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Feeding the Aging Heart

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Abstract

More than 10% of all dogs and cats have cardiac disease that, especially in dogs, becomes increasingly common with age. However, even in the absence of obvious disease, changes in cardiac function occur with aging. Nutrition plays an important role in the management of cardiac disease and also may help to optimize cardiac function.

Glossary of Abbreviations

ACE: Angiotensin-Converting Enzyme
AAFCO: Association of American Feed Control Officials
ATE: Arterial Thromboembolism
CHF: Congestive Heart Failure
CVD: Chronic Valvular Disease
DCM: Dilated Cardiomyopathy
DHA: Docosahexaenoic Acid
EPA: Eicosapentaenoic Acid
HCM: Hypertrophic Cardiomyopathy
IL-1: Interleukin-1β
ISACHC: International Small Animal Cardiac Health Council
TNF: Tumor Necrosis Factor-α

Cardiac Disease in Dogs and Cats

Cardiac disease is one of the most common disorders in both dogs and cats. Approximately 11% of dogs have cardiac disease;[1] 95% of these dogs have adult-onset (acquired) cardiac disease. For dogs with acquired disease, the majority have endocardiosis (commonly referred to as mitral regurgitation due to chronic valvular disease or [CVD]), with smaller numbers having dilated cardiomyopathy (DCM) or the less common pericardial disease, endocarditis, primary arrhythmias, or heartworm disease. Small- to medium-sized dog breeds are predisposed to CVD, while DCM is the most common cause of congestive heart failure (CHF) in large-breed dogs.

Hypertrophic cardiomyopathy (HCM) currently is the most common form of cardiac disease in cats, but other forms of cardiomyopathy (i.e., dilated, restrictive or unclassified) and other diseases (e.g., heartworm disease, pericardial disease) also can occur. Cardiac disease often is perceived as a relatively uncommon disease in cats, but results of recent studies suggest that up to 16% of apparently healthy cats may have cardiomyopathy.[2,3] Thus, cardiac disease appears to be a very common disease in the feline population and, regardless of the cause, often leads to CHF, arterial thromboembolism (ATE), syncope or sudden death.

Age-Associated Cardiac Changes

In both dogs and cats, most cardiac disease occurs more commonly in older animals. However, even in the absence of disease, the aging heart undergoes a variety of changes that affect the susceptibility to disease and response to stress, as well as contributing to functional deficits. Changes that have been identified in aging are listed in Table 1 and include cardiac hypertrophy, altered vascular resistance and abnormal endothelial function.[4] Note that these changes have been most often studied in rodents or humans; much research needs to be done in the aging canine and feline heart.

Treatment of Cardiac Disease

Few of the common cardiac diseases in dogs and cats currently are easily corrected. Pacemaker implantation can be successfully accomplished in animals with severe bradycardia, certain pericardial diseases are much improved following surgery, and surgical replacement or repair of the mitral valve is possible where cardiopulmonary bypass is available. However, the vast majority of the common cardiac diseases in dogs and cats do not have a surgical solution, yet these diseases can often be successfully managed medically. Medical therapy of cardiac disease has improved in recent years, with newer and more effective drugs, but medical therapy still is only palliative. Goals of medical care include controlling clinical signs, slowing the progression of disease, and improving quality of life. Maintaining good quality of life is particularly important in dogs and cats, as owners often prefer quality of life to "quantity" of life.

Careful attention to the diet of animals with cardiac disease is a key component of optimal medical treatment of these patients. In the past, the goal of nutritional management for animals with cardiac disease was purely symptomatic and focused mostly on sodium restriction. This was primarily due to the limited number of medications available for treatment, and in that situation, sodium restriction was beneficial for

Table 1

Cardiac Changes	Vascular Changes
Structural • Increased o Heart weight o Cardiomyocyte size o Collagen o Inflammatory mediators (e.g., cytokines, reactive oxygen species) • Decreased o Cardiomyocyte number o Functional atrial pacemaker cells o Mitochondrial function	*Structural* • Increased o Vascular stiffness o Arterial wall thickness o Total peripheral resistance o Endothelial permeability • Decreased o Endothelial nitric oxide o Elasticity and distensibility of the vasculature
Functional • Increased o End-diastolic filling o Contraction duration • Decreased o Early diastolic filling o Responsiveness to β-adrenergic stimulation (chronotropic and inotropic) o Peak cardiac output with exercise o Lusitropic function o ATP production o Energy reserve	*Functional* • Increased o Blood pressure • Decreased o Endothelial function o β-adrenergic-mediated vasodilation

Adapted from: Ferrari, et al. *J Appl Physiol*. 2003;95:2591-2597.[4]

reducing fluid accumulation in animals with CHF. The development of more effective medications has made severe sodium restriction less important in most animals with cardiac disease. Current goals for the nutritional management of animals with cardiac disease are to maintain optimal body condition, avoid nutritional deficiencies and excesses, and to gain potential benefits from pharmacologic doses of certain nutrients. Integrating nutrition into the care of animals with cardiac disease may reduce the number or doses of medications an animal requires, reduce complications, improve quality of life, and may slow the progression of the disease.

Optimal Body Condition

Cardiac cachexia is the loss of lean body mass that occurs in CHF. In healthy animals, weight loss is associated primarily with reductions in fat, and lean tissue is relatively spared. However, weight loss in CHF comes from the metabolically active lean body mass. Cachexia has important detrimental effects in the cardiac patient and is an independent risk factor for mortality. One study found that 50% of dogs with CHF had some degree of cachexia.[5]

The deleterious effects of cachexia and the role of body weight and body composition in heart failure have been emerging. While obesity is a risk factor for development of heart disease in people, obesity may actually be associated with a protective effect once heart failure is present. This is known as the obesity paradox. A recent large meta-analysis on body condition in people with heart failure concluded that obesity and being overweight were associated with lower all-cause and cardiovascular mortality and that underweight patients consistently had a higher risk[6] of death. Given the adverse effects associated with cachexia, the association between obesity and improved survival in heart failure appears to be due to a lack of cachexia, rather than obesity per se. This is likely due to the increased reserve of lean body mass in overweight and obese people. The obesity paradox also has been demonstrated in dogs and cats with heart failure, and recent studies suggest a "U-shaped" curve with the worst survival for those with the lowest and highest weights.[7] These data emphasize the importance of avoiding weight (and muscle) loss, as well as severe obesity, in the patient with heart failure.

Cardiac cachexia is a multifactorial syndrome with contributing factors including reduced or altered appetite, increased energy requirements, and increased production of inflammatory

cytokines (e.g., tumor necrosis factor-α [TNF], interleukin-1β [IL-1]). These cytokines cause anorexia, increased energy requirements and loss of lean body mass. Omega-3 fatty acids reduce inflammatory mediators, including cytokines. Fish oil, which is high in omega-3 fatty acids, decreases cachexia, and, in some dogs with CHF-induced anorexia, improves food intake.[5]

Nutritional Excesses

Sodium

Sodium excretion is reduced in cardiac disease, so sodium restriction is recommended for dogs and cats with heart disease. However, dietary sodium restriction can further activate the renin-angiotensin-aldosterone system so the authors do not recommend severe sodium restriction in early heart disease. For animals with asymptomatic cardiac disease ISACHC (International Small Animal Cardiac Health Council) Stage 1a and 1b,<100 mg sodium/100 kcals in the diet is recommended. For animals in ISACHC Stage 2, <80 mg sodium/100 kcal is recommended, and for those in ISACHC Stages 3a and 3b, <50 mg sodium/100 kcals is recommended. However, further research is needed to develop optimal recommendations for dose and timing of sodium restriction in animals with cardiac disease.

Potassium

Angiotensin-converting enzyme (ACE) inhibitors are commonly used in animals with cardiac disease. These drugs cause increased serum potassium and a small proportion of animals develop hyperkalemia. Spironolactone, now used in some dogs and cats with CHF, is an aldosterone antagonist and a potassium-sparing diuretic. Some animals receiving ACE inhibitors or spironolactone can develop hyperkalemia. As some commercial cardiac diets contain increased potassium concentrations to counteract the theoretical potassium loss due to diuretics, these diets can contribute to hyperkalemia. Therefore, monitoring serum potassium and consideration of the dietary potassium content is recommended.

Nutritional Deficiencies/Nutritional Pharmacology

Protein and Amino Acids

Protein

The authors strongly recommend avoiding restriction of dietary protein intake in dogs and cats with cardiac disease, unless warranted by concurrent disease, as dietary protein restriction can contribute to loss of lean body mass. Dietary protein restriction could occur unintentionally in an animal with cardiac disease by recommending a renal diet. In addition, some of the commercial cardiac diets are very low in protein. Reduced protein diets should be avoided in all animals with cardiac disease unless severe renal dysfunction is present, and dietary protein should be fed to at least meet Association of American Feed Control Officials (AAFCO) minimums for adult dogs (5.1 gm/100 kcal) or cats (6.5 gm/100 kcals).

Taurine

There has been a dramatic reduction in the incidence of feline dilated cardiomyopathy (DCM) since the late 1980s when increased dietary supplementation of taurine was instituted in commercial pet foods. Most current cases of feline DCM are unrelated to taurine deficiency, but taurine deficiency should be suspected in all cases of feline DCM, especially in cats fed a poor quality, homemade, vegetarian, or otherwise unbalanced diet.

Taurine deficiency is now suspected in some cases of canine DCM. Unlike cats, most dogs should be able to synthesize adequate amounts of taurine and so are not thought to require dietary taurine. Most dogs with DCM do not have taurine deficiency, but low taurine concentrations have been found in some dogs with DCM. Low blood taurine concentrations are most commonly reported for American Cocker Spaniels, and large-breed dogs such as Golden Retrievers, Labrador Retrievers, Newfoundlands, Portuguese Water Dogs, and Irish Wolfhounds.

Taurine deficiency in dogs may be related to dietary factors as it appears to be associated more commonly with certain lamb meal and rice-based diets or high-fiber diets, and has been induced by feeding a low-protein, low-taurine diet long term to dogs. Taurine deficiency also may be the result of increased renal or fecal loss of taurine, or other metabolic defects present in certain breeds. Taurine supplementation may be beneficial in some dogs with taurine deficiency, but even in dogs that respond, the response is typically not as dramatic as in taurine-deficient cats with DCM. Further research is required to determine the role of taurine in canine DCM. In addition to the potential for correcting a deficiency, some of the potential benefits of taurine may be due to its positive inotropic effects or role in calcium regulation in the myocardium.

Fat

Omega-3 Fatty Acids

The omega-3 fatty acids, eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), are normally present in low concentrations in the diet and subsequently low levels are in cell membranes. However, dietary and membrane concentrations can be increased by a food or supplement enriched in omega-3 fatty acids. Dogs with CHF have a relative deficiency of EPA and DHA compared to unaffected dogs, and fish oil supplementation normalizes these abnormalities.

Omega-3 fatty acids also have a number of other effects that may be beneficial in animals with cardiac disease, including a reduction in inflammatory mediators (e.g., eicosanoids, TNF, IL-1). In addition, omega-3 fatty acids have antiarrhythmic effects. Although an optimal dose of omega-3 fatty acids has not been determined, the authors currently recommend a dosage of fish oil to provide 40 mg/kg EPA and 25 mg/kg DHA for dogs and cats with cardiac disease. Omega-3 fatty acids appear to be particularly useful in animals with anorexia or cachexia but also may be beneficial in animals with less advanced cardiac disease

(e.g., DCM, CVD, HCM).

A few specially designed therapeutic diets contain high levels of omega-3 fatty acids, but for most animals, supplementation will be necessary to achieve a sufficiently high omega-3 fatty acid dose. When recommending a supplement, it is important to know the exact amount of EPA and DHA in the specific brand fish oil since supplements vary widely. The most common formulation of fish oil, however, is one gram capsules that contain approximately 180 mg EPA and 120 mg DHA. At this concentration, fish oil can be administered at a dose of one capsule per 10 pounds of body weight to achieve the authors' recommended EPA and DHA dosage.

Fish oil supplements should contain vitamin E as an antioxidant, but other nutrients should not be included to avoid toxicities. Similarly, cod liver oil should not be used to provide omega-3 fatty acids because it contains high levels of vitamins A and D, which can result in toxicity. Flaxseed or flaxseed oil also should not be used because its omega-3 fatty acids cannot be efficiently converted to EPA and DHA in dogs (and particularly in cats).

Vitamins

B Vitamins

Anorexia and increased urinary loss of water-soluble vitamins associated with diuretic use could predispose animals with CHF to low B vitamin concentrations. Thiamine deficiency is known to be a cause of cardiomyopathy and a consequence of CHF in people, but there has been little investigation into the role of B vitamins in animals with cardiac disease.

Minerals

Magnesium

Magnesium plays an important role in normal cardiac function. Animals with CHF may be at increased risk for magnesium deficiency due to diuretics and other cardiac medications. Hypomagnesemia can increase the risk of arrhythmias, decrease cardiac contractility, cause muscle weakness, contribute to renal potassium loss, and can potentiate the adverse effects of certain cardiac medications. Hypomagnesemia has not been a consistent finding in studies of animals with heart disease but this may be because serum magnesium concentration is a poor indicator of total body stores. Monitoring magnesium status in animals with CHF can identify those that would benefit from supplementation.

Other Nutrients

L-Carnitine

L-carnitine is critical for fatty-acid metabolism and energy production and is concentrated in skeletal and cardiac muscle. While carnitine deficiency is associated with primary myocardial disease in a number of species, including a family of Boxer dogs, L-carnitine supplementation may have benefits even if a deficiency is not present by improving myocardial energy production.

Table 2. Treats for Dogs with Heart Disease

Acceptable Treats and Foods That Can Be Used to Increase Palatability

Note: All foods in this list should be prepared without salt.

- Pasta
- Rice (plain white or brown rice, not flavored rice)
- Honey
- Maple syrup
- Low-sodium cheese
- Lean meats, cooked (chicken, turkey, beef, or fish) – not sandwich meats/cold cuts
- Eggs, cooked
- Homemade soup — not canned soups
- Low-salt breakfast cereal (such as Frosted Mini-Wheats) — the label should read "this is a low-sodium food"
- Fresh vegetables/fruit (such as carrots, green beans, apple, orange, banana [avoid grapes])
- Dog treats that are low in sodium:
  Purina® Alpo® Variety Snaps Treats;
  Purina Veterinary Diets® Lite Snackers;
  Iams® Adult Original Formula Small Biscuits;
  Science Diet® Simple Essentials Training Treats™;
  Stewart® Fiber Formula® Medium Dog Biscuits

Foods to Avoid

- Fatty foods (meat trimmings, cream, ice cream)
- Baby food
- Pickled foods
- Bread
- Pizza
- Condiments (ketchup, soy sauce, barbeque sauce, etc.)
- Sandwich meats/cold cuts (ham, corned beef, salami, sausages, bacon, hot dogs)
- Most cheeses, including "squirtable" cheeses (unless specifically labeled as "low sodium")
- Processed foods (such as potato mixes, rice mixes, macaroni and cheese)
- Canned vegetables (unless "no salt added")
- Potato chips, packaged popcorn, crackers and other snack foods
- Soups (unless homemade without salt)
- Most dog biscuits and other dog treats

Coenzyme Q10

Coenzyme Q10 also is a co-factor required for energy production. In addition, it has antioxidant properties. There are many anecdotal reports of benefits to coenzyme Q10 supplementation, but controlled prospective studies are necessary to accurately judge the efficacy of this product. Most human studies of coenzyme Q10 supplementation have not been well-controlled and results are conflicting. Possible reasons

Tips for Administering Medications

Foods commonly used to administer medications can provide a large amount of additional sodium in your dog's diet. Better ways clients can give medications include:

- Have a doctor or technician teach them how to give medications without using food
- Insert medications into one of the following foods:
  - Fruit (for example, banana, orange, melon [avoid grapes])
  - Low-sodium cheese
  - Low-sodium canned pet food
  - Peanut butter (labeled as "no salt added")
  - Home-cooked meat such as chicken or hamburger (without salt), not lunch meats

for the reported benefits of supplementation include correction of a deficiency, improved myocardial metabolic efficiency, or increased antioxidant protection.

Antioxidants

Reactive oxygen species are a normal byproduct of oxygen metabolism and are typically adequately compensated through the production of endogenous antioxidants. Normally, there should be a balance between oxidants and antioxidants, but dogs with CHF have been shown to have an imbalance between oxidant production and antioxidant protection. Antioxidants are produced endogenously but also can be supplied exogenously with either enzymatic antioxidants (e.g., superoxide dismutase, catalase, glutathione peroxidase) or oxidant quenchers (e.g., vitamin C, vitamin E or glutathione). While dietary antioxidants have been shown to increase antioxidants and to reduce oxidants, clinical benefits of antioxidant supplementation in animals with cardiac disease have not yet been demonstrated.

Clinical Issues

The process of choosing an appropriate diet for an animal with cardiac disease involves examining the patient, the diet, and the owner's feeding practices and considering all the issues at hand. It is important to assess all these factors to determine which diet or diets might best suit an individual patient.

The Patient

In general, the nutrients of concern in cardiac patients are calories, sodium and chloride, protein, potassium, and magnesium. However, patients with cardiac disease vary tremendously in terms of their clinical signs, laboratory parameters, and food preferences, and these all affect diet selection. For example, dogs with asymptomatic heart disease require less severe sodium restriction than those with CHF. Thin cats require a more calorically dense diet than would a normal or overweight cat. Laboratory results (e.g., hypokalemia versus hyperkalemia) and concurrent diseases also influence diet choice. For animals with acute CHF, dietary changes should be avoided until the patient is stabilized. Once the animal is home and stabilized on medications, a gradual change to a new diet can be made. Forced dietary changes when the animal is sick can induce food aversions.

The Diet

Based on these and other patient parameters, a diet or diets can be matched to the individual patient. For an animal with cardiac disease without CHF (i.e., an asymptomatic dog with CVD or cat with HCM), the authors recommend only mild sodium restriction and counseling the owner to avoid diets high in sodium, and treats or table food high in sodium. Most owners need very specific instructions regarding which foods are appropriate. When CHF first arises, additional sodium restriction is recommended, but attention to providing adequate protein and optimal levels of other nutrients of concern also is important.

As CHF becomes more severe, more severe sodium restriction may allow lower dosages of diuretics to be used to control clinical signs. Careful selection is important to achieve not only the desired sodium level, but also appropriate levels of protein, potassium, magnesium and other nutrients. Above all, the diet must be palatable enough that the animal will willingly eat it to aid in maintaining optimal weight.

There is usually not a single "best" diet for any patient. The authors typically select several diets that are appropriate for an individual patient based on the patient, diet and feeding practices. These diets are offered as choices for the owner and for the pet so they can determine the one that works best. Having multiple appropriate dietary choices is particularly beneficial for animals with more advanced CHF, in which a cyclical or selective loss of appetite is common.

Feeding Practices

While it is important to find a diet or diets that the individual animal likes and will willingly eat to maintain optimal body condition, it also is necessary to meet the owner's expectations in terms of diet. A pet's quality of life is of tremendous importance to owners of pets with cardiac disease so providing diets that are palatable and readily eaten is critical. Also, be sure to address not only the pet food but also the treats, table food and foods used to administer medication.

Most dogs and many cats receive treats, so it is important to specifically discuss treats with the owner. Most owners are unaware of treats that would be contraindicated (e.g., high salt treats or table food). The author typically provides a list of foods that are appropriate and foods to avoid as treats to assist the owner in wise selection (see Table 2).

In addition, most dog owners (and many cat owners) give medications with "people food." Including this information in

the overall diet plan is important to achieve success with nutritional modification. The type of food preferred also varies between owners (and pets, e.g., canned versus dry versus homemade) and must be taken into consideration. Finally, cost preferences should be considered as veterinary therapeutic diets may be out of the price range for long-term use by some owners. In these cases, lower-priced alternatives should be offered.

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For Further Reading

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Q&A Discussion

Q: Dr. Margie Scherk, Vancouver: Can you clarify in the U-shaped curve showing survival versus body weight in the cats, was the weight taken at the time of diagnosis or during progression of disease?

A: Dr. Freeman: The curve of survival in the cats was their weight or their body condition at the time of diagnosis of heart failure — not just heart disease but heart failure. We also did look at changes in weight over time in both feline and canine patients. And, in dogs, it was significant. Dogs that gained weight actually did better than if they lost weight. But we did not find that in the cats, in terms of the change in the weight.

Q: Dr. Bob Backus, University of Missouri: I have a quick question on magnesium that you have listed as a nutrient to consider. How do you assess status? You mentioned, and I think a lot of us believe, that serum is not a very accurate indicator of status. Do you have any suggestions on how we should assess magnesium status?

A: Dr. Freeman: We measure total magnesium in our hospital, but we also measure ionized magnesium. You are absolutely right that serum magnesium is a poor indicator of the patient's status. Certainly, if they are low on total magnesium, that is something to pay attention to, but I think that ionized magnesium is a little bit more accurate indicator, so we would keep track of that as well. We also start looking a little bit more critically if the animal is having arrhythmias or other issues that may be the result of low magnesium status. And then the other thing is looking at the diet. If they are also on a low magnesium diet or on a lot of diuretics, I may have a higher index of suspicion for magnesium depletion.