CARDIAC EDUCATION group
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CIRCULATIONS
conversations with a cardiologist

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Ask the Expert:
Part 1: Nutritional Management of Heart Disease

WHY CAN'T WE USE ONE OR TWO SPECIFIC DIETS TO "COVER" OUR HEART DISEASE PATIENTS' NEEDS?

The same diet is not optimal for every dog or every cat with heart disease. Needs may vary based on body pre-existing condition at the time illness occurs, the metabolic changes caused by the disease itself, concurrent illnesses, and the preferences of the individual patient (and its owner). Therefore, to optimize care for patients, it is important to individualize the nutritional plan, just as veterinary clinicians are comfortable individualizing medication plans. General recommendations such as "just feed a low salt diet" or "give a senior diet" will result in suboptimal outcomes.

WHY DO PATIENTS LOSE CONDITION WHEN THEY HAVE HEART DISEASE? IS THAT WHAT "CARDIAC CACHEXIA" MEANS?

A common misconception is that "cachexia" refers only to end-stage, emaciated animals. The term "cachexia" actually refers to a loss of lean body mass and, in fact, is subtle in the early stages of heart disease. Muscle loss usually is noted first in the epaxial, gluteal, scapular, or temporal muscles. Cachexia can even occur in obese animals, because body composition changes in heart disease. Animals with congestive heart failure (CHF) demonstrate changes in appetite, ranging from changes in food preferences (dysrexia), to a reduction in food intake (hyporexia), to a complete loss of appetite (anorexia). While these changes in appetite and intake may contribute to reduced food intake and weight loss in any animal, the weight loss that occurs in CHF is very different than that which occurs in a healthy animal. If a healthy animal receives insufficient calories, it will lose primarily fat, but a dog or cat with CHF that does not eat adequate calories primarily loses lean body mass or muscle. This muscle loss (cachexia) is common in CHF, particularly as disease progresses, and has negative effects on muscle strength, immune function, and survival. Changes in food intake and increased energy requirements both contribute to this loss of lean body mass, but increased production of inflammatory cytokines [e.g., tumor necrosis factor-alpha (TNF) and interleukin-1-beta (IL-1)] is a primary mediator of cardiac cachexia.

WHAT CAN I DO TO MANAGE MY PATIENT'S LOSS OF WEIGHT OR LEAN BODY MASS?

In addition to recording body weight and body condition score (BCS), careful assessment of the muscle condition score (MCS) at every visit is important to recognize muscle loss at an early stage when there is a better opportunity to successfully manage it. Ensuring adequate protein intake is important. Diets that are restricted in protein (e.g., renal diets or diets with protein < 5.1 gm/100 kcal for dogs or 6.5 gm/100 kcal for cats) should be avoided, unless concurrent advanced kidney disease is present. Adequate protein intake is a key goal in animals with heart disease, particularly as CHF becomes more severe. Feeding a diet restricted in protein (e.g., renal diets, some senior diets) may contribute to unwanted muscle loss.

Animals with CHF often have periods of dysrexia, hyporexia and anorexia. Changes in appetite may suggest a need to adjust medications, but fluctuating appetite occurs even when CHF is well controlled. Owners often describe the animal preferring different foods than it used to or eating one food well for several days and then not wanting it anymore (but that the pet will eat a different food). It's helpful to warn the owner about these typical appetite changes and to provide multiple appropriate diet options for the owner to feed (i.e., they can feed one diet for several days until the animal no longer has interest for that food and then switch to a different food). The pet will often be willing to eat the original food again at a future time so owners can use a "rotation" of foods to keep the animal eating. It is important to advise the owner that if the animal also

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is acting lethargic, is tachypneic, won't eat any foods or if their pet has been eating very well and then suddenly stops eating, this usually indicates the need for reassessment and adjustment of medications.

WHAT TYPES OF NUTRITIONAL DEFICIENCIES ARE ASSOCIATED WITH HEART DISEASE?

There are a variety of deficiencies that can occur in animals with heart disease. Nutritional deficiencies may directly cause heart disease [e.g., taurine deficiency-induced dilated cardiomyopathy (DCM) in cats] or may occur secondary to CHF or heart failure therapy (e.g., hypokalemia, hypomagnesemia). There are also some nutrients that, if provided at levels above and beyond their nutritional requirements, may have pharmacologic benefits (i.e., nutritional pharmacology). It is not always clear whether benefits are being derived because of correction of deficiency or from pharmacologic effects. For example, providing taurine supplementation to a cat with taurine deficiency can correct the deficiency and result in reversal of DCM. However, taurine also has antioxidant and positive inotropic effects. Therefore, some benefits of nutrients in animals with heart disease may also be derived from these non-nutritional effects.

▶ Deficiencies that may occur in heart disease patients

- **Taurine deficiency:** Taurine can play a role in the development of feline DCM and in some cases of canine DCM. Taurine deficiency-induced DCM is now uncommon in cats but should be suspected if cats are eating diets that are vegetarian, home-prepared, or made by a manufacturer with suspect quality control measures. Some dogs with DCM also can have taurine deficiency, and the index of suspicion should be higher in predisposed breeds (e.g., American cocker spaniels, St. Bernard dogs, Newfoundland dogs, golden retrievers), particularly if they are eating lamb and rice, high fiber, or very low protein diets.
- **Omega-3 fatty acids:** Dietary fat composition can have significant benefits with regard to inflammation, immune function, and hemodynamics. Most pet foods contain primarily omega-6 fatty acids (e.g., linoleic acid, arachidonic acid). Omega-3 fatty acids are normally are found in very low concentrations in the cell membrane compared with the omega-6 fatty acids, but their levels can be increased by consumption of a food or supplement enriched in omega-3 fatty acids. In one study, dogs with CHF had a relative deficiency of omega-3 fatty acids, which was corrected after supplementation of the omega-3 fatty acids (eicosapentanoic acid [EPA] and docohexaenoic acid [DHA]) from fish oil. The benefit of having a higher concentration of omega-3 fatty acids in cell membranes is that breakdown products of the omega-3 fatty acids (eicosanoids) are less potent inflammatory mediators than eicosanoids derived from omega-6 fatty acids. This decreases the production of cytokines and other inflammatory mediators. Omega-3 fatty acids also reduce the production of TNF and IL-1, minimize muscle loss, and have antiarrhythmic effects. In some animals, omega-3 fatty acid supplementation improves appetite.
- **Minerals:** Hypokalemia and hypomagnesemia can occur in CHF, especially when high doses of loop diuretics are used. Hypokalemia and hypomagnesemia can cause weakness and increase risk for arrhythmia.
- **Vitamins:** B vitamin deficiencies appear to be relatively common in people with CHF due to urinary losses from diuretic use. Similar studies have not been published in dogs with CHF but one study in cats with cardiomyopathies showed reduced serum concentrations of vitamins B6 and B12. If owners are feeding their animals home-cooked diets, a variety of vitamin (and other) deficiencies are possible. Studies have shown that, unless formulated by a board-certified veterinary nutritionist (American College of Veterinary Nutrition or European College of Veterinary Comparative Nutrition), nearly all home-cooked diets are nutritionally unbalanced. Nutritional deficiencies also can occur in animals fed commercial foods if the foods are made by companies with lax nutritional/quality control.

HOW DO I DETECT THESE DEFICIENCIES AND WHAT CAN I DO ABOUT THEM?

▶ Taurine deficiency:

- **Cats with DCM:** Although most current cases of feline DCM appear to be independent of taurine status, taurine deficiency still should be suspected whenever the diagnosis of feline DCM is made. Ideally, both plasma and whole blood taurine concentrations should be analyzed since whole blood is the best indicator of status during depletion, but plasma taurine is the best indicator if repletion has already begun (if cost is an issue, at least whole blood taurine concentrations should be analyzed). Taurine supplementation (125 to 250 mg PO q 12 h) and medical therapy should begin concurrently while test results are pending. If the cat is eating an unconventional diet that is likely to be unbalanced or a food produced by a small manufacturer, the owner also should be counseled to switch to a nutritionally balanced commercial cat food made by a well-known manufacturer.
- **Dogs with DCM:** Plasma and whole blood taurine concentrations (or at least whole blood concentrations) should be evaluated in predisposed breeds or dogs eating lamb and rice, high fiber, or very low protein diets. The

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benefits of taurine supplementation are far less certain in canine DCM than in feline taurine deficiency-induced DCM. Although several small studies have shown that taurine supplementation can improve some clinical or echocardiographic parameters in taurine-deficient dogs, the response is generally not as dramatic as is seen in cats with taurine deficiency–induced DCM. Nonetheless, supplementing taurine is recommended until taurine assay results are available. The optimal dose of taurine for correcting a deficiency has not been determined, but recommended dosages range from 250-1000 mg PO q 12 h.

Omega-3 fatty acids:

- There is no routine clinical test to evaluate omega-3 fatty acid deficiency, but dogs with CHF in one study had lower concentrations of omega-3 fatty acids compared to healthy controls. Supplementation of omega-3 fatty acids will increase the concentration of omega-3 fatty acids in blood and in cell membranes. The breakdown products of the omega-3 fatty acids (eicosanoids) are less potent inflammatory mediators than eicosanoids derived from omega-6 fatty acids. Increased concentration of omega-3 fatty acids decreases the production of cytokines and other inflammatory mediators. Omega-3 fatty acids also reduce the production of TNF and IL-1, minimize muscle loss, and have antiarrhythmic effects. In some animals, omega-3 fatty acid supplementation improves appetite.
- The author currently recommends a daily dose of fish oil to provide 40 mg/kg of EPA and 25 mg/kg of DHA for dogs with anorexia or cachexia and also for most dogs and cats with heart disease if there are no contraindications (e.g., dietary fat intolerance, coagulopathies). Unless the diet is one of a few specially designed veterinary diets, supplementation is necessary to achieve this omega-3 fatty acid dose. When using fish oil supplements, it is important to know the exact amount of EPA and DHA in a specific fish oil brand since supplements vary widely. However, the most common formulation of fish oil is 1-g capsules that contain approximately 180 mg of EPA and 120 mg of DHA. At this concentration, fish oil can be administered at a dose of 1 capsule per 10 pounds of body weight per day to achieve the recommended EPA and DHA dosages. Cod liver oil and flaxseed oil should not be used to provide omega-3 fatty acids to dogs and cats, and fish oil supplements containing nutrients other than vitamin E (a small amount of vitamin E is required to protect the fish oil) should be avoided due to potential toxicities.

Minerals:

- **Potassium:** Serum potassium should be monitored regularly in animals with CHF. Commercial pet foods vary widely in their potassium content so if hypokalemia is present, commercial pet foods should be selected that have the other desired nutrient properties (i.e., adequate protein, restricted in sodium) and are high in potassium. Alternatively, potassium supplements can be given.
- **Magnesium:** Serum magnesium concentrations also should be measured in animals with CHF, although serum magnesium concentrations are a less than ideal indicator of total body stores (ionized magnesium is a better indicator if this measurement is possible in your hospital). Nonetheless, serial evaluations in an individual patient may be useful, especially in patients with arrhythmias or in those receiving high doses of diuretics. Diets also vary in their magnesium content so careful diet selection may help to maintain normal magnesium concentrations in a hypomagnesemic animal. However, magnesium supplements may be required if diet alone is insufficient.

Vitamins:

- A complete diet history should be performed on every animal, at every visit, to assess the patient for risk of deficiencies, i.e., if an unbalanced diet is suspected or reported (e.g. the pet eats a homemade diet that has not been formulated by a board-certified veterinary nutritionist, will eat only cooked meat or processed lunchmeat, etc.) or if high doses of diuretics are used.

WHICH NUTRITIONAL EXCESSES SHOULD I BE CONCERNED ABOUT IN MY PATIENTS, AND WHAT CAN I DO TO LIMIT THE PROBLEM?

Increased sodium and water retention:

- Because increased retention of sodium and water occurs in animals with heart disease due to activation of the renin-angiotensin-aldosterone system and other neuroendocrine systems, excess sodium in the diet may hinder successful medical therapy. Sodium and water retention can begin even before overt CHF is present.
- Sodium restriction is recommended for all stages of heart disease, but the degree of restriction should vary with the stage of disease. The author does not recommend severe sodium restriction in the early stages of disease, since this can further activate the renin-angiotensin-aldosterone system. Instead, mild restriction (<100 mg sodium/100 kcal) is recommended when heart disease but not heart failure is present (i.e. ACVIM Stage B or International Small Animal

Cardiac Health Council [ISACHC] Class 1a and 1b heart disease). It's important to begin to educate owners about nutrition and heart disease, particularly with respect to foods that are high in sodium, while their pet is in the early phases of heart disease. Most owners are unaware of the sodium content of pet and human foods and need very specific instructions regarding appropriate pet foods, acceptable low-sodium treats, and methods for administering medications. Owners also should be counseled on specific foods to avoid, such as baby food; bread; cheeses; lunch meats and cold cuts (e.g., ham, corned beef, salami, sausages, bacon, hot dogs); most pet treats; rawhides and bully sticks; etc). As heart disease progresses, additional dietary sodium restriction is recommended: In the early stages of CHF (first occurrence, ACVIM Stage C, ISACHC Class 2) the author aims for a diet <80 mg sodium/100 kcal and in late stage CHF (ACVIM Stage C or D, ISACHC Class 3), further sodium restriction may be useful.

▶ Potassium:

- Hypokalemia can occur in animals with CHF, especially those receiving moderate or higher doses of furosemide and anorexic or hyporexic patients. Hyperkalemia also is possible in patients receiving angiotensin converting enzyme (ACE) inhibitors or aldosterone receptor antagonists.
- Serum potassium should be monitored regularly in animals with CHF. Commercial pet foods vary widely in their potassium content so if hyperkalemia is present, commercial pet foods should be selected that have the other desired nutrient properties (i.e., adequate protein, restricted in sodium) and are low in potassium.

▶ Other nutrient excesses:

- If owners are providing dietary supplements to their animals, a variety of vitamin, mineral, and other excesses are possible.
- A complete diet history should be performed on every animal at every visit to assess them for risk of nutritional excesses or interactions between the supplements and cardiac medications (or between the supplements themselves).

Recommended Reading

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