Dietary considerations for dogs suffering from cardiac disease

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Managing the diet of a dog with cardiac disease depends on the animal's clinical signs and stage of heart failure (Figure 1**), as well as any concurrent diseases.**

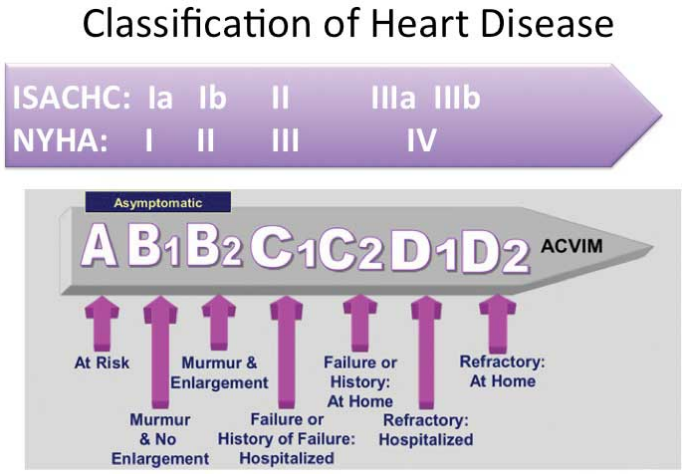

Figure 1. American College of Veterinary Internal Medicine graphic showing the stages of heart failure. IMAGE: ©Clarke Atkins.

In the past, severe sodium restriction was the main recommendation for patients with heart disease.
This idea has been modified and the roles of other nutritional factors are becoming more important.

Maintenance of optimal weight

Cardiac cachexia is a common problem in dogs with chronic heart failure. Cachexia involves a loss of lean body mass, with direct and deleterious effects on strength, immune function and survival.

Cachexia is sometimes thought of as the classic end-stage picture of an emaciated dog or cat.

Actually, cachexia can be very subtle initially and can even occur in an obese animal.
Loss of lean body mass is usually first seen in the epaxial, gluteal, scapular and temporal muscles.

Anorexia is present in up to 75 per cent of dogs with heart disease. Other causes of cachexia include increased energy requirements and metabolic alterations. It may be secondary to fatigue, dyspnoea, medication toxicity or unpalatable diets.

The inflammatory cytokines, particularly tumour necrosis factor alpha (TNF?), interleukin-1 (IL-1) beta and interleukin-6 (IL-6), appear to be the primary mediators of cachexia.

These inflammatory cytokines directly cause anorexia, increase lean body mass loss and may increase energy requirements.

Tumour necrosis factor, IL-1 and IL-6 also cause cardiac myocyte hypertrophy and fibrosis and have negative inotropic effects, so may directly contribute to progression of disease.

Supplementation of omega-3 (n-3) polyunsaturated fatty acids has been recommended for cardiac cachexia.

Fish oil, which is high in n-3 fatty acids, can decrease cytokine production in dogs with congestive heart failure (CHF) and improve cachexia. In some dogs with CHF-induced anorexia, fish oil supplementation improves food intake.

In addition, reduction of cytokines has been correlated with survival in dogs with CHF (Freeman et al, 1998).

Obesity

The implications of obesity in small animals with heart disease are not well studied, though the coronary artery disease of humans is not common in dogs or cats.

However, obesity may negatively affect cardiac output, pulmonary function, neurohumoral activation, blood pressure and heart rate.

In some dogs with CHF, exercise restriction is recommended so their weight loss must come entirely from caloric restriction.

Once heart disease has been diagnosed, extra bodyweight appears to result in longer survival – a feature known as the obesity paradox. This is likely to be due to the increased lean body mass in heavier individuals rather than the increased fat.

Sodium

Table 1. Sodium recommendations for dogs, based on stage of disease			
ACVIM cardiac classification	**Description**	**Diagnostic findings and signs**	**Dietary sodium recommendation**
A	No heart failure, but breed at risk.	None.	Advise owner about high sodium treats and human foods.
B1	Asymptomatic; heart disease is detectable, but dog does not show signs of heart failure.	Could include a heart murmur, arrhythmia, cardiac chamber enlargement. No signs of volume or pressure overload.	Advise owner to avoid high sodium diets (more than 100mg/100kcal), high salt treats and human food.
B2	Mild to moderate heart failure; signs are evident at rest or mild exercise and affect quality of life.	Signs include coughing, tachypnoea, respiratory distress, mild to moderate ascites.	Dietary sodium content of 50mg/100kcal to 80mg/100kcal and avoid high salt foods as above.
C	Advanced heart failure; home care is possible.	Signs are obvious. May include respiratory distress, marked ascites, exercise intolerance or hypoperfusion at rest.	Sodium content less than 50mg/100kcal.
D	Advanced heart failure; hospitalisation necessary.	Dog may be in life-threatening shock, oedema, ascites or pleural effusion.	Diet changes not done while dog is in hospital.

Table 1. Sodium recommendations for dogs, based on stage of disease.

Sodium excretion is reduced even in early cardiac disease, so sodium restriction has been typically recommended.

However, as dietary sodium restriction can further activate the renin-angiotensin-aldosterone (RAA) system, it is not clear whether sodium restriction in early heart disease is beneficial or harmful.

Studies of sodium restriction plus the use of furosemide in normal dogs resulted in hyperkalaemia of some of the dogs (Roudebush et al, 1994).

Although one study in dogs with CHF showed measures of cardiac size decreased significantly on a low-sodium diet, the effect of low-sodium diets on survival or progression of disease has not been studied (Rush et al, 2000).

Further, sodium has not been shown to directly affect the blood pressure of dogs or cats.

Due to the stimulation of the RAA system, excessive sodium restriction could be detrimental to dogs with early cardiac disease. Recommendations for sodium are summarised in **Table 1**.

Figure 2. Breeds at risk of congestive heart failure include cavalier King Charles spaniels.

For dogs with heart disease, or breeds at risk of heart disease (**Figure 2**), the clinician should discuss with the owner the sodium content of treats and human food, especially if these are being used to administer medications.

Foods such as pizza, lunch meats, sausages, bacon, most cheeses and most processed foods are high in sodium.

Potassium

Angiotensin converting enzyme (ACE) inhibitor therapy has gained widespread use in the management of dogs with CHF. These drugs can cause increased serum potassium and some animals develop hyperkalaemia.

Spironolactone, now used in some dogs and cats with heart disease, is an aldosterone antagonist and a potassium-sparing diuretic. Animals receiving ACE inhibitors or spironolactone can develop hyperkalaemia.

Some commercial cardiac diets contain increased potassium concentrations to counteract the theoretical potassium loss due to diuretics, so can contribute to hyperkalaemia.

Nutritional deficiencies

Deficiencies – for example, deficient thiamine, magnesium, vitamin E, selenium and taurine – may cause or complicate cardiac disease.

These are unlikely to be the primary or only cause of cardiac disease, other than in some predisposed breeds or in pets on unbalanced diets.

Protein and amino acids

Protein

Recommendations for restricting the dietary protein intake of animals with CHF were once common. The thinking behind this was to reduce the “metabolic stress” on the kidneys and liver.

There is no evidence to show protein restriction is necessary for dogs and cats with CHF. In fact, it probably is deleterious since these patients are predisposed to loss of lean body mass.

Unfortunately, many people recommend a diet designed for renal disease for animals with heart disease, because many renal diets are restricted in sodium. Protein is also restricted, even in some

diets designed specifically for pets with cardiac disease. Unless severe renal dysfunction is present, high-quality protein should be fed to meet maintenance requirements.

Taurine

There has been a dramatic reduction in cases of feline dilated cardiomyopathy (DCM) since the late 1980s when increased dietary supplementation of taurine was instituted, after the landmark research of Pion et al (1987).

While most current cases of feline DCM are not taurine deficient, this should be ruled out in all cases.

Cats fed a home-made, vegetarian, poor quality or otherwise unbalanced diet are at risk of taurine deficiency.

Taurine deficiency is now suspected in some cases of canine DCM. Unlike cats, dogs are able to synthesise adequate amounts of taurine, so are thought not to require dietary sources.

While most dogs with DCM do not have taurine deficiency, low taurine concentrations have been found in some cases – mostly in American cocker spaniels, golden retrievers, Labrador retrievers, Newfoundlands, Dalmatians, Portuguese water dogs and bulldogs.

Taurine deficiency in dogs may be related to dietary factors, as it appears to be more common in dogs eating high-fibre diets or some lamb and rice-based diets. It has also been induced by long-term low-protein, low-taurine diets.

Further, taurine deficiency may be the result of increased renal or faecal taurine loss or metabolic defects present in certain breeds.

Taurine supplementation may be beneficial in some deficient dogs but, even in animals that respond, the response generally is not as dramatic as in taurine-deficient cats with DCM.

The exact role of taurine in canine DCM is unclear and some of the potential benefits may be due to its positive inotropic effects or role in calcium regulation in the myocardium.

The optimal dose for correcting a deficiency is not known, but the recommendation is 500mg to 1,000mg every 8 hours to 12 hours for dogs and 250mg PO every 12 hours to 24 hours for cats.

Because taurine is safe and inexpensive, it is recommended for use in any case of myocardial failure.

Arginine

Nitric oxide is an endogenous vascular smooth muscle relaxant. It is synthesised from L-arginine and molecular oxygen and is catalysed by the nitric oxide synthase (NOS) enzyme.

There are three forms of NOS: inducible NOS (iNOS), endothelial NOS (eNOS) and neuronal NOS (nNOS).

eNOS and nNOS are constitutive forms and always produced in low levels. eNOS is also required for maintenance of normal vascular tone and as a physiologic messenger.

iNOS, on the other hand, is inducible by a variety of inflammatory mediators including the cytokines TNF and IL-1, and free radicals.

High levels of iNOS are induced as a mediator of the inflammatory response and in the host defence mechanism.

Circulating nitric oxide is elevated in dogs and cats with CHF. However, while iNOS is up-regulated in patients with CHF producing high circulating levels, eNOS is down-regulated – thus reducing endothelium-dependent vasodilation.

Arginine supplementation improves endothelial dysfunction in people with CHF, with no negative effects on cardiac contractility or other echocardiographic variables.

Fat and n-3 polyunsaturated fatty acids

Fat provides calories and increases the palatability of pet foods, but can also significantly affect immunological, inflammatory and haemodynamic parameters.

The n-3 polyunsaturated fatty acids, eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) are normally in low concentrations in the plasma membrane, but levels can be increased by a food or supplement enriched in n-3 fatty acids.

Dogs with CHF have been shown to have plasma fatty acid abnormalities, including decreased concentrations of EPA and DHA compared to normal dogs. Fish oil supplementation can normalise these plasma fatty acid abnormalities (Freeman et al, 1998).

There are a number of potential benefits of n-3 fatty acids supplementation. The eicosanoids derived from n-3 fatty acids are less potent inflammatory mediators than those from n-6 fatty acids, and n-3 fatty acids decrease production of TNF and IL-1. In addition, n-3 fatty acids have anti-arrhythmic properties.

While optimal doses are not known, 40mg/kg EPA and 25mg/kg DHA has been safely used for dogs with cardiac cachexia.

B vitamins

Thiamine deficiency can cause cardiomyopathy in people, but there has been little investigation into the role of B vitamin deficiencies as a cause of heart disease in dogs and cats.

Anorexia and urinary loss of water-soluble vitamins can contribute to low B vitamin concentrations in patients with heart failure.

In a human study, more than 90 per cent of patients with CHF had low thiamine concentrations (Seligmann et al, 1991).
In a study of cats with cardiomyopathy, vitamin B_{12} – but not vitamins B_6 or folate – was significantly lower compared to healthy controls. This effect was unrelated to diet or furosemide use (McMichael et al, 2000).

Magnesium

As magnesium plays an important role in normal cardiac function, hypomagnesaemia can play an important role in many cardiovascular conditions, including hypertension, coronary artery disease, congestive heart failure and cardiac arrhythmias.

As some cardiac drugs are associated with magnesium depletion, animals with CHF can be at increased risk.

Hypomagnesaemia can increase the risk of arrhythmias, decrease cardiac contractility, cause muscle weakness, contribute to renal potassium loss and potentiate the adverse effects of certain cardiac medications.

Hypomagnesaemia has not been consistently found in animals with heart disease. However, this may be because serum magnesium concentrations are a poor indicator of total body stores.

Other nutrients

L-carnitine

L-carnitine is critical for fatty acid metabolism and energy production and is concentrated in skeletal and cardiac muscle.

Carnitine deficiency is associated with primary myocardial disease in a number of species, including a family of boxer dogs. Some boxers and American cocker spaniels with DCM respond to carnitine supplementation (Keene et al, 1991; Costa and Labuc, 1994; Kittleson et al, 1997).

Further anecdotal reports exist regarding the efficacy of carnitine in canine DCM (Sanderson,

2001).

One study of dogs with heart failure induced by rapid pacing showed myocardial concentrations of carnitine decreased and plasma concentrations increased (Pierpont et al, 1993).

Damage to the heart cells from the pacing may have caused the carnitine to leak from the myocardiocytes.

Even if carnitine deficiency is not the inciting cause of DCM, L-carnitine supplementation could be beneficial by improving myocardial energy production. It is especially recommended for boxers and cocker spaniels with DCM.

The recommended canine dose is 50mg/kg to 100mg/kg PO every eight hours.

Coenzyme Q10

Like carnitine, coenzyme Q10 is a cofactor in many reactions required for energy production. It is also an antioxidant.

Many anecdotal reports claim benefits of supplementation, but controlled prospective studies will be 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.

Reasons 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 by-product of oxygen metabolism and are, typically, adequately compensated for through the production of endogenous antioxidants.

Antioxidants are produced endogenously, but can be supplied exogenously with enzymatic antioxidants (for example, superoxide dismutase, catalase and glutathione peroxidase) or oxidant quenchers (for example, vitamin C, vitamin E, glutathione and ?-carotene).

However, some investigators claim an imbalance between oxidant production and antioxidant protection could increase the risk for certain types of heart disease.

Also, dogs with heart failure have increased levels of biomarkers of oxidative stress and a reduction in some antioxidants, particularly vitamin E (Freeman et al, 1999).

Commercial diets for cardiac disease

A number of commercial veterinary diets are available specifically for animals with cardiac disease.

Specific characteristics of these foods vary, but they usually are mildly to severely restricted in sodium and generally contain increased levels of B vitamins. Some cardiac diets may also be enriched with other nutrients. The choice should consider the level of sodium restriction desired for a specific patient.

General diet issues

When selecting a diet for a patient with cardiac disease, it is important to find one with the desired nutritional properties for the animal.

Many dogs with heart disease have concurrent diseases that could affect dietary selection. The presence of partial or complete anorexia, which occurs in many animals with CHF, can dramatically alter diet choice.

It is important to devise a dietary plan that meets the owner's expectations. This includes finding a diet the owner perceives the pet to enjoy, providing acceptable treats and devising a satisfactory method for administering medications.

In one study, more than 60 per cent of dogs with heart disease received treats often high in sodium (Freeman et al, 2002).
In addition, most people administering medications to their dogs use foods as a way to administer them.
Therefore, recommending appropriate treats and methods of administering medications is an important part of a successful nutritional programme.

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