**Nutritional Considerations**

Important metabolic changes may occur in animals with heart failure. Upregulation of the RAAS leads to increased plasma volume, largely mediated by increased sodium retention. Increased production of inflammatory cytokines such as tumor necrosis factor and interleukin 1 may promote increased metabolic demand and contribute to anorexia, thus worsening “cardiac cachexia.” Studies in people and a recent one in dogs have shown that patients with CHF who lost weight during the course of these studies had a poorer prognosis. In some patients, nutrient deficiencies (taurine, carnitine, coenzyme Q10) have been associated with decreased myocardial performance. Decreased levels of circulating fatty acids have been documented in people and dogs with heart failure. The overall nutritional goals in the management of animals with heart failure should therefore include supplying adequate calories, modulating the production of proinflammatory cytokines, managing sodium balance, and supplementing nutrients that may be deficient.

The idea that **sodium restriction** reduces circulating plasma volume and thus preload is well established. However, sodium restriction is known to activate the RAAS, and there is some debate as to the role of sodium restriction in animals with asymptomatic cardiac disease or mild or moderate CHF. In contrast, there is almost universal acceptance that moderate to severe sodium restriction is indicated in animals with advanced CHF. Only mild sodium restriction (<80–90 mg/100 kcal) is recommended in patients with moderate to severe cardiac remodeling not yet in CHF (International Small Animal Cardiac Health Council [ISACHC] Class IB). It is also important to counsel owners to avoid foods and treats with high sodium content, because an acutely high sodium load (as can occur in animals fed human snacks or table foods) may precipitate CHF in animals with compensated heart disease. For animals in mild to moderate heart failure (ISACHC Class II), moderate sodium restriction (50–80 mg/100 kcal) is recommended. In patients with severe refractory CHF, more aggressive sodium restriction (<50 mg/100 kcal) is recommended. This becomes a therapeutic challenge with some animals in which cardiac cachexia is now present, because lower sodium foods can be less palatable.

Supplementation with **n-3 fatty acids** has shown multiple benefits in people with CHF, and a recent study in dogs suggests antiarrhythmic benefits as well. These fatty acids may reduce circulating inflammatory cytokine levels and seem to improve appetite in some dogs with cardiac cachexia. Daily doses of 40 mg/kg EPA and 25 mg/kg DHA are recommended.

**Taurine** supplementation is indicated in animals with documented taurine deficiency and DCM. The incidence of DCM has declined dramatically in cats since taurine deficiency was identified as a primary cause in the late 1980s. Taurine deficiency is still documented in some cats with DCM fed noncommercial diets. Supplementation at 250 mg, sid-bid can be started in cats while awaiting results of plasma and whole blood taurine levels. Dogs are able to synthesize endogenous taurine, so deficiency is less common in this species. However, breeds such as the American Cocker Spaniel, Golden Retriever, and Newfoundland are relatively predisposed to taurine deficiency, especially when fed lamb and rice, or high-fiber low-protein/taurine diets. Whole blood and plasma taurine levels should be obtained in any dog suspected of having a taurine-deficient cardiomyopathy, and supplementation can be started at 500–1,000 mg, PO, bid-tid while awaiting results.

**l-Carnitine** plays an important role in fatty acid metabolism and energy production. Carnitine deficiency has been documented in a family of Boxers, and carnitine has been supplemented in other breeds with DCM with some anecdotal success. It is unclear whether this deficiency is a cause, or a result of cardiomyopathy in these dogs. Diagnosis of carnitine deficiency is difficult and requires an endomyocardial biopsy. Supplementation is also expensive, and given our limited knowledge of the role carnitine plays in canine cardiomyopathy, supplementation is not routinely recommended. Nevertheless, supplementation can be offered at 50–100 mg/kg, PO, bid-tid to dogs with DCM, especially Boxers.

**Coenzyme Q**10 is involved with mitochondrial energy production and possesses general antioxidant properties. Anecdotal benefits of supplementation in people and dogs with DCM have been reported, but well-controlled studies are lacking, and some reports are conflicting. The current recommended dosage in dogs is 30–90 mg, PO, bid.

Source: <http://www.merckmanuals.com/vet/circulatory_system/heart_disease_and_heart_failure/heart_failure.html>