

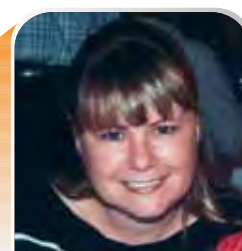


Nutritional management of early cardiac disease: ACT with SPEED



Daniel Baker, DVM
Scientific Communications, Royal Canin USA

Dr. Baker graduated from the University of Massachusetts-Amherst with a Bachelor of Science degree in biology with Honors in 1999. As an undergraduate he was one of the eighteen members from around the world who took part in the East/West marine biology program at Northeastern University, allowing students to compare and contrast marine flora and fauna in three distinct parts of the world. Daniel received his DVM degree from Ross University in 2003 having completed his clinical year at the University of Minnesota. Following graduation he worked as a small animal clinician for 4 years. During this time he focused mainly in the area of emergency and critical based medicine. Daniel is currently a member of the Scientific Communications team for Royal Canin USA.



**Denise Elliott, BVSc (Hons), PhD,
Dipl. ACVIM, Dipl. ACVN**
Scientific Affairs, Royal Canin USA

Dr. Elliott received her degree in Veterinary medicine with honors from the University of Melbourne in 1991.

After completing an internship in Small Animal Medicine and Surgery at the University of Pennsylvania, Denise completed a residency in Small Animal Internal Medicine and Clinical Nutrition at the University of California-Davis. Denise became a Diplomate of the American College of Internal Veterinary Medicine in 1996 and the American College of Veterinary Nutrition in 2001. She received her PhD in Nutrition from the University of California-Davis in 2001, for her work on multifrequency bioelectrical impedance analysis in healthy cats and dogs. Denise is currently the Director of Scientific Affairs for Royal Canin USA.

Accurate and timely diagnosis of early stage (*Class 1 & 2, Table 1*) cardiac disease historically has been difficult. Most patients show no outward evidence of being sick. Owners themselves usually do not report clinical signs related to a cardiac abnormality until later stages occur. Typical treatment of early stage cardiac disease has largely focused on benign neglect with subsequent monitoring of the patients cardiac rate, rhythm, size and associated clinical signs at six to twelve month intervals. The question that must be asked is a topic of much debate within the veterinary community. "If a patient shows no outward clinical signs

associated with a disease, such as early cardiac disease, what if anything should be done clinically to aid in the improvement of the animal's health?"

Through the collaboration with some of the worlds leading cardiologists and nutritionists in conjunction with the innovative research performed at Royal Canin and the WALTHAM Centre for Pet Nutrition, we now realize the beneficial effects of implementing early nutritional support in patients faced with cardiac abnormalities. Nutrition by itself can have profound beneficial effects at minimizing metabolic disturbances, while at the same time improving the patients over all quality of life.

Table 1.
Classification of heart disease

Grade	Clinical description
Class I	No limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, or dyspnea.
Class II	Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in fatigue, palpitation, or dyspnea.
Class III	Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity causes fatigue, palpitation, or dyspnea.
Class IV	Unable to carry out any physical activity without discomfort. Symptoms of cardiac insufficiency at rest. If any physical activity is undertaken, discomfort is increased.

Cardiac disease is now the second leading cause of death in dogs. Chronic valvular disease (CVD) accounts for approximately 75% of these cases while dilated cardiomyopathy (DCM) is responsible for 10-15% of cases observed. Over the past decade more advanced diagnostic tools such as: electrocardiography (ECG), Holter monitoring and echocardiography are now available to the general practitioner. This allows for early diagnosis and subsequent intervention of early cardiac disease.

Nutrition's primary role in the prevention and treatment of cardiac disease is multimodal. Through the use of key nutrients, dietary intervention seeks to provide optimal amounts of energy, minimize oxidative stress, reduce inflammation, balance electrolytes and ultimately to improve cardiac performance. Through the use of the ACT with SPEED acronym we can better understand each key nutrients role in slowing the progression of heart disease.

ACT

Arginine, an essential amino acid, is the precursor for endogenously synthesized nitric oxide. Nitric oxide is well known for its role as an endothelium-derived relaxation factor, which in turn is responsible for maintaining normal vascular tone (1). Endothelial dysfunction has been linked, in humans and dogs, with congestive heart failure

(CHF) (2). Arginine supplementation appears to improve cardiac output in patients with cardiac disease by positively influencing preload and afterload ($CO = HR \times \text{contractility} \times \text{preload/afterload}$).

Carnitine, a quaternary amine, is composed of two essential amino acids, lysine and methionine. It is commonly found concentrated in both skeletal and cardiac muscle. L-carnitine (the biologically active form) is critical for oxidation of fatty acids within mitochondria. It serves as a transport shuttle of fatty acids from outside the mitochondria into the inner membrane and in turn is a key component in the regulation of the citric acid cycle (3). L-carnitine is also responsible for transporting metabolic waste products out of the mitochondria that might otherwise become cardiotoxic. L-carnitine is normally provided to the animal in sufficient amounts through intestinal absorption or following hepatic and renal synthesis. In certain breeds (Boxers, Doberman Pinschers and American Cocker Spaniels) there have been reported myocardial carnitine deficiencies (4,5). In a great many of these cases plasma carnitine levels were within normal limits. These findings suggest a membrane transport defect may be present preventing L-carnitine from entering into the myocardial cells from the plasma. Providing optimal amounts of carnitine in the diet may improve overall myocardial function.

Taurine, a non-essential amino acid in the dog, is well known for its powerful antioxidant effects throughout the body. Taurine also is a key nutrient in the treatment of certain cardiomyopathies (6-8). Recent evidence also suggests that feeding certain lamb-based diets and severely protein restricted diets, may lead to marked taurine deficiencies with associated clinical signs (9-10). Breed associated deficiencies (e.g. American Cocker Spaniel, Portuguese Water dog) have manifested themselves in the form of dilated cardiomyopathy. Clinical improvement has resulted in several cases where the proper taurine levels were present within the diet (5-11). Taurine has also proven to have positive inotropic effects in animals with experimentally induced heart failure (12). This suggests taurine supplementation may prove beneficial in patients with cardiac disease without true taurine deficiencies.

ROYAL CANIN VIEWPOINT

With SPEED

Sodium restriction has long been thought of as the nutritional mainstay of treating cardiac disease. While sodium restriction has its place adjunctively along side additional nutritional modalities, it is the degree of sodium restriction based on the severity of cardiac disease present that must be taken into account. Indeed, sodium restriction that is too severe early on in the disease process may lend itself to an exaggerated response by the renin-angiotensin aldosterone system (13-18). This can exacerbate clinical signs and the progression of the disease itself. The advent of angiotensin converting enzyme (ACE) inhibitors has further decreased the need for severe sodium restriction in most patients (19). Based on our current understanding of sodium's relationship to cardiac physiology during certain stages of cardiac disease, tailoring the degree of sodium restriction is of the utmost importance.

Protein restriction has erroneously worked its way into the management of cardiac disease. Many of today's diets are formulated on the antiquated thought process that protein restriction lends itself to reduced metabolic stress on the kidneys and liver (20). No known peer reviewed publications exist proving this hypothesis. What's worse is that protein restricted diets favor the development of cardiac cachexia. This in turn will lead to the perception by the owner of a poor

quality of life and likely hasten euthanasia. Cardiac diets should contain optimal amounts of highly digestible proteins that allow for the preservation of lean body mass.

Energy requirements in patients with cardiac disease should be considered with both the patients' body condition score (BCS) and degree of cardiac cachexia (**Table 2**) in mind. The goal should be to manage caloric intake to prevent both obesity and emaciation while preserving lean body mass. Special focus needs to be placed on patients with poor BCS and/or cachexia already prevalent. Poor doing is often associated with anorexia in patients with cardiac disease. A recent study revealed the daily caloric intake in dogs with dilated cardiomyopathy was 72-84% of their expected daily energy requirements (21). Nutrient profiles therefore need be formulated with nutrients that are highly digestible and readily bioavailable.

Eicosapentaenoic acid (EPA)

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Docosahexaenoic acid (DHA) are essential long chain fatty acids derived from marine sources that provide profound anti-inflammatory effects throughout the body but especially within the heart. Studies have revealed that dogs with congestive heart failure have decreased concentrations of plasma EPA/DHA in comparison with healthy dogs (21). Their combined effects target the reduction of pro-inflammatory mediators actions on the cardiac infrastructure (21). Supplementation with marine derived omega-3 fatty acids have also been shown to improve cardiac cachexia scores in dogs with cardiac disease. A recent study revealed that EPA/DHA from fish oils given over a six-week period may decrease the severity and frequency of arrhythmias in Boxers who suffer from Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC) (22).

Several other key nutrients related to the heart should be taken into consideration. For example, vitamin E has positive effects on inhibiting lipid peroxidation of the cellular membranes of cardiac cells. Vitamin E can be looked at as a biomarker of oxidative stress. In patients with heart disease, low

Table 2.
Cachexia scoring system

Cachexia score	Description
0	Good muscle tone with no evidence of muscle wasting
1	Early, mild muscle wasting, especially in the hindquarters and lumbar region
2	Moderate muscle wasting apparent in all muscle groups
3	Marked muscle wasting as evidenced by atrophy of all muscle groups
4	Severe muscle wasting

*Modified from Freeman (29).

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Vitamin E concentrations have been negatively correlated with the severity of the disease (23). Vitamin B deficiencies have been reported in cats with cardiomyopathy (24). Hypomagnesemia may potentiate cardiac arrhythmias, decrease myocardial contractility and contribute to muscle weakness (25,26). This is often observed in the Cavalier King Charles Spaniel (27). Potassium, traditionally hyper-supplemented in historic cardiac diet formulations to compensate for the urine wasting secondary to diuretic therapy, is no longer necessary. With the advent of ACE inhibitor therapy that increases the renal absorption of potassium, modern cardiac diets should contain normal levels of potassium (28).

In summary, we must first identify early stage cardiac disease in our patients. Then we need to educate pet owner's that early cardiac disease left untreated may have drastic life long complications. Many of these consequences stem from a sub-clinical disease process not necessarily manifesting itself as outward clinical signs. As clinicians, we now have the opportunity to offer our clients early dietary therapeutic intervention in conjunction with close monitoring of their pet's disease state. Together we can give owners and their beloved pets, the nutritional tools necessary to aid in the management of heart disease and to improve the chance of living longer, healthier lives.

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