FELINE HYPERTROPHIC CARDIOMYOPATHY AND THE ROLE OF NUTRITION

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Heart disease is a common cause of illness in domestic cats. Studies show ~ 20% of apparently healthy cats examined at random were found to have cardiac murmurs.[1,2] Prevalence data shows that up to 20% of feline populations are affected by cardiac disease.[3,4] Cardiomyopathies are the principal cause of cardiovascular morbidity and mortality in cats, and hypertrophic cardiomyopathy is the most common of these disorders.[5] Although the majority of affected cats are assumed to remain free of clinical signs, a proportion experiences serious complications, chief among which are congestive heart failure (CHF), arterial thromboembolism (ATE), and sudden cardiac death (SD).[5] Cats tend to hide pain and illness and thus owners do not recognize early signs of disease, thus heart disease in cats is known as the 'silent killer'. While other species may exhibit early signs such as coughing or exercise intolerance, cats often present without signs, until the disease has become more advanced.

Many types of heart disease exist in cats, but the most common are cardiomyopathies.[6] Other types of heart disease may include valve disease, birth defects, or other conditions. The most common heart disease in cats is Hypertrophic Cardiomyopathy (HCM). HCM is characterized by a gradual thickening and weakening of the heart muscle. Left ventricular hypertrophy, impaired diastolic filling, and often secondary left atrial enlargement result. As the muscle thickens, there is less room for blood to fill the heart and the muscle is less able to pump blood effectively as it weakens. There are two primary forms of HCM – a genetically linked form that is often early onset and occurs in some breeds (Maine Coons, Ragdolls, Rexes, Sphynxes, and others) and a sporadic form that may occur in any cat at any age.

In cats with HCM, CHF and ATE are common clinical manifestations, with ATE reportedly developing in up to 48% of affected cats.[7,8] The most common site of ATE in cats is the terminal aorta, resulting in hind limb paresis/paralysis. Other limbs and sites can be affected, but this occurs less frequently. Additional clinical manifestations of HCM in cats include syncope, arrhythmias, and sudden death. The median survival time for cats with HCM is less than 2 years, with cats presenting for CHF or ATE having even shorter survival times.[7,8] Disease-causing mutations have not been identified in most cats with HCM, except for the myosin binding protein C mutation in Maine Coon and Ragdoll cats.[7,9,10] At this time, management of cats with HCM is limited to a combination of medication and diet.

Dilated Cardiomyopathy (DCM) was once a common disease in cats. DCM is characterized by a thin and "flabby" heart muscle. It was discovered that DCM was almost exclusively the result of a deficiency of taurine in the diet. Following the discovery that taurine deficiency was the principal cause of dilated cardiomyopathy in cats in 1987, the prevalence of this disease has decreased significantly. [10-12]

Since this time, commercial foods have been supplemented with taurine, rendering the disease less common. DCM may still occur for other reasons, or in cats fed homemade or poorly formulated diets deficient in taurine. It is important to note that taurine supplementation is likely not helpful in cats with other forms of heart disease.

SIGNS

Early signs of heart disease in cats are easy to miss as they are either non-existent, or so subtle and non-specific that they are rarely noticed by owners. In addition, cats appear to know their own capabilities and limitations and thus, restrict their level of activity, which can further mask clinical signs.

Some cats may show signs of congestive heart failure, including labored or rapid breathing, open-mouthed breathing, blue tinged gums, and lethargy. These symptoms occur when fluid accumulates in or around the lungs.

A serious and life-threatening consequence of HCM is the formation of blood clots in the heart. These clots may travel through the bloodstream and result in a thromboembolism. The effect of the clot depends on its location. In cats with HCM, clots most commonly result in blockage of blood flow to the hind limbs, causing acute hind limb pain or hind

limb paralysis. Diagnosing HCM and managing the condition may help decrease the severity of clinical signs and the possibility of thromboembolism.

Cats with FATE generally lose the function of one or more limbs. Initially, the embolism is very painful, with loss of pain sensation occurring over the next few hours. The affected limbs are cold to the touch and without pulses in the arteries to the affected leg(s), and the limbs are weak or paralyzed. This is a very serious condition and is an immediate medical emergency for which prompt therapy is essential. While FATE typically affects the limbs, cats may also have clots to their brain, lungs, heart, gut, or in other locations.[6]

Although FATE results in dramatic and disturbing clinical signs, treatment for at least 48 to 72 hours should be suggested strongly to the owner, as within this time frame some cats will remain stable or improve, whereas others may worsen.[13] The goals in managing acute FATE are:

1. reduction in continued thrombus formation associated with the embolus
2. improvement in arterial blood flow (either aortic or collateral)
3. pain management
4. treatment of concurrent CHF
5. supportive care.

RISK

HCM has particular breed predispositions, and at least one identified genetic cause in each of two breeds – Maine Coons and Ragdolls.[14,15] The following breeds are considered to be predisposed to cardiomyopathies: Sphynx, Norwegian Forest Cats, American Shorthairs, Scottish Folds, Persians, Siamese, Abyssinians, Himalayans, and Birmans.[16] Whether there are any gender differences in expression of genetic traits or in prevalence of congenital disorders is not well defined.

Risk factors for causing or complicating cardiovascular disease include breed, gender, obesity, renal disease, drug therapy, endocrinopathies. Obesity occurs frequently in dogs and cats with cardiovascular disease. Obesity not only produces clinical signs that mimic those of early heart failure (i.e., exercise intolerance, tachypnea, weakness), but may also result in cardiovascular changes that can exacerbate underlying cardiovascular disease.[12] Obesity has potentially profound cardiovascular consequences. From a cardiovascular perspective, obesity is a disease of blood volume expansion with:[12] 1) elevated cardiac output, 2) increased plasma and extracellular fluid volume, 3) increased neurohumoral activation, 4) reduced urinary sodium and water excretion, 5) increased heart rate, 6) abnormal systolic and diastolic ventricular function, 7) exercise intolerance and 8) variable blood pressure response.

Chronic progressive kidney disease and failure often occur in cats with cardiovascular disease - especially older cats. Cardiac disease often intensifies underlying renal disease as a large proportion of the cardiac output is normally destined for the kidneys. Renal disease influences the types and dosages of medications that are used to treat patients with cardiovascular disease. Chronic kidney disease is also a risk factor for secondary hypertension in cats. Hyperthyroidism is a risk factor for hypertrophic cardiomyopathy and secondary hypertension in older cats.

DIAGNOSIS

The veterinary healthcare team should take an in depth and thorough history along with a tip to tail physical exam, as these form the basis of diagnosis of heart disease. The examination will include listening carefully to the heart and lungs and checking for normal pulses. However, it is important for the veterinary team to remember that many cats with cardiac disease have no indicative clinical signs or physical examination findings.

Chest radiographs are often recommended as part of the diagnostic workup. The radiographs may show heart disease definitively, but more often are used as one part of a multistep diagnostic process. Identification of cardiomegaly from radiographs in cats is difficult. Left ventricular concentric hypertrophy, as occurs with HCM, is not identified by

radiographs. It is important to note that cats can have profoundly thickened left ventricular walls that are not detectable via radiographs.

Blood tests may be needed to rule out other conditions. Some specific tests (such as nt-pro-BNP) may be useful in determining if heart disease is present. Biochemical indicators of heart disease include cardiac troponin I; atrial natriuretic peptide (ANP) and its prohormone, NT-proANP; and B-type natriuretic peptide (BNP) and its prohormone, NT-proBNP.[1,6] These are proteins either secreted or released by cardiomyocytes in response to stretch or injury and can be measured in serum or plasma.

In cats, electrocardiography (ECG) is ineffective as a screening tool for occult cardiac disease. The foundation for using ECG as a screening tool relies on its ability to reveal either chamber enlargement or shifts in the mean electrical axis (MEA). Consequently, studies show that the sensitivity of ECGs in detecting morphologic changes consistent with chamber enlargement or HCM is at best 20%.[1,17]

The best way to determine the type and extent of heart disease is with echocardiography. An echocardiograph allows images and videos of the heart to be collected and analyzed by the veterinarian and/or veterinary cardiologist.

MANAGEMENT AND TREATMENT

Heart disease in cats is seldom curable with management focused on medical and nutritional therapy. There is little proof that therapy before the onset of CHF is effective at slowing the progression of disease in most conditions, though some potential therapies exist. Cats with CHF may require oxygen therapy, chest taps, and in some individual patient's more aggressive care. Cats with blood clots (FATE) will require pain control medication, anticoagulants, and aggressive hospital care.[6]

Cats with CHF will almost always be treated with a diuretic to keep fluid off the lungs. They may also be treated with beta blockers to slow the heart rate and reduce certain symptoms, ACE-inhibitors to help decrease fluid buildup, blood thinners to prevent clots, or one of several other medications based on veterinary recommendations.

The prognosis for FATE is much more guarded, especially if severe signs are present. Many cats with saddle thrombosis do not survive hospitalization, while others may recover fully. Treatment with blood thinners such as clopidogrel is commonly lifelong.

Nutritional Assessment

The veterinary team should not wait until later stages of cardiac disease to begin nutritional management. Rather, we should optimize nutritional therapy early; thus, benefitting from nutritional therapy during the longer, and more common, earlier stages of cardiac disease. Adjustment of certain nutrients may be beneficial in asymptomatic disease. In addition, optimizing body composition, preventing deficient or excessive intake of various nutrients, and avoiding nutritionally unbalanced diets may help to slow progression of disease and improve quality of life. Nutrition should be considered an integral part of the overall care for *all* stages of heart disease, from cats with a predisposition for heart disease, to cats with asymptomatic heart disease, to cats with CHF.

A nutritional assessment aids in determining if specific modifications might be beneficial for a cat with heart disease. A nutritional assessment includes an assessment of the cat and its current diet. Nutritional assessment consists of a nutritional history, body weight, body condition score (BCS), and muscle condition score (MCS).

It is important for the veterinary team member to ask open ended questions to facilitate discussion. In addition to what the cat is being fed, we also what to ascertain if any supplements are being given. Cats with heart disease are more likely to be receiving dietary supplements than cats in the general population.[18,19] Also, the giving of snacks, treats, and table food should be investigated. Also, asking owners about how they administer medications can reveal issues not suspected by the owner (e.g., high-sodium foods like cheese or lunch meats being used to hide medications).

Changes in body composition are common and important concerns in cats with heart disease or heart failure and can negatively affect the outcome and quality of life.

BCS should be performed at least weekly and can be done by the owner and reported into the veterinary team. Ensure the BCS method is consistent so as to assess changes over time.

The muscle condition score (MCS) evaluates muscle mass. Cats can be very obese and yet have severe muscle loss, and conversely, cats can be thin but have normal muscle mass. Evaluation of muscle mass includes visual examination and palpation over the temporal bones, scapulae, lumbar vertebrae, and pelvic bones. Assessing MCS is important because cats with heart disease lose primarily muscle. Early identification of muscle loss is beneficial for successful intervention.

Reductions in food intake may indicate the need for dietary modifications but also may be an early sign of decompensation of the cardiac disease or the need for medication adjustment. Body weight, BCS, and MCS should be monitored carefully at every visit and addressed if not optimal. Also, it is important to reassess the diet to ensure that it remains optimal for the cat's stage of disease, laboratory values, and clinical signs. Owners often change the diet (e.g., the cat food, treats, and/or table food), add supplements, or use different methods for administering medications. Therefore, just because the diet was appropriate at a previous visit does not mean that it is still optimal.

Cachexia

Cachexia is a multifactorial syndrome characterized by severe, chronic, and progressive weight loss and muscle wasting, oftentimes accompanied by anorexia. Cachexia is a complex syndrome characterized by severe, chronic, undesired, and progressive weight loss and muscle wasting, with or without loss of fat mass.[20] This syndrome is associated with an underlying disease, anorexia, inflammation, insulin resistance, and increased lean muscle breakdown.

In patients with cardiac disease, anorexia may be a result of the following:

- clinical signs of heart failure
- the presence of concomitant disease
- use of medications that cause nausea
- the presence of elevated levels of inflammatory cytokines
- sudden nutritional changes.

The rate of loss of lean body mass with cardiac cachexia exceeds that attributable to anorexia alone and indicates the excessive caloric expenditures of the increased work of respiration and elevated heart rate. Cachexia involves depletion of lean body mass.[12] Physical inactivity due to exercise restriction in cardiac patients may also contribute to loss of lean body mass.

Nutritional Management Overview

For cats with HCM, the dietary management depends upon the stage of disease. For cats with HCM but no clinical signs, it is an ideal time to begin talking to the owner about the animal’s overall dietary patterns (i.e., the pet food, treats, table food, and medication administration) and achieving ideal body weight/body condition, as it is easier to institute dietary modifications before clinical signs have arisen. Mild sodium restriction (<100 mg/100 kcal) is recommended. At this point, severe sodium restriction (<50 mg/100 kcal) is not recommended since severe restriction may result in early and prolonged activation of the renin-angiotensinaldosterone (RAA) system. When cats with HCM develop mild-moderate CHF, moderate sodium restriction (i.e., <80 mg Na/100 kcal) is indicated along with medical therapy.[7] Anorexia becomes more of a problem in cats with CHF. Anorexia may be the result of side effects from medications so the veterinary team must carefully monitor drug doses and the cat’s serum biochemistry profile for alterations in BUN, creatinine, and electrolytes. Providing a more palatable diet can help to improve appetite. Supplementation with fish oil, which is high in n-3 fatty acids, can decrease inflammatory cytokine production and improve appetite in some cats with CHF. Anorexia can contribute to B vitamin deficiencies, as can increased urinary losses of water soluble vitamins due to diuretic use. Research has shown that plasma concentrations of vitamins B6, B12, and folate were significantly lower in cats with cardiomyopathy than in healthy controls, an effect that was unrelated to diet or furosemide use.[21,22] Therefore, animals with cardiac disease (at least those receiving diuretics) may have higher B vitamin requirements. Although most commercial feline diets contain relatively high levels of water

soluble vitamins, B vitamin supplementation may be useful for cats with CHF, particularly those receiving large doses of diuretics. In severe CHF, greater restriction of dietary sodium (<50 mg) may allow lower dosages of diuretics to be used to control clinical signs; however, it is critical that adequate food intake is maintained.[7] Therefore, compromises often must be made between the "ideal diet" and one that the cat will eat; at this stage, anorexia can be a significant problem. Nonetheless, maintaining at least mild sodium restriction is recommended. In these situations, feeding tubes can be an important consideration to address calorie and other nutrient needs. There also is a higher risk for potassium and magnesium abnormalities in cats with severe CHF because of the high doses of diuretics usually required, so monitoring of electrolytes is important for optimal care.[7]

Key Nutrients in Managing HCM

Sodium and Chloride

Retention of sodium, chloride, and water is associated with CHF. Consequently, the healthcare team should focus on these nutrients in patients with cardiovascular disease. A few hours after the ingestion of high levels of sodium, healthy cats easily excrete any excess in their urine. Patients in early cardiac disease may lose this ability to excrete excess sodium. As heart disease worsens and CHF arises, the ability to excrete excess sodium is worsened. Historically, sodium retention was primarily implicated in the pathogenesis of CHF and some forms of hypertension. A number of studies have examined the interaction of sodium with other ions, including chloride. Chloride may also act as a direct renal vasoconstrictor. According to the NRC the minimum recommended allowance for sodium and chloride in foods for adult cats is 0.068% for sodium and 0.096% for chloride (DM).[23] When dealing with cats with cardiovascular disease the sodium levels in foods designed to manage these patients should be limited to 0.07 to 0.3% DM for cats. Recommended chloride levels are typically 1.5 times sodium levels.

Avoiding excess sodium chloride in cat foods is more difficult vs. dog foods as ingredients used to meet the higher protein requirement of cats also contain sodium and chloride and thus increase the sodium chloride content of cat food.

Taurine

Taurine is an important amino acid in cats with myocardial failure. The mechanism of heart failure in taurine-deficient cats is not well understood. Taurine may function in inactivation of free radicals, osmoregulation, and calcium modulation. Taurine is also known for its direct effects on contractile proteins. Additionally, there may be other factors responsible for contributing to the development of myocardial failure in patients with taurine deficiency. As we know, taurine is an essential amino acid in cats, therefore a minimum recommended allowance for taurine is necessary in cat foods. Taurine should be 0.04% DM.[23]Taurine content of foods for cats with cardiovascular disease should contain at least 0.3% DM. Levels of taurine typically recommended for supplementation of feline cardiovascular patients (250 to 500 mg taurine/day) provide approximately twice that much.

Phosphorus

Patients with cardiac disease are often suffering from concurrent disease conditions. It is understood that phosphorus is a nutrient of concern in patients with concurrent chronic kidney disease and that kidney disease is one of the more prevalent diseases seen concurrently with cardiac disease. Therefore, nutritional management should avoid excess phosphorus in patients with concurrent chronic kidney disease. The recommended amount of phosphorous in nutritional management of cardiac disease is 0.3 to 0.7% DM in cats.

Potassium and Magnesium

Another concern in cardiac disease patients is the metabolism of potassium and magnesium. Hypokalemia, hyperkalemia, and hypomagnesemia, all have the potential for complications when medication therapy is introduced in patients with cardiovascular disease. Veterinary team members should be aware that potassium or magnesium homeostasis abnormalities can:

- cause cardiac dysrhythmias
- decrease myocardial contractility
- produce profound muscle weakness
- potentiate adverse effects from cardiac glycosides and other cardiac drugs.

The amounts of potassium and magnesium recommended for adult maintenance in cats (0.52% DM potassium, and 0.04% DM magnesium) should be the minimum amounts included in nutritional management of CHF. If abnormalities in these electrolytes occur, the healthcare team should consider supplementation or switching to a different food.

Protein

Cardiac cachexia is a major concern in patients with cardiac disease. The protein requirements of patients with cardiac cachexia have not been investigated extensively to date. The metabolic changes associated with cachexia and their effect on overall nutrient requirements is only recently being investigated. Many patients with cachexia present with concomitant disease (i.e., chronic kidney disease), which also significantly affects nutrient requirements. Nutritionists do know that profound anorexia enhances protein-energy malnutrition in patients with cachexia. Subsequently, patients with cachexia or exhibiting signs potentially leading to cachexia should be encouraged to eat a complete and balanced food that contains adequate calories and adequate high-quality, highly digestible protein.

Omega-3 Fatty Acids

In cardiac cachexia, TNF and IL-1cytokines have been implicated as pathogenic mediators. Fish oil (known to be high in omega-3 (n-3) fatty acids) has been shown to alter cytokine production. Early investigations involving fish oil suggest that fish-oil-mediated alterations in cytokine production may help dogs with CHF. Consequently, it is believed that heart failure patients with cachexia may benefit from the alterations of cytokine production through omega-3 fatty acid supplementation.

It is believed that omega-3 fatty acids electrically stabilize heart cells through modulation of the fast voltage-dependent Na(+) currents and the L-type Ca(2+) channels which results in the heart cells becoming resistant to dysrhythmias. Clinical studies of fish oil as a source of long-chain omega-3 fatty acids have confirmed the reduction in frequency of ventricular arrhythmia in boxer dogs.

Omega- 3 fatty acids have been shown to have a significant effect on survival times when used in dogs diagnosed with DCM or chronic valvular disease. The effect of the omega-3 fatty acids may be attributed to: anti-inflammatory effects, cachexia prevention, improved appetite, or anti-arrhythmic effects. The veterinary healthcare team should also be aware of further effects of omega-3 fatty acids on the patient. The healthcare team must be mindful of the fact that omega-3 fatty acids have the potential to alter immune function. This alteration in immune function may contribute to the cardiovascular effects of omega-3 fatty acids. Also, omega-3 fatty acids reduce platelet aggregation resulting from the production of thromboxane B5. The reduction in platelet aggregation might be of benefit in cats with cardiac disease and at risk for thrombus formation. However, this effect is also important to be mindful of when using omega-3 fatty acids in animals with coagulopathies.

Additional studies and discussion is needed in the long term, but it is believed that dogs and cats with cardiac disease may benefit from omega-3 fatty acid supplementation. However, the healthcare team must consider a number of factors: 1) dose, 2) timing, and 3) omega-3 fatty acid form.

At this point in time, no optimal dose of omega-3 fatty acids has been established for humans, cats, or dogs. The current recommendation from nutritionists studying fatty acids and cardiac disease is a dose of 40 mg/kg EPA and 25 mg/kg DHA for both dogs and cats.[7, 23]

Timing also needs to be taken into consideration when supplementing omega-3 fatty acids. The healthcare team should remember and educate owners that the majority of omega-3 fatty acids benefits occur after peak plasma and tissue concentrations have been achieved. Although plasma concentrations may increase significantly in the first week of omega-3 fatty acid supplementation, typically 4 to 6 weeks are required to reach peak plasma concentrations.

EPA and DHA can be provided via the diet or as a dietary supplement. There are a few therapeutic pet foods with high levels of EPA and DHA, but the majority of foods manufactured today do not achieve the recommended level of EPA and DHA. The current recommended dose is 40 mg/kg EPA + 25 mg/kg DHA. Therefore, a manufactured food would need to contain between 80 and 150 mg/100 kcal EPA + DHA. Other factors that would need to be taken into consideration would be the size of the pet and the amount of food consumed. If the pet is not prescribed one of the high fatty acid foods, a recommendation of fish oil supplementation would be necessitated. However, caution must be given

when making a supplement recommendation, as fish oil supplements vary widely in the amount of EPA and DHA they contain. The healthcare team should be familiar with various brands of fish oil supplements and plan to make a recommendation based on a specific brand with which the concentration of EPA and DHA have been researched and confirmed.

Water

With all patients, veterinary team members must remember to talk with clients about the importance of water for pets. Veterinary nurses/technicians need to remind clients that pets should be offered water free choice and it should be clean and fresh. Healthcare teams must also keep in mind that water quality varies considerably, even within the same community. We must be cognizant of the fact that water can be a significant source of sodium, chloride, and other minerals. Veterinary healthcare teams should be familiar with the mineral levels in their local water supply. Water samples can be submitted to state or other government laboratories for analysis. Also, municipal water companies can be contacted to ask about mineral levels in local water supplies. Distilled water or water with less than 150 ppm sodium is recommended for patients with advanced heart disease and failure.

Good client communication is important for achieving desired outcomes, particularly in cats with cardiac disease. Discussion of diet, treats, table food, dietary supplements, and effective medication administration is beneficial for both the owner's and the cat's quality of life. Try to engage the client in decision making and defining expectations so that recommendations can address the cat's preferences, as well as the client's time, lifestyle, and financial limitations. Owners appear much more likely to change dietary factors (e.g., diet, treats, and/or supplements) than they would a medication. Therefore, it is relatively common to have various aspects of the diet change from one visit to the next. This is why it is important to perform a screening nutritional evaluation and to make specific recommendations at every visit (even if just to say that what the owner is doing is ideal). Demonstrating and teaching the client to effectively administer medications and to evaluate the body weight, BCS, and MCS is beneficial for engaging the client in their cat's care.

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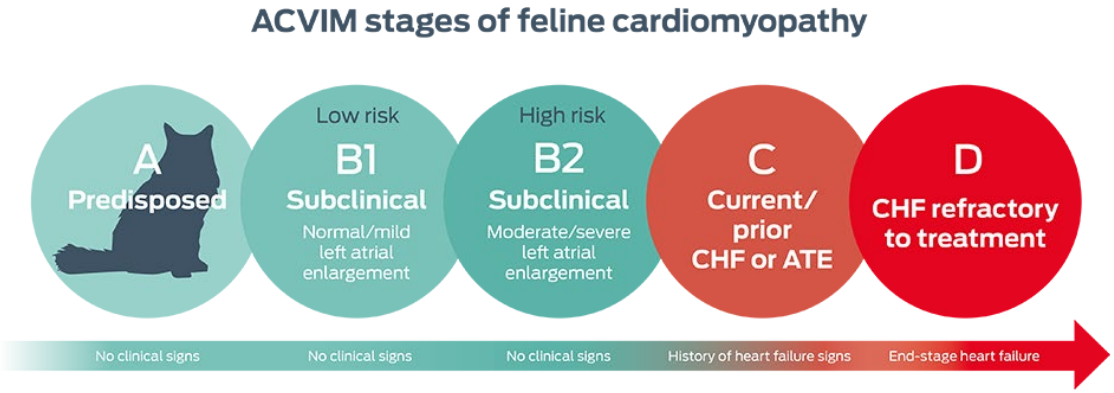

Table 3. The feline classification system as published by the ACVIM Consensus Statement (Luis Fuentes et al, 2020)

Stage	Description
A	Cats that are at risk of, or are predisposed to, developing cardiomyopathy but currently have no evidence of disease
B	Cats in this group have been diagnosed with hypertrophic cardiomyopathy (HCM) (because they have increased left ventricular (LV) wall thickness) but have **no clinical signs**. This group is divided in to two separate categories: • B1 — cats at low risk of developing congestive heart failure (CHF) or aortic thromboembolism (ATE) imminently. This is because they have no or mild left atrial enlargement • B2 — cats at higher risk of developing CHF or ATE imminently. This is because they have moderate to severe left atrial enlargement
C	Cats that currently have, or have had, signs of CHF or ATE
D	Cats that have become refractory to conventional CHF treatment