Population and survival characteristics of cats with hypertrophic cardiomyopathy: 260 cases (1990–1999)

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Objective—To determine current population characteristics of, clinical findings in, and survival times for cats with hypertrophic cardiomyopathy (HCM).

Design—Retrospective study.

Animals—260 cats with HCM.

Procedure—Information was obtained from the medical records. Cats were classified into 1 of 4 clinical groups (congestive heart failure [CHF] group, arterial thromboembolism [ATE] group, syncope group, or cats without clinical signs [subclinical group]) on the basis of the primary clinical signs at the initial examination.

Results—120 cats were classified in the CHF group, 43 in the ATE group, 10 in the syncope group, and 87 in the subclinical group. Antecedent events that may have precipitated CHF included IV fluid administration, anesthesia, surgery, and recent corticosteroid administration. Median survival time was 709 days (range, 2 to 4,418 days) for cats that survived > 24 hours. Cats in the subclinical group lived the longest (median survival time, 1,129 days; range, 2 to 3,778 days), followed by cats in the syncope group (654 days; range, 28 to 1,505 days), cats in the CHF group (563 days; range, 2 to 4,418 days), and cats in the ATE group (184 days; range, 2 to 2,278 days). Causes of death included ATE (n = 56), CHF (49), sudden death (13), and noncardiac causes (27). In univariate analyses, survival time was negatively correlated with left atrial size, age, right ventricular enlargement, and thoracentesis. Cats with systolic anterior motion of the mitral valve lived longer than cats without this echocardiographic finding. In multivariate analyses, only age and left atrial size remained significant predictors of survival time.

Conclusions and Clinical Relevance—Although overall survival time for cats with HCM was similar to earlier reports, survival times for cats with CHF or ATE were longer than previously reported. (*J Am Vet Med Assoc* 2002;220:202–207)

Hypertrophic cardiomyopathy (HCM) is a primary myocardial disease affecting cats, people, and a variety of other species. Hypertrophic cardiomyopathy is recognized by veterinary cardiologists as the most common cardiac disease in cats and is characterized by hypertrophy of the left ventricle without dilatation, impaired diastolic filling, and often secondary left atrial enlargement.[1,2] **Congestive heart failure (CHF)** and **arterial thromboembolism (ATE)** are common clinical manifestations in cats with HCM,[1,3] with ATE reportedly developing in up to 48% of affected cats.[4,5] Other clinical manifestations include syncope, arrhythmias, and sudden death.[6,7] In many cats, HCM is initially diagnosed following identification of a cardiac murmur or gallop rhythm, and the disease may remain without clinical signs for many years.[1,3]

A study[3] published in 1992 was the first large retrospective study of risk factors for, clinical signs in, and survival times of 74 cats treated for HCM during the 1980s. The prognosis for affected cats was linked to clinical signs at the time of initial examination. Cats without clinical signs had a median survival time of 1,830 days, cats with CHF had a median survival time of 92 days, and cats with ATE had a median survival time of 61 days. In addition, cats with tachycardia had shorter survival times than cats with heart rates < 200 beats/min. In a retrospective study[6] published in 1995, Fox et al provided data on 46 cats with HCM. Cats that died of CHF had larger left atrial size and greater ventricular hypertrophy and were less likely to have systolic anterior motion of the mitral valve. Peterson et al[8] reported that cats that survived longer than 3 months had a significantly smaller left atrial dimension and a higher fractional shortening.

It is critical to be able to provide an accurate prognosis for owners of cats with HCM. Results of a study[9] involving dogs with CHF documented that the veterinarian's prognosis was the single most important factor in an owner's decision to have euthanasia performed, and similar findings may be expected for cats with HCM. However, survival times for cats with HCM may have changed since the study[3] by Atkins et al because of improvements in diagnosis and changes in treatment. The purpose of the study reported here, therefore, was to determine current population characteristics of, clinical findings in, and survival times and prognostic factors for cats with HCM.

Criteria for Selection of Cases

Cats with HCM were identified retrospectively by searching the computer database containing information on all cats examined by the cardiology service at the Foster Hospital for Small Animals at Tufts University School of Veterinary Medicine. The database

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was searched to identify all cats examined between 1990 and 1999 in which the left ventricular free wall or interventricular septum at end-diastole was > 0.55 cm. Cats were subsequently selected for inclusion in the study if the attending cardiologist diagnosed HCM and retrospective review of the record was consistent with HCM. The decision to include or exclude a case was based on findings from the physical examination, electrocardiography, radiography, blood pressure measurement, laboratory testing, and echocardiography (ie, hypertrophy of the left ventricle without dilatation and evaluation of other echocardiographic findings including systolic anterior motion of the mitral valve, papillary muscle hypertrophy, and left atrial enlargement). Cats with hypertension, hyperthyroidism, aortic stenosis, intermediate forms of cardiomyopathy, and other cardiac diseases were excluded.

Procedures

Medical records of cats included in the study were reviewed, and information was collected on signalment; initial clinical signs; and results of cardiovascular examinations, laboratory testing, and initial treatment. Cats were classified into 1 of 4 clinical groups (CHF, ATE, syncope, or without clinical signs [subclinical]) on the basis of the primary complaint of the owner. Any antecedent event that may have precipitated CHF was recorded, and concurrent diseases were tabulated for each cat. The long-term medication protocol (dosage, adverse effects, and medication adjustments required) was also evaluated. Owners of cats for which outcome (alive, died, euthanatized) could not be determined from the medical record were contacted by telephone to determine current status and medications or date and cause of death. Survival time was calculated for cats that survived > 24 hours after the initial examination. In addition, any episodes of ATE that developed after diagnosis of HCM were recorded.

Statistical analyses—Data are given as mean ± SD (for normally distributed data) or median and range (for skewed data). Descriptive statistics were used to define the population characteristics of cats with HCM and survival times for cats in each clinical group (CHF, ATE, syncope, subclinical HCM). Categorical variables were compared by use of χ^2 tests; continuous data were analyzed with independent *t*-tests. Analysis of variance with Tukey HSD post-hoc analysis was used to compare continuous data among the 4 clinical categories. Multivariate linear regression analysis was used to correct for confounding factors. Values of $P < 0.05$ were considered significant. Statistical analyses were performed with commercial software.[a]

Results

Six hundred ninety-seven cats examined between 1990 and 1999 in which the left ventricular free wall or interventricular septum at end-diastole was > 0.55 cm were identified. After cats with other forms of heart disease were excluded, 260 cats with HCM were found to be eligible for inclusion in the study.

Median age of the cats with HCM was 5.6 years (range, 0.2 to 18.3 years). Two hundred six cats were male (193 castrated), and 54 were female (50 spayed). The most common breeds were domestic shorthair (n = 170) and domestic longhair (56). Other breeds that were represented included Persian (n = 8), Himalayan (6), Maine Coon (3), and other breeds (17). Mean ± SD body weight was 5.1 ± 1.3 kg (11.2 ± 2.9 lb).

At the time of initial examination, 120 cats were classified in the CHF group, 43 in the ATE group, 10 in the syncope group, and 87 in the subclinical group. There were no significant differences in regard to age, sex, or breed among clinical groups. Many of the cats had concurrent diseases including **lower urinary tract disease** (**LUTD**; n = 42), chronic renal failure (13), neoplasia (8), diabetes mellitus (3), and a variety of other diseases (55). Nine cats had 2 concurrent diseases, and 1 cat had 3 concurrent diseases.

For 61 of the 120 cats with CHF, an antecedent event that may have precipitated CHF was identified. These included IV fluid administration (n = 17), recent anesthesia or surgery (15; mean ± SD time prior to initial examination, 5.1 ± 5.5 days), recent corticosteroid administration (13; mean time prior to initial examination, 6.8 ± 7.7 days), trauma (7), upper respiratory tract infection (3), and miscellaneous causes (18). Twelve of the cats had > 1 antecedent event. For the 10 cats in which the type of corticosteroid administered could be confirmed, 7 had been treated with a long acting formulation of methylprednisolone,[b] and 3 had been treated with an injectable form of triamcinolone. The anesthetic regimen could be confirmed in 9 of the 15 cats in which recent anesthesia or surgery was identified as a precipitating event; ketamine hydrochloride had been used in 8 cats, and tiletamine-zolazepam[c] had been used in 1.

At admission, 59 of 260 cats had a cardiac gallop rhythm, 123 had a murmur, and 30 had both; however, the initial examination was not necessarily performed by a cardiac specialist. When cats were assessed by a cardiologist during the same hospital visit, 87 were found to have a gallop rhythm, 166 were found to have a murmur, and 50 were found to have both. Cats with mitral regurgitation ($P < 0.001$), systolic anterior motion of the mitral valve ($P < 0.001$), or a subjective assessment of left ventricular outflow tract obstruction ($P < 0.001$) had significantly higher-grade murmurs than did cats without these echocardiographic abnormalities.

Significant differences were found among cats in the 4 clinical groups in regard to rectal temperature, respiratory rate, heart rate, total CO_2 concentration, blood glucose concentration, and chloride concentration at admission (**Table 1**). During the initial hospitalization, cats with CHF were more likely to develop hypochloremia (n = 39; $P < 0.001$), hyponatremia (11; $P = 0.05$), hypokalemia (27; $P = 0.02$), and a high total CO_2 concentration (19; $P \leq 0.001$) than were cats in the other 3 clinical groups. Hepatic enzyme activities (alanine aminotransferase or aspartate aminotransferase) were high in 94 of the 139 (68%) cats in which they were measured.

Mean systolic blood pressure at the time of admission was 129 ± 32 mm Hg, and mean diastolic blood pressure was 86 ± 19 mm Hg. One hundred eighty-one

Table 1—Physical examination and laboratory findings for 260 cats with hypertrophic cardiomyopathy (HCM) grouped on the basis of clinical manifestation at the time of initial examination (congestive heart failure [CHF], arterial thromboembolism [ATE], syncope, and subclinical HCM)

Variable	CHF (n = 120)	ATE (n = 43)	Syncope (n = 10)	Subclinical HCM (n = 87)
Rectal temperature (C)	37.7 ± 1.3[a]	37.0 ± 1.5[b]	38.6 ± 0.7[a,c]	38.5 ± 0.6[c]
Heart rate (beats/min)	176 ± 41[a]	200 ± 36[b]	173 ± 37[a,b]	178 ± 32[a]
Respiratory rate (breaths/min)	62 ± 23[a]	60 ± 23[a]	43 ± 14[a,b]	42 ± 17[b]
Blood urea nitrogen (mg/dl)	43 ± 30	43 ± 58	32 ±10	42 ± 42
Creatinine (mg/dl)	1.9 ± 1.1	2.1 ± 2.7	1.8 ± 1.0	2.2 ± 2.0
Blood glucose (mg/dl)	165 ± 81[a]	199 ± 73[b]	111 ± 46[a,c]	133 ± 50[a,d]
Total CO_2 (mEq/L)	20 ± 6[a]	17 ± 3[a,b]	17 ± 4[a,b]	16 ± 3[b]
Sodium (mEq/L)	149 ± 5[a]	149 ± 4[a,b]	152 ± 5[a,b]	152 ± 5[b]
Chloride (mEq/L)	110 ± 9[a]	115 ± 6[b]	119 ± 4[b]	117 ± 7[b]
Potassium (mEq/L)	4.0 ± 0.8	4.0 ± 0.6	4.5 ± 0.5	4.2 ± 0.7

Values are given as mean ± SD. In each row, values with different superscript letters are significantly ($P < 0.05$) different from each other.

Table 2—Echocardiographic measurements in 260 cats with HCM grouped on the basis of clinical manifestation at the time of initial examination

Variable	CHF (n = 120)	ATE (n = 43)	Syncope (n = 10)	Subclinical HCM (n = 87)
Left atrium (cm)	1.80 ± 0.43[a]	1.99 ± 0.49[b]	1.63 ± 0.39[a,b,c]	1.50 ± 0.27[c]
Aorta (cm)	0.92 ± 0.14[a]	0.95 ± 0.12[a,b]	0.97 ± 0.13[a]	1.01 ± 0.11[b]
LVIDs (cm)	0.68 ± 0.23[a]	0.84 ± 0.27[b]	0.57 ± 0.22[a,c]	0.64 ± 0.18[a,c]
LVIDd (cm)	1.28 ± 0.26[a]	1.38 ± 0.29[a,b]	1.26 ± 0.22[a,b]	1.37 ± 0.21[b]
LVFWs (cm)	0.98 ± 0.18	0.97 ± 0.20	1.08 ± 0.17	1.00 ± 0.13
LVFWd (cm)	0.78 ± 0.19[a]	0.79 ± 0.17[a,b]	0.81 ± 0.19[a,b]	0.71 ± 0.16[b]
IVSs (cm)	0.94 ± 0.17	0.91 ± 0.16	1.01 ± 0.16	0.96 ± 0.17
IVSd (cm)	0.72 ± 0.17	0.70 ± 0.15	0.71 ± 0.15	0.67 ± 0.14
RVd (cm)	0.32 ± 0.16	0.30 ± 0.18	0.38 ± 0.18	0.28 ± 0.12
FS (%)	47.5 ± 13.2[a,b]	38.7 ± 20.4[a]	55.6 ± 12.6[a,b]	50.6 ± 23.0[b]

LVIDs = Left ventricular internal diameter at end-systole. LVIDd = Left ventricular internal diameter at end-diastole. LVFWs = Left ventricular free wall thickness at end-systole. LVFWd = Left ventricular free wall thickness at end-diastole. IVSs = Interventricular septum thickness at end-systole. IVSd = Interventricular septum thickness at end-diastole. RVd = Right ventricle diameter at end-diastole. FS = Fractional shortening.
See Table 1 for remainder of key.

cats underwent thoracic radiography. Abnormalities specifically recorded on the radiology or cardiology report included cardiomegaly (n = 165; 91%), pulmonary edema (119; 66%), pleural effusion (61; 34%), vascular enlargement (76; 42%), and hepatomegaly (21; 12%). Pulmonary edema was judged by a cardiologist to be the most important cause of respiratory distress in 111 of the 129 (86%) cats with CHF, and a large volume of pleural effusion was thought to be the most important cause of respiratory distress in 18 (14%).

We did not detect any significant differences among cats in the 4 clinical groups with regard to thickness of the interventricular septum at end-systole or end-diastole or thickness of the left ventricular free wall at end-systole (**Table 2**). Cats in the CHF group had a significantly larger left atrium ($P < 0.001$), smaller aorta ($P < 0.001$), smaller left ventricular internal dimension at end-diastole ($P = 0.04$), and thicker left ventricular free wall at end-diastole ($P = 0.04$) than did cats in the subclinical group. Cats in the ATE group had a significantly ($P < 0.001$) larger left atrium than did cats in the subclinical and CHF groups. Cats in the ATE group had a significantly ($P < 0.001$) larger left ventricular internal dimension at end-systole than did cats in the other clinical groups. Subjective echocardiographic abnormalities that were identified included left ventricular outflow tract obstruction (n = 146), mitral regurgitation (97), right ventricular enlargement (77), systolic anterior motion of the mitral valve (75), and pulmonary artery enlargement (22).

Cats with CHF received a mean of 30.3 ± 21.8 mg of furosemide during the first 24 hours of hospitalization and a cumulative dose of 38.9 ± 26.7 mg during the first 48 hours of hospitalization. Thoracentesis was performed in 24 cats, all of which had CHF. The median volume of fluid removed was 70 ml (range, 2 to 360 ml). Other medications administered on an emergency basis included nitroglycerine (n = 63), heparin (28), and streptokinase (19).

Cats received a variety of different medications, including combinations of medications, for treatment of HCM (**Table 3**). Among the 146 cats reevaluated at the Tufts University School of Veterinary Medicine, 86 (59%) were judged by the attending clinician to require an adjustment in medication. Changes included addition of a medication (n = 48), discontinuation of a medication (17), reduction of a dosage (23), and increase in a dosage (16).

During the course of long-term treatment of HCM, a treatment problem or complication was identified 92 times. Owner compliance issues were identified 56 times, and adverse effects of medications were identified 36 times. Adverse effects attributable to medications included vomiting or gastrointestinal tract bleed-

Table 3—Medications used for long-term treatment of 260 cats with HCM

Medication	No. treated	Dosage (mg/kg/d)		Duration of treatment (d)	
		Median	Range	Median	Range
Enalapril	112	0.35	0.11–1.00	396	1–3,029
Furosemide	119	1.25	0.33–6.25	155	1–3,032
Aspirin	132	5.30	2.65–11.25	429	1–3,365
Coumadin	11	0.06	0.01–0.14	80	1–669
Atenolol	57	1.39	0.30–3.91	527	2–2,051
Propranolol	10	1.34	0.63–2.21	589	6–2,925
Diltiazem	93	4.09	1.15–12.25	368	1–4,236
Long-acting diltiazem[d,e]	69	6.67	4.00–18.75	242	1–1,830
Low molecular weight heparin[f]	7	109	85–189	199	2–681
Hydrochlorothiazide-spironolactone	2	1.48	1.39–1.56	47	2–43

Most cats received more than 1 medication.

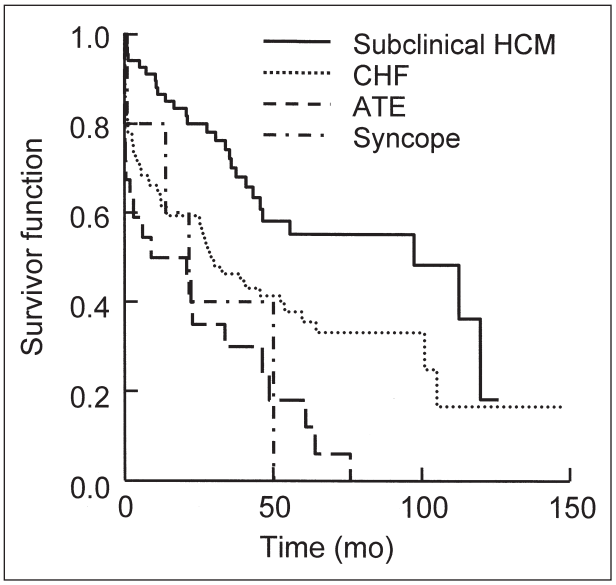

Figure 1—Survival times for cats with hypertrophic cardiomyopathy (HCM) grouped on the basis of clinical manifestation at the time of initial examination (congestive heart failure [CHF], arterial thromboembolism [ATE], syncope, and subclinical HCM).

ing, transient severe or persistent azotemia, hypotension, weakness, and complications secondary to streptokinase, such as reperfusion injury or hemorrhage.

Of the 260 cats, 72 were alive at the time of the study, 66 had died, 84 had been euthanatized, and 38 had been lost to follow-up. The proximate cause of death or reason for euthanasia was identified for 145 of the 150 cats that died or were euthanatized; these included ATE (n = 56), CHF (49), noncardiac causes (27), and sudden death (13). Thromboembolic disease was common among cats in all clinical groups; 73 cats developed ATE at some time during the course of their disease. Cats that developed ATE after the initial examination had a significantly ($P = 0.001$) larger mean left atrial size (1.92 ± 0.51 cm) at the time of initial examination than did cats that did not develop ATE (1.65 ± 0.38 cm). Cats in the subclinical group were significantly ($P < 0.001$) more likely to die of noncardiac causes than were cats in any of the other clinical groups. Median survival time, calculated for cats that survived > 24 hours after the initial examination, was 709 days (range, 2 to 4,418 days). Median survival time for cats in the subclinical group was 1,129 days (range, 2 to 3,778 days; **Fig 1**). Median survival time was 654 days (range, 28 to 1,505 days) for cats in the syncope group, 563 days (range, 2 to 4,418 days) for cats in the CHF group, and 184 days (range, 2 to 2,278 days) for cats in the ATE group. In univariate analyses, detection of systolic anterior motion of the mitral valve was positively ($P = 0.002$) associated with survival time. Left atrial size ($P < 0.001$), age ($P = 0.001$), subjective evidence of right ventricular enlargement ($P = 0.004$), and thoracentesis ($P = 0.005$) were negatively associated with survival time. There was no significant association between heart rate or an antecedent event and survival time. In multivariate analyses, only age ($P = 0.003$) and left atrial size ($P < 0.001$) remained significant predictors of survival time.

Discussion

Although overall survival time in the present study (median, 709 days) was similar to that reported by Atkins et al,[3] cats in the CHF and ATE groups in the present study lived longer than previously reported. These longer survival times may relate to changes in the natural history of the disease, geographical differences in disease severity, or use of newer medical treatments for cats with HCM. In particular, use of angiotensin-converting enzyme inhibitors to treat cats with HCM was not reported at the time of the earlier study.[3] Long-acting diltiazem formulations[d,e] are now available, and use of these long-acting formulations might improve treatment by maintaining blood concentrations for longer periods or enhancing owner compliance. As the present study was a retrospective study and most cats were receiving multiple medications, it was not possible to determine the independent effects of medications on outcome or survival time. In addition, the option of euthanasia makes survival analysis of veterinary patients difficult. Thus, prospective studies are needed to separate out factors that affect outcome and survival time. Nonetheless, the present study does provide up-to-date information on survival times that will assist veterinarians in giving a more accurate prognosis to cat owners.

Left atrial size was negatively associated with survival time in the present study. Left atrial enlargement is a result of reduced left ventricular diastolic compliance and often leads to CHF. It has also been reported

that severe enlargement of the left atrium is associated with development of ATE.[6,8,10] Cats in the present study that developed ATE after the initial examination had a significantly larger mean left atrial size at the time of initial examination than did cats that did not develop ATE. Previous studies have shown that interventricular septal hypertrophy[8] and overall left ventricular hypertrophy[6] were negatively associated with survival time, whereas no association between ventricular hypertrophy and survival time was detected in the current study. Fox et al[6] also showed that cats that survived were more likely to have the obstructive form of HCM. Although systolic anterior motion of the mitral valve (a manifestation of obstructive disease) was significantly associated with survival time in univariate analyses in the current study, it was not significant in multivariate analyses. Recent publications have indicated that newer Doppler techniques may be useful to characterize diastolic dysfunction in cats with HCM.[11-13] However, there were not enough cats in the present study in which Doppler studies measuring transmitral inflow and left ventricular outflow had been performed to be able to examine the effects of these data on survival. Unlike the study by Atkins et al,[3] a heart rate > 200 beats/min was not associated with worse survival time in the present study. The reason for this discrepancy is unclear; however, clinical decisions that might be made on the basis of initial heart rate in cats with HCM should be reevaluated in light of the present results.

Cats with HCM examined at a referral institution will usually die as a result of their cardiac disease.[3,6,14] In the present study, the most common cause of death or euthanasia was ATE. Of the 222 cats with adequate follow-up at the time of the study, 73 (32.8%) experienced an episode of ATE. There are still a limited number of strategies to treat acute ATE and to prevent its occurrence, so improved management of this manifestation of HCM is needed. Congestive heart failure was also a common cause of death in the present study. In many instances, cats that were initially examined because of CHF had stable disease for some time and then had a recurrence of CHF. In contrast to dogs, in which CHF tends to be a progressive disorder, many cats in the present study that initially had severe CHF went for many months or years before experiencing another episode of CHF.

Despite the longer survival times for cats in the CHF and ATE groups in the present study, the overall survival time was similar to that in the study by Atkins et al.[3] Possible reasons for this are the higher proportion of cats in the present study in the CHF group (46%), compared with cats in the study by Atkins et al[3] (32%), and the lower proportion of cats with subclinical HCM (33%), compared with cats in the study by Atkins et al (55%). Because sudden death is the first indication of HCM in some cats,[14] survival times in the present study may be skewed by underrepresentation of cats in which the initial indication of disease was sudden death. Finally, in the present study, most cats with subclinical HCM had concurrent diseases that eventually proved lethal, reducing the overall survival time for this group. In fact, significantly more cats with subclinical HCM died of noncardiac causes than did cats in any of the other clinical groups. The most common concurrent disease in this study was LUTD, which was identified in 16% of the cats. In a previous study,[15] 33% of cats with HCM had clinical signs compatible with LUTD. The association between these 2 diseases is unclear but may be an effect of IV fluid administration, initiation of diets with modified nutrient composition, or the stress of hospitalization.

Although a causal relationship could not be proven in this retrospective study, an antecedent event that could have contributed to CHF, such as IV fluid therapy, recent anesthesia or surgery, or corticosteroid administration, was identified in 61 of 120 cats with CHF. It has been proposed that cats with HCM may be intolerant of stress and tachycardia, because tachycardia shortens the duration of diastole and, therefore, limits coronary perfusion.[16,17] In theory, sustained tachycardia could worsen diastolic dysfunction in cats with HCM and precipitate CHF in cats with previously compensated disease.[16,17] This is in contrast to cats with healthy ventricles, in which sympathetic stimulation typically results in improved diastolic function. Invasive hemodynamic studies[4,16] in 4 cats with HCM identified an increase in left ventricular diastolic pressures in response to administration of isoproterenol, supporting the hypothesis that cats with stable HCM may have an acute decompensation in response to certain stimuli. Although corticosteroids may contribute to fluid retention because of their mineralocorticoid properties, methylprednisolone has minimal mineralocorticoid activity.[18] Ketamine or tiletamine-zolazepam was used in most cats in which anesthesia was identified as an antecedent event of CHF, and ketamine administration has previously been identified as a cause of myocardial damage.[19] Although identification of an antecedent event prior to development of CHF was not associated with outcome of cats in the present findings, results of this study do suggest that a cat that develops dyspnea after a potentially stressful event should be carefully evaluated for underlying cardiac disease.

As in previous studies,[3,6,8] cats in the present study were generally middle-aged male cats, although the age range was wide. On initial physical examination, most cats in this study had a cardiac murmur, a cardiac gallop rhythm, or both. Nonetheless, 57 of 260 (22%) cats did not have a murmur or gallop rhythm, even when examined by a cardiologist; therefore, screening cats for HCM by auscultation alone will miss a proportion of affected cats.

There were no differences in blood urea nitrogen or serum creatinine concentrations at the time of initial evaluation among cats in the 4 clinical groups in the present study. However, follow-up blood urea nitrogen concentration during the initial visit was higher for cats in the CHF group than for cats in the subclinical group. Cats in the CHF group also had a higher total CO_2 concentration and lower sodium and chloride concentrations during initial laboratory testing, compared with cats in the other clinical groups. This may be explained by the fact that many of these cats were treated for dyspnea with furosemide prior to the time

that the initial laboratory samples were obtained. During the initial hospitalization, cats in the CHF group were more likely to develop hypochloremia, hyponatremia, hypokalemia, and high total CO_2 concentration than were cats in the other 3 clinical groups. These abnormalities had resolved by the time of the first reevaluation and were most likely a result of furosemide administration, which interferes with chloride and electrolyte absorption in the ascending limb of Henle and can cause electrolyte depletion and metabolic alkalosis.

During reevaluation of cats at Tufts University, a medication adjustment was judged to be appropriate for 86 of 146 (59%) cats. The proportion requiring a medication adjustment was even higher among cats in the CHF group. This finding supports the suggestion that cats with HCM be reevaluated at regular intervals by an individual who is familiar with cardiac disease and cardiac medications. Medication adjustments were particularly likely to be needed within the first month for cats in the CHF group. Owner compliance issues were common in this study and were, in fact, even more common than adverse medication effects. Cats that need long-term treatment with oral medications can be difficult to manage as a result of problems with pill administration or reluctance of owners to administer medications long-term. Owner compliance issues were a common reason for changing medications in the present study, and many cats were changed from medications that required 3 times a day administration to medications that could be administered once a day for this reason. Future studies aimed at identifying novel methods to administer medications to cats would be helpful in improving owner compliance with medical management of cats with chronic cardiac disease.

[a]Systat 9.0, SPSS, Chicago, Ill.
[b]Depo-medrol, Pharmacia & Upjohn, Kalamazoo, Mich.
[c]Telazol, Fort Dodge Animal Health, Fort Dodge, Iowa.
[d]Dilacor XR, Watson Pharmaceuticals Inc, Corona, Calif.
[e]Cardizem-CD, Aventis Pharmaceuticals, Parsippany, NJ.
[f]Fragmin, Pharmacia & Upjohn, Kalamazoo, Mich.

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