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Longitudinal assessment of systolic anterior motion of the mitral valve in cats with hypertrophic cardiomyopathy

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Abstract

Background: The proportion of cats with hypertrophic cardiomyopathy (HCM) that lose systolic anterior motion of the mitral valve (SAM) in the long term is unknown.

Hypothesis/Objectives: Cats with HCM will lose SAM in the long term. Loss of SAM will be associated with greater age, longer scan-interval, and altered left ventricular (LV) dimensions.

Animals: Sixty unsedated cats with HCM, not receiving beta blockers or pimobendan.

Methods: A retrospective cohort study from 2 referral centers. Cats were eligible if they had been diagnosed with HCM and had a repeat echocardiogram ≥1 year later. Clinical and echocardiographic data of the left heart variables were collected.

Results: Thirty-eight cats had SAM at the initial scan. After a median follow-up time of 2.1 years (range: 1.0-5.9), 7 cats had lost SAM (18%) and 5 cats (23%) gained SAM. On follow-up, cats with SAM at the initial scan had a larger left atrium ($P = .037$), lower left atrial fractional shortening ($P = .014$), greater LV internal diameter in end-systole ($P = .002$), and lower LV fractional shortening ($P < .001$). Four cats with SAM developed congestive heart failure. There were no new cases of congestive heart failure or change in left heart variables in cats without SAM at the initial scan. The gain or loss of SAM was not associated with age or time between scans.

Conclusions and Clinical Importance: Similar proportions of cats gained or lost SAM. Cats with SAM at baseline had more evidence of disease progression than cats without SAM.

KEYWORDS
cardiology, echocardiogram, echocardiograph, feline, obstructive cardiomyopathy

1 | INTRODUCTION

Hypertrophic cardiomyopathy (HCM) is defined as left ventricular (LV) hypertrophy because of an unknown cause or a sarcomeric mutation.[1] Systolic anterior motion of the mitral valve (SAM) is present in 30% to

Abbreviations: AMVL, anterior mitral valve leaflet; 2D, 2-dimensional echocardiography; DLVOTO, dynamic left ventricular outflow tract obstruction; HCM, hypertrophic cardiomyopathy; IVS, interventricular septum; LA, left atrial; LV, left ventricular; LVFW, left ventricular free wall; LVOT Vmax, maximal left ventricular outflow tract velocity; MV, mitral valve; RPLax, right parasternal long axis; RPSax, right parasternal short axis; SABP, systolic arterial blood pressure; SAM, systolic anterior motion of the mitral valve.

50% of cats at the time of HCM diagnosis,[2,3] and is characterized as abnormal movement of the mitral valve (MV) toward the interventricular septum, narrowing the LV outflow tract (LVOT) and causing dynamic LVOT obstruction (DLVOTO).

Severe DLVOTO is associated with increased risk of symptoms and death in people with HCM.[4,5] In studies of prognosis in cats with HCM, SAM is not a risk factor for heart failure or death.[3,6-9] However, most studies of SAM in cats have been retrospective, with cats evaluated at a single time-point. Recent prospective studies report that some cats gained or lost SAM over time.[10,11] If the presence of SAM is evaluated at only a single time point in a study, any cats that had previously lost SAM would be mistakenly classified as nonobstructive HCM. Any association of SAM with the frequency of subsequent progression of cardiac disease would then be underestimated.

Several morphological and functional abnormalities of the left heart are associated with SAM in humans.[12-14] These include longer MV leaflet length and area; reduced mitral-aortic angle; reduced distance between MV coaptation point and interventricular septum (IVS); septal bulge; and abnormal papillary muscle position and orientation.[13,14] Additionally, greater LV systolic function and enhanced adrenergic tone alter the flow vector and drag the MV leaflets toward the LVOT in humans.[15] In cats, longer anterior MV leaflet (AMVL) length, papillary muscle hypertrophy, and increased numbers of false tendons in the LVOT are associated with the presence of SAM.[16] Many of these factors require specific views for optimal imaging, so should ideally be studied either prospectively, or using computerized tomography or cardiac magnetic resonance imaging. However, echocardiographic details of left heart dimensions such as LV wall thickness, LV fractional shortening and left atrial (LA) size can be reported in retrospective studies.

Hypertrophic cardiomyopathy can take years to progress.[9,10] The aims of this study were to describe the incidence of change in the presence of SAM between baseline and follow-up assessment, and any clinical and echocardiographic features associated with the gain or loss of SAM. The hypotheses were (1) some cats with HCM will gain or lose SAM between assessments ≥12 months apart, (2) the gain or loss of SAM are associated with cat's age, scan-interval, changes in the LV dimensions, and (3) cats with SAM will show more disease progression than cats without SAM. Greater understanding of SAM will better define HCM and help design future treatment trials or outcome studies in cats with HCM.

2 | MATERIALS AND METHODS

2.1 | Study design

This was a retrospective cohort study involving 2 referral centers (Animal Referral Centre, New Zealand and Queen Mother Hospital for Animals, The Royal Veterinary College, United Kingdom). Ethical approval for data collection in the United Kingdom was provided by the Royal Veterinary College Ethics Committee (URN: M20170143). Ethical approval for data collection in New Zealand was not required as this data was collected retrospectively.

Electronic medical records between October 2009 to January 2018 from Queen Mother Hospital for Animals, and between July 2020 to January 2023 from Animal Referral Centre, were searched for cats referred to a cardiologist for cardiac screening before breeding or blood donation, or for investigation of a heart murmur. Cats were included if they were diagnosed with HCM and had at least 2 echocardiograms ≥12 months apart performed by a board-certified cardiologist or supervised cardiology resident. Cats were excluded if they met any of the following criteria at baseline or recheck: incomplete clinical records, echocardiographic studies with low 2D temporal resolution (<60 frames per second), use of sedation, beta blockers or pimobendan in between or during the 2 time points, presence of clinical dehydration, hyperthyroidism, diabetes mellitus, cardiac neoplasia, congenital heart disease, anemia (packed cell volume <20%), and systemic hypertension. Systemic hypertension was defined as Doppler systolic arterial blood pressure (SABP) measurement of ≥180 mm Hg, or ≥160 mm Hg if the cat had creatinine ≥140 μmol/L (≥1.6 mg/dL) or was ≥6 years old.

Clinical and echocardiographic data were collected at baseline and last recheck. For the clinical data, the age, sex, breed, body weight, reason for cardiac exam, medication, physical examination findings, Doppler SABP recordings, presence of arrhythmias, electrocardiogram findings, and clinical signs were recorded.

Each echocardiogram was reviewed and measured over 3 different cardiac cycles by a single observer (J.S.). The diagnosis of HCM was confirmed when end-diastolic left ventricular wall thickness (LVWT Max) measured ≥6 mm, and other known differentials of an HCM phenotype ruled out based on the clinical summary.[3] The measurements of 2-dimensional echocardiography (2D) variables were LV internal diameter in end-diastole and end-systole measured using a right parasternal short-view axis (RPSax) view[17]; LV fractional shortening calculated based on LV internal diameters[17]; maximal thickness of the IVS and LV free wall (LVFW) in end-diastole in right parasternal long axis (RPLax) 4-chamber, 5-chamber and RPSax views[18]; maximal LA diameter using a RPLax 4-chamber view,[18] LA to aortic ratio using a RPSax view,[19] and AMVL length from a RPLax 5-chamber view.[20] The RPSax view at the aortic valve level was used to guide M-mode for measurement of LA fractional shortening.[7] Maximal LVOT velocity (LVOT Vmax) was measured from a left parasternal apical 5-chamber view using spectral Doppler echocardiography guided by color Doppler.[3] Maximal LV wall thickness was recorded based on the IVS and LVFW measurements (whichever was greater).[10,21] Additionally, the presence of dynamic right ventricular outflow tract obstruction (defined as turbulence of right ventricular outflow tract in the absence of an anatomical abnormality with blood flow velocities exceeding 1.7 m/s)[22]; DLVOTO (defined as LVOT Vmax exceeding 2.5 m/s)[3,16]; mid-LV obstruction (defined as the presence of turbulence on color Doppler during systole)[23]; end-systolic cavity obliteration (defined as the disappearance of the LV cavity at end-systole on 2D imaging),[17] and regional LV wall hypokinesis (defined as reduced segmental LV wall motion)[24] were recorded. The heart rate was calculated from the echocardiographic loop used to diagnose SAM.

The presence of SAM was defined as the anterior movement of the AMVL toward the LV outflow tract during systole using 2D

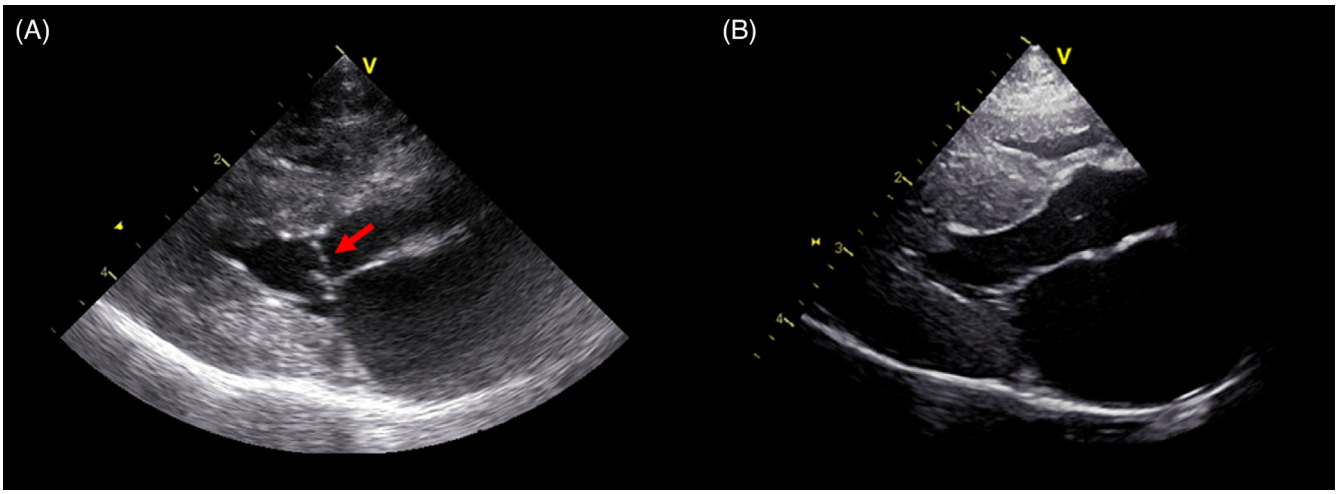

FIGURE 1 Still images of the right parasternal long-axis 5-chamber view at end-systole in 2 cats with hypertrophic cardiomyopathy. The left (A) image shows systolic anterior motion of the mitral valve (arrow), with the anterior mitral valve leaflet touching the interventricular septum during systole. The right (B) image shows a cat without systolic anterior motion of the mitral valve.

imaging (Figure 1).[14,16,21] In addition to the primary observer, a second observer (V.L.F.) independently assessed the presence of SAM. The second observer was not provided with a clinical summary, timing of the echocardiogram, or the assessment from the primary observer (J.S.). When there was disagreement between the primary and second observer, the echocardiogram was viewed together, and the final consensus was used for the statistical analysis.

2.2 | Statistical analysis

Normality of continuous data was tested by visual assessment and Shapiro-Wilk test. Normally distributed data were presented as mean (95% confidence interval [95% CI]) and nonnormally distributed data were presented as median (range). The baseline and recheck data of the study population were compared by a paired *t* test or a Wilcoxon signed ranked test depending on the normality of the data. The study population was then further analyzed by dividing them into 2 groups: those that had SAM at baseline and those that did not. Continuous variables in these 2 groups were compared by independent *t*-test or a Mann-Whitney test. Categorical demographic variables were compared by Pearson chi-square or Fisher's exact test, as appropriate. The baseline characteristics, time interval between 2 echocardiograms, reasons for the second cardiac scan, and the percentage change in heart rate, body weight, and all echocardiographic variables were compared between cats with SAM at baseline and follow-up vs those that lost SAM at recheck, and between cats without SAM at baseline and follow-up vs those that gained SAM at recheck. The level of agreement between 2 observers on diagnosing SAM was assessed by Cohen's kappa (κ). 95% CI was calculated by 1.96 $\times$ SE. The frequency of cats gaining or losing SAM was presented as incidence rate, where the numerator equaled the number of cats that gained or lost SAM during the follow-up period and the denominator equaled the number of cat-years at risk. Statistical significance was set at <.05.

3 | RESULTS

A total of 130 cats met the inclusion criteria from the retrospective data search. Of these, 87 cats were excluded for the following reasons: 15 for incomplete medical records, 4 for SABP ≥180 mm Hg, 2 for unavailable SABP with creatinine ≥140 μmol/L (≥1.6 mg/dL), 10 for hyperthyroidism, 6 for diabetes mellitus, 1 for packed cell volume <20%, 35 for receiving atenolol, 5 for receiving sedation, 8 for receiving pimobendan, 1 for possible cardiac neoplasia, 1 for limited echocardiographic study, and 4 for having low temporal resolution (<60 FPS). This resulted in 38 cats. An additional 22 cats were recruited by being prospectively invited for a recheck at the Queen Mother Hospital for Animals. The final sample size was 60 cats. There was no statistical difference between the baseline variables between the original study group and those of the 22 additional cats.

The median temporal resolution of the cineloop used to diagnose SAM was 112 frames per second (minimum 60; maximum 317). There was good agreement between 2 observers on the diagnosis of SAM ($\kappa = 0.831$, 95% CI: 0.727-0.934, $P < .001$). The 9 out of 120 scans where the disagreement arose was thought to be because of end-systolic cavity obliteration, which made the accurate observation of mitral valve leaflets difficult. These scans occurred in 2 cats at both time points and 5 cats at a single time point. Both observers ultimately agreed not to have these cats classified as having SAM.

At baseline, 38 cats (63%; 95% CI: 50%-75%) had SAM and 22 cats (37%; 95% CI: 25%-50%) did not have SAM. Two cats without SAM had congestive heart failure. Three cats were receiving medications: furosemide and clopidogrel (n = 1), furosemide and benazepril (n = 1), and aspirin (n = 1). One cat without SAM had ventricular

TABLE 1 Baseline study population characteristics.

	Whole group (n = 60)	SAM (n = 38)	No SAM (n = 22)	*P* value
Age (years)[cont]	5.6 [0.3-14.2]	3.9 [0.3-11.2]	8.0 [1.5-14.2]	<.001
Body weight (kg)[cont]	4.7 [2.8-8.8]	4.3 [2.8-7.6]	5.7 [3.8-8.8]	.008
Body condition score (/9)[cat/o]	5 [4-9]	5 [4-8]	6 [4-9]	.266
Sex				
Female : male[cat/b]	14 : 46	11 : 27	3 : 19	.219
Entire : neutered[cat/b]	2 : 58	2 : 36	0 : 22	.251
Purebred : Nonpurebred[cat/b]	22 : 38	11 : 27	11 : 11	.163
Balinese		1		
Bengal		1	1	
British Shorthair		1	1	
Burmese				
Exotic Shorthair			1	
Himalayan			1	
Maine Coon		2		
Norwegian Forest Cat			1	
Persian		1	3	
Ragdoll			1	
Russian Blue		1	2	
Siberian		1	1	
Sphynx		3		
Presenting complaint[cat/n]				.041
Cardiac screening[cat/b]	12	4	8	.022
Lethargy[cat/b]	2	1	1	1.000
Heart murmur[cat/b]	43	32	11	.007
Respiratory signs[cat/b]	3	1	2	.548
Heart rate (per minute)[cont]	182.6 [131-236]	181.1 [133-236]	183.5 [131-236]	.921
Murmur (%)[cat/b]	49 (81.7%)	36 (94.7%)	13 (59.1%)[a]	.001
Murmur grade[cat/o]	3 (2-4)	3 (2-4)	2 (2-4)	<.001
Gallop (%)[cat/b]	5 (8.3%)	3 (7.9%)	2 (9.1%)	.872
Medication	3 cats[a]	None	3 cats[b]	–
SABP (mm Hg)[cont]	128.9 (121.1-136.8; n = 32)	126.8 (118.5-135.1; n = 18)	131.6 (115.8-147.5; n = 14)	.565

Note: Variable types are annotated using superscripts: [cont] for continuous, and [cat/o] for ordinal, [cat/b] for binary and [cat/n] for nominal categorical variables. Normally distributed data are presented as mean (95% confidence interval) and nonnormally distributed data as median [range]. Continuous variables are presented in mean (± SD) when normally distributed and median (range) when nonnormally distributed.

Abbreviations: SABP, systolic arterial blood pressure; SAM, systolic anterior motion of the mitral valve.

[a]One cat without SAM had dynamic right ventricular outflow tract obstruction and 3 had midventricular obstruction. The cause of a heart murmur was not identified in the remaining 9 cats without SAM.

[b]One cat was receiving aspirin, 1 cat was receiving furosemide and clopidogrel, and 1 cat was receiving furosemide and benazepril.

premature complexes. Cats with SAM were younger, weighed less, and had higher LVOT Vmax, longer AMVL length, and greater LV hypertrophy than those without SAM. A summary of the baseline clinical and echocardiographic characteristics of the 2 groups is presented in Tables 1, 2, and Figure 2.

The median follow-up period was 2.1 years (minimum 1.0 years; maximum 5.9 years), which was not different between the 2 groups (No SAM at baseline, median 1.8 years [minimum 1.0 year; maximum 5.9 years] vs SAM at baseline, 2.4 years [minimum 1.0 year; maximum 5.7 years], $P = .067$, Figure S1). With the entire study population assessed as a single group, at recheck the LA size had increased (LA to aortic ratio, mean 1.4 [95% CI: 1.4-1.5] to 1.6 [95% CI: 1.4-1.7]), LA function reduced (LA fractional shortening, mean 29.5% [95% CI: 27.2-31.8] to 27.1% [95% CI: 24.1-30.1]), and the LV diameter in end-systole increased (mean 5.0 mm [95% CI: 4.5-5.6] to 5.8 mm [95% CI: 5.1-6.4], Table 3). Further analysis showed that this progression in left

TABLE 2 Baseline echocardiography results.

	Study group (n = 60)	SAM (n = 38)	No SAM (n = 22)	*P* value
ATE (%)[cat/b]	0	0	0	–
CHF (%)[cat/b]	2	0	2	.131
Arrhythmias (%)[cat/b]	1	0	1	.373
Scan interval (years)[cont]	2.1 [1.0-5.9]	2.4 [1.0-5.7]	1.8 [1.0-5.9]	.067
Left atrium				
LA/Ao[cont]	1.4 [1.1-2.1]	1.4 [1.1-2.1]	1.3 [1.2-2.0]	.730
LAD Max (mm)[cont]	16.2 [14.7-25.7]	15.5 [14.6-25.7]	16.5 [12.8-21.7]	.586
LA FS%[cont]	29.5 (27.2-31.8)	28.7 (26.0-31.4)	30.9 (26.3-35.4)	.379
Left and right ventricle				
IVSd Max (mm)[cont]	6.6 [5.4-9.7]	6.9 [6.0-9.7]	6.3 [5.4-9.1]	.024
LVFWd Max (mm)[cont]	6.1 [4.3-8.5]	6.2 [4.3-8.5]	5.9 [4.7-7.5]	.079
LVWT Max (mm)[cont]	6.7 [6.0-9.7]	6.9 [6.0-9.1]	6.5 [6.0-9.1]	.027
LVIDd (mm)[cont]	14.9 (14.4-15.4)	14.8 (14.1-15.5)	15.0 (14.2-15.9)	.686
LVIDs (mm)[cont]	5.0 (4.5-5.6)	4.6 (3.9-5.2)	5.8 (5.0-6.7)	.020
LV FS%[cont]	66.5 (63.2-69.7)	69.5 (65.7-73.4)	61.2 (55.7-66.7)	.012
End-systolic cavity obliteration[cat/b]	41 (68.3%)	29 (76.3%)	12 (54.5%)	.081
Regional wall hypokinesis[cat/b]	0	0	0	–
Midleft ventricular obstruction[cat/b]	11/23 (47.8%)	7/12 (58.3%)	4/11 (36.4%)	.292
LVOT Vmax (m/s)[cont]	1.9 [0.7-5.4]	2.9 [0.9-5.4]	1.0 [0.7-2.1] (n = 18)	<.001
DLVOTO (%)[cat/b]	25 (41.7%)	25 (65.8%)	0	<.001
DRVOTO (%)[cat/b]	9/54 (16.7%)	8/35 (22.9%)	1/19 (5.3%)	.137
Mitral valve apparatus				
AMVL length (mm)[cont]	11.1 [7.0-18.4]	12.2 [9.7-18.4]	10.7 [7.0-14.2]	.002

Note: Variable types are annotated using superscripts: [cont] for continuous and [cat/b] for binary categorical variables. Normally distributed data are presented as mean (95% confidence interval) and nonnormally distributed data as median [range].

Abbreviations: AMVL, anterior mitral valve leaflet; ATE, aortic thromboembolism; CHF, congestive heart failure; DLVOTO, dynamic left ventricular outflow tract obstruction; DRVOTO, dynamic right ventricular outflow tract obstruction; IVSd Max, maximal interventricular septal thickness in end-diastole; LA/Ao, left atrium to aortic ratio; LAD Max, maximal left atrial diameter; LA FS%, left atrial fractional shortening; LV FS%, left ventricular fractional shortening; LVFWd Max, maximal left ventricular free wall thickness in end-diastole; LVIDd, left ventricular internal diameter in end-diastole; LVIDs, left ventricular internal diameter in end-systole; LVOT Vmax, maximal left ventricular outflow tract velocity; LVWT Max, maximal left ventricular wall thickness in end-diastole; SAM, systolic anterior motion of the mitral valve.

heart dimensions was only seen in cats with SAM at baseline. At recheck, cats with SAM at baseline had increased LA size (LA to aortic ratio, mean 1.4 [95% CI: 1.4-1.5] to 1.4 [95% CI: 1.4-1.7]; LA maximal diameter, mean 15.5 mm [95% CI: 14.9-17.4] to 16.0 mm [95% CI: 16.3-19.3]), reduced LA function (LA fractional shortening, mean 28.7% [95% CI: 26.0-31.4] to 25.1% [95% CI: 21.7-28.5]), increased LV diameter in end-systole (mean 4.3 mm [95% CI: 3.9-5.2] to 5.3 mm [95% CI: 5.1-6.5]), decreased LV fractional shortening (mean 69.5% [95% CI: 65.7-73.4] to 61.3% [95% CI: 57.5-65.0]), and lost any mid-LV obstruction (58.3% [95% CI: 27.7%-84.8%] to 24.0% [95% CI: 9.4-45.1]). There was no change in left heart variables in cats without SAM at baseline.

Four cats with SAM at the initial scan subsequently developed congestive heart failure (Table 4). Two of these cats had lost SAM at recheck. Seven cats with SAM at the initial scan developed arrhythmias. All cats with arrhythmias had an electrocardiogram performed by the echocardiographic machine or a separate 6 lead electrocardiogram. The new arrhythmias consisted of ventricular premature complexes (n = 4), atrial premature complexes (n = 1), atrial fibrillation (n = 1), and supraventricular tachycardia (n = 1). None of the cats without SAM at initial scan developed new congestive heart failure or arrhythmias.

At recheck, the 2 cats with congestive heart failure at baseline were also receiving aspirin and spironolactone. Additionally, 10 new cats were on medications, all of which had SAM at baseline. Medications administered were furosemide, benazepril, aspirin, and spironolactone (n = 1); furosemide, benazepril, and aspirin (n = 1); furosemide and clopidogrel (n = 1); furosemide alone (n = 1); benazepril (n = 2); and clopidogrel (n = 4).

The incidence rate of cats to either gain or lose SAM was 8 per 100 cat-years. The incidence rate of cats gaining SAM was 10 per 100 cat-years. The incidence rate of cats losing SAM was 7 per 100 cat-years. None of the cats that gained or lost SAM had disagreement of the SAM diagnosis between 2 observers.

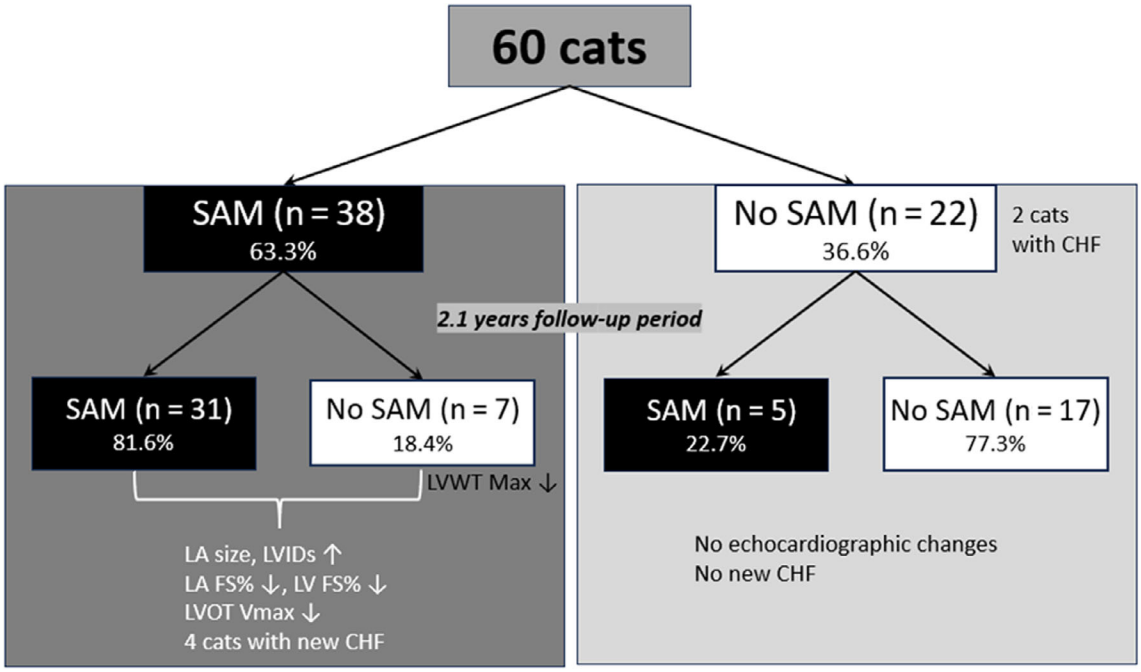

FIGURE 2 Summary of the study population and the results. Out of 60 cats, 38 cats had systolic anterior motion of the mitral valve (SAM) and 22 did not at baseline. After a median follow-up period of 2.1 years, 7 cats lost SAM and 5 cats gained SAM. The loss of SAM was associated with reduced maximal left ventricular wall thickening (LVWT Max). Only the cats with SAM at baseline showed progression in disease severity. These included increase in left atrial (LA) size and left ventricular internal diameter in end-systole (LVIDs), reduced left atrial fractional shortening (LA FS%), reduced left ventricular fractional shortening (LV FS%), reduced left ventricular outflow tract obstruction (LVOT Vmax). Four cats that had SAM at baseline developed congestive heart failure (CHF). Two cats without SAM at baseline had congestive heart failure. But none of the cats without SAM developed new congestive heart failure or changes in the echocardiographic variables.

Cats that gained SAM at recheck had a higher baseline LVOT, thinner LVFW and a greater ratio of maximal IVS to maximal LVFW thickness than those that did not. None of the baseline characteristics were associated with cats that later lost SAM (Table 5).

Gaining SAM was associated with increased LVOT Vmax and decreased maximal IVS to maximal LVFW thickness ratio (Table 6). There was no statistical difference between maximal IVS and maximal LVFW as individual variables between 2 time points.

Losing SAM was associated with reduction in maximal LVFW thickness and loss of DLVOTO (Table 7). The change in LA size, LA function, LV internal diameter in end-systole, LV fractional shortening, loss of end-systolic cavity obliteration, and development of new CHF or arrhythmia were not associated with the loss of SAM ($P > .05$). The changes in scan interval, LV dimensions, and LVOT Vmax in cats that gained or lost SAM are shown in Figures 3 and 4.

4 | DISCUSSION

In this retrospective cohort study, the incidence rate of a change in the presence of SAM in cats with HCM was 8 per 100 cat-years. That is, in the order of 8 out of every 100 cats with HCM are expected to experience a change in the presence of SAM over a 12-month period. This high number of cats that gained or lost SAM further demonstrates that SAM is labile, and its presence can change over time. In a previous study, 3 of 14 cats were found to gain SAM (21.4%) in a median follow-up period of 5.6 years, whereas 4 of 7 cats lost SAM (57.1%) during the same time period.[10] In another study, 1 of 8 cats lost SAM (12.5%) and 1 of 8 cats gained SAM (12.5%) during a 6 month time period.[11] The results of the present study support these previous observations that cats with HCM can gain or lose SAM.

An unexpected finding was that cats with SAM at the initial scan were more likely to show disease progression than cats without SAM. In previous studies of feline HCM, cats with obstructive HCM are reported to have a similar or more benign outcome to nonobstructive cats.[3,7-9] Possible reasons for a different result in our study to previous studies include selection bias. In previous studies, cats with SAM have tended to be younger than those without.[3,7-9] Cats with obstructive HCM would be more likely to present with a heart murmur than cats with nonobstructive HCM, and this might lead to investigations at a younger age in cats with SAM than those without. Cats with nonobstructive HCM are more likely to remain undiagnosed until the development of clinical signs.

Furthermore, the labile nature of SAM would likely have influenced the result. Young cats might be more excitable than old cats, and more likely to exhibit SAM in a stressful environment. It is difficult to predict whether cats diagnosed with obstructive HCM remain obstructed at home, further biasing outcomes data of these studies. However, in our study the heart rate during echocardiography was similar between cats with SAM and those without SAM, suggesting

TABLE 3 Comparison of baseline and recheck characteristics (whole cohort).

	Baseline	Recheck	*P* value
Age (years)[cont]	5.6 [0.3-14.2]	8.0 [2.5-18.0]	–
Body weight (kg)[cont]	4.7 [2.8-8.8]	4.9 [3.2-9.8]	.409
Body condition score (/9)[cat/o]	5 [4.0-9.0]	5.0 [2.0-9.0]	.891
Medication	3 cats	14 cats	–
ATE (%)	0	0	–
CHF (%)[cat/b]	2 (3.3%)	6 (10.0%)	.272
Arrhythmias (%)[cat/b]	1 (1.7%)	8 (13.3%)	.032
Murmur (%)[cat/b]	49 (81.7%)	42 (70.0%)	.154
Murmur grade (/6)[cat/o]	3 (2-4)	3 (2-4)	.595
Gallop sound[cat/b]	5 (8.3%)	9 (15.0%)	.255
Heart rate (bpm)[cont]	182.6 [131-236]	186 [140-246]	.125
SABP (mm Hg)[cont]	128.9 (121.1-136.8; n = 32)	138.9 (130.3-147.5; n = 35)	.178
Left atrium			
LA : Ao[cont]	1.4 [1.1-2.1]	1.4 [1.0-3.0]	.024
LAD Max (mm)[cont]	16.2 [14.7-25.7]	16.0 [11.5-30.7]	.060
LA FS%[cont]	29.5 (27.2-31.8)	27.1 (24.1-30.1)	.043
Left and right ventricle			
LVWT Max (mm)[cont]	6.7 [6.0-9.7]	7.2 [5.6-10.2]	.297
LVIDd (mm)[cont]	14.9 (14.4-15.4)	15.0 (14.4-15.6)	.561
LVIDs (mm)[cont]	5.0 (4.5-5.6)	5.8 (5.1-6.4)	.038
FS%[cont]	66.5 (63.2-69.7)	62.1 (58.6-65.6)	.051
End-systolic cavity obliteration[cat/b]	41 (68.3%)	33 (55.0%)	.133
Regional wall hypokinesis[cat/b]	0	3 (5.0%)	.244
Midleft ventricular obstruction[cat/b]	11/23 (47.8%)	9/33 (27.3%)	.114
LVOT Vmax (m/s)[cont]	1.9 [0.7-5.4]	1.6 [0.7-5.2]	.076
DLVOTO (%)[cat/b]	25/60	19/56	.391
DRVOTO (%)[cat/b]	9/54	16/52	.087
Mitral valve apparatus			
SAM (%)[cat/b]	38 (63.3%)	36 (60.0%)	.707
SAM group[cat/b]	38/38 (100%)	31/38 (81.6%)	.012
No SAM group[cat/b]	0/22 (0%)	5/22 (22.7%)	.048
AMVL length (mm)[cont]	11.1 [7.0-18.4]	11.7 [8.9-16.0]	.880

Note: Variable types are annotated using superscripts: [cont] for continuous, and [cat/o] for ordinal and [cat/b] for binary categorical variables. Normally distributed data are presented as mean (95% confidence interval) and nonnormally distributed data as median [range].

Abbreviations: AMVL, anterior mitral valve leaflet; ATE, aortic thromboembolism; CHF, congestive heart failure; DLVOTO, dynamic left ventricular outflow tract obstruction; DRVOTO, dynamic right ventricular outflow tract obstruction; LA/Ao, left atrium to aortic ratio; LAD Max, maximal left atrial diameter; LA FS %, left atrial fractional shortening; LV FS%, left ventricular fractional shortening; LVIDd, left ventricular internal diameter in end-diastole; LVIDs, left ventricular internal diameter in end-systole; LVOT Vmax, maximal left ventricular outflow tract velocity; LVWT Max, maximal left ventricular wall thickness in end-diastole; SABP, systolic arterial blood pressure; SAM, systolic anterior motion of the mitral valve.

that cats with or without SAM were similarly stimulated during echocardiography. Further studies are required to determine if more accurate identification of latent SAM will reveal an association between SAM or DLVOTO and disease outcome in cats.

The labile nature of SAM is well described in human cardiology. To accurately assess the clinical significance of SAM, pharmacologic and physiologic provocative methods have been proposed.[25] These methods stimulate adrenergic tone, myocardial contractility, and

TABLE 4 Clinical characteristics at baseline and recheck in cats with and without SAM at baseline.

	SAM at baseline (n = 38)			No SAM at baseline (n = 22)		
	Baseline	**Recheck**	***P* value**	**Baseline**	**Recheck**	***P* value**
Age (years)[cont]	3.9 [0.3-11.2]	6.8 [2.5-14.9]	–	8.0 [1.5-14.2]	10.8 [3.3-18.0]	–
Body weight (kg)[cont]	4.3 [2.8-7.6]	4.4 [3.2-8.4]	.378	5.7 [3.8-8.8]	5.4 [3.3-9.8]	.917
Body condition score (/9)[cat/o]	5 [4-8]	5 [3-7]	.639	6 [4-9]	5 [2-9]	.824
Medication	0 cats	9 cats	–	3 cats	3 cats	–
ATE (%)[cat/b]	0	0	–	0	0	–
CHF (%)[cat/b]	0	4 (10.5%)	.115	2 (9.1%)	2 (9.1%)	1.000
Arrhythmias (%)[cat/b]	0	7 (18.4%)	.012	1 (4.5%)	1 (4.5%)	1.000
Heart murmur (%)[cat/b]	36 (94.7%)	32 (84.2%)	.051	13 (59.1%)	10 (45.5%)	.554
Murmur grade (/6)[cat/o]	3 [3-4]	3 [2-4]	.469	2 [2-4]	3 [2-4]	.459
Gallop sound (%)[cat/b]	3 (7.9%)	6 (15.8%)	.480	2 (9.1%)	3 (13.6%)	1.000
HR (bpm)[cont]	181.1 [133-236]	184.0 [140-245]	.528	183.5 [131-236]	193.5 [146-246]	.102
SABP (mm Hg)[cont]	126.8 (118.5-135.1; n = 18)	137.4 (126.4-148.3; n = 23)	.363	131.6 (115.8-147.5; n = 14)	141.8 (125.7-157.8; n = 12)	.331

Note: Normally distributed data are presented as mean (95% confidence interval) and nonnormally distributed data as median [range]. The variables are annotated according to their type using superscripts: [cont] for continuous, and [cat/o] for ordinal and [cat/b] for binary categorical variables.
Abbreviations: ATE, aortic thromboembolism; CHF, congestive heart failure; SABP, systolic arterial blood pressure; SAM, systolic anterior motion of the mitral valve.

TABLE 5 Baseline characteristics that were statistically different between cats that gained systolic anterior motion of the mitral valve (SAM) and cats that did not gain SAM during the follow-up period.

Baseline characteristics	Gained SAM at recheck (n = 5)	Unchanged at recheck (n = 17)	*P* value
LVOT Vmax (m/s)	1.6 (0.7-2.4)	1.0 (0.9-1.2)	.017
LVFW (mm)	5.5 (4.8-6.3)	5.9 (5.7-6.4)	.009
IVS/LVFW	1.2 (1.0-1.6)	1.1 (1.0-1.1)	.004

Note: All data are normally distributed and are presented as mean (95% confidence interval).
Abbreviations: IVS, maximum interventricular septum in end-diastole; LVFW, maximum left ventricular free wall thickness in end-diastole; LVOT Vmax, maximum left ventricular outflow tract velocity.

TABLE 6 Variables that were statistically different between cats that gained systolic anterior motion of the mitral valve (SAM) and cats that did not gain SAM during the follow-up period.

% change from baseline to recheck	Gained SAM at recheck (n = 5)	Unchanged at recheck (n = 17)	*P* value
△% LVOT Vmax	+135% (−305 to +574%)	+6.7% (−18.0 to +31.3%)	.012
△% IVS/LVFW	−0.3% (−21.4 to +0.1)	6.5% (−5.2 to +11.2)	.036

Note: All data are normally distributed and are presented as mean (95% confidence interval).
Abbreviations: IVS, maximum interventricular septum in end-diastole; LVFW, maximum left ventricular free wall thickness in end-diastole; LVOT Vmax, maximal left ventricular outflow tract velocity.

TABLE 7 Variables that were statistically different between cats that lost systolic anterior motion of the mitral valve (SAM) and cats that did not lose SAM during the follow-up period.

% change from baseline to recheck	Lost SAM at recheck (n = 7)	Unchanged at recheck (n = 31)	*P* value
△% LVFW	−10% (−18 to +9%)	+5.8% (−1.5 to +9.4%)	.040
△% Loss of DLVOTO[a]	71.4% (56.5-89.7)	22.6% (3.7-71.9)	.032

Note: All data are normally distributed and are presented as mean (95% confidence interval).
Abbreviations: DLVOTO, dynamic left ventricular outflow tract obstruction; LVFW, maximum left ventricular free wall thickness in end-diastole.
[a]95% confidence interval is also reported for the categorical data.

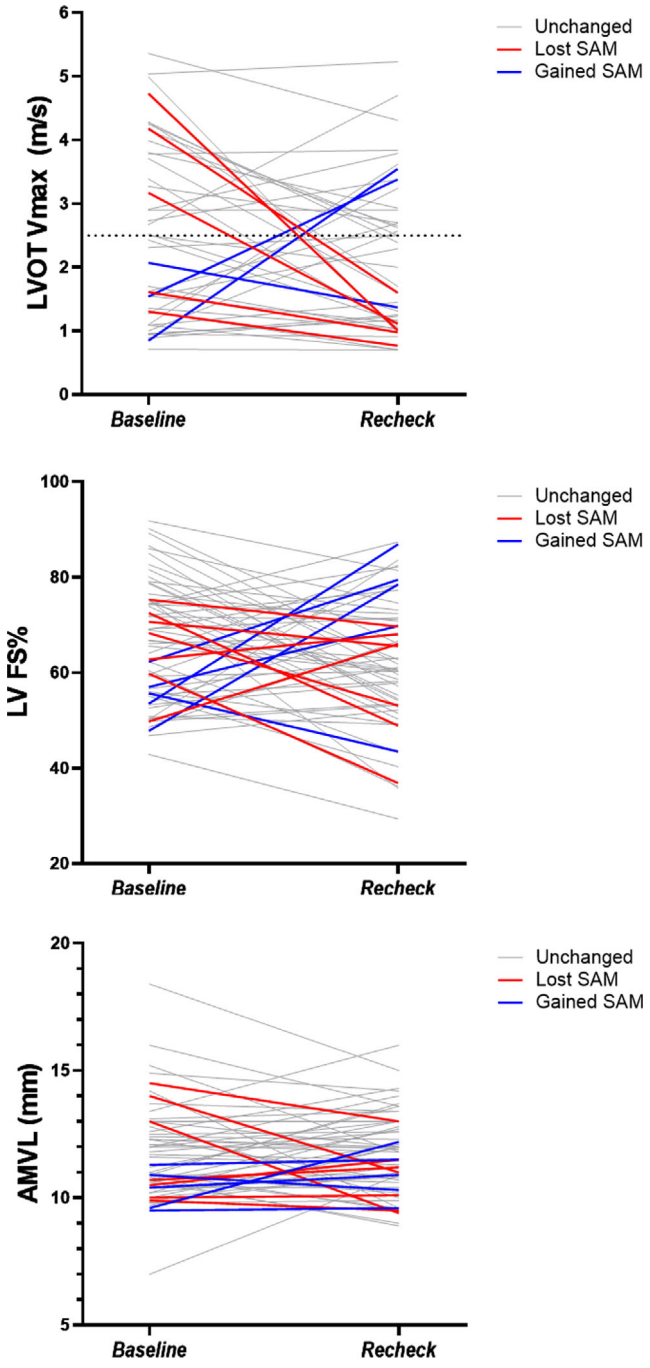

FIGURE 3 Changes of maximal left ventricular outflow tract velocities (LVOT Vmax), anterior mitral valve leaflet (AMVL) length, left ventricular fractional shortening (LV FS%), and age (scan interval). Cats that persistently had SAM at both time points are shown in gray. Cats that have lost SAM at recheck are shown in red. Cats that never had SAM at either time point are shown in light blue. Cats that gained systolic anterior motion of the mitral valve (SAM) at recheck are shown in blue. A horizontal line in LVOT Vmax shows 2.5 m/s, which was used as a cutoff to define dynamic left ventricular outflow tract obstruction.[16] Cats that gained SAM had faster baseline LVOT Vmax than those that did not ($P = .017$).

vascular tone to induce SAM and potentially worsen DLVOTO.[15] For example, a treadmill exercise test can allow detection of patients with latent SAM, where SAM is neither present nor causing DLVOTO at rest.[26,27] Documenting patients with latent SAM is important as they can still develop significant DLVOTO with activity and require therapy. In contrast, there is no standardized method of assessing SAM and DLVOTO in cats with HCM. Furthermore, a clear distinction between SAM and DLVOTO are not always made in the literature. One could consider that the echocardiography in cats is, by itself, a provocative maneuver. However, cats can still display varying degrees of stress in each hospital visit. Provocative maneuvers could not be used in this study as all baseline data and most of the follow up data were collected retrospectively. The authors acknowledge that this is a limitation of this study.

Cats that had faster LVOT Vmax, thinner LVFW, and greater IVS to LVFW ratio at baseline were significantly more likely to develop SAM. These features support the hypothesis that underlying anatomical and functional substrates of the heart predispose to the development of SAM.[12,14,16] No other baseline variables predicted the subsequent development or loss of SAM. This highlights the difficulty of predicting the gain or loss of SAM by a single echocardiogram.

Age, time between the 2 scans, and indices of LV systolic function were not associated with the gain or loss of SAM. This occurred despite the decline in LV systolic function over time in cats with SAM. However, most of these cats were still hyperdynamic in LV systolic function when compared to an established normal reference interval in cats.[28] Hyperdynamic LV systolic function is an important factor for developing SAM in humans and likely cats.[14,29] A longer follow-up period could have documented further reduction in LV systolic function and the number of cats that lose SAM may have been similar to a previous study.[10] Similarly, cats in this study were relatively young at recheck because of a relatively short follow-up period for a disease with a long preclinical period. The presence of SAM is often associated with young cats with HCM.[3,9,21,30] It is possible that these young cats lose SAM as they get older. A study with a longer follow up period is needed to further test this hypothesis.

The presence of SAM is associated with greater LV hypertrophy.[16] In the present study, cats that lost SAM had reduced LVFW thickness. However, it is unknown if the reduction in LVFW thickness was the cause or result of the loss of SAM. Furthermore, a reduction in LV wall thickness was also seen in cats that maintained SAM. Recently, LV thinning was shown to represent intramural myocardial fibrosis in cats with HCM.[24] Regional wall hypokinesis was not common in the present study. It is unclear whether the loss of LV wall thickness is a result of myocardial fibrosis in some cats. Additionally, an increase in LV hypertrophy were seen in both cats that maintained SAM and cats that lost SAM. Increase in LV hypertrophy is an expected disease progression in most cats with HCM.[10]

The lack of standardized protocol for provoking SAM during echocardiography is a limitation of this study. Day-to-day variation in stimulation and adrenergic tone is an important confounder, although echocardiography performed at a veterinary clinic is arguably a provocative maneuver in itself. Second, the relatively short-term follow up period did not allow many cats with advanced heart disease to be included in the study. Third, the sample size in each group was small. The small sample size was the result of strict exclusion criteria at both time points. This was necessary for carefully assessing the presence

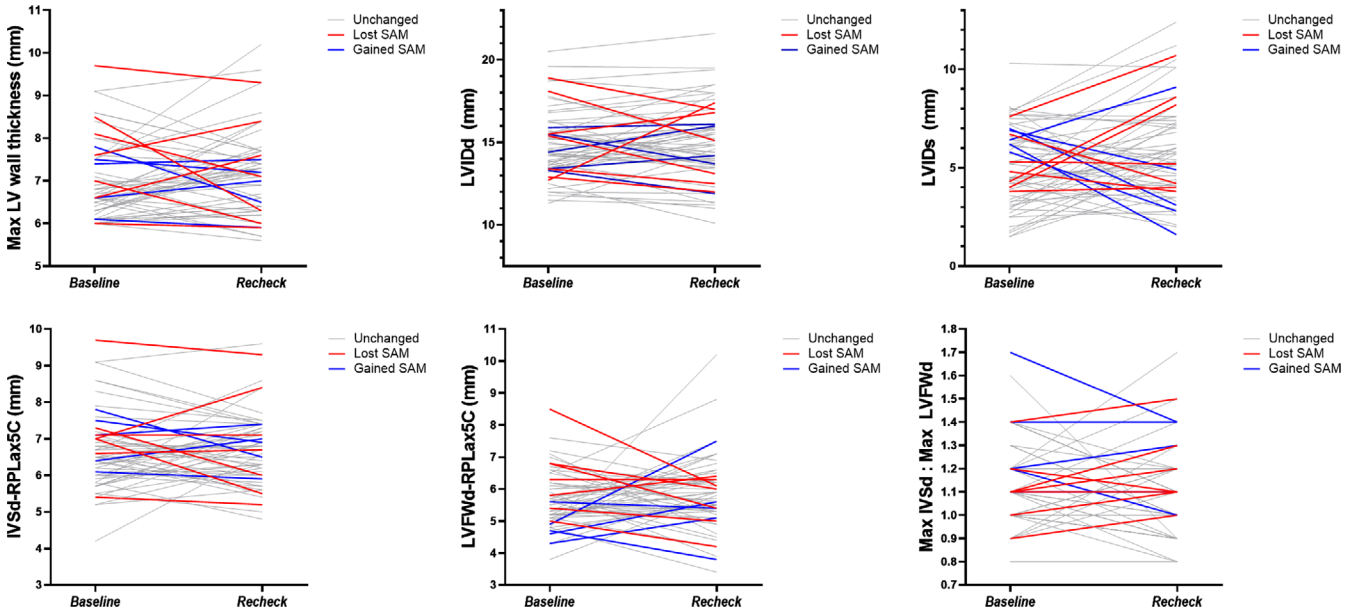

FIGURE 4 Change in maximal left ventricular (LV) wall thickness, LV internal diameters in end-diastole (LVIDd) and end-systole (LVIDs), maximal interventricular septal thickening in right parasternal long axis 5-chamber view (IVSd-RPLAX5C), maximal left ventricular free wall thickness in right parasternal long axis 5-chamber view (LVFWd-RPLAX5C), and a ratio of maximal interventricular septum to maximal left ventricular free wall (Max IVS : Max LVFW). Cats that persistently had systolic anterior motion of the mitral valve (SAM) at both time points are shown in gray. Cats that lost SAM at recheck are shown in red. Cats that never had SAM at either time point are shown in light blue. Cats that gained SAM at recheck are shown in blue. Cats that gained SAM had thinner LVFWd-RPLAX5C ($P = .009$) and a greater Max IVS : Max LVFW at baseline ($P = .004$).

of SAM over time. Nevertheless, some features associated with gain or loss of SAM could have been missed because of type II error. A larger sample size would also allow a multivariable analysis because many anatomical and functional features of HCM are thought to cause SAM.[12] A multivariable analysis would allow identification of most important variables associated with SAM by ranking variables according to their magnitude of effect on the development of SAM. Fourth, the study involved only 2 time points. The presence of SAM may change multiple times in some cats. Lastly, certain anatomical features, including aortic-septal angle, and papillary muscle morphologies were not studied in the present study. Many of these variables would be better assessed by an advanced cardiac imaging such as cardiac magnetic resonance imaging.[14]

The incidence rate was calculated to describe the frequency of gain or loss of SAM while accounting for the inconsistent follow-up period among cats. Interpreting the incidence rate requires an understanding of the interval censoring problem, where the disease or event could occur anywhere in between 2 time points. A common approach to account the interval censoring problem is by using a "midpoint" incidence rate, where the event is assumed to have occurred half-way between the 2 time points.[31] However, this approach could underestimate the incidence near the end of the observation period.[31,32] Alternatively, random-point and Poisson binomial methods have been described to potentially reduce underestimation and bias.[31,32] An ideal approach to account the interval censoring problem was not investigated in the present study.

Interobserver agreement of SAM diagnosis was not 100% in this study. To overcome an observation error, the consensus between 2 board certified cardiologists (J.S. and V.L.F.) was used to diagnose SAM. Nine cineloops (7.5%) needed a review but the final consensus did not increase the number of cats that gained or lost SAM. If both cardiologists made an error on the echocardiographic diagnosis of SAM, the number of cats that gain or lose SAM would further increase.

Lastly, there is an argument that cats with SAM and LV hypertrophy have MV dysplasia instead of HCM. Indeed, cats with SAM and LV hypertrophy have elongated AMVL and changes to the papillary muscles that could suggest MV dysplasia. However, these changes to the AMVL and papillary muscles are a recognized feature of HCM in both cats and humans.[16,20] Based on the results of experimental studies, and longitudinal studies of humans and cats, abnormalities of the MV appear to occur after birth, but before LV hypertrophy.[20,33-36] The argument on the disease definition remains unresolved, but the current literature supports the use of HCM as the disease name in these cats.

5 | CONCLUSION

The incidence rate of cats with HCM to gain or lose SAM was 8 per 100-cat years. Age and scan-interval between 2 time-points were not associated with the gain or loss of SAM. Cats that had faster LVOT Vmax, thinner LVFW, and greater IVS to LVFW ratio at baseline were

significantly more likely to develop SAM. Cats with SAM at baseline had more advanced disease progression than those without SAM at baseline.

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CONFLICT OF INTEREST DECLARATION

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OFF-LABEL ANTIMICROBIAL DECLARATION

Authors declare no off-label use of antimicrobials.

INSTITUTIONAL ANIMAL CARE AND USE COMMITTEE (IACUC) OR OTHER APPROVAL DECLARATION

Approved by the Royal Veterinary College Ethics Committee (URN: M20170143). Ethical approval for data collection in New Zealand was not required as this data was collected retrospectively.

HUMAN ETHICS APPROVAL DECLARATION

Authors declare human ethics approval was not needed for this study.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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