Prevalence of cardiomyopathy in apparently healthy cats

Christopher F. Paige, MS, DVM, DACVIM; Jonathan A. Abbott, DVM, DACVIM; François Elvinger, Dr med vet, PhD, DACVP; R. Lee Pyle, VMD, MS, DACVIM

Objective—To determine the prevalence of cardiomyopathy and the relationship between cardiomyopathy and heart murmurs in apparently healthy cats.

Design—Cross-sectional study.

Animals—103 privately owned, apparently healthy domestic cats.

Procedures—Cats were physically and echocardiographically examined by 2 investigators independently. Left ventricular wall thickness was determined via 2-dimensional echocardiography in short-axis and long-axis planes. Left ventricular hypertrophy was identified when end-diastolic measurements of the interventricular septum or posterior wall were ≥ 6 mm. Cats with left ventricular hypertrophy but without left ventricular dilatation were considered to have hypertrophic cardiomyopathy (HCM). The associations between heart murmurs and Doppler echocardiographic velocity profiles indicative of dynamic ventricular outflow tract obstruction were evaluated.

Results—Heart murmurs were detected in 16 (15.5%; 95% confidence interval, 9.2% to 24.0%) cats; of these, 5 had cardiomyopathy. Cardiomyopathy was also identified in 16 (15.5%; 95% confidence interval, 9.2% to 24.0%) cats; 15 had HCM, and 1 had arrhythmogenic right ventricular cardiomyopathy. Of the cats with HCM, 11 had segmental left ventricular hypertrophy, 3 had diffuse left ventricular hypertrophy, and 1 had borderline left ventricular hypertrophy with marked systolic anterior motion of the mitral valve. Sensitivity and specificity of auscultatory detection of a heart murmur for diagnosing cardiomyopathy were 31% and 97%, respectively. Echocardiographic evidence of late systolic acceleration within ventricular outflow tracts was associated with the existence of a heart murmur.

Conclusions and Clinical Relevance—Cardiomyopathy was common in the healthy cats evaluated in this study. In apparently healthy cats, detection of a heart murmur is not a reliable indicator of cardiomyopathy. (J Am Vet Med Assoc 2009;234:1398–1403)
Web that was used to identify cats meeting the inclusion criteria and collect zoographic data. Cats were considered healthy when they had not already undergone an echocardiographic examination, were not receiving treatment for cardiovascular disease, and did not have a history of chronic illness such as inflammatory bowel disease, hyperthyroidism, renal disease, systemic hypertension, or diabetes mellitus. Cats were excluded when veterinary care had been sought for a systemic illness in the previous 3 months. Cats that had a history of a heart murmur but had not been examined echocardiographically were included. The study protocol was approved by the Animal Care and Use Committee and Institutional Review Board of Virginia Polytechnic Institute.

**Study procedures**—Physical examination, Doppler arterial blood pressure estimation, ECG, and then echocardiography were performed in that sequence in 3 rooms. The physical and echocardiographic examinations were performed by 2 board-certified veterinary cardiologists (RLP and JAA); the echocardiographer (JAA) was unaware of the physical examination findings. Echocardiographic images were digitally recorded for subsequent quantitative and qualitative analysis. After digital echocardiographic records were randomized and cat identifiers were concealed, echocardiographic measurements were obtained by a third investigator (CFP). The final echocardiographic diagnosis consisted of the consensus opinion of the echocardiographer and the investigator (CFP) that performed the echocardiographic measurements and was determined without knowledge of the physical examination findings.

**Physical examination**—All cats underwent a systematic, dynamic cardiac auscultatory examination. Auscultation was first performed when cats were at rest and then after provocation (a maneuver in which the examiner quickly lifted the cat in the air at least twice). When identified, heart murmurs were described in terms of intensity, which was graded on a 6-point scale according to the recommendations of Levine, point of maximal intensity, and timing. The existence or lack of a gallop sound was recorded, as was a description of the cardiac rhythm. Heart rate was recorded for all cats.

**Echocardiography**—Echocardiographic examinations were performed without chemical restraint. A sonograph with a 7.5-MHz transducer was used to perform transthoracic echocardiography as described elsewhere. Two-dimensional short-axis and long-axis right parasternal images of the left ventricle were used to measure end-diastolic thickness of the interventricular septum and posterior wall. In the short-axis plane, end of diastole was defined by the onset of the QRS complex in a simultaneously recorded ECG. However, some cats did not tolerate ECG monitoring, and in other situations, motion artifact obscured the onset of the QRS complex. For those examinations, the maximal diastolic excursion of the ventricle was used to define end of diastole. In the long-axis plane, the end of diastole was defined as the first frame in which mitral valve closure was visible. The dimensions LVIDd, IVSd, and LVPWd were obtained from the short-axis image.

From the short-axis image, the IVSd was also measured in 2 additional sites. Specifically, the maximal thickness of the septum was measured in the 2 septal segments that extended from the papillary muscles to the central point of the septum (Figure 1). The line of measurement was parallel to a chord that extended through the centroid of the ventricular lumen (Figure 2). End-diastolic basilar septal thickness was measured in the long-axis plane; the greatest septal dimension between the aortic root and the point at which the anterior mitral valve leaflet most closely approached the interventricular septum during diastole was recorded (Figure 3). Septal measurements included left ventricular
endocardial echoes, but excluded echoes arising from the right ventricular endocardium. Measurements of the LVPW included endocardial echoes but did not include the pericardium.

The left atrial and aortic root diameters were determined from M-mode images, with the M-mode beam directed by use of right parasternal short-axis 2-D images. A ratio of these 2 values was calculated, and left atrial enlargement was defined as a ratio > 1.54. The cats were also evaluated by means of conventional color flow, pulsed-wave, and sometimes continuous spectral Doppler echocardiographic examination. Left and right ventricular outflow tract velocities were recorded. Echocardiographic dimensions and spectral Doppler echocardiographic velocities were calculated as the mean of 3, usually consecutive, cardiac cycles.

Hypertrophic cardiomyopathy was echocardiographically defined by an end-diastolic wall thickness ≥ 6 mm that affected > 50% of any region of the interventricular septum or LVPW. The existence or lack of systolic anterior motion of the mitral valve was also detected. Diffuse left ventricular hypertrophy was diagnosed when abnormal wall thickness was identified in at least 3 wall segments. Other types of cardiomyopathy in cats were classified as described elsewhere. Dynamic RVOT obstruction was defined as detection of a systolic jet that originated proximal to the pulmonary valve and had spectral Doppler echocardiographic characteristics that indicated late-systolic acceleration.

Arterial blood pressure measurement and electrocardiography—Systemic arterial blood pressure was estimated for all cats by use of the Doppler cuff–flow method. All estimates were obtained from the left or right forelimb. Cats were considered hypertensive when the mean of 3 consecutive arterial blood pressure measurements was ≥ 180 mm Hg. After systemic arterial blood pressure was measured, cats were restrained in right lateral recumbency and a 6-lead ECG was recorded. Mean ECG heart rate was determined from the 6-lead ECG only, whereas the existence of arrhythmias was determined from the 6-lead ECG or the ECG that was recorded during echocardiography.

Thyroid gland function—After completion of the cardiovascular examination, a blood sample was obtained via jugular venipuncture from all cats that were ≥ 6 years old. Blood samples were centrifuged, and serum was harvested and refrigerated at 4°C until analyzed. Serum thyroxine concentration was determined by use of a commercial assay and an automated chemistry analyzer. Cats with a serum thyroxine concentration that exceeded the upper limit of the laboratory reference range were considered hyperthyroid and excluded from additional analysis.

Statistical analysis—Prevalence of murmurs and cardiomyopathy and the corresponding 95% CIs were calculated by use of statistical software. Associations between detection of a heart murmur and dynamic ventricular outflow tract obstruction were assessed with the \( \chi^2 \) test, and prevalence odds ratios were calculated. Sensitivity, specificity, positive and negative predictive values, positive and negative likelihood ratios, and respective 95% CIs were calculated by use of other software to determine the diagnostic usefulness of cardiac auscultation for identifying cardiomyopathy. Echocardiographic detection of cardiomyopathy was considered the gold standard with which auscultation was compared, and auscultatory detection of an unprovoked heart murmur was considered a positive test result. Continuous data are reported as mean ± SD. A value of \( P < 0.05 \) was considered significant for all analyses.

Results

Animals—One hundred sixty-five people responded to the electronic survey, and 145 cats were identified as meeting the initial inclusion criteria. On 6 nonconsecutive days from late August 2005 to November 2005, 132 cats were examined. Twenty-nine of these cats were later excluded because they resisted manual restraint (n = 22), were hyperthyroid (4), or had an incomplete echocardiographic examination (3). Therefore, 103 apparently healthy cats were included in the study. Forty-three cats were female. Mean ± SD body weight was 5.01 ± 1.13 kg (11.02 ± 2.50 lb). Precise ages were not always available, so the following discrete age categories were used instead: < 1 year (n = 6), 1 to 5 years (62), 6 to 10 years (27), 11 to 15 years (7), and ≥ 16 years (1). Most cats were of mixed breed (75 domestic shorthair, 9 domestic medium hair, and 10 domestic longhair). Purebred cats included Himalayan (n = 3), Siamese (3), Ocicat (2), and Maine Coon (1).

Physical examination—Heart murmurs were detected in 16 (15.5%; 95% CI, 9.2% to 24.0%) cats. Grades of heart murmur intensities were distributed as follows: grade 1/6 (n = 5), grade 2/6 (9), and grade 3/6 (2). After the provocative maneuver, heart murmurs
were detected in 28 cats, 13 of which did not have a heart murmur at rest. One cat had a heart murmur at rest but not after the provocative maneuver. Based on results of auscultation, the mean heart rate of the 103 cats was 173.9 ± 16.4 beats/min. Additional cardiac abnormalities identified through auscultation were a gallop rhythm in 2 cats and bradycardia in another.

Arterial blood pressure and ECG—All cats were normotensive, and systolic arterial blood pressure in 87 echocardiographically normal cats was 131.1 ± 17.8 mm Hg. In 15 cats with HCM, systolic arterial blood pressure was 136.4 ± 19.6 mm Hg. Based on results of electrocardiography, the heart rate of 80 cats without cardiomyopathy was 189.4 ± 23.8 beats/min and that for cats with HCM was 188.7 ± 27.2 beats/min. Electrocardiography or echocardiography revealed ECG abnormalities in 7 cats without cardiomyopathy. Two of these cats had ventricular preexcitation, 4 had ventricular premature complexes, and 1 had ventricular tachycardia. Of the cats with cardiomyopathy, 2 with HCM had ventricular premature complexes. In addition, a cat with arrhythmogenic right ventricular cardiomyopathy also had ventricular tachycardia.

Echocardiography—Cardiomyopathy was identified in 16 (15.5%; 95% CI, 9.2% to 24.0%) cats; 15 had HCM, and 1 had arrhythmogenic right ventricular cardiomyopathy. Of the 15 cats with HCM, 11 had segmental left ventricular hypertrophy, and in 3 of these cats, hypertrophy was localized to the basilar septum. The magnitude and distribution of ventricular hypertrophy in the cats with HCM varied among cats (Table 1). Of the cats with basilar septal hypertrophy, 1 cat was between 1 and 5 years of age and 2 were > 10 years of age. Three cats had diffuse left ventricular hypertrophy. One cat with marked systolic anterior motion of the mitral valve and a maximal ventricular wall thickness of 5.9 mm was classified as having HCM. The cat with a diagnosis of arrhythmogenic right ventricular cardiomyopathy had mild right ventricular enlargement, abnormal septal motion, an end-systolic left ventricular diameter of 11.9 mm, tricuspid valve regurgitation, ventricular tachycardia, and an intermittent gallop sound.

Dynamic RVOT obstruction with a peak velocity ≥ 1.7 m/s was identified in 5 cats, and 3 other cats had lower velocities with evidence of late systolic acceleration. Dynamic LVOT obstruction was identified in 8 cats. Two cats with HCM had systolic anterior motion of the mitral valve with supraphysiologic LVOT velocities (4.2 and 2.1 m/s) and mitral valve regurgitation and 2 had mild left ventricular obstruction and supraphysiologic velocities (1.8 and 1.7 m/s), whereas the remaining 4 had late systolic acceleration with lower velocities (1.0 m/s [n = 2], 1.1 m/s [1], and 1.6 m/s [1]). Of the 6 cats with dynamic LVOT obstruction in which systolic anterior motion of the mitral valve was not detected, 2 had HCM and 4 did not. Two cats, 1 with and 1 without HCM, had right and left dynamic ventricular outflow tract obstructions. Trace or mild tricuspid valve regurgitation was detected in 7 cats, and 1 of these also had slight mitral valve regurgitation. In 1 cat that was classified as free of cardiomyopathy, echocardiography revealed an equivocally enlarged right ventricle, slight tricuspid valve regurgitation, and brief paroxysm of nonsustained ventricular tachycardia.

All evaluated cats had normal left atrial size. In 1 cat without cardiomyopathy, the M-mode image did not include 3 measurable cardiac cycles, but size of the left atrium was unremarkable in 2-D short-axis and long-axis views. In 3 additional cats also free of cardiomyopathy, the M-mode ratio of the left atrial and aortic root diameters marginally exceeded 1.54. However, the M-mode–derived end-systolic dimension of the left atrium did not exceed 1.45 cm, and left atrial size was unremarkable in the 2-D short-axis and long-axis views.10

Association between heart murmurs and dynamic outflow tract obstruction—Of the 16 cats with heart murmurs, 5 had HCM. Six of 16 cats with unprovoked

<table>
<thead>
<tr>
<th>Cat</th>
<th>Wt (kg)</th>
<th>LVIDd (cm)</th>
<th>LVES (cm)</th>
<th>IVSd (cm)</th>
<th>LVPWd (cm)</th>
<th>IVS1 (cm)</th>
<th>IVS2 (cm)</th>
<th>IVSb (cm)</th>
<th>Ao (cm)</th>
<th>LA (cm)</th>
<th>LA:Ao</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>6.03</td>
<td>1.25</td>
<td>0.24</td>
<td>0.44</td>
<td>0.61*</td>
<td>0.42</td>
<td>0.61*</td>
<td>0.69*</td>
<td>1.07</td>
<td>1.45</td>
<td>1.35</td>
</tr>
<tr>
<td>2</td>
<td>7.59</td>
<td>1.68</td>
<td>0.62</td>
<td>0.57</td>
<td>0.68*</td>
<td>0.47</td>
<td>0.37</td>
<td>0.47</td>
<td>1.14</td>
<td>1.32</td>
<td>1.15</td>
</tr>
<tr>
<td>3</td>
<td>4.31</td>
<td>1.49</td>
<td>0.61</td>
<td>0.46</td>
<td>0.38</td>
<td>0.60*</td>
<td>0.44</td>
<td>0.84*</td>
<td>0.84</td>
<td>1.14</td>
<td>1.36</td>
</tr>
<tr>
<td>4</td>
<td>4.46</td>
<td>1.68</td>
<td>0.59</td>
<td>0.57</td>
<td>0.67*</td>
<td>0.76*</td>
<td>0.49</td>
<td>0.74*</td>
<td>1.33</td>
<td>1.57</td>
<td>1.18</td>
</tr>
<tr>
<td>5</td>
<td>6.66</td>
<td>1.36</td>
<td>0.56</td>
<td>0.53</td>
<td>0.45</td>
<td>0.52</td>
<td>0.51</td>
<td>0.64*</td>
<td>0.82</td>
<td>1.39</td>
<td>1.52</td>
</tr>
<tr>
<td>6</td>
<td>9.80</td>
<td>1.45</td>
<td>0.52</td>
<td>0.52</td>
<td>0.61*</td>
<td>0.45</td>
<td>0.44</td>
<td>0.42</td>
<td>1.04</td>
<td>1.45</td>
<td>1.40</td>
</tr>
<tr>
<td>7†</td>
<td>6.15</td>
<td>1.15</td>
<td>0.2</td>
<td>0.59</td>
<td>0.58</td>
<td>0.56</td>
<td>0.56</td>
<td>0.58</td>
<td>1.03</td>
<td>1.24</td>
<td>1.21</td>
</tr>
<tr>
<td>8</td>
<td>4.66</td>
<td>1.62</td>
<td>0.38</td>
<td>0.37</td>
<td>0.33</td>
<td>0.47</td>
<td>0.56</td>
<td>0.71*</td>
<td>0.95</td>
<td>1.36</td>
<td>1.43</td>
</tr>
<tr>
<td>9</td>
<td>4.23</td>
<td>1.04</td>
<td>0.27</td>
<td>0.40</td>
<td>0.59</td>
<td>0.46</td>
<td>0.41</td>
<td>0.61*</td>
<td>1.07</td>
<td>1.10</td>
<td>1.03</td>
</tr>
<tr>
<td>10</td>
<td>4.28</td>
<td>1.26</td>
<td>0.31</td>
<td>0.64*</td>
<td>0.42</td>
<td>0.54</td>
<td>0.51</td>
<td>0.71*</td>
<td>0.82</td>
<td>1.14</td>
<td>1.24</td>
</tr>
<tr>
<td>11</td>
<td>4.29</td>
<td>1.18</td>
<td>0.44</td>
<td>0.39</td>
<td>0.61*</td>
<td>0.53</td>
<td>0.38</td>
<td>0.37</td>
<td>0.89</td>
<td>1.08</td>
<td>1.21</td>
</tr>
<tr>
<td>12</td>
<td>5.10</td>
<td>0.88</td>
<td>0.11</td>
<td>0.50</td>
<td>0.60*</td>
<td>0.53</td>
<td>0.51</td>
<td>0.71*</td>
<td>0.92</td>
<td>1.25</td>
<td>1.35</td>
</tr>
<tr>
<td>13</td>
<td>7.03</td>
<td>1.35</td>
<td>0.55</td>
<td>0.54</td>
<td>0.61*</td>
<td>0.68*</td>
<td>0.57</td>
<td>0.59</td>
<td>0.89</td>
<td>1.36</td>
<td>1.53</td>
</tr>
<tr>
<td>14</td>
<td>4.82</td>
<td>1.14</td>
<td>0.36</td>
<td>0.48</td>
<td>0.50</td>
<td>0.61*</td>
<td>0.52</td>
<td>0.48</td>
<td>0.93</td>
<td>1.38</td>
<td>1.48</td>
</tr>
<tr>
<td>15</td>
<td>4.54</td>
<td>1.25</td>
<td>0.38</td>
<td>0.68*</td>
<td>0.61*</td>
<td>0.53</td>
<td>0.37</td>
<td>0.61*</td>
<td>0.79</td>
<td>1.14</td>
<td>1.45</td>
</tr>
</tbody>
</table>

*Indicated value is ≥ 6 mm, indicating the existence of HCM. This cat had a diagnosis of HCM on the basis of values that were close to the 6-mm cutoff and detection of marked systolic anterior motion of the mitral valve.

Wt = Body weight; LVES = Left ventricular wall thickness at end-systole; IVS1 = Thickness of the interventricular septum segment 1, IVS2 = Thickness of the interventricular septum segment 2, IVSb = Thickness of the basilar septum, Ao = Aortic diameter at end-diastole, LA = Left atrial diameter at end-systole, LA:Ao = Ratio of left atrial diameter to aortic diameter.

Hypertrophic cardiomyopathy was echocardiographically defined by an end-diastolic wall thickness ≥ 6 mm that affected > 50% of any region of the interventricular septum or LVPW.
heart murmurs had a dynamic ventricular outflow tract obstruction: 3 cats without HCM had RVOT obstruction only, 1 cat with HCM had RVOT and LVOT obstructed, and 2 cats with HCM had LVOT obstruction and mitral regurgitation associated with systolic anterior motion of the mitral valve. The other 10 cats had no evidence of a dynamic ventricular outflow tract obstruction; two of these cats had HCM. On the other hand, 10 of 85 (12%) cats without an unprovoked heart murmur had a dynamic ventricular outflow tract obstruction, whereas the other 75 (88%) did not.

Ten of the 28 (36%) cats with heart murmurs detected after provocation had Doppler echocardiographic evidence of dynamic ventricular outflow tract obstruction. The other 18 (64%) cats with provoked heart murmurs had no dynamic ventricular outflow tract obstruction. Of the cats with heart murmurs detected after provocation, 5 without HCM had RVOT obstruction only, 1 with HCM and 1 without had LVOT only, 1 with HCM had RVOT and LVOT obstructions, and 2 with HCM had LVOT obstruction and mitral valve regurgitation associated with systolic anterior motion of the mitral valve. The 6 cats that had a heart murmur at rest and dynamic ventricular outflow tract obstruction also had a heart murmur after provocation. On the other hand, 6 of 72 (8%) cats without a provoked heart murmur had a dynamic ventricular outflow tract obstruction, whereas the other 66 (92%) did not.

The odds that a cat with an unprovoked heart murmur had a dynamic ventricular outflow tract obstruction as detected via Doppler echocardiography were 4.5 times as high as the odds that a cat without an unprovoked heart murmur had the same problem (95% CI, 1.3 to 15.1; P = 0.01; n = 101). The odds of a cat with a provoked heart murmur having a dynamic ventricular outflow tract obstruction were 6.1 times as high as the odds of a cat without a provoked heart murmur having the same problem (95% CI, 2.0 to 19.1; P < 0.001; n = 100).

Diagnostic usefulness of auscultation for detecting cardiomyopathy—Comparison of results of auscultation with those of echocardiographic indicated that auscultation of an unprovoked heart murmur had poor sensitivity but moderate specificity in the detection of cardiomyopathy (Table 2). The P values for the likelihood ratios were not significant. Given the prevalence of cardiomyopathy in the sample as determined via echocardiography (15.3%), the probability that a cat with an unprovoked heart murmur would have cardiomyopathy (ie, the positive predictive value of the test) was only 31% (95% CI, 9% to 54%). In addition, 13% of cats without unprovoked heart murmurs would have cardiomyopathy (negative predictive value, 87%; 95% CI, 80% to 94%).

Table 2—Results for the evaluation of auscultation of unprovoked heart murmurs as a means of detecting cardiomyopathy in 103 apparently healthy cats, with echocardiography used as the gold standard.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prevalence of cardiomyopathy (%)</td>
<td>15.5</td>
<td>9.2–24.0</td>
</tr>
<tr>
<td>Sensitivity (%)</td>
<td>31.3</td>
<td>8.5–54.0</td>
</tr>
<tr>
<td>Specificity (%)</td>
<td>87.4</td>
<td>80.4–94.3</td>
</tr>
<tr>
<td>Positive likelihood ratio</td>
<td>2.5</td>
<td>1.0–6.2</td>
</tr>
<tr>
<td>Negative likelihood ratio</td>
<td>0.8</td>
<td>0.6–1.1</td>
</tr>
</tbody>
</table>

Discussion

In the present study, the relationship between auscultatory abnormalities and echocardiographic findings was evaluated in a sample of apparently healthy cats that were prospectively enrolled. Results indicated that the prevalence of subclinical cardiomyopathy in the population of nonreferred cats in the area (Southwest Virginia) was approximately 16%; most affected cats had HCM, of which approximately a third had heart murmurs. Only 5 of 16 cats with heart murmurs had cardiomyopathy.

Other researchers have evaluated epidemiologic characteristics of cardiomyopathy in cats, but many of these studies involved referral-based populations or excluded cats with heart murmurs. One group reported a 21% prevalence of heart murmurs in 103 overtly healthy cats. However, cats that had a heart murmur or history of cardiac disease were excluded from that study; therefore, that prevalence estimate cannot be directly compared with our findings. Because we included cats on the basis of apparent health status, we believe we obtained a better estimate of the true prevalence within the target population (apparently healthy cats). Importantly, our study design removed the bias that would have been introduced by an echocardiographer attempting to identify the source of a heart murmur.

Results of the present study indicated that HCM in cats is more prevalent than it is in humans (estimated prevalence, 0.2%). This prevalence of HCM in apparently healthy cats was high, but to our knowledge, there are no similar data with which to compare our findings. It is important to mention that M-mode echocardiography, which has been used in many studies of HCM in cats, is likely to be less sensitive than 2-D echocardiography for detection of hypertrophy, as is true in people with HCM. Most (11/15) cats with HCM had segmental left ventricular hypertrophy. The cats identified with HCM were apparently mildly affected. The degree of hypertrophy was not marked and was generally segmental; furthermore, none of the cats had echocardiographic evidence of atrial enlargement. The high prevalence of HCM in apparently healthy cats was consistent with the diversity of this disorder in humans. In the past, the clinical impact of HCM in humans and perhaps in cats has been exaggerated because data have typically been obtained from referral facilities. In humans, HCM is a genetic disorder that is associated with diverse phenotypic expression and a variable clinical course. In Maine Coon and Ragdoll breeds of cats, HCM is heritable and the associated genetic mutations have been reported. Familial HCM has been identified in other breeds of cats; therefore, it is possible that HCM is generally a genetic disorder in cats.

Abnormal auscultatory findings often prompt an echocardiographic examination, which is the next logical diagnostic step when cardiomyopathy is suspected in cats. However, retrospective studies have revealed that some cats with cardiomyopathy do not have heart murmurs. In the present study, 11 of 16 cats with cardiomyopathy did not have heart murmurs. Sensitivity and specificity as relevant to the present study characterized the proportion of cats with or without cardiomyopathy that were identified by the presence or absence a heart murmur, respectively. Interestingly, only 31% of cats with cardiomyopathy had heart murmurs, although it may be relevant that both cats that had hypertrophic obstructive cardiomyopathy associated with systolic an-
terior motion of the mitral valve also had heart murmurs. Because the sensitivity was low, use of auscultation as a diagnostic screening test for cardiomyopathy would likely yield a high proportion of false-negative test results.20

The calculation of likelihood ratios is another approach to evaluate the usefulness of a diagnostic test.21 In the present study, neither likelihood ratio was significant. Considered with the other measurements of diagnostic accuracy, auscultation of heart murmurs in healthy cats does not usefully discriminate between those with cardiomyopathy and those without.

In general, diagnostic tests that effectively screen populations for disease have high sensitivity. When detection of a heart murmur was considered a positive test result, cardiac auscultation had low sensitivity and moderate specificity for detection of cardiomyopathy. In this regard, auscultation had limitations as a diagnostic test for cardiomyopathy.

The association between abnormal ventricular outflow tract velocities and heart murmurs was evaluated through the calculation of prevalence odds ratios.22 In our study, cats with Doppler echocardiographic evidence of dynamic ventricular outflow tract obstruction were 4.5 times as likely to have a heart murmur at rest and 6 times as likely to have a heart murmur after provocation, compared with cats that did not have obstruction. Because the study was cross-sectional, we could not infer a causal association between echocardiographic findings and the existence of a heart murmur. However, we could conclude that cats with Doppler echocardiographic evidence of abnormal ventricular outflow velocity were more likely to have a heart murmur at rest and after provocation. Heart murmurs in cats are reportedly labile and vary in intensity with heart rate.11 Because auscultation was not performed during the echocardiogram, it is plausible that some, if not all, cats with dynamic ventricular outflow tract obstruction could develop a heart murmur in certain conditions.

Results of the present study must be interpreted in the context of the study limitations. The CIs for the descriptive statistics were broad, and this was a reflection of the sample size, which was chosen on the basis of practicality. The cats enrolled in the study were pets; therefore, it was not possible to confirm the diagnosis of cardiomyopathy by use of postmortem examination. Several (n = 22) cats resisted manual restraint, and exclusion of these cats may have influenced the prevalence results. Furthermore, although we attempted to echocardiographically examine each cat in a consistent and systematic manner, some cats did not tolerate ECG monitoring or, in other situations, motion artifact obscured the onset of the QRS complex in the ECG. For those examinations, the maximal diastolic excursion of the left ventricle and not the onset of the QRS complex was used to define the end of diastole. Although the magnitude of the difference between the 2 methods was likely small, this inconsistency constitutes a minor limitation of our work. Finally, a few cats had isolated basilar septal hypertrophy, and a change in aortic septal angle (i.e., the development of a so-called sigmoid septum) could have resulted in an artifactual appearance of septal hypertrophy.23

References