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Systolic Anterior Motion and Dynamic Right Ventricular Outflow Obstruction as Leading Causes of Murmurs in 856 Clinically Examined Cats

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ABSTRACT

Cardiac auscultation remains a key diagnostic approach for detecting potential heart disease. While multiple studies have assessed murmur prevalence in cats, little is known about the precise origin of the blood flow disturbances causing these murmurs. This study aimed to assess both the prevalence and clinical implications of heart murmurs identified during routine examinations in cats. Clinical records and echocardiographic data from feline patients evaluated for heart murmurs were retrospectively analyzed. Complete clinical information was available for 856 cats. The underlying cause of the murmur was identified in 93.1% of cases—72.3% with a single source of turbulence, 26.4% with two, and 1.3% with three. The most common cause was systolic anterior motion of the mitral valve (SAM) (39.2%), followed by dynamic right ventricular outflow tract obstruction (DRVOTO) (32%) and physiological (flow) murmurs (6.9%). Over half of the cats (56.7%) with murmurs exhibited no detectable structural heart abnormality. Findings suggest that specific murmur characteristics—such as timing, intensity, and point of maximal intensity—can serve as useful indicators for identifying underlying heart disease.

Keywords: Feline, Heart murmur, Cardiology, SAM, DRVOTO, Congenital

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Introduction

In human medicine, heart murmurs are the leading cause of cardiology referrals, with approximately 50–70% being benign, particularly in children [1]. In feline practice, heart murmurs are likewise a frequent reason for specialist referral, accounting for roughly 50% of all cardiology cases at our institutions. Similar to human findings, many murmurs detected in seemingly healthy cats are not associated with any cardiac pathology, with up to 50% of affected cats showing no structural heart disease [2, 3]. However, most feline studies have focused on identifying underlying structural disorders—such as cardiomyopathies or ventricular hypertrophy—rather than pinpointing the exact source of turbulent blood flow [2–7], though a few attempts have been made to determine murmur etiology more precisely [2, 6].

Accordingly, this retrospective study aimed to establish the prevalence of heart murmurs found during standard clinical examinations in cats, emphasizing the echocardiographic determination of the precise flow disturbance responsible. Additionally, it sought to identify potential links between murmur and patient characteristics that could predict the presence of congenital or acquired cardiac disease.

We hypothesized that, consistent with prior studies [2, 3], most cats with an auscultated heart murmur would show no structural cardiac abnormality on echocardiography, and that certain patient variables (age, breed, sex, weight) or murmur attributes (timing, grade, PMI, dynamic or intermittent behavior) might predict the presence of heart disease.

Materials and Methods

Data collection

Clinical and echocardiographic records from cats examined between June 2009 and June 2022 for murmur investigation at two centers—Specialist Veterinary Cardiology Consultancy (SVCC Ltd., Four Marks, Hampshire, UK) and The Ralph Veterinary Referral Centre (TRVRC, Marlow, Buckinghamshire, UK)—were retrospectively reviewed. Extracted data included examination date, signalment, murmur details, echocardiographic evidence of flow turbulence (single or multiple), cardiac measurements, structural abnormalities, ECG results, and whether sedation was administered before imaging. Cats lacking complete clinical descriptions or full echocardiographic datasets were excluded.

Heart murmur description

Each murmur was classified by timing (systolic, diastolic, continuous, or to-and-fro), intensity (Levine scale 1–6) [8], and point of maximal intensity (PMI)—categorized as left or right parasternal, sternal/bilateral, left base, or left apex. Murmurs that varied in strength were defined as dynamic, while those inconsistently heard were labeled intermittent. All auscultations were conducted by the attending cardiologists (LF or HF) in a quiet examination room, prior to echocardiography, using the same stethoscope model (Littmann Classic II Pediatric, 3M, Maplewood, MN, USA).

Echocardiographic examination

Echocardiographic assessments were undertaken by a diplomate in cardiology (LF) or an RCVS-certified veterinary cardiologist (HF). Examinations employed either an Esaote MyLab 30 Gold Cardiovascular, MyLab Omega (MyLab, Esaote S.p.A., Genoa, Italy) at SVCC Ltd., or an Esaote MyLab X8 (MyLab, Esaote S.p.A.) at TRVRC Genoa, Italy. The probe used was a phased-array transducer of 7.0–10.0 MHz or 2.0–9.0 MHz frequency, selected according to the cat's body size. Each scan was re-evaluated later with specialized offline software (MyLab Desk and MyLab Desk Evo, Esaote S.p.A., Genoa, Italy) to confirm the accuracy of blood-flow disturbance recognition and to validate the final interpretation.

A complete transthoracic study—covering two-dimensional, M-mode, and Doppler (color and spectral) analyses—was carried out using standard imaging windows [9]. Cats were calmly positioned in both right and left lateral recumbencies on a cushioned echocardiography table; their forelimbs were placed slightly forward to allow access between the ribs. A small area between the fourth and fifth intercostal spaces was clipped and coated with ultrasound gel to optimize visualization. Sedation was administered only when an animal was uncooperative. Continuous ECG recording was achieved using soft clips attached near or below the elbows and the patellae, with ultrasound gel serving as the conductive interface [10]. Whenever feasible, image collection and measurements occurred during a stable sinus rhythm.

Left-ventricular dimensions were determined from right-parasternal short-axis views at the papillary-muscle level, using high-frame-rate B-mode cine loops. For this work, left-ventricular hypertrophy was defined as an interventricular septum or left-ventricular free wall diastolic thickness greater than 6.0 mm (measured from leading-to-trailing and leading-to-leading edges, respectively), excluding regions with false tendons, focal thickening, or localized echogenicity [11, 12]. Left-atrial diameter (LA) was compared with aortic diameter on right-parasternal short-axis views obtained at the heart base in early diastole—the first frame following aortic-valve closure. An LA-to-aortic ratio ≥ 1.5 was classified as enlargement. Each parameter was averaged across three sequential cardiac cycles. The source of a murmur was identified first by color-flow turbulence and then confirmed by spectral Doppler.

Diagnostic criteria

Systolic anterior motion of the mitral valve (SAM)

SAM was diagnosed when the septal mitral leaflet moved toward the interventricular septum during systole, evident on a right-parasternal short-axis M-mode image at mitral valve level. This was accompanied by left-ventricular outflow turbulence, mid-systolic mitral regurgitation forming a characteristic "double-jet," and a narrow dagger-shaped velocity pattern on spectral Doppler of the LV outflow tract [13–16]. Cases showing systolic displacement of the chordae tendineae were also categorized here [17]. Inducible SAM denoted a transient outflow obstruction triggered by sympathetic activation caused by a sudden increase in Doppler sound volume from the ultrasound speakers (provocative maneuver).

Dynamic right-ventricular outflow-tract obstruction (DRVOTO)

DRVOTO was identified as systolic aliasing on color Doppler within the right ventricle, immediately cranial to the tricuspid valve, extending into the outflow tract. The Doppler spectrum displayed dispersion and a scimitar-like contour, consistent with previous reports [18, 19]. The inducible form was produced by applying gentle probe pressure over the right parasternal area, generating a temporary, mechanically induced outflow narrowing [18].

Flow murmurs

Flow, or "innocent," murmurs were characterized by a soft, audible murmur during auscultation in the absence of detectable functional or structural cardiac abnormalities on echocardiography [20, 21].

Mitral and tricuspid valve regurgitation and stenosis

Regurgitation of the mitral or tricuspid valves was confirmed by recognizing systolic turbulent flow directed respectively toward the left or right atrium, verified in left-apical views with spectral Doppler assessment. Conversely, mitral stenosis was indicated by diastolic turbulence aimed toward the left ventricle and a lengthened E-wave deceleration time on inflow Doppler tracings [22].

Mid-Left-ventricular outflow obstruction

Mid-left-ventricular outflow obstruction (also termed mid-cavitary obstruction) was defined as systolic turbulence originating in the mid-portion of the left ventricle, linked to localized mid-septal hypertrophy and abnormal attachment of papillary muscles or chordae tendineae in cats showing left-ventricular hypertrophy [23, 24].

Pulmonic and aortic stenosis and insufficiency

Unless specified otherwise, pulmonic stenosis (PS) and aortic stenosis (AS) were classified as valvular in origin. Diagnosis was confirmed through the presence of partly fused valve cusps and an observed rise in systolic blood velocity across the pulmonic and aortic valves. Subaortic and supravalvular aortic stenosis were recognized by identifying systolic turbulence produced by a membranous structure situated just beneath or above the aortic valve, leading to fixed narrowing. A double-chambered right ventricle (DCRV) was recorded when disturbed flow with high velocity originated from a mid-ventricular obstruction dividing the right ventricle into a proximal high-pressure and a distal low-pressure area. Aortic insufficiency (AI) was determined by detecting marked diastolic regurgitation from the aorta into the left ventricle, typically linked to aortic valve dysplasia or endocarditis [25, 26].

Ventricular and atrial septal defects (VSD and ASD)

Heart murmurs resulting from VSD or ASD were verified after visualization of an incomplete septum and recognition of systolic turbulence across the defect [27].

Tetralogy of Fallot

Disturbed blood flow related to tetralogy of Fallot was defined by the coexistence of pulmonic stenosis with a right-to-left shunting ventricular septal defect [27].

Patent ductus arteriosus (PDA)

Patent ductus arteriosus (PDA) was identified through the detection of the ductus arteriosus and continuous turbulent blood movement across the vessel, flowing toward the main pulmonary artery. All PDAs in this dataset showed a left-to-right shunting pattern [28].

Atrioventricular canal defect

In cases of atrioventricular canal defect, also known as endocardial cushion defect, the hallmark finding was systolic shunting of blood from the left ventricle through the right ventricle into the pulmonary artery [29].

Coronary artery to pulmonary artery fistula

The murmur associated with a coronary-to-pulmonary artery fistula was due to continuous left-to-right turbulent flow through an abnormal vascular link [30].

Statistical analysis

All information gathered was entered into Microsoft Excel 365 (Microsoft Corp., Redmond, DC, USA) and reviewed for precision by three independent reviewers (LF, NC, and AC). The Shapiro–Wilk test was applied to

assess the normality of quantitative data. Normally distributed variables were expressed as mean ± standard deviation, whereas non-normally distributed data were summarized as median and range. Categorical variables were presented as counts and percentages.

To test whether most cats with auscultated murmurs lacked anatomic cardiac defects, the proportion of such cases without echocardiographic abnormalities was determined.

Univariable logistic regression was applied to examine whether age, sex, breed, body weight, and murmur features (timing, intensity, point of maximal intensity, and whether dynamic or intermittent) could predict structural cardiac disease. All predictors were treated as categorical. Murmur intensity was classified as "soft" (1/6–2/6), "moderate" (3/6), "loud" (4/6), or "palpable" (5/6–6/6), following the simplified grading approach introduced by Rishniw (2018), which conveys equivalent diagnostic meaning to the traditional Levine system but with easier interpretation [8].

Variables showing significance in univariable testing were included in a multivariable logistic regression to evaluate independent relationships with cardiac structural defects. Statistical significance was defined as $p < 0.05$. Analyses were carried out using MedCalc Software (MedCalc Software Ltd., Ostend, Belgium).

Ethical approval

Because the study was retrospective and non-invasive, formal ethical review was not required.

Results and Discussion

Population Characteristics

Database screening identified 1,521 cats referred for murmur evaluation from June 2009 to June 2022. Of these, 856 animals had complete medical and echocardiographic data available, including all images and video clips. Every included patient was stable enough for a comprehensive echocardiographic examination.

Animals

The median age of the studied cats was 5.6 years (range: 1 month–19.5 years), and the median body weight was 4.4 kg (range: 0.3–12.4 kg). There were 539 males (63.0%) and 317 females (37.0%), of which 463 were neutered males (54.1%) and 267 spayed females (31.2%). Domestic shorthair (DSH) cats made up the majority of the cohort (60.5%), followed by domestic longhair (DLH) (6.8%) and British shorthair (BSH) (6.1%). The distribution of all other breeds is summarized in **Table 1**.

Table 1. Breeds represented in the 856-cat sample with documented heart murmurs.

Breed	Number of Cats	Percentage (%)
Domestic Shorthair	518	60.51
Domestic Longhair	58	6.78
British Shorthair	52	6.07
Bengal	49	5.72
Maine Coon	30	3.50
Persian	30	3.50
Ragdoll	16	1.87
Birman	16	1.87
Sphynx	16	1.87
Siamese	10	1.17
Siberian	9	1.05
British Blue	8	0.93
Scottish Fold	7	0.82
Burmese	6	0.70
Exotic	6	0.70
Norwegian Forest Cat	4	0.47
Tonkinese	2	0.23
Savannah	2	0.23

Russian Blue	2	0.23
Devon Rex	2	0.23
Selkirk Rex	2	0.23
Chinchilla	2	0.23
Ocicat	2	0.23
Oriental Shorthair	2	0.23
Himalayan	1	0.12
Egyptian Mau	1	0.12
Cyprus Shorthair	1	0.12
Korat	1	0.12
Turkish Van	1	0.12

DSH: Domestic Shorthair; DLH: Domestic Longhair; BSH: British Shorthair.

Sedation

Out of the total cases, 73 cats (8.5%) underwent sedation before echocardiographic assessment, either because they did not tolerate the procedure adequately or were already sedated for other diagnostic purposes unrelated to the cardiac exam. Over the study period, sedation regimens varied according to clinician preference. The combinations employed included injectable ketamine/midazolam, butorphanol/acepromazine, butorphanol/alfaxalone, as well as single-agent buprenorphine (injectable) or gabapentin (oral).

ECG findings

Most cats maintained a sustained normal sinus rhythm (91.4%). Occasionally, sinus rhythm occurred alongside ventricular ectopic beats (3.8%) or atrial premature contractions (2.1%). Persistent rhythm and conduction abnormalities included atrial fibrillation (1.0%), isorhythmic atrioventricular dissociation (0.9%), ventricular tachycardia (0.4%), atrial standstill (0.2%), and complete atrioventricular block (0.2%).

3.3. Heart Murmur Characteristics

Timing: The majority of murmurs detected in this population were systolic (98.7%), with smaller portions being diastolic (0.7%), continuous (0.5%), or to-and-fro (0.1%). Diastolic murmurs were found in association with aortic insufficiency (AI), cor triatriatum sinister, and mitral valve stenosis (MS). However, some cats with notable AI lacked an audible diastolic murmur, which was considered incidental when another—typically systolic—murmur was evident on echocardiography. Continuous murmurs were linked to left-to-right patent ductus arteriosus (PDA) in four cats and a coronary artery–pulmonary artery fistula in one cat. A single to-and-fro murmur was documented in a case involving mitral valve dysplasia with concurrent regurgitation and stenosis.

Intensity: The distribution of murmur loudness was as follows: soft (56.7%), moderate (28.7%), loud (12.6%), and palpable (2.1%) **(Figure 1)**. The most intense murmurs consistently correlated with severe congenital disorders such as VSD, PDA, DCRV, PS, and mitral valve dysplasia, whether isolated or combined. Roughly 27.8% of murmurs were described as intermittent, while 27.3% were dynamic, meaning their volume fluctuated with heart rate changes (either spontaneous or stimulated) or following gentle thoracic pressure with the stethoscope [31].

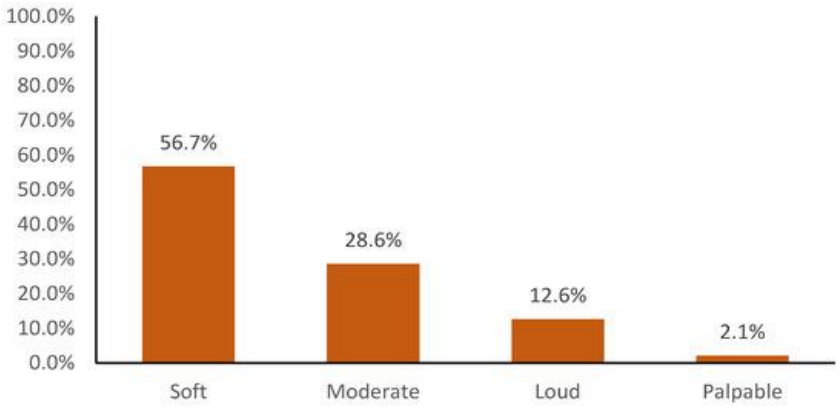

Figure 1. Histogram illustrating the distribution of soft, moderate, loud, and palpable murmurs in **856 cats** evaluated for cardiac murmur investigation.

Point of maximal intensity (PMI): The PMI was categorized as sternal (or both parasternal) in 38.3%, left parasternal in 37.2%, right parasternal in 21.7%, left base in 2.0%, and left apex in 0.8% of cases.

3.4. Cause of Heart Murmurs

Echocardiographic studies identified the murmur source in 93.1% of cats, determined through the detection of marked blood flow turbulence on color and spectral Doppler imaging. In most cases (72.3%), a single turbulent jet corresponded to the audible murmur, whereas 226 cats (26.4%) and 11 cats (1.3%) showed two or three distinct causes, respectively. Altogether, 1,093 significant turbulent blood flow sites were recorded among the 856 cats included.

Heart murmurs in kittens

Heart murmurs were found in 30 kittens under four months old, commonly noticed after initial vaccination. Congenital disorders accounted for 12 cases (40.0%), while the remaining 18 (60.0%) were classified as flow murmurs. The youngest kittens diagnosed with an acquired cardiac disorder (HCM phenotype) were six months old. In both instances, echocardiography revealed findings consistent with acute myocarditis, followed by complete reverse remodeling and disappearance of the murmur on subsequent re-evaluation after several weeks.

Most common causes of heart murmur

The systolic anterior motion (SAM) of the mitral valve was the leading murmur etiology (39.2%), followed by dynamic right ventricular outflow tract obstruction (DRVOTO) at 32%, and flow murmurs at 6.9%. A summary of all turbulent blood flow origins contributing to murmur formation is shown in **Table 2** and **Figure 2**.

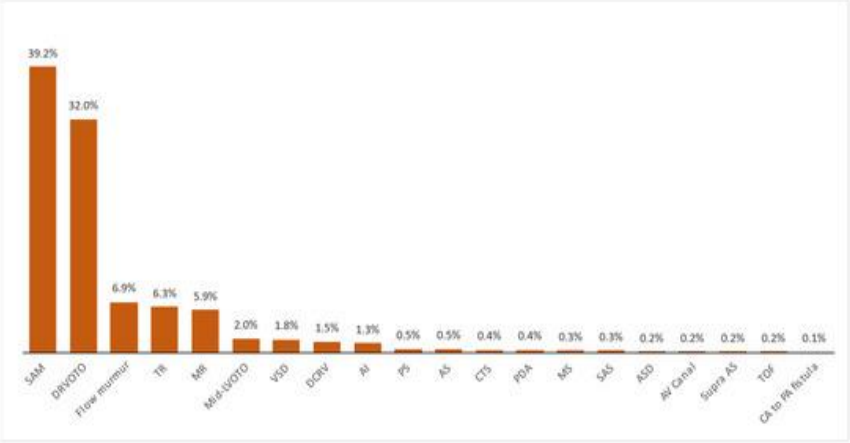

Figure 2. Histogram illustrating the distribution of turbulent flow sources responsible for murmurs in 856 cats.

Abbreviations: SAM = systolic anterior motion of the mitral valve; DRVOTO = dynamic right ventricular outflow tract obstruction; TR = tricuspid regurgitation; MR = mitral regurgitation; Mid-LVOTO = mid-left ventricular outflow tract obstruction; VSD = ventricular septal defect; DCRV = double-chambered right ventricle; AI = aortic insufficiency; PS = pulmonic stenosis; AS = aortic stenosis; CTS = cor triatriatum sinister; PDA = patent ductus arteriosus; MS = mitral valve stenosis; SAS = subaortic stenosis; ASD = atrial septal defect; AV canal = atrioventricular canal defect; Supra AS = supravalvular aortic stenosis; TOF = tetralogy of Fallot; CA–PA fistula = coronary-to-pulmonary artery fistula.

Table 2. Causes of the 1,093 turbulent blood flows observed during echocardiographic evaluation of **856 cats** with murmurs.

Etiology	Number of Cases	Percentage (%)	Additional Notes
Systolic Anterior Motion of Mitral Valve	429	39.25	23 inducible
Dynamic Right Ventricular Outflow Tract Obstruction	350	32.02	71 inducible

Physiologic Flow Murmur	75	6.86	1 associated with anemia
Tricuspid Valve Regurgitation	69	6.31	
Mitral Valve Regurgitation	64	5.86	
Mid-Ventricular Left Outflow Tract Obstruction	22	2.01	
Ventricular Septal Defect	20	1.83	
Double-Chambered Right Ventricle	16	1.46	
Aortic Valve Insufficiency	14	1.28	
Pulmonic Valve Stenosis	6	0.55	
Aortic Valve Stenosis	5	0.46	
Cor Triatriatum Sinister	4	0.37	
Patent Ductus Arteriosus	4	0.37	
Mitral Valve Stenosis	3	0.27	
Subaortic Stenosis	3	0.27	
Atrial Septal Defect	2	0.18	
Atrioventricular Canal Defect	2	0.18	
Supravalvular Aortic Stenosis	2	0.18	
Tetralogy of Fallot	2	0.18	
Coronary-to-Pulmonary Artery Fistula	1	0.09	

Abbreviations: SAM, DRVOTO, LVOTO = left ventricular outflow tract obstruction.

Relationship between heart murmurs and structural cardiac defects

Among cats that received a complete echocardiographic evaluation following the detection of a heart murmur, more than half exhibited no anatomical cardiac defect (n = 485; 56.7%). In most of these animals, the origin of the turbulent flow was related to SAM (50.3%), DRVOTO (19.9%), or physiological (flow) murmurs (14.6%). When considered independently as the sole contributor, 56.1% of cats showing SAM and 85.0% of those with DRVOTO demonstrated no structural alteration on echocardiography. By definition, none of the cats with flow murmurs showed evidence of any structural defect. DRVOTO was more frequent in older animals, with a median age of 9.3 years (range: 6 months–19.5 years).

The remaining murmur etiologies were associated either with congenital malformations (14.0%) or with acquired heart diseases (29.3%), with proportional frequencies varying by diagnosis, as summarized in **Table 2**. All cases with VSD or ASD revealed a left-to-right shunt across the septal defect, except for Tetralogy of Fallot, which featured a right-to-left flow through the VSD. Of note, only 19.7% of cats presenting SAM displayed echocardiographic evidence of left ventricular hypertrophy.

Physical variables (age, sex, breed, body weight) and murmur traits (timing, grade, PMI, dynamic pattern, or intermittence) were examined using both univariable and multivariable logistic regression models to determine the likelihood of detecting a structural heart disorder via echocardiography. All continuous, diastolic, to-and-fro, and palpable murmurs were significantly associated with congenital cardiac abnormalities (100%). Conversely, soft to moderate systolic murmurs, intermittent murmurs, and those with a PMI located at the right parasternal region were inversely correlated with the presence of cardiac pathology on echocardiography **(Table 3)**.

Table 3. Results of the multivariable model illustrating associations between the probability of detecting a structural cardiac lesion on echocardiography and feline or murmur characteristics identified as significant in the univariable analysis.

Variable	Coefficient	Odds Ratio	95% Confidence Interval	p-Value
Age	−0.01	0.99	0.96 to 1.02	0.5130
Soft murmur	−0.94	0.39	0.24 to 0.65	0.0003
Moderate murmur	−0.84	0.43	0.26 to 0.71	0.0010
Dynamic murmur	−0.35	0.70	0.49 to 1.02	0.0660
Intermittent murmur	−1.35	0.26	0.17 to 0.39	<0.0001
PMI = “RPA”	−0.69	0.50	0.32 to 0.78	0.0020

PMI: point of maximal intensity; RPA: right parasternal area.

Inducible Heart Murmurs

A subset of cats that exhibited audible murmurs during auscultation showed no apparent flow disturbance on echocardiography until a provocative maneuver was applied; these were categorized as inducible heart murmurs (93 cats, 10.9%). Within this subset, inducible DRVOTO accounted for 8.3% and inducible SAM for 2.6%. Specifically, a provocative test was required to uncover DRVOTO in 20.3% and SAM in 5.1% of the affected animals.

To the authors' best understanding, this investigation represents the most extensive and detailed assessment of the causes and clinical attributes of feline heart murmurs to date, and differs from prior studies by systematically identifying the precise site of the turbulent blood flow responsible for murmur generation.

The median age and body weight of cats referred for cardiologic evaluation were comparable to those documented in previous reports involving cats examined by veterinary cardiologists [2, 6].

Most murmurs identified during auscultation were systolic, mild to moderate in intensity, and typically heard over both parasternal regions ("sternal" murmurs), showing no clear relationship with either the cause or severity of the underlying cardiac disorder. This indicates that, for most cats, murmur characteristics hold limited diagnostic value—though louder murmurs are more commonly linked to significant structural heart disease, primarily congenital defects, as earlier described [32].

The presence of intermittent murmurs may have multiple explanations. They may become audible when sympathetic tone rises under stress or excitement, leading to elevated heart rate, contractility, and blood pressure. Another plausible cause is the induction of iatrogenic murmurs due to excessive pressure from the stethoscope during auscultation [31]. A comparable mechanism may contribute to dynamic murmurs, which remain audible but vary in loudness through the cardiac cycle.

Echocardiography successfully identified the turbulent blood flow responsible for the murmur in 93.1% of examined cats. In the remaining 6.9%, no detectable flow disturbance or structural defect was found, resulting in a diagnosis of flow (innocent) murmurs according to human-based diagnostic standards [20, 21].

Roughly one quarter of the studied cats exhibited multiple regions of blood flow turbulence on echocardiography, believed to contribute collectively to the auscultated murmur. This reinforces the idea that auscultation alone—without echocardiographic confirmation—cannot reliably determine murmur etiology, except in cases of intermittent murmurs induced by chest pressure or those revealed by variable stethoscope pressure [31], and in kittens, where loud murmurs are usually linked to major congenital anomalies (40.0%) and softer ones typically correspond to benign conditions (60.0%).

Contrary to previous publications in which feline murmurs were mostly connected with cardiac pathology [2, 3, 5, 7], our findings show that over half (56.7%) of feline heart murmurs occur in the absence of structural heart disease.

The most frequent source of cardiac murmurs in these cats was a dynamic obstruction of the left ventricular outflow tract associated with systolic anterior motion (SAM), accounting for roughly 40% of all murmurs identified in this feline cohort. Among cats exhibiting an HCM phenotype accompanied by a murmur on auscultation, 80% presented with SAM. Systolic anterior motion of the mitral valve was also observed in other cardiomyopathy forms, including 27.3% of restrictive cardiomyopathy (RCM) cases, 33.3% of non-specific cardiomyopathy, and 22.2% of end-stage HCM, according to the classification outlined by Luis Fuentes *et al*. [33]. Additionally, SAM was present in 24.2% of cats diagnosed with mitral valve dysplasia. Nevertheless, 56.1% of all cats with SAM displayed no echocardiographic evidence of structural heart disease, indicating that this dynamic left ventricular outflow tract obstruction is not specific to HCM. This observation supports the hypothesis first proposed and later verified by Ferasin *et al*. [13, 14].

The second most frequent cause of cardiac murmurs was dynamic right ventricular outflow tract obstruction (DRVOTO), which occurred in approximately one-third of the cases. In 85.0% of DRVOTO cases, no underlying cardiac abnormality was present. Since the echocardiographic irregularities identified in the remaining 15.0% did not impact the right outflow tract, it is likely that DRVOTO in those cats represented an incidental, non-pathological finding.

Flow (innocent) murmurs represented the third most prevalent cause of murmur in this investigation (6.9%). However, their true prevalence may have been slightly overestimated because provocative maneuvers were not always applied during echocardiography to uncover inducible SAM or iatrogenic DRVOTO [18].

Misinterpretation of respiratory sounds synchronized with the heartbeat as murmurs may also have contributed to diagnostic inaccuracies in some instances.

Blood flow turbulence related to various congenital malformations was identified as the cause of audible murmurs in the remaining 21.9% of cases. However, based on the available data, it was not possible to determine whether ASDs alone were responsible for producing an audible murmur, as they were consistently associated with additional significant flow disturbances.

This finding carries substantial clinical relevance, as early detection may alter patient outcomes by facilitating corrective or palliative procedures (e.g., PDA, PS, AS) or through regular monitoring of resting respiratory rate to enable early recognition of symptoms linked to congestive heart failure [34, 35].

Because this research was retrospective, several data points were unavailable for some cats, including blood pressure readings, hematocrit levels, and serum thyroxine concentrations, which may have influenced murmur development. The absence of information on systemic health status prevents definitive exclusion that, in certain individuals, the primary or contributing cause of the murmur might have been inaccurately identified.

Another limitation involved the uncertainty of whether outflow tract obstructions (SAM or DRVOTO) could have been provoked in some "innocent" murmur cases, as the aforementioned provocative maneuvers were not consistently performed, particularly when these techniques were not yet well established. Limited classification of murmur types for each echocardiographic abnormality also introduces potential bias.

An additional constraint concerns the possible influence of various sedative protocols on echocardiographic structure and function, although only a small percentage of cats underwent sedation. Similarly, data regarding the administration of cardioactive medications were absent in many cases, preventing evaluation of their potential effects on clinical and imaging outcomes. Furthermore, because multiple clinicians participated in diagnosing and managing the cats in this study, variations in interpretation could have influenced accuracy; nonetheless, all clinical files and echocardiographic images were retrospectively reviewed by a board-certified cardiologist to verify measurements and diagnostic.

Conclusion

More than half of the cats referred for evaluation of an audible murmur during thoracic auscultation exhibited no echocardiographic indicators of heart disease. This finding suggests that many feline heart murmurs are benign or may be related to subclinical cardiac alterations undetectable via echocardiography. Moreover, this investigation confirms that specific murmur features—such as timing, loudness, and localization—can occasionally aid in distinguishing healthy cats from those with cardiac pathology, except for loud (usually systolic) and palpable murmurs, which almost always correspond to significant disease. However, since most feline murmurs are systolic and of mild to moderate intensity, echocardiographic assessment—preferably by an experienced cardiologist—should always be pursued following murmur detection during routine examination, even in cats lacking overt clinical signs of heart disease.

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Ethics Statement: None

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