Heart murmurs in young dogs and cats: differentials, tips and additional testing

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Heart murmurs can occasionally be auscultated in young dogs and cats; their discovery can be in conjunction with other signs of cardiac disease or it may be an incidental finding. The identification of heart murmurs can cause apprehension for owners, and depending on the location, intensity and characteristics, the veterinary professional can perform a list of differentials and suggest the best diagnostic approach.

CARDIAC AUSCULTATION

The first stethoscope was invented in 1816 by René Laennec in Paris (Rishniw, 2018) and it has been, since then, a very powerful instrument to aid clinicians in their everyday cardiac evaluation. Auscultation takes time and practice, but it can help in stratifying the risk associated with the identification of an abnormal finding. It is very important that the stethoscope is in good condition too.

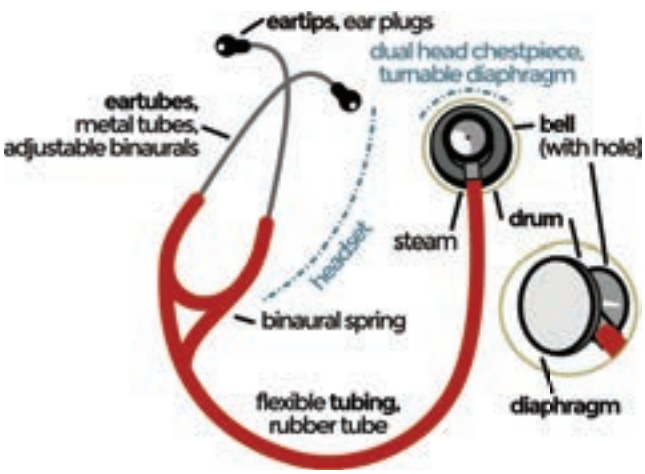

Figure 1: Anatomy of a stethoscope. The stethoscope is composed of earpiece tips, binaural headpiece, tubing and chest piece. The latter is composed of bell and diaphragm. The bell held lightly against the chest transmits both low and high frequency (which is generally preferred for auscultating both lung and low frequencies heart sounds), whereas the diaphragm allows for a more filtered auscultation of high frequencies (cardiac auscultation). Most stethoscopes allow the locking of one of the two components (either bell or diaphragm) to permit better alignment with the tubes and increase quality of sound (image courtesy of Jaroald, 2017 under Creative Commons Licence, https://commons.wikimedia.org/wiki/File:Stethoscope.svg).

In dogs and cats, normal auscultation is characterised by two audible heart sounds (S1 and S2), which separate systole (S1-S2) from diastole (S2-S1). A quiet environment and a relaxed patient provide the best clinical condition to detect abnormalities; however, this sometimes may not happen in everyday practice. Strategies to reduce environmental noise levels and calm the patient, as well as repeated auscultation can be attempted to increase efficacy or confirm the initial suspicion. The patient should be standing, and palpation of the chest walls should be performed prior to applying the stethoscope to identify the precordium and look for thrills, if present (Smith *et al*, 2006).

Inching (moving the stethoscope between the cardiac apex and base) helps in identifying any abnormal heart sound and its point of maximal intensity (PMI). Traditionally, three areas are heard on the left hemithorax (pulmonary, aortic and mitral [PAM] from a more cranial to caudal position) and one on the right (tricuspid valve [T] [Smith *et al*, 2006]). Some murmurs may be well localised, whereas some others irradiate to the other hemithorax. Auscultation of the thoracic inlet may aid in identifying particular murmur irradiation (ie. aortic stenosis).

Heart murmurs are the result of turbulent blood flow. The Reynolds number gives the probability of a flow to be laminar (low) or turbulent (high). The Reynolds number has no practical use but is helpful in explaining which conditions can increase the chances of a flow to become turbulent, and therefore to cause a heart murmur (Sisson DD and Ettinger SJ, 1999). These include increased blood velocity, decreased blood viscosity (such as in anaemic patients), changes in vessel/valve radius (such as stenosis, which is generally associated with increased velocity) or changes in blood density. Heart murmurs can either be associated with heart disease (pathological murmur) or may occur in an otherwise normal heart (non-pathological, normal, innocent, functional or physiological murmur). A slight difference in meaning can be given when using the words 'functional' or 'innocent' murmur. Functional murmurs can be defined as heart murmurs in the absence of structural heart disease, but a plausible explanation for the murmur can be identified (ie. anaemia), whereas an innocent murmur defines a heart murmur in the absence of structural heart disease without any obvious causative explanation (Coté *et al*, 2015). Non-pathological murmurs may also come with peculiar auscultatory characteristics that can aid in their recognition (see Table 1).

Table 1: Heart murmur characteristics that are more likely associated with a non-pathological heart murmur in dogs.

Characteristics	
Change with rest/exercise	Softer murmur at rest or even absent, increased intensity after exercise
Murmur intensity	Grade 1 to 3/6; can be variable
Timing	Early/mid-systolic or late systolic (does not last the entire systole)
Murmur localisation	Left heart base, no murmur irradiation
Other auscultation abnormalities	Absent

Non-pathological heart murmurs have been identified in young puppies (Szatamari *et al*, 2015), boxer dogs (Höglund *et al*, 2004), retired racing hounds (Fabrizio *et al*, 2006) and whippets (Bavegem *et al*, 2011), but they can also be auscultated in animals with systemic disease (ie. anaemia, fever).The most common murmur grading scheme proposed in veterinary medicine comes from Ettinger and Suter, a six-level scheme adapted from the Detweiler's first veterinary grading scheme (see Table 3).

Table 2: Heart murmur grading scales.

6-level murmur grading	Description	Descriptive 4-level murmur grading	Description
I/VI	Nearly imperceptible, may be heard with very careful auscultation in a quiet environment; always focal	Soft (I-II/VI)	Softer than heart sounds. Both heart sounds can be easily heard and are louder than the murmur
II/VI	Heard readily but very soft; always focal	Moderate (III/VI)	Equal to heart sounds. Heart sounds can be easily heard and are equal to the murmur in intensity
III/VI	Heard readily, moderate intensity; usually regional (can be heard in several auscultatory regions of the heart)	Loud (IV/VI)	Louder than heart sounds. One or both heart sounds often obliterated by the murmur or distinctly softer than the murmur
IV/VI	Heard readily, loud, and usually radiates widely (can be heard in most or all auscultatory regions of the heart), but without a palpable thrill	Palpable (V-VI/VI)	Has a palpable thrill, regardless of auscultated intensity
V/VI	Heard readily, loud, and associated with a precordial thrill, but the murmur is not heard with the stethoscope lifted off the surface of the thorax		
VI/VI	Heard readily, loud, associated with a precordial thrill, and the murmur remains audible with the stethoscope lifted 1cm off the surface of the thorax		

More recently, a simplified grading scheme in dogs with mitral valve disease seemed to provide enough clinical information when only four grading categories were applied (soft – grade I+II/VI; moderate – grade III; loud – grade IV; and thrilling – grade V+VI/VI [Ljungvall *et al*, 2014]). In dogs with pulmonic stenosis and subaortic stenosis, an even simpler classification was considered to be already informative in discriminating mild disease from moderate/severe disease, the latter of which would require further workup (anything louder than soft murmurs; ie. murmur grades greater than III/VI [Caivano *et al*, 2018]).

Figure 2: Cardiac auscultation. On the left hemithorax murmurs at the pulmonary and aortic valve are heard at the heart base, whereas murmurs affecting the mitral valve are located more toward the cardiac apex. At the thoracic inlet some murmurs can still be auscultated, and on the right hemithorax murmurs at the tricuspid valve are heard. Note that to fully auscultate the characteristic continuous water hammer murmur typical of patent ductus arteriosus, the stethoscope needs to be further cranial in the left hemithorax; ventricular septal defects murmurs are loudest on the right hemithorax but can also be auscultated on the left hemithorax between the heart base and apex.

Table 3: Differentials and associated clinical signs in puppies with a murmur

Murmur	Location	Differentials	Possible associated clinical signs/physical examination findings
Systolic murmur	Left heart base		
		Pulmonic stenosis (true or relative)	Stunted growth, exercise intolerance, syncope, ascites arrhythmias
		Subaortic stenosis	Stunted growth, exercise intolerance, syncope, respiratory distress, arrhythmias. Weak pulses ('pulsus parvus et tardus')
		Non-pathologic murmur	No clinical signs
Systolic murmur	Left apex		
		Mitral valve dysplasia	Syncope, respiratory distress
		Mitral regurgitation due to mitral annular dilation (juvenile dilated cardiomyopathy, arrhythmia-induced cardiomyopathy, patent ductus arteriosus)	Exercise intolerance, respiratory distress, arrhythmias. Regular alternation of femoral pulse amplitude ('pulsus alternans')
Systolic murmur	Right hemithorax		
		Ventricular septal defect (murmur heard also between L heart base and L apex, loudest on R hemithorax)	Respiratory distress
		Tricuspid valve dysplasia	Ascites. Jugular distension
Continuous murmur	Left heart base		
		Patent ductus arteriosus	Stunted growth, exercise intolerance, respiratory distress. Bounding femoral pulses
Diastolic murmurs	Left cardiac base		
		Aortic valve stenosis with insufficiency	Bounding pulses
		Pulmonic insufficiency (most likely due to pulmonary hypertension)	Exercise intolerance, syncope
Diastolic murmurs	Left cardiac apex		
		Mitral valve stenosis	Syncope, respiratory distress
No murmurs			
		Tetralogy of Fallot	Cyanosis, exercise intolerance, syncope-hypercyanotic spells
		Cor triatriatum dexter	Ascites. No jugular distension
		R-L shunting PDA	Exercise intolerance, syncope, respiratory distress, differential cyanosis

HEART MURMUR IN PUPPIES – DIFFERENTIALS

Congenital heart disease (CHD) is probably the number one differential in a young dog with a murmur. Depending on the location and timing, a list of most likely differentials can be drawn (see Table 3).

Most young dogs with murmurs may present without additional clinical signs, but some others may present for exercise intolerance, respiratory effort, ascites or syncopal events. The list of differentials may be different in these two categories of patients, as well as the diagnostic approach. When clinical signs develop, particularly if they lead to the diagnosis of congestive heart failure, it is necessary to treat heart failure prior to performing further investigations.

The prevalence of CHDs in the dog population ranges between 0.13% in mixed-breed dogs from an American shelter (Schrope *et al*, 2015) to 22% of all cardiac cases in a secondary referral hospital (Oliveira *et al*, 2011). Congenital heart defects' prevalence is likely linked to breeds' prevalence, but the most common simple CHD have not changed much since the first disease prevalence studies were reported in the 1970s. These include pulmonic stenosis, subaortic stenosis and patent ductus arteriosus. Depending on the murmur quality, a list of differentials can be drawn (Table 3).

Some breeds may be more prone to developing specific CHDs, which may not be very common in the overall population. Mitral valve dysplasia is very common in bull

terrier dogs (Oliveira *et al*, 2011), as tricuspid valve dysplasia is for Labrador retrievers (Oliveira *et al*, 2011).

Some dogs with CHDs may present with clinical signs but may have very soft murmurs or none at all. These CHD are less common but may be worth considering whenever you are assessing a young patient with history of exercise intolerance or syncope. The list of differentials should include Tetralogy of Fallot (most common cyanotic CHD), which can be associated with a left base systolic murmur with different intensity (the softer the more severe the disease), reversed right to left shunting patent ductus arteriosus or ventricular septal defect (both not associated with any heart murmur). Because some of the clinical signs associated with right-to-left shunting are due to hypoxia and polycythaemia, a blood haematocrit is a valid ancillary test to determine the chronicity of the disease process. Atrial septal defects or less common congenital heart defects (cor triatriatum dexter, atrioventricular septal defects to cite a couple) lack heart murmurs.

Another noteworthy differential in a dog with low-grade systolic heart murmur is non-pathological heart murmur. The prevalence of non-pathological murmurs reported in a study of young dogs (one to five years old) varied between 6-12% (Szatamari *et al*, 2015) and was 16% in another study including only mixed-breed dogs (Schrope *et al*, 2015). The murmurs were all systolic, low-grade, and mainly at the left cardiac base. Some breeds of athletic dogs may also have a higher prevalence of innocent murmurs (up to 58% in whippets and 67% in retired racing greyhounds [Bavegem *et al*, 2011] [Fabrizio *et al*, 2004]). Boxer dogs may present innocent murmurs, but mild subaortic stenosis may also be present or should be excluded by echocardiography (Höglund *et al*, 2006). Any chamber dilation leading to mitral annular dilation can result in a soft, left apical heart murmur.

Juvenile dilated cardiomyopathy has been identified in Portuguese water dogs (Sleeper *et al*, 2002). This is associated with an autosomal recessive mutation causing myocardial atrophy. Clinical signs, heart murmurs and auscultation abnormalities tend to develop close to decompensation and death (mean age four months, range two to 32 weeks). A gallop sound (audible S3) may also be auscultated in these patients. Toy Manchester terrier dogs also seem to present a similar form of juvenile dilated cardiomyopathy, with similar sudden decompensation or sudden death (Legge *et al*, 2013).

Arrhythmia-induced cardiomyopathy (previously known as tachycardia-induced cardiomyopathy) may also be a differential in young dogs with irregular heart rhythms and a soft, left apical murmur. Congenital arrhythmias due to accessory pathways may cause sustained supraventricular tachycardia leading to a dilated cardiomyopathy phenotype and heart murmur due to mitral and/or tricuspid regurgitation (Gopinathannair *et al*, 2015).

Up to 46.1% of dogs with an accessory pathway had a dilated cardiomyopathy phenotype and 38% of dogs were in congestive heart failure on presentation. Concurrent CHDs were identified in 13% of dogs as well (Wright *et al*, 2018; Santilli *et al*, 2018). Labrador retrievers are over-represented, but other breeds are affected as well. Radiofrequency catheter ablation was successful in ablating the accessory pathways in nearly all patients in both centres.

Diet-induced dilated cardiomyopathy has also been recently identified in dogs fed grain-free diets that were not standard breeds affected by dilated cardiomyopathy (Adin *et al*, 2019); mean age at presentation was four years, but a thorough anamnesis should include the puppy's diet given these recent findings and the risk of developing diet-induced cardiomyopathy.

HEART MURMUR IN KITTENS – DIFFERENTIALS

Heart murmurs are common in cats (from 15.5% to 40.8% depending on the study, Payne *et al*, 2015). It is even more difficult to define the location of a heart murmur in a cat; inching can be performed, and listening at the sternum sometimes can help identifying murmurs in a cat. Purring is one of the most common limitations to cardiac auscultation in a cat; an attempt to stop purring can be made by opening a water tap or gently closing the nose to the cat. Alcohol smell has been anecdotally used, but this is generally unpleasant and can upset some of the cats, so should be avoided.

Murmur intensity, timing and variability should be noted. Some murmurs are labile and may not always be present. Whenever a murmur is heard in a cat, an echocardiogram should be suggested to the owner as an additional step to investigate its source, given the high prevalence of non-pathological disease.

The top two differentials in a cat with murmur are hypertrophic cardiomyopathy and non-pathological murmurs, with a nearly 50:50 ratio. Fewer studies investigated the prevalence of CHD in kittens; a prevalence of 0.14-0.5% has been reported (Payne *et al*, 2015). The most common CHD included ventricular septal defects, tricuspid valve dysplasia, pulmonic stenosis, atrial septal defect or aortic stenosis (Tidholm *et al*, 2015). Pedigree cats may also have more complex or rare CHD.

Juvenile hypertrophic cardiomyopathy is a very common cause of heart murmur in young cats. Pedigree cats (mainly Ragdolls, Maine Coons, Bengals, Sphynxes and Persians) are reported to be at higher risk, with Ragdoll kittens developing severe disease at a young age – as young as a few months (Silverman *et al*, 2012; Trehiou-Sechi *et al*, 2012; Borgeat *et al*, 2014). Non-pedigree cats can show hypertrophic cardiomyopathy (HCM) as well. Hypertrophic cardiomyopathy can also be present in the absence of a heart murmur and undiagnosed HCM is not an uncommon cause of unexpected death in the feline population (Wilkie LJ *et al*, 2017).

Systolic anterior motion of the mitral valve leading to left ventricular dynamic outflow tract obstruction is generally responsible for heart murmur in a cat with HCM, but may be present in a minority of cats without obvious or equivocal left ventricular hypertrophy (4.4%; Payne *et al*, 2015).

Dynamic right ventricular outflow tract obstruction is another identified cause of functional murmur (Rishniw *et al*, 2002).

FURTHER TESTING

Depending on the presence or absence of clinical signs,

the choice of additional diagnostic work-up varies.
Echocardiography is probably the best diagnostic test to perform once a heart murmur is heard because it allows identification of the cardiac abnormality, if present, or ruling it out, if not. If the patient is in congestive heart failure, focused thoracic ultrasound to detect B-lines, thoracic or abdominal effusion and to assess chamber size is a valid tool (Boysen *et al*, 2013). Once the patient is stable or if the patient is asymptomatic, a full echocardiogram can be performed to assess chamber size and function as well as valve morphology or abnormal chamber communications.
Electrocardiography is strongly advised whenever heart rate or rhythm is abnormal on auscultation (tachycardias or bradycardias) to identify the type of the arrhythmia and whether this requires medications. It is also helpful to identify for any sign of concealed accessory pathway (ventricular pre-excitation), although this may be present only in the minority of dogs with accessory pathways (30-48% of all cases). Twenty-four-hour Holter monitoring should be considered should a persistent or intermittent arrhythmia be identified on auscultation.
Thoracic radiographs can provide insights into abnormal lung parenchyma, which may be secondary to heart disease or may be an unrelated cause of tachypnoea/respiratory distress in a dog or cat with a heart murmur. Cats can present a more variable radiographic appearance of cardiogenic pulmonary oedema, as well as a not-so-obvious cardiomegaly, in contrast with dogs, where these findings are more standardised (Guglielmini *et al*, 2015). Patient triage is important as critically ill patients should not be imaged immediately, but should be stabilised prior to taking radiographs; generally, oxygen administration and mild sedation with butorphanol can be provided in order to allow the patient to settle. Focused thoracic ultrasound can also be a quicker initial way to assess for free fluid that could be drained in order to stabilise the patient enough to perform thoracic radiographs.
Cardiac biomarkers are substances specifically produced by the cardiac cells and released in proportion to cardiac damage (troponin I) or atrial/ventricular stretch (atrial and brain natriuretic peptides; ANP/BNP) (Oyama *et al*, 2015). Biomarkers such as ANP or BNP are very useful in the context of screening for causes of dyspnoea or in older patients with a heart murmur. The most commonly used is BNP, but due to its short half-life, a fragment of the pro-peptide is analysed (N-terminal pro BNP, NT-proBNP). In a prospective study including mainly Cairn terriers and few other breeds, NT-proBNP concentration was not different between dogs with an innocent murmur compared to those without a murmur (300pmol/L vs 326pmol/L). In contrast, pathological murmurs had a higher median NTproBNP (1102 pmol/L). However, few cases of pulmonic stenosis did not show an increase in NT-proBNP values, resulting in a false positive result (Marinus *et al*, 2017). Dogs with patent ductus arteriosus showed elevated NT-proBNP, which correlated with radiographic and echocardiographic parameters of cardiac enlargement (vertebral heart score and indexed left ventricular systolic chamber) [Hariu CD *et al* 2013]).

Table 4: Heart murmur in kittens – differentials.

Murmur type	Differentials
Systolic murmur	
	Hypertrophic cardiomyopathy
	Non-pathological murmur
	Ventricular septal defect
	Tricuspid valve dysplasia
	Pulmonic stenosis
Continuous murmur	
	Patent ductus arteriosus

No studies are available for CHD in relation to cats, but NT-proBNP is higher in cats with cardiomyopathy compared to healthy cats, with more severe heart disease being associated with higher NT-proBNP values. A cut-off of 99 or 100pmol/L allowed good differentiation between healthy cats and those affected by cardiomyopathy. The qualitative NTproBNP snap test currently available has a positive result with an average cut-off of 100pmol/L.
Certain parameters also affect NT-proBNP concentration, including breed and kidney disease. Labrador retrievers and Newfoundland dogs have higher basal values compared to other breeds (Sjöstrand *et al*, 2014) as well as retired healthy greyhounds (Couto KM *et al*, 2015). Elevated serum creatinine was also associated with elevated NT-proBNP, but stable kidney disease did not seem to be affecting glomerular excretion of either NT-proBNP and troponin I, indicating no major increase in these values in patients with stable kidney disease (Pelander *et al*, 2017).
NT-proBNP is also higher in cats with dynamic left ventricular outflow tract and hypertrophic cardiomyopathy who are not in congestive heart failure (Payne JR, unpublished data) compared to cats with similar wall thickness but no dynamic left ventricular outflow tract.
Exercise has also been shown to increase NT-proBNP values (Hunt *et al*, 2018), so working dogs or any active dog should be tested before any strenuous activity is performed, or if this is not possible, then considering exercise as a possible differential for raised NT-proBNP values and a repeated testing when the dog is at rest should be performed.
Troponins indicate acute myocardial damage; they are therefore, increased in cases of acute myocardial damage (ie. myocardial infarcts, myocarditis, etc.), but are not specific for the underlying cause. The most common assessed troponin is troponin I (TnI), but troponin T has also been measured in cats (Langhorn *et al*, 2014).
Troponins are increased in acquired heart disease but not in the most common CHDs, where the TnI value is similar to healthy dogs (Spratt DP *et al*, 2005). Cardiac catheterisation following pulmonary balloon valvuloplasty or transvenous pacemaker implantation was associated with raised post-procedural TnI, in contrast with patent ductus arteriosus

closure, which had no significant change from baseline (Shih *et al*, 2009).

Cats with HCM have variable TnI increase, which is generally mild in asymptomatic cats but slightly higher than in healthy cats (median 0.027 for healthy vs 0.103 for asymptomatic cats, Hori *et al*, 2018). An elevated TnI value (greater than 0.7ng/ml) was associated with cardiac death in a prospective cohort of cats with hypertrophic cardiomyopathy (Borgeat *et al* 2014), and similar results were identified in another prospective study (Langhorn *et al*, 2014), where both TnI and troponin T were of prognostic importance. However, troponins are not disease-specific, and TnI is elevated in other cardiac or systemic disease as well, such as transient myocardial thickening, arrhythmias, bartonellosis and thoracic trauma among others (Novo Matos *et al*, 2018; Oxford *et al*, 2018; Joseph *et al*, 2018; Bartoszuk *et al*, 2017).

CONCLUSIONS

Heart murmurs are the result of turbulent blood flow and can be associated with underlying heart disease, as well as being non-pathological, with different conditions in the dog or cat. Further testing may be necessary to fully rule in/out heart disease, but auscultation is an art that needs practice and time and, in experienced hands, with ideal environmental conditions and compliant patients, can aid in identifying a more pathological vs non-pathological murmur.

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READER QUESTIONS AND ANSWERS

1. WHICH OF THE FOLLOWING BREEDS HAVE NON-PATHOLOGICAL MURMURS?

A. Whippet
B. German shepherd
C. Jack Russell terriers
D. Toy Manchester terriers

2. WHICH OF THE FOLLOWING CONGENITAL HEART DISEASES IS NOT ASSOCIATED WITH A HEART MURMUR?

A. Left-to-right shunting patent ductus arteriosus
B. Pulmonic stenosis
C. Pulmonary hypertension
D. Right-to-left shunting ventricular septal defect

3. WHICH THORACIC AREA CAN INCREASE THE CHANCE OF DETECTING A HEART MURMUR IN A CAT WITH HYPERTROPHIC CARDIOMYOPATHY?

A. Mitral area
B. Left heart base
C. Sternal
D. Tricuspid area

4. IF I HAVE AN ELEVATED NT-PROBNP RESULT AND I WANT TO MAKE SURE IT IS TRULY ELEVATED, WHAT SHOULD I ENQUIRE ABOUT WITH THE OWNER?

A. Diet
B. Exercise
C. Probiotic use
D. Treatment with homeopathic drugs

5. WHICH OF THE FOLLOWING DISEASES CAN INCREASE TROPONIN I CONCENTRATION?

A. Transient myocardial thickening
B. Hyperthyroidism
C. Patent ductus arteriosus closure via interventional cardiology
D. Asthma

ANSWERS: 1 A; 2 D; 3 C; 4 B; 5 A