The practitioner should be familiar with the common cardiac malformations, its associated clinical signs, the repercussions for breeding and the possibilities of curative or palliative treatment. Thorough auscultation is a good screening tool and should be performed at the animal’s first presentation to the veterinarian (primary vaccination or earlier), and repeated at every representation. Innocent murmurs should be differentiated from pathological murmurs. Accurate assessment may require Doppler echocardiography and referral to a veterinary specialist in cardiology.

This is the first article of a series of three written to help the veterinarian in judging the importance and the long-term implications when a murmur is detected in a young animal. However, one has to remember that thoracic auscultation is only a piece of the puzzle, and that the observations should be correlated with the history and the findings of a full physical examination.

**SIGNALMENT**

**Age**

Innocent murmurs are most common in puppies less than 16 weeks old. Kittens can have innocent, dynamic murmurs for a much longer time, like adult cats. These innocent murmurs are always systolic (decrescendo), located over the left heart base, and should never exceed a grade 3/6. In puppies they should have disappeared by the age of 3.5 months. The continuous murmur of a patent ductus arteriosus (PDA) should have disappeared by the age of 5 days. However, a persistent left-to-right shunting defect might not manifest before a couple of weeks after birth because of the declining pulmonary vascular resistance for 1–2 months after birth. Some murmurs might increase in severity with age due to growth (e.g. stenotic lesions). A progression of clinical signs with age is expected in animals with severe malformations.

**Gender**

A sex predisposition has not well been established in dogs. Exceptions include PDA (females outnumber males by 3/1), and the possible male predominance in tricuspid dysplasia. Male English Bulldogs appear to have a higher prevalence of pulmonic stenosis.

**Breed**

Knowledge of the breed is very important because a genetic basis is suspected for many of the cardiac malformations. The frequency of congenital heart disease is greater in purebread than in mongrel dogs.

A list with breed predispositions can be be downloaded from www.acapulco-vet.be

**HISTORY**

**Family history**

The prevalence of congenital heart disease differs between breeds and countries and often within regions of a given country. This indicates the importance of regional gene pools in the aetiology of congenital heart disease.

**Clinical signs**

Most animals with incidental detection of a heart murmur at young age (vaccination, pre-neutering) will not have any clinical signs.

In affected animals the clinical signs can vary. Stunted growth is a common finding. Other clinical signs will often only show at the time of cardiac decompensation. These include decreased exercise (play) tolerance, shortness of breath, syncope, cyanosis (differential cyanosis in case of R–L PDA: cranial mucous membranes are pink, caudal mucous membranes are cyanosed because the brachycephalic trunk leaves the aorta before the PDA shunt reaches it), abdominal distension, hind limb weakness and cough.

**PHYSICAL EXAMINATION**

**Head**

Examination of the head includes oral inspection for accuracy of age (teething), mucous membrane colour (pink, pale, dark red, cyanosis) and capillary refill time.

**Neck**

The jugular veins should be inspected carefully in any case.
of suspected cardiac disease. This might warrant clipping or wetting of the hair.

Patients with overt right heart failure may have obviously distended jugular veins. The hepatojugular reflex might accentuate the presence of subtle heart failure.

Pulsating jugular veins can be seen with decreased right ventricular compliance as in severe right ventricular hypertrophy or with severe tricuspid regurgitation. Pulsation can also be seen with atrioventricular dissociation (3rd degree AV-block) because the atria contract against a closed AV-valve (canon a-waves).

**Thorax**

The thorax should always be palpated for deformities, for the strength and location of the precordial impulse (best felt in the region of the left apex), and the presence of a thrill.

Feeling a strong apex beat does not mean a strongly contracting heart. The apex beat is not the heart striking against the thoracic wall but the tension generated by the myocardium. This wall tension is calculated by multiplying the left ventricular chamber pressure by the left ventricular chamber diameter. Consequently a dog with a dilated ventricle will have a stronger apex beat.

A thrill is a vibrating feeling created by turbulence within the heart through the chest wall and best felt in the region of the heart murmur.

**Abdomen**

The abdomen should be inspected for the presence of free fluid or organomegaly. Right heart failure commonly produces ascites in dogs and the aspirated fluid can be a transudate, a modified transudate or even a chylous effusion.

**Extremities**

The femoral pulse, which represents the systolic blood pressure (BP) minus the diastolic pressure (=pulse pressure), might be altered in strength or formation.

A weak pulse (decrease in pulse pressure) indicates a decreased stroke volume (e.g. myocardial failure, severe aortic stenosis, severe pulmonic stenosis), lowering of the systemic peripheral vascular resistance, or increased arterial compliance.

A bounding pulse (increase in pulse pressure) is observed with an increase in systolic BP, a decrease in diastolic BP (e.g. aortic insufficiency), or a combination of both (PDA).

In dogs with severe aortic stenosis the pulse often peaks later into systole (pulsus tardus et parvus). Dogs with mitral regurgitation might have a brisk pulse because the left ventricle ejects blood at a higher velocity and the ejection time is shortened.

Pulse deficits are often associated with tachyarrhythmias (ventricular and supraventricular) because the left ventricle has not enough time to fill.

The temperature of the extremities should always be compared with the rest of the body. If they are colder it might suggest poor cardiac output and/or peripheral vasoconstriction. The presence of peripheral oedema is very uncommon in small animals but can occur with right-sided congestive heart failure.

**THORACIC AUSCULTATION**

Thoracic auscultation should be performed in a systematic fashion. Failure to do so will result in mis- or non-diagnosis.

**Principles of thoracic auscultation**

*The modern stethoscopes*

Auscultation is accomplished with the binaural stethoscope first introduced by George Cammann in 1855. Stethoscopes vary in quality of design and of sound transmission. The best instrument is the one that fits the ears comfortably (Fig. 1) and transmits both normal and abnormal heart and lung sounds clearly.

There seems to be no major difference between single and double tubing (although in theory double tubing should be better). The tubing should be as short as possible, not compromising your personal comfort. The chest piece should accentuate high (diaphragm) or low frequency (bell, Fig. 2) vibrations (S3 and S4). Diaphragm size (Fig. 3) should be adapted depending on the size of the animal. Small diaphragms (paediatric or neonate) are extremely useful for determining the point of maximum intensity and to avoid interfering noises.
Electronic stethoscopes will become more practical in the future as frequency shifting will permit accentuation or attenuation of particular band frequencies.

**Psychoacoustics**

The human ear does not hear all sounds of equal intensity with equal ease, and the threshold of audibility differs from person to person upon individual hearing ability. Higher volume (in an exponential way) is required for low frequency noise to be heard.

A quiet environment is required for successful auscultation. This includes internal (harsh lung sounds, upper airway noise) and external noise.

**Cardiac auscultation**

**Normal heart sounds**

**First heart sound (S1):** S1 occurs at the onset of ventricular systole. S1 is louder, longer (80 ms) and lower pitched than the second heart sound. The major audible portion of S1 is associated with closure of mitral and tricuspid valves and is best heard over that area. The sound is not generated by the valve cusps slapping together but rather by the sudden acceleration and deceleration of blood and tensing of the valve apparatus. Other elements that contribute to S1 are vibrations due to early ventricular muscle contraction, opening of the semilunar valves at the end of isovolumetric contraction, and vibrations related to rapid early acceleration of blood in the ventricles.

**Second heart sound (S2):** This sound is of higher frequency and shorter duration (60 ms) than the first heart sound, and is best heard over the pulmonic and aortic valves. Vibrations contributing to S2 are produced by early muscular relaxation, blood vibration in the great vessels and opening of the AV valves. S2 occurs at the end of ventricular systole, near the end of the T-wave on ECG and correspond to closure of the aortic (first) and pulmonic valves.

**Normal rhythm**

Normal rhythms in young dogs include sinus rhythm (a regular rhythm) and sinus arrhythmia (regular irregular rhythm). In cats only a regular sinus rhythm is normal (unless they are asleep when a sinus arrhythmia can be noticed). Sinus arrhythmia is due to high vagal tone and is most commonly observed in brachycephalic breeds. It is represented by alternating periods of slower (expiration) and more rapid (inspiration) heart rates, which are usually, but not always, related to respiration. The intensity of S1 and S2 will vary with sinus arrhythmia.

**Areas of auscultation**

Traditional areas of auscultation include the heart apex and heart base at both sides of the chest (left and right). More experienced auscultators will try to narrow these areas down to mitral, tricuspid, aortic and pulmonic valve areas.

**Variations in intensity and rhythm of heart sounds**

**First heart sound (S1):** The first heart sound is loudest in young, thin animals and in those with high sympathetic tone, tachycardia, systemic hypertension, or anaemia.

The first heart sound can be diminished in intensity in obese animals or when there is obscuring for other reasons (pleural and pericardial effusions, diaphragmatic or pericardial hernia). Decreased myocardial contractility or prolonged P-R intervals can also give diminished S1 sounds.

Splitting of S1 can be heard with asynchronous valve closure (3rd degree AV block) or conditions that delay either mitral or tricuspid valve closure (mitral and tricuspid stenosis, bundle branch blocks, ectopic beats).
**Second heart sound (S2):** Physiological audible splitting of S2 is sometimes heard in healthy large breed dogs on inspiration. Pathological splitting of S2 is caused by asynchronous closure of aortic and pulmonic valves. This can be heard with pulmonary hypertension (varying with respiration), atrial septal defects (fixed) and bundle branch blocks.

**Third heart sound (S3):** The third heart sound is of low frequency and is generated by rapid ventricular filling. It is not auscultable in most healthy dogs. The intensity is determined by the rapidity of early diastolic filling, the pressure in the atrium, and the distensibility of the ventricle during early diastole.

Loud S3 often implies myocardial failure (e.g. in dogs with dilated cardiomyopathy) or increased left ventricular stiffness (e.g. cats with hypertrophic cardiomyopathy).

**Fourth heart sound (S4):** The fourth heart sound is produced by atrial systole. Vibrations initiated by forceful ejection of the blood into an already distended or non-compliant ventricle will generate a low-pitched, low frequency heart sound.

**Gallops:** A gallop rhythm is a sequence of three sounds consisting of S1 and S2 combined with S3 and/or S4. In the dog, they often indicate advanced myocardial disease or heart failure. In the cat they are indicators of the presence of reduced compliance of the left ventricle (S3), as seen with hypertrophic and restrictive cardiomyopathy, and/or increased left atrial pressures (S4).

They are classified as protodiastolic (S3), presystolic (S4), or as summation gallops (fusion of S3 and S4).

**Abnormal rhythms:** The intensity of S1 and S2 will vary with most arrhythmias. These alterations are produced by variations in the location of the valves and by the different degrees of filling of the ventricles.

Premature heart beats are heard as early low intensity heart sounds followed by a pause. They are often associated with a pulse pressure variation or deficit. It is impossible to differentiate by the means of auscultation only if the beats are ventricular or supraventricular in origin.

Supraventricular and ventricular tachycardias are characterised by bursts of rapid beats that usually cease abruptly. Differentiation by auscultation only is impossible and electrocardiography is warranted to differentiate them.

**Murmurs** represent sounds of longer duration than the normal heart sounds. They are generated from turbulent blood flow created by abnormal communications between cardiac chambers or through insufficient valves or stenotic in/outflow tracts, or by alterations in blood viscosity.

**Timing and intensity profile**

a. **Systolic (Fig. 4):** A murmur is called holosystolic when S1 and S2 can still clearly be distinguished. A murmur is called pansystolic when S1 and S2 cannot be distinguished. However, there is in the current literature a lot of disagreement about this terminology and both terms are interchanged. ‘Pan’ and ‘holo’ are both Greek terms for ‘the whole of’.

![Murmur timing and profile](image)

**PLATEAU MURMURS**

1. Atrioventricular valve regurgitation:
   - Mitral and tricuspid dysplasia
   - Mitral regurgitation associated with the volume overload of a Patent Ductus Arteriosus
   - AV-canal defects in cats
2. Left-to-right shunting ventricular septal defects

**EJECTION MURMURS (DECRESCEndo AND CRESCendo-DECRESCEndo)**

1. Aortic:
   - obstructive (fixed or dynamic) (aortic stenosis)
   - increased flow (anaemia, stress, fever) (physiological murmur)
2. Pulmonic:
   - obstructive (Pulmonic stenosis)
   - increased flow (ASD, large VSD, as a part of Tetralogy of Fallot)

**Descriptive characteristics of cardiac murmurs**

UK VET - Online
www.ukvet.co.uk
b. Diastolic (very uncommon) (Fig. 5)

![Murmur timing and profile](image)

**Fig. 5: Murmur timing and profile.**

**EARLY DIASTOLIC MURMUR**
1. Aortic regurgitation
2. Pulmonic regurgitation (rarely audible, except after balloon dilation of the valve)

**MID-DIASTOLIC MURMUR**
1. Mitral stenosis
2. Tricuspid stenosis
3. Cor triatriatum sinister

c. Continuous (Fig. 6)

![Murmur timing and profile](image)

**Fig. 6: Murmur timing and profile.**

1. Patent Ductus Arteriosus (99%)
2. Aorticopulmonary window
3. A-V shunts in the thorax

d. To and fro (Fig. 7)

![Murmur timing and profile](image)

**Fig. 7: Murmur timing and profile.**

1. VSD with aortic insufficiency
2. Aortic stenosis with significant aortic regurgitation
3. Pulmonic stenosis and significant pulmonic regurgitation

**Point of maximal intensity (PMI) and radiation (Fig. 8)**

The **left ventricular area** is centred at the cardiac apex. This is a good location to hear mitral murmurs (regurgitation and stenosis). Also gallop sounds generated at the left side of the heart will be best heard in this area. The murmurs of mitral regurgitation often radiate to the **left atrial area**, slightly dorsal and caudal to the left ventricular area.

![Areas of auscultation](image)

**Fig. 8: Areas of auscultation.**

The **aortic area** consists of the traditional aortic valve area on the left side at the level of the mid-heart to heart base. It also includes another area on the right cranial thorax and sometimes the murmur of aortic stenosis is louder on the right hand side.

The murmur of aortic insufficiency is usually best heard in the area of the traditional aortic valve area on the left side at the level of the mid-heart to heart base often radiating to the left heart apex.

The **pulmonic area** consists of the traditional pulmonic valve area cranially at the left heart base but also includes a corresponding area on the right cranial thorax. The murmurs of pulmonic stenosis and pulmonic insufficiency are usually best heard in the pulmonic area on the left, as is the pulmonic component of the second heart sound.

The murmur of a PDA is best heard at the **left heart base area** and it can be very localised in the animal’s axilla. However most continuous murmurs are loud (90% grade 4 and louder) and radiate to the right side of the chest.
The murmurs of a ventricular septal defect (and tricuspid stenosis) are usually best heard in the right ventricular area, as are gallops generated in the right side of the heart.

The right atrial area, located dorsal to the right ventricular area, is where the murmur of tricuspid regurgitation is best heard.

In cats, the sternum often plays the role of an acoustic enhancer and murmurs can be heard best over the sternum (Fig. 9).

Loudness: Murmurs are currently graded from I to VI.
I: very soft and is only detected in a quiet room after prolonged auscultation
II: faint but easily heard
III: moderately loud
IV: very loud but no associated thrill
V: same as grade 4 in loudness but with associated thrill
VI: can be heard with the stethoscope removed from the chest wall

PHONOCARDIOGRAPHY
Phonocardiography is the graphic recording of heart sounds and murmurs. Ideally an ECG should be recorded simultaneously to identify the timing in the cardiac cycle, but if S1 is clearly distinguishable it can be used as the beginning of systole. Examples are illustrated in Figs. 4-8.

REMARKS AND CONCLUSIONS
If a murmur is auscultated in a young animal it can be physiological or pathological. Although the intensity of a heart murmur is not directly correlated with the severity of a lesion, in certain diseases as mitral dysplasia (except for the musical murmurs), aortic and pulmonic stenosis a rough correlation exists. However in other diseases such as ventricular septal defects the louder the murmur the smaller the defect. Small atrial septal defects are not associated with a heart murmur, whereas large ASDs often create an ejection murmur at the level of the pulmonic valve, due to right ventricular volume overload. No exact correlation between heart murmur grade and severity has been established in patent ductus arteriosus, although low frequency continuous murmurs are more frequently associated with small ducti. Tricuspid dysplasia can create quite variable murmurs in intensity, with sometimes complete absence in case of a large defect creating severe laminar, but non-audible, tricuspid regurgitation.

The pathological murmurs are most commonly secondary to congenital heart disease, but one has to be aware that some of the acquired conditions, although rare, can occur at a very young age (for example hypertrophic cardiomyopathy in Maine Coon kittens, dilated cardiomyopathy in Great Dane pups, arrhythmogenic right ventricular cardiomyopathy in Boxer pups). On the other hand, some of the rare congenital heart malformations (e.g. reverse shunts) might not be associated with a heart murmur, but the rest of the clinical examination will indicate the presence of heart disease. Accurate assessment may require Doppler echocardiography and referral to a specialist in veterinary cardiology.

FURTHER READING AND REFERENCES
KVART C. and HÄGGSTRÖM J. (2002) Cardiac auscultation and phonocardiography in dogs, horses and cats. TK I Uppsala AB.

© Illustrations Nicole Van Israël.
These multiple choice questions are based on the above text. Readers are invited to answer the questions as part of the RCVS CPD remote learning program. Answers appear on page 99. In the editorial panel’s view, the percentage scored, should reflect the appropriate proportion of the total time spent reading the article, which can then be recorded on the RCVS CPD recording form.

1. What is the most likely differential diagnosis if a continuous murmur is detected at the left heart base in a puppy:
   a. Subaortic stenosis
   b. Pulmonic stenosis
   c. Patent Ductus arteriosus
   d. Ventricular septal defect
   e. Mitral dysplasia

2. What is the most likely diagnosis if a grade V systolic murmur is heard and felt at the right hemithorax in a cat:
   a. Subaortic stenosis
   b. Pulmonic stenosis
   c. Patent Ductus arteriosus
   d. Ventricular septal defect
   e. Mitral dysplasia

3. What is the most likely differential diagnosis if a systolic murmur with point of maximal intensity at the right hemithorax is detected in a puppy:
   a. Tricuspid dysplasia
   b. Pulmonic stenosis
   c. Patent Ductus arteriosus
   d. Ventricular septal defect
   e. Mitral dysplasia

4. What differentiates a murmur grade IV from a murmur grade V/VI:
   a. Grade V is much louder than grade IV and can be heard with the stethoscope of the chest wall
   b. The difference can only be heard by experienced examiners
   c. Grade IV and V are both loud murmurs but grade V is associated with a thrill
   d. The animal with a grade V murmur shows clinical signs of heart failure
   e. Only phonocardiography can differentiate both