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EFFECTIVE CARDIAC AUSCULTATION

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CARDIAC AUSCULTATION – INTRODUCTION

The stethoscope (and its clinical use) was first described by the French physician, Laennec in 1816. He used a rolled tube of paper to transmit sounds from thorax to ear and subsequently described the relationships between these sounds and underlying thoracic diseases in his treatise De l’Auscultation Mediate. His observations, along with those of other clinical investigators, continue to influence clinical decision-making. Auscultation of the heart and lungs remains an important examination technique for the detection of thoracic disease. This presentation reviews some of the key features of auscultation in dogs and cats, including examination techniques and the interpretation of auscultatory findings relative to acquired and congenital cardiac disorders.

The auscultatory exam is expedient and cost effective. When completed by an experienced clinician, auscultation carries a high predictive value for identification of certain serious heart diseases. Some diagnoses can be attained with high sensitivity and specificity through auscultation. Two examples include the diagnosis of a patent ductus arteriosus (PDA) in a puppy with a continuous murmur and the identification mitral regurgitation (MR) in the older dog with a typical holosystolic, left apical murmur. Auscultation also carries high sensitivity, but lower specificity, for the diagnosis of outflow tract obstructions, septal defects, and sustained or recurrent cardiac arrhythmias. Often the results of auscultation, combined with the signalment, clinical history, and findings on physical diagnosis point to a tentative cardiac or respiratory diagnosis. This presumptive diagnosis is then confirmed, refined, or refuted through Doppler echocardiography, radiography, or electrocardiography. It is emphasized that auscultation is less sensitive for the recognition of patients with cardiomyopathies, pericardial diseases, pulmonary hypertension, trivial valve lesions, or infrequent or sporadic arrhythmias. These are challenging conditions to recognize or confirm without ancillary studies such as cardiac ultrasound or ambulatory electrocardiography.

The essential abnormalities of cardiac auscultation include the following: abnormal heart rate or irregular rhythm (arrhythmia); abnormal intensity of heart sounds (loud, soft, or variable); extra sounds (gallops and clicks); split sounds; cardiac murmurs; and pericardial friction rubs. Different classification systems for heart sounds and murmurs are designed to foster communication among clinicians and carry clinical relevance. This subject is addressed below. A respiratory examination is also performed using the stethoscope, and the breath sounds detected should be classified and assessed as well (these are summarized at the end).

METHODS

Heart sounds are generally too low in frequency or amplitude to be detected by the human ear. Accordingly, careful auscultation is necessary to identify the limited vibrations that fall within our audible frequency-amplitude spectrum. This requires a stethoscope to transfer (not amplify) the sounds from the patient to the examiner.

The choice of a stethoscope is one of personal preference, and there is no ‘best’ model for everyone. Some reasonable stethoscope choices advocated by the author are included in this section. The traditional stethoscope design has a diaphragm and shallow bell (selected by twisting the chest piece head). Many of these instruments permit an exchange to a different-sized (pediatric) diaphragm or (deep) bell (Tyco Harvey Elite®; MDF Procardial C3®). Newer stethoscope models carry one or two tunable diaphragms. A single tunable diaphragm is found on some instruments (3M Littman® Master Classic II and Master Cardiology models); this chest piece is acoustically superior in many ways. However, it is designed for adults and is somewhat large for cats and smaller dogs so the examiner must learn to place it carefully. Another popular stethoscope (3M Littman Cardiology III®) uses both “adult” and “pediatric" sized tunable diaphragms in a single rotating head. For clinicians preferring a light pediatric stethoscope with a small footprint there are very good and economical models available (e.g. 3M Littman Classic II® Pediatric Stethoscope; MDF® Pediatric Stainless Steel Dual Head Stethoscope). These pediatric scopes are especially good for small dogs, cats, ferrets, and birds. While amplified stethoscopes generally are not recommended because of the potential for artifacts and distortion, they can be useful for those hard of hearing or for recording documenting sounds (3M Littman Electronic® 3000 series). Ultimately, the most important part of the stethoscope are the “bits between the earpieces”! A practiced and knowledgeable examiner will succeed with any number of stethoscope models.

Stethoscope designs have improved the acoustics of the instrument, but it must be used properly to obtain optimal clinical results. Of importance are acquiring an instrument of acceptable quality and tube length...
(typically 22 to 28 inches; longer tubes are not better); directing the binaural rostrally (towards the nose) and aligning these to the ear canals by gently adjusting the headset; inserting comfortable earpieces snugly to obtain an airtight seal; and applying the chest pieces with proper technique. The stethoscope chest pieces include two general types: traditional and “tunable”. A traditional flat diaphragm is applied gently but firmly to the chest to accentuate higher frequency sounds such as normal heart and breath sounds. This chest piece is used for 90% of the examination. The traditional shallow or deep bell chest pieces are applied lightly to achieve an airtight seal and enhance auscultation of lower pitched sounds. Examples of the latter include the third and fourth heart sounds and some diastolic murmurs. Combination chest pieces (“tunable diaphragms”) change their frequency response with varying pressure such that flattening the chest piece accentuates higher-pitched sounds (like a typical diaphragm) while gentle pressure brings out the lower pitched sounds like a traditional bell. There are also intermediate levels of pressure that can optimize certain sounds and murmurs; however, careful attention must be directed to subtle pressure changes to optimize the chest piece functionality.

The conditions for auscultation exert a substantial influence on the results of the examination. The room must be quiet, the patient gently restrained by an assistant, and the examiner relaxed. It is preferable for the dog to stand in order to locate the valve areas accurately. A cat can be gently restrained with one hand under the caudal abdomen; this encourages the cat to rest on the forelimbs. The patient must be calm and ventilation and purring controlled if possible. Ventilation (especially panting) that is synchronous to the heart rhythm can mimic cardiac murmurs. Gently holding the mouth closed, whistling, or briefly obstructing the nares are effective maneuvers for reducing ventilation artifacts in dogs. Showing a cat water dripping in a sink, holding the cat, or gently pressing the larynx might reduce the degree of purring. Sound artifacts can be misinterpreted as abnormal heart or lung sounds. These include ventilation and panting (mimics murmurs); twitching (sounds like an extra heart sounds or premature beats); and friction from rubbing the chest piece across hair (sounds like pulmonary crackles or rales). Excessive pressure on the chest can distort the thorax of small animals and create abnormal flow patterns and murmurs.

The clinical examination method involves integration of auscultation with palpation of the precordium and examination of the pulses. Auscultation is preceded by palpation of the arterial pulse to estimate heart rate, rhythm, and pulse strength and character. Ideally, the jugular venous pulse should also be inspected, but practically this is rarely done in healthy patients because of the need to clip neck hair. The thoracic wall over the heart (precordium) is palpated on both sides in order to assess the apical beats. The prominent left apical impulse occurs coincident with opening of the semilunar valves (a systolic thrust). The impact is normally strongest at the left fifth intercostal space near the costochondral junction. A weaker impulse is normally palpable on the right hemithorax at approximately the right third to fourth ICS. Dilatation of the left ventricle displaces the apex beat caudoventrally. Hypertrophy of the left or the right ventricle can produce an impulse more prominent than normal; this is termed a precordial heave. Interpretation of these changes requires considerable experience and practice. Of great value is the identification of a precordial vibration or ‘thrill’. A precordial thrill is the palpable manifestation of a loud murmur and it typically indicates the point of maximal murmur intensity, a descriptor that informs the differential diagnosis.

The entire precordium is examined, with particular attention directed to the cardiac valve areas and the first and second heart sounds, which indicate the onset of systole and diastole, respectively. While the exact anatomic location of the valve areas depends on the species, breed, chest conformation, and size of the heart, a common relative location can be identified. From caudal to cranial are the mitral–tricuspid–pulmonic–aortic with the tricuspid valve on the right side and other valves areas on the left. The author approaches the patient from the caudal perspective, allowing the stethoscope to follow the natural curve of the arm; if one can learn to be ambidextrous in holding the chest piece, the examination can be very efficient. A useful approach in dogs begins with palpation of the left apical impulse where mitral sounds radiate well and the lower-pitched, first heart sound is best heard. The mitral listening area is there and immediately dorsal to the apex. Other valve areas are found from this point. The aortic valve area is located one or two intercostal spaces craniodorsal to the mitral area, and the sharper, higher-pitched second heart sound is usually loudest at that location. Once the aortic second sound is identified, the stethoscope can be moved one interspace cranial and slightly ventral (over the pulmonary valve area). The tricuspid valve area is over the right hemithorax, cranial to the mitral area, and covers a relatively wide area. The pulmonary artery extends dorsally from the pulmonic valve. The left ventricular outflow tract (LVOT) is located near the center of the heart and aortic sounds and aortic ejection murmurs usually radiate to each hemithorax, although these are usually loudest over the aortic area and craniodorsal to the valve.

Cardiac “apex” and cardiac “base” are commonly used expressions to designate the region ventral (ventricles) and dorsal to the atroventricular groove but are not specific for any cardiac valve. Mitral and tricuspid valve sounds and murmurs generally project ventrally to the apical regions. Ventricular septal defects are also louder ventrally (along the sternal edges) in most patients. In contrast, murmurs originating at the semilunar valves or the great arteries are detected best over the base, generally over the left, craniodorsal...
cardiac base. Murmurs that originate in the subvalvular regions of the outflow tracts are often heard both ventrally and craniodorsally. For example, it is common for a loud murmur of subvalvular aortic stenosis (SAS) in dogs to radiate to be well detected at aortic valve area, the mitral valve area, and in the ascending aorta on both left and right sides of the chest.

Valve areas in cats are less distinct and the edges of the chest piece usually cover multiple valves. Consequently, most clinicians do not identify specific feline valve areas but instead use descriptors such as “apical”, “caudal”, “cranial”, and “sternal”. In many cats, the apical impulse is located close to the midline and most auscultation is conducted along the left and the right sternal borders. The first sound is loudest along the left apical (caudal) sternal border. The normal second sound is louder over the left cranial sternal border. A typical murmur of mitral regurgitation is loudest over the caudal left sternal border (apex) whereas typical ejection and functional murmurs are loudest along the left or right cranial sternal borders. However, these are generalizations and distinguishing the source and cause of feline heart murmurs is quite challenging.

TRANSIENT CARDIOVASCULAR SOUNDS

Transient sounds are vibrations of short duration because their genesis depends on abrupt changes in pressure and blood flow. The transient sounds, as well as cardiac murmurs, are timed relative to the first ($S_1$) and second ($S_2$) heart sounds. Following initial activation of the ventricles and the rapid development of ventricular pressure, the first sound is heard indicating the onset of systole for the clinician. At the end of ventricular ejection, the second sound is generated, heralding the onset of diastole to the clinician. Any transient sounds detected during systole or diastole are considered abnormal in small animals.

Both $S_1$ and $S_2$ are relatively high-frequency sounds. The first sound is associated with vibrations of the cardiac structures and blood pool near the time of atrioventricular (AV) valve closure, while the second sound is caused by vibrations occurring at the time of closure of the aortic and pulmonic valves. The first sound is lower-pitched (duller), longer, and more obvious over the left apex. The relatively sharper and shorter second sound is more prominent over the aortic and pulmonic valve areas and in some normal dogs is closely split during inspiration. These two transient sounds can become abnormal in certain conditions. Pericardial or pleural effusions and myocardial failure (as with dilated cardiomyopathy) decrease the intensity of the heart sounds. Conversely, both heart sounds tend to be relatively loud in healthy animals under high sympathetic drive or those with thin body conformation.

Diastolic sounds or gallops are abnormal in dogs and in cats. These are audible manifestations of filling sounds (that would be considered normal sounds in larger animal species). Gallops are lower-frequency sounds and associated with vibrations surrounding either sudden termination of early ventricular filling ($S_2$) or atrial contraction and end-diastolic ventricular filling ($S_3$). These sounds indicate diastolic dysfunction when detected in dogs or cats. A third sound is typical of a very diseased ventricle, reduced diastolic chamber compliance with ventricular filling occurring under high venous pressures. Hence, a ventricular gallop is sometimes considered a heart failure sound. An atrial gallop is typically associated with impaired ventricular relaxation (as occurs with feline hypertrophic cardiomyopathy or hypertensive heart disease) and probably stems from the brief, compensatory increase in atrial pressure needed to fill the ventricle at end-diastole. This enhancement of end-diastolic filling leads to an audible sound timed between the P-wave and QRS-complex of the ECG (it is called $S_4$).

Diastolic sounds or gallops can be loudest over the pulmonic valve or pulmonary artery (left craniodorsal). Systolic clicks are also detected in some cats with hypertrophic cardiomyopathy, but without a phonocardiograph recording, the distinction between an $S_4$ and early systolic click in a cat can be very challenging. These are also detected in some dogs and cats with mitral valve dysplasia. Ejection clicks are infrequently detected in dogs affected by valvular pulmonic stenosis or pulmonary hypertension; these tend to be loudest over the pulmonic valve or pulmonary artery (left craniodorsal).

In many patients, the first recognition of a cardiac arrhythmia occurs during auscultation and palpation of the femoral arterial pulse. Auscultatory findings in arrhythmias and conduction disturbances include abnormal heart sounds and associated with vibrations surrounding either sudden termination of early ventricular filling ($S_2$) or atrial contraction and end-diastolic ventricular filling ($S_3$). These sounds indicate diastolic dysfunction when detected in dogs or cats. A third sound is typical of a very diseased ventricle, reduced diastolic chamber compliance with ventricular filling occurring under high venous pressures. Hence, a ventricular gallop is sometimes considered a heart failure sound. An atrial gallop is typically associated with impaired ventricular relaxation (as occurs with feline hypertrophic cardiomyopathy or hypertensive heart disease) and probably stems from the brief, compensatory increase in atrial pressure needed to fill the ventricle at end-diastole. This enhancement of end-diastolic filling leads to an audible sound timed between the P-wave and QRS-complex of the ECG (it is called $S_4$).

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Cardiac murmurs are prolonged audible vibrations. Although murmurs are a hallmark of many cardiac diseases, very often murmurs are innocent or functional (i.e., the heart is structurally normal). Murmurs are associated with high velocity blood flow (typically >1.6 m/sec) and vibrations that develop about disturbed or turbulent flow; these generate changes in the flow stream capable of generating audible sound waves. Turbulence and wake fluctuations are more common when flow velocity increases, viscosity of the blood decreases, or when flow moves through a larger blood vessel.

Clinically the causes of cardiac murmurs relate to a number of common conditions. Adrenergic stimulation, secondary to anxiety, fear, exercise, fever, drugs, or hyperthyroidism, is a common denominator underlying many functional heart murmurs. Both sympathomimetic activation (by increasing contractility) and peripheral vasodilation (by reducing afterload) can increase ventricular systolic function. This can lead to higher ejection velocity into the great vessels, dynamic obstruction in either ventricle and foster high-velocity or turbulent blood flow. Anemia decreases viscosity of blood and like hyperthyroidism is associated with increased sympathetic activity and peripheral vasodilation. Increased ventricular stroke volume can cause mild-to-moderate increases in ejection velocities and result in an ejection murmur in the absence of any valvular obstruction. Examples include bradycardias (pronounced sinus arrhythmia, AV block, and athletic heart) and atrial septal defect. Structural heart lesions offer pathways for blood to flow from high to low-pressure zones; this pathophysiologic representation is the most common explanation for pathologic (organic) murmurs. Examples include flow across a restrictive ventricular septal defect (VSD), a stenotic valve, an incompetent valve, or through an aortic to pulmonary shunt (as with PDA). A common reason for heart murmurs in cats are the primary or secondary cardiomyopathies; murmurs stem from either secondary mitral regurgitation or dynamic ventricular obstruction. Another common cause of murmurs in older cats is aortic dilatation (aortoannular ectasia) with discrete upper septal thickening (in the subaortic ventricular septum).

Cardiac murmurs should be described based on timing and shape, intensity (loudness), and point of maximal intensity (PMI). Additional qualifiers include the murmur radiation, pitch and quality. The general timing of the murmur is designated as systolic, diastolic, continuous, or to-and-fro (systolic – diastolic with a pause a S₂). The adjectives “proto”, “meso”, and “tele” are sometimes used to indicate early, middle, and late. Within this timing are descriptions qualifying the onset/end and relative loudness of the murmur as characterized by a phonocardiographic recording. This creates an impression of the murmur’s “shape”. For example, ejection murmurs cannot begin until after S₁ and must end by S₂ and peak in early to mid-systole unless caused by a severe obstruction to flow. The shape of ejection murmurs is crescendo-decrescendo (diamond-shaped). In
The point of maximal intensity is communicated by indicating the location, valve area, or intercostal space where the murmur is loudest. A murmur usually projects from the PMI in the direction of abnormal blood flow. Murmurs can also radiate through solid structures to the chest wall, as often occurs with MR radiating to the left apex. The radiation of a loud cardiac murmur can be extensive and can off some clues about the genesis of the murmur. However, very loud murmurs tend to radiate widely and can sometimes be confusing. Pitch and quality pertain to the frequency and subjective assessment of the murmur by the examiner. Murmurs consisting of one fundamental frequency with overtones are described as “musical”, whereas murmurs of mixed frequencies are typically noted to be “harsh.” Most murmurs are of mixed frequency.

Functional Murmurs are those unassociated with obvious cardiac pathology. These murmurs arise from physiologic changes (see above) or undefined causes (so-called innocent murmurs). In small animals, nearly all functional murmurs are systolic and the murmur is typically ejection in shape, meaning it begins after $S_2$ and ends prior to $S_3$. The PMI of the functional ejection murmur is at or adjacent to the aortic or the pulmonary valve and the murmur radiates craniodorsally into the aorta or pulmonary artery. In general, functional murmurs are soft, grade 1-2/6, and relatively brief with both heart sounds evident. In puppies, most innocent murmurs become softer during the vaccination sequence and eventually disappear (at about 4 months of age). A classical innocent murmur has a humming or musical character likely related to a vibrating intracardiac structure. Functional ejection murmurs in larger canine breeds can persist throughout life. In many of these dogs, the murmur is protomesosystolic, grade 2 to 3/6 in intensity, and transiently accentuates following exercise to a grade 3 to 4/6 intensity. These ejection murmurs are evident without any accompanying structural lesions on 2D echocardiography. In fact, echocardiographic correlates to a functional murmur are inconsistent with the most frequent being mildly increased aortic ejection velocity on spectral Doppler imaging (1.7 to 2.4 m/s). It is nearly impossible to distinguish functional ejection murmurs from those due to trivial LVOT stenosis based on velocity alone. Some breeds, such as boxers and bull terriers, have a relatively narrow aorta, and ejection murmurs in some of these dogs could represent the effect of ventriculo-annular disproportion as opposed to true subvalvular aortic stenosis (SAS). This issue is controversial and largely unresolved. It should be noted, however, that in breeds rarely affected by SAS (such as greyhounds), functional ejection murmurs are also associated with mildly elevated aortic ejection velocities. Systolic murmurs over the cranial sternal borders are common in cats without echocardiographic evidence of significant heart disease. Many of these murmurs arise from flow across the LVOT (often in to a dilated aorta). Others are caused by dynamic intraventricular obstruction (see next section).

Dynamic mid-ventricular and outflow tract obstructions are another potential cause of systolic murmurs in dogs and cats. The classic auscultatory feature is a mid-to-late systolic murmur associated with emptying of ventricular blood and physical contract between cardiac walls, papillary muscle, or mitral valve elements. The Doppler signal is dagger-shaped, typical of obstruction that begins dynamically, as ejection progresses and ventricular volume decreases. The heart can be structurally normal or hypertrophied in these cases. Mid-ventricular obstruction of the left ventricle (LV) is often from dehydration or concentric hypertrophy of the chamber. When the murmur arises in the right ventricle (RV), the obstruction is often between the free-wall and the supraventricular crest and is often explained by high sympathetic tone, ventricular septal thickening, or right ventricular hypertrophy. Dynamic RV obstruction is especially common in stressed and dehydrated cats.

Whether these murmurs should be considered functional or a consequence of pathology depends largely on the findings on 2D echocardiography. Some cats with hypertrophic cardiomyopathy (HCM) have prominent mid-ventricular obstruction. The finding is also common in cats with hypertrophy thickening from systemic hypertension and hyperthyroidism. Mid-ventricular obstruction differs from dynamic LVOT obstruction caused by systolic anterior motion of the mitral valve associated with HCM in cats and mitral valve malformations in cats and dogs. In that situation, elements of the anterior (septal) mitral leaflet are pulled towards the ventricular septum creating both dynamic obstruction and an eccentric jet of MR.
Aortic Stenosis  Obstruction in the LVOT is most often causes by subvalvular aortic stenosis (SAS) in dogs and by HCM in cats. The lesions of SAS include a congenital, subvalvular, fibrous obstruction below the aortic valve that can be discrete or envelop the outflow tract to the base of the aortic valve proper. Other causes of LVOT obstruction are valvular aortic stenosis (AS), mitral valve malformations, and chronic infective endocarditis of the aortic valve. Depending on the lesion, aortic regurgitation (AR) will occur to varying degrees. In most cases, AR is a Doppler diagnosis and the regurgitation is silent to auscultation.

The murmur of (S)AS is systolic, flow dependent, and crescendo-decrescendo in configuration. As with most ejection murmurs, the murmur intensifies following exercise, inotropic stimulation, a ventricular premature beat, a long pause in sinus arrhythmia, or with increases in venous return and stroke volume. The typical PMI of SAS is over the left thorax at the aortic/subaortic region with strong radiation of the murmur apically towards the mitral area and craniodorsally into the ascending aorta. The murmur tends to project into the carotids and even radiate to the skull. The murmur of SAS can be loudest over the right dorsal cardiac base owing to radiation into a dilated ascending aorta. In pure valvular or rare supravalvular AS, the murmur can be loudest on either side of the thorax. With moderate to severe (S)AS the murmur peaks later in systole – sounding more holosystolic than ejection in configuration; at the same time the arterial pulses are hypokinetic and late-rising. If a diastolic murmur of aortic regurgitation develops with (S)AS, both a systolic and a decrescendo diastolic murmur are evident. This leads to the to-and-fro murmur of AS/AR and to bounding arterial pulses.

Pulmonic Valve Stenosis (PS) is a congenital malformation of the pulmonary valve characterized by varying degrees of valve thickening, leaflet fusion and tethering, and hypoplasia of the annulus or along the base of the valve. It is most common in dogs and relatively rare in cats (and more likely to be infundibular in this species). The resultant murmur is systolic, crescendo–decrescendo, and loudest over the pulmonary valve (at the left second to third ICS) with strong radiation dorsally into the post-stenotic dilatation of the main pulmonary. Thus, the murmur tends to be heard very well over the left, cranial to dorsal cardiac base and is sometimes confused with a PDA for this reason. An early systolic ejection click might be detected with careful auscultation (from a fused but mobile valve). A concurrent murmur of tricuspid regurgitation (TR) is often heard over the tricuspid valve area caused by secondary right ventricular enlargement or tricuspid dysplasia. Pulmonary regurgitation is invariably present on Doppler studies but rarely audible. However, following successful balloon catheter valvuloplasty, there is often moderate to severe pulmonary insufficiency creating an early to mid-diastolic decrescendo murmur of pulmonary regurgitation. Rarely severe pulmonary regurgitation is present with native valvular dysplasia and the associated to-and-fro murmur of PS/PR is easily confused with a PDA. Congenital PS is often associated with a prominent jugular pulse (giant A-wave). In contrast to dogs with SAS, the arterial pulses should be normal in character unless there is heart failure.

Mitrail Regurgitation is one of the most common and important flow disturbances responsible for a pathologic systolic heart murmur. MR develops secondary to malfunction of any portion of the mitral apparatus. Causes include congenital mitral dysplasia, myxomatous degeneration of the valve (endocardiosis) in the dog, infective endocarditis, redundancy or rupture of a chordae tendineae (in the dog), and causes of left ventricular dilatation or hypertrophy, such as cardiomyopathy, hyperthyroidism, systemic hypertension, and PDA. This murmur is loudest over the mitral valve area or the palpable left apex where it is projects well (or near the left sternal edge in cats). MR murmurs radiate both dorsally and to the right (usually one grade softer on the right hemithorax). The resultant murmurs are crescendo in peracute leakage (from equilibration of LV-LA pressures) or in mild cases (as the regurgitant orifice closes in late systole). The murmur is soft in hypotensive patients; alternatively, systemic hypertension can amplify the intensity. The musical systolic “whoop” is a striking frequency phenomenon in some dogs. Progressive increase in the intensity of the first heart sound is a unique feature of MR in dogs with valvular endocardiosis and probably indicates cardiac dilatation with maintenance of left ventricular systolic function. Additionally, the intensity of the MR murmur usually increases with the degree of valvular incompetency (assuming normal ABP) in dogs with myxomatous disease. The intensity of a MR murmur in other causes of MR is not as reliably correlated to the severity. Cats with hypertrophic cardiomyopathy often have a labile murmur of MR related to dynamic left ventricular outflow tract obstruction and systolic anterior motion of the mitral valve.

Tricuspid regurgitation is another common cardiac murmur and in many ways is similar to mitral regurgitation. Causes include valve malformation, degenerative valve disease (endocardiosis), right ventricular enlargement (from pulmonic valve stenosis, right sided cardiomyopathy, chronic bradycardia, or pulmonary hypertension), dicrofilariasis, transvenous pacing leads, and (very rarely) tricuspid endocarditis. The PMI of this murmur is the tricuspid valve area on the right, and dorsal radiation is typical. In young puppies with TV dysplasia (e.g., Labrador retrievers), the murmur can be very soft and readily missed. It can be difficult to distinguish TR from the radiating murmur of MR. The following support concurrent TR in the this setting: a
prominent jugular pulse, right precordial thrill, different frequency murmur than that heard at the left side, louder right than left-sided murmur, or right-sided CHF. The murmur of TR is often very loud in the setting of pulmonary hypertension. Eccentric jets of TR from degenerative TV prolapse can also lead to very prominent murmurs of TR along with a right sided thrill.

**Ventricular Septal Defect** is the most common congenital heart malformation of cats, and also occurs with some frequency in dogs. A large nonrestrictive VSD (i.e., no substantial pressure difference between the ventricles) is also part of the tetralogy of Fallot. When the defect communicates the left ventricular outlet with the perimembranous ventricular septum, the murmur is loudest just below the tricuspid valve, along the right sternal border. When the defect is subarterial, communicating the subaortic septum with the subpulmonary septum, the murmur may be most prominent over the craniodorsal left cardiac base (similar to PS). If the aortic valve has prolapsed into the ventricular septal defect, there may be a diastolic murmur of aortic regurgitation as well.

**Aortic Regurgitation** is the most important diastolic cardiac murmur. Causes include infective endocarditis, congenital aortic valve disease, prolapse of an aortic valve cusp into a subaortic ventricular septal defect, and aortic root dilatation in aged cats. This murmur is a long, diastolic, decrescendo murmur heard best over the aortic valve at the left hemithorax. The murmur is also well heard at the right cardiac base.

Diastolic murmurs are otherwise rare in dogs and cats. Differential diagnosis includes the soft, low-pitched rumble of congenital mitral or tricuspid stenosis but these are very rare conditions. There is often concurrent ativoventricular valvular regurgitation, which may lead to a systolic murmur.

**Patent Ductus Arteriosus** is the most important cause of a continuous murmur and is described as “distant” and “machinery” (like a machine shop) in quality. The murmur must be carefully located dorsally on the cranial left base, over the main pulmonary artery (which is the sink for the shunt). Although it is continuous, the murmur does vary and loudness peaks around the time of S2. Often there is concurrent mitral regurgitation (due to left ventricular dilatation) which can be responsible for a systolic murmur over the left apex. The stethoscope should be “inched” up and back from the left apex to the PMI of the continuous murmur at the left base. In terms of differential diagnosis, continuous murmurs (bruits) can be detected over congenital or acquired arteriovenous fistulas, including those associated with thyroid carcinomas or limb injuries. Within the thorax, coronary artery to pulmonary artery fistulas and systemic arterial to pulmonary artery vascular malformations can result in a continuous cardiac murmur. Rare cases of reversed PDA usually have no murmur or a soft ejection murmur with a tympanic and sometimes split second heart sound.

References