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Clinical significance of cardiac murmurs:

Get the sound and rhythm!

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Murmurs arise when blood flow becomes turbulent, causing vibration of cardiovascular structures. Physiological murmurs or functional murmurs result from turbulent flow associated with normal blood flows at high velocity. Pathological murmurs are caused by a pathological condition such as valvular regurgitation or congenital cardiac disease. When a murmur is diagnosed, we first need to identify the source of the murmur and then assess its significance. We therefore need to classify murmurs based upon their timing (systolic or diastolic) and duration, location and radiation, intensity (1 to 6 scale) and character. The intensity of the murmur is often related to the severity of the regurgitation with louder murmurs being more severe. However, this is not always the case, especially not for a ventricular septal defect, for a musical murmur or when heart failure is present. The timing of the murmur, such as systolic (between S1 and S2), diastolic (between S2 and S1) or continuous, and the point of maximal intensity already allow to determine the origin of the murmur. The duration is described as early, mid, late, holo- or pansystolic or –diastolic. Murmurs that obscure the normal heart sounds (pansystolic or pandiastolic) are more severe than those that do not obscure heart sounds (holosystolic or holodiastolic). The radiation and character are also related to the significance of the murmur.

The most important cardiac murmurs are listed below.

Systolic murmurs

During systole, the mitral valve and tricuspid valve should be closed and blood is ejected into the great arteries. Thus, in general, a left-sided systolic murmur is caused by mitral regurgitation or by the normal ejection of blood into the great arteries. A right-sided systolic murmur is caused by tricuspid regurgitation or, if the murmur is loud, harsh and located slightly below the tricuspid valve area, by a ventricular septal defect.

LEFT side systolic: Ejection murmur or flow murmur (physiological murmur)

During systole a large volume of blood is ejected into the great arteries which might be associated with a systolic (functional) flow murmur degree 1-3/6, best heard over the aortic

valve area. The intensity often varies with slightly changing heart rates. Typically, this soft murmur has a crescendo-decrescendo shape, occurs in early to mid-systole, and ends before the onset of the second heart sound. Flow murmurs do not tend to obscure heart sounds. Up to 60% of normal horses present an ejection-type murmur in early to mid-systole. Flow murmurs are also frequently detected in animals in which blood viscosity is low (hypoproteinaemia, anaemia) or cardiac output is high (foals, colic). Therefore, when an ejection-type murmur is detected in a colic horse, re-examination should be performed after the colic episode.

LEFT side systolic: Mitral regurgitation

Mitral regurgitation usually results in a holosystolic plateau type murmur over the mitral valve area, radiating dorsally with an intensity varying between 1/6 and 5/6. The backflow of blood into the left atrium results in an increased atrial filling with more blood flow during early ventricular filling often producing a louder third heart sound. Increased atrial pressures impede pulmonary blood flow. Therefore, clinical signs to be expected from severe mitral regurgitation are especially decreased performance with increased respiratory rates, excessive sweating and slower recovery after exercise.

Murmurs of 1-2/6 are usually of no clinical importance and are associated with trivial or mild regurgitation. If the intensity is 3/6 or more, the murmur is more likely to have clinical importance or may become clinically important in future. Cardiac ultrasound is indicated to assess the importance of the regurgitation and determine if secondary changes (e.g. left atrial dilatation) have occurred. Often, ultrasound examination reveals that the regurgitation is mild to moderate, is still well compensated, with no or limited left atrial dilatation, having no or only limited impact on performance. But, although the regurgitation *per se* may not affect performance, one should be aware that such regurgitation increases the risk for development of atrial fibrillation. In case AF develops, performance capacity is likely to reduce. Severe mitral regurgitation is often associated with a 4/6 or 5/6 holo- or pansystolic murmur, and affects performance. It is likely to progress to heart failure. During this evolution towards heart failure, horses may remain relatively stable for a certain period but when atrial fibrillation develops, clinical signs suddenly get worse. Chordal rupture is often associated with a very loud, rough or vibrating pansystolic murmur, degree 4/6 or often 5/6. This condition is usually severe, especially when a 'major' chorda is ruptured, rapidly progressing and often results in development of respiratory distress, tachycardia, pulmonary hypertension, heart failure and death. Rupture of a 'minor' chord may be tolerated for a prolonged period of time, depending on the importance of the chord.

Less frequently, mitral regurgitation presents as a 1/6 -4/6 holo (or mid-to-late) systolic crescendo murmur. The crescendo character is caused by a valvular prolapse with increasing regurgitation towards the end of systole. Such crescendo murmurs tend to be tolerated well and show less progression than the holosystolic band-shaped murmurs.

In general, it can be said that mitral regurgitation occurs frequently in horses. Most commonly, horses still perform up to their expectations. Mitral regurgitation often progresses over time but progression is usually slow. Atrial fibrillation is a common sequel to mitral regurgitation. Cardiac failure is relatively rare, but if it occurs, it is often associated with severe mitral regurgitation.

RIGHT side systolic: Tricuspid regurgitation

Tricuspid regurgitation is very frequently encountered in normal horses, probably because of the complex structure of the valvular apparatus. Mild to moderate regurgitation generally does not affect performance although in some occasions it may increase the risk for development of atrial fibrillation. Asymptomatic tricuspid regurgitation has reported to be more prevalent in Thoroughbreds and Standardbreds, presumably due to training related cardiac hypertrophy.

The murmur usually is holosystolic, soft blowing and grade 1/6 to 4/6. The point of maximal intensity is located near the 3rd- 4th intercostal space just below the level of the shoulder joint.

Only severe tricuspid regurgitation may be related to exercise intolerance. The most common cause of severe tricuspid regurgitation is left cardiac failure. In these horses, pulmonary artery dilatation and right cardiac dilatation with paradoxical septal motion may be found. Tricuspid regurgitation may result in increased right atrial pressure and the occurrence of pathological jugular venous pulsation and ventral oedema.

RIGHT side systolic: interventricular septal defect

Ventricular septal defect (VSD) is the commonest congenital heart defect in horses and is most commonly located in the membranous or semi-membranous portion of the interventricular septum, just below the aortic root. A harsh band-shaped pansystolic murmur, degree 3/6 or more is present slightly ventral to the tricuspid valve area. Generally, a very loud murmur accompanied by a precordial thrill (degree 5/6) is found. The murmur of a VSD radiates ventrally toward the sternum, while the murmur of tricuspid valve regurgitation radiates dorsally. Due to the left-to-right shunt, there is a right ventricular volume overload. During systole, this increased volume of blood is ejected into the pulmonary artery producing

a systolic ejection murmur over the pulmonary valve area (left side), which is referred to as a 'relative pulmonary stenosis'. In some animals, an aortic regurgitation murmur is associated with the VSD because the aortic root is no longer 'supported' by the interventricular septum, resulting in aortic valve instability or prolapse into the defect.

When the VSD is located in the muscular portion of the interventricular septum, the murmur not necessarily radiates toward the sternum. A subpulmonic VSD results in a pansystolic murmur with the point of maximal intensity on the left side, just underneath the pulmonary valve. Horses with a Tetralogy of Fallot show a left-sided and right-sided holo- to pansystolic murmur, with the loudest murmur over the pulmonic valve area (due to pulmonary stenosis).

It should be emphasized that the intensity of the VSD murmur poorly correlates to the size of the VSD. Therefore, cardiac ultrasound is mandatory to determine the clinical importance of the VSD. The size of the defect and the blood flow velocity through the defect should be measured to assess the consequences on performance. The defect should be measured in 2 perpendicular planes. The maximal flow velocity should be measured with continuous wave Doppler. A small VSD will only result in a small amount of blood flowing from left to right during systole. As a result the pressure difference between left and right ventricle will be maintained and therefore the defect is called 'restrictive'. Because of the big pressure difference between both ventricles the flow velocity is very high, which carries a better prognosis.

Restrictive VSDs may only have little or even no effect on performance. As such, the VSD might be found by coincidence in otherwise healthy animals. Larger VSDs lead to pulmonary artery dilatation and left atrial dilatation, atrial fibrillation and eventually, cardiac failure and death. As a general rule of thumb, mature horses with a VSD of less than 2.5 cm or less than $1/3^{\text{rd}}$ of the aortic root, and a shunt velocity of more than 4-5 m/s have a relatively favourable prognosis and might even compete in more demanding sports. Prognosis is worse when the condition is associated with aortic regurgitation or prolapse of the aortic valve into the VSD.

Diastolic murmurs

Physiological murmurs in diastole are short in duration and occur in early or late diastole. Pathological diastolic murmurs are holo- or pandsystolic, often decrescendo.

LEFT side short early or late diastolic: physiological murmur

A functional or physiological diastolic murmur is soft and short in duration. Early in diastole, between S2 and S3, a short murmur may be heard in some horses. The murmur may be soft and blowing or may be rather musical ('whoop'). Although it is often referred to as '2-year old squeak' it may occur at any age. This murmur can be heard from the left hemithorax. It is associated with rapid ventricular filling and therefore physiological. It may increase in intensity after mild exercise or stress, or disappear completely.

Late diastolic or presystolic murmurs are also short, occur between S4 and S1 and may sometimes be difficult to distinguish from S4 and S1. The murmur can be heard at either side of the chest. This is also a physiological murmur and has no clinical significance at all.

LEFT side diastolic: aortic regurgitation

The murmur of aortic insufficiency is best heard from the left thorax with the point of maximal intensity over the aortic valve area in the left fourth intercostal space, radiating ventrally. The quality is often musical which is a typical feature of aortic regurgitation and is caused by vibration of cardiac structures. However, the murmur may also be soft blowing (easily missed!) or harsh. Generally, the murmur is decrescendo because of the gradual fall in pressure gradient between left ventricle and aorta throughout diastole. Especially for the musical murmurs, the intensity of the murmur does not reflect its clinical significance.

Aortic regurgitation might be caused by congenital, degenerative, inflammatory or idiopathic valvular lesions and appears more commonly in older horses. Small nodules on the aortic valve are commonly found. Nodules on the aortic valve are due to degenerative lesions and are most frequently found in older horses, especially on the right coronary cusp. Because of the large diameter of the equine aorta, the nodules will not result in aortic stenosis. Although endocarditis is not frequently encountered in horses, the aortic valve is most commonly affected. Ventricular septal defects, located just beneath the aortic root can cause severe aortic regurgitation because of instability of the aortic root and aortic valves. On echocardiography, the aortic valves may be seen prolapsing toward the septal defect.

Mild to moderate aortic regurgitation will have no effect on performance: the left ventricle is able to compensate for the limited volume overload and, during exercise, the increase in heart rate shortens the diastolic time and thus the time of regurgitation. Aortic regurgitation is likely to slowly deteriorate with time. Moderate to severe regurgitation results in volume overload of the left ventricle, which results in left ventricular dilatation. Horses with aortic regurgitation should be monitored regularly because ventricular dilatation may cause

dilatation of the mitral valve annulus, resulting in mitral regurgitation and a left-sided holosystolic murmur. These animals carry a poor prognosis.

Palpation of the arterial pulse provides an assessment of the severity of the condition, rather than the grade of the murmur. Severe aortic regurgitation results in a bounding pulse because the left ventricular volume overload results in an increased stroke volume and increased systolic pressure, while the rapid diastolic run-off results in a decreased diastolic pressure. Therefore, a larger arterial pressure difference is present between systole and diastole, making the pulse bounding, hyperkinetic.

Most important in horses with aortic regurgitation is that they tend to have more ventricular arrhythmias compared to their age-matched peers. The exact mechanism for the arrhythmias is not fully understood yet but ventricular arrhythmias carry a risk for sudden collapse or death. Animals with aortic valve incompetence should therefore always be monitored using ambulatory and especially exercising ECG recordings to diagnose the presence of ventricular arrhythmias.

As it is difficult to assess the significance of aortic regurgitation based upon auscultation, ultrasound examination is necessary, especially when intensity is 3/6 or more. Horses should be re-examined by ultrasound and exercise ECG at least yearly when there is moderate to severe aortic regurgitation. On two-dimensional echocardiography left ventricular dilatation might be seen and the left ventricular apex might become more rounded, giving the ventricle a turgid, globoid appearance. Left atrial dilatation may become apparent especially if mitral regurgitation occurs as a result of mitral annulus dilatation. On M-mode diastolic vibration of the aortic valve and the mitral valve are often detected. Colour-flow Doppler echocardiography allows demonstrating the size of the regurgitant jet and continuous wave Doppler can be used to assess pressure gradients between left ventricle and aorta.

Continuous murmurs

The most common reasons for a continuous murmur are a combination of a systolic and diastolic murmur (e.g. mitral regurgitation and aortic regurgitation), an aortocardiac fistula or an aortopulmonary fistula.

Aortocardiac fistula

The typical findings of an aortocardiac fistula are a continuous machinery murmur with the point of maximal intensity over the right side of the thorax, a bounding arterial pulsation and ventricular tachycardia. Many horses present signs of colic. The fistula can be identified on ultrasound. Such horses often carry a grave prognosis.

Aortopulmonary fistula

The condition is typically seen in Friesian horses (can occur at any age but most frequently between 2 and 6 years of age). It represents a connection between the aorta and pulmonary artery due to a wall rupture, near the location of the ductus arteriosus. On auscultation, a soft holosystolic and an early-mid or holodiastolic murmur dorsal to the aortic valve area might be heard. Aortopulmonary fistulation is typically associated with bounding arterial pulsation and often with (sinus) tachycardia. The condition can be acute (hours), subacute or chronic (weeks to months) but is always fatal. The fistula can be detected on ultrasound but requires specific images of the region of the ductus arteriosus.

Conclusion

Auscultation provides an excellent tool to diagnose valvular regurgitation or other congenital or acquired cardiovascular disease. Classification of the murmur allows to make an accurate diagnosis and to decide whether or not further examinations are required. Besides the effect of the murmur itself on blood flow, presence or risk for arrhythmias play a major role in the assessment of the cardiac patient. It is important to realize the association between mitral regurgitation and atrial fibrillation, and between aortic regurgitation and ventricular arrhythmias (increased risk for sudden death).