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CARDIAC MURMURS IN HORSES: SHOULD WE WORRY?

Physiological murmurs (or functional murmurs or flow murmurs) result from turbulent flow associated with normal blood flow at high velocity. Pathological murmurs are caused by a pathological condition such as valvular regurgitation or congenital cardiac disease. The most important cardiac murmurs are listed below.

SYSTOLIC MURMURS

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. Up to 60% of normal horses present an ejection-type murmur.

LEFT side systolic: Mitral regurgitation

Mitral regurgitation (MR) 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. Murmurs of 1-2/6 are usually of no clinical importance. If the intensity is 3/6 or more, the murmur is more likely to have clinical importance or may become clinically important in future. 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 future. Severe MR is often associated with a 4/6 or 5/6 holosystolic murmur, and affects performance. 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.

RIGHT side systolic: Tricuspid regurgitation

Mild to moderate regurgitation is very frequently encountered in normal horses and generally does not affect performance. In some occasions it may increase the risk for development of atrial fibrillation. The murmur usually is holosystolic, soft blowing and grade 1/6 to 4/6.

Only severe tricuspid regurgitation may be related to exercise intolerance. The most common cause of severe tricuspid regurgitation is left cardiac failure.

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, the murmur is very loud and accompanied by a precordial thrill (degree 5/6). The murmur of a VSD radiates ventrally toward the sternum. In some animals, an aortic regurgitation murmur is associated with the VSD because of associated aortic regurgitation. The point of maximal intensity is often located elsewhere in case of a muscular or subpulmonary VSD.

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.

DIASTOLIC MURMURS

LEFT side short early or late diastolic: physiological murmur

A functional or physiological diastolic murmur is soft and short in duration and occurs in early diastole, between S2 and S3 as a whoop or squeaky sound, or in late diastole, between S4 and S1.
"LEFT side diastolic aortic regurgitation"

The murmur of AR 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 AR and is caused by vibration of cardiac structures. However, the murmur may also be soft blowing (easily missed!) or harsh. Especially for the musical murmurs, the intensity of the murmur does not reflect its clinical significance.

Aortic regurgitation appears more commonly in older horses. Mild to moderate AR will have no effect on performance. Moderate to severe regurgitation results in ventricular volume overload and dilatation and may affect performance. Palpation of the arterial pulse provides an assessment of the severity of the condition. Severe AR results in a bounding, hyperkinetic pulse.

Most important in horses with AR is that they tend to have more ventricular arrhythmias compared to their age-matched peers. The ventricular arrhythmias carry a risk for sudden collapse or death. Animals with aortic valve incompetence should therefore always be monitored using exercising ECG recordings to diagnose the presence of ventricular arrhythmias.

As it is difficult to assess the significance of AR 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 AR.

CONTINUOUS MURMURS

The most common reasons for a continuous murmur are a combination of a systolic and diastolic murmur (e.g. MR and AR), 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

This condition occurs in (adult) Friesian horses as a connection between the aorta and pulmonary artery due to a wall rupture. On auscultation, a soft (often 1/6-3/6) holosystolic and an early-mid or holodiastolic murmur dorsal to the aortic valve area might be heard. Usually a bounding arterial pulsation and (sinus) tachycardia are found. 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.
CARDIAC ARRHYTHMIAS IN HORSES: ‘IS THERE A RISK DOCTOR?’

Horses commonly show cardiac dysrhythmias which may occur at rest and/or during exercise. Careful auscultation, which means sufficiently long at both sides of the thorax, will often allow to make a presumptive diagnosis or at least have a strong suspicion about the origin of certain dysrhythmias at rest, especially whether they are physiological or pathological. For most pathological dysrhythmias, assessment during exercise (exercise ECG test) is essential. Attention should also be paid to presence of murmurs as these might be associated with arrhythmias (e.g. mitral regurgitation and atrial fibrillation or aortic regurgitation and ventricular ectopy). The definitive diagnosis of the type of dysrhythmia can only be made by electrocardiography (ECG). Nowadays, ECG equipment is easy to use and affordable to private practice which allows on site recording at rest, during exercise or during 24 hours. If one is not familiar with interpretation of the data, digital files can be easily sent to more experienced colleagues or online services.

When assessing performance horses, it is important to differentiate between innocent, physiological dysrhythmias that do not require further examination, and pathological dysrhythmias that require additional examinations. The additional exam obviously includes an ECG at rest and exercise but often also requires cardiac ultrasound and blood examination (electrolytes, troponins, CBC and chem panel). The exercise ECG test is essential in horses with poor performance and horses with aortic regurgitation, which show an increased prevalence of ventricular arrhythmias. When determining the clinical importance of arrhythmias, one should attempt to identify the possible effect on performance but also the potential risk to horse and rider. Naturally, all decisions and interpretations must always be made in the light of type of sport, level of sport, owner expectations, insurance, future re-sale, etc.

In normal horses, sinus arrhythmia appears as a waxing and waning of heart rate (‘accordion’ effect) during recovery from exercise. Although the arrhythmia is easily identifiable on an ECG recording, it might be more difficult on auscultation to differentiate it from atrial or ventricular ectopy or from (paroxysmal) atrial fibrillation.

The two most important physiological and pathological arrhythmias to differentiate are second degree atrioventricular block (2°AVB) and atrial fibrillation (AF).

2°Atrioventricular Block is the most common physiological dysrhythmia in horses appearing at rest when vagal tone is high. It is completely innocent as long as it disappears (often temporarily) with slight excitation of the horse or with exercise. In 2°AVB the underlying rhythm is regular but at regular intervals one beat (or sometimes 2) is blocked producing a pause that is exactly double (triple) the normal RR interval. Careful auscultation will often reveal an atrial sound (fourth heart sound) during the pause. 2°AVB should be clearly differentiated from the most important pathological dysrhythmia in horses, atrial fibrillation (AF). Although AF can occasionally present as paroxysmal AF, e.g. during and immediately after racing, in general, it will be permanent AF which means it sustains for ever, unless treated. On auscultation AF typically appears as an irregularly irregular rhythm with a loud first heart sound, but be careful: in some horses the rhythm may mimic 2°AVB (although the atrial sound is absent during a pause). Therefore one needs to listen long enough because during AF, besides long pauses, a too early beat will always be found. The combination of loud first heart sounds, pauses without fourth heart sound, AND too early beats is by far most likely to be atrial fibrillation. Differential diagnosis includes an ectopic atrial and/or ventricular rhythm. The only way to get certainty is to obtain an ECG. When auscultating an AF horse after exercise, the irregularity of the rhythm becomes slightly more difficult to appreciate because of the high heart rate. Horses with AF (in the absence of other cardiac disease) usually will return to full athletic capacity after successful treatment. It is important to realize that, even when AF is an incidental finding in a normally performing horse (often at lower levels), the arrhythmia often results in exercise-related disproportionate tachycardia, abnormal QRS complexes and R-on-T, leading to an increased risk for collapse or even sudden death during exercise. Horses showing any of these ECG abnormalities should be converted. Recurrence risk after successful cardioversion is about 30-35%.

Atrial premature beats are usually found at rest or slow exercise and tend to disappear (or become invisible on the ECG) during exercise. They rarely seem to have an effect on performance. However, atrial premature beats are a trigger for induction of atrial
fibrillation. Horses with a high burden of atrial premature beats should regularly be checked by ECG to show the absence of atrial fibrillation.

**Ventricular premature beats** can be found at rest and/or during exercise and suggest myocardial or other underlying disease. Especially when ventricular premature beats increase with exercise, they are considered a risk for deterioration into ventricular tachyarrhythmia’s which can result in collapse or sudden death. Exercise ECG recording is mandatory to rule out exercise-related ventricular arrhythmias in poor performing horses. In case of maximal exercise (racing), an occasional, isolated ventricular ectopic beat in the immediate post-exercise period is regarded as normal. Also in horses with aortic regurgitation (degree 3/6 or more) an appropriate exercise ECG is mandatory as these horses are known to show an increased prevalence of ventricular ectopy during exercise. When no underlying cause is found horses should be rested, might be treated with anti-inflammatory drugs, and should be followed-up carefully before they are allowed to continue work.

**Bradydysrhythmias**, such as 2°AVB, sinus arrest, sinus block or sinus bradycardia are usually vagally driven and should disappear immediately with exercise or excitation. If not, one should consider advanced 2°AVB, or rarely, 3°AVB, and further cardiac exams are required.

Dysrhythmias are common in horses. Some can be readily identified and differentiated on auscultation but the ECG is needed to prove the exact origin. As ventricular arrhythmias often only appear during intense exercise, an exercise ECG test is an essential part in the evaluation of a poor performer or a horse with aortic regurgitation. Besides the effect of an arrhythmia on performance, the clinician should inform the owner of possible risks for horse and rider associated with exercise. A full cardiac work-up, including blood examination and cardiac ultrasound, might be necessary to get a full picture of the disease.
IMPLICATIONS OF CARDIAC DISEASE FOR SEDATION AND ANAESTHESIA

The function of the cardiovascular system is to deliver adequate volumes of oxygenated blood to peripheral tissue. This function is determined by the heart, blood and blood vessels. Especially the interaction between heart and blood vessels is complex and has a major influence on final delivery of blood. In a healthy horse, cardiac function mainly depends on blood circulating volume, heart rate and on cardiac contractile function. Vascular tone has a major impact on the blood pressure. Sufficiently high pressures (generated by the heart) are needed to allow the blood to circulate to the periphery. However, higher blood pressures (due to vasoconstriction) will impede the ejection of blood by the left ventricle and therefore reduce cardiac output. So both hypertension and hypotension may be associated with impaired circulation of blood. Monitoring of cardiovascular function during sedation and anaesthesia in horses is often limited to blood gases, ECG (or at least heart rate) and invasive blood pressure. Often cardiac output is not monitored. Heart rhythm should be a sinus rhythm and heart rate should be within normal ranges. Blood pressure, however, is dependent on cardiac function (e.g. preload, contractile function) and on vasomotor tone (afterload due to vasoconstriction or vasodilation). Blood pressure does not accurately reflect cardiac output and the delivery of blood to peripheral tissue. Vasoconstriction has a positive influence on blood pressure (blood pressure increases) but negatively influences cardiac output (reduced output due to increased afterload). Therefore, despite a normal blood pressure cardiac output might be too low. Inducing vasodilation in such a patient will increase cardiac output but on the other hand, the decreased blood pressure may hamper peripheral circulation. Because of the large individual patient differences, the limited anaesthetic monitoring and the complex interactions within the cardiovascular system, the exact effect of the applied anaesthetic protocol or drug is often speculative and difficult to predict in the equine patient. Presence of heart disease further complicates these interactions. For this reason, there is hardly any evidence of the implications of heart disease on sedation or anaesthesia in horses. The suggestions below are based upon general principles that should be taken into account but currently lack hard evidence in the equine patient.

Cardiovascular depression is the most important side effect of anaesthetics, increasing the likelihood of cardiovascular failure and death. Cardiac disease might increase this risk. Firstly, it is of imperative importance that an accurate diagnosis of cardiac disease is made and, if possible, that reversible risk factors are treated preoperatively. Despite the fact that anaesthesia in most horses with mild to moderate cardiac disease is uneventful, owners and insurance should be well informed before starting sedation or anaesthesia. Horses generally compensate well for most cardiovascular disease at rest so that no clinical signs are seen. If clinical signs related to the heart are present at rest, cardiac failure is usually present and prognosis for life and for anaesthesia is probably grave and the anaesthetic procedure should be questioned.

In general, intravenous anaesthetic techniques are said to be less cardio-respiratory depressant than volatile agents. When using inhalation anaesthesia, isoflurane and sevoflurane are less myocardially depressant than halothane. During inhalational anaesthesia, continuous low dose alpha2-agonists may reduce the use of volatile agents which is beneficial. However, these agents also produce bradycardia, possible hypotension (after initial hypertension) and the less alpha2agonist agents (xylazine) may increase myocardial sensitivity to catecholamine-induced ventricular arrhythmias. Lidocaine infusion also reduces the use of volatile agents, does not induce bradycardia and has anti-arrhythmic properties. Ketamine infusion also reduces the use of volatile agents and improves cardiovascular function by its sympathomimetic properties. Acepromazine causes hypotension due to vasodilation. However, at low dose administered intramuscularly, it is often used as a premedication before anaesthesia because of its sedative and anti-arrhythmic effects (less circulating catecholamines). Premedication with opioids might also be associated with vasodilation and lower blood pressures during anaesthesia. Dobutamine is a positive inotropic and positive chronotropic agent used during anaesthesia to increase blood pressure and cardiac output. However, at high doses, it also increases the occurrence of atrial and ventricular ectopic rhythms. Finally, besides the administration of drugs, one should be aware that restoration of blood volume (crystalloids, plasma expanders,…) is an important tool to improve blood pressure and cardiac output. Spontaneous breathing results in a better cardiovascular function than controlled respiration (IPPV). Body position also has an
important effect on cardiovascular function, dorsal recumbency having a profound negative effect on venous return and hence pre-load.

Some specific conditions require special attention. Vagally-induced bradycardia is usually treated with atropine. Foals with bladder rupture present with hyperkalaemia and are at increased risk for profound bradycardia (atrioventricular block), asystole and ventricular fibrillation. Right ventricular pacing, using a temporary pacing device, and electrical defibrillation, using external paddles, have been successfully applied in foals with bradycardia, asystole and ventricular fibrillation (unpublished results). Sinus tachycardia can be treated with alpha2-agonist drugs. Ventricular tachycardia should be treated with lidocaine; also magnesium sulphate can be used. As horses with atrial fibrillation might have slightly lower blood pressures, acepromazine premedication is usually avoided. In these horses, intra-operative dobutamine use should be limited (lower dose) as this agent is more likely to result in enhanced atrioventricular conduction and therefore excessive increases in heart rate.

Mitral regurgitation results in left atrial volume overload, which might impair lung circulation and might be associated with decreased systemic blood pressure. Sufficiently high heart rates are thus important to maintain cardiac output and blood pressure. In these horses, it is important to avoid bradycardia and hypertension (the latter will increase the regurgitant volume). Such horses benefit from premedication with acepromazine. In severe cases, pre-treatment with ACE-inhibitors (eg benazepril), and potentially digoxin, might be useful but remains unproven. Horses with aortic regurgitation will have lower diastolic and mean blood pressures with reduced cardiac output. Lower diastolic pressures might also lead to myocardial hypoxia and increased risk for ventricular ectopy. Similar measures as for horses with mitral regurgitation should be taken for general anaesthesia. Regarding sedation in horses with severe aortic regurgitation, a low dose should be used as these horses might be more likely to collapse due to severe hypotension as a result of slow heart rate (long diastole further decreases diastolic pressure). Horses with a ventricular septal defect will present left-to-right shunting with pulmonary volume overload. Again bradycardia and especially arterial hypertension should be avoided. The latter would decrease cardiac output, arterial pressure and increase pulmonary overload.
ELECTRICAL CONVERSION OF ATRIAL FIBRILLATION: SHOCKING?

Atrial fibrillation (AF) is the most important arrhythmia in horses. Although horses performing at lower exercise levels might not present clinical signs, one must appreciate that some individuals with AF have excessively high ventricular rates, increased prevalence of ventricular arrhythmias and even R-on-T phenomenon during low level exercise. As a consequence, such horses can be at an increased risk of collapse or sudden death during exercise compared to their age-matched peers. Atrial fibrillation can be converted to sinus rhythm by administration of drugs, such as quinidine sulphate or amiodarone, or by electrical cardioversion. Treatment with quinidine sulphate has a success rate of about 60-80%; success tends to be highest in race horses (younger and more acute AF) and lower in sport horses (older and more chronic AF), but this treatment is often associated with side effects of which some may carry a risk (colic, diarrhoea, laminitis, tachycardia and potentially dangerous ventricular arrhythmias). Transvenous electrical cardioversion has a higher success rate (90-98%), is often effective even when quinidine sulphate treatment failed, and is associated with fewer side effects. Because of the size of the horse's chest, the direct current electrical shock must be applied through internal electrodes (catheters in the heart). The procedure requires general anaesthesia, specialized equipment and experienced operators which are familiar with all aspects of equine cardiology and with cardiac catheterization.

PROCEDURE

In the standing horse, two cardioversion catheters are inserted via the jugular vein. These catheters possess a large surface area electrode at the tip for shock delivery. Under ultrasound guidance, 1 catheter needs to be positioned in the right atrium while the second catheter is guided 10–20 cm into the left pulmonary artery. A pressure trace recording from the catheter may be used to give additional information about the position of the catheter. A third catheter, a pacing catheter, is inserted into the right ventricular apex and can be used for temporary right ventricular pacing for the rare occasion where shock delivery would result in temporary (usually seconds up to two minutes) asystole. When all catheters are in place, general anaesthesia is induced. During anaesthesia, catheter position is confirmed by ultrasound and the catheters may be repositioned, as necessary. Radiography has also been used to check catheter positions. The right ventricular pacing catheter is first connected to a temporary pacing unit. Both cardioversion catheters and a surface ECG are then connected to the biphasic defibrillator, which is operated in synchronous mode. The defibrillator detects the R wave of the ECG and will automatically deliver the shock timed exactly on the R wave after the button of the defibrillator is pressed. Synchronous shock delivery with correct detection of the R wave is vital to avoid induction of fatal ventricular fibrillation. When the defibrillator wrongly identifies the T wave as an R wave, position of the surface electrodes must be changed. Incremental shocks are applied starting at 125 to 300 Joules, with attempt being made to minimize total energy delivered. Most horses convert between 125 and 250 J with an impedance of 25 to 50 Ohm. If cardioversion is not achieved, shock delivery after administration of an antiarrhythmic drug, such as amiodarone, usually results in successful cardioversion. After successful cardioversion, anaesthesia is continued for at least 10 minutes to monitor for atrial ectopy. If there is a high burden of atrial premature beats immediately after cardioversion, antiarrhythmic drugs can reduce the risk for immediate recurrence of AF. After the cardioversion procedure, catheters are gently withdrawn and the horse is allowed to recover.

AFTERCARE

Following successful conversion to normal sinus rhythm aftercare is similar irrespective of the method used to achieve it.

A high burden of atrial premature depolarisations and especially the presence of runs of atrial tachycardia on a 24-hour ECG monitoring one week after cardioversion have been associated with an increased risk for AF recurrence (unpublished data). In these circumstances, treatment with anti-inflammatory, antiarrhythmic drugs, or both coupled with prolonged rest (2-4 months) have been suggested in an attempt to reduce the risk of the AF recurring.

AF results in progressive electrical and contractile remodeling of the atrial myocardium. Reverse remodeling after successful conversion depends on the chronicity of the
Arrhythmia. Experimental studies showed that after one week of AF reverse remodeling was complete within 1–2 days, while AF of 4–6 months duration took longer (about 3–4 weeks) to fully return to normal baseline values. A resting period that allows full recovery is optimal but not always possible due to financial and time constraints. Owners, trainers and treating veterinarians are advised to monitor cardiac rhythm regularly by palpation of the apex beat or auscultation, especially before and after competitive work and if the horse ever performs below expectation.

PROGNOSIS
Transvenous electrical cardioversion has a high success rate of 90–98% and is known to be more effective in chronic cases compared to quinidine. Therefore, transvenous electrical cardioversion can still be efficacious in horses that fail to convert with quinidine. When treatment of AF is successful, horses can return to their previous exercise levels. Recurrence rate of AF at one year post cardioversion is around 30–35% (unpublished data). If AF recurs it most-frequently happens in the first 4 months post conversion.