

Equine cardiology: when to worry about murmurs and arrhythmias

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Introduction

‘Abnormal’ findings are common during cardiovascular examination of horses, and determining the significance of these in terms of performance or clinical status can be challenging. The emphasis in equine cardiology is mostly on diagnosis and prognosis, rather than the treatment of cardiac disease. This lecture aims to help you interpret your clinical examination in order to provide likely differentials, and to understand the significance of those differentials and when further investigation is warranted.

History & Signalment

The most common reason for presentation of a horse for a cardiovascular workup is poor performance/recovery. ***Despite this, lameness and respiratory disease/dysfunction are far more common causes of poor performance than cardiovascular disorders.*** Cardiovascular abnormalities are often detected on routine clinical or pre-purchase examination, leading to a full cardiovascular workup. ***A massive functional cardiac reserve means that horses rarely present with signs of obvious cardiac failure.***

Consider:

- Age – congenital vs. acquired disease
- Breed
- Usage
- Fitness/training history
- Onset of signs
- Concurrent/previous history of disease
- Poor performance/recovery
- History of collapse/“seizures” (syncope an important DDX)
- colic – acute cardiac dysfunction (V.tach, aortic root rupture) may present as acute colic.

Clinical Examination

Remember to include a thorough examination of all body systems. In a horse presented for poor performance, lameness and respiratory evaluations are always warranted.

1) Distant Examination

Body condition should be assessed. Weight loss is a feature of decompensated (end stage) cardiac disease.

The horse should be examined for the presence of oedema. Cardiac disease is not a common cause of oedema in horses. Oedema can occur in the brisket region/under the cranial abdomen and in the prepuce of horses with cardiac failure. Distal limb oedema can occur with cardiac disease, although other causes (hypoproteinaemia, vasculitis etc) are far more common.

2) Peripheral Pulse

The pulse can be palpated at several different sites. Palpation of the facial artery (under the jaw) is the usual site for assessment, though the transverse facial artery (located just ventral and caudal to the lateral canthus of the eye) is also a useful site.

The pulse rate and quality can be assessed. Pulse strength represents the difference between systolic and diastolic pressures. Certain conditions may result in a pulse deficit (e.g. atrial fibrillation, VPCs) whilst others may cause changes in pulse strength e.g. bounding pulses in aortic regurgitation.

3) Mucous membranes

Oral mucous membrane colour and capillary refill time should be assessed, although they are not sensitive or specific indicators of cardiac disease. Low output cardiac failure may result in pale mucous membranes and a prolonged capillary refill time. Cyanosis may be apparent with right to left shunting such that occurs in some congenital disorders.

4) Jugular veins

The jugular veins are an indicator of venous pressure. In the normal horse, only the lower ~1/3 of the veins should be distended. Normal 'pulsations' or waves can be observed in this region. Right heart failure can result in jugular vein distension further up the neck. ***Be careful to assess this with the head held in a normal position – lowering***

the head will result in distention of the jugular veins in normal horses. Tricuspid regurgitation and atrioventricular dissociation (ventricular tachycardia, 3rd degree AV block) can lead to abnormal ‘pulsations’ or ‘waves’ up the jugular veins. These causes need to be distinguished from pulses that are referred from the carotid arteries.

5) Auscultation

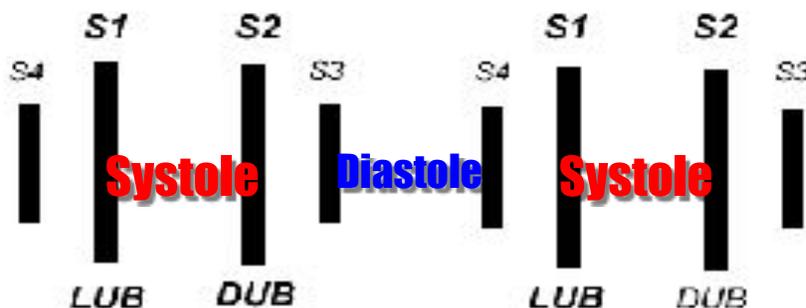
Careful cardiac auscultation can be a precise diagnostic tool. It is important to find a quiet place to listen to the horse. *Always listen to the left and right sides, and always include the lung fields in your examination.* A good quality stethoscope is essential. The diaphragm is best for picking up high frequency sounds, the bell for lower frequency sounds. The newer Littmann™ stethoscopes are equipped with a tunable diaphragm – pressing softly accentuates low frequency sounds, pressing harder accentuates higher frequency sounds.

Auscultate the heart before and after exercise or stimulation – changes in heart rate can help distinguish some functional murmurs & arrhythmias – see later.

Normal resting heart rate varies from 25-45 beats per minute in adults and 60-80 bpm in foals. Resting heart rate depends on fitness and excitement. During maximal exercise heart rates approach 240 bpm. *The resting heart rate is an important indicator of cardiac function* – a horse with a heart rate of 30 bpm is unlikely to be in decompensated congestive heart failure.

i) The cardiac cycle

The two readily audible heart sounds S1 (Lub) and S2 (Dub) can be heard in every horse. S1 (Lub) is associated with closure of the atrioventricular valves (the left = mitral, the right = tricuspid) and marks the beginning of systole (ventricles contracting). S2 is associated with closure of the semilunar valves (aortic and pulmonary), as blood slows in the aorta and pulmonary artery, and marks the end of systole.



In addition to the two main heart sounds, two other normal sounds, S3 and S4, can be heard in some horses (usually fit horses). S4 is associated with atrial contraction, just prior to ventricular contraction, and may sound like a ‘split’ first heart sound. You should be able to hear S4 in most horses – and it is important to detect the presence of this sound – see later. S3 is associated with passive ventricular filling. It is low pitched and can be difficult to hear. It is most obvious in fit athletic animals.

Sounds associated with each of the valves can be heard in different regions on the chest wall. These regions do not necessarily correlate with the position of the underlying valve.

ii) Technique

a) Begin by palpating the left side of the chest for an ‘apex beat’. Place your stethoscope on the chest wall at this region – usually 5th intercostal space, ~10cm below the point of the shoulder. In this area you will hear a loud S1. This area is the best place to listen for S3, and sounds associated with the *mitral valve*.



b) Move your stethoscope cranially and a little dorsally, just under the triceps muscle, to ~ the 4th intercostal space. You will hear S2 loudly in this area. This is the best place to listen for sounds associated with the *aortic valve*.



c) Move your stethoscope further cranially and slightly ventrally, to around the 3rd intercostal space. It may help to move the limb forward for better access. S2 will be loud in this region. This is the best place to listen to sounds associated with the *pulmonary valve*. This is the best place to listen for S4.



d) **Right hemithorax.** You may be able to palpate an apex beat on the RHS. Start listening at this area, or around the 4th-5th intercostal space. Move cranially under the triceps muscle to around the 4th intercostal space, midway between the point of the shoulder and olecranon. It may help to move the limb forward for better access. This is the best place to listen for sounds associated with the **tricuspid valve**, and S1 will be loudest in this area.



Establish the heart rate and rhythm prior to examining each area in detail. The normal transient heart sounds (S1-S4) vary in intensity depending on fitness, body condition, and individual variation. Pericardial or pleural fluid may result in muffled sounds. Consolidated lung between the heart and thoracic wall may result in louder sounds that are heard over a wider area than normal. Intrathoracic masses may change the position of the heart within the thoracic cavity and change the loudness or positioning of heart sounds at the chest wall.

iii) Murmurs

Murmurs are prolonged sounds heard during a stage of the cardiac cycle which is normally silent. They are caused by turbulent blood flow which results in vibration of cardiac structures.

Murmurs can be classified using a number of criteria in order to attempt to define their source and significance:

a) Timing & duration:

- Early, mid or late systolic/diastolic
- Pansystolic – from the start of S1 to the end of S2
- Holosystolic – from the end of S1 to the start of S2
- Holodiastolic – from the end of S2 to the start of S1
- Presystolic – between S4 and S1
- Machinery – continuous throughout the cardiac cycle

b) Location & Radiation:

The point of maximal intensity (PMI) of a murmur is the position on the chest wall where the murmur can be heard best. The position is correlated with a valvular region (see above) or can be expressed as being toward the base or apex region of the underlying heart.

Whether or not a murmur ‘radiates’ to other regions of the chest wall other than the PMI can give an indication of the severity of the underlying pathology.

Pathological murmurs often radiate in the direction of turbulent flow (e.g. the direction of a regurgitant 'jet')

c) Intensity (loudness):

Grade 1 – quiet & localised

Grade 2 – quiet, localised, heard immediately

Grade 3 – Moderately loud

Grade 4 – Loud & widespread area, faint thrill

Grade 5 – Loud with a precordial thrill

Grade 6 – Can hear with the stethoscope off the chest

A vibration of the chest wall that can be palpated is called a precordial thrill. Precordial thrills are associated with high velocity turbulence & significant disturbance of flow.

d) Quality:

Murmurs may get louder (crescendo) or softer (decrescendo) from start to end, or may have a constant intensity from beginning to end (plateau). They can also be described as harsh, soft or musical.

Functional murmurs

High velocity blood flow and large vessel diameter increase the potential for turbulent flow. The properties of the equine heart (large chambers & vessels, large stroke volume) mean that turbulent flow can readily occur even when pathology is not present. ***Non-pathological murmurs associated with normal blood flow are relatively common especially in fit, young horses. These murmurs are called 'functional', 'flow', or 'innocent' murmurs.***

- ***Early-mid systolic functional murmurs:*** These murmurs are most common in fit athletic horses, but can be heard in ~40% of all horses. They are usually heard best over the base of the heart on the left side of the chest (pulmonary and aortic areas) and are localized (do not radiate). They are often high pitched, and end well before S2. They usually vary with heart rate – they may disappear or get louder at higher heart rates. ***These murmurs are not associated with any pathology, and are thought to be the result of high velocity flow within the great vessels during ventricular 'ejection'.***
- ***Early diastolic functional murmurs:*** most common in young fit horses. Often called the 'two year-old squeak'. These murmurs are very short in

duration and high pitched, can often be heard over both sides of the chest, and occur between S2 and S3. ***They are not associated with any underlying pathology.***

Additional Diagnostic Procedures

1) Laboratory medicine:

A complete blood profile is useful in all cases of suspected cardiac disease to determine the presence of inflammation, infection and/or systemic disease.

- **Haematology:** Anaemia may result in signs similar to that of cardiac disease, or may exacerbate existing cardiac failure. Changes in white cell count and differential may support a diagnosis of bacterial endocarditis or pericarditis (i.e. neutrophilia with a left shift). A lymphocytosis +/- monocytosis may support a diagnosis of viral pericarditis or myocarditis.

- **Blood biochemistry:** Electrolyte disturbances may contribute to cardiac arrhythmias, and therefore K, Mg, and Ca levels should be determined. High plasma fibrinogen indicates an inflammatory process is present, though this is not specific to the heart. Cardiac muscle isoenzymes of creatinine kinase (CK) and lactate dehydrogenase (LDH) can be assayed from plasma. The levels of these enzymes may be increased when active myocardial damage is present, although specificity and sensitivity of these tests is questionable. **Plasma cardiac troponin I (cTnI) is a sensitive and (fairly) specific indicator of myocardial damage and/or inflammation in horses.**

2) Electrocardiography (ECG)

The ventricular depolarization process in the horse is different from that of small animals. There is a widespread Purkinje network within the ventricles of the horse resulting in multiple depolarisation points across the myocardium. ***As a result the ECG gives no information on chamber size/enlargement in the horse. The ECG provides useful information on heart rate and rhythm.***

- Lead systems

As ECGs give no information on chamber size/ enlargement in the horse, only a single (base-apex lead system) is usually needed to adequately diagnose the rhythm disturbance.

In the base apex system the negative electrode (RA) is attached over the base of the heart (Right jugular groove or in front of the right scapula) and the positive electrode (LA) is attached over the apex of the heart (left chest wall behind the elbow). Alternatively, in the Y lead system the positive electrode is attached over the xiphoid and

the negative over the manubrium. A neutral electrode is required in mains powered machines, attached at any convenient site. **For the base-apex or Y-lead systems the machine is set to lead I (LA +, RA -).**



Above: Normal ECG Trace using a base apex lead system

ECGs can be recorded at rest, after exercise, continuously (24 h Holter monitoring) or during exercise via telemetry

Examining the trace:

- Establish the heart rate
- Is the rhythm regular?
- Is there a P wave for every QRS?
- Is there a QRS for every P wave?
- Are the wave forms normal?
- Are the wave forms consistent? (**Especially QRS**)
- Measure P, QRS, and QT intervals.

ECG: Normal findings: The P wave is notched, and may be biphasic). The P wave may change morphology from beat to beat in normal horses (wandering pacemaker). The QRS complex: Q is the first negative deflection, R the first positive deflection, and S the second negative deflection. The Q wave is often absent in some leads. It is best to consider the QRS as a whole rather than each separate component. The T wave varies greatly in polarity and morphology in normal horses but should be the same or similar in an individual horse.

Normal Measurements:

P wave duration	$\leq 0.16s$
P-R interval	0.26 to 0.52s
QRS complex duration	$\leq 0.14s$
Q-T interval	$\leq 0.6s$



Normal base-apex ECG trace at rest

Heart Score: The heart score is obtained by measuring the average QRS duration in the three limb leads (I,II,III). The heart score has been used as an indicator of ventricular size, and a predictor of performance. It is a dated concept and is of little value

Cardiac Disease

Arrhythmias

1) Atrioventricular block (AV block)

a) 1st degree AV block

1st degree AV block is a variable or consistent increase in the P-R interval. It is often associated with high vagal tone, and is sometimes seen in fit athletic horses at rest. It is rarely if ever pathological, and is abolished at higher heart rates in normal horses. Diagnosis is by measurement of the P-R interval on ECG. Rarely, an increased interval may be heard on auscultation between S4 and S1.

b) 2nd Degree AV Block

2nd Degree AV block is the most common arrhythmia in horses. ***It occurs commonly in normal horses at rest (up to 44% of normal horses), and is a homeostatic mechanism to reduce cardiac output at rest. It is most common in fit athletic horses, and is associated with high vagal tone.***

On auscultation, 2nd degree AV block sounds like a ‘dropped’ or ‘missed’ beat. A low resting heart rate is usually present. An S4 sound may be heard that is not followed by S1,S2,S3. ***In normal horses 2nd degree AV block is abolished by removing the influence of high vagal tone, i.e. by exciting or exercising the horse.***

ECG Findings: A P wave is present that is not followed by a QRS complex. This usually occurs at regular intervals. The most common form of 2nd Degree AV block in horses is Mobitz type I, where there is a progressive lengthening of the P-R interval prior to the dropped beat.



2nd Degree AV block – a P wave is not followed by a QRS complex. Note the progressive lengthening of the P-R interval prior to the dropped beat.

Generally, 2nd degree AV block can be distinguished from pathological arrhythmias by auscultation. ***A regularly irregular rhythm, occurring at low heart rates, that is abolished with exercise, are the auscultatory hallmarks of 2nd degree AV block.*** The presence of an S4 sound that is not followed by S1 S2 confirms the diagnosis. Pathological arrhythmias such as atrial fibrillation sound irregularly irregular, and will not be abolished at higher heart rates. In any case an ECG is diagnostic.

‘High- grade’ 2nd degree AV block, when the block results in more than 2 consecutively dropped P waves, can occur and is considered abnormal. ‘High-grade’ 2nd degree AV block may not be abolished by exercise or stimulation, and may cause loss of performance or even syncope. Advanced 2nd degree AV block can be associated with abnormally high vagal tone (will be abolished by atropine) digitalis toxicity, electrolyte imbalances or AV nodal disease (will not be abolished by atropine). Holter monitoring or treadmill testing with ECG telemetry may assist in diagnosis. Advanced 2nd degree AV block may be characterized by frequent dropped beats (1 blocked for every 1 or 2 normal beats). Recommendations for high-grade second degree AV block are as follows:

- Horses with high grade second degree Av block that disappears with exercise should only be ridden by an informed adult and the heart rate and rhythm should be frequently monitored.
- Horses with high-grade second degree AV block during exercise should be rested then re-evaluated. They are considered less safe to ride than their peers.
- Horses with symptomatic bradyarrhythmias are not safe to ride and generally have a poor prognosis.

c) 3rd degree AV block (complete heart block)

This rhythm disturbance is rare. 3rd degree AV block occurs when there is no transmission through the AV node between the atria and ventricles. A relatively slow junctional or ventricular ‘escape’ rhythm takes over (<20 bpm). Because there is no relationship between the atria and ventricles (AV dissociation) they may contract simultaneously against each other, resulting in a characteristic ‘cannon’ wave up the jugular veins. This rhythm disturbance is always pathological, and carries a grave prognosis. Severe exercise intolerance and syncope are often associated with 3rd degree AV block. A specific cause is often not found although inflammatory and degenerative changes of the AV node, electrolyte imbalances and metabolic abnormalities can cause it. Treatment can be attempted with vagolytic drugs (often unsuccessful), corticosteroids are indicated (dexamethasone 0.05-0.22 mg/kg iv) in the hope that there is reversible inflammatory disease. Pacemaker implantation is often required.

2) Sinus arrhythmia

Sinus arrhythmia is an intermittent, often cyclic, change in heart rate. It is usually associated with changes in vagal tone in horses at rest, or in the period just following exercise. Changes in heart rate may be associated with respiration. It is much less common than 2nd degree AV block in horses that are examined at rest, though it is more commonly seen on 24 hour Holter traces. Sinus arrhythmia is rarely, if ever, pathological.

3) Sinus Block/Arrest

Sinus block is thought to be a manifestation of high vagal tone, though it is much less common than 2nd degree AV block. Sinus block is a pause in SA node depolarization that is up to two R-R intervals long, whilst sinus arrest is defined as a pause greater than 2 R-R intervals long. No S4 is heard during the pause, and no P wave is seen on the ECG. This arrhythmia is usually abolished at higher heart rates, though in some cases it may be associated with poor performance. In severe cases an escape beat or rhythm originating in the junctional or ventricular tissue may be generated. Treatment involves correcting the underlying cause, corticosteroids, sympathomimetic drugs, or even pacemaker implantation in persistent cases.

4) Atrial Premature Complexes (APCs)

Atrial premature complexes are caused by impulses generated in the atrial myocardium from a site other than the SA node (i.e. an ectopic site). They occur prior to the normal SA node impulse (i.e. prematurely). APCs are a relatively common arrhythmia in horses. ***Occasional APCs (<1/hour) are common in clinically normal horses and in the immediate post exercise period. If they are more frequent at rest, associated with runs of paroxysmal supraventricular tachycardia, or the horse presents for poor performance, further investigation is warranted.*** APCs are rarely a cause of poor performance themselves, however, horses with frequent APCs at rest may develop supraventricular tachycardia (4 or more APCs in a row) or atrial fibrillation during maximal exercise. Atrial enlargement, inflammation, electrolyte abnormalities, hypoxia, pyrexia or sepsis/toxaemia may predispose a horse to APCs.

On auscultation APCs sound like a premature normal beat (short diastolic interval), followed by a normal length diastolic period. A pulse deficit may accompany an APC if it is particularly premature (not enough time for ventricular filling).

On ECG the P wave of the APC is usually different in conformation (due to its origin away from the SA node) than other P waves in the trace. The QRS complex of the APC is often normal as it is conducted through the ventricles via the normal pathway (although aberrant conduction is possible). The R-R interval preceding the APC is short, whereas the R-R interval following the APC is normal. This differentiates APCs from premature complexes of ventricular origin, which are always followed by a compensatory pause. APCs may occur without conduction through the AV node (i.e. an early P wave with no QRS following).



The 6th beat is an APC. Note the different conformation of the P wave, the short R-R interval prior to the APC, and the normal R-R interval following the APC.

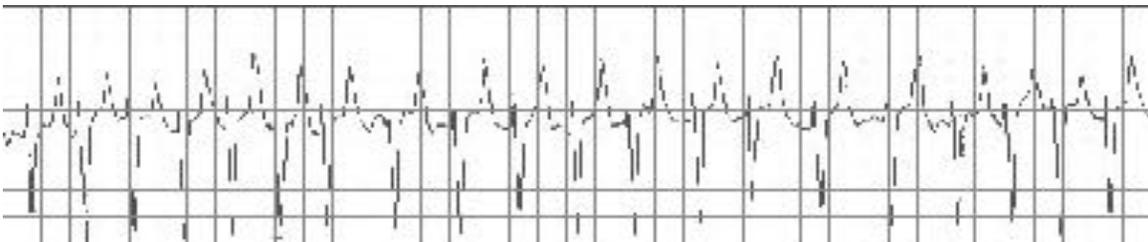
Further investigation of a horse with APCs should include haematology and biochemistry to detect the presence of inflammation, infection and/or electrolyte abnormalities which may be responsible for the rhythm abnormality. An 24 hour ECG is recommended to assess frequency. ECG telemetry during maximal exercise can help establish the frequency of APCs or progression to atrial tachycardia or atrial fibrillation in athletic horses.

In all cases treatment of any possible underlying extra-cardiac cause of APCs is recommended. Isolated or infrequent APCs that are overdriven during exercise and those with only occasional APCs during exercise are considered safe to ride/drive. When there is an impact on performance or large numbers of APCs a rest period is indicated (1-2 months). Corticosteroid therapy (~4 weeks of dexamethasone or prednisolone) may be useful in refractory cases, or when myocardial inflammation is suspected. **Horses with a significantly increased number of APCs may be at increased risk of developing atrial fibrillation.**

5) Supraventricular Tachycardia (SVT)

SVT is a rapid rhythm originating from an ectopic centre(s) within the atria (atrial tachycardia) or AV nodal tissue ('nodal' or 'junctional' tachycardia). It may be paroxysmal (short 'bursts') or sustained. SVT may occur in horses with primary cardiac disease, or may be secondary to systemic disease.

Auscultation will reveal an inappropriately rapid, though often regular rhythm. SVT may be difficult to distinguish from sinus tachycardia on ECG unless a transition is seen on the trace. The P waves are different from those of SA node origin in atrial tachycardia, though often they are 'lost' within the preceding T wave. There may be inconsistent conduction of P waves to the ventricle.



Supraventricular tachycardia. Notice the fairly regular (and short) R-R intervals and normal appearance of the QRS complexes. P waves can be seen in some places on the trace. HR ~ 180bpm

Haematology and serum biochemistry are indicated to detect the presence of underlying systemic disease or metabolic imbalance. Any abnormality should be treated accordingly. An echocardiogram should be performed if primary cardiac disease is suspected. Specific therapy is rarely indicated. In horses with severe signs related to SVT digoxin may be used to control the ventricular response rate.

6) Atrial fibrillation (AF)

AF is the most common arrhythmia affecting performance in horses. It occurs most commonly in athletic horses without underlying cardiac disease, but also occurs in horses with cardiac disease, particularly where atrial enlargement has occurred.

AF is caused by multiple re-entrant wave fronts of electrical activity circling the atria. Large atria and high vagal tone allow the rhythm to perpetuate in horses without underlying cardiac disease. In other species AF generally only occurs in the presence of cardiac disease. Electrolyte imbalances (particularly potassium depletion) may predispose.

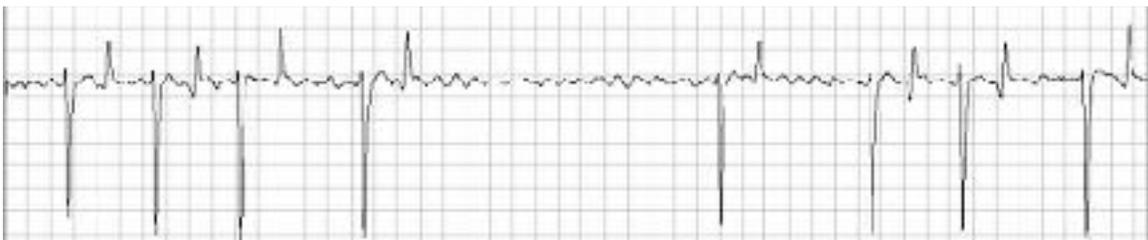
AF results in a loss of coordinated atrial contraction, and therefore loss of the atrial contribution to ventricular filling. At rest the atria contribute little to ventricular filling, and therefore ***most horses with AF are asymptomatic at rest.*** However, atrial contraction can contribute up to 25% of ventricular filling at maximal exercise. ***AF therefore markedly reduces peak performance in athletic horses.***

Horses with AF and underlying cardiac disease may have a history of progressive exercise intolerance. The most common presentation however is that of sudden loss of performance in a standardbred or thoroughbred racehorse. Horses that develop AF during a race suddenly slow down and may become ataxic. Exercise induced pulmonary haemorrhage may occur.

Atrial fibrillation can be paroxysmal (reverts spontaneously within 24-48h) or sustained. Paroxysmal AF can be difficult to diagnose, as spontaneous reversion to sinus rhythm often occurs before an examination can be carried out.

Auscultation reveals an irregularly irregular (random) rhythm. S1 and S2 may vary in intensity. ***S4 is not heard.*** In horses with underlying cardiac disease murmurs may be noted. Variable intensity peripheral pulses and pulse deficits may be palpable.

The ECG lacks P waves. Instead there is a constantly undulating baseline (so-called 'f' waves). The QRS complexes are normal, but the R-R interval is highly irregular, particularly at lower rates. The ventricular response rate varies, but is usually similar to normal sinus rate in horses without underlying cardiac disease.

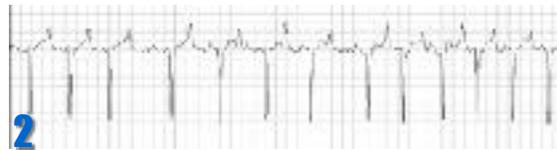


Atrial fibrillation. Note the absence of P waves, the irregularly irregular R-R interval, normal QRS complexes and undulation of the baseline ('f' waves).

Haematology and serum biochemistry should be performed on all horses with AF to detect the presence of systemic disease and/or metabolic or electrolyte abnormalities. An echocardiogram is indicated to rule out the presence of primary cardiac disease, in particular atrial enlargement.

Horses with AF can be converted to sinus rhythm using antiarrhythmic drug therapy or transvenous electrical cardioversion. However, **horses with significant cardiac disease (cardiac failure) are not suitable candidates for conversion** – it may be dangerous to attempt to convert these horses and they tend to revert back to AF after therapy. Horses without underlying cardiac disease (almost all athletic horses presented with acute-onset AF) may convert to sinus rhythm spontaneously. If this does not occur within 24-48 h therapeutic intervention is indicated. The level of intended activity influences whether cardioversion is required. Cardioversion is generally recommended for horses performing high intensity athletic activities, or if the average maximal heart rate during exercise (at a level mirroring the horses intended use) is greater than 220/minute. Collapse during exercise has been reported with AF and ventricular ectopy can occur so an exercising ECG should always be performed in horses where cardioversion is not intended.

The antiarrhythmic drug quinidine is most commonly used for conversion to sinus rhythm. Quinidine sulfate is administered at 22mg/kg via nasogastric tube every 2 hours until a maximum dose of 40-60g (4-6 treatments for a 500kg horse) is reached, toxic or adverse reactions occur, or conversion is achieved. Most horses are successfully converted to sinus rhythm within 6 treatments. If not, dosing can be repeated the next day. Alternatively, dosing may be continued with the frequency reduced to every 6 hours until conversion is affected. Digoxin may be added to the regimen in refractory cases or in horses that are tachycardic/have previously become severely tachycardic during a conversion attempt. A lower than usual dose of digoxin (0.011 mg/kg PO *s.i.d*) is used as quinidine displaces digoxin from albumin, increasing its concentration. Intravenous quinidine gluconate (1.0 mg/kg q 5-10 min, up to maximum 10 mg/kg) is used successfully where available (USA) when the duration of AF is < 1 week.



ECG recordings from a horse undergoing conversion of AF using quinidine sulfate. 1) After 1 dose (10g). 2) After 3 doses (30g). 3) After 4 doses. 4) After 5 doses (50g) conversion is successful – sinus tachycardia now present.

Side effects of quinidine therapy commonly encountered include:

- Depression
- sweating,
- paraphimosis
- flatulence
- colic,
- diarrhea
- tachycardia

Reduced myocardial contractility and congestive heart failure can occur in horses with underlying myocardial dysfunction. Quinidine treatment should be avoided in horses with CHF.

- **Toxic effects include nasal mucosal oedema, neurological signs and arrhythmias.**
- cardiac arrhythmias (SVT, VPCs, VT, VF) may occur along with widening of the QRS as plasma levels approach the toxic (>5 ug/ml) range (Therapeutic range 2-5 ug/ml).

At each treatment interval an ECG should be performed to monitor progress and detect signs of toxicity. Widening of the QRS complexes by 25% or more is considered an early indicator of toxicity. If toxicity occurs treatment should be ceased immediately. Sodium bicarbonate (1mEq/kg IV) can be used to treat quinidine toxicity: it increases quinidine binding to albumin and therefore reduces active (free) drug levels. Digoxin can be used to treat supraventricular tachycardia, and MGSO₄ can be used for ventricular tachycardia.

Following conversion an echocardiogram and 24-h ECG are recommended to establish the presence of residual atrial arrhythmias and myocardial dysfunction. A rest period is ideal prior to returning to work. Most horses return to athletic activity without a reduction in performance. Prognosis is good in horses with minimal underlying heart disease 90% of these horses are converted to normal sinus (quinidine or electrical cardioversion). Atrial fibrillation may recur in up to 25 % (duration of AF < 4 mo.) and 65 % (duration of AF > 1 year) of horses at a later date but, if treated and converted, horses once again return to previous performance level. The longer the arrhythmia has been present the harder it is to convert, the higher the risk of side effects, and the more likely AF is to recur. Occasionally, a horse will convert to normal sinus rhythm without treatment. If this happens, it usually does so within the first 24–48 hours (paroxysmal atrial fibrillation).

Horses that do not convert to sinus rhythm can function normally as breeding animals, and can usually tolerate mild athletic activity (dressage, pony club etc. exercising ECG should be performed to check that an appropriate heart rate is achieved for level of exercise and that there is no ventricular ectopy). As long as there is no concurrent cardiac failure, horses with persistent AF are generally safe to ride.

7) Ventricular premature complexes (VPCs)

VPCs are the result of abnormal impulse formation from an ectopic focus within the ventricular myocardium. ***A single VPC detected at rest warrants further investigation as primary cardiac disease may be present.*** Systemic disease or metabolic/electrolyte imbalances may predispose horses to the development of VPCs.

Horses with isolated, infrequent VPCs may be asymptomatic. VPCs may become more frequent during exercise, or progress to ventricular tachycardia, resulting in exercise intolerance and possibly syncope.

Auscultatory findings for VPCs are similar to that of APCs. There may be variability in the intensity of S1 and S2. A pulse deficit may be palpated.

On ECG the QRS of the VPC appears wide (often within normal limits however) and different to that of the normally conducted QRS complexes. The VPC is not preceded by a P wave. The T wave of the VPC is large, prolonged and often of a different polarity to normal T waves. VPCs are followed by a compensatory pause, this occurs because the next sinus impulse (P wave) is blocked by the VPC. This P wave is often superimposed upon the end of the T wave. If all the abnormal QRS and T complexes have the same morphology in 1 lead the VPCs are described as being uniform. If several different abnormal QRS complexes are present in 1 lead the VPCs are described as multiform.



An isolated VPC. Note the wide and bizarre QRS complex, with no preceding P wave. The T wave of the VPC is large and prolonged. The VPC is followed by a full compensatory pause, with the following P wave superimposed on the end of the T wave (arrow). Note the regular P-P interval, hence the compensatory pause

Further investigation of a horse with VPCs should include haematology and serum biochemistry. Non cardiac causes of VPCs should be ruled out or corrected if possible. A complete cardiovascular examination is indicated, including electrocardiography (24 hour holter, exercise test) an echocardiogram and cardiac isoenzymes (cTnI).

Occasional uniform VPCs (around 1 per hour) occur in 14% of normal horses. Occasional VPCs may be clinically insignificant and do not require treatment. Frequent uniform VPCs and multiform VPCs are suggestive of myocardial pathology. In these cases a rest period (4-8 weeks) followed by reevaluation is recommended. Corticosteroid

therapy may be of use if an inflammatory myocarditis is suspected. If R on T phenomenon is present, antiarrhythmic drug therapy is indicated as this may predispose the horse to ventricular tachycardia or ventricular fibrillation. Recommendations for horses with VPCs include:

- Horses with occasional VPCs at rest or during exercise can be ridden by an informed adult.
- Horses with complex ventricular ectopy should be rested and treated. The safety of these horses is uncertain and they should only be ridden by an informed adult
- Rigorous work is not recommended for horses that have VPCs and moderate or severe structural heart disease. These horses should only be ridden by an informed adult rider

8) Ventricular tachycardia

Ventricular tachycardia (VT) is defined as four or more VPCs in a row, and may be paroxysmal (short ‘bursts’) or sustained. VT can occur in horses with severe systemic disease, electrolyte imbalances or cardiac disease. If all other causes of VT are ruled out primary myocardial disease should be suspected.

Clinical examination will often reveal signs of severe systemic disease (colic, toxemia etc.). Clinical signs of congestive heart failure may develop due to the high heart rate. Most horses with sustained VT and heart rates around 120 bpm will develop generalized venous distension, jugular pulses and ventral edema. Syncope may occur in horses with uniform VT with heart rates >150 bpm. Pulmonary edema may also develop. On auscultation the rhythm will be rapid (>60bpm) and regular if the VT is sustained and uniform. It will be irregular with paroxysmal and/or multiform VT. Heart sounds are loud and of variable intensity. A weak pulse, with deficits is palpable.

On ECG there is AV dissociation (no relationship between P waves and QRS complexes). The P-P interval is regular and at a normal atrial rate but P waves are often superimposed upon the QRS complexes. The R-R interval is often regular unless multiform VT is present (irregular) or the VT is paroxysmal (periodically irregular). Sustained VT may be difficult to differentiate from rapid supraventricular tachycardias (and rapid atrial fibrillation). R on T phenomenon, where the QRS begins on the T wave of the previous beat, may be seen. This is considered an extremely unstable rhythm, with progression to ventricular fibrillation possible.



Sustained uniform VT. Note the regular R-R interval. The QRS complexes are high amplitude and wide. Some P waves can be seen, but they have no relationship to the QRS complexes

If underlying disease is present treatment should be directed at reversing the underlying disease whilst maintaining cardiac output. Antiarrhythmic therapy for ventricular tachycardia is indicated if the rate is excessively high, the rhythm is multiform, or R on T complexes are detected. If pulmonary edema is present antiarrhythmic treatment should be initiated as soon as possible in addition to intravenous frusemide (1-2 mg/kg). The drug of choice for VT, particularly if under anaesthesia is lignocaine (without adrenaline) at 0.5mg/kg slowly IV, repeated after 5-10 minutes if required (generally only repeated 3 times prior to progressing to an alternative antiarrhythmic drug). Hyperexcitability may occur in conscious patients – can pre-treat with 0.05 mg/kg diazepam IV. Quinidine (ideally quinidine gluconate 0.5-2.2 mg/kg iv bolus), propafenone (0.5-1 mg/kg in 5% dextrose iv), procainamide (1 mg/kg/min iv to a dose of 20 mg/kg) and magnesium sulphate (25g slow iv) can also be used.

If the VT is due to primary cardiac disease the prognosis is poor. Euthanasia is the best management in these cases. Horses with VT that is converted to NSR should be rested for at least 4 weeks prior to re-evaluation, including exercising ECGs. Horses affected with a single episode generally have a favourable prognosis. Follow up should be performed annually

9) Accelerated idioventricular rhythm

This is where a ventricular or nodal/junctional ectopic focus dominates the rhythm at a rate only slightly faster than sinus rate (usually around 50bpm). The QRS complexes are wide and bizarre with an idioventricular rhythm, but will be normal with an idionodal/junctional rhythm. The QRS complexes have no relationship with the P waves (AV dissociation). If the rate is stable there may be no severe effects on cardiac output at rest. This arrhythmia is of unknown aetiology, the significance of this abnormality in horses is unclear, but further cardiac work up is indicated.

10) Ventricular pre-excitation

In ventricular pre-excitation an abnormal or 'accessory' pathway exists between the atria and ventricles, bypassing the AV node. On ECG the P-R interval is very short or absent, and the QRS is wide and abnormal. Horses with ventricular pre-excitation may be asymptomatic. In other species re-entrant SVT can occur secondarily. The significance of this abnormality in horses is currently unclear.

Acquired valvular disease

Murmurs associated with valvular dysfunction are relatively common in horses. They must be differentiated from functional murmurs on auscultation. In contrast to small animals, valvular stenosis is extremely rare in horses. Most pathological murmurs are associated with valvular incompetence and regurgitation.

1) Aortic regurgitation

Aortic regurgitation is a common finding in teenage and older horses. The murmur is characteristic – a decrescendo, holodiastolic murmur with the point of

maximal intensity (PMI) over the left side heart base (~4th intercostal space). The murmur radiates toward the apex of the heart. The murmur is often musical in quality, and may also radiate to the right side of the chest. Bounding arterial pulses are an indication of hemodynamically significant aortic regurgitation.

Aortic regurgitation is usually the result of age-related changes to the valve leaflets. ***Although left-sided volume overload is inevitable, progression may be extremely slow.*** Horses may be asymptomatic, or may show signs of progressive exercise intolerance. Clinical signs of left and/or right-sided heart failure are rare.

The intensity of the murmur is not a good indicator of the significance of the regurgitation. An echocardiogram is indicated to establish the degree of valvular dysfunction and the presence/degree of chamber enlargement.

Generally, horses with mild aortic regurgitation that is not associated with clinical signs of congestive heart failure or exercise intolerance are safe to ride. However, if bounding arterial pulses are present/ moderate to severe AR is present an exercising ECG is essential as ventricular arrhythmias may develop secondary to decreased myocardial perfusion by the coronary arteries associated with severe aortic regurgitation and the resultant reduction in diastolic pressure (leads to reduced coronary filling). Progression to cardiac failure is usually extremely slow. In all cases an echocardiogram is indicated to assist in making a prognosis for future athletic activity, along with future examinations to assess disease progression. Aortic regurgitation will eventually result in left atrial enlargement which places the horse at increased risk of developing atrial fibrillation.

2) Mitral regurgitation

Mitral regurgitation is more common in middle-aged to older horses. It is usually the result of degenerative changes to the mitral valve leaflets, though the aetiology is unclear. It results in a pan/holosystolic, plateau (band-shaped) murmur with the PMI over the apex of the heart (5th intercostal space) on the left hand side. The murmur radiates toward the heart base. There is no variation in the quality of the murmur with exercise.

Rupture of a mitral valve chord is a less common cause of mitral regurgitation in horses, and frequently results in acute signs of left-sided cardiac failure (pulmonary edema, tachypnoea, exercise intolerance). The murmur may have a harsh coarse vibrant or musical honking quality. It radiates widely over the left side of the chest and is usually associated with a thrill.

Mitral regurgitation may be an incidental finding in horses with no clinical signs of cardiac disease. Progression may be very slow depending on the degree of insufficiency, though it is difficult to predict. Atrial enlargement, left-sided heart failure, pulmonary hypertension, and right-sided heart failure are sequelae.

Murmurs with greater intensity (> grade 2) are generally associated with more severe pathology, although it can be difficult to gauge the significance of mitral regurgitation using clinical examination alone. ***Moderate mitral regurgitation is likely to***

limit performance in athletic horses. An echocardiogram is indicated in all cases of mitral regurgitation. Exercising ECGs are recommended in all cases of moderate to severe MR. Follow-up examinations are useful to track the progression.

A horse with a low grade mitral regurgitant murmur, with no clinical signs of cardiac disease and no apparent exercise intolerance is generally safe to ride. Such horses should always be monitored carefully with repeat examinations. Owners should be offered an echocardiogram for a more accurate prognosis.

3) Tricuspid regurgitation

Tricuspid regurgitation occurs frequently in horses (particularly Standardbreds). The murmur is pan/holosystolic, plateau (band-shaped) with the PMI over the right side of the chest (4th-5th intercostal space). The murmur usually radiates cranially. There is no variation in intensity with exercise.

Often the tricuspid valve is morphologically normal in cases of tricuspid regurgitation. Pulmonary hypertension (from severe obstructive airway disease) or normal concentric hypertrophy of the right side of the heart (in response to athletic training) may be aetiological factors. Tricuspid regurgitation usually does not change or progresses slowly. Severe tricuspid regurgitation may lead to exercise intolerance, atrial fibrillation, and signs of right sided heart failure (venous distension, jugular pulsations, peripheral edema, ascites, hepatic congestion).

Tricuspid regurgitation is only rarely associated with a reduction in performance, and does not generally progress to cardiac failure. In horses with a low-moderate grade murmur that are presented for poor performance, tricuspid regurgitation should only be considered if other (more likely) potential causes are ruled out. An echocardiogram is indicated in horses with signs of cardiac disease or exercise intolerance, when the source of the murmur is in question, if the murmur is grade 4 or louder and/or in the presence of thrombophlebitis/ fever of unknown origin.

4) Valvular endocarditis

Valvular endocarditis is rare in horses. It is caused by bacterial infection of the endocardium, with subsequent damage to the valves. The aortic and mitral valves are more commonly affected than the tricuspid or pulmonary valves.

Horses with valvular endocarditis are systemically ill. Pyrexia, depression, tachypnoea, tachycardia, weak pulse and injected mucous membranes are commonly present. Intermittent lameness, signs of heart failure, and peripheral edema may also be present. In more protracted cases, intermittent fever with weight loss may be the dominant features. A murmur associated with insufficiency (occasionally stenosis) of the affected valve(s) will be present, and may be severe. Arrhythmias may be present. The

source of infection may not be apparent, although jugular phlebitis may occasionally be involved.

Further investigation should include haematology and serum biochemistry, as well as an echocardiogram. High globulin and fibrinogen, together with a leukocytosis (neutrophilia) are commonly seen. Blood culture can occasionally lead to isolation of the offending organism.

Horses with valvular endocarditis generally have a grave prognosis, depending on the extent of damage to cardiac structures. Treatment is often unrewarding. High dose, prolonged, broad spectrum antimicrobials (eg high doses of penicillin & gentamicin) together with supportive therapy (fluids, electrolytes and non-steroidal anti inflammatory), as well as antithrombotic therapy may be successful in some cases. Blood culture, if successful, may enable more precise antimicrobial therapy. Repeat echocardiographic examinations are essential to monitor the disease process. Valvular regurgitation may continue to progress after a bacteriologic cure is achieved due to damage to the valve leaflets.

Congenital cardiac disease

1) Ventricular septal defect (VSD)

VSD is the most common congenital cardiac disorder in horses. The defect is usually in the membranous portion of the septum, just below the aortic valve. VSD commonly occurs on its own, but may be part of a more complex disorder such as tetralogy of Fallot.

Clinical signs vary widely depending on the size of the defect and the consequent haemodynamic effects. ***The most common presentation of a horse with VSD is poor performance on beginning athletic training as a yearling/young adult.*** Because of the pressure gradient VSDs result in left to right shunting of blood. Oxygenated blood is shunted across the VSD to the right ventricle and out the pulmonary artery. This primarily leads to a volume overload of the left atrium, left ventricle, and pulmonary vasculature due to location of the VSD. Large defects can result in severe signs soon after birth including tachypnoea, dyspnoea, collapse and pulmonary oedema. Smaller defects may be asymptomatic.

VSDs result in 2 murmurs:

1. **A plateau-type pansystolic murmur with the PMI on the right side** of the chest (3rd-4th intercostal space) will be present associated with shunting of blood through the VSD into the right side of the heart. There is usually a palpable thrill. The murmur may radiate widely, and can sometimes be heard over the sternum and left side of the chest.

2. The second murmur is ausculted over the **pulmonary area (L side of the thorax)**. This murmur is generally 1 grade softer than the right sided murmur and is crescendo decrescendo in quality. It is an ejection murmur associated with increased blood flow through the pulmonary artery (**relative pulmonic stenosis**).

Echocardiography will confirm the presence of a VSD, its size, and hemodynamic significance, as well as give an indication of the degree of cardiac compromise. This will assist in providing a prognosis. Horses with a small perimembranous VSD (<2.5cm) may be asymptomatic and can perform high intensity athletic activities. They have a good prognosis for life, and occasionally can even compete successfully as athletic horses. Horses with larger, more hemodynamically significant VSDs with concurrent myocardial or valvular pathology may have a shortened life expectancy and develop congestive heart failure. Exercise ECGs should be performed on all horses with moderate to large VSDs that are intended for ridden use.

2) Patent ductus arteriosus (PDA)

The ductus arteriosus closes in normal foals within 24h of birth. Patent ductus arteriosus is an extremely rare condition in horses. PDA results in a machinery (continuous) murmur with the PMI over the left heart base. The prognosis is usually poor if the PDA is present longer than 96 hours after birth. The condition is usually associated with a more complex condition (eg tetralogy of Fallot).

4) Tetralogy of Fallot

1. Ventricular septal defect.
2. Overriding, dextroposed aorta.
3. Pulmonic stenosis
4. Right ventricular hypertrophy (secondary lesion).

The overall consequences are reduced blood flow to the lungs, and non-oxygenated blood entering the systemic circulation (from the right ventricle, via the dextroposed aorta). The clinical presentation depends upon the severity of the right ventricular outflow tract obstruction and the size of the ventricular septal defect. Often there is marked growth retardation, severe exercise intolerance, dyspnea, tachycardia and syncope. There may be cyanosis, particularly after exercise.

There is a characteristic, loud (Grade 4-6) pansystolic murmur that radiates widely over both sides of the chest (PMI over the pulmonic and tricuspid areas on the left and right sides). Prognosis is grave.

Myocardial disease

Primary myocardial disease can occur as the result of inflammatory, degenerative or toxic processes.

Primary myocarditis may be idiopathic or associated with viral infection. This condition often results in poor performance in athletic horses, but can also result in exercise intolerance collapse and sudden death. Arrhythmias may be present. Echocardiography is useful in diagnosing the condition. Resting heart rates may be normal or elevated but exercising heart rates are usually elevated and the horse shows prolonged recovery after exercise. Haematology and serum biochemistry will confirm the presence of inflammation and or infection. After ruling out other causes of poor performance, complete cardiac examination including echocardiogram is indicated.

Primary myocardial disease can result from monensin or persin toxicoses – see your toxicology notes.

Pericardial disease

Pericarditis is rare in horses. It may be idiopathic, viral, bacterial or associated with concurrent pleuropneumonia. Pericardial effusion (usually a modified transudate) results from inflammation of the pericardial sac.

Clinical signs are variable, from poor performance in mild cases to overt signs of acute cardiac failure with more severe effusions. Venous distension, jugular pulsations ventral edema, depression, pyrexia, weight loss, dyspnoea and tachycardia may be present depending on the initiating disease process and the severity. Cardiac auscultation reveals tachycardia, muffled heart sounds and/ or pericardial friction rubs and absent lung sounds in the ventral portion of the thorax.

Echocardiography provides a definitive diagnosis. Severe pericardial effusion with cardiac tamponade may be life threatening, and drainage is required. Pericardiocentesis is performed under ultrasound guidance and is a specialist procedure. Antibiotics and antiinflammatories are administered as indicated. Mild pericarditis secondary to viral lung disease may require no specific treatment. In these cases rest, anti-inflammatory therapy and re-evaluation are indicated.

Congestive heart failure (CHF)

Horses rarely present with overt signs of CHF due to the large cardiac reserve. There is usually a history of progressive exercise intolerance. Weight loss, weakness and lethargy may be present. Mucous membrane colour is often normal, though capillary refill time may be prolonged. The pulse may be weak, and pulse deficits may be present if there is an arrhythmia. Jugular vein distension and pulsations are common. Oedema, particularly under the chest may be a feature in advanced cases. Tachypnoea is often present, particularly if there is pulmonary oedema. ***The heart rate is elevated (>45 bpm) at rest.*** A murmur will usually be audible depending on the aetiology.

An echocardiogram is always indicated in cases of CHF to gauge the degree of cardiac decompensation. Provision of an accurate prognosis is the most important aspect in these cases. An ECG should be performed if an arrhythmia is auscultated.

Specific treatment is rarely indicated in horses with CHF. Specific antiarrhythmic therapy may be provided as required. Therapy for CHF in horses follows the same principals as for small animals. Diuretics (furosemide 1mg/kg IM 3-4 times/day), ace inhibitors (enalapril 0.25-0.5 mg/kg PO *s.i.d.* or *b.i.d.*) and positive inotropic drugs (digoxin 10µg/kg *b.i.d.*) have been used. Even with treatment prognosis is poor once significant clinical signs have developed (maximum of 6 months life expectancy).