Recommendations for Equine Athletes with Cardiovascular Abnormalities
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Abstract

Murmurs and arrhythmias are commonly detected in equine athletes. Assessing the significance of these cardiovascular abnormalities in the performance horse can be challenging for the veterinary clinician. Determining the impact of the cardiovascular abnormality on the horse’s performance, life expectancy, safety (horse and rider) and the owner’s expectations for the future are paramount. This consensus statement presents a general approach to the assessment of cardiovascular abnormalities, followed by a discussion of the common murmurs and arrhythmias. The description, diagnosis, evaluation and prognosis for each cardiovascular abnormality are discussed. A comprehensive assessment of the cardiovascular abnormality detected is essential to determine its severity and formulate a recommendation for the equine athlete. The recommendations presented are based the available literature and the consensus of the panelists. While, the majority of horses with cardiovascular abnormalities have a useful performance life, periodic re-examinations are indicated for horses with significant cardiovascular disease. Horses with pulmonary hypertension, congestive heart failure, complex or malignant ventricular arrhythmias are unsafe to ride.

Keywords: Murmurs, arrhythmias, echocardiography, exercise testing
Cardiac murmurs and arrhythmias are often identified in horses engaged at all levels of performance across the entire spectrum of equine sports. The challenge for the clinician is to determine the impact of any cardiovascular (CV) abnormality on the horse’s performance (present and future), on rider and horse safety, and to consider any long-term effects on the horse’s health and longevity.

The recommendations contained herein were developed during recurrent and protracted discussions with active participation by all seven panelists. The available literature (including, high-grade research evidence, retrospective studies, case reports) was used whenever possible and a comprehensive list of the references used along with other supporting content has been included at <URL>. However, since very limited literature is available in many important areas, our collective personal experience constitutes the basis of most of these guidelines. As might be expected, some clinical issues were associated with more divergent opinions than others; but overall, the panelists achieved “consensus” (indicating that 6 to 7/7 panelists supported the recommendation). We emphasize that the following guidelines pertain specifically to horses used in performance-based activities and are not necessarily generalizable to all horses. Furthermore, the reader should anticipate that modifications in these recommendations will occur based on the results of future research, including that recommended by the panel at the conclusion of this report.

**Abbreviations**

AF = atrial fibrillation  
AIVR = accelerated idioventricular rhythm  
AR = aortic regurgitation  
AVB = atrioventricular block  
CHF = congestive heart failure  
Echo = echocardiogram  
EIPH = exercise induced pulmonary hemorrhage  
HR = heart rate  
LA = left atrium  
LV = left ventricle  
LVD = left ventricular dysfunction  
MR = mitral regurgitation  
MVP = mitral valve prolapse  
NSR = normal sinus rhythm  
PAC = premature atrial complex  
PHT = pulmonary hypertension  
PMI = point of maximal intensity  
PVC = premature ventricular complex  
RA = right atrium  
RCT = ruptured chordae tendineae  
RV = right ventricle  
TR = tricuspid regurgitation  
VA = ventricular arrhythmias  
VT = ventricular tachycardia  
VSD = ventricular septal defect

**General Approach**

Before discussing specific recommendations for equine athletes, familiarity and expertise in equine echocardiography (Echo), electrocardiography (ECG), exercise testing, interpretation of laboratory tests and a thorough understanding of the pathophysiology of equine heart disease are needed. Most management decisions and risk assessments are based on these evaluations, along with the performance history, physical examination findings, athletic demands placed on the horse and owner expectations.

With this approach, most cardiac lesions can be “graded” as mild, moderate, or severe. Prognosis tends to track these grades, accepting that forecasting the performance longevity and lifespan of an equine athlete with a “mild” or “severe” heart disease is relatively straightforward when compared to one of “moderate” lesion that may be surprisingly well tolerated or rapidly
decompensating. Horses with a defined cardiac lesion warrant a schedule of follow-up examinations to refine the prognosis, reassure the horse owner, and anticipate and manage medical complications. Objective, negative prognostic factors in the setting of structural heart disease include the development of progressive chamber remodeling (dilatation and altered chamber shape) and dysfunction, great vessel enlargement, pulmonary hypertension (PHT), pathologic arrhythmia, or overt congestive heart failure (CHF). Horses with PHT and CHF are unsafe to ride and standard treatment for CHF is indicated.

**Echocardiography**

The *history*, cardiac rhythm, and the results of auscultation should be known before interpreting the Echo. The Panel specifically recommends an Echo when the following *indications* exist: 1) a previously-diagnosed “functional” murmur that is louder on serial examinations; 2) a grade 3-6/6 left-sided murmur compatible with mitral regurgitation (MR) or aortic regurgitation (AR); 3) a grade 4-6/6 right sided systolic murmur compatible with tricuspid regurgitation (TR); 4) suspected ventricular septal defect (VSD) or other congenital heart lesion; 5) continuous or combined systolic-diastolic murmurs; 6) pathologic arrhythmias, whether a murmur is present or not, and 7) suspected myocardial injury/damage.

A *complete echocardiographic study* should address the following: 1) morphologic lesions; 2) motion abnormalities; 3) cardiac chamber and great vessel size; 4) cardiac valve function; 5) blood flow disturbances; 6) global and regional ventricular systolic function; 7) hemodynamic estimates, and 8) ventricular diastolic function and filling pressures (accepting these two are challenging in mature horses). A *comprehensive assessment* requires the application of complementary 2D, M-mode, and Doppler modalities. This standard does not mitigate the value of a directed 2D Echo for specific clinical issues such as cardiac size, ventricular function, and myocardial grayscale in a horse with an isolated arrhythmia.

**Exercise Testing**

Exercise testing is an important component of CV assessment and should be at an intensity that is at or slightly exceeding the horse’s normal activities. In horses with significant structural lesions, intermittent premature complexes, or lone AF that cannot be converted to sinus rhythm, exercise testing can determine if the HR is appropriate for the work performed or if an arrhythmia deteriorates over the course of the test. Exercise testing is also indicated during a prepurchase examination when a non-functional heart murmur or sporadic arrhythmia is identified. An exercise test should not be performed when there is CHF, severe valvular regurgitation with secondary AF, PHT, or suspected myocardial disease or when a ventricular arrhythmia of dangerous complexity is present.

Specific noninvasive cardiac assessments include the following: 1) the effects of exercise on auscultation (rate, rhythm, and murmurs); 2) peak HR during exercise; 3) time interval and HR responses during the warm-up to peak exercise; 4) time interval and HR responses during recovery from work; 5) heart rhythm before, during, and immediately following exercise; and optionally, 6) pre- and post-exercise (stress) Echo. Some method of inducing unexpected sympathetic stimulation should be included in the exercise test to identify an inappropriate HR, aberrant conduction, or ectopy associated with adrenergic stimulation. Exercising ECGs should only be obtained with a device that has permanent storage and playback capabilities. Additional tests that might be indicated are analysis of gait, airway dynamics, arterial blood gas tensions and other clinical laboratory tests.
Cardiac Murmurs

A complete Echo is the diagnostic test of choice when evaluating a horse with a cardiac murmur, although occasionally horses are not further evaluated when an examiner is confident a murmur is functional and performance has been satisfactory. However, an Echo is generally advised when auscultatory findings are incongruous with a physiologic murmur, the murmur is moderate to loud, or when a murmur is detected as part of a pre-purchase examination. Loudness does not correlate to severity, especially with musical murmurs. Mild valvular regurgitation is often detected in horses with soft murmurs, and seemingly does not affect performance or health.

A number of Doppler findings can be used to assess severity of valvular regurgitation, but none of these are reliable in isolation. Both width of the vena contracta and jet area can be used to assess severity. Doppler studies often also reveal physiological valvular regurgitation silent to auscultation. Serial echocardiographic evaluations are more meaningful than findings from a single examination.

Mitral Regurgitation

Mitral regurgitation (MR) is a common finding in horses performing in all sports disciplines. MR is usually mild and associated with normal performance and life expectancy. If significant MR develops at an early age, it is the valvular lesion, most likely to shorten life expectancy.

Some of the underlying lesions responsible for MR can be visualized by 2D Echo such as mitral valve dysplasia, degenerative or inflammatory valve thickening (including bacterial endocarditis), prolapse (MVP), thickened or ruptured chordae tendineae (RCT), and flail leaflet. MR also can develop secondary to dilatation of the MV annulus (as with severe AR, nonrestrictive VSD or rarely, dilated cardiomyopathy) or papillary muscle ischemia or fibrosis. Diagnosis and Evaluation

The mitral valve and support apparatus should be thoroughly examined by 2D and M-mode Echo for structural and motion abnormalities and identification of the most likely etiology for MR. Valvular or chordal thickening may be evident, but in mild cases, judgments will be subjective. The echocardiographic diagnosis of MVP has not been well defined in the horse, and the saddle-shape of the valve makes the diagnosis challenging. Convex bulging of a mitral leaflet into the LA with an associated jet of MR on Doppler studies is consistent with MVP. A RCT is detected as an echoic, whip-like structure moving into the LA during systole which may flip into and out of the imaging plane. A flail leaflet is diagnosed when part of the valve leaflet is imaged in the LA and moving independent of the rest of the mitral valve. Endocarditis may appear as focal raised lesions on the atrial surface of the valve or as oscillating vegetations or thrombi attached to the valve.

Except in cases of acute MR associated with endocarditis, chordal rupture or papillary muscle dysfunction, there should be progressive changes in LA and LV size and shape that reflect the severity of MR. An absence of remodeling is consistent with mild MR. Marked enlargement and signs of PHT are typical of severe MR. Assessment of LV systolic function can be confusing due to increased preload and reduced afterload. When MR is acute and severe, the LV will be hyperdynamic with increased fractional shortening. Diastolic compression of the right ventricle with exuberant septal motion also suggests severe LV volume overload. With chronic MR associated with progressive remodeling and LVD, the fractional shortening returns...
to the normal range or becomes lower than normal.

Importantly, a MR jet that is eccentric, wall-hugging, or flat can lead to underestimation of severity. Multiple jets are often present and the short-axis image plane and other cardiac windows can be useful to identify these. The duration, density and shape of the CW Doppler regurgitant signal are also useful in assessing MR severity.

**Prognosis (Table Two)** Assessing the severity of MR and predicting its clinical course is difficult. Although MR is unlikely to affect performance unless it is relatively severe, LA enlargement increases the risk for AF. Major negative prognostic indicators for horses with MR include moderate to severe regurgitation, endocarditis, RCT, flail leaflet, severe valvular thickening, concurrent PHT, or significant MR with AF.\(^9,^{12}\)

**Summary: Recommendations for Mitral Regurgitation**

- Determine the most likely etiology
- Assess severity based on combined assessment of performance history, exercise testing, clinical examination and echocardiographic findings
- Re-examine at least annually or every other year for mild MR
  - Horses with mid-to-late crescendo murmur and mild MR usually have a favorable prognosis
- Ensure that HR and rhythm are monitored on a regular basis in horses with moderate to severe MR
  - Increased HR or irregularly irregular rhythm suggests AF or may indicate progression
- If AF develops or if significant progression of MR occurs, in the absence of signs of CHF, perform an ECG exercise test.
- Manage complications of advanced disease. Limited data have been published about ACE inhibitors and no consensus could be reached regarding their use in MR in the absence of CHF.

**Aortic Regurgitation**

Aortic regurgitation (AR) is a common finding in older horses, often detected incidentally.\(^{13}\) Any holodiastolic murmur is assumed to indicate AR unless proven otherwise. The underlying lesions may be evident on 2D Echo. Degenerative valve thickening and aortic valve prolapse are most frequently detected.\(^{13,^{14}}\) Other causes include congenital malformations, leaflet tearing, infective endocarditis, valvulitis, fenestrations, and aortic root disease. AR is also observed in association with some VSDs (see below).

The condition is often mild and associated with normal performance and life expectancy.\(^{11}\) However, when AR is moderate or severe or first recognized in a younger horse (<10 years of age), the risks for reduced performance life and longevity are higher. Sudden death associated with fatal ventricular arrhythmias has been observed in horses with moderate to severe AR and can occur in isolation, without poor performance or CHF.

**Diagnosis and Evaluation** Bounding or hyperdynamic arterial pulses suggest hemodynamically severe AR with LV volume overload.\(^{13}\) Noninvasive blood pressure measurement is useful to identify widening of pulse pressure.\(^{15}\) An exercising ECG is recommended if there is cardiac remodeling and/or performance issues, with emphasis placed on exercise-induced PVCs and the appropriateness of the exercising HR. A continuous 24h-Holter electrocardiogram can be considered to further identify and quantify ventricular arrhythmias.
2D Echo may demonstrate valve thickening as a fibrous band like-lesion, which appears as an echoic line parallel to the free edge of the leaflet. It is most often observed on the left coronary cusp. Less often, nodular thickening or a generalized increase in echogenicity of the leaflet free edge is detected. Prolapse, especially involving the noncoronary cusp, is another common finding with convex, apical bulging of a portion of the leaflet into the LVOT. False positives are common with misalignment of the ultrasound beam. A jet of AR surrounding the prolapsed portion of the leaflet should be evident by color Doppler imaging. Diastolic fluttering or vibrations affecting the free edge of a leaflet, especially the left coronary cusp, are often observed in horses with musical AR murmurs, consistent with degeneration and redundancy or fenestration of the leaflet. A flail leaflet is detected rarely and indicates a torn or avulsed portion of the valve. Endocarditic lesions (nearly as frequent here as on the mitral valve) are an uncommon cause of AR and may be evidenced as raised lesions, thickenings, or a large oscillating mass depending on the severity and chronicity of disease.

M-mode studies may demonstrate diastolic fluttering of the mitral or aortic valves, aortic root, or ventricular septum should the jet of AR impinge one of these structures. Eccentric jets directed towards the mitral valve can prevent full opening of that leaflet. Premature closure of the mitral valve on M-mode Echo is a sign of markedly increased LV end-diastolic pressure and indicates severe AR.

Comments made previously about volume overload, remodeling, PHT and LV systolic function for MR are also appropriate for AR. Enlargement of the aortic root also can occur with significant, long-standing AR. LA dilatation may indicate ventricular dysfunction, volume retention, or concurrent MR. The latter can develop due to AR-induced dilatation of the LV, mitral annulus, and LA or from degenerative mitral disease. Assessment of LV systolic function is difficult, especially in acute AR, but with chronic disease, overt LVD may be present. CW Doppler studies including regurgitant signal duration and density, pressure half-time, and the velocity time integral of AR compared to forward flow may have merit.

Prognosis (Table Three) Assessing AR severity and predicting its clinical course is challenging. Findings compatible with mild AR and examinations that are static over time relate to a better prognosis for work and life. The detection of hyperkinetic arterial pulses and a pulse pressure of > 60 mm Hg suggest that progression of AR is likely. The detection of exercise-induced ventricular arrhythmias is considered an important negative prognostic indicator and these horses are considered less safe to ride than their age-matched peers by most panelists. All agreed that these horses should not be ridden by a child or used as a lesson horse or in a high-risk sport owing to a risk for sudden death. Although AR is unlikely to affect performance unless it is severe, once LA enlargement occurs, the risks of AF, PHT, and CHF are higher.

Summary: Recommendations for Aortic Regurgitation

- Determine the most likely etiology
- Assess severity based on combined assessment of performance history, exercise testing, clinical examination and echocardiographic findings
- Re-examine twice yearly when there is moderate to severe AR and at least annually thereafter if minimal progression is evident. Similarly, longer follow-up intervals are appropriate for horses with mild AR after the first re-evaluation.
- Ensure HR and rhythm are monitored on a regular basis in cases of moderate to severe AR; an increased resting HR or an irregularly irregular rhythm suggesting AF or PVCs indicate progression.
If AF develops in a horse with mild to moderate AR, perform an ECG exercise test
Manage complications of advanced disease. Limited data have been published about ACE inhibitors and no consensus could be reached regarding their use in AR in the absence of CHF.

Tricuspid Regurgitation
Tricuspid regurgitation (TR) is a common finding in equine athletes, especially in horses performing high-intensity work.\textsuperscript{7,16} The prevalence of and severity of TR appears to be influenced by both age and level of training.\textsuperscript{7,17,18} The precise mechanisms of training-associated TR are unknown, and leaflets abnormalities are infrequently detected on 2D Echo. Infrequently, thickening of valve leaflet or RCT are observed, but much less often than for MR. Horses with severe MR and PHT associated with CHF as well as (less commonly) horses with severe respiratory disease develop TR. Endocarditis infrequently affects the TV and if it does, it is usually secondary to septic jugular thrombophlebitis. Congenital tricuspid dysplasia is rare.

Diagnosis and Evaluation
Although a Doppler Echo confirms TR, it is not an essential study for horses with a grade 1-3/6 murmur. Exceptions include: 1) poor performance, 2) pre-purchase examination, 3) concurrent thrombophlebitis, and 4) fever of unknown origin.

With benign or training-related TR the valve is structurally normal, the RA and RV are normal in size, and the regurgitant jet is usually thin and directed towards the aorta. With clinically significant TR there may be structural or motion abnormalities of the TV with RA and RV enlargement. The jet width is wider at the origin, tends to occupy a larger area in the RA, and is often directed centrally or towards the lateral RA wall. Finding a zone of proximal flow convergence on color Doppler echocardiography supports moderate to severe TR. When the TR jet velocity is >3.5 m/s, PHT is suspected, in which case the patient should be scrutinized for left-sided heart disease or cor pulmonale. The effect of inflammatory airway disease and recurrent airway obstruction on TR is unknown but is likely to be minor and associated cardiac disease is potentially reversible with therapy.\textsuperscript{19} Significant TR also can progress to atrial flutter, AF or CHF, though less often than MR.

Prognosis
Negative prognostic indicators include TR associated with: 1) structural valve lesions (endocarditis, RCT, flail leaflets); 2) clinical signs of right-sided CHF; 3) severe MR and PHT; or 4) severe cor pulmonale.

Summary: Recommendations for Tricuspid Regurgitation
- Appreciate the high prevalence of training-induced TR in high-performance horses
- Perform comprehensive clinical and echocardiographic examinations when indicated (see above)
- An annual echocardiographic re-examination is indicated for horses with moderate and severe TR.

Ventricular septal defect
VSD is the most common congenital heart defect with some breed predispositions suspected (Section A Welsh Mountain ponies\textsuperscript{20}, Standardbreds,\textsuperscript{21} and Arabian horses\textsuperscript{21,22}). The location of the defect influences both auscultation and imaging. The typical VSD is perimembranous located ventral to the tricuspid leaflet, and below the junction of the right and
non-coronary cusps of the aortic valve. Less common lesions are subarterial (also called subpulmonic, or outlet) – beneath each semilunar valve – and muscular (apical) VSDs. When the aorta straddles part of ventricular septum, the descriptor “malalignment” is used and the risk of aortic valve prolapse increases. VSDs are often a component of more complex congenital cardiac defects, whereupon, the associated cardiac murmurs may differ from those described below. Multiple VSDs are rare.

**Diagnosis & Evaluation**

Definitive diagnosis requires an Echo. Complex congenital disease must be excluded. The typical defect is below the aortic valve and adjacent to the septal tricuspid valve leaflet, ventral to the junction of the right and noncoronary aortic cusps. The size of the VSD is often underestimated with 2D Echo and overestimated with color-flow Doppler Echo. The largest systolic diameter of the defect in two mutually-perpendicular planes should be measured. In general, defects ≤ 2.5 cm in a 450 – 500 kg horse are less likely to be hemodynamically significant.

Usually there is some enlargement of the left side of the heart with a left-to-right shunt; however, cardiac dimensions may fall within the normal range if the shunt is small. Moderate to severe enlargement of the LA and LV are concerning and increase the risk for AF, PHT, and CHF. Noticeable systolic enlargement of the pulmonary artery is an indication of pulmonary over-circulation. Marked pulmonary artery enlargement during both systole and diastole suggests PHT; a finding that can be substantiated by CW Doppler interrogation of any TR and/or PR jet.

Assuming parallel alignment between ultrasound beam and shunt flow, the maximal shunt velocity measured by CW Doppler estimates the pressure gradient across the defect. Peak velocities >4.5 m/s suggest a restrictive defect with better prognosis. With good alignment peak velocities > 5 m/s are expected. The functional size of the defect can be reduced by prolapse of the aortic valve into the VSD, tricuspid valve adhesions, or fibrous tissue proliferation. Lower velocities (<4 m/s) along with higher PA ejection and mitral inflow velocities indicate a more significant shunt.

It is important to identify aortic malalignment, aortic valve prolapse into the VSD, and AR, and to detect MR or TR and any other comorbidity (such as multiple VSDs). If moderate to severe right ventricular wall hypertrophy is found, a large shunt, PHT, or complex congenital disease should be assumed.

**Prognosis (Table Four at [URL])**

The most important prognostic criteria include: 1) size of VSD; 2) size of the cardiac chambers; 3) maximal shunt velocity; 4) presence of significant AR or MR; 5) PHT; 6) CHF. The horse with a small VSD has an excellent prognosis and should experience a normal performance life. Closure of a small VSD has been reported but is believed rare. Moderate defects may be tolerated with a normal performance life competing in disciplines that are less strenuous. These horses could deteriorate depending on concurrent lesions, especially when progressive MR or AR is identified on follow-up examinations. The finding of a large diameter, unrestricted VSD portends a poor prognosis, limited or no performance life, and shortened life expectancy. Progressive LA enlargement can lead to AF and CHF.

**Summary: Recommendations for Ventricular Septal Defect**

- Perform comprehensive clinical and echocardiographic examinations
- Re-examine at least annually
- Perform exercise (ECG) testing of horses with moderate to large VSDs, in prepurchase situations, or when performance is suboptimal
Consider horses with a small VSD and minimal cardiomegaly as safe to compete; evaluate larger defects on a case-by-case basis in consultation with a specialist experienced in equine cardiology.

Consider affected horses unsuitable for breeding.

**Aorto-cardiac fistula**

A continuous machinery murmur loudest on the right side of the thorax with bounding arterial pulses is characteristic of a horse with an aorto-cardiac fistula. Many horses present with an acute onset of exercise intolerance and pain, often perceived as colic, and ventricular tachycardia (VT). Horses with an aorto-cardiac fistula are **not safe to use** for performance.

**Cardiac Arrhythmias**

Arrhythmias can develop as isolated electrical disorders (e.g., lone AF or complete heart block) or secondary to other etiological factors, including: 1) structural heart disease, 2) metabolic and endocrine disorders; 3) systemic inflammation; 4) hypotension, hemorrhage, anemia, and ischemia; 5) autonomic influences; 6) toxicosis/envenomation; and 7) drugs.

**General Comments**

Electrocardiography (ECG) is the test of choice for confirming the diagnosis of heart rhythm disturbances. A base-apex (rhythm strip) ECG is usually sufficient, but additional surface leads, intracardiac leads, or transesophageal leads may be needed to better evaluate the ECG waveforms in some cases. A portable ECG unit may be useful in the field for documenting arrhythmias. An exercising ECG is usually indicated to determine if an arrhythmia has potential for impairing performance or might become a safety issue.

The workup of the horse with an arrhythmia should include: 1) a history including all drugs and supplements administered; 2) a complete Echo; and 3) appropriate laboratory tests.

The examiner must be familiar with physiologic, vagally-mediated arrhythmias that include sinus arrhythmia, second degree AVB, and sinoatrial block. These are normal at rest and in the immediate post-exercise period.

**Second Degree Atrioventricular block (AVB)**

This rhythm is normal in athletic horses. The HR is in the low normal range and there are usually several conducted beats before each blocked beat. Auscultation is characterized by a repetitive pattern with an irregular rhythm, with occasional fourth (atrial) heart sounds that are not followed by first and second heart sounds. Physical activity or startling to increase sympathetic tone should cause the arrhythmia to disappear, although AVB is likely to resume very quickly.

**Diagnosis and Evaluation** In some cases, an exercising ECG is needed to confirm the physiologic basis of the arrhythmia. When second degree AV block results in a P:QRS conduction sequence of 2:1, 3:1, or more, the term “high-grade” is used and the rhythm considered abnormal. If the horse with high-grade second degree AVB cannot be exercised, an atropine response test can be used instead to determine if a 1:1 conduction occurs. A continuous 24 hour ECG (Holter) monitoring with simultaneous video recording should be obtained when there is a history of collapse.

**Summary: Recommendations for High-Grade Second Degree AV Block**
- Horses with high-grade second degree AVB that disappears with exercise should only be ridden by an informed adult, but the HR and rhythm should be frequently monitored.
- Horses with high-grade second degree AVB during exercise or after atropine administration should be rested and re-evaluated; they are not considered as safe to ride as their age-matched peers.
- Horses with symptomatic bradyarrhythmias generally have a poor prognosis and are not as safe to ride as their age-matched peers.

**Atrial Fibrillation (AF)**

AF is the most common arrhythmia affecting performance and appears more often and as a heritable lesion in some Standardbred racehorses. With acute onset of AF, spontaneous conversion to normal sinus rhythm (NSR) can occur, usually within 24-48 hours. This is referred to as *paroxysmal AF*. AF in the absence of detectable underlying heart disease is called *lone AF*. Microstructural lesions or channelopathies that predispose to AF are likely present in some of these horses, but cannot be detected using routine diagnostic tests. When AF follows structural heart disease, the term *secondary AF* is used. Affected horses are predisposed to *persistent*, *recurrent* and *permanent* AF.

AF is usually recognized during auscultation and is characterized by an irregularly irregular rhythm that can sound like a combination of premature beats and long pauses. The atrial (fourth) sound is absent. The resting HR is usually normal. Resting tachycardia suggests underlying heart disease or sympathetic nervous system stimulation because of stress or pain. Some horses have AF with a patterned irregularity that must be distinguished from second degree AVB. Although AF often sounds more regular at higher HRs, the rhythm remains irregular and careful prolonged auscultation will reveal this. Horses with AF also should be examined carefully for murmurs of valvular regurgitation that may predispose to atrial enlargement and remodeling, creating a substrate for AF.

**Diagnosis and Evaluation** The diagnosis is confirmed with an ECG and is characterized by an irregularly irregular R-R interval with normal QRS morphology, the absence of P waves and the presence of “f” waves. Atrial flutter represents a slow macro-reentry variation on AF. Flutter waves resemble saw-toothed P waves and have a regular rate of about 170-275/min, while fibrillation waves are less organized and faster (275-500/minute on intracardiac electrogram; its rate cannot be derived from surface ECG). AV conduction in atrial flutter is usually variable, resulting in a ventricular rate response that can be irregular or regular during periods of increased sympathetic tone. Patterns of 3:1, 2:1 or 1:1 atrial-to-ventricular conduction may be observed. With atrial fibrillation, the R-R interval is irregular. Concurrent PVCs may be found.

Ideally, a *complete echocardiogram* should be performed to identify any underlying structural heart disease, valvular regurgitation, and cardiac (atrial) enlargement as described above. It should be noted that slight LA enlargement can result from AF, even in the absence of MR. Additionally, an *ECG exercise test* should always be performed when a horse is used for performance and cardioversion is not an option.

The suspected *duration of AF* should be determined when possible because it affects the prognosis for successful conversion and the likelihood of recurrence. AF induces time-dependent electrical and structural remodeling within the atria, factors known to promote its persistence. These changes may also decrease the chance of successful cardioversion and...
increase the risk of recurrent or persistent AF, even after successful treatment. Additionally, atrial disease may be associated with recurrent PACs that can act as triggers for recurrent AF after successful treatment. A sudden change in performance and results of previous veterinary examinations provide the best estimate for the onset of the AF. When there is no history of poor performance and no recent information from an examination, it should be assumed that the AF is long-standing. While many horses with AF associated with LA enlargement can be successfully cardioverted to NSR, the likelihood of a successful cardioversion is reduced and the chance for recurrence increases with persistent LA dilatation. Horses with AF secondary to CHF or with PHT have a grave prognosis and should be retired.

Evidence of LVD, typically a depressed shortening fraction, may indicate underlying myocardial disease in the horse with AF, but assessment of LV function is difficult during AF because of secondary ventricular dyssynchrony, tachycardia-induced LV dysfunction and preload and heart rate dependence of many of the 2D, M-mode and Doppler derived indices to assess left ventricular function. If horses fail to return to their previous level of performance after AF has been successfully corrected, persistent LVD should be suspected.

The level of intended activity influences clinical decision making and some horses with persistent AF are able to perform successfully when used for less intense athletic work. Cardioversion is recommended when the average maximal HR during exercise with AF exceeds 220/minute. Additionally, ventricular ectopy during exercise or during sympathetic nervous system stimulation indicates a possible risk for sudden death, particularly when short coupling intervals (R-on-T phenomenon) are observed. For this reason, treatment for AF is also recommended when concurrent ventricular arrhythmias are observed; these usually resolve after cardioversion.

Management strategies for AF include no treatment, pharmacologic cardioversion, transvenous electrical cardioversion (TVEC), and pharmacologic control of ventricular response rate. Details have been described elsewhere. Horses with CHF and AF should be treated for CHF and are not candidates for cardioversion.

Cardioversion is generally not performed for the first 24-48 hours of a documented, recent onset of AF because of the possibility that spontaneous cardioversion might occur. However, even with spontaneous conversion to NSR, an evaluation should be done, including serum K⁺, Mg²⁺ and Ca²⁺, fractional excretion of K⁺ (in racehorses), Echo, continuous 24-hour ECG, and optimally an exercising ECG test to identify atrial triggers or other arrhythmias. These tests are also appropriate in a horse with NSR if paroxysmal AF is suspected from the clinical history. However, if AF persists beyond 48 hours, prompt treatment of AF is recommended to deter progressive atrial remodeling.

Cardioversion to NSR  Cardioversion is desirable in all horses performing rigorous athletic work. Successful treatment allows a return to the previous level of performance, assuming an absence of significant underlying cardiac disease. Cardioversion of AF should only be performed in a controlled setting with continuous (ECG) monitoring, regardless of the treatment method. There are no prospective, randomized studies directly comparing the efficacy of quinidine to transvenous cardioversion (TVEC). The success rate depends on the patient population and success rates of 65-90% have been reported for both. Young racehorses with lone AF have the best prognosis for successful cardioversion, independent of treatment modality. Horses with advanced valvular heart disease and moderate to severe atrial enlargement are poor candidates for cardioversion and long-term maintenance of normal sinus rhythm.

Quinidine sulfate is the mainstay of pharmacological cardioversion of AF. Although
other drugs have been used with varying success to convert AF, these are not first line choices. Indications for quinidine therapy include lone AF, AF with mild LA enlargement, and comorbidities in which general anesthesia or TVEC are not options. Relative or absolute contraindications to quinidine cardioversion include rapid ventricular response to AF and complex ventricular ectopy owing to the potential for proarrhythmia and polymorphic VT. Quinidine also carries the risk for adverse drug effects that may necessitate close monitoring or discontinuation of therapy. The reader is directed elsewhere for details.  

Transvenous electrical cardioversion (TVEC) involves a timed shock delivery on the R-wave. The procedure should be performed by experienced operators. Specialized equipment is needed, including a biphasic defibrillator/cardioversion system and custom-made equine cardioversion catheters. TVEC can be used to treat lone AF, AF with mild LA enlargement, and horses either intolerant of, unresponsive to quinidine therapy or in which quinidine is contraindicated (see above). The risks of TVEC include general anesthesia or rarely, development of a fatal arrhythmia. The immediate recurrence of AF (IRAF) within the first 24 hours after cardioversion, although infrequent, is more likely than with quinidine cardioversion. Pretreatment with antiarrhythmic drugs prior to TVEC or administration of an antiarrhythmic drug during and after anesthesia may minimize the likelihood of IRAF. In the long term, recurrence rates following TVEC and quinidine cardioversion are believed to be similar. The reader is referred elsewhere for a more detailed description of the procedure.

Heart Rate Control
Drugs to control excessively high HR during exercise can be considered as a final option for management of persistent AF, when cardioversion is not an option. but most panelists recommended that if ventricular rate response cannot be controlled, the horse should be retired. If pharmacologic control of an excessively high HR during exercise is to be tried, it should only be performed under the supervision of a specialist experienced in equine cardiology. The efficacy and safety of this approach to control HR in AF in performance horses has not been evaluated critically. If attempted, repeated exercising ECGs and therapeutic drug monitoring are essential.

Summary: Post-Treatment Recommendations for Atrial Fibrillation
Continuous 24-hour ECG is recommended by most panelists after cardioversion, but more data are needed to determine the optimal timing and its prognostic value. A complete Echo after cardioversion can evaluate LV and LA mechanical function and reassess heart size and valvular function. LV function should return to normal within three days. Recovery of LA contractile function can occur within a few days or may take several weeks when AF has been long-lasting. Persistent LA contractile dysfunction can be caused by AF-induced atrial remodeling or underlying primary cardiomyopathy and may predict recurrent AF.

Most recommendations to minimize the risk of AF recurrence are based on human studies or experimental animal models. Chronic antiarrhythmic drug therapy potentially effective against atrial arrhythmias includes propafenone, sotalol, flecainide, and phenytoin. While these could be beneficial to horses with frequent atrial ectopy after cardioversion, clinical studies of efficacy and safety are needed. Drugs known to predispose to ectopic impulse formation should be avoided, including: furosemide, supplements containing sodium bicarbonate, and thyroid hormones. Potassium chloride (KCl) supplementation is indicated in most horses racing on furosemide for EIPH or in those with low fractional excretion of potassium.

Exercise recommendations after cardioversion vary according to the type of AF, AF
duration and concurrent cardiac abnormalities. Ideally, rest is enforced until atrial electrical and contractile function has returned to normal or near normal. Horses with paroxysmal AF and short-duration, lone AF can return to training within one week unless LA stunning or post-conversion arrhythmias are detected. Horses with long-standing AF may need a month or longer of rest.31

Sustained AF is likely to limit rigorous athletic work. Occasionally, it also impairs performance at mid to low levels of activity. Horses with sustained AF that have not been converted should only be used by informed adult riders and limited to an exercise level considered relatively safe based on exercising ECG. The use of a HR monitor can enable the rider to monitor rate during exercise and modify the rigor of the work performed. Although very unlikely, collapse during exercise has been reported with AF. AF associated with exercise-induced ventricular arrhythmias resulting in sudden death has been documented in at least one horse.4

Recurrence of AF The recurrence rate of AF is lowest (about 15%) with recent onset (< 1 month) lone AF.32,37 Recurrence rate may be higher in older horses that are more likely to have underlying cardiac disease, especially chronic valvular regurgitation with atrial enlargement. Horses with a high number of PACs or runs of atrial tachycardia are more likely to experience recurrent AF, and these ECG findings should be considered a poor prognostic indicator. Persistent LA mechanical dysfunction is thought to indicate irreversible atrial remodeling and may also represent a poor prognostic sign.

Premature atrial complexes (PACs)
PACs are usually detected during auscultation as premature beats interrupting an otherwise regular rhythm. At times, PACs are difficult to differentiate from marked sinus arrhythmia. The greatest concern about PACs relates to their potential to incite atrial flutter and atrial fibrillation.

The ECG is needed for definitive diagnosis. PACs are characterized by premature atrial activation (P’), usually with changes in normal P-wave morphology. PACs can be conducted with a variable P’-R interval or blocked at the AV node. PACs are easily missed when buried in the ST-T wave. The conducted QRS is generally normal in morphology, but ventricular conduction can be aberrant resulting in wider, taller or bizarre QRS complexes with secondary ST-T changes.

Summary: Recommendations for Premature Atrial Complexes (PACs)
- PACs are an uncommon cause for poor performance
- Horses with occasional PACs that are overdriven during exercise and those with occasional PACs during exercise are considered as safe to ride as their age-matched peers
- Underlying causes should be sought
- The risk for AF should be appreciated

Premature Ventricular Complexes (PVCs) and Ventricular Tachycardia (VT)
Premature ventricular complexes (PVCs) are also usually detected during auscultation and are characterized by premature beats interrupting an otherwise regular rhythm, usually followed by a compensatory pause.

Ventricular tachycardia (VT) is an abnormal rhythm caused by repetitive or linked PVCs.
Auscultation of VT is characterized by a rapid, usually regular rhythm, with variable intensity and often booming heart sounds ("bruit de cannon"). Due to intermittent aortic valve opening, the rhythm may sound irregular on auscultation and an intermittent pulse deficit may be present. Abnormal jugular pulses (cannon waves) are frequently observed.

The ECG is needed for definitive diagnosis. PVCs are characterized by premature ventricular activation without an associated P-wave. The QRS complex is typically wide and bizarre and followed by a large T-wave of opposite polarity. Impulses arising from high in the ventricle (near the bundle of His) may be difficult to distinguish from a junctional (nodal) rhythm. Following one or more PVCs, sinus impulses are usually blocked in the AV node resulting in atrioventricular dissociation. If the ectopic ventricular rate and sinus rate are close, fusion complexes may be observed.

It is important to characterize the morphology, timing, and rate of PVCs and VT because they are believed to affect the risk for hypotension or sudden death from ventricular fibrillation (VF). The distributional patterns can include PVCs that are isolated, haphazard, or in patterns (such as bigeminy), while runs of VT are classified as nonsustained (paroxysmal) or sustained. The morphology of the ventricular complexes is defined as uniform, multiform or polymorphic. Multiform or polymorphic morphologies suggest widespread myocardial disease. The timing of isolated or repetitive PVCs is important to determine because PVCs coinciding with the preceding T wave (R-on-T) may trigger VF. Repetitive ventricular ectopic activity can be classified as couplets, triplets, or runs of VT. Repetitive ventricular rhythms include idioventricular escape rhythms (20-40/minute associated with high-grade or complete AVB), idioventricular tachycardias (or accelerated idioventricular rhythms [AIVR]) at rates slightly higher than the sinus rate (50-80 beats/minute at rest), monomorphic VT, multiform VT, and polymorphic VT (torsades de pointes).

As a general rule, the following are features of “complex” or “malignant” ventricular arrhythmias: multiform QRS morphology, highly-premature or R-on-T timing, repetitive activity (couplets, triplets), paroxysmal or sustained VT (exceeding 120 beats/minute), multiform tachycardia and polymorphic tachycardia.

Occasional monomorphic PVCs overdriven with exercise or only detected in the immediate post-exercise period are not usually a cause for poor performance. A wide range of ventricular arrhythmias have been reported during and immediately following intense exercise in normally performing horses. The significance of these arrhythmias and the relationship to sudden death in equine athletes requires further investigation. PVCs may also occur during exercise and are a cause for concern. Their relationship to poor performance is also uncertain and requires further investigation.

Assessment of the overall clinical picture is important because ventricular arrhythmias can be associated with medical or surgical disorders and often resolve with correction of the underlying problem. A clinical laboratory profile, including cTnI should be obtained from all horses. An echocardiogram should be performed if cTnI is significantly elevated, a murmur is detected or ventricular arrhythmias persist. The Echo should include imaging for abnormal myocardial echo texture, thickness, or scar, and exclusion of dissecting aortic aneurysm or aortocardiac fistula. LVD may be secondary to tachycardia-induced cardiomyopathy or ventricular dyssynchrony. In horses with sustained VT the Echo should be repeated once the horse has returned to NSR. Further work-up of a horse with PVCs or AIVR, in the absence of underlying systemic disease, should include an exercising ECG. Horses with severe ventricular arrhythmias should not be exercise tested.
Summary: Recommendations for Premature Ventricular Complexes and Ventricular Tachycardia

- Underlying causes should be sought and managed if possible
- Horses with occasional PVCs, with sustained AIVR that is overdriven by exercise or multiple PVCs during exercise may be used with caution by an informed adult rider. Owing to ongoing concerns about underlying myocardial or electrical disease and increased risks of exercise associated collapse and sudden death, these horses should not be used by a child rider or as a lesson horse.
- Horses with sustained monomorphic VT should be rested and treated. NSR should be present for at least 4 weeks before re-evaluation is performed. A continuous 24-hour ECG is indicated prior to returning the horse to work. If normal, an exercising ECG should be performed, followed by another exercising ECG once the horse has returned to full work. Horses affected by a single episode generally have a favorable prognosis, but on occasion monomorphic VT can recur.
- Horses with complex or malignant ventricular arrhythmias should be rested and treated. A 24-hour ECG is indicated before returning the horse to work. Once NSR has resumed, an exercising ECG should be performed, although the safety of these horses remains uncertain. These horses should only be ridden by an informed adult rider.
- Rigorous athletic work is not recommended for horses that showed VT in the setting of structural heart disease, including focal myocardial fibrosis. These horses should only be used by an informed adult rider due to the risk of possible recurrence of VT. These horses are not safe for use by a child rider or as a lesson horse.
- For horses that remain in work with a history of VT follow up 24-hour and exercising ECGs should be performed at least annually.

Areas for Future Research Investigation

The panel has identified some areas for future research including further investigations into the pathophysiology of mitral, tricuspid and aortic regurgitation and the development of a comprehensive assessment to determine the severity of valvular regurgitation. Large multicenter clinical studies to better understand the progression and outcome of horses with valvular regurgitation and VSDs are indicated. Long-term studies evaluating the effect of ACE inhibitors in horses with moderate to severe mitral and aortic regurgitation are needed. Critical assessments of the efficacy of different treatments for AF need to be performed with the selection criteria clarified for each treatment. Large multicenter trials to study the factors affecting recurrence of AF in a varied AF patient population are indicated. Large studies in different populations of performance horses are needed to further study the arrhythmias present during exercise and their relationship to horse and rider safety and the occurrence of sudden cardiac death. The reproducibility of continuous 24-hour ECG monitoring and exercising ECGs also needs to be determined. Collaboration between different investigators on these and other studies will help to further advance the knowledge of cardiac disease in the equine athlete and the development of appropriate recommendations.
Selected References