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Recommendations for Management of Equine Athletes with Cardiovascular Abnormalities

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Murmurs and arrhythmias are commonly detected in equine athletes. Assessing the relevance of these cardiovascular abnormalities in the performance horse can be challenging. Determining the impact of a cardiovascular disorder on performance, life expectancy, horse and rider or driver safety relative to the owner's future expectations is paramount. A comprehensive assessment of the cardiovascular abnormality detected is essential to determine its severity and achieve these aims. 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 are considered for each cardiovascular abnormality. The recommendations presented herein are based on available literature and a consensus of the panelists. While the majority of horses with cardiovascular abnormalities have a useful performance life, periodic reexaminations are indicated for those with clinically relevant cardiovascular disease. Horses with pulmonary hypertension, CHF, or complex ventricular arrhythmias should not be ridden or driven.

Key words: Arrhythmias; Echocardiography; Exercise testing; Murmurs.

Cardiac murmurs and arrhythmias are often identified in horses engaged in 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 present and future performance, on rider or driver and horse safety, and to consider any long-term effects on health and longevity.

The recommendations contained herein were developed during recurrent and protracted discussions with active participation by all 7 panelists. The available lit-

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Abbreviations:

AF	atrial fibrillation
AIVR	accelerated idioventricular rhythm
AR	aortic regurgitation
AVB	atrioventricular block
AVP	aortic valve prolapse
CHF	congestive heart failure
cTnI	cardiac troponin I
CV	cardiovascular
ECG	electrocardiogram
HR	heart rate
IRAF	immediate recurrence of AF
LA	left atrium or left atrial
LVD	left ventricular dysfunction
LV	left ventricle or left ventricular
MR	mitral regurgitation
MVP	mitral valve prolapse
NSR	normal sinus rhythm
PAC	premature atrial complex
PHT	pulmonary hypertension
PVC	premature ventricular complex
RA	right atrium or right atrial
RCT	ruptured chordae tendineae
RV	right ventricle or right ventricular
SCD	sudden cardiac death
SNS	sympathetic nervous system
TR	tricuspid regurgitation
TVEC	transvenous electrical cardioversion
VA	ventricular arrhythmias
VSD	ventricular septal defect
VT	ventricular tachycardia

erature (including high-grade research evidence, retrospective studies, and case reports) was used whenever possible. However, since pertinent literature in many important areas is quite limited, 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.

General Approach

These recommendations for equine athletes presume familiarity and clinical expertise in equine auscultation, echocardiography, electrocardiography (ECG), exercise testing, and interpretation of laboratory testing, along with a thorough understanding of the pathophysiology of equine heart disease.[1–4] 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.

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 a lesion of "moderate" severity that might be 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 progressive chamber remodeling (dilatation and altered chamber shape) and dysfunction, great vessel enlargement, as well as the development of pulmonary hypertension (PHT), congestive heart failure (CHF), and potentially dangerous arrhythmias. Horses with PHT, CHF, or complex ventricular arrhythmias (VA) are unsafe to ride or drive and should be managed with standard therapy for these problems.

Echocardiography

The history, cardiac rhythm, and the results of auscultation should be known before interpreting the echocardiogram. The Panel specifically recommends an echocardiogram in the following situations: (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) clinically important arrhythmias, whether a murmur is present or not; (7) suspected myocardial injury; or (8) suspicion of CHF. However, there are other clinical situations when echocardiography also provides valuable information.

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) estimates of hemodynamic variables including pressure gradients and volumetric flow; and (8) ventricular diastolic function and filling pressures (accepting these two are challenging to measure in mature horses). A comprehensive assessment requires the application of complementary 2D, M-mode, and Doppler modalities.

Exercise Testing

Exercise testing is an important component of CV assessment and includes continuous recording of the ECG. Exercising ECGs should only be obtained with a device that has permanent storage and playback capabilities.[4–6] Work intensity should be at or slightly exceeding the horse's customary activities. 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. In horses with clinically important 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 nonfunctional 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 severely reduced systolic function or when a ventricular arrhythmia of dangerous complexity is present.

Specific noninvasive cardiac assessments include (1) the effects of exercise on auscultation (rate, rhythm, and murmurs); (2) peak HR during exercise; (3) HR and rhythm during the different phases of the exercise test and during recovery; and, optionally, (4) echocardiography before and after exercise (stress echocardiogram). Additional tests that might be indicated are analysis of gait, airway dynamics, arterial blood gas tensions, and other clinical laboratory tests.

Cardiac Murmurs

Both functional and clinically relevant cardiac murmurs are common in horses (Table 1). Mild valvular regurgitation is often detected in horses with soft

Table 1. Cardiac murmurs.

Left/Right Side Thorax	Diagnosis	Typical Auscultatory Findings	Comments
Left side thorax			
Systolic murmur	Physiologic (flow) murmur	PMI over aortic and pulmonic valve area Early-to-midsystolic Crescendo-decrescendo or decrescendo Usually grade 1–3/6	Assumed to be caused by blood flow in aorta and pulmonary artery in early systole Generally localized and brief in duration Intensity might change with exercise
	Mitral regurgitation	PMI over mitral to aortic valve Holo- or pansystolic or mid-to-late systolic Plateau (band-) shaped or mid-to-late crescendo Grade 1–6/6	Crescendo mid-to-late systolic murmur suggestive of mitral valve prolapse Musical holosystolic or mid-to-late systolic honking murmur suggestive of ruptured chordae tendineae
	Ventricular septal defect (subarterial)	PMI pulmonic valve area Plateau (band)-shaped Grade 4–6/6	Often heard on right side but less loud Less common location Must distinguish from right ventricular outflow obstruction
Diastolic murmur	Physiologic (flow) murmur	PMI over mitral & tricuspid valve area Early-diastolic (S2-S3) or late-diastolic (S4-S1) Quality often musical/squeaking Grade 1–3/6	Assumed to be caused by ventricular filling
	Aortic regurgitation	PMI over aortic valve area Holo- or pandiastolic Decrescendo, musical, or both Grade 1–6/6	Usually radiates to the right as a slightly softer, but otherwise similar murmur
Systolic with diastolic component	Aorto-pulmonary fistula (predominantly reported in Friesians)	PMI dorsal to aortic valve area Holosystolic and early-to-middiastolic	Can be fairly localized Bounding arterial pulses Usually tachycardic
Right side thorax			
Systolic murmur	Tricuspid regurgitation	PMI over tricuspid valve Holo- or pansystolic Crescendo or band shaped Grade 1–6/6	Usually soft and blowing
	Ventricular septal defect (perimembranous)	PMI ventral to tricuspid valve Holo- or pansystolic Plateau (band-) shaped Grade 4–6/6	Usually coarse Often a softer crescendo-decrescendo murmur over pulmonic valve heard because of relative pulmonic stenosis
Continuous	Aorto-cardiac fistula	PMI right side, also audible on left Harsh machinery Grade 3–6/6	Bounding arterial pulses Ventricular arrhythmias common initially

murmurs, and seemingly does not affect performance or health.[7] Loudness of the murmur need not correlate with severity, especially with musical murmurs or in the setting of CHF.

A complete echocardiogram is the diagnostic test of choice when evaluating a horse with a cardiac murmur[2,3] and is advised (1) when auscultatory or clinical findings are not consistent with a physiologic murmur; (2) if the murmur is moderate to loud; or (3) on occasion, when a murmur is detected as part of a prepurchase examination. Serial echocardiographic evaluations are more meaningful for prognostication than findings from a single examination. A number of Doppler findings, including the width of the vena contracta and the regurgitant jet area, are used to assess valvular regurgitation,[8] but these variables are unreliable in isolation. For example, in the absence of cardiac chamber enlargement, it is unlikely that chronic, severe regurgitation is present, regardless of the Doppler findings. Furthermore, Doppler studies often reveal physiological valvular regurgitation that is silent on auscultation.

Mitral Regurgitation

MR is a common finding in horses performing in all sports disciplines. Murmurs of MR can be mid-to-late

systolic or holo/pansystolic (Table 1). When MR is mild, it is usually associated with normal performance[7] and life expectancy.[9–12]

Some of the underlying lesions responsible for MR can be visualized by 2D echocardiography, including 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 valve annulus or ventricular dilatation (as with severe AR, nonrestrictive VSD or rarely, dilated cardiomyopathy).

Diagnosis and Evaluation. The mitral valve and support apparatus should be thoroughly examined by 2D and M-mode echocardiography for structural and motion abnormalities and identification of the most likely etiology for MR. Valvular or chordal thickening might 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 left atrium (LA) along with a mid-to-late systolic murmur and Doppler confirmation of MR are findings consistent with MVP. A RCT is detected as an echoic, whip-like structure moving into the LA during systole which flips into and out of the imaging plane.[13] A flail leaflet is diagnosed when part of the valve leaflet is imaged in the LA and moving chaotically or independently from the rest of the mitral valve.[13] Endocarditis often appears as focal-raised lesions on the 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 left ventricular (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.[13] In the absence of pulmonary artery (PA) catheterization, TR velocity and PA dilatation are used as surrogates for the identification of PHT. Assessment of LV systolic function can be confusing because of 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 (RV) with exuberant septal motion also suggests severe LV volume overload. Chronic MR associated with progressive remodeling and left ventricular dysfunction (LVD) can lead to the fractional shortening returning to the normal range or being obviously reduced.

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. A number of color and spectral Doppler methods are used to assess severity of MR in humans[8] and are also used by some panelists in horses. However, the examiner must fully appreciate the limitations of these Doppler techniques.

Prognosis (Table 2). Assessing the severity of MR and predicting its clinical course are difficult. Horses with a mid-to-late crescendo murmur and mild MR usually have a favorable prognosis. 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 PA dilatation, increased TR velocity, or significant MR with AF or tachycardia.[10,13]

Summary: Key Recommendations for MR

- Determine the most likely etiology.
- Assess severity based on combined assessment of performance history, exercise testing, clinical examination, and echocardiographic findings.
- Reexamine at least annually or, for mild MR, every other year.
- Ensure that HR and rhythm are monitored on a regular basis in horses with moderate to severe MR.
- Perform ECG exercise test in (1) all horses with moderate to severe MR; (2) if AF becomes established (see below); or (3) if MR progresses more rapidly than expected, in the absence of signs of CHF.
- 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

AR is a common finding in older horses. The murmur is often detected incidentally.[14] Any left-sided holodiastolic murmur is assumed to indicate AR unless proven otherwise. Degenerative valve thickening and aortic valve prolapse are most frequently detected.[14,15] 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.[12] However, when AR is moderate to severe or first recognized in a younger horse (<10 years of age), the risks for reduced performance life and longevity are higher. SCD associated with fatal VA has been observed in horses with moderate to severe AR and can occur in isolation, without a history of poor performance or CHF.

Diagnosis and Evaluation. Bounding or hyperdynamic arterial pulses suggest hemodynamically severe AR with LV volume overload.[14] Noninvasive blood pressure measurement can identify widening of pulse pressure.[16] An exercising ECG is recommended when moderate to severe AR or performance issues are evident. This evaluation should be focused on identifying exercise-induced premature ventricular complexes

Table 2. Prognostic guidelines for performance of horses affected with mitral and aortic regurgitation.[a]

	Excellent	Fair	Guarded to Poor
Mitral regurgitation			
Arterial pulses	Normal	Normal	Weak
Valve lesion	None, mild MVP	Mild thickening, mild dysplasia	Severe thickening, RCT, flail leaflet, endocarditis, severe dysplasia
LA enlargement	Absent or mild	Mild to moderate	Moderate to severe or progressive
LA shape	Normal	Normal or slightly rounded	Might appear round and turgid
Interatrial septum	Normal	Normal	Might be bulging toward RA
LV volume overload	Absent	Mild	Moderate to severe
LV systolic function	Normal	Normal	Hyperdynamic, normal (less than expected) or decreased
Pulmonary vein enlargement	Absent	Absent	Usually present
PA enlargement	Absent	Absent	Present or developing
MR jet[b]	One or multiple small & narrow jets	One or more medium-sized jets	Large single or large multiple jets
AF, PACs	Absent	Pre-existing	Secondary
Aortic regurgitation			
Arterial pulses	Normal	Normal or slightly bounding	Bounding or weak
Arterial blood pressure	Normal		Pulse pressure >60 mmHg
Valve lesion	None, parallel fibrous band, mild AVP	Nodular thickening, moderate AVP suspected fenestration	Severe thickening or AVP, flail leaflet, endocarditis, congenital malformation
Aortic root	Normal	Normal or mild enlargement	Mild to severe enlargement
LV volume overload	Absent or mild	Mild to moderate	Moderate to severe
LV systolic function	Normal	Hyperdynamic	Hyperdynamic or decreased
AR jet[b]	One or 2 small & narrow jets Long pressure half-time[c]	One or more medium-sized jets	Large single or large multiple jets Short pressure half-time[c]
LA enlargement	Absent	Absent or mild	Mild to severe
Concurrent MR	Absent	Absent or pre-existing	Secondary to AR
PA enlargement	Absent	Absent	Present
Ventricular arrhythmias	Absent	Absent	PVCs, VT
AF	Absent	Pre-existing	Secondary
Age at onset	Older	Middle age	Young

[a]Combined assessments are essential in accurately formulating a prognosis for life and performance and should include lesions detected, size of cardiac chambers, myocardial function, color Doppler assessment of jet, Doppler (CW)/hemodynamic estimates, age at onset, and intended use of horse.

[b]"Jet" includes both the width of the regurgitant flow at the valve orifice (at the vena contracta) and the jet area relative to the receiving chamber area, in which case the "jet" likely includes entrained RBCs. Width of the jet at the valve orifice is difficult to measure accurately because of dynamic, nonuniform, three-dimensional structure at the vena contracta. Jet area is highly dependent on technical factors including ultrasound beam angle, transducer distance from the region of interest, transducer frequency, 2D and color gain and filter settings, pulse-repetition frequency, and image plane.

[c]Pressure half-time cannot be accurately measured unless the interrogating beam is maintained at a constant angle with the regurgitant jet.

AF, atrial fibrillation; AR, aortic regurgitation; AVP, aortic valve prolapse; LA, left atrial; LV, left ventricular; MR, mitral regurgitation; MVP, mitral valve prolapse; PA, pulmonary artery; PAC, premature atrial complex; PVC; premature ventricular complex; RA, right atrium; RCT, ruptured chordae tendineae; VT, ventricular tachycardia.

(PVCs) and appropriateness of the exercising HR. A continuous 24 hour-Holter ECG can be considered to further identify and quantitate any VA.

2D echocardiography frequently demonstrates valve thickening as a fibrous band-like lesion, which appears usually as an echoic line parallel to the free edge of the left coronary leaflet.[14] Less often, nodular thickening or a generalized increase in echogenicity of the leaflet's free edge is detected. Leaflet prolapse into the LV outflow tract, especially involving the noncoronary cusp, is another common finding. False positive diagnoses are common with misalignment of the ultrasound beam.[15] 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 are an uncommon cause of AR and can be evidenced as raised lesions, an oscillating thrombus, or mild to severe thickenings depending on the severity and chronicity of disease.

M-mode studies demonstrate diastolic fluttering of the mitral[14] or aortic valves, aortic root, or interventricular

septum should the jet of AR impinge one of these structures. Eccentric jets directed toward the mitral valve can prevent full opening of that leaflet. Premature closure of the mitral valves on M-mode echocardiography 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 can occur with long-standing moderate to severe AR.[14] LA dilatation suggests ventricular dysfunction, volume retention, or concurrent MR. The latter can develop because of 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 might be observed. CW Doppler studies including regurgitant signal duration and intensity, pressure half-time, and velocity time integral of AR compared to forward flow are used in humans[8] and could have merit in horses.

Prognosis (Table 2). Assessing AR severity and predicting its clinical course are challenging. Findings compatible with mild AR that are unchanged at follow-up examinations relate to a better prognosis for work and life. The detection of hyperkinetic arterial pulses or a pulse pressure of >60 mmHg suggests that progression of AR is likely.[16] All panelists agreed that horses with severe AR should not be ridden or driven by a child, used as a lesson horse, or participate in a high-risk sport owing to a risk for SCD. 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: Key Recommendations for AR

- Determine the most likely etiology.
- Assess severity based on combined assessment of performance history, exercise testing, clinical examination, and echocardiographic findings.
- An exercise ECG is indicated in a horse with moderate to severe AR.
- Reexamine twice yearly when there is moderate to severe AR (including echocardiography and ECG exercise test) and at least annually thereafter if progression has been minimal. Similarly, longer follow-up intervals are appropriate for horses with mild AR after the first re-evaluation.
- If AF develops in a horse with mild to moderate AR, a reexamination is indicated at that time, including an ECG exercise test.
- 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 indicates progression.
- The detection of exercise-induced VA is considered an important negative prognostic indicator.
- Horses with AR and PVCs during exercise are considered less safe to ride or drive than their age-matched peers.

- 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

TR is a common finding in equine athletes.[7,17] The prevalence and severity of TR appear to be influenced by both age and level of training.[7,18,19] The precise mechanisms of training-associated TR are unknown. Horses with severe MR and PHT associated with CHF as well as (less commonly) horses with severe respiratory disease can develop TR. Abnormalities such as leaflet thickening or RCT are infrequently detected on 2D echocardiography. Endocarditis infrequently affects the TV and is typically secondary to septic jugular thrombophlebitis.[20]

Diagnosis and Evaluation. A Doppler echocardiogram confirms TR but is mainly indicated with a grade 4/6 or louder murmur in the setting of poor performance, concurrent thrombophlebitis, or with fever of unknown origin. An echocardiogram also could be requested in the setting of a prepurchase examination.

With benign or training-related TR, the valve is structurally normal, the right atrium (RA) and RV are normal in size, and the regurgitant jet is usually thin and directed toward the aorta. With clinically significant TR, there might 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 toward the lateral RA wall. When the TR jet velocity is >3.5 m/s, PHT is suspected, in which case the horse should be scrutinized for left-sided heart disease or severe pulmonary disease.[21] TR with RA enlargement can predispose to atrial flutter, AF or CHF, although less often than for MR.

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

Summary: Key Recommendations for TR

- Appreciate the high prevalence of training-induced TR in high-performance horses.
- Perform comprehensive clinical and echocardiographic examinations when indicated (see above).
- An annual echocardiogram 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,[22] Standardbreds,[23] and Arabian horses[23,24]). 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 noncoro-

nary cusps of the aortic valve.[23] 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 could differ from those described in Table 1.

Diagnosis and Evaluation. Definitive diagnosis requires an echocardiogram. Complex congenital disease must be excluded.[23,25,26] The size of the VSD is often underestimated with 2D echocardiography and overestimated with color-flow Doppler echocardiography. 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 important.[23]

There is usually some enlargement of the left side of the heart with a left-to-right shunt; however, cardiac dimensions usually fall within the normal range if the shunt is small. Moderate to severe enlargement of the LA and LV is concerning and increases the risk for AF, PHT, and CHF. Noticeable systolic enlargement of the pulmonary artery is an indication of pulmonary overcirculation. Marked pulmonary artery enlargement during both systole and diastole suggests PHT.

Provided that there is 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 a better prognosis.[23] 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 shunt velocities (<4 *m/s*), along with higher pulmonary artery ejection and mitral inflow velocities, indicate a greater shunt volume.

It is important to identify aortic malalignment, aortic valve prolapse into the VSD with AR, and to detect MR, TR, or other comorbidities. If moderate to severe right ventricular wall hypertrophy is found, a large shunt, PHT, or complex congenital disease should be assumed.

Prognosis. The most important prognostic criteria for an isolated VSD 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. Moderate defects are often well-tolerated at rest; although performance in high-intensity sports might be affected. Progressive MR or AR and cardiac remodeling can facilitate the development of AF, PHT, or LVD with progressively negative impact on exercise capacity and eventually on survival. The finding of a large diameter, unrestrictive VSD portends a poor prognosis and shortened life expectancy.

Summary: Key Recommendations for VSD

- Perform comprehensive clinical and echocardiographic examinations.
- Reexamine 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 and Aorto-Pulmonary 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.[27,28] Many horses present with an acute onset of exercise intolerance and pain, often perceived as colic, and ventricular tachycardia (VT).[27,28]

Friesian horses with aorto-pulmonary fistulation present with bounding arterial pulses, tachycardia, and a grade 1–3/6 holosystolic and early-to-mid diastolic murmur loudest dorsal to the aortic valve.[29] Although rare, it often results in acute heart failure or even SCD, but might also present with a chronic history of poor performance.

Summary: Key Recommendations for Aorto-cardiac and Aorto-pulmonary Fistula

- Consider aorto-cardiac fistula in horses with acute colic and VT, or when a continuous right-sided murmur is detected.
- Affected horses are not safe to use.
- These horses can experience SCD at any time.

Cardiac Arrhythmias

Arrhythmias can develop as isolated electrical disorders (eg, lone AF or complete heart block) or secondary to other etiologic 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/envenomations; 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,[30] or transesophageal leads[31] are needed to better evaluate the ECG waveforms in some cases. A portable ECG unit is useful in the field for documenting arrhythmias. A continuous 24-hour Holter ECG is indicated to characterize intermittent arrhythmias. An exercising ECG is often indicated to determine if an arrhythmia

has potential for impairing performance or might become a safety issue.

The workup of the horse with a nonphysiologic arrhythmia should also include (1) a history including all drugs and supplements administered; (2) a complete echocardiogram; and (3) appropriate laboratory tests.

The examiner must be familiar with physiologic, vagally mediated arrhythmias that include sinus arrhythmia, second degree AVB, and sinoatrial bock. These are normal at rest and immediately after exercise.

Second Degree Atrioventricular Block (AVB)

This rhythm is normal in equine athletes. The HR is in the low normal range and there are usually several conducted P waves before the AVB. Auscultation is characterized by an irregular rhythm with a repetitive pattern. Fourth (atrial) heart sounds that are not followed by first and second heart sounds might be audible in the regular pauses in some horses. Physical activity or increased sympathetic tone should cause the arrhythmia to disappear, although AVB is likely to resume 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 more than 2 consecutively blocked P waves, 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 to determine if a 1 : 1 conduction develops. A continuous 24-hour Holter ECG with simultaneous video recording should be obtained when there is a history of collapse.

Summary: Key Recommendations for High-Grade Second Degree AVB

- Horses with high-grade second degree AVB that disappears with exercise should only be ridden or driven by an informed adult, and 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 considered less safe to ride or drive than their age-matched peers.
- Horses with symptomatic bradyarrhythmias generally have a poor prognosis and are not safe to ride or drive.

Atrial Fibrillation

Atrial fibrillation (AF) is the most common arrhythmia affecting performance. In some Standardbred racehorses, it has been identified as a heritable lesion.[32] 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 might be present in some of these horses, but cannot be detected using routine diagnostic tests. Structural heart diseases can predispose horses to recurrent or persistent 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, sympathetic nervous system (SNS) stimulation because of stress or pain, or in unusual cases, presence of an accessory atrioventricular conduction pathway. Some horses have AF with a patterned AV conduction sequence that must be distinguished from second degree AVB. Although AF often sounds more regular at higher HRs, the rhythm remains irregular and careful auscultation will reveal this. Horses with AF also should be examined critically for relevant murmurs that might indicate the presence of atrial enlargement and remodeling, creating a substrate for AF.

Diagnosis and Evaluation. The diagnosis is confirmed with an ECG, which is characterized by an irregularly irregular R-R interval with normal QRS morphology, the absence of P waves and the presence of "f" waves. Concurrent PVCs might be found. Atrial flutter represents a slow macro-reentry variation on AF. Flutter waves resemble saw-toothed P waves without an isoelectric shelf and have a regular atrial rate of about 170–275/min, while fibrillation waves are less organized and faster (275–500/min on intracardiac electrograms). 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 can be observed.

A complete echocardiogram should be performed to identify any underlying structural heart disease, valvular regurgitation, and cardiac (atrial) enlargement as described above. A slight increase in LA size 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 or could not be attained.[33]

The suspected duration of AF should be determined when possible because it affects the prognosis for successful conversion and the likelihood of recurrence.[34] AF induces time-dependent electrical and structural remodeling within the atria, factors known to promote its persistence.[30,35–37] These changes might also decrease the chance of successful cardioversion and increase the risk of recurrent or persistent AF, even after successful treatment.[30] Additionally, atrial disease might be associated with recurrent premature atrial complexes (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 this information is lacking, it should be assumed that AF is long-standing. Significant LA

enlargement reduces the likelihood of successful cardioversion and increases the risk for recurrence. Horses with AF secondary to CHF or with PHT have a grave prognosis and should be retired.

Evidence of LVD, typically a decreased shortening fraction, might indicate underlying myocardial disease in the horse with AF.[38] However, assessment of LV function is hampered during AF because of ventricular dyssynchrony, tachycardia-induced LV dysfunction and preload and HR dependence of many of the echocardiographic indices used to assess LV function. If horses fail to return to their previous level of performance after AF has been successfully corrected, then persistent LVD should be suspected.

The level of intended activity influences clinical decision making as sustained AF is likely to limit rigorous athletic work and occasionally impairs performance at mid to low levels of activity. Other horses with persistent AF are able to perform successfully when used for less intense athletic work. However, cardioversion of AF is recommended when the average maximal HR during exercise at an intensity that is at or slightly exceeding the horse's normal activities is greater than 220/min. Although uncommon, collapse during exercise has been reported with AF. Additionally, ventricular ectopy during exercise or during SNS stimulation indicates a possible risk for SCD, particularly when short R-R intervals or R-on-T phenomenon are observed. AF associated with exercise-induced VA resulting in SCD has been documented in at least one horse.[4] For this reason, treatment for AF is also recommended when concurrent VA are observed.[33] These usually resolve after cardioversion.

Management strategies for AF include no treatment, pharmacologic cardioversion, and transvenous electrical cardioversion (TVEC). Details have been described elsewhere.[38–49] 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 spontaneous cardioversion might occur. However, after spontaneous cardioversion, an evaluation is still indicated, including measurement of serum K^+, and Mg^{++}, fractional excretion of K^+ (in racehorses), echocardiography, 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 clinically relevant 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

TVEC. Success rates of 65–90% have been reported for both. Young racehorses with lone AF probably have a better prognosis for successful cardioversion, independent of treatment modality. Horses with advanced valvular heart disease and moderate to severe atrial enlargement are poorer candidates for cardioversion and long-term maintenance of NSR.

Quinidine sulfate is the mainstay of pharmacologic cardioversion of AF.[34] Indications for quinidine treatment 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 proarrhythmic effect of quinidine and risk of polymorphic VT. Quinidine also carries the risk for adverse drug effects that necessitates close monitoring, and sometimes discontinuation of treatment or coadministration of other drugs (such as digoxin) to control the ventricular response rate. The reader is directed elsewhere for details.[34,38,43,44]

TVEC involves a timed shock delivery on the R-wave. The procedure should be performed by experienced operators using specialized equipment. TVEC can be used to treat lone AF, AF with mild LA enlargement, and horses either intolerant of or unresponsive to quinidine treatment or horses in which quinidine is contraindicated (see above).[39] The risks of TVEC include general anesthesia[50,51] or rarely, development of a fatal arrhythmia.[52] 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 before TVEC or administration of an antiarrhythmic drug during and after anesthesia might minimize the likelihood of IRAF.[45,53] In the long term, recurrence rates after TVEC and quinidine cardioversion are believed to be similar. The reader is referred elsewhere for a more detailed description of the procedure.[39–42,45,54]

PostCardioversion. Recurrent atrial arrhythmias can be observed in some horses following successful cardioversion and these are best identified using a continuous 24-hour ECG. However, the optimal timing of this examination, the influence of premature atrial complexes on long-term prognosis, and the best approach to management of recurrent atrial ectopy are unknown. A complete echocardiogram after cardioversion can evaluate LV and LA mechanical function and reassess heart size and valvular function. LV function should return to normal within 3 days.[38] Recovery of LA contractile function can occur within a few days or might take several weeks when AF has been long-lasting.[53,55,56] Persistent LA contractile dysfunction can be caused by AF-induced atrial remodeling or underlying primary cardiomyopathy and might portend recurrent AF.

Most recommendations to minimize the risk of AF recurrence are based on human studies or experimental animal models. Chronic antiarrhythmic drug treatment potentially effective against atrial arrhythmias in

human patients includes propafenone, sotalol, flecainide, amiodarone, 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 supplementation is indicated in most horses administered furosemide before racing or in those with low fractional urinary excretion of potassium.

The type of AF, AF duration, and concurrent cardiac abnormalities help determine the time out of training following cardioversion. Ideally, rest is enforced until atrial electrical and contractile function has normalized or nearly so. Horses with paroxysmal AF and short-duration, lone AF can return to training within 1 week unless LA stunning or postconversion arrhythmias are detected. Horses with long-standing AF might need a month or longer of rest.[53]

Recurrence of AF. The recurrence rate of AF is lowest (about 15%) with lone AF of recent onset (≤1 month).[34,39] Recurrence rate of AF is higher in horses with 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 might also represent a poor prognostic sign.

Summary: Key Recommendations for AF

- Select the most appropriate method of cardioversion based on the risk factors for pharmacologic cardioversion or TVEC.
- Perform a continuous 24-hour ECG and ideally evaluate LA function after cardioversion.
- Return to training within 1 week with paroxysmal AF or short-duration lone AF if normal after cardioversion; rest horses with long-standing AF for 4–6 weeks.
- Avoid the use of furosemide, supplements containing sodium bicarbonate, and thyroid hormones after cardioversion.
- Supplement oral potassium chloride to horses administered furosemide before racing or demonstrating low fractional excretion of potassium.
- Because safety is a concern with persistent AF, the horse should be cardioverted or retired when the exercising HR during sustained maximal exercise exceeds 220 beats/min or if concurrent VA are detected during exercise or with SNS stimulation.
- Horses with persistent AF should only be ridden or driven by an informed adult and limited to an exercise level considered relatively safe based on an exercising ECG. The use of a HR monitor might be useful to track heart rate during exercise and modify the rigor of the work performed.

Premature Atrial Complexes

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. PACs are an uncommon cause for poor performance. The greatest concern about PACs relates to their potential to incite atrial flutter and AF.

The ECG is needed for definitive diagnosis, and continuous ECG monitoring enables the clinician to more completely characterize the atrial ectopy. PACs are characterized by ectopic, 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 segment or T wave, especially at higher HR. 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 segment and T wave changes.

Summary: Key Recommendations for PACs

- A continuous 24-hour ECG is recommended to assess frequent PACs.
- Horses with occasional PACs that are overdriven during exercise and those with occasional PACs during exercise are considered as safe to ride or drive as their age-matched peers.
- Underlying causes should be sought.
- The risk for AF should be appreciated.

Premature Ventricular Complexes and Ventricular Tachycardia

PVCs are also usually detected during auscultation and are characterized by premature beats interrupting an otherwise regular rhythm, usually followed by a compensatory pause. VT is an abnormal rhythm caused by 3 or more 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"). Because of intermittent aortic valve opening, the rhythm can sound irregular on auscultation and an intermittent pulse deficit could be present. Abnormal jugular pulses (cannon waves) are frequently observed.

An 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) can be difficult to distinguish from a junctional (nodal) rhythm.

Defining the safety risks to the horse and to the rider or driver is paramount in cases of ventricular ectopy. The complexity of a ventricular arrhythmia (see below) is presumed to relate to the risk of hypotension and sudden cardiac death (SCD) because of ventricular fibrillation (VF). However, risk stratifica-

tion for VA is imperfect. In the absence of clear evidence, the panel believes recommendations should be biased toward safety, as opposed to maintaining athletic activity. A history of collapse raises great concern in a horse with PVCs. Similarly ventricular ectopy in association with important structural heart disease (and cardiomegaly) poses another long-term safety concern (as discussed above, exercise ECGs are generally recommended for those horses). Detection of systemic hypotension during a documented run of VT is another indication of a serious arrhythmia. But in the absence of clinical signs or of serious structural heart disease, the risk of ventricular ectopy is usually defined by electrocardiographic characteristics, accepting the limitations of this analysis. This assessment includes the morphology, timing, and rate of the ectopic activity.

PVCs can be characterized by QRS complex morphology, including uniform or multiform appearance; number (singles, couplets, or VT); average frequency (per hour or per 24 hours); distributional pattern (eg, haphazard, bigeminy, interpolated); coupling interval relative to the previous QRS complex (fixed versus variable; short coupling versus late-diastolic); and prematurity or timing relative to the previous QRS-T (as with 'R-on-T' complexes). Runs of VT are further classified by their rate (absolute rate/min and rate relative to the sinus node); duration of VT (nonsustained versus sustained), and morphology (uniform versus polymorphic, including torsades de pointes). As a general rule, the following are features of complex or potentially "malignant" VA: multiform or polymorphic QRS morphology; short coupling intervals (especially R-on-T timing); sustained VT; rapid ventricular rate (exceeding 120 beats/min); and repetitive ectopic activity (couplets, VT). Polymorphic VT can be observed with diffuse myocardial disease or with drug toxicity (quinidine toxicosis) and induces both hemodynamic and electrical instability. Conversely, the well-tolerated accelerated idioventricular rhythm (AIVR) tends to be monomorphic, start with a relatively long coupling interval, and become established at relatively slow ventricular rates (50–80/min at rest).

Occasional monomorphic PVCs overdriven with exercise or only detected in the immediate postexercise period are not usually a cause for poor performance. A wide range of VA occur during and immediately after intense exercise in normally performing horses.[57–60] The importance of these arrhythmias and the risk of SCD requires further investigation. PVCs can also occur during exercise and are a cause for concern. Their relationship with poor performance is also uncertain and requires further investigation.

Assessment of the overall clinical picture is important because VA can be associated with medical or surgical disorders and often resolve with correction of the underlying problem. A clinical laboratory profile, including plasma or serum cardiac troponin I (cTnI) concentration should be obtained from affected horses. While an echocardiogram might be valuable in any horse with VA, this test is specifically recommended for horses with VT or complex VA; when VA is recurrent or persistent; or when VA is identified in the clinical settings of poor performance, collapse, a clinically relevant cardiac murmur, or a moderately to severely increased cTnI. The echocardiogram should include imaging for abnormal myocardial echo texture, thickness, or scar, and exclusion of dissecting aortic aneurysm or aorto-cardiac fistula. LVD might be secondary to tachycardia-induced cardiomyopathy or ventricular dyssynchrony. In horses with sustained VT, the echocardiogram should be repeated once the horse has returned to NSR. A continuous 24-hour ECG should also be obtained to more completely evaluate the VA as they are often intermittent. Further workup of a horse with PVCs or AIVR, in the absence of underlying systemic disease, should include an exercising ECG. Horses with VT or complex VA should not be exercise tested.

Summary: Key Recommendations for PVCs and VT

- Underlying causes should be sought and managed if possible.
- Horses with occasional PVCs at rest or during exercise or with sustained AIVR that is overdriven by exercise can be ridden or driven with caution by an informed adult. Owing to ongoing concerns about underlying myocardial or electrical disease and increased risks of exercise associated collapse and SCD, these horses should not be used by a child 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 before 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 symptomatic or complex VA should be rested and treated. Follow-up examinations are similar as for horses with sustained monomorphic VT although the safety of these horses remains uncertain. These horses should only be ridden or driven by an informed adult.
- Rigorous athletic work is not recommended for horses that showed VA in the setting of moderate or severe structural heart disease, including echocardiographic lesions suspected to indicate myocardial fibrosis or scar, and moderate to severe AR. These horses should only be ridden or driven by an informed adult because of the risk of possible recurrence of VT. These horses are not safe for use by a child or as a lesson horse.
- For horses with a history of VT that remain in work follow-up 24-hour and exercising ECGs should be performed at least annually.

Areas for Future Research Investigation

Large multicenter clinical studies are indicated to develop a comprehensive assessment of the severity of valvular regurgitation, its progression, outcome, and the effect of ACE inhibitors in horses with moderate to severe MR and AR. The methods for echocardiographic assessment of cardiac structures, size, and function should be standardized. Critical assessments of the efficacy of different treatments for AF and the factors affecting its recurrence are indicated. The reproducibility of continuous 24-hour Holter ECG monitoring and exercising ECGs also needs to be determined. Large studies in different populations of performance horses are needed to further study the arrhythmias present during exercise and their relationship with horse and rider or driver safety and SCD.

Summary: Key Recommendations for Research Investigation

- Standardization of echocardiographic methods.
- Comparative efficacy of quinidine and TVEC.
- Pharmacotherapy to prevent recrudescence of AF.
- Risk of VA and SCD with AR.
- Refining exercise testing for specific sports.

Collaboration between different investigators on these and other studies will help further advance the knowledge of cardiac disease in the equine athlete and the development of appropriate recommendations.

Conflict of Interest Declaration

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