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REVIEW ARTICLE

Response to equine cardiac adverse events during sports

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Summary

Cardiac adverse events and sudden death are a feared scenario for equine veterinarians, with serious consequences for animal health, riders' safety and the social licence to operate equestrian sports. The response to equine cardiac adverse events (CAEs) during sports is poorly defined. In this review, we provide information to help assess horses post-exercise and suggest a rapid assessment and treatment protocol for veterinarians responding to CAEs during equestrian sports.

KEYWORDS
Arrhythmia, collapse, Exercise Associated Sudden Death, horse

INTRODUCTION

The response to equine cardiac adverse events (CAEs) during sports is poorly defined and complicated by the difficulty of rapidly and safely evaluating, treating and discriminating CAEs from other clinical problems that cause post-exercise distress. The most feared scenario is the collapse and death of the horse, injury of the rider and the impact on the social licence to operate equestrian sports (Bennet & Parkin, 2022; Cameron-Whytock et al., 2025; Douglas et al., 2022; Nath, Stent, et al., 2022; Williams et al., 2023). However, not all CAEs are associated with collapse, cardiac arrest and death of the horse, and possibly for some horses, deterioration might be preventable or the condition could be successfully treated.

Arrhythmias are suspected to be the most common cause of CAEs in horses. Tachyarrhythmias may cause exercise-related distress with or without immediate collapse. Ventricular tachycardia (VT) and atrial fibrillation (AF) with a fast ventricular response rate are most likely to cause this type of distress as these can result in poorly coordinated and weak ventricular contractions, reduced cardiac output and subsequent poor oxygen delivery to the brain (Navas de Solis, 2020; Reimer et al., 1992). Signs may be more subtle if the heart rate is not very high. Horses may also exhibit neurologic, aggressive, panicked, colicky or other inappropriate and potentially dangerous behaviours, and it could be speculated that this is analogous to the anxiety or panic that humans experience due to the sensation of a fast, irregular rhythm (palpitations, tachycardia awareness) even when the rhythm is not dangerous (Pohl et al., 1988). Because AF with fast ventricular response and VT may degenerate into ventricular fibrillation, collapse and sudden death, they should be treated urgently. Atrial fibrillation (AF) and other supraventricular tachyarrhythmias (SVTs), when detected at rest, rarely result in marked behavioural distress and seldom need urgent, life-saving treatment. Atrial fibrillation is the most common

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performance limiting arrhythmia in horses and can be observed in horses as an incidental post-race finding. In addition to poor performance, it is also encountered in horses with nonspecific signs of poor recovery, and mildly to severely distressed horses post-exercise (Nath et al., 2023). Atrial fibrillation was a common arrhythmia identified in a recent case series describing electrocardiographic findings in horses with exercise-associated sudden death (EASD) (Navas de Solis et al., 2026). The clinical signs of AF, or other SVTs, may overlap with VT and can degenerate into VT or other malignant rhythms when associated with high ventricular response rates, sympathetic stimulation or sudden onset during or after exercise. While differentiating AF and other SVTs from VT in post-exercise ECGs is a skill worth developing, it is worth recognising that it is not always easy, possible or safe to obtain or interpret a rhythm diagnosis in the distressed horse. Consequently, treatment protocols will need to take this into account. Bradyarrhythmias are also a potential cause of arrhythmogenic collapse that is less likely to manifest during or shortly after exercise (Keen, 2020; Lyle et al., 2010).

Other cardiovascular events that may be associated with exercise-related distress include exercise-associated fatal pulmonary haemorrhage (Rocchigiani et al., 2022), epistaxis associated with sudden onset of atrial fibrillation (Hansen et al., 2024; Nath et al., 2023), rupture of the aorta or other large vessels (Marr et al., 1998; Sleeper et al., 2001) or rupture of chordae tendineae of the mitral valve with rapid onset of pulmonary oedema (Brown et al., 1983; Holmes & Miller, 1984). Exercise-induced pulmonary haemorrhage (EIPH) and/or epistaxis can be seen in horses with CAEs, and the nature of the association (cause, consequence or parallel) of EIPH with the rest of the clinical presentation is uncertain and a frequent topic of debate. Previously undiagnosed degenerative, inflammatory or infectious cardiac diseases may be coincidentally detected in the post-exercise period. The potential association between myocarditis and systemic inflammation with arrhythmogenesis is interesting (Tran et al., 2025) but poorly understood in horses. These inflammatory diseases are often first detected outside the post-exercise period and are more commonly thought to trigger different clinical signs than post-exercise distress, although they may contribute to the development of arrhythmogenic myocardial substrates.

Cardiac arrest, the sudden and complete loss of cardiac function, almost invariably leads to sudden cardiac death in the adult horse unless it occurs in a controlled setting, like the anaesthetised horse (Duggan et al., 2022; Munerato et al., 2025). Some equine veterinarians believe a coordinated response to CAEs in equine sports is futile, given the difficulty of assessing and treating horses in this context and the likely outcome. The survival rate after human sudden cardiac arrest during sports is high, which contrasts with the lower survival or arrest rates during critical illness. The overall survival after sudden cardiac arrest during sports is approximately 63% when a bystander provides CPR and approximately 83% when a certified athletic trainer assists in resuscitation. The highest rate of survival (89%) occurs in cases with a shockable rhythm, where a defibrillator is used (Drezner et al., 2019; Olasveengen et al., 2020). Defibrillation and CPR, hopefully, will become part of the equine sports-associated CAE discussion in the future; however, they are currently impractical in the field setting and, for this reason, are not discussed in this manuscript.

In this review, we aim to provide clinicians with information useful for evaluating and treating horses experiencing CAEs during sports. For this purpose, we will use the following definitions.

Exercise-related distress

Clinical presentation of horses around the time of exercise that includes the presence of collapse, recumbency, stupor, excitability, inappropriate behaviour, difficult walking, moderate or severe ataxia, colic-like behaviour, stumbling, falling, trembling, dyspnoea or tachypnoea.

Cardiac adverse event (CAE)

A cardiac problem that is associated with exercise-related distress. Non-perfusing rhythms, arrhythmias with severe haemodynamic consequences or sudden cardiac arrest are some potential causes of CAE.

EVALUATION OF THE HORSE AFTER EXERCISE

Healthy horses with a normal response to high-intensity exercise exhibit post-exercise vital signs and diagnostic aid results that differ from the reference ranges of the resting horse.

Cardiac auscultation

The normal heart rate after exercise is high and variable. The peak heart rate during the preceding exercise, the interval between exercise and auscultation and the level of excitement need to be considered when assessing heart rate after exercise. The maximal sinus heart rate of horses is individually dependent and typically ranges from 210 to 240 beats per minute. The peak heart rate during a specific exercise bout is primarily related to exercise intensity and the stimulation of the sympathetic nervous system. Maximal heart rate is only achieved during exercises like racing, breezing or treadmill training and occasionally during uphill gallops, cross-country competition or foxhunting. The normal heart rate deceleration response after maximal or near-maximal high-intensity training is characterised by a precipitous fall during approximately the first

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minute after exercise. Then, the heart rate decreases rapidly and linearly until approximately 5 min post-exercise before declining at a consistent but slower rate to approximately 100–120/min at approximately 15 min post-maximal exercise (Schrurs et al., 2024). The average heart rate of a population of horses auscultated while standing at 30 min and 60 min after racing was reported to be 82 and 67 beats/min, respectively (Witherington, 1971). In other modalities of high intensity but submaximal exercise, such as cross-country competition or training gallops in eventers, the heart rate may be somewhat lower and generally approximates 55%, 50%, 47.5%, 42.5% and 37.5% of the peak heart rate at 1, 2, 5, 10 and 15 min, respectively (Kirsch et al., 2019, 2020). The peak heart rate in these types of high-intensity exercise in non-racehorses varies among horses and the type of workout and is often 180–210/min (Durando et al., 2024; Kirsch et al., 2020; Navas de Solis et al., 2026). This means that, due to the frequently lower peak heart rate, the expected heart rates during recovery will be somewhat lower than those of a racehorse after breezing or racing. Horses participating in sports that do not require maximal or submaximal exercise often return to low and near-resting heart rates more quickly. From the information described above, general rules are that horses with sustained non-exercising heart rates higher than 150 beats per minute at any time, heart rates over 140 beats per minute 20 min after high-intensity exercise, 120 beats per minute 30 min after high-intensity exercise or 100 beats per minute 60 min after high-intensity exercise have inappropriate tachycardia (Kirsch et al., 2020; Witherington, 1971) and should be closely monitored.

The cardiac rhythm is normally regular in the late post-exercise period; however, intermittent or occasional irregularities are frequent during fast heart rate deceleration in the immediate (1–2 min) post-exercise period (Physick-Sheard & McGurrin, 2010). Horses with occasional premature complexes, sinus pauses or physiologic arrhythmias such as second-degree AV block, sinus block or sinus arrhythmia without underlying severe tachycardia are at low risk of CAE and generally do not need antiarrhythmic therapy or other interventions. Complex rhythms like ventricular or supraventricular tachycardias, multiform QRS morphology, R on T phenomenon or atrial fibrillation with a high ventricular response rate may have a higher risk of collapse and sudden death, but the quantification of this risk is currently not possible (Navas de Solis et al., 2026; Physick-Sheard, 2013; Reef et al., 2014; Verheyen et al., 2013).

Murmurs are common in athletic horses. Murmurs that are left-sided, grade 2/6 or quieter; that are early to mid or mid to late systolic; and that are variable in intensity are often benign and associated with ejection or mild mitral valve prolapse/insufficiency. Murmurs that are grade 3/6 or louder, holosystolic, pansystolic or diastolic are likely to require evaluation but rarely require emergency therapy. Exceptions may be murmurs associated with severe regurgitation, a ruptured chorda tendinea of the mitral valve or an aortic root fistula. Ruptured chordae tendineae tend to be associated with murmurs that are at least 3/6 in grade, have a musical or honking component and are holosystolic or pansystolic. Aortocardiac fistulae, often causing right-sided continuous murmurs, are likely to need emergency therapy and may be associated with arrhythmias (Reef et al., 2014). Aorto-pulmonary fistulae, mainly seen in Friesian horses, usually cause soft systolic and early diastolic murmurs over the pulmonary or aortic valve area.

Causes for concern during cardiac auscultation and the potential need for further evaluation and/or intervention are:

- Horses that have resting heart rates over 150/min at any time, 140/min 20 min or 120/min 30 min or 100/min 60 min after exercise
- Horses with irregularly irregular rhythms that are suggestive of atrial fibrillation
- Horses with intermittent or sustained tachyarrhythmias at high rates (>100/min approximately) are suggestive of ventricular or supraventricular tachycardias
- Horses with murmurs that are grade 3/6 or louder, musical, honking, holo/pansystolic, holodiastolic or continuous, particularly if they are a new occurrence

Respiratory auscultation

The respiratory rate of horses in the post-exercise period will be increased. A persistently high respiratory rate can indicate pain, heat illness or respiratory disease. Pulmonary oedema can cause adventitious lung sounds, such as crackles, which may be difficult to detect in some horses. Wheezes might indicate bronchoconstriction due to asthma, anaphylactic reaction or other underlying lung disease; however, this is an uncommon cause of exercise-related distress in horses without previously recognised clinical signs. 'Thumps' (synchronous diaphragmatic flutter) may be associated with electrolyte abnormalities (often calcium or magnesium), dehydration and alkalosis. Although it is rarely an emergency when identified in the post-exercise period in the otherwise healthy horse, as it may self-correct with sufficient time, treatment is generally directed at electrolyte supplementation with calcium- and magnesium-containing fluids. Endurance horses and horses with clinical abnormalities beyond 'thumps' need special attention (Faircloth et al., 2025).

Causes for concern during respiratory evaluation/auscultation and the potential need for further evaluation and/or intervention are:

- A persistently high respiratory rate that indicates respiratory distress
- Panting/mouth breathing
- Foam in the nostrils, especially if blood-tinged

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- Epistaxis
- Adventitious lung sounds such as crackles and wheezes

Temperature

The body temperature increases during exercise. Intense exercise is often associated with increases in temperature to 42°C (107.6°F), and severe clinical signs are frequently described with temperatures over 43.5°C (110.3°F). For temperatures between these two thresholds, there is a variable display of clinical signs, and it is plausible that the degree of heat-related tissue damage depends upon temperature, the area under the curve of temperature and duration and the interaction with other variables. Independent of the definition of heat stroke or illness and the specific rectal temperature that triggers signs, cooling in the post-exercise period often helps distressed horses (Brownlow & Mizzi, 2023).

Causes for concern from the thermoregulation standpoint, indicating a potential need for further evaluation and/or intervention, are as follows:

- A fast and shallow (panting) respiratory rate
- Inappropriately dull or manic (e.g. thrashing, circling, striking, repeated vocalising) behaviour
- Rectal temperature over 107°F (42°C) at 30–60 min after exercise

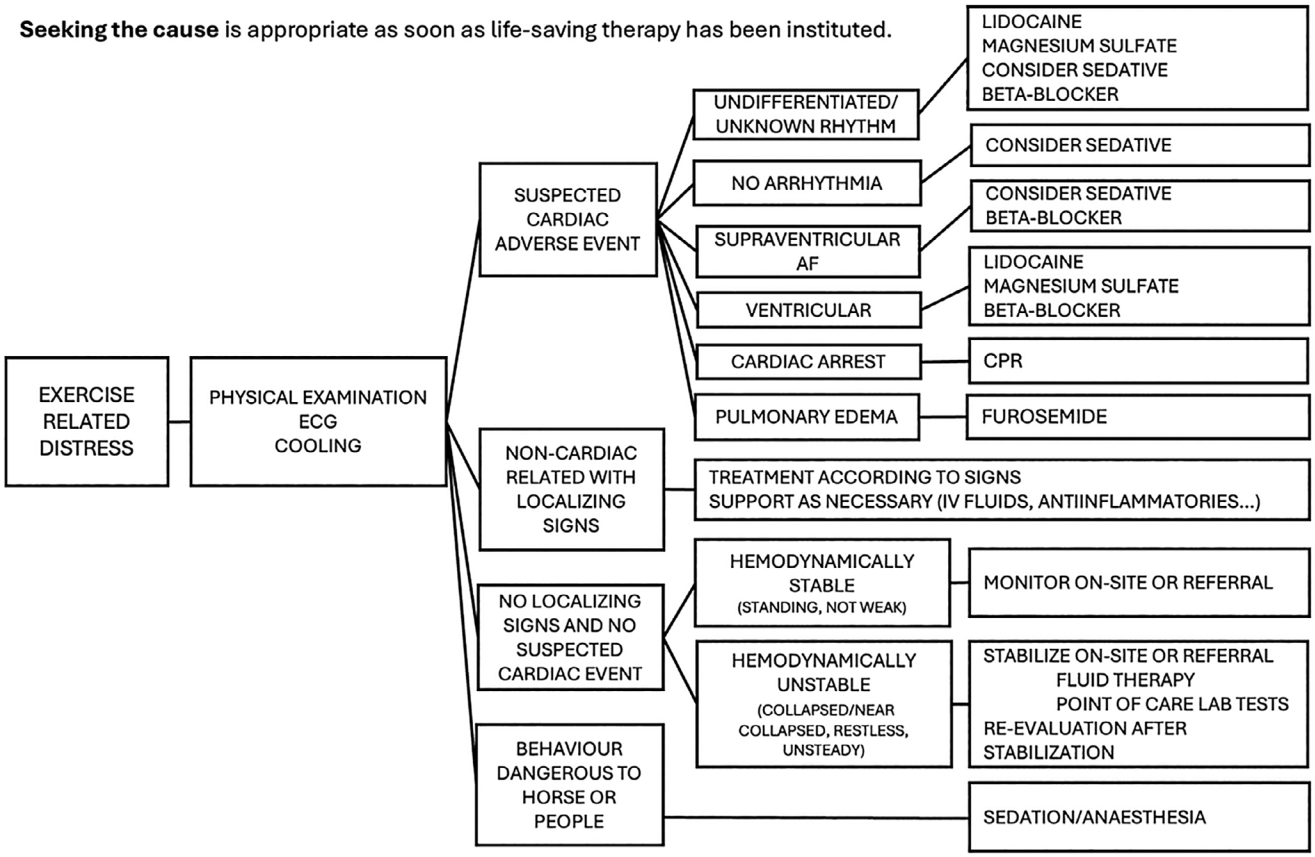
Cardiac rhythm evaluation

Inappropriate tachycardia or an irregular rhythm during auscultation in the post-exercise period is indication to obtain an electrocardiogram (ECG) if the horse's behaviour allows. Different devices are available for obtaining ECGs in a field setting. Small pocket/handheld devices (Kardia) or digital stethoscopes (EKO) with a coupled ECG may be suitable for rapid acquisition, digital storage and teletriage. Telemetry units (Televet) or ECGs embedded in heart rate monitors, wearable devices or fitness trackers designed for horses (Arioneo Equimetre, Etrakka, Equimetrics) or adapted from humans (Movesense, Polar) may be helpful when more than a short ECG (30–60 s) is needed. All these devices have advantages and disadvantages, and Kardia, for short recordings, and Televet, for long or multi-lead recordings, have been used more frequently in peer-reviewed literature (Haughan et al., 2021; Nath, Forbes, et al., 2022; Ter Woort et al., 2022). Small handheld devices are often useful post-exercise but can display a single lead of low amplitude, and it can be challenging to determine if the QRS morphology is normal. Using the Kardia 6L with a lead extender (an electrode placed in front of the left scapula) will provide a base-apex ECG in addition to five unconventional leads, which may help overcome problems with small amplitude and the availability of a single lead. ECGs obtained with some handheld devices can be sent via email or instant messaging applications to an expert if remote assistance is needed. Many of these devices have algorithms for automatically analysing rhythm and, in the case of stethoscopes, for detecting murmurs. The use of the Equifib application, which utilises Polar Equine hardware, is the only one of these devices that features an algorithm optimised for detecting AF in horses (Van Steenkiste et al., 2025). Algorithms embedded in devices designed for humans (Kardia, EKO) are often inaccurate. It is likely that soon, other devices and software will become available for rhythm analysis in horses. Table S1 describes features of available devices, and Data S1 provides instructions on using the Kardia 6L in horses. For all other devices, instructions are available through the manufacturer's links in Table S1. These devices and their interfaces are frequently updated, and it is likely that shortly after the submission of this manuscript, the table will become obsolete. However, the aim for it is to serve as a starting point for the interested reader.

Standardisation of lead position is a notable current goal in equine electrocardiography. This may help share data sets and create new knowledge in electrocardiographic interpretation (Paulussen et al., 2024). For systems with four electrodes, the Delta configuration (based down) has been proposed to maximise information about the equine conduction system. This is: 'right arm' (RA, red) electrode right to the withers at the level of the top of the scapular spine, 'left arm' (LA, yellow) just caudal to the right olecranon, 'left foot' (LF, green) just caudal to the left olecranon, 'right leg' (RL, black) left to the withers at the level of the top of the scapular spine (note: European colour standards for ECG). For most wearable devices that can be useful in the triage setting, standardisation is difficult and may be less relevant or set by manufacturers; however, approximating a standard lead is worth considering. General guidelines for basic ECG acquisition using wearables and potential response to arrhythmias are given in this document, but the interpretation of complex ECGs is beyond the scope of this review. Examples of common arrhythmias obtained with different devices are included in Figures S1–S11. We also discuss a general approach that could be useful when a diagnosis is not possible. It is essential to recognise that an electrocardiographic diagnosis will generate the most appropriate therapy and that the clinical variability of individual situations must take precedence over general guidelines.

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TRIAGE AND TREATMENT ALGORITHM FOR EXERCISE-RELATED DISTRESS AND SPORTS-RELATED EQUINE CARDIAC ADVERSE EVENTS



THERAPY

Several aspects of the proposed triage and treatment algorithm require further explanation.

Seek cause

Physical examination and ECGs are helpful aids if the patient is compliant. If the heart rate is >150/min at any time at rest, >140/min 20 min after exercise, >120/min 30 min after exercise or >100/min >60 min after exercise, a cause should be sought and the horse monitored. When an ECG confirms sinus tachycardia or the rhythm is regular and there are no signs of cardiovascular collapse (such as weakness or recumbency), the clinical signs should guide the therapy, and no medications to control the rate may be necessary. Pain, hyperthermia, systemic disease and disproportionate excitement/anxiety, among others, are potential causes of inappropriate sinus tachycardia. Of note, however, is that uniform VT and SVT will also be rapid regular rhythms on auscultation and may occur with or without clinical signs. An ECG to distinguish sinus tachycardia, SVT and VT or empirical therapy in clinically affected horses may be necessary. Rapid irregular rhythms on auscultation should always raise suspicion for malignant arrhythmias, particularly if accompanied by weakness, recumbency, poor recovery from exercise or altered mentation or behaviour. Both multiform VT and rapid atrial fibrillation are irregular rhythms that can sound similar on auscultation and must be distinguished from rapid irregular decelerations and accelerations in the early recovery period after intense exercise. ECG distinction of ventricular versus atrial arrhythmias is best as therapy will differ, but empirical therapy directed at rate control or prioritising the treatment of potential ventricular arrhythmias may be necessary in the realistic clinical scenario.

Cooling

Showering the whole body with tap water for 5 min or intermittent cold water (e.g. 3 treatments of 16 L of iced water over 3 min) over the whole body with or without scraping is an effective method of cooling (Marlin et al., 1998; Takahashi et al., 2020). These methods can be repeated as required. Fans and keeping horses in shaded

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areas between and after water application may improve cooling. Cooling could be helpful in most scenarios of the distressed horse post-exercise to centralise blood volume and reduce heart rate. Exertional heat illness can exhibit clinical signs that overlap with CAE (Brownlow & Mizzi, 2023), and the increase in temperature can have compounding effects on tachycardia and arrhythmogenesis (Goetz & Manohar, 1990; Kaur et al., 2022; Morita et al., 2007). For these two reasons, considering exercise-related hyperthermia and heat illness in the treatment of CAEs is relevant. If heat illness is suspected, further treatment for this condition can be implemented. This has been recently reviewed (Brownlow & Mizzi, 2023), and considering the use of sedatives and anti-inflammatory agents, in addition to cooling, is an important aspect of treatment for heat illness.

Sedatives

When excitement or pain is the suspected cause of persistent tachycardia, sedatives at low to standard dosages can be given. Clinical experience among racetrack veterinarians suggests that low-dose sedation, such as 2 mg of detomidine IV/500 kg, may be beneficial in the clinical situations of the standing horse or the recumbent, agitated horse with undifferentiated tachycardia, as well as in the management of SVTs or rapid AF. It is suggested to repeat this dose up to three times if the heart rate remains markedly elevated. Dexmedetomidine (0.5–1 mg/500 kg horse) may have a similar effect with a proposed additional antiarrhythmic effect in humans (Jiang et al., 2024) but is currently a less commonly used drug in equine practice. Alpha-2 agonists at doses significantly higher than those suggested above have been shown to be safe in the post-exercise period in experimental settings (Hubbell et al., 1999). The initial hypertensive response (particularly with higher doses of detomidine) must be considered, particularly in horses with suspected haemorrhage, and the later hypotensive response has to be considered in all individuals (Bettschart-Wolfensberger et al., 2005; Hubbell et al., 1999, 2025; Yamashita et al., 2000). QT prolongation and the promotion of long-short RR sequences/pauses are suggested disadvantages of alpha-2 agonists (Trachsel et al., 2021) and a potential concern in the early recovery period, particularly when the heart rate is rapidly decelerating. The clinical perception is that a decrease in heart rate outweighs this risk in other clinical scenarios. Acepromazine has been suggested as a potentially helpful drug for anxiety, but the slow onset of action, hypotension and tachycardia after administration must be considered.

Lidocaine and magnesium sulfate

These antiarrhythmics are often helpful for the treatment of VT. Confirming VT is ideal, but sometimes not possible. Adverse effects are very uncommon when lidocaine and magnesium sulfate are appropriately dosed and delivered. Therefore, empiric treatment with these potentially lifesaving drugs in the severely distressed horse may be acceptable in many clinical situations, even in the absence of a definitive diagnosis. A standard dose for lidocaine is 250 mg slowly IV/500 kg horse repeated up to four times at 3–5 min intervals. A standard dose of magnesium sulfate is 25 g/500 kg in 1 L of IV fluids over 15–25 min, and an intravenous catheter is needed for this treatment. Hypotension is the main risk of rapid infusion. Magnesium sulfate is the drug of choice for torsades de pointes (TdP), a polymorphic ventricular tachycardia (PVT) seen in the setting of prolonged QT intervals (Mitchell, 2017). Magnesium blocks calcium channels thereby reducing the early afterdepolarisations that initiate TdP. Magnesium is generally considered less effective for other types of PVTs. Because magnesium also slows conduction through the AV node, it is potentially useful for slowing the ventricular response rate to AF. When an abnormal rhythm has been successfully treated pharmacologically, continuous electrocardiogram monitoring is indicated, as the short half-lives of these drugs can be associated with recurrences. Dosages above may need to be followed with constant rate infusions or other medications after assessment.

Beta-blockers

Beta-blockers, such as metoprolol (20 mg IV/500 kg horse), can be effective for supraventricular tachycardias, AF with a high ventricular response rate or ventricular arrhythmias that do not respond to lidocaine or magnesium sulfate (Luethy et al., 2022). Sotalol (2 mg/kg orally) is a class III antiarrhythmic with beta-blocking effects (Broux et al., 2018; Decloedt et al., 2018). However, oral administration of sotalol limits its usefulness in the emergency setting. Propranolol can be administered at a dose of 0.2–1 mg/kg IV, and its half-life is approximately 2 h. The co-administration of propranolol and alpha 2 agonists enhances bradycardia, the sedative effect and causes ataxia, raising safety concerns (Aramaki et al., 2000; Cenani et al., 2017) about the combination of alpha 2 agonists and beta-blockers. Propranolol has more potential to cause bronchoconstriction due to its non-selective beta effects (vs. more selective β-1 effect of metoprolol or esmolol). Esmolol is another β-blocker with the potential benefit of having a shorter half-life (9 min described in humans vs. 21 min half-life of metoprolol described in the horse), but the pharmacokinetics of esmolol in the horse have not been described. There is scarce information about the use of beta-blockers in equine species at the time of exercise (Snow et al., 1979), and no information is available during the post-exercise period or in horses with CAE. Lower-than-expected heart rate, rapidly decreasing heart rate, arrhythmias that include pauses, suspected myocardial/systolic dysfunction, hypovolaemia or hypotension may be scenarios when adverse effects of beta-blocker could be more likely. Although this needs to be acknowledged, beta-blockers can be considered mechanistically promising in a potentially common scenario like AF with rapid ventricular response or other ventricular or undifferentiated arrhythmias that do not respond to lidocaine and magnesium sulfate. Some regulatory organisations, such as the Horseracing Integrity

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and Safety Authority (HISA) in the US or the FEI, consider most beta-blockers banned substances. Other medications that can be useful in the distressed horse are included in Table S2.

CONCLUSION

In this review, we provide information to help assess horses post-exercise and suggest a rapid assessment and treatment protocol for veterinarians responding to CAEs during equestrian sports. We want to emphasise that diagnostic algorithms cannot fully capture the complexity of many clinical scenarios, and the standard of care can often be improved when a clinician's judgement is not limited by an algorithm. It is unknown if developing a coordinated approach to equine CAE during sports can improve survival. However, it seems possible that a practical and organised response to CAEs, further understanding of drugs useful in equine CAEs, and development of technology to improve resuscitation efforts could be a worthwhile investment in equine health and welfare. The documentation of responses to therapy in horses with CAEs is sparse; establishing a registry of treatment responses would be a worthwhile endeavour.

AUTHOR CONTRIBUTIONS

C. Navas de Solis: Conceptualization; investigation; writing – original draft; writing – review and editing. **J. Keen:** Conceptualization; investigation; writing – review and editing. **A. Decloedt:** Conceptualization; investigation; writing – review and editing. **B. Delvescovo:** Conceptualization; investigation; writing – review and editing. **K. Hopster:** Conceptualization; investigation; writing – review and editing. **K. Mitchell:** Conceptualization; investigation; writing – review and editing. **J. Slack:** Conceptualization; investigation; writing – review and editing. **G. van Loon:** Conceptualization; investigation; writing – review and editing. **G. Van Steenkiste:** Conceptualization; investigation; writing – review and editing. **L. Nath:** Conceptualization; investigation; writing – review and editing.

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ETHICS STATEMENT

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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