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CASE REPORT

# Early recognition with capnography, and successful resuscitation, of severe cardiac dysfunction in a horse receiving amiodarone during transvenous electric cardioversion (TVEC)

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## Summary

A 19-year-old Cob mare with atrial fibrillation (AF) presented for transvenous electric cardioversion (TVEC). Under general anaesthesia, three successive incremental electric shocks were given (total 750 J) and sinus rhythm was temporarily restored. After 13 s, AF recurred so a fourth shock (300 J) was given and intravenous (IV) amiodarone started. Shortly after, capnography revealed an acute drop in the end-tidal carbon dioxide, which was followed by agonal gasping. Echocardiography showed severe cardiac dysfunction. Successful cardiac resuscitation consisted, among others, of cessation of amiodarone and administration of dobutamine and adrenaline. This case report highlights the importance of capnography as a non-invasive means of cardiac output monitoring in equine anaesthesia which definitely contributed to a positive outcome in this case. It also emphasises the critical importance of early recognition and communication of complications within anaesthetic and cardiology teams.

KEYWORDS
horse, amiodarone, capnography, TVEC

## INTRODUCTION

Atrial fibrillation (AF) is a commonly diagnosed arrhythmia in horses, associated with limited performance (McGurrin, 2015) and safety concerns for riders (Decloedt et al., 2020; Reef et al., 2014). Treatment aims to restore sinus rhythm to improve performance and safety (Reef et al., 2014). This contrasts with treating AF in humans and dogs where rate control is the primary treatment objective (Bright & zumBrunnen, 2008; Wyse et al., 2002), except in patients with haemodynamic instability when electric cardioversion is used to restore normal sinus rhythm (Kirchhof et al., 2016; Wilson et al., 2021). Historically, medical treatment with quinidine sulfate, a class IA antiarrhythmic/$Na^+$ channel blocker, has been used for cardioversion of horses in AF (Reef et al., 1988). However, this drug has potentially severe side effects occurring in up to 75% of cases including colic, diarrhoea, ataxia, hypotension, upper respiratory tract stridor, supraventricular tachycardia and prolonged QRS complex (Reef et al., 1995). Additionally, quinidine sulfate has become difficult to source recently. Therefore, transvenous electric cardioversion (TVEC) of horses with AF is becoming more common, as it is increasingly available and shows good success (McGurrin et al., 2008; Vernemmen et al., 2022).

This report describes a case of acute severe cardiac dysfunction during TVEC in a horse presenting with AF after receiving amiodarone during the procedure. The importance of early recognition of critical events with adequate monitoring and good communication between personnel to optimise outcomes is discussed, as well as potential aetiologies and resuscitation.

## CASE HISTORY

### Clinical findings

A 19-year-old, 596 kg Cob mare presented for evaluation of a cardiac arrhythmia consistent with AF. Initial cardiovascular clinical examination showed pink mucous membranes with a capillary refill time <2 s and an irregular pulse rate, with variable pulse quality. Cardiac auscultation revealed an irregularly irregular rhythm with a rate of 42 beats/min, and no significant murmurs.

A baseline resting electrocardiogram (ECG) confirmed AF, showing normal QRS complexes with irregularly irregular R–R intervals, absence of P waves and an undulating baseline consistent with F waves. An ECG recorded during exercise revealed neither abnormal ventricular complexes, nor rapid conduction through the atrioventricular node. The heart rate at each exercise level was slightly above the normal reference range. Further investigation with two-dimensional and Doppler echocardiography revealed no significant abnormalities of cardiac dimension or indices of systolic and diastolic function.

These findings suggested lone atrial fibrillation, that is, not associated with significant structural cardiac disease and therefore electrical cardioversion was attempted. Treatment with oral sotalol 2 mg/kg (Bristol Laboratories Ltd.) twice daily was initiated 2 days prior to TVEC. The last sotalol dose was given 3 h 45 min prior to induction of anaesthesia.

With all these findings, the owners were informed about the procedure and written owner consent was obtained.

### Pre-anaesthetic management and catheters/lead placement

Based on the information above, the patient (non-food-producing animal) was assigned an ASA (American Society of Anaesthetists) score of II. On the day of the TVEC, the horse was fasted for 6 h overnight with free access to water. A long stay 14G 7-inch cannula (MILACATH; MILA International) was inserted aseptically under local anaesthesia into the left jugular vein for routine IV access. The horse received flunixin meglumine 1.1 mg/kg (Meflosyl; Zoetis UK Ltd.) IV. Acepromazine 0.04 mg/kg (AceSedate; Jurox) was administered IV, followed by two romifidine doses (0.015 mg/kg IV total) (Sedivet; Boheringer Ingelheim) 60 and 110 min later respectively, to facilitate aseptic placement into the right jugular vein of three specialised introducer cannulae (Terumo 9Fr introducer RSB9ON10SQ; Terumo UK). Under echocardiographic and pressure waveform guidance, the introducer cannulae allowed the positioning of two cardioversion catheters (Gaeltec Devices Ltd.); one into the right ventricle for later withdrawal into the right atrium (RA) via the most cranial introducer and one into the left pulmonary artery (PA) via the middle introducer. A pacing catheter (7 Fr Electrode; ZUCKER™ Lumen SKU:051513P Bard Medical) was placed in the right ventricle via the most caudal introducer (Decloedt et al., 2021). Correct placement was confirmed using thoracic radiography. A total sedation time of 74 min was required for the placement of introducer cannulae and catheters.

### Anaesthetic management

Immediately after, the horse was moved to the induction box in the same building of The Dick Vet Equine Hospital and was premedicated IV with romifidine 0.04 mg/kg and morphine 0.1 mg/kg (Martindale). General anaesthesia was induced 10 min later with IV ketamine 2.2 mg/kg (Ketamidor; Chanelle) and diazepam 0.06 mg/kg (Hameln). Induction, behind a swing gate in the induction box, was smooth. Induction time will be considered T0 for chronological explanation of events (Figure 1). The horse's trachea was intubated blindly without difficulty, with a cuffed 30 mm internal diameter equine silicone endotracheal tube, before the horse was hoisted onto a surgical table and positioned in left lateral recumbency.

Anaesthesia was maintained with sevoflurane (Sevotek; Animalcare) vaporised in oxygen ($O_2$) (1–4 L/min) and delivered via a large animal anaesthetic machine (Tafonius; Vetronic Services Ltd.). The horse was allowed to breathe spontaneously. Monitoring using a Datex-Ohmeta™ S/5 multiparameter included ECG, pulse oximetry, capnography, invasive arterial pressure (IBP) via a 20G arterial cannula in the right transverse facial artery, fraction inspired (FI)/fraction expired (FE) sevoflurane, FI/FE $O_2$ and a peripheral nasal temperature probe. Intravenous fluid therapy with Hartmann's at 5 mL/kg/h was initiated with a continuous rate infusion (CRI) of romifidine at 0.04 mg/kg/h.

### Cardioversion procedure

Cardioversion to sinus rhythm was performed via sequential shocks using a biphasic defibrillator (Lifepak 12) as previously described (De Clercq, van Loon, Schauvliege, et al., 2008; Decloedt et al., 2021; McGurrin, Physick-Sheard, & Kenney, 2005, McGurrin, Physick-Sheard, Kenney, Kerr, et al. 2005). The first (T17m 28s) and second (T18m 00s) conversion attempts (energy 200 and 250 J, respectively) did not achieve normal sinus rhythm (NSR). The third conversion attempt (energy 300 J, T18m 32s) resulted in atrioventricular blockade for a period of 7 s, followed by transient NSR before returning to AF after 6 s.

The fourth conversion attempt (energy 300 J, T19m 08s) resulted in 13 s of atrioventricular blockade before sustained NSR. As standard, a 10-min observation period to ensure sustained NSR post-conversion began.

At T25m 00s, with the horse in sinus rhythm, an IV infusion of amiodarone (Hameln Pharma Ltd.) was initiated (2.5 mg/kg over 15 min in 0.9% NaCl) via the distal introducer cannula. Initial resistance to amiodarone infusion was noted and flow improved after removal of the pacing lead and flushing of the distal introducer with saline. At this time, the atrial cardioversion catheter and proximal

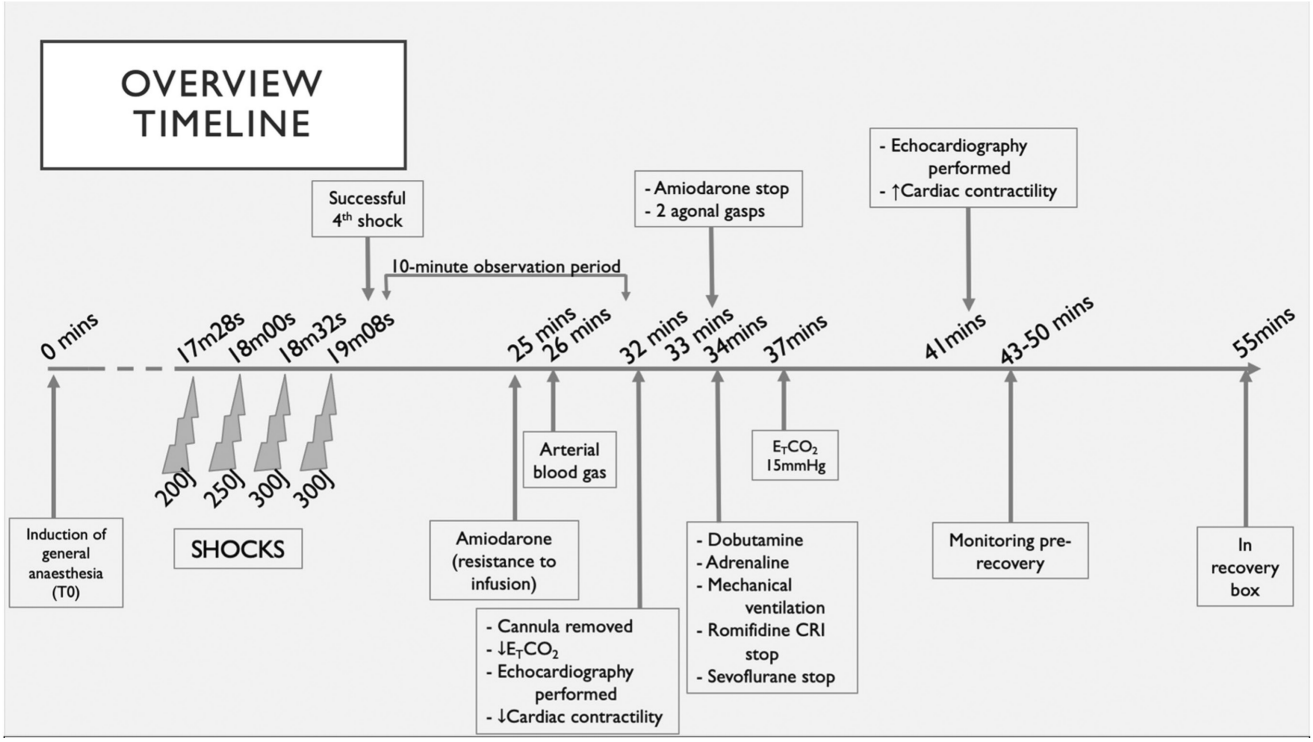


**FIGURE 1** Timeline of case events.

**TABLE 1** Arterial blood gas (reference ranges [Hughes & Bardell, 2019]).

| Parameters | Result |
|---|---|
| pH (7.37–7.49) | 7.39 |
| $PaCO_2$ (36.3–54.0 mmHg) | 52 |
| $PaO_2$ (82.6–112.3 mmHg) | 69 |
| $Na^+$ (133–141 mmol/L) | 142 |
| $K^+$ (3.05–4.65 mmol/L) | 3.3 |
| $Cl^-$ (100–110 mmol/L) | 103 |
| BE (0.51–8.80 mmol/L) | 4.5 |
| $HCO_3^-$ (23.87–32.45 mmol/L) | 30.3 |
| $SpO_2$ (90%–100%) | 90 |
| HCT (%) | 27 |

introducer were withdrawn from the right jugular vein and pressure was applied to the site to limit haematoma formation. Also, at T26m 00s an arterial blood sample was taken anaerobically from the arterial cannula for blood gas analysis, which revealed a moderate hypoxaemia (Table 1).

## Diagnosis

After the 10-min observation period and removing the proximal cannula (T32m 00s), the capnograph showed an acute drop, within 15 s, in end-tidal carbon dioxide ($E_TCO_2$) from 42 to 30 mmHg, which was immediately communicated from the anaesthetic to the cardiology team. The first action taken by the anaesthetic team was confirming that no leak was present in the cuff of the endotracheal tube. Echocardiography showed poor thickening of the right (RV) and left ventricular (LV) free walls and interventricular septum (IVS), indicative of biventricular hypocontractile function and an enlarged right heart, flattening of the IVS and left-sided deviation in diastole, and poor left ventricular filling (Figure 2; Video S1).

## Treatment

One minute later, at T33m 00s, the amiodarone administration was discontinued (total administered 300 mg; 0.5 mg/kg over possibly 60 s) and the horse progressed to an agonal breathing pattern. At least, two agonal gasps were detected at this time. Immediately after (T34m 00s), a rapid bolus of dobutamine (Dobutamine; Hameln) 0.25 mg/mL via manual pressure over 60 s was initiated, aiming to rapidly increase cardiac contractility and, therefore, cardiac output. Simultaneously, adrenaline 0.01 mg/kg (Martindale) was prepared according to an emergency drugs chart attached to the portable anaesthetic trolley and administered IV. The romifidine infusion was discontinued. Also, the vaporiser was switched off for 4 min and end-tidal sevoflurane decreased from 1.9% to 1.7%. Mechanical ventilation was initiated with a tidal volume ($V_T$) of 7.0 L and a respiratory rate (RR) of 12 breaths/min. During this time, $E_TCO_2$ decreased further, reaching 15 mmHg at its lowest (T37m 00s). Hypotension (mean arterial pressure < 70 mmHg) was not detected before the episode of cardiac dysfunction. Unfortunately, exact blood pressure values during the event were not recorded on the anaesthetic record

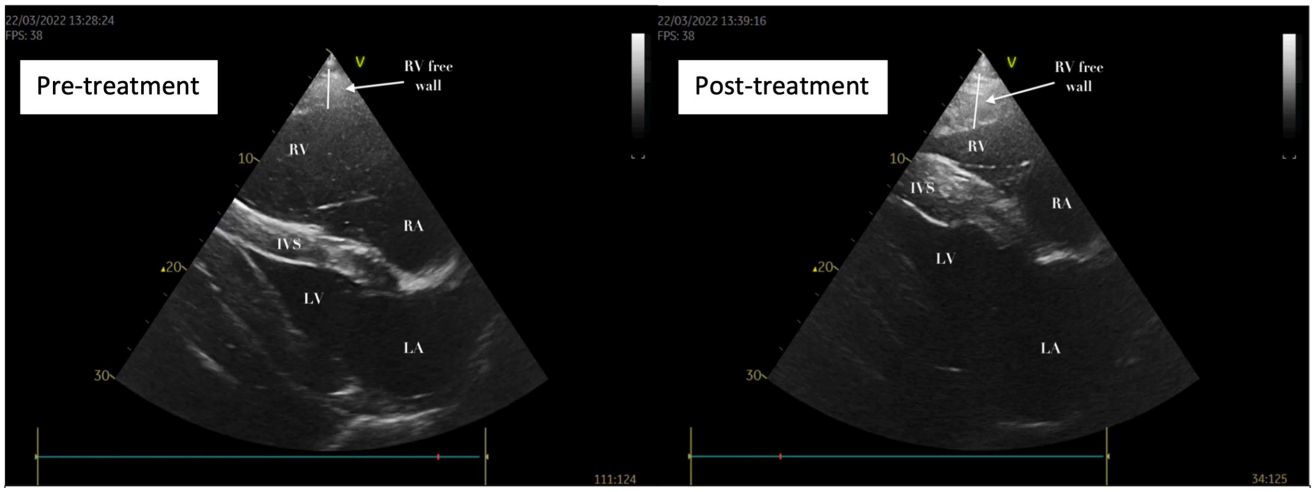


**FIGURE 2** Stills from an echocardiographic examination taken during the event (pre-treatment, T 32m 00s) and after resuscitation had been performed (post-treatment, T41m 24s); RA/LA, right atrium/left atrium; RV/LV, right ventricle/left ventricle; IVS, interventricular septum (Courtesy of Professors L.E Young and J.A Keen).

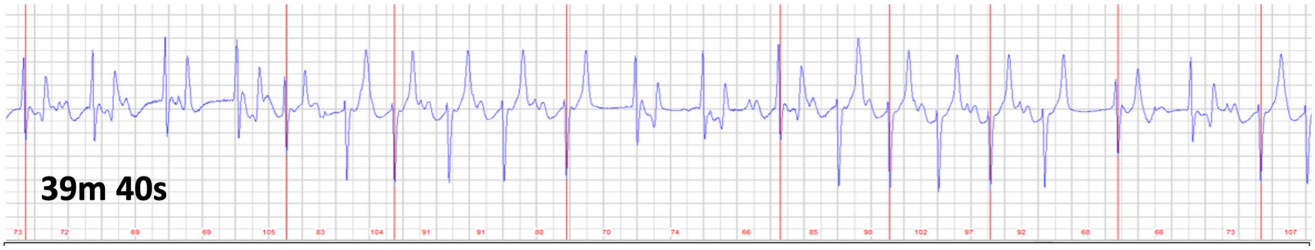


**FIGURE 3** Electrocardiogram (ECG) recorded at 39 m 40 s showing periods of sinus tachycardia interrupted by a slower ventricular rhythm (Courtesy of Professors L.E Young and J.A Keen).

due to prioritising resuscitation of the horse. One minute after adrenaline administration, the horse developed a supraventricular tachycardia (from 43 to 160 beats/min) with a concurrent increase in arterial blood pressure (from 90 to 220 mmHg systolic pressure; and 70–150 mmHg mean arterial pressure, MAP). During this period of sustained tachycardia, multiple arrhythmias developed including ventricular complexes, supraventricular complexes and atrioventricular dissociation (Figure 3). The heart rate decreased from 160 to 91 beats/min and a new echocardiographic examination at T41m 24s revealed improved thickening of the RV and LV free walls and IVS, indicative of improved myocardial contractility, a reduction in RV volume, improved LV filling and no IVS deviation (Figure 2; Video S2). The heart rate continued to decrease from 91 beats/min and sinus rhythm was sustained at a normal rate of 35 beats/min at T43m 00s.

### Outcome

After 10 min of continuous monitoring, with all the monitored variables within normal limits and the horse spontaneously breathing, romifidine 0.01 mg/kg was administered IV while still on the table. The horse was then hoisted and positioned in the left lateral recumbency in the recovery box. The total duration of general anaesthesia was 55 min. Head and tail rope-assisted recovery was uneventful and the horse stood after one attempt within 30 min.

Once back in her stable, the horse remained hospitalised for continued monitoring. The horse underwent clinical examination 3 h after the procedure and had an irregularly irregular heart rhythm, indicating early recurrence of AF. At discharge 2 days later the horse remained in AF. No further complications following anaesthesia were reported.

## DISCUSSION

This case report describes the successful resuscitation of a horse with severe cardiac dysfunction receiving amiodarone during TVEC. It highlights the importance of early recognition of abnormalities with appropriate monitoring during equine anaesthesia. Furthermore, it demonstrates that efficient communication between trained personnel contributes to improved patient outcomes.

Interventional options for AF in racehorses comprise pharmacological cardioversion or TVEC. Determining the duration of AF is prognostically important for conversion success and AF recurrence, as electrical and structural remodelling of the atria occurs over time (De Clercq, van Loon, Tavernier, et al., 2008).

Pharmacological conversion using quinidine sulfate avoids the need for general anaesthesia, showing conversion success rates of 88% in horses without concurrent cardiac disease and a recent (<2 months) history of AF (McGurrin, 2015; Reef et al., 1988). However, there are multiple, well-described systemic side effects of this therapy (Reef et al., 1995), as previously discussed in the Introduction section.

In this case, we chose to use electrical cardioversion as described in humans, horses, dogs and cats (Bright et al., 2005; Klainbart et al., 2021; McGurrin et al., 2008; Nathan et al., 1984; Nusair et al., 2010), to avoid any side effects associated with the use of quinidine sulfate. A recent retrospective study of 231 TVECs demonstrated a conversion rate of 94.4% (Vernemmen et al., 2022). Currently, this is the preferred treatment for horses with suspected prolonged duration AF (>3 months), for cases in which pharmacological conversion is contraindicated e.g. rapid ventricular conduction of AF, complex ventricular ectopy (Reef et al., 2014), and for horses that have previously shown severe adverse reactions to pharmacological conversion. However, the nature of TVEC in horses makes general anaesthesia a prerequisite, which increases the perioperative risks (Gozalo-Marcilla et al., 2021; Johnston et al., 2002). The ASA grade assigned to our case (II) is the same as that assigned to a similar case (Asorey & Corletto, 2022). However, in the paper by Hubbell et al. (2022) AF was proposed to be equivalent to an ASA III (patient with severe systemic disease). While the authors do not believe that all AF cases are equivalent to ASA III, nor that the dysfunction described in this case was attributable to the AF alone, it is worth appreciating that greater cardiovascular dysfunction than expected from an ASA II may occur during anaesthesia for the treatment of AF in horses, due to the techniques and drugs used. The proposed Combined Horse Anaesthetic Risk Identification and Optimisation (CHARIOT) may become a useful tool in the future when grading risk status in horses (Hubbell et al., 2022).

Sotalol prior to, and amiodarone during, general anaesthesia were given to this mare in order to reduce the number of shocks and energy required for conversion, and to decrease the risk of an immediate or early recurrence of AF (IRAF/ERAF) following cardioversion (Broux et al., 2018; Vernemmen et al., 2020). Sotalol is a class III antiarrhythmic with beta-adrenergic and potassium channel blocking activity, which may result in a prolonged atrial fibrillation cycle length (AFCL), Q-T interval and atrial and ventricular effective refractory period (Broux et al., 2018; Decloedt et al., 2018). Amiodarone is another class III anti-arrhythmic. It acts as a potassium channel blocker which increases the duration of the cardiac action potential and prolongs the refractory period, therefore, decreasing cardiac myocyte excitability and preventing ectopic foci-derived tachyarrhythmias. It also has actions as a beta adrenergic blocker, $Ca^{2+}$ and $Na^{+}$ channel blocker (Freedman & Somberg, 1991). Amiodarone's pharmacodynamics result in a prolonged Q-T interval (Decloedt et al., 2018; Hohnloser et al., 1991; Trachsel et al., 2004), reduction in sinoatrial node automaticity and reduction in atrioventricular node conduction velocity (Singh et al., 1976). All of these actions contribute to its anti-fibrillatory effects (Freedman & Somberg, 1991). Amiodarone has been shown to decrease the chances of IRAF, therefore it was infused in this horse given the return of AF 13 s following the first successful shock. The combined use of sotalol and amiodarone could increase the risk of Q-T interval prolongation, which could cause ventricular arrhythmias and a secondary effect on blood pressure (Broux et al., 2018; Decloedt et al., 2018). However, ECG recordings taken just before the agonal gasping, in this case, showed normal sinus rhythm. Therefore, it is unlikely that the cardiac dysfunction we describe here was secondary to the potential cardiac arrhythmias caused by sotalol and amiodarone combination therapy.

The horse in our report demonstrated a severe reduction in myocardial contractility during the IV administration of amiodarone. This was initially detected by capnography as a decreasing $E_TCO_2$ and confirmed with an echocardiographic examination. Capnography is a simple but essential part of anaesthetic monitoring which allows non-invasive, continuous respiratory assessment, as well as being an indicator of pulmonary blood flow (Karlsson et al., 2018). Breath-by-breath assessment provides advanced warning of reduction in cardiac output or ventilation efficiency (Karlsson & Lönnqvist, 2022). However, a short delay may occur when using side-stream capnographs, which are the most commonly used in equine anaesthesia (Dagnall et al., 2021). Our ability to quickly detect the failure in cardiac output enabled immediate communication with the cardiology team and resulted in rapid identification of the cause. Point-of-care echocardiography during the event confirmed a significant impairment of left and right ventricular contractility, resulting in reduced forward flow into the pulmonary artery and aorta. The reduced output from the RV would have led to right ventricular dilatation and poor LV filling. Reduced LV preload would have further decreased LV contractility and cardiac output. Specific measurements to quantify any reduction in cardiac output were not performed due to the emergent nature of the case. Once the cause had been confirmed by echocardiography, appropriate treatment to improve cardiac contractility was implemented and the rapid return of cardiac contractility following this treatment documents a positive outcome.

After confirming the underlying problem, dobutamine and adrenaline were administered in an attempt to improve contractility (Muir & Hubbell, 2009; Schauvliege & Gasthuys, 2013). Dobutamine was given first as it was already prepared and immediately available. When ventricular contractility and relaxation are poor, the time taken for drugs to reach the heart is prolonged; it was felt, as described earlier, that contractility needed to be improved as soon as possible in this horse to increase the chance of a successful outcome. Dobutamine was therefore started while adrenaline was being prepared according to an emergency drug chart, as required by safety guidelines adapted from small animals (Fletcher et al., 2012). The use of both drugs was successful in restoring and maintaining cardiac function before the uneventful recovery. If the pharmacological treatment would have not worked, cardiac compressions would have been considered. However, only limited reports of success exist in adult horses (Ruiz et al., 2018) and, in this particular large-sized Cob, its potential efficacy remains uncertain (Hodgson & Steffey, 1993; Hubbell et al., 1993).

Once in the stable and after an uneventful recovery from general anaesthesia, recurrence of an irregularly irregular heart rhythm was noted. In horses, IRAF and ERAF post-conversion is a complication of both pharmacologic and electric cardioversion. This is more common in patients with a longer history of AF (>4 months), atrial structural changes, and electrophysiologic dysfunction, which all increase the risk of AF recurrence (De Clercq et al., 2014; Reef et al., 1988). This horse had IRAF after the third (first successful) shock, which is why an amiodarone infusion was started. However, despite the use of systemic anti-arrhythmic agents prior to and during the TVEC, unfortunately, ERAF occurred in this case. In human medicine, radiofrequency ablation would have been used to treat the underlying cause of the AF. Although this technology has not yet been fully developed in horses, the treatment of other arrhythmias, such as sustained atrial tachycardia and an accessory pathway, has been reported (Buschmann et al., 2023; Phillips et al., 2022; Van Steenkiste et al., 2022).

Side effects after IV amiodarone have previously been reported in humans, horses, dogs and rabbits, including hypotension and negative inotropy progressing to cardiovascular collapse (Asorey & Corletto, 2022; Cushing et al., 2010; Lessa & Tibiriçá, 2005; Somberg et al., 2005). It has been suggested that the cosolvents polysorbate 80 and benzyl alcohol in the standard IV formulation (Cordarone® Intravenous amiodarone hydrochloride) could drive these cardiovascular effects (Cushing et al., 2010). A recent case report of a horse undergoing TVEC and receiving amiodarone (two infusions of 2.5 mg/kg IV each given over 15 min) described arterial hypotension (MAP 47 mmHg), that responded to treatment with dobutamine (Asorey & Corletto, 2022). In our case, the first indicator of cardiac dysfunction was a decreasing $E_TCO_2$ detected using capnography, rather than an acute drop in MAP. The intended amiodarone dose in our case was 1490 mg given over 15 min, translating to a rate of 99 mg/min. The actual dose administered in our case (total 300 mg; 0.5 mg/kg) was smaller than intended, and smaller than the dose used by Asorey and Corletto (2022), due to complications arising and drug cessation. However, despite the small total dose in our case, we cannot rule out the delivery of a rapid amiodarone bolus (suspected to be ~300 mg/min) after re-establishing the patency of the IV cannula. As with other species, there appears to be individual variation in the cardiovascular response to the infusion of amiodarone in horses. Therefore in future cases, it may be prudent to start with a slower infusion rate over the first 5 min and closely monitor cardiac performance before infusing the full dose.

Finally, it is worth mentioning that our patient signalment is not typical for AF. The majority of equine TVEC studies comprise Standardbred, Thoroughbred and Warmblood populations (Decloedt et al., 2021; McGurrin et al., 2008; Reef et al., 1988; Vernemmen et al., 2022). The age, breed and reduced athletic level in our patient, compared to typical TVEC candidates, could indicate a different underlying mechanism for causing and maintaining the AF in this case when compared to the more common athletic caseload. However, while this remains speculative and may have influenced the recurrence of AF, it is difficult to know how this would influence the response to an amiodarone infusion.

In summary, (i) although the causes of the complications in this case are not clear, IV administration of a bolus of amiodarone during TVEC cannot be excluded. This case (ii) contributes to reports of adverse cardiovascular effects during TVEC and amiodarone administration. Lastly, (iii) it highlights the importance of early recognition and accurate diagnosis with appropriate monitoring, and communication of critical events within trained teams, leading to better patient outcomes.

## AUTHOR CONTRIBUTIONS

E. Pye contributed to case management, writing of the first draft, final preparation of the manuscript, and preparation of the revised manuscript. M. Gozalo-Marcilla and K. Blissitt contributed to case management, and manuscript review and processing. All authors gave their final approval of the manuscript.

## CONFLICT OF INTEREST STATEMENT

No conflicts of interest have been declared.

## FUNDING INFORMATION

None.

## ETHICS STATEMENT

This case report describes the urgent management of an animal which developed a significant anaesthetic complication during anaesthesia for a recognised veterinary procedure and therefore no ethical committee approval was sought. Written owner consent was obtained for the procedure.

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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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