

CASE REPORT

Suspected vagal reflex and hyperkalaemia inducing asystole in an anaesthetised horse

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Summary

A 10-year-old 466 kg mustang gelding presented to an equine referral hospital for surgical repair of nasal, frontal and lacrimal bone fractures from an unknown trauma. Surgical repair was performed under general anaesthesia, including a right-sided maxillary regional anaesthetic block with mepivacaine hydrochloride. Progressive hyperkalaemia was documented perianaesthetically (T-3 mins; 134 mins after induction; potassium 6.4 mmol/L (ref 3.5-5.1 mmol/L). Perianaesthetic bradycardia was attributed to alpha-2 agonist infusion administration, and other characteristic ECG changes (flattened P waves, narrow T waves of increased amplitude, prolonged QRS complex) were not documented. Asystole occurred 137 min after induction of anaesthesia; however, a review of the available literature suggests the degree of hyperkalaemia documented was unlikely to be the primary cause of asystole but may have been a contributing factor. It is hypothesised that a trigeminocardiac reflex was the primary contributory factor to asystole in the described case, and may represent a maxillomandibulocardiac reflex that has not been previously described in the horse.

KEYWORD

horse,

1 | INTRODUCTION

Vagal reflexes have been well documented in both human and veterinary anaesthetic literature. A trigeminocardiac reflex (TCR) is a vagal reflex frequently documented in human medicine, and subtypes such as the oculocardiac reflex (OCR) are well documented in veterinary medicine throughout numerous species.¹⁻³ A further TCR subtype, the maxillomandibulocardiac reflex (MCR) has been documented in human literature associated with facial fractures and has recently been described in a sole case report in a dog.⁴ Thus far, it has not been documented in a horse. The current case report aims to describe the perianaesthetic management of a horse that developed asystole due to a suspected TCR of either OCR or MCR in origin, in combination with hyperkalaemia of unknown cause which may or

may not have contributed to the suspected vagal event, a relationship that has been previously documented.^{5,6} Both causes will be discussed as independent or contributory factors.

2 | CASE PRESENTATION

A 10-year-old, 466 kg, mustang, gelding presented to a European equine referral hospital for surgical repair of nasal, frontal and lacrimal bone fractures from an unknown trauma sustained in the field 6 days previously. Computed tomography (CT) had been performed the preceding day under general anaesthesia uneventfully. The anaesthetic protocol prior to CT was identical to that used for the surgical repair, without the use of a maxillary loco-regional

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technique or lidocaine continuous rate infusion. Blood gas analysis was not performed throughout this anaesthesia due to the short duration of anaesthesia (20 min) and lack of arterial catheterisation. General anaesthesia was elected for surgical repair. Clinical examination prior to general anaesthesia was otherwise unremarkable (heart rate [HR]-36 bpm, respiratory rate [RR]-8 bpm, temperature-37.6°C). Pre-anaesthetic haematology and biochemistry revealed a mild leukopenia (3910/ μ L; ref 5000-10 000/ μ L), mild hypokalaemia (3.4 mmol/L; ref 3.5-5.1 mmol/L) and elevated serum amyloid A (720.8 mg/L; ref <10 mg/L) consistent with recent trauma and a 4-hour period of starvation with ad libitum water prior to general anaesthesia.

3 | ANAESTHETIC MANAGEMENT

A 12G intravenous catheter had been pre-placed in the right jugular vein. Pre-anaesthetic medication consisted of acepromazine (0.03 mg/kg; intramuscularly (IM); Vanastress 10 mg/ml; Vana GmbH) 35 min before premedication with xylazine (0.6 mg/kg; intravenously (IV); Sedaxylan 20 mg/ml; Dechra Veterinary Products GmbH) and butorphanol (0.02 mg/kg; IV; Butomidor 10mg/ml; Richter Pharma AG) to good effect. Assisted wall induction of anaesthesia was performed with ketamine (2.2 mg/kg; IV; Narketan 100 mg/ml; Vetoquinol) and midazolam (0.1 mg/kg; IV; miDAZolam 5 mg/ml). Endotracheal intubation was performed with a 24-mm cuffed endotracheal tube when the horse was in lateral recumbency. The horse was shortly hoisted by all four feet and placed in left lateral recumbency. The endotracheal tube was attached to a large animal circle breathing system delivering isoflurane (1.1%-1.3%; Iso-Vet 1000 mg/g; Vana GmbH) vaporised in a mixture of medical air and oxygen (FiO₂-60%) adjusted to maintain adequate depth of anaesthesia, monitored by the absence of the perineal reflex

due to the inability to access the head of the horse perioperatively. The lungs of the horse were ventilated throughout the anaesthetic (HorseVent; 6-8 bpm; tidal volume 15 ml/kg; peak inspiratory pressure 22-30 cmH₂O; positive end-expiratory pressure 3-5 cmH₂O; to maintain normocapnia [EtCO₂ 35-45 mmHg]). Continuous rate infusions (CRIs) of ketamine (1.2 mg/kg/h), midazolam (0.02 mg/kg/h) and xylazine (0.3 mg/kg/h) were infused IV combined as a triple drip in one CRI as per the hospital protocol. Lidocaine (1.8 mg/kg/h) was infused IV for further analgesia with a 1 mg/kg initial loading bolus. Crystalloid therapy (5 ml/kg/h; lactated Ringer's solution; Vetfundin; Bad Ischl Apotheke) was infused IV. Instrumentation included electrocardiography (ECG), pulse oximetry (SpO₂), capnography (EtCO₂), invasive arterial blood pressure monitored from a 20G catheter placed in the right metatarsal artery, arterial blood gas analysis, temperature, inspired and end-tidal isoflurane concentration using a multi-parameter monitor (Carescape Monitor B650; GE Healthcare). The horse was prepared for surgery and a right-sided maxillary loco-regional anaesthetic technique⁷ with mepivacaine hydrochloride (10 ml; 0.02 ml/kg; 0.43 mg/kg; Mepinaest Purum 20 mg/ml; Gebro Pharma) was performed by blindly inserting a 3.5-inch spinal needle into the pterygopalatine fossa.

The first hour of anaesthesia was uneventful- cardiovascular parameters were stable (mean arterial blood pressure [MAP] 80-90 mmHg; heart rate [HR] 25-29 bpm). Arterial blood gas analysis 30 mins after induction (Table 1; T-107 mins) revealed adequate oxygen tension (FiO₂- 61%; PaO₂; 363 mmHg) and mild hyperglycaemia (glucose 8.3 mmol/L; ref 4-5.6 mmol/L). Potassium concentration was within normal limits (4.6 mmol/L; ref 3.5-5.1 mmol/L). Subsequent arterial blood gas analysis (Table 1; T-51 mins) performed 56 min later, revealed potassium concentration had increased marginally (4.8 mmol/L) and increased hyperglycaemia (glucose 13.6 mmol/L). It was anticipated there was approximately 30 min before the end of surgery; therefore, the lidocaine infusion

TABLE 1 Perianaesthetic blood gas analysis of a mustang gelding presenting for surgical repair of nasal, frontal and lacrimal bone fractures

Time before asystole/Parameter (reference range)	T-107 mins	T-51 mins	T-3 mins	T+19 mins	T+31 mins	T+50 mins
Arterial pH (7.35-7.45)	7.48	7.46	7.41	7.37	7.37	7.40
Arterial partial pressure of carbon dioxide (32-48 mmHg)	36.4	41.2	50.9	57.8	48.3	32.1
Arterial partial pressure of oxygen (83-108 mmHg)	362.8	324.4	313.6	311.7	261.6	269
Bicarbonate (22-29 mmol/L)	26.8	28.3	31.3	32.8	27.5	19.3
Base excess	3.4	4.1	5.3	5.2	1.5	-4.4
Sodium (136-145 mmol/L)	133.9	134.5	132.4	134.2	133.8	139.4
Potassium (3.5-5.1 mmol/L)	4.6	4.8	6.4	5.7	4.1	2.9
Calcium ⁺ (1.15-1.33 mmol/L)	1.5	1.5	1.5	1.5	1.4	1.2
Chloride (98-107 mmol/L)	103.5	103.2	100	98.8	101.9	112.3
Glucose (4-6.9 mmol/L)	8.3	13.5	16.6	19.5	20.4	14.7
Lactate (1.0-1.8 mmol/L)	1.1	1.3	1.6	1.6	2.2	2.0

was discontinued in order to avoid ataxia in recovery. Mean arterial pressure dropped acutely to 51 mmHg and a dobutamine (Dobutamin 12.5 mg/ml; ERWO Pharma) infusion was started at an incorrect dose of 0.5 mg/kg/h, instead of 0.5 mcg/kg/min, equivalent to 8.3 mcg/kg/min. A 5-10 s period of no ECG or blood pressure trace was observed approximately one minute after the start of the dobutamine infusion, assumed at the time to be artefactual or machine error. Blood pressure increased acutely (HR-55; MAP-120 mmHg) and the dobutamine infusion was stopped within 5 min of it being commenced as the incorrect dosage had been noted. Cardiovascular parameters normalised within 10 min of stopping the dobutamine infusion, with a decrease in both HR and MAP (HR-30; MAP-90 mmHg). Anaesthesia continued to be stable for a further 20 min, with blood pressure gradually decreasing (MAP-65 mmHg), thought to be the effects of dobutamine decreasing. A third arterial blood sample taken 134 min after induction (T-3mins) revealed potassium concentration was 6.4 mmol/L. Crystalloid therapy was changed from lactated Ringers solution to sodium chloride (NaCl 0.9% Fresenius, Spullösung; Fresenius Kabi). Mean arterial pressure continued to decrease alongside concurrent bradycardia (HR-24; MAP-60 mmHg), with a poor arterial waveform. Asystole (T) occurred 3 min after the third arterial blood sample. Surgical stimulus at the time of asystole consisted of twisting of cerclage wire placed in the nasal and frontal bone fragments to reposition the bone fragments. The inhalational agent was discontinued and mechanical ventilation was continued. The surgeon was asked to refrain from applying any further surgical stimulus. Chest compressions were initiated by an 85 kg person dropping on to their knees from a standing position behind the horse's elbow; atropine (0.005 mg/kg; Atropinum Sulfuricum 0.5 mg/ml; Takeda) and adrenaline (0.01 mg/kg; Supranenin 1 mg/ml; Sanofi) were administered IV followed by IV crystalloid therapy. Spontaneous circulation returned (return of arterial waveform; heartbeat auscultated; EtCO₂-37 mmHg) within 2 min of CPR commencing (1 cycle). Atrial fibrillation was noted. Treatment for hyperkalaemia was initiated with insulin (0.4 IU/kg IV), glucose (3.2 ml/kg 5% glucose over 30 min; IV; Landes Apotheke) and prednisolone (0.2 mg/kg IV; Prednisolut 1000 mg). The horse returned to sinus rhythm. Serial blood gas measurements (Table 1; T +19 mins, T + 31 mins, T +50 mins) were performed under general anaesthesia over the following 60 min with potassium reducing to 2.9 mmol/L in response to therapy. The horse was transferred to the recovery box via a hoist, and recovered uneventfully from 190 min of anaesthesia via rope assistance, standing on the first attempt after 30 min in recovery. Post-operatively, the horse received flunixin meglumine (1.1 mg/kg IV BID; Niglumine 50 mg/ml; Laboratorios Calier), lidocaine (1.8 mg/kg/h CRI; lidocainhydrochlorid; Bad Ischl Apotheke) penicillin (20 000 IU/kg; slow IV BID, penicillin G-natrium; Sandoz GmbH) gentamycin (6.6 mg/kg SID IV, Gentavan 50 mg/ml; Vana GmbH), sodium chloride (NaCl 0.9% Fresenius, Spullösung; Fresenius Kabi) IV fluid therapy (2 ml/kg/h). Venous blood gas analysis 1 h after recovery showed potassium to be within normal limits (3.6 mmol/L). Over the following

days, serial venous blood gas analysis measurements were performed and potassium remained within normal limits. The horse was sedated 2 days following general anaesthesia uneventfully. The horse was readmitted 2 months after discharge for treatment of sequestration and was sedated on multiple occasions uneventfully. On both occasions, the sedation protocol consisted of detomidine (0.02 mg/kg; IV; Equidor 10 mg/ml, Richter Pharma AG) and butorphanol (0.02 mg/kg; IV; Butomidol 10 mg/ml; Richter Pharma AG). Testing for hyperkalaemic periodic paralysis (HYPP) and glycogen synthase 1 gene (GYS1) were negative.

4 | DISCUSSION

4.1 | Trigemino-cardiac reflex

The trigemino-cardiac reflex (TCR) is a well-documented vagal reflex in human medicine, particularly in maxillofacial surgeries^{8,9} and is defined as a decrease of at least 20% in heart rate and blood pressure upon surgical manipulation at or in the vicinity of any branch of the trigeminal nerve.¹⁰ The trigeminal nerve provides sensory innervation to the equine head via the ophthalmic and maxillary nerves, and mixed sensory and motor innervation via the mandibular nerve.¹¹ The ophthalmic nerve divides into the frontal, lacrimal and nasociliary nerves, providing sensory innervation to the eyelids and forehead.¹¹ The maxillary nerve provides sensory innervation to ipsilateral cheek teeth, paranasal sinuses and the nasal cavity.¹¹ The zygomatic branch of the maxillary nerve does also provide sensory innervation to the lower eyelid.¹¹

Four subtypes of a TCR exist- a central TCR involving intracranial stimulation of the trigeminal nerve, and two peripheral subtypes; the oculo-cardiac reflex (OCR) and the maxillomandibulocardiac reflex (MCR), referring to the involved branches of the trigeminal nerve.¹² Stimulation of afferent trigeminal nerve sensory fibres at their respective branch generate action potentials through the Gasserian ganglion to the trigeminal nerve sensory nucleus on the floor of the fourth ventricle. This action potential continues to the efferent motor nucleus of the vagus nerve via internuncial nerve fibres of the reticular formation.¹⁰ Motor fibres terminate in the myocardium causing negative inotrope and chronotrope, resulting in bradycardia, arrhythmias, hypotension and asystole. Other possible autonomic reactions include apnoea and gastric hypermotility. The fourth TCR subtype, known as the Gasserian ganglion subtype involves direct stimulation around the ganglion and differs from the other TCRs in that it can present as either parasympathetic or sympathetic stimulation.¹³

The OCR was first documented in human medicine in 1908¹⁴ and is widely documented in veterinary medicine, particularly in ophthalmic surgery.¹⁵ A human descriptive case series evaluating the TCR in patients with facial fractures found all patients with midfacial fractures exhibited bradycardia perioperatively, due to stimulation of the maxillary nerve and subsequent suspected TCR.⁸ The same study reported asystole associated with zygomatic fracture

manipulation, which has previously been documented in human medical literature.¹⁶

At the time of asystole, cerclage wire fixating the frontal and nasal bone fractures was being tightened, which may have caused direct traction of a trigeminal nerve branch, leading to a TCR. Mechanical stretch is reported in human medical literature to be the most powerful predisposing factor in the generation of a TCR.¹⁰ Other potential stimuli include pressure on the orbit or muscles of the eyelid by the surgeon, stimulating the sensory ophthalmic nerve leading to an OCR. If the TCR was of MCR origin, local anaesthetic techniques as used in the present case could have prevented the TCR from occurring due to the sensory blockade of the maxillary nerve, although human literature reports local anaesthetic techniques do not always prevent TCR episodes and stretch of an anaesthetised nerve can still provoke a TCR.¹⁰ This could be due to differential blockade first described in 1929,¹⁷ where different nerve fibres are selectively blocked by local anaesthetics dependent on numerous factors including nerve diameter and local anaesthetic used. The maxillary nerve was anaesthetised at the pterygopalatine fossa, providing anaesthesia to the cheek teeth, paranasal sinuses (including frontal and nasal bones) and nasal cavity.^{7,11} If the MCR was of zygomatic nerve origin, which leaves the maxillary nerve caudal to the site of local anaesthetic deposition, the block may still have been efficacious but the reflex still of MCR origin.¹¹

Mepivacaine is reported to have a duration of action of 90–180 min in the horse.¹⁸ Asystole occurred 95 min following the maxillary nerve block; therefore, it is plausible that asystole occurred due to sensory blockade waning and a MCR occurred. Assessing block efficacy or waning of the blockade was challenging due to the administration of other analgesics as continuous rate infusions alongside the inhalational agent, as is the protocol in the referral hospital. Alternatively, the maxillary nerve block may still have been efficacious and the vagal reflex was of OCR origin. The ethmoidal nerve, a branch of the nasociliary nerve and, therefore, branch of the ophthalmic nerve also provides sensory innervation to some sinus structures and could have been stimulated by tightening of the cerclage wire. A local anaesthetic approach to the ethmoidal nerve has been described in response to the experience of incomplete anaesthesia of the paranasal sinuses with a maxillary nerve block alone.¹⁹ An ethmoidal nerve block alongside a maxillary block is thought to fully anaesthetise the ipsilateral sinus structures and may have been beneficial in the present case depending on the origins of the observed TCR, as well as for additional analgesia.

4.2 | Hyperkalaemia

Hypokalaemia is commonly encountered in horses under general anaesthesia, primarily those with abdominal disease due to reduced gastrointestinal absorption, which is the principal source of potassium in the horse.²⁰ Hyperkalaemia is much less commonly encountered, but can be life-threatening. There are three primary causes for hyperkalaemia in horses²¹: (i) Trans-cellular movement – hyperkalemic periodic paralysis; acidosis; cell lysis (rhabdomyolysis,

haemolysis, hyperthermia induced); increased metabolic rate; insulin deficiency, drug-induced (beta antagonists, cardiac glycosides); (ii) Decreased urinary excretion- uroperitoneum, anuric or oliguric renal failure, drugs (NSAIDs, alpha-2 agonists, trimethoprim) and (iii) Increased intake – iatrogenic intravenous administration.

The most common findings of hyperkalaemia under anaesthesia are due to changes in cell membrane excitability and include arrhythmias (bradycardia, altered ECG morphology (flattened P waves, narrow T waves of increased amplitude, prolonged QRS complex)) due to prolonged depolarisation and repolarisation of the myocardium,²² slowing the rate of cardiac pacemaker discharge.²³ Early experimental studies inducing hyperkalaemia in horses found ECG changes to be progressive according to the degree of hyperkalaemia and speed of potassium administration, with rapid administration having more marked effects on the ECG.^{24,25} Although P wave changes occurred with plasma concentrations over 6.2mmol/L, asystole did not occur until plasma potassium was over 9.4 mmol/L.²⁴ In the case reported here, HR was consistently 22–29 bpm; however, this was thought to be related to the administration of xylazine as a continuous rate infusion and not necessarily a manifestation of hyperkalaemia. No abnormalities on the ECG were detected, however, it is documented that ECG changes are not always present in cases of hyperkalaemia.^{26,27}

There are numerous reports of confirmed HYPP in the veterinary literature, including case reports of peri-anaesthetic management.^{28,29} HYPP was first identified in 1985 in quarter horse breeds related to the stallion, impressive. Clinical signs in conscious patients include muscle fasciculations and weakness, involuntary recumbency, with a report of lingual swelling due to suspected myotonia,³⁰ 1994), none of which were reported in the history of this horse. Intraoperative findings of HYPP are varied in the previously mentioned case reports, ECG changes became apparent above 7.2 mmol/L but other signs of HYPP were noted prior to this such as muscle fasciculations, hypercapnia and respiratory acidosis. Other findings have included sinus tachycardia and hyperthermia. None of these findings besides hyperkalaemia was observed in the present case report, and testing for HYPP was negative.

The GYS1 gene has been identified in the mustang breed in relation to a subset of horses affected by polysaccharide storage myopathy (EPSSM), although horses can be negative for the gene and still be affected by EPSSM.³¹ Creatine kinase activity can be increased in EPSSM-affected horses and was moderately elevated the day following surgery in the present case (Table 2), with AST being within

TABLE 2 Pre- and post-operative blood biochemistry results from a mustang gelding presenting for surgical repair of nasal, frontal and lacrimal bone fractures

Parameter (reference range)	Preoperatively	Day following surgery
Creatinine Kinase (<200 U/L)	229	1115
Aspartate aminotransferase <550 U/L	Not obtained	481
Serum Amyloid A <10 mg/L	720.8	405.8

normal limits. These findings are consistent with the previous study findings of expected biochemical changes associated with anaesthesia in the horse.³² Furthermore, a case report of post anaesthesia recumbency in a horse confirmed with EPSSM documented normal intraoperative potassium concentration.³³ The current case recovered from anaesthesia uneventfully and testing for the GYS1 gene was negative. Muscle biopsy was not performed as clinical suspicion of EPSSM was low.

There have been multiple case reports attributing intraoperative hyperkalaemia in large non-domesticated felids to the administration of alpha-2 agonists,³⁴⁻³⁶ which induce hyperglycaemia by inhibiting insulin secretion from pancreatic beta cells. Furthermore, hyperglycaemia as a stress response to surgery has been shown in both human and equine patients.^{37,38} Both hyperglycaemia and insulin deficiency can cause hyperkalaemia by cellular water loss and decreased cellular uptake of potassium, respectively,²² which has been documented in human medicine.³⁹ Xylazine as both a bolus and continuous rate infusion as used in the case presented causes hyperglycaemia in horses,⁴⁰⁻⁴² however, these studies did not document concurrent hyperkalaemia. Perioperative hyperglycaemia was documented in the present case, and further glucose was administered after asystole as an emergency treatment for hyperkalaemia alongside insulin, which may or may not have been the correct approach given the hyperglycaemia. Further studies in equine patients are needed to draw definitive conclusions regarding the relationship between hyperglycaemia, hyperkalaemia and alpha-2 agonist administration.

Vasopressors and inotropes used in the current case included dobutamine, atropine and adrenaline. Dobutamine has primarily beta-1 adrenergic activity, with some alpha-1 and beta-2 adrenergic activity at higher doses (>7.5 µg/kg/min), however, the alpha-1 mediated vasoconstriction is antagonised by the vasodilatory beta-2 effect.^{43,44} Thirty-five minutes prior to asystole, MAP dropped acutely from 80 mmHg to 51 mmHg and a dobutamine infusion was started at an incorrect dose of 8.3 µg/kg/min resulting in an increase in HR to 55bpm and MAP of 120 mmHg. Equine studies assessing haemodynamic changes associated with dobutamine infusions used doses up to 10 µg/kg/min although cardiac arrhythmias were sometimes noted at higher doses.⁴⁵⁻⁴⁷ Veterinary texts suggest doses up to 5 µg/kg/min for horses and doses up to 20µg/kg/min for small animal patients.⁴⁸⁻⁵⁰ It is possible that this initial drop in MAP was in fact also a vagal event which resolved with the adrenergic action of dobutamine, and when the dobutamine infusion was stopped, the parasympathometric effects predominated, leading to asystole. Stimulation of alpha receptors causes a transient increase in plasma potassium concentration due to activation of calcium-dependent potassium channels, whereas activation of beta receptors induces a decrease in plasma potassium concentration due to activation of sodium-potassium-ATPase in skeletal muscle.⁵¹ For this reason, beta-agonists are used in human medicine to treat hyperkalaemia.⁵² Therefore, it is possible that the dobutamine infusion affected plasma potassium concentrations as the higher infusion rate may have preferentially stimulated alpha receptors causing an increase in plasma potassium concentration, however, the extent of this effect is unknown.

In metabolic or respiratory acidosis, potassium moves from the intracellular space into the extracellular space in exchange for hydrogen to maintain electroneutrality; therefore, increasing plasma potassium levels.²¹ In the case reported here, acidosis was not documented on any blood gas analysis prior to asystole. Extensive and invasive debridement occurred throughout surgery, which in theory may have released potassium due to cellular damage, as in cases of rhabdomyolysis,²⁷ however, this would not be sufficient to markedly increase plasma potassium concentrations. Studies have found increases in serum potassium concentration to be variable in cases of rhabdomyolysis, with hyperkalaemia not consistently documented, concluding that renal function is assessed in horses with rhabdomyolysis before attributing any hyperkalaemia to solely rhabdomyolysis.⁵³⁻⁵⁵ Urinary output was adequate both throughout anaesthesia and post-operatively in the case reported, and haematology and biochemistry were not supportive of any haemolysis or renal insufficiency. Therefore, hyperkalaemia in the case reported was unlikely related to rhabdomyolysis or decreased urinary excretion of potassium.

Rapid intravenous administration of potassium is another possible cause of hyperkalaemia. Physiological lactated Ringers solution (4mEq/L potassium) was administered at 5 ml/kg/h throughout anaesthesia, a rate insufficient to induce significant increases in plasma potassium.⁵⁶ Crystalloid therapy was changed to sodium chloride (0.9%) as soon as hyperkalaemia was documented so as to avoid any additional potassium entering the circulation, although this approach to hyperkalaemia has been questioned in cats with urinary obstruction, with studies finding lactated Ringers solution better at restoring acid-base balance, due to the presence of a lactate buffer which is metabolised to bicarbonate and has an alkalinising effect.⁵⁷ Comparatively, the administration of sodium chloride (0.9%) is associated with hyperchloraemic metabolic acidosis, which may lead to worsening hyperkalaemia due to extracellular movement of potassium,⁵⁸ as previously discussed.

5 | CONCLUSION

After considering the literature, it was concluded that the most likely primary cause for asystole was a trigemino-cardiac reflex (TCR), the origins of which could be a maxillomandibulocardiac reflex (MCR) which has not previously been documented in the horse. This may or may not have been influenced by the hyperkalaemia affecting myocardial susceptibility to the vagal event, which has been previously reported.^{5,6} The horse responded quickly to vagolytic agents (dobutamine, adrenaline, atropine) and return of spontaneous circulation occurred before any significant changes in plasma potassium concentrations occurred, as documented by the potassium still being elevated 19 min following asystole (Table 1), suggesting a primarily vagal component to asystole as opposed to hyperkalaemia. Furthermore, previous reported cases of hyperkalaemia under anaesthesia document clinical signs at higher plasma potassium concentrations than those documented in the present case report and ECG changes prior to cardiac arrest. Therefore, due to the comparatively mild increase in

plasma potassium concentration (6.4 mmol/L), it was concluded that hyperkalaemia was unlikely to be the cause of asystole, and a vagal reflex of a subtype of TCR, an MCR was presumed.

ETHICAL ANIMAL RESEARCH

Research ethics committee oversight not required by this journal: a retrospective study of clinical records.

INFORMED CONSENT

Explicit owner-informed consent for the inclusion of animals in this study was not stated.

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CONFLICT OF INTERESTS

No competing interests have been declared.

AUTHOR CONTRIBUTIONS

A. Ryan was responsible for manuscript preparation. M. Gurney and R. Steinbacher were responsible for manuscript review.

PEER REVIEW

The peer review history for this article is available at <https://publons.com/publon/10.1111/evj.13535>.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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