




CASE REPORT

Horses and other equids

Suspected severe post-anaesthetic myopathy or myelopathy in a Clydesdale horse resulting in euthanasia

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Abstract

This case report describes suspected myopathy or myelopathy in a 5-year-old Clydesdale gelding following general anaesthesia for sarcoid removal. The lowest mean arterial pressure was 67 mmHg. Hyperlactataemia and tachycardia were observed during anaesthesia prompting abortion of surgery. The horse was unable to stand with assistance from a specialised sling. Azotaemia and hyperkalaemia developed in recovery and worsened despite therapeutic interventions. Euthanasia was performed given the grave prognosis. Postmortem examination was not carried out but could have provided a definitive diagnosis. Specific factors have been identified in the prevention of post-anaesthetic myopathy, including maintenance of adequate tissue perfusion and oxygenation and careful positioning. Potential improvements in the anaesthetic management of this case in relation to these factors are discussed.

KEYWORDS

anaesthesia, horses, neuromuscular disorders

BACKGROUND

General anaesthesia in horses carries a relatively high risk of morbidity and mortality. Preliminary results from an ongoing investigation report a peri-operative mortality rate of 0.6% for anaesthetised horses not undergoing colic surgery.¹ A historical iteration of this study reported an equivalent mortality rate of 0.9%.²

Myopathy is a notable complication of general anaesthesia in horses, accounting for 7% of all deaths in a non-emergent population.² It is often associated with intraoperative hypotension or hypoxaemia.^{3,4} Post-anaesthetic myelopathy is comparably rare, although likely under-reported. There is a lack of research into myelopathy, but a higher incidence has been shown in larger framed individuals positioned in dorsal recumbency.⁵

Increasing plasma lactate concentration can be a result of tissue hypoperfusion which is implicated in muscle damage, but it is not considered a pathognomonic sign of myopathy or myelopathy. Abnormal lactate values are not always reported in horses succumbing to these conditions.

Inadequate perfusion of the dorsal musculature and/or the spinal cord is likely to have occurred in this horse. Several features of this anaesthetic, including low intravenous fluid administration, potentially excessive anaesthetic depth and

delayed recognition of developing pathology could have contributed to the period of inadequate perfusion. Identification of and reflection on problems in equine anaesthetic practice is vital to ensure improvements in patient safety.

Use of a specialised sling to achieve a standing position in this horse is a notable feature of the management protocol. Employment of mobility aids in horses has important practical and safety implications.

CASE PRESENTATION

A 5-year-old male Clydesdale horse, weighing 725 kg (body condition score 4/9), presented for surgical excision of inguinal, axillary and preputial sarcoids. Endoscopic exploration of the prepuce and penis was also scheduled due to incomplete exteriorisation of the penis during urination and an inability to fully extrude the penis under sedation. Male pseudohermaphroditism was suspected due to measurement of anti-Müllerian hormone suggesting gelded status (0.1 ng/ml), despite no record of castration and absent testicles. The horse had an amenable but nervous temperament and was deemed healthy on pre-anaesthetic clinical examination; no blood tests were performed. The pre-anaesthetic heart rate was 44 bpm, suspected to be mildly elevated due to anxi-

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ety. No abnormal heart sounds or arrhythmias were detected. The respiratory rate was 20 bpm and oral mucous membranes were pink and moist. Hydration status was adequate.

The horse was restricted from hay for 12 hours and fed a small soaked forage-based feed 3 hours before general anaesthesia. Water was freely available until pre-medication. Acepromazine 0.02 mg/kg IV and flunixin 1.1 mg/kg intravenously (IV) were administered, and penicillin 20 mg/kg was given intramuscularly 40 minutes before induction of anaesthesia. A 14 Ga, 9 cm cannula was inserted aseptically into the left external jugular vein. Romifidine 0.04 mg/kg IV followed by morphine 0.2 mg/kg IV, once sedation was apparent, were given 10 minutes before induction with ketamine 2.2 mg/kg and diazepam 0.05 mg/kg IV. Anaesthetic induction was performed with the use of a swing-gate in a padded induction box and was uneventful. The trachea was intubated with a 30-mm (internal diameter) cuffed endotracheal tube with the horse in right lateral recumbency. The horse was hoisted via fabric hobbles placed around each limb and lowered onto the surgery table into dorsal recumbency atop a deeply padded foam surface with an uncompressed depth of 15 cm (Karomed, UK). Care was taken to ensure even distribution of weight across the body surfaces in contact with the padding. The head and neck were elevated from the horizontal plane at approximately 30°. The lungs were mechanically ventilated via a large animal circle system (Mallard Medical, USA) with the following settings: tidal volume 8 L, respiratory rate 10 bpm and peak inspiratory pressure 20 cm H₂O. Anaesthesia was maintained with isoflurane in oxygen at an end-tidal concentration (ET_{iso}) of 1.4%–1.5%, targeting absence of spontaneous movement, an absent or 'sluggish' palpebral reflex and relaxed cervical musculature. Fresh gas flow was 6 L/min of oxygen. Hartmann's solution was infused freely under gravity through a standard giving set (20 drops per ml) from the beginning of isoflurane anaesthesia. A romifidine infusion was administered at 0.03 mg/kg/h 5 minutes after the commencement of isoflurane anaesthesia. A 20 G cannula was inserted into the left mandibular artery for continuous measurement of invasive blood pressure and intermittent monitoring of arterial blood gases. Dobutamine was initially administered at 0.5 µg/kg/min from the beginning of isoflurane anaesthesia in an attempt to ensure adequate mean arterial pressure (MAP) before obtaining invasive blood pressure measurements. It was discontinued after 35 minutes due to consistent blood pressure readings >67 mmHg. Physiological variables were acceptable for the first 60 minutes of anaesthesia (heart rate 32–40 bpm, PE'CO₂ 4.2–4.6 kPa, MAP 67–80 mmHg).

Analysis of an arterial blood sample 50 minutes into anaesthesia revealed acceptable ventilation and oxygenation and normal electrolyte status (see Table 1). Lactate was not measured. A second arterial sample taken 105 minutes after induction revealed hyperlactataemia (6.04 mmol/L) with normal PaCO₂ and oxygenation. Twenty minutes after this result, the horse's heart rate increased from 35 to 55 bpm over 5 minutes; other physiological variables remained unchanged. Lactate had increased to 10.21 mmol/L, 135 minutes after induction.

Surgery was aborted after the second lactate result due to high suspicion of myopathy. Total anaesthesia time was 170 minutes and total fluid administration (Hartmann's) was 5 L (2.5 ml/kg/h). Mechanical ventilation, isoflurane and romifidine were discontinued. The horse was transported to the recovery box via hoist and placed in right lateral recum-

LEARNING POINTS/TAKE-HOME MESSAGES

- Post-anaesthetic myopathy can occur in healthy horses undergoing general anaesthesia.
- Maintenance of adequate tissue perfusion and oxygenation is essential during general anaesthesia to reduce the risk of myopathy and myelopathy—adequate mean arterial pressure (MAP) is essential but interventions such as deep padding of surfaces, careful positioning, appropriate intravenous fluid therapy and avoidance of excessive anaesthetic depth are also important.
- Development of hyperlactataemia under anaesthesia (>3.0 mmol/L) is a cause for concern and action should be taken to investigate and identify the problem immediately.
- A pre-anaesthetic 'minimum database' comprising packed cell volume (PCV), total solids and renal parameters may be of value in horses deemed at high risk of peri-anaesthetic complications.
- Specialised slings can be employed in the management of recumbent horses to achieve a standing position.

bency. A 14-mm (internal diameter) nasopharyngeal tube was placed through the right nostril and the horse's trachea was extubated following resumption of spontaneous breathing. Sedation for the immediate recovery period was provided by xylazine 0.2 mg/kg IV, administered at the appearance of nystagmus. Morphine 0.2 mg/kg IV was also given at this time to provide analgesia in case of myopathy. Oxygen was insufflated at 15 L/min via tubing inserted into the left ventral nasal meatus. A venous blood sample at this time revealed a packed cell volume of 36% and a total protein of 68 g/L. A 2.5 L bolus of Hartmann's solution was administered intravenously over 20 min to augment circulating volume.

Voluntary movement of the head, neck and forelimbs occurred 25 minutes after discontinuation of isoflurane anaesthesia. No movement occurred in the hindlimbs. The horse became distressed with erratic head movements. Romifidine 0.02 mg/kg IV repeated twice at a 5-minute interval had no significant effect. Further sedation was administered with detomidine 0.007 mg/kg IV 5 minutes later and this was repeated after another 5 minutes with a moderate effect: the horse rested in lateral recumbency with occasional head movements. The pulse rate was 60 bpm and the respiratory rate was 22 bpm. Nociceptive testing of the hind feet, consisting of firm pinching of the fetlock skin with artery forceps, was inconclusive due to intermittent movement of the head and neck. Withdrawal reflexes were absent, but anal tone was present. Swelling and hardening of the epaxial and gluteal musculature was evident bilaterally (see Figure 1).

INVESTIGATIONS

Investigations were performed through repeated clinical examination and biochemical testing in the recovery period (see Table 1).

TABLE 1 Blood gas, electrolyte, biochemical and haematological results at different stages of case progression (blank boxes indicate test not performed at specific time point)

Time from first induction min	Period	Arterial/venous	Blood gases					Electrolytes					Biochemistry					
			pH	PO ₂ mmHg	PCO ₂ mmHg	HCO ₃ ⁻ mmol/L	BE	Na ⁺ mmol/L	K ⁺ mmol/L	iCa ²⁺ mmol/L	Glucose mmol/L	Lactate mmol/L	BUN mmol/L	Creatinine mmol/L	CK U/L	PCV%	TP g/dl	Hb g/dl
	Reference range		7.35–7.45 ^a	90–110 ^a	36–46 ^a	25–30 ^a	-5 – +5 ^a	126–146 ^b	2.5–5.2 ^b	1.25–1.75 ^b	3.6–6.1 ^b	0.3–1.5 ^a	2.5–8.9 ^b	53–194 ^b	120–470 ^b	30–45 ^a	57–80 ^a	12–17 ^b
50	Intraoperative	Arterial	7.404	358	45.5	28.4	4	133	3.5	1.52	8.2				25.0		8.5	
110	Intraoperative	Arterial	7.390	388	43.0	26	1				6.04							
135	Intraoperative	Arterial	7.267	379	48.8	22.3	-5				10.21							
170	Recovery (following surgery)	Venous													36.0	68.0		
290	Recovery (following sling application)	Venous	7.261	29	44.2	19.9	-7				14.19							
310	Recovery (following first sling raise attempt)	Venous	7.164	24	53.6	19.3	-9	131	5.1	1.14	7.5				49.0		16.7	
410	Recovery (following second sling raise attempt)	Venous						135	6.8	Total calcium 3.25	7.3		315	>470 (off-scale high)				

Abbreviation: CK; creatine kinase; Hb, haemoglobin; PCV, packed cell volume.

^a Vetscan VS2 Biochemical Analyser, Zoetis Animal Health, <https://vet.abaxis.co.uk/vs2-support/>.

^b Vetscan ISTAT1 Critical Care Analyser, Zoetis Animal Health, <https://vet.abaxis.co.uk/istat-support/>.

Case Timeline

- 08.50** – Pre-medication (acepromazine)
- 09.20** – Pre-medication (romifidine and morphine)
- 09.30** – First induction (ketamine and diazepam)
- 10.20** – First intra-operative blood test performed (arterial): blood gases & electrolytes
- 11.20** – Second intra-operative blood test performed (arterial): blood gases & lactate; hyperlactataemia revealed
- 11.45** – Third intra-operative blood test performed (arterial): blood gases & lactate; hyperlactataemia worsening
- 12.15** – End of surgery
- 12.20** – End of general anaesthesia; horse hoisted to recovery box and positioned in right lateral recumbency
- 12.30** – Extubation; xylazine & morphine administered
- 13.15** – Signs of distress; romifidine administered
- 13.20** – Repeat romifidine
- 13.25** – Detomidine administered
- 13.55** – Second induction (ketamine and diazepam); horse hoisted & sling applied
- 14.05** – Horse positioned in right lateral recumbency; xylazine administered
- 14.20** – Blood test performed: blood gases & lactate; hyperlactataemia worsening (venous)
- 14.40** – Blood test performed: blood gases & electrolytes; hypocalcaemia & hyperkalaemia revealed; calcium supplementation with IVFT
- 15.00** – Horse hoisted via sling into standing position; horse unable to bear weight on limbs
- 15.25** – Horse lowered into right lateral recumbency; flunixin administered
- 16.10** – Horse hoisted via sling into standing position for second time; horse unable to bear weight on limbs
- 16.20** – Blood test performed: electrolytes & biochemistry; azotaemia, worsening hyperkalaemia & high creatine kinase revealed
- 16.28** – Horse lowered into sternal position
- 17.10** – Euthanasia

FIGURE 1 Timeline of case detailing important developments

DIFFERENTIAL DIAGNOSIS

Post-anaesthetic myopathy
 Post-anaesthetic myelopathy
 Polysaccharide storage myopathy

TREATMENT

A second general anaesthetic was carried out 95 minutes after recovery, within the recovery box, to apply a suspension sling consisting of broad mesh abdominal panels and straps (Animal Rescue and Transportation Sling, Switzerland) to enable hoisting of the horse into a standing position. Ketamine 2.2 mg/kg and diazepam 0.05 mg/kg IV were administered. The horse was hoisted in dorsal recumbency to fit the sling and then lowered into right lateral recumbency. Sling application took 15 minutes; a further 0.7 mg/kg ketamine IV was administered to maintain anaesthesia. Endotracheal intubation was not performed. The horse breathed spontaneously without oxygen supplementation. Xylazine 0.3 mg/kg IV was administered to provide sedation during recovery. A venous blood sample retrieved after sling application revealed mild hypocalcaemia and hyperkalaemia (see Table 1). Calcium gluconate (1.2 g/L) was added to 5 L of Hartmann's solution and administered intravenously over 40 minutes.

The horse began moving the head, neck and forelimbs, and attempted to move into sternal recumbency 50 minutes after sling application. Absence of movement of the hindlimbs persisted. The horse was raised in the sling via the hoist to achieve

a standing position, but did not bear weight on the hindlimbs. The horse was lowered to the ground in the sling after approximately 10 minutes and straw bales were used to support it in sternal recumbency. Flunixin 1.1 mg/kg and Hartmann's solution at 5 ml/kg/h were administered. The environment was kept calm and quiet.

The horse was unable to bear weight on the hindlimbs during a second raising of the sling 65 minutes later; the horse was again lowered into sternal recumbency. A venous blood sample at this time revealed worsening hyperkalaemia and elevated creatinine and creatine kinase (CK) (see Table 1). The horse had not produced any urine since the beginning of the first general anaesthetic, with a total anuric period of 410 minutes. Catheterisation of the urinary bladder was impossible due to the inability to extrude the penis.

OUTCOME AND FOLLOW-UP

Due to the grave prognosis, euthanasia was performed 5 hours after the initial recovery from general anaesthesia with 28 g quinalbarbitone sodium and 17.5 g cinchocaine hydrochloride intravenously.

DISCUSSION

The outcome of this case unfortunately lacks a definitive diagnosis, as a postmortem examination was not carried out due to financial reasons. However, the clinical signs and

association with general anaesthesia make post-anaesthetic myopathy highly likely.

The main signs of pathology in this horse were hyperlactataemia, absence of movement of the hindlimbs and swollen gluteal and epaxial musculature. These clinical signs are highly suggestive of post-anaesthetic myopathy. Alternative aetiologies include myelopathy and polysaccharide storage myopathy (PSSM). Tachycardia under anaesthesia is a relatively rare event in horses, and usually reflects nociception, hypotension, hypercarbia or hypoxia.⁶ The presence of tachycardia, in combination with the hyperlactataemia, alerted the anaesthetist to a potential pathological process and prompted further investigation. Azotaemia could be explained by secondary acute renal injury from circulating myoglobin, pre-existing chronic kidney disease or dehydration.

Myelopathy or myelomalacia is considered in this horse due to the absence of withdrawal reflexes of the hindlimbs and the inability to confirm the presence of deep pain, but severe muscle pain and lack of muscular function, as would occur in myopathy, could also cause these signs. Additional findings expected in myelopathy include loss of anal tone and cutaneous trunci reflex from the caudal thorax distally.⁵ Regrettably, specific assessment of the cutaneous trunci reflex was not carried out and could have helped to refine the diagnosis. This, together with the presence of anal tone, makes it difficult to be confident of myelopathy in this horse, but it remains a plausible differential, especially because this is a progressive condition where individuals may possess anal tone or even the ability to stand initially.⁵

Myelopathy following general anaesthesia in the horse has been documented in case reports and series.^{5,7–13} The underlying pathology is ischaemic damage to the spinal cord and its blood vessels. There is a tendency for 'large-framed' breeds to be affected.⁵ Dorsal recumbency is also a common feature in horses with suspected spinal cord pathology or bilateral neuropathy.^{8–13} Subclinical hypovitaminosis E and selenium deficiency may also be implicated.⁵ Measurement of vitamin E levels could be considered on an individual basis in horses that may be predisposed to myelopathy.

PSSM describes an accumulation of glycogen in muscle tissue. Clinical signs include stiffness, lameness, sweating and muscle tremors. PSSM has been associated with Draught breeds¹⁴ and has been proven as a cause of post-anaesthetic myopathy.¹⁵ PSSM is a possible condition in this horse but was not considered due to an absence of clinical signs.

This horse was most likely suffering from severe post-anaesthetic myopathy, given the clinical findings and association with general anaesthesia. The term 'myopathy' describes muscle damage resulting from compromised perfusion and/or oxygenation.⁶ Myopathies typically cause painful, swollen and firm muscle tissue leading to lameness or complete disuse. Increased CK, as seen in this horse, is consistent with myopathy^{3,16} as it reflects muscle damage. However, elevations in CK have also been shown to occur normally following general anaesthesia, with increases to >5000 U/L at 4 hours post-anaesthetic.¹⁷

The pathophysiology underlying myopathy is inadequate muscle tissue perfusion and/or hypoxia.^{3,4} Muscle perfusion in the horse decreases under general anaesthesia.¹⁸ Reduced cardiac output, hypotension and/or high intracompartmental muscle pressure can all compromise muscle

perfusion. Multiple factors specific to the anaesthesia of this case could be implicated.

Tissue oxygen delivery (DO_2) is primarily dependent on the oxygen content of arterial blood (CaO_2) and perfusion pressure.¹⁹ CaO_2 is dictated by the partial pressure of oxygen in arterial blood (PaO_2 , mmHg), haemoglobin concentration ($[\text{Hb}]$, g/dl) and percentage saturation of haemoglobin with oxygen (SaO_2 , %) and is given by the following equation²⁰:

$$\text{CaO}_2 = (1.34 \times [\text{Hb}] \times \text{SaO}_2) + (0.003 \times \text{PaO}_2)$$

Using the results from the first arterial blood gas, the CaO_2 in this horse is 12.46 ml O_2 per 100 ml blood (see Table 1). An applicable reference range for CaO_2 is lacking in the literature, but calculating this value using data obtained from five dorsally recumbent isoflurane-anaesthetised horses, mechanically ventilated with an $\text{FiO}_2 > 95\%$,²¹ gives an approximate value of 18 ml O_2 per 100 ml blood. The relatively lower $[\text{Hb}]$ in this horse may be responsible for the lower CaO_2 , but lower haematocrit and $[\text{Hb}]$ values of this horse fall within the normal reference range for its breed type (24%–44% and 8.0–14.0 g/dl, respectively).²² The effect of anaesthetic drugs, such as acepromazine²³ and alpha-2 agonists,^{24,25} on lowering packed cell volume (PCV) has also been demonstrated. It is therefore impossible to know if the comparatively lower CaO_2 contributed significantly to inadequate DO_2 in this case. Sufficient tissue perfusion pressure is essential for the delivery of oxygenated blood to tissues. The role of inadequate perfusion in compromising DO_2 must be considered and, on balance, was probably the more relevant factor in this horse owing to several features of the peri-anaesthetic period.

The most common cause of reduced tissue perfusion during anaesthesia is hypotension. The lowest MAP recorded in this horse was 67 mmHg. The recommended minimum MAP for horses is contentious, with some reference texts advising 60–70 mmHg,⁶ and others recommending a minimum of 70 mmHg,²⁶ which has been associated with a reduced risk of myopathy.²⁷ However, although hypotension increases the risk of severe myopathy,²⁸ normotension does not guarantee adequate muscle perfusion.²⁹ This is because intracompartmental muscle pressures (ICMP) may exceed MAP, even in apparent normotension—ICMP is an opposing force to blood flow. Several studies have attempted to measure muscle pressure in anaesthetised horses,^{4,30–32} with results varying widely according to recumbency, muscle location and the type of surface the horse is in contact with, but typical ICMP in dependent muscles can reach 30–40 mmHg.^{12,26} It has been suggested that MAP should be at least 30 mmHg higher than ICMP to ensure adequate muscle perfusion.³³ It is possible that the MAP, despite being close to or within the accepted range for general anaesthesia,⁶ was not high enough to overcome the ICMP in the dependent musculature of this individual. 'Normotension' is therefore a context-dependant concept and, in this horse, a MAP of 70 mmHg may have been insufficient to maintain muscle perfusion. Excessive ICMP occurs because of the high body weight of the recumbent horse pressing onto muscle tissue and impaired venous drainage³⁰; consequent muscular swelling further increases ICMP.

The contribution of systemic vascular resistance (SVR) to maintaining normotension must also be considered. A high SVR due to vasoconstriction may produce a normal MAP while compromising tissue perfusion. The resultant increase

in afterload will decrease cardiac output, further hindering tissue perfusion. Insufficient tissue perfusion in the face of an apparently adequate MAP seems particularly possible in this case considering the intraoperative infusion of romifidine. Alpha-2 agonists decrease heart rate, stroke volume and consequently cardiac output,³⁴ increase SVR through vasoconstriction³⁵ and have been shown to reduce muscle perfusion.³⁶

Romifidine was chosen as an adjunct to inhalational anaesthesia because of its potential MAC-sparing effect.³⁷ It was rationalised that a lower ET_{iso} would improve blood pressure, as inhalant agents reduce cardiac contractility and heart rate in a dose-dependent manner.²⁶ Blood pressure is reduced by halo-ethers (isoflurane, sevoflurane) mostly through reducing SVR, whereas halo-alkanes (halothane) tend to have a more pronounced deleterious effect on cardiac contractility.³⁸ A blinded clinical trial in 30 horses undergoing arthroscopy failed to demonstrate any anaesthetic-sparing effect of romifidine at 0.04 mg/kg/h,³⁹ but a lower dose of 0.0003 mg/kg/h has been shown to reduce inhalant anaesthetic requirements in a separate study.³⁷ MAC-sparing effects have been shown for detomidine infusions,³⁵ and xylazine reduces MAC requirements by approximately 25%–30%.^{40,41} These contrasting findings could suggest that romifidine has an inferior sedative effect when administered as an infusion, so may not have reduced the ET_{iso} in this horse as desired.

End-tidal isoflurane was recorded as 1.4%–1.5% for the majority of the anaesthetic period, which is above the reported MAC of 1.31%.^{6,42} The anaesthetic requirements of any individual horse may indeed be higher than the MAC, but the use of pre-medication would likely reduce inhalant requirement. This horse's plane of anaesthesia could have been excessively deep, implicating a reduction in tissue perfusion. Omitting a romifidine infusion, thereby avoiding the decreases in cardiac output attributed to alpha-2 agonists, and/or maintaining a lower ET_{iso} , may have improved global tissue perfusion in this case. The increase in SVR expected with romifidine in combination with reduced cardiac output may have compromised tissue perfusion further in this horse. Alternatives for a partial intravenous anaesthetic protocol include ketamine or lidocaine, both of which provide analgesia and may reduce MAC requirements.⁴³ However, it is impossible to say whether a different anaesthetic protocol would have improved the outcome for this horse.

Minimising anaesthetic time as much as possible is important, as anaesthetic duration over 60 minutes is associated with a higher risk of death in horses.⁴⁴ Duration of anaesthesia was 170 minutes. Minimising anaesthesia time as much as possible, for example by pre-clipping surgical sites, is always advisable.

The horse was premedicated with morphine at 0.2 mg/kg IV. Although this dose falls within the normal range,²⁶ the principles of allometric scaling⁴⁵ suggest a lower dose may have been more appropriate. Larger animals require lower drug doses on a mg/kg basis because they have a lower metabolic rate.³⁶ Morphine was chosen for this horse due to its analgesic properties and its association with improved recoveries in this species.^{46,47} It is unlikely that the cardiovascular effects of morphine itself contributed to reducing perfusion, as these have been shown to be minimal in anaesthetised horses.^{46,48} Morphine has unpredictable effects on inhalant anaesthetic requirement and may increase or decrease the

MAC.⁴⁹ It is possible that the relatively high dose of morphine may have increased the depth of anaesthesia in this horse, which could have contributed to inadequate tissue perfusion.

The peri-operative crystalloid fluid administration rate of 2.5 ml/kg/h likely contributed to the hypoperfused state in this horse, as this is considerably lower than the commonly targeted rate of 5–10 ml/kg/h,⁵⁰ which is intended to augment intravascular volume and maintain haemodynamic homeostasis. Progressive haemoconcentration is evident from the increasing PCV (see Table 1), possibly attributable to dehydration as well as the gradual elimination of acepromazine and romifidine. The reliance on gravity to drive fluid administration and omission of assessment of this rate contributed to the discrepancy in fluid provision for this horse, which may have contributed to an insufficient circulating volume. Elevation of the fluid bag can markedly increase fluid flow via gravity. Other means to increase fluid administration include fluid pumps or high-pressure infusion systems, a wider bore giving set and/or intravenous cannula, and placing an additional intravenous cannula.

Muscle perfusion in horses under general anaesthesia is significantly affected by positioning, with hypoperfusion being more likely in dependant body regions owing to high intracompartmental pressure in dependant muscles.¹⁹ Non-dependant body regions can also be at risk of hypoperfusion due to decreased hydrostatic pressure in vessels above the heart.³³ Although recumbency has not been shown to be associated specifically with mortality,⁵¹ it does dictate which anatomical structures will be subject to the highest pressures and which areas are involved in weight distribution through their direct contact with the table surface. The surface area in contact with the surgery table is smaller for a horse in dorsal recumbency than for lateral recumbency, so more pressure will be exerted onto dependent tissues per unit of area in the dorsal position. These areas are consequently most at risk of pathology. In this horse, the bilateral distribution of affected muscles is highly suggestive that dorsal recumbency played a key role. 'Side flaps' on the table support the hindlimbs in an axial direction and are intended to prevent splaying. Although the hindlimbs of this horse appeared evenly positioned, it is possible the side flaps provided insufficient or excessive support, which would have created tension in either the medial or lateral hindlimb musculature respectively. This would have caused stretching of the blood vessels running within, thereby compromising muscle perfusion.

Because both hindlimbs were affected in this horse, bilateral femoral neuropathy is a possibility; multiple reports exist of this condition.⁵² Nerve injury is commonly associated with compression or stretching of the affected nerve/s and consequent nerve ischaemia and hypoxia,⁷ either from the horse's own body weight, or external factors such as positioning or restraint devices. However, neuropathies are typically characterised by non-painful, non-swollen musculature⁷ which is in contrast to the signs exhibited in this case. Hoisting of the horse onto the surgery table could have caused trauma to nerves, but this is deemed unlikely due to the short time period of this intervention (approximately 3–5 minutes).

A well-padded surface is required for the even distribution of body weight³² for even distribution of pressure exerted on dependent musculature. The padding used in this case was deep foam, employed as standard for all anaesthetised horses at this facility.

Hyperlactataemia (6.04 mmol/L) was identified 105 minutes into anaesthesia. Normal lactate measurements in adult horses are typically <0.7 mmol/L.⁵³ Systemic lactate gradually increases in anaesthetised horses⁵⁴ up to approximately 3.0 mmol/L,¹⁷ but this is significantly lower than the values observed here. Most accounts of post-anaesthetic myopathy do not report any specific biochemical changes, although lactate testing may not have been available in all previous reports and anaesthesia may be uneventful until the recovery stage.⁶ Type B hyperlactataemia describes an increase in lactate production despite adequate DO₂. Type A hyperlactataemia occurs secondary to inadequate tissue oxygen delivery (DO₂), as a by-product of anaerobic cell metabolism⁵³ and seems most plausible in this case.

Quicker recognition of and response time to the first confirmation of hyperlactataemia would have been beneficial in this case. A second sample was checked to verify the findings 25 minutes after the first lactate result. Although it was clear that a pathological process was already underway at the time of the first lactate result, earlier action may have contributed towards a better clinical outcome for this horse. Immediate action to abort anaesthesia at the first confirmation of hyperlactataemia, or at least increasing intravenous fluid provision to try to augment circulating volume and thereby improve tissue perfusion, would have been appropriate. Careful evaluation of the positioning of the horse, which could have contributed to inadequate tissue perfusion, would have been a logical addition to the decision making process at this stage. Measurement of respiratory blood gases was the primary reason for blood testing in this case initially, due to the expectation that respiratory derangements were the most likely abnormality to occur. The first blood sample was tested 50 minutes into anaesthesia using a cartridge that did not include lactate measurement. Obtaining a 'baseline' lactate at the beginning of general anaesthesia to monitor for serial increases over time may be helpful, especially in horses deemed more susceptible to tissue hypoxia. Monitoring of biochemical and respiratory blood gas variables as early into the anaesthetic period as possible is essential to identify problems at their onset. Taking aggressive corrective action in response to any hyperlactataemia identified is key.

The first arterial blood gas sample revealed a ventilation-perfusion (V/Q) mismatch in this horse. The PaCO₂ was 45.5 mmHg which contrasts markedly with the PE'CO₂ at this time which was 31–34.5 mmHg. Differences in arterial and end-tidal CO₂ have been shown to accurately reflect V/Q mismatching in humans.⁵⁵ Mismatching occurs due to a discrepancy between the relative ventilation and perfusion of the alveoli. A V/Q ratio < 1 represents relatively less ventilation than perfusion; V/Q > 1 occurs when there is relatively less perfusion than ventilation.⁵⁶ Either scenario would cause the difference seen here between the arterial and end-tidal CO₂ concentrations, but the low PE'CO₂ specifically indicates a relative increase in alveolar 'dead space' due to altered perfusion.⁵⁷ An appreciation that this process was occurring may have alerted the anaesthetist to the potential of hypoperfusion in this horse and the consequent risk of myopathy.

Application of a suspension sling was performed to assess the horse's ability to bear weight on the hindlimbs and enable repositioning into sternal recumbency. Resumption of sternal positioning after a period of lateral or dorsal recumbency improves tissue oxygenation.⁵⁸ Suspension slings are an

accepted system for recovery in horses⁵⁹ and appear to be well-tolerated,⁶⁰ but concerns exist over their potential to create focal pressure points and impede spontaneous ventilation.⁶¹ Due to the severity of the clinical presentation in this case, the sling was attempted as a 'last resort' intervention. A second short period of general anaesthesia was necessary to apply the sling safely. The effect of a standard dose of diazepam for this short anaesthetic may have exacerbated muscular weakness, as plasma half-life is approximately 1 hour,⁶² and reducing the dose accordingly may have been beneficial. Supplementing oxygen during this anaesthetic would also have been prudent given the likelihood of tissue hypoxia, but proved logistically difficult due to the limited space in the recovery box.

Increased serum creatinine (>315 µmol/L) in the recovery period influenced the decision to euthanase this horse. Acute kidney injury was suspected due to inadequate renal perfusion secondary to low circulating volume, dehydration and renal insult secondary to myoglobinuria from muscle damage. A post-renal cause of azotaemia cannot be ruled out. Myoglobinuria is a known consequence of myopathy,⁵⁰ but its presence was unable to be verified in this horse due to the inability to place a urinary catheter. Myoglobin is a nephrotoxin⁶³ and decreases renal blood flow, consequently reducing renal oxygenation, resulting in ischaemic injury.⁶⁴ The possibility of pre-existing renal disease in this horse cannot be excluded as pre-anaesthetic biochemistry testing was not carried out. Prior assessment of renal parameters and electrolytes may have been beneficial in this horse given the urinary system abnormalities.

Hyperkalaemia can result from anuric renal failure,⁶⁴ urinary obstruction causing reabsorption of potassium ions from the urinary bladder, iatrogenic overdose of potassium or the release of intracellular potassium secondary to muscle cell damage.⁶⁵ It is not clear whether the horse was truly anuric: urinary catheterisation was impossible due to the conformational abnormalities of the genitalia, so measurement of urine output was not possible. No urine production was documented at any point following the first anaesthetic induction. Hyperkalaemia worsened despite fluid therapy (see Table 1) and treatment of this became the most acute consideration because of the potential for life-threatening cardiac arrhythmias.⁶⁴ The inability to place a urinary catheter made it impossible to facilitate diuresis, a key treatment for hyperkalaemia. Additional treatments for hyperkalaemia include administration of dextrose and insulin, to promote intracellular uptake of potassium ions and calcium gluconate to protect temporarily against the adverse cardiac effects of hyperkalaemia.⁶⁶

Although this horse was deemed healthy before anaesthesia, one peculiarity is the suspicion of male pseudohermaphroditism. Links have been made in the human medical literature between pseudohermaphroditism and reduced aldosterone and cortisol production,⁶⁷ which could compromise the stress response mounted to general anaesthesia and surgery. Recognition of the possible deficiencies with this condition and testing of electrolytes and cortisol levels could be useful for future cases.

Avoiding general anaesthesia in this horse would have prevented this outcome. However, the ventral location of some sarcoids, the need for aggressive local excision and the requirement for invasive genital examination precluded the procedure being performed 'standing' with the horse under

sedation. It was of course impossible to know the actual risk posed to this individual horse by general anaesthesia before the event, but considering the clinical outcome, its large size certainly played a role in increasing the risk above that for a 'standard' sized adult horse (e.g. 500 kg).

There is significant room for improvement in particular factors pertaining to the anaesthesia of this case; namely the identification and response time to hyperlactataemia, the provision of intravenous fluid therapy in the peri-operative period, potentially excessive anaesthetic depth and, possibly, the choice to perform partial intravenous anaesthesia with romifidine. A full and frank audit of this case has been undertaken by the anaesthetist involved and the wider clinical care team to reflect on the areas for improvement.

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CONFLICT OF INTEREST

The authors declare they have no conflicts of interest.

AUTHOR CONTRIBUTIONS

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Ethical approval is not applicable as this report describes a clinical case.

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