Post-anaesthetic myelomalacia in a horse

K E Joubert[a*], N Duncan[b] and S E Murray[c]

ABSTRACT

This article describes a rare neurological complication of anaesthesia in a 2 year-old Clydesdale colt undergoing castration. Anaesthesia was induced with glyceryl guaiacol ether and ketamine and maintained with halothane. Following an uneventful anaesthetic of 40 minutes, the horse recovered from anaesthesia in a padded recovery stall. After approximately 70 minutes in the recovery stall, the horse attempted to stand and adopted a dog sitting position. One hundred and fifty minutes later, the horse became distressed and was sedated with xylazine. Clinical examination of the horse did not reveal any evidence of myositis or fractures. A neurological examination revealed an intact anal reflex, deep pain response in the hind legs, tail tone and voluntary movement of the hind legs was possible. The horse deteriorated neurologically over the next 24 hours and was euthanased on humane grounds. The horse was submitted for necropsy. Gross pathology was unremarkable except for a small amount of haemorrhage around the right kidney. Histopathology revealed no abnormalities in any muscle groups or peripheral nerves. Congestion and axonal swelling of the spinal cord was evident from T16 to S1. Ischaemic neurons were evident from L 1 to L 6. The most prominent lesions were at L4 and L5. A diagnosis of myelomalacia was made. This is a rare complication of anaesthesia in horses with 9 case studies appearing in the literature since 1979. This is the 1st case to be reported in South Africa. The speculated pathophysiology and risk factors for this complication are discussed.

Key words: anaesthetic complications, equine, myelomalacia.

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INTRODUCTION

Anaesthetic mortality in horses is high (approximately 2 %)[15] when compared with small animals[5,7]. The most prominent causes of equine mortality are cardiovascular collapse and arrest (33.2 %), fractures (25.6 %), myopathes (7 %) and abdominal complications (13.1 %)[15].

The literature on anaesthetic-related neurological complications in horses is limited to case studies[2,3,16,19,20,28,29]. Myelomalacia is a rare anaesthetic complication in horses and there have been no controlled studies. Our understanding of this condition is largely based on case studies reported in the literature[26]. In order to improve our understanding of this condition it is vital that each case is carefully documented to allow for a retrospective study of the literature, an assessment of risk factors and identification of possible pathophysiological mechanisms. This article presents the first recorded case of myelomalacia in a horse in South Africa and discusses the current evidence in the literature.

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CASE HISTORY

A 2 year-old 480-kg Clydesdale colt was presented for castration under general anaesthesia. The horse was difficult to examine but was clinically normal. Acetylpromazine (15 mg, i/m) (Aceprom 10, Centaur Labs, Isando), procaine penicillin (30 ml, i/m)(Depocillin, Intervet, Isando) and tetanus toxoid (1 ml, i/m) (Tetanus, Onderstepoort Biological Products, Onderstepoort) were administered one and a half hours before the induction of anaesthesia. Anaesthesia was induced with glyceryl guaiacol ether (30 g, i/v) (GGE, Kyron Laboratories, Benrose) until the horse was unstable, followed immediately by ketamine (1.2 g, i/v) (Anaket V, Centaur Labs). The horse was then intubated and maintained on halothane (Fluothane, AstraZeneca, Sandton). The duration of anaesthesia and surgery was 40 minutes. The heart rate was initially high (50 bpm) after induction but settled down to between 42–33 bpm for the remainder of the anaesthetic. Blood pressure (mean blood pressure 68.5 ± 3.4 mm Hg, systolic 94.3 ± 6.1 mm Hg and diastolic 55.6 ± 3.8 mm Hg) was maintained through the administration of fluids (PlasmaVet, Sabax, Midrand) and a constant-rate infusion of dobutamine (Dobutrex, Eli Lilly, Bryanston). Peripheral blood saturation was maintained above 99 % throughout the procedure. Respiratory rate varied between 12–15 bpm with the horse breathing spontaneously. A standard castration was performed and no surgical anomalies were encountered. Following completion of the surgery, the horse was placed in a padded recovery stall. The endotracheal tube was removed as soon as the swallowing reflex returned and a naso-pharyngeal tube was placed to maintain a patent airway.

One hour and ten minutes after the end of anaesthesia the horse attempted to stand on its front limbs but no support was seen from the hind limbs. Examination of the horse at this time revealed an elevated pulse rate (50) and respiratory rate (30) and normal lung sounds. The large muscle masses of the hindquarters were warm, soft and non-painful (making myositis unlikely), the anal and tail tone were normal. Two hours after the end of anaesthesia the horse attempted to rise in the front again but was unable to use the hind limbs. Phenylbutazone (15 ml, i/v) (Phenylarthrite, Centaur Labs) was administered. Thirty minutes later the horse was alert and eating in sternal recumbency. Three hours after the end of anaesthesia the horse had made numerous attempts to stand and was exhausted. Xylazine (40 mg, i/v) (Chanazine, Centaur Labs) and butorphanol (10 mg, i/v) (Torbugesic, Forte Dodge, Kempton Park) were administered to allow the horse to relax. Fluids (PlasmaVet) and a calcium-magnesium combination (500 ml, i/v) (MFC, Merial, Midrand) were administered. Body temperature was raised after the physical effort of trying to stand (38.5 °C) and urine was dribbling from the penis. A urinary catheter was placed and the bladder was emptied. An attempt was made to assist the horse to stand but no support from the hindquarters was

Table 1: **Signalment, procedure, position, duration of anaesthesia and outcome for the 9 case studies and the present case study.**

Year of publication	Author	Breed	Sex	Weight (kg)	Age (months)	Procedure	Position	Duration of anaesthesia (minutes)	Outcome
1979	Schatzmann[20] (1)	Inlanders	Colt	360	14	Infected mandibular lymph node	Dorsal	95	Euthanased
1979	Schatzmann[20] (2)	Inlanders	Filly	470	18	Umbilical hernia	Dorsal	55	Euthanased
1984	Blakemore[2]	Shire	Filly	300	6	Umbilical hernia	Dorsal	60	Euthanased
1985 later	Zink[29]	Quarter horse	Colt	360	24	Cryptorchid	Dorsal	105	Died 8 days
1986	Yovich[28]	Quarter horse	Colt	350	12	Cryptorchid	Dorsal	45	Euthanased
1986	Brearley[3]	Shire	Filly	370	8	Umbilical hernia	Dorsal	40	Died
1993	Lerche[16]	Belgian	Colt	645	18	Cryptorchid	Dorsal	60	Euthanased
1994	Wan[27]	Appaloosa	Colt	234	12	Castration & arthroscopy	Dorsal	75	Euthanased
1997	Raidal[19]	Thoroughbred	Colt	560	24	Castration	Right lateral	25	Euthanased
1997	AFIP[1]	Not stated	Colt	307	12	Castration	Dorsal	Not reported	Euthanased
2004	Present study	Clydesdale	Colt	480	24	Castration	Dorsal	40	Euthanased
Mean				**403**	**15.6**			**60**	
SD				**122**	**6.4**			**25.3**	

evident. Prednisolone (100 mg)(Solu-Cortef, Pharmacia Upjohn, Sandton) was administered intravenously. The horse remained alert in sternal recumbence and readily ate and drank.

During the evening the horse was still able to eat, drink, defaecate and urinate. The following morning the horse showed signs of weakness in the front limbs. The horse was sedated with xylazine (120 mg, i/v) and butorphanol (20 mg i/v) and moved so that it could be slung. The attempt to sling the horse was unsuccessful. The horse was treated with procaine penicillin (20 ml, i/m), dimethyl sulphoxide (100 ml, i/v) (Kyron DMSO, Kyron Labs), Vitamin E-Selenium (20 ml, i/m) (Selevit E, Kyron Labs), vitamin B_1 (20 ml, i/v) (KyroVite B1, Kyron) and a vitamin B_6, B_{12} and glycerol-phosphate solution (40 ml, i/v) (Phosamine Stimulans, Centaur Labs). The horse's ability to prehend food decreased and muscle weakness progressed through the day. By the afternoon, he was no longer able to maintain sternal recumbency. The horse was euthanased on humane grounds with owner consent and submitted for a *post mortem*.

On *post mortem*, the only gross lesion observed was haemorrhage involving the perirenal tissue of the right kidney and adjacent musculature. The spinal cord was removed, cut into transverse slices at each spinal level and fixed in 10 % buffered formalin. Prior to fixation, the grey matter was observed to be noticeably congested and soft from T16 to S1. Histological examination of the spinal cord revealed oedema and congestion of the grey matter from T15 to S1, haemorrhage within the grey matter from L1 to S1 and the presence of ischaemic neuronal changes characterised by axonal swelling, dispersal of the Nissle substance (chromatolysis) and disappearance of the nucleus (karyolysis) from T18 to S1. The area showing the most severe change was L3–L6. No vascular lesions were observed, neither were any areas of glial proliferation or inflammatory cell infiltrate. The adjacent white matter also showed varying degrees of congestion. The remainder of the spinal cord and brain were normal. The changes observed were consistent with ischaemic myelopathy.

DISCUSSION

This article reports the first documented case of myelomalacia following general anaesthesia in South Africa. The 1st report of this complication appeared in the literature in 1979[20]. This has been followed by the addition of 8 other cases from different parts of the world[1–3,16,19,28]. A further 15 horses have been reported in a review article of neurodegenerative disease of horses by Gruys[12] and again in a congress abstract[24]. The review article and congress abstract unfortunately do not supply a complete history and clinical data of each horse, making it difficult to assess risk factors. The CEPEF study indicates that 18 horses were euthanased or died due to neurological complications that included myelomalacia[15]. Unfortunately no further details are given. The discussion in this article is based mainly on the case reports summarised in Tables 1, 2 and 3.

The failure of a horse to stand following anaesthesia is usually the result of musculo-skeletal damage. Myositis and fractures are the most common. Myositis is inducible with hypotensive anaesthesia[11,17] but may occur in patients that have had a normotensive anaesthetic[6]. The affected muscles in myositis are usually firm, swollen and painful on palpation[13]. Lameness and paresis are usually evident[13]. Fractures are usually extremely painful and associated with swelling in the area. These conditions were excluded in this patient based on clinical examination, making a neurological lesion more likely. Neurological complications in horses are devastating due to the logistical limitation of therapy. Complications in horses with neurological dysfunction include fracture of the cervical vertebra[4],

Table 2: **Anaesthetic agents used in the 9 case studies and the present case.**

Author	Anaesthetic drugs
Schatzmann[20] (1)	Proprionylpromazine, GGE, halothane, nitrous oxide
Schatzmann[20] (2)	Not reported
Blakemore[2]	Acepromazine, thiopentone, halothane
Zink[29]	acepromazine, Chloral hydrate, thiopentone, halothane
Yovich[28]	Xylazine, GGE, ketamine, halothane
Brearley[3]	Acepromazine, thiopentone, halothane
Lerche[16]	Xylazine, GGE, thiopentone, halothane
Wan[27]	Acetylpromazine, GGE, ketamine, halothane
Raidal[19]	Xylazine, ketamine
AFIP[1]	Not reported
Present study	Acetylpromazine, GGE, ketamine, halothane

Table 3: **Myelomalacia lesion site and evaluation of neurological reflexes.**

Author	Lesion*	Paniculus	Front limbs	Hind limbs	Deep pain	Tail tone	Anal tone
Schatzmann[20] 1	T12–L intumesces	Present	Paretic	Paraplegic	Not reported	Not reported	Not reported
Schatzmann[20] 2	Caudal T–L–cranial S	Not reported	Not reported	Not reported	Not reported	Not reported	Absent
Blakemore[2]	L4–S1	Absent	Normal	Paretic	Present	Present/absent	Not reported
Zink[29]	T–L	Present	Normal	Paretic	Not reported	Absent	Absent
Yovich[28]	C6–T8	Absent	Normal	Paraplegic	Absent	Hyper	Hyper
Brearley[3]	LS	Not reported	Not reported	Not reported	Not reported	Not reported	Not reported
Lerche[16]	T15–S1	Not reported	Normal	Paraplegic	Absent	Absent	Absent
Wan[27]	T2–S2	Not reported	Normal	Paretic	Absent	Hypo	Hypo
Raidal[19]	T18–L 4	Not reported	Normal	Paraplegic	Not reported	Absent	Absent
AFIP[1]	T2–S2	Not reported	Normal	Paraplegic	Not reported	Absent	Absent
Present study	T18–S1	Not done	Normal	Paretic	Present	Present	Present

*T, thoracic vertebrae; L, lumbar vertebrae; C, cervical vertebrae.

cerebral necrosis[21] fibrocartilaginous infarct[10,14,25] and myelomalacia. Myelomalacia was suspected in this case before euthanasia based on its signalment.

Myelomalacia is most commonly described in young (mean 15 months) large breed (or large for their size) horses (mean 403 kg) undergoing surgery in dorsal recumbence usually for an elective procedure. Anaesthetics are usually of a short duration (mean 60 minutes) and are usually uneventful. Short-duration anaesthetics are usually associated with a reduction in perioperative mortality[15]. There appears to be no relation to the drugs administered as multiple anaesthetic protocols have been used. Most cases have been reported in colts undergoing a castration and fillies undergoing an umbilical hernia repair. These signalment characteristics are common to this case. All reported cases died or were euthanased. (Tables 1 and 2)

Neurologically this condition usually appears as a progressive hind limb paresis to paralysis with the horse usually assuming a dog sitting position. Some horses may stand before becoming recumbent[3,19]. The front limbs are usually normal but with time may become paretic. This is easily explained as most lesions have been reported in the thoraco-lumbar region. Deep pain, the anal reflex, tail tone and the paniculus reflex may or may not be normal, making these neurological tests a poor discriminator for this condition. Neurological evaluation is important to monitor the progression of this condition and differentiate out other neurological conditions. (Table 3)

The pathophysiology of myelomalacia is most likely to be the result of an ischaemic insult to the spinal cord during anaesthesia[2,3,16,19,20,29]. A number of postulates have been put forward as a mechanism of action. Most cases have been reported in dorsal recumbency[2,3,16,20,28,29] and it is speculated that the weight of the abdominal organs compresses the abdominal vasculature with a resultant hypoperfusion of the spinal cord. Compression of the venous drainage is as important as the arterial system. Venous congestion of paravertebral sinuses may play a role[20]. Wan suggested that due to the anatomical relationship between the large colon and the vena cava this may be a causative mechanism in dorsal recumbency[27]. Blakemore suggested positioning the horse in a slightly oblique position to prevent compression of the vasculature[2]. One case has been documented in lateral recumbency[19] which confounds this suggestion. Schatzmann suggested that the vasculature in young animals was unable to adapt to the change in position resulting in compression[20]. Adult horses were able to adapt and hence myelomalacia did not occur. This may be challenged by the review article of Gruys *et al.* who describe the condition in an 11 and a 16 year-old horse undergoing enterotomies[12].

A reduction in cardiac output and hypotension is speculated to reduce spinal blood flow and result in spinal hypoxia[3,27]. Inhalation anaesthetic agents reduce cardiac output in a dose-dependent fashion[8,22,23]. Alpha$_2$ adrenergic agents are known to decrease cardiac output[9,18]. This may further compromise the blood supply to the spinal cord and promote ischaemic injury[3]. The case studies document the use of xylazine or acetylpromazine as premedicants, thiopentone or ketamine with or without glyceryl guaiacol ether for induction of anaesthesia and halothane as a maintenance agent. One horse was maintained intravenously[19]. This makes it difficult to identify a specific anaesthetic agent as a causal agent. The effect of these agents on cardiovascular function may be the common theme. Positional changes are also known to effect cardiac function[27].

Histological evidence indicates that post-anaesthetic myelomalacia may be related to equine degenerative myeloencephalopathy[12,24]. This disorder has been associated with a Vitamin E and selenium deficiency[12,24]. This has led to the suggestion that vitamin E and selenium are involved in the pathogenesis of post-anaesthetic myelomalacia[12,16,24]. Vitamin E and selenium were not assessed in the 1 case that speculated this as a cause[16].

The pathophysiology of this condition is speculative. This makes it difficult to reproduce the condition for scientific research and improve our understanding. This condition carries a poor prognosis in the reported literature. Mild cases most probably recover and remain undiagnosed as no histopathology is done. This condition should be considered in horses that have hind limb paresis/paralysis following general anaesthesia with the typical signalment. No method of management can currently be suggested to prevent this condition.

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