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The second greatest cause of perioperative death in horses is catastrophic orthopaedic injury sustained during recovery from anaesthesia (Johnston et al. 1995). Orthopaedic injuries have 3 fundamental causes: abnormal (high) forces; force applied through abnormal angles; and abnormal bone strength. The nature of orthopaedic injuries sustained in horses recovering from anaesthesia is unknown. The author believes pelvic limb (femur and tibia) and cervical vertebral luxations are the most common. The underlying aetiology may differ according to the nature of the injury.

Excessive force may be applied through limb bones when recoveries are uncharacteristically violent. This seems probable when inadequate analgesia in pain-intolerant subjects initiates a characteristic ‘fight-or-flight’ reaction. Horses damaging cervical vertebrae may do so because of vestibular and/or visual impairment and/or proprioceptive dysfunction. Alternatively, a failure to avoid wall collision may result from muscle weakness.

Normal forces applied at abnormal angles may result from the imposition of casts or thick bandaging, the physical properties of the substrate (high friction vs. slippery) or abnormal leg positioning during a normal rising attempt. The latter may occur because of proprioceptive defects caused by neurapraxia, the persistent action of anaesthetics, or by pre-existing pathology, e.g. cervical vertebral anomalies. Alternatively, myasthenia arising from ischaemic muscle damage or physical exhaustion may be present. The effects of misdirected force will be greater in heavier animals. ‘Fight or flight’ reactions may conceivably result in additional action of anaesthetics, or by pre-existing pathology. The horse’s temperament is probably important, and its role in the quality of assisted recoveries is currently being investigated. The animal’s size should also be considered, smaller ponies are more readily restrained with ropes, even when they have arthritis. Abnormally weak bone is probably a factor in many post-operative catastrophes. The Confidential Enquiry into Perioperative Equine Fatalities (CEPEF-1) revealed that the relative risk in orthopaedic cases undergoing internal fixation was 3.2. After nonorthopaedic surgery the author believes that pelvic limb fracture is most likely to occur in older (>12 years), overweight, physically compromised cases and its role in the quality of assisted recoveries is currently being evaluated. The animal’s size should also be considered, smaller ponies are more readily restrained with ropes, even when they precipitate a violent reaction.

For a horse is to stand post-operatively, either ‘assisted’ or otherwise, it must: a) want to stand; and b) be able to stand. Normally, the author allows 20 min recovery time per hour of ‘down-time’ before expecting to see some sort of standing attempt made. A horse may be unwilling to stand because of foot, limb or abdominal pain, myositis or the loss of will (and/or energy) after multiple failed attempts to rise. Pain should be treated and floor ‘purchase’ provided. Any unexpected cause of weakness should be investigated, e.g. myelomalacia. In these cases a blood sample should be taken for haemoglobin and electrolyte analysis. There is currently no evidence that intervening in recovery after an equine anaesthetic reduces morbidity or mortality figures. It is likely that certain cases benefit from intervention but equally undeniable that intervention becomes interference in some cases.
References


NOTES


The paradox of 100% oxygen leading to hypoxia in anaesthetised horses

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During general anaesthesia and recumbency, pulmonary gas exchange and arterial oxygenation commonly deteriorate in the horse (Nyman and Hedenstierna 1989). Even when horses are ventilated with a high fraction of oxygen, it is sometimes difficult to keep the animal well oxygenated (Hubbell et al. 1989). The major contributor to hypoxaemia in the horse is atelectasis and, to some degree, ventilation-perfusion mismatch (Nyman et al. 1990). During inhalation anaesthesia atelectasis occurs in the dependent lung regions. Shunting of blood comprises approximately 20% of the total lung tissue in the average 500 kg horse positioned in lateral recumbency and 33% during dorsal recumbency. There are 2 prerequisites for atelectasis to develop during anaesthesia; firstly loss of respiratory muscle tone with subsequent reduction of lung volume and secondly ventilation with highly absorbable gas, which initiates resorption atelectasis.

Little attention has been paid to the effect of different inspired oxygen fractions in horses although Cuveliez et al. (1990) several years ago reported that horses breathing 85% oxygen during 4 h of inhalation anaesthesia displayed a progressive increase in the alveolar - arterial oxygen partial pressure difference P(A-a)O2, compared to when breathing 30% oxygen. Since pure oxygen is commonly used as the carrier gas during inhalation anaesthesia in horses, investigations of equine pulmonary dysfunctions occurring with use of different anaesthetic protocols and positions have mainly been conducted during inhalation with 100% oxygen. During field anaesthesia, air breathing has not been recommended for longer periods than 60 min irrespective of injectable drugs chosen because of the danger of hypoxia and myositis (Matthews et al. 1991). It has also been proposed that horses under i.v. anaesthesia could be intubated and connected to the demand valve, allowing administration of oxygen and ventilation periodically to prevent atelectasis (Riebold et al. 1980).

In human studies, inspiration of 100% oxygen during anaesthesia has been found to promote intrapulmonary shunt and atelectasis, in contrast to inspiration of 30% oxygen in nitrogen (Rothen et al. 1995a). In the border area between ventilated and nonventilated lung areas the composition of the inhaled gas will affect the ability of the alveoli to stay open. If a lung unit is entirely closed off, oxygen as a highly absorbable gas will easily be taken up by the blood flow so that the gas pocket behind closed airways is emptied. The time taken for the alveoli to collapse has been estimated to be 6–9 h if the unit contains air (79% of which is the poorly soluble gas N2) and about 6–8 min if it contains pure oxygen (Joyce et al. 1993). In man, it has been shown that atelectasis in the anaesthetised subject occurs already during the induction of anaesthesia when pre-oxygenation with 100% oxygen is provided. Rapid collapse of alveoli on induction of anaesthesia and more widespread closure of airways seem to explain the oxygenation impairment and may also contribute to post operative pulmonary infection (Lindberg et al. 1992). Lowering of oxygen fraction from 100% to 80%, drastically reduce the atelectasis incidence but the safety time of apnoea period is not shorter which is particularly useful for a difficult intubation (Edmark et al. 2003). Thus, in human anaesthesia, avoiding high inspired oxygen fractions during both induction and maintenance of anaesthesia prevents or reduces atelectasis, while intermittent ‘vital capacity’ manoeuvres recruit atelectatic lung regions. A recruitment manoeuvre in anaesthetised human patients atelectasis recurred within 5 min if the patients were ventilated with FiO2 = 1.0 in contrast to the group ventilated with FiO2 = 0.4 where atelectasis was eliminated for at least 40 min (Rothen et al. 1995b). Therefore, a modern recommendation in human anaesthesia is that ventilation if possible should be performed with a moderate oxygen concentration (30–40%).

To investigate the pulmonary function and gas exchange during and after breathing gas of a high fraction of oxygen compared to air breathing, horses were studied during dissociative anaesthesia (Marntell et al. 2005). All horses developed intrapulmonary shunt, irrespective of inhaled gas composition, but the magnitude of shunt was significantly larger during inspiration of high oxygen as compared to air breathing. After induction of anaesthesia, the shunt thus initially may be a result of compression atelectasis due to the relaxed diaphragm tone and the recumbent position. The significantly larger shunt developing after breathing of >95% oxygen may be a result of a concomitant transformation of intermittently closed alveoli to atelectasis by resorption of oxygen by the blood. Breathing a high fraction of oxygen resulted in increased PaO2 compared to air breathing, but concomitantly hypoventilation, i.e. increased arterial carbon dioxide tension was evident. Interestingly, in horses positioned in lateral recumbency the average shunt of 5% following air breathing and 13% following breathing of >95% oxygen during dissociative anaesthesia was less pronounced than an average shunt of 20%, observed previously in horses during inhalation anaesthesia with an inspired oxygen concentration >90% (Nyman and Hedenstierna 1989). Although no direct comparison is possible, the difference might be attributable to the use of different fractions of inhaled oxygen, drug-induced differences or better preserved muscular tone, particularly the diaphragm tone, during dissociative anaesthesia. The intrapulmonary shunt created during breathing of high oxygen concentrations remained when oxygen concentration was reduced from 95% to 21% during anaesthesia. This indicates that resorption atelectasis produced during breathing of high oxygen concentrations subsequently persists throughout recumbency and anaesthesia.

In summary, atelectasis is present in most horses during anaesthesia and is the major cause of impaired oxygenation. Causative mechanisms to atelectasis and airway closure seem to be loss of respiratory muscle tone and gas resorption. Avoiding high fractions of oxygen in inspired gas during induction and maintenance of anaesthesia may prevent formation of atelectasis in the anaesthetised horse.

References


Safe balanced anaesthesia techniques for horses

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Balanced anaesthesia is a technique of general anaesthesia based on the concept that administration of a mixture of small amounts of several neuronal depressants summates the advantages but not the disadvantages of the individual components of the mixture.

Drugs used in combination with inhalation anaesthesia

Lidocaine

Lidocaine is highly metabolised by the liver and has a very short half life (Doherty and Frazier 1998). It has to be administered as a bolus followed by CRI (constant rate infusion). The first report (Doherty and Frazier 1998) used a bolus of lidocaine (2.5 mg/kg bwt/min) followed by 50 µg/kg/min or 100 µg/kg bwt/min for 1 h. This resulted in a dose dependent minimum alveolar concentration (MAC) reduction, but lidocaine plasma levels were very variable. In another clinical study (Dzikiti et al. 2003) lidocaine was combined with isoflurane anaesthesia and administered for 75 min. A bolus dose of 2.5 mg/kg bwt (given over 10 min) followed by 50 µg/kg bwt/min resulted in an average MAC reduction of 25%. The horses in the lidocaine group recovered with less excitement from anaesthesia. In contrast to this, horses recovering form anaesthesia after a mean lidocaine infusion duration of 125 min showed significantly worse recoveries with lidocaine in comparison to balanced anaesthesia with medetomidine (Ringer et al. 2007). Also in a clinical study that investigated the influence of lidocaine on recovery from isoflurane or sevoflurane anaesthesia it was shown, that horses receiving lidocaine until the end of surgery showed a significantly higher degree of ataxia (Valverde et al. 2005). It was recommended to discontinue lidocaine CRI 30 min before the end of surgery to reduce ataxia during the recovery period.

Faery et al. (2005) showed in clinical cases that anaesthesia with sevoflurane has a profound effect on lidocaine disposition. Lidocaine plasma levels were considerably higher during anaesthesia than in awake horses. They recommended the use of lower dose rates in anaesthetised horses, as general anaesthesia might mask neurologic manifestations of toxicity. In another study (Brianceau et al. 2002) in horses with colic dose rates were considerably lower than in previous studies (0.65 mg/kg bwt loading dose followed by 25 µg/kg bwt/min). Nevertheless these authors measured toxic plasma levels in one horse and advocated prudent intraoperative dosing. Contrary to this, a retrospective clinical study that the successful use of lidocaine in combination with isoflurane or sevoflurane in 25 horses undergoing colic surgery. A bolus of 1.5 mg/kg bwt lidocaine was given just before surgery and the infusion of 30 µg/kg bwt/min was stopped when the surgeon started to close the abdomen. In this study horses with lidocaine did not show worse recoveries than without and no signs of toxicity were noted.

To summarise a lidocaine bolus (0.65–2 mg/kg bwt) administered over 10–15 min followed by CRI 25–50 µg/kg bwt/min can be used as part of a balanced anaesthesia regime in horses. It decreases MAC dose dependently. Higher dose rates might induce toxicity especially in compromised patients. Toxicosis only becomes apparent after the effect of the inhalant anaesthetic has vanished and might negatively influence recovery. The administration of lidocaine should be with care and stopped at least 30 min prior to the end of anaesthesia, to reduce the occurrence of ataxia and uncontrolled recoveries following lidocaine balanced anaesthesia regimes.

Alpha2-adrenoceptor agonists

Alpha2-adrenoceptor agonists are potent analgesics, and reduce the MAC of inhalation agents dose dependently (Steffey et al. 2000). All available alpha2-adrenoceptor agonists have already been used for balanced anaesthesia in horses (Clarke et al. 1991; Yamashita et al. 2000a), but medetomidine was investigated most intensively (Kamerling et al. 1991; Bettschart-Wolfensberger et al. 1999a,b, 2001; Neges et al. 2003; Kalchofner et al. 2006). It has been shown to provide potent analgesia (Kamerling et al. 1991). Medetomidine’s high clearance rate and short half life necessitate its use as a CRI (Bettschart-Wolfensberger et al. 1999b). A CRI of medetomidine (3.5 µg/kg bwt/h) decreased desflurane MAC by 28% (Bettswart-Wolfensberger et al. 2001). In 40 clinical patients the use of medetomidine CRI (3.5 µg/kg bwt/h) - isoflurane compared to just isoflurane anaesthesia resulted in significantly reduced isoflurane requirements (Neges et al. 2003). A study that compared lidocaine isoflurane with medetomidine isoflurane anaesthesia balanced anaesthesia in 69 clinical patients (Ringer et al. 2007) showed, that following a mean anaesthesia time of 2 h, recovery with medetomidine is longer but of better quality. Maintenance of anaesthesia was also easier with medetomidine and less additional drugs had to be administered in order to maintain a stable plane of anaesthesia. A retrospective study that reported the use of medetomidine isoflurane in 300 clinical cases (Kalchofner et al. 2006) with a mean anaesthesia duration of 146 min and a range of 40–420 min outlines the safety of this drug combination in horses. These authors (Kalchofner et al. 2006) outline that the anaesthetist has to be aware that judgement of depth of anaesthesia is different from other inhalation anaesthesia regimes. Under medetomidine isoflurane anaesthesia eye reflexes should be brisker. Only when nystagmus occurs this is an indicator of insufficient depth of anaesthesia. Further alpha2-adrenoceptor agonists and especially medetomidine increase urinary production. Catheterisation of the urinary bladder is mandatory.

The use of romifidine for balanced anaesthesia was tested in a clinical study in 20 horses (Kuhn et al. 2004). Although horses with romifidine CRI needed less isoflurane, showed more often sufficient spontaneous ventilation and needed less often dobutamine for maintenance of blood pressure, the results of this study should not be overinterpreted. There were 2 different anaesthetists administering isoflurane to effect and the duration of anaesthesia was only 45–80 min. Description of recovery was lacking in this study either.

The use of detomidine CRI for balanced anaesthesia in combination with halothane was used in a study in horses, that also investigated the effect of neurectomy on cardiopulmonary function (Wagner et al. 1992). Duration of detomidine administration was 1 h 40 min–2 h 50 min and the average dose rate 0.18 µg/kg/min. With halothane only, horses had higher heart rates but otherwise no other differences between the groups concerning cardiopulmonary function or recovery were noted.

In conclusion alpha2-adrenoceptor agonists reduce MAC considerably and the only concern is their depressive effect on cardiovascular function. Detailed studies of medetomidine...
showed, that during CRI at a doserate of 3.5 µg/kg bwt/h cardiovascular function is depressed minimally. Large clinical trials showed, that recovery with medetomidine isoflurane is better than with lidocaine isoflurane or S-ketamine isoflurane and in comparison with other regimens seems to be generally of better quality. Data of balanced anaesthesia including other alpha2-agonists is limited.

**Ketamine**

Ketamine is a dissociative agent that induces analgesia, amnesia and immobility without depressing cardiovascular function. During inhaled anaesthesia, racemic ketamine can be administered as incremental i.v. doses (0.1–0.2 mg/kg bwt) or as a constant rate infusion for additional analgesia (Hall et al. 2000). Muir and Sams (1992) investigated racemic ketamine halothane-sparing effects. They found a positive correlation between ketamine plasma concentration and a reduction of up to 37% of halothane MAC. Knobloch et al. (2006) administered racemic ketamine for 2 h to ponies anaesthetised with isoflurane at 1 MAC with a target controlled infusion pump. The initial racemic ketamine loading dose was approximately 0.3–0.4 mg/kg i.v. followed by a linearly decreasing infusion rate of 9–5 mg/kg bwt/h. At these infusion rates, racemic ketamine was found to decrease nociception in a more pronounced fashion than during isoflurane anaesthesia alone (Spadavecchia et al. 2006).

Unfortunately, ketamine as well as its metabolites, have undesirable central nervous system excitatory properties. When ketamine is administered as a sole agent or following prolonged ketamine infusions (>1–2 h) or repetitive i.v. boluses (more than 2 mg/kg bwt total) horses might suffer from these side effects. Ketamine can induce emergence reactions during the anaesthetic recovery period that can turn into a fatal event in horses. In order to minimise such reactions, ketamine infusions can be reduced progressively and/or be stopped 15–20 min before the end of the anaesthetic procedures (Spadavecchia et al. 2002). Thereafter, patients should receive additional post anaesthetic sedation with alpha2-adrenoceptor agonists. S-ketamine was administered to horses undergoing elective arthroscopy as an induction agent at the dose of 1.1 mg/kg bwt to horses sedated with xylazine (1.1 mg/kg bwt). Anaesthesia maintenance was with a CRI of S-ketamine (0.5 mg/kg bwt) and isoflurane in oxygen (Larenza et al. 2008). That balanced regime resulted in better quality of anaesthetic recovery than when horses received double dose of racemic ketamine (induction: 2.2 mg/kg bwt i.v., CRI 1 mg/kg bwt/h). Similarly, Filzek et al. (2003) found that guaifenesin-S-ketamine-xylazine combinations provided better recovery qualities than guaifenesin-racemic ketamine-xylazine combinations.

In conclusion, racemic ketamine or S-ketamine low dose infusions or repetitive boluses might be beneficial when associated with other anaesthetic agents, especially when additional analgesia or improved haemodynamics are required. When ketamine is used, the top-up boluses should not exceed 2 mg/kg bwt or a CRI (1 mg/kg bwt/h) should not be used for anaesthetics longer than 90–120 min, in order to avoid rough recoveries. A CRI should be discontinued 15–20 min prior to transferring the patient to the recovery box. The use of S-ketamine is currently under investigation and might prove advantageous over the use of the racemate. Administration of an alpha2-adrenoceptor agonist before emergence from anaesthesia is highly recommended.

**Opioids**

If in horses the intraoperative use of opioids as part of a balanced anaesthesia regime in horses is useful is debated by many authors. Several experimental and clinical studies have tried to measure influence of opioids on MAC. Morphine, butorphanol or alfentanil did not consistently alter MAC (Matthews and Lindsay 1990; Prevoo et al. 1993; Steffey et al. 2000). Individual horses within each study showed either an increase in MAC, a decrease or no change at all. Following the potent opioid agonists, Fentanyl and Alfentany, individual horses showed bad recoveries full of excitement, especially with higher dose rates. A clinical study (Clark et al. 2008) tested the use of a bolus of morphine (0.15 mg/kg bwt) followed by an infusion of morphine (0.1 mg/kg bwt/h) in comparison to unbalanced halothane anaesthesia. No significant differences between the groups were identified. The same authors further tested the influence of morphine on recovery (Clark et al. 2008). Recovery quality was not different but the horses needed fewer attempts to standing with morphine. Morphine’s influence on MAC of halothane was also tested (Bennett et al. 2004) when it was administered concurrently to xylazine. The result was that xylazine reduced MAC but morphine did not further reduce it.

In conclusion the results of these studies do not provide convincing, objective evidence to support the opinion that systemically administered opioids reduce MAC in a reliable manner or have any other beneficial effects in combination with inhalation anaesthetics (Bennett and Steffey 2002). Nevertheless is the author of the current abstract convinced, that morphine (0.1 mg/kg bwt), administered towards the end of surgery followed by alpha2-adrenoceptor agonists during recovery, helps to smoothen recovery.

**Centrally acting muscle relaxants**

**Guaifenesin**

Guaifenesin is used as an adjunct of balanced anaesthesia in horses to induce muscle relaxation. Guaifenesin has a wide margin of safety and sedative properties that can potentiate other sedative drugs (Schatzmann 1974). Usual clinical doses normally do not affect the diaphragmatic function and preserve the respiratory function, and exert no significant effect over cardiac output and arterial blood pressure (Hubbell et al. 1980).

Spadavecchia et al. (2002), combined guaifenesin (1–0.3 mg/kg bwt/min) with ketamine (39–13 µg/kg bwt/min) to reduce the dose requirement of halothane in horses that presented for a variety of procedures. Surgical anaesthesia was maintained more stable with this combination compared with halothane alone with fewer episodes of subjects moving in response to surgery. The quality of recovery was acceptable and similar to horses receiving only halothane. Similarly, infusions of ketamine-guaifenesin or ketamine-guaifenesin-romifidine allowed for reductions in isoflurane concentrations in horses undergoing several surgical procedures and resulted also in more stable and better cardiovascular performance than when isoflurane was used alone (Nannarone et al. 2005). An infusion of guaifenesin, ketamine and medetomidine to horses anaesthetised with sevoflurane, resulted in better transition and maintenance phases while improving the cardiovascular function and reducing the attempts needed to stand up during the recovery phase, when compared with inhalation of sevoflurane alone (Yamashita et al. 2000b). Thrombophlebitis can occur especially with solutions containing 10% (Hersch et al. 1992) or more guaifenesin and haemolysis has been reported after administering i.v. solutions containing more than 10% of guaifenesin (Grandy and McDonell 1980).

In conclusion, although the effect of guaifenesin alone on MAC has never been quantified nor its analgesic properties, it can be added to balanced anaesthesia protocols. It improves muscle relaxation. Administration to horses at risk of thrombophlebitis as for example severely compromised patients, is not recommended.
**Benzodiazepines**

Recently, benzodiazepines have been incorporated into inhaled balanced regimes aiming to potentiate muscle relaxation and to reduce the delivered concentration of volatile agents. A combined CR of ketamine, medetomidine and midazolam during 4 h of sevoflurane reduced MAC considerably (Kushiro et al. 2005). Horses recovered from anaesthesia without incidents, although ataxia was seen for 15–20 min after standing. Application of head-tail ropes was advised. In order to minimise the post anaesthetic ataxia induced by benzodiazepines, it has been suggested to antagonise their effects by means of administration of specific benzodiazepine antagonists like sarmazenil or flumazenil.

In conclusion, water-soluble benzodiazepines can be administered together with alpha-2 adrenoceptor agonists or ketamine to enhance muscle relaxation. Antagonisation with a specific antagonist is advised to reduce the post operative ataxia. Their role as analgesic coadjuvants remains to be determined.

**References**


Bennett, R., Steffey, E.P., Kollas-Baker, C. and Sams, R. (2004) Influence of morphine administered together with alpha-2 adrenoceptor agonists or sarmazenil or flumazenil. Beta antagonist has been suggested to antagonise their effects by means of reduction of volatile agents. During 4 h of sevoflurane reduction of MAC considerably (Kushiro et al. 2005). Horses recovered from anaesthesia without incidents, although ataxia was seen for 15–20 min after standing. Application of head-tail ropes was advised. In order to minimise the post anaesthetic ataxia induced by benzodiazepines, it has been suggested to antagonise their effects by means of administration of specific benzodiazepine antagonists like sarmazenil or flumazenil.

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**References**


NOTES
Four types of movement may occur during surgery: 1) volitional: the animal is conscious and attempts to get off the table (presumably beginning with a ‘neck-tensing’ righting reflex); 2) involuntary: simple reactive movement results from local spinal reflexes in response to noxious stimuli but may initiate central pattern generators (CPGs) to produce rhythmic activity and thus create the impression of a volitional response; 3) tonus: cyclical, slowly developing weak contractures arising from repetitive asynchronous discharge in the ventral horn of the spinal cord; and 4) muscular contraction resulting as a direct, myogenic response to surgical traction, e.g. contraction of the external cremaster muscle during castration (this depends on mechanisms peripheral to the muscle’s nerve supply and neuromuscular junction and is largely beyond the anaesthetists’ control).

Movement - the best indicator of inadequate anaesthesia - can occur in well-anaesthetised animals when nocistimulation is marked. Slight movement will complicate surgery and compromise surgical cleanliness whilst violent reactions may injure operating room staff and damage equipment. Unexpected movement may be catastrophic in intraocular surgery. Muscle tone necessitates greater traction at the surgical site for exposure which is undesirable; excessive traction increases post operative inflammation (and pain) at the surgical wound.

Surgical muscle relaxation can be produced in 4 ways: 1) ‘deep’ general anaesthesia; 2) ‘spinal’ drugs, e.g. benzodiazepines or GGE; 3) local anaesthetics in the extradural space; and 4) neuromuscular blocking agents (NMBs).

Depolarising and nondepolarising NMBs are seldom used in clinical practice because of: a) unfamiliarity with NMBs; b) the small number of operations requiring profound muscle relaxation; c) the availability of alternative techniques for producing relaxation; d) inexperience with, and, or lack of facilities for positive pressure ventilation (PPV); e) concern with the adequacy of anaesthesia and analgesia; f) complicating effect of positive pressure ventilation (PPV); g) concern with the adequacy of anaesthesia and analgesia; h) lack of accurate, straightforward dosing information; i) absence of sound guidelines for monitoring; j) poor understanding of NMB behaviour in different circumstances; k) fears of prolonged incoordinated recoveries; l) concerns over the effects of PPV on cardiac output and ventilation/perfusion matching; m) difficulty in restoring adequate spontaneous breathing; n) cost of antagonism; o) fear of side effects of antagonism; and p) the widespread use of gentamycin. These are largely unfounded and in general, horses are good subjects for receiving intermediate duration NMBs, providing a means of PPV is available and unconsciousness/analgesia is assured.

Advantages include a reduced requirement for ‘anaesthetic’ and straightforward antagonism. Neuromuscular blocking agents may be used in horses to provide predictable profound relaxation for: a) improved surgical access, e.g. ophthalmic and laparotomy procedures - particularly in sick horses; b) or where persistent, idiopathic movement compromises normal procedures; c) ventilation management (idiopathic, diaphragmatic splinting, deranged blood-gas); or d) as an adjunct to restraint/capture?

The decision to use muscle relaxants in any animal must be determined by the ability to guarantee the provision of adequate levels of insensibility and analgesia. Clinical signs of responses to noxious stimulation may be difficult to identify and interpret. Further prerequisites to the safe administration of NMBs are a knowledge of: a) pathophysiological factors, e.g., extremes of pH or electrolyte levels; b) pharmacological factors, e.g. other anaesthetics, affecting pharmacodynamic and pharmacokinetic behaviour; c) incidence, types and severity of side effects; and d) the effectiveness and safety of antagonism.

Monitoring neuromuscular transmission: allows the effects of NMBs to be titrated with respect to the amount of relaxation required and in the presence of influential pathophysiological and pharmacological factors; provides information on the timing of antagonism. It is not straightforward in horses and frequently cannot be employed when most needed. Baseline values cannot be recorded when nondepolarising drugs are used at induction to anaesthesia to gain control of breathing. The peroneal nerve - pelvic limb digital extensor muscle unit has been studied most extensively, but is only possible with the animal in lateral recumbency. Limb-fixation, i.e. casting, is required for accurate TO4 ratio measurement although the TO4 count can be monitored easily in the uncast limb. The peroneal nerve - pelvic limb digital extensor muscle unit is relatively sensitive to atracurium and cis-atracurium and so its use during ophthalmic surgery may lead to inadequate conditions – the facial and extraocular muscles of the horse being resistant to neuromuscular blockade.

Facial muscle activity may be monitored during laparotomy or in operations where pelvic limb access is difficult. The dorsal buccal branch of the facial nerve has been stimulated and evoked responses in the ‘lip’ muscles measured. However, variability in responses to fixed experimental conditions can be expected because movement in the nasolabial tissue probably reflects contraction of more than one muscle. Responses are frequently artefactual at high stimulation frequencies, including the 2 Hz employed in TO4 stimulation. Furthermore, gravity, i.e. position, affects the nature of evoked response. Mechanomyographic techniques may be applied to the caninus muscle as these are position independent and avoids artefacts associated with multiple muscle contraction. However, equipment must be purpose built and is difficult to set up. The author favours atracurium for use in (sevoflurane or isoflurane-anaesthetised) horses. Doses of 0.1–0.2 mg/kg bwt i.v. cause apnoea in 120–150 s, followed by abolition of T1 at the pelvic limb digital extensors for 6–20 min. Train of 4 stimulation of facial muscles will not reveal diminution in the TO4 count after these doses, although mechanomyography may reveal a modest and short-lived reduction in the TO4R. Doses of mivacurium and vecuronium that are effective in other species appear to have minimal effect upon NMT in horses at the pelvic limb digital extensor muscles. Atracurium-induced blockade is effectively antagonised using edrophonium (0.1 mg/kg bwt) when evidence of spontaneous recovery is present. Autonomic nervous disturbances are not seen when this dose is injected over 2 mins and so atropine coadministration is unnecessary.

Reference