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Case Report Rapport de cas

Anesthetic management of a horse with traumatic pneumothorax

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Abstract — A traumatic pneumothorax and severe hemorrhage were present in a mare with a large thoracic wall defect, lung perforation, and multiple rib fractures. General anesthesia was induced to allow surgical exploration. We describe the anesthetic technique, and discuss the management of the ventilatory, hemodynamic, and metabolic disturbances encountered.

Résumé — Gestion de l'anesthésie générale d'un cheval avec un pneumothorax d'origine traumatique. Nous décrivons le cas d'une jument présentée avec un important déficit de la paroi thoracique d'origine traumatique, une perforation pulmonaire, ainsi que de multiples fractures costales, résultant en un pneumothorax et une hémorragie sévère. L'exploration chirurgicale a été réalisée sous anesthésie générale. Nous détaillons la gestion de l'anesthésie. De plus, les perturbations ventilatoires, hémodynamiques et métaboliques observées sont décrites et leur traitement discuté.

(Traduit par les auteurs)

Pneumothorax refers to the presence of air in the pleural space. It results from any lesion allowing atmospheric air ingress into the interpleural space. Pneumothoraces are classified as closed or opened, depending on thoracic wall integrity, and are rare in horses but have been reported (1–3). The most common reported causes are penetrating thoracic injuries and pleuropneumonia (3). Pneumothorax in horses is normally treated conservatively by packing or closing the wound and draining pleural gas until healing occurs (2,3). However, wounds involving foreign bodies, lung lacerations, or significant thoracic wall defects must be repaired surgically, either under standing surgical or general anesthesia. The few case reports of horses with pneumothorax undergoing general anesthesia fail to emphasize the anesthetic aspect of the management and the observed physiological disturbances (1,2).

The aim of this report is to describe the anesthetic management of, and the physiological perturbations observed in an adult horse presented with a traumatic open pneumothorax, lung lacerations, and diaphragmatic hernia.

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Case description

A 7-year-old Irish Sport mare, estimated body mass 500 kg, was presented at the Royal (Dick) School of Veterinary Studies for the investigation of a wound on the right thoracic and abdominal wall. At admission the mare was alert but quiet. A large wound extending from the right axilla caudo-dorsally over approximately 40 cm was visible on the thoracic wall. Palpation revealed extensive muscle laceration and the fracture of at least 3 ribs. Thoracic and abdominal cavity involvement was suspected. Arterial hemorrhage was limited by using a compressive thoraco-abdominal bandage. The horse's owner was unable to quantify the blood lost before admission, although approximately 5 L were shed thereafter. General anesthesia was scheduled to allow surgical exploration, wound debridement, and wound repair.

Pre-anesthetic examination revealed a respiratory rate (fR) of 40 breaths/min and pulse rate of 65 pulsations/min; oral mucous membranes were pale pink and the capillary refill time was 1.5 s. The facial arterial pulse was regular and of good quality. Thorough auscultation of the lungs and heart was not possible because of the large bandage covering the thorax and abdomen. Subcutaneous emphysema was present over the thorax and neck.

A 13-gauge cannula was inserted in the external jugular vein. Venous blood analysis revealed a hematocrit of 0.37 L/L and total solids of 67 g/L. In an attempt to replace lost circulating volume, hypertonic saline solution 7.2% (Vetivex 20; Arnolds Veterinary Products, Shrewsbury, UK) was administered intravenously (IV) at 5 mL/kg of body weight (BW) over 10 min. The mare was then sedated with romifidine (Sedivet; Boehringer Ingelheim, Berkshire, UK), 0.08 mg/kg BW, IV followed by morphine (Morphine Sulphate Injection; Martindale Pharmaceuticals, Romford, UK), 0.12 mg/kg BW,

IV. Ten minutes later, anesthesia was induced with IV ketamine (Vetalar V; Pharmacia & Upjohn Animal Health Ltd, Crawley, UK), 2.2 mg/kg BW and diazepam (Diazepam Injection; Hameln Pharmaceuticals, Gloucester, UK), 50 μg/kg BW. Induction was assisted by restraining the mare with her left flank along the wall whilst she descended to the floor of the padded induction box. When fully relaxed, the horse was carefully rolled into right lateral recumbency.

Although induction was smooth, the animal became immediately dyspneic (supranormal thoracic excursions with nostril "flaring") and tachypneic (60 breaths/min). The trachea was intubated with a 30 mm cuffed PVC endotracheal tube and the cuff was inflated. An additional dose of ketamine (Vetalar; Pharmacia & Upjohn Animal Health), 0.3 mg/kg BW, IV and midazolam (Hypnovel; Hameln Pharmaceuticals, Gloucester, UK), 5 μg/kg BW, IV were administered to "deepen" anesthesia. The mare was mechanically hoisted, positioned in dorsal recumbency on a padded operating table and the endotracheal tube immediately connected to the circle breathing system of a large animal anesthetic machine with integrated ventilator (Tafonius; Vetronic service/Hallowell EMC, Abbotskerswell, UK) pre-filled with 6% sevoflurane (Sevoflo; Abbott Laboratories, Maidenhead, UK) in oxygen. Intermittent positive pressure ventilation (IPPV) was immediately initiated with a tidal volume of 6.0 L at a rate of 6 breaths/min.

Anesthesia was maintained with sevoflurane in oxygen and an IV constant rate infusion of morphine of 0.1 mg/kg BW/h. Flunixin meglumine was administered (Flunixin; Norbrook Laboratories, Carlisle, UK), 1.1 mg/kg BW, IV. Isotonic poly-ionic solution (Vetivex 11; Arnolds Veterinary Products) was infused as quickly as gravity allowed via the jugular cannula, and 5 L were administered over the 55 min of anesthesia. A 20-gauge cannula was placed in the left facial artery for the continuous monitoring of arterial pressure and collection of blood samples. Blood gas analysis was performed with an OPTI AVL Critical Care Analyzer (Osmetech, Roswell, Georgia, USA). The arterial blood pressure [systolic (SAP), mean (MAP), diastolic (DAP)], heart rate (HR), electrocardiogram (ECG), end-tidal partial pressure of carbon dioxide ($PE'CO_2$), inspired fraction of oxygen (FIO_2), end-tidal concentration of sevoflurane ($FE'SEVO$) and peak inspiratory pressure (PIP) were continuously monitored using a multi-channel physiological monitor (Datex-Engstrom Compact; Datex-Engstrom, Tewksbury, Massachussetts, USA), and recorded every 5 min along with specific ventilator settings: fR, tidal volume (V_T).

Removal of the dressing revealed that the hemorrhage had stopped. A right hemi-pneumothorax was obvious, and no lung sounds were audible during auscultation of the left hemithorax. The smell of sevoflurane at the surgical site indicated lung perforation had occurred. Arterial blood gas analysis carried out 10 min after induction revealed hypoxemia ($PaO_2 < 60$ mmHg) with a combined metabolic and respiratory acidosis (Table 1). At this time the average PIP was 7 cm H_2O. The V_T was subsequently increased to 7.5 L and respiratory rate increased to 9 breaths/min. The average PIP increased to 10 cm H_2O. Ten actuations of salbutamol aerosol (Ventolin; Allen & Hanburys, Greenford, UK), 2 μg/kg BW were administered into the

Table 1. Arterial blood gas analysis, FIO_2 and $PE'CO_2$ at 10, 30, and 50 minutes after induction of anesthesia in a horse with a pneumothorax

Time post induction of general anesthesia	10 min	30 min	50 min
pH	7.29	7.32	7.38
$PaCO_2$ (mmHg)	59	54	45
PaO_2 (mmHg)	49	73	89
Base excess (mmol/L)[a]	−0.2	0.5	0.5
HCO_3^- (mmol/L)[a]	27.4	27.3	25.9
SaO_2 (L/L)	0.77	0.9	0.96
FIO_2 (L/L)	0.87	0.77	0.91
$PE'CO_2$ (mmHg)	42	43	35

[a] Reference values for anesthetized horses on the blood gas analyzer used: Base excess (4.0 ± 2.0 mmol/L); HCO_3^- (30.8 ± 2.3 mmol/L) [determined on 175 anesthetized horses, ASA status 1 or 2, with a normal pH (7.35 to 7.45); unpublished data].

endotracheal tube at the onset of the ventilator's inspiratory cycle. At 30 min post-induction of general anesthesia, hypoxemia had resolved although PaO_2 was still low, and despite some improvement, acidemia remained uncorrected (Table 1). Consequently, the V_T was increased to 9.0 L which caused PIP to increase to 12 cm H_2O and the blood pressure to fall: SAP 71 mmHg; MAP 49 mmHg; DAP 35 mmHg. Hypotension was treated with 30 mg ephedrine IV (Ephedrine Hydrochloride Injection; Martindale Pharmaceuticals) and the infusion of 500 mL of 6% hydroxyethyl starch 130/0.4 (Voluven; Fresenius Kabi, Runcorn, UK), and within 5 min, the blood pressure was restored to physiological limits (SAP 83–85, MAP 61–65, DAP 49–55 mmHg) where it was maintained for the remainder of surgery. A third arterial blood gas sample analyzed 50 min after induction revealed that all variables were within acceptable limits (Table 1).

Surgical exploration revealed a right-sided pneumothorax, a 25-cm tear in the thoracic wall (extending caudal to the axilla over 5 ribs in the ventral third of the thorax) and comminuted fractures of the 5 ribs involved in the wound. These fractures had lacerated the right lung lobe and the diaphragm along its ventral surface. Apprised of the poor prognosis, the owner consented to euthanasia which was carried out in the anesthetized animal.

Discussion

When the thoracic cage is broached, air enters the pleural space down the pressure gradient generated by the subambient intra-pleural pressure. This varies from −5 cm H_2O to −10 cm H_2O during resting breathing but can fall as low as −30 cm H_2O during forceful breathing (4). Accumulating air disrupts the cohesion between the visceral and parietal pleura, causing the lung to collapse under the combined effects of gravity, surface tension, and elastic forces. The lack of intrapleural negative pressure renders respiratory muscle contraction inefficient in expending lung during inspiration. This results in a decreased functional residual capacity, airway closure, and uneven distribution of inspired gas which, collectively, promotes hypoxemia and increases the alveolar-arterial oxygen tension difference as a result of intrapulmonary shunting and ventilation-perfusion mismatch. In healthy subjects, compensatory mechanisms such as tachypnea (5) and hypoxic pulmonary vasoconstriction

(HPV) (6–8) improve alveolar ventilation and arterial blood oxygenation, respectively. During anesthesia with volatile agents these compensatory mechanisms are obtunded and the ability to oxygenate blood can dangerously deteriorate (6,9–11). In anesthetized horses, positioning in dorsal and (to a lesser extent) lateral recumbency contributes importantly to arterial hypoxemia because of increased ventilation/perfusion mismatching (12).

Blood gases were not measured before induction of anesthesia in the case described. Had this been otherwise, the severity of the respiratory dysfunction could have been quantified. However, blood gas data would probably not have altered case management because severe compromise could be assumed on the basis of the injuries and the suspicion of pneumothorax. The prompt induction of anesthesia without extensive pre-operative laboratory testing allowed immediate control of ventilation, the provision of high inspired oxygen fraction (FiO_2), a rapid assessment of the injury and the establishment of a prognosis.

Positive pressure ventilation (PPV) is mandatory in cases of severe pneumothorax. When PPV is imposed, a V_T of 10 to 15 mL/kg BW to healthy horses is normally achieved with PIP values of 20 to 35 cm H_2O (13). However, there are no data reporting the PIP required for ventilating the lungs of adult horses during thoracotomy. In the case described, the mean PIP remained relatively low despite increasing the V_T (from 14 to 18 mL/kg BW). This normally occurs when thoracic or lung compliance increases; however, in our case the most likely explanation is the loss of delivered V_T through the perforated lung. Increasing fR, V_T, and FiO_2, and administering salbutamol were associated with an improvement in PaO_2 and $PaCO_2$ (Table 1). This was not unexpected; salbutamol increases PaO_2 in hypoxemic horses during mechanical ventilation (14). On the other hand, increasing V_T during IPPV in horses does not change the ventilation ratio between different lung regions, and may not improve PaO_2 when hypoxemia results from intrapulmonary shunting (15). Consequently, the improvement of PaO_2 as well as $PaCO_2$ following the increase in V_T in the current case, suggests that the hypoxemia observed was at least partly due to severe hypoventilation caused by lung collapse and leakage of inspired gas from the lacerated lung. Blood-gas derangements caused by gas leakage may have been minimized by isolating the lacerated lung through surgical ligation or insertion of a bronchial blocker; both would have allowed effective ventilation in the left (undamaged) lung. Bronchial blockers have been used in horses (16) but the required equipment was unavailable. Surgical isolation was not attempted as the decision to conduct euthanasia had already been taken.

Hypoxic pulmonary vasoconstriction (HPV) is the physiological response to alveolar hypoxia that redirects blood from severely hypoxic to less hypoxic alveoli. In cases of pneumothorax, it is probably an important mechanism for improving ventilation-perfusion matching. The effect of sevoflurane on HPV and pulmonary shunt fraction is unclear. *In vitro* studies suggest that it decreases HPV in a dose related manner (17) but clinical concentrations in anesthetized dogs have no significant effect on HPV (18). Studies in humans have failed to demonstrate the superiority of intravenous anesthetics (propofol) over sevoflurane in terms of improving PaO_2 or lowering shunt frac-

tion (8). Unfortunately, the relevance of these results in horses is unknown as HPV response is variable between species and, the impact of anesthetic agent on HPV has not been studied in horses. There is evidence, however, for an inverse relationship between HPV and cardiac output which implies that the net effect of volatile agents on HPV is the balance between direct inhibition, and indirect stimulation arising from reduced cardiac output (9). In short, the choice of anesthetic may be less important than the preservation of hemodynamic variables within suitable, though undefined, ranges.

In this case, the persistence of abnormally low PaO_2 despite the administration of a high FiO_2 (Table 1) suggests the presence of an intrapulmonary right to left shunt. Severe right-to-left shunting impairs pulmonary uptake of volatile anesthetic agents (19) to the extent that the inspired fraction of the anesthetic agent must be increased to achieve a desired depth of anesthesia (20). Despite the intrapulmonary shunt, anesthesia was successfully maintained with $Fi'sevo$ and $Fe'sevo$ levels indistinguishable from those required in other horses undergoing non-thoracic surgery in dorsal recumbency at this institution. This may be explained by the use of partial intravenous anesthesia, i.e., morphine infusion. Morphine continuous rate infusion decreases volatile anesthetic requirement in several species (21,22), although evidence for a similar effect in horses is experimentally inconclusive (23). In the clinical context, Clark et al (24) found that during halothane anesthesia, horses that received a constant rate infusion of morphine tended to require fewer and lower doses of additional anesthetic drug compared with horses that did not receive morphine.

In the case described, the blood losses were estimated at 5 L (~10 mL/kg). Considering that the average blood volume in "hot blooded" horses is 109.6 mL/kg (isotope dilution technique; 25), this would represent a blood loss of 9% of the total blood volume. Acute hypovolemia was treated pre-operatively with intravenous hypertonic saline (5 mL/kg BW), which was given in an attempt to ameliorate the hemodynamic effects of anesthetics and body position. In experimentally induced hemorrhagic shock in anesthetized horses, administration of 2 L (3.8 to 4.5 mL/kg) of 7.2% hypertonic saline resulted in an average increase of 6 L of total blood volume, and restored cardiac output, stroke volume, and the overall hemodynamic status to the pre-hemorrhage values for up to 2 h (26). Consequently, we believe that the 2.5 L administered in our case improved the hemodynamic status and contributed to maintain normotension. However, resuscitation with hypertonic saline during uncontrolled hemorrhage can induce re-bleeding (27). This probably results from disruption of the hemostatic plug when blood pressure is restored, coinciding with increased blood flow, vessel dilatation, decreased viscosity, and clotting factor dilution (28). Based on experimental trials, several strategies have been recommended in order to limit rebleeding including permissive hypotension (29) and delayed fluid resuscitation using slow infusion rates (30). In the case reported here, hypertonic saline administration was delayed over 30 min after a packing and compressive bandage was applied to the wound. No major arterial bleeding was observed when the compressive bandage was removed, suggesting an efficient hemostatic plug had formed.

In spite of pre-operative hypertonic saline administration and a progressive decrease in $Fe'sevo$, the horse became hypotensive 30 min after induction of anesthesia. This may have resulted from: 1) the waning effect of infused hypertonic saline; 2) occult ongoing hemorrhage; or 3) a delayed romifidine-induced decrease in blood pressure. Ephedrine 30 mg (~0.06 mg/kg BW) and the simultaneous infusion of 500 mL of Voluven (~1 mL/kg BW) caused a satisfactory and sustained increase in blood pressure during the last 15 min of anesthesia. This was expected, as in hypotensive anesthetized horses, 0.06 mg/kg of ephedrine increases the mean arterial blood pressure for at least 15 min (31). The effect of hypertonic saline is short-lived and the combined use of hydroxyethyl starch solution (HES) is sometimes recommended in order to sustain its hemodynamic effect. In this case, Voluven was preferred over other proprietary HES's for intravascular volume replacement because it has less effect on coagulation (32) and allows better tissue oxygenation (33) — albeit in humans. To our knowledge, its effects have not been evaluated in horses. The total volume administered was relatively small (1 mL/kg BW), but administration was discontinued when blood pressure became acceptable. Volumes ranging from 5 to 20 mL/kg BW of 6% HES have been reported to have a significant intravascular volume expansion effect in horses, but lower volumes have not been evaluated (34,35). The plasma volume expansion resulting from infusion of 6% HES is theoretically slightly greater than 100% of the infused volume, so in this case, an increase in total blood volume of approximately 1%, which would have been modest, if not negligible. The plasma volume expansion effect of Voluven is unknown in the horse, but is reported to be greater than that of larger molecular weight HESs in humans (36). In conclusion, the hemodynamic benefits of Voluven in the current case remain undetermined.

In conclusion, anesthetizing horses with acute traumatic pneumothorax is complicated by an aggravation of the ventilation/perfusion mismatching which is already an inevitable consequence of general anesthesia and recumbency in this species. Precise control and monitoring of the ventilatory variables such as V_T, fR and PIP helped in the management of this case. The use of high V_T together with aggressive cardiovascular support improved the metabolic and ventilatory status of the animal. Adding an opioid infusion may have reduced the sevoflurane requirement allowing a safe and predictable anesthetic depth despite the decreased uptake of volatile agent resulting from lung perforation.   CVJ

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