

Anesthesia in a horse with diaphragmatic hernia

Anesthesie bij een paard met hernia diafragmatica

A.J.H.C. Michielsen, A. Binetti, J. Brunsting, F. Gasthuys, S. Schauvliege

Vakgroep Heelkunde en Anesthesie van de Huisdieren
Faculteit Diergeneeskunde, Universiteit Gent, Salisburylaan 133, B-9820 Merelbeke, België

anneleen.michielsen@ugent.be, stijn.schauvliege@ugent.be

ABSTRACT

An eight-year-old Thoroughbred mare was presented with acute colic symptoms. Clinical and ultrasonographic examination revealed a suspicion of diaphragmatic hernia, which was confirmed during an emergency midline laparotomy performed the same day. Patients with diaphragmatic hernia pose a challenge for the surgeon and the anesthesiologist, because of the disturbed function of the diaphragm and the displacement of the abdominal organs into the thoracic cavity. Achieving optimal ventilation and oxygenation without causing damage to the lungs is not simple. In this case, assisted-controlled, intermittent positive pressure ventilation with a low tidal volume, low pressure and relatively high respiratory rate was applied. Alongside the difficulties during ventilation, the patient was cardiovascularly compromised. Due to the extent and position of the hernia, euthanasia was performed after obtaining the owner's consent during surgery.

SAMENVATTING

Een acht jaar oude volbloedmerrie werd aangeboden op de Faculteit Diergeneeskunde (UGent) met acute kolieksymptomen. Een hernia diafragmatica werd vermoed aan de hand van het klinisch en echografisch onderzoek. Deze diagnose werd nadien bevestigd tijdens een spoedlaparotomie uitgevoerd diezelfde dag. Patiënten met een hernia diafragmatica vormen een uitdaging voor zowel de chirurg als de anesthesist omwille van de verstoorde functie van het middenrif en de verplaatsing van de abdominale organen in de thorax. Het bekomen van optimale ventilatie en oxygenatie zonder schade aan de longen toe te brengen, is niet vanzelfsprekend. In de voorliggende casus werd een geassisteerde-gecontroleerde ventilatie met een intermitterend positieve druk met een laag tidaal volume, lage druk en een iets hogere ademhalingsfrequentie toegepast. Naast problemen tijdens de ventilatie was de patiënt cardiovasculair gecompromitteerd. Gedurende de operatie werd, met de eigenaars toestemming, besloten het paard te euthanaseren gezien de ernst en locatie van de hernia.

INTRODUCTION

The diaphragm consists of a central fibrous and a peripheral muscle part. Its main function is to contribute to respiration. In addition, the diaphragm separates both the abdominal and the thoracic cavities from each other (Maish, 2010; McMaster et al., 2014). Important structures cross the diaphragm: vena cava, aorta and esophagus (Maish, 2010; McMaster et al., 2014). During inspiration, the diaphragm and external intercostal muscles contract, resulting in an expansion of the thorax. The intra-thoracic pressure

diminishes and air flows into the lungs. During expiration, which is mainly passive, the diaphragm relaxes and air flows out of the lungs. However, there is an active component at the end of every expiration in horses, consisting of a small upward movement of the diaphragm. Following this active contraction, the diaphragm relaxes, whereby air flows passively into the lungs during the first part of inspiration (Maish, 2010; Sprayberry and Barret, 2015).

A diaphragmatic hernia is either a congenital or acquired interruption of the diaphragm (Kummer and Stick, 2012; McMaster et al., 2014), which is a rare

type of abdominal hernia in the horse (Clutton et al., 1992; Hart et al., 2009; Kelmer et al., 2012; Kummer and Stick, 2012; Auer, Röcken et al., 2013). An acquired diaphragmatic hernia can be caused by either trauma (Santschi et al., 1997; Hassel, 2007; Kelmer et al., 2012, Röcken et al., 2013; Sprayberry and Barret, 2015) or an acute rise of the intra-abdominal pressure (Hassel, 2007; Kelmer et al., 2012; Röcken et al., 2013; Sprayberry and Barret, 2015). This type of defect is often larger than a congenital hernia (Auer et al., 2012; Röcken et al., 2013). Usually, the tears are located ventrally, just above the processus xiphoideus of the sternum (McMaster et al., 2014). During the acute phase of the trauma, the acquired lesions have rough and irregular borders combined with bleeding and inflammation. The more chronic lesions of an acquired diaphragmatic hernia have smoother and thicker borders with fibrous tissue. These chronic lesions are sometimes combined with visceral adhesions (McMaster et al., 2014).

Patients suffering from a diaphragmatic hernia may be presented with a history of chronic, repeated colic symptoms, but also with severe acute respiratory symptoms (Hassel, 2007; Hart et al., 2009; Kelmer et al., 2012; Kummer and Stick, 2012; Sprayberry and Barret, 2015). Additionally, exercise intolerance, presentation of an apathetic state and sudden death may be other clinical signs associated with diaphragmatic hernia in horses (Santschi et al. 1997; Hassel, 2007; Hart et al., 2009; McMaster et al., 2014; Sprayberry and Barret, 2015). During thoracic auscultation, intestinal borborygmi may often be heard in the thorax (Kelmer et al., 2012; Kummer and Stick, 2012). To improve diagnostics, intestines can be visualized in the thoracic cavity using ultrasonography (Hart et al., 2009; Kelmer et al., 2012; Kummer and Stick, 2012; Sprayberry and Barret, 2015). Besides ultrasonography, radiology may also be used to aid in establishing a likely diagnosis (Hassel, 2007; Kummer and Stick, 2012; McMaster et al., 2014; Sprayberry and Barret, 2015). However, the definitive diagnosis can only be made during an exploratory laparotomy (Hassel, 2007; Hart et al., 2009; Kelmer et al., 2008; Kummer and Stick, 2012; McMaster et al., 2014; Sprayberry and Barret, 2015) and it is also the only option to repair the diaphragm and to treat the patient. This type of hernia has a reserved prognosis (Hart et al., 2009; Kummer and Stick, 2012; Röcken et al., 2013), which is mainly determined by the location and size of the lesions, as well as by the involvement and condition of the intra-abdominal and -thoracic organs (Hart et al., 2009; McMaster et al., 2014).

CASE REPORT

An eight-year-old Thoroughbred mare (BWT 446 kg) was referred to the Faculty of Veterinary Medicine (Ghent University) for acute colic symptoms after be-

ing treated once with natriummetamizole 25 mg kg⁻¹ combined with butylhyoscine bromide 0.2 mg kg⁻¹ (Buscopan Compositum ad us. vet., SCS Boehringer Ingelheim Comm V, Brussels, Belgium) by a local veterinarian at home before transport. The day before, the horse had already been examined by the veterinarian for symptoms of apathy, but clinical examination and general blood work hadn't revealed a conclusive diagnosis for the local veterinarian at that time.

The horse was presented at the clinic with sinus tachycardia (88 beats per minute), a regular pulse of good quality, an abdominal respiration pattern with a frequency of 36 breaths per minute (tachypnea), pink mucous membranes and both normal skin turgor and capillary refill time on clinical examination. The rectal temperature was 38°C and the horse had a diminished lumbar reflex. Increased respiratory sounds and reduced gut sounds were respectively heard during auscultation of both sides of the thorax and the abdomen. On a jugular venous blood sample were only some mild deviations from the reference values visible: packed cell volume: 38% (35-45%); pH: 7.47 (7.35-7.45); base excess: 7.8 mEqL⁻¹ (-5 - +5 mEqL⁻¹); glucose: 188 g dL⁻¹ (80-180 g dL⁻¹); sodium: 131 mmol L⁻¹ (135-150 mmol L⁻¹); potassium: 2.8 mmol L⁻¹ (3.0-5.9 mmol L⁻¹); calcium: 1.28 mmol L⁻¹ (1.4-1.7 mmol L⁻¹); lactate: <1 mmol L⁻¹ (<2 mmol L⁻¹). The colon and cecum could not be palpated rectally. Ultrasonography revealed a large amount of free fluid in both the thoracic and abdominal cavity, with an irregular delineation of the diaphragm between the stomach and ventral side of the abdominal wall. Abdominocentesis was performed after a small stab incision in the linea alba at the lowest level of the ventral abdomen and a small amount of serohemorrhagic fluid was collected with the aid of a blunt teat cannula. Further examination of the transudate revealed a white blood cell count of 2.3*10⁹ L⁻¹ (< 3.5*10⁹ L⁻¹), a total protein concentration of 15 g L⁻¹ (< 20 g L⁻¹) and a lactate of 2.4 mmol L⁻¹ (<4 mmol L⁻¹). During thoracocentesis, which was performed under ultrasonographic guidance with a 19G needle (Agani™ needle, Terumo Europe N.V., Hamburg, Germany) and a 10 cc syringe (Terumo 10 cc syringe without needle, Terumo corporation, Laguna, the Philippines), pure blood was aspirated. Based on these findings, the presence of a diaphragmatic hernia was suspected. After obtaining the owner's consent, the horse was prepared for an emergency laparotomy. A catheter (Intraflon 2, 12G, Vygon, Ecoen, France) was placed in the left jugular vein. Gentamicin 6.6 mg kg⁻¹ (Genta-kel 5%, Kela NV, Hoogstraten, Belgium), benzylpenicillin-sodium 15 000 I.U. kg⁻¹ (Natrium-Penicilline G, Kela Pharma, Sint-Niklaas, Belgium) and flunixin meglumine 1.1 mg kg⁻¹ (Emdoflunin 50, Emdoka BVBA, Hoogstraten, Belgium) were administered intravenously. A nasogastric tube was placed in the right ventral nasal meatus to evacuate fluid and gas from the stomach. Subsequently, the horse was sedated with intravenous

administration of 0.7 mg kg^{-1} xylazine (Xyl-M, VMD, Arendonk, Belgium). After five minutes, anesthesia was induced using an intravenous combination of ketamine 2.2 mg kg^{-1} (Ketamidor, Richter Pharma AG Wels, Austria) and midazolam 0.06 mg kg^{-1} (Midazolam B Braun 5 mg mL^{-1} , Braun, Melsunger, Germany). After induction, the horse's trachea was intubated with a 28 mm-outer-diameter endotracheal tube (Rusch Teleflex endotracheal tube red rubber, Kernen, Germany) and then the horse was placed in dorsal recumbency on the padded operation table. General anesthesia was maintained with isoflurane (IsoFlo, Aesica Queenborough Limited, Kent, U.K.) in oxygen (4 L minute^{-1}) and medical air ($0.5 \text{ L minute}^{-1}$) via a half-closed circle system. Intermittent positive pressure ventilation was immediately applied in an assisted-controlled mode, trigger pressure of $-3 \text{ cm H}_2\text{O}$, with a respiratory rate of 10 breaths per minute, a tidal volume of 11 mL kg^{-1} and a peak inspiratory pressure limit of $20 \text{ cm H}_2\text{O}$ (Smith Respirator, BDO Medipass Medical Divison and Dräger Sulla 909 V, Dräger werk AG, Lübeck, Germany). The resultant tidal volumes varied between 4 and 4.8 L . An infusion with lactated Ringer's solution (Vetivex 5000 mL, Dechra Limited, North Yorkshire, U.K.) (10 mL kg h^{-1}) was immediately started together with a constant rate infusion of lidocaine $2 \text{ mg kg}^{-1} \text{ h}^{-1}$, preceded by an intravenous

bolus of 1.5 mg kg^{-1} administered over 15 minutes (Laocaïne 20 mg mL^{-1} , Intervet, Beaucauzé, France). A butterfly needle (Surflo Winged infusion Set 21G, Leuven, Belgium) was placed in the right facial artery for monitoring arterial blood pressure (Datex-Ohmeda S/5, Helsinki, Finland), packed cell volume and blood gas analysis (Radiometer ABL5, USA). Further monitoring included electrocardiography, pulse oximetry and measurement of body temperature, inspired and expired concentrations of oxygen, carbon dioxide and isoflurane (Datex-Ohmeda GE Healthcare, Helsinki, Finland OY).

Because of the low arterial blood pressure, i.e. $[80/50 (65) \text{ mmHg (systolic/diastolic (mean))}]$ and weak pulsation of the facial artery, an intravenous constant rate infusion of dobutamine hydrochloride (Dobutrexmylan, Synthron BV, Nijmegen, the Netherlands) was started at a rate of $0.75 \text{ } \mu\text{g kg minute}^{-1}$ (Figure 1). Within five minutes, the arterial pressure dropped to $65/40 (45) \text{ mmHg}$. Then, the dobutamine administration rate was increased to $1 \text{ } \mu\text{g kg minute}^{-1}$ and an intravenous bolus of phenylephrine $2 \text{ } \mu\text{g kg}^{-1}$ (Phenylephrine 10 mg mL^{-1} , Beaucon Pharmaceuticals, Kent, U.K.) was administered. The mean arterial pressure (MAP) improved to 70 mmHg , but decreased again to 55 mmHg after only two minutes. A second intravenous bolus of phenylephrine $2 \text{ } \mu\text{g kg}^{-1}$

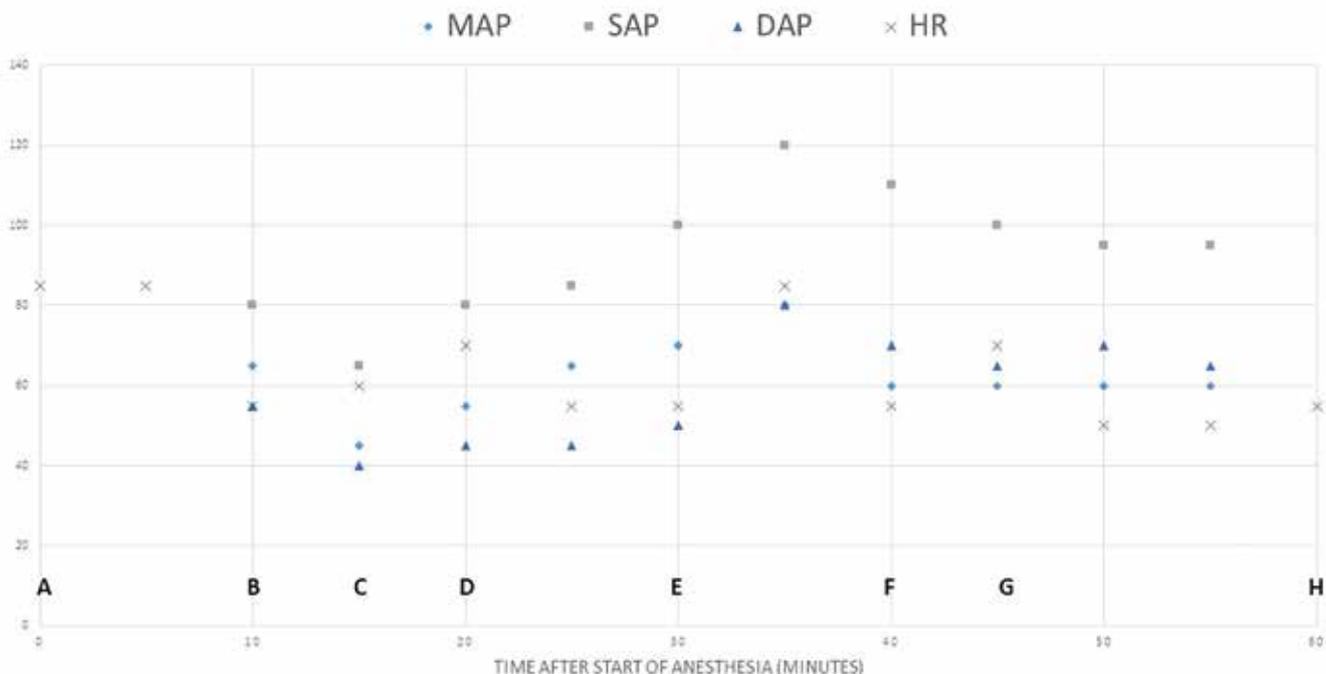


Figure 1. Graphic representation of the monitoring during a case of diaphragmatic hernia in a horse.

MAP= mean arterial pressure, SAP = Systolic arterial pressure, DAP= Diastolic arterial pressure, HR= Heart rate. The blood pressure is expressed in mmHg and the heart rate in beats per minute. A. Bolus of lidocaine 1.5 mg kg^{-1} I.V. over 15 minutes, B. Start dobutamine CRI at $0.751 \text{ } \mu\text{g kg}^{-1} \text{ min}^{-1}$, C. Start CRI Lidocaine IV $2 \text{ mg kg}^{-1} \text{ h}^{-1}$ I.V., increase in dobutamine CRI to $1 \text{ } \mu\text{g kg}^{-1} \text{ min}^{-1}$ and bolus of $2 \text{ } \mu\text{g kg}^{-1}$ of phenylephrine I.V., D. Second bolus of $2 \text{ } \mu\text{g kg}^{-1}$ of phenylephrine I.V. E. Bolus of 150 mg ketamine I.V. due to nystagmus at surgical incision, 5.5 mL kg^{-1} of synthetic colloids over 15 minutes and decrease dobutamine CRI to $0.56 \text{ } \mu\text{g kg}^{-1} \text{ min}^{-1}$ F. $100 \text{ } \mu\text{g}$ ipratropium bromide through aerosol, G. Bolus of 100 mg ketamine I.V. due to nystagmus, H. Euthanasia with 50 mg kg^{-1} sodium pentobarbital I.V.

kg⁻¹ was administered. After a short improvement of the MAP to 85 mmHg, it decreased to 65 mmHg and remained stable at this level until the patient was euthanized. The heart rate increased further from 55 to 85 beats per minute 35 minutes after the start of the dobutamine CRI, coinciding with the time of surgical incision. Nystagmus was observed at this time and a bolus of 0.34 mg kg⁻¹ of ketamine was administered. The dobutamine administration rate was decreased to 0.56 µg kg⁻¹ minute⁻¹, a second catheter was placed in the right jugular vein and synthetic colloids (5.5 mL kg⁻¹ over 15 minutes) (Geloplasma 500 mL Fresenius Kabi NV, Schelle, Belgium) were administered followed by lactated Ringer's solution.

An arterial blood gas analysis five minutes after induction of anesthesia revealed a modest degree of hypercapnia (arterial partial pressure of carbon dioxide - PaCO₂ - 60 mmHg), severe hypoxemia (arterial partial pressure of oxygen - PaO₂ - 28 mmHg, arterial oxygen saturation - SaO₂ - 51%) and metabolic acidosis (base excess 10 mEqL⁻¹; pH: 7.1; HCO₃⁻: 31 mmol L⁻¹). Despite the relatively high PaCO₂, the end tidal CO₂ concentration was normal at 40 mmHg. This difference between the arterial and alveolar CO₂ concentrations indicates the presence of alveolar dead space. The ventilation was adapted to a tidal volume of 9 mL kg⁻¹, reaching maximal peak inspiratory pressures of 20 cm H₂O and a respiratory rate of 12 breaths/minute. The inspired fraction of oxygen (FIO₂) was increased from 50 to 60% by reducing the medical air in the gas mixture. After 30 minutes, the oxygenation and ventilation parameters evaluated by means of arterial blood gas analyses, had not improved significantly (average PaO₂ of 30 mmHg and PaCO₂ of 60 mmHg over five arterial blood gas analyses), and ipratropiumbromide 100 µg (Atrovent 20mcg aerosol, Boehringer Ingelheim Pharma GmbH and Co. KG, Ingelheim am Rhein, Germany) was administered as an aerosol at the level of the Y-piece of the breathing circuit, at the beginning of inspiration. Although the SaO₂ and PaO₂ improved slightly ten minutes after the administration of ipratropium bromide (59% and 34 mmHg, respectively), the PaCO₂ increased to 68 mmHg and the packed cell volume decreased from 41% at the time of the first arterial blood gas sample to 36%. A couple of boluses of ketamine were administered at 30 and 45 minutes after the start of the anesthesia (150 mg and 100 mg, respectively), because of nystagmus and an increase in heart rate.

During surgery, the diagnosis of a diaphragmatic hernia was confirmed. A tear in the diaphragm was palpated from the left dorsal attachment on the abdominal wall towards the centre of the diaphragm until the level of the esophagus. The stomach was dilated and completely displaced into the thoracic cavity, embedded in a large amount of blood. Considering the position and size of the tear and the reserved prognosis, the horse was euthanized 60 minutes after induction of anesthesia with sodium pentobarbital

50 mg kg⁻¹ I.V. (Release 300 mg mL⁻¹, WDT, Garbsen, Germany). During pathological examination, the presence, localization and size of the diaphragmatic hernia were confirmed, with displacement of the stomach, part of the spleen and the duodenum into the thoracic cavity.

DISCUSSION

Anesthesia of horses requiring emergency surgery for acute colic can be quite challenging (Boesch, 2013). Any horse is predisposed to develop hypercapnia (PaCO₂ > 40 to 45 mmHg) and hypoxemia (PaO₂ < 60 to 70 mmHg) during anesthesia (Robertson, 2002; Moens, 2013). In case of a diaphragmatic hernia, these deviations are inevitable. The respiratory function is grossly disturbed and patients are often presented with respiratory symptoms such as tachypnea, which was also the case with this patient (36 breaths per minute). In addition, compression atelectasis of the lungs, which is normally caused by relaxation of the diaphragm during anesthesia, and compression of the lungs by the abdominal organs during anesthesia, are increased significantly in the presence of a diaphragmatic hernia (Clutton et al., 1992; Hassel, 2007; Kelmer et al., 2008; Moens, 2013). In addition, extreme pain and hemodynamic, acid-base and electrolyte imbalances may cause multiple anesthetic complications in these patients (Boesch, 2013). The anesthetist's primary aim is to maintain an optimal ventilation and adequate blood pressure (Clark et al., 2014), not only during maintenance of anesthesia, but also during preoperative preparation, premedication and induction of anesthesia.

Preparation of the patient and equipment prior to anesthesia is important in order to prevent complications (Clarke et al., 2014). Stabilization before anesthesia must be accomplished as much as possible (Boesch, 2013). Any fluid deficits should, at least partly, be addressed already before anesthesia. In the present case, the pre-anesthetic venous blood sample did not reveal important electrolyte or PCV imbalances requiring fluid resuscitation. As described in this case, a nasogastric tube should be placed prior to anesthesia to evacuate fluids and/or gas from the stomach as much as possible.

As stated earlier, the anesthetic protocol should have minimal negative effects on the cardiovascular and respiratory system (Taylor and Clarke, 2007; Boesch, 2013). However, despite their negative effects on the cardiovascular and gastro-intestinal system, α₂-agonists are needed to achieve adequate sedation in horses before induction of anesthesia. In colic horses, a short acting α₂-agonist such as xylazine is preferred (Taylor and Clarke, 2007), and was therefore chosen in this case. Opioids were not administered. The use of opioids in colic cases is still controversial due to their negative effect on the gastro-intestinal motility

(Boesch, 2013). Analgesia is however important in anesthesia and was accomplished by administration of the non-steroidal anti-inflammatory drug, flunixin. Flunixin meglumine provides effective (visceral) analgesia and reduces the signs of endotoxemia (Taylor and Clarke, 2007; Cook et al., 2009; Clarke et al., 2014). However, flunixin meglumine will inhibit the recovery of the ischemic injured mucosal barrier function (Cook et al., 2009; Marshall and Blikslager, 2011). Administration of polymyxin B (Taylor and Clarke, 2007) before induction could have been considered to reduce the risk of cardio-vascular collapse due to endotoxemia, which is common in colic cases (Doherty and Valverde, 2006; Rowland, 2013). General anesthesia was induced with ketamine and midazolam. The use of guaifenesin as muscle relaxant instead of benzodiazepines during induction has been described but may have a negative effect on the cardiovascular system (Boesch, 2013). Ketamine provides short-term anesthesia with stable cardiorespiratory function. In addition, ketamine provides analgesia and has potent anti-inflammatory effects (Muir, 2010).

During either the induction of anesthesia or when the horse is placed in dorsal recumbency, the general condition of a horse with a diaphragmatic hernia may quickly deteriorate and apnea may occur. Moreover, in this case, pronounced hypercapnia and hypoxemia were present (mean PaCO₂ of 61.5 mmHg, mean PaO₂ 30.6 mm Hg and mean SaO₂ 55.8 %). The potential consequences of hypercapnia are myocardial depression, sympathetic activation with catecholamine release (sinus tachycardia, cardiac arrhythmias), central nervous system depression, decreases in blood pH with an increase of serum potassium and impairment of cellular homeostasis and enzymatic function (Kerr and McDonnell, 2009). Hypoxemia appears because of the diminished residual volume, the changes in the ventilation/perfusion ratio and the increased intra-pulmonary shunt flow (Clutton et al., 1992; Robertson, 2002; Moens, 2013).

Mechanical ventilation with the use of oxygen is essential during anesthesia of horses and should be initiated as soon as possible after induction (Kelmer et al., 2008; Clarke et al., 2014; Melis et al., 2014). However, in patients with a diaphragmatic hernia, providing a good ventilation and oxygenation without causing further damage to the lungs is very challenging (Clutton et al., 1992; Kelmer et al., 2008). In this case, assisted-controlled intermittent positive pressure ventilation was applied immediately after induction of anesthesia, with a modest volume and pressure but a relatively high respiratory rate for adult horses. A low pressure with a maximum peak inspiratory pressure (PIP) of 20 cmH₂O and a low tidal volume of 9 to 11 mL kg⁻¹ are indeed advised in case of a diaphragmatic hernia to avoid overinflation of (parts of) the lungs, given the decreased available lung volume (Melis et al., 2014). Quick reinflation of atelectatic lungs may

cause iatrogenic damage to the alveoli, such as ventilation-induced barotrauma and lung edema. The aim of a conservative type of ventilation in these cases is not to reinflate the lungs but to maintain normocapnia and oxygenation (Melis et al., 2014; Ionnindis et al., 2015). The combination with a high respiratory rate, in this case 10-12 breaths per minute, is necessary to maintain an adequate minute volume ventilation and reach a sufficient oxygenation and normocapnia.

Ventilation induced lung trauma is one of the largest complications during mechanical ventilation and can be induced through three mechanisms: volutrauma (Carney et al., 2015; McMaster et al., 2014; Melis et al., 2014), atelectrauma (Carney et al., 2015; Melis et al., 2014; Güldner et al., 2016) or biotrauma (Carney et al., 2015; Melis et al., 2014; Güldner et al., 2016). With volutrauma, the alveoli are overfilled with a large volume, potentially in combination with a large pressure (Carney et al., 2005; Melis et al., 2014; Ionnindis et al., 2015; Güldner et al., 2016). Atelectrauma arises through stress in the alveolar membranes when the alveoli collapse and re-open during the breathing cycle (Carney et al., 2005; Melis et al., 2014; Güldner et al., 2016). Positive end expiratory pressure (PEEP) can be applied during ventilation to stabilize the lung and prevent the collapse and re-opening of the alveoli (Güldner et al., 2016). This mode was not used in the present case because of the unstable cardiovascular status of the horse. Indeed, PEEP is known to have a negative influence on the cardiovascular system, with a decrease of the cardiac output (Robertson, 2002; Rowland, 2013). Biotrauma is a more secondary, chemically induced damage to the lungs, caused by the release of inflammatory products such as cytokines, which are released in response to mechanically induced trauma through inappropriate ventilation (Melis et al., 2014; Ionnindis et al., 2015). Melis et al. (2014) described the use of dexamethasone 0.5 mg kg⁻¹ intravenously in a dog with diaphragmatic hernia in an attempt to stabilize the membranes to reduce the inflammatory response caused by hypoxemia and to prevent lung trauma due to re-expansion of the lungs. Chao et al. (2012) reported the positive effect of dexamethasone in the prevention of the development of the inflammatory response caused by alveolar hypoxemia. The administration of dexamethasone could have been considered in case the horse was not euthanized.

Because PEEP cannot be applied safely in the presence of pronounced cardiovascular depression, 100 µg ipratropiumbromide was administered at the Y-piece of the breathing circuit in an attempt to improve the oxygenation. Bronchodilators such as salbutamol (β₂ receptor agonists) (Robertson, 2002) and ipratropiumbromide (antimuscarinic) are used to improve oxygenation in horses with chronic obstructive pulmonary disease (COPD) by causing bronchodilation (Robinson et al., 2001; Bayly et al., 2002). The bronchial muscle tone is mainly regulated by the autonomic

nervous system and in addition by β -adrenoreceptors (Beeh et al., 2002). Ipratropium bromide causes dose-dependent bronchodilation in humans (Beeh et al., 2002). Reductions of the total pulmonary resistance and of the maximum change in intrapleural pressure, and an increase in the dynamic lung compliance, have been shown after the use of anticholinergics such as atropine, glycopyrrolate or ipratropium bromide (Bayly et al., 2002). To the authors' knowledge the effect of ipratropium bromide has not yet been investigated in anesthetized, hypoxemic horses. Salbutamol however has been investigated by Robertson et al. (2002). Eighty-one hypoxemic anesthetized horses were monitored to compare the arterial pH and blood gas values, heart rate and mean arterial pressure before and after treatment with salbutamol aerosol ($2 \mu\text{g kg}^{-1}$). In this study, an improvement of arterial oxygenation was noticed twenty minutes after treatment with salbutamol aerosol. Unfortunately, no control group was included in this study, making it difficult to draw clear conclusions. In the present case, a limited increase in arterial oxygenation was seen after the administration of ipratropium bromide.

However, as long as the abdominal organs remain in the thoracic cavity and the negative intrathoracic pressure is not restored, the inflation of the lungs is inadequate and hypoventilation will follow (Kelmer et al., 2008). In this case, the surgeons failed to reduce the abdominal organs out of the thorax. Some authors have described the use of a reverse Trendelenburg position (30° , hind quarter down) to improve visualization of the diaphragm and to facilitate reduction of the abdominal organs (Kelmer et al., 2008; McMaster 2014; Melis et al., 2014). It could additionally be hypothesized that this position would improve respiration by reducing the pressure of the intra-abdominal organs on the lungs. This position was not applied in this case, because the position may reduce venous return, affecting arterial blood pressure, which would not have been optimal in this patient. In horses undergoing elective surgery in a mild reverse Trendelenburg position, more dobutamine is indeed required to maintain arterial pressure than in horses on a horizontal table (Schauvliege et al., 2015). Besides a sufficient PaO_2 , a good blood pressure and cardiac output are fundamental for sufficient perfusion and oxygenation of the tissues (Robertson, 2002; Rowland, 2013).

The second major concern of the anesthesiologist is to maintain an adequate blood pressure during anesthesia. Decreased perfusion and oxygenation may contribute to damage to the liver and muscles, resulting in post-anesthetic myositis (Robertson, 2002; Rowland, 2013). Because of a compensatory increase of the cardiac output, the oxygen transport can usually be maintained under anesthesia despite reductions in the arterial oxygen content, but this becomes difficult in patients with a diaphragmatic hernia, which are already cardiovascularly compromised (Clutton et al., 1992; Hassel, 2007; Maish, 2010). Indeed, venous re-

turn is decreased in these horses due to compression of the different vessels by the displaced abdominal organs. In addition, during a normal inspiration, blood is drawn to the heart because of the negative intrathoracic pressure, but this mechanism is disturbed in case of a diaphragmatic hernia (Clutton et al., 1992).

In the present case, hypotension ($\text{MAP} < 60 \text{ mmHg}$) was indeed noted [$80/50$ (65) mmHg decreasing to $65/40$ (45) mmHg] (Figure 1). Hypotension can be counteracted using fluid therapy, positive inotropic agents and vasopressors as well as reducing the dose of the volatile anesthetic required. The latter can cause hypotension mainly through vasodilatation and a negative inotropic effect (Doherty and Valverde, 2006; Taylor and Clarke, 2007, Rowland, 2013). Fluid therapy can be performed with crystalloids or colloids, which were both used in this case to improve the blood pressure through two catheters in the jugular veins. Because crystalloids diffuse to the interstitial space, large quantities are needed to increase the plasma volume significantly (Doherty and Valverde, 2006). Colloids are more effective to correct hypotension due to hypovolemia. Because these fluids consist of large molecules, they are better retained in circulation and the intravascular volume rises more quickly. Small amounts of colloids are often already quite effective (5 mL kg^{-1}) (Doherty and Valverde, 2006; Rowland, 2013). In this case, 2.5 L of colloids I.V. were quickly administered. In case of severe hypovolemia, hypertonic fluids can be considered, as they quickly increase the intravascular volume (Rowland, 2013). This type of fluid therapy can be given quite quickly and small amounts are effective (Doherty and Valverde, 2006). This therapy draws fluid from the intracellular and interstitial space to the blood plasma, causing further dehydration and electrolyte disbalances. Hypertonic solutions are therefore best used in combination with crystalloids (Rowland, 2013). Nevertheless, administration of hypertonic saline during anesthesia is not advised, since it may result in bradycardia and hypotension (Rowland, 2013).

In addition to fluid therapy, dobutamine, an inotropic β_1 -agonist, was administered as a constant rate infusion. In case of severe hypovolemia, dobutamine may cause tachycardia (Boesch, 2013; Rowland, 2013), as in the present case. However, the persisted tachycardia could also partly have been the result of the light plane of anesthesia, pain, toxemia (Clarke et al., 2014) or hypoxemia (Boesch, 2013). Phenylephrine, a short-acting vasopressor α_1 -agonist, can be added as bolus or CRI to increase blood pressure by causing vasoconstriction without affecting cardiac output (Rowland, 2013; Southwood, 2013). In this case, a bolus of phenylephrine ($2 \mu\text{g kg}^{-1}$) was administered twice during anesthesia. After the second bolus, arterial blood pressure could be maintained above $65\text{-}70 \text{ mmHg}$ using continued crystalloid fluid therapy and administering of dobutamine by CRI until euthanasia was performed. The volatile anesthetic

isoflurane was used at the lowest possible end tidal concentration, because of its vasodilating and negative inotropic properties (Taylor and Clarke, 2007). To reduce the isoflurane requirements, a constant rate infusion of lidocaine was provided (Enderle et al., 2008). The patient nevertheless developed nystagmus twice, and an increase in heart rate was noted during anesthesia. Therefore, a bolus of ketamine was administered I.V. each time (150 mg and 100 mg, respectively). Alternatively, a constant rate infusion of ketamine could have been considered to further decrease the inhalation agents requirements (Matthews, 2004; Canola et al., 2015) and for its analgesic properties (Boesch, 2013), but it was not necessary in this case due to euthanasia of the patient (Matthews, 2004; Canola et al., 2015).

CONCLUSIONS

Optimizing the anesthesia of a patient with a diaphragmatic hernia is quite a challenge for the anesthesiologist. In these cases, the respiratory and cardiovascular systems are compromised and require extra attention during anesthesia. As long as the abdominal organs are not reduced and compress the lungs, adapted ventilation is necessary to maintain adequate ventilation and oxygenation without causing further damage to the lungs. A low volume positive pressure ventilation with a high respiratory rate is preferably used. Support of the cardiovascular system is also an important issue to focus on during anesthesia and can be performed in different ways.

REFERENCES

- Bayly W.M., Duvivier D.H., Votion D., Vandenput S., Art T., Lekeux P. (2002). Effects of inhaled ipratropium bromide on breathing mechanics and gas exchange in exercising horses with chronic obstructive pulmonary disease. *Equine Veterinary Journal*, 34(1), 36-43.
- Beeh K.M., Welte T., Buhl R. (2002). Anticholinergics in the treatment of chronic obstructive pulmonary disease. *Respiration* 69(4), 372-379.
- Boesch J.M. (2013). Anaesthesia for the horse with colic. *Veterinary Clinics of North America: Equine Practice* 29(1), 193-214.
- Canola P.A., Valadao C.A.A., Borges J.H.S., Canola J.C. (2015). Evaluation of cardiovascular function during S(+)-ketamine constant rate infusion in dorsally recumbent halothane-anesthetized horses, *Journal of Equine Veterinary Science*, 35, 41-48.
- Carney D., Dirocco J., Nieman G. (2005). Dynamic alveolar mechanics and ventilator-induced lung injury. *Critical Care Medicine* 33, 122-128.
- Chao J., Viets Z., Donham P. (2012). Dexamethasone blocks the systemic inflammation of alveolar hypoxia at several sites in the inflammatory cascade, *American Journal of Physiology: Heart and Circulatory Physiology* 303(2), 177-168.
- Clarke K.W., Trim C.M., Hall L.W. (2014). Anaesthesia of the horse. In: *Veterinary Anaesthesia*. Eleventh edition, Saunders Elsevier, London (UK), 245-311.
- Clarke K.W., Trim C.M., Hall L.W. (2014). Anaesthesia for intrathoracic procedures. In: *Veterinary Anaesthesia*. Eleventh edition, Saunders Elsevier, London (UK), 599-609.
- Clutton R.E., Boyd C., Richards D.L.S., Welker F.W., Moderansky P. (1992). Anesthetic problems caused by a diaphragmatic hernia in the horse: a review of four cases, *Equine Veterinary Journal* 24(11), 30-33.
- Clutton R.E. (2010) Opioid analgesia in horses. *Veterinary Clinics of North America: Equine Practice* 26(3), 493-514.
- Cook V.L., Meyer C.T., Campbell N.B., Bliklager A.T. (2009). Effect of firocoxib or flunixin meglumine on recovery of ischemic-injured equine jejunum, *American Journal of Veterinary Research* 70(8), 992-1000.
- Enderle A.K., Levionnois O.L., Kuhn M., Schatzmann U. (2008). Clinical evaluation of ketamine and lidocaine intravenous infusions to reduce isoflurane requirements in horses under general anesthesia. *Veterinary Anaesthesia and Analgesia*, 35(4), 297-305.
- Güldner A., Braune A., Ball L., Silva P.L., Samary C., In-sorsi A., Huhle R., Rentzsch I., Becker C., Oehme L., Andreeff M., Vidal Melo M.F., Winkler T., Pelosi P., Rocco P.R.M., Kotzerke J., Gama de Abreu M. (2016). Comparative effect of volutrauma and atelectrauma on lung inflammation in experimental acute respiratory distress syndrome *Critical Care Medicine* 44(9), 854-865.
- Hart S.K., Brown MS, Brown J. (2009). Diaphragmatic hernia in 44 horses (1989-2006), *Journal of Veterinary Emergency and Critical Care* 19(4), 357-362.
- Hassel D.M. (2007) Thoracic trauma in horses, *Veterinary Clinics: Equine Practice* 23 (1), 67-80.
- Ionnidis G., Lazardis G., Baka S., Mpoukovinas I., Karavasilis V., Lampaki S., Kioumis I., Pitsiou G., Papaiwannou A., Karavergou A., Katskigiannis N., Sarika E., Tsakitidis K., Korantzis I., Zarogoulidis K., Zarogoulidis P. (2015). Review article: Barotrauma and pneumothorax, *Journal of Thoracic Disease* 7, 38-43.
- Kelmer G., Kramer J., Wilson D.A. (2008). Diaphragmatic hernia: etiology, clinical presentation and diagnosis, *Compendium Equine*. January/February, 28-36.
- Kelmer G., Kramer J., Wilson D.A. (2008) Diaphragmatic hernia: treatment, complications and prognosis. *Compendium Equine*. January/February, 37-46.
- Kerr C.L., McDonnell W.N. (2009). Oxygen supplementation and ventilation support, In: Hubbell J.A.E., Muir W.W. (editors). *Equine Anaesthesia: Monitoring and Emergency Therapy*. Second edition, Elsevier Health Sciences, London (UK), 332-352.
- Kummer M.R. and Stick J.A. (2012). Abdominal hernia's, In: Auer J.A. and Stick J.A. (editors). *Auer Equine Surgery*. Fourth edition, Elsevier Saunders, Missouri (USA), 512-513.
- Maish M.S. (2010). The diaphragm. *Surgical Clinics of North America* 90(5), 955-968.
- Marshall J.F., Bliklager A.T. (2011). The effect of nonsteroidal anti-inflammatory drugs on the equine intestine, *Equine Veterinary Journal* 43(39), 140-144.
- Matthews N.S., Fielding C.L., Swinebroad E. (2004). How to use ketamine constant rate infusion in horses for analgesia, *IVIS. 50th Annual Convention of the American Association of Equine Practitioners*. Denver, Colorado.

- McMaster M., Spirito M., Munsterman A. (2014). Surgical repair of diaphragmatic tear in a thoroughbred broodmare. *Journal of Equine Veterinary Science* 34 (1-2), 1333-1337.
- Melis S.M., De Rooster H., Waelbers T., Polis I. (2014). Anaesthesia case of the month, *Journal of the American Veterinary Medical Association* 245, 1230-1234.
- Moens Y. (2013). Mechanical ventilation and respiratory mechanics during equine anaesthesia. *Veterinary Clinics of North America: Equine Practice*, 29(1), 51-67.
- Muir W.W. (2010). NMDA receptor antagonists and pain: ketamine. *Veterinary Clinics of North America: Equine Practice* 26(3), 565-578.
- Robertson S.A., Bailey J.E. (2002) Aerosolized salbutamol (albuterol) improves PaO₂ in hypoxaemic anaesthetized horses – a prospective clinical trial in 81 horses. *Veterinary Anaesthesia and Analgesia* 29, 212-218.
- Robinson N.E., Derksen F.J., Jackson C.A., Peroni D., Gerber V. (2001). Satellite Article: Management of heaves, *Equine Veterinary Education* 13(5), 247-259.
- Röcken M., Mosel G., Barske K., Witte T.S. (2013). Thoracoscopic diaphragmatic hernia repair in a warmblood mare. *Veterinary Surgery* 42, 591-594.
- Rowland S. (2013). Blood pressure management in equine anaesthesia, *Veterinary technician*, E1-E6.
- Santschi E.M., Juzwiak J.S., Moll H.D., Slone D.E. (1997). Diaphragmatic hernia repair in three young horses. *Veterinary Surgery* 26, 242-245.
- Schauvliege S., Van Dyck J.J., Duchateau L., Rodrigo-Mocholi D., Cerasoli I., Binetti A., Van Hende D., Neumeyer A., Gasthuys F. (2015). Cardiorespiratory effects of mild reverse Trendelenburg position in horses. In: *Proceedings World Congress of Veterinary Anaesthesia 2015*. Kyoto, Japan.
- Sprayberry K.A. and Barret E.J. (2015) Thoracic trauma in horses, *Veterinary Clinics of North America: Equine practice*, 31(1), 199-219.
- Taylor P.M. (1998). Effects of hypoxaemia on endocrine and metabolic responses to anaesthesia in ponies. *Research in Veterinary Science*, 66, 39-44.
- Taylor P.M., Clarke K.W. (2007). Inhalation anaesthesia, In: Taylor P.M., Clarke K.W. (editors). *Handbook of Equine Anaesthesia*. Second edition, Saunders Elsevier, Philadelphia (USA), 54-84.
- Taylor P.M., Clarke K.W. (2007). Anesthetic problems, In: Taylor P.M., Clarke K.W. (editors). *Handbook of Equine Anaesthesia*. Second edition, Saunders Elsevier, Philadelphia (USA), 123-175.
- Taylor P.M., Clarke K.W. (2007). Anaesthesia in special situations, In: Taylor P.M., Clarke K.W. (editors). *Handbook of Equine Anaesthesia*. second edition, Saunders Elsevier, Philadelphia (USA), 176-207.
- Valverde A. (2010). Alpha-2 agonists as pain therapy in horses, *Veterinary Clinics of North America: Equine Practice* 26(3), 515-532.

Uit het verleden

VLOEKEN BIJ HET VEE DRIJVEN ALS OORZAAK VAN DE ‘PLAGEN DER BEESTEN’

‘De plagen der beesten zijn dikwijls de gevolgen van de godslasteringen, vloeken en verwenschingen der meesters en dienstboden. Wij moeten dit doen, zeggen goddeloze landlieden, om de beesten voort te stuwen! Wat een ijdele verschooning. Het zijn de godslasteringen, de vloeken, de verwenschingen niet die deze beesten zullen doen voortgaan. Andere woorden zullen hetzelfde uitwerksel hebben. Waarom dan goddeloze en heiligschennende woorden gebruikt, die den Heiligen Naam des Heeren lasteren, de plagen over uwe huizen trekken en u plichtig maken aan het helsche vuur, waar gij voor eeuwig met den duivel wiens taal gij spreekt, zult liggen te branden. Neemt dus nooit die gruwelijke gewoonte aan.’

Uit: Caytan, L.A.. *De Godvruchtige Landman*. Eerste druk 1786. Het fragment werd overgenomen uit de ‘Nieuwe uitgaaf, verbeterd volgens de noodwendigheden van onzen tijd’, uitgegeven door Beyaert, Brugge, 1895, p. 247.