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Use of continuous positive airway pressure (CPAP) in a horse with diaphragmatic hernia

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Summary

Continuous positive airway pressure (CPAP) is a ventilation mode where airway pressure is kept above ambient during the entire breathing cycle. We describe a case of a diaphragmatic hernia in a horse where CPAP was applied during anaesthesia to treat hypoxaemia. A 15 year-old mare weighing 556 kg was anaesthetised for colic surgery. After induction, hypoxaemia (PaO_2: 8.8 kPa = 66 mmHg) and severe hypotension (mean arterial pressure: 24 mm Hg) were noticed. CPAP at a level of 20 cm H_2O was applied to the airways. This resulted in a marked increase of PaO_2 to 53.2 kPa (400 mmHg) within 90 minutes after induction of anaesthesia. Treatment of hypotension included dobutamine, ephedrine, phenylephrine and hypertonic saline. Total anaesthesia time was 120 minutes. Surgical examination revealed an impaction of the large colon with no other gastrointestinal abnormality. After disconnecting the horse from the circle system and transferring it with the overhead hoist into the recovery box, it suffered from cardio-respiratory arrest and died. Pathology revealed a diaphragmatic hernia with 25 L of blood and the whole stomach in the thoracic cavity. In this case CPAP appeared to be a reliable technique to treat hypoxaemia during anaesthesia in a horse with a pre-existing pulmonary abnormality. However, its effects on the cardio-vascular system must be closely monitored and adequate support provided.

Keywords: Anaesthesia, hypoxaemia, ventilation mode, horse, continuous positive airway pressure, CPAP

Verwendung eines neuen Ventilationsmodus genannt "Kontinuierlicher positiver Atemwegsdruck" bei einem Pferd mit Zwerchfellruptur

Der Ventilationsmodus "Kontinuierlicher positiver Atemwegsdruck" oder continuous positive airway pressure (CPAP) wird in der Humanmedizin routinemässig eingesetzt um kollabierte kleine Atemwege (Bronchiloes) zu öffnen oder offen zu halten. Dies wird erreicht durch einen kontinuierlich positiven Atemwegsdruck während des gesamten Atemzyklus. Der Patient atmet dabei spontan. Bis vor kurzem war es nicht möglich CPAP beim Pferd anzuwenden, da uns kein adequater Ventilator zur Verfügung stand. Seit kurzer Zeit ist ein Kolben-getriebener Ventilator erhältlich, der durch die Messung des Atemwegdruckes im Bereich des Y-Stückes und durch computergesteuerte Anpassung der Kolbenstellung den Atemwegsdruck trotz der hohen Flussraten während des Expiriums und des Inspiriums beim Pferd konstant halten kann. Dieser Fallbericht beschreibt die Anästhesie einer Stute (556 kg) während einer Kolikoperation. Nach der Einleitung wurde eine Hypoxämie (PaO_2: 8.8 kPa = 66 mmHg) und eine hochgradige Hypotension (mittlerer arterieller Blutdruck: 24 mmHg) diagnostiziert. Der Atemwegsdruck wurde auf 20 cmH_2O gesetzt (CPAP = 20 cmH_2O). Das bewirkte einen starken Anstieg des PaO_2 auf 53.2 kPa (400 mmHg) innerhalb von 90 Minuten nach Anästhesieeinleitung. Die Hypotension wurde mit Dobutamin, Ephedrin, Phenylephrin und hypertoner Kochsalzlösung behandelt. Die gesamte Anästhesiezeit betrug 120 Minuten. Während der chrirugischen Exploration wurde eine Obstipation des Kolons diagnostiziert. Es wurden keine weiteren Abnormalitäten des Gastrointestinaltraktes bemerkt. Nachdem das Pferd vom Ventilator diskonnektiert und mittels Kran in die Aufwachbox transportiert wurde, erlitt es einen Herz-Atemstillstand und starb. In der Pathologie wurde eine Zwerchfellruptur mit 25 Liter Blut und dem gesamten Magen im Thorax diagnostiziert. In diesem Fall war der CPAP-Ventilationsmodus eine erfolgreiche Technik um die Hypoxämie in einem Pferd mit hochgradiger Lungenpathologie während der Anästhesie zu therapieren. Allerdings muss das kardiovaskuläre System genau überwacht und notwendige begleitende Therapien durchgeführt werden.

Schlüsselwörter: Anästhesie, Hypoxämie, Ventilationsmodus, Pferd, kontinuierlicher positiver Atemwegsdruck, CPAP

Introduction

Diaphragmatic hernia is a rare condition in horses that has been associated with a high mortality (*Branson* and *Kramer* 2000). During anaesthesia the compression of the lungs by the herniated organs plus the absence of subatmospheric intrapleural pressure results in a massive alveolar collapse and decrease in functional residual capacity (FRC). This on top of the rapid occurrence of atelectasis after induction of anaesthesia and resulting high intrapulmonary shunting reported in healthy horses (*Nyman* et al. 1990) can lead to severe hypoxaemia, impaired ventilation and high intraoperative mortality in these patients (*Clutton* et al. 1992, *Pearson* et al. 1977).

Continuous positive airway pressure (CPAP) is a ventilation mode used to treat hypoxaemia in man (*Lindner* et al. 1987). CPAP is the application of a continuous pressure above ambient in the airway in spontaneously breathing patients. A large animal ventilator (Tafonius, Vetronic Services, Abbotskerswell, Devon, UK), able to provide CPAP in horses, has only become available recently; therefore no scientific data are available for the use of CPAP in horses.

This case report describes the use of CPAP in a horse with diaphragmatic hernia presented for exploratory laparotomy. Hypoxaemia during anaesthesia was successfully treated by the use of CPAP, but its use was also associated with severe cardiovascular depression.

Case report

A 15 year-old thoroughbred mare weighting 556 kg was admitted with acute signs of colic. The horse was agitated and uncomfortable; rectal examination revealed impactions of the large colon. It was decided to perform an exploratory laparatomy.

Preoperative resuscitation consisted of pentastarch 1L (HAES-steril, Fresenius Kabi Ltd, Warrington, UK) and Hartmann's solution 5L (Hartmann's solution; Vetivex 11, Dechra ltd.Shrewsbury UK) intravenously (IV) and two boli of xylazine 0.3 mg kg^{-1} IV (Virbaxyl 10%; Virbac Ltd, Bury St. Edmunds, Suffolk, UK). Flunixin (Flunixin; Norbrook Laboratories, Carlisle, Cumbria, UK) 1.1 mg kg^{-1} IV, gentamycin 2.2 mg kg^{-1} IV (Genta-100; cp-pharma, Burgdorf, Germany) and procaine penicillin 20 000 iu kg^{-1} (Norocillin; Norbrook Laboratories Ltd) were administered intramuscularly (IM) before premedication.

The mare was pre-medicated with xylazine 0.6 mg kg^{-1} and morphine 0.1 mg kg^{-1} IV (Morphine Sulphate Injection BP, CB Pharma Ltd., Slough, Berkshire UK). Anaesthesia was induced with diazepam (Diazepam Injection, Hameln Pharmaceuticals Ltd., Gloucester, UK) 0.1 mg kg^{-1} followed by ketamine 2 mg kg^{-1} IV. The trachea was intubated and the mare transferred to the surgery table. The tube was connected to a circle system (Tafonius, Vetronic Services, Abbotskerswell, Devon, UK).

Anaesthesia was maintained with sevoflurane (SevoFlo, Abbott Laboratories Ltd, Queensborough, UK) in oxygen 100% supplemented with a lidocaine infusion (Lidocaine Hydrochloride Intravenous infusion 0.2%, Fresenius Kabi Ltd., Runcorn, UK) (1.7 mg kg^{-1} h^{-1}) and methadone boli (Physeptone Injection, Martindale Pharmaceuticals Ltd. Romford, Essex UK) (0.1 mg kg^{-1} every hour). Hartmann`s solution was infused at a rate of 10 mL kg^{-1} h^{-1} in combination with pentastarch 3 L during the first hour. The horse was allowed to breath spontaneously after induction. Heart rate (HR), systolic, diastolic and mean invasive arterial pressure (SAP, DAP and MAP), pulse oximetry (SpO_2) end-tidal sevoflurane concentration ($P_{ET}SEVO$), end-tidal CO_2 ($P_{ET}CO_2$), respiratory rate (f_R), tidal volume (TV) and minimal and maximal airway pressures (PI_{min} and PI_{max}), were continuously monitored using multiparameter monitors (Tafonius and Datex-Ohmeda S5, Datex-Ohmeda Finland OY, Helsinki, Finland) and recorded every five minutes.

Dobutamine (Dobutamine, Hameln pharmaceuticals ltd. Gloucester, UK) was started immediately after induction (1 μg kg^{-1} min^{-1}) and infusion rate was increased over the next 10 minutes to 4 μg kg^{-1} min^{-1} due to severe hypotension (MAP of 24 mm Hg and weak pulse). A blood gas analysis 15 minutes after induction of anaesthesia (ABL5 machine; Radiometer Medical A/S, Copenhagen, Denmark) revealed hypoxaemia (PaO_2: 8.8 kPa [66 mmHg]) and hypercapnia ($PaCO_2$: 8.3 kPa [62 mmHg]). Alveolar arterial oxygen difference (D(A-a)O2) was calculated retrospectively and was 77 kPa (579 mmHg). Changes in the partial pressures of $PaCO_2$ and PaO_2 over time are summarised in table 1.

Intermittent positive pressure ventilation was initiated (f_R 4 bpm, TV 8 L, PI_{max} 30 cmH_2O) to treat the respiratory component of the moderate blood acidosis (pH: 7.19; base excess: -4.3 mmol L^{-1}). Despite adequate depth of anaesthesia the horse did exert respiratory efforts against the ventilator up to a point where it was decided to let it breath spontaneously while applying a CPAP of 20 cmH_2O. The horse was breathing 4 to 6 times a minute with a tidal volume of 10 to 12 litres with only minor changes throughout anaesthesia. A blood gas analysis performed 45 minutes after induction revealed a PaO_2 of 28.7 kPa (216 mm Hg) with a raise in $PaCO_2$ to 10.8 kPa (81 mmHg). $D(A\text{-}a)O_2$ decreased to 54,3 kPa (408 mmHg).

While oxygenation of the horse was improving over the first 45 minutes mean arterial pressure remained below 50 mm Hg despite dobutamine infusion and aggressive fluid therapy. Heart rate increased up to 120 bpm. In addition to dobutamine, two boli of ephedrine 0.1 mg kg^{-1} were administered. Arterial pressure reached a nadir of 60 mm Hg and HR remained at 120 bpm. An infusion of hypertonic saline 7.5%

Table 1 Changes in arterial oxygen tension (PaO_2), carbon dioxide arterial tension ($PaCO_2$), alveolar arterial difference ($D(A\text{-}a)O_2$), heart rate (HR), mean arterial pressure (MAP) and respiratory rate (RR) during anaesthesia. Spont = spontaneous ventilation; FiO_2 = fraction of inspired oxygen

Veränderung von arterieller Sauerstoffspannung (PaO_2), arterieller Kohlendioxidspannung ($PaCO_2$), alveolär-arterieller Differenz ($D(A\text{-}a)O_2$), Herzschlag (HR), mittlerem arteriellem Druck (MAP) und Atemfrequenz (RR) während der Narkose. Spont = spontane Atmung; FiO_2 = Fraktion des eingeamtmeten Sauerstoffs

Minutes after induction	Ventilation mode	FiO_2	PaO_2 kPa (mmHg)	$PaCO_2$ kPa (mmHg)	$D(A\text{-}a)O_2$ kPa (mmHg)	HR bpm	MAP mmHg	RR bpm
15	Spont	1	8.8 (66)	8.3 (62)	77 (579)	65	40	4
45	CPAP	1	28.7 (216)	10.8 (81)	54.3 (408)	105	70	5
90	CPAP	1	53.2 (400)	7.9 (59)	33 (248)	90	75	6

(4 mL kg^{-1} over 15 minutes) and phenylephrine 60 μg kg^{-1} h^{-1} were started 45 minutes after induction of anaesthesia. This resulted in a marked improvement in arterial pressure (MAP of 70 mm Hg) and a HR of 90 bpm; mucous membranes appeared pink with CRT of 2 seconds and a good pulse. Phenylephrine infusion was stopped and dobutamine was decreased to 2 μg kg^{-1} min^{-1} for the remaining anaesthesia time. Ninety minutes after induction of anaesthesia PaO_2 was 53.2 kPa (400 mmHg), $PaCO_2$ 7.9 kPa (59 mmHg), $D(A\text{-}a)O_2$ 33 kPa (248 mmHg) and a mild acidosis (pH: 7.24, BE: -2.5 mmol L^{-1}) was present. PCV and TP were 26% and 50 g L^{-1}, respectively.

During surgery, a large colon impaction was found with no other gastrointestinal abnormalities. Stomach and liver could be grossly felt.

At the end of anaesthesia (120 minutes after induction) the horse was transferred to the recovery box with an overhead hoist. When reaching the recovery box, the horse stopped breathing with dilated pupils, cyanotic mucous membranes and reflux from the nose. No pulse was palpable. Immediately initiated cardiopulmonary resuscitation manoeuvres were ineffective.

Necropsic examination revealed a diaphragmatic hernia, with approximately 25 L of blood (mainly clotted) and the entire stomach in the thoracic cavity.

Discussion

This case describes the use of CPAP to treat hypoxaemia in a horse with diaphragmatic hernia. Partial pressure of oxygen increased markedly within 75 minutes during anaesthesia despite the engagement of parts of the stomach and blood in the thoracic cavity.

Diaphragmatic hernia is a condition associated with a high anaesthetic mortality rate related to impaired ventilation and hypoxaemia (*Clutton* et al. 1992). Hypoxaemia was also noted in the case described here 15 minutes after induction. Several mechanisms may have contributed to the marked hypoxaemia. Intrapulmonary shunting and decrease of ventilation perfusion ratio due to atelectasis are undoubtedly involved as shown by the increase in alveolar arterial oxygen difference. Anaesthetised healthy horses have values for $D(A\text{-}a)O_2$ of approximately 26 kPa when placed in dorsal recumbency (*Nyman* et al. 1990, *Wilson* and *Soma* 1990). The massive lung collapse in this case described here was likely caused by decreased functional residual capacity due to the herniated viscera and presence of blood in the thorax, and the absence of transpulmonary pressure to oppose the elastic recoil force of the lung (*Clutton* et al. 1992). Hypoventilation and possible diffusion impairment caused by lung tissue inflammation due to the underlying disease might have enhanced hypoxaemia (*Brigham* 1982).

The use of IPPV in horses suffering from diaphragmatic hernia has been associated with better outcome compared to spontaneous ventilation (*Clutton* et al. 1992). The "panting respiration" and "fighting the ventilator" seen in our horse has been described by *Clutton* et al. (1992). The reason for this phenomenon is unclear. In babies an expiratory reflex has been described causing life-threatening pneumothorax during IPPV (*Greenough* et al. 1986). This reflex is comparable with the enormous respiratory drive seen in these horses. The minute volume of this horse was around 60 litres per minute. This is higher than the normally expected 10 ml/kg/min, which would have been 55 litres for this horse.

We chose CPAP in an attempt to treat hypoxaemia after failure of IPPV. To provide CPAP, the ventilator must deliver a high gas flow to keep the pressure positive despite the high respiratory flow generated during inspiration and expiration (4.5 L s during inspiration, 4 L s^{-1} during expiration) (*McMurphy* et al. 2002). A new large animal ventilator (Tafonius, Vetronic & Halowell corporations) driven by a servo-controlled piston can keep the airway pressure positive throughout the breathing cycle. The piston is controlled by a software which also integrates airway pressure readings taken at the Y-piece of the circle system. Using the pressure information the piston moves at different speeds to meet the corresponding flow rates.

Continuous positive airway pressure was remarkably efficacious to improve PaO_2 in our horse. It has been used in man in a wide range of lung pathologies especially as part of the treatment of acute respiratory distress syndrome. By maintaining a positive airway pressure, CPAP decreases alveolar collapse and improves FRC. In our case CPAP recruited collapsed alveoli as shown by a decrease in arterial to alveolar oxygen gradient (from 77 to 33 mm kPa). Although this phenomenon has already been described in man (*Pilbeam* 1998) and noticed in healthy horses (unpublished data), this is still remarkable as in this case blood and viscera additionally compressed the lung.

The high level of 20 cmH_2O of CPAP chosen in our case was related to the high inspiratory effort of the horse resulting in a high inspiratory flow. Therefore, a high level of basic airway pressure had to be applied to ensure a permanent pressure over ambient. Values of CPAP usually range between 5 to 15 cmH_2O in man (*Pilbeam* 1998) and a value of 10 cmH_2O has been used successfully in healthy horses (unpublished data).

A major concern of applying a high level of CPAP was the potential risk of cardio-vascular depression related with decreased venous return and cardiac output. A decrease in cardiac output using IPPV and/or positive end-expiratory pressure has been reported in healthy horses and horses suffering from colic (*Nyman* et al. 1988, *Wilson* and *McFeely* 1991, *Wilson* and *Soma* 1990). On the other hand *Wettstein* et al. (2006) have shown that peak inspiratory pressures of up to 55 cmH_2O in combination with positive-end expiratory pressures of 20 cmH_2O increase oxygenation with limited cardiovascular compromise in healthy ponies. No statement on the real impact of CPAP on the cardiovascular system in our horse can be given as too many other factors like hypovolaemia, probably compression of thoracic vessels and the absence of the pumping effect due to the huge defect in the diaphragm were contributing to the poor performance of the cardiovascular system during anaesthesia. In the case described here the clinically measurable parameters of the cardiovascular system like heart rate and blood pressure were sta-

ble at the end of anaesthesia after aggressive fluid therapy with minor sympathomimetic support using the high CPAP level. However, the heart rate was still abnormally high. It also remains questionable why the $PaCO_2$ remained high while PaO_2 was increasing steadily. Beside hypoventilation, which was not present in our horse proven by the high minute volume, one explanation would be the stabilisation of the peripheral vascular system and wash out of "trapped" metabolites as CO_2 from the periphery into the central compartment. Obviously the application of CPAP was necessary to keep the balance between respiratory and cardiovascular system as the horse died within several minutes after CPAP was stopped.

In our case, the surgeon palpated the stomach during laparotomy but pathology reported the entire stomach in the thoracic cavity. These discordances might be attributed to the use of CPAP. The continuous positive pressure applied to the airways may have prevented viscera from entering the thorax. After disconnection from CPAP and the transfer of the animal with the overhead hoist into the recovery box the stomach was probably pushed through the diaphragm. This would explain the rapid development of cardio-pulmonary arrest.

Conclusion

In conclusion, CPAP was a valuable option to treat hypoxaemia in this horse. It is the first description of a decrease in $D(A\text{-}a)O_2$ in a horse anaesthetised for diaphragmatic hernia. The cardio-vascular performance was only adequate after aggressive cardio-vascular support.

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