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Technical Note

Oxygen supplementation before induction of general anaesthesia in horses

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Summary

Reasons for performing study: Hypoventilation or apnoea, caused by the induction of general anaesthesia, may cause hypoxaemia. Preoxygenation may lengthen the period before this happens. No scientific studies are published on preoxygenation in equine anaesthesia.
Objectives: To determine whether supplementation of oxygen at a flow rate of 15 l/min for 3 min via a nasal cannula before induction of general anaesthesia is effective in elevating the arterial partial pressure of oxygen (PaO_2) directly after induction.
Study design: Randomised, prospective clinical trial.
Methods: A total of 18 American Society of Anesthesiologists physical status 1 or 2 adult horses undergoing elective anaesthesia were randomly allocated to one of 2 groups. The first group (control) received no oxygen supplementation before induction of general anaesthesia, whereas the second group (oxygen) did. All horses were anaesthetised with intravenous detomidine, butorphanol, ketamine, midazolam and isoflurane. Directly after induction (T = 0) and 30 min later (T = 30) an arterial blood sample was taken for blood gas analysis. At T = 30 an estimate of intrapulmonary shunt fraction (Qs/Qt) was calculated.
Results: At T = 0 arterial partial pressure of oxygen (PaO_2) was significantly higher in the oxygen group compared with the control group (11.0 ± 2.6 kPa vs. 7.4 ± 1.6 kPa; mean ± s.d., P = 0.005) and at T = 30 differences were not statistically significant. Partial pressure of carbon dioxide ($PaCO_2$) and Qs/Qt did not differ between groups.
Conclusions: Supplementing oxygen by a nasal cannula before induction of general anaesthesia in horses is feasible and does effectively elevate the PaO_2 immediately after induction. Future research is needed to determine whether supplementation of oxygen before induction of general anaesthesia in horses will affect outcomes.

Keywords: horse; preoxygenation; general anaesthesia; hypoxaemia; F shunt

Introduction

Preoxygenation is a common practice in anaesthesia in small animals and man to lengthen the period before development of hypoxaemia, caused by hypoventilation or apnoea resulting from induction of general anaesthesia. There are no scientific reports on techniques, strategies, benefits and potential adverse effects of preoxygenating horses before induction of general anaesthesia.

Although a previous study has shown that supplementing oxygen through a nasal cannula in standing awake horses is effective in increasing the arterial partial pressure of oxygen (PaO_2) [1], it can be questioned whether supplementing oxygen to horses before induction of general anaesthesia is feasible and effective in preventing potential hypoxaemia.

The aim of the current study was to determine whether supplementing oxygen via a nasal cannula before induction of general anaesthesia in horses is effective in increasing the PaO_2 immediately after induction of general anaesthesia, whether it leads to disturbance of the induction process and whether it affects venous admixture (Qs/Qt) by increasing resorption atelectasis.

Materials and methods

Animals

A total of 18 adult horses, American Society of Anesthesiologists physical status 1 or 2, undergoing elective anaesthesia were enrolled. An *a priori* sample size calculation, using the expected PaO_2 values of [1], showed that one animal per group was needed for a power of 0.8 and alpha of 0.05. However, more animals were included in the study to allow for statistical testing and subsequent generalisation of the results.

The animals were randomly allocated to either the control or oxygen group concealed by drawing prewritten lots from an opaque envelope. Supplementary Item 1 outlines characteristics of horses recruited for the study.

Procedures

All horses were premedicated with butorphanol (Dolorex)[a] 20.0 μg/kg bwt i.v. and detomidine (Domosedan)[b] 10.0 μg/kg bwt i.v. After adequate sedation, the animals were placed behind a swing door for induction of general anaesthesia. A nasal cannula (silicon tube with an external diameter of 10 mm and 50 cm in length) was held over the face with the tip at the medial canthus of the left eye and a mark placed on the tube at the level of the left nostril. After marking, the cannula was advanced into the left ventral nasal meatus until the mark on the tube was at the level of the nostril. Acceptance of the cannula was subjectively scored on a 3 point scale (good, moderate, poor) by the same observer. When in place, the oxygen flow was turned on and immediately increased to 15 l/min in the oxygen group. In the control group the oxygen flow was not turned on. The cannula was left in place for 3 min before the induction agents were given.

In all horses, anaesthesia was induced with ketamine (Narketan)[c] 2.2 mg/kg bwt i.v. and midazolam (Midazolam Actavis)[d] 0.05 mg/kg bwt i.v. Directly after induction (T = 0, all horses in left lateral recumbency), an arterial blood sample was drawn anaerobically from the right facial artery, collected in a balanced heparinised syringe[e] (Rapidlyte) and directly analysed on a blood gas analyser[e] (Rapidlab 1200 series). All samples were obtained within 1 min after the facial artery could be safely accessed. After orotracheal intubation the endotracheal tube was attached to a circle breathing system and a fresh gas mixture of isoflurane in oxygen and air given to both groups at a total flow rate of 4–6 l/min with an inspiratory oxygen fraction (FiO_2) of 50%. End-tidal isoflurane concentration (ET-Iso) was targeted at 1.3%. A second arterial blood sample was taken 30 min after

TABLE 1: Arterial artial pressures of oxygen (PaO_2) and carbon dioxide ($PaCO_2$) and shunt fraction (Qs/Qt) in horses with and without oxygen supplementation before induction of general anaesthesia

	Control group	Oxygen group
PaO_2 (kPa) (mean ± s.d.)		
T = 0 [a]	7.4 ± 1.6 (range: 5.0–10.9)	11.0 ± 2.6 (range: 7.0–15.7)
T = 30	15.9 ± 6.9 (range: 7.9–27.2)	14.2 ± 5.7 (range: 8.21–24.8)
$PaCO_2$ (kPa) (mean ± s.d.)		
T = 0	6.8 ± 0.6 (range: 5.5–7.6)	7.5 ± 1.3 (range: 6.5–9.4)
T = 30	7.8 ± 1.4 (range: 5.3–9.7)	8.3 ± 1.2 (range: 6.1–10.3)
Qs/Qt (%) (mean ± s.d.)	20 ± 11 (range: 6–41)	24 ± 9 (range: 12–40)

[a]Significant difference between groups P = 0.005.

taking the first arterial blood sample (T = 30) and directly analysed for blood gasses and haemoglobin content. The latter was analysed on a haematology analyser (Medonic CA 530 Vet analyzer)[f]. At this point the horses were either in lateral or dorsal recumbency (7 lateral, 2 dorsal in each group). The actual FiO_2, measured by the anaesthesia monitor (Datex S/5)[g] and atmospheric pressure measured by the blood gas analyser[e], were noted. Based on these parameters, an estimate of shunt fraction (Qs/Qt) was calculated (Supplementary Item 2) [2–4]. During the 30 min period in which blood samples were taken, the animals were breathing spontaneously. After this period, the anaesthetist involved changed ventilation, fresh gas flow and ET-Iso as needed for appropriate maintenance of anaesthesia. All procedures in the first 30 min period were carried out by one researcher (H.v.O.).

Data analysis

Data were analysed using Microsoft Excel 2010[h] and SPSS 20[i]. Values for age, weight, PaO_2, $PaCO_2$ and Qs/Qt were tested using an independent samples *t* test (2-sided), as assumptions on normality and homogeneity of variance were met. Bonferroni correction was used to correct for multiple testing (P value × 2). Group distribution regarding gender, ASA physical status and position during the maintenance phase of anaesthesia was tested using a Fisher's exact test. Differences were considered statistically significant when $P \leq 0.05$.

Results

No statistically significant differences in data on group composition were found (S1). In 2 horses (one in each group), acceptance to the silicon tube in the nose was scored as 'moderate'; the observed reactions were sneezing, head shaking and head lift. In all other animals it was scored as 'good'.

At T = 0, the PaO_2 of the oxygen group was significantly higher compared with the control group at T = 0 (P = 0.005) but not T = 30 (Table 1). At T = 0, in all but one control animal, PaO_2 values were below the cut-off point at which intervention to treat the hypoxaemia is deemed necessary, i.e. <8–9.3 kPa [5]. In contrast, in the oxygen group only one animal had a PaO_2 value <8–9.3 kPa. There were no significant differences in $PaCO_2$ and Qs/Qt between groups.

Discussion

Horses breathing room air before induction of general anaesthesia are likely to develop hypoxaemia in the early phase after induction and supplementing oxygen and using the technique described in this study may prevent this.

There is limited scientific literature on the effect of hypoxaemia on short- and long-term outcome in anaesthetised horses. Although this lack of evidence does not mean that hypoxaemia does not affect outcome in horses, we know that horses possess a large cardiopulmonary reserve capacity which may allow them to be less affected by hypoxaemia than other species. This large reserve results from a superior O_2 delivery system consisting of a large heart with high stroke volumes, a high maximum heart rate, a highly contractive spleen that can increase haematocrit levels up to 60–70% and a slightly left shifted O_2 dissociation curve (P50 of ~3.3 kPa) [6]. However, stroke volume, myocardial inotropy, venous return, heart frequency, sympathetic tone, splenic contraction and local tissue perfusion pressure are negatively affected by anaesthetic agents and recumbency. As a consequence, general anaesthesia can decrease the cardiopulmonary reserve of both healthy and sick horses impairing the capacity to cope with hypoxaemia. A recent study shows that hypoxaemia (next to duration of anaesthesia and type of suture material) is a risk factor for developing wound infection in horses undergoing emergency surgery for colic [7]. This supports the view that it is relevant to prevent hypoxaemia during general anaesthesia in horses, as it may affect outcome.

It could be argued that, for better comparison between the groups, instead of no flow, air (FiO_2 0.21) at a flow rate of 15 l/min should have been given to the control group. In the oxygen group, the flow itself might have stimulated the animals, leading to increased ventilation and improved oxygenation. However, we assume that this effect was minimal as in both groups only one horse was disturbed by the placement of the nasal cannula and all animals were adequately sedated in preparation for induction of general anaesthesia. In addition, no differences in $PaCO_2$ were found between groups, suggesting that the oxygen group did not have an increased alveolar minute volume compared with the control group.

In theory, oxygen flow rates of higher than 15 l/min could have been given to further increase the PaO_2 at T = 0. Our oxygen flow meter did not allow for accurate measurement of higher oxygen flow rates but it is possible to deliver the oxygen bilaterally in the nose [1].

Preoxygenation could potentially increase intrapulmonary shunt by increasing resorption atelectasis [8]. Our results show no significant difference in intrapulmonary shunt fraction (Qs/Qt) between the oxygen and control group at T = 30, making it less likely that the oxygen supplementation before induction of general anaesthesia as described in this study increases intrapulmonary shunt by promoting resorption atelectasis. In this study Qs/Qt was estimated by using a previously reported equation [2–4]. Calculating the true Qs/Qt requires mixed venous blood samples obtained via a pulmonary artery catheter. Placing a pulmonary artery catheter is not without risk and was therefore considered nonfeasible in studies using client-owned horses. The equation used here assumes a fixed oxygen content difference between arterial and mixed venous blood of 3.5 ml/dl. This assumption is based on previous work in man [2] and removes the need for mixed venous blood samples. Although this equation for calculating Qs/Qt has been published in equine studies [3,4], the formula has not yet been validated in this species. Nevertheless, at present it seems the best surrogate to noninvasively calculate Qs/Qt in client-owned horses.

Supplementing oxygen to horses before induction of general anaesthesia could potentially lead to hypercapnia and respiratory acidosis in the early maintenance phase after induction, due to absence of a hypoxic respiratory drive. This phenomenon was observed previously in anaesthetised elk [9]. However, the PaO_2 of the elk that demonstrated hypoxaemic respiratory drive (~4 kPa) was lower than the mean PaO_2 in our control group (7.4 kPa). Also, in awake horses, hypoxaemic respiratory drive is reported to occur at PaO_2 of 5 kPa [10], below the PaO_2 in our control group. As our results show that the arterial carbon dioxide concentration does not differ between groups, we conclude that supplementing oxygen to horses before induction of anaesthesia does not lead to hypercapnia and the hypercapnia found in both groups in this study is most likely due to the respiratory depressant effect of general anaesthesia induced with ketamine and midazolam and maintained by an inhalant [11].

We conclude that supplementing oxygen to horses before induction of general anaesthesia is effective in increasing the PaO_2 during the early anaesthesia maintenance phase, does not disturb the induction process and does not increase Qs/Qt. However, future research is needed to determine whether this technique is equally effective in nonhealthy horses and whether preventing hypoxaemia during the early maintenance phase will improve overall short- and long-term outcome.

Authors' declaration of interests

No competing interests have been declared.

Ethical animal research

The study complied with the institutional ethical guidelines of the University of Utrecht Equine Hospital. Animals included in this study were client owned horses, for which owner consent was obtained.

Sources of funding

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Authorship

H. van Oostrom contributed to study design, study execution, data analysis and interpretation, preparation of the manuscript and final approval of the manuscript. M.W.H. Schaap contributed to data analysis and interpretation, preparation of the manuscript and final approval of the manuscript. T. van Loon contributed to study design, study execution, data analysis and interpretation, preparation of the manuscript and final approval of the manuscript.

Manufacturers' addresses

[a]MSD Animal Health, Boxmeer, the Netherlands.
[b]Zoetis Animal Health, Capelle a/d IJssel the Netherlands.
[c]Vetoquinol, Breda, the Netherlands.
[d]Actavis, Hafnarfjordur, Iceland.
[e]Siemens Healthcare Diagnostics, Tarrytown, New York, USA.
[f]Boule Medical AB, Spånga, Sweden.
[g]Datex Ohmeda, Helsinki, Finland.
[h]Microsoft, Redmond, Washington, USA.
[i]IBM Software, Armonk, New York, USA.

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Supporting Information

Additional Supporting Information may be found in the online version of this article at the publisher's website:

Supplementary Item 1: Characteristics of horses recruited for the study.

Supplementary Item 2: Equation used for calculation of shunt fraction [2–4].