ARTICLES

Alleviation of postanesthetic hypoxemia in the horse

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Abstract

This study was designed to investigate the effect of the nasotracheal insufflation of oxygen at a flow rate of 15 L/min on the arterial partial pressure of oxygen during the recovery period following inhalation anesthesia in the horse. It has been stated that this is a suitable flow rate to prevent postoperative hypoxemia but without any experimental evidence to support those statements. Horses being used for the study of healing of cartilage were anesthetized on two separate occasions. Following one period of anesthesia they were allowed to recover breathing room air, and following the other period of anesthesia oxygen was insufflated into the trachea at 15 L/min throughout the recovery period. This permitted each horse to act as its own control and allowed statistical analysis using Student's *t*-test for paired samples.

The insufflated horses had a higher arterial partial pressure of oxygen during the recovery period than did the noninsufflated horses ($p < 0.05$).

Résumé

La réduction de l'hypoxémie post-anesthésie chez le cheval

Cette étude avait pour but d'évaluer l'effet de l'insufflation naso-trachéale d'oxygène à un débit de 15 L/min sur la pression partielle d'oxygène artériel durant la période de réveil d'une anesthésie par inhalation chez le cheval. Le débit utilisé a déjà été suggéré comme étant adéquat pour contrôler l'hypoxémie post-anesthésie chez le cheval quoique ceci n'ait jamais été appuyé par une étude expérimentale. Des chevaux utilisés pour une étude sur la guérison du cartilage ont été anesthésiés à deux reprises. Les chevaux ont respiré l'air ambiant durant la période de réveil de la première anesthésie tandis qu'on leur a administré l'oxygène à 15 L/min dans la trachée lors du réveil de la seconde anesthésie. Chaque animal a donc été son propre contrôle, ce qui a permis de compléter une analyse statistique à l'aide du test de Student pour échantillons pairés.

Les chevaux qui ont reçu l'oxygène par insufflation naso-trachéale avaient une pression partielle d'oxygène artérielle plus grande durant la période de réveil que ceux qui n'en recevaient pas ($p < 0,05$).

Can Vet J 1989; 30: 37-41

Introduction

Hypoxemia (arterial partial pressure of oxygen less than 60 mm Hg) commonly develops in horses in the immediate postanesthetic period regardless of the position, mode of ventilation, or arterial oxygen levels during the anesthetic period (1-3). Horses under general anesthesia frequently have high alveolar-arterial partial pressure oxygen differences (A-a PO_2), largely due to inequalities of ventilation and perfusion of the lungs (4-6). If the horse receives 100% oxygen, the arterial partial pressure of oxygen (PaO_2) during anesthesia is generally adequate, even with ventilation/perfusion mismatch. During recovery however, horses are usually breathing room air and become hypoxemic because of the existing pulmonary problems. Increasing the concentration of inspired oxygen during the recovery period has been used in attempts to prevent this period of hypoxia. Recommendations for flow rates for insufflation of oxygen into the nares or trachea have been made varying from 5-15 L/min (1,7,8). A recent study using an oxygen insufflation rate of 10 L/min found no significant difference in PaO_2 between horses which were insufflated and those that were not, however, these horses were insufflated for only the first ten minutes of the recovery period (2). Use of a demand valve that delivered 50 L/min of oxygen improved oxygenation in these horses. However, use of a demand valve requires that the horse be intubated. Once the valve is removed and the horse extubated, PaO_2 rapidly declines.

Our purpose in this study was to determine if a higher insufflation flow rate of 15 L/min throughout the recovery period would significantly raise the arterial oxygen content. At least two previous reports recommended the use of this flow rate, however one of these provided no data to support the claim (7) and the second measured arterial oxygen once only, at 15 minutes into the recovery period (1). This second

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Supported by Dean's CDA grant, Western College of Veterinary Medicine.

trial had no control group for the horses in dorsal recumbency. We had the opportunity to carry out a trial on twelve horses that were to be anesthetized on two occasions, at intervals of six weeks. We could therefore use each horse as its own control to study the effects of insufflation of 15 L/min^{-1} of oxygen throughout the recovery period compared with breathing room air during recovery.

Materials and methods

Twelve healthy adult horses undergoing arthroscopy for determination of the healing processes in cartilage were to be anesthetized on two separate occasions. Horses were of both sexes and weighed 428 ± SD 43 kg. A physical examination and a complete blood count were done the morning before each surgery.

Immediately prior to anesthesia, an arterial blood sample was obtained by percutaneous puncture from the carotid artery and analyzed immediately for pH, PaO_2, partial pressure of carbon dioxide ($PaCO_2$), bicarbonate concentration (HCO_3), total carbon dioxide content (Total CO_2), base excess (ABE), hemoglobin concentration, and hemoglobin saturation.

The same anesthetic regimen was used for each surgery. The horses were premedicated with xylazine (Rompun: Haver, Bayvet Division, Chemagro Ltd., Etobicoke, Ontario) 0.4 mg/kg, intravenously. Guaifenesin, 5% solution in 5% dextrose, was infused intravenously until the horse showed signs of unsteadiness, and then sodium thiamylal (Thialean, MTC Pharmaceuticals, Canada Packers Inc., Cambridge, Ontario) 6 mg/kg, was given intravenously over a period of thirty seconds. An endotracheal tube was placed and the horse positioned in dorsal recumbency and connected to a large animal anesthetic machine with an integral ventilator (Drager NA Large Animal Control Center, Hoechst Ltd., Montreal, Quebec) and an out-of-circle vaporizer (Drager 19, Hoechst Ltd., Montreal, Quebec). Anesthesia was maintained with halothane (Somnothane, Hoechst Ltd., Montreal, Quebec) in 100% oxygen and ventilation was controlled using a tidal volume of 15 mL/kg at a respiratory rate of six breaths per minute. Following induction of anesthesia, a 5 cm 20 gauge teflon catheter was placed in a transfacial artery, and blood pressure was measured via a Bell and Howell blood pressure transducer (CEC Division, Bell and Howell, Pasadena, California) and heart rate and rhythm were monitored via an oscilloscope (Burdick M200 Monitor, The Burdick Corporation, Milton, Wisconsin). End tidal CO_2 (ET CO_2) was monitored with a capnograph (CO_2 Monitor and Recorder, Puritan Bennet Corp., Los Angeles, California). Ringer's solution was administered at a rate of 10 mL/kg/h. Tidal volume was varied as required to keep blood gases within normal range according to the results obtained from blood gas analysis (ABL 330: Radiometer, Copenhagen) and the capnograph readings. Blood samples were corrected for hemoglobin and body temperature. The ABL 330 automatically performs a one point calibration every two hours and a two point calibration every four hours.

All horses were allowed to recover in left lateral recumbency under the same conditions. Following the first period of anesthesia, six of the horses received oxygen insufflation into the trachea at 15 L/min during the entire recovery period and six horses breathed room air. Oxygen was administered via a 15 mm outside diameter plastic tube inserted into the trachea as the endotracheal tube was withdrawn. The insufflation tube was taped to the horse's head to retain it in place until after the horse was standing. Arterial blood gas samples were drawn prior to induction, twice during anesthesia (once during controlled ventilation and once during spontaneous ventilation), immediately after the horse was placed in the recovery room, and every five minutes thereafter, until the horse regained sternal recumbency. A sample was drawn immediately after the horse rolled into sternal recumbency, and again when standing. Samples were analyzed immediately. After the second period of anesthesia the situation was reversed, and those horses that had been insufflated were allowed to recover breathing room air, while those that had not received oxygen were insufflated. Each horse thus served as its own control.

Group A horses were those horses breathing room air and group B horses were those horses insufflated with oxygen at 15 L/min during recovery. There were no abnormalities in the automatic calibration of the blood gas analyzer at any time during the investigations. Comparisons were made within each group between baseline values and values at each time period. Comparisons were also made between the values of the final blood sample obtained from the horses in lateral recumbency (last lateral) and the samples obtained when the horses assumed sternal recumbency and the samples when they were standing. Results were analyzed using Student's *t*-test for paired samples and a statistical analysis program (StatView 512K+, Brain-Power Inc., Calabas, California) on a Macintosh computer (Apple Computer Inc., Cupertino, California). $p < 0.05$ was considered statistically significant.

The guidelines, "Guide to the Care and Use of Experimental Animals, Volumes I and II", published by the Canadian Council on Animal Care were followed, and the protocol was approved by the Animal Care Committee of the University of Saskatchewan.

Results

All horses were healthy and had no detectable cardiopulmonary abnormalities. The total anesthetic time for group A horses was 101 ± 26 min and for group B horses was 93 ± 20 min. There was no significant difference in anesthetic time between the two groups. Two horses in group A went from lateral recumbency directly to standing without assuming sternal recumbency long enough for a sample to be collected. This also occurred with two other horses in group B. Three of the horses in group B pulled the nasotracheal catheter out as they rose to standing, so no standing blood gas samples were taken. Group A horses took 41.4 ± 13.5 min from the end of anesthesia until they were standing; group B horses took 39.1 ± 12.2 min. There was no significant difference between the two groups.

TABLE 1

Arterial blood gas analysis results in horses recovering from anesthesia, breathing room air (group A) or insufflated with oxygen at 15 L/min (group B)

	Baseline	Spont. vent	3 min	8 min	13 min	18 min	23 min	Last lateral	Sternal	Standing
Group A (room air)										
pH	7.415	7.286[a]	7.374[a]	7.381[a]	7.381[a]	7.381[a]	7.389	7.385[a]	7.369[a]	7.402
	±0.027	±0.040	±0.062	±0.037	±0.041	±0.035	±0.034	±0.036	±0.031	±0.037
$PaCO_2$	43.9	64.0[a]	48.7[a]	48.2[a]	48.1[a]	47.9[a]	49.2[a]	48.1[a]	48.2[a]	46.7[a]
	±2.9	±7.1	±6.6	±4.2	±4.7	±3.5	±3.8	±4.0	±3.3	±3.8
PaO_2	102.9	188.6[a]	65.4[a]	62.8[a]	61.5[a]	60.8[a]	61.4[a]	58.5[a]	75.2[a,d]	88.3[a,d]
	±12.9	±86.2	±8.6	±8.8	±8.3	±8.9	±8.7	±7.4	±12.4	±9.8
HCO_3	27.53	29.16[a]	27.70	27.78	27.60	27.97	27.92	28.2	27.28	28.34
	±1.65	±2.58	±2.40	±2.43	±2.24	±2.61	±2.38	±2.2	±3.10	±2.78
BE	3.0	1.5[a]	2.2	2.5	2.3	2.8	2.6	2.9	1.9	2.8
	±1.6	±2.4	±2.7	±2.5	±2.3	±2.2	±2.3	±2.2	±3.1	±2.8
(n)	(12)	(12)	(12)	(12)	(11)	(10)	(9)	(12)	(10)	(12)
Group B (insufflated)										
pH	7.424	7.286[a]	7.321[a,c]	7.324[a,c]	7.341[a,c]	7.345[a]	7.338[a,b]	7.360[a]	7.332[a,c]	7.371[a]
	±0.018	±0.051	±0.053	±0.053	±0.053	±0.049	±0.034	±0.030	±0.028	±0.035
$PaCO_2$	45.0	62.4[a]	56.7[a,c]	55.0[a,c]	54.5[a,c]	55.0[a,c]	52.5[a,b]	51.7[a,c]	54.8[a,c]	48.0[a]
	±3.3	±8.8	±8.0	±7.4	±7.4	±6.2	±5.3	±4.7	±6.3	±4.0
PaO_2	109.2	204	91.4[c]	101.0	100.2[c]	102.7[c]	100.5[a,b]	94.3[c]	107.4[c]	118.7[c]
	±14.7	±101.5	±28.9	±40.3	±42.0	±41.6	±40.5	±30.75	±24.9	±21.8
HCO_3	28.82	28.33	28.03	27.60[a]	28.09	28.29	27.18[a,b]	28.20	28.12	27.09[a]
	±1.69	±1.25	±1.73	±1.74	±1.67	±1.73	±2.71	±1.80	±2.48	±1.49
BE	4.2	0.7[a]	1.3[a]	1.0[a]	1.9[a]	1.9[a]	1.1[a,b]	2.2[a]	1.4[a]	1.6[a]
	±1.4	±1.6	±1.9	±2.2	±1.9	±2.1	±2.5	±1.6	±2.1	±1.6
(n)	(12)	(12)	(12)	(12)	(12)	(8)	(6)	(12)	(10)	(9)

Data expressed as mean ±1 SD

[a]Significantly different ($p < 0.05$) from baseline values

[b]Insufficient number of paired samples for statistical analysis between group A and group B

[c]Significantly different ($p < 0.05$) from values on room air

[d]Significantly different ($p < 0.05$) from last lateral values

There were no significant differences in pH, $PaCO_2$, PaO_2, HCO_3 or ABE between group A (room air) or group B (insufflated) horses prior to anesthesia (baseline values). The pH was significantly lower than baseline for group A horses for the first 18 min of the recovery period, and then it gradually increased and there was no significant difference from the values at 23 min and the values when standing (Table 1). For group B horses, pH remained significantly lower than baseline at all times. The $PaCO_2$ was significantly higher than baseline at each sampling time for both groups of horses. The $PaCO_2$ was significantly higher in the group B horses than in group A horses (room air) from 3 min to 18 min. Due to variations in recovery time, we were only able to match sufficient horses for the paired Student's *t*-test between groups A and B through 18 min. At 23 min there were insufficient matched pairs in groups A and B for valid comparison. The $PaCO_2$ was also significantly higher in group B horses when in sternal recumbency than in group A horses, although this difference disappeared when both groups were standing. There was a significant difference between baseline HCO_3 in group A and the value when spontaneously breathing on the anesthetic machine, but at no other times. For group B, there was a significant difference from baseline at 8 min and at standing, but not at any other sampling time. There was no significant difference in HCO_3 between group A and group B at any sampling time. There were no significant differences in ABE between baseline and any other sampling time for either group, and there were no significant differences between groups.

PaO_2 was significantly higher than baseline for group A horses when they were spontaneously breathing on the anesthetic machine at the end of the anesthetic period. PaO_2 dropped rapidly, and from 3 min to standing values were significantly lower than baseline. For group B horses there was no significant difference between the spontaneously breathing horses on the anesthetic machine and baseline (Table 1). There was no significant difference in PaO_2 from baseline at any other sampling time either. At 3, 8, 13, 18 min and in sternal and standing samples, PaO_2 in group B (insufflated) horses was significantly higher than in the group A (room air) horses. At 23 min there were insufficient pairs for valid statistical analysis.

In addition to comparing values at fixed time periods, an analysis was made on the last samples collected when each horse was still in lateral recumbency (last lateral) (Table 1). The time for collecting these samples ranged from 13 to 43 min. For group A horses, there was a significant difference between last lateral and baseline. pH was lower, $PaCO_2$ and PaO_2 were elevated, in last lateral group compared to baseline. Last lateral pH was significantly lower than baseline for group B horses and $PaCO_2$ levels were elevated. There was no significant difference between last lateral pH values for the group A horses and last lateral values for the group B horses. There was no significant difference between last lateral and sternal pH and $PaCO_2$ values in either group A or in

group B horses (Table 1). There was a significant difference between group A last lateral $PaCO_2$ and group B last lateral $PaCO_2$, and between group A sternal $PaCO_2$ and group B sternal $PaCO_2$; group B horses had a significantly higher $PaCO_2$. There were no significant differences in HCO_3 or ABE in last lateral or sternal recumbency, either within or between groups.

There was a significant difference between last lateral PaO_2 and sternal PaO_2 for group A horses. There was an immediate rise in PaO_2 when the horses rolled into sternal recumbency. There was no significant difference between last lateral PaO_2 and sternal PaO_2 in group B horses, but there was a large standard deviation between horses in that group (the PaO_2 ranged from 61–148 mm Hg in last lateral and from 73–146 mm Hg in sternal). There was a significant difference in last lateral PaO_2 between group A and group B horses and also for sternal PaO_2 between groups (Table 1). In both group A and group B horses, the last lateral pH was significantly lower than baseline.

Discussion

Hypoxemia concurrent with normocarbia may result from low inspired oxygen concentration, diffusion impairment, right-to-left pulmonary vascular or cardiac shunts, ventilation/perfusion inequalities, and decreased mixed venous oxygen content (9). The most likely cause for hypoxemia without increased levels of carbon dioxide in healthy horses under general anesthesia on 100% oxygen is ventilation/perfusion inequalities or decreased mixed venous oxygen due to decreases in cardiac output, or a combination of both.

Both groups of horses became slightly hypercarbic by 3 min into the recovery period when taken off controlled ventilation, with a mean $PaCO_2$ of 64 mm Hg for group A (room air) and 62.4 mm Hg for group B (insufflated). Group A horses (room air) had their arterial CO_2 tensions back to near normal, around 48 mm Hg at the 8 min sample, but the horses that were insufflated with oxygen (group B) had values that remained greater than 50 mm Hg throughout, and were significantly higher than the group A horses. Group B horses had a resulting decrease in arterial pH due to respiratory acidosis. In awake horses that become hypoxemic, there is a compensatory increase in respiratory rate with a resulting decrease in arterial CO_2 due to stimulation of peripheral chemoreceptors (11–13). Carbon dioxide response curves have been used to demonstrate the depressant effects that anesthetics have on central chemoreceptor responses to rising CO_2 (14,15), but the response of peripheral chemoreceptors to hypoxemia may be active in the recovery period. This could explain why the horses that were not insufflated and became hypoxemic had significantly lower $PaCO_2$ values than those which received oxygen.

Although many horses under anesthesia cannot be classed as hypoxemic ($PaO_2 < 60$ mm Hg), they have significant differences in alveolar and arterial oxygen levels (4–6). When these horses are receiving 100% oxygen, they seldom have arterial oxygen tensions low enough for oxygen saturation of hemoglobin to fall below 90–95%, since this would require a PaO_2 of less than 60 mm Hg. However, when horses are recovering from anesthesia, their inspired oxygen concentration is only 21%, if it is not supplemented with oxygen, and arterial oxygen tensions will rapidly fall to levels as low as 50 mm Hg (1,2).

Tissue hypoxia may result from hypoxemia, low levels of hemoglobin or inability of hemoglobin to carry oxygen, poor perfusion, or failure of oxygen utilization by the cells such as occurs with cyanide or alcohol poisoning (10). Horses that are anesthetized or recovering from anesthesia often have less than optimal cardiac output and perfusion pressures and simultaneous hypoxemia, such as that which often occurs during recovery, would increase the chance of tissue hypoxia occurring. Therefore it is important to try to prevent this hypoxemia from occurring in order to minimize the risks of anesthetic-related decreases in cardiovascular function.

Authors of previous reports on the use of oxygen insufflation during the recovery period make various and conflicting recommendations. Donawick and Alexander (7) stated that an insufflation rate of 5–8 L/min would maintain the PaO_2 at 100 mm Hg yet no supporting data were provided. DeMoor *et al* (1) investigated the use of 15 L/min of insufflated oxygen during recovery and found that the horses which were insufflated had significantly higher arterial oxygen tensions than the horses which were not insufflated, but only measured this one time during the recovery, at 15 min. Also, only one group of their horses (those that had been in lateral recumbency) had control horses to compare with. The horses that had been in dorsal recumbency were all insufflated so comparisons could not be made. In the horses that had been in dorsal recumbency, insufflation did not prevent hypoxemia in all horses since they reported a low value of 53 mm Hg. Mason *et al* (2) studied the effects of 10 L/min of oxygen insufflation during the recovery period and found that those horses which breathed room air had a lower PaO_2 than those which were insufflated, but they were unable to show a statistically significant difference between the groups. In our study, horses insufflated with a flow rate of 15 L/min had a significantly higher PaO_2 than when not supplemented with oxygen, suggesting that insufflation with this higher flow rate is a useful technique to prevent hypoxemia during recovery. One of the group B horses in this study did have a PaO_2 of 61 mm Hg, so even at this flow level a degree of hypoxemia may occurr. The lowest PaO_2 measured was 52 mm Hg in a group A horse. Even so, this horse had an oxygen saturation of 82.3%. The lowest PaO_2 in group B was 61 mm Hg with an oxygen saturation of 88.5%. It may be that even this small increase in saturation would significantly increase the amount of oxygen available to the tissues, which could be critical in the recovery period in a sick horse. Conditions such as acidosis, metabolic or respiratory, shift the oxyhemoglobin dissociation curve to the right. In these circumstances, a higher partial pressure of oxygen is required for hemoglobin to bind a given amount of oxygen (16).

Also noted in our study is that the group that was not supplemented with oxygen had a significant and immediate rise in PaO_2 when they rolled into a sternal position. Thus, encouraging or rolling a horse into sternal recumbency may also be helpful in alleviating hypoxemia during recovery. This is in agreement with the findings of Mason *et al* (2).

Insufflation of oxygen into the trachea of horses during the recovery period is very simply done. In this study we were using oxygen from a wall outlet and, because of our recovery room set-up, we used a separate flow meter for oxygen delivery. However, in most instances an oxygen insufflation tube can be run directly from the flowmeter of the anesthetic machine using a length of plastic tubing. The tubing is inserted through the nasal cavity and into the trachea as the endotracheal tube is withdrawn. A flow of at least 15 L/min of oxygen should be used, which would prevent hypoxemia in most horses. Oxygen insufflation should certainly be used for all sick horses, particularly those suffering from acidosis and shock, and those with respiratory difficulties, where even a small increase in arterial oxygen partial pressure could be the difference between life and death. If horses are not given supplemental oxygen, they should be rolled into sternal recumbency as soon as possible. CVJ

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