Anesthesia Case of the Month

Sponsored by the American College of Veterinary Anesthesiologists. Comments and contributions should be directed to Dr. Jeff C. H. Ko, Department of Medicine and Surgery, College of Veterinary Medicine, Oklahoma State University, Stillwater, OK 74078, telephone: 405/744-8584; e-mail: jeff_ko@okway.okstate.edu

History

An 8-year-old 494-kg (1,086-lb) Thoroughbred gelding was referred because of a history of abdominal discomfort of 8 hours' duration. The horse had been treated with mineral oil via a nasogastric tube as well as IV administration of flunixin meglumine, xylazine hydrochloride, and butorphanol tartate prior to admission to the hospital. Upon admission, the horse had signs of abdominal pain and depression, with a heart rate of 75 beats/min and a respiratory rate of 18 breaths/min. The horse was moderately dehydrated and had a low number of borborygmic sounds. Findings on thoracic auscultation were normal, and abdominal palpation per rectum revealed a slightly distended cecum. Results of abdominal and thoracic ultrasonography were unremarkable. No abnormalities were detected on CBC, serum biochemical analysis, and peritoneal fluid analysis. An arterial blood sample was obtained for pH and blood gas analysis (**Table 1**).[a] Initial treatment consisted of IV administration of lactated Ringer's solution at the rate of 6 L/h. The horse's signs of abdominal pain increased during the next 2 hours, and exploratory celiotomy was performed.

The horse was given a dose of xylazine (540 mg, IV), and anesthesia was induced with ketamine hydrochloride (1,100 mg, IV) approximately 5 minutes later. The horse was intubated with a 26-mm-diameter cuffed endotracheal tube that was connected to a large animal circuit, and the horse was allowed to breathe spontaneously. Isoflurane in oxygen was delivered at initial settings of 4% and 10 L/min, respectively. An arterial cannula was placed in the right facial artery.[a] Throughout the anesthesia period, lactated Ringer's solution was administered IV at approximately 12 L/h. After 30 minutes, the oxygen flow was reduced to 5 L/min, and the vaporizer setting was incrementally decreased starting 15 minutes after inhalation anesthesia was begun.

The **mean arterial pressure (MAP)** was within reference range or slightly high during anesthesia and ranged from 92 to 115 mm Hg without inotropic support. The **respiratory rate (RR)**, 20 breaths/min, increased after intubation, and the arterial blood gas values obtained 20 minutes after induction indicated hypoxemia and hypercapnia (Table 1). **Intermittent positive-pressure ventilation**[b] **(IPPV)** was instituted with a RR of 10 breaths/min, but the horse continued to breathe spontaneously. Another arterial pH and blood gas tension analysis was performed 30 minutes after induction (Table 1), which indicated a further decrease in Pa_{O_2} and no further change in Pa_{CO_2}. It was also observed that the peak inspiratory pressure was higher than expected. The surgeons were advised of the difficulty encountered ventilating the horse and that the celiotomy should begin as soon as possible to relieve any abdominal distention. After the abdomen was opened by a ventral midline incision, no visceral distention was apparent. Another arterial pH and blood gas analysis was performed (40 minutes after induction), and there was no change in Pa_{O_2} and a slight increase in Pa_{CO_2}. At this time, the horse was becoming increasingly difficult to ventilate (ie, increased spontaneous ventilation and increased peak inspiratory pressures without improvement in Pa_{O_2}). Fifty minutes after induction, the ventilator settings were as follows: 10 breaths/min, controlled with spontaneous respirations; inspiratory time, 2.65 seconds; and **inspiratory-to-expiratory ratio (I:E)**, 1:1.2. The resulting tidal volume and peak inspiratory pressure were 6 L/min and 38 cm H_2O, respectively. There were no substantial changes in the blood gas values at this time (Table 1).

Question

Why was this horse so difficult to ventilate?

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Table 1—Arterial pH and blood-gas values immediately before induction (at admission) and during anesthesia and exploratory celiotomy in a Thoroughbred

Variable	Admission	20 min*	30 min*	40 min*	50 min*	70 min*
pH	7.450	7.350	7.357	7.329	7.363	7.364
Pa_{CO_2} (mm Hg)	44.1	54.8	54.7	61.0	53.6	52.2
Pa_{O_2} (mm Hg)	83.3	53.6	48.2	50.4	53.4	109.8
HCO_3^- (mmol/L)	30.9	30.7	31.0	32.4	30.8	30.0
BE (mmol/L)	+6.1	+4.0	+4.4	+4.8	+4.1	+3.3

*Times represent minutes after induction, and the 70-minute results are from an arterial blood sample obtained after reduction of a diaphragmatic hernia (removal of the ascending colon from the thorax).
HCO_3^- = Bicarbonate. BE = Base excess.

Answer

When no abnormalities were identified involving the visible intestine, the cranial region of the abdomen was explored, and a diaphragmatic hernia was discovered in the left dorsal quadrant of the diaphragm. The rent was approximately 10 × 25 cm in length, and most of the ascending colon was displaced into the thoracic cavity. Enlargement of the rent was required to allow the removal of the colon from the thoracic cavity.

The arterial blood gas values improved immediately after the colon was removed (70 minutes after induction) from the thorax (Table 1). In addition, the spontaneous breathing stopped within 5 minutes, and the horse was much easier to ventilate (no spontaneous ventilation and decreased peak inspiratory pressures with improvement in Pa_{O_2}). At this point, the ventilator settings were as follows: 10 breaths/min, controlled with no spontaneous respirations; inspiratory time, 2.2 seconds; and I:E, 1:2.2. The resulting tidal volume and peak inspiratory pressure were 8 L/min and 22 cm H_2O, respectively. Because of the poor prognosis for repair resulting from the dorsal location and extent of the defect, the owners elected to have the horse euthanatized. A subsequent necropsy did not reveal other abnormalities, and the hernia was determined to be the result of recent trauma as evidenced by the rent's swollen hemorrhagic edges. The owners did not know of any recent traumatic incident.

Discussion

Hypoxemia is defined as a Pa_{O_2} < 80 mm Hg.[1] In the horse of our report, hypoxemia during anesthesia was likely a result of the combination of the pulmonary changes commonly seen in horses during anesthesia and the additional effect of the colon displaced into the thorax. The role of the hernia in the horse's pulmonary dysfunction is evidenced by the improvement seen immediately after it was reduced.

Changes in the pattern of pulmonary perfusion when horses are anesthetized have been documented by Dobson et al.[2] Anesthetized horses have increased perfusion in the caudodorsal lung fields irrespective of positioning. This selective caudodorsal perfusion differs from what is observed in conscious horses where the pulmonary perfusion is determined by hydrostatic forces and, therefore, is greatest in whatever portion of the lung field is dependent. Robinson[3] has summarized this phenomenon and described other physiologic factors in pulmonary function in anesthetized horses. In addition, there is a decrease in hypoxic vasoconstriction within the lung during isoflurane-induced anesthesia.[4] Nyman and Hedenstierna[5] documented a dramatic decrease in Pa_{O_2} in anesthetized horses attributable to alveolar collapse, which results in an increased intrapulmonary shunt fraction. They reported the primary mechanism producing the increased shunt fraction was alveolar collapse rather than airway closure.

The extreme difficulty in oxygenating arterial blood encountered in our horse and its lack of response to IPPV prior to hernia reduction suggests a similar mechanism involving alveolar collapse. The improvement seen in Pa_{O_2} after reduction of the hernia was more pronounced than the response to IPPV often seen in an otherwise clinically normal horse anesthetized and positioned in dorsal recumbency. This difference was likely the result of the large amount of the lungs that had alveolar collapse. Once the compression induced by the entrapped colon was removed, IPPV of the entire lung field had an effect similar to that seen in Nyman's[6] study. In that study, selective ventilation of the dependent lung fields, the areas with the most alveolar collapse in an anesthetized horse in dorsal recumbency, resulted in profound improvement in Pa_{O_2}. Because of the widespread alveolar collapse in our horse, IPPV of the entire lung field had an effect similar to selective ventilation of the area of collapsed alveoli in Nyman's study.[6]

Reports of herniation of the large colon through the diaphragm are uncommon.[7-11] Because of the size of the large colon, the effect on ventilation may be more pronounced when herniation through the diaphragm involves the large colon. In one report,[9] 2 of 7 horses undergoing general anesthesia that had large-colon herniation into the thorax died shortly after anesthesia induction because of an inability to ventilate. In our horse, the presence of the large colon in the thorax contributed substantially to the difficulty in ventilating, and adequate ventilation was achieved only after reduction of the hernia.

[a]SenDx 100 acid-base, blood gas, electrolyte analyzer, SenDx Medical Inc, Carlsbad, Calif.

[b]Rachel Model 2800 large animal anesthesia ventilator, Mallard Medical Inc, Shasta Lake, Calif.

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