Isoflurane anesthesia in a rhinoceros

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- Endotracheal intubation and maintenance of anesthesia with isoflurane in oxygen following induction with injectable agents is a feasible anesthetic protocol for adult rhinoceroses.
- Complications encountered during the monitoring period may include respiratory depression and ventilation/perfusion inequality, as indicated by hypercapnia and hypoxemia.
- Arterial blood pressure monitoring appears to be an effective method for monitoring anesthetic depth and response to surgical stimulation.

A 15-year-old female white rhinoceros (*Diceros simus*), weighing approximately 2,000 kg, was anesthetized for surgical exploration of the reproductive tract. The rhinoceros was infected with *Mycobacterium bovis*, and euthanasia was planned as part of the decontamination process being undertaken in its habitat. The animal was transported in a metal crate on a flatbed semitrailer to Louisiana State University's Veterinary Teaching Hospital and Clinic for general anesthesia, surgery, euthanasia, and necropsy.

On arrival, 2.5 mg of etorphine HCl[a] was injected into the left triceps muscle by use of a dart rifle and projectile syringe. Approximately 20 minutes after injection, the rhinoceros was leaning against the side of the crate and muscle tremors were noticed in the forelimbs, which persisted for 30 minutes. Fifty minutes after etorphine injection, the rhinoceros was pulled from the crate, using large bands and a fork lift, and positioned in left lateral recumbency on 2 dunnage bags[b] (1.3 × 2.3 m), which were then filled with air to provide padding for the dependent side. A 16-gauge, 2-inch catheter[c] was placed in an auricular vein on the lateral aspect of both ears. Twenty-gauge, 2-inch catheters[c] were placed in 2 auricular arteries on the medial aspect of the right ear. One arterial catheter

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was connected to a pressure transducer that was positioned and calibrated at heart level to record the arterial blood pressure (ABP), and was traced on a multichannel video monitor.[d] A base-apex ECG was continuously recorded in lead II.[d] The second arterial catheter was used to collect serial blood samples for blood gas analyses and creatine kinase activity.

After baseline heart rate, respiratory rate, and ABP values were recorded, endotracheal intubation was performed to facilitate delivery of isoflurane in oxygen and intermittent positive-pressure ventilation. Intubation was difficult initially because of the small size of the mouth and lack of muscle relaxation. An additional 1 mg of etorphine, 10 mg of acepromazine maleate (0.005 mg/kg of body weight), 35 g of guaifenesin (17.5 mg/kg), and 1,400 mg of thiamylal sodium (0.075 mg/kg) were administered over a 10-minute period to improve muscle relaxation. The technique used to perform intubation proved to be a combination of techniques described for horses and cattle. The intubator's arm was introduced into the oral cavity to palpate the larynx, but only the tip of the epiglottis could be reached because of the distance from the mouth to the larynx. A 30-mm internal-diameter low-pressure, high-volume cuffed endotracheal tube[e] was guided into the oral cavity until the tip was positioned at the glottis, as indicated by movement of air in and out of the tube. Attempts to advance the tube resulted in its movement into the esophagus. At this time, a 10-mm internal-diameter nasogastric tube was introduced into the endotracheal tube and advanced into the trachea. The endotracheal tube was then advanced into the trachea, with the nasogastric tube acting as a guide, and the cuff inflated to form an effective seal.

A large-animal anesthetic machine,[f] with a precision vaporizer[g] for isoflurane[h] and a circle breathing system with a 30-L rebreathing bag, was used to deliver 2% isoflurane in 100% oxygen (10 L/min). At this time, a pulse oximeter[i] probe was attached to the tongue to record arterial oxyhemoglobin saturation (Sa_{O_2}) for comparison to the Pa_{O_2} values obtained with blood-gas analyses. The contribution of errors incurred by motion and tracking

of each pulsation was minimized by ensuring that the oximeter was accurately assessing pulse rate when each Sa_{O_2} reading was taken. Although ABP and heart rate (mean ABP, 86 mm of Hg; heart rate, 86 beats/min) indicated adequate cardiovascular function, the slow respiratory rate (2 breaths/min) and absence of a palpebral reflex prompted a reduction in the delivered isoflurane concentration to 1%. Forty minutes after the initial delivery of isoflurane in oxygen, intermittent positive-pressure ventilation was initiated in an attempt to correct the severe hypercapnia (Pa_{CO_2}, 100 mm of Hg) and relative hypoxemia (Pa_{O_2}, 91 mm of Hg, with FiO_2 = 1.0). A bag-in-a-barrel-type ventilator with a 30-L capacity, cycled by a pressure-cycled ventilator,[j] was set at 6 breaths/min and a peak inspiratory pressure of 25 cm of H_2O.

Surgery was initiated via a right flank celiotomy 1 hour after isoflurane delivery was initiated and 95 minutes after the second injection of etorphine. Because of an increase in ABP at the start of surgery (mean ABP increased from 88 to 120 mm of Hg), isoflurane concentration was increased to 2% and maintained at that concentration until the end of the anesthetic period. At the end of the 60-minute surgical procedure, intermittent positive-pressure ventilation and isoflurane in oxygen delivery were discontinued to allow monitoring of the degree of change in arterial oxygenation associated with breathing room air (Pa_{O_2}, 61 mm of Hg, with FiO_2 = 0.2) after breathing 100% oxygen (Pa_{O_2}, 131 mm of Hg, with FiO_2 = 1.0). Unassisted ventilation resumed within 60 seconds, and the palpebral reflex returned 7 minutes after discontinuation of isoflurane delivery. The rhinoceros was then euthanatized by lethal injection.[k]

The data collected during general anesthesia of this white rhinoceros expand on previous reports of immobilization techniques for and data collected from white and black rhinoceroses.[1-8] Tachycardia, high arterial blood pressure values, respiratory depression, muscle tremors, and hyperpyrexia are adverse effects of etorphine administration that have been reported in several species[3] and also were observed in the rhinoceros of this report. The rectal temperature (39.7 C) recorded 120 minutes after the initial etorphine injection was high compared with most reported rectal temperatures for white[1,7] and black[4-6] rhinoceroses immobilized with etorphine. One study of black rhinoceroses reported a rectal temperature range of 36.5 to 41.2 C, and the investigators suggested that a temperature of 36.5 C may represent resting body temperature of that species. Another investigator reported that temperature changes in black rhinoceroses were directly related to behavior and to variations in the ambient temperature.[9] The high body temperature recorded for the rhinoceros of this report may have been influenced by the high ambient temperature on the day of the study (mean temperature, 30 C) and by the prolonged period of transport (4 hours) that immediately preceded anesthesia. Rectal temperature decreased during the anesthetic period to within those ranges previously reported.[4,5]

Heart rates recorded from the rhinoceros of this report during anesthesia varied between 86 and 116 beats/min. These values were high compared with most mean heart rates of chemically immobilized black rhinoceroses.[4-6] When compared with the heart rate range of 1 immobilized adult white rhinoceros,[2] these values are high, but are similar to the heart rate reported for another white rhinoceros.[1] The importance of the heart rates recorded from the rhinoceros of this report was difficult to determine, because reference values for rhinoceroses are not available. One author reported heart rates of awake juveniles to range from 70 to 140 beats/min[10]; however, this information may not apply to adult rhinoceroses because juveniles of domestic species have higher heart rates than adults, which is attributable to inherent differences in vagal tone.[11] The heart rate of our rhinoceros varied throughout the anesthetic monitoring period and may have been influenced by body temperature, effects of the various anesthetic agents administered, hypoxemia, hypercapnia, or sympathetic stimulus related to the surgical procedure.

Respiratory rate of the rhinoceros of this report (2 to 10 breaths/min) was similar to the rate reported for 1 immobilized white rhinoceros,[2] but low compared with the rates of the white rhinoceros of another report[1] or for black rhinoceroses.[4-6] Compared with reported ranges for respiratory rate of awake rhinoceroses (20 to 40 breaths/min),[10] respiratory rate was low in our rhinoceros (4 breaths/min) after administration of etorphine injection, and was further decreased by the other parenteral agents used and the isoflurane anesthesia (2 breaths/min). Hypoventilation was confirmed by the detection of hypercapnia (Pa_{CO_2}, 91 to 100 mm of Hg) on the serial blood gas analyses.

Apparent hypertension (mean ABP, 86 to 175 mm of Hg; systolic ABP, 108 to 208 mm of Hg; diastolic ABP, 60 to 160 mm of Hg) was detected after administration of etorphine. Etorphine-induced hypertension has been reported in domestic and exotic animal species, including the white rhinoceros.[1-3,12-15] Hypercapnia and hypoxia, which cause endogenous catecholamine release, may have influenced the high arterial pressure values recorded early during the anesthetic period. Although the initial arterial pressure values seemed high compared with anesthetized horses,[16] the values were difficult to interpret because reference values for awake standing rhinoceroses are not available. The initial decrease in ABP (mean ABP decreased from 175 to 130 mm of Hg) was probably influenced by injection of acepromazine, guaifenesin, and thiamylal. The further decrease in ABP (mean ABP decreased from 130 to 86 mm of Hg) during isoflurane anesthesia and positive-pressure ventilation may have been influenced by a combination

of factors, including the cardiodepressant effects of the injectable agents and isoflurane, the vasodilatory effects of acepromazine and isoflurane, the decrease in venous return secondary to positive-pressure ventilation, the increase in Pa_{O_2}, and the decrease in Pa_{CO_2}. Arterial blood pressure during maintenance with isoflurane was comparable to those reported for anesthetized horses.[16]

Blood gas analyses revealed hypercapnia and hypoxemia (Pa_{O_2}, 61 mm of Hg, with $FIO_2 = 0.2$). Similar effects associated with etorphine immobilization and recumbency in rhinoceroses have been reported.[1,17] General anesthesia is a common cause of hypoventilation, therefore, the hypercapnia, which was more severe than the hypercapnia reported for an etorphine-anesthetized white rhinoceros,[2] was not unexpected. Intermittent positive-pressure ventilation only partially corrected the hypercapnia (Pa_{CO_2} decreased from 100 to 64 mm of Hg), because an adequate minute ventilation was not delivered. Delivered tidal volume decreases over time as lung compliance decreases when a pressure-cycled ventilator is used, but peak inspiratory pressure does not increase; this factor also may have contributed to the inability to achieve and maintain normocapnia. Ventilation/perfusion inequality has been determined to adversely affect carbon dioxide elimination,[18,19] and may have contributed to the severity of hypercapnia in the rhinoceros of this report.

Hypoxemia was attributed to a combination of factors, including hypoventilation and venous admixture. Causes of hypoxemia include hypoventilation, diffusion abnormality, low ventilation: perfusion ratios, and right-to-left shunt.[20] The latter 2 abnormalities are collectively termed venous admixture.[20] Hypoventilation contributed to the hypoxemia, as indicated by an increase in Pa_{O_2} and a decrease in Pa_{CO_2} after initiation of intermittent positive-pressure ventilation. Hypoxemia caused by hypoventilation, diffusion abnormality, and ventilation/perfusion inequality, but not right-to-left shunt, may be corrected by increasing the inspired fraction of oxygen to 1.0.[18] The Pa_{O_2} in our rhinoceros increased to within normal limits after administration of 100% oxygen (Pa_{O_2}, 131 mm of Hg); however, this was a small improvement in Pa_{O_2}, indicating that venous admixture was a cause of the hypoxemia. The alveolar-arterial PO_2 difference for $FIO_2 = 0.2$ and $FIO_2 = 1.0$, which were calculated assuming a normal respiratory quotient of 0.8, also supported venous admixture as the cause of hypoxemia. Venous admixture associated with recumbency and general anesthesia in horses has been well described[21,22] and was not unexpected in this large species. A diffusion abnormality was ruled out as the cause of hypoxemia, as there was no clinical evidence of respiratory disease and there was no evidence of lung abnormalities on postmortem examination that would limit respiratory gas exchange. Also, Pa_{O_2} should have been

much higher while the animal was breathing 100% oxygen if a diffusion abnormality was the primary cause of hypoxemia.[23] Because intermittent positive-pressure ventilation failed to totally correct the hypoventilation and because the necessary data for the calculation of shunt fraction were not collected, the relative impact of hypoventilation, ventilation/perfusion inequality, and right-to-left shunt on the abnormal Pa_{CO_2} and Pa_{O_2} values could not be accurately assessed.

Values for oxyhemoglobin saturation recorded from the oximeter throughout the anesthetic period were inconsistent with the Pa_{O_2} values reported with blood gas analyses. Oxyhemoglobin saturation as an indicator of arterial oxygenation was reported to be a useful monitoring tool during immobilization in an elephant, as indicated by improvement in oxyhemoglobin saturation after the nasal delivery of supplemental oxygen.[24] In the rhinoceros of this report, the correlation between oximetry values for oxyhemoglobin saturation and Pa_{O_2} values obtained from blood gas analyses provided a crude indication of arterial oxygenation status. Oxyhemoglobin saturation values did reflect the large increases and decreases in Pa_{O_2}, which resulted from changing FIO_2 but did not appear to be a sensitive indicator of more subtle changes in Pa_{O_2}.

Creatine kinase activity was high throughout the period of anesthesia (range, 1,767 to 2,030 U/L), compared with 2 reported values for white rhinoceroses (reference ranges, 27 to 159 U/L and 24 to 72 U/L, respectively).[25,26] Enzyme activity actually decreased slightly over the anesthetic period. These high values probably reflected the influence of the 4-hour transport of the rhinoceros just prior to induction of anesthesia.

Because the rhinoceros was euthanatized, duration and quality of recovery could not be evaluated. Recovery was reported to be uncomplicated for a black rhinoceros maintained with halothane in oxygen.[8] It is tempting to speculate that appropriate padding and positioning and maintenance of adequate arterial oxygenation and arterial blood pressure would have resulted in an uncomplicated recovery in the rhinoceros of this report; however, until successful recoveries after inhalation anesthesia in rhinoceroses can be further documented, the usefulness of this technique in this species remains to be determined.

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