CARDIOPULMONARY CHANGES ASSOCIATED WITH CHEMICAL IMMobilIZATION AND RECUMBENCY IN A WHITE RHINOCEROS (CERATOTHERIUM SIMUM)

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Abstract: A 28-yr-old female white rhinoceros (Ceratotherium simum) was immobilized with etorphine twice for reproductive examination and manipulation prior to euthanasia for bilateral blindness. Prolonged etorphine immobilization and recumbency were associated with hypoxemia, hypercapnia, and apparent hypertension. Although the hypoxemia was probably due to hypoventilation, left lung atelectasis after prolonged lateral recumbency suggests that pulmonary shunting and ventilation/perfusion mismatching may also contribute to hypoxemia in the recumbent rhinoceros.

Key words: Etorphine, white rhinoceros, Ceratotherium simum, anesthesia, hypoxemia, hypertension.

INTRODUCTION

As a large perissodactylid, the immobilized and recumbent white rhinoceros (Ceratotherium simum) would be expected to experience the same adverse effects of recumbency and anesthesia observed in the horse,17 including hypoventilation, pulmonary shunting, and progressive lung atelectasis, leading to associated hypoxemia and hypercapnia. The use of etorphine and carfentanil for immobilization of free-ranging and captive white and black (Diceros bicornis) rhinoceroses has been well described.1,3,5-11,18 Although several studies have reported heart and respiration rates,8-12,18 arterial blood pressures12,13 and arterial blood gas values12 are rarely mentioned. This case report contains observations made during two immobilizations of a white rhinoceros that have implications for research and management of anesthesia in other rhinoceroses.

CASE REPORT

A 28-yr-old female white rhinoceros (~1,500 kg) was chemically immobilized on two consecutive days for reproductive examination and manipulation prior to euthanasia for bilateral blindness.

First immobilization

The rhinoceros was confined in a metal chute, and 1.5 mg etorphine HCl (M99, Lemmon Co., Sellersville, Pennsylvania 18960, USA) was injected into the semitendinosus muscle using a dart pistol and projectile syringe. An additional 0.5 mg etorphine administered i.m. at 17 min provided good standing restraint until 55 min, when 0.25 mg etorphine injected i.v. caused the animal to fall into sternal recumbency. This last dose of 0.25 mg i.v. was repeated at 78 and 165 min. At 95 min, 1 L of 5% guaifenesin (Guailaxin, A. H. Robins Co., Animal Health Group, Richmond, Virginia 23220, USA) was administered i.v. without apparent effect. At 213 min after the initial etorphine injection, immobilization was reversed with 3.0 mg diprenorphine i.v. (M50-50, Lemmon Co.). After 7 min, the rhinoceros was alert and sternal and was standing by 12 min.

Beginning at 50 min after initial etorphine injection, direct systolic, mean, and diastolic blood pressures were measured every 5 min using an 18-gauge over-the-needle catheter (Angiocath, Deseret Medical, Sandy, Utah 84070, USA) placed in an auricular artery and connected by a heparin-
blood pressures ranged from 148 to 212, 107 to 168, and 62 to 82 mmHg, respectively (Fig. 1). Indirect blood pressure measurements at 175 (170 vs. 162 mmHg direct) and 190 min (170 vs. 171 mmHg direct) were obtained from the coccygeal artery with an ultrasonic flow detector (Ultrasonic Doppler Flow Detector, Parks Medical Electronics, Aloha, Oregon 97007, USA) and blood pressure cuff using the technique described for the horse.\textsuperscript{15} Heart rate, determined either from direct cardiac auscultation or from the pressure tracing, and respiration rate, determined from direct observation of thoracic movements, were measured every 5 min over 30-sec intervals. Heart and respiration rates decreased slowly over the duration of the procedure and varied from 60 to 88 bpm and from 2 to 6 breaths/min, respectively (Fig. 1). Heparinized arterial blood samples for blood gas analysis were taken from the auricular catheter every 15 min, placed in glass syringes into crushed ice, and analyzed within 4 hr. The blood gas values were corrected to the body temperature of the animal. The arterial carbon dioxide partial pressure (Paco$_2$), which rose from a low of 43 mmHg at 50 min to a peak of 69 at 165 min, had declined to 53 by the end of the procedure (Fig. 1). The arterial oxygen partial pressure (Pao$_2$) of 83 mmHg in the standing rhinoceros at 50 min rapidly decreased to 61 mmHg at 65 min in the sternally recumbent animal (Fig. 1). Inspired oxygen supplementation (66 L/min) at the level of the left nostril between 90 and 195 min elevated Pao$_2$ to a peak of 139 mmHg at 135 min, with a subsequent decline to 61 mmHg at 165 min. (Fig. 1).

Second immobilization

The following day, the rhinoceros was immobilized in its stall with 1.5 mg etorphine i.m. in the shoulder using a dart gun and projectile syringe. The animal was ataxic and head pressing at 7 min and, following a further injection of 0.25 mg etorphine i.v., was pulled into left lateral recumbency at
35 min using ropes. The rhinoceros was maintained in lateral recumbency for 3 hr using intermittent administration of etorphine (0.25–0.5 mg i.v.) every 30–45 min. Toward the end of the procedure, additional doses of etorphine exacerbated muscle trembling and central nervous system excitement. Xylazine (Rompun, Mobay Corp., Animal Health Division, Shawnee, Kansas 66201, USA) (150 mg and 100 mg i.v. at 70 and 90 min, respectively) appeared to enhance muscle relaxation, whereas 1 L of 5% guaifenesin at 120 min had little effect.

Several attempts, including blind, were made to intubate the animal with an endotracheal tube (26 mm internal diameter). Although the epiglottis was palpated, oral intubation was inhibited by poor muscle relaxation and the inability to extend the head and neck of the animal. Nasal intubation was made difficult by the acute angle between the nares and the ventral nasal meatus. Even when the well-lubricated tube was passed beyond this point, it was not able to pass the nasopharynx because of the angle of the head and neck.

Necropsy

At the end of the second immobilization, the rhinoceros was euthanized and a necropsy performed. When the thorax was examined, the left lung (the “down” lung) was atelectatic, and the right lung appeared normal. The anatomy of the respiratory tract is similar to that of the horse:4 the epiglottis is dorsal to the soft palate, the larynx contains lateral ventricles, and there is an absence of external evidence of lung lobulation, except for the accessory lobe and bilateral cardiac notches. The larynx of this animal was large enough to accommodate a 26-mm endotracheal tube.

DISCUSSION

Prolonged chemical immobilization and recumbency were associated with hypoxemia and hypercapnia in this adult white rhinoceros. Similar effects have been reported during etorphine immobilization in another white and an Indian (Rhinoceros bicornis) rhinoceros.12 The observed hypoxemia was probably due to hypoventilation, as indicated by the hypercapnia immediately after the animal fell to sternal recumbency. Although ventilation/perfusion mismatching and shunting in this rhinoceros could not be evaluated without providing ventilation, the left lung atelectasis after prolonged lateral recumbency suggests their possible contribution to the observed hypoxemia.14,17 Hypoxemia associated with increased pulmonary shunting is a well-described complication of anesthesia and recumbency in the horse.17

Although the Pao2 values observed in this animal were probably adequate for tissue oxygenation because of the higher oxygen affinity hemoglobin (lower PaO2) and lower tissue metabolic rate of large mammals,16 hypoxemia may be one cause of perianesthetic morbidity/mortality in the rhinoceros. Hypoxemia, compounded by maldistribution of blood flow caused by etorphine-induced peripheral vasoconstriction (D. J. Heard, unpubl. data), may contribute to localized or generalized myositis,17 renal failure, abortion, or cardiac failure and death. The observed hypercapnia was due either to hypoventilation associated with decreased ventilatory drive caused by the etorphine and/or the pressure of viscera pushing forward on the diaphragm.17

Suspected hypertension associated with etorphine use in white rhinoceroses has been described previously.12,13 Etorphine also produces hypertension in the horse2 and several species of ruminants.7 The similar pressures reported here, although hypertensive relative to the domestic horse,13 are difficult to interpret because of the absence of normal values from awake standing rhinoceroses. Other possible causes of suspected hypertension in this rhinoceros include the observed hypercapnia and hypoxemia and pain associated with the reproductive manipulations. The indirect blood pressure measurements from the coccygeal artery closely approximated those measured di-
rectly, suggesting that this indirect method may be a useful technique for systolic blood pressure determination in the rhinoceroses.

Etorphine doses and behavioral responses were similar to those reported in other white rhinoceroses, except cumulative doses of etorphine resulted in increased muscle movement and central excitement. Although this paradoxical response could have been the result of sequential immobilizations, it may still confuse assessment of anesthetic depth when using etorphine for prolonged immobilization. The addition of xylazine enhances induction and maintenance of etorphine immobilization in the black rhinoceros. Although xylazine also improved muscle relaxation in the white rhinoceroses in this report, further studies are needed to assess the potential adverse cardiopulmonary effects of this combination in these animals.

The use of inhalation anesthesia in adult rhinoceroses has not been described but may be a useful alternative to the repeated administration of etorphine, with its associated hypertension and poor muscle relaxation, for prolonged procedures. Although nasal intubation was not successful in this animal, oral intubation by direct palpation appears feasible. Intubation would allow delivery of a high inspired oxygen concentration, partial protection of the airway from oral secretions and regurgitated material, and ventilation of the animal if hypoventilation is severe.

**LITERATURE CITED**


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