MULTIPLE ISOFLURANE ANESTHESIA IN A CAPTIVE BLACK RHINOCEROS (*Diceros bicornis*)

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Abstract

Inhalant anesthesia in rhinoceros has been reported on a couple of occasions. In all instances reported, euthanasia was performed[1] or the rhino expired in the perianesthetic period from surgical complications[2]. This report describes the successful maintenance and recovery of a black rhinoceros anesthetized twice for diagnostics evaluations of a nasal fracture and obstructive respiratory disease. A 4-yr-old male black rhinoceros weighing an estimated at 1000 kg was anesthetized twice and maintained on isoflurane and ventilated on both occasions for diagnostics evaluation of a fractured maxillary sinus. The animal was considered a high risk for anesthesia due to the known respiratory compromise from the fractured maxilla and obvious respiratory stridor and the possibility of pulmonary disease secondary to the trauma and resultant infection. On the first occasion, the rhino was premedicated with oral halperidol. This was followed by 40 mg detomidine and 20 mg butorphanol i.m. Induction was accomplished with 2 mg etorphine i.m. Standing immobilization occurred at this point and the animal was safe to approach. An additional 0.25 mg etorphine i.v. and 300 mg xylazine i.v. was utilized to facilitate sternal recumbency and intubation. A 28-mm endotracheal tube was placed with the aid of a 1.7-m colonoscope. Antagonism consisted of 125 mg of naltrexone given simultaneously i.v. and i.m. The rhino stood in 1 min but was slightly sedate for an additional 20 min.

Seventy-five days later the rhinoceros was again premedicated with 40 mg detomidine and 20 mg butorphanol i.m. followed by 2 mg etorphine i.m. Standing immobilization was again achieved and 1000 mg ketamine i.v. was used to induce recumbency and intubation. This time the rhinoceros went down in lateral recumbency. The respiratory rate dropped to 3/min before it was moved into sternal recumbency and intubated. Naltrexone was again given, 100 mg i.v. and i.m. plus 4000 mg of tolazaline i.m. and the rhinoceros stood within 5 min but was sedate for an additional 40 min.

On both anesthetic episodes respirations were assisted at 8-10 per minute with a 20-L bellow large animal ventilator. Isoflurane was kept between 2-3 % based on heart rate and rate and depth of spontaneous efforts to breathe and response to surgical or other procedural stimulation. Serial blood gas taken from an auricular artery, non-invasive blood pressure monitoring with the cuff on the base of the tail, oxygen saturation via pulse oximetry, and base apex electrocardiogram (ECG) monitoring were followed during the procedure when feasible. The rhino was maintained in sternal recumbency for the entire procedure. The elapsed time of isoflurane anesthesia for the first anesthesia was 172 min and 230 min for the second procedure. Recoveries were smooth and uncomplicated with the

animal exhibiting normal behaviors within 2 hr of each event.

For the first episode, body temperature was maintained at 100.9° F (38.3° C) for the duration of monitoring, heart rate averaged 44/min (range: 36-58), oxygen saturation averaged 92% (range: 100-84%). Blood pressures averaged 130/99/114 mm Hg and deviated very little during the procedure. Initial arterial blood gas taken prior to intubation revealed relative hypoxemia (PaO_2, 96 mm Hg) and mild hypercapnia ($PaCO_2$, 57.9 mm Hg). Once intubated and provided with ventilator support, both values essentially resolved with PaO_2 at 218 mm Hg and $PaCO_2$ at 44.8mg Hg. They remained near these levels for the duration of monitoring. Electrocardiogram tracing revealed a normal sinus rhythm.

For the second episode, body temperature was again maintained at 100.9° F (38.3° C) for the duration of monitoring. During the first 30 min following ketamine and while in lateral recumbency and not yet intubated, heart rates where elevated (average 57/bpm, range 49-76), respirations decreased to 3-4/min, oxygen saturations decreased (mean 83%, range 80-86%) while blood pressures where elevated (average 190/143/166). Compared to the first episode, initial arterial blood gas sampling obtained prior to intubation revealed hypoxemia (PaO_2, 72 mm Hg) and hypercapnia ($PaCO_2$, 44.2 mm Hg). Further sampling revealed an increase of PaO_2 (average 203 mm Hg, range 167-259) but the hypercapnia persisted and even worsened ($PaCO_2$, average 56 mm Hg, range 34.1-68.5). After this initial 30 min elapsed, heart rate returned to what was deemed a more acceptable level (average 43/bpm, range 35-52), blood pressures stabilized at an average of 136/79/104 mm Hg. Electrocardiogram tracing again revealed a normal sinus rhythm.

Several elements were key to the success of these anesthetic episodes. The first is that the rhino was tractable to the point of allowing the premedications to be given by hand. In fact, this animal had received 1 wk's worth of i.v. antibiotics prior to the second episode due to septicemia. The halperidol did not seem to affect the ability to allow this hand injection nor did it appear to enhance the overall quality of anesthesia so it was not included in the second procedure. It was essential to maintain the rhino in sternal recumbency to prevent a ventilation/perfusion mismatch (V/Q) on top of any pre-existing pulmonary compromise. The fact that the rhinoceros was in lateral recumbency combined with the intravenous ketamine may have combined to produce the prolonged hypercapnia seen in the second event. The V/Q mismatch may have been from the recumbency and produced an area of lung consolidation on the dependent lung that was not thoroughly expanded even with assisted ventilation. Venous admixture associated with lateral recumbency is well documented in horses and suspected in a white rhinoceros under isoflurane anesthesia[1]. Ketamine was chosen in the second instance based on personal preference and in an effort to avoid additional narcotics. In this case it may have been better to repeat the supplemental dosing as was done in the first episode based on the initial parameters collected following ketamine. Depth of anesthesia was judged primarily by depth and rate of spontaneous respirations and heart rate. Position of the eye was not useful in determining depth of anesthesia. Recoveries were quick and extremely smooth in both cases and in fact surprisingly fast in the first case. At no time during either event was there any muscle rigidity or hyperexcitability noted so common with ultra potent narcotics.

LITERATURE CITED

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