Rectal prolapse associated with a healed pelvic fracture in a pregnant free-ranging African black rhinoceros (*Diceros bicornis*). Part 1: anaesthesia

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ABSTRACT

Anaesthesia was required in a heavily-pregnant, adult, free-ranging African black rhinoceros *Diceros bicornis* with a rectal prolapse for examination and possible treatment. The animal was immobilised with 4.5 mg etorphine and 60 mg azaperone. For continued observation, the immobilised animal was transported to a boma. Additional etorphine and azaperone were administered to keep the animal anaesthetised during treatment and transport. In addition, 15 mg nalorphine was administered during this time to improve ventilation and reduce muscle rigidity. Sixty hours later, in preparation for surgery, 2.5 mg etorphine and 40 mg azaperone were administered, followed by endotracheal intubation and halothane anaesthesia. During anaesthesia, a decrease in tidal volume was observed. Venous blood-gas analysis indicated a decrease in the oxygen partial pressure, and a mixed respiratory and metabolic acidosis. Cardiac arrest was preceded by an increase in heart rate and tidal volume after 80 min of inhalation anaesthesia.

Key words: anaesthesia, black rhinoceros, *Diceros bicornis,* etorphine, halothane, midazolam, pelvic fracture, rectal prolapse.

Stegmann G F, Hofmeyr M, Olivier A, Lane E, Volkman D H **Rectal prolapse associated with a healed pelvic fracture in a pregnant free-ranging African black rhinoceros (*Diceros bicornis*). Part 1: anaesthesia.** *Journal of the South African Veterinary Association* (2001) 72(4): 239–241 (En.). Department of Companion Animal Clinical Studies, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, Onderstepoort, 0110 South Africa.

INTRODUCTION

A heavily pregnant, adult, free-ranging African black rhinoceros (*Diceros bicornis*) with a suspected rectal or vaginal prolapse was seen in the Pilanesberg National Park, North West Province, South Africa. The age and body mass of the animal were estimated to be 6 years and 900 kg respectively. Apart from the prolapse, she appeared healthy. Her 2- to 3-year-old female calf accompanied her. A puncture wound in the escutcheon had been observed some months previously.

Anaesthesia resulting in the death of the rhinoceros is described in this part of the case report. Surgery undertaken to correct a rectal prolapse and necropsy findings are described in Part 2[9].

ANAESTHESIA

The rhinoceros was initially immobilised using a dart gun and projectile syringe (Palmer CapChur) from a helicopter for clinical examination and possible medical treatment. A combination of 4.5 mg etorphine (M99, Logos Agvet), 60 mg of azaperone (Stresnil, Janssen) and 2500 IU of hyaloronidase (Kyron Laboratories) were used. The drugs were administered in the gluteus muscle group, and she became immobilised in sternal recumbency after 4.5 min. Eight minutes after she had been darted, 10 mg nalorphine hydrobromide (Nalorphine Injection, Kyron Laboratories) was administered intramuscularly to improve ventilation and reduce muscle rigidity (induced by etorphine), followed by an additional 5 mg nalorphine, 30 min later. The initial examination revealed a rectal prolapse, and treatment was attempted.

For further observation and continued treatment, the immobilised rhinoceros was transported on a trailer to a boma some 10 km away. Fifty minutes after immobilisation, an increase in ventilation rate and a decrease in the depth of ventilation were observed, and additional intravenous etorphine (2 mg) and azaperone (50 mg) were administered. During transport the animal was kept in lateral recumbency, during which the anaesthetic plane lightened to the point of being responsive to noise stimulation and displaying occasional involuntary movement. At the boma, an additional 1.5 mg etorphine had to be administered intramuscularly to off-load her and to continue treatment for an additional 20 min. The total anaesthetic time was 130 min. Anaesthesia was reversed by intravenous administration of 24 mg diprenorphine hydrochloride (M5050, Logos Agvet). Within a minute, depth of ventilation increased and movement of the ears was observed, and she was able to stand after 4 min. Recovery was uneventful. By the following day, her rectum had prolapsed again.

To allow for possible residual effects from the diprenorphine, treatment under anaesthesia was delayed for a period of 60 h following the first immobilisation. The rhinoceros was darted with a reduced dose of 1 mg etorphine and 40 mg azaperone in an attempt to obtain standing immobilisation. However, an additional 1.5 mg etorphine was required to immobilise the rhinoceros in the standing position for the rectal examination.

For inhalation anaesthesia, the rhinoceros was pulled into sternal recumbency with the aid of ropes tied around her legs. For endotracheal intubation, a gag was placed between the upper and lower molars on the right-hand side. The epiglottis was identified by internal palpation of the oropharynx. As the swallowing and laryngeal reflexes were present, it delayed the placement of the endotracheal tube. A 25 mm diameter stomach tube was introduced into the trachea for a distance of at least 50 cm. The anaesthetist's arm was then retracted from the oral cavity. The free end of the stomach tube was fed into a cuffed, 30 mm silicon endotracheal tube, and the latter was advanced forward until the trachea was intubated, with the stomach tube acting as a guide. The stomach tube was then removed. For maintenance of anaesthesia, the endotracheal tube was connected to a circle anaesthetic machine equipped with a 30-ℓ reservoir bag, carbon dioxide absorption, and an out-of-circle Fluotec Mk II precision vaporiser (Cyprane). Two to three per cent halothane-in-oxygen (Fluothane, Zeneca)

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Received: April 2001. Accepted: September 2001.

was delivered to the breathing circuit at a flow rate of 5 ℓ/min for the first 20 min, and thereafter reduced to 3 ℓ/min during maintenance of anaesthesia. Ten milligram midazolam (Dormicum, Roche) and 0.25 mg etorphine were administered twice intravenously, at 30-min intervals to maintain anaesthesia and reduce muscle rigidity. Percutaneous venous puncture of a superficial ear vein on the lateral surface was performed with a 18 G teflon catheter (Jelco, Johnson & Johnson) for the administration of a balanced electrolyte solution (Plasmavet, Sabax). A total of 6 ℓ was administered during surgery that lasted 75 min.

After approximately 75 min of inhalation anaesthesia, heart rate and depth of ventilation increased. Five mg nalorphine was injected intravenously, but no change in heart rate occurred. As no apparent reason for the increase in heart rate and tidal volume were evident, the changes in the variables were regarded as possible signs of an impending cardiovascular collapse. Five minutes later, signs of apparent partial recovery from anaesthesia occurred as evidenced by involuntary movement of the head and neck. The delivered halothane concentration was increased to 6 %, the fresh gas flow rate increased to 5 ℓ/min, and an additional 0.25 mg etorphine administered intravenously. Within 3 min, respiratory arrest occurred with a rapid decrease in the audible heart sounds. The antidote, 12 mg diprenorphine was injected intravenously, and the rhinoceros was turned into left lateral recumbency. External chest compression over the heart was initiated and adrenaline administered intravenously, but the attempted resuscitation failed to restore cardiac function.

MONITORING DURING SURGERY

Before immobilisation, the habitus of the animal appeared normal. After immobilisation, and during inhalation anaesthesia, heart rate was evaluated with a stethoscope. Depth of ventilation was subjectively estimated from reservoir bag movement during inspiration. During sternal recumbency, and before halothane anaesthesia the ventilation rate was 10 breaths/min. During halothane-etorphine anaesthesia the heart rate varied between 56 and 68 (mean 64 ± 6 SD) beats/min. After 75 min inhalation anaesthesia, the heart rate increased to 96 beats/min. Ventilation rate varied between 8 and 12 breaths/min during etorphine-immobilisation and inhalation anaesthesia. The tidal volume was estimated to be between 3 and 4 ℓ during anaesthesia, but appeared to decrease over time during inhalation anaesthesia. The first administration of midazolam resulted in a transient decrease in ventilation rate to 2 breaths/min.

The estimated blood loss during surgery was 2–3 ℓ. Heparinised venous blood was anaerobically collected in 3 mℓ plastic syringes from an ear vein and stored in iced water until blood-gas analysis in a calibrated blood-gas analyser (ABL 300, Radiometer), approximately 4 h after collection (Table 1). Samples were collected at the start of surgery, 30 min later and before the end of surgery, and reported in Table 1. A mixed respiratory and metabolic acidosis was present from the 1st sample that tended to worsen until the 3rd (last) sample was taken. The partial pressure of oxygen (PO_2) decreased and the partial pressure of carbon dioxide (PCO_2) increased during inhalation anaesthesia. Routine haematological and blood lactate examination were performed 9 h after sample collection. Blood samples for lactate were stored in iced water, and haematological samples at 4 °C until analysis. Haematological examination revealed a haemoglobin concentration of 14.1 g/ℓ, red cell count 4.89 × 10^{12}/ℓ, haematocrit 0.348 ℓ/ℓ, and a white cell count of 7.2 × 10^9/ℓ. Plasma concentrations of Na^+, K^+ and blood lactate were respectively 167, 5.22 and 1.6 mmol/ℓ.

Table 1: **Venous blood-gas analysis from an African black rhinoceros (*Diceros bicornis*) during etorphine-halothane anaesthesia.**

Variable	Sample 1[a]	Sample 2[b]	Sample 3[c]
pH (units)	7.3	7.2	7.2
PCO_2 (kPa)	6.8	8.2	9.3
PO_2 (kPa)	25.6	26.5	15.5
HCO_3 (mmol/ℓ)	22.6	24.3	23.6
ABE (mmol/ℓ)	–4.4	–3.6	–5.8
SBE (mmol/ℓ)	–3.7	–2.6	–4.2
SAT (%)	99.3	99.3	96.3

[a]Beginning of inhalation anaesthesia.
[b]After 30 min of inhalation anaesthesia.
[c]After 60 min of inhalation anaesthesia.
PCO_2 = partial pressure of carbon dioxide; PO_2 = partial pressure of oxygen; HCO_3 = bicarbonate concentration; ABE = actual base excess; SBE = standard base excess; SAT = oxyhaemoglobin saturation.

DISCUSSION

Chemical immobilisation of the black rhinoceros has been described previously by numerous authors[4,6,7,10]. Isoflurane anaesthesia in an Indian rhinoceros has been described[11]. An essential component of the medical treatment of free-ranging wild animals is their capture and translocation to a suitable holding pen or boma, and possible repeated exposure to immobilising drugs. As published data on the clinical effects of the repeated administration of immobilising drugs and the antidote in rhinoceros are not available, it was suggested that repeated exposure to these drugs may increase the risk of morbidity or mortality. The 2nd exposure to immobilising drugs was therefore delayed by 60 h. A total dose of 8 mg etorphine, 15 mg nalorphine, 110 mg azaperone were administered on the 1st day of treatment. For immobilisation on the 3rd day, the etorphine dose was reduced from the recommended dose for adult free-ranging rhinoceros[7] of 4.5 mg (used during the first immobilisation) to 1 mg. The dose was reduced to allow for previous drug exposure, stress of capture, and an attempt to keep the animal standing. The subsequent failure to immobilise the rhinoceros was probably the result of the low etorphine dose and possible subcutaneous injection of the drugs. An additional 1.5 mg of etorphine was therefore administered, bringing the total dose for etorphine to 2.5 mg.

Intubation in sternal recumbency was complicated by the limited vertical movement of the mandible. In addition, the weight of the head and neck, the long distance from the mouth opening to the larynx, high muscle rigidity, and partial suppression of the swallowing and laryngeal reflexes contributed to the difficulty experienced during intubation. The use of a gag, and an assistant lifting the head and neck, facilitated internal palpation of the pharynx, and the introduction of the stomach tube into the larynx and trachea.

Black rhinoceros have been reported to recover from etorphine-immobilisation without preceding signs of decreased anaesthetic plane[10]. To prevent possible injury to the surgical team in the event of an unexpected recovery, etorphine was not reversed during halothane anaesthesia despite the possible increase in ventilatory depression from the combined use of the anaesthetic agents. Both etorphine[1] and halothane[12] may depress ventilation in animals. Additional etorphine was administered after 30 min of anaesthesia, based on previous experience with etorphine-halothane anaesthesia in the African elephant[13] where partial recovery occurred after 60 min of halothane anaes-

thesia. The potent analgesic effect of etorphine may contribute towards a reduction in the inspired halothane concentration required to maintain surgical anaesthesia. In contrast to our previous experience with the African elephant during etorphine-halothane anaesthesia where 1–2 % halothane was required for maintenance of anaesthesia during dental surgery[15], no anaesthetic-sparing effect was observed, as the vaporiser setting remained at 2–3 % during maintenance. Muscle relaxation was improved after the administration of midazolam during anaesthesia. Midazolam causes a transient ventilatory depression in domestic goats[14]. The ventilation rate observed in the rhinoceros (10 breaths/min) is in agreement with previously reported rates[4]. It is suggested that the ventilatory depression imposed by etorphine is partially countered by the hypoxic ventilatory drive, thus increasing minute ventilation. Hypoxia is a common finding during etorphine immobilisation in large mammals[3]. Inhalation of high oxygen concentrations results in increased partial pressure of oxygen in comparison to the pressures when breathing air, resulting in a decrease in the hypoxic ventilatory drive and decreased minute ventilation.

It is recognised that prolonged storage of blood in plastic syringes may detract from the validity of the results from the blood-gas analysis, and a decrease in the O_2 and CO_2 tensions may result from diffusion from the plastic syringe. Metabolic changes may result in increases in the CO_2 and a decrease in the O_2 tension and blood lactate. The values obtained from the first blood sample were within the range of expected values, especially for the O_2 and CO_2 tension. The changes that took place over time should be regarded as of value in the search for factors that could have contributed towards the mortality.

In comparison to values obtained from venous blood in a horse during halothane anaesthesia[13], ventilatory depression from the halothane-etorphine combination was not excessive. The increase in the CO_2 tension (37 %) indicated an increase in ventilatory depression in time that was confirmed by subjective observation of the reservoir bag. However, the CO_2 tension was higher compared to partial pressures reported for etorphine-acepromazine-immobilised horses breathing air (7.3 kPa)[3]. The decrease in the PO_2 from 26.5 to 15.5 kPa as was observed in the rhinoceros may have been an indication of deterioration in pulmonary function. Ventilation-perfusion mismatch and pulmonary shunts are common findings during inhalation anaesthesia in horses[2]. The O_2 tension for mixed venous blood reported for horses during halothane anaesthesia (6.4 kPa)[13], and etorphine-acepromazine anaesthesia[3] (4 kPa) were considerably lower than the minimum value (15.5 kPa) observed in this rhinoceros. The inhalation of high oxygen concentrations minimises the effects of ventilation/perfusion mismatch, and therefore hypoxia was likely the result of pulmonary shunting. Atelectasis is commonly associated with pulmonary shunting during inhalation anaesthesia[8], but was not observed at necropsy in this rhinoceros (Part II). Although the animal was kept in sternal recumbency during anaesthesia, restrictive effects from the foetus and abdominal organs on diaphragmatic function should be considered as contributory to hypoventilation.

Haematological values were somewhat below reported values for black rhinoceros[5]. Analysis of plasma electrolytes indicated Na^+ concentrations above reported values[5], but this probably resulted from the addition of an anticoagulant containing sodium (Heparin sodium, Intramed) to the blood samples. The plasma K^+ concentration was increased in comparison to the reported value in black rhinoceros[5], and probably the result of the severe acidosis. A mixed respiratory- and metabolic acidosis resulted in a decrease in the pH to 7.16 and the standard base excess to 4.2 mmol/ℓ. In comparison, the pH for mixed venous blood only decreased from 7.35 to 7.32 units in etorphine-acepromazine anaesthetised horses[3]. Acidosis may adversely affect cardiac contractility[16] if the arterial pH decreases below 7.2. The reported mixed venous pH for horses[13] during halothane anaesthesia decreased to 7.26, which is considerably better than the value of 7.16 observed in this rhinoceros. Although arterial blood pressure was not monitored, observations from the surgical site did not indicate hypotension or hypovolaemia during surgery, and an intraoperative blood loss of approximately 3 ℓ may be considered minimal in an animal with a body weight of 900 kg.

In conclusion, the normal homeostatic mechanisms in the rhinoceros were probably compromised by a combination of factors such as the advanced stage of pregnancy, stress associated with the prolapsed rectum, capture, previous exposure to immobilising drugs, anaesthesia and surgery, and severe acidosis. It appeared that the administration of halothane was associated with deterioration of pulmonary function. The suggested cause of death in this rhinoceros was cardiac arrest after a relative anaesthetic overdose.

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