Journal of Zoo and Wildlife Medicine 38(3): 380–387, 2007

THE CARDIOPULMONARY EFFECTS OF ETORPHINE, AZAPERONE, DETOMIDINE, AND BUTORPHANOL IN FIELD-ANESTHETIZED WHITE RHINOCEROSES (*CERATOTHERIUM SIMUM*)

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Abstract: White rhinoceroses (*Ceratotherium simum*) anesthetized with etorphine combinations develop severe pathophysiologic changes, including hypoventilation, hypoxemia and metabolic acidosis. The aim of this study was to evaluate the addition of butorphanol to the immobilizing mixture on the cardiopulmonary effects in free-ranging white rhinoceroses darted from the helicopter. In the control group ($n = 15$), the rhinoceroses were anesthetized with etorphine, azaperone, detomidine, and hyaluronidase administered intramuscularly. In the treatment group ($n = 16$), 10–20 mg of butorphanol was added to the combination. Within 10 min of becoming immobile, vital parameters (heart rate, respiratory rate, and temperature) and blood gas analyses were taken, and measurements were repeated after 10 (treatment group) and 20 min (control group). Both groups showed respiratory and metabolic acidosis, hypoxemia, and hypercapnia. In the control group, the arterial partial pressure of oxygen was significantly higher and the alveolar-to-arterial oxygen pressure gradients were significantly lower in all body positions compared with the butorphanol group. Oxygen hemoglobin saturation in the control group was higher than in the butorphanol group only in the lateral position. Improvements in arterial oxygen levels were observed in all animals when placed in sternal recumbency. There were no significant differences in the mean induction times between groups, but the distance the butorphanol group ran was significantly less after darting than in the control group. By adding butorphanol to the immobilizing mixture, no benefits in ventilation were seen; although, size differences make comparisons difficult. Running for a shorter distance during induction could be beneficial in the prevention of severe acid-base imbalances and capture myopathy.

Key words: *Ceratotherium simum*, rhinoceros, anesthesia, hypoxemia, butorphanol, etorphine.

INTRODUCTION

Safe and reliable anesthesia of white rhinoceroses (*Ceratotherium simum*) is an important tool for conservation-based programs of free-ranging animals. White rhinoceroses are routinely anesthetized for marking, sample collection, translocation, and treatment, such as the removal of snares.[22] Many drug combinations have been used successfully for the immobilization of white rhinoceroses. Commonly used opioids include etorphine, fentanyl, and carfentanil.[9,15,17,19,28,33] To improve muscle relaxation, reduce hyperexcitability, and shorten induction times, supplemental drugs such as α_2-adrenoreceptor agonists, butyrophenones, and hyaluronidase are often added to the opioid components.[15,22,26]

Respiratory depression with hypoxemia and hypercapnia regularly develop in rhinoceroses immobilized with protocols using potent opioids such as etorphine.[2,10,18] This depression is dose-dependent, and it may be compounded by the rigidity of the thoracic musculature. Anesthesia of free-ranging animals usually results in more marked respiratory depression than in captive animals, because higher doses of etorphine are used to shorten induction times.[28] Many reports recommend oxygen supplementation, partial reversal of anesthesia with nalorphine, and respiratory stimulants to correct hypoxemia.[2,28]

Butorphanol tartrate is a synthetic opioid that acts as a κ-receptor agonist and μ-receptor antagonist.[3] One of its major advantages as an anesthetic agent is its minimal respiratory and cardiovascular side effects.[25] It has been used in captive white rhinoceroses in combination with either azaperone or detomidine, etorphine, and acepromazine.[29,35]

The deleterious side effects of μ-receptor activation, such as respiratory depression, can be completely reversed by competitive inhibition, using pure opioid antagonists such as naloxone. However, by using pure opioid antagonists, analgesia and se-

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dation also will be reversed.[34] In an attempt to selectively antagonize the undesirable side effects of pure opioid agonists while preserving their potent analgesic effects, opioid agonist-antagonists, such as butorphanol, have been used with varying success in humans and animals.[12,13,21] The aim of this study was to evaluate the addition of butorphanol to the immobilizing mixture on selected cardiopulmonary and behavioral effects in free-ranging white rhinoceroses.

MATERIALS AND METHODS

The study animals were 31 white rhinoceroses captured during conservation-based programs in Malilangwe Wildlife Reserve, Zimbabwe (control group, $n = 15$) and game capture operations in Kruger National Park, South Africa (butorphanol group, $n = 16$). The control animals were anesthetized in May and June 2005 with ambient temperatures ranging from 20 to 30°C and barometric pressure from 722 to 730 mm Hg. In the butorphanol group, the rhinoceroses were captured in June and July 2006, ambient temperatures were below 20°C, and barometric pressure ranged from 735 to 742 mm Hg. The animals were divided into three age groups: calves (birth until 2.5 yr), subadults (2.5–7 yr), and adults (older than 7 yr). The weight ranged up to 1,000 kg in calves, between 1,200 and 1,800 kg in subadults, and more than 1,800 kg in adults. In the control group, there were 10 males and five females. Twelve animals were classified as calves and three as subadults. In the butorphanol group, there were eight males and eight females. Of these, two animals were calves, nine were subadults, and five were adults.

The animals were located using a combination of ground tracking, fixed wing aircraft, and helicopter. The size of the animal was assessed from the air, and the rhinoceroses were darted from the helicopter in the dorsal thigh/rump area with a mixture of etorphine (M99®, Novartis, Kempton Park, South Africa), azaperone (Stressnil®, Janssen Pharmaceutical Ltd., Halfway House, South Africa), detomidine (Domosedan®, Novartis), and hyaluronidase (Hyalase, Kyron Laboratories, Benrose, South Africa). In Zimbabwe, calves received 1–2 mg of etorphine, 20–25 mg of azaperone, 1–2 mg of detomidine, and 2,500 IU of hyaluronidase. Subadults received 2.5–4 mg of etorphine, 40–80 mg of azaperone, 2.5–4 mg of detomidine, and 5,000 IU of hyaluronidase. The mixture was placed in plastic air-pressurized darts (Palmer Cap-Chur® syringe, Palmer Cap-Chur Equipment, Powder Springs, Georgia 30127, USA) with 45–60-mm collared needles attached firmly to the projectile syringe and delivered with a powder charge projector (PneuDart® projector, PneuDart Inc., Williamsport, Pennsylvania 17701, USA).

In South Africa, calves received 1.5–2 mg of etorphine, 20 mg of azaperone, 1–2 mg of detomidine, and 1,000 IU hyaluronidase. Subadults received to 3–4 mg of etorphine, 35–40 mg of azaperone, 2.5–4 mg of detomidine, and 2,500 IU of hyaluronidase. Adults received to 4–5 mg of etorphine, 40 mg of azaperone, 4.5–5 mg detomidine, and 2,500 IU of hyaluronidase. Butorphanol, 10 mg for calves and 20 mg for subadults and adults (Torbugesic®, Fort Dodge Animal Health, Fort Dodge, Iowa 50501, USA), was added to the immobilization mixture. The delivery system was 4.5 ml aluminium darts with 45–60 mm collared needles using a sodium bicarbonate/acetic acid injection system propelled by a modified shotgun (20-gauge Miroku O/U) (Rhorr, pers. comm.).

If no signs of induction, such as stumbling or increasing incoordination, were observed within 10 to 15 min, the animal was reevaluated from the helicopter and redarted if appropriate. The induction time was noted for every rhinoceros, and it refers to the time from dart impact to when the animal became immobile, including both recumbent and standing sedation. The distance the animal ran before and after darting was estimated using the global positioning system of the helicopter.

When an animal was recumbent, or it was judged ready for roping, it was approached cautiously from behind and blindfolded. Two ropes were attached, one rope around the rear horn and lower jaw, and the other rope around one of the hind legs. After the initial examination, the animals were placed into sternal (Zimbabwe) or lateral (South Africa) recumbency. Rate of respiration and heart rate were carefully monitored in all animals. Rectal body temperature was recorded, and if necessary, the animals were actively cooled by spraying water on them. Physiologic data and body and horn measurements were obtained; blood samples were collected from an ear vein; and in Zimbabwe, earnotching for marking purposes was performed. The dart wounds were treated with an intramammary antibiotic treatment (Rilexine 200 LC, Virbac RSA, Halfway House, South Africa). A descriptive score ranging from 1 to 6 was used for scoring the immobilization level (Table 1).

In animals with a respiratory rate below 3 to 4 breaths per minute and an oxygen saturation of hemoglobin in the blood (SpO_2) below 80%, the immobilization was partially reversed using 5–10 mg of nalorphine (Nalorphine, 20 mg/ml, Kyron Laboratories), combined with 0.6 to 1.2 mg of dipren-

Table 1. Descriptions used to score the immobilization level of rhinoceroses during the anesthetic period.

Immobilization level score	Description
1	no sedation
2	very light sedation with the animal having to be roped to go down; or showing head or leg movements; or trying to get up
3	light anesthesia with some ear movements and muscle tremors present
4	moderate anesthesia with no or just a few muscle tremors
5	fully relaxed rhinoceros with no muscle tremors
6	an excessive anesthetic level with a respiratory rate below 3 breaths/min

orphine (M5050®, Novartis), depending on the amount of etorphine used, given i.v. into an auricular vein.

In Zimbabwe, anesthesia was reversed using 20–55 mg of naltrexone (Naltrexone, 50 mg/ml i.v., Kyron Laboratories), depending on the amount of etorphine used. In South Africa, once all work was completed on the rhinoceros, it was stimulated to stand and walk into the crate by using an electric cattle prod directed at the perineal area and limbs. If necessary, partial reversal of anesthesia was accomplished by administering 10 mg of nalorphine and 1.2 mg of diprenorphine i.v. Up to 10 people were stationed on the head rope to assist in pulling the animal into the crate. The animals would walk with an unstable gait, and it could be guided by lateral pulling and pushing of the horn. The rope on the hind foot could be used to brake the rhinoceros should it start to run. Diprenorphine, 4.8–14.4 mg, was administered i.v. once the animal was safely loaded into the crate.

In both Zimbabwe and South Africa, blood gas analyses were performed on all animals with blood collected anaerobically into heparinized syringes from the auricular artery located on the inside of the pinna. The first sample was taken within 10 min of the animal becoming immobile (T1), and the second sample was taken 10 min (butorphanol group) or 20 min (control group) later (T2). The blood samples were analysed immediately after withdrawal using a portable blood gas analyzer (i-STAT 1®, Axonlab AG, Baden-Dättwil, Switzerland) and disposable cartridges (i-STAT CG 4^+ cartridges®, Axonlab). Concurrent with each blood sample, heart rate, respiratory rate, temperature, and immobilization level were noted. The blood gas values were corrected for the actual body temperature. Arterial partial pressure of carbon dioxide ($PaCO_2$), arterial partial pressure of oxygen (PaO_2), pH, and lactate were measured directly by the machine, whereas base excess (BE), bicarbonate (HCO_3^-), and arterial hemoglobin oxygen saturation (SaO_2) were calculated.

Statistical analyses were performed using SPSS, version 14.0 (SPSS Inc., Chicago, Illinois 60606, USA). The independent-samples *t*-test was used to compare the differences in the blood gas values, lactate, induction time, immobilization level, and distance the animal ran before and after darting in both treated and control animals. One-way analysis of variance (ANOVA) and post hoc Bonferroni test were used to detect differences between the blood gas values in regard to the age class and body position. A P value below 0.05 was considered significant. The data were normally distributed, and data are presented as mean values with confidence intervals.

RESULTS

In the studied rhinoceroses, no mortality occurred during the anesthetic procedures. Recovery was smooth in all animals, and it occurred within 1 to 2 min of administering the reversal agent. In the animals that were observed during the following days, no complications were noted in the postanesthetic period. There were no significant differences in the mean induction times between the control and butorphanol groups, and the values were 436 sec (331–540) and 452 sec (341–562), respectively. There were no significant differences in the distance the animal ran before darting. In contrast, the distance the butorphanol group ran was significantly less after darting compared with that of the control group. The values were 503 m (88–919) in the butorphanol group and 1,273 m (813–1,732) in the control group. There were significant differences in heart rate and respiratory rate between the two groups only at T2. In contrast, there were significant differences in body temperature at T1 and T2. In the butorphanol group, mean heart rate was 93 beats/min (85–100), mean respiratory rate 12 breaths/min (11–13), and mean body temperature

Table 2. Summary of the arterial blood gas values from rhinoceroses in lateral, sternal, and standing positions.

Parameter	Position	Control group (n = 15)	Butorphanol group (n = 17)
pH	lateral	7.22 (7.16–7.28)	7.26 (7.22–7.30)
	sternal	7.28 (7.22–7.32)	7.22 (7.12–7.32)
	standing	na[a]	7.30 (7.21–7.40)
	overall	7.25 (7.21–7.29)	7.26 (7.23–7.30)
$PaCO_2$ (mmHg)	lateral	63.9 (59.7–68.1)	66.0 (63.2–68.7)
	sternal	64.6 (60.1–69.1)	68.4 (63.2–73.5)
	standing	na	56.6 (49.2–64.0)
	overall	64.3 (61.4–67.1)	64.3 (61.6–67.0)[b]
PaO_2 (mmHg)	lateral	54.1 (46.3–61.8)[c]	36.3 (33.3–40.0)
	sternal	63.0 (55.8–70.0)[c]	45.8 (34.7–57.0)
	standing	na	49.9 (39.7–60.0)
	overall	58.9 (53.7–64.1)[c]	41.8 (37.9–45.6)[b]
HCO_3^- (mmol/L)	lateral	25.7 (22.5–28.9)[c]	29.4 (26.8–32.0)
	sternal	29.4 (26.2–32.2)	28.2 (22.0–34.1)
	standing	na	28.2 (24.6–31.8)
	overall	27.7 (25.6–29.9)	28.9 (27.1–30.7)
BE (mmol/L)	lateral	−1.45 (−5.3 to +2.4)	+2.3 (−0.9 to +5.5)
	sternal	+3.2 (−0.3 to +6.8)	+0.5 (−7.6 to +8.6)
	standing	na	+1.7 (−3.1 to +6.5)
	overall	+1.1 (−1.5 to +3.7)	+1.8 (−0.5 to +4.1)
SaO_2 (%)	lateral	71 (64–78)[b,c]	57 (52–63)
	sternal	81 (75–86)	65 (45–85)
	standing	na	78 (67–88)[b]
	overall	76 (72–81)[c]	64 (58–69)
$P(A\text{-}a)O_2$ (mm Hg)	lateral	12.6 (4.7–20.4)[b,c]	31.1 (21.3–40.9)
	sternal	3.1 (−3.1 to 9.3)[c]	14.8 (6.9–22.8)
	standing	na	25.1 (15.3–35.0)
	overall	7.4 (2.5–12.4)[c]	26.6 (20.4–32.9)

[a] na, not applicable.
[b] Represents significant differences ($P < 0.05$) within a group in different body positions.
[c] Represents significant differences ($P < 0.05$) between control and butorphanol groups.

was 37.3°C (36.8–37.7). In the control group, mean heart rate was 79 beats/min (67–92), respiratory rate was 9 breaths/min (8–10), and body temperature was 39.3°C (38.9–39.7). There were no significant differences in the immobilization level. The mean values were 3.3 in both the butorphanol and control groups.

The results of the blood gas analyses are summarized in Tables 2 and 3. Both groups showed respiratory and metabolic acidosis, hypoxemia, and hypercapnia. In the control group, PaO_2 values were significantly higher and the alveolar to arterial oxygen pressure gradients [$P(A\text{-}a)O_2$] were significantly lower in all body positions than in the bu-

Table 3. Summary of the arterial blood gas values from the rhinoceroses immobilized with etorphine, azaperone, detomidine ± butorphanol divided into age classes.

Parameter	Calves (n = 14)	Subadults (n = 12)	Adults (n = 5)
pH	7.23 (7.19–7.27)	7.26 (7.22–7.30)	7.30 (7.27–7.34)
$PaCO_2$ (mm Hg)	65.0 (62.1–67.9)	65.7 (63.1–68.4)	59.7 (53.8–65.5)
PaO_2 (mm Hg)	56.7 (51.0–62.5)[a]	41.6 (37.4–45.9)	44.1 (34.6–53.6)
HCO_3^- (mmol/L)	27.1 (24.9–29.3)	29.7 (27.4–31.9)	29.5 (27.6–31.4)
BE (mmol/L)	+0.2 (−2.6 to +3.0)	+2.6 (−0.2 to +5.4)	+3.1 (+1.1 to +5.1)
SaO_2 (%)	73 (67–78)	64 (58–70)	68 (56–81)
$P(A\text{-}a)O_2$	8.1 (3.1–13.0)[a]	26.5 (16.9–36.0)	27.2 (20.1–34.3)

[a] Represents significant differences ($P < 0.05$) between different age groups.

torphanol group. SaO_2 saturation in the control group was higher than the butorphanol group only in the lateral position. A change in body position from lateral to sternal recumbency showed a significant improvement in the PaO_2 and SaO_2 as well as a decrease in $P(A\text{-}a)O_2$ in both groups. Compared with the subadults and adults, PaO_2 values were significantly higher and $P(A\text{-}a)O_2$ was significantly lower in the calves. Between the subadults and adults, there were no marked differences.

There were no significant differences in the lactate values between the groups, but there was a trend toward higher values in the control group. In both groups, the lactate values were higher in the first measurement compared with the second sample. In the control group, the mean lactate value was 9.5 mmol/L (6.5–12.4) in the first measurement and 6.0 mmol/L (2.2–9.8) in the second measurement. In the butorphanol group, the values were 6.4 mmol/L (3.6–9.2) and 4.6 mmol/L (1.6–7.5), respectively.

DISCUSSION

Arterial blood gas analyses during anesthesia of white rhinoceroses presented in this study revealed hypoxemia ($PaO_2 < 70$ mm Hg), hypercapnia, and metabolic acidosis in both the control and butorphanol group. These derangements persisted throughout the duration of the anesthesia, despite partial reversal of the etorphine and changes in body postures. Severe hypoxemia with PaO_2 values below 60 mm Hg have been reported in captive as well as in free-ranging white rhinoceroses anesthetized with etorphine.[2,10,11] Metabolic acidosis in the rhinoceroses in this study probably resulted from lactic acid accumulation due to muscle activity before and after darting, and hypoxemia during the recumbent period. Lactate values decreased during the anesthetic period.

Hypoxemia is defined as abnormally low PaO_2 in the arterial blood, and it can result in abnormal organ function, cellular damage due to decreased oxygen delivery to the tissues, or both.[36] Respiratory causes that can lead to hypoxemia include hypoventilation and ventilation/perfusion imbalance (V/Q mismatch).[20] $PaCO_2$ is a measure of the ventilatory status of the patient, and it normally ranges between 35 and 45 mm Hg in domestic herbivores.[4] In 30 of 32 animals in this study, the $PaCO_2$ values were above 50 mm Hg, and in 22 of 32 rhinoceroses, the $PaCO_2$ values were even above 60 mm Hg. These elevated $PaCO_2$ values indicate hypoventilation. No differences were seen between the two groups. Similar elevated $PaCO_2$ values have been reported both in free-ranging and in captive rhinoceroses, but higher values were reported in captive rhinoceros immobilized with etorphine, detomidine, acepromazine, and butorphanol.[10,11,35] Increased scatter of ventilation and perfusion leads to increased $P(A\text{-}a)O_2$ gradients and decreased PaO_2. It is generally accepted that V/Q mismatch develops during anesthesia in large animals.[8,24] Presumably, in nondependent lung zones, regions exist without perfusion (alveolar dead space), whereas in the atelectatic lung zones, regions exist without ventilation (shunted perfusion). In this study, $P(A\text{-}a)O_2$ gradients ranged from 0 to 45 mm Hg. In 19 of 32 animals, the gradients were above 20 mm Hg, indicating some shunted perfusion. It is thus likely that hypoventilation with areas of ventilation/perfusion imbalances were the most probable cause for the low PaO_2 levels seen in these animals.

The use of potent opioids such as etorphine has respiratory depressant effects in rhinoceroses, and it is the most likely cause of hypoventilation in the studied rhinoceroses.[2] α2-Adrenoreceptor agonists, such as detomidine, are also known to have cardiopulmonary side effects in domestic animals, and they have synergistic effects with opioids.[6,14,27] Although only small doses were used, detomidine may have further aggravated the respiratory depression induced by etorphine.

Butorphanol is a synthetic opioid with both agonist and antagonist properties.[3] It is a κ-receptor agonist and μ-receptor antagonist. When used alone, butorphanol provides only mild sedation. One of its major advantages as an anesthetic agent is its minimal respiratory and cardiovascular side effects.[34] Respiratory depression seems to reach a ceiling beyond which higher doses do not produce an appreciably greater depression. In rats, butorphanol given subcutaneously produced only a small increase in $PaCO_2$ and a decrease in pH.[25] Pure opioid agonists such as etorphine can be reversed using specific antagonists such as naloxone or naltrexone. The deleterious side effects of μ-receptor activation, such as respiratory depression, are completely reversed; however, analgesia and sedation also are reversed.[34] In an attempt to selectively antagonize the undesirable side effects of pure opioid agonists while preserving their potent analgesic effects, opioid agonist-antagonists such as butorphanol have been used with varying success in humans and animals.[12,13,21] McCrackin et al.[21] demonstrated that a single bolus of butorphanol administered to dogs at the end of surgery caused partial reversal of oxymorphone-induced respiratory depression and bradycardia. The treatment group had increased respiratory rates, tidal volumes, and minute volumes as well as normal end-tidal CO_2 values within

2 min of butorphanol administration. Butorphanol was also effective in reversing the respiratory depression that was produced by fentanyl in rats.[12] To improve ventilation, butorphanol has been used in recumbent white rhinoceroses immobilized with etorphine.[22] In our study, no advantageous effects were seen in PaO_2 levels by adding butorphanol to the immobilizing mixture. The control group had significantly higher PaO_2 and SaO_2 levels than the butorphanol group. One reason that no beneficial effects were seen in this study could be because insufficient doses of butorphanol were used or because the body weight differences between the two groups minimized the potentially beneficial effects of the butorphanol. Further research is needed to evaluate whether, by increasing the butorphanol dose and decreasing the etorphine dose, beneficial effects regarding oxygenation are observed. Nevertheless, the etorphine dose can only be decreased and the butorphanol dose increased to a certain level; otherwise, the rhinoceros will not become recumbent.

In this study, PaO_2 values were significantly higher and $P(A\text{-}a)O_2$ gradients were lower in the calves compared with the subadults and adults. The massive size of the white rhinoceros, and in particular its large digestive tract, is thought to be a major factor in reducing the ability of the recumbent animal to breathe adequately.[5] This finding is compatible with reports in horses indicating a positive correlation between body weight and pulmonary shunt.[24] In the control group, predominantly calves were immobilized, whereas in the butorphanol group, there was a higher proportion of subadults or adult animals. This could have contributed to the lower PaO_2 levels and higher $P(A\text{-}a)O_2$ gradients seen in the butorphanol group. Further research, in which comparable age groups are studied, is required to see whether this would result in different PaO_2 values and $P(A\text{-}a)O_2$ gradients. Another limitation of this study is the small number of animals in each group.

A change in body position from lateral to sternal recumbency increased PaO_2 values and decreased $P(A\text{-}a)O_2$ gradients in the studied rhinoceroses. This finding is compatible with studies indicating that changes in posture of anesthetized horses exert profound effects on arterial oxygenation.[7,8,31,32]

The administration of partial antagonists within 10 min of recumbency has been suggested with the objective to improve hypoventilation.[18,28] In this study, the partial reversal of the immobilization showed no definite improvement in the oxygen and $PaCO_2$ levels. In a recently published article, it was reported that free-ranging white rhinoceroses that received nalorphine after becoming recumbent showed only a minimal improvement of PaO_2 of 7 mm Hg after partial reversal of immobilization.[2]

Oxygen supplementation has been used effectively in rhinoceroses during immobilization to increase PaO_2 levels. In a captive white rhinoceros, insufflation of oxygen at the level of the left nostril increased PaO_2 to a peak of 135 mm Hg.[11] In a study by Bush et al.,[2] PaO_2 and SpO_2 values increased rapidly after nasotracheal oxygen supplementation, whereas the rhinoceroses that did not receive oxygen remained very hypoxemic throughout the monitoring period. Oxygen supplementation had no influence on hypercapnia or metabolic acidosis. In fieldwork, supplementation of oxygen can be cumbersome when equipment needs to be carried over long distances, but, in our case, it would have been a good option to improve oxygenation.

There was a trend of lower lactate values and higher BE in the butorphanol group, indicating that these animals had a less severe metabolic acidosis. One of the reasons could be the result of having run a significantly shorter distance after darting than the control group. The mean distance covered during induction was 500 m, which is less than values reported in free-ranging black rhinoceroses (*Diceros bicornis*) immobilized with etorphine, xylazine, and hyaluronidase in Zimbabwe.[15] The addition of butorphanol could have a positive effect by reducing the running and pacing commonly observed after administration of etorphine.[33] Capture myopathy is a multifactorial disease that has been described in a wide range of species and often leads to the loss of the animal.[23,30,37] The pathophysiology is associated with the duration and intensity of the physical effort expended during the capture event.[30] The addition of butorphanol could be beneficial in the prevention of capture myopathy as animals run for a shorter distance. Further studies are needed to evaluate whether this could reduce the risk of losing an animal in the bush and reduce muscle damage.

The control group had lower mean heart and respiratory rates at time T2 when the second blood sample was taken. There are many explanations for this, but the most probable reason is that the second sample was taken approximately 10 min later than in the butorphanol group. This was due to limited personnel availability in the control group and to the rapid accomplishment of the game capture in the butorphanol group. In most animals, heart and respiratory rates decreased over time, and they stabilized at a certain level 20 to 30 min after becoming immobile. The control group also had significantly higher body temperatures, which is probably

due to the higher ambient temperatures and because they ran for a longer distance during the darting procedure. In a study performed in captive rhinoceros anesthetized with etorphine, detomidine, butorphanol, and acepromazine, mean heart rate was similar to the values found in the butorphanol group and it decreased over time.[35] Other studies in free-ranging animals immobilized with similar protocols have shown comparable heart rates, whereas protocols using no α_2-agonists report higher heart rates.[2,18]

In this study, no anesthetic mortality or morbidity was observed in the rhinoceroses. Recovery was smooth in all animals, and it occurred within 1 to 2 min of administering the opioid antagonist. No complications were noted in the animals observed during the subsequent days. Due to the high oxygen affinity of the rhinoceros (low P_{50} of about 2.66 kPa [20 mm Hg]) and lower tissue metabolic rate of large mammals, the rhinoceros is probably better able to maintain adequate tissue oxygenation with low PaO_2 values than smaller animals.[1,11] Healthy animals can tolerate these severe physiological alterations for short periods during field immobilizations with etorphine combinations.[2] Nevertheless, during capture and translocation procedures, complications can occur, and hypoxemia may be one cause of perianesthetic mortality in the rhinoceros.[11,16,18]

CONCLUSIONS

In this study, severe hypoxemia and hypoventilation were seen in the rhinoceroses immobilized with etorphine in both the control and butorphanol group. Improvements in arterial oxygen levels were observed when animals were placed in sternal recumbency. Partial reversal of the immobilization with nalorphine or diprenorphine to increase ventilation showed only minimal improvements in PaO_2 levels. By adding butorphanol to the immobilizing mixture, no benefits in ventilation were seen, although the body weight differences may have influenced the results. The rhinoceroses ran for a shorter distance during induction, which could be beneficial in the prevention of capture myopathy. Further research is needed to evaluate the effects on respiratory parameters by increasing butorphanol doses in the immobilizing mixtures.

Acknowledgments: This study was carried out in fulfilment of the Wild Animal Health M.Sc. degree (to S.W.) at the Royal Veterinary College and the Zoological Society of London. We thank Miss Aviva Petrie for help with statistical analyses and Dr. Tony Sainsbury for comments with the manuscript. We also thank Axonlab AG for lending the i-STAT blood gas analyzer; the veterinary department of the Zoological Society of London for providing consumables; and Zebra Foundation, England, for assisting with funding.

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Received for publication 26 October 2006