Severe hypoxaemia in field-anaesthetised white rhinoceros (*Ceratotherium simum*) and effects of using tracheal insufflation of oxygen

M Bush[a*], J P Raath[b], D Grobler[c] and L Klein[d]

ABSTRACT

White rhinoceros anaesthetised with etorphine and azaperone combination develop adverse physiological changes including hypoxia, hypercapnia, acidosis, tachycardia and hypertension. These changes are more marked in field-anaesthetised rhinoceros. This study was designed to develop a technique to improve safety for field-anaesthetised white rhinoceros by tracheal intubation and oxygen insufflation. Twenty-five free-ranging white rhinoceros were anaesthetised with an etorphine and azaperone combination for translocation or placing microchips in their horns. Once anaesthetised the rhinoceros were monitored prior to crating for transportation or during microchip placement. Physiological measurements included heart and respiratory rate, blood pressure and arterial blood gas samples. Eighteen rhinoceros were intubated using an equine nasogastric tube passed nasally into the trachea and monitored before and after tracheal insufflation with oxygen. Seven rhinoceros were not intubated or insufflated with oxygen and served as controls. All anaesthetised rhinoceros were initially hypoxaemic (percentage arterial haemoglobin oxygen saturation ($\%O_2S_a$) = 49 % ± 16 (mean ± SD) and P_aO_2 = 4.666 ± 1.200 kPa (35 ± 9 mm Hg)), hypercapnic (P_aCO_2 = 8.265 ± 1.600 kPa (62 ± 12 mm Hg)) and acidaemic (pH_a = 7.171 ± 0.073). Base excess was –6.7 ± 3.9 mmol/ℓ, indicating a mild to moderate metabolic acidosis. The rhinoceros were also hypertensive (systolic blood pressure = 21.861 ± 5.465 kPa (164 ± 41 mm Hg)) and tachycardic (HR = 107 ± 31/min). Following nasal tracheal intubation and insufflation, the $\%O_2S_a$ and P_aO_2 increased while blood pH_a and P_aCO_2 remained unchanged. Tracheal intubation via the nose is not difficult, and when oxygen is insufflated, the P_aO_2 and the $\%O_2S_a$ increases, markedly improving the safety of anaesthesia, but this technique does not correct the hypercapnoea or acidosis. After regaining their feet following reversal of the anaesthesia, the animals' blood gas values return towards normality.

Key words: anaesthesia, arterial blood gases, azaperone, *Ceratotherium simum,* etorphine, hypercapnia, hypoxia, insufflation, intubation, oxygen, rhinoceros.

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INTRODUCTION

Safe and reliable anaesthesia of rhinoceros is an important tool for health care of both captive and free-ranging animals and for conservation-based programmes. Physiological monitoring of white rhinoceros anaesthetised with etorphine hydrochloride (M99) reveals physiological alterations, including hypertension, tachycardia, acidaemia and respiratory depression with hypoxia and hypercapnia[2,5–8]. The addition of azaperone to M99 decreased blood pressure in a series of 6 animals[6]. Many reports recommend oxygen (O_2) supplementation, partial reversal with nalorphine, and/or respiratory stimulants to correct the hypoxia[3,6,12]. An M99-anaesthetised captive white rhinoceros, intubated and maintained on isoflurane with intermittent partial pressure ventilation (IPPV) remained hypoxic and hypercapnic. This was attributed to a ventilation perfusion inequality[2].

Field anaesthesia of rhinoceros usually results in more marked physiological changes than seen in captive animals, since higher dosages of M99 are used to shorten induction times, which leads to additional respiratory depression. Fortunately, most healthy rhinoceros tolerate this brief period of physiological insult. In a captive situation longer induction times are usually acceptable, therefore lower dosages of M99 can be used.

Oral tracheal intubation of the rhinoceros is difficult due to 1) the heavy head, 2) general muscle rigidity associated with M99 anaesthesia, and 3) the large molar teeth with a narrow oral passage between them.

Nasotracheal intubation for delivery of O_2, anaesthetics and IPPV is used in domestic animals, including adult horses, foals, llamas and calves[11,14,17]. This study records a technique of nasotracheal intubation of field-anaesthetised white rhinoceros combined with O_2 insufflation, to improve oxygenation.

MATERIALS AND METHODS

The study animals were 22 white rhinoceros captured during a conservation-based relocation programme in the Kruger National Park, South Africa. There were 8 adult females and 14 subadults (8 males and 6 females) in the study. Three additional female white rhinoceros (1 adult and 2 subadults) were studied during a capture operation at the Mpumalanga Parks to place microchips the animals' horns. The capture method used at both locations has been reported in a recent summary of anaesthesia procedures in white rhinoceros[12]. All animals were darted from a helicopter with dosages ranging from 1.3 to 4 mg of etorphine hydrochloride (M99, C-Vet, SA) plus 20–80 mg of azaperone (Stressnil, Janssen Animal Health, SA) depending on the animal's size. To shorten induction time, 7500 IU of hyaluronidase (Sigma Chemical Co, St Louis, MO, USA.) was added to each dart. The delivery system was either 1) a modified shotgun (20-gauge Miroku O/U) to propel a 3 ml aluminum dart using a bicarbonate/acetic acid injection system (Gunsmith, Kruger National Park, Skukuza, SA) or 2) a CO_2-powered remote injection device delivering a 3 ml plastic air-pressurised dart with a 60 mm needle (Dan-Inject, SA). Once down the animals were place in sternal recumbency,

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blindfolded and earplugs were inserted prior to the monitoring.

To ensure the safety of the rhinoceros during this study, 20–50 mg nalorphine (Lethidrone, Wellcome) and/or 100–400 mg Doxapram HCL (Dopram-V, Fort Dodge Animal Health, Fort Dodge, Iowa 50501 USA) were administered intravenously (i.v.), at the discretion of the veterinarian in charge (JPR or DG) to reverse apnoea both before and during the data-collection period.

Arterial blood gases (ABG) were collected following catheterisation of the auricular artery located on the inside of the pinna, using a 22 gauge catheter (Jelco, Critikon, SA, Johnson and Johnson (Pty) Ltd. SA). The samples were collected anaerobically into heparinised syringes, which were sealed and stored on ice until analysed within 3 hours of collection. The catheter was maintained with a heparin saline flush and 1–2 mℓ of blood was withdrawn and discarded prior to collection of the ABG sample. Arterial haemoglobin oxygen saturation ($\%O_2S_a$) and base excess (BE) were calculated according to standard human formulae. Samples were collected prior to administration of O_2 and at various intervals during O_2 administration. Samples from control animals (4 from the Kruger Park and 3 from Mpumalanga Parks) were also collected at various intervals during the monitoring period.

Tracheal intubation was accomplished with an equine nasogastric tube (0.9 or 1.4 cm internal diameter) (Kalayjian Industries, Inc, Long Beach, CA 90806, USA) passed along the ventral floor of the nasal cavity to avoid the nasal turbinates. Once the tube was in the posterior pharynx, the operator listened for an exhalation and then advanced the tube into the trachea to approximately the level of the tracheal bifurcation (Fig. 1). The tube never entered the oesophagus during this study, but on occasion was reflected by the posterior pharyngeal wall and came out the other nostril. Tracheal intubation was verified by listening to the air passing through the tube during respiration. Oxygen was administered at a rate of 15–30 ℓ/min depending on the size of the animals.

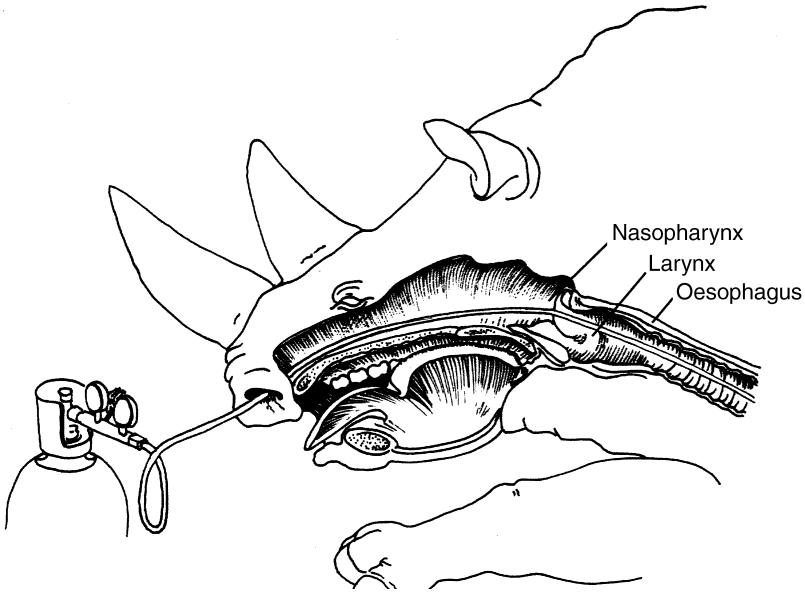


Fig. 1: **Drawing of a recumbent rhinoceros showing the placement of the nasotracheal tube passing through the larynx into the trachea, with the tube attached to an oxygen tank with a pressure regulator and a flow meter to control the O_2 flow.**

The data-gathering period varied among animals since it was related to the time required to crate each animal. When 2 animals were anaesthetised simultaneously, 1 animal was chosen to be crated last, thus obtaining a longer monitoring period. The control animals were selected randomly.

Systolic blood pressure was measured indirectly from the tail (Dinamap, Critikon Inc. Tampa, Florida 33607 USA). Respiration rate was obtained by feeling the expiration of air and a heart rate was obtained using a stethoscope. Rectal temperature was taken to calibrate the blood gas machine.

Following the monitoring period in the Kruger Park rhinoceros, M99 effects were antagonised with either 6–7.5 mg diprenorphine (M50-50, C-Vet) or 25–40 mg nalorphine i.v. and each animal was directed to the crate using the pre-placed ropes after it regained its feet. In 10 rhinoceros it was possible to obtain an ABG sample from the arterial catheter in the auricular artery after the animal was standing in the crate. The 3 control rhinoceros from Mpumalanga were given 40–80 mg naltrexone i.v. and allowed to recover and return to their habitat.

RESULTS

No mortality or post-anaesthetic morbidity occurred in the rhinoceros that were studied.

The results of physiological monitoring of rhinoceros that received O_2 supplementation and the controls are summarised in Table 1. Physiological data were not recorded for all animals studied. Note that the means of all ABG parameters except the P_aO_2 and the $\%O_2S_a$ are similar in the groups. Both groups showed respiratory and metabolic acidosis, hypercapnia, tachycardia, decreased respiratory rate and moderate hypertension.

The summary of all ABG samples collected over the monitoring period divided into the group that received O_2 supplementation and the non-supple-

Table 1: **Summary of the physiological monitoring and arterial blood gas values from both O_2-supplemented and control animals. Mean values for all parameters (except P_aO_2 and $\%O_2S_a$, which increased markedly in the supplemented animals) are comparable and show acidosis, hypercapnia, tachycardia, decreased respiratory rate and moderate hypertension.**

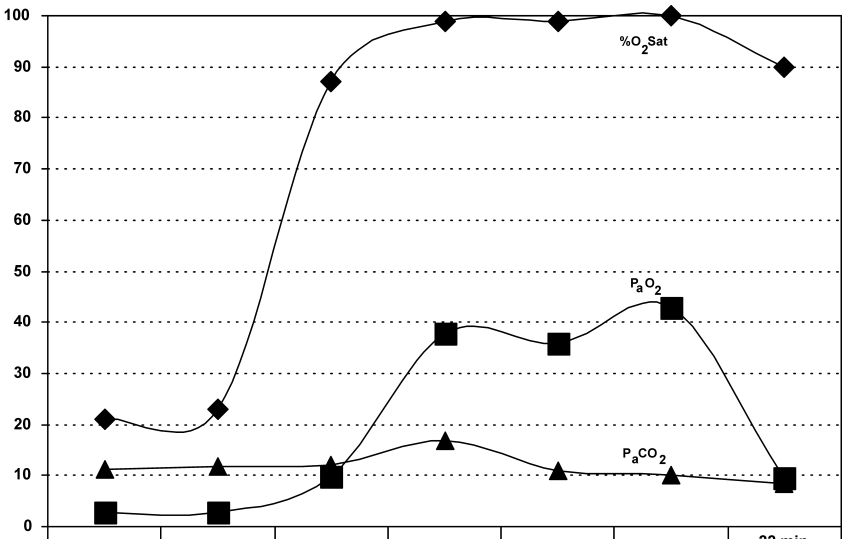
		S_aO_2 (%)	pH_a	P_aO_2 (kPa)	P_aCO_2 (kPa)	HCO_3 (mmol/ℓ)	BE (mmol/ℓ)	Heart rate (per min)	Respiration rate (per min)	Systolic BP (kPa)
O_2 Suppl.	Mean	88	7.148	13.863	9.464	23.5	–5.8	104	9	24.93
	SD	15	0.097	8.265	2.666	4.2	4.4	38	3	7.73
	n	38	39	39	39	39	39	15	13	1.47
No O_2	Mean	57	7.206	5.065	8.665	23.7	–3.7	106	10	24.93
	SD	18	0.078	1.466	1.600	4.8	5.0	32	3	3.20
	n	36	36	36	36	32	36	7	10	1.07

Table 2: **Summary of the arterial blood gas values during the monitoring period from O_2-supplemented and control animals. Time 0 combines the initial sample on the supplemented animals and control animals. Following the oxygen supplementation the rise in P_aO_2 and $\%O_2S_a$ is rapid while the remaining values remain constant.**

		Time 0	O_2 supplementation			No O_2 supplementation		
		Arterial sample No O_2	First 10 min	11–20 min	20–40 min	First 10 min	11–20 min	20–35 min
pHa	Mean	7.175	7.133	7.142	7.213	7.246	7.244	7.246
	SD	0.076	0.114	0.082	0.036	0.051	0.084	0.067
S_aO_2 (%)	Mean	49	84	89	95	62	69	70
	SD	16	17	15	3	23	13	8
P_aO_2 (kPa)	Mean	4.666	12.797	15.330	13.597	4.932	5.465	5.599
	SD	1.200	7.865	10.131	3.333	1.866	1.600	0.933
P_aCO_2 (kPa)	Mean	8.265	9.731	9.731	8.398	9.331	8.531	8.931
	SD	1.600	3.333	2.266	0.533	0.933	1.200	2.000
HCO_3 (mmol/ℓ)	Mean	22	23	24	25	28	24	27
	SD	4	5	4	2	5	3	3
BE (mmol/ℓ)	Mean	–6.4	–6.77	–5.7	–3.2	–0.26	–1.4	0.3
	SD	4.27	5.3	3.4	1.9	3.49	5.73	2.7
Sample time (min)	Mean		7	15	28	10	17	24
	SD		3	3	7	0.1	3	3
	n	21	19	14	6	5	5	5

mented control group are shown in Table 2. Time 0 shows values obtained from the 1st ABG sample collected in both groups, and serves as a baseline for both the control animals and the animals that received O_2 following Time 0. The P_aO_2 and the $\%O_2S_a$ values increased rapidly and markedly following O_2 supplementation while the rhinoceros that did not receive O_2 remained very hypoxaemic throughout the monitoring period, with only a slight elevation at 30 min. The very low P_aO_2 values, combined with acidosis, resulted in severe haemoglobin desaturation with initial $\%O_2S_a$ values below 50 % recorded in the 7 control animals breathing ambient air, while during O_2 supplementation, 30 of 39 samples showed the $\%O_2S_a$ to be greater than 90 % and in only 1 sample was $\%O_2S_a$ below 80 %. The pH_a and $P_a CO_2$ in both groups showed consistent acidaemia and hypercapnoea throughout the monitoring period. Base excess remained below 0 mmol/ℓ except for the 20–25 min monitoring period in the non-supplemented rhinoceros (0.3 mmol/ℓ), indicating that metabolic acidosis persisted throughout the recumbent period. The mean bicarbonate value in the anaesthetised rhinoceros ranged between 22 and 28 mmol/ℓ, (Table 2), reflecting the decrease due to metabolic acidosis, combined with the increase resulting from hypercapnoea due to anaesthetic-induced respiratory depression.

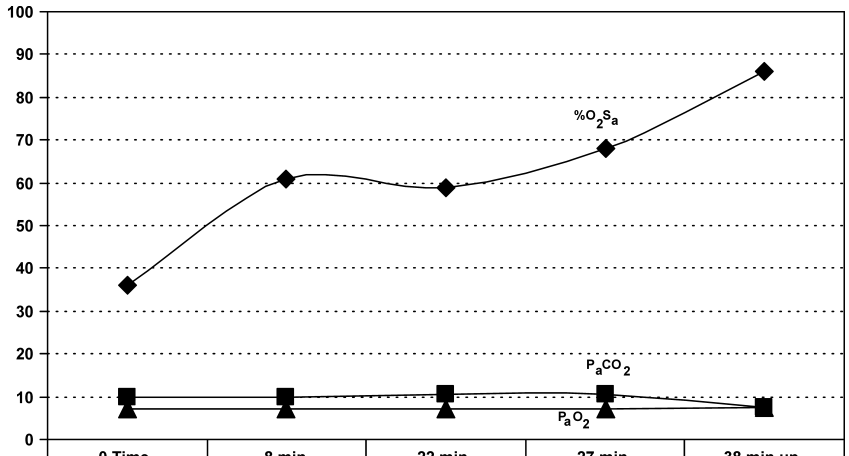
Arterial blood gas values from 2 subadult female rhinoceros in this study during the monitoring period and initial recovery are shown in Fig. 2 (an O_2 -supplemented animal) and Fig. 3 (a control animal) with the P_aO_2 and the $\%O_2S_a$ and P_aCO_2 plotted and the other ABG values listed in the table below the chart. Figure 2 plots P_aO_2 and $\%O_2S_a$ and P_aCO_2, before and during O_2 supplementation. The P_aO_2 and $\%O_2S_a$ increased rapidly during supplementation while the P_aCO_2 remained elevated. The ABG values from the standing animal following the reversal by M99 showed a drop in both P_aCO_2 and P_aO_2 while the pH_a and BE increased, indicating a partial correction of both the respiratory and the metabolic acidosis. Figure 3 shows that, in the non-O_2-supplemented animal, hypoxia and hypercapnoea persisted until the animal was standing after reversal. Both animals were also acidotic during recumbency, with the ABG values returning towards normal by the time the animals were standing.



	T = 0	1 min	4 min	9 min	11 min	15 min	22 min Up in crate
$\%O_2$ S_a	21	23	87	99	99	100	90
P_aO_2 kPa	2.93	2.93	9.84	37.72	35.99	42.92	9.46
P_aCO_2 kPa	11.2	11.86	12	16.93	11.06	10.13	8.53
pH_a	7.162	7.162	7.111	7.027	7.166	7.177	7.270
BE mmol/l	-2.9	-1.4	-3.4	-4.5	-2.2	-2.9	0.3

Fig. 2: **Chart and associated table showing the results of arterial blood gas values of a subadult female rhinoceros that received O_2 supplementation via a nasotracheal tube. Note the rapid initial rise of both the P_aO_2 and $\%O_2S_a$ once supplementation began. When the animal was standing in the crate the arterial blood gas values started to return towards normality.**

Figure 4 shows the initial effect of O_2



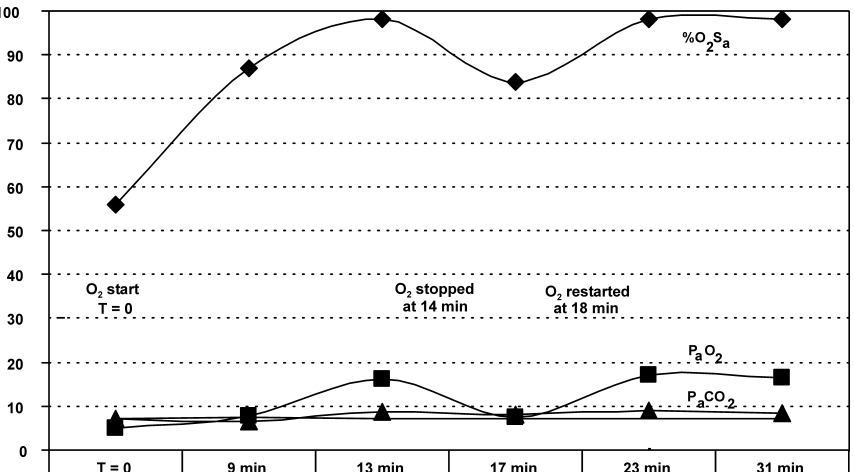
	0 Time	8 min	22 min	27 min	38 min up
$\%O_2S_a$	36	61	59	68	86
P_aO_2 kPa	5.2	5.33	5.2	5.87	7.73
P_aCO_2 kPa	9.86	10	10.53	10.53	7.46
pH_a	7.138	7.179	7.176	7.202	7.316
BE mmol/l	-6.1	-2.6	-2.1	-0.4	1.2

Fig. 3: **Chart and associated table showing the results of arterial blood gas values of a subadult female rhinoceros control that did not receive O_2 supplementation. Note hypoxia with low P_aO_2 and $\%O_2S_a$ and hypercapnia with elevated P_aCO_2, which persisted until the animal was standing in the crate at 38 min.**

supplementation on an adult rhinoceros. The O_2 supplementation was discontinued for 4 min at 14 min with a resultant decrease of both P_aO_2 and $\%O_2S_a$, which increased rapidly again once O_2 supplementation was resumed. This illustrates the rapid changes that occur in the ABG values

A common procedure is the administration of nalorphine (10–30 mg) i.v. to white rhinoceros within 10 min of recumbency.[7,12] The objective is to improve the safety of the procedure by lightening the opioid anaesthesia and improving respiration, which is observed clinically. Twelve animals that received nalorphine were monitored. Follow-up data, within 10 min of injection, showed very little improvement in the physiological parameters compared to the 5 control animals that did not receive nalorphine or O_2 supplementation (Table 3). The major physiological alterations persisted. When nalorphine was given the net change in P_aO_2, after an average of 7 minutes, was an increase of 0.93 kPa (7 mm Hg) from 4.27 to 5.20 kPa (32 to 39 mm Hg).

Table 4 shows the ABG values of 10 rhinoceros, including the animals in Figs 2 and 3, sampled at various times when they were standing in their crates after having received the antagonist to M99. The ABG values returned towards normal within an average of about 3 min after standing.



	T = 0	9 min	13 min	17 min	23 min	31 min
$\%O_2S_a$	56	87	98	84	98	98
P_aO_2 kPa	4.93	7.73	16.26	7.6	16.93	16.53
P_aCO_2 kPa	7.06	6.53	8.80	8.00	8.93	8.53
pH_a	7.176	7.321	7.221	7.216	7.241	7.224
BE mmol/l	-8.40	-1.00	-1.20	-0.30	0.00	-1.60

Fig. 4: **Chart and associated table showing the results of arterial blood gas values of an adult female rhinoceros that initially received O_2 supplementation. The supplementation was discontinued for 4 minutes with a marked drop in both the P_aO_2 and $\%O_2S_a$, which rapidly rose again once O_2 was restarted.**

DISCUSSION

This study documents 5 observations: 1) rhinoceros anaesthetised with doses of M99 and azaperone appropriate for free-ranging conditions experience severe respiratory depression and hypoxaemia, moderate hypercapnia, and combined respiratory and metabolic acidosis. 2) Ten rhinoceros required administration of nalorphine within the first 10 min to ensure their safety, while the resultant monitoring within 10 min of administration showed little physiological improvement. 3) Healthy rhinoceros can tolerate these severe physiological alterations for short periods during field anaesthesia with these agents. 4) Tracheal intubation and O_2 insufflation will rapidly and markedly improve oxygenation as shown by P_aO_2 and the $\%O_2S_a$, but does not affect hypercapnoea or metabolic acidosis. 5) Following antagonism of M99 and the end of recumbency the ABG values rapidly return towards normal levels.

The respiratory depression and resultant hypoxia is probably due to 4 factors: 1) the high dose of M99 required to shorten the induction time when anaesthetising an animal under field conditions; 2) white rhinoceros are very sensitive to opioids such as M99;[12,15] 3) a large animal recumbent while anaesthetised is subject to physiological alterations that lead to cardiopulmonary depression, as has been shown in domestic animals[9,16], elephants[3,4] and humans[10]; 4) a large recumbent animal will develop a perfusion/ventilation disparity[2,16].

There also seems to be variation between white and black rhinoceroses, with the former being much more sensitive to the respiratory depressant effects of opioids (J P R and D G, pers. obs., 2003).

In the group of white rhinoceros under study, the negative BE values indicate some degree of metabolic acidosis. Formulae used in the instrument software for the calculation of BE are based on the relationship of pH_a to P_aCO_2 in humans, with the 'normal' range for BE centred around a value of 0 mmol/ℓ. Using these formulae, normal domestic herbivores typically have BE values above 0, from +4 to +8 mmol/ℓ, depending on the specific

Table 3: **Effect of receiving nalorphine in 12 rhinoceros compared to 5 animals that did not receive nalorphine or O_2 supplementation. Note there is very little improvement of P_aO_2 and $\%O_2S_a$ following administration of nalorphine in these 12 animals.**

	Respiration (per min)		Heart rate (per min)		P_aCO_2 (kPa)		P_aO_2 (kPa)		O_2S_a (%)	
Time:	**T = 0**	**10 min**	**T = 0**	**10 min**	**T = 0**	**10 min**	**T = 0**	**10 min**	**T = 0**	**10 min**
12 Nalorphine	10	10	138	115	9.60	9.60	4.67	5.47	71	75
5 Controls	9	11	113	87	9.20	9.20	5.20	5.20	74	79

Table 4: **Arterial blood gas values from 10 rhinoceros while they were standing in a crate after reversal of M99. Note that all values are returning towards normal compared with values during anaesthesia.**

			Arterial samples					
	Total time down (min)	**Sample after up** (min)	S_aO_2 (%)	pH_a	P_aO_2 (kPa)	P_aCO_2 (kPa)	HCO_3 (mmol/ℓ)	**BE** (mmol/ℓ)
	12	1	87	7.279	8.265	7.878	26.9	–0.3
	49	12	82	7.238	7.598	8.225	25.4	–2.5
	21	1	90	7.266	9.464	8.345	27.6	1.0
	21	7	90	7.270	9.464	8.478	28.3	0.3
	41	4	86	7.316	7.731	7.558	28.2	1.2
	60	1	91	7.315	9.064	6.572	25.4	–0.8
	41	3	82	7.304	6.932	6.238	23.5	–2.6
	35	3	76	7.306	5.999	6.092	23.0	–2.9
	26	1	88	7.292	8.531	8.131	28.7	0.8
	29	1	92	7.340	9.998	7.505	29.7	2.9
Mean	33.5	3.4	86.5	7.293	8.291	7.505	26.7	–0.3
SD	14.6	3.6	5.1	0.030	1.253	0.893	2.3	1.91
n =	10	10	10	10	10	10	10	10

blood gas analyser (L K, pers. obs., 2003). Although normal values for unrestrained, free-ranging white rhinoceros are not available, it is likely that their acid-base balance is similar to other herbivores and that the negative BE values reported here represent a true metabolic acidosis. Metabolic acidosis in these animals probably resulted from lactic acid accumulation due to muscle activity before and after darting, and hypoxaemia during the recumbent period. The persistence of metabolic acidosis even in those animals that received O_2 supplementation may be due to the muscle activity and rigidity, which typically accompanies restraint with M99 in white rhinoceros.

Arterial haemoglobin oxygen saturation values in these rhinoceros were calculated, also using formulae based on the O_2 affinity and Bohr effect (effect of a change in pH on the P50 (P_aO_2 at which the haemoglobin is half-saturated with O_2)) of normal adult human haemoglobin. Domestic horses have a P50 of ≅ 3.33 kPa (25 mm Hg), similar to humans and therefore the $\%O_2S_a$ values calculated from human formulae are reasonably accurate. No reports of respiratory characteristics of white rhinoceros haemoglobin using whole blood could be found in the literature, but results of 1 study of the effects of various anions and other factors on the O_2 affinity of white rhinoceros haemoglobin in solution suggested that the P50 might be ≅ 2.67 kPa (20 mm Hg)[1]. If this is the case, then the true $\%O_2S_a$ in the rhinoceros in this study may be somewhat higher than the values reported here although still profoundly desaturated in those animals did not receive O_2 supplementation.

Nasal tracheal intubation is not a difficult procedure in the field. The insufflation with O_2 improves the safety of the procedure by increasing the P_aO_2 and the $\%O_2S_a$, but does not correct the acidosis or hypercapnia, which has also been reported in wapiti[13]. Giving O_2 does not seem to worsen the respiratory depression by removing hypoxic respiratory drive, since significant increases in $P_a CO_2$ were not detected during O_2 supplementation. Care must be taken, when insufflating oxygen though a nasotracheal tube or catheter, to ensure that the tube is in the trachea, and not the oesophagus. Insufflation of oxygen into the oesophagus can result in gastric rupture, which has occurred in horses (L K, pers. obs., 2003), but in this study we did not pass the tube in to the oesophagus, probably thanks to pharyngeal anatomy that renders it difficult.

Methods to correct the acidosis and hypercapnoea would include IPPV, which would be difficult in the field, since it would require placement of a cuffed endotracheal tube and a large-capacity ventilator, or 2 demand valves joined in parallel with a larger source of compressed O_2 than required for insufflation as used in this study. Even with IPPV in large species, hypoxia may persist in some individuals due to ventilation/perfusion disparity, and hypercapnoea persists because of insufficient tidal and minute volume[2].

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