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PULMONARY DEAD SPACE IN FREE-RANGING IMMOBILIZED BLACK RHINOCEROSES (*DICEROS BICORNIS*) IN NAMIBIA

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Abstract: It was observed previously that end-expired carbon dioxide ($P_{\dot{E}}CO_2$) decreased when immobilized black rhinoceroses (*Diceros bicornis*) were moved from sternal to lateral recumbency. These experiments were designed to test whether greater alveolar ventilation or greater pulmonary dead space in lateral recumbency explains this postural difference in $P_{\dot{E}}CO_2$. Twenty-one (9 male, 12 female; 15 [3.5–26] yr old) wild black rhinoceroses were immobilized with etorphine and azaperone and positioned in either sternal or lateral recumbency. All rhinoceroses were hypoxemic and had lactic and respiratory acidemia. The animals in lateral recumbency were more acidemic, had higher lactate, and lower arterial oxygen that those in sternal recumbency; however, arterial carbon dioxide was similar between groups. Both $P_{\dot{E}}CO_2$ and mixed expired carbon dioxide pressure were lower in lateral than sternal recumbency. Although there was no difference in tidal volume or arterial carbon dioxide, both the breathing rate and minute ventilation were greater in lateral recumbency. The physiologic dead space ratio and dead space volume were approximately two times larger in lateral recumbency; hence, the decrease in $P_{\dot{E}}CO_2$ in lateral recumbency can be attributed to increased dead space ventilation not increased alveolar ventilation. Positioning immobilized rhinoceroses in lateral recumbency does not confer any advantage over sternal in terms of ventilation, and the increase in minute ventilation in lateral recumbency can be considered an energetic waste. Although arterial oxygen was superior in sternal recumbency, further studies that measure oxygen delivery (e.g., to the muscles of locomotion) are warranted before advice regarding the optimal position for immobilized rhinoceroses can be given with confidence.

Key words: Anesthesia, black rhinoceros, capnography, dead space, *Diceros bicornis*, oxygenation, posture.

INTRODUCTION

The black rhinoceros (*Diceros bicornis*) is critically endangered, with just over 5,000 animals surviving in the wild in southern Africa. Optimal management of rhinoceros populations requires capture of individuals for procedures, including translocation to minimize regional genetic homogeneity and for repatriation. However, in such a small population, occasional morbidity and mortality associated with capture is unacceptable.[15] The harsh nature of black rhinoceros' habitat usually requires that free-ranging animals be immobilized by darting from a helicopter; this procedure inevitably induces an extreme flight response. Established immobilization protocols for black rhinoceroses use potent opioids, such as etorphine, injected by dart; unfortunately, these drugs produce important side effects, including hypoventilation, hypoxemia, hypercapnea, hypertension, and acidemia that may contribute to mortality during capture.[11,10,3,4,6,19] These pharmacologic perturbations exacerbate the effects of the exertion that usually precedes capture.

In large quadrupeds, both the pulmonary and cardiovascular systems are affected by posture under anesthesia.[24,25] In anesthetized horses (*Equus caballus*) that have not undergone exertion, alveolar ventilation and arterial oxygenation are greater in sternal recumbency than in lateral recumbency.[9] On the other hand, in anesthetized adult elephants, the pulmonary system is generally considered to be more disadvantaged in sternal recumbency than it is in lateral recumbency.[12,15] The optimal posture for rhinoceroses under anesthesia has not been determined.

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Figure 1. Field collection of expired gases in a sternally recumbent free-ranging black rhinoceros (*Diceros bicornis*) after capture in Etosha National Park, Namibia.

End-expired carbon dioxide ($P_{\dot{E}}CO_2$) decreased when immobilized black rhinoceroses were moved from sternal to lateral recumbency and increased when they were moved from lateral to sternal recumbency.[17] These observations could be explained by greater alveolar ventilation in lateral recumbency or reduced alveolar ventilation in sternal recumbency or by incremental and decremental changes in pulmonary dead space, respectively. These experiments were designed to test which mechanism explains those observations. The results will also add to the knowledge base on which decisions regarding positioning are made for rhinoceroses during capture and immobilization.

MATERIALS AND METHODS

Study site and subjects

The research protocol was approved by the Cornell University Institutional Animal Care and Use Committee (Protocol No. 2006-0170) and by the Namibian Ministry of Environment and Tourism (MET). Study sites were located in eastern Etosha National Park (latitude: 19°0′0″S, longitude: 16°0′0″E). Twenty-one (9 male, 12 female; age 15 [3.5–26] yr; median (minimum–maximum)] desert-adapted black rhinoceros were captured in March and April of 2011 for routine identification and biologic data gathering. No rhinoceroses were immobilized specifically for this study. The subjects were immobilized by remote intramuscular injection from a helicopter using etorphine HCl 4.5 (3.0–5.5) mg (M99, Novartis, Kempton Park 1619 South Africa), azaperone 80 (60–80) mg (Stressnil, Janssen Pharmaceutical Ltd., Halfway House 1685 South Africa), and hyaluronidase 2,500 (2,500–5,000) IU (Hyalase, Kyron Laboratories, Benrose 2011 South Africa). Global Positioning System coordinates marked the distance each rhinoceros moved from the time the animal started running to recumbency. As soon as the rhinoceros could be approached, it was sprayed with water to assist with thermoregulation. The first rhinoceros captured was assigned to the posture into which it fell (sternal or lateral recumbency), and subsequent animals were assigned to alternating postures, thus ensuring similar numbers of animals in the sternal and lateral treatment groups; when necessary, the animal was rolled into the assigned posture soon after it could be approached. Prior to data collection, animals were allowed to stabilize for a minimum of 10 min without further change in posture. Inspection of previously collected data suggested that any systematic differences between right lateral and left lateral recumbency in $P_{\dot{E}}CO_2$ were much smaller than differences between lateral and sternal recumbency;[17] with this in mind and to have sufficient numbers for meaningful statistical analysis, data were pooled from all animals in lateral recumbency a priori. After all data collection and management procedures were completed, the etorphine was specifically antagonized with a mixture of naltrexone and diprenorphine by intravenous injection. All animals recovered satisfactorily.

Measurements

Environmental temperature and atmospheric pressure were measured at the capture site (Kestrel® 4300 Weather Meter, Nielsen-Kellerman, Boothwyn, Pennsylvania 19061, USA). Cuffed tubes were placed inside each nostril (26 mm ID; SurgiVet, Smiths Medical North America, Dublin, Ohio 43017, USA) and attached to a purpose-built system of polyvinyl chloride tubes, one-way valves, and two 200-L collection bags (Fig. 1). Expired gas was collected for 2 min, while the respiratory rate was counted. At the end of this period, the collecting system was disconnected from the animal and sealed. A sampling tube was placed inside the nasal passage (VitaLine™ H Set, Oridion Capnography Inc., Bedford, Massa-

chusetts 01730, USA), and $P_{\dot{E}}CO_2$ was measured during expired gas collection using a capnograph designed for side-stream sampling (Microcap® Plus, Oridion Capnography Inc.). Mixed expired carbon dioxide pressure ($P_{\dot{E}}CO_2$) was measured from a small volume aspirated (at 50 ml/min) from the collection bags with the Microcap Plus. The expired gas volume was measured by completely expressing the gas from the collection bags through a respirometer (Wright's Mark 8 Respirometer, Ferraris Medical, Holland, New York 14080, USA) that was calibrated (RT-200 Calibration Analyzer, Timeter Instrument Corp., St. Louis, Missouri 63110, USA). The temperature of the gas leaving the respirometer was measured with a weather meter (Kestrel 4300).

Equipment for measuring body weight was unavailable; however, a flexible rule was used to measure the distance between the most caudal aspect of the occiput and the base of the tail (spine length). Rectal temperature was measured with a thermistor (Fluke Corporation, American Fork, Utah 84003, USA) during collection of the expired gas. Blood was collected from an auricular artery during or at the end of gas collection using a vented syringe (Smiths Medical Pro-Vent® Plus Arterial Blood Gas Sampling Kit, Fisher HealthCare, Houston, Texas 77038, USA); arterial respiratory gas partial pressures (P_aO_2, P_aCO_2), and acid-base variables, including lactate, were measured immediately (VetScan i-STAT® 1, Handheld Clinical Analyzer, Abbott Laboratories, Abbott Park, Illinois 60064, USA). Values for P_aO_2, P_aCO_2, and pH were corrected to rectal temperature, and oxygen saturation of the arterial blood (S_aO_2) at rectal temperature was calculated from the hemoglobin/oxygen dissociation curve.[1,13]

Analyses

Alveolar oxygen pressure (P_AO_2) was calculated from the alveolar gas equation (1)

$$P_AO_2 = 0.21(P_{atm} - P_{H_2O}) - (P_aCO_2/R), \quad (1)$$

where P_{atm} was the atmospheric pressure, P_{H_2O} was the saturated water vapor pressure at the rectal temperature, and R was the respiratory quotient (in the absence of a value for this species, an R value of 0.8 was assumed). Alveolar to arterial oxygen pressure difference ($P_{A\text{-}a}O_2$) and arterial to end-tidal carbon dioxide pressure difference ($P_{a\text{-}\dot{E}}CO_2$) were then calculated. Tidal volume (VT) was calculated by dividing expired minute ventilation (VE) by the breathing rate. VE and VT were corrected to body temperature, atmospheric pressure, and for saturation with water vapor. Total dead space ratio (VD/VT_{TOT}) was calculated by applying the Enghoff modification of the Bohr equation (2):

$$VD/VT_{TOT} = (P_aCO_2 - P_{\dot{E}}CO_2)/P_aCO_2. \quad (2)$$

Total dead space (VD_{TOT}) (comprised of both mechanical dead space [VD_{MECH}] and physiologic dead space [VD_{PHYS}]) was the product of VT and VD/VT_{TOT}. VD_{MECH} measured in the collection system was 4.5 L; this was allowed for in calculating VD_{PHYS} by applying the formula by Singleton et al., where $P_{\dot{E}}CO_2$ was used to estimate P_ACO_2 (3):[22]

$$VD_{PHYS} = VD_{TOT} - VD_{MECH} \times (P_ACO_2/P_aCO_2)^2. \quad (3)$$

VD/VT_{PHYS} was determined by dividing VD_{PHYS} by VT. Alveolar ventilation per breath (VA/breath) was calculated by subtracting VD_{PHYS} from VT, and VA/min was calculated by multiplying VA/breath by breathing frequency.

The significance of differences between groups for environmental temperature, atmospheric pressure, time to recumbency, distance run, rectal temperature, spine length, and time in posture before sampling were tested using Student's *t*-test. All remaining data were analyzed by descriptive and inferential methods using statistical software (SPSS v.20, IBM SPSS Inc., IBM Corporation, Somers, New York 10589, USA). Descriptive statistics and tests for normality were performed in a series of univariate analyses. Continuous normally distributed data were described by mean (95% confidence limits), and continuous non-normal variables were described by median (first and third quartile) values. To test which predictor variables were associated with dead space (total and physiologic), a multivariable general linear model analysis was performed with dead space as the dependent variable. To minimize variability related to rhinoceros size, measures of ventilation were normalized to body size by controlling for spine length (meters) as a covariate in multivariate models (i.e., analysis of covariance [ANCOVA], where breathing rate, VE, VT, VD/VT_{TOT}, VD/VT_{PHYS}, VD_{TOT}, VD_{PHYS}, VA/breath, and VA/min were the dependent variables, respectively). The creation of an index or ratio to normalize physiologic data for variation in body size has fallen into disfavor because of the tendency of such ratios to introduce bias and mislead researchers. Therefore, variation in animal body size was controlled for in the model by including

Table 1. Measurements from rhinoceroses positioned in lateral or sternal recumbency after capture with etorphine, azaperone, and hyaluronidase. Significance was tested by ANCOVA controlling for spine length. Data are reported as marginal means (95% confidence limits) at average spine length of 2.29 m for normally distributed data or as medians (first quartile; third quartile) for data that are not normally distributed.

Variable[a]	Lateral (n = 11)	Sternal (n = 10)	*P* value[b]
pH	7.27 (7.24, 7.30)	7.31 (7.27, 7.34)	0.02[c]*
Lactate (mmol/L)	7.9 (6.3, 9.4)	2.9 (1.2, 4.5)	<0.001*
P_aCO_2 (mm Hg)	50 (48, 52)	53 (51, 56)	0.07
P_aO_2 (mm Hg)	41 (37, 46)	56 (52, 61)	<0.001*
S_aO_2 (%)	62 (57, 67)	82 (77, 88)	<0.001*
$P_{\dot{E}}CO_2$ (mm Hg)	36 (34, 38)	46 (44, 49)	<0.001*
$P_{\dot{E}}CO_2$ (mm Hg)	22 (20, 24)	26 (24, 27)	0.006*
P_AO_2 (mm Hg)	66 (63, 70)	62 (59, 66)	0.09
$P_{A\text{-}a}O_2$ (mm Hg)	26 (20, 29)	2 (0, 10)	<0.001[c]*
$P_{a\text{-}\dot{E}}CO_2$ (mm Hg)	14 (12, 16)	7 (5, 9)	<0.001*
Rectal temperature (°C)	38.2 (37.8, 38.7)	37.9 (37.4, 38.4)	0.37

[a] P_aCO_2 and P_aO_2, arterial respiratory gas partial pressures; S_aO_2, oxygen saturation of the arterial blood; $P_{\dot{E}}CO_2$, end-expired carbon dioxide partial pressure; $P_{\dot{E}}CO_2$, mixed expired carbon dioxide partial pressure; P_AO_2, alveolar oxygen partial pressure; $P_{A\text{-}a}O_2$, alveolar to arterial oxygen partial pressure difference; $P_{a\text{-}\dot{E}}CO_2$, arterial to end-tidal carbon dioxide partial pressure difference.

[b] * significant difference at $P < 0.05$.

[c] Modeled by ANCOVA on ranks controlling for spine length due to non-normality.

spine length as a covariate rather than creating a size-specific index.[18] ANCOVA was applied to raw data that were normally distributed as appropriate; while ANCOVA on rank-transformed variables was applied to data that were not normally distributed. Significance was set at $P \leq 0.05$.

RESULTS

Immobilizations were performed in an environmental temperature of 27.8 ± 2.8°C (mean ± standard deviation, pooled data) and an atmospheric pressure of 665 ± 5 mm Hg. In lateral recumbency, there were seven females and four males; in sternal recumbency, there were five females and five males. There was no difference between groups in the time from darting to becoming recumbent (lateral 4.4 ± 1.2 min; sternal 6.3 ± 4.2 min; $P = 0.2$) nor the distance that rhinoceroses traveled from the time the animal started running to becoming recumbent (lateral 918 ± 604 m; sternal 903 ± 495 m; $P = 0.96$). Rectal temperature was similar in animals between postures (lateral 38.3 ± 0.8°C; sternal 37.9 ± 0.7°C; $P = 0.24$) as was spine length (lateral 230 ± 10 cm; sternal 231 ± 11 cm; $P = 0.24$). Rhinoceroses were in lateral recumbency longer than they were in sternal recumbency before sampling was started (27.1 ± 8.9 min, and 16.5 ± 9.5 min, respectively; $P = 0.02$).

The rhinoceroses in both groups were hypoxemic and had lactic and respiratory acidemia (Table 1). Nevertheless, the animals in lateral recumbency were more acidemic, had higher lactate, greater $P_{A\text{-}a}O_2$ gradients, and lower arterial oxygen that those in sternal recumbency; however, P_aCO_2 and P_AO_2 were similar between groups. Both $P_{\dot{E}}CO_2$ and $P_{\dot{E}}CO_2$ were lower in lateral recumbency than sternal recumbency, while $P_{a\text{-}\dot{E}}CO_2$ was higher. Time in posture was not significantly associated with dead space in the multivariate ANCOVA.

Although there was no difference in tidal volume between postures, breathing rate and minute ventilation were greater (22 and 38%, respectively) in lateral than in sternal recumbency (Table 2). Total dead space ratio was not significantly different between treatment groups; however, physiologic dead space ratio and physiologic dead space volume were both greater in lateral recumbency (89 and 128%, respectively; Table 2). Minute ventilation increased at 141 L/m of spine length ($\beta = 141.43$, $P = 0.03$). No difference in alveolar ventilation was found between the two postures.

DISCUSSION

The nature of their habitat requires that free-ranging rhinoceroses are usually captured by darting from a helicopter. This process inevitably induces an extreme flight response, and this, in turn, produces exertion-related physiologic perturbations (e.g., increased metabolic oxygen demand, hypoxemia, hypercapnea, metabolic and

Table 2. Measurements from rhinoceroses positioned in lateral or sternal recumbency after capture with etorphine, azaperone, and hyaluronidase. Abbreviations for variables are defined in the text. Significance was tested by ANCOVA controlling for spine length. Data are reported as marginal means (95% confidence limits) at average spine length of 2.29 m for normally distributed data or as medians (first quartile; third quartile) for data that are not normally distributed.

Variable[a]	Lateral (n = 11)	Sternal (n = 10)	*P* value[b]
Breathing rate (per minute)	8.4 (7.6, 9.2)	6.9 (6.0, 7.7)	0.02*
VE (L/min)	96.8 (79.8, 113.7)	69.9 (52.1, 87.8)	0.04*
VT (L)	11.7 (9.5, 13.9)	10.4 (8.1, 12.6)	0.39
VD/VT_{TOT} (%)	58 (54, 59)	51 (46, 57)	0.07[c]
VD/VT_{PHYS} (%)	32 (24, 41)	17 (8, 26)	0.02*
VD_{TOT} (L)	6.6 (5.4, 7.9)	5.3 (4.0, 6.6)	0.16
VD_{PHYS} (L)	4.2 (2.8, 5.7)	1.9 (0.4, 3.4)	0.03*
VA/breath (L/breath)	6.4 (5.9, 9.1)	8.0 (7.5, 10.2)	0.13[c]
VA/min (L/min)	54.5 (53.0, 73.0)	57.9 (54.7, 64.2)	0.87[c]

[a] VE, minute ventilation; VT, tidal volume; VD/VT_{TOT}, total dead space ratio; VD/VT_{PHYS}, physiologic dead space ratio; VD_{TOT}, total dead space; VA, alveolar ventilation.

[b] * significant difference at $P < 0.05$.

[c] Modeled by ANCOVA on ranks controlling for spine length due to non-normality.

respiratory acidemia, lactic acidemia, hyperthermia, and electrolyte abnormalities). It is likely that recovery from these perturbations is, at best, delayed and is, at worst, exacerbated by the drugs used for capture and by mechanical impairment of ventilation imposed by recumbency. Hence, the hypercapnea and hypoxia that was observed might be the result of a complex interaction of exertional, drug, and postural effects. Previous observations suggested that manipulation of posture might be used to mitigate some of the adverse pathophysiologic consequences of capture in free-ranging black rhinoceroses. This study was designed to investigate that postulate.

Animals recovering from exertion have several factors that encourage hyperpnea, including oxygen debt, acidemia, and hyperthermia. The respiratory rate of anesthetized black rhinoceroses in this study was less than half of nonanesthetized white rhinoceroses (*Ceratotherium simum*).[5] These black rhinoceroses had VE and VT values consistent with those observed by others under similar circumstances after capture (Bush and Citino, unpub. data). It is remarkable that both VE and VT are less than those observed in adult Thoroughbred horses after exertion, even though horses weigh approximately half of these adult rhinoceroses. Although this may reflect a physiologic difference between species or an impediment due to recumbency in the immobilized rhinoceros, or some other factor, the lower volumes observed in rhinoceroses are probably attributable to the potent etorphine used for rhinoceros capture. Opioids, such as etorphine, act via μ and δ opioid receptors to reduce central respiratory drive and decrease the sensitivity of chemoreceptors to increased carbon dioxide. Opioids can also increase chest wall rigidity and upper airway resistance. Alone, they have been implicated in hypoventilation, hypoxemia, and increased physiologic dead space.[28]

The data presented here confirm earlier observations that P_ECO_2 is lower in lateral recumbency than it is in sternal recumbency in recently captured black rhinoceroses.[17] Theoretically, this could be due to greater alveolar ventilation in lateral recumbency compared with sternal recumbency. However, in previous observations, P_ECO_2 changed to a new plateau within 2 min after a change in posture; this rapid change to a new plateau is unlikely to reflect a change in alveolar ventilation. Although greater minute ventilation was observed in lateral recumbency, alveolar ventilation was not correspondingly greater, nor was P_aCO_2 correspondingly less, in this posture. Hence, the lower P_ECO_2 seen in anesthetized rhinoceroses in lateral recumbency cannot be attributed to superior alveolar ventilation.

It was hypothesized that the likely cause for the lower P_ECO_2 in lateral recumbency is greater physiologic dead space ventilation in this posture. Dead space ventilation is that part of expired air that has not participated in gas exchange and, therefore, has had no carbon dioxide added to it; hence it dilutes the carbon dioxide added to that fraction of tidal breath that has participated in gas exchange. Physiologic dead space in lateral recumbency was more than twice that in sternal recumbency; this is consistent with the hypothesis

that the lower $P_{\dot{E}}CO_2$ in lateral recumbency represents greater dead space ventilation.

Physiologic dead space is composed of anatomic dead space and alveolar dead space. Anatomic dead space is roughly that part of the tidal volume that remains in conducting airways (nasal cavity, larynx, trachea, bronchi, and bronchioles). Alveolar dead space is that part of the tidal volume that ventilates alveoli that are not perfused or that have suboptimal perfusion (ventilation:perfusion ratio >1). It is unlikely that anatomic dead space will change substantially with posture. On the other hand, increased alveolar dead space can be caused by various factors, such as a decrease in overall pulmonary blood flow (i.e., decreased cardiac output), redistribution of pulmonary blood flow away from ventilated alveoli, and regional changes in chest wall movement; all of these factors could be influenced by the posture into which a rhinoceros is positioned.[8,16] Hence, it is postulated that higher alveolar dead space in lateral recumbency accounts for the majority of the higher physiologic dead space and lower $P_{\dot{E}}CO_2$ seen in this posture. Alveolar dead space fraction has been calculated using a modification of the Bohr equation where $P_{\dot{E}}CO_2$ is substituted for $P_{E}CO_2$.[21] This method was not applied to the data because it may systematically overestimate alveolar dead space when alveoli empty nonuniformly, as is likely in these large animals in the period after exertion.[2,26] Nevertheless, using the mean data in Table 1 gives estimates of the alveolar dead space ratio of 27.5% for rhinoceroses in lateral recumbency and 13.0% in sternal recumbency; this is consistent with greater alveolar dead space in lateral recumbency in these rhinoceroses.

The higher breathing rate and minute ventilation observed in lateral recumbency is compatible with anecdotal reports that rhinoceros appear to breathe better in this posture. However, the data show that the apparently superior ventilation in lateral recumbency is caused by an increase in dead space ventilation and not alveolar ventilation. Hence, the practice of moving a recently captured rhinoceros from sternal to lateral recumbency is unlikely to improve their ability to eliminate carbon dioxide. In quadrupeds that are standing or in sternal recumbency, each hemithorax moves laterally to an approximately similar extent during inspiration. When such animals lie on their side, the dependent chest wall is fixed by the ground; under these circumstances, ordinary thoracic wall movement during inspiration displaces the whole chest upward, and the movement of the upper hemithorax may appear to be increased. This may explain the suggestion that tidal volume is greater in laterally recumbent rhinoceroses; an increase in tidal volume could not be measured in the rhinoceroses in lateral posture.

VT and P_aCO_2 were similar in both postures, despite the greater VE and physiologic dead space in lateral recumbency. The extra dead space ventilation in lateral recumbency could be viewed as an energetic waste. However, dead space ventilation can be important for dissipating excess body heat; nevertheless, because rectal temperature was not lower in the rhinoceroses in lateral recumbency, no evidence supports greater dead space being advantageous to the animals in this regard. It is possible that rectal temperature would have declined faster in the animals in lateral recumbency had data been collected for a longer period of time.

The values for VD/VT_{PHYS} are lower than those reported in nonanesthetized, standing horses, and cows (*Bos primigenius*)[7] but consistent with those reported in anesthetized horses. Rainger et al.[20] did not observe differences in dead space with positioning in lateral or dorsal recumbency; however, those horses were mechanically ventilated, while the rhinoceroses in the present study were breathing spontaneously.

Both treatment groups were hypoxemic based on normal values for other mammals; nevertheless, the rhinoceroses in lateral recumbency were significantly more hypoxemic than those in sternal recumbency. Hypoxia is particularly important because it is likely to contribute to postcapture myopathy, which is a cause of mortality in rhinoceroses and other large ungulates.[15] Field observations of immobilized rhinoceroses demonstrate hypoxemia as a predominant finding, with hypoventilation as a presumptive cause.[3,6,14] Hypoventilation leads to carbon dioxide displacing oxygen from the alveolar gas; if these rhinoceroses had been normocapnic, by using the pooled data from Table 1 and substituting $P_aCO_2 = 40$ mm Hg in the alveolar gas equation, the P_AO_2 would be substantially increased to ~79 mm Hg for both treatment groups. This suggests that hypoventilation contributed to the hypoxemia of both groups but cannot explain the inferior arterial oxygen of those in lateral recumbency. Elevation above sea level and the associated reduction in inspired oxygen partial pressure will reduce oxygen tension in the blood. At an altitude of ~1,000 m and with an average barometric pressure of 665 mm Hg, the inspired PO_2 of the

rhinoceroses in this study was ~20 mm Hg less than at sea level. If the rhinoceroses had been at sea level, substitution in the alveolar gas equation suggests that P_AO_2 would have been 86 mm Hg for those in lateral recumbency and 83 mm Hg for those in sternal. Consequently, it should be anticipated that P_aO_2 and S_aO_2 would be reduced from that expected at sea level. However, the lower oxygen environment was common to all subjects in this study, hence, it could not explain the differences in oxygenation associated with posture.

Arterial oxygen (P_aO_2 and S_aO_2) was lower in the rhinoceroses in lateral recumbency than in those in sternal recumbency. This was associated with a larger alveolar-arterial oxygen pressure gradient in lateral recumbency; although pulmonary shunt fraction was not measured in this study, this finding is compatible with substantially greater pulmonary venous admixture in this posture. Pulmonary venous admixture represents venous blood that passes through the lung without participating in gas exchange and could contribute to the arterial hypoxemia observed in both groups of rhinoceroses. Pulmonary venous admixture can be caused by impeded gaseous diffusion across the alveolar membrane; spatial mismatching of ventilation to perfusion in the lung (specifically areas of lung with V/Q in the range of <1 to >0); true shunt through collapsed alveoli (with $V/Q = 0$) or through anastomotic vessels. Mild, transient, interstitial pulmonary edema, secondary to pulmonary hypertension, has been suggested as a cause of impeded oxygen diffusion across the alveolar membrane and, thus, arterial hypoxemia during exertion in racehorses.[23,27] Without measurements of oxygen diffusion rate and pulmonary artery pressure, it is undetermined whether this mechanism contributed to the hypoxia in these rhinoceroses; however, because the animals in both groups traveled similar distances over similar time periods prior to recumbency, the level of exertion (and any pulmonary edema-induced hypoxemia) should have been similar among all subjects. Hypoxic pulmonary vasoconstriction is a physiologic mechanism that minimizes spatial mismatching of ventilation to perfusion in the lung by regulating vascular tone to direct flow away from poorly ventilated regions of lung; although inhaled agents can obtund hypoxic vasoconstriction, the drugs used in this study are unlikely to affect it.[24] At maximal exertion, the pulmonary vascular tree of horses is thought to be maximally dilated;[27] this suggests that hypoxic pulmonary vasoconstriction is superseded to accommodate the increase in cardiac output that occurs with exertion; however, exertion may also be associated with reduced transit time for red cells in the lung and reduced time for the hemoglobin to acquire oxygen in the pulmonary bed. It is not known whether alterations in hypoxic vasoconstriction pertain to the hypoxemia in the rhinoceroses in this study; however, it is unlikely that perturbation of hypoxic vasoconstriction could explain the differences in arterial oxygen observed between postures.

If the hypoxemia is due to diffusion limitation or mismatching of ventilation to perfusion (but not true shunt), then it will respond to supplementation of inspired oxygen. The fraction of oxygen inspired was not supplemented in this study. However, the practice of increasing the inspired oxygen fraction by insufflation of the upper airway with oxygen may be beneficial during immobilization in this species, regardless of posture, and others have shown that oxygen insufflation of the upper airways increases arterial oxygen tension in recently captured white rhinoceroses.[4] Should oxygen supplementation not be available, this study suggests that sternal recumbency may be preferred to facilitate oxygenation.

Plasma lactate concentration was greater in lateral recumbency. Lactate was probably produced by anaerobic metabolism in muscle prior to capture if oxygen demand exceeded oxygen supply as the animal ran to avoid the helicopter. There is no evidence to suggest the animals in lateral recumbency had exerted themselves more during capture than those in sternal recumbency; hence, the higher plasma lactate concentration in lateral can be attributed to the more severe hypoxemia in this posture causing quantitatively more ongoing anaerobic metabolism after the animal goes down. However, the possibility cannot be excluded that reduced lactate clearance (e.g., due to inferior liver perfusion) in lateral recumbency contributes to higher lactate.

This study was incorporated into the conservation management activities of the MET; this included placement of radiotelemetry transmitters in the horn of rhinoceroses in lateral recumbency. In turn, this resulted in the animals in lateral recumbency being in that position for significantly longer before data collection than those in sternal recumbency. It is possible that this confounding factor biased the data. Indeed, in anesthetized horses, the dead space ratio increased with duration of procedure;[20] however, the rise in dead space was observed only after 60 min, and all of the rhinoceroses in this study were

recumbent for periods of less than 1 hr. Nevertheless, to determine if the time an animal was in posture prior to data collection could have influenced the data, the time in posture variable was analyzed in the regression model. Time in posture showed no correlation with dead space in the multivariate ANCOVA. These findings suggest that time in posture prior to collection of data does not account for the observed differences in dead space. The respiratory quotient of herbivores is usually considered to be >0.8 and <0.9. In the absence of a particular value for rhinoceroses or other large herbivores, it was determined to use 0.8 in the alveolar gas equation; if 0.9 was used, substitution in the alveolar gas equation using pooled values from Table 1 gives P_AO_2 values of 73 mm Hg for the animals in lateral recumbency and 70 mm Hg for those in sternal recumbency. These values would cause an apparent increase in $P_{A\text{-}a}O_2$ but would not account for the difference between postures in arterial oxygenation.

CONCLUSIONS

Capture-related mortality in rhinoceroses is likely to be related to cardiopulmonary derangements, which might be influenced by positioning. The current practice of many field veterinarians is to position recently captured black rhinoceroses in lateral recumbency because they appear to breath better in this position than they do in sternal recumbency. The present study's data confirm that minute ventilation is, indeed, superior in lateral recumbency. Nevertheless, these data also show that the fraction of dead space is greater in this position so that the greater ventilation in lateral recumbency is entirely wasted in dead space. Thus, lateral recumbency offers no advantage over sternal recumbency in terms of alveolar ventilation and P_aCO_2.

Data from this study also show that P_aO_2 and oxygen saturation of arterial hemoglobin are greater in sternal recumbency than in lateral recumbency, suggesting that oxygenation of tissues would be better in this posture. Because neither posture is preferable from the point of view of alveolar ventilation but sternal recumbency produces superior arterial oxygen, one might suggest that sternal recumbency is preferable to lateral recumbency in recently captured black rhinoceroses. However, because tissue oxygen delivery is the product of arterial oxygen content and blood flow, such a conclusion is premature until data are available showing that regional blood flow and net oxygen delivery to the muscles of locomotion are not disadvantaged in rhinoceroses in sternal recumbency.

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