TREATMENT OF HYPOXEMIA DURING ANESTHESIA OF BROWN BEARS (URSUS ARCTOS)

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Abstract: This study assessed whether arterial oxygenation could be increased by treatment with intranasal oxygen supplementation in brown bears (*Ursus arctos*) with hypoxemia during anesthesia with medetomidine-zolazepam-tiletamine. Arterial blood samples were collected anaerobically from the femoral artery before and during oxygen supplementation. An oxygen flow rate of 2–5 L/min administered intranasally to brown bears weighing 12–120 kg markedly increased arterial oxygenation. Intranasal oxygen supplementation proved to be a simple and efficient method for treatment of hypoxemia in anesthetized bears.

Key words: Arterial blood gases, brown bear, hypoxemia, immobilization, oxygen supplementation, Ursus arctos.

BRIEF COMMUNICATION

Both free-ranging and captive brown bears (*Ursus arctos*) anesthetized with medetomidinezolazepam-tiletamine (MZT) at different doses commonly develop mild to marked hypoxemia, as detected by arterial blood gas analysis.⁵ Hypoxemia can lead to insufficient oxygen delivery and tissue hypoxia, which can rapidly cause cell damage in sensitive organs. During anesthesia of wild animals, hypoxemia is often not treated, or not even recognized,¹⁰ even though hypoxemia can result in brain cell death, myocardial ischemia, and multiorgan damage. Because the consequences of hypoxemia can be difficult to measure, a negative effect on an organ system does not have to be proven before therapy can be instituted.⁸ If the potential for improving overall organ function outweighs the risks and disadvantages of the therapy, it should be given strong consideration.⁸ If arterial oxygenation is evaluated only by pulse oximetry (SpO₂), hypoxemia can be missed, as shown in polar bears (Ursus maritimus) anesthetized with zolazepamtiletamine.² For example, despite excellent SpO₂ values of 99% and 98% in one polar bear, the concurrent partial pressures of arterial oxygen (PaO₂) were 61 and 55 mmHg, respectively.² Arterial blood gas analysis is a valuable tool for assessment of ventilation, and portable analyzers also enable measurement in the field. The objective of this study was to assess whether arterial oxygenation could be increased by treatment with intranasal oxygen supplementation in brown bears with hypoxemia during anesthesia with MZT.

In April 2006 and 2007, oxygen supplementation was provided to nine brown bears (two captive, seven free-ranging) that were part of a larger study on physiologic evaluation of capture and anesthesia in brown bears.⁵ Captive bears were darted while in their indoor quarters at the zoo; free-ranging bears were darted from a helicopter. For anesthesia, medetomidine at 0.02–0.14 mg/kg (Domitor[®] vet., 1 mg/ml, or Zalopine, 10 mg/ml, Orion Pharma Animal Health, Espoo, Finland) was used in combination with zolazepam-tiletamine at 1.9–6.9 mg/kg (Zoletil forte vet., Virbac S.A., Carros, France). A detailed description of capture methods, drug doses, monitoring, and arterial blood gas analysis

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Age, sex, body mass ^c	Body position ^c	O ₂ flow rate (L/min)		SpO ₂ (%)	$\mathrm{SaO}_2~(\%)^d$	PaO ₂ (mmHg) ^d	$PaCO_2 \ (mmHg)^d$
Captive							
Subadult ♂ 87 kg	RL RL	5	Pre-O ₂ 18 min of O ₂	NR° NR	84 100	55 192	43 45
Subadult Q 62 kg	RL RL	5	Pre-O ₂ 2 min of O ₂ 32 min of O ₂	NR 87 89	86 98 100	58 113 306	46 47 50
Free-ranging							
Yearling ♀ 19 kg	LL LL	2	Pre-O ₂ 8 min of O ₂	NR 93	77 100	55 216	45 49
Yearling ♀ 15 kg	dorsal LL	2	Pre-O ₂ 25 min of O ₂	91 94	83 100	59 290	48 51
Yearling Q 12 kg	RL RL RL	2	Pre-O ₂ 14 min of O₂ 11 min post-O ₂	82 86 88	71 99 81	47 165 55	43 46 49
Adult ♀ ^ь 72 kg	dorsal dorsal	2	Pre-O ₂ 16 min of O ₂	90 83	78 98	58 133	48 47
Adult Q 120 kg	RL dorsal LL	2	Pre-O ₂ 16 min of O₂ 10 min post-O ₂	NR NR 84	85 97 91	59 109 70	39 46 41
Adult Q ^b 93 kg	RL/dorsal RL dorsal	2	$\begin{array}{c} \text{Pre-O}_2 \\ \textbf{12 min of O}_2 \\ \text{25 min post-O}_2 \end{array}$	84 86 91	82 97 90	63 124 77	40 49 43
Subadult ♀ ^ь 65 kg	RL RL	2	$\frac{\text{Pre-O}_2}{25 + 15 \min^{\text{f}} \text{ of }}$	NR 95	91 96	88 110	38 44

 a SpO₂, hemoglobin oxygen saturation measured by pulse oximetry; SaO₂, arterial hemoglobin oxygen saturation (calculated value); PaO₂ and PaCO₂, partial pressures of arterial oxygen and carbon dioxide, respectively (measured values, temperature corrected). Boldface indicates values during oxygen supplementation.

 $^{\rm b}$ Hyperthermia, rectal temperature $>40.0^{\circ}{\rm C}.$

^cRL, right lateral; LL, left lateral.

^d Increased significantly after oxygen supplementation (P < 0.009).

 $^{\rm e}\,NR,$ not recorded because the pulse oximeter failed to produce a reading.

^fOxygen supplementation was discontinued after 25 min for a short period because of procedures performed during anesthesia.

have been previously described.5 Intranasal oxygen was administered for treatment of hypoxemia (PaO₂ range 47–63 mm Hg) in two captive and six free-ranging bears. In addition, a presumably normoxemic free-ranging bear (PaO₂ 88 mm Hg) received intranasal oxygen because of hyperthermia (rectal temperature > 40° C). Age, sex, and body mass of the bears are presented in Table 1. Oxygen was provided from a portable cylinder via a tube that was inserted approximately 2 cm into the nasal cavity of the bears. An oxygen flow rate of 5 L/min was administered to the two captive brown bears (Table 1). The remaining bears received a flow rate of 2 L/min. Arterial blood samples were collected anaerobically from the femoral artery before and during oxygen supplementation. In three bears, arterial blood samples were also collected after oxygen supplementation was discontinued. The samples were processed immediately in the field with the use of a portable analyzer and cartridges (i-STAT[®] 1 Portable Clinical Analyzer and i-STAT® cartridges CG4+ and 6+, Abbott Laboratories, Abbott Park, Illinois 60064, USA). The analysis included measured values for partial pressures of arterial oxygen (PaO₂) and arterial carbon dioxide (PaCO₂) and calculated values for arterial hemoglobin oxygen saturation (SaO₂). Blood gas values were corrected to the rectal temperature. SpO₂ was monitored with the pulse oximeter probe attached to the tongue (Nellcor NPB-40 Handheld Pulse Oximeter, Nellcor Inc., Pleasanton, California 94588, USA). Data from arterial blood samples were treated nonparametrically because of the low animal number and assessed by the Wilcoxon signed-rank test in Minitab[®] 15 Statistical Software (Minitab Inc., State College, Pennsylvania 16801, USA). Differences were considered significant at p < 0.05.

Oxygen supplementation markedly improved the PaO₂ and SaO₂ in hypoxemic brown bears (Table 1). The small but statistically significant increase in PaCO₂ was probably unrelated to oxygen supplementation because a similar increase was noted in anesthetized brown bears that did not receive oxygen.⁵ Because hypoxemia can occur at any time during anesthesia⁵ and recur if oxygen supplementation is discontinued, it is essential to provide oxygen continuously throughout anesthesia. During field work in remote settings, it is desirable to give the minimum effective flow rate to prolong the life of the oxygen cylinder. Oxygen was initially administered at a flow rate of 5 L/min, but because the PaO₂ increased well above the assumed normal range of 80-100 mm Hg, the flow rate was decreased for subsequent bears. A flow rate of 2 L/min, administered to brown bears weighing 12-120 kg, was sufficient to increase the PaO₂ above 100 mm Hg. Optimal oxygen flow rates for different sizes of bears remain to be determined.

The only study available reporting oxygen supplementation in bears used pulse oximetry as the objective measurement for hypoxemia and effect of treatment.¹ Although pulse oximetry has been recommended to adjust the flow rate when supplementing bears with oxygen,³ it might not reliably indicate hypoxemia or normoxemia. In the present study, despite a $PaO_2 > 100 \text{ mm Hg}$ and calculated hemoglobin oxygen saturation values (SaO₂) \geq 96%, pulse oximetry-derived saturation was <90% in five of eight comparable measurements during oxygen supplementation (Table 1). Similar inconsistent pulse oximetry values have been reported in immobilized whitetailed deer (Odocoileus virginianus) supplemented with oxygen9 and in critically ill humans under intensive care.11 Pulse oximetry requires adequate perfusion at the probe site, and the accuracy of the readings can be disturbed by a reduced peripheral blood flow because of vasoconstriction, hypotension, hypovolemia, and hypothermia.7 In addition, the accuracy can vary between different pulse oximeters, probe sites, and species.^{7,11} The tendency for pulse oximetry to underestimate saturation at high ranges of SaO₂ and to overestimate saturation at lower ranges of SaO_2 can lead to a significant risk of undiagnosed hypoxemia and makes it unsuitable as the sole monitor of oxygenation.

Because of a high risk of hypoxemia developing during anesthesia with MZT in free-ranging as well as captive brown bears, even though lower drug doses can be used in captivity,⁵ oxygen supplementation is recommended to improve safety for the animals. Free-ranging bears darted from a helicopter commonly develop hyperthermia,⁵ which increases oxygen consumption.⁶ This is an additional reason why oxygen supplementation should be administered. Furthermore, nasal insufflation with oxygen is a safe and simple method to protect the brain against hyperthermal damage.⁴

Intranasal oxygen supplementation proved to be a simple and effective technique for treatment of hypoxemia in brown bears anesthetized with MZT. An oxygen flow rate of 2–5 L/min given to brown bears weighing 12–120 kg markedly improved arterial oxygenation. Oxygen supplementation is strongly recommended to improve safety during anesthesia by prevention or treatment of hypoxemia. Further study is needed to determine optimal flow rates for different sizes of bears.

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