Journal of Zoo and Wildlife Medicine 41(1): 161–164, 2010

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

Åsa Fahlman, D.V.M., Vet. Med. Lic., Ph.D., John Pringle, D.V.M., D.V.Sc., Ph.D., Dipl. A.C.V.I.M., Dipl. E.C.E.I.M., Jon M. Arnemo, D.V.M., Ph.D., Jon E. Swenson, Ph.D., Dr. habil., Sven Brunberg, and Görel Nyman, D.V.M., Ph.D., Dipl. E.C.V.A.A.

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 medetomidine-zolazepam-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 (SpO_2), hypoxemia can be missed, as shown in polar bears (*Ursus maritimus*) anesthetized with zolazepam-tiletamine.[2] For example, despite excellent SpO_2 values of 99% and 98% in one polar bear, the concurrent partial pressures of arterial oxygen (PaO_2) 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

From the Department of Clinical Sciences, Faculty of Veterinary Medicine and Animal Science, Swedish University of Agricultural Sciences, P.O. Box 7054, SE-750 07 Uppsala, Sweden (Fahlman, Pringle); the Department of Pathology and Wildlife Diseases, National Veterinary Institute, SE-751 89 Uppsala, Sweden (Fahlman); the Faculty of Forestry and Wildlife Management, Hedmark University College, NO-2480 Koppang, Norway, and the Department of Wildlife, Fish, and Environmental Studies, Faculty of Forest Sciences, Swedish University of Agricultural Sciences, SE-901 83 Umeå, Sweden (Arnemo); the Department of Ecology and Natural Resource Management, Norwegian University of Life Sciences, NO-1432 Ås, Norway, and the Norwegian Institute for Nature Research, NO-7485 Trondheim, Norway (Swenson); the Scandinavian Brown Bear Research Project, Tackåsen, SE-794 98 Orsa, Sweden (Brunberg); and the Department of Environment and Health, Faculty of Veterinary Medicine and Animal Science, Swedish University of Agricultural Sciences, P.O. Box 234, SE-532 23 Skara, Sweden (Nyman). Correspondence should be directed to Dr. Fahlman (asa_fahlman@hotmail.com).

Table 1. Effects of intranasal oxygen supplementation on pulmonary gas exchange[a] during medetomidine-zolazepam-tiletamine anesthesia of brown bears. Oxygen was administered to these bears because of hypoxemia, hyperthermia, or both.[b]

Age, sex, body mass[c]	Body position[c]	O_2 flow rate (L/min)		SpO_2 (%)	SaO_2 (%)[d]	PaO_2 (mmHg)[d]	$PaCO_2$ (mmHg)[d]
Captive							
Subadult ♂	RL	5	Pre-O_2	NR[e]	84	55	43
87 kg	RL		**18 min of O_2**	**NR**	**100**	**192**	**45**
Subadult ♀	RL	5	Pre-O_2	NR	86	58	46
62 kg	RL		**2 min of O_2**	**87**	**98**	**113**	**47**
			32 min of O_2	**89**	**100**	**306**	**50**
Free-ranging							
Yearling ♀	LL	2	Pre-O_2	NR	77	55	45
19 kg	LL		**8 min of O_2**	**93**	**100**	**216**	**49**
Yearling ♀	dorsal	2	Pre-O_2	91	83	59	48
15 kg	LL		**25 min of O_2**	**94**	**100**	**290**	**51**
Yearling ♀	RL	2	Pre-O_2	82	71	47	43
12 kg	RL		**14 min of O_2**	**86**	**99**	**165**	**46**
	RL		11 min post-O_2	88	81	55	49
Adult ♀[b]	dorsal	2	Pre-O_2	90	78	58	48
72 kg	dorsal		**16 min of O_2**	**83**	**98**	**133**	**47**
Adult ♀	RL	2	Pre-O_2	NR	85	59	39
120 kg	dorsal		**16 min of O_2**	**NR**	**97**	**109**	**46**
	LL		10 min post-O_2	84	91	70	41
Adult ♀[b]	RL/dorsal	2	Pre-O_2	84	82	63	40
93 kg	RL		**12 min of O_2**	**86**	**97**	**124**	**49**
	dorsal		25 min post-O_2	91	90	77	43
Subadult ♀[b]	RL	2	Pre-O_2	NR	91	88	38
65 kg	RL		**25 + 15 min[f] of O_2**	**95**	**96**	**110**	**44**

[a] SpO_2, hemoglobin oxygen saturation measured by pulse oximetry; SaO_2, arterial hemoglobin oxygen saturation (calculated value); PaO_2 and $PaCO_2$, partial pressures of arterial oxygen and carbon dioxide, respectively (measured values, temperature corrected). Boldface indicates values during oxygen supplementation.

[b] Hyperthermia, rectal temperature $> 40.0°C$.

[c] RL, right lateral; LL, left lateral.

[d] Increased significantly after oxygen supplementation ($P < 0.009$).

[e] NR, not recorded because the pulse oximeter failed to produce a reading.

[f] Oxygen 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 (PaO_2 range 47–63 mm Hg) in two captive and six free-ranging bears. In addition, a presumably normoxemic free-ranging bear (PaO_2 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 (PaO_2) and arterial carbon dioxide ($PaCO_2$) and calculated values for arterial hemoglobin oxygen saturation (SaO_2). Blood gas values were corrected to the rectal temperature. SpO_2 was monitored with the pulse oximeter probe attached to the tongue (Nellcor NPB-40 Handheld Pulse Oximeter, Nellcor Inc., Pleasan-

ton, 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 PaO_2 and SaO_2 in hypoxemic brown bears (Table 1). The small but statistically significant increase in $PaCO_2$ 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 anesthesia[5] 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 PaO_2 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 PaO_2 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 mm Hg and calculated hemoglobin oxygen saturation values (SaO_2) ≥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 white-tailed deer (*Odocoileus virginianus*) supplemented with oxygen[9] 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 SaO_2 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.

Acknowledgments: The authors thank the Swedish Environmental Protection Agency, the Norwegian Directorate for Nature Management, and the Michael Forsgren Foundation for generous support of this study. Also, the authors thank helicopter pilot Ulf Grinde at Jämtlands Flyg and the Orsa Bear Park for collaboration. The study was performed within the Scandinavian Brown Bear Research Project, which was supported by the Swedish Environmental Protection Agency, the Norwegian Directorate for Nature Management, the Research Council of Norway, and the World Wide Fund for Nature in Sweden.

LITERATURE CITED

1. Cattet, M. R. L., N. A. Caulkett, and G. B. Stenhouse. 2003. Anesthesia of grizzly bears using xylazine-zolazepam-tiletamine or zolazepam-tiletamine. Ursus 14: 88–93.

2. Cattet, M. R., N. A. Caulkett, K. A. Streib, K. E. Torske, and M. A. Ramsay. 1999. Cardiopulmonary response of anesthetized polar bears to suspension by net and sling. J. Wildl. Dis. 35: 548–556.

3. Caulkett, N. 2007. Bears. *In:* West, G., D. Heard, and N. Caulkett (eds.). Zoo Animal and Wildlife

Immobilization and Anesthesia, 1st ed. Blackwell Publishing Professional, Ames, Iowa. Pp. 409–415.

4. Einer-Jensen, N., K. E. Baptiste, F. Madsen, and M. H. Khorooshi. 2002. Can intubation harm the brain in critical care situations? A new simple technique may provide a method for controlling brain temperature. Med. Hypotheses 58: 229–231.

5. Fahlman, Å. 2008. Advances in Wildlife Immobilisation and Anaesthesia: Clinical and Physiological Evaluation in Selected Species. PhD Thesis, Swedish University of Agricultural Sciences, Uppsala, Sweden. http://diss-epsilon.slu.se/archive/00001908/.

6. Hurst, R. J., N. A. Oritsland, and P. D. Watts. 1982. Body mass, temperature and cost of walking in polar bears. Acta Physiol. Scand. 115: 391–395.

7. Matthews, N. S., S. Hartke, and J. C. Allen, Jr. 2003. An evaluation of pulse oximeters in dogs, cats and horses. Vet. Anaesth. Analg. 30: 3–14.

8. McDonell, W. N., and C. L. Kerr. 2007. Respiratory system. *In:* Tranquilli, W. J., J. C. Thurmon, and K. A. Grimm (eds.). Lumb & Jones' Veterinary Anesthesia and Analgesia, 4th ed. Blackwell Publishing, Ames, Iowa. Pp. 117–151.

9. Mich, P. M., L. L. Wolfe, T. M. Sirochman, M. A. Sirochman, T. R. Davis, W. R. Lance, and M. W. Miller. 2008. Evaluation of intramuscular butorphanol, azaperone, and medetomidine and nasal oxygen insufflation for the chemical immobilization of white-tailed deer, *Odocoileus virginianus*. J. Zoo Wildl. Med. 39: 480–487.

10. Read, M. R., N. A. Caulkett, A. Symington, and T. K. Shury. 2001. Treatment of hypoxemia during xylazine-tiletamine-zolazepam immobilization of wapiti. Can. Vet. J. 42: 861–814.

11. Van de Louw, A., C. Cracco, C. Cerf, A. Harf, P. Duvaldestin, F. Lemaire, and L. Brochard. 2001. Accuracy of pulse oximetry in the intensive care unit. Intensive Care Med. 27: 1606–1613.

Received for publication 25 February 2009