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OXYGEN SUPPLEMENTATION IN ANESTHETIZED BROWN BEARS (*URSUS ARCTOS*)—HOW LOW CAN YOU GO?

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ABSTRACT: Hypoxemia is anticipated during wildlife anesthesia and thus should be prevented. We evaluated the efficacy of low flow rates of supplemental oxygen for improvement of arterial oxygenation in anesthetized brown bears (*Ursus arctos*). The study included 32 free-ranging brown bears (yearlings, subadults, and adults; body mass 12–250 kg) that were darted with medetomidine-zolazepam-tiletamine (MZT) from a helicopter in Sweden. During anesthesia, oxygen was administered intranasally from portable oxygen cylinders at different flow rates (0.5–3 L/min). Arterial blood samples were collected before (pre-O_2), during, and after oxygen therapy and immediately processed with a portable analyzer. Rectal temperature, respiratory rate, heart rate, and pulse oximetry-derived hemoglobin oxygen saturation were recorded. Intranasal oxygen supplementation at the evaluated flow rates significantly increased the partial pressure of arterial oxygen (PaO_2) from pre-O_2 values of 9.1±1.3 (6.3–10.9) kPa to 20.4±6.8 (11.1–38.7) kPa during oxygen therapy. When oxygen therapy was discontinued, the PaO_2 decreased to values not significantly different from the pre-O_2 values. In relation to the body mass of the bears, the following oxygen flow rates are recommended: 0.5 L/min to bears <51 kg, 1 L/min to bears 51–100 kg, 2 L/min to bears 101–200 kg, and 3 L/min to bears 201–250 kg. In conclusion, low flow rates of intranasal oxygen were sufficient to improve arterial oxygenation in brown bears anesthetized with MZT. Because hypoxemia quickly recurred when oxygen was discontinued, oxygen supplementation should be provided continuously throughout anesthesia.

Key words: Anesthesia, brown bear, flow rate, hypoxemia, immobilization, oxygen therapy, wildlife.

INTRODUCTION

Impaired oxygenation during anesthesia can lead to morbidity and mortality during and after the anesthetic event. Most physiologic studies that include blood gas analysis demonstrate hypoxemia with the drugs and doses used for anesthesia in free-ranging and captive wild animals (Read 2003; Mich et al. 2008; Fahlman et al. 2012). Mild to marked hypoxemia has been documented in free-ranging and captive brown bears (*Ursus arctos*) anesthetized with medetomidine-zolazepam-tiletamine (MZT; Fahlman et al. 2011). Hypoxemia is anticipated and thus should be prevented during wildlife anesthesia.

Intranasal oxygen supplementation is a simple and efficient method to increase arterial oxygenation, as documented in some wildlife species (Cattet et al. 2003; Fahlman et al. 2010, 2012). In brown bears, the use of intranasal oxygen at flow rates as high as 6–10 L/min has been reported (Mortenson and Bechert 2001; Cattet et al. 2003). Lower flow rates (2–5 L/min) efficiently treated hypoxemic brown bears on the basis of arterial blood gas analysis (Fahlman et al. 2010), but optimal flow rates to maintain adequate arterial oxygenation have not been established.

Oxygen sources used in the field include oxygen cylinders that store gaseous oxygen under high pressure and portable battery-driven oxygen concentrators that extract nitrogen from the air and produce oxygen on site (Fahlman et al. 2010, 2012). Small light-weight oxygen cylinders are easy to

TABLE 1. Animal data and intranasal oxygen flow rates for 32 free-ranging brown bears (*Ursus arctos*) included in a study on low flow oxygen therapy during anesthesia.

	n[a]			O_2 flow rate	
Age	M	F	Body mass (kg)	L/min	mL/min per kilogram
Adult	11	7	60–250	1–3	9–28
Subadult	1	3	28–65	1–2	16–36
Yearling	6	4	12–25	0.5–2	20–167

[a] n = number of animals; M = male; F = female.

carry during fieldwork, but refilling cylinders may entail complex logistics in remote areas. Thus, administration of the minimum effective flow rate will reduce the number of oxygen cylinders needed and decrease costs. We evaluated the efficacy of low flow rates of intranasal oxygen for improvement of arterial oxygenation in anesthetized brown bears. We also evaluated the effect of discontinued oxygen therapy on arterial oxygenation.

MATERIALS AND METHODS

The study included 32 free-ranging brown bears (Table 1) anesthetized for individual marking for ecologic studies in the Scandinavian Brown Bear Research Project. The brown bears were anesthetized in the county of Dalarna, Sweden (approximately 61°N, 15°E) in April 2006–2012. For anesthesia, medetomidine was used at a mean ± SD (range) dose of 0.10±0.04 (0.05–0.23) mg/kg (Domitor® vet., 1 mg/mL, or Zalopine, 10 mg/mL, Orion Pharma Animal Health, Espoo, Finland) in combination with zolazepam-tiletamine at 4.7±1.6 (2.3–9.1) mg/kg (Zoletil forte vet., Virbac S.A., Carros, France). All animals were darted from a helicopter. A detailed description of the capture method, monitoring, and physiologic responses during anesthesia of brown bears with this drug combination has been published (Fahlman et al. 2011). Approval was given by the Ethical Committee on Animal Experiments in Uppsala, Sweden.

Blood sampling, analysis, and physiologic monitoring

Arterial blood samples were collected anaerobically from the femoral artery before (pre-O_2, n=31), during (n=32), and after (post-O_2, n=11) intranasal oxygen therapy. Time of sampling varied depending on individual procedures in the field. The second sample was collected 18±6 (7–40) min after initiation of oxygen supplementation. The post-O_2 sample was collected 9±6 (3–25) min after discontinued oxygen supplementation and removal of the nasal line(s). All samples were collected in preheparinized syringes, capped, and immediately processed with the use of a portable analyzer and cartridges (i-STAT®1 Portable Clinical Analyzer and i-STAT® cartridges CG4+, Abbott Laboratories, Abbott Park, Illinois, USA). The analyses included measured values for partial pressures of oxygen (PaO_2) and carbon dioxide ($PaCO_2$), pH, and lactate, and a calculated value for arterial hemoglobin oxygen saturation (SaO_2). Rectal temperature, respiratory rate, and heart rate were recorded at the time of arterial sampling, and if feasible, pulse oximetry-derived hemoglobin oxygen saturation (SpO_2) was also recorded. The pulse oximeter probe was attached to the tongue (Nellcor NPB-40 handheld pulse oximeter, Nellcor Inc., Pleasanton, California, USA). Blood gas values were corrected to the rectal temperature, except in one hyperthermic bear that received a cold-water enema, which influenced the accuracy of rectal temperature measurements. For this bear, blood gases and pH at standard temperature (37 C) were used.

Oxygen therapy and target PaO_2

Oxygen was supplemented from portable oxygen cylinders via one or two nasal lines inserted 2–10 cm into the nasal cavity of the bears. Light-weight aluminum cylinders with a pressure regulator and flowmeter were used. Initially, a flow rate of 2 L/min provided to seven bears, independent of their body mass, resulted in various degrees of hyperoxemia (PaO_2 range 14.5–38.7 kPa; 109–290 mmHg; Fahlman et al. 2010). Thereafter, the flow rate was adjusted based on body mass to approach the minimum required flow rate necessary to meet a target PaO_2. Calculation of the target PaO_2 has been described (Fahlman et al. 2012). The target PaO_2 represents the estimated PaO_2 value that could be expected in a

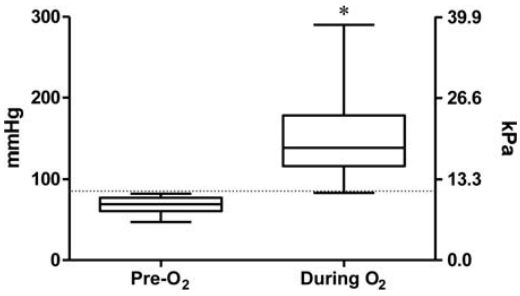

FIGURE 1. Partial pressure of arterial oxygen (PaO_2) before (pre-O_2) and during intranasal oxygen therapy (0.5–3 L/min) administered from a portable oxygen cylinder to 25 anesthetized brown bears (*Ursus arctos*) that were hypoxemic before oxygen therapy was instituted. The whiskers represent minimum and maximum values, and the asterisk indicates a significant difference ($P<0.05$) from the previous sample. The dotted line represents the mean target PaO_2 of 11.1 kPa (individual range 10.1–11.6 kPa).

normal awake brown bear at the altitude of each capture site. Using local barometric pressure for each individual, the resulting target PaO_2 ranged from 10.1 to 11.6 kPa (mean 11.1 kPa) [76–87 mmHg (mean 83 mmHg)]. Brown bears were considered hypoxemic if their PaO_2 was below their calculated target value. Normoxemic bears with increased rectal temperatures (≥39.7 C) received intranasal oxygen therapy as part of the treatment protocol for hyperthermia.

Statistical analysis

Physiologic values measured before and during oxygen therapy were compared with a two-tailed *t*-test. A one-way, repeated measures analysis of variance followed by a post hoc Bonferroni's multiple comparison test was used for animals with PaO_2 data available pre-, during and post-O_2 therapy. All analyses were performed with GraphPad Prism (GraphPad Software Inc., La Jolla, California, USA). A *P*-value ≤0.05 was considered significant. Data are presented as mean ± SD (range).

RESULTS

Arterial oxygenation increased significantly during oxygen supplementation (Fig. 1). Of the 31 bears that were sampled before oxygen therapy was initiated, 25 bears were hypoxemic and six bears were normoxemic. In the hypoxemic bears, pre-O_2 values of PaO_2 increased significantly from 9.1±1.3 (6.3–10.9) kPa to 20.4±6.8 (11.1–38.7) kPa during oxygen therapy [68±10 (47–82) mmHg to 153±51 (83–290) mmHg] (Fig. 1), and the PaO_2 reached or increased above the calculated target PaO_2 in all. The SaO_2 increased significantly from 86±6% (71–94%) to 98±2% (93–100%) during oxygen therapy ($n=25$). The SpO_2 increased significantly 86±6% (75–93%) to 91±5% (82–97%) during oxygen therapy ($n=17$).

In the six normoxemic bears that received intranasal oxygen as part of treatment of hyperthermia (rectal temperature 39.7–40.8 C), the PaO_2 increased significantly from pre-O_2 values of 11.7±0.7 (11.1–13.1) kPa to 16.9±4.3 (9.9–22.3) kPa during oxygen therapy [88±5 (83–98) mmHg to 127±32 (74–167) mmHg]. In one of these bears (adult male, 111 kg), the PaO_2 decreased from 11.1 kPa to 9.9 kPa (83 mmHg to 74 mmHg) during oxygen supplementation at 2 L/min.

The PaO_2 response to the administered oxygen flow rates showed wide interindividual variability (Fig. 2). The lowest flow rate of 0.5 L/min was evaluated in bears weighing from 14 to 25 kg, whereas 1 L/min was used in bears weighing up to 102 kg, and 2 L/min in bears up to 230 kg (Fig. 2). The heaviest bear in the study weighed 250 kg and received 3 L/min initially, followed by an increase to 5 L/min. Based on actual body weights, in bears receiving oxygen at a flow rate of 10–20 mL/min per kilogram, the PaO_2 ranged from 9.9 to 22.1 kPa (74–166 mmHg), and in bears receiving 22–133 mL/min per kilogram, the PaO_2 ranged from 11.1 to 38.7 kPa (83–290 mmHg).

In the 11 bears for which data were available pre-, during, and post-O_2, the PaO_2 increased significantly during oxygen supplementation (Fig. 3). When oxygen therapy was discontinued, the PaO_2 decreased significantly to values that were not significantly different from the pre-O_2 values (Fig. 3).

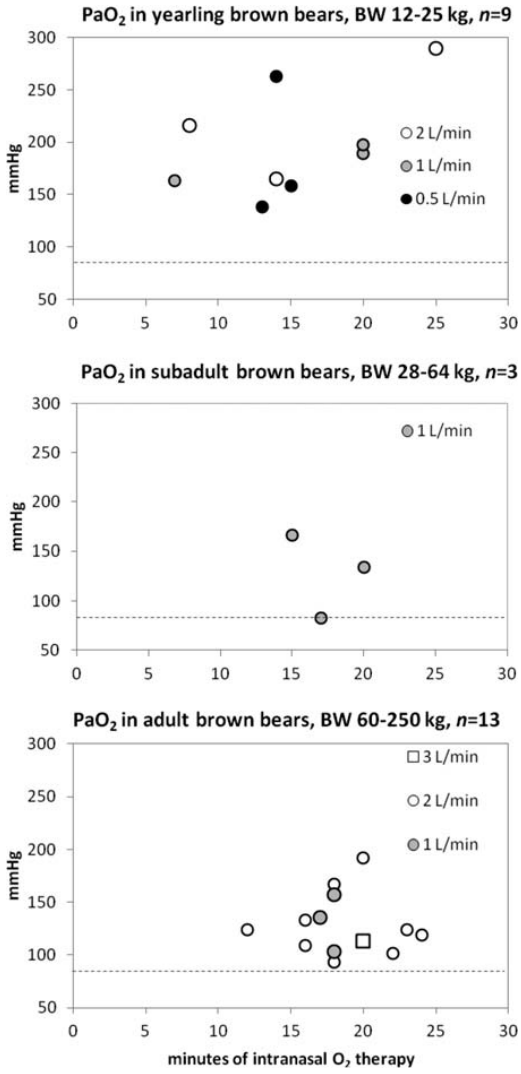

FIGURE 2. Partial pressure of arterial oxygen (PaO_2) in anesthetized brown bears (*Ursus arctos*) supplemented with different flow rates of intranasal oxygen from portable oxygen cylinders. All bears included in this figure were hypoxemic before oxygen therapy was initiated. The flow rate was progressively adjusted between individuals to approach the minimum effective flow rate to meet an individual target PaO_2 ranging from 10.1 to 11.6 kPa. The dotted line represents the mean target PaO_2 of 11.1 kPa.

The $PaCO_2$ increased significantly from pre-O_2 values of 5.6±0.8 (0.1–8.3) kPa to 6.3±0.9 (4.8–9.9) kPa during oxygen therapy [42±6 (31–62) mmHg to 47±7 (36–74) mmHg] (n=31). Mild hypercapnia was recorded in seven bears before, during, and after oxygen therapy ($PaCO_2$ 6.1–6.7 kPa; 46–50 mmHg). In 10 additional bears, a mild hypercapnia was recorded during oxygen therapy only ($PaCO_2$ 6.1–7.3 kPa; 46–55 mmHg). Marked hypercapnia was recorded only in one bear, both before and during oxygen therapy ($PaCO_2$ 8.3 and 9.9 kPa; 62 and 74 mmHg, respectively). Heart rate decreased significantly from pre-O_2 values of 73±18 (36–110) beats/min to 68±16 (32–100) beats/min during oxygen therapy (n=30). Lactate decreased significantly from pre-O_2 values of 5.7±3.4 (1.7–13.0) mmol/L to 3.1±1.7 (0.8–7.3) mmol/L during oxygen therapy (n=31). There was no significant change in respiratory rate [pre-O_2 11±9 (3–48) breaths/min, n=31], rectal temperature [pre-O_2 39.3±1.0 C (37.4–41.3 C), n=30], or pH [pre-O_2 7.27±0.06 (7.12–7.40), n=31].

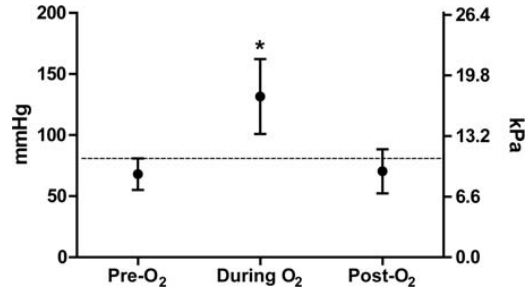

FIGURE 3. Partial pressure of arterial oxygen (PaO_2, mean ± SD) before (pre-O_2), during, and after (post-O_2) intranasal oxygen therapy administered at 0.5–3 L/min from a portable oxygen cylinder to 11 anesthetized brown bears (*Ursus arctos*). The asterisk indicates a significant difference ($P<0.05$) from the pre- and post-O_2 samples. The dotted line represents the mean target PaO_2 of 11.1 kPa (individual range 10.1–11.6 kPa).

The pulse oximetry-derived hemoglobin saturation values (SpO_2) were lower than the SaO_2 in 20 of 21 paired measurements during oxygen supplementation. In eight paired measurements, SpO_2 values ≤90% were recorded, whereas all SaO_2 values were ≥95% in all 21 bears.

DISCUSSION

Low flow oxygen therapy efficiently treated hypoxemia in anesthetized brown

bears. Flow rates from 0.5 to 3 L/min markedly improved arterial oxygenation in the study bears, which had a body mass from 12 to 250 kg. The goal of oxygen therapy in the hypoxemic bears was to increase arterial oxygenation to reach a calculated target PaO_2, representing the assumed normal level. The oxygen flow rates were gradually reduced between individuals, according to the arterial blood gas results, to see whether minimum effective flow rates could be established for different sized bears. Based on actual body weights of the brown bears in this study, we recommend intranasal supplementation at a flow rate of approximately 10–20 mL/min per kilogram. Similarly, low oxygen flow rates (14–18 mL/min per kilogram) effectively treated hypoxemic white-tailed deer when supplemented intranasally with oxygen at 1 L/min (Fahlman et al. 2014). For simplicity in the field, a quick chart for oxygen flow rates has been established for brown bears anesthetized with MZT: 0.5 L/min to bears <50 kg, 1 L/min to bears 51–100 kg, 2 L/min to bears 101–200 kg, and 3 L/min to bears 201–250 kg. It is possible that even lower flow rates may be adequate, depending on the individual response to oxygen therapy.

Use of the minimum effective flow rate will decrease cost by reducing the number of oxygen cylinders and the logistics involved in refilling oxygen cylinders, which also is time consuming during remote fieldwork. A further advantage is a considerable space and weight reduction in the amount of equipment needed in the field, when lower flow rates and thus fewer oxygen cylinders can be used. Based on the current study, the low flow of 0.5 L/min recommended for brown bears weighing up to 50 kg will make a D-cylinder that contains 425 L of oxygen last for 14.2 hr. In comparison, it will only last 1.2 hr at a flow rate of 6 L/min, and 0.7 hr at 10 L/min, which are the previously published flow rates used in brown bears (Mortenson and Bechert 2001; Cattet et al. 2003).

The wide individual variation in PaO_2 between bears receiving the same oxygen flow rate may be due to differences in breathing pattern, ventilatory minute volume, and depth of anesthesia. In general, increasing the oxygen flow rate increases the fraction of inspired oxygen (FIO_2) and the PaO_2, but individual variation has also been shown in dogs (*Canis lupus familiaris*; Fitzpatrick and Crowe 1986). Unnecessary high FIO_2 can lead to resorption atelectasis, which causes increased intrapulmonary shunting, as shown in humans and horses (Nyman et al. 1990; Edmark et al. 2003; Marntell et al. 2005). Inappropriate high flow oxygen therapy commonly results in hyperoxia (abnormally high oxygen content in tissues and organs), but oxygen toxicity depends on both the oxygen dose (i.e., the concentration delivered; FIO_2) and the duration of treatment. With intranasal administration of oxygen the FIO_2 does not reach possible toxicity levels, and wild animals are rarely anesthetized for prolonged periods. Nevertheless, the lowest FIO_2 possible should be used to achieve normoxemia and to prevent hypoxemia and hyperoxemia (supranormal PaO_2). Measurement of the FIO_2 requires a gas analyzer and placement of a tracheal catheter, which makes it unfeasible in field situations; thus, FIO_2 values in anesthetized wildlife are seldom reported.

Our second aim was to evaluate the effect of discontinued oxygen therapy on arterial oxygenation, because the beneficial increases in PaO_2 and FIO_2 associated with oxygen administration diminish rapidly when oxygen is discontinued (Dunphy et al. 2002). In dogs the FIO_2 falls to baseline within 10 sec when oxygen therapy is interrupted (Fitzpatrick and Crowe 1986). In the current study, hypoxemia resumed when oxygen supplementation was interrupted, as also described in other wildlife species such as white-tailed deer (*Odocoileus virginianus*) and bongo antelope (*Tragelaphus eury-*

cerus; Schumacher et al. 1997; Fahlman et al. 2014). Thus, continuous oxygen supplementation is imperative throughout anesthesia.

In agreement with our results, blood gas studies in other species of bears have also demonstrated hypoxemia during anesthesia with MZT (Caulkett and Cattet 1997; Caulkett et al. 1999). Hypoxemia is often clinically silent, and pulse oximetry can be unreliable for detection of hypoxemia in bears and other wildlife species (Schumacher et al. 1997; Cattet et al. 1999; Mich et al. 2008; Fahlman et al. 2010; Muller et al. 2012). Pulse oximetry underestimated hemoglobin oxygen saturation during oxygen therapy in all but one of the 21 paired measurements of SaO_2 and SpO_2 from brown bears in the present study. When these subjects were breathing air, pre- or post-O_2 supplementation, SpO_2 unpredictably overestimated or underestimated the saturation compared with SaO_2. Because pulse oximetry and clinical signs are insufficiently sensitive to ensure recognition of hypoxemia, routine administration of oxygen is recommended. However, previously recommended flow rates in published literature might not be optimal, or evaluated on the basis of arterial blood gases. In brown bears, flow rates of 6–10 L/min were reported to increase hemoglobin oxygen saturation based on pulse-oximetry (Cattet et al. 2003). Another study reports that brown bears supplemented with intranasal oxygen at 6 L/min had relatively low PaO_2 values (mean±SE; 8.5±0.4 kPa; 64±3 mmHg), possibly because of low body temperatures or mixed venous-arterial samples when sampling sublingual vessels (Mortenson and Bechert 2001). Flow rates of 2–5 L/min resulted in hyperoxemia in some brown bears, with PaO_2 values up to 40.8 kPa (306 mmHg) (Fahlman et al. 2010). Unnecessarily high flow rates are wasteful of oxygen and less economical, and the only advantage to the animal is increased time to desaturation of the hemoglobin (Edmark et al. 2003). Oxygen flow rates should preferably be adjusted according to arterial blood gas determinations.

Intranasal oxygen administration is a practical and noninvasive method that is simple to perform in the field. Nasal lines were easily placed in the anesthetized bears and should be secured close to the nares to prevent displacement. Arterial oxygenation improved whether unilateral or bilateral lines were used for the bears, as also demonstrated in dogs (Dunphy et al. 2002). When comparing a specific flow rate administered through one nasal catheter to that same flow rate divided between two nasal lines, there was no difference in the PaO_2 and the FIO_2 in the dog study (Dunphy et al. 2002).

In addition to treatment of hypoxemic brown bears, intranasal oxygen was administered as part of the treatment protocol for hyperthermia to brown bears with elevated rectal temperatures. It has been shown in pigs (*Sus scrofa*) that nasal flushing with oxygen induces a rapid, flow-dependant decrease in brain temperature (Einer-Jensen et al. 2001). Since both hypoxemia and hyperthermia adversely affect brain and nervous tissue, local cooling of the nasal cavities with oxygen aid in prevention of brain injury.

Although the mean $PaCO_2$ increase was statistically significant, the hypercapnia was not considered clinically relevant. Similar levels of hypercapnia and an increase in $PaCO_2$ over time during anesthesia have been recorded in anesthetized bears that were not given oxygen (Fahlman et al. 2011).

The adult male bear that was hypoxemic (PaO_2 8.7 kPa; 65 mmHg) despite oxygen therapy at 3 L/min was tachypneic (70 breaths/min) and hyperthermic (40.9 C). After treatment with a 0.5-L cold-water enema, 1 L of intravenous fluids, and an increased oxygen flow rate of 5 L/min, the respiratory rate had decreased to 28 breaths/min and normoxemia was established. The initial poor response in arterial oxygenation to oxygen therapy in this bear

was probably due to the rapid shallow breathing pattern, with increased dead space ventilation and decreased alveolar ventilation, leading to an impaired ventilation/perfusion (V/Q) matching. An impaired V/Q mismatch can be treated with increased oxygen concentration.

In conclusion, low flow rates of intranasal oxygen were sufficient to improve arterial oxygenation in anesthetized brown bears. Intranasal administration is a practical and noninvasive method of oxygen supplementation that is simple to perform in the field. Oxygen supplementation should be provided continuously throughout anesthesia, because hypoxemia quickly recurred when oxygen was discontinued.

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