Physiologic Evaluation of Capture and Anesthesia with Medetomidine–Zolazepam–Tiletamine in Brown Bears (*Ursus arctos*)

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PHYSIOLOGIC EVALUATION OF CAPTURE AND ANESTHESIA WITH MEDETOMIDINE–ZOLAZEPAM–TILETAMINE IN BROWN BEARS (*URSUS ARCTOS*)

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Abstract: Physiologic variables during anesthesia with medetomidine–zolazepam–tiletamine were evaluated in 52 free-ranging brown bears (*Ursus arctos*) darted from a helicopter and in six captive brown bears darted at a zoo. During anesthesia, rectal temperature, respiratory rate, heart rate, and pulse oximetry derived hemoglobin oxygen saturation were recorded. Arterial blood samples were collected and immediately analyzed for evaluation of pulmonary gas exchange, acid–base status, and selected hematologic and plasma variables. At the end of anesthesia, atipamezole was administered intramuscularly at five times the medetomidine dose. Capture-induced hyperthermia and lactic acidemia were documented in free-ranging bears. Hypoxemia during anesthesia was documented in both free-ranging and captive bears. In free-ranging bears, rectal temperature, heart rate, lactate, hematocrit, and hemoglobin decreased significantly during anesthesia, whereas partial pressure of arterial carbon dioxide, pH, potassium, and glucose increased. Yearlings had a significantly higher heart rate, pH, base excess, bicarbonate, and glucose, and had a significantly lower rectal temperature, sodium, hematocrit, and hemoglobin when compared with subadult and adult brown bears. In conclusion, alterations in pulmonary gas exchange and acid–base status in brown bears during anesthesia with medetomidine–zolazepam–tiletamine with the doses and capture methods used in this study were identified. Oxygen supplementation is recommended to counteract hypoxemia during anesthesia.

Key words: Acid–base status, anesthesia, arterial blood gases, brown bear, immobilization, lactate, *Ursus arctos*.

INTRODUCTION

When immobilizing wildlife, the capture event and the anesthetic drugs can influence the animal's welfare by altering physiologic variables. Patterns of physiologic disturbance vary with method of capture.[10,18,23,24] The health status of the animal can be affected both during and after the actual capture, and death may occur.[31] A low mortality rate is sometimes reported as the single measure of a safe capture method, with no or a limited description of physiologic responses.[1,16] However, it is important to stress that mortality does not reflect morbidity. Safe handling should ensure stable physiology to minimize the risk of complications. To state that capture methods, drug combinations, or doses are safe, physiologic effects must be studied. In addition, evaluation and improvement of wildlife-handling techniques are important, because events that occur with capture can influence research results, e.g., movement rates or reproduction can be negatively altered after capture.[6,15]

For immobilization of brown bears (*Ursus arctos*) and other bear species, zolazepam–tiletamine has been used extensively, although prolonged recoveries are common.[4,5,11,30,32,33] Ketamine in combination with xylazine or medetomidine has also been used in bears, but sudden recoveries can occur.[1,9,21,33] Zolazepam–tiletamine in combination with xylazine or medetomidine has been reported to immobilize different bear species effectively,[7–9,13,28] including brown bears,[2,11,29] both in captivity and in the wild. Advantages of the

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Table 1. Age, sex, body mass, and drug doses of medetomidine–zolazepam–tiletamine used for anesthesia of 52 free-ranging and six captive brown bears. During helicopter capture of free-ranging bears, additional darts were required subsequent to the initial dart dose in 16 bears.

Age	*n*, sex[a]	Body mass (kg)	Medetomidine[b]	Zolazepam–tiletamine[c]
Free-ranging bears			Initial dart dose[d] (mg)	Initial dart dose[d] (mg)
Yearling	14F, 5M	12–41	1.25	62.5
Subadult	5F, 1M	42–65	2.5 or 5	125 or 250
Adult	19F	62–120	5 or 10	250 or 500
Adult	8M	80–241	10 or 15	500 or 750
Single dart			Actual mean ± SD dose (range), mg/kg[e]	Actual mean ± SD dose (range), mg/kg[e]
Yearlings ($n = 16$)			0.07 ± 0.02 (0.04–0.10)	3.2 ± 0.8 (1.5–5.2)
Subadults ($n = 5$) + adults ($n = 15$)			0.08 ± 0.02 (0.05–0.13)	4.1 ± 1.3 (2.3–6.6)
2 or 3 darts[f]				
Yearlings ($n = 3$)			0.12 ± 0.02 (0.10–0.14)	6.1 ± 1.1 (5.0–7.2)
Subadults ($n = 1$) + adults ($n = 12$)			0.12 ± 0.03 (0.06–0.20)	6.0 ± 1.7 (3.1–10.0)
Captive bears			Initial dart dose (mg)/ actual dose (mg/kg)	Initial dart dose (mg)/ actual dose (mg/kg)
Yearlings	2F, 1M	37, 38, 48	1.25/0.03 in all 3	62.5/1.7, 1.6, 1.3
Subadults	1F, 2M	62, 86, 87	2/0.03, 0.02, 0.02	166/2.7, 1.9, 1.9

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

[b] For yearlings: Domitor® vet., 1 mg/ml, Orion Pharma Animal Health, Espoo, Finland; for subadults and adults: Zalopine, 10 mg/ml, Orion Pharma Animal Health.

[c] Zoletil forte vet., Virbac S.A., Carros, France.

[d] The total dose in milligrams, chosen after estimation of the bear's body size. The lower dose of medetomidine was combined with the lower dose of zolazepam–tiletamine, e.g., medetomidine 2.5 mg + zolazepam–tiletamine 125 mg, and vice versa.

[e] Actual dose is the dose in milligrams per kilogram, according to body weight obtained during anesthesia.

[f] Three darts were only needed to two yearlings.

medetomidine–zolazepam–tiletamine combination (MZT) include a small drug volume suitable for remote darting and reversibility by the use of atipamezole. In polar bears (*Ursus maritimus*) and black bears (*Ursus americanus*), hypertension and hypoxemia have been described as the major disadvantages of MZT.[13,14]

Between 1992 and 2009, the Scandinavian Brown Bear Project has conducted more than 1,300 immobilizations with MZT in free-ranging brown bears by darting from a helicopter. Mortality has been low (0.7%, 9 bears) (Arnemo, unpubl. data), but physiologic effects have not yet been reported. The objective of this study was to evaluate physiologic variables, including pulmonary gas exchange and acid–base status, during MZT anesthesia of free-ranging and captive brown bears. A pilot study was also performed to assess whether arterial oxygenation could be increased by intranasal oxygen (O_2) supplementation.[19]

MATERIALS AND METHODS

Study area and animals

The study included the anesthesia of 52 free-ranging and six captive brown bears between April and June 2004 and 2006–2007. Age, sex, and body mass of all the bears are presented in Table 1. The bears were categorized as yearlings (1 yr), subadults (2–3 yr), or adults (≥4 yr) based on the actual birth year if followed since birth, or age determination of an extracted premolar tooth.[26] No bears were darted the year they were born. Forty-nine free-ranging bears were anesthetized in the county of Dalarna, Sweden (approximately 61°N, 15°E) at altitudes of 300–700 m above sea level (barometric pressure 690–746 mm Hg). Three free-ranging bears were anesthetized in the Sarek National Park, Norrbotten, Sweden (approximately 67°N, 17°E) at altitudes of 520–725 m above sea level (barometric pressure 691–719 mm Hg). Ambient temperature ranged from 2°C to 23°C.

Captive bears were anesthetized for microchipping in their indoor quarters at Orsa Bear Park, a zoo in Dalarna at an altitude of 550 m above sea level (barometric pressure 708 mm Hg). Captive bears were fasted overnight before anesthesia. Free-ranging bears were anesthetized for individual marking for ecologic studies in the Scandinavian Brown Bear Research Project. Bears immobilized for the first time received an ear

tag, a lip tattoo, and a microchip. Adult and subadult bears received either very high frequency (VHF) radiocollars (Telonics MOD-500, Telonics, Inc., Mesa, Arizona 85204, USA), or global positioning system radiocollars (Vectronics GPS PLUS, Vectronic Aerospace GmbH, Carl-Scheele-Str. 12, DE-12489 Berlin, Germany). Twelve adult bears (11 female, 1 male) and 14 yearlings (all female) received intraperitoneal VHF transmitters (IMP/400/L, Telonics, Inc.). For access to the peritoneal cavity, a ventral midline incision was made by using standard surgical procedures, as described previously.[3] Approval was given by the Ethical Committee on Animal Experiments, Uppsala, Sweden.

Capture methods, drugs, and darting equipment

Free-ranging radiomarked bears were located by radiotracking from a helicopter and were darted at a distance of 4–6 m. Most bears were darted in the hindquarters while running across a marsh, a frozen lake, or a clear cut in the forest. The time of intensive helicopter pursuit, when the helicopter approached the bear for the actual darting, was always <1 min and usually <30 sec. A radiomarked bear, or a family group, could be tracked and driven (sometimes without being observed from the air because of dense vegetation) for longer periods, with the helicopter flying at a distance from the animal, to move the bear to an open area where darting could take place. The helicopter crew subjectively estimated the distance that the bear moved from first sighting from the helicopter (before darting) until recumbency (after darting). When capturing family groups ($n = 17$), the yearlings were darted before the radiomarked mother. After successful darting of the first yearling, it was observed from a high altitude throughout the induction period, before darting of the next family member. When a darted bear slowed down, as the drugs started to have effect, the other family members left it and were then not followed by the helicopter. It was often 10–20 min between the intensive pursuits for darting of different family members. All yearlings and five adults were naïve to capture, whereas the other bears had been captured 1–12 times before.

All the bears were anesthetized with medetomidine, zolazepam, and tiletamine by using standard drug doses according to body size, estimated from the helicopter (Table 1). The drugs were delivered in 1.5- or 3-ml dart syringes with 1.5 × 25-mm or 2.0 × 40-mm barbed needles fired from a carbon dioxide (CO_2) powered rifle (Dan-Inject, DK-7080 Børkop, Denmark). If no sign of drug effect was noted within 5–10 min of initial darting, then the animal was darted again with either another full dose or a lower dose. If a darted bear was moving toward risky terrain, such as open water, then it could be darted a second time, within 5 min of the first dart, to rapidly induce recumbency. All animals were weighed during anesthesia, and the actual drug doses were calculated (Table 1).

During anesthesia, an eye gel (Viscotears®, CIBA Vision AG, CH-8424 Embrach, Switzerland) was applied to the cornea to prevent desiccation, and a blindfold was placed. The animals were primarily positioned in lateral recumbency, but, for surgery ($n = 26$), they were moved to dorsal recumbency. For analgesia, 4 mg/kg carprofen (Rimadyl® vet. 50 mg/ml, Orion Pharma Animal Health, FI-02200 Espoo, Finland) was administered subcutaneously before surgery. To minimize the risk of wound infection, procaine benzylpenicillin and benzathine benzylpenicillin were injected intramuscularly at 100,000 IU/kg (PENI-kél L.A. 15+15, Kela Laboratoria NV, B-2320 Hoogstraten, Belgium).

The bears needed to be sufficiently anesthetized to permit handling for fitting of radiocollars as well as surgery for placement of intraperitoneal radio transmitters. In case of an inadequate plane of anesthesia or spontaneous recovery, supplemental doses of 0.5–1.0 mg medetomidine were administered intramuscularly, depending on the depth and prolongation of anesthesia needed.

Antagonism of medetomidine was achieved with atipamezole (Antisedan® vet., 5 mg/ml, Orion Pharma Animal Health) administered intramuscularly at five times the total dose of medetomidine. The time from darting until injection of atipamezole was recorded. Family members were placed together, and the animals were left undisturbed to recover in lateral recumbency. Long-term survival after capture and anesthesia of free-ranging bears was documented by radiotracking.

Monitoring

Time from darting to recumbency (induction time) was recorded. Rectal temperature was monitored with a digital thermometer with continuous reading and a measurement range from 28.9°C to 42.2°C (Welch Allyn Diatec 600, Welch Allyn, Inc., Skaneateles Falls, New York 13153, USA). To prevent or treat hyperthermia (rectal temperature ≥ 40.0°C), one or several of

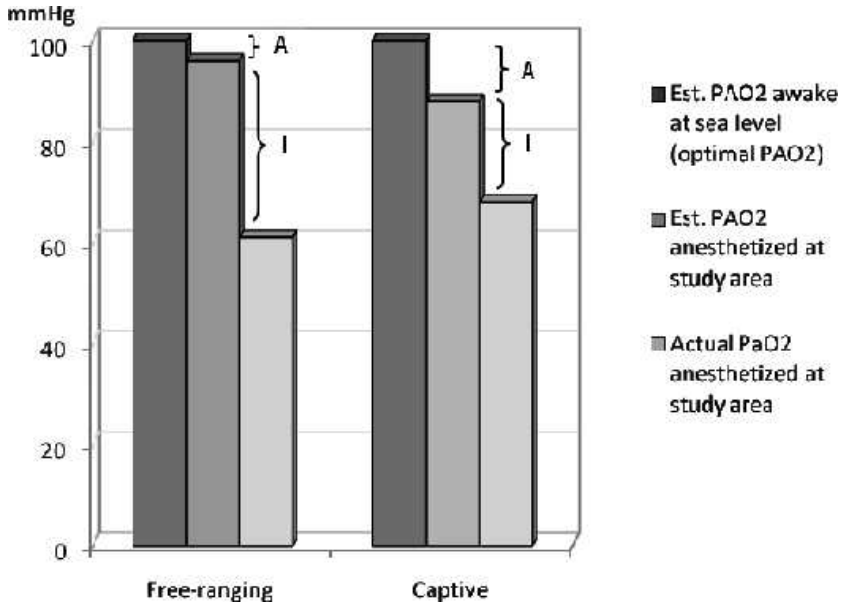

Figure 1. Mean alveolar-arterial oxygen tension difference [P(A-a)O_2] estimated in 46 free-ranging and five captive brown bears 30 min after induction with medetomidine–zolazepam–tiletamine. Influence on arterial oxygenation (PaO_2) by altitude (A) (barometric pressure; P_B) and intrapulmonary factors (I), i.e., ventilation–perfusion mismatch, including shunt or diffusion impairment. Mean values at standard temperature are presented. Free-ranging bears were anesthetized at altitudes of 300–700 m (P_B 690–746 mm Hg) and captive bears at an altitude of 550 m (P_B 708 mm Hg). Hypoventilation influenced PaO_2 in individual bears but did not contribute to the decrease in the mean P(A-a)O_2.

the following cooling measures were applied: shading, fanning, cooling with water, snow, and/or intravenous fluid. The respiratory rate was monitored by observing thoracic movements. Heart rate and hemoglobin oxygen saturation (SpO_2) were monitored continuously by pulse oximetry, with the pulse oximeter probe attached to the tongue (Nellcor NPB-40 Handheld Pulse Oximeter, Nellcor Inc., Pleasanton, California 94588, USA). Rectal temperature, respiratory rate, heart rate, and SpO_2 were monitored throughout anesthesia. Capillary refill time was measured and muscle relaxation was assessed subjectively by control of muscular tone in an extremity at the time of blood sampling.

Arterial blood samples were collected for analysis of blood gases, acid–base status, and selected hematologic and plasma variables. Up to three samples were collected from each animal. At first, blood samples were collected only 30 and 60 min after the initial darting. From 2006, one more sample was collected as early as possible after recumbency (<25 min after darting), to enable evaluation of early physiologic alterations. The samples were collected anaerobically from the femoral artery by using 0.8 × 40-mm needles and self-filling arterial syringes with heparin (PICO™70, Radiometer Copenhagen, DK-2700 Brønshøj, Denmark). The femoral pulse was palpated in the groin, and the needle was inserted percutaneously into the artery, confirmed by pulsating blood. Firm pressure was applied to the sample site for 2 min after sampling to avoid bleeding. The samples were processed immediately in the field by using 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). Because the i-STAT®1 analyzer only operates at 16°C to 30°C, it was kept on a warm water bottle in a polystyrene foam box in an insulated cooler bag. The analysis included measured values for pH, partial pressures of arterial CO_2 ($PaCO_2$) and arterial oxygen (PaO_2), lactate, hematocrit, sodium, potassium, chloride, urea, and glucose. Blood gas values and pH were corrected to the rectal temperature. Calculated values were provided for actual base excess (BE), actual bicarbonate (HCO_3), arterial hemoglobin oxygen saturation (SaO_2), and hemoglobin.

The alveolar-arterial oxygen tension difference, P(A-a)O_2, at standard temperature (37°C) was

estimated from the equation: $PAO_2 = F_IO_2 (P_B - P_{H_2O}) - (PaCO_2/RQ)$. PAO_2 = partial pressure of alveolar oxygen, F_IO_2 = fraction of inspired O_2 (0.21), P_B = barometric pressure, P_{H_2O} = saturated vapor pressure for water at 37°C (47 mm Hg), and RQ = respiratory quotient (assumed to be 0.8).

To describe the contribution of different factors that influence the arterial oxygenation (PaO_2), the difference between the estimated PAO_2 awake at sea level (optimal PAO_2) and the actual PaO_2 anesthetized at the study area was set to 100% (Fig. 1). The relative contribution of the following factors was calculated for the following: Altitude = the difference between estimated PAO_2 awake at sea level (optimal PAO_2) and estimated PAO_2 awake at study area; Hypoventilation = the difference between estimated PAO_2 awake at study area and the estimated PAO_2 anesthetized at study area; Intrapulmonary factors = the difference between the estimated PAO_2 anesthetized at study area and the actual PaO_2 anesthetized at study area.

The strong ion difference (SID) was calculated as (Na + K) − (Cl + lactate).[17] Acidemia was defined as a pH < 7.35, and marked acidemia if pH < 7.20. Hypocapnia was defined as a $PaCO_2$ < 35 mm Hg, and hypercapnia was defined as mild ($PaCO_2$ 45–60 mm Hg) or marked ($PaCO_2$ > 60 mm Hg). Hypoxemia was defined as mild (PaO_2 60–80 mm Hg) or marked (PaO_2 < 60 mm Hg). From 2006, bears with a marked hypoxemia were administered intranasal O_2, and these results are published separately.[19]

Statistical analysis

Physiologic data from free-ranging bears were analyzed by using a three-way mixed model in SAS® (SAS® System 9.1, SAS Institute Inc., Cary, North Carolina 27513, USA), with the fixed factors Sex, Age group, ''Post-darting time point,'' and the interaction Age group * ''Post-darting time point.'' ''Post-darting time point'' was the within subjects factor and Sex and Age group were the between subjects factors. Different covariance structures were tested: compound symmetry and heterogeneous compound symmetry with and without between-subject heterogeneity. The covariance structure with the smallest value of the Akaike's Information Criterion was considered to be the most informative. The interaction Age group * ''Post-darting time point'' refers to the statistical test of whether the effect of one of these factors, as measured by differences in the response average, is different for different levels of the other factor. For significant interactions, simple main effects tests were conducted, i.e., which evaluated the effects of one factor when holding the other factor fixed. Respiratory rate and lactate concentration were log-transformed before the analyses because they had positively skewed distributions. Data are presented as mean ± standard deviation (SD) (range). Differences were considered significant when $P < 0.05$. Spearman rank order correlation was used to investigate the correlation between different variables.

RESULTS

Free-ranging bears

Free-ranging bears were successfully immobilized with a single dart dose during 36 of 52 captures, and the time from darting until recumbency ranged from 1 to 13 min. For 16 of the captures, the bears had to be darted with a second ($n = 14$) or third dose ($n = 2$), and the time from first dart until recumbency ranged from 4 to 26 min. Five of these bears required additional darts because of a poor injection site of the first dart (subcutaneous or in small muscle group). The estimated distance that the bears moved after they were sighted from the helicopter until recumbency averaged 870 ± 570 m (range 180–2,400 m). During anesthesia, muscle relaxation was good in all bears, and capillary refill times ranged from 1 to 3 sec.

Hyperthermia was recorded initially in 24 bears (46%) and at 30 min after darting in 15 bears (29%). Rectal temperature was significantly higher in adults and subadults than in yearlings (Table 2); only two yearlings developed hyperthermia. The rectal temperature decreased to ≤37.0°C in two yearlings 60–90 min after darting (lowest temperature 36.2°C). There was no correlation between rectal temperature and body mass or ambient temperature.

Respiratory rate decreased significantly over time but did not correlate to rectal temperature, lactate concentration, or pH. Heart rate was the only variable that differed significantly between the sexes; being higher in females than in males. Yearlings had a significantly higher heart rate than subadult and adult bears, and heart rate decreased over time for all age groups (Table 2).

A significant change over time occurred in PaO_2, $PaCO_2$, SaO_2, PAO_2, and $P(A\text{-}a)O_2$ (Table 2). A mild hypoxemia was recorded in 30 bears (PaO_2 60–80 mmHg), and a marked hypoxemia was recorded in 8 bears (PaO_2 47–59 mm Hg). Marked hypoxemia was recorded

Table 2. Physiologic variables and arterial blood gases in free-ranging brown bears darted with medetomidine–zolazepam–tiletamine from a helicopter. Values presented are exclusive of those episodes where bears were given oxygen supplementation. Mean ± standard deviation (range) is presented.

	Adult and subadult brown bears			Yearling brown bears		
Variable[a]	14 ± 5 min[b], n = 10	30 min, n = 29	60 min, n = 27	18 ± 4 min[b], n = 7	30 min, n = 18	60 min, n = 16
Respiratory rate[c] (breaths/min)	15 ± 8 (6–36)	14 ± 21 (2–92)	16 ± 22 (2–80)[d]	13 ± 6 (7–26)	11 ± 4 (4–24)[d]	13 ± 4 (4–18)
Heart rate[e,f] (beats/min)	83 ± 15 (54–100)	70 ± 17 (44–115)[d]	63 ± 14 (41–90)[d,e]	81 ± 16 (60–104)	79 ± 20 (34–110)	72 ± 18 (40–104)[d]
Rectal temperature[c,f,g] (°C)	40.3 ± 0.6 (39.3–41.2)	40.0 ± 0.9 (38.0–41.8)	40.0 ± 0.7 (38.9–41.5)	39.4 ± 0.6 (38.8–40.5)	39.2 ± 0.6 (38.0–40.4)	38.6 ± 0.7 (37.4–39.6)[d,e]
SpO_2 (%)	89 ± 5 (83–97)	88 ± 5 (80–98)	90 ± 4 (81–98)	91 ± 5 (85–96)	89 ± 4 (81–96)	90 ± 5 (75–97)
SaO_2[c] (%)	90 ± 3 (83–95)	88 ± 5 (73–94)	89 ± 5 (69–94)	90 ± 6 (78–95)	88 ± 6 (71–94)	92 ± 3 (83–96)[e]
PaO_2[c,g] (mmHg)	91 ± 13 (63–110)	75 ± 12 (50–96)[d]	76 ± 10 (49–89)[d]	76 ± 11 (56–89)	71 ± 11 (47–82)	78 ± 8 (59–87)[e]
PAO_2[c] (mmHg)	100 ± 7 (92–110)	96 ± 6 (82–105)	94 ± 6 (82–107)[d,e]	98 ± 5 (91–104)	96 ± 6 (85–104)	93 ± 3 (87–97)[d,e]
P(A-a) O_2[c,g] (mmHg)	26 ± 10 (14–40)	36 ± 9 (23–57)[d]	32 ± 7 (23–49)[d,e]	32 ± 7 (24–44)	35 ± 9 (21–47)	23 ± 10 (9–44)[d,e]
$PaCO_2$[c] (mmHg)	39 ± 6 (28–47)	42 ± 6 (33–54)	43 ± 5 (32–52)[d,e]	39 ± 3 (35–44)	40 ± 4 (34–48)	42 ± 3 (37–47)[d,e]

[a] SpO_2 = hemoglobin oxygen saturation measured by pulse oximetry; SaO_2 = arterial oxygen saturation (calculated value); PaO_2 and $PaCO_2$ = partial pressures of arterial oxygen and carbon dioxide (measured values). Blood gas values were corrected to the rectal temperature. PAO_2 = partial pressure of alveolar oxygen, and P(A-a)O_2 = alveolar-arterial oxygen tension difference (calculated at standard temperature 37°C).

[b] Time after darting. The first sample was collected as early as possible after recumbency.

[c] Significant difference over time, regardless of age. Analysis included the full data set of all age groups.

[d] Significant difference from the early value (first sample) within the age group.

[e] Significant difference from the 30-min value (second sample) within the age group.

[f] Significant difference between age groups.

[g] Significant interaction (Age group * "Post-darting time point").

Table 3. Acid–base status, hematologic, and plasma variables in arterial blood from free-ranging brown bears darted from a helicopter with medetomidine–zolazepam–tiletamine. Values presented are exclusive of those episodes where bears were given oxygen supplementation. Mean ± standard deviation (range) is presented.

	Adult and subadult brown bears			Yearling brown bears		
Variable[a]	14 ± 5 min[b], n = 10	30 min, n = 29	60 min, n = 27	18 ± 4 min[b], n = 7	30 min, n = 18	60 min, n = 16
pH[c,f,g]	7.15 ± 0.09 (6.96–7.27)	7.27 ± 0.04 (7.15–7.33)[d]	7.31 ± 0.03 (7.25–7.37)[d,e]	7.28 ± 0.03 (7.24–7.32)	7.29 ± 0.03 (7.25–7.35)	7.33 ± 0.02 (7.29–7.35)[d,e]
Lactate[c,g] (mmol/L)	10.7 ± 5.2 (2.6 to >20.0)	5.4 ± 3.8 (0.9–14.1)[d]	3.1 ± 1.7 (0.5–6.0)[d,e]	5.6 ± 2.7 (3.1–9.7)	4.0 ± 1.6 (2.0–7.5)[d]	2.2 ± 0.8 (1.0–3.9)[d,e]
Base excess[c,f,g] (mmol/L)	−14 ± 5 (−22 to −5)	−7 ± 3 (−14 to 0)[d]	−4 ± 3 (−9 to 2)[d,e]	−7 ± 2 (−10 to −6)	−6 ± 2 (−9 to −3)[d]	−4 ± 2 (−7 to −1)[d,e]
HCO_3[c,f,g] (mmol/L)	14 ± 4 (8–21)	19 ± 3 (13–26)[d]	21 ± 3 (16–28)[d,e]	18 ± 1 (15–20)	19 ± 2 (16–23)[d]	22 ± 2 (19–25)[d,e]
SID[c] (mEq/L)	20 ± 7 (14–32)	26 ± 4 (18–33)[d]	29 ± 3 (24–37)[d,e]	24 ± 3 (18–27)	25 ± 2 (22–30)	28 ± 1 (27–31)[d,e]
Sodium[f] (mmol/L)	131 ± 2 (128–134)	132 ± 2 (128–135)	132 ± 2 (128–136)	129 ± 1 (127–131)	128 ± 2 (125–133)	129 ± 2 (124–132)
Potassium[c] (mmol/L)	3.5 ± 0.6 (2.5–4.6)	3.8 ± 0.5 (2.9–6.0)	3.7 ± 0.4 (2.9–5.1)	3.2 ± 0.4 (2.8–3.9)	3.6 ± 0.3 (3.1–4.3)[d]	3.5 ± 0.3 (3.2–4.3)[d]
Chloride (mmol/L)	105 ± 3 (102–110)	104 ± 3 (98–110)	104 ± 3 (96–111)	103 ± 3 (100–109)	103 ± 2 (98–108)	102 ± 1 (99–104)
Glucose[c,f,g] (mmol/L)	6.0 ± 1.6 (3.8–9.4)	6.6 ± 2.2 (2.7–11.8)	8.1 ± 2.7 (3.7–14.7)[d,e]	7.6 ± 1.2 (6.1–8.8)	8.0 ± 1.2 (5.7–9.9)[d]	8.5 ± 1.8 (5.3–11.6)[d,e]
Hematocrit[c,f] (%)	47 ± 4 (41–55)	46 ± 4 (38–53)[d]	44 ± 4 (34–50)[d,e]	44 ± 2 (40–46)	42 ± 2 (38–47)[d]	41 ± 3 (37–47)[d,e]
Hemoglobin[c,f] (mmol/L)	16 ± 1 (14–19)	16 ± 1 (13–18)[d]	15 ± 2 (12–17)[d,e]	15 ± 1 (14–16)	14 ± 1 (13–16)[d]	14 ± 1 (13–16)[d,e]

[a] HCO_3 = bicarbonate; SID = strong ion difference calculated as (sodium + potassium) − (chloride + lactate).
[b] Time after darting. The first sample was collected as early as possible after recumbency.
[c] Significant difference over time, regardless of age. Analysis included the full data set of all age groups.
[d] Significant difference from the early value (first sample) within the age group.
[e] Significant difference from the 30-min value (second sample) within the age group.
[f] Significant difference between age groups.
[g] Significant interaction (Age group * "Post-darting time point").

early as well as 30 and 60 min after darting. Altitude (barometric pressure) was responsible for approximately 10% of the reduction in the mean value of PaO_2 at 30 min after darting, whereas intrapulmonary factors contributed, with approximately 90% (Fig. 1). The effect of treatment with intranasal O_2 supplementation in seven free-ranging bears with a marked hypoxemia, hyperthermia, or both is published separately.[19] A mild hypercapnia was recorded in 17 bears ($PaCO_2$ 45–54 mm Hg), whereas no bears developed a marked hypercapnia. Hypocapnia was recorded in seven bears ($PaCO_2$ 28–35 mm Hg).

A pH $<$ 7.35 was recorded in 50 bears (96%) of all age groups. A pH $<$ 7.2 was recorded in adults and subadult bears only. In six of 17 arterial samples collected early in the sampling period, and in two of 47 samples collected 30 min after darting, the pH was $<$7.2. The initial lactate concentration ranged from 2.6 to $>$20.0 mmol/L, which is the upper limit of measurability of the i-STAT®1. Lactate concentration decreased significantly during anesthesia, whereas pH, BE, HCO_3, and SID increased significantly (Table 3). Adult and subadult bears initially had significantly lower pH, BE, and HCO_3 than yearlings. Lactate concentration correlated significantly to pH, whereas there was no correlation between lactate concentration and respiratory rate. There was no correlation between the distance moved and the lactate concentration, the rectal temperature, or the glucose concentration.

Potassium and glucose increased significantly during anesthesia, whereas hematocrit and hemoglobin decreased significantly (Table 3). In 14 bears, the exact urea concentration could not be measured because it was $<$1.0 mmol/L, which is the lower detection limit of the i-STAT®1. The highest urea concentration recorded was 25.1 mmol/L.

All the bears were immobilized sufficiently to allow at least 1 hr of handling time, except two yearlings, which required hand-injection of medetomidine 13 min after darting. Supplemental doses of medetomidine were given after $>$1 hr of anesthesia to five yearlings, one subadult, and one adult bear because of an inadequate plane of anesthesia (spontaneous blinking, or slight body movement) 69–101 min after darting. One of the yearlings was undergoing surgery when additional dosing was required. Supplemental medetomidine was also given to two yearlings that recovered spontaneously without any preceding signs 87 and 116 min after darting. No sudden recoveries occurred in adult or subadult bears.

Atipamezole was injected 64–151 min after darting. No capture-related mortalities occurred during or within 1 mo after anesthesia.

Captive bears

None of the six captive bears developed hyperthermia. The lowest rectal temperature recorded was 36.3°C in a yearling 60 min after darting. A mild hypoxemia was recorded in four bears. Two bears developed a marked hypoxemia and were treated with intranasal O_2 supplementation.[19] Altitude (barometric pressure) was responsible for approximately 40% and intrapulmonary factors contributed with approximately 60% of the reduction in the mean value of PaO_2 in captive bears, at 30 min after darting (Fig. 1). A mild hypercapnia was recorded in four bears. The PAO_2 and $P(A\text{-}a)O_2$ were 88 $\pm$ 4 mm Hg and 20 $\pm$ 10 mm Hg, respectively, at 30 min, and 87 $\pm$ 4 mm Hg and 13 $\pm$ 9 mm Hg, respectively, at 60 min after darting.

A pH $<$ 7.35 was recorded in all six captive bears, whereas none developed a pH $<$ 7.2. The initial lactate concentration ranged from 0.7 to 2.7 mmol/L and decreased over time in all bears. Glucose ranged from 7.3 to 9.3 mmol/L throughout anesthesia. Three bears had urea concentrations $<$1.0 mmol/L, whereas the highest urea concentration recorded was 2.4 mmol/L.

DISCUSSION

Advantages of MZT anesthesia in brown bears included a small drug volume for darting, reliable induction after good dart placement, and a predictable duration of anesthesia. A common adverse effect reported during anesthesia with MZT in sun bears (*Helarctos malayanus*) was vomiting during the induction, despite previous fasting.[28] In the current study, no brown bears vomited, even though only the captive animals were fasted. Excessive salivation and brief tremors have been reported in grizzly bears (*Ursus arctos horribilis*) anesthetized with zolazepam–tiletamine,[32] but none of these adverse effects during anesthesia with MZT were observed in this study. An increased intensity of the palpebral reflex was an early indicator of a light plane of anesthesia in the brown bears studied, which has also been described in bears immobilized with xylazine–ketamine or medetomidine–ketamine.[12]

Physiologically, a marked hypoxemia was recorded in both free-ranging and captive brown bears in the current study, whereas only free-ranging bears developed hyperthermia and a

marked lactic acidemia. Transient hypoxemia with a $PaO_2 < 60$ mm Hg and mild or minimal hypoventilation have been reported in polar bears and in a black bear immobilized with MZT at various doses.[13,14] In comparison, during immobilization with zolazepam–tiletamine alone, brown bears did not develop hypoxemia, whereas polar bears did.[5,14]

The alveolar-arterial oxygen tension difference, calculated based on standard temperature, indicates an impaired O_2 exchange [$P(A\text{-}a)O_2 >$ 10 mm Hg], in both free-ranging and captive bears, but the impairment was more severe in the free-ranging bears. Deterioration of the pulmonary gas exchange may be of both extrapulmonary origin, such as low barometric pressure (altitude) or hypoventilation (increased $PaCO_2$), and intrapulmonary origin, i.e., ventilation–perfusion mismatch including shunt, or diffusion impairment. The effect of altitude on arterial oxygenation is illustrated by the following equations: at sea level (P_B 760 mm Hg), it would be expected that the PAO_2 in awake bears would be approximately 100 mm Hg, when assuming a $PaCO_2$ value of 40 mm Hg [$PAO_2 = 0.21 \times (760 - 47) - (40/0.8)$]. At the barometric pressure in the different study areas, the PAO_2 was predicted in the range of 85–97 mm Hg in the free-ranging bears (P_B 690–746 mm Hg) [$PAO_2 = 0.21 \times (690 - 47) - (40/0.8)$; $PAO_2 = 0.21 \times (746 - 47) - (40/0.8)$]; and of 87 mm Hg in the captive bears (P_B 708 mm Hg) [$PAO_2 = 0.21 \times (708 - 47) - (40/0.8)$]. Based on the above results, altitude would result in a decrease in PAO_2 of 3–15 mm Hg in the free-ranging bears and of 12 mm Hg in the captive bears. This altitudinal effect on PAO_2 would have the same decreasing effect on PaO_2. During anesthesia, hypoventilation also contributed to hypoxemia in some free-ranging bears (17%), but, when considered in the entire group, did not further decrease the mean $P(A\text{-}a)O_2$. Every increase in $PaCO_2$ by 1 mm Hg will actually decrease PaO_2 by 1.25 mm Hg, when assuming an RQ of 0.8. Based on measured $PaCO_2$ values in the immobilized study bears, this would result in a decrease in PAO_2 of 36–57 mm Hg. The largest decrease in PAO_2 (>50 mm Hg) was in eight bears in which hypoventilation contributed to hypoxemia. Thus, extrapulmonary factors contributed to the reduction in PaO_2: altitude affected PAO_2 in all bears, whereas hypoventilation was evident in some individuals. In addition, increased metabolic production of CO_2 during hyperthermia probably contributed to a reduction in PaO_2 in hyperthermic bears. The remaining decrease in PaO_2 would be because of intrapulmonary factors. As suggested in polar bears and wolverines (*Gulo gulo*) immobilized with drug combinations, including medetomidine,[14,18] ventilation–perfusion mismatch is the most probable reason for hypoxemia, which is consistent with the findings in brown bears of the current study. The presence of intrapulmonary shunting could not be determined in this study. However, a large shunt was unlikely because PaO_2 improved in bears supplemented with O_2, as previously reported.[19] Interestingly, intrapulmonary factors contributed to the increase in the mean calculated alveolar-arterial O_2 tension difference to a higher degree in free-ranging bears than in captive bears, which suggests that the impaired pulmonary gas exchange could be dose dependent, because lower drug doses were used in captivity. Specifically, the impairment was probably related to the medetomidine, because $alpha_2$-agonists can increase the pulmonary vascular pressure and disturb the matching of pulmonary perfusion in relation to ventilation, which results in a reduction in PaO_2.[25] Because hypoxemia was a common finding in the current study, further studies are required to develop effective treatment strategies. The pilot study clearly illustrates the efficacy of intranasal O_2 supplementation.[19]

Initial decreases in pH, SID, HCO_3, and base excess indicate a lactic acidemia in immobilized free-ranging bears, which agrees with acid–base disturbances recorded during anesthesia of free-ranging adult wolverines.[18] Although $PaCO_2$ increased slightly during anesthesia in free-ranging bears, the pH also increased over time, which probably reflects the reduction in the lactate concentration. Thus, although MZT anesthesia had an effect on pulmonary function, it did not further impair the capture-induced acid–base changes. Because hyperthermia and lactic acidemia were recorded only in free-ranging bears, it was probably a result of physical exertion and stress during induction in the wild. Even though capture myopathy has been reported in brown bears trapped with leg-hold snares,[6,22] lactate concentration has not been reported before in any bear species. In the absence of reference values from nonanesthetized bears, lactate concentrations from bears anesthetized in captivity are useful as baseline data when evaluating the effect of a capture method in the wild. Interestingly, in free-ranging adult wolverines, the muscular activity related to resisting

manual restraint after capture by hand with a pole snare resulted in similar lactate concentrations as after helicopter pursuit.[18] As in wolverines, lactate concentration did not correlate to the distance the brown bears ran before recumbency, probably because several factors may influence the work load during the induction, such as terrain (i.e., steepness, snow depth), ambient temperature, and travel speed. Capture-induced hyperthermia can be caused predominantly by stress, as shown in impala (*Aepyceros melampus*), but with possible additional effects of physical activity before recumbency, ambient temperature, and the immobilizing drugs.[27] In the current study, five of seven bears with rectal temperatures $\geq 41.0^{\circ}C$, had respiratory rates of more than 30 breaths per minute. Hyperthermia and concurrently high respiratory rates have also been documented in grizzly bears darted from a helicopter with zolazepam–tiletamine and in snare-trapped black bears anesthetized with xylazine–ketamine.[20,32] Death from heat-related causes has been reported in two black bears, of which one had a rectal temperature of $43.0^{\circ}C$.[20] Aggressive treatment of hyperthermic brown bears in the current study was necessary to prevent further temperature increase during anesthesia, which emphasizes the importance of physiologic monitoring throughout the procedure. It is also imperative to start measuring physiologic variables as early as possible after recumbency, when the effects of physical exertion, induction stress, and the anesthetic drugs are the strongest. For example, in free-ranging bears, a $pH < 7.2$ was measured in 35% of the samples collected early during anesthesia compared with in only 4% of the samples collected 30 min after darting. The time of sampling is an important variable when evaluating anesthesia, and a large sample size and serial samples can be necessary to detect physiologic alterations. There are probably no completely safe wildlife capture methods or drug combinations, but, if physiologic effects are quantified, documented, and published, then the safety for the animals can be improved by prevention or treatment of physiologic alterations.

CONCLUSIONS

Mild-to-marked alterations in pulmonary gas exchange and acid–base status were identified in brown bears anesthetized with MZT at the doses evaluated in this study. Hyperthermia and marked lactic acidemia were recorded only in free-ranging bears, whereas hypoxemia developed in both free-ranging and captive bears. Oxygen supplementation is recommended to counteract hypoxemia during anesthesia with MZT.

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