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EVALUATION OF CHEMICAL IMMOBILIZATION IN CAPTIVE BLACK BEARS (*URSUS AMERICANUS*) RECEIVING A COMBINATION OF NALBUPHINE, MEDETOMIDINE, AND AZAPERONE

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ABSTRACT: To assess potential seasonal differences in responses to immobilization, we sedated eight orphaned yearling black bears (*Ursus americanus*) being held for rehabilitation at a wildlife facility in Colorado, US, using a premixed combination of nalbuphine (40 mg/mL), azaperone (10 mg/mL), and medetomidine (10 mg/mL; NalMed-A) in October (autumn) prior to hibernation and again after emergence in May (spring) prior to their release. We dosed all bears at 1 mL NalMed-A per estimated 45 kg body mass (1 mL NalMed-A/45 kg), delivered by intramuscular injection using a pole syringe, to facilitate routine examination and ear tagging. Arterial blood gases were measured to assess oxygenation and acid-base status of bears both pre and post oxygen supplementation. The mean (SE) dose calculated post hoc was 0.9 (0.04) mg nalbuphine/kg, 0.2 (0.01) mg azaperone/kg, and 0.2 (0.01) mg medetomidine/kg. The mean induction time was 8 (1) min for six of the bears in October and 6 (1) min for eight bears in May. The NalMed-A combination provided good sedation in captive yearling black bears in autumn and spring and was effectively antagonized with a combination of naltrexone and atipamezole. Mild hypoxemia (PaO_2: 53.5–54.4 mmHg) was the most significant side effect and was corrected (PaO_2: 68.4–150.1 mmHg) with supplemental oxygen administered at 2–5 L/min for 5 min (point of sampling).

Key words: Azaperone, black bear, blood gas, chemical immobilization, medetomidine, nalbuphine, tranquilizer, *Ursus americanus*.

INTRODUCTION

American black bears (*Ursus americanus*) are routinely captured and handled by wildlife managers and researchers throughout North America. Historically, a variety of chemical compounds have been used for immobilizing black bears, but most common are combinations involving a dissociative such as tiletamine or ketamine (Gibeau and Paquet 1991; White et al. 1996). When dissociatives are used in synergistic combination with alpha-2 adrenoreceptor agonists (usually xylazine, medetomidine, or detomidine), the total drug volume often can be reduced and immobilization can be partially antagonized (Addison and Kolenosky 1979; Caulkett and Cattet 1997; Laricchiuta et al. 2008). The combination of butorphanol, azaperone, and medetomidine (BAM; Wolfe et al. 2008) also provides more-completely reversible immobilization of black bears without the use of a dissociative.

A combination of nalbuphine, azaperone, and medetomidine has shown utility for reversible chemical immobilization under field conditions in a variety of species including Rocky Mountain elk (*Cervus canadensis*; Wolfe et al. 2014) and black bears (Wolfe et al. 2014, 2016). Nalbuphine hydrogen chloride is a synthetic opioid agonist antagonist with moderate analgesic properties. Azaperone is a neuroleptic in the butyrophenone class of tranquilizers. Medetomidine is a highly specific, alpha-2 adrenoreceptor agonist with good sedative and analgesic properties. As with BAM, NalMed-A (nalbuphine, azaperone, medetomidine) immobilization can be antagonized with atipamezole and naltrexone. In addition to being an effective immobilization combination, NalMed-A lacks components subject to the US Drug Enforcement Administration's scheduling and oversight and thus should be more readily

available to field biologists and wildlife officers than are combinations involving dissociative drugs or other controlled drugs (Wolfe et al. 2014).

We evaluated the NalMed-A combination in orphaned black bears held in captivity at two handling events to assess potential physiologic differences in responses to immobilization between autumn (just prior to hibernation) and spring (post hibernation). These time periods represent the relative extremes of black bear condition in the mountains of Colorado, US, and neighboring jurisdictions. To complement a previous evaluation of this drug combination in black bears (Wolfe et al. 2016), we measured arterial blood gases both pre and post oxygen supplementation and compared the responses between October and May.

MATERIALS AND METHODS

We evaluated responses to NalMed-A (Wildlife Pharmaceuticals, Windsor, Colorado, USA) in eight orphaned yearling black bears held for rehabilitation at the Colorado Parks and Wildlife Frisco Creek Wildlife Facility in Del Norte, Colorado (37°33′18″N, 106°23′43″W; elevation 2,886 m). During most of the rehabilitation period, bears were held in chain link pens (125 m^2 or 242 m^2) with three to five bears per pen. Immediately prior to hibernation, all eight bears were moved to a single pen covered with a metal roof, measuring about 4×12 m. During the winter, seven of the eight bears hibernated in a single 2×4 m hibernaculum built of wooden pallets and hay bales and filled with grass hay while the eighth bear chose to hibernate alone in a nearby aluminum den box filled with grass hay and covered with a tarpaulin.

Prior to hibernation all bears were fed an ad libitum diet consisting mainly of a commercial diet formulated specifically for bears (Mazuri® Bear Diet, Brentwood, Missouri, USA). The commercial diet was supplemented with trout from a local hatchery, choke cherries (*Prunus virginiana*), and wild rose hips (*Rosa woodsii*) still attached to the stems. In the spring, following emergence from hibernation, bears were fed a limited diet much lower in protein. Their spring diet consisted of the first tender grasses and dandelion greens, catkins from aspen trees, and varied fruits and vegetables including raspberries, apples, oranges, melons, lettuce, spinach, carrots, and potatoes. A gradually increasing amount of commercial diet also was provided along with the vegetable matter until the bears were released later that spring.

All bears were in captivity for an average of 71 (range 23–87) d prior to the first handling event. We sedated each bear twice to facilitate physical exam and ear tagging, first in October (autumn) prior to hibernation and then again about 7 mo later in May (spring) prior to release. Handling in May was about 63 d post hibernation, although the precise day of emergence for each bear was uncertain because human activity was minimized in order to minimize habituation.

For both handling sessions, we used a premixed NalMed-A combination (40 mg nalbuphine, 10 mg azaperone, and 10 mg medetomidine/mL) prepared by Wildlife Pharmaceuticals. We delivered NalMed-A by intramuscular injection using a pole syringe (Zoolu pole syringe, Animal Care Equipment and Services, Boulder, Colorado, USA). We estimated each bear's mass by visual exam and based dosing on 1 mL of the premixed NalMed-A per estimated 45 kg body mass (target dosage: 0.9 mg/kg nalbuphine, 0.2 mg/kg azaperone, 0.2 mg/kg medetomidine).

Induction time was measured as the time (rounded to the nearest minute) from injection to sternal recumbency. We considered a bear to be fully sedated when there was no obvious response (such as ear twitch or head movement) to auditory (such as hand clapping) or noxious stimuli (such as repositioning or lifting on to a carrier). Sedated bears were blindfolded and maintained in sternal recumbency throughout immobilization. We measured body mass on a platform scale (VSSI Way, Carthage, Missouri, USA). Arterial blood samples were collected from the femoral artery in preheparinized, self-filling arterial syringes (Pro-Vent, Smiths Medical, Keen, New Hampshire, USA). We immediately processed all arterial blood samples with an Element POC® rapid blood analyzer (Heska, Loveland, Colorado, USA). Oxygen was delivered in October via portable size E oxygen cylinders (0–15 L/min click style flow selection and single stage regulator; Airgas Puritan Medical, Fort Collins, Colorado, USA) through a clear plastic nasal cannula with two outlets placed just inside the nares (Jorgensen Laboratories, Loveland, Colorado, USA). In May, we used the same cylinder and regulator but changed to a clear plastic nasal catheter inserted about 3–4 cm inside the naris (14 F×40.6 cm, Jorgensen Laboratories). Arterial samples were collected before oxygen supplementation and again approximately 5 min after beginning intranasal oxygen supplementation at a rate of 2 L/min. We maintained oxygen supplementation until the antagonist was administered. During the May handling event, the oxygen flow rate was increased to 5 L/min in three bears to

evaluate responses to a higher flow rate (approximately 5 min after the 2 L/min blood sample was taken). Heart rate (beats/min) and oxygen saturation (SpO_2) were measured with a Nellcor (Covidien, Boulder, Colorado, USA) or Masimo Rad (Masimo Corp., Irvine, California, USA) pulse oximeter using a lingual clip. Respiratory rate was measured by observing thoracic excursions. Unless otherwise noted, data are reported as mean (SE).

We subjectively monitored and scored levels of sedation throughout the handling period using jaw tone, muscle tension, and response to stimulus (based on response to elements of processing including ear tagging, sampling, and carrying the bear to the sampling area) as follows: 1=not able to handle, 2=sedate but moved away when stimulated, 3=sedate and did not move away but responded to stimulus, 4=slight response to stimulus (e.g., ear twitch), 5=no response to handling or stimulus. The overall score was the highest achieved; however, immobilized bears regressing in sedation score (e.g., from level 5 back to level 4) prior to antagonist administration received the lower score.

Once handling was completed, antagonists were administered by intramuscular hand injection at a rate of 5 mg atipamezole/mg medetomidine (5:1) and 1.3 mg naltrexone/mg nalbuphine (1.3:1). We measured recovery time as time (to the nearest minute) from antagonist injection to first attempt of lifting head up. Bears that had not recovered by 10 min were stimulated with either hand clapping or physical pressure (rocking or repositioning the bear).

The target or expected partial pressure of oxygen (PaO_2) for the study location was determined by calculating the partial pressure of alveolar oxygen (PAO_2) from the alveolar gas equation and subtracting the alveolar-arterial gradient (A-a gradient; Fahlman et al. 2012): $PaO_2 = PAO_2 - P(A\text{-}a)O_2 = F_iO_2(P_{ATM} - P_{H20}) - (PaCO_2/RQ) - P(A\text{-}a)O_2$. Using this equation, the PAO_2 for this study site elevation was 60.6 mmHg where F_iO_2=fraction of inspired O_2 (assumed 0.21), P_{ATM}=atmospheric pressure (544 mmHg at an elevation of 2,886 m), P_{H20}=water vapor pressure at 37 C (assumed 47 mmHg), $PaCO_2$=partial pressure of arterial carbon dioxide (assumed 35 mmHg), and RQ=respiratory quotient (assumed 0.8; Gessman and Nagy 1988). The alveolar-arterial oxygen gradient, or $P(A\text{-}a)O_2$, is not well established in black bears. Fahlman et al. (2012) and Mathieu et al. (2017) estimated 15 mmHg for the A-a gradient. However, because age influences the A-a gradient and all bears in this study were young, we used a conservative estimate using $P(A\text{-}a)O_2$=5 mmHg. The expected PaO_2 for black bears at our study site was 55.6 mmHg.

A two-sided Student's *t*-test was used for comparison of blood gas values between October (pre hibernation) and May (post hibernation), and between pre and post oxygen supplementation. A paired, one-sided Student's *t*-test was used to compare between 2 L/min and 5 L/min oxygen supplementation. We used alpha=0.05 to ascribe statistical difference. This study was reviewed and approved by the Colorado Parks and Wildlife Animal Care and Use Committee (file no. 08-2015).

RESULTS

All eight captive yearling black bears were similar in age and size. The mean (SE) estimated mass by visual observation in autumn (October) handling was 43 (2) kg and the measured mass was 42.5 (2.5). As expected, all bears lost weight during hibernation so the mean mass in spring (May) was 38.0 (2.8). We had estimated mass based on an expected 10% weight loss from their previous measured mass. The mean (SE) dosage for both October and May immobilizations based on measured masses was 0.9 (0.04) mg nalbuphine/kg, 0.2 (0.01) mg azaperone/kg, and 0.2 (0.01) mg medetomidine/kg. Two bears during the October handling needed supplemental injections for adequate sedation, in one case likely due to the injection site in the hind quarter (rump fat) and in the other most likely because we underestimated body mass. Mean (SE) induction for the six bears that were given a single injection in October was 8 (1) min and mean induction in May (n=8) was 6 (1) min. Induction time for one of the remaining October bears was 34 min; induction for the second bear was not observed. Mean (SE) recovery time for seven bears in October was 10 (1) min and for seven of the bears observed in May was 11 (1) min. The two remaining bears did recover, but times were not recorded.

The mean (SE) PaO_2 measured before oxygen supplementation in October was 53.6 (3.3) mmHg and in May it was 54.4 (2.0) mmHg. Both were slightly below the target PaO_2 of $\geq$55.6 mmHg and did not differ between October and May (two-sided P=0.821; Table 1). Overall, the mean PaO_2 at both sampling times increased with 2 L/min

supplemental oxygen (Table 1; P=0.046 [October] and P=0.002 [May]) and was above the target level of 55.6 mmHg and also did not differ between seasons (P=0.141). The three bears that were increased to 5 L/min supplemental oxygen showed a further increase in PaO_2 (Table 1; P=0.025) as well as in $PaCO_2$ (P=0.017). The mean (SE) calculated A-a gradient was somewhat elevated (>15 mmHg; Fahlman 2014) in October (24.3 [1.4]; Table 1) and within reported ranges for other species (Trulock 1990) in May (11.4 [1.5]). The mean (SE) $PaCO_2$ (25.6 [1.9]) in October suggested hypocapnia and a reduced mean bicarbonate (HCO_3; 17.9 [0.9]) at the same time point was likely due to a compensated respiratory alkalosis (Table 1). Both $PaCO_2$ and HCO_3 increased to normal ranges by the second arterial sample. Three bears had insufficient arterial samples for analysis, two from the October pre oxygen and one from the May post oxygen sample interval. The mean SpO_2 before oxygen supplementation was 84.2 (2.9) in October and 84.5 (2.1) in May. Measured oxygen saturation did not increase when oxygen was supplemented at 2 L/min ($P \geq 0.131$; Table 1); however, in the three bears from May that had been further supplemented with 5 L/min, measured oxygen saturation increased to 92.3 ([1.7]; Table 1).

During the October handling we subjectively scored the sedation in seven bears as level 5, meaning that there was no response to stimulus throughout the handling and sampling (including ear tag application, blood draw, repositioning the animal from side to side); one animal was scored as level 4 because slight jaw tone was noted. We scored all eight bears as level 5 during the May handling.

DISCUSSION

The NalMed-A combination provided acceptable sedation in all eight captive yearling bears in both autumn and spring, and immobilization was effectively antagonized with a combination of naltrexone and atipamezole. We observed mild hypoxemia prior to oxygen supplementation at both handling events, but this condition was effectively corrected with 2 L/min and 5 L/min of oxygen delivered via a nasal catheter or cannula. Evaluating blood gases at higher elevations can be challenging because, as barometric pressure increases, inspired oxygen tension is reduced and subsequently PAO_2 is reduced (Trulock 1990). The amount of oxygen present in the arterial bloodstream is a function of the amount of oxygen in the alveoli (PAO_2) minus some factor of loss between the alveoli and arterial blood (i.e., A-a gradient). In general, an A-a gradient in a normal healthy animal with no respiratory issues should be in the range of 5 mm to 20 mmHg and is affected by age, with younger individuals having lower A-a gradients (Trulock 1990). Because we could not find an established reference for the normal A-a gradient in black bears, and all bears in our study were young, we regarded a conservative estimate of 5 mmHg as appropriate. As a result, an expected PaO_2 of 55.6 at the study elevation may be an overestimate. The SpO_2 values were below 90% prior to and after supplementation with 2 L/min oxygen. The SpO_2 did not exceed 90% in immobilized bears until we supplemented with 5 L/min oxygen. Lack of a demonstrable increase in SpO_2 corresponding to the increased PaO_2 was surprising because SpO_2 often overestimates oxygenation. It is worth noting that we based mean SpO_2 on a single measurement at a specified time point rather than using an average over several minutes, which may have been more reflective of true SpO_2. In addition, Luks and Swenson (2011) list factors such as altitude, movement, probe fit, and poor perfusion as affecting the accuracy of the pulse oximeter readings.

It is important to note that blood gas data were not corrected for rectal temperature because corresponding temperatures were not available at all sampling points in October. The solubility of oxygen and carbon dioxide in water changes with temperature. As temperature increases, the solubility decreases, leading to lower PaO_2 and $PaCO_2$ measurements (Bacher 2005). Our blood gas analyzer

TABLE 1. Vital measurements, hemoglobin saturation, and arterial blood gas values in eight captive black bears (*Ursus americanus*) sedated with a combination of nalbuphine, azaperone, and medetomidine at two handling events in October and May (before and after hibernation). Values are not temperature corrected. All eight bears were supplemented with nasal oxygen at 2 L/min and three of the bears were then increased to 5 L/min when immobilized in May.[a]

	October sampling				May sampling					
	Pre oxygen supplementation		Post 2 L/min oxygen supplementation		Pre oxygen supplementation		Post 2 L/min oxygen supplementation		Post 5 L/min oxygen supplementation	
	n	Mean (SE)	*n*	Mean (SE)	*n*	Mean (SE)	*n*	Mean (SE)	*n*	Mean (SE)
pH	6	7.4 (0.01)	8	7.4 (0.01)	8	7.4 (0.02)	7	7.4 (0.01)	3	7.3 (0.01)
$PaCO_2$ (mmHg)	6	25.6 (1.9)	8	37.0 (1.4)	8	29.2 (2.3)	7	35.0 (2.0)	3	39.9 (0.9)
PaO_2 (mmHg)	6	53.6 (3.3)	8	68.4 (7.2)	8	54.4 (2.0)	7	84.6 (7.3)	3	150.1 (8.1)
PAO_2 (mmHg)	6	77.9 (2.3)	8	110.7 (1.7)	8	65.5 (2.8)	7	102.4 (2.3)	3	144.7 (1.3)
A-a gradient (mmHg)	6	24.3 (1.4)	8	NA	8	11.4 (1.5)		NA		NA
SpO_2 (%)	6	84.2 (2.9)	8	88.1 (1.9)	8	84.5 (2.1)	7	85.9 (2.0)	3	92.3 (1.7)
$cHCO_3^-$ (mmol/L)	6	17.0 (0.9)	8	21.1 (0.6)	8	17.0 (0.7)	7	19.3 (0.5)	3	21.2 (0.4)
BE(b) (mmol/L)	6	−5.1 (0.5)	8	−2.6 (1.2)	8	−6.6 (0.4)	7	−5.3 (0.2)	3	−4.4 (0.3)
cSO_2 (%)	6	88 (2.2)	8	90.3 (2.5)	8	87 (1.7)	7	94.9 (1.4)	3	99.2 (0.1)
AGapK (mmol/L)	6	18 (0.6)	7	14 (.9)	8	13.3 (0.6)	7	11 (0.4)	3	10.7 (0.3)
Heart rate (beats/min)	8	61 (7)	8	49 (3)	8	52 (3)	7	51 (3)	NA	ND
Respiratory rate (breaths/min)	8	15 (2)	8	15 (1)	8	10 (0.5)	NA	9 (1)	NA	ND
Rectal temperature (C)	4	37.9 (0.4)	NA	ND	8	38.6 (0.2)	NA	38.4 (0.2)	NA	ND

[a] $PaCO_2$ = arterial carbon dioxide; PaO_2 = arterial oxygen; PAO_2 = alveolar oxygen; A-a gradient = alveolar arterial gradient; SpO_2 = hemoglobin saturation; $cHCO_3-$ = calculated bicarbonate; BE(b) = base excess in blood; cSO_2 = calculated hemoglobin saturation; AGapK = anion gap; NA = not applicable; ND = not done.

was optimized to evaluate samples at a temperature of 37 C. Mean (SE) rectal temperatures were 38.0 (0.4) C for four bears in October and 38.5 (0.2) C for bears in May, so blood gas analytes were likely somewhat higher than reported. Bears sampled in May showed a better response to oxygen supplementation than did bears in October (Table 1), which seems most likely due to our using a longer nasal catheter rather than a nasal cannula in May. Three bears given oxygen at a rate of 5 L/min showed a marked increase in oxygen saturation, although we believe flow rates that high are likely unnecessary with this drug combination. The elevated A-a gradient in October may reflect hypoventilation as well as a ventilation-perfusion mismatch or physiologic shunt that are often associated with alpha-2 adrenoreceptor agonists (Read 2003). Hypoxemia is a common side effect in wildlife immobilization and was seen in bears with other immobilizing combinations such as medetomidine, zolazepam, and tiletamine (Caulkett and Cattet 1997; Fahlman et al. 2012) and butorphanol, azaperone, and medetomidine (Wolfe et al. 2008), and is likely associated with hypoventilation (Evans et al. 2012).

The initial hypocapnia that we observed was an unexpected finding but may be related to a reduced PaO_2 and a compensatory increase in alveolar ventilation. Hypocapnia also can be associated with hyperventilating (increased clearance carbon dioxide; Muir 2015) and we suspect that, in our study, the observed hypocapnia may have been the result of bears "chuffing" in response to human activity prior to sedation. The subsequent increase in carbon dioxide over time was most likely the result of hypoventilation associated with sedation-induced depression of the respiratory center.

Overall, NalMed-A provided reversible, good-quality sedation with a low delivery volume in captive black bears. Hypoxemia was a side effect in some individuals and we recommend oxygen supplementation for bears and other species immobilized with this drug combination.

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