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THE USE OF KETAMINE-XYLAZINE OR BUTORPHANOL-AZAPERONE-MEDETOMIDINE TO IMMOBILIZE AMERICAN BLACK BEARS (*URSUS AMERICANUS*)

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ABSTRACT: Wildlife anesthetic protocols must offer rapid inductions and recoveries, be physiologically safe, and be minimally regulated. With this in mind, we evaluated differences in induction and recovery times and physiological parameters in 33 American black bears (*Ursus americanus*) anesthetized with ketamine-xylazine (KX) or immobilized with a commercial drug combination of butorphanol, azaperone, and medetomidine (BAM). Dose was based on mass estimated from field observations. Bears were housed at Appalachian Bear Rescue, Townsend, Tennessee, US, or free-ranging within the Great Smoky Mountains National Park (Tennessee and North Carolina, US) and chemically immobilized for management purposes. From 11 April to 29 June 2016, we immobilized bears with injection via pole syringe or disposable dart projected from an air-powered dart rifle. Once immobilized, we measured each bear's temperature, respiration (breaths/min), heart rate (beats/min), hemoglobin oxygen saturation (via pulse oximetry), arterial blood gases, and mass (kg). We found no differences in the induction parameters, partial pressures of CO_2, and rectal temperatures. The BAM-treated bears had lower heart and respiratory rates that led to lower hemoglobin oxygen saturation levels (from blood gas analysis, SaO_2). The SaO_2 after treatment with BAM (91.1±0.8%) was lower than with KX (93.4±0.9%). After handling, we reversed KX-treated bears with a $\bar{x}$=0.2±0.02 mg/kg yohimbine and BAM-treated bears with $\bar{x}$=1.5±0.1 mg/kg atipamezole and 0.8±0.1 mg/kg naltrexone. We found no differences in the recovery times to increased respiration and to the bear assuming a head-up position. The BAM-treated bears stood and recovered quicker than did KX-treated animals. Based on our observations, BAM appears to offer safe, predictable immobilizations with fewer drawbacks and faster recovery times than KX-treated bears.

Key words: Arterial blood gases, BAM, black bear, chemical immobilization, pulse oximetry, *Ursus americanus*.

INTRODUCTION

Capture and handling of American black bears (*Ursus americanus*) are often necessary for research and management. Ketamine-xylazine (KX) or tiletamine-zolazepam-xylazine drug combinations have long been the standards for immobilizing black bears, yet both have undesirable side effects for management purposes (Addison and Kolenosky 1979; Stewart et al. 1980; Cook 1984). Ketamine/xylazine (at a ratio of 2:1) offers rapid induction and moderately fast recoveries and has a high therapeutic index, but often bears have problems with thermoregulation and spontaneous arousals (Cattet et al. 1999). Antagonizing the xylazine with yohimbine hydrochloride decreases recovery times, although an early reversal leads to rough recoveries associated with ketamine immobilization (Ramsay et al. 1985; Garshelis et al. 1987). The tiletamine-zolazepam-xylazine combination produces an effective, predictable, and safe immobilization, but extended recoveries are problematic for bear management activities (White et al. 1996). Replacing xylazine with medetomidine in the tiletamine/zolazepam mixture allowed lower dosages of the tiletamine/zolazepam and rapid reversal of the medetomidine with atipamezole in black bears (Caulkett and Cattet 1997). However, the authors reported bears were disoriented, possibly due to the residual effects of tiletamine/zolazepam. The US Drug Enforcement Agency (DEA) regulates both ketamine and

tiletamine-zolazepam as schedule III–controlled substances.

Wolfe et al. (2008) found a combination of butorphanol tartrate, azaperone tartrate, and medetomidine HCl (BAM) produced reliable and reversible anesthesia in black bears. Butorphanol is regulated as a schedule IV opioid drug (DEA). The remaining components of BAM are not regulated by DEA. The BAM combination is partially reversible, cost effective, and potent in small volumes, and it has application in multiple species. These advantages may offer agencies and field personnel alternative options to traditional bear anesthesia in the field.

Butorphanol, azaperone, and medetomidine act synergistically on the central nervous system to immobilize wildlife (Kreeger et al. 1989). The butorphanol produces only mild sedation and analgesia when used alone but when combined with an α_2-adrenergic agonist (e.g., xylazine, medetomidine) has produced profound sedation (Kreeger et al. 1989). Butorphanol can be antagonized with naltrexone. However, butorphanol may offer postcapture analgesic effects in captive animals when reversal is not necessary (Wolfe et al. 2008). Azaperone, a butyrophenone, has been used as a sedative in the swine industry to reduce aggression. Azaperone combined with an opioid (e.g., butorphanol, etorphine) or cyclohexane produces smoothing effects that shorten induction and reduce the amount of primary immobilizing agent needed (Colly 1992; Kreeger and Arnemo 2012). Medetomidine is a potent α_2-adrenergic agonist that can be completely antagonized (Kreeger and Arnemo 2012). Medetomidine can cause hypoxemia in wild ruminants and multiple species of bears (Read 2003). Medetomidine caused bradycardia in polar bear (*Ursus maritimus*) under ketamine-medetomidine combinations (Caulkett et al. 1999). Caulkett and Cattet (1997) reported mild bradycardia and hypoxemia in black bears with medetomidine-tiletamine-zolazepam treatment.

Physiological monitoring is critical when immobilizing wildlife. Portable pulse oximeters have been used to measure hemoglobin oxygen saturation (SpO_2) in the field. However, Caulkett and Fahlman (2014) reviewed work with polar and brown bears (*Ursus arctos*) and found pulse oximetry may overestimate SpO_2 at lower ranges of partial pressure of arterial oxygen. Therefore, arterial blood gas measurements may be required for accurate physiological monitoring until the full efficacy and safety of a chemical immobilization agent can be characterized and the accuracy of portable pulse oximetry is verified.

Recent research has focused on anesthetic regimes that minimize DEA oversight, reduce record keeping, and lessen storage restrictions. Several drug combinations require no DEA oversight but seem to extend induction times (Wolfe et al. 2014, 2016) making immobilization of free-ranging, wild bears difficult. Our purpose was to compare chemical immobilization with KX, the traditional protocol currently used by Great Smoky Mountains National Park biologists, and BAM with appropriate antagonists on induction times, recovery times, and physiological effects to determine the best option for immobilizing American black bears in the field. We also wanted to compare hemoglobin oxygen saturation measured by two different methods (arterial blood gas analysis and pulse oximetry) to determine the best approach for physiological monitoring.

MATERIALS AND METHODS

The University of Tennessee Institutional Animal Care and Use Committee (2451-0416) approved all animal procedures. We anesthetized 24 orphaned, yearling black bears that were rehabilitated at Appalachian Bear Rescue Facility (ABR; 35°40′N, 83°47′W; elevation 354 m), Townsend, Tennessee, US, in May or June 2016. The ABR bears were housed in 35×35 m outdoor enclosures until time of capture. We transferred the bears to a small acclimation pen or lured them within darting range for immobilization at ABR. We also captured nine free-ranging black bears in Great Smoky Mountains National Park in Tennessee and North Carolina, US (elevation 396–1,463 m) by culvert trap or dart projector with disposable darts. We used intramuscular (IM) injection in shoulders or hindquarters using either a pole syringe (Cap-Chur, Powder Springs, Georgia, USA), darting (Dan-Inject, Dan-Inject North America, Fort Collins, Colorado, USA with

darts from Pneu-Dart, Williamsport, Pennsylvania, USA), or hand injection with needle and syringe.

We estimated body mass based on size and condition of bears for calculating the drug dose. We weighed ABR and free-ranging bears to the nearest kilogram after immobilization. We used KX in initial trials due to drug availability and added BAM treatments later. We randomly assigned the KX or BAM protocol to each capture that occurred after the initial KX trials.

Bears were immobilized with an estimated 6.6 mg/kg of ketamine (200 mg/mL, ZooPharm, Fort Collins, Colorado, USA) and 3.3 mg/kg xylazine (300 mg/mL, ZooPharm) or 0.5 mL 23 kg BAM. We used commercial BAM (ZooPharm) that contained 27.3 mg/mL butorphanol tartrate, 9.1 mg/mL azaperone tartrate, and 10.9 mg/mL medetomidine HCL, for an estimated dosage of 0.6 mg/kg butorphanol, 0.2 mg/kg azaperone, and 0.2 mg/kg medetomidine. We measured the time (to the nearest minute) to first effect (misstep, loss of coordination), to sternal recumbency with responsiveness, to lateral recumbency (bear immobilized), and to time of approach. We measured rectal temperature (Vet-Temp DT-10, Advanced Monitoring Corp., San Diego, California, USA), respiration (from thoracic movements), and peripheral capillary oxygen saturation (SpO_2) and heart rate (using pulse oximetry; PM-60Vet, Shenzhen Mindray Bio-Medical Electronics, Shenzhen, China), beginning when immobilization was verified and the bear had been moved to a processing facility or to level ground (>5 min). The pulse oximeter probe was placed on the tongue, labia, or prepuce. We collected <1 mL of blood from the femoral artery into heparinized syringes and processed samples immediately to measure arterial blood gases (IDEXX VetStat Analyzer, Westbrook, Maine, USA). We recorded arterial partial pressure of oxygen (mmHg), arterial partial pressure of CO_2 (mmHg), and blood pH. Hemoglobin oxygen saturation as measured by the VetStat analyzer (SaO_2) was validated for humans. For the ABR bears, we performed a prerelease examination, attached a Global Positioning System collar (Vectronic Aerospace GmbH, GPS Iridium, Berlin, Germany), and applied a lip tattoo. Bears captured in Great Smoky Mountains National Park were marked similarly to the ABR bears, with an additional passive integrated transponder tag, and we removed the first premolar for aging. These wild bears were released onsite after marking and handling.

Upon completion of the procedures, we moved the bears into a transfer cage or placed the bear in a safe area and administered 0.2 mg/kg of the antagonist yohimbine (ZooPharm) intravenously in the femoral vein to those that were immobilized with KX. For bears immobilized with BAM, we administered atipamezole (25 mg/mL stock solution; ZooPharm) at twice the volume of initial BAM dose and naltrexone (50 mg/mL stock solution; ZooPharm) at a dose of 0.5 mL per animal intramuscularly. Bears were physically stimulated with slight pressure from a blunt pole 5 min after the antagonists were administered. Recovery was measured to the nearest minute at the first effect (change in respiration) of drug antagonism, when the bear recovered its ability to hold its head up, when it stood up, and at full recovery.

We tested physiological parameters and timing for normality with a Shapiro-Wilk test (Cody and Smith 2006). We used a *t*-test (PROC TTEST, SAS 9.4; Cody and Smith 2006) for those parameters that were normally distributed. We evaluated the equality of variance and used the appropriate *t*-test *P*-value for equal or unequal variance. For parameters that were not normally distributed, we used a nonparametric test, the Wilcoxon two-sample test (PROC NPAR1WAY; Cody and Smith 2006). We considered significance level at alpha 0.05, and means are reported ($\pm$SE). We compared SpO_2 and SaO_2 using the Bland-Altman method (Bland and Altman 1986), similarly to Muller et al. (2012) in SAS 9.4 (SAS Institute, Cary, North Carolina, USA).

RESULTS

For ABR yearling bears, we used KX on 12 (eight males and four females) from 11 to 12 April 2016 and BAM on 12 (six males and six females) from 13 April to 10 June 2016. For wild bears, we used KX on three (two males and one female) from 17 June to 16 July 2016 and BAM on six males from 12 June to 16 July 2016. The final dosage $\bar{x}$=6.9$\pm$0.4 mg/kg for ketamine and $\bar{x}$=3.5$\pm$0.2 mg/kg xylazine calculated after weighing bears was close to the estimated dosage. The dosage for BAM-treated bears was $\bar{x}$=0.9$\pm$0.1 mg/kg butorphanol, $\bar{x}$=0.3$\pm$0.02 mg/kg azaperone, and $\bar{x}$=0.3$\pm$0.02 mg/kg medetomidine, or about 40% higher than the estimate.

Ten (66%) of the 15 KX-treated bears were successfully immobilized with one injection (nine by pole syringe and one by dart). Four bears (26%) required additional drugs to achieve sufficient depth of anesthesia. One bear (6%) injected with a pole syringe and initially immobilized required an additional

TABLE 1. Comparisons of drug effects and physiological parameters for bears (*Ursus americanus*). Bears were injected with ketamine/xylazine ($\bar{x}$=6.9±0.4 mg/kg ketamine and $\bar{x}$=3.5±0.2 mg/kg xylazine) reversed with $\bar{x}$=0.2±0.02 mg/kg yohimbine or BAM ($\bar{x}$=0.9±0.1 mg/kg butorphanol, $\bar{x}$=0.3±0.02 mg/kg azaperone, and $\bar{x}$=0.3±0.02 mg/kg medetomidine) reversed with $\bar{x}$=1.5±0.1 mg/kg atipamezole and $\bar{x}$=0.8±0.1 mg/kg naltrexone from 11 April to 16 June 2016 at the Appalachian Bear Rescue, Townsend, Tennessee, USA, and Great Smoky Mountains National Park, Tennessee, and North Carolina, USA.

Measurement	BAM		Ketamine-xylazine		
	Mean (*n*)	SE	Mean (*n*)	SE	*P*
Time to stages of immobilization (min)					
First effect	1.8 (18)	0.2	2.7 (15)	0.7	1.000[a]
Sternal recumbency	3.6 (18)	0.4	5.2 (15)	1.5	0.530[a]
Lateral recumbency	4.6 (18)	0.4	6.4 (15)	1.6	0.934[a]
Able to approach	6.2 (18)	0.4	7.6 (15)	1.8	0.364[a]
Increased respiration after reversal	8.4 (18)	1.6	7.0 (11)	2.0	0.240[a]
Head-up after reversal	13.3 (18)	2.0	17.5 (11)	2.8	0.191[a]
Standing after reversal	19.3 (18)	2.9	36.0 (11)	5.1	0.005[a]
Total recovery time after reversal	19.4 (18)	3.0	36.0 (11)	5.1	0.007[a]
Physiological parameters					
SaO_2 (%)[b]	91.1 (17)	0.8	93.4 (14)	0.9	0.053[c]
pH	7.39 (17)	0.01	7.42 (14)	0.01	0.007[c]
$PaCO_2$ (mmHg)[d]	37.24 (17)	1.08	38.57 (14)	1.05	0.387[c]
HCO_3	20.66 (17)	0.50	23.06 (14)	0.58	0.004[c]
Rectal temperature (C)	37.9 (18)	0.1	37.6 (15)	0.2	0.205[c]
Heart rate (beats/min)	53.3 (15)	2.8	93.8 (15)	4.2	<0.001[c]
Respiratory rate	14.1 (18)	1.4	21.4 (15)	1.6	0.002[c]

[a] *P* value from Wilcoxon two-sample test.

[b] SaO_2 = hemoglobin saturation from blood gas analysis.

[c] *P* value from *t*-test.

[d] $PaCO_2$ = partial pressure of CO_2.

dose of ketamine after partial arousal from anesthesia during processing. Seventeen (94%) of the 18 BAM-treated bears were effectively immobilized with one injection (14 by pole syringe and four by dart). The remaining bear injected with a needle and syringe was not completely immobilized at 20 min after injection, so we gave it an additional dose (0.3 mL/23 kg) of BAM to allow processing. This bear may have received a partial dose initially.

The induction parameters, times to first effect, sternal recumbency, lateral recumbency, and approach, did not differ between bears treated with KX and with BAM (Table 1). Qualitatively, there were differences between treatments: BAM-treated individuals exhibited head droop and extension and laxation of the tongue, whereas KX-treated animals exhibited nystagmus and excessive licking.

Not surprisingly, the mean heart rate for BAM-treated bears was 53.3±2.8 compared to 93.8±4.2 for KX-treated bears (Table 1). The higher rate for KX-treated bears was probably because cyclohexane drugs such as ketamine tend to increase heart rate (Kreeger and Arnemo 2012). Respiratory rate (breaths/min) was lower for BAM-compared to KX-treated bears (Table 1). The respiration rate of BAM-treated bears was also irregular with periods of apnea. The peripheral vasoconstriction associated with medetomidine also made obtaining arterial blood much more difficult. Hemoglobin saturation from blood gas analysis was lower with BAM than with KX (Table 1), although no animals had an SaO_2 <89.5%. We did not detect differences in partial pressure of CO_2 nor rectal temperature, and no animals exhibited hyperthermia (≥40 C; Fahlman et al. 2011). Bicarbonate

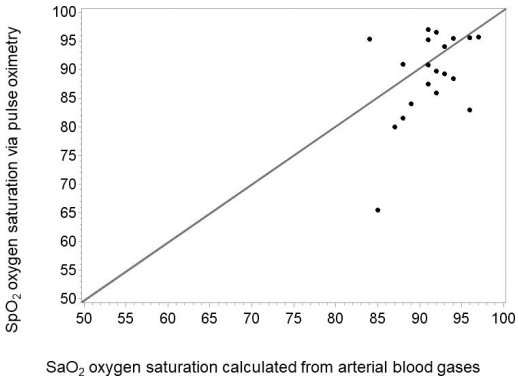

FIGURE 1. Comparison of hemoglobin oxygen saturation using a Bland-Altman analysis plot of identity. Oxygen saturation was measured by two methods, pulse oximeter (SpO_2) and arterial blood gases (SaO_2), in American black bears (*Ursus americanus*). Bears were injected with ketamine/xylazine ($\bar{x}$=6.9±0.4 mg/kg ketamine and $\bar{x}$=3.5±0.2 mg/kg xylazine) reversed with $\bar{x}$=0.2±0.02 mg/kg yohimbine or BAM ($\bar{x}$=0.9±0.1 mg/kg butorphanol, $\bar{x}$=0.3±0.02 mg/kg azaperone, and $\bar{x}$=0.3±0.02 mg/kg medetomidine) reversed with $\bar{x}$=1.5±0.1 mg/kg atipamezole and $\bar{x}$=0.8±0.1 mg/kg naltrexone from 11 April to 16 June 2016 at the Appalachian Bear Rescue, Townsend, Tennessee, USA, and Great Smoky Mountains National Park, Tennessee and North Carolina, USA. The solid black line is the line of identity where SaO_2=SpO_2.

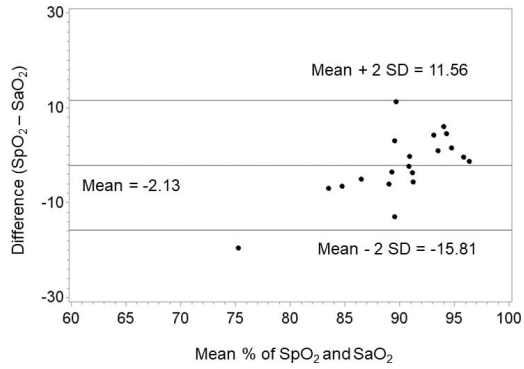

FIGURE 2. Comparison of hemoglobin oxygen saturation using a Bland-Altman analysis. Oxygen saturation was measured by two methods, pulse oximeter (SpO_2) and arterial blood gases (SaO_2), in American black bears (*Ursus americanus*). Bears were injected with ketamine/xylazine ($\bar{x}$=6.9±0.4 mg/kg ketamine and $\bar{x}$=3.5±0.2 mg/kg xylazine) reversed with $\bar{x}$=0.2±0.02 mg/kg yohimbine or BAM ($\bar{x}$=0.9±0.1 mg/kg butorphanol, $\bar{x}$=0.3±0.02 mg/kg azaperone, and $\bar{x}$=0.3±0.02 mg/kg medetomidine) reversed with $\bar{x}$=1.5±0.1 mg/kg atipamezole and $\bar{x}$=0.8±0.1 mg/kg naltrexone from 11 April to 16 June 2016 at the Appalachian Bear Rescue, Townsend, Tennessee, USA, and Great Smoky Mountains National Park, Tennessee and North Carolina, USA. Both methods provided clinically useful measurements when the mean of the two measurements was above 83%.

(HCO_3) and blood pH differed between treatments with BAM being more acidic and HCO_3 lower than KX (Table 1).

Although there was general agreement on hemoglobin oxygen saturation for both methods (n=20 for both measurements), SaO_2 was higher than SpO_2 below 89% (Figs. 1, 2). Above 93%, SpO_2 exceeded the value obtained from SaO_2 (Figs. 1, 2). Both methods provided clinically useful measurements when the mean of the two measurements was above 83%. Since we were not always able to measure SpO_2 (n=13), we reported SaO_2 for comparison between the two protocols (Table 1).

We administered the appropriate antagonists for each drug combination after bears were processed and measurements were completed ($\bar{x}$=35.7±6.3 min; range=18–65 min). The KX-treated bears were reversed with $\bar{x}$=0.2±0.02 mg/kg yohimbine and BAM-treated bears with $\bar{x}$=1.5±0.1 mg/kg atipamezole and $\bar{x}$=0.8±0.1 mg/kg naltrexone. Times to the recovery stages, increased respiration, and head-up positions did not differ between bears treated with KX and with BAM (Table 1). However, time to standing after reversal and total recovery were faster for bears treated with BAM than with KX (Table 1). Bears in both treatments received tactile stimulus 5 min after reversal injection to speed recovery.

DISCUSSION

Our processing times averaged 35.7±6.3 min from approach to reversal. Both KX and BAM provided effective and safe immobilization for American black bears during the handling period. As part of additional research projects, no documented mortality events or unusual movements were noted during continual monitoring of these bears for greater

than 6 mo. The bears treated with BAM were mildly hypoxemic, but the physiological measurements were above those considered easily tolerated in healthy black bears (Caulkett and Cattet 1997). Cattet et al. (2003) considered an SpO2 of less than 85% as the point where grizzly bears showed clinical signs of hypoxia and oxygen supplementation was required. The lowest SaO2 for a BAM-treated bear was 89.5%.

The actual dose for KX we used was close to the estimated dose; however, the actual dose for BAM was almost 40% higher than the estimated dose. Overdosing with a drug such as medetomidine would affect the hypoxia as well as the rapid induction times, yet that higher drug dose did not compromise the bears. Estimation of body mass was difficult to do in the field and could have led to overdosing BAM due to the small volume of the drug combination used.

In order to mimic backcountry bear capture where access is difficult, we did not treat any bears with supplemental oxygen. This approach seemed acceptable for short durations of handling, based on our physiological monitoring. However, if longer immobilization periods are required (e.g., animal relocation, family capture, difficult processing), more research may be needed to assess physiological parameters under BAM anesthesia. Lower respiration and heart rates with resultant hypoxemia may be problematic with extended periods after treatment with BAM. Additionally, research should evaluate the effects of BAM on hibernating bears because of the seasonal decline in metabolic rate.

The advantages of BAM over KX included lower volume for darting, faster recovery after handling, and a lower DEA schedule. For a 23-kg bear we used 1.05 mL for KX and 0.5 mL for BAM at the concentrations of each drug commercially available. Reduction by half of the volume of the drug given reduces dart size and impact velocity while increasing long-range ballistics making BAM preferable for free range darting. Total recovery time was shorter with less variability for BAM-treated bears (Table 1).

Tactile stimulation 5 min after drug antagonism sped the recovery of animals. Qualitatively, bears treated with KX and reversed with yohimbine exhibited uncoordinated standing and apparent ataxia while attempting to walk. The recovery of BAM-treated bears after reversal was consistently smooth and quick with no loss of coordination or stumbling when standing or walking. These BAM-treated animals typically stood and walked or ran away immediately after raising their heads. If recovery is prolonged with BAM treatment, we recommend an additional half dose of atipamezole given 20 min after the initial reversal dose. Only one bear was given an additional dose of atipamezole during our research.

Individual variability of each bear must be considered when analyzing mode of action for chemical immobilization. The majority of these bears were rehabilitated juveniles, housed in a captive facility with minimal human contact. Due to their lack of human interaction, the captive bears possessed a natural fear of people. The interactions with humans resulted in variable levels of stress, which could affect dosage needed and injection method available. Although induction appeared complete, several bears were still able to respond to tactile stimulus after injection. There were four BAM-treated bears (26%) that required additional drugs to complete immobilization. The additional doses may have been needed due to incomplete injection, subcutaneous injection, injection into adipose tissue, or variable physiological stress levels of captured bears (Addison and Kolenosky 1979).

Food was constant and readily available at the ABR facility, which resulted in yearling bears with unnatural layers of adipose tissue for their age class. Estimating these uncommon juvenile bear masses led to inflated BAM doses. This may be an important consideration when intramuscularly administering pharmaceuticals. Wolfe et al. (2016) recommended IM injections of BAM be targeted at the shoulder or legs, due to medetomidine being lipophilic. Age class can directly affect dose with juvenile animals requiring more and

senior animals requiring less for immobilization (Kreeger and Arnemo 2012). Adequate needle length to penetrate beyond poorly vascularized tissue also should be considered based on seasonality and morphological parameters.

We experienced difficulty obtaining a reliable reading using a pulse oximeter on the yearling bears. In adults, we were able to use the probes on the tongue. This attachment was not always effective in the yearling bears, so we attached leads to the labia and the prepuce with variable results. We also tried different pulse oximetry units with varying success. When comparing the pulse oximeter to the blood gas analyzer, both methods provided clinically useful measurements when the mean of the two measurements was above 83%. However, there was great variability in the readings with the pulse oximeter. We were successful in collecting arterial blood and using the portable blood gas analyzer, but we realize the equipment may not be readily available and may be difficult to use under most field conditions.

Choice of immobilizing agents may also depend on human safety for personnel working with the drugs. Human exposure to the medetomidine and butorphanol components of BAM may severely affect human respiratory and cardiorespiratory function and require medical assistance. In humans, exposure to opioids such as butorphanol can be reversed with naloxone (Kreeger and Arnemo 2012). While atipamezole will reverse the effects of medetomidine in animals, it is not approved for use in humans. However, human trials have been conducted with atipamezole reversal of dexmedetomidine without severe side effects (Pertovaara et al. 2005). Recent research by Greenberg et al. (2017) provides a recommended dose of 100 mg atipamezole IM for a significant medetomidine exposure where medical aid is not available. Every agency should have human drug exposure protocols in place and should have discussed those with emergency medical facilities prior to any incident. Human safety after animal workup is also important for any bears that may be potentially harvested or consumed. Recent research found no evidence of butorphanol, azaperone, medetomidine, or atipamezole within white-tailed deer (*Odocoileus virginianus*) muscle or liver 11 d after immobilization with BAM (Cook et al. 2016). Short retention time of BAM in the tissue may allow agencies more time to address management issues after immobilization while maintaining the utilization of harvested bears on the landscape.

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