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BUTORPHANOL-AZAPERONE-MEDETOMIDINE FOR IMMOBILIZATION OF CAPTIVE WHITE-TAILED DEER

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ABSTRACT: Drug combinations are commonly used to immobilize white-tailed deer (*Odocoileus virginianus*) for capture or handling. Although efficacy of various compatible and complementary drugs has been tested in clinical trials with deer, extensive negative side effects, impractical drug volume, and slow recovery from immobilization sometimes make these combinations less than ideal for routine field use. We hypothesized that a combination of butorphanol, azaperone, and medetomidine (BAM) would provide safe and effective immobilization of captive white-tailed deer while minimizing these complicating factors. We tested two dosages of this drug combination (BAM-1 and BAM-2) and two dosages of a naltrexone, tolazoline, and atipamezole antagonist combination (NTA-1 and NTA-2) with captive white-tailed deer. We characterized efficacy of drug for immobilization, quality of drug induction, and recovery after drug reversal, and we compared our findings with those of previous drug trials. Complete immobilization and excellent induction quality was achieved with a low volume dosage of BAM-2. Time to drug induction and deer recumbency for BAM-2 compared favorably with results from previous trials involving xylazine/ketamine and medetomidine/ketamine but without risk of hyperthermia. We found no differences in time to deer recovery for NTA-1 and NTA-2, with deer treated with either combination standing by 30 min postinjection. Regardless of immobilizing drugs used, we suggest practitioners monitor for signs of circulatory deficiency in deer and provide supplemental oxygen when needed.

Key words: Azaperone, BAM, butorphanol, immobilization, medetomidine, *Odocoileus virginianus*, white-tailed deer.

INTRODUCTION

Drugs have been used to immobilize free-ranging and captive deer since the 1950s. Early research focused on neuromuscular blocking agents, like d-tubocurarine, gallamine, and succinylcholine, or ganglionic stimulators, like nicotine (Jones, 1973). Although immobilization of deer with these drugs was reported (Hall et al., 1953; Jenkins, 1955; Crockford, 1957; Post, 1959; Miller, 1968), extensive negative side effects and high deer mortality limited their usefulness (Jones, 1973). During the 1960s, practitioners began using drug combinations that depressed an animal's central nervous system, providing relatively safe, humane, and sometimes reversible anesthesia (Jones, 1973; Wenkler, 1998). Today, combinations of compatible and complementary drugs routinely are used for immobilizing ungulates (Wenkler, 1998; Walsh and Wilson, 2002; Caulkett and Haigh, 2004). Route and volume of delivery, duration and quality of anesthesia, potential side effects, and availability of antagonists are important considerations when selecting drug combinations (Wenkler, 1998).

For >30 yr, xylazine, a powerful α_2-adrenergic agonist, has been used to immobilize deer (Roughton, 1975; Seal and Bush, 1987; Caulkett and Haigh, 2004). Xylazine is partially reversed by available antagonists and use of this prescription drug in deer has been approved by the US Food and Drug Administration (Greene and Thurmon, 1988; Webb et al., 2004). When combined with schedule II opioids (like etorphine or carfentanil) or schedule III cyclohexamines (like ketamine or tiletamine), xylazine works synergistically, improving efficacy and reducing drug volume (Wenkler, 1998; Kilpatrick and Spohr, 1999; Walsh and Wilson, 2002). However in deer, xylazine and drug combinations including

xylazine are known to cause acidemia, bloating and regurgitation, bradycardia, temperature regulation problems, hypoxemia, glycosuria, and anorexia (DelGiudice et al., 1988; Kreeger, 2000; Read et al., 2001; Miller et al., 2003; Storms et al., 2005, 2006). Furthermore, a metabolite of xylazine is carcinogenic in rats (Chamberlain and Brynes, 1998), and researchers caution about the use of xylazine in deer intended for human consumption (Walsh and Wilson, 2002).

The prescription drug medetomidine, the most site specific α_2-adrenergic agonist, has been suggested as an alternative to xylazine for veterinary use because it is more potent and reliable, is quickly reversed with an α_2-antagonist, and is unlikely to be carcinogenic (Virtanen et al., 1988; Nielsen, 1999; Walsh and Wilson, 2002). Thus far, medetomidine or drug combinations including medetomidine have been tested with axis deer (*Axis axis*), fallow deer (*Dama dama*), hog deer (*Axis porcinus*), mule deer (*Odocoileus hemionus*), red deer (*Cervus elaphus*), reindeer (*Rangifer tarandus*), sika deer (*Cervus Nippon*), and white-tailed deer (Wolkers et al., 1994; Ranheim et al., 1997; Tsuruga et al., 1999; Caulkett et al., 2000; Fernandez-Moran et al., 2000; Millspaugh et al., 2004; Arnemo et al., 2005; Smith et al., 2006; Muller et al., 2007).

Butorphanol, a morphine-based, schedule IV opioid, has been combined with α_2-adrenergic agonists, like medetomidine or xylazine, to achieve synergistic central nervous system sedation of captive wildlife (Kreeger et al., 1989). However, to our knowledge, butorphanol has been tested only as a postsurgical analgesic in white-tailed deer (Posner et al., 2005). Likewise, although the prescription drug azaperone, a neuroleptic sedative, has been combined with xylazine and/or fentanyl to immobilize fallow deer (Haigh, 1977; Wilson et al., 1996) and with etorphine to immobilize mule deer (Krausmann et al., 1986), its use in white-tailed deer is limited, to our knowledge, to a single report without characterization of resulting immobilizations (Read and McCorkell, 2002).

We believed medetomidine in combination with butorphanol and azaperone (BAM) might provide a low-volume, reversible alternative to other immobilizing drug combinations. We evaluated this combination for immobilization of captive white-tailed deer at two dose levels in an effort to demonstrate an effective dose rate. We measured efficacy of the combination to immobilize deer, characterized quality of induction, and characterized quality of recovery following administration of tolazoline, atipamezole, and naltrexone antagonists. We qualitatively compared our findings to those of similar, published experiments conducted by members of our research team on the same deer population at the same facility.

MATERIALS AND METHODS

Research was conducted at the University of Georgia Daniel B. Warnell School of Forestry and Natural Resources Whitehall Deer Research Facility (33°53′N, 83°21′W) in Athens, Georgia, USA. Research protocols received prior approval from the University of Georgia Animal Care and Use Committee (UGA AWA No. A3437-01).

Deer were housed in large (0.4–0.8 ha) forested outdoor pens and were fed 17% protein ration (Godfrey 17-pt deer feed, Godfrey Warehouse, Madison, Georgia, USA), with fresh hay and water available ad libitum. The average ambient temperature at time of immobilizations was 14.4 C. Temperatures ranged from −1.7 to 25.6 C.

We moved deer from outdoor pens 16–24 hr before immobilizations and individually directed them into a drop floor-type squeeze chute equipped with an electronic scale (Cervid Solutions, Tellico Plains, Tennessee). After obtaining body weights ($\bar{X}\pm$SD, 57.5±12.2 kg), we moved each deer into individual 3×6 m barn stalls where food was withheld until after the drug trial to minimize ruminal tympany (Caulkett, 1997). On 18 and 20 December 2006, we moved each deer into a squeeze chute and administered a single intramuscular injection (IM) of BAM-1 (0.34 mg/kg butorphanol [30 mg/ml, ZooPharm, Laramie, Wyoming, USA]+0.27 mg/kg azaperone [50 mg/ml, ZooPharm]+ 0.11 mg/kg medetomidine [20 mg/ml, Zoo-

Pharm] in the left hindquarter of 20 (10 male and 10 female; ≥1.5 yr old) captive white-tailed deer. On 9 and 11 January 2007, 19 of the above deer and one replacement deer (male, 1.5 yr old) received IM injections of BAM-2 (0.30 mg/kg butorphanol+0.16 mg/kg azaperone+0.20 mg/kg medetomidine in the left hindquarter.

Immediately after drug injection, each deer was released into a 15×20-m observation pen, where we recorded time to first noticeable drug effect (e.g., stumbling, gait changes), sternal time (when deer collapsed without rising again), lateral time (when deer lowered head and became immobile), and approachable time (when deer remained immobile and seemed oblivious to its surroundings). Upon approaching an immobile deer, we placed it in a sternal position to minimize rumen bloat, treated its eyes with ophthalmic ointment (Paralube® Vet Ointment, Pharmaderm, Melville, New York, USA) to minimize corneal drying, and covered its eyes with a cloth mask to minimize visual stimulation.

We recorded each deer's heart rate (determined by auscultation), respiration rate (determined by thoracic movements), rectal temperature (B–D Digital Fever Thermometer, Becton Dickinson, Franklin Lakes, New Jersey, USA), and hemoglobin saturation level (Rad-5 Masimo SET Handheld Pulse Oximeter, Irvine, California, USA) at 0, 10, and 20 min after recumbency. We attached the pulse oximeter's finger probe to each deer's tongue and shielded the photodiode from direct sunlight to minimize optical interference (Tungjitkusolmun, 1997). To further ensure accuracy, we compared heart rate readings from pulse oximetry to heart rates as determined by auscultation.

After the last set of physiologic measurements was recorded, two observers independently reported scores of three qualitative parameters evaluating quality of induction (modified from Storms et al., 2005). A quality rating score of 0–3 was assigned to each of three categories: excitability, muscle rigidity, and overall quality. Ratings for both observers were summed and reported as a combined total quality score for each induction, with a potential maximum quality score of 18. A desirable induction was defined as having a quality score of ≥12, as defined by Storms et al. (2005).

After each trial, deer were returned to individual stalls where we administered antagonists via separate IM injections. All deer treated with BAM-1 and 10 deer treated with BAM-2 received NTA-1 (50 mg naltrexone [50 mg/ml, ZooPharm]+200 mg tolazoline [100 mg/ml, Tolazine, Lloyd Laboratories, Shenandoah, Iowa, USA]+10 mg atipamezole [5.0 mg/ml, Pfizer Animal Health, Exton, Pennsylvania, USA]. Ten deer treated with BAM-2 (five males and five females) received NTA-2 (75 mg naltrexone+300 mg tolazoline+15 mg atipamezole).

For deer immobilized with BAM-2, we recorded elapsed time from antagonist injection until each deer raised its head and until each deer stood. Beginning 30 min after antagonist injection, a sedation score was assigned to each deer at 30-min intervals for 5 hr. Scores were based on the following behavioral criteria and evaluated on a scale from 5 to 0: 5=lateral recumbency with no sign of reversal; 4=lateral recumbency, unable to maintain an erect head, and noticeable eye or ear movement; 3=unable to stand, dazed and unsteady, but able to hold head up; 2=standing with moderate ataxia, braced stance, and sometimes lowered head; 1=minimal sedation characterized by drooping eyelids; and 0=no sign of sedation. Additionally, deer were monitored at 8, 12, and 16 hr after antagonist administration to monitor for signs of resedation.

We used a one-tailed Student's *t*-test to test for differences between treatments for induction times, reversal times, sedation scores, and quality ratings (SAS Institute, Cary, North Carolina, USA). A one-way analysis of variance for repeated measures and a Tukey's honestly significant difference test was performed to determine differences in physiologic values between observation intervals. All differences between treatments were evaluated at $P<0.05$.

RESULTS

Eighteen of 20 deer (90%) injected with BAM-1 were successfully immobilized. One female was not recumbent at 30 min postinjection. Thus, we returned her to a barn stall, where we administered antagonists. Another female was laterally recumbent and appeared approachable after injection, but when we positioned her for physiologic monitoring, she stood. She did not collapse again during 19 min of subsequent observation. We returned her to a barn stall, where we administered antagonists (NTA-1). Both deer subsequently recovered normally.

Each of 20 deer (100%) injected with BAM-2 were effectively immobilized.

TABLE 1. Efficacy and quality of immobilization for 10 adult male and 10 adult female, captive white-tailed deer (*Odocoileus virginianus*) hand-injected with either 0.34 mg/kg butorphanol+0.27 mg/kg azaperone+0.11 mg/kg medetomidine (BAM-1) or 0.30 mg/kg butorphanol+0.16 mg/kg azaperone+0.20 mg/kg medetomidine (BAM-2), December 2006 to January 2007, Athens, Georgia, USA.

	BAM-1			BAM-2		
Efficacy and quality of immobilization	$\bar{X}$±SD	Range	*n*	$\bar{X}$±SD	Range	*n*
Time to first effect (min)[a]	5.0±2.7	2.6–12.3	20	3.8±1.5	1.8–8.4	20
Time to sternal recumbency (min)	10.2±8.1	4.8–39.0	20	8.3±5.5	3.6–25.8	20
Time to lateral recumbency (min)	12.8±7.6	5.0–30.8	19[b]	11.3±6.6	4.1–25.8	20
Time to approach (min)	17.0±7.6	6.7–32.4	18[c]	13.5±6.9	5.5–27.5	20
Quality rating [d]	12.7±4.6	4–18	20	15.6±2.1	12–18	20

[a] Significant difference between treatments ($P<0.05$).

[b] One adult female did not become laterally recumbent.

[c] Two adult females did not become approachable.

[d] Optimal quality rating=18.0.

Time to first effect was shorter ($P<0.05$) for deer treated with BAM-2 than with BAM-1 (Table 1). Times to sternal and lateral recumbency and approach did not differ ($P>0.05$) between deer treated with BAM-1 and with BAM-2 (Table 1).

Quality ratings of drug inductions were higher ($P<0.05$) for deer treated with BAM-2 than for deer treated with BAM-1 (Table 1). Hemoglobin saturation, respiration, and rectal temperature of deer treated with BAM-1 or with BAM-2 did not differ ($P>0.05$) within any monitoring period (Table 2). Heart rate was higher ($P<0.05$) for deer treated with BAM-2 than for BAM-1–treated deer at 0, 10, and 20 min postrecumbency (Table 2). For deer treated with BAM-1, heart rate was greater ($P<0.05$) at 0 min versus 20 min postrecumbency, trending downward over time (Table 2). For deer treated with BAM-2, heart rate also trended downward

TABLE 2. Physiologic data recorded at three intervals postrecumbency (min) for 10 adult male and 10 adult female, captive white-tailed deer (*Odocoileus virginianus*) immobilized with either 0.34 mg/kg butorphanol+0.27 mg/kg azaperone+0.11 mg/kg medetomidine (BAM-1) or 0.30 mg/kg butorphanol+0.16 mg/kg azaperone+0.20 mg/kg medetomidine (BAM-2), December 2006 to January 2007, Athens, Georgia, USA.

		BAM-1			BAM-2		
Physiologic parameter	Min[a]	$\bar{X}$±SD[b]	Range	*n*[c]	$\bar{X}$±SD	Range	*n*
SpO_2	0	85.3±5.3 A	72–91	18	87.5±5.8 A	71–95	20
	10	82.2±8.1 B	64–93	18	84.5±6.7 B	71–92	20
	20	81.4±7.6 B	66–94	18	83.1±6.8 B	70–92	20
Heart rate	0*	48.4±10.8 A	34–72	18	56.9±10.9 A	50–64	20
	10*	46.2±8.9 AB	40–68	18	52.1±9.4 B	34–68	20
	20*	43.2±8.9 B	40–64	18	49.1±7.8 C	36–60	20
Respiration rate	0	23.9±14.2 A	8–66	18	22.3±12.5 A	8–62	20
	10	20.7±9.4 A	10–46	18	21.1±7.8 A	8–42	20
	20	19.3±7.6 A	8–36	18	19.3±7.2 A	8–36	20
Temperature (C)	0	39.5±1.0 A	37.9–41.3	18	39.2±0.8 A	37.7–40.7	20
	10	39.5±1.0 A	37.8–41.8	18	39.3±0.8 A	37.6–40.5	20
	20	39.2±1.1 B	37.6–41.6	18	39.2±0.7 A	37.8–40.3	20

[a] Asterisks indicate differences ($P<0.05$) between drug treatments for that time interval.

[b] Means with the same letter within a column are not significantly different ($P>0.05$).

[c] Physiologic data were not collected on two unapproachable females.

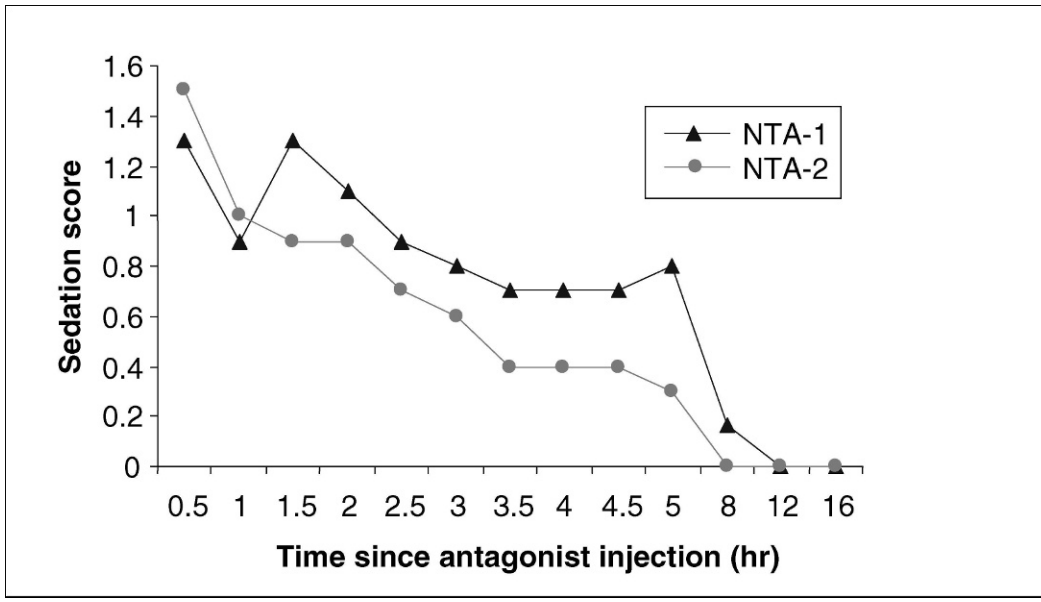

FIGURE 1. Mean sedation scores for 10 adult (five males and five females), captive white-tailed deer immobilized with 0.30 mg/kg butorphanol+0.16 mg/kg azaperone+0.20 mg/kg medetomidine (BAM-2) and antagonized with either 50 mg naltrexone+200 mg tolazoline+10 mg atipamezole (NTA-1) or 75 mg naltrexone+300 mg tolazoline+15 mg atipamezole (NTA-2). Sedation score of 2=standing with moderate ataxia. Sedation score of 0=no sedation.

over time but differed ($P<0.05$) at 0, 10, and 20 min postrecumbency (Table 2). Similar time-related downward trends were observed in hemoglobin saturation for BAM-1 and BAM-2 treatments with different ($P<0.05$) values at 0 and 20 min postrecumbency (Table 2). Respiration rates of deer immobilized with BAM-1 and BAM-2 generally trended downward over time with five deer treated with BAM-1 and seven deer treated with BAM-2 dropping to 8–16 breaths per min at 20 min postrecumbency. Pale-colored gums and mucous membranes of 5 of 18 deer (28%) immobilized with BAM-1 were indicative of bradypnea. Only 1 of 20 deer (5%) immobilized with BAM-2 had pale gums.

Hyperthermia (≥41.1 C) was observed in 2 of 18 deer (11%) treated with BAM-1 and 0 of 20 deer (0%) treated with BAM-2. Rectal temperatures for BAM-1–treated deer were higher ($P<0.05$) at 20 min than at 0 or 10 min postrecumbency. For BAM 2–treated deer, there was no difference in rectal temperature ($P>0.05$) at 0, 10, and 20 min postrecumbency.

For deer treated with BAM-2, time (min) to head up did not differ ($P>0.05$) for those receiving NTA-1 ($\bar{X}\pm$SD, 21.4±40.8; range, 1.5–136.7; $n=9$) and NTA-2 ($\bar{X}\pm$SD, 11.4±6.1; range, 0.1–21.7; $n=10$). Similarly, time (min) to standing did not differ ($P>0.05$) for deer receiving NTA-1 ($\bar{X}\pm$SD, 28.8±40.0; range, 6.0–137.7; $n=10$) and NTA-2 ($\bar{X}\pm$SD, 15.4±7.5; range, 6.1–29.7; $n=10$). No differences ($P>0.05$) in mean sedation scores were detected for BAM-2 treated deer receiving either NTA-1 or NTA-2, with each deer ($n=19$) standing at 30 min postinjection of antagonist (Fig. 1). However, a large, tame, adult male (84.6 kg, 5.5 yr old) antagonized with NTA-1 showed no signs of recovery at 5 hr postinjection of antagonist. We then administered a subsequent IM injection of 250 mg of tolazoline. Within about 15 min postinjection, he began recovering from immobilization, with minimal ataxia.

As his recovery progressed, muscle tremors and low rectal temperature (35.1 C) were suggestive of hypothermia. We walked him into an enclosed, insulated room, which was warmed by a space heater. His temperature rose to 36.1 C and 37.8 C at about 25 and 75 min after we began therapy, respectively. We subsequently returned him to a barn stall and observed no further complications. However, another tame male (80.5 kg, 4.5 yr old) immobilized with BAM-1 and antagonized with NTA-1 was found dead in an outdoor pen 11 days after immobilization. Superficial injuries to his body indicated he may have died from fighting with other males; however, an exact cause of death is unknown.

DISCUSSION

Pharmacologic advances during the past 40 yr have resulted in the development of many drugs suitable for immobilization and general anesthesia of deer (Nielsen, 1999; Walsh and Wilson, 2002). Synergistic effects of modern drug combinations enable the use of smaller drug volumes (i.e., smaller, lighter darts), which improves precision of remote delivery systems and reduces impact site trauma (Cattet et al., 2006). However, drug combinations must be thoroughly tested on captive deer before field immobilizations are attempted. Hand-placed injections in a controlled, laboratory environment eliminate drug lost from darts on impact (Cattet et al., 2006) and standardize factors affecting induction times (Schultz et al., 1992; Caulkett and Haigh, 2004). The Whitehall Deer Research Facility at The University of Georgia has proven valuable for scientifically evaluating promising drug combinations because of its captive deer herd and efficient deer-handling system (Miller et al., 2003, 2004; Storms et al., 2005, 2006; Muller et al., 2007). Furthermore, because multiple experiments conducted by researchers at this facility have had identical drug application and deer handling and observation protocols, and similar randomization of deer, it is possible to make qualitative comparisons among drug treatments relative to their efficacy, induction quality, and physiologic measurements. Of course, ambient temperature during a particular drug trial was influenced by date and must be considered when evaluating across-trial comparisons.

This research revealed that, at appropriate dosages, medetomidine, when combined with butorphanol and azaperone provided complete immobilization of white-tailed deer. Deer treated with BAM-2 exhibited faster drug induction and better induction quality than those treated with BAM-1. Induction quality for BAM-2 was excellent (i.e., score of 15.6 out of a possible 18.0) when compared with previous experiments involving combinations of xylazine and carfentanil ($\bar{X}\pm$SD, 9.3$\pm$2.9; Storms et al., 2005) or two combinations of xylazine, carfentanil, and ketamine ($\bar{X}\pm$SD, 11.1$\pm$3.0 and 11.3$\pm$2.1; Storms et al., 2006). Infrequent behavioral side effects with BAM-1 and BAM-2 that influenced induction-quality scores included mild body muscle tremors, leg paddling, and side-to-side head movements.

When considering previous research at this facility, time to first effect for BAM-1 and BAM-2 was most similar to that of xylazine/ketamine ($\bar{X}\pm$SD, 3.6$\pm$0.8; Muller et al., 2007) and medetomidine/ketamine ($\bar{X}\pm$SD, 3.1$\pm$0.5; Muller, 2007). Previous experiments involving combinations of Telazol/xylazine, carfentanil/xylazine, carfentanil/xylazine/ketamine, and medetomidine/ketamine/butorphanol each produced first effects in $<$2.0 min (range, 0.9$\pm$0.4 to 1.9$\pm$0.2; Miller et al., 2003; Storms et al., 2005, 2006). Likewise, time to sternal recumbency for deer treated with BAM-1 and BAM-2 was more similar to that of xylazine/ketamine ($\bar{X}\pm$SD, 6.9$\pm$0.9; Muller et al., 2007) and medetomidine/ketamine ($\bar{X}\pm$SD, 5.8$\pm$0.9; Muller et al.,

2007) than to Telazol/xylazine, carfentanil/xylazine, carfentanil/xylazine/ketamine, and medetomidine/ketamine/butorphanol (range, 2.3±0.9 to 4.7±3.3; Miller et al., 2003; Storms et al., 2005, 2007). Based on our findings, deer treated with BAM-1 or BAM-2 took an average of 17.0 and 13.5 min, respectively, to become approachable, with muscular relaxation and loss of reflexes. However, they would be unable to effectively walk or run in response to flight-evoked stimuli at about 8–10 min postinjection. Thus, our results suggest BAM-1 or BAM-2 are appropriate for field applications because it is common to wait >20 minutes after darting a deer to ensure it is recumbent and unaware of being pursued (Kilpatrick et al., 1996; Kilpatrick and Spohr, 1999).

A pulse oximeter is useful for monitoring oxygen hemoglobin saturation of immobilized deer. Normal saturation of deer blood ranges from 95% to 97%, with SpO_2 values below 85–90% possibly requiring supportive oxygen therapy (Caulkett, 1997). However, this recommendation is based on speculation rather than biologic data on pathologic effects, if any, of depressed SpO_2 on immobilized wildlife (Wenker, 2007). Deer in our study had mean SpO_2 values <85% at 10 and 20 min postrecumbency. For comparison, at 20 min postrecumbency, 10 immobilizing drug combination treatments previously tested at this facility resulted in mean SpO_2 values ranging from 85.2±10.4 to 91.0±4.8 (Miller et al., 2003, 2004; Storms et al., 2005, 2006; Muller et al., 2007). Because of relatively low SpO_2 values in our study and in previous studies at this facility and because pulse oximeters are poorly calibrated for saturation levels below 80%, practitioners must concurrently monitor color of oral mucous membranes and capillary refill time for signs of circulatory deficiency (grayish and bluish white and >2.0 sec, respectively; Reddy, 1997; Tungjitkusolmun, 1997; Nielsen, 1999) when immobilizing deer with BAM-1, BAM-2, or other drug combinations. Also, to minimize risk of a deer becoming hypoxemic, it should be positioned in sternal recumbency with its neck fully extended (Caulkett and Haigh, 2004). Corrective, supportive, or emergency treatments should be initiated if pulse oximetry and visual signs indicate SpO_2 levels have become critically low (Caulkett, 1997; Caulkett and Haigh, 2004).

Normal heart rate for a deer at rest is 70–80 beats/min (Nielsen, 1999). A heart rate of <30 beats/min is suggestive of heart failure, and one >150 beats/min suggests other physiologic problems (Caulkett and Haigh, 2004). Heart rates for deer treated with BAM-1 or BAM-2 were well within the limits for animal welfare. For purposes of comparison, heart rates for deer in previous studies at this facility ranged from about 61–74 beats/min at 10 min postrecumbency to 53–67 beats/min at 20 min postrecumbency (Miller et al., 2003; Storms et al., 2005, 2006).

Normal respiration rate for a deer at rest is 16–20 breaths/min, but depth of respiration is equally important, with deep and smooth breathing desirable (Nielsen, 1999). Mean respiration rates for deer in our study compared favorably with those of previous drug combination experiments (range at 20 min postrecumbency, 19.3±8.8 to 33.8±26.4; Miller et al., 2003, 2004; Storms et al., 2005, 2006; Muller et al., 2007). However, because respiration rates of 12 deer treated with BAM-1 or BAM-2 were low, we suggest using a nasal catheter to supply 6–8 l/min of supplemental oxygen when respiration rate drops below 16 breaths/min (Caulkett and Haigh, 2004). Although some practitioners might consider field application of supplemental oxygen impractical, it is warranted whenever a deer will be chemically immobilized for >15 min, regardless of immobilizing drugs used (Caulkett, 1997).

Because immobilized deer are prone to hyperthermia it is important to monitor rectal temperature (Nielsen, 1999; Caulk-

ett and Haigh, 2004). Normal rectal temperature of deer is 35–41 C ($\bar{X}$=38 C; Nielsen, 1999). When rectal temperature reaches 40 C, hyperthermia becomes a concern and a temperature >41 C constitutes a medical emergency, requiring aggressive efforts to cool the deer (Nielsen, 1999; Caulkett and Haigh, 2004). Rectal temperatures of deer in our study were within normal limits with the exception of two deer treated with BAM-1, which exceeded 41 C. At 20 min postrecumbency, mean rectal temperatures for deer treated with BAM-1and BAM-2 compared favorably with means reported for other drug combinations tested at this facility during December to April (range, 40.1±1.1 to 42.1±1.6; Miller et al., 2003; Storms et al., 2005, 2006; Muller et al., 2007). However, as referenced by Walsh and Wilson (2002), nontame deer require higher drug dosages than tame deer. Only because we monitored rectal temperature of each deer in our study were we able to recognize when the tame buck treated with BAM-2 became hypothermic, and we were able to act accordingly. Regardless of drugs administered, it is the responsibility of the practitioner to recognize inherent risks (e.g., ambient temperature, individual personalities, etc.) and to take appropriate actions to ensure animal welfare.

Deer immobilized with BAM-2 and reversed with NTA-1 or NTA-2 were standing at 30 min postantagonist injection, with no differences (P>0.05) in sedation scores. Therefore, if cost of antagonists is considered, we suggest that NTA-1 is the best choice for reversing BAM-2 because it contains the least amounts of naltrexone and atipamezole. As discussed by Muller et al. (2007), naltrexone, the antagonist for butorphanol, is relatively expensive and adds considerably to the cost of reversal when compared with immobilizing drug combinations not including an opioid. Furthermore, synergism associated with the tolazoline and atipamezole combination facilitates complete agonist reversal while lowering cost, when compared with atipamezole use alone. Efficacy of NTA-1 and NTA-2 to reverse the effects of BAM-2 was superior in time (min) to standing for deer at this facility immobilized with the popular Telazol/xylazine combination and reversed with yohimbine (half intravenous [IV] and half IM; $\bar{X}$±SD, 165.5±66.4; Miller et al., 2003). For additional comparison, after censoring data for a xylazine/ketamine immobilization of a single deer (Muller et al., 2007), time (min) to standing for nine other experimental drug combination treatments followed by antagonist injection appeared shorter than those for BAM-2 followed by NTA-1 or NTA-2 (range, 1.9+1.1 to 12.3+15.4; Miller et al., 2003, 2004; Storms et al., 2005, 2006; Muller et al., 2007). However, we believed times to standing following injections of NTA-1 or NTA-2 (<30 min.) were reasonable for field applications. If NTA-1 or NTA-2 had been injected IV in addition to IM, recovery times might have been shorter. However, we administered them IM only because IV injections of tolazoline and atipamezole have been reported to cause anxiety and seizures in ruminants (Read et al., 2000).

In summary, a small volume of butorphanol, azaperone, and medetomidine resulted in safe and reversible immobilizations of captive deer. Of the two dosages we tested, BAM-2 provided faster induction, superior induction quality, and less negative side effects than BAM-1. Quality of induction with BAM-2 was excellent. Both BAM-1 and BAM-2 were effectively reversed by an injection of NTA-1. Both NTA-1 and NTA-2 were equally effective at reversing the effects of BAM-2. When comparing our results to previous immobilizing drug experiments at this facility, induction time (min) for BAM-2 was most similar to induction times for xylazine/ketamine or medetomidine/ketamine combinations but without the associated risk of hyperthermia. As with all immobilizing drug combinations, when using BAM-2 to

immobilize a group of deer with a range of individual drug tolerance, practitioners should monitor respiration, heart rate, rectal temperature, and hemoglobin saturation and be prepared to take therapeutic measures when needed. In addition, although human health risk related to accidental exposure of drugs in BAM are less than for drug combinations including more potent opioids (Radcliffe et al., 2000), practitioners should follow appropriate safety protocols (Nielsen, 1999). Future clinical research should evaluate physiologic responses to BAM-2, which were outside the scope of our study (e.g., blood pressure, blood analysis, serum analysis) and test its efficacy for capturing free-ranging deer.

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