Immobilizing Captive White-tailed Deer Using Medetomidine-Ketamine versus Xylazine-Telazol

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Abstract: Chemical immobilization often is the most effective method for capturing white-tailed deer (*Odocoileus virginianus*). Numerous chemical immobilization agents are available. We compared the efficacy and physiological effects of 2 white-tailed deer immobilizing agents: medetomidine-ketamine (M-K) antagonized with atipamezole, and xylazine-Telazol (X-T) antagonized with tolazoline. Mean induction time was longer and more variable for M-K. Mean reversal time and total down time was longer and more variable for X-T. Mean blood oxygen saturation (SpO₂) in subjects treated with M-K was lower immediately following induction. We detected no differences in mean SpO₂ at >5 minutes post induction or for mean rectal temperature or pulse rate at any time during the monitoring period between the 2 groups. Each agent appeared to offer advantages and disadvantages, depending on the specific circumstances of the capture event.

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Managing white-tailed deer often necessitates live capture of specific individuals. Selective capture of white-tailed deer may be necessary for research such as to attach or remove radio-transmitters, to translocate nuisance animals, or to capture injured or sick animals for treatment. A variety of techniques are used to capture deer for research and management purposes. These include drop nets, drive nets, rocket nets, and live traps (Schemnitz 1994). Most of these methods limit the biologists' ability to select specific animals for capture. However, chemical immobilization using a remotely delivered anesthetic agent is a selective capture technique which allows specific individuals to be targeted. This technique does not require pre-positioning of equipment such as traps or nets, which are subject to tampering by humans, and causes minimal disturbance to both wildlife and humans. Also, it does not require animals to be attracted to the capture site by bait, as is the case with preset traps. Therefore, it is especially useful in urban or suburban environments, where other methods may be impractical or socially unacceptable, and which offer abundant food supplies in the form of landscape vegetation that can negate the effectiveness of bait. Finally, anesthetized animals can be handled with less stress to the animal and a lower risk of injury to the animal and human workers.

Attributes of an ideal anesthetic for wildlife capture include short induction time (time to immobilization), rapid and complete reversal, and minimal physiological distress (Kreeger 1996). Deer are often frightened and flee after being struck with a dart. Hence, long induction times allow the animal to travel a greater distance prior to immobilization which decreases the chance of locating the immobilized individual. Animals that are anesthetized but not found face increased risk of injury or death due to physical obstacles (i.e., drowning in standing water, entanglement in fences), complications (i.e., bloat, obstruction of the airway), or attacks by predators.

Immobilization can cause physiological stress in the anesthetized animal. Even under human care, anesthetized animals are at risk of complications such as the depression of the cardiovascular or respiratory system and disruption of the thermoregulatory mechanisms. These effects may require supportive treatment by workers or initiation of anesthetic reversal prior to completion of the procedure and may result in animal injury or death.

Effects of many immobilization agents can be reversed with the administration of a chemical antagonist. Reversal decreases recovery times, thus reducing the risk of complications and the amount of time required to monitor and care for the immobilized animal. If the management or research protocol requires the immobilization of multiple animals, rapid recovery may allow more animals to be handled during a given period of time.

Numerous compounds are used to immobilize white-tailed deer. One of the most commonly used agents is a combination of xylazine and Telazol (zolazopamtiletamine). Xylazine is a commonly-used alpha₂-adrenergic antagonist marketed under several brand names, including Rompun and Xyla-ject. Telazol is a potent cyclohexane compound. It is classified by the U.S. Drug Enforcement Administration as a Schedule III controlled substance. As such, its purchase and use is strictly regulated.

Kilpatrick and Spohr (1999) assessed the efficacy of xylazine-Telazol (X-T) for capturing free-ranging white-tailed deer in Connecticut. They concluded that X-T was more effective than a xylazine-ketamine combination because induction time, measured as the distance traveled following administration, was shorter. Based on their results, they recommended X-T for capture of white-tailed deer.

A possible alternative to X-T is medetomidine-ketamine (M-K). Medetomidine is an alpha₂-adrenergic compound, like xylazine, but 40–200 times more potent (Kreeger 1996). It is marketed under the trade name Domitor. Ketamine is a cyclohexane, related to Telazol. It is marketed under such trade names as Ketaset and Ketaject.

M-K has been used to successfully immobilize a number of cervids, including moose (*Alces alces;* Arnemo et al. 1994), sike deer (*Cervus nippon;* Tsuruga et al. 1999), Eld's deer (*C. eldi;* Klein et al. 1996) fallow deer (*Dama dama;* Fernandez-Moran and Peinado 1996), reindeer (*Rangifer tarandus;* Tyler et al. 1990), and mule deer (*O. hemionus;* Caulkett et al. 2000). Jalanka (1989) used M-K to anesthetize 20

white-tailed deer at the Helsinki Zoo. He reported satisfactory results, but did not discuss physiological effects. Kreeger (1996) also suggested M-K as an alternative to X-T. We are unaware of any studies which directly compare the efficacy and physiological effects of these 2 combinations for immobilizing white-tailed deer. Therefore, our objective was to compare the efficacy (induction time, recovery time, and total down time) and physiological effects (cardiorespiratory and thermoregulatory function) of M-K and X-T for immobilizing white-tailed deer.

Methods

We used 16 captive-raised, female white-tailed deer for this study. All deer were adults, 2–9 years of age. Each individual was randomly assigned to 1 of 2 groups ("X-T" and "M-K") of 8 deer each. Prior to treatment, we visually estimated live weight of the study subjects to be 45kg, and calculated drug dosages based on this estimate.

Each animal was placed in an individual 2.5×2.5 m stall and captured by hand. We then administered an intramuscular injection of the appropriate immobilizing agent. Animals in the X-T group were given 175.50 mg xylazine (Xyla-ject; Phoenix Pharmaceuticals, St. Joseph, Mo.) and 216.00 mg Telazol (Fort Dodge Animal Health, Ft. Dodge, Iowa; Kilpatrick and Spohr 1999). Individuals in the M-K group received 3.15 mg medetomidine (Domitor; Pfizer Animal Health, Exton, Pa.) and 90.00 mg ketamine (Ketaject; Phoenix Pharmaceuticals, St. Joseph, Mo.; Kreeger 1996).

Personnel withdrew from the stall immediately following administration of the drug and observed the subject from a concealed location. The animal was allowed to remain alone and undisturbed until induction, which we judged to have occurred once the animal had become recumbent and lost consciousness.

Immediately following induction, we recorded each subject's blood oxygen saturation (SpO₂), pulse rate, and rectal temperature. The measurement of SpO₂ ("pulse oximetry") is recognized as a standard monitoring procedure in anesthesiology as an index of respiratory function (Tremper and Barker 1989). We used a Nellcor NBP-40 pulse oximeter (Nellcor Puritan Bennett, Inc., Pleasanton Calif.) to monitor SpO₂ and pulse rate. We monitored rectal temperature as an index of thermoregulatory function, using a ReliOn digital thermometer (Wal-Mart, Inc., Bentonville, Ark.). We continued to measure SpO₂, pulse rate, and rectal temperature at 5-minute intervals until administration of the antagonist at 30-minutes post-induction. Due to technician error, some data were not collected at all time intervals for 3 subjects. Therefore, 1 subject from each group was excluded from SpO₂ and pulse rate analysis, and 1 subject from the M-K group was excluded from analysis of body temperature.

Approximately 30 minutes following induction, we administered an antagonist to each subject. X-T anesthesia was reversed with 180.00 mg tolazoline (Tolazine; Lloyd Laboratories, Shenandoah, Iowa), given intravenously (Kreeger et al. 1986). Animals in the M-K group were given 15.75 mg atipamezole (Antisedan; Pfizer Animal Health, Exton, Pa.) intramuscularly (Jalanka and Roeken 1990, Pfizer Animal

Health1999).

We used a handheld stopwatch to record time from injection of the anesthetic until (1) induction, (2) administration of the antagonist, and (3) recovery. We used this data to calculate induction, recovery, and total down time. We defined induction time as time elapsed between administration of the anesthetic and loss of consciousness; recovery time as time elapsed between administration of the antagonist and recovery of consciousness and mobility (ability to stand, walk, and avoid capture); and total down time as the sum of induction time, recovery time, and a 30-minute monitoring period. We chose a 30-minute period to simulate a procedure that might be performed during a typical capture, such as transmitter attachment.

Immediately following recovery, all animals were euthanized (as required for another study) and weighed. We then calculated the actual drug dosage administered to each animal.

We calculated means and 95% confidence intervals (C.I.s) for induction, recovery, and total down time for the M-K and X-T groups. We tested physiological data for normality using the Shapiro-Wilk test and equality of variance between samples using Levine's test. Data were found to violate the parametric assumptions of normality and equal variance; therefore, we tested for differences in physiological parameters between groups using the Mann-Whitney test (Ott and Longnecker 2001).

Results

Mean body weight of the X-T group was 36 (range: 27–43) kg, resulting in mean dosages of 4.3 (range: 4.08–6.50) mg/kg xylazine, 6.06 (range: 5.02–8.00) mg/kg Telazol, and 5.05 (range: 4.19–6.67) mg/kg tolazoline. Mean body weight of the M-K group was 39 (range: 29–47) kg, resulting in mean dosages of 0.08 (range: 0.07–0.11) mg/kg medetomidine, 2.35 (range: 1.91–3.10) mg/kg ketamine, and 0.41 (range: 0.34–0.54) mg/kg atipamezole. Due to overestimation of body weight prior to calculation of dosage, actual dosages exceeded those recommended by Kilpatrick and Spohr (1999) and Kreeger (1996).

Induction time for the M-K group was longer and more variable ($\bar{x} = 10.4$ minutes; 95% C.I. = 6.1–14.7) than for the X-T group ($\bar{x} = 2.8$ minutes; 95% C.I. = 1.8–3.8; Fig. 1). However, recovery time (Fig. 2) for the M-K group was shorter and less variable ($\bar{x} = 9.7$ minutes; 95% C.I. = 7.4–12.1) than for the X-T group ($\bar{x} = 82.7$ minutes; 95% C.I. = 60.1–105.4). Similarly, total down time (Fig. 3) for the M-K group was shorter and less variable ($\bar{x} = 54.4$ minutes; 95% C.I. = 50.4–58.4) than for the X-T group ($\bar{x} = 120.7$ minutes; 95% C.I. = 97.8–143.5).

The SpO₂ of the X-T group was significantly lower (U = 43, P= 0.017) than that of the M-K group immediately following induction, but levels were similar throughout the rest of the monitoring period (Table 1). Mean SpO₂ levels for both groups remained above 80% for the duration of the monitoring period. Mean body temperature and pulse rate did not differ between groups at any time during the monitoring period. Both groups exhibited elevated body temperature following induction, which declined slightly throughout the procedure but remained above normal levels. Mean



Figure 1 Mean induction time for whitetailed deer given medetomidine-ketamine (M-K) and xylazine-Telazol (X-T). Vertical bars indicate 95% confidence intervals for the mean.



Treatment





Figure 3. Mean down time for whitetailed deer given medetomidine-ketamine (M-K) and xylazine-Telazol (X-T). Vertical bars indicate 95% confidence intervals for the mean.

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Table 1. Group mean, test-statistic (Mann-Whitney U), and *P*- value of SpO_2 , body temperature, and pulse rate at 0–30 minutes post induction for white-tailed deer given medetomidine-ketamine (M-K) and xylazine-Telazol (X-T).

		SpO ₂		
		5002		
Minutes	X-T Group	M-K Group		
post-	mean	mean		_
induction	(N = 7)	(N = 7)	U	Р
0	80.0	91.4	43.0	0.017
5	80.2	89.4	39.0	0.063
10	87.5	88.0	35.5	0.157
15	90.5	87.7	26.5	0.797
20	90.5	87.3	27.5	0.700
25	91.7	88.9	27.0	0.749
30	91.3	89.7	25.0	0.949
		Body tempe	rature	
Minutes	X-T Group	M-K Group		
post-	mean	mean		
induction	(N = 8)	(N = 7)	U	Р
0	39.7	40.2	36.5	0.336
5	39.7	40.1	39.5	0.318
10	39.8	39.9	29.0	0.955
15	39.8	39.7	29.0	0.955
20	39.6	39.6	28.5	0.955
25	39.6	39.4	29.5	0.867
30	39.4	39.2	30.0	0.867
		Pulse ra	te	
Minutes	X-T Group	M-K Group		
post-	mean	mean		
induction	(N = 8)	(N = 7)	U	Р
0	83.7	72.0	36.5	0.128
5	81.7	70.6	36.0	0.165
10	76.6	67.7	33.5	0.259
15	71.4	64.3	32.0	0.383
20	68.0	61.1	33.0	0.318
25	65.6	59.0	31.0	0.456
30	62.9	57.6	25.5	0.902
50	02.7	57.0	20.0	0.702

pulse rate of both groups declined steadily during the monitoring period to slightly below normal levels (Table 1).

Discussion

Induction time following M-K injection was substantially longer than the median of 6.2 minutes reported by Jalanka and Roeken (1990), despite the fact that we administered a higher mean dosage of anesthetic (0.068 and 1.55 mg/kg of medetomidine and ketamine, respectively) than in their study. This difference may have resulted from the rather subjective nature of defining induction (or, in the case of Jalanka and Roeken [1990], "down").

Our induction time of 2.8 minutes for X-T anesthesia was similar to the mean value of 2.9 minutes reported by Kilpatrick and Spohr (1999); however, our dosages were considerably higher. Further, induction time in our study was based on observation of captive animals, while Kilpatrick and Spohr (1999) defined induction time as the search time for darted, free-ranging deer. These factors may make the results less comparable, but suggests that X-T has a fairly high therapeutic index.

While both M-K and X-T appear to satisfactorily anesthetize captive whitetailed deer, significant differences exist between the 2 treatments. Deer treated with X-T succumb to anesthesia more quickly, and remain immobilized longer than those given M-K at the dosages we administered. Further, X-T appears to depress respiratory function, at least for a few minutes immediately following treatment. Tremper and Barker (1989) defined SpO₂ \leq 85% in human patients as severe hypoxemia. Our data suggests that X-T suppresses SpO₂ below this level. It is reasonable to assume that this may have some untoward effects, although data on the pathological effects of short-term reduced SpO₂ on white-tailed deer are lacking.

Normal body temperature for deer is 38.4 C (Nielsen 1999). Both groups exhibited elevated body temperature during anesthesia. Whether this resulted from physiological effects of the drugs, excitation of the animal during capture, or a combination of these factors, we do not know. However, hyperthermia is commonly encountered when chemically immobilizing wild animals and was not unexpected. Other studies have reported similar results among cervids during immobilization (DelGuidice et al. 1989, Tsuruga et al. 1999).

Seal et al. (1978) reported that immobilized white-tailed deer in northern Minnesota with rectal temperatures >40 C were at greater risk of capture-related mortality. Kreeger (1996) recommended immediate reversal of animals whose temperature exceeded 41 C. One individual in the X-T group had a rectal temperature of 41.3 C and 1 individual in the H-K group had a rectal temperature of 41.0 C during part of the monitoring period. Three individuals give X-T and 4 individuals give M-K exhibited rectal temperatures of 40.1–40.9 C during all or part of the monitoring period. Despite similarities in response, the longer periods of hyperthermia under X-T anesthesia arising from a longer down time may place animals at greater risk.

Late in the monitoring period, mean pulse rate of both groups (Table 1) fell below the normal range of 70–80 beats per minute given by Nielsen (1999). This was consistent with an animal at rest and did not cause concern. The pulse rate of all subjects remained regular throughout the procedure.

Management Implications

Our results suggest that the physiological effects of the 2 agents are comparable. However, a trade-off exists between rapid induction and long recovery periods. As such, the selection of an immobilizing agent will depend upon the specific capture situation and the anticipated time required to complete the procedure. Free-ranging animals face greater risk when search times are long and the possibility of losing an animal is high. Therefore, rapid induction, resulting in reduced travel distance and search times, is desirable under field conditions. Under these circumstances, a fastacting agent such as X-T would appear to be desirable, despite the increased down time which demands considerable investment of manpower to monitor recovering animals. Conversely, when immobilizing captive or confined animals, where search times are less important and risk of loss is low or non-existent, considerable savings in time may be realized by using M-K, because of the shorter total down time.

We acknowledge several shortcomings in our study. First, our results may not be applicable to wild deer, as our study was conducted using captive deer in a controlled environment. We chose to conduct the experiment under these circumstances because the study subjects were part of a separate study, which required close monitoring of the animals. Also, manpower investment would have been much greater under field conditions. Second, we could have more accurately calculated drug dosages had we weighed each animal prior to treatment. However, our facilities were not equipped to do this easily, and we judged that we could accurately estimate animal weights. In hindsight, this was an error in judgment which may have weakened our conclusions.

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