Comparative cardiopulmonary effects of carfentanil-xylazine and medetomidine-ketamine used for immobilization of mule deer and mule deer/white-tailed deer hybrids

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Abstract

Three mule deer and 4 mule deer/white-tailed deer hybrids were immobilized in a crossover study with carfentanil (10 µg/kg) + xylazine (0.3 mg/kg) (CX), and medetomidine (100 µg/kg) + ketamine (2.5 mg/kg) (MK). The deer were maintained in left lateral recumbency for 1 h with each combination. Deer were immobilized with MK in 230 ± 68 s (mean ± SD) and with CX in 282 ± 83 seconds. Systolic, mean and diastolic arterial pressure were significantly higher with MK. Heart rate, PaO_2, $PaCO_2$, pH, and base excess were not significantly different between treatments. Base excess and pH increased significantly over time with both treatments. Both treatments produced hypoventilation ($PaCO_2$ > 50 mmHg) and hypoxemia (PaO_2 < 60 mmHg). PaO_2 increased significantly over time with CX. Body temperature was significantly ($P < 0.05$) higher with CX compared to MK. Ventricular premature contractions, atrial premature contractions, and a junctional escape rhythm were noted during CX immobilization. No arrhythmias were noted during MK immobilization. Quality of immobilization was superior with MK, with no observed movement present for the 60 min of immobilization. Movement of the head and limbs occurred in 4 animals immobilized with CX. The major complication observed with both of these treatments was hypoxemia, and supplemental inspired oxygen is recommended during immobilization. Hyperthermia can further complicate immobilization with CX, reinforcing the need for supplemental oxygen.

Résumé

Dans une étude en dispositif chassé-croisé trois cerf-mulets et quatre hybrides cerf-mulet/cerf de Virginie furent immobilisés à l'aide de carfentanil (10 µg/kg) avec xylazine (0,3 mg/kg) (CX), et de la médétomidine (100 µg/kg) avec kétamine (2,5 mg/kg) (MK). Pour chacune de ces combinaisons, les animaux furent maintenus en décubitus latéral pendant 1 h. Le temps d'immobilisation avec la combinaison MK était de 230 ± 68 s (moyenne ± écart-type) et de 282 ± 83 s pour CX. Les pressions artérielles systolique, diastolique et moyenne étaient significativement plus élevées avec la combinaison MK. Aucune différence significative entre les groupes de traitement ne fut notée pour ce qui est du rythme cardiaque, de la PaO_2 et $PaCO_2$, du pH et de l'excès de base. Pour les deux traitements, l'excès de base et le pH augmentèrent significativement avec le temps, et on nota une hypoventilation ($PaCO_2$ > 50 mmHg) et une hypoxémie (PaO_2 < 60 mmHg). Une augmentation significative de la PaO_2 dans le temps fut notée avec la combinaison CX. La température corporelle était significativement (P < 0,05) plus élevée avec le mélange CX qu'avec la combinaison MK. Des contractions ventriculaires et auriculaires prématurées, ainsi qu'un rythme d'échappement atrio-ventriculaire furent notés lors de l'immobilisation avec CX. Aucune arythmie n'a été observée durant l'immobilisation avec la combinaison MK. La qualité de l'immobilisation avec MK était meilleure étant donné qu'aucun mouvement observable n'a été noté, alors que des mouvements de la tête et des membres ont été observés chez quatre animaux immobilisés avec la combinaison CX. L'hypoxémie fut la complication majeure observée avec les deux combinaisons de traitement, et un apport supplémentaire en oxygène est recommandée durant l'immobilisation. L'hyperthermie peut être une complication supplémentaire avec la combinaison CX, ce qui supporte le besoin d'un apport supplémentaire en oxygène.

(Traduit par docteur Serge Messier)

Introduction

Wild and captive deer may need to be immobilized for a variety of reasons. Restraint of these animals can be difficult, and chemical immobilization may be required in some situations. Drug options for immobilization of deer include: xylazine or xylazine-ketamine combinations (1); Fentazine, a mixture of fentanyl, azaperone, and xylazine, is available in New Zealand and Australia. Potent narcotics, such as carfentanil and etorphine, can also be used for deer immobilization (2). Xylazine as the sole agent produces unreliable immobilization. Combining ketamine with xylazine increases reliability, but results in a high volume if commercial ketamine is used.

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Table I. Arterial blood pressure, heart rate, rectal temperature and respiratory rate following the administration of medetomidine + ketamine[a] (MK), or carfentanil + xylazine[b] (CX), in mule deer and mule deer/white-tailed deer hybrids

		Time from immobilization (min)									
Parameter	Tx	15	20	25	30	35	40	45	50	55	60
DAP (mmHg)[c]	MK	115 ± 13	115 ± 11	115 ± 10	113 ± 10	111 ± 10	111 ± 13	110 ± 9	110 ± 13	109 ± 13	107 ± 14
	CX	90 ± 11	87 ± 11	86 ± 19	82 ± 18	78 ± 6	82 ± 7	81 ± 12	81 ± 12	82 ± 14	84 ± 14
MAP (mmHg)[c]	MK	123 ± 12	123 ± 12	124 ± 12	123 ± 12	123 ± 12	122 ± 12	121 ± 12	120 ± 13	119 ± 14	117 ± 17
	CX	102 ± 10	97 ± 12	96 ± 8	93 ± 7	89 ± 8	90 ± 7	91 ± 9	92 ± 12	94 ± 16	95 ± 14
SAP (mmHg)[c]	MK	134 ± 13	134 ± 13	135 ± 12	134 ± 12	136 ± 7	134 ± 11	132 ± 11	131 ± 14	131 ± 13	129 ± 13
	CX	112 ± 9	109 ± 11	106 ± 10	102 ± 12	100 ± 11	99 ± 11	100 ± 9	103 ± 13	104 ± 18	105 ± 12
HR (beats/min)	MK	70 ± 19	68 ± 15	64 ± 17	61 ± 18	57 ± 17	55 ± 19	54 ± 15	53 ± 15	54 ± 15	53 ± 12
	CX	59 ± 14	68 ± 14	57 ± 12	58 ± 8	62 ± 13	60 ± 12	59 ± 14	58 ± 22	53 ± 13	52 ± 12
RR (breaths/min)[c]	MK	34 ± 14	37 ± 21	37 ± 20	33 ± 18	32 ± 20	37 ± 24	36 ± 22	36 ± 23	34 ± 22	36 ± 21
	CX	29 ± 9	24 ± 8	23 ± 10	21 ± 9	20 ± 9	20 ± 9	22 ± 13	22 ± 11	23 ± 14	21 ± 12
TEMP (°C)[c]	MK	39.8 ± 0.9	39.8 ± 0.8	39.8 ± 0.8	39.8 ± 0.8	39.7 ± 0.7	39.6 ± 0.6	39.5 ± 0.5	39.5 ± 0.5	39.5 ± 0.5	39.3 ± 0.6
	CX	40.7 ± 1.4	40.4 ± 1.3	40.3 ± 1.4	40.5 ± 1.4	40.9 ± 0.9	40.9 ± 0.9	40.9 ± 0.9	40.8 ± 0.9	40.7 ± 0.9	40.9 ± 1.8

Tx — treatment; DAP — diastolic arterial pressure; MAP — mean arterial pressure; SAP — systolic arterial pressure; HR — heart rate; RR — respiration rate; TEMP — body temperature

[a] medetomidine 100 µg/kg; ketamine 2.5 mg/kg

[b] carfentanil 10 µg/kg; xylazine 0.3 mg/kg

[c] significant difference between treatments

[d] significant difference within treatment (compared to reading at 15 min)

Data are expressed as mean ± standard deviation

Fentazine is a very useful combination for immobilization of deer, but is unavailable in Canada. Carfentanil is labeled for immobilization of deer in Canada and is often combined with sedatives, such as xylazine, to improve muscle relaxation (3,4). Advantages of carfentanil include rapid, reliable induction (2,3) and low volume required (2,3). Carfentanil-induced immobilization is readily antagonized with naltrexone (5). One major disadvantage encountered with carfentanil is the hazard to human safety (3). Hypoventilation and hypoxemia has been observed during carfentanil immobilization (6–8), and the cardiopulmonary effects of this drug have not been well described in deer. One of the objectives of this study is to describe the cardiopulmonary effects of carfentanil-xylazine-induced immobilization in deer.

Medetomidine is a potent, selective α_2-agonist that is readily antagonized with atipamezole (9). Medetomidine can be combined with ketamine to produce reliable immobilization in a wide variety of domestic and non-domestic animals (9–12). Medetomidine is not currently commercially available for wildlife immobilization, but once it is, it should prove to be a useful drug for immobilization of game-farmed species. The second objective of this study is to describe the cardiopulmonary effects of medetomidine-ketamine immobilization in deer, and compare the combination to carfentanil-xylazine.

Materials and methods

Three mule deer (*Odocoileus hemionus*) and 4 mule deer/white-tailed deer hybrids (*Odocoileus hemionus* × *Odocoileus virginianus*) (3 male, 4 female) were used in this study. This study was approved by the University of Saskatchewan Animal Care Committee. On the day of the study, the deer were moved into a handling facility and weighed. Each deer received an intramuscular (IM) injection of carfentanil (Wildnil, 10 µg/kg; Wildlife Laboratories, Fort Collins, Colorado, USA) + xylazine (Rompun, 0.3 mg/kg; Haver, Bayer Division, Chemagro, Etobicoke, Ontario) (CX) or medetomidine (100 µg/kg; Farmos Group, Turku, Finland) + ketamine (Rogarsetic, 2.5 mg/kg; Rogar STB, London, Ontario) (MK). Drugs were administered into the gluteal muscle mass by hand injection. Treatments were administered in random order at least 5 d apart. Immediately following drug injection, the deer was moved into a holding pen and the time from injection to sternal recumbency (TI) was noted. Once the deer was safely immobilized, the eyes were lubricated with methylcellulose drops and the animal was loaded onto a cart and moved to a heated facility for the study. The deer were maintained in left lateral recumbency and a 20-gauge, 5-cm catheter (Surflo, Terumo Medical Corporation, Irvine, California, USA) was placed in the auricular artery. The catheter was connected with non-compliant tubing to a pressure transducer (Uniflow, Baxter Healthcare Corporation, Irvine, California, USA), which was in turn connected to a physiological monitor (Propaq 400 EL, Protocol Systems Inc., Beaverton, Oregon, USA). The transducer was zero-calibrated at the level of the sternum. Direct systolic (SAP), mean (MAP), and diastolic (DAP) arterial pressures were recorded from the arterial catheter every 5 min. Heart rate (HR) was calculated from the arterial pressure tracing, and was recorded every 5 min. Esophageal temperature (TEMP) was monitored with a temperature probe connected to the physiological monitor and was recorded every 5 min. Blood samples were collected from the arterial catheter at 15, 30, 45, and 60 min post injection (PI). These samples were stored on ice and analyzed within 3 h of collection. The PaO_2, $PaCO_2$, pH, and base excess (BE) values were determined from the arterial samples with a blood gas analyzer (Copenhagen

Table II. Blood gas values following the administration of medetomidine + ketamine[a] (MK) or carfentanil + xylazine[b] (CX) in mule deer and mule deer/white-tailed deer hybrids

Parameter	Treatment	Time from immobilization (min)			
		15	30	45	60
pH[c]	MK	7.29 ± 0.07	7.31 ± 0.05[d]	7.35 ± 0.04[d]	7.37 ± 0.03[d]
	CX	7.30 ± 0.03	7.34 ± 0.02[d]	7.38 ± 0.03[d]	7.42 ± 0.03[d]
Base excess	MK	−2.7 ± 4.2	0.3 ± 4.2[d]	2.4 ± 2.9[d]	3.6 ± 2.4[d]
	CX	−1.9 ± 2.3	1.7 ± 2[d]	3.4 ± 2[d]	4.1 ± 1.6[d]
PaO_2 (mmHg)	MK	59 ± 11	58 ± 9	59 ± 11	62 ± 15
	CX	52 ± 9	58 ± 12	63 ± 12	68 ± 10[d]
$PaCO_2$ (mmHg)	MK	51 ± 4	53 ± 5	52 ± 3	52 ± 5
	CX	50 ± 6	52 ± 6	50 ± 6	46 ± 5

[a] medetomidine 100 µg/kg; ketamine 2.5 mg/kg

[b] carfentanil 10 µg/kg; xylazine 0.3 mg/kg

[c] significant difference between treatments

[d] significant difference within treatment (compared to reading at 15 min)

Data are expressed as mean ± standard deviation

Radiometer, Acid Base Laboratory 330, Copenhagen, Denmark). Arterial samples were corrected for body temperature and hemoglobin concentration. Hemoglobin concentration was measured by the cyanomethemoglobin method. Respiratory rate (RR) was determined at 5-minute intervals from observation of chest excursions. A lead II electrocardiogram was constantly monitored for the presence of arrhythmias. Quality of immobilization was a subjective determination based on observation, and included factors such as: presence of muscle rigidity and spontaneous movement. The deer were also observed for signs of rumenal tympany. Deer that demonstrated excessive movement or muscle rigidity following the administration of CX received supplemental xylazine in 0.1 mg/kg increments, intravenously (IV), to improve muscle relaxation and facilitate the experimental procedures.

At 60 min post injection, the deer were loaded onto a cart and returned to the handling facility for recovery. Medetomidine was antagonized with 500 µg/kg of atipamezole (Farmos Group), with the dose split half IV into the jugular vein, and half IM into the gluteal muscle mass. Carfentanil was antagonized with 1 mg/kg of naltrexone (Sigma Chemical Company, St. Louis, Missouri, USA), administered into the gluteal muscle mass. The time from drug administration to standing (TR) was recorded.

Data were analyzed with a commercial statistics package (Stat View 4.0, Abacus Concepts, Berkeley, California, USA). Differences between treatment groups were compared with 2-way analysis of variance (ANOVA). Differences over time within treatments were compared with repeated measures ANOVA, and a Dunnett's test at specific time points. A significance level of $P < 0.05$ was used in the analysis.

Results

The deer weighed 46.4 ± 25 kg (all values are mean ± SD). Time to sternal recumbency was 282 ± 83 s with CX and 230 ± 68 s with MK. Values for SAP, MAP, DAP and RR were significantly higher with MK. Heart rate was not significantly different between treatments. The TEMP value was significantly higher with CX. These values are presented in Table I. Base excess, $PaCO_2$, and PaO_2 were not significantly different between treatments, pH was significantly higher during CX immobilization and increased significantly over time with both treatments. Base excess also increased significantly over time with both treatments. With CX, the PaO_2 increased significantly over time, while it did not change during immobilization with MK. Hypoxemia ($PaO_2 < 60$ mmHg) was observed with both treatments. The lowest PaO_2 recorded during CX immobilization was 38.8 mmHg and the lowest PaO_2 with MK was 42.8 mmHg. Both of these values were from the same individual. Blood gas data are illustrated in Table II.

Several arrhythmias and ECG changes were observed with CX. One animal demonstrated a large, spiked T-wave early in the immobilization, atrial premature contractions were noted in another animal, a junctional rhythm was noted in one animal, and ventricular premature contractions were noted in one animal. Arrhythmias were not noted with MK.

Immobilization with CX was characterized by increased activity prior to induction. Spontaneous movement was common with CX; such movement of the head and limbs was noted in 4 animals. Three deer received 0.1 mg/kg of xylazine, IV, to prevent excessive movement. One animal developed mild rumenal tympany during immobilization with MK. Immediately following atipamezole administration the deer began to eructate and the rumenal tympany rapidly resolved.

Recovery from immobilization was rapid, with a time to standing from MK of 74 ± 37 s following atipamezole administration, and a time to standing from CX of 195 ± 84 s following administration of naltrexone. Deer were monitored for signs of re-narcotization for the 24-hour period following recovery. Re-narcotization was not noted in any individual with either of the protocols.

Discussion

Induction time was rapid with both of these combinations. Rapid induction is desirable, particularly if the animal is free-ranging. Recovery was also rapid following administration of antagonist drugs.

Mean arterial pressure was significantly higher during immobilization with MK. Medetomidine increases MAP by peripheral activation of α_2 receptors and increased systemic vascular resistance (6,11,12). Hypertension is commonly encountered during MK anesthesia (6,11,12). Without baseline data it is difficult to comment on the magnitude of increase in MAP. The average MAP in these deer throughout immobilization was 121.5 ± 12.6 mmHg. This is relatively low compared to other studies of medetomidine-based protocols (6,11,12), and is probably of minimal significance. Significant hypertension (MAP > 200 mmHg) was noted during carfentanil-xylazine immobilization in bongos (8).

Both treatments produced hypoxemia and hypercarbia. Hypercarbia was never severe, with all $PaCO_2$ values being less than 60 mmHg. Hypoxemia was severe, particularly in some individuals. The animal that developed the lowest recorded PaO_2 values appeared to be in good health and demonstrated no abnormalities in the complete blood count. Hypoxemia tended to resolve over time with CX, but they also appeared to be in a lighter plane of anesthesia towards the end of the immobilization. Oxygenation did not change over time with MK. A similar change in PaO_2 was observed in sheep (6). Hypoxemia with both treatments is the result of hypoventilation and ventilation perfusion mismatch. Hypoxemia, resulting from hypoventilation, should respond to supplemental inspired oxygen, but supplemental inspired oxygen will be of less benefit in improving hypoxemia resulting from ventilation perfusion mismatch, particularly if shunt flow is > 30% (13). Given the fact that supplemental inspired oxygen should tend to offset some of the decreased PaO_2 resulting from hypoventilation, and the fact that supplemental inspired oxygen should help to improve oxygenation if shunt flow is < 30%, animals receiving these combinations should receive supplemental inspired oxygen and increased inspired oxygen is recommended when animals are immobilized with either of these protocols. Further studies are indicated to determine the benefits of supplemental inspired oxygen with these combinations.

Mild hyperthermia was noted during immobilization with CX. Hyperthermia may be the result of increased activity prior to induction. Catecholamine release has been documented during CX immobilization (8). Catecholamines increase oxygen consumption and heat production (14) and could contribute to hyperthermia. Hyperthermia increases the risk of complications during CX immobilization, as it has been linked with capture myopathy (15). Hyperthermia in the face of hypoxemia is of particular concern, as hyperthermia increases oxygen demand, and would increase the risk of complications from hypoxemia. An effort should be made to avoid hypoxemia in the face of hyperthermia, reinforcing the recommendation to administer supplemental oxygen to deer immobilized with CX. Ambient temperature probably did not contribute to hyperthermia in these animals. The immobilizations were performed in mid-March. Environmental temperature during induction was 0–10°C, and snow was still on the ground.

Changes in BE, pH, and $PaCO_2$ were similar to those observed in domestic sheep immobilized with these combinations (6). Although baseline data was not available in the deer, the low pH early in the immobilization with MK and CX is probably due to increased $PaCO_2$ and represents a respiratory acidosis. The increase in BE over time may reflect early metabolic compensation for the acidosis. With CX, $PaCO_2$ tended to decrease over time. This would contribute to the increase in pH over time, it would also contribute to the increase in PaO_2 over time.

Several arrhythmias and ECG changes were noted during immobilization with CX. One animal developed an enlarged, spiked T-wave during CX immobilization. This animal had a PaO_2 of 40 mmHg when this occurred. The T-wave abnormality is probably the result of myocardial hypoxia (16). Similar T-wave changes were observed in hypoxemic sheep during CX immobilization (6). One animal developed sinus pauses and junctional escape beats. At the time of the arrhythmia the animal had a heart rate of 44–48 beats/min, and the arrhythmia was probably due to increased vagal tone. Both xylazine and the potent narcotics can increase vagal tone, resulting in bradyarrhythmias (16). Atrial premature contractions were observed in one individual and ventricular premature contractions were noted in another individual. Both of these arrhythmias can be caused by many factors (16). These animals were only mildly hypoxemic. Hypercarbia will result in catecholamine release, which may have contributed to these arrhythmias. Catecholamine release has been documented during carfentanil-induced immobilization in bongos (8). Changes in autonomic tone due to catecholamine release could have contributed to the development of ventricular premature contractions in these animals (16). Xylazine has been shown to decrease the arrhythmogenic threshold in the face of catecholamine release (17). The combination of catecholamine release, resulting from carfentanil administration, and myocardial sensitization, produced by xylazine, could have contributed to the development of ventricular premature contractions in these deer. In dogs, the major reasons to treat ventricular tachycardias are to improve hemodynamic stability and prevent sudden death (18). Rapid ventricular tachycardias can result in severe hemodynamic compromise or sudden death from ventricular tachycardia (18). The arrhythmias in these deer were probably of little physiological significance, as they were sporadic and blood pressure was well maintained during the arrhythmia.

Quality of immobilization was superior with MK. Immobilization with CX was characterized by head movement and sudden limb movement in several animals. It is possible that increasing the dose of xylazine in these animals could decrease spontaneous movement and improve muscle relaxation. Immobilization with MK was characterized by good muscle relaxation and no spontaneous movement. One individual immobilized with MK developed mild rumenal tympany. Ruminants frequently develop rumenal tympany during xylazine-induced sedation (19), and it is not surprising that it could occur during immobilization with medetomidine.

Recovery was more rapid following the administration of atipamezole. Since the atipamezole dose was administered by both IV and IM routes, and naltrexone was only administered IM, the more rapid recovery is probably reflective of the route of administration.

Both of these combinations will produce effective immobilization of mule deer and mule deer/white-tailed deer hybrids. The major complication with both combinations is hypoxemia, and supplemental oxygen should be administered during immobilization. A further complication with CX is hyperthermia. Immobilization with this combination is probably best performed during the cooler hours of the day, and supplemental inspired oxygen is further

recommended to avoid hypoxemic complications from increased oxygen demand.

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