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An Evaluation of Acepromazine/Ketamine for Immobilization of White-Tailed Deer (*Odocoileus virginianus*)

A.F. Ahmed, F.A. Al-Sobayil and Y. Al-Rauji
College of Agriculture and Veterinary Medicine, Qassim University, Buraydah 51452,
P.O. Box 6622, Al-Qassim, Saudi Arabia

Abstract: Effect of combination of acepromazine (0.2 mg kg⁻¹ IM) and ketamine (2.5 mg kg⁻¹ IM) on immobilization, physiological and hematological constituents of six healthy female white-tailed deer (*Odocoileus virginianus*) was studied. Onset and duration of anaesthesia were recorded. Physiological parameters (body temperature, heart, respiratory rates and arterial oxygen hemoglobin saturation) were investigated before administration of acepromazine and every 5 min after its injection and then every 10 min after administration of ketamine until recovery. Depth of anaesthesia was determined by recording reflexes (palpebral and corneal reflexes, lacrimation, jaw and anal reflexes, skin pinch, ear and pedal withdrawal reflexes and tongue protrusion). Blood samples were collected before injection of acepromazine, 20 min after injection of acepromazine, 30 min after injection of ketamine and after recovery. Animals showed marked decrease in spontaneous activity following administration of acepromazine and went to lateral recumbency 10 min after administration of ketamine. However, most of reflexes were either in mild or moderate stage. A significant stress-related increase in body temperature was seen after administration of acepromazine and this increase gradually decreased until end of the study. There was marked decrease in (Red blood cells) RBC and (White blood cells) WBC counts 20 and 30 min after administration of acepromazine and ketamine, respectively. In addition, there was significant increase in percentage of neutrophils and significant decrease in percentage of lymphocytes. It was concluded that anesthetic regime used in the present study was quite convenient and safe to immobilize white-tailed deer but not enough to maintain an adequate plane of surgical anesthesia.

Key words: Wild animals, anaesthesia, phenothiazines, dissociative anesthetics, white-tailed deer

INTRODUCTION

White-tailed deer (*Odocoileus virginianus*) is a medium-sized deer found throughout most of continental United States, southern Canada, Mexico, Central America, northern portions of South America as far south as Peru and some countries in Europe (Halls, 1984).

Chemical immobilization is necessary for capturing wild animals including white-tailed deer that often need to be captured for management and research. Many drugs have been used either individually or in combinations for chemical immobilization of deer (Jones, 1984; Walsh and Wilson, 2002). Chemical immobilizing agents have a role in reducing stress and pain-induced distress during velvet antler removal. Phenothiazine derivatives are commonly used drugs to calm animals. These drugs act on the central nervous system by blocking postsynaptic dopamine receptors and may also inhibit release of dopamine and depress the activity of the brain stem and its connections to the cerebral cortex

(Thurmon *et al.*, 1996). They also have anticholinergic, antihistaminic, antispasmodic and alpha adrenergic blocking effects (Plumb, 2002).

Acepromazine, a short-acting phenothiazine, has been widely used as a preanaesthetic or sedative agent in domestic and wild animals including wild ungulates (Montane *et al.*, 2003). In wild animals, many studies on acepromazine have focused on its effects on capture stress (Manziano and Manziano, 1978; Montane *et al.*, 2003; Lopez-Olvera *et al.*, 2007). Ketamine is the most commonly used dissociative for animal anaesthesia. After bolus intravenous injection, ketamine rapidly crosses the blood-brain barrier, quickly entering the brain and the brain/plasma concentration ratio becomes constant in less than 1 min. It is often used in combination with tranquilizers or sedatives to avoid the adverse effect of muscular rigidity and violent recovery (Muir *et al.*, 2000).

Acepromazine in combination with ketamine has been used in different species including horses and donkeys (Harbison *et al.*, 1974; Fisher, 1984), sheep (Harbison *et al.*, 1974; Thurmon *et al.*, 1975), dogs (Manziano and Manziano, 1978), cats (Ingwersen *et al.*, 1998), wolves (Sillero-Zubiri, 1996), rabbits (Vachon, 1999), ferrets (Ko *et al.*, 1998) and monkeys (Lopez *et al.*, 2002). The immobilization effects of acepromazine combined with ketamine have not been investigated yet in deer.

The objective of the present study was to evaluate a combination of Acepromazine/Ketamine (AK) for immobilizing white-tailed deer.

MATERIALS AND METHODS

This study was approved under the authority of Scientific and Research Deanship. Six healthy adult female white-tailed deer (ages 2 years, weight 31.3±2.0 kg) were used in this study. The deer were housed together in a large pen and fed green and dry alfalfa. The study was performed at the end of February 2008 and the environmental temperature ranged from 15-25°C. Food and water were withheld 24 h prior to immobilization. After manual capturing, the deer were tied at four limbs, weighed and left to get rest for 2 h. Each animal initially received an intramuscular injection of acepromazine (0.2 mg kg⁻¹ IM; Calmivet®, Vetoquinol S.A., France) (Janovsky *et al.*, 2000) and was followed 20 min later by ketamine (Ketavet, Delvet Pvt. Ltd., Australia) 2.5 mg kg⁻¹ IM (Caulkett *et al.*, 2000). Drugs were administered into the gluteal muscle mass by hand injection. The onset and duration of sedation and anesthesia were recorded. The parameters of physiological functions including body temperature (BT), heart rate (HR), respiratory rate (RR) and oxygen hemoglobin saturation were investigated before the administration of acepromazine and every 5 min (after the injection of acepromazine) and every 10 min (after the injection of ketamine) until complete recovery. HR was calculated by auscultation, RR by observing thoracic movements and BT was recorded using a digital thermometer. Pulse oximeter (504DX Digital Oximeter, Criticare Systems Inc. Waukesha, WI, USA) with probe placed either on the tongue or ear was used to determine the concentration of arterial oxygen hemoglobin saturation. A lead II electrocardiogram (Kenz Cardico 302, Suzuken Co. Ltd., Japan) was used to constantly monitor the patient for the presence of arrhythmias. The depth of anaesthesia was determined by recording various reflexes including palpebral, corneal reflexes, lacrimation, jaw, tongue, ear, anal, skin pinch and pedal reflexes. The reflexes were categorized into absent, mild, moderate and strong reflex (0-3). Jugular blood samples were collected from each animal by venipuncture into ethylenediaminetetraacetic acid (EDTA)-containing vacutainer tubes immediately before and 20 min after the injection of acepromazine, 30 min after the injection of ketamine and immediately after the full recovery. The count of red and white blood cells (RBC, WBC), the differential count of White Blood Cells (dWBC), Packed Cell Volume (PCV) and hemoglobin concentration (Hb) were determined using an automated machine (Cell-Dyn 3500, Abbott Diagnostics Santa Clara, CA, USA).

Data were expressed as Mean±SEM and analyzed with a commercial statistics package, SAS®. A repeated measures ANOVA was used as the statistical model to evaluate the differences over time.

When there was a significant difference, Dunnett's test was used to make individual comparisons. The differences were decided to be significant when p-value was less than 0.05.

RESULTS

The deer showed a marked decrease in spontaneous activity following the administration of acepromazine ($p < 0.05$). The calming effects occurred shortly (8 ± 1.2 min) after the injection of acepromazine and were characterized by laying down on the sternal recumbency and resting the head on the left flank (Fig. 1). Ten minute after the administration of ketamine, the deer were put in lateral recumbency. The mean duration of total immobilization was 80 (80 ± 4.1) min from injection of acepromazine to full recovery. Recovery was smooth without major complications. Signs of recovery started 30 min after ketamine administration as gradual increase in the strength of reflexes. Average time for the full recovery was 55 min (Range = 43-70) after ketamine administration. Full recovery was characterized by animal standing on its feet and running after being un-tied.

A non significant increase (65.2 ± 7.7) in the respiratory rate ($p > 0.05$) was seen after the administration of acepromazine and this increase returned to the normal levels (50.7 ± 5.2) 40 min after the injection of ketamine (Fig. 2). A non significant reduction in the heart rate (73.5 ± 5.7) was observed after the administration of acepromazine. Ten minutes after the administration of ketamine, HR increased non significantly (80.5 ± 3.1) and then stabilized until the end of the study (Fig. 2). Neither the injection of acepromazine nor ketamine produced significant effects on the concentration of oxygen saturation (Fig. 3). A significant increase (41.1 ± 0.4) was seen in BT after the administration of acepromazine ($p < 0.05$) and this increase gradually decreased until the end of the study (40.1 ± 0.1) (Fig. 3).

ECG changes were variable and included bradycardias and arrhythmias that appeared 15 min after administration of acepromazine. There were elevations in the QRS complex waves (0.8 ± 0.1 mV vs. 0.4 ± 0.03 mV at 0 time), P waves (0.3 ± 0.01 mV vs. 0.15 ± 0.03 mV at 0 time) and T waves (0.8 ± 0.06 mV vs. 0.2 ± 0.09 mV at 0 time). Bradyarrhythmias continued after ketamine administration, however, the intensity of the QRS complex waves increased (1.0 ± 0.1 mV) and T waves decreased (0.4 ± 0.03 mV) 30 min of ketamine administration.

Tongue and ear reflexes decreased and became moderate 10 and 15 min, respectively after the injection of acepromazine and became mild 30 min after the injection of ketamine and gradually increased until returning to the normal level at the end of the study (Table 1). Pedal and anal reflexes became moderate 15 min after the administration of acepromazine and became almost mild 30 min after the administration of ketamine and gradually elevated until the end of the study (Table 1). Jaw reflex



Fig. 1: The calming effects of acepromazine

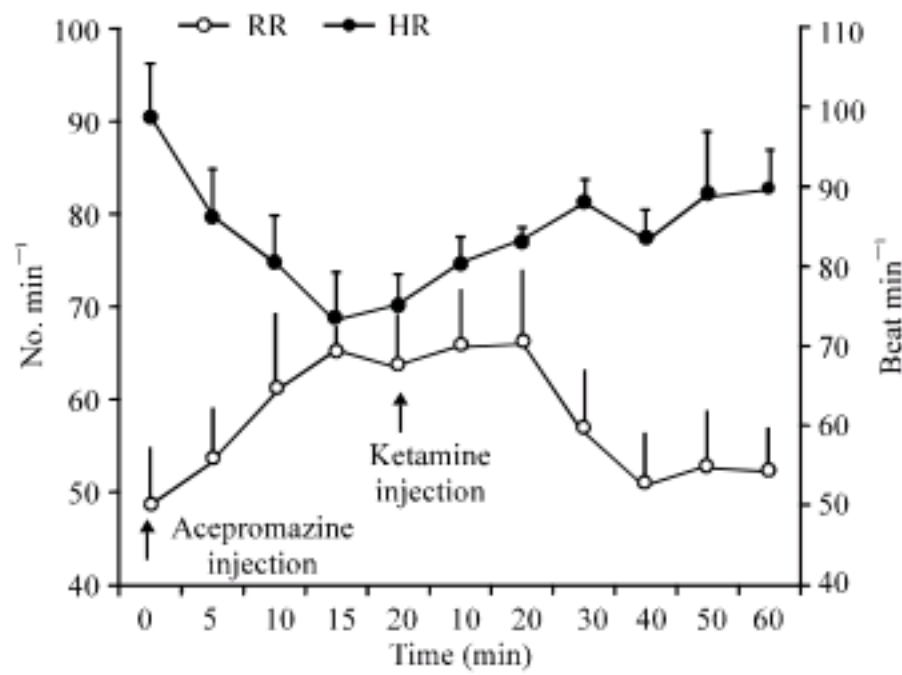


Fig. 2: Mean (\pm SE) of respiratory and heart rates of white-tailed deer (n = 6) immobilized by acepromazine/ketamine combination

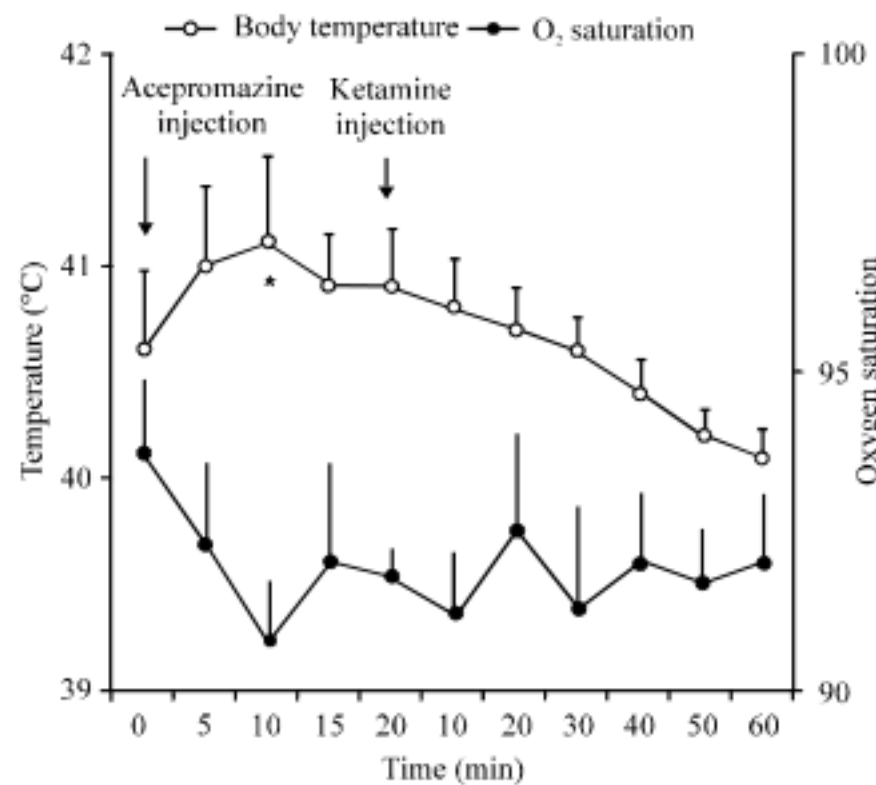


Fig. 3: Mean (\pm SE) of body temperature and oxygen saturation of white-tailed deer (n = 6) immobilized by acepromazine/ketamine combination. *Temperature value is significantly different from base line value at $p < 0.05$

was moderate ten minutes after the injection of acepromazine and became completely absent 20 min after the injection of ketamine for a period of 10 min. After that, jaw reflex gradually increased until it became normal at the end of the study (Table 1). Fifteen minute after the administration of acepromazine, the palpebral reflex was moderate (Table 1). It became mild 20 min after the injection of ketamine and continued to be so for 10 min thereafter. A gradual increase in the palpebral reflex was seen after that until reaching its normal value at the end of the study. Corneal reflex was moderate 10 min after the administration of ketamine and became mild 30 min after the injection of ketamine (Table 1). After that, corneal reflex gradually increased until it became normal at full recovery. Skin pinch and lacrimation reflexes were not observed in deer neither before nor after the administration of acepromazine/ketamine.

Mild salivation was seen 15 min after the administration of acepromazine and the amount of salivation increased 10 min after the administration of ketamine (Table 1). The amount of salivation gradually decreased and became mild. However, salivation still continued even after full recovery.

Table 1: Means (\pm SE) of reflexes of white-tailed deer (n = 6) immobilized by acepromazine/ketamine combination

Time (min)	Reflexes							
	Tongue	Ear	Pedal	Anal	Jaw	Salivation	Palpebral	Corneal
0	3.0 \pm 0.0	3.0 \pm 0.00	3.0 \pm 0.00	3.0 \pm 0.00	3.0 \pm 0.00	0.0 \pm 0.00	3.0 \pm 0.0	3.0 \pm 0.00
5(A)	3.0 \pm 0.0	2.8 \pm 0.16	3.0 \pm 0.00	2.8 \pm 0.16	2.8 \pm 0.16	0.3 \pm 0.20	3.0 \pm 0.0	3.0 \pm 0.00
10(A)	2.0 \pm 0.2	2.6 \pm 0.20	2.6 \pm 0.20	2.4 \pm 0.16	1.8 \pm 0.16	0.8 \pm 0.16	2.3 \pm 0.2	2.8 \pm 0.16
15(A)	2.0 \pm 0.16	2.0 \pm 0.24	2.0 \pm 0.00	1.9 \pm 0.16	1.3 \pm 0.20	1.0 \pm 0.00	2.0 \pm 0.0	2.5 \pm 0.20
20(A)	2.0 \pm 0.0	2.0 \pm 0.24	2.0 \pm 0.00	1.8 \pm 0.16	1.0 \pm 0.24	1.0 \pm 0.00	2.0 \pm 0.0	2.3 \pm 0.20
10(K)	1.8 \pm 0.16	1.8 \pm 0.16	2.0 \pm 0.00	1.5 \pm 0.20	1.0 \pm 0.00	1.6 \pm 0.20	1.3 \pm 0.2	2.0 \pm 0.00
20(K)	1.6 \pm 0.2	1.6 \pm 0.20	1.6 \pm 0.20	1.5 \pm 0.29	0.0 \pm 0.16*	1.5 \pm 0.20	1.0 \pm 0.2*	2.0 \pm 0.00
30(K)	1.1 \pm 0.2*	1.3 \pm 0.2*	1.5 \pm 0.20	1.2 \pm 0.2*	0.0 \pm 0.16*	1.5 \pm 0.20	1.0 \pm 0.2*	1.2 \pm 0.16*
40(K)	1.5 \pm 0.2	1.6 \pm 0.20	1.8 \pm 0.16	1.2 \pm 0.16*	0.8 \pm 0.16	1.3 \pm 0.20	1.3 \pm 0.2	1.5 \pm 0.16
50(K)	2.0 \pm 0.24	2.0 \pm 0.24	2.2 \pm 0.16	2.0 \pm 0.00	1.7 \pm 0.20	1.2 \pm 0.24	2.0 \pm 0.0	2.0 \pm 0.00
60(K)	3.0 \pm 0.0	3.0 \pm 0.24	3.0 \pm 0.00	3.0 \pm 0.00	3.0 \pm 0.00	0.8 \pm 0.16	3.0 \pm 0.0	3.0 \pm 0.00

(A) = Acepromazine injection, (K) = Ketamine injection, *Value is significantly different from 0 min value (base value) at $p < 0.05$

Table 2: Means (\pm SE) of hematological parameters of white-tailed deer (n = 6) immobilized by acepromazine/ketamine combination

Time (min)	Hematological parameters							
	RBCs count (cell $\times 10^6$ μ L $^{-1}$)	WBCs count (cell $\times 10^3$ μ L $^{-1}$)	Neutrophils	Lymphocytes	Monocytes	Eosinophils	Hb (g dL $^{-1}$)	PCV (%)
0	6.4 \pm 0.05	11.7 \pm 1.2	67.5 \pm 3.9	26.5 \pm 3.8	2.3 \pm 0.7	3.0 \pm 1.9	15.9 \pm 0.6	35.3 \pm 0.6
20 (A)	6.2 \pm 0.08*	8.9 \pm 0.6*	78.0 \pm 3.1*	15.3 \pm 2.2*	3.6 \pm 0.9	3.6 \pm 1.3	14.0 \pm 0.7	33.6 \pm 0.5
30 (K)	6.2 \pm 0.05*	8.5 \pm 0.5*	80.0 \pm 3.4*	13.6 \pm 3.1*	1.8 \pm 0.4	4.2 \pm 1.8	13.2 \pm 0.7	32.9 \pm 0.7
Recovery time	6.3 \pm 0.08	9.3 \pm 0.4	76.1 \pm 2.5	17.5 \pm 2.5	2.5 \pm 0.5	3.8 \pm 1.5	13.1 \pm 0.5	31.6 \pm 1.6

(A) = Acepromazine injection, (K) = Ketamine injection, *Value is significantly different from 0 min value (base value) at $p < 0.05$

Twenty minute after injection of acepromazine, there was significant decrease in the counts of RBC and WBC ($p < 0.05$) and 30 min after the injection of ketamine (Table 2). After complete recovery, there was a non significant increase in the counts of RBC and WBC. The percentage of neutrophils in the blood significantly increased 20 and 30 min after the administration of acepromazine and ketamine, respectively. The percentage of lymphocyte in the blood significantly decreased 20 and 30 min after the administration of acepromazine and ketamine, respectively (Table 2). Non significant increase was seen in the percentage of blood monocytes and eosinophils 20 min after the administration of acepromazine. Non significant decrease in the percentage of blood monocytes was observed 30 min after the injection of ketamine. Thirty minute after the administration of ketamine, the percentage of eosinophils slightly increased. There were non significant decrease in the concentration of Hb and the percentage of PCV after the injection of acepromazine and ketamine (Table 2).

DISCUSSION

The present study showed rapid effects and long duration after the administration of AK (8 \pm 1.2; 80 \pm 4.1 min, respectively) in white-tailed deer. Moreover, there was slight increase in the RR after the administration of acepromazine in white-tailed deer. After the injection of acepromazine, the pattern of respiration became shallow and this might be the reason for the increase in the respiratory rate. The dose of acepromazine that we used in the present study was quite high when it is compared with the recommended dose (Nielsen, 1999). It has been reported that acepromazine may decrease tidal volume when administered in large dose (Muir *et al.*, 2000). Therefore, this might be the reason for the slight increase of RR in the present study. In dogs, Farver *et al.* (1986) have reported that acepromazine significantly decrease breathing rate. Over all in the present study, combination of acepromazine/ketamine did not significantly alter RR. Similar result has been reported in cats (Ingwersen *et al.*, 1998).

In the present study, there was slight decrease in HR after administration of acepromazine, but it increased after the injection of ketamine. A significant decrease in HR after the administration of acepromazine has been reported in roe deer (*Capreolus capreolus*) (Montane *et al.*, 2003) and in Southern chamois (*Rupicapra pyrenaica*) (Lopez-Olvera *et al.*, 2007). In horses, it has been reported that HR did not change after the administration of acepromazine (Marroum *et al.*, 1994), whereas in another study, the administration of acepromazine increased the HR (Muir and Mason, 1996). Ketamine generally stimulates the cardiovascular system due to central effects that mimic the effect of sympathetic nervous system stimulation which overrides any direct peripheral cardiovascular depressant effects of this drug (Reves *et al.*, 2000). The result is an increase in HR and an increase in cardiac output with stroke volume remaining unchanged (Muir *et al.*, 2000) or became lower (Farver *et al.*, 1986). In healthy cats, HR was not significantly altered after the administration of acepromazine/ketamine (Ingwersen *et al.*, 1998). The concentration of oxygen saturation did not change over time a result that was similar to that reported in healthy cats (Ingwersen *et al.*, 1998).

A significant elevation was seen in BT after the administration of acepromazine and gradual decrease was seen after the injection of ketamine. In fact, acepromazine did not have any effect on BT (Montane *et al.*, 2003), although hypothermia is a well known non-desired effect of phenothiazines (Plumb, 2002). In certain stressful situation, increases in BT cannot only be explained by physical activity, but another component called Stress-Induced Hyperthermia (SIH) might contribute in that explanation (Bakken *et al.*, 1999; Moe and Bakken, 1997). SIH is a regulated shift of the thermoregulatory set point (Briese and Cabanac, 1991) mediated by prostaglandin E and interleukins one and six (Le May *et al.*, 1990). SIH in mice is time dependent that takes ten minutes to reach a stable high level which is 1-1.5°C higher than the baseline and it takes 60 min to return to baseline (Zethof *et al.*, 1994). In farmed silver foxes (*Vulpes vulpes*), SIH lasted 60-90 min after a short stress or presentation (Moe and Bakken, 1997). Therefore, the changes in BT observed in the present study might be due to SIH.

ECG changes included bradycardias and arrhythmias that appeared 15 min after administration of acepromazine in the current study. Generally speaking, acepromazine produced sinus tachycardia and rarely sinus bradycardia (Muir and Mason, 1996). However, when given in high doses (0.4-1.0 mg kg⁻¹) prior to epinephrine administration; acepromazine prevents cardiac dysrhythmias and ventricular fibrillation in dogs during barbiturate, methoxyflurane and halothane anaesthesia (Wiersig *et al.*, 1974). Moreover, acepromazine produces minimal direct myocardial depression in patients with impaired cardiac output (Paddleford and Harvey, 1996).

The moderate to mild responses of reflexes after injection of acepromazine/ketamine in the present study might indicate insufficient depth of anaesthesia to perform surgical operations; however, it was sufficient to immobilize white-tailed deer. Disappearance of jaw reflex 20 min after administration of ketamine supports that jaw reflex in white-tailed deer is a good indicator for muscle relaxation, but not for the depth of anaesthesia. Moreover, no need to use anticholinergic drugs when immobilizing white-tailed deer with acepromazine/ketamine since a slight amount of salivation was observed after their administration. The present study showed that skin pinch is not a good parameter for evaluating pain reflex in the white-tailed deer because the reflex was absent before and after the administration of the anesthetic agents. Skin pinch reflex may be weak in white-tailed deer or even absent.

Hematological findings have shown significant decrease in the RBC and WBC counts 20 min after acepromazine administration and 30 min after ketamine administration. Hb and PCV slightly decreased 20 min after the administration of acepromazine and 30 min after the administration of ketamine. This can be explained by the alpha-adrenergic blocking effect of acepromazine which might induce relaxation to the spleen and consequently cause splenic sequestration of erythrocytes (Jain, 1993). In horses, PCV decreased over time after the administration of acepromazine (Marroum *et al.*, 1994; Parry and Anderson, 1983). The reduction of PCV percentage after the administration of acepromazine has been

reported as the most sensitive variable among the other physiological and hematological variables in horses (Marroum *et al.*, 1994). Therefore, it is not recommended to administer acepromazine in cases with blood loss. Crooks *et al.* (2003) have found that hematological parameters including RBC, PCV and Hb were lower in blood samples collected from skunks anesthetized with the combination of acepromazine/ketamine.

The present study has shown that there was significant neutrophilia and lymphopenia 20 and 30 min following the administration of acepromazine and ketamine, respectively. Stressful events such as capture and handling can affect total and differential leukocyte counts. In domestic animals, neutrophilia and lymphopenia appear after exposure to stress (Jain, 1993). Although deer in this study were injected with acepromazine and ketamine, it appeared that they were still under stress which might be associated with the environment stress factors. It seems that stress have lasted until 20-30 min after ketamine administration. At this time, elevated body temperature and respiratory rate had returned to base line values. Levels of stress indicators such as blood glucose and cortisol were not measured at the current study.

CONCLUSION

Combination of acepromazine (0.2 mg kg⁻¹ IM) and ketamine (2.5 mg kg⁻¹ IM) produced safe and effective immobilization of white-tailed deer, but was not enough to maintain an adequate plane of surgical anaesthesia. Recovery was smooth without major complications. Stress-related hyperthermia was observed after the administration of acepromazine, but it decreased following the injection of ketamine. This might indicate that deer were still under stress even though they were injected with acepromazine/ketamine combination and this stress might be associated with environmental stress factors. Immobilization regimen used in the present study might be useful prior to transportation and for performing minor surgical procedures of deer in zoological parks and farms.

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