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Comparison of the electrocardiographic findings, heart rates, cloacal temperatures, respiratory rates, and anaesthetic effects of medetomidine-ketamine and detomidine-ketamine combination in the buzzards (*Buteo buteo*)

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ABSTRACT: The aim of the present study was to compare the electrocardiographic findings, heart rate, cloacal temperature, respiratory rate, and anesthetic effects of medetomidine-ketamine to detomidine-ketamine in buzzards. Fourteen buzzards of unknown sex and age and weighing a mean \pm standard deviation (SD) of 789.29 ± 82.97 g were included in the study. The buzzards were randomly assigned to two groups: MK group (0.1 mg kg⁻¹ medetomidine and 10 mg kg⁻¹ ketamine 10 minutes after medetomidine application) and group DK (0.3 mg kg⁻¹ detomidine and 10 mg kg⁻¹ ketamine 10 minutes after medetomidine application). Heart rates (HR), respiratory rates (f_R), and cloacal temperatures (CT) were recorded before administration of any drug (0) and then 5 and 10 minutes after medetomidine and 5, 10, 15, 30, 45, 60 minutes after ketamine. At this point, atipamezole (0.5 mg/kg, IM) was administered and measurement recorded at 5, 10 and 15 minutes. Serum electrolyte and blood gases were measured before and during anesthesia. Electrocardiogram was recorded before and during anesthesia. Distribution and suitability of the data were evaluated by the Shapiro-Wilks test. Statistical tests were performed via two-way variance analysis in repeated measures, and multiple comparisons were corrected with the post-hoc Generalized Linear Model (GLM). Differences were considered to be statistically significant if $p < 0.05$. Onset of anesthesia time was 4.1 ± 2.3 and 3.9 ± 1.3 minutes (mean \pm standard deviation) in groups MK and DK, respectively. Reflexes (righting reflex, toe pinch reflex, feather plucking reflex, palpebral reflex) disappeared studied in both groups during anesthesia. HR, f_R , and CT were significantly decreased in both groups ($p < 0.001$). Baseline blood gases values showed significantly increases in pCO₂ and HCO₃ and decreases in pH and pO₂ during anesthesia in both groups ($p < 0.05$).

In conclusions medetomidine-ketamine and detomidine-ketamine combinations were both sufficient to provide desired level of anesthesia, analgesia and muscle relaxation.

Keywords: Anesthesia, Buteo Buteo, Detomidine, ECG, Ketamine, Medetomidine.

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INTRODUCTION

In recent years, avian anesthesia has been used more widely to facilitate physical restraint, clinical and radiographic examinations, and minor or major surgical interventions. General anesthesia in various avian species may be produced by administration of either inhalational or injectable agents. Injectable anesthetic agents may offer advantages such as requiring only minimal equipment, rapid administration and low cost (Durrani et al., 2009; Machin and Caulkent 1998). Several injectable anesthetic agents have been used in avian species including α_2 adrenergic agonist (xylazine, medetomidine, detomidine), barbiturates, dissociatives, alphaxalone, propofol and benzodiazepines (Christensen et al., 1987; Durrani et al., 2014; Saqib and Parkaash 2017). The α_2 adrenergic agonists (medetomidine, detomidine) are widely used parenterally as sedatives and analgesic agents for bird species. They are easy to administer, very effective, and safe (Paddleford and Harvey 1999; Pollock et al., 2001). Some groups have reported that medetomidine and detomidine are usually used in combination with ketamine (Christensen et al., 1987; Ashraf et al., 2009). Medetomidine (M) is a potent and selective α_2 adrenoceptor agonist that produces reliable sedation and analgesia. Detomidine is a α_2 adrenoceptor agonist with sedative, muscle relaxant, and analgesic properties (Paddleford and Harvey 1999). Ketamine is phencyclidine that induces dissociative anesthesia. Ketamine anesthesia is associated with poor muscle relaxation, muscle tremors, myotonic contractions, and opisthotonus. Therefore, it is usually necessary to administer a drug such as medetomidine, an α_2 agonist compound, to prevent seizures and produce muscle relaxation (Christensen et al., 1987; Mahmud et al., 2014). Atipamezole (AT) is a strong and selective α_2 receptor antagonist that increases the release of noradrenaline in both the central nervous system and the peripheral nervous system. It is used to reverse the effects of α_2 adrenoceptor agonists such as detomidine and medetomidine (Jager et al., 1998).

The aim of the present study was to compare the electrocardiographic findings, heart rates, cloacal temperatures, respiratory rates, and anesthetic effects of medetomidine- ketamine or detomidine-ketamine anesthetics in buzzards (*Buteo buteo*).

MATERIALS AND METHODS

The study was approved by Aydin Adnan Menderes University's Institutional Animal Care and Use Committee (file number 2017/114) and Turkish Re-

public Ministry of Agriculture and Forestry General Directorate of Nature Conservation and National Park (Approval no: 16.10.2019/E.3157257).

Animals

Fourteen buzzards of unknown gender which were given general anesthesia for clinical examination and orthopedic problems (4 tibiatarsus, 2 tasometatarsus, 2 humerus, 3 antebrachium, 2 carpometacarpus fracture, 1 metacarpophalangeal luxation) in Aydin Adnan Menderes University Veterinary Faculty Surgery Department were enrolled in the present study. Body weights were 789.29 ± 82.97 g [mean \pm standard deviation (SD)]. The buzzards were housed in individual cages of sufficient size (2.5x3x2.5 m) at the Kanat- Ger Rehabilitation Center. Buzzards were fasted for 6 hours before the induction of anesthesia, but water was not withdrawn. Physical examination on each buzzards were performed prior to general anesthesia.

Anesthesia protocols

The buzzards were divided randomly into two equal groups: Medetomidine-Ketamine (MK, group 1) and Detomidine-Ketamine (DK, group 2). In group 1 (MK), the buzzards were injected with 0.1 mg/kg medetomidine (1 mg ml⁻¹, Domitor® Pfizer, Turkey) and 10 mg kg⁻¹ ketamine (100 mg/ml, Alfamine® Ege Vet, Turkey) intramuscularly (IM) 10 minutes after medetomidine application. In group 2 (DK), each buzzard was given 0.3 mg kg⁻¹ detomidine (10 mg/ml, Domesedan® Pfizer, Turkey) and 10 mg kg⁻¹ ketamine (IM) 10 minutes after detomidine application. All drugs were administered IM into the deep pectoral muscle using an insulin syringe. In both groups, the anesthetic effects of the drugs were reversed by the administration of 0.5 mg kg⁻¹ Atipamezol (5 mg/ml Antisedan®, Pfizer, Turkey) IM at 60 min after ketamine administration into the buzzards. The buzzards were gently restrained in lateral recumbency during administration.

Assessment of the clinical effect of anesthesia

Onset time of sedation and body reflexes (righting reflex, toe pinch reflex, feather plucking reflex, palpebral reflex and table knock reflex) were evaluated in this study (Durrani et al., 2009).

Evaluation of the depth of anesthesia

Anesthetic effects of the drug combination during the anesthetic period were assessed according to Uzun *et al.*, 2003.

Physiological Parameters

Cloacal temperature (CT, °C), heart rate ECG tracing (HR, beats minute⁻¹) and respiratory rate (f_R breaths minute⁻¹) of the buzzards from the two groups were recorded before administration of any drug (0) and then 5 min and 10 min after medetomidine and 5, 10, 15, 30, 45, 60 min after ketamine, and 5, 10, 15 min after administration of AT. Heart rate (HR) was calculated from ECG recordings respiratory rate (f_R) was determined by direct observation of movement of the sterna. Cloacal temperature (CT) was measured by with a digital thermometer

Blood Analysis

All measurements during anesthesia were taken after 20 minutes administer ketamine. Serum electrolyte [sodium (Na), ionized calcium (Ca⁺⁺), potassium (K)] and blood gases [arterial pH (pH), arterial carbon dioxide partial pressure (pCO₂), arterial oxygen partial pressure (pO₂) bicarbonate concentration (HCO₃) hematocrit (HCT) and oxyhemoglobin saturation (O₂Sat)] were evaluated in blood samples (1 ml) taken before (at time 0) and during (20 min after on ketamine) anesthesia. Venous blood sample (superficial Ulnar vein, 1 mL) were collected in vacuum plasma tubes (heparinized) for the measurement of electrolytes and blood gases with an automated blood gas analyzer (Model ITC Irma Tripoint, Blood Analysis System, USG).

Electrocardiography Analysis

ECG was recorded before and during anesthesia with MP 30 Ultimate System (Biopac®) with fre-

quency response (0.05-35 Hz). Alligator clip electrodes were attached to the propatagium of the right and left wings and to the inguinal skin-fold of the left and right legs. Good clip to skin contact was established by using alcohol. Nomenclature and ECG interpretation were performed according to the standard methods (Wiemeyer *et al.*, 2013). The amplitudes of waves (P, QRS, T) the durations of waves (P, QRS, T), intervals (PR and QT) and cardiac rhythm were manually determined on lead II at 50 mm/sec and 1 cm = 1 mV.

Statistical analysis

Statistical analysis of the data was performed using the SPSS 19.0 statistical package program (IBM Corp., Waltham, MA, USA). All data are presented as the arithmetic mean (\bar{x}) and the standard error of the mean (S \bar{x}). Distribution and fitness of the data were evaluated by the Shapiro-Wilks test. Statistical tests were performed via two-way variance analysis in repeated measures, and multiple comparisons were corrected with the post-hoc Generalized Linear Model (GLM). Differences were considered statistically significant if $p < 0.05$.

RESULTS

Evaluation of the depth of anesthesia, values for physiological parameters (HR, f_R and CT), blood gas values and ECG findings (Figure 1) are shown in Tables 1, 2, 3 and 4, respectively. The onset time of sedation in the MK group was 4.1 ± 2.28 minutes and 3.96 ± 1.31 minutes in the DK group. There were no intraoperative mortality and body reflexes (righting

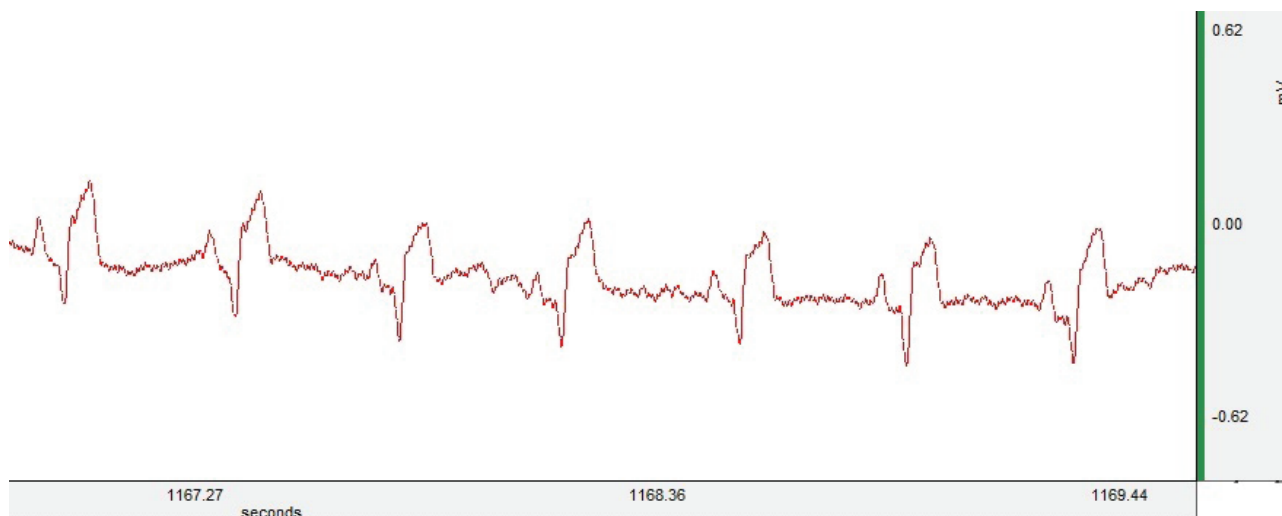


Figure 1: The electrocardiogram from a Buteo Buteo (10 mins after ketamine)

Table 1. The assessment of sedation-anesthesia quality for buzzards (*buteo buteo*) medetomidine - ketamine (MK) or detomidine-ketamine (DK) combination and alterations following atipamezole (AT) administration

Buzards No	Baseline Value		M/D 10 min		K 5 min		K 10 min		K 15 min		K 20 min		K 30 min		K 45 min		K 60 min		AT 5 min		AT 10 min		AT 15 min		
	MK	DK	MK	DK	MK	DK	MK	DK	MK	DK	MK	DK	MK	DK	MK	DK	MK	DK	MK	DK	MK	DK	MK	DK	
1	0	0	1	1	2	2	4	3	4	3	4	4	4	4	4	4	4	4	4	4	3	3	3	2	
2	0	0	1	1	3	3	4	4	4	4	4	4	4	4	4	4	4	4	3	4	3	4	2	3	2
3	0	0	1	1	2	2	4	4	4	4	4	4	4	4	4	4	4	4	4	4	3	3	2	2	
4	0	0	1	1	2	2	4	4	4	4	4	4	4	4	4	4	4	4	4	4	3	3	3	2	
5	0	0	1	1	2	2	4	3	3	3	3	3	3	3	3	4	3	4	3	3	2	2	2	2	
6	0	0	1	1	2	2	4	4	4	4	4	4	4	3	3	3	3	3	3	3	2	2	2	2	
7	0	0	1	1	2	2	4	4	4	4	4	4	4	4	4	4	4	4	4	4	4	3	3	2	

0; baseline values, 10 minutes after medetomidine (M) and 5, 10, 15, 20, 30, 45, 60 minutes after ketamine (K), and 5, 10, 15 minutes after injection of Atipamezol (AT).

1; Able to stand up, partly responsive to environmental objects and walk voluntarily when stimulated. 2; Unable to stand up and tend to stay in lateral recumbency. Partly has needle prick stimuli and hardly responsive to environmental stimulation. 3; Inability to restore body posture, hardly has foot withdrawal response against needle prick. Good muscle relaxation. 4; Deep general anesthesia, no reflexes available including pedal, palpebral and corneal. Complete closure of third eyelids. Satisfactory muscle relaxation. No response to any pain reflexes. MK: Medetomidine-Ketamine DK: Detomidine-Ketamine

Table 2. Mean ± standart deviation of heart rates, respiratory rates, and cloacal temperatures in buzzards (*Buteo buteo*) after medetomidine - ketamine (MK) or detomidine -ketamine (DK) combination

N=7 (for each group)	Medetomidine-Ketamine			Detomidine-Ketamine		
	Cloacal Temperature $\bar{X} \pm S_x$	Respiratory Rate $\bar{X} \pm S_x$	Heart Rate $\bar{X} \pm S_x$	Cloacal Temperature °C $\bar{X} \pm S_x$	Respiratory Rate $\bar{X} \pm S_x$	Heart Rate $\bar{X} \pm S_x$
Baseline Value	41.9 ± 0.2	42. ± 2	272±6	41.9 ± 0.1	42±2	314±13
(Me) detomidine 5 min	41.± 0.2	18.± 3***	118±5***	41.5 ± 0.2	26±2***	170±24***
(Me) detomidine 10 min	40.3 ± 0.3**	26.± 4***	122±2.***	41.2 ± 0.2*	22±2***	122±8***
Ketamine 5 min	40.2 ± 0.2**	27 ± 4***	111±3***	40.7 ± 0.4*	22±2***	117 ±5***
Ketamine 10 min	39.5 ± 0.5**	28 ± 4***	107±4***	40.4 ± 0.4*	24±3***	112 ±3***
Ketamine 15 min	38.8 ± 0.5**	29± 4***	102±4***	39.8± 0.4*	22±3***	109.±4***
Ketamine 30 min	38.7 ± 0.5***	31 ± 4***	100 ±5***	39.6 ± 0.4*	24±4***	105±3***
Ketamine 45 min	38.2 ± 0.4***	34 ± 6***	96±4***	38.8 ± 0.5***	25±4***	102 ±2***
Ketamine 60 min	37.0 ± 0.5***	33 ± 5***	91±3***	38.1 ± 0.4***	25±4***	105 ±4***
AT 5 min	36.1 ± 0.5***	24± 3***	89±4***	38.1 ± 0.5***	30 ±4***	100 ±4***
AT 10 min	35.9 ± 0.4***	25 ± 3***	87±4***	37.6 ± 0.5***	36±5***	98±4***
AT 15 min	36.2 ± 0.5***	27 ± 4***	92±6***	37.6 ± 0.4***	37± 5***	122 ±7***

* $P < 0.05$ ** $P < 0.01$ *** $P < 0.001$ Significant difference between baseline values with anesthesia

Baseline values: before anaesthesia

5, 10 minutes after medetomidine and 5, 10, 15, 30, 45, 60 minutes after ketamine (K), and 5, 10, 15 minutes after injection of Atipamezol (AT).

Table 3. Mean \pm standard deviation of serum electrolyte and blood gases in the buzzards (*Buteo buteo*) medetomidine - ketamine (MK) and detomidine -ketamine (DK) combination

Variable	Groups	Baseline Value	During Anesthesia
pH	MK	7,33 \pm 0,43	7,316 \pm 0,024*
	DK	7,40 \pm 0,030	7,24 \pm 0,045*
pCO ₂ (mmHg)	MK	42,32 \pm 3,04	50,00 \pm 2,04*
	DK	38,15 \pm 1,99	61,18 \pm 6,22*
pO ₂ (mmHg)	MK	88,98 \pm 26,00	56,15 \pm 3,59*
	DK	73,30 \pm 9,57	56,85 \pm 5,38*
HCT(%)	MK	32,47 \pm 2,58	34,04 \pm 3,43
	DK	25,39 \pm 2,51	26,44 \pm 2,27
Na ⁺ (mmol/L)	MK	135,88 \pm 4,30	142,00 \pm 4,99
	DK	145,28 \pm 2,17	137,57 \pm 2,52
K ⁺ (mmol/L)	MK	7,17 \pm 0,67	5,99 \pm 0,78
	DK	5,08 \pm 0,65	4,58 \pm 0,30
Ca ⁺⁺ (mmol/L)	MK	0,61 \pm 0,08	0,73 \pm 0,15
	DK	0,75 \pm 0,05	0,63 \pm 0,065
HCO ₃ ⁻ (mmol/L)	MK	21,67 \pm 1,64	24,92 \pm 1,44*
	DK	23,34 \pm 1,95	24,81 \pm 1,09*
O ₂ Sat (%)	MK	87,82 \pm 3,53	79,28 \pm 4,08*
	DK	84,94 \pm 4,20	73,67 \pm 3,78*

* Significant difference between baseline values with anesthesia ($P < 0.05$) ($P < 0.05$)

Baseline value: Anesthesia before

During Anesthesia: 20 min after ketamine

Table 4. Effects of the amplitudes and the durations of waves the groups Medetomidine-Ketamine, Detomidine -Ketamine

Values	N:7	Baseline values $\bar{X} \pm S$	Med 10 min $\bar{X} \pm S$	Ket 10 min $\bar{X} \pm S$	Ket 15 min $\bar{X} \pm S$	Ket 30 min $\bar{X} \pm S$	Ket 45 min $\bar{X} \pm S$	Ket 60 min $\bar{X} \pm S$	REV 5 min $\bar{X} \pm S$	REV 15 min $\bar{X} \pm S$
P-wave duration (s)	MK	0.033 \pm 0.004	0.036 \pm 0.001	0.041 \pm 0.003	0.038 \pm 0.001	0.037 \pm 0.001	0.039 \pm 0.001	0.042 \pm 0.003	0.041 \pm 0.003	0.042 \pm 0.002
	DK	0.032 \pm 0.004	0.042 \pm 0.005	0.034 \pm 0.001	0.038 \pm 0.004	0.037 \pm 0.001	0.036 \pm 0.004	0.033 \pm 0.002	0.041 \pm 0.005	0.042 \pm 0.003
P-wave amp. (mV)	MK	0.009 \pm 0.005	0.006 \pm 0.003	0.003 \pm 0.001	0.003 \pm 0.001	0.004 \pm 0.001	0.004 \pm 0.001	0.004 \pm 0.001	0.003 \pm 0.001	0.005 \pm 0.004
	DK	0.014 \pm 0.003	0.020 \pm 0.003	0.021 \pm 0.006	0.019 \pm 0.006	0.014 \pm 0.001	0.014 \pm 0.001	0.016 \pm 0.002	0.037 \pm 0.020	0.013 \pm 0.003
QRS complex duration(s)	MK	0.052 \pm 0.003	0.038 \pm 0.001	0.049 \pm 0.002	0.044 \pm 0.003	0.050 \pm 0.003	0.043 \pm 0.003	0.043 \pm 0.003	0.050 \pm 0.003	0.045 \pm 0.004
	DK	0.054 \pm 0.002	0.059 \pm 0.004	0.053 \pm 0.002	0.045 \pm 0.002	0.058 \pm 0.002	0.054 \pm 0.002	0.124 \pm 0.072	0.126 \pm 0.072	0.047 \pm 0.002
QRS complex amp.(mV)	MK	0.123 \pm 0.005	0.121 \pm 0.004	0.119 \pm 0.002	0.114 \pm 0.001	0.118 \pm 0.003	0.115 \pm 0.002	0.117 \pm 0.003	0.113 \pm 0.002	0.115 \pm 0.003
	DK	0.207 \pm 0.047	0.160 \pm 0.007	0.132 \pm 0.015	0.122 \pm 0.016	0.102 \pm 0.017	0.124 \pm 0.23	0.108 \pm 0.027	0.080 \pm 0.013	0.129 \pm 0.007
T-wave duration(s)	MK	0.038 \pm 0.004	0.052 \pm 0.004	0.051 \pm 0.003***	0.052 \pm 0.004***	0.56 \pm 0.004***	0.060 \pm 0.004***	0.061 \pm 0.003***	0.059 \pm 0.003***	0.064 \pm 0.003***
	DK	0.034 \pm 0.003	0.044 \pm 0.002	0.052 \pm 0.002***	0.053 \pm 0.003***	0.60 \pm 0.003***	0.053 \pm 0.00***	0.062 \pm 0.001***	0.072 \pm 0.005***	0.067 \pm 0.003***
T-wave amp. (mV)	MK	0.005 \pm 0.001	0.004 \pm 0.001	0.002 \pm 0.001	0.007 \pm 0.002	0.005 \pm 0.001	0.004 \pm 0.001	0.006 \pm 0.001	0.006 \pm 0.001	0.006 \pm 0.001
	DK	0.016 \pm 0.003	0.022 \pm 0.004	0.021 \pm 0.005	0.019 \pm 0.003	0.017 \pm 0.003	0.018 \pm 0.003	0.023 \pm 0.006	0.017 \pm 0.004	0.018 \pm 0.001
PR (sn)	MK	0.048 \pm 0.003	0.072 \pm 0.003	0.077 \pm 0.005	0.077 \pm 0.003	0.74 \pm 0.002	0.079 \pm 0.003	0.079 \pm 0.003	0.084 \pm 0.005	0.084 \pm 0.004
	DK	0.053 \pm 0.005	0.074 \pm 0.005	0.079 \pm 0.002	0.084 \pm 0.002	0.84 \pm 0.003	0.085 \pm 0.003	0.084 \pm 0.003	0.086 \pm 0.003	0.083 \pm 0.004
QT interval (s)	MK	0.094 \pm 0.005	0.079 \pm 0.003	0.010 \pm 0.004	0.118 \pm 0.004**	0.122 \pm 0.006**	0.131 \pm 0.004**	0.131 \pm 0.007**	0.135 \pm 0.007**	0.138 \pm 0.007**
	DK	0.094 \pm 0.003	0.109 \pm 0.002	0.117 \pm 0.004	0.112 \pm 0.005**	0.117 \pm 0.004**	0.118 \pm 0.005**	0.126 \pm 0.005**	0.130 \pm 0.005**	0.124 \pm 0.004**

Significant difference between time points at anesthesia protocol ($P < 0.01$).*Significant difference between time points at anesthesia protocol ($P < 0.001$).

reflex, toe pinch reflex, feather plucking reflex, palpebral reflex and table knock reflex) 10 minutes after ketamine administration in all study groups (Tablo 1). The eyes of all the buzzards during anesthesia were closed. Two cases in the MK group showed irregular respiration (cases 1 and 6). Three animals in the MK group showed defecation during anesthesia and one had muscular contractions. All animals in the

DK group showed regular respiration and exhibited no complications during anesthesia. Hypoxia and hypothermia were observed during anesthesia in both groups.

Changes in HCT, K⁺, Na⁺, and Ca⁺⁺ levels in both the MK and DK groups were not statistically significant when compared to baseline values, and the dif-

ference in these changes between the two groups was not statistically significant ($p > 0.05$).

DISCUSSION

Injectable anesthesia is widely used for sedation and anesthesia in birds. Injectable anesthesia is cheap and easy to administer, and is thus widely used. Ketamine is probably the most commonly used drug for general anesthesia in birds (Patrik 2005). However, it should not be used as the sole agent for anesthetizing birds because it is associated with poor muscle relaxation, muscle tremors, myotonic contractions, opisthotonus, and rough recoveries (Christensen et al., 1987; Mahmut et al., 2014). Therefore, it is usually necessary to administer an α_2 agonist such as xylazine, medetomidine or detomidine (Mohammad et al., 1993; Durrani et al., 2005; Durrani et al., 2014; Kamiloglu et al., 2014). Some researchers (Mohammad et al., 1993; Sandmeier (2000) reported that a detomidine-ketamine anesthesia combination can provide long and deep anesthesia for birds that require painful procedures. In our study we used 0.1 mg/kg of medetomidine or 0.3 mg/kg of detomidine, with 10 mg kg⁻¹ of ketamine. The sedative effect started approximately 4.1 ± 2.28 minutes after medetomidine administration and 3.96 ± 1.31 minutes after detomidine administration. All birds of both groups showed deep analgesia, good muscle relaxation, no reflexes, and closed eyes during the whole duration of the anesthesia. Some studies reported side effects such as hypothermia, and partial bradycardia with AV block associated with the dose of the α_2 agonist and ketamine combination. No complications were observed in either group bradycardia during anesthesia in our study.

It is known that α_2 agonists have depressant and arrhythmogenic effects on the cardiovascular system. First, hypertension develops. This is followed by bradycardia and reduced cardiac contractility. Then there is a reduction in cardiac output (Uzun et al., 2006). Some studies (Uzun et al., 2003; Durrani et al., 2008) have reported that the combined use of α_2 agonists and ketamine leads to a significant decrease in heart rate in birds. In the present study, decrease in heart rate was statistically significant ($p < 0.001$) in both groups, but there was no difference between the groups. This property of α_2 agonist agents has been reported to result from depression of sympathetic activity coupled with an increase in parasympathetic effects, decreasing heart rate (Güzel 2003).

ECG is a widely used method to evaluate the elec-

trical activity of the heart. It is also an objective examination method used in the evaluation of the cardiopulmonary system during anesthesia (Kaya and Soylu 2013; Wiemeyer et al., 2013). In our study we performed an ECG examination before and during anesthesia. The evaluations before anesthesia did not show any abnormal findings and just showed normal sinus rhythms. In both groups the ECG evaluations revealed prolongation in the PR range and in the QT range on the T-wave. If there is a prolongation in the QRS complex or T-wave duration or both, a prolongation can also be observed in the QT range. The duration of QT interval varies inversely to heart rate. While a quick heart rate leads to a shorter QT duration, a slow heart rate leads to a longer QT duration. In our study, the decrease in the number of heart rate can explain by the prolongation of the PR and QT ranges and the T wave.

Avian cloacal temperature ranges from 40 to 44.4°C (Harrison and Ritchie 1994; Chan et al., 2013). Some authors (Uzun et al., 2003; Qazi et al., 2015) have reported a decrease in cloacal temperature following administration of the α_2 agonist and ketamine combination in birds. This was caused by depression of the thermoregulatory center, reduction of basal metabolism, reduction of heat production, and an increase of the loss of heat through the respiratory tract. Therefore, during avian anesthesia, supplementary heat is recommended to counter the decrease in cloacal temperature over time (Chan et al., 2013). In the present study, a decrease in cloacal temperature was statistically significant ($p < 0.05$) in both groups, but there was no difference between the groups. A decrease in cloacal temperature was reported in pigeons (Atalan et al., 2002, Uzun et al., 2003, Lumeij et al., 2003) and buzzards (Kilic and Paşa 2009) during medetomidine-ketamine anesthesia and in quails (Durrani et al., 2008; Yayla et al., 2015; Akgül et al. 2017;) and pigeons during detomidine-ketamine anesthesia.

Baseline f_R values in buzzards of 30 breaths min⁻¹ have been reported in previous studies (Harrison et al., 1994; Chan et al., 2013). The f_R of the anesthetized buzzards in the present study was significantly decreased from baseline values, and during anesthesia the respiration was deep but regular. There was no difference between the groups ($p > 0.05$). Similar data were reported in birds anesthetized with either medetomidine-ketamine (Mohammad et al., 1993; Uzun et al., 2003) or detomidine-ketamine combinations (Machin and Caulkett 1998). α_2 Agonists induced a

dose-dependent decrease in f_R (Machin & Caulkett 1998). Respiratory rate may be decreased due to direct depression of respiratory centers. In addition, increases in pCO_2 and HCO_3 and decreases pH and pO_2 were associated with respiratory depression caused by both MK and DK during anesthesia (20 min after on ketamine). However, there was no statistically significant difference in pCO_2 , HCO_3 , RR, or pH values between the two groups. Previous studies have reported respiratory acidosis in buzzards during MK anesthesia. Our findings for buzzards were similar to those of previous reports. Birds should be supplemental oxygen during anesthetized with medetomidine-ketamine or detomidine- ketamine.

In recent years, α_2 adrenoceptor antagonists (atipamezol, yohimbine, and talozoline) have been widely used in veterinary practice. Atipamezole is a strong and selective α_2 receptor blocking agent that increases the release of noradrenaline in both the central nervous system and the peripheral nervous system. α_2 adrenoceptor antagonists are used to eliminate or inhibit the effects of α_2 adrenoceptor agonists such as such as detomidine and medetomidine (Jager et al., 1998). Birds have very high metabolic rates. A high metabolism provides a faster utilization of food. Six to eight hours of fasting before anesthesia can cause fatal hypoglycemia and ketosis. Therefore, the ef-

fect of α_2 adrenoceptor agonists in birds should be reversed after completion of anesthesia. Total recovery after AT injection was observed within 10 min in dogs (Pypendop et al., 1996) and 15 min in lambs (Ko and McGrath, 1995). In our study, recovery was very quick after injection with atipamezole.

In conclusion, medetomidine-ketamine and detomidine-ketamine combinations administered in Buteos can potentially produce a sufficient degree of anesthesia, analgesia and muscle relaxation. Hypoxemia, hypercapnia, primary respiratory acidosis and hypothermia were present in both groups. Therefore, oxygen support and heat are required during the anesthesia protocol of the birds.

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CONFLICT OF INTEREST

None declared.

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