Vol. 22, No. 1, Spring, 2026, 28-36 *Original Article*
DOI: 10.22055/ivj.2025.522754.2794
Journal Homepage: http://www.ivj.ir/

Evaluation of sedation status, clinical and cardiorespiratory effects of clonidine hydrochloride on dogs

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Received: 14.05.2025 Accepted: 20.10.2025

Abstract

Clonidine is an alpha-2 agonist which has been widely known as an antihypertensive agent in human medicine. Due to the importance of the alpha-2 agonists as sedatives and analgesics; it seems that there is a lack of information about clonidine in the veterinary science. The aim of the present study was to evaluate sedation status, clinical and cardiorespiratory effects of clonidine alone, and compare it with xylazine and acepromazine in dogs. To do this, five adult mixed-breed male dogs were used. Health status was assessed by means of physical examination, a complete blood count and serum biochemical analysis. All dogs respectively received intramuscular acepromazine (0.2 mg/kg), xylazine (1 mg/kg), and clonidine (0.03 mg/kg) within a 10-day interval. The degree of sedation, heart rate, respiratory rate, and rectal temperature was recorded before injection until 90 min post-injection. Blood samples were also collected before injection and 1, 3, 6 and 24 hours after that. As a result, there were no significant differences between the sedation status of the three groups at any time. Cortisol levels, except for a significant increase between acepromazine and xylazine 3 hours post-injection, were not significantly different. The reduction of heart rate and respiratory rate was more significant in the xylazine and clonidine group than in the acepromazine group. According to the results, it seems that the sedative effects of clonidine were acceptable compared with the other groups in the study. With further studies, administration of clonidine (0.03 mg/kg) as a sedative and preanesthetic drug could be recommended intramuscularly.

Key words: Alpha-2 agonist, Clonidine, Dog, Sedation

Introduction

Delivery of drugs to achieve sedation is an important aspect of veterinary practice. Sedative drugs are administered for diagnostic procedures, preanesthetic sedation, postsurgical sedation, and many other reasons (Carter et al, 2013). Alpha-2 agonists are the only single class of anesthetic drugs that induce reliable, dose-dependent sedation, analgesia, and muscle relaxation in dogs and cats. Used at low doses, as adjuncts to injectable and inhalational anesthetics, selective alpha-2 agonists dramatically reduce the amount of anesthetic drug required to induce and maintain anesthesia. They can also be readily reversed by administration of selective antagonists. The most commonly used alpha-2 agonists in veterinary medicine are xylazine, detomidine, dexmedetomidine and romifidine. The activity of these drugs depends on their relative specificity for the A-2 and A-1

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receptors. Of the agents listed above xylazine has the least and dexmedetomidine has the highest specificity for A-2 receptors (Lemke, 2004; Carter, 2014). Clonidine is an alpha-2 agonist typically used in human medicine as an anti-hypertensive agent. Like the other drugs of this class, it also has sedation and analgesic effects (Keranen et al, 1978; Lehner, 2022). It has also been mentioned for possible use in treating dogs for fear-based behavior problems (Landsberg et al, 2003; Ogata and Dodman, 2011). The aim of the current study was to evaluate the effects of clonidine alone and in comparison, with other sedative agents in dogs. The hypothesis was that it could be used for restraining and simple procedures.

Materials and methods

Five adult mixed-breed male dogs weighing 20.3 ± 5.0 kg (mean ± standard deviation) were used for this study. Health status was assessed by means of physical examination, complete blood count and serum biochemical analysis. Animals were housed separately for one week before the experiment. Food but not water was withheld for 12 hours prior to the experiment.

All the dogs in three groups, respectively, received acepromazine 1% (Neurotranq, Alfasan, Netherlands, 0.2 mg/kg), xylazine 2% (Xylazine, Alfasan, Netherlands, 1 mg/kg), and clonidine (12 mg clonidine HCL powder mixed in 20 ml of 10% ethanol solution which were prepared in isotonic saline solution and autoclaved to make a 6% injectable clonidine solution, Clonidine HCL, Toliddaru, Unichem India, 0.03 mg/kg) intramuscularly within 10-day intervals to washout between treatments. To inject the same level of drugs in the hamstring group muscles, acepromazine was diluted 0.05 ml/kg in sterile water (Dirikolu et al, 2006; Monteiro et al, 2008; Cruz et al, 2013).

Physiological variables (heart rate, respiratory rate, and rectal temperature) were recorded at 30 min before, and 0, 5, 10, 15, 20, 25, 30, 45, 60, 75 and 90 min after injection of each agent. Times of sternal recumbency, lateral rec., back to sternal rec., and standing were also recorded.

2 ml of blood was collected from the cephalic vein for determining the hematological (WBCs count, RBCs count, hemoglobin level, HCT, MCV, MCH, MCHC, RDW, Plt) and biochemical parameters (cortisol level) at 0, 1, 3, 6 and 24 hours following the early described procedure. Hematological variables were analyzed with an automatic cell counter (BC-2800 Vet, Mindray, China), and the cortisol level was determined through a cortisol ELISA kit (Monobind, Germany).

Sedation scoring was performed by a single observer. Each dog was housed separately at least 12 hours before initiation of the study. They were also acclimatized to the observer for approximately 30 min before the scoring began. During data collection, the dogs were able to roam freely in the room. Sedation was scored using a simple descriptive scale (SDS) and a numeric rating scale (NRS) in the present study. The SDS included four scores from 0 (no sedation), 1 (mild sedation: quieter but still bright and active), 2 (moderate sedation: quiet, reluctant to move, possibly ataxic but still able to walk) and 3 (Profound sedation: unable to walk). NRS was based on the sum of the scores of vocalization (0.-1, -2, -3), posture (3, 2, 1, 0, -1), appearance (3, 2, 1, 0, -1, -2, -3), interactive behaviors (3, 2, 1, 0, -1, -2, -3), restrainability (2, 1, 0, -1, -2) and noise response (3, 2, 1, 0) (Table 1). (Mair et al., 2009; Zapata et al., 2013; Monteiro et al., 2016). Sedation scores were obtained before time 0, at 5, 10, 15, 20, 25, 30, 45, 60, 75 and 90 min post-injection.

The Data were analyzed through SPSS software. Comparison of the parametric data of three groups with each other was based on the Tukey post Hoc test and one-way ANOVA. For comparison of each time per group Repeated measures ANOVA test was used. Sedation scoring analysis as nonparametric data was also accomplished through Kendall and Kruskal-Wallis test. Significance was set at $P<0{\cdot}05$.

Table 1: Sedation scoring system (Zapata et al, 2013)

Observation	Score	Criteria
Vocalization	0	Quiet
	−1	Whining softly but quiets with soothing touch
	−2	Whining continuously
	−3	Barking continuously
Posture	3	Lateral recumbency
	2	Sterna recumbency
	1	Sitting or ataxic
	0	Standing
	−1	Continuous movement
Appearance	3	Eyes sunken, glazed, unfocused, ventromedial rotation
	2	Eyes glazed but follow movement
	1	Nictitating membrane protruded; normal visual Responses
	0	Normal appearance
	−1	Pupils dilated; abnormal facial expression
Interactive Behaviors	3	Recumbent; no response to voice or touch
	2	Recumbent; lifts head in response to voice or touch
	1	Recumbent but rises in response to voice or touch
	0	Standing or sitting up; normal response to voice or touch
	−1	Moves away from voice or touch; "jumpy"
	−2	Growls/hisses when approached or touched
	−3	Bites/swats when approached
Restrainability	2	Lies on floor with minimal restraint needed
	1	Lies on floor with light restraint of head/neck
	0	Sits up on floor; attempts to jump despite restraint
	−1	Struggles against restraint continuously
	−2	Cannot be restrained for less than 20 seconds
Noise Response	3	No response to a hand clap near the head
	2	Minimal response to a hand clap near head
	1	Slow/moderate response to hand clap near head
	0	Brisk response to a hand clap; raises head, eyes open

Results

Physiological variables

Heart rate (HR) of the dogs in the acepromazine (A) group was significantly higher than in the xylazine (X) and clonidine (C) groups, respectively, at time 0 until 30 min after administration and all times mentioned previously ($P<0·05$). Considering differences in each group at each time separately, HR in the xylazine group was significantly decreased in contrast with baseline. This result was also seen in the clonidine group ($P<0·05$) (Table 2). The least HR was in the xylazine group at 20 min after administration (28.60 ± 1.17) (p in comparison with baseline = 0.004) (Table 2).

Respiratory rate (RR) of the dogs in the acepromazine group was significantly higher than the xylazine group at 10 to 90 min after administration and also higher than clonidine group at 5 to 25 min and 60 to 90 min after administration ($p<0·05$) The least RR was in xylazine group at 20 min (8.20 ± 0.8) after administration (further information at table 2).

Rectal temperature (RT) of the dogs in the clonidine group was significantly higher than that of the acepromazine and xylazine groups at 75 min after administration (p respectively 0.04, 0.031). RT of the clonidine group was also significantly higher than the xylazine group at 90 min after injection (p=0.041) (Table 2).

Hematological and biochemical variables

RBCs count, hemoglobin level, and PCV of the clonidine group were significantly higher than the acepromazine group at 6 hours (p respectively 0.049, 0.022, 0.014) (Table 3).

Cortisol level of the acepromazine group was significantly higher than the xylazine group at 3 hours (p=0.037). There were no other significant changes in the serum cortisol level during the present study (Table 3).

Table 2: Mean ± SE of HR, RR, RT in 5 dogs receiving acepromazine (0.2 mg/kg), xylazine (1 mg/kg), clonidine (0.03 mg/kg)

Parameters	HR			RR			RT		
Times/ Groups	A	X	C	A	X	C	A	X	C
Baseline	104±8.2 (XC) (k)	70.20±7.81 (A)(bcdefghijk)	62.40±3.7 (A)(bcdefghijk)	40.00±7.3 efgh	46.40±11.1 cdefghijk	32.40±6.7 cdefghijk	38.84±0.9 j	38.50±0.2 cdf	38.34±0.2 bcdefghijk
5 min	118.40±8.6 (XC) (k)	34.20±3.0 (A) (ae)	40.80±3.3 (A)(ak)	27.60±3.4 C	20.00±2.2 cdefghij	16.80±2.1 (A)(f)	38.96±0.1 hijk	38.68±0.2 f	38.78±0.2 a
10 min	104.40±8.3 (XC) (k)	35.40±4.2 (A) (a)	36.40±2.0 (A)(a)	28.00±3.6 XC	13.20±2.2 (A) (abh)	15.00±1.3 (A)(adk)	38.96±0.7 ijk	38.86±0.2 ai	38.84±0.2 agijk
15 min	100.40±7.5 XC	31.40±2.5 (A) (a)	36.20±2.1 (A)(a)	24.80±2.9 XC	9.20±1.0 (A) (ab)	13.40±1.5 (A)(ac)	38.92±0.1 (ijk)	38.84±0.2 ajk	38.92±0.1 a
20 min	92.80±14	28.60±1.2 (A)(abfhijk)	35.60±0.4 (A)(a)	26.40±3.4 (XC) (a)	8.20±0.8 (A) (ab)	12.60±1.5 (A)(ag)	38.92±0.9 ijk	38.80±0.2 fik	39.08±0.2 aij
25 min	98±15.19 (XC) (k)	31.60±1.4 (A)(ae)	35.00±2.4 (A)(a)	22.80±3.4 (XC) (a)	8.60±0.7 (A) (ab)	12.00±1.1 (A)(ab)	38.94±0.1 ijkh	38.94±0.2 abeijk	39.02±0.2 abij
30 min	97.20±15.7 (XC) (k)	32.60±1.6 (A)(a)	35.40±1.6 (A)(a)	23.60±3.3 (X) (a)	9.60±1.3 (A) (ab)	15.40±2.1 e	38.88±0.1 ijk	38.82±0.2 ijk	39.16±0.2 abc
45 min	87.60±14.6 (C)	32.00±1.8 (aej)	36.80±1.6 (A)(a)	25.20±6.5 (X) (a)	8.40±1.1 (A)(abc)	14.60±1.7 a	38.70±0.1 bf	38.68±0.3	39.18±0.2 ab
60 min	87.60±14.2 (C)	35.40±1.9 (ae)	36.60±1.6 (A)(a)	30.80±7.0 XC	11.40±1.2 (A) (ab)	13.8±1.2 (A)(a)	38.72±0.1 bcdefgk	38.60±0.3 cdefg	39.24±0.2 abcef
75 min	81.60±16.9 (C)	38.40±3.5 (aeh)	36.60±0.9 (A)(a)	29.60±7.0 XC	11.80±2.1 (A)(ab)	11.60±1.2 (A)(a)	38.60±0.8 (C)(abcdefg)	38.56±0.24 (C)(fg)	39.32±0.2 (AX)(abcef
90 min	78.00±11.8 (C)(abcdefg)	37.80±2.9 (ae)	33.00±2.7 (A)(ab)	28.80±4.2 XC	12.40±2.2 (A)(a)	11.60±1.2 (A)(ac)	38.60±0.15 Bcdefgi	38.44±0.2 (C)(defg)	39.20±0.2 (X)(abc)

Small letters (a to k) indicating significant changes at each group separately during times.
Capital letters (A, X, C) indicating significant changes of groups in comparison at each time.

Table 3: Mean ± SE of hematological and chemical (cortisol) parameters in 5 dogs receiving acepromazine (0.2 mg/kg), xylazine (1 mg/kg), clonidine (0.03 mg/kg)

Parameter	Groups/times	Baseline (a)	1 hr (b)	3 hr (c)	6 hr (d)	24 hr (e)
WBC	A	13.00±1.74	12.02±1.79	14.48±2.38	13.08±1.62	12.00±2.06
	X	10.20±0.68	10.40±0.40	10.04±0.68	9.68±0.32	10.38±1.39
	C	8.96±0.44	11.12±0.71	9.84±0.54	9.94±0.37	10.36±0.31
RBC	A	5.82±0.28 bd	4.79±0.16 ae	4.90±0.24	4.29±0.08 (C)(ae)	5.54±0.15 bd
	X	5.39±0.21 bd	5.06±0.19 a	4.92±0.25	4.85±0.22 ae	5.27±0.27 d
	C	5.57±0.35	5.24±0.14	5.09±0.09 e	5.04±0.25 A	5.43±0.18 c
PCV	A	39.04±2.62 d	32.08±1.49	32.78±2.25	27.98±1.01 (C)(ae)	36.58±1.71 d
	X	35.96±1.44 bd	34.18±1.30 a	32.72±1.52	31.88±1.23 a	35.18±2.14
	C	37.80±1.79	34.50±0.87	33.74±0.95	33.94±1.45 A	36.16±1.59
Cortisol	A	4.03±0.94	4.60±1.44	3.34±0.79 X	2.41±0.52	1.85±0.58
	X	1.50±0.36 d	1.66±0.38	1.24±0.33 (A)(d)	2.05±0.21 ace	1.40±0.13 d
	C	2.32±0.63	2.27±0.67	1.42±0.31 e	2.02±0.42	2.60±0.26 c

Small letters (a to e) indicating significant changes at each group separately during times.
Capital letters (A, X, C) indicating significant changes of groups in comparison at each time.

Sedation score

According to both sedation score scales, there was no significant difference between the three groups. The SDS score in each group separately showed that the score of the acepromazine group was significantly higher than the baseline, except for 5 min after administration (P<0.05). Sedation score of the xylazine group was significantly higher than the baseline at all times (P<0.05). The score of the clonidine group was significantly higher than the baseline except for 25 min after administration (P<0.05). The highest sedation score was in the acepromazine group at 45 min (median sedation score: 3) and in the xylazine group at 30-45 min (median sedation score: 3) after administration. The NRS in each study group separately indicated that the acepromazine group at 10-90 min, the xylazine group, and the clonidine group at 5-90 min after administration were significantly higher than the baseline (P<0.05). The highest sedation score was in the acepromazine group (9) and the xylazine group (9) at 45 min (p 0.000, 0.000) (Table 4).

Table 4: Median (min-max) of sedation score in 5 dogs receiving acepromazine (0.2 mg/kg), xylazine (1 mg/kg), clonidine (0.03 mg/kg)

Scores	SDS			NRS		
Times/Groups	A	X	C	A	X	C
Baseline (a)	0 (0-0) cdefghijk	0 (0-0) bcdefghijk	0 (0-0) bcdeghijk	0 (0-1) cdefghijk	-1 (-2-0) bcdefghijk	-1 (-3-0) bcdefghijk
5 min (b)	1 (1-1)	2 (1-2) agh	1 (1-2) aeh	1 (0-5) efghijk	3 (0-5) acdefghij	3 (0-5) adhijk
10 min (c)	1 (1-2) afgh	2 (1-2) agh	2 (1-2) a	3 (2-4) ahijk	5 (2-7) abeghi	2 (1-8) a
15 min (d)	2 (1-2) a	2 (1-3) a	2 (1-2) a	3 (2-9) ahi	7 (3-7) abefgh	6 (1-7) ab
20 min (e)	2 (1-2) a	2 (1-3) a	2 (2-3) ab	5 (2-9) ab	8 (4-9) abcd	4 (1-7) a
25 min (f)	2 (1-3) ac	2 (1-3) a	2 (2-2)	5 (2-9) abhi	8 (4-9) abd	4 (1-9) a
30 min (g)	2 (1-3) ac	3 (1-3) abc	2 (2-3) a	5 (2-10) ab	8 (4-10) abcd	7 (2-9) a
45 min (h)	3 (1-3) a	3 (2-3) abc	2 (2-3) ab	9 (6-10) abcdf	9 (6-9) abcd	7 (6-8) ab
60 min (i)	2 (1-3) a	2 (2-3) a	2 (2-3) a	8 (6-10) abcdf	8 (6-9) abck	7 (4-8) ab
75 min (j)	2 (1-3) a	2 (2-3) a	2 (2-2) a	8 (3-10) abc	7 (5-8) abk	6 (4-7) ab
90 min (k)	2 (1-3) a	2 (1-2) a	2 (1-2) a	7 (2-8) abc	6 (3-7) aij	7 (6-7) ab

Small letters (a to k) indicating significant changes at each group separately during times.
Capital letters (A, X, C) indicating significant changes of groups in comparison at each time.

Due to the comparison of recumbency times, sternal rec., back to sternal rec (mean times of change in position, respectively, 18.40±5.33, 22.60±6.92, 55.20±14.18 min), and standing times of the acepromazine group occurred at a shorter time. Time of lateral rec. was also shorter in the acepromazine group (191.60±43.02 min). The Longest sternal rec., lateral rec., back to sternal rec., and standing times were in the clonidine group (mean times of change in position, respectively, 17.20±5.20 min, 33.20±15.55 min, 68.40±14.08 min, 147.60±28.88 min). Vomiting was also observed in 4 dogs of the clonidine group and 3 dogs of the xylazine group.

Discussion

This study was conducted according to the importance of the alpha-2 agonist family as sedatives and analgesics and the lack of information on clonidine as an alpha-2 agonist in veterinary medicine. Clonidine was studied alone and in comparison with other sedatives in dogs.

The acepromazine group provided a stable HR compared to the other two groups, which may result from greater hypotensive actions and concurrent mild sympathetic attenuation (Vaisanen et al, 2002). Alpha-2 agonists cause bradycardia regardless of their amount. The sympatholytic and parasympathomimetic effects produced by the clonidine and the xylazine group were the actual reason for bradycardia (Klide et al, 1975; Kroin et al, 2003). In a study that was conducted on 6 healthy dogs, 5µg/kg of intramuscular clonidine, as a pre-anesthetic medication associated with tramadol (analgesia), propofol (induction of anesthesia), and isoflurane (maintenance) increased the time domain HRV by blocking sympathetic outflow and increasing parasympathetic tonus, with minor effects on arterial blood pressure (Pascon et al, 2022). Based on another study that was conducted on 6 dogs, intrathecal clonidine (250 µg/h) reduced heart rate by 45.8%, and five of six animals had bradyarrhythmia which was similar to the current study (Kroin et al., 2003). The effects of acepromazine on the HR are variable. A slight increase in response to hypotension might be seen (Turner et al, 1974). In return, it might also decrease (Popvic et al, 1972; Monteiro et al, 2008).

Despite the desirable clinical properties of the alpha-2 agonists, the main limiting factor of their use is the adverse cardiovascular effect (Sinclair, 2003). Similar to HR, the effects of the acepromazine group on RR were significantly less than those of the alpha-2 agonist family ($P<0.05$). The effects of alpha-2 agonists may be due to direct inhibition of sympathetic control by central or peripheral mechanisms (Eisenach, 1991).

Alpha-2 agonist and phenothiazine family effect on respiratory variables depend on the kind, dosage, route of administration and fellow drugs. Acepromazine has a low effect on the lungs; although it might also decrease (Popvic et al, 1972; Turner, 1974; Monteiro et al, 2016). Alpha-2 agonists show a significant decrease in RR without changes in PaO_2, $PaCO_2$ and pH which was similar to the current study (Klide et al, 1975; Monteiro et al, 2008).

Changes of RT were variable in all three groups. The only significant change of RT was at 75 min after administration, where the clonidine group was significantly higher than the xylazine and acepromazine groups ($P<0.05$). But all changes of RT were within the normal range.

Phenothiazines are known for their intervention mechanisms in body temperature and for decreasing it. Vasodilation of peripheral vessels following acepromazine administration participates in resonant loss of body heat (Hall et al, 2001). Studies have also indicated that alpha-2 agonists decrease body temperature in dogs, which might be a result of muscle relaxation or the direct effect of the drug on the body's thermoregulatory systems (Cassu et al, 2014). However, in dogs, only slight reductions in rectal temperature were observed with alpha-2 agonists like medetomidine or romifidine, while no reductions were noted with romifidine sedation (Lemke, 1999; Sinclair, 2003).

Hematological variables (RBC, Hb, PCV) of the clonidine group were significantly higher than those of the acepromazine group at 6 hours after injection. A decrease in these variables in each group, even if it is not significant, is probably due to sequestration of erythrocytes in the spleen. Lower levels of the acepromazine group at 6 hours could also have been due to the longer duration of

action (Biemann et al, 2012; Volpato et al, 2014).

The only significant change in the serum cortisol level between groups at each time was at 3 hours after injection. Cortisol level of the acepromazine group was higher than that of the xylazine group (p=0.037). Various studies have shown that alpha-2 agonists reduce the perioperative levels of stress-related hormones and thus attenuate the stress response of surgery in dogs (Benson et al, 2000; Sinclair, 2003). Although the alpha-2 agonist family can decrease the levels of cortisol, tracking it is not easy (Posner, 2018). In dogs undergoing stressful events, medetomidine as premedication reduced serum cortisol levels, indicating a reduction in stress response (Vaisanen et al, 2002). Our study had limitations in tracking it properly.

Based on both sedation score scales, there was no significant difference between the 3 groups at each time. All groups demonstrated levels of sedation. As it is indicated in the present study, acepromazine shows mild to moderate sedative effects, and its sedative effects do not increase with higher doses. Xylazine and clonidine groups also showed desirable sedative effects. Although clonidine acts more specifically than xylazine and has fewer unfavorable effects, the reason for the difference between the highest sedation score of alpha-2 agonist groups (although not significant) could be due to the amount of drug. Unlike acepromazine, this family's sedative effects are dose-dependent (Hall et al, 2001). Based on a study that has examined the pharmacokinetic and sedative effects of a single dose IV clonidine on four thoroughbred horses, the horses were clinically sedated within minutes of clonidine administration. The maximum sedative effect was observed within 15-20 min after IV injection and persisted for 2.5 to 3 hours after injection (Dirikolu et al, 2006).

In conclusion, due to the results achieved from evaluation of physiological, hematological and sedative parameters, it seems that the sedative effects of clonidine were acceptable compared to the other groups in the study, and with further studies, administration of clonidine (0.03 mg/kg) as a sedative and preanesthetic drug could be recommended intramuscularly.

Acknowledgment

The authors would like to thank members of surgery division, veterinary hospital, Faculty of veterinary medicine, Shahid Chamran University of Ahvaz, Iran.

Conflict of Interest

The authors declare that they have no known competing financial interest or personal relationships that could have appeared to influence the work reported in the present study.

Funding

This study was supported by a grant from the Shahid Chamran University of Ahvaz, Iran.

Ethics approval

This study was approved by Shahid Chamran University of Ahvaz Ethical Committee (Number Ethical: EE/1401.2.24.146228/scu.ac.ir).

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Received: 14.05.2025
Accepted: 20.10.2025

DOI: 10.22055/ivj.2025.522754.2794

ارزیابی وضعیت آرام‌بخشی و اثرات بالینی و قلبی – تنفسی کلونیدین هیدروکلراید در سگ‌ها

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تاریخ دریافت: ۱۴۰۴/۲/۲۴ تاریخ پذیرش: ۱۴۰۴/۷/۲۸

چکیده

کلونیدین یک آگونیست آلفا-۲ است که به عنوان یک عامل ضد فشار خون در پزشکی انسانی به طور گسترده شناخته شده است. با توجه به اهمیت آگونیست‌های آلفا-۲ به عنوان آرام‌بخش و مسکن؛ به نظر می‌رسد که اطلاعات کمی در مورد کلونیدین در دامپزشکی وجود دارد. هدف از مطالعه حاضر ارزیابی وضعیت آرام‌بخشی، اثرات بالینی و قلبی تنفسی کلونیدین به تنهایی و مقایسه آن با زایلازین و آسپرومازین در سگ‌ها بود. پنج سگ نر بالغ نژاد مخلوط مورد استفاده قرار گرفتند. وضعیت سلامت با معاینه فیزیکی، شمارش کامل خون و آنالیز بیوشیمیایی سرم ارزیابی شد. همه سگ‌ها به ترتیب آسپرومازین عضلانی (۰/۲ میلی‌گرم بر کیلوگرم)، زایلازین (۱ میلی‌گرم بر کیلوگرم) و کلونیدین (۰/۰۳ میلی‌گرم بر کیلوگرم) را به فاصله ۱۰ روز دریافت کردند. میزان آرام‌بخشی، ضربان قلب، تعداد تنفس و دمای رکتوم قبل از تزریق تا ۹۰ دقیقه پس از تزریق ثبت شد. همچنین نمونه خون قبل از تزریق و ۱، ۳، ۶ و ۲۴ ساعت پس از آن گرفته شد. هیچ تفاوت معنی‌داری بین وضعیت آرام‌بخشی سه گروه در طول زمان‌ها وجود نداشت. سطح کورتیزول به جز افزایش معنی‌دار بین آسپرومازین و زایلازین ۳ ساعت پس از تزریق تفاوت معنی‌داری نداشت. کاهش ضربان قلب و تعداد تنفس در گروه زایلازین و کلونیدین نسبت به گروه آسپرومازین بیش‌تر بود. با توجه به نتایج، به نظر می‌رسد اثرات آرام‌بخشی کلونیدین در مقایسه با سایر گروه‌های مورد مطالعه قابل قبول بوده است و با مطالعات بیشتر، می‌توان تجویز کلونیدین (۰/۰۳ میلی‌گرم بر کیلوگرم) را به عنوان داروی آرام‌بخش و پیش بیهوشی به صورت عضلانی توصیه کرد.

کلمات کلیدی: آلفا-۲ آگونیست، کلونیدین، سگ، آرام‌بخشی

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