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Original Study

A Comparison Between the Effects of Xylazine-Ketamine and Xylazine-Thiopental Combinations on Cardiac Rhythm in Dogs

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Abstract

Objective- To compare the effects of xylazine-ketamine and xylazine-thiopental on cardiac rhythm in dogs.

Design- Experimental study.

Animals- 15 mixed-bred, adult dogs of either sex.

Procedures- Following premedication with xylazine HCl, either ketamine HCl or sodium thiopental were administered to dogs. Cardiac rhythm was evaluated before as well as 15, 30, 45, and 60 minutes and 3 days after induction of anesthesia.

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Results- Sinus arrest/block and first degree atrioventricular (A-V) block were the most common arrhythmias observed after ketamine administration. During thiopental-induced anesthesia, ventricular premature beats with bigeminy pattern and sinus bradycardia were also recorded.

Conclusion and Clinical Relevance-Concerning the documented effects of the aforementioned drugs on cardiac rhythm, arrhythmias might be induced by the combination of xylazine with both anesthetic drugs.

Key Words: Cardiac arrhythmia, dog, ketamine, thiopental, xylazine

Introduction

Cardiac arrhythmias are among important complications, which may occur during general anesthesia. During anesthesia and surgery, cardiac monitoring by means of electrocardiogram (ECG), particularly in the critically disabled animals, may be inevitable.

Dogs suffering from heart diseases are likely to show cardiac arrhythmia before and/or during anesthesia. In small animals causes of cardiac arrhythmias may be divided into 3 general categories: 1. dysfunction of the autonomic nervous system, 2. cardiac diseases, and 3. non-cardiac causes. Disturbances in the electrolyte and acid-base balance and administration of various drugs (e.g., general anesthetics) are among non-cardiac causes of cardiac arrhythmias[1]. Xylazine is commonly used in combination with ketamine for reduced muscle tonicity. The combination of xylazine-ketamine produces a good general anesthesia and has several advantages such as an easy administration, rapid onset/termination of anesthesia and few apparent clinical complications[2].

In a study performed by Folts *et al.*[3] alterations in systemic and coronary haemodynamic in, animals anesthetized with ketamine were observed. They reported the following hemodynamic effects of ketamine in dogs: increased in sinus blood circulation, decreased vascular resistance, decreased oxygen delivery to the sinus coronary, increased heart rate, increased cardiac output, and decreased pulmonary ventilation. Wright *et al.*[4] showed that application of ketamine and xylazine for induction of anesthesia would lead to ventricular arrhythmia. It is believed that the ketamine-xylazine combination may cause severe cardiovascular disturbances, and it is suggested to avoid their simultaneous application when cardiovascular complications are present. In addition, administration of sodium thiopental in dogs may be followed by arrhythmogenic effects in 40% of cases.

Ability in the correct and on-time diagnosis and therapy of cardiac arrhythmias is of major importance for the anesthetist as well as for survival of the patient[5].To achieve this goal, ECG of the anesthetized animal can be considered as a monitoring procedure. Since ketamine HCl and sodium thiopental are widely used in small animal clinical practice, their effect was studied in dogs, and cardiac rhythm and ECG parameters were investigated.

Materials and Methods

1) Animals

This study was performed using 5 mixed-bred, adult dogs of either sex, weighing 25 kg in average. The animals did not show any disease symptoms as determined by clinical examinations. Moreover, to overcome any parasitic worm infestation, animals were treated by praziquantel (Droncit®; Bayer AG, Luverkusen, Germany) and levamisole (Rouz Darou Labs, Tehran, Iran). After a 2-week adaptation period, the animals were fasted for 24 hours before anesthesia.

2) Experimental design

Five to eight minutes before anesthesia, animals were premedicated with a 2% solution of xylazine HCl (Rompun ; Bayer AG, Luverkusen, Germany). Xylazine was injected intramuscularly, at a dose 0.6 mg/kg b.w., according to Branson and Booth[6]. Thereafter, skin in the anterior side of the forearm bone was shaved

and disinfected in order to gain access to the cephalic vein into which a 5% dextrose solution was continuously infused by means of a gauge 21 intravenous catheter. First, anesthesia was induced by a bolus IV injection of 5.5 mg/kg ketamine HCl 10% (Rotexmedica GmbH). Anesthesia was maintained for 75 minutes by injections of the drug at a dose of 2.75 mg/kg; every 25 minutes. (based on results of pilot study).

After the recovery period, vital signs were controlled and the animals were kept in their cages, receiving food and water for 2 weeks. This time, the animals were anesthetized with of sodium thiopental 2.5% (Nesdonal : Isochemie) at a dose of 25 mg/kg intravenously, according to Paddleford[7].

Six ECGs were recorded on I, II, III, aVR, aVL, and aVF leads, before, and 15 min, 30 min, 45 min, 60 min, and 3 days after the onset of anesthesia. During the ECG recordings, dogs were placed on an appropriate operating table on their right lateral sides, with their legs vertical on their body surfaces. After shaving the necessary areas and application of ECG jelly, alligator-form electrodes were attached to the points on top of the elbow and stifle joints. For recording ECG, a 1 -channel Cardiofax ECG apparatus (Nihon Kohden Corporation, Model 6511, Japan) was used (paper speed = 25 mm/sec; apparatus sensitivity: 1 mV=10 mm). Finally, variables required for evaluation of cardiac rhythm and rates were analyzed, using a creditable reference .

3) Statistics & data analysis

The results are presented as means ± SEM. In order to compare heart rate and PR interval, before and at different time points after anesthesia induction, the acquired data were analyzed using Sigmastat Software (Jandle Scientific). The means were compared by using the repeated-measure ANOVA, and $P < 0.05$ was considered a significant difference. For comparison of the results before and after anesthesia, the Dunnet post-hoc test was applied. Considering the physical body size of the animals in this study, values mentioned for large-breed dogs[8,9] were used as reference for evaluation of the heart rhythm, rate (to determine tachycardia and bradycardia) and the PR interval (to determine the first degree AV block).

Results

1) Before anesthesia

Eighty percent of the dogs showed respiratory sinus arrhythmia before administration of xylazine and induction of anesthesia. This rhythm irregularity is considered to be normal in the dog. No other type of arrhythmia was determined under control condition.

2) Xylazine- ketamine combination

In the ECGs taken after anesthesia, all dogs demonstrated one or more of the following rhythm abnormalities: sinus block/arrest, first-degree AV block, sinus block/arrest plus first-degree AV block (Figure 1), and sinus arrhythmia. Frequency and relative frequency of each of these rhythm abnormalities before and after ketamine-induced anesthesia are shown in Table 1. In some cases, there were more than one type of arrhythmia; sinus block/arrest accompanied by first-degree AV block was among these complex rhythm abnormalities. Of 25 recorded traces after anesthesia, sinus block/arrest and first-degree AV block were the dominant arrhythmias with 19 and 13 frequencies, respectively (Table 1). Ketamine injection prolonged the PR interval at 15 and 30 minutes after anesthesia induction (Table 2), whereas, no significant difference in the heart rate was observed (Table 3).

3) Xylazine- thiopental combination

In animals treated with thiopental one or more of the following rhythm abnormalities occurred. Sinus block/arrest, sinus block/arrest plus first-degree AV block, sinus block/arrest plus first-degree AV block plus sinus bradycardia, sinus block/arrest plus escape beat (Figure 2), sinus arrhythmia, and ventricular premature beat (Figure 3). Frequency of each of these rhythm abnormalities before and after thiopental-induced anesthesia is shown in Table 4. As with ketamine, in some cases of thiopental-treated animals, more than one type of arrhythmia were observed; sinus block/arrest plus first-degree AV block plus sinus bradycardia was among these complex rhythm irregularities. Sinus block/arrest with 10 frequencies (out of 25) was the dominant arrhythmia (Table 4).

Thiopental injection did not influence the PR interval after anesthesia induction (Table 5). However, significant decreases in the heart rate were observed at 30, 45, and 60 minutes after the onset of anesthesia (Table 6).

Table 1. Heart rhythm abnormalities, induced by xylazine-ketamine HCl in the dog

heart rhythm	evaluation time						
	before an.	15 min a.an.	30 min a.an.	45 min a.an.	60 min a.an.	3 days a.an.	Total
Regular	1	0	0	1	0	1	3
Sin. block/arrest	0	2	1	0	1	2	6
Sin. block/arrest + FAVB	0	2	4	3	3	1	13
FAVB	0	0	0	1	1	0	2
sinus arrhythmia	4	1	0	0	0	1	6
Total	5	5	5	5	5	5	30

an.= anesthesia, a.an.= after anesthesia , FAVB first-degree atrioventricular block, sin= sinus

Table 2. PR interval (in seconds, Lead II), before and after anesthesia, induced by xylazine-ketamine in the dog.

time	PR interval			significant difference ($p < 0.05$)
	Min.	Max.	mean ± SEM	
before an.	0.08	0.14	0.11 ± 0.01	-
15 min a.an.	0.12	0.16	0.13 ± 0.01	No
30 min a.an.	0.12	0.16	0.14 ± 0.01	Yes
45 min a.an.	0.12	0.16	0.15 ± 0.01	Yes
60 min a.an.	0.12	0.16	0.14 ± 0.01	No
3 days a.an.	0.08	0.16	0.13 ± 0.01	No

* The PR intervals at different time points have been compared with the control value (before anesthesia).
an.= anesthesia, a.an. = after anesthesia, Max.= maximum, Min.= minimum, min= minutes, SEM= standard error of the means.

Table 3. Heart rate (beats per minutes), before and after anesthesia, induced by xylazine-ketamine in the dog.

Time	Heart rate			significant difference ($p < 0.05$)*
	Min.	Max.	mean ± SEM	
before an.	60	120	94.00 ± 14.00	-
15 min a.an.	65	110	83.00 ± 8.00	No
30 min a.an.	70	90	77.00 ± 3.74	No
45 min a.an.	70	110	81.80 ± 7.24	No
60 min a.an.	75	95	81.60 ± 3.47	No
3 days a.an.	60	100	85.00 ± 9.22	No

* Heart rate at different time points has been compared with the control value (before anesthesia).
a.an.= after anesthesia, an.= anesthesia, Max.= maximum, Min.= minimum, min= minutes, SEM= standard error of the means.

Table 4. Heart rhythm abnormalities, induced by xylazine-thiopental in the dog

	evaluation time						
heart rhythm	before an.	15 min a.an.	30 min a.an.	45 min a.an.	60 min a.an.	3 days a.an.	Total
Regular	2	0	1	0	1	3	7
SB/arrest	0	0	1	1	3	2	7
SB/arrest + FAVB	0	1	1	0	1	0	3
SB/arrest + Sin. Brad.	0	0	1	1	0	0	2
SB/arrest + SB + FAVB	0	1	0	2	0	0	3
SB/arrest + escape beat	0	0	1	0	0	0	1
sinus arrhythmia	3	0	0	1	0	0	4
ventricular premature beat	0	3	0	0	0	0	3
Total	5	5	5	5	5	5	30

an.= anesthesia, a.an.= after anesthesia, Sin. Brad.= Sinus bradycardia, FAVB= first-degree atrioventricular block, SB = sinus block.

Table 5. PR interval (in seconds, Lead II), before and after anesthesia, induced by xylazine-thiopental in the dog.

time	PR interval			significant difference ($p < 0.05$)*
	Min.	Max.	mean ± SEM	
before an.	0.10	0.14	0.12 ± 0.01	-
15 min a.an.	0.10	0.19	0.13 ± 0.02	No
30 min a.an.	0.12	0.20	0.14 ± 0.02	No
45 min a.an.	0.12	0.18	0.14 ± 0.01	No
60 min a.an.	0.12	0.22	0.14 ± 0.02	No
3 days a.an.	0.08	0.12	0.11 ± 0.01	No

* The PR intervals at different time points have been compared with the control value (before anesthesia).
an.= anesthesia ,a.an.= after anesthesia, Max.= maximum, Min.= minimum, min= minutes, SEM = standard error of the means.

Table 6. Heart rate (beats per minutes), before and after anesthesia, induced by xylazine-thiopental in the dog.

Time	Heart rate			significant difference ($p < 0.05$)*
	Min.	Max.	mean ± SEM	
before an.	70	130	100.00 ± 13.42	-
15 min a.an.	50	140	77.00 ± 16.9	No
30 min a.an.	50	110	66.00 ± 11.22	Yes
45 min a.an.	30	90	61.40 ± 10.72	Yes
60 min a.an.	50	110	68.00 ± 10.68	Yes
3 days a.an.	70	115	93.00 ± 9.03	No

* Heart rate at different time points has been compared with the control value (before anesthesia).
an.= anesthesia, a.an.= after anesthesia , Max.= maximum, Min.= minimum, min= minutes, SEM= standard error of the means.

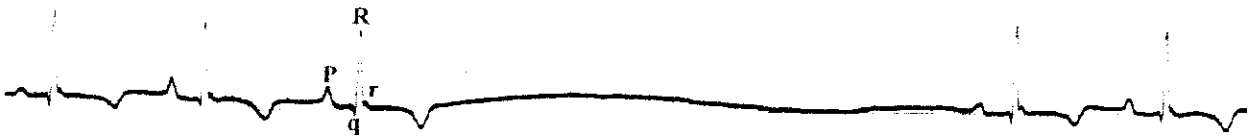

Fig 1. Sinus arrest accompanied by first-degree AV block in a dog. 15 minutes after induction of anesthesia with a ketamine-xylazine combination (Lead II: 25 mm/sec., 10 mm = 1 mV).

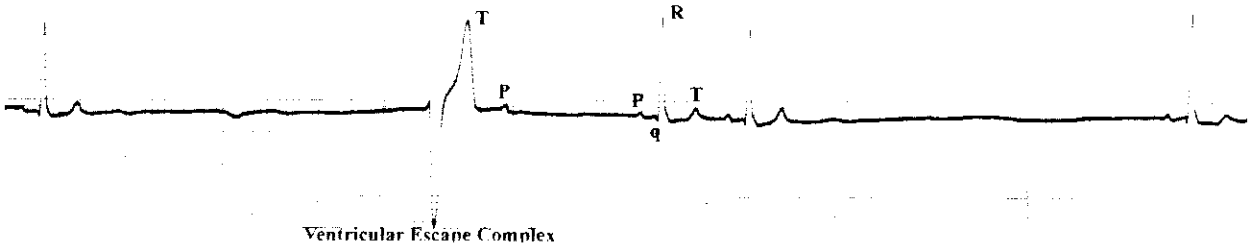

Fig 2. Sinus arrest and ventricular escape complex in a dog. 30 minutes after induction of anesthesia with a thiopental-xylazine combination. Notice that the P wave appeared after the escape complex, during ventricular refractory period, hence, was not followed by ventricular depolarization (Lead aVF: 25 mm/sec., 10 mm = 1 mV).

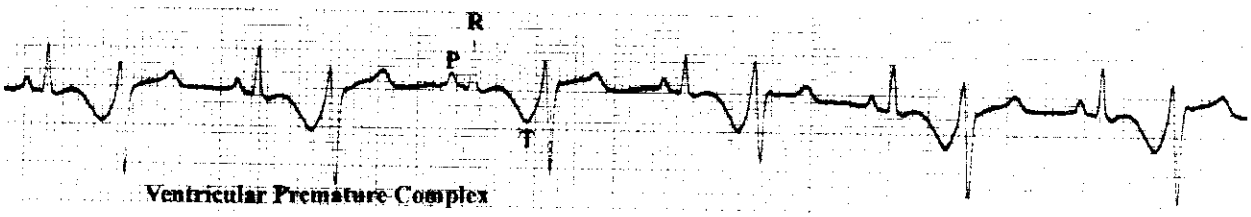

Fig 3. Ventricular premature complexes with a bi-geminal pattern in a dog. 15 minutes after induction of anesthesia with a thiopental-xylazine combination (Lead aVF: 25 mm/sec., 10 mm = 1 mV).

Discussion

Electrocardiography was used to evaluate cardiac arrhythmias during general anesthesia induced by combinations of xylazine-ketamine and xylazine-thiopental. Sinus arrest/block and first degree A-V block were the most common arrhythmias observed after xylazine-ketamine administration. During xylazine-thiopental anesthesia, ventricular premature beats with bigeminy pattern and sinus bradycardia were also recorded.

The α_2-adrenoceptor agonist, xylazine, has prominent vagotonic effect. Sinus bradycardia, sinoatrial block, first and second degree A-V block, A-V dissociation and advanced sinus arrhythmia have been reported following administration of this drug [9].

Despite claimed anti-arrhythmic effect of ketamine[9], this drug reduces the ventricular arrhythmogenic dose of epinephrine in halothane-anesthetized cats[10]. In lower doses, this dissociative anesthetic is believed to have an inhibitory effect on the centrogenic arrhythmias. However, at higher doses, it may induce centrogenic arrhythmias in spontaneously breathing rats[11]. Ketamine increase sympathetic tone and has inhibitory effect on parasympathetic nervous system[12, 13]. However, the nature of arrhythmias observed in

the present study, indicates ketamine depressant action on cardiac impulse transmission by a potentiating effect on the parasympathetic activity. Considering the arrhythmogenic activity of xylazine and the effects of ketamine on cardiac electrophysiology, we concluded that the cardiac arrhythmias observed in anesthetized dogs mainly aroused either from the action of xylazine or interaction between xylazine and ketamine on the heart. In agreement with our findings, Kim *et al.*[4], reported several kinds of arrhythmias including sinus arrest and first-degree heart block following the administration of xylazine/ketamine combination. They concluded that inhibited cardiac function is likely due to xylazine. Their findings are in agreement with the pervious records[3, 10, 17, 18, 19, 20, 21] that xylazine inhibited cardiopulmonary function even when administered together with ketamine by increasing vagal tone occurring in response to hypertension.

Thiopental has direct depressant effect on myocardium and can cause ventricular premature contractions[7]. Ventricular bigeminy is the most characterized arrhythmia in animals anesthetized by thiopental[6, 13]. The clinical importance of this abnormality is unknown, but it does not seem to progress to ventricular fibrillation[7]. In dogs, thiopental sensitizes the heart to epinephrine in a dose-dependent manner. This sensitizing action would in part explain the potentiation by thiopental of hydrocarbon anesthetic-epinephrine arrhythmias[22]. In addition, again in dogs, thiopental (20 mg/kg) potentiates various types of epinephrine-induced ventricular arrhythmias (ventricular ectopy, bigeminy and tachycardia) with enflurane, and ventricular tachycardia with isoflurane[23]. This ultra-short acting barbiturate reduces the epinephrine dose needed for AV dissociation and ventricular, but not atrial, arrhythmias[24]. In addition, epinephrine-induced ventricular arrhythmias in halothane-anesthetized dogs are potentiatied by thiamylal and thiopental[25]. In the present study, besides arrhythmias observed in anesthesia with ketamine, sodium thiopental caused ventricular premature complexes with bigeminy rhythm and sinus bradycardia. Again, the contribution of xylazine on genesis of these arrhythmias is perceivable.

One of the striking findings in this study was continuation of some of the arrhythmias (e.g. sinus block/arrest) 3 days after recovery in three of five dogs anesthetized with both xylazine-ketamine and xylazine-thiopental combination. The half life of these drugs are too short to cause such effect after such a long time[6]. The cause and importance of this unexpected finding needs to be determined by future studies.

From the data presented in this study, it is concluded that both xylazine-ketamine and xylazine-thiopental combinations may precipitate serious cardiac arrhythmias in the dog. Therefore, it is suggested to use such combinations with more caution in the animals prone to cardiac disturbances.

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چکیده:

مقایسه اثرات ترکیب زایلازین-کتامین و زایلازین- تیوپنتال

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هدف: بررسی اثرات ترکیبی داروهای زالازین- کتامین و زایلازین- تیوپنتال.
طرح: مطالعه تجربی.
حیوانات: ۵ قلاده سگ بالغ از هر دو جنس.
طرح: مطالعه تجربی.
روش کار: در این بررسی، متعاقب تزریق داروی پیش بیهوشی زایلازین هیدروکلراید، داروهای کتامین هیدروکلراید و تیوپنتال سدیم برای ایجاد بیهوشی عمومی در حیوانات مذ (سگ) تجویز گردید. ریتم قلب قبل و همچنین ۱۵، ۳۰، ۴۵ و ۶۰ دقیقه و **سه روز بعد از القای بیهوشی ارزیابی گردید.**
نتایج: انسداد و ایست سینوسی و انسداد دهلیزی-بطنی نوع اول متداولترین بی نظمی ریتم قلبی مشاهده شده متعاقب تجویز کتامین هیدروکلراید بود. در طول بیهوشی القاء شده توسط تیوپنتال سدیم، ضربان نارس بطنی با الگوی دو تایی و برادیکاردی سینوسی ثبت گردید.
نتیجه گیری: با توجه به اثرات مستند داروهای ذکرشده بر ریتم قلب، بی نظمی ایجاد شده احتمالاً ناشی از ترکیب زایلازین هیدروکلراید با داروهای بیهوش کننده کتامین هیدروکلراید و تیوپنتال سدیم بوده است.
کلید واژه ها: آریتمی قلبی، سگ، کتامین، تیوپنتال، زایلازین