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Electrocardiography study in the dogs subjected to ovariohysterectomies anaesthetized with tiletamine-zolazepam and ketamine-diazepam combinations

P.D. Salve[1], M.G. Thorat[2†], R.V. Raulkar[3] and R.I. Hatzade[4]

Maharastra Animal and Fishery Science University, Nagpur-440001 (MS)

[1]MVSc Scholar, [2]Professor & Head, [3]Assistant Professor, Department of Veterinary Surgery and Radiology, [4]Ph.D. Scholar, Department of Veterinary Medicine, Post Graduate Institute of Veterinary and Animal Sciences, Akola

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The study was conducted to evaluate the electrocardiographic changes in 12 healthy female dogs undergoing ovariohysterectomy pre-anaesthetized with xylazine. These dogs were randomly divided into two equal groups. In Group I induction and maintenance of anaesthesia was achieved using Tiletamine – Zolazepam (3 mg/kg BW IV) combination whereas in Group II induction and maintenance of anaesthesia was achieved using Ketamine (3 mg/kg BW IV) preceded by administration of Diazepam (0.25 mg/kg BW IV). Significant increase in the value of P wave duration was observed at 30 min. P-R and Q-T interval was significantly increased at different time intervals. Amplitude of T wave was increased in both groups. Sinus arrhythmia and sinus bradycardia were recorded in both groups, however, first degree AV block was observed in Group I.

Keywords: *Dogs, ECG, Anaesthesia, Xylazine*

Electrocardiography (ECG) is a non-invasive and affordable tool used frequentlyto determine heart rate, cardiac rhythm, conduction integrity and mean electrical axis. ECG monitoring is an inevitable part of anaesthesia monitoring as most of the anaesthetic agents cause cardio-pulmonary depression.

Electrocardiography was conducted in 12 dogs subjected to ovariohysterectomies anaesthetized with tiletamine- zolazepam and ketamine- diazepam combinations. These dogs were randomly divided into two equal groups.

In group I dogs, induction was achieved using Inj. Tiletamine-Zolazepam (Zoletil) combination @ 3mg/kg BW I/V and in group II dogs, induction was achieved using Inj. Ketamine @ 3mg/kg BW I/V preceded by intravenous administration of Diazepam @ 0.25mg/kg BW. Xylazine was used as pre-anaesthetic agent in both the groups @ 1 mg/kg BW IM. Heart rate was calculated from the lead II on ECG paper at 0 min i.e. before sedation, 15, 30, 45 min after induction and at the time of recovery from anesthesia.

Amplitude and duration of P wave and QRS complex, P-R and Q-T interval were recorded before sedation (0 min), at 15, 30, 45 min during anaesthesia and at the time of recovery from anaesthesia. Similarly, T wave amplitude and presence or absence as well as classification of arrhythmia was recorded by performing ECG and reading lead II on ECG paper. The ECG was recorded with the help of hand-held ECG recorder BPL CARDIART 108T-DIGI at speed 50 mm/sec and amplitude at 10 mV.

The data was statistically analysed using the ICAR WASP 2.0 software's Two Way Factorial Experimental Design.

In both the groups heart rate was decreased significantly ($p<0.01$) at 15, 30 and 45 min when compared with base value (0 min). The heart rate might be decreased due to depression of sympathetic activity (Yaygingül and Belge 2018).

There was non-significant difference in the amplitude of P wave at different intervals in both the groups as compared to their base values. These findings are in agreement with the findings of Demirkan *et al.* (2002). P wave duration remained same throughout anaesthetic period except at 30 min where it was increased significantly ($p<0.05$) in both the groups. This increased in the duration of P wave might be attributed to cardio-depressant effect of xylazine (Samimi *et al.* 2020).

P-R interval was significantly ($p<0.01$) increased at different time intervals. The increased in the duration of P-R interval might be due to depression of sympathetic activity and increase in parasympathetic tone due to α2 agonist that leads to decreased heart rate (Yaygingül and Belge, 2018). When heart rate decreases P-R interval increases and vice-versa (Miller *et al.,* 1999). P-R interval in both the groups was clinically insignificant as the values were within normal physiological limit.

The changes in the QRS amplitude and QRS duration were non-significant. Q-T interval was increased significantly ($p<0.01$) in both groups. QT interval is depended on the heart rate i.e. slower the heart rate, longer the QT interval and vice versa (Sarchahi *et al.* 2009).

In present study the amplitude of T wave in all the dogs of both groups was normal i.e. 25% of amplitude of QRS complex before sedation. But during

†Corresponding author; E-mail: milindthrt@yahoo.co.in

Table 1. Mean±SE of Heart Rate (beats/min), P wave amplitude (mV), P wave duration (sec), P-R Interval (sec), QRS amplitude (mV), QRS duration (sec) and Q-T Interval (sec)

	Group	Time Interval (min)				
		0	15	30	45	At Recovery
Heart Rate	**I**	124.50±9.34	80.00±9.30**	73.16±7.93**	69.83±9.13**	64.16±4.16**
	II	107.00±8.79	62.16±6.92**	79.83±7.37**	76.33±4.52**	63.00±4.03**
P WaveAmplitude	**I**	0.20±0.02	0.19±0.02	0.19±0.02	0.16±0.03	0.20±0.02
	II	0.15±0.02	0.17±0.01	0.18±0.01	0.20±0.02	0.20±0.02
P WaveDuration	**I**	0.040±0.000	0.040±0.000	0.042±0.002*	0.040±0.000	0.040±0.000
	II	0.040±0.000	0.040±0.000	0.043±0.002*	0.040±0.000	0.040±0.000
P-R Interval	**I**	0.10±0.00	0.13±0.00**	0.13±0.00**	0.13±0.00**	0.13±0.00**
	II	0.10±0.00	0.12±0.00**	0.11±0.00**	0.12±0.00**	0.11±0.00**
QRS Amplitude	**I**	0.88±0.11	0.92±0.15	0.81±0.10	0.87±0.14	0.85±0.14
	II	0.93±0.13	0.86±0.13	0.77±0.12	0.78±0.12	0.83±0.12
QRS Duration	**I**	0.04±0.00	0.05±0.00	0.05±0.00	0.05±0.00	0.05±0.00
	II	0.05±0.00	0.05±0.00	0.05±0.00	0.05±0.00	0.05±0.00
Q-T Interval	**I**	0.18±0.00	0.24±0.00**	0.23±0.01**	0.26±0.01**	0.24±0.00**
	II	0.20±0.00	0.26±0.01**	0.25±0.01**	0.25±0.01**	0.26±0.01**

**Significant at 1% level, *Significant at 5% level

anaesthesia it was observed that the amplitude of T wave was spiked i.e. increased beyond normal level in four dogs from group I and three dogs from group II. The spiked T wave amplitude in present study could be due to bradycardia observed in both the groups (Miller *et al.* 1999).

In current study sinus arrhythmia was recorded in two dogs of groups I and in four dogs of group II. Sinus arrhythmia observed in this study might be due to administration of xylazine as xylazine enhances the vagal tone (Kwon *et al.* 2003). Sinus bradycardia (Fig. 1) was recorded in one dog of both the groups, which might be attributed to administration of anaesthetic agents and sedatives (Tilley and Smith, 2008) or due to significant declined in heart rate below normal range (Samimi *et al.* 2020). In the present study first degree AV block was observed in one dog of group I, whereas it was observed in combination with sinus bradycardia in two dogs of group I and in one dog of group II. First degree AV block was observed in this study might be due to vagal stimulation by anaesthetic and sedative agents (Samimi *et al.*, 2020).

It can be concluded that xylazine has an arrhythmogenic effect characterised by bradycardia, sinus arrhythmia, sinus bradycardia and first-degree AV block.

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