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ELECTROCARDIOGRAPHY IN CANINE CARDIAC DISEASES

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ABSTRACT

In this article, electrography for diagnosis and screening of fifteen dogs for cardiac diseases were done. ECG waveform measurement and interpretation was performed using standard bipolar (Lead I, II & III) and augmented limb leads (aVR, aVL & aVF). The ECG waveform measurements revealed significant ($p<0.01$) increase in P (sec) and P (mV) in both hypertrophic cardiomyopathy and valvular disease groups indicating atrial enlargement. Arrhythmias observed in cardiac diseases were increased QRS duration, electrical alternans, atrial fibrillation, ST depression, P mitrale, increased QRS amplitude, ventricular premature complexes (VPCs), deep Q wave, atrioventricular (AV) blocks and right bundle branch block (RBBB). Timely performed electrocardiography is helpful in screening and diagnosing canine cardiac diseases and arrhythmias.

Key words: Atrial fibrillation, Arrhythmia, Electrical alternans, Ventricular premature complex.

Introduction

Electrocardiography (ECG) is a non-invasive, inexpensive, non-hazardous easy to use diagnostic technique to identify cardiac arrythmias, cardiac chamber enlargements, myocardial diseases, ischemia, heart failure, conduction defects (heart blocks, bundle branch blocks), monitoring antiarrhythmic drugs and pericardial effusions. Detection of cardiac arrhythmias in the early and advanced stages of heart disease is essential for diagnostic and prognostic purposes (Wess et al., 2010). Cardiac arrhythmia is defined as a disturbance in heart rhythm, including abnormalities of impulse formation and/ or conduction (Ware 2009). Disturbances of excitability and impulse formation are the most common causes of arrhythmias in dogs (Aptekmann et al., 2010). Cardiac arrhythmias and intracardiac conduction abnormalities are common problems in dogs (Gugjoo et al., 2014a). ECG patterns in animals can change with body weight, age, and fat deposition, resulting in changes in electrical activity and heart rhythm seen on the ECG (Neto et al., 2010). Kraus et al. (2008) recommended that 24-hour Holter recording is essential to establish a definite diagnosis for in-depth examination of the number and quality of the arrhythmia and to ascertain medication effectiveness. DeFransesco, 2001 and Velhankar, 2013 opiniated that electrocardiography is very useful as an inexpensive diagnostic tool for identifying cases of rhythm disorders and DCM.

Materials and methods

Selection of animals

The present study was carried out on 3166 dogs presented at Department of Veterinary Medicine, Advanced Veterinary Multispeciality Complex of DGCN, COVAS, CSKHPKV, Palampur (H.P.) over a period from May 2021 to September 2022 and 15 dogs were diagnosed with various cardiac disorders as Dilated cardiomyopathy (DCM), Hypertrophic cardiomyopathy (HCM), valvular diseases (MMVD) and Pericardial effusion (PE). The cardiac disorders were confirmed based on signalment, medical history, clinical examination, electrocardiography, laboratory examination, radiography, cardiac troponin-I biomarker and echocardiography.

Procedure

ECG was recorded in a quiet place according to standard procedure described by Tilley and Smith (2016) using alligator type electrode clips to obtain standard bipolar limb leads (Lead I, II & III) and augmented limb leads (aVR, aVL & aVF). The animals were placed in right lateral recumbency on a non-conducting surface and all conductive wearables were removed. The limbs were kept perpendicular without contacting each other to the long axis of body and parallel to each other.

Equipment

The electrocardiogram was obtained using a three channel ECG machine RMS Vesta 301i (Recorders & Medicare Systems Ltd, Industrial area Phase 1, Panchkula, Haryana).

ECG measurement & interpretation

The ECG was recorded @25mm/s and 50mm/s in all six leads and measurements were recorded using lead II @50mm/s speed and 1mm/mV sensitivity. ECG waveform measurement and interpretation was performed as described by Tilley and Smith (2016).

Statistical analysis

The mean and standard error for electrographic parameters were calculated and Computer Software Instat from Graphpad software, 2008 was used for the statistical analysis of data at 1% and 5% level of significance.

Table 1: Classification of Arrhythmias (Tilley and Smith, 2016)

Sr.no	Arrhythmia classification	Type of Arrhythmias
1.	Normal sinus impulse formation	Normal sinus rhythm, Sinus arrhythmia, Wandering pacemaker
2.	Disturbances of sinus impulse formation	Sinus arrest, Sinus bradycardia, Sinus tachycardia
3.	Disturbances of supraventricular impulse formation	Atrial premature complexes, Atrial tachycardia Atrial flutter, Atrial fibrillation
4.	Disturbances of ventricular impulse formation	Ventricular premature complexes, Ventricular tachycardia, Ventricular fibrillation
5.	Disturbances of impulse conduction	Atrial standstill, Sinoatrial block, Heart blocks (AV blocks), Bundle branch blocks
6.	ECG waveform morphology	Deep Q, Tall T waves, Increased/ Decreased QRS, P pulmonale, P mitrale
7.	Miscellaneous	Electrical alternans

Results and discussion

The electrocardiographic waveform parameters and arrhythmia findings in Cardiac diseases are presented in Table 2 and Table 3 respectively. The ECG waveform measurement revealed significant ($p<0.01$) increase in P (sec) (0.14 ± 0.01) and P (mV) in both HCM (0.40 ± 0.00) and valvular disease (0.29 ± 0.06) groups as compared to healthy control. Arrhythmia findings in overall cardiac cases were increased QRS duration (n=7, 46.67%), electrical alternans (n=5, 33.33%), atrial fibrillation (n=3, 20%), ST depression (n=3, 20%), P mitrale (n=3, 20%), increased QRS amplitude (n=3, 20%), ventricular premature complexes (n=2, 13.33%), deep Q wave (n=2, 13.33%), atrioventricular blocks (n=2, 13.33%) and right bundle branch block (n=1, 6.66%).

In dogs suffering from DCM, P (sec) was significantly ($p<0.01$) increased with respect to control group. Increased QRS duration, electrical alternans, atrial fibrillation, ST depression, P mitrale, increased QRS amplitude were other findings in this group. Similarly, Velhankar (2013) reported atrial fibrillation (21.73%) as the most common arrhythmia in 23 DCM-affected dogs along with ventricular premature complexes (VPCs), ventricular tachycardia, extended P waves (LAE) and

QRS complexes (LVE) associated with structural changes. Dogs with bilateral DCM showed taller P wave indicating right atrial enlargement. Atrium is often dilated in DCM, reflecting raised filling pressures and/or associated valvular abnormalities. According to Tilley (1992), right atrial enlargement is indicated by a P wave amplitude >0.4 mV and left atrial enlargement by a P wavelength >0.04 sec (P mitrale). It was also stated that deep Q > 0.5 mV in Lead II and deep S in Leads I, II, III and aVF is suggestive of right ventricular enlargement or hypertrophy. Atrial fibrillation is mainly caused by atrial volume overload and subsequent atrial enlargement, or by spontaneous electrical activity in the pulmonary veins (Chen et al., 2000). According to Cote (2010), ST segment depression or ST coving may be related to myocardial hypoxia, nonspecific electrolyte abnormalities, or cardiac hypertrophy. Atrial fibrillation is characterised by the presence of "f" fibrillation waves, absence of P waves, fast ventricular rate, and irregular ventricular depolarizations due to numerous ectopic atrial depolarization locations and varied AV nodal refractoriness. VPCs are recognised by their wide and peculiar appearance and their lack of a P wave but association with a large T wave (Tilley and Smith, 2016). According to Ettinger and Feldman, (2006) ventricular extra systole also known as VPC, is the most common of all arrhythmias and is caused by an ectopic focus in the ventricular myocardium. R-wave amplitude is commonly used to assess left ventricular function and is a good indicator of ventricular contraction (Gugjoo et al., 2014b).

In HCM (group II), P (mV) was significantly ($p<0.01$) increased with respect to control group and had increased QRS duration, P mitrale, increased QRS amplitude and deep Q wave as ECG abnormalities. The increased width of QRS complexes and their increased amplitude – implied left ventricular enlargement. Liu et al. (1979) reported ventricular premature complexes, paroxysmal ventricular tachycardia, first degree and third-degree AV block in dog affected with HCM. The in humans are impaired diastolic filling and LVOT obstruction are the main functional abnormalities associated with HCM.

In MMVD group, P (mV) was significantly ($p<0.01$) increased with respect to control group and only one case had increased QRS duration. It is assumed that the duration of the P-wave reflects the electrophysiological properties of the atrium muscle. As the electrical activity of the cardiac muscle displayed on the electrocardiogram is closely correlated with the conduction of specific areas of the atrium; the regional depolarization disturbances may lead to variety of the duration of the P-wave at different ECG leads. Chiavegato et al. (2009) reported LA and LV enlargement as important electrocardiographic indicators of MMVD along with presence of systolic and diastolic dysfunctions and pulmonary hypertension.

In PE, there was no significant change in ECG parameters when compared with control group. ST depression was present in one case while all cases were having low QRS complexes and electrical alternans as ECG findings. Similarly, Saini (2014) reported low voltage QRS complexes (83%) and electrical alternans (25%) as major ECG findings in pericardial effusions. When there is a lot of fluid in the pericardial sac, the heart may swing back and forth within the pericardium, causing electrical alternans, which is an alternation of the T wave or QRS complex with each pulse (Ware, 2000). Electrical alternance is caused by mechanical anterior–posterior swinging of the heart in the large fluid and is frequently seen in malignant causes such as metastatic lung cancer.

Table 2: ECG waveform parameters in Cardiac diseases

PARAMETER	CONTROL (n=96)	CARDIAC (n=15)	DCM (n=8)	HCM (n=2)	MMVD (n=3)	PE (n=2)
P (mV)	0.117 ± 0.006	0.20 ± 0.40	0.14 ± 0.05	0.40 ± 0.00*	0.29 ± 0.06*	0.10 ± 0.00
P (sec)	0.036 ± 0.001	0.03 ± 0.05	0.14 ± 0.01*	0.05 ± 0.01	0.04 ± 0.00	0.03 ± 0.01
QRS (mV)	0.818 ± 0.061	1.33 ± 0.48	1.64 ± 0.38	1.73 ± 0.65	1.33 ± 0.23	0.45 ± 0.05
QRS (sec)	0.049 ± 0.001	0.06 ± 0.04	0.06 ± 0.01	0.06 ± 0.00	0.05 ± 0.01	0.04 ± 0.00
T (mV)	0.190 ± 0.038	0.24 ± 0.43	0.30 ± 0.07	0.24 ± 0.05	0.18 ± 0.06	0.14 ± 0.05
T (sec)	0.056 ± 0.005	0.06 ± 0.04	0.07 ± 0.01	0.06 ± 0.00	0.05 ± 0.01	0.05 ± 0.01
PR (sec)	0.086 ± 0.003	0.08 ± 0.11	0.06 ± 0.02	0.10 ± 0.00	0.09 ± 0.01	0.09 ± 0.01
QT (sec)	0.202 ± 0.004	0.18 ± 0.06	0.20 ± 0.01	0.17 ± 0.01	0.17 ± 0.01	0.15 ± 0.01

Values with * are highly significant as compared to control group at p≤ 0.01

Table 3: Arrhythmia findings in Cardiac diseases

ARRHYTHMIAS	CARDIAC DISEASES				
	DCM (n=8)	HCM (n=2)	MMVD (n=3)	PE (n=2)	Total (n=15)
	Cases (%)	Cases (%)	Cases (%)	Cases (%)	Cases (%)
ST depression	2 (25%)	0.00%	0.00%	1 (50%)	3 (20%)
Atrial fibrillation	3 (37.50%)	0.00%	0.00%	0.00%	3 (20%)
P mitrale	2 (25%)	1 (50%)	0.00%	0.00%	3 (20%)
↑ QRS amplitude	2 (25%)	50.00%	0.00%	0.00%	3 (20%)
↑ QRS duration	5 (62.50%)	1 (50%)	1 (33.33%)	0.00%	7 (46.67%)
Low QRS complexes	0 (0.00%)	0.00%	0.00%	2 (100%)	2 (13.33%)
VPCs	2 (25%)	0.00%	0.00%	0.00%	2 (13.33%)
Electrical alternans	3 (37.50%)	0.00%	0.00%	2 (100%)	5 (33.33%)
Deep Q wave	1 (12.50%)	1 (50%)	0.00%	0.00%	2 (13.33%)
AV blocks	2 (25%)	0.00%	0.00%	0.00%	2 (13.33%)
RBBB	1 (12.50%)	0.00%	0.00%	0.00%	1 (6.66%)

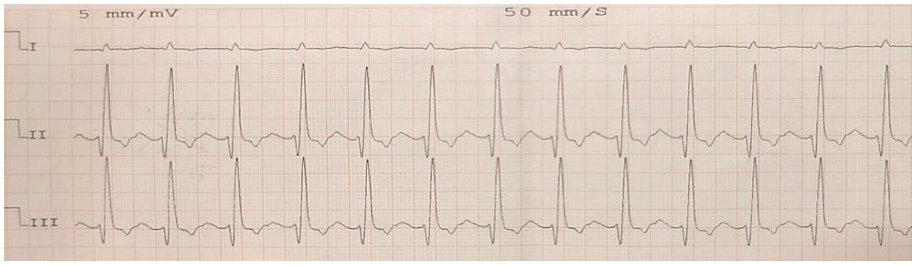

Figure 1: ECG showing increased QRS amplitude = 4mV @5mm/mV (Half sensitivity) indicative of ventricular enlargement

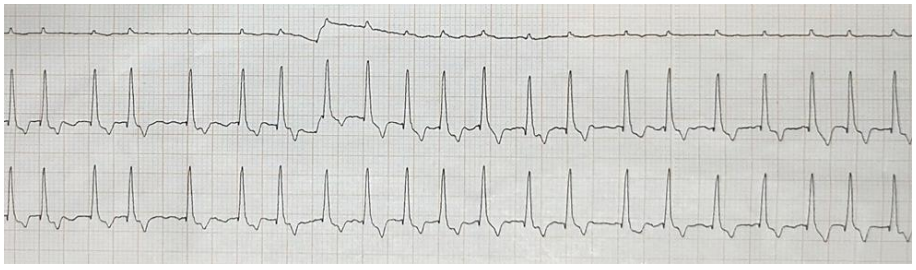

Figure 2: ECG showing Atrial fibrillation with electrical alternans in a dog with DCM

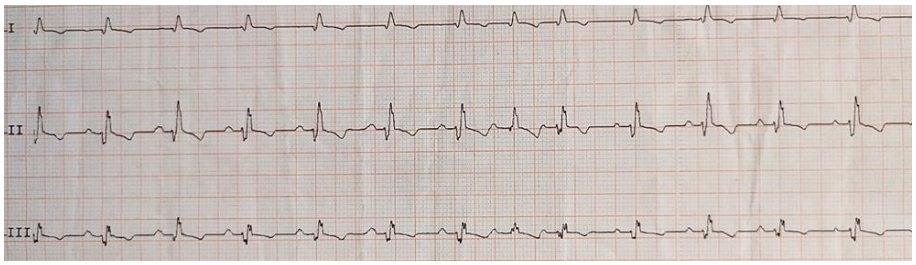

Figure 3: ECG showing low QRS complexes with electrical alternans in a dog with Pericardial effusion

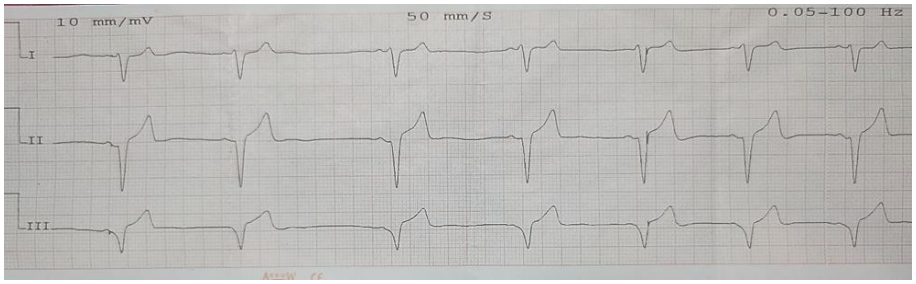

Figure 4: ECG showing right bundle branch block (RBBB)

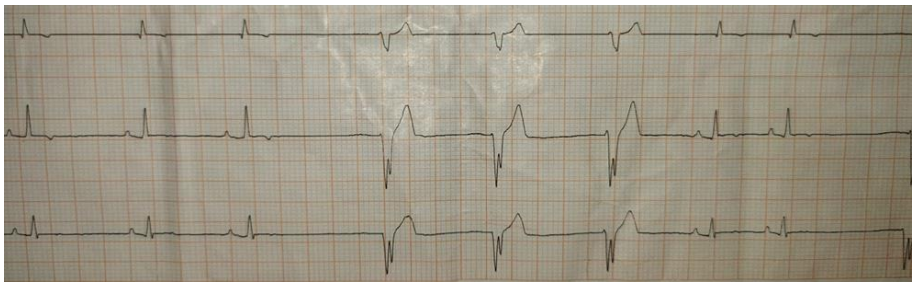

Figure 5: ECG showing triplet of ventricular premature complexes (VPCs)

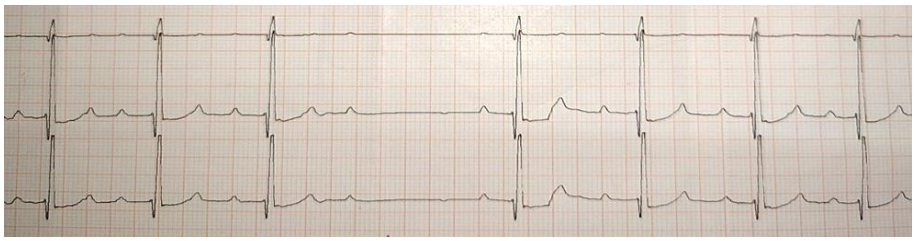

Figure 6: ECG showing 2nd degree heart block (Mobitz type II)

Conclusion

Arrhythmias are common in dogs, hence its important for the veterinarian to understand what is happening to the patient and what affect it is having on cardiac output. Therefore, timely performed electrocardiography is helpful in screening and diagnosing canine cardiac diseases and arrhythmias.

References

1. Aptekmann K. P., Vailati M. F., Fortuna T. M., Schwartz D. S. (2010). *Prevalence of cardiac arrhythmias and conduction disturbances in dogs and cats in Botucatu, Brazil (2003–2007)*. J Vet Res An Sci 47: 371–79.
2. Chen Y.J., Chen S.A., Chang M.S., Lin C.I. (2000). *Arrhythmogenic activity of cardiac muscle in pulmonary veins of the dog: Implication for the genesis of atrial fibrillation*. Cardiovasc Res 48, 265–273.
3. Chiavegato D., Borgarelli M., D'Agnolo G., Santilli R. A. (2009). *Pulmonary hypertension in dogs with mitral regurgitation attributable to myxomatous valve disease*. Vet Radiol Ultrasound 50: 253–258.
4. Cote E., (2010). *Electrocardiography and cardiac arrhythmias*. In: Ettinger, S. J., Feldman, E. C., (eds): Textbook of Veterinary Internal Medicine, Edn. 7th, W. B. Saunders Co., Philadelphia: 1159–1187.
5. DeFransesco. (2001). *Advanced discussions in the diagnosis of heart failure*. In a publication on "Advances in management of cardiac patients" Waltham and Merial, USA: 3–11.
6. Ettinger, S. J. and Feldman E.C. (2006*). In "Textbook of Veterinary Internal Medicine Diseases of the Dog and Cat" 6th* Ed. W. B. Saunders Company, Philadelphia, USA.
7. Gugjoo M. B., Hoque M., Saxena A. C., Samsuz Zama M. M., (2014a). *Reference values of six-limb-lead electrocardiogram in conscious Labrador retriever dogs.* Pak J Biol Sci 17(5): 689–695.
8. Gugjoo M. B., Hoque., M., Saxena A. C., Shamsuz Zama, M. M., Dey, S. (2014b). *Reference values of M-mode echocardiographic parameters and indices in conscious Labrador retriever dogs*. Iran J Vet Res 15(4): 341–346.
9. Kraus M. S., Gelzer A. R. M., Moise S. (2008*). Treatment of cardiac arrhythmias and conduction* disturbances. Manual of canine and feline cardiology, Edn. 4th, W. B. Saunders Co., Philadelphia, USA: 315–32.
10. Liu S.K., Barry J. M., Lawrence P.T. (1979). Hypertrophic cardiomyopathy in the dog. Am J Path 94: 497–508.
11. Neto, G. B. P., Brunetto, M. A., Sousa, M. G., Carciofi, A. C., Camacho, A. A. (2010). *Effects of weight loss on the cardiac parameters of obese dogs*. Pesq Vet Bras. Rio De Janeiro 30:2.
12. Saini N. (2014*). Diagnosis and Therapeutic Management of Myocardial and Valvular Diseases in Dogs*. Ph.D. Dissertation, Guru Angad Dev Veterinary and Animal Sciences University Ludhiana, India.
13. Tilley L. P. (1992). *Essentials of canine and feline electrocardiography*. 3rd Edn. Lea and Fabiger, Philadelphia 1–252.
14. Tilley L., Smith F. (2016). *Electrocardiography. Manual of canine and feline cardiology 5th Edn*. W.B Saunders, Philadelphia, USA: 49–76.
15. Velhankar RD. (2013). *Two dimensional echocardiographic studies on dilated cardiomyopathy in dog*. Ph.D. Thesis. Maharashtra Animal and Fishery Sciences University, Nagpur, India.
16. Ware W. A. (2000). *Pericardial diseases*. In: Abbott A. J. (ed.) Small Animal Cardiology Secrets. Hanley and Belfus, Philadelphia: 276–285.
17. Ware W A. (2009). *Cardiovascular system disorder*. In: Nelson R. W. and Couto C. G. (Eds). Small Animal Internal Medicine, 4th Edn. Mosby Elsevier: 1–95.
18. Wess G., Schulze A., Simak J., Killich M., Keller L. J., Maeurer J. and Hartmann K. (2010). *Prevalence of dilated cardiomyopathy in Doberman Pinschers in various age groups*. J Vet Intern Med 24(3): 533–38.