ELECTROCARDIOGRAPHIC STUDIES ON ARRHYTHMIA IN DOGS

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DECLARATION

I hereby declare that the thesis entitled **"ELECTROCARDIOGRAPHIC STUDIES ON ARRHYTHMIA IN DOGS"** is a record of research work done by me during the course of research and this thesis has not previously formed the basis for the award of any degree, diploma, fellowship or associateship or other similar title, of any other University or Society.

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CERTIFICATE

Certified that the thesis entitled **"ELECTROCARDIOGRAPHIC STUDIES ON ARRHYTHMIA IN DOGS"** is a record of research work done independently by **Ranimol Antony** under my guidance and supervision and that it has not previously formed the basis for the award of any degree, diploma, fellowship or associateship to her.

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Ranimol Antony

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LIST OF ABBREVIATIONS

@- At the rate of

cu. mm- Cubic Millimetre

bpm-beats per minute

ECG-Electrocardiogram

EDTA- Ethylene Diamine Tetra Acetate

°F- Fahrenheit

g- Gram

g/dl- Grams per decilitre

MEA- Mean electrical axis

mEq/l- milli equivalent per litre

mg – Milligram

mg/dl- Milligram per decilitre

ml – Millilitre

MHz- Mega hertz

mv- Milli volts

SE-Standard Error

sec – second

U/L- units per litre

Introduction

1. INTRODUCTION

Cardiac arrhythmias are defined as abnormalities of rate, rhythm or conduction of electrical activity due to abnormal impulse formation or conduction. Arrhythmias are classified by the origin of the impulse (sinus node, atrial, junctional and ventricular) and the deviation of the rate from the normal sinus rate (tachycardia and bradycardia).

The presence of potential for development of arrhythmias should be suspected in patients with conditions resulting in poor tissue perfusion, hypoxemia, multiple trauma, primary cardiac diseases and serum electrolyte abnormalities.

The significance of an arrhythmia is dependent on two major factors: the type of arrhythmia (timing and frequency) and the associated clinical condition of the affected animal.

Electrocardiogram, a recording of the sum of the electrical potential generated by the heart during impulse formation and propagation, is the definitive tool for evaluating most cardiac arrhythmias. But the underlying mechanism usually can not be determined from the electrocardiogram. In such condition, radiography and echocardiography proved to be useful techniques in recognising cardiac disease.

Managing cardiac arrhythmia in small animals is a challenging task to the clinicians. Many arrhythmias do not require specific treatment and are self limiting with treatment of underlying disease. Serious arrhythmias, however can significantly impair cardiac output, decrease coronary perfusion and impair myocardial performance leading to congestive heart failure or sudden death.

Considering these views, the present study entitled "Electrocardiographic Studies on Arrhythmia in Dogs" was undertaken with the following objectives,

1. Screening of dogs for cardiac disorders.

2. To study the different type of arrhythmia based on ECG.

Review of Literature

2. REVIEW OF LITERATURE

2.1. INTRODUCTION

Arrhythmia substantially affected morbidity and survival of animals with Congestive Heart Failure (CHF) and should be considered when selecting therapy. (Goodwin and Strickland a, 1998)

Bosswood (2001) stated that cardiac arrhythmias were commonly encountered in veterinary patients and these animals might be suffering from cardiac disease, non- cardiac disease or apparently normal.

DeFransesco (2004) reported that heart disease was a major health problem in dogs world wide and according to a survey conducted by Morris Animal Foundation, cardiac failure was believed to be the second most common cause of death in dogs.

Bhatt *et al.* (2005) stated that arrhythmias are disorders of heart rhythm due to abnormalities in impulse generation, impulse conduction or a combination of both.

2.2. ETIOLOGY

Bolton (1975) reviewed that arrhythmias might be secondary to complicated disease conditions like diabetes mellitus, adrenocortical insufficiency, pyometra, idiopathic cardiomyopathy, aortic stenosis, endocarditis, intestinal obstructions and toxemias.

Cardiac arrhythmias were associated with thoracic trauma, neurologic injury, severe shock and extensive tissue trauma. (Macintire and Snider, 1984)

Ventricular arrhythmias occurred rarely and resulted from myocardial ischemia in dogs with atherosclerosis. Atrial fibrillation had also occurred with increased frequency in hypothyroid dogs. (Panciera, 1994)

Goodwin and Strickland (1998 b) stated that heart disease could cause cardiac chamber dilatation which could physically disrupt cell to cell communication leading to arrhythmias.

The mechanical activity of the heart was controlled by electrical impulses and these impulses were intrinsic to the heart but also modulated by neuronal activity through the autonomic nervous system. (Keating and Sanguinetti, 2001)

Goodwin (2001) reported that cardiac arrhythmias might occur either in the presence or in the absence of specific cardiac disease and numerous systemic disorders might be correlated with abnormal cardiac rhythms.

Martin (2003) reported that in heart failure, a reduction in cardiac output led to activation of the sympathetic system and chronic activation exerted adverse effect on the cardiovascular system followed by myocardial cell dysfunction and cell death. Peripheral vasoconstriction, myocardial hypertrophy and fibrosis, induced tachycardia and provoked arrhythmias.

Observations in animals and humans had implicated that enhanced cardiac sympathetic or decreased vagal influences were contributors to ventricular dysarrhythmias. (Vaisanen *et al.*, 2005)

2.3. SIGNIFICANCE OF ARRHYTHMIAS

Arrhythmias caused low cardiac output by either decreasing the stroke volume (tachyarrhythmias) or slowing heart rate (bradyarrhythmias). (Leib and Monroe, 1997)

Abbott (1998) reported that sinus bradycardia in dogs with clinical and radiographic findings characteristic of CHF suggested sinus node disease. The

author also added that CHF was usually accompanied by tachycardia and the rhythm was regular unless pathologic arrhythmias were present

Keating and Sanguinetti (2001) stated that cardiac arrhythmias were one of the leading causes of morbidity and mortality in man and in most cases, it was assumed that the underlying cause of a sudden death was ventricular tachyarrhythmia.

Goodwin (2001) reported that many cardiac arrhythmias were benign and clinically insignificant and required no specific therapy while some might cause severe clinical signs or progressed into malignant arrhythmias leading to cardiac arrest and sudden death.

Some arrhythmias led to haemodynamic compromise or sudden death especially in animals with underlying cardiac disease. Sinus tachycardia and atrial or ventricular tachyarrhythmias were common with cardiac tamponade associated with pericardial effusion. (Ware, 2001)

Martin (2002 b) observed that abnormalities in the conduction system of heart were associated with faults either in the generation of the impulse from the sinoatrial node or in the abnormalities in conduction through specialized tissues such as the atrioventricular node, bundle of His or the Purkinje system.

Hanna *et al.* (2004) observed that there were qualitative and quantitative differences in atrial versus ventricular remodeling in experimental ventricular tachypacing induced CHF.

Ristic (2004) stated that it was unusual to have clinically significant cardiac disease without an auscultable abnormality like murmur or arrhythmia.

The origin and mechanism of arrhythmia were of considerable significance, since knowledge of these might provide a basis for successful therapy. (Bhatt *et al.*, 2005)

2.4. OCCURRENCE

Brownlie and Cobb (1999) studied the electrocardiography and echocardiography of 39 Irish wolf hounds with cardiac failure and reported that eighteen dogs (46 percent) had atrial fibrillation .Other ECG abnormalities included ventricular and supraventricular premature contractions, first and second degree atrioventricular block, P mitrale, left anterior fascicular block and right bundle branch block.

Medical records and electrocardiographic rhythm strips of 51 dogs and cats with history of syncope, episodic collapse or intermittent weakness revealed a specific arrhythmia as the cause of clinical signs in 18 of the (35 percent) 51 animals.(Bright and Cali, 2000)

Meurs *et al.* (2001) evaluated 24 hour ambulatory ECG of 50 healthy dogs and concluded that 16 dogs had between one and 24 VPC; 34 had no VPC and the presence of numerous or sequential VPC might be suggestive of cardiac or systemic disease.

Chetboul *et al.* (2004) observed several episodes of unifocal ventricular tachycardia on ECG of a one year old female Great Dane which had no history of clinical signs related to respiratory or cardiovascular system.

Ninety-four boxers with arrhythmogenic right ventricular cardiomyopathy were selected and severity was estimated by use of 24 hour ambulatory ECG, signal averaged electrocardiography and echocardiography. Dogs were grouped into four categories based on VPC frequency as group one = < 10 VPCs / 24 hour; group two = 10 to 100 VPC / 24 hour; group three = 101 to 1,000 VPC s / 24 hour and group four = >1,000 VPCS / 24 hour. The percentage of affected dogs in group one, two, three, and four were 52, 18, 11 and 18 respectively (Spier and Meurs, 2004).

2.5. SIGNALMENT

Lombard (1984) studied the clinical signs and electrocardiogram of 12 dogs with dilated cardiomyopathy and congestive heart failure. The author concluded that there was a high prevalence in males (11 dogs).Among breeds, Dobermann (seven dogs) had high prevalence and seven dogs had atrial fibrillation.

Thomas (1987) observed a high incidence of chronic congestive heart failure in male English Cocker Spaniels.

Hamlin (1990) reported that ventricular arrhythmias were more common in geriatric dogs than in young ones. Geriatric dogs particularly Miniature Schnauzers, Cocker Spaniels and Dachshunds had high prevalence of weakness or fainting caused by short periods of cardiac arrest.

Ambulatory ECG obtained from 113 male and 115 female of clinically normal Beagles revealed bradycardia in 27 of 44 males (61.4 percent) and 18 of 46 females (39.1 percent). Sinus pause was identified in 33 of 44 males (75 percent) and 30 of 46 females (65.2 percent).Ventricular ectopic complexes were detected in 18.8 to 26.1 percent animals; second degree atrioventricular block was observed in 6 of 69 males (8.7 percent) and 14 of 69 females (20.3 percent); supraventricular escape complex occurred in 2 of 69 females (2.9 percent) and multiple types of abnormal complexes were observed in 2 of 69 males (2.9 percent) and 6 of 69 females(8.7 percent). (Ulloa *et al.*, 1995)

Jacobs (1996) reported that 57 percent of dogs with idiopathic dilated cardiomyopathy were Dobermann Pinschers and other frequently affected breeds included Boxers, Great Danes, Saint Bernards, German Shepherds and Retrievers.The author also added that males were more frequently affected than females and could be diagnosed most commonly in middle aged dogs.

Leib and Monroe (1997) stated that German Shepherds had an inherited problem with ventricular arrhythmias and sudden cardiac death. Brachycephalic

breeds of dogs often had upper respiratory abnormalities that might cause bradyarrhythmias such as sinus bradycardia or second degree heart block.

Abbott (1998) stated that dilated cardiomyopathy was most often seen in middle aged or older large and giant breed dogs such as Dobermann Pinschers, Boxers and Great Danes

Paslawska (1998) studied the electrocardiographic features of clinically healthy dogs representing nine selected purpose breed dogs and revealed that the most significant factor influencing the electrocardiographic curve morphology was the dog breed; affiliation to purpose group and age were influenced only some of ECG curve components and effects of animal sex and size on the ECG parameters were not found.

Brownlie and Cobb (1999) conducted electrocardiographic and echocardiographic examinations of 39 Irish Wolfhounds with cardiac failure and found that the mean age at which CHF diagnosed was 77 months in males and 86 months in females. The mean age at which atrial fibrillation (AF) first detected was 45 months in males and 59 months in females and the mean time for the first detection of atrial fibrillation to congestive heart failure was 27 months in males and 24 months in females.

McEwan (2000) observed that cardiac arrhythmias such as atrial fibrillation or ventricular ectopy associated with dilated cardiomyopathy were more prevalent in certain breeds like Dobermanns and Boxers.

2.6. GENERAL CLINICAL EXAMINATION

Fisher (1967 b) stated that auscultation was the most important part of cardiological examination and diagnosis with reasonable certainity of some arrhythmias was possible by auscultation.

Bolton (1975) reported that any animal that showed vague signs of weakness, fatigue, loss of stamina, seizures, coma or collapse should be screened for the presence of an arrhythmia.

Lombard and Goldschmidt (1980) reported lethargy and exercise intolerance in a one year old male Irish setter which had congestive heart failure secondary to fibroma of the right atrium.

Reason for weakness in congestive heart failure was reduced perfusion to skeletal muscles caused by poor myocardial function, persistent tachyarrhythmias or bradyarrhythmias, outflow obstructions, pericardial effusion or hypotension (Leib and Monroe, 1997)

Abbott (1998) stated that body temperature and pulse quality was usually normal in animals with CHF and pathologic arrhythmia resulted in varying pulse strengths and rates. The author also added that in many patients with dilated cardiomyopathy, a murmur of functional mitral valve insufficiency was present. Heart sound became less intensive in animals with pericardial effusion.

Strickland and Goodwin (1998) reported that clinical signs such as exercise intolerance, coughing and tachypnea were frequently associated with heart failure and it occurred secondary to either decrease in stroke volume or abnormal heart rate or rhythms.

Cardiac cachexia, the muscle wasting associated with congestive heart failure was a common disorder of patients with cardiac disease and it was present in more than 50 percent of dogs with dilated cardiomyopathy. (Roudebush and Freeman, 2000)

Most common cardiac cause of dyspnea was left heart failure causing pulmonary oedema. (Goodwin, 2001)

Cardiac causes of weakness or syncope included bradyarrhythmias and tachyarrhythmias which failed to generate adequate cardiac output. (DeFransesco, 2004)

Physical examination of 54 dogs with pulmonary hypertension revealed respiratory crackles, wheezes or rubs in eight dogs. (Pyle *et al.*, 2004)

Ristic (2004) reported that pulse deficits associated with arrhythmias might be clinically significant.

Hyun (2005) reported several episodes of syncope, dyspnoea and tachypnoea in a Cavalier King Charles Spaniel suffering from mitral valve regurgitation.

Schrope and Kelch (2006) studied the clinical signs of 124 dogs with high grade second or third degree atrioventricular blocks and reported that weakness, lethargy, exercise intolerance and syncope were the most common clinical signs.

2.7. ELECTROCARDIOGRAPHY

Fuentes (1998) reported that P wave represented atrial depolarization; PR interval - conduction through A-V node; QRS complex- ventricular depolarization; ST segment - period between ventricular depolarization and repolarization and T wave represented repolarization of ventricles.

ECG was a graphic record of the voltage produced by cardiac muscle cells during depolarization and repolarization plotted against time. (Ettinger *et al.*, 2000)

Goodwin (2001) reported that electrocardiogram was a basic and valuable diagnostic test in veterinary medicine.

ECG interpretation was based on a good understanding of the electrical activity of the heart which related directly to the complexes seen on an ECG. Results of any diagnostic tool such as electrocardiography were always evaluated with a sound appreciation of the history and clinical findings. (Martin, 2002 a)

Ristic (2004) stated that main indication for performing electrocardiography was to categorize arrhythmias noted during the clinical examination.

2.7. 1. IMPORTANCE OF ELECTROCARDIOGRPHY

Littlewort (1967) reported that electrocardiogram is a valuable aid in detecting arrhythmias, abnormalities of conduction, variations in the normal distribution of cardiac muscle within the thoracic cavity and disturbances of myocardial metabolism.

An electrocardiogram was quickly and easily made. Interpretation and information obtained were reliable. It was also stated that electrocardiography was useful in cardiac diseases; differentiation of non specific diseases that caused fatigue, fever, lethargy, collapse or seizures; monitoring during anaesthesia and surgery; for documentation of data and for sharing information and seeking consultation service. (Bolton, 1975)

Goodwin and Lombard (1990) stated that ECG was an integral part of the diagnostic evaluation of puppies that were suspected to have congenital heart disease.

McEWan (2000) stated that electrocardiography was essential in any dog with an arrhythmia to document the condition and also to find out if any treatment was indicated.

DeFransesco (2004) reported that ECG provided a rapid and relatively cost effective method of determining heart rate and rhythm and was a valuable monitoring tool. It was also stated that the ECG was most useful in determining the type of arrhythmia discovered on physical examination and provided information regarding cardiac chamber enlargement, state of the conduction system and the health of the myocardium.

2.7.2. Limitations of electrocardiography

Electrocardiogram provided information only on the spread of depolarization and repolarization through the heart muscle and no direct information concerning valvular disease and force of contraction. (Littlewort, 1967)

Electrocardiogram has its own limitations and the electrocardiographic findings must always be correlated with the other findings. (Bolton, 1975)

Ettinger *et al.* (2000) reported that electrocardiographic parameters for dogs or cats based on breed, body size, age and sex were not established and ECG was not an absolute indicator of normalcy or disease

Ware (2001) stated that while a routine electrocardiogram documented arrhythmias, presented during recording, it provided only a glimpse of the cardiac rhythms occurring over the course of a day and potentially critical arrhythmias could easily be missed.

2.7.3. Artifacts observed in ECG

Artifacts were abnormal deflections reproduced in an ECG recording which were not associated with the electrical activity of the heart viz-electrical interference, muscle tremor artifact and movement artifact. (Martin, 2002 c)

Sixty cycle interference, respiratory artifacts, nervous trembling, shaking and struggling were the common artifacts in ECG. (Bolton, 1975)

2.7.4. Procedure for taking Electrocardiogram

Littlewort (1967) reported that through electrodes and associated wires, electric currents arising in the heart were transmitted through the tissues of the body to skin and were conducted to the voltmeter of the electrocardiograph where they were measured and recorded.

Bolton (1975) stated that the clips and the skin were moistened to establish good clip to skin contact and to prevent electrical interference and it was preferable to place the dog on an insulated table.

Goodwin and Lombard (1990) reported that for electrocardiographic recording, attach the front limb leads (labeled RA and LA)to the skin, just distal to the elbow and rear limb leads (LL and RL) to the cranial aspect of the stifle over the patellar ligament. The exploring electrode (C) was used to obtain chest lead recordings and the locations for the various chest leads were the 6^{th} left intercostal space near the edge of the sternum (CV6LL or V_1), the 6^{th} left inter costal space at the costochondral junction (CV6LU or V_2), the 5^{th} right inter costal space near the edge of the sternum (CV5RL or V_4) and the skin over the dorsal spinous process of the 7^{th} thoracic vertebra (V_{10} or V_3).Record four to six complexes for each lead and if an arrhythmia was detected, run the rhythm strip for one to two minutes longer to check for additional abnormalities.

The electrical ground wire (RL) connected the patient to the ground and in lead I, LA was the positive terminal and RA was the negative terminal; in lead II, LL was positive and LA was the negative and in lead III, LL was positive and LA was the negative terminal and in theory the bipolar limb leads formed an equilateral triangle (Einthoven's triangle) with the origin of cardiac vector, the zero point of the electrical field, at the centre point of the triangle. (Ettinger *et al.*, 2000)

Lead II was the most popular lead used for rhythm analysis in dogs and cats but it was advisable to record all standard limb and chest leads and to examine every lead especially when the rhythm was difficult to identify in lead II. (Rishniw and Thomas, 2000)

Goodwin (2001) reported that the electrocardiogram should be recorded in a quiet area free of distraction and the patient should be placed in right lateral recumbency because electrocardiographic reference values were obtained from animals in right lateral recumbency.

Martin (2002 a) reported that for normal heart rate, set the paper speed at 25mm/second and for a fast heart rate set the paper speed at 50mm/ second and calibration was usually set at one cm/ mV. The author also added that chemical restraint should be avoided prior to recording an ECG.

2.7. 5. Determination of heart rate

Bolton (1975) reported that the normal heart rate in the dog was 70 to 160 beats per minute and if the heart rate was above normal, the dog had tachycardia and if below normal it was called bradycardia.

Heart rate could be obtained by multiplying the number of QRS complexes in a three second interval by 20 or in a six second interval by 10 or if it was regular rhythm divide, 3000 (if the paper speed was 50mm/sec) or 1,500 (if the paper speed was 25mm/sec) by the number of millimeters between adjacent R waves. (Goodwin and Lombard, 1990)

The heart rate variability might provide valuable information to the clinician when assessing the severity of mitral regurgitation caused by chronic valvular disease. (Haggstrom *et al.*, 1996)

2.7.6. Determination of Mean Electrical Axis (MEA)

The mean electrical axis of ventricular depolarization in the frontal plane in most normal dogs lies between $^{+}40$ and $^{+}100$ degrees and variations within normal are not significant where as major shifts to the right ($>^{+}90$ degrees) or the left ($<$ $^{+}40$ degrees) are relevant. (Ettinger *et al.*, 2000)

According to Goodwin (2001) MEA could be determined by following methods 1) Using the six standard leads, if there was a lead with isoelectric QRS complexes, the MEA equated to the lead on the Bailey axis perpendicular to the isoelectric lead. 2) By plotting the net amplitude of a lead I QRS complex and the interaction would provide the vector equal to the MEA.

Martin (2002 a) reported that measurement of mean electrical axis (MEA) was of limited value in small animals partly because the vector in the front plane was less representative of the true direction of the vector in three dimensions than it was in humans. The author also added that MEA was mainly used to assist in the assessment of ventricular enlargement and in the recognition of intraventricular conduction defects.

2.7.7. Determination of heart rhythm

Bolton (1975) reported that the normal rhythm in the dog was sinus in origin and if there was a perfectly regular rhythm it was called normal sinus rhythm.

Goodwin and Lombard (1990) reported that in a normal sinus rhythm each QRS complex was preceded by a P wave and each P wave was followed by a QRS complex.

Goodwin (2001) stated that cardiac rhythm was highly influenced by changes in autonomic tone in which elevated sympathetic tone would increase the rate of SA nodal discharge and elevated vagal tone would decrease the rate of SA nodal discharge.

2.7.8. Normal canine electrocardiogram

Bernal *et al.* (1995) studied lead II electrocardiogram of 70 Mastin Espanol dogs, aged between one day and three years. The mean P wave duration ranged between 0.022 and 0.038 seconds and it increased with age and body weight. PR interval was shorter in animals less than one month of age (0.058 to 0.060 seconds) and longer in adult animals (0.140 seconds). Deep S waves were observed in all one-day-old animals. Mean value of Q waves ranged between 0.27 and 0.43 mv. The amplitude of the R wave increased greatly after the first week of life with mean values of 0.73± 0.065 mv and QRS duration also increased with age. (0.029 to 0.055 seconds)

Paslawska (1998) made electrocardiographic observation of 487 clinically healthy dogs which were subdivided into defensive, hunting and draught dogs. The highest average P amplitude was noted in hunting dogs (0.235±0.0067). In five out of 487 of examined dogs P pulmonale was noted, when the amplitude in outlets II and III exceeded 0.4 mv and the duration was within standard limits. Longer PQ section was noted in adult dogs (0.0596±0.0012 seconds) than the young dog group (0.0578±0.00132 seconds).the hunting dog group had shorter PQ interval (0.1516±0.00132 seconds) than the defensive dog groups (0.0628±0.00174 seconds). The average amplitude of QRS complex was 1.562 mv and it was not influenced by animal sex, age and body size.

The P wave should be positive in lead II and aVF, isoelectric or positive in lead I and it should be negative in leads III, aVR, aVL, and V_{10}. The maximum normal amplitude and duration of P wave in lead II was 0.4 mv and 0.04 seconds respectively. The duration of the PR interval varied inversely with the heart rate and it was usually 0.06 to 0.14 second in dogs. The upper normal amplitude of the canine R wave was 2.5 mv and above 3 mv was abnormal. Abnormal deviations of the ST segment were depression of 0.2 mv or elevation of 0.15 mv in leads II and III and if the amplitude of the T wave was greater than 25 percent of that of the R wave, hypoxia and electrolyte changes might be suspected. (Ettinger *et al.*, 2000)

2.8. EVALUATION OF ARRHYTHMIAS USING ELECTROCARDIOGRAM

2.8.1 Sino-atrial arrhythmias

Fisher (1967 a) stated that sinus arrhythmia was a normal regular irregularity of rhythm observed in healthy dogs associated with respiration.

According to Bolton (1975) sinoatrial arrhythmias included sinus arrhythmia, sinus arrest, wandering pacemaker, sinus tachycardia and sinus bradycardia and all were normal variations of the canine cardiac rhythm. It was also stated that these arrhythmias were of no clinical significance.

Sinus tachycardia was a sinus rhythm occurring at an elevated rate and sinus bradycardia was the one in which the heart rate was abnormally low. (Ettinger *et al.*, 2000)

Goodwin (2001) reported that in sinus arrest there was a pause in the rhythm and the pause was at least twice the preceding R-R interval.

Wandering pacemaker occurred as a result of the dominant pacemaker shifting from the sinoatrial node to other pacemaker cells within the atria and ECG features included variation in P wave morphology and it could be positive, negative, biphasic or even isoelectric. (Martin, 2002 a)

2.8.2. Supra ventricular arrhythmias

2.8.2.1 Supra ventricular premature complexes

An atrial premature complex was consisted of a P wave that differed in configuration from that of a sinus beat however the QRS should look like that of a sinus complex because conduction from the A-V node down was the same as in sinus complex. (Leib and Monroe, 1997)

Goodwin (2001) reported that atrial premature complexes were usually seen in association with atrial enlargement, degenerative valvular diseases, congenital heart diseases, cardiomyopathy, hypoxia, atrial neoplasia and chronic obstructive pulmonary disease.

2.8.2.2. Supraventricular tachycardia

Bolton (1975) stated that atrial tachycardia should be the prime suspect in older dogs which had mitral insufficiency with syncopal episodes.

Ettinger *et al.* (2000) reported that an atrial tachycardia was a series of atrial extra systoles occurring at a rate greater than the sinus rhythm. In paroxysmal atrial tachycardia ECG showed a burst of atrial extra systoles occurring at a rate greater than the sinus rhythm; while in sustained atrial tachycardia P waves might

be buried within the previous QRS complexes and were difficult or impossible to identify.

Durham (2005) reported chronic atrial tachycardia in an eight-month old spayed Beagle with a history of spontaneous collapse and loss of consciousness and it was diagnosed as a case of early congestive heart failure.

2.8.2.3. Atrial flutter

In dogs atrial flutter was an unstable rhythm occurred intermittently and ECG features included saw tooth- shaped rapid undulations (F waves) instead of P waves with cycle length usually ranged from 300 to 600 beats / min. (Moise, 1988)

D'Souza *et al.* (1990) reported a case of leptospirosis with atrial flutter in a 65-year-old man.

Strickland (1998) stated that atrial flutter was commonly encountered in dogs with chronic heart failure secondary to degenerative valve disease or idiopathic dilated cardiomyopathy.

2.8.2.4. Atrial fibrillation

Jacobs (1996) stated that atrial fibrillation was a common sustained rhythm disturbance associated with idiopathic DCM of dog. Heart sounds in atrial fibrillation were described as chaotic as they were rapid, irregular and of variable intensity.

Coronary artery disease was a significant risk factor for atrial fibrillation in man. (Sinno *et al.*, 2003)

Cote *et al.* (2004) reported that because of absence of P waves it was not possible to confirm the presence or absence of atrio-ventricular dissociation and therefore atrial fibrillation might have been mistaken for other arrhythmias such as ventricular ectopy and supra-ventricular ectopy with aberrancy.

DeFransesco (2004) stated that atrial fibrillation was common in large breed dogs and typically associated with dilated cardiomyopathy or advanced valvular heart disease. ECG revealed, no P waves because of unrecognized atrial activity, an irregular R-R interval with normal QRS complexes and slight fluctuations in the baseline representing fibrillation waves.

Atrial fibrillation was the most common arrhythmia that required treatment in veterinary medicine. (Moise, 2005)

2.8.3. Ventricular arrhythmias

Ventricular arrhythmias were the most common findings in Boxers with dilated cardiomyopathy and might be the first sign of cardiomyopathy in Dobermann Pinschers. (Leib and Monroe, 1997)

2.8.3.1. Ventricular premature complexes

According to Ulloa *et al.* (1995) a ventricular premature complex was a premature QRS complex originating at or distal to the bundle of His and not associated with a P wave. Complexes originating near the bundle of His appeared normal and those arising more distally were wide and bizarre in shape.

Causes for ventricular premature complexes were numerous viz-structural heart disease, boxer cardiomyopathy, hypoxia, anemia, uremia, gastric-dilatation-volvulus, myocarditis and drug induced. (Goodwin, 2001)

The T wave of a ventricular premature complex was often large and opposite in direction to the QRS complex and when a ventricular premature complex was so premature, it was superimposed on the T wave of the preceding complex such that the ventricles were depolarized before they had completely repolarized from the preceding contraction and this was termed R on T. (Martin, 2002 a)

2.8.3.2. Ventricular tachycardia

Ventricular tachycardia was a series of three or more ventricular extra systoles occurring at a high rate and it might be continuous (sustained) or intermittent (paroxysmal). Typical appearance was a series of QRS complexes that were widened, associated with giant T waves, not linked to P waves and might include capture beats and fusion beats. (Ettinger *et al*, 2000)

Bosswood (2001) stated that rapid periods of ventricular tachycardia even in the absence of outward clinical signs might indicate an increased risk of development of ventricular fibrillation.

2.8.3.3. Ventricular fibrillation

Littlewort (1967) reported that the chief electrocardiographic feature of ventricular fibrillation was an entire absence of recognizable P, QRS and T waves and presence of only irregularly shaped waves of varying amplitude and duration.

Ventricular fibrillation was the most serious consequences of ventricular myocardial irritability and it was resulted from uncontrollable myocarditis or aortic stenosis. Clinical signs were acute collapse with or without seizures followed by death. (Bolton, 1975)

2.8.4. Atrio-ventricular dissociation

In atrio-ventricular dissociation, the atria and ventricles were depolarized by separate, independent foci due to disturbed atrio-ventricular conduction or depressed sino-atrial nodal function and ECG features showed independent P wave and QRS complexes appearing to catch up on the P waves. (Martin, 2002 a)

Kadar and Rush (2004) reported a case of atrio ventricular dissociation in a 13 year old male Dobermann Pinscher diagnosed with hypercalcemia of malignancy associated with adenocarcinoma of anal sacs.

2.8.5. Conduction disturbances

2.8.5.1. Atrio-ventricular block

Littlewort (1967) reported that in atrio- ventricular block, the impulse was delayed or interrupted between the atria and the ventricles and it included; a) first degree atrio-ventricular block in which the interval between atrial and ventricular contraction was unduly long and manifested in the electrocardiogram as a prolonged P-R interval. b) second degree atrio-ventricular block in which occasional impulse failed to reach the ventricles because it was blocked from transmission through the A-V node and in this one or more normal P waves, unaccompanied by any QRS complex or T wave and c) third degree or complete A-V block in which impulses were completely blocked from the ventricles which contracted independently at a slow intrinsic rate and it was represented in the electrocardiogram as regularly repeated P waves, entirely dissociated from much less frequent QRS complexes.

Abbott and king (1993) reported third degree atrio-ventricular block in a dog suffering from non-penetrating chest trauma and it was found that syncope was the most common clinical sign. It was also added that other causes for the formation of third degree atrio-ventricular block included digitalis toxicities, fibrosis, neoplasia, infective endocarditis of the aortic valve, hypertrophic cardiomyopathy and repeated cardiac punctures.

2.8.5.2. Bundle branch blocks

Bundle branch blocks were interruption of conduction involving one or more of the ventricular branches of bundle of His and blocks might be functional or structural. (Ettinger *et al.*, 2000)

2. 8.5.2.1. Left Bundle Branch Block (LBBB)

Goodwin (2001) stated that left bundle branch block (LBBB) occurred due to a delay in conduction or block in both the posterior and the anterior fascicles of

the left bundle and it was a marker of significant heart disease. Electrocardiographic features included prolonged QRS complexes (> 0.08 second) which were wide and positive in leads I, II, III and aVF.

Vernooy *et al.* (2004) studied left bundle branch block in eight dogs and it was observed that LBBB immediately and persistently induced mechanical asynchrony and in the long run it led to left ventricular dilatation and asymmetric hypertrophy and it appeared to be a greater threat to long term prognosis than the reduced septal blood flow.

2.8.5.2.2. Right Bundle Branch Block (RBBB)

Bolton (1975) reported that electrocardiogram recorded from a 10 year old beagle dog revealed complete right bundle branch block which had normal sinus rhythm and normal P waves but the QRS complexes were prolonged which had large wide S waves in lead I, II, III, aVF and in lead V_{10}.

Martin (2002 c) stated that in RBBB, depolarization of the left ventricle occurred normally but depolarization of the right ventricular mass occurred through the myocardial cell tissue instead of right bundle branch, which resulted in a very prolonged QRS complex.

2.8.6. Escape rhythms

2.8.6.1 Junctional escape beats

When not activated by atrial depolarization, the junctional A-V nodal area might spontaneously discharge and electrocardiographic features included inverted P wave, occurring before, during or just after QRS complex; and it usually occurred after a significant pause in the sinus rhythm. (Goodwin, 2001)

2.8.6.2. Ventricular escape beats

Ventricular escape beats might occur singly after long pauses in the rhythm and electrocardiographic features showed normal or slow heart rate, normal P

waves when they occurred but the QRS complexes might be bizarre. (Bolton, 1975)

2.8.7. Atrial stand still

In atrial stand still, there was absence of any atrial activity due to failure of atrial muscle depolarization and impulses were conducted from the sino- atrial node by inter nodal pathways to the atrio-ventricular node (sino- atrial rhythm) and hyperkalemia was the most common metabolic cause of atrial stand still in dogs. (Martin, 2002 b)

2.8.8. Wolf-Parkinson White syndrome (WPW)

According to Ettinger *et al.* (2000) WPW produced a shortened PR interval (<0.08 second) and a notching of the QRS complexes by delta wave indicating the premature depolarization of part of the ventricles.

Wolf Parkinson-White syndrome (WPW) was the commonest form of ventricular pre-excitation and characterised by the presence of accessory pathway between the atria and the ventricles that provided an alternative route for ventricular activation. (Keating *et al.* 2003)

2.8.9. Sick-Sinus Syndrome (SSS)

Disorders of the sinus node were known as sinus node dysfunction or sick sinus syndrome (SSS) and they were resulted from inadequate impulse formation or conduction. (Moise, 1988)

Goodwin (2001) reported that sick-sinus syndrome was a progressive heart disease characterized by a variety of arrhythmias including sinus bradycardia, sinus arrest, paroxysmal atrial tachycardia, intermittent AV nodal block and lack of ventricular escape complex. It was also stated that most cases were presented with a history of intermittent weakness and collapse.

2.9. RADIOGRAPHY

Wyburn and Lawson (1967) reported that the left ventricle was the cardiac chamber most commonly involved in hypertrophy or dilatation and on the lateral plane; the caudal border of the heart became bulged so that it overlied the diaphragmatic shadow to an increased extent.

Thomas (1987) studied features of congestive cardiomyopathy in eight Cocker Spaniels and the appearance of the cardiac silhouettes on thoracic radiographs indicated a marked heart enlargement, typically of a biventricular nature with left atrial enlargement and tracheal elevation in all cases.

Goodwin and Lombard (1990) reported that thoracic radiographs provided essential information about overall cardiac size, shape, enlargement of individual cardiac chambers and the presence of aneurismal or post stenotic dilatations.

According to McEwan (2000) in DCM radiographic changes affecting the cardiac silhouette included left atrial enlargement alone, left atrial and left ventricular enlargement, right sided enlargement or generalized cardiomyopathy.

According to Ware (2001), massive pericardial effusion resulted in the classic globoid cardiac shadow seen on both lateral and dorsoventral radiographs and other radiographic findings of cardiac tamponade included pleural effusion, caudal venacava distension, hepatomegaly and ascites.

Overall radiographic signs of a mitral valve prolapse in Cavalier King Charles Spaniel implied a severe left atrial and ventricular enlargement with pulmonary oedema which might be caused by congestive heart failure. (Hyun, 2005)

2.10. ECHOCARDIOGRAPHY

In a review of case records of pericardial effusion in three dogs, the abnormalities found in M-mode echocardiography were, echo-free separation of the visceral and parietal pericardium, dampening of parietal pericardial motion,

exaggerated or paradoxic motion of intracardiac structures and thickened epicardial echoes. (Bonagura and Pipers, 1981)

M-mode echocardiography proved to be a useful technique in recognizing left heart dilatation and poor contractile function in dogs with dilated cardiomyopathy and congestive heart failure. (Lombard, 1984)

A left apical four chamber view of a two year old female mixed breed dog that developed third degree atrio-ventricular block following non-penetrating chest trauma revealed the presence of an echogenic mass arising from the caudoventral aspect of the atrial septum. (Abbott and King, 1993)

According to Abbott (1998) echocardiographic examination was a sensitive mean for detecting chamber enlargement, structural valvular abnormalities, pericardial effusions and also the most practical method for evaluating systemic ventricular performance.

Echocardiography is the method of choice for the confirmative diagnosis of DCM and for excluding other acquired or congenital heart disease. (McEwan, 2000)

Sisson *et al.* (2000) reported that short axis end-systolic and end- diastolic dimensions of the left ventricle, indexed for body weight were usually much larger in dogs with DCM than normal dogs.

Echocardiography was highly sensitive for detecting even small quantity of pericardial effusions and might document an underlying neoplasm and other cardiac condition. (Ware, 2001)

Thirty four adult healthy dogs (16 males and 18 females) were studied by both two-dimensional and M-mode echocardiography and it was found that many values provided good indicators for evaluation of cardiac function and they were ejection fraction, fractional shortening, fractional thickness, systolic time

intervals, left ventricular diastolic dimension ratio and stroke volume indices. (Su *et al.*, 2003)

According to Ristic (2004), a hypoechoic region surrounding the heart provided definitive diagnosis of pericardial effusion.

2.11. CILINICO-PATHOLOGY

The results of a complete blood count and a serum chemistry profile were often normal in patients with CHF although severe failure might be complicated by prerenal azotemia, hyponatremia and a stress leukogram. (Abbott, 1998)

Haematobiochemical values were not of much useful for the diagnosis of heart disease however, they could be helpful to investigate potential concurrent disease. (Ristic, 2004)

2.11.1. Haematology

In healthy dogs, normal haemoglobin concentration (Hb), volume of packed red cells (VPRC) and total leucocytes count (TLC) ranged from 12 to 18 gram per cent, 37 to 55 per cent and 6,000 to 17,000 per cu.mm respectively. Normal differential count (DC) include 60 to 77 per cent neutrophils, 12 to 30 per cent lymphocytes, three to ten per cent monocytes and two to ten per cent eosinophils. (Schalm *et al.*, 1975)

Thomas *et al.* (1984) reviewed the clinico-pathological features of constrictive pericardial disease in 13 dogs and observed that two had mild non regenerative anemia (PCV<37 per cent) and three had slight leukocytosis (WBC between 18,000 and 20,000 per cu.mm) with neutrophilia.

Wilbanks (1992) reported that the haematological parameters (WBC, RBC, Hb, PCV and DLC) were within the reference change in a 13 year old spayed female Schnauzer presented with complaints of lethargy, fainting, cyanosis and

abdominal enlargement and it was diagnosed as a case of third degree heart block.

Ristic (2004) stated that many cardiac cases would have a stress leukogram causing considerable elevations in mature neutrophil counts.

2.11.2. Serum biochemistry

Gavaghan and Kittleson (1997) reported the normal value for sodium and potassium in dog as 145-154 mmol/l and 4.1-5.3 mmol/l respectively

Normal serum creatinine concentration in dogs ranged from 0.3 to 1.3 mg/dl. (DiBartola, 2000)

According to Wortinger (2001) normal serum calcium levels ranged from 8.9 to 12.2 mg/dl in dogs.

According to Forrester and Brandt (1994) hyperkalemia was a life threatening abnormality in acute renal failure and the electrocardiographic changes associated with hyperkalemia included prolonged PR intervals, spiked T waves, missing P waves and widened QRS complexes. It was also stated that immediate treatment was indicated to lower serum potassium concentrations in patients with severe cardiac arrhythmias or serum potassium concentrations > 8 mEq/l.

According to Jacobs (1996), hyponatremia and hypoprotenemia might develop in DCM and usually associated with severe congestive heart failure.

Aroch *et al.* (1998) reported a case of hypocalcaemia associated with hypomagnesaemia in a lactating bitch and ECG revealed a heart rate of 120 beats / min, low amplitude P waves (0.1 to 0.2 mV), tall spiked T waves (0.9 mV) in lead II, III, aVL and aVF. It was also added that extreme hypocalcaemia could cause low myocardial contractility and low cardiac output.

Wyatt *et al.* (1998) compared the values of CK-MB concentration in eight dogs without heart disease and 11 dogs with heart disease including mitral valve

insufficiency, atrial fibrillation, supraventricular and ventricular ectopy, dilated cardiomyopathy, heart worm disease and tricuspid insufficiency. It was found that the median and mean CK-MB concentrations for the non-cardiac diseased groups were 7.7 pmol/l and 20.1 pmol/l where as for the dogs with cardiac disease they were 4.8 pmol/l and 14.7 pmol/l respectively and they were not significantly different. In conclusion CK-MB concentration was not a suitable indicator of cardiac failure in dogs.

Increased serum potassium concentration led to bradyarrhythmias that ranged from simple sinus bradycardia (serum k^+ 6 to 7 mEq/l) to a sino-ventricular rhythm (serum $k^+ \geq 7$ to 8 mEq/l) (Knight, 2000)

Sisson *et al.* (2000) stated that in DCM serum electrolyte and protein concentrations were often in the normal range but in some cases there might be mild hypoprotenemia and hyponatremia and modestly elevated serum liver enzymes ,bile acids, increased serum urea and creatinine concentrations.

Acid base disturbances and fluid and electrolyte imbalances resulted in the development of rhythm disturbances in which hyperkalemia led to atrial stand still and hypocalcaemia predisposed to ventricular rhythm disturbances. (Bosswood, 2001)

Ravindran (2001) found out that electrocardiographic signs of electrolyte imbalance due to renal disease were tall peaked T waves in the chest leads and prolonged Q-T interval indicated hyperkalemia and hypocalcaemia respectively.

Hypokalemia could predispose the patients to ventricular arrhythmias and many antiarrhythmic drugs were ineffective in the presence of hypokalemia. (Ware, 2001)

Materials and methods

3. MATERIALS AND METHODS

The study was conducted in the Department of Clinical Medicine, College of Veterinary and Animal Sciences, Mannuthy during the period of May 2005 to June 2006.

Dogs brought to the Veterinary College Hospital, Mannuthy and University Veterinary Hospital, Kokkalai with signs of cardiac problems like lethargy, weakness, dyspnoea, cough, syncope and exercise intolerance were used for the study. They were screened for cardiac involvement by detailed clinical examination.

Standard electrocardiogram patterns were worked out from six healthy dogs.

Based on the clinical signs and electrocardiogram abnormalities, eight dogs having arrhythmia were selected and detailed examinations were carried out.

The types of arrhythmia were as follows;

1. Supraventricular tachycardia – two dogs
2. Ventricular premature complexes – four dogs
3. First degree atrio-ventricular block – one dog
4. Right bundle branch block – one dog

3.1 PARAMETERS STUDIED

1. Signalment and History.
2. Clinical examination.
3. Electrocardiography.
4. Radiography.
5. Echocardiography.
6. Clinical pathology.

A. Haematology

a. Volume of Packed Red Cells (VPRC) (per cent)

b. Haemoglobin (Hb) (g/dl)

c. Total Leucocyte Count (TLC) (per cu.mm)

d. Differential Count (DC) (per cent)

B. Serum biochemistry

a. Creatine kinase (CK) (U/L)

b. Serum sodium, potassium (mEq/l) and calcium (mg/dl)

c. Serum creatinine (mg/dl)

3.2. PROCEDURES ADOPTED

3.2.1. Clinical examination

Detailed history and results of clinical examination of eight selected dogs were recorded as per the method of Houston (2000), paying special attention to the cardiovascular system.

3.2.2. Electrocardiography

Electrocardiograms of the patients were recorded using BPL - CARDIART-6108® ECG machine. Three standard bipolar limb leads (I, II, III) and three augmented unipolar limb leads (aVR, aVL, aVF) were used for the study.

According to the procedure of Goodwin (2001) and Martin (2002a), animals were placed in right lateral recumbency and the electrodes were attached over the elbow and stifle joints of the forelimbs and hind limbs respectively after applying electrode jelly. Electrocardiograms were recorded in recorded in **BPL - CARDIART®** paper strips at 25 mm per second speed and 1 mv = 10 mm

Plate 1. ECG recording

sensitivity. The recorded ECG tracings were evaluated and were photographed later (PLATE 1).

3.2.3. Radiography

Lateral plain thoracic radiographs were taken for cardiac evaluation. Size of the X-ray film and radiographic factors varied depending upon the size and chest girth of the patients.

3.2.4. Echocardiography

Selected animals were subjected to ultrasound scanning of the heart using **L&T SYMPHONY® 4.0** scanner. Both two – dimensional and M – mode echocardiography in left and right parasternal view of the heart was obtained using 7.5 MHz transducer.

3.3. CLINICAL PATHOLOGY

3.3.1. Haematology

Clinical materials were collected on the day of admission. Five milliliter of whole blood was collected from saphenous or cephalic vein of the patient in dry glass vials with EDTA @ 1-2 mg per milliliter of blood as anticoagulant. (Benjamin, 1998) The values of haemogram of six healthy animals were used for the comparison with that of the diseased animals. The values of Hb, VPRC, TLC and DC of diseased and control animals were determined as per the method of Schalm *et al.* (1975).

3.3.2. Serum biochemistry

Blood samples (approximately 5 ml) were collected from saphenous or cephalic veins of each animal having arrhythmia and six control animals. Samples were taken in a clean, dry test tube for separating serum for biochemical analysis. Sera thus separated were stored at $^{-}20^{0}$C till further analysis. The mean serum

biochemical values of six apparently healthy animals were used for comparison with that of diseased.

Serum creatine kinase, creatinine, serum sodium, potassium and calcium were estimated. Serum creatine kinase, creatinine and calcium were estimated by spectrophotometry in Merck 200 spectrophotometer using commercially available kits. Serum sodium and potassium were estimated using SYSTRONICS 128® flame photometer.

Results

4. RESULTS

Dogs presented to the Veterinary College Hospital, Mannuthy and University Veterinary Hospital, Kokkalai with signs of cardiac problems formed the material for the study. All the cases were subjected to detailed clinical examination, electrocardiography, radiography, echocardiography and haemato-biochemical assays. According to the test results and findings, a total of 8 cases having arrhythmia were selected. The types of arrhythmia were as follows.

1. Supraventricular tachycardia – two dogs
2. Ventricular premature complexes – four dogs
3. First degree atrio-ventricular block – one dog
4. Right bundle branch block – one dog

4.1 PREVALENCE

Data on the occurrence of cardiac disease in dogs for a period of one year (2005- 2006) were collected from the records maintained at the University Veterinary Hospitals of Mannuthy and Kokkalai. Out of 11,230 canine patients, 106 (0.9 per cent) were brought with clinical signs like lethargy, dyspnoea, cough, syncope and exercise intolerance. Out of these 106 cases, 25 (23.6 per cent) were confirmed as cardiac diseases and of which eight (32.0 per cent) had arrhythmia. Cardiac diseases and arrhythmia were accounted for 0.22 per cent and 0.07 per cent of the total canine cases respectively.

4.2 SIGNALMENT

Signalment of 8 selected dogs having arrhythmia are given in Table 1.

4.2.1 Age

Age wise prevalence of arrhythmia revealed that it was more commonly observed in middle aged dogs. Dogs of seven to ten years of age (62.5 per cent) were more frequently affected followed by three to six year (25.0 per cent) and ten to thirteen year old dogs (12.5 per cent).

4.2.2. Sex

Sex wise prevalence of arrhythmia in dogs revealed that it was more commonly seen in males (75.0 per cent) than females (25.0 per cent).

4.2.3. Breed

The breed wise prevalence of arrhythmia indicated that Dobermann Pinschers were more prone to arrhythmia (37.5 per cent) followed by German Shepherd (25.0 per cent), Boxer (12.5 per cent), Cocker Spaniel (12.5 per cent) and Mongrel (12.5 per cent).

4.3. CONTROL GROUP

4.3.1. Electrocardiographic parameters

The mean ± SE values of various parameters like heart rate, P wave amplitude and duration, P-R interval, R wave amplitude, QRS duration, S-T segment and T wave amplitude are given in Table 2.(PLATE 2.)

4.3.2. Haematological parameters

The mean ± SE values various parameters like VPRC, Haemoglobin, TLC and DC are given in Table 3.

The mean ± SE values of VPRC, Haemoglobin and TLC were 36.5±2.8 per cent, 11.8±0.5 g per cent and 11,375.5±936.3 per cu.mm respectively and DC obtained were neutrophil = 70.4±1.5 per cent, Lymphocyte = 27.1 ± 1.1 per cent, monocytes = 1.3±0.4 and eosinophil = 1.0±0.5.

4.3.3. Serum biochemical parameters

The mean ± SE values of various parameters like creatine kinase, sodium, potassium , calcium and creatinine are given in table 4.

The mean ± SE values of creatine kinase, Sodium, Potassium, Calcium and Creatinine were 38.3 ± 4.7(U/L), 138.5 ± 1.2 (mEq/l), 4.1 ± 0.3 (mEq/l), 9.3 ± 0.5 (mg/dl), 0.7 ± 0.1(mg/dl) respectively.

4.4. DETAILS OF DIFFERENT TYPES OF ARRHYTHMIA

Types of arrhythmia occurred in dogs are given in Table 5.

4.4.1 Supraventricular tachycardia

Supraventricular tachycardia was observed in two out of eight dogs (25.0 per cent). The signalments of these dogs are presented in table 1 (case 1 and 2). Ages of the affected dogs were 9 year and 5 year and they belonged to Dobermann and German Shepherd breed. Both of them were males.

4.4.1.1 Clinical findings

Clinical parameters are given in Table 6.

Case 1 was presented with a history of exercise intolerance, anorexia and occasional cough. The rectal temperature was 102.2°F and mucous membrane was pale. Vaccination and deworming were regular. Wet film examination of the blood revealed microfilaria.

Case 2 had a history of fear and excitement on hearing loud noise. Vomiting, anorexia, diarrhoea, polidypsea and poliurea were also reported. Conjuctival mucous membrane was pale roseate and rectal temperature was 101.8°F. The dog was weak and showed respiratory discomfort on lateral recumbency.

Pulse deficit was observed in both cases and in Case 2 pulse rate was much slower than the heart rate. In Case 1, heart rate was 214 bpm. The heart rate varied from 120-140 bpm in Case 2. Thoracic auscultation revealed respiratory

distress and loud heart sounds in Case 1. Case 2 upon auscultation showed paroxysms of rapid irregular beats.

4.4.1.2 Electrocardiographic findings

In Case 1, lead II of the ECG tracing revealed morphologically abnormal P waves with generally normal QRS complexes which were suggestive of supraventricular tachycardia(PLATE 2.).

Typical ECG findings in lead II of case 2 revealed ectopic P waves, which were superimposed on preceding T waves. The P-R intervals were very short. A compensatory pause was also seen following this and after that comparatively normal P wave and QRS complexes were observed. This short burst of supraventricular premature complexes was suggestive of paroxysmal supraventricular tachycardia (PLATE 2.).

4.4.1.3 Radiographic findings

In Case 1, mild cardiac enlargement was evident in the lateral plain radiograph. There was increased contact between cardiac silhouette and sternum and the cranial border of the cardiac silhouette was vertically oriented indicating right ventricular enlargement.

In Case 2, no changes were evident in the cardiac silhouette.

4.4.1.4 Echocardiographic findings

On two-dimensional echocardiographic examination, biventricular enlargement was noticed in Case 1 with hypertrophied right and left ventricle.

Echocardiographic findings of Case 2 were comparatively normal except for the mild mitral valve insufficiency. Enlargement of right kidney (7.1 cm) was observed on ultrasonography. There was slight increase in the echogenecity of renal cortex and cortico-medullary distinction was not clear. The left kidney was comparatively normal.

4.4.1.5 Haematology

Haematological values are given in Table 7. Case 1 had mild degree of anaemia. Haemoglobin and VPRC were 8.0 g per cent and 24.0 per cent respectively and TLC was 10,600.0 per cu.mm. Differential count was almost normal (N = 68.0 per cent, L = 30.0 per cent, M = 2.0 per cent.)

In Case 2, haemoglobin and VPRC were within the normal reference range (14.0 g per cent and 40.0 per cent). Total leucocyte count was 13,500 per cu. mm and differential count (N = 80.0 per cent, L = 18.0 per cent, E = 2.0 per cent) suggestive of neutrophilia.

4.4.1.6. Serum biochemistry

Serum biochemical values are given in Table 8

Serum creatinine was within the normal reference range in Case 1 (0.6 mg per cent) whereas the serum creatinine was abnormally high in Case 2 (12.8 mg per cent) suggestive of a renal failure. Serum creatine kinase values in Case 1 and 2 were 35.0 U/L and 54.0 U/L respectively. Serum sodium, potassium and calcium values were 136.0 mEq/l, 3.4 mEq/l and 10.5 mg/dl in Case 1 and 147.0 mEq/l, 4.5 mEq/l and 10.0 mg/dl in Case 2

4.4.2 Ventricular premature complexes (VPC)

Ventricular premature complexes were observed in four out of eight cases (Cases 3, 4, 5 and 6), which formed 50.0 per cent of the total cases. The mean age of the affected dogs was 10.5 years. Each one belonging to Cocker Spaniel, Boxer, Doberman and German Shepherd was affected. Out of four dogs having VPC, three were males.

4.4.2.1 Clinical findings

Clinical parameters are given in Table 6.

Cases 3 and 4 had a common history of exercise intolerance, generalized weakness, lethargy and nocturnal cough. Case 3 had dyspnoea whereas occasional syncope was the prominent clinical sign in Case 4. Cardiac cachexia

was also evident in Case 4. Conjunctival mucous membrane was pale roseate in both cases. In Case 3, rectal temperature was 101.8°F and in Case 4, it was 103.0°F. Case 4 was earlier treated for microfilaria. Both dogs were inappetant. Hind limb oedema also noticed in Case 4. The heart rate in Case 3 and 4 were 110 bpm and 124 bpm respectively. Gallop rhythm could be appreciated by auscultation in Case 3. In Case 4 haemic murmurs heard on auscultation of the heart. Pulse deficit was noticed in both cases.

Cases 5 and 6 were presented with complaints of anorexia, vomiting, lethargy, weakness and oligurea. Severe melena and anemia observed in case 5 whereas diarrhoea and polydipsia reported in Case 6. Their heart rate was 120 bpm and 84 bpm respectively. The character of the pulse in these dogs was hyperkinetic and haemic murmurs were heard upon auscultation of the heart.

4.4.2.2 Electrocardiographic findings

Evaluation of lead II ECG tracing revealed wide, bizarre QRS complexes, which were not associated with P waves. T waves were directed opposite to the QRS complexes. All these findings were suggestive of ventricular premature complexes. Out of the 4 cases of VPC, 3 were paroxysmal VPC's in which occasional VPC's followed by normal P waves and QRS complexes were observed. One case was ventricular bigeminy in which every other complex was a VPC (Case 4) (PLATE 3).

4.4.2.3 Radiographic findings

In Case 3 and 4, cardiac enlargement was evident in the lateral radiograph. Pulmonary congestion was also noticed in Case 3. No abnormal findings were observed on radiography in Case 5 and 6(PLATE 4).

4.4.2.4 Echocardiographic findings

The left parasternal four chamber view of the heart in Case 3 revealed enlargement of all the four chambers of the heart. Mitral valve insufficiency was also noticed in this case.

The left parasternal four-chamber view of the heart in Case 4 revealed dilated left atrium and left ventricle with relatively thin walls. Typical M-mode changes were larger systolic dimension with thin interventricular septal and left ventricular posterior wall measurements (PLATE 5).

4.4.2.5 Haematology

Haematological parameters are given in table 7. Case 3 had normal values of haemoglobin and VPRC (10.0 g per cent and 32.0 per cent). Anaemia observed in Case 4 with a haemoglobin concentration of 8.0 g per cent and VPRC of 26.0 per cent. Haemoglobin concentrations in Case 5 and 6 were 9.0 g per cent and 7.0 g per cent respectively and their VPRC were 30.0 per cent and 22.0 per cent.

In Case 3 and 4, TLC values were 11,500.0 per cu.mm and 9,800.0 per cu.mm respectively. Neutrophilia observed in Case 4 (N=76.0 per cent, L=23.0 per cent, M=1.0 per cent) whereas it was normal in Case 3 (N=71.0 per cent, L=28.0 per cent, M=1.0 per cent). Total leucocyte count of Case 5 and 6 were 11,000.0 per cu.mm and 12,800.0 per cu.mm respectively. Neutrophilia was observed in both cases (N=74.0 per cent and 82.0 per cent respectively).

4.4.2.6. Serum biochemistry

The serum biochemical values are given in table 8. Cases 3 and 4 had normal creatinine concentrations (1.3 mg per cent and 1.1 mg per cent). In the case of Creatine Kinase (CK), a high value (181.0 U/L) was obtained in Case 4 whereas in Case 3, CK value was within the normal reference range (48.0 U/L) serum sodium, potassium and calcium values in Case 3 were 132.0 mEq/l, 4.2 mEq/l and 10.2 mg/dl respectively and in Case 4 they were 146.0 mEq/l, 3.4 mEq/l and 9.3 mg/dl.

Creatinine levels were high in Case 5 and 6 (5.4 mg per cent and 3.3 mg per cent) whereas their CK values were within the normal reference range (32.0 U/L and 41.0 U/L). Serum sodium potassium and calcium values in Case 5 were 138.0 mEq/l, 5.2 mEq/l and 10.3 mg/dl respectively and corresponding values in Case 6 were 126.0 mEq/l, 3.8 mEq/l and 9.2 mg/dl respectively.

4.4.3. First degree atrio-ventricular block

First degree atrio-ventricular block was observed in one out of eight dogs (12.5 per cent). The signalment of the dog is given in table 1. The dog was a 10-year-old male Dobermann (Case 7) (PLATE 6).

4.4.3.1. Clinical findings

Clinical parameters are given in the Table 6.

The dog had a history of severe weakness, lethargy and progressive wasting. Appetite of the dog was normal. Rectal temperature was 102°F and mucous membrane was pale roseate. Heart rate was 82 bpm. Muffled heart sound was heard on auscultation and pulse was hyper kinetic.

4.4.3.2 Electrocardiographic findings

The P wave and QRS complex were normal in configuration but the P-R interval was prolonged (0.18 seconds) suggestive of first degree atrioventricular block. The P wave amplitude and duration were 0.2 mv and 0.04 seconds respectively. The Q wave amplitude was 0.6 mv. QRS duration was 0.04 sec and the amplitude of R wave was 0.4 mv. Q-T interval was 0.16 second. The polarity of T wave was negative.

4.4.3.3 Radiographic findings

Thoracic radiography revealed enlargement of the cardiac silhouette. The outline was smooth and globoid.

4.4.3.4 Echocardiographic findings

The left parasternal four-chamber view of the heart in B-mode echocardiography revealed slight increase in right ventricular chamber dimension. Mild hypoechoic region was also seen surrounding the heart.

4.4.3.5. Haematology

Haematological values are given in Table 7.

Haemoglobin and VPRC were 11.0 g per cent and 28.0 per cent respectively. Total leucocyte count was 14,000.0 per cu.mm and Differential count was almost normal (N = 74.0 per cent, L = 24.0 per cent and M = 2.0 per cent).

4.4.3.6. Serum biochemistry

Serum biochemical values are given in the Table 8.

Serum creatinine was 1.4 mg per cent. Creatine kinase was within the normal reference range (29.0 U/L). Serum sodium potassium and calcium values were 133.0 mEq/l, 4.8 mEq/l and 9.5 mg/dl respectively.

4.4.4. Right bundle branch block (RBBB)

Right bundle branch block was noticed in one out of eight dogs. (12.5 per cent).The dog was an eight year old female Mongrel (Case 8) Signalment of this dog is given in Table 1.

4.4.4.1. Clinical findings

Clinical parameters are given in Table 6.

The dog had a history of weakness, lethargy and exercise intolerance. Mucous membrane was slightly congested and rectal temperature was 103.2°F. The dog exhibited mild dyspnoea. Appetite was normal and the dog was not having proper records of vaccinations and deworming. Wet film examination of the blood revealed microfilaria.

The heart rate was 140 bpm. The heart sound and rhythm appeared normal on auscultation with associated palpable pulses.

4.4.4.2 Electrocardiographic findings

The QRS duration was prolonged (>0.07 second). Prominent, slurred S waves were noticed in leads II, III and aVF (PLATE 6).

4.4.4.3 Radiographic findings

Mild degree of pulmonary congestion was noticed on lateral plain radiograph. No changes were evident in the cardiac silhouette.

4.4.4.4 Echocardiographic findings

The dog had no cardiac abnormality in both 2-D echocardiography and M-mode echocardiography. The myocardial and vulvular kinesis was also normal.

4.4.4.5. Haematology

Haematolgoical values are given in Table 7.

Haemoglobin and VPRC values were 12.0 g per cent and 40.0 per cent respectively. Total leucocyte count was 12,400.0 per cu. mm and Differential count was also normal (N = 70.0 per cent, L = 27.0 per cent and E = 3.0 per cent).

4.4.4.6. Serum biochemistry

Serum biochemical values are given in Table 8.

Serum creatinine was within the normal reference range (0.9 mg per cent). Serum creatine kinase was 40.0 U/L. Values of serum sodium, potassium and calcium were 129.0 mEq/l, 3.8 mEq/l and 8.1 mg/dl respectively.

Table.1.Signalment of dogs having arrhythmia

Dog Number	Breed	Age (year)	Sex
1.	Dobermann	9	Male
2.	German Shepherd	5	Male
3.	Cocker Spaniel	9	Male
4.	Boxer	7	Male
5.	Dobermann	12	Male
6.	German Shepherd	3	Female
7.	Dobermann	10	Male
8.	Mongrel	8	Female

Table 2. Electrocardiographic parameters – control group

Parameters	Mean ± SE
P duration (sec)	0.040 ± 0.00
P amplitude (mv)	0.167 ± 0.012
PR interval (sec)	0.117 ± 0.016
QRS duration (sec)	0.037 ± 0.0043
R amplitude (mv)	1.467 ± 0.192
Q-T interval (sec)	0.190 ± 0.012
T wave (mv)	0.116 ± 0.012
ST segment (mv)	0.100 ± 0.021
Heart rate (bpm)	106 ± 4.79

Table 3. Haemogram of the control group

Parameters		Mean ± SE
VPRC (per cent)		36.5 ± 2.8
Haemoglobin (g per cent)		11.8 ± 0.5
TLC (per cu.mm)		11,375.5 ± 936.3
Differential Count (per cent)	Neutrophils	70.4 ± 1.5
	Lymphocytes	27.1 ± 1.1
	Monocytes	1.3 ± 0.4
	Eosinophils	1.0 ± 0.5
	Basophils	0

Table 4. Serum chemistry of control group

Parameters	Mean ± SE
Creatine kinase (U/L)	38.3 ± 4.7
Sodium (mEq/l)	138.5 ± 1.2
Potassium (mEq/l)	4.1 ± 0.3
Calcium (mg/dl)	9.3 ± 0.5
Creatinine (mg/dl)	0.7 ± 0.1

Table.5.Different types of arrhythmia observed in dogs

Dog Number	Heart Rate	Type of arrhythmia
1.	214	Supra-ventricular tachycardia
2.	130	Paroxysmal supra ventricular tachycardia
3.	110	Ventricular premature complexes (VPC)
4.	125	Ventricular bigeminy
5.	120	Ventricular premature complexes (VPC)
6.	84	Ventricular premature complexes (VPC)
7.	82	First degree atrio ventricular block
8.	140	Right bundle branch block (RBBB)

Table 6.Clinical parameters of dogs with arrhythmia

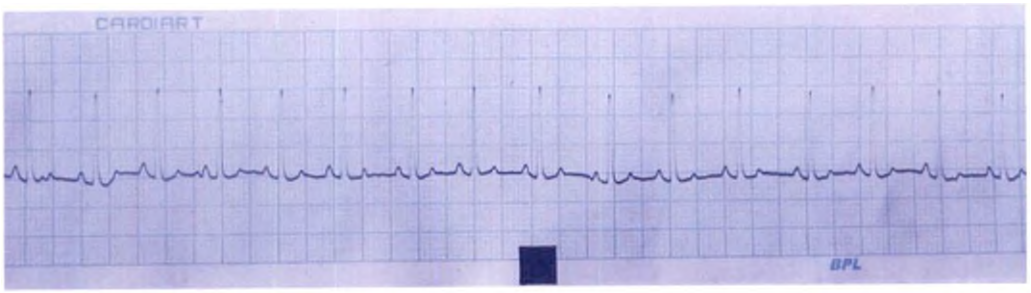
Dog number	Respiration (per minute)	Pulse (per minute)	Temperature (in °F)	Mucous membrane
1	32.0	180.0	102.2	Pale
2	36.0	126.0	101.8	Pale roseate
3	20.0	90.0	101.8	Pale roseate
4	35.0	98.0	103.0	Pale roseate
5	25.0	106.0	102.4	Pale
6	32.0	80.0	102.8	Pale
7	28.0	78.0	102.0	Pale roseate
8	30.0	132.0	103.2	Slightly congested

Table 7. Haemogram of dogs having arrhythmia

Day number	Hb (g%)	VPRC (%)	TLC (per cu.mm)	DC				
				N (%)	L (%)	M (%)	E (%)	B (%)
1	8.0	24.0	10600.0	68.0	30.0	2.0	0	0
2	14.0	40.0	13500.0	80.0	18.0	0	2.0	0
3	12.0	32.0	11500.0	71.0	28.0	1.0	0	0
4	8.0	26.0	9800.0	76.0	23.0	1.0	0	0
5	9.0	30.0	11000.0	74.0	26.0	0	0	0
6	7.0	22.0	12800.0	82.0	17.0	1.0	0	0
7	11.0	28.0	14000.0	74.0	24.0	2.0	0	0
8	12.0	40.0	12400.0	70.0	27.0	0	3.0	0

Table 8. Serum chemistry of dogs having arrhythmia

Dog number	Creatine kinase (U/L)	Creatinine (mg per cent)	Sodium (mEq/l)	Potassium (mEq/l)	Calcium (mg/dl)
1	35.0	0.6	136.0	3.4	10.5
2	54.0	12.8	147.0	4.5	10.0
3	48.0	1.3	132.0	4.2	10.2
4	181.0	1.1	146.0	3.4	9.3
5	32.0	5.4	138.0	5.2	10.3
6	41.0	3.3	126.0	3.8	9.2
7	29.0	1.4	133.0	4.8.0	9.5
8	40.0	0.9	129.0	3.8	8.1



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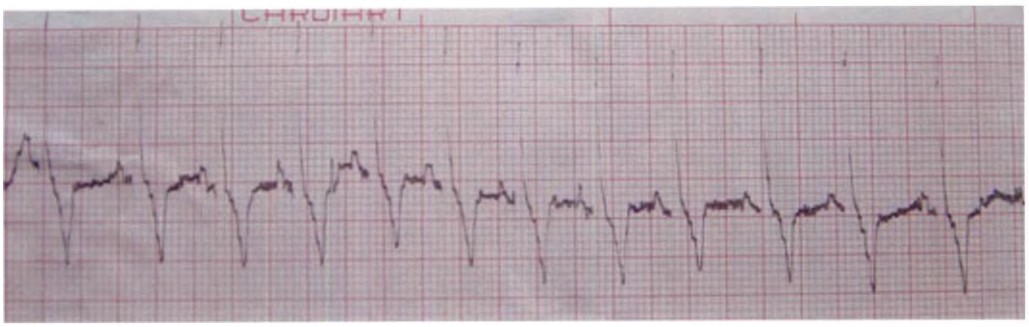

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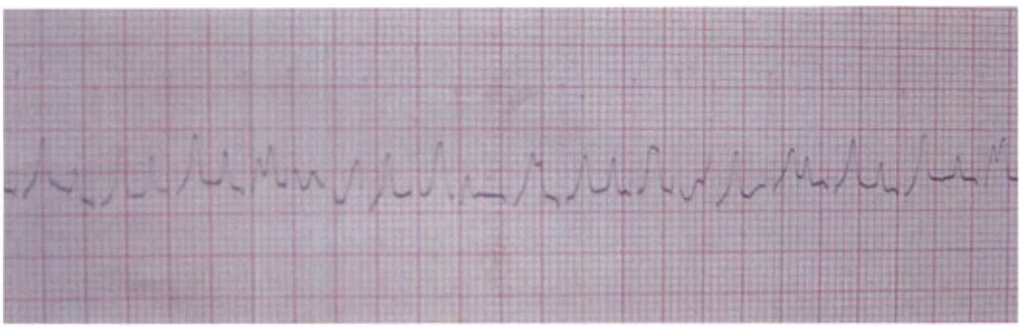


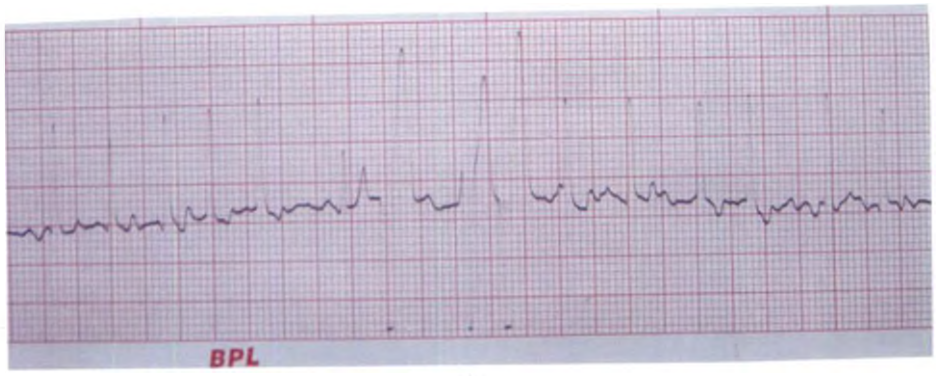
C

Plate 2. ECG of normal and arrhythmic dogs

A. Normal Electrocardiogram

B. Dog 1; Supraventricular tachycardia - Abnormal P waves with normal QRS complexes

C. Dog 2; Supraventricular tachycardia - Ectopic P waves superimposed on preceding T waves



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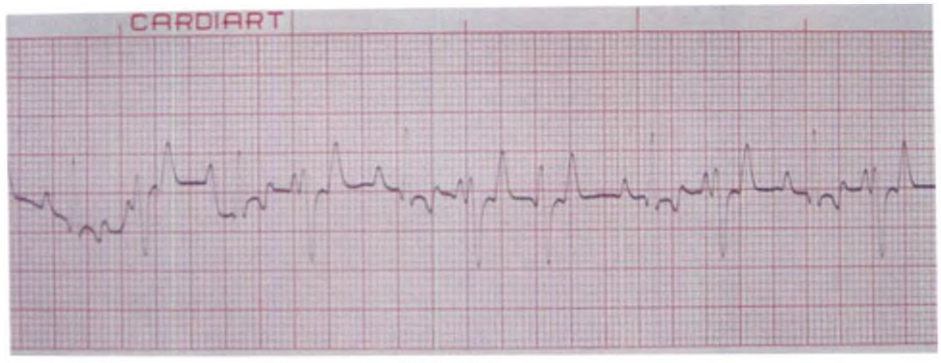

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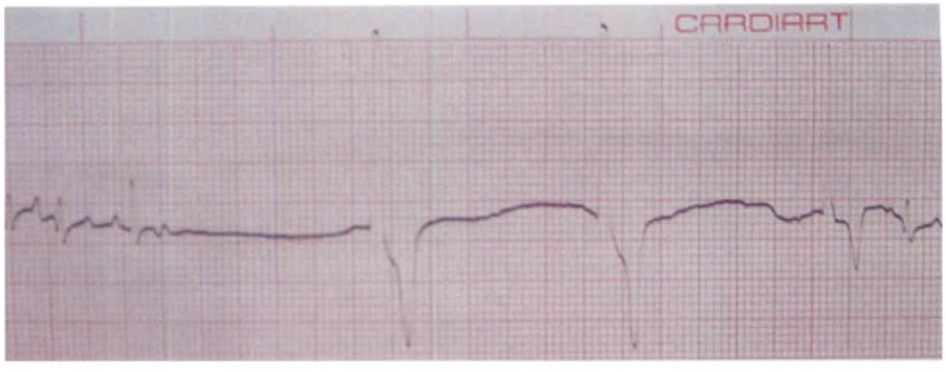

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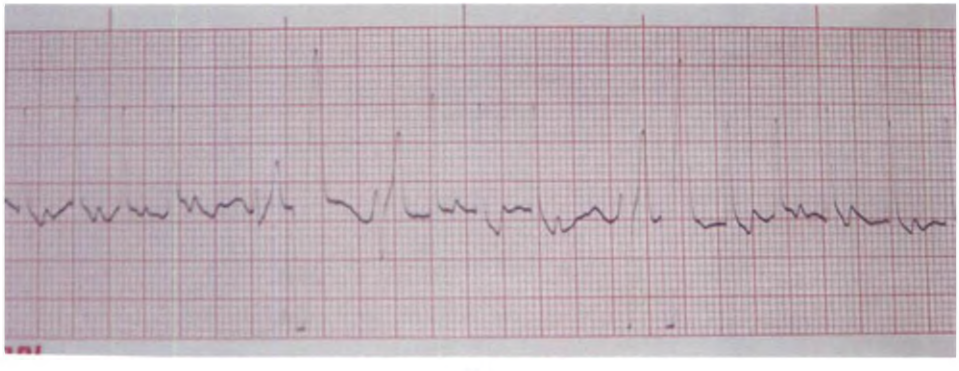
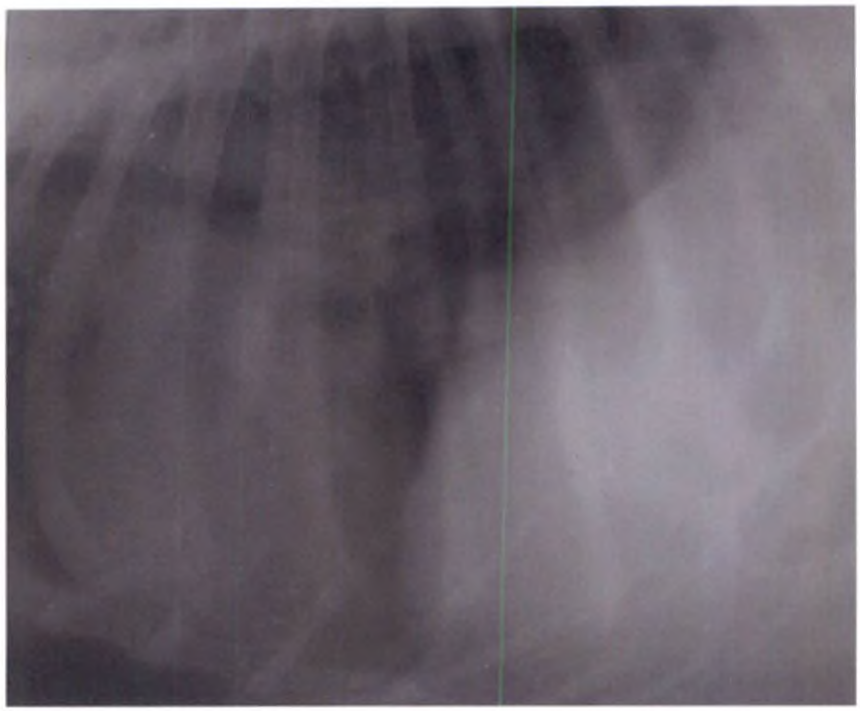


D

Plate 3. Lead II ECG tracing of dogs with ventricular premature complexes

A. Dog : 3; B. Dog : 4; C. Dog : 5; D. Dog : 6.

Wide bizarre QRS complexes not associated with P waves.



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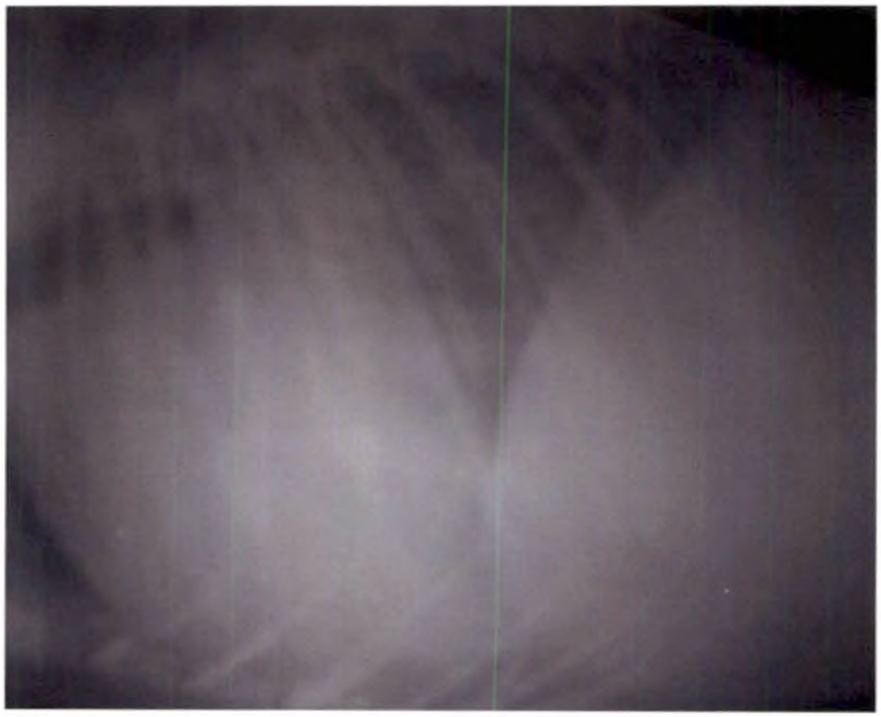


B

Plate 4 . Radiography of arrhythmic dogs

A. Dog : 3; B. Dog : 4.

Enlarged cardiac silhouette

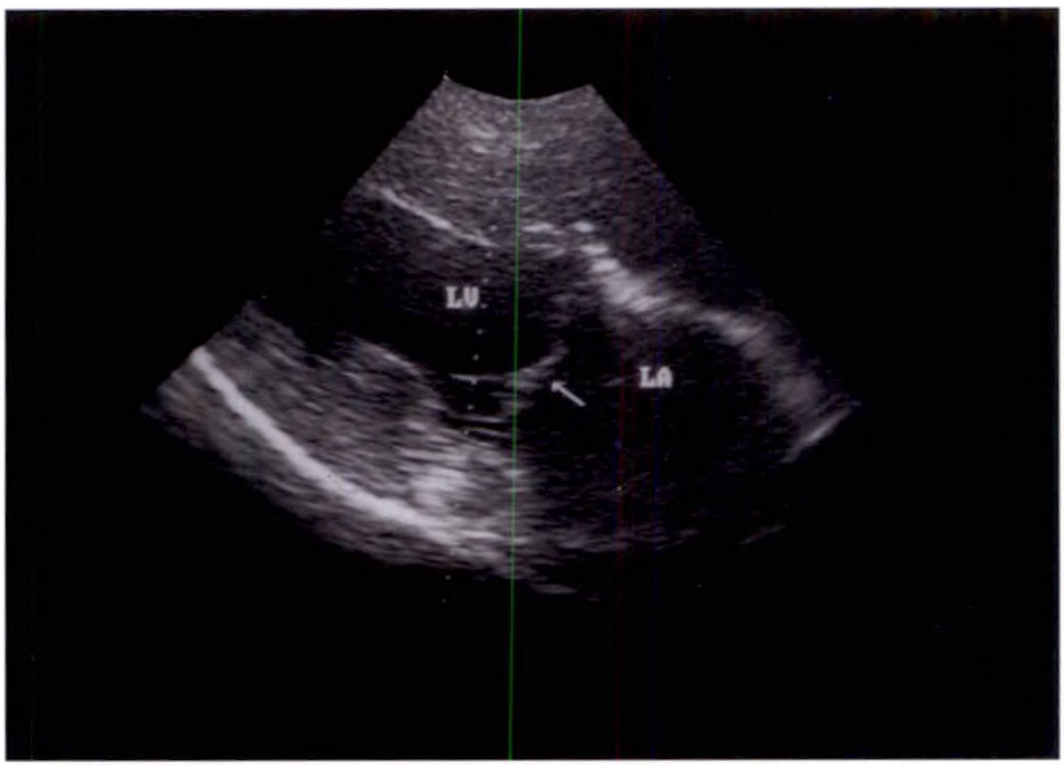
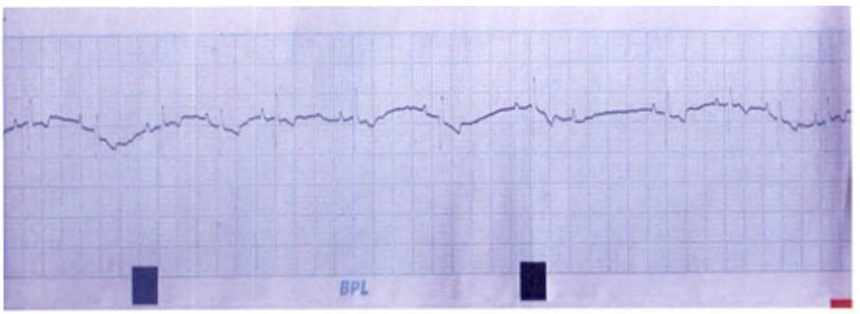


Plate 5. Echocardiogram of a dog with DCM (Dog : 4)
Left parasternal four chamber view
Dilated left atrium and ventricles with thin interventricular septum



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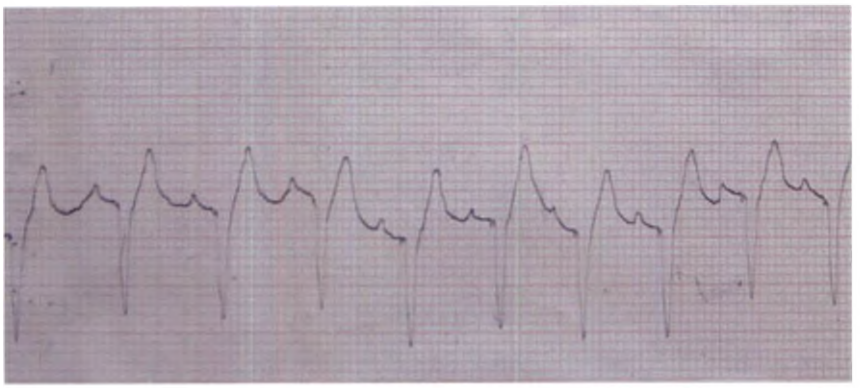

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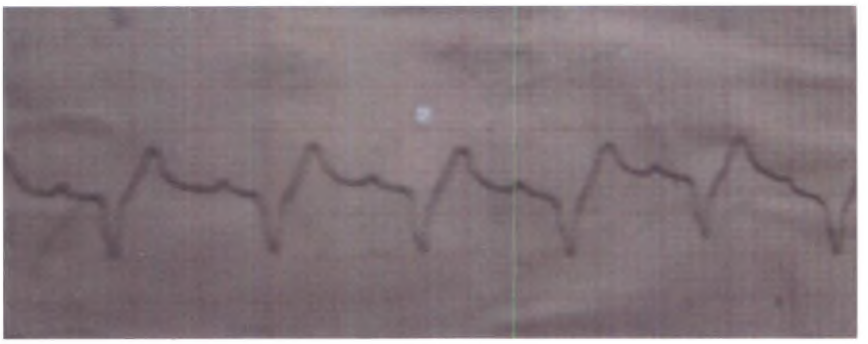

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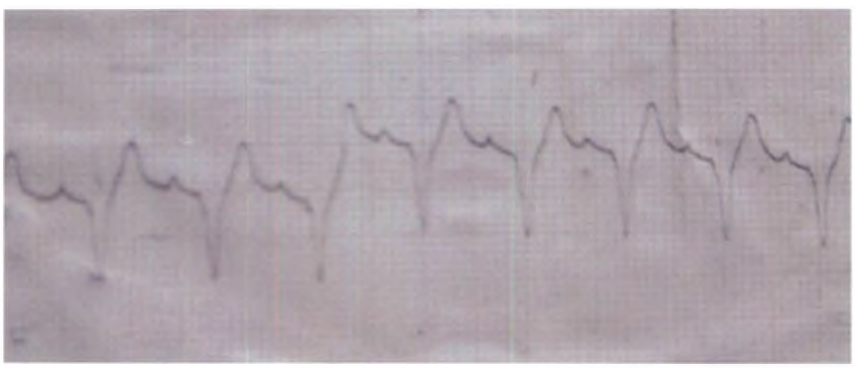

D

Plate 6. ECG of arrhythmic dogs

A. Dog : 7 - First degree atrioventricular block (Prolonged P-R interval)

B, C & D Dog : 8 - Right bundle branch block

B. Lead II; C. Lead III; D. Lead aVF

Prolonged QRS duration

Discussion

5. DISCUSSION

In the present study, eight dogs having arrhythmia were studied and the results are discussed in detail.

5.1 PREVALENCE

The prevalence of cardiac disease and arrhythmia obtained in the present study was 0.22 per cent and 0.07 per cent of the total canine cases respectively. The occurrence of cardiac disorders obtained in the present study was very less when compared with the results of earlier workers. This may be due to comparatively small population included in the present study. A higher occurrence of 1.3 per cent were observed by Ravindran (2001)

5.2 SIGNALMENT

5.2.1. Age

In the current study arrhythmia was found to be more in middle aged dogs and 62.5 percent of dogs having arrhythmia were between seven and ten year old. Cardiac disease usually affects middle aged, large pure- bred dogs (Gompf, 2001)

5.2.2. Sex

The occurrence of arrhythmias were higher in males (75.0 percent) than in females (25.0 percent). The predisposition of male animals for cardiac diseases has been reported by Gompf (2001) and DeFransesco (2004).this may be because of over representation of males in the canine population. (Vijayakumar, 2002)

5.2.3. Breed

Dobermann Pinschers were more prone to arrhythmia (37.5 percent) followed by German Shepherd (25.0 percent), Boxer (12.5 percent), Cocker Spaniel (12.5 percent) and Mongrel (12.5 percent). Among breeds, a high incidence of electrocardiographic abnormalities was observed in Dobermann Pinschers (Lombard, 1984 and Jacobs,

1996).An autosomal dominant mode of inheritance is suspected in Dobermann (McEwan, 2000; Gompf, 2001).

German Shepherds had an inherited problem with ventricular arrhythmias and sudden cardiac death. (Leib and Monroe, 1997). McEwan (2000) observed that English Cocker Spaniels were more predisposed to cardiac diseases. Severe arrhythmias often occur in Boxers with normal cardiac chamber size (Calvert, 2001).

5.3. CONTROL GROUP

5.3.1.Haematological parameters

The heamatological parameters of control animals were within the normal range as mentioned by Schalm *et al.* (1975)

5.3.2. Serological parameters

The serological values of control animals were within the normal range (Gavaghan and Kittleson, 1997; Dibartola, 2000; Wortingter, 2001)

5.3.3. Electrocardiographic parameters

Electrocardiographic parameters of control group are in agreement with the findings made by Bernal *et al.* (1995); Paslawska (1998) and Ettinger *et al.* (2000).

5.4. DETAILS OF DIFFERENT TYPES OF ARRHYTHMIA OCCURRED.

5.4.1. Supraventricular tachycardia

Among the eight dogs, supraventricular tachycardia was observed in two dogs (Case 1 and 2), which form about 25.0 per cent of total cases. This value is high when compared with the studies made by Ettinger and Suter (1970).They found out that out of 124 cases, only three dogs (2.4 per cent) had supraventricular tachycardia. Ages of the affected dogs were nine and five year and they belonged to Dobermann Pinscher and German Shepherd breed respectively. Both of them were males.

Case 1 was presented with a history of exercise intolerance, anorexia and occasional cough. Clinical signs such as exercise intolerance, cough and tachypnoea were frequently associated with heart failure and it occurred secondary to either decrease in stroke volume or abnormal heart rate or rhythms (Bolton, 1975; Strickland and Goodwin, 1998; DeFransesco, 2004).

The rectal temperature was within the normal range (102.2^0F). This is in agreement with the observations made by Abbott (1998). Mucous membrane was pale. It was probably due to poor cardiac output or anaemia because of the presence of microfilaria. A mild non regenerative anaemia was commonly observed in dogs with chronic heart worm infection (Calvert *et al.*, 1988).

Case 2 had a history of fear and excitement on hearing loud noise. Vomiting, anorexia, diarrhoea, polydipsia and poliurea were also reported in this case. These clinical findings were suggestive of acute renal failure (Cowgill and Elliott, 2000; Kraje, 2002).

In both cases pulse deficit was observed. Ristic (2004) reported that pulse deficits associated with arrhythmias might be clinically significant. Heart rate was normal in Case 2 (140 bpm), but it was above the normal reference range in Case 1. (214bpm). Thoracic auscultation revealed dyspnoea and loud heart sounds in Case 1. Gompf (2001) reported that most common cardiac cause of dyspnoea was left heart failure causing pulmonary oedema. Case 2 upon auscultation showed paroxysms of rapid irregular beats. Rapid heart rate and paroxysms of rapid irregular beats were suggestive of supraventricular tachycardia. (Bolton, 1975).

In Case 1, lead II of the ECG tracing revealed morphologically abnormal P waves with generally normal QRS complexes which were suggestive of supraventricular tachycardia. Bolton (1975) observed that P waves of supraventricular tachycardia were different from the P waves when the heart rate was normal.

In Case 2, typical ECG findings in Lead II included ectopic P waves which were superimposed on preceding T waves. The P-R intervals were very short. A compensatory pause was also seen following this and after that comparatively normal P

wave and QRS complexes were observed. This short burst of supraventricular premature complexes was suggestive of paroxysmal supraventricular tachycardia. This is in agreement with the observations made by Ettinger *et al.* (2000).

In Case 1, mild cardiac enlargement was evident in the lateral plain radiograph. There was increased contact between cardiac silhouette and sternum and the cranial border of the cardiac silhouette was vertically oriented indicating right ventricular enlargement. In congestive cardiomyopathy, radiographic changes affecting the cardiac silhouette included marked heart enlargement, typically of a biventricular nature. (Thomas, 1987; McEwan, 2000).

No radiographic changes were evident in the cardiac silhouette in Case 2.

On two dimensional echocardiographic examination, biventricular enlargement was noticed in Case 1 with hypertrophied right and left ventricle. These findings are suggestive of hypertrophic cardiomyopathy. (Moise and Fox, 1988).

Echocardiographic findings of Case 2 were comparatively normal except for the mild mitral valve insufficiency. Ravindran (2001) observed thickening of mitral valve in left side heart failure. In this case, enlargement of right kidney (7.1 cm) was observed on ultrasonography. There was slight increase in the echogenicity of renal cortex and cortico medullary distinction was not clear. The left kidney was comparatively normal. The ultrasonographic appearance of kidneys in acute renal failure was mild to marked increase in cortical echogenicity with variable degrees of lesser echogenicity at the cortico medullary junction (Cowgill and Elliott, 2000). Case 1 had mild degree of anemia (Hb = 8.0 g percent, VPRC = 24.0 percent). This could be due to the presence of microfilaria (Calvert *et al.*, 1988)

In case 1, TLC was 10,600.0 per cu.mm. DC was also normal (N=68.0 per cent, L = 30.0 per cent, M = 2.0 per cent) Haematological parameters were often normal in patients with congestive heart failure (Abbott, 1998).

In case 2, haemoglobin and VPRC were within the normal reference range with slight leukocytosis and neutrophilia, suggestive of infection or intoxication. Neutrophilia in renal failure could be due to systemic intoxication that occurs in uremia.

It is prominent in patients with pericarditis or other inflammation related to severe azotemia (Wintrobe *et al.*, 1981).

Serum creatinine was within the normal reference range in Case 1 (0.6 mg percent) whereas the serum creatinine was abnormally high in Case 2 (12.8 mg percent) suggestive of a renal failure. Increased blood urea nitrogen and creatinine concentration resulted from a decreased glomerular filtration rate causing limited excretion of these substances (Kraje, 2002).

Serum Creatine Kinase (CK) values were normal in both cases. In domestic species, CK isoenzymes analysis has not been shown to be of significant value as serum creatine kinase activity rapidly returns to normal after a muscle damaging incident. Creatine kinase concentration may not be of value for diagnosis of cardiac failure in dogs (Wyatt *et al.*, 1998).

Serum sodium, potassium and calcium values were within normal range in both cases. These findings are not in agreement with the observations made by Forrester and Brandt (1994) and Jacobs (1996). According to them hyperkalemia and a mild hypocalcaemia frequently observed in dogs with acute renal failure.

Sisson *et al.* (2000) stated that in DCM serum electrolytes were within the normal reference range.

Based on the above findings it was concluded that in case 1, supraventricular tachycardia was occurred as a result of congestive heart failure. According to Abbott (1998) some patients with severe CHF and markedly reduced cardiac output, develop tachycardia. In case 2 acute renal failure was the cause of supraventricular tachycardia. Supraventricular or ventricular tachycardia are consistent electrocardiographic abnormalities in acute renal failure. Arrhythmias are developed as a result of myocardial contractility and excitability, which may be triggered or worsened by hypervolemia, acidosis or uremic toxins. (Cowgill and Elliott, 2000).

5.4.2 Ventricular premature complexes (VPC)

Ventricular premature complexes were observed in four out of eight cases (50.0 per cent) (Cases 3, 4,5 and 6). This was comparatively high compared with the findings of earlier workers. According to Ettinger and Suter (1970), 34.7 per cent was the incidence of VPC. The mean age of the affected dogs was 10.5 years. Each one belonging to Cocker Spaniel, Boxer, Dobermann and German Shephered breed was affected. Out of four dogs having VPC, three were males.

Case 3 and 4 had a common history of exercise intolerance, generalized weakness, lethargy and nocturnal cough. Exercise intolerance, cough and tachypnoea were frequently associated with heart failure (Strickland and Goodwin (1998). Leib and Monroe (1997) reported that tachyarrhythmias could cause weakness in congestive heart failure due to reduced perfusion to skeletal muscle caused by poor myocardial function. Case 3 had dyspnoea. Occasional syncope was the prominent clinical sign in Case 4. Occasional syncope in cardiac disorders was also reported by DeFransesco (2004) and Schrope and Kelch (2006). Some Boxers had frequent ventricular arrhythmias in early stages of DCM and these arrhythmias might be responsible for syncopal episodes. (McEwan, 2000). Syncope observed in ventricular tachycardia could be attributed to the rapid heart beats resulting in reduced diastolic filling and cardiac output (Durham, 2005). Cardiac cachexia was also evident in Case 4. Cardiac cachexia is a usual complication of congestive heart failure. Cardiac cachexia is most commonly seen in dogs with dilated cardiomyopathy especially those with right sided failure. Cardiac cachexia might have resulted from malabsorption, maldigestion, increased energy requirements and hypermetabolic state associated with CHF (Moneva – Jordan, 2003).

Physiological parameters were normal in both cases. In both cases, inappetance was observed. Moneva – Jordan (2003) opined that inappetance could be directly related to congestive heart failure, malaise and dyspnoea. Hind limb oedema also noticed in Case 4 which is a usual clinical sign in CHF (Abbott, 1998). The heart rate in Case 3 and 4 were 110 bpm and 124 bpm respectively. Gallop rhythm was observed

during auscultation in Case 3. Gallop rhythm indicated myocardial failure. (Sisson *et al.*, 2000)

In Case 4 haemic murmurs heard on auscultation of the heart. A murmur of functional mitral valve insufficiency was seen in many dogs with dilated cardiomyopathy and this murmur occurred in systole (Abbott, 1998). Pulse deficit was noticed in both cases. The premature contractions occurred before adequate ventricular filling and resulted in no ejection and a pulse deficit (Goodwin, 2000).

Cases 5 and 6 were presented with complaints of anorexia, vomiting, lethargy, weakness and oliguria. Severe melena and anaemia observed in Case 5 where as diarrhoea and polydipsia reported in Case 6. Similar clinical signs were reported in acute renal failure by Forrester and Brandt (1994). Their heart rates were normal. The pulses in these dogs were hyperkinetic and haemic murmurs were heard upon auscultation of the heart. The haemic murmurs observed were physiological murmursproduced by functional changes such as increased cardiac output seen anaemia (Detweiler and Patterson, 1967). The hyperkinetic pulse observed in those dogs in the present study was due to decreased vascular resistance in anemia (Goodwin, 2000).

Evaluation of lead II ECG tracing in all these 4 cases revealed wide, bizarre QRS complexes which were not associated with P waves. T waves were directed opposite to the QRS complexes. All these findings were suggestive of ventricular premature complexes. Out of the 4 cases of VPC, 3 were paroxysmal VPC's in which occasional VPC's followed by normal P waves and QRS complexes were observed. One case was ventricular bigeminy in which every other complex was a VPC (Case 4). All these findings were same as described by Goodwin (2001) and Martin (2002a). Ulloa *et al* (1995) opined that complexes originating near the bundle of His appeared normal and those arising more distally were wide and bizarre in shape Ventricular premature contractions were evident in dogs with dilated cardiomyopathy. (Calvert and Jacobs, 2000).

In Case 3 and 4, cardiac enlargement was evident in the lateral plain radiograph. Pulmonary congestion was also noticed in Case 3. Radiographic changes in DCM included left atrial enlargement alone, left atrial and left ventricular enlargement, right atrial and right ventricular enlargement or generalized cardiomegaly. Pulmonary venous congestion where pulmonary veins became larger than pulmonary arteries was also a common finding in DCM (McEwan, 2000).

No abnormal findings on radiography in Case 5 and 6.

The left parasternal four chamber view of the heart in Case 3 revealed hypertrophy of all the four chambers of the heart. Mitral valve insufficiency was also noticed in this case. Abbott (2001) observed that severe mitral valve insufficiency might cause systolic fluttering of the septal leaflet. The more chronic and common causes of canine heart failure, were associated with cardiomegaly (DeFranseco, 2004).

The left parasternal four chamber view of the heart in Case 4 revealed dilated left atrium and left ventricle with relatively thin walls. Typical M- mode changes were larger systolic dimension with thin interventricular septal and left ventricular posterior wall measurements suggestive of dilated cardiomyopathy. (Moise and Fox, 1988).

Case 3 had normal values of haemoglobin and VPRC. In Case 4 haemoglobin concentration and VPRC concentration were 8.0 g percent and 26.0 percent respectively suggestive of anaemia.

Haemoglobin concentrations in Case 5 and 6 were 9.0 g percent and 7.0 g percent respectively. These values were considerably low when compared with the control group suggestive of anemia. Anemia of renal disease might be due to decreased erythropoietin production, decreased intake of iron and Vitamin B12 and toxic suppression of haemopoiesis in the bone marrow (Kraje, 2002).

In Case 3 and 4 TLC values were 11,500.0 per cu.mm and 9,800.0 per cu.mm respectively. Differential count was normal in Case 3 where as slight neutrophilia observed in Case 4. (N=76.0 percent, L=23.0 percent, M=1.0 percent). Ristic (2004) opined that many cardiac cases would have a stress leukogram causing considerable elevations in mature neutrophil counts. The values of TLC in Case 5 and 6 were normal.

But neutrophilia observed in both cases (N=74.0 percent and 82.0 percent respectively). Neutrophilia in renal failure could be due to systemic intoxication that occurs in uraemia (Ravindran, 2001).

Cases 3 and 4 had normal serum creatinine concentrations. In Case 3, CK value was around the normal reference range (48.0 U/L) where as a high value of 181.0 U/L was obtained in Case 4. Creatine kinase might be elevated in many cardiac diseases like acute myocardial infraction and skeletal muscle damage. (Wyatt *et al.*, 1998). Serum sodium, potassium and calcium values were normal in Case 3 and 4.

Creatinine values were high in Case 5 and 6. (5.4 mg percent and 3.3 mg percent), suggestive of renal failure (Kraje, 2002). Their CK values were within the normal reference range. Serum sodium, potassium and calcium values were normal in Case 5 where as values of sodium and potassium in case 6 was slightly below from the normal level. (Sodium = 126.0 mEq/l and Potassium= 3.8 mEq/l)

The values of calcium were also within the normal reference range in Case 5 and 6. According to Forrester and Brandt (1994), hypocalcaemia was reported in 44.0 to 50.0 per cent of dogs with renal failure. This variation may be due to difference in the severity of renal failure.

From the above findings it was clear that, mitral valve insufficiency was the cause of ventricular premature complexes in Case 3 and dilated cardiomyopathy was the cause in Case 4. Arrhythmias were reported in 53.0 percent of dogs with mitral valve insufficiency (Ettinger and Suter, 1970). Chronic valvular heart disease may lead to degenerative myocardial lesions which may stimulate ectopic ventricular foci and resulted in ventricular premature complexes.

Acute renal failure was the cause of arrhythmia in Case 5 and 6. In renal failure, uraemic toxins caused ventricular myocarditis by irritation or inflammation of ventricular myocardium which predisposed the patients to ventricular premature beats or ventricular tachycardia. (Ettinger and Suter, 1970; Bolton, 1975).

5.4.3 First degree atrio -ventricular block

First degree atrio -ventricular block was observed in one out of eight dogs. (12.5 per cent).This is less when compared with the findings of Ettinger and Suter (1970).According to their studies, the incidence of first degree atrio -ventricular block was 23.3 percent. The dog was a 10 year old male Dobermann Pinscher (Case 7)

The dog was presented with a history of severe weakness, lethargy and progressive wasting. Weakness, lethargy, exercise intolerance and syncope were the most common clinical signs in atrio- ventricular blocks (Schrope and Kelch, 2006). Roudebush and Freeman (2000) observed that cardiac cachexia, the muscle wasting associated with congestive heart failure was a common disorder of patients with cardiac disease. Appetite of the dog was normal. Rectal temperature and mucous membrane were normal. Heart rate was 82 bpm. Miller *et al.* (1988) observed that heart rate was usually normal in first degree atrio- ventricular block unless associated with drug intoxication or severe underlying cardiac disease. Muffled heart sound was heard on auscultation. Large volumes of pleural or pericardial effusion altered the effort and pattern of respiration and muffled both heart and lung sounds. (Sisson and Thomas, 1988) Hyperkinetic pulse was observed in association with fever or anemia (Goodwin, 2000).

The P wave and QRS complex were normal in configuration but the P-R internal was prolonged (0.18 seconds) suggestive of first degree heart block. (Littlewort, 1967). Normal P-R interval ranged from 0.06 – 0.13 second (Bolton, 1975 and Goodwin, 2001). Martin (2002b) stated that first degree atrio -ventricular block occurred due to a delay in conduction through the atrio-ventricular node. The P wave amplitude and duration were 0.2 mv and 0.04 seconds respectively which were normal when compared with the control group. The Q wave amplitude was 0.6 mv. According to Bolton (1975) Q waves deeper than 0.5 mv in lead II were suggestive of right ventricular hypertrophy. The QRS duration and Q-T interval was within the normal reference range. R wave amplitude was less when compared with the reference range of control group. This could be due to pericardial or pleural effusion (Ravindran, 2001). According to Ware

(2001) complexes of diminished amplitude were seen in association with pericardial effusion.

Thoracic radiography revealed enlargement of the cardiac silhouette. The outline was smooth and globoid suggestive of pericardial effusion. These findings are in concur with the findings of Ware (2001).

The left parasternal four chamber view of the heart in B-mode echocardiography revealed slight increase in right ventricular chamber dimension, suggestive of right ventricular hypertrophy. Mild hypoechoic region was also seen surrounding the heart, which provided a definitive diagnosis of pericardial effusion (Ristic, 2004).

All haematological values were within the normal reference range when compared with the control group. This is in concur with the findings of Wilbanks (1992).

Serum creatinine, creatine kinase and electrolytes were within the normal range. According to Ristic (2004) haematobiochemical values were not of much useful for the diagnosis of heart disease.

First degree atrio-ventricular block is an ECG abnormality which does not usually indicate cardiac disease.

5.4.4 Right bundle branch block (RBBB)

Right bundle branch block was observed in one out of eight dogs. (12.5 per cent).This value is high when compared to that of Ettinger and Suter (1970). They observed two cases of RBBB (1.6 per cent) out of 124 canine cases. This variation may be due to less number of cases included in the present study. In this case the dog was an eight year old female Mongrel.

The dog had a history of weakness, lethargy and exercise intolerance. According to Bolton (1975) no clinical sings were attributable to right bundle branch block. Mucous membrane was slightly congested and rectal temperature was 103.2^0F which were slightly above from the normal reference range when compared with the control group. The dog exhibited mild dyspnoea. All these signs might be due to the presence of

microfilaria in the blood stream. Coughing and dyspnoea were the most common signs in canine heart worm disease and usually associated with parenchymal disease of the caudal lung lobe (Calvert *et al.*, 1988).

Heart rate was normal (140bpm). The heart sound and rhythm appeared normal on auscultation with associated palpable pulses. Similar observations were made by Martin (2002 b).

Lead II of the electrocardiogram revealed prolonged QRS complexes (>0.07 second) suggestive of right bundle branch block. (Miller *et al*, 1988 and Martin, 2002b) Prominent, slurred S waves were noticed in leads II, III and aVF. This is in concur with the findings of Bolton (1975); Ravindran (2001) and Martin (2002 b)

Mild degree of pulmonary congestion was noticed on lateral plain radiograph. According to Sisson and Thomas (1988) in canine heart worm disease, common pulmonary arterial abnormalities included increased prominence of the main pulmonary artery and enlargement or abnormal tapering of the peripheral pulmonary arteries.

On radiography and echocardiography no changes were evident in the cardiac silhouette Right bundle branch block does not directly impair cardiac function (Bolton, 1975; Goodwin, 2001).

All haematoligical parameters were within the normal range when compared with the control group. This is in consistent with the findings of Abbott (1998).

Serum creatinine and creatine kinase values were normal in this case. Values of serum sodium and potassium were 129.0 mEq/l and 3.8 mEq/l which were less when compared with the values of control group. Value of calcium (8.1 mg/dl) was within the normal reference range.

Right bundle branch block is an incidental finding in normal dogs. Clinical signs of weakness, lethargy and cough occurred in the present study may be the effect of circulating microfilaria.

Summary

6. SUMMARY

The present study entitled "Electrocardiographic Studies on Arrhythmia in Dogs" was conducted to study the incidence and to delineate the etiopathogenesis and clinical manifestation of arrhythmias in dogs. Study was conducted in the Department of Clinical Medicine, College of Veterinary and Animal Sciences, Mannuthy for a period of one year (2005 –2006).

Dogs brought to the Veterinary College Hospital, Mannuthy and University Veterinary Hospital, Kokkalai, with signs of cardiac problem like lethargy, weakness, dyspnoea, cough, syncope and exercise intolerance were used for the study. They were screened for cardiac involvement by detailed clinical examination.

Standard electrocardiogram patterns and haematobiochemical parameters were worked out from six healthy dogs.

Out of 11,230 canine patients, 106 (0.9 per cent) cases were suspected to have cardiac disorders. Out of these 106 cases, 25 (23.6 per cent) were confirmed as cardiac diseases, of which eight (32.0 per cent) had arrhythmia. Cardiac disease and arrhythmia accounted for 0.22 per cent and 0.07 per cent of the total canine cases respectively

Based on clinical examination, electrocardiography, echocardiography, haematology and serum biochemical assays, a total of eight cases having arrhythmia were selected.

Arrhythmia was more commonly observed in dogs between the age group of seven to ten years. Out of the eight cases, six were found to be males and remaining were females. Arrhythmia was recorded more in Dobermann Pinschers followed by German Shepherd, Boxer, Cocker Spaniel and Mongrel.

Out of the eight cases of arrhythmia two were supraventricular tachycardia (25.0 percent), four were ventricular premature complexes (50.0 per cent), one

was first degree atrio- ventricular block (12.5 per cent) and the remaining was right bundle branch block (12.5 percent).

The most common clinical signs in arrhythmia observed were lethargy, weakness, exercise intolerance, cough and dyspnoea. Syncope and cardiac cachexia were also observed.

Pulse deficits and haemic murmurs were also noticed in few cases.

In supraventricular tachycardia, typical ECG findings in lead II were morphologically abnormal P waves, very short P-R intervals and ectopic P waves which were superimposed on preceding T waves. Out of the two cases encountered, test results and findings revealed that congestive heart failure was the cause in one case and acute renal failure was the cause in the other.

Ventricular premature complexes were obtained in four cases. Typical ECG findings were wide bizarre QRS complexes, not associated with a P wave and T waves were directed opposite to the QRS complexes. Ventricular Premature Complexes (VPC) were also noticed in two cases of acute renal failure. They had a history of vomiting, diarrhoea, oligurea, melena and anaemia.

Radiography and echocardiography revealed cardiac enlargement and mitral value insufficiency in one case and dilated cardiomyopathy in another case.

In the case of VPC with dilated cardiomyopathy, creatine kinase value was very high. In cases of renal failure, serum creatinine values were above the normal level. Other biochemical values were within the normal reference range.

In first degree atrio -ventricular block, P wave and QRS complexes were normal in configuration, but the P-R interval was prolonged. (0.18 seconds). R wave amplitude was less due to pericardial effusion and it was confirmed by radiography and echocardiography. Hematological and serum biochemical values were normal. First degree atrio -ventricular block is an ECG abnormality which does not usually indicate cardiac disease.

In the case of right bundle branch block, duration of QRS complex was more than 0.07 seconds. Prominent, slurred S waves were also noticed in lead II, III and aVF.

The dog exhibited mild dyspnoea which might be due to the presence of microfilaria in the blood stream. On radiography, mild degree of pulmonary congestion was noticed whereas no echocardiographic changes were evident in the cardiac silhouette. Haematological parameters were normal and serum potassium level was slightly below the normal range. Right bundle branch block was found to be an incidental finding in the present case.

Based on the present study it was concluded that,

1. Arrhythmias were frequently associated with cardiac disorders and secondary to renal diseases.
2. Electrocardiography was a useful clinical tool for the diagnosis of arrhythmias.
3. Haematology and biochemistry were not particularly useful for the diagnosis of arrhythmia but helpful to investigate concurrent diseases.

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ELECTROCARDIOGRAPHIC STUDIES ON ARRHYTHMIA IN DOGS

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ABSTRACT

Study entitled "Electrocardiographic Studies on Arrhythmia in Dogs" was conducted in eight dogs. It was conducted to study the incidence and to delineate the etiopathogenesis and clinical manifestation of arrhythmias in dogs. Out of 11,230 canine patients, cardiac disease observed in 0.22 per cent and arrhythmia observed in 0.07 per cent of cases. The parameters observed were signalment, history and detailed clinical examination, electrocardiography, radiography, echocardiography, haematology and serum biochemical assays.

Dog between the groups of seven to ten years were more frequently affected and it was more commonly observed in males. Arrhythmia was recorded more in Dobermann Pinschers followed by German Shepherd, Boxer, Cocker Spaniel and Mongrel.

The types of arrhythmia observed in the present study included supraventricular tachycardia, ventricular premature complexes, first degree atrio-ventricular block and right bundle branch block. In supra ventricular tachycardia typical ECG findings in lead II were morphologically abnormal P waves and very short P-R intervals. In ventricular premature complexes, typical ECG findings were wide bizarre QRS complexes and T waves directed opposite to QRS complexes. Prolonged P-R interval was the typical finding in first degree atrio-ventricular block. In right bundle branch block typical ECG findings were prolonged QRS complexes with prominent slurred S waves.

The frequently observed clinical signs of arrhythmia were lethargy, weakness, exercise intolerance, cough and dyspnoea. Syncope and cardiac cachexia were also observed. Pulse deficits and haemic murmurs were also noticed in a few cases.

Arrhythmias were noticed in association with cardiac diseases and secondary to renal diseases.

Radiography and echocardiography were useful to find out cardiac chamber enlargement and mitral valve insufficiency.

Haematological and serum biochemical values did not show any significant changes in arrhythmias.