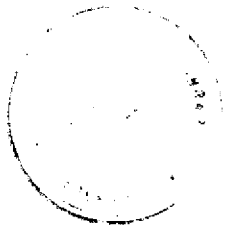
ELECTROCARDIOGRAM ABNORMALITIES IN CARDIAC DISORDERS OF DOGS

By

RAVINDRAN. P.



THESIS

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DECLARATION

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

Mannuthy,

RAVINDRAN, P.

CERTIFICATE

Certified that the thesis, entitled **"ELECTROCARDIOGRAM ABNORMALITIES IN CARDIAC DISORDERS OF DOGS"** is a record of research work done independently by **Dr. P. Ravindran,** under my guidance and supervision and that it has not previously formed the basis for the award of any degree, fellowship or associateship to him.

Dr. S. Ajithkumar
(Chairman, Advisory Committee)
Assistant Professor
Department of Clinical Medicine
College of Veterinary and
Animal Sciences, Mannuthy

Mannuthy,
5/4/2021

CERTIFICATE

We, the undersigned members of the Advisory committee of **Dr. P. Ravindran,** a candidate for the degree of Master of Veterinary Science in Clinical Medicine, agree that the thesis entitled **"ELECTROCARDIOGRAM ABNORMALITIES IN CARDIAC DISORDERS OF DOGS"** may be submitted by Dr. P. Ravindran, in partial fulfilment of the requirement for the degree.

Dr. S. Ajithkumar
(Chairman, Advisory Committee)
Assistant Professor
Department of Clinical Medicine
College of Veterinary and Animal Sciences, Mannuthy

Dr. P.C. Alex
(Member, Advisory Committee)
Associate Professor and Head
Department of Clinical Medicine
College of Veterinary &
Animal Sciences, Mannuthy

Dr. R. Vijayan
(Member, Advisory Committee)
Associate Professor
Department of Animal Husbandry
College of Agriculture
Vellayani, Thiruvananthapuram

Dr. K. Rajankutty
(Member, Advisory Committee)
Associate Professor
Department of Surgery
College of Veterinary &
Animal Sciences, Mannuthy

External Examiner

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Dedicated to
My Parents and Brothers

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Introduction

1. INTRODUCTION

The function of the heart is to maintain the blood circulation. It carries out this function in a very complex and delicate manner along with a vast network of blood vessels and capillaries. Pure oxygenated blood supply is maintained throughout the body with constant clearing of impure blood. Thus the whole body is constantly bathed with oxygen and substances that are essential for the survival of the cells.

Right side of the heart receives impure blood from the body and pumps it into the pulmonary vascular bed where it gets purified and is brought back to the left side of the heart. Each and every aspect of the structure of the heart, its anatomy, its position, the bicuspid and tricuspid valves, the chordae tendinae etc. are so well oriented to serve specific purpose associated with pumping of blood. Heart is also well adapted to adjust and alter its rate, rhythm and capacity when need arises.

The heart beat is an electrical process and each time the heart muscle contracts the electrical currents flow through it. Waller (1887) was the first to demonstrate that the electrical impulses of the heart could be recorded from the surface of the body. He utilized his pet dog to record the first electrocardiogram ever known.

Any abnormality or disease, however minute can cause variation in the conduction of electrical impulses from the heart and these impulses can be traced by an electrocardiogram. The electrocardiogram has special importance in animals especially in canines. Unlike in humans, the animals cannot complain verbally the symptoms. Heart diseases may manifest as signs of involvement of various other systems of the body and clinical investigations may proceed in the wrong direction if special techniques like ECG has not been carried out.

In veterinary patients heart diseases are the most unnoticed and misdiagnosed cases in field conditions. Hence electrocardiogram becomes imperative for diagnosing heart diseases or even for a routine check up. The electrocardiogram can detect both the functional and anatomical changes of heart to a greater extent. A thorough clinical examination is a must before proceeding to electrocardiography. The electrocardiogram is very specific for changes of the myocardium and it forewarns the type of impending heart failure. However it may require ancillary diagnostic techniques like radiography and ultrasonography for further confirmation.

With good interpretation, electrocardiography is the cheapest, noninvasive, easily accessible and accurate diagnostic technique in cardiology. With sound knowledge one can perceive or correlate the graphs on the ECG paper with the structure and function of the heart. The statement of Lewis (1912) is most appropriate in this regard i.e., "The time is at hand, if it has not

already come, when an examination of the heart is incomplete if electrocardiography is neglected."

Much work has been done in western countries regarding canine heart diseases, but it is still in a budding stage in India especially in Kerala. With the increasing pet population especially the exotic one's in this State, the cardiac problems have been on a rise. But these diseases are not often properly diagnosed and treated obviously due to the lack of diagnostic facilities, proper knowledge and experience.

The study of electrocardiogram abnormalities in dogs is a new and emerging field of research in our country and this research work entitled "Electrocardiogram abnormalities in cardiac disorders of dogs" was formulated with the following objectives.

1. Standardisation of the three standard limb leads, three augmented unipolar leads and chest leads in the common breeds of dogs in Kerala of varying age groups.

2. Identification of the ECG abnormalities in dogs affected with cardiac disorders.

3. Correlation of the ECG abnormalities detected, with other clinical and biochemical observations.

Review of Literature

2. REVIEW OF LITERATURE

2.1 Occurrence

Detweiler and Patterson (1965b) reported that in their study, heart disease (congenital and acquired) was present in 545 out of 4831 dogs (11.3 per cent) examined consecutively at the University of Pennsylvania.

Taylor and Sittinikow (1968) stated that out of 4126 dogs examined in the Veterinary College Clinic, Helsinki, Finland in 1967, 166 had clinical signs suggestive of heart disease. On special clinical examination, of 4126 dogs, 124 (3 per cent) were found to have definite heart disease, 15 (0.34 per cent) had pulmonary diseases and the remaining dogs had other diseases. Of the 124 dogs with heart disease, 112 dogs (90.3 per cent) had acquired lesions and 12 dogs (9.7 per cent) had congenital heart diseases.

Myocardial disease was recognised with increasing frequency in dogs. Out of the total dogs examined 1.1 per cent had dilated cardiomyopathy .(Fioretti and Dellicarri, 1988).

Tidholm (1997) reported that out of 151 dogs, with congenital heart diseases, the commonest defect was aortic stenosis (35 per cent), followed by pulmonic stenosis (20 per cent), ventricular septal defect (12 per cent), patent ductus arteriosus (11per cent), mitral valve dysplasia (8 per cent), tricuspid

valve dysplasia (7 per cent), endocardial fibroelastosis (1.9 per cent) and tetralogy of Fallot (0.6 per cent).

2.2 Signalment

The incidence of congestive heart failure was two and a half times greater in males than in females (Detweiler *et al.*, 1961).

One study on endocardiosis with mitral or tricuspid regurgitation and another investigation of pulmonary fibrosis/bronchitis/emphysema showed that, for every year after five years of age, dogs have a dramatic increase in the incidence of these two types of disease (Hamlin, 1990).

Buchanan (1992) reported that cardiac disease was relatively common in dogs, with acquired cardiac disease comprising the majority of such cases.

Clinical signs of congestive heart failure resulting from mitral valve endocardiosis were observed almost exclusively in geriatric small breed dogs (Abbot, 1998).

2.3 General Clinical Examination Findings

Diastolic murmurs could also be sometimes appreciated in association with anaemia (Detweiler and Patterson, 1965a).

Physiological murmurs were produced by functional changes such as increased cardiac output as seen in anaemia and fever (Detweiler and Patterson, 1967).

Fisher (1967) reported that in primary right sided heart failure due to vascular obliterative changes in the lungs, the clinical signs included exercise intolerance, coughing, tachypnoea, emphysematous crackling,, systemic congestion, oedema, fast weak pulse and some times cyanosis of mucosa.

A jerky pulse with rapid upstroke and downstroke but with less accentuation could be palpated in mitral insufficiency (Gould *et al.*, 1968).

Ettinger (1969) observed fainting associated with cardiac conditions such as tachycardia, bradycardia and heart blocks.

Ettinger and Suter (1970) stated that cardiac cough was low pitched and resonant and occurred in paroxysms, first noticed during the early morning hours. It was further characterized by terminal gagging or coughing up of a white or blood-tinged phlegm. Other clinical signs observed in congestive heart failure were open mouth breathing with extension of neck, abduction of elbows, left ventricular heave, precordial thrill, muffling of heart and lung sounds during auscultation of thorax, paroxysmal dyspnoea, anorexia, diarrhoea, ascites and cardiac cachexia.

Calvert *et al.* (1982) observed weight loss, episodic weakness, exercise intolerance, coughing, irregular heart rhythm, gallop rhythm, and pulmonary oedema in Dobermann pinschers with congestive cardiomyopathy.

Scheel and Williams (1985) reported that in chronic anaemia both the right and left ventricles were hypertrophied in dogs.

Calvert (1986) observed syncope and weakness in Dobermann pinschers with dilated cardiomyopathy.

Kuehn (1986) reported that chronic coughing was a common complaint at rest or upon minimal exertion in heart failure due to chronic mitral regurgitation and was often due to bronchial irritation from left atrial compression on the left main stem bronchus. Tachypnoeic dyspnoea and cyanosis were other clinical signs associated with the development of pulmonary oedema. Lethargy, fatigue and exercise intolerance were associated with diminished cardiac output.

Fleming *et al.* (1989) reported a sudden onset of vomiting, diarrhoea or constipation, depression, oliguria, anuria, bradycardia, dehydration, hypothermia, mucosal injection, azotemia, hyper-phosphatemia and hyperkalemia in dogs affected with acute renal failure.

Mikiciuk *et al.* (1989) reported polyuria and polydipsia in dogs affected with chronic renal failure. The dogs also exhibited clinical signs of uraemia such as weight loss, anorexia, vomiting, diarrhoea, rough hair coat, oral

ulceration, lingual discoloration and necrosis, mucosal pallor, scleral injection and dehydration.

Wilbanks (1992) reported lethargy, fainting, cyanosis and abdominal enlargement of four months duration in a 13 year old spayed female Schnauzer having complete atrioventricular (third degree) heart block. When forced to walk, the patient became weak and collapsed. Palpation of the femoral pulse revealed a rate of 30 beats/minute. Auscultation of the thorax revealed muffled heart sounds and a respiratory rate of 60 breaths/minute. A fluid wave was noted on percussion of the abdomen.

Tilley (1992) had reported sinus tachycardia in animals affected with congestive heart failure

Elwood *et al.* (1993) observed pyrexia, lameness, lethargy, inappetance, epistaxis, hind limb weakness, joint swelling, heart murmurs, tachycardia, dysrrhythmias, hyperkinetic pulses and oral ulcerations in dogs affected with bacterial endocarditis.

The clinical signs reported by Bonagura and Darke (1995) in congenital heart disease included failure to grow, shortness of breath, abdominal swelling, cyanosis, weakness, syncope, seizures and sudden death. Cardiac murmurs of congenital heart disease often were loud and accompanied by a precordial thrill. Sometimes a murmur might not be audible with very large defects even in the presence of severe cyanotic heart disease. Additional auscultatory

abnormalities, such as a loud or split second heart sound might offer further evidence of congenital heart disease. Abnormalities of the arterial pulse, mucous membranes, jugular venous pulse, or precordium might substantiate a clinical suspicion of congenital disease. Cyanosis often indicated pulmonary to systemic shunting, but could develop in animals with severe congestive heart failure and secondary pulmonary dysfunction.

Goodwin (1995) stated that hyperkinetic pulse refers to an arterial pulse that is abnormally strong and is seen in conditions associated with decreased vascular resistance like anaemia, fever and hyperthyroidism.

Ihle (1995) stated that cardiac disease could suppress the appetite, absorption and transport of nutrients from the intestine. He also reported that gastrointestinal parasitism, resulting in a relative deficiency in nutrition was the most common cause of reversible retarted growth in puppies.

Lunney and Ettinger (1995) stated that animals having supraventricular premature contractions were symptomatic for the primary disease and asymptomatic for the cardiac arrhythmia although clinical signs like syncope, paroxysmal weakness of congestive heart failure might accompany the frequent premature contractions. In atrial fibrillation, weakness ,collapse and syncopal episodes could be seen. In ventricular tachycardias reduced cardiac out put could be manifested as weakness, collapse, syncope, seizures and loss of appetite. Congestive heart failure could also result. A rapid rhythm, weak pulse, pulse deficits and pale mucous membranes might appear or were of

variable intensity. In animals in which the jugular furrow was visible, cannon "a" waves might be seen in the jugular vein as the atria contracted against a closed atrio-ventricular (AV) valve.

O'Grady (1995) reported that the earliest physical finding in chronic mitral valve insufficiency (CMVI) was the detection of an incidental soft left apical mid systolic murmur. The murmur had a mixed high and low frequency sound quality or might in some cases had a high frequency "whooping" quality. As the disease progressed, the murmur became more intense and holosystolic and radiated well to the right thoracic wall. In advanced CMVI, third (S_3) and fourth (S_4) heart sounds might be ausculted in addition to the normal heart sounds (S_1 and S_2). These were gallop sounds and were indicative of myocardial failure. A murmur of tricuspid valve insufficiency might also be detected over the tricuspid valve area in addition to mitral regurgitation (MR). This murmur was soft and mid systolic. It was also stated that the signs of right heart failure were related to the right atrial pressure and might include respiratory distress due to pleural effusion; abdominal distension due to ascites, hepatomegaly, or splenomegaly, or gastrointestinal signs such as diarrhoea, vomiting or anorexia.

Del Palacio *et al.* (1997) reported distended abdomen, dyspnoea, cyanosis, orthopnoea, mouth-breathing at rest, fast and hypokinetic femoral pulse, distended jugular veins and systolic murmur (grade II/VI) in a six week

old male puppy having persistent left cranial venacava associated with other multiple congenital anomalies.

Gavaghan and Kittleson (1997) observed severe dyspnoea, loud crackles and wheezes by auscultation throughout the respiratory cycle, II/VI left apical systolic murmur, pulse rate of 190 bpm, regular hypokinetic femoral pulse, normal capillary refill time, cyanotic oral mucous membranes and cold extremities in an American Cocker Spaniel with dilated cardiomyopathy.

White *et al.* (1997) reported lethargy, exercise intolerance, grade IV/VI systolic murmur with its point of maximum intensity (PMI) over the left cardiac base and hypokinetic femoral pulse in a Golden retriever having sub-valvular aortic stenosis and mitral dysplasia.

Abbot (1998) reported that body temperature and pulse quality were often normal in patients with congestive heart failure and pathological arrhythmia might result in varying pulse strengths and rates. It was also stated that alterations in respiratory rate and character depend on the severity of heart failure.

2.4 Electrocardiography

Littlewort (1967) had reported electrocardiography as a clinical method of choice for detecting cardiac disorders in dogs.

The electrocardiogram (ECG) is a graphic record of the voltage produced by cardiac muscle cells during depolarization and repolarisation plotted against time. In most dogs with acquired heart disease, the history and physical examination provide a great portion of the information needed for the final diagnosis and electrocardiography and radiography confirm or amplify the tentative diagnosis (Ettinger and Suter, 1970).

Tilley (1992) stated that in ECG the 'P' wave corresponds to atrial depolarisation or contraction, QRS to ventricular depolarisation or contraction and T wave represents ventricular repolarisation or relaxation.

2.4.1 Importance of electrocardiography

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

Electrocardiography is useful in the evaluation of cardiac diseases, differentiation of non specific diseases that cause weakness, fatigue, fever, lethargy, collapse or seizures and monitoring during anaesthesia and surgery (Bolton, 1975).

Electrocardiography is a useful tool in two major areas (1) diagnosing most cardiac arrhythmias, since the electrocardiogram can determine the source of the rhythm and the frequency with which the impulse arises; and

(2) providing information on the status of the myocardium, since the P-QRS-T deflections of the electrocardiographic tracing are often altered by either pathologic or physiologic factors (Tilley, 1992).

2.4.2 Indications for taking an electrocardiogram

Cardiac enlargement, arrhythmias, drug therapy, electrolyte disturbances, pericardiocentesis, adrenal insufficiency, diabetic ketoacidosis, renal insufficiency, eclampsia, badycardia, myocarditis, cardiac neoplasia, anesthesia and monitoring ventillation-oxygenation changes are the indications for taking an electrocardiogram (Bolton, 1975).

Tilley (1992) stated that the important indications for taking an electrocardiogram were tachycardia, bradycardia or arrhythmia heard on auscultation, acute onset of dyspnoea, shock, fainting or seizures, during surgery, cardiac murmurs, cardiomegaly on thoracic radiographs, cyanosis, evaluating the effect of cardiac drugs, electrolyte disturbances, pericardiocentesis and systemic diseases that affect the heart.

2.4.3 Techniques of electrocardiography

Hamlin and Stalnaker (1989) reported that there appeared to be no advantage for recording ECGs at 50mm/sec versus 25 mm/sec in dogs. Recording at the lower speed saved paper, permitted recording for longer periods and easier for identification of deflections of low amplitude and short duration.

Tilley (1992) reported that the lead systems necessary to view the heart from different directions were biopolar standard leads (I, II, III), augmented unipolar limb leads (aVR, aVL, aVF), unipolar precoralial chest leads (CV5RL, CV6LL, CV6LU and V10), modified orthogonal lead systems (X,Y,Z) and invasive leads (Oesophageal and intracardiac).

2.4.4 Limitations of Electrocardiography

The electrocardiogram tells nothing about the mechanical status of the heart, since an animal with congestive heart failure may have a normal electrocardiogram, and a perfectly normal animal may show non specific electrocardiographic abnormalities (Tilley, 1992).

2.4.5 Common Artifacts during recording

Ettinger and Suter (1970) stated that technical errors like reversal of electrode placement and panting and shivering by the dog could cause artifacts during recording.

Bolton (1975) stated that 60 cycle electrical interference, respiratory artifacts and movement artifacts are trouble areas during recording and interpretation of an electrocardiogram.

Electrical interference, wandering baseline, poorly defined baseline and inadequate frequency response were common artifacts seen during recording of an electrocardiogram (Tilley, 1992).

2.4.6 Determination of Heart rate Rhythm and Mean Electrical Axis

The heart rate could be determined by counting the number of ventricular complexes in 6 sec period and multiplying by 10. (Ettinger and Suter, 1970).

Bolton (1975) recommended to evaluate heart rhythm in lead II strip of ECG. The P waves should be related to the QRS complexes and this could be verified by checking that P-R intervals of all the beats are same. The normal rhythm in dog is sinus in origin, and if this sinus rhythm is perfectly regular it is called normal sinus rhythm. If the sinus rhythm has some irregularity it is called as sinus arrhythmia (a normal rhythm for the dog). Wandering pacemaker is another normal variation in the dogs.

Tilley (1992) stated that there are three basic methods for estimating the mean electrical axis in the frontal plane like (a) finding an isoelectric lead, the algebraic sum of the QRS deflections being zero, (b) choosing the lead with the largest net QRS deflection, (c) Measuring the algebraic sum of the QRS deflections in lead I and lead III and plotting the values on the triaxial system.

2.4.7 Normal Canine Electrocardiogram

Pouchelon *et al.* (1973) studied normal electrocardiograms of 72 healthy male German Shepherd dogs, aged 1-12 years (average 4.5). The average value of the P wave was 0.05 sec, QRS complex 0.056 sec, T wave 0.095 sec, PR interval 0.11sec and QT interval 0.22 sec. Respiratory sinus arrhythmia caused

difficulties in the interpretation of certain abnormal tracings. Sinoauricular blocks and extrasystoles were established in certain normal dogs.

Eckenfels (1986) reported that the amplitude (voltage) of the R-wave in 118 Beagles averaged 1.6 mv (range 0.5 to 2.7 mv) in lead II..

Bernal *et al.* (1995) studied serial electrocardiograms of lead II from 70 Mastin Espanol dogs in right lateral recumbency, aged between one day and three years. The P wave was positive and Monophasic in all recordings. The mean P wave duration ranged between 0.022 and0.038 seconds and increased with age and body weight. There was a highly significant correlation between PR interval and age and bodyweight, with shorter intervals in animals less than one month of age (0.058 to 0.060 seconds) and longer intervals in adult animals (0.140 seconds). In all the one-day-old animals, the authors found deep S waves. A 'Q' wave was observed in nearly all the recordings with an increase in the amplitude of Q wave until 45 days of age, then a decrease until the age of three months and the final mean values ranged between 0.27 and 0.43 mv. Sometimes the Q Waves were of a large amplitude, reaching 0.8mv. The amplitude of the R wave increased greatly after the first week of life. The older the animals, the greater the R Wave values. S waves greater than 0.3 mv were only found in one-day-old animals, with mean values of 0.73± 0.065 mv. QRS duration increased with age (0.029 to 0.055 seconds).

Blumenthal *et al.* (1996) evaluated P wave duration in lead II ECG in 364 healthy mongrel dogs weighing 13 to 35 kg. Mean P-wave duration for all dogs

(44.9 milli seconds) was greater than published accepted normal values for the dog (640 milli seconds). Dogs weighing >20 kg had longer mean P-wave durations (45.3 milli seconds) than dogs weighing <20 kg (41.6 milli seconds). Female dogs had a greater mean duration (45.4 milli seconds) than males (43.8 milli seconds).

Venkateshwarlu *et al.* (1997) made electrocardiographic observations in healthy mongrel dogs. Heart rate ranged from 60 to 100 beats/min with a mean of 85 ± 3.59. P wave was positive in all the animals in leads II, III and aVF. It was either positive or isoelectric in lead I. It was either negative or isoelectric in lead aVL. The P wave was always negative in aVR in all the animals. The duration of 'P' wave varied from 0.04 to 0.08 sec with a mean duration of 0.04 ± 0.003 sec. The amplitude of 'P' wave varied from 0.0 5 mv to 0.3 mv with a mean if 0,17 ± 0.02 mv. The duration of QRS interval ranged from a minimum of 0.04 sec. to 0.08 sec. with a mean of 0.05 sec. The amplitude of QRS complex varied from 0.4 to 1.0 mv with a mean of 0.93 ± 0.117mv. R wave was always negative in aVR lead. It was always positive in leads II, III and aVF. PR interval ranged from 0.08 sec. to 0.16 sec with a mean of 0.10 ± 0.007 sec. QT interval ranged from 0.16 sec to 0.2 sec with a mean of 0.18 ± 0.005 sec.

Roukolamine *et al.* (1998) recorded ECG using the precordial leads (CV_6LU, CV_6LL, CV_6RL,V10) from 100 Iranian German Shepherd dogs. In most dogs normal sinus arrhythmia was noticed. The P waves were positive in CV_6LL and CV_6LU. In lead V10, 81.8 per cent were isoelectric and 11.1 per

cent positive and 7.1 per cent were negative. The QRS complexes in all leads were of different shapes. The P.R. interval between dogs over 12 months and under 12 months of age in leads V10, CV_6LL was significantly different. Mean Q-T interval was also significant in leads CV_6LL, CV_6LU. Mean height of S wave in CV5RL was lower in dogs younger than 12 months than in dogs older than 12 months of age.

Konacevic *et al.* (1999) studied electrocardiographic values of 44 normal Dobermann pinscher dogs, 11 months to 8.5 years old, weighing 34.4 ± 4.9 kg. The following electrocardiographic values were recorded: heart rate 127.9 ± 23.6 beats per minute (range 70 to 176) , P wave amplitude 0.211 ± 0.072 mv (range 0.1 to 0.35), P wave duration 0.04 ± 0.003s (range 0.03 to 0.05) , QRS complex duration 0.053 ± 0.01s (range 0.02 to 0.07), R wave amplitude 1.66 ± 0.56 mv (range 0.6 to 3), Q wave amplitude 0.65 ± 0.4 mv (range 0.05 to 1.8), Q – T interval duration 0.187 ± 0.024 (range 0.14 to 0.26) and S-T segment level 0.049 ± 0.068 mv (range 0.1 to 0.2). These values were similar to the standard values, but values of mean electrical axes (50.9° ± 26°), with a range of 42° to 101° were considerably different.

2.4.8 Electrocardiography changes in cardiac diseases and other conditions

Detweiler (1959) stated that 1st degree and 2nd degree atrioventricular blocks in the dogs usually indicate primary myocardial disease which has involved the conducting system of the heart. Complete A-V block was almost always an indication of severe heart disease and carried a very poor prognosis.

Persistent, frequent premature beats usually indicated either severe myocardial disease or sometimes severe digitalis intoxication.

Buchanan (1965) reported that left bundle branch block indicated severe myocardial disease in dogs. Right bundle branch block was not always associated with heart disease. Atrial fibrillation led to reduced cardiac output and was usually secondary to severe heart disease.

Littlewort (1967) opined that in sino-atrial (S-A) block the impulse arising in the pacemaker was not conducted to the atria. First degree block manifested in the electrocardiogram as a prolonged interval between the P-wave, and the QRS complex. Second degree block results in a break in the regular rhythm of the heart, the length of which was a simple multiple of the normal interval between beats. During the interval one or more normal P-waves, unaccompanied by any QRS complex or T-wave, might be seen. In third degree or complete heart block all impulses from the atria were blocked in the A-V node and the atrial and ventricular beats become dissociated from one another.

When the right atrium hypertrophied, the P-waves become tall and peaked but remain normal in its width. This P wave is called "P pulmonale" and indicates right atrial hypertrophy. When the left atrium enlarges, the P waves become wide and notched. This P wave is termed "P mitrale" and indicates left atrial hypertrophy. Complexes of diminished amplitude might be seen on the electrocardiogram in pericardial effusion (Ettinger and Suter, 1970).

The R wave should not be taller than 3.0 mv in any dog and should not be taller than 2.5 mv in smaller dogs. An R wave that was too tall indicated left ventricular hypertrophy (Bolton, 1975).

Yoshike (1976) stated that the electric potential change of the S-T segment on the electrocardiogram offered a reliable method of diagnosis of myocardial infarction in dogs.

Lombard and Goldschmidt (1980) reported low voltage and small negative P waves in leads II, III and aVF in a dog with primary fibroma of the right atrium.

The major electrocardiogram findings in association with canine cardiac diseases were left atrial enlargement, left ventricular enlargement and right ventricular enlargement (McIntosh, 1981).

Gooding *et al.* (1982) reported that dogs with cardiomyopathy showed electrocardiogram changes compatible with left or biventricular hypertrophy in the absence of radiographic changes in dogs with concentric hypertrophy of the heart.

Boeve *et al.* (1984) documented significant relationships between the survival period and bodyweight, R-wave voltage and QRS duration in 59 cases of atrial fibrillation in dogs.

Baba and Arakawa (1984) observed increased depth of T-waves and depression of the S-T segment in myocardial hypoxia in an obese Beagle dog.

Lombard (1984) reported atrial fibrillation in 12 dogs with dilated cardiomyopathy.

Macintire and Snider (1984) reported premature ventricular contractions and ventricular tachycardia in dogs after trauma. Cardiac arrhythmias were associated with thoracic trauma, neurological injury, severe shock and/or extensive tissue trauma.

Thomas *et al.* (1984) stated that atrial fibrillation in dog was a cardiac dysrrhythmia frequently associated with marked atrial enlargement following chronic mitral and tricuspid valvular insufficiency, idiopathic cardiomyopathy and congenital heart defects. He also reported that increase P-wave duration and diminished QRS voltages on the electrocardiogram was a consistent diagnostic feature in constrictive pericardial disease of dogs.

Thomas (1987) reported supraventricular dysrrhythmias (atrial premature complexes, atrial tachycardia and atrial fibrillalion) in congestive cardiac failure in Cocker spaniels. Mean electrical axis in the frontal plane was normal in each case.

Jacobs (1989) reported that supraventricular tachycardias, such as atrial fibrillation and paraoxysmal atrial tachycardias, often developed in small animal patients with congestive heart failure.

Goodwin and Lombard (1990) reported that electrocardiogram was an integral part of the diagnostic evaluation of puppies that were suspected to have congenital heart disease. In left atrial enlargement the P-waves were wider than normal. The maximum normal width on a lead II tracing with a paper speed of 50 mm/sec is 0.04 sec. In right atrial enlargement the height of the P-waves were excessive (taller than 0.4 mv) in lead II, III or in aVF. In left ventricular enlargement, the height of the R-wave was increased (taller than 2.5 mv in leads II, aVF and in CV_6LU and taller than 3.0 mv in lead CV_6LL). The QRS complex might be wider than normal (greater than 0.06 sec in lead II). The mean electrical axis might on rare occasion be shifted to the left. In right ventricular enlargement deep S-waves were present in leads I, II, III, aVF, CV_6LL and CV_6LU (>0.05 mv in lead I; >0.035 mv in lead II; >0.8 mv in lead CV_6LL; and >0.7 mv in lead CV_6LU). The mean electrical axis was deviated to the right.

Hamlin (1990) stated that myocardial fibrosis might be detected by routine electrocardiography. The QRS complex for a normal young dog usually contained an R-wave (the high voltage positive deflection) that had a brisk rise and fall. However, the QRS complex of dogs with myocardial fibrosis often had a brisk ascent but a relatively slow descent containing slurs or notches. This made the QRS complex relatively long in duration. It was common to observe ventricular premature depolarizations occurring singly or paroxysmally in association with myocardial fibrosis.

Pattarakosol *et al.* (1991) reported bundle branch block and ventricular premature beat in dogs infested with *Dirofilaria immitis*. Right atrial hypertrophy, right ventricular hypertrophy and myocardial stress were evident in some dogs.

Ventricular premature complexes (VPC's) are impulses that arise from an ectopic focus in the ventricles. The impulse do not travel through the specialised conduction system but through ordinary muscle, spreading through both ventricles with delay and causing a bizarre widened QRS complex. VPCs are the most frequent type of abnormal rhythm in dogs (Tilley, 1992).

Zipes (1992) stated that ventricular tachyarrhythmias were associated with cardiomyopathies, myocarditis, advanced congestive heart failure with myocardial anoxia and intramural microscopic myocardial infarctions.

Bossabaly *et al.* (1993) reported atrial fibrillation in 19 per cent cases of idiopathic cardiomyopathy.

Baatz (1993) diagnosed arrhythmia in dogs with gastrointestinal form of canine parvovirus infection. Often supraventricular arrhythmia and conduction disturbances in the AV node and the ventricle were seen. Electrocardiographic changes were seen mostly during acute infection.

Elwood *et al.* (1993) reported that among ten cases of bacterial endocarditis in dogs, electrocardiographic findings included normal sinus rhythm (30 per cent), sinus tachycardia (20 per cent), ventricular premature

complexes (20 per cent), second degree atrioventricular block (10 per cent) and multifocal ventricular premature beats with paroxysmal ventricular tachycardia (10 per cent).

Marcks (1993) recorded sinus tachycardia and ventricular premature contraction in cases of canine cardiomyopathy.

Panciera (1994) reported that the most common ECG abnormality in canine hypothyroidism was decreased amplitude of R-wave. Experimental studies of hypothyroid dogs confirmed that atrioventricular conduction time, functional refractory period of the atrioventricular node and duration of ventricular action potential were prolonged.

Little and Julu (1995) reported severe sinus arrhythmia caused by increased parasympathetic tone in a dog with upper respiratory tract obstruction.

Elevated sympathetic or parasympathetic tone can provoke certain supraventricular arrhythmias, such as atrial fibrillation (Coumel *et al.*, 1996).

Freeman *et al.* (1996) studied nine Dalmatian dogs with idiopathic dilated cardiomyopathy. ECG findings include sinus rhythm (1 dog), sinus arrhythmia (1 dog), sinus tachycardia (6 dogs) and paroxysms of supra ventricular tachycardia (1 dog). Of the 6 dogs with sinus tachycardia at the time of diagnosis, 4 converted to a sinus rhythm, 1 developed first degree

AV block with infrequent ventricular premature deplolarisations and I continued to have sinus tachycardia.

Calvert *et al.* (1997) documented ventricular tachyarrhythmias in Dobermann pinschers with occult cardiomyopathy.

Del Palacio *et al.* (1997) observed narrow complex tachycardia (260 beats/min) with positive P-waves in lead I, II, aVL and aVF and negative P-waves in leads III and aVR, deep S-waves in leads I, II, III and aVF and a mean electrical axis of 90° in a six week old puppy having persistent left cranial venacava associated with multiple congenital anomalies.

Gavaghan and Kittleson (1997) reported that in an American Cocker Spaniel with dilated cardiomyopathy, the ECG showed sinus tachycardia at 170 bpm with infrequent (<2/min) ventricular premature contractions.

White *et al.* (1997) reported respiratory sinus arrhythmia in which the R-wave height was 3.0 mv and QRS duration was 0.05 seconds in a 12 month old neutered male Golden retriever with subvalvular aortic stenosis and mitral dysplasia.

Choi-In Hyuk *et al.* (1998) reported high T-wave, low R-wave and low S-T segment, high R-wave in hyperkalemia and hypokalemia respectively.

Driehuys *et al.* (1998) studied 37 cases of myocardial infarction in dogs and cats. Electrocardiogram abnormalities in dogs included ventricular

tachycardia (16%), atrial fibrillation (9%) and premature ventricular contractions (6.5%).

Brownlie and Cobb (1999) reported atrial fibrillation, ventricular and supra ventricular premature contractions, first and second degree atrioventricular block, P mitrale and left anterior fascicular and right bundle branch blocks in Irish Wolf hounds with dilated cardiomyopathy (DCM). They suggested that certain ECG abnormalities, especially atrial fibrillation and/or progressive ventricular and atrial dilatation, might be the indicators of DCM in Wolf hounds,

2.5 Radiography in cardiac disorders

Fisher (1967) reported that in primary right sided heart failure, radiography might revealed lung changes and would demonstrate an enlargement particularly of the right side of the heart.

Wyburn and Lawson (1967) stated that dilation of the left atrium was considerably more common in dogs than dilatation of the right atrium. Enlargement of either atria were always due to dilatation rather than hypertrophy. The left ventricle was the cardiac chamber most commonly involved in hypertrophy or dilatation.

Suter and Chan (1968) stated that pulmonary oedema was recognised radiographically by an alveolar density characterized by ill defined, fluffy margins of the infiltrate fading into the surrounding unaffected lung, tendency

of the infiltrate to coalesce and presence of "air-bronchograms" and "air"alveolograms".

Ettinger and Suter (1970) stated that transudation of fluid into the pleural space indicated impaired lymphatic drainage due to pulmonary hypertension or more frequently, elevated systemic venous pressure and was thus seen in both left and right heart failure.

Lombard and Goldschmidt (1980) reported that pleural effusion and right atrial enlargement were evident on radiographs of a dog with primary fibroma in the right atrium.

Calvert *et al.* (1982) reported that in congestive cardiomyopathy in twenty Dobermann pinschers, all the dogs had some degree of left atrial and left ventricular enlargement evident on radiography.

Thomas *et al.* (1984) reported pleural effusion and mild to moderate cardiomegaly on radiographs of a dog with constictive pericardial disease.

Thomas (1987) reported that in eight cases of congestive cardiac failure in Cocker spaniels, 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 the cases.

Fagin (1988) stated that radiographic signs of left sided cardiac silhouette enlargement were loss of the caudal cardiac waist, elongation and

straightening of the caudal cardiac border, separation of the main stem bronchi with compression of the left main stem bronchus, elevation of trachea and increased triangular density representing the left atrium in the region of the hilus.

Thoracic radiographs provided essential information about over all cardiac size and shape, enlargement of all individual cardiac chambers and the presence of aneurysmal or post stenotic dilatations. radiographs also indicated the size of the pulmonary arteries and veins (Goodwin and Lombard, 1990).

Radiographic findings of different heart worm-infected dogs suggested right heart enlargement, lung oedema, pulmonary knob and whole heart enlargement (Pattarakosol *et al.*, 1991).

Elwood *et al.* (1993) reported that out of ten cases of bacterial endocarditis in dogs, thoracic radiographs showed no abnormalities in 40 per cent, cardiomegaly in 40 per cent and or an alveolar pulmonary pattern in 30 per cent.

Burk and Ackerman (1996) stated that on lateral radiograph, the silhouette of the enlarged left atrium produced a "wing-shaped" shadow caudal to the tracheal bifurcation and dorsal to the caudal venacava.

Freeman *et al.* (1996) after studying nine Dalmatian dogs with idiopathic dilated cardiomyopathy stated that radiography revealed generalized

cardiomegaly and pulmonary oedema. Pleural effusion was not detected in any of the dogs.

Thoracic radiograph included global enlargement of the cardiac silhouette, marked elevation of the trachea and wide pulmonary arteries that tapered towards the periphery in a six-week old puppy having persistent left cranial venacava associated with multiple congenital anomalies (Del Palacio *et al.*, 1997).

Thoracic radiographs did not detect specific changes attributable to myocardial infarction, but were useful for evaluating associated pulmonary and cardiac abnormalities (Driehuys *et al.*, 1998).

2.6 Clinico-pathology

2.6.1 Haematology in cardiac disorders

Bacterial endocarditis was frequently associated with an increase in total white blood cell count, principally polymorphonuclear leucocytes and band cells (Ettinger and Suter, 1970).

Eyster *et al.* (1977) reported a PCV of 55 per cent and Hb of 19 g% in a dog with tetralogy of Fallot.

In most anaemias the erythrocyte sedimentation rate (ESR) was accelerated due to the small number of cells that could settle more easily in the large volume of fluid (Benjamin, 1998).

Baba *et al.* (1981) reported azotemia and marked neutrophilic leukocytosis with a left shift in a dog with bacterial endocarditis.

A complete blood count (CBC) of a chronic renal failure patient will reveal normochromic, normocytic anaemia with neutrophilia and lymphopenia (Mikiciuk *et al.*, 1989).

Goodwin and Lombard (1990) stated that diseases like tetralogy of Fallot and reversed Patent ductus arteriosus (PDA) often induced a polycythemia (PCV >55).

Wilbanks (1992) reported that the haematological parameters (WBC, RBC, Hb, PCV, MCV, MCH, MCHC, DLC) were within the reference range in a 13 year old spayed female Schnauzer presented with complaints of lethargy, fainting, cyanosis and abdominal enlargement. The dog was having a complete atrioventricular (third degree) heart block.

Elwood *et al.* (1993) suggested that haematological and biochemical abnormalities in canine bacterial endocarditis were non-specific and were attributed to the effects of septicaemia, embolism or chronic inflammation. They observed neutrophilia, monocytosis and thrombocytopenia.

Roger (1995) stated that in anaemia there would be reduction in red cell mass.

Haematology was normal in a six week old puppy with persistent left cranial venacava associated with multiple congenital anomalies (Del Palacio, 1997).

Premalatha *et al.* (1997) reported that there were no significant haematological changes between dogs with atrial fibrillation and control animals without heart disease.

White *et al.* (1997) reported that routine haematology and biochemistry were unremarkable in a case of subvalvular aortic stenosis and mitral dysplasia in a Golden Retriever.

Aird (2000) reported that endogenous production of erythropoietin was low in chronic renal failure and it would result in decreased production of haemoglobin.

In human beings anaemia is considered as the most important factor for the development of left ventricular hypertrophy in uraemic patients (Berweck *et al.*, 2000).

In human beings, haemoglobin levels below 10 g/dl could lead to left ventricular (LV) hypertrophy/LV dilation and a lower quality of life (Foley *et al.*, 2000).

Harvey (2000) stated that a mild to moderate non regenerative anaemia often accompanied chronic inflammatory and neoplastic disorders and the

anaemia was generally normocytic, but might be microcytic in long-standing cases. He also reported that Mean Corpuscular Volume (MCV) was usually in the low end of the reference range, but might occasionally be slightly less than the reference range.

Anaemia and hypertension play an important role in the pathogenesis of left ventricular hypertrophy as well as in the development of cardiac dysfunction (Jeren-Strujic *et al*., 2000).

2.6.2 Serum biochemistry in cardiac disorders

The serum glutamic oxaloacetic transaminase (SGOT) and serum glutamic pyruvic transaminase (SGPT) were greater than 60 SFunits when myocardial necrosis was present (Ettinger and Suter, 1970).

Knob and Seidl (1980) measured serum activities of total creatine kinase (CK) and CK isoenzymes in 103 healthy dogs. Normal values (U/litre) were total CK 14.4-83.6, CK-BB (Brain type) 6.1-77.1, CK-MM (muscle type) 1.3-50.3, and the CK-MB (myocardial type) was not detected (<1.0 U/litre).

Pozza *et al.* (1983) studied various enzymes in 21 normal dogs of several breeds, aged 4 months to 13 years. The following mean values were obtained (IU/L) AST - 15.61, ALT - 17.6, CK - 22.71, CK isoenzyme MB 7.33, Lactic dehydrogenase (LD) 56.04, bilirubin 0.09 mg /dl, creatinine 1.13 mg/dl.

Fleming *et al.* (1989) stated that hyperkalemia was a common finding in acute renal failure and could lead to life threatening cardiotoxicity and early death.

In domestic species, CK isoenzymes analysis has not been shown to be of significant value (Hoffmann, 1990).

Wilbank (1992) reported that the serum chemistry profile (creatinine, albumin, globulin, A:G ratio, SGPT, CPK, sodium and potassium were within the reference range in a 13 year old spayed female Schnauzer presented with complaints of lethargy, fainting, cyanosis and abdominal enlargement. The dog was having a complete atrioventricular block. The normal values for the above parameters were creatinine 1 to 2.0 (mg/dl), albumin 2.3 to 3.8 (g/dl), globulin 1.8 to 5.2 (g/dl), A:G ratio 0.8 to 2.0, ALT 1 to 150 (U/L), CPK 10 to 400 (U/L), sodium 140 to 155 (MEq/l) and potassium 4.3 to 5.5 (MEq/l).

In human medicine serum CK isoenzymes serve as sensitive and specific indicators of cardiac infarction (Moss and Henderson, 1994).

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.

Premalatha *et al.* (1997) reported significant increase in serum potassium and creatine kinase, muscle brain (CK-MB), a heart specific enzyme in 10 dogs with atrial fibrillation.

Abbot (1998) reported that in congestive heart failure the renin-angiotensin-aldosterone system caused vasoconstriction, potentiated adrenergic arm of the autonomic nervous system and promoted sodium and water retension.

As a result of the short serum half-life the CK activity rapidly returns to normal after a muscle damaging incident (Hoffmann and Kramer, 1999).

2.7 Ultrasonography in cardiac disorders

Ecchocardiographic changes in chronic degenerative valvular disease included left atrial and left ventricular dilation left ventricular eccentric hypertrophy, hyperdynamic systolic function and a deformed mitral valve (Kienle and Thomas, 1995).

Freeman *et al.* (1996) reported marked diminished contractibility (fractional shortening ranged from 4 to 17%; mean, 11%) in Dalmatians with idiopathic dilated cardiomyopathy. The size of the left atrium ranged from 2.49 to 4.50 cm (mean, 3.83 cm), and the aortic size ranged from 2.00 to 3.20 cm (mean, 2.25 cm). The internal dimension of the left ventricle ranged from 5.53 to 8.20 cm (mean, 6.97 cm) in diastole and 4.96 to 7.60 cm. (mean, 6.22 cm) in systole. The thickness of the interventricular septum ranged from 0.51 to 0.80 cm (mean, 0.72 cm) in diastole and 0.56 to 1.08 cm (mean, 0.81 cm.) in systole. The left ventricular free wall thickness ranged from 0.66 to 1.10 cm (mean, 0.79 cm) in diastole and 0.60 to 0.94 cm (mean, 0.84 cm) in systole.

Brownlie and Cobb (1999) reported that seven out of eight dogs with dilated cardiomyopathy showed increase in left atrial (LA) diameter and LA/AO. They also showed a slight or marked increase in left ventricular systolic and diastolic diameters.

Bossabaly *et al.* (1993) reported a thin walled, poorly contracting left ventricle with a moderately enlarged left atrium (5.1 cm) as observed from the right parasternal short-axis view in a Dobermann pinscher with aortic body carcinoma and myocardial infarction. The diastolic diameter of the left ventricle, obtained from the right parasternal short axis M-mode recording, was enlarged (6.1cm) and fractional shortening was calculated at 15 per cent. The interventricular septum and left ventricular free wall during diastole measured 0.80 and 0.85 cms respectively.

2.8 Autopsy findings in cardiac disorders

Postmortem findings reported in dilated cardiomyopathy were those of congestive heart failure. All chambers, particularly the left ventricle were markedly dilated and hypertrophied. The atrio-ventricular rings were dilated (the endocardium may be opaque due to subendocardial fibrosis and there may be atrial thrombosis). In mitral valvular insufficiency anatomically, there was an enlarged annulus, short thick leaflets, short thickened chordae tendineae, upward malposition of atrophic or hypertrophic papillary muscles, and enlargement of the left atrium and ventricle. There was also diffuse endocardial fibrosis (Robinson and Maxie, 1993).

Materials and Methods

3. MATERIALS AND METHODS

The study was conducted in the Department of Clinical Medicine, College of Veterinary and Animal Sciences, Mannuthy for a period of four semesters, during the years 1998 to 2000.

Canine patients brought to the Veterinary College Hospital, Mannuthy and University Veterinary Hospital, Kokkalai were used for the study. They were screened for cardiac involvement by detailed clinical examination.

Standard electrocardiogram patterns were worked out from sixty normal animals. Control animals were grouped according to different age groups (below one year and above one year) and breeds (German Shepherd, Dobermann pinscher, Dachshund, Spitz and Mongrel), so that six animals were there in each group.

Based on the clinical signs and electrocardiogram abnormalities, 13 dogs were classified into three groups (Table 1).

Group I - Congestive heart failure

Group II - Ventricular enlargement secondary to anaemia

Group III - Electrolyte imbalance due to renal disease

3.1 Parameters studied

1. Signalment and history
2. Clinical examination

3. Electrocardiography

4. Radiography

5. Clinical pathology

A. Haematology

a. Packed cell volume (PCV) (per cent)

b. Haemoglobin (Hb) (g/dl)

c. Total erythrocyte count (TEC) (x 10^6/mm^3)

d. Erythrocyte sedimentation rate (ESR) (mm/30 min)

e. Total leucocyte count (TLC) (/mm^3)

f. Differential leucocyte count (DLC) (per cent)

g. Erythrocytic indices

(i) Mean corpuscular volume (MCV) (fl)

(ii) Mean corpuscular haemoglobin (MCH) (pg)

(iii) Mean corpuscular haemoglobin concentration (MCHC) (per cent)

B. Serum biochemistry

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

b. Alanine aminotransferase (ALT) (IU/L)

c. Serum sodium and potassium (mEq/l)

d. Serum total protein (g/dl)

e. Serum albumin (g/dl)

f. Serum creatinine (mg/dl)

g. Albumin : Globulin ratio (A:G ratio)

6. Ultrasonography

7. Autopsy

3.2 Procedures adopted

3.2.1 Clinical examination of the patient

Detailed history and results of clinical examination of the patient were recorded in the proforma (Annexure I). Clinical examination of the patient was conducted as per the modified protocol suggested by Ettinger and Suter (1970).

3.2.2 Electrocardiography

Electrocardiogram of the patients were recorded by CARDIART-108 ECG machine (BPL). Three standard bipolar limb leads (I, II, III), three augmented unipolar limb leads (aVR, aVL, aVF) and precordial chest leads (CV_6LL, CV_6LU and V_{10}) were used for the study. The machine was standardised before positioning the dogs in right lateral recumbency. The electrodes were attached to the specific points on the skin after applying electrode gelly. ECG was recorded at a paper speed of 25 mm and 50 mm per second.

The sensitivity control switch was adjusted to 1 or 0.5, so as to adjust the complexes within the ruled area of the ECG paper (Bolton, 1975).

3.2.3 Radiography

Thoracic radiographs were taken after the patients were kept in right lateral recumbency. Plain radiographs were used for cardiac evaluation. Size

of the X-ray film and radiographic factors varied depending upon the size and chest girth of the patients.

3.3 Clinico-pathology

3.3.1 Haematology

Blood samples (3-5 ml) were collected from saphenous/cephalic veins of each animal with clinical signs of cardiac involvement and six healthy animals selected randomly from the control group in a sterile vial with EDTA (1 mg per ml of blood) for the haemogram. The values of haemogram of these six healthy animals were used for comparison with that of the diseased. Complete blood count was done as per Schalm *et al.* (1975).

3.3.2 Serum Biochemistry

Blood samples (approximately 10 ml) were collected from saphenous/ cephalic veins of each animal having clinical signs of cardiac involvement and six control animals in a sterile screw capped test tube without anticoagulant for serum separation. The mean serum biochemical values of the six apparently healthy animals were used for comparison with that of the diseased.

All the biochemical analyses were carried out by using Photometer 5010 (Boehringer Mannheim) under standard conditions of operation.

The following tests were conducted according to the corresponding procedures.

a. Creatine kinase (CK): The method used was the "optimized standard method" as per the recommendations of the Dentsche Gesellschaft fir Klinische Chemie (Anon., 1977).

b. Serum alanine aminotransferase (ALT): Scandinavian Committee on Enzymes (SCE) method (Anonymous, 1974).

c. Serum sodium and potassium : Flame Photometer (Oser, 1971).

d. Serum total protein : Biuret method (Weichselbaum, 1946).

e. Serum albumin: Bromocresol-green method (Doumas, 1971).

f. Serum globulin and A:G ratio: Calculated from values of total protein and albumin (Benjamin, 1998).

g. Serum creatinine: Jaffe method, without deproteinization (Bartels, 1971).

3.4 Ultrasonography

Dogs with signs of cardiac involvement were subjected for echocardiography randomly. 2-D echocardiography was done using mechanical real time sector scanner at the frequencies of 3.5 MHz and 5.0 MHz. The transducer location or windows used were right parasternal long axis and short axis views, left apical parasternal location (apical two chamber and four-chamber views) and left cranial parasternal location (long axis and short axis views).

3.5 Autopsy

One dog that died of congestive heart failure was subjected to post-mortem examination.

3.6 Statistical analysis

Data obtained were analysed statistically as per Snedecor and Cochran (1994).

Results

4. RESULTS

4.1 Occurrence

Out of the total 1000 canine cases brought with various conditions and screened at University Veterinary Teaching hospitals at Mannuthy and Kokkalai, during the period 1998 to 2000, 13 dogs were found to have electrocardiogram abnormalities (1.3%).

4.2 Signalment

4.2.1 Breed, Age and Sex

Out of the 13 cases with electrocardiogram abnormalities, seven cases (53.85%) were with congestive heart failure (CHF) and three cases (23.08%) were with cardiac involvement secondary to anaemia and three cases (23.08%) were with electrolyte imbalance due to renal disease. The total male:female ratio was 12:1.

Out of the six cases of secondary cardiac involvement, three cases (50%) were with anaemia and three cases (50%) were with renal disease.

Out of the seven dogs with congestive heart failure, two were Dachshunds (28.57%), three were Spitz (42.86%) and two were Dobermann pinschers (28.57%). All the dogs with CHF were males. Their age ranged from six to eleven years.

Out of the three cases with anaemia two were Dobermann Pinschers (66.67%) and one was Labrador (33.33%). All the dogs of this group were males. Their age ranged from five months to two years.

Among the three cases of electrolyte imbalance due to renal disease, two dogs were Mongrel (66.67%) and one was a German Shepherd (33.33%). Mongrels were males and German Shepherd was a female. Their age ranged from seven to nine years.

4.3 Clinical signs

4.3.1 Group 1

Four dogs out of seven (Case No. 1, 2, 3 and 4) exhibited orthopnoea, cough and during auscultation crackles were heard from the lung area. Exertional dyspnoea and exercise intolerance were present in all the cases. Two dogs (Case No. 1 and 2) had grade IV/VI holosystolic murmur of "whooping quality" (seagull murmur) best heard at the left caudal sternal border. Those two dogs had intermittent syncopic episodes. One dog with ventricular tachycardia had syncope while climbing the steps (Case No. 3). Three dogs out of seven had ascites (Case No. 5, 6 and 7). All the dogs were anorectic. Pulse deficit was noticed in two cases with ventricular tachycardia (Case No. 3 and 4). A jerky pulse with rapid upstroke and downstroke was noticed in two dogs (Case No. 1 and 2). Gallop rhythm could be appreciated by auscultation in two dogs (Case No.1 and 2). The dogs with ventricular tachycardia (Case No.3 and 4), upon auscultation showed paroxysms of rapid

regular beats. Cannon 'a' waves were observed in the jugular vein of these dogs. Two dogs exhibited vigorous open mouth breathing and extension of neck on little exertion (Case No.1 and 6). These dogs were standing with abducted elbows. Left ventricular heave and precordial thrill were noticed in one dog between fourth to sixth intercostal space at the ventral portion on the left side (Case No.1). One dog (Case No.6) had muffled heart and lung sounds upon auscultation of thorax. Paroxysmal dyspnoea was observed in four dogs (Case No.1, 2, 3 and 4). Expectoration of pink stained frothy fluid was observed in one case (Case No.1). In one dog cardiac cachexia was observed (Case No.6). The mean ± SE values of the clinical parameters of this group were tabulated (Table 2).

4.3.2 Group II

Two dogs out of three of this group had inappetance and stunted growth (Case No.8 and 9). One dog was anorectic (Case No. 10). All the dogs were anaemic and had pale visible mucous membranes. All the three dogs were weak and showed respiratory distress upon exertion. In one dog, the intensity of cardiac sounds and area of cardiac auscultation were increased (Case No.10). Two dogs had haemic murmurs on auscultation of the heart (Case No.8 and 10). They had hyperkinetic pulse. There was muffling of heart sounds in one dog (Case No.9). This dog was severely infected with tape worms (*Dipyllidium caninum*). The mean ± SE values of the clinical parameters of this group were tabulated (Table 2).

4.3.3 Group III

All the dogs of this group had anorexia and polydipsia. Two had oliguria (Case No.11 and 12). One dog had anuria (Case No.13). Vomiting and diarrhoea with melena were observed in all the dogs of this group. All the dogs were weak and with varying degrees of dehydration. They were anaemic and haemic murmurs were heard upon auscultation of the heart. Aortic thudding was heard on auscultation of the flank. The pulse in all animals were hyperkinetic. One had stomatitis and ulcers in the mouth with necrosis of the tip of the tongue (Case No.11).

The mean ± SE values of the clinical parameters of this group were tabulated (Table 2).

4.4 Electrocardiography

4.4.1 Normal values of Electrocardiographic parameters - control group

The mean ± SE values of various parameters like heart rate, MEA (mean electrical axis), 'P' wave amplitude and duration, P-R interval, 'R' wave amplitude, 'QRS' duration, 'Q-T' interval, 'S-T' segment, 'T' wave amplitude, 'Q' wave amplitude and 'S' wave amplitude in different lead systems (I, II, III, aVR, aVL, aVF, V10, CV6LL and CV6LU) of different breeds (Spitz, German Shepherd, Dobermann pinscher, Dachshund and Mongrel) of two age groups (below one year and above one year of age) were tabulated (Table 3 to 23).

4.4.2 Electrocardiographic parameters in clinical groups

4.4.2.1 Group I

The Dachshund with left sided congestive heart failure (Case No.1) had a heart rate of 144 bpm and a mean electrical axis of 70° in the frontal plane. The 'P' wave was notched in all the leads (I, II, III, avR, avL, avF, V_{10}, CV_6LU and CV_6LL) with a duration of 0.08 sec uniformly. The 'P-R' interval was 0.12 sec in lead II. The amplitude of 'Q' wave was 1.0 mv in lead I, 0.8 mv in lead II and 0.7 mv in avF. The 'R' wave amplitude was 2.1 mv in lead I, 5.0 mv in lead II, 2.3 mv in lead III and 3.4 mv in avF. The Q-T interval was 0.2 sec and the QRS duration was 0.04 sec in lead II. There was 'S-T' segment coving in lead II with deep negative 'T' waves. The amplitude of 'T' wave was 0.7 mv in lead II (Plate 1 and 2).

The other Duchshund dog with right sided congestive heart failure (Case No.6) had a heart rate of 156 bpm and mean electrical axis of –90° in the frontal plane. Deep 'S' waves were evident in lead II, III, avF, CV6LL and CV6LU with an amplitude of 1.5, 1.6, 1.5, 1.5 and 2.0 mvs respectively. The R/S ratio in CV6LU was o.3 mv. The 'R' amplitude in lead II, III, avF and CV6LU were 1.0, 1.0, 1.0 and 0.6 mvs respectively. In lead II the 'P' wave amplitude and duration were 0.1 mv and 0.4 sec. The 'P-R' interval was 0.1 sec and the QRS duration was 0.04 sec (Plate 7).

Among Spitz with clinical signs suggestive of left sided congestive cardiac failure (Case No.2, 3 and 4), two dogs had ventricular tachycardia with

a ventricular rate of 100 and 140 bpm respectively. The 'P' waves were not associated with the QRS complex in one (Case No.4) and in the other (Case No.3) it was hidden in the QRS complex. The QRS complexes were wide and bizzare. There was no relationship between 'P' waves and QRS complexes. Since the QRS complexes were of the same shape it was considered as unifocal ventricular tachycardia. 'T' waves were directed opposite to the QRS complexes. Since the major QRS deflection was negative in lead II, the ectopic focus was in the left ventricle (Plate 10).

One dog had no dysrrhythmias (Case No.2). The heart rate of that animal was 150 bpm with a mean electrical axis (MEA) of 83° in the frontal plane. The rhythm was normal sinus rhythm. The amplitude of 'P' wave in lead II was 0.4 mv with a duration of 0.04 sec. The 'P-R' interval was 0.12 sec in lead II. The amplitude of 'R' wave was 1.0 mv in lead I, 4.0 mv in lead II, 3.4 mv in lead III, 3.7 mv in avF and 4.6 mv in CV_6LU. The 'Q' wave was 0.5 mv and 0.4 mv in lead I and II respectively. The QRS duration was 0.04 sec in lead II. There was 'S-T' segment coving with deep negative 'T' waves. The amplitude of 'T' wave was about 0.7 mv.

The Dobermann pinschers with congestive heart failure had clinical signs suggestive of right sided failure (Case No.5 and 7). The QRS complexes in lead II was 0.1 secs in duration. The QRS complex was positive in avR, avL and CV_5RL. It was associated with 'P' wave in all the leads. The 'P-R' interval was constant with a duration of 0.1 sec (Case No.5) and 0.12 sec (Case No.7) in

lead II. The lead V10 showed 'W' pattern. Both the dogs had a normal sinus rhythm with a heart rate of 160 and 100 bpm respectively. The mean electrical axis in frontal plane was 120° and –90° (Case No.5 and 7 respectively) indicating right axis deviation. The 'S' waves were large and wide in lead I, II, III, avF and Cv_6LU with an amplitude of 0.3, 1.5, 1.1, 1.1, 1.2 mvs respectively (Case No.5) and 0.2, 1.5, 1.2, 1.3 and 1.5 mvs respectively (Case No.7) (Plate 11, 12, 13 and 14).

4.4.2.2 Group II

In this group two dogs (Case No.8 and 9) had right ventricular enlargement and one dog had left ventricular enlargement (Case No.10).

In the dogs with right ventricular enlargement the 'P' wave amplitude and duration were 0.1 mv and 0.04 secs. The QRS duration was 0.04 sec. The 'P-R' interval was 0.16 sec (Case No.8) and 0.12 sec (Case No.9) in lead II. The amplitude of 'R' wave was 0.4 mv (Case No.8) and 1.0 mv (Case No.9) in lead II. The prominent change noticed were deep 'S' waves in lead II, III and avF with amplitudes of 0.5, 0.7 and 0.7 mvs respectively (Case 8) and 0.5, 0.6 and 0.6 mvs respectively (Case No.9). The 'S' wave in lead CV_6LU was 1.0 mv (Case No.8) and 0.9 mv (Case No.9). The amplitude of 'R' wave in lead I and III were only 0.35 and 0.3 mvs respectively (Case No.8) and 0.2 and 0.7 mvs respectively (Case NO.9). The heart rate was 132 bpm (Case No.8) and 108 bpm (Case No.9) with a normal sinus rhythm. The mean electrical axis

(MEA) in the frontal plane were –90° (Case No.8) and 70° (Case No.9) (Plate 16).

In the dog with left ventricular enlargement (Case No.10) the heart rate was 130 bpm with a normal sinus rhythm. The mean electrical axis (MEA) in the frontal plane was 90°. In lead II amplitude and duration of 'P' wave were 0.2 mv and 0.04 secs respectively. The 'P-R' interval in lead II was 0.12 sec. The 'Q' wave amplitudes in lead I and II were 0.4 and 0.6 mv respectively. The 'QRS' duration was 0.04 sec. The amplitude of 'R' wave in lead I, II, III, avF and CV_6LU were 0.5, 3.6, 3.0, 3.4 and 4.0 mvs respectively. The 'Q-T' interval was 0.2 sec.

4.4.2.3 Group III

All the dogs of this group had a normal sinus rhythm with a heart rate of 72, 110 and 84 bpm (Case No.11, 12 and 13) respectively. The amplitude of 'P' wave was 0.1 mv and duration 0.04 sec in lead II for all the dogs. The 'P-R' interval was 0.12 sec. The QRS duration was 0.04 sec. The amplitude of 'R' wave in lead II was 2.3 mv (Case No.11), 1.6 mv (Case No.12) and 2.2 mv (Case No.13). The 'Q-T' interval was 0.24 sec (Case No.11) and 0.28 sec (Case No.13). The prominent change was peaking of the 'T' wave in lead II tracing of all the dogs. The 'T' wave amplitude in lead II was 0.5 mv (Case No.11), 0.3 mv (Case No.12) and 0.5 mv (Case No.13). The 'R' wave amplitude in CV_6LU was 2.6 mv (Case No.11), 2.4 mv (Case No.12) and 3.0 mv (Case No.13). The 'T' wave amplitude in CV_6LU were 1.8, 0.4 and

1.8 mv (Case No.11, 12 and 13) respectively. In CV_6LL the 'R' amplitude was 1.8 mv and that of 'T' was 1.5 mv (Case No.11) (Plate 18, 19 and 20).

In Case No.12 the chest leads like V10, CV_6LL and CV_5RL, the amplitudes of 'R' wave were 0.3, 0.9, 0.6 and that of 'T' wave were 0.3, 0.6 and 0.6 respectively. The 'R' amplitude in CV_6LL was 2.1 mv and that of 'T' wave was 1.8 mv (Case No.13). The 'Q-T' interval was 0.24 sec in lead II (Case No.11). In the chest leads like CV_6LL the 'Q-T' interval was 0.28 seconds (Case No.11 and 13).

4.5 Radiography

4.5.1 Group I

In all the cases of congestive heart failure, cardiac enlargement was evident in the lateral radiograph.

The lateral radiograph of the cardiac silhouette showed an enlarged left atrium producing a " wing shaped " shadow caudal to the tracheal bifurcation and dorsal to the caudal venacava (Case No.1). There was marked tracheal elevation in some dogs (Case No.1, 3, 4, 6). The left ventricular enlargement was evident by straightened and more up right caudal cardiac margin (Case No.1) and the caudal border of the silhouette was straight and formed a sharp angle with the dorsal cardiac margin. There was accentuation of the convexity of the caudal cardiac border in some dogs (Case No.2, 3, 4). The tracheal

bifurcation was elevated and the thoracic trachea was parallel to the thoracic vertebral bodies (Case No.1) (Plate 3).

The thoracic trachea sloped dorsally from the thoracic inlet to its bifurcation (Case No.3). In some dogs there was increased contact between cardiac silhouette and the sternum (Case No.4, 5, 6 and 7) (Plate 15). In some dogs the cranial border of the cardiac silhouette was vertically oriented (Case No.5, 6 and 7) indicating right ventricular enlargement. The caudal lung lobe had interstitial (reticular) pattern of density (Case No.7). Pleural effusion was evident in the lateral radiograph in one dog (Case No.6).

4.5.2 Group II

In one dog the outline of the cardiac silhouette was smooth, suggesting pericardial effusion (Case No.9). Tracheal elevation was noticed at the base of the heart and there was accentuation of the normal convexity of the caudal cardiac margin (Case No.10).

4.5.3 Group III

In this group no changes were evident in the cardiac silhouette.

4.6 Clinico-pathology

4.6.1 Haematology

4.6.1.1 Total erythrocyte count ($x10^6/mm^3$)

The mean ± SE value of total erythrocyte count in healthy controls was 6.67 ± 0.36 with a range of 6 to 8 millions per cubic millimetre of blood (Table 24).

The total erythrocyte count of Group I animals had a mean ± SE value of 6.88 ± 0.37. This value was within the normal range (Table 25).

The mean ± SE value of Group II was 3.67 ± 0.17, and was considerably less when compared with the control group (Table 25).

The mean ± SE value of Group III was 3.67 ± 0.44, which was also notably less than that of the control group (Table 25).

4.6.1.2 Haemoglobin (g/dl)

he haemoglobin level of the healthy control group had a mean ± SE value of 13.67 ± 0.67 with a range of 12 to 16 g% (Table 24).

The haemoglobin level of Group I was 14.43 ± 0.37, which was within the normal range (Table 24). The mean ± SE value of haemoglobin of Group II was 7 ± 0.58, which was very less when compared with the control group (Table 25).

The haemoglobin level of group III had a mean ± SE value of 6 ± 0.58, which was also less when compared with the control group (Table 25).

4.6.1.3 Packed cell volume (per cent)

The packed cell volume of the control group had a mean ± SE value of 43.33 ± 2.25, with a range of 40 to 50 per cent (Table 23).

The packed cell volume of the Group I was 44.43 ± 1.82, within the range of the control group (Table 24).

The packed cell volume of the Group II was 23.33 ± 1.33 and Group III was 26.67 ± 1.67, which were considerably less when compared with the controls (Table 24).

4.6.1.4 Mean corpuscular volume (MCV) (fl)

The mean corpuscular volume of the control group had a mean ± SE value of 65.04 ± 0.50 with a range of 60 to 72.72 (Table 23).

The mean MCV of the Group I and Group II, had a mean ± SE value of 64.88 ± 1.52 and 60.95 ± 3.04, both were within the range of the control group (Table 24). The MCV of the group III was 57.14 ± 11.98, which was less when compared with the control group (Table 24).

4.6.1.5 Mean corpusculr haemoglobin (MCH) (pg)

The mean MCH of the control group had a mean ± SE value of 20.54 ± 0.34, with a range of 20 to 21.8 (Table 23).

The MCH of Group I was 21.19 ± 0.83 within the range for the controls (Table 24).

The MCH for the Group II had a mean ± SE value of 18.21 ± 0.89 and was less when compared with the controls (Table 24).

The MCH for the group III had a mean ± SE value of 22.06 ± 1.04 and was more or less within that of the control groups (Table 24).

4.6.1.6 Mean corpuscular haemoglobin concentration (MCHC) (per cent)

The MCHC in control group had a mean ± SE value of 31.59 ± 0.59, with a range of 30 to 34.28 (Table 23).

The MCHC of Group I had a mean ± SE value of 32.63 ± 0.82, which was within the range for the control group (Table 24).

The mean cell haemoglobin concentration of Group II and Group III had a mean ± SE value of 29.95 ± 1.37 and 30.00 ± 1.15 respectively, which were within the range for the control group (Table 24).

4.6.1.7 Total leucocyte count (TLC) (/mm^3)

The mean ± SE value of TLC of the control group had a mean ± SE value of 10,833.33 ± 945.75 with a range of 9000 to 14,000 per cubic millimetre of blood (Table 23).

The Group I, II and III had a mean ± SE value of 12,428.57 ± 751.42; 11,000 ± 577.35; 12,000 ± 577.35 within the range for the control group (Table 24).

4.6.1.8 Differential leucocyte count (per cent)

a. Neutrophils

The mean ± SE value of the control group was 70.33 ± 1.69 percentage with a range of 65 to 76 per cent (Table 23).

The mean ± SE values of the group I and II were 73.57 ± 1.45 and 75.67 ± 0.67 respectively and they were within the range for the controls (Table 24).

The mean ± SE values of the Group III was 84 ± 2, which was considerably higher than that for the control group (Table 24).

b. Lymphocytes

The mean ± SE value for the control group was 23.17 ± 1.08, with a range of 20 to 26 per cent (Table 23).

The mean ± SE values for Group I and II are 21.86 ± 0.94 and 20 respectively, which lies within the range for the control group (Table 24).

The mean ± SE value for Group III was 13 ± 1.53 and showed a decrease when compared with the control group (Table 24).

c. Monocytes

The mean ± SE value for the control group was 3.33 ± 0.56, with a range of 2 to 5 per cent (Table 23).

The mean ± SE value for group I, II and III are 2.86 ± 0.46, 3 and 2 respectively. All the values were within the range for the control group (Table 24).

d. Eosinophils

The mean ± SE value for the control group was 3.17 ± 0.60, with a range of 2 to 5 per cent (Table 23).

The mean ± SE value of Group I, II and III are 1.86 ± 0.40, 1.33 ± 0.67 and 1 ± 0.58 respectively, which were less when compared to the control group (Table 24).

4.6.1.9 Erythrocyte sedimentation rate (ESR) (mm/30 min)

The mean ± SE value of the control group was 3 ± 0.73, with a range 1 to 6 mm/30 minute (Table 23).

The mean ± SE value of Group I and II animals were 3.43 ± 0.37 and 5 ± 1.73 respectively which were within the range for the control group (Table 24).

The mean ± SE value of Group III animals (9.33 ± 0.67), was greater than that for the control group (Table 24).

4.6.2 Serum biochemistry

4.6.2.1 Creatine kinase (CK) (U/L)

The mean ± SE value for the control group of animals was 42.5 + 5.46, with a range of 20 to 60 U/L (Table 25).

The Group I, II and III animals had a mean ± SE value of 46.14 ± 2.19, 43.33 ± 3.33, 33.33 ± 1.76, all within the range for the control group (Table 26).

4.6.2.2 Alanine amino transferase (ALT) (IU/L)

The mean ± SE value for the control group of animals was 17 ± 1.78, with a range of 12 to 24 IU/L (Table 25).

The group I, II and III animals had a mean ± SE value of 19.43 ± 0.81, 18.33 ± 1.66, 16.67 ± 0.88 IU/L, all within the range for the control group (Table 26).

4.6.2.3 Sodium (mEq/l)

The mean ± SE value for the control group of animals was 145 ± 1.41, with a range of 140-150 MEq/l (Table 25).

The group I animals had a mean ± SE value of 174.14 ± 2.38, which was considerably higher than that of the control group (Table 26).

The Group II and III animals had a mean ± SE value of 143 ± 1.73 and 144.33 ± 2.73 respectively which was within the range for the control group (Table 26).

4.6.2.4 Potassium (mEq/l)

The mean ± SE value for the control group of animals was 4.33 ± 0.24, with a range of 3.8-5.3 MEq/l (Table 25).

The group I and II animals had a mean ± SE value of 4.80 ± 0.08 and 4.5 ± 0.28 respectively, which were within the range for the control group (Table 26).

The Group III animals had a mean ± SE value of 7.67 ± 0.33 which was considerably higher when compared with that of the control group (Table 26).

4.6.2.5 Total protein (g/dl)

The mean ± SE value for the control group of animals, was 6.7 ± 0.15, with a range of 6.2 to 7.2 g/dl) (Table 25).

The mean ± SE value for the group I and III animals were 6.54 ± 0.18 and 6.07 ± 0.07 respectively, which were within the range for the control group (Table 26).

The mean ± SE value for the group II animals was 5.73 ± 0.93, which was less than that of the control group (Table 26).

4.6.2.6 Albumin (g/dl)

The mean ± SE value for the control group of animals, was 3.73 ± 0.07, with a range of 3.6 to 4.0 g/dl (Table 25).

The mean ± SE value for group I animals was 3.91 ± 0.07, which was within the range for the control group (Table 26).

The mean ± SE value for the Group II and III animals were 3.2 ± 0.70 and 1.9 ± 0.06, which were less when compared with the value of control group (Table 26).

4.6.2.7 Albumin: Globulin ratio (A:G)

The mean ± SE value for the control group of animals was 1.27 ± 0.07, with a range of 1.11 to 1.58 (Table 25).

The mean ± SE value for the group I and II animals were 1.54 ± 0.13 and 1.15 ± 0.19 respectively, which were within the range for the control group (Table 26).

The group III animals had a mean ± SE value of 0.46 ± 0.03, which was considerably less than that of the control group (Table 26).

4.6.2.8 Creatinine (mg/dl)

The mean ± SE value for the control group of animals was 0.93 ± 0.12, with a range of 0.5-1.3 (Table 25).

The Group I and II animals had a mean ± SE value of 1.25 ± 0.08 and 0.93 ± 0.06, which were within the range for the control group of animals (Table 26).

The Group III animals had a mean ± SE value of 19.77 ± 1.29, which was very high when compared with that of the control group (Table 26).

4.7 Ultrasonography

The left apical four chamber view of the heart in one dog (Case No.1) revealed dilated left atrium, left ventricle and thickened mitral valve (Plate 4). The left parasternal short axis view revealed hypertrophied right ventricle in one dog (Case No.6) (Plate 8 and 9) and pericardial effusion in two cases (Case No.6 and 9) (Plate 17).

4.8 Autopsy findings

The post-mortem examination of the heart (Case No.1) revealed a dilated left atrium and ventricle, thick valvular leaflets and an enlarged annulus of the mitral valve (Plate 5 and 6).

Table 1. Experimental design and procedures adopted

Group	Condition	Case No.	Breed	Age	Sex	Procedures adopted
I	Congestive heart failure	1	Dachshund	10 years	Male	Clinical examination Electrocardiography Radiography Clinico-pathological investigation Ultrasonography
		2	Spitz	11 years	Male	
		3	Spitz	10 years	Male	
		4	Spitz	9 years	Male	
		5	Dobermann pinscher	8 years	Male	
		6	Dachshund	4 years	Male	
		7	Dobermann pinscher	6 years	Male	
II	Ventricular enlargement secondary to anaemia	8	Dobermann pinscher	6 months	Male	-do-
		9	Dobermann pinscher	5 months	Male	
		10	Labrador	2 years	Male	
III	Electrolyte imbalance due to renal disease	11	Mongrel	7 years	Female	-do-
		12	Mongrel	8 years	Male	
		13	German Shepherd	9 years	Male	

Table 2. Respiration rate, pulse rate and rectal temperature in dogs with ECG abnormalities (Mean ± SE)

Parameters	Congestive heart failure (n=7)	Ventricular enlargement secondary to anaemia (n=3)	Electrolyte imbalance due to renal disease (n=3)
Respiration rate (per minute)	37.85 ± 2.39	39 ± 3.21	28.33 ± 3.53
Pulse rate (per minute)	127.85 ± 11.91	123.33 ± 7.68	88 ± 11.71
Rectal temperature (°C)	38.69 ± 0.10	38.66 ± 0.168	37.77

Table 3 The mean ± SE values of various parameters in lead I of different breeds below one year of age (control group) (n=6)

Breed	P-du	P-amp.	P-R	R-amp.	QRS-du	Q-T	S-T-seg	T-amp.	Q-amp.	S-amp.
Spitz	0.027± 0.004 (0.02-0.04)	0.092± 0.008 (0.05-0.1)	0.097± 0.012 (0.06-0.14)	0.717± 0.159 (0.3-1.2)	0.027± 0.004 (0.02-0.04)	0.157± 0.004 (0.14-0.16)	0.100	0.108± 0.020 (0.1-0.2)	0.300± 0.103 (0.1-0.6)	0.017± 0.017
GSD	0.041	0.100	0.103± 0.008 (0.08-0.12)	0.233± 0.049 (0.1-0.4)	0.037± 0.004 (0.02-0.04)	0.200	0.100	0.100	0.183± 0.040 (0.1-0.3)	Absent
Dobermann	0.040	0.100	0.127± 0.008 (0.1-0.14)	0.417± 0.143 (0.1-1.0)	0.037± 0.004 (0.02-0.04)	0.200± 0.012 (0.16-0.24)	0.100	0.100	0.083± 0.031 (0.1-0.2)	Absent
Dachshund	0.033± 0.004 (0.02-0.04)	0.100	0.111± 0.008 (0.08-0.12)	0.750± 0.204 (0.1-1.6)	0.034± 0.004 (0.02-0.04)	0.160	0.100	0.092± 0.008 (0.05-0.1)	0.500± 0.137 (0.3-0.9)	0.033± 0.033
Mongrel	0.030± 0.004 (0.02-0.04)	0.092± 0.008 (0.05-0.1)	0.110± 0.004 (0.1-0.12)	0.640± 0.143 (0.2-1.0)	0.030± 0.004 (0.02-0.04)	0.177± 0.012 (0.14-02)	0.100	0.100	0.267± 0.067 (0.1-0.5)	Absent

P-du = 'P' wave duration, P-amp. = 'P' wave amplitude, P-R = 'P-R' interval, R-amp. = 'R' wave amplitude, QRS-du = QRS duration, Q-T = 'Q-T' interval, S-T-seg. = 'ST' segment, T-amp. = 'T' wave amplitude, Q-amp. = 'Q' wave amplitude, S-amp. = wave amplitude, Ranges for the above values are given in parenthesis

Table 4. The mean ± SE values of various parameters in lead I of different breeds above one year of age (control group) (n=6)

Breed	P-du	P-amp.	P-R	R-amp.	QRS-du	Q-T	S-T seg.	T-amp.	Q-amp.	S-amp.
Spitz	0.033± 0.004 (0.02-0.04)	0.101	0.150± 0.012 (0.1-0.2)	0.617± 0.127 (0.2-1.1)	(0.02-0.04) (0.02-0.04)	0.197± 0.016 (0.16-0.26)	0.100	0.100	0.167± 0.095 (0.2-0.6)	0.017± 0.017
GSD	0.037± 0.004 (0.02-0.04)	0.100	0.127± 0.008 (0.12-0.16)	0.400± 0.184 (0.12-1.3)	0.038± 0.004 (0.02-0.04)	0.233± 0.008 (0.2-0.24)	0.100	0.100	0.050± 0.034 (0.1-0.2)	Absent
Dobermann	0.040	0.100	0.117± 0.008 (0.08-0.14)	0.333± 0.078 (0.2-0.7)	0.040	0.195± 0.012 (0.16-0.22)	0.100	0.100	0.233± 0.092 0.1-0.6)	Absent
Dachshund	0.038± 0.004 (0.02-0.04)	0.092± 0.008 (0.05-0.1)	0.082± 0.016 (0.08-0.12)	0.883± 0.110 (0.6-1.3)	0.033± 0.004 (0.02-0.04)	0.193± 0.016 (0.16-0.26)	0.100	0.100	0.350± 0.043 (0.2-0.5)	Absent
Mongrel	0.040	0.093± 0.008 (0.05-0.1)	0.113± 0.008 (0.08-0.12)	0.217± 0.016 (0.2-0.3)	0.043± 0.004 (0.04-0.06)	0.187± 0.008 (0.16-0.2)	0.100	0.092± 0.008 (0.05-0.1)	0.067± 0.033	Absent

P-du = 'P' wave duration, P-amp. = 'P' wave amplitude, P-R = 'P-R' interval, R-amp. = 'R' wave amplitude, QRS-du = QRS duration, Q-T = 'Q-T' interval, S-T-seg. = 'ST' segment, T-amp. = 'T' wave amplitude, Q-amp. = 'Q' wave amplitude, S-amp. = wave amplitude, Ranges for the above values are given in parenthesis

Table 5. The mean ± SE values of various parameters in lead II of different breeds below one year of age (control group) (n=6)

Breed	P-du	P-amp.	P-R	R-amp.	QRS-du	Q-T	S-T seg.	T-amp.	Q-amp.	S-amp.
Spitz	0.031± 0.004 (0.02-0.04)	0.133+ 0.020 (0.1-0.2)	0.100± 0.012 (08-0.14)	1.617± 0.180 (09-2.1)	0.027± 0.004 (0.02-0.04)	0.160	0.100	0.192± 0.037 (0.1-0.3)	0.300± 0.115 (0.2-0.7)	0.183± 0.048 (0.1-0.3)
GSD	0.040	0.150± 0.020 (0.1-0.2)	0.103± 0.008 (0.08-0.12)	1.300± 0.171 (0.5-1.7)	0.037± 0.004 (0.02-0.4)	0.200	0.100	0.100	0.233± 0.071 (0.1-0.5)	0.033± 0.033
Dobermann	0.040	0.133± 0.020 (0.1-0.2)	0.127± 0.004 (0.1-0.14)	1.600± 0.269 (1.0-2.9)	0.040	0.200± 0.012 (0.16-0.24	0.100	0.133± 0.021 (0.1-0.2)	0.267± 0.049 (0.2-0.5)	0.050± 0.050
Dachshund	0.037± 0.004 (0.02-0.04	0.183± 0.016 (0.1-0.2)	0.113± 0.008 (0.08-0.12)	2.867± 0.151 (2.5-3.0)	0.033± 0.004 (0.02-0.04)	0.16	0.133± 0.021 (0.1-0.2)	0.100± 0.022 (0.05-0.2)	0.300± 0.068 (0.1-0.5)	0.100± 0.052 (0.1-0.3)
Mongrel	0.030± 0.004 (0.02-0.04)	0.117± 0.016 (0.1-0.2)	0.110± 0.004 (0.1-0.12)	2.083± 0.376 (0.6-2.8)	0.043± 0.008 (0.02-0.06)	0.177± 0.012 (0.14-0.2)	0.100	0.117± 0.017 (0.1-0.2)	0.483± 0.070 (0.3-0.7)	0.067± 0.042

P-du = 'P' wave duration, P-amp. = 'P' wave amplitude, P-R = 'P-R' interval, R-amp. = 'R' wave amplitude, QRS-du = QRS duration, Q-T = 'Q-T' interval, S-T-seg. = 'ST' segment, T-amp. = 'T' wave amplitude, Q-amp. = 'Q' wave amplitude, S-amp. = wave amplitude, Ranges for the above values are given in parenthesis

Table 6. The mean ± SE values of various parameters in lead II of different breeds above one year of age (control group) (n=6)

Breed	P-du	P-amp.	P-R	R-amp.	QRS-du	Q-T	S-T seg.	T-amp.	Q-amp.	S-amp.
Spitz	0.033± 0.004 (0.02-0.04)	0.167± 0.020 (0.1-0.2)	0.150± 0.012 (0.12-0.2)	1.900± 0.212 (1.2-2.7)	0.030± 0.004 (0.02-0.04)	0.203± 0.012 (0.16-0.26)	0.100	0.183± 0.031 (0.1-03)	0.117± 0.065 (0.1-0.4)	0.100± 0.037 (0.1-0.2)
GSD	0.037± 0.004 (0.02-0.04)	0.117± 0.016 (0.1-0.2)	0.127± 0.008 (0.12-0.16)	1.733± 0.220 (1.2-2.4)	0.037± 0.004 (0.02-0.04)	0.233± 0.008 (0.2-0.24)	0.100	0.200± 0.052 (0.1-0.4)	0.433± 0.056 (0.2-0.6)	0.017± 0.017
Dobermann	0.040	0.167± 0.020 (0.1-0.2)	0.117± 0.008 (0.08-0.12)	1.850± 0.294 (0.9-2.8)	0.040	0.193± 0.012 (0.16-0.22)	0.100	0.167± 0.033 (0.1-0.3)	0.367± 0.120 (0.1-0.7)	0.150± 0.067 (0.1-0.4)
Dachshund	0.040	0.167± 0.033 (0.1-0.3)	0.103± 0.008 (0.08-0.12)	2.433± 0.192 (1.6-3.0)	0.033± 0.004 (0.02-0.04)	0.190± 0.016 (0.16-0.26)	0.100	0.167± 0.021 (0.1-0.2)	0.267± 0.042 (0.1-0.4)	0.100± 0.045 (0.1-0.3)
Mongrel	0.040	0.133± 0.020 (0.1-0.2)	0.113± 0.008 (0.08-0.12)	1.467± 0.220 (1.1-2.5)	0.040	0.187± 0.008 (0.16-0.2)	0.100	0.117± 0.017 (0.1-0.2)	0.233± 0.056 (0.1-0.2)	Absent

P-du = 'P' wave duration, P-amp. = 'P' wave amplitude, P-R = 'P-R' interval, R-amp. = 'R' wave amplitude, QRS-du = QRS duration, Q-T = 'Q-T' interval, S-T-seg. = 'ST' segment, T-amp. = 'T' wave amplitude, Q-amp. = 'Q' wave amplitude, S-amp. = wave amplitude, Ranges for the above values are given in parenthesis

Table 7. The mean ± SE values of various parameters in lead III of different breeds below one year of age (control group) (n=6)

Breed	P-du	P-amp.	P-R	R-amp.	QRS-du	Q-T	S-T seg.	T-amp.	Q-amp.	S-amp.
Spitz	0.027± 0.004 (0.02-0.4)	0.125± 0.024 (0.05-0.2)	0.100± 0.012 (0.08-0.14)	1.133± 0.237 (0.5-2.0)	0.027± 0.004 (0.02-0.04)	0.160	0.100	0.157± 0.033 (0.1-0.3)	0.083± 0.048 (0.1-0.3)	0.317± 0.114 (0.1-0.8)
GSD	0.040	0.133± 0.020 (0.1-0.2)	0.103± 0.008 (0.08-0.12)	1.21±7 0.065 (0.5-1.3)	0.037± 0.004 (0.02-0.04)	0.197± 0.004 (0.18-0.2)	0.100	0.100	0.233± 0.062 (0.1-0.4)	0.067± 0.067
Dobermann	0.040	0.133± 0.020 (0.1-0.2)	0.127± 0.004 (0.12-0.14)	1.133± 0.302 (0.3-2.5)	0.040	0.200± 0.012 (0.16-0.24)	0.100	0.133± 0.021 (0.1-0.2)	0.150± 0.056 (0.1-0.4)	0.100± 0.063 (0.1-0.4)
Dachshund	0.037± 0.004 (0.02-0.04)	0.183± 0.016 (0.1-0.2)	0.100± 0.008 (0.08-0.12)	2.450± 0.192 (1.9-3.0)	0.037± 0.004 (0.32-0.04)	0.150± 0.008 (0.12-0.16)	0.100	0.100± 0.022 (0.05-0.2)	0.150± 0.062 (0.1-0.4)	0.200± 0.068 (0.1-0.5)
Mongrel	0.031± 0.004 (0.02-0.04)	0.117± 0.016 (0.1-0.2)	0.110± 0.004 (0.01-0.12)	1.300± 0.233 (0.8-2.4)	0.043± 0.008 (0.02-0.06)	0.177± 0.012 (0.14-0.2)	0.100	0.100	0.383± 0.087 (0.1-0.6)	0.133± 0.088

P-du = 'P' wave duration, P-amp. = 'P' wave amplitude, P-R = 'P-R' interval, R-amp. = 'R' wave amplitude, QRS-du = QRS duration, Q-T = 'Q-T' interval, S-T-seg. = 'ST' segment, T-amp. = 'T' wave amplitude, Q-amp. = 'Q' wave amplitude, S-amp. = wave amplitude, Ranges for the above values are given in parenthesis

Table 8. The mean ± SE values of various parameters in lead III of different breeds above one year of age (control group) (n=6)

Breed	P-du	P-amp.	P-R	R-amp.	QRS-du	Q-T	S-T seg.	T-amp.	Q-amp.	S-amp.
Spitz	0.027± 0.004 (0.02-0.04)	0.133± 0.020 (0.1-0.2)	0.153± 0.016 (0.12-0.22)	1.517± 0.184 (0.1-2.3)	0.030± 0.004 (0.02-0.04)	0.203± 0.015 (0.16-0.26)	0.100	0.150± 0.034 (0.1-0.3)	0.067± 0.033 (0.1-0.2)	0.133± 0.056 (0.1-0.3)
GSD	0.037± 0.004 (0.02-0.04)	0.133± 0.020 (0.1-0.2)	0.127± 0.008 (0.12-0.16)	1.450± 0.192 (0.9-1.9)	0.037± 0.004 (0.02-0.04)	0.233± 0.004 (0.22-0.24	0.100	0.150± 0.034 (0.1-0.3)	0.333± 0.067 (0.1-0.6)	0.017± 0.017
Dobermann	0.040	0.133± 0.020 (0.1-0.2)	0.113± 0.008 (0.08-0.14)	1.217± 0.290 (0.6-2.5)	0.040	0.203± 0.009 (0.16-0.22)	0.10	0.133± 0.021 (0.1-0.2)	0.283± 0.105 (0.2-0.6)	0.183± 0.075 (0.1-0.4)
Dachshund	0.040	0.117± 0.016 (0.1-0.2)	0.103± 0.008 (0.8-0.12)	1.567± 0.163 (0.9-1.9)	0.033± 0.004 (0.02-0.04)	0.187± 0.016 (0.16-0.26)	0.100	0.133± 0.021 (0.1-0.2)	0.083± 0.047 (0.1-0.3)	0.167± 0.049 (0.1-0.3)
Mongrel	0.040	0.117± 0.016 (0.1-0.2)	0.113± 0.008 (0.08-0.12)	1.167± 0.229 (0.8-2.3)	0.040	0.187± 0.008 (0.16-0.2)	0.100	0.100	0.183± 0.040	Absent

P-du = 'P' wave duration, P-amp. = 'P' wave amplitude, P-R = 'P-R' interval, R-amp. = 'R' wave amplitude, QRS-du = QRS duration, Q-T = 'Q-T' interval, S-T-seg. = 'ST' segment, T-amp. = 'T' wave amplitude, Q-amp. = 'Q' wave amplitude, S-amp. = wave amplitude, Ranges for the above values are given in parenthesis

Table 9. The mean ± SE values of various parameters in lead aVR of different breeds below one year of age (control group) (n=6)

Breed	P-du	P-amp.	P-R	R-amp.	QRS-du	Q-T	S-T seg.	T-amp.	Q-amp	S-amp.
Spitz	0.027± 0.004 (0.03-0.04)	0.108± 0.020 (0.05-0.02)	0.100± 0.012 (0.08-0.14)	1.150± 0.122 (0.8-1.5)	0.027± 0.004 (0.02-0.04)	0.160	0.100	0.150± 0.022 (0.1-0.2)	0.333± 0.141 (0.2-0.9)	0.150± 0.056 (0.1-0.4)
GSD	0.037± 0.004 (0.02-0.04)	0.100	0.103± 0.008 (0.08-0.12)	0.733± 0.098 (0.3-1.0)	0.037± 0.004 (0.02-0.04)	0.200	0.100	0.100	0.200± 0.073 (0.1-0.5)	0.017± 0.017
Dobermann	0.040	0.100	0.127± 0.004 (0.12-0.14)	0.950± 0.163 (0.6-1.7)	0.040	0.200± 0.012 (0.16-0.24)	0.100	0.100	0.233± 0.102 (0.1-0.3)	0.050± 0.034 (0.1-0.2)
Dachshund	0.037± 0.004 (0.02-0.04)	0.108± 0.008 (0.1-0.15)	0.103± 0.012 (0.06-0.12)	1.650± 0.184 (1.4-2.2)	0.037± 0.004 (0.02-0.04)	0.160± 0.004 (0.14-0.18)	0.117± 0.017 (0.1-0.2)	0.100± 0.022 (0.05-0.2)	0.330± 0.112 (0.1-0.8)	0.050± 0.034 (0.1-0.2)
Mongrel	0.030± 0.004 (0.02-0.04)	0.092± 0.008 (0.05-0.1)	0.110± 0.004 (0.1-0.12)	1.367± 0.363 (0.1-2.3)	0.043± 0.008 (0.02-0.06)	0.177± 0.012 (0.14-0.2)	0.100	0.100	0.400± 0.082 (0.1-0.6)	0.017± 0.017

P-du = 'P' wave duration, P-amp. = 'P' wave amplitude, P-R = 'P-R' interval, R-amp. = 'R' wave amplitude, QRS-du = QRS duration, Q-T = 'Q-T' interval, S-T-seg. = 'ST' segment, T-amp. = 'T' wave amplitude, Q-amp. = 'Q' wave amplitude, S-amp. = wave amplitude, Ranges for the above values are given in parenthesis

Table 10. The mean ± SE values of various parameters in lead aVR of different breeds above one year of age (control group) (n=6)

Breed	P-du	P-amp.	P-R	R-amp.	QRS-du	Q-T	S-T seg.	T-amp.	Q-amp.	S-amp.
Spitz	0.023± 0.004 (0.02-0.04)	0.092± 0.008 (0.5-0.1)	0.147± 0.012 (0.1-0.2)	1.150± 0.204 (0.6-1.7)	0.030± 0.004 (0.02-0.04)	0.190± 0.008 (0.16-0.22)	0.100	0.108± 0.020 (0.05-0.2)	0.117± 0.054 (0.2-0.3)	0.017± 0.017
GSD	0.037± 0.004 (0.2-0.04)	0.100	0.130± 0.008 (0.12-0.18)	0.917± 0.118 (0.6-1.3)	0.033± 0.004 (0.02-0.04)	0.230± 0.008 (0.18-0.24)	0.100	0.117± 0.017 (0.1-0.2)	0.267± 0.042 (0.1-0.4)	Absent
Dobermann	0.040	0.100	0.117± 0.008 (0.08-0.14)	1.083± 0.122 (0.7-1.5)	0.040	0.193± 0.012 (0.16-0.22)	0.100	0.117± 0.017 (0.1-0.2)	0.300± 0.100 (0.1-0.7)	0.067± 0.049 (0.1-0.3)
Dachshund	0.040	0.133± 0.020 (0.1-0.2)	0.100± 0.008 (0.08-0.12)	1.683± 0.204 (1.1-2.3)	0.030± 0.004 (0.02-0.04)	0.180± 0.016 (0.16-0.26)	0.100	0.100	0.350± 0.072 (0.2-0.05)	Absent
Mongrel	0.040	0.083± 0.012 (0.05-0.1)	0.114± 0.008 (0.08-0.12)	0.750± 0.135 (0.12-0.13)	0.040	0.187± 0.008 (0.16-0.2)	0.100	0.100	0.117± 0.040 (0.1-0.3)	Absent

P-du = 'P' wave duration, P-amp. = 'P' wave amplitude, P-R = 'P-R' interval, R-amp. = 'R' wave amplitude, QRS-du = QRS duration, Q-T = 'Q-T' interval, S-T-seg. = 'ST' segment, T-amp. = 'T' wave amplitude, Q-amp. = 'Q' wave amplitude, S-amp. = wave amplitude, Ranges for the above values are given in parenthesis

Table 11. The mean ± SE values of various parameters in lead aVL of different breeds below one year of age (control group) (n=6)

Breed	P-du	P-amp.	P-R	R-amp.	QRS-du	Q-T	S-T seg.	T-amp.	Q-amp.	S-amp.
Spitz	0.027± 0.004 (0.02-0.04)	0.092± 0.008 (0.05-0.1)	0.100± 0.012 (0.08-0.14)	0.583± 0.049 (0.4-0.7)	0.027± 0.004 (0.02-0.04)	0.160	0.100	0.125± 0.017 (0.1-0.2)	0.050± 0.034 (0.1-0.2)	0.283± 0.130 (0.1-0.9)
GSD	0.037± 0.004 (0.02-0.04)	0.100	0.103± 0.008 (0.08-0.12)	0.417± 0.049 (0.2-0.5)	0.037± 0.004 (0.2-0.04)	0.200	0.100	0.100	0.217± 0.065	0.067± 0.067 (0.1-0.2)
Dobermann	0.040	0.100	0.127± 0.004 (0.12-0.14)	0.483± 0.110 (0.2-0.9)	0.040	0.200± 0.012 (0.16-0.24)	0.100	0.100	0.033± 0.021	0.083± 0.054 (0.2-0.3)
Dachshund	0.033± 0.004 (0.02-0.04)	0.092± 0.008 (0.05-0.1)	0.110± 0.008 (0.08-0.12)	1.167± 0.220 (0.7-2.1)	0.037± 0.004 (0.02-0.04)	0.160	0.117± 0.017 (0.1-0.2)	0.083± 0.010 (0.05-0.	0.033± 0.033	0.183± 0.087
Mongrel	0.031± 0.004 (0.02-0.04)	0.075± 0.012 (0.05-0.1)	0.110± 0.004 (0.1-0.12)	0.517± 0.118 (0.2-0.9)	0.040± 0.008 (0.02-0.06)	0.177± 0.012 (0.14-0.2)	0.100	0.100	0.133± 0.061	0.067± 0.049

P-du = 'P' wave duration, P-amp. = 'P' wave amplitude, P-R = 'P-R' interval, R-amp. = 'R' wave amplitude, QRS-du = QRS duration, Q-T = 'Q-T' interval, S-T-seg. = 'ST' segment, T-amp. = 'T' wave amplitude, Q-amp. = 'Q' wave amplitude, S-amp. = wave amplitude, Ranges for the above values are given in parenthesis

Table 12. The mean ± SE values of various parameters in lead aVL of different breeds above one year of age (control group) (n=6)

Breed	P-du	P-amp	P-R	R-amp	QRS-du	Q-T	S-T seg.	T-amp.	Q-amp.	S-amp
Spitz	0.027± 0.004 (0.02-0.04)	0.092± 0.008 (0.05-0.1)	0.147± 0.012 (0.12-0.2)	0.800± 0.122 (0.5-1.3)	0.023± 0.004 (0.02-0.04)	0.193± 0.012 (0.16-0.24)	0.100	0.092± 0.008 (0.05-1)	0.033± 0.033	0.067± 0.033 (0.1-0.2)
GSD	0.037± 0.044 (0.2-0.04)	0.092± 0.008 (0.05-0.1)	0.127± 0.008 (0.12-0.16)	0.617± 0.106 (02-0.9)	0.033± 0.004 (0.02-0.04)	0.233± 0.008 (0.2-0.24)	0.100	0.125± 0.025 (0.05-0.2)	0.117± 0.040 (0.1-0.3)	Absent
Dobermann	0.040	0.1	0.117± 0.008 (0.08-0.14)	0.567± 0.143 (0.1-1.1)	0.040	0.193± 0.012 (0.16-0.26)	0.100	0.117± 0.017 (0.1-0.2)	0.02	0.150± 0.056 (0.1-0.3)
Dachshund	0.04	0.1	0.097± 0.008 (0.08-0.12)	0.600± 0.073 (0.3-0.8)	0.030± 0.004 (0.02-0.04)	0.180± 0.016 ((0.16-0.26)	0.100	0.100	Absent	0.133± 0.061 (0.1-0.4)
Mongrel	0.040	0.092± 0.008 (0.05-0.1)	0.113± 0.008 (0.08-0.12)	0.500± 0.110 (0.2-1.0)	0.040	0.187± 0.008 (0.16-0.2)	0.100	0.100	0.067± 0.021	0.033± 0.021

P-du = 'P' wave duration, P-amp. = 'P' wave amplitude, P-R = 'P-R' interval, R-amp. = 'R' wave amplitude, QRS-du = QRS duration, Q-T = 'Q-T' interval, S-T-seg. = 'ST' segment, T-amp. = 'T' wave amplitude, Q-amp. = 'Q' wave amplitude, S-amp. = wave amplitude. Ranges for the above values are given in parenthesis

Table 13. The mean ± SE values of various parameters in lead aVF of different breeds below one year of age (control group) (n=6)

Breed	P-du	P-amp.	P-R	R-amp.	QRS-du	Q-T	S-T seg.	T-amp.	Q-amp.	S-amp.
Spitz	0.027± 0.004 (0.02-0.04)	0.150± 0.020 (0.1-0.2)	0.090± 0.008 (0.08-0.14)	1.350± 0.180 (0.7-1.8)	0.023± 0.004 (0.02-0.04)	0.160	0.100	0.200± 0.045 (0.1-0.3)	0.150± 0.076 (0.1-5)	0.167± 0.042 (0.1-0.3)
GSD	0.040	0.117± 0.016 (01-0.2)	0.103± 0.008 (0.08-0.12)	1.050± 0.114 (0.5-1.3)	0.037± 0.004 (0.02-0.04)	0.200	0.100	0.100	0.217± 0.065 (0.1-0.5)	0.067± 0.67
Dobermann	0.04	0.117± 0.016 (0.1-0.2)	0.127± 0.004 (0.12-0.14)	1.383± 0.245 (1-2.6)	0.040	0.200 (0.16-0.24)	0.100	0.117± 0.017 (0.1-0.2)	0.250± 0.106 (0.1-0.7)	0.083± 0.065 (0.1-0.4)
Dachshund	0.037± 0.004 (0.02-0.04)	0.200± 0.024 (0.1-0.3)	0.113± 0.008 (0.08-0.12)	2.367± 0.237 (1.5-3.2)	0.037± 0.004 (0.02-0.04)	0.160	0.100	0.083± 0.011 (0.05-0.1)	0.233± 0.067 (0.2-0.5)	0.200± 0.068 (0.2-0.4)
Mongrel	0.030± 0.004 (0.02-0.04)	0.117± 0.016 (0.1-0.2)	0.110± 0.004 (0.1-0.12)	1.623± 0.290 (0.6-2.6)	0.043± 0.008 (0.02-0.06)	0.177± 0.012 (0.14-0.2)	0.100	0.100	0.367± 0.080 (0.2-0.6)	0.083± 0.054

P-du = 'P' wave duration, P-amp. = 'P' wave amplitude, P-R = 'P-R' interval, R-amp. = 'R' wave amplitude, QRS-du = QRS duration, Q-T = 'Q-T' interval, S-T-seg. = 'ST' segment, T-amp. = 'T' wave amplitude, Q-amp. = 'Q' wave amplitude, S-amp. = wave amplitude, Ranges for the above values are given in parenthesis

Table 14. The mean ± SE values of various parameters in lead aVF of different breeds above one year of age (control group) (n=6)

Breed	P-du	P-amp.	P-R	R-amp.	QRS-du	Q-T	S-T seg.	T-amp.	Q-amp.	S-amp.
Spitz	0.030± 0.004 (0.02-0.04)	0.133± 0.020 (0.1-0.2)	0.147± 0.012 (0.1-0.2)	1.567± 0.163± (1-2)	0.030± 0.004 (0.02-0.04)	0.193± 0.012 (0.16-0.24)	0.100	(0.1-0.3)	0.100± 0.063 (0.1-0.4)	0.117± 0.048 (0.1-0.3)
GSD	0.037± 0.004 (0.02-0.04)	0.100	0.127± 0.008 (0.12-0.16)	1.517± 0.92 (0.9-2.0)	0.037± 0.004 (0.02-0.04)	0.230± 0.008 (0.2-0.24)	0.100	0.150± 0.034 (0.1-0.3)	0.417± 0.065 (0.2-0.7)	Absent
Dobermann	0.040	0.133± 0.020 (0.1-0.2)	0.117± 0.008 (0.12-0.16)	1.567± 0.294 (0.6-2.6)	0.040	0.193± 0.012 (0.16-0.22)	0.100	0.150± 0.034 (0.12-0.3)	0.350± 0.112 (0.1-0.7)	0.167± 0.061 (0.1-0.3)
Dachshund	0.040	0.067± 0.033 (0.1-0.3)	0.107± 0.004 (0.1-0.12)	2.00± 0.245 (1-2.7)	0.030± 0.004 (0.02-0.04)	0.183± 0.013 (0.16-0.26)	0.100	0.133± 0.021 (0.1-0.2)	0.167± 0.061 (0.2-0.4)	0.133± 0.049 (0.1-0.3)
Mongrel	0.040	0.134± 0.020 (0.1-0.2)	0.113± 0.008 (0.08-0.12)	1.233± 0.269 (0.6-2.5)	0.040	0.187± 0.008 (0.16-0.2)	0.100	0.100	0.133± 0.042	0.083± 0.065

P-du = 'P' wave duration, P-amp. = 'P' wave amplitude, P-R = 'P-R' interval, R-amp. = 'R' wave amplitude, QRS-du = QRS duration, Q-T = 'Q-T' interval, S-T-seg. = 'ST' segment, T-amp. = 'T' wave amplitude, Q-amp. = 'Q' wave amplitude, S-amp. = wave amplitude, Ranges for the above values are given in parenthesis

Table 15. The mean ± SE values of various parameters in lead V_{10} of different breeds below one year of age (control group) (n=6)

Breed	P-du	P-amp.	P-R	R-amp.	QRS-du	Q-T	S-T seg.	T-amp.	Q-amp.	S-amp.
Spitz	0.040	0.100	0.140	1.100± 0.045 (1-1.2)	0.040	0.160	0.100	0.150± 0.022 (0.1-0.2)	Absent	0.450± 0.022 (0.4-0.5)
GSD	0.040	0.100	0.107± 0.008 (0.08-0.12)	0.733± 0.041 (0.6-0.9)	0.037± 0.004 (0.2-0.04	0.200	0.100	0.150± 0.034 (0.1-0.3)	Absent	0.267± 0.049 (0.1-0.4)
Dobermann	0.040	0.100	0.120	0.950± 0.110 (0.7-1.2)	0.040	0.200	0.100	0.200± 0.045 (0.1-0.3)	Absent	0.200
Dachshund	0.037± 0.004 (0.02-0.04)	0.092± 0.088 (0.05-0.10)	0.113± 0.008 (0.08-0.12)	0.983± 0.086 (0.8-1.3)	0.037± 0.004 (0.02-0.04)	0.160	0.100	0.100	Absent	0.233± 0.021 (0.2-0.3)
Mongrel	0.031± 0.004 (0.02-0.04)	0.100	0.113± 0.004 (0.1-0.12)	0.700± 0.118 (0.4-1.1)	0.040± 0.008 (0.02-0.06)	0.177± 0.012 (0.14-0.2)	0.100	0.150± 0.022 (0.1-0.2)	Absent	0.100± 0.063

P-du = 'P' wave duration, P-amp. = 'P' wave amplitude, P-R = 'P-R' interval, R-amp. = 'R' wave amplitude, QRS-du = QRS duration, Q-T = 'Q-T' interval, S-T-seg. = 'ST' segment, T-amp. = 'T' wave amplitude, Q-amp. = 'Q' wave amplitude, S-amp. = wave amplitude, Ranges for the above values are given in parenthesis

Table 16. The mean ± SE values of various parameters in lead V_{10} of different breeds above one year of age (control group) (n=6)

Breed	P-du	P-amp.	P-R	R-amp.	QRS-du	Q-T	S-T seg.	T-amp.	Q-amp.	S-amp.
Spitz	0.033± 0.004 (0.02-0.04)	0.083± 0.012 (0.05-0.1)	0.140± 0.008 (0.12-0.16)	0.833± 0.102 (0.5-0.9)	0.033± 0.004 (0.02-0.04)	0.193± 0.008 (0.16-0.22)	0.100	0.142± 0.027 (0.05-0.2)	Absent	0.200± 0.026 (0.1-0.3)
GSD	0.033± 0.004 (0.02-0.04)	0.100	0.120	0.667± 0.180 (0.3-1.2)	0.033± 0.004 (0.02-0.04)	0.240	0.100	0.133± 0.033 (0.1-0.3)	Absent	0.167± 0.095 (0.2-0.6)
Dobermann	0.04	0.100	0.121± 0.008 (0.08-0.14)	1.250± 0.114 (0.9-1.5)	0.040	0.197± 0.012 (0.16-0.22)	0.100	0.150± 0.034 (0.1-0.3)	Absent	0.300± 0.037 (0.2-0.4)
Dachshund	0.040	0.100	0.097± 0.008 (0.08-0.12)	0.717± 0.078 (0.4-0.9)	0.030± 0.004 (0.02-0.04)	0.180± 0.016 (0.16-0.26)	0.100	0.133± 0.021 (0.1-0.2)	Absent	0.200± 0.063 (0.1-0.4)
Mongrel	0.040	0.100	0.120	0.567± 0.127 (0.2-0.9)	0.033± 0.004 (0.02-0.04)	0.200	0.100	0.100	Absent	0.200± 0.073 (0.2-0.4)

P-du = 'P' wave duration, P-amp. = 'P' wave amplitude, P-R = 'P-R' interval, R-amp. = 'R' wave amplitude, QRS-du = QRS duration, Q-T = 'Q-T' interval, S-T-seg. = 'ST' segment, T-amp. = 'T' wave amplitude, Q-amp. = 'Q' wave amplitude, S-amp. = wave amplitude, Ranges for the above values are given in parenthesis

Table 17. The mean ± SE values of various parameters in lead CV_6LL of different breeds below one year of age (control group) (n=6)

Breed	P-du	P-amp.	P-R	R-amp.	QRS-du	Q-T	S-T seg.	T-amp.	Q-amp.	S-amp.
Spitz	0.033± 0.004 (0.02-0.04)	0.100	0.120± 0.012 (0.08-0.14)	2.033± 0.208 (1.4-2.5)	0.033± 0.004 (0.02-0.04)	0.160	0.100	0.167± 0.021 (0.1-0.2)	Absent	0.700
GSD	0.040	0.133± 0.020 (0.1-0.2)	0.107± 0.008 (0.08-0.12)	2.417± 0.229 (1.6-3.0)	0.037± 0.004 (0.02-0.04)	0.200	0.100	0.183± 0.048 (0.1-0.4)	0.050± 0.034 (0.1-0.2)	0.500± 0.052 (0.3-0.7)
Dobermann	0.040	0.117± 0.016 (0.1-0.2)	0.127± 0.004 (0.12-0.14)	2.383± 0.461 (1.2-3.7)	0.040	0.213± 0.008 (0.2-0.24)	0.100	0.300± 0.077 (0.1-0.6)	0.050± 0.050	0.383± 0.091 (0.1-0.6)
Dachshund	0.033± 0.004 (0.02-0.04)	0.117± 0.016 (0.1-0.2)	0.113± 0.008 (0.08-0.12)	2.867± 0.294 (1.8-3.6)	0.037± 0.004 (0.02-0.04)	0.153± 0.008 (0.12-0.16)	0.117± 0.017 (0.1-0.2)	0.117± 0.017 (0.1-0.2)	Absent	0.048± 0.070 (0.2-0.07)
Mongrel	0.030± 0.004 (0.02-0.04)	0.117± 0.016 (0.1-0.2)	0.110± 0.004 (0.1-0.12)	2.283± 0.400 (1.5-4.0)	0.043± 0.008 (0.02-0.06)	0.183± 0.008 (0.16-0.2)	0.100	0.366± 0.138 (0.1-1.0)	Absent	0.333± 0.099 (0.1-0.7)

P-du = 'P' wave duration, P-amp. = 'P' wave amplitude, P-R = 'P-R' interval, R-amp. = 'R' wave amplitude, QRS-du = QRS duration, Q-T = 'Q-T' interval, S-T-seg. = 'ST' segment, T-amp. = 'T' wave amplitude, Q-amp. = 'Q' wave amplitude, S-amp. = wave amplitude. Ranges for the above values are given in parenthesis

Table 18. The mean ± SE values of various parameters in lead CV_6LL of different breeds above one year of age (control group) (n=6)

Breed	P-du	P-amp.	P-R	R-amp.	QRS-du	Q-T	S-T seg.	T-amp.	Q-amp.	S-amp.
Spitz	0.030± 0.004 (0.02-0.04)	0.117± 0.016 (0.1-0.2)	0.150± 0.012 (0.12-0.2)	1.767± 0.331 (1.1-3.3)	0.030± 0.004 (0.02-0.04)	0.183± 0.008 (0.16-0.2)	0.100	0.250± 0.081 (0.1-0.5)	Absent	0.417± 0.065 (0.2-0.6)
GSD	0.040	0.100	0.127± 0.008 (0.12-0.16)	2.283± 0.351 (1.1-3.2)	0.033± 0.004 (0.02-0.04)	0.234± 0.008 (0.2-0.24)	0.100	0.283± 0.060 (0.1-0.5)	Absent	0.300± 0.086 (0.1-0.5)
Dobermann	0.040	0.100	0.120± 0.008 (0.08-0.14)	3.117± 0.269 (2.3-3.8)	0.040	0.193± 0.012 (0.16-0.22)	0.100	0.367± 0.076 (0.2-0.6)	0.033± 0.033	0.583± 0.056 (0.1-0.5)
Dachshund	0.040	0.117± 0.016 (0.1-0.2)	0.097± 0.008 (0.08-0.12)	1.850± 0.253 (1.3-2.9)	0.030± 0.004 (0.02-0.04)	0.183± 0.016 (0.16-0.26)	0.100	0.200± 0.068 (0.1-0.5)	Absent	0.483± 0.108 (0.4-0.7)
Mongrel	0.040	0.117± 0.016 (0.1-0.2)	0.113± 0.008 (0.08-0.12)	1.500± 0.327 (0.03-2.5)	0.043± 0.004 (0.04-0.06)	0.187± 0.008 (0.16-0.2)	0.100	0.270± 0.080 (0.1-0.6)	Absent	0.483± 0.083 (0.1-0.7)

P-du = 'P' wave duration, P-amp. = 'P' wave amplitude, P-R = 'P-R' interval, R-amp. = 'R' wave amplitude, QRS-du = QRS duration, Q-T = 'Q-T' interval, S-T-seg. = 'ST' segment, T-amp. = 'T' wave amplitude, Q-amp. = 'Q' wave amplitude, S-amp. = wave amplitude. Ranges for the above values are given in parenthesis

Table 19. The mean ± SE values of various parameters in lead CV_6LU of different breeds below one year of age (control group) (n=6)

Breed	P-du	P-amp.	P-R	R-amp.	QRS-du	Q-T	S-T seg.	T-amp.	Q-amp.	S-amp.
Spitz	0.030± 0.004 (0.02-0.04)	0.133± 0.020 (0.1-0.2)	0.110± 0.012 (0.08-0.14)	2.167± 0.351 (0.8-2.9)	0.032± 0.004 (0.02-0.04)	0.160	0.100	0.200± 0.026 (0.1-0.3)	0.050± 0.022	0.367± 0.061 (0.2-0.5)
GSD	0.040	0.117± 0.016 (0.1-0.2)	0.107± 0.008 (0.08-0.12)	1.550± 0.225 (1.1-2.6)	0.037± 0.004 (0.02-0.04)	0.200	0.100	0.217± 0.048 (0.1-0.4)	0.133± 0.021 (0.1-0.2)	0.267± 0.084 (0.1-0.5)
Dobermann	0.040	0.117± 0.016 (0.1-0.2)	0.124± 0.004 (0.12-0.14)	2.500± 0.416 (1.3-3.8)	0.040	0.207± 0.008 (0.2-0.24)	0.100	0.300± 0.052 (0.1-0.4)	0.117± 0.098 (0.1-0.6)	0.333± 0.112 (0.4-0.6)
Dachshund	0.037± 0.004 (0.02-0.04)	0.167± 0.020 (0.1-0.2)	0.113± 0.008 (0.08-0.12)	3.150± 0.167 (2.5-3.5)	0.037± 0.004 (0.02-0.04)	0.153± 0.008 (0.12-0.16)	0.117± 0.016 (0.1-0.2)	0.133± 0.021 (0.1-0.2)	0.150± 0.050 (0.2-0.3)	0.167± 0.056 (0.2-0.3)
Mongrel	0.030± 0.004 (0.02-0.04)	0.117± 0.016 (0.1-0.2)	0.110± 0.004 (0.1-0.12)	2.250± 0.331 (1.4-3.5)	0.043± 0.008 (0.02-0.06)	0.183± 0.008 (0.16-0.2)	0.100	0.217± 0.060 (0.1-0.5)	0.117± 0.040 (0.1-0.2)	0.167± 0.067 (0.1-0.4)

P-du = 'P' wave duration, P-amp. = 'P' wave amplitude, P-R = 'P-R' interval, R-amp. = 'R' wave amplitude, QRS-du = QRS duration, Q-T = 'Q-T' interval, S-T-seg. = 'ST' segment, T-amp. = 'T' wave amplitude, Q-amp. = 'Q' wave amplitude, S-amp. = wave amplitude, Ranges for the above values are given in parenthesis

Table 20. The mean ± SE values of various parameters in lead CV_6LU of different breeds above one year of age (control group) (n=6)

Breed	P-du	P-amp.	P-R	R-amp.	QRS-du	Q-T	S-T seg.	T-amp.	Q-amp.	S-amp.
Spitz	0.030± 0.004 (0.02-0.04)	0.133± 0.020 (0.1-0.2)	0.150± 0.012 (0.12-0.2)	2.400± 0.314 (1.3-3.5)	0.030± 0.004 (0.02-0.04)	0.190± 0.012 (0.16-0.24)	0.100	0.292± 0.127 (0.05-0.09)	Absent	0.333± 0.084 (0.2-0.6)
GSD	0.037± 0.004 (0.02-0.04)	0.100	0.127± 0.008 (0.12-0.16)	2.483± 0.274 (1.7-3.5)	0.040	0.227± 0.008 (0.2-0.24)	0.100	0.333± 0.056 (0.2-0.5)	0.067± 0.033 (0.1-0.2)	0.217± 0.101 (0.3-0.5)
Dobermann	0.040	0.100	0.120± 0.008 (0.08-0.14)	3.417± 0.180 (2.2-4.0)	0.040	0.210± 0.024 (0.16-0.22)	0.100	0.417± 0.117 (0.1-0.9)	0.050± 0.034 (0.1-0.2)	0.467± 0.102 (0.4-0.7)
Dachshund	0.040	0.167± 0.020 (0.1-0.2)	0.097± 0.008 (0.08-0.12)	2.633± 0.253 (1.6-3.3)	0.033± 0.004 (0.02-0.04)	0.183± 0.016 (0.16-0.26)	0.100	0.183± 0.048 (0.1-0.4)	0.017± 0.017	0.367± 0.067 (0.2-0.4)
Mongrel	0.040	0.117± 0.016 (0.1-0.2)	0.113± 0.008 (0.08-0.12)	1.867± 0.253 (1.2-2.7)	0.043± 0.004 (0.04-0.06)	0.187± 0.008 (0.16-0.2)	0.100	0.283± 0.070 (0.1-0.5)	0.033± 0.033	0.333± 0.033 (0.2-0.4)

P-du = 'P' wave duration, P-amp. = 'P' wave amplitude, P-R = 'P-R' interval, R-amp. = 'R' wave amplitude, QRS-du = QRS duration, Q-T = 'Q-T' interval, S-T-seg. = 'ST' segment, T-amp. = 'T' wave amplitude, Q-amp. = 'Q' wave amplitude, S-amp. = wave amplitude, Ranges for the above values are given in parenthesis

Table 21. Mean ± SE values of heart rate and mean electrical axis of different breeds below one year of age (control group) (n=6)

Parameters	Spitz	German Shepherd	Dobermann	Dachshund	Mongrel
Heart Rate (per minute)	145.83 ± 20.43 (60-200)	123.33 ± 3.33 (120-140)	110.83 ± 8.79 (90-150)	153.67 ± 7.94 (120-180)	113.33 ± 10.85 (70-150)
MEA (Mean Electrical Axis) (in degrees)	69.33 ± 7.60 (50-90)	91.67 ± 1.67 (90-100)	75.00 ± 8.47 (40-90)	84.83 ± 2.37 (78-90)	80.00 ± 6.82 60-90)

Ranges are given in parenthesis

Table 22. Mean ± SE values of heart rate and mean electrical axis of different breeds above one year of age (control group)

Parameter	Spitz	German Shepherd	Dobermann	Dachshund	Mongrel
Heart rate (per minute)	88.5 ± 5.07 (72-105)	96.83 ± 8.57 (72-132)	100 ± 4.47 (90-120)	122.83±9.17 (90-150)	129.17 ± 6.88 (105-150)
MEA (Mean Electrical Axis) (in degrees)	79.5 ± 3.63 (70-90)	76.67 ± 6.15 (60-90)	79.33 ± 8.19 (40-90)	67 ± 6.70 (40-90)	78.33 ± 8.33 (40-90)

Ranges are given in parenthesis

Table 23. Haemogram of the control group

Parameters	Mean ± SE
RBC (x 10^6/mm^3)	6.67 ± 0.36 (6-8)
Haemoglobin (g%)	13.67 ± 0.67 (12-16)
PCV (per cent)	43.33 ± 2.25 (40-50)
MCV (fl)	65.04 ± 0.90 (62.5-68.57)
MCH (pg)	20.54 ± 0.34 (20-21.8)
MCHC (per cent)	31.59 ± 0.59 (30-34.28)
TLC (mm^3)	10833.33 ± 945.75 (9000-14000)
DIFFERENTIAL COUNT (per cent)	
Neutrophils	70.33 ± 1.69 (65-76)
Lymphocytes	23.17 ± 1.08 (20-26)
Monocytes	3.33 ± 0.56 (2-5)
Eosinophils	3.17 ± 0.60 (2-5)
Basophils	0
ESR (mm/30 min)	3 ± 0.73 (1-6)

Table 24. Haematology of dogs with electrocardiogram abnormalities (clinical group)

	RBC	Hb	PCV	MCV	MCH	MCHC	TLC	NEU	LYM	MON	EOS	BAS	ESR
Gp I CHF (n=7)	6.88 ± 0.37 (5.5-8.2)	14.43 ± 0.37 (13-16)	44.43 ± 1.82 (40-52)	64.88 ± 1.52 (60-72.72)	21.19 ± 0.83 (18.75-24.45)	32.63 ± 0.82 (30-35.71)	12428.57 ± 751.42 (9000-15000)	73.57 ± 1.45 (68-77)	21.86 ± 0.94 (18-24)	2.86 ± 0.46 (1-4)	1.86 ± 0.40 (1-4)	0	3.43 ± 0.37 (2-5)
Gp II Ventricular enlargement (n=3)	3.67 ± 0.17 (3.5-4)	7 ± 0.58 (6-8)	23.33 ± 1.33 (22-26)	60.95 ± 3.04 (55-65)	18.21 ± 0.89 (17.14-20)	29.95 ± 1.37 (27.27-31.81)	11000 ± 577.35 (10000-12000)	75.67 ± 0.67 (75-77)	20 (20)	3	1.33 ± 0.67 (2)	0	5 ± 1.73 (2-8)
Gp III Electrolyte imbalance due to renal disease (n=3)	3.67 ± 0.44 (3-4.5)	6 ± 0.58 (7-9)	26.67 ± 1.67 (25-30)	57.14 ± 11.98 (33.33-71.42)	22.06 ± 1.04 (20-23.33)	30 ± 1.15 (28-32)	12000 ± 577.35 (11000-13000)	84 ± 2 (80-86)	13 ± 1.53 (11-16)	2	1 ± 0.58 (0-2)	0	9.33 ± 0.67 (8-10)

Ranges are given in parenthesis

Table 25. Serum chemistry of control group

Parameters	Mean ± SE
CK (U/L)	42.5 ± 5.46 (20-60)
ALT (IU/L)	17.00 ± 1.78 (12-24)
Sodium (mEq/l)	145 ± 1.41 (140-150)
Potassium (mEq/l)	4.33 ± 0.24 (3.8-5.3)
Total protein(g/dl)	6.7 ± 0.15 (6.2-7.2)
Albumin (g/dl)	3.73 ± 0.07 (3.6-4.0)
Albumin: Globulin ratio	1.27 ± 0.07 (1.11-1.58)
Creatinine (mg/dl)	0.93 ± 0.12 (0.5-1.3)

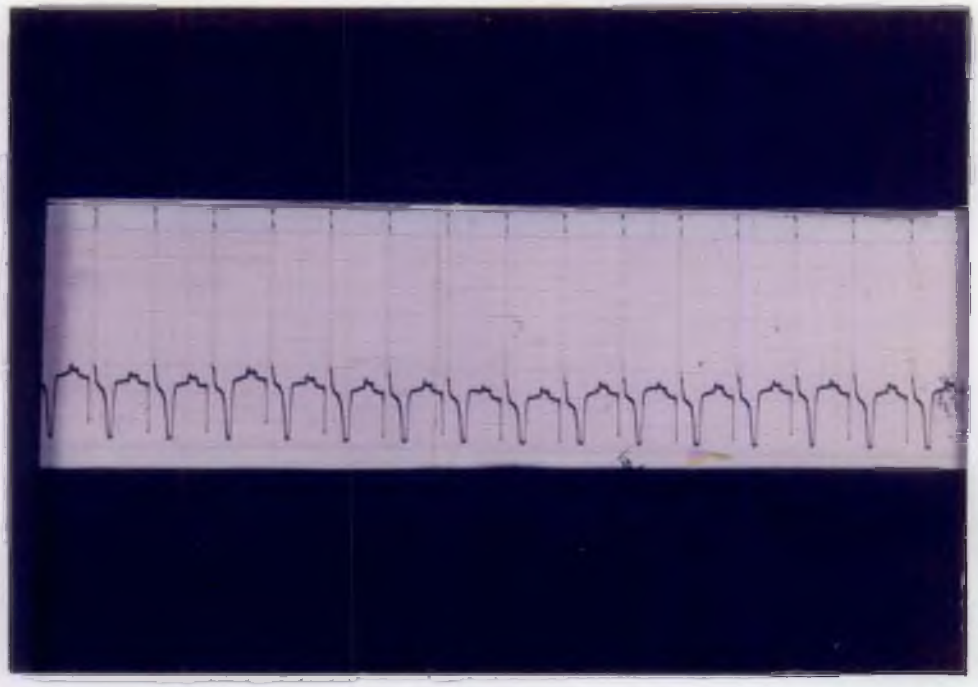
Table 26. Serum chemistry of dogs with electrocardiogram abnormalities (clinical group)

Group	CK	ALT	SOD	POT	TP	ALB	A:G	CRE
Group I Congestive heart failure (n=7)	46.14 ± 2.19 (38-55)	19.43 ± 0.81 (17.22)	174.14 ± 2.38 (166-183)	4.80 ± 0.08 (4.5-5)	6.54 ± 0.18 (6.0-7.2)	3.91 ± 0.073 (3.6-4.2)	1.54 ± 0.13 (1.125-2)	1.25 ± 0.08 (0.9-1.47)
Group II Ventricular enlargement (n=3)	43.33 ± 3.33 (40-50)	18.33 ± 1.66 (15-20)	143.00 ± 1.73 (140-146)	4.50 ± 0.28 (4.0-5.0)	5.73 ± 0.93 (4.0-7.2)	3.20 ± 0.702 (1.8-4.0)	1.15 ± 0.19 (0.818-1.5)	0.93 ± 0.06 (0.8-1)
Group III Electrolyte imbalance due to renal disease (n=3)	33.33 ± 1.76 (30-36)	16.67 ± 0.88 (15-18)	144.33 ± 2.73 (139-148)	7.67 ± 0.33 (7-8)	6.07 ± 0.07 (6-6.2)	1.9 ± 0.06 (1.8-2)	0.46 ± 0.03 (0.409-0.5)	19.77 ± 1.29 (18-22.3)

Ranges are given in parenthesis
CK – Creatine Kinase, ALT – Alanine amino transferase, SOD – Sodium, POT – Potassium, TP – Total Protein, ALB – Albumin, A:G – Albumin: Globulin ratio, CRE - Creatinine

Plate 1 Lead II ECG tracing showing notched and widened 'P' wave, tall 'R' wave and deep 'Q' wave in a dog with congestive heart failure indicating left atrial and ventricular enlargement and interventricular septal hypertrophy (paper speed 25 mm/sec, 1 cm = 1 mv)

Plate 2 Lead II ECG tracing of the same dog (as shown in Plate 1 in 1 $\frac{mV}{cm}$ = 0.5 $\frac{cm}{}$ sensitivity (paper speed 25 mm/sec)



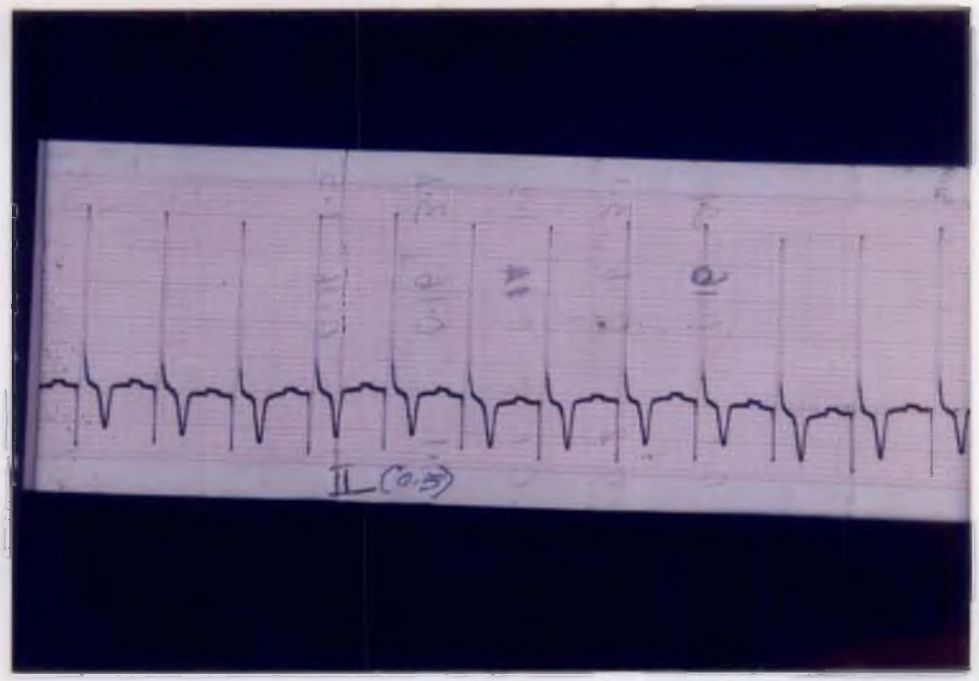

II (0.5)

Plate 3 **Radiograph showing enlarged cardiac silhouette with left atrial and ventricular enlargement with straightening of the caudal waist (arrow) of the same dog (as shown in Plate 1) with congestive heart failure**

Plate 4 **Echocardiogram showing enlargement of left atrium (LA) and thickened mitral valve (MV) in a dog with congestive heart failure (same dog shown in Plate 1)**

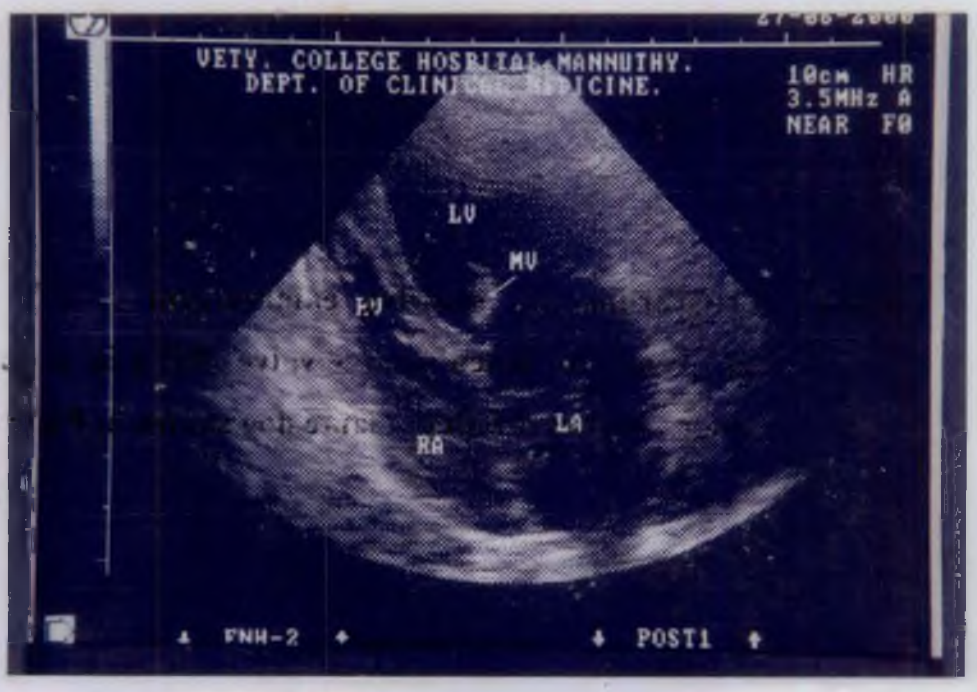
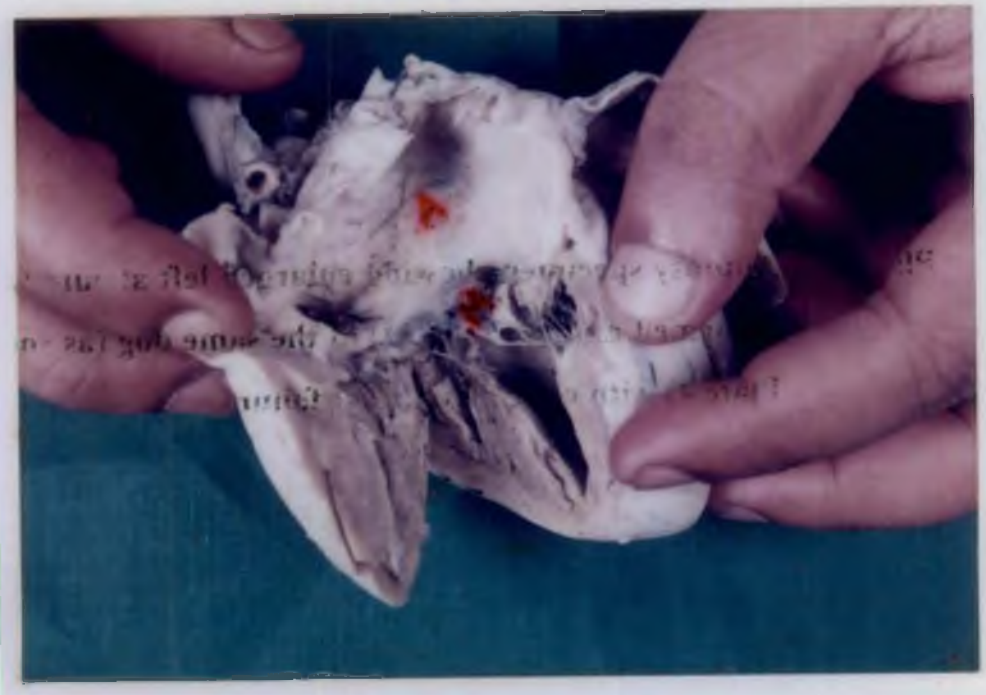

VETY. COLLEGE HOSPITAL, MANNUTHY.
10cm HR
3.5MHz A
NEAR F0
LV
MV
RV
LA
RA
FNH-2
POST1

Plate 5 Autopsy specimen showing enlarged left atrium (A) and deformed mitral valve (M) in the same dog (as shown in Plate 4) with congestive heart failure

Plate 6 Gross autopsy specimen showing enlarged left atrium (arrow) of the same dog (as shown in Plate 5) with congestive heart failure



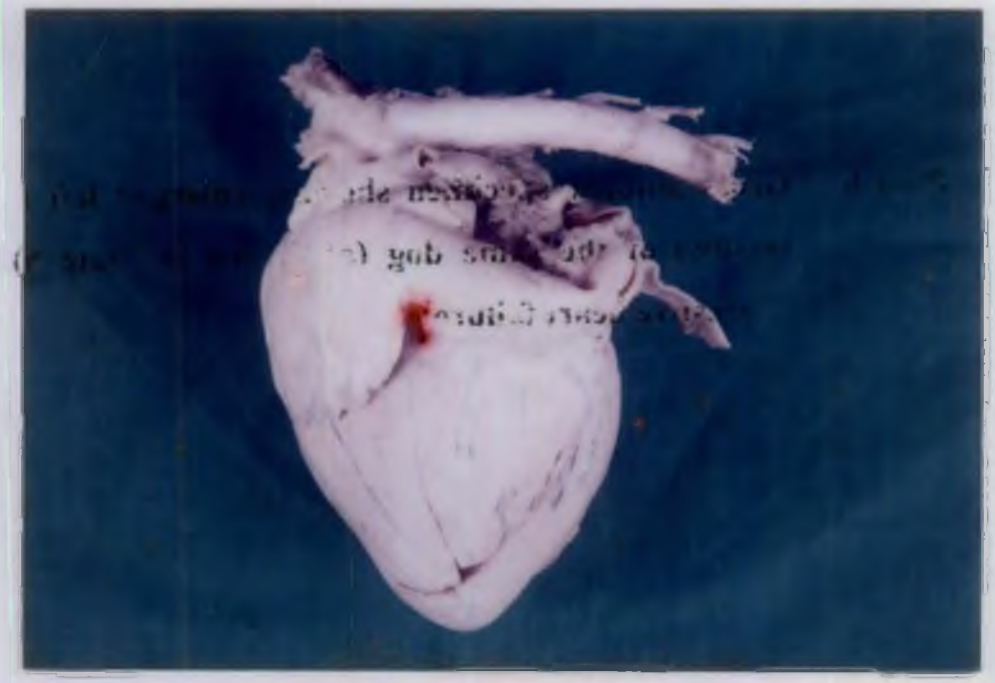
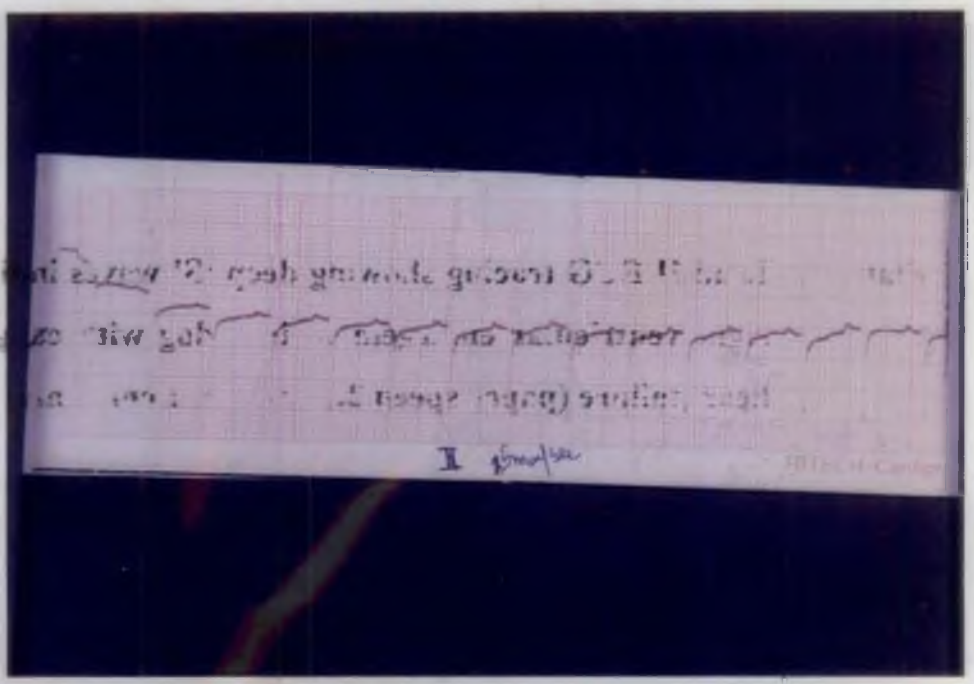


Plate 7 **Lead II ECG tracing showing deep 'S' waves indicating right ventricular enlargement in a dog with congestive heart failure (paper speed 25 mm/sec; 1 cm 1 mv)**

Plate 8 **Echocardiogram showing thickened and hypertrophied right ventricle (arrow) of the same dog (as shown in Plate 7) with congestive heart failure**



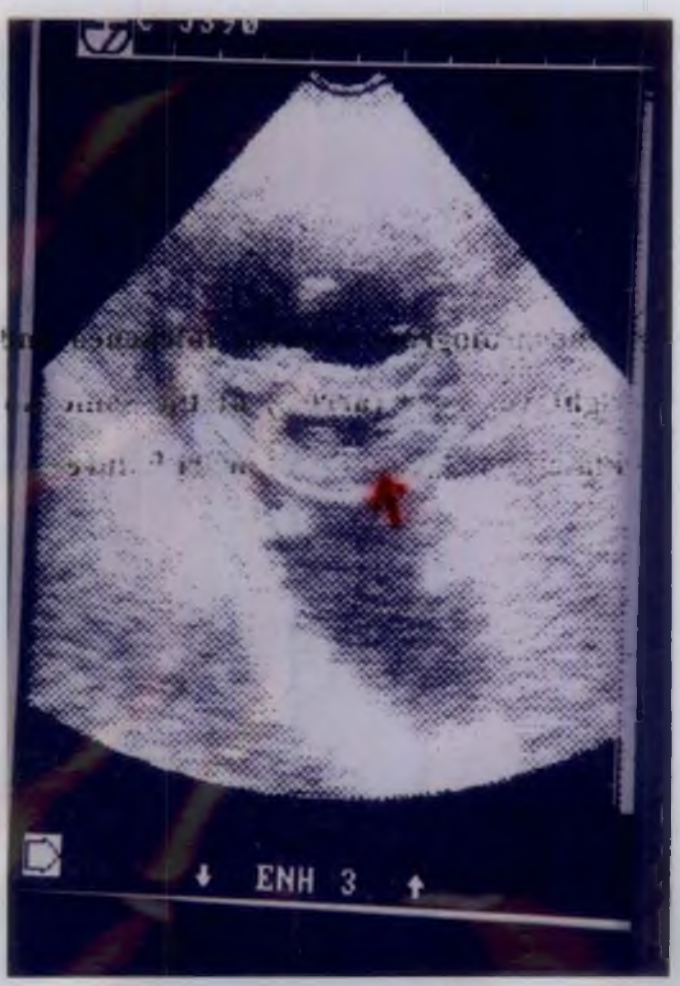
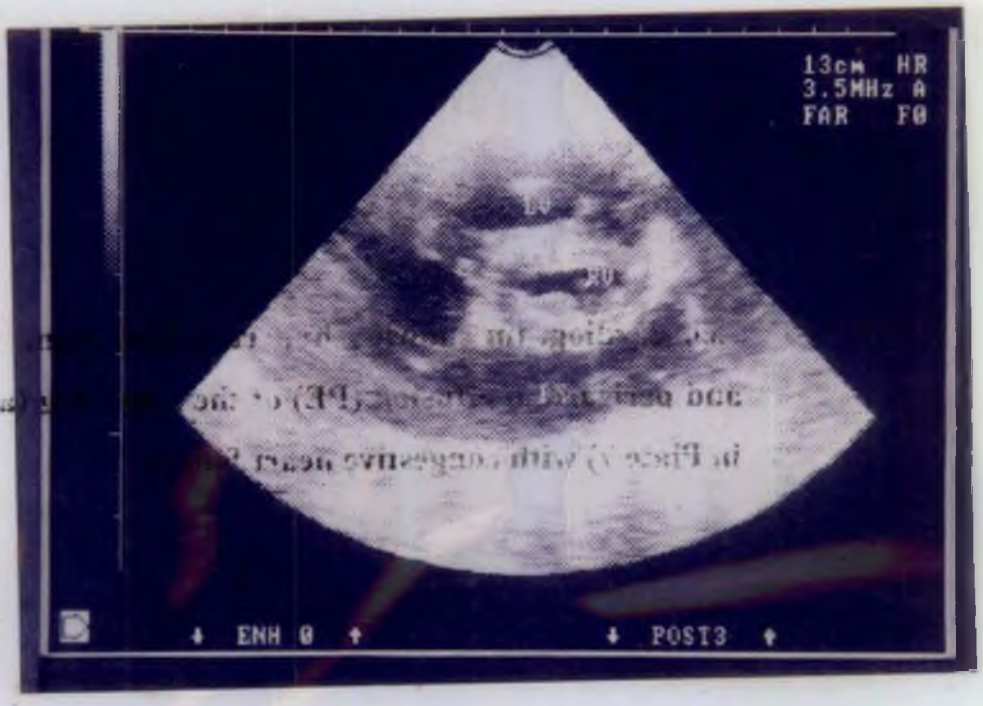

ENH 3

Plate 9 **Echocardiogram showing hypertrophied right ventricle and pericardial effusion (PE) of the same dog (as shown in Plate 7) with congestive heart failure**

Plate 10 **Lead II ECG tracing showing ventricular tachycardia in a dog with congestive heart failure (paper speed 25 mm/sec; 1 cm = 1 mv)**


13cm HR
3.5MHz A
FAR F0
ENH 0
POST3

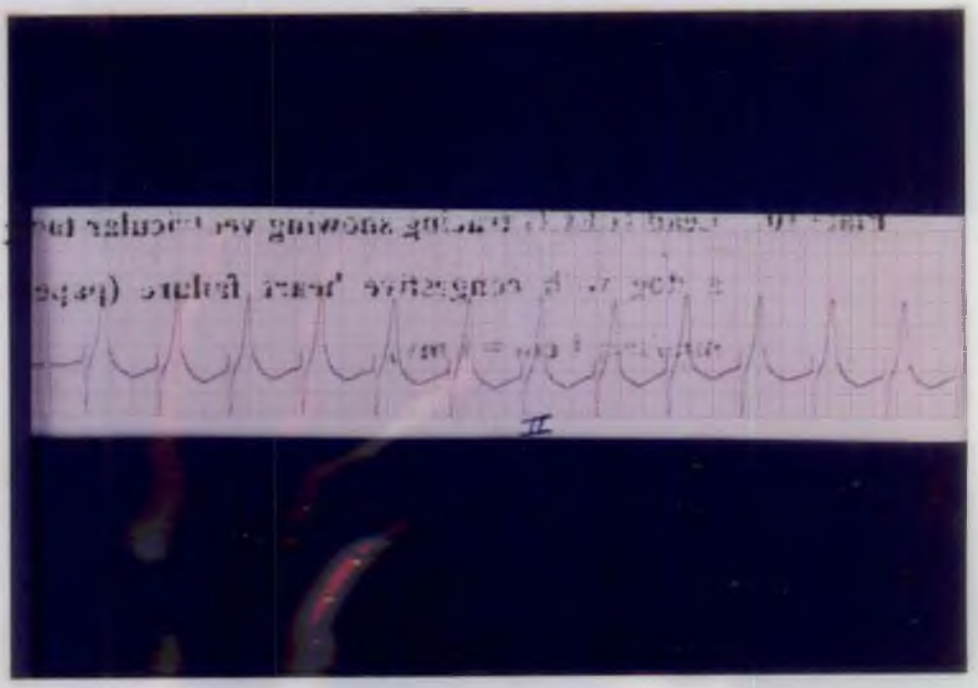
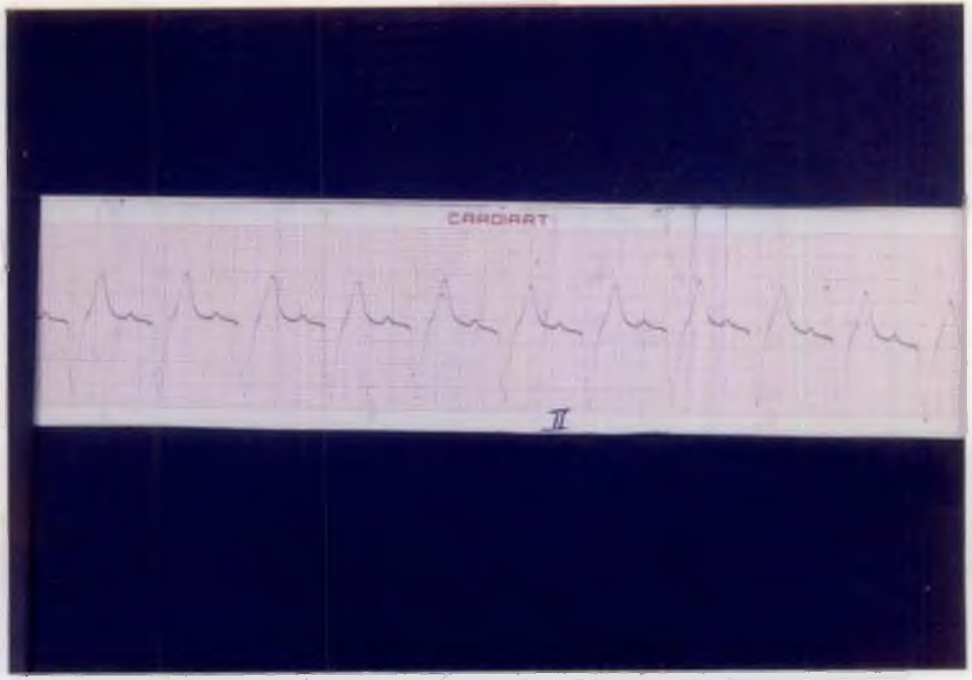


Plate 11 **Lead II ECG tracing showing right bundle branch block in a dog with congestive heart failure (paper speed 50 mm/sec; 1 cm = 1 mv)**

Plate 12 **Lead aVF ECG tracing showing right bundle branch block of the same dog (as shown in Plate 11) with congestive heart failure (paper speed 50 mm/sec; 1 cm = 1 mv)**


CARDIART
II

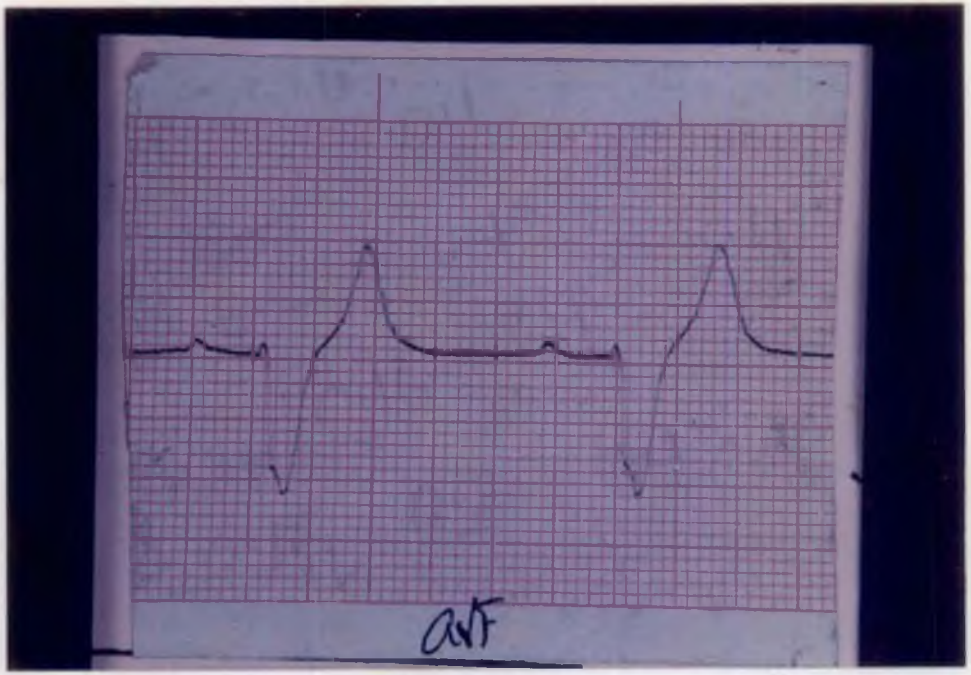
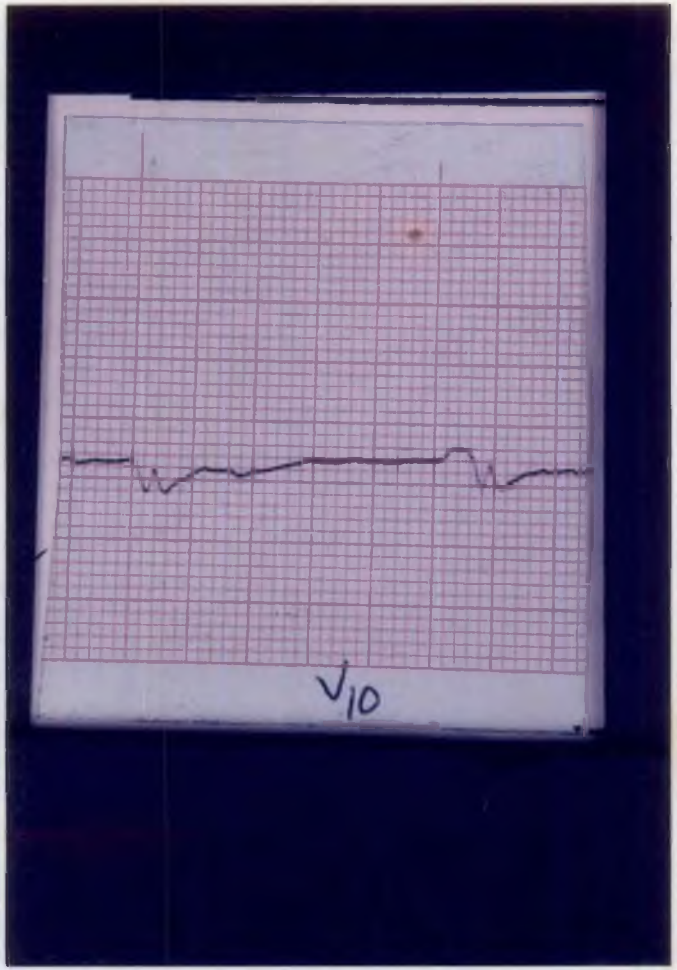

aVF

Plate 13 Lead V_{10} ECG tracing showing 'W' pattern in right bundle branch block of the same dog (as shown in Plate 11) with congestive heart failure (paper speed 50 mm/sec; 1 cm = 1 mv)

Plate 14 Lead avL ECG tracing showing right bundle branch block of the same dog (as shown in Plate 11). The QRS complex is in the opposite direction (positive) (paper speed 50 mm/sec; 1 cm = 1 mv)


V10

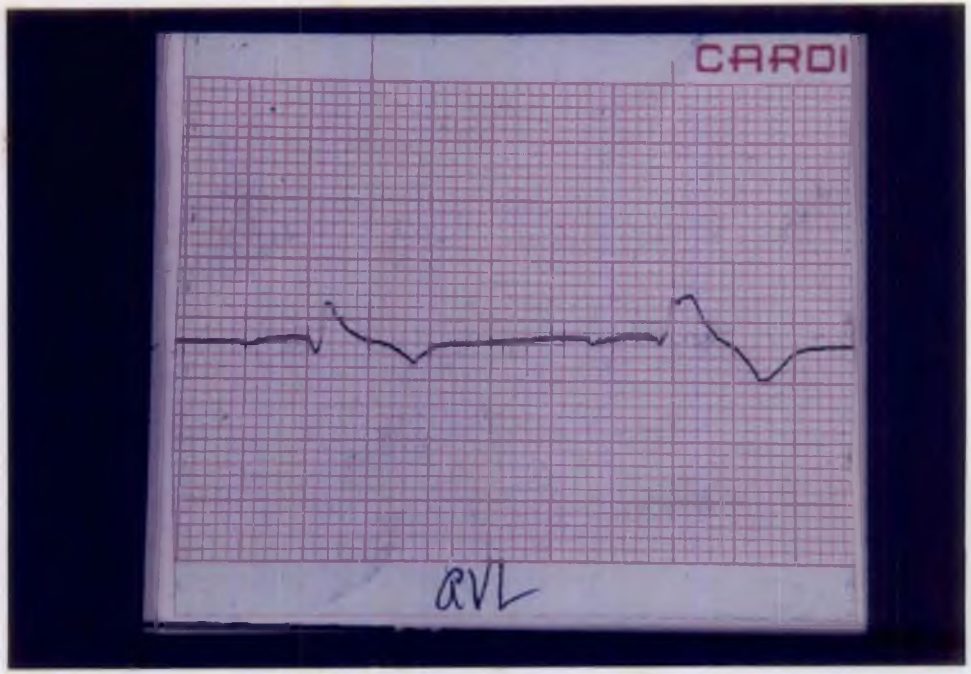
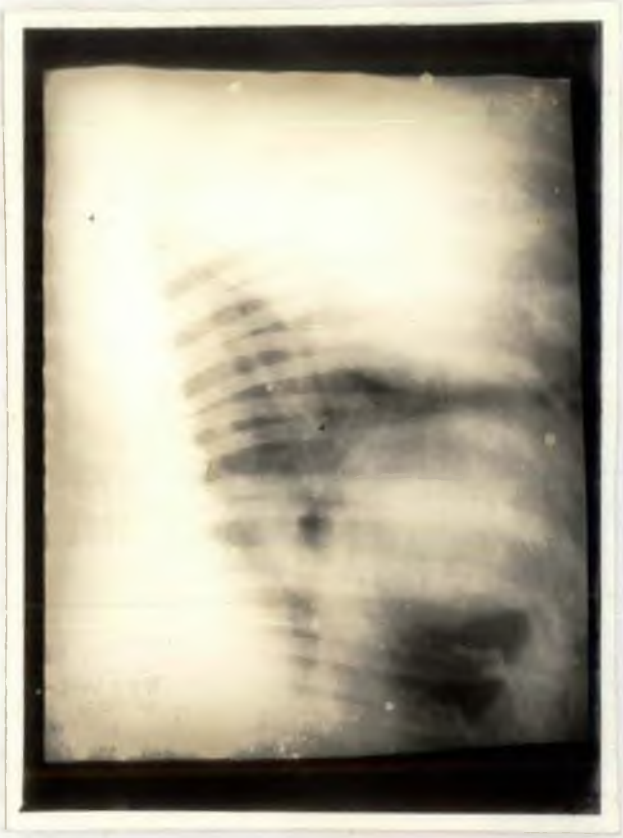

CARDI
aVL

Plate 15 **Radiograph showing increased contact between cardiac silhouette and sternum indicating right ventricular enlargement of the same dog (shown in Plate 11) with congestive heart failure**

Plate 16 **Lead II ECG tracing showing deep 'S' wave (S_2 pattern) in a dog with right ventricular enlargement secondary to anaemia (paper speed 25 mm/sec; 1 cm = 1 mv)**



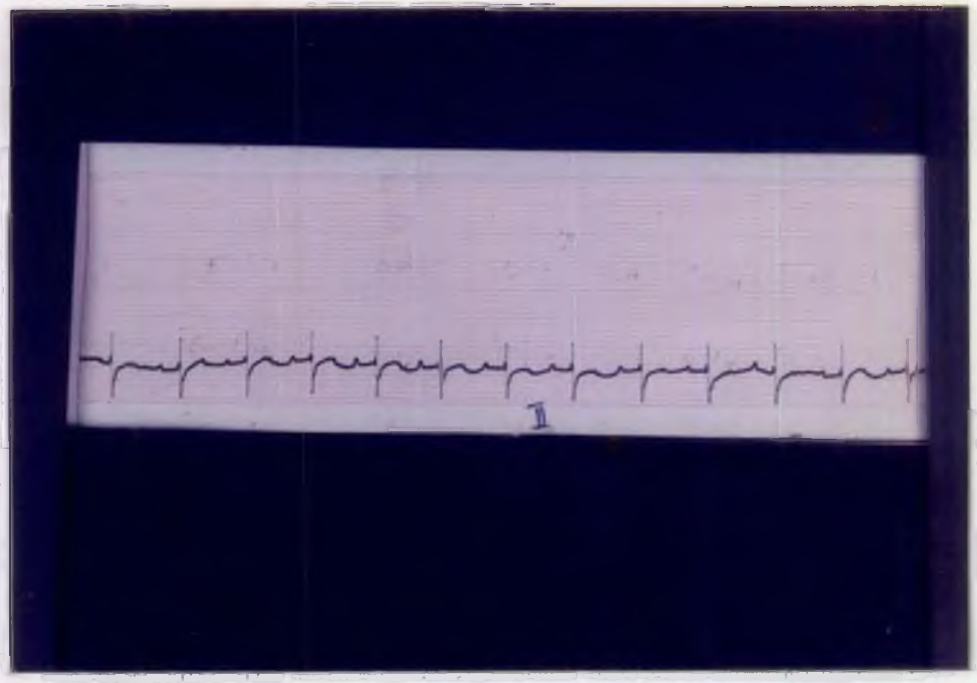
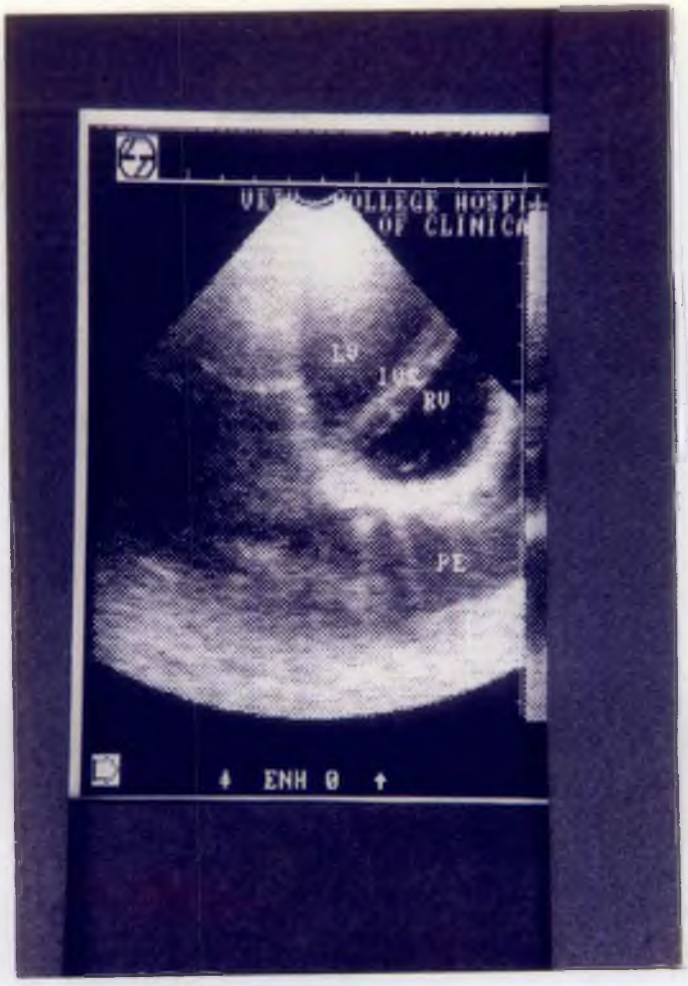

II

Plate 17 **Echocardiogram showing pericardial effusion (PE) in a dog with right ventricular enlargement secondary to anaemia**

Plate 18 **Lead II ECG tracing showing peaked 'T' wave in a dog with hyperkalaemia due to renal disease (paper speed 25 mm/sec; 1 cm = 1 mv)**


RV
PE
ENH 0

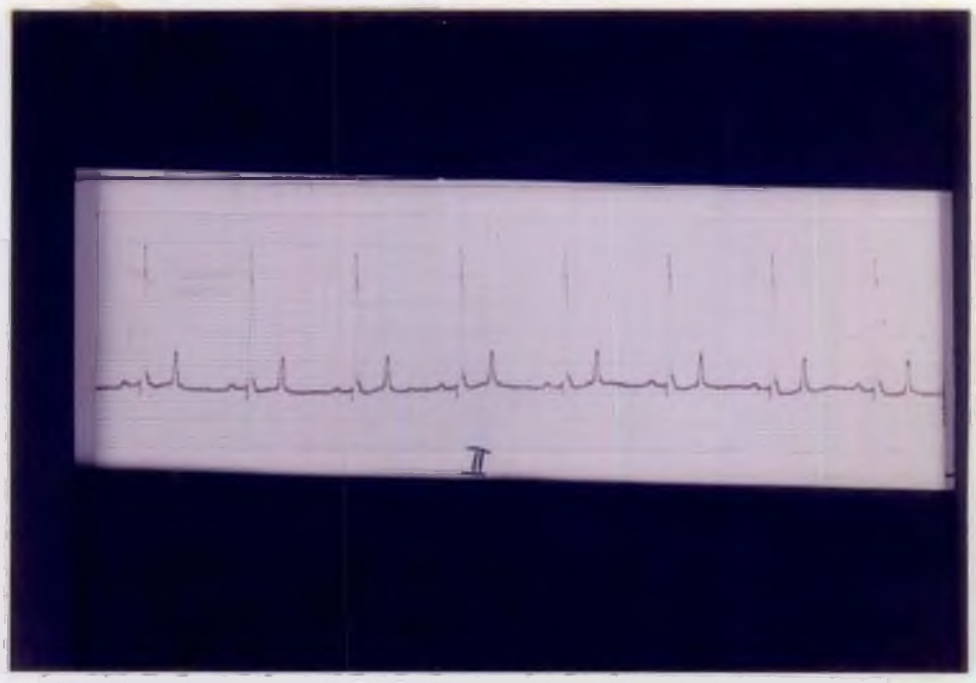
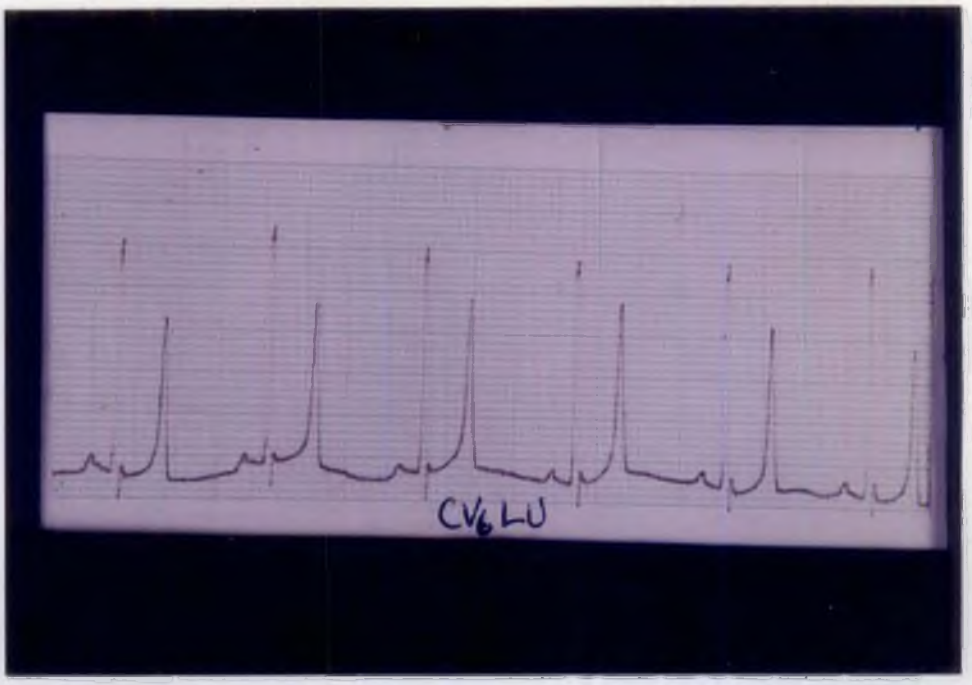
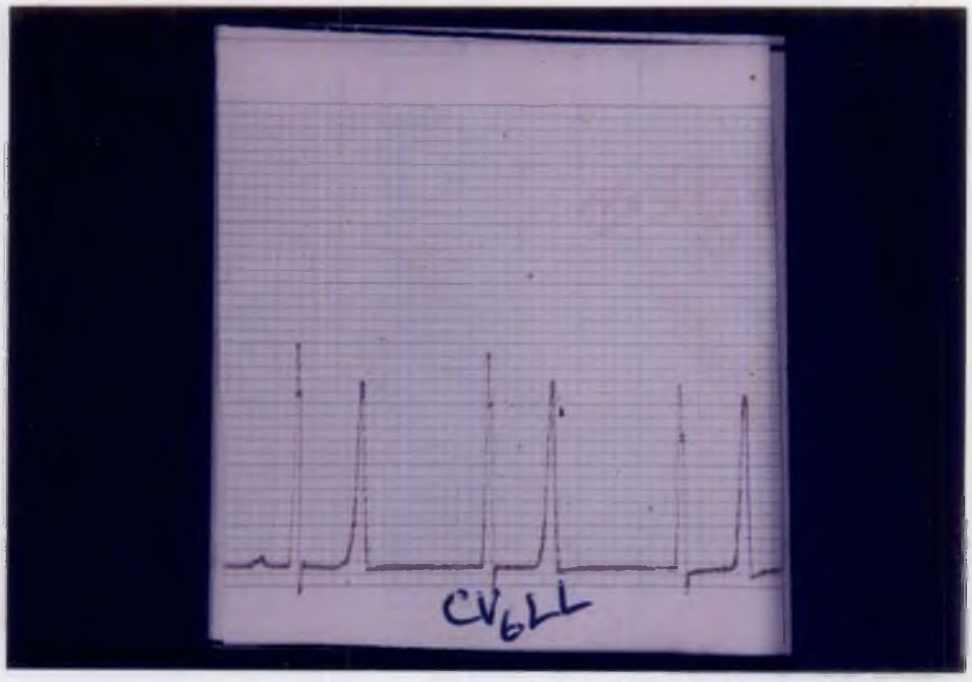

II

Plate 19 **Lead CV_6LU ECG tracing showing tall and peaked 'T' wave of the same dog (as shown in Plate 18) with hyperkalaemia due to renal disease (paper speed 25 mm/sec; 1 cm= 1 mv)**

Plate 20 **Lead CV_6LL ECG tracing showing tall and peaked 'T' wave of the same dog with hyperkalaemia (as shown in Plate 18) due to renal disease (paper speed 25 mm/sec; 1 cm = 1 mv)**


CV6LU


CV6LL

Discussion

5. DISCUSSION

The results of various parameters under the study like occurrence, signalment, clinical and physical examination findings, electrocardiography, radiography, ultrasonography, autopsy examination findings, haematological and serum biochemical results of dogs having electrocardiogram abnormalities were discussed in detail.

5.1 Occurrence

The occurrence (1.3 per cent) of cardiac disorders obtained in the present study was very less when compared with those reported by earlier workers. A higher occurrence of 11.3 and 3 per cent were observed by Detweiler and Patterson (1965b) and Taylor and Sittinikow (1968).

5.2 Signalment

5.2.1 Breed, Age and Sex

The most common cardiac syndrome encountered in the present study was congestive heart failure (53.85 per cent). Cardiac involvement secondary to anaemia (23.08 per cent) and renal disease (23.08 per cent) were also observed.

In the present study the occurrence of cardiac disorders were high in males than in females (12:1). Detweiler *et al.* (1961) had reported the incidence of congestive heart failure, two and a half times greater in males than in

females. The incidence of heart disease observed by earlier workers was only slightly greater in male than in female dogs (Detweiler and Patterson, 1965b). The reason for high incidence in males, in the present study could be due to the preference of male dogs to female dogs by the people of Kerala so as to avoid the breeding nuisance of female dogs.

The breeds affected with congestive heart failure (CHF) in the present study were Dachshund, Spitz and Dobermann pinschers. Five out of seven dogs with CHF were small breeds like Dachshund and Spitz.

In the current study CHF was found to be more in geriatric small breeds. In one Dachshund dog (Case No.1) the cause for congestive heart failure was mitral insufficiency. In other dogs the specific etiology for CHF were not elucidated. The clinical signs of CHF resulting from mitral valve endocardiosis were observed almost exclusively in geriatric small breeds (Abbot, 1998). Chronic mitral valvular fibrosis resulting in mitral insufficiency was the most frequent cause of CHF in dogs (Ettinger and Suter, 1970). In the present study the age of the small breeds with CHF ranged from four to eleven years.

Two out of seven dogs with CHF were Dobermann pinschers with an age of six and eight years. They had right sided heart failure secondary to pulmonary affections. Both the dogs were males. An earlier study showed that after five years of age, dogs had a dramatic increase in the incidence of endocardiosis and pulmonary fibrosis (Hamlin, 1990). In the present study,

also, the Dobermann pinschers with congestive heart failure had pulmonary fibrosis in the lateral radiograph.

In the group II with ventricular enlargement secondary to anaemia all the dogs were males. Two out of the three dogs were Dobermann pinschers with an age of five and six months. One dog was a Labrador with two years of age.

In the group III with renal disease, there were two Mongrels and one German Shepherd. Their age ranged from seven to nine years. One of the Mongrel was a female and the other two in this group were males.

5.3 Clinical signs

5.3.1 Group I

Congestive Heart Failure (CHF) is the most common form of heart failure in small animals. It could result from any number of cardiac diseases. In dogs it usually resulted from mitral valve regurgitation due to endocardiosis or dilated cardiomyopathy (Abbot, 1998).

The clinical signs observed in the present study associated with CHF were orthopnoea, cough, crackles upon auscultation of the lung area, exertional dyspnoea and exercise intolerance which were similar to those observed by Fisher (1967). Orthopnoea is the inability to breathe except in an upright position (Ettinger and Suter, 1970). This occurs because the circulating blood volume increases in CHF, resulting in increased venous return, pulmonary

congestion and diminished pulmonary compliance (Friedberg, 1966). Cardiac dyspnoea resulted from increased pulmonary blood volume and elevated pulmonary venous and capillary pressures (Ettinger and Suter, 1970). The increased pulmonary blood volume and elevated pulmonary venous and capillary pressures were responsible for the changes in alveolar capillary diffusion causing blood-tinged frothy fluid to be expectorated during coughing (Friedberg, 1966). In the present study also blood tinged phlegm was expectorated by one dog with left sided CHF (Case No.1). Crackles occurred when there was pulmonary oedema. The paroxysmal dyspnoea noticed in all the dogs with left sided heart failure (Case No. 1, 2, 3, 4) were attacks of respiratory distress. Paroxysmal dyspnoea was common when the dogs were in the recumbent position (Ettinger and Suter, 1970).

The two dogs with left sided congestive heart failure in the present study had holosystolic murmur in the left caudal sternal border with grades IV/VI (Case No.1) and III/VI (Case No.2) and with a whooping quality could be due to the mitral insufficiency (O'Grady, 1995). Syncope was noticed in three dogs, two with mitral insufficiency and other with ventricular tachycardia associated with left sided congestive heart failure.

Syncope in those dogs with CHF was suggestive of reduced cardiac output and cerebral hypoxia. Syncope of cardiac origin are called Stokes-Adams Seizures (Ettinger and Suter, 1970). In mitral insufficiency syncope

could result from reduced left ventricular forward flow as a result of regurgitation of blood into the left atrium. It could also be due to paroxysms of coughing called "post-tussive syncope" (O'Grady, 1995). He also opined that coughing associated with mitral insufficiency was due to compression of left main stem bronchus from an enlarged left atrium. In the present study also an enlarged left atrium was observed in the Dachshund with congestive heart failure due to mitral insufficiency which was confirmed by electrocardiography, radiography, ultrasonography and postmortem examination. Syncope observed in ventricular tachycardia could be attributed to the rapid heart beats and loss of atrioventricular synchrony resulting in reduced ventricular filling and cardiac output (Lunney and Ettinger, 1995).

In the current study alterations in the characters of pulse could be observed in three dogs with congestive heart failure. Two dogs with left sided CHF due to mitral insufficiency had a jerky pulse (Case No.1 and 2). A jerky but less accentuated pulse could be palpated in mitral insufficiency (Gould *et al.*, 1968). The reason for this type of pulse could be attributed to the volume overloading of the left ventricle and shortened ejection time resulting in loss of diastolic pressure (Goodwin, 1995).

In the dogs with ventricular tachycardia associated with congestive heart failure (Case No.3 and 4) pulse deficits, paroxysms of rapid regular heart beats and cannon 'a' waves in the jugular furrow were noticed. Similar observations were recorded by Lunney and Ettinger (1995).

Cannon 'a' waves were intermittent, bounding jugular venous pulsations that could occur when disturbances of the cardiac rhythm caused the right atrium to contract on a closed atrioventricular valve, thus forcing the blood to move retrograde into the jugular veins (Ettinger and Suter, 1970). Ventricular tachycardia interfered with diastolic filling and caused insufficient filling of the ventricle. The premature contraction occur before adequate ventricular filling and result in no ejection and a pulse deficit (Goodwin, 1995).

In two dogs with left sided congestive heart failure (Case No.1 and 2) due to mitral insufficiency, gallop rhythm was observed during auscultation. Gallop rhythm indicated myocardial failure (O'Grady, 1995).

Two dogs (Case No.1 and 6) with congestive heart failure exhibited open mouth breathing with extension of neck and abduction of the elbows. Patients with thoracic fluid accumulation secondary to heart failure might stand with their elbows abducted and with the neck extended in an attempt to improve ventillation (Bond, 1997a).

Left ventricular 'heave' and precordial thrill were noticed in one dog with left sided CHF (Case No.1). An apical thrust (left ventricular heave) and a precordial thrill could be observed in dogs with advanced mitral insufficiency. Extensive movement of the left thoracic wall, referred as left ventricular heave suggested cardiac enlargement. Thrills are palpable vibrations synchronous with the heart beat present over the precordium in the presence of a loud heart murmur (Ettinger and Suter, 1970).

In the present study all the dogs in this group, were found to be anorectic. Anorexia and gastrointestinal disturbances might occur in association with cardiac disease (Ettinger and Suter, 1970). Ascites was noticed in all the dogs with right sided congestive heart failure (Case No.5, 6 and 7). Cardiac cachexia was observed in one dog with right sided congestive heart failure (Case No.6). Weight loss, especially with abdominal distension (ascites) was common in right sided heart failure in dogs and might accompany weakness. Severe weight loss due to a heart problem is known as cardiac cachexia (Bond, 1997). Serous effusions into the peritoneal cavity developed as a result of increased portal or capillary hydrostatic pressure. Cachexia in congestive heart failure was due to malnutrition as a result of poor appetite, malabsorption from the gastrointestinal tract and a variety of gastrointestinal disturbances (Ettinger and Suter, 1970).

In one dog (Case No.6) with right sided congestive heart failure muffling of heart sounds was observed due to pleural and pericardial effusion. This was confirmed by radiography. Animals that were dyspnoeic from pleural effusion had decreased heart and lung sounds (Bond, 1997a).

The mean values of the clinical parameters like respiration, temperature and pulse rates in this group with congestive heart failure were within the reference range eventhough pulse deficits were noticed in two dogs. The body temperature and pulse quality were often normal in patients with congestive

heart failure and pathological arrhythmias might result in varying pulse strengths and rates (Abbot, 1998).

5.3.2 Group II

In the present study all the dogs in this group were anaemic (mean haemoglobin level of 7 ± 0.58) and it resulted in ventricular enlargement confirmed by electrocardiography and radiography. In the present study two dogs had right ventricular enlargement and one had left ventricular enlargement. In chronic anaemia both the right and left ventricles were hypertrophied in dogs (Scheel and Williams, 1985). In human beings haemoglobin levels below 10 g/dl could lead to left ventricular (LV) hypertrophy/dilatation and a lower quality of life. Further the normalisation of haemoglobin does not lead to regression of already established concentric LV hypertrophy or dilatation, however it could prevent further progression of LV dilatation (Foley *et al.*, 2000).

In the current study, the reason for anaemia could not be elucidated in most of the cases. One dog had severe cestodiasis. Other clinical signs observed in the dogs of this group were anorexia, stunted growth, pale visible mucous membranes, weakness and respiratory distress. Lethargy, depression, anorexia, weakness and dyspnoea are the signs of chronic anaemia (Straus, 1998). Stunted growth observed in two dogs (Case No.8 and 9) aged five and six months could be due to gastrointestinal parasitism, resulting in a relative deficiency in nutrition (Ihle, 1995).

In the dog having cestodiasis pericardial effusion was evident in the lateral radiograph and echocardiogram. Muffling of heart sounds observed in that dog was due to pericardial effusion. Muffled, soft or tinny sounds suggested pericardial or pleural effusion (Ettinger and Suter, 1970). The increased intensity and area of cardiac sound auscultated in another dog (Case No.10) with left ventricular enlargement could be attributed to the increased cardiac output seen in anaemia to preserve oxygen delivery to the tissues (Lewis, 1977). Increase in cardiac output is brought about by an increase in the stroke volume associated with cardiac dilatation and hypertrophy. On auscultation the heart sounds might be accentuated (Dunn, 1991).

The haemic murmurs observed in the dogs of this group were physiological murmurs produced by functional changes such as increased cardiac output seen in anaemia (Detweiler and Patterson, 1967). The low haemoglobin level decreased the viscosity and increased the velocity of blood to produce the turbulence in the valvular area to produce murmurs (Detweiler and Patterson, 1965a). The hyperkinetic pulse observed in those dogs in the present study was due to decreased vascular resistance in anaemia (Goodwin, 1995).

The mean values of the clinical parameters like respiration, temperature and pulse rates of this group were within the normal reference range for dogs.

5.3.3 Group III

Anorexia, polydipsia, oliguria, anuria, vomiting, diarrhoea and melaena were the major clinical signs observed in this group. These clinical findings were suggestive of renal disease. Vomiting, diarrhoea or constipation, depression, oliguria or anuria, bradycardia, dehydration, hypothermia, mucosal injection, azotemia, hyperphosphatemia and hyperkalaemia were reported in renal failure (Fleming *et al.*, 1989).

Anaemia, haemic murmurs and hyperkinetic pulse were also observed in the dogs of this group. One dog had oral ulceration and tongue tip necrosis. Polyuria, polydipsia, weight loss, anorexia, vomiting, diarrhoea, oral ulceration, lingual discoloration and necrosis, mucosal pallor, scleral injection and dehydration were reported in chronic renal failure (Mikiciuk *et al.*, 1989). Haemic murmurs, hyperkinetic pulse and aortic thudding observed in the present study were due to anaemia evidenced by low haemoglobin level (6 ± 0.59 g/dl).

Among the clinical parameters the mean value for the pulse rate and temperature showed a decrease among the animals of this group (Table 2). Bradycardia and hypothermia were reported in renal failure by Fleming *et al.* (1989). They also stated that bradycardia could occur in renal disease as a result of hyperkalemia.

5.4 Electrocardiography

5.4.1 Group 1

The heart rates of all the dogs with CHF were within the normal limits except in a Dachshund with right sided failure (Case No.6) (Table 21). This indicated sinus tachycardia. Sinus tachycardia could be seen in right sided congestive heart failure due to acute cor pulmonale (Tilley, 1992).

The notching of the 'P' waves with increased duration in all the leads in a Dachshund with left sided CHF (Case No.1) due to mitral insufficiency was suggestive of left atrial enlargement. When the left atrium enlarged, the 'P' waves became wide and notched (P intake) (Ettinger and Suter, 1970).

The increased amplitude of 'Q' wave in leads, I, II and aVF in two dogs (Case No.1 and 2) with left sided heart failure indicated septal hypertrophy or biventricular enlargement (Tilley, 1992). The increased amplitude of 'R' wave in lead II in dogs with left sided heart failure (Case No.1 and 2) and slurring of the 'S-T' segment indicated left ventricular enlargement (Bolton, 1975; Aronson and McCaw, 1984).

Deep 'S' waves in lead II, III (S_2 and S_3 pattern) and in aVF, CVLL, CV_6LU and low R/S ratio in dogs with right sided congestive heart failure (Case 5, 6 and 7) were suggestive of right ventricular enlargement (McCaw and Aronson, 1984; Tilley, 1992; Bolton, 1975).

In the Dachshund with right sided congestive heart failure (Case No.6) the amplitude of 'R' wave in lead II and CV_6LU (1.0 and 0.6 mvs respectively) were less when compared with the reference range for Dachshunds used as controls (Table 6 and 20). This could be due to pericardial or pleural effusion. Complexes of diminished amplitude were seen on the electrocardiogram in association with pericardial effusion (Ettinger and Suter, 1970). The low voltage of the QRS complexes were due to the 'short-circuiting' action of the pericardial fluid (Tilley, 1992).

In one dog (Case No.3) there was idioventricular tachycardia associated with left sided heart failure with a heart rate of 100 bpm. The ventricular tachycardia between 60 and 100 bpm is termed idioventricular tachycardia or enhanced ventricular rhythm (Tilley, 1992).

The wide and bizzare QRS complexes with 'P' waves hidden in the QRS complexes or QRS complexes without an associated 'P' wave indicated ventricular premature complexes (VPC's) (Bolton, 1975; Tilley, 1992; Bond, 1997b). Three or more ventricular extrasystoles in a row are defined as ventricular tachycardia (Conway, 1977 and Bond, 1997b).

In dogs with ventriculr tachycardia in the present study, the QRS complexes were wide, bizzare and of the same shapes. Ventricular premature complexes with same shape are called as unifocal ventricular tachycardia (Tilley, 1992). The major QRS deflection was negative in lead II and this indicated that the ectopic focus was in the left ventricle (Bond, 1997b).

The Dobermann pinschers with right sided congestive heart failure had electrocardiogram changes suggestive of right bundle branch block (RBBB). The electrocardiogram changes were in agreement with Tilley (1992) who stated that in RBBB the QRS complex was of greater duration than 0.08 sec with right axis deviation. In RBBB the QRS complex is positive in aVR, aVL and CV5RL leads, with large wide 'S' waves in leads I, II, III, aVF, CV6LL and CV6LU (Bond, 1997b). Also the 'W' pattern is usually seen in lead V10 (Tilley, 1992). Both the dogs (Case No.5 and 7) in the present study had right axis deviation in the frontal plane. These electrocardiographic features were similar to those observed by Bond (1997b) and Tilley (1992).

Electrocardiographically, right bundle branch block could mimic severe right ventricular hypertrophy because of deep 'S' waves in lead I, II and III. Right ventricular hypertrophy however, seldom causes the 'S' waves to be so wide. Confirmatory diagnosis of right ventricular enlargement could be made by thoracic radiograph in such cases (Bolton, 1975).

5.4.2 Group II

In this group two dogs (Case No.8 and 9) had electrocardiographic signs suggestive of right ventricular enlargement and one dog (Case No.10) had left ventricular enlargement.

The prominent electrocardiographic changes observed in the dogs with right ventricular enlargement were deep 'S' waves in lead II, III (S_2 and S_3

pattern), aVF, CV_6LL, CV_6LU and right axis deviation suggestive of right ventricular enlargement. These findings were in agreement with those of Aronson and McCaw (1984), Tilley (1992) and Bolton (1975). The increased amplitude of 'R' wave observed in one dog (Case No.10) was indicative of left ventricular enlargement (Bolton, 1975). The deep 'Q' wave observed in that dog (Case No.10) indicated interventricular septal hypertrophy or biventricular enlargement (Tilley, 1992).

5.4.3 Group III

The decreased heart rate observed in two dogs (Case No.11 and 13) in this group indicated sinus bradycardia. Sinus bradycardia could occur in renal disease due to hyperkalemia (Fleming *et al.*, 1989).

The prominent electrocardiographic changes observed in this group were peaking of 'T' wave in lead II and chest leads like CV_6LU and CV_6LL and prolongation of 'Q-T' interval. Prolongation of Q-T interval and peaked narrow 'T' waves of high amplitude were seen in the electrocardiogram as a result of hypocalcemia and hyperkalemia respectively (Ettinger and Suter, 1970; Bolton, 1975).

Generally T wave should not be larger than one-fourth the height of the 'R' wave (Ettinger and Suter, 1970). In the present study the chest leads like CV_6LL and CV_6LU were found to be more sensitive in detecting hyperkalemia. In this group, the 'T' wave of both CV_6LU and CV_6LL were much greater than

one-fourth of the amplitude of the 'R' waves. An interesting observation was that in lead II the amplitude of 'T' wave was not greater than one-fourth of the amplitude of 'R' wave even in the presence of hyperkalemia. So chest leads were more sensitive in detecting hyperkalemia in renal diseases when compared to the usually used second lead.

5.5 Radiography

5.5.1 Group I

Prominent radiographic signs observed in animals with congestive heart failure in the present study were left atrial enlargement, left and right ventricular enlargement and tracheal elevation.

The lateral radiograph of the cardiac silhouette producing a "wing shaped" shadow caudal to the tracheal bifurcation and dorsal to the caudal venacava was indicative of left atrial enlargement. The enlarged left atrium produced a "wing-shaped" shadow caudal to the tracheal bifurcation and dorsal to the caudal venacava (Burk and Ackerman, 1996).

One dog with left sided congestive heart failure due to mitral insufficiency showed an enlarged cardiac silhouette with straightened, upright caudal cardiac margin. The caudal cardiac margin in that dog formed a sharp angle with the dorsal cardiac margin. The tracheal bifurcation was markedly elevated. The radiographic signs of left sided cardiac silhouette enlargement

were loss of the caudal cardiac waist, elongation and straightening of the caudal cardiac border and elevation of trachea (Fagin, 1988).

The accentuation of the convexity of the caudal cardiac border noticed in some dogs (Case No.2, 3 and 4) was indicative of mild left ventricular enlargement (Burk and Ackerman, 1996).

In some dogs there was increased contact between cardiac silhouette and the sternum (Case No.4, 5, 6 and 7). In right sided cardiac silhouette enlargement there was increased sternal contact (Fagin, 1988). In dogs with right sided congestive heart failure (Case No.5, 6 and 7) the cranial border of the cardiac silhouette was vertically oriented. This indicated right ventricular enlargement. In the lateral radiograph the initial change that occurred as the right ventricle enlarged, was that the angle formed between the cranial outline of the heart and the vessels in the cranial mediastinum became more obtuse. As the degree of enlargement increased, the cranial aspect of the cardiac silhouette started to bulge cranially, becoming rounded so that more of the heart shadow appeared to be in contact with the sternum (Wyburn and Lawson, 1967).

In two dogs with right sided congestive heart failure (Case No.5 and 7) the caudal lung lobes had interstitial (reticular) pattern of density. The interstitial pattern infiltrates are described as fine linear, reticular or nodular pattern of density and may be due to the presence of fluid or cells (including neoplastic) or fibrosis within the supporting tissues of lung (Burk and Ackerman, 1996). One dog with right sided failure (Case No.6) had pleural

effusion evident in the lateral radiograph. Transudation of fluid into the pleural space observed in the present study indicated impaired lymphatic drainage due to pulmonary hypertension or due to the elevated systemic venous pressure seen in both left and right heart failure (Ettinger of Suter, 1970).

5.5.2 Group II

In this group one dog with right ventricular enlargement exhibited pericardial effusion evidenced by the smooth cardiac silhouette in the lateral radiograph. This was later confirmed by ultrasonography. If significant amount of pericardial effusion surrounds the heart, it would smooth the outline of the cardiac silhouette and heart base retained its normal shape (Burk and Ackerman, 1996).

In another dog with left ventricular enlargement, the tracheal elevation at the base of the heart and the accentuation of the normal convexity of the caudal cardiac margin were observed which indicated left ventricular enlargement (Burk and Ackerman, 1996).

5.5.3 Group III

No changes were evident in the cardiac silhouette in this group. In human beings anaemia is considered as the most important factor for the development of left ventricular hypertrophy in uremic patients (Berweck *et al.*, 2000).

Anaemia and hypertension in renal insufficiency could cause left ventricular hypertrophy. They play an important role in the pathogenesis of left ventricular hypertrophy as well as in the development of cardiac dysfunction (Jeren-Strujic' *et al.*, 2000).

In the present study eventhough anaemia was evident in this group, the renal disease might be acute and possibly the course of the disease was not sufficient to cause gross anatomic changes in the cardiac silhouette to be appreciated in the lateral radiograph.

5.6 Clinico-pathology

5.6.1 Haematology

5.6.1.1 Total erythrocyte count, haemoglobin, packed cell volume

In the present study, the mean ± SE values of total erythrocyte count, haemoglobin and packed cell volume for group I were within the normal range. Group II and III exhibited a considerably low value for the above parameters when compared with the control group. This was due to anaemia seen in group II and III animals. In anaemia there will be reduction in red cell mass (Roger, 1995). Group III animals had renal disease. The complete blood count of a patient with renal failure invariably reveals anaemia (Mikiciuk *et al.*, 1989). Anaemia of renal disease might be due to decreased erythropoietin production, decreased intake of iron and vitamin B_{12} as animals suffer from anorexia and vomiting and/or toxic suppression of haemopoiesis in the bone marrow (Sastry, 1983). It was also stated that blood sucking worms in the gastrointestinal tract

of growing young dogs could cause chronic haemorrhagic anaemia. One dog in group II had severe cestodiasis. The group II animals were young and occult parasitism might be the reason for anaemia seen in them. The endogenous production of erythropoietin is low in renal failure and it would result in decreased production of haemoglobin (Aird, 2000).

5.6.1.2 Mean corpuscular volume, mean corpuscular haemoglobin and mean corpuscular haemoglobin concentration

The mean corpuscular volume of the group I and II animals had a mean ± SE value of 64.88 ± 1.52 and 60.95 ± 3.04 respectively. Both these values were within the range for the control group (Table 24). The value of mean corpuscular volume of the group III animals was less when compared with the control group. This indicated microcytic anaemia. Group III animals had renal disease. In deficiencies of haemopoietic factors like erythropoietin there is decreased MCV (Benjamin, 1998). Microcytic anaemia could be seen in iron deficiency anaemia. Iron deficiency anaemia could develop in renal insufficiency due to loss of blood from gastrointestinal tracts (Wintrobe *et al.*, 1981). In the present study also there was gastrointestinal haemorrhage in the dogs of this group. Anaemia of renal disease might also be due to decreased erythropoietin production (Sastry, 1983 and Aird, 2000).

The mean corpuscular haemoglobin (MCH) value of group I and III animals were within the reference range (Table 24). The MCH for the group II had a mean ± SE of 18.21 ± 0.89 which was slightly less when compared with

the controls. The Mean Corpuscular Haemoglobin Concentration (MCHC) is a more valuable measurement than MCH, because in anaemias MCH is not altered proportionately with MCV (Benjamin, 1998).

In group II animals the mean cell volume (MCV) and mean cell haemoglobin concentration (MCHC) were within the reference range (Table 24). The slight drop in MCH was insignificant.

The mean ± SE values of MCHC of group I, II and III were 32.63 ± 0.82, 29.95 ± 1.37 and 30 ± 1.15 respectively. All these values were within the reference range of the control group of animals.

5.6.1.3 Total leucocyte count and differential count

The mean ± SE values of group I, II and III animals were within the referene range of the control group.

The mean ± SE values of the neutrophil and lymphocyte count of group I and II animals were within the normal range. Group III animals showed neutrophilia and lymphopenia. Group III animals had renal disease. In renal failure there will be anaemia with neutrophilia and lymphopenia (Mikiciuk *et al.*, 1988). Neutrophilia in renal failure could be due to systemic intoxication that occur in uraemia. It is prominent in patients with pericarditis or other inflammation related to severe azotemia (Wintrobe *et al.*, 1981).

The mean ± SE value of the monocyte count for group I, II and III were 2.86 ± 0.46, 3 and 2 respectively. All the values were within the normal range (Table 24).

The mean ± SE values of eosinophil count for group I, II and III were only slightly less which was clinically insignificant.

5.6.1.4 Erythrocyte sedimentation rate (ESR)

The mean ± SE value of group I and II animals were 3.43 ± 0.37 and 5 ± 1.73 respectively. The erythrocyte sedimentation rate of group II was more than the mean ± SE value of 3 ± 0.73 for the control group eventhough it was in the upper limit of the reference of 1 to 6 mm/30 minutes for the control group. Group III animals with renal disease had more severe anaemia than group II. The mean ± SE value of ESR for group III was 9.33 ± 0.67 which was greater than that of the control group. In anaemia ESR was accelerated due to small number of cells that could settle more easily in the large volume of fluid (Benjamin, 1998).

5.6.2 Serum biochemistry

5.6.2.1 Creatine Kinase (CK) (U/L)

The CK level for group I, II and III were all within the normal range. In human medicine serum CK isoenzymes serve as sensitive and specific indicators of cardiac infarction (Moss and Henderson, 1994). Cardiac infarction was infrequently encountered in clinical veterinary medicine where

serum CK isoenzymes were of no diagnostic value than total serum CK determination. In domestic species, CK isoenzymes analysis has not been shown to be of significant value (Hoffmann, 1990) as serum CK activity rapidly returns to normal after a muscle damaging incident (Hoffmann and Kramer, 1999). In the present study since the total CK level was not elevated, there was no indication for estimating the isoenzymes. CK might be elevated in acute myocardial infarction, skeletal muscle damage and pulmonary embolism (Benjamin, 1998). In acute myocardial infarction CK occurs in serum within 12 hrs, reaches a peak within 24 to 36 hrs and returns to normal within 4 days. In the present study, dogs in all the three groups might not be having any myocardial infarction or skeletal muscle damage of recent origin as evidenced by the normal CK level.

5.6.2.2 Alanine amino transferase (ALT) (IU/L)

The mean ± SE values of group I, II and III were 19.43 ± 0.81, 18.33 ± 1.66 and 16.67 ± 0.88 IU/L all within the range for the control group (Table 26).

In congestive heart failure due to liver congestion the ALT level could be increased (Benjamin, 1998). But in the present study in the group I with congestive heart failure the ALT level was within the reference range. This was not in agreement with that of Benjamin (1998). This could be due to the fact that the plasma half-life of this enzyme in dog is approximately sixty hours (Turk and Casteel, 1999). It is so common in animal practice that most of dogs

with congestive heart failure are brought to the hospital only in the advanced stage. By that time the rise in serum level of ALT following hepatocellular injury due to liver congestion would have come to the normal range. This may be the reason for normal ALT value in the serum of dogs with congestive heart failure in the present study. No significant elevations in ALT were usually observed in uremia (Cornelius, 1980). Group III animals had renal disease and was uraemic. The ALT level in this group was within the reference range and was in agreement with Cornelius (1980).

5.6..2.3 Sodium (mEq/l)

The group II and III animals had a mean ± SE value of 143 ± 1.73 and 144.33 ± 2.73 respectively which was within the range for the control group (Table 26). The group I animals had a mean ± SE value of 174.14 ± 2.38 which was considerably higher than that of the control group. In congestive heart failure the renin-angiotensin-aldosterone system promotes sodium and water retension (Abbot, 1998).

5.6.2.4 Potassium (mEq/l)

The group I and II animals had a mean ± SE value of 4.80 ± 0.08 and 4.5 ± 0.28 respectively, which were within the range for the control group (Table 26). In group III animals with renal disease, the potassium level (7.67 ± 0.33) was higher when compared with that of the control group. Hyperkalemia was a common finding in renal failure and could lead to life-threatening

cardiotoxicity and early death (Fleming *et al.*, 1989). Group III animals had electrocardiographic signs of hyperkalaemia, peaking and increased amplitude of 'T' wave in the ECG tracings, which indicated hyperkalaemia.

5.6.2.5 Total protein (g/dl)

The mean ± SE value for the group I and III animals were 6.54 ± 0.18 and 6.07 ± 0.07 respectively, which were within the range for the control group (Table 26). But the group II animals had a mean ± SE value of 5.73 ± 0.93 which was less than that of the control group. Group II animals were anaemic. Anaemia could lead to anorexia (Benjamin, 1998). Reduction in dietary protein cause hypoproteinaemia and hypoalbuminaemia in dogs (Allison, 1957).

5.6.2.6 Albumin (g/dl)

The mean ± SE value for group I animals was 3.91 ± 0.07 which was within the range for the control group. The albumin level for group II animals was 3.2 ± 0.70 which was slightly less when compared to the control group. Group II animals were anaemic. Anaemia could lead to anorexia (Benjamin, 1998). Dietary protein deficiency or depletion could manifest as hyproteinaemia and hypoalbuminaemia in dogs (Allison, 1957). Group III animals had renal disease and very low albumin value of 1.9 ± 0.06. In renal disease there could be proteinuria and hypoalbuminaemia (Mikiciuk *et al.*, 1989).

5.6.2.7 Albumin: Globulin ratio (A:G)

The mean ± SE value for the group I and II animals were 1.54 ± 0.13 and 1.15 ± 0.19 respectively, which were within the range for the control group (Table 26). The group III animals had a mean ± SE value of 0.46 ± 0.03 which was less when compared to the control group. This was due to marked hypoalbuminemia in this group. In renal disease there could be proteinuria and hypoalbuminemia (Mikiciuk *et al.*, 1989).

5.6.2.8 Creatinine (mg/dl)

The creatinine level of group I and II were 1.25 ± 0.08 and 0.93 ± 0.06 which were within the range for the control group. The group III animals had creatinine value (19.77 ± 1.29) which was higher than that of the control group. Increased serum creatinine indicated renal failure (Finco, 1980).

5.7 Ultrasonography

In the present study, one dog with left sided congestive heart failure revealed dilated left atrium, left ventricle and thickened mitral valve in two dimensional left apical four chamber view of the heart. This was suggestive of mitral insufficiency. Two dimensional examination was superior to M-mode for demonstrating left atrial and left ventricular dilation and also allowed more complete examination of the entire mitral valve structure (Kienle and Thomas, 1995). They also stated that abnormalities of the valve included irregular,

smooth thickening and knobby enlargements at the ends of the leaflets which had an echogenicity similar to or less than the normal valve.

One dog with right sided congestive heart failure (Case No.6) had increased thickness of right ventricular wall evident in the 2-D echocardiogram. Moderate to severe right ventricular hypertrophy is usually identified as thickening of the right ventricular wall and septum. The septum becomes flattened toward the left ventricle in systole, resulting in an ovoid left ventricular shape in the short-axis view. Pericardial effusion was evident in the echocardiogram of two dogs (Case No.6 and 9) as a hypoechoic space surrounding the heart between the pericardial sac and the ventricular walls (Kienle and Thomas, 1995).

5.8 Autopsy findings

In the present study, one dog with left sided congestive heart failure had dilated left atrium and ventricle, thick valvular leaflets and an enlarged annulus of the mitral valve on postmortem examination. This was suggestive of mitral insufficiency. In mitral valvular insufficiency, anatomically there was an enlarged annulus, short thick leaflets, short thickened chordae tendinae, upward malposition of atrophic or hypertrophic papillary muscles and enlargement of the left atrium and ventricle (Robinson and Maxie, 1993).

Summary

6. SUMMARY

The present study was conducted in the Department of Clinical Medicine, College of Veterinary and Animal Sciences, Mannuthy for a period of four semesters, during the years 1998 to 2000.

Canine patients brought to the Veterinary College Hospital, Mannuthy and University Veterinary Hospital, Kokkalai were used for the study. They were screened for cardiac involvement by detailed clinical examination.

Standard electrocardiogram patterns were worked out from sixty normal animals. Control animals were grouped according to different age groups (below one year and above one year) and breeds (German Shepherd, Dobermann pinscher, Dachshund, Spitz and Mongrel), so that six animals were there in each group.

Out of the total 1000 canine cases screened, 13 dogs were found to have electrocardiogram abnormalities (1.3 per cent).

Based on the clinical signs and electrocardiogram abnormalities, thirteen dogs were classified into three groups.

Group I : Dogs with congestive heart failure

Group II : Dogs with ventricular enlargement secondary to anaemia

Group III : Dogs with electrolyte imbalance due to renal disease

The parameters studied in these dogs were signalment and history, clinical examination, electrocardiography, radiography, haematology, serum biochemistry, ultrasonography and autopsy.

Out of the 13 cases (1.3 per cent) of electrocardiogram abnormalities, seven cases (53.85 per cent) were congestive heart failure, three cases (23.08 per cent) were with cardiac involvement secondary to anaemia and three cases (23.08 per cent) were with electrolyte imbalance due to renal disease.

Out of the seven dogs with congestive heart failure, two were Dachshunds (28.57 per cent), three were Spitz (42.85 per cent) and two were Dobermann pinschers (28.57 per cent). All the dogs with CHF were males. Their age ranged from six to eleven years.

Out of the three cases with anaemia two were Dobermann pinschers (66.66 per cent) and one was Labrador (33.33 per cent). All the dogs of this group were males. Their age ranged from five months to two years.

Among the three cases of electrolyte imbalance due to renal disease, two dogs were Mongrel (66.66 per cent) and one was a German Shepherd (33.33 per cent). Mongrels were males and German shepherd was a female. Their age ranged from seven to nine years.

The major clinical findings in the dogs with CHF (Group I) were anorexia, cough, exertional dyspnoea, exercise intolerance, orthopnoea, crackles on auscultation of lung area, murmurs, syncopic episodes, cardiac cachexia and ascites. Alteration in pulse characters were noticed in some dogs.

Clinical signs exhibited by the dogs with ventricular enlargement secondary to anaemia (Group II) were anorexia, pale visible mucous membranes, weakness and respiratory distress. Some dogs exhibited haemic murmur and all of them had a hyperkinetic pulse.

The dogs with electrolyte imbalance due to renal disease (Group III) exhibited anorexia, vomiting, diarrhoea, melaena, polydipsia, oliguria and anuria as the major clinical findings.

The major electrocardiogram abnormalities observed in the dogs with congestive heart failure (Group I) were notched P wave with increased duration indicating left atrial enlargement, increased amplitude of R wave indicating left ventricular enlargement, increased amplitude of Q wave indicating interventricular septal hypertrophy or biventricular enlargement, deep S waves indicating right ventricular enlargement and patterns of ventricular tachycardia and right bundle branch block.

The electrocardiographic features of dogs with ventricular enlargement secondary to anaemia (Group II) were tall R waves and deep 'S' waves indicating left and right ventricular enlargement respectively.

The electrocardiographic signs of electrolyte imbalance due to renal disease (Group III) were tall peaked 'T' waves in the chest leads and prolonged 'Q-T' interval indicated hyperkalaemia and hypocalcaemia respectively. These changes were prominent in chest leads rather than on the lead II.

The prominent radiographic signs observed in dogs with congestive heart failure in the present study were left atrial enlargement, left and right ventricular enlargement and tracheal elevation. Radiographic signs exhibited by dogs with ventricular enlargement secondary to anaemia included pericardial effusion, left ventricular enlargement and tracheal elevation.

Dogs with electrolyte imbalance due to renal disease had no radiographic signs of cardiac enlargement.

In the present study one dog with left sided CHF showed ultrasonographic evidence of left atrial and left ventricular dilatation and mitral valve thickening. Another dog with right sided CHF showed increased thickening of right ventricular wall. Pericardial effusion was evident in two dogs.

Post-mortem examination of one dog with left sided congestive heart failure due to mitral insufficiency revealed a dilated left atrium and ventricle, thick valvular leaflets and an enlarged annulus of the mitral valve.

In the present study Group II and III animals had anaemia evidenced by low value for total erythrocyte count, haemoglobin and packed cell volume.

The mean corpuscular volume of Group III animals were low indicating microcytic anaemia. Group III animals had neutrophilia and lymphopenia. Erythrocyte sedimentation rate was moderately high in Group III animals.

The serum creatine kinase and alanine amino transferase level were within the reference range for all the three groups. Group I animals with CHF had their serum sodium level high when compared with the control group while serum potassium level was increased in group III with renal disease. Total protein and albumin level, was low in group II with anaemia. Group III animals with renal disease had severe hypoalbuminaemia and low albumin: globulin ratio. Serum creatinine level was very high in group III animals with renal disease.

Conclusion

It was found that male geriatric small breeds of dogs were more affected with congestive heart failure. Chest leads were more sensitive than other leads in detecting hyperkalemia in renal diseases. Various leads including chest leads were standardized for the common breeds of dogs in Kerala of varying age groups which could serve as ready reference to the field practitioners.

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* Originals not seen

Appendices

ANNEXURE-I

Case No. Date:

Hospital

Owners name and address:

Patient data

- Colour
- Breed
- Sex
- Age
- Body weight

Owners complaint

History

- Past
- Present

Environment

- Indoor
- Outdoor

General clinical examination

- General appearance
- Behaviour
- Expression
- Bodily condition
- Condition of skin and coat
- Appearance of abdomen
- Posture
- Gait

Abnormal acts

External surface of the body

Respiration

- Rate
- Rhythm
- Character

Pulse

- Rate
- Rhythm
- Character

Jugular pulse

Temperature

Mucous membrane of the eye

Capillary refil time

System wise examination

Cardiac examination

- Physical examination
- Electrocardiogram findings (Annexure I)
- Radiographic findings

Respiratory system

Digestive system

Nervous system

Skin

Lymphatic system

Genitalia

Locomotor system

Laboratory examination

- Wet film and blood smear for blood parasites

Haematology

- RBC
- Hb
- PCV
- MCV
- MCH
- MCHC
- TLC
- DC N L M E B
- ESR

Serum biochemistry

CPK	Total protein
ALT	Albumin
Sodium	A/G ratio
Potassium	Creatinine

ANNEXURE – II

Electrocardiogram Report

Case No. Date:

Owners name and address

Heart rate

Rhythm

Parameters Lead II

- P-amplitude (mv)
- P-duration (sec)
- P-R interval (sec)
- QRS duration (sec)
- R-wave amplitude (mv)
- Q-T interval (sec)
- S-T segment
- T-amplitude (mv)

Other findings (including changes in other leads)

MEA

Report

ELECTROCARDIOGRAM ABNORMALITIES IN CARDIAC DISORDERS OF DOGS

By
RAVINDRAN. P.

ABSTRACT OF A THESIS

Submitted in partial fulfilment of the
requirement for the degree of

Master of Veterinary Science

Faculty of Veterinary and Animal Sciences
Kerala Agricultural University

Department of Clinical Medicine
COLLEGE OF VETERINARY AND ANIMAL SCIENCES
MANNUTHY, THRISSUR - 680651
KERALA, INDIA

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ABSTRACT

In the present study, standard electrocardiogram patterns were worked out from sixty normal dogs. These dogs were grouped according to age groups like dogs below one year and above one year and different breeds like German Shepherd, Dobermann pinscher, Dachshund, Spitz and Mongrel.

Based on the clinical signs and electrocardiogram abnormalities, thirteen dogs were grouped into (1) dogs with congestive heart failure, (2) dogs with ventricular enlargement secondary to anaemia and (3) dogs with electolyte imbalance due to renal disease.

Congestive heart failure was found to be more in geriatric small breeds like Dachshund and Spitz.

The clinical signs observed in dogs with CHF were anorexia, cough, exertional dyspnoea, exercise intolerance, orthopnoea, crackles, murmurs, syncope, cachexia and ascites.

Clinical signs exhibited by the dogs with ventricular enlargement secondary to anaemia were anorexia, pale visible mucous membranes, weakness and respiratory distress.

Dogs with electrolyte imbalance due to renal disease exhibited anorexia, vomiting, diarrhoea, malena, polydipsia, oliguria and anuria.

The electrocardiographic signs exhibited by dogs with congestive heart failure were consistent with left atrial enlargement, left and right ventricular enlargement, ventricular tachycardia and right bundle branch block. Dogs in group II had right and left ventricular enlargement as major electrocardiogram abnormality. Dogs in group III with renal disease exhibited tall and peaked 'T' wave and prolonged 'Q-T- interval in electrocardiogram specifically on the chest leads.

The prominent radiographic signs observed in dogs with CHF in the present study were left atrial enlargement, left and right ventricular enlargement and tracheal elevation. Dogs in group II showed left ventricular enlargement, pericardial effusion and tracheal elevation. Group III animals had no radiographic signs of cardiac enlargement.

Ultrasonographic evidence of cardiac involvement were present in three dogs, two with CHF and one with ventricular enlargement secondary to anaemia. The findings of postmortem examination of the heart in one dog with CHF were suggestive of mitral valvular insufficiency.

Haematology showed no variations in dogs with CHF. Group II and III animals exhibited anaemia evidenced by haematological changes like low TEC, Hb and PCV. Group III animals had neutrophilia with lymphopenia. Group III also had microcytic anaemia.

Serum values of CK and ALT were normal in all the three groups. Dogs with CHF showed elevated sodium level while group III with renal disease showed elevated potassium level. Group II animals had hypoproteinaemia and hypoalbuminaemia. Group III animals revealed severe hypoalbuminaemia, low albumin: globulin ratio and elevated serum creatinine.