PATHOLOGY OF CARDIO-PULMONARY DISORDERS IN CANINES

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Thesis submitted in partial fulfilment of the requirement for the degree of

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DECLARATION

I hereby declare that the thesis, entitled **"PATHOLOGY OF CARDIO-PULMONARY DISORDERS IN CANINES"** 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.

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CERTIFICATE

Certified that the thesis entitled ""**PATHOLOGY OF CARDIO-PULMONARY DISORDERS IN CANINES**" is a record of research work done independently by **Dr. Pradeep, M.**, 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.

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Pradeep, M.

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Introduction

1. INTRODUCTION

The lungs and heart can be considered as a dual organ which plays a vital role in maintaining the life process by supplying the tissues with oxygen and removing carbon-dioxide. They are structurally and functionally interdependent in their mechanism of action.

Though the heart is an organ with wonderful ability to withstand stress and strain imposed upon it and remarkable in its ability to accommodate itself to the changes in diseases, the early symptoms of the heart disease are either unnoticed or are so slight as to be disregarded. It is also a fact that many animals, which revealed gross lesions in the heart on post mortem examination, had not manifested any related symptoms. The lesions are usually encountered as a coincidence or along with other unassociated disease which caused the death.

Two of the most important symptoms of congestive heart failure, namely dyspnea and cyanosis are pulmonary in origin. Due to the imbalance of the two sides of the heart, increased output of the right side or diminished output of the left side results in congestion and dysfunction of the lungs. On the other hand, diffuse pulmonary lesions may throw an intolerable strain on the right side of the heart.

The lungs are vulnerable to injury due to the constant exposure to air which are likely to contain numerous microbes, particles, toxic gases etc. The lungs are also exposed to the haematogenous microbes, toxins and emboli. The lungs are also a common site of metastasis of neoplasms.

Extensive studies have been conducted focusing on the pathology of either the respiratory system or the cardiovascular system. But only few studies have been conducted correlating the pathological lesions of the pulmonary and the cardiovascular system.

A study of this nature can serve as an index of the environmental health prevalent in different geographical areas where the study is conducted, since the cardio-pulmonary system is directly exposed to the external environment.

Hence the present study was designed to reveal the nature of the cardio-pulmonary lesions, to help the clinicians in the understanding of the disease process affecting cardio-pulmonary system and also to monitor the changing environmental health status using dog as the animal model. The study is also intended to correlate the prevalence of cardiovascular disorders with age, breed and sex.

Review of Literature

2. REVIEW OF LITERATURE

2.1. INCIDENCE OF HEART AND LUNG LESIONS

Patterson (1971) reported an incidence of congenital cardiac defects as one percentage while Darke (1986) reported only 0.5 per cent in dogs.

Liu *et al.* (1986) in a retrospective study of 12,348 canine necropsies during the period of 1970 to1983, observed only 21 cases of atherosclerosis.

The incidence of hypertrophic cardiomyopathy was predominant in male dogs. German shepherd was the most frequently affected breed (Van Vleet and Ferrans, 1986).

Out of the 30 dogs studied by Rajendrasing (1988) aortic lesions were found in 18 dogs and coronary artery lesions in three. Coronary artery had only microscopic lesions characterised by intimal thickening and disrupted internal elastic laminae.

Stalis *et al.* (1995) conducted 1472 necropsies of dog and found 37 cases of endomyocarditis and 25 cases of left ventricular endocardial fibrosis. Out of 35 cases of endomyocarditis, 25 had interstitial pneumonia. Interstitial pneumonia was seen in seven out of 25 cases of thrombo embolism of left atrium, right ventricle and pulmonary artery.

Freeman et al. (1996) reported nine cases of idiopathic cardiomyopathy in Dalmatians in a period of five years.

Hahn *et al.* (1996) reported 40 neoplastic conditions affecting lungs in a study conducted on 216 dogs.

Calvert *et al.* (1997) reported predilection of dilated cardiomyopathy in males.

Braslasu (2000) observed Cardiomyopathies in 12.39 per cent of 355 dogs suffering from diverse cardiovascular diseases. Out of these 59.09per cent had hypertrophic cardiomyopathy and 40.09per cent had dilated cardiomyopathy. Males were affected more frequently (58.33%) than female. Cardiomyopathy was frequent in dogs aged more than 10 years (50.6%). Congenital cardiomyopathy was diagnosed at the age of one-year.

Vail (2000) reported that 80 per cent cases of lymphoma in dogs were of multicentric origin and were mostly of stage III or IV of WHO grading. Occurrence of mediastinal lymphoma was three per cent.

Aleksik-Kovasevic and Jelesijevic (2001) conducted morphological, histopathological and immunohistochemical study of malignant lymphoma in 11 dogs. According to histological classification they got six cases of poorly differentiated, four cases of intermediate and one case of well differentiated lymphomas; and by histochemical method they noted that eight cases were B cell predominant and three cases T cell predominant lymphomas.

Guarda *et al.* (2001) described gross and histopathological finding of primary and metastatic heart neoplasia and heart base tumor which included seven cases of haemangiosarcoma, four chemodactoma, four lymphosarcoma and three metastatic tumors like osteosarcoma, broncho-alveolar adeno carcinoma and sarcoma.

2.2. CARDIAC LESIONS

2.2.1. Cardiomyopathy

2.2.1.1. Hypertrophic Cardiomyopathy

Myocardial fiber disarray was cited as most consistent feature of hypertrophic cardiomyopathy in human (Maron, 1985).

Van Vleet and Ferrans (1986) reported myocytes disarray in the ventricular septum of 20 percentages of the dogs with hypertrophic cardiomyopathy. They also noticed that the hearts were grossly enlarged and showed ventricular hypertrophy with decreased left ventricular cavity size and left atrial dilatation and asymmetric septal hypertrophy (septal /free wall thickness ratio, >1.1) was often present.

In the obstructive form of hypertrophic cardiomyopathy, fibrous endocardial plaques developed on the septum directly opposite to thickened anterior mitral wall leaflet (Sisson and Thomas, 2000).

2.2.1.2. Dilated Cardiomyopathy

Focal degeneration of Bundle of His, and bone and cartilage in the central fibrous body were reported in Doberman Pinschers, by James and Drake (1968) which might have some pathogenic role in dilated cardiomyopathy by narrowing the lumen of the coronary arteries near the bundle of His.

Lesions of dilated cardiomyopathy were seen in the left ventricular free wall, left ventricular papillary muscle and interventricular septum (Calvert *et al.*, 1982).

Dilated cardiomyopathy was characterised by flabby and dilated heart and might show some degree of endocardial fibroelastosis. Mural thrombi were also common (Van Vleet and Ferrans, 1986).

Liu and Tilly (1980), Sandusky *et al.* (1984), Tidholm and Jonsson (1996), and Everett *et al.* (1999) considered wavy fibers as good indicators of dilated cardiomyopathy.

Tidholm *et al.* (1998) found that 98 per cent of the animals having the dilated cardiomyopathy showed attenuated wavy fibers and concluded that presence of wavy fibers was a sensitive index for dilated cardiomyopathy.

Everett *et al.* (1999) reported that in stained longitudinal (base to apex) section of the left ventricle, the lesion of the dilated cardiomyopathy was usually apparent to the unaided eye, appearing as a central linear pale zone. In his retrospective evaluation of dilated cardiomyopathy in 32 Doberman Pinschers revealed left ventricular free wall as a highly specific location for the characteristic myocardial lesions. The lesion involved more than 50 percentage of the ventricular wall. The specific myocardial lesions were characterised by myofiber degeneration and atrophy, and replacement of myocardial dense bundles of collagen and clusters of adepocytes.

2.2.1.3. Right Ventricular Cardiomyopathy

In a case of right ventricular cardiomyopathy reported by Bright and McEntee (1995) right ventricle and right atrium were greatly distended. The right atrium and right ventricular myocardium showed remarkable degree of fibro-adipose replacement with complete sparing of left ventricular myocardium. This kind of lesion established the diagnosis of isolated right ventricular cardiomyopathy and differentiated this order from idiopathic dilated cardiomyopathy and from chronic right ventricular overload.

Dilated right ventricular cardiomyopathy similar to human was reported in Boxer, Dachshund and Bullmastiff. (Sisson and Thomas, 2000)

2.2.2. Myocardial Degeneration

Rafiquzzaman *et al.* (1976) reported generalised cytoplasmic glycogen deposition in dogs with type III glycogenesis.

Basophilic degeneration was occasionally present in the myocardium of dogs with mitral endocardiosis and myocardial hypertrophy. The affected cells had a mass of perinuclear, Periodic acid Schiff (PAS) positive basophilic material. (Bishop, 1979).

2.2.3. Myocardial Infarction

2.2.3.1. Gross Pathology

Klinosky (1960) observed demonstrable degree of reduction of myocardial enzymes in the infracted area of the heart.

Nachlas and Shanitka (1963) demonstrated efficacy of Nitro- blue tetrazolium salt (Nitro-BT) in early detection of myocardial infarction, experimentally in dogs after coronary artery ligation. Nitro-BT a redox indicator gives dark blue formazan dye, staining the normal myocardium dark blue coloration leaving the infarcted area unstained. Carcasses stored at 4°C up to 24 hours gave reliable results.

Derias and Adams (1978) using Nitro-BT showed areas of focal diminution of staining as early as one hour after the suspected onset of infarction.

Shaper *et al.* (1979) measured the relationship of infarct sizes to area at risk using p - Nitro-BT and histological examination at 48 hours after coronary artery occlusion and demonstrated a close agreement between macrohistochemical and histological infarct size.

Dungworth (1993) noticed that pulmonary infarction was more common in the caudal lobes. The infarct was red blue to black in colour, firm in consistency and bulged out on the pleural aspect.

2.3.2.3. Mineralization and Ossification

Liu *et al.* (1969) reported pulmonary alveolar microlithiasis in a dog with ruptured chordae tendineae in the mitral and tricuspid valves.

Pulmonary ossification could be associated with mitral valve stenosis (Spencer, 1969).

Brix *et al.* (1994) reported a case of alveolar microlithiasis and ossification in a male English Setter dog. Grossly the lung was relatively rigid and had a grainy consistency on palpation. Decalcified histopathological section of the lung tissue contained many circular to irregular shaped, slightly basophilic, nonbirefrengent, Periodic acid Schiff-positive concretions within most alveolar walls or free within alveoli and variable conversion of microliths in to bone without any association of inflammatory cells.

Day *et al.* (1996) observed crystalline materials with in the lungs of 12 dogs. The crystals were always associated with aggregated black granular pigment (carbon) and were located adjacent to bronchioles and/or with in the intra alveolar interstitium. The crystals were small, needle like birefringent particles located with in the cytoplasm of macrophages or extracellularly.

2.3.2.4 Inflammatory Condition

Pulmonary edema or alveolitis were observed in dogs died of complicated septicemia (Turk *et al.* 1990).

Corcoran *et al.* (1992) reported a case of lipoid pneumonia in a rough collie dog.

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Klein *et al.* (1981) suggested that normal myocardium reduces tetrazolium salts to formazan pigment by the activity of membrane bound diaphorase that use NADH (NADPH) as electron donors.

According to Knight (1983) myocardium became paler, edematous, lost its normal wet lusture and matted after a lapse of 15 to 24 hours of infarction. It progressed to haemorrhagic streaks with yellowish background and gelatinous centre.

In contrast to the concept that right ventricle of the dogs were immune to infarction as they were having a well developed thebesian system, Francis *et al.* (1989) identified right ventricular infarction as an important clinical entity in association with myocardial infarction of the posterior free wall of the left ventricle as well as chronic lung diseases.

In a study conducted by Driehuys *et al.* (1998) on myocardial infarction, 25 dogs had infarction extending from the endocardium to 25 to 100 per cent of the myocardial thickness. Ninety-four per cent of dogs had infarcts of the left ventricle, and many dogs had involvement of the right ventricle and interventricular septum. Grossly areas of infarct ranged from well-circumscribed pale tan to red areas in the myocardium.

2.2.3.2 Histopathology

Ratliff *et al.* (1967) reported that in hypoxic cases of dogs sub-endocardial haemorrhage and necrosis were concentrated in the ventricular sub endocardium and were especially pronounced in the papillary muscles of both ventricles and in the middle of the ventricular septum.

Wavy fibers were a criterion for histological diagnosis of myocardial infarction and even a 24-hour autolysis did not affect it (Bouchardy and Majno, 1974).

Isner *et al.* (1982) contradicted that the presence of wavy fibers, an indicator of left ventricular infarction, as an unreliable indicator for acute right ventricular infarction.

Jones *et al.* (1985) reported that myocardial vacuolisation occur in infarct border zones and was severe in subendocardial region.

Francis *et al.* (1989) observed histopathologically, edema, extravasation of blood cells, hyper eosinophilia and a reduction or absence of PAS reaction with in 60 minutes of the right coronary artery occlusion. Purkinji cells retained glycogen deposit along the basilar region of the cells after three hours of right ventricular ischemia.

Discrete focal or multifocal areas of eosinophilic myocardial necrosis with or without inflammatory cells, along with oedema and loss of striations were reported by Driehuys *et al.* (1998)

2.2.4. Endocardial Lesions

2.2.4.1. Gross Pathology

Zook (1974) reported sporadic cases of endocardial fibroelastosis in dogs.

Liu and Tilley (1976) studied dysplasia of tricuspid valves in 14 dogs and the lesions observed macroscopically included long, thick septal leaflet adhered to the septum; short, stout and fused chordae tendineae; hypertrophic fused papillary muscles directly in to lateral leaflets; incomplete development of the valvular tissue; and enlargement of the right atrium and ventricle.

Olson and Miller (1986) reported epicardial, endocardial and myocardial haemorrhage in dogs infected with pseudorabies.

Takeda *et al.* (1991) reported blood cyst in the cardiac valves of dogs. He observed that the red blood cells were surrounded by fibrous tissue and cartilaginous metaplasia in the valve of chronic cases.

The endocardial fibroelastosis was characterised by left ventricular and atrial dilatation, with severe endocardial thickening (Sisson *et al.*, 2000).

2.2.4.2. Histopathology

Endocardial edema and dilated lymph vessels were seen in early stages of endocardial fibroelastosis suggesting the role of lymphatic obstruction in its pathogenesis (Miller et al., 1963).

The histopathological lesions found in the tricuspid dysplasia of the dogs were proliferation of the spongiosa, hypoelastification, and irregular arrangement of the fibrosa in the affected leaflet tissue. Endothelium-lined blood filled cysts were also observed in the valvular tissue (Liu and Tilley, 1976).

Sisson *et al.* (2000) observed histological lesion of endocardial fibroelastosis as diffuse intracellular fibro elastic thickening of endocardium with layering of thin, randomly organised collagen and elastic fibers.

2.2.5. Inflammatory Conditions of Heart

Scattered pale foci of myocardial necrosis were noted in puppies of five to seven days of age that were experimentally infected with Canine Distemper virus but not in those which were infected at 10- 21 days of age (Higgins *et al.*, 1981).

Experimental intrauterine infection of puppies during the second trimester of pregnancy with canine herpes virus resulted in foetal and prenatal death showing focal necrotizing myocarditis with inclusion bodies (Hashimoto *et al.*, 1983).

Necropsy of dogs done by Lenghaus and Studdert (1984) with parvo viral infection revealed lesions of acute congestive heart failure with pulmonary edema.

Wright and Heard (1979) reported that primary pulmonary hypertension could cause pulmonary congestion.

In dogs with chronic bronchitis emphysematous changes were usually confined to edges of pulmonary lobes (Anderson, 1987).

Anderson *et al.* (1989) reported multifocal bullous emphysema with bronchial hyperplasia in dogs.

Asymmetry of pulmonary infiltration with predominantly right side involvement was reported by Kerr (1989) in animals with noncardiogenic pulmonary edema resulted from upper airway obstruction. The cause for this was unknown.

Pulmonary edema characterised by patchy interstitial edema, intra alveolar haemorrhage and inflammatory cell infiltration were reported in acute pancreatitis (Zhou *et al.*, 1992).

Edema due to permeability defect was found to be more acidophilic than cardiogenic edema and frequently contained strands or clumps of fibrin (Dungworth, 1993).

The greatest degree of pulmonary involvement was noticed in the noncardiogenic pulmonary edema (NPE) caused by airway obstruction, followed by cranial trauma, seizures and then electric shock (Drobatz *et al.*, 1995).

2.3.2.2. Pulmonary Infarction

Pulmonary infarction was characterised by stasis of blood in the vessel, accumulation of blood in the bronchiole and alveoli losing their normal architecture (Runnels, 1956).

Dungworth (1993) noticed that pulmonary infarction was more common in the caudal lobes. The infarct was red blue to black in colour, firm in consistency and bulged out on the pleural aspect.

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Corcoran *et al.* (1992) reported a case of lipoid pneumonia in a rough collie dog.

Greene (1998) reported that the lesions of streptococcal pneumonia depended on the virulence of the organism. It varied from vascular leakage and edema and damage to type I and type II pneumocytes.

Diffuse interstitial pneumonia with giant cell formation was seen in canine distemper viral infection in dogs (Greene and Appel, 1998).

Study on rats by Clarke *et al.* (1999) revealed that inhaled urban air particles increase the susceptibility to pulmonary inflammation.

The studies conducted by Clarke (2000) suggested that concentrated air particles (CAPs) inhalation was associated with subtle alterations in pulmonary and systemic cells profiles and specific components of CAPs may be responsible for these biologic responses.

Histologically, exogenous lipid pneumonia was characterised by large extra cellular fat globule surrounded by phagocytic lipid laden macrophages and multinucleated giant cells; whereas with endogenous lipid pneumonia, all lipids confined intracellularly in foamy lipid laden macrophages that fill the alveoli (Jones *et al.* 2000).

2.3.2.5. Pneumoconiosis

Suzuki *et al.* (1972) in their study on hamster lungs with pulmonary asbestosis found that epithelial cells lost their original type I and type II cell structure and change in to form intermediate between epithelial cell and alveolar macrophages due to constant irritation.

Canfield *et al.* (1991) reported silicios pneumoconiosis in two dogs.

Hawkins (2000) reported that mineralization of airway or pleura occur in aged dogs of any breeds and have no correlation with the disease.

Results obtained from experiments on rats by Saldiva (2002) confirmed that short-term exposures to concentrated air particles induced a significant inflammatory reaction in rat lungs and the reaction was influenced by particle composition.

2.4. CARDIOPULMONARY LESIONS

Borst *et al.* (1956) showed that with in certain physiological limit, increased left atrial pressure may actually result in pulmonary capillary dilatation.

Torack (1958) listed the factors predisposing for the pulmonary infarction as (a) left ventricular failure, (b) collapse of the lung, (c) any condition which mechanically interfere with the oxygenation of alveolar capillary blood, such as bronchial obstruction or pneumonic consolidation, and (d) conditions of shock.

Spencer (1969) reported that nodular heterotropic ossification might occur within the lung alveoli in mitral stenosis, in organised inflammatory tissue, occasionally in the walls of a thoracic cavity and very rarely by a process of metaplasia within normal lung. In conditions like Hamman-Rich lung (Idiopathic chronic interstitial fibrosis), bronchial carcinoid, local pulmonary hamartoma and sclerosing angioma, ossification of lung could be noticed.

Cardiac disease, neoplasia, disseminated intravascular coagulation, sepsis and hyperadrenocortisism were the most commonly identified coexisting factors with pulmonary thromboembolism (La Reu and Murtaugh, 1990).

McCarten and Patton (1993) noted thrombosis of the pulmonary artery, right ventricular dilatation and hepatic congestion in the feline dirofilariasis.

Cardiac edema was not fatal if the cardiac insufficiency can be controlled, but pulmonary edema was often the cause of death from sudden cardiac decompensation.

(Robinson and Maxie, 1993). They also reported that pulmonary congestion was most commonly caused by left sided or bilateral cardiac failure.

A study conducted by Caideron-Garciduenas *et al.* (2001) in clinically healthy mongrel dogs revealed that air pollution cause both cardiac and pulmonary lesions. Cardiac lesions included, apoptotic myocytes, endothelial and immune effecter cells, degenerated mast cells associated with scattered pool of mononuclear cells in ventricles. Vascular changes included scattered polymorphonuclear leucocytes margination and microthrombi in capillaries, small venules and arterioles. Small vessels exhibited smooth muscle cell hyperplasia, and arteriolar blood vessel showed deposition of particulate matter in the media and adventitia. Lung showed patchy chronic mononuclear cell infiltrate along with macrophages loaded with particulate matter surrounding the bronchiolar walls and adjacent vascular structure. Bronchiolar epithelial and smooth muscle cell hyperplasia, peribronchiolar fibrosis, microthrombi and capillary and venule polymorphonuclear leucocytes margination were also seen.

Subarachnoid haemorrhage was found to cause pulmonary edema (Neurogenic pulmonary edema) and cardiac failure (Macmillan *et al.*, 2002).

2.5. VASCULAR LESIONS

Hamilton and Angevine (1946) reported that pulmonary emboli were more common in the right lobes than the left lobes.

Liebo *et al.* (1950) experimentally produced medial degeneration of pulmonary artery (in the pre-stenotic segment) in a puppy by artificially stenosing the pulmonary artery.

MacLeod and Grant (1954) studied the distribution of the pulmonary emboli in man and animals and found that the majority of them lodge in the lower lobes particularly in the posterior basal and apical segments of the lower lobes.

Spencer (1969) reported that exaggeration of bronchopulmonary anastomosis was known to occur in several pathological states affecting the lung like acute bronchiolitis and pulmonary hypertension.

Jones *et al.* (1984) observed that the proliferation of muscle in the media of small arteries along with vessel occlusion was due to increased vascular resistance.

According to Chazova *et al.* (1995) the most frequent changes in the primary pulmonary hypertension (PPH) were concentric and eccentric intimal thickening, medial hypertrophy, appearance of muscle in smaller and most peripheral arteries more than normal, arterial obliteration and recanalisation.

Lesions in pulmonary artery reported by Karlstam *et al.* (2000) in King Charles Spaniel were moderate to severe intimal thickening due to slight sub endothelial fibrosis, and abundance of vacuolated, Alcian bluer-PAS positive intercellular substances compatible with glycosminoglycans (GAGs).

Wu *et al.* (2002) studied the effect of acute multiple pulmonary thromboembolism in canines and found it to cause lung injury including pulmonary haemorrhage, consolidation and microembolism.

2.6. LYMPH NODE LESIONS

Pearson *et al.* (1986) published first report of a granulomatous lymphadenopathy in two dogs associated with alumino silicate minerals.

In a study conducted by Day *et al.* (1996) on lymph nodes in canines showed reddish brown coloration and diffuse congestion on cases of lymphadenopathy with birefringent materials inside. Cortico-medullary differentiation was apparent in majority of the lymph nodes. The most common microscopic feature of the lymph nodes with mineral deposition was the presence of large numbers of macrophages with in the medullary sinuses or, less frequently, in medullary cords. In all nodes

these macrophages had extensive cytoplasm that was often vacuolated and contained variable quantities of intracytoplasmic crystalline materials.

2.7. NEOPLASIA

2.7.1. Heart

Sanford *et al.* (1984) reported primary cardiac granular cell tumor in a dog histopathologically showing areas of spindle shaped cells and sheet of globoid cells with foamy granular cytoplasm.

Mesfen (1990) reported spontaneous epicardial fibrous fronds on the atria of a Beagle dog. This was seen as an irregular nodule on the base of the left atrium.

McDonough *et al.* (1992) observed a case of pericardial mesothelioma in dog. Neoplastic nodules were also present in the lung and tracheobronchial lymph node.

Mansfield *et al.* (2000) reported intra atrial rhabdomyoma in a bull terrier dog having a size of three-centimetre diameter.

2.7.2. Lung

Lungs were the most common sites for metastasis in osteosarcoma. The tumor was most commonly seen in large breeds of dogs than in the smaller breeds (Theilen and Madewell, 1987).

Leblanc *et al.* (1990) reported lymphomatoid granulomatosis in the lung and lymph nodes of a Beagle dog. The accessory lobe was enlarged firm and pale yellow. The cut section revealed coalescing multinodular pale tissue centered on the bronchi. Sheets of dense infiltrates of pleomorphic lymphoid cells that obliterated normal structures were noticed histologically.

Schulman *et al.* (1992) reported three cases of intracranial meningioma with pulmonary metastasis. The tumor in the lung was seen macroscopically as multiple nodules and histopathologically as meningiomatous meningioma.

Weller *et al.* (1992) reported primary pulmonary chondrosarcoma in a Beagle dog.

Hahn *et al.* (1996) reported 40 neoplastic conditions of dogs affecting lung. Thirty five neoplasms were carcinomas, classified as papillary adenocarcinoma (20), bronchio alveolar carcinoma (9), adeno squamous carcinoma (5) and bronchial gland carcinoma (1). The other five neoplasms were a fibrinohistiocytoma, three cases of adenoma and a fibroma.

The lungs and the lymph nodes were the most common sites for tumor metastasis (Avadhani, 1998).

Takhashi *et al.* (2000) reported a two centimetres size pulmonary hamartoma beneath the pleura without any connection to tracheo-bronchial tree in a dog.

Materials and Methods

3. MATERIALS AND METHODS

The present study was conducted utilizing, the data maintained during the period 1997-2002 and the carcasses of dog brought for autopsy at the Centre of Excellence in Pathology, College of veterinary and animal sciences, Mannuthy, to investigate the prevalence and pathology of cardio-pulmonary disorders of canines and to classify the lesions encountered in these organs.

3.1. PREVALENCE STUDY

A retrospective study for the period of five years (1997 to 2002) was conducted to find out the prevalence of cardio-pulmonary lesions by scanning the autopsy records, maintained at the centre. The available data pertaining to the post mortem diagnosis and the gross pathological lesions of the heart and lungs were collected and analysed.

3.2. SAMPLE COLLECTION

Seventy eight samples of heart, lung and lung associated lymph nodes collected from the carcasses brought for autopsy at the centre formed the materials for the study. Animals of all age groups, were utilised for the study.

3.3. GROSS EXAMINATION

Detailed post mortem examination of the dogs that were brought for autopsy at the Centre of Excellence in Pathology was conducted. Heart and lungs were removed from the carcass as single piece and examined for pathological lesions and were recorded. The pericardium was removed to detect abnormal fluid accumulation and for other changes on epicardium if any. Size and shape of the heart were noted. The heart was examined for hypertrophy and dilatation. Heart was then dissected from the apex to base through both ventricle and atrium and checked for internal lesions. Incisions were made through pulmonary artery

and pulmonary vein from the respective atrium to see thrombus formation if any. The hearts suspected for myocardial infarction were grossly stained with Nitro-Blue tetrazolium chloride solution as described by Nachlas and Shanitka (1963).

Lung was then separated from the heart. The pleural surface and borders of the lungs were examined for changes in lusture, color, consistency and any adhesion. The size and color of the bronchial and mediastinal lymph nodes were noted. The bronchi were opened and the contents of the lumen were examined for color and nature of the exudate. Each lobe of the lungs were examined separately for changes on the surface and the parenchyma, starting from the apical lobe and proceeds in the order of cardiac and diaphragmatic lobe. The lobes were palpated in order to detect the changes in consistency.

3.4. HISTOPATHOLOGY

Heart, lung and lymph nodes were cut into small pieces for histopathological examination. Pieces of heart included longitudinal section of left ventricular free wall with papillary muscle and left atrium; right ventricular free wall with right atrium; and cross section of heart that included ventricular septum and parts of ventricular free walls. The representative parts of lungs, showing gross pathological changes, and lung-associated lymph nodes were collected. All the tissue pieces were fixed in ten per cent formalin for the histopathological examination and were processed by routine paraffin embedding technique (Sheehans and Hrapachak, 1980). The paraffin embedded tissues were cut at three micron thickness and stained with routine Haematoxylin and Eosin stain (Bancroft and Cook, 1995). Histopathological lesions of these organs were recorded and classified. Special stains like Mallory's Phosphotungstic acid haematoxylin, Gomori's Trichrome, Alizarin Red and Periodic acid Schiff (Luna, 1968) were used wherever necessary.

Gross and histopathological lesions were correlated with that of breed, sex, age; and prevalence of the lesions were assessed.

Results

4. RESULTS

4.1. RETROSPECTIVE STUDY

A total of 746 carcasses were autopsied during the period 1997 to 2002. Cardiac lesions were detected grossly in 47.05 per cent (351) cases and pulmonary lesions in 83.38 per cent (622) cases. The lesions were present in both the lung and heart in 42.76per cent (319) cases.

4.1.1. Pulmonary Lesions

Pulmonary congestion was seen in 66 per cent cases and pulmonary edema in 25.88 per cent cases. Pulmonary collapse was seen in 11.25 per cent, pulmonary emphysema in 4.50 per cent and pulmonary haemorrhage in 8.84 per cent cases. Pneumonia was recorded in 13.67 per cent cases. Miscellaneous conditions like anthracosis (0.64%), abscess (0.64%), tumor (0.16%) were also noticed.

4.1.2. Cardiac Lesions

Among the cardiac lesions, the most commonly found lesion was right ventricular dilatation (39.88%). Left ventricular hypertrophy was seen in 17 per cent while right ventricular dilatation with left ventricular hypertrophy seen in 11.39 per cent cases. Right ventricular dilatation alone without any gross abnormality of the left ventricle was detected in 15 per cent cases. Biventricular dilatation was found in 13.68 per cent.

Petechial haemorrhage was noticed on the epicardium in 11.78 per cent cases. Epicardial vessels were congested in 13.68 per cent cases. Atrio-ventricular valves showed thickening in 5.13 per cent, haemorrhage in 6.21 per cent and endocardial haemorrhage in 20.51 per cent cases.

Pericarditis, hydro pericardium and haemopericardium were found in 1.14 per cent, 2.56 per cent and 1.71 per cent cases respectively. Congestion of pericardial blood vessels was seen in 0.28 per cent while petechiae were seen in 1.42 per cent cases.

Myocardial degeneration was observed in 2.56 per cent cases.

4.2 DISORDERS OF THE HEART AND LUNGS

In this study, 61 cases were males and 17 cases females. The age wise distribution was below one-year age (21 cases), between the age group of one and five (31) and above five-year of age (26). Breeds studied were non-descript (35 cases), German Shepard (26) and Doberman (7). Other breeds included were very few in number and they include Pomeranian (4), Great Dane (3), Cocker Spaniel (1), Boxer (1) and Labrador (1)

The age-wise incidence of cardiac and pulmonary lesions is illustrated in Fig. 1. The sex-wise incidence of cardiac and pulmonary lesions is shown in Fig. 2. The breed- wise incidence of cardiac and pulmonary lesions is presented in Fig. 3.

4.2.1. Gross Pathology

The post mortem examination revealed lesions of varying intensity and nature, both in the lungs and heart of almost all the carcasses. Gross pulmonary lesions without the involvement of the heart; and cardiac lesions without the involvement of the lungs were also observed.

4.2.1.1. Pulmonary Lesions

The pulmonary lesions were evident in 77 cases. The lesions were mainly located in the right cranial lobe (97.44%). The involvement of the right middle lobe and right caudal lobes were 94.87 per cent and 92.41 per cent respectively. The

accessory lobe was also involved in 92.41 per cent of cases. The least involved was the left cranial lobe (79.49%)

The main pulmonary lesions observed in different lung lobes are given in Table 1.

Bronchi and bronchioles revealed the presence of contents ranging from frothy to sanguineous fluid along with congestive changes of the bronchial mucosa (31.17%).

4.2.1.1.1. Pulmonary congestion

Pulmonary congestion of mild to severe nature was evident in 70.51 per cent. The congestion was marked in the accessory lobe. Focal or diffuse congestion affecting all the lobes was noticed in 36.36 per cent cases. The congestive changes were severe in 27.78 per cent, moderate in 38.39 per cent and mild in 33.33 per cent of cases.

4.2.1.1.2. Pulmonary haemorrhage

Pulmonary haemorrhage was noticed in 32.47 per cent of cases. The haemorrhage varied from petechiae to suffusion. Petechiae distributed in all the lobes of lungs were seen in 44 per cent. The haemorrhage was more frequent in the right cranial lobe (88%) and the left caudal lobe was the least affected (44%).

4.2.1.1.3. Pulmonary emphysema

Emphysematous changes were found to coexist along with other changes in 49.35 per cent of cases. The emphysematous areas appeared pale and distended. In 92.2 per cent cases the lesions were restricted to the borders of the lung lobes. Emphysematous changes were more prominent in the right cranial lobes (81.57%).

The degree of involvement by parenchyma in the cranial lobes was more and least in the caudal lobe.

4.2.1.1.4. Pulmonary edema

In 31.17 per cent cases pulmonary edema was evident. The lungs appeared wet and heavy (Fig. 4). Blood tinged or frothy fluid oozed out from the cut surface.

4.2.1.1.5. Pulmonary collapse

A patchy depressed area with dark colour and firm consistency were the gross features observed in 7.79 per cent. The lesions were mostly seen in the right middle and caudal lobes in about 83.33 per cent of cases. The left lobes were relatively less affected. The collapse was also noticed grossly in close proximity to the tumor lesions.

4.2.1.1.6. Pneumonia

The consolidated appearance of lungs were detected in eight cases. The lesion was more frequent in right lobes. Middle lobe lesions were found to be more severe and were mostly lobar type. Grey hepatisation was noticed in the right middle lobe in one case

4.2.1.1.7. Pleural lesions

Pleural adhesion of the lung lobes was seen in two cases. In one case adhesion was between the caudal part of left cranial lobe and the caudal lobe (Fig.5) and in the other case all the left lung lobes were fused together.

4.2.1.1.8 Mineralization and ossification

In a case showing hydrothorax and ascites multiple white hard masses of one to three millimetre diameters were seen grossly in the collapsed right and left middle lobes (Fig.6).

Sub-pleural gritty, translucent crystals distributed in all the lobes of the lung were observed in three cases. The crystals measured one to two millimetres in diameter. The grits were detected in dogs of above one year of age (Fig.1).

4.2.1.1.9 Pneumoconiosis

The surface of the lungs from the older age groups showed pinhead sized blackish discoloration affecting all the lobes of the lungs suggestive of anthracosis (16.67 %). The lesion was more frequent in above five year of age (Fig.1). Non-descript breeds showed higher incidence when compared to other breeds (Fig.3).

4.2.1.2. Cardiac Lesions

Congenital anomalies were not observed in any of the cases.

4.2.1.2.1. Pericardial lesions

Pericardium was found to be thickened and opaque with deposition of fibrin plaques in about 1.29 per cent cases of the heart affected cases. Moderate to severe degree of congestion of the pericardial vessels were noticed in 5.19 per cent of the cases. Hydro pericardium with clear to turbid pericardial fluid was observed in about 5.19 per cent (Fig.7).

One case of cardiac tamponade revealed the presence of current jelly clot filling the pericardial sac (Fig.8). A wound of approximately one centimetre diameter was observed on the pulmonary vein near its junction with the left atrium.

The wound was extending in to the atrium internally as a linear tear in the endocardium.

4.2.1.2.2. Epicardial and endocardial lesions

The gross pathological changes observed in the epicardium were mainly vascular changes like congestion (Fig.9) and haemorrhage. Degenerative and necrotic changes were also noticed as focal as well as diffuse, pale patches on the surface of the epicardium. Gelatinisation of epicardial fat was found in one case.

Petechial haemorrhagic streaks on epicardium were noticed in 11.69 per cent of cardiac lesions. Petechiae were more confined to the epicardial fat around the atrio-ventricular groove.

Endocardial haemorrhage was noticed in 25.97 per cent in the left ventricle (Fig.10), which was confined to the papillary muscle in most of the cases. Haemorrhage in the right ventricular endocardium was relatively less (6.49%).

Mitral valve showed moderate thickening in 5.19 per cent and diffuse haemorrhage in 9.09 per cent cases (Fig.11). Tricuspid valve showed mild thickening and diffuse haemorrhage in 2.59 per cent cases each.

A blood cyst of five millimetres diameter was detected in the mitral valve of a dog.

4.2.1.2.3. Cardiac dilatation and hypertrophy

Many cases showed enlargement of the heart due to dilatation along with hypertrophic changes. Right ventricular dilatation was seen in 75.32 per cent cases right ventricular dilatation along with left ventricular hypertrophy could be detected in 15.58 per cent. Rounding up of heart with the dilatation of both ventricles was noticed in 31.17 per cent. Dilatation of the right and left atrium was 12.99 per cent

and 7.79 per cent respectively. Male dogs showed comparatively higher incidence of cardiomyopathies (Fig.2) and the incidence was more in aged dogs (Fig.1).

4.2.1.2.4. Myocardial infarction

In two cases, the left ventricular free wall and inter ventricular septum showed well demarcated pale patchy area with cooked up appearance affecting nearly half of the left ventricular length from the apex (Fig.12).

On gross staining with Nitro-Blue tetrazolium chloride (Nitro-BT), unstained areas surrounded by blue stained myocardium in the left ventricular wall were observed in 5.19 per cent cases (Fig.13).

4.2.1.3. Neoplasia

In a Doberman Pinscher male dog, aged eight years, a soft tumor mass measuring about 10 centimeters in diameter was observed subcutaneously on the right side of the thoracic wall in the fifth intercostal space. On opening the carcass, partially necrotic large mass of tumor was seen extending into the thoracic cavity. The parietal pleura and diaphragm had small pedunculated nodules on its surface. Multiple nodules of size varying from one to four centimeters in diameter were seen in the visceral pleura extending into the lung parenchyma (Fig.14). Some nodules were hard in consistency while others were soft. Middle tracheobronchial lymph node was enlarged. On sectioning it revealed a white nodular mass. The heart revealed biventricular dilatation and was free of nodules.

In another female, Doberman Pinscher dog of two years of age multiple nodules of size varying from five to ten millimeters were detected in the heart (Fig.15) and lung parenchyma. In the lung only one nodule of size, one and half centimetre in the left caudal lobe was visible externally. Larger palpable nodules were present in right middle and caudal lobes and very small nodules were palpable

4.2.2.1.2. Pulmonary congestion

The hyperaemic changes observed grossly were evident as congestion of the muscular arteries as well as veins and septal capillaries. The engorgement of septal capillaries resulted in thickening of alveolar septa (Fig.20).

4.2.2.1.3. Pulmonary haemorrhage

In the cases of pulmonary haemorrhage extravasation of erythrocytes in to the alveolar space, interstitium as well as in to the sub-pleural tissues were noted.

4.2.2.1.4. Pulmonary edema

Pulmonary edema was present in 24 (31.17%) cases, which appeared histologically as homogenous pink staining fluid in the alveoli (Fig.21) as well as in the interstitium and in the bronchioles. The colour of the edema varied from pale to dark pink. Haemosiderosis was evident in a few cases.

4.2.2.1.5. Pulmonary collapse

The collapsed lung revealed the alveolar walls in apposition giving a cleft like appearance. In some of the cases the thickening of the alveolar wall along with dilatation of septal capillaries were also evident.

4.2.2.1.6. Pulmonary emphysema

Pulmonary emphysema was evident histologically as macro alveoli. Thinning as well as thickening of inter-alveolar septa were observed. Remnants of ruptured alveolar walls were also noticed (Fig.20).

4.2.2.1.7. Pulmonary infarction

Small patches of pulmonary infarction were noticed in two cases. The lumen of the alveoli was diffusely filled with erythrocytes along with necrosis and disappearance of the alveolar lining cells (Fig.22). The small bronchioles and blood vessels were also found to be damaged. Some of the blood vessels revealed thrombus formation.

4.2.2.1.8. Pneumonia

Histologically all the pneumonic lobes were infiltrated with inflammatory cells predominantly of mononuclear type. Bronchioles showed desquamation of epithelial cells, partially filling the lumen with debris and edema fluid and showed infiltration of inflammatory cells. Alveolar lumen was filled with proliferated type II pneumocytes, alveolar macrophages and fibrin materials (Fig.23). Alveolar space was lined with hyaline membrane in two cases.

Haemolytic streptococci were isolated from a pneumonic lung. Histological sections showed inflammatory cells predominantly of mononuclear type and proliferated type II pneumocytes and alveolar macrophages. Blood vessel wall and pleura were moderately thickened. Fibrous tissue hyperplasia was evident in sections stained with Gomori's trichrome (Fig. 24).

In another pneumonic case infiltration of mononuclear type inflammatory cells were recorded along with vacuolar degeneration of type II cells resulting in the formation of foamy cells (Fig.25).

4.2.2.1.9. Mineralization and ossification

Though the translucent, gritty materials were seen grossly in sub-pleural region, it could not be demonstrated histologically.

Multiple whitish masses observed grossly in the lung on histological examination revealed the presence of pink staining osseous tissue in the interstitial area (Fig.26). Mild to moderate degree of infiltration with inflammatory cells were seen around the osseous tissue giving a granulomatous appearance. The neighbouring alveoli showed rupture of inter alveolar septa and formation of macro alveoli. Out of the three cases of osseous metaplasia in lungs two cases were between the age group of one to five year and one case was above five years. The sections on staining with Alizarin red revealed red colour to the osseous tissue.

4.2.2.1.10. Pneumoconiosis

Histologically macrophages laden with anthracotic dust particle were present in sub-pleural region, peribronchiolar and perivascular area (Fig.27) and in the alveoli. Mild to moderate degree of fibrous tissue proliferation was evident. Pulmonary lymph nodes also revealed anthracotic particles (Fig.28).

4.2.2.1.11. Pulmonary vascular lesions

Thickening of the wall of the pulmonary arteries was noticed in 76.92 per cent cases. Endarteritis obliterans (Fig.29) and muscle fibre disarray were the other major lesions noticed in the pulmonary blood vessels. Pulmonary thrombi were noticed in four cases.

4.2.2.2. Cardiac lesions

4.2.2.2.1. Epicardial lesions

The pale areas observed grossly, on histological examination revealed degenerating and necrotic changes of the cardiac muscle fibres. Fibrous tissue proliferation and extensive areas of fat deposition in the sub-epicardial space extending into the myocardium were also observed.

4.2.2.2.2. Myocardial lesions

Myocardium of dilated heart showed widely separated thin muscle fibres. Attenuated wavy fibres were observed in 61.03 per cent cases of dilated hearts (Fig.30). In cases of cardiac hypertrophy the muscle fibres were swollen and crowded. The myocardial fibres revealed increase in thickness, enlargement of the nuclei. Intermuscular fibrosis was seen in 31.17 per cent cases (Fig.31). Twenty three per cent cases revealed haemorrhage in the myocardium. Large amount of extravasated red blood cells were seen in between widely separated muscle fibres with damaged collateral vessels (Fig.32) in a few cases.

Fragmentation and vacuolation (Fig.33) of the muscle fibres were also noticed. Infiltration of inflammatory cells mainly mononuclear type was noticed in one case (Fig.34).

Cartilaginous metaplasia at the tip of the papillary muscle was noticed in one case.

4.2.2.2.3. Myocardial infarction

Histologically infarcted myocytes showed hyper eosinophilia, pyknosis, karyolysis, loss of striations and vacuolation of myocardial fibres. The Nitro-B'T stained tissues on histopathological examination revealed normal fibres with blue colored formazan deposit and infarcted areas without the deposit (Fig.35).

4.2.2.2.3. Endocardial lesions

Thickening of the endocardium by fibrous tissue proliferation was observed in 12 cases. Endocardial fibro-elastosis characterised by extensive proliferation of the fibrous tissue in the endocardium extending into the myocardium (Fig.36) was evident in some cases. Valvular endocarditis (Fig.37) and mural thrombus (Fig.38) were observed in one case each.

Histologically the blood cyst contained large number of red blood cells and fibrous tissue was seen around the cyst (Fig.39). The valve showed cartilaginous metaplasia and congested blood vessels.

Cartilaginous metaplasia of mitral valve at the site of attachment of chordae tendineae was observed in one case (Fig.40).

4.2.2.2.4. Vascular lesions

Thickening of coronary vessels with or without narrowing of the lumen were noticed in several cases. Thickening was caused either by intimal layer proliferation or by medial layer hyperplasia. Special stain like Phosphotunstic acid haematoxylin was employed to differentiate the thickened layer (Fig.41). Vacuolar changes in the wall of the vessels were also found in some cases.

4.2.2.3. Neoplasia

4.2.2.3.1. Osteosarcoma

The soft tumor tissues in the lung were made up of large number of spindle shaped neoplastic cells along with multinucleated giant cells resembling osteoclasts (Fig.42). The hard tumor tissues on the lung and in the thoracic cavity, histologically revealed presence of malignant spindle shaped cells surrounded by poorly formed osteoid tissue (Fig.43). The tumor was diagnosed histologically as osteosarcoma.

The other lesions observed were collapse of the bronchiole (Fig.47) and alveoli adjacent to the tumor mass. The sub pleural pulmonary arterioles were obliterated by intimal layer proliferation (Fig.29). The pleura adjacent to the tumor tissue showed vacuolar changes (Fig.44).

Middle tracheobronchial lymph node revealed the presence of tumor cells beneath the capsule (Fig.45).

4.2.2.3.2. Lymphosarcoma

The tumor mass revealed the presence of large number of lymphoblasts. Pleomorphic type of lymphoblastic cells infiltrated both lung parenchyma and in between the cardiac muscle fibres (Fig.46). The cardiac muscle fibres showed fragmentations. The cells observed were of mixed type. Some cells of intermediate differentiation and plasmacytoid differentiation were observed.

Pulmonary lymph nodes showed diffuse lymphoid cells predominantly of lymphoblasts and lymphocytes of plasmacytoid differentiation.

4.3. CARDIO-PULMONARY LESIONS

Out of the 78 cases studied 26.92 per cent only showed primary lesions of the cardio-pulmonary system. 73.08 per cent lesions of the lung and heart were secondary to other lesions like nephritis, hepatitis, pyometra, metastatic tumor conditions and traumatic injuries.

The cardiac lesions like dilatations and hypertrophy were consistently associated with pulmonary involvement.

Cases in which left ventricular hypertrophy and right ventricular dilatation were detected showed congestion of all the lung lobes.

The association of pulmonary congestion, edema and haemorrhage with the cardiac lesion is given in Table 2. Left ventricular hypertrophy along with the right ventricular dilatation and biventricular dilatation were the most frequently seen cardiac lesions with the pulmonary congestion and edema.

The cardiac lesions in pneumonic cases are given in Table 3. Bilateral dilatation was the frequently encountered cardiac lesion with an incidence of 50 per cent of pneumonic cases.

Table 1. Distribution of lesions in different lobes of lungs

Pulmonary lesion	Right cranial	Right middle	Right caudal	Accessory	Left cranial		Left caudal
					Cranial part	Caudal part	
Congestion (54 nos.)	32 (59.26)	36 (66.67)	36 (66.67)	41 (75.93)	32 (59.26)	31 (57.41)	36 (66.67)
Haemorrhage (25 nos.)	22 (88)	18 (72)	18 (72)	14 (56)	16 (64)	12 (48)	11 (44)
Emphysema (38 nos.)	31 (81.58)	19 (50)	16 (42.11)	14 (36.84)	17 (44.74)	13 (34.21)	10 (26.32)
Collapse (6 nos.)	3 (50)	5 (83.33)	5 (83.33)	2 (33.33)	2 (33.33)	2 (33.33)	2 (33.33)
Pneumonia (8 nos.)	1 (12.50)	4 (50)	4 (50)	1 (12.5)	1 (12.5)	2 (25)	3 (37.5)
Anthracosis (13 nos.)	13 (100)	13 (100)	13 (100)	13 (100)	13 (100)	13 (100)	13 (100)

Values given in parenthesis shows percentage

Table 2. Lung lesions in cardiomyopathies

Lung lesions	Congestion	Haemorrhage	Edema
LVH&RVD (12cases)	8(66.67)	2(16.67)	5(41.67)
BD (24 cases)	16(66.67)	5(20.83)	10(41.67)
RVD (22 cases)	11(50)	8(36.36)	4(18.18)
LVH (2 cases)	2(100)	1(50)	0

Table 3. Cardiac lesions in pneumonia

Cardiac lesions	LVH &RVD	BD	RVD	LVH	EC	MI	E/V.H
Pneumonia (8 cases)	0	4(50)	1(12.5)	1(12.5)	3(37.5)	1(12.5)	3(37.5)

Values given in parenthesis shows percentage

LVH &RVD-left ventricular hypertrophy and right ventricular dilatation
BD-biventricular dilatation
RVD- right ventricular dilatation
LVH-left ventricular hypertrophy
EC-epicardial congestion
MI-myocardial infarction
E/V.H-endocardial or valvular haemorrhage

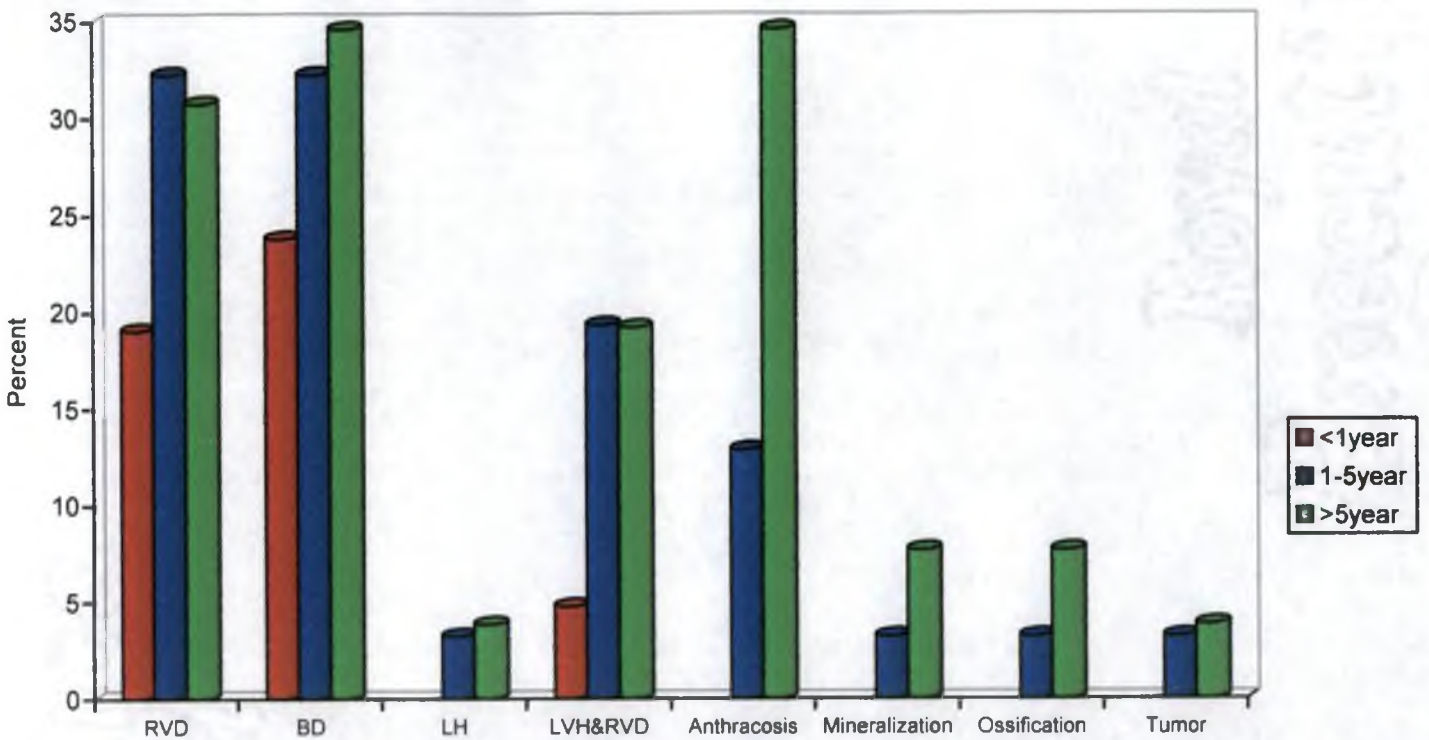


Fig. 1. Age-wise incidence of cardiac and pulmonary lesions

R.V.D. – Right ventricular dilatation		BD	– Biventricular dilatation
L.H	– Left ventricular hypertrophy	LVH&RVD	– Left ventricular hypertrophy and Right ventricular dilatation

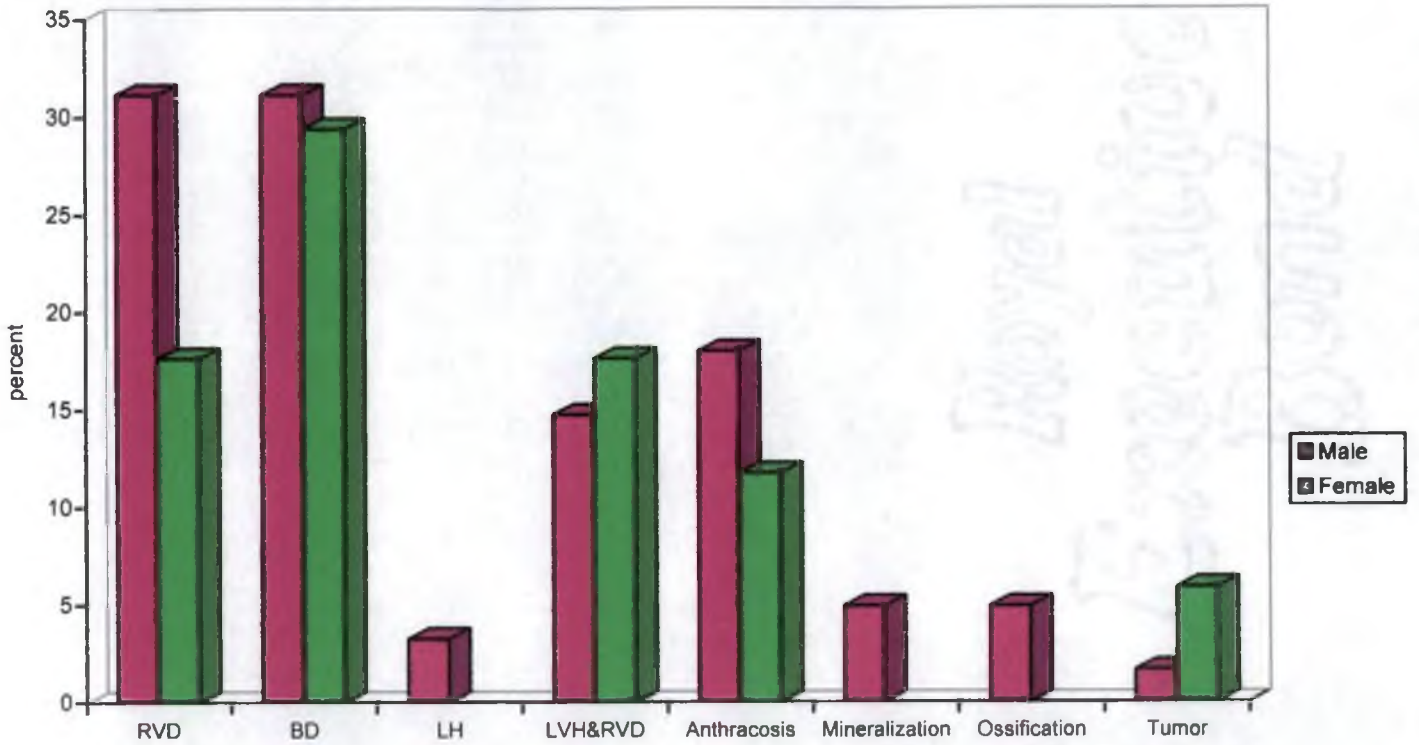


Fig. 2. Sex-wise incidence of cardiac and pulmonary lesions

R.V.D.	– Right ventricular dilatation	BD	– Biventricular dilatation
L.H	– Left ventricular hypertrophy	LVH&RVD	– Left ventricular hypertrophy and Right ventricular dilatation

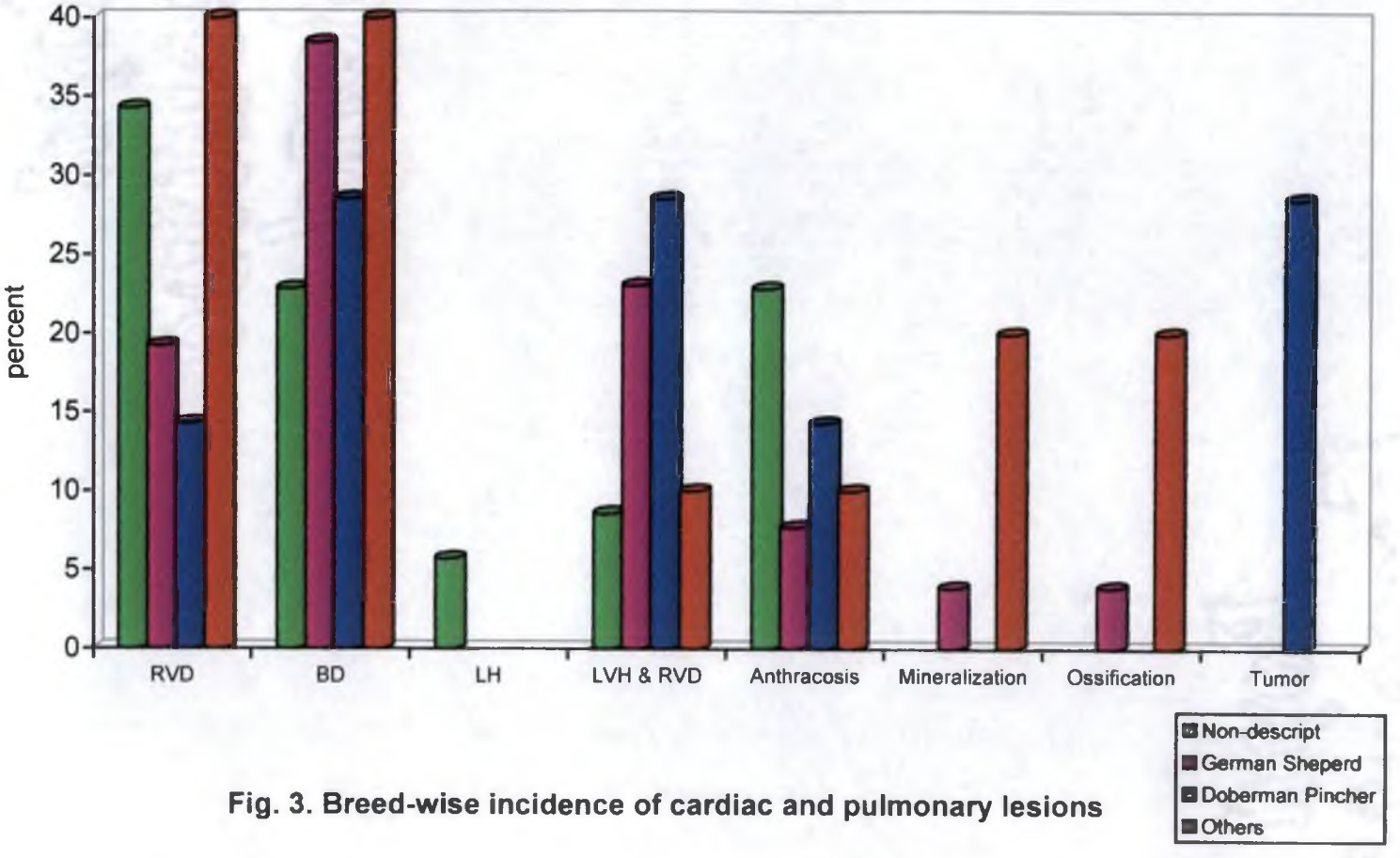


Fig. 3. Breed-wise incidence of cardiac and pulmonary lesions

R.V.D.	– Right ventricular dilatation	BD	– Biventricular dilatation
L.H	– Left ventricular hypertrophy	LVH&RVD	– Left ventricular hypertrophy and Right ventricular dilatation

Fig.4. Oedamatous lung

Fig. 5 Pleural adhesion between left caudal part of cranial lobe and the caudal lobe

Fig.6 White hard osseous tissues in the collapsed middle lobes of the lung

Fig.7 Hydropericardium with engorged blood vessels

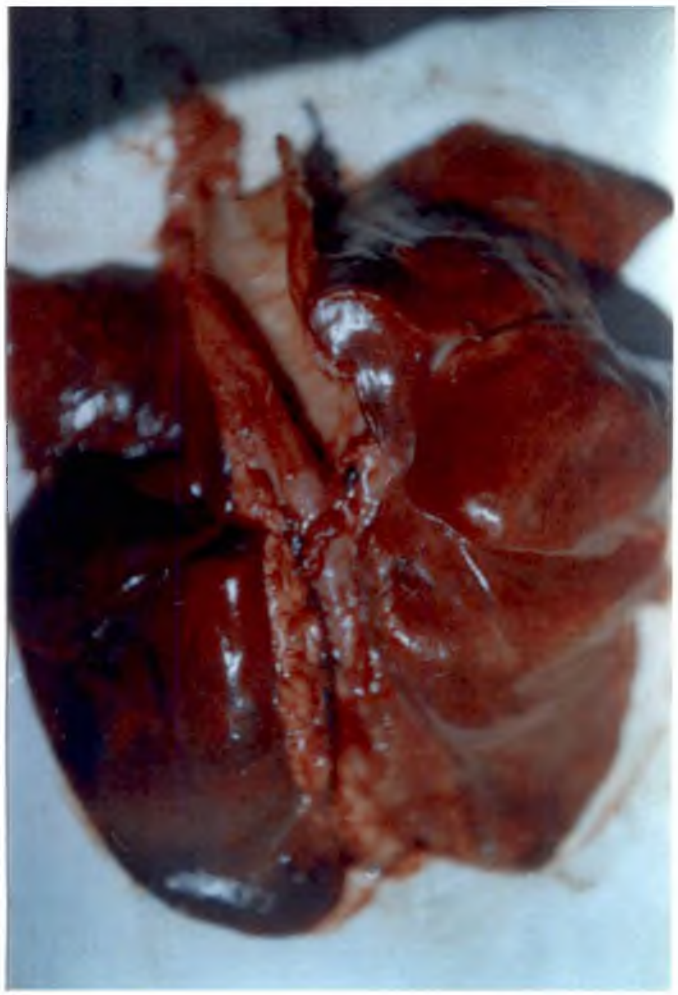
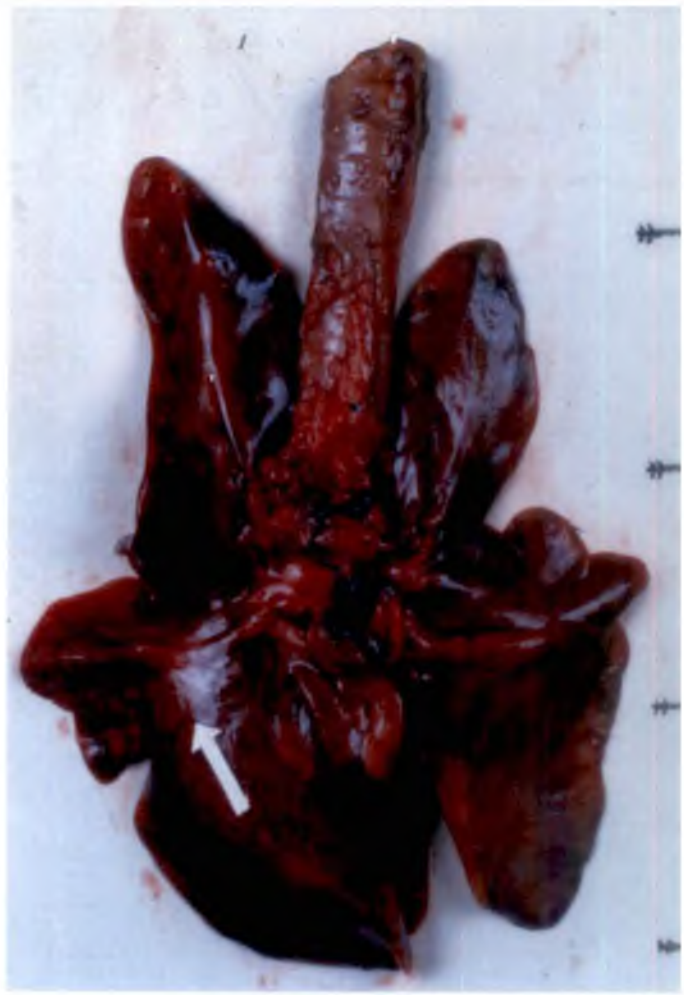


Fig. 4.



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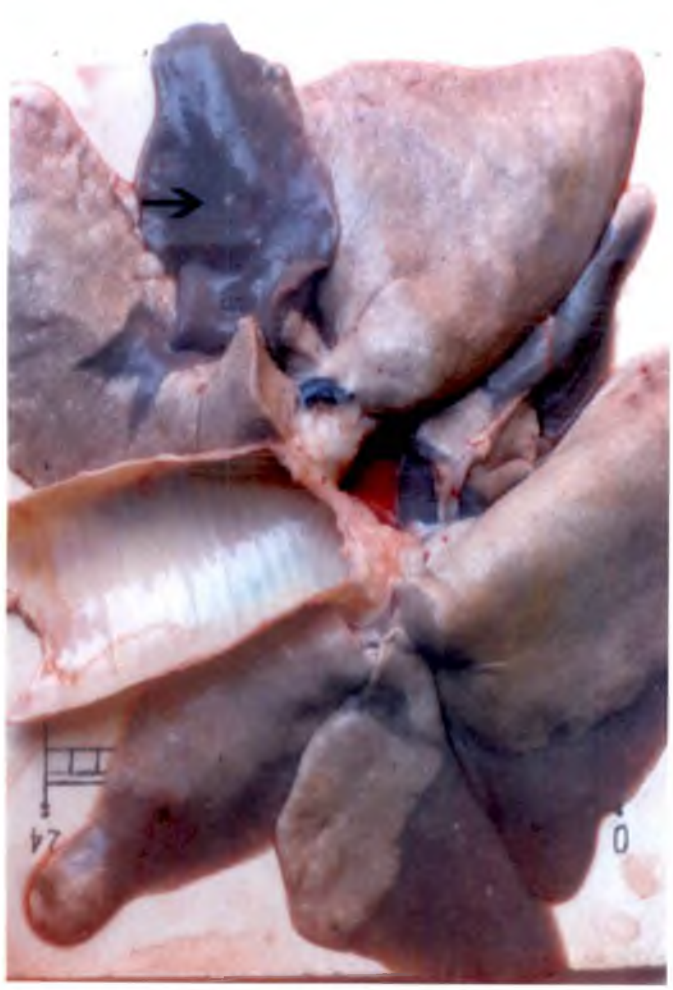


Fig. 6

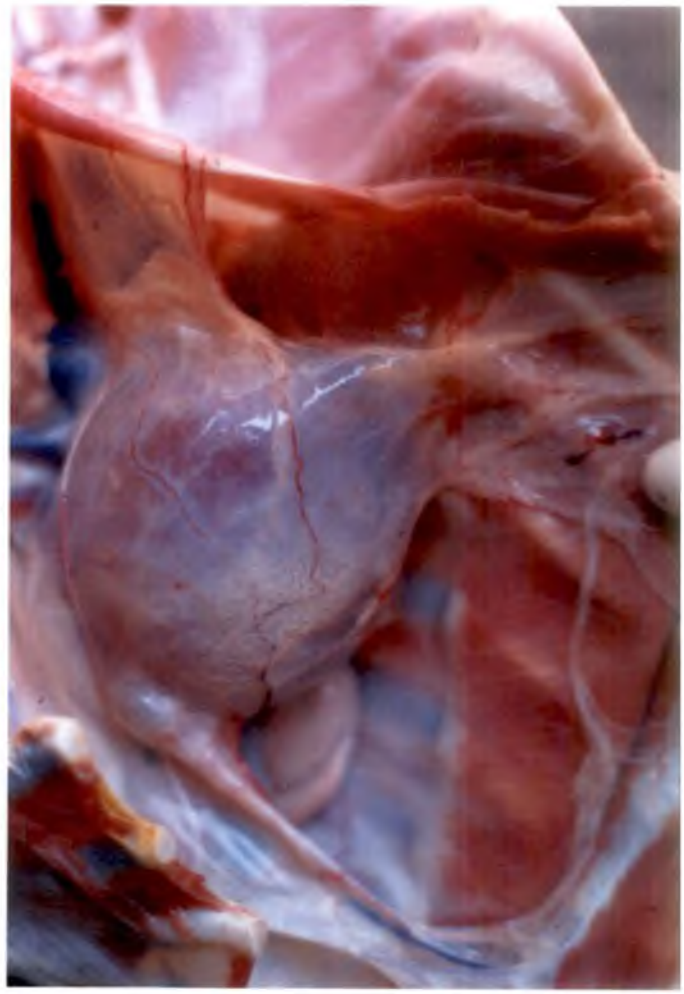


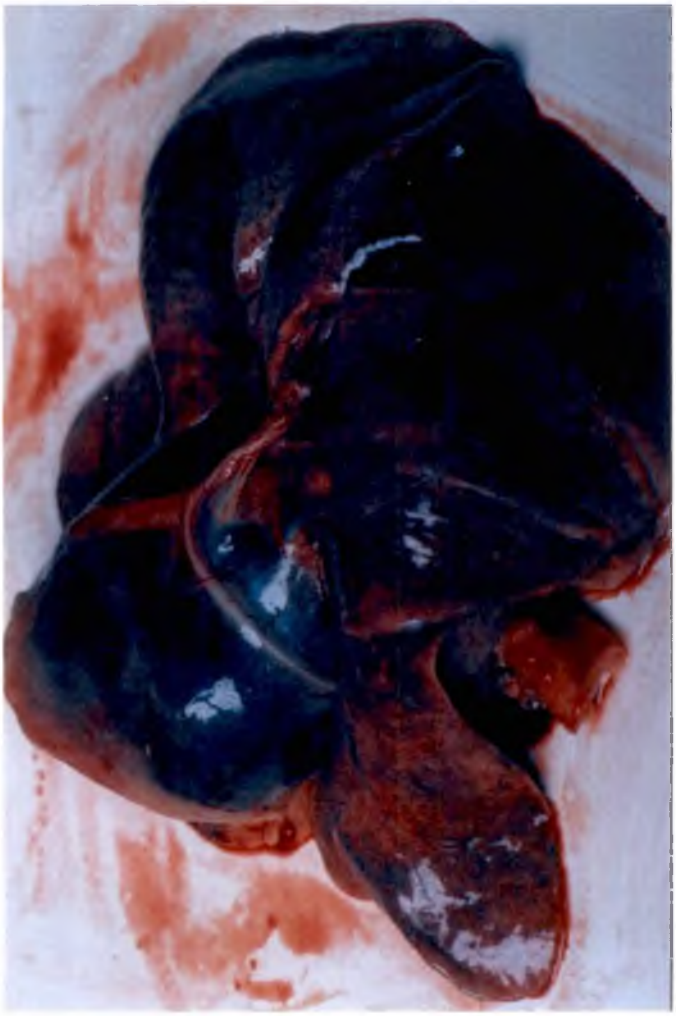
Fig. 7.

Fig.8 Cardiac tamponade

Fig.9 Cardiac dilatation with engorged epicardial vessels

Fig.10 Endocardial haemorrhage

Fig.11 Ecchymosis on mitral valve



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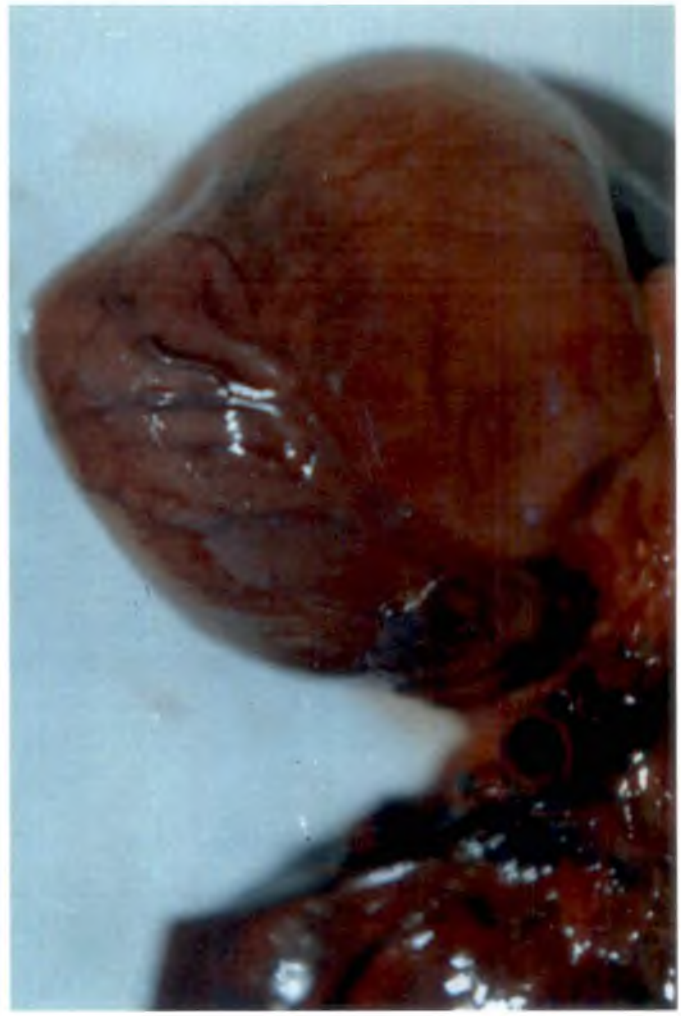

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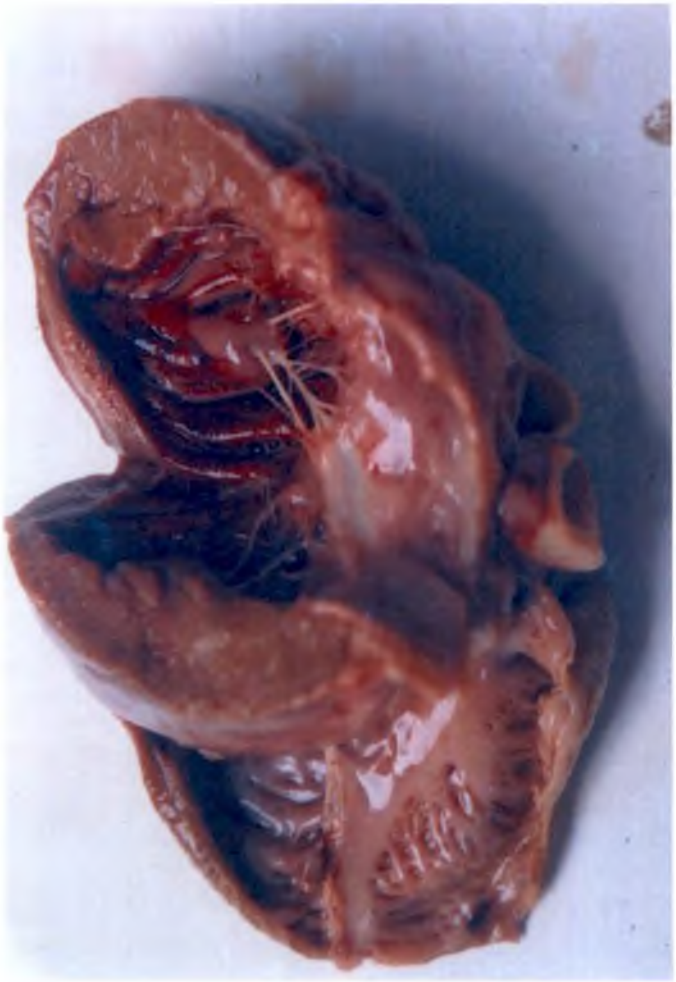


Fig. 10.

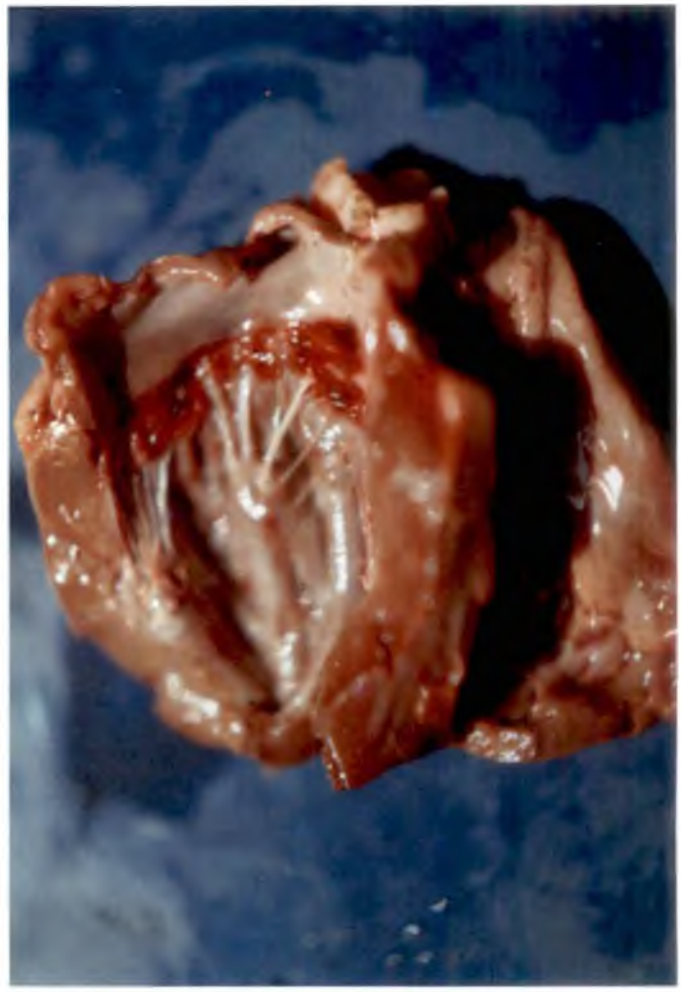


Fig. 11.

Fig.12 Infarcted pale areas in the left ventricular myocardium

Fig.13 Myocardial infarction grossly stained with Nitro-BT. Normal areas are blue coloured and infarcted area unstained

Fig.14 Multiple nodules in the lung- Osteosarcoma

Fig.15 Greyish white nodules in the heart and lung- Lymphosarcoma

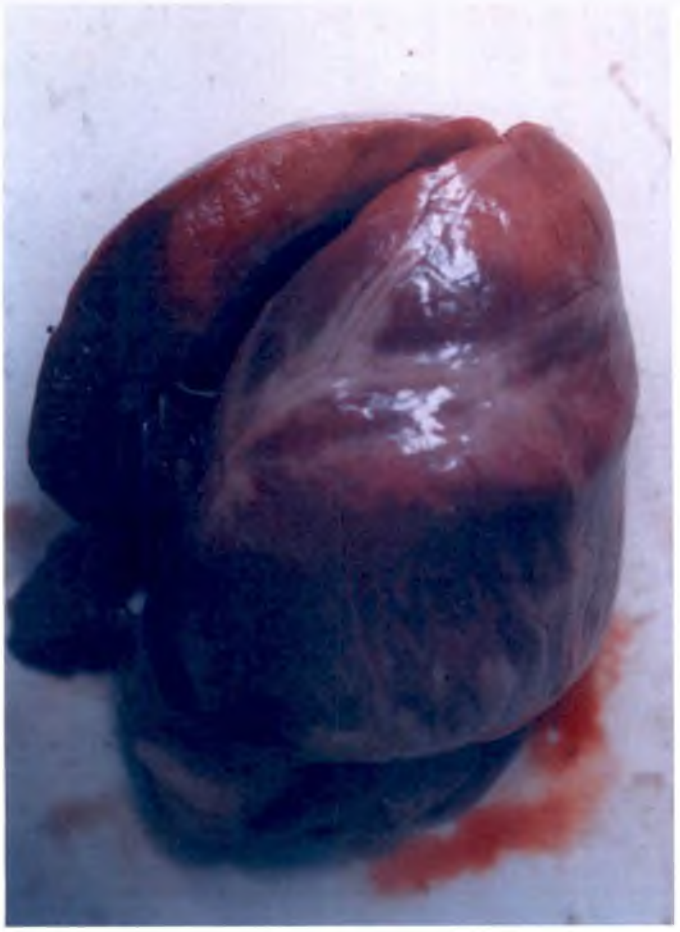


Fig. 12.

Fig. 13.

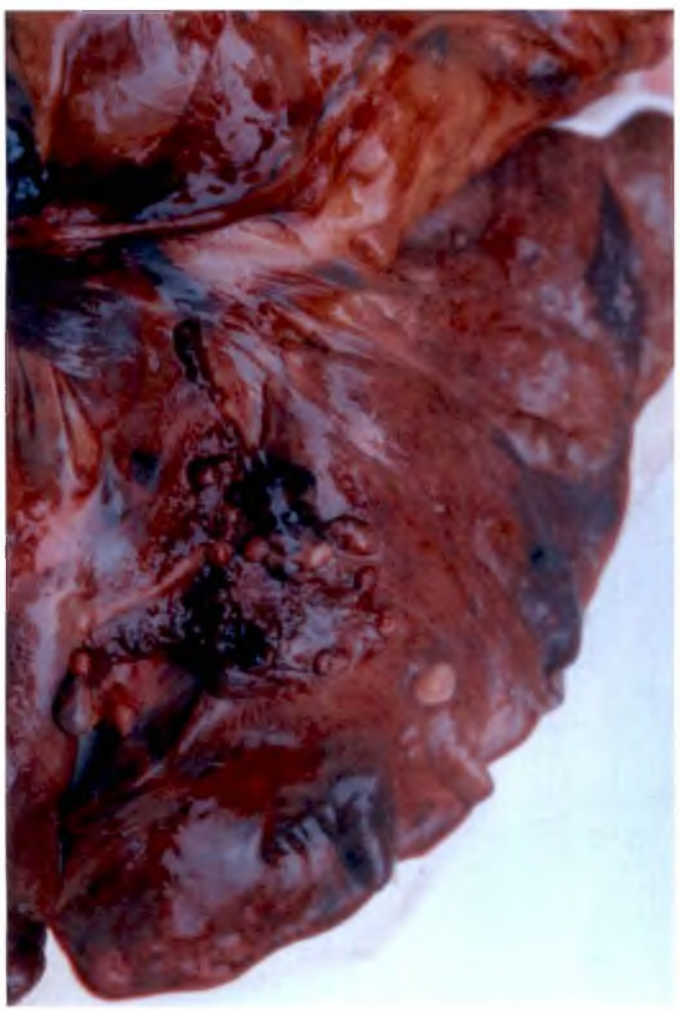


Fig. 14.

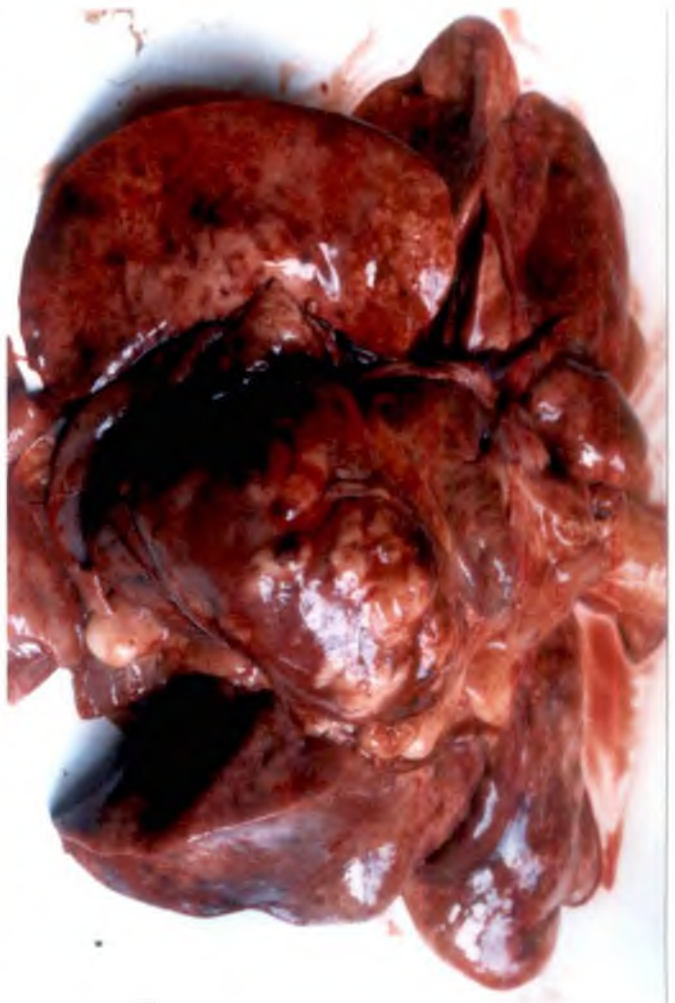


Fig. 15.

Fig.16 Multiple nodules in the endocardium- Lymphosarcoma

Fig.17 Lungs- Desquamation of bronchial epithelia and ulceration. Hyperplasia of peribronchial fibrous tissue and infiltration of inflammatory cells. Gomory's trichrome. x250

Fig.18 Lungs-Obliteration of bronchiole with papillary projection and necrotic materials. H&E x250

Fig.19. Lungs- Hyaline membrane formation in bronchiole. H&E x250

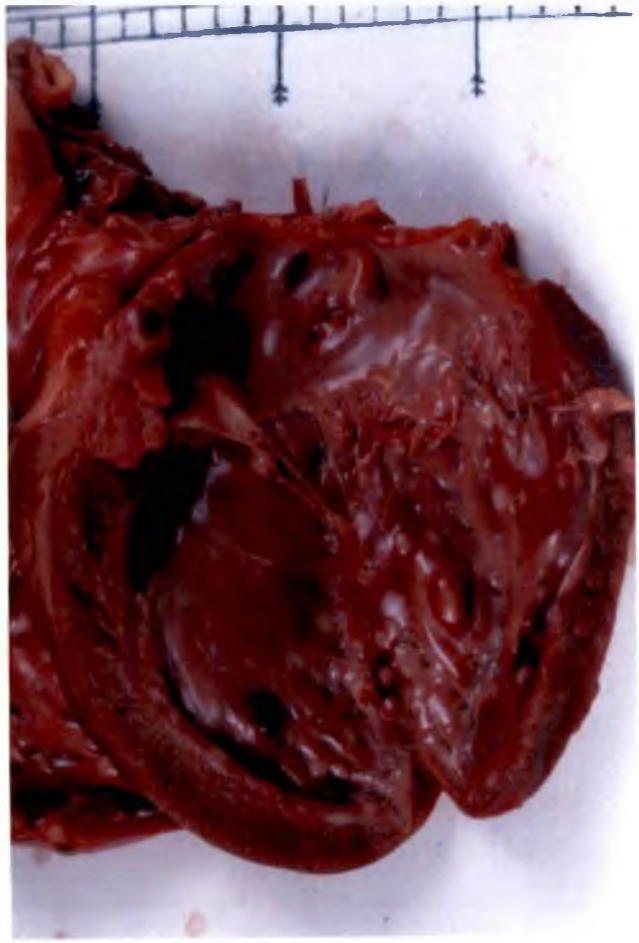
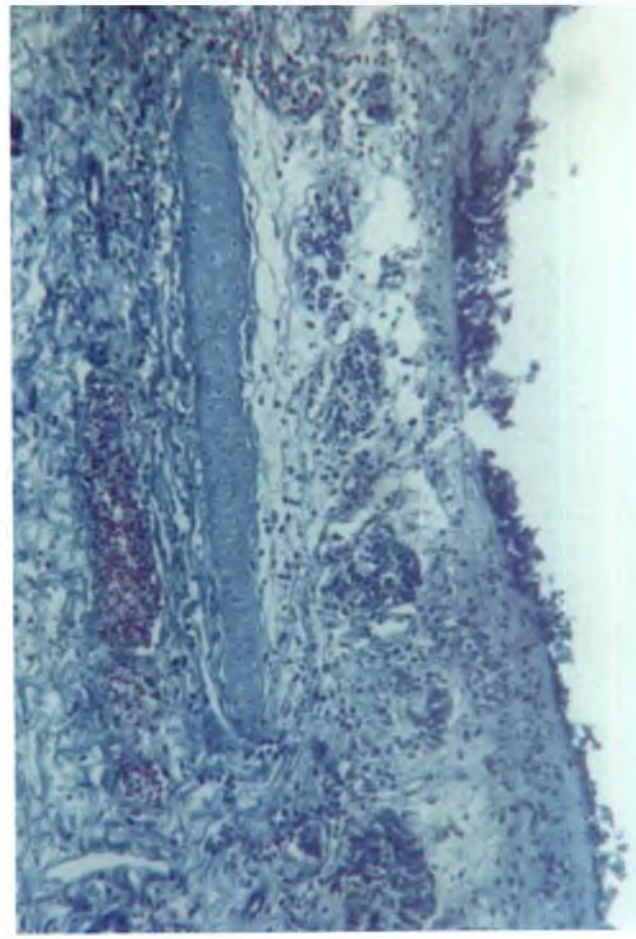


Fig. 16.



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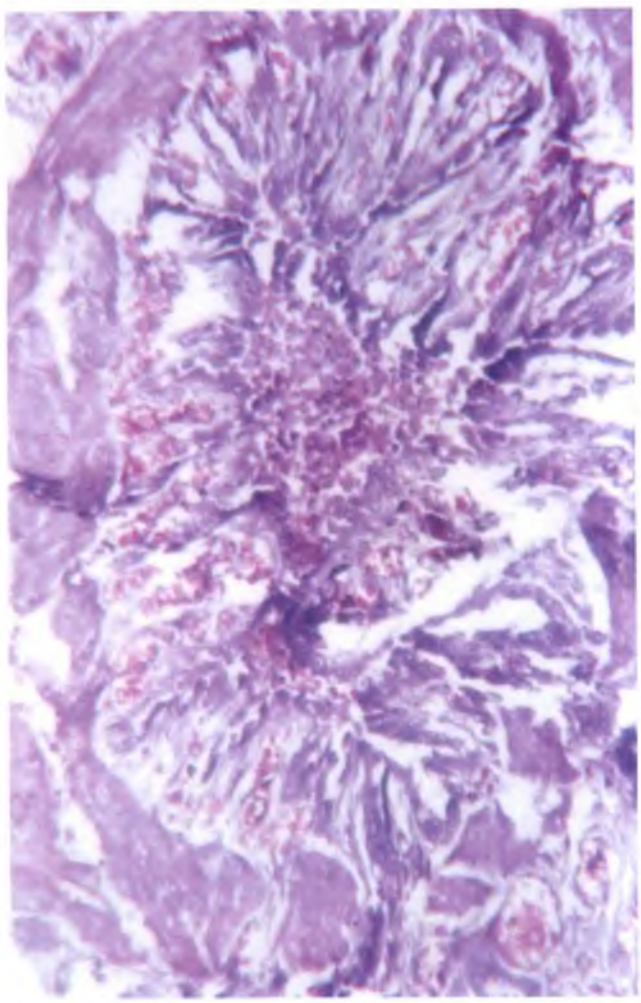

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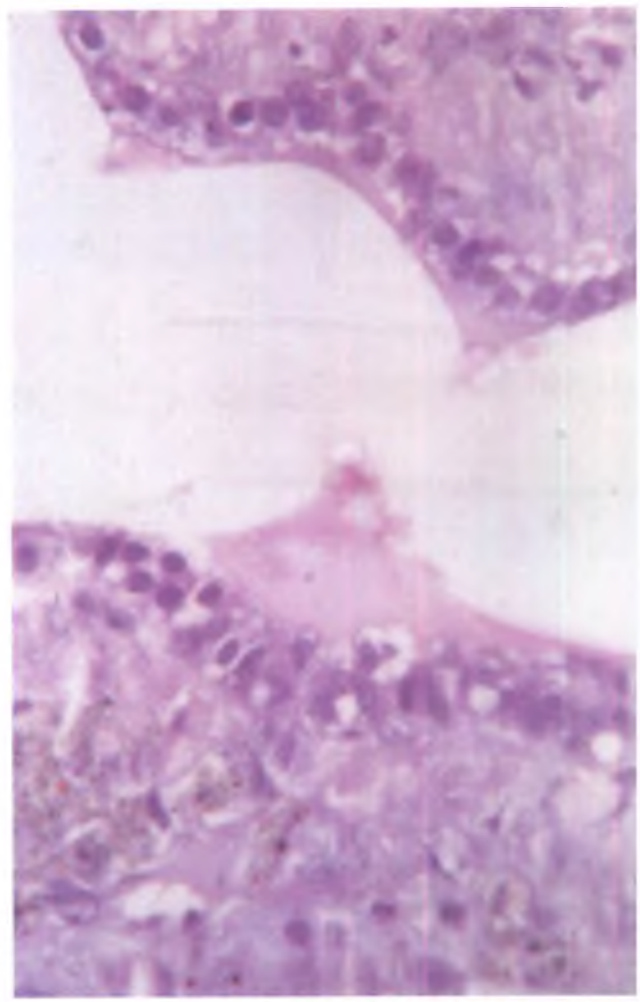


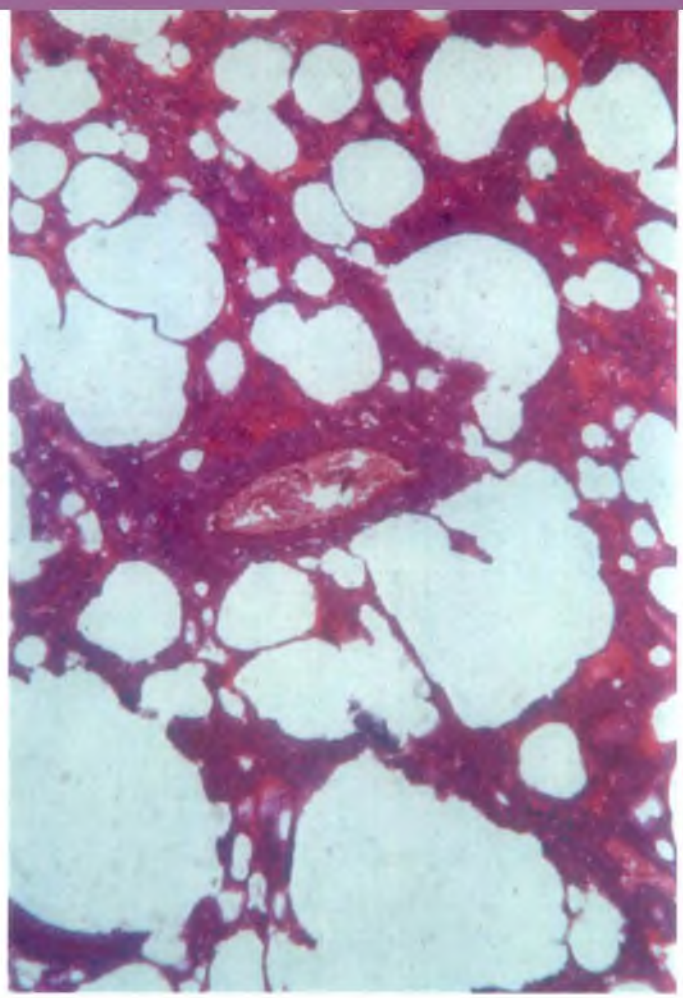
Fig. 19.

Fig.20. Lungs- Pulmonary congestion, septal thickening and emphysema. H&E x250

Fig.21 Lungs- Alveolar edema and hemosiderosis. H&E x250

Fig.22. Lungs- Alveoli filled with erythrocytes, necrotic changes- Pulmonary infarction. H&E x250

Fig.23. Lungs- Alveolar lumen filled with fibrin, alveolar macrophages and Pneumocytes- Pneumonia. H&E x1000



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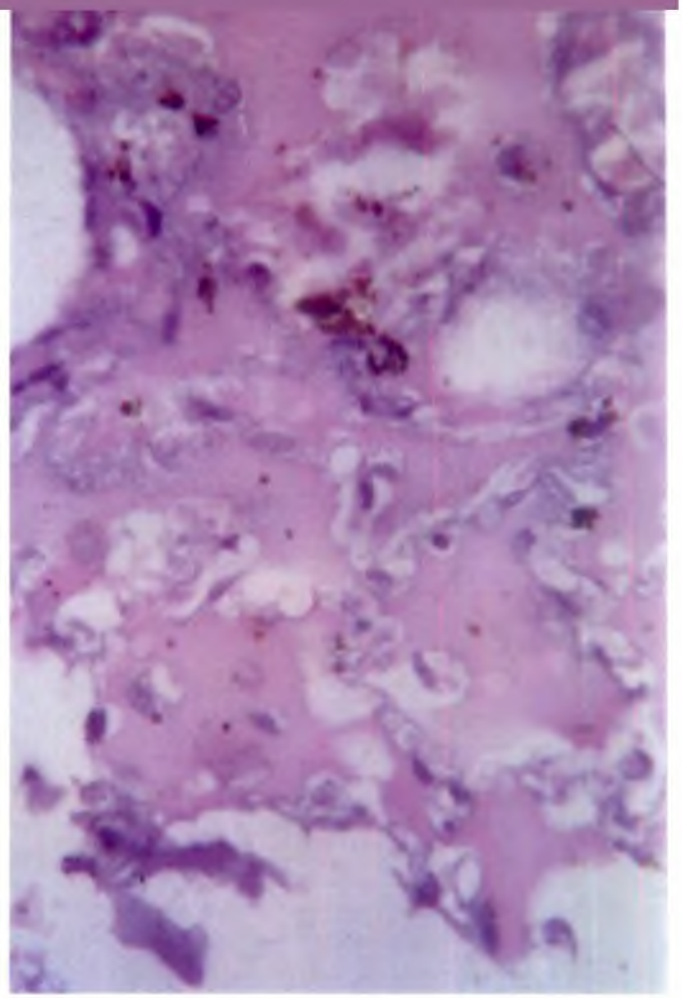

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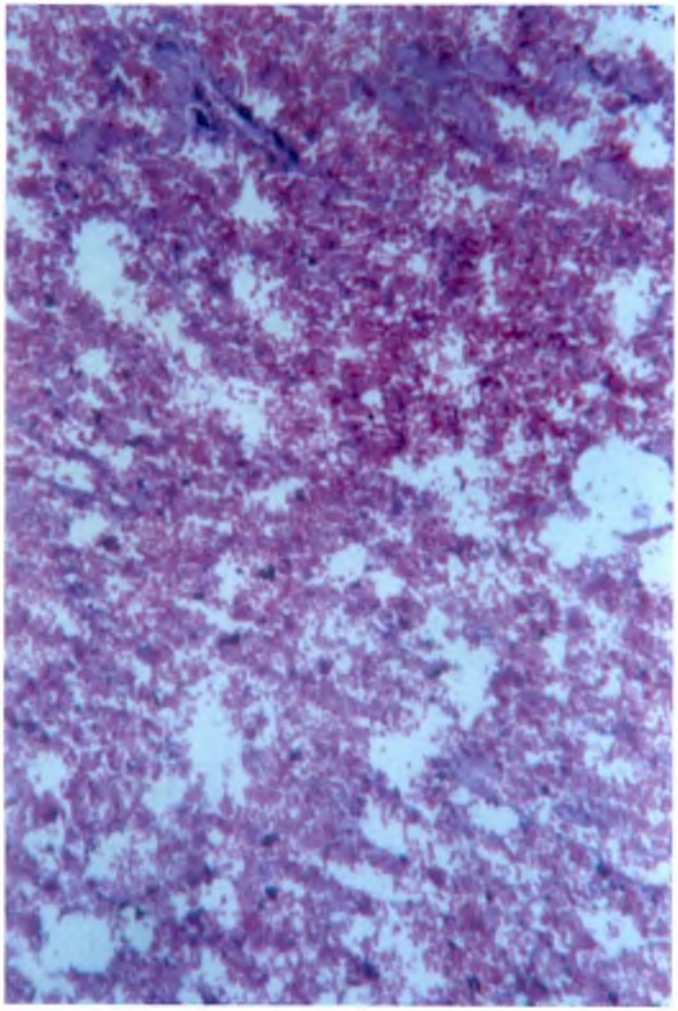

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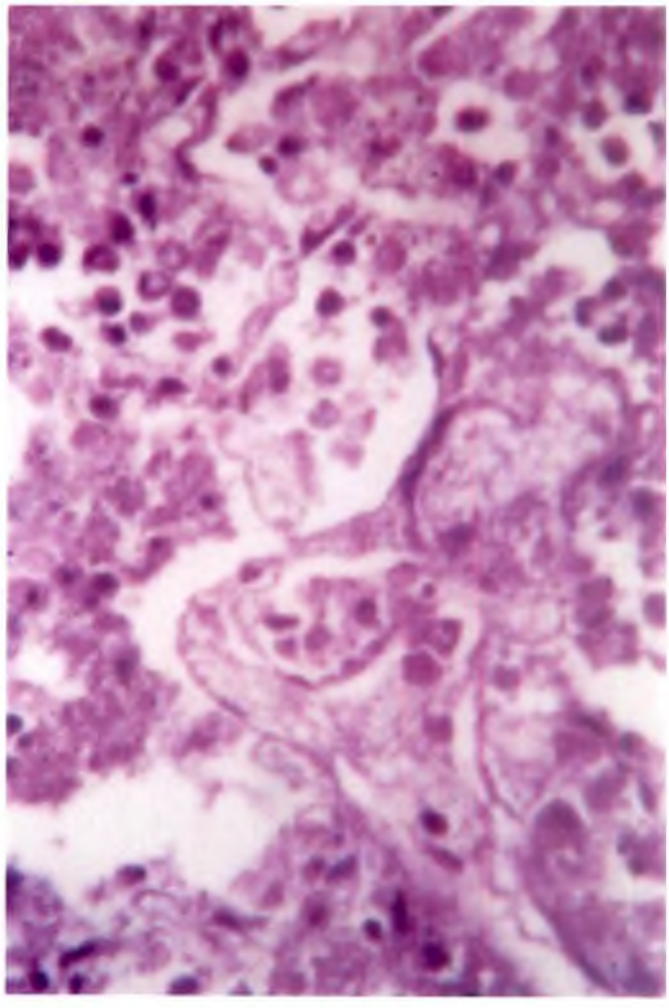

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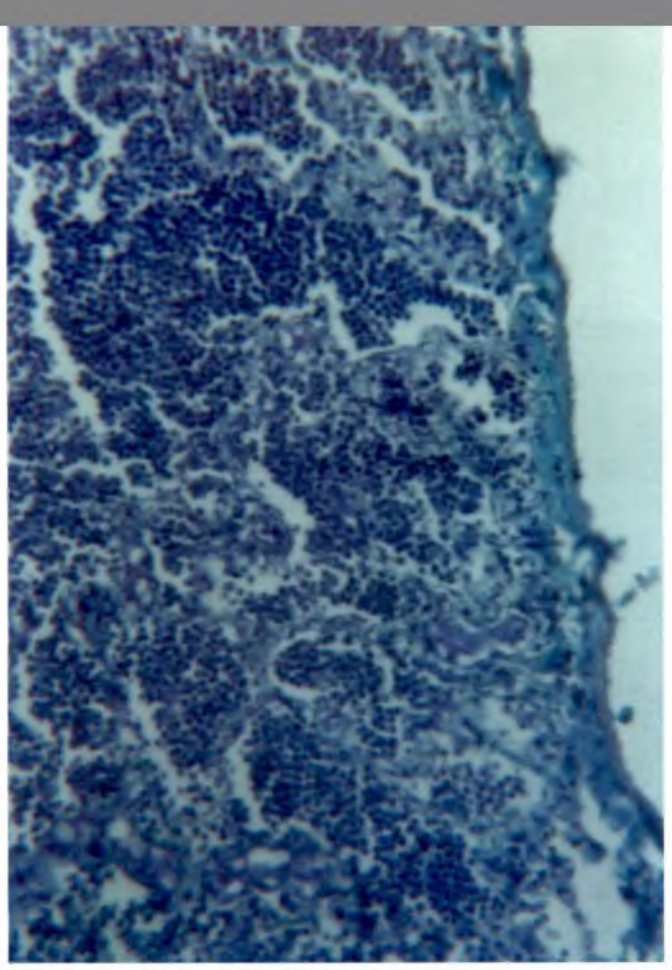

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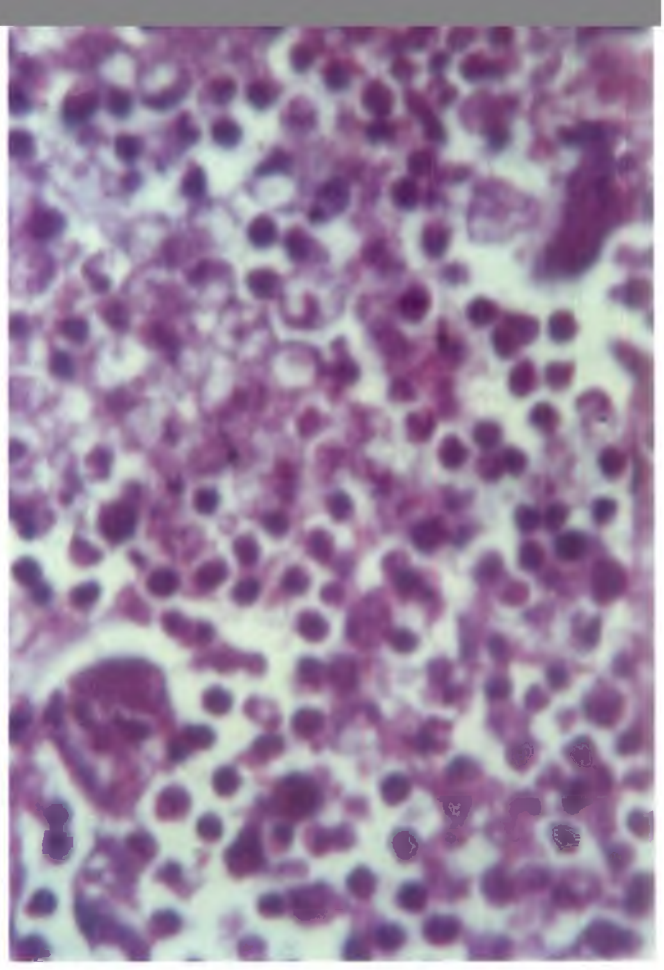

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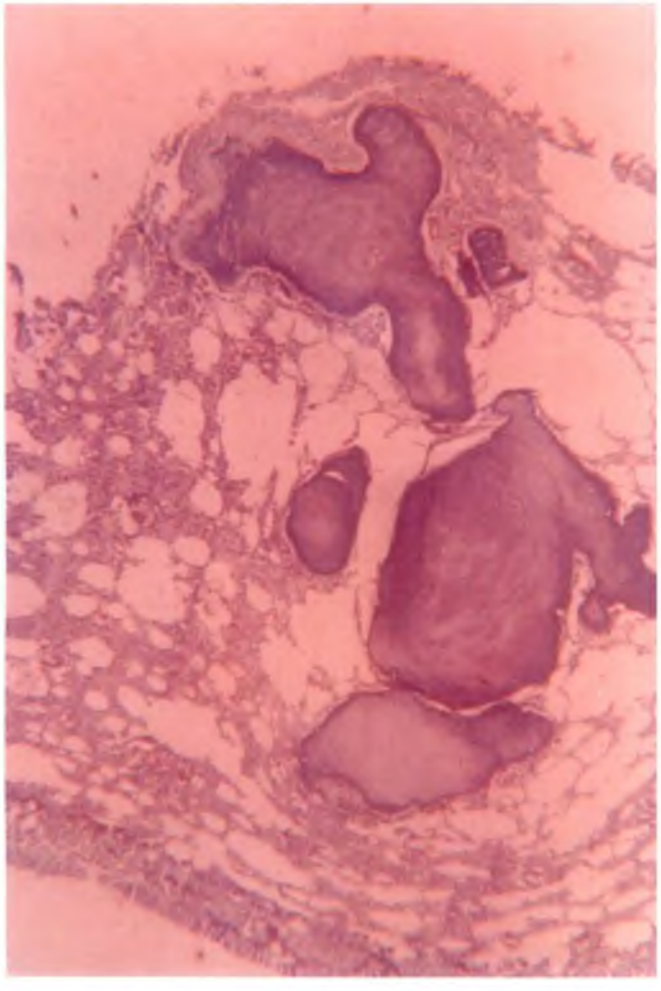


Fig. 26.

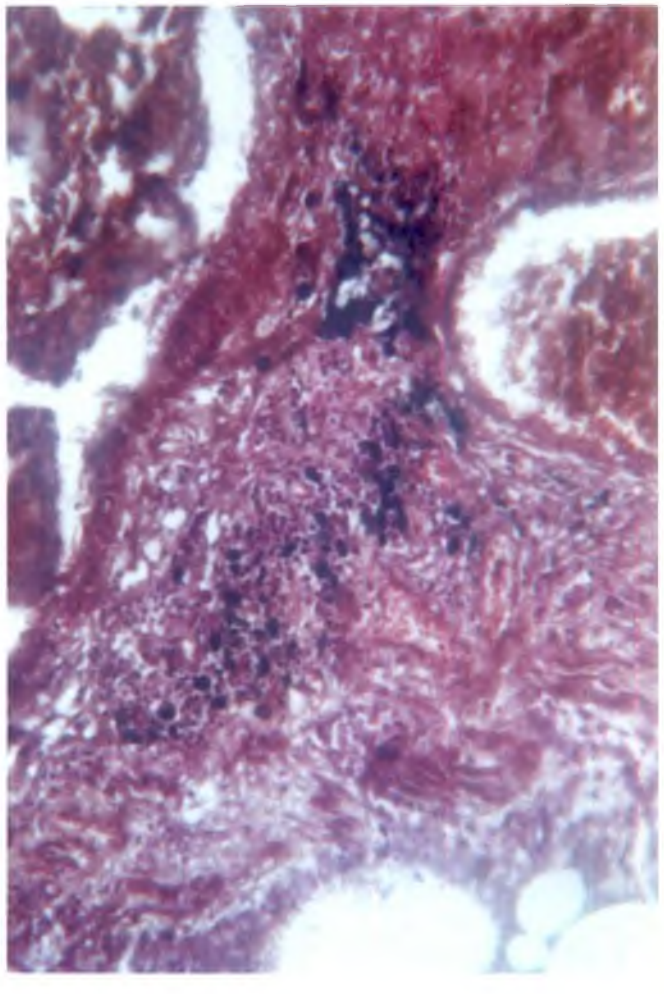


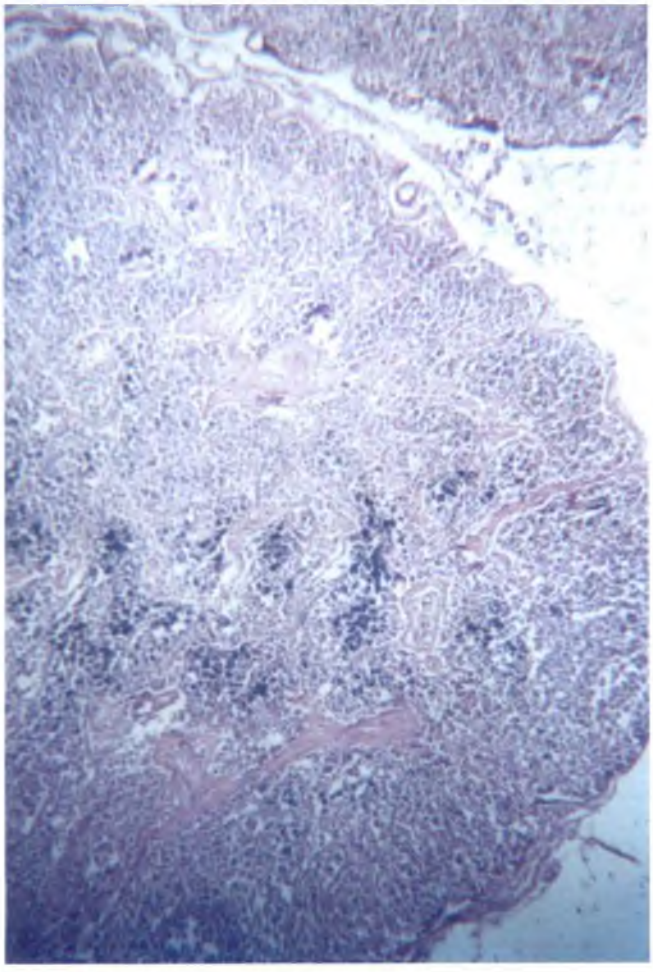
Fig. 27.

Fig. 28. Lymph node- Anthracosis. H&E x63

Fig.29 Lungs-Endarteritis obliterans. H&E x250

Fig.30. Heart- Attenuated wavy fibres. H&E x250

Fig. 31. Heart - Fibrous tissue proliferation in between necrotic cardiac muscle fibres. H&E x1000



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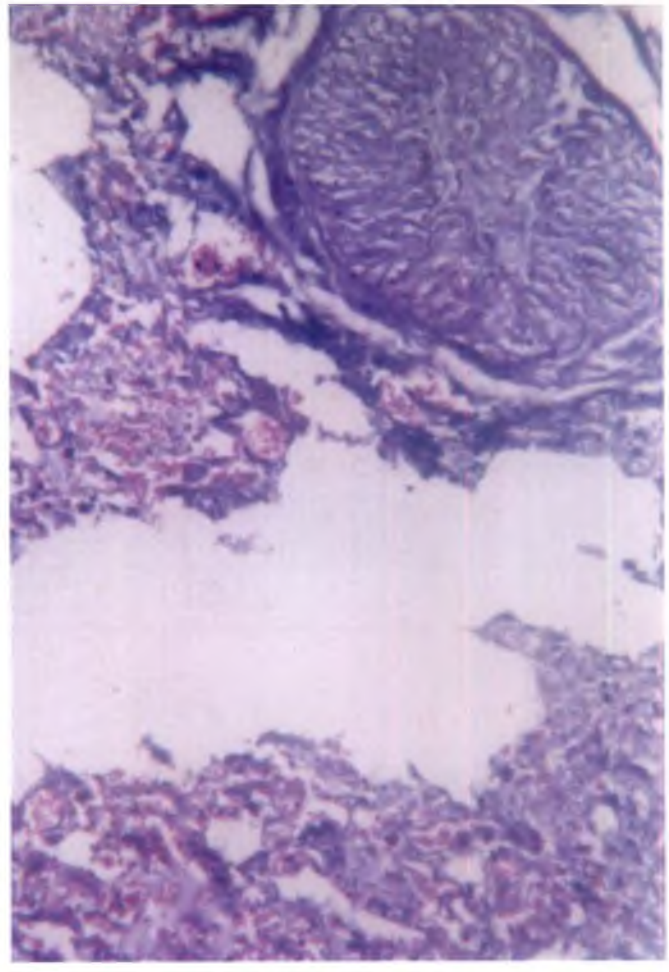

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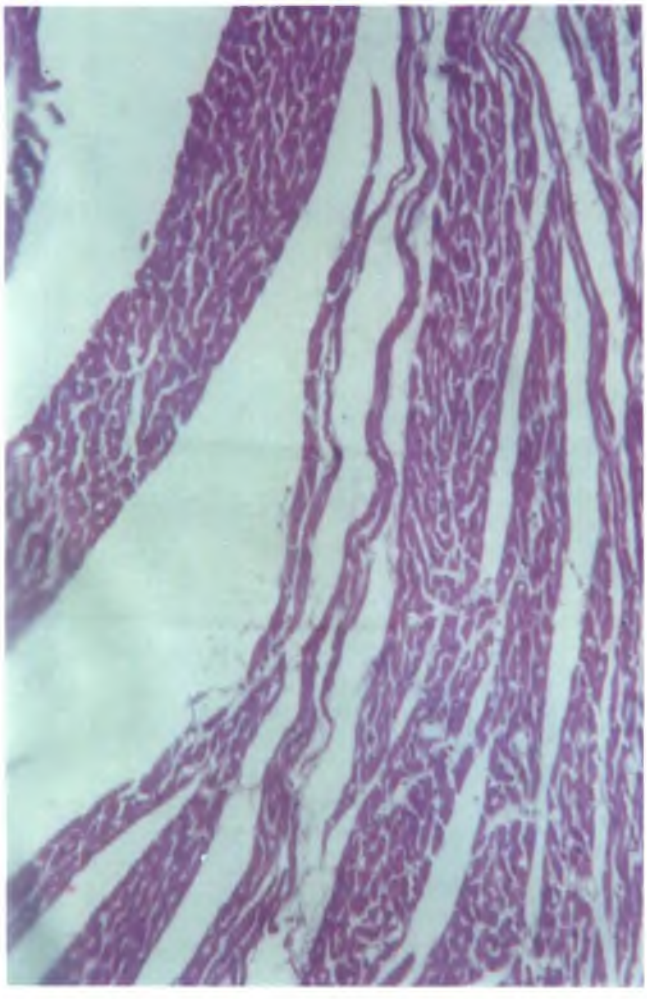

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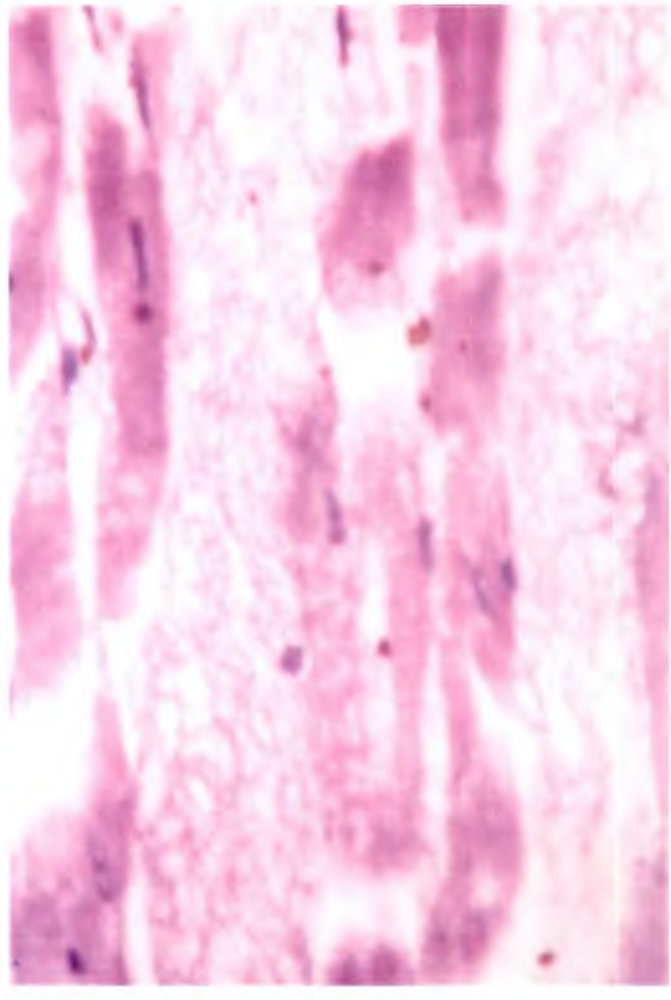


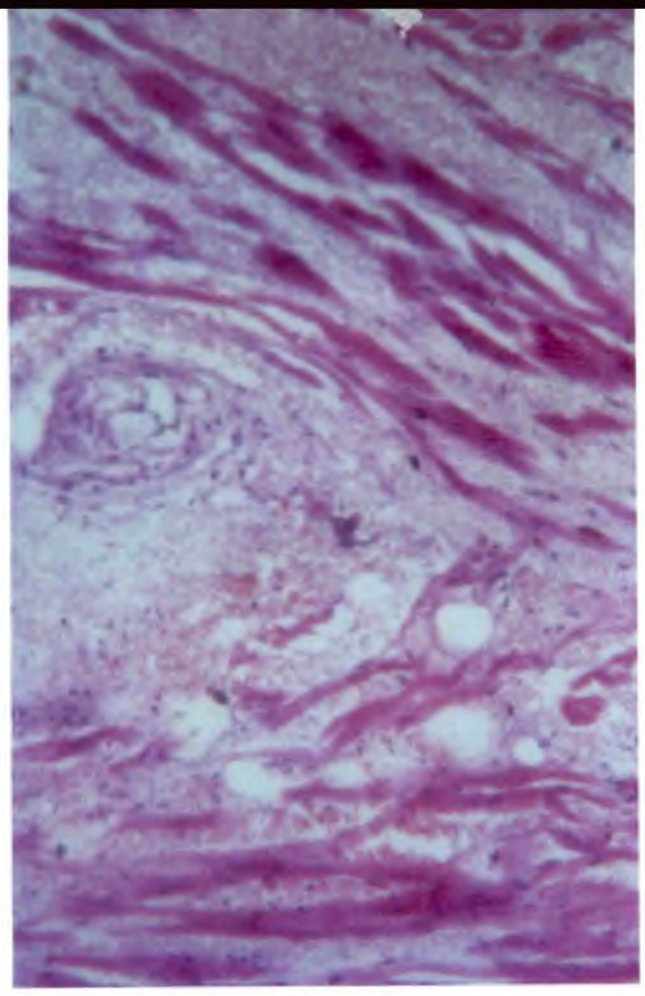
Fig. 31.

Fig. 32. Heart- Severe haemorrhage in between muscle fibres with blood vessel damage. H&E x250

Fig. 33. Heart- Vacuolation of cardiac fibres in the sub endocardial region. H&E x1000

Fig. 34. Heart- Inflammatory cell infiltration in the myocardium. H&E x1000

Fig. 35. Heart- Nitro-BT stain, differentiating normal dark blue stained muscle fibres with infarcted red stained fibres. H&E x250



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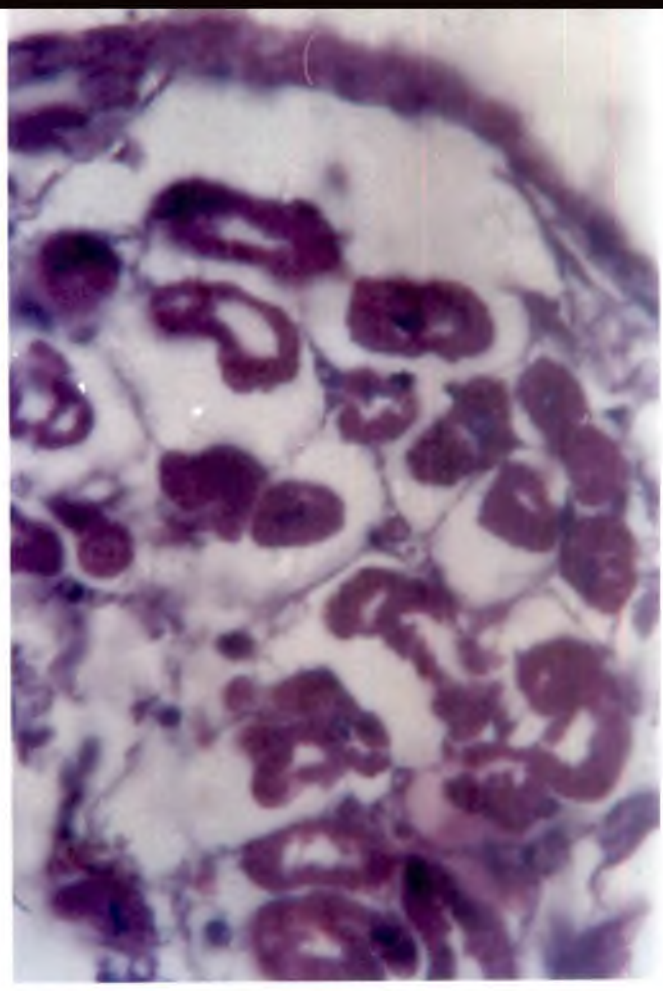
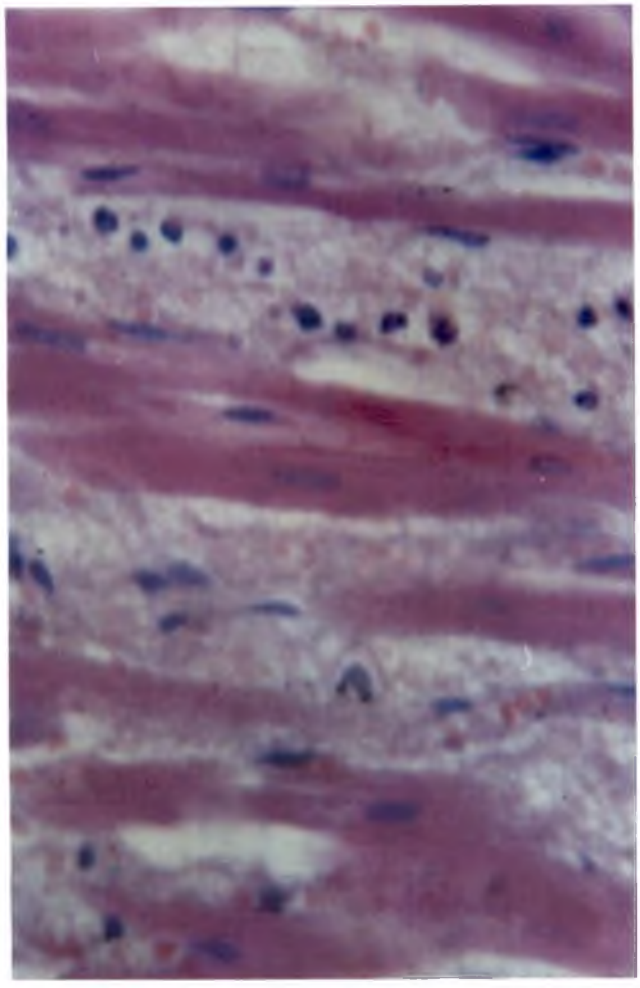


Fig. 33.



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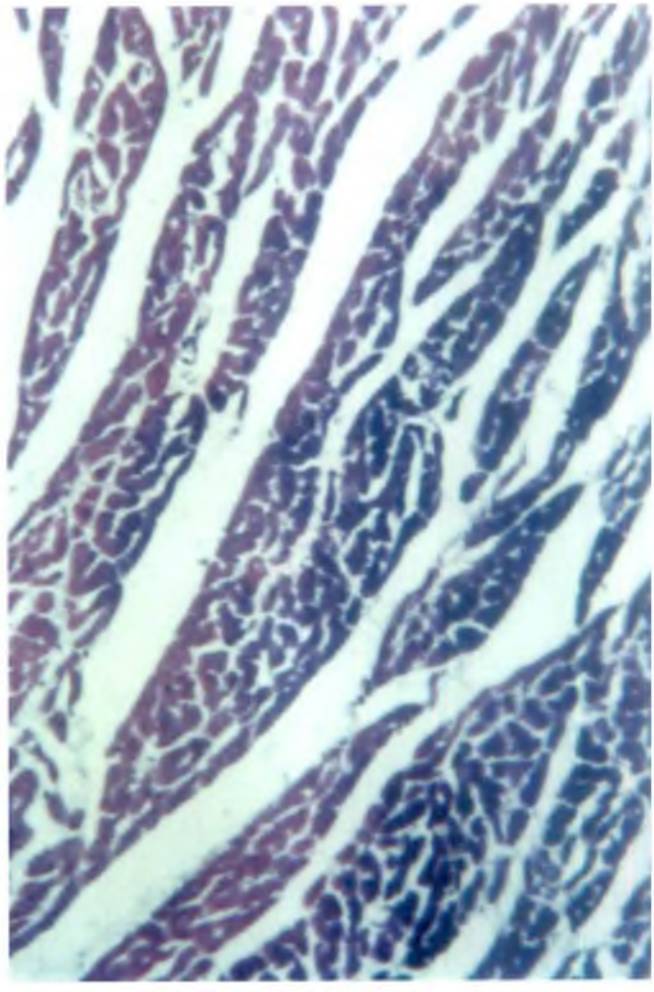


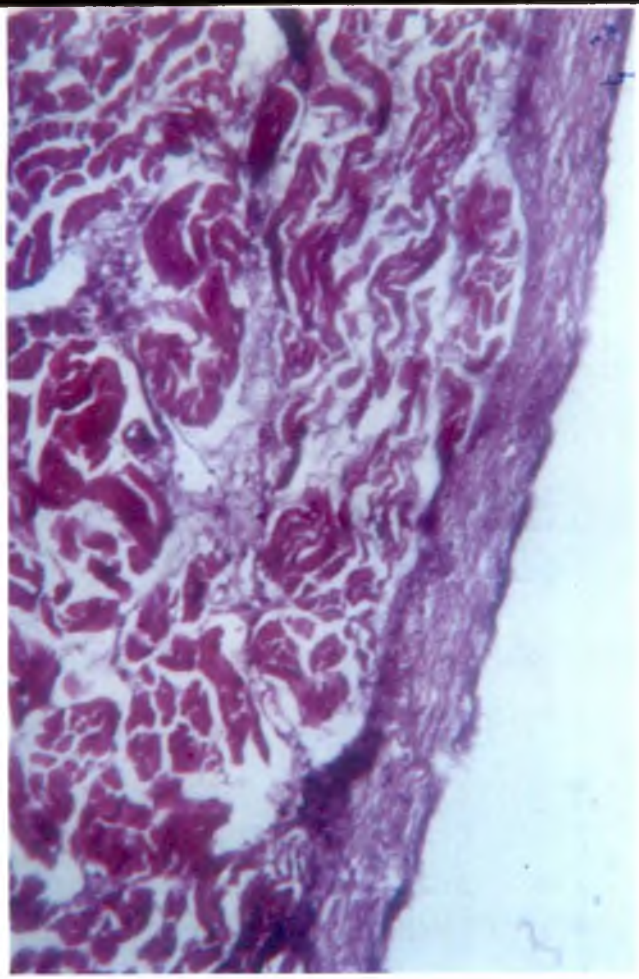
Fig. 35.

Fig.36 Heart- Endocardial fibroelastosis. H&E x250

Fig.37. Heart- Valvular endocarditis. H&E x160

Fig.38. Heart- Mural thrombus. H&E x250

Fig.39. Mitral valve- Blood cyst. H&E x63



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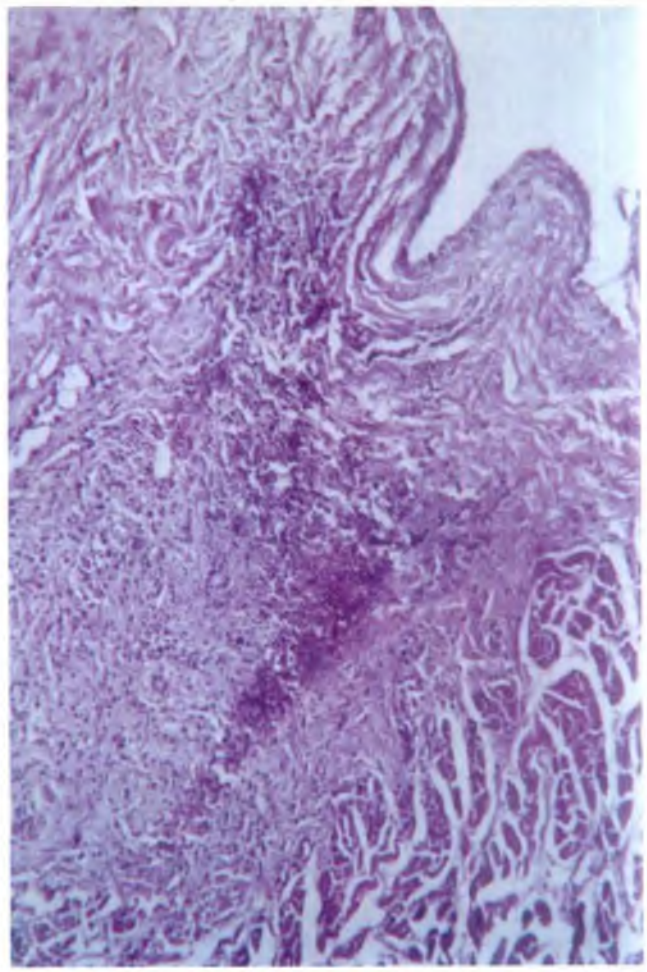

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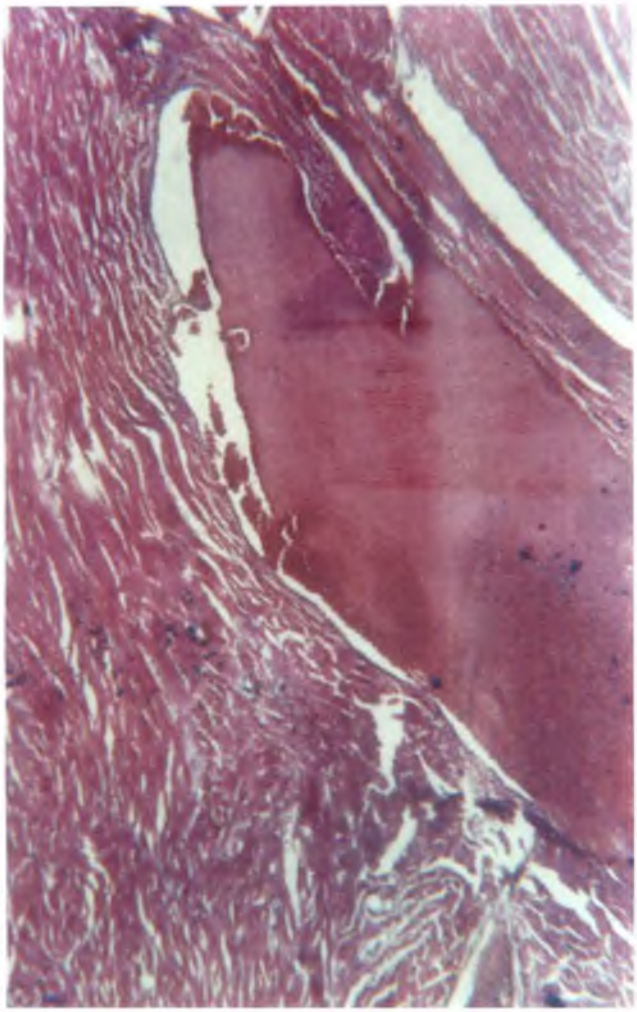

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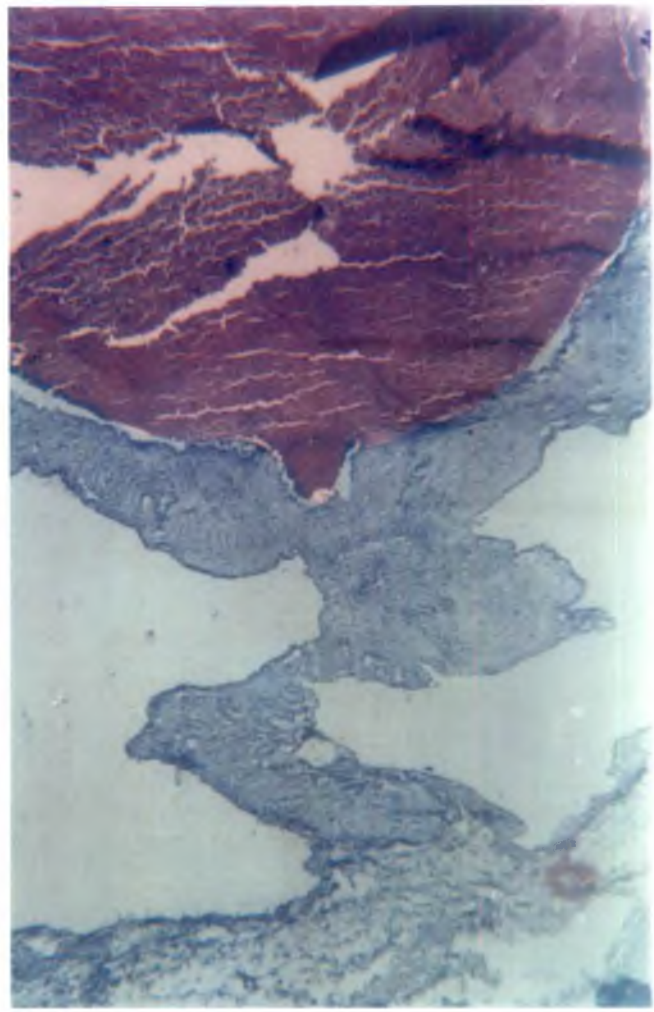


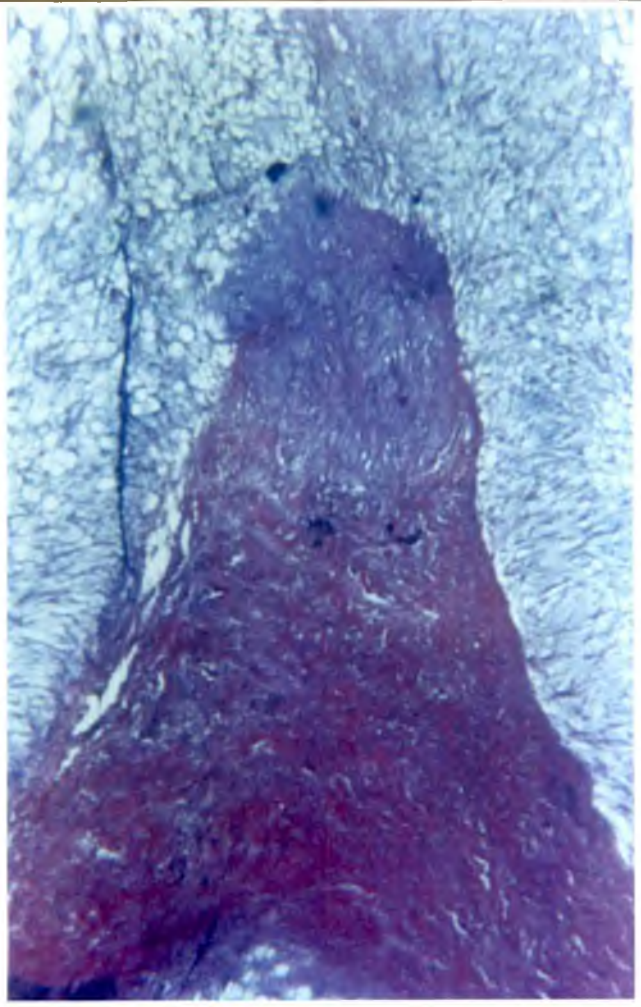
Fig. 39.

Fig.40. Mitral valve- Cartilagenous metaplasia at the region of attachment of chordae tendinae. H&E x63

Fig.41. Heart- Coronary arteriolosclerosis and vacuolisation in the medial layer. Mallory's Phosphotungstic acid-Haematoxylin. X250

Fig. 42. Lungs- Tumor nodule- Spindle shaped neoplastic cells and multinucleated giant cells- Osteosarcoma. H&E x250

Fig.43 Lungs- Poorly formed osseous tissue in the tumor nodule and collapse of adjacent alveoli- Osteosarcoma. H&E x250



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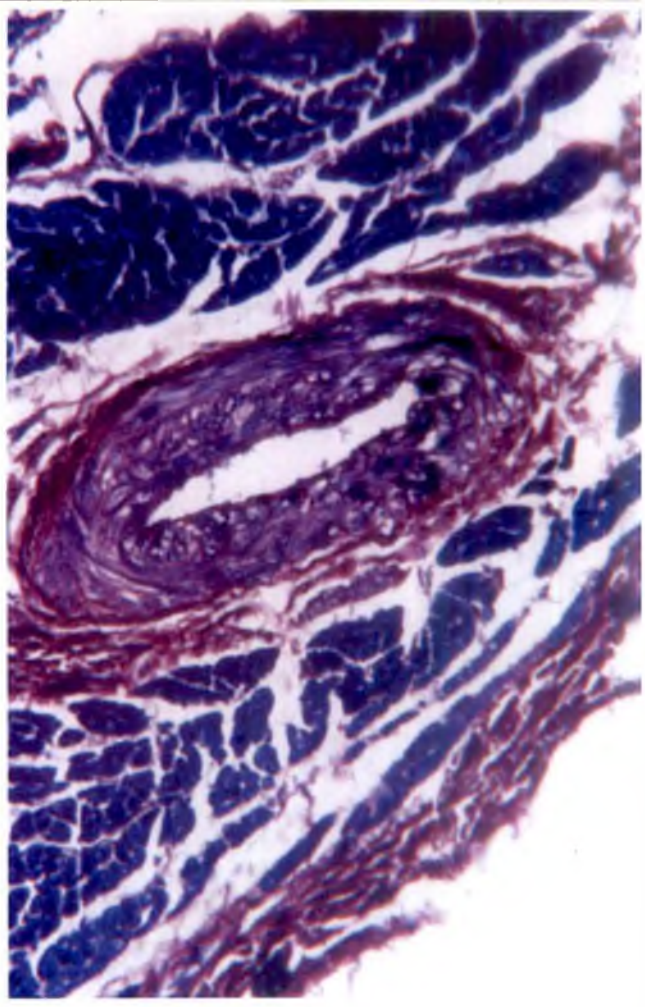

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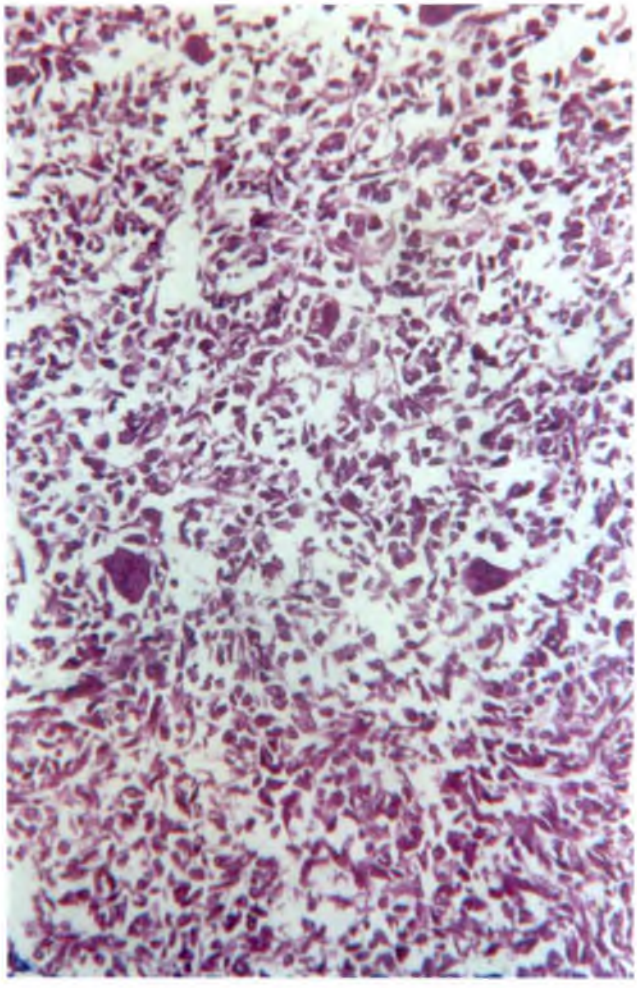


Fig. 42.

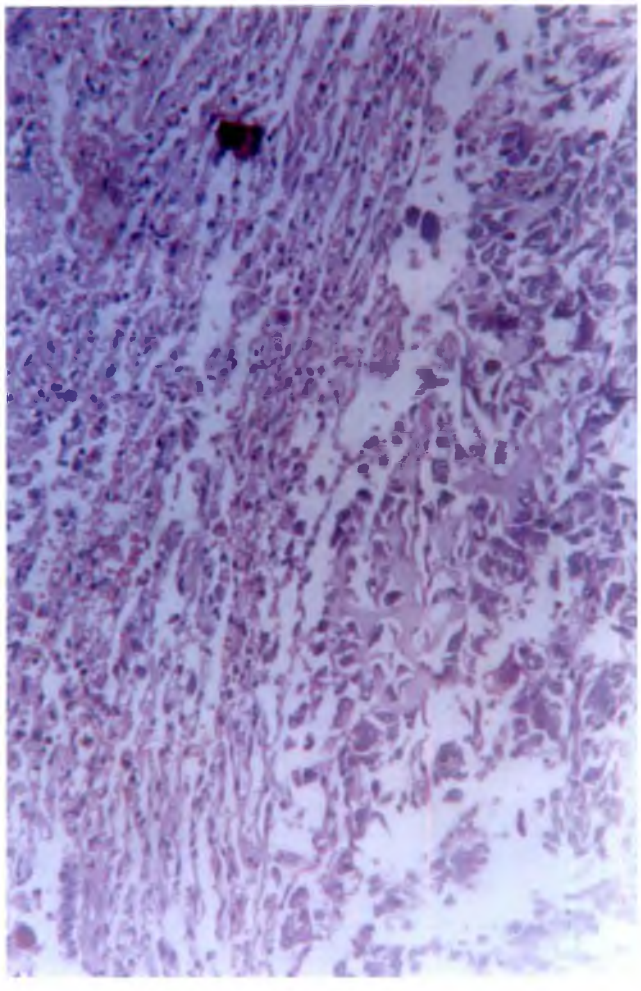


Fig. 43.

Fig.44 Lungs- Vacuolation of mesothelial cells- Osteosarcoma. H&E x250

Fig. 45. Lymph node- Neoplastic cells in the subcapsular area- Ostosarcoma H&E x250

Fig. 46. Heart- Lymphoblasts in between the myocardial fibres- Lymphosarcoma H&E x250

Fig. 47. Lungs- Collapse of bronchioles and peribronchiolar fibrous tissue hyperplasia H&E x250

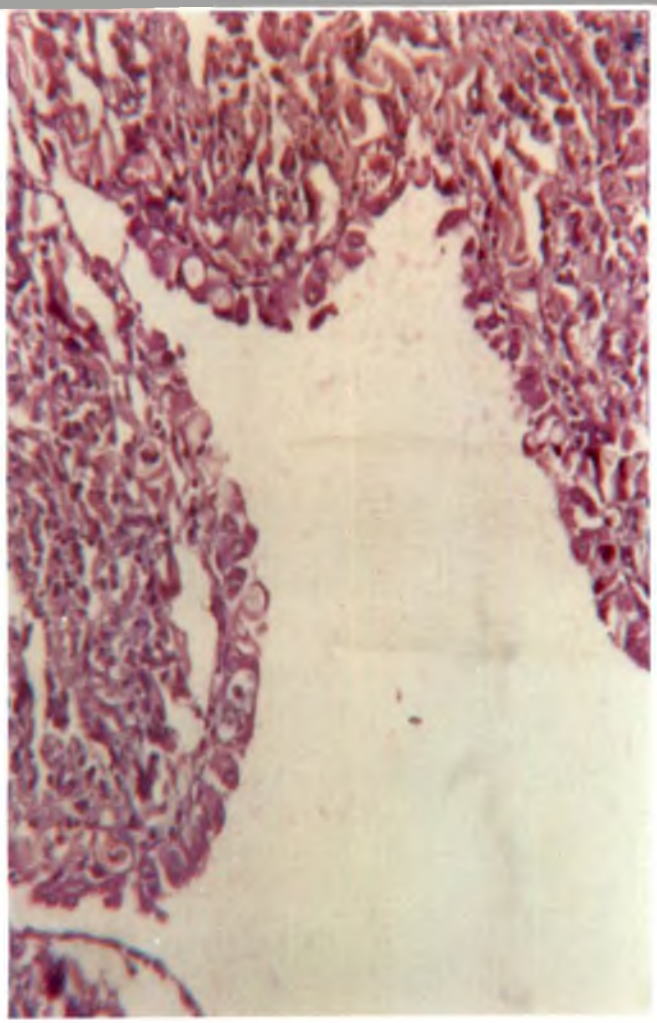
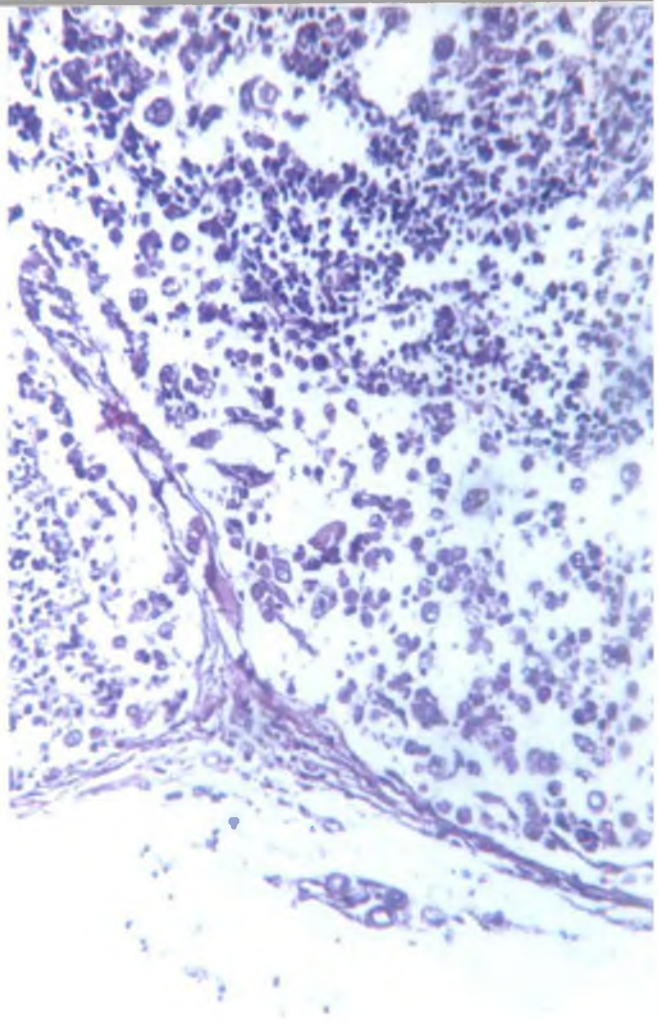


Fig. 44.



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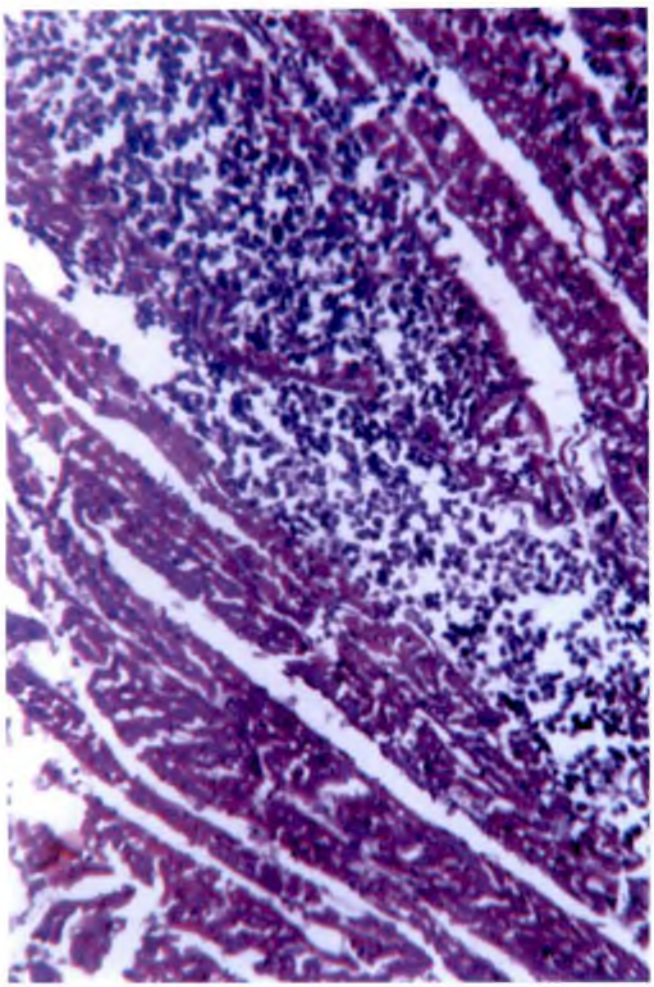

Fig. 46.

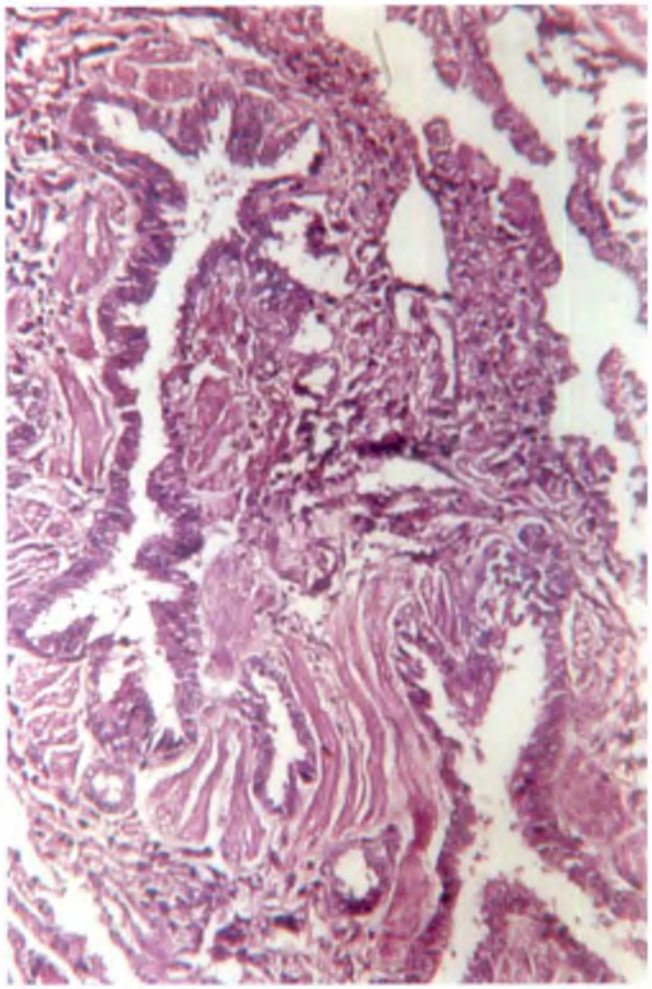

Fig. 47.

Discussion

5. DISCUSSION

The present study was undertaken to investigate the prevalence and pathology of cardio-pulmonary disorders in dogs. The data collected from the autopsy records maintained at the Centre of Excellence in Pathology, College of Veterinary and Animal Sciences, Mannuthy during the period of 1997-2002, gross and the histopathological features of samples collected from the necropsy cases formed the materials for the study.

Analysis of the data collected from the autopsy records revealed 47.05 per cent occurrence of various cardiac lesions and 83.38 per cent of pulmonary lesions for the past five year period. On the other hand, the present investigation revealed 98.71 per cent incidence of both cardiac and pulmonary lesions. This significant increase in the cardiac and pulmonary lesions could be attributed to a more focussed study as well as an increase in the environmental pollution.

Various cardiac and pulmonary lesions were classified and recorded on the basis of gross and histopathological examination

The most frequently encountered pulmonary lesions were congestion, oedema, haemorrhage and pneumonia. The conditions like emphysema, collapse, abscess, mineralization and ossification were also recorded. Though one case of abscess in the lung was recorded in retrospective study, such cases were not observed in the present study. This may be due to the less number of cases available during the period of study. The lung lesions were more frequent in right lobes when compared to the left lobes. This is in accordance with the observations of Kerr (1989).

The main histological lesions observed in the bronchi and bronchioles were desquamation of epithelial cells; proliferation of epithelial cells, squamous metaplasia, infiltration of inflammatory cells, dilatation of the bronchioles and

peribronchial fibrous tissue hyperplasia. Spencer (1969) reported that desquamation of the surface epithelial cells occurs at an early stage of ulcerative bronchiolitis. Bacteria like *Streptococcus* spp. and virus like adeno virus infection could result in ulcerative bronchitis. In the present study haemolytic Streptococci were isolated from one such case and another case was clinically suggestive of canine distemper. The proliferation of bronchial epithelium noticed in some cases could be attributed to atmospheric pollution as opined by Calderon-Garcideiunas *et al.* (2001) as most of the dogs autopsied were from urban areas.

Obliteration of a bronchiole found in one case was similar to the findings of Mayer and Katzenstein (1988) in human beings with bronchiolitis obliterans.

In a case where hypertrophic cardiomyopathy and renal lesions were noticed hyaline membrane formation in the bronchiole, eosinophilic proteinacious fluid accumulation in the alveoli and alveolar ducts along with haemosiderosis were observed. The lesions of the bronchioles and lungs could be attributed to the uremia associated with nephritis. Spencer (1969) also reported similar lesions in uraemic lung. He also reported that 'uraemic lung' can also be present in non-uraemic conditions like the left ventricular failure.

Squamous metaplasia with or without peribronchiolar fibrous tissue hyperplasia and dilatation of small bronchioles seen in a case of osteosarcoma were similar to the lesions described by Boyd (1985) for the atelectic bronchectasis.

Alveolar septal thickening found in some cases were caused by dilated capillaries with or without congestion, septal cell proliferation and/or interstitial fibrosis. In one of the cases from which haemolytic Streptococci was isolated, inter alveolar septa was thickened due to pneumocyte proliferation as well as infiltration of the mononuclear cells. This is in accordance with the observation of septal cell proliferation in Streptococcal pneumonia by Greene (1998).

Proliferation of type II pneumocyte could occur in air pollution as describe dby Takenaka *et al.* (1999).

Biventricular dilatations was mostly seen along with pulmonary congestion in this study which is in agreement with the observations of Dungworth (1993) that pulmonary congestion could be caused by left sided or biventricular cardiac failure.

Right ventricular dilatation and biventricular dilatation were the most frequent cardiac lesions seen with pulmonary haemorrhage and hemosiderosis. This finding is in accordance with Cotran *et al.* (1994). Pulmonary thrombosis was seen in some cases of pulmonary haemorrhage. Wu *et al.* (2002) also reported the occurrence of pulmonary haemorrhage along with multiple thrombo embolisms.

Pulmonary emphysema was found to be confined to the border areas of the lung lobes in most of the cases. Anderson (1987) also reported the occurrence of similar lesions. In the opinion of Dungworth (1993) other than the bullous type, emphysema has no clinical significance in dogs. Bullous emphysema were not seen in any of the cases.

Pulmonary oedema was prominent in cardiomyopathies like biventricular dilatation. Pulmonary oedema has been reported as secondary lesions in many extra pulmonary conditions like cardiac failure, subarachnoid haemorrhage (Macmillan *et al.*, 2002), cranial trauma (Drobatz et al., 1995) and acute pancreatitis (Lees *et al.*, 1978). The pulmonary oedema noticed in two dogs which had fatal injuries in the head in the present study is relevant in the above context.

Pulmonary infarction was noticed in two cases with extravasated erythrocytes flooding the alveoli and bronchioles and necrosis of alveolar structures. This is in close agreement with reports of Spencer (1969) that pulmonary infarcts in dogs were hemorrhagic owing to the high collateral

circulation. The heart showed biventricular dilatation. This finding is in accordance with the findings of Torack (1958) that pulmonary venous congestion and heart failure can cause pulmonary infarction. Though he stated that, bronchial obstruction, pneumonic consolidation and shock can also produce pulmonary infarction such lesions were not encountered in the above stated cases.

Pneumonic lesions were more frequent and severe in the middle lobes. Ramachandran (1970) also noted the frequent middle lobe affection in pigs. Most of the pneumonic cases showed proliferation of type II pneumocytes, alveolar macrophages and mononuclear inflammatory cells. *Streptococcus pyogens* was isolated form one pneumonic case. This is in accordance with the findings of Greene (1998). Calvert (1982) reported endocarditis caused by haemolytic Streptococci, however no such lesions were noticed in the heart of streptococcal pneumonic case. In most of the pneumonic cases both the bronchioles and alveoli were found to be affected. This points to the chances of viraemic infection as described by Dungworth (1993). In another case of pneumonia infarction of the myocardium, kidney and spleen were evident by gross examination and inflammation of the mitral valve on microscopic examination, which could be attributed to septicaemia.

Translucent gritty materials found in the sub pleural region could not be demonstrated histologically. This may be due to the loss of crystals during the processing or cutting of the tissues. These lesions were found mostly in animals of above five years of age. No such lesions were detected in animals below one year of age. Day *et al.* (1996) reported black gritty deposits of carbon aggregates in dogs. Hawkins (2000) reported that mineralization of airway or pleura occur in aged dogs of any breeds and have no correlation with the disease.

Osseous tissue formation in the lung was noticed in three cases. Spencer (1969) opined mitral stenosis and pulmonary hypertension as the main causes for bone formation. One case showed hydropericardium, left ventricular hypertrophy with right ventricular dilatation and ascites indicative of heart failure. In other

two cases biventricular dilatation was evident, one of which showed endarteritis obliterans of pulmonary arteriole. Heart affection might have caused pulmonary hypertension and osseous tissue formation in all the three cases. Though Brix *et al.* (1994) had opined that "ectopic ossification of the interstitium in the lung of dog has no pathological significance", the findings of the present study are strongly contradictory to this statement and support the findings of Spencer (1969).

All the cases of anthracosis noticed in the study were above one year of age and mostly above five years. The nondescript breeds were more affected when compared to other breeds. The observation of higher incidence of anthracosis in non descript breeds points to the fact that they are not reared under ideal management conditions and the chances of getting exposed to automobile exhausts is more. Anthracosis in older age group is indicative of prolonged exposure to the environmental carbon particles. It is again an indication of increased carbon pollution in the atmosphere.

Pulmonary arterial wall thickening was noticed in many cases. Cardiomyopathic changes and congestive changes of the lungs were associated with most of the cases of pulmonary arterial wall thickening. One of the cases showing endarteritis obliterans had osteosarcoma in the lung. The pressure from the tumor mass might have caused the obliterative changes. This correlates with the findings of Jones *et al.* (1984) that proliferation of muscle in the media of small arteries along with vessel occlusion was due to increased vascular resistance. The findings of the present study is in agreement with the observation of Chazova *et al.* (1995) that intimal thickening of pulmonary artery could be resulted from cardiac disorders.

Pulmonary thrombosis was observed in three cases. In all the cases septal capillaries were congested. Haemorrhage into bronchioles and alveoli were observed in one case. This finding is in accordance with that of Wu *et al.* (2002).

Right ventricular dilatation was seen in two cases of pulmonary thrombosis. The thrombi might have caused vascular resistance and heart failure.

Hydropericardium was observed in four cases. In one case along with hydropericardium, ascites was also present and the heart showed right ventricular dilatation with left ventricular hypertrophy and bone formation in the lung. Cardiomyopathy was evident histologically in all the cases of hydropericardium. Nephritis was also present in one case. This is in accordance with the findings of Wright and Heard (1979) that hydropericardium can be induced by renal and cardiac failure.

In the case of cardiac tamponade the blood clot in the pericardium might had been formed from oozing of blood through the wound present in the pulmonary vein. Though the lesion was suspected to be a bullet wound, no bullet could be recovered. The right auricle was empty, heart showed left ventricular hypertrophy and the lung showed diffuse congestion. As described by Boyd (1985) the tamponade might have compressed the auricle and prevented its filling and death occurred by cardiac failure.

The animal that showed gelatinisation of the epicardial fat was chronically ill for a long period and anorectic. Utilization of fat as a source of energy might have led to depletion and subsequent gelatinisation of fat as described by Robinson and Maxie (1993). Petechiae noticed in many could be caused by agony of death or by infectious conditions. Olson and Miller (1986) reported epicardial, endocardial and myocardial haemorrhage in dogs infected with pseudorabies.

Ecchymotic haemorrhage in the endocardium was more frequent in left ventricle. Runnels (1956) observed endocardial and myocardial haemorrhage in viral diseases. Bisi (2002) recorded endocardial haemorrhage in a rabid cattle. In the present study a rabies case confirmed by Negri body demonstration in the hippocampus didn't show any endocardial haemorrhage.

Endocardial fibrosis was more frequent in older animals. This is in agreement with the findings of Whitney (1974), Van Vleet and Ferrans (1986) and Sisson and Thomas (2000). In the present investigation cardiomyopathies were present in all the cases showing fibroelastosis and the findings of the above authors holds good in the present study also.

Cartilaginous metaplasia of the endocardium was seen at the tip of the papillary muscle of the left ventricle in a two-year-old German Shepherd dog. Mitral valve also showed the cartilaginous metaplasia at the site of attachment of the chordae tendineae (the collagenous area). The above findings correlated with the report of cartilage formation in the collagenous endocardial tissue in a case of fibroelastosis in Great Dane pup by Krahwinkle and Coogan (1971). The lung lesion noted in the case was diffuse congestion, thickening and vacuolation of the pulmonary artery and obliteration of the bronchiole. The chronic heart lesion might have attributed the lesions in the lungs.

A blood cyst was observed in the mitral valve of a two-year-old female dog. The lesions found, were in accordance with the report of Takeda *et al.* (1991). They suggested that mechanical effect such as an increase in tension, friction and impact could trigger the enlargement of valvular blood vessels in a sort of valvular telengectiasis with subsequent cyst formation. Marcato *et al.* (1996) also reported that cyst formation was due to dilatation of blood and lymph vessels. The heart of the present case showed dilatation of all the chambers. The lungs were diffusely congested and oedematous. Cyst might have caused the mitral stenosis and the lung lesions.

Endocarditis was noticed in one case, in which generalised inflammation was found in the lungs and rectum and infarction was noticed in the heart and kidney. These lesions indicated that the endocarditis might have resulted from septicaemia. No isolation was done in the case. Robinson and Maxie (1993) reported that endocarditis was seldom seen in dogs. Calvert (1982) reported that most of the endocarditis in dogs was caused by *Streptococci* spp. In the present

study endocarditis could not be detected from the case in which *Streptococci* were isolated.

Both hypertrophic and dilated cardiomyopathies were noticed in the present investigation. The heart with ventricular hypertrophy showed histological lesions like swelling, crowding and fragmentation of cardiac muscle fibres, interstitial fibrosis, myocyte disarray and endocardial fibrosis. These lesions are in accordance with the findings of Maron (1985); Van Vleet and Ferrans (1986) and Sisson and Thomas (2000). Oslen (1971) reported fibrous tissue replacement of the cardiac fibres in human hypertrophic cardiomyopathy.

Heart with right ventricular dilatation and biventricular dilatation showed lesions suggestive of dilated cardiomyopathy as described by Liu and Tilly (1980); Sandusky *et al.* (1984) Tidholm and Jonsson (1996) and Everett *et al.* (1999). Occurrence of endocardial fibroelastosis in some cases of dilated ventricle is in accordance with the findings of Van Vleet and Ferrans (1986). Cartilaginous metaplasia at the tip of the left ventricular papillary muscle in the biventricular dilated heart of a German Shepherd dog was similar to the lesion as reported by James and Drake (1968) in a Doberman Pinscher. Though fragmentation, fibrous tissue replacement and fat deposition were seen in the myocardium of the dilated right ventricles, the lesion reported by Bright and Mc Entee (1995) with complete sparing of left ventricle could not be detected in any case. The incidence of the cardiomyopathies was more frequent in male and aged dogs. This is in accordance with the findings of Calvert *et al.* (1997) and Braslasu (2000). Tidholm and Jonsson (1997) reported that hypothyroidism might predispose the dogs to dilated cardiomyopathy while infectious etiology was recorded by Van Vleet and Ferrans (1986). In the present study also, this could be attributed to the infectious etiology.

Myocardial infarction was detected in five cases. Nitro-BT staining was used for macroscopical identification of myocardial infarction. In histological section also Nitro- BT differentiated normal and infarcted areas. This shows that

the gross staining of the suspected infarct does not affect the result of the histological interpretation; rather, it enables a better differentiation of the infarcted area. This is in the support of the findings of Nachalas and Shantika (1963). All the myocardial infarctions detected in present study were in the left ventricular free wall and in the interventricular septum. This correlates with the findings of Ratliff *et al.* (1967). No infarction could be detected in the right ventricle as reported by Francis *et al.* (1989). In one of the cases of myocardial infarction, Leptospira could be detected. Myocardial necrosis in leptospirosis in human was reported by McKinney (1974). The lungs of this case showed septal congestion, haemorrhage and epithelial cell proliferation. Vacuolation of myocardial fibres were also noted. Vacuolation was also present in other cases of myocardial infarction and was severe in sub-endocardial region in one case. Jones *et al.* (1985) reported myocardial vacuolisation in infarct border zones and sub-endocardial region.

Coronary artery thickening was noticed in many cases. Intimal thickening was more frequent than medial thickening. Vacuolation in the coronary vessel wall was also noticed in some cases. In the cases with coronary wall thickening, congestion, haemorrhage and septal cell proliferation were the main pulmonary lesions. In cases of myocardial infarction also, coronary arteries were found thickened. In the opinion of Robinson and Maxie (1993), intimal proliferation may occur in response to decreased coronary perfusion and increased intramural tension during systole. Intimal thickening due to fibro elastic and smooth muscle proliferation can result in myocardial infarction and scarring. Liu *et al.* (1986) reported vacuolation or foamy cells and cystic spaces found in the coronary arteries with or without eroded intima and degenerated muscle cells as the major histological features of atherosclerosis. Similar lesions were encountered in the present study also. According to Cliff *et al.* (1988) a strong correlation exists between infection and atherosclerosis of the coronary arteries.

the pulmonary pressure, caused the cardiac failure. Moreover, cardiac lesions caused lung lesion like congestion and hemosiderosis. In cases like Streptococcal pneumonia, contrary to the expectation, there were no extension of inflammation to the cardiac chambers. There are lot of reports on lung lesions caused by air pollutants. In the current study, lesions like anthracosis and epithelial cell proliferation, which could be attributed to air pollution, were frequently encountered. This finding is a pointer to the need for thorough investigation on the various air pollutants present in different localities and its effect on the cardiopulmonary system can be taken as a model for human diseases.

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Two tumors were encountered in the present investigation. One case of lymphosarcoma and an osteosarcoma. Both the tumors were found in Doberman Pincher dogs.

In the case of osteosarcoma a large tumor mass was found in the thoracic cavity extending to the subcutaneous through the right intercostal muscle. The tumor might have originated from the rib. The histological lesions were in accordance with the discription of Ashley (1978) for the osteosarcoma. Though the heart was not affected with tumor it showed biventricular dilatation. Endarteritis obliterans was evident in the pulmonary arteriole. The compression pressure from the tumor mass might have caused increased pulmonary pressure and heart failure.

In retrospective study one case of osteosarcoma was found in the lung of a German Shepherd dog. Forrester and Moor (1997) reported that more than 90 per cent of osteosarcoma were found in dogs weighing 20 kg and this correlates with the observation of both the tumor cases in large breeds in the present study. One case of tumor was found in male and other in female. No sex predilection can be made from the cases. The animal with osteosarcoma was eight years old. This is in agreement with the findings of Forrester and Moor (1997) that osteosarcoma was most frequent in older dogs with a median age of seven years.

The lymphosarcoma was found in a two-year-old Doberman Pincher dog. From the severity of hepatic lesions, the liver appeared to be the primary site of tumorogenesis. The tumor nodules were also present in the lung, heart, kidney and sub scapular lymph nodes. The histological observation of diffuse small lymphocytic type cells correlated with the description of Carter *et al.* (1986) in histological classification of canine lymphomas.

In this investigation, all the dogs revealed cardio-pulmonary lesions of varying intensities. It was further revealed that lesions in the lung, affected heart and vice versa in most of the cases. The conditions like tumor, which increases

the pulmonary pressure, caused the cardiac failure. Moreover, cardiac lesions caused lung lesion like congestion and hemosiderosis. In cases like Streptococcal pneumonia, contrary to the expectation, there were no extension of inflammation to the cardiac chambers. There are lot of reports on lung lesions caused by air pollutants. In the current study, lesions like anthracosis and epithelial cell proliferation, which could be attributed to air pollution, were frequently encountered. This finding is a pointer to the need for thorough investigation on the various air pollutants present in different localities and its effect on the cardiopulmonary system can be taken as a model for human diseases.

Summary

6. SUMMARY

An investigation was carried out to study the prevalence and pathology of cardio- pulmonary disorders in dogs. The prevalence of cardiac and pulmonary disorders for the five years was found to be 47.05 per cent and 83.38 per cent respectively by analysis of the data collected from the autopsy records maintained at the Centre of Excellence in Pathology, Mannuthy. Forty three per cent cases showed lesions in both heart and lungs.

Seventy eight samples of canine heart and lungs collected from the carcasses brought for autopsy at the Centre of Excellence in Pathology, Mannuthy were used for the study. The samples collected were subjected to detailed gross as well as histopathological examination. The pathological lesions observed were classified and recorded. Each lesion was explicated giving possible etiopathogenesis. The study revealed a high prevalence of both heart and lung lesions (98.7 per cent each). The major cardiac lesions were endocardial haemorrhage (25.97 per cent), left ventricular hypertrophy (15.58 per cent), biventricular dilatation (13.17 per cent) and epicardial petechiae (11.69 per cent). The other lesions observed were hydropericardium, cardiac tamponade, mitral valve haemorrhage, myocardial infarction, myocarditis, endocarditis, endocardial fibroelastosis, cartilaginous metaplasia of the endocardium, valvular blood cyst and lymphosarcoma.

Pulmonary lesions were congestion (70.51 per cent), haemorrhage (32.47 per cent), emphysema (49.35 per cent), edema (31.17 per cent), collapse (7.79 per cent), pneumonia (10.39 per cent) and anthracosis (16.67 per cent). The other lesions were pleural adhesion, subpleural mineral deposition, osseous tissue formation, infarction, bronchial epithelial cell desquamation and proliferation, bronchiolar obliteration, hyalinisation, osteosarcoma and lymphosarcoma.

Dilated ventricles revealed thin, atrophic and widely separated muscle fibres with wavy appearance, while the hypertrophic cases revealed swollen and crowded muscle fibres, histologically. Fragmentation of the muscle fibres, fibrous tissue proliferation, vacuolation and infiltration of inflammatory cells were the other histological features observed along with the cardiomyopathy. This could be attributed to nutritional, hormonal or infectious aetiology. Congestion and edema were the frequently observed pulmonary changes along with cardiomyopathies. The cardiomyopathies may be the primary cause for the above pulmonary lesions.

Valvular blood cyst was observed in one case. This could be due to stress induced dilatation of blood and lymph vessels in the valve.

In one case, focal valvular endocarditis was noticed. Infiltration of mononuclear cells was seen in the lungs along with other visceral organs indicative of septicaemia with subsequent localisation.

Myocardial infarct was present in a few cases, which were confirmed by gross staining with Nitro-BT. Infarction was confined to the left ventricle and interventricular septum. The presence of coronary arteriosclerosis observed in some of the infarcted cases substantiates the view that arteriosclerosis might lead to myocardial infarction. Infectious conditions could be a predisposing cause as evidenced by confirmation of leptospirosis in a case of infarction.

Pulmonary congestion, edema and haemorrhage showed high prevalence in this study. These lesions were closely associated with cardiac disorders like cardiomyopathies.

Pneumonia was found in eight cases. The severity of pneumonia was prominent in the middle lobes. Histological examination revealed septal cell proliferation, infiltration of mononuclear inflammatory cells and alveolar macrophages. Alveolar lumen was filled with fibrin in some cases. Pleural thickening by fibroplasia was demonstrated by Gomori's trichrome stain.

Haemolytic Streptococci were isolated from one such case. Damage to both bronchioles and alveoli in most of the pneumonic cases pointed to the chance of viral infection and possible secondary bacterial infection.

Osseous tissue formation in the interstitium of the lungs was evident in three cases. High incidence of biventricular dilatation in such cases indicates the cardiac failure and subsequent pulmonary hypertension and ossification. Presence of mononuclear inflammatory cells in one case, also pointed to the possibility of chronic inflammation and ossification.

Subpleural mineral deposition found in aged animals might be due to chronic exposure to air pollutants. Any correlation with disease condition could not be observed.

Anthracosis found in aged animals indicated prolonged exposure to air pollutants.

Arteriosclerosis was evident in the pulmonary and coronary arteries in many cases. Endarteritis obliterans observed in the lungs might be caused by pressure from tumour tissue in one case. Thickening of the coronary arteries along with vacuolar changes was confirmed by Mallory's Phosphotungstic acid - haematoxylin stain. Thickening and narrowing of the coronary arteries resulted in cardiac infarction in some cases.

Lymphosarcoma was detected in the heart and lungs in one case. The tumour might have metastasised from the liver or could be of multicentric origin. The tumour was of diffuse small leucocyte type.

Osteosarcoma was seen in the lung of an eight year old Doberman Pinscher. This tumour was metastasised from the ribs. Histological sections revealed spindle shaped cells, osteoclasts and poorly formed osseous tissue. Osteosarcoma was also recorded in a German Shepherd dog in the retrospective study. This indicated high prevalence of osteosarcoma in large breeds.

The systematic investigation undertaken has helped to focus attention on he prevalence of various cardiac and pulmonary disorders in dogs. The nyocardial as well as pulmonary disorders of the dogs observed in this study rovide many unique opportunities to explore diverse aspects of cardio-ulmonary medicine. Some of these diseases correspond closely to conditions nown to affect humans. Others constitute model systems for monitoring the nvironmental status. Therefore, the findings of the current investigation can act s basis for future studies, which can throw light on pathomechanisms of cardio-ulmonary disorders.

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PATHOLOGY OF CARDIO-PULMONARY DISORDERS IN CANINES

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ABSTRACT OF A THESIS

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ABSTRACT

The present study was undertaken to assess the prevalence and pathology of various cardiac and pulmonary disorders in dogs. The data collected from the autopsy records maintained at the Centre of Excellence in Pathology, Mannuthy and seventy eight samples of heart and lungs from necropsy cases formed the basis of the study. Ninety nine per cent cases showed varying types of cardiac and pulmonary lesions. The highest incidence in the heart was dilated (75.32 per cent) and hypertrophic (15.58 per cent) cardiomyopathies. The other lesions found were hydropericardium, cardiac tamponade, haemorrhage, myocarditis, endocarditis, endocardial fibroelastosis, valvular blood cyst coronary arteriosclerosis and mural thrombus. Myocardial infarction was found to be confined to the left ventricles and interventricular septum in all the encountered cases (5.19 per cent). Pulmonary congestion (70.51 per cent), haemorrhage (32.47 per cent) and edema (31.17 per cent) were observed with higher incidence rate. The other lesions found in the lungs were bronchitis, emphysema, pneumonia, collapse, infarction, mineralization, ossification, anthracosis and endarteritis obliterans. Metastatic osteosarcoma in the lungs, metastatic lymphosarcoma in the heart and lungs were also observed. The incidence of cardio-pulmonary disorders encountered was relatively high. The need and scope for investigation into the pathological disorders of the heart and lungs of canines were highlighted.