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**PATHOLOGY OF CARDIAC DISORDERS
IN CATTLE**



**By
BISI T V**

THESIS

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requirement for the degree of**

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**Centre of Excellence in Pathology
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2002**

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I hereby declare that this thesis entitled **"PATHOLOGY OF CARDIAC DISORDERS IN CATTLE"** 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

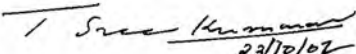
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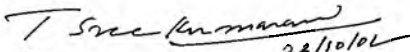

Dr. T. Sreekumaran
23/10/02
(Chairman Advisory Committee)

Professor
Centre of Excellence in Pathology
College of Veterinary and
Animal Sciences Mannuthy


Mannuthy


CERTIFICATE


We the undersigned members of the Advisory Committee of **Smt Bisi, TV** a candidate for the degree of **Master of Veterinary Science in Pathology** agree that the thesis entitled **"PATHOLOGY OF CARDIAC DISORDERS IN CATTLE"** may be submitted by **Smt Bisi TV** in partial fulfilment of the requirement for the degree

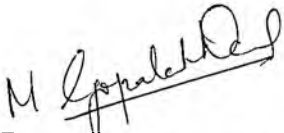

Dr. T. Sreekumaran 23/10/02

Professor
(Chairman Advisory Committee)
Centre of Excellence in Pathology
College of Veterinary and Animal Sciences Mannuthy


Dr. K.V. Valsala
Professor and Head
Centre of Excellence in Pathology
(Member)


Dr. N. Vijayan
Associate Professor
Centre of Excellence in Pathology
(Member)


Dr. N. Ashok
Assistant Professor
Department of Anatomy
(Member)


EXTERNAL EXAMINER
Dr. M. Gopalakrishnan
Nair
Assoc Professor
Dept of Pathology
Rajiv Gandhi College
of Veterinary and
Animal Sciences (Pondicherry)

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Introduction

1 INTRODUCTION

The cardiovascular system with the heart as its epicentre assists the body in maintaining life and health by circulating the blood through the lungs and tissues adequate for respiration and nutrition. The heart has an inherent capacity to withstand the stress and strain which the changing body conditions impose upon it. The strong and elastic muscular walls, the perfectly seated cardiac valves and the efficient pacemaker mechanisms

work in harmony to maintain the functional needs of the organ under all circumstances. Another important characteristic of the heart musculature is its ability to adapt itself to varying physiological needs and pathological conditions by compensation and the failure of it leads to decompensation. To meet the increased demand the heart musculature undergoes hypertrophy as a compensatory mechanism. Decompensation is gradual and results in dilatation of the ventricles.

In vertebrates the cardiac muscle is capable of generating action potential without any nervous stimulation. This type of heart is called "myogenic heart". The action potential developed in one myocyte spreads to all other myocytes that are joined together by gap junctions and they become excited and contract

as a single functional syncytium. There is little or no regenerative capacity for the cardiac muscle fibres after injury or destruction. Healing is accomplished by scar formation. Likewise, in young ones, growth of the heart is accomplished by an increase in the size of the fibres and not by an increase in the number of the cells.

Investigations on cardiomyopathies had begun long back in human medicine. Previously it was described under different terminologies like "primary myocardial disease", "cardiomyopathy" and "myocardiopathy". Extensive studies have been conducted in this field in human beings and in companion animals. Pathology of cardiac valves was an area of great interest and now the transplantation of cardiac valves with prosthetic materials has become a common practice in canines. In many of the experimental studies in human cardiology, the swine heart is used as a paradigm. Owing to the difficulties encountered in physical and clinical examinations and in diagnostic methods like electrocardiography and ultrasound scanning in large animals, large animal cardiology is a poorly attended field of investigation. Literature regarding the clinical diagnosis of cardiac disorders in large animals is scarce. Here lies the

significance of postmortem diagnosis of cardiac problems in large animals

Bovine cardiomyopathies have been reported from different parts of the world and extensively discussed in literature. The major etiological factors attributed to cardiac lesions in bovines are infectious agents like virus, pathogenic bacteria, protozoan parasites and toxic as well as nutritional factors. The deficiency of trace elements particularly Selenium and Vitamin E produce cardiomyopathies in animals.

Cardiac cachexia, the muscle wasting associated with congestive heart failure, is present in more than 50 per cent of dogs affected with dilated cardiomyopathy (Roudebush and Freeman, 2000). Obesity can also lead to cardiovascular disorders as there is an increase in the plasma and extra cellular fluid volume and the cardiac output will be elevated. There will be an increase in the neurohormonal activation, increased heart rate, abnormal systolic and diastolic ventricular functions, exercise intolerance and elevated blood pressure. At this juncture, the compensatory mechanisms set in and as a consequence of all these changes, the heart undergoes hypertrophy and/or dilatation. Excessive feeding and lack of proper exercise in farm

conditions may produce obesity in cattle and may predispose the heart to diseases

So far no systematic study has been made on bovine cardio pathology in India and much data are not available regarding the nature and prevalence of cardiac lesions in cattle. Since a wide variety of cardiac disorders have been reported in small animals especially in dogs the chances of getting similar conditions in cattle cannot be over looked. The present study therefore was designed to delve on the prevalence and pathology of cardiac disorders in cattle and to make a detailed study of the gross and histopathological lesions.

Review of Literature

2 REVIEW OF LITERATURE

2.1 Congenital anomalies

Silva Krott and Wilkinson (1991) reported a case of hypoplastic left ventricle along with aortic atresia in a two day old female Holstein calf. The foramen ovale was patent. The heart revealed severe dilatation and hypertrophy of the myocardium. In addition, the ductus arteriosus was patent.

Congenital aortic valvular dysplasia was reported by Watson *et al* (1991) in a heifer. There was severe enlargement of the left atrium with ventricular dilatation and hypertrophy. The aortic valve had an increase in the circumference, the dorsal cusp was incomplete and the borders were thickened.

Besser and Knowlen (1992) recorded the occurrence of ventricular septal defect (VSD) in two co-twin cows. The heart revealed a VSD of 3 cm diameter in the upper part of the septum. Hypertrophy of both the ventricles, endocardial fibrosis and vegetative endocarditis were also noticed. Myocardial necrosis was observed in one of them.

McLennan and Sutton (1993) reported co-occurrence of VSD and atrio-ventricular valvular anomaly in a heifer. There was transposition of a part of the support structures of the septal

cusp into the left ventricle as well as an additional cusp that inserted into the rim of the defect

Three cases of bovine extreme tetralogy of Fallot were reported by Nakade *et al* (1993) All the intrapericardial pulmonary arteries were completely absent in one of the calves In the other two cases there was an atretic pulmonary trunk with a bicuspid valve All the three cases revealed ventricular septal defect also

Robinson and Maxie (1993) reported that congenital haematomas on the margin of atrioventricular valves were common in calves

Reppas *et al* (1996) observed an unusual congenital cardiac anomaly in a Dexter calf characterised by a persistent truncus arteriosus overlying a 2cm VSD The calf also had an aneurysmal rudiment of the lower aortico pulmonary trunk arising from the right ventricle

A case of total ectopia cordis in a calf was reported by Eroksuz *et al* (1998) The heart was completely extruded from the chest cavity and the pericardium was adherent to the sternum There was also epicardial ecchymosis along with minute papillary folds of the pericardium on the posterior surface of the heart

2 2 Cardiomyopathies

2 2 1 Genetic predisposition

Watanabe *et al* (1979) reported the occurrence of an autosomal recessive lethal gene responsible for the causation of idiopathic cardiomyopathy

Cook (1981) and later Morrow and McOrist (1984) demonstrated the association of primary cardiomyopathy in Poll Hereford cattle with curly hair coat and suggested a simple autosomal recessive mode of inheritance

Baird *et al* (1986) observed an inherited predisposition to dilated cardiomyopathy in Holsteins

Association of an autosomal recessive allele in the causation of bovine dilated cardiomyopathy (BDCMP) was supported by Dolf *et al* (1998) based on a pedigree analysis of a herd comprising of 75 cattle with dilated cardiomyopathy

2 2 2 Hypertrophic cardiomyopathy

Alexander *et al* (1960) observed right ventricular hypertrophy along with pulmonary hypertension in cattle reared at high altitude in an experiment conducted to study the effect of residence at high altitude upon the pulmonary arterial pressure

of cattle and to determine the relationship of pulmonary arterial pressure to ventricular mass

Danzl (1995) reported 47 cases of incurable heart disease in cross bred cattle during a period of 10 years (1985-1994). The heart was enlarged with hypertrophy and fibrosis of the myocardium.

Machida *et al* (1996) recorded two cases of hypertrophic cardiomyopathy in Holstein cattle. Microscopically there was markedly disarrayed cardiac myocytes which were extremely hypertrophied and contained large hyperchromatic and bizarre shaped nuclei.

2.2.3 Nutritional cardiomyopathy

Sykes and Moore (1942) reported that rations low in potassium when fed to calves produced degenerative changes in the Purkinje fibres of the heart characterised by vacuolation and granular appearance of the fibres.

Van Vleet and Ferrans (1986) reported myocardial necrosis in calves deficient in vitamin E and Selenium. They further reported that in Copper and Iron deficiency the heart was atrophic, pale and flabby with extensive myocardial fibrosis.

Clinical evidence of cardio pathological alterations were noticed by Osame *et al* (1989) in calves suffering from Selenium and vitamin E responsive white muscle disease. There was an increase in the activities of serum GOT, CPK and LDH and the ECG revealed sinus tachycardia.

Kennedy and Rice (1992) conducted an experimental study to evaluate the effect of diets containing polyunsaturated fatty acids along with a low level of vitamin E and selenium on the myocardium in calves. The myocardial alterations comprised of multifocal or diffuse cardiocyte degeneration and necrosis. Ultrastructurally damaged cardiocytes showed lysed contractile material, vacuolated sarcoplasm, altered mitochondria, sarcoplasmic myelin figures, lipofuscin granules and multiple nuclei.

Orr and Blakley (1997) recorded that selenium deficiency in bovine fetus caused myocardial necrosis and heart failure characterised by lymphocytic myocarditis and myocardial fibrosis.

2.2.4 Toxic cardiomyopathy

O Hara and Pierce (1974) reported fatal cardiomyopathy in rabbits caused by ground endosperms of *Coffee senna* and the disease was characterised by mitochondrial degeneration, lipid

accumulation myofibrillar degeneration myocytolysis and other reparative changes. The microscopic lesions in the heart of poisoned rabbits resembled those in cattle poisoned with *C. senna*.

Acute toxicity of lasalocid and monensin was studied experimentally in calves by Galitzer *et al* (1986). They observed gross and microscopic lesions consistent with cardiomyopathy. Grossly the lesions included cardiac dilatation^{and} petechial and ecchymotic hemorrhages. Microscopically multifocal areas of necrosis of myocytes were noticed.

Graham *et al* (1988) conducted experimental studies in veal calves for evaluating the toxic effects of dietary zinc. The heart revealed petechiae and myocardial infarcts.

Odrozla *et al* (1991) reported an outbreak of poisoning in Angus bulls by winter forage *Vicia villosa*. Along with the generalised petechiae in the abdominal organs there were pale areas in the ventricular walls of the heart. Microscopically an extensive granulomatous myocarditis characterised by necrosis and eosinophilic infiltration was evident.

Cattle fed *ad libitum* with poultry litter containing residues of ionophore antibiotic maduramicin produced dilated

cardiomyopathy with congestive heart failure (Shlosberg *et al* 1992 and Bastianello *et al* 1995)

Bastianello *et al* (1996) reported chronic cardiomyopathy in cattle due to toxic levels of Salinomycin in the feed. Histologically this was characterised by extensive myocardial fibre atrophy with multifocal hypertrophy and interstitial fibrosis.

Oryan *et al* (1996) conducted an experimental study on oleander poisoning in cattle. Fibromyolysis and infiltration with mononuclear and polymorphonuclear cells in the cardiac muscle fibres, endocardium and epicardium were seen.

Wouters *et al* (1997) reported degenerative cardiomyopathy characterised by myocardial fibrosis in narasin poisoning in cattle.

2 2 5 Myocardial degeneration

Bradley and Duffell (1982) recorded the occurrence of Xanthosis (Brown atrophy) in the heart muscles of cattle in the age group 4-13 years. The heart was uniformly pigmented brown. Microscopically the pigment granules could be demonstrated both in the cardiac muscle cells and in Purkinje cells.

Van Vleet and Ferrans (1986) described myocardial lipofuscinosis in aged or cachectic animals characterised by auto fluorescent pigment granules in the myocytes

Machida *et al* (1991) reported a case of myocardial infarction secondary to a disseminated coagulopathy in a cow. The lesions in the heart were concentric hypertrophy of the left ventricle with a large area of pale yellowish discoloration in the anterior aspect. Microscopically the myocardium revealed areas of acute necrosis characterised by hyalinisation of the fibres.

2 2 6 Non specific cardiomyopathies

Goodwin *et al* (1961) described cardiomyopathy as sub acute or chronic disorder of the heart muscle of unknown or obscure etiology often endocardial or sometimes with pericardial involvement but not atherosclerotic in origin.

Ishikawa *et al* (1984) described histopathological lesions in idiopathic cardiomyopathy which included severe vacuolation, hyaline degeneration and nuclear deformation in myocardial fibres including the Purkinje fibres.

Idiopathic congestive cardiomyopathy with congestive heart failure and myocardial degeneration in dry cows and steers were reported by Ishikawa *et al* (1985).

Martig and Tschudi (1985) studied cardiomyopathy and associated congestive heart failure and pulmonary hypertension in cattle. There was an inherited predisposition to cardiomyopathy triggered by an exogenous factor possibly a feed toxin.

McLennan and Kelly (1990) described the gross and histopathological appearance of heart in dilated (congestive) cardiomyopathy in a Friesian heifer. The heart was enlarged with dilatation of both the ventricles. Hypertrophy of the myocardial fibres with central vacuolation was observed histologically.

Graber *et al* (1995) reported cardiomyopathy in Holstein breeds and was clinically characterised by signs of congestive heart failure.

2.3 Cardiac lesions of infectious etiology

The macroscopic appearance of myocarditis in FMD virus infected calves was characterised by a swollen heart muscle with whitish streaks and foci described as the "tiger heart" (Jennings 1970). Microscopically the initial reaction was an interstitial myocarditis followed by hyaline degeneration of the fibres and cellular infiltration around the conducting bundle.

Power and Rebhun (1983) reviewed 31 cases of bacterial endocarditis in adult cattle. Out of these 31 cases 20 cases were

diagnosed at autopsy *Corynebacterium pyogenes* was the most common causative agent. The right atrioventricular valve was greatly affected (12 cases) and the left atrioventricular valve to a lesser degree (9).

Rajan *et al* (1983) recorded the incidence of sarcocystis in the bovine heart. Out of the 556 hearts examined 47 (16%) were positive for sarcocystis and they were found both in cardiac muscle fibres and Purkinje cells.

Neonatal calves affected with FMD virus had "tiger heart" appearance. Microscopically lesions were characterised by lymphocytic myocarditis with hyaline degeneration and neutrophilic infiltration (Van Vleet and Ferrans 1986 and Radostits *et al* 2000).

Carcasses of 13 Holstein cows died of peripartum complications were examined by Yamagishi *et al* (1995) and they observed myocardial necrosis accompanied by neutrophilic and mononuclear cellular infiltrates with interstitial fibrosis.

Shehab *et al* (1996) observed myocarditis in calves affected with IBR and Rota virus infections.

Zaid (1996) observed hydropericardium, petechial hemorrhages on the pericardium and congestion of the heart in cattle affected with respiratory bacterial infections.

Disseminated focal to extensive and diffuse bleeding in the heart was reported by Heckert and Appel (1997) in cattle affected with Bovine Diarrhoea virus infection

Wouda *et al* (1997) recorded myocarditis in the bovine foetus affected with neosporosis with a higher incidence of tachyzoites in the heart of younger foetuses

Radostits *et al* (2000) reported that parasitic myocarditis occurred in neonatal calves due to cysticercosis migration of strongyle larva sarcocystis and neospora

2 4 Pathology of the cardiac valves

Davies (1980) explained the conditions of infective valvular endocarditis and non bacterial thrombotic endocarditis in cardiac valves The lesions in the non bacterial thrombotic endocarditis were characterised by thrombus vegetation and absence of infectious organisms

Silva Krott and Wilkinson (1991) recorded a hemocyst of one centi metre diameter in the right atrioventricular valve in a Holstein calf died of hypoplastic left ventricle and aortic atresia

Marcato *et al* (1996) reported 5 1% incidence of blood cyst and 5 8% incidence of serous cyst in the atrioventricular valves of cattle out of the 30907 cases examined

2 5 Pathology of the coronary vessels

Neumann *et al* (1970) observed intimal thickening and damaged elastic lamina in the intramural arteries particularly in the ventricular septum in bovine foetuses

Machida *et al* (1991) found large partially organized thrombi in the intra mural coronary arteries adjacent to an area of myocardial infarction in a cow

Machida *et al* (1996) reported thickening of the walls of intra mural coronary arteries and a decrease in the luminal size in and around the areas of replacement fibrosis in the ventricular walls. The increase in wall thickness was due to proliferation of smooth muscle and fibrous tissue in the intima

2 6 Neoplasia

Jennings (1970) reported the right auricle of the bovine heart as a predilection site for lymphosarcoma and other neoplasms and attributed this to the loose sub epicardial connective tissue and a potential for hematopoiesis at this site

Ivany and Illanes(1999) recorded a case of congestive heart failure in a Holstein cow due to epicardial lymphosarcoma. The heart was flabby covered partially with irregular sized polypoid or papillary projections of vascularised tissue attached to the

ventricular epicardium Microscopically the epicardium revealed papillary projections of hyperplastic mesothelium heavily infiltrated by solid sheets of anaplastic lymphoid cells admixed with neutrophils macrophages and red blood cells

2 7 Miscellaneous conditions

Bretzinger (1974) observed endothelial and sub endothelial calcification in the ventricle and auricle in the bovine heart Histologically localised areas of calcium deposition in the elastic fibres of sub endothelial tissue and their disintegration were seen

Baker *et al* (1993) reported incidence of myocardial epithelial inclusions in bovine heart The lesions were single or multifocal The white non capsulated well demarcated masses were within the myocardium with a dominant pattern of simple cuboidal epithelial cells forming acinar and tubular structures

Potter and Besser (1994) enlisted cardiovascular lesions in 10 animals affected with bovine Marfan syndrome as cardiac tamponade secondary to aortic rupture and dissecting aneurysms of the aorta as well as pulmonary artery Cardiac tamponade due to rupture of the pulmonary artery was observed in the fetuses of pregnant animals Microscopically Verhoeff

Van Gieson stained sections of the aorta contained severe fragmentation of the elastic laminae in the aortic media

Guarda *et al* (1996) classified the conduction tissue alterations in the heart as

- (a) primary alterations caused by pathogenic factors which cause damage to both the myocardial and Purkinje cells
- (b) alterations secondary to endocardial and myocardial lesions

The histopathological lesions included atrophy of the Purkinje cells surrounded by cellular infiltration vacuolation of cells along with Zenker's necrosis fragmentation of Purkinje cells and dystrophic calcification

Materials and Methods

3 MATERIALS AND METHODS

The present study was conducted at the Centre of Excellence in Pathology College of Veterinary and Animal Sciences Mannuthy to investigate the prevalence and pathology of cardiac disorders in cattle

3 1 Prevalence study

To assess the prevalence of cardiac disorders in cattle the autopsy records for the past 10years (1991 2000) kept at the Centre of Excellence in pathology College of Veterinary and Animal Sciences Mannuthy were screened The lesions were classified and the prevalence was assessed

3 2 Sample collection

One hundred and one samples of bovine hearts collected at random from the cattle slaughtered at the Meat Technology Unit College of Veterinary and Animal Science Mannuthy and from the cattle slaughtered at the Municipal Slaughter House Thrissur as well as from the carcasses brought for autopsy at the Centre of Excellence in Pathology College of Veterinary and Animal Science Mannuthy were utilized for the study

3 3 Morphometry

3 3 1 Relative weight of the heart

The body weight of the carcass and the weight of the heart were recorded. The relative weight of the heart was determined by the method used by Robinson and Maxie (1993)

3 3 2 Measurements of the heart

Following morphometrical parameters for individual heart were recorded as per the method described by Malik *et al* (1972)

- Total length from the base to the apex of the heart
- total circumference at the coronary groove region

After dissecting the heart transversely at the coronary groove region the following parameters were measured

- wall thickness of the left ventricle
- wall thickness of the right ventricle
- thickness of the interventricular septum
- cross sectional diameter at the coronary groove region

The data collected were analysed statistically using ANACOVA Student T test and Regression analysis (Pense and Sukhatme 1978)

3 4 Gross examination

The heart was subjected to detailed ^{gross} examination. The pericardium was examined and dissected out. The coronary fat was dissected and the coronary arteries were traced out, dissected and examined for gross lesions on the surface and in the lumen. The organ was examined in detail especially for congenital anomalies, hypertrophy, dilatation, epicardial and endocardial hemorrhages, myocardial degeneration and for any inflammatory conditions. The cardiac valves were examined for the presence of thickening, blood or serous cysts.

3 5 Histopathology

Representative samples of tissues from the pericardium, right and left ventricles, right and left auricles, cardiac valves and coronary vessels were collected and preserved in 10% Neutral Buffered Formalin. The tissues were processed by routine paraffin embedding techniques (Sheehan and Hrapchak 1980). Sections were cut at 4 micron thickness and stained with routine Haematoxylin and Eosin stain (Bancroft and Cook 1995) for histopathological studies. Special stains like Mallory's

phosphotungstic acid haematoxylin stain Gomori's Aldehyde Fuchsin and Van Gieson's technique for collagen fibres (Luna 1968) and Masson's Trichrome stain Verhoeff Van Gieson's technique Alizarin red and Oil Red O staining methods (Sheehan and Hrapchak 1980) were used wherever required. The stained sections were subjected to detailed examination under the light microscope and the lesions were classified.

Results

4 RESULTS

4.1 Prevalence study

During the past 10 year period (from 1991 to 2000) a total number of 498 bovine carcasses of different age groups were examined at the Centre of Excellence in Pathology College of Veterinary and Animal Sciences Mannuthy. Out of the 498 cases examined 172 carcasses ^(35 per cent) showed various cardiac lesions. The lesions were classified and the prevalence was assessed (Table 1). The highest incidence was haemorrhages of varying degrees (40 per cent) and the least incidence was congenital cardiac anomaly of inter atrial septal defect (0.6 per cent). The other lesions recorded were ventricular hypertrophy (32 per cent), inflammatory or degenerative conditions (29 per cent), haemopericardium (22 per cent), cardiac dilatation (19 per cent), hydropericardium (6 per cent) and neoplasia of the heart (2 per cent).

4.2 Relative weight of the heart

The weight of the animal and the heart weight were determined and the relative weight of the organ was calculated. The data recorded for ten animals having ventricular hypertrophy are presented in Table 2. The highest weight of the

Table 1 Prevalence study

No	Year	Total no of bovine cases	No of cases with cardiac lesions	Hyper trophy	Dilatation	Haem orrhage	Inflammatory/ degenerat on	Haemo peri card um	Hydro peri card um	Neoplasm	Congenital anomaly (other at al septal defect)
1	1991	56	15 (28)	3 (20)	5 (33)	2 (13)	5 (33)		4 (27)		
2	1992	91	20 (22)	2 (10)	3 (15)		15 (75)		1 (5)		
3	1993	71	17 (24)	1 (5)	7 (41)	7 (41)	4 (24)		2 (12)		
4	1994	68	22 (32)	6 (27)	6 (27)	7 (32)	11 (50)	1 (5)			
5	1995	37	7 (19)	2 (29)	2 (29)	2 (29)	2 (29)	2 (29)		1 (14)	1 (14)
6	1996	64	46 (72)	33 (72)	7 (15)	31 (67)	4 (9)	28 (61)			
7	1997	33	10 (30)	2 (20)		4 (40)		1 (10)			
8	1998	38	18 (47)	5 (28)	2 (11)	7 (39)	6 (33)		3 (17)		
9	1999	19	9 (47)			4 (44)	2 (22)	2 (22)		1 (11)	
10	2000	21	8 (38)	1 (13)	1 (13)	4 (50)	2 (26)		1 (13)	1 (13)	
	Total	498	172 (35)	55 (32)	33 (19)	68 (40)	51 (30)	34 (20)	11 (6)	3 (2)	1 (0.6)

* The values in parenthesis indicate percentage

Table 2 Relative weight of the heart of animals with ventricular hypertrophy

Sl No	Animal weight (kg)	Heart weight (kg)	Relative organ weight (%)
1	473	1 92	0 41
2	227	0 87	0 38
3	265	1 09	0 41
4	296	1 62	0 55
5	530	2 09	0 38
6	422	1 74	0 41
7	342	1 19	0 35
8	520	2 21	0 43
9	242	1 25	0 52
10	296	1 40	0 47
			Mean 0 414 + 0 209

heart was 2.21 Kg for an animal weighing 520 Kg and the lowest heart weight recorded was 0.87 Kg for an animal weighing 227 Kg. The relative weight of the heart ranged from 0.35 per cent to 0.55 per cent with a mean value of 0.414 ± 0.209 .

4.3 Morphometry

The relative weight of the heart, the left ventricular wall thickness, the right ventricular wall thickness, thickness of the inter-ventricular septum, circumference of the heart at the coronary groove region, total length of the heart and the horizontal and the vertical cross-sectional diameter at the groove for adult animals with ventricular hypertrophy, adult animals without ventricular hypertrophy and for calves without hypertrophy taken at random are presented in Tables 3, 4 and 5 respectively. Among the hypertrophic hearts, the total length of the heart was maximum for that with maximum organ weight. The left ventricular wall thickness also showed a proportional increase with the organ weight.

All the parameters were analysed and they were not statistically significant except for the left ventricular wall thickness. The proportional increase in the thickness of the left ventricular wall in hypertrophic hearts was significant ($p < 0.05$).

Table 3 Measurements of the heart of adult animals with ventricular hypertrophy

Sl No	A	B	C	D	E	F	G	H
1	0.38	5.5*	2.8	4.4	37.5	24.5	11.7	12.8
2	0.41	5.5*	2.5	4.0	36.8	20.2	11.2	12.2
3	0.35	4.5*	2.8	3.7	32.0	18.3	9.5	10.7
4	0.43	5.5*	3.0	4.3	40.0	24.5	12.0	14.0
5	0.52	2.5*	1.0	2.0	36.0	18.0	12.3	13.5
6	0.47	2.3*	1.0	2.0	25.0	13.3	8.7	10.8

* Statistically significant ($P < 0.05$)

Table 4 Measurements of the heart of adult animals without ventricular hypertrophy

Sl No	A	B	C	D	E	F	G	H
1	0.41	3.5	2.5	3.5	36.5	19.0	11.0	14.5
2	0.38	2.5	0.8	1.5	29.5	17.0	11.0	12.5
3	0.41	3.0	1.0	2.5	30.0	20.0	12.0	14.0
4	0.36	2.5	1.5	2.3	24.0	16.0	8.0	10.5
5	0.55	2.0	0.8	2.3	25.0	14.5	9.0	13.0
6	0.30	3.3	1.8	3.0	20.8	15.0	6.8	8.3

Table 5 Measurements of the heart of calves

Sl No	A	B	C	D	E	F	G	H
1	0.45	2.0	0.6	1.3	13.2	10.9	5.8	6.6
2	0.45	1.7	0.9	1.4	11.8	9.5	5.3	6.2
3	0.45	2.0	0.8	1.5	12.6	10.0	4.5	5.0
4	0.43	1.8	0.6	1.5	12.8	10.0	6.0	6.5
5	0.41	2.0	0.3	1.4	13.0	11.0	6.0	6.5
6	0.43	2.5	1.5	1.8	15.5	9.6	6.0	8.5

- A Relative weight of the heart in percentage
 B Thickness of left ventricular wall in cm
 C Thickness of right ventricular wall in cm
 D Thickness of interventricular septum in cm
 E Circumference of the heart at the coronary groove in cm
 F Length of the heart in cm
 G Horizontal cross sectional diameter at the coronary groove in cm
 H Vertical cross sectional diameter at the coronary groove in cm

4 4 Gross pathology

Out of the 101 hearts examined, 56 hearts (55 per cent) revealed gross pathological lesions. Classification of the lesions based on the age and the sex is given in Table 6 and Table 7 respectively. Higher incidence of cardiac disorders was observed in animals of age between six months and three years. Females (57 per cent) were affected more than the males (50 per cent). The lesions were also classified based on the source from which they were collected (Table 8) and the incidence of cardiac lesions was higher in animals died due to various disease conditions. The prevalence of each lesion is illustrated in Graph 1.

4 4 1 Haemorrhage

4 4 1 1 Petechial haemorrhage

Out of the 55 hearts with cardiac lesions, petechial haemorrhages were observed in 11 cases (19.6 per cent). Small pin point accumulations of blood were seen in the epicardium of the ventricles and atria as well as in the ventricular endocardium. In seven cases, haemorrhagic foci were found in the epicardial fat around the coronary groove. Three cases revealed diffuse areas of pinpoint haemorrhages throughout the organ. A focus of petechial haemorrhage was observed in the

Table 6 Classification of lesions based on the age

Age Group	Total number of cases	Cases with gross cardiac lesions	
		Number	Percentage
0 - 6 months	29	17	59
6 months to 3 years	6	5	83
> 3 years	66	34	52

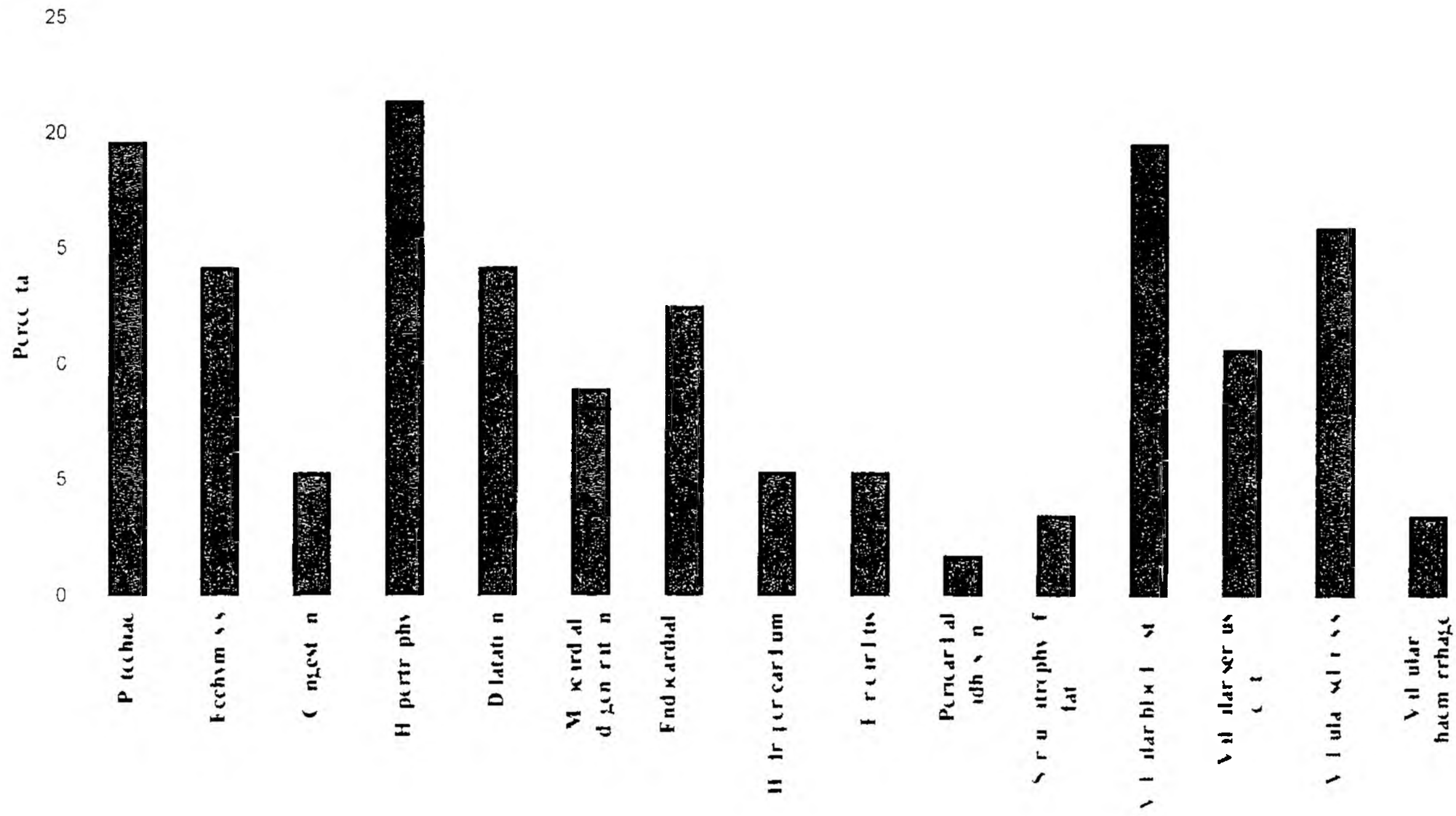
Table 7 Classification of lesions based on the sex

	Total number of cases	Cases with gross cardiac lesions	
		Number	Percentage
Male	24	12	50
Female	77	44	57

Table 8 Classification of lesions based on the source of samples obtained

	Total number of cases	Cases with gross cardiac lesions	
		Number	Percentage
From slaughter house	72	33	46
From carcasses brought for autopsy	29	23	79

Graph 1 Prevalence of cardiac lesions



endocardium of the left ventricle at the base of the papillary muscle in one case(Fig1)

4 4 1 2 Ecchymosis

Extensive areas of haemorrhage were observed in eight cases (14.2 per cent) out of which six hearts revealed endocardial ecchymosis in the left and the right ventricles. In two cases there were also epicardial haemorrhage in the left ventricle.

4 4 2 Congestion

Congestion of the coronary vessels was noticed in 3 cases (5.3 per cent). Both the extra mural and the intra mural arteries and veins were engorged with blood giving the heart a reddish discoloration.

4 4 3 Ventricular hypertrophy

Ventricular hypertrophy of varying intensity was observed in 12 cases (21.4 per cent). The heart was enlarged due to the increased ventricular musculature especially that of the left ventricle. The lumen of the ventricle was reduced considerably and in one case the right ventricular lumen was completely occluded (Fig 2). The ventricular walls were thickened, firm and rubber like. The average weight of the organ was 1.65 kg and the average thickness of the left ventricular wall was 4.3 cm. The

relative cardiac weight was higher for hearts with ventricular hypertrophy when compared to that of the normal hearts

4 4 4 Cardiac dilatation

Cardiac dilatation was observed in eight cases (14.2 per cent). The right ventricular wall was thin and flabby. The luminal size was either normal or increased. The apex of the heart was rounded giving the heart a globular appearance (Fig 3).

4 4 5 Degeneration

In five cases (8.9 per cent) the myocardium of the heart revealed focal areas of yellowish white discoloration and fish flesh appearance. The focal areas of degeneration were observed at the apex region in one case, middle parts of the left ventricular free wall in three cases and near the coronary groove in the right ventricular free wall in one case.

4 4 6 Endocardial fibroelastosis

Endocardial fibroelastosis was noticed in seven cases (12.5 per cent). The endocardium was thickened in both the ventricles and atria and it had a glistening white appearance. There was diffuse thickening of the endocardium throughout the right atrium in three cases. In two cases there were focal areas of endocardial thickening in the left ventricle and in one case

endocardial thickening was noticed in the right ventricle in a focal area (Fig 4)

4 4 7 Hydropericardium

In three cases (5 3 per cent) the pericardial sac was distended and filled with straw coloured clear fluid measuring approximately 20 to 30 ml

4 4 8 Serous pericarditis

In three cases (5 3 per cent) the pericardium was affected with serous pericarditis The glistening appearance of the pericardium was lost and the pericardium appeared opaque Focal deposition of pale yellow coloured fine fibrin like material was also observed

4 4 9 Pericardial adhesion

In one case (1 7 per cent) the pericardium was adherent to the thoracic wall at the apex The external surface of the pericardium was haemorrhagic and rough due to the deposition of fibrin

4 4 10 Serous atrophy of the epicardial fat

The adipose tissue deposited at the epicardium especially around the coronary groove revealed serous atrophy in two emaciated calves (3 5 per cent) The fat was replaced by oedema

fluid which appeared as yellowish brown jelly like material (Fig 1)

4 4 11 Cystic valves

4 4 11 1 Blood cyst

Sessile round or oval cysts filled with blood protruding above the atrial surface of the leaflets of the atrio ventricular valves were seen in 11 cases (19.6 per cent). The cysts were observed both in calves and adult animals but higher incidence was noticed in young animals. The size of the cyst varied from 1mm to 5mm. The colour of the cysts ranged between light red to greenish black (Fig 5). The cysts occurred in singles as well as in multiples.

4 4 11 2 Serous cyst

Larger sized (3-10 mm) single serous cysts were observed in the atrio ventricular valves in six cases (10.7 per cent). The cysts were filled with straw coloured clear fluid.

4 4 12 Valvular thickening

Thickening of the valvular cusps was noticed in nine cases (16 per cent). The thickening was of nodular type in the aortic valves whereas in the atrio ventricular valves it was diffuse (Fig 6).

4 4 13 Valvular haemorrhage

Diffuse haemorrhage in the atrio ventricular valvular leaflets was observed in two cases (3.5 per cent)

4 5 Histopathology

4 5 1 Pericardium

4 5 1 1 Haemorrhage

Pericardial haemorrhage was recorded in two per cent cases. There were accumulations of erythrocytes in focal and diffuse areas. Oedema fluid was observed as homogeneously pink stained areas adjacent to the area of haemorrhage.

4 5 1 2 Pericarditis

There was thickening of the pericardium due to infiltration with mononuclear phagocytes and lymphocytes. A mild degree of fibrinous exudation was also observed in one case.

4 5 1 3 Lymphoid hyperplasia

Nodular lymphoid accumulations encapsulated by fibrous tissue were noticed in one case in a black cattle. In addition, loosely scattered lymphoid accumulation was seen throughout the pericardium (Fig 7).

4 5 1 4 Fibrosis

The non inflammatory thickening of the pericardium was observed in one case There was proliferation of the pericardial fibrous tissue with a predominance of collagen fibres which on PTAH staining appeared reddish purple

4 5 2 Epicardium

4 5 2 1 Haemorrhage

Epicardial haemorrhage was recorded in five per cent cases Extravasated erythrocytes were seen as focal or diffuse collections in the subepicardial adipose tissue and between the connective tissue layers of the epicardium In one case there was severe haemorrhage in the epicardium extending into the myocardium

4 5 2 2 Fatty infiltration

Excessive amount of adipose tissue in the epicardium was observed in 10 per cent cases There was infiltration of the subepicardial space with fat vacuoles seen extending even into the myocardium in one case Ganglionic plexus were observed in the subepicardial space (Fig 8) The fat droplets appeared as empty vacuoles in the Haematoxylin Eosin stained sections and as red droplets in Oil Red O stained sections

4.5.2.3 Fibrous tissue proliferation

Focal thickening of the epicardium was recorded in five per cent of the cases. There was proliferation of the epicardial collagen fibres which appeared purple red in colour in Van Gieson Picric acid stained sections (Fig 9)

4.5.2.4 Lymphoid hyperplasia

Accumulation of lymphoid cells in the epicardium was recorded in one case. There were focal and diffuse collections of lymphocytes in the epicardium. Some of the lymphoid nodules were encapsulated by fibrous tissue. Focal accumulation of oedema fluid was also observed (Fig 10a, 10b)

4.5.2.5 Serous atrophy of the epicardial fat

Serous atrophy of the epicardial fat was recorded in one case. There was homogeneously pink stained serous fluid seen in between the fat vacuoles subepicardially. Erythrocytes were also seen scattered in the interstitial space along with the oedema fluid (Fig 11)

4 5 3 Myocardium

4 5 3 1 Vascular changes

4 5 3 1 1 Congestion

Out of the 101 hearts screened myocardium of 34 hearts revealed congestion of varying degree. Based on the intensity it was classified into mild (11 per cent), moderate (19 per cent) and severe (four per cent). Both the extramural and intramural coronary vessels were engorged with erythrocytes in severe cases of congestion (Fig 12). The congested blood vessels were more pronounced in the ventricular myocardium than in the atrial myocardium.

4 5 3 1 2 Haemorrhage

Twenty six per cent hearts revealed haemorrhages of which six per cent had mild haemorrhage, 15 per cent showed moderate haemorrhage and five per cent had severe haemorrhage. The extravasated erythrocytes were seen scattered in between the myocardial fibres and in the connective tissue septum (Fig 13). Focal or diffuse collection of erythrocytes was seen in the perivascular area in one case.

4 5 3 1 3 Oedema

Oedema was observed in 16 per cent cases. It varied from mild (11 per cent) to moderate oedema (five per cent). Faintly pink stained homogenous fluid was observed in the interstitial spaces and in between the myofibrils in H&E stained sections. The muscle fibres were displaced and compactly arranged. The capillaries in the surrounding area were engorged. Intravascular oedema along with venous stasis was observed in three cases.

4 5 3 2 Cardiomyopathies

4 5 3 2 1 Dilatation

Attenuated wavy fibres with widening of the intermuscular spaces were recorded in nine per cent cases. The individual muscle fibres were thin and atrophic. The muscle fibres were widely separated giving a wavy appearance to the muscle bundle (Fig 14). In one case a mild degree of fibrosis was also recorded. These changes were more prominent in the right ventricular myocardium compared to the left ventricle.

4 5 3 2 2 Hypertrophy

Hypertrophy of the muscle fibres was recorded in 13 per cent cases. The muscle fibres were swollen with active nuclear changes. There was hypercellularity with hyperchromatic nuclei.

in three cases. In one case there was disarray of the muscle fibres. The adjacent areas showed crowding of muscle bundles with a reduction in the inter muscular spaces.

4 5 3 3 Myocardial degeneration

4 5 3 3 1 Fatty changes

Fatty changes were recorded in 10 per cent cases. Out of this one case showed extensive changes in the myocardium of the left ventricle. In all other cases there was a moderate degree of fatty changes. Fat droplets appeared in between the muscle fibres as vacuoles. In severe case there were diffuse fat vacuoles replacing the myofibrils (Fig 15). The vacuoles were spherical to oval in shape and of varying size. The fat vacuoles appeared red stained in Oil red O stained sections.

4 5 3 3 2 Hyaline degeneration

Hyaline degeneration was recorded in 12 per cent cases. Focal (eight per cent) and diffuse (four per cent) areas of homogenously pink stained glassy material were present in the muscle bundles (Fig 16a, 16b). The nuclei of the myocytes were seen accumulated in clumps in focal areas.

4 5 3 3 3 Degeneration of the Purkinje fibres

Degenerated Purkinje fibres were observed in 13 per cent cases. The Purkinje fibres appeared swollen and vacuolated. In one case there was a mild degree of infiltration of the mononuclear leucocytes within the fibres (Fig 17)

4 5 3 4 Myocarditis

Focal or diffuse myocarditis was recorded in eight per cent cases. There was infiltration of the myocardium with mononuclear cells (seven per cent) and polymorphonuclear cells (one per cent) along with mild degree of degenerative changes. In one case, there was focal coagulation necrosis of the atrial myocardium along with mononuclear infiltration. The sarcoplasm had lost its structural architecture and it appeared hazy (Fig 18)

4 5 3 5 Sarcocystosis

Myocardial sarcocystosis was recorded in 45 per cent cases. Out of this 40 per cent cases revealed sarcocysts in the muscle fibres (Fig 19) and in five per cent cases they were present both in the muscle fibres and in the Purkinje fibres (Fig 20). The cysts were of varying sizes. The shape of the cyst was oval, spherical or elongated and they contained the merozoites. The cyst wall was intact and the surrounding areas

revealed no inflammatory reactions. The fibres were distended and distorted particularly in the case of fibres harbouring large cysts.

4 5 3 6 Haemosiderosis

Foci of haemosiderin pigment deposition were recorded in three per cent cases. The pigment appeared golden brown in colour in H&E stained sections. There were extensive areas of haemorrhage in the epicardium and myocardium.

4 5 4 Endocardium

4 5 4 1 Haemorrhage

Haemorrhages of varying degrees were recorded in five per cent cases. Mild to moderate haemorrhages (four per cent) and extensive haemorrhage (one per cent) were seen in the endocardium of the ventricles. Extravasated erythrocytes were seen accumulated in focal or diffuse areas in the endocardial and subendocardial spaces.

4 5 4 2 Endocardial fibro elastosis

Endocardial fibro elastosis was recorded in 14 per cent cases. There was thickening of the endocardium of both the ventricles and the atria particularly of the left side. An increase in the amount of collagen fibres along with large number of long

and wavy elastic fibres were seen which could be demonstrated by PTAH staining wherein the collagen fibres appeared reddish purple in colour (Fig 21)

4 5 4 3 Mural thrombus

In one case an infected mural thrombus was observed in the ventricular endocardium. The thrombus was large in size and appeared as a spherical mass attached to the endocardium. The thrombotic mass was characterised by heavy infiltration with neutrophils and eosinophils. There were flecks of fibrin and hyaline droplets seen as an admixture within the thrombus. The surrounding endocardial and myocardial tissue revealed no pathologic alterations.

4 5 5 The cardiac valves

4 5 5 1 Haemorrhage

Valvular haemorrhages were observed in seven per cent cases. Haemorrhage was observed both in the aortic valve (two per cent) and in the atrio ventricular valves (five per cent). Erythrocytes were seen accumulated either focally or diffusely in the valvular leaflet more towards the free border. In one case an extensive area of haemorrhage was seen at the base of the cusp of the atrio ventricular valve. Both the mitral and tricuspid valves were equally affected.

4 5 5 2 Fatty infiltration

Excessive infiltration with fat was recorded in three per cent cases. Fat vacuoles were seen diffusely arranged in the valvular leaflet in the subendocardial space replacing the connective tissue. There was segmental loss of connective tissue in focal areas adjacent to the area of excessive liposis.

4 5 5 3 Valvular endocarditis

Inflammation of the valvular endocardium was recorded in three per cent cases. There was focal or diffuse infiltration of the endocardium with neutrophils, eosinophils and lymphocytes (Fig 22). Erythrocytes were also seen in clumps or scattered throughout the valvular leaflet of the atrio ventricular valve.

4 5 5 4 Valvular sclerosis

Valvular sclerosis was seen in nine per cent cases. There was thickening of the cusp of the aortic (five per cent) and atrio ventricular valve (four per cent) due to proliferation of fibrous tissue. In the aortic valve the thickening was due to the proliferation of elastic fibres which could be demonstrated using Aldehyde Fuchsin staining technique (Fig 23). The proliferated elastic fibres were distributed throughout the cusp between which the collagen fibres were also observed. In all other cases, there was proliferation of collagen fibres. There was large

number of sprouting capillaries seen throughout the cusp particularly towards the atrial wall. All the capillaries were engorged with erythrocytes (Fig 24). In one case the collagen fibres of the valvular leaflet had undergone metaplasia to chondrocytes. The chondrocytes appeared in focal aggregations admixed within the collagenous matrix. Hyalinisation of some of the fibres was also noticed (Fig 25).

4 5 5 5 Blood cyst

Eleven per cent cases revealed the presence of blood cyst in the atrio ventricular valve. The cysts were of varying size and seen in the loose areolar part of the valvular leaflet. The cyst contained intact red blood cells and leucocytes. The cyst wall was organised and composed of collagen and elastic fibres (Fig 26). In one case, there was haemosiderin pigmentation in the cyst. Cartilaginous metaplasia of the cyst wall characterised by multiple layers of chondrocytes in focal areas was observed in one of the cases (Fig 27).

4 5 6 The coronary vessels

4.5 6 1 Embolism

The intramural coronary artery in the atrial wall revealed an embolus in the lumen in one case. The embolus contained

pink stained glassy material embedded with fibrin and cellular infiltrates

4 5 6 2 Fibrinoid degeneration

Fibrinoid degeneration of the vessel wall was observed in one case. There were several degenerative foci in the adventitial layer of the intramural coronary artery in the atrial wall. The vessel wall was swollen and had a homogenous hyalinized appearance in focal areas. There was also oedema of the adventitial layer of the vessel wall.

4 5 6 3 Hyperplasia of the wall

Hyperplastic vessel wall was recorded in three per cent cases. There was marked increase in the thickness of the wall in the case of coronary venules due to proliferation of collagen fibres. The lumen of the vessel was reduced considerably (Fig 28). Mild degree of mononuclear infiltration along with fibrous tissue proliferation was observed in the perivascular area.

4 5 7 Aorta

4 5 7 1 Calcification

Calcification of the aorta was recorded in four per cent cases. The tunica media revealed areas of calcification which appeared as basophilic patches (Fig 29a) and could be

demonstrated as red coloured areas in Alizarin Red stained sections (Fig 29b) There was loss of elastic fibres in the media and has seen replaced by calcium salts

Fig 1 Haemorrhage and serous atrophy of epicardial fat

Fig 2 Concentric hypertrophy of the ventricle showing reduction in the lumen size

Fig 3 Cardiac dilatation Right ventricular wall is thin and flabby

Fig 4 Endocardial fibroelastosis Thickening of the endocardium appeared as glistening white areas



Fig 3

Fig 5 **Blood cysts in the atrio ventricular valves**

Fig 6 **Diffuse thickening of the atrio ventricular valves**

Fig 7 **Nodular hyperplasia of the pericardial lymphoid collections H&E x160**

Fig 8 **Ganglionic plexus seen in the sub epicardial areolar tissue H&E x250**



Fig 5

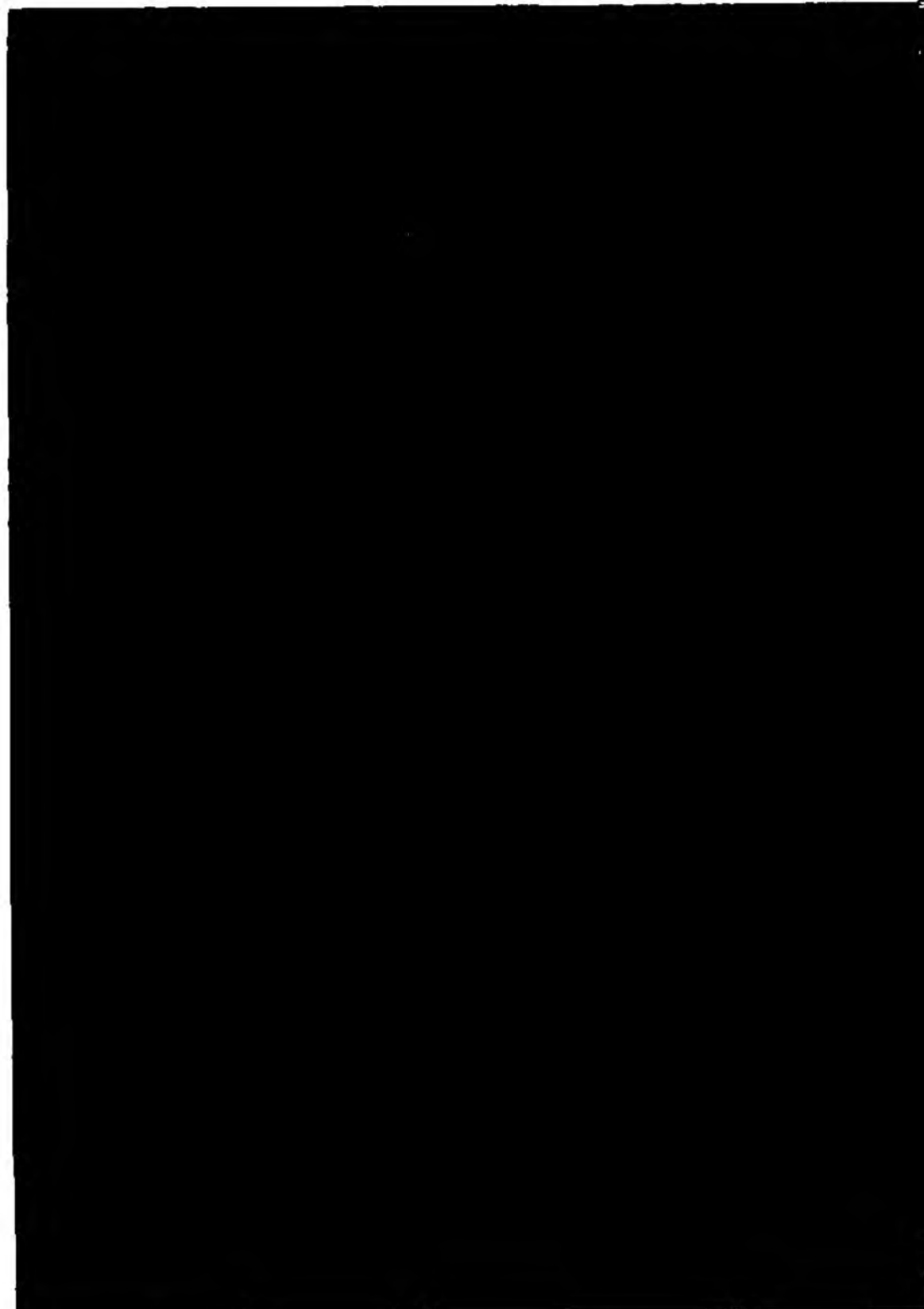


Fig 9 **Thickening of the epicardium due to proliferation of collagen fibres Van Gieson's Picric acid stain x250**

Fig 10a **Hyperplasia of the epicardial lymphoid collections extending into the myocardium H&E x160**

Fig 10b **Proliferated lymphoid collections and oedema fluid in the epicardium H&E x160**

Fig 11 **Serous atrophy of the sub epicardial adipose tissue along with haemorrhage H&E x250**

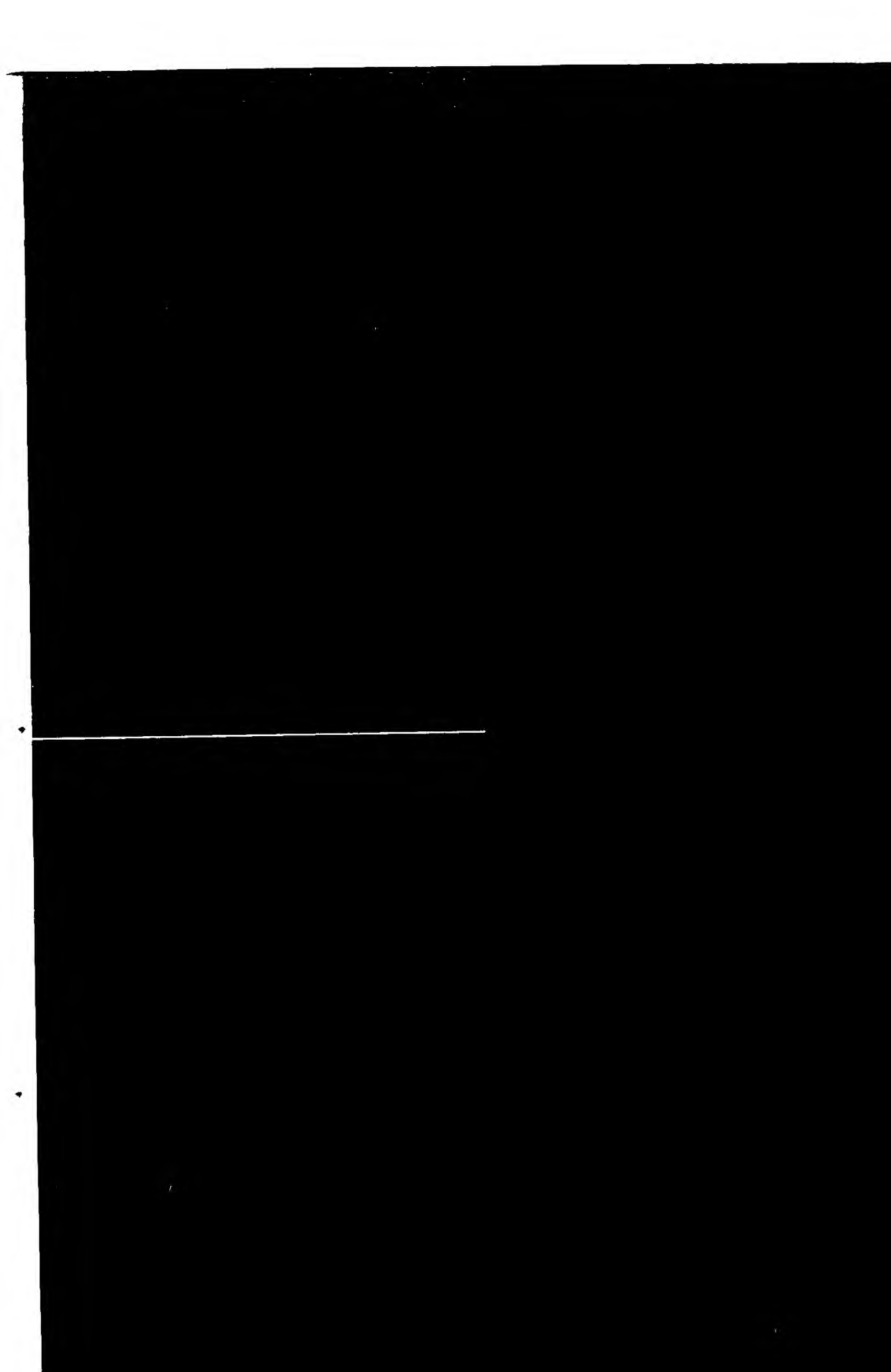


Fig 12 Severely congested intramural coronary vessel with excessive perivascular adipose tissue H&E x63

Fig 13 Haemorrhage in the intermuscular space in the ventricular myocardium H&E x160

Fig 14 Attenuated wavy fibres and widening of the septal space H&E x63

Fig 15 Fatty change in the myocardium Focal and diffuse areas of fat globules replacing the myofibres H&E x160



Fig. 12



Fig. 13



Fig. 14



Fig. 15

Fig 16a Hyalinization of the muscle fibres
H&E x160

Fig 16b Diffuse areas of hyaline degeneration
and fragmentation of muscle fibres
H&E x250

Fig 17 Purkinje fibre degeneration
Vacuolation and infiltration with
mononuclear leucocytes H&E x160

Fig 18 Area of coagulation necrosis in the
atrial musculature along with
mononuclear infiltration H&E x160



Fig 19 Presence of large sized sarcocysts in the muscle fibre H&E x250

Fig 20 Large sized sarcocysts present in the Purkinje fibre H&E x160

Fig 21 Endocardial fibroelastosis in the atrium Collagen fibres appear reddish purple PTAH x250

Fig 22 Valvular endocarditis Mononuclear infiltration extending into the endocardium H&E x160



Fig 23 **Valvular sclerosis Proliferation of
elastic fibres Gomori's Aldehyde
Fuchsin x160**

Fig 24 **Sprouting capillaries in the valvular
leaflets of the atrio ventricular valve
H&E x160**

Fig 25 **Cartilagenous metaplasia of the
valvular leaflet in the aortic valve H&E
x160**

Fig 26 **Blood cyst in the atrio ventricular
valve H&E x160**



Fig. 25

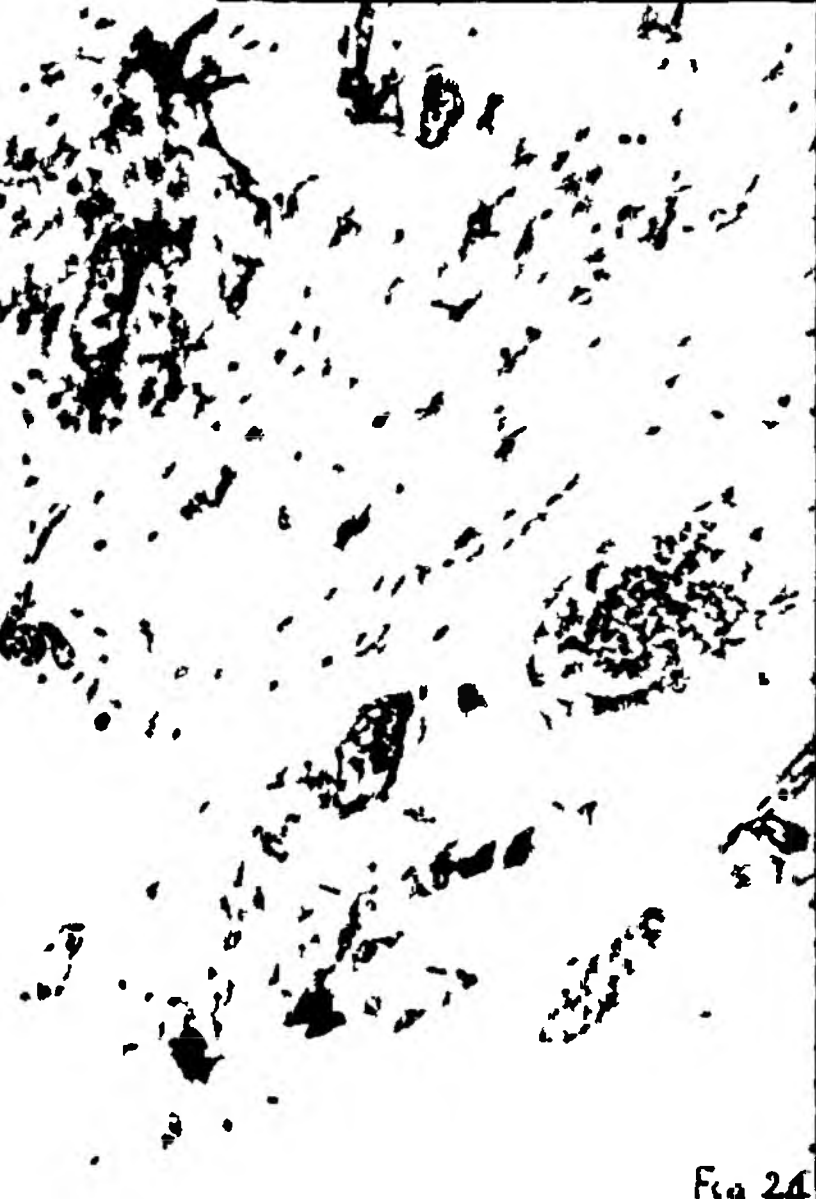


Fig. 24

Fig 27 Cartilagenous metaplasia of the cyst wall in the atrio ventricular valve H&E x250

Fig 28 Hyperplasia of the wall of the intramural coronary artery with reduction in the lumen size H&E x160

Fig 29a Atherosclerotic lesion in the aorta with areas of calcium deposition H&E x63

Fig 29b Areas of calcium deposition stained red in Alizarin - red staining x63



Fig. 29.

Discussion

5 DISCUSSION

The present study was undertaken to investigate the prevalence and pathology of cardiac disorders in cattle. The data collected from the autopsy records maintained at the Centre of Excellence in Pathology, Mannuthy, the morphometry and gross as well as the histopathological features of the samples collected both from the slaughtered cattle and the necropsy cases formed the materials for the study. Analysis of data collected from the autopsy records revealed 35 per cent occurrence of various cardiac lesions in bovines for the period of past 10 years. On the other hand, the present investigation during the period from January 2001 to October 2002 revealed 55 per cent prevalence of cardiac disorders in cattle. This moderate increase in the prevalence of cardiac disorders could be attributed to the small sample size and short duration of the period of study. There was a marked difference in the prevalence of cardiac lesions among the animals brought for autopsy and those observed at slaughter. The higher incidence of lesions in animals between the age groups of six months and three years observed at necropsy indicated the underlying systemic involvement. In the adult animals, the disorders were limited as majority of them were slaughtered in apparently healthy condition. There was no

significant difference between sexes in the prevalence of cardiac disorders

The relative weight of the hypertrophic hearts showed marked difference between individual animals but the average relative weight of the organ was within the normal range as observed by Robinson and Maxie (1993) and Ocal and Cakir (1993). In addition the difference between the different morphometric parameters of the normal and the hypertrophic hearts was not significant except for the left ventricular wall thickness. A positive correlation was observed between the left ventricular wall thickness and the total circumference as well as the cross sectional diameter in the hypertrophic hearts. This clearly indicated the cohesive compensatory adjustments that the heart takes in the phase of an abnormal event and the major role of the left ventricle in such a situation.

The various cardiac disorders were categorised and catalogued based on the gross and histopathological features. Histopathologically the lesions varied from simple degeneration haemorrhage fatty infiltration to inflammation. These could be attributed to hypoxic injury malnutrition and toxic conditions. In almost all the cases fatty change in the cardiac muscle was associated with congestion and haemorrhages of varying degree.

In two cases the calves were severely emaciated and anaemic. Hypoxic injury to the cell due to anaemia could be a reason for fatty change in the myocardium. Malnutrition is also one of the factors causing fatty change. Malnutrition mobilizes the fat and hence leads to an enhancement in the entry of fat into the cells. In addition, the deficiency of lipotropic factors produces defective synthesis of phospholipids thus inhibiting the transport of lipid into the body reserves (Perez Tamayo 1966). The presence of aflatoxin in the livestock feed producing fatty change cannot be ruled out. Zenker's necrosis characterised by hyaline degeneration of the cardiac muscle fibres was observed in the myocardium along with mild fibromyolysis and nuclear pyknosis in 12 per cent cases. Myocardium was diffusely infiltrated with mononuclear leucocytes. McKinney (1974) observed similar lesions in human beings in various myopathies and attributed these to injury produced by toxic plants. Van Vleet and Ferrans (1986) also described similar lesions in selenium vitamin E deficiency in calves. Hence, the underlying etiology of this type of degenerative myopathies could either be toxic or nutritional factors. Degenerated Purkinje fibres characterised by swelling and vacuolation along with mild infiltration with mononuclear cells were observed in a few cases. Sykes and Moore (1942) reported degeneration of Purkinje fibres characterised by

vacuolation as well as the presence of yellowish brown granules in the cytoplasm due to deficiency of potassium in the diet. However in the present study no granules were observed in the cytoplasm of the degenerated fibres. Guarda *et al* (1996) associated the Purkinje fibre degeneration with endocardial lesions but there was no evidence of pathological changes in the endocardium in these cases.

Focal and diffuse myocarditis observed in the study were characterised by mild infiltration with mononuclear and polymorphonuclear leucocytes in the muscle fibres. There was a mild degree of oedema in the interstitium in a few cases. Robinson and Maxie (1993) reported that myocardial lesions could be an extension of lesions from any other location in systemic infections. The lymphocytic myocarditis observed in the study suggested a viral infection. Van Vleet and Ferrans (1986) reported lymphocytic myocarditis with hyaline necrosis and neutrophilic infiltration in foot and mouth disease. Myocarditis characterised by predominant eosinophil infiltration was noticed in a few cases which indicated the presence of parasites. However no evidence of any parasitic cross section could be seen in the tissue sections. Mild inflammation and fibrosis were observed in the myocardium in one case as described by Olsen

(1992) in resolving or healing myocarditis. A healing form of myocarditis characterised by abated inflammatory infiltrate with normal or fibrotic interstitium in peripartum heart disease in human beings was reported by Sanderson *et al* (1986). Waisman and Mahoney (1978) also reported similar lesions in rheumatic myocarditis in human beings. Mononuclear and neutrophilic infiltration between the cardiac muscle fibres was observed in Oleander (*Nerium oleander*) poisoning in cattle by Oryan *et al* (1996). A focal area of coagulation necrosis in the atrial myocardium along with mononuclear infiltration was noticed in one case. The sarcoplasm of the myofibres had lost their structural architecture and it appeared hazy. These lesions are in agreement with the observations recorded by Robinson and Maxie (1993) in porcine stress syndrome. These types of lesions are observed primarily in ischemic states due to an excessive release of endogenous catecholamines. Hence, the presence of the above described lesions in slaughtered cattle could be explained on the basis of various stress conditions encountered during transportation and stunning before slaughter. Cappell and Anderson (1971) also described similar lesions in human beings in toxic conditions. Solitary infected mural thrombi were observed in the endocardium of the ventricle and atrium. The animal had a septicæmic disease and the carcass revealed

hydropericardium hydrothorax ascites hepatic necrosis and suppurative arthritis Hence the thrombotic lesion could be the result of lodging of a septic embolus on the endocardium Yamagishi *et al* (1995) reported a mural thrombus in the endocardium of a cow affected with peripartum heart disease and suggested a multifactorial etiology

In the present investigation presence of sarcocystis in the myocardium was observed in 45 per cent cases The prevalence of sarcocystis in bovine heart was studied by Rajan *et al* (1983) they recorded 47.16 per cent incidence of sarcocystosis in cattle The cysts varied from smaller to larger size and the wall appeared intact with the surrounding myocardium showing no lesions Ninety per cent incidence of sarcocystis pseudocysts without associated myocarditis was reported by Edwards *et al* (1995) Gharagozlou *et al* (2001) observed lymphocytic infiltration of the myocardial muscle fibres in association with presence of sarcocystis Eosinophilic myocarditis in association with degenerated sarcocysts was observed by Ali and Mohamed (1997) they stated that the intensity and number of sarcocysts infecting the myocardium played a highly significant role in the development of eosinophilic myocarditis Contrary to this statement in the present study no such inflammatory reactions

could be observed inspite of the presence of large number of cysts in the muscle fibres This could probably be due to the high adaptability of the particular species of the parasite in forming an intact cyst wall in the host tissue However the presence of unusually large sized cysts in the Purkinje fibres point to the functional disturbance of the conducting system of the heart and the same can not be ruled out

Vascular disturbances like congestion and haemorrhage observed could not be ascribed to any specific etiology except in one case where similar lesions were observed in a cow died of rabies Runnells (1956) also observed endocardial and myocardial haemorrhages in viral diseases Majority of the samples for the study were obtained from the slaughtered cattle These types of vascular changes could be attributed to the violent convulsions at the time of slaughter Serous atrophy of the epicardial adipose tissue observed in a severely emaciated animal indicated the progressive mobilization of depot fat and the replacement of the lipid vacuoles with proteinacious fluid as described by Robinson and Maxie (1993)

Cardiomyopathies were characterised by the presence of attenuated wavy fibres with widening of the inter muscular spaces The individual muscle fibres were thin and atrophic

There was marked disarray of the muscle fibres. These changes were more prominent in the right ventricular musculature. The muscle fibres at focal areas appeared hypertrophic reducing the width of the intermuscular spaces. These findings are in agreement with the observations recorded by Tidholm and Jonsson (1996) and Tidholm (2000) in dilated cardiomyopathy in dogs. Machida *et al* (1996) described similar lesions in Holstein cattle and suggested that a primary myocardial disease similar to hypertrophic cardiomyopathy in human beings also occurred in cattle. The lesions observed in the present study were suggestive of a toxic etiology. Bastianello *et al* (1996) observed similar lesions in Salinomycin toxicity in feedlot cattle. The hypertrophic cardiomyopathy in pigs, dogs and cats is less frequently associated with myocyte disarray and this type of hypertrophic cardiomyopathy is described as symmetrical type (Van Vleet and Ferrans 1986). Huang *et al* (1999) reported that in hypertrophic cardiomyopathy in pigs the histological lesions were more intense in the interventricular septum and apex of the heart. Ferrans (1980) had reported that neither gross anatomic findings nor microscopic features are absolutely diagnostic of hypertrophic cardiomyopathy. Myocyte hypertrophy, interstitial fibrosis and nuclear hyperchromasia were recorded in peripartum cardiomyopathies in human beings (O'Connell *et al* 1986). Eventhough the lesions observed in the present study simulate

the above observations peripartum heart disease could be ruled out as none of the animals came under this category. All the animals were either calves, heifers or culled animals. Black *et al* (1991) hypothesised an autosomal recessive inheritance for cardiomyopathy in cattle. In Kerala the cross breeding policy is followed and there is chance of inbreeding thus leading to expression of the recessive genes.

In hypertrophic hearts fibrinoid degeneration of the blood vessel along with oedema of the tunica adventitia and hyperplasia with proliferation of the connective tissue of the media reducing the lumen size were observed. Similar lesions were described by Van Vleet and Ferrans (1986) in human beings and Machida *et al* (1996) in bovines. Perivascular infiltration with mononuclear cells and fibrous tissue proliferation around the coronary vessels were observed in the myocardium associated with hypertrophic cardiomyopathy. McKinney (1974) reported marked thickening of the small intracardiac arterioles due to fibrous tissue proliferation within and around the blood vessel wall along with lymphocytic infiltration in human beings affected with cardiomyopathy of unknown etiology.

In the present study aged animals with cardiac hypertrophy revealed atherosclerotic lesions

in the aorta along with calcification. The large sized plaques may cause hindrance to the flow of blood leading to ventricular overload and subsequent hypertrophy of the fibres.

Endocardial thickening characterised by proliferation of collagen fibres along with long and straight elastic fibres was observed in a few cases as described in human cardiomyopathy by McKinney (1974). The left ventricles and the left atrium were involved mostly and the condition was observed in the animals of all the age groups. Similar lesions were observed in cattle by Gopal *et al* (1986) and Scarratt *et al* (1987) and diagnosed the condition as a congenital heart disease. Robinson and Maxe (1993) reported that primary endocardial thickening may precede cardiac dilatation for a prolonged period in the absence of any associated cardiac malformation and hypothesised that the endocardial fibroelastosis could have an inherited predisposition. Endocardial fibroelastosis in dogs characterised by deposition of elastic lamellae interspersed with fibrous tissue in the endocardium was reported by Larsson *et al* (1997).

Valvular sclerosis was observed in both the aortic and atrio ventricular valves in aged cattle characterised by thickening of the leaflet due to deposition of dense connective tissue as described in pigs by Gagna *et al* (1998). Calcific aortic stenosis

in old age people characterised by sclerosis and calcification of the aortic cusps was reported by Cappell and Anderson (1971) and Davies (1980). In one case there was cartilaginous metaplasia of the elastic laminae. The focal accumulations of the chondrocytes produced gross nodular thickening of the aortic cusp. In some cases there was mild inflammatory reaction also. The inflammatory reaction was seen more towards the base of the leaflet indicating an extension from the endocardial lesions. The presence of thick walled sprouting capillaries in the valvular leaflets indicate a repair process. Blood cysts were present in the atrio ventricular valves of both calves and adult animals but the incidence was more in the young animals. The observations made in this study were in agreement with the findings of Marcato *et al* (1996) in cattle and Gupta (1969) in pigs. It was suggested by Takeda *et al* (1991) that the mechanical effects such as an increase in tension, friction and impact could trigger the enlargement of valvular blood vessels in a sort of valvular telangiectasis with subsequent cyst formation. According to Marcato *et al* (1996), the valvular cyst formation was due to the dilatation of blood and lymph vessels. Robinson and Maxie (1993) recorded congenital hematomas present on the margin of the atrioventricular valves in calves.

In the present investigation the pericardial lesions observed were limited in number. In one case the pericardium was thickened due to proliferation of the collagen fibres. This may be a sequel of previous inflammatory condition. Constrictive pericarditis in a horse characterised by fibrosis and thickening as a sequel to pericardial inflammation was described by Worth and Reef (1998). In the present study hyperplasia of the focal lymphoid collections of the pericardium was observed in a black cattle. Bacteriological examination of the lymphnode impression smear revealed acid fast organisms but no typical tuberculous nodules could be observed. On the other hand, all the serous membranes revealed hyperplastic lymphoid nodules without any extension into the parenchyma which suggested an initial immune response to the tuberculous organisms.

The systematic investigation undertaken has helped in categorising and documenting the different cardiac lesions and to assess the prevalence of various conditions affecting the heart in cattle. The conditions known to be associated with myocardial lesions include hypothyroidism, hyperthyroidism, anaemia, malnutrition, parasitic infections, toxaeemias and poisoning, neoplasia, pyometra and endometritis as well as diabetes mellitus (Ettinger and Sutter 1970). Despite the fact that the

study was focussed mainly on slaughtered cattle which were apparently healthy a high incidence of cardiac lesions could be observed The findings of the present study suggest that many of the sudden death cases of unknown etiology in field conditions could probably be the result of underlying cardiac problems The results of the study suggest the need for a detailed investigation on the etiological and clinico pathological diagnosis of cardiac disorders in cattle

Summary

6 SUMMARY

An investigation was carried out to study the prevalence and pathology of cardiac disorders in cattle. The prevalence of cardiac disorders in cattle for the past 10 years was found to be 35 per cent by analysis of the data collected from the autopsy records maintained at the Centre of Excellence in Pathology Mannuthy.

Hundred and one bovine hearts collected from the carcasses brought for autopsy at the Centre of Excellence in Pathology Mannuthy as well as from the cattle slaughtered at the Meat Technology Unit, College of Veterinary and Animal Sciences Mannuthy and from the Municipal Slaughter House Thrissur were used for the study. The samples collected were subjected to morphometrical analysis and detailed gross as well as histopathological examinations. The average relative weight of hypertrophic hearts was within the normal range. In hypertrophic hearts, the left ventricular wall showed significant increase in thickness, thus giving a major contribution to the increased circumference and cross-sectional diameter of the heart. The conditions encountered were classified and pathological features were recorded and each lesion was

explicated giving possible etiopathogenesis. The study revealed a high prevalence (55 per cent) of cardiac disorders in cattle. The lesions were haemorrhage (33.8 per cent), congestion (5.3 per cent), ventricular hypertrophy (21.4 per cent), cardiac dilatation (14.2 per cent), myocardial degeneration (8.9 per cent), endocardial fibroelastosis (12.5 per cent), hydropericardium (5.3 per cent), pericarditis (5.3 per cent), pericardial adhesion (1.7 per cent), serous atrophy of the epicardial fat (3.5 per cent), blood cyst in the valve (19.6 per cent), serous cyst in the valve (10.7 per cent), valvular thickening (16 per cent) and valvular haemorrhage (3.5 per cent).

The most common lesion encountered was the presence of sarcocystis tissue cysts in the cardiac musculature. It was observed in 45 per cent cases. Cysts of varying size with intact cyst wall were present both in the cardiac muscle fibres and in the Purkinje fibres. The unusually large sized sarcocysts present in the Purkinje fibres pointed to the functional disturbance of the conducting system of the heart.

Moderately high incidence of fatty change and hyaline degeneration was observed in the myocardium and this could be attributed to hypoxic injuries resulting from anaemia as well as to malnutrition. The presence of aflatoxin in the cattle feed can

produce fatty change in the myocardium. Degeneration of Purkinje fibres was observed in a few cases which could be ascribed to deficiency of minerals especially potassium.

In a few cases the myocardium revealed focal and diffuse infiltration with mononuclear and polymorphonuclear leucocytes. Predominance of eosinophils in a few cases indicated a parasitic infection whereas the lymphocytic myocarditis noticed in one case was suggestive of viral disease. In most of the cases the lesions were of a mild degree and could be considered as a healing or resolving form of myocarditis. An area of coagulation necrosis was observed in the atrial myocardium in one case. This ~~lesion could~~ be due to the release of endogenous catecholamines in various stresses during the process of slaughter. The presence of solitary infected mural thrombi in the atrial and ventricular endocardium in one case could be due to lodging of the septic emboli on the endocardium. The higher incidence of vascular disturbances could be attributed to both viral infections and the violent convulsions occurring during slaughter.

Histopathological lesions suggestive of dilated or hypertrophic cardiomyopathies were observed in a few cases. The thin and atrophic muscle fibres formed a wavy appearance with

an increase in the intermuscular space. On the other hand muscle fibres in focal areas were hypertrophic with active nuclear changes. Based on the observations recorded this condition could be attributed to hereditary toxic or nutritional etiology. Endocardial fibroelastosis was noticed in a few cases. The predominance of collagen fibres could be demonstrated by PTAH staining.

Valvular sclerosis was observed in a few aged animals with cardiac hypertrophy. The proliferated connective tissue fibres could be demonstrated by special staining technique using Gomori's Aldehyde Fuchsin. In one case there was cartilagenous metaplasia of the connective tissue laminae of the valvular cusp. These changes could be attributed to senility. A higher incidence of valvular cysts of varying size was noticed especially in the young animals. This could be due to stress induced dilatation of blood and lymph vessels in the valve and could be congenital. Hyperplastic nodules of lymphoid collections in the pericardium was observed in a black cattle. It could be ascertained that the lymphoid hyperplasia was due to the immune response evoked against the tuberculus organisms.

The systematic investigation undertaken has helped to focus attention on the prevalence of various cardiac disorders in

cattle. As believed, cardiac disorders in cattle are not less frequent. A high incidence of cardiac lesions could be recorded in spite of the fact that most of the samples subjected to the study were from slaughtered cattle which were apparently healthy. Many of the conditions encountered in this study were of detrimental effect and could result in death of the animal. The findings suggest that many of the sudden death cases of unknown etiology in field conditions could probably be the result of underlying cardiac problems. Therefore, a detailed investigation can bring light to the unknown disorders and the etiopathogenesis of the known conditions can be clarified.

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PATHOLOGY OF CARDIAC DISORDERS IN CATTLE

By
BISI T V

ABSTRACT OF A THESIS

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**Centre of Excellence in Pathology
COLLEGE OF VETERINARY AND ANIMAL SCIENCES
MANNUTHY THRISSUR 680651
KERALA INDIA
2002**

ABSTRACT

The present study was undertaken to assess the prevalence and pathology of various cardiac disorders in cattle. The data collected from the autopsy records maintained at the Centre of Excellence in Pathology, Mannuthy, and hundred and one samples of heart obtained from both the slaughtered cattle and necropsy cases formed the basis of the study. Fifty five per cent hearts showed lesions of varying type. The highest incidence was for sarcocystosis (45 per cent). Vascular lesions were present in 33.8 per cent cases. Other myocardial lesions encountered were myocardial degeneration, myocarditis, and other cardiomyopathies of unknown etiology. Endocardial fibroelastosis and valvular sclerosis were observed in four cases. Valvular and coronary vessel lesions showed correlation with hypertrophic cardiomyopathy. The incidence of pathological disorders encountered were relatively high. The need and scope for investigation into the pathological disorders of bovine heart were highlighted.