THE PATHOLOGY OF SOME SPONTANEOUS CARDIOVASCULAR DISEASES IN THE DOG

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SUMMARY

This thesis describes the nature, frequency and severity of some spontaneous cardiovascular diseases in a series of 200 unselected dogs. This study concentrated on two conditions, chronic valvular disease and arteriosclerosis.

It was established that chronic valvular disease was a mucoid degeneration of the spongiosa and fibrosa of the valves. These lesions occurred with the greatest frequency and severity in the left atrioventricular valve where they gave rise to valvular insufficiency and congestive heart failure in 6.0% of cases. Similar lesions occurred in the other heart valves but they were rarely associated with clinical disease. It was shown that chronic valvular disease increases in frequency and severity with advancing age, particularly in the left atrioventricular valve. These lesions tended to develop more rapidly in the male than in the female and did not appear to have any breed predisposition, nor were they associated with diseases in any particular body system. Age emerged as the most important single aetiological factor.

Two different changes were seen in the posterior aorta and coronary circulation. One, a fibrous intimal plaque of no clinical significance and the other an amyloid degeneration of the intramural coronary vessels of the left ventricle. This lesion may have some clinical significance when associated with chronic valvular disease.

Other cardiovascular diseases were observed and recorded.

THE PATHOLOGY OF SOME SPONTANEOUS CARDIOVASCULAR DISEASES IN THE DOG

Introduction

The volume of research into the nature of the spontaneous cardiovascular diseases of animals has increased in the last fourteen years. The stimulus was provided by the report of a meeting of the World Health Organisation held in Washington in 1957. This meeting recommended "that the investigation of Arterial diseases in Man should be developed to embrace the whole Animal Kingdom and not be confined to Man and the common laboratory animals."

Since then an extensive range of wild and domestic animals have been 'screened' to determine their suitability as experimental animals for atherosclerosis research, but little effort has been directed towards the study of any other spontaneous cardiovascular disease that may occur in these animals. The main reasons for this deficiency lies in the world-wide shortage of funds and trained observers to undertake these investigations.

The dog is one exception to this rule. This species has been a popular experimental animal for many years and the great advances in small animal medicine in the post-war years has increased our knowledge of some aspects of cardiovascular disease in the dog to a point far in advance of other species. Much of the credit for this is due to Dr D. K. Detweiler and his colleagues at the University of Pennsylvania for their studies in canine clinical cardiology. As a result of their investigations, it has been possible to determine three important cardiovascular diseases of the dog.

Chronic valvular disease is regarded as being the most important

of these conditions as it results in an insufficiency of the left atrio-ventricular valve which may lead on to congestive heart failure in some cases. However, little is known of the aetiology, pathology or pathogenesis of this condition.

Changes have been observed in the myocardium of the left ventricle of the heart which are characterised by degeneration and fibrosis of muscle fibres. These changes are associated with hyalinisation and thickening of the walls of the intramyocardial coronary vessels. While this condition in itself is of little clinical importance, it may assume clinical significance when found in association with chronic valvular disease.

The abdominal aortae of ageing dogs may show fibrous arteriosclerotic plaques in a proportion of cases and these lesions are of considerable interest to the comparative cardiologist.

The bulk of this thesis is devoted to the study of these three conditions.

In addition to these three, certain other cardiovascular diseases were observed which will receive some consideration in the text.

This investigation was based upon the examination of 200 unselected dogs that were presented for post-mortem examination.

Material and Methods

The material which forms the basis of this study was drawn from 200 unselected dogs which were presented for post-mortem examination to the Department of Veterinary Pathology, Royal (Dick) School of Veterinary Studies, in the years 1960-1964.

In Table 1, the age and sex of these dogs are recorded and the same dogs were also divided into four age groups (Table 2) as follows:-

(a) Young dogs (0-4 yrs. inclusive)

(b) Adult dogs (5-8 yrs. inclusive)

(c) Middle-aged dogs (9-12 yrs. inclusive)

(d) Old dogs (13-16 yrs. inclusive)

In Table 1 it will be seen that the number of individuals in any one group are small, while in Table 2 the numbers of individuals are larger in each of the four age groups. It is apparent that there are more males than females (45:31) in age group 0-4 yrs. inclusive, and that the situation is reversed (31:39) in the age group 9-12 years inclusive. The distribution is closer in the other two groups. In this series of 200 unselected dogs, the mean age for the males was 6.4 years, while the mean age for the females was 6.58 years. The overall mean age was 6.48 years.

Table 3 summarises the age and breed distribution of these dogs and Table 4 divides them into the same four age groups described above. A total of 33 breeds and a large group of cross-breds are represented. Only five breeds are represented by more than ten individuals, i.e. Alsatian, Border Collie, Boxer, Greyhound and Labrador.

These 200 dogs were presented to the Small Animal Practice Teaching Unit, Royal (Dick) School of Veterinary Studies, for a variety of reasons. They were, in some cases, subjected to varying forms of therapy appropriate to their condition prior to euthenasia, while in other cases they

were subjected to immediate euthenasia at the owners' request, or upon the advice of the clinical staff. Clinical cardiovascular disease was detected in some cases, while in others no such disease was detected or suspected. In Table 5, the reasons for death or destruction are recorded.

All these dogs were presented to the Department of Veterinary Pathology for post-mortem examination which was undertaken by the author in all cases.

Post-mortem examination

The carcases were subjected to a standard post-mortem technique. The heart and lungs were subjected to a surface examination and then the pericardium was opened at the apex, so that the volume, colour and nature of any excessive pericardial fluid could be determined. Then, the pericardial sac was removed. The brachio-cephalic artery and the aorta were freed from the mediastinum and the lungs were separated from the heart by cutting the bronchii, pulmonary arteries and pulmonary veins. The aortic arch was severed at a point immediately proximal to the origin of the anterior aorta and placed in a dish of warm normal saline for examination later.

The examination of the heart

The isolated heart was washed in normal saline and subjected to a surface examination, particular note being taken of any variations in shape, colour or consistency of the organ.

The heart was dissected as follows (Fig.1).

The points of scissors were introduced through the walls of the left and right atria beneath the atrial appendages and two incisions (a.1 & 3) were made parallel to and above the left and right circumflex coronary arteries across the dorsal face of the heart. These two incisions were continued on either side of the interatrial septum (a.2 & 4) to expose the atrial aspects of the left and right atrio-ventricular valves and the atrial endocardium. Again, the heart was washed in normal saline and the blood clots were removed from the heart chambers by gentle digital manipulation.

Ligatures were applied to the pulmonary artery and the aortic arch (b.5 & 6) and the ventricles were filled with fresh, warm normal saline by immersion. By means of gentle digital pressure, it was possible to obtain an assessment of the competency of the atrio-ventricular valves.

The left and right ventricles were opened by two parallel incisions down (c.7 & 8) either side of the inter-ventricular septum extending from the atrio-ventricular ring to the apex of both ventricles. These incisions passed through the commissural areas of both valves. The lumina of both ventricles were examined, particular attention was paid to the valve cusps, chordae tendineae and the endocardium. At this point, the aortic and pulmonary inlet and the heart chambers were packed with cotton wool and the whole heart was immersed in a bucket of 10% neutral formol saline for a period of five days.

At the end of this period, a medial incision through the ventral (anterior) cusp of the left atrio-ventricular valve (d.9 & 10), the posterior cusp of the aortic valve and the aortic arch exposed the aortic valve and the related structures. On the right side, an incision lateral to the supra-ventricular crest exposed the pulmonary conus and was extended into the pulmonary artery, through the pulmonary valve (d.11 & 12).

After removal of the appropriate blocks for histological examination, the remainder of the heart was subjected to multiple transverse incisions across the extra-myocardial coronary vessels and adjacent myocardium.

The examination of the aorta

The aorta was removed from normal saline and all excess fascia and fat was removed from the surface of the adventitia. The 'stumps' of aortic branches were trimmed off 'flush' with the adventitial surface, with the exception of the iliac vessels. Then the aorta was opened on its long axis with a pair of blunt pointed scissors to expose the intima. The vessel was washed in warm normal saline and laid out, intimal surface uppermost on an appropriately-sized piece of cardboard and placed in 10% neutral formol saline for 24 hours.

In all, 50 aortae were stained grossly with Sudan IV (Holman 1958) and examined. A further 150 aortae were fixed by the method described above but were not subjected to Sudan IV staining.

The examination of the remainder of the carcase

The remainder of the carcase was subjected to the standard post-mortem examination technique.

Histopathological Methods

Fixation All tissues were fixed in 10% neutral formol saline.

(a) The heart

The blocks, selected for examination, were taken in accordance with recommendations of the Armed Forces Institute of Pathology (cit Detweiler 1960). A minimum of ten blocks were taken from each heart as follows:

(i) Ventral cusp of left atrio-ventricular valve and including the dorsal cusp of the aortic valve.

(ii) Dorsal cusp of left atrio-ventricular valve and a related segment of left ventricular myocardium.

(iii) Left ventral papillary muscle and adjacent wall of left ventricle.

(iv) Left atrial appendage.

(v) Vertical section through inter-ventricular septum to include the Bundle of His and septal cusp of right atrio-ventricular valve.

(vi) Mural cusp of right atrio-ventricular valve and a related segment of the right ventricle.

(vii) Conus arteriosus, pulmonary valve and pulmonary artery.

(viii) Transverse section through proximal centimetre of left circumflex coronary artery.

(ix) Similar section through right circumflex coronary artery.

(x) Left dorsal papillary muscle and apex of left ventricle.

After 24 hours further fixation, the blocks were trimmed and given five further days of fixation in fresh neutral formol saline.

(b) The aorta

After gross fixation and in some cases, Sudan IV staining, appropriate blocks were taken from the aortae and fixed in 10% neutral formol saline for five days. Routine blocks were taken from the thoracic portion, the region of the renal artories and the iliac bifurcation.

(c) Other organs

Routine blocks were taken from the liver and lungs in all cases and blocks of other organs were taken appropriate to the condition represented at post-mortem. All these blocks were fixed in 10% formol saline for five days final fixation.

Staining Methods

Selected blocks were processed by the frozen section technique and stained by Sudan IV, Oil Red O and by Sudan Black B for fat.

The remainder of the blocks were processed in paraffin wax and stained routinely by Harris' haematoxylin and eosin.

Following an examination of these sections, a selected number were subjected to one or a number of the following staining methods:

The Verhoeff-Van Gieson method was regularly employed to demonstrate elastic (and collagen) fibres in the heart valves and blood vessels. The Weigert stain was also employed with some sections.

Van Gieson's method was used for collagen and the Gordon and Sweet's method was used to stain reticulum. Masson's Trichrome and Heidenhain's Azan stain upon occasion.

An extensive range of carbohydrate stains were employed in this study. The Periodic acid-Schiff method for glycogen and other carbohydrates was

used both with and without diastase. The metachromatic stains, Toluidine Blue and Azur A were employed. Alcian Blue with and without Methylation and Saponification was used in addition to the Hale's colloidal iron method, Halmi's modification of the Gomori aldehyde fuchsin technique (combined Alcian Blue/PAS and Alcian Blue/aldehyde fuchsin).

To detect amyloid, Congo Red, methyl violet and Thioflavine T were used, followed by exposure to ultra-violet light.

Sundry other methods were used including Perl's Prussian Blue reaction for ferric salts, Mallory's phosphotungstic acid-haematoxylin for muscle striations and Lendrum's acid Picro-Mallory method for fibrin.

Other Investigational Techniques

In addition to the 200 dogs' hearts that were examined in the pathological investigation, three further series of hearts were examined in the course of the anatomical and histological investigations.-

(a) A series of 50 unselected dogs' hearts were examined in order to determine the normal histology of the canine heart valve. These hearts were processed in a similar way to that described above.

(b) A series of 14 young greyhound hearts were examined to determine the distribution of the blood supply from major coronary vessels to the myocardium. For these purposes a radio-opaque material was injected into the coronary vessels (p.77).

(c) A series of 16 young greyhound hearts were examined to investigate various aspects of the coronary circulation. For these purposes, three different injection masses were employed.

The injection technique

The greyhounds used in this study were anaesthetised with nembutal and exsanguinated until dead. The thoracic contents were removed from the carcase as rapidly as possible and the pulmonary veins and venae cavae ligated. The aortic arch was severed proximal to the origin of the anterior aorta. The heart was isolated from the other thoracic contents and washed gently in warm normal saline. The organ was gently massaged to remove as much blood as possible from the heart chambers and coronary circulation. The pulmonary artery was severed proximal to its bifurcation then the preliminary preparation was concluded by washing the heart in clean, warm, normal saline and storing it for 24 hours in normal saline at $4^{\circ}C$.

The following day the heart was warmed slowly to room temperature and massaged to remove rigor mortis and any residual blood from the vessels

or heart chambers. Then the heart was ready for injection, the final preparation depending upon the particular requirement.

If it was intended to inject the left ventricular cavity, the proximal centimetre of the left and right coronary vessels were isolated and ligated with strong cotton thread. A large glass cannula was introduced into the aorta and tied firmly with string (after flushing with warm normal saline). The heart was introduced into a bath of warm normal saline and the injection commenced.

If it was proposed to inject the coronary vessels as well, fine glass cannulae were introduced into the left (and sometimes the right) coronary artery and tied in situ. The ligature was removed from the posterior vena cava and the system was 'flushed' with warm normal saline. Then the injection mass was introduced as described below.

The subsequent treatment of the heart depended upon the nature of the injection mass that was employed; three different injection masses were used:-

(i) Vinyl resin (VYHH) was prepared as a 12% solution in acetone and coloured with a variety of dyes, e.g. Red (Calco Oil Red), Yellow (Luxol fast yellow G). This injection mass was introduced into the heart at 3-5 lbs. pressure. This pressure was maintained until the level in the stock bottle of injection mass had remained constant for 2-3 minutes. In the case of the coronary vessels, the catheters were removed and the vessel was tied off. In the case of injection into the heart chambers, the rubber tubing above the cannula was clamped off. In some cases, multiple injections were made into the major coronary vessels with different coloured injection masses which required the isolation of each individual vessel. The technique otherwise, was identical.

When the injection was completed, the heart was left in a bath of normal saline for 1-2 hours to enable the vinylite to set, then the heart was transferred to a bath of concentrated hydrochloric acid for maceration which was completed within 24-36 hours. The residual debris

was washed off in a gentle stream of tap water. The cast was stored in 5% formol saline to prevent fungal growth.

(ii) The commercial radiopaque injection mass 'Micropaque' was introduced into the coronary vessels and heart chambers at 1½ lbs. pressure. The technique of injection was identical in every other respect to that employed for Vinylite. Subsequent to the injection, the treatment was different. Xrays were taken on a Watson portable machine. No details of exposure, etc. were available on these plates due to loss of records in the department.concerned.

The heart was placed in 10% neutral formol saline and fixed for 10 days. Then the atria were opened to permit drainage of the Micropaque from the heart chambers. The hearts were then placed on an electric bacon slicer and cut transversely into slices 0.3-0.5 cm thick. These slices were then washed thoroughly to remove residual Micropaque, particularly from the cut surfaces and dried with blotting paper.

The slices were then Xrayed and restored to 10% formol saline for storage. In two cases, some slices were placed in Oil of Wintergreen and 'cleared' for naked-eye examination of the vessels within them.

(iii) Gelatin-carmine This injection mass was introduced into the chambers of the left heart under 3 lbs. pressure and then transferred to iced water. The heart was fixed intact in 10% neutral formol saline and after ten days the appropriate blocks were selected for histological examination, trimmed and given a further five days fixation in 10% formol saline before processing in paraffin-wax and staining with Harris' haematoxylin and eosin.

The Review of the Literature

This section is divided into three sub-sections as follows:

1. The anatomy of the canine cardiovascular system.
2. The histology of the canine cardiovascular system.
3. The pathology of spontaneous cardiovascular diseases in the dog.

The Anatomy of the Dog's Heart

While the general structure and appearance of the dog's heart is similar to that of other species, there are certain variations peculiar to the dog which are pertinent to this thesis and require description. In describing the anatomy of animals' hearts, veterinary anatomists have tended to use medical terms to 'label' the various structures; this has been avoided here, but an attempt has been made to correlate the newer terminology with the old. (Most of these modified terms are associated with variations in the posture of the dog in relation to man.)

The atria

The heart of the dog is composed of four heart chambers; there are two atria, left and right, which are similar in structure to that of other species. The atria are separated from the two ventricles by the cardiac skeleton which also provides the main attachment for the heart valves.

The cardiac skeleton

The cardiac skeleton is a complex of four fibrous and partly cartilagenous rings which separate the thin atrial and the thick ventricular muscle from each other. The cardiac skeleton is traversed by one single specialised bundle of muscle, the atrio-ventricular bundle, which is part of the conduction mechanism of the heart. (P.32) These four fibrous rings of the cardiac skeleton surround the following structures:-

(a) The aortic fibrous ring surrounds the root of the aorta and is best developed at the points of attachment of the three semi-lunar cusps of the aortic valve. This gives the aortic fibrous ring a 'scalloped' appearance in transverse section and creates three shallow concavities which form the sinuses of the aortic valve. The cardiac skeleton is best developed dorsal to the aortic fibrous ring between this structure and the left and right atrioventricular fibrous rings where two triangular fibro-cartilagenous areas are formed - the left and right fibrous trigones.

(b) The pulmonary fibrous ring is a more delicate structure than the aortic fibrous ring but similar in structure. The myocardium of the conus arteriosus is attached to its ventral surface and the media of the pulmonary artery to its dorsal surface.

(c) The left and right atrioventricular fibrous rings are both relatively delicate structures which join the myocardium of the atria to that of the ventricles and support the atrio-ventricular valves. The greater bulk of the ventricular myocardium causes it to bulge outwards so that it tends to surround the atrioventricular fibrous rings.

The left ventricle

The left ventricle is a thick-walled conical structure whose apex also forms the apex of the heart. The wall of the left ventricle is three times thicker than the right ventricle (Truex and Warshaw 1942).

The two large papillary muscles in the left ventricle are protruberances of myocardium which support the left atrioventricular valve and the chordae tendineae during systole. The ventral (anterior) papillary muscle lies close to the ventral (anterior) interventricular groove where it is associated with a network of fine muscular strands which traverse the ventricular lumen from the inter-ventricular septum. These muscular strands represent terminal divisions of the left bundle branch of the Conduction Mechanism. The ventral (anterior) papillary muscle is separated from the dorsal (posterior) papillary muscle by a deep cleft. The dorsal papillary muscle rises, in part, from the wall of the left ventricle and partly from the interventricular septum. Both papillary muscles terminate within a few millimetres of the apex of the left ventricle. The chordae tendineae are inserted in the crest of each papillary muscle and pass to both cusps of the left atrioventricular valve.

The trabeculae carneae are long myocardial projections (separated by deep, oblong depressions) which run parallel to the long axis of the ventricle. These tend to be more concentrated at the apex of the heart.

The right ventricle

The right ventricle which lies ventral and cranial to the left ventricle is crescentic in transverse section and does not form part of the apex of the heart. The right ventricular wall is thin when compared with the left ventricle.

The papillary muscles are variable in number, but there are usually three arising in the distal third of the ventricle. The largest papillary muscle arises at the junction between the dorsal margin of the right ventricular wall and the interventricular septum. Another papillary muscle, which is constant in position, lies in the lower third of the conus arteriosus. The chordae tendineae of the right atrioventricular valve arise from these papillary muscles and from the mural endocardium (Truex and Warshaw 1942). Trabeculae carneae are seen lining the wall of the right ventricle and a thin, branching, muscular strand (trabecula septomarginalis or moderator band) runs across the lumen from the interventricular septum to the lateral wall in the vicinity of the dorsal papillary muscle.

The interventricular septum consists of two parts, a small vestigeal fibrous portion which lies beneath the septal cusp of the right atrioventricular valve and a thick muscular portion formed from the fusion of the myocardium of the left and right ventricles.

The Heart Valves

The heart valves may be classified into two groups:-

(a) The atrioventricular valves

(b) The semi-lunar valves.

The left atrioventricular valve (Mitral Valve)

The left atrioventricular valve is a more substantial structure than the right atrioventricular valve, due to the higher blood pressure that exists in the systemic circulation. The valve consists basically of two cusps, one ventral (anterior) and one dorsal (posterior) with abundant commissural areas, particularly on the medial aspect. Sisson (1945) describes the left atrioventricular valve as consisting of two large cusps and four or five small ones. Sheridan (1967) found difficulty in defining the commissural areas, but considered that this valve was basically bi-cuspid. Miller et al (1964) described three types of chordae tendineae as follows:-

(i) Thick chords which originate at the attached border of the valve and extend over the ventricular face.

(ii) Medium-sized chords which extend to the middle of the valve on the ventricular face.

(iii) Fine chords which are attached to the margins of the valve.

Quain (1929) classified the chordae tendineae of the human heart into three groups as follows:-

(a) Thick primary chordae tendineae which run a short direct course to the crest of the papillary muscle and are parallel to its long axis.

(b) Secondary chordae tendineae which are longer and finer, and are attached to the margins of the valve cusp.

(c) Short, thick tertiary chordae tendineae which are inserted into the wall of the left ventricle.

Brock (1952) regarded the points of origin of the primary chordae tendineae as the most important points on the valve cusp in man and called them the 'critical areas'. In consequence, each cusp had two 'critical areas' which divided the cusp into three parts:-

(i) A distal, central, or horizontal portion which lay between the two 'critical areas'.

(ii) Two lateral or receding oblique portions.

The primary chordae tendineae were confined to the 'critical areas' and the secondary chordae tendineae were almost completely confined to the lateral, oblique pathways. The central pathway had few, if any, chordae tendineae. In diastole, the blood entered the ventricle through the central pathway, the lateral, oblique pathways being filled by the chordae tendineae and the papillary muscles (Fig. 2).

In the left heart, the anterior mitral cusp is responsible for the closure of the greater part of the atrioventricular orifice in systole (Brock 1952). Sheridan (1967) in a diagram suggests that the same is true for the dog; that the ventral (anterior) cusp closes the greater part of the left atrioventricular orifice.

The right atrioventricular valve (Tricuspid)

The lower pulmonary blood pressure on the right side results in less substantial valve cusps and attendant structures in the right atrioventricular valve. The valve consists of two cusps, one septal (dorsal) cusp and one mural (ventral) cusp. Again, there are abundant commissural areas with secondary cusps. The septal cusp is associated with the interventricular septum, the mural cusp with the lateral wall of the right ventricle. The septal cusp is more substantial than the mural cusp. The chordae tendineae from each valve cusp are inserted into the crests of the papillary muscles and the mural endocardium. The variability in number of the papillary muscles results in variations in the distribution of the chordae tendineae. The chordae tendineae of the septal cusp are all inserted into the mural endocardium.

The semilunar valves

The semilunar valves are associated with, and based upon, the aortic and pulmonary fibrous rings of the cardiac skeleton. They are known as the aortic and pulmonary valves respectively. Each valve consists of three cusps, whose function is to prevent blood returning to the ventricles from the great vessels during diastole.

The aortic valve

The aortic valve has one dorsal, one left and one right semilunar cusp. The areas of apposition of each of these cusps is defined by two semi-circular areas (lunulae) in the distal third of the cusp, with a central fibrous nodule (nodulus valvularum aorta).

Behind each valve cusp is a slightly concave space - the aortic sinus - which holds a small reserve of blood and prevents the valve cusp being pressed too tightly against the wall of the aorta (during systole), thereby facilitating closure during the subsequent diastole.

The left and right coronary arteries originate beneath the left and right aortic cusps.

The pulmonary valve

The pulmonary valve is less substantial than the aortic valve and lies cranial to that valve. In its structure it is similar to the aortic valve, having three cusps, one ventral, one left and one right semilunar cusp. This valve prevents a reflux of blood from the pulmonary artery into the right ventricle during diastole.

THE BLOOD SUPPLY TO THE HEART

The coronary circulation which supplies blood to the heart is derived from two vessels, the left and right coronary arteries. These vessels originate respectively from beneath the left and right cusps of the aortic valve.

The left coronary artery

The left coronary artery originates from a single opening in 95% cases and as a double opening in 5% cases (Blair 1961). It is a short vessel, up to 5 mm. long, which divides regularly into two divisions, the circumflex branch and the ventral (anterior) descending branch. A third vessel may originate from the left coronary artery but it shows some variation in its point of origin. This is the ventral (anterior) septal artery which, according to Blair (1961) may originate at the bifurcation of the left coronary artery (55%) along the course of the ventral descending branch (36%), along the course of the left coronary artery (5%) or along the course of the circumflex branch (4%). Carmella, Vane, Anderson, Vitmanson and Hay (1957) support Blair's observations, but these findings are at variance with Donald and Essex (1954a).

The circumflex branch

This vessel, lying in the coronary groove, runs around the left side of the heart to its caudo-dorsal surface where, in the vicinity of the dorsal (posterior) interventricular groove, it turns towards the apex of the heart and is known as the dorsal (posterior) descending branch. In the first part of its course, the circumflex branch is covered by the left auricular appendage.

The circumflex branch gives off both ascending and descending divisions to the left heart:-

Ascending divisions

These vessels are small and variable in number but Moore (1930) describes two major divisions, one a proximal division which supplies the left atrium on its deep surface, the left auricular appendage and

part of the interatrial septum; a distal division supplies the remainder of the left atrium and interatrial septum. Meek, Keenan and Theisen (1929) regarded the proximal division as the most important vessel as its anastomoses with ascending divisions of the right coronary artery in the vicinity of the sinoatrial node. Christensen and Campeti (1959) also describe an intermediate division.

Descending divisions

The number of descending divisions of the circumflex branch are variable. Kazzaz and Shanklin (1950) describe 4-11 vessels, Moore (1930) gives a range of 6-8 vessels and Pianetto (1939) found 2-6 principal descending divisions. It is clear that a considerable variation exists and that this is due in part to variations in the origin of the descending vessels of the left ventricle. When the number of descending divisions from the circumflex branch is low, there are larger numbers of left lateral divisions from the ventral (anterior) descending branch and vice-versa.

The two most constant divisions are, firstly, the angular division (Moore 1930) which arises in the angle between the circumflex and ventral (anterior) descending branches and runs obliquely across the left lateral face of the left ventricle. This vessel originates from the circumflex branch in 75% of hearts and from the ventral descending branch in 25% of hearts (Moore 1930). Secondly, the largest of the descending divisions is the left marginal or median division which follows the left margin of the heart to the apex of the heart where it is associated with terminal divisions of the ventral descending branch.

As described above, the dorsal (posterior) descending branch is a direct continuation of the main trunk of the circumflex branch. This vessel is shorter than the ventral (anterior) descending branch and it gives off left and right lateral divisions and septal vessels

to the interventricular septum and the dorsal (posterior) papillary muscle on the left side.

The left lateral divisions are shorter than those on the right side which supply the dorsal (posterior) margin of the right ventricle. The largest of the right lateral divisions is the first (which may arise from the circumflex branch). This vessel runs a parallel course to its parent vessel down towards the apex of the right ventricle. The terminal divisions of this and other right lateral vessels are found near the apex of the right ventricle which is wholly supplied by these vessels. The terminal divisions of the dorsal descending branch do not extend to the apex of the left ventricle.

The most important septal vessel is the first, the dorsal (posterior) septal branch which runs beneath the coronary sinus and terminates in the vicinity of the aortic root. This vessel supplies the atrioventricular node, atrioventricular bundle and the related myocardium of the interventricular septum (Lumb, Shacklett & Dawkins 1959). Other septal divisions supply the dorsal border of the interventricular septum and contribute to the supply of the left dorsal (posterior) papillary muscle.

Ventral (anterior) descending branch

This vessel arises, in conjunction with the circumflex branch, from the left coronary artery and runs caudally across the sterno-costal face of the heart and frequently terminates around the apex of the left ventricle on the dorso-caudal face (Christensen 1962). This vessel gives off left and right lateral and septal vessels. In the first part of its course, it is closely associated with the pulmonary artery.

The left lateral divisions are variable in size and number (see p.20) and may supply the greater part of the left ventricular wall. Pianetto (1939) found 3-7 vessels and Blair (1964) describes three major divisions. The right lateral vessels which supply the ventral margin of the right

ventricle are three to five in number. The first division when present is the largest and supplies the conus arteriosus in conjunction with divisions of the right coronary artery. Blair (1961) found a first right lateral division in 65% of hearts. The septal vessels are small and range from 12-20 in number (Moore 1930). These vessels supply the ventral periphery of the interventricular septum. Christensen and Campeti (1959) consider that these vessels are of major importance in the blood supply to the interventricular septum.

The ventral (anterior) septal branch

This vessel is variable in origin (p.19). It passes directly through the wall of the left ventricle and then turns to run obliquely towards the apex, giving off ascending and descending divisions to the interventricular septum. In the early part of its course, this vessel runs in the fibrous septum beneath the septal cusp of the right atrioventricular valve. In the middle third of the interventricular septum, the vessel penetrates more deeply into the myocardium. Donald and Essex (1954a) estimate that this vessel supplies 70-75% of the interventricular septum, the dorso-caudal portion and the periphery of the interventricular septum being supplied by the septal divisions of the dorsal and ventral descending branches.

The blood supply to the atrioventricular node and bundle is derived from the dorsal (posterior) septal branch (p.32) and the blood supply to the left and right bundle branches and the distal portion of the Atrioventricular bundle is supplied by the ventral (anterior) septal branch which anastomoses with the posterior septal branch (Lumb et al 1959). Christensen and Campeti (1959) consider that the ventral septal branch is of minor importance in the blood supply to the interventricular septum.

The right coronary artery

This vessel arises from one or two inlets beneath the right semilunar cusp of the aortic valve and runs around the right coronary groove

to a point at or just short of the dorsal (posterior) interventricular groove (Kazzaz and Shanklin 1950). In the first part of its course, the vessel is covered by the right atrium and lies caudal to the pulmonary artery and conus arteriosus. The right coronary artery gives off ascending and descending branches.

Ascending divisions

These vessels are small and vary in number from 3-5. Moore (1930) found two major vessels, one proximal vessel which supplies the interatrial septum and right auricular appendage and a distal vessel which supplied the right atrium, interatrial septum and the sinoatrial node, in conjunction with ascending divisions of the circumflex branch of the left coronary artery.

Descending divisions

These vessels are relatively small and vary in number from 4-8. The first branch supplies the conus arteriosus (in conjunction with divisions of the ventral descending branch of the left coronary artery) and may arise (20% hearts) as a separate vessel from the aorta when it is known as the accessory right coronary artery (Moore 1930). The remaining branches are given off at right angles to the main vessel and supply of the right ventricle. The right marginal branch is the largest of these vessels. Donald and Essex (1954) found that the right coronary artery supplied blood to 66% of the wall of the right ventricle. The remainder of this ventricle was supplied by right lateral divisions of the dorsal and ventral descending branches of the left coronary artery.

It is clear from this description of the coronary arterial supply that, in the dog, there is a left preponderance which occurred in 100% of animals studied (Blair 1961).

Anastomoses in the Coronary Circulation

There is abundant evidence of anastomoses between various divisions of the coronary arterial tree of the dog (Moore 1930; Pianetto 1939; Blumgart et al 1941; Blair 1961 and Christensen 1962). These anastomoses have been demonstrated by the use of a variety of injection masses, i.e. barium sulphate and gelatin (Moore 1930), lead-agar mass (Pianetto 1939; Blumgart et al 1941) and vinyl acetate (Christensen 1962).

These inter-arterial anastomoses are seen in almost all dog hearts and may be visible to the naked eye in appropriately treated specimens. This means that they are larger than 60-80 microns in diameter and correspond in size to arterioles and arteries. These anastomoses are of considerable importance in interpreting the results of ligation experiments using dogs and may be responsible for the inconsistency of the results that have been obtained (Blumgart, Zoll, Freedberg and Gilligan 1936).

In addition to the inter-arterial anastomoses, there are anastomoses between coronary arteries and capillaries and the heart chambers themselves through the agency of myocardial sinusoids and arterio-luminal vessels (Thorn et al 1933).

The Cardiac Veins

The majority of cardiac veins are satellites of the corresponding coronary arteries, but the names of the veins do not always correspond. All the major coronary veins drain into the coronary sinus which enters the right atrium through the interatrial septum and just beneath the opening of the posterior vena cava.

The principal tributary of the coronary sinus is the great coronary vein which originates near the apex of the heart and ascends the heart in the ventral (anterior) interventricular groove as a paired vessel. At the atrioventricular groove, the vessel turns and runs around the left side of the heart as a single vessel, to enter the coronary sinus. In the ascending portion of this vein, paired tributaries enter the vessel from the left and right ventricle.

At the junction between the coronary sinus and the great coronary vein, two other vessels enter the coronary sinus. The dorsal vein of the left ventricle runs across the dorsal face of the heart from the apex of the left ventricle and a smaller oblique vein runs from the left atrium.

A middle cardiac vein lies in the dorsal (posterior) interventricular groove and collects tributaries from the left and right ventricle. This vessel is a large paired vein. The small cardiac vein runs around the right atrioventricular groove from the region of the pulmonary ring, collecting tributaries from the right atrium and ventricle. Some of the ventral branches may enter the coronary sinus directly.

Other Vascular Communications with the Heart Chambers

The majority of the venous blood drains from the myocardium via the coronary sinus, but a small proportion returns to all the heart chambers through small channels which anastomose with coronary arteries and veins. These vessels are known as small cardiac veins (Miller et al. 1964). Thebesian veins (Thebesius 1706) and arterio-luminal vessels (Wearn, Mettier, Klumpp and Zschiesche 1933).

Vieussens (1706) first described vessels entering the heart chambers of man and expressed the view that these vessels originated from the coronary circulation. Vieussens saw similar openings in the hearts of calves and sheep. Thebesius (1708) saw similar openings but concluded that these were connected to the cardiac veins. Abernethy (1798) injected two different coloured waxes into the coronary arteries and veins and demonstrated that these openings connected both with coronary arteries and veins. Wearn (1928) was able to demonstrate a connection between these openings and the coronary arteries in celloidin-injected hearts.

Wearn et al. (1933) described the nature and distribution of these vascular channels and their relationship to the coronary arteries. They described vessels of two types:-

Myocardial sinusoids Thin-walled vessels of variable diameter which run a meandering course between the muscle bundles to drain through a common opening into the heart chambers. These vessels anastamose with capillaries and with each other.

Arterio-luminal vessels Shorter, thicker-walled vessels which run a direct course from coronary arteries to the heart chambers.

These authors suggested that both types of vessel may be of great importance in normal cardiac function, but did not suggest what this function might be.

Blumgart, Schlesinger, Davis & Zoll (1941) were unable to demonstrate

any connection between the heart chambers and the various sub-divisions of the coronary tree of the dog used for lead-paper injection meas.

The anatomy of the aorta

The aorta of the dog does not differ significantly from that of any other species. No anatomical features of the aorta are relevant to this study.

The histology of the pericardium

The pericardium is composed of two basic elements. The fibrous pericardium is a dense collagenous sac which enclosed the heart and continues at its apex as a fibrous ligament to the diaphragm. This sac blends with the adventitia of the great vessels at its base.

The serous pericardium covers the surface of the heart and lines the pericardial sac to leave a potential space between them which contains 0.3 - 1.0 ml of a clear, light-yellow, fluid - liquor pericardii. Both layers are composed of mesothelial tissue and the parietal pericardium is supported by collagen fibres. The visceral pericardium is closely applied to the heart and is rich in elastic fibres. This layer is regularly referred to as the epicardium.

The histology of the heart valves

The heart valves are a fold of endocardium supported by fibrous tissue and near the base of the atrioventricular valves. They are also supported by a tongue of atrial myocardium. The atrio-ventricular valves have a series of chordae tendineae extending from the ventricular face of the valves to the papillary muscles.

The histology of the myocardium

The myocardium constitutes the majority of the thickness of the walls of the heart as it lies between the epicardium and endocardium.

The myocardium is composed of branching striated muscle fibres which do not possess a distinct sarcolemmal sheath. The fibres themselves are divided transversely and longtitudinally by a series of disc-like structures - the intercalated discs which divide the muscle fibres into a series of short segments.

Thomas (1957) described the muscular architecture of the dog's heart. This author showed that every muscle fascicle in the ventricles originated from, and was inserted into, the fibrous skeleton of the heart. These fascicles are arranged in three layers, a thin outer layer, a thicker middle layer and a thinner inner layer. In the main, these fascicles pass down to the apex and return to the fibrous skeleton in a figure-of-eight pattern. In the left ventricle only, a cylinder of fascicles encompasses the ventricular lumen in the middle third. These latter fascicles are not seen in the right heart.

The histology of the conduction mechanism of the heart

This system is best described by relating structure with function. The role of the conduction mechanism is to disseminate the regular stimulus originating from vagal and sympathetic nerves throughout the atrial and ventricular myocardium and thereby maintaining a regular and ordered cardiac cycle.

The initial point of excitation lies in the sino-auricular (S.A.) node which may be found at the junction between the anterior vena cava and the right atrium. The S.A. node is a focus of modified myocardial fibres which are directly continuous with the conventional myocardial fibres of the right atrium. These modified muscle fibres show less striation and have a greater diameter than do ordinary myocardial fibres. The connective tissue stroma is more abundant in the S.A. node and many terminal branches of sympathetic and vagal nerves are seen. In addition, numerous small blood vessels are present, these being derived from the anterior septal branch of the left coronary artery.

The impulse spreads from the S.A. node extending, cell-by-cell throughout the right atrial myocardium and on into the left atrium. The fibrocartilagenous skeleton (the atrioventricular rings) of the heart forms a barrier to further extension of this impulse into the ventricular myocardium. However, this barrier is breached at one point, just anterior to the coronary sinus and above the interventricular septum, where a second focus of modified fibres are found, the atrio-ventricular (A.V.) node.

The A.V. node provides a 'bridge' for the extension of this wave of excitation from the atria to the ventricles. The A.V. node has a short caudal extension, the bundle of His, which runs sub-endocardially beneath the base of the septal cusp of the right atrioventricular valve. The bundle of His is short and it soon divides into two branches, the left and right bundle branches, which run down either side of the interventricular septum,

in the sub-endocardium. The muscle fibres of the A.V. node show a transition from those forming the muscle of the node to that of normal myocardial fibres of the right atrium. The A.V. node is rich in connective tissue and the modified muscle fibres lie within this meshwork. These muscle fibres themselves are large and are striated only at the periphery, the centre of these cells being clear and rich in glycogen. Many blood vessels are seen which are derived, in part, from the anterior septal and partly from the posterior septal branches of the left coronary artery. The muscle fibres of the A.V. node, bundle of His and the bundle branches, are all similar in their morphology and it is these cells which transmit the impulse to the ventricular myocardium.

The left and right bundle branches, the former being the larger, give off a series of branches which form a complex interwoven network within the sub-endocardium of both ventricles, these are the Purkinje fibres which transmit the impulse throughout the ventricular myocardium. These cells are similar in character to those of the A.V. node. Some of the Purkinje fibres cross the ventricular cavities in such structures as the moderator band, found in the right ventricle. The impulse spreads from the Purkinje fibres, cell by cell, throughout the ventricular myocardium.

In this way, an ordered sequence of events takes place within the heart during the cardiac cycle. Should any of the modified or conventional muscle fibres of the conduction system be damaged, the cardiac cycle may be disturbed.

The histology of the arteries

All the larger blood vessels, the aorta, arteries and arterioles, have a similar structure, being composed of three basic layers, the tunica intima, tunica media and the tunica adventitia.

The tunica intima or inner coat is lined by a thin, single layer of pavement epithelium, the vascular endothelium. These cells are elongated in the long axis of the vessel, and form a smooth lining. In the larger vessels there is a delicate layer of loose connective tissue, the sub-endothelium, which separates the vascular endothelium from the internal elastic lamina. The internal elastic lamina or fenestrated membrane is a sheet of elastic fibres which defines the lower limit of the tunica intima.

The tunica media or middle coat is composed of a mixture of elastic and smooth muscle fibres. The majority of the muscle fibres are arranged annularly around the vessel and varying numbers of elastic fibres are interspersed between them. Some of these elastic fibres are directly continuous with those of the internal elastic lamina. The muscle fibres themselves are short when compared with those in other viscera and their nuclei are rod-shaped. The tunica media is the thickest of the three basic layers of the arteries and constitutes the majority of the vessel wall. The relative proportions of elastic and smooth muscle fibres varies, being predominantly muscular in medium-sized arteries and arterioles while the elastic tissue is prominent in such vessels as the aorta, particularly the thoracic aorta.

The tunica adventitia or outer coat is composed of loose areolar connective tissue. It is rich in elastic tissue where it joins the tunica media but elastic fibres are less prominent when the tunica media is rich in elastica, as in the aorta for example. The collagen fibres in the tunica adventitia provide protection and support to the walls of these vessels. The outer limits of this layer are ill-defined as it merges

into the surrounding connective tissue.

The aorta shows certain differences from the basic structure of the arteries. In the intima, the sub-endothelium is a thicker and more prominent layer and the internal elastic lamina is less prominent here. The tunica media is rich in elastic fibres, particularly in the thoracic aorta where it forms membranous layers or lamellar networks which alternate with smooth muscle fibres. In addition to these tissues, a considerable number of collagen fibres are present and all these fibrillar elements combine to make the aortic wall very strong. Unlike the tunica media of the majority of the arteries, the aorta and some of its major branches have longtitudinal, as well as annular smooth muscle fibres. These are particularly prominent in the inner third of this layer. The amount of elastic tissue is greater in the thoracic than in the abdominal artery.

In the larger pulmonary arteries, the tunica media is thinner than in vessels of comparable size in the systemic circulation.

3. The pathology of spontaneous cardiovascular diseases in the dog.

This review is prepared in four sections as follows:

A - Chronic valvular disease.

B - The diseases of the circulation.

C - Diseases of the myocardium.

D - Other cardiovascular conditions.

A - Chronic valvular disease

All veterinary surgeons recognise that insufficiency of the left atrioventricular valve is the most common cardiac abnormality that is detected during the clinical examination of the dog (Detweiler 1958; Joshua 1959; Das and Tashjian 1965; Pirie 1965). In addition, this condition has been described as the one most closely associated with congestive heart failure in the dog (Detweiler 1952). Insufficiency of the left atrioventricular valve has been associated with three different cardiac conditions, namely bacterial endocarditis (Shouse and Meier 1956), spontaneous rupture of the chordae tendineae (Ettinger and Buergelt 1969) and chronic valvular disease (also known as valvular endocardiosis and chronic valvular endocarditis). The first record of chronic valvular disease was by Cadiot (1892).

Luginbühl and Detweiler (1965) described the typical lesion in the left atrioventricular valve as smooth, nodular, firm and commonly located at the free margin of the cusp. Whitney (1967) emphasises the close relationship between these lesions and the origins of the chordae tendineae. The early lesion is oedematous, while the later lesions are greyish-white in colour (Das and Tashjian 1965; Sheridan 1967). In the more severe examples of this disease, the cusps bulge convexly on the atrial surface, sometimes curling under the free margin to effectively shorten the cusp. In some instances, the chordae tendineae are also thickened, especially where they join the free edge of the cusp. Severely affected valves may be reduced to a series of smooth, glistening, 'knob-like' swellings which could not possibly be competent (Luginbühl and Detweiler 1965). In a minority of cases the chordae

tendineae may be ruptured (Sheridan 1967). Ettinger and Buergelt (1969) recorded eighteen such cases found in association with chronic valvular disease. Areas of haemorrhage may be seen within the affected valve cusps at any stage of the disease (Das and Tashjian 1965; Luginbühl and Detweiler 1965; Sheridan 1967; Whitney 1967).

The lesions of chronic valvular disease are not confined to the left atrioventricular valve. Luginbühl and Detweiler (1965) found lesions in the left atrioventricular valve in 61.5% of cases, in the right atrioventricular valve in 22.6% of cases, the aortic valve in 2.9%, but no lesions were seen in the pulmonary valve. Bretschneider (1962) found lesions in 57.3% of left atrioventricular valves, in the left and right atrioventricular valves of 26.6% of cases, in the right atrioventricular valve alone of 7.5% of cases, 2.1% of aortic valves and 0.4% of pulmonary valves. Das, Patnaik, Lin and Irschlinger (1965) found lesions in 46.0% of left atrioventricular valves, 30.0% of right atrioventricular valves, 9.0% of aortic valves and 2.5% of pulmonary valves. Sheridan (1967) found lesions in 88.0% of left atrioventricular valves, 70.0% of right atrioventricular valves (34.0% in septal cusp only, 34.0% septal and mural cusp, 2.0% in mural cusp only), 36.0% in aortic valves and 8.0% in the pulmonary valve. Munich (1935) and Stunzi and Teuscher (1962) confirmed these observations. It is evident from these results that chronic valvular disease occurs, in descending order of frequency, in the left atrioventricular valve, right atrioventricular valve, aortic valve and pulmonary valve.

In a clinical study of 4,831 dogs, Detweiler and Patterson (1965) found 545 cases (11.3%) with cardiovascular disease. Of these, 27 (0.6%) had congenital cardiac diseases, 391 cases (8.1%) had chronic valvular disease (which included 94 cases (1.9%) which were complicated by myocardial disease), myocardial disease (without chronic valvular disease) in 68 cases (1.4%) and a further 59 cases (1.2%) had a variety of other

cardiovascular conditions. Bretschneider (1962) examined a series of 688 cases of valvular endocarditis at post-mortem and found chronic valvular disease in 83.3% of left atrioventricular valves. In a series of 550 unselected post-mortems, Das and Tashjian (1965) saw lesions in 42.0% of left atrioventricular valves. Sheridan (1967) found 88.0% with lesions in the left atrioventricular valves. Whitney (1967) saw lesions in the left atrioventricular valve of 97.0% of dogs, nine to twelve years old inclusive.

A number of authors have described the histopathological changes in chronic valvular disease. Das and Tashjian (1965) described the lesion as a proliferation of loose connective tissue with no reactive cells, Aschoff bodies or endocarditis. In a few cases, myxomatous changes and acid mucopolysaccaride material were identified. The initial change was an oedema of the valve cusp. Luginbühl and Detweiler (1965) saw a fibrous and elastic connective tissue proliferation with oedema and mucinous degeneration. These changes involved the auricularis (atrialis), the spongiosa and, in the more severe lesions, the fibrosa. Fragmented collagen and elastic fibres were seen in these lesions. Cells resembling fibroblasts and other cells, which could not be identified, were also observed. Recent and old haemorrhages and haemosiderin were found with some frequency. Jones and Zook (1965) found masses of hyaline or 'fibrinoid' material and a few leucocytes (lymphocytes or plasma cells) in the thickened valves. Wallace (1965) found that the leaflets were thickened with a mucoid connective tissue. Pirie (1965) saw a disruption of the normal architecture and a proliferation of the atrialis, in the area of contact, and, secondly, a mucinous degeneration of the spongiosa and fibrosa. Such mucinous material in the valves is derived from the fibroblasts (Bunting 1950). A concensus of these opinions suggests that these lesions are both proliferative and degenerative and that there is an accumulation of metachromatic ground substance within the valve.

In an attempt to investigate the pathogenesis of chronic valvular disease, Whitney (1967) classified the gross lesions into five groups. Normal valves, Type I and Type II lesions which were discrete nodular lesions of a type that was not associated with insufficiency, and Type III and Type IV, larger nodules and plaque-like deformities where there was evidence of valvular insufficiency and congestive heart failure in some cases.

Certain aspects of the epidemiology of chronic valvular disease have been considered. One aspect that has received particular attention is the age distribution; Whitney (1967) found chronic valvular disease in 35.0% of dogs under five years and in 97.0% of dogs nine to twelve years inclusive . Wallace (1965) found that 40.0% of cardiovascular disease in dogs over six years was due to chronic valvular disease. Munich (1935) found 77.4% of a series of 93 cases of chronic valvular disease were over six years old. Das and Tashjian (1965) demonstrated an apparent linear age relationship in 550 unselected post-mortems but they also emphasized that chronic valvular disease was not confined to aged dogs. Jones and Zook (1965) showed a similar age relationship and concluded that age appeared to be a significant factor. Detweiler, Hubben and Patterson (1960) commented on the sharp rise in the frequency of chronic valvular disease in dogs over seven years old. Pirie (1965) suggested that the disease was more severe in dogs over four years than in dogs under four years. Detweiler and Patterson (1965) state that chronic valvular disease shows a marked increase in prevalence with age. Thus, there is evidence on the data at present available, that ageing is concerned in the pathogenesis of chronic valvular disease (Pomerance and Whitney 1970).

It is not clear whether sex influences the frequency of chronic valvular disease. Munich (1935) found no sex predisposition. Das and Tashjian (1965) supported this view but added that congestive heart failure was more common in the male. This view was supported by Detweiler and

Patterson (1965). In a later paper (Detweiler 1966), the senior author reaffirmed this opinion and suggested that acquired heart disease developed more rapidly in the male than in the female.

The position seems clearer in relation to breed predisposition to chronic valvular disease. While Munich (1935) found no breed predisposition, other authors found a greater susceptibility in the Boxer (Das and Tashjian 1965) and the Cocker Spaniel (Das and Tashjian 1965; Detweiler and Patterson 1965).

Only Munich (1935) attempted to correlate chronic valvular disease with other intercurrent diseases, namely, chronic interstitial nephritis and canine distemper. He found no direct correlation between them.

Having considered the nature, the gross and microscopic changes, the distribution of lesions and some aspects of the epidemiology of chronic valvular disease, some consideration must be given to the effect of these lesions upon the cardiovascular system as a whole. It should be remembered that the valve mechanism is not restricted to the valve cusp, with or without the chordae tendineae; the valve mechanism includes the annulus, the valve cusp, the chordae tendineae (in the case of the atrioventricular valves) the papillary muscles and the root of the aorta or pulmonary artery (in the case of the semi-lunar valves). Changes in any one, or a number of these structures may influence the function of the valve as a whole. Another basic factor worthy of consideration is the fact that an insufficiency of the left atrioventricular valve, for example, does not mean that a clinical condition exists in the absence of other signs, e.g. dyspnoea, cyanosis, ascites, enteritis, etc (Joshua 1959). A dog will survive for months or even a year or two with a valvular insufficiency before any congestive heart failure develops (Detweiler and Patterson 1965).

These valve changes seem to develop slowly within the cusp and the heart possesses a great ability to accommodate to these defects in its

function; this is the cardiac reserve. From the clinical standpoint, supported by post-mortem examinations, the insufficiency of the left atrioventricular valve appeared to be the principle problem. Sheridan (1967) found moderate or severe insufficiency in the left atrioventricular valve in 13.0% of dogs. He classified these malformations into three groups, according to the changes that took place at the free borders of the cusps. In most cases, insufficiency was the result of contraction of the cusps so that the regurgitant jet passed through the central area of the annulus (central pathway) during systole. In some cases, there was excessive convolution of the free border so that a series of small apertures developed in the central pathway during systole. In 4.0% of cases, the insufficiency developed due to 'ballooning' and eversion of the cusp, during systole. This may be further complicated by rupture of one or more of the chordae tendineae. In these cases, described by Sheridan, it was clear that both the ventral and dorsal cusps of the left atrioventricular valve were involved. No information appears to be available for the dog on the relative size of these two cusps but in man Brock (1952) showed that the anterior mitral (ventral cusp) was 33.3% larger than the posterior mitral (dorsal cusp). If one cusp only was affected, the flexibility of the other will enable it to conform to many of the irregularities of the diseased partner and reduce insufficiency to a minimum.

In the right atrioventricular valve, Sheridan (1967) found 10% with insufficiency, eight of which involved the mural cusp, two involved the septal cusp. It is not known what the relative contributions these two cusps make to the closure of the right atrioventricular orifice during systole.

As a result of these valve deformities and the insufficiency of the cusp, a regurgitant stream escapes back into the atrium during systole. This blood may 'jet' on to the anterior endocardium from the central path-

way or laterally from a 'ballooning' defect to cause a similar, local fibro-elastic proliferation of the atrial endocardium (Das and Tashjian 1965; Sheridan 1967).

This reflux of blood causes a dilatation of the atrium which is followed later by hypertrophy of the atrial myocardium. This increased volume in the atrium passes into the ventricle, causing it to dilate, also. As the ventricle has now to pump additional blood to compensate for the insufficiency, the ventricular myocardium hypertrophies too. Therefore, in cases of insufficiency of the left atrioventricular valve in the dog, the left atrium and ventricle show hypertrophy with dilatation. In this way the cardiac output is restored to a normal 3.3-4.0 litres/minute (Hülsmann 1967).

It is suggested that chronic valvular disease develops in early life, causes mitral, and possibly tricuspid, insufficiency in middle life and may cause clinical disease in later life. This insufficiency is well tolerated in many dogs (Detweiler and Patterson 1965). If this assumption is correct, the heart has to continue to compensate for the further valvular insufficiency until a point is reached when no further compensation is possible. The first breakdown that appears is in the lungs which develop a pulmonary congestion and oedema due to a 'back-up' of pressure from the left atrium (Hamlin, Smetzer and Smith 1965). This, in turn, causes increased peripheral resistance in the pulmonary circulation (and hypoxia) which results in hypertrophy and later dilatation of the right ventricle. Detweiler (1958) suggests that tricuspid insufficiency is secondary, being due to dilatation of the valve ring. The failure of the right heart leads to chronic venous congestion of the abdominal viscera and ascites which, in turn, increases the peripheral resistance in the systemic circulation. This, coupled with hypoxia, results in dilatation of the left heart and the vicious circle is completed - left and right heart failure. Detweiler and

Patterson (1965) estimated the prevalence of congestive heart failure in a clinical study in the Philadelphia area as 14:1000. Das and Tashjian (1965) found congestive heart failure in 12.0% of a series of 550 unselected post-mortems. It is clear that valve lesions are present in many clinically normal dogs. The heart becomes compensated to these valvular abnormalities and the normal cardiac output may be maintained for a considerable period; congestive heart failure only occurs in a distinct minority of cases.

In essence, heart failure is myocardial failure and the ability of the heart to compensate for valvular insufficiency depends to a considerable extent upon the integrity of the myocardium. Any condition which damages the myocardium reduces the cardiac reserve and hastens the onset of congestive heart failure. So-called 'small vessel disease' is such a condition which will be referred to in detail later. Small vessel disease affects the intra-mural coronary vessels and is associated with circumscribed foci of myocardial necrosis and fibrosis. These two changes, coupled with chronic valvular disease, make up a triad of changes that are frequently associated with congestive heart failure (Luginbühl and Detweiler 1965). Mitral stenosis is rarely reported in the dog - one report exists where stenosis was seen in association with chronic valvular disease in the mitral valve (Tashjian and McCoy 1960).

In considering the aetiology of chronic valvular disease, it must be borne in mind that these valve lesions are age dependent (Pomerance and Whitney 1970). Thus in considering the changes that take place in the valve, one must consider how many of these lesions are normal ageing changes and how many are due to a pathological process. No such information is available at this time to differentiate between normal ageing changes and disease processes in the heart valves of the dog.

Pensinger (1965) reports that the chronic changes which involve

the cusp tissue are due to a progressive fibro-elastic proliferation of the valve. Whitney (1967) described a fibro-elastic proliferation of the atrialis (in the area of contact) which he suggested was due to friction and impact, during systole. Valve lesions are more common on the left side of the heart where the blood pressure is higher than on the right side. In an experimental study, Nedzel (1937) used pitressin, by injection, for varying periods of time to produce oedema, haemorrhage and fibrosis in the area of contact of dogs. The pitression increased the work-load and the impact on the valve cusps - there was also a relative local anoxia. Lillehei, Bobb and Visscher (1950) produced lesions, similar to chronic valvular disease, in young and old dogs by means of arterio-venous fistulae in four to six weeks. These fistulae lowered the peripheral resistance and increased the work-load on the heart. There was no significant increase in blood pressure. Pirie, Mackey and Fisher (1965) did not see any significant valve lesions in cases of renal hypertension in the dog.

Altland, Highman and Roshe (1959) exposed a series of dogs to decompression, following mechanical injury to the aortic and/or left atrio-ventricular valve, and produced valve lesions similar to chronic valvular disease. These authors suggested that hypoxia and/or decompression was concerned in the pathogenesis of these valve lesions.

Shouse and Meier (1956) studied a series of cases of bacterial endocarditis in dogs and suggested this condition was common in the dog and that chronic valvular disease represented the healed phase of a bacterial endocarditis. In a series of 69 cases of heart disease, Detweiler et al (1960) found two cases of bacterial endocarditis. Das et al (1965) found 23 cases of bacterial endocarditis in a series of 730 unselected post-mortems which included 193 cases of chronic valvular disease. Munich (1935) saw 8 cases of bacterial endocarditis in a series of 100 cases of valvular disease in the dog. Sheridan (1967) saw four cases in a series of similar

size of what he implied were healing cases of bacterial endocarditis. (Chronic valvular disease was seen in 88 cases in this series.) Stunzi (1962) states that acute valvulitis is a response to a bacterial infection, whereas, chronic valvular disease is not inflammatory in origin but is a dystrophy. Sheridan (1967) saw acute valvulitis in young dogs and suggested that such charges may lead to chronic valvular disease in later life.

Patterson (1963) and Hamlin, Smetzer and Smith (1965) described cases of congenital mitral insufficiency due to myocardial failure. This was a secondary incompetence due to dilatation of the valve ring.

Bohmig and Klein (1953) commented upon the ready allergic reaction of the sub-endothelium of the heart valve of the rabbit. Detweiler (1959) suggested that a similar antigen-antibody relationship may produce valve lesions in the dog. There is no direct evidence to confirm or refute this suggestion.

Angrist, Oka, Nakao and Marquiss (1960) demonstrated the ability of various forms of non-specific stress to initiate changes in the heart valves of the rat. These lesions are characterised by oedema, swelling and eosmophilia of the collagen and vacuolation of the ground substance. The endothelial cells are lost and micro-thombi develop on the surface - later these thrombi become organised. These authors referred to this condition as non-bacterial thrombotic endocarditis which they regarded as the basic valve lesions which may develop into a bacterial endocarditis or a lesion of a degenerative type. With organisation, the micro-thrombi may form Lambi's excresences. Oka and Angrist (1963) found these valve lesions rich in acid mucopolysaccharide and hyaluronic acid. Angrist and Marquiss (1954) suggest that there is a basic fault in the collagen metabolism which accounted for these changes. Oka and Angrist (1963) felt that there was some hormonal disturbance on the pituitary-adrenal axis involved in the

pathogenesis of these lesions.

Angrist (1965) stated that the lesions in the dog were similar in many respects to those which occur in nodular or senile sclerosis in man. Pomerance (1966) described the lesions of nodular sclerosis in the aged as a fibro-elastic proliferation of the atrialis in the area of contact of the atrioventricular valves. She suggested that these lesions were a response to repeated impact, analagous to cutaneous callus formation and that hypoxia was important in the pathogenesis of these lesions. Ashworth (1946), McMillan and Lev (1964) and, more recently, Pomerance (1967) have described atheromatosis of the mitral and aortic valves. Such lesions are most readily observed around the margins of the aortic valve cusps and in the proximal portions of the mitral valve, particularly the anterior cusp. Such lesions involve the inflow surface of the cusps, affecting the ventricularis (arterialis) and the subjacent fibrosa in these areas where fine fat droplets appear associated with cholesterol deposits - this fat being extra-cellular initially. Mönckeberg's sclerosis develops in these same sites.

Cka (1962), Pomerance (1967; 1969) and others have described the changes associated with mucoid degeneration of the valve cusp in man. These changes were only seen as naked-eye lesions in 7.0% of cases over forty-five years old. Severe lesions with ectasia of the cusps ('parachute' deformity, 'ballooning' degeneration, 'floppy valve' disease) were seen in less than 1.0% of 3000 cases examined (Pomerance 1969). Pomerance and Whitney (1970) compared these human conditions with chronic valvular disease in the dog and stated that these lesions were similar in the two species. These authors concluded that the nodular thickenings and lipoid depositions were possibly related to haemodynamic stimulae, a view supported by McMillan and Lev (1964) who considered that these atheromatous lesions are a response to local turbulence on the cusps. However, these valve changes are of

little clinical significance. In contrast, mucoid degeneration may produce a 'ballooning' degeneration and ectasia of the cusp which results in valvular incompetence. Such lesions are more prevalent in the dog.

McMillan and Lev (1964) described the ageing changes in the human heart valves. They described the development of the fibro-elastic plaques in the areas of contact and concluded that these were a response to repeated impact on the opposing surfaces. These authors described a proliferation of elastic and collagen fibres in the spongiosa opposite the origins of the chordae tendineae and a fibro-elastic thickening around the chordae tendineae themselves. They suggest that these changes and a response to 'tensing' the chordae tendineae and, in part, to local turbulence around them. All these changes are more pronounced on the left side than on the right side and these authors suggest that this is due to the higher systemic pressure. No similar study has been undertaken in the dog.

B The diseases of the circulation

There has been a great interest in the study of the vascular diseases of animals in the last decade. Much of this work has stemmed from an urgent need to increase our understanding of atherosclerosis in Man by these comparative studies. The dog has been a popular experimental animal for the investigation of a variety of cardiovascular conditions, including atherosclerosis. A considerable volume of work had been undertaken long before the anatomy, histology and pathology of the spontaneous cardiovascular diseases of the dog were known. There is little doubt that a fuller appreciation of experimental results would have been possible had this basic data been available at that time. In 1971, our understanding and knowledge of canine vascular diseases is improved.

The term 'arteriosclerosis' (Lobstein 1933) is employed as a generic name for any arterial thickening. Thus, this term may be used to cover a variety of conditions, including intimal fibrous thickenings, medial thickening, intimal and medial thickening, generalised atherosclerosis or xanthomatosis, medial calcification, polyarteritis nodosa, hypertensive changes, necrotic endarteritis, hyalinosis of intramural coronary vessels, hyalinosis of splenic vessels and intimal and medial hyperplasia of pulmonary vessels (Dahme 1962). In this context, attention is directed towards the aorta and the coronary circulation but arteriosclerosis may be found anywhere in the circulatory system.

In the majority of the common domestic species, arteriosclerotic changes are uncommon, probably due to their limited life-span under current commercial farming practice, but the dog, cat and domestic cage-bird have a more normal life-span and all show marked changes in their blood vessels (Dahme 1962).

Atherosclerosis is not a term that is applied to vascular lesions in animals for, in the main, fat deposition and gross disorganisation of the vessel wall are not observed.

The earliest paper on arteriosclerosis in the dog described the aortic lesions (Kollisch 1910). This paper and others largely concerned with the aorta were reviewed by Ackermecht and Krause (1929). In 1952, Lindsay, Chaikoff and Gilmore published a most excellent paper on coronary and aortic arteriosclerosis which was a significant landmark in this field. Morehead and Little (1945) emphasised the importance of the changes in the aortic media of the older dogs.

The gross changes may be seen in the aorta of dogs at all ages. The earliest detectable lesions appeared as smooth, oval, raised areas, 1-2 mm in diameter which were situated at points close to the inlets to branches from the abdominal aorta and common iliac vessels (Davies and Reinert 1965). In older dogs, these lesions appeared as greyish raised streaks, irregular plaque formations and diffuse intimal thickenings. These various manifestations were seen throughout the descending aorta but they were always more numerous and extensive in the abdominal aorta towards the bifurcation than elsewhere (Luginbühl, Jones and Detweiler 1965). These lesions were most readily observed on the posterior (dorsal) wall of the aorta (Davies and Reinert 1965).

Lindsay et al (1952) described the gross lesions of canine arteriosclerosis at different levels of the aorta. In the aortic arch there was a diffuse wrinkling and thickening of the intima on the concave (cranial) curve of the arch.

In the thoracic aorta, small oval plaques from 1 mm up to 2 cm were observed, caudal to the inlets to the intercostal branches. Similar plaques were sometimes seen around the inlets to the left and right coronary arteries which tended to occlude these inlets in some cases. These lesions tended to increase in number rather than in individual size with advancing age (Davies and Reinert 1965).

Lesions were more frequently observed in the abdominal aorta than

in the thoracic aorta. As in the thoracic aorta, these lesions were associated with the inlets to branches. They varied in shape from round to oval or elongated. These lesions were most severe on the posterior (dorsal) wall of the abdominal aorta and became more extensive caudally as the aortic bifurcation was approached. These lesions extended into the common iliac arteries where they appeared as a series of linear elevations at right angles to long axis of the vessel. No gross changes were detected in the anterior aorta (Davies and Reinert 1965).

Grossly, no fat was detected in these lesions (Lindsay et al 1952; Davies and Reinert 1965) but macroscopic staining with Sudan IV did reveal the presence of lipid in one case in a series of 315 dogs (Hindawy 1959).

Upon histological examination of these lesions, the earliest detectable change was an oedema of the intima and inner third of the media which Dahme (1962) interpreted as an indication of disturbed permeability within the endothelium and sub-endothelium. Associated with these changes, there was a beading, splitting, fraying or re-duplication of the internal elastic lamina. Furthermore, in many cases the internal elastic lamina was absent within the lesion (Lindsay et al 1952; Davies and Reinert 1965). A loose meshwork of delicate reticulin and collagen fibres developed within the sub-endothelium and a mucoid ground was demonstrable in some cases. Within this fibrillar matrix, linearly arranged strands or clumps of smooth muscle cells were seen, each surrounded by fine elastic fibres (Luginbühl et al 1965). Smooth muscle cells were also seen lying at right angles to the intimal surface within gaps in the internal elastic lamina.

In the early arteriosclerotic lesion, the fibrillar elements were loosely arranged. In the thoracic aorta, the larger arterioclerotic lesions were rich in elastic tissue which may be disorganised or arranged in thick fibres parallel to the internal elastic lamina. The larger lesions in the abdominal aorta had less elastic tissue and more collagen. The intima

between neighbouring arteriosclerotic plaques may show a variable degree of diffuse intimal hyperplasia (Lindsay et al 1952).

The larger intimal plaques were laminated, the superficial layer being loose-knit and cellular, while the deeper layers were relatively a-cellular and composed of densely packed collagen which may show a hyaline degeneration. A variable amount of elastic tissue was also present, particularly in the 'cap-like' lesions at the entrance to the smaller branches. The deeper layers were sometimes mucoid (Lindsay et al 1952) and vascularisation was sometimes evident (Luginbühl et al 1965). Fat was rare in these lesions (Lindsay et al 1952; Davies and Reinert 1965; Lugenbühl et al 1965).

In association with these intimal lesions, changes were also seen in the inner third of the media. In this area, there was a loss of elastic fibres and the development of the cyst-like spaces filled with mucin. Later this mucin was replaced by fibrous tissue. Areas of medial calcification may also be seen in association with degeneration of the media. Such changes were most regularly seen in the aortic arch. Hindawy (1959) found six such cases in a series of 92 cases of aortic arteriosclerosis. Aggregates of smooth muscle cells were seen (Morehead and Little 1945).

Similar fibrous plaques may be seen in the renal arteries (Davies and Reinert 1965).

Hindawy (1959) found 92 (29.2%) cases of aortic arteriosclerosis in a series of 315 dogs. No gross lesions were detected in dogs under one year old; this finding was not supported by Davies and Reinert (1965). Hindawy found two cases in 52 dogs, one to two and a half years old. In older animals, the incidence increased with age. Das et al (1965) found 256 (35.0%) cases of aortic arteriosclerosis in a series of 730 dogs. Morehead and Little (1945) found intimal plaques in dogs aged ten days.

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Gross lesions are uncommon in the coronary vessels, excepting those in the rare cases of xanthomatosis (Bloom 1946) and polyarteritis nodosa (Dahme 1962). The only gross lesions that are regularly seen in association with coronary disease are areas of myocardial necrosis and fibrosis that have been associated with so-called small vessel disease.

In describing the histopathology of coronary disease, it is necessary to distinguish between those lesions found in extramural, coronary vessels and those found in intramural vessels as there are certain differences between them. The lesions found in extramural vessels are similar in character to those seen in the aorta. Similar changes were seen in the internal elastic lamina and smooth muscle cells were evident within the lesion and lying in gaps within the elastica (Luginbuhl et al 1965). Reticulin, collagen and elastic fibres were seen within the intima, while collagen was predominant in the larger plaques (Lindsay et al 1952). Lesions such as these were seen in the anterior descending and circumflex branches of the left coronary artery and in the right coronary artery. (Morehead and Little 1945; Lindsay et al 1952; Davies and Reinert 1965; Luginbuhl et al 1965). Alcian blue positive material was present in some of these lesions but no fat was observed (Davies and Reinert 1965). None of these lesions were sufficiently large to cause occlusion of the vessels and no thrombosis was observed.

In the intramural coronary vessels, similar intimal lesions were observed to those seen in extramural branches, namely, a predominantly collagenous plaque. Smooth muscle cells are seen in these plaques and Luginbuhl et al (1965) also found single or multinucleate giant cells, some of which had branching cytoplasmic processes. These intimal plaques may reach considerable proportions, involving the whole circumference of the intima, thereby reducing the lumen of the vessel. The endothelium was unaffected and no thrombosis was seen.

In addition to these basic lesions, certain other changes took place,

particularly in those vessels supplying the papillary muscles of the left ventricle. These changes were characterised by the accumulation of a homogeneous, PAS-positive material within the vessel wall. In mildly affected vessels, this material accumulated on either or each side of the internal elastic lamina both interstitially and within the smooth muscle cells. Luginbuhl et al (1965) referred to this material as hyalin. In more severely affected vessels, hyalin may be observed in eccentric plaques, in part of the circumference, or involving the whole vessel wall. The internal elastic lamina was generally absent from such vessels and the lumen was considerably reduced. Indeed, the lumen may be reduced to a series of small clefts or channels containing red blood cells and/or haemosiderin. Some of these cavities were lined with a vascular endothelium. A few foam cells may be seen in some of these severely affected vessels (Luginbuhl et al 1965). Lindsay et al (1952) stained such vessels by the iron-Prussian blue method to demonstrate a metachromasia which could not be removed by testicular hyaluronidase.

Braunmühl (1956) and Dahme (1962) described changes in the medium-sized and larger cerebral vessels of old dogs in a condition now known as congophilic angiopathy. Dahme (1965) pointed to the similarity of these lesions to the hyalin lesions found in the intramural coronary vessels. Luginbuhl (1966) found cardiac and cerebral hyalin vascular deposits in 28 or 30 dogs over twelve years old. These lesions stained positively with congo red, Thioflavine T and were positive with polarised light. Dahme and Deutschlander (1967) concluded that this lesion was a primary amyloidosis.

Detweiler and Patterson (1965) state that clinical evidence of myocardial disease is relatively rare in dogs under nine years, after which time the diagnosis may be made in 9.0% of cases. One common cause of myocardial disease in the dog was a myocardial necrosis and fibrosis which has been

associated with intramural coronary disease. Detweiler et al (1960) found such coronary lesions in 54 of 69 hearts examined histologically. These lesions were associated with myocardial necrosis and fibrosis in many cases. The extramural coronary vessels were unaffected in these cases. Detweiler and Patterson (1965) emphasise the close relationship between chronic valvular disease, small vessel disease and myocardial necrosis and fibrosis. This triad of changes is frequently associated with cases of congestive heart failure in the dog. Das et al (1965) found 39 cases of myocardial necrosis in 730 unselected dogs.

In addition to the coronary lesions described above, certain other arteriosclerotic conditions have been described in the dog. One of the most spectacular of these is generalised atherosclerosis or medial Xanthomatosis, a condition first described by Bloom (1946) in the dog. Later Luginbuhl et al (1965) described a further three cases in which the vessels supplying the brain and heart were the most severely affected. Grossly, the vessels are yellow in colour, thickened and tortuous. This rare condition affected both the intima and media of muscular and musculo-elastic arteries, throughout the body. These vessels showed great accumulations of extra-cellular and intra-cellular fat associated with cholesterol and calcium deposits. The endothelium was intact and thrombosis, ulceration and haemorrhage were not seen. In all cases, the thyroid glands were atrophic. Serum lipids and cholesterol levels were greatly elevated (Dahme 1965; Head and Whitney 1967).

Dahme (1962) recorded three cases of polyarteritis nodosa affecting the coronary arteries of the dog. The affected hearts showed white focal lesions scattered throughout the atria and ventricles. Histological examination of the affected vessels showed a necrosis and an acute inflammatory reaction in and around the affected vessel.

Pirie, Mackay and Fisher (1965) described changes in a variety of vessels,

including coronary vessels that they associated with renal hypertension. These changes were characterised by fibrinoid necrosis, hyalinosis and medial hyperplasia. In addition to these changes, necrotic endocarditis and endarteritis may be seen, particularly in the left atrium and around the inlets of the pulmonary veins. The exact aetiology of these lesions is not known but they are believed to arise from the uraemia that is associated with renal failure.

In surveying the lesions of the blood vessels of the dog, it is clear that in the fibrous intimal plaque is the common lesion, occurring as it does in the descending aorta, extra and intramural coronary vessels. This lesion is akin to the prototype lesion described by Gresham and Howard (1961). Morehead and Little (1945) and Lindsay et al (1952) suggest that the medial lesion is age-related. This view is not supported by Davies and Reinert (1965). Hyalinosis or primary amyloidosis of the intramyocardial coronary vessels appears to be the only condition of any real clinical significance.

C Diseases of the myocardium

Cardiac failure is essentially myocardial failure and so any condition that affects the myocardium directly or indirectly is significant. Nonetheless, the myocardium possesses the ability to compensate for functional defects within the heart; this is the cardiac reserve. There are three important factors that have to be considered in relation to the cardiac reserve, firstly, the extent of the lesion, secondly, the speed with which a lesion develops. Spontaneous rupture of the chordae tendineae results in acute cardiac failure and death within a few hours - the magnitude and speed of onset of the lesion does not allow the heart the time for compensation (Ettinger and Buergelt 1969). When rupture of the chordae tendineae occurred as a complication of chronic valvular disease, although the cardiac condition deteriorated, no acute cardiac crisis developed. The heart had already compensated for the valvular insufficiency and despite the ruptured chordae, the dog was able to survive. The third important consideration in cardiac compensation is the integrity of the myocardium itself, a diseased myocardium cannot necessarily compensate for every functional defect that may arise. This is most clearly seen in relation to chronic valvular disease which is often associated with hyalinosis of intramural coronary vessels and myocardial necrosis (Detweiler et al 1960). These myocardial lesions reduce the ability of the heart to compensate for the valve lesions.

The relative size of the heart chambers and the thickness of the ventricular walls is the measure of the presence or absence of cardiac compensation. Heart weight is a useful guide, the heart weight/body weight ratio in dogs is wide, 5.0-11.0. Northrup et al (1957) state that any ratio in excess of 10 suggested a cardiac hypertrophy but Luginbuhl and Detweiler (1965) found that ratios of 11.5-17.3 in racing greyhounds were not uncommon. The ratio of the weight of left ventricular free wall :

right ventricular free wall has been assessed as 1.82 to 0.205 in a group of 46 dogs (Latimer 1961). Therefore, cardiac hypertrophy of one or both sides of the heart is a useful measure of cardiac compensation. As a result of functional disturbances, the myocardium has to work harder and therefore undergoes a hypertrophy to compensate for the defect.

Hypertrophy may or may not be associated with dilatation of the heart chambers. The heart chambers are dilated in cases of valvular insufficiency (see p.42) but not in cases of increased peripheral resistance. In the systemic circulation, left ventricular hypertrophy has been associated with chronic interstitial nephritis and other types of renal disease (Platt 1952, Dahme 1955, 1957; Persson, Persson and Asheim 1961; Pirie et al 1965). There is some doubt as to whether renal hypertension exists in the dog but Anderson and Fisher (1967) have been able to demonstrate elevated blood pressure readings in such cases. In the pulmonary circulation, right ventricular hypertrophy has been seen in cases of pulmonary oedema and Diofilaria immitis infection of the pulmonary vessels.

Dilatation of the heart may follow hypertrophy or may occur spontaneously as in cases of acute congestive heart failure. The atria may dilate in any condition which increases inter-atrial pressure and this occurs most commonly in association with valvular insufficiency. Sometimes the atria may rupture, Stunzi and Teuscher (1962) describe atrial rupture in cases of valvular insufficiency, which occurred most regularly in Dachshunds. Ventricular dilatation with thinning of the walls, dilatation of the valve ring and flattening of the papillary muscles is seen most regularly in the right ventricle. Such a dilatation is often observed in association with pneumonia or congestion and oedema of the lungs. Aneurysmal dilatation of the apex of the right ventricular is occasionally observed (Danhof 1960).

A variety of degenerative lesions have been described in the myocardium. Myocardial atrophy may be seen in association with chronic wasting

diseases. Cloudy swelling and fatty change have been associated with febrile diseases, anaemia and poisoning by heavy metals. Amyloid has not been seen outside the coronary circulation (Stunzi and Teuscher 1962). Calcification has been seen in association with myocardial necrosis and over-dosage with Vitamin D (Blazek 1959).

Myocardial necrosis will follow advanced stenosis or total occlusion of the coronary circulation. Necrosis was often associated with hyalinosis or primary amyloidosis of the intramural coronary vessels. This condition causes a narrowing of the lumen of the vessel and it has been suggested that this change results in a peripheral ischaemia and necrosis of the related myocardium. Detweiler et al (1960) found 17 such hearts in a series of 69 cardiac cases. Das et al (1965) found 39 cases in a series of 760 post-mortems. These ischaemic lesions were small (up to several millimetres in diameter) and were sometimes soft and friable or firm; they varied in colour from reddish-brown to grey and they were surrounded by a zone of congestion and haemorrhage in some cases. Histologically, they were composed of muscle fibres in varying stages of necrosis, resorption and fibrosis. These lesions were seen most regularly in the left ventricle and particularly in the papillary muscles (Detweiler et al 1960).

Embolism of the coronary vessels may cause a myocardial necrosis in association with the rare cases of bacterial endocarditis. Frank myocardial infarction was observed in six cases in a series of 314 post-mortems (Luginbühl and Detweiler 1965) but only one of these was detected clinically in a total of 4,010 examinations. Similar cases may arise from a pulmonary thrombus, associated with Diofilaria immitis infestation.

Inflammation of the myocardium, myocarditis, may be found in up to 10.0% of dogs (Luginbühl and Detweiler 1965). In many cases, the aetiology of these lesions was unknown and unsuspected (Pirie 1965). Acute interstitial myocarditis has been associated with rupture of the left atrium (Stunzi and Teuscher 1962). Acute non-suppurative myocarditis was a feature

in conditions such as leptospirosis, hepatitis, contagiosa canis and toxoplasmosis. Acute suppurative myocarditis may be associated with septic emboli from a bacterial endocarditis. Chronic granulomatous lesions of the myocardium were seen in cases of tuberculosis, blastomycosis and coccidiomycosis (Stunzi and Teuscher 1962).

Nematode larvae may also produce granulomatous focal lesions in the myocardium. Generally, such lesions are a-symptomatic but they may produce a cardiac arrythmia (Patterson, Detweiler, Hubben and Botts 1961).

Tumours are not uncommon in the dog's heart. Luginbühl and Detweiler (1965) found seventeen (5.5%) in a series of 309 post-mortems, only one, a haemangiosarcoma, was considered to be a primary tumour. Das et al (1965) found three primary tumours, two of which were haemangiomas and fourteen secondary tumours, seven of which were melanotic tumours. Luginbühl and Detweiler (1965) listed the common primary cardiac tumours of the dog as haemangioendothelioma, mixed cell sarcoma, spindle cell sarcoma, round cell sarcoma, lymphangioendothelioma, haemangioma, fibroma, rhabdomyoma, chondroma, fibromyxoma, myxoma and teratoma. A series of 31 primary cardiac haemangiosarcomas were seen most frequently in old dogs affecting the right atrium (Kleine, Zook and Munson 1970). These tumours which were found most commonly in Alsatians, were located near the entrance to the auricle. Dobberstein and Tamaschke (1962) found that the most common secondary tumours were adenocarcinomas of the mammary gland and thyroid. Sandersleben (1961) states that lymphosarcomas are rare in the myocardium but Luginbühl and Detweiler found five in their series of seventeen cardiac tumours.

With regard to the so-called 'heart base tumour', some controversy surrounds their tissue of origin. They are not regarded as primary cardiac tumours, as they appear to arise either from thyroid cell 'rests', parathyroid or aortic body tissue (Kast 1958). Kast suggest that these tumours probably all arise from thyroid tissue. This view is disputed by Hubben

et al (1960), Guarda and Luginbühl (1965) and Nillson (1955). Stunzi and Teuscher (1953) found two tumours arising from thyroid tissue in a series of three cases. Nonetheless, it is generally accepted that these tumours arise from aortic body tissue (Hubben et al 1960, Guarda and Luginbühl 1965, Nillson 1955). These tumours are white in colour ('potato' tumours) and lie at the base of the heart within the pericardial sac between the aorta and pulmonary artery in most cases. They grow slowly and only the larger ones cause any functional disturbance by pressing on the roots of the great vessels, particularly the pulmonary artery and the atria. Some larger tumours may encroach on the oesophagus. The majority of these tumours are benign but occasionally a malignant tumour is found (Sander and Whitenach 1970).

Heart base tumours are more common in brachiocephalic breeds such as the Boxer, Boston Terrier and Bulldog when they are eight years old or more.

D Other cardio-vascular conditions

Congenital cardiac disease

Congenital cardiac disease is a significant cause of clinical heart disease in dogs under four years old. Patterson (1965) estimated the frequency of congenital cardiac diseases as between 5.6 and 10 per 1000; 58.5% of these cases were under one year old. It is probable that many severe anomalies go undetected because few neonatal deaths in puppies are subjected to a post-mortem. Bowden, Hodgeman and Hime (1963) estimated neonatal deaths of 12% of 3,065 puppies. It is probable that a proportion of these had severe cardiac anomalies. Less severe anomalies permit the survival of the dog for months or even years before the development of a clinical disease.

Patterson (1965) found the six commonest congenital anomalies to be patent ductus arteriosus, pulmonic stenosis, subaortic stenosis, right-sided aortic arch, tetralogy of Fallot and ventricular septal defects, in that order of frequency. Das et al (1965) found patent ductus arteriosus, aortic stenosis and interatrial septal defects were the most common anomalies in a series of 22 cases of congenital cardiac disease. Pirie (1965) stated that pulmonic stenosis, subaortic stenosis and right-sided aortic arch had the greatest frequency. Patterson (1965) found no evidence of cases of coarctation of the aorta but Das et al (1965) found one such case.

Pulmonic stenosis occurs at the level of the valve which is itself smaller than normal, in some cases the cusps may be fused. The right ventricle is hypertrophied and the hyperplastic myocardium may aggravate the stenosis by encroaching upon the outflow tract.

Subaortic stenosis is characterised by the development of a ring of fibrous tissue beneath the valve cusps. The left ventricle is hypertrophied.

Due to the development of the right fourth aortic arch instead of

the left fourth aortic arch, the ductus arteriosus (or ligamentium arteriosium) passes dorsal to the oesophagus from the pulmonary arch to the aortic arch, enclosing the oesophagus in a vascular ring. Under these circumstances, there is obstruction of the oesophagus at this point which leads to dysphagia and dilatation of the cervical oesophagus. Occasionally, both the left and right fourth aortic arches persist with similar results.

The Tetralogy of Fallot is a combination of four defects, a ventricular septal defect, pulmonary stenosis, dextaposition of the aorta and a right ventricular hypertrophy.

Ventricular septal defects are usually found in the fibrous septum and may vary in size. Large defects are associated with left and right ventricular hypertrophy and dilatation.

Patent ductus arteriosus of the vessel may be variable in the degree of patency. If the patency is large, the right ventricle is hypertrophied. Such lesions are difficult to detect in younger animals.

Other lesions such as truncus communis, persistent left cephalic vein, patent foramen ovale and endocardial fibro-elastosis have been recorded.

Patterson (1965) suggested that there was a genetic basis for these congenital defects, although the mode of inheritance was not known. The same parents could have normal and later abnormal litters after successive matings.

Certain breeds appeared to be more susceptible to certain malformations, for example, Boxers and Alsatians are more susceptible to subaortic stenosis. Females appeared to be more susceptible to patent ductus arteriosus than the male, otherwise there did not appear to be any sex predisposition to any particular cardiac anomaly.

Diseases of the pericardium

Pericardial effusions are not common in the dog. These effusions may be classified into two groups, non-inflammatory and inflammatory.

Hydropericardium and haemopericardium are examples of non-inflammatory effusions. Hydropericardium may be seen in conjunction with congestive heart failure where a clear, serous, relatively a-cellular, transudate is seen within the pericardial sac. A sero-sanguinous fluid may be found in the pericardial sac in association with some heart-base tumours and haemangio-sarcomas (Detweiler 1955). Haemopericardium may be seen in association with traffic accidents and rupture of the atrium (Buchanan and Kelly 1964). Blood in the pericardial sac is rapidly defibrinated and reabsorbed (Wilson 1962).

Inflammatory effusions are associated with pericarditis. The classic pericarditis of the dog with a sero-sanguinous exudate, is chronic tuberculous pericarditis. This lesion causes some distension of the pericardial sac which is thickened itself and has greyish-white nodules, 2-4 mm in diameter, scattered over its surface. Occasionally, a sero-sanguinous exudate is seen in cases of nocardiosis in the dog.

Fibrinous pericarditis is rare and confined to puppies.

Detweiler (1952) first described four cases of sub-acute pericarditis with a sero-sanguinous exudate which was of unknown aetiology. Smears and culture failed to reveal the causal organisms. This condition may recur long after an apparent clinical recovery (Luginbuhl and Detweiler 1965; Price and Mullen 1966).

Cardiac tamponade is rare in the dog and when it occurs, it is usually associated from rupture of the atria or from traumatic cardiac injury.

Bacterial endocarditis

Bacterial endocarditis is a relatively uncommon disease which affects dogs over four years old in the majority of cases. The condition arises following a localised bacterial condition such as pneumonia, oral infections, infected anal glands, prostatitis or metritis. However, in some cases, it is not easy to locate this primary focus of infection.

The left atrioventricular valve is more frequently affected than the aortic valve and lesions are rare in the right heart. The affected valves vary in their appearance, according to the age of the lesion. In a recent lesion, the vegetation is a soft, friable, red, cauliflower-like mass of thrombus material, situated on the outflow surface of the area of contact. Older vegetations are smaller and less haemorrhagic. ( ). The histological examination of the vegetation shows it to be composed of organising thrombus material, imposed upon an acute valvulitis. A variety of organisms have been isolated from these lesions, including streptococci, staphylococci, Escherischia coli and Pseudomonas aeruginosa.

The effects of these lesions upon the host are two-fold. Firstly, they result in an incompetence of the affected valve and, secondly, due to the nature of the early valve lesion, sterile or, more regularly, septic emboli are shed from the vegetation into the systemic circulation. These emboli give rise to infarcts in the heart and/or kidney and, less regularly, in other viscera. These myocardial and renal infarcts may cause the death of the animal.

Defects of the conduction mechanism

Patterson et al (1961) found cardiac arrhythmias in 95 of a series of 3000 unselected dogs that were subject to a clinical cardiac examination. In all, 124 abnormal rhythms were found in these 95 dogs of which atrial premature beats, atrial fibrillation, incomplete atrioventricular block, incomplete atrioventricular block with dropped beats, ventricular premature beats and paroxysmal tachycardia were the most common irregularities. Subsequently, 34 of these dogs were subjected to post-mortem and 20 of these showed a chronic valvular disease, hyalin deposition in intramyocardial coronary vessels and myocardial necrosis and fibrosis.

In those cases where the chronic valvular disease was severe, the left atrium was dilated, the atrial myocardium showed a focal necrosis and fibrosis with oedema and/or mononuclear cellular infiltration. The endocardium was thickened and fibrotic. These changes have been associated with a variety of atrial arrhythmias, all of which culminate in an atrial fibrillation (Rubin 1968).

Ventricular arrhythmias have been associated with such conditions as hypertrophy and dilatation which cause variations in the amplitude of the QRS complex. Ischaemia and necrosis of the myocardium cause alterations in the amplitude of the QRS complex and modification of the S-T segment and the T-wave (Rubin 1968). It is clear that this triad of changes (chronic valvular disease, hyalinosis of coronary vessels and myocardial necrosis and fibrosis) produce a range of atrial and ventricular arrhythmias which vary according to the duration and severity of the lesions (Rubin 1968).

Ventricular premature beats with modified T-waves are seen in association with myocarditis. Pericardial effusions, pleural effusions and excess pericardial fat modify the amplitude of all complexes by 'dampening' the current flow. Endocarditis and epicarditis alter the S-T segment and the T-wave. Thoracic tumours and diaphragmatic hernia produce variations in

the amplitude of the QRS complex. Cardiac tumours and heart base tumours may cause atrial and/or ventricular premature beats.

It is clear that there are a wide range of conduction defects in the dog but these have little specificity to any particular lesion (Pirie 1965, Rubin 1968). In contrast, Patterson et al (1961) found atrial arrhythmias consistently in cases of congestive heart failure. Furthermore, it would appear that conduction defects may be found in hearts devoid of any morphological lesion (Pirie 1965).

Anatomical Investigations

These investigations were undertaken to assist in the interpretation of the pathological and histopathological results obtained in this study.

A review of the literature failed to reveal any detailed description of the histology of the normal heart valves of the dog. Such a study was necessary in order to provide reliable 'base-lines' for the histopathological study.

The correct interpretation of the findings in study of the intramyocardial coronary disease necessitated a study of the principle areas of distribution of the blood supply from the major coronary vessels, the assessment of the importance of myocardial sinusoids and arterio-luminal vessels in the blood supply of the heart and further confirmation of the importance of inter-arterial anastomoses in the coronary circulation of the dog's heart.

These results are reported in following order:-

(a) The histology of the normal heart valve.

(b) The definition of the principle areas of supply of the major coronary vessels.

(c) Inter-arterial anastomoses in the coronary circulation.

(d) Myocardial sinusoids and arterio-luminal vessels in the left heart.

(a) The histology of the normal heart valves of the dog

A preliminary study of the normal heart valves in 50 dogs was undertaken as an essential pre-requisite of a histopathological study of the valve lesions, there being no detailed reports of the normal histology in the literature. The terminology of Gross and Kugel (1931) has been employed.

Both the semi-lunar and atrio-ventricular valves may be said to be composed of five basic layers which are taken, in sequence, from the outflow to the inflow surfaces. (Figs. 5 and 6)

(1) Endothelium The outflow and inflow surfaces of the valve cusps are covered by a single layer of flattened endothelial cells which have an elongated, darkly stained nucleus. The endothelium is directly continuous with that of the mural endothelium and the endothelium of the great vessels.

(2) Atrialis The atrialis is a continuation of the atrial endocardium across the outflow surface of the valve cusp (when describing this layer in the semi-lunar valves it is called the ventricularis). The atrialis itself may be divided into four layers:-

(i) Endothelium

(ii) Sub-endothelial layer - a thin, vestigeal layer of loose connective tissue.

(iii) Collagen layer - a thin sheet of collagen and reticulin fibres.

(iv) Elastic layer - a delicate sheet of elastic fibres which tends to be more deeply situated in the area of apposition. The elastic layer is the lower limit of the atrialis and marks its junction with the spongiosa.

(3) Spongiosa The spongiosa is a layer of loose connective tissue containing elastic, reticulin and collagen fibres and variable numbers of fibroblasts, histiocytes and undifferentiated mesenchymal cells. These cells are more numerous in young animals and become progressively less numerous with advancing age. Even in older animals these cells are more numerous at the valve tip. The elastic fibres are most numerous opposite the points of origin of the chordae tendineae. At the valve tip, the spongiosa constitutes the bulk of the valve tissue and extends over the atrial face of the fibrosa to the valve ring where it is directly continuous with the sub-epicardial connective tissue.

(4) Fibrosa The fibrosa, bounded on its atrial surface by the spongiosa, is composed of dense collagenous tissue. At the base of the valve the fibrosa is directly continuous with the fibro-cartilagenous cardiac skeleton. The bulk of the collagen fibres run transversely across the face of the valve. A few fibroblasts are evident between the collagen bundles. The fibrosa provides the main support to the valve cusp, in conjunction with the chordae tendineae and the papillary muscles.

(5) Ventricularis There is an intimate relationship between the ventricularis and the fibrosa on the inflow surface of the valve cusp. (When describing this layer in the semilunar valves, it is called the arterialis.) Structurally the ventricularis is composed of the same four layers as the atrialis but these layers are ill-defined. At its base the ventricularis is directly continuous with the ventricular endocardium.

The Chordae Tendineae

The chordae tendineae consist of a core of dense collagenous tissue, cloaked in endocardium. The collagen fibres are directly continuous with

those of the fibrosa and the myocardial fascia of the papillary muscles. The endocardium is directly continuous with the ventricularis and the mural endocardium of the papillary muscles.

This section covers the general structure of the heart valves but the more detailed structure is also of great importance.

The Atrio-ventricular Valves

The atrio-ventricular valve rings support the valve cusps at their base. This area may be defined as follows (Fig. 5). Into each of the atrio-ventricular valves, a wedge-shaped tongue of atrial myocardium extends for a variable distance into the valve cusp between the atrialis and the spongiosa, this is termed the atrial myocardial prolongation. The tip of this myocardial wedge marks the lower limit of the valve ring on a line drawn at right angles across the cusp at this point. A second line of equal distance is drawn along the atrialis towards the base of the valve from the tip of the myocardial prolongation. A third line joins the two ends of the first two lines to form an equilateral triangle - this is the valve ring which is based upon the atrioventricular rings of the fibro-cartilagenous cardiac skeleton. The ring spongiosa is poorly defined in the atrio-ventricular valves in contrast to the semi-lunar valves.

The valve ring of the septal cusp of the right atrio-ventricular valve is longer and it is inserted in part onto the fibrous interventricular septum and, in part, onto the right atrio-ventricular fibrous ring.

The Left Atrio-ventricular Valve (Mitral valve)

The left atrio-ventricular valve consists essentially of two valve cusps with extensive commissural areas, particularly on the medial aspect.

The Ventral (Septal) cusp (Fig. 3)

This cusp lies dorsal to the aortic orifice and is intimately associated with the dorsal (posterior) aortic cusp on the inflow surface (Fig. 4).

The atrialis is directly continuous with the left atrial endocardium and terminates just short of the valve tip. In the proximal third of the valve, the atrialis is bounded on its ventral face by the atrial myocardial prolongation, the latter being provided with numerous blood vessels.

At the valve base, the spongiosa is directly continuous with the adventitia of the aorta and this layer extends to the valve tip where it constitutes the bulk of the valve tissue.

The fibrosa is directly continuous with the collagen of the aortic media at the base of the valve cusp. The fibrosa extends across the valve to a point just distal to the chordae tendineae with which the fibrosa has an intimate association.

The ventricularis, at the base of the cusp, is separated from the fibrosa by the aortic ring spongiosa but these two layers are intimately associated with each other in the distal two-thirds of the valve (Fig 5).

The ventral (septal) cusp has a Y-shaped base, one limb being provided by the aortic media, one limb by the fibrosa of the dorsal aortic cusp and the stem by the aortic fibrous ring and the fibrosa of the ventral cusp of the left atrio-ventricular valve.

The Dorsal (Mural) cusp

In the dorsal cusp, the atrial myocardial prolongation is shorter than in the ventral cusp. The spongiosa is continuous with the sub-epicardial wedge of loose connective tissue. The base of the fibrosa is continuous with the left atrio-ventricular ring of the cardiac skeleton and is intimately associated with the fascia of the crest of the ventricular myocardium which is dome-shaped. The ventricularis is directly continuous with the left ventricular endocardium. (Fig. 6)

In general, all layers of the dorsal (mural) cusp are less substantial than those of the ventral (septal) cusp. The spongiosa of the

posterior cusp is less rich in elastic fibres than the anterior cusp but otherwise there is little difference.

Both the anterior and posterior cusps contain a variable amount of neutral fat within the spongiosa.

The Right Atrio-ventricular Valve (Tricuspid)

The right atrio-ventricular valve has two cusps, one septal and one mural cusp. The commissural areas of this valve are extensive and may give rise to secondary cusps in systole. In general, the cusps of the right atrio-ventricular valve are less substantial than those of the left side.

The Septal Cusp

The septal cusp is shorter and more substantial than the mural cusp. The ventral (anterior) portion of this cusp is based on the fibrous portion of the inter-ventricular septum and the right fibrous trigone of the cardiac skeleton. Cartilagenous plates may be seen in sections from this area.

More caudal sections through this valve show the valve to be based upon the myocardial ridge of the inter-ventricular septum. The atrial myocardial prolongation is very short in this cusp and is only seen within the cusp in the more caudal sections. (Fig. 7)

The atrio-ventricular bundle and node and the left and right bundle branches of the cardiac conduction mechanism may be seen on the crest or lateral to the margins of the inter-ventricular septum (depending upon the point at which the section is taken).

The structure of the cusp itself is similar, though less substantial than the left atrio-ventricular valve. The spongiosa is a narrower structure in transverse section.

The Mural Cusp

The mural cusp is longer and less substantial than the septal cusp.

In sections taken from the ventral (anterior) portion of this cusp, the crest of the ventricular myocardium is U-shaped due to the presence of the right coronary artery lying above it. In sections taken from the dorsal (posterior) portion, the myocardial crest is dome-shaped and similar to the dorsal cusp of the left atrio-ventricular valve.

The atrial myocardial prolongation is short and delicate and extends for a short distance into the valve cusp. All layers of the valve are delicate by comparison, particularly the spongiosa.

The Semi-lunar Valves

The Aortic Valve

The three semilunar cusps of this valve may be differentiated from each other by their relationships and mode of insertion onto the aortic fibrous ring.

As the aortic media which is rich in elastic fibres approaches the aortic fibrous ring, the deeper elastic fibres are lost progressively until only the superficial fibres remain. This gives the junction an oblique 'wedge-shaped' appearance. The aortic fibrous ring is thickest at the three commissures of the valve cusps and this produces three shallow concavities, the sinus pockets, beneath the valve cusps.

A narrow sheet of collagen runs around the floor of the sinus pocket and extends to a point short of the valve tip in the aortic cusps, this is the fibrosa. Beneath the root of the fibrosa and the aortic fibrous ring is a triangular area of loose connective tissue, the ring spongiosa, which is continued on the cranial (dorsal) aspect to the tip of the aortic cusp; this is the valve spongiosa. Beneath the ring spongiosa the aortic fibrous ring continues for a variable distance beneath the ventricular endocardium, the sub-valvular ring fibrosa. (Fig. 65)

The outflow surface of the valve is supported by a layer directly continuous with the ventricular endocardium, the ventricularis which is

composed of similar fibrillar elements, elastic, reticulin and collagen fibres.

The inflow surface of the valve faces the intima of the aorta. The superficial elastic and collagen fibres pass around the sinus pocket and extend a short distance along the valve- this layer is called the arterialis. The inflow and outflow surfaces are covered by a delicate vascular endothelium.

Certain modifications occur in the ventricularis in adult animals along the line of closure of the semilunar cusps. The ventricularis shows localised fibro-elastic proliferation along the margins of the lunulae. A pronounced localised fibro-elastic proliferation occurs in the centre of the cusp in sections through the nodule valvularum aortae.

There are few cells in the spongiosa of the valve tip distal to the line of closure.

The individual cusps may be recognised by variations in their insertion and by their relationships to neighbouring structures.

The insertion of the right aortic cusp is U-shaped, one arm of the U being formed by the media of the aorta, the other by the fibrosa of the valve cusp as it curls around the base of the sinus pocket and extends along the valve cusp. The right aortic cusp is also associated with the myocardium of the pulmonary conus which is closely associated with the aortic root. The opening of the right coronary artery may be observed in some sections through the centre of this cusp. The left aortic cusp has a similar U-shaped insertion but has no adjacent myocardium as has the right cusp. A section through the centre of the sinus pocket will show the entrance to the left coronary artery or, at least, a branch of that artery.

The dorsal (posterior) cusp has a different insertion from the other two cusps (p.71). The aortic fibrous ring in this cusp is represented

by the base of the fibrosa of the ventral (anterior) cusp of the left atrio-ventricular valve. The ventricularis of the aortic cusp is continuous with that of the atrio-ventricular cusp and separated from the aortic fibrous ring by the broad triangular area of ring spongiosa which lies ventral (caudal) to the sinus pocket.

The Pulmonary Valve

All three semilunar cusps of the pulmonary valve have a U-type insertion and an identical structure to the aortic cusps. They may be differentiated from the aortic cusps by their more delicate structure, the relative thinness of the pulmonary artery when compared with the aorta and the ill-defined sub-valvular annulus. (Fig. 68)

The left cusp is associated with the interatrial septum and therefore it shows a trough-shaped fold of atrial myocardium on its epicardial surface. Alternatively, it may be associated with the root of the aorta in certain sections. The right cusp has no relationships or particular features. The ventral (anterior) cusp is based on a thinner myocardium than the right cusp. In all three cusps the spongiosa is ill-defined.

Ageing Changes in the Heart Valves

In the course of this study of the normal histology of the canine heart valves, consideration was given to the ageing changes in these valves. However, it became apparent that many of the ageing changes of these valves could also be regarded as significant pathological lesions. In consequence, the ageing changes are only summarised here and are described in detail on p.159

These changes may be summarised as follows:

(a) Elastic proliferation of the atrialis followed by fibrosis and plaque formation in the area of apposition.

(b) Fibro-elastic proliferation in the spongiosa.

(c) Progressive reduction in the number of cells, particularly in the spongiosa.

(d) Progressive accumulation of lipid within the spongiosa of the left atrio-ventricular valve.

(e) These changes were observed initially in the ventral (septal) cusp of the left atrio-ventricular valve. At all ages the ageing changes seemed to be more advanced in this cusp.

(f) Ageing changes were the least pronounced in the pulmonary valve.

(b) The definition of the principal areas of supply of the major coronary arteries.

A series of normal greyhound hearts were injected with Micropaque, each into a single major coronary artery, to define the area supplied by that vessel. A second series of hearts were injected with vinyl acetate to provide the 'third dimension' to this study. The results of this investigation are summarised in Table 6 .

A clear definition of the areas of supply was obtained in X-rays of the micropaque-injected hearts and it was possible to build up a picture of the coronary circulation (Figs. 8-9-). There was some 'overlap' of the areas of supply with those of neighbouring vessels which was considered to be due to the presence of anastomoses between them (page 79).

Particular interest was directed towards the blood supply to the left dorsal (posterior) and the left ventral (anterior) papillary muscles and the apex of the left ventricle, all of which was derived from divisions of the left coronary artery:-

(a) Left dorsal papillary muscle

The blood supply to this muscle is derived from the marginal division of the circumflex branch and, to a limited extent, by the septal and left lateral divisions of the dorsal descending branch. The latter vessel supplies a narrow area on the medial aspect of the papillary muscle which obtains the majority of its blood from divisions of the circumflex branch (Fig. 10)

(b) Left ventral papillary muscle

In this muscle, the great majority of the blood supply is derived from the left lateral divisions of the ventral descending branch and, to a lesser extent, by the marginal division of the circumflex branch. The latter vessel supplies only the dorsal aspect

of the crest of the papillary muscle (Fig.9), the remainder of this muscle being supplied from the ventral descending branch. It is possible that variations in the origin of the descending divisions of the left heart (p.20) may modify the relative importance of the ventral descending branch in relation to the circumflex branch in some cases, although no such variation was observed in this series.

(c) The apex of the left ventricle

The apex is supplied over two-thirds of its area by terminal divisions of the ventral descending branch, the remaining one-third being provided by terminal vessels from the marginal division of the circumflex branch (Fig.11).

In a minority of cases, the ventral descending branch extends over the apex of the heart and on to the dorsal aspect. In these cases, the apex is wholly supplied by this vessel (p.21).

(c) Interarterial anastomoses in the coronary circulation

Anastomoses between coronary vessels could be readily demonstrated in hearts injected with vinyl acetate of different colours. These anastomoses were recognised by the naked eye as vessels joining divisions of two different major coronary arteries with a mixing of the different-coloured injection masses within them. (Fig. 12)

Anastomoses were seen between the following vessels:-

(a) Terminal divisions of the dorsal and ventral ascending branches of right coronary artery and the circumflex branch of the left coronary artery, in particular, in the region of the S.A. node.

(b) Terminal divisions of the ventral descending branch and the marginal division of the left circumflex coronary artery in the vicinity of the apex of the left ventricle.

(c) Terminal vessels of the marginal division of the circumflex branch and the dorsal descending branch of the left coronary artery. (Fig. 13)

(d) Terminal divisions of the ventral and dorsal septal branches of the left coronary artery.

(e) Terminal divisions of the dorsal and ventral descending branches of the left coronary artery with the right coronary artery.

(d) The myocardial sinusoids and arterio-luminal vessels of the left heart.

The injection of vinyl acetate, Micropaque and carmine-gelatin into a series of greyhound hearts revealed the presence of a system of vessels joining the coronary circulation to the heart chambers.

When vinyl acetate was injected into the lumina of the left heart, a series of 'loops' and 'spurs' were revealed which arose from the main mass of the cast (the heart chambers). These 'loops' and 'spurs' were vessels lying within the myocardium which entered the heart chambers at one or two points. The 'loops' had two openings into the heart chambers which were joined by a semi-circular vessel (Fig.14). The 'spurs' were shorter, thicker vessels which ran a straight or slightly curved course and entered the heart chambers in the left atrium and ventricle. The 'loops' were the most readily demonstrated, surrounding the base of the papillary muscles. In vinyl acetate casts they appeared as a series of semi-circular vascular loops, lying at right angles to the long axis of the papillary muscles. The casts of these vessels showed them to be irregular in diameter and shape and to have numerous branches and anastomoses with neighbouring 'loops' (Fig.15). These vessels were also demonstrated in hearts injected with Micropaque (Fig.16). However, the vascular 'loops' were not confined to the papillary muscles and they were observed running around the base of the columnae carneae, their openings lying in the clefts on either side of these bands of muscle, particularly towards the apex of the left ventricle (Figs. 17). Similar vessels were observed beneath the pectinate muscles of the left auricular appendage, their openings lying in the clefts between these muscles.

The 'spurs' were shorter vessels of a more uniform diameter and they were disposed fairly uniformly over the endocardial surface of the left ventricle but tending to be more numerous on the outflow tract.

In hearts simultaneously injected with vinyl acetate (into the lumen of the ventricles and into the coronary tree), it was possible to demonstrate a contiguity between the two types of vessel. Thus these vascular 'loops' and 'spurs' were connected directly to the coronary tree. These findings were confirmed with Micropaque in hearts injected into the ventricular cavity and, on occasion, in hearts injected via the coronary vessels (Fig. 18). In both cases, vascular 'loops' could be demonstrated around the base of the papillary muscles.

Hearts injected with carmine-gelatin, processed in paraffin wax and stained by haematoxylin and eosin, showed these to be thin-walled vessels, composed of a vascular endothelium and a few elastic and reticulin fibres (Fig. 19). As these vessels penetrated deeper into the wall of the myocardium, their walls became thicker and they were less readily distinguishable from the vessels of the vascular tree (Figs. 20, 21). These vessels were similar to those described by Wearn et al (1938) who called them myocardial sinusoids. Similar vessels were seen beneath the Trabeculae carneae.

The short 'spurs' seen in the vinylite casts and Micropaque-injected hearts could not be demonstrated histologically but were probably the arterioluminal vessels (Wearn et al 1934).

THE PATHOLOGY OF CHRONIC VALVULAR DISEASE

Introduction

Chronic valvular disease is recognised by the presence of greyish-white, smooth, glistening nodules or plaque-like deformities situated in the area of apposition, on the outflow surface of the valve cusp.

The early lesions appear to be oedematous while advanced lesions may show areas of haemorrhage beneath the endothelium on the outflow surface of the cusp.

The cut surface of an early lesion, which shows slight nodularity, is thickened at the points of origin of the chordae tendineae. In contrast, advanced lesions with apparent nodularity and plaque-like deformities, show a thinning of the valve cusps between thickened origins of the chordae tendineae on the inflow surface of the valve cusps. The cusp is 'ballooned' towards the atrial surface of the valve.

Chronic valvular disease is observed with greater regularity and severity in the left atrioventricular valve. Lesions are less frequent in the right atrioventricular valve where they affect the septal cusp rather than the mural cusp of that valve. Lesions are uncommon in the aortic valve and are rarely, if ever, observed in the pulmonary valve.

The higher incidence of severe lesions in both cusps of the left atrioventricular valve leads to incompetence of the valve and, in some cases, congestive heart failure. Valvular incompetence in the dog's heart is largely confined to the left side.

At the outset of this investigation it was assumed that the various valvular changes that were observed were different manifestations of the same disease process known as chronic valvular disease. It was accepted that it might be necessary to modify this view in the light of experience.

The Grading of Valve Lesions

The various macroscopic valvular deformities that were observed, coupled with the assessment of valvular competence described on p.4, formed the basis of a grading system which divided the hearts into five groups as follows:-

(a) Normal heart valves (see p.16) (Fig. 3)

(b) Type I lesions A few small, discrete, oedematous foci situated in the area of apposition which may be associated with irregular, diffuse areas of opacity in the proximal portions of the valve cusps. These oedematous foci are associated with the origins of the chordae tendineae which, themselves, show no abnormality. There is no evidence of valvular incompetence. (Fig. 22)

(c) Type II lesion Larger, more numerous, nodular foci, greyish-white in colour or oedematous, are seen in the area of apposition. These nodules lie close together and tend to coalesce with their neighbours. Such lesions are clearly associated with the origins of the chordae tendineae which, themselves, show no abnormality. Again, diffuse areas of opacity may be seen in the proximal portions of the valve cusp. There is no evidence of valvular incompetence. (Fig. 23)

(d) Type III lesion A variable number of the greyish-white nodules are evident within the valve cusp but, in addition, areas of 'ballooning' of the cusp are seen in the area of apposition. Areas of haemorrhage may also be seen. The origins and proximal portions of the chordae tendineae related to these lesions are thickened and irregular in their outline. The diffuse areas of opacity are reduced by the encroachment of these lesions. In many cases the lesions are associated with an apparent valvular incompetence. (Fig. 24)

(e) Type IV lesion The valve cusps are grossly distorted by nodules and 'ballooning' which has resulted in shortening of the cusp in its long axis. The chordae tendineae show thickening and irregularity of their outline and an occasional chordae may be ruptured.

The primary chordae tendineae are most frequently involved. All valves examined in this group were apparently incompetent. (Fig. 25)

The Distribution, Frequency and Relative Severity of Chronic Valvular Disease

The results are summarised in tables and in figures.

Table 7 shows that the left atrioventricular valve was the most frequently affected valve (139 dogs, 69.5%). In addition, this valve was the most seriously affected, Type I and II lesions (lesions of little apparent clinical significance) occurred in 73 dogs (36.5%) and Type III and IV lesions (lesions of apparent clinical significance) were seen in 66 dogs (33%).

Chronic valvular disease occurred with greater frequency in the septal cusp than in the mural cusp of the right atrioventricular valve. Lesions were seen in the septal cusp in 105 dogs (52.5%) compared with 23 dogs (11.5%) with lesions in the mural cusp. Furthermore, Type I and II lesions were seen in the septal cusp in 73 dogs (36.5%) compared with 23 dogs (11.5%) with lesions in the mural cusp. Lesions of apparent clinical significance (Type III and IV) were confined to the septal cusp which was affected in 32 dogs (16%).

A comparison of the severity of lesions in the left atrioventricular valve and the septal cusp of the right atrioventricular valve shows that lesions of equal or lesser severity occurred in 91 dogs (45.5%) in the septal cusps. More advanced lesions in the septal cusps were seen in 10 dogs (5%). In 34 dogs (17%) lesions were detected in the left atrioventricular valve while the septal cusp was normal. Of these, 29 dogs (14.5%) showed Type I or II lesions and 5 dogs (2.5%) had Type III or IV lesions. Only 4 dogs (2.0%) showed lesions in the septal cusp in the absence of lesions in the left atrioventricular valve. It was concluded that lesions generally occur with greater frequency and severity in the left atrioventricular valve than in the septal cusp of the right atrioventricular valve.

Lesions in the mural cusp of the right atrioventricular valve are generally mild (Type I & II) and of no apparent clinical importance. They were regularly associated with lesions in the left atrioventricular valve and the septal cusp of the right atrioventricular valve.

Only 14 dogs (7.0%) showed lesions in the aortic valve of which 7 dogs (3.5%) had Type III or IV lesions. In all cases, these were associated with lesions of equal or greater severity in the left atrioventricular valve and 6 dogs (3.0%) had associated lesions in the septal cusp of the right atrioventricular valve.

The pulmonary valve showed no macroscopic abnormalities in this series.

The age distribution of Chronic Valvular Disease

In Tables 1 and 2, two hundred unselected dogs are classified by age and by age groups. The macroscopic valve lesions are graded into four types as described on p. 83.

The relationship between age and the distribution, frequency and severity of these valve lesions is described for each heart valve as follows:-

A. The left atrioventricular valve

Chronic valvular disease was observed in 2 dogs under one year (7%) as Type I or II lesions (lesions of little apparent clinical significance). Type III lesions were not seen in dogs under two years and severe Type IV lesions were not seen in dogs under eight years. (Tables 8 & 9)

Type I and II lesions occurred in 43 dogs (37.4%) under nine years and in 30 dogs (35.3%) nine years and over. Type III and IV (lesions of apparent clinical significance) occurred in 16 dogs (13.8%) under nine years, compared with 50 dogs (58.9%) nine years and over (Table 8).

In Table 10, the age group four years and under shows 11 dogs (14.5%) with Type I lesions and 10 dogs (13.2%) with Type II lesions, i.e. 27.7% with lesions of little apparent clinical significance. Type III lesions occurred in 7 dogs (9.2%) of this age group.

Dogs five to eight years inclusive show Type I lesions in 11 dogs (28.2%) and the same number show Type II lesions, i.e. 56.4% show lesions of little apparent clinical significance. Only 9 dogs (23%) showed Type III or IV lesions in this age group, of which one (2.5%) was a severe lesion (Type IV).

The age group nine to twelve years inclusive, showed 8 dogs (11.4%) with Type I lesions and 20 dogs (28.6%) with Type II lesions, i.e. 40% show lesions of little apparent clinical significance. Type III and

IV lesions occurred in 37 dogs (53%), of which 14 (20%) showed severe lesions (Type IV).

In dogs thirteen years and over, 1 dog (6.7%) showed Type I lesions and one other showed Type II lesions (6.7%). Thirteen dogs (86.6%) showed lesions of apparent clinical significance of which eight (53.3%) were severe lesions (Type IV).

From a study of these results, it is clear that Type I lesions increase in frequency up to the ninth year and then decline thereafter. Type I and II lesions may be seen in dogs under one year. Type II lesions increase in frequency until after the twelfth year and then decline. Type III lesions occur as early as the third year of life and increase in frequency up to the thirteenth year and then tend to decline. The severe Type IV lesion is not seen in dogs under eight years and this lesion increases in frequency thereafter (Fig.26).

It is concluded from these results that chronic valvular disease of the left atrioventricular valve increases in frequency and severity with advancing age. These lesions, once established, are progressive. Type I lesions increase in frequency in early life in close association with an increasing frequency of Type II. In later life the Type I and II lesions decline in frequency as the Type III and IV lesions increase in frequency.

The right atrioventricular valve

The septal and mural cusps of this valve receive separate consideration in this text.

B. The septal cusp

Of the 2 dogs (6.9%) under one year, one showed a Type I lesion and the other showed a Type IV lesion. In the latter case, this severe lesion was associated with a congenital pulmonary stenosis. The remaining 27 dogs (93.1%) under one year showed no abnormality of the septal cusp (Table 3).

Type II lesions occurred in 1 dog (8.3%) at one year.

With the exception of the case recorded above, Type III lesions were not seen in dogs under four years and Type IV lesions were not seen in dogs under eight years.

Type I and II lesions occurred in 34 dogs (29.5%) under nine years and in 39 dogs (45.8%) nine years and over. Type III and IV lesions occurred in 9 dogs (8.8%) under nine years and in 23 dogs (27%) over nine years (Table 8).

In Table 12, dogs under five years old showed 7 dogs (9.2%) with Type I lesions and 10 dogs (13.2%) with Type II lesions, i.e. 22.4% with lesions of little apparent clinical significance. Type III and IV lesions occurred in 2 dogs (2.6%) of which one (1.3%) was a severe lesion (Type IV).

In the age group five to eight years inclusive, 8 dogs (20.5%) showed Type I lesions and 9 dogs (23.1%) showed Type II lesions, i.e. 43.6% showed lesions of little apparent clinical significance. Type III and IV lesions occurred in 7 dogs (17.9%) of which one (2.5%) was a severe lesion (Type IV).

In dogs nine to twelve years inclusive, 11 dogs (15.7%) showed Type I lesions and 23 dogs (32.8%) showed Type II lesions, i.e. 48.5% showed lesions of little apparent clinical significance. Type III and IV lesions occurred in 16 dogs (22.8%) of which five (7.1%) showed severe lesions (Type IV).

Dogs thirteen years and over showed 1 dog (6.7%) with Type I lesions and four (26.7%) with Type II lesions, i.e. 33.4% with lesions of little apparent clinical significance. Type III and IV lesions were seen in 7 dogs (46.6%) of which five (33.3%) showed severe lesions (Type IV).

The frequency of Type I lesions increases up to the ninth year and then declines. The frequency of Type II lesions increases up to the

twelfth year and then tends to decline. Type III lesions increase in frequency between the fourth and ninth year but thereafter there is little evidence of increasing frequency. Indeed, after the twelfth year the frequency tends to decline with the exception of 1 dog (8s). No Type IV lesions were observed in dogs under eight years. Type IV lesions do not show any marked increase in frequency with advancing age until these dogs pass their twelfth year (Fig.27).

The frequency of chronic valvular disease in the septal cusp of the right atrioventricular valve increases with age, but the increase in severity of these lesions is less striking than is observed in the case of the left atrioventricular valve.

The Type III and IV lesions appear to have less influence on the competence of the right atrioventricular valve.

C. The mural cusp

No dogs under three years old showed any evidence of chronic valvular disease. At three years, 1 dog (10%) showed a Type I lesion. At seven years, 2 dogs showed Type II lesions. No dogs at any age showed lesions of apparent clinical significance (Type III and IV).(Table 14)

Dogs under nine years showed 8 dogs (7%) with Type I lesions, while dogs nine years and over showed 12 dogs (14.1%) with Type I lesions and 3 dogs (3.5%) with Type II lesions, i.e. 17.6% with lesions of little apparent clinical significance.(Table 8)

In the youngest age group (dogs under five years) 5 dogs (6.6%) showed Type I lesions and 3 dogs (five to eight years inclusive) showed 7.7% with Type I lesions. In the group of dogs nine to twelve years, 11 dogs (15.7%) showed Type I lesions and 2 dogs (2.9%) showed Type II lesions, i.e. 18.6% had lesions of little apparent clinical significance. Dogs thirteen years and over showed 1 dog (6.6%) with Type I lesions and another (6.6%) with Type II lesions, i.e. 13.2% had lesions of little apparent clinical significance. (Table 14)

The frequency of chronic valvular disease in the mural cusp of the right atrioventricular valve is low and shows a very limited increase with advancing age. No lesions of apparent clinical significance (Type III and IV) were observed in this series (Fig. 28).

The low frequency and mildness of chronic valvular disease in the mural cusp permits a considerable degree of accommodation to the gross abnormalities in the septal cusp.

The semilunar valves

The semilunar valves are considered individually as follows:-

D. The aortic valve

One dog showed a Type I lesion at two years old and no abnormalities were detected in dogs under this age. Type II lesions were not seen in dogs under three years and no dog under five years showed a Type III lesion. Severe lesions (Type IV) were not observed in dogs under ten years. (Table 17)

Dogs under nine years showed Type I and II lesions in 3 dogs (2.6%), while dogs nine years and over showed 4 dogs (4.7%) with these lesions. Type III and IV lesions occurred in 2 dogs (1.7%) of the group under nine years and 5 dogs (5.8%) of the group, nine years and over (Table 8).

In the youngest age group (dogs under five years) 1 dog (1.3%) showed Type I lesions and another (1.3%) showed Type II lesions, i.e. 2.6% with lesions of little apparent clinical significance. (Table 16)

Dogs five to eight years inclusive, showed 1 dog (2.6%) with Type II lesions and 2 dogs (5.1%) with Type III lesions.

In the group nine to twelve years inclusive, 1 dog (1.4%) had Type I lesions and 3 dogs (4.3%) had Type II lesions, i.e. 5.7% showed lesions of little apparent clinical significance. Type III and IV lesions occurred in 3 dogs (4.4%) of which 2 dogs (3.0%) showed severe lesions (Type IV).

In dogs thirteen years and over, 2 dogs (13.3%) showed Type III lesions.

The frequency of chronic valvular disease in the aortic valve was so low that no firm conclusions could be drawn as to the progressive nature of these lesions in this valve (Fig.29) but it appeared that there was little progression of these lesions.

E. The pulmonary valve

No macroscopic valve lesions were detected in this series.

A comparison of the frequency and severity of Chronic Valvular Disease in the heart valves

It has been established (p 82) that chronic valvular disease occurs with the greatest frequency and severity in the left atrioventricular valve and the septal cusp of the right atrioventricular valve (Fig. 34). The frequency and severity of lesions in the mural cusp of the right atrioventricular valve and the aortic valve is low. No macroscopic lesions were detected in the pulmonary valve.

Table 8 shows the earliest ages of onset of chronic valvular disease in each of the heart valves. Dogs under one year showed Type I and II lesions in the left atrioventricular valve and Type I lesions in the septal cusp of the right atrioventricular valve. The septal cusp showed Type II lesions in some dogs at one year old. Type III lesions were seen in the left atrioventricular valve at two years, but similar lesions were not seen in the septal cusp of the right atrioventricular valve before the fourth year. No Type IV lesions were seen in either of these valves in dogs under eight years (excepting one puppy with a congenital pulmonary stenosis 137/61).

The mural cusp of the right atrioventricular valve showed Type I lesions from the third year onwards, while Type II lesions were not observed in dogs under eleven years. No Type III or IV lesions were observed in this cusp.

In the aortic valve, Type I lesions were observed at two years. Type II lesions at three years, Type III lesions at five years and Type IV lesions were seen in dogs over ten years.

The pulmonary valve showed no macroscopic abnormality at any age.

In Tables 8 · 9 · , the frequency of Type I and II lesions in the left atrioventricular valve of dogs under nine years is compared with the frequency of similar lesions in dogs nine years and over. The

dogs under nine years showed 22 (19.1%) with Type I lesions and 21 (18.3%) with Type II lesions. The dogs nine years and over showed 9 (10.6%) with Type I lesions and 21 (24.7%) with Type II lesions. These results are compared with the results of the examination of the septal cusp of the right atrioventricular valve. The septal cusp of dogs under nine years showed 15 (13.0%) with Type I lesions and 19 (16.5%) with Type II lesions. In the group nine years and over, 12 dogs (14.1%) showed Type I lesions and 27 dogs (31.7%) showed Type II lesions.

A similar comparison of the frequency of Type III and IV lesions in the same age groups showed that dogs under nine years have Type III lesions in 15 cases (13%) and one dog (0.8%) had a Type IV lesion in the left atrioventricular valve. Dogs nine years and over showed 28 (33.0%) with Type III lesions and 22 (25.9%) with Type IV lesions. In the septal cusp of the right atrioventricular valve, dogs under nine years showed 7 (6.1%) with Type III lesions and 2 (1.7%) with Type IV lesions (including one dog with a congenital pulmonary stenosis). Dogs nine years and over showed 13 (15.3%) with Type III lesions and 10 (11.7%) with Type IV lesions.

There is a greater frequency of chronic valvular disease in the left atrioventricular valve (69.5%) than in the septal cusp of the right atrioventricular valve (52.5%). Type I and II lesions develop in the left atrioventricular valve and in the septal cusp within the first two years of life and while the frequency of Type I lesions may be higher in the left atrioventricular valve, there is a closer relationship in the frequency of Type II lesions in this cusp when compared with the septal cusp of the right atrioventricular valve in dogs under nine years. Dogs over nine years show a decline in Type I lesions and an increase in Type II lesions in the left atrioventricular valve. The septal cusp of the right atrioventricular valve in dogs nine years and over shows a slight increase in the frequency of Type I lesions and a sharp increase in Type II lesions.

In dogs under nine years, Type III lesions in the left atrioventricular valve have twice the frequency observed in the septal cusp of the right atrioventricular valve. If one case of congenital pulmonary stenosis is excluded, there is a close parity in the frequency of Type IV lesions in the left atrioventricular valve and the septal cusp of the right atrioventricular valve. Type III and IV lesions have more than twice the frequency in the left atrioventricular valve when compared with the septal cusp of the right atrioventricular valve in dogs nine years and over.

In Fig.30 the distribution of grades of chronic valvular disease in the left atrioventricular valve is plotted for dogs under nine years and for dogs nine years and over. It is clear that this condition has a greater frequency in dogs nine years and over. While Type I lesions are more common in young dogs, all three other grades are more common in the older dogs. The frequency of Type III and IV lesions is far higher in the older dogs.

In Fig.31, a similar histogram records the relative frequency of grades of chronic valvular disease in the septal cusp of the right atrioventricular valve. In dogs under nine years, the frequency of Type I and II lesions is lower than in the left atrioventricular valve and a similar reduction is evident for Type III lesions (Fig.31). Compared with the left atrioventricular valve (Fig.30), the frequency of lesions in the dogs nine years and over is very different. There is a greater frequency of Type I lesions and a considerable increase in Type II lesions, while Type III and IV lesions showed a modest increase in frequency in the older age group. It was concluded that while chronic valvular disease is essentially progressive in the left atrioventricular valve, the frequency of these lesions is lower in the septal cusp of the right atrioventricular valve, and these lesions are less progressive

and tend to become arrested at the Type II stage.

In Fig. 8, it will be seen that the frequency and severity of lesions in the mural cusp is low. No Type III or IV lesions were observed in this cusp. Type I lesions only were observed in dogs under nine years. In dogs over nine years, there was some increase in Type I lesions and a few Type II lesions were seen. In Fig. 32, it is seen that the frequency of Type I lesions is much lower than that observed in the septal cusp. In dogs over nine years, the frequency of Type I lesions is the same, but Type II lesions are uncommon in the mural cusp, in sharp contrast to the situation observed in the septal cusp of this valve. It was concluded that, while it is clear that the mural and septal cusps have little in common with each other, both cusps show a lower frequency of lesions and a reduced tendency to progression when they are compared with the left atrioventricular valve.

In Fig.33, a similar histogram shows a very low frequency of chronic valvular disease in the aortic valve. In dogs nine years and over, there is a slight increase in frequency when compared with dogs under nine years, but the numbers are so small that no firm conclusions can be reached. The fact that Type III and IV lesions may be observed suggests that these lesions are more progressive than those in the mural cusp of the right atrioventricular valve.

In Fig. 8, the age group under five years inclusive, normal left atrioventricular valves were seen in 48 dogs (63.2%) compared with 57 dogs (75%) with normal septal cusps of the right atrioventricular valve and 71 dogs (93.4%) with normal mural cusps of that valve. The aortic valve was normal in 74 dogs (97.4%) and all pulmonary valves were normal in this series. Type I lesions were seen in 11 dogs (14.5%) in the left atrioventricular valve, compared with 7 dogs (9.2%) with lesions in the septal cusp of the right atrioventricular valve, and

5 dogs (6.6%) with Type I lesions in the mural cusp. The aortic valve showed 1 dog (1.3%) with Type I lesions.

Type II lesions affected the left atrioventricular valve in 10 dogs (13.2%) and a similar number showed lesions in the septal cusp of the right atrioventricular valve. No lesions of this type affected the mural cusp but the aortic valve was affected in one case (1.3%). Type III lesions were observed in 7 (9.2%) of the left atrioventricular valves and 1 (1.3%) of septal cusps. The mural cusp of the right atrioventricular valve and the aortic valve showed no Type III lesions in this age group. Type IV lesions were seen in 1 (1.3%) septal cusp of the right atrioventricular valve associated with a congenital pulmonary stenosis (137/61).

In the age group five to eight years inclusive, normal left atrioventricular valves were seen in 8 dogs (20.5%) compared with 15 dogs (38.5%) with normal septal cusps of the right atrioventricular valves and 36 dogs (92.3%) with normal mural cusps and aortic valves. No pulmonary valves were affected in these dogs.

A total of 11 dogs (28.2%) showed Type I lesions in the left atrioventricular valves compared with 8 dogs (20.5%) with similar lesions in the septal cusp of the right atrioventricular valve. The mural cusp showed 3 dogs (7.7%) with Type I lesions and no lesions of this type were seen in the aortic valve. Type II lesions affected 11 (28.2%) left atrioventricular valves, while 9 (23.1%) septal cusps of the right atrioventricular valve were affected. One aortic valve (2.6%) showed Type II lesions. Type III lesions occurred in 8 (20.5%) of left atrioventricular valves compared with 6 (15.4%) septal cusps of the right atrioventricular valve. The aortic valve showed 2 cases (5.1%) with Type III lesions. Type IV lesions were confined to the left atrioventricular valve and the septal cusp of the right atrioventricular valve

where one (2.5%) was affected in each case.

The age group, nine to twelve years inclusive, showed 5 dogs (7.1%) with normal left atrioventricular valves compared with 20 (28.6%) septal cusps of the right atrioventricular valve, 57 (81.4%) of the mural cusp and 63 (90%) of the aortic valves. Type I lesions occurred in the left atrioventricular valves of 8 dogs (11.4%) compared with 11 (15.7%) of the septal cusps of the right atrioventricular valves and a similar number in the mural cusp. The aortic valve showed Type I lesions in 1 dog (1.4%). Type II lesions were seen in 20 (28.6%) of left atrioventricular valves and 23 (32.8%) of septal cusps of the right atrioventricular valve. Two dogs (2.9%) showed Type II lesions in the mural cusp and 3 dogs (4.3%) showed similar lesions in the aortic valve. Type III lesions were seen in the left atrioventricular valve of 23 dogs (32.8%) while similar lesions occurred in the septal cusp of the right atrioventricular valve in 11 dogs (15.7%). No Type III lesions were seen in the mural cusp but the aortic valve showed 1 (1.4%) such lesion. Type IV lesions occurred in the left atrioventricular valve of 14 dogs (20%) compared with 5 dogs (7.1%) with similar lesions in the septal cusp of the right atrioventricular valve. No Type IV lesions were seen in the mural cusp but these lesions were seen in 2(3%) of the aortic valves.

In the age group thirteen to sixteen years inclusive, no normal left atrioventricular valves were observed, while 3 (20%) dogs showed no lesions in the septal cusp of the right atrioventricular valve; 13 (86.6%) showed normal mural cusps and aortic valves. The left atrioventricular valve, septal and mural cusps of the right atrioventricular valve each showed 1 (6.7%) with Type I lesions. No Type I or II lesions were seen in the aortic valve. Type II lesions were seen in 1 (6.7%) of the left atrioventricular valves, 4 (26.7%) of septal cusps and 1 (6.7%) of mural cusps of the right atrioventricular valves. No Type II were seen in the

aortic valve. Type III lesions occurred in 5 (33.3%) of left atrioventricular valves, 2 (13.3%) of septal cusps of the right atrioventricular valve and a similar number involving the aortic valve. No Type III or IV lesions occurred in the mural cusp of the right atrioventricular valve. Type IV lesions were seen in 8 (53.3%) of the left atrioventricular valves, compared with 5 (33.3%) of septal cusps of the right atrioventricular valve. No Type IV lesions were seen in the aortic valve.

In Fig.26, the frequency of all four grades of chronic valvular disease in the left atrioventricular valve are plotted for the four different age groups. In dogs under four years, the frequency of Type I and II lesions is similar, and greater than the frequency of Type III lesions. Between five and eight years, the frequency of Type I and II lesions increases rapidly, in association with a less rapid increase in Type III lesions. A few Type IV lesions appear during this period. In the years nine to twelve, the frequency of Type I lesions declines sharply, while there is a further slight increase in Type II lesions; a steeper increase in Type III lesions is seen, until the frequency exceeds the frequency of Type II lesions. There is a sharp increase in Type IV lesions. Dogs thirteen years and over show a further diminution in the frequency of Type I lesions, and a steep decline in Type II lesions to a low level. In contrast, Type III lesions show a slight increase, while Type IV lesions increase rapidly.

It was concluded that chronic valvular disease in the left atrioventricular valve increases in frequency with advancing age. These four grades of valve lesions show an initial increase in frequency with a subsequent decline. Mild Type I lesions reach a peak in dogs under nine years old and then decline progressively in the older age groups. The more advanced Type II lesion increases to a peak which is maintained from the five to eight year old group through the nine to twelve year

old group and then declines progressively thereafter. The more severe Type III lesions do not reach their peak of frequency until the ninth to twelfth year and this level is maintained in the group thirteen to sixteen years. Type IV lesions are low in frequency in dogs five to eight years but increase rapidly in the two subsequent age groups and are the predominant lesions in dogs thirteen years and over.

These results suggest that chronic valvular disease in the left atrioventricular valve is a progressive lesion, low in frequency and severity in young dogs, and increasing in frequency and severity with advancing age.

Fig.47 shows the frequency of all four grades of chronic valvular disease in the septal cusp of the right atrioventricular valve in all four age groups. The frequency of Type I and II lesions in dogs under five years is at a lower level than in the left atrioventricular valve and the frequency of these lesions does not increase at the same rate in age group five to eight years. In the period five to eight years, the frequency of Type III lesions increases rapidly, but there is little increase in the frequency of Type IV lesions. The increase in frequency of Type III lesions from a very low level under five years is more dramatic than that observed in the left atrioventricular valve in the period five to eight years. In the age group nine to twelve years, Type I lesions decline, but less dramatically than seen in the left atrioventricular valve. The frequency of Type II lesions continues to increase to reach a peak during this period of life. (They persist for a longer period than in the left atrioventricular valve.) There is no further increase in the frequency of Type III lesions and only a limited increase in Type IV lesions. In the left atrioventricular valve, Type III lesions continue to increase in frequency during this period and Type IV lesions show a more rapid increase in frequency. In dogs thirteen years and over, the decline in frequency of Type I lesions continues, Type II

lesions decline slightly but persist at a far higher level than such lesions do in the left atrioventricular valve. There is a slight decline in the frequency of Type III lesions which persist at a far lower level than in the left atrioventricular valve. There is a rapid increase in Type IV lesions but to a lower peak than in the left atrioventricular valve.

The overall frequency of chronic valvular disease in the septal cusp of the right atrioventricular valve is lower than in the left atrioventricular valve. In relation to the left atrioventricular valve, the frequency of Type III and Type IV lesions is much lower. The frequency of Type I lesions declines progressively in dogs nine years and over. Type II lesions are the predominant lesions and tend to persist in dogs over nine years. It is concluded that chronic valvular disease is less progressive in the septal cusp than in the left atrioventricular valve and that the more severe Type III and IV lesions are less common.

In Fig.23, the frequency of Type I and II lesions in the mural cusp of the right atrioventricular valve in the four age groups are recorded. Type III and IV lesions were not observed in this cusp. Only Type I lesions were observed at a low level in dogs under nine years old. In the period nine to twelve years, dogs showed a moderate increase in Type I lesions and the appearance of a few Type II lesions. Dogs thirteen to sixteen years show a distinct decline in Type I lesions and a compensating increase in Type II lesions.

The overall frequency of chronic valvular disease in the mural cusp of the right atrioventricular valve is low, far lower than that observed in the septal cusp of that valve. The predominant lesion is a Type I lesion. Type II lesions are generally uncommon. It was concluded that lesions in the mural cusp are generally non-progressive.

In Fig. 34, the frequency of all four grades of chronic valvular disease in the aortic valve in the four age groups is recorded. The overall frequency of lesions in the aortic valve is so low that no meaningful results can be extracted. The fact that all four grades may be observed, suggests that chronic valvular disease in the aortic valve may be a progressive lesion.

In comparing the frequency and severity of chronic valvular disease in three different valves (Fig. 34), we have seen that the left atrioventricular valve is most frequently and severely involved and that the lesion, once established, is progressive throughout the life span of the dog. In the right atrioventricular valve, there are distinct differences in severity and frequency between the two cusps. The septal cusp has reduced frequency and a lesser tendency to progression to Type III and IV lesions than the left atrioventricular valve. The mural cusp has a far lower frequency of lesions than the other two cusps and few of these lesions progress beyond Type I. The overall frequency of chronic valvular disease in the aortic valve is very low but as all four grades of lesion are observed in this small number of cases, the lesions appear to be progressive.

To underline these variations, Figs. 35-38, outline the frequency of each grade of chronic valvular disease in the four age groups. In Fig. 35, Type I lesions follow a similar pattern in the left atrioventricular valve and the septal cusp of the right atrioventricular valve. The frequency of Type I lesions is higher in the left atrioventricular valve, but both cusps reach a peak in the age group five to eight years and decline progressively in older dogs. The frequency of Type I lesions in the mural cusp of the right atrioventricular valve is lower still and does not reach a peak until the nine to twelve year group and thereafter declines. Little or nothing can be said of the

aortic valve, other than that lesions are uncommon in this valve.

In Fig.36, Type II lesions in the left atrioventricular valve reach a peak between the fifth and twelfth year and decline rapidly in frequency in dogs over that age. In the septal cusp of the right atrioventricular valve, Type II lesions develop less rapidly to reach a higher peak in the ninth to twelfth year and the frequency declines slightly thereafter. The frequency of Type II lesions in the mural cusp is very low and occur only in dogs over nine years old. The frequency of chronic valvular disease Type II in the aortic valve is very low but tends to increase in frequency up to the ninth to twelfth year and then declines.

Fig.37, shows that Type III lesions rise to reach a plateau in dogs over twelve years in the left atrioventricular valve. In the septal cusp of the right atrioventricular valve such lesions reach a lower peak in dogs over eight years but this is maintained through to the twelfth year and then shows a slight decline. The frequency of Type III lesions in the aortic valve is difficult to interpret. The lower frequency of Type III lesions makes the interpretation difficult but it would appear that Type III lesions increase in frequency with advancing age.

Fig.38, shows that Type IV lesions are uncommon in dogs under nine years. In the left atrioventricular valve, the frequency of these lesions increases in the age group nine to twelve years inclusive and shows a sharp increase again in dogs over this age. There is only a slight increase in the frequency of Type IV lesions in the septal cusp of the right atrioventricular valve in dogs under thirteen years. Dogs thirteen years and over show a sharp increase in frequency but the peak is far lower than that attained in the left atrioventricular valve. The results as far as the aortic valve are concerned are inconclusive.

No Type III or IV lesions were observed in the mural cusp of the right atrioventricular valve.

The Sex Distribution of Chronic Valvular Disease

The 200 hearts which form the basis of this study were derived from 105 entire males, 89 entire females and a further 6 females which had been subjected to an ovaro-hysterectomy.

There was a disparity in the age distribution of the sexes in this series. Table 2 shows the sex distribution by age groups and the relative parity of the sexes in age groups 5-8 years inclusive and 13-16 years inclusive. But there is a disparity of the sexes in age group 0-4 years inclusive with a predominance of males. This situation is reversed in age group 9-12 years inclusive, although the disparity is less pronounced.

Each heart valve will be considered separately when considering and comparing the frequency and severity of chronic valvular disease in the two sexes. The six females subjected to an ovaro-hysterectomy are excluded from this comparison.

A The left atrioventricular valve

The various grades of chrcnic valvular disease were seen in 74 males (70.5%) and 60 entire females (67.4%).

Table 18A shows that Type I lesions occurred in 16 males (15.2%) and 14 females (15.7%) while Type II lesions were seen in 26 males (24.8%) ahd 14 females (15.7%). Therefore, lesions of little apparent clinical significance were seen in 40.0% of males and 31.4% of females. Type III lesions were seen in 17 males (16.2%) and 24 females (27.0%) while Type IV lesions occurred in 15 males (14.3%) and 8 females (9.0%). Therefore, lesions of apparent clinical significance (Types III and IV) were seen in 32 males (30.5%) and 32 females (36.0%).

In Table 19A the frequency of chronic valvular disease in the two sexes is compared in dogs under and over nine years. In dogs under nine years, the males show 31 (46.9%) with no abnormality of the left

atrioventricular valve compared with 24 females (51.1%). In the male, Type I lesions were seen 13 (19.9%) compared with 8 females (17.0%). Type II lesions were seen in 15 males (22.7%) and 6 females (12.8%). Lesions of little apparent clinical significance (Types I and II) occurred in 28 males (42.6%) and 14 females (29.8%). Type III lesions were seen in 6 males (9.0%) and 9 females (19.1%) while Type IV lesions occurred in 1 male (1.5%) only. Lesions of apparent clinical significance occurred in 7 males (10.5%) and 9 females (19.1%). These results suggest that the lesions develop more rapidly in those females that become affected for the overall frequency of chronic valvular disease is greater in the male.

In dogs over nine years old, 5 females (11.9%) only had normal left atrioventricular valves. In the male Type I lesions were seen in 3 (7.7%) compared with 6 females (14.3%). Type II lesions were seen in 11 males (28.2%) and 8 females (19.0%). Lesions of little apparent clinical significance occurred in 14 males (35.9%) and 14 females (33.3%). Type III lesions were seen in 11 males (28.2%) and 15 females (35.7%) while Type IV lesions occurred in 14 males (35.9%) and 8 females (19.0%). Lesions of apparent clinical significance (Types III and IV) occurred in 25 males (64.1%) and 23 females (54.7%). These results suggest that while the valve lesions may develop more rapidly in the female under nine years, the male ultimately surpasses the female in the frequency and severity of these lesions.

Table 20A shows the sex distribution of chronic valvular disease by age groups. In dogs under five years the males showed 29 (64.4%) with no abnormality of the left atrioventricular valve compared with 19 females (61.3%). In the male, Type I lesions seen in 6 (13.3%) compared with 5 (16.1%) in the female. Type II lesions were seen in 9 males (20.0%) and 1 female (3.2%). Lesions of little apparent clinical

significance occurred in 15 males (33.3%) and 6 females (19.3%). Type III lesions were seen in 1 male (2.2%) and 6 females (19.3%) but no Type IV lesions were observed in either sex. Lesions of apparent clinical significance (Type III) were observed in 1 male (2.2%) and 6 females (19.3%). These results suggest that while lesions develop less frequently in the female they develop more rapidly in those females that are affected than in the male.

In the age group five to eight years inclusive, 2 males (9.5%) had normal left atrioventricular valves compared with 5 females (31.3%). Of the males, 19 (90.5%) showed valve lesions while 11 females (68.7%) showed changes in their left atrioventricular valves. In the male, Type I lesions were seen in 7 (33.3%) compared with 3 females (18.7%) Type II lesions were seen in 6 males (28.6%) and 5 females (31.3%). Lesions of little apparent clinical significance occurred in 13 males (61.9%) and 8 females (50.0%). In the male, Type III lesions were seen in 5 (23.8%) compared with 3 females (18.7%) while Type IV lesions were seen in 1 male (4.8%) only. Lesions of apparent clinical significance (Types III and IV) occurred in 6 males (28.6%) and 3 females (18.7%). These results suggest that frequency and severity of chronic valvular disease is greater in the male than in the female in dogs between five and eight years.

In the age group nine to twelve years inclusive only, 5 females (14.3%) had normal left atrioventricular valves. Of the males, 31 (100.0%) showed valve lesions compared with 30 females (85.7%). Type I lesions were seen in 3 males (9.7%) and 5 females (14.3%), while Type II lesions occurred in 11 males (35.5%) and 7 females (20.0%). Lesions of little apparent clinical significance occurred in 14 males (45.2%) and 12 females (34.3%). Type III lesions were seen in 8 males (25.8%) and 13 females (37.1%) while Type IV lesions occurred in 9 males (29.0%)

and 5 females (14.3%). Lesions of apparent clinical significance occurred in 17 males (54.8%) and 18 females (51.4%). These results suggest that the frequency and severity of chronic valvular disease in the left atrioventricular valve is greater in the male than in the female in dogs between nine to twelve years inclusive.

In the age group thirteen to sixteen years inclusive, both sexes showed valve lesions in every case. Type I lesions occurred in 1 female (14.3%) only and Type II lesions occurred in 1 other female (14.3%). Lesions of little apparent clinical significance occurred in 2 females (28.6%). Type III lesions occurred in 3 males (37.5%) and 2 females (28.6%) while Type IV lesions were seen in 5 males (62.5%) and 3 females (42.8%). Lesions of apparent clinical significance (Types III and IV) were seen in 8 males (100.0%) and 5 females (71.4%). These results suggest that lesions of greater severity occur more regularly in the male than in the female in dogs thirteen to sixteen years old.

To summarise all these results on Table A20, it is clear that while in the group under five years lesions may develop more rapidly in the female than in the male, in the other three age groups the lesions in the left atrioventricular valve have a greater frequency and severity in the male than in the female.

In Tables A21, the year-by-year frequency and severity of chronic valvular disease in the left atrioventricular valve is shown for both sexes. The number of individuals of any one age and sex is small and this makes any interpretation of this table difficult. However, it is possible to draw some conclusions.

Both Type I and Type II lesions are seen in the male under one year, while similar lesions are not seen until one to two years later in the female. Conversely, however, 2 females (25.0%) showed Type

III lesions in their second year while similar lesions did not develop in the male until one year later. Type IV lesions were first seen in the male in the eighth year but did not appear in the female until one year later. In general, the frequency and severity of chronic valvular disease in the male is greater than in the female.

In Figure A31, the sex distribution of chronic valvular disease in the left atrioventricular valve is compared. The frequency of lesions in the male was 70.5% compared with 67.4% in the female. Type I lesions were seen in 15.2% of males and 15.7% of females, while Type II lesions occurred in 24.8% of males and 15.7% of females. Therefore lesions of little apparent clinical significance (Types I and II) occurred in 40.0% of males and 31.4% of females. Type III lesions occurred in 16.2% of males and 27.0% of females while Type IV lesions were seen in 14.3% of males and 9.0% of females. Lesions of apparent clinical significance (Types III and IV) occurred in 30.5% of males and 36.0% of females. There is a greater proportion of Type III lesions in the females than in the males. In general the frequency and severity of lesions in the left atrioventricular valve is greater in the male than in the female. In a previous section it was concluded that the frequency of the more advanced grades of chronic valvular disease (Types III and IV) were age related. Therefore the age distribution of the sexes becomes relevant. In Table A32, there were 115 dogs under nine years of which 57.4% were males and 42.6% were females. In the 85 dogs over nine years 45.9% were male and 54.1% were female. If these lesions are age related, the higher proportion of Type III lesions in the females is not so surprising with their numerical superiority. The greater frequency of Type IV lesions in the male reinforces the view that, in general, lesions are of greater severity in the male than in the female.

It is difficult to explain the greater frequency of Type III lesions in females under nine years. It may be that lesions develop more rapidly in a proportion of females and the situation becomes stabilised thereafter. It is tempting to suggest that these younger females may have been subjected to pregnancy and parturition, a feature not shared with others of their sex. There is no evidence available that would confirm or refute this theory.

The right atrioventricular valve

The septal and mural cusps of the right atrioventricular valve are considered separately.

B. The septal cusp

The various grades of chronic valvular disease were seen in 55 males (52.3%) and 47 females (52.8%).

Table B 22 shows that Type I lesions were seen in 15 males (14.3%) and 12 females (13.5%) while Type II lesions occurred in 20 males (19.0%) and 25 females (28.1%). Therefore, lesions of little apparent clinical significance (Types I and II) occurred in 35 males (33.3%) and 37 females (41.6%). Type III lesions were seen in 10 males (9.5%) and 8 females (9.0%) while Type IV lesions occurred in 10 males (9.5%) and 2 females (2.2%). Thus, lesions of apparent clinical significance (Types III and IV) were seen in 20 males (19.0%) and 10 females (11.2%).

In Table B 22, the frequency and severity of chronic valvular disease in the septal cusp is compared in dogs under and over nine years. In dogs under nine years, 42 males (63.6%) and 28 females (59.6%) had normal septal valves. Type I lesions occurred in 10 males (15.2%) compared with 5 females (10.6%) while Type II lesions were seen in 8 males (12.1%) and 11 females (23.4%). Lesions of little apparent clinical significance were seen in 18 males (27.3%) and 16 females (34.0%). Type III lesions were observed in 4 males (6.1%) and 3 females (6.4%) while Type IV lesions were confined to 2 males (3.0%). Therefore lesions of apparent clinical significance (Types III and IV) occurred in 6 males (9.1%) and 3 females (6.4%). There is a greater frequency of chronic valvular disease (Types I and II) in the female.

In dogs over nine years, 8 males (20.5%) and 14 females (33.3%) had normal septal valves. Type I lesions occurred in 5 males (12.8%) compared with 7 females (16.7%) while Type II lesions were seen in 12

males (30.8%) and 14 females (33.3%). Thus, lesions of little apparent clinical significance (Types I and II) occurred in 17 males (43.6%) and 21 females (50.0%). Type III lesions were seen in 6 males (15.4%) and 5 females (11.9%) while Type IV lesions were evident in 8 males (20.5%) and 2 females (4.8%). Therefore, lesions of apparent clinical significance occurred in 14 males (35.9%) and 7 females (16.7%). The results of the comparison of frequency and severity of lesions in the septal cusp in males and females under and over nine years suggest that while these lesions tend to develop more rapidly in the young female, the male ultimately surpasses the female in the frequency and severity of lesions.

Table B 23, shows the sex distribution by age groups of chronic valvular disease in the septal cusp. In dogs under five years old, a total of 34 males (75.6%) had normal septal valves compared with 23 females (74.2%). Type I lesions were seen in 5 males (11.1%) and 2 females (6.5%) while Type II lesions occurred in 5 males (11.1%) and 5 females (16.1%). Therefore, lesions of little apparent clinical significance occurred in 10 males (22.2%) and 7 females (22.6%). Type III lesions were seen in 1 female (3.2%) and Type IV lesions in 1 male (2.2%). In the latter case the Type IV lesion was associated with a congenital pulmonary stenosis. It was concluded from these results that chronic valvular disease tends to develop more rapidly in the female than in the male in dogs under five years old.

In the age group five to eight years inclusive, a total of 8 males (38.1%) and 5 females (31.3%) had normal septal valves. Type I lesions were seen in 5 males (23.8%) and 3 females (18.7%) while Type II lesions occurred in 3 males (14.3%) and 6 females (37.5%). Lesions of little apparent clinical significance (Types I and II) occurred in 8 males (38.1%) and 9 females (56.2%). Type III lesions were seen in 4 males (19.0%)

and 2 females (12.5%) and Type IV lesions in 1 male (4.8%) only. Therefore, lesions of apparent clinical significance occurred in 5 males (23.8%) and 2 females (12.5%). A study of these results indicates that while the overall frequency of chronic valvular disease is greater in the female, the frequency of Type III and IV lesions is greater in the male, i.e. the lesions are more progressive.

In the age group nine to twelve years inclusive, a total of 8 males (25.8%) and 11 females (31.4%) had normal septal valves. Type I lesions were seen in 5 males (16.1%) and 6 females (17.1%), while Type II lesions occurred in 9 males (29.0%) and 13 females (34.1%). Therefore, lesions of little apparent clinical significance (Types I and II) occurred in 14 males (45.1%) and 19 females (54.2%). Type III lesions were seen in 5 males (16.1%) and 4 females (11.4%) while Type IV lesions occurred in 4 males (12.9%) and 1 female (2.9%). Lesions of apparent clinical significance occurred in 9 males (29.0%) and 5 females (14.3%). These results support the view that chronic valvular disease of the septal cusp is of greater frequency and severity in the male than in the female.

In the age group thirteen to sixteen years inclusive, 3 females (43.0%) had normal septal valves. Type I lesions occurred in 1 female only (14.3%) while Type II lesions were seen in 3 males (37.5%) and 1 female (14.3%). Lesions of little apparent clinical significance occurred in 3 males (37.5%) and 2 females (28.6%). Type III lesions were seen in 1 male (12.5%) and 1 female (14.3%) while Type IV lesions occurred in 4 males (50.0%) and 1 female (14.3%). Therefore, lesions of apparent clinical significance (Types III and IV) occurred in 5 males (62.5%) and 2 females (28.6%). These results show that chronic valvular disease was of greater frequency and severity in the septal cusp of dogs between thirteen and sixteen years old.

If we summarise the conclusions on the relative sex incidence of

chronic valvular disease in the septal cusp while lesions tend to develop more frequently in the young female than in the male; in general, these lesions have a greater frequency and severity in the male.

A comparison of the year-by-year frequency and severity of chronic valvular disease in both sexes (Table B 24) shows that the number of individuals in any one group by sex and age is small, which creates difficulties in the interpretation of the results. Type I lesions were observed in a male (5.5%) under one year, while similar lesions were first seen in 2 females (25.0%) at two to three years. Type II lesions were first seen in 1 male (14.3%) at one year but were not seen in the female until one year later. In contrast, Type III lesions were first seen in the female in the fourth year and in the male in the following year. Type IV lesions were seen in a male puppy under one year in association with a congenital pulmonary stenosis and again in the male in the eighth year. Similar lesions were not seen in the female until the ninth year.

In Table B22, the sex distribution of chronic valvular disease in the septal cusp of the right atrioventricular valve is compared. The frequency of lesions in the male was 52.4% compared with 52.8% of females. Type I lesions were seen in 14.3% of males and 12.6% of females while Type II lesions occurred in 19.0% of males and 26.3% of females. Therefore, lesions of little apparent clinical significance (Types I and II) occurred in 33.3% of males and 38.9% of females. Type III lesions occurred in 9.5% of males and 8.9% of females, while Type IV lesions occurred in 9.5% of males and 2.1% of females. Thus, lesions of apparent clinical significance (Types III and IV) occurred in 19.0% of males and 11.0% of females. While the frequency of lesions shows a close relationship in the two sexes, the lesion is more progressive in the male than in the female.

C The mural cusp

Chronic valvular disease (Types I and II only) were seen in 10 males (9.5%) and 11 females (12.4%) involving the mural cusp of the right atrioventricular valve. No Type III or IV lesions were observed in this valve cusp.

Table C 25 shows that Type I lesions occurred in 8 males (7.6%) and 10 females (11.2%) while Type II lesions occurred in 2 males (1.9%) and 1 female (1.1%). Lesions of little apparent clinical significance (Types I and II) occurred in 10 males (9.5%) and 11 females (12.3%).

In Table C 25, the frequency of Type I and II lesions in the two sexes is compared in dogs under and over nine years. In dogs under nine years, 63 males (95.5%) and 42 females (89.4%) show normal mural valves. Type I lesions were seen in 3 males (4.5%) and 5 females (10.6%). No Type II lesions were observed. Therefore, lesions of little apparent clinical significance occurred in 3 males (4.5%) and 5 females (10.6%). These results suggest that these lesions may be of greater frequency in the female but there is no evidence of progression in either sex. No Type III or IV lesions were observed.

In dogs over nine years old, 32 males (82.1%) and 36 females (85.7%) showed no abnormality of the mural valves. Type I lesions were seen in 5 males (12.8%) and 5 females (11.9%) while Type II lesions were seen in 2 males (5.1%) and 1 female (2.4%). Therefore lesions of little apparent clinical significance occurred in 7 males (17.9%) and 6 females (14.3%). These results show that these lesions have little progressive tendency and that there may be a slightly greater frequency in the male than in the female but the total number of dogs showing lesions was small.

Table C 26, shows the sex distribution of chronic valvular disease of the mural cusp by age groups. In dogs under five years, 42 males (93.3%) and 29 females (93.6%) had normal mural valves. Type I lesions were seen in 3 males (6.7%) and 2 females (6.4%), these lesions represent

the only manifestation of chronic valvular disease in this age group. These results suggest that there is no sex difference in the frequency or severity of chronic valvular disease of the mural cusp.

In the age group five to eight years inclusive, 21 males (100.0%) and 13 females (81.3%) had normal mural valves. A total of 3 females (18.7%) had Type I lesions only. These results suggest that Type I lesions are more prevalent in the female than in the male in this age group.

In the age group nine to twelve years inclusive, 26 males (83.9%) and 29 females (82.9%) had normal mural valves. Type I lesions were seen in 4 males (12.9%) and 5 females (14.3%) while Type II lesions occurred in 1 male (3.2%) and 1 female (2.9%). Therefore lesions of little apparent clinical significance (Types I and II) occurred in 5 males (16.1%) and 6 females (17.2%). These results suggest that there is no significant sex difference in the frequency or severity of chronic valvular disease of the mural cusp in dogs nine to twelve years old inclusive.

In dogs thirteen to sixteen years inclusive, 6 males (75.0%) and all 7 females (100.0%) had normal mural valves. Only 1 male (12.5%) had a Type I lesion and one other male (12.5%) had a Type II lesion. Therefore, lesions of little apparent clinical significance (Types I and II) occurred in 2 males (25.0%) only. These results suggest that Type I and II lesions are more frequent in the male than in the female.

To summarise all these results on Table C 26, it is clear that there is no significant difference in the frequency or severity of chronic valvular disease of the mural cusp of the right atrioventricular valve.

The comparison of the year-by-year frequency and severity of chronic valvular disease in the mural cusp of both sexes (Table C 27) shows that the number of individuals of either sex and any particular age is small. Thus the interpretation of any results is difficult. However, it is clear that Type I lesions are not seen in males under three years and not seen

in females under four years. Type II lesions are not seen in dogs under 11 years.

In Figure C 25, the sex distribution of chronic valvular disease of the mural cusp is compared. The frequency of lesions in the male was 9.5% compared with 12.3% in the female. Type I lesions were seen in 7.6% of males and 11.2% of females while Type II lesions occurred in 1.9% of males and 1.1% of females. No Type III or IV lesions were observed.

While it is clear that the frequency of lesions in the mural cusp is so low that no valid conclusions can be reached with regard to any sex difference, the mild nature of these lesions is striking, especially when it is compared with the closely related septal cusp.

D The Aortic Valve

The various grades of chronic valvular disease were observed in 8 males (7.7%) and 6 females (6.3%).

Table D 28 shows that Type I lesions occurred in 2 females (2.1%) while Type II lesions were seen in 3 males (2.9%) and 2 females (2.1%). Therefore, lesions of little apparent clinical significance (Types I and II) occurred in 3 males (2.9%) and 4 females (4.2%). Type III lesions were seen in 3 males (2.9%) and 2 females (2.1%) while Type IV lesions were seen in 2 males (1.9%) only. Thus lesions of apparent clinical significance (Types III and IV) occurred in 5 males (4.8%) and 2 females (2.1%).

In Table D 28 , the frequency and severity of chronic valvular disease of the aortic valve in both sexes under and over nine years is compared. In dogs under nine years, 63 males (95.5%) and 45 females (95.7%) had normal aortic valves. Type I lesions were seen in 1 female (2.1%) and Type II lesions in 1 male (1.5%) and 1 female (2.1%). Thus, lesions of little apparent clinical significance (Types I and II) were seen in 1 male (1.5%) and 2 females (4.2%). Type III lesions were seen in 2 males (3.0%) and no Type IV lesions were observed. Lesions of apparent clinical significance (Type III) were seen in 2 males (3.0%).

In dogs over nine years old 34 males (87.2%) and 38 females (90.5%) had normal aortic valves. Type I lesions were seen in 1 female (2.4%) and Type II lesions occurred in 2 males (5.1%) and 1 female (2.4%). Lesions of little apparent clinical significance occurred in 5.1% of males and 4.8% females. Type III lesions were seen in 1 male (2.6%) and 2 females (4.7%), and Type IV lesions occurred in 2 males (5.1%) only. Lesions of apparent clinical significance (Type III and IV) occurred in 3 males (7.7%) and 2 females (4.7%).

While the small number of affected animals involved limit the validity of any conclusions, lesions in the aortic valve do appear to be more progressive than in the mural cusp of the right atrioventricular valve.

Table D 29, shows the sex distribution of chronic valvular disease in the aortic valve by age groups. In dogs under five years, 45 males (100.0%) and 29 females (93.6%) had normal aortic valves. Type I lesions were seen in 1 female (3.2%) and Type II lesions were seen in 1 female (3.2%) too. Therefore, lesions of little apparent clinical significance (Types I and II) were confined to 2 females (6.4%). No Type III or IV lesions were observed. The results suggest that chronic valvular disease occurs more readily in the aortic valve of the female.

In the age group five to eight years inclusive, 18 males (85.7%) and 16 females (100.0%) had normal aortic valves. Type II lesions were seen in 1 male (4.8%) and Type III lesions were seen in 2 males (9.5%). No lesions were seen in females in this age group. The results suggest that chronic valvular disease of the aortic valves occurred more readily in the male than in the female.

In the age group nine to twelve years inclusive, 27 males (87.1%) and 32 females (91.4%) had normal heart valves. Type I lesions were seen in 1 female (2.9%) only and Type II lesions occurred in 2 males (6.5%) and 1 female (2.9%). Lesions of little apparent clinical significance occurred in 2 males (6.5%) and 2 females (5.8%). Type III lesions were seen in 1 female (2.9%) only and Type IV lesions occurred in 2 males (6.5%) only. Lesions of apparent clinical significance (Types III and IV) occurred in 1 female (2.9%) and 2 males (6.5%). These results suggest that these lesions in the aortic valve may be slightly more progressive in the male than in the female.

In the age group thirteen to sixteen years, 7 males (87.5%) and 6 females (85.7%) had normal aortic valves. Type III lesions were seen in 1 male (12.5%) and 1 female (14.3%) only. These results are largely inconclusive.

To summarise all these results on Table D29, it is clear that chronic valvular disease is uncommon in the aortic valve but when it occurs these lesions are more progressive than in the case of the mural cusp of the right atrioventricular valve.

The comparison of the year-by-year frequency and severity of chronic valvular disease in the aortic valve of both sexes (Table D 30) shows that the number of affected individuals of any one sex and age is so low that it is impossible to draw any valid conclusions. Lesions were not detected in the aortic valves of dogs under two years, at which age 1 female (12.5%) showed a Type I lesion. A Type II lesion was seen in 1 female (25.0%) in the third year of life. Type III lesions were first observed in 1 five-year old male (16.7%) and Type IV lesions in 1 ten year old male (7.1%).

In Figure D 28, the sex distribution of chronic valvular disease in the aortic valve is compared. The frequency of these lesions in the male was 7.7% and in the female 6.6%. Type I lesions were seen in 2.2% of females and Type II lesions occurred in 2.9% of males and 2.2% of females. Type III lesions were seen in 2.9% of males and 2.2% of females and Type IV lesions were confined to 1.9% of males. No conclusion with regard to a sex predisposition can be drawn from these results but what is clear is that although chronic valvular disease of the aortic valve has a low frequency, these lesions, when they occur, are progressive, unlike lesions in the septal cusp of the right atrioventricular valve.

E The pulmonary valve

No gross lesions were detected in this valve in this series of 200 unselected dogs.

The comparison of the frequency and severity of chronic valvular disease in the heart valves of the two sexes

The frequency and severity of valve lesions in the two sexes is compared one valve with another.

Left atrioventricular valve and septal cusp of right atrioventricular valve

Chronic valvular disease of the left atrioventricular valve was seen in 70.5% of males and 67.4% of females (Table A 19) while these lesions in the septal cusp of the right atrioventricular valve occurred in 52.4% of males and 52.8% of females (Table B 22). Lesions of apparent clinical significance (Types III and IV) occurred in 30.5% of males and 36% of females in the left atrioventricular valve and in 19.0% of males and 11.2% of females in the septal cusp (Table 31). It is clear that there is a close parity in the overall frequency of lesions in the sexes but that there is a higher frequency of severe lesions in the left atrioventricular valve of the female and a lower overall frequency of these lesions in the septal cusp.

If we consider lesions of little apparent clinical significance (Types I and II), 40% of males and 31.4% of females showed lesions in the left atrioventricular valve while 33.3% of males and 41.6% of females showed lesions in the septal cusp (Table 31). It would appear that lesions in the left atrioventricular valve develop more rapidly in the female while in the septal cusp the converse applies.

If we compare the frequency and severity of lesions in dogs under and over nine years in the two sexes, it is seen that in dogs under nine years the lesions occurred in 53.1% of males and 48.9% of females in the left atrioventricular valve (Table A 19) and 40.4% of females and 36.4% of males (Table B 22) in the septal cusp. Lesions of apparent clinical significance (Types III and IV) occurred in 10.5% of males and 19.1% of females in the left atrioventricular valve, while 9.1% of males and 6.4% of females showed lesions in the septal cusp (Table 33). Lesions of little apparent clinical

significance (Types I and II) occurred in 42.6% of males and 29.8% of females in the left atrioventricular valve and in 27.3% of males and 34% of females in the septal cusp (Table 33).

In dogs nine years and over, lesions occurred in 100% of males and 88.0% of females in the left atrioventricular valve (Table A 19) while the septal cusp showed lesions in 79.5% of males and 66.7% of females (Table B 22). Lesions of apparent clinical significance (Types III and IV) were seen in 64.1% of males and 54.7% of females in the left atrioventricular valve, and the septal cusp showed similar lesions in 35.9% of males and 16.7% of females (Table 33). Lesions of little apparent clinical significance (Types I and II) occurred in the left atrioventricular valves of 35.9% of males and 33.3% of females, while 43.6% of males and 50.0% of females showed similar lesions in the septal cusp of the right atrioventricular valve.

There is a close parity in the frequency and severity of chronic valvular disease in the two sexes. There is a greater frequency of Type III and IV lesions in the left atrioventricular valves of females under nine years old. The overall frequency of lesions in the septal cusp is greater in the female under nine years. In dogs nine years and over, chronic valvular disease has a greater frequency and severity in the male than in the female. It would appear that there is a factor which induces the more rapid development of chronic valvular disease in a proportion of younger females.

If these dogs are re-classified into four age groups (Table 32) we see that in dogs under five years, 35.5% of males and 38.6% of females show lesions in their left atrioventricular valves compared with 24.4% of males and 25.8% of females with similar lesions in the septal cusps. Lesions of apparent clinical significance (Types III and IV) occurred in 2.2% of males and 19.3% of females in the left atrioventricular valve while similar lesions were seen in 2.2% of males (one case of congenital pulmonary

stenosis) and 3.2% of females in the septal cusp. Lesions of little apparent clinical significance (Types I and II) were seen in 33.3% of males and 19.3% of females in the left atrioventricular valve compared with 22.2% of males and 22.6% of females in the septal cusp.

In the age group five to eight years inclusive (Table 32), lesions occurred in the left atrioventricular valve of 90.5% of males and 68.7% of females while 61.9% of males and 68.7% of females showed lesions in the septal cusp. Lesions of apparent clinical significance (Types III and IV) were seen in 28.6% of males and 18.7% of females in the left atrioventricular valve, compared with 23.8% of males and 12.5% of females with similar lesions in the septal cusp. Types I and II lesions were seen in the left atrioventricular valve of 61.9% of males and 50% of females, while 38.1% of males and 56.2% of females show similar lesions in the septal cusp.

Of the dogs aged nine to twelve years inclusive, all the males and 85.7% of the females showed chronic valvular disease compared with 74.1% of males and 68.5% of females with similar lesions in the septal cusp (Tables A 20 B 23). In the left atrioventricular valves Types III and IV lesions were seen in 54.8% of males and 51.4% of females, while in the septal cusp similar lesions were seen in 29.0% of males and 14.3% of females (Table 32). Lesions of little apparent clinical significance (Types I and II) were detected in 45.1% of males and 54.2% of females in the septal cusp compared with 45.2% of males and 34.3% of females with similar lesions in the left atrioventricular valve.

In dogs thirteen to sixteen years inclusive, all the males and females showed lesions in the left atrioventricular valve, while all the males and 57.2% of the females showed lesions in the septal cusp (Table A 20 : Table B23). Types III and IV lesions were observed in the left atrioventricular valve of all the males and 71.4% of the females and similar lesions affected the septal cusps of 62.5% of males and 28.6% of females. Lesions of little

apparent clinical significance (Types I and II) affected the left atrioventricular valve of 28.6% of females only and similar septal cusp lesions were seen in 37.5% of males and 28.6% of females (Table 32).

While the smaller sample size of the age groups five to eight years and thirteen to sixteen years inclusive may have influenced the results to some extent, it is clear in the three age groups five years and over that the frequency and severity of chronic valvular disease is greater in the male than in the female. In dogs under five years the situation is reversed and there is a greater frequency and severity of lesions in the female than in the male. It appears that there is some factor which accelerates the development of these lesions in some females.

The septal and mural cusps of the right atrioventricular valve

Chronic valvular disease was seen in the septal cusps of 52.4% of males and 52.8% of females compared with 9.5% of males and 12.4% of females with similar lesions in the mural cusps. Lesions of apparent clinical significance (Types III and IV) occurred in 19.0% of males and 11.2% of females in the septal cusp but no similar lesions were seen in the mural cusp (Tables 22.24). It is clear that there are considerable differences in the frequency and severity of chronic valvular disease in these two cusps of the same valve.

Lesions of little apparent clinical significance (Types I and II) were seen in the septal cusp of 33.3% of males and 41.6% of females while these lesions were seen in 9.5% of males and 12.4% of females (Table 31). Although these lesions have a greater frequency in the septal than in the mural cusp, lesions in the latter cusp persist without developing into Type III and IV lesions in later life.

The comparison of frequency and severity in dogs under and over nine years in both sexes shows that in dogs under nine years, valve changes occurred in the septal cusps of 36.4% of males and 40.4% of females and in 4.5% of males and 10.6% of females with lesions in the mural cusps (Table 33).

Lesions of little apparent clinical significance (Types I and II) were seen in 27.3% of males and 34.0% of females affecting the septal cusps while similar lesions were seen in 4.5% of males and 10.6% of females in the mural cusps (Table 33). Types III and IV lesions were confined to the septal cusps of 9.1% of males and 6.4% of females.

In dogs nine years and over, chronic valvular disease was seen in the septal cusps of 79.5% of males and 66.7% of females while 17.9% of males and 14.3% of females showed similar lesions in the mural cusps. Lesions of little apparent clinical significance (Types I and II) were observed in 43.6% of males and 50.0% of females in the septal cusps compared with 17.9% of males and 14.3% of females with similar lesions in the mural cusps (Tables 33). Lesions of apparent clinical significance (Types III and IV) were confined to the septal cusps of 35.9% of males and 16.7% of females (Table 33).

There is a greater frequency of chronic valvular disease in the septal cusp than in the mural cusp, despite the fact that these two cusps comprise the right atrioventricular valve. In dogs under nine years, lesions are more frequent in the female than in the male, whereas, in dogs over nine years, this situation is reversed. No Type III or IV lesions were seen in the mural cusps.

If these same dogs are re-classified into four age groups and the frequency and severity of chronic valvular disease in the two sexes is compared, we see that in dogs under five years old 24.4% of males and 25.8% of females show lesions in the septal cusps compared with 6.7% of males and 6.4% of females with lesions in the mural cusp (Table 32). Lesions of little apparent clinical significance (Types I and II) were seen in the septal cusps of 22.2% of males and 22.6% of females while 6.7% of males and 6.4% of females had similar lesions in the mural cusps. Type III and IV lesions were confined to the septal cusps of 2.2% of males (one case of congenital pulmonary stenosis) and 3.2% of females (Table 32).

In the age group five to eight years inclusive, lesions occurred in the septal cusps of 61.9% of males and 68.7% of females while similar lesions were confined to 18.7% of females in the mural cusps. Type I and II lesions were seen in 38.1% of males and 56.2% of females in the septal cusp and in the mural cusps of 18.7% of females only. Type III and IV lesions were confined to the septal cusps of 23.8% of males and 12.5% of females (Table 32).

In dogs nine to twelve years old inclusive, 74.1% of males and 68.5% of females showed lesions in the septal cusp compared with 16.1% of males and 17.2% of females with similar lesions in the mural cusps (Table 32). Lesions of little apparent clinical significance occurred in the septal cusps of 45.1% of males and 54.2% of females compared with 16.1% of males and 17.2% of females showing these changes in the mural cusps. Type III and IV lesions were confined to the septal cusps of 29.0% of males and 14.3% of females (Table 32).

In the age group thirteen years and over, all the males and 57.2% of females showed lesions in the septal cusps while lesions were confined to 25.0% of males in the mural cusps (Table 32). Types I and II lesions were seen in 37.5% of males and 28.6% of females in the septal cusps and in the mural cusps of 25.0% of males only. Lesions of apparent clinical significance (Types III and IV) were confined to 62.5% of males and 28.6% of females in the septal cusps only.

There is little evidence for the progression of lesions in the mural cusp. The inconsistency in the frequency of lesions in the mural cusp is probably due to the low overall incidence of such lesions in this situation. These results are even more striking when compared with the far higher frequency and severity of chronic valvular disease in the septal cusp. There appears to be a closer relationship of frequency and severity of lesions between the left atrioventricular valve and the septal cusp

of the right atrioventricular valve.

The left atrioventricular valve and the aortic valve

Chronic valvular disease was seen in the left atrioventricular valve of 70.5% of males and 67.4% of females in the left atrioventricular valve while similar lesions occurred in the aortic valves of 7.7% of males and 6.7% of females. Lesions of apparent clinical significance (Types III and IV) occurred in 30.5% of males and 36.0% of females in the left atrioventricular valve compared with 4.8% of males and 2.1% of females with lesions in the aortic valve (Tables 19-29). There is a gross disparity in the frequency of lesions in these two valves.

Lesions of little apparent clinical significance (Types I and II) were seen in the left atrioventricular valve of 40.0% of males and 31.4% of females while in the aortic valve 2.9% of males and 4.2% of females showed lesions of similar types (Table 31).

A comparison of the frequency and severity of chronic valvular disease in dogs under and over nine years shows that in dogs under nine years, the left atrioventricular valve was affected in 53.1% of males and 48.9% of females compared with 4.5% of males and 4.2% of females with lesions in the aortic valves. Type III and IV lesions occurred in the left atrioventricular valves of 10.5% of males and 19.1% of females, while the aortic valves showed these lesions in 3.0% of males only (Table 33). Lesions of little apparent clinical significance (Types I and II) occurred in the left atrioventricular valves of 42.6% of males and 29.8% of females compared with 1.5% of males and 4.2% of females with similar lesions in the aortic valves (Table 33).

In dogs nine years and over, chronic valvular disease of the left atrioventricular valve occurred in all the males and 88.0% of the females compared with 12.8% of males and 9.5% of females with lesions in the aortic valves. Types III and IV lesions occurred in 64.1% of males and 54.7% of females in the left atrioventricular valve, while in the aortic valve

7.7% of males and 4.7% of females showed similar lesions (Tables 33). Types I and II lesions were seen in the left atrioventricular valves of 35.9% of males and 33.3% of females while 5.1% of males and 4.8% of females had similar lesions in the aortic valve (Table 33).

Chronic valvular disease has a far greater frequency in the left atrioventricular valve than in the aortic valve. The low overall incidence of lesions in the latter valve makes it difficult to interpret these results, but it would appear that the aortic valve lesion is progressive as Types III and IV lesions were observed. These lesions appear to have a greater frequency in the male than in the female.

If these dogs are divided into four age groups, we see that in dogs under five years, 35.5% of males and 38.6% of females show lesions in the left atrioventricular valves, while only 6.4% of females showed lesions in the aortic valve (Table 32). Lesions of apparent clinical significance (Types III and IV) were seen only in the left atrioventricular valves of 2.2% of males and 19.3% of females. Types I and II lesions were seen in the left atrioventricular valves of 33.3% of males and 19.4% of females and in the aortic valves of only 6.4% of females (Table 32).

In dogs five to eight years old inclusive, lesions were seen in the left atrioventricular valves of 90.5% of males and 68.7% of females compared with 14.3% of males only with lesions in the aortic valve. Lesions of apparent clinical significance were seen in 28.6% of males and 18.7% of females in the left atrioventricular valves, while similar lesions were seen only in 9.5% of males in the aortic valve (Table 32). Lesions of little apparent clinical significance were seen in the left atrioventricular valves of 61.9% of males and 50.0% of females, compared with 4.8% of males only with lesions in the aortic valves.

In the age group nine to twelve years inclusive, all the males and 85.7% of the females showed lesions in the left atrioventricular valves

compared with 13.0% of males and 8.7% of females with lesions in the aortic valve. Type III and IV lesions were seen in the left atrioventricular valves of 54.8% of males and 51.4% of females while similar lesions were observed in the aortic valves of 6.5% of males and 2.9% of females (Table 32). Type I and II lesions occurred in 45.2% of males and 34.3% of females affecting the left atrioventricular valve while the aortic valve was affected with similar lesions in 6.5% of males and 5.8% of females.

In dogs thirteen years and over, all the males and females had lesions in the left atrioventricular valve while only 12.5% of the males and 14.3% of the females showed lesions in the aortic valve. Type III and IV lesions were seen in the left atrioventricular valve of all the males and in 71.4% of the females compared with 12.5% of the males and 14.3% of the females with similar lesions in the aortic valve (Tables 32). Lesions of little apparent clinical significance were confined to the left atrioventricular valve of 28.6% of females.

In general, lesions in the left atrioventricular valve increase in frequency and severity with advancing age and are more severe in the male than in the female (the exception is the higher prevalence and severity of lesions in some females under five years). In the aortic valve, these lesions have a far lower frequency and no clear sex predisposition is apparent. It is evident, however, that chronic valvular disease may progress to its more severe forms (Types III and IV) unlike the mural cusp of the right atrioventricular valve.

The pulmonary valve

No gross lesions were detected in any pulmonary valve in this series of dogs.

The comparative age of onset of chronic valvular disease in the heart valves of the two sexes

Type I lesions (the earliest gross manifestation of chronic valvular disease) were detected in the left atrioventricular valves and the septal cusps of the right atrioventricular valve of males under one year old

while such lesions were not seen in females under two years old (Table 34). Such lesions were not seen in the mural cusp of the right atrioventricular valve until the males had reached three years old and the females were four years old. In the aortic valve Type I lesions were seen in only one female at two years old.

Type II lesions were seen in the left atrioventricular valve of males under one year did not appear in the females until they were one year old. In the septal cusp of the right atrioventricular valve such lesions appeared in the male at one year and in the female at two years, while in the mural cusp these lesions did not occur in either sex under eleven years old. Type II lesions were seen in the aortic valves of the male in the seventh year and in the female in the third year of life.

In the left atrioventricular valve, Type III lesions were first seen in males at three years and females at two years while in the septal cusp of the right atrioventricular valve such lesions appeared at five years in the male and at four years of age in the female. Type III lesions were not seen in the mural cusp of the right atrioventricular valve but in the aortic valve these lesions were seen in the male at five years and in the female at nine years.

Type IV lesions were seen in the left atrioventricular valve and the septal cusp of the right atrioventricular valve of males of eight years and of females of nine years old. Type IV lesions were not seen in the mural cusp of the right atrioventricular valve but these lesions were seen in the aortic valves of males from ten years old.

The various grades of chronic valvular disease tend to occur at an earlier age in the left atrioventricular valve than in the septal cusp of the right atrioventricular valve. Lesions occur much earlier in both these cusps than do similar lesions in the aortic valve or in the mural cusp of the right atrioventricular valve. Furthermore, these lesions tend to

occur at an earlier age in the male than in the female, with the exception of Type III lesions in the left atrioventricular valve and septal cusp of the right atrioventricular valve where the situation was reversed. The close relationship in frequency, severity, age of onset of lesions and sex distribution between the left atrioventricular valve and the septal cusp of the right atrioventricular valve is striking.

No Type III or IV lesions were observed in the mural cusp of the right atrioventricular valve. The low frequency of lesions in the aortic valve makes it impossible to assess any degree of sex predisposition. No gross lesions were detected in the pulmonary valve.

The breed susceptibility to chronic valvular disease

All the breeds represented by more than ten individuals have been listed separately and the frequency and severity of their chronic valvular disease compared with that of the whole two hundred dogs in this series (control group). Five breeds, Alsatian, Border Collie, Boxer, Greyhound and Labrador, were examined, a total of ninety dogs (45% of the total number in this series). It was necessary to assure that each of these breeds were comparable with the control group and so the mean age and sex distribution was established for each breed and compared with the control group.

The Alsatian

A total of sixteen Alsatians (nine males and seven females) were presented with a mean age of 6 years 5 months (Table 35).

In the left atrioventricular valve (Tables 36) normal valves were seen in four Alsatians (25.0%) and 61 controls (30.5%). Type I lesions were seen in three Alsatians (18.7%) and 31 controls (15.5%), while Type II lesions were seen in three Alsatians (18.7%) and 42 controls (21.0%). Lesions of little apparent clinical signifance (Types I and II) occurred in six Alsatians (37.4%) and 73 controls (36.5%) (Tables 37). Type III lesions were seen in three Alsatians (18.7%) and 43 controls (21.5%) and Type IV lesions in three Alsatians (18.7%) and 23 controls (11.5%). Lesions of apparent clinical significance (Types III and IV) were seen in six Alsatians (37.4%) and 66 controls (33.0%). Considering the small number of Alsatians involved, the variations in frequency and severity of chronic valvular disease are not considered to be significant.

In the septal cusp of the right atrioventricular valve, normal valves were seen in seven Alsatians (43.8%) and 95 controls (47.5%) (Tables 37). Type I lesions were seen in three Alsatians (18.7%) and 27 controls (13.5%) while Type II lesions occurred in four Alsatians (25.0%) and 46 controls (23.0%). Thus, lesions of little apparent clinical significance occurred

in seven Alsatians (43.7%) and 73 controls (36.5%) (Tables 37). Type III lesions were seen in two Alsatians (12.5%) and 20 controls (10.0%) while Type IV lesions were confined to 12 controls (6.0%). Lesions of apparent clinical significance were seen in two Alsatians (12.5%) and 32 controls (16.0%). There is some elevation in Type I and II lesions in the Alsatian but this is not considered to be significant.

In the mural cusp of the right atrioventricular valve, normal valves were seen in thirteen Alsatians (81.2%) and 177 controls (88.5%). Type 1 lesions were seen in two Alsatians (12.5%) and 20 controls (10.0%) while Type II lesions were seen in one Alsatian (6.3%) and 3 controls (1.5%). Thus, lesions of little apparent clinical significance were seen in three Alsatians (18.8%) and 23 controls (11.5%). No Type III or IV lesions were observed.

In the aortic valve, normal valves were seen in fourteen Alsatians (87.5%) and 186 controls (93.0%). Type I lesions were seen in two controls (1.0%) only and Type II lesions in one Alsatian (6.3%) and 5 controls (2.5%). Lesions of little apparent clinical significance occurred in one Alsatian (6.3%) and 7 controls (3.5%). Type III lesions occurred in 5 controls only and Type IV lesions in one Alsatian (6.3%) and 2 controls (1.0%). Therefore, lesions of apparent clinical significance occurred in one Alsatian (6.3%) and 7 controls (3.5%).

No gross lesions were observed in the pulmonary valve.

To summarise on the breed susceptibility of the Alsatian, although there is a slightly greater frequency of Type III and IV lesions in the left atrioventricular valve and a greater frequency of Type I and II lesions in the septal cusp of the right atrioventricular valve, these variations must be seen against the small, albeit representative, number of Alsatians that were examined. Therefore, it is concluded that there is no evidence of breed susceptibility to chronic valvular disease in the Alsatian.

The Border Collie

A total of 31 Border Collies, sixteen males, fourteen females and one castrated female, were examined with a mean age of 8 years 6 months. The sex distribution compared well with the control group but the mean age was elevated by 2 years (Table 35).

In the left atrioventricular valve (Tables 36), normal valves were seen in four collies (12.9%) and 61 controls (30.5%). Type I lesions were seen in twelve Collies (38.7%) and 31 controls (15.5%), while Type II lesions were observed in three Collies (9.7%) and 42 controls (21.0%). Lesions of little apparent clinical significance occurred in fifteen Collies (48.4%) and 73 controls (36.5%) (Tables 37). Type III lesions were seen in ten Collies (32.3%) and 43 controls (21.5%) while Type IV lesions were seen in two Collies (6.5%) and 23 controls (11.5%). Lesions of apparent clinical significance (Type III and IV) were seen in twelve Collies (38.8%) and 66 controls (33.0%). The frequency and severity of chronic valvular disease is greater in the Border Collie than in the control group.

In the septal cusp of the right atrioventricular valve (Tables 36) normal septal cusps were seen in eleven Collies (35.5%) and 95 controls (47.5%). Type I lesions were seen in eight Collies (25.8%) and 27 controls (13.5%), while Type II lesions occurred in ten Collies (32.3%) and 46 controls (23.0%). Lesions of little apparent clinical significance (Tables 37) were seen in eighteen Collies (58.1%) compared with 73 controls (36.5%). Type III lesions were seen in one Collie (3.2%) and 20 controls (10.0%) and Type IV lesions in one Collie 3.2% and 12 controls (6.0%). Thus lesions of apparent clinical significance were seen in two Collies (6.4%) and 32 controls (16.0%). There is a far greater frequency, although a lesser severity of lesions in the septal cusp of the Border Collie.

In the mural cusp of the right atrioventricular valve (Tables 36) normal mural cusps were seen in all the Border Collies but only in 177

(88.5%) of the controls. The total absence of lesions in the mural cusps of the Border Collie was unexpected.

In the aortic valve (Tables 36) normal valves were seen in 29 Collies (93.6%) and 186 controls (93.0%). Type I lesions were seen in one Border Collie (3.2%) and 2 controls (1.0%) and Type II lesions were confined to 5 controls (2.5%). Lesions of little apparent clinical significance (Tables 37) were seen in one Collie (3.2%) and 7 controls (3.5%). Type III lesions occurred in one Collie (3.2%) and 5 controls (2.5%) while Type IV lesions were confined to 2 controls (1.0%). Lesions of apparent clinical significance were seen in one Collie (3.2%) and 7 controls (3.5%). There is a close parity of frequency and severity of lesions between the Border Collies and the control group.

No gross lesions were observed in the pulmonary valve.

To summarise on the breed susceptibility of the Border Collie to chronic valvular disease, these lesions have a greater frequency and severity in the left atrioventricular valve. These lesions are far more frequent, if less severe, in the septal cusp of the right atrioventricular valve. The overall frequency of lesions in the mural cusp of the right atrioventricular valve and the aortic valve is low and so, where small numbers of dogs are involved, irregularities tend to occur, as in this case where no lesions were observed in the mural cusp. However, there is a clear-cut increase in frequency and, to some extent, an increase in the severity of lesions in the left atrioventricular valve and septal cusp, but the mean age of the Border Collie is two years older than the control group. It has already been demonstrated how these lesions are age-dependent and, therefore, it is suggested that this apparent breed susceptibility is not a true one but is associated with the greater mean age of these Border Collies.

The Boxer

A total of eleven Boxers, six males and five females, were examined with a mean age of 7 years 0 months.

In the left atrioventricular valve (Tables 36) normal valves were seen in two Boxers (18.2%) and 61 controls (30.5%). Type I lesions were seen in one Boxer (9.1%) and 31 controls (15.5%) while Type II lesions were seen in 3 Boxers (27.3%) and 42 controls (21.0%). Thus lesions of little apparent clinical significance (Tables 37) were seen in four Boxers (36.4%) and 73 controls (36.5%). Type III lesions occurred in four Boxers (36.4%) and 43 controls (21.5%) and Type IV lesions in one Boxer (9.1%) and 23 controls (11.5%). Lesions of apparent clinical significance occurred in five Boxers (45.5%) and 66 controls (33.0%). Chronic valvular disease appears to have a greater frequency and severity in the left atrioventricular valve of the Boxer.

In the septal cusp of the right atrioventricular valve (Tables 36) normal septal cusps were seen in eight Boxers (72.7%) and 95 controls (47.5%). Type I lesions were confined to 27 controls (13.5%), while Type II lesions were seen in one Boxer (9.1%) and 46 controls (23.0%). Lesions of little apparent clinical significance(Tables 37) occurred in one Boxer (9.1%) and 73 controls (36.5%). Type III lesions were observed in one Boxer (9.1%) and 20 controls (10%) while Type IV lesions occurred in one Boxer (9.1%) and 12 controls (6.0%). Thus lesions of apparent clinical significance were seen in two Boxers (18.2%) and 32 controls (16.0%). Chronic valvular disease has an equal severity but a far lower frequency in the septal cusp of the right atrioventricular valve of the Boxer.

In the mural cusp of the right atrioventricular valve (Tables 36) normal mural cusps were found in ten Boxers (90.9%) and 177 controls (88.5%). Type I lesions were seen in one Boxer (9.1%) and 20 controls (10.0%) while Type II lesions were confined to 3 controls (1.5%). Lesions of little apparent clinical significance (Tables 37) were seen in 1 Boxer (9.1%) and 23 controls (11.5%). No Type III or IV lesions were observed. There is no evidence of a breed susceptibility in respect of this valve cusp.

In the aortic valve (Tables 36) normal valves were seen in eight Boxers (72.7%) and 186 controls (93.0%). Type I lesions were confined to 2 controls (1.0%), while Type II lesions were seen in one Boxer (9.1%) and 5 controls (2.5%). Lesions of little apparent clinical significance (Tables 37) were seen in one Boxer (9.1%) and 7 controls (3.5%). Type III lesions were seen in one Boxer (9.1%) and 5 controls (2.5%) while Type IV lesions occurred in one Boxer (9.1%) and 2 controls (1.0%). Lesions of apparent clinical significance were seen in two Boxers (18.2%) and 7 controls (3.5%).

No gross lesions were seen in the pulmonary valve .

To summarise on the breed susceptibility of the Boxer, the variations in the frequency and severity of chronic valvular disease in the different valves suggest that the sample size (11) is too small and unrepresentative to give any meaningful result. Therefore, it is assumed that there is no breed susceptibility in the Boxer.

The Greyhound

A total of 14 Greyhounds, eight males and six females, were examined with a mean age of 1 year 9 months. While there is a slight preponderance of males, the sex distribution is reasonable but the mean age is well below the control group (6 years 5 months) (Table 36).

In the left atrioventricular valve (Tables 36) normal valves were seen in 8 Greyhounds (57.1%) and 61 controls (30.5%). Type I lesions were seen in 2 Greyhounds (14.3%) and 31 controls (15.5%) while Type II lesions occurred in 3 Greyhounds (21.4%) and 42 controls(21.0%). Lesions of little apparent clinical significance (Tables 37) were seen in 5 Greyhounds (35.7%) and 73 controls (36.5%). Type III lesions occurred in 1 Greyhound (7.1%) and 43 controls (21.5%), while Type IV lesions were seen only in 23 controls (11.5%). Lesions of apparent clinical significance were observed in 1 Greyhound (7.1%) and 66 controls (33.0%). Overall, there is a marked

reduction in frequency and the severity of chronic valvular disease in the left atrioventricular valve of the Greyhound.

In the septal cusp of the right atrioventricular valve (Tables 36) normal septal cusps were seen in nine Greyhounds (64.3%) and 95 controls (47.5%). Type I lesions occurred in three Greyounds (21.4%) and 27 controls (13.5%) while Type II lesions were seen in two Greyhounds (14.3%) and 46 controls (23.0%). Lesions of little apparent clinical significance (Tables 37) were seen in five Greyhounds (35.7%) and 73 controls (36.5%). Lesions of apparent clinical significance (Types III and IV) were confined to 32 controls (16.0%). It is clear that the frequency and severity of chronic valvular disease in the septal cusp is much lower in the Greyhound than in the control group.

In the mural cusp of the right atrioventricular valve (Tables 36) normal mural cusps were seen in twelve Greyhounds (85.7%) and 177 controls (88.5%). Type I lesions were seen in two Greyhounds (14.3%) and 20 controls (10.0%) while Type II lesions were confined to 3 controls (1.5%). Lesions of little apparent clinical significance (Tables 37) were seen in two Greyhounds (14.3%) and 23 controls (11.5%). No Type III or IV lesions were observed. On the basis of this result, both the Greyhounds and control group have a similar frequency of lesions in the mural cusp.

In the aortic valve (Tables 36) normal valves were seen in thirteen Greyhounds (92.9%) and 186 controls (93.0%) while Type I lesions were confined to 2 controls (1.0.). Type II lesions were seen in one Greyhound (7.1%) and 5 controls (2.5%). Lesions of little apparent clinical significance were seen in one Greyhound (7.1%) and 7 controls (3.5%). Type III and IV lesions (Tables 36 . 37) were confined to 7 controls (3.5%). Lesions in the aortic valve of the Greyhound are less severe than in the control group.

No gross lesions were seen in the pulmonary valve.

Chronic valvular disease has far lower frequency and severity in these Greyhounds than in the control group. The age dependence of chronic valvular disease has already been established and it is suggested that the significantly lower mean age of these Greyhounds is largely responsible for this reduction in frequency.

The Labrador

A total of eighteen Labradors, ten males, six females and two castrated females, were examined with a mean age of 6 years, 8 months. (Table 36)

In the left atrioventricular valve (Tables 36) normal valves were seen in two Labradors (11.1%) and 61 controls (30.5%). Type I lesions were seen in six Labradors (33.3%) and 31 controls (15.5%) while Type II lesions were seen in five Labradors (27.8%) and 42 controls (21.0%). Lesions of little apparent clinical significance occurred in eleven Labradors (61.1%) and 73 controls (36.5%). Type III lesions were seen in three Labradors (16.7%) and 43 controls (21.5%) and Type IV lesions occurred in two Labradors (11.1%) and 23 controls (11.5%). Thus, lesions of apparent clinical significance (Tables 37) were seen in five Labradors (27.8%) and 66 controls (33.0%). Chronic valvular disease in the left atrioventricular valve of the Labrador has a greater frequency than in the control group.

In the septal cusp of the right atrioventricular valve (Tables 36) normal septal cusps were seen in seven Labradors (38.8%) and 95 controls (47.5%). Type I lesions were seen in three Labradors (16.7%) and 27 controls (13.5%), while Type II lesions occurred in two Labradors (11.1%) and 46 controls (23.0%). Lesions of little apparent clinical significance (Tables 37) occurred in five Labradors (27.8%) and 73 controls (36.5%). Type III lesions were seen in three Labradors (16.7%) and 20 controls (10.0%) and Type IV lesions in a further three Labradors (16.7%) and only 12controls (6.0%). Thus, lesions of apparent clinical significance were

seen in six Labradors (33.3%) and 32 controls (16.0%). Clearly the frequency and severity of chronic valvular disease in the septal cusp of the right atrioventricular valve is greater in the Labrador than in the control group.

In the mural cusp of the right atrioventricular valve (Tables 36) normal mural cusps were seen in fourteen Labradors (77.8%) and 177 controls (88.5%). Type I lesions occurred in 4 Labradors (22.2%) and 20 controls (10.0%) while Type II lesions were confined to 3 controls (1.5%). Thus, lesions of little apparent clinical significance (Tables 37) were seen in four Labradors (22.2%) and 23 controls (11.5%). Chronic valvular disease in the mural cusp of the right atrioventricular valve is more frequent in the Labrador than in the control group.

In the aortic valve (Tables 36) normal valves were seen in seventeen Labradors (94.4%) and 186 controls (93.0%). Type I lesions were seen in one Labrador (5.6%) and 2 controls (1.0%) while Type II lesions were confined to 5 controls (2.5%). Thus, lesions of little apparent clinical significance (Tables 37) occurred in one Labrador (5.6%) and 7 controls (3.5%). Type III and IV lesions were confined to 7 controls (3.5%). The frequency and severity of chronic valvular disease in the aortic valve is greater in the control group than in the Labrador but the low overall incidence of lesions in this valve makes it difficult to assess the significance, if any, of this observation.

To summarise on the breed susceptibility to chronic valvular disease in the Labrador, the sex distribution and mean age are close to those of the control group. Thus, the far higher frequency of lesions in the left atrioventricular valve, the higher the frequency and severity of lesions in the septal cusp of the right atrioventricular valve, and the higher frequency of lesions in the mural cusp, suggest there may be a measure of breed susceptibility in the Labrador, even accepting that the number of dogs is small. Such an observation can only be confirmed by a further survey.

General conclusions on breed susceptibility

In this series only five breeds were represented by more than ten individuals and these were considered to be worthy of further study with regard to breed susceptibility.

Of these, the Alsatian and the Boxer were considered to show no significant variation with regard to frequency or severity from that witnessed in the control group

Two further breeds, namely, the Border Collie and the Greyhound did show variations from the normal which were not deemed to be due to breed susceptibility or resistance. It has been established that chronic valvular disease is an age-dependent lesion and thus the irregularities witnessed in these two breeds were attributed to the ages of the dogs concerned. The mean age of the Border Collies was over two years older than the control group; thus the increased frequency and severity of lesions in these dogs was attributed to their age rather than to a breed susceptibility. Conversely, the mean age of the Greyhounds was more than four years below that of the control group; thus the markedly lowered frequency and severity of the lesions in this breed was again attributed to age rather than to a breed resistance.

But, in the case of the Labrador, where the mean age and sex distribution was close, there did appear to be a breed susceptibility, despite the small number of dogs involved.

The true position with regard to the Border Collie, Greyhound and Labrador can only be established by the collection of a further larger series of cases.

The relationship between chronic valvular disease and other intercurrent conditions

In this section, the major causes of death or destruction are listed under the appropriate system (i.e. the principal system involved in any particular disease syndrome) and the frequency and severity of chronic valvular disease in those dogs that succumbed to these intercurrent conditions is also recorded (where more than ten individuals were concerned). A total of seven systems are recorded in this, plus one further group (systemic disease) of 17 dogs which died of Hepatitis contagiosa canis (Rubarth's Disease) and Canine distemper, both systemic virus diseases.

As in the previous section, the frequency and severity of chronic valvular disease in these groups is compared with a control group formed by the whole series of 200 dogs, representing the normal population. In an attempt to assess the comparability of each group with the controls, a mean age and sex distribution has been established for each group (Tables 38).

The Alimentary system

This group included 20 affected dogs, eleven males (55.0%) and eight females (40.0%), and one castrated female (5.0%) with a mean age of 7 years 6 months. These dogs had died or were destroyed as a result of a variety of conditions, including foreign bodies in the intestine, anal fistula, three cases of acute haemorrhagic gastro-enteritis, chronic enteritis, megacolon, two cases of gastric and duodenal ulceration, hepatitis, bile duct carcinoma, acute pancreatitis and adenocarcinoma of the pancreas (Tables 38).

In the left atrioventricular valve (Tables 39), normal valves were seen in three affected dogs (15.0%) and 61 controls (30.5%). Type I lesions were seen in seven affected dogs (35.0%) and 31 controls (15.5%), while Type II lesions were seen in four affected dogs (20.0%) and 42 controls

(21.0%) (Tables 40). Thus, lesions of little apparent clinical significance (Types I and II) were seen in eleven affected dogs (55.0%) and 73 controls (36.5%). Type III lesions were seen in three affected dogs (15.0%) and 43 controls (21.5%), while Type IV lesions occurred in a further three affected dogs (15.0%) and 23 controls (11.5%). Thus lesions of apparent clinical significance (Types III and IV) occurred in six affected dogs (30.0%) and 66 controls (33.0%). There is a greater frequency of Types I and II lesions in the left atrioventricular valves of alimentary cases.

In the septal cusp of the right atrioventricular valve (Tables 39) normal septal cusps were seen in four affected dogs (20.0%) and 95 controls (47.5%). Type I lesions occurred in seven affected dogs (35.0%) and 27 controls (13.5%) and Type II lesions in four affected dogs (20.0%) and 46 controls (23.0%). Thus, lesions of little apparent clinical significance (Types I and II) were seen in eleven affected dogs (55.0%) and 73 controls (36.5%). Type III lesions occurred in two affected dogs (10.0%) and 20 controls (10.0%) while Type IV lesions were seen in three affected dogs (15.0%) and twelve controls (6.0%). Lesions of apparent clinical significance (Tables 40) were seen in five affected dogs (25.0%) and 32 controls (16.0%). There is a greater frequency and severity of chronic valvular disease in the septal cusp of cases of alimentary disease.

In the mural cusp of the right atrioventricular valve (Tables 39), normal mural cusps were seen in nineteen affected dogs (95.0%) and 177 controls (88.5%). Type I lesions were seen in one affected dog (5.0%) and twenty controls (10.0%) while Type II lesions were confined to three controls (1.5%). Thus, lesions of little apparent clinical significance (Types I and II) were seen in one affected dog (5.0%) and 23 controls (11.5%). Chronic valvular disease in the mural cusp of alimentary cases is less common than in the controls, no Type III or IV lesions were observed.

In the aortic valve (Tables 39), normal valves were seen in 18 affected dogs (90.0%) and 186 controls (93.0%). Type I lesions were confined to 2 controls (1.0%) while Type II lesions were seen in 1 affected dog (5.0%) and 5 controls (2.5%). Lesions of little apparent clinical significance (Tables 40) were seen in 1 affected dog (5.0%) and 7 controls (3.5%). Type III lesions were seen in 1 affected dog (5.0%) and 5 controls (3.5%) while Type IV lesions were seen in 2 controls (1.0%) only. Lesions of apparent clinical significance were seen in 1 affected dog (5.0%) and 7 controls (3.5%). There is no significant difference between these two groups. No gross lesions were seen in any pulmonary valve.

In conclusion, while there is a greater frequency of chronic valvular disease in the left atrioventricular valve and the septal cusp of the right atrioventricular valve, no other significant differences were observed in these two groups. This greater frequency of Type I and II lesions may be due to the greater mean age of the alimentary cases.

The cardiovascular system

This group included 31 affected dogs, eighteen males (58.1%), twelve females (38.7%) and one castrated female (3.2%) with a mean age of 6 years 1 month. There is a slight preponderance of males but the mean age is comparable. These dogs died or were destroyed as a result of a variety of conditions which will form the basis of a separate section in this thesis and will merely be listed here. The conditions included six cases of congenital cardiac disease (sub-aortic stenosis, pulmonary stenosis and persistent right aortic arch), two cases of bacterial endocarditis, non-suppurative and suppurative myocarditis, parasitic myocarditis, constrictive pericarditis, twelve cases of insufficiency of the left atrioventricular valve and one heart base tumour (Tables 38).

In the left atrioventricular valve (Tables 39), normal valves were seen in 9 affected dogs (29.0%) and 61 controls (30.5%). Type I lesions

were observed in 5 dogs (16.1%) and 31 controls (15.5%) and Type II lesions occurred in 3 dogs (9.7%) and 42 controls (21.0%). Thus lesions of little apparent clinical significance (Tables 40) were seen in 8 affected dogs (25.8%) and 73 controls (36.5%). Type III lesions were seen in 10 affected dogs (32.3%) and 43 controls (21.5%), while Type IV lesions were seen in 4 affected dogs (12.9%) and 23 controls (11.5%). Lesions of apparent clinical significance occurred in 14 affected dogs (45.2%) and 66 controls (33.0%). There is no significant increase in the frequency of chronic valvular disease in the cardiovascular group but there is a marked increase in severity in a group where twelve dogs died of congestive heart failure due to chronic valvular disease.

In the septal cusp of the right atrioventricular valve (Tables 39), normal septal cusps were seen in 16 affected dogs (51.6%) and 95 controls (47.5%). Type I lesions were seen in 2 affected dogs (6.4%) and 27 controls (13.5%), while Type II lesions were seen in 8 affected dogs (25.8%) and 46 controls (23.0%). Lesions of little apparent clinical significance (Types I and II) occurred in 10 affected dogs (32.2%) and 73 controls (36.5%). Type III lesions were seen in 3 affected dogs (9.7%) and 20 controls (10.0%), while Type IV lesions were seen in 2 affected dogs (6.4%) and 12 controls (6.0%). Lesions of apparent clinical significance occurred in 5 affected dogs (16.1%) and 32 controls (16.0%). There is little to differentiate between the affected and the control group insofar as septal valve lesions are concerned.

In the mural cusp of the right atrioventricular valve (Tables 39), normal mural valves were seen in 25 affected dogs (80.6%) and 177 controls (88.5%). Type I lesions were seen in 4 affected dogs (12.9%) and 20 controls (10.0%) and Type II lesions occurred in 2 affected dogs (6.4%) and 3 controls (1.5%). Lesions of little apparent clinical significance (Tables 40) occurred in 6 affected dogs (19.3%) and 23 controls (11.5%).

No Type III or IV lesions were observed. Lesions of the mural cusp were more prevalent in cardiovascular cases.

In the aortic valve (Tables 39), normal valves were seen in 30 affected dogs (96.8%) and 186 controls (93.0%). Type I and II lesions were confined to 7 controls (3.5%) and Type III lesions were confined to 5 controls (2.5%). However, Type IV lesions were seen in 1 affected dog (3.2%) and 2 controls (1.0%). Lesions of apparent clinical significance (Tables 40) were seen in 1 affected dog (3.2%) and 7 controls (3.5%). There is no greater frequency of valve lesions in the aortic valve of the cardiovascular case.

To summarise, with the exception of the mural cusp of the right atrioventricular valve, there is no significant variation in the frequency of chronic valvular disease in the cardiovascular case. There is, however, a far greater severity of lesions in the left atrioventricular valve which is associated in this group with twelve cases of congestive heart failure due to insufficiency of the left atrioventricular valve.

The endocrine system

This group included 14 affected dogs, three males (21.4%) and 11 females (78.6%), with a mean age of 9 years 9 months. The mean age is grossly elevated above the control group and the sex distribution is heavily in favour of the female. These imbalances are due to the nature of the diseases included in this group, one case of diabetes insipidus, eight cases of diabetes mellitis, one case of adrenal atrophy and four cases of thyroid carcinoma. The majority of the cases recorded here were diabetes mellitis, which occurs almost exclusively in females in middle or old age (Tables 38).

In the left atrioventricular valve (Tables 39), normal valves occurred in 1 affected dog (7.1%) and 61 controls (30.5%). Type I lesions were seen in 2 affected dogs (14.3%) and 31 controls (15.5%), while Type II lesions occurred in 3 affected dogs (21.4%) and 42 controls (21.0%). Lesions of little apparent clinical significance (Tables 40) occurred in 5 affected

dogs (35.7%) and 73 controls (36.5%). Type III lesions occurred in 5 affected dogs (35.7%) and 43 controls (21.5%), while Type IV lesions were seen in 3 affected dogs (21.4%) and 23 controls (11.5%). Lesions of apparent clinical significance were seen in 8 affected dogs (57.1%) and 66 controls (33.0%). Chronic valvular disease has a far greater frequency and severity in the left atrioventricular valve of endocrine cases.

In the septal cusp of the right atrioventricular valve (Tables 39), normal septal cusps were seen in 1 affected dog (7.1%) and 95 controls (47.5%). Type I lesions were seen in 2 affected dogs (14.3%) and 27 controls (13.5%) while Type II lesions occurred in 8 affected dogs (57.1%) and 46 controls (23.0%). Lesions of little apparent clinical significance (Tables 40) were seen in 10 affected dogs (71.4%) and 73 controls (36.5%). Type III lesions were observed in 2 affected dogs (14.3%) and 20 controls (10.0%), while Type IV lesions were seen in 1 affected dog (7.1%) and 12 controls (6.0%). Lesions of apparent clinical significance occurred in 3 affected dogs (21.4%) and 32 controls (16.0%). These valve lesions have a far greater frequency in cases of endocrine disease than in the control group.

In the mural cusp of the right atrioventricular valve (Tables 39), normal mural cusps were seen in 11 affected dogs (78.6%) and 177 controls (88.5%). Type I lesions were seen in 2 affected dogs (14.3%) and 20 controls (10.0%) while Type II lesions occurred in 1 affected dog (7.1%) and 3 controls (1.5%). Thus, lesions of little apparent clinical significance (Tables 40) were seen in 3 affected dogs (21.4%) and 23 controls (11.5%). No Type III or IV lesions were observed. Cases of endocrine disease show a greater frequency of chronic valvular disease of the mural cusp of the right atrioventricular valve.

In the aortic valve (Tables 39), normal valves were observed in 13 affected dogs (92.9%) and 186 controls (93.0%). Type I lesions were observed in 1 affected dog (7.1%) and 2 controls (1.0%), while Type II lesions were confined to 5 controls (3.5%). Lesions of little apparent clinical significance (Types I and II) were seen in 1 affected dog

(7.1%) and 7 controls (3.5%). Type III and IV lesions were confined to 7 controls (3.5%). There is no clear difference between these two groups.

To summarise, with the exception of the aortic valve, there is a greater frequency and, in the case of the left atrioventricular valve, a far greater severity of chronic valvular disease in cases of endocrine disease. It is suggested that much of this increase is due to the greater age of the dogs in this group.

The Locomotor system

This group includes 15 affected dogs, twelve males (80.0%), two females (13.3%) and one castrated female (6.6%), with a mean age of 5 years 7 months. There is a considerable sex imbalance towards the male. The causes of death or destruction included four cases of disc protrusion, two cases of rickets, three osteosarcomas, two cases of Spondylitis deformans and two cases of septic arthritis and myositis (Tables 38).

In the left atrioventricular valve (Tables 39), normal valves were seen in 5 affected dogs (33.3%) and 61 controls (30.5%), Type I lesions were seen in 2 affected dogs (13.3%) and 31 controls (15.5%), while Type II lesions occurred in 3 affected dogs (20.0%) and 42 controls (21.0%). Thus, lesions of little apparent clinical significance (Tables 40) were seen in 5 affected dogs (33.3%) and 73 controls (36.5%). Type III lesions were observed in 2 affected dogs (13.3%) and 43 controls (21.5%) and Type IV lesions in 3 affected dogs (20.0%) and 23 controls (11.5%). Thus, lesions of apparent clinical significance were seen in 5 affected dogs (33.3%) and 66 controls (33.0%). There is no significant difference between these two groups.

In the septal cusp of the right atrioventricular valve (Tables 39), normal septal cusps were seen in 7 affected dogs (46.6%) and 95 controls (47.5%). Type I lesions were confined to 27 controls (13.5%) while Type II lesions were seen in 3 affected dogs (20.0%) and 46 controls (23.0%).

Therefore, lesions of little apparent clinical significance occurred in 3 affected dogs (20.0%) and 73 controls (36.5%). Type III lesions were seen in 3 affected dogs (20.0%) and 20 controls (10.0%), while Type IV lesions occurred in 2 affected dogs (13.3%) and 12 controls (6.0%). Lesions of apparent clinical significance occurred in 5 affected dogs (33.3%) and 32 controls (16.0%). Lesions in the septal cusp of cases of locomotor disease have a greater severity than in the control group.

In the mural cusp of the right atrioventricular valve (Tables 39), normal mural cusps were seen in 13 affected dogs (86.6%) and 177 controls (88.5%). Type I lesions were seen in 2 affected dogs (13.3%) and 20 controls (10.0%), while Type II lesions were seen in 3 controls (1.5%) only. Lesions of little apparent clinical significance (Tables 40) were seen in 2 affected dogs (13.3%) and 23 controls (11.5%). No Type III or IV lesions were observed. There is no significant difference in the frequency or severity of chronic valvular disease in the mural cusp of either group.

In the aortic valve (Tables 39), normal valves were observed in 14 affected dogs (93.3%) and 186 controls (93.0%. Type III lesions occurred in 1 affected dog (6.6%) and 5 controls (2.5%) but Type IV lesions were confined to 2 controls (1.0%). Lesions of apparent clinical significance (Tables 40) were seen in 1 affected dog (6.6%) and 7 controls (3.5%). Type I and II lesions were confined to 7 controls (3.5%). One cannot differentiate between these two groups with regard to the valve lesions.

To summarise, with the exception of the high level of severity of valve lesions in the septal cusp, there is little evidence of any differences in the frequency or severity of chronic valvular disease in cases of locomotor disease when compared with the control group.

<u>The Reproductive system</u>

This group includes 16 affected dogs, 3 male (18.8%) and 13 female (81.2%), with a mean age of 9 years 7 months. There is a marked sex imbalance

towards the female and a grossly elevated mean age which is largely controlled by the nature of the diseases in this group. The causes of death or destruction were, in the female, three cases of post-parturient metritis, three cases of chronic endometritis and seven cases of adenocarcinoma of the mammary gland. In the male, one Sertoli cell tumour, one prostatitis and one case of cystic hyperplasia of the prostate (Tables 38).

In the left atrioventricular valve (Tables 39), normal valves were seen in 1 affected dog (6.3%) and 61 controls (30.5%). Type I lesions were seen in 1 affected dog (6.3%) and 31 controls (15.5%), while Type II lesions occurred in 7 affected dogs (43.7%) and 42 controls (21.0%). Lesions of little apparent clinical significance (Tables 40) were seen in 8 affected dogs (50.0%) and 73 controls (36.5%). Type III lesions were seen in 7 affected dogs (43.7%) and 43 controls (21.5%) but Type IV lesions were confined to 23 controls (11.5%). Thus lesions of apparent clinical significance occurred in 7 affected dogs (43.7%) and 66 controls (33.0%). Chronic valvular disease is more frequent and severe in cases of reproductive disease than in the control group.

In the septal cusp of the right atrioventricular valve (Tables 39), normal septal valves were seen in 7 affected dogs (43.7%) and in 95 controls (47.5%). Type I lesions occurred in 3 affected dogs (18.7%) and in 27 controls (13.5%), while Type II lesions were observed in 5 affected dogs (31.3%) and 46 controls (23.0%). Lesions of little apparent clinical significance (Tables 40) occurred in 8 affected dogs (50.0%) and 73 controls (36.5%). Type III lesions were seen in 1 affected dog (6.3%) and 20 controls (10.0%) while Type IV lesions were confined to 12 controls (6.0%). Thus lesions of apparent clinical significance occurred in 1 affected dog (6.3%) and 32 controls (16.0%). Lesions have a greater frequency in the septal cusps of cases of reproductive disease.

In the mural cusp of the right atrioventricular valve (Tables 39),

normal mural cusps were seen in 14 affected dogs (87.5%) and 177 controls (88.5%). Type I lesions were seen in 2 affected dogs (12.5%) and 20 controls (10.0%), while Type II lesions were confined to 3 controls (1.5%). Thus, lesions of little apparent clinical significance (Tables 40) were seen in 2 affected dogs (12.5%) and 23 controls (11.5%). No Type III or IV lesions were observed. There is no significant difference between these two groups.

In the aortic valve (Tables 39), normal valves were seen in 14 affected dogs (87.5%) and 186 controls (93.0%). Type I and II lesions were confined to 7 controls (3.5%) but Type III lesions occurred in 1 affected dog (6.3%) and 5 controls (2.5%) and Type IV lesions to 1 affected dog (6.3%) and 2 controls (1.0%). Thus lesions of apparent clinical significance (Types III and IV) were seen in 2 affected dogs (12.5%) and 7 controls (3.5%).

No gross lesions were seen in the pulmonary valves.

To summarise, chronic valvular disease has a far greater frequency and severity in cases of reproductive disease in the left atrioventricular valve and a greater frequency in the septal cusp of the right atrioventricular valve. It is suggested that this is due to the greater mean age of the dogs in this group.

The Respiratory System

This group includes 16 dogs, 8 males (50.0%) and 8 females (50.0%) with a mean age of 3 years 11 months. This is a lower mean age group than the control group and a slight imbalance towards the female in the sex ratio. The causes of death or destruction in this group include seven cases of acute broncho-pneumonia and pleurisy, five cases of thoracic nocardiosis, one case of pulmonary oedema, foetal atalectasis, carcinoma of the tonsil and bronchogenic carcinoma (Tables 38).

In the left atrioventricular valve (Tables 39), normal valves were

seen in 8 affected dogs (50.0%) and 61 controls (30.5%). Type I lesions were seen in 2 affected dogs (12.5%) and 31 controls (15.5%), while Type II lesions were seen in 4 affected dogs (25.0%) and 42 controls (21.0%). Thus, lesions of little apparent clinical significance (Tables 40) occurred in 6 affected dogs (37.5%) and 73 controls (36.5%). Type III lesions were seen in 2 affected dogs (12.5%) and 43 controls (21.5%), while Type IV lesions were confined to 23 controls (11.5%). Thus lesions of apparent clinical significance occurred in 2 affected dogs (12.5%) and 66 controls (33.0%). The frequency and severity of chronic valvular disease in the left atrioventricular valve of cases of respiratory disease is much lower than in the control group.

In the septal cusp of the right atrioventricular valve (Tables 39) normal septal cusps were seen in 8 affected dogs (50.0%) and 95 controls (47.5%). Type I lesions occurred in 4 affected dogs (25.0%) and 27 controls (13.5%), while Type II lesions were seen in 4 affected dogs (25.0%) and 46 controls (23.0%). Thus lesions of little apparent clinical significance (Tables 40) were seen in 8 affected dogs (50.0%) and 73 controls (36.5%). Type III and IV lesions were confined to 32 controls (16.0%). Lesions in the septal cusp are less frequent and far less severe in cases of respiratory disease.

In the mural cusp of the right atrioventricular valve (Tables 39), normal mural cusps were seen in 15 affected dogs(93.7%) and 177 controls (88.5%). Type I lesions were seen in 1 affected dog (6.3%) and 20 controls (10.0%), while Type II lesions were confined to 3 controls (1.5%). Lesions of little apparent clinical significance (Tables 40) were seen in 1 affected dog (6.3%) and 23 controls (11.5%). No Type III or IV lesions were observed. Lesions in the mural cusp are less common in cases of respiratory disease than in the controls.

In the aortic valve (Tables 39) normal aortic valves were seen in

15 affected dogs (93.7%) and 186 controls (93.0%). Type I lesions were confined to 2 controls (1.0%), while Type II lesions were seen in 1 affected dog (6.3%) and 5 controls (2.5%). Lesions of little apparent clinical significance (Types I and II) occurred in 1 affected dog (6.3%) and 7 controls (3.5%). Type III and IV lesions were confined to 7 controls (3.5%).

To summarise, chronic valvular disease is consistently less frequent and severe in cases of respiratory disease when compared with the controls.

The Urinary System

This group includes 24 dogs, thirteen males (54.1%), ten females (41.7%) and one castrated female (4.1%), with a mean age of 8 years 4 months. The mean age is elevated over the control group but the sex distribution is similar.

The causes of death or destruction include thirteen cases of chronic interstitial nephritis, five renal tumours and cases of acute and sub-acute interstitial nephritis, one septic nephritis, one urethral obstruction and two cases of massive renal infarction.

In the left atrioventricular valve (Tables 39), normal valves were seen in 4 affected dogs (16.7%) and 61 controls (30.5%). Type I lesions were seen in 4 affected dogs (16.7%) and 31 controls (15.5%), while Type II lesions were observed in 7 affected dogs (29.1%) and 42 controls (21.0%). Thus lesions of little apparent clinical significance (Tables 40) were seen in 11 affected dogs (45.8%) and 73 controls (36.5%). Type III lesions occurred in 5 affected dogs (20.8%) and 43 controls (21.5%), while Type IV lesions were seen in 4 affected dogs (16.7%) and 23 controls (11.5%). Thus lesions of apparent clinical significance occurred in 9 affected dogs (37.5%) and 66 controls (33.0%). Lesions in the left atrioventricular valve of cases of urinary disease have a greater frequency and somewhat greater severity than in the control group.

In the septal cusp of the right atrioventricular valve (Tables 39)

normal septal cusps occurred in 13 affected dogs (54.2%) and 95 controls (47.5%). Type I lesions were observed in 1 affected dog (4.2%) and 27 controls (13.5%), while Type II lesions were evident in 5 affected dogs (20.8%) and 46 controls (23.0%). Thus lesions of little apparent clinical significance (Types I and II) were seen in 6 affected dogs (25.0%) and 73 controls (36.5%). Type III lesions were seen in 2 affected dogs (8.3%) and 20 controls (10.0%), while Type IV lesions were seen in 3 affected dogs (12.5%) and 12 controls (6.0%). Thus, lesions of apparent clinical significance (Tables 40) were evident in 5 affected dogs (20.8%) and 32 controls (16.0%), While the frequency of chronic valvular disease is less in the septal cusp, there is a somewhat higher proportion of severe lesions in cases of urinary disease than in the control group.

In the mural cusp of the right atrioventricular valve (Tables 39), normal mural cusps were seen in 21 affected dogs (87.5%) and 177 controls (88.5%). Type I lesions were seen in 3 affected dogs (12.5%) and 20 controls (10.0%) while Type II lesions were seen in 3 controls (1.5%) only. Thus lesions of little apparent clinical significance (Tables 40) were observed in 3 affected dogs (12.5%) and 23 controls (11.5%). There is little to chose between these two groups with regard to lesions in this valve.

In the aortic valve (Tables 39) normal valves were seen in 22 affected dogs (91.7%) and 186 controls (93.0%). Type I lesions were confined to 2 controls (1.0%), while Type II lesions were seen in 1 affected dog (4.1%) and 5 controls (2.5%). Thus lesions of little apparent clinical significance (Tables 40) occurred in 1 affected dog (4.1%) and 7 controls (3.5%). Type III lesions were confined to 5 controls (2.5%) and Type IV lesions were seen in 1 affected dog (4.1%) and 2 controls (1.0%). Therefore, lesions of apparent clinical significance occurred in 1 affected dog (4.1%) and 7 controls (3.5%). There is little difference in the frequency or severity

of valve lesions in the aortic valve of these two groups.

To summarise, chronic valvular disease in cases of urinary disease have a greater frequency and severity in the left atrioventricular valve and a greater severity in the septal cusp of the right atrioventricular valve than in the control group.

The Systemic Diseases

This group contains 17 dogs, twelve males (70.6%) and 5 females (29.4%), with a mean age of 11 months. The mean age is well below the control group and the sex distribution is biased towards the male.

The causes of death or destruction of these dogs were two generalised virus diseases which affect a range of body systems, namely, 7 cases of hepatitis contagiosa canis (Rubarth's disease) and 10 cases of canine distemper.

In the left atrioventricular valve (Tables 39), normal valves were seen in 13 affected dogs (76.5%) and 61 controls (30.5%). Type I lesions were seen in 1 affected dog (5.9%) and 31 controls (15.5%), while Type II lesions occurred in 2 affected dogs (11.7%) and 42 controls (21.0%). Thus lesions of little apparent clinical significance (Types I and II) occurred in 3 affected dogs (17.6%) and 73 controls (36.5%). Type III lesions occurred in 1 affected dog (5.9%) and 43 controls (21.5%), while Type IV lesions were confined to 23 controls (11.5%). Therefore, lesions of apparent clinical significance (Tables 40) were seen in 1 affected dog (5.9%) and in 66 controls (33.0%). It is clear that both the frequency and severity of chronic valvular disease is greatly reduced in these cases of virus disease.

In the septal cusp of the right atrioventricular valve (Tables 39) normal septal cusps were seen in 12 affected dogs (70.6%) and 95 controls (47.5%). Type I lesions were seen in 2 affected dogs (11.7%) and 27 controls (13.5%), while Type II lesions were observed in a further 2 affected dogs (11.7%) and 46 controls (23.0%). Thus lesions of little apparent clinical significance (Tables 40) occurred in 4 affected dogs (23.4%)

and 73 controls (36.5%). Type III lesions were observed only in 20 controls (10.0%), while Type IV lesions occurred in 1 affected dog (5.9%) and 12 controls (6.0%). Therefore lesions of apparent clinical significance (Types III and IV) occurred in 1 affected dog (5.9%) and 32 controls (16.0%). Chronic valvular disease has a reduced frequency and severity in the septal cusp of cases of virus disease when compared with the control group.

No gross lesions were observed in this group in the mural cusp of the right atrioventricular valve, the aortic valve or the pulmonary valve.

To summarise, the frequency and severity of chronic valvular disease in cases of virus disease is greatly reduced in comparison to the control group.

The miscellaneous group is not considered in this section.

Conclusion on the relationship of chronic valvular disease to other intercurrent conditions

It is clear that disease in some body systems is associated with higher levels of frequency and severity of chronic valvular disease while the converse applies in other body systems. Consideration of the nature of the diseases in these body systems is illuminating, for example, in the endocrine system the predominant condition was diabetes mellitis, a disease known to be associated with middle-aged or elderly female dogs (which is also largely responsible for the imbalance of the sexes in this group). Thyroid carcinoma is characteristically associated with old dogs. Hence the mean age of the endocrine group was 9 years, 9 months (Table 41).

In the group of dogs which died of Hepatitis contagiosa canis and canine distemper, the mean age was under one year. Both of these diseases occur within the first two years of life in the vast majority of cases. The frequency and severity of chronic valvular disease in this group is very low (Table 41).

In the group of dogs with disease of the Reproductive system, nearly

half the members of this group had an adenocarcinoma of the mammary gland, a condition associated with the elderly bitch. In the group of Respiratory diseases, both Nocardiosis and broncho-pneumonia are more prevalent in dogs under six years and again, the occurrence and severity of chronic valvular disease is low.

Thus it will be seen that in all these systems the nature of the predominant disease condition and its typical age incidence influences the mean age of these groups. Thus, if a table is constructed (Table 41) based on the mean age of the dogs in each group, we see that the frequency and severity of chronic valvular disease declines progressively as the dogs become younger. There are obviously inconsistencies which are probably associated with the small sample size of these groups (more particularly in the case of the septal cusp of the right atrioventricular valve) but the trend is clear. Diseases of the cardiovascular system are a special case where the predominant condition was congestive heart failure due to chronic valvular disease. This fact must obviously influence the results.

Thus it is concluded that there is no direct relationship between chronic valvular disease and any disease or group of diseases in any particular body system. However, there does appear to be an indirect relationship, related to the age incidence of the predominant disease in a particular system, examples of which have been given above.

The histopathology of chronic valvular disease

The normal histology of the heart valves of the dog is described on pages 68-76. Each valve is composed of four basic layers, atrialis, spongiosa, fibrosa and ventricularis; these layers are similarly disposed in all the atrioventricular valves but are different in the semi-lunar valves. These differences will be referred to later.

It would appear that chronic valvular disease is an age-related condition and thus one must attempt to differentiate the lesions of chronic valvular disease from the changes associated with the normal ageing process. In order to differentiate between these two, the principle has been adopted that the normal ageing changes are represented by those valves showing the least changes at any given age. A study of the normal ageing changes was undertaken, using the ventral cusp of the left atrioventricular valve as a model. This valve was employed because it was the largest and probably the most labile of the atrioventricular valves. The valve was labile in the sense that it was more subject to changes than the other valves and its larger size made it possible to identify these changes more readily.

The normal ageing changes of the atrioventricular valves

The ages of the dogs in this series ranged from one day to fourteen years.

At one day old, the individual layers of the valve cannot be appreciated readily. The atrialis was most easily identified at the base of the cusp but it was ill-defined in the distal two-thirds. Similarly, the fibrosa and ventricularis could only be appreciated in the vicinity of the annulus. The bulk of the valve at this age was composed of a loose connective tissue, rich in cells, which was represented by the spongiosa in older dogs (Fig 39).

At ten weeks old, the atrialis extended into the area of contact and consisted of a few layers of delicate elastic fibres and some reticulin fibres. Similarly, the fibrosa and ventricularis extended throughout the

valve terminating some little distance short of the valve tip. The spongiosa was still abundant, particularly in the distal third of the cusp (Fig 40).

At twenty weeks old, the atrialis was more clearly defined and was composed of many layers of dense elastic fibres in proximal third of the valve cusp (Fig 41) and in the line of contact (Fig 42). Many young collagen fibres were seen within the atrialis and many adult fat cells were seen in the vicinity of the atrial myocardial prolongation. The fibrosa was now a dense sheet of collagen fibres which were directly continuous with those of the core of the chordae tendineae. The spongiosa was most prominent in the triangular areas opposite the origins of the chordae tendineae where numerous delicate elastic fibres ran in all directions. The spongiosa was still abundant at the valve tip but the cellularity was less pronounced. Delicate elastic fibres could be appreciated along the length of the ventricularis in appropriately stained sections.

By the end of the first year, further proliferation, particularly of collagen fibres, was evident in the distal third of the atrialis, in the area of contact (Fig 43). The fibrosa was now a robust structure and the ventricularis could not be differentiated from the fibrosa due to the intimate associationship between the two. In the spongiosa, neutral fat aggregates and further elastic fibres were evident in the triangular areas opposite the origins of the chordae tendineae (Fig 44).

At two years, the changes were confined to the atrialis, in the area of contact. In this situation there had been continued proliferation of collagen fibres with little or no further multiplication of elastic fibres so that the atrialis in this area was thickened to form a fibro-elastic plaque (Fig 45). A continued accumulation of fat in the spongiosa was also evident in some cases.

In dogs over two years old, the ageing changes were confined to the atrialis in the area of contact. Continued proliferation and hyalinisation of collagen gave rise to extensive fibrous plaques in which elastic fibres were generally confined to the base of the plaque and marked the boundary

between the atrialis and the spongiosa (Fig 46).

The extent to which these plaques developed varied in different individuals at the same age. These plaques were more prominent in the ventral cusp of the left atrioventricular valve than in any other cusp. These plaques did not seem to cause any gross distortion of the valve cusp, although they must have caused some 'stiffening' of the valve cusp in the area of contact. In the main, the vascular endothelium remained unaltered in the area overlying these plaques, although localised areas of proliferation were observed occasionally. No fibrin deposition was seen. One case in a ten-year old female had developed Lambl's excescences over a hyaline plaque.

The Lesions of Chronic Valvular Disease

The previous section has dealt with the normal ageing changes that are seen in the heart valves. These ageing changes are often obscured by the degenerative lesions of chronic valvular disease.

The description of the histopathology of chronic valvular disease uses the ventral cusp of the left atrioventricular valve as a model. The appearance of the lesions in the other valves are compared and contrasted with that seen in the ventral cusp.

The ventral cusp of the left atrioventricular valve

The earliest change detectable by light microscopy was seen in the sub-endothelium of the distal third of the ventricularis (Fig. 47). In such a lesion there was a localised oedema which dissected and separated the elastic and collagen fibres of the ventricularis. These fibrillar elements became fragmented and disorientated, lost their affinity for eosin and ultimately disappeared. The oedema, coupled with the loss of fibrillar elements, made these lesions pale-staining and facilitated their detection at an early stage (Fig. 47).

These early lesions may occur at any point along the length of the ventricularis but such lesions occurred most regularly at the junction between the valve cusp and the chordae tendineae (Fig. 48) where the reaction resulted in swelling of the proximal portion of the chordae and degeneration of their collagenous core. These degenerative lesions were seen in the distal ventricularis (and chordae tendineae) in 54% of cases, in the proximal ventricularis in 2.0% of cases and extended throughout the ventricularis in a further 8.0% (Table 42). When this lesion was extensive, it gave the valve a 'lace-like' border (Fig. 49).

As these lesions developed at the junction between the chordae and the valve, the resultant swelling tended to incorporate the proximal chordae into the valve cusp (Fig. 48). This incorporation became more pronounced as the lesion progressed.

These early lesions were even detected in the left atrioventricular valves of dogs under six months old.

Although these lesions appeared to originate in the ventricularis, they soon extended to involve the subjacent fibrosa with which the ventricularis is intimately associated and where a similar dissection, degeneration and fragmentation of collagen fibres took place. The normal fibrillar components of the fibrosa and ventricularis were replaced by a very delicate

reticulin network which supported the cellular components of the lesion. The predominant cell was an immature fibroblast which was regularly observed in association with lesser numbers of undifferentiated mesenchymal cells, lymphocytes, plasma cells and occasional polymorphs (Figs. 50).

As the reticulin network developed, certain tinctorial changes took place. Sections stained by Toluidine Blue showed a β metachromasia which was confined to the surfaces of the reticulin fibres in some cases but was more regularly seen extending throughout the abundant ground substance (Fig. 51). The ground substance also stained with Azure A and Alcian Blue, was PAS-negative (Fig.52). Sections stained by Sudan Black B failed to reveal any fat within these lesions and no cholesterol deposits were seen. Calcium deposition was only observed in one 7 year old dog.

These lesions extended throughout the fibrosa progressively destroying the normal tissues and replacing them with a loose myxomatous tissue. There is a similar progressive destruction of collagen in the proximal chordae tendineae in 73% of cases (Fig. 53). The chordae become uniformly thickened or 'beaded' with the destruction of their collagenous core.

The destruction of the collagen of the fibrosa, ventricularis and chordae tendineae, coupled with the replacement of this supporting tissue by a loose myxomatous tissue inevitably weakened the valve cusp.

But these degenerative changes were not confined to the septal areas of the valve. A similar degenerative process was seen in 62.0% of cases, involving the spongiosa. The earliest changes were seen in the triangular areas of spongiosa opposite to the origins of the chordae tendineae (Fig. 54). The earliest lesions could be most readily detected in sections stained by Toluidine blue (Fig.55). These small degenerative foci increased in size and ultimately coalesced to give rise to extensive areas of degeneration similar in appearance to those seen in the fibrosa and ventricularis (Fig. 54).

In the normal spongiosa, this loose meshwork of collagen and elastic fibres also contained aggregates of neutral fat which were destroyed as the lesion developed. The lesion was similar to that described above, a delicate reticulin network supporting numerous immature fibroblasts with an abundant metachromatic ground substance which caused the valve to swell. In 12.0% of cases, haemorrhage was seen into the spongiosa, particularly beneath the area of contact. This haemorrhage was usually recent with accumulations of intact red blood cells (11.0%) but four cases were seen where aggregates of haemosiderin marked a previous haemorrhage (Fig. 56).

Again, a similar degenerative lesion to that described above was seen in the distal third of the atrialis (16.0% of cases). Such changes involved the fibrous atrial plaque described in a previous section (p.159) and completely destroyed the fibrillar elements replacing others with the same loose myxomatous tissue. In advanced cases, it was difficult to define the lower limit of the atrialis. Sometimes these lesions were extremely localised, throwing up nodular excrescences in the area of contact (Fig. 57).

While lesions in the atrialis and spongiosa do not significantly weaken the valve, as do similar lesions in the fibrosa and chordae tendineae, they do result in distortion and swelling of the valve. These swellings occurred initially at points opposite to the origins of the chordae tendineae and resulted in nodular swellings on the outflow surface opposite to the origins of the chordae tendineae (Fig. 54). Later, as these lesions tended to coalesce with neighbouring lesions through the intervening spongiosa and valve tip, diffuse plaque-like deformities developed in the distal third of the valve (Fig. 24).

Thus we see that all four basic layers of the valve are involved - lesions in the spongiosa and, to some extent, the atrialis cause distortion, nodularity and plaque-like deformities on the outflow surface in the area

of contact. These may give rise to valvular insufficiency, particularly when the dorsal cusp is similarly affected.

Similar lesions may be seen in the fibrosa and ventricularis and, while these may contribute to the swelling of the cusp, the loss of supporting collagen is more important. The chordae tendineae are also involved; lesions in the proximal chordae are incorporated into the inflow surface of the valve, thereby shortening the chordae and adding to the incompetence of the valve. The lesions in the chordae may extend as far as the related papillary muscle and the strength of the chordae may be reduced to a point where they rupture or stretch.

The principal effect of lesions in the fibrosa, the ventricularis, to some extent, and the related chordae tendineae, is to reduce the ability of these structures to resist the stress of systole causing the valve and the chordae to stretch and 'balloon' during systole, resulting in severe incompetence.

In these ways, the valve function is destroyed and the incompetence develops. Such changes are more pronounced where the dorsal cusp is involved, as it almost invariably is. Swelling and distortion of the valve cusp and/or 'ballooning' are responsible for valvular incompetence.

The dorsal cusp of the left atrioventricular valve

The basic nature of the degenerative lesion seen in the dorsal cusp was similar to that already described in the ventral cusp. There was, however, some variation in the distribution of these lesions within the cusp itself.

The initial lesion was most regularly observed (62.0%) in the distal third of the ventricularis in the ventral cusp but only 17.5% of dorsal cusps showed lesions in this situation. In the proximal two-thirds of the ventricularis the dorsal cusp showed lesions in 34.0% of cases compared with 10.0% in the ventral cusp. In the dorsal cusp, 30.0% of these lesions in the proximal two-thirds of the ventricularis affected the base of the valve and extended across the sinus pocket and a few millimetres into the mural endocardium (Fig 58).

In association with the higher frequency of lesions in the proximal ventricularis, 12.0% of proximal chordae tendineae showed lesions and 43.5% showed lesions in distal chordae tendineae; in all, 55.5% of cases showed lesions in the chordae tendineae, lesions which had originated at the junction between the chordae and the ventricular face of the cusp (Fig 53). In the ventral cusp, 73.0% of cases showed lesions in the chordae tendineae.

Degenerative changes were confined to the distal half of the spongiosa in both cusps, these lesions were observed in 62.0% of ventral cusps and 58.5% of dorsal cusps. The valve tip was affected in 68.0% of ventral cusps and 45.0% of dorsal cusps. As the spongiosa and valve tip are directly continuous with each other, it was reasonable to suppose that the frequency of lesions in the valve tip would be equal (within limits) in both the dorsal and ventral cusp, particularly as the frequency of lesions in the spongiosa was so close. The reasons for the discrepancy are not known.

Neutral fat was less common in the dorsal cusp (22.5%) than in the

ventral cusp (62.0%). Furthermore, the bulk of this fat was confined to the vicinity of the atrial myocardial prolongation.

In the atrialis, the normal fibro-elastic plaque was more readily observed in the ventral than in the dorsal cusp - plaques were present in the dorsal cusp of all older dogs, but they were less prominent. These fibro-elastic plaques in the dorsal cusp were subject to a similar frequency of degenerative changes as those observed in the ventral cusp (Fig 57).

To summarise, there were two basic differences in the distribution of the degenerative lesion in the dorsal cusp when compared with the ventral cusp. Firstly, there was a far greater frequency of lesions in the proximal two thirds of the ventricularis in the dorsal cusp compared with the ventral cusp (34.0%:10.0%). Secondly, there was a greater frequency of lesions in the valve tip of the ventral cusp compared with the dorsal cusp (68.0%:45.0%). In all other respects, the frequency and distribution of these degenerative changes was similar in both valves.

The right atrioventricular valve

The right atrioventricular valve consists of two cusps, each of which will be considered separately.

The septal cusp

The basic pathological process is similar to that seen in the left atrioventricular valve but differs in the distribution of these lesions within the cusp.

The initial lesion was seen in the proximal two thirds of the ventricularis in 10.0% of ventral cusps and 34.0% of dorsal cusps in the left atrioventricular valve and in 76.0% of septal cusps in the right atrioventricular valve (Fig 59). The distal third of the ventricularis showed lesions in 62.0% of ventral cusps and 17.5% of dorsal cusps in the left atrioventricular valves and in 15.5% of septal cusps. The lesions in the proximal two thirds of the ventricularis of the septal cusp extended around the sinus pocket to include a few millimetres of the mural endocardium (Fig 59).

The proximal chordae tendineae showed degenerative lesions in 77.5% of septal cusps (Fig 59) while only 12.0% of dorsal cusps of the left atrioventricular valve showed lesions. The distal chordae tendineae of 73.0% of ventral cusps and 43.5% of dorsal cusps in the left atrioventricular valves showed lesions compared with 9.0% in the septal cusps. All these lesions in the chordae tendineae originated at the junction between the chordae and the ventricular face of the cusps (Fig 48).

Similar degenerative lesions were seen in the distal spongiosa of 62.0% of ventral cusps and 58.5% of dorsal cusps in the left atrioventricular valve and in 18.0% of septal cusps in the right atrioventricular valve. No lesions were seen in the proximal spongiosa of the left atrioventricular valve whereas 46.0% of septal cusps showed lesions in this situation (Fig 59). It seemed that lesions in the spongiosa of the septal cusp originated near,

or at the base of the cusp and extended distally from that point (Fig 59). In 8.5% of cases degenerative lesions were seen in the valve ring (Fig 60). In the valve tip 68.0% of ventral cusps and 45.0% of dorsal cusps in the left atrioventricular valve showed lesions compared with 22.5% of septal cusps (Fig 61). In the septal cusp, 12.0% of these lesions were mild.

Neutral fat when it occurred in the septal cusp, was confined to the proximal cusp and particularly to the vicinity of the atrial myocardial prolongation.

Fibro-elastic plaques were less well defined in the septal cusp than in the ventral cusp of the left atrioventricular valve but, nevertheless, they were seen in 33.5% of septal cusps and a further 48.5% showed plaques with secondary degenerative lesions (Fig 62).

The most striking feature in the distribution of these degenerative lesions was the frequency of lesions in the proximal spongiosa, ventricularis and chordae tendineae when compared with the left atrioventricular valve where lesions occurred principally in the distal portion of the cusp.

The mural cusp

Lesions were far less common and severe in the mural cusp than in the septal cusp of the right atrioventricular valve.

Degenerative lesions were seen in the proximal two-thirds of the ventricularis in 76.0% of septal cusps and 3.0% of mural cusps. The distal third of the ventricularis showed lesions in 15.5% of septal cusps and 10.0% of mural cusps, 2.0% of these latter cases showed more pronounced lesions (Fig 63). The overall frequency and severity of these lesions was far lower in the mural cusp.

Similarly, 47.5% of septal cusps showed lesions in the proximal chordae tendineae while only 9.0% of these cusps showed lesions in the distal chordae tendineae. In the mural cusp lesions were confined to 20.0% of distal chordae tendineae (Fig. 63).

In the proximal half of the spongiosa, 46.0% of septal cusps and only 1.0% of mural cusps showed lesions. In the distal half of the spongiosa 18.0% of septal cusps and 40.5% of mural cusps showed lesions (32.5% of these were very mild lesions). In the valve tip 22.5% of septal cusps and 39.0% of mural cusps also showed lesions, 24.0% of the latter were mild lesions (Fig 63).

In the mural cusp, neutral fat was uncommon, being seen in 4.0% of cases only.

Fibro-elastic plaques were seen in 26.5% of mural cusps and mild secondary degenerative lesions in a further 12.0% (Fig 63). In the septal cusp, such lesions were seen in 33.5% and 48.5% respectively.

It is clear that degenerative lesions have a far greater frequency and severity in the septal cusp than in the mural cusp.

The semi-lunar valves

There are fundamental terminological and structural differences between the semi-lunar and atrioventricular valves. Because of these differences, the distribution of the four basic layers is altered so that the atrialis of the atrioventricular valve becomes the ventricularis of the semi-lunar valve. The spongiosa and fibrosa retain the same relationship and the ventricularis of the atrioventricular valve becomes the arterialis of the semi-lunar valves. There are no chordae tendineae in the semi-lunar valves.

Ageing changes in these valves are most readily observed in the aortic valve where they are confined to the ventricularis. In the ventricularis, a localised fibro-elastic proliferation develops along the line of closure (lunulae). These changes are most obvious in the middle of the lunulae, where there is a pronounced fibro-elastic proliferation in the nodulae valvularum aortae (Fig.64). Similar, but less obvious changes, are seen in the pulmonary valve.

The aortic valve

In the normal semi-lunar valve, beneath the fibrosa of the valve cusp, there is a triangular wedge of loose connective tissue, the ring spongiosa (sub-valvular spongiosa) which is directly continuous with the spongiosa of the valve cusp. The endocardium of the ventricle is directly continuous with the ventricularis of the valve cusps as it lies over the ring spongiosa (Fig. 65).

The ring spongiosa was particularly susceptible to the same degenerative lesions; 84.0% of cases showed lesions at this site (Fig.66) and in 1.0% of cases, this lesion extended into the proximal spongiosa of the valve cusp (Fig. 66). Lesions were detected in the distal spongiosa, in the vicinity of the line of contact of 15.0% of cases and a further 6.0% showed lesions in the free border of the cusp (Fig. 67).

The ventricularis showed the degenerative lesion in 12.0% of cases at

the base of the cusp and overlying the ring spongiosa (Fig. 66). Secondary degenerative changes were seen in the fibro-elastic proliferations of the lunulae and nodulae valvularum aortae in 17.0% of cases. The latter lesions were very mild in 12.0% of these (Fig. 67).

The arterialis could not be differentiated from the fibrosa in the majority of cases, so these two layers were considered to be one. Mild degenerative lesions were seen proximally in 6.0% (Fig. 66). Distally, 1.0% of valves showed this characteristic lesion.

To summarise, the most striking and consistent change seen in the aortic valve was located in the ring spongiosa. In the valve cusp, the principle changes were seen in the distal spongiosa and the ventricularis, around the line of contact.

The Pulmonary Valve

The pulmonary valve is similar in structure to the aortic valve but it has a more delicate structure (Fig. 68).

As with the aortic valve, degenerative lesions were most frequently observed in the ring spongiosa, where 43.0% of valves showed lesions and in 3.0% of these the changes extended into the proximal spongiosa of the valve cusp (Fig.69). A further 7.0% of cases showed mild degenerative lesions in the distal spongiosa, in the vicinity of the line of contact (Fig. 70). Only 2.0% of cases showed lesions in the free border of the cusp (Fig. 70).

In the proximal ventricularis 14.5% of cases showed mild degenerative lesions over the ring spongiosa and extending into the base of the cusp in some cases (Fig.69). The distal ventricularis, in the vicinity of the line of contact, showed mild lesions in 10.0% of cases (Fig.70).

The arterialis and fibrosa showed changes proximally in 8.0% of cases and in the distal half in 4.0% of cases (Fig. 69).

Lesions in the pulmonary valve are generally less frequent and severe in the pulmonary valve than in the aortic valve.

In comparison with the atrioventricular valves, lesions are far less frequent and severe in the semi-lunar valves. Undoubtedly the most striking and consistent lesion was seen in the ring spongiosa of both valves.

Lesions, visibly by the naked eye, were confined to the aortic valve and could be detected histologically in the distal spongiosa and ventricularis in the vicinity of the line of contact (Fig.67).

The histo-chemical nature of the valve lesion

It was apparent that a similar lesion was seen in all the heart valves and that this lesion was characterised by a degeneration of the fibrillar elements of the valve and their replacement by an abundant, structureless, ground substance, containing a delicate reticulin network (Fig 71).

The ground substance gave a negative reaction to the Periodic acid Schiff method which limited the possible alternatives to two. Either this material was predominantly a simple (acid) mucopolysaccharide or a complex (sulphated) mucopolysaccharide.

This material stained positively with Hale's colloidal iron (Fig 72) and Alcian Blue (Fig 73) and gave a violet (Betametachromasia with Toluidine Blue and Azur A (Fig 54). There was a positive reaction with Gomori's aldehyde fuchsin and a negative reaction with Sudan Black and Herxhaimer Scarlet R.

Methylation and saponification resulted in a negative reaction with Alcian Blue. This combined Alcian Blue/P.A.S. technique gave a positive reaction. A combined Alcian Blue/Aldehyde fuchsin method gave a blue reaction within the valve substance (Fig 73). It was concluded that this material was an acid mucopolysaccharide.

The Changes in the Blood Vessels

To facilitate a description of the nature, frequency and distribution of the pathological changes in the blood vessels, a grading system has been devised. The different manifestations of the arteriosclerotic process have been classified into two broad groups and seven different grades as follows:

Group I (Grades A to D)

Group I changes were seen in the posterior aorta, the common iliac arteries, the renal arteries and left and right coronary arteries. These Group I changes were characterised by the development of a fibrous intimal plaque. In association with the intimal changes, clefts containing a metachromatic material were seen in the inner third of the media of the posterior aorta.

Grade A Changes

These changes were the earliest that were detected, characterised by a localised intimal oedema and degeneration or fraying, splitting or re-duplication of the internal elastic lamina. (Such changes were often difficult to observe in the posterior aorta and thus are not recorded in this situation.)

Grade B Changes

In these lesions the intimal oedema was more pronounced and the vascular endothelium was elevated to form a plaque. The internal elastic lamina showed degenerative changes and fibrocytes and smooth muscle cells were seen passing through defects in the internal elastic lamina and lying within the plaque itself. There was little or no fibrillar material within the intimal lesion.

Grade C Changes

The lesion remained oedematous to some extent but fibrillar elements were now evident, notably collagen fibres, but some elastic tissue was also evident in some vessels. The fibrillar elements appeared to develop more readily in the deeper layers of the plaque while the more superficial layers were oedematous.

Grade D Changes

These lesions were almost wholly fibrous and usually multiple inasmuch as several plaques appeared in one transverse section of a vessel. A variable degree of diffuse intimal hyperplasia was seen in the areas between neighbouring fibrous plaques.

Where plaques of different grades were seen in the same vessel, the case was classified according to the most advanced grade that was observed.

Group II (Grades E - F)

Group II changes were confined to the intramural coronary vessels of the inner third of the myocardium of the left ventricle. These vessels were infiltrated by a PAS - positive hyalin material which replaced the normal components of the vessel wall. These lesions were grades as follows:

Grade E Changes

Limited segmental or circumferential infiltration of the intima and the inner third of the media by hyalin material.

Grade F Changes

More advanced segmental or circumferential infiltration of the vessel wall without a significant narrowing of the lumen of the vessel.

Grade G Changes

Extensive infiltration of the vessel wall with stenosis or complete occlusion of the vessel wall. Such lesions were often associated with ischaemic changes in the myocardium.

The Group I and Group II changes described above form the basis of the description of lesions in particular vessels that follows.

The Posterior Aorta

Lesions in the posterior aorta and common iliac arteries were the only ones that could be detected macroscopically.

The Gross Lesions

These lesions were detected in 79 cases (39.5%) as greyish-white

smooth or irregular plaques or streaks and areas of diffuse intimal thickening. These lesions were slightly elevated above the surrounding normal intima. The earliest lesions appeared as smooth rounded elevations, a few millimetres in diameter, situated in close proximity to the inlets to the branches of the aorta (Fig 74).

In the aortic arch a diffuse 'roughening' and wrinkling was regularly observed in aged dogs on the cranial or concave surface of the aortic arch (Fig 75).

In the thoracic portion of the aorta, the lesions were always mild, compared with those in the abdominal portion. These lesions consisted of small, smooth plaques up to 5 mm. in diameter, situated on the caudal or 'downsteam' side of the inlets to the intercostal vessels (Fig 74).

In the abdominal aorta, the lesions were often more pronounced, particularly as one approached the iliac bifurcation. Early lesions were associated with the inlets to branches of the posterior aorta, notably the renal and common iliac vessels. In more advanced cases, however, these lesions were more extensive and diffuse, particularly over the dorsal (posterior) face of the aortic intima (Fig 77).

A total of 200 aortae were examined, of which 50 were subjected to gross staining by Sudan IV (Holman et al 1958). Only in one case, a 10 yo. Labrador male, a limited fatty streaking was observed (Fig 76). The remainder were negative for fat, although arteriosclerotic plaques were present in some.

The frequency and distribution of these aortic lesions are recorded in a series of tables, following a gross and histological examination of these vessels. The vessels are classified in accordance with the grading scheme described above. The details of the histopathological examination will be described below (page 192), but the early changes in the internal elastic lamina could not be readily appreciated in the aorta due to the

nature of its normal structure. Therefore, Grade A changes are not described or recorded for the aorta.

Age distribution

In dogs under five years (Table 43), Grade B lesions only were seen in 5 dogs (7.0%). In the age group five to eight years inclusive, 10 dogs (25.6%) had Grade B lesions, 4 dogs (10.2%) had Grade C lesions and 1 dog (2.5%) had a Grade D lesion. Thus, in dogs under nine years, 15 (13.0%) had Grade B lesions, 4 (3.5%) had Grade C lesions and 1 (0.8%) had a Grade D lesion (Table 43).

In the age group nine to twelve years inclusive (Table 43), 25 dogs (35.7%) had Grade B lesions, 10 dogs (14.3%) had a Grade C lesion and 16 (22.9%) had a Grade D lesion. In dogs thirteen years and over, Grade C lesions only were seen in 8 dogs(53.3%). Thus, in dogs nine years and over, 25 dogs (29.4%) had Grade B lesions, 18 dogs (21.2%) had Grade C lesions and 16 (18.8%) had Grade D lesions (Table 43).

It is clear that aortic arteriosclerosis increases considerably in frequency and severity with advancing age. Grade B lesions are first seen in dogs at two years, Grade C lesions at five years and Grade D lesions at seven years (Table 44). Aortic arteriosclerosis may be seen in any dog over two years old.

The Sex Distribution

Grade B lesions were seen in 19 males (18.1%) and 21 females (22.1%). Grade C lesions were seen in 13 males (12.2%) and 9 females (9.5%), while Grade D lesions were seen in 11 males (10.5%) and 6 females (6.3%). In all, aortic arteriosclerosis was seen in 43 males (40.9%) and 36 females (37.9%). While there is a tendency towards a greater frequency and severity in the male, these differences in the two sexes are not considered to be significant.

Breed Susceptibility

In this series of 200 dogs only five breeds are represented by more than

ten individuals. The frequency and distribution of the various grades in these five breeds are compared with the other 110 dogs (Table 46).

In the Alsatians, 3 dogs (18.7%) had Grade B lesions only. In the Border Collies, 6 (19.3%) had Grade B lesions, 6 (19.3%) had Grade C lesions and 3 (9.7%) had Grade D lesions. The Boxers had 4 (36.4%) with Grade B lesions, 1 (9.1%) with a Grade C lesion and 3 (27.2%) with a Grade D lesion. Only 1 Greyhound (7.1%) had a Grade B lesion. In contrast, 2 Labradors (11.1%) had Grade B lesions, 1 Labrador (5.6%)had a Grade C lesion and 3 Labradors (16.6%) had a Grade D lesion.

In the control group 24 dogs (21.8%) had Grade B lesions, 14 dogs (12.7%) had Grade C lesions and 8 (7.3%) had Grade D lesions. If age is a factor in the frequency of aortic arteriosclerosis, the mean age of these five breeds must be taken into account in any consideration of breed susceptibility. The overall frequency in the control group is 41.8% with a mean age of 6 years 5 months (Table 46). The Alsatian has a frequency of 18.7% (Grade B only) with a mean age of 6 years 5 months. Thus it is clear that the age factor is not important here but the sample size (16 dogs) is small and may not be representative.

In the Border Collie, the breed frequency was 48.4% with a higher frequency of Grade C lesions in particular. Furthermore, the mean age of the Border Collies was 8 years 6 months; considerably higher than that of the control group. The age of these dogs may be a more important factor than the small sample size shown in the higher frequency in this breed.

The Boxers have 72.8% with aortic arteriosclerosis due to a greatly increased frequency of Grade B and Grade D lesions. There is little variation in the mean age of the Boxers compared with the control group (Table 35), but the small sample size (11) must be significant here.

In the Greyhound 1 (7.1%) showed a Grade B lesion only, the low mean age of the Greyhounds (1 year 9 months) must influence the frequency in this breed.

The Labradors have an overall frequency of 33.4%, largely due to a high incidence of Grade D lesions, the mean age is comparable to that of the control group.

There is no evidence of any breed susceptibility to aortic arteriosclerosis any variations from the control group can be attributed to small sample size or variations in the mean age of a particular breed.

Extramural Coronary Arteries

Despite the fact that multiple transverse sections were taken across the major extramural branches, no gross lesions were detected in these vessels. In three cases only, arteriosclerotic plaques were seen around the inlets to the left coronary artery (two cases) and the right coronary artery (one case).

In consequence, the detection of lesions depended upon the histological examination and vessels were then graded according to the scheme described on page 176.

The left coronary artery

Lesions were detected at all levels in the left coronary artery but principally in the extramural divisions. The changes in intramural branches will be described below (page 187). While the majority of arteriosclerotic lesions occurred in the left circumflex coronary artery, 11 cases (5.5%) showed changes in the descending branches. In seven of these cases, these lesions were associated with arteriosclerotic lesions in the left circumflex coronary, while in the remaining four cases lesions were only detected in the descending branches. Of these 3 (1.5%) were Grade A lesions and 1 (0.5%) was a Grade C lesion.

A routine block was taken from the first few millimentres of the left circumflex coronary and arteriosclerotic lesions were detected at this site in 64.0% of affected vessels.

In all, 54 dogs (27.0%) showed arteriosclerotic lesions in their extramural vessels. The internal elastic lamina could be clearly defined in the coronary vessels, so Grade A changes could be readily seen and are recorded.

Age distribution

In dogs under five years, 8 (10.5%) showed Grade A changes only (page 178). In dogs five to eight years inclusive, 9 (23.1%) showed Grade A changes,

3 (7.7%) showed Grade B lesions and 2 (5.1%) showed Grade C changes (Table 47). Therefore, dogs under nine years showed 17 (14.7%) with Grade A changes, 3 (2.7%) with Grade B changes and 2 (1.7%) with Grade C changes (Table 47).

In the age group nine to twelve years inclusive, 19 dogs (27.1%) showed Grade A changes, 6 dogs (8.6%) showed Grade B lesions and 1 dog (1.4%) showed a Grade C lesion. Dogs over twelve years showed 5 (33.3%) with Grade A lesions and 1 (6.6%) with a Grade C lesion. Therefore, dogs nine years and over had 24 (28.2%) with Grade A changes, 6(7.0%) with Grade B lesions and 2 (2.4%) with Grade C changes. No Grade D lesions were seen in any coronary artery (Table 47).

Grade A changes were seen in one dog under one year old in association with a congenital pulmonary stenosis. Dogs six years and over may show Grade B and Grade C changes (Table 44).

There is some evidence of an increase in frequency of arteriosclerosis in the left coronary artery with advancing age. The overall frequency in dogs under nine years was 19.9% compared with 37.6% in older dogs but there is no evidence of increase in the severity of lesions in dogs of advancing age. Coronary arteriosclerosis may be seen in dogs at any age.

Sex distribution

Grade A lesions were seen in 23 males (21.9%) and 18 females (18.8%). Grade B changes were seen in 7 males (6.6%) and 2 females (2.1%) while Grade C lesions were seen in 1 male (1.0%) and 3 females (3.1%). The overall frequency in the male was 29.5% compared with 24.2% for the female. There is no significant difference in frequency between the male and the female.

Breed susceptibility

In the Alsatian, 5 dogs (31.3%) had Grade A changes and 3 dogs (18.7%) had Grade B changes, an overall frequency of 50.0% compared with 17.3% for the control group, the mean age of the Alsatian being identical to

that of the control group. The small sample size of the Alsatians (16) may be responsible for the variation. (Table 49)

In the Border Collie, 12 dogs (38.7%) had Grade A changes, 1 dog (3.2%) had a Grade B lesion and 1 dog had a Grade C lesion, an overall frequency of 45.2% compared with 17.3% for the control group. The higher frequency in the Border Collie, particularly of Grade A lesions, may be due in part to the higher mean age (8 years 6 months) for this breed (Table 36).

The Boxers showed 2 (18.2%) with Grade A lesions, 2 (18.2%) with Grade B lesions and 1 (9.1%) with a Grade C lesion, an overall frequency of 45.5% compared with 17.3% for the control group. There is little difference in the mean age of the Boxers compared with the control group (Table 36) but the small sample size (11 Boxers) is probably the factor here which modifies the frequency.

The Greyhounds showed 3 (21.4%) with Grade A lesions only and a mean age of 1 year 9 months. The relative youth of the Greyhounds and the relatively small sample size, no doubt, influenced this result.

In the Labrador, 5 dogs (27.7%) showed Grade A lesions only, the mean age (6 years 8 months) was close of that of the control group (Table 36). Such variations as occurred were probably a consequence of the small sample size.

The higher frequency seen in the Alsatian and Boxer are probably a consequence of the small sample size of these breeds, while the higher frequency in the Border Collie may be due to variations in the mean age of that breed compared with the control group.

The Right Coronary Artery

Lesions in the right coronary artery were confined to the extramural branches. In a total of 17 cases (8.5%), 35.3% showed lesions in the first few millimetres of the vessel. Only 1 case (0.5%) showed lesions in descending branches (Grade A) and that in the absence of lesions in the main trunk of the vessel.

Age distribution

In dogs under five years old, 3 (3.9%) showed Grade A changes and 2 (2.6%) showed Grade B changes. In dogs five to eight years inclusive, 3 (7.6%) had Grade A lesions, 3 (7.6%) had Grade B changes, and 1 (2.6%) had a Grade C lesion (Table 50). Therefore, in dogs under nine years 6 (5.2%) had Grade A changes, 5 (4.4%) had Grade B changes and 1 (0.9%) had Grade C changes (Table 50).

In dogs nine to twelve years inclusive, 3 (4.3%) had Grade A changes and 1 (1.4%) had Grade C changes. Dogs over twelve years showed 1 (6.6%) with Grade B changes (Table 50). Therefore, dogs nine years and over showed 3 (3.5%) with Grade A changes, 1 (1.2%) with Grade B changes and 1 (1.2%) with Grade C changes. (Table 50). No Grade D lesions were seen in the right coronary artery.

Grade A lesions were seen in two-year old dogs, Grade B changes in two dogs under one year and Grade C lesions were not seen in dogs under five years.

There is little evidence, based upon these results, of an increase in the frequency or severity of arteriosclerosis of the right coronary artery with advancing age. Dogs under nine years have an overall frequency of 10.5% compared with 5.9% at nine years or over. The low frequency of lesions in the right coronary artery is a significant factor here. Arteriosclerosis of the right coronary artery may be seen at any age.

Sex Distribution

A total of 6 males (5.7%) and 3 females (3.2%) had Grade A lesions, 6 males (5.7%) only had Grade B lesions and 1 male (0.9%) and 1 female (1.0%) had Grade C lesions. The overall frequency in the male was 12.4% compared with 4.2% in the female. This disparity may be due to the low overall frequency of lesions in the right coronary artery. (Table 51)

Breed Susceptibility

In the Alsatian 3 (18.7%) showed Grade A changes and 1 (6.3%) showed Grade B changes, an overall frequency of 25.0% compared with 6.4% in the control group (Table 52). As both the Alsatian and the control group have an identical mean age, it is possible that this gross discrepancy may be related to the low overall frequency of lesions in this vessel.

In the Border Collie, 2 (6.5%) had Grade A lesions and 2 (6.5%) had Grade B lesions, an overall frequency of 12.9% compared with 6.4% in the control group. This discrepancy, while related to the higher mean age of the Border Collies (8 years), is also a consequence of the low overall frequency of lesions in this vessel.

In the Boxer, only 1 (9.1%) had a Grade B lesion. Here again, the low overall frequency of lesions and the low sample size in this breed may account for the discrepancies observed.

No lesions were observed in the right coronary arteries of the Greyhounds.

In the Labrador, 1 dog (5.5%) showed a Grade C lesion. Probably similar factors operated here to those mentioned in relation to the other breeds.

No clear evidence of any breed susceptibility could be detected here. The low frequency of lesions in the right coronary artery played a major part in the inconsistent results obtained.

The renal artery

No routine examination of the renal arteries was undertaken but lesions similar to those described above were seen in a number of instances.

The common iliac artery

Here again, while no routine examination of these vessels was undertaken, lesions were regularly observed in the common iliac arteries in those cases where severe arteriosclerotic lesions were present in the terminal (caudal) portion of the posterior aorta. Upon gross examination these vessels were distended, the intima being thrown up into a series of annular ridges. In all, 35 examples of severe arteriosclerosis of these vessels were observed (Fig 78).

The Intramyocardial Vessels

Two different lesions were seen in intramyocardial vessels:-

(a) A fibrous intimal plaque similar to that seen in the posterior aorta and extramyocardial coronary vessels.

(b) A hyalin degeneration of the intima and media of the coronary vessels that lie within the papillary muscles and innermost third of the myocardium of the left ventricle.

(a) The fibrous intimal plaque

These lesions were less frequent than similar lesions seen in the extramyocardial coronary vessels and they were confined to the left heart in this series.

Age distribution

Fibrous intimal plaques were seen in the vessels supplying the papillary muscles and apex of the left ventricle of 11 dogs (5.5%). This low overall frequency influenced all the results that have been obtained from these epidemiological studies.

Dogs under nine years showed 2 (1.7%) with Grade A lesions and 4 (3.5%) with Grade B changes. Dogs nine years and over showed 3 (3.5%) with Grade A lesions, 1 (1.2%) with Grade B lesions and 1 (1.2%) with a Grade C lesion. No Grade D lesions were observed (Table 53). This gave dogs under nine years an overall frequency of 5.2% compared with 5.9% in dogs nine years and over. There is no evidence of an increase in the frequency or severity of the fibrous intimal plaque.

In dogs under five years, only 3 (3.9%) showed Grade B lesions. In dogs five to eight years inclusive, 2 (5.1%) showed Grade A lesions and 1 (2.6%) showed a Grade B lesion(Table 53). Dogs nine to twelve years old inclusive, showed 2 (2.9%) with Grade A lesions, 1 (1.4%) with a Grade B lesion and 1 (1.4%) showed a Grade C lesion. Dogs thirteen years and over showed only 1 (6.6%) with a Grade A lesion. There is no evidence

of an increase in the frequency or severity of these lesions in this study. Grade A lesions were not seen in dogs under six years, Grade B lesions were first seen in two-year old dogs and Grade C lesions in one twelve-year old dog.

Similarly, with the small number of cases involved, no reliable sex distribution or breed predisposition could be determined for these lesions.

The essential histopathological features of these lesions are similar to those seen in the posterior aorta and extramural coronary arteries.

(b) Hyalin change in intramyocardial coronary vessels

In this study these lesions were confined to vessels located in the papillary muscles and innermost third of the left ventricular myocardium. The nature of these lesions differs from those already described, so the grading system has been extended to include these lesions (page 177).

Age distribution

The overall frequency of hyalin degeneration was 29.5%, a total of 59 cases were observed.

Dogs under nine years showed 11 (9.5%) with Grade E lesions, 3 (2.6%) with Grade F lesions and 1 (0.9%) with Grade G lesions. In dogs nine years and over 20 (23.5%) had Grade E lesions, 15 (17.6%) had Grade F lesions and 9 (10.6%) had Grade G lesions. The frequency and severity of hyalin degeneration increases with age (Table 56).

Dogs five years and under had 3 (4.0%) with Grade E lesions and 1 (1.3%) with a Grade F lesion. In dogs five to eight years inclusive, 8 (20.5%) showed Grade E lesions, 2 (5.1%) showed Grade F lesions and 1 (2.6%) showed a Grade G lesion. In the age group nine to twelve years inclusive, 16 (22.8%) showed Grade E lesions, 13 (18.6%) showed Grade F lesions and 7 (10.0%) showed Grade G lesions. In dogs thirteen years and over, 4 (26.6%) showed Grade E lesions, 2 (13.3%) had Grade F lesions and 2 (13.3%) had Grade G lesions. Grade E and F lesions were seen in dogs at two years old, while Grade G lesions were seen in dogs five years and over.

Hyalin degeneration of the intramyocardial coronary vessels increased in frequency and severity with advancing age. There was a sharp increase in frequency in dogs between five and eight years inclusive and a sharp increase in the severity in dogs between nine and twelve years inclusive. In dogs thirteen years and over, 53.2% showed evidence of hyalin degeneration and 26.6% of these cases showed early Grade E lesions only. The overall frequency of hyalin degeneration was 29.5% of which 15.0% were Grade E lesions, 9.5%

were Grade F lesions and 5.0% were Grade G lesions. It was in the Grade G lesions that evidence of an associated myocardial ischaemia was seen, that is in 10 cases (5.0%). In the remaining 49 (24.5%) cases of hyalin change, no evidence of myocardial ischaemia was observed.

Sex distribution

Grade E lesions were seen in 17 males (16.2%) and 14 females (14.7%). Grade F lesions occurred in 7 males (6.6%) and 11 females (11.6%), while Grade G lesions occurred in 4 males (3.8%) and 6 females (6.3%). In all, hyalin degeneration occurred in 28 males (26.7%) and 31 females (32.7%). This sex difference is not considered to be significant. (Table 57)

Breed susceptibility

In the Alsatian, 4 dogs (25.0%) had Grade E lesions, 1 dog (6.2%) had Grade F lesions and 2 dogs (12.5%) had Grade G lesions, an overall frequency of 43.5% (Table 58). This is far higher than that seen in the control group (27.3%). The breed susceptibility appears to be greater in the Alsatian.

In the Border Collie, 7 dogs (22.6%) had Grade E lesions, 1 dog (3.2%) had a Grade F lesion and 3 dogs (9.7% had Grade G lesions, giving an overall frequency of 35.5%. This elevation of the frequency may be due to the higher mean age of the Border Collies (8 years 6 months) compared with the controls (Table 35).

In the Boxers, 2 dogs (18.2%) showed Grade E lesions, 1 dog (9.1%) had a Grade F lesion and 1 further dog (9.1%) had a Grade G lesion, giving an overall frequency of 36.4% compared with 27.3% for the control group. It is doubtful if this difference is significant, bearing in mind the small sample size of the Boxers (11 dogs).

The Greyhounds showed no cases of hyalin degeneration; the mean age of this group was 1 year 9 months. As stated above, hyalin degeneration was not seen in dogs under two years, so that absence of lesions in the Greyhound may not be unexpected.

In the Labradors, 1 dog (5.6%) had a Grade E lesion and 6 dogs (33.3%) had Grade F lesions, giving an overall frequency of 38.9%. This is considerably higher than the control group (27.3%). No Grade G lesions were observed.

Bearing in mind the limitations discussed in relationship to the breed susceptibility of chronic valvular disease, further study is required to determine if there is a true breed susceptibility to hyalin degeneration in the Alsatian and the Labrador.

The histopathology of canine arteriosclerosis

The earliest signs of arteriosclerosis were seen in the intima and internal elastic lamina. The latter showed a variety of changes which included beading, reduplication (Fig. 79), areas of degeneration (Fig. 80), fragmentation, and total loss of elastic fibres in one or more segments (Fig. 81). Total loss of elastic fibres was the most frequent abnormality. These changes were associated with a slight intimal oedema. No fat was seen in these lesions and the vascular endothelium was normal. These changes constituted a Grade A lesion. Lesions of this type were not recorded in the posterior aorta, particularly in the thoracic portion, where no clear-cut internal elastic lamina was seen.

As the lesions developed, radially-orientated fusiform smooth muscle cells clustered around these defects and some of the cells were seen, passing from the media, into the intima through the defects in the internal elastic lamina (Fig. 82). Disorganised smooth muscle cells were observed in the intima of some vessels, while in other cases they were seen lying in rows parallel to the long axis of the vessel. (Fig. 83). In these, the fusiform shape of the smooth muscle cells could not be seen in the standard transverse section. In some vessels, smooth muscle cells constituted the bulk of the lesion (Fig. 84), but delicate elastic , reticulin and collagen fibres were seen also. The presence of these new tissues thickened the intima over part or all of its circumference. These lesions were called intimal plaques. Some of these plaques were rich in amorphous ground substance and some contained mucopolysaccharides which could be demonstrated by the appropriate stains (Fig. 85). No lipid was seen in these plaques. These changes constituted the Grade B lesion. These Grade B changes were regarded as a basic arteriosclerotic lesion which could be seen in all the vessels that were examined. From this stage of development the lesions developed

different characteristics which appeared to depend upon the type and location of the vessel that was affected.

For example, the small plaques that developed in the thoracic portion of the posterior aorta were rich in elastic as well as collagen fibres. The lesions were related to the inlets to the intercostal branches where they formed 'cap-like' lesions which extended from the thoracic aorta around the junction and into the branch vessel(Fig.86). The arteriosclerotic lesions in the abdominal portion of the posterior aorta were more extensive than those in the thoracic portion. Two different types of lesion were seen. In one, a variety of tissue elements were observed which included smooth muscle cells, reticulin and elastic fibres and an abundant amorphous ground substance, rich in mucopolysaccharide. These elements were confined to the base of the plaque, nearest to the media. These tissues were covered by a layer of collagen fibres situated beneath the vascular endothelium of the plaque(Fig. 87). The relative proportions of each of these elements varied in different lesions but no fat was seen in any of them. These lesions were graded C and they were detected in the posterior aorta,coronary circulation and in the iliac and renal arteries on occasions.

The second, more advanced, lesion was confined to the abdominal aorta, being composed almost entirely of collagen fibres.(Fig,88) These lesions showed 'layering' in some cases, when the layers of collagen fibres were separated by delicate layers of elastic fibres (Fig. 89). These lesions, when severe, were associated with diffuse areas of intimal hyperplasia (Fig.75). No lipid was seen in these plaques and the overlying vascular endothelium was unaltered. (No fibrin deposition was seen on the vascular endothelium of any arteriosclerotic plaque, irrespective of their grade). The lesions, described above, were characteristic of a Grade D lesion, and these were confined to the abdominal portion of the posterior aorta.

Grade A lesions were not recorded in the posterior aorta, Grade B lesions were seen in 40 cases (20.0%), Grade C changes in 22 cases (11.0%) and Grade D changes in 17 cases (8.5%). In all, 79 dogs (39.5%) showed arteriosclerotic lesions in the posterior aorta.

Similar lesions were seen in the coronary circulation. In the left coronary artery, arteriosclerotic changes were seen in the ventral descending branch, the circumflex branch, and in the extramural divisions of these two vessels. All the coronary vessels had a clearly defined internal elastic lamina, so Grade A changes were recorded in all these vessels. Grade A lesions were seen in 41 cases (20.5%), Grade B lesions in 9 cases (4.5%) and Grade C changes in 4 cases (2.0%). No Grade D changes were seen in these vessels. A total of 54 dogs (27.0%) showed arteriosclerotic changes in the extramural vessels of the left coronary artery. Approximately 75.0% of these affected vessels showed Grade A changes which had no clinical significance whatsoever. All the Grade B lesions and three of the four Grade C lesions were rich in smooth muscle cells, with a minimal content of elastic and collagen fibres (Fig. 84). The other Grade C lesion showed extensive fibrous intimal plaques in descending branches of the left circumflex artery of a 10 y.o. Corgi female (Fig. 90). No lipid was seen in any of these affected vessels, there was no fibrin deposition, and the vascular endothelium was normal. These lesions in the extramural branches of the left coronary artery were less frequent and severe than in those seen in the posterior aorta.

In the right coronary artery, arteriosclerotic plaques were seen in 16 cases in the main trunk and, in one case, in a descending branch of that vessel. Grade A lesions were seen in 9 cases (4.5%), Grade B changes in 6 cases (3.0%) and Grade C changes in 2 cases (1.0%). In all 17 dogs (8.5%) showed arteriosclerotic changes in the extramural branches of this vessel. Lesions in the right coronary artery were less frequent

than those that were seen in the left coronary artery. They were similar in character to those described in the left coronary artery (Fig. 80).

Arteriosclerotic changes in the intramural coronary vessels were confined to the left ventricle. Here, lesions of two different types were observed.

Firstly, lesions similar to those described in the extramural branches were seen in those intramural branches that supplied the innermost third of the left ventricular myocardium, the papillary muscle and apex of the heart. Grade A lesions were seen in 5 cases(2.5%) and Grade C changes in 1 case (0.5%). In all, 11 dogs (5.5%) showed this arteriosclerotic lesion which, in six cases, was characterised by the presence of numerous smooth muscle cells which caused some narrowing of the lumen of the vessel (Fig. 84). Mucopolysacchanide may also be a feature of some of these lesions (Fig. 91). No lipid was seen and the vascular endothelium showed no abnormality. These lesions in intramural vessels had a very low frequency.

The low frequency of these lesions in the intramural vessels was due to the fact that a secondary change took place within them. In consequence, these initial lesions were obscured and could not be recorded. The secondary changes were characterised by an infiltration and deposition of a hyalin material into the intima and media of these, and other, vessels (Fig. 92). The grading system that was applied to the other arteriosclerotic lesions was extended to include this Hyalin change. These vessels were classified in Grades E to C, according to the extent of the hyalin change within their walls.

In a Grade E lesion, the hyalin material was confined to the vicinity of the internal elastic lamina, in most cases. This material was deposited on one, or both, sides of this elastic layer (Fig.93) Hyalin may be extracellular or, in some cases, it could be seen within the

cytoplasm of smooth muscle cells. On occasion, hyalin could be seen lying beneath the endothelium rather than around the internal elastic lamina. There was no stenosis of the vascular lumen and no lipid was seen in these lesions.

Grade F lesions showed an increase in the amount of hyalin in the vessel wall. This hyalin replaced the normal tissues and the internal elastic lamina, intima and media were destroyed. Lesions in the intima appeared as hyalin plaques or as circumferential thickenings of the intima which resulted in a variable degree of stenosis of the vascular lumen (Fig. 94). Lesions in the media appeared to extend outwards from the vicinity of the internal elastic lamina towards the adventitia, which was unaffected by this process. The hyalin may extend circumferentially or segmentally and the normal smoothmuscle was destroyed. As the hyalin accumulated, the media became thickened causing stenosis of the vascular lumen. The vascular endothelium was unaltered and no lipid was seen in these lesions.

In the Grade G lesion, the intima, media and internal elastic lamina have been destroyed and replaced by a mass of hyalin material which caused marked stenosis or total occlusion of the vascular lumen (Fig. 92). The vessel wall was grossly thickened and, in some cases, slit-like spaces and clefts were seen within these vessels. Some of these clefts were lined with a vascular endothelium and many contained red blood cells and haemosiderin . These channels were considered to be examples of re-canalisation, following vascular occlusion (Fig. 95). In an occasional severely affected vessel foam cells, containing lipid, were observed(Fig. 92)

Grade E lesions were seen in 31 cases (15.5%, Grade F lesions in 18 cases (9.0%) and Grade G lesions in 10 cases (5.0%). A total of 59 cases (29.5%) showed hyalin changein intramyocardial coronary vessels of the left ventricle. No changes of any type were seen in the intramural

coronary vessels of the right ventricle.

The Grade G lesions were associated with areas of myocardial degeneration and fibrosis in the inner third of the left ventricular myocardium. These myocardial lesions appeared to be a consequence of the vascular occlusion.

In association with intimal lesions, changes may be seen in the inner third of the media of the posterior aorta (Fig. 96). Indeed, such changes may be seen in media in the absence of intimal lesions. These medial lesions may be seen throughout the posterior aorta, but they are more frequent in the thoracic aorta. Here, there was a focal degeneration of elastic fibres and cystic spaces developed which were filled with a mucopolysaccharide material (Fig. 96). Later this defect was filled with collagen. Similar lesions were seen in some extramural coronary vessels (Fig. 97). These changes were observed in dogs of all ages.

Arteriosclerotic plaques in the renal arteries were similar to those seen in the abdominal portion of the posterior aorta, while the iliac arteries showed lesions, rich in elastic tissue, that were similar to those seen in the thoracic aorta.

The histochemistry of the hyalin material in the walls of the intramural vessels

This hyalin material was PAS-positive and diastase negative. There was no metachromasia with Toluidine blue or Azur A but a faint blue colour was evident with Alcian blue. A dark blue focal response was obtained by staining with Hale's colloidal iron but there was no response to aldehyde fuchsin.

Frozen sections stained with Sudan IV revealed the presence of fat in some of the Grade F and Grade G lesions.

Sections stained by Lugol's iodine showed a mahogany-brown colouration in the walls of affected vessels. These same vessels showed a pink to red colouration using Bennhold's congo red and the alkaline congo red method. (Fig 102) Other sections stained by methyl violet gave these vessel walls a bright red colour (Fig 100). After staining with Thioflavine T, the walls of these affected vessels fluoresced under ultra-violet light (Fig 101). In mildly affected vessels (Grade E lesion), this hyalin material was seen lying between the smooth muscle cells of the media and intima.

A similar response was seen on the inflow surface of the ventral cusp of the left atrioventricular valve and in the main trunk of the left circumflex coronary artery in two cases.

It was concluded that the hyalin material, observed in these situations, was amyloid. No such material was detected in the interstitial tissues of the myocardium.

Medial calcification of the posterior aorta

Medial degeneration with secondary calcification was seen in 10 dogs (5.0%). These lesions were confined to the thoracic portion of the posterior aorta and 7 (3.5%) occurred in the aortic arch.

With one exception, all these dogs were over five years old with an age range of one to fourteen years and a mean age of eight years, four months.

A total of 7 (3.5%) were male and 3 (1.5%) were female.

These cases of medial calcification were seen in 7 different breeds and 1 crossbred. These included two Border Collies and two Labradors.

These dogs died from a wide range of conditions which did not include chronic renal failure or vitamin D poisoning.

Only 3 (1.5%) of these cases were associated with aortic arteriosclerosis but all were associated with various grades of chronic valvular disease in the left atrioventricular valve. Only one case showed chronic valvular disease in the aortic valve.

Histologically, these lesions appeared as foci of degeneration in the inner half of the media with a secondary deposition of calcium into the necrotic tissue.

The Pathology of the Myocardium

A number of myocardial conditions were observed involving both the atria and ventricles of the heart.

Hypertrophy, with or without dilatation was seen in association with the Type III and IV lesions of chronic valvular disease and also in cases of chronic interstitial nephritis. In a number of these dogs both conditions occurred in the same animal and it was difficult to determine the responsibility for the hypertrophy that was observed.

Cardiac dilatation was seen in cases of congestive heart failure. It was apparent that dilatation of the right ventricle was more readily detected than dilatation on the left side.

No gross myocardial infarcts were observed but recent small septic emboli were evident in one case of bacterial endocarditis.

Myocardial degeneration and fibrosis was seen in 10 cases (5.0%). In four cases these lesions were detected as small, red or depressed, grey foci, a few millimetres in diameter, lying beneath the endocardium of the left ventricle. These lesions were located in the papillary muscles and apex of the left ventricle (Fig. 98). On cut surface, more of these lesions were evident in the innermost third of the left ventricular myocardium. Histological examination of these and other hearts revealed foci of degeneration, necrosis and fibrosis of the myocardium, in these areas (Fig. 98). No similar lesions were seen in the right ventricle. These lesions were associated with hyalin degeneration of the related intramyocardial coronary vessels and these myocardial lesions are considered to be due to a local myocardial ischaemia.

Myocarditis was associated with a variety of intercurrent conditions in seventeen cases (8.5%). These lesions were suppurative or non-suppurative. Suppurative myocarditis was seen in six cases (3.0%) in association with bacterial endocarditis and septic emboli, post-operative infection

following intra-cardiac surgery, chronic rhinitis, metritis and secondary infections following a traumatic injury. Four of these dogs were males, two were females and their ages ranged from ten months to nine years. These lesions were multifocal and widespread in some cases, the papillary muscles were frequently involved.

Non-suppurative myocarditis was associated with conditions such as broncho-pneumonia, congenital cardiac disease, visceral larva migrans, chronic interstitial nephritis and myocarditis of unknown aetiology. A total of eleven dogs (5.5%) showed these lesions of which seven were males and four were females. Their ages ranged from two months to sixteen years.

In eight of these cases (4.0%) these myocardial lesions were considered to be the primary cause of death.

Only two cardiac tumours were observed in this series, one primary (?) tumour and one secondary tumour. The heart base tumour is not a true primary tumour as it has its origin in the aortic body. This tumour was seen in a twelve-year old West Highland White terrier male. It was located between the aortic root and the pulmonary artery and was 1.5 cm in diameter. One secondary tumour was seen in the ventral papillary muscle of a twelve-year old Cocker spaniel female with a malignant adenocarcinoma of the mammary gland.

Extensive sub-endocardial haemorrhage with bleeding into the adjacent myocardium was observed in a seven-year old Border Collie female who died of Warfarin poisoning.

In the left atrium, myocardial degeneration and fibrosis was observed in seven cases (3.5%). Lesions of this type were associated with insufficiency of the left atrioventricular valve in five of the seven cases. Three of these dogs were Boxers and two were Alsatians and their ages ranged from four to fourteen years.

Congenital cardiac disease

A total of six cases (3.0%) of congenital cardiac disease were observed.

There were three cases of pulmonic stenosis with constriction of the pulmonary inlet and a right ventricular hypertrophy. Two of these cases were seen in crossbred males aged twelve and sixteen weeks respectively and the third case was in a ten-month old Greyhound female.

Sub-aortic stenosis was seen in two dogs, one male and one female. A two year old Boxer female died of congestive heart failure a few days after parturition. The second case was seen in a six-month old Staffordshire Bull terrier male. Both showed a dense ring of fibrous tissue beneath the aortic valve and a dilated left ventricle.

One seven-week old Whippet female showed a persistent right aortic arch with strangulation of the oesophagus. The condition was diagnosed by x-ray and the dog was destroyed.

The Pericardium

A serous transudate was observed in cases of congestive heart failure in amounts ranging from 1-5 ml. This fluid was clear and pale yellow in colour. Several millilitres of a sero-sanguinous fluid were observed in association with a case of heart base tumour.

Haemorrhage and fibrin deposition on to the epicardium was seen in one dog with fractured ribs following a traffic accident.

Four cases of pericarditis were observed. One in a ten-year old Labrador male which had a localised, organising, pericarditis over the left atrium; this lesion was an incidental finding. An acute serous pericarditis was observed in a three-year old Alsatian male, who died of an acute leptospiral nephritis.

A suppurative pericarditis was observed in a two-year old Border Collie male following intra-cardiac surgery. A chronic constrictive pericarditis was seen in a ten-year old Sealyham female which showed extensive fibrous adhesions between the visceral and parietal pericardium. No acid-fast organisms were observed, the aetiology was unknown.

Bacterial endocarditis

Two cases (1.0%) of bacterial endocarditis were seen in this series. One, in a four year old Boxer female which had recent lesions on the outflow surfaces of the left atrioventricular valve. This dog died of congestive heart failure.

The second case was in a three year old Springer spaniel male which was destroyed as incurable after antibiotic treatment for a septic arthritis. At post-mortem an organizing pleurisy and bacterial endocarditis of the aortic and left atrioventricular valve were found. Staphylococcus pyogenes was isolated from the original joint lesion.

Discussion

This study had two basic objectives, firstly, to investigate the frequency and nature of some of the common spontaneous cardiovascular diseases of the dog, i.e. chronic valvular disease, arteriosclerosis and hyalinosis of the intramyocardial coronary vessels. The second objective was to compare these conditions with perallel conditions in Man. A number of other cardiovascular conditions were found in this study and these have received brief consideration.

The material which formed the basis of this study was obtained from 200 unselected dogs which were presented to the Department of Veterinary Pathology for post-mortem examination by the author. Great emphasis has been laid on the frequency and distribution of cardiovascular lesions in these dogs and, thus, it is important to know if these 200 dogs were representative of the canine population as a whole.

In the absence of any realistic dog licensing system in the United Kingdom, no information was available on the normal canine population. The only available population statistics are based on animals presented to veterinary hospitals. Robinson (1968) compared a 'hospital' population with the normal population from which they were drawn and found many discrepancies. A higher proportion of young dogs were seen in the 'hospital' population than in the normal population. Any recent breed 'fancy' could result in larger numbers of young dogs of a particular breed in the 'hospital' population which was not reflected in the normal population. In contrast, middle-aged animals were poorly represented in the 'hospital' population compared with the normal population where they represented a large proportion of the total. Dogs over nine years were found in increasing numbers in the 'hospital' population, while they declined steadily in the normal population. In the 200 dogs in this study, a similar age distribution was found to that seen in the Robinson's 'hospital' population.

There were 76 (38.0%) dogs under five years old, 39 (19.5%) dogs five to eight years inclusive and 85 (42.5%) dogs nine years and over (Table 2). Other variations between a 'hospital' population and the normal population included local breed fancies and the susceptibility of those breeds to certain diseases. Therefore, it was clear that there were many discrepancies between a 'hospital' population and the normal population but this 'hospital' population of 200 dogs was the only one that was available, imperfect though it was.

A standard technique was adopted for the examination of the heart which included an assessment of the function of the atrioventricular valves (pages 4-5). Both atria were opened to expose the outflow surfaces of these valves, the aortic root and pulmonary artery were ligated, and the ventricles filled with warm, normal, saline. Both atrioventricular valves were closed by gentle manual pressure on the ventricles and an assessment of valvular competence was made. A similar technique was employed by Sheridan (1967). The variables in this technique (e.g. degree of rigor mortis in the myocardium, the intensity of the manual pressure applied to the ventricles, etc) made this technique unreliable by itself but, coupled with the gross examination of the heart, this technique was of some value. It should be noted that the terms 'no apparent clinical significance' have been used regularly in the text.

The anatomical studies that have been undertaken had two objectives. One, to establish the normal structure of the heart valves and the second, to establish the nature of the blood supply to certain defined areas of the left ventricular myocardium, using injection techniques.

As far as could be determined, no detailed study of the histology of the canine heart valve had been undertaken. The structure of the canine heart was similar to that of the human heart valve, as described by Gross & Kugel (1931). The only variation was the presence of cartilagenous

plates (in the interventricular septum) in the vicinity of the right fibrous trigone of the cardiac skeleton (p.13). Ageing changes were also seen in these valves but these will be discussed later in conjunction with the pathology.

Detweiler et alia (1960) described areas of myocardial degeneration in the papillary muscles and apex of the left ventricle which they associated with hyalinosis of the related intramyocardial vessels. Injection studies, using vinyl acetate, carmine-gelatin and the commercial radio-opaque material 'Micropaque', were employed to determine the blood supply to the ventral (anterior) papillary muscle, the dorsal (posterior) papillary muscle and the apex of the left ventricle. All three injection masses possessed excellent powers of penetration of the smaller intramyocardial vessels. These studies confirmed many of the observations made by other authors on the basic structure of the coronary circulation (Blair 1961; Christensen 1962; Christensen and Campeti 1959; Donald and Essex 1954; Garmella et alia 1957; Kazzaz and Shanklin 1950; Lumb et alia 1959; Meek et alia 1929; Moore 1930; Pianetto 1939). There was a strong left dependence of the canine coronary circulation; the left coronary artery supplying the free wall and papillary muscles of the left ventricle, the left atrium, the interventricular septum, the margins and apex of the right ventricle and this vessel also sent branches to the right atrium. The right coronary artery also supplied the right atrium and 66.0% of the free wall of the right ventricle.

There are differences between the coronary circulation of the dog and Man. The dorsal (posterior) descending branch originates from the left circumflex coronary artery and not the right coronary artery as it does in the majority of cases in Man. The ventral (anterior) septal artery of the dog is a large single vessel which supplies 70-75% of the interventricular septum, but in Man the septum is supplied by a series of small vessels derived from the anterior descending branch. The normal coronary circu-

lation of the dog is rich in anastomses between all the major divisions of the coronary artery (p.79). These anastomoses can be readily seen by the naked eye which gives them a diameter of approximately 60-80 microns. Such natural anastomoses are delicate and uncommon in the normal coronary circulation of Man (Blumgart et alia 1950). These abundant, natural coronary anastomoses do much to protect the heart of the dog by limiting the effects of vascular occlusion. Spontaneous gross myocardial infarction is uncommon in the dog. The great variability in the results of experimental coronary occlusion studies in the dog are probably due to these abundant natural anastomoses (Blumgart et alia 1950). The consequences of vascular obstruction by hyalinosis of intramyocardial coronary vessels are extremely limited and the natural intercoronary anastomoses undoubtedly play a part in limiting this response.

All these characteristics are well documented in the literature although the significance of some of these characteristics have not been appreciated by some pathologists. But the prime objective of these investigations was to determine the details of the coronary supply to the papillary muscles and apex of the left ventricle. This objective was achieved by isolating the major divisions of the left coronary artery and injecting them with Micropaque In undertaking this study, allowance had to be made for the natural anastomoses that occurred but a good level of correlation was obtained, despite a certain 'overlap' in the areas supplied by each vessel.

As a result of these studies (p.77-78) it was established that these particular areas were supplied almost entirely by the ventral (anterior) descending branch and by divisions of the median branch of the left circumflex coronary artery. A small area of the dorsal papillary muscle was supplied by the dorsal descending branch of the left coronary artery.

In the course of this study, another system of blood vessels was recognised which joined the coronary vessels to the heart chambers themselves.

These vessels were similar in their character and distribution to the luminal VESSELS of the human heart which have been described by Wearn et alia (1933) and others. These vessels were of two types, the myocardial sinusoid and the arterio-luminal vessel, both of which were found in the innermost third of the left atrial and ventricular myocardium. The myocardial sinusoids formed vascular loops around the bases of the papillary muscles and the trabeculae carneae. They were extremely thin-walled vessels which meandered between the muscle bundles and were ultimately connected to the coronary vessels. These myocardial sinusoids were extremely variable in diameter. In the papillary muscles, these vessels were concentrated around the base of the muscle and were far removed from the hyalinised vessels (Fig. 21). It was possible to build up a picture of the blood supply to the papillary muscles. The major myocardial sinusoids running around the base of the papillary muscle with divisions of the left coronary artery running into the substance of that muscle - it is the coronary vessels which show the hyalin change. These affected vessels are far thicker-walled than the myocardial sinusoids. Fig. 99 gives a three-dimensional picture of the blood supply to the papillary muscles.

The arterior-luminal vessel is a shorter, thicker-walled, straight vessel which anastomoses with the coronary vessels directly. The arterio-luminal vessels are particularly common in the outflow tract of the left ventricle. The exact function and direction of blood flow in the luminal vessels is not clear, possibly the blood moves in both directions at different stages of the cardiac cycle.

To summarise the conclusions on the injection studies that have been undertaken, it is clear that the canine coronary circulation has a strong left dependence, unlike the majority of human hearts. This view is supported by Blumgart et alia (1950) who also confirmed the fact that many normal anastomoses existed in the canine coronary circulation, unlike Man where such

anastomoses were few. It was the fact that hyaline degeneration of intramyocardial coronary vessels occurred most commonly in those vessels which supplied the papillary muscles and apex of the left ventricle (Detweiler et alia 1960) that prompted a detailed study of the blood supply to these areas. It was found that these vessels were derived from the ventral (anterior) descending branch and the median branch of the left circumflex coronary artery. In addition, it was found that luminal vessels were also present in the dog's heart which have not been reported previously, but the hyalin change did not appear to occur in these vessels.

It is generally accepted that insufficiency of the left atrioventricular valve is the most common cardiac abnormality that is detected upon the clinical examination of the dog's heart (Detweiler 1958; Joshua 1959; Das and Tashjian 1965; Pirie 1965). This insufficiency was most frequently associated with chronic valvular disease which may lead to congestive heart failure in some cases (Detweiler 1952). Chronic valvular disease is recognised by the presence of greyish-white smooth, glistening nodules or irregular, plaque-like thickenings in the area of apposition of the valve cusp. Thickening of the proximal portions of the chordae tendineae are also seen. Since this condition was first described (Cadiot 1892) numerous papers have been published on the clinical aspects and gross pathology of this condition but little information is available on the histopathology of the condition.

In 1967, Whitney devised a grading system which has been employed in this study. The system is based upon a gross visual examination of the valves without any direct measurement of the valve lesions. The extreme variation in the size of lesions due to the variations in the age and breed of the subjects would make any direct measurements meaningless (pp. 83-84). The Type I lesion is the earliest gross manifestation of this condition that can be detected by naked-eye examination. These lesions increase in size causing greater distortion of the valve cusp until, in their most severe form, they are

graded a Type IV lesion. The grading system forms the basis of the epidemiological studies that have been reported here.

Although the clinical manifestations of chronic valvular disease are primarily associated with the left atrioventricular valve, lesions are not confined to this valve alone but may be found in the right atrioventricular valve, the aortic valve and, on occasion, in the pulmonary valve.

In this study, chronic valvular disease was seen in 69.5% of left atrioventricular valves. These lesions were evident in both the ventral and dorsal cusps, but often appeared to be more pronounced in the ventral cusp. In the right atrioventricular valve, however, the lesions were of greater frequency and severity in the septal cusp than in the mural cusp. In the septal cusp, chronic valvular disease was seen in 52.5% of cases while only 11.5% of mural cusps showed lesions. In the semi-lunar valves, 7.0% of aortic valves showed lesions while no gross abnormality was detected in any of the pulmonary valves. Thus, it is seen that chronic valvular disease occurs in descending order of frequency in the left atrioventricular valve, right atrioventricular valve, aortic valve and pulmonary valve. These conclusions are in agreement with other published work (Munich 1935; Bretschneider 1962; Stunzi and Teuscher 1962; Das et alia 1965; Luginbuhl and Detweiler 1965). Sheridan (1967) saw a greater frequency of lesions in the septal cusp than in the mural cusp of the right atrioventricular valve.

By applying the grading system to these lesions, Type III and IV lesions (lesions of apparent clinical significance) were seen in 33.0% of left atrioventricular valves, 16.0% of septal cusps and no mural cusps in the right atrioventricular valve, 3.5% of aortic valves and none in the pulmonary valve. Type III and IV lesions represent the most severe manifestations of chronic valvular disease so that we may conclude that not only the most frequent but also the severe lesions are seen in the left atrioventricular valve, right atrioventricular valve, aortic valve and pulmonary valve in descending order of frequency.

Chronic valvular disease was seen in 17.0% of the left atrioventricular valves without lesions in the right atrioventricular valve. Lesions occurred in both valves in 45.5% of cases and a further 2.0% showed lesions in the right atrioventricular valve only. These observations suggest that lesions tend to occur in the left atrioventricular valve before they occur in the right atrioventricular valve.

It is generally acknowledged that chronic valvular disease increases in frequency with advancing age. Das and Tashjian (1965) and Jones and Zook (1965) have demonstrated a linear relationship between the frequency of chronic valvular disease and advancing age. Other authors have shown that chronic valvular disease is more prevalent in older dogs (Munich 1935; Detweiler et alia 1960; Detweiler and Patterson 1965; Pirie 1965; Wallace 1965; Whitney 1967; Pomerance and Whitney 1970). But none of these authors has attempted to demonstate an increase in the severity of these lesions, although this may have been implied in their text.

In this study, the frequency and severity of chronic valvular disease was compared in dogs under and over nine years old. In the left atrioventricular valve, the frequency rose from 51.2% under nine years to 94.2% at nine years and over. Type III and IV lesions rose from 13.8% to 58.9% at nine years and over. There are considerable differences in the frequency and severity of lesions in the right atrioventricular valve so each cusp was considered separately. In the septal cusp, the frequency rose from 38.3% under nine years to 72.8% in the older group. The severity rose from 8.8% in the younger group to 27.0% in dogs nine years and over. Lesions in the left atrioventricular valve have a greater frequency and severity than lesions in the septal cusp of the right atrioventricular valve.

In the mural cusp of the right atrioventricular valve, no Type III or IV lesions were observed and the frequency of chronic valvular disease was much lower, rising from 7.6% under nine years to 17.6% in dogs nine years and over. Even Type II lesions were uncommon in this cusp.

The frequency of lesions in the aortic valve is even lower but Type III and IV lesions were observed. In dogs under nine years 4.3% showed lesions compared with 10.5% nine years and over. The severity increased from 1.7% Type III and IV lesions under nine years to 5.8% at nine years and over. No gross lesions were seen in the pulmonary valve.

These observations support the view that both the frequency and severity of chronic valvular disease is higher in older dogs than in young dogs. Lesions have a greater frequency and progress more rapidly in the left atrioventricular valve than in the septal cusp of the right atrioventricular valve. Lesions are less frequent and severe in the mural cusp of the right atrioventricular valve and in the aortic valve.

Detweiler and Patterson (1965) suggested that chronic valvular disease began in the first third of life and that valvular insufficiency of the left atrioventricular valve and, perhaps, the right atrioventricular valve developed in middle age. This insufficiency was well tolerated in many individuals who survived to reach nine to twelve years or more. The implication is that these lesions develop early in life and progress throughout the life-span of the dog causing valvular insufficiency and congestive heart failure in some cases in later life.

In an attempt to test this hypothesis in this study, these 200 dogs were divided into four age groups. Ideally, the dogs should have been examined year by year but the numbers were insufficient. In the left atrioventricular valve a progressive increase in the frequency and severity of lesions was demonstrated. In dogs under five years, the frequency rose from 36.9% to 100.0% in dogs thirteen years and over. There was a similar increase in severity (Type III and IV lesions) from 9.2% under five years to 86.6% in dogs thirteen years and over.

A similar progression was observed in the septal cusp of the right atrioventricular valve but the lesions here were less frequent and severe.

In the septal cusp the frequency rose from 25.0% under five years to 80.0% at thirteen years and over. Type III and IV lesions rose from 2.6% to 46.6% in dogs thirteen years and over. The rate of progression of lesions was less pronounced than that seen in the left atrioventricular valve. There was, in both valves, a sharp increase in the frequency of Type III and IV lesions (which wa associated with valvular insufficiency) in dogs five to eight years inclusive. These observations are in agreement with Detweiler and Patterson's (1965) hypothesis and the conclusions of some other authors (Detweiler et alia 1960; Pirie 1965; Wallace 1965).

There was no conclusive evidence on the progressive nature of chronic valvular disease in the mural cusp of the right atrioventricular valve. The absence of Type III and IV lesions in this cusp suggests that these lesions are not progressive. In the aortic valve Type III and IV lesions were observed, so these lesions may be more progressive in this valve. The low frequency of lesions in these valves prevents any conclusions on their progressiveness.

The frequency and severity of chronic valvular disease has been plotted against these four age groups in the form of a series of graphs (Figs. 26-38). In the left atrioventricular valve, the essentially progressive nature of these lesions is clearly demonstrated. The Type I lesion predominates in the young dog, giving way in turn to the Type II, the Type III and, ultimately, the Type IV lesion. As these individual grades give way in precedence, their own frequency declines. The progressive nature of lesions in the septal cusp of the right atrioventricular valve is also evident, but this progression is a slower process and Type II lesions tend to persist throughout the life span of these dogs. The low frequency of lesions in the mural cusp of the right atrioventricular valve and aortic valve obscures the picture and prevents any conclusions being made.

In Tables 8 and 9 the age of onset of lesions in the heart valves are recorded and reinforce the view that lesions progress more rapidly in the left atrioventricular valve than in the septal cusp of the right atrioventricular valve.

If the frequency and severity of lesions in the septal cusp is compared with that of the mural cusp of the right atrioventricular valve, striking differences are evident. The frequency of lesions in the septal cusp is 52.5% compared with 11.5% in the mural cusp. Type III and IV are confined to 16.0% of septal cusps. Indeed Type II lesions are only seen in 1.5% of mural cusps. Only Sheridan (1967) has noted this disparity between these two cusps. No lesions were seen in the mural cusps of dogs under three years. No explanation can be offered for this disparity in frequency and severity of valve lesions in these two cusps of the right atrioventricular valve.

A considerable effort has been directed towards the determination of any sex predisposition to chronic valvular disease. Munich (1935) and Das and Tashjian (1965) were unable to see any such predisposition based on the frequency of lesions in the two sexes. Das and Tashjian (1965) and Detweiler and Patterson (1965) found that congestive heart failure was more prevalent in the male. Detweiler (1966) also states that acquired heart disease (including chronic valvular disease) developed more rapidly in the male than in the female. In this study, there were 105 males, 89 entire females and 6 females that had been ovariectomised. These female castrates have not been considered in this context. The distribution of the sexes was imbalanced in this study, but it is not considered that these imbalances have affected these conclusions to any extent.

It is seen that the frequency of chronic valvular disease in the left atrioventricular valve of the male was 70.5% and 67.4% in the female. An even balance which supports the views of Munich (1935) and Das and

and Tashjian (1965). A comparison of severity of lesions shows Type III and IV lesions in 30.5% of males and 36.0% of females. A comparison of the frequency of lesions in dogs under and over nine years shows that in dogs under nine years, lesions occurred in 53.1% of males and 48.9% of females, but Type III and IV lesions occurred in 10.5% of males and 19.1% of females. In dogs nine years and over 100.0% of males and 88.0% of females showed chronic valvular disease and Type III and IV lesions occurred in 64.1% of males and 54.7% of females. While there was a greater overall frequency of chronic valvular disease in the male, the young female had a greater frequency of Type III and IV lesions. This ascendancy was lost in later life when the male had the greater number of severe lesions.

In the septal cusp of the right atrioventricular valve, chronic valvular disease occurred in 52.3% of males and 52.8% of females; Type III and IV lesions were seen in 19.0% of males and 11.2% of females. In the septal cusp, unlike the left atrioventricular valve, severe lesions occurred more regularly in the male. Furthermore, in the septal cusp, lesions were more progressive in the male than in the female.

The low overall frequency of lesions in the mural cusp of the right atrioventricular valve and aortic valve prevents any clear-cut conclusion.

It would seem that some young females (under nine years) develop Type III lesions in the left atrioventricular valve at a younger age than in the male and in the septal cusp of the right atrioventricular valve there is a greater number of Type I and II lesions in the female. In dogs over nine years, the ascendancy passes from the female to the male, who shows a greater frequency and severity in both valves.

In order to compare the rate of progression of lesions in both sexes, the frequency and severity of chronic valvular disease was assessed in the four age groups.

In dogs under five years, chronic valvular disease was seen in 35.5%

of males and 38.6% of females. Type III and IV lesions were seen in 2.2% of males and 19.1% of females. Lesions of apparent clinical significance were far more prevalent in females under five years old. In all three age groups over five years old, chronic valvular disease had a greater frequency and severity in the male than in the female.

No excess was seen in the septal cusp of females under five years, but the frequency and severity of lesions was higher in the male than in the female in dogs over five years old. The low frequency of lesions in the mural cusp of the right atrioventricular valve and the aortic valve did not allow any conclusions on the sex predisposition in these valves.

With the exception of Type III lesions, all other grades of chronic valvular disease developed at a younger age in the male than in the female in the left atrioventricular valve and the septal cusp of the right atrioventricular valve.

There is little variation in the overall frequency of chronic valvular disease in the two sexes which confirms the view expressed by Munich (1935) and Das and Tashjian (1965). However, the employment of the grading system has shown that lesions are more progressive and ultimately more severe in the male than in the female. The higher frequency of Type III and IV lesions in the male means a higher level of valvular incompetence in the male and these severe lesions developed at a younger age in the male. These conclusions lend support to the view expressed by Das and Tashjian (1965) and Detweiler and Patterson (1965) that congestive heart failure was more prevalent in the male. Chronic valvular disease is associated with congestive heart failure in the dog (Detweiler 1952; Wallace 1965). The more rapid progression of lesions in the male supports the view of Detweiler (1966) that acquired heart disease developed more rapidly in the male than in the female. One anomaly also emerged, namely, the higher frequency of Type III lesions in females under five years old. Why this

should be so is not entirely clear since this predominance of lesions is not evident in older females. In addition, there is a slightly higher frequency of lesions in the septal cusp of these young females which is not evident in the older females. What could be responsible for this is not certain. What might accelerate this process in some young bitches? It is suggested that pregnancy and parturition may be responsible, the tendency is always to breed females at a young age, largely for commercial reasons. The stress on the systemic circulation produced by pregnancy may, in some way, accelerate the process.

Little has been said on the breed predisposition to chronic valvular disease. Munich (1935) found no evidence of a breed predisposition in a small series of cases. Das and Tashjian (1965) and Detweiler and Patterson (1965) found that the Boxer and Cocker Spaniel were more susceptible than other breeds. In this study, five breeds were represented by more than 10 individuals, namely the Alsatian, Border Collie Boxer, Greyhound and Labrador. The frequency and severity of chronic valvular disease in each of these breeds was compared with a control group. As it already has been shown that the age and sex may influence the frequency and severity of the valve lesions, the mean age and distribution of the sexes was assessed for each of these breeds and compared with the control group. It was clear that the small number of individuals involved severely restricted the value of any conclusions, the smallest number was 11 Boxers, the largest was 31 Border Collies.

In consequence, no clear-cut breed predisposition could be demonstrated, although there was suggestion that the Labrador was more susceptible, a greater number of this breed would need to be examined before any firm conclusion could be made. The importance of mean age in relation to breed susceptibility emerged as an important factor. The mean age of the Border Collies was 8 years 6 months, far higher than the control

group. Chronic valvular disease had a greater frequency and severity in the left atrioventricular valve and a greater frequency in the septal cusp of the right atrioventricular valve than in the control group. In contrast the mean age of the Greyhounds was low (1 year 9 months), when compared with the control group. Both the frequency and severity of chronic valvular disease was much lower than the controls. The mean age emerged as an important factor in any consideration of breed susceptibility. The Sex distribution in these groups was not sufficiently disproportionate to significantly alter the results.

It was possible neither to confirm nor refute the suggestion that the Boxer and Cocker Spaniel had any particular breed susceptibility , the numbers involved were too small.

Munich (1935) was unable to correlate the frequency of chronic valvular with other intercurrent diseases e.g. chronic interstitial nephritis and Canine Distemper. In this study, the causes of death or destruction are summarised in Table 5. The frequency of any one particular disease in this series was considered to be too small to produce any meaningful results. Also, this study showed that in some cases the post-mortem revealed not one, but a number of conditions present in the one dog. Under these circumstances , the prime cause of the clinical condition was considered to be the cause of death and the case was recorded under the appropriate heading.

As the frequency of any particular intercurrent diseases was too small to produce satisfactory results, these diseases were classified according to the body systems that were principally involved e.g. Locomotor, Respiratory etc. (Table 5). In each of these groups the frequency and severity of chronic valvular disease was assessed and compared with the control group, represented by the whole series of 200 dogs. In order to test the comparability of each group the mean age

and distribution of the sexes was assessed and compared with the controls.

A study of these results showed an extreme variability in mean age, distribution of the sexes and the frequency and severity of chronic valvular disease in the left atrioventricular valve and septal cusp of the right atrioventricular valve which was summarised in Table 41. These groups, including the controls, were classified according to the mean age and, when the mean age was higher than the control group, both the frequency and severity of chronic valvular disease were higher than in the control group. There were some exceptions, in the Alimentary group of diseases the severity of Chronic valvular disease was less than was seen in the control group. In the septal cusp of the right atrioventricular valve, the severity was generally greater than the control group, with the exception of the Reproductive group of diseases and the frequency of lesions was greater in all excepting the Urinary group of diseases. But, with these two exceptions , it is true to say that both the frequency and severity of chronic valvular disease is greater than that seen in the control group.

In those groups of diseases where the mean age was lower than that of the control group, the picture is more complex. Not surprisingly, the frequency and severity of chronic valvular disease was higher in the group of cardiovascular diseases, which included 12 cases of congestive heart failure following insufficiency of the left atrioventricular valve. Here, the mean age of the Cardiovascular group was lowered by the inclusion of 6 cases of congenital heart disease. Similarly in the Locomotor group, these too had a frequency and severity close to that seen in the control group but this group included cases of rickets and osteomyelitis which lowered the mean age of this group. In the two youngest groups, Respiratory diseases and Systemic diseases, the frequency and severity of chronic valvular disease was well below that of

the control group. So it is clear, with certain exceptions already mentioned, that when the mean age is lower than that of the control group, the frequency and severity of chronic valvular disease is reduced below that of the controls, and furthermore the mean age of these various groups can be correlated with the frequency and severity of chronic valvular disease.

The reasons for the variation in the mean age of these groups has been indicated in the previous paragraph. Each predominant disease (or diseases) have their own characteristic age incidence and distribution between the sexes, and it is these factors which influence the mean age of the group as a whole. For instance, in the group of Endocrine diseases (mean age 9 years 9 months) the predominant disease was diabetes mellitis, a condition which occurs in elderly females almost exclusively. This group also contained four cases of adenocarcinoma of the thyroid gland, a condition found in aged dogs. These diseases, in particular diabetes mellitis , raised the mean age of the group and modified the balance of the sexes towards the female. Similarly, in the group of Reproductive diseases (mean age 9 years 7 months) the predominant condition was an adenocarcinoma of the mammary gland, another condition associated with aged females. It is these females which were responsible for the high mean age and the imbalance of the sexes in this group.

At the lower end of the age range, the group of Respiratory diseases included cases of bronchopneumonia and Nocardial pleurisy which are known to occur in young dogs, thereby lowering the mean age of the group as a whole. Two diseases, Canine Distemper and Hepatitis contagiosa canis (Rubarth's disease) constitute the group of Systemic diseases. Both these diseases occur, characteristically, in young dogs,hence the low mean age of this group (11 months). In both these groups, the frequency and severity of chronic valvular disease is low. While it would appear to be

possible to correlate disease in particular body systems with the frequency and severity of chronic valvular disease, there is no direct relationship between them. The age incidence of the predominant diseases influences the mean age of each group and the mean age also influences the frequency and severity of Chronic Valvular disease.

It is evident that age plays an important part in the epidemiology of chronic valvular disease and so the normal aging changes in the heart valves must be considered when the histopathology of this condition is studied. It is necessary to distinguish between the normal ageing changes and any pathological lesions that may be present. The principle was adopted that the normal ageing changes in the valves were represented by the least changes that were detectable at any given age. Ageing changes were evident in the atrioventricular and semi-lunar valves but these were more pronounced on the left side of the heart. These changes were most readily detectable in the ventral cusp of the left atrioventricular valve.

A synopsis of the ageing changes seen in dogs between one day and fourteen years (page 159) shows that the most pronounced lesions were seen in the atrialis of the area of apposition. In this area, a fibro-elastic plaque developed which persisted throughout the life-span of the dog. As the plaque aged, the elastic tissue decreased and the collagen proliferated and became hyaline. The overlying endothelium was unaltered and no fibrin deposition was observed, however Lambl's excrescences were seen in one ten year-old female. The existence of these plaques was first recorded by Whitney (1967) following their description in the heart valves of Man. (McMillan and Lev 1964; Pensinger 1965; Pomerance 1966). Pomerance(1966) considers that these lesions in the area of apposition are a response to impact and are analagous to cutaneous callus formation. This view was supported by McMillan and Lev (1964).

In addition to these changes in the atrialis, an increase in fibrillar elements, notably elastic fibres, was seen in the spongiosa, opposite to the origins of the chordae tendineae. Adult fat cells accumulated in the spongiosa, originating in the vicinity of the valve ring and extending downwards towards the free border of the valve. The spongiosa was very cellular in the young dog but this cellularity declined with the passage of the years.

All these changes were most readily detectable in the ventral cusp of the left atrioventricular valve. They were less prominent in the dorsal cusp and less prominent still in the right atrioventricular valve. In the semi-lunar valves, similar changes were observed, particularly in the aortic valve where fibro-elastic plaques developed along the lunulae and the nodulae valvularum aortae were enlarged. These ageing changes were most pronounced on the left side, due to the greater systemic blood pressure, according to McMillan and Lev (1964). All these changes tended to be obscured when chronic valvular disease developed within the valve. The earliest histopathological changes of chronic valvular disease appeared as foci of oedema which separated and disorientated the fibrillar elements of the ventricularis and fibrosa. These lesions appeared in the distal third of the ventricularis, particularly at the junction between the valve cusp and the chordae tendineae, in the ventral cusp of the left atrioventricular valve. As these lesions increased in size, the process extended along the inflow surface of the cusp and downwards along the chordae tendineae so that the latter tended to become incorporated into the inflow surface of the valve.

The contiguity of the fibrosa with the ventricularis facilitated the extension of this process from the one to the other and a similar swelling and disorientation of fibrillar elements in the fibrosa occurred. With the enlargement of these oedematous foci certain other

changes became evident. The disorientated fibres fragmented, underwent degeneration and disappeared, to be replaced by a delicate reticulin network which supported the cellular elements of the lesion. Two predominant cell types were observed, one resembled the young fibroblast (fibrocyte) the other an undifferentiated mesenchymal cell; small numbers of plasma cells and lymphocytes were also seen. In addition, a tinctorial change took place characterised by the development of a metachromasia within this abundant ground substance.

This essentially degenerative process extended from the original foci, destroyed the normal fibrillar elements of the fibrosa and replaced them by this loose connective tissue. The same process extended into the proximal chordae tendineae causing these structures to swell and to become further incorporated into the inflow surface of the valve cusp. The overall effect of these lesions was to reduce the amount of supporting collagenous tissue in the fibrosa, ventricularis and chordae tendineae and replace it by a bulkier loose connective tissue which was rich in ground substance. In consequence, the affected areas became swollen but the ability of these new tissues to support the valve was poor so the valve was weakened.

A similar degenerative process occurred in the spongiosa. The early lesions were located in the triangular areas of spongiosa opposite to the origins of the chordae tendineae. These early lesions could be most readily detected in sections stained by Azur A or Toluidine Blue. They appeared as multiple foci of metachromasia which increased in size, coalesced with each other and ultimately gave rise to nodular swellings in these areas. The normal fibrillar structure and the neutral fat deposits in the affected areas were destroyed. Haemorrhage into the spongiosa in the distal third of the valve was seen in some dogs.

In 16.0% of cases similar lesions may be seen in the

distal half of the atrialis where the fibro-elastic plaque was destroyed and the definition between the atrialis and spongiosa was obscured.

The overall effect of these lesions was to cause a swelling of the valve distal to the line of apposition. The shape of this swelling depended upon the extent of the degenerative lesions. If the degenerative changes were confined to the areas opposite to the origins of the chordae tendineae the gross lesions appeared as nodular swellings. Where the swelling extended throughout the distal spongiosa and valve tip, the gross lesion was an irregular plaque-like deformity (Type III and IV). The lesions in the ventricularis and fibrosa only contributed to the swelling of the cusp to a limited extent; their importance lay in the destruction of the collagen of the fibrosa and chordae tendineae and the resultant loss of support to the valve during systole. The limited swelling that occurred was located on the inflow surface of the valve, in association with the swelling and incorporation of the proximal chordae tendineae. The destruction of the collagenous core of the chordae tendineae weakened these structures, causing them to stretch or, in some cases, to rupture spontaneously. Involvement of the atrialis was not common so, when the valve deformities developed, the atrialis proliferated to give rise to extensive fibrous plaques over the nodular or plaque-like deformities that arose as a result of changes in the spongiosa.

The foregoing discussion has been confined to the nature of the lesions and the effects of these lesions upon the ventral cusp of the atrioventricular valve. This valve has been used as a model for similar lesions in other valves. While the basic pathological process is the same, the lesions differed in their distribution in other heart valves. These differences may be linked to the different anatomy of these other valves to some extent.

These degenerative lesions were not confined to the ventral cusp as similar lesions were seen in the dorsal cusp of the left atrioventricular valve. These changes differed in their distribution, as they appeared to originate in the proximal two-thirds of the ventricularis and extended around the sinus pocket and into the mural endocardium. Otherwise, these lesions had a similar distribution to those in the ventral cusp.

Lesions in the septal cusp of the right atrioventricular valve originated in the proximal ventricularis and spongiosa with great regularity; changes were less common in the distal third of the valve cusp. These changes extended into the ring spongiosa in 8.5% of cases. Lesions in the mural cusp of the right atrioventricular valve were mild by comparison and were confined to the distal third of the valve cusp.

In the semi-lunar valves, lesions were more severe in the aortic valve. In these valves the most common site for lesions was the ring spongiosa which was affected in 84.0% of aortic valves. These lesions extended to involve the proximal ventricularis and spongiosa in a minority of cases. Lesions were seen occasionally in the area of contact. In the pulmonary valve the degenerative changes had a similar distribution but were less severe.

The special staining methods on the valve lesions showed that they were PAS negative and gave a B metachromasia with Toluidine Blue and Azur A. This material was positive with Alcian blue and Hale's colloidal iron method. Methylation and saponification 'blocked' a positive staining reaction with Alcian Blue. The combined Alcian Blue/aldehyde fuchsin method gave a blue reaction. Sections stained by Sudan Black B were negative for fat. It was concluded that these valve lesions were rich in acid mucopolysaccharide. Das and Tashjian (1965) described the lesions of chronic valvular disease as a proliferation of connective tissue, coupled with a myxomatous change; oedema was a feature of the early lesion. Luginbühl

and Detweiler (1965) saw cells resembling fibroblasts and other unidentified cells in these affected valves. Wallace (1965) found that the valves were thickened by a mucoid connective tissue. Pirie (1965) comments upon the proliferation of the atrialis coupled with a mucinous degeneration of the spongiosa and fibrosa. These authors' observations are in broad agreement with the findings in this study but they failed to differentiate between ageing changes and the degenerative lesions of chronic valvular disease (Whitney 1967).

The effects of these valve lesions upon cardiac function vary according to the extent of the valve damage and the location of the affected valve. It is clear that the left atrioventricular valve is the valve that is most frequently and seriously affected. Both the ventral and dorsal cusps show lesions with an equal frequency and severity. These valve lesions originate at two particular sites within the valves. The spongiosa lesions originate in the triangular areas opposite to the origins of the chordae tendineae. These foci of mucoid degeneration increase in size and give rise to the nodular Type I and II lesions on the outflow surface of the valve cusp. These nodules continue to increase in size by extension into the spongiosa, and later coalesce with neighbouring nodules to give rise to the plaque-like thickenings characteristic of the Type III and IV lesions. As these changes occur with equal frequency in the dorsal and ventral cusps, the nodules vary in size and shape and they are randomly distributed over the areas of apposition; leaking occurs between the valves during systole. Another feature of the Type IV lesion was the contraction and shortening of the valve cusp that occurred. This feature, coupled with incorporation of the proximal portions of the chordae tendineae, resulted in an inability to close the valve in the central pathway during systole. Both these defects have been described by Sheridan (1967) as types of valvular insufficiency.

The second site of origin of lesions lies in the ventricularis, fibrosa and the closely related chordae tendineae. In these structures, there is a progressive destruction of the fibrous 'backbone' of the valve cusp and supporting chordae tendineae. Ultimately a point is reached when these structures cannot resist systolic pressure and the valve cusp 'balloons' into the left atrium during systole. The fibrosa and chordae tendineae stretch to allow this 'ballooning' to take place. As a result, there is a reflux of blood through the lateral oblique pathways into the left atrium. On occasion, severely affected chordae tendineae may rupture with disastrous results (Ettinger and Buergelt 1969).

The loose connective tissue of the spongiosa permits a more rapid development of mucoid degeneration than can occur in the denser fibrosa. This may account for the lower frequency of 'ballooning' degeneration (Sheridan 1967).

These changes combine to make insufficiency of the left atrioventricular valve the most common cardiac defect of the dog, a view supported by a number of authors (Detweiler 1958; Joshua 1959; Das and Tashjian 1965; Pirie 1965).

In the right atrioventricular valve there are profound differences in the frequency and severity of lesions in the two cusps. The septal cusp shows a mucoid degeneration of the proximal ventricularis, fibrosa and spongiosa which rapidly leads to a weakening of the proximal fibrosa and a 'ballooning' degeneration during systole. Lesions are less common in the area of contact and the characteristic, nodular Type I and II lesions are rarely observed in this cusp. Lesions are less frequent in the mural cusp and these lesions are mild in character. Observation has shown that the major responsibility for closure of the right atrioventricular orifice rests on the mural cusp, the septal cusp contributing little to the closure (Fig 2). Therefore, the low frequency of lesions in the larger of the two cusps enables the mural cusp to accommodate to defects in the septal

cusp. As a result, primary insufficiency of the right atrioventricular valve is uncommon. This view is supported by Detweiler (1958) who states that insufficiency of the right atrioventricular valve is usually secondary and is associated with the terminal stages of a congestive heart failure.

In the semi-lunar valves, the frequency and severity of chronic valvular disease is lower. The most common site lies in the ring spongiosa and these changes may extend into the proximal spongiosa of the valve cusp in some cases. Lesions may also be seen in the area of apposition. In those few cases where severe lesions were seen, aortic stenosis and insufficiency was apparent due to the thickening of the valve cusps and ring spongiosa.

To summarise, the principle clinical consequence of chronic valvular disease is an insufficiency of the left atrioventricular valve. Changes may be detected in the ventricularis of dogs under six months old. These lesions develop progressively in the months and years that follow until a point is reached when a valvular insufficiency developed but cardiac compensation maintained the cardiac output. There was evidence of insufficiency of the left atrioventricular valve in 33.0% of cases but only 6.0% of these showed signs of congestive heart failure. Cardiac compensation was effective in 27.0% of cases. Primary insufficiency of the right atrioventricular valve was not seen in this series but Sheridan (1967) found such an insufficiency in 10.0% of his cases.

Where the reflux of blood from an insufficient valve strikes the atrial endocardium, a fibro-elastic proliferation was seen similar to that described by Das and Tashjian (1965).

The determination of the aetiology of chronic valvular disease presents many problems. One factor which emerges strongly from this study is the age-dependence of these lesions. Both in the dog and Man the normal ageing changes are characterised by the development of fibro-elastic plaques

in the atrialis and an elastic and collagen proliferation in the spongiosa opposite to the origins of the chordae tendineae (McMillan and Lev 1964; Pomerance 1966; Whitney 1967; Pomerance and Whitney 1970). McMillan and Lev (1964) also described a fibro-elastic thickening of sub-endocardium of the proximal chordae tendineae of Man which has not been seen in the dog. All these changes are a consequence of mechanical forces, i.e. friction and impact, 'tensing' of the valve and turbulence acting upon the valve cusp and chordae tendineae. All these ageing changes were more severe on the left side, due to the higher systemic blood pressure (McMillan and Lev 1964).

If one accepts these interpretations of the aetiology of these ageing changes, the atrialis, certain areas of the spongiosa and the junctions between the ventricularis and the chordae tendineae, are points particularly susceptible to 'wear and tear' as they appear to require further reinforcement during life to maintain the integrity of the valve.

If we look at the points of origin of mucoid degeneration in the ventral cusp of the left atrioventricular valve of the dog (and Man?), lesions develop at these same points, i.e. in the spongiosa, opposite to the origins of the chordae tendineae, in the ventricularis at the points of origin of the chordae tendineae, and in the atrialis in the area of apposition. In the dorsal cusp, lesions develop at the same points in the atrialis and spongiosa, but in the ventricularis lesions tend to develop in the sinus pocket and proximal ventricularis. In the septal cusp of the right atrioventricular valve, lesions originate in the sinus pocket and proximal ventricularis, while lesions in the mural cusp show a similar distribution to the ventral cusp of the left atrioventricular valve. In the semi-lunar valves, lesions originate and are generally confined to the ring spongiosa; all these areas of points of particular turbulence during systole.

Spongiosa lesions develop at the points opposite to the origins of

the chordae tendineae in the ventral cusp of the left atrioventricular valve which are regions of mechanical strain and 'tensing' during systole (McMillan and Lev 1964). Lesions in the atrialis originate in the area of apposition in pre-existing fibro-elastic plaques. (Lesions are usually well developed in the spongiosa and fibrosa before lesions develop here.)

While the ageing changes may represent the normal response to increased mechanical stress in the maturing individual, mucoid degeneration may represent a failure to accommodate to these additional stresses in the dog.

These valve lesions were more severe in the left heart which could be due to the higher systemic blood pressure on the left side. There is experimental evidence that this degenerative process can be accelerated by increasing the workload on the heart. Medzel (1937) was able to produce fibrosis of the valves by long courses of pitressin. Lillihei et alia (1950) induced lesions similar to chronic valvular disease by means of arteriovenous fistulae. Altland et alia (1959) induced similar lesions by decompression and hypoxia.

It is evident that these mechanical factors are of vital importance in the aetiology of chronic valvular disease but other factors are probably involved as well. The essential lesion is a mucoid degeneration of the fibrillar elements of the valve. What circumstances result in the accumulation of an acid mucopolysaccharide in these lesions? Mucopolysaccharide accumulates in the tissues in a variety of conditions of known and unknown aetiology, for example, in granulomatous tissue, scurvy, starvation, thyroid deficiency. Mucopolysaccharide accumulates in the tissues of some animals under the influence of the sex hormones.

Conditions of unknown aetiology such as Marfan's syndrome, Erheim's medio-necrosis, the Hurler syndrome and other congenital diseases; in some tumours such as the myxoma, myxofibroma, etc. Certain toxic agents such as Sweet Pea meal, β-aminoproprionitrile and other nitriles may produce

accumulations of mucopolysaccharide in the tissues. However, none of these conditions give any real 'pointer' to the aetiology of chronic valvular disease.

This mucopolysaccharide material is derived from a variety of cells including fibrocytes, chondrocytes, osteocytes and, possibly, mast cells (Robb-Smith 1964). The degenerative lesion of chronic valvular disease is rich in fibrocytes which are, presumably, the origin of this mucopolysaccharide. Due to some defect in their function, they are unable to condense this material into collagen (Glynn 1960).

Various other proposals have been made for the aetiology of chronic valvular disease. Shouse and Meier (1956) suggested that chronic valvular disease was the chronic stage of a previous bacterial endocarditis. This is not so, for the lesion is clearly degenerative and not post-inflammatory. Secondly, this proposal implies a high incidence of bacterial endocarditis but only two cases (1.0%) were seen in this series. Bacterial endocarditis has a low frequency in the dog (Munich 1935; Detweiler et alia 1960; Stunzi 1962; Das et alia 1965; Sheridan 1967; Whitney 1967; Pomerance and Whitney 1970).

Bohmig and Klein (1953) demonstrated the ready allergic reaction of the sub-endothelium of the rabbit heart valve. Detweiler (1959) suggested that a similar antigen-antibody reaction may precipitate the lesions of chronic valvular disease in the dog. There is no evidence to confirm or refute this hypothesis.

Angrist et alia (1960) showed how various forms of non-specific stress may initiate valve lesions in the rat. These lesions were similar to chronic valvular disease in the dog (Angrist 1965). The lesions in the rat were a consequence of a disturbed collagen metabolism (Angrist and Marquise 1954). Non-specific stress may be a factor in the aetiology of chronic valvular disease.

The aetiology of chronic valvular disease is unresolved but it appears that ageing and mechanical stresses are intimately involved in its development.

In comparing these valve changes in the dog with similar changes in Man, it is clear that there are many similarities. There is little doubt that the normal ageing changes are similar in both species and that their aetiology is the same (Pomerance and Whitney 1970).

The atheromatous condition of the mitral and aortic valves of Man (Ashworth 1946; McMillan and Lev 1964; Pomerance 1967) has no counterpart in the dog where lipid deposition was confined to the spongiosa.

Mucoid degeneration of the heart valves occurs in both species but is more prevalent in the dog. In Man, 7.0% of patients forty-five years and over showed lesions in their heart valves compared with 94.2% of dogs nine years and over. Severe mucoid degeneration was seen in 1.0% of human beings and 59.0% of dogs (Pomerance and Whitney 1970). If mechanical factors are significant in the aetiology of this condition, it would appear that the human heart valve is better able to accommodate to additional mechanical stresses than the heart valves of the dog.

In the study of the changes in the blood vessels of the dog the generic name 'arteriosclerosis' has been employed after Lobstein (1933). Basically, two different lesions were seen, one a fibrous intimal plaque and secondly, a hyalin degeneration of the intima and media. Both of these changes originated from a basic intimal lesion which was seen in all the vessels that were examined.

The fibrous intimal plaque could be seen by the naked eye in the posterior aorta where they appeared as greyish-white elevations of the intima. These changes were more severe in the abdominal than in the thoracic portion of this vessel. A total of 50 aortae were subjected to gross staining with Sudan IV (Holman et alia 1958) but only one showed a doubtful positive

for sub-intimal fat. Hindaway (1959) found one positive reaction in 315 dogs. It is concluded that lipid is uncommon in aortic arteriosclerosis in the dog. Histological examination of these aortae revealed many more lesions at an earlier stage of development.

In an attempt to follow the pathogenesis of these lesions and to provide a basis for an epidemiological study, a grading system was devised based upon the histopathological findings in these vessels. The basic lesion and the fibrous intimal plaques were placed in Group I and were graded A-D. Grade A changes were largely confined to the internal elastic lamina but, due to the nature of the internal elastic lamina in the posterior aorta, Grade A changes were not recorded in this vessel.

Grade B changes could be readily detected in all vessels as a loose plaque, rich in smooth muscle cells and ground substance. These lesions contained a few delicate collagen or elastic fibres. The Grade B changes constituted the basic arteriosclerotic lesion and all further modifications of the arteriosclerotic process appeared to stem from these lesions. Gresham and Howard (1963) saw a similar lesion in a wide range of species, including Man.

The Grade C lesion showed an increase in the fibrillar content of the plaque. In the thoracic portion of the posterior aorta the plaques were rich in elastic fibres while similar lesions in the abdominal portion were rich in collagen fibres. In addition to these fibres, smooth muscle cells and an amorphous ground substance was also present which contained muco-polysaccharide in some cases (Lindsay et alia 1952; Davies and Reinert 1965).

Grade D lesions were confined to the abdominal portion of the posterior aorta and they were composed almost entirely of collagen fibres. Some lesions showed 'layering' when the collagen fibres were interspersed with delicate layers of elastic fibres. Smooth muscle cells and ground substance were not seen. In severely affected aortae a diffuse intimal hyperplasia was seen between neighbouring fibrous plaques. No vascularisation was

seen as described by Lüginbuhl et alia (1965).

The posterior aorta showed arteriosclerosis in 39.5% of cases compared with 29.2% (Hindaway 1959) and 35.0% (Das et alia 1965). Hindaway (1959) states that intimal lesions increase in frequency with advancing age. By using the grading system in dogs of different ages, it was possible to confirm that aortic arteriosclerosis increased in frequency and severity with advancing age. A total of 17.3% of dogs under nine years showed arteriosclerosis, while 69.4% of dogs nine years and over showed similar lesions. Grade D lesions increased from 0.8% to 18.8% in dogs nine years and over. No fibrin deposition or thrombosis of the posterior aorta was seen, nor any sub-intimal lipid. Aortic arteriosclerosis was seen in dogs at two years but Davies and Reinert (1965) saw such lesions in dogs under two years.

No gross lesions were detected in the coronary circulation although fibrous plaques were seen around the inlets to the coronary vessels in three cases. In the left coronary artery 27.0% showed arteriosclerotic lesions while 8.5% of right coronary arteries showed similar lesions. Grade A lesions were recorded in the coronary circulation but Grade D lesions were not seen in these vessels. No Grade B or C lesions were sufficiently large to obstruct these vessels and there was no lipid in these lesions and no thrombosis. A total of 19.1% of dogs under nine years showed arteriosclerosis in the left coronary artery compared with 37.6% of dogs nine years and over. Grade C lesions rose from 1.7% to 2.4% nine years and over. While lesions increase in frequency, there is little increase in severity with advancing age. In the right coronary artery, arteriosclerosis occurred in 10.5% of dogs under nine years compared with 5.9% in dogs nine years and over. Grade C lesions rose from 0.9% to 1.2% in dogs nine years and over. The overall frequency is far lower in the right coronary artery which may account for the apparent absence of an increase in frequency or severity.

Lesions were seen in the left coronary artery of one dog under one year with a congenital cardiac defect. Lesions were not seen in the right coronary artery in dogs under two years. While arteriosclerosis may be seen in dogs under one year, lesions do not generally appear until the dog reaches 1-2 years old.

In general, arteriosclerotic lesions in the extramural coronary circulation and posterior aorta have no apparent clinical significance, a view supported by Luginbühl et alia (1965).

In the coronary circulation of the dog, 64.0% of the lesions in the left coronary artery and 35.3% of lesions in the right coronary artery occurred within 1 cm of each of these vessels. In Man 70.0% of vascular occlusions occur within 4.0 cm of the coronary ostia and more than half the lesions occur in the main trunk of the vessels (Blumgart et alia 1959). While no vascular occlusion occurred in the dog, arteriosclerotic lesions occurred proximally in both species.

There was no conclusive evidence of any sex or breed predisposition to arteriosclerosis.

Arteriosclerosis in intramural coronary vessels was of two types. The basic lesion was observed and also a hyalin degeneration of some vessels. Intramural arteriosclerosis was confined to the left coronary artery. In dogs under nine years, 5.2% showed Group I arteriosclerosis compared with 5.9% nine years and over. Grade C changes occurred in 1.2% of dogs nine years and over. There was little evidence of an increase in the frequency or severity of these lesions. Indeed, only one dog showed a Grade C lesion. Luginbühl et alia (1965) described the presence of a large, sometimes multinucleate cell, with long branching cytoplasmic processes in these lesions which could not be demonstrated in this series.

The low frequency of lesions of this type in these intramural vessels was due to the fact that these same vessels were invaded by a hyalin material

at an early stage. This hyalin change was classified a Group II lesion and graded E - G according to the extent of the hyalin change within the intima and media. In the Grade E lesion, hyalin was confined to the vicinity of the internal elastic lamina. In the Grade F lesion, the infiltration was more extensive, involving both the intima and media in some cases. In other cases only one of these was involved. In the Grade G lesion the intima and media were totally replaced by hyalin with marked stenosis or total occlusion of the lumen of the vessel. The hyalin may accumulate segmentally or circumferentially, and it may give rise to large hyalin intimal plaques.

In the media of the vessel, this material originates beneath the internal elastic lamina and extends peripherally towards the adventitia. The normal structure of the vessel wall is destroyed by this process. In some cases a series of clefts were observed in severely damaged vessels which might be interpreted as evidence of recanalisation. A variety of staining techniques were employed to establish that this hyalin material in the vessel walls possessed all the staining characteristics of amyloid. No amyloid was detected in the myocardial connective tissues. The connective tissue fibres in the intima and media swell and exert pressure on the muscle fibres of the media and ultimately destroy them.

Braunmühl (1956) first described congophilic angiopathy and 'senile' plaques in old dogs. Later, Dahme (1965) stressed the similarity between these brain lesions and the lesions in the intramural coronary vessels. Luginbühl (1966) found amyloid deposits in the cerebral and/or coronary vessels of 28 of 30 dogs over twelve years old. These authors confirm the view that this hyalin material is amyloid. Small quantities of lipid material were seen in some Grade F and G lesions.

Amyloid degeneration was seen in 29.5% of cases of which 5.0% were Grade G lesions. These changes were seen in 13.0% of dogs under nine years

and in 51.7% of dogs nine years and over. Grade G lesions rose from 0.9% to 10.6% in dogs nine years and over. Amyloid degeneration increases in frequency and severity with advancing age. Detweiler and Patterson (1965) state that clinical myocardial disease is rare in dogs under nine years, after which time this diagnosis may be made in 9.0% of cases. Grade E and F lesions were not associated with any other tissue changes but Grade G lesions were associated with small areas of myocardial necrosis and fibrosis. These myocardial lesions were considered to be due to myocardial ischaemia following the stenosis or total occlusion of the vessel walls (Detweiler et alia 1960). These ischaemic areas were located in the papillary muscles and apex of the left ventricle in the innermost third of the myocardium. The injection studies showed that these areas were supplied by divisions of the ventral descending branch and the median branch of the left circumflex coronary artery. Similar degenerative lesions were occasionally seen in divisions of the anterior septal branch in the interventricular septum. It is the terminal arterioles of these vessels that show amyloid degeneration and, when the changes are severe, these result in a peripheral ischaemia and myocardial degeneration.

The vascular lesions and the associated myocardial degeneration may not occur in the same section and can only be seen in serial sections. Amyloid degeneration of considerable severity may occur without myocardial degeneration. These areas of myocardial ischaemia are small, the majority can only be detected histologically. What factors limit the frequency and extent of these areas of myocardial degeneration.

Firstly, the fact that the smaller intramural branches are affected limits the potential size of the ischaemic areas. Had these lesions been located in the larger extramural branches the effects would have been more dramatic. Secondly, the injection studies revealed the presence of many large natural anastomoses, 60-80 microns in diameter, which were capable of restoring

the blood supply or limiting the effects of myocardial ischaemia through collateral vessels. The amyloid degeneration appears to develop slowly so that these anastomses have time to develop to a point where the blood supply can be maintained despite severe vascular damage. A third point of importance is the extent of the involvement of these intramural vessels. Once a large number of vessels become involved, the collateral circulation cannot be maintained and ischaemia develops. If only a few vessels are occluded the collateral circulation can maintain the viability of the myocardium. The role of the arterioluminal vessels is uncertain but it is possible that they could help to maintain the myocardium to some extent. All these factors combine to limit the effects of severe amyloid degeneration in intramural vessels. But where myocardial ischaemia is present, the cardiac reserve is reduced.

In this series, 5.0% of 200 dogs showed evidence of severe amyloid degeneration and myocardial ischaemia. This figure compares favourably with the 5.3% in a series of 730 dogs (Das et alia 1965). Detweiler et alia (1960) found 54 cases of so-called hyalin degeneration in a series of 69 cases of cardiac disease. Detweiler and Patterson (1965) refer to a triad of changes, chronic valvular disease, intramural arteriosclerotic change and myocardial necrosis and fibrosis which they associated with a substantial percentage of cases of congestive heart failure in the dog. In this series, all 10 cases (5.0%) of amyloid degeneration of intramural vessels and myocardial ischaemia were associated with chronic valvular disease (Type III and IV in eight cases) but only one of these died of congestive heart failure. There is little doubt that myocardial necrosis and fibrosis is of some significance when associated with severe insufficiency of the left atrioventricular valve.

It is clear that arteriosclerosis in the dog is of little clinical importance. If Gresham and Howard's (1963) observations are accepted, both

the dog and Man demonstrate a similar basic arteriosclerotic lesion but here the similarity ends. Lipid and cholesterol deposition, characteristic of human atheroma, are confined to the rare cases of primary Xanthomatosis in the dog (Bloom 1946; Luginbühl et alia 1965; Head and Whitney 1967). Lipid deposition in Group I and II lesions is confined to a few cases of Grade G amyloid degeneration. No complicated lesions are seen in the dog. Another basic difference is the absence of fibrin deposition and coronary thrombosis in the dog. Where coronary occlusion occurs in the dog, the small intramural branches are involved, in Man the larger extramural branches are occluded with extensive myocardial infarction. Myocardial infarction in the dog is associated with rare cases of septic embolism. Where arteriosclerosis is of major importance in Man, it has little if any clinical significance in the dog.

Medial calcification of the posterior aorta was seen in the aortic arch in 10 cases (5%), only 1.5% of these were associated with aortic arteriosclerosis. Hindaway (1959) found 6 cases (6.5%) in a series of 92 dogs.

Hypertrophy of the left ventricle can be associated with insufficiency of the left atrioventricular valve and chronic interstial nephritis. In 7 cases (3.5%) both conditions were present in the same dog; it was difficult to apportion the responsibility for the hypertrophy of the left ventricle.

Cardiac dilatation was associated with 12 cases (6.0%) of congestive heart failure.

No myocardial infarcts were seen in this series but myocardial necrosis and fibrosis was seen in 10 cases (5.0%) in association with hyalin degeneration of intramural coronary vessels.

Myocarditis was seen in 17 cases (8.5%). Luginbühl and Detweiler (1965) saw myocarditis in 10.0% of their cases. Suppurative myocarditis

was seen in 3.0% of cases in association with a variety of purulent conditions. Non-suppurative myocarditis was seen in 5.5% of cases; often these were an incidental finding at post-mortem (Pirie 1965).

One heart base tumour and one metarstasis from a mammary adenocarcinoma was found in these 200 dogs.

Myocardial degeneration and fibrosis of the left atrium was seen in 7 cases (3.5%) in association with atrial dilatation and insufficiency of the left atrioventricular valve. Detweiler and Patterson (1965) consider these myocardial lesions to be the source of a variety of conduction defects in the dog.

Congenital cardiac defects, pulmonary stenosis, aortic stenosis and persistent right aortic arch were seen in a total of 6 dogs (3.0%). No cases of patent foramen ovale were detected.

A number of cases of pericardial effusion were observed and four cases of pericarditis of which one case of chronic constrictive pericarditis was considered to be clinically significant.

Bacterial endocarditis was seen in 2 cases (1.0%) of which one was associated with a suppurative arthritis.

From this study of the spontaneous cardiovascular conditions of the dog, one condition of prime importance emerges, namely, insufficiency of the left atrioventricular valve, due to chronic valvular disease. This condition causes valvular insufficiency in 33.0% of cases and 6.0% of these showed congestive heart failure as a result of these valve lesions.

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THE TABLES

TABLE I

The age and sex distribution of 200 unselected dogs

Age Group (years)	0-1	1-2	2-3	3-4	4-5	5-6	6-7	7-8	8-9	9-10	10-11	11-12	12-13	13-14	14-15	15-16	16-17	Total
Male	18	7	10	6	4	6	5	5	5	9	14	4	4	3	4	0	1	105
Female	11	5	8	4	3	5	4	6	3	11	10	9	9	2	4	0	1	95
Total	29	12	18	10	7	11	9	11	8	20	24	13	13	5	8	0	2	200

TABLE 2

Sex distribution by age groups of 200 unselected dogs

Age group	0-4	5-8	9-12	13-16	Total
Male	45	21	31	8	105
Female	31	18	39	7	95
Total	76	39	70	15	200

TABLE 3 The age and breed distribution of 200 unselected dogs

Age Group(years)	0-1	1-2	2-3	3-4	4-5	5-6	6-7	7-8	8-9	9-10	10-11	11-12	12-13	13-14	14-15	15-16	16-17	Total
Alsatian ..	2		1	2	1	1	1	1	1	1	1	3	1					16
Beagle ..	1	1	1						1									4
Border Collie	2	-	5			3	2	1		3	4	2	3	2	2		2	31
Border Terrier		1							1	1	1		1					5
Boxer ..			1		1	1	2	2			4							11
Bulldog ..	1																	1
Bull Mastiff											1							1
Cairn ..					1		1	1		2	1							6
Cocker Spaniel	1		1							1	1	1						5
Corgi ..	1	1		2					1		2				1			8
Cross-bred ..	5	2	1	1		2	1	3	2	3	2	2	1	2	3			30
Dachshund ..	3					2												5
Dalmatian ..								1										1
Foxhound ..									1									1
Great Dane ..										1								1
Greyhound ..	3	5	2	3	1													14
Golden Retriever									1	1				1				3
Irish Terrier					1													1
Kerry Blue ..												1						1
Labrador ..	2	1	1	1		1	2	2		2	4		2					18
Lakeland Terrier													1		1			2
Maltese Terrier					1													1
Miniature Poodle	3		1															4
Papillon ..	1																	1
Pekinese ..			1							1								2
Pomeranian ..												1						1
Scottish Terrier												1	1					2
Sealyham Terrier											1							1
Shetland Collie	1																	1
Springer Spaniel				1								1						2

TABLE 3 (Contd)

Age Group(years)	0-1	1-2	2-3	3-4	4-5	5-6	6-7	7-8	8-9	9-10	10-11	11-12	12-13	13-14	14-15	15-16	16-17	Total
Staffordshire Bull Terrier	1									2	1		2					6
West Highland White Terrier		1	2		1	1				1		1	1		1			9
Whippet ..	1																	1
Wire-haired Fox Terrier ..	1		1							1	1							4
Total ..	29	12	18	10	7	11	9	11	8	20	24	13	13	5	8	-	2	200

TABLE 4

BREED DISTRIBUTION BY AGE GROUPS

	0-4	5-8	9-12	13-16	Total
Alsatian	6	4	6	0	16
Beagle	3	1	0	0	4
Border Collie	7	6	12	6	31
Border Terrier.. ..	1	1	3	0	5
Boxer	2	5	4	0	11
Bulldog	1	0	0	0	1
Bull Mastiff	0	0	1	0	1
Cairn	1	2	3	0	6
Cocker Spaniel.. ..	2	0	3	0	5
Corgi	4	1	2	1	8
Cross-bred	9	8	8	5	30
Dachshund	3	2	0	0	5
Dalmatian	0	1	0	0	1
Foxhound	0	1	0	0	1
Great Dane	0	0	1	0	1
Greyhound	14	0	0	0	14
Golden Retriever ..	0	1	1	1	3
Irish Terrier	1	0	0	0	1
Kerry Blue	0	0	1	0	1
Labrador	5	5	8	0	18
Lakeland Terrier ..	0	0	1	1	2
Maltese Terrier ..	1	0	0	0	1
Miniature Poodle ..	4	0	0	0	4
Papillon	1	0	0	0	1
Pekinese	1	0	1	0	2
Pomeranian	0	0	1	0	1
Scottish Terrier ..	0	0	2	0	2
Sealyham	0	0	1	0	1
Shetland Collie ..	1	0	0	0	1
Springer Spaniel ..	1	0	1	0	2
Staffordshire Bull Terrier	1	0	5	0	6
West Highland White Terrier	4	1	3	1	9
Whippet	1	0	0	0	1
Wire Haired Fox Terrier	2	0	2	0	4
Total ..	76	39	70	15	200

TABLE 5

CAUSES OF DEATH OR DESTRUCTION IN 200 UNSELECTED DOGS.

System	Condition	No.	Total
ALIMENTARY	Acute gastro-enteritis	3	
	Chronic enteritis	2	
	Gastric and/or duodenal ulcers	2	
	Foreign body - small intestine	2	
	Megacolon	1	
	Anal fistula	1	
	Acute pancreatitis	2	
	Adenocarcinoma - pancreas	2	
	Hepatitis	2	
	Bile duct carcinoma	3	20
CARDIOVASCULAR	Congenital abnormalities	6	
	Bacterial endocarditis	2	
	Chronic constrictive pericarditis	1	
	Chronic valvular disease	12	
	Myocarditis	8	
	Heart base tumour	1	
	Secondary tumours	1	31
CENTRAL NERVOUS SYSTEM	Chronic encephalitis	4	
	Brain tumour	1	5
ENDOCRINE	Adrenal atrophy	1	
	Diabetes insipidus	1	
	Diabetes mellitis	8	
	Thyroid adenocarcinoma	4	14
LOCOMOTOR	Contracture of hind limbs	1	
	Disc protrusion	4	
	Fracture of femur	1	
	Rickets	2	
	Septic arthritis	2	
	Spondylitis deformans	2	
	Osteosarcoma	3	15
REPRODUCTIVE	Acute metritis	3	
	Chronic endometritis	3	
	Cystic hyperplasia prostate	1	
	Mammary adenocarcinoma	7	
	Sertoli cell tumour	2	16

TABLE 5 (Cont.)

System	Condition	No.	Total
RESPIRATORY	Acute broncho-pneumonia	7	
	Acute pulmonary oedema	1	
	Foetal atalectasis	1	
	Nocardial pleurisy	5	
	Carcinoma tonsil	1	
	Bronchogenic carcinoma	1	16
LYMPHOID	Lymphosarcoma	6	
	Thymic tumour	1	7
SYSTEMIC DISEASE	Hepatitis contagiosa canis (Rubarth's Disease)	7	
	Canine distemper	10	17
URINARY	Acute interstitial nephritis	2	
	Chronic interstitial nephritis	13	
	Pyleonephritis	1	
	Renal infarcts	2	
	Urethral obstruction	1	
	Melanosarcoma	1	
	Nephro-blastoma	2	
	Fibrosarcoma	2	24
MISCELLANEOUS	Owners' request	19	
	Other conditions	11	
	Trauma	5	35

TABLE 6 The definition of the areas of supply of the major coronary vessels.

Region of heart	Source of blood supply
Left atrium	Ascending divisions of the circumflex branch of left coronary artery.
Right atrium	Ascending divisions of right coronary artery and some vessels from the circumflex branch of the left coronary artery.
Left ventricle	Left coronary artery.
Right ventricle	Right coronary artery and right lateral divisions of ventral and dorsal descending branches and ventral septal branch of the left coronary artery.
Inter-atrial septum	Ascending divisions of circumflex branch of the left coronary artery and the right coronary artery.
Inter-ventricular septum	Ventral (anterior) and dorsal (posterior) septal branches and minor septal divisions of ventral and dorsal descending branches of the left coronary artery.
Sino-atrial node	Proximal ascending division of circumflex branch of left coronary artery and posterior ascending branch of the right coronary artery.
Atrio-ventricular node	Dorsal (posterior) septal branch of left coronary artery.
Atrio-ventricular bundle	Ventral (anterior) and dorsal (posterior) septal branches of left coronary artery.

TABLE 6 (Contd)

Region of heart	Source of blood supply
Left and right bundle branches	Ventral and dorsal septal branches of left coronary artery and other divisions of the left and right coronary arteries.
Left ventral papillary muscle	Left lateral divisions of the ventral descending branch and the marginal divisions of circumflex branch of left coronary artery.
Left dorsal papillary muscle	Marginal division of circumflex branch and left lateral and septal divisions of dorsal descending branch of left coronary artery.
Right papillary muscles	Right coronary artery divisions and the ventral septal branch and right lateral divisions of dorsal descending branch of left coronary artery.
Apex of left heart	Terminal divisions of ventral descending branch and marginal division of circumflex branch of left coronary artery.
Apex of right ventricle	Terminal divisions of ventral septal branch and right lateral and terminal divisions of dorsal descending branch of left coronary artery.

TABLE 7

The distribution, frequency and relative severity of Chronic Valvular Disease

The distribution of grades of Chronic Valvular Disease in heart valves

Grades of chronic valvular disease	Left atrio-ventricular valve	Right atrio-ventricular valve		Aortic valve	Pulmonary valve
		Septal cusp	Mural cusp		
Normal	61	95	177	186	200
Type I	31	27	20	2	-
Type II	42	46	3	5	-
Type III	43	20	-	5	-
Type IV	23	12	-	2	-

The percentage distribution of grades of chronic valvular disease in the heart valves.

Grades of chronic valvular disease	Left atrio-ventricular valve	Right atrio ventricular valve		Aortic valve	Pulmonary valve
		Septal cusp	Mural cusp		
Normal	30.5	47.5	88.5	93.0	100.0
Type I	15.5	13.5	10.0	1.0	-
Type II	21.0	23.0	1.5	2.5	-
Type III	21.5	10.0	-	2.5	-
Type IV	11.5	6.0	-	1.0	-

TABLE 8 Summary on Age Distribution of Chronic Valvular Disease

	Grade	Left atrioventricular valve	Right atrioventricular valve		Aortic valve	Pulmonary valve
			Septal Cusp	Mural Cusp		
Age of onset of mild lesions	Type I	Under 1 yr.	Under 1 yr.	3 years	2 years	-
	Type II	Under 1 yr.	1 year	11 years	3 years	-
Age of onset of advanced lesions	Type III	2 years	4 years	-	5 years	-
	Type IV	8 years	8 years +	-	10 years	-
Frequency of mild lesions in dogs under 9 years	Type I	22	15	8	1	-
	Type II	21	19	-	2	-
Frequency of mild lesions in dogs 9 years and over	Type I	9	12	12	1	-
	Type II	21	27	3	3	-
Frequency of advanced lesions in dogs under 9 years	Type III	15	7	-	2	-
	Type IV	1	1(2) +	-	-	-
Frequency of advanced lesions in dogs 9 years and over	Type III	28	13	-	3	-
	Type IV	22	10	-	2	-
Age Group 0 - 4 years inclusive	Normal	48	57	71	74	76
	Type I	11	7	5	1	-
	Type II	10	10	-	1	-
	Type III	7	1	-	-	-
	Type IV	-	(1) +	-	-	-
Age Group 5 - 8 years inclusive	Normal	8	15	36	36	39
	Type I	11	8	3	-	-
	Type II	11	9	-	1	-
	Type III	8	6	-	2	-
	Type IV	1	1	-	-	-
Age Group 9 - 12 inclusive	Normal	5	20	57	63	70
	Type I	8	11	11	1	-
	Type II	20	23	2	3	-
	Type III	23	11	-	1	-
	Type IV	14	5	-	2	-
Age Group 13 - 16 years inclusive	Normal	0	3	13	13	15
	Type I	1	1	1	-	-
	Type II	1	4	1	-	-
	Type III	5	2	-	2	-
	Type IV	8	5	-	-	-

TABLE 9 The percentage summary on age distribution of chronic valvular disease.

	Grade	Left atrioventricular valve	Right atrioventricular valve		Aortic valve	Pulmonary valve
			Septal Cusp	Mural Cusp		
Percentage frequency of mild lesions in dogs under 9 years	Type I	19.1	13.0	7.0	0.9	-
	Type II	18.3	16.5	-	1.7	-
Percentage frequency of mild lesions in dogs 9 years and over	Type I	10.6	14.1	14.1	1.2	-
	Type II	24.7	31.7	3.5	3.5	-
Percentage frequency of advanced lesions in dogs under 9 years	Type III	13.0	6.1	-	1.7	-
	Type IV	0.8	1.7 +	-	-	-
Percentage Frequency of advanced lesions in dogs 9 years and over	Type III	33.0	15.3	-	3.5	-
	Type IV	25.9	11.7	-	2.3	-
Age Group 0 - 4 years inclusive	Normal	63.2	75.0	93.4	97.4	100
	Type I	14.5	9.2	6.6	1.3	-
	Type II	13.2	13.2	-	1.3	-
	Type III	9.2	1.3	-	-	-
	Type IV	-	1.3 +	-	-	-
Age Group 5 - 8 years inclusive	Normal	20.5	38.5	92.3	92.3	100
	Type I	28.2	20.5	7.7	-	-
	Type II	28.2	23.1	-	2.6	-
	Type III	20.5	15.4	-	5.1	-
	Type IV	2.6	2.6	-	-	-
Age Group 9 - 12 years inclusive	Normal	7.1	28.6	81.4	90.0	100
	Type I	11.4	15.7	15.7	1.4	-
	Type II	28.6	32.8	2.9	4.3	-
	Type III	32.8	15.7	-	1.4	-
	Type IV	20.0	7.1	-	3.0	-
Age Group 13 - 16 years inclusive	Normal	-	20.0	86.6	86.6	100
	Type I	6.7	6.7	6.7	-	-
	Type II	6.7	26.7	6.7	-	-
	Type III	33.3	13.3	-	13.3	-
	Type IV	53.3	33.3	-	-	-

Table 10A The left atrioventricular valve

The distribution of grades of Chronic Valvular Disease by age groups

Age Group	Normal Valves	Grades of Chronic Valvular Disease				Total
		Type I	Type II	Type III	Type IV	
0 - 4	48	11	10	7	0	76
5 - 8	8	11	11	8	1	39
9 - 12	5	8	20	23	14	70
13 - 16	0	1	1	5	8	15
Total	61	31	42	43	23	200

Table 10 A

The percentage distribution of grades of Chronic Valvular Disease by age groups

Age Group	Normal Valves	Grades of Chronic Valvular Disease			
		Type I	Type II	Type III	Type IV
0 - 4	63.2	14.5	13.2	9.2	0
5 - 8	20.5	28.2	28.2	20.5	2.5
9 - 12	7.1	11.4	28.6	33.0	20.0
13 - 16	0	6.7	6.7	33.3	53.3

TABLE IIA

The Left Atrioventricular Valve

The distribution of grades of Chronic Valvular Disease by age

Age (in years)	Normal valves	Grades of Chronic Valvular Disease				Total
		Type I	Type II	Type III	Type IV	
0-1	27	1	1	-	-	29
1-2	9	1	2	-	-	12
2-3	8	6	2	2	-	18
3-4	2	3	3	2	-	10
4-5	2	-	2	3	-	7
5-6	3	3	3	2	-	11
6-7	1	5	2	1	-	9
7-8	3	1	4	3	-	11
8-9	1	2	2	2	1	8
9-10	3	1	4	6	6	20
10-11	1	4	6	9	4	24
11-12	1	1	6	4	1	13
12-13	-	2	4	4	3	13
13-14	-	-	1	2	2	5
14-15	-	1	-	2	5	8
15-16	-	-	-	-	-	-
16-17	-	-	-	1	1	2
Total	61	31	42	43	23	200

TABLE II A

The left atrioventricular valve

The percentage distribution of grades of Chronic Valvular Disease by age

Age (in years)	Normal valves	Grades of Chronic Valvular Disease			
		Type I	Type II	Type III	Type IV
0-1	93.0	3.5	3.5	-	-
1-2	75.0	8.3	16.7	-	-
2-3	44.5	33.3	11.1	11.1	-
3-4	20.0	30.0	30.0	20.0	-
4-5	28.6	-	28.6	42.8	-
5-6	27.3	27.3	27.3	18.1	-
6-7	11.1	55.6	22.2	11.1	-
7-8	27.3	9.1	36.3	27.3	-
8-9	12.5	25.0	25.0	25.0	12.5
9-10	15.0	5.0	20.0	30.0	30.0
10-11	4.1	16.7	25.0	37.5	16.7
11-12	7.7	7.7	46.1	30.8	7.7
12-13	-	15.4	30.8	30.8	23.0
13-14	-	-	20.0	40.0	40.0
14-15	-	12.5	-	25.0	62.5
15-16	-	-	-	-	-
16-17	-	-	-	50.0	50.0
All ages	30.5	15.5	21.0	21.5	11.5

TABLE 12B

The Right Atrioventricular Valve - Septal Cusp

The distribution of grades of Chronic Valvular Disease by age groups

Age Group	Normal valves	Grades of Chronic Valvular Disease				Total
		Type I	Type II	Type III	Type IV	
0-4	57	7	10	1	1	76
5-8	15	8	9	6	1	39
9-12	20	11	23	11	5	70
13-16	3	1	4	2	5	15
Total	95	27	46	20	12	200

The percentage distribution of grades of Chronic Valvular Disease by age groups

Age Group	Normal valves	Grades of Chronic Valvular Disease			
		Type I	Type II	Type III	Type IV
0-4	75.0	9.2	13.2	1.3	1.3
5-8	38.5	20.5	23.1	15.4	2.5
9-12	28.6	15.7	32.8	15.7	7.1
13-16	20.0	6.7	26.7	13.3	33.3

Table 13 B The right atrioventricular valve - Septal Cusp

The distribution of grades of Chronic Valvular Disease by age

Age (in years)	Normal Valves	Grades of Chronic Valvular Disease				Total
		Type I	Type II	Type III	Type IV	
0 - 1	27	1	0	0	1	29
1 - 2	10	1	1	0	0	12
2 - 3	12	3	3	0	0	18
3 - 4	6	1	3	0	0	10
4 - 5	2	1	3	1	0	7
5 - 6	4	2	4	1	0	11
6 - 7	5	3	1	0	0	9
7 - 8	3	2	3	3	0	11
8 - 9	3	1	1	2	1	8
9 - 10	7	3	4	4	2	20
10 - 11	7	5	9	1	2	24
11 - 12	3	2	6	2	0	13
12 - 13	3	1	4	4	1	13
13 - 14	1	1	1	0	2	5
14 - 15	2	0	2	1	3	8
15 - 16	0	0	0	0	0	0
16 - 17	0	0	1	1	0	2
Total	95	27	46	20	12	200

TABLE 13 B

The Right Atrioventricular Valve - Septal Cusp

The percentage distribution of grades of Chronic Valvular Disease by age

Age in years	Normal valves	Grades of Chronic Valvular Disease			
		Type I	Type II	Type III	Type IV
0-1	93.1	3.4	-	-	3.4
1-2	83.3	8.3	8.3	-	-
2-3	66.6	16.7	16.7	-	-
3-4	60.0	10.0	30.0	-	-
4-5	28.6	14.3	42.8	14.3	-
5-6	36.4	18.2	36.4	9.0	-
6-7	55.6	33.3	11.1	-	-
7-8	27.3	18.1	27.3	27.3	-
8-9	37.5	12.5	12.5	25.0	12.5
9-10	35.0	15.0	20.0	20.0	10.0
10-11	29.2	20.8	37.5	4.2	8.3
11-12	23.0	15.4	46.2	15.4	-
12-13	23.0	7.7	30.8	30.8	7.7
13.14	20.0	20.0	20.0	-	40.0
14-15	25.0	-	25.0	12.5	37.5
15-16	-	-	-	-	-
16-17	-	-	50.0	50.0	-
All ages	47.5	13.5	23.0	10.0	6.0

TABLE 14 C

The Right Atrioventricular Vale - Mural Cusp

The distribution of grades of Chronic Valvular Disease by age groups

Age Group	Normal valves	Grades of Chronic Valvular Disease				Total
		Type I	Type II	Type III	Type IV	
0-4	71	5	-	-	-	76
5-8	36	3	-	-	-	39
9-12	57	11	2	-	-	70
13-16	13	1	1	-	-	15
Total	177	20	3	-	-	200

The percentage distribution of grades of Chronic Valvular Disease by age groups

Age Group	Normal valves	Grades of Chronic Valvular Disease			
		Type I	Type II	Type III	Type IV
0-4	93.4	6.6	-	-	-
5-8	92.3	7.7	-	-	-
9-12	81.4	15.7	2.9	-	-
13-16	86.6	6.6	6.6	-	-

Table 15C The right atrio-ventricular valve - Mural Cusp

The distribution of grades of Chronic Valvular Disease by age

Age in years	Normal Valves	Grades of Chronic Valvular Disease				
		Type I	Type II	Type III	Type IV	Total
0 - 1	29	0	0	0	0	29
1 - 2	12	0	0	0	0	12
2 - 3	18	0	0	0	0	18
3 - 4	9	1	0	0	0	10
4 - 5	3	4	0	0	0	7
5 - 6	9	2	0	0	0	11
6 - 7	9	0	0	0	0	9
7 - 8	10	1	0	0	0	11
8 - 9	8	0	0	0	0	8
9 - 10	17	3	0	0	0	20
10 - 11	21	3	0	0	0	24
11 - 12	9	2	2	0	0	13
12 - 13	10	3	0	0	0	13
13 - 14	4	1	0	0	0	5
14 - 15	7	0	1	0	0	8
15 - 16	0	0	0	0	0	0
16 - 17	2	0	0	0	0	2
TOTAL	177	20	3	0	0	200

Table 150 The right atrioventricular valve - Mural Cusp

The percentage distribution of grades of Chronic Valvular Disease by age.

Age (in years)	Normal Valves	Grades of Chronic Valvular Disease			
		Type I	Type II	Type III	Type IV
0 - 1	100.0	0	0	0	0
1 - 2	100.0	0	0	0	0
2 - 3	100.0	0	0	0	0
3 - 4	90.0	10.0	0	0	0
4 - 5	42.9	57.1	0	0	0
5 - 6	81.8	18.2	0	0	0
6 - 7	100.0	0	0	0	0
7 - 8	90.9	9.1	0	0	0
8 - 9	100.0	0	0	0	0
9 - 10	85.0	15.0	0	0	0
10 - 11	87.5	12.5	0	0	0
11 - 12	69.2	15.4	15.4	0	0
12 - 13	76.9	23.1	0	0	0
13 - 14	80.0	20.0	0	0	0
14 - 15	87.5	0	12.5	0	0
15 - 16	0	0	0	0	0
16 - 17	100.0	0	0	0	0
All ages	88.5	10.0	1.5	0	0

TABLE 16

D THE AORTIC VALVE

The distribution of grades of Chronic Valvular Disease by age groups.

Age Group	Normal valves	Grades of Chronic Valvular Disease				Total
		Type I	Type II	Type III	Type IV	
0-4	74	1	1	0	0	76
5-8	36	0	1	2	0	39
9-12	63	1	3	1	2	70
13-16	13	0	0	2	0	15
Total	186	2	5	5	2	200

The percentage distribution of grades of Chronic Valvular Disease by age groups.

Age Group	Normal valves	Grades of Chronic Valvular Disease			
		Type I	Type II	Type III	Type IV
0-4	97.4	1.3	1.3	0	0
5-8	92.3	0	2.6	5.1	0
9-12	90.0	1.4	4.3	1.4	3.0
13-16	86.6	0	0	13.3	0

TABLE 17

D THE AORTIC VALVE

The distribution of grades of Chronic Valvular Disease by age

Age (in years)	Normal valves	Grades of Chronic Valvular Disease				Total
		Type I	Type II	Type III	Type IV	
0-1	29	0	0	0	0	29
1-2	12	0	0	0	0	12
2-3	17	1	0	0	0	18
3-4	9	0	1	0	0	10
4-5	7	0	0	0	0	7
5-6	10	0	0	1	0	11
6-7	9	0	0	0	0	9
7-8	9	0	1	1	0	11
8-9	8	0	0	0	0	8
9-10	17	1	1	1	0	20
10-11	22	0	1	0	1	24
11-12	12	0	1	0	0	13
12-13	12	0	0	0	1	13
13-14	5	0	0	0	0	5
14-15	7	0	0	1	0	8
15-16	0	0	0	0	0	0
16-17	1	0	0	1	0	2
Total	186	2	5	5	2	200

TABLE 17

D THE AORTIC VALVE

The percentage distribution of grades of Chronic Valvular Disease by age.

Age (in years)	Normal valves	Grades of Chronic Valvular Disease			
		Type I	Type II	Type III	Type IV
0-1	100.0	0	0	0	0
1-2	100.0	0	0	0	0
2-3	94.4	5.6	0	0	0
3-4	90.0	0	10.0	0	0
4-5	100.0	0	0	0	0
5-6	90.9	0	0	9.1	0
6-7	100.0	0	0	0	0
7-8	81.8	0	9.1	9.1	0
8-9	100.0	0	0	0	0
9-10	85.0	5.0	5.0	5.0	0
10-11	91.7	0	4.2	0	4.2
11-12	92.3	0	7.7	0	0
12-13	92.3	0	0	0	7.7
13-14	100.0	0	0	0	0
14-15	87.5	0	0	12.5	0
15-16	0	0	0	0	0
16-17	50.0	0	0	50.0	0
All ages	93.0	1	2.5	2.5	1

TABLE 18

A THE LEFT ATRIOVENTRICULAR VALVE

The sex distribution of grades of chronic valvular disease

Sex	Normal valves	Grades of Chronic Valvular Disease				Castrated	Total
		Type I	Type II	Type III	Type IV		
Male	31	16	26	17	15	-	105
Female	29	14	14	24	8	6	95
TOTAL	60	30	40	41	23	6	200

The percentage sex distribution of grades of chronic valvular disease

Sex	Normal valves	Grades of Chronic Valvular Disease			
		Type I	Type II	Type III	Type IV
Male	29.5	15.2	24.8	16.2	14.3
Female	32.6	15.7	15.7	27.0	9.0

TABLE 19

A THE LEFT ATRIOVENTRICULAR VALVE

The sex distribution of grades of chronic valvular disease by age.

Age Group	Sex	Normal valves	Grades of Chronic Valv. Disease				Castrated	Total
			Type I	Type II	Type III	Type IV		
Under 9 years	M	31	13	15	6	1	-	66
	F	24	8	6	9	0	2	49
9 years and over	M	0	3	11	11	14	-	39
	F	5	6	8	15	8	4	46

The percentage sex distribution of grades of chronic valvular disease by age.

Age group	Sex	Normal valves	Grades of Chronic Valvular Disease			
			Type I	Type II	Type III	Type IV
Under 9 years	M	46.9	19.9	22.7	9.0	1.5
	F	51.1	17.0	12.8	19.1	0
9 years and over	M	0	7.7	28.2	28.2	35.9
	F	11.9	14.3	19.0	35.7	19.0

TABLE 20

A THE LEFT ATRIOVENTRICULAR VALVE

The sex distribution of grades of chronic valvular disease by age groups.

Age group (years)	Sex	Normal valves	Grades of Chronic Valv. Disease				Castr.	Total
			Type I	Type II	Type III	Type IV		
0-4	Male	29	6	9	1	0	-	45
	Female	19	5	1	6	0	-	31
5-8	Male	2	7	6	5	1	-	21
	Female	5	3	5	3	0	2	18
9-12	Male	0	3	11	8	9	-	31
	Female	5	5	7	13	5	4	39
13-16	Male	0	0	0	3	5	-	8
	Female	0	1	1	2	3	-	7
Total	..	60	30	40	41	23	6	200

The percentage sex distribution of grades of chronic valvular disease by age groups.

Age group (years)	Sex	Normal valves	Grades of chronic valvular disease			
			Type I	Type II	Type III	Type IV
0-4	Male	64.4	13.3	20.0	2.2	0
	Female	61.3	16.1	3.2	19.3	0
5-8	Male	9.5	33.3	28.6	23.8	4.8
	Female	31.3	18.7	31.3	18.7	0
9-12	Male	0	9.7	35.5	25.8	29.0
	Female	14.3	14.3	20.0	37.1	14.3
13-16	Male	0	0	0	37.5	62.5
	Female	0	14.3	14.3	28.6	42.8

TABLE 21

A THE LEFT ATRIOVENTRICULAR VALVE

The sex distribution of grades of chronic valvular disease by age.

Age (in yrs.)	Sex	Normal valves	Grades of Chronic Valv. Disease				Castr.	Total
			Type I	Type II	Type III	Type IV		
0-1	Male	16	1	1	0	0	0	18
	Female	11	0	0	0	0	0	11
1-2	Male	5	0	2	0	0	0	7
	Female	4	1	0	0	0	0	5
2-3	Male	5	4	1	0	0	0	10
	Female	3	2	1	2	0	0	8
3-4	Male	1	1	3	1	0	0	6
	Female	1	2	0	1	0	0	4
4-5	Male	2	0	2	0	0	0	4
	Female	0	0	0	3	0	0	3
5-6	Male	1	3	1	1	0	0	6
	Female	2	0	2	1	0	0	5
6-7	Male	0	2	2	1	0	0	5
	Female	1	2	0	0	0	1	4
7-8	Male	1	0	2	2	0	0	5
	Female	1	1	2	1	0	1	6
8-9	Male	0	2	1	1	1	0	5
	Female	1	0	1	1	0	0	3
9-10	Male	0	1	3	2	3	0	9
	Female	3	0	1	4	3	0	11
10-11	Male	0	1	5	4	4	0	14
	Female	1	3	1	5	0	0	10
11-12	Male	0	0	2	1	1	0	4
	Female	1	1	2	3	0	2	9
12-13	Male	0	1	1	1	1	0	4
	Female	0	1	3	1	2	2	9
13-14	Male	0	0	0	1	2	0	3
	Female	0	0	1	1	0	0	2
14-15	Male	0	0	0	2	2	0	4
	Female	0	1	0	0	3	0	4
15-16	Male	0	0	0	0	0	0	0
	Female	0	0	0	0	0	0	0
16-17	Male	0	0	0	0	1	0	1
	Female	0	0	0	1	0	0	1
Total		60	30	40	41	23	6	200

TABLE 21

A THE LEFT ATRIOVENTRICULAR VALVE

The sex distribution of grades of chronic valvular disease by age.

Age (in years)	Sex	Normal valves	Grades of Chronic Valvular Disease			
			Type I	Type II	Type III	Type IV
0-1	Male	88.8	5.6	5.6	0	0
	Female	100.0	0	0	0	0
1-2	Male	71.4	0	28.6	0	0
	Female	80.0	20.0	0	0	0
2-3	Male	50.0	40.0	10.0	0	0
	Female	37.5	25.0	12.5	25.0	0
3-4	Male	16.6	16.6	50.0	16.6	0
	Female	25.0	50.0	0	25.0	0
4-5	Male	50.0	0	50.0	0	0
	Female	0	0	0	100.0	0
5-6	Male	16.6	50.0	16.6	16.6	0
	Female	40.0	0	40.0	20.0	0
6-7	Male	0	40.0	40.0	20.0	0
	Female	33.3	66.6	0	0	0
7-8	Male	20.0	0	40.0	40.0	0
	Female	20.0	20.0	40.0	20.0	0
8-9	Male	0	40.0	20.0	20.0	20.0
	Female	33.3	0	33.3	33.3	0
9-10	Male	0	11.1	33.3	22.2	33.3
	Female	27.3	0	9.9	36.3	27.3
10-11	Male	0	7.1	35.7	28.5	28.5
	Female	10.0	30.0	10.0	50.0	0
11-12	Male	0	0	50.0	25.0	25.0
	Female	14.3	14.3	28.6	42.8	0
12-13	Male	0	25.0	25.0	25.0	25.0
	Female	0	14.3	42.8	14.3	28.6
13-14	Male	0	0	0	33.3	66.6
	Female	0	0	50.0	50.0	0
14-15	Male	0	0	0	50.0	50.0
	Female	0	25.0	0	0	75.0
15-16	Male	0	0	0	0	0
	Female	0	0	0	0	0
16-17	Male	0	0	0	0	0
	Female	0	0	0	100.0	100.0

TABLE 22

B THE RIGHT ATRIOVENTRICULAR VALVE
Septal Cusp

The sex distribution of grades of chronic valvular disease

Sex	Normal valves	Grades of chronic valvular disease				Castrated	Total
		Type I	Type II	Type III	Type IV		
Male	50	15	20	10	10	-	105
Female	42	12	25	8	2	6	95
Total	92	27	45	18	12	6	200

The percentage sex distribution of grades of chronic valvular disease

Sex	Normal valves	Grades of chronic valvular disease			
		Type I	Type II	Type III	Type IV
Male	47.6	14.3	19.0	9.5	9.5
Female	47.2	13.5	28.1	9.0	2.2

The sex distribution of grades of chronic valvular disease by age

Age Group	Sex	Normal valves	Grades of Chronic Valvular Ds				Castrated	Total
			Type I	Type II	Type III	Type IV		
Under 9 years	M	42	10	8	4	2	-	66
	F	28	5	11	3	0	2	49
9 years and over	M	8	5	12	6	8	-	39
	F	14	7	14	5	2	4	46

The percentage sex distribution of grades of chronic valvular disease by age

Age Group	Sex	Normal valves	Grades of Chronic Valvular Disease			
			Type I	Type II	Type III	Type IV
Under 9 years	M	63.6	15.2	12.1	6.1	3.0
	F	59.6	10.6	23.4	6.4	0
9 years and over	M	20.5	12.8	30.8	15.4	20.5
	F	33.3	16.7	33.3	11.9	4.8

TABLE 23

B THE RIGHT ATRIOVENTRICULAR VALVE
Septal Cusp

The sex distribution of grades of chronic valvular disease by age groups

Age Group (years)	Sex	Normal valves	Grades of Chronic Valvular Dis. Type I	Type II	Type III	Type IV	Castrated	Total
0–4	Male	34	5	5	0	1	-	45
	Female	23	2	5	1	0	-	31
5–8	Male	8	5	3	4	1	-	21
	Female	5	3	6	2	0	2	18
9–12	Male	8	5	9	5	4	-	31
	Female	11	6	13	4	1	4	39
13–16	Male	0	0	3	1	4	-	8
	Female	3	1	1	1	1	-	7
Total		92	27	45	18	12	6	200

The percentage sex distribution of grades of chronic valvular disease by age groups

Age Group (years)	Sex	Normal valves	Grades of chronic valvular disease Type I	Type II	Type III	Type IV
0–4	Male	75.6	11.1	11.1	0	2.2
	Female	74.2	6.5	16.1	3.2	0
5–8	Male	38.1	23.8	14.3	19.0	4.8
	Female	31.3	18.7	37.5	12.5	0
9–12	Male	25.8	16.1	29.0	16.1	12.9
	Female	31.4	17.1	37.1	11.4	2.9
13–16	Male	0	0	37.5	12.5	50.0
	Female	43.0	14.3	14.3	14.3	14.3

TABLE 24

B THE RIGHT ATRIOVENTRICULAR VALVE - Septal Cusp

The sex distribution of grades of chronic valvular disease by age.

Age (in years)	Sex	Normal valves	Grades of Chronic Valvular Dis. Type I	Type II	Type III	Type IV	Cast.	Total
0-1	Male	16	1	0	0	1	0	18
	Female	11	0	0	0	0	0	11
1-2	Male	5	1	1	0	0	0	7
	Female	5	0	0	0	0	0	5
2-3	Male	8	1	1	0	0	0	10
	Female	4	2	2	0	0	0	8
3-4	Male	3	1	2	0	0	0	6
	Female	3	0	1	0	0	0	4
4-5	Male	2	1	1	0	0	0	4
	Female	0	0	2	1	0	0	3
5-6	Male	2	2	1	1	0	0	6
	Female	2	0	3	0	0	0	5
6-7	Male	3	1	1	0	0	0	5
	Female	1	2	0	0	0	1	4
7-8	Male	1	1	1	2	0	0	5
	Female	1	1	2	1	0	1	6
8-9	Male	2	1	0	1	1	0	5
	Female	1	0	1	1	0	0	3
9-10	Male	3	1	1	3	1	0	9
	Female	4	2	3	1	1	0	11
10-11	Male	3	4	4	1	2	0	14
	Female	4	1	5	0	0	0	10
11-12	Male	1	0	2	1	0	0	4
	Female	1	2	3	1	0	2	9
12-13	Male	1	0	2	0	1	0	4
	Female	2	1	2	2	0	2	9
13-14	Male	0	0	1	0	2	0	3
	Female	1	1	0	0	0	0	2
14-15	Male	0	0	2	0	2	0	4
	Female	2	0	0	1	1	0	4
15-16	Male	0	0	0	0	0	0	0
	Female	0	0	0	0	0	0	0
16-17	Male	0	0	0	1	0	0	1
	Female	0	0	1	0	0	0	1
Total		92	27	45	18	12	6	200

TABLE 24

B THE RIGHT ATRIOVENTRICULAR VALVE - Septal Cusp

The sex distribution of grades of chronic valvular disease by age.

Age (in years)	Sex	Normal valves	Grades of Chronic Valvular Disease			
			Type I	Type II	Type III	Type IV
0-1	Male	88.9	5.5	0	0	5.5
	Female	100.0	0	0	0	0
1-2	Male	71.4	14.3	14.3	0	0
	Female	100.0	0	0	0	0
2-3	Male	80.0	10.0	10.0	0	0
	Female	50.0	25.0	25.0	0	0
3-4	Male	50.0	16.6	33.3	0	0
	Female	75.0	0	25.0	0	0
4-5	Male	50.0	25.0	25.0	0	0
	Female	0	0	66.6	33.3	0
5-6	Male	33.3	33.3	16.6	16.6	0
	Female	40.0	0	60.0	0	0
6-7	Male	60.0	20.0	20.0	0	0
	Female	33.3	66.6	0	0	0
7-8	Male	20.0	20.0	20.0	40.0	0
	Female	20.0	20.0	40.0	20.0	0
8-9	Male	40.0	20.0	0	20.0	20.0
	Female	33.3	0	33.3	33.3	0
9-10	Male	33.3	11.1	11.1	33.3	11.1
	Female	36.4	18.2	27.3	9.1	9.1
10-11	Male	21.4	28.6	28.6	7.1	14.3
	Female	40.0	10.0	50.0	0	0
11-12	Male	25.0	0	50.0	25.0	0
	Female	14.3	28.6	42.8	14.3	0
12-13	Male	25.0	0	50.0	0	25.0
	Female	28.6	14.3	28.6	28.6	0
13-14	Male	0	0	33.3	0	66.6
	Female	50.0	50.0	0	0	0
14-15	Male	0	0	50.0	0	50.0
	Female	50.0	0	0	25.0	25.0
15-16	Male	0	0	0	0	0
	Female	0	0	0	0	0
16-17	Male	0	0	0	100.0	0
	Female	0	0	100.0	0	0

TABLE 26

C THE RIGHT ATRIOVENTRICULAR VALVE - MURAL CUSP

The sex distribution of grades of chronic valvular disease

Sex	Normal valves	Grades of chronic valvular disease				Castrated	Total
		Type I	Type II	Type III	Type IV		
Male	95	8	2	0	0	-	105
Female	78	10	1	0	0	6	95
Total	173	18	3	0	0	6	200

The percentage sex distribution of grades of chronic valvular disease

Sex	Normal valves	Grades of chronic valvular disease			
		Type I	Type II	Type III	Type IV
Male	90.5	7.6	1.9	0	0
Female	87.6	11.2	1.1	0	0

The sex distribution of grades of chronic valvular disease by age

Age Group	Sex	Normal valves	Grades of Chronic Valv. Disease				Castrated	Total
			Type I	Type II	Type III	Type IV		
Under 9 years	M	63	3	0	0	0	-	66
	F	42	5	0	0	0	2	49
9 years and over	M	32	5	2	0	0	-	39
	F	36	5	1	0	0	4	46

The percentage sex distribution of grades of chronic valvular disease by age.

Age Group	Sex	Normal valves	Grades of Chronic Valvular Disease			
			Type I	Type II	Type III	Type IV
Under 9 years	M	95.5	4.5	0	0	0
	F	89.4	10.6	0	0	0
9 years and over	M	82.1	12.8	5.1	0	0
	F	85.7	11.9	2.4	0	0

TABLE 26

C THE RIGHT ATRIOVENTRICULAR VALVE - MURAL CUSP

The sex distribution of grades of chronic valvular disease by age groups.

Age Group (years)	Sex	Normal valves	Grades of chronic valv. disease				Castrated	Total
			Type I	Type II	Type III	Type IV		
0-4	Male	42	3	0	0	0	-	45
	Female	29	2	0	0	0	-	31
5-8	Male	21	0	0	0	0	-	21
	Female	13	3	0	0	0	2	18
9-12	Male	26	4	1	0	0	-	31
	Female	29	5	1	0	0	4	39
13-16	Male	6	1	1	0	0	-	8
	Female	7	0	0	0	0	-	7
Total		173	18	3	0	0	6	200

The percentage sex distribution of grades of chronic valvular disease by age groups.

Age Group (years	Sex	Normal valves	Grades of chronic valvular disease			
			Type I	Type II	Type III	Type IV
0-4	Male	93.3	6.7	0	0	0
	Female	93.6	6.4	0	0	0
5-8	Male	100.0	0	0	0	0
	Female	81.3	18.7	0	0	0
9-12	Male	83.9	12.9	3.2	0	0
	Female	82.9	14.3	2.9	0	0
13-16	Male	75.0	12.5	12.5	0	0
	Female	100.0	0	0	0	0

TABLE 27

THE RIGHT ATRIOVENTRICULAR VALVE - MURAL CUSP

The sex distribution of grades of chronic valvular disease by age.

Age (in years)	Sex	Normal valves	Grades of Chronic Valv. Disease				Castrated	Total
			Type I	Type II	Type III	Type IV		
0-1	Male	18	0	0	0	0	0	18
	Female	11	0	0	0	0	0	11
1-2	Male	7	0	0	0	0	0	7
	Female	5	0	0	0	0	0	5
2-3	Male	10	0	0	0	0	0	10
	Female	8	0	0	0	0	0	8
3-4	Male	5	1	0	0	0	0	6
	Female	4	0	0	0	0	0	4
4-5	Male	2	2	0	0	0	0	4
	Female	1	2	0	0	0	0	3
5-6	Male	6	0	0	0	0	0	6
	Female	3	2	0	0	0	0	5
6-7	Male	5	0	0	0	0	0	5
	Female	3	0	0	0	0	1	4
7-8	Male	5	0	0	0	0	0	5
	Female	4	1	0	0	0	1	6
8-9	Male	5	0	0	0	0	0	5
	Female	3	0	0	0	0	0	3
9-10	Male	9	0	0	0	0	0	9
	Female	8	3	0	0	0	0	11
10-11	Male	12	2	0	0	0	0	14
	Female	9	1	0	0	0	0	10
11-12	Male	2	1	1	0	0	0	4
	Female	6	0	1	0	0	2	9
12-13	Male	3	1	0	0	0	0	4
	Female	6	1	0	0	0	2	9
13-14	Male	2	1	0	0	0	0	3
	Female	2	0	0	0	0	0	2
14-15	Male	3	0	1	0	0	0	4
	Female	4	0	0	0	0	0	4
15-16	Male	0	0	0	0	0	0	0
	Female	0	0	0	0	0	0	0
16-17	Male	1	0	0	0	0	0	1
	Female	1	0	0	0	0	0	1
Total		173	18	3	0	0	6	200

TABLE 27

C THE RIGHT ATRIOVENTRICULAR VALVE - MURAL CUSP

The sex distribution of grades of chronic valvular disease by age.

Age (in years)	Sex	Normal valves	Grades of chronic valvular disease			
			Type I	Type II	Type III	Type IV
0-1	Male	100.0	0	0	0	0
	Female	100.0	0	0	0	0
1-2	Male	100.0	0	0	0	0
	Female	100.0	0	0	0	0
2-3	Male	100.0	0	0	0	0
	Female	100.0	0	0	0	0
3-4	Male	83.3	16.6	0	0	0
	Female	100.0	0	0	0	0
4-5	Male	50.0	50.0	0	0	0
	Female	33.3	66.6	0	0	0
5-6	Male	100.0	0	0	0	0
	Female	60.0	40.0	0	0	0
6-7	Male	100.0	0	0	0	0
	Female	100.0	0	0	0	0
7-8	Male	100.0	0	0	0	0
	Female	80.0	20.0	0	0	0
8-9	Male	100.0	0	0	0	0
	Female	100.0	0	0	0	0
9-10	Male	100.0	0	0	0	0
	Female	72.7	27.3	0	0	0
10-11	Male	85.7	14.3	0	0	0
	Female	90.0	10.0	0	0	0
11-12	Male	50.0	25.0	25.0	0	0
	Female	85.7	0	14.3	0	0
12-13	Male	75.0	25.0	0	0	0
	Female	85.7	14.3	0	0	0
13-14	Male	66.6	33.3	0	0	0
	Female	100.0	0	0	0	0
14-15	Male	75.0	0	25.0	0	0
	Female	100.0	0	0	0	0
15-16	Male	0	0	0	0	0
	Female	0	0	0	0	0
16-17	Male	100.0	0	0	0	0
	Female	100.0	0	0	0	0

TABLE 28

D THE AORTIC VALVE

The sex distribution of grades of chronic valvular disease by age.

Age Group	Sex	Normal valves	Grades of Chronic Valv. Disease				Castrated	Total
			Type I	Type II	Type III	Type IV		
Under 9 years	M	63	0	1	2	0	-	66
	F	45	1	1	0	0	2	49
9 years and over	M	34	0	2	1	2	-	39
	F	38	1	1	2	0	4	46

The percentage sex distribution of grades of chronic valvular disease by age.

Age Group	Sex	Normal valves	Grades of Chronic Valvular Disease			
			Type I	Type II	Type III	Type IV
Under 9 years	M	95.5	0	1.5	3.0	0
	F	95.7	2.1	2.1	0	0
9 years and over	M	87.2	0	5.1	2.6	5.1
	F	90.5	2.4	2.4	4.7	0

The sex distribution of grades of chronic valvular disease.

Sex	Normal valves	Grades of Chronic Valv. Disease				Castrated	Total
		Type I	Type II	Type III	Type IV		
Male	97	0	3	3	2	-	105
Female	83	2	2	2	0	6	95
Total	180	2	5	5	2	6	200

The percentage sex distribution of grades of chronic valvular disease.

Sex	Normal valves	Grades of Chronic Valvular Disease			
		Type I	Type II	Type IVI	Type IV
Male	92.3	0	2.9	2.9	1.9
Female	93.3	2.1	2.1	2.1	0

TABLE 29

D THE AORTIC VALVE

The sex distribution of grades of chronic valvular disease by age groups.

Age Group (years)	Sex	Normal valves	Grades of Chronic Valv. Disease				Castr.	Total
			Type I	Type II	Type III	Type IV		
0-4	Male	45	0	0	0	0	-	45
	Female	29	1	1	0	0	-	31
5-8	Male	18	0	1	2	0	-	21
	Female	16	0	0	0	0	2	18
9-12	Male	27	0	2	0	2	-	31
	Female	32	1	1	1	0	4	39
13-16	Male	7	0	0	1	0	-	8
	Female	6	0	0	1	0	-	7
Total		180	2	5	5	2	6	200

The percentage sex distribution of grades of chronic valvular disease by age groups.

Age Group	Sex	Normal valves	Grades of Chronic Valvular Disease			
			Type I	Type II	Type III	Type IV
0-4	Male	100.0	0	0	0	0
	Female	93.6	3.2	3.2	0	0
5-8	Male	85.7	0	4.8	9.5	0
	Female	100.0	0	0	0	0
9-12	Male	87.1	0	6.5	0	6.5
	Female	91.4	2.9	2.9	2.9	0
13-16	Male	87.5	0	0	12.5	0
	Female	85.7	0	0	14.3	0

TABLE 30

D THE AORTIC VALVE

The sex distribution of grades of chronic valvular disease by age.

Age (in years)	Sex	Normal valves	Grades of Chronic Valv. Disease				Castrated	Total
			Type I	Type II	Type III	Type IV		
0-1	Male	18	0	0	0	0	0	18
	Female	11	0	0	0	0	0	11
1-2	Male	7	0	0	0	0	0	7
	Female	5	0	0	0	0	0	5
2-3	Male	10	0	0	0	0	0	10
	Female	7	1	0	0	0	0	8
3-4	Male	6	0	0	0	0	0	6
	Female	3	0	1	0	0	0	4
4-5	Male	4	0	0	0	0	0	4
	Female	3	0	0	0	0	0	3
5-6	Male	5	0	0	1	0	0	6
	Female	5	0	0	0	0	0	5
6-7	Male	5	0	0	0	0	0	5
	Female	3	0	0	0	0	1	4
7-8	Male	3	0	1	1	0	0	5
	Female	5	0	0	0	0	1	6
8-9	Male	5	0	0	0	0	0	5
	Female	3	0	0	0	0	0	3
9-10	Male	8	0	1	0	0	0	9
	Female	9	1	0	1	0	0	11
10-11	Male	12	0	1	0	1	0	14
	Female	10	0	0	0	0	0	10
11-12	Male	4	0	0	0	0	0	4
	Female	6	0	1	0	0	2	9
12-13	Male	3	0	0	0	1	0	4
	Female	7	0	0	0	0	2	9
13-14	Male	3	0	0	0	0	0	3
	Female	2	0	0	0	0	0	2
14-15	Male	3	0	0	1	0	0	4
	Female	4	0	0	0	0	0	4
15-16	Male	0	0	0	0	0	0	0
	Female	0	0	0	0	0	0	0
16-17	Male	1	0	0	0	0	0	1
	Female	0	0	0	1	0	0	1
Total		180	2	5	5	2	6	200

TABLE 30

D THE AORTIC VALVE

The sex distribution of grades of chronic valvular disease by age

Age (in years)	Sex	Normal valves	Grades of Chronic Valvular Disease			
			Type I	Type II	Type III	Type IV
0-1	Male	100.0 100.0	0 0	0 0	0 0	0 0
1-2	Male	100.0 100.0	0 0	0 0	0 0	0 0
2-3	Male	100.0 87.5	0 12.5	0 0	0 0	0 0
3-4	Male	100.0 75.0	0 0	0 25.0	0 0	0 0
4-5	Male	100.0 100.0	0 0	0 0	0 0	0 0
5-6	Male	83.3 100.0	0 0	0 0	16.7 0	0 0
6-7	Male	100.0 100.0	0 0	0 0	0 0	0 0
7-8	Male	60.0 100.0	0 0	20.0 0	20.0 0	0 0
8-9	Male	100.0 100.0	0 0	0 0	0 0	0 0
9-10	Male	88.9 81.8	0 9.1	11.1 0	0 9.1	0 0
10-11	Male	85.7 100.0	0 0	7.1 0	0 0	7.1 0
11-12	Male	100.0 85.7	0 0	0 14.3	0 0	0 0
12-13	Male	75.0 100.0	0 0	0 0	0 0	25.0 0
13-14	Male	100.0 100.0	0 0	0 0	0 0	0 0
14-15	Male	75.0 100.0	0 0	0 0	25.0 0	0 0
15-16	Male	0 0	0 0	0 0	0 0	0 0
16-17	Male	100.0 0	0 0	0 0	0 100.0	0 0

TABLE 31

Percentage frequency and severity of grades of chronic valvular disease in the two sexes.

Valve	Left atrio-ventricular valve		Right atrioventricular valve				Aortic valve		Pulmonary valve	
			Septal cusp		Mural cusp					
	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
Normal valve	29.5	32.6	47.6	47.2	90.5	87.6	92.3	93.3	100.0	100.0
Type I	15.2	15.7	14.3	13.5	7.6	11.2	-	2.1	-	-
Type II	24.8	15.7	19.0	28.1	1.9	1.1	2.9	2.1	-	-
Type III	16.2	27.0	9.5	9.0	-	-	2.9	2.1	-	-
Type IV	14.3	9.0	9.5	2.2	-	-	1.9	-	-	-

TABLE 34

Age of onset of chronic valvular disease in the two sexes.

Grade of valve lesion	Left atrio-ventricular valve		Right atrioventricular valve				Aortic valve		Pulmonary valve	
			Septal cusp		Mural cusp					
	M	F	M	F	M	F	M	F	M	F
Type I	1	2	1	2	3	4	-	2	-	-
Type II	1	1	1	2	11	11	7	3	1	1
Type III	3	2	5	4	-	-	5	9	-	-
Type IV	8	9	8	9	-	-	10	-	-	-

TABLE 32

Percentage frequency and severity of grades of chronic valvular disease in the two sexes by age.

Age Group (years)	Sex	Left atrio-ventricular valve		Right atrioventricular valve				Aortic valve		Pulmonary valve	
				Sept. Cusp		Mural Cusp					
		I II	III IV	I II	III IV	I II	III IV	I II	III IV	I II	III IV
0–4	M	33.3	2.2	22.2	2.2	6.7	-	-	-	-	-
	F	19.3	19.3	22.6	3.2	6.4	-	6.4	-	-	-
5–8	M	61.9	28.6	38.1	23.8	-	-	4.8	9.5	-	-
	F	50.0	18.7	56.2	12.5	18.7	-	-	-	-	-
9–12	M	45.2	54.8	45.1	29.0	16.1	-	6.5	6.5	-	-
	F	34.3	51.4	54.2	14.3	17.2	-	5.8	2.9	-	-
13–16	M	-	100.0	37.5	62.5	25.0	-	-	12.5	-	-
	F	28.6	71.4	28.6	28.6	-	-	-	14.3	-	-

TABLE 33

Percentage frequency and severity of grades of chronic valvular disease in the two sexes by age

Age Group	Sex	Left atrio-ventricular valve		Right atrioventricular valve				Aortic valve		Pulmonary valve	
				Sept. cusp		Mural cusp					
		I II	III IV	I II	III IV	I II	III IV	I II	III IV	I II	III IV
Dogs under 9yr	M	42.6	10.5	27.3	9.1	4.5	-	1.5	3.0	-	-
	F	29.8	19.1	34.0	6.4	10.6	-	4.2	-	-	-
Dogs over 9 years	M	35.9	64.1	43.6	35.9	17.9	-	5.1	7.7	-	-
	F	33.3	54.7	50.0	16.7	14.3	-	4.8	4.7	-	-

TABLE 35

Mean age and sex distribution by breed

Breed	Mean Age	Sex Distribution			Total Number
		Male	Female	Castrated	
ALSATIAN	6 yrs. 5 mos.	9	7	0	16
BORDER COLLIE	8 " 6 "	16	14	1	31
BOXER	7 " 0 "	6	5	0	11
GREYHOUND	1 yr. 9 mos.	8	6	0	14
LABRADOR	6 yrs. 8 mos.	10	6	2	18
CONTROL GROUP	6 " 5 "	105	89	6	200

Mean age and percentage sex distribution by breeds.

Breed	Mean	Sex Distribution		
		Male	Female	Castrated
ALSATIAN	6 yrs. 5 mos.	56.3	43.7	0
BORDER COLLIE	8 " 6 "	51.6	45.1	3.2
BOXER	7 " 0 "	54.5	45.4	0
GREYHOUND	1 yr. 9 mos.	57.1	42.9	0
LABRADOR	6 yrs. 8 mos.	55.6	33.3	11.1
CONTROL GROUP	6 " 5 "	50.3	44.5	3.0

TABLE 36

Breed frequency and severity of chronic valvular disease

Breed (No. of individuals)	Left atrioventricular valve					Right atrioventricular valve										Aortic Valve					Pulmonary Valve				
						Septal Cusp					Mural Cusp														
	Normal	C V D				Normal	C V D				Normal	C V D				Normal	C V D				Normal	C V D			
		I	II	III	IV		I	II	III	IV		I	II	III	IV		I	II	III	IV		I	II	III	IV
ALSATIAN (16)	4	3	3	3	3	7	3	4	2	0	13	2	1	0	0	14	0	1	0	1	16	0	0	0	0
BORDER COLLIE (31)	4	12	3	10	2	11	8	10	1	1	31	0	0	0	0	29	1	0	1	0	31	0	0	0	0
BOXER (11)	2	1	3	4	1	8	0	1	1	1	10	1	0	0	0	8	0	1	1	1	11	0	0	0	0
GREYHOUND (14)	8	2	3	1	0	9	3	2	0	0	12	2	0	0	0	13	0	1	0	0	14	0	0	0	0
LABRADOR (18)	2	6	5	3	2	7	3	2	3	3	14	4	0	0	0	17	1	0	0	0	18	0	0	0	0
CONTROL (200)	61	31	42	43	23	95	27	46	20	12	177	20	3	0	0	186	2	5	5	2	200	0	0	0	0

TABLE 36

Percentage breed frequency and severity of chronic valvular disease.

Breed	Left atrioventricular valve					Right atrioventricular valve (Septal Cusp)				
	Normal	% CVD Grades				Normal	% CVD Grades			
		I	II	III	IV		I	II	III	IV
ALSATIAN (16)	25.0	18.7	18.7	18.7	18.7	43.8	18.7	25.0	12.5	0
BORDER COLLIE (31)	12.9	38.7	9.7	32.3	6.5	35.5	25.8	32.3	3.2	3.2
BOXER (11)	18.2	9.1	27.3	36.4	9.1	72.7	0	9.1	9.1	9.1
GREYHOUND (14)	57.1	14.3	21.4	7.1	0	64.3	21.4	14.3	0	0
LABRADOR (18)	11.1	33.3	27.8	16.7	11.1	38.8	16.7	11.1	16.7	16.7
CONTROL (200)	30.5	15.5	21.0	21.5	11.5	47.5	13.5	23.0	10.0	6.0

Breed	Right atrioventricular valve (Mural Cusp)					Aortic Valve					Pulmonary Valve				
	Normal	% CVD Grades				Normal	% CVD Grades				Normal	% CVD Grades			
		I	II	III	IV		I	II	III	IV		I	II	III	IV
ALSATIAN (16)	81.2	12.5	6.3	0	0	87.5	0	6.3	0	6.3	100.0	0	0	0	0
BORDER COLLIE (31)	100.0	0	0	0	0	93.6	3.2	0	3.2	0	100.0	0	0	0	0
BOXER (11)	90.9	9.1	0	0	0	72.7	0	9.1	9.1	9.1	100.0	0	0	0	0
GREY-HOUND (14)	85.7	14.3	0	0	0	92.9	0	7.1	0	0	100.0	0	0	0	0
LABRADOR (18)	77.8	22.2	0	0	0	94.4	5.6	0	0	0	100.0	0	0	0	0
CONTROL (200)	88.5	10.0	1.5	0	0	93.0	1.0	2.5	2.5	1.0	100.0	0	0	0	0

TABLE 37

Breed frequency and severity of chronic valvular disease

Breed (No. of individuals)	Left atrioventricular valve			Right atrioventricular valve						Aortic Valve			Pulmonary Valve		
				Septal Cusp			Mural Cusp								
	Normal	C V D		Normal	C V D		Normal	C V D		Normal	C V D		Normal	C V D	
		I II	III IV		I II	III IV		I II	III IV		I II	III IV		I II	III IV
ALSATIAN (16)	4	6	6	7	7	2	13	3	0	14	1	1	16	0	0
BORDER COLLIE(31)	4	15	12	11	18	2	31	0	0	29	1	1	31	0	0
BOXER (11)	2	4	5	8	1	2	10	1	0	8	1	2	11	0	0
GREYHOUND (14)	8	5	1	9	5	0	12	2	0	13	1	0	14	0	0
LABRADOR (18)	2	11	5	7	5	6	14	4	0	17	1	0	18	0	0
CONTROL (200)	61	73	66	95	73	32	177	23	0	186	7	7	200	0	0

TABLE 37

Breed frequency and severity of chronic valvular disease

Breed (No. of Individuals)	Left atrioventricular valve				Right Atrioventricular Valve Septal Cusp				Mural Cusp				Aortic Valve				Pulmonary Valve			
	Normal	% CVD Grades			Normal	% CVD Grades			Normal	% CVD Grades			Normal	% CVD Grades			Normal	% CVD Grades		
		I	II	III IV		I	II	III IV		I	II	III IV		I	II	III IV		I	II	III IV
ALSATIAN (16)	25.0	37.4		37.4	43.8	43.7		12.5	81.2	18.8		0	87.5	6.3		6.3	100.0	0		0
BORDER COLLIE (31)	12.9	48.4		38.8	35.5	58.1		6.4	100.0	0		0	93.6	3.2		3.2	100.0	0		0
BOXER (11)	18.2	36.4		45.5	72.7	9.1		18.2	90.9	9.1		0	72.7	9.1		18.2	100.0	0		0
GREYHOUND(14)	57.1	35.7		7.1	64.3	35.7		0	85.7	14.3		0	92.9	7.1		0	100.0	0		0
LABRADOR (18)	11.1	61.1		27.8	38.8	27.8		33.4	77.8	22.2		0	94.4	5.6		0	100.0	0		0
CONTROL (200)	30.5	36.5		33.0	47.5	36.5		16.0	88.5	11.5		0	93.0	3.5		3.5	100.0	0		0

TABLE 38

The age and sex distribution related to diseased body systems.

System	Mean Age	Sex distribution			Total
		Male	Female	Castrated	
Alimentary	7yrs 6mos	11	8	1	20
Cardiovascular	6yrs 1mos	18	12	1	31
Endocrine	9yrs 9mos	3	11	0	14
Locomotor	5yrs 7mos	12	2	1	15
Reproductive	9yrs 7mos	3	13	0	16
Respiratory	3yrs 11mos	8	8	0	16
Urinary	8yrs 4mos	13	10	1	24
Systemic	0yrs 11mos	12	5	0	17
Miscellaneous	6yrs 3mos	25	20	2	47
Control	6yrs 5mos	105	89	6	200

TABLE 38

The percentage age and sex distribution related to diseased body systems.

System	Mean Age	Sex Distribution		
		Male	Female	Castrated
Alimentary	7 yrs 6 mos	55.0	40.0	5.0
Cardiovascular	6 yrs 1 mos	58.1	38.7	3.2
Endocrine	9 yrs 9 mos	21.4	78.6	0
Locomotor	5 yrs 7 mos	80.0	13.3	6.6
Reproductive	9 yrs 7 mos	18.8	81.2	0
Respiratory	3 yrs 11 mos	50.0	50.0	0
Urinary	8 yrs 4 mos	54.1	41.7	4.1
Systemic	0 yrs 11 mos	70.6	29.4	0
Miscellaneous	6 yrs 3 mos	53.2	42.6	4.2
Control	6 yrs 5 mos	52.5	44.5	3.0

TABLE 39

The frequency and severity of chronic valvular disease related to diseased body systems.

System	Left atrioventricular valve					Right Atrioventricular Valve										Aortic Valve					Pulmonary Valve				
						Septal Cusp					Mural Cusp														
	N	Grades of CVD				N	Grades of CVD				N	Grades of CVD				N	Grades of CVD				N	Grades of CVD			
		I	II	III	IV		I	II	III	IV		I	II	III	IV		I	II	III	IV		I	II	III	IV
Alimentary (20)	3	7	4	3	3	4	7	4	2	3	19	1	0	0	0	18	0	1	1	0	20	0	0	0	0
Cardiovascular(31)	9	5	3	10	4	16	2	8	3	2	25	4	2	0	0	30	0	0	0	1	31	0	0	0	0
Endocrine (14)	1	2	3	5	3	1	2	8	2	1	11	2	1	0	0	13	1	0	0	0	14	0	0	0	0
Locomotor (15)	5	2	3	2	3	7	0	3	3	2	13	2	0	0	0	14	0	0	1	0	15	0	0	0	0
Reproductive (16)	1	1	7	7	0	7	3	5	1	0	14	2	0	0	0	14	0	0	1	1	16	0	0	0	0
Respiratory (16)	8	2	4	2	0	8	4	4	0	0	15	1	0	0	0	15	0	1	0	0	16	0	0	0	0
Urinary (24)	4	4	7	5	4	13	1	5	2	3	21	3	0	0	0	22	0	1	0	1	24	0	0	0	0
Systemic (17)	13	1	2	1	0	12	2	2	0	1	17	0	0	0	0	17	0	0	0	0	17	0	0	0	0
Miscellaneous (47)	17	7	9	8	6	27	6	7	7	0	42	5	0	0	0	43	1	2	1	0	47	0	0	0	0
Control (200)	61	31	42	43	23	95	27	46	20	12	177	20	3	0	0	186	2	5	5	2	200	0	0	0	0

TABLE 39

The percentage frequency and severity of chronic valvular disease related to diseased body systems

System	Left atrioventricular valve					Right atrioventricular valve Septal Cusp					Right atrioventricular valve Mural Cusp					Aortic Valve					Pulmonary Valve				
	N	% CVD				N	% CVD				N	% CVD				N	% CVD				N	% CVD			
		I	II	III	IV		I	II	III	IV		I	II	III	IV		I	II	III	IV		I	II	III	IV
Alimentary	15.0	35.0	20.0	15.0	15.0	20.0	35.0	20.0	10.0	15.0	95.0	5.0	0	0	0	90.0	0	5.0	5.0	0	100.0	0	0	0	0
Cardiovascular	29.0	16.1	9.7	32.3	12.9	51.6	6.4	25.8	9.7	6.4	80.6	12.9	6.4	0	0	96.8	0	0	0	3.2	100.0	0	0	0	0
Endocrine	7.1	14.3	21.4	35.7	21.4	7.1	14.3	57.1	14.3	7.1	78.6	14.3	7.1	0	0	92.9	7.1	0	0	0	100.0	0	0	0	0
Locomotor	33.3	13.3	20.0	13.3	20.0	46.6	0	20.0	20.0	13.3	86.6	13.3	0	0	0	93.3	0	0	6.6	0	100.0	0	0	0	0
Reproductive	6.3	6.3	43.7	43.7	0	43.7	18.7	31.3	6.3	0	87.5	12.5	0	0	0	87.5	0	0	6.3	6.3	100.0	0	0	0	0
Respiratory	50.0	12.5	25.0	12.5	0	50.0	25.0	25.0	0	0	93.7	6.3	0	0	0	93.7	0	6.3	0	0	100.0	0	0	0	0
Urinary	16.7	16.7	29.1	20.8	16.7	54.2	4.2	20.8	8.3	12.5	87.5	12.5	0	0	0	91.7	0	4.1	0	4.1	100.0	0	0	0	0
Systemic	76.5	5.9	11.7	5.9	0	70.6	11.7	11.7	0	5.9	100.0	0	0	0	0	100.0	0	0	0	0	100.0	0	0	0	0
Miscellaneous	36.2	14.9	19.2	17.0	12.7	57.4	12.8	14.9	14.9	0	89.4	10.6	0	0	0	91.5	2.1	4.2	2.1	0	100.0	0	0	0	0
Control	30.5	15.5	21.0	21.5	11.5	47.5	13.5	23.0	10.0	6.0	88.5	10.0	1.5	0	0	93.0	1.0	2.5	2.5	1.0	100.0	0	0	0	0

TABLE 40

The frequency and severity of chronic valvular disease related to diseased body systems.

Systems	Left atrioventricular valve			Right atrioventricular valve						Aortic Valve			Pulmonary Valve		
				Septal Cusp			Mural Cusp								
	N	Grades of CVD		N	Grades of CVD		N	Grades of CVD		N	Grades of CVD		N	Grades of CVD	
		I II	III IV		I II	III IV		I II	III IV		I II	III IV		I II	III IV
Alimentary (20)	3	11	6	4	11	5	19	1	0	18	1	1	20	0	0
Cardiovascular(31)	9	8	14	16	10	5	25	6	0	30	0	1	31	0	0
Endocrine (14)	1	5	8	1	10	3	11	3	0	13	1	0	14	0	0
Locomotor (15)	5	5	5	7	3	5	13	2	0	14	0	1	15	0	0
Reproductive (16)	1	8	7	7	8	1	14	2	0	14	0	2	16	0	0
Respiratory (16)	8	6	2	8	8	0	15	1	0	15	1	0	16	0	0
Urinary (24)	4	11	9	13	6	5	21	3	0	22	1	1	24	0	0
Systemic (17)	13	3	1	12	4	1	17	0	0	17	0	0	17	0	0
Miscellaneous (47)	17	16	14	27	13	7	42	5	0	43	3	1	47	0	0
Control (200)	61	73	66	95	73	32	177	23	0	186	7	7	200	0	0

TABLE 40

The percentage frequency and severity of chronic valvular disease related to diseased body systems.

Systems	Left atrio-ventricular valve			Right Atrioventricular valve						Aortic Valve			Pulmonary Valve		
				Septal Cusp			Mural Cusp								
	N	% CVD		N	% CVD		N	% CVD		N	% CVD		N	% CVD	
		I II	III IV		I II	III IV		I II	III IV		I II	III IV		I II	III IV
Alimentary	15.0	55.0	30.0	20.0	55.0	25.0	95.0	5.0	0	90.0	5.0	5.0	100.0	0	0
Cardiovascular	29.0	25.8	45.2	51.6	32.2	16.1	80.6	19.3	0	96.8	0	3.2	100.0	0	0
Endocrine	7.1	35.7	57.1	7.1	71.4	21.4	78.6	21.4	0	92.9	7.1	0	100.0	0	0
Locomotor	33.3	33.3	33.3	46.6	20.0	33.3	86.6	13.3	0	93.3	0	6.6	100.0	0	0
Reproductive	6.3	50.0	43.7	43.7	50.0	6.3	87.5	12.5	0	87.5	0	12.5	100.0	0	0
Respiratory	50.0	37.5	12.5	50.0	50.0	0	93.7	6.3	0	93.7	6.3	0	100.0	0	0
Urinary	16.7	45.8	37.5	54.2	25.0	20.8	87.5	12.5	0	91.7	4.1	4.1	100.0	0	0
Systemic	76.5	17.6	5.9	70.6	23.4	5.9	100.0	0	0	100.0	0	0	100.0	0	0
Miscellaneous	36.2	34.1	29.7	57.4	27.7	14.9	90.0	10.0	0	91.5	6.3	2.1	100.0	0	0
Control	30.5	36.5	33.0	47.5	36.5	16.0	88.5	11.5	0	93.0	3.5	3.5	100.0	0	0

TABLE 4-1

The relationship of age to diseased body systems and the frequency and severity of chronic valvular disease.

System (No.of individuals)	Mean Age	Left Atrioventr. Valve			Right Atrioventric. Valve (Septal Cusp)		
		N	% CVD		N	% CVD	
			I II	III IV		I II	III IV
Endocrine (14)	9 years 9 mos.	7.1	35.7	57.1	7.1	71.4	21.4
Reproductive (16)	9 years 7 mos.	6.3	50.0	43.7	43.7	50.0	6.3
Urinary (24)	8 years 4 mos.	16.7	45.8	37.5	54.2	25.0	20.8
Alimentary (20)	7 years 6 mos.	15.0	55.0	30.0	20.0	55.0	25.0
Control (200)	6 years 5 mos.	30.5	36.5	33.0	47.5	36.5	16.0
Miscellaneous (47)	6 years 3 mos.	36.2	34.1	29.7	57.4	27.7	14.9
Cardiovascular (31)	6 years 1 mos.	29.0	25.8	45.2	51.6	32.2	16.1
Locomotor (15)	5 years 7 mos.	33.3	33.3	33.3	46.6	20.0	33.3
Respiratory (16)	3 years11 mos.	50.0	37.5	12.5	50.0	50.0	0
Systemic (17)	0 years11 mos.	76.5	17.6	5.9	70.6	23.4	5.9

TABLE 42 - Distribution of histopathological lesions in the heart valves.

Site of Lesion	Grading	L.atriovent. valve		R.atrioven.valve		Aortic Valve	Pulmonary Valve
		Ventral	Dorsal	Septal	Mural		
Ventricularis and Fibrosa (Arterialis)	**Proximal**						
	+	10.0	26.5	27.0	3.0	5.0	5.0
	++	0	7.5	22.0	0	1.0	3.0
	+++	0	0	27.0	0	0	0
	Distal						
	+	54.0	6.5	1.0	8.0	1.0	3.0
	++	5.0	7.5	10.0	2.0	0	1.0
	+++	3.0	3.5	4.5	0	0	0
Chordae Tendineae	**Proximal**						
	+		5.5	20.0	0		
	++	-	5.5	26.5	0	-	-
	+++		1.0	1.0	0		
	Distal						
	+	43.0	25.5	2.5	19.0		
	++	22.0	17.0	5.5	1.0	-	-
	+++	8.0	1.0	1.0	0		
Spongiosa	**Proximal**						
	+			19.0	0	1.0	3.0
	++	-	-	12.5	1.0	0	0
	+++			14.5	0	0	0
	Distal						
	+	48.0	24.5	11.0	32.5	6.0	7.0
	++	7.0	18.0	4.5	8.0	5.0	
	+++	7.0	16.0	2.5	0	4.0	

TABLE 42 (Contd)

Site of lesion	Grading	L.atriovent. valve		R.atrioven.valve		Aortic valve	Pulmonary valve
		Ventral	Dorsal	Septal	Mural		
Valve tip	+	42.0	14.0	12.0	24.0	2.0	2.0
	++	19.0	19.0	6.0	11.0	4.0	0
	+++	7.0	12.0	4.5	4.0	0	0
Neutral fat (spongiosa)	+	39.0	12.0	21.0	3.0	-	-
	++	14.0	7.0	12.0	1.0		
	+++	9.0	3.0	9.0	0		
Atrialis (ventricularis)	Fibrous Plaque						
	+	42.0	7.0	19.0	22.5	8.0	7.0
	++	19.0	7.5	11.0	3.0	2.0	7.5
	+++	4.0	7.5	3.5	1.0	2.0	0
	Degeneration						
	+	10.0	5.5	8.0	12.0	6.0	5.5
	++	3.0	7.0	34.5	0	6.0	4.5
	+++	3.0	4.5	6.0	0	5.0	0
Ring Spongiosa	+	-	-	-	-	46.0	34.5
	++					28.0	7.0
	+++					10.0	1.5

TABLE 43

THE AORTA

The distribution of grades of arteriosclerosis by age groups.

Age Group	Normal	Grade A	Grade B	Grade C	Grade D
0-4	71	0	5	0	0
5-8	24	0	10	4	1
9-12	19	0	25	10	16
13-16	7	0	0	8	0
Total	121	0	40	22	17

The percentage distribution of grades of arteriosclerosis by age groups.

Age Group	Normal	Grade A	Grade B	Grade C	Grade D
0-4	93.0	0	7.0	0	0
5-8	61.6	0	25.6	10.2	2.5
9-12	27.1	0	35.7	14.3	22.9
13-16	46.6	0	0	53.3	0

The distribution of grades of arteriosclerosis by age groups.

Age Group	Normal	Grade A	Grade B	Grade C	Grade D
Under 9 years	95	0	15	4	1
Over 9 years	26	0	25	18	16

The percentage distribution of grades of arteriosclerosis by age groups.

Age Group	Normal	Grade A	Grade B	Grade C	Grade D
Under 9 years	82.6	0	13.0	3.5	0.8
Over 9 years	30.6	0	29.4	21.2	18.8

TABLE 44

The age of onset of arteriosclerotic lesions

Vessel (lesion)	Age of Onset (years)						
	Grade A	Grade B	Grade C	Grade D	Grade E	Grade F	Grade G
Posterior Aorta	-	2	5	7	-	-	-
L C A	Under 1	6	6	-	-	-	-
R C A	2	Under 1	5	-	-	-	-
I/m vessels	6	2	12	-	-	-	-
S V D	-	-	-	-	2	2	5

L C A - Left coronary artery

R C A - Right coronary artery

I/m vessels - Intramyocardial vessels

S V D - Small vessel disease

TABLE 45

THE AORTA

The percentage sex distribution of grades of arteriosclerosis

Sex	Normal	Grade A	Grade B	Grade C	Grade D
Male	59.1	0	18.1	12.2	10.5
Female	62.1	0	22.1	9.5	6.3

TABLE 46

THE AORTA

The breed distribution of grades of arteriosclerosis.

Breed	Normal	Grade A	Grade B	Grade C	Grade D
Alsatian	13	0	3	0	0
Border Collie	16	0	6	6	3
Boxer	3	0	4	1	3
Greyhound	13	0	1	0	0
Labrador	12	0	2	1	3
Others	64	0	24	14	8
Total	121	0	40	22	17

The percentage breed distribution of grades of arteriosclerosis.

Breed	Normal	Grade A	Grade B	Grade C	Grade D
Alsatian	81.2	0	18.7	0	0
Border Collie	51.6	0	19.3	19.3	9.7
Boxer	27.2	0	36.4	9.1	27.2
Greyhound	92.9	0	7.1	0	0
Labrador	66.6	0	11.1	5.6	16.6
Others	58.2	0	21.8	12.7	7.3

The sex distribution of grades of arteriosclerosis.

Sex	Normal	Grade A	Grade B	Grade C	Grade D
Male	62	0	19	13	11
Female	59	0	21	9	6
Total	121	0	40	22	17

TABLE 47

THE LEFT CORONARY ARTERY

The distribution of grades of arteriosclerosis by age groups

Age Group	Normal	Grade A	Grade B	Grade C	Grade D
0-4	68	8	0	0	0
5-8	25	9	3	2	0
9-12	44	19	6	1	0
13-16	9	5	0	1	0
Total	146	41	9	4	0

The percentage distribution of grades of arteriosclerosis by age groups

Age Group	Normal	Grade A	Grade B	Grade C	Grade D
0-4	89.5	10.5	0	0	0
5-8	64.1	23.1	7.7	5.1	0
9-12	62.9	27.1	8.6	1.4	0
13-16	60.0	33.3	0	6.6	0

The distribution of grades of arteriosclerosis by age groups

Age Group	Normal	Grade A	Grade B	Grade C	Grade D
Under 9 years	93	17	3	2	0
Over 9 years	53	24	6	2	0

The percentage distribution of grades of arteriosclerosis by age groups

Age Group	Normal	Grade A	Grade B	Grade C	Grade D
Under 9 years	80.9	14.7	2.7	1.7	0
Over 9 years	62.4	28.2	7.0	2.4	0

TABLE 48

THE LEFT CORONARY ARTERY

The sex distribution of grades of arteriosclerosis

Sex	Normal	Grade A	Grade B	Grade C	Grade D
Male	74	23	7	1	0
Female	72	18	2	3	0
Total	146	41	9	4	0

The percentage sex distribution of grades of arteriosclerosis

Sex	Normal	Grade A	Grade B	Grade C	Grade D
Male	70.5	21.9	6.6	1.0	0
Female	75.8	18.8	2.1	3.1	0

TABLE 49

THE LEFT CORONARY ARTERY

The breed distribution of grades of arteriosclerosis

Breed	Normal	Grade A	Grade B	Grade C	Grade D
Alsatian	8	5	3	0	0
Border Collie	17	12	1	1	0
Boxer	6	2	2	1	0
Greyhound	11	3	0	0	0
Labrador	13	5	0	0	0
Others	91	14	3	2	0
Total	146	41	9	4	0

The percentage distribution of grades of arteriosclerosis

Breed	Normal	Grade A	Grade B	Grade C	Grade D
Alsatian	50.0	31.3	18.7	0	0
Border Collie	54.8	38.7	3.2	3.2	0
Boxer	54.5	18.2	18.2	9.1	0
Greyhound	78.5	21.4	0	0	0
Labrador	72.2	27.7	0	0	0
Others	82.7	12.7	2.7	1.8	0

TABLE 50

THE RIGHT CORONARY ARTERY

The distribution of grades of arteriosclerosis by age groups

Age Group	Normal	Grade A	Grade B	Grade C	Grade D
0–4	71	3	2	0	0
5–8	32	3	3	1	0
9–12	66	3	0	1	0
13–16	14	0	1	0	0
Total	183	9	6	2	0

The percentage distribution of grades of arteriosclerosis by age groups

Age Group	Normal	Grade A	Grade B	Grade C	Grade D
0–4	93.4	3.9	2.6	0	0
5–8	82.3	7.6	7.6	2.6	0
9–12	94.3	4.3	0	1.4	0
13–16	93.3	0	6.6	0	0

The distribution of grades of arteriosclerosis by age groups

Age Group	Normal	Grade A	Grade B	Grade C	Grade D
Under 9 years	103	6	5	1	0
Over 9 years	80	3	1	1	0

The percentage distribution of grades of arteriosclerosis by age groups

Age Group	Normal	Grade A	Grade B	Grade C	Grade D
Under 9 years	89.5	5.2	4.4	0.9	0
Over 9 years	94.1	3.5	1.2	1.2	0

TABLE 51

THE RIGHT CORONARY ARTERY

The sex distribution of grades of arteriosclerosis

Sex	Normal	Grade A	Grade B	Grade C	Grade D
Male	92	6	6	1	0
Female	91	3	0	1	0
Total	183	9	6	2	0

The percentage sex distribution of grades of arteriosclerosis

Sex	Normal	Grade A	Grade B	Grade C	Grade D
Male	87.6	5.7	5.7	0.9	0
Female	95.8	3.2	0	1.0	0

TABLE 52

THE RIGHT CORONARY ARTERY

The breed distribution of grades of arteriosclerosis

Breed	Normal	Grade A	Grade B	Grade C	Grade D
Alsatian	12	3	1	0	0
Border Collie	27	2	2	0	0
Boxer	10	0	1	0	0
Greyhound	14	0	0	0	0
Labrador	17	0	0	1	0
Others	103	4	2	1	0
Total	183	9	6	2	0

The percentage breed distribution of grades of arteriosclerosis

Breed	Normal	Grade A	Grade B	Grade C	Grade D
Alsatian	75.0	18.7	6.3	0	0
Border Collie	87.1	6.5	6.5	0	0
Boxer	91.0	0	9.1	0	0
Greyhound	100.0	0	0	0	0
Labrador	94.4	0	0	5.5	0
Others	93.6	3.6	1.8	0.9	0

TABLE 53

THE INTRA-MYOCARDIAL VESSELS

The distribution of grades of arteriosclerosis by age groups

Age Group	Normal	Grade A	Grade B	Grade C	Grade D
0-4	73	0	3	0	0
5-8	36	2	1	0	0
9-12	66	2	1	1	0
13-16	14	1	0	0	0
Total	189	5	5	1	0

The percentage distribution of grades of arteriosclerosis by age groups

Age Group	Normal	Grade A	Grade B	Grade C	Grade D
0-4	96.1	0	3.9	0	0
5-8	92.3	5.1	2.6	0	0
9-12	94.3	2.9	1.4	1.4	0
13-16	93.3	6.6	0	0	0

The distribution of grades of arteriosclerosis by age groups

Age	Normal	Grade A	Grade B	Grade C	Grade D
Under 9 years	109	2	4	0	0
Over 9 years	80	3	1	1	0

The percentage distribution of grades of arteriosclerosis by age groups

Age	Normal	Grade A	Grade B	Grade C	Grade D
Under 9 years	94.8	1.7	3.5	0	0
Over 9 years	94.1	3.5	1.2	1.2	0

TABLE 54

THE INTRAMURAL VESSELS

The sex distribution of grades of arteriosclerosis

Sex	Normal	Grade A	Grade B	Grade C	Grade D
Male	100	3	2	0	0
Female	89	2	3	1	0

The percentage sex distribution of grades of arteriosclerosis

Sex	Normal	Grade A	Grade B	Grade C	Grade D
Male	95.2	2.9	1.9	0	0
Female	93.7	2.1	3.2	1.0	0

TABLE 58

THE INTRAMURAL VESSELS

The breed distribution of grades of arteriosclerosis

Breed	Normal	Grade A	Grade B	Grade C	Grade D
Alsatian	15	0	0	1	0
Border Collie	29	2	0	0	0
Boxer	10	1	0	0	0
Greyhound	12	0	2	0	0
Labrador	17	1	0	0	0
Others	106	1	3	0	0
Total	189	5	5	1	0

The percentage distribution of grades of arteriosclerosis

Breed	Normal	Grade A	Grade B	Grade C	Grade D
Alsatian	93.7	0	0	6.3	0
Border Collie	93.5	6.5	0	0	0
Boxer	90.9	9.1	0	0	0
Greyhound	85.7	0	14.3	0	0
Labrador	94.4	5.6	0	0	0
Others	96.4	0.9	2.7	0	0

TABLE 56

THE INTRAMURAL VESSELS

The distribution of grades of hyalin degeneration by age groups.

Age Group	Normal	Grade E	Grade F	Grade G
0-4	72	3	1	0
5-8	28	8	2	1
9-12	34	16	13	7
13-16	7	4	2	2
Total	141	31	18	10

The percentage distribution of grades of hyalin degeneration by age groups.

Age Group	Normal	Grade E	Grade F	Grade G
0-4	94.7	4.0	1.3	0
5-8	71.8	20.5	5.1	2.6
9-12	48.6	22.8	18.6	10.0
13-16	46.6	26.6	13.3	13.3

The distribution of grades of hyalin degeneration by age.

Age Group	Normal	Grade E	Grade F	Grade G
Under 9 years	100	11	3	1
Over 9 years	41	20	15	9

The percentage distribution of grades of hyalin degeneration by age groups.

Age Group	Normal	Grade E	Grade F	Grade G
Under 9 years	87.0	9.5	2.6	0.9
Over 9 years	48.2	23.5	17.6	10.6

TABLE 57

THE INTRAMURAL VESSELS

The sex distribution of hyalin degeneration

Sex	Normal	Grade E	Grade F	Grade G
Male	77	17	7	4
Female	64	14	11	6
Total	141	31	18	10

The percentage sex distribution of hyalin degeneration

Sex	Normal	Grade E	Grade F	Grade G
Male	73.3	16.2	6.6	3.8
Female	67.3	14.7	11.6	6.3

TABLE 58

THE INTRAMURAL VESSELS

SVD - The breed distribution of grades of hyalin degeneration

Breed	Normal	Grade E	Grade F	Grade G
Alsatian	9	4	1	2
Border Collie	20	7	1	3
Boxer	7	2	1	1
Greyhound	14	0	0	0
Labrador	11	1	6	0
Others	80	17	9	4
Total	141	31	18	10

The percentage breed distribution of grades of hyalin degeneration

Breed	Normal	Grade E	Grade F	Grade G
Alsatian	56.3	25.0	6.2	12.5
Border Collie	64.5	22.6	3.2	9.7
Boxer	63.6	18.2	9.1	9.1
Greyhound	100.0	0	0	0
Labrador	61.1	5.6	33.3	0
Others	72.7	15.5	8.2	3.6