DETECTION OF CARDIOVASOPATHY IN DOGS BY
QUANTITATIVE ANALYSIS OF ANGIOCARDIOGRAMS

A Thesis
Presented in Partial Fulfillment of the Requirements
for the Degree Master of Science

by
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*** *** ***

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# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACKNOWLEDGMENTS</td>
<td>ii</td>
</tr>
<tr>
<td>TABLES</td>
<td>iv</td>
</tr>
<tr>
<td>ILLUSTRATIONS</td>
<td>v</td>
</tr>
<tr>
<td>INTRODUCTION</td>
<td>1</td>
</tr>
<tr>
<td>LITERATURE REVIEW</td>
<td>3</td>
</tr>
<tr>
<td>MATERIALS AND METHODS</td>
<td>11</td>
</tr>
<tr>
<td>Normal Dogs</td>
<td>11</td>
</tr>
<tr>
<td>Diseased Dogs</td>
<td>13</td>
</tr>
<tr>
<td>Structure Measurements</td>
<td>15</td>
</tr>
<tr>
<td>Statistical Methods</td>
<td>21</td>
</tr>
<tr>
<td>Structure Size Analysis</td>
<td>22</td>
</tr>
<tr>
<td>RESULTS AND DISCUSSION</td>
<td>25</td>
</tr>
<tr>
<td>Normal Cases</td>
<td>25</td>
</tr>
<tr>
<td>Cardiovascular Defects</td>
<td>25</td>
</tr>
<tr>
<td>Patent Ductus Arteriosus</td>
<td>30</td>
</tr>
<tr>
<td>Mitral Insufficiency</td>
<td>34</td>
</tr>
<tr>
<td>Atrial Septal Defect</td>
<td>39</td>
</tr>
<tr>
<td>Ventricular Septal Defect</td>
<td>42</td>
</tr>
<tr>
<td>Aortic Stenosis</td>
<td>45</td>
</tr>
<tr>
<td>Pulmonary Stenosis</td>
<td>49</td>
</tr>
<tr>
<td>Tricuspid Insufficiency</td>
<td>53</td>
</tr>
<tr>
<td>Differential Diagnosis</td>
<td>59</td>
</tr>
<tr>
<td>COMMENTS</td>
<td>63</td>
</tr>
<tr>
<td>Importance of Ratio Analysis</td>
<td>63</td>
</tr>
<tr>
<td>Basic Shortcomings of Ratio</td>
<td>65</td>
</tr>
<tr>
<td>Hypertrophy</td>
<td>65</td>
</tr>
<tr>
<td>AH:ARW</td>
<td>65</td>
</tr>
<tr>
<td>AH:Wt</td>
<td>66</td>
</tr>
<tr>
<td>Evaluation of a Single Patient</td>
<td>73</td>
</tr>
<tr>
<td>SUMMARY AND CONCLUSIONS</td>
<td>80</td>
</tr>
<tr>
<td>BIBLIOGRAPHY</td>
<td>82</td>
</tr>
</tbody>
</table>

iii
<table>
<thead>
<tr>
<th>Table</th>
<th>Description</th>
<th>Page</th>
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<tbody>
<tr>
<td>1.</td>
<td>Survey of normal dogs</td>
<td>12</td>
</tr>
<tr>
<td>2.</td>
<td>Survey of cardiovascular diseased dogs</td>
<td>14</td>
</tr>
<tr>
<td>3.</td>
<td>Ratio analysis of normal dogs</td>
<td>26</td>
</tr>
<tr>
<td>4.</td>
<td>Correlation matrix for cardiovascular structure measurements of normal dogs</td>
<td>27</td>
</tr>
<tr>
<td>5.</td>
<td>Decisions about the ratios of diseased dogs</td>
<td>57</td>
</tr>
<tr>
<td>6.</td>
<td>Deductive decisions about the relative size of cardiovascular structures in diseased dogs</td>
<td>61</td>
</tr>
<tr>
<td>7.</td>
<td>Range of values for each ratio by categories</td>
<td>78</td>
</tr>
</tbody>
</table>
## ILLUSTRATIONS

<table>
<thead>
<tr>
<th>Figure</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Left ventricular and aortic angiocardiogram with outlined structures</td>
<td>16</td>
</tr>
<tr>
<td>2. Right ventricular and pulmonary artery angiocardiogram with outlined structures</td>
<td>18</td>
</tr>
<tr>
<td>3. Regression of the area of the heart (AH) on body weight</td>
<td>28</td>
</tr>
<tr>
<td>4. Plotted ratio values for dogs with patent ductus arteriosus</td>
<td>32</td>
</tr>
<tr>
<td>5. Plotted ratio values for dogs with mitral insufficiency</td>
<td>37</td>
</tr>
<tr>
<td>6. Plotted ratio values for dogs with atrial septal defect</td>
<td>40</td>
</tr>
<tr>
<td>7. Plotted ratio values for dogs with ventricular septal defect</td>
<td>43</td>
</tr>
<tr>
<td>8. Plotted ratio values for dogs with aortic stenosis</td>
<td>47</td>
</tr>
<tr>
<td>9. Plotted ratio values for dogs with pulmonary stenosis</td>
<td>51</td>
</tr>
<tr>
<td>10. Plotted ratio values for dogs with tricuspid insufficiency</td>
<td>55</td>
</tr>
<tr>
<td>11. Relationship between body weight and the ratio AH:Wt for dogs with pulmonary stenosis</td>
<td>69</td>
</tr>
<tr>
<td>12. Relationship between body weight and the area of the heart (AH) in dogs with pulmonary stenosis</td>
<td>71</td>
</tr>
<tr>
<td>13. Normality-abnormality distribution curve</td>
<td>74</td>
</tr>
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</table>
INTRODUCTION

Angiocardiography affords the opportunity to visualize clearly and measure various structures of the heart and great vessels in the living animal. This is an advantageous diagnostic method for two different reasons. First, it allows one to observe specific spontaneous cardiovascular lesions. The techniques for this have become established by veterinary cardiologists (6, 7, 24, 51, 53). Secondly, it provides a means to analyze quantitatively the morphologic and dynamic changes which have occurred due to the stress placed upon the heart and great vessels by the circulatory changes caused by a specific cardiac lesion. The potential value of quantitating cardiovascular measurements has been explored during recent years for human diagnostics. Parameters which have been studied angiocardiographically in a variety of heart diseases in man include: left ventricular volume (14, 19, 40, 48); left ventricular wall thickness (11, 29, 37); size of the pulmonary artery (12, 16, 31, 52); size of the aorta (2, 15).

Left ventricular aberrations due to cardiovascular diseases have received considerable attention by quantitative angiocardiographic techniques. The reason for this is because the left ventricle is an ellipsoid of revolution (1) and geometric measurements in either biplane or single plane projections are performed easily, reliably, and provide a great deal of information about
left ventricular function (8, 14, 21, 34).

In contrast, very few studies have been made of the right ventricle. It is crescent-shaped (47) with deep trabeculations, consequently, angiograms of this chamber are cumbersome for geometric analysis. An angiographic volume study of the right ventricle has been performed, however (42).

In the present era of increased needs for animal models in cardiovascular research, and with the rapid advancements in the surgical corrections of cardiac deformities in dogs, the need existed for a precise diagnostic technique for both acquired and congenital cardiovasopathies which was reliable, rapid, and economical. The recognition of this need provided the stimulus for developing a quantitative method for evaluating cardiovascular structures from angiograms to assist in the differentiation of commonly occurring spontaneous heart diseases in dogs.
LITERATURE REVIEW

The ratio of the thickness of the left to the right ventricular wall in the newborn dog was 1.036 (35). Maturation of the electrocardiographic pattern occurred in less than thirty days in dogs (27), indicating completion of physiological growth adjustment of the left ventricle. Linzbach (38) explained that during physiologic growth the number of fibers remained constant; the fibers enlarged but their length-to-width ratio remained constant. The number of fibers and nuclei were roughly the same for both ventricles. The left ventricle, however, became heavier than the right because its fibers thickened to a greater extent.

This increased ratio of left to right ventricle thickness postnatally was due to adaptation of structure to function. Immediately following birth, the ductus arteriosus closed, heralding a reversal of increased right to left ventricular pressure gradient, so that the left ventricular pressure was markedly dominant (3). Outflow resistance (R) of the left ventricle was then increased while volume flow (Q) remained constant. It holds then that left ventricular work (W) was increased in accordance with the formula \( W = QR \) (10). Increased work was accompanied by an increase in oxygen consumption. Physiological hypertrophy was then initiated according to Badeer's (4) theory that the greatest stimulus for hypertrophy was oxygen consumption per beat per unit mass of myocardium. As the myocardial mass increased progressively, the stroke metabolic rate per unit weight of myocardium gradually decreased and eventually reached a normal value. At this
point physiological hypertrophy stopped.

With the advent of a practical method of angiocardiography in 1938 (43), measurements of structures of the heart and great vessels became simple and reliable.

Hatam, Rudhe, and Wallgren (29) measured the free wall of the left ventricle using frontal plane projection angiocardiograms of children ranging from one to fifteen years of age and in whom left ventricular involvement was not present. Measurements were made in late diastole and the left ventricular wall thickness was found to correlate well with body weight, height, and body surface area.

Levine, Rachoff, and Braunwald (37), using a single plane angiocardiographic technique, found an increase in left ventricular wall thickness without an increase in chamber size in human patients with valvular or discrete subvalvular aortic stenosis. They failed to find any consistent increase in left ventricular wall thickness in a group of patients with mitral or aortic regurgitation, despite the increased chamber volume suggested by the greater diameter of the ventricle measured from the angiocardiograms.

Castellanos and Hernandez (11), using biplane angiocardiograms, measured the left ventricular free wall thickness in diastole at three different sites in an attempt to differentiate various forms of congenital aortic and sub-aortic stenotic lesions and aortic insufficiency in man. They found, that in all forms of ventricular outflow and aortic obstructive lesions, the left ventricular wall thickness was increased but differentiation of the type of lesion was not possible. The wall thickness was not observed to be increased in aortic insufficiency.
By angiocardiographic and biophysical measurements, Grant, Greene, and Bunnell (19) explained left ventricular adaptive changes characterized by the type of external ventricular work performed in various cardiovascular diseases in man. In a group of twenty-five patients, the adaptations of the left ventricle to a pressure overload (outflow obstruction) and to a volume overload (regurgitant valvular disease) were compared with the normal function of the left ventricle and its function in the presence of myocardial disease. Adaptation to a pressure overload was by concentric hypertrophy which was defined as an increase in wall thickness without chamber enlargement. Filling pressure may be raised in these normal-sized chambers. With a volume overload there was enlargement of the ventricle and a proportionate increase in wall thickness; end-diastolic pressure may be normal in these large chambers, indicating that they were not stretched by a rise in filling pressure secondary to ventricular failure. The apparent paradox of normal filling pressure in an enlarged chamber can be explained if the enlargement was attributed to a growth process similar to the conversion of an infants’ ventricle into that of an adult. This form of pathologic growth has been called eccentric hypertrophy; the concept was supported by Linzbach’s (38) morphologic work indicating that elongation of tissues take place by growth rather than by stretching. He showed that sarcomeres were added in series to lengthen the fiber. A precise definition of eccentric hypertrophy was proposed as a cavity enlargement without a change in shape or relative wall thickness. The mass of the myocardium is increased in both eccentric and concentric hypertrophy. In eccentric hypertrophy, the fiber increase
was primarily by lengthening and its width was increased in proportion to the enlargement of the chamber. In concentric hypertrophy, a marked thickening of the fibers occurred by the addition of sarcomeres in parallel, but whether lengthening or hyperplasia occurred to account for the greater circumferential epicardial length is not known. When an enlarged left ventricle was produced by adaptive hypertrophy it was probably wrong to consider this a sign of ventricular failure.

Miller and Swam (40) studied left ventricular volumes, determined angiocardioiographically, in human patients with pressure overload and volume overload of the left ventricle and compared them with normals. The left heart volumes were in the normal range in the presence of pressure overload. With volume overload, end-diastolic and end-systolic volumes were in excess of normal. The ejection fractions were constant regardless of the pressure load on the left ventricle. Therefore, stroke volume increased in chronic volume overload.

According to angiocardioiographic and necropsy appraisals, the degree (28) or the type (49) of left ventricular hypertrophy cannot be determined by electrocardiography.

 Adaptation of the right ventricle to work was described by Rushmer (47). The normal right ventricle is crescent shaped with a relatively large radius and a large surface area per unit volume. Consequently, it is particularly suited for pumping large volumes of blood efficiently. For this reason, it can adjust to chronic volume overloads without dilatation providing pulmonary resistance is low. Adaptation to chronic pressure overload was similar to
left ventricular changes. The wall thickness increased and the cavity tended toward an ellipsoidal shape with no change in volume.

Dotter and Steinberg (15, 16) measured the diameter of the main pulmonary artery, angiographically, in normal human patients and found it was increased in patients with patent ductus arteriosus, atrial septal defect, ventricular septal defect, idiopathic aneurysmal dilations, Eisenmenger complex, pulmonic stenosis, mitral stenosis, congestive heart failure, chronic pulmonary disease, and kyphoscoliosis. Measurements were not correlated to or corrected for body dimensions.

From pulmonary artery measurements on angiograms of human patients with pulmonary stenosis, van Buchem (52), observed no correlation between the degree of "poststenotic" dilatation and the gradient across the pulmonary valve. Another observer found the amount of enlargement of the pulmonary trunk did not bear a relationship to the degree of stenosis (18). Also, according to human necropsy findings, the dilatation of the pulmonary artery may be absent in both mild and severe cases of pulmonary stenosis (20). These features constitute arguments in favor of Laubry's (36) suggestion that two different congenital abnormalities exist; one being valvular stenosis and the other dilatation of the artery. Although there evidently exists a primary abnormality of the pulmonary artery, hydraulic forces may certainly contribute to the final development of the dilatation (32).

Hernandez and Castellanos (31) found that the systolic cross-sectional area of the main pulmonary artery correlated well with body surface area in normal patients and they constructed a normal linear
regression line with which to predict its size. It was increased in 84 percent of both atrial septal defects and ventricular septal defects. When pulmonary resistance was elevated in the presence of these diseases, the pulmonary artery size was markedly enlarged. In pulmonary stenosis, the main pulmonary artery size was increased in 90 percent of the cases. The increase in the size was quite variable and it was postulated that this did not depend solely upon the basis of hydraulic laws, but more importantly upon the mechanical dynamics of the blood (17, 33).

Using techniques described previously, (12, 31), the cross-sectional area of the right pulmonary artery in the majority of the cases was found to be related to the size of the main pulmonary artery in ventricular septal defects and atrial septal defects, but no relationship was found between the two vessels in pulmonary stenosis.

Dotter and Steinberg (15) measured the aortic diameter in four locations from angiograms of normal human patients. They found an increase in aortic size with increasing age, but did not attempt to correlate the aortic diameter to body size. They suggested that the progressive increase in size with age was due to the incidence of atherosclerosis in advancing age groups.

From angiograms, Arvidsson (2) measured the systolic cross-sectional areas of the ascending and descending aorta in normal children and in the following congenital heart disease cases: ventricular septal defect, atrial septal defect, pulmonary valvular stenosis, patent ductus arteriosus, ventricular septal defect with patent ductus arteriosus, tetralogy of fallot, aortic valvular stenosis, subvalvular aortic stenosis, and aortic stenosis with insufficiency. The size of
the ascending aorta was correlated to the body surface area of the normal group and all the disease groups as an aggregate (except aortic stenosis and tetralogy of Fallot) and a common regression line was constructed. He compared the slope of the regression line of the individual defects to the common regression line rather than comparing the size of the ascending aorta in each disease group separately against the normal group to determine absolute acceptance or rejection of enlargement. Therefore, a comparison of the relative enlargement of the ascending aorta within the diseases studied combined with normals was made. He concluded that:

1. The ascending aorta was definitely enlarged in aortic valvular stenosis, but not in subvalvular aortic stenosis. In the former, the explanation for poststenotic dilatation is purely hemodynamic (32), and the degree of dilatation is independent of the degree of stenosis.

2. A slight increase in aortic size was observed in Fallot's tetrad.

3. No significant enlargement was observed in the other disease groups.

The size of the descending aorta was not changed in any of the diseases. This finding is substantiated by Moss (41).

Ross (45) measured the diameter of the aorta at four sites from angiograms of dogs with patent ductus arteriosus. He compared the ratio of the ascending aorta to the descending aorta and found that the values of the diseased dogs were elevated over those of normal dogs, and he concluded that the ascending portion of the aorta is dilated in
patent ductus arteriosus. No statistical analysis was obtained.

It was reported that body surface area of normal humans correlates well with many structural dimensions of the heart and great vessels such as: left ventricular wall thickness (11, 29, 37); left ventricular volume (40); left ventricular diameter (37); main pulmonary artery size (31); right main pulmonary artery size (12); and ascending and descending aorta size (1). It was assumed that the same dimensional relationships hold true for dogs.

The hypothesis was forwarded that the analysis of a battery of pertinent ratios should permit detection of cardiovascular structure deviations and thus be useful in differentiating commonly occurring spontaneous heart diseases in dogs.
MATERIALS AND METHODS

Angiocardiograms from 16 normal dogs, 35 with various types of congenital cardiovascular defects, and 2 with acquired heart disease were studied. The cardiovascular diseases included patent ductus arteriosus (PDA), mitral insufficiency (MI), atrial septal defect (ASD), ventricular septal defect (VSD), aortic stenosis (AS), pulmonary stenosis (PS), and tricuspid insufficiency (TI). No attempt was made to subclassify any of the diseases in this evaluation. Information about the normal and diseased dogs is given in tables 1 and 2 respectively.

Normal Dogs. The selection of normal dogs was based upon the absence of detectable heart disease by auscultation, thoracic radiographs, and electrocardiographic evidence. A necropsy verification followed the angiocardiographic procedure and was negative for heart disease in all cases.

Angiocardiography was performed with the dogs in the left-to-right lateral projection. Morphine, 6 mg. per Kg., subcutaneously, was given as premedication followed in 30 minutes by sodium pentobarbital, 20 mg. per Kg., intravenously. Dogs were intubated in all cases. The contrast medium used was 75 per cent sodium diatrizoate (Hypaque) in a dosage of 1.5 ml. per Kg. of body weight. The medium was injected into the right atrium or right ventricle through a no. 8 NIH catheter during a 1.5 - 2 second period. The angiocardiograms were obtained with a single plane Sanchez-Perez
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automatic rapid cassette changer, at a rate of two exposures per second. The left ventricle was demonstrated after passage of the contrast medium through the pulmonary circulation. Opacification of both ventricles and great vessels was satisfactory throughout the series. The electrocardiogram was followed visually on an oscilloscope for detection of arrhythmias during the exposure period. If arrhythmias occurred, the injection was repeated in 5 to 10 minutes, and after fluoroscopic examination of the bladder to evaluate the urinary excretion of the contrast material.

No attempt was made to standardize this control group with regard to breed, age, sex, or weight.

**Diseased Dogs.** The angiocardiograms selected for study were obtained from clinical cases which had a single hemodynamic abnormality which was visualized clearly in the angiocardiographic series. The verification of the specific lesion obtained from the clinical records of either catheterization techniques, the surgical correction, or the necropsy report was the other restriction placed upon the selection of the cases studied. The angiocardiographic methods in the control and diseased group were similar with the exception that the selective angiocardiography of the left side in the diseased dogs was used when indicated. This resulted in the absence of right heart measurement data in those cases and accounted for many unavailable ratio observations.

The dogs with congenital cardiovascular defects ranged in age from 8 weeks to 4 years. Therefore, ventricular maturation was present in all cases. The two cases with acquired mitral
Table 2

SURVEY OF CARDIOVASCULAR DISEASED DOGS

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<thead>
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*Data Unavailable
insufficiency were 12 and 14 years of age. The clinical cases contained mostly purebred dogs of a variety of breeds. The most marked variation in this group was body weight range which spanned 1.9 to 36.4 Kg.

Structure Measurements. Lateral projection angiocardiographic exposures which corresponded to a late diastolic phase of the cardiac cycle were selected for measurement. The opacified left and right ventricles, their respective great vessels, and their free walls were outlined as illustrated in figures 1 and 2 respectively.

The left ventricular length (LVL) was a line drawn from the center of the aortic orifice to the peak of the apex. Left ventricular diameter (LVD) was drawn perpendicular to LVL at its midpoint. The indentation of the papillary muscle in the posterio-medial margin of the ventricular shadow obscured the visualization of the majority of the left ventricular border and was ignored when making linear and area measurements and the broken line was used. The left ventricular free wall thickness (LW) was measured perpendicular to a line tangent to a point estimated to be slightly above the midpoint of the ventricle. The diameter of the aorta was measured at three sites. The aortic sinus of Valsalva (svA) was measured at the point of its greatest width. The ascending aorta (aa) was measured at a point midway between the aortic valves and the brachiocephalic trunk. The descending aorta (da) measurement was made at a point slightly past the posterior margin of the base of the heart.
A left ventricular and aortic angiocardiogram showing the manner in which structural measurements were determined. LVL, left ventricular length; LVD, left ventricular diameter; LW, left ventricular free wall thickness; svA, sinus of Valsalva; aA, ascending aorta; dA, descending aorta.
Figure 2

A right ventricular and pulmonary artery angiocardiogram showing the manner in which structural measurements were determined. RV, right ventricular cavity; RW, right ventricular free wall thickness; sPA, sinus of the pulmonary artery; mPA, main pulmonary artery.
The right ventricular free wall thickness (RW) was measured perpendicular to a line tangent to a point on the epicardium which was estimated to be at a level slightly dorsal to the ventral portion of the pulmonary outflow tract. The diameter of the pulmonary artery was measured at two sites. The sinus of the pulmonary artery (SPA) was measured at the point of its greatest width. The main pulmonary artery (mPA) was measured at a point midway between the pulmonary valves and the bifurcation of the trunk.

The area of the left ventricle (ALV) and the area of the right ventricle (ARV) were obtained by planimetering the perimeter of the outlined respective ventricles. The total area of the heart silhouette (AH) was also planimetered.

Great vessel diameters were measured on a line perpendicular to their walls. In areas where the vessel wall was not straight, the measurement was made perpendicular to a line tangent to a point on the outer curvature of the vessel. With respect to dilation of aA or mPA, the measurement was taken at its greatest diameter.

Linear measurements such as diameters, lengths, and thicknesses were measured to the nearest millimeter. Areas were measured in square centimeters, and body weight (Wt) was recorded in kilograms. Two individuals duplicated the measurements independently and their error was negligible. This indicates the reproducibility and reliability of the measurement method.

These measurements were constructed into the following systematized ratio assortment which would cross reference the
the relative sizes of the structures: 1) LW:RW, 2) sPA:mPA,
3) sPA:sVA, 4) mPA:sA, 5) sVA:sA, 6) sVA:dA, 7) aA:dA,
8) AH:ALV, 9) AH:AV, 10) LVL:LW, 11) LVD:LW, 12) LVL:LVD,
13) AH:WT. Since the decision about the size of structures was
based upon the ratio of one structure to another and was therefore
a relative rather than an absolute measurement system, it was
not necessary to correct for X-ray distortion, nor was it
necessary to make corrections for body size.

Statistical Methods. All the ratios satisfied the Kolmogorov-
Smirnov test for normality.

There were enough observations (greater than 6) in the ratios
of the clinical cases of pulmonary stenosis and in the majority
of those with patent ductus arteriosus to perform Student's t test
and make a significant decision (alpha error) about the population in
the ratios of both diseases with respect to the normal population.
In the other diseases, smaller sample sizes (less than 6) were
available and decisions about the population in the ratios were
made by inductive analysis. The analysis was used to compare a
single observation, or the mean of a small number of observations,
in a manner which permits a relatively certain decision. By this
method it was determined that although a particular observation
(or the mean of a small number of observations) falls within 2
standard deviations of the mean of the normal population, if it
falls sufficiently far from the mean, it was probably not in the
normal population. The inductive decisions (P) for small sample
sizes were made using the following mathematical model:
\[
\frac{\bar{x} - \mu}{\sigma(\mu)} \geq \frac{Z_a}{\sqrt{n}}
\]

where: \( \bar{x} \) = the mean of the sample.  
\( \mu \) = the mean of the normal population  
\( \sigma(\mu) \) = the standard deviation of the normal population.  
\( Z_a \) = the \( 100 - \alpha/2 \) percent point of the \( N(0, 1) \) distribution.  
\( n \) = the number of observations in the sample.

This model implied that if the actual standard deviation difference from the normal mean for the sample being studied was greater than the estimate of the significant standard deviation difference from the normal mean, then the sample being studied was different from the normal population.

Structure Size Analysis. A conclusion about the relative size of each cardiovascular structure was formed by comparing the inductive decisions made from an appropriate set of ratios by deductive reasoning. To construct an example of this method, let us compare the ratios \( svA/dA, svA/AA \), and \( AA/dA \) in a given heart diseased dog and deduce the relative sizes of \( svA, AA, \) and \( dA \). If, for example, \( svA/dA \) was not significantly different from the corresponding mean of the normal dogs, one would then conclude that both structures \( (svA \) and \( dA) \) were either unchanged or that they were proportionally increased or decreased in size. While, on the other hand, if the
ratio svA:aA was significantly decreased, one could surmise the following: 1) if svA was decreased in size, the size of aA would be either unchanged or decreased; 2) if svA was increased in size, the size of aA would be increased proportionately greater than svA; 3) if svA was unaffected in size, the size of aA would be increased. The conclusion that aA was, in fact, significantly increased in size was verified when the ratio aA:dvA was found to be significantly increased. This decision was by virtue of obtaining significantly deviated ratios when aA was related to both svA and dvA respectively, while the relationship between svA and dvA was not significantly changed. These two latter structures were concluded to be unaltered by the heart defect.

To assist in making deductive decisions about the relative size of the cardiovascular structures, several points must be realized.

1. The size of dvA was not affected by any disease in this study (2, 41). Therefore, any significant ratio alteration involving this measurement was due to the other structure in the ratio.

2. The ratio LW:RW was a good estimate of either ventricular wall being significantly thickened when a significant alteration occurs.

When the LW thickens relative to the increased left ventricular volume (LVV) as in eccentric hypertrophy, a significant increase in LW:RW may not be observed, but assumptions about the relative thickness of the wall can be made based upon the relative changes of the left ventricular dimensions.
may not be affected by volume overload of the right ventricle unless the pulmonary resistance becomes elevated.

3. The product of LVL and LVD was a good estimate of ALV (21, 22). In this study ALV was planimetered, and the calculated estimate of ALV will help to verify the deducted estimate.

4. The determination of left ventricular volume (LVV) by single plane angiocardiography has as its major mathematical function the calculation of the area of the left ventricle in the lateral projection. The only non-constant mathematical variable with the exception of ALV in the calculation of LVV was the minor axis measurement which was perpendicular to LVL and LVD and which has a 1:1 relationship to LVD (21). Therefore, the relative size of ALV was an estimate of the relative size of LVV.

5. AH in the lateral projection was an estimate of relative heart size in some diseases studied. An increase in AH:Wt was assumed to indicate heart enlargement.
RESULTS AND DISCUSSION

Normal Cases. Sixteen dogs verified to be free from heart disease were studied. The statistical information about the ratios of pertinent cardiovascular structures of these dogs measured from angiocardiograms are shown in table 3. The majority of structural ratios appeared to have a relatively constant relationship judging from the coefficients of variation (C). C values ranged between 7 and 30 percent. 22 percent or below was considered satisfactorily low. Only one ratio (AR:ARV) was unsatisfactory.

An interrelationship among the measured cardiovascular structures and body weight existed. Table 4 shows that there was a high correlation in four or more variables for each measurement studied, with the exception of ARV and RW. This was also suggestive that the ratios of the normal dogs had a relatively constant relationship.

Figure 3 illustrates that the regression of the lateral area of the heart silhouette (cm²) on body weight (Kg.) was linear for normal dogs. For instance, it was expected for the heart to increase in size with larger dogs.

Cardiovascular Defects

Thirty-seven dogs with either pressure overload or flow overload diseases of either the left or right ventricle respectively
### Table 3

**RATIO ANALYSIS OF NORMAL DOGS**

<table>
<thead>
<tr>
<th>Ratios</th>
<th>No. of Obs.</th>
<th>Mean</th>
<th>Range</th>
<th>S.D.*</th>
<th>C (%)**</th>
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<td>1.4 - 3.0</td>
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<td>sPA:mPA</td>
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*standard deviation

**coefficient of variation
Table 4

CORRELATION MATRIX FOR CARDIOVASCULAR STRUCTURE MEASUREMENTS OF NORMAL DOGS

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\( r = \) Correlation Coefficients (\( N = 16; \) except ARV, \( N = 7 \)).
\( \alpha = \) Significance Levels: *, <.05; **, <.025; ***, <.01; ****, <.005; ***** , <.0005.
NS = Not Significant (\( \alpha > .05 \)).
Figure 3

Relationship between body weight and the planimetered area of the heart silhouette in sixteen normal dogs. Note the calculated linear regression equation.
Figure 3

\[ AH = 11.254 \times 3.811 \text{ (Wt)} \]

\[ \Gamma = 0.8630 \]

N = 16
were studied angiocardio graphically. Cardiovascular structure measurement ratios corresponding to those of the normal dog study were analyzed.

**Patent Ductus Arteriosus.** Fourteen cases were studied. Levograms were taken in all cases but one, so decisions about ratios which contained right ventricular or pulmonary artery measurements were made by inductive analysis due to small sample sizes. All structure measurements, pertinent to the left side, contained enough observations to permit decisions about the ratios to be made by the t test. The statistical details from figure 4 and table 5 were used to make deductions about cardiovascular structural sizes.

The mean of $AH:Wt$ was significantly increased ($\alpha<.01$). Since $AH$ was disproportionately increased over the variable $Wt$, heart size was concluded to be enlarged.

The mean of $LW:RW$ was not significantly different from the mean of the normal dogs. Since this study includes only dogs with single defects and would affect the ventricular wall of just one side of the heart, the conclusion can be made that neither the left nor the right ventricular wall thickness was altered.

Left ventricular diameter and left ventricular length were both increased in size since $LW$ was concluded to be relatively unchanged in size and the means of $LVD: LW$ and $LVL: LW$ were significantly increased ($\alpha<.01$ and $\alpha<.02$ respectively). $LVD$ was
increased proportionately more than LVL since there was a significant decrease ($\alpha < .05$) in the mean of LVL:LVD with respect to the corresponding mean ratio of the normal dogs. The area of the left ventricle was concluded to be significantly increased for two reasons. First the product of LVL and LVD was an estimate of ALV, and since both LVL and LVD were significantly increased, it holds that ALV was significantly increased. Secondly, the mean of AH:ALV was significantly less than the mean of the same ratio for normal dogs. Since AH was already established to be increased, then ALV was deduced as being increased in size. LVV was increased since it was an estimate of the relative size of ALV.

The mean of svA:dA was not significantly different from the mean of the normal dogs. Since dA has been described to be unaffected by any of the diseases studied, svA was concluded to be unaffected. In the ratios svA:aA and aA:dA, the mean of the former was significantly decreased ($\alpha < .01$) while the mean of the latter was significantly increased ($\alpha < .01$). Therefore, aA was deduced as being significantly increased in size.

The mean of sPA:svA was significantly increased ($P < .05$). This indicates, by deduction, that sPA was significantly increased in size since svA was not affected. The ratio mPA:aA was not significantly different from the mean of the normal dogs. Since aA was determined to be significantly increased in size, it holds that mPA was also significantly increased in size. The mean of sPA:mPA was not significantly different from the normal dogs. Therefore, both sPA and mPA were deduced as being significantly increased in size.
Figure 4

Ratio values for each dog with patent ductus arteriosus are plotted within the individual ratio variables. A statistical decision about the mean of the aggregate of the plotted values with respect to the mean of the normal dogs for each ratio variable is indicated.
Figure 4

PATENT DUCTUS ARTERIOSUS

AH: Wt

sPA:svA

LVL:LW LVD:LW

AH:ALV

AH:ARV

LWL:LV

mPA:aA svA:aA

svA:dA

aA:dA

sPA:svA

LW:RW

a<.01

P<.05

NS

a<.01

NS

a<.01

NS

a<.01

NS

a<.01

a<.01

a<.01

α = SIGNIFICANT DECISION (α ERROR)
P = INDUCTIVE DECISION
NS = NOT SIGNIFICANT
* = DATA UNAVAILABLE
--- = NORMAL MEAN
--- = TWO STANDARD DEVIATIONS FROM NORMAL MEAN
The important deductive findings in this analysis of the cardiovascular structures involved in PDA were enlargement of the heart by virtue of a dilated left ventricular chamber. A proportionally greater LVD than LVL occurred. Although the left ventricular wall thickness was not significantly altered, it can be concluded that it was either unchanged or was increased in thickness proportional to the increase in chamber size. This would concur with the response of the left ventricular wall to eccentric hypertrophy (19, 38) created by PDA. The ascending aorta, the sinus of the pulmonary artery, and the main pulmonary artery were all increased in size.

**Mitral Insufficiency.** Three cases were studied. One case occurred in a nine month old dog and was congenital, the other two cases were acquired forms of the disease in dogs 12 and 14 years old respectively. While variability of the plotted ratio values did exist among the dogs, it was not attributable to age difference. In every ratio where samplings from all three dogs were obtained it was important to note that the ratio value of the young dog was either very close to the ratio value of one of the old dogs or it fell between their ratio values.

The statistical details from figure 5 and table 5 were used to make deductions about cardiovascular structural sizes. Levograms were obtained in the old dogs, so ratio comparisons relating both left and right ventricular and arterial structures were obtained in only the young dog with congenital MI. In cases where two plotted
points are observed in a ratio, they are those of the young dog and one of the old dogs.

The mean of AH:Wt was significantly increased (P<.01), so the heart size was concluded to be enlarged.

The means of AH:ALV and AH:ARV were not significantly different from the mean of normal dogs in spite of the deductive decision that AH was increased. We know that an enlarged heart silhouette is created by enlargement of one or both of the ventricles, so let us take the logical approach and assume that enlargement of the area of the left ventricle occurred which was proportional in size to the enlargement of the heart silhouette, thus the mean value for AH:ALV was not different from the mean of the normal dogs. Let us logically reason also that ARV was unaffected. In retrospect, the ratio LVL:ARV would have proven this to be true and will be discussed in the comments section. Since, by deduction, ALV was significantly increased in size, one can deduct further that LVL and LVD were significantly increased since the product of these measurements was an estimate of ALV. The mean of LW:RW was greater than the mean of the normal dogs, but not significantly greater. This was enough evidence to conclude that the right ventricular wall thickness was unaltered since the right ventricle was not involved. However, since the mean of LW:RW is 1.5 standard deviations greater than the mean of the normal dogs for this ratio, it was deduced that the left ventricular wall was probably thickened in proportion to the increased volume of the chamber. This means that LW was slightly increased in size and
would verify that LVL and LVD were, in fact, also significantly increased since there was no significant change in the mean ratio values from the normal means when they were related to LW. Based on the inductive decision that the mean of LVL:LVD was not significantly different from the normal mean, there was no proportional difference in the increases of LVL and LVD. The left ventricular chamber was concluded to be, indeed, dilated and LVW was significantly increased. This reasoning is compatible with the changes that occur in left ventricular eccentric hypertrophy created by MI (19).

The means of svA:dA and aA:dA were significantly increased (P<.01). This was enough evidence to conclude that both svA and aA were significantly increased in size, since dA has been shown to be unaffected by any disease in this study. svA:aA was not different from the normal means, so these two structures were proportionally increased in size.

The mean of spa:svA was not significantly different from the mean of the normal dogs, while the mean of mPA:aA was significantly decreased (P<.05). Since SPA and mPA were related to structures (svA and aA respectively) which were proportionally increased in size, it was concluded that SPA was also significantly increased in size, while mPA was either unchanged or decreased in size. The mean of SPA:mPA was significantly increased (P<.01). This verifies that SPA was significantly increased in size, but still no decision was reached which excluded mPA from being decreased in size. However, with the single hemodynamic lesion
Figure 5

Ratio values for each dog with mitral insufficiency are plotted within the individual ratio variables. A statistical decision about the mean of the aggregate of the plotted values with respect to the mean of the normal dogs for each ratio variable is indicated.
Figure 5

MITRAL INSUFFICIENCY

\[ \text{LW:RW} \]
\[ \text{mPA:aA} \]
\[ \text{svA:aA} \]
\[ \text{AA:AA} \]
\[ \text{P<.01} \]
\[ \text{NS} \]
\[ \text{P<.05} \]

\[ \text{LVD:LW} \]
\[ \text{AH:ALV} \]
\[ \text{AH:ARV} \]
\[ \text{P<.01} \]
\[ \text{NS} \]

\[ \text{P = INDUCTIVE DECISION} \]
\[ \text{NS = NOT SIGNIFICANT} \]
\[ \text{--- = NORMAL MEAN} \]
\[ \text{--- = TWO STANDARD DEVIATIONS FROM NORMAL MEAN} \]
of MI, it was unlikely that a circulatory perturbation existed which
would decrease the size of mPA and it was assumed to be unaffected.

The important deductive findings in this analysis of the
cardiovascular structures involved in MI were enlargement of the
heart by virtue of a dilated left ventricular chamber. The left
ventricular wall thickened in proportion to the increase in left
ventricular volume, and eccentric hypertrophy existed. The
ascending aorta, aortic sinus of Valsalva, and sinus of the
pulmonary artery were all increased in size.

**Atrial Septal Defect.** One case, a six month old female Boxer
dog, was available for study. Right ventricular and pulmonary
artery measurements were not available because a levogram study
was performed. Statistical details from figure 6 and table 5
were used to make deductions about cardiovascular structure sizes
of this single case study.

The mean of AH/WT was not significantly different from the
mean of the normal dogs, so heart size was deduced as being
unaffected. The absence of a significant positive response to
this ratio value was not understood since it is well known that
the heart enlarges with this disease.

Mean ratio values relating to left ventricular structure
measurements were not significantly different from the mean values
of the normal dogs. This was also not understood for it seems
reasonable that the circulatory dynamics which effectuate an
enlargement of the ascending aorta (as will be shown later) should
also alter either the left ventricular wall thickness or the left
Figure 6

Ratio values for each dog with atrial septal defect are plotted within the individual ratio variables. A statistical decision about the mean of the aggregate of the plotted values with respect to the mean of the normal dogs for each ratio variable is indicated.
Figure 6

ATRIAL SEPTAL DEFECT

**P**: INDUCTIVE DECISION
**NS**: NOT SIGNIFICANT
***: DATA UNAVAILABLE

---

SP, LV: PA, LA
LW, RV, WA
AH, LV, LA
AH: LV, WA

---

= NORMAL MEAN
= TWO STANDARD DEVIATIONS FROM NORMAL MEAN

---

P<.05
P: INDUCTIVE DECISION
NS: NOT SIGNIFICANT
*: DATA UNAVAILABLE
-: NORMAL MEAN
- - : TWO STANDARD DEVIATIONS FROM NORMAL MEAN
ventricular chamber size to an extent that they would be detected by comparing ratio measurements.

The mean of svA:dA was not significantly different from the mean of the normal dogs. Since dA has been described to be unaffected by any of the diseases studied, svA was concluded to be unaffected. The mean of svA:aA was significantly decreased (P<.05) while the mean of aA:dA was significantly increased (P<.01). By deduction, aA was significantly increased in size.

Because there was only a single sample and many ratios with data unavailable, the only salient deductive findings in this case of ASD was enlargement of the ascending aorta.

**Ventricular Septal Defect.** Two cases were available for study. In one dog, just a levogram was available, so the ratios which related the right and left ventricular and arterial structures were obtained in the other dog only. Statistical details from figure 7 and table 5 were used to make deductions about cardiovascular structure sizes. There was a wide separation of ratio values between the two dogs in several variables (AH:Wt, aA:dA, svA:dA, svA:aA). The reason for this variation in values was not known.

The mean of AH:Wt was significantly increased (P<.01), so heart size was concluded to be enlarged.

The mean of LW:RW was not significantly different from the corresponding mean of the normal dogs, so both left and right ventricular free wall thicknesses were concluded to be unaffected.

Mean ratio values relating to left ventricular structure
Figure 7

Ratio values for each dog with ventricular septal defect are plotted within the individual ratio variables. A statistical decision about the mean of the aggregate of the plotted values with respect to the mean of the normal dogs for each ratio variable is indicated.
sizes were not significantly altered from the mean values of the normal dogs. As in ASD, this was not understood.

The right ventricle was not adequately opacified for planimetry, so ARV was unavailable.

The means of dA:dA and svA:dA were significantly increased (P<.01). Since dA was shown not to be affected by any disease in this study, both AA and svA were deduced to be significantly increased in size. Proportionally, AA was increased more than svA according to the mean of svA:AA which was significantly decreased (P<.05).

The mean of sPA:mPA was not significantly different from the mean of the normal dogs. This means that sPA and mPA were either normal in size or that they were proportionally increased or decreased in size. The means of sPA:svA and mPA:AA also were not significantly different from the mean of the normal dogs. svA and AA were already shown to be significantly increased in size so deductively this means that sPA and mPA were also significantly increased in size.

The salient deductive findings in this analysis of the cardiovascular structures involved in VSD were enlargement of the heart for which the enlarged chamber contributing to this was undecided. The aortic sinus of Valsalva, ascending aorta, sinus of the pulmonary artery, and the main pulmonary artery were all increased in size.

**Aortic Stenosis.** Three cases were available for study. All were in German Shepherd dogs; two were four months old, and the other was four years old. Age did not appear to affect structural relationships for the ratio plots of all three dogs were relatively
uniform.

Dextrograms and levograms were performed. Unfortunately, in all three dogs, the right ventricle and pulmonary artery were not opacified clearly enough to make measurements of their structures, except for the free wall of the right ventricle. Statistical details from figure 8 and table 5 were used to make deductions about cardiovascular structure sizes.

The mean of AH:Wt was not significantly different from the mean of the normal dogs. Therefore, heart size in the lateral projection was deduced as being unaffected. The absence of a significant positive response to this ratio value was not understood since it is well known that the heart enlarges with this disease.

The mean of LVL:LVD was not significantly different from the mean of the normal dogs, therefore, LVL and LVD could be either normal in size or proportionally increased or decreased in size. The mean of AH:ALV was not significantly different from the mean of the normal dogs, thus ALV was unaffected since AH was previously concluded to be unaffected. This was enough evidence to deduct that LVL and LVD were unaffected since their product was an estimate of ALV. Left ventricular volume, which was a relative estimate of ALV, was concluded to be unchanged.

The means of LVL:LW and LVD:LW were significantly decreased (P<.01). Since LVL and LVD were normal in size, LW, by deduction, was increased in size. This was verified by the inductive decision that LW:RtW was significantly increased (P<.01) since it is known that right ventricular structures are not affected by this disease.
Figure 8

Ratio values for each dog with aortic stenosis are plotted within the individual ratio variables. A statistical decision about the mean of the aggregate of the plotted values with respect to the mean of the normal dogs for each ratio variable is indicated.
AORTIC STENOSIS

LW:RW

aA:dA

sPA:mPA
mPA:aA
sPA:sVA
svA:aA

P<.01

AHL:ALV

LVL:LVD

LVL:LW

AH:ARV

LVD:LW

AH:Wt

NS P<.01

NS

NS

P<.01

P<.01

P<.01

NS

P = INDUCTIVE DECISION
NS = NOT SIGNIFICANT
* = DATA UNAVAILABLE

--- = NORMAL MEAN
----- = TWO STANDARD DEVIATIONS FROM NORMAL MEAN
The mean of svA:da was not significantly different from the mean of the normal dogs. Since da has been described to be unaffected by any of the diseases studied, svA was concluded to be unaffected. The mean of svA:aa was significantly decreased \( (P<.01) \) while the mean of aa:da was significantly increased \( (P<.01) \). By deduction, aa was significantly increased in size.

The important deductive findings in this analysis of the cardiovascular structures involved in AS were increased thickness of the left ventricular wall with no change in the structure dimensions which influence left ventricular volume. The ascending aorta was increased in size. There was an absence of total heart enlargement which was not explainable.

**Pulmonary Stenosis.** Thirteen cases were available for study. They ranged from 4 months to 3.5 years of age. All structure measurements contained enough observations to permit significant decisions about the ratios. The statistical details from figure 9 and table 5 were used to make deductions about cardiovascular structure sizes. There was a wide range of ratio values in the majority of ratio variables. The reason for this variation in values is not known.

The mean of AH:Wt was not significantly different from the mean of the normal dogs. Therefore, heart size in the lateral projection was deduced as being unaffected. The absence of a significant positive response to this ratio value was not understood since it is well known that the heart enlarges with this disease.
A discussion was presented later to show that an absence of a positive response to this ratio in the heart diseases studied was probably an aberration.

Mean ratio values relating to left ventricular structure sizes, and to the aortic diameters were not significantly altered from the mean values of the normal dogs.

The mean of $AH:ARV$ was not significantly different from the mean of the normal dogs, so the chamber size was deducted as being unaffected. This was the expected response from pressure overload defects.

The mean of $mPA:aA$ was significantly increased ($\alpha < .01$), while the mean of $sPA:svA$ was significantly decreased ($\alpha < .01$). Both pulmonary artery structures were related respectively to an aortic structure which was concluded to be unaffected. Therefore, by deductive reasoning, $mPA$ was significantly increased while $sPA$ was significantly decreased in size. This was verified by the mean of $sPA:mPA$ which was significantly decreased ($\alpha < .01$).

$LW$ was deduced as being unaffected since the means of $LVL:LW$ and $LVD:LW$ were not significantly different from the mean of the normal dogs. Therefore, by deduction, the right ventricular wall thickness was significantly increased since the mean of $LW:RW$ was significantly decreased ($\alpha < .01$).

The important deductive findings in this analysis of the cardiovascular structures involved in PS were increased thickness of the right ventricular wall with no apparent change in the area of the right ventricle. The main pulmonary artery was increased in size while the sinus of the pulmonary artery was decreased in
Figure 9

Ratio values for each dog with pulmonary stenosis are plotted within the individual ratio variables. A statistical decision about the mean of the aggregate of the plotted values with respect to the mean of the normal dogs for each ratio variable is indicated.
Figure 9

PULMONARY STENOSIS

mPA:aA

AH:ALV

•

AH:ARV

svA:aA

•

LVL:LV

svAidA:Aa:idA

LVL:LV

•

wT:AH

•

LVD:LV

NS: NS

a < .01

•

NS: NS

a < .01

•

NS: NS

a < .01


α = SIGNIFICANT DECISION (α ERROR)

NS = NOT SIGNIFICANT

_- - NORMAL MEAN

- - TWO STANDARD DEVIATIONS FROM NORMAL MEAN
size. There was an absence of total heart enlargement which was not explained.

Tricuspid Insufficiency. One case, a four-month-old female Standard Poodle, was available for study. Statistical details from figure 10 and table 5 were used to make deductions about cardiovascular structure sizes of this single case study.

The mean of AH:Wt was not significantly different from the mean of the normal dogs. Therefore, heart size was deduced as being unaffected, and this was not understood since it is well known that the heart enlarges with this disease. The possible explanation for the absence of a positive significant response to this ratio in the lateral projection was discussed later.

The mean of AH:ALV was significantly increased (P<.01). Since the area of the heart silhouette was unaffected, ALV, be deduction, was significantly decreased. The mean of LVL:LVD was not significantly different from the mean value of the normal dogs. This indicates that there was no proportional difference between the two dimensions. Therefore, both LVL and LVD were concluded to be significantly decreased since the product of these two measurements was an estimate of ALV. An important deduction involved in the decreased ALV was that left ventricular volume was significantly decreased (since its size was a relative estimate of ALV), and this was not understood. Because LVL and LVD were deduced as being significantly decreased, the relationship of LW to these two structures in the ratios LVL:LW and LVD:LW, indicates that LW was also significantly decreased since there was no significant
difference between the mean ratio values and the corresponding mean of the normal dogs.

The mean of AH:ARV was not significantly different from the mean of the normal dogs. This was in contradistinction to the subjective angiocardiographic observation that the right ventricular chamber appeared dilated. In retrospect, the ratio LVL:ARV would have proven this to be true and will be discussed in the comments section.

The mean of LW:RW was not significantly different from the mean of the normal dogs. LW was previously demonstrated to be significantly decreased by its relationship to LVL and LVD, so RW was deduced as being proportionately decreased in thickness.

Mean ratio values relating to the aortic diameters were not significantly different from the mean of the normal dogs.

The mean of sPA:svA was significantly decreased (P<.01). svA was previously demonstrated unaffected, so sPA, by deduction, was significantly decreased in size. The mean of mPA:AA was not significantly different from the mean of the normal dogs, so mPA was concluded to be unaffected. It was a unique aberration to have sPA significantly decreased in size while mPA remained normal, but this was verified by the decision that the mean of sPA:mPA was significantly decreased (P<.05).

The important deductive findings in the analysis of the cardiovascular structures involved in TI were decreased measurements of the structures which were involved in left ventricular volume (LVD, LVL, and ALV), a decreased thickness of the wall of
Figure 10

Ratio values for each dog with tricuspid insufficiency are plotted within the individual ratio variables. A statistical decision about the mean of the aggregate of the plotted values with respect to the mean of the normal dogs for each ratio variable is indicated.
Figure 10

TRICUSPID INSUFFICIENCY

P = INDUCTIVE DECISION
NS = NOT SIGNIFICANT
--- = NORMAL MEAN
---- = TWO STANDARD DEVIATIONS FROM NORMAL MEAN

P < .05
P < .01
Table 5

Summary of the decisions about the ratios of diseased dogs. A significance level decision is given about the mean of the population in the ratios of the various diseases and indicates the direction (increase or decrease) the population ratio has deviated if it is different from the normal population.
Table 5

DECISIONS ABOUT RATIOS

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α = SIGNIFICANT DECISION (α ERROR)  NS = NOT SIGNIFICANT (α OR P > .05)
P = INDUCTIVE DECISION  * = DATA UNAVAILABLE

↑ = INCREASED MEAN
↓ = DECREASED MEAN
both the right and left ventricle, and a decrease in the sinus of the pulmonary artery without a change being reflected also in the main pulmonary artery. In retrospect, right ventricular enlargement was found to have occurred.

Differential Diagnosis. Ratio analysis of structures measured from angiocardiograms in heart diseased dogs showed that it was possible to detect aberrant structures. Table 6 summarizes the deductive decisions made regarding the relative size of cardiovascular structures in diseased dogs compared to normal dogs. It can be used as a guide to assist in differential diagnosis.

The cardiovasopathy of the left ventricular flow overload diseases, PDA and MI, was similar. The response of the left ventricular cavity in both diseases was enlargement; thereby increasing the measurements associated with LVV (LVD, LVL, and ALV). AA as well as sPA increased in diameter in both diseases. The following three structures differentiated the two diseases: 1) LW was unaffected in PDA while it was increased proportional to the left ventricular enlargement in MI, 2) sVA was unaffected in PDA while it was increased in MI, 3) mPA diameter was increased in PDA and was unaffected in MI.

The deviated cardiovascular structure response to VSD reflected the aberrant architecture of PDA and MI by an increased area of the cardiac silhouette, an increased diameter of AA, and an increased diameter of sPA. VSD differed from PDA and MI by not involving any of the structures associated with LVV (LVD, LVL, and ALV). VSD's variegated structure paralleled PDA and contrasted MI
by not affecting LW and by having an increased mPA diameter. The reverse association of these two diseases to VSD was demonstrated by the increased diameter of svA.

Abnormal cardiovascular structures due to the circulatory stress of ASD and VSD lesions should be closely allied since both diseases shunt blood most commonly from the left to the right side of the heart. Although a dearth of pertinent data existed for ASD analysis, the comparison of available information showed similar cardiovascular responses in the two diseases. Aside from the unexplained normal heart size deduced in ASD, the only difference between the two diseases in the available data was the unaffected size of svA in ASD. This was also difficult to explain and was possibly attributed to a pseudodecision due to the paucity of observations in both diseases.

AS and PS are pressure overload diseases of the left and right ventricles respectively and are easily differentiated from the other diseases. The left ventricular free wall thickness (LW) was greatly increased and aA was increased in diameter in AS. The right ventricular free wall thickness (RW) was greatly increased; mPA was increased in diameter while sPA was decreased in diameter in PS. All other structures were unaffected in both diseases.

The one case of TI was easily differentiated from all other diseases by decrescence in various structures to the circulatory stress created by the lesion. All left ventricular parameters (LW, LVD, LVL, ALV, and LVV) were decreased as was RW and sPA. Right ventricular enlargement, however, was detected.
Table 6

Summary of the deductive decisions about the relative size of the cardiovascular structures in diseased dogs compared to normal dogs. It indicates the direction (increased or decreased) that the structure has deviated if it is different from the normal population.
Table 6

RELATIVE CARDIOVASCULAR STRUCTURE SIZES

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⊗ = relative wall thickness
NA = Not Affected
↑ = Increased
↓ = Decreased
* = Data Unavailable
**Importance of Ratio Analysis.** Cardiovascular lesions are not seen in a conventional radiograph. Just the adaptations of the heart (ventricular chamber and free wall enlargements) and great vessels (dilatation) due to the circulatory stress caused by the lesions are visualized.

In this quantitative angiocardiographic study, the size of aA was increased in PDA, MI, ASD, VSD, and AS. Also, the size of mPA was determined to be increased in PDA, VSD, PS, and has been reported to be increased in ASD (16, 25, 31). Three (PDA, ASD, and VSD) of the six diseases affecting aA and mPA cause enlargement in both of these structures. On a radiograph, when one or both of the great vessels are determined by subjective observation or quantitative analysis to be enlarged, it is necessary to perform angiocardiographic techniques for differential diagnosis. If the lesion causing the enlargement is not visualized directly, deductive ratio analysis of the structure measurements can be used for differentiation.

It was recognized that the conclusions about the results in this study had limitations due to unavailable data and a paucity of observations in the majority of the diseases studied. Nevertheless, the decisions made by deductive ratio analysis with regard to the relative structure sizes of the diseased dogs was consistent with those reported in humans with corresponding diseases and in which absolute structure measurements were corrected for body
surface area (11, 12, 29, 31, 37, 40). This concurrence greatly
strenthened the assurance of the reliability of the ratio analysis
method of determining relative cardiovascular structure size. It
also gave this indirect analysis method importance for usage in
dogs since there was no accepted reliable method for determining
body surface area in dogs which utilizes both weight and body
length whereby absolute direct corrected measurements could be
obtained as in humans.

While angiocardiography is primarily used to identify specific
cardiopaties, during the interpretation, some quantitative
estimates about the relative size of anatomical cardiovascular
structures can be obtained. Although the measurements described
in this report were admittedly gross, the deductive analysis was
reliable, simple, and was made rapidly enough to be incorporated
into the routine interpretation of all angiocardiograms. An
assessment of the derived ratios of measurements of these structures
would be a valuable complement to auscultation, electrocardiography,
radiography, catheterization procedures, laboratory tests, and
clinical symptomatology in order to arrive at a definite diagnosis.

The distinct advantage of angiocardiographic analysis of
cardiopaties is that it allows an "in vivo" deter-
mination of structural sizes under physiologic conditions which
approach normality. Furthermore, the accuracy of angiocardiography
is superior to other methods for the determination of cardiovascular
structure dimensions. Necropsy measurement of vessel dimensions
yield lower results than angiocardiographic measurement due to
their collapsed state (31). Ventricular free wall necropsy measurements are greater than angiocardiographic measurements attributed to the fact that the ventricles contract in the post-mortem state and distort the configuration (11, 29, 37). It has also been shown that electrocardiographic patterns could not distinguish concentric from eccentric hypertrophy (49) nor the degree of left ventricular hypertrophy (28). Both conditions can be identified by quantitative angiocardiography.

By analyzing the alterations in the gross structure of the heart that occur in various forms of heart disease, one becomes cognizant of the pathophysiological and biophysical processes that exist. For instance, the fact that svA was increased in diameter in some diseases that affected aA and not others may be of importance, but can not be explained.

**Basic Shortcomings of Ratio Analysis.** The following conditions and variables produced difficulties in the ratio analysis determination of relative structure size in certain diseases:

1. *Eccentric Hypertrophy.* Left ventricular wall thickness (LW) that was increased proportional to the enlargement of the left ventricular cavity, as occurred in MI, was not detectable by statistical decision for the small number of available observations. Logical reasoning was necessary to deduce an increase in LW.

2. *AH:ARV.* Right ventricular enlargement, as occurred in TI, was not detected by the ratio
AH:ARV. This is because the correlation
coefficient between AH and ARV was insignificant.
In retrospect, ARV correlates well with LVL
\( r = .8230, \alpha < .025, N = 7 \), and if the ratio
LVL:ARV had been used, right ventricular enlarge-
ment would have been detected for this disease.
Furthermore, LVL:ARV would have shown positively
that ARV was of normal size in MI which would
have confirmed that ALV was indeed increased.
This would have obviated the conjecture that
ALV was increased in size.

3. AH:Wt. It was expected that either right
of left ventricular enlargement would be evident
by an increased lateral area of the heart
silhouette so that the ratio AH:Wt would always
be significantly increased with respect to the
concerning mean of the normal dogs. However,
in spite of the excellent correlation coefficient
between AH and Wt \( r = .8630, \alpha < .0005, N = 16 \), this
ratio was unreliable and led to the false decision,
that heart size was not affected in the diseases
of ASD, AS, PS, and TI. In these diseases, the
spread of observation points for this ratio around
the mean of normal dogs was wide and no significant
difference existed. In particular, the ratio
points in pulmonary stenosis spanned beyond two
standard deviations on both sides of the mean ratio value for normal dogs. Figure 11 illustrates this point. It was important to note that there was a size separation in this group of dogs; the mean of this ratio for the smallest seven dogs (3.6 to 10.0 Kg.) was significantly increased ($\alpha<.01$) and the mean of the five largest dogs (15.9 to 36.4 Kg.) was significantly decreased ($\alpha<.01$) with respect to the mean of the normal dogs. This made AH appear to be dependent upon body weight which, in fact, was not linear in certain types of heart disease.

The reason for this seems logical. The ratio of the area of the heart silhouette to body weight in the lateral projection was markedly increased when eccentric hypertrophy of the left ventricle (PDA and MI) occurred. Since the ellipsoidal geometry of the left ventricle is spatially oriented almost perpendicular to the central beam of the X-rays in the lateral projection (5), an enlargement of this chamber would be easily detected. The inconsistent AH:Wt ratio values which occurred in concentric hypertrophy of the left ventricle (AS) cannot be explained.

The right ventricle's crescent-shaped geometry is spatially oriented at a diagonal to the central
beam of the X-ray in the lateral projection. This makes it easy to reason that an enlargement of this ventricle would not be detected consistently in this plane.

Another possible explanation for the inconsistent results with this ratio in all weight ranges could be that the relationship between AH to Wt decreases with increasing body weight. When dogs with pulmonary stenosis were divided into the same two weight groups as described before, figure 12 indicates there was a more narrow scattering of observation points around the regression of AH on Wt in the smaller dogs ($r=.9275$, $\alpha<.005$, $N=7$), than in the larger dogs ($r=.8495$, $\alpha<.05$, $N=5$). The smaller dogs were in a weight range slightly lower than the weight range of the normal dogs while the larger dogs were markedly heavier than the normal dogs.

Assuming the two above factors (decremental ratio shift with weight increase, and the incongruous ventricular geometry and spatial orientation) did, in fact, cause the inconsistent AH:Wt ratio results, this could be circumvented by the following two ways: 1) by calculating mean ratio values of normal dogs in weight ranges of 5 Kg. increments and comparing the ratio of the diseased dogs individually to the mean of the normal dogs with corresponding
Figure 11

Relationship between body weight and the ratio AH:Wt for dogs with pulmonary stenosis. There were two distinct areas of observation points on opposite sides of the mean ratio value for normal dogs. The smaller dogs had a significantly increased (α<.01) mean ratio value, and the larger dogs had a significantly decreased (α<.01) mean ratio value.
Figure 11

PULMONARY STENOSIS

α = SIGNIFICANT DECISION (α-ERROR)

=- NORMAL MEAN

- = TWO STANDARD DEVIATIONS FROM NORMAL MEAN

↑ = INCREASED MEAN

↓ = DECREASED MEAN

RATIO VALUE (AH:Wt.)

WEIGHT (Kg.)
Relationship between body weight and the planimetered area of the heart silhouette (AH) in dogs with pulmonary stenosis. Note that the scattering of observation points around the regression of AH on Wt was more narrow in the smaller dogs than in the larger dogs.
Figure 12

PULMONARY STENOSIS

$\text{AH (cm}^2\text{)}$

$\text{WEIGHT (Kg.)}$

$r = .8495$

$\alpha = .05$

$N = 5$

$r = .9675$

$\alpha = .005$

$N = 7$
weights, or 2) obtain AH/Wt values from a large number of dogs with as wide a weight range as could be expected. The 95% confidence limits from the regression line would be useful for dogs of all body weights.

A recently developed measurement technique for dorso-ventral projection radiographs was demonstrated to be capable of detecting total heart enlargement, selective ventricular enlargement, and enlargement of the great vessels in all commonly occurring heart diseases in dogs (26).

Evaluation of a Single Patient. Quantitative analysis of a patient's angiocardiogram can be used to detect and differentiate heart diseases. The principle used was similar to that which was already discussed. The mean ratio values of the dogs without heart disease were shown to follow a normal distribution. For this reason, it was justifiable to establish the following categories for making a judgement about the ratio values of a patient: "normal," "probably normal," "probably abnormal," "abnormal." The selection of measures, corresponding to the categories, were arbitrarily chosen, as shown in figure 13, because they were based upon accepted statistical theory (54). The considerations for the limits of each category are as follows:

1. "Normal" was any value between the mean of the distribution and +0.67C and -0.67C. This area includes 50 percent of the observations in the normal range, and the measures are known as probable
Figure 13

Distribution curve for the evaluation of cardiovascular measurement ratios in a single case study. It gives the criteria for the placement of ratio values into the categories of "normal," "probably normal," "probably abnormal," and "abnormal."
Figure 13

NORMALITY-ABNORMALITY DISTRIBUTION CURVE

2.5%

11.25%

25%

1.96σ

4.06σ

1.10σ

1.06σ

0.06σ

0.95σ

0.50σ

95%

22.5%

ABNORMAL

NORMAL
error in statistical theory.

2. "Abnormal" was any value at or beyond 
\[ \pm 1.96\sigma \] from the mean. This corresponded to 
the commonly used 5 percent level of significance, 
of which 2.5 percent was higher and 2.5 percent 
was at the lower end of the distribution. The 
area between \(+1.96\sigma\) and \(-1.96\sigma\) includes 
95 percent of the observations. For convenience, 
one might use values of \(+2.00\sigma\) and \(-2.00\sigma\).

3. The limits between the "normal" and the 
"abnormal" leave an area that includes 22.5 
percent on each half of the distribution. These 
areas between \(+0.67\sigma\) and \(+1.96\sigma\) and between 
\(-0.67\sigma\) and \(-1.96\sigma\) were divided into equal 
parts. This corresponded to the values \(+1.10\sigma\) 
and \(-1.10\sigma\) for the right and left tails of the 
distribution, leaving approximately 11.25 percent 
of the observations in each new category.

"Probably normal" was any value between \(+0.67\sigma\) 
and \(+1.10\sigma\) or between \(-0.67\sigma\) and \(-1.10\sigma\).

"Probably abnormal" was any value between 
\(+1.10\sigma\) and \(+1.96\sigma\) or between \(-1.10\sigma\) and 
\(-1.96\sigma\).

For each ratio, the range of values which corresponds to the 
appropriate diagnostic category, as shown in table 7, were constructed 
from the statistical parameters of the normal dogs. A given ratio 
value was categorized by determining the range of values within which
it was located. When a judgement was made for all of the ratios obtained from a patient's angiocardiograms, deductive analysis, as previously described, was used to form a conclusion about the relative size of each cardiovascular structure. These conclusions were compared with the findings in table 6 to assist in differentiating heart diseases. Admittedly, while this method is reliable, it should be used as a complement to all other available diagnostic techniques to assure accuracy of diagnosis.
Table 7

Range of values for each ratio by categories. A given ratio value was determined as "normal," "probably normal," "probably abnormal," and "abnormal."
<table>
<thead>
<tr>
<th>RATIO (µ)</th>
<th>NORMAL (µ ± 0.67σ)</th>
<th>PROBABLY NORMAL (±0.67σ to ±1.10σ)</th>
<th>PROBABLY ABNORMAL (±1.10σ to ±1.96σ)</th>
<th>ABNORMAL (≥ ±1.96σ)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LW:HW (2.05)</td>
<td>1.74-2.36</td>
<td>1.54-1.73 and 2.37-2.56</td>
<td>1.15-1.53 and 2.57-2.95</td>
<td>&lt;1.15 and &gt;2.95</td>
</tr>
<tr>
<td>sPA:mPA (1.22)</td>
<td>1.14-1.30</td>
<td>1.09-1.13 and 1.31-1.35</td>
<td>0.98-1.08 and 1.36-1.46</td>
<td>&lt;0.98 and &gt;1.46</td>
</tr>
<tr>
<td>sPA:sVA (1.12)</td>
<td>1.07-1.17</td>
<td>1.03-1.06 and 1.18-1.21</td>
<td>0.96-1.02 and 1.22-1.28</td>
<td>&lt;0.96 and &gt;1.28</td>
</tr>
<tr>
<td>mPA:aA (1.24)</td>
<td>1.10-1.38</td>
<td>1.01-1.09 and 1.39-1.47</td>
<td>0.83-1.00 and 1.48-1.65</td>
<td>&lt;0.83 and &gt;1.65</td>
</tr>
<tr>
<td>sVA:aA (1.33)</td>
<td>1.25-1.41</td>
<td>1.20-1.24 and 1.42-1.46</td>
<td>1.09-1.19 and 1.47-1.57</td>
<td>&lt;1.09 and &gt;1.57</td>
</tr>
<tr>
<td>sVA:dA (1.79)</td>
<td>1.70-1.88</td>
<td>1.64-1.69 and 1.89-1.94</td>
<td>1.52-1.63 and 1.95-2.06</td>
<td>&lt;1.52 and &gt;2.06</td>
</tr>
<tr>
<td>aA:dA (1.35)</td>
<td>1.26-1.44</td>
<td>1.20-1.25 and 1.45-1.50</td>
<td>1.08-1.19 and 1.51-1.62</td>
<td>&lt;1.08 and &gt;1.62</td>
</tr>
<tr>
<td>Ah:ALV (3.32)</td>
<td>2.97-3.67</td>
<td>2.75-2.96 and 3.68-3.89</td>
<td>2.30-2.74 and 3.90-4.34</td>
<td>&lt;2.30 and &gt;4.34</td>
</tr>
<tr>
<td>Ah:ARV (2.20)</td>
<td>1.75-2.65</td>
<td>1.47-1.74 and 2.66-2.93</td>
<td>0.91-1.46 and 2.94-3.49</td>
<td>&lt;0.91 and &gt;3.49</td>
</tr>
<tr>
<td>LVL:LV (6.23)</td>
<td>5.76-6.70</td>
<td>5.46-5.75 and 6.71-7.00</td>
<td>4.86-5.45 and 7.01-7.60</td>
<td>&lt;4.86 and &gt;7.60</td>
</tr>
<tr>
<td>LVD:LV (3.95)</td>
<td>3.36-4.54</td>
<td>2.99-3.35 and 4.55-4.91</td>
<td>2.24-2.98 and 4.92-5.66</td>
<td>&lt;2.24 and &gt;5.66</td>
</tr>
<tr>
<td>LVL:LVD (1.63)</td>
<td>1.45-1.81</td>
<td>1.33-1.44 and 1.82-1.93</td>
<td>1.10-1.32 and 1.94-2.16</td>
<td>&lt;1.10 and &gt;2.16</td>
</tr>
<tr>
<td>AH:Wt (4.73)</td>
<td>4.35-5.11</td>
<td>4.10-4.34 and 5.12-5.36</td>
<td>3.61-4.09 and 5.37-5.85</td>
<td>&lt;3.61 and &gt;5.85</td>
</tr>
</tbody>
</table>
SUMMARY AND CONCLUSIONS

Angiocardiography is an advantageous diagnostic method for visualizing specific cardiovascular lesions and for quantitatively analyzing cardiovascular structures. The need existed, in veterinary cardiology, for a precise diagnostic technique for both acquired and congenital cardiovasopathies which was reliable, rapid, and economical. The recognition of this need provided the stimulus for developing a quantitative method for evaluating cardiovascular structures from angiocardiograms in order to assist in the differentiation of commonly occurring spontaneous heart diseases in dogs.

Measurements of selected cardiovascular structures were made from angiocardiograms. Those measurements which correlated well in normal dogs were constructed into a ratio assortment. All structures in the ratio assortment were cross referenced, so by selecting a set of pertinent ratios, the relative size of any structure could be analyzed. The ratio values of dogs with heart defects were compared statistically to the mean ratio values of the normal dogs. By deductive analysis of the decisions made from the ratio values of diseased dogs, cardiovascular structure size deviations were detected. The comparison of the structure deviations in the diseases studied provided the necessary information for making a differential diagnosis.

The distinct advantage of quantitative angiocardiographic analysis is that it allows an "in vivo" determination of structural sizes under physiologic conditions which approach normality.
Furthermore, the accuracy of angiocardiology is superior to other methods (electrocardiography and necropsy) for the determination of cardiovascular measurements. For this reason it should be a valuable procedure in experimental physiology for the quantitative estimate of the relative size of anatomical cardiovascular structures.

The usefulness of cardiovascular structure ratio analysis far surpassed its original expectations as a clinical diagnostic method. The procedure was reliable, simple, and was made rapidly enough to be incorporated into the routine interpretation of angiocardograms.
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ADDENDUM