APPENDIX III

Left atrial splitting

Divisions:

A. Epidemiology

B. Postmortem Cardiotomy Method

C. Hemopericardium

D. Acquired Atrial Septal Defect

E. Nonperforating Atrial Splits

F. Endocardial Degeneration with Lipid Deposition

A. Epidemiologic aspects of left atrial splitting.

Tables

TABLE 3.1

SPONTANEOUS LEFT ATRIAL SPLITTING IN 30 DOGS:

AGE, SEX, BREED AND SYNDROME

A. Left atrial perforation causing Hemopericardium (9 Dogs)

Dog No.	Breed	Age (yrs.)	Sex
1.	Cocker Spaniel	11	Male
2.	Cocker Spaniel	12	Male
3.	Dachshund	8	Male
4.	Standard Poodle	9	Male
5.	Mongrel	11	Male
6.	Mongrel	12	Female
7.	Mongrel	13	Male
8.	Mongrel	14	Male
9.	Mongrel	Aged	Male

B. Interatrial perforation causing Atrial Septal Defect (4 Dogs)

10.	Cocker Spaniel	10	Male
11.	Dachshund	10	Male
12.	Dachshund	10	Male
13.	Beagle	8	Male

TABLE 3.1 (CONT.)

C. Non-perforating left atrial splits (17 Dogs)

14.	Cocker Spaniel	9	Male
15.	Cocker Spaniel	11	Male
16.	Cocker Spaniel	11	Female
17.	Cocker Spaniel	13	Male
18.	Cocker Spaniel	13	Male
19.	Dachshund	8	Male
20.	Dachshund	8	Male
21.	Dachshund	8	Male
22.	Dachshund	9	Male
23.	Dachshund	13	Male
24.	Dachshund	15	Male
25.	Minature Poodle	9	Female
26.	Mongrel	10	Male
27.	Mongrel	10	Male
28.	Mongrel	12	Male
29.	Mongrel	13	Male
30.	Mongrel	13	Female

D. Totals

1. Sex: Males 26 Females 4
2. Age: 10.8 years (range: 8-15)
3. Breeds: Mongrels - 10; Cocker Spaniels - 8; Dachshunds - 9; Other - 3.

Table 3.2

Sex distribution of 30 dogs
with Left Atrial Splitting.
Univ. of Pa. Veterinary Clinic 1961-1966

	Observed	Expected*
Male	26	15.2
Female	4	14.8
Total	30	30

$x^2 = 14.148$**

$P < .001$

* Based upon the sex distribution of dogs of similar age in the clinic population from July 1963 to Dec. 1965 in Table 3.3: 1172 males = 50.8%; 1135 females = 49.2%. The expected number of male dogs with left atrial splitting was .508 x 30 = 15.2

** Adjusted for smallness of numbers by using the formula:

$$X^2 = \frac{[(\text{Observed} - \text{Expected}) - 0.5]^2}{\text{Expected}}$$

From Snedcor, G.W.: Statistical Methods, p.224
Iowa University Press, Ames, Iowa, 1956.

TABLE 3.3

Left Atrial Splitting:

Sex and Breed Distributions of an Age Selected Clinic Population Sample and 30 Dogs with Left Atrial Splitting (L.A.S.)

University of Pennsylvania Veterinary Clinic, July 1963 to Dec. 1965.

	Clinic population (8 years and older)		Dogs with L.A.S. (8-15 years old)*		
Breed	No. of dogs	% of total	No. observed	No. expected**	
Mongrel	792	34.3	10	10.3	
Dachshund	115	5.0	9	1.5	
Cocker Spaniel	193	8.3	8	2.5	
Beagle	44	1.9	1	.6	
Poodle	174	7.5	2	2.2	
Boxer	207	9.0	0	2.7	
Collie	97	4.2	0	1.3	
Fox Terrier	56	2.4	0	.7	
German Shep.	85	3.7	0	1.1	
Other breeds	549	23.7	0	7.1	
TOTALS	2,312	100%	30	30	$x^2 = 61.201$*** $P < .001$ (df=4)
Number of males	1172	50.8%	26		
Number of females	1135	49.2%	4		

*Confirmed or detected at necropsy 1961-1966.

**Number expected = total cases of L.A.S. (30 dogs) multiplied by % of breed in clinic population sample of similar age.

***Contingency table was reduced to a 5 x 2 table for calculation of Chi square because of excessively small expected values. The computation was based upon the four breeds with more than one observed case; all other breeds were grouped as a single population.

TABLE 3.4

CHRONIC VALVE DISEASE:

Sex and Breed Distributions of an Age Selected Clinic Population Sample and 365 Dogs with Chronic Valve Disease (C.V.D.)

University of Pennsylvania Veterinary Clinic, Jan. 1958 - July 1960

	Clinic population (5 years and older)		Dogs with C.V.D. (5 years and older)*		
Breed	No. of dogs	% of total	No. observed	No. expected**	Chi Square
Mongrel	616	31.8	98	115.7	1.183
Dachshund	58	3.0	15	11.0	1.454
Cocker Spaniel	262	13.5	72	49.3	10.452
Beagle	36	1.9	11	6.9	2.436
Poodle	48	2.5	12	9.1	.924
Boxer	180	9.3	35	33.9	.036
Collie	71	3.7	10	13.5	.907
Fox Terrier & Irish Terrier	140	7.2	19	26.3	2.026
German Shep.	67	3.4	3	12.8	7.503
Other breeds	462	23.7	90	86.5	.142
TOTALS	1,940	100 %	365	365	$x^2 = 27.063$ $P<.01$ (df=9)
Number of Males	994	51.2%	213	186.9	3.645
Number of Females	946	48.8%	152	178.1	$x^2 = \frac{3.825}{7.470}$ $P<.01$***

*Subgroup of Table 1.2 accounting for 93% of the dogs with C.V.D. in a clinic population sample of 4,831 dogs excluding those referred for heart disease.

**Number expected = total cases of C.V.D. observed (365 dogs) multiplied by % of breed in clinic population sample of similar age.

***Overall age and sex adjusted prevalence ratios presented in Table 1.3.

TABLE 3.5 a

INTERVERTEBRAL DISC SYNDROME:

Sex and Breed Distributions of the Total Clinic Population and 236 Dogs with the Intervertebral Disc Syndrome (I.D.S.)

University of Pennsylvania Veterinary Clinic, July 1963 - Dec. 1965

Clinic Population (all ages)			Dogs with I.D.S. (2-12 years old)		
Breed	No. of dogs	% of total	No. observed	No. expected*	Chi square
Mongrel	3,711	31.6	28	74.6	29.109
Dachshund	507	4.3	121	10.1	1217.704
Cocker Spaniel	520	4.4	21	10.4	10.804
Beagle	370	3.1	22	7.3	29.601
Poodle	1,164	9.9	12	23.4	5.554
Boxer	489	4.2	0	9.9	9.900
Collie	390	3.3	0	7.8	7.800
Fox Terrier	251	2.1	0	5.0	5.000
German Shep.	1,336	11.4	0	26.9	26.900
Other breeds	3,022	25.7	32	60.6	13.498
TOTALS	11,760	100 %	236	236	x^2 =1355.870
					P<.001 (df=9)
Number of Males	6,335	54.1%	134	127.7	0.311
Number of Females	5,364	45.9%	102	108.3	$x^2 = \frac{0.366}{0.677}$
					Diff. not signif.

*Number expected = total cases of I.D.S. observed (236 dogs) multiplied by % of breed in total clinic population.

TABLE 3.5 b

Intervertebral Disc Syndrome in Dogs other than Dachshunds:

Sex and Breed Distributions of a Clinic Population

Sample and 115 Dogs with the Intervertebral Disc Syndrome (I.D.S.)

University of Pennsylvania Veterinary Clinic, July 1963 - Dec. 1965

	Clinic population* (all ages)		Dogs with I.D.S.*		
Breed	No. of dogs	% of total	No. observed	No. expected**	Chi square
Mongrel	3,711	33.0	28	38.0	2.632
Cocker Spaniel	520	4.6	21	5.3	46.508
Beagle	370	3.3	22	3.8	87.168
Poodle	1,164	10.3	12	11.8	.003
Boxer	489	4.3	-	4.9	4.900
Collie	390	3.5	-	4.0	4.000
Fox Terrier	251	2.2	-	2.5	2.500
German Shep.	1,336	11.9	-	13.8	13.800
Other breeds	3,022	26.9	32	30.9	.039
TOTALS	11,253	100 %	115	115.0	$x^2 = 161.550$ P<.001 (df=8)
No. of Males	6,057	54.1	64	62.2	.052
No. of Females	5,137	45.9	51	52.8	.061
					$x^2 = 0.113$ Diff. not signif.

* Dachshund excluded

** Number expected = cases of I.D.S. observed (115 dogs) multiplied by % of breed in the clinic population sample.

TABLE 3.6

Comparison of the Occurrence of Three Chronic Diseases in Various Purebreds in Descending Orders of Frequency Based Upon Tables 3.3-3.5 University of Pennsylvania Veterinary Clinic 1958-1966

1961-1966	1958-1960	1963-1965
Left atrial splitting	Chronic valve disease	Intervertebral disc syndrome
1. Dachshund *	1. Cocker Spaniel *	1. Dachshund *
2. Cocker Spaniel *	2. Beagle	2. Beagle *
3. Beagle	3. Dachshund	3. Cocker Spaniel *
4. Poodle	4. Poodle	4. Poodle
5. Not observed in other purebreds	5. Less common breeds as a group ** (excluding the following)	5. Less common breeds as a group ***
	6. Boxer	6. Not observed in this series in Boxers, Collies, Fox Terriers, or German Shepherds
	7. Collie	
	8. Fox Terriers	
	9. German Shepherd	
Marked male predominance	Moderate male predominance	No sex predominance

* Breeds showing frequency distributions significantly greater than the rest of the population.

** The frequency of chronic valve disease was higher in Chihuahuas than in Poodles.

*** The frequency of intervertebral disc syndrome appeared to be higher in Pekingese, Chihuahuas and Basset Hounds than in Poodles, based upon small numbers of dogs examined.

B. Post-mortem Cardiotomy Method

Right atrium

The right atrium is incised along its dorsal aspect from the appendage into the caudal vena cava.

Right ventricle

The right ventricular incision is begun in the outflow tract region adjacent to the ventricular septum. It is extended into the main pulmomary artery through the commissure between the left and caudal pulmonary valve cusps. The right ventricular incision is then continued in the opposite direction along the junction of the septum and free wall towards the apex; then caudally toward the base as far as the tricuspid annulus. After reflecting the free wall, the tricuspid valve leaflets, chordae tendinea and papillary muscles are inspected. The two caudal papillary muscles are transected at their origin on the ventricular septum and the ventricular incision is extended into the atrium through the caudal commissural region.

Left atrium

The left atrium is incised along its dorsal aspect close to the entrance of the left pulmonary veins. This incision is begun at the left atrial appendage and extended into the right caudal pulmonary vein. Blood is removed and the atrial aspect of the mitral valve is examined. When ruptured chordae tendineae are present, the segment of valve which they supported often is elevated above the remainder of the valve. The unsupported free edge is usually tilted upward and is rigid owing to fibrosis. Broken chordae tendineae stumps are occasionally visible at this stage of the examination.

Left ventricle

The first left ventricular incision is made halfway between the left ventricular apex and the left anterior interventricular groove. After ascertaining entrance into the left ventricle, the incision is extended in a basal direction a few mm. caudal and parallel to the left anterior descending coronary artery. After partial reflection of the left ventricular free wall, papillary muscles, and mitral valve apparatus, the incision is extended along the left ventricular outflow tract and into the aorta. The intact mitral valve apparatus is inspected from the ventricular aspect.

Mitral valve

A parallel incision is made in the apex of the left ventricle 0.5 to 1 cm. caudal to the first incision. After penetrating the endocardium, the incision is extended basally until it bisects the left anterior papillary muscle. This is done between the separate attachments of the first order chordae tendineæ to the septal and mural leaflets of the mitral valve. Under direct vision, the small remaining commissural cusp is bisected between the insertion of its two primary chordae tendineae. With the valves and chordae tendineae separated, the mitral annulus is cut by extending the ventricular incision until it reaches the previous atrial incision. The entire mitral valve apparatus with intact or spontaneously ruptured chordae tendineae can be flattened out into a two dimensional plane by extending the second ventricular incision in a basal direction along the caudal aspect of the left ventricle until it partially bisects the posterior papillary muscle.

C. Hemopericardium

caused by

Left atrial splitting

Gross Pathology

Figure 3.1 Hemopericardium

Figure 3.2 Recent and old endomyocardial splits

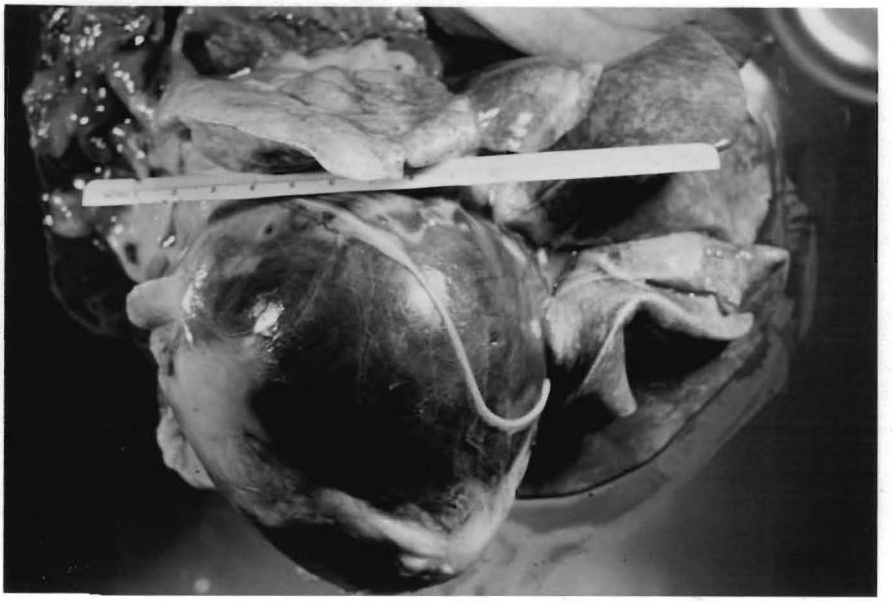
Figure 3.3 Epicardial perforation

Figure 3.4 Recent and old endomyocardial splits.

Clinical and Radiologic Aspects.

(Reprint).

Figure 3.1 Photographs of the heart and lungs of dog # 1 with left atrial perforation and hemopericardium before (A) and after (B) removal of the pericardium. Evidence of pericardial effusion was seen in radiographs made 36 hours before death. At necropsy, the pericardium was thicker than normal and was distended more than usually occurs in acute cardiac tamponade. A large unattached blood clot was present in addition to about 200 cc. of nonclotted blood. The left atrium was very dilated and thin walled. A band of subepicardial hemorrhage was visible in the caudal wall of the left atrium overlying a large, recent endomyocardial split (see next illustration). The specific epicardial site of hemorrhage was not apparent. The lungs were heavy, did not collapse normally, and had rounded edges. Pulmonary edema was observed when cross sections were made.



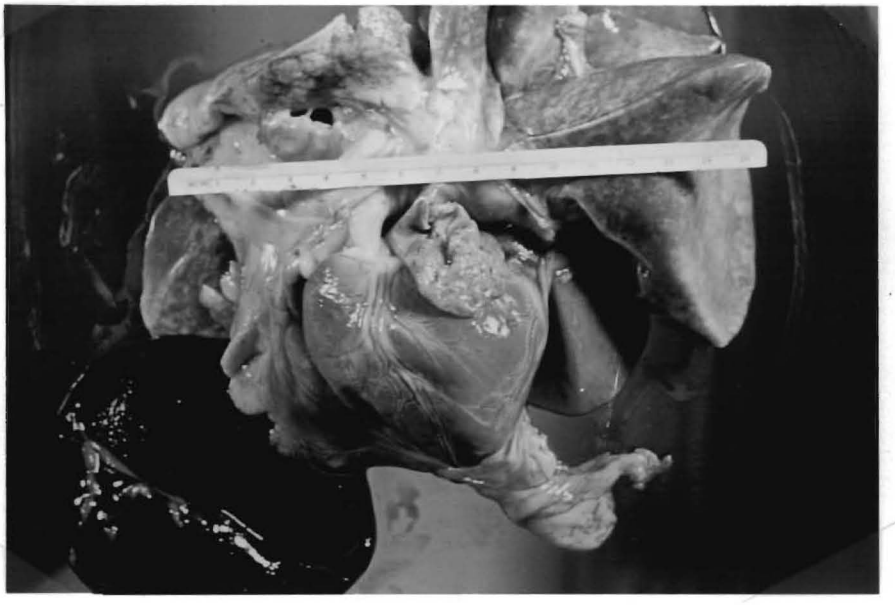


Figure 3.2 Photograph of the left heart chambers of dog # 1 with severe chronic mitral valve disease, left atrial splitting and hemopericardium. A large, thrombus covered, endomyocardial split was present in the left atrium at the most common site: dorsal to the posterior mitral leaflet and parallel to the annulus. In this case it extended partly into the left atrial appendage. Above and parallel to the large recent split was a smaller healed split. Other recent splits were present in the opposite wall of the left atrium.

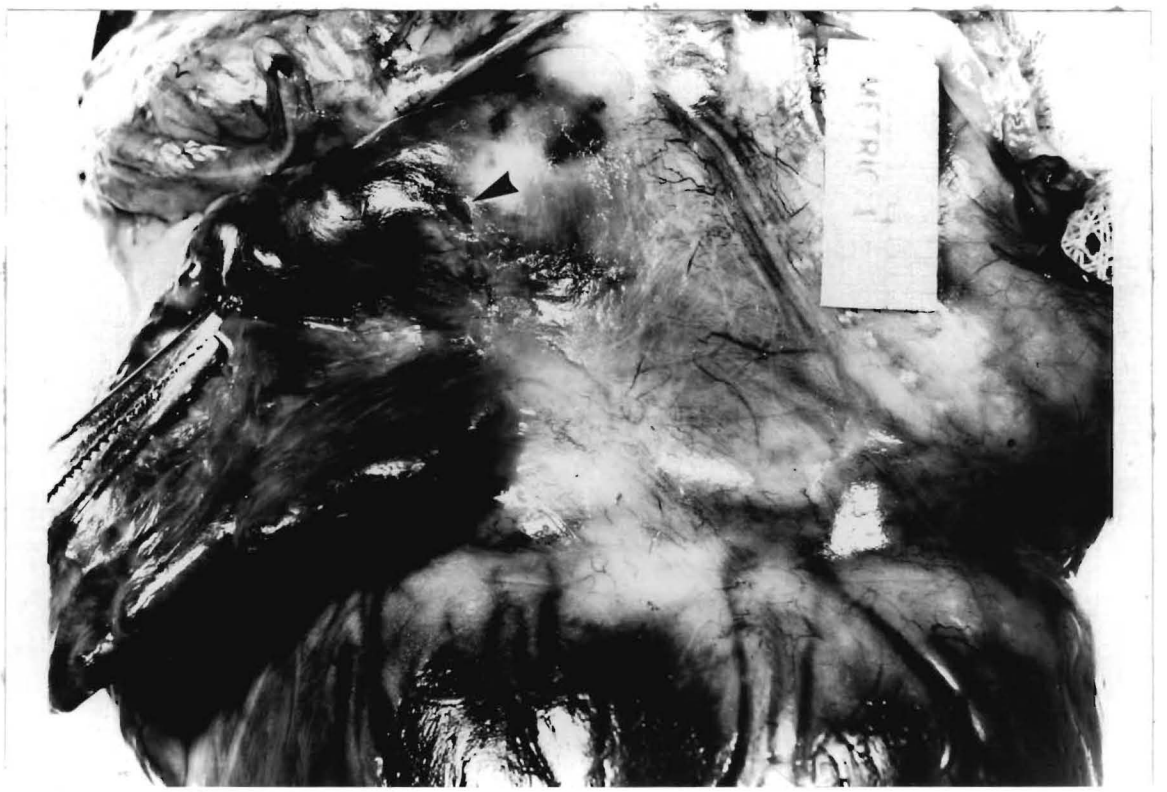
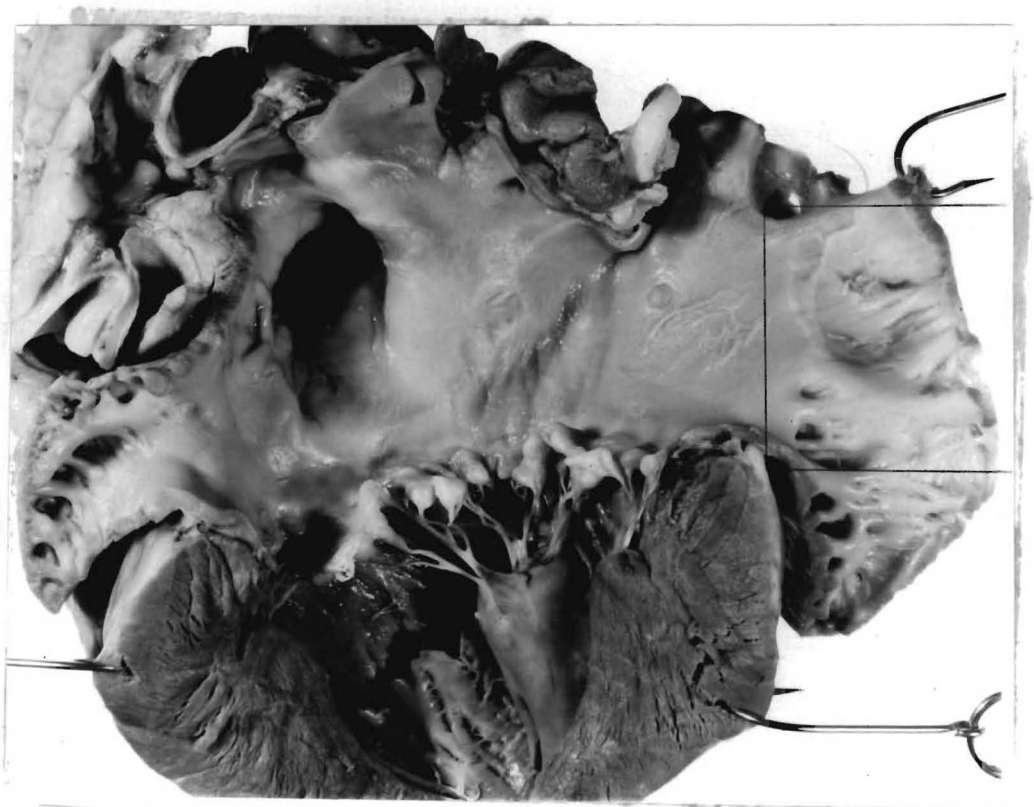
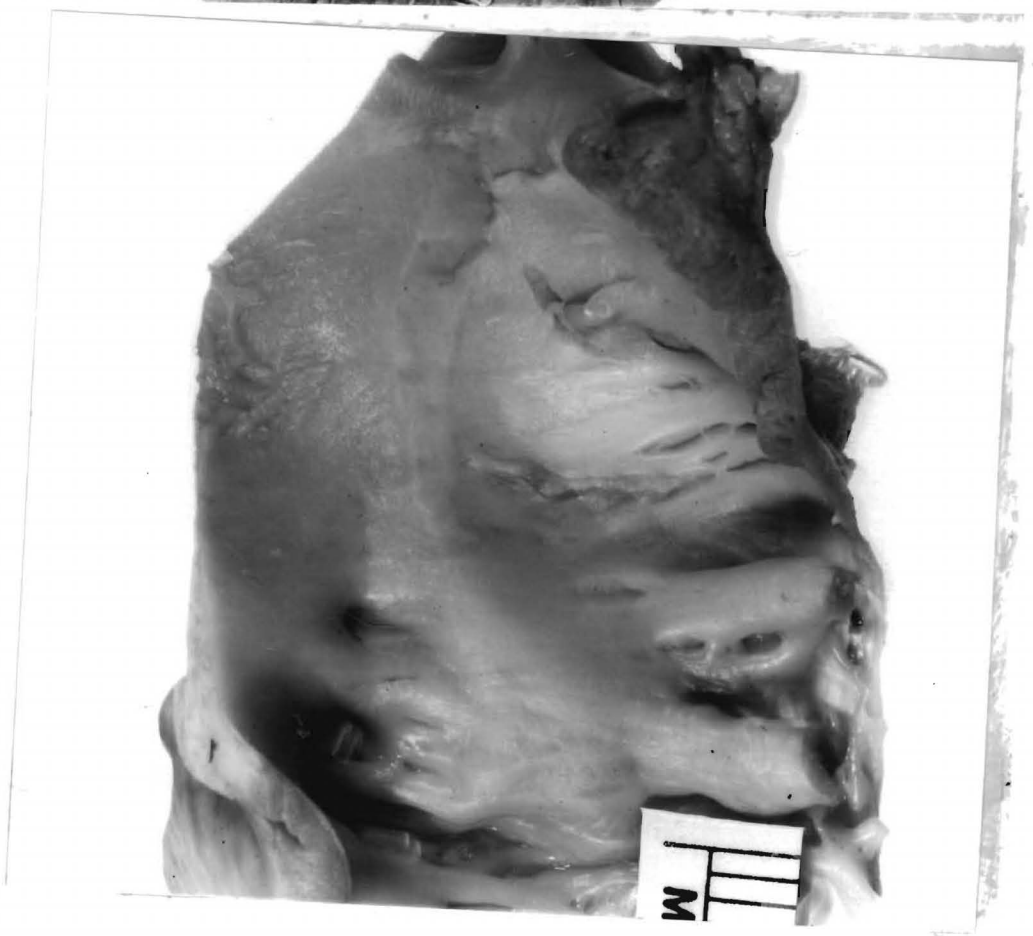


Figure 3.3 Photograph of the left atrial epicardium of dog # 6 which died of acute cardiac tamponade. A 1 x 2 mm. epicardial split was present in the dorsocaudal wall of the left atrium (arrow). Nonclotted blood was present in the pericardial sac in addition to a blood clot which weighed 75 grams.

Figure 3.4 Photographs of the left heart chambers of dog # 6 which died of acute cardiac tamponade. Severe chronic valve disease was present and ruptured primary chordae tendineae to the anterior mitral leaflet were found. Typical jet impact lesions caused by a regurgitant stream of blood were visible on the posterior left atrial wall. Diagonally, above this region, a fibrosed endocardial split was present. Perpendicular to the healed split were two recent endomyocardial splits about one cm. long. The split nearest the ruler in the close-up photograph communicated with the epicardial split in figure 3.3. The other recent split was associated with a corresponding area of subepicardial hemorrhage near the ligament of Marshall in figure 3.3.





D. Acquired atrial septal defect

caused by

left atrial splitting

Case History

Table 3.7 Clinical laboratory results

Table 3.8 Intracardiac pressures and oxygen determinations

Fig. 3.5 Lateral radiographs

Fig. 3.6 Dorsoventral radiographs

Fig. 3.7 Angiocardiograms

Fig. 3.8 Gross pathology - left atrial aspect

Fig. 3.9 Gross pathology - right atrial aspect

Case History of Dog # 10

On March 20, 1964, a 7½-year-old male Cocker Spaniel was examined because of heavy breathing, panting, restlessness at night, and decreased exercise tolerance over a two-month-period. A diagnosis of congestive heart failure had been made by the referring veterinarian who instituted digitalis therapy. The previous medical history consisted of distemper and hepatitis vaccinations and treatment of intestinal parasites.

Examination revealed an alert, intact male dog weighing 31 pounds in apparently good physical condition and of normal body conformation. No edema, ascites, or venous distension was present. Harsh grade 3 (out of 5) holosystolic murmurs of mitral and tricuspid insufficiency were heard. No precordial thrills were palpable and no pulmonary rales were heard. High amplitude R waves (3.2 millivolts) in lead II were present in an electrocardiogram. In thoracic radiographs, moderate generalized cardiac enlargement and moderate to severe left atrial enlargement were observed (Fig. 3.5a and 3.6a). Evidence of pulmonary congestion was also present. No microfilaria were found in a centrifuged blood sample. One nucleated red blood cell was reported in the hemogram along with a normal total white blood cell count and differential (Table 3.7).

A diagnosis was made of mitral and tricuspid insufficiency due to chronic valve disease resulting in cardiac enlargement in general and left atrial enlargement in particular.

Treatment with digitoxin was stopped for a trial period of 10 days during which the dog began coughing more frequently. Digoxin therapy was then instituted and after a brief episode of toxicity, the signs of congestive heart failure were nearly eliminated with only infrequent coughing noted.

On April 6, 1964, two separate atrial premature beats and one 3 beat Wenckebach series were recorded in an electrocardiogram. Six months later, on October 10, 1964, the mitral insufficiency murmur had increased to grade 4 intensity and was associated with a precordial thrill. The heart size was slightly increased in radiographs. The electrocardiogram still had high amplitude R waves; one ventricular premature beat was recorded. The dog's general activity and other clinical signs were unchanged, and digoxin therapy was continued at the maintenance level.

Seven months later, on May 8, 1965, the animal was re-examined. The dog had been fairly stable with only one episode of vomiting 2-3 hours after digoxin treatment. Occasional coughing had been noted during the period between examinations. For a few days prior to this examination, the coughing was more frequent (5 times or more per day). Upon auscultation, increased vesicular lung sounds were heard. The mitral insufficiency murmur was still of grade 4 intensity and was associated with a precordial thrill. The tricuspid insufficiency murmur, although still classified as grade 3, was louder and was also associated with a precordial thrill. Marked left ventricular and left atrial enlargement with pulmonary congestion were observed in radiographs. The mucous membranes were very pale, and the dog became slightly cyanotic when restrained for an electrocardiogram. The heart rate was 140 per minute. The P waves were increased in duration (0.06 sec.) as was the QRS complex (0.07 sec.). Atrial activation was apparently originating from the sinus node most of the time; however, evidence of a wandering pacemaker was seen in one lead. One atrial premature beat and two ventricular premature beats were also recorded. Digoxin therapy was continued and chlorthiazide treatment was prescribed for one week.

During this time, the dog improved clinically; however, when examined on May 15, 1965, the mitral insufficiency murmur was of grade 5 intensity and the tricuspid insufficiency murmur was grade 4.

On June 4, 1965, both murmurs were of grade 5 intensity and were audible over the entire thorax including the vertebrae. Ballotement indicated a slight amount of ascites in the ventral 1-2 inches of the abdominal cavity. No edema was present. The lung sounds were normal. Frequent atrial premature beats and paroxysmal atrial tachycardia (3-8 beats) were heard and recorded in an electrocardiogram. Non-premature inverted P waves occurred during a portion of lead II and two ventricular premature beats were recorded. No definite evidence of ascites was detected by radiography. Moderate right heart enlargement and marked left heart enlargement were noted in addition to pulmonary congestion. Blood samples were obtained for a hemogram, and blood urea nitrogen, glucose, cholesterol, SGPT, SGOT, and LDH determinations (Table 3.7).

An additional diagnosis of atrial myocardial disease and possible left atrial splitting with progressive congestive heart failure was made. The digoxin dosage was increased and chlorthiazide therapy was started.

When examined one month later, on July 3, 1965, moderate ascites was present. The owners had noted abdominal enlargement over the preceding 10 days but had detected no change during the last 3-4 days. Coughing was less frequent and weaker. No fainting, and no detectable change in water intake or frequency of urination was noted. In an electrocardiogram, sinus tachycardia (170 per minute) with wide P waves and high amplitude R waves were present. No atrial or ventricular premature beats occurred. Digoxin therapy was continued at the previous higher maintenance level and chlorthiazide dosage was doubled.

Examinations on July 16, August 6, August 23, and September 11, 1965 showed progressive accumulation of ascitic fluid to a marked degree in spite of further increase in dose levels of digoxin and chlorthiazide and institution of a low sodium diet. The weight of the dog fluctuated slightly but was not a reliable guide, since the increasing amount of ascitic fluid was coupled with progressive emaciation. Only one atrial and one ventricular premature beat were recorded out of four electrocardiograms made on the above dates. Sinus tachycardia varied from 160 to 190 beats per minute. The PR interval lengthened beyond the limits of normal probably owing to digoxin therapy. The right ventricular and right atrial portions of the cardiac silhouette became enlarged to a degree equal to the marked enlargement of the left atrium and ventricle (Fig. 3.5b and 3.6b). These changes were associated timewise with the development of a slightly louder tricuspid than mitral insufficiency murmur and a more palpable thrill on the right precordium.

Although the dog was active and alert, he reportedly was no longer able to jump up on a couch because of weakness and marked ascites. The severe abdominal distension was causing difficult breathing, and the dog became reluctant to lie down. For this reason, 1400 cc. of slightly blood tinged ascitic fluid was removed. Although the abdomen was still moderately enlarged, the dog was able to lie down and breathe easily.

A tentative diagnosis of acquired atrial septal defect due to left atrial splitting was made; however, splitting of the second heart sound could not be detected by auscultation or phonocardiography because of the loud holosystolic murmurs of mitral and tricuspid insufficiency.

When examined one month later, on October 16, 1965, marked ascites was again present, and the dog's breathing was shallow and rapid (140 per minute); however, he was still active and alert. No coughing had occurred since the previous examination. A further increase in the markedly enlarged heart was noted in radiographs. One atrial premature beat occurred in leads I and AVF respectively. The heart rate was 145 per minute.

Four days later, on October 20, 1965, atrial fibrillation was present. The ventricular rate was 170 per minute. The owners had noted no change in the dog's activity or condition since the examination four days earlier. An abdominal paracentesis was performed without incident under local anesthesia; 1100 cc. of slightly blood tinged ascitic fluid was removed.

On November 6, 1965, the dog had increased ascites. Moderate tachypnea was noted. No coughing or regurgitation had occurred, and no edema was present.

On November 11, 1965, blood samples were obtained for clinical laboratory studies (Table 3.7). Paracentesis was again performed and 750 cc. of ascitic fluid was removed. Cardiac catheterization was done under local anesthesia. The pressure recordings were consistent with left and right heart failure and marked mitral and tricuspid insufficiency. Blood oxygen saturation studies were consistent with the presence of an atrial septal defect (Table 3.8).

During the next three weeks, the ascites again increased, although the animal's weight declined. Emaciation and weakness progressed, but the dog remained very alert and responsive. He had difficulty going up one step and became unable to maintain posture normally when defecating.

Some increase in frequency of urination was noted when spironolactone was used as a diuretic; however, the dog became anorectic. Prior to this, his appetite had always been excellent.

The dog was last examined on December 6, 1965 at the age of 9½ years. This was 23 months after the initial signs of congestive heart failure had been noted. He was severely emaciated and although he weighed 21 pounds, marked ascites was present and his actual body weight was probably under 15 pounds as contrasted to 31 pounds initially. His mucous membranes were very pale and he had difficulty supporting his weight on his hindlegs. Urine and feces staining were noted along with ulcers around the rectum. Before euthanasia was performed, cardiac catheterization was again done, and the previous findings were confirmed. In addition, lateral and dorsoventral angiocardiograms were made after injecting a contrast medium into the main pulmonary artery. These showed the absence of pericardial effusion and the presence of a left-to-right intracardiac shunt (Fig. 3.7).

Pathologic examination confirmed the presence of an acquired atrial septal defect. A large fibrosed endocardial and subendocardial split extended from the left atrial appendage to the fossa ovalis parallel to the mitral annulus. A one cm. long oval atrial septal defect was present where the split extended over the thin part of the fossa ovalis. Smaller scars of other healed splits also were present as were several broken chordae tendineae and chronic valve disease (Fig. 3.8).

TABLE 3.7

CLINICAL LABORATORY DATA ON DOG #10 WITH ACQUIRED ATRIAL SEPTAL DEFECT

	3-20-64	6-4-65	7-3-65 & (8-23-65)	11-11-65
Hematocrit	52	54	43	37
Hemoglobin	18.4	18.4	14.9	12.4
Red blood count	7,650,000	7,950,000	6,106,000	5,254,000
White blood count	9,800	12,000	6,800	9,100
Segmented	69	79	64	72
Nonsegmented	2	-	7	6
Lymphocyte	22	14	24	22
Monocyte	3	1	-	-
Eosinophil	4	6	5	-
Basophil	-	-	-	-
Cholesterol		261	292 (300)	292
Glucose		89	-	118
B.U.N.		14	-	21
SGPT		50	(42)	38
SGOT		32	(29)	22
LDH		670	(580)	200
Misc.	Microfil. negative 1 Nuc. RBC			Mod.Aniso- and Poikilocytosis. Few Howell Jolly Bodies.

Blood culture 12-6-65 negative

TABLE 3.8

CATHETERIZATION DATA ON DOG #10 WITH

ACQUIRED ATRIAL SEPTAL DEFECT

Systolic*/Diastolic (mean) blood pressures in mm. Hg.				% O_2 saturation	
Site	11-11-65	12-6-65	12-6-65**	11-11-65	12-6-65
Azygos vein	45/10 (18)	40/10 (--)	-	54	71
Cranial vena cava	45/12 (18)	40/10 (16)	-	53	51
Caudal vena cava	25/12 (17)	32/12 (17)	-	60	68
Right atrium	38/11 (17)	40/10 (16)	20/9 (12)	74	80
Right ventricle	70/10	72/15	46/8	78	80
Main pulm. art.	70/20 (48)	65/20 (38)	45/17 (25)	78	79
Left atrium	-	-	25/10 (15)	-	99**
Left ventricle	-	-	98/8	-	-

* Systolic values at venous and atrial sites represent V wave pressures.

** Recording made during thoracotomy and 30% oxygen positive pressure ventilation.

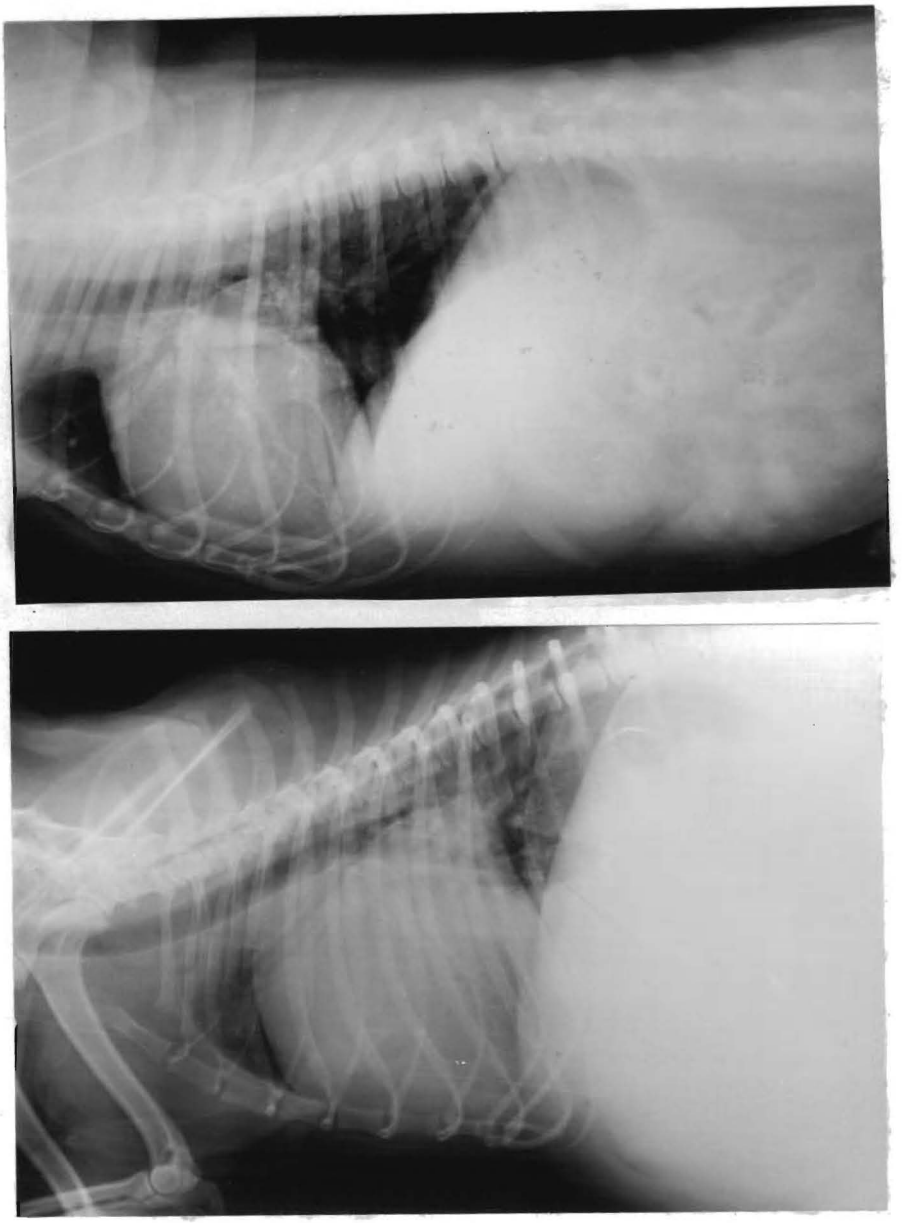


Figure 3.5 Lateral radiographs of dog # 10*

A: At the time of initial examination, March 20, 1964, moderate generalized cardiac enlargement was present with particular dorso-caudal prominence of the left atrium. This caused dorsal elevation of the trachea and left mainstem bronchus and classical forking of the tracheal bifurcation.

B: Radiograph made 17 months later on August 23, 1965. Severe ascites was present in addition to marked generalized cardiac enlargement.

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* In these and all subsequent lateral radiographs, the dog's head is to the reader's left.

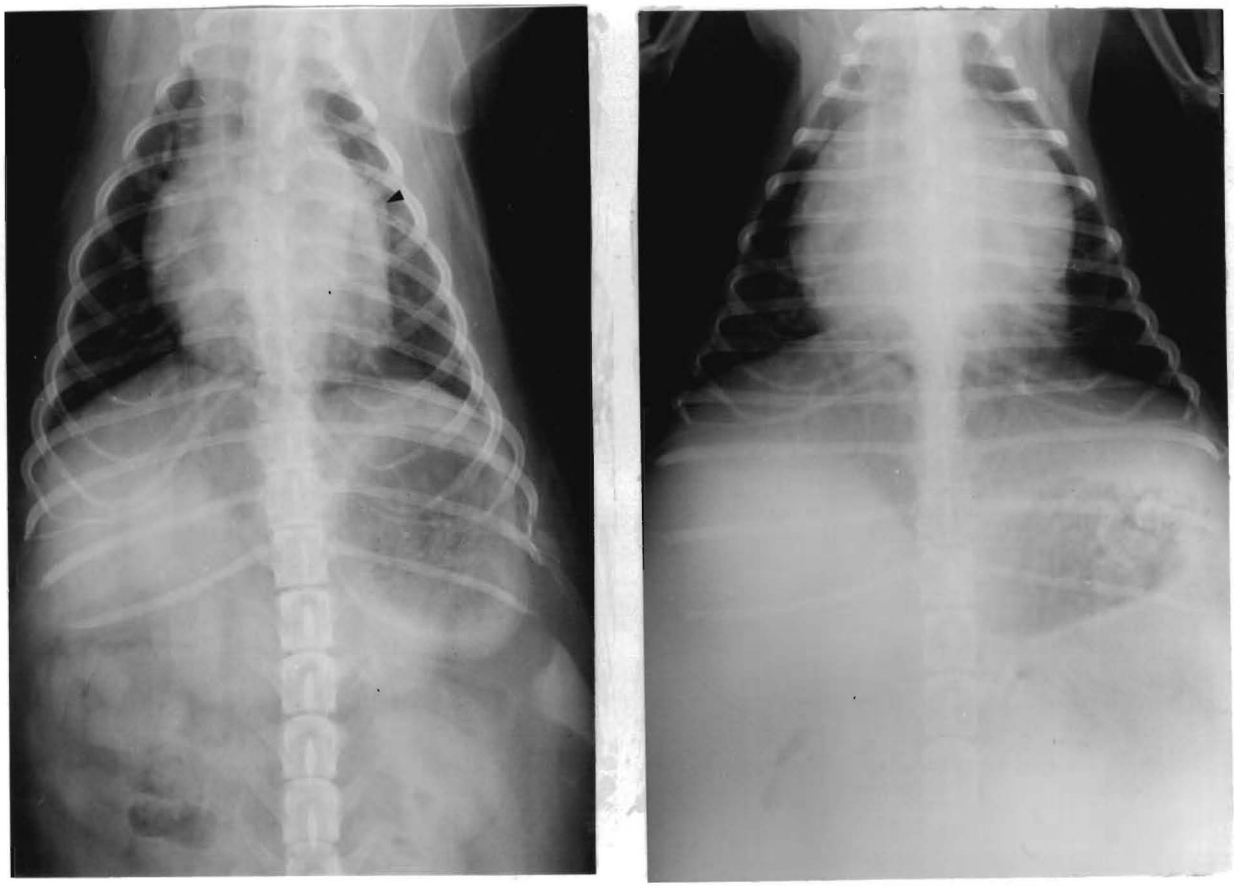

Figure 3.6 Dorsoventral radiographs of dog # 10.*

A: In the initial radiograph made on March 20, 1964, generalized cardiac enlargement was apparent. Particular enlargement of the left atrial appendage was indicated by a leftward bulge at about 2-3 o'clock (arrow).

B: Subsequent radiograph made on August 23, 1965. Marked ascites and generalized cardiac enlargement were evident. The left atrial appendage bulge was more apparent, as was greater dilatation of the right side of the heart.

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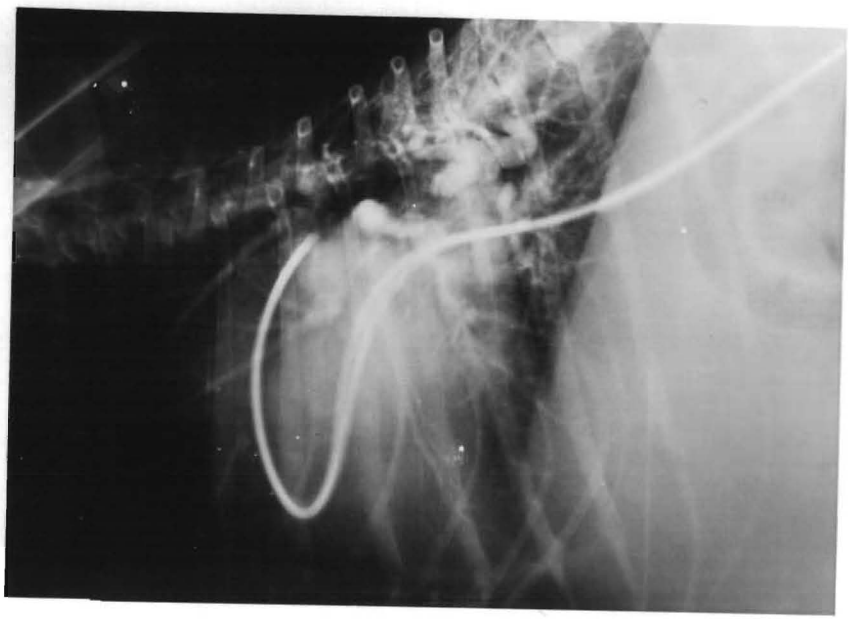
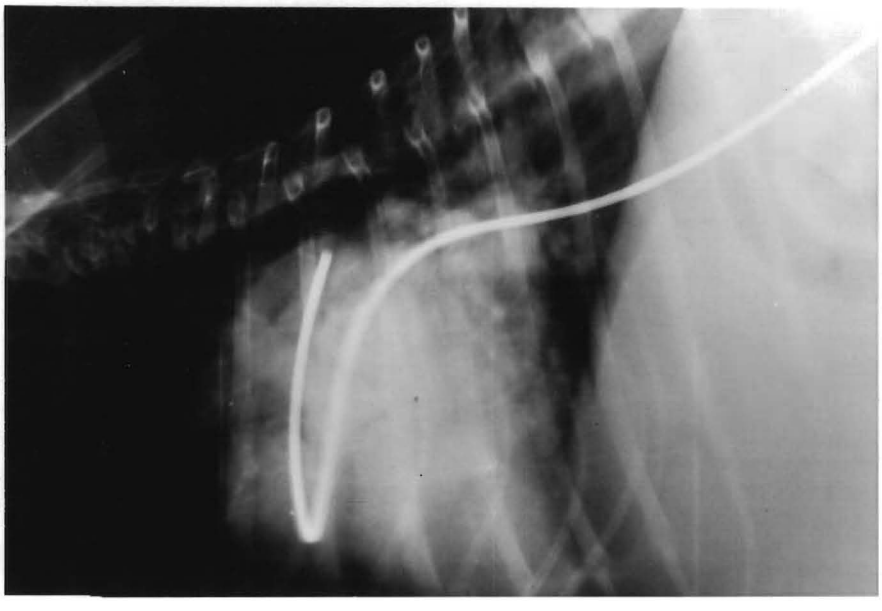
* In these and all subsequent dorsoventral radiographs, the dog's left is to the reader's right.

Figure 3.7 Pulmonary artery injection angiocardiograms of dog # 10 made on December 6, 1965 after two catheterization studies had indicated the presence of an atrial septal defect.

A: In a lateral angiocardiogram, made 2 seconds after the start of an injection of a contrast medium into the pulmonary artery, tortuous, dilated pulmonary veins were visualized. No contrast medium was present in the right ventricle where the sharp ventral curve of the catheter was situated.

B: In a lateral angiocardiogram, made 6 seconds after the start of the injection, the left heart chambers and aorta were opacified as were the right atrium and ventricle. The right atrium could be seen above the level of the superimposed aortic sinus of Valsalva and the ascending aorta. The right ventricle was visible below the level of the aortic sinus of Valsalva in the region where the catheter appeared sharply bent before entering the nonopacified pulmonary artery. The individual chamber sequence of opacification of the right atrium and right ventricle could not be determined from the angiocardiograms; therefore, the level of the left-to-right shunt could not be determined from this study.





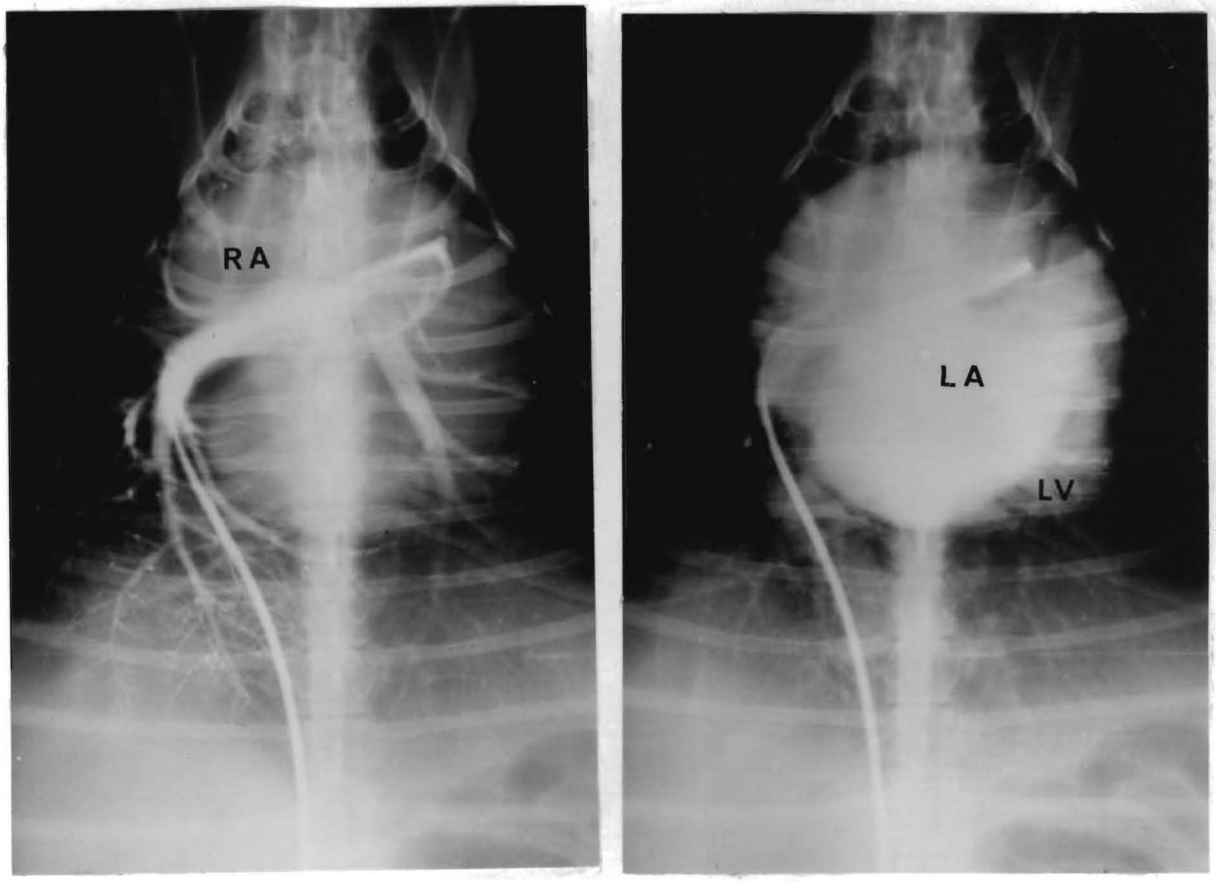


Figure 3.7 (continued)

C: In a dorsoventral angiocardiogram made ½ second after the start of another injection, the pulmonary arteries were opacified. The cranial and rightward segments of the cardiac silhouette in the region of the right atrium (RA) were not clearly visible.

D: In an angiocardiogram made 8 seconds later, the left atrium (LA) and apex of the left ventricle (LV) were visible. The cranial and rightward segments of the cardiac silhouette were sharply defined and the chamber area of the right atrium was slightly more opaque. These observations supported the diagnosis of left-to-right shunting through an atrial septal defect.

A tracing of the original film used for Fig. 3.7D demonstrated better the contribution of the dilated left atrium to the dorsoventral cardiac silhouette. It is presented in Figure 4.10 in the appendix on radiology of left atrial enlargement.

Figure 3.8 Postmortem photograph of the left heart chambers of dog # 10. Left ventricular and left atrial dilatation were noted at necropsy in addition to chronic valve disease and ruptured chordae tendineae of the anterior mitral leaflet. A fibrosed endocardial split, covered by endothelium, was observed in the caudal left atrial wall parallel to the mitral annulus. Where the split extended into the septum, an atrial septal defect was present. Other healed splits in the endocardium were present.

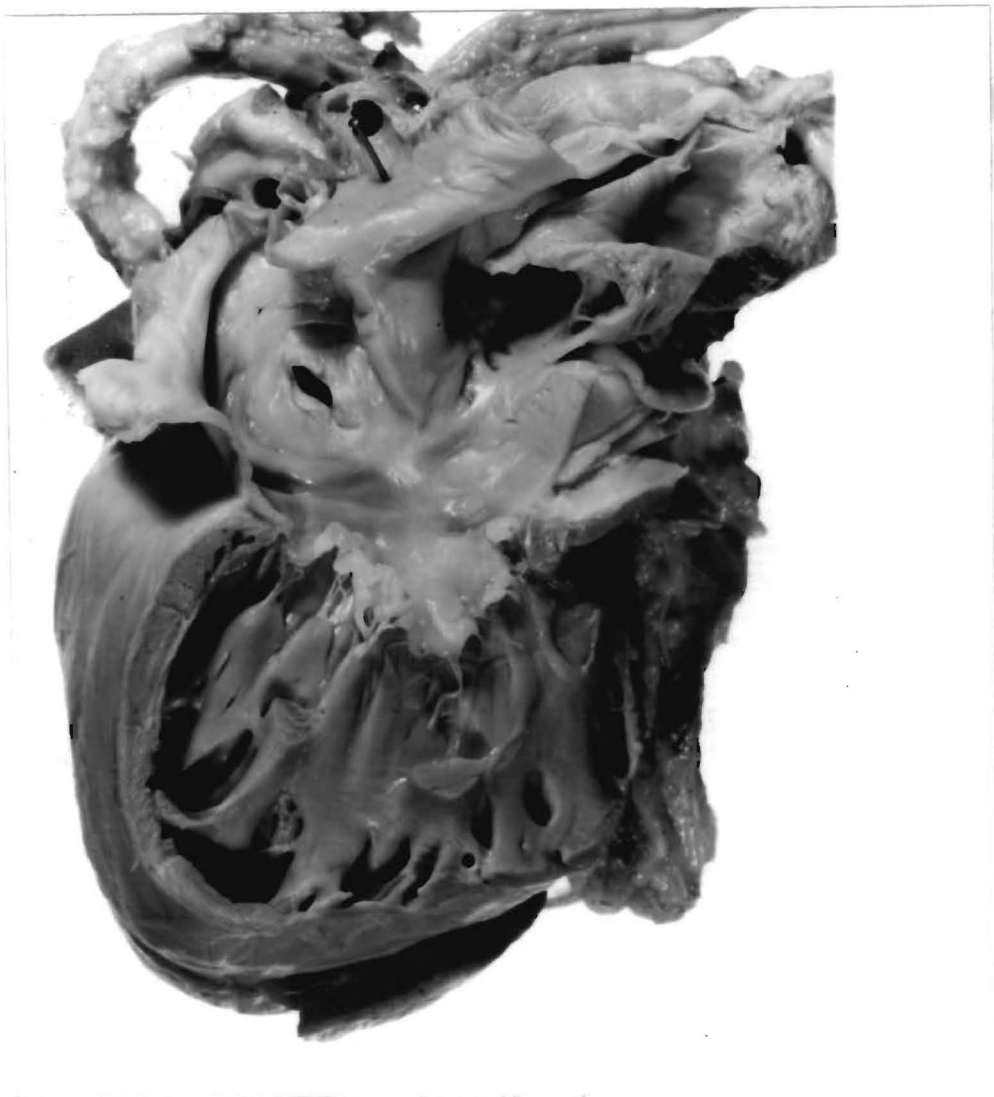


Figure 3.9 Postmortem photograph of the right heart chambers of dog # 10. Chronic thickening of the septal leaflet of the tricuspid valve as well as thickening of several chordae tendineae were observed. An oval atrial septal defect with sharp edges could be seen in the usually translucent portion of the fossa ovalis.

E. Nonperforating left atrial splits.

Case History

Figure 3.10 Subepicardial hemorrhage

Figure 3.11 Endocardial and endomyocardial splits

Case history on dog # 19

A three year old male Dachshund was brought to the Veterinary Clinic on August 8, 1958 because of chronic dermatitis of 2 years duration. A grade 2 (out of 5) intensity holosystolic murmur was detected in the mi-ral area. Thoracic radiographs revealed mild left-sided cardiac enlarge-ment. An electrocardiogram and hemogram were within normal limits. A diag-nosis of mitral insufficiency (chronic valve disease) was made. Five months later, the murmur was increased slightly in intensity; however, it still was not classed as grade 3.

At seven years of age, the dog started coughing, especially when exer-cising, and fainted twice during a two week period of time prior to exami-nation by a local veterinarian. A diagnosis of congestive heart failure was made and the dog was digitalized. The frequency of coughing was subsequent-ly reduced until a few days prior to examination in the heart station on August 9, 1963 at the age of 8 years.

At this time, the dog had grade 4 holosystolic murmurs and precordial thrills strongest in the mitral and tricuspid areas; however, a holosysto-lic murmur could be heard anywhere on the dog's thorax. The first heart sound was very loud, and the second heart sound was normal. Sinus tachy-cardia at a heart rate of 180 beats per minute was noted in an electrocar-diogram. The P waves were prolonged, and the P wave vector was directed more dorsally than normal. The P wave duration equaled the duration of the PR segment. These changes were considered evidence of left atrial enlarge-ment which was confirmed by thoracic radiography. Left and right ventricu-lar and right atrial enlargement were also seen in addition to evidence of pulmonary congestion in the radiographs.

The digitalis dosage was increased, and the owners were advised to limit the dog's activity. The dog died suddenly on September 3, 1963, after a 3 day period of severe coughing (mostly during the night time). The owners reported that at 6 A.M. the dog was not coughing but appeared weak, and was breathing with difficulty. Blood tinged froth was noted at the corner of the dog's mouth. Vomitus was present on the floor near the dog. It died at 6:15 A.M. from apparent pulmonary edema.

The dam of this dog had been examined in the heart station at the age of 6 years and no sign of heart disease was found. She was euthanatized at the age of 9 years for apparently non-cardiovascular reasons. Nothing was known about the sire. One offspring of the dog No. 19 had had "a heart attack" and had been placed on digitalis therapy by a local veterinarian. Based upon dates and time factors, this offspring must have been less than 7 years old.

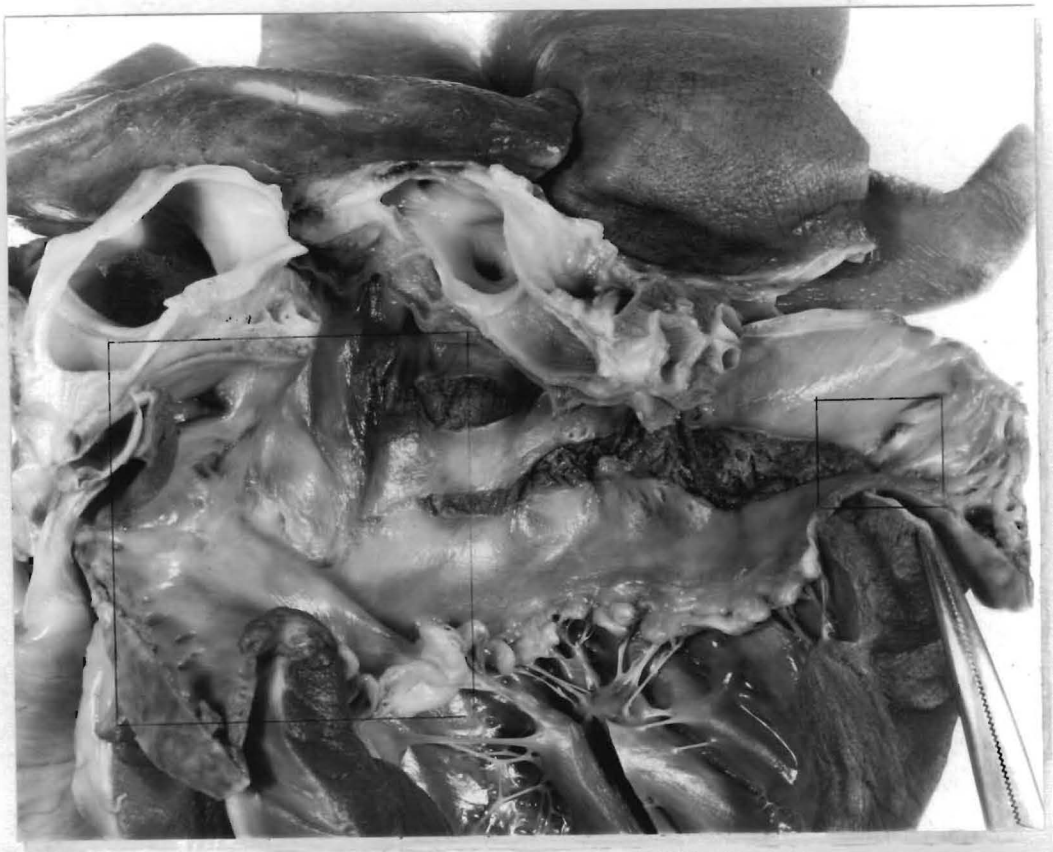
Postmortem examination of dog # 19 revealed marked pulmonary edema. Slightly more than a normal amount of clear serous pericardial fluid was present. All chambers of the heart were enlarged, and a transverse band of subepicardial hemorrhage was present in the caudal wall of the left atrium (Fig. 3.10). This area was underneath the largest of several endocardial and endomyocardial splits of the left atrium (Fig. 3.11). Severe chronic valve disease and several broken chordae tendineae were present.

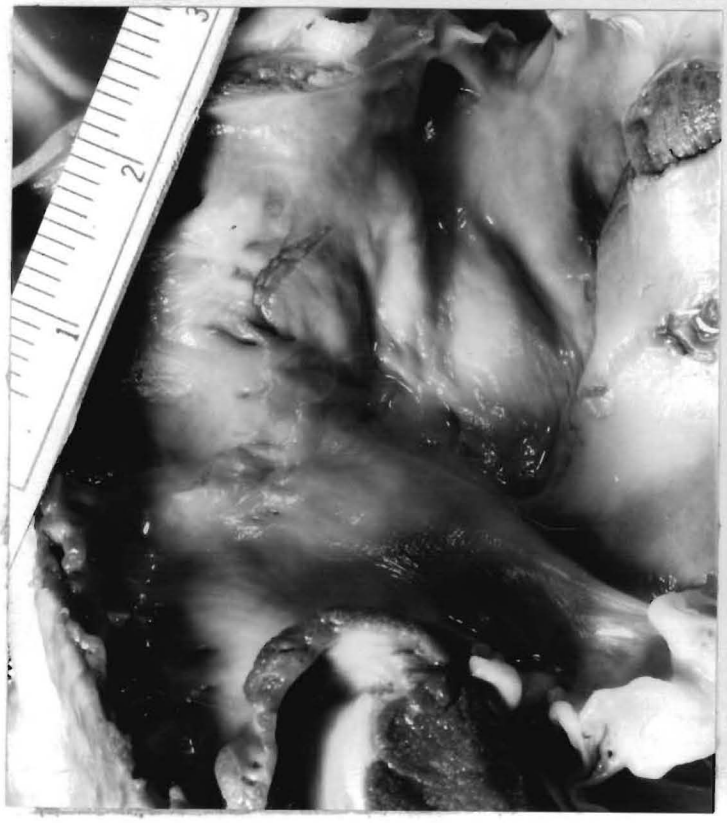
Figure 3.10 Caudal aspect of the left atrium of dog # 19. The transverse band of subepicardial hemorrhage was produced by the largest of several nonperforating endomyocardial splits (see next illustration).

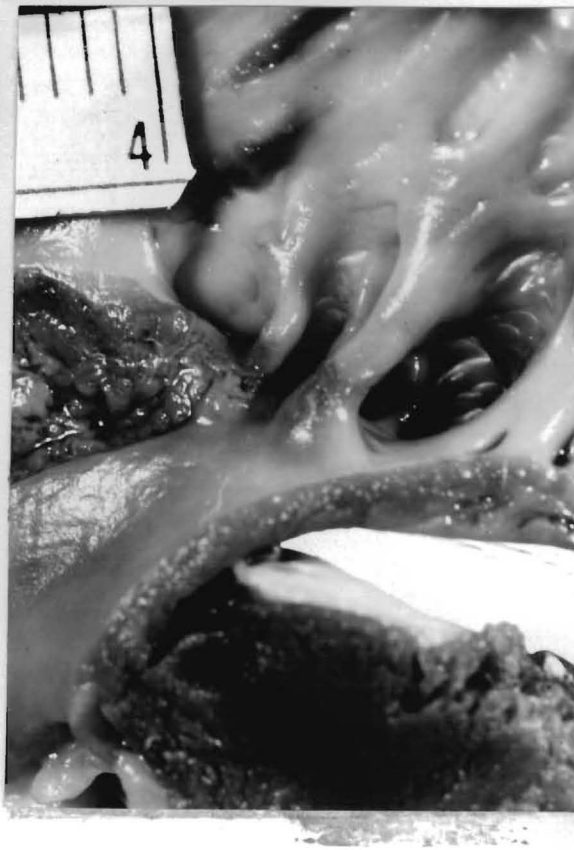
Figure 3.11 Mitral valve and left atrium of dog # 19 with severe chronic valve disease and marked left atrial dilatation. Several ruptured primary chordae tendineae of the anterior mitral leaflet (large block) were observed but were not well photographed. The unsupported free edge of the leaflet was rigid and directed toward the left atrium; a thick fibrous ridge extended between intact secondary chordae tendineae on the ventricular aspect of the leaflet. These changes were considered evidence for the existence of ruptured chordae tendineae over a long period of time.

A large (6 cm. long), recent, endomyocardial split was present in the caudal left atrial wall extending from the interatrial septum into the appendage. In the same plane as the large lesion, but separated from it, an endocardial split could be seen involving a pectinate muscle band in the appendage (small block). At the opposite extent, the large split terminated as a partial thickness endocardial separation which did not have superimposed thrombus (large block). Just above this area, several small (1-2 mm. long) partial endocardial splits were visible.

In the photograph of the cranial wall of the left atrium, three types of lesions could be seen (large block). Immediately below the ruler segment, a white, raised, jet impact lesion was present. At the ventral edge of this was a recent thrombus covered endocardial split. Parallel and 3 mm. ventral to the recent split was a scar from a previous endocardial split. The latter was 2.5 cm. long and 0.3 cm. wide at its center.







F. Endocardial degeneration with lipid deposition

Case History

Figure 3.12 Gross left atrial pathology

Figure 3.13 Microphotograph of endocardium

Figure 3.14 Ruptured chordae tendineae

Figure 3.15 Microphotograph of chordae tendineae

Case History of Dog # 15

Dog # 15 was an eleven-year-old male Cocker Spaniel presented for examination on March 14, 1964 primarily because a male offspring had heart disease and was being periodically examined at this institution. Radiographs of the latter dog are shown in Figures 4.7-4.9.

Since 6 years of age, dog # 15 had coughed occasionally after exercise and excitement and had been checked every 6 months for a "heart condition" by a local veterinarian. Physical examination on March 14, 1964 revealed a grade 3 (out of 5) intensity holosystolic murmur and a slight precordial thrill in the mitral area. Increased vesicular lung sounds were also heard. In an electrocardiogram, sinus tachycardia (185/min.), high amplitude R waves, and one ventricular premature beat were recorded. Radiographic signs of pulmonary congestion, left ventricular and left atrial enlargement were found. The diagnosis made was chronic valve disease with mitral insufficiency, left ventricular hypertrophy, and early congestive heart failure. Digoxin therapy was initiated, and the owner was advised to return the dog in 6 months for a follow-up examination.

Ten weeks later, the dog was presented for examination because of increased coughing (especially at night), decreased activity, anorexia, and progressive abdominal enlargement over the preceding 10 day period. Vomiting and diarrhea had been observed in the last 3 days of this period.

Physical examination on May 25, 1964 revealed a grade 4 holosystolic murmur and diffuse palpable thrills in both the mitral and tricuspid valve precordial areas. Moist rales were heard over all lung fields. Marked ascites was present as well as ventral abdominal and hindleg edema. The heart rate was 190 beats/minute and irregular.

Evidence of atrial fibrillation was noted in an electrocardiogram in addition to several ventricular premature beats. The R wave amplitude in lead II was 75 % of that noted in the previous electrocardiogram. Radiographically, marked generalized cardiac enlargement was noted along with evidence of pleural effusion. Although somewhat rounded, the cardiac silhouette was not considered quite characteristic of significant pericardial effusion. In addition to the initial diagnosis, a tentative diagnosis of left atrial splitting with possible hemopericardium was made.

The dog was hospitalized for nine days prior to euthanasia. The latter was performed at the owner's request because of the poor prognosis and lack of response to treatment.

At necropsy, marked generalized cardiac enlargement was noted and only 30 cc. of clear serous pericardial effusion was present. Several broken mitral chordae tendineae were observed along with severe chronic valve disease. The left atrial endocardium was white, diffusely opaque and was interrupted in several locations by large endocardial and endomyocardial separations with thrombi on the exposed areas of myocardium. Upon closer examination of the fixed specimen, a yellowish segment was noted in two intact chordae tendineae near the ruptured primary chordae tendineae inserting into the anterior mitral leaflet. The yellowish chordae tendineae were cut longitudinally and processed by frozen section technique. Histologic examination after staining with oil-red-O revealed large amounts of lipid material primarily in the central collagenous portions of the grossly abnormal chordae tendineae segments. Varying amounts of endocardial lipid material were also found in preliminary frozen section studies of the left atrium.

Figure 3.12 Postmortem photograph of the left heart chambers of dog # 15. Chronic mitral valve disease, ruptured chordae tendineae, left atrial jet impact lesions, and several nonperforating endomyocardial splits of the left atrium were observed at necropsy.

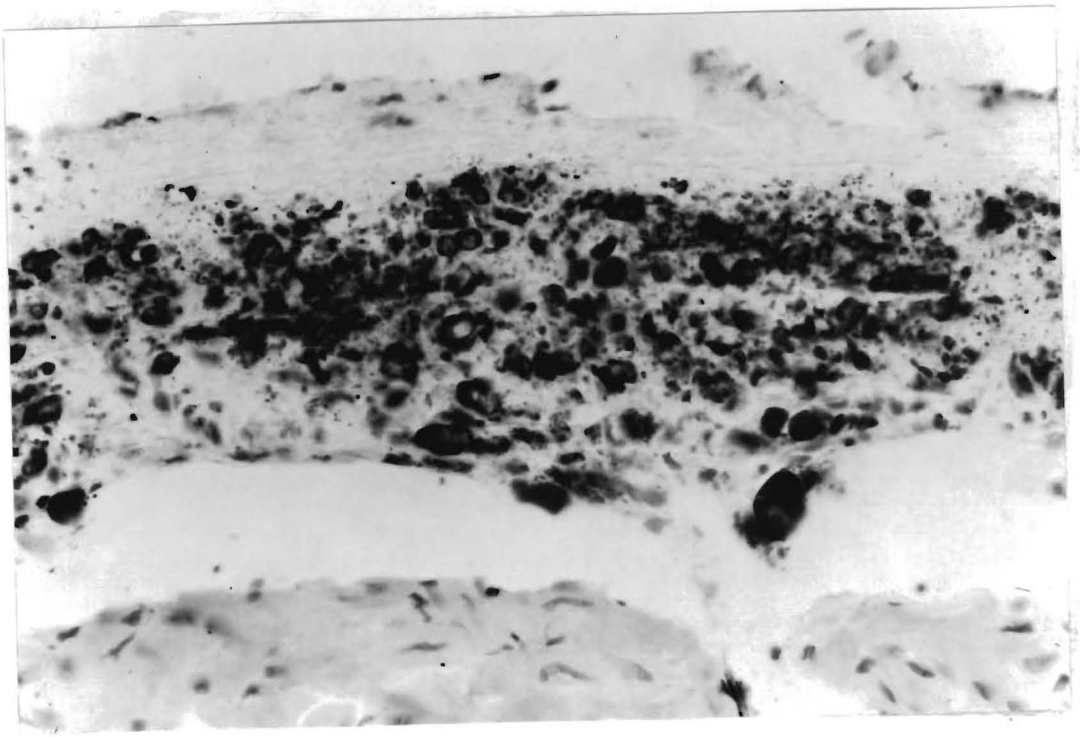


Figure 3.13 Microphotograph of a focal area of lipid deposition in the left atrial endocardium of dog # 15. The endocardium was generally thickened and a zone of lipid deposition was observed in several areas in the subendocardial region. In some focal areas, the sudanophilic material was present in all layers of the endocardium (oil-red-O stain; 415x).

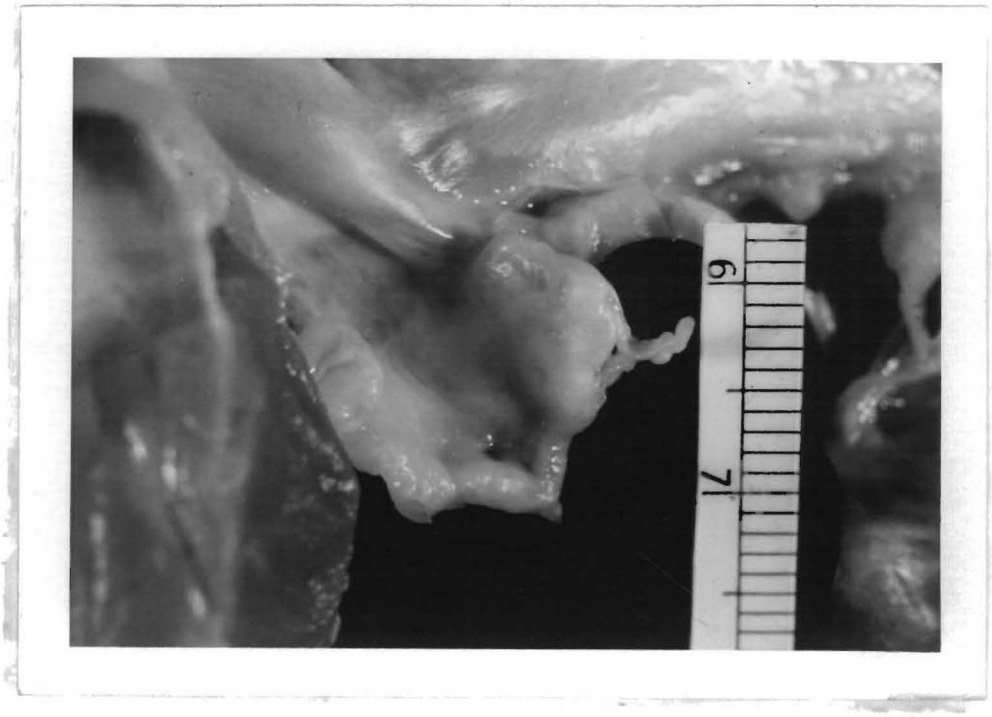


Figure 3.14 Close-up photograph of the anterior mitral leaflet and several ruptured chordae tendineae in dog # 15.

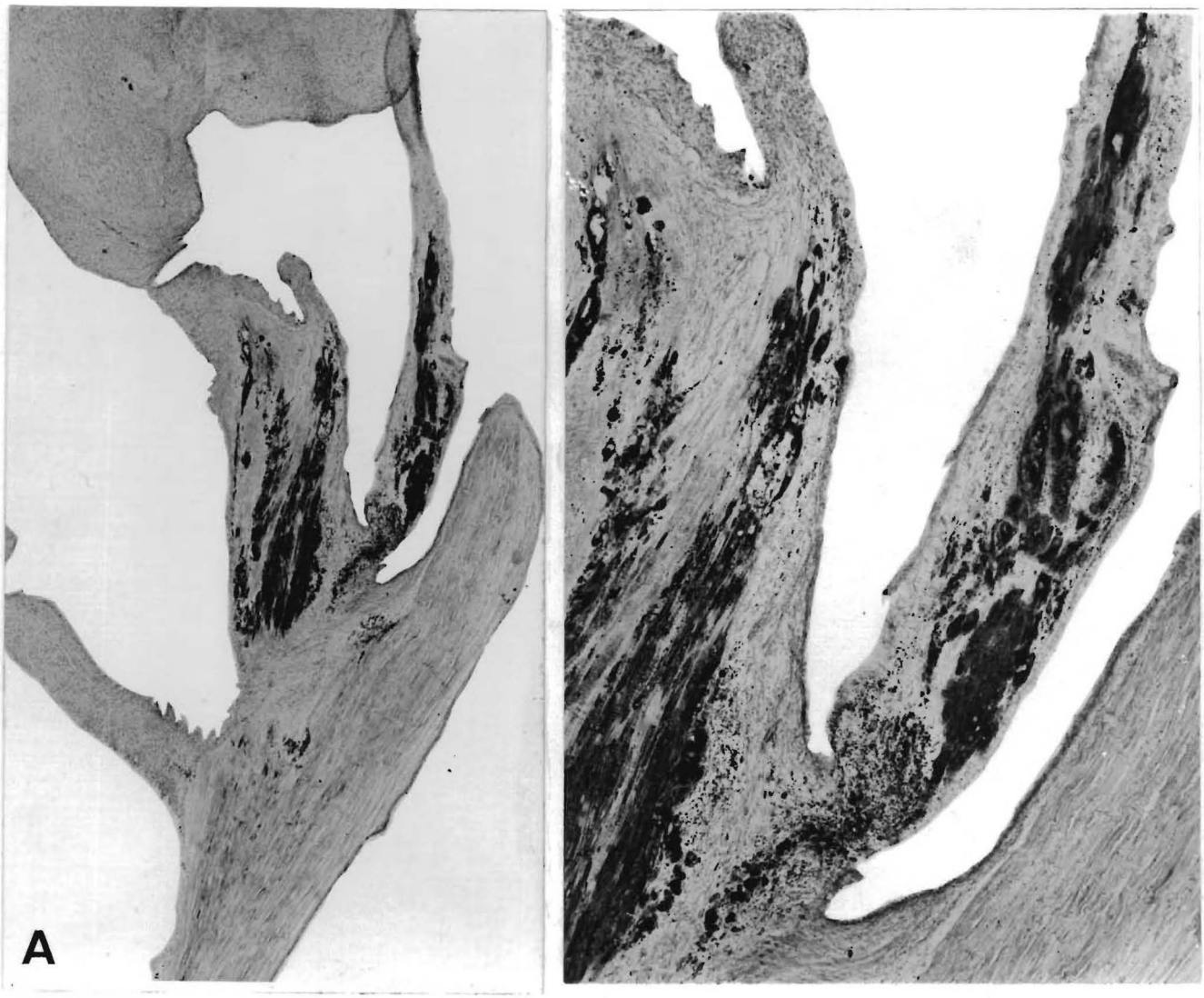


Figure 3.15 Microphotographs of nonruptured chordae tendineae of the anterior mitral valve leaflet in dog # 15. These were located underneath the most leftward chorda tendina visible in the previous illustration. The two chordae tendineae segments which contain large amounts of lipid material were selected for longitudinal sectioning by frozen technique because they appeared yellow upon gross examination. (Oil-red-O stain; @ 30X and 80X)