

University of Pennsylvania **ScholarlyCommons**

Departmental Papers (Vet)

School of Veterinary Medicine

9-15-1961

Clinico-Pathologic Conference: Pulmonic Stenosis and Patent Foramen Ovale in a Dog

James W. Buchanan University of Pennsylvania, jwb@vet.upenn.edu

David K. Detweiler University of Pennsylvania

Klaus Hubben University of Pennsylvania

Follow this and additional works at: https://repository.upenn.edu/vet papers



Part of the Veterinary Medicine Commons

Recommended Citation

Buchanan, J. W., Detweiler, D. K., & Hubben, K. (1961). Clinico-Pathologic Conference: Pulmonic Stenosis and Patent Foramen Ovale in a Dog. Journal of the American Veterinary Medical Association, 139 (6), 701-707. Retrieved from https://repository.upenn.edu/vet_papers/53

Appendix 6.1 of James W. Buchanan's dissertation Chronic Valve Disease and Left Atrial Splitting in the Dog

This paper is posted at ScholarlyCommons. https://repository.upenn.edu/vet_papers/53 For more information, please contact repository@pobox.upenn.edu.

Clinico-Pathologic Conference: Pulmonic Stenosis and Patent Foramen Ovale in a Dog

Disciplines

Medicine and Health Sciences | Veterinary Medicine

Comments

Appendix 6.1 of James W. Buchanan's dissertation Chronic Valve Disease and Left Atrial Splitting in the Dog

CLINICO-PATHOLOGIC CONFERENCE

From the School of Veterinary Medicine University of Pennsylvania

Presentation of Case

Dr. J. W. Buchanan. *-On Nov. 7, 1960. a male Miniature Poodle 8 months old was admitted to the University of Pennsylvania veterinary hospital. The dog had been operated on for an umbilical hernia in July. 1960, at which time it was given antidistemper-hepatitis serum. Since that time, severe attacks of dyspnea had occurred. sometimes as often as three times a day. They were characterized by rapid, openmouthed, deep respirations which were associated with generalized ataxia, weakness in the hindlegs, occasional fainting, restlessness, and drooling of foamy material. The dog had intermittent ascites but no coughing.

On physical examination, the body temperature was 102 F., respiration rate 44 (labored), heart rate 152 (no pulse deficit),

*Dr. Buchanan is instructor in cardiology.

and blood pressure 115/70. There was a loud (grade 4), systolic murmur with a palpable thrill at the left third to fourth intercostal space, as well as a grade 3 systolic

Prepared by:

R. S. Brodey, D.V.M., M.Sc. (V.S.)

Assistant Professor of Veterinary Surgery

J. E. Prier, D.V.M., Ph.D.

Associate Professor of Virology

murmur with a thrill at the right third intercostal space. Cyanosis and loud, moist rales in all lung fields were also present. There was no ascites. The other organs and systems were not abnormal. The mean

LABORATORY DATA

Data	11-7-60	12-2-60	Percent 02 saturation		Pressure (mm. Hg)
Total white				-	001140
blood cells	20,700	9,700	Aorta	75	100/65*
Segmented					
neutrophils	67%	65%	Lt. ventricle	71	114-128/6-8
Nonsegmented					
neutrophils	2%	5%	Lt. atrium	66	•••••
Lymphocytes	19%	11%	Rt. atrium	51	
Monocytes	11%	16%	Rt. ventricle	52	190/10
Eosinophils	1%	3%	Cranial	56.5	•••••
	(8.5)	100 May 1	vena cava		
Packed cell					
volume	53%	46%	Caudal	45	
		/ 0	vena cava		
Hemoglobin	20.7 Gm./	14.7 Gm./			
	100 ml.	100 ml.			
3 nucleated red	blood cells/100 v	white blood cells;	micro-		
		n: light trichuriasi			

^{*}Not determined simultaneously.

electrical axis in the electrocardiogram was plus 250 degrees. Marked right and some left cardiac enlargement was seen on survey radiographs. Angiocardiography was performed by inserting a catheter into the right jugular vein. There was a prominent bulge in the region of the cranial heart base, a thickened right ventricle, and the presence of radiopaque dye in the aorta within three seconds after injection. Right-and left-side heart catheterizations were performed November 22.

Following these diagnostic procedures, the temperature elevations which developed were controlled with antibiotic therapy. Intermittent bloody diarrhea also was a problem but did not seriously affect the animal. The dog did not have noticeable dyspnea while in the hospital, and it was not digitalized until December 8 when coughing and heavier respirations were noticed.

Case Discussion

Dr. D. K. Detweiler.*—The first thing to note in this case is that the condition occurred in a young dog. This immediately makes one think of a congenital lesion. The history reports clinical signs referable to the respiratory tract and evidence of failure of the right ventricle (intermittent ascites). The loud moist rales in all lung fields suggest the possibility of a left-sided hear't lesion. The hematologic findings suggest polycythemia, since the hemoglobin exceeds 20 Gm./100 ml. of blood. Since cyanosis was observed during the initial physical examination, it is possible that we are dealing with polycythemia secondary to chronic anoxemia. However, in the second blood study the hemoglobin level decreased. The history of bloody diarrhea could account for this significant change in hemoglobin level between the two determinations. The presence of a loud systolic murmur at the left third or fourth intercostal space in a young dog immediately suggests two possibilities based on the prevalence rates of congenital heart disease in the dog. The most common congenital lesion in the dog is pure pulmonic stenosis. This produces a harsh systolic murmur. often in the third left intercostal space and in the third right intercostal space at the edge of the sternum. Thus, a diagnosis of pulmonic stenosis would be consistent with these auscultatory findings. However, aortic stenosis is also not rare in the dog. It occurs as a congenital lesion which may produce a harsh systolic murmur in the same general area on the left side of the thorax. Thus the auscultatory findings can only be considered suggestive but not diagnostic.

We may now add to that the electrocardiographic findings. The mean electrical axis in the electrocardiogram has rotated markedly to the right. This indicates marked right ventricular enlargement; aortic stenosis would cause left ventricular enlargement. Thus far the findings favor a diagnosis of pulmonic stenosis.

The presence of cyanosis does not fit in with the diagnosis of pure pulmonic stenosis and must be explained. Ordinarily, in a young animal, cyanosis in the presence of heart disease suggests the possibility that there is a "right to left" shunt in which venous blood bypasses the lungs and reaches the systemic arterial circulation.

Now, turning to the radiographic and angiocardiographic findings, the first thing I notice in the right lateral thoracic radiograph is that the cardiac silhouette appears to be longer than normal in its dorsoventral diameter. This is indicated by the fact that the trachea is parallel with the spine; that is, the normal angle between the trachea and the spine has been decreased. Second, at the terminal end of the trachea, there is a definite bowing upward of the trachea as it passes over the region of the right atrium. This suggests enlargement of the right ventricle or the right atrium or both. Third, the two mainstem bronchi are somewhat separated so that they form a V-like structure. This is a change that we often see when there is enlargement of the left atrium. The cranial border of the shadow is straightened to some extent, suggesting enlargement of the right side of the heart. The caudal border of the heart is also straightened to some extent suggesting left-sided heart enlargement. The dorsoventral radiograph is confusing to some degree. Two findings usually associated with pure pulmonic stenosis are absent. First, poststenotic dilatation of the pulmonary artery is usually seen as an enlargement along the left cranial border of the heart. Second, the right cranial border of the cardiac silhouette often has an an-

^{*}Dr. Detweiler is associate professor of pharmacology.

gular appearance which is probably caused by massive enlargement of the right atrium. However, a third finding is present—that of a greatly enlarged cardiac silhouette to the right of the midline. This view of the heart does not strongly support the diagnosis of pure pulmonic stenosis; however, it is not inconsistent with it. In summary, these two radiographs indicate enlargement of the entire heart, involving chiefly the right ventricle.

The initial series of angiocardiograms were taken with the dog in the left lateral position. The first angiocardiogram, taken one second after the rapid injection of contrast medium, shows filling of the right atrium and cranial vena cava. In the second angiocardiogram, taken at three seconds, there is contrast medium present in the right atrium, the right ventricle, and possibly in the left ventricle. The pulmonary arteries have begun to opacify and, although it is debatable, I think the aorta is opacifying simultaneously with the pulmonary arteries. The left atrium and pulmonary veins leading into the left atrium appear to have about the same opacity as the aorta. This suggests that the aorta is opacifying at about the same time as the left atrium, obviously an abnormal situation. In the third angiocardiogram, taken at five seconds (Fig. 1), opacification of the aorta and pulmonary arteries is about equal. Some contrast medium is still evident in both ventricles and both atriums. Thus, in this series of angiocardiograms, it appears that the contrast medium has somehow gained access to the left side of the heart shortly after it was injected into the right atrium, since the aorta and pulmonary arteries are opacifying simultaneously. This establishes the presence of a right to left shunt, but I don't think we can be absolutely sure from this series of angiocardiograms where the shunt is located.

The second series of angiocardiograms, also taken with the dog in the lateral position, are extremely revealing with respect to the location of the shunt. In taking these records, the catheter was advanced through the cranial vena cava and the right atrium into the caudal vena cava so that the contrast medium was actually injected into

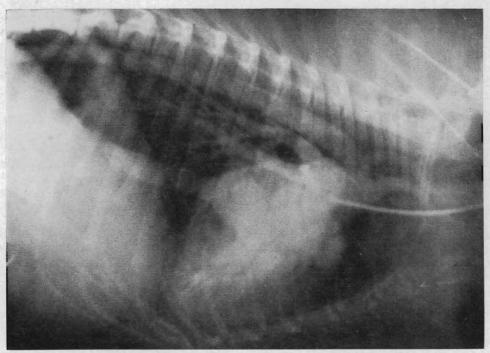


Fig. 1—Left lateral angiocardiogram taken five seconds after injection of radiopaque dye into the cranial vena cava. The aorta and pulmonary arteries are simultaneously opacified, indicating some type of right to left shunting of blood in the heart. The wall of the right ventricle is markedly thickened. The main artery is dilated.

the caudal vena cava. In the first picture, taken at one half second, the contrast medium is seen filling part of the caudal vena cava and the right atrium. The second angiocardiogram, taken at three seconds, indicates that the contrast medium flowed almost immediately into the left ventricle and strongly opacified the aorta (Fig. 2); the aorta contains much more contrast medium than the pulmonary arteries. This flow of blood is reminiscent of the fetal circulation; that is, the oxygenated blood in the fetal circulation approaches the heart through the ductus venosus into the caudal vena cava, into the right atrium, through the foramen ovale into the left atrium, so that much of the blood bypasses the lungs in reaching the aorta. In the fetus, there is relatively little mixing of this oxygenated blood coming from the placenta into the right side of the heart through the cephalic vena cava. Thus, in this case, the pattern of circulation found in the fetus appears to have persisted, because it is evident that the contrast medium has passed into the left ventricle with relatively little mixing with the blood in the right atrium, thus accounting for the fact that the aorta contains so much more contrast medium than the pulmonary vessels in this series. This difference in behavior of the contrast medium when injected into the caudal vena cava, as contrasted with its behavior when injected into the cranial vena cava, suggests that in this heart the foramen ovale has failed to close. It does not rule out the possibility of an atrial septal defect, however, although in the presence of a large atrial septal defect one would anticipate better mixing of the contrast medium in the right atrium than is apparent here.

In the third angiocardiogram, taken five seconds after injection of contrast medium, the aorta still contains some contrast medium. Both atriums, the pulmonary veins, the aorta, and the left ventricle are slightly opacified but at no time in this series of three angiocaridograms have the pulmonary arteries been opacified. It appears that in this series most of the contrast medium bypassed the pulmonary circula-

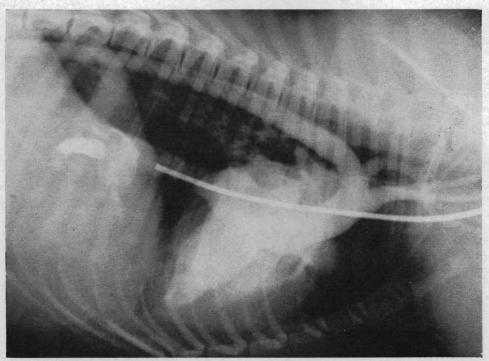


Fig. 2—Left lateral angiocardiogram taken three seconds after injection of radiopaque dye into the caudal vena cava. The left ventricle and aorta are densely opacified while little dye is presented in the right ventricle and pulmonary arteries. This suggests a patent foramen ovale, since blood from the caudal vena cava apparently is reaching the left side of the heart with less mixing than blood from the anterior vena cava.

tion through a defect between the two atria. I can think of no other circumstance which would account for the course followed by the blood in this series of angiocardiograms. The ventricular walls, especially the right, are markedly thickened in these angiocardiograms. The dorsoventral angiocardiograms do not add diagnostic information.

Looking at the oxygen saturation, we see that the blood in the right atrium was less saturated than the blood in the cranial vena cava. Such variations are likely to occur in a normal dog. The blood in the left atrium (66% saturated) contained relatively less oxygen than the blood obtained from the left ventricle and aorta, suggesting that there is a shunt passing unoxygenated blood into the left atrium. Presumably, at the time the sample was taken, this venous blood was not completely mixed with the oxygenated blood also arriving at the left atrium, so that a relatively greater amount of unoxygenated than oxygenated blood was drawn up in the sampling syringe. This would account for the fact that the oxygen content was lower in the blood samples from the left atrium than in those from the left ventricle and aorta.

The blood pressure values were higher in the right ventricle than in the left ventricle or aorta. That the pressure in the right ventricle exceeded that of the systemic pressure was strong presumptive evidence that the interventricular septum was intact.

Adding all of these observations together, the diagnosis would be congenital stenosis of the pulmonic valve (we have not ruled out the possibility of subvalvular stenosis), in the presence of a patent foramen ovale or possibly a very small atrial septal defect. I think it must be patent foramen ovale.

Clinical Diagnosis

- 1) Congenital pulmonic valvular stenosis.
- 2) Patent foramen ovale.

Surgical Findings

Dr. D. F. Patterson.*—A thoracotomy was performed at the right fifth intercostal

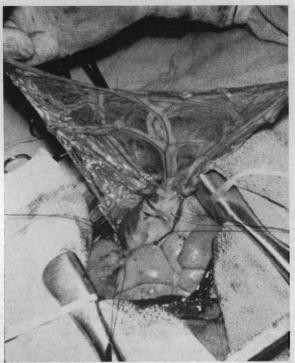


Fig. 3—Omentum in the pericardial sac. This evidence of a rarely seen diaphragmatic-pericardial hernia was an unexpected finding at the time of surgery. The omentum was ligated and removed without difficulty.

space on December 21. Upon opening the pericardial sac, a 10 cm. portion of omentum (Fig. 3) was seen to cover the lateral surface of the ventricles. The origin of the omentum was traced to a small opening between the apex of the pericardial sac and the peritoneal cavity, a true diaphragmatic-pericardial hernia. The omentum was ligated and amputated, and the stump was replaced into the peritoneal cavity.

The right ventricle was extremely enlarged and a thrill could be felt in the dilated pulmonary artery during systole. A small incision was made in the right ventricular outflow tract and a Brock valvulotome was introduced into the incision and passed several times through the pulmonary valve area. Only slight lessening of the thrill resulted and it was felt that the procedure was unsuccessful due to marked obstruction of the right ventricular outflow tract by the hypertrophied myocardium. (Adequate correction of this stenosis would require open heart surgery performed with heart-lung bypass.)

^{*}Dr. Patterson is instructor in cardiology.



Fig. 4—Pulmonary semilunar valves viewed from the main pulmonary artery. All the semilunar cusps were fused with the exception of a small 1 mm. orifice. The main pulmonary artery is dilated (poststenotic dilation).



Fig. 5—The right ventricle is thickened, with narrowing of its outflow tract. A 2 mm. applicator stick reduced to 1 mm. diameter has been passed through the orifice of the stenotic pulmonary semilunar valves.

The myocardial, pericardial, and thoracic incisions were closed and the dog recovered from the surgery without complication. However, on Jan. 15, 1961, the dog became cyanotic, staggered, and collapsed. It was revived by oxygen therapy and progressed with mild dyspnea for five more days. On January 20, the dog appeared all right at 9:00 p.m. but was found dead at midnight.

Pathologic Findings

Dr. K. Hubben.*—The postmortem findings substantiate the clinical diagnosis. On gross examination, the heart had the following abnormalities: the right ventricle was markedly thickened (Fig. 5) and on cut section the myocardium was pale and mottled. The pulmonic valve opening (Fig. 4) was narrowed by fusion of the cusps to

*Dr. Hubben is assistant professor of veterinary pathology.



Fig. 6—Patent foramen ovale viewed from the right atrium.

a 1-mm. valve orifice. The foramen ovale was patent (Fig. 6). Other findings grossly and histologically were minor and mostly related to congestive heart failure.

Comments

Dr. J. Buchanan.—I would like to point out that normally, in doing cardiac catheterizations, particularly in cases of suspected valvular stenosis, we always attempt to get a pressure recording on both sides of the involved valve to determine the presence of a gradient (difference in pressure across the valve). In this case, the pulmonic valve was so stenotic that we could not get a catheter through it.

Dr. R. S. Brodey.—How often do you find these two lesions present in the same animal?

Dr. Buchanan.—This is the second case I have seen in three months; however, in the earlier one, the atrial septal defect was larger than just a patent foramen ovale. I would like to refer your question to Dr. Patterson who has had more experience in this field than I.

Dr. Patterson.—In a series of 3,000 dogs screened for cardiovascular disease in our clinic, we found 6 with pulmonic stenosis—about 2 per 1,000 dogs examined. One of these also had a patent foramen ovale with a right to left shunt.

The coexistence of these two lesions is not surprising when one considers the effects of pulmonic stenosis on the circulation. Normally, at birth the pulmonary vascular resistance falls as the lungs expand. This allows more venous blood to flow through the lungs and causes a decrease in pressure in the right side of the heart. At this time, left atrial pressure usually rises above right atrial pressure. and a membranous flap on the left side of the septum forms a valve over the foramen ovale. This valve prevents blood from flowing from left to right, but should right atrial pressure again rise above left for any reason, flow through the foramen from right to left might be re-established. In pulmonic stenosis, the right ventricular pressure remains high because outflow through the small valve orifice is impeded. Right ventricular pressure can be considerably elevated for some time without any significant rise in right atrial pressure. If the ventricle becomes unable to empty itself adequately, however, as occurs in congestive heart failure, right atrial pressure will rise. At this point, perhaps after the animal has reached maturity, the foramen ovale may open, and right to left shunting may occur, at which time the dog becomes cyanotic.

Pathologic Diagnosis

- 1) Congenital pulmonic valvular stenosis.
- 2) Patent foramen ovale.
- 3) Diaphragmatic pericardial hernia.

Reference

¹Detweiler, D. K., Brodey, R. S., and Flickinger, G. L.: Diagnosis and Surgical Correction of Peritoneopericardial Diaphragmatic Hernia in a Dog. J.A.V.M.A., 137, (Aug. 1, 1960): 177-182.