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Abstract
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Disciplines
Cardiology | Medicine and Health Sciences | Veterinary Medicine

Comments
Appendix 6.10 of James W. Buchanan's dissertation Chronic Valve Disease and Left Atrial Splitting in the Dog

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J. W. Buchanan, D.V.M., and R. L. Pyle, V.M.D.

Summary

Perforation of the heart, which was confirmed by contrast radiography, occurred during venous cardiac catheterization of a dog. As tamponade developed within the next hour, changing physical signs (progressive muffling of heart sounds and murmur, disappearance of a precordial thrill, fall in blood pressure, and increase in heart rate with a weakening pulse) indicated the need for surgical relief in spite of insignificant fluoroscopic evidence. Treatment by thoracotomy, pericardiotomy, and blood replacement was successful. Evidence of a bidirectional ventricular septal defect and probable pulmonic stenosis was observed in angiocardiograms made an hour after the thoracotomy. The dog was discharged in good condition 12 days later. Examination 3 months later revealed no after effects of the cardiac catheterization and thoracotomy.

CARDIAC tamponade is a rare complication of cardiac catheterization in man and is occasionally diagnosed early enough to permit successful corrective measures.[4] The purpose of this report is to present the clinical, fluoroscopic, angiocardiographic, hemodynamic, and surgical aspects of cardiac perforation and tamponade in a dog with congenital heart disease. In this case, tamponade resulted from perforation of the right ventricle during cardiac catheterization.

History and Clinical Findings

On Sept. 21, 1965, a 17-lb. (8-kg.) female Shetland Sheepdog, 1 year old (Case 8085 J), was submitted for examination because of suspected congenital heart disease. The dog had been bred and raised by the owner and had been vaccinated against distemper, hepatitis, and rabies. For 2 months prior to examination, the owners had noticed a decrease in the dog's activity and exercise tolerance.

Two weeks prior to our examination, the dog had been taken to a local veterinarian because of anorexia of 3 days' duration. A precordial thrill and a heart murmur were detected; a blood examination for microfilaria was negative. A diagnosis of heart disease was made, and the dog was treated with digitalis, penicillin, and an oral sulfonamide.

The results of the physical examination on September 21 were within normal limits except for the cardiovascular findings. Intense thrills were palpated on the left side of the thorax in the pulmonic region (3rd intercostal space) and over the right 3rd intercostal space. A grade 4 (out of 5), medium-pitched, holosystolic murmur was heard best on the left side of the thorax. Although most intense in the pulmonic area, the murmur was audible over the entire left side of the thorax and over a large portion of the right thorax. On the right side, the murmur

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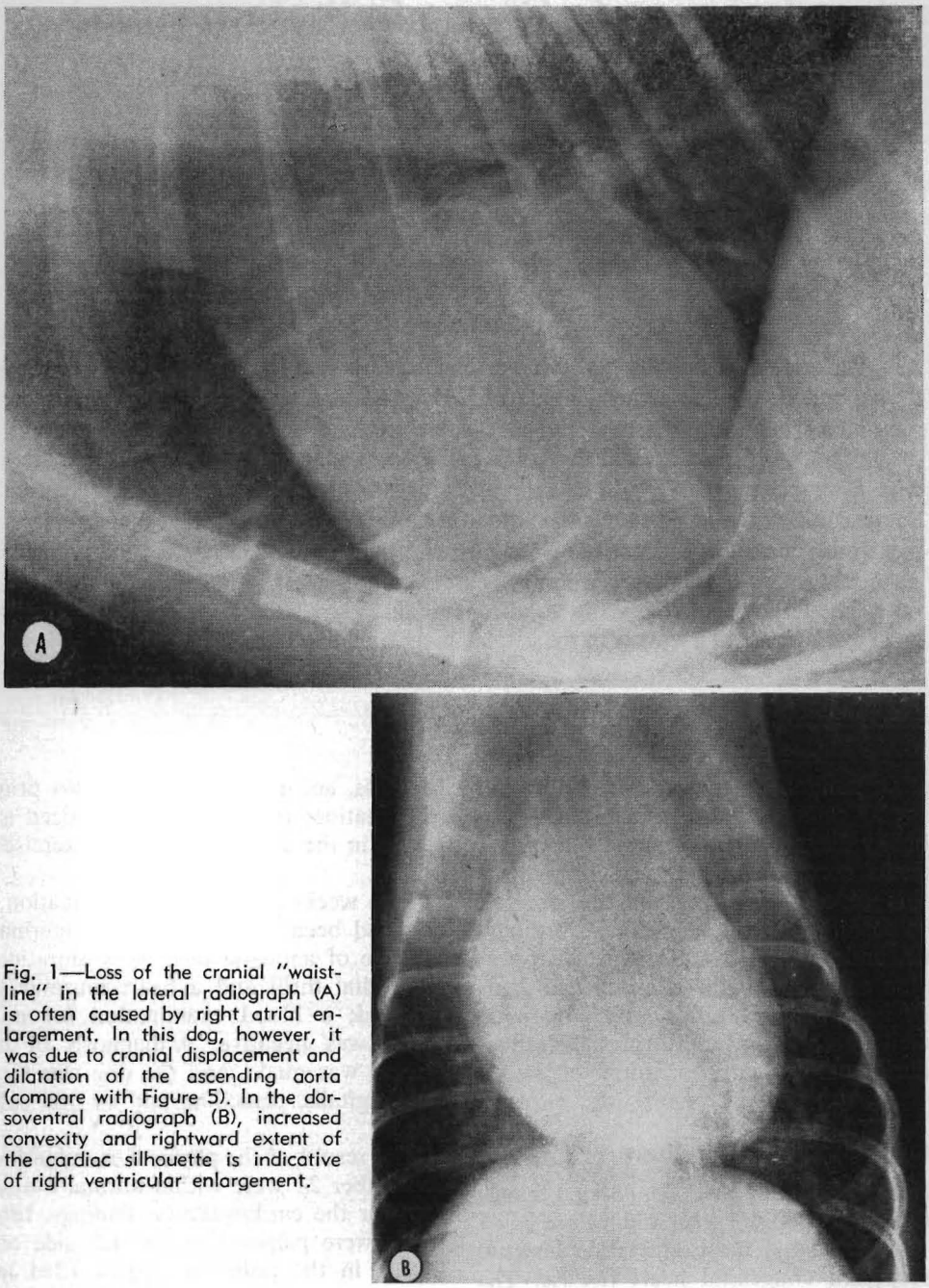
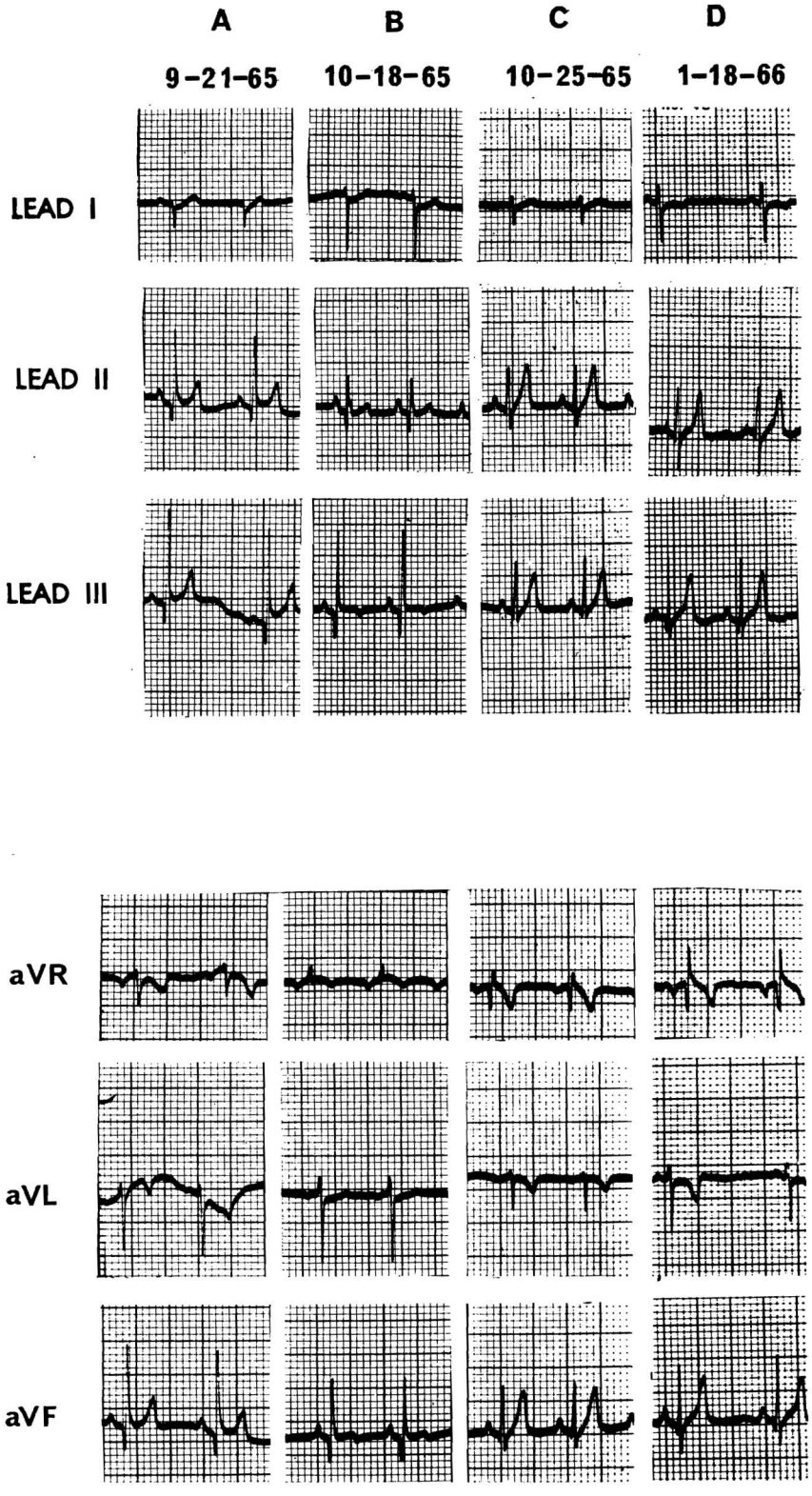


Fig. 1—Loss of the cranial "waistline" in the lateral radiograph (A) is often caused by right atrial enlargement. In this dog, however, it was due to cranial displacement and dilatation of the ascending aorta (compare with Figure 5). In the dorsoventral radiograph (B), increased convexity and rightward extent of the cardiac silhouette is indicative of right ventricular enlargement.

was of grade-4 intensity in the 3rd and 4th intercostal spaces near the sternum. It also could be heard well at the thoracic inlet. Cyanosis was not observed.

In thoracic radiographs, there was evidence of right ventricular enlargement (Fig. 1). In the lateral radiograph, loss of the cranial "waistline" was noticed. In the dorsoventral radiograph, the right half of the cardiac silhouette was more prominent and


A
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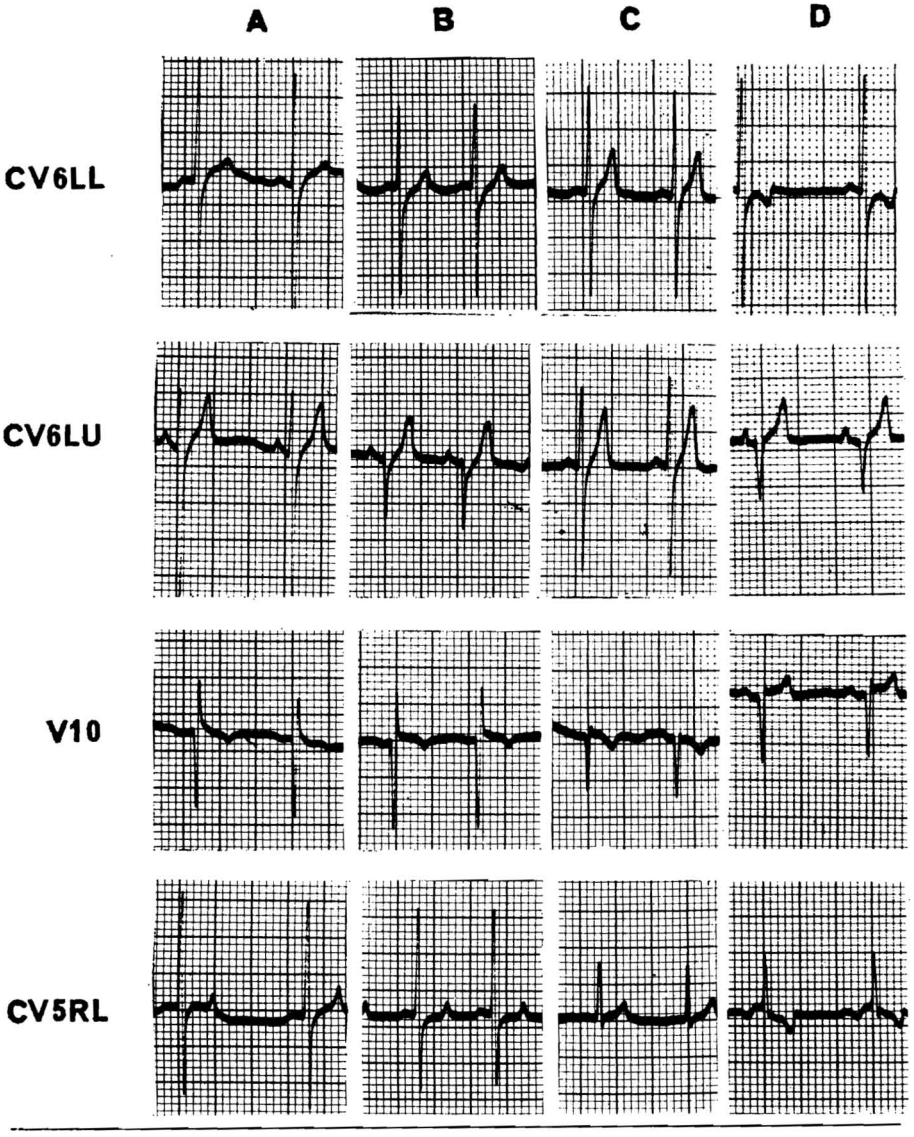

Legend for illustrations on left-hand page and above

Fig. 2—Four different 10-lead electrocardiograms recorded before (A, B) and after thoracotomy (C, D). Unipolar-exploring thoracic leads were recorded over the left 6th intercostal space to the left of the sternum (CV6LL); left 6th intercostal space at the level of the costochondral junction (CV6LU); dorsal to the 7th thoracic vertebra (V10); and over the right 5th intercostal space to the right of the sternum (CV5RL). All leads were recorded at a paper speed of 25 mm. per second and a sensitivity of 1 cm./mv. except for CV6LL in B to D and CV6LU in B where a sensitivity of 0.5 cm./mv. was used. In the initial electrocardiogram (A), the mean manifest frontal plane QRS axis of 107 degrees coupled with a clockwise vector loop and large S waves in leads CV6LL and CV6LU were compatible with a diagnosis of right ventricular hypertrophy. Four weeks later (B), the QRS axis was 145 degrees and larger S waves were present in CV6LL. The presence of S waves in leads II, III, and aVF 6 days after perforation of the right ventricle (C) was considered indicative of interference with right ventricular conduction, however, the QRS duration remained within normal limits. Negative T waves in CV5RL and positive T waves in V10 as noticed 3 months later (D) have been consistently associated with right ventricular hypertrophy in other dogs.

convex than normal. Prominence of the main pulmonary artery segment was not regarded as definitely abnormal.

In a 10-lead electrocardiogram (ECG), the frontal plane mean manifest QRS axis was 107 degrees (Fig. 2 A). In lead I and in exploring unipolar left thoracic leads over the apex (CV6LL) and free wall of the left ventricle (CV6LU), abnormally large S waves were observed, indicating cranial and rightward direction of the terminal QRS vector forces. These features were compatible with a diagnosis of right ventricular hypertrophy. In other respects, the ECG was within normal limits.

On the basis of the foregoing findings, the dog was considered to have congenital heart disease, and a diagnosis of ventricular septal defect or pulmonic stenosis or both was made. Since the dog had previously been given digitalis, it was kept on digitalis while arrangements for cardiac catheterization were being made.

Cardiac Catheterization

The dog was hospitalized on October 18 for definitive studies. Clinical findings were unchanged from the previous examination; however, increased right axis deviation (QRS axis, 145 degrees) was noticed in an ECG (Fig. 2 B). The following day, catheterization via the right jugular vein was begun after preanesthetic administration of 0.5 mg. of atropine sulfate and 30 mg. of meperidine hydrochloride* and after induction of anesthesia with intravenous pentobarbital sodium. Under fluoroscopic guidance, a curved, 7 French, teflon catheter with a tapered tip, open end, and side holes was positioned in the right ventricle. Simultaneous recordings of a lead II ECG, right 4th intercostal space phonocardiogram, and right ventricular pressure were made (Fig. 3 A). The right ventricular pressure was 100/0 mm. Hg. Repeated attempts to position the catheter in the pulmonary artery were unsuccessful. It was removed and replaced with a more flexible 6 French woven nylon catheter with a closed end and 4 side holes near the tip.

This catheter was positioned in the right ventricle using fluoroscopy and pressure monitoring and then advanced cranially in the right ventricular outflow tract toward the main pulmonary artery. When the tip of the catheter reached the level of the juncture of the cardiac silhouette and the cranial mediastinum, abrupt resistance was met; so no attempt was made to advance it further. Because the catheter tip was adjacent to the cranial border of the cardiac silhouette and the blood pressure monitored through the catheter had fallen below zero, it was thought that the catheter had penetrated the myocardium and was being stopped by the craniodorsal reflection of the pericardium. The catheter was quickly withdrawn until the tip was in the cranial vena cava.

It was advanced again into the right ventricle and toward the main pulmonary artery where it followed the previous course with no resistance until it stopped abruptly at the same level as before, and the pressure was again below zero.

Because of the similar course of the catheter, the previous assumption of myocardial puncture was considered a false alarm, and the catheter was thought to be in the main pulmonary artery on both occasions. This was based also on findings in some other dogs with confirmed pulmonic stenosis. In these dogs, poststenotic dilatation of the main pulmonary artery sometimes causes it to contribute significantly to the cranial border of the cardiac silhouette.[2] Although not previously observed by the authors, a catheter within a dilated main pulmonary artery could lie adjacent to the cranial border of the cardiac silhouette. The second factor which was considered a false alarm was the marked drop in pressure being recorded through the catheter. The cardinal proof of the existence of pulmonic stenosis is the demonstration of a pressure gradient across the pulmonary valve or the right ventricular outflow tract. The pressure at this point in this dog fell below zero; however, this was attributed to possible baseline drift in the recorder and occlusion of a stenotic pulmonary orifice by the presence of the catheter. Since the latter event was considered responsible for ventricular fibrillation and death in another dog, a recording of the pressure was immediately begun while the catheter was

*Demerol, Winthrop Laboratories, New York, N.Y.

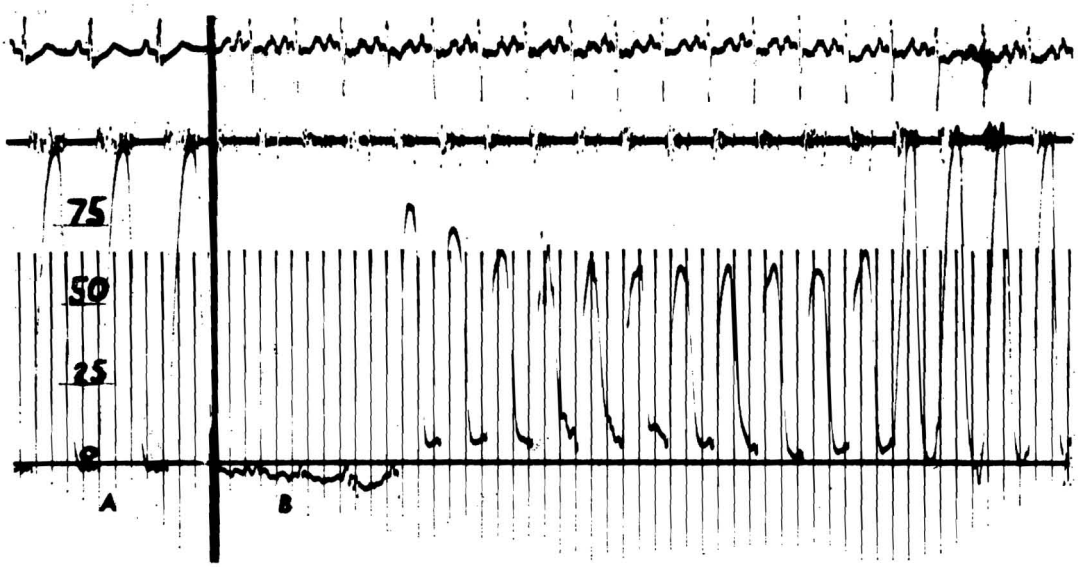

Fig. 3—Lead II electrocardiogram, right 4th intercostal space phonocardiogram, and pressure recordings. The right ventricular pressure was 100/0 mm. Hg (A). The pressure in the pericardial cavity (B) was −5 to −10 mm. Hg. It increased to 60 to 75 mm. Hg as the catheter was withdrawn toward the right ventricle before suddenly increasing to 100/0 mm. Hg inside the right ventricle.

being withdrawn to the right ventricle (Fig. 3 B). By observing the catheter through the fluoroscope and the monitored pressure on an oscilloscope, it was noticed that the pressure did not increase until the catheter tip was near the apex of the right ventricle. The recorded pressure gradients were nearly typical for combined valvular and subvalvular pulmonic stenosis; however, the levels at which they were recorded were distinctly more ventral than in any other animal with pulmonic stenosis previously observed.

For this reason, the catheter was advanced again toward the main pulmonary artery, and it followed the same course as before without resistance until it stopped abruptly at the juncture of the cranial mediastinum and the border of the cardiac silhouette. This time, 7 cc. of a contrast material* was injected through the catheter using hand pressure on a glass syringe. By fluoroscopic observation during the injection, the contrast material was seen leaving the catheter and diffusely spreading out over the entire cardiac silhouette, thus confirming that the catheter was in the pericardial sac. The catheter was then withdrawn to the right ventricle, and a radiograph was made (Fig. 4).

Being aware that the right ventricular myocardium had been perforated probably 3 times, a period of watchful waiting ensued. During the next 20 minutes the right ventricular pressure gradually fell to 50 mm. Hg. The heart rate increased moderately, and the femoral pulse became very weak. The precordial thrill disappeared, and the intensity of the murmur decreased to grade 2. Fluoroscopic examination during this time revealed no significant increase in the size of the cardiac silhouette.

After instituting positive pressure ventilation, a thoracotomy was performed through the right 5th intercostal space, and the heart was exposed within 10 minutes. At this time, the right ventricular pressure was below 25 mm. Hg. Approximately 50 cc. of nonclotted blood was aspirated from the pericardial sac, and an intravenous transfusion of 200 cc. of whole blood was started. Right ventricular pressure immediately began to increase, and the heart action became more vigorous. The pericardial sac was then opened and the epi-

*Cardiografin 85%, E. R. Squibb and Sons, New York, N.Y.

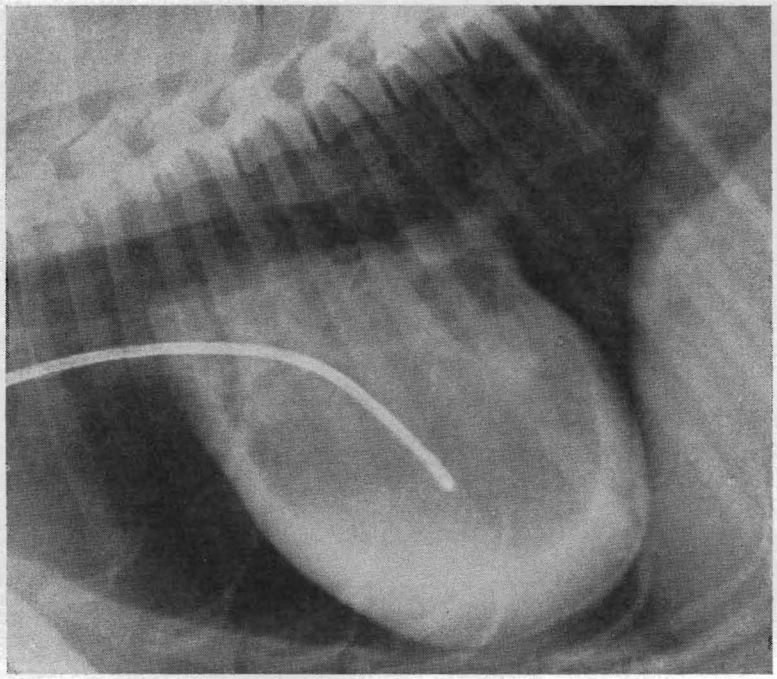

Fig. 4—Radiograph made after 7 cc. of a contrast material had been injected into the pericardial sac. The amount of blood in the pericardial sac at this time was not known, and clinical evidence of cardiac tamponade was not yet detectable.

cardium was examined to see if active bleeding still was occurring.

By elevating the apex of the heart out of the pericardial sac, 3 sites of epicardial perforation were made visible in the outflow tract of the right ventricle near the left anterior descending coronary artery halfway between the base of the heart and the apex. Each site was identified as a 3- to 4-mm. (diameter) subepicardial hematoma, with a break in the epicardium over the center of each. All were located about 1 cm. from each other, roughly forming a triangle. Active hemorrhage was not noticed even though the heart action was good, and the right ventricular pressure was again elevated. For this reason, no sutures were placed in the perforation sites. On direct palpation of the heart, a systolic thrill was felt over the right ventricular outflow tract and the main pulmonary artery. A thrill was not felt on the right atrium and ascending aorta.

Passing a dilator through the pulmonic valve was considered but rejected, since the accessibility of the pulmonary valve from a right-sided thoracotomy is poor. The ability of the right ventricle to tolerate a ventriculotomy at this time was questionable, and a diagnosis of pulmonic stenosis was not certain.

The pericardium was loosely sutured, and the thorax was closed in routine fashion after insertion of a pleural drainage tube. The catheter was replaced in the right ventricle without further attempts to measure pulmonary artery pressure.

After injecting 10 cc. of a contrast medium into the right ventricle near the apex, angiocardiograms were made at intervals of approximately 2 per second. Simultaneous opacification of both ventricles indicated right-to-left shunting of blood through a ventricular septal defect (Fig. 5 A). Probable dilatation of the main pulmonary artery was also present, which supported a diagnosis of coexisting pulmonic stenosis. In subsequent angiocardiograms, the right ventricle was no longer radiopaque as the contrast material circulated into the pulmonary vessels (Fig. 5 B). When the contrast material reached the left ventricle, the right ventricle became opacified again, indicating left-to-right shunting of blood through the ventricular septal defect (Fig. 5 C).

On the bases of the loud systolic murmur in the pulmonic area, the palpable thrill on

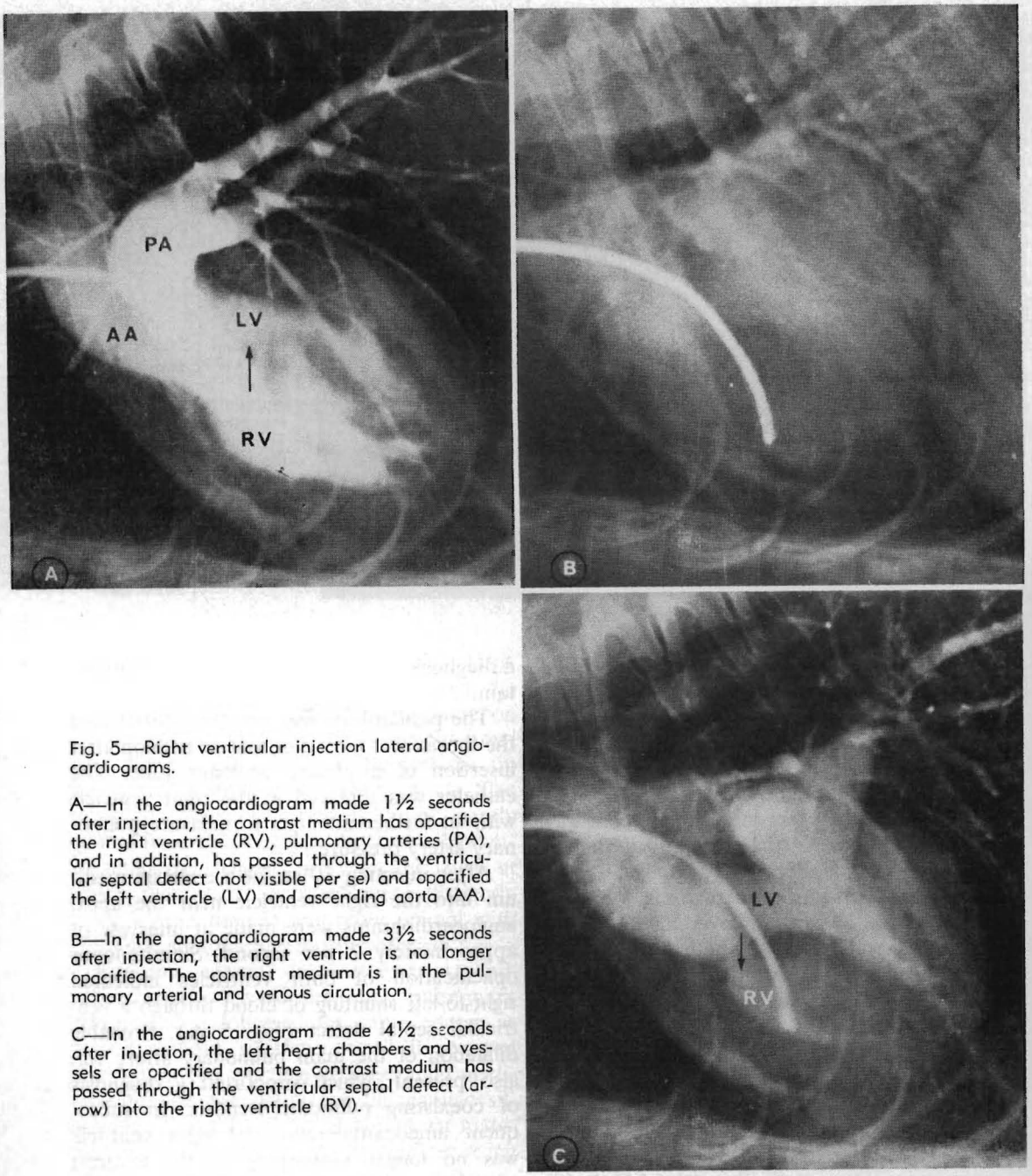

Fig. 5—Right ventricular injection lateral angiocardiograms.

A—In the angiocardiogram made 1½ seconds after injection, the contrast medium has opacified the right ventricle (RV), pulmonary arteries (PA), and in addition, has passed through the ventricular septal defect (not visible per se) and opacified the left ventricle (LV) and ascending aorta (AA).

B—In the angiocardiogram made 3½ seconds after injection, the right ventricle is no longer opacified. The contrast medium is in the pulmonary arterial and venous circulation.

C—In the angiocardiogram made 4½ seconds after injection, the left heart chambers and vessels are opacified and the contrast medium has passed through the ventricular septal defect (arrow) into the right ventricle (RV).

the main pulmonary artery and borderline evidence of main pulmonary artery enlargement in angiocardiograms, the final diagnosis in this case was pulmonic stenosis and bidirectional ventricular septal defect. Although not accepted by all authorities, this has also been referred to as a mild form of "tetralogy of Fallot."

For 3 hours following completion of the thoracotomy, the body temperature gradually increased to 106.4 F. (41.4 C.), and the dog was still in an unresponsive anesthetic

state. Ice packs were applied and the temperature gradually declined. Seven hours after the thoracotomy, body temperature was 103.4 F. (39.7 C.), and the dog was able to support itself in a sitting position. Subsequent daily treatment consisted of penicillin, dihydrostreptomycin, and digoxin. The post-thoracotomy course was uncomplicated

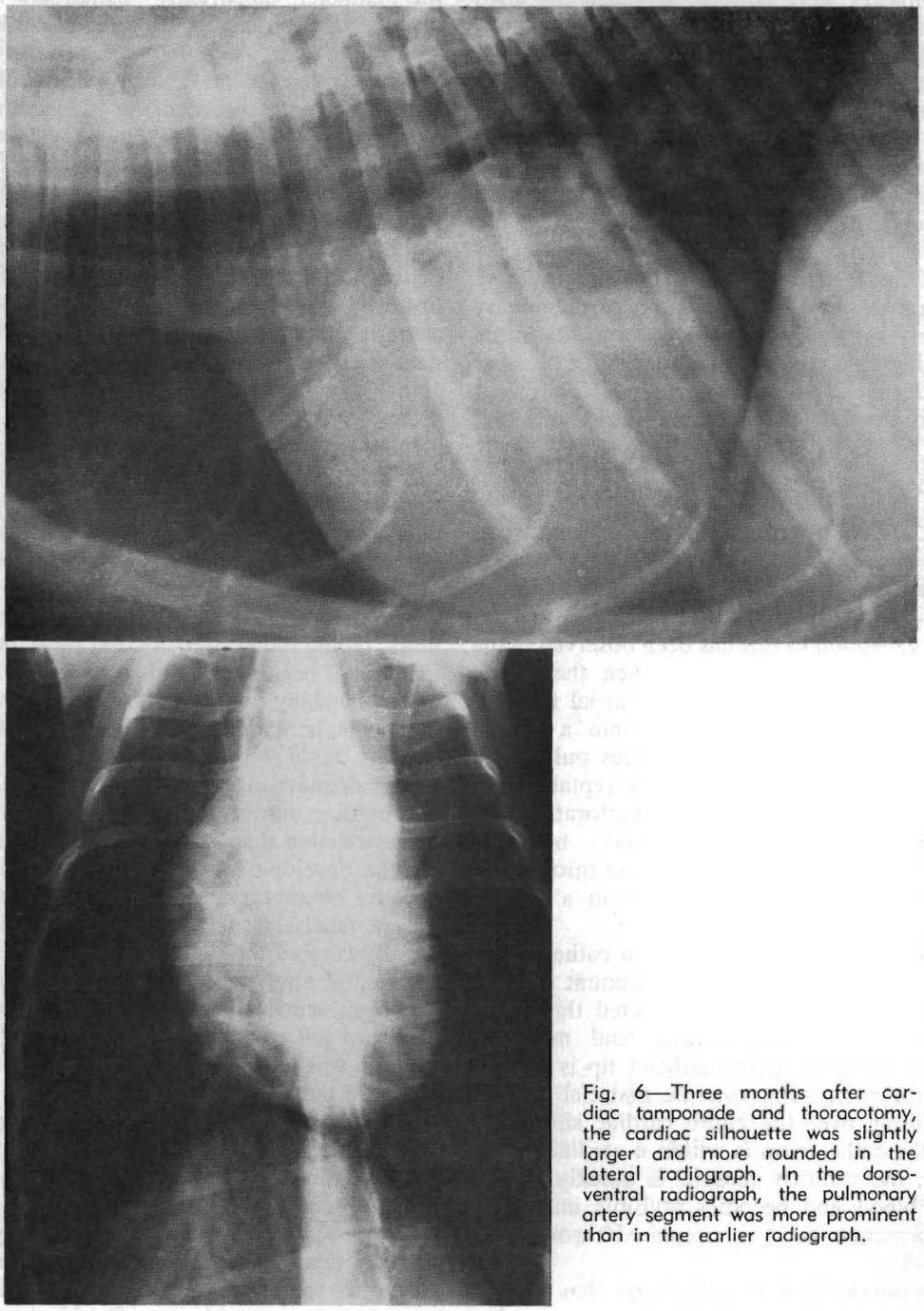

Fig. 6—Three months after cardiac tamponade and thoracotomy, the cardiac silhouette was slightly larger and more rounded in the lateral radiograph. In the dorsoventral radiograph, the pulmonary artery segment was more prominent than in the earlier radiograph.

except for a mild temperature elevation beginning on the 6th day and lasting 3 days. The dog was discharged in good condition 12 days after admission.

In several ECG's made after the thoracotomy, progressive changes in the QRS-T complexes were noticed, although the QRS duration remained basically unchanged (Fig. 2 C). In the last tracing (Fig. 2 D), terminal QRS forces were directed more cranially (S wave in leads II, III, and AVF); the T waves were negative in CV5RL and positive in V10. These changes were compatible with increasing degrees of right ventricular hypertrophy, although they could have been caused at least in part by myocardial damage. In thoracic radiographs made 3 months later (Fig. 6), the main pulmonary artery segment was larger than in the initial radiographs. This was considered further evidence of pulmonic stenosis coexistent with the known ventricular septal defect.

Discussion

Cardiac perforation into the pericardial sac should be suspected during fluoroscopy when an intracardiac catheter is observed close to the border of the cardiac silhouette. An exception to this has been observed in the cranial cardiac silhouette when the catheter tip is located in the right atrial appendage. Entrance of a catheter into a pulmonary vein because of anomalous pulmonary venous drainage or an atrial septal defect may also resemble cardiac perforation fluoroscopically. In this instance, however, withdrawal of oxygenated blood through the catheter identifies its location in a pulmonary vein.

To confirm penetration of a catheter into the pericardial sac, a small amount of radiopaque material can be injected through it using an ordinary syringe and moderate hand pressure. If the catheter tip is located in the pericardial sac, the material spreads diffusely over the entire cardiac silhouette. If the catheter tip is within a cardiac chamber, the contrast material is quickly diluted by blood and becomes invisible under the fluoroscope as it is circulated through the heart.

Realization that a catheter has passed through the myocardium into the pericardial sac is based primarily upon fluoroscopic observation of the position of the catheter. In this dog and in a previous dog where treatment was delayed and unsuccessful, no noticeable resistance was encountered when the catheter apparently penetrated the myocardium. Similar findings have also been reported in man.[3]

Observation of an abrupt fall in blood pressure being recorded by the catheter gives an indication that the catheter may have entered the pericardial sac. This is not pathognomonic, however, since a similar drop to zero may occur when an open-ended catheter is impinged upon the endocardium or when a kink develops. This has also been observed in dogs with severe pulmonic stenosis in which the pulmonic orifice is nearly occluded by a catheter being passed from the right ventricle into the pulmonary artery.

Although perforation into the pericardial sac may be proved or suspected, cardiac tamponade does not necessarily develop in all patients. Before a pericardiocentesis or thoracotomy is performed, the patient should be observed for signs of tamponade (progressive muffling of heart sounds and murmurs, falling blood pressure and weak pulse, increasing heart rate and respiratory rate, and blanching of mucous membranes). Since the pericardial sac does not distend acutely, rapid accumulation of blood prevents adequate atrial and ventricular filling, and cardiac output diminishes. Fluoroscopy is less reliable than physical signs in assessing the development of cardiac tamponade, because circulatory arrest may occur when only a relatively small amount of blood has rapidly entered the pericardial sac. In an experimental study of dogs, systemic arterial and right ventricular systolic blood pressure began to fall when 30 ml. of isotonic NaCl solution was injected into the pericardial sac. Maximal tamponade (precipitous fall in systemic blood pressure) occurred in dogs of various sizes when 80 to 180 ml. of NaCl solution was injected.[5] In animals with diseased hearts, it is not unlikely that smaller amounts of blood could cause circulatory arrest. In this dog, no significant change occurred in the size of the cardiac silhouette

during the development of clinical signs of tamponade. This is in contrast to the distensibility of the pericardial sac in instances of chronic pericardial effusion. As much as 1,200 ml. of sanguineous fluid has been withdrawn from the pericardial sac of dogs with heartbase tumors.[1]

Although it was not done in this dog, pericardiocentesis perhaps should be attempted in animals with suspected tamponade before resorting to thoracotomy. Against this, the following arguments can be made:

The volume of blood in the pericardial sac in acute tamponade is relatively small; thus, it would be difficult to tap, and the chances of entering the heart would be great. An actively hemorrhaging site might be lacerated further or a new site of hemorrhage might be made. If a patient is rapidly deteriorating, too much time might be lost. If a thoracotomy is performed, the site of hemorrhage can be located and sutured.

The catheter which initially perforated the myocardium of this dog could not be determined because 2 catheters were used. It is likely that the 3 holes in the epicardium were caused by the 2nd catheter, since it appeared to follow the same course each of the 3 times it was advanced toward the pulmonary artery. It is possible that an endocardial laceration could have been made by the stiffer catheter used initially; and through this, the more flexible blunt-tipped catheter may have entered the myocardium. The close proximity of the 3 epicardial perforation sites and the flexibility of the 2nd catheter make other explanations less satisfactory.

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