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Tetralogy of Fallot in a Cat

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SUMMARY

An antemortem diagnosis of tetralogy of Fallot was made in a 7-month-old cat by means of physical and hematologic examinations, radiography, angiography, electrocardiography, and phonocardiography. Two palliative surgical procedures were performed. The Blalock-Taussig operation (anastomosis of subclavian artery to pulmonary artery) was successful while the anastomosis was patent. The Pott's operation (anastomosis of pulmonary artery to aorta) failed and the cat died from postoperative hemorrhage due to an inadvertent tear in the pulmonary artery.

TETRALOGY OF Fallot is a congenital defect which, in man, includes the following multiple anomalies of the heart and great vessels: (1) a ventricular septal defect; (2) pulmonary arterial stenosis; (3) dextroposition of the aorta; and (4) right ventricular hypertrophy. It is the most common cause of cyanosis due to congenital heart disease and leads to retarded growth, intolerance to exercise, and frequent episodes of syncope in affected individuals.[7] Congenital heart defects with the same anatomic malformations as tetralogy of Fallot, and similar clinical and pathologic sequelae, have been reported in dogs[3,6,9] and cattle[5] and have been given the same eponym. The reported prevalence of cardiac anomalies in cats is low.[4] Four cases of tetralogy of Fallot have been reported in cats—1 case was referred to in a necropsy survey of feline cardiovascular lesions[1,2] and 3 cases were diagnosed clinically.[2] This report is concerned with the clinical diagnosis and surgical treatment of tetralogy of Fallot in a cat.

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Clinical History

An underweight 7-month-old female domestic long-haired cat was referred to the Division of Animal Medicine, Johns Hopkins University School of Medicine, with respiratory difficulty and a suspected cardiac abnormality.

The cat's dam had been adopted as a pregnant stray animal ill with "pneumonia." The dam was treated successfully and subsequently gave birth to 3 kittens. One of the kittens died the first week of life without clinical examination or necropsy; thus a diagnosis was not available. The remaining 2 kittens were reported to be "sickly" and had "colds." The owner noticed that the patient was small, appeared inactive, had bluish mucous membranes, and developed seizures after brief periods of sustained exercise. The seizures progressed to the extent that even the exertion of eating would precipi-

tate a generalized episode. During a seizure, the cat developed muscle rigidity with opisthotonos and curling of the toes; the cat would cry out, and bilateral dilatation of the pupils would occur. Episodes lasted 20 to 40 seconds, after which the cat would relax but remain in a confused state, with dilated pupils, for 3 to 5 more minutes.

Physical Findings

The cat weighed 2.15 kg., was extremely listless, and had marked dyspnea. There was marked cyanosis of oral and ocular mucous membranes, nose, and foot pads. A 2-cm. ventral hernia was near the umbilicus. On auscultation, harsh lung sounds were heard. A heart murmur was not detected and the cardiac rhythm was regular. These findings were unchanged on subsequent examinations. During the initial examination, conducted with minimal handling, the cat had 3 seizures related to anoxia.

Hematologic Findings

The total white blood cell and differential counts were normal. The packed cell volume (74%); red blood cell count (16,700,000/cmm.); and Hb. (greater

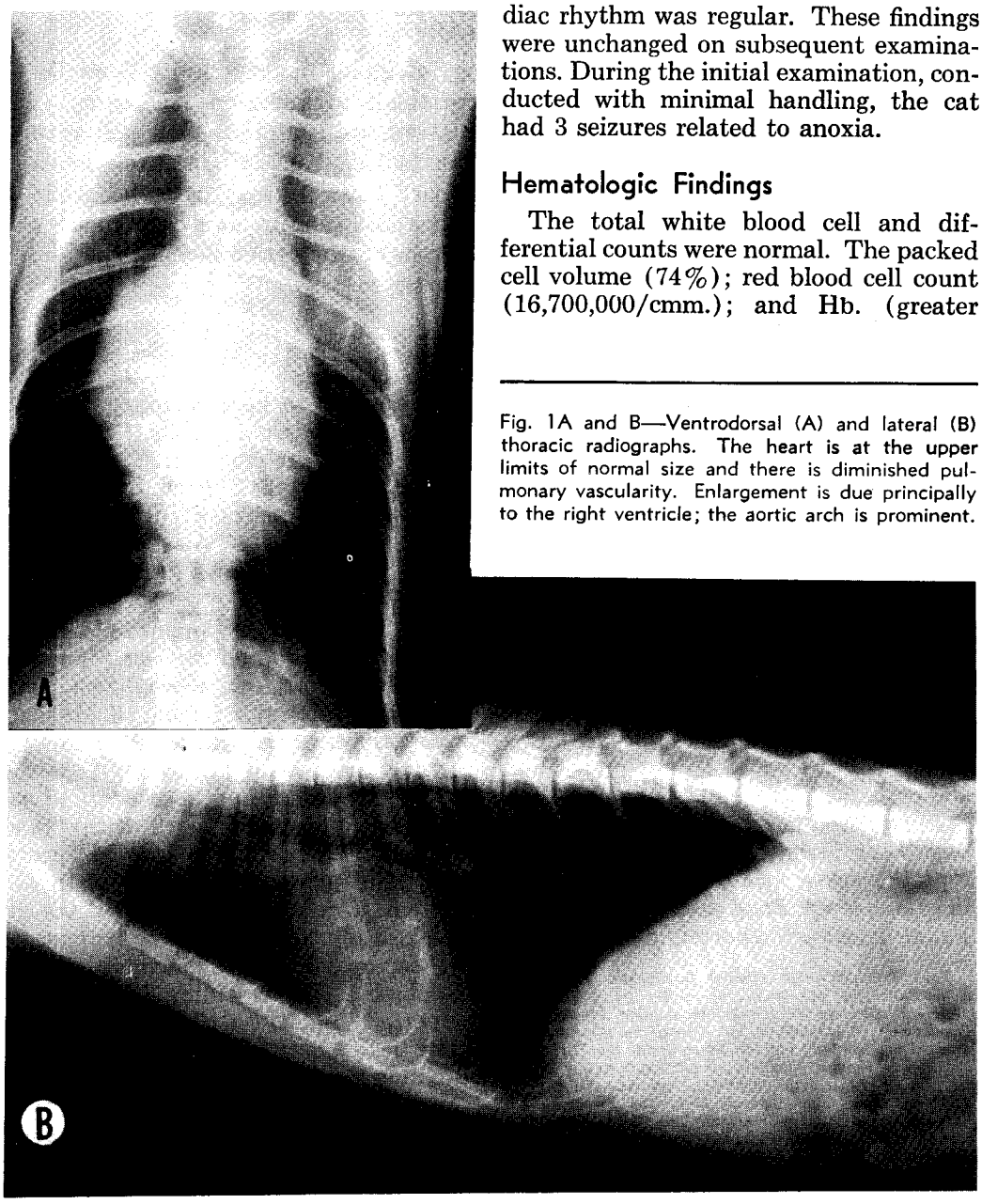

Fig. 1A and B—Ventrodorsal (A) and lateral (B) thoracic radiographs. The heart is at the upper limits of normal size and there is diminished pulmonary vascularity. Enlargement is due principally to the right ventricle; the aortic arch is prominent.

than 20.0 Gm./100 ml.) indicated marked polycythemia. Blood urea nitrogen content was 35 mg./100 ml. Examination of bone marrow aspirated from the iliac crest revealed erythroid hyperplasia and indicated that the polycythemia was of secondary type, inasmuch as there was no maturation arrest in the erythrocytic series and no atypical cells were noticed among the mature erythrocytes or their precursors. The polycythemia was in response to the anoxia occurring due to the heart defect.

Radiographic Findings

The thoracic radiograph revealed a decrease in pulmonary vascularity (Fig. 1). A cardiac angiogram was obtained to document the specific congenital abnormality. The cat was lightly sedated (5 mg. ketamine and 5 mg. promazine), and the jugular vein was surgically exposed and catheterized. Although some difficulty was encountered with the placement of the catheter (No. 4 angiographic), a diagnostic biplane angiogram was obtained by using 3 ml. of contrast material that was introduced by a mechanical injector at 160 p.s.i. The filming was 6 exposures/second for 2 seconds and 2 exposures/second for 5 seconds. There were right ventricular hypertrophy, infundibular pulmonary arterial stenosis,

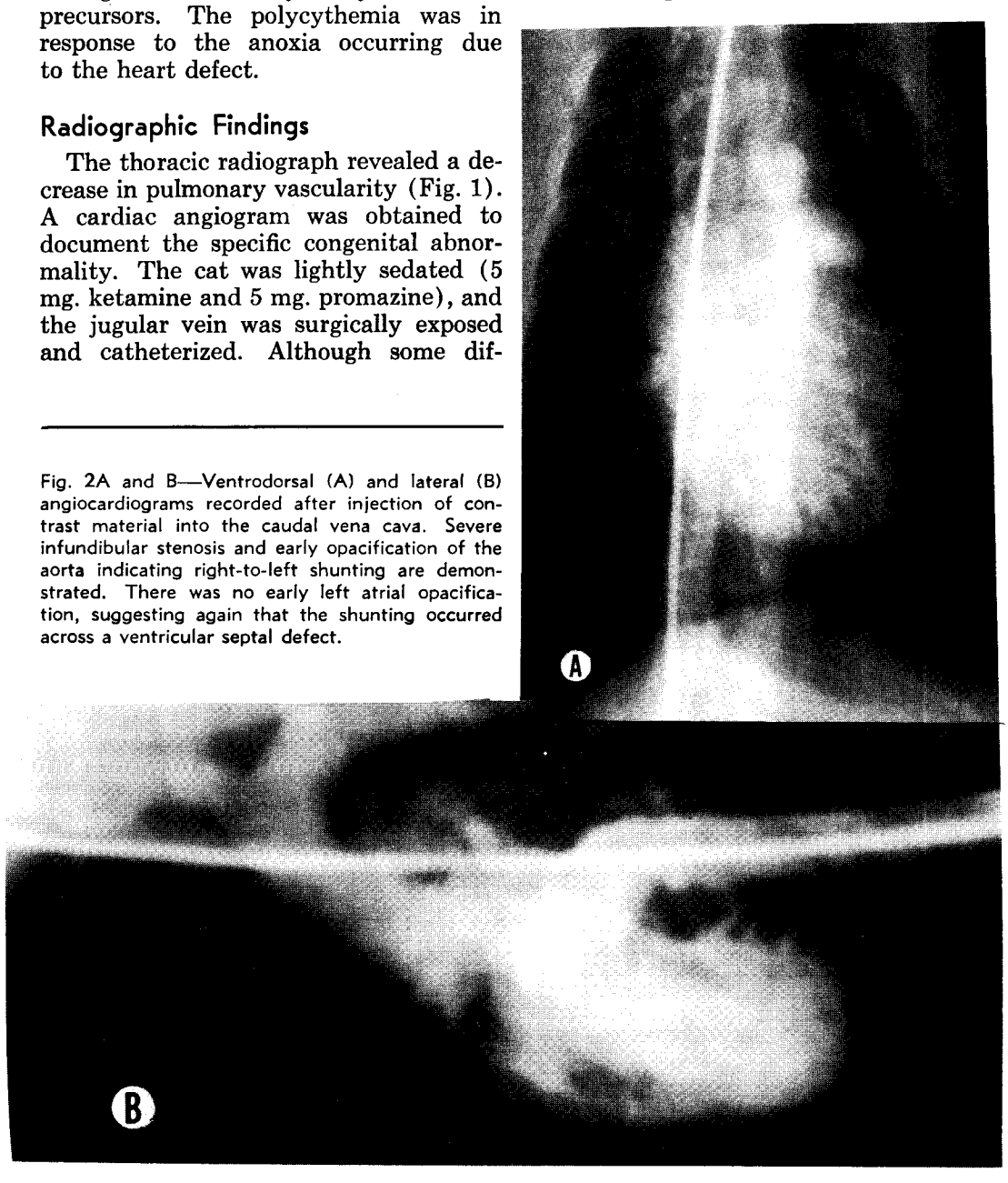

Fig. 2A and B—Ventrodorsal (A) and lateral (B) angiocardiograms recorded after injection of contrast material into the caudal vena cava. Severe infundibular stenosis and early opacification of the aorta indicating right-to-left shunting are demonstrated. There was no early left atrial opacification, suggesting again that the shunting occurred across a ventricular septal defect.

and a high intraventricular septal defect associated with right-to-left shunting of contrast material and an overriding aorta (Fig. 2)—all characteristics of tetralogy of Fallot. Selective right ventriculography was not done because of the cat's poor physical condition.

Phonocardiographic and Electrocardiographic Findings

The phonocardiogram revealed a normal S_1 with two components and a duration of 0.04 second. The presence of a single S_2 and the absence of a systolic murmur were of particular importance (Fig. 3). Diminution in the pulmonary component of the second heart sound is a well-documented finding in patients with tetralogy of Fallot. In severe right ventricular outflow obstruction, the second heart sound is obliterated because blood does not move the pulmonary valve.

The electrocardiogram (Fig. 4) demonstrated a severe right axis deviation of +210 degrees.[7] In the absence of right bundle branch block this was probably secondary to severe right ventricular hypertrophy. The tall peaked P waves seen in L-2 AVR and AVF were typical of right atrial enlargement.[11] Although neither the electrocardiographic nor phonocardiographic findings were pathognomonic, they were both consistent with the clinical diagnosis of tetraology of Fallot.

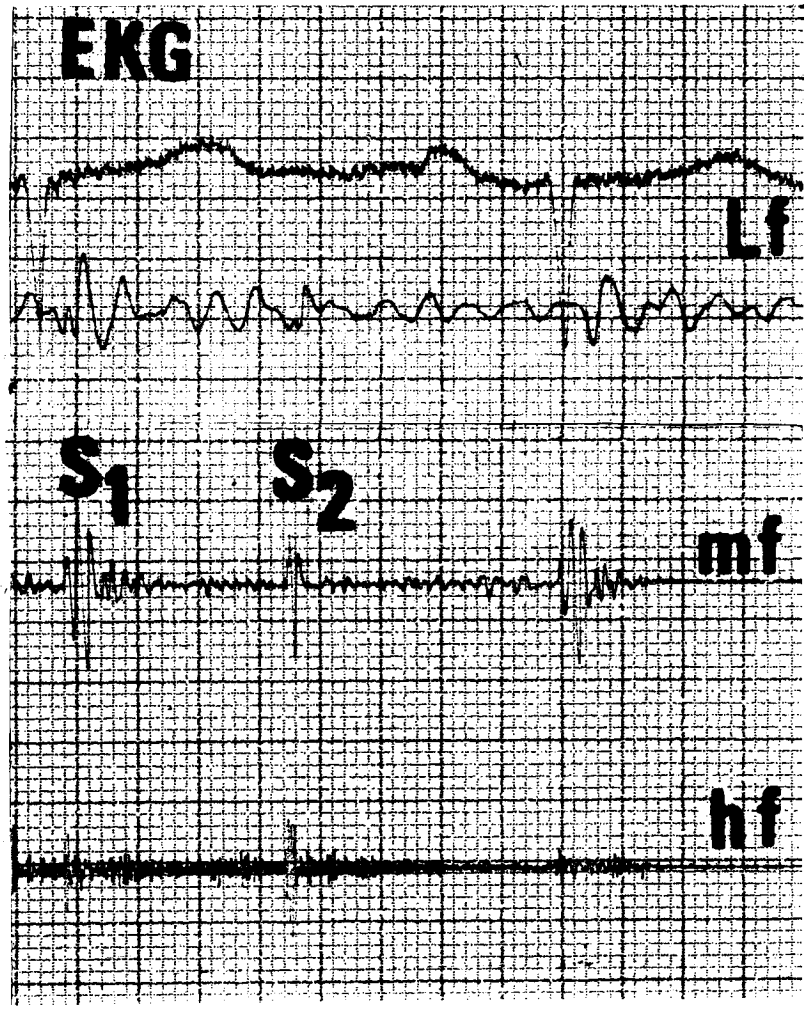

Fig. 3—Phonocardiographic tracings on high (hf), medium (mf), and low (lf) frequency bands demonstrate a normal first heart sound (s_1) followed by a single second heart sound (s_2). A murmur was not recorded. Respiratory sounds are recorded on hf and background sounds on lf bands. Time lines equal 0.01 second.

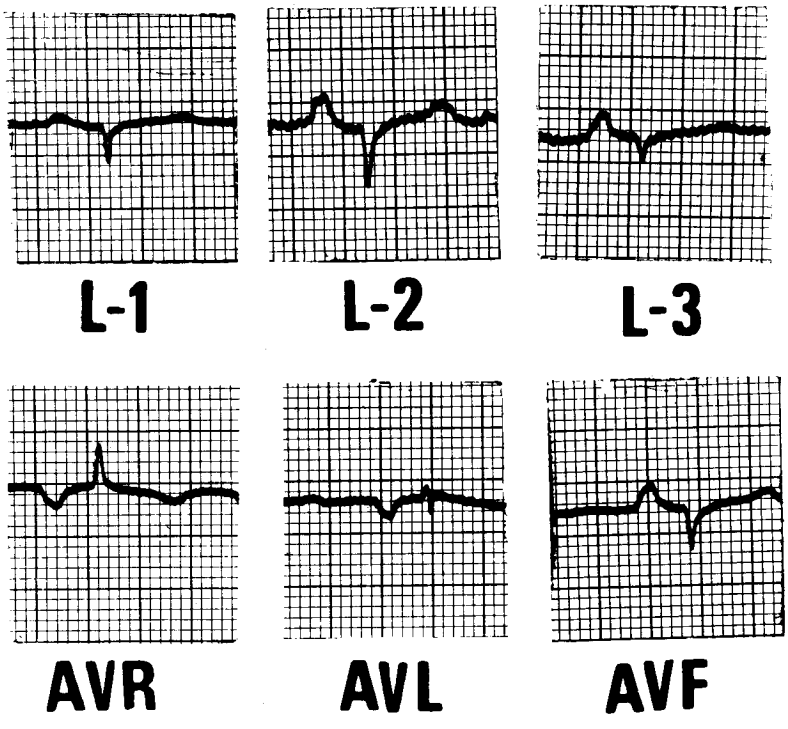

Fig. 4—Electrocardiographic limb-lead tracings, demonstrating severe right axis deviation. The prominent P waves represent right atrial enlargement. Time lines equal 0.02 second.

Surgery

The cat was fasted for 18 hours and was given atropine sulfate (0.1 mg.) and ketamine (5.0 mg.). Halothane (1½%) and O_2 was administered via a face mask until anesthesia was induced sufficiently to allow passage of an endotracheal tube, and then used in a semiclosed system to maintain surgical anesthesia. After the thorax was opened surgically, respirations were controlled manually.

After thoracotomy through the 4th intercostal space, a Blalock-Taussig operation was performed.[1] An anastomosis, using 6-0 cardiovascular silk sutures, was created between the divided proximal end of the subclavian artery, and an opening was made in the side of the pulmonary artery distal to the stenosis. After completion of the anastomosis, there was a marked increase in the diameter of the left pulmonary artery and a thrill was palpated. Routine closure was performed without difficulty. The cat was placed in an oxygen incubator for 72 hours and given antibiotics for 5 days. The respiratory sounds were harsh on auscultation the 1st day after surgery but became less so over the next 10 days. A loud, continuous left precordial murmur, characteristic of a patent anastomosis, was heard on ausculation.

The cat's physical activity and appetite increased steadily and were near normal on the 7th postoperative day. She was discharged on the 10th day but was readmitted 4 days later when she refused to eat. The cat then had signs of estrus and minimal respiratory distress. The anastomotic murmur was not as loud as it had been in the hospital immediately after surgery and could not be heard on the 16th postoperative day. The cat appeared clinically normal and a second palliative repair was not attempted at this time. Episodes of anoxia began to recur, however, and the cat was readmitted with severe dyspnea on the 37th postoperative day. Because of cyanosis and severe state of collapse, the cat was placed in an oxygen cage. Marked clinical improvement was noticed within 1 hour.

A second surgical procedure was performed 7 days later. Anesthesia and sur-

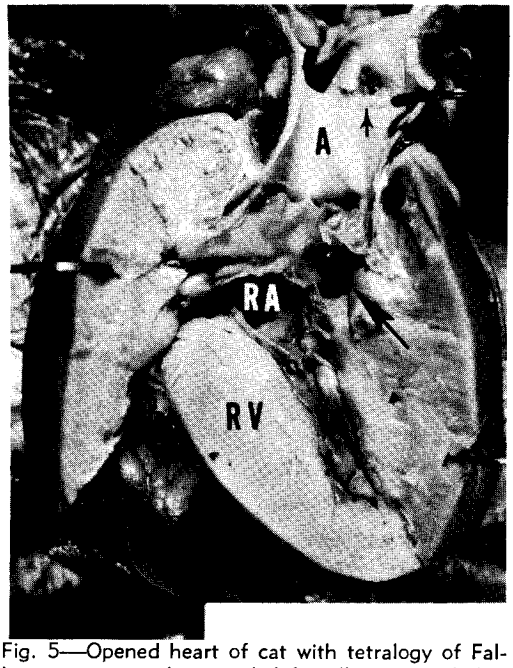

Fig. 5—Opened heart of cat with tetralogy of Fallot: interventricular septal defect (large arrow), hypertrophied right ventricular wall (RV), and the aortic side of the Pott's anastomosis (small arrow). Right atrium (RA), aorta (A).

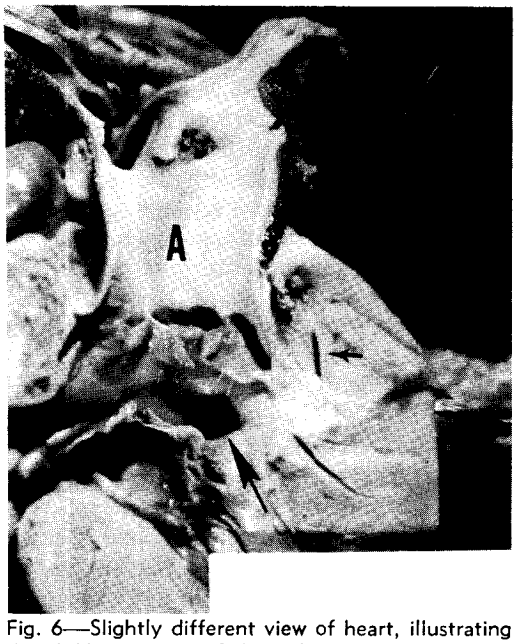

Fig. 6—Slightly different view of heart, illustrating the slitlike opening of the pulmonary outflow tract (small arrow). Interventricular septal defect (large arrow), aorta (A).

gical exposure were the same as in the first operation. There were no adhesions or other complications from the first procedure, but the left subclavian artery was obstructed at the site where it entered the pulmonary artery. The Pott's procedure[10]—a side-to-side anastomosis of the descending aorta to the left pulmonary artery—was performed. During dissection and isolation of the pulmonary artery, a small tear that was inadvertently created was controlled by digital pressure and absorbable hemostatic material. The Pott's procedure was facilitated by partial occlusion of both the aorta and pulmonary artery while a 3-mm. opening was made in each vessel. These openings were then sutured together with 6-0 cardiovascular silk. The pericardial sac was left open to prevent tamponade if hemorrhage should occur and the surgical incision was routinely closed. The cat was placed in an O_2 incubator and given antibiotics. She developed marked dyspnea after recovery from anesthesia and died 8 hours after surgery. A cosmetic necropsy was performed.

Pathologic Findings

At necropsy approximately 20 ml. of blood, which appeared to have originated from anterior mediastinal soft tissues, was in the pericardial sac and left hemithorax. The left lung was atelectatic. The heart weighed 32.5 Gm. and appeared to be enlarged and rounded. The right atrium was markedly dilated and the right ventricular myocardium was markedly hypertrophied (11 mm. thick) (Fig. 5). The atrioventricular valves were normal. A 6-mm. diameter defect was in the membranous portion of the intraventricular septum. The pulmonary infundibulum was markedly stenotic. The opening from the right ventricle was a 3-mm. long slit (Fig. 6) that passed as a narrow channel to a small but structurally normal pulmonary valve and main pulmonary artery. The left atrium and pulmonary veins were normal. The left ventricular myocardium was 7 mm. thick. There

was overriding of the aortic valve, which was situated over the ventricular septal defect and a portion of the right ventricle (Fig. 5). The ascending aorta and aortic arch were of a much larger caliber than the descending aorta or the pulmonary artery. The anastomosis of the left subclavian artery and pulmonary artery was occluded due to constriction at the suture line and subsequent reendothelialization of each vessel. The side-to-side anastomosis between the pulmonary artery and aorta was probe-patent.

Microscopically, there was moderate centrilobular congestion of the liver. There was evidence of hematopoietic activity in the spleen and there was marked erythroid hyperplasia of the bone marrow. Incidental findings were small foci of chronic pleuritis and focal chronic conjunctivitis. Death was attributed to hemopericardium and hemothorax.

Discussion

Clinical signs and electrocardiographic and radiographic findings in this cat were similar to those described in man and other animals with tetralogy of Fallot. One exception was the lack of a typical cardiac murmur, which in other species usually arises from blood flow across either the narrowed pulmonary infundibulum or, less commonly, the stenotic pulmonary valve. A murmur is not generated across the ventricular septal defect because both ventricles are at systemic or equal pressure. The absence of a cardiac murmur is entirely consistent with the diagnosis; the lack of a murmur in this cat could be associated with small size of pulmonary outflow tract diminishing the amount of blood flow necessary to cause turbulence. Limited blood flow was noted previously in the phonocardiogram by a single S_2.

Clinical manifestations of tetralogy of Fallot are secondary to a reduction of pulmonary blood flow, resulting in severe anoxia. Surgical correction is performed, therefore, to augment the blood supply to the lungs by surgically creating a patent ductus arteriosus. The Blalock-Taussig operation increases the pulmonary blood flow by shunting blood from the left side of the heart (via subclavian artery) and diverting it to the pulmonary trunk distal to the pulmonary stenosis. The purpose of the Pott's anastomosis (descending aorta to left pulmonary artery) is in theory identical to that of the Blalock-Taussig shunt.

In this instance, the Blalock-Taussig operation failed due to constriction of the suture line at the anastomotic site. This frequently occurs when the subclavian artery is small. Results were satisfactory while the shunt was functioning. The cat had marked increase in activity, tolerated exercise, and had no further episodes of anoxia.

The polycythemia noticed on initial examination decreased after the Blalock-Taussig shunt was created. At the 15th postoperative day, PCV was 63% and Hb. was 18 Gm./100 ml.; however, polycythemia became progressively worse after the shunt was no longer patent. The PCV increased to 77% and Hb. increased to greater than 20 Gm./100 ml. just before the second surgical procedure was performed. All of the previous signs returned when the shunt was no longer patent. The hemopericardium and hemothorax that occurred following the Pott's operation were probably a result of bleeding from the tear in the pulmonary artery. The initial hemorrhage from this tear was controlled at the time of surgery by pressure and by absorbable hemostatic material; however, the increase in pulmonary arterial pressure that followed the anastomosis apparently dislodged the clot that had formed.

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