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Aortic Embolism in Cats: Prevalence, Surgical Treatment and Electrocardiography

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Appendix 6.11 of James W. Buchanan's dissertation *Chronic Valve Disease and Left Atrial Splitting in the Dog*

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Aortic Embolism in Cats: Prevalence, Surgical Treatment and Electrocardiography

Abstract

Aortic embolism (caudal arterial thromboembolism) was diagnosed over a four-year-period in 14 out of 2,000 cats in a hospital clinic population (7/1,000). Including 35 cases reported in the literature, the average age of 50 cats with aortic embolism was 6-8 years (range one to 16 years). Of these, 37 were males and 13 were females. Endocarditis with thrombosis was the most frequently observed cause of aortic embolism, although aortic arteriosclerosis was reported in one cat.

The clinical and pathological features of aortic embolism in five cats are described in this report. In electrocardiograms of four of these, arrhythmias or conduction disturbances were recorded. Intact emboli in the aorta and external iliac arteries were removed by abdominal aortic embolectomy in two cats within hours after the onset of posterior paralysis. Death resulted in one case from cardiac complications and in the other by euthanasia at the later date because of probably recurrent aortic embolism. In the other three cases, multiple sections of the aorta with the embolus *in situ* were examined, but no microscopic changes in the aortic wall were noted.

Surgical removal of an aortic embolus is technically and economically feasible and is considered the treatment of choice when treatment is requested within hours after the onset of clinical signs. Although embolectomy can yield a good immediate result; the long range justification for such therapy requires further evaluation, since recurrent embolization may develop.

Disciplines

Cardiology | Medicine and Health Sciences | Veterinary Medicine

Comments

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Aortic Embolism in Cats: Prevalence, Surgical Treatment and Electrocardiography

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SUMMARY.—Aortic embolism (caudal arterial thrombo-embolism) was diagnosed over a four-year-period in 14 out of 2,000 cats in a hospital clinic population (7/1,000). Including 35 cases reported in the literature, the average age of 50 cats with aortic embolism was 6.8 years (range one to 16 years). Of these, 37 were males and 13 were females. Endocarditis with thrombosis was the most frequently observed cause of aortic embolism, although aortic arteriosclerosis was reported in one cat.

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Introduction

THE clinical recognition of aortic embolism in cats has been reported frequently in recent years. Treatment by aortic embolectomy has been suggested by several authors, but the results of such treatment were mentioned in only three reports.

This communication presents the case histories of five cats with occlusion of the abdominal aorta at its bifurcation and describes the techniques and results of aortic embolectomy in two of these. Abnormal electrocardiograms were found in four cats. The prevalence rate, age, and sex distribution of this condition in 15 cats examined at the School of Veterinary Medicine, University of Pennsylvania, and 36 cases in the literature have been tabulated.

Literature Review

Arterial obstruction, causing a syndrome of paralysis, absence of pulsation, pain, coldness, and hardening of muscles in the hind legs of cats has been termed

variously aortic thrombosis, saddle thrombus, aortic embolism, or saddle embolism. These terms are defined as follows: a thrombus is a blood clot formed *in situ* in a blood vessel or one of the cavities of the heart (Stedman, 1961); thrombosis is the process of thrombus formation, and embolism is the transportation and impaction of abnormal material in a blood vessel. Although a variety of material may enter the circulation, the most common cause of embolism is dislodgement of the whole or part of a thrombus, and unless otherwise specified, the term embolism usually refers to this process (Florey, 1962). A saddle embolus is one which straddles the bifurcation of the aorta (Stedman, 1961).

On the basis of *post-mortem* information in previous reports and the cases presented in this paper, the most appropriate of these terms in many cases appears to be saddle embolism. This term, however, is not entirely adequate for this condition since additional thrombus formation may occur distal to the embolus as has been noted also by Holzworth, Simpson and Wind (1955). In addition, microscopic changes in the aortic wall of one cat could justify a diagnosis of primary aortic thrombosis in that case (Kasbohm & Riedel, 1963). In another cat, emboli were found in both external iliac arteries, but not in the aorta (Palumbo & Hubbard, 1966). Emboli may be located in the abdominal aorta cranial to the bifurcation and not directly occluding the orifices of the external iliac arteries (Holzworth *et al.*, 1955; Freak, 1956). In a few instances, emboli have been found lodged at more than one site in the abdominal aorta (Holzworth *et al.*, 1955; Imhoff and Tashjian, 1961).

Because of the variety of pathological findings in cats having a similar clinical syndrome, it might be useful to suggest a new term which would be more general than those which have been used. A term which seems to be appropriate for all the forms observed is "caudal arterial thrombo-embolism".

Although an abbreviation of this term provides a tempting catchy label for a syndrome in cats, the dictates of conservative medical writing speak against the addition of a new term when an older one is not entirely unsatisfactory. For this reason,

TABLE I

Age in years Sex	(0-2)		(3-5)		(6-8)		(9-11)		(12+)		(? yrs.)		Totals
	M	F	M	F	M	F	M	F	M	F	M	F	
Collet (1930)	-	-	-	-	1	-	-	-	-	-	-	-	1
Holzworth <i>et al.</i> (1955)	2	-	2	2	1	-	1	1	2	-	-	-	11
Freak (1956)	-	-	-	-	-	-	1	1	-	-	-	-	2
Shouse & Meier (1956)	-	-	1	-	1	1	1	-	1	-	-	-	5
Joshua (1957)	-	-	1	-	2	-	1	-	-	-	1	-	5
Zervins (1958)	-	-	1	-	-	-	-	-	-	-	-	-	1
Kasbohm & Riedel (1963)	-	-	1	-	2	-	-	-	-	-	-	-	1
Tashjian <i>et al.</i> (1965)	-	-	-	-	2	2	1	-	1	-	-	-	6
Das (1966)	-	-	1	1	-	-	-	-	1	-	-	-	3
Palumbo & Hubbard (1966)	-	-	-	-	-	-	1	-	-	-	-	-	1
Univ. Pa (1961-65)	-	1	5	2	3	2	2	-	-	-	-	-	15
Totals	3		17		15		10		5		1		51*

*Two additional cases of unknown age and sex were reported by Imhoff & Tashjian (1961).

the authors have selected aortic embolism as the best of those available, since it applies to those cases with saddle embolism, those where an embolus lodges in the aorta cranial to its bifurcation, and those where more than one aortic embolus is found.

At least 38 cats with aortic embolism have been reported previously. A summary of 36 of these cases in which the age or sex was given, is presented in Table I. Also included are the age and sex of 15 additional cases examined at the School of Veterinary Medicine, University of Pennsylvania.

Holzworth *et al.* (1955) reviewed the literature on the occurrence of "aortic thrombosis" in the cat and quoted Collet (1930) as being the first to describe a case in which the diagnosis was made before death. Other clinical reports of "aortic thrombosis" have been published by Freak (1956) and by Joshua (1957).

Holzworth (1958) reported that "arterial thrombosis" was the principal cause of sudden paralysis in the cat and mentioned having encountered about 30 cases. She cautioned against the advisability of surgery and apparently relied entirely upon supportive medical therapy. Emphasis was placed upon the fact that the primary trouble was usually in the heart.

The report of Shouse and Meier (1956) on animals with acute vegetative endocarditis included 13 cats. In five cats with sudden posterior paralysis, "aortic thrombosis" was considered secondary to vegetative endocarditis at *post-mortem* examination.

Imhoff and Tashjian (1961) described a technique for aortography and illustrated its use in the diagnosis of aortic embolism.

Imhoff (1961) was unable to reproduce the clinical syndrome of acute aortic embolism *i.e.* paraplegia and pain, in any of 10 cats by single or two separate aortic ligatures just cranial to the bifurcation. However, in three other cats, paralysis did occur after combined ligation and administration of bovine thrombin. This substance was injected into the aorta between two ligatures 1.5 cm. apart. After formation of a soft blood clot, the cranial ligature was removed. The following day, all three cats treated in this manner were paraplegic. The effect of bovine thrombin without ligation was not mentioned.

Bardens and Walker (1962) reported their surgical approach for the removal of "thrombi from the external iliacs" of cats. Clinical data were not published, but the recurrent nature of the problem was mentioned.

Tashjian, Presinger, Das, Reid, and Crescenzi (1963) found two cases of aortic embolism in 102 cats examined *post mortem*. The site of occlusion was illustrated by angiography in one of these. In this cat, one of the iliac arteries distal to the embolus was rendered opaque by collateral blood flow; however, the animal was reportedly unable to use either hind leg.

Tashjian, Presinger, Palich, Hamlin and Yamo (1965), in a report on the cardiovascular lesions found in 202 consecutive feline necropsies, listed six cats with aortic embolism. Two of these were included in the report by Tashjian *et al.* in 1963 (Das, 1966 personal communication). A clinical report of one cat was included in which embolectomy was performed.

Palumbo and Hubbard (1966) successfully removed two separate emboli from the external iliac arteries of a cat. They detected no abnormalities in electrocardiograms before and after surgery.

Case Histories

Case 1: (1198 J)

A five-year-old castrated male cat was found collapsed on the front doorstep by its owners 45 minutes before presentation. They assumed that the animal had been the victim of a car accident.

Upon physical examination, bilateral hind leg paralysis was apparent. The cat appeared greatly distressed and when handled would roll onto its back and cry out. There was rapid open-mouth breathing and the heart rate exceeded 160 per minute. The mucous membranes were pale. Abdominal palpation was resented. The hindlegs were cool and gastrocnemius muscle spasm was not present. Capillary flush did not occur following release of pressure on the digital pads, and femoral artery pulses were absent. Pulsation of the abdominal aorta could be palpated per rectum, but the iliac arteries could not be felt. No visible abnormalities were seen in a lateral abdominal radiograph. A diagnosis of aortic embolism was made, and surgery was begun within two hours after the onset of clinical signs.

The animal was pre-medicated with 0.25 mg. atropine sulphate and 20 mg. promazine hydrochloride*. Anaesthesia was induced with intravenous sodium thiopentone†. The trachea was intubated, and the cat was maintained on oxygen and ether. At this time, the hind legs were noticeably colder than when first examined.

A 10 cm. mid-line abdominal incision was made which extended cranially from the pubis. The intestines and omentum were retracted cranially, and the bladder was lifted out caudally with moist sponges. After exposure of the aorta, the site of occlusion was immediately apparent. A blue-red mass was visible through the wall of the terminal aorta which extended into both external iliac arteries (0.5 cm. into the right and 3 cm. into the left). The connective tissue was freed from the terminal aorta, and an umbilical tape loop was placed around the aorta 2 cm. cranial to the obstruction. By elevating this loop, effective occlusion of the aorta was obtained. A longitudinal aortic incision of 1 cm. was made just cranial to the bifurcation. As the incision was made, the soft embolic material bulged through it and the intact embolus was extracted by gentle traction using a mosquito haemostat. Some blood was lost by retrograde flow from the iliac vessels before these were occluded. The aortic incision was closed with a continuous suture of 5-0 cardiovascular silk. There was immediate pulsation in the right femoral artery. Capillary flow was detected in the left digital pads, and flow was apparent in the left external iliac artery, but there was no left femoral artery pulse. The abdominal incision was closed in a routine manner. The operation lasted 45 minutes. Blood loss was estimated at 15 ml. and the cat received 50 ml. of saline intravenously. During aortic manipulations, the pulse rate rose from 150 to 200 per minute, but stabilised at 150 after aortic closure.

Post-surgically, the cat was given 50 mg. oxytetracycline‡ twice daily and 1,000 units of heparin sodium once daily.

Twelve hours after surgery, there was a strong right femoral artery pulse, but none was evident in the left limb. There was lameness as well as some hardening of the gastrocnemius muscle in the left leg, although the cat was able to walk. The day following surgery, a holosystolic murmur was audible in the right fourth intercostal space. The murmur varied in intensity from grades one to three (out of five), depending upon the position of the animal. The heart sounds were neither accentuated nor split, and no precordial thrills were palpable. In an electrocardiogram, the mean QRS axis in the frontal plane was $+95^\circ$. There was slight ST segment depression (0.05 mv.) in a unipolar exploring lead at the fifth right intercostal space just lateral to the sternum. A regular sinus rhythm was present at a rate of 165 per minute.

In lateral and ventrodorsal thoracic radiographs, enlargement of the left ventricle was evident (Fig. 1).

The intra-aortic specimen removed at surgery

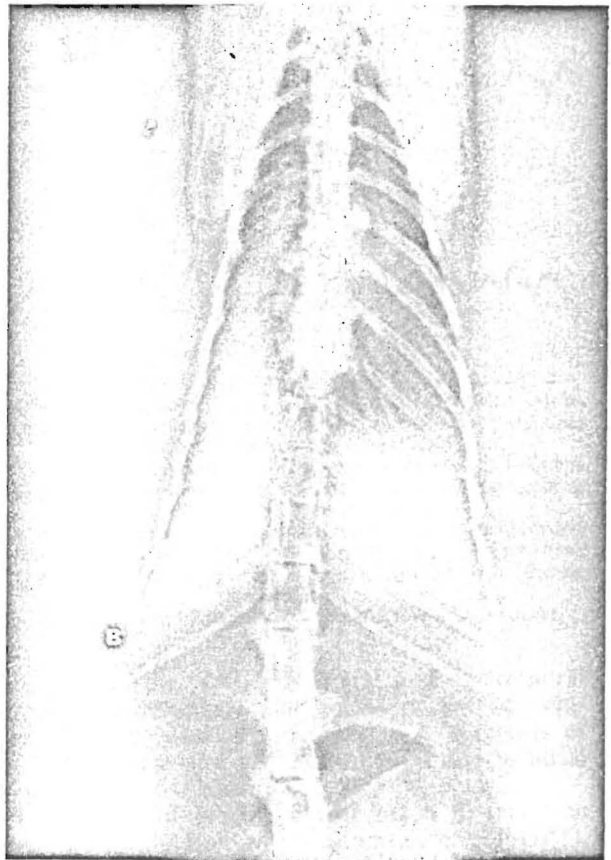
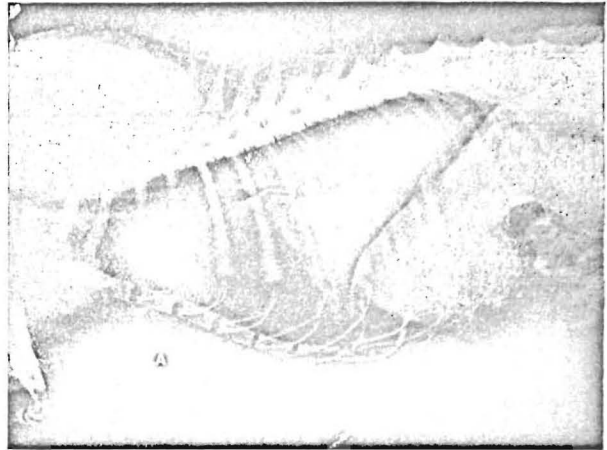


FIG. 1.—In lateral (A) and dorsoventral (B) radiographs of Case 1, enlargement, primarily of the left ventricle, is visible. In this and all subsequent dorsoventral radiographs, the cat's left is to the reader's right.

was examined histologically. It consisted of clotted fibrin with islands of mature neutrophils and red blood cells and was interpreted as being a thrombotic embolus.

The only post-operative complication was a subcutaneous haematoma at the surgical site which was possibly related to the anticoagulant therapy. Heparin was discontinued four days after surgery. At this

*Sparine: Wyeth Laboratories Inc., Philadelphia, P.A.

†Pentothal: Abbott Laboratories, North Chicago, Ill.

‡Tetramycin: Chas. Pfizer & Co. Inc., New York, N.Y.

time, the cat had no signs of lameness and appeared to be in good health. It was discharged five days after surgery, and skin sutures were removed on the ninth day after surgery. The cat appeared healthy and had adequate circulation to the left rear leg, even though a pulse rate could be detected only high in the femoral triangle. The right femoral artery pulsated normally.

Six weeks later, the cat suddenly appeared distressed and developed paralysis of the right front leg. Upon examination the following day, the temperature was 100°F., the respiratory rate was 60 per minute, and both femoral artery pulses were present. The right elbow was dropped and the carpus was flexed simulating a radial nerve paralysis. The right foreleg was cold. Induration of the triceps, biceps, and antebrachial extensors was noted. No brachial pulse was palpable but by pressing the digital pads, some capillary flushing and refilling could be detected. A diagnosis of embolic occlusion of the right brachial artery was made.

The cat was admitted and treated with 100 mg. of chloramphenicol* and 1,000 units of heparin sodium daily. Electrocardiographically, there was a regular sinus rhythm at a rate of 175 per minute. ST segment elevation (0.1 mv.) was present in lead II and to a lesser extent in leads III and CV6LL. The haemogram was within normal limits.

During the five days in hospital, there was a gradual return of circulation to the affected limb, although functional usage of the limb was delayed. At the time of discharge, there was still some knuckling of the right forepaw and active extension of the elbow was 50 per cent. of normal. The owner reported that function of this limb returned to nearly normal over the next month.

Three months later, the owners were contacted and it was learned that hind leg collapse had occurred the week before. Euthanasia had been performed elsewhere, and a necropsy was not carried out. Our assumption was that either embolism had recurred at the aortic bifurcation, or that thrombosis had developed at the site of aortic embolectomy. The latter is unlikely because four and a half months had elapsed.

Case II: (2128 H)

A six-year-old spayed female domestic cat was presented with bilateral hind leg paralysis of sudden onset approximately three hours earlier. No previous medical problems had been noted by the owner.

Upon physical examination, the recorded rectal temperature was 95.2°F., heart rate was over 200, and respirations were rapid and laboured. No pulse was palpable in either femoral artery, although the skin, toe pads, and toe nails had normal colour. The gastrocnemius muscles were firmer than normal. By auscultation, a rapid irregular heart rate was noted; however, no murmurs were detected. A right axis deviation (plus 260° in the frontal plane) was present in the electrocardiogram (Fig. 2). The ventricular rate was 240 per minute and irregular. Definite P waves could not be seen, even though carotid sinus pressure was applied in an attempt to slow the ven-

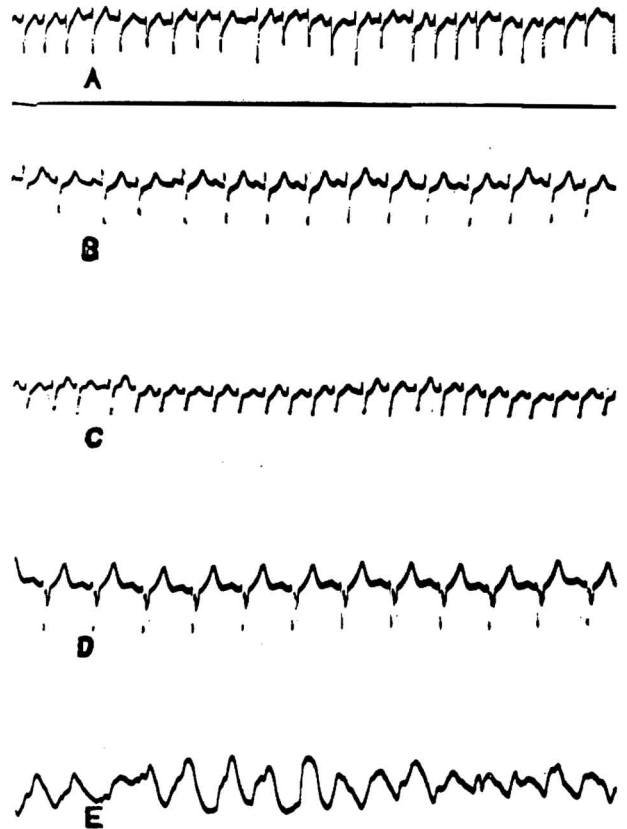


FIG. 2.—Lead II electrocardiograms from Case II.
 A—Irregular supraventricular tachycardia (probable atrial tachycardia with frequent atrial premature beats or possible atrial fibrillation) before anaesthesia.
 B—Slower atrial rhythm (probably sinus origin) with frequent atrial premature beats after treatment of cardiac arrest during induction of anaesthesia.
 C—Gradual return to irregular supraventricular tachycardia.
 D—Regular sinus rhythm with intraventricular conduction delay after 40 minutes of treatment for cardiac arrest during thoracotomy.
 E—Irreversible ventricular fibrillation a few minutes later.

tricular rate (Fig. 2A). In lateral and dorsoventral thoracic radiographs, enlargement of the left ventricle and left atrium were seen. A diagnosis of aortic embolism and atrial fibrillation due to atrial myocardial disease was made.

The results of blood samples taken at this time were: serum glutamic pyruvic transaminase (SGPT), 86 Sigma Frankel units (normal below 16); serum glutamic oxalacetic transaminase (SGOT), 180 Sigma Frankel units (normal below 19); blood urea nitrogen (BUN), 25 mg. per cent. (urograph test).

One hour after admission, the gastrocnemius muscles were in noticeably greater spasm and some hardening of the biceps femoris muscles was detected. Two hours after admission, the cat was anaesthetised with 90 mg. of pentobarbital sodium. During the induction of anaesthesia under ECG monitoring, cardiac arrest occurred. Circulation was maintained by external cardiac massage and spontaneous cardiac activity resumed following ventilation with oxygen

*Chloromycetin: Parke, Davis & Co., Detroit, Mich.

under intermittent positive pressure and an intracardiac injection of 2 cc. of epinephrine (1:10,000). About two minutes after the resumption of spontaneous circulation, the ventricular rate slowed from 200 to 140 per minute and was governed predominantly by a sinus rhythm with frequent atrial premature beats (Fig. 2B). The sinus rhythm persisted for approximately 20 minutes after which the sinus rate became more rapid, atrial premature beats were more frequent, and later atrial tachycardia with frequent atrial premature beats became manifest (Fig. 2C). It was not possible to be certain that the original tracing interpreted as atrial fibrillation might not represent atrial tachycardia and atrial premature beats also. Attempts to record the atrial activity with an intraoesophageal electrode were unsuccessful.

Approximately four hours after admission, angiocardigrams were made. In these, marked delay in passage of a contrast medium* through the heart was noted (Fig. 3). In addition, the lack of opacity in the dilated left atrium was considered evidence

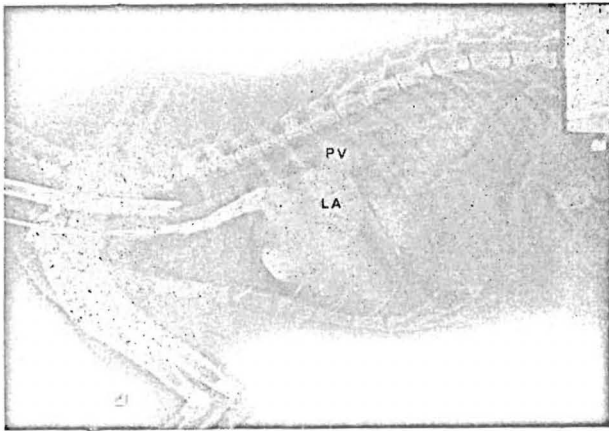


FIG. 3.—Lateral angiocardigram of Case II made 15 seconds after right atrial injection of 5 cc. of contrast material. Circulation of dye through the heart was markedly delayed. The pulmonary veins (PV) and left atrium (LA) are dilated. Because of the roundness and decreased opacification of the left atrium, a left atrial ball thrombus was suspected. None was found at surgery.

of a left atrial ball thrombus as described in another cat with a similar angiocardigram (Buchanan, 1965). By the time the angiocardigrams were made, cyanosis in the hind leg tow pads and nails had developed. An hour later when the cat was clipped and prepared for surgery, a sharp line of demarcation was apparent between the pink abdominal skin and the cyanotic skin of both legs.

The surgical approach to the abdominal aorta was the same as that in Case 1. The dark saddle embolus was visible through the aortic wall in the terminal 1 cm. of the abdominal aorta and extended into each external iliac artery for a distance of about 1 cm. Small vascular bulldog clamps were placed on the aorta 2 cm. cranial to the embolus and 2 cm. distal to the aortic bifurcation on each iliac artery. A longi-

tudinal aortic incision of 1 cm. was made cranial to the occlusion. By gently "milking" the embolus toward the incision, it was removed intact. Individual temporary release of each iliac artery clamp permitted a retrograde flush of blood from each vessel; however, no additional embolic material was noted. The aortic incision was closed with a continuous suture of 5-0 silk on a fine cardiovascular needle with sutures placed 1 mm. apart. As soon as the aortic clamp was removed, strong femoral artery pulses were noted in both legs, and the skin on the legs became pink. The abdomen was closed in routine fashion.

The thorax was then entered through the left fifth intercostal space to remove the suspected left atrial ball thrombus through an atriotomy during temporary occlusion of venous return. Upon exposure of the heart, the left atrium was noted to be markedly distended, and the atrial myocardium was bright red in colour to an abnormal degree. Cautious finger tip ballotment of the atrium did not confirm the presence of a ball thrombus. It did result in the onset of atrial fibrillation followed by ventricular asystole. Resuscitative measures for 40 minutes including cardiac massage, epinephrine, sodium bicarbonate, and calcium gluconate resulted in a return to normal sinus rhythm with effective cardiac output (Fig. 2D). This lasted for 10 minutes, following which the heart action deteriorated ending in irreversible ventricular fibrillation (Fig. 2E).

At necropsy, no thrombus was present in the left atrium, ventricle, or arterial system although a severe myocarditis of the left atrium was present. The aorta at its bifurcation was histologically normal. No explanation was found for the electrocardiographic evidence of a cranially directed mean QRS vector in the frontal plane.

Case III: (102 H)

A five and a half-year-old spayed female Siamese cat was presented with bilateral hind leg paralysis of 15 hours duration and no other pertinent medical history. (This cat was not included when the 14/2,000 prevalence ratio was determined, since it was known to be referred by a local veterinarian.)

The cat was alert and without respiratory distress, but appeared to be uncomfortable. Rectal temperature was 98.2° F. The heart rate was 160 per minute and both femoral pulses were absent. The hind legs were in extension, and the cat was unable to support itself. No reflex could be elicited in either hind leg using a needle. Both hind legs were cold and the nail beds were dark blue compared to the pink nail beds of the forepaws. The tail was cold also, but reacted feebly to needle pricking. The gastrocnemius muscles were very hard, and other muscles of the hind legs were slightly firmer than normal.

A lateral radiograph of the thorax was considered within normal limits. In the dorsoventral radiograph, however, evidence of left and right atrial enlargement was seen (Fig. 4). The electrocardiogram was technically unsatisfactory because of excessive muscle tremor; however, one ventricular premature beat was noted. In an exploring unipolar chest electrode over the free wall of the right ventricle (CV5RL), the ST

*Cardiografin 85 per cent.: E. R. Squibb & Sons, New York.

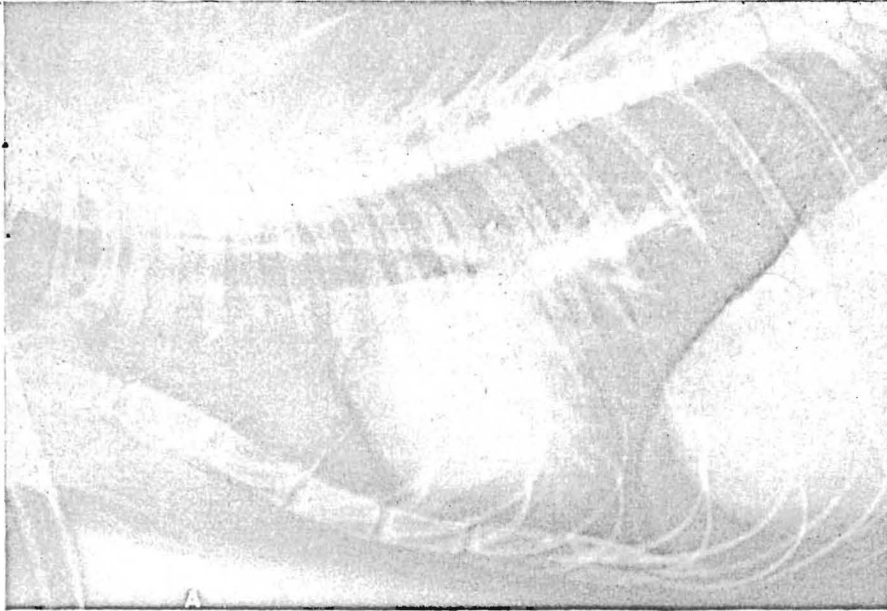


FIG. 4.—The cardiac silhouette of Case III in the lateral radiograph (A) was within normal limits. In the dorsoventral radiograph (B), moderate enlargement of both atria was considered present.

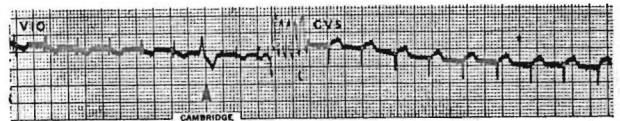
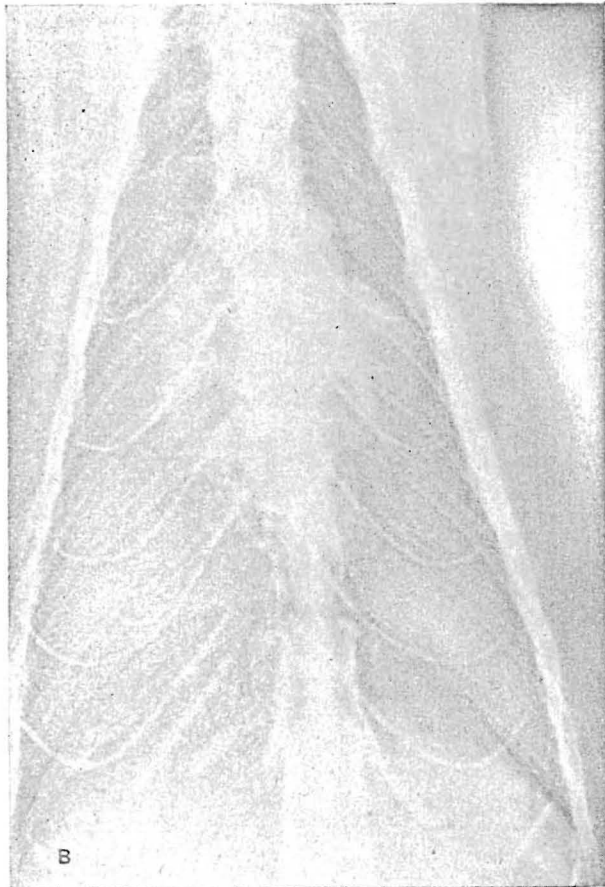


FIG. 5.—Electrocardiogram from Case III. A ventricular premature beat (arrow) is present in the initial half of the tracing (lead V10). In the second half of the tracing (lead CV5), the S-T segment is elevated 0.15 mv. and the QRS complexes are primarily negative. In this and all other electrocardiograms in this report, a recording sensitivity of 1 cm. per mv. was used.

barbital followed by immediate *post-mortem* examination.

A large myocardial infarct was found in the apical third of the left ventricular wall extending into the interventricular septum. Microscopically, this was well defined and in an early stage or organisation (capillary proliferation, macrophages, mild leucocytic and lymphoid cellular infiltration, beginning fibrous repair). Focal hyperaemia and haemorrhage (erythrocytes and haemosiderin) surrounded the infarct. No pathologic changes were observed in the coronary arteries in the sections examined, and the cause of infarction was not determined. The age of the infarct was considered in excess of the 15 hour duration of *ante-mortem* paralysis. In retrospect, the electrocardiographic waves in lead CV5RL were similar to changes seen in electrocardiograms of human patients and experimental animals with myocardial infarction.

Thrombo-embolic material was present in the abdominal aorta beginning 3 cm. cranial to its bifurcation and extending distally into all the examined arteries of both hind legs at least as far as the metatarsal region. Microscopically, no abnormalities were seen in four different sections of the wall of the aorta which were cut with the embolus undisturbed.

segment was elevated 0.15 mv., and the ventricular complex (QRS) was nearly all negative (Fig. 5).

A diagnosis of aortic embolism was made and euthanasia was performed with intravenous sodium pento-

Case IV: (1523 H)

A six-year-old spayed female domestic cat was presented during the night as a suspected accident case. The cat had been out-of-doors and when let into the house, it began crying out and running in circles.

Upon physical examination, the cat was very excited, rectal temperature was 99.0° F., heart rate was 220 per minute, and the respiratory rate was 100 per minute. The cat could walk and was difficult to handle. The chief clinical sign was apparent pain on palpation of the pelvic region. A diagnosis of pelvic fracture or hip luxation was considered. The cat was sedated with 24 mg. promazine hydrochloride, and pelvic radiographs were taken which were normal.

Approximately 12 hours later, the cat was found in a semicomatose condition and had all the signs of saddle embolism: bilateral hind leg paralysis, cold hind legs, cyanotic skin and nailbeds, absence of reflexes and femoral artery pulses, and hardness of hind leg muscle groups, especially the gastrocnemius muscles. In an electrocardiogram, sinus tachycardia was present at a rate of 210 per minute (Fig. 6). The R wave amplitude in lead II was 2.8 millivolts which probably exceeds normal limits. Elevation of the S-T segment (0.15 mv.) was noted in leads II, III, and AVF. In thoracic radiographs, the cardiac silhouette was considerably enlarged with special prominence of the right side of the heart. The results of blood tests made at this time were as follows: packed cell volume 27 per cent.; haemoglobin 8.5 gm. per cent.; RBC—6,120,000 per cm. WBC—15,850 per cm.; mature neutrophils 78 per cent.; immature neutrophils 3 per cent.; lymphocytes 19 per cent. The blood clotting time was two minutes 15 seconds; SGPT 240 Sigma Frankel units; SGOT greater than 1,000 Sigma Frankel units. A blood culture taken before antibiotics were given was negative. The cat was then given supportive therapy consisting of fluids and anti-

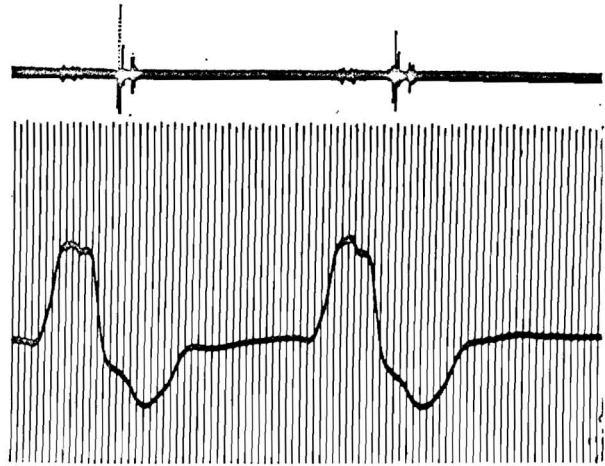


Fig. 7.—Phonocardiogram from Case IV made just prior to death illustrating splitting of the second heart sound and a slow idioventricular rhythm.

biotics in addition to streptokinase 10,000 units, streptodornase 2,500 units, and human plasminogen* 50 units intravenously.

On the second morning after admission, the cat was moribund. An electrocardiogram was made which revealed an idioventricular rhythm at a rate of 65 per minute with prolonged QRS and T complexes and no P waves (Fig. 6). A split second heart sound was noted by auscultation and confirmed by phonocardiography (Fig. 7). The cause of this was probably asynchronous ventricular contraction and relaxation evidenced by the prolonged QRS and T complexes. Hyperkalaemia was considered as a possible cause of these abnormalities, although similar changes have been noted in moribund animals dying from other diseases. Blood studies were requested for electrolyte determinations; however, the cat died before these could be obtained.

At necropsy, an embolus was present in the aorta at the level of the external iliac arteries. Microscopically, no pathologic changes in the vessel wall were seen in sections of four blocks of the aorta in the region of the embolus.

Case V: (6710 J)

A nine-year-old castrated male domestic cat was presented with a history of illness of 20 hours duration characterised by intermittent diarrhoea and bilateral hind leg weakness which progressed to paralysis.

On physical examination, the cat was panting; pupils were widely dilated; rectal temperature was 99° F.; and the mucous membranes were pale. The femoral pulses were absent; the hind legs were cold and paralysed in extension; the gastrocnemius muscles, and later the quadriceps muscles were hard. The nail beds were cyanotic.

In an electrocardiogram, definite P waves could not be seen, and the heart rate appeared to be governed by two idioventricular pacemakers (Fig. 8). It is possible, however, that the slower pacemaker could

*Varizyme: American Cynamid Co., Princeton, N.J.

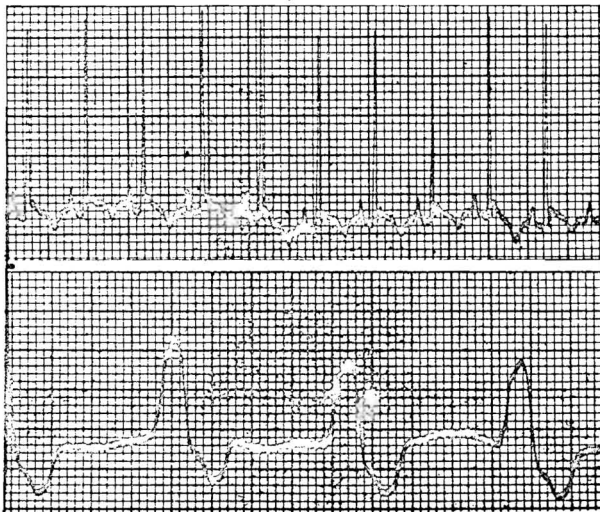


Fig. 6.—Lead AVF electrocardiograms from Case IV. In the upper tracing made on the day after admission, sinus tachycardia (210 per min.) is present. The R wave amplitude is 2.5 mv. and the S-T segment is elevated 0.15 mv. In the lower tracing made the following day, a slow idioventricular rhythm with prolonged QRS-T complexes is present. P waves are absent.



FIG. 8.—Non-simultaneous lead AVR and AVF electrocardiograms from Case V. The cardiac rhythm is apparently governed by two ventricular foci. No P waves are visible in the tracings. The slower rhythm (110 per min.) is interrupted near the end of AVR by the onset of a more rapid ventricular tachycardia (150 per min.). In lead AVF, a similar change-over is noted and a fusion beat occurred in the middle of the tracing (arrow). See text for additional comments.

have been located in the AV node. Although neither pacemaker had an absolutely fixed rate, the slower one was fairly constant at 115 beats per minute, and the faster one was more regular at 140 beats per minute. Occasionally, nearly simultaneous discharge of both pacemakers produced fusion beats of various forms.

Although paroxysmal ventricular tachycardia is a reliable clinical indication of myocardial disease and always occurs in dogs with experimental myocardial infarction, it is not usually associated with an absence of P waves. The latter does occur in advanced stages of hyperkalaemia. Although the serum potassium level was not determined in this case, evidence that hyperkalaemia could have been present was seen in related findings: elevated BUN (68 mg. per cent.), 20 hours duration of illness with probable muscle necrosis, and the presence of a large renal infarct on *post-mortem* examination. The characteristics of the QRS-T complexes occurring at a rate of 100 per minute in lead III were similar to classical changes observed in dogs and man with an apparent A-V nodal rhythm when serum potassium reaches a level of 8 to 9 mEq. per litre: absence of P waves, elevation and peaking of T waves, and prolongation of the QRS duration.

In a dorsoventral thoracic radiograph, the heart appeared enlarged in the regions of the right and left atria (Fig. 9). A diagnosis of aortic embolism was made, and a poor prognosis was given because of the time elapsed and the severe electrocardiographic abnormalities.

An angiogram was attempted to demonstrate the site of occlusion, but the cat died during the procedure.

At autopsy, both atria were dilated. No thrombi were visible grossly in the heart. The aorta was occluded throughout a 4 cm. length extending cranially from the external iliac arteries. The embolus did not enter into the external iliac vessels. A large haemorrhagic infarct was present in the right kidney. The abdominal aorta was removed with the embolus intact and fixed in formalin. In multiple sections from two blocks, no pathologic changes were seen in the aortic wall. On this basis, a diagnosis of aortic embolism of probable cardiac origin was made.

Discussion

The question of nomenclature for this condition has been raised by Holzworth *et al.* (1955) and by

Imhoff and Tashjian (1961). Terms which have been used in the past are defined in the literature review (q.v.). The present authors regard the term caudal arterial thrombo-embolism as inclusive for all the forms of arterial obstruction reported in cats with bilateral hind leg paralysis. If a new term is to be avoided, however, the most satisfactory of those in the literature is considered to be aortic embolism. The basis for this is the consistent absence of microscopic changes in the aortic wall at the level of the occlusion which would account for blood clotting at this site. Cardiac disease, on the other hand, is an associated finding either clinically or at necropsy.

Kasbohm and Riedel (1963) are the only authors to describe degenerative changes in the aortic wall in association with "aortic thrombosis" in one cat. In other cases reported in the literature, and in four cases in this series in which histopathological studies from multiple sections of several tissue blocks of the intact area of aortic occlusion were done, no changes in the aortic wall were seen which could account for formation of a thrombus *in situ*.

An additional complication regarding terminology was found in Case III of this series where distal extension of the embolus by thrombosis had occurred in practically all arteries of the hindlegs. This probably occurs, but to a lesser degree, in most cases of aortic embolism. The obstruction usually extends for a few



FIG. 9.—Dorsoventral radiograph of Case V. Marked enlargement of both atria was confirmed at necropsy an hour later.

millimetres into each iliac artery which gives it the characteristic "saddle shape" when removed at surgery or necropsy.

Of the cases in this report and in the literature, necropsy information was available on 36 animals. In 32 of these (89 per cent.), evidence of various types of heart disease was found, including myocardial infarction, chamber enlargement, myocarditis, vegetative valvular endocarditis, and mural endocarditis with thrombosis. In four cases in this report, electrocardiograms were obtained before treatment and all had ECG abnormalities compatible with myocardial disease.

Although most of the clinical and *post-mortem* evidence supports a diagnosis of primary heart disease with secondary aortic embolism, gross examination of the heart does not always reveal a site of thrombus formation even when myocarditis is found histologically. Shouse and Meier (1956) commented on the slender attachment that atrial thrombi may have, which after dislodgement, would only be revealed by an extensive histopathologic study. Further studies are indicated to assess the correct relationship between endocardial lesions and aortic embolism in cats.

Shouse and Meier (1956) also discussed the aetiology of vegetative endocarditis and the difficulty in isolating micro-organisms from the blood stream. In two cases (I and IV) in the present series, *ante-mortem* blood cultures were negative. At necropsy of case IV however, an *Aerobacter sp.* was isolated from the brain and *post-mortem* blood and was considered a probable contaminant. An alpha *Haemolytic streptococcus* was isolated from the left atrium and pericardial fluid.

The cardinal sign of aortic embolism in cats is hind leg paralysis of sudden onset without a history of trauma. An absence of femoral pulses, combined with increasing coldness of the extremities, is confirmation of arterial obstruction. Muscle spasm and cyanosis may not be present at the onset of paralysis and on the basis of three cases reported here, possibly more than two hours time must elapse before spasm occurs, and even longer before cyanosis becomes evident.

Additional signs which have been observed include respiratory distress, tachycardia, cardiac murmurs, and arrhythmias. Pain appears to be present in cats with aortic embolism at an early stage. After complete paralysis, and when muscle spasm and cyanosis are evident, signs of pain on palpation usually regress.

Temperature recordings per rectum are not reliable, since the coldness of the hind legs and pelvic muscle groups may falsely suggest systemic hypothermia. In Case II, for example, the recorded rectal temperature was 95.2° F., although the animal was warm in parts of the body other than the hind legs and pelvic area.

At this institution, an average of 480 cats per year are examined. In a total of approximately 2,000 cats, 14 cases of aortic embolism were diagnosed (prevalence ratio 7/1,000). Two factors must be mentioned in describing the cat population sample from which this prevalence ratio was determined because they probably influence the prevalence of this particular condition in this particular sample.

As an academic institution with established clinical specialities, more animals are known to be referred at the time of presentation than is the case in private practice. In addition, a few other cats have been presented without our knowledge of their having been referred until a later date. None of the 14 cases used to determine the prevalence ratio were known to be referred.

A second factor to be considered is that this condition is manifested by acute clinical signs; thus, owners request immediate veterinary service. It is common practice for some local practitioners and the police department to refer all night time emergencies to the University Veterinary Hospital where students and an intern are available at all times.

A third factor, which may, or may not, have affected the prevalence ratio concerns the number of young cats presented for elective surgery. During a one-year-period, 28 cats were spayed and 48 cats were castrated. This total accounts for approximately 15 per cent. of the cats presented to this hospital. If other veterinary hospitals have a greater percentage of cats presented for elective surgery, a corresponding decrease in the prevalence ratio of aortic embolism probably would be noted.

From another institution, Tashjian *et al.* (1965) reported six cases of aortic embolism in 202 necropsied cats. The number of cats clinically examined during this period was not given, therefore, a comparison of clinical prevalence ratios cannot be made.

The average age of 50 cats with this lesion at this institution and in the literature was 6.8 years (range: one to 16 years). Of these, 94 per cent. were three years of age or older (Table I). Based upon age specific prevalence ratios of 14 cases in 2,000 cats, aortic embolism appeared to occur most often in six to eight year old animals (Table II). Because of the small numbers available for these determinations, the differences between groups more than two years of age was not statistically significant. The conclusion that can be drawn from Tables I and II is that aortic embolism seldom occurs before three years of age and is chiefly a disease of middle aged and older cats.

In the sex ratio of cases reported in the literature, there was a predominance of male cats (male to

TABLE II
AGE SPECIFIC PREVALENCE RATIOS OF 14 CASES IN 2,000 CATS

Age in years	No. cats examined*	No. observed cases	Prevalence ratio: N/1,000
0—<1	794	—	
1—<3	549	1	2/1,000
3—<6	312	6	19/1,000
6—<9	123	5	41/1,000
9—<12	72	2	27/1,000
12+	78	—	
unknown	72	—	
	2,000	14	7/1,000 (overall)

*Estimated number, based upon age group percentages in a sample of 1,352 cats examined consecutively at the Veterinary Hospital, University of Pennsylvania.

female ratio of 3·2:1). This was in contrast to cases at this institution where the male to female ratio of cats with the lesion (1·8:1) was not significantly different from the sex ratio of the clinic population (1·4:1). Holzworth *et al.* (1955) concluded that in the Angell Memorial Hospital series, the age and sex incidence of the disease in 11 cats was distributed similarly to the "ill cat" hospital population. Tabulation of their reported cases, however, revealed a male to female ratio of 2·7:1. The total male to female sex ratio of cases in the literature and those seen at this institution was 2·9:1. A possible explanation for the apparently higher prevalence in males might be found in their propensity for being involved in cat fights. This could account for the higher incidence of bacterial endocarditis in males (10 out of 13 cats) reported by Shouse and Meier (1956).

The finding of Imhoff (1961) that clot formation, in addition to aortic ligation, was essential in reproducing the clinical syndrome has been mentioned. The aetiological rôle of clot formation in the production of paralysis was not determined, however, the author did not regard mechanical blockage of collateral vessels as a factor.

The possible rôle of some humoral agent in causing direct or reflex vasospasm would be another avenue of investigation. In addition, comparative studies of the clotting mechanism in normal and affected cats are required. These should be compared and contrasted to clot formation in dogs where embolic occlusion of the caudal aorta is a distinct rarity.

Whether or not surgery should be recommended in cats with aortic embolism depends upon the duration of time which has elapsed. If paralysis has been present for more than 12 hours, surgical benefit is doubtful, since distal extension of the clot by secondary thrombosis and irreversible muscle damage will probably have occurred. The surgical procedure is simple, and the only special requirement is a cardiovascular quality 5-0 silk suture.

The medical treatment of aortic embolism in cats is basically supportive in nature. Fibrinolytic enzymes have been used in man with variable results. No information is available in the literature concerning their use in cats. In Case IV, fibrinolytic enzymes were given, but death occurred before their effects could be evaluated. Broad spectrum antibiotics are recommended because of the frequent occurrence of endocarditis and myocarditis. Heparin was given to two cats at a dosage of 1,000 i.u. once daily, but the effect of this dosage on the clotting time was not ascertained. The efficacy of anticoagulant therapy in man has been questioned by Slade (1963), who reported recurrent aortic embolism in a patient on anticoagulant therapy. He also noted that recurrent embolism occurred in three out of five other reported patients being treated with anticoagulants following aortic embolectomy.

Holzworth *et al.* (1955) stated that "if the patient's primary heart lesion does not cause death, the peripheral disturbance is, in time, overcome". Our experience generally has been that if the occlusion is complete and cyanosis is present, recovery does not occur. In some of the cats which died, there was in-

sufficient evidence of cardiac disease to explain the cause of death. In one of these, the BUN was 68 mg. per cent., and the SGOT was over 1,000 Sigma Frankel units. In another case, the SGOT was also above 1,000 units. In these cases, death may have been due to renal failure secondary to severe muscle necrosis or renal infarction. It is, therefore, possible that some of the other fatal cases in this series and in the literature did not die of "primary heart lesions" and may have survived, had surgery been performed and muscle necrosis been avoided.

There are a few reported cases in which spontaneous recovery has occurred. This was observed in a four-year-old spayed female cat at this institution. The cat was presented with sudden posterior paralysis and no femoral pulses. A diagnosis of aortic embolism was made. In this case, however, the syndrome was not complete in that capillary filling in the toe pads persisted; cyanosis did not develop, and hardening of the gastrocnemius muscles was minimal. The cat was treated with antibiotics, heparin, and ataractics. The animal was discharged three days later after there had been a partial return of motor and sensory reflexes, although femoral pulses were still absent. The cat was able to support its weight on its hind legs but moved reluctantly. Upon examination nine months later, the cat appeared healthy and had complete and normal use of its hind limbs. Bilateral femoral pulsations were present.

The recurrent nature of the problem has been mentioned by others (Holzworth, 1955; Shouse & Meier, 1956; Joshua, 1957; Bardens & Walker, 1962). This occurred in Case I in spite of prolonged antibiotic therapy for the treatment of suspected bacterial endocarditis. Atrial thrombi, by nature of their insecure attachment, are prone to dislodge. The resultant emboli may again lodge in the aorta or at some other arterial bifurcation. The prognosis, therefore, should always be guarded, and this in turn may affect the decision to undertake surgery.

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