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AORTIC THROMBOSIS

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[WITH SPECIAL PLATE]

Aortic thrombosis (Leriche's syndrome) is generally considered to be a rare condition. The true incidence may be roughly assessed from post-mortem findings, and there are reports of several large necropsy series in the literature. Straus *et al.* (1946), combining the results from several such series, arrived at an incidence of 0.12%, a figure frequently quoted subsequently. Other figures are given in the Table :

Authors	No. of Cases	No. of Necropsies
Gross and Philips (1940)	7	5,350
Greenfield (1943)	24	16,935
Barnett, Moorman, and Merrick (1952)	10	4,394
Siegel and Garvin (1941)	9	6,547

Combining these figures produces 50 cases in 33,226 necropsies—an incidence of 0.15%, agreeing quite closely with the figure quoted by Straus *et al.*

Turning now to cases diagnosed clinically, Milanés *et al.* (1950, 1952) saw 30 such patients out of 4,560 referred to a special vascular clinic. The incidence of 0.61% is naturally higher than that found at necropsies on general hospital patients.

During a period of four years 32 patients with aortic thrombosis have been seen in the Diagnostic Radiological Department of St. Mary's Hospital, which has a large and very active unit specializing in the surgery of peripheral vascular disease and draws patients from all over the country. In all our 32 patients the diagnosis was confirmed by aortography, and the subsequent report is concerned with some of the findings in these patients, and in particular with the radiological findings. In addition to the cases of aortic thrombosis investigated by us, others have been referred to St. Mary's Hospital Surgical Unit after investigation and aortography at other hospitals, but these cases are not included in the present report.

Sex Incidence

There were 24 male and 8 female patients in the series. This sex difference has been observed previously and corresponds to the greater incidence of arterial disease in the male in sites other than the aorta. The reported proportions between male and female vary in different series, as they are bound to do when figures are small. Milanés *et al.* (1952) had five females among their 30 patients. These Cuban workers also claim four to one preponderance of whites as compared with negroes. Theis (1952) found 13 women among 29 patients, the highest proportion of females yet reported. The numbers of well-authenticated reports on single female cases is too great to mention here individually; those of Gough (1935), Horwitz (1948), and Brock (1953) are of particular interest and will be referred to later. There is therefore no justification for the statement made by Good-

win and Petrie (1951) that all indubitable cases reported to that date had been in males. It is hoped to show, however, that taken as a group there are differences in the aetiological factors between the female and male groups.

Age Distribution

The youngest patient in our series was 37 years when first seen here, the oldest 78 years. The average age for the whole series was 51.3 years. The age distribution is shown in the diagram (Fig. A). It will be seen that there is a fairly even distribution up to the age of 65 years; the single patient aged 78 years slightly exaggerates the average age of the series.

Aortic thrombosis has been reported at all ages. Cases have even been recorded in infancy and childhood, but these should probably be considered as a separate group, because they differ in aetiology. In 1935 Rothstein reported the case

FIG. A.—Diagram showing age and sex incidence in 32 cases of aortic thrombosis. Cases considered to follow embolus in mitral stenosis are in black.

of a mongol boy who died on the 13th day after birth. At necropsy the aorta was found to be occluded. The cause was thought to have been umbilical sepsis. The paper contains a review of childhood cases to that date, all derived from necropsy records; 12 patients between the ages of 2 weeks and 12 years are described. All the cases seem to have been associated with some form of sepsis, though one case occurred during the course of diphtheria and one was superimposed on syphilitic mesaortitis. More recent reports on this condition in infants are by Morison (1945) and by Marks and Fehler (1953). Morison reported necropsy findings in three infants. In one aortic thrombosis was associated with a peculiar form of arteritis; the second infant, with an aneurysm of the ductus arteriosus, showed thrombosis of the thoracic aorta only; while in the third the renal arteries were occluded. The patient reported by Marks and Fehler was an 11-months-old boy who died during the course of an upper respiratory infection and was found to have developed a thrombosed aorta. While in some of these infants cyanosis and gangrene of lower limbs was prominent,

it was not invariable, and in some arterial disease was unsuspected until necropsy. The papers mentioned here do not constitute an exhaustive review of the subject, but are quoted to draw attention to a little-known aspect of the problem. A case of aortic thrombosis complicating syphilitic aortitis and occurring in a 20-year-old female is recorded by Gough (1935).

At the other extreme of age Pinkerton (1948) reported a case of aortic thrombosis in a man aged 81.

Theis (1952) found the average age in 19 men as 55, and in 13 women as 56 years. De Wolfe *et al.* (1954) found the average age of 47 patients with aortic or iliac thrombosis to be 54.4 years, and Beaconsfield and Kunlin (1953) 52 years in 35 patients. DeBakey *et al.* (1954) try to distinguish between patients with complete and incomplete thrombosis; they found those with complete thrombosis to be somewhat younger. The significance of this at first sight rather paradoxical finding is not certain. It is clear that aortic thrombosis is not a disease of senility, but, on the contrary, is commonest in the middle period of life, an age before degenerative changes have reached their height. The aetiology of the more common adult cases is quite different from that of the rarer childhood cases already mentioned. In adults sepsis is of no importance and atheroma is the predominant cause. Aetiology is further discussed below.

Onset of Symptoms

In 20 of our patients the onset of symptoms was very gradual—that is, the patients had difficulty in dating the onset. Twelve patients experienced a more rapid beginning of their symptoms and could date the origin more definitely. The established picture of aortic occlusion, whether embolic or thrombotic in origin, is the same, however, and these patients will be discussed together as regards symptomatology.

Relation to Trauma

In three patients in the present series the onset of symptoms may have been related to trauma.

Case 1.—Man aged 44. Received a crushing injury to the abdomen when he was trapped between two lorries. He was admitted to hospital and kept in bed for several days. Two weeks after the injury he began to complain of intermittent claudication in the right calf. This spread to involve both legs and thighs and left buttock. An aortogram showed occlusion of the aorta from the level of the second lumbar vertebra to the bifurcation of the common iliac arteries.

Case 2.—A 58-year-old man involved in a car accident. Exact details of the extent of his injuries are not now available, but they necessitated a stay in bed of nine days. On getting up he complained of intermittent claudication in both legs. Aortography revealed the presence of aortic occlusion.

Case 3.—A Pole, now aged 63, received an injury to his left leg, resulting in a fracture of the left tibia and fibula, in 1948. When he resumed walking he began to experience intermittent claudication in that leg. In 1954 he spent several weeks in hospital, suffering from jaundice (probably infective hepatitis). On his discharge from hospital intermittent claudication rapidly spread to involve the whole of the left lower extremity, left buttock, and right leg. An aortogram showed occlusion of the lower abdominal aorta.

Symptoms and Signs

Intermittent Claudication

Intermittent claudication was the outstanding symptom in the cases here described. It was present in nearly all, and consisted of an aching or burning sensation brought on by exercise and relieved within a few minutes by rest. In the early stages the pain would involve the calf muscles of one leg, beginning after the patient had walked several hundred yards. Gradually the claudication distance becomes less, and the pain comes to involve the opposite calf, thighs, and sometimes hips. The full development of this symptom may take years. Eventually the patient's walking distance may be reduced to 10–15 yards (9–14 m.), and many patients have expressed embarrassment at having to stop frequently when in the street; many will pretend to look at a shop window while resting. Some patients, however, can con-

tinue to walk for considerable distances, provided they do so slowly enough. Most patients find it impossible to continue walking when the pain has reached a certain intensity, and it may then be accompanied by marked weakness. Occasionally a patient may force himself to carry on; pins and needles in the calves may then be added to the pain. The time of rest needed before pain abates becomes progressively longer, and eventually aching may persist even at rest.

Pain occurring in addition at rest was complained of by six patients only. The only patient without intermittent claudication had suffered an attack of severe paralytic poliomyelitis many years previously and was unable to walk. His complaint was continuous pain in both legs. In a few patients the distribution of the claudication pain differed from that described above and involved mainly the buttocks or was apparently situated in the hip. However, it maintained its relation to exercise and its aching or burning characteristics. Rarely pain may be replaced by tingling and weakness caused by exercise.

We have found intermittent claudication to be the most reliable symptom of arterial insufficiency. It is our experience that where requests were received for the performance of arteriography and a history of true intermittent claudication was not obtained, the arteriograms usually showed a normal arterial tree. Conversely, claudication pain may be misdiagnosed as due to other causes.

Loss of Power and Muscle Wasting

Loss of power in the legs, frequently accompanied by muscle wasting (see below), appeared to be the symptom next in frequency, and occurred in about half the cases referred to us. The loss of power and the wasting may at times be very severe. Occasionally wasting was more marked on one side than the other. Weakness and global atrophy of legs, thighs, and buttocks were features particularly stressed by Leriche (1940), who also pointed out the ease with which these patients became fatigued. Loss of power, fatigability, and wasting are presumably related directly to the impoverished blood supply to the limbs. Another possible factor, however, is ischaemic neuropathy. Burt *et al.* (1952) have observed wasting, fasciculation, and stocking anaesthesia in their patients, and regard these as a result of ischaemic neuritis. Good evidence for the occurrence of such lesions is advanced in the papers by Edwards (1952) and Edwards and LeMay (1955). However, in one of our patients with very severe wasting the sciatic and sural nerves, examined histologically after death, showed no abnormalities.

Impotence

Leriche (1940) also stressed the diagnostic significance of impotence in the male. He accurately described this as "failure of maintained erection." Seligman (1954) described one patient said to be suffering from loss of libido. This seems unusual, and one wonders whether there was in fact some confusion in terminology. Of the present patients, four males admitted to impotence; in one 64-year-old man it appears to have been temporary, beginning two years after the first onset of symptoms, lasting six months, and clearing up without treatment. It may be presumed with some confidence that the true incidence was actually higher than this, because, unfortunately, several patients were not directly questioned regarding this symptom, and because, even when approached tactfully, men are more likely to deny than admit such a deeply distressing inability. A similar incidence has been found in other series. Beaconsfield and Kunlin (1953) saw six men with impotence in a series of 35 patients. This symptom is not confined to aortic thrombosis; it has also been observed in thrombosis of the common iliac artery. Shapiro (1952), Covey (1955), and others have reported such cases. It is accepted that operative treatment may bring about a cure of this symptom in some patients. Milanés *et al.* (1950) and Ortnor and Griswold (1950) observed this after lumbar sympathectomy, and Oudot and Beaconsfield (1953) and DeBakey *et al.* (1954) after treat-

ment by arterial grafting. As stated above, however, spontaneous cure of the symptom may also occur, and the significance of operation in sometimes relieving it is not yet clear. The pathogenesis of impotence in aortic thrombosis is of some interest and will be discussed later.

Other Symptoms and Signs

Of the eight women in the series, menstruation was normal in four and four were post-menopausal. We have not observed disturbances of micturition or of bowel function. This has been reported in only one case, the patient of Tocker and Cauble (1956). She suffered from urinary incontinence, which was relieved following operation on the aorta. Since she also had a cystocele it is difficult to assess this case.

Persistent central backache is a fairly frequent complaint, though again its exact incidence cannot be assessed in the present series. In Leriche's view it was due to the intense periarteritis found in relation to the thrombosed segment of aorta. He claimed that resection of the occluded aorta relieved this symptom. We have not had an opportunity to confirm or deny this claim.

The diminished blood supply to the legs resulted almost invariably in marked coldness of the feet. Usually the patients or their spouses were very conscious of this. Exceptions were those patients who had had previous lumbar sympathectomy. While this operation does not seem to make any difference to the patient's progress, it does in many cases result in a warmer skin. Other, minor, results of the impoverished blood supply observed were dermal trophic changes such as thin, white skin over the shins, sometimes with loss of hair. The toenails were frequently ridged and deformed.

In contrast to these minor effects of ischaemia, major sequelae such as ulceration and gangrene have been uncommon. This was first pointed out by Leriche and confirmed by all subsequent reports; ulceration and gangrene are not a common feature of insidious aortic thrombosis, and should they arise may occur late in the disease. If ulceration does appear it is frequently the result of a small local injury or infection which fails to heal, and the prognosis then immediately becomes more serious. Healing of ulcers and of amputation wounds is difficult, and a distal amputation may be followed by further progressively higher operations. In the present series ulcers of a malleolus were seen in three patients and gangrene of a big toe in two. In one of the latter amputation of a big toe was required and was followed by satisfactory healing. The ages of these five patients were 44, 55, 58, 62, and 63 years—an average of 56.5 years. (Late gangrene also occurred in our oldest patient, aged 78, shortly before death. She died of myocardial infarction and not as a direct result of the aortic thrombosis.) Edwards and LeMay (1955) suggest that obstruction of branches of the aorta may give rise to visceral symptoms, without, however, quoting actual observations. In the present series one patient, a man aged 55, complained of nausea after exercise. He had an aortic occlusion below the level of the third lumbar vertebra. The inferior mesenteric artery was involved, as in most of our cases (see "Radiology" below), but the superior mesenteric artery was patent. The definite association with exercise is unusual and difficult to understand. Visceral symptoms were not encountered in any other cases of our series.

Covey (1955) mentions perineal pain and sacral formications as symptoms in patients with aortic occlusion. No such complaints were made by our patients.

Bonney (1956) and de Wolfe *et al.* (1954) drew attention to cases with atypical claudication of the hip, presenting in the first place as supposed orthopaedic problems.

Symptomless Aortic Occlusion

Finally, a number of cases have been reported in which thrombosis of the abdominal aorta was found at necropsy, having been unsuspected during life. All these patients had

been seriously ill and bedridden for some time before death; symptoms of aortic occlusion would therefore not be prominent. It is possible that thrombosis occurred in these cases at a late stage in the illness. Greenfield (1943) reported three such cases; one patient died of miliary tuberculosis, the second of bronchopneumonia, and the third of acute yellow atrophy of the liver. Frelick and Shellenberger (1954) were able to demonstrate aortic occlusion in a patient seriously ill with malignant hypertension; he had had no symptoms referable to aortic occlusion. Evidently symptoms need not normally be expected unless the patient is mobile, when the demand for oxygen by the muscles will exceed the supply. The following history, however, is of interest in this connexion.

Case 4.—The patient had an attack of paralytic poliomyelitis at the age of 21. This left him with paralysis of both legs and he has since been confined to a wheel-chair. When he was 47 he began to complain of pain in the left foot; this later spread to involve both legs. The pain was always worse at night. Examination revealed absence of femoral pulses and an aortogram proved the presence of aortic thrombosis.

The reason for his continuous rest pain is uncertain. It may be that because of his immobility there was lack of stimulus for the development of a collateral circulation, and that long disuse had been followed by hypoplasia of the vascular supply to the legs. When thrombosis supervened, therefore, the blood supply to the wasted muscles or tissues became insufficient even for their resting demands.

The most obvious sign to inspection is the profound wasting of the lower limbs and buttocks. It is not always present, however, and presumably takes some time to develop. It has already been mentioned, together with skin and nail changes, loss of hair, and possibly ulceration.

Undoubtedly the most important sign is loss of palpable arterial pulsation below the block. The femoral arteries are never felt to pulsate, nor, in our experience, the popliteal, dorsalis pedis, or posterior tibial arteries. Pulsation below arterial obstruction is said to occur at times when the patient is at rest; this disappears after exercise. The absence of pulsation in the femoral arteries does not imply that they are necessarily occluded. Arteriograms nearly always show these vessels to be patent even though no pulse is felt in them. The phenomenon is due to filling from numerous small collaterals, carrying blood over unequal distances from the aorta over a tortuous course, and producing damping of the pulse. The position is the same as in coarctation of the aorta, where the femoral pulse is usually impalpable but femoral blood flow is present.

Confusion may at times be caused by strongly pulsating collaterals in the groin. We have not encountered it to any serious extent, though faint pulsations have been felt in the groins of some patients in an unusual position, and clearly not caused by the femoral arteries. Burt *et al.* (1952) state, however, that pulsation in an inferior epigastric artery may at times be strong enough to be mistakenly attributed to the femoral artery. These authors also mention the possibility of an anatomical anomaly in which one external iliac artery arises direct from the aorta at a high level and thus fails to be occluded by thrombus lower down.

In patients with a thin abdominal wall the level of the aortic block can sometimes be made out by palpation. Above the block pulsation of the abdominal aorta may be particularly vigorous. It should be borne in mind, however, that the level of the aortic bifurcation is somewhat variable, and an apparent "block" may be due to a high bifurcation. Nevertheless there is little doubt that the actual level of the obstruction can be thus determined in some cases.

The presence of collaterals has already been mentioned. They may sometimes be seen and felt in the groins and lower abdominal quadrants, but on the whole are not prominent. They are not so widespread as in patients with coarctation of the aorta.

Burt *et al.* (1952) state that in most cases of aortic thrombosis neurological disturbances will be found, consisting not

only of wasting but also of fasciculation and "stocking" analgesia. We have certainly observed these signs in a number of patients.

Covey (1955) described as a sign of aortic thrombosis a loud musical systolic murmur over the spinous processes of the lumbar vertebrae and lower abdomen. He found this sign in two out of four patients. In this connexion it is of interest to note the apparently contradictory claim made by Edwards (1952), according to whom a systolic murmur may frequently be heard over an arteriosclerotic vessel, but disappears when this vessel becomes occluded. It may be inferred that the murmur described by Covey is a result of the collateral circulation. Since reading his article we have had an opportunity to examine ten patients with aortic thrombosis with particular reference to murmurs; a soft blowing systolic murmur over the lumbar region was heard in four. DeBakey *et al.* (1954) also observed similar murmurs over the lower abdomen.

Haematuria resulting from emboli into the kidneys is mentioned as a sign by Straus *et al.* (1946). It does not seem to have been observed by others.

Plethysmography and Ballistocardiography

We have no personal experience of plethysmography in this condition, and are therefore unable to discuss this procedure. It was not carried out in the majority of cases, and it seems unlikely that much of value was lost by omitting that investigation. The same applies to the use of the ballistocardiogram. Elkin and Cooper (1949) described changes in the tracings which they implied were characteristic, but their work does not seem to have been followed up. In view of the more complete information obtained by arteriography, these indirect methods would seem unnecessary refinements.

Associated Cardiovascular Disease

In the present series there were six patients with mitral stenosis and auricular fibrillation. These will be described separately (Starer and Sutton, in preparation). One further patient, a male aged 55, had suffered a coronary thrombosis several years before his admission to St. Mary's Hospital.* In the other patients clinical, radiological, and electrocardiographic examination failed to reveal any evidence of heart disease. Theis (1952) in his report on 29 patients stated that cardiac disease or anaemia was present in 75%, without, however, giving further details. A high incidence of heart disease was also found by Burt *et al.* (1952). Little mention of associated cardiac disease is made in the recent papers from surgical clinics (for instance, DeBakey *et al.*, 1954), presumably because of selection of patients reaching those clinics.

Hypertension is not particularly associated with aortic thrombosis. There are some patients, however, who have hypertension which is related to aortic thrombosis. These form an important group, and space is given to some discussion of this problem below. Of the present series, the diastolic pressure was below 90 mm. Hg in 18; from 90 to 100 mm. in 8; from 100 to 110 mm. in 4; and above 120 mm. in 2 patients.

Thrombophlebitis and pulmonary embolus have been mentioned as frequently associated conditions (Elliot and Peck, 1952). One of our patients, whose case is described in more detail later, developed recurrent attacks of thrombophlebitis at an early stage of his disease. No pulmonary emboli have been encountered in our patients.

Length of History

It is now well recognized that the history in these cases may extend over many years. Beaconsfield and Kunlin (1953), for instance, quote the case of a patient who is believed to have had symptoms due to aortic thrombosis for

*Another patient with a history of previous coronary thrombosis has been encountered amongst eight further cases seen since this paper was written.

21 years. In the present series the longest history was 13 years. The following table shows the length of history in the 32 cases:

Length of History (Years)	No. of Patients	Length of History (Years)	No. of Patients
13	1	4	5
8	3	3	4
7	1	2	2
6	1	1	1
5	1	Less than 1 ..	13

Examination of the histories of many of these patients suggests a relatively rapid evolution of symptoms over a period of months in the early stages. Thereafter symptoms remain unchanged or progress very slowly.

Diabetes Mellitus

No cases of diabetes mellitus were encountered in the present series.

Radiology

Plain Radiographs

The plain radiograph has little importance in the diagnosis of aortic thrombosis. The presence or absence of calcification in the abdominal aorta is of no help. It was thought that the tortuous and dilated lower intercostal and subcostal arteries which develop as part of the collateral circulation might cause notching of the lower ribs. This is seen in some of the rare cases of coarctation of the abdominal aorta. However, examination of the radiographs in our cases has failed to reveal this sign. Aortography shows that the tortuosity of the intercostal and subcostal vessels in these cases is not as marked as in coarctation. (See Special Plate, Fig. 1.)

Aortography

Aortography is the most useful procedure in making a positive diagnosis, and all our patients were so examined.

Price and Wagner (1947) are often credited with being the first to have used aortography to demonstrate thrombosis of the abdominal aorta. They examined two patients during 1945, and in the second patient were able to confirm their radiological diagnosis at necropsy. However, Leriche in his original paper already refers to cases in which aortography has been performed in his clinic before 1942. Since 1947 the method has been used increasingly. The diagnosis from bilateral iliac occlusion cannot be made with certainty without aortography (except, of course, at operation or post mortem), and the earlier case reports suffer to some degree from this diagnostic uncertainty.

In this connexion it may be useful to mention some of the pitfalls of clinical diagnosis. In our experience it is not possible always to distinguish clinically between thrombosis of the lower aorta and bilateral thrombosis of the common iliac arteries. This was also found by Milanés *et al.* (1952). We see this error made from time to time by experienced physicians and surgeons. Further, one may be misled by pulsation in a groin into believing that the femoral pulse is present, when in fact one is feeling a dilated collateral—for example, the inferior epigastric; or the disposition of vessels may be unusual.

The two constant features which between them make a diagnosis at aortography certain are an abruptly ending column of contrast medium, the block lying anywhere between the level of the renal arteries and the aortic bifurcation; and the presence of a collateral circulation. When the block is well below the origin of the renal arteries the aorta below these arteries was generally narrow and tapering. The narrowing thus produced was distinctly in excess of the normal diminution of calibre of the lower aorta. About half of the patients in the present series had aortic occlusions well below, the rest directly below, the level of the renal arteries (Fig. B and Special Plate, Figs. 2-4).

In five patients the aortic thrombus appeared to be extending into one or other renal artery (Fig. B and Special Plate, Fig. 5). This appeared on the aortogram as an area of narrowing or a filling defect at the mouth of the renal artery. In the remaining patients, however, thrombus had stopped just short of these arteries. Extensive renal arterial

involvement is likely to be rapidly fatal, but appears to be uncommon. No such cases have been seen in the Pathology Department at St. Mary's Hospital; consequently we believe that the progress of thrombosis is often slowed or halted on reaching the renal arteries. This suggests that spreading aortic thrombosis is a self-limiting disease in the large majority of patients. The reason for this is not altogether clear. When the iliac arteries become blocked the

FIG. B.—Diagrams illustrating extent of vascular occlusion in 32 consecutive cases of aortic thrombosis examined by aortography. In cases marked " ? " the lower limit of the block was not clearly defined. In cases where a renal artery is marked by an arrow (top row) the origin of the renal artery was involved.

effluents from the aorta below the renal arteries are relatively small, and a cul-de-sac is formed in which clotting can proceed rapidly and easily. At the level of the renal arteries, however, flow is torrential and clotting cannot take place so readily. Whether the degree of atheroma found at different levels in the aorta plays any part is uncertain, but it is true that atheroma maximally involves the lower abdominal aorta below the renal arteries. As thrombosis extends upwards the lumbar arteries and inferior mesenteric artery become occluded. Involvement of the latter vessel is not, as a rule, associated with any symptoms to mark its occurrence. Blood is carried from the superior mesenteric artery through its middle colic branch to the left colic branch of the inferior mesenteric artery and hence to the superior haemorrhoidal artery. These anastomotic vessels form a very constant and characteristic loop in the left upper quadrant (Special Plate, Figs. 1, 4, 5, 6). Theis (1952) has reported one death from gangrene of the bowel as a result of inferior mesenteric arterial thrombosis; this appears to be unique.

The anastomotic channels in aortic occlusion have been extensively described by Burt *et al.* (1952) and by Edwards and LeMay (1955). It is unnecessary to describe them in detail here. Although lumbar aortography gives some idea of the extent of the collateral circulation, it is unable to show the anastomosis in the anterior abdominal wall deriving from the internal mammary arteries. Further, the

extent of filling may depend on the phase at which the radiographic exposure was made. We have not found it possible to correlate the degree of the patient's disability with the extent of the collateral circulation as shown by angiography.

In most of our cases the common iliac arteries were occluded, and flow could be seen to start about the iliac bifurcation (Special Plate, Fig. 7). In one patient thrombosis extended to the commencement of the femoral artery (Special Plate, Fig. 6). Rarely the internal iliac artery appeared blocked when the external iliac artery was patent, or vice versa. Frequently a large anastomotic vessel may be seen entering the iliac arteries at the level where flow recommences. This occurs often enough to suggest that the lower limit of the block is determined by collaterals able to carry sufficient blood into the iliac arteries, thereby producing a relatively fast flow of blood which will prevent clotting.

An idea may be gained of the extent of the collateral circulation required when it is remembered that the quantity of liquid flowing through a tube is as the fourth power of the radius (Poiseuille's law) and comparing the radii of the blocked aorta with even the largest collateral.

In addition to the aortic block it may be possible to demonstrate additional and unsuspected blocks in distal leg arteries, a point of great importance in assessing prognosis and treatment.

Case 5.—Male aged 56. This man had suffered from progressive intermittent claudication, beginning in the right and spreading to the left calf, for 18 months. The legs were cold, but there was no ulceration, muscle wasting, or impotence. The femoral pulses could not be felt. A clinical diagnosis of aortic thrombosis was made. It was confirmed by aortography, and incidentally the presence of a horseshoe kidney demonstrated. There was, however, in addition occlusion of the left popliteal artery, well shown on the serial films.

Similar cases have been described by DeBakey *et al.* (1954).

Complications of Aortography

While it is proper to stress the diagnostic importance of aortography, it should be pointed out that it is a procedure which involves discomfort to the patient and carries an appreciable morbidity (McAfee, 1957). It is not an examination to be undertaken casually by the inexperienced, whether radiologist or surgeon.

The interpretation of films is, on the whole, straightforward, but Gaylis and Laws (1956) have pointed out that appearances superficially similar to those due to aortic occlusion may be obtained by subintimal injection of the contrast medium. The absence of anastomotic vessels and the persistence of the contrast medium in rapid serial films should help to avoid this diagnostic error. It should be added, however, that in a large experience of aortography by one of us (D.S.) this accident has proved very rare.

Brock (1953) has expressed the fear that puncturing the aorta may lead to an extension of thrombosis. He quotes the history of a young female patient of his own in support of this. The danger must, however, be very remote. We have seen no evidence of further spread following arteriography, and related to it, in the cases we describe, nor has thrombosis ever developed in any of the many patients without aortic thrombosis on whom aortography has been carried out at this hospital. It must also be kept in mind that thrombus in the occluded aorta may tend to spread even when nothing is done to that vessel, and that spread consists in a building up of the pre-existing clot. This appears to have happened in Brock's patient. Thrombosis in relation to the needle puncture might reasonably be expected to occur at the site of puncture, which is several inches above the level of the block. This in fact does not occur, and we do not consider extension of thrombosis to be a serious hazard of aortography.

Among the complications of aortography described, the most important is undoubtedly anuria and renal failure (Dormandy *et al.*, 1957). At least 12 deaths from this cause are now recorded in the literature.

A recent survey (McAfee, 1957) suggests that there is a greater danger of renal complications in cases of aortic thrombosis than in others. This is because, with the blockage of the aorta, a much higher proportion of the contrast medium enters the kidneys than would normally do so. As a result there is a greater risk of a toxic dose being delivered to the kidneys. We are also aware of at least one unpublished instance at another London hospital where aortography in a case of aortic thrombosis was followed by renal failure and death. In our own large series of cases of aortic thrombosis in which aortography was performed we have not encountered any such complication. However, there is no question that extra caution as regards the dose injected is needed in these cases, and we would stress that adequate diagnostic results can be obtained using only 15–20 ml. of 60% "urografin." With doses such as this we do not think there is any real danger of renal damage.

Paraplegia following aortography has also been recorded several times, and is discussed in detail by Hare (1957). Other complications which have been noted are pneumothorax, perforation of the bowel, peritoneal injection, and haemothorax.

All these recorded complications serve to emphasize that aortography is not a procedure to be lightly undertaken by the occasional dabbler. The incidence of complications in aortography does, in fact, appear to be directly related to the experience of the operators.

At St. Mary's Hospital, where large numbers of aortograms are performed, the procedure is normally carried out by the radiologists in the x-ray department under strictly standardized and controlled conditions. Morbidity and complications have been thus reduced to a minimum. In the past four years some 500 aortograms have been undertaken, mainly on patients with arterial disease. The only significant complication encountered has been the single case of temporary anuria mentioned above (Dormandy *et al.*, 1957).

Thus, provided adequate precautions are taken and the problem of dosage is fully understood, aortography in suspected cases of aortic thrombosis is fully justified. This is emphasized by the confidence with which these cases are entrusted to us for investigation by our colleagues.

Discussion

Aetiology

Mitral Stenosis.—Six of the present patients had mitral stenosis and auricular fibrillation. In these there was evidence of multiple emboli. They will be discussed in detail elsewhere, and we shall bring forward evidence for believing that in these cases the aortic thrombosis was a result of more distal emboli causing retrograde clotting into the aorta (Starer and Sutton, in preparation). Four of these six cases were in women—that is, half the female cases in the series.

Trauma.—In three cases described above aortic thrombosis may have been related to trauma, but the relationship is uncertain.

Atheroma.—There would seem to be little doubt that in most of our cases thrombosis was superimposed on underlying atheroma. Previous pathological findings and our own arteriographic experience show that this is the case. A study of our arteriographic material leads us to believe that thrombosis usually starts in the common iliac arteries. This appears to be the general opinion also of other writers on the subject—for instance, Leriche (1951). The reason for this is not known. Straus *et al.* (1946) consider that thrombosis is facilitated by heavy atheroma in that region caused by local eddy currents. Like Leriche, we have, however, seen thrombosis starting at a higher level, and we have even observed occasional patients with localized atheromatous stenosis of the aorta and apparently healthy iliac vessels at aortography. This can give rise to a most puzzling clinical and radiological picture, particularly in a young patient. It may be extremely difficult to differentiate from the very rare syndrome of coarctation of the

abdominal aorta. Despite these exceptional cases, it is clear that, as already stated, most cases result from atheroma affecting the common iliacs and terminal aorta.

Case 6.—A 55-year-old gravedigger. He had been complaining of intermittent claudication for ten years, beginning on the right but finally involving both calves and thighs. More recently he had also suffered from pain at rest, but there had been no impotence or muscle wasting. A bilateral lumbar sympathectomy (L 1–5) produced no change. An aortogram in January, 1956, showed partial occlusion of the aorta $1\frac{1}{2}$ in. (3.75 cm.) above the bifurcation and complete occlusion of the right internal iliac artery (Special Plate, Fig. 8). Between January and December, 1956, there appeared to have been no change in his condition. But, since his disablement was severe, disobliteration of the aorta and iliacs was carried out (Mr. Eastcott). At operation complete occlusion of the aorta was found up to the level of the renal arteries. (It may be noted that the level of puncture was considerably above the level of the occlusion.) A successful disobliteration was achieved.

We have not studied blood cholesterol levels in our patients. Theis (1952) reported that 62% of his patients had a cholesterol level above 300 mg. per 100 ml. of blood. Of greater significance may be the studies on serum lipoprotein concentrations carried out by de Wolfe and his colleagues (1954, 1956). In a study of 84 non-diabetic subjects with peripheral aortic atherosclerosis they found in about half the cases an increase in cholesterol and low-density lipoproteins with a decrease in high-density lipoprotein content as compared with normal subjects. The increase was greater in patients with aortic than in those with femoral occlusion.

Not all writers have accepted the atheromatous origin of aortic thrombosis. Leriche's view seems to have been that it was probably the result of a peculiar arteritis. The arguments in favour of this are summed up by Shapiro (1952), who mentions the following points: (1) thrombosis is rare, whereas atheroma is common; (2) thrombosis begins in the iliac arteries, whereas atheroma is maximal in the aorta; (3) age—these patients tend to be middle-aged and have not reached the period of most advanced degenerative change; (4) the presence of a definite arteritis in some cases; and (5) the presence of an intensive periarteritis. Similar views were expressed in the discussion following the paper of DeBakey *et al.* (1954). It should, however, be remembered that marked cellular infiltration and intensive periarteritis are found in relation to atheromatous occlusion of vessels. Several case reports exist of aortic thrombosis due to alleged Buerger's disease. Bohle (1950), for instance, reported two such cases, neither of which showed any other manifestations of thromboangiitis obliterans. His interpretation of the histological appearances must be treated with reserve, since the appearances could equally well be due to the cellular reaction often seen in relation to a thrombosed atheromatous vessel. We have, however, seen one patient with a history compatible with Buerger's disease who developed aortic thrombosis.

Case 7.—A male aged 38, diagnosed as a case of Buerger's disease because of arterial insufficiency to the legs with repeated attacks of phlebitis. The femoral arteries appear to have been normal, and his blood pressure 125/60 mm. Hg. A lumbar sympathectomy was carried out without benefit; following this operation femoral arterial thrombosis was diagnosed. He developed increasingly severe intermittent claudication and impotence and began to complain of headaches. A diagnosis of aortic thrombosis was made, and his blood pressure was found to have risen to 180/110 mm. Aortography demonstrated aortic occlusion at the level of the renal arteries, and the left renal artery was narrowed at its origin. Resection of the thrombosed segment of aorta was carried out and a polyvinyl graft inserted; at the same time a left nephrectomy was performed (Professor Rob). There was marked alleviation of symptoms and the blood pressure fell to normal levels. Pathological examination of the excised aorta suggested atheroma as the underlying abnormality. Here also Buerger's disease could not definitely be implicated. Histologically, the kidney showed changes of chronic pyelonephritis, but no arterial changes as are seen in hypertension.

Other Aetiological Factors.—A large number of other possible aetiological factors have been mentioned. Gross

and Philips (1940) found one case complicating dissecting aneurysm. Pinkerton (1948) reported an 81-year-old man in whom the aorta was pressed on by extensive spinal osteophytes and these may have been responsible for aortic thrombosis; the author recalls the subclavian and brachial thrombosis resulting from pressure by a cervical rib or a crutch. We have observed similar cases associated with large osteophytes, but doubt whether it was a significant aetiological factor. Horwitz (1948) observed thrombosis of the aorta in a 58-year-old woman with Addison's disease occurring shortly after the performance of a Cutler test. Syphilitic aortitis has been recorded on a number of occasions as an aetiological factor (Greenfield, 1943; Beaconsfield and Kunlin, 1953), but was not found in any of our cases. Rothstein (1935) gives a lengthy list of conditions that have been found together with aortic occlusion; their relationship is not always clear. Dexter *et al.* (1957) described a patient with aortic thrombosis who also suffered from Marfan's syndrome. Finally, Harrison *et al.* (1955) report a remarkable and unique family history: aortic thrombosis occurred in two brothers and one cousin, while four other members of the family had severe arterial insufficiency of the legs.

Unusual associated conditions found in our series were fused kidneys in two cases and poliomyelitis in one case. Of the two patients with fused kidneys, one had a horseshoe kidney (Special Plate, Figs. 2 and 7), the other a crossed fused ectopia. The pelves of the fused kidneys in this latter case contained staghorn calculi, and the patient also suffered from cystinuria and retinitis pigmentosa.

Hypertension

The relationship between aortic occlusion and hypertension is of considerable interest and merits further discussion. Raised blood pressure is a common finding in the general population, and therefore a number of cases of aortic thrombosis may be expected to have incidental hypertension. In so far as a raised blood pressure predisposes to atheroma it may become one aetiological factor in aortic thrombosis. We have already remarked on the relatively small number of patients with aortic thrombosis who also have hypertension.

A second possibility is that occlusion of the aorta below the renal arteries in itself is responsible for a rise in blood pressure. This possibility seems to have attracted several German authors (see Bohle, 1950; Krautwald *et al.*, 1955). Brotcher (quoted by Horwitz, 1948) obtained small rises in blood pressure in dogs by clamping the aorta below the origin of the renal arteries. Apart from this, however, there appears to be little experimental or clinical evidence to support this view of hypertension in aortic occlusion. The absence of hypertension in so many of these patients, on the contrary, is definite evidence against it.

The third possibility, and the one of the greatest interest, is that hypertension of renal origin may be caused by narrowing of a renal artery from extension of thrombus. Goodman (1952) has given an excellent account of the occurrence of the Goldblatt kidney in man. The kidney supplied by the occluded artery remains histologically normal, being protected by the arterial obstruction—a state of affairs similar to that found in experimental hypertension in rats (compare Case 7). Such hypertension, if treated early, should be reversible by nephrectomy.

We have observed renal arterial stenosis on five occasions. In one of these patients the right renal artery and right kidney were abnormally small, but appeared to function normally. This patient died following an attempted grafting operation, and at necropsy the kidney was considered to be congenitally hypoplastic. Of the five patients mentioned above, two had normal blood pressure. Similar cases are reported in the literature, and narrowing of the renal artery is obviously not always associated with hypertension. One patient was mildly hypertensive, and the relationship between raised blood pressure and renal arterial involvement is not clear. One patient has already been described (Case 7); the history of another is of some interest.

Case 8.—A 38-year-old woman of gipsy origin. Four and a half years before admission to St. Mary's Hospital she awoke one night complaining of tingling in both legs. This seems to have been only transient, and she was subsequently well enough to work on a farm. Three years later she suddenly developed pulmonary oedema, and she was found to be hypertensive. Cardiac failure persisted, and a year later her hypertension had become frankly malignant. Her blood pressure was 220/120 mm. Hg, there were retinal haemorrhages with blurring of the optic disks, and proteinuria was present. She was admitted to the local hospital, where a diagnosis of phaeochromocytoma was made and an operation for its removal performed. No such tumour could be found, but it was noticed that the aorta was not pulsating. The diagnosis was changed to one of aortic thrombosis and the patient transferred to St. Mary's Hospital. Here an aortogram showed aortic occlusion at the level of the renal arteries: the right renal artery appeared to be blocked (Special Plate, Fig. 5). Renal catheter studies, however, showed some function of the right kidney. At a later date the abdominal aorta was excised (Professor Rob and Mr. Eastcott) and replaced by a freeze-dried graft. On the day following operation her blood pressure was 280/170 mm. It was lowered to 230/110 mm. by hexamethonium. She was discharged at her own insistence before further blood-pressure or renal-function studies could be carried out, and it has not been possible to follow her up further.

Reports of similar cases are now sufficiently frequent to make it clear that aortic thrombosis may be a factor in the inception of malignant hypertension. The physician dealing with such a case must consider this possibility, and aortography would seem to have a limited place in the investigation of these patients. Martorell (1954) described two deaths from malignant hypertension in patients with aortic thrombosis and renal arterial involvement, and two others cured by operation. Roberts (1955) operated successfully on a similar case. Krautwald *et al.* (1955) studied one such case in which both renal arteries were involved. They found normal inulin clearance, but reduced renal plasma flow. The filtration fraction was increased. On the basis of comparison with "ordinary" hypertensives the authors conclude that renal blood flow in this case was diminished beyond that otherwise seen. Price and Wagner (1947) reported a case of malignant hypertension in which there was bilateral occlusion of polar arteries. Other cases have been reported by Frelick and Shellenberger (1954) and by Barnett *et al.* (1952).

The combination of hypertension and absent femoral pulses found in these cases clearly require differentiation from coarctation of the aorta.

Impotence

The nature of the inability appears to be failure to maintain an erection. This was so described by Leriche in his original publication. It is presumably due to failure of adequate blood supply to the corpora cavernosa through the internal pudendal artery. In those patients who develop an adequate collateral supply to that artery impotence does not develop. Presumably it is the development of such a collateral circulation following sympathectomy or grafting which may bring about the disappearance of this symptom; or the natural improvement in the collateral circulation with time, as appears to have happened in one of our cases. With regard to lumbar sympathectomy, it should be remembered that bilateral section below L2 abolishes ejaculation, though erection and sensation may remain normal.

An alternative mechanism is discussed by Straus *et al.* (1946). They postulate that weakness of lower limbs, especially after exercise, may be due to Dejerine's syndrome or "intermittent claudication of the cord," and that this may also result in impotence. A lesion involving the sacral nerves below S1 abolishes erection and produces relative anaesthesia of the penis (impotence may be found in cauda equina tumours, presumably a somewhat comparable situation). Burt *et al.* (1952) have, however, drawn attention to the fact that disturbances of micturition are not found in aortic thrombosis, and would be expected in cord lesions at the level demanded to cause impotence.

Johnson (1954) reported testicular atrophy found at necropsy in one man. We have seen one patient who,

clinically, appeared to have somewhat atrophic testes; histological examination showed them to be normal, however. We have not had an opportunity to estimate the serum gonadotrophin level in any of our cases; this may be not without interest. In fact the testicular artery can frequently be seen on aortograms and the collateral supply to the testes appears good (Neuhof and Mencher, 1940).

Differential Diagnosis

It is our experience that the diagnosis is often—perhaps usually—missed by the doctor referring the case to hospital, and it is part of the purpose of this paper to make the condition better known. If the diagnosis of aortic thrombosis is kept in mind, suspicion should be easily aroused, and a fair degree of certainty in diagnosis readily attained by physical examination. The diagnosis can then be proved unequivocally by aortography.

A number of other conditions may be confused with thrombosis of the aorta. Bilateral iliac arterial thrombosis cannot be differentiated from it without an aortogram. De Wolfe *et al.* (1954) and Bonney (1956) described patients complaining of intermittent claudication of the hip who were referred for orthopaedic treatment with the mistaken diagnosis of hip-joint disease. We have seen one patient diagnosed and treated for some time as a case of sciatica. Progressive muscular wasting may suggest a primarily neurological lesion, and the finding of impotence, peroneal pain, fasciculation, and sensory loss apparently confirm it. The patient whose main complaint is impotence may well be referred to a psychiatrist for lengthy and fruitless treatment. Vigorous aortic pulsation above the block may suggest the presence of an aortic aneurysm—an error of less importance.

When hypertension is present a number of considerations arise. We have described a case in which a mistaken diagnosis of pheochromocytoma was made. It has also been suggested that in patients with hypertension the absent femoral pulses may lead to an erroneous diagnosis of coarctation. The presence of intermittent claudication and other features noted above should prevent such an error. Also, as previously mentioned, rib-notching, even in the lower ribs, does not seem to occur in aortic thrombosis.

We believe that the diagnosis of aortic thrombosis is sometimes considered and dismissed because of the absence of gangrene. It is therefore worth emphasizing that nutritional changes in the lower limbs are the exception rather than the rule, and that a diagnosis of aortic thrombosis is perfectly compatible with a normal nutritional state of the feet.

Prognosis

The first report of aortic thrombosis appears to have been that by Severinus in 1632, made as a result of post-mortem examination (for a history of aortic thrombosis see Burt *et al.*, 1952). The scanty reports following this were similarly based on post-mortem examinations, and in 1935 Rothstein was able to collect 123 cases from the literature with an alleged 91% mortality. It should be remembered that before the introduction of aortography and the development of major vascular surgery the diagnosis in the living subject had to remain speculative. As a result the impression of aortic thrombosis as an inexorably fatal disease arose. It was perhaps only to be expected that a block of the major vessel should give rise to rapid gangrene and death. In 1942 Leriche published his experience with such cases, and pointed out that the outlook for these patients was by no means hopeless. From his article it is evident that he had the greatest difficulty in persuading his colleagues in France of the correctness of his observations. While, in general, it is now agreed that his description of the clinical picture in aortic thrombosis was substantially correct, the earlier and gloomier view still lurks in the background. That disablement is severe is without doubt; we also know that some of these patients will die from renal involvement or gangrene. Nevertheless, from our review of the condition it is clear that the danger to life has been grossly exaggerated in the past, and that most patients

with aortic thrombosis can continue to lead useful and moderately active lives for many years. Indeed, there is some evidence that the patient is more likely to succumb to associated coronary vascular disease or other illness than to the aortic thrombosis.

Recent advances in arterial surgery have made operative treatment of aortic thrombosis a practical procedure, and many such operations have now been performed at St. Mary's Hospital. The assessment of the long-term results in vascular surgery is not yet possible, and is in any case outside our province. It is clear, however, that the dangers of major surgery in these patients, who are often very poor operative risks, must be carefully weighed against the prognosis of the untreated disease. This, as we have pointed out, is less gloomy than has been generally believed.

Summary

A clinical and radiological study is presented of patients with aortic thrombosis examined by aortography at St. Mary's Hospital in the four years prior to June, 1957, and the previous literature on the subject is reviewed. During the period under review 32 patients with this condition were examined. (A further eight cases have been seen since this paper was written, bringing our total to 40 cases in five years.)

The clinical and radiological findings are discussed in detail.

The diagnosis should be readily suspected on clinical grounds, but differentiation from bilateral iliac thrombosis is not possible without aortography. The latter procedure has been carried out in all our cases without complication. It is considered that the recorded hazards of aortography have been mainly due to faulty techniques.

Aortic thrombosis is nearly always due to atheroma of the lower abdominal aorta and the iliac vessels. Buerger's disease and other forms of "aortitis" which have been cited as responsible in previous papers are considered to be of little importance. There was a possible relationship to trauma in three of our cases. In six cases aortic thrombosis was associated with mitral stenosis. In these cases retrograde thrombosis was considered to have followed peripheral emboli.

The problem of renal arterial involvement is discussed in detail, together with its possible relationship to hypertension.

It is concluded that aortic thrombosis is neither as rare nor, in most cases, as dangerous to life as was previously thought.

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PATHOLOGICAL STUDY OF THE LIVER IN KWASHIORKOR*

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[WITH SPECIAL PLATE]

In Calcutta a syndrome is frequently seen in children which presents a picture of severe malnutrition characterized by diarrhoea, wasting, oedema of varying degree without gross albuminuria, glossitis, anaemia, cutaneous changes with black pigmentation, enlargement of the liver, hypoproteinaemia, eye lesions of vitamin-A deficiency, failure of growth, sometimes mental changes, and anomalies of hair growth (Fig. A). A similar syndrome has also been observed in parts of tropical Africa, and was described under the name "kwashiorkor" by Williams (1933, 1935, 1940). It has also been called infantile pellagra (Trowell, 1937, 1940, 1941), malignant malnutrition (Trowell, 1944; Trowell and Muwazi, 1945a), ariboflavinosis (Hughes, 1946), and fatty liver disease of infants (Waterlow, 1948).

In pathological studies of this syndrome in children in Africa, Williams (1935) and Gillman and Gillman (1944) found only fatty changes of varying degree in the liver parenchyma and no necrosis or haemorrhage. Waterlow (1948) considered fatty change in the liver to be a constant accompaniment of the disease; he also observed increase of fibrous tissue from the portal tracts with a tendency to progress to Laennec's cirrhosis.

In serial biopsies in 120 cases of pellagra, all but two in patients over 10 years of age, Gillman and Gillman (1945) found fatty changes in the liver and cirrhosis. In the adults the cirrhosis was preceded by a deposit of haemosiderin in the portal tracts in fatty livers, giving a picture of haemosiderosis or cytosiderosis. Liver

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puncture showed that fatty liver is as constant a feature of the syndrome as the rash or oedema, and that it develops early before other known complications such as pneumonia, tuberculosis, or gastro-enteritis. Of the 120 cases investigated, 15 (12.5%) had frankly pigmentary cirrhosis, while in 20% the liver was pre-cirrhotic and about 30% showed signs of serious hepatic damage amounting to cirrhosis. In 1% of cases the liver had no recognizable damage. In none of the livers was

FIG. A.—Typical case of kwashiorkor in Hindu child aged 2 years 2 months. Oedema and skin pigmentation are well shown.

there necrosis or haemorrhage. In two cases in children not included in the above series there was irrefutable evidence of non-pigmentary cirrhosis.

Achar (1950), who studied 78 cases of severe malnutrition in Madras, on histological examination of 12 cases found an extreme degree of fatty infiltration; in two of these cases there was commencing periportal fibrosis, and in one case diffuse fibrosis around the lobules.

The present paper reports the histopathological findings in the liver in 34 cases of kwashiorkor studied post mortem with a view to elucidating the evolution of cirrhosis from hepatic damage.

Method of Investigation

The gross specimens of liver in all cases were fixed in formalin and kept in No. III Kaiserling solution (glycerin and water). After necropsy small pieces of liver tissue 3–5 mm. thick were immersed in Zenker's fluid for 24 hours, washed in running water for 24 hours, and finally preserved in 80% alcohol in tissue jars. Blocks were made by paraffin embedding from different areas of liver in both lobes and studied. Staining methods were as follows: (1) for routine staining—paraffin sections stained by Weigert's iron-haematoxylin and eosin; (2) for connective tissue—Mallory's aniline blue method; (3) for fat—frozen sections stained with Sudan III and counterstained with haematoxylin; (4) for reticulin—Foot and Menard's method as modified by

F. STARER AND D. SUTTON: AORTIC THROMBOSIS

FIG. 1.—Aortic thrombosis just below level of renal arteries. Note small subcostal arteries, atherosclerotic stenosis of left renal artery, and inferior mesenteric artery supplied by left colic branch of superior mesenteric artery.

FIG. 2.—Aortic thrombosis below inferior mesenteric artery just above bifurcation of aorta.

FIG. 3.—Aortic thrombosis at origin of inferior mesenteric artery. (Note tapering of aorta.)

FIG. 4.—Aortic thrombosis just below renal arteries.

FIG. 5.—Aortic thrombosis at level of renal arteries with involvement of one renal artery. Note that inferior mesenteric artery is supplied by left colic branch of superior mesenteric. There is a left nephrogram but no right nephrogram.

FIG. 6.—Aortic thrombosis above level of inferior mesenteric artery. As in Fig. 1, latter is supplied by hypertrophied left colic branch of superior mesenteric artery. Lower limit of block is in common iliac artery on right, but in femoral artery on left. Left internal iliac artery, however, is filled from collaterals.

FIG. 7.—Lower limits of aortic block shown on serial films. This is same case as Fig. 2, and demonstrates in nephrogram phase the associated horseshoe kidney.

FIG. 8.—Aortic stenosis due to atheroma just above bifurcation. Operative clips are from a previous sympathectomy. At operation condition had progressed to complete thrombosis of aorta.