CURRENT SURVEY

Thoracic aneurysms

L. L. BROMLEY

M.Chir., F.R.C.S.

Surgeon in Charge of the Thoracic Department, St Mary's Hospital, London, W.2

IN THE past 15 years it has become possible to treat most varieties of thoracic aneurysms by surgical excision. This is due to technical advances especially in anaesthesia, blood transfusion, the use of homografts and more recently the development of prosthetic grafts of synthetic fibre (Wesolowski, *et al.*, 1966). Hypothermia, temporary arterial shunts, partial left heart bypass (left atrio-femoral), and total cardiopulmonary bypass have allowed maintenance of viability of the brain, heart, kidneys and spinal cord during aneurysmal resection and repair. Nevertheless there remain very considerable difficulties in this type of surgery; the operative mortality and morbidity are high and firm indications should exist before advising surgical treatment. The natural history of most thoracic aneurysms is of steady expansion leading to rupture (Holman, 1955), nevertheless enlargement of some aneurysms is remarkably slow and in many cases conservative management is wise whilst the behaviour of the aneurysm is observed.

Left ventricular aneurysm

This results from a full thickness myocardial infarction most commonly of the anterior descending branch of the left coronary artery, the aneurysm being anterior and apical. As healing of the infarct proceeds the young scar tissue is stretched by the force of the ventricular impulse and a bulging pouch develops with paradoxical pulsation. The pericardium is adherent, rough clot lines the internal aspect and the neck is wide and not particularly well defined. It seems likely that hypertension assists the development of the aneurysm but there is no convincing evidence that an aneurysm is likely to arise in a patient who is active very early after a cardiac infarction. In many cases the wall of the aneurysm gradually calcifies and systemic emboli do not occur. Such patients usually have no symptoms and expansion of the sac is unlikely.

Other aneurysms; however, increase in size; the effective stroke output of the left ventricle is seriously reduced as a great deal of blood is washed in and out of the sac in systole and diastole. Such patients may be greatly disabled. Surgical treatment may be advised if there is increasing dyspnoea on exertion, recurrent attacks of pulmonary oedema,

intractable angina, systemic emboli or steady enlargement of the aneurysm both clinically and radiologically. Using cardiopulmonary bypass the aneurysm is excised and the left ventricular wall repaired (Cooley *et al.*, 1959; Telling & Wooler, 1961; Lillehei *et al.*, 1962). The surgical mortality is low but as the patient still has coronary arterial disease the prognosis is generally poor as further infarctions may occur (Figs. 1 and 2).

FIG. 1. Chest radiograph showing large left ventricular aneurysm 6 months after infarction.

Sinus of Valsalva aneurysm

This is usually congenital, there being a weakness of the medial coat of the aortic wall at its junction with the annulus fibrosus (Edwards & Burchell, 1957). A sock-shaped sac develops most commonly below the right coronary ostium in the sinus, less commonly in the noncoronary sinus, and this

FIG. 2. Left ventricular aneurysm at operation just before excision.

projects into the right atrium or right ventricle. The diagnosis is not usually made before rupture which results in an aortoatrial or aortoventricular fistula with a collapsing pulse, a continuous murmur at the left sternal edge and cardiac enlargement. The chest radiograph shows overfilling of the pulmonary vessels. Aortography distinguishes the deformity from patent ductus arteriosus or a ventricular septal defect with aortic regurgitation and simple rupture of an aortic cusp, whilst right heart catheterization confirms the left to right shunt. Surgical treatment is indicated as the natural history is of progressive heart failure (Besterman, Goldberg & Sellors, 1963).

Using cardiopulmonary bypass the neck of the aneurysm is closed by suture, the approach being through the right ventricle, the right atrium or the ascending aorta, or a combination of these. Co-existent defects are not uncommon, a small V.S.D. or A.S.D. being sometimes found.

Syphilitic aneurysm

Generally aneurysms of the ascending aorta are fusiform, whilst saccular aneurysms are most common on the arch or descending thoracic aorta. Combinations of fusiform and saccular aneurysms are common and fusiform dilatation may extend into the proximal parts of the branches of the arch (Fig. 3). The time taken for the development of an aneurysm from infection is remarkably variable, being from 5 to 40 years. Saccular aneurysms may have a relatively narrow neck and arise from a

transverse tear in the intima and media. Syphilitic aneurysms show progressive enlargement, chemotherapy having little effect in stopping the inexorable expansion and sooner or later symptoms develop. These are generally due to pressure or rupture: compression of the trachea or superior vena cava, left recurrent nerve palsy, severe pain due to vertebral or sternal bone erosion and internal or external rupture are examples. Aortic regurgitation from stretching of the annulus of the valve may also develop. Surgery is indicated where life is threatened by mediastinal compression, imminent rupture or severe intractable pain. The history of surgery for syphilitic aneurysm is long and distinguished, a number of palliative operations being devised but it is now accepted that the most effective treatment is excision of the aneurysm with restoration of continuity (Waldhausen, 1964). This is major surgery in the truest sense of the word and is not undertaken unless bypass facilities are immediately available. Many saccular aneurysms may be excised following simple clamping of the neck and associated fusiform dilatation treated by reinforcement of the aorta by suturing a suitably tailored dacron sleeve in position (Bickford & Glennie, 1960).

In the distal arch or descending thoracic aorta left atrio-femoral bypass can aid removal and local repair or grafting, thus ensuring a continuing blood supply to the kidneys and spinal cord during the period of aortic clamping. For combined fusiform and saccular aneurysms of the ascending aorta total arch replacement may be required but is a formidable procedure entailing the use of temporary

FIG. 3. Arch aortogram showing syphilitic aneurysm of the ascending aorta in a female aged 23 years. There is fusiform dilatation extending into the proximal innominate artery and a small saccular aneurysm arising anteriorly.

shunts, cardiopulmonary bypass, perhaps with profound hypothermia in order to maintain cerebral, cardiac and renal viability whilst the aorta is excised and a graft sutured in position (Barnard & Schrire, 1963).

Atherosclerotic aneurysm

These are mostly fusiform and, though usually arising in the abdomen below the renal arteries, are not uncommon in the descending thoracic aorta. Most are symptom free and the natural history is more benign than a syphilitic aneurysm; however, sudden chest pain indicating leaking or persistent pain from vertebral erosion are indications for surgery.

Traumatic aneurysm

Closed chest injury involving rapid deceleration may result in a transverse tear of the aorta usually just beyond the isthmus (Strassman, 1947). The tear may involve the complete circumference and full thickness of the aorta with immediate exsanguination into the left pleural cavity. In other cases the adventitia remains intact but a large mediastinal false aneurysm rapidly develops. The condition can be recognized by chest pain and dyspnoea after injury with radiological signs of a widened upper mediastinal shadow and a left pleural effusion, heavily bloodstained if tapped. Within a few hours rupture of the remaining adventitia and overlying pleura occurs with exsanguination. Emergency surgery is indicated in the hours before rupture (Bromley, Hobbs & Robinson, 1965).

In other patients the tear involves intima and part of the media only, and a chronic but nevertheless steadily expanding aneurysmal sac develops. Definitive surgery can be planned: via a left thoracotomy the aorta is mobilized above and below the aneurysm and with partial left heart bypass the aneurysmal sac can be excised and the aorta repaired by direct suture or by grafting (McClenathan & Brettschneider, 1965).

Dissecting aneurysm

In recent years this variety of aneurysm has been more frequently recognized, studied and surgically treated. It is a lethal form of aortic disease affecting males aged 40–70 years usually with essential hypertension. It occurs also in association with Marfan's syndrome, coarctation of the aorta and in pregnancy. The fundamental pathology is degeneration or weakness of the media of the proximal aorta (cystic medionecrosis) with an initial dissection occurring within the media from rupture of the wall of a vasa vasorum (Gore, 1952). The inner layers of the media and the intima then tear and the full force of the aortic pressure can now attack the intimomedial split, dissection proceeding in the

outer layers of the media. The intimal split is usually single, transverse, and sharply defined, situated in the proximal 3 cm of the ascending aorta and not related to any coexistent atheromatous plaque. Another common site of origin is at the aortic isthmus. Within hours the dissection spreads proximally and distally, the extent varying but in the grossest form to the aortic root and to the iliac vessels. Spontaneous re-entry may occur in an estimated 5–10% of cases and a chronic state can be achieved, the aorta possessing two lumens. In the majority of cases death occurs from intrapericardial rupture, rupture into the pleura, mediastinum, peritoneum, or into a viscus; or it can be due to cerebral or renal ischaemia.

In a series of 505 patients reviewed by Hirst, Johns & Kime (1958) the survival time was indicated in 425. Eight per cent died within 6 hr, 21% within 24 hr, 49% within 4 days, 74% within 2 weeks and 91% within 6 months. The clinical picture is of an abrupt onset with anterior chest pain, severe and sometimes described as of a tearing quality. As dissection proceeds the pain may move to the abdomen and even to hips and legs. The patient presents a shocked appearance but the blood pressure is normal or raised, pulse variations in the carotids and limb vessels are common and an aortic diastolic murmur may be heard. Disorders of consciousness occur and paraplegia may develop. The chest radiographs show a progressively widening thoracic aortic shadow and a left pleural effusion. The electrocardiograph shows left ventricular hypertrophy or strain and there may be changes suggesting coronary insufficiency. The main differential diagnosis is from cardiac infarction and the points of importance are the characteristics of the pain, the normal or elevated blood pressure, the variable pulses found on repeated examination and the radiological features.

In the past 10 years surgical treatment has been more frequently attempted, initially by making a re-entry ostium in the lower thoracic or abdominal aorta (Rob & Kenyon, 1960), and more recently by direct surgery. De Bakey *et al.* (1965) have classified aneurysms into three types. Type 1 where the dissection starts in the ascending aorta and extends distally along the rest of the aorta. Early surgical treatment was undertaken, a re-entry procedure being done in six patients with four surviving, but in fourteen, using cardiopulmonary bypass, the ascending aorta was transected, the false lumen obliterated by suture and the ascending aorta then reanastomosed. Ten patients survived the operation. In Type 2 the dissection is limited to the ascending aorta. Seventeen patients were treated by excision and graft replacement with twelve surviving. The largest group was Type 3 where the dissecting had begun at the isthmus and

progressed mainly distally. Using left atriofemoral by pass the aneurysm was excised and grafted in 139 patients (there were three patients also having a re-entry procedure) with 123 surviving. This type of dissection was operated upon when a chronic state had been reached as opposed to Types 1 and 2 where surgery was undertaken at an earlier stage. Wheat *et al.* (1965) consider that as the chief cause of death is not due to the initial tear of the intima but to the subsequent dissection an effort should be made to limit those forces promoting dissection, thus reducing it. Surgery, if required, can then be undertaken when the aneurysm is chronic. The main force promoting dissection is the cardiac impulse, the acceleration of left ventricular output, a factor of the rate of ventricular fibre shortening. By the use of trimethapham in the first 48 hr after admission and later by guanethidine alone the systemic blood pressure is held around 100 mmHg systolic. Aortography and full assessment of the need for surgery can be made some weeks later. Indications for surgery are progressive aortic regurgitation with impending left ventricular failure, intractable pain due to the aneurysm and steady increase in the size of the aneurysm. Six patients were treated conservatively, one patient later underwent successful elective resection of the ascending aorta; the other five were living 9–15 months on continued medical treatment, without symptoms and no apparent change in the size of the aneurysm. There is no doubt that the technical difficulties are very much greater when surgery is done in the acute stage.

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