There were serious complications in two patients. In one, a woman of 72, the upper oesophagus suffered a small perforation which led to mediastinitis, the patient dying shortly afterwards for reasons which were not entirely clear. In the other patient severe haemorrhage occurred. There was haemorrhage in nine patients (2.1%), three of whom had an atrophic mucosa. Grounds for caution in performing gastric biopsy are suggested.

The Australian type of biopsy instrument is reasonably safe provided there is some selection of patients and the patient is observed for some hours after its use. Our results with it so far indicate that it is useful in the diagnosis of some types of gastric lesion. Possible clinical uses to which the histological findings may be put are mentioned.

The Australian biopsy tube is made by D.H.A. (Victoria) Pty., Ltd., 21, Alfred Place, Melbourne, C.I, Australia (formerly Messrs. Felton, Grimwade and Duerdins Pty., Ltd., of the same address). It can be imported through Drug Houses of Australia Export Ltd., 2/3, Norfolk Street, Strand, London, W.C.2.

The main modifications to the Australian instrument were worked out with the Genito-Urinary Manufacturing Co. They and Messrs. T. H. Spicer and Son have altered the original instruments for us. The hand pump was obtained from Messrs. A. Gallenkamp and Co., Ltd.

It is a pleasure to record our thanks to the following people who have helped us: to Dr. Avery Jones for his interest, and the loan of an instrument at the start of the work; to Professor J. S. Young for his help and advice; to Dr. A. C. Counsell for so generously providing facilities in his laboratory for the preparation and staining of the biopsy specimens; to Mr. W. J. Ferguson for allowing us to include details of Case 6; to Drs. T. H. C. Lewis, W. B. Hennessy, and Felicity Edwards, who have obtained many of the specimens; to Mr. C. N. King, of the West Middlesex Hospital Pharmaceutical Department, for much help with the instruments; to Sisters C. K. Chambers, I. E. S. Seaney, and H. Hendey, and particularly Staff Nurse J. H. MacDonald, for help in the care of the patients; to Mr. R. Drummond for the photomicrographs; to Mr. D. A. Vinton for Fig. 7; and to Miss Susan Robinson for the drawing.

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In his annual report for the year 1954 Dr. I. GORDON, medical officer of health for the borough of Ilford, has these remarks to make about voluntary work in the Health Service: "Many people have complained of the division of the National Health Service into three parts—hospitals, general practitioners, and local authorities—each of which is concerned with the welfare of old people and often working independently of each other. There is actually, of course, a fourth part, the voluntary services. We are fortunate in this town in having a committee run by a section of the fourth part, the Ilford Social Service Association, which concerns itself with the aged. . . . I have heard the Minister of Health state that it was often said that the strange thing about the National Health Service is that, while theoretically it should not work, nevertheless in practice it does. May I suggest here that the reason why it does work is that local people, who know local needs, and feel strongly on the matter, join together to patch up its defects, by arrangements that do not appear in the National Health Service Act, 1946?"

HEALED DISSECTING ANEURYSM OF THE AORTA

BY

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Dissecting aneurysms of the aorta usually have a short clinical course. In Shennan's (1934) series of 143 recent dissecting aneurysms with clinical data 84 (58%) of the patients died within the first 24 hours and 38 (26%) within the first week. This short duration is also emphasized by the fact that dissecting aneurysms are commoner in deaths reported to coroners than in routine hospital necropsies (Mote and Carr, 1942). Crowell (1921) estimated that 20% of patients survived the initial episode, and a smaller number recover completely. The following two cases are reported in order to emphasize that there is a characteristic clinical and pathological picture in healed cases of dissecting aneurysm of the aorta. Syphilitic aneurysms are now uncommon, and it is believed that healed dissecting aneurysms assume a relatively more important place in the incidence of chronic aortic aneurysm.

Case 1

A Spanish dental mechanic aged 49 had an attack of severe pain in his chest and back in 1949; this was associated with hoarseness of voice. After 10 days in bed he made a good recovery and was subsequently perfectly well except for occasional pains in the left shoulder and left arm; these were not related to exercise.

In May, 1953, he had another similar sudden attack of thoracic pain, mainly on the left side and radiating to the back and down to the left groin. He was admitted at once to hospital, where he was found to be cold and sweating; pulse 84, regular; B.P. 90/30. The radial and femoral pulses were equal; the heart was slightly enlarged and there were no abnormal heart sounds. A few rales were heard at both bases. The epigastrium was tender and the abdominal wall rigid. The remainder of the physical examination was normal. The E.C.G. showed left ventricular preponderance only. A small left pleural effusion was seen in the chest x-ray film and the left mediastinal shadow was enlarged. The patient improved with sedation, and the pain disappeared; the blood pressure rose to 130/70. After eight hours a large pleural effusion was detected; this was confirmed by a second chest x-ray film, in which the mediastinal shadow was found to be posterior, projecting anteriorly. Twelve hours later the pain returned, the blood pressure fell, and the patient was more shocked. Pure blood was aspirated from the left pleural cavity, and the diagnosis of ruptured dissecting aneurysm of the aorta was made. There was no further improvement, and the patient died four days after admission.

At necropsy 2 litres of blood was found in the left pleural cavity. This had originated from a ruptured dissecting aneurysm of the aorta, which involved the descending thoracic aorta and the upper part of the abdominal aorta.

FIG. 1.—Diagram of circulation through healed dissecting aneurysm (Case 1) before terminal rupture.

This dissecting aneurysm communicated with the lumen of the aorta by two smooth rounded apertures—one at the commencement of the descending aorta, the other 1 cm. above the coeliac axis (Fig. 1). The sac was anterior to the aorta, and on opening the aneurysm it was found to be lined by smooth endothelium on which there were a little laminated blood clot and some flecks of fatty change resembling atheroma with early calcification. The aneurysm was confined to the aorta, and no branches were dissected. The true abdominal aorta was grossly atheromatous with ulceration, but there was only slight atheroma of the thoracic aorta. Recent rupture of the dissecting aneurysm had occurred into the left pleural cavity just above the diaphragm and also into the retroperitoneal tissues on the left side. The heart was enlarged (580 g.) by left ventricular hypertrophy; the left lung was collapsed by the haemothorax, and was adherent to the dissecting aneurysm by dense adhesions. It was clear that the dissecting aneurysm was several years old, and corresponded with the clinical history of pain in 1949. The proximal primary tear and the distal re-entrant aperture were also old, and blood was shown to have circulated through the sac by the presence of ante-mortem blood clot of varying ages.

Histological examination showed that there were smooth muscle and elastic fibres between the aneurysmal sac and the lumen of the aorta; hence the aneurysm was in the medial coat, but a false fibroid intimal coat was found in the sac, in which fatty change resembling early atheroma was present. Mucoid degeneration without cyst formation was found in the medial coat.

Case 2

A 66-year-old retired clerk collapsed in the street, vomited, and lost consciousness. A year earlier he had had a stroke, from which he had completely recovered. For 18 months he had been treated for hypertension by repeated venesections. There was no history of any thoracic pain.

On arrival at hospital he was in deep coma. The heart was moderately enlarged and the B.P. 210/130. Apart from an accentuated second sound at the aortic area the heart sounds were normal and the peripheral pulses equal. Moist rales were heard at both lung bases. The eyes were fixed and the pupils small and equal in size. There were signs of a right-sided paralysis. The clinical course was progressively downhill, and the patient died on the fifth day.

The necropsy showed that there was a left cerebral haemorrhage with a large haematoma in the left lateral ventricle. Anterior to the recent haemorrhage an old pigmented scar was found, presumably representing the cause of the stroke one year before death. The cerebral arteries were grossly atheromatous and there was generalized arteriosclerosis. The whole length of the descending thoracic and the abdominal aorta was converted into a patent double tube by a healed dissecting aneurysm

FIG. 2.—Diagram of circulation through healed dissecting aneurysm (Case 2).

which communicated with the aorta by two apertures and with both common iliac arteries (Fig. 2). These inter-communications were all old, rounded, and covered with smooth endothelium. The lining of the aneurysmal sac was also smooth and shiny, and in the proximal part there was some old laminated ante-mortem blood clot. In addition there were some fatty flecks of early atheroma. No major branches of the aorta had been dissected, and there was no evidence of leakage or rupture of the aneurysm.

The aneurysmal sac in this case was approximately the same diameter as that of the aorta itself, and the sac

appeared to have been carrying an equal quantity of blood. It was more rigid and appeared older than that of Case 1, and, although there was no history by which this aneurysm can be dated, it was estimated that the primary dissection occurred about five years before death.

Microscopy confirmed the presence of long-standing processes, which included organizing thrombosis and the production of a false fibrous intimal coat in the sac of the dissecting aneurysm. Mucoid degeneration was found in the aorta, but there was no evidence of cystic change.

Comment

The establishment of a circulation within a dissecting aneurysm with a distal re-entrant was described by Peacock (1863) as the "imperfect natural cure." The perfect cure is effected by fibrous obliteration of the sac, but this occurs very rarely with sheath-like aneurysms (Shennan, 1932). The majority of healed dissecting aneurysms described in the literature fall into a surprisingly uniform pattern; approximately 50 of the cases reported by Shennan (1934), Peery (1936), Glendy *et al.* (1937), Weiss *et al.* (1940), Graybiel and Sprague (1941), and Mote and Carr (1942) are "double-barrelled" aortas resembling the two cases described above, and most of these extend from the distal part of the arch of the aorta to the bifurcation. These aneurysms show clear evidence of patency, and their sacs are lined with endothelium containing varying degrees of atheromatous change.

There are two anatomical factors that encourage survival with the formation of this characteristic type of healed aneurysm. The first is the situation of the dissection. The commonest cause of death is haemorrhage: 86% of dissecting aneurysms involving the intrapericardial aorta rupture into the pericardial sac (Shennan, 1934) and 74% of all haemorrhages from dissecting aneurysms are into the pericardium (Frei, 1921). Thus it is clear that the majority of cases that survive will be those with a normal ascending aorta, and most healed dissecting aneurysms begin at or beyond the arch. If the site of a recent dissecting aneurysm can be determined by radiology the prognosis will be directly proportional to the distance of the dissection from the intrapericardial aorta. The second factor that appears to encourage healing is the length of the aneurysm. The short dissections in Shennan's (1934) series ruptured early, and most healed dissecting aneurysms are long and sheath-like, extending from the arch to the iliac arteries. This long distance between the primary tear and the re-entrant aperture may well be due to the fact that the pressure within the aneurysm will approximate to that of the thoracic aorta from which it arises, while the pressure within the aorta at the site of re-entrance will be lowered by successive branching. A re-entrant tear will be favoured by this pressure difference, which increases with the length of the aneurysm, though local factors in the structure of the distal aorta and iliac arteries may also be important.

It is difficult to assess the frequency of healed dissecting aneurysms of the aorta. A suggestive clinical history may be absent (Case 2), and the pathological changes seen in old aneurysms—old thrombosis, endothelialization, and atheroma in the sac—may not be recognizable or may be obscured by recent extension with haemorrhage and thrombosis. The re-entrant aperture, like the primary tear, becomes smooth and inconspicuous, so that it may not be appreciated that the aneurysm was carrying blood and its age may be underestimated; this confusion is especially likely in healed aneurysms that have recently extended, since the clinical and pathological pictures are also those of an acute dissection. The two cases presented here were found in a series of 18 dissecting aneurysms, and Shennan (1934) reported two old dissections with 15 recent cases. Weiss *et al.* (1940) estimated that one dissecting aneurysm in 10 is apt to heal, and, allowing for errors due to selection, it appears that between 5 and 10% of dissecting aneurysms are spontaneously repaired and canalized.

A number of patients with healed dissecting aneurysms have no history of an acute attack (Case 2); they have no history of pain, and they commonly die of cardiac failure (Flaxman, 1942). However, it is not clear if this is a characteristic feature of dissecting aneurysms that heal, since Baer and Goldburgh (1948) found that 50% of acute aneurysms presented without pain. It would be interesting to relate the speed of development of the dissection—of which pain may be an index—with the incidence of healing. Tyson (1931) has described dissecting aneurysms due to vasal haemorrhage that have no communication with the aorta; as these are of capillary origin they must develop more slowly than those associated with an early intimal tear. Tyson believed that intimal rupture was a secondary event in the development of a dissecting aneurysm, and there is no reason why both entrant and re-entrant apertures in healed aneurysms should not follow the production of a silent medial split.

The signs of healed dissecting aneurysms differ from those of the commoner acute type. Since the aortic valve is not often involved, regurgitant aortic murmurs will be unusual. However, Frothingham *et al.* (1939) believed that aortic regurgitation in dissecting aneurysms was caused by ectasia of the aortic ring rather than by deformity of the ring due to the dissection, and if this is accepted it is possible to have an aortic diastolic murmur in the presence of a distal aneurysm which does not involve the intrapericardial aorta. Shennan (1934) described one case with a rough, strong, diffuse bellows murmur over the arch and another with a rough systolic murmur over the manubrium, but these were not clearly related to circulatory abnormalities through the sac, and he concluded that there were no specific diagnostic signs of healed dissecting aneurysms. In most healed aneurysms the heart is found to be enlarged as in both the present cases, but this is not invariable (Weiss *et al.*, 1940).

Differences in pulse pressures are less characteristic of healed aneurysms than of acute cases. This is due to the fact that most healed aneurysms begin at the level of the attachment of the ductus arteriosus distal to the left subclavian artery, and the arteries of the head, neck, and upper limbs will not be involved. In addition, the canalized sac appears to transmit enough blood to equalize the pressures in the upper and lower limbs, and any such differences may be expected to diminish with the development of a patent sac. Radiological changes seem to be the most characteristic findings; Kienbock and Weiss (1931) and Weiss *et al.* (1940) reported that the light shadow of the dissection could be distinguished from the darker aortic shadow in cases of typical "double-barrelled" healed dissecting aneurysms. As in the case of aortic murmurs, these radiological changes will be complicated by the presence of ectasia of the proximal aorta if this coexists with the aneurysm.

In conclusion, it appears that if 5–10% of all dissecting aneurysms heal, then patients with long-sheath-like dissections distal to the arch have a good chance of recovery, and the location of the aneurysm is of prognostic importance. Careful clinical and radiological examination of patients who survive the initial episode should be expected to demonstrate the development of a circulation through a healed dissecting aneurysm.

Summary

The establishment of a circulation through the sac of a dissecting aneurysm of the aorta is the characteristic finding in healed dissecting aneurysm.

It is suggested that patients with a distal sheath-like dissection have a reasonably good prognosis compared with those with proximal dissecting aneurysms involving the intrapericardial aorta. Two cases of distal dissecting aneurysms in which the patients survived for several years are described.

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TWO SURVEYS TO INVESTIGATE THE RELATION OF SICKLE-CELL TRAIT AND MALARIA

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Raper (1949), Brain (1952), Mackey and Vivarelli (1954), and Allison (1954a and b) have suggested that the presence of the sickle-cell trait is a protection against infection with falciparum malaria. Allison further stated that the high sickle-cell-trait rates found in many African tribes can be accounted for on this assumption, and he attempted to relate sickle-cell-trait rates in tribal groups to the severity of malaria. In our view this work was of doubtful validity, since only the trait rates were determined by Allison. His estimations of the malaria distribution were those of other workers; in fact he compared his own sickling rates calculated in 1953 with malaria rates based on Wilson's map for Tanganyika of 1943 (Atlas of Tanganyika, 1948).

It has been shown that sickle-cell-trait rates vary widely, not only within small areas, but also within small subdivisions of the same tribe inhabiting the same area (Foy *et al.*, 1954; Moore *et al.*, 1954; Hiernaux, 1952). Even greater variations occur in malaria incidence at different times and in different places, so that much of Allison's work on this aspect of the subject must be discounted. Our surveys in the field have shown that (a) two adjacent tribal groups with almost identical malaria parasite rates (60%) had very different sickle-cell rates (33% Kambe and 9% Duruma), examinations for the trait and malaria parasites being made on the same individuals (Moore *et al.*, 1954); (b) the sickle-cell-trait rates in the Dinka, Nuer, and Shilluk of the Equatorial Province of the Sudan are 2 to 4%, but malaria is stated to be hyperendemic. It is therefore apparent that, even if the sickle-cell phenomenon and malaria were very closely associated, the attempt to relate the two from data giving the rates for tribal groups as a whole would have little chance of success.