adenocarcinoma of the cervix uteri, demonstrated by histological sections. The younger died of the disease on the day of admission, having had vaginal bleeding for about one month. The second was treated by total hysterectomy eight days after the onset of vaginal bleeding, and was reported well and developing normally 21 months after the operation.

DISSECTING ANEURYSM OF THE AORTA WITH RENAL COMPLICATIONS

BY

H. BREBNER, M.B., Ch.B., M.R.C.P.

Senior Registrar, Kingston Hospital

[WITH SPECIAL PLATE]

Chronic dissecting aneurysm has ceased to be a medical curiosity, and the absence of pain at the time of dissection has often been noted (Gouley and Anderson, 1940; Flaxman, 1942). In the following case there was no history of pain until one month before death, and at necropsy a long-standing dissecting aneurysm was found.

Case Report

The patient, a land-surveyor aged 52, had always enjoyed good health. His parents were over 80 years of age. He consulted his doctor in September, 1947, because of breathlessness on exertion. There was no history of chest pain. His blood pressure was 140/80 and there was no albuminuria. He continued to lead a normal active life. On November 18, 1949, he developed acute pain in the epigastrium radiating to the left side and left shoulder and finally settling in the left loin. He was admitted to a surgical ward, where tenderness was noted in the left loin. His blood pressure was 170/110 and the urine contained albumin but no red cells. Renal colic was diagnosed; the pain ceased in a few days, and he went home. On December 6 he was admitted suffering from increasingly severe dyspnoea. There

FIG. A.—Aorta at site of dissection. The normal ascending part is seen, then the slit of entry. The smooth surface of the false sac is seen just to the left of the probe at the top.

FIG. B.—Kidneys. Dissection of intrarenal branches of left renal artery can be seen. Multiple infarcts in left kidney are visible. (The left kidney is the lower in the Figure.)

had been no recurrence of abdominal pain and he had no pain during his stay in hospital. He gave no history of chest pain at any time.

On examination he was distressed, with acidotic breathing and bleeding from the lips and gums. His complexion was sallow and the mucous membranes were pale. Cardiac enlargement and gallop rhythm were present; there were no murmurs. Blood pressure was 220/150. Numerous moist rales were audible over both lower lobes, but there was no increase in jugular venous pressure nor any oedema. Abdominal palpation was negative. The fundi showed numerous exudates and a few haemorrhages. The urine contained a trace of albumin and very occasional white and red blood cells but no casts. The blood urea was 120 to 169 mg. per 100 ml.; haemoglobin, 74%; red blood cells, 4,020,000. The urinary output was maintained above 40 oz. (1.14 litres) daily, but uraemia persisted and the patient died on December 21.

Necropsy

The heart was enlarged, the right and left ventricular walls being thickened (0.6 cm. and 1.5 cm. respectively). The aorta (Fig. A) was normal up to a slit-shaped opening in the wall 1.2 by 0.3 cm. across, which lay between the left subclavian and the left common carotid arteries, and was the apex of a dissecting aneurysm. This did not involve either of these arteries. The false sac had a smooth lining and extended almost completely round the true descending aorta in its upper 3 in. (7.5 cm.). The dissection then veered more to the left side, where it continued, narrowing, to the iliac bifurcation and then along the left iliac artery to the inguinal ligament, but only a little way along the right iliac artery. No re-entrant was found. There was dissection of the left renal artery, the true lumen being flattened and narrowed. The dissection continued into the main intrarenal branches (Fig. B). The right renal artery was normal. The left kidney (9 by 5 by 4.5 cm., weight 106 g.) had an irregular shape and on section showed a number of pale yellow infarcts. The right kidney (10 by 7.5 by 5.5 cm., weight 210 g.) appeared normal. The lungs showed congestion and

FIG. C.—Section across aorta, showing splitting of media.

bronchopneumonic mottling of the lower lobes. The liver and spleen showed venous congestion.

Histology.—Fig. 1 (Plate) is a section through the aorta at the site of entry, stained for elastic tissue by Verhoeff's method. At the edge of the slit there is disruption of the media, but elsewhere the wall shows no abnormality. Cystic necrosis of the media cannot be demonstrated. (Sections were also examined after staining with van Gieson and H. & E.)

Fig. C is a section across the aorta 4 in. (10 cm.) below the site of rupture, showing splitting of the media.

Fig. 2 (Plate) is a section across the left renal artery showing that dissection has occurred along the outer part of the media in the same way as in the aorta.

Figs. 3 and 4 (Plate) are sections of the right kidney and of a non-infarcted portion of the left. Arteriolar sclerosis is present in both, the appearances being similar to those in hypertension.

Discussion

Clinical Features.—The main finding in chronic dissecting aneurysm is intractable cardiac failure, and renal symptoms are seldom prominent. Buckley (1940) described two cases with haematuria. Baer and Goldburgh (1948), who divided their cases into five clinical groups, mention the renal group as the least common. The presenting symptom in this patient was pain, presumably due to the most recent renal infarction, and thereafter the picture, although it included a slight degree of left ventricular failure, was mainly one of uraemia. The exact nature of the renal lesion was not determined before death. Unfortunately the patient's condition prevented pyelography, which in cases of renal infarction is usually diagnostic (Regan and Crabtree, 1948).

Aetiology.—Hypertension is generally accepted as a causal factor, but it is not always present in cases of

dissecting aneurysm (Mote and Carr, 1942; Schnitker and Bayer, 1944). The last-named authors consider that it plays only a small part in the aetiology. In the present case the date of dissection is unknown, but it was apparently long-standing. This opinion is based mainly on the discrepancy in size between the two kidneys. The right had hypertrophied to one and a half times the average normal size, and the left had shrunk to less than half normal. These changes, particularly the hypertrophy, must have occurred over a period of months rather than weeks. The smooth appearance of the false sac was consistent with dissecting aneurysm of long standing. Since the blood pressure was normal two years before death, it is unlikely that hypertension was an important factor in causing dissection.

Renal Infarction and Hypertension.—In a period of two years the blood pressure rose to a severe hypertensive level, and retinopathy, left ventricular failure, and uraemia developed. Is it possible that unilateral renal infarction was responsible for this? It is known that renal ischaemia may cause hypertension (Goldblatt *et al.*, 1934); and Loomis (1946) produced hypertension in rats by causing renal infarction. There is clinical evidence that unilateral kidney disease in man may lead to hypertension, probably in individuals with a hereditary predisposition (Palmer *et al.*, 1940). In patients suffering from rheumatic and coronary heart disease renal infarction has caused the rapid development of hypertension (Fishberg, 1942; Wainwright, 1949). Yuile (1944) maintains that in order to prove the presence of the "Goldblatt mechanism" one must demonstrate arteriolar sclerosis in the kidney with a normal blood supply without such changes in the ischaemic kidney. He could find only three cases to conform to these strict criteria. In this case the arteriolar changes were of equal degree in both kidneys.

In the case here described there is no evidence to prove that unilateral infarction of the kidney was the cause of hypertension. It is certain, however, that renal infarction was present at a time when the hypertensive process (whatever its cause) was progressing with great rapidity, and there is little doubt that the infarcted kidney exerted an important adverse influence on this process.

Summary

A case of chronic dissecting aneurysm of the aorta is described, with dissection of the renal artery and renal infarction.

The presenting symptom was renal colic, and the main clinical finding was uraemia.

It is suggested that the infarcted kidney was responsible for the rapid progress of hypertension.

I wish to thank Dr. J. C. Lees for the pathological report and for much valuable advice on the case. I am indebted to Dr. J. W. Joule for helpful criticism, and to Dr. M. H. Fridjohn for providing details of his examination of the patient in 1947.

REFERENCES

- Baer, S., and Goldburgh, H. L. (1948). *Amer. Heart J.*, **35**, 198.
- Buckley, T. I. (1940). *J. Urol.*, **44**, 816.
- Fishberg, A. M. (1942). *J. Amer. med. Ass.*, **119**, 551.
- Flaxman, N. (1942). *Amer. Heart J.*, **24**, 654.
- Goldblatt, H., *et al.* (1934). *J. exp. Med.*, **59**, 347.
- Gouley, B. A., and Anderson, E. (1940). *Ann. intern. Med.*, **14**, 978.
- Loomis, D. (1946). *Arch. Path.*, **41**, 231.
- Mote, C. D., and Carr, J. L. (1942). *Amer. Heart J.*, **24**, 69.
- Palmer, R. S., Chute, R., Crone, N. L., and Castleman, B. (1940). *New Engl. J. Med.*, **223**, 165.
- Regan, F. C., and Crabtree, E. G. (1948). *J. Urol.*, **59**, 981.
- Schnitker, M. A., and Bayer, C. A. (1944). *Ann. intern. Med.*, **20**, 486.
- Wainwright, J. (1949). *Lancet*, **1**, 62.
- Yuile, C. L. (1944). *Amer. J. med. Sci.*, **207**, 394.

T. TOMASZEWSKI: SIDE-EFFECTS OF CHLORAMPHENICOL AND AUREOMYCIN

FIG. 1.—Scraping of the tongue before treatment. Mixed bacterial flora. ($\times 1,000$.) The patient, who was treated with chloramphenicol, 4 g. daily to a total of 96 g., developed hypertrophic glossitis with brown coloration of the tongue.

FIG. 2.—Third day of chloramphenicol treatment. Absence of bacteria; scattered groups of fungi.

FIG. 3.—Twenty-fourth day of chloramphenicol treatment. Absence of bacteria; rich growth of fungi. Treatment stopped on that day.

H. BREBNER: DISSECTING ANEURYSM OF AORTA

FIG. 1.—Section through aorta at site of dissection, showing disruption of media.

FIG. 2.—Renal artery, showing dissection.

FIG. 4.—Seventeen days after stopping chloramphenicol. Bacteria reappeared after a week, and are here seen in large numbers; scattered groups of fungi are still present.

FIG. 3.—Section of right kidney. Arteriolar sclerosis is seen.

FIG. 4.—Section of left kidney. Arteriolar sclerosis is seen. The edge of an infarcted portion is visible.