already reported for F.D.P. and again the rise of C3 and F.D.P. excretion tended to coincide.

Until now little attention has been given to the excretion of C3 in the urine of patients with glomerulonephritis. Indeed the published reports (Lange and Wenk, 1954; Lagrue *et al.*, 1969) have been concerned with establishing that the low serum C3 concentrations have not been produced by excessive urinary C3 loss. Recently C3 has been shown to be present in the urine of patients with proliferative glomerulonephritis and after renal transplantation using the relatively insensitive radial immunodiffusion assay (Hoq *et al.*, 1974). Our preliminary results indicate that C3 excretion occurs often in patients with glomerulonephritis and suggest that further studies may be useful.

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MEDICAL MEMORANDA

Hypertension with Dissecting Abdominal Aortic Aneurysm

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A 46-year-old bricklayer presented with a three-hour history of sharp upper abdominal pain. The onset was sudden and occurred while he was seated watching television. The pain was midline and extended from the lower sternum to the umbilicus; there was no radiation. He was given 5 mg intramuscular diamorphine and had no further pain. The referring doctor noted slight epigastric tenderness but on arrival at hospital there were no abnormal signs in the abdomen. Both femoral pulses were normal and no bruit was audible. The heart rate was 100/min and the blood pressure 185/155 mm Hg. A hypertensive retinopathy with arteriovenous nicking was noted but the apex beat was not displaced. An E.C.G. was normal.

The patient had been found to be hypertensive three years previously and at that time an intravenous pyelogram was normal, blood urea was 43 mg/100 ml, and urinary catecholamine excretion was within normal limits at 20 µg/24 hr. His blood pressure had been maintained at 160/100 mm Hg supine and 150/90 mm Hg standing on 80 mg debrisoquine daily. He had taken 20 mg debrisoquine two hours before admission but four hours after arrival his blood pressure was 220/140 mm Hg, and by eight hours it had risen to 300/180 mm Hg. He was nursed in a head-raised position and in addition to debrisoquine 20 mg six-hourly was treated with subcutaneous injections of pentolinium every two hours, initially 2.5 mg and increasing to 15 mg. This regimen was continued for 60 hours but the diastolic blood pressure never fell below 130 mm Hg. He remained symptom-free throughout. A sinus tachycardia of 150/min then developed and 24

hours later he was first noted to have a diminished left femoral pulse with a bruit over the upper abdomen. A satisfactory urinary output was maintained; however, the urine was found to contain some red cells. A right transfemoral aortogram (see fig.) confirmed a dissection just below the renal artery on the right side extending down to the aortic bifurcation. There was evidence of involvement of the right renal artery; the left renal artery appeared to be normal. It was felt that absolute indications for surgical intervention were present in view of uncontrollable hypertension with evidence of major branch occlusion.

At operation the radiological findings were confirmed though there was some retrograde dissection for a distance of 1-2 cm above the level of the renal artery. The right kidney appeared to be adequately

Aortogram showing aortic dissection affecting right renal artery.

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perfused though the pulsation in the right renal artery was diminished. The left renal artery was normal. The major part of the dissection had occurred distally and affected about two-thirds of the circumference of the aorta. The left common iliac artery was involved at its origin by an extension of the dissection.

In view of the involvement of the renal artery in the dissection it was decided that a simple re-entry procedure would be more suitable than any form of major vascular replacement (De Bakey *et al.*, 1955, 1965). The aorta was mobilized and all branches were controlled. In order to maintain renal perfusion the aorta was cross-clamped distal to the renal arteries and therefore distal to the site of the dissection. To prevent further extension of the retrograde dissection it was necessary to achieve and maintain a moderate degree of hypotension, a point emphasized by other workers (De Bakey *et al.*, 1955). The systolic blood pressure was maintained at 100 mm Hg by using low concentrations of halothane (0.5-1%) in conjunction with *d*-dubocurarine. An infusion of 500 ml 10% mannitol was given to protect renal function (Barry *et al.*, 1961).

The aorta was then transected 2 cm above the bifurcation. The re-entry procedure consisted simply in suturing back the dissected intima to the adventitia and media of the distal segment, thereby occluding the dissection. A window about 1.5 by 2 cm was then excised from the proximal detached intima to effect an adequate re-entry point. The aorta was then repaired with continuous 4/0 Dacron and the anastomosis supported by a sleeve of woven Dacron. During the procedure fluids were infused to maintain the central venous pressure at 12 cm of water to obviate any possible uncontrollable hypotension on release of the arterial clamps. All the pulses returned to normal when the clamps were removed.

Both to ensure a stable respiratory state and to aid intravenous pentolinium in the control of hypertension (Enderby, 1954) it was decided to ventilate the patient for the first 12 hours postoperatively.

In the immediate postoperative period the previously uncontrollable hypertension became easily manageable with occasional small doses of pentolinium. The blood pressure at that time was maintained at 150/90 mm Hg. Throughout the postoperative period a good urinary output was maintained though the blood urea rose to 92 mg/100 ml by the third postoperative day but rapidly returned to normal. From then on the patient made an uninterrupted recovery. By the seventh postoperative day his blood pressure was controlled on 60 mg debrisoquine daily. Subsequently his blood pressure was maintained at 150/90 mm Hg supine and 130/80 mm Hg standing on this dose of debrisoquine. He remained well and was working full-time one year after the operation. At the time of writing his blood pressure was 140/80 mm Hg on a reduced dose of his antihypertensive agent.

Comment

Goldblatt *et al.* (1934) showed in experimental animals that partial renal artery occlusion is followed by rapid-onset hypertension. The present case is a striking clinical example of the Goldblatt phenomenon. The patient, a well-controlled hypertensive, suffered an abrupt and uncontrollable rise in blood pressure produced by partial occlusion of the right renal artery secondary to aortic dissection.

The hypertension caused by renal artery occlusion does not appear to be due to renal ischaemia but probably results from a fall in perfusion pressure to the affected kidney. The juxtaglomerular apparatus responds by releasing renin, which reacts with plasma angiotensinogen to form the vasoconstrictor angiotensin.

Hypertension is a well known clinical manifestation of aortic dissection (Hirst *et al.*, 1958). It has been suggested that many factors may play a part in the causation of this rise in blood

pressure (Burchell, 1955). Since hypertension is so common with aortic dissection when patients first present it is difficult to be sure how often the Goldblatt phenomenon occurs. It may, however, be more common than previously recognized, particularly as anuria due to renal artery occlusion is a well known complication of aortic dissection.

The management of acute aortic dissection is a complex clinical problem. The conservative management, using sustained hypotension, is now well established (McFarland *et al.*, 1972). In the presence of major branch involvement, however, surgical treatment becomes mandatory.

The surgical management of aortic dissection has been discussed by various authors (De Bakey *et al.*, 1955, 1965; Eastcott, 1969). Since the treatment is still evolving, however, it is difficult to be certain of the best approach (Bloor, 1973). For dissection involving the ascending aorta aortic arch, and the remaining thoracic aorta major reconstruction with excision and grafting of the affected segment appears to produce the most satisfactory results (De Bakey *et al.*, 1965). The simple re-entry procedure (De Bakey *et al.*, 1955) when applied to the proximal aorta carries a high operative mortality rate associated with a large number of late failures. In the rare cases of dissection limited to the abdominal aorta, however, particularly with renal artery involvement, the simple re-entry procedure with reinforcing of the aorta using a Dacron sleeve has much to commend it.

At operation in this case the right kidney was adequately perfused though under reduced pressure. After the partial occlusion of the right renal artery by aortic dissection there was a rapid rise in blood pressure which was followed by an equally rapid fall when the compression was relieved, thus satisfying the Goldblatt postulate.

Unfortunately no renin or angiotensin estimations were performed and it could be suggested that in the presence of a raised renin level temporary control of the blood pressure might have been achieved using propranolol, which lowers plasma renin (Michelakis and McAllister, 1972). Undoubtedly, however, in this case successful control of hypertension was achieved by surgery.

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