99% complete (Hughes and Tanner, 1970) and the equivalent age in human terms (based on bone age) would be about 16 years. In adult life changes in weight in rats (Hollenberg and Vost, 1968) and in humans (Hirsch *et al.*, 1966; Sims *et al.*, 1968) have been found to correlate well with changes in adipose cell size, but total cell numbers are not affected.

It therefore seems that the total number of adipose cells becomes fixed towards the end of childhood, and the results of our investigations in obese children suggest that the period during which the *rate* of fat cell multiplication is most affected by overnutrition extends to about the age of 1 year. It may be relevant that also at about this age the skinfold thicknesses in normal infants stop increasing (Tanner and Whitehouse, 1962). It seems likely that during this sensitive period the basic complement of fat cells for an individual is established and that thereafter cell multiplication proceeds at a normal rate, regardless of nutritional circumstances, changes in which are reflected by changes in adipose cell size.

It has been shown that the total number of adipose cells cannot be reduced by dieting, either in obese adults (Salans *et al.*, 1968) or in children (Brook, 1971c). It is not yet known, however, whether the rate of multiplication of adipose cells during later childhood can be arrested by relative undernutrition, nor whether infants who have laid down an increased number of cells during the first year of life may be expected to maintain this increase even if dietary intake is subsequently curtailed. Studies of the natural history of childhood obesity (Lloyd *et al.*, 1961) show that obesity persists into adult life in about 80% of children, but no investigation has been made relating the age of onset of the obesity to the outcome in individual children. Prospective studies are required to determine whether children whose obesity started in the first year of life and who have an increased number of adipose cells have a different prognosis from those whose obesity started later and whose adipose cell number is normal.

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MEDICAL MEMORANDA

Reversible Renal Failure with Renal Artery Occlusion

O. P. QUANTOCK, G. N. THATCHER

British Medical Journal, 1972, **2**, 27–28

Acute occlusion of the main renal artery whether by trauma, embolism, or thrombosis produces acute failure of function of that kidney. Return of renal function can be expected if the obstruction is removed within a few hours. There are reports of return of renal function after delayed surgical revascularization, however, even when this is delayed up to 39 days (Perkins *et al.*, 1967). Viability but not function is presumably maintained by collateral renal vessels (Love and Bush, 1968). Spontaneous recovery of function after unilateral renal artery occlusion has been described (Bellman and Odén, 1960; Heitzman and Perchik, 1961; Fergus *et al.*, 1969). We report a case of acute renal failure in a patient with only one kidney in whom occlusion of the renal artery occurred and in whom life-sustaining renal function returned spontaneously after prolonged dialysis.

Groote Schuur Hospital, Cape Town, South Africa
O. P. QUANTOCK, M.B., B.CH., F.C.P.(S.A.), Senior Registrar
G. N. THATCHER, M.B., CH.B., F.C.P.(S.A.), Physician to Renal Service

A 48-year-old man was investigated for accelerated hypertension in March 1966. His blood pressure was 240/170 mm Hg, blood urea 66 mg/100 ml, and creatinine clearance 47·5 ml/min. I.V.P. showed a non-functioning right kidney. An aortogram showed gross stenosis at the origin of the right renal artery. There was stenosis at the origin of the left renal artery and poststenotic dilatation. A right nephrectomy was performed and a Teflon graft placed between the left common iliac artery and the poststenotic portion of the left renal ortery (Fig. 1). Biopsy of the left kidney showed features of gross ischaemia. Postoperatively his blood

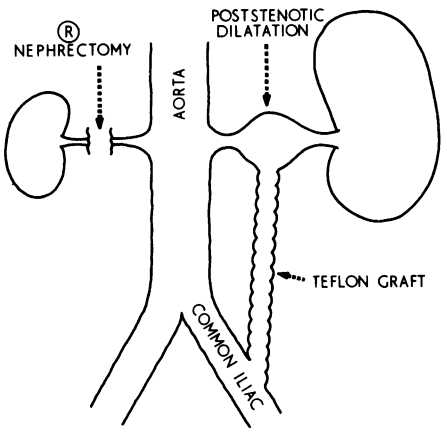

FIG. 1—Diagram of postoperative renovascular status (1966).

pressure fell to about 190/110 mm Hg and his retinopathy regressed. He was treated with methyldopa and remained well apart from moderate hypertension.

In January 1971 he was admitted with gastrointestinal haemorrhage. He did not appear oligaemic. His blood pressure was 170/90 mm Hg, and apart from left ventricular hypertrophy examination showed nothing abnormal. During blood transfusion (900 ml) he developed pulmonary oedema with a rise in blood pressure. Morphine and frusemide (Lasix) (80 mg) were followed by a prompt diuresis.

The next day pulmonary oedema recurred. Frusemide 600 mg was ineffective. He remained oliguric for two months, requiring repeated peritoneal dialyses. Ten days after onset of the oliguria a high-dose I.V.P. showed a faint nephrogram on the left. Aortography showed an aneurysmal abdominal aorta. The left renal artery was totally occluded just beyond its origin. A dilated ureteric collateral rising from the left internal iliac artery emptied into the distal left main renal artery and filled the intrarenal vessels. There was no evidence of cortical necrosis. The Teflon graft inserted in 1966 was not functioning (Figs. 2 and 3). On 9 March, 66 days after admission, his urine output rose to over 1,000 ml/24 hr under the influence of intravenous frusemide 1 g daily. His creatinine clearance rose gradually to 15 ml/min and his blood urea fell to 156 mg/100 ml on the 70th day. Six months after admission he was reasonably well, with blood pressure 190/100 mm Hg, blood urea 110 mg/100 ml, and serum creatinine 2·2 mg/100 ml on 800 mg of frusemide daily (Fig. 4).

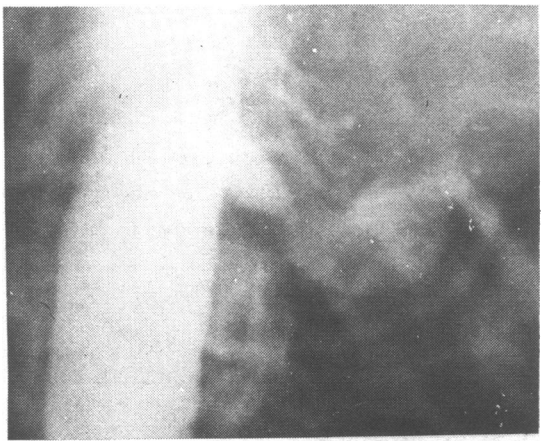

FIG. 2—Aortogram showing occluded left renal artery (January 1971).

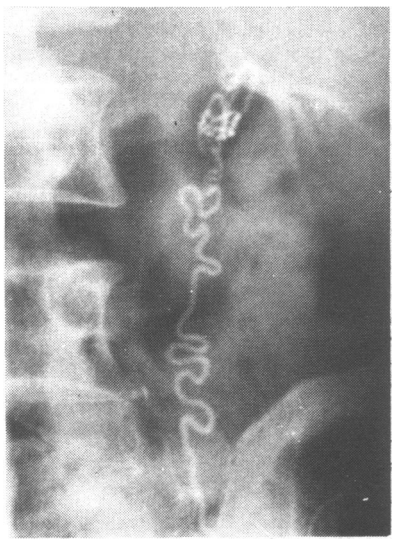

FIG. 3—Aortogram. Delayed film showing large periureteric collateral artery (January 1971).

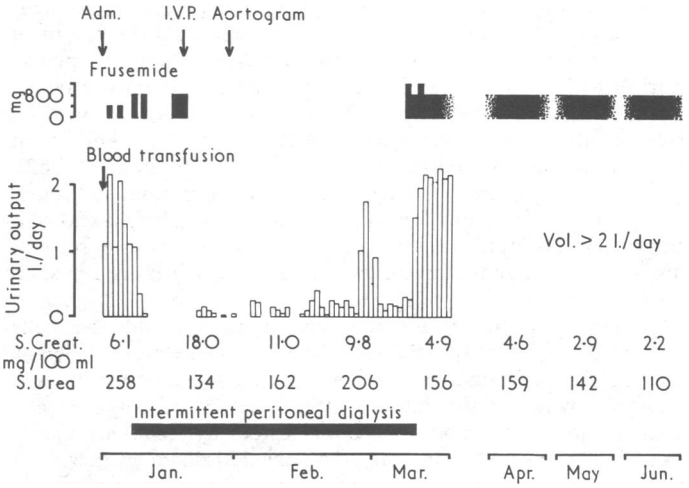

FIG. 4—Course of renal failure. ADM = admission.

Comment

This patient developed acute oliguric renal failure owing to acute thrombotic occlusion of his stenosed renal artery or to acute tubular necrosis consequent on the haemodynamic disturbance occasioned by the gastrointestinal bleed, transfusion, and subsequent heart failure. In the latter instance one must suppose that gradual occlusion of the renal artery had preceded the present illness, with renal function being maintained by the simultaneous development of a collateral circulation (Wong and Chow, 1964; Kolsaker and Ofstad, 1966). Recovery of renal function may have been the result of recanalization of the renal artery (Bellman and Odén, 1960; Fergus *et al.*, 1969) or due to repair of tubular necrosis despite continuing ischaemia.

In patients with severe atheromatous vascular disease sudden deterioration of renal function may result from occlusion of the renal arteries. Prompt or sometimes delayed surgical revascularization may improve renal function. Where surgery is contraindicated or delayed recovery may still occur, as in this case, provided an adequate collateral circulation is present. Recovery of renal function may be slow and a lengthy period of dialysis may be required.

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