HYPERTENSION IN UNILATERAL KIDNEY DISEASE: A SHORT DISCUSSION AND PRESENTATION OF CASES*

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The death rate from hypertension in the USA has been quoted as being 4 times that of cancer and 20 times that of diabetes mellitus or tuberculosis. The surgical treatment to date is empirical and medical treatment is difficult and requires constant supervision. The recognition, therefore, of hypertension due to unilateral kidney disease† is of importance, for this form of hypertension is often curable if dealt with in the early stages.

The classical work of Goldblatt and others (1934) showed the close association between unilateral renal ischaemia and hypertension, and since then the subject has been opened up by a number of publications, some of which are included in the bibliography. The mechanism of unilateral renal ischaemia as a cause of hypertension is not clearly understood, but something instrumental in increasing blood pressure is elaborated or produced by the ischaemic kidney and this pressor substance has been isolated from both normal and ischaemic renal tissue. It is thought to be a proteolytic ferment, renin, which acts on a globulin substrate in the plasma to produce a powerful pressor agent termed angiotonin, hypertensin (Braun-Menéndez) or now, by mutual agreement, angiotensin, and this substrate has been determined and synthesized. A great deal of experimental work has been done on the renin-angiotensin system but, although not fully understood, the mechanism is thought to be active in the acute stage of the hypertensive process that may follow a unilateral renal injury, but to almost disappear after the chronic stage has become established.

It is all-important in hypertension from unilateral disease that the treatment should be instituted as early as possible and before the chronic irreversible stage develops.

We have confirmed the clinical features in the successful cases quoted by Perera and Haelig. The onset is usually relatively abrupt in a subject previously normotensive, but there are a few cases in which the condition was preceded by benign essential hypertension. The disease tends to run a severe rapid course and to develop into the malignant phase. The onset may be marked by headaches, progressing rapidly to frank encephalopathy and retinopathy with visual disturbances and grave changes in the retinal artery. Later, general involvement develops, with albuminuria and a rapid drop in kidney function. The disease may begin at any age, but an onset before 20 or after 45 is highly suggestive, because essential hypertension is not commonly seen at these ages. The condition may be precipitated by trauma to the renal region, whether accidental or surgical. In embolism or thrombosis of the renal artery there is often a history of an acute attack of pain in the flank or lower abdomen and renal colic or appendicitis may be simulated. So a history of hypertension, developing within a few days of an injury or in an illness in a subject known to have had normal blood pressure, is important.

When these cases are investigated the renal function tests are usually not of help, for the function of the good kidney is usually temporarily reduced by the hypertension. *Intravenous pyelography* is sometimes helpful and it should, if possible, be carried out. A small renal shadow on one side is important; a fainter shadow on one side suggests reduced circulation or impaired tubular function, and failure of the intravenous pyelogram to visualize the pelvis of the kidney, which later yields a normal retrograde pyelogram, strongly points to an obstruction in the renal artery. However, normal pyelograms are frequently obtained with serious unilateral disease.

Differential renal-function tests by means of bilateral ureteral catheterization, so well described by Howard and his associates, are useful investigations. A diminished volume of urine, together with a lower concentration of sodium from one kidney in specimens taken simultaneously, is highly significant, and a reduction in the volume of urine of at least 50% and a reduction in sodium concentration of at least 15% as compared with the opposite kidney predicts a favourable outcome from surgery. The most dependable single investigation is aortography‡ and this procedure we have found practical and the risk involved relatively slight. Poutasse and Dustan (1956) believe that it is indicated in the following groups of patients with hypertension:

1. Those with unexplained disparity of size or function of the kidneys as shown by intravenous pyelography.
2. Young patients with no family history of hypertension and no apparent cause for it.
3. Elderly patients with sudden onset of accelerated hypertension.
4. Patients with essential hypertension, whose disease suddenly accelerates, especially when this acceleration follows an attack of unexplained pain in the flank.

Lipo-protein studies have been found of use in cases of atherosclerosis and have in a number of cases predicted to some degree what has later been demonstrated by aortography.

CASE REPORTS

Case 1

Y.H., aged 9 years. This girl was first seen on 16 August 1952 as an urgent case presenting with recurring convulsions, which required control by general anaesthesia on 2 occasions and by continuous deep sedation.

The mother reported that the girl was healthy until 18 months previously, and that since then she often woke up with a headache, suffered recurring attacks of nausea and vomited occasionally, but attended school until the day before the first convulsion.

On examination. A pale, very ill-looking child. Fundi showed grade-4 eye changes. Blood pressure 250/180 mm.Hg. Femoral pulses present and equal. Heart not clinically enlarged. Heart sounds closed with a marked accentuation of the aortic

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† See Editorial 'Narrowing of the renal artery', *S. Afr. Med. J.*, 6 February 1960 (**34**, 110).

‡ See article by Drs. Denny and Tinker on page 852 of this issue of the *Journal*.

second sound. No clinical evidence of an abdominal tumour. Urine: + albumin; centrifuged specimen showed numerous red cells, an occasional leucocyte, and no casts. Alkaline phosphatase 11·4 units %, inorganic phosphate 3·0 mg.%, serum calcium 8·7 mg.%, blood urea 45 mg.%, blood creatinine 0·8 mg.%. Benzodioxane intravenous test negative. Before this child was referred, an intravenous pyelogram had revealed some doubtful flattening of the upper calyx of the right kidney, but a retrograde pyelogram done immediately afterwards was reported to be within normal limits. X-ray of the wrists and ankles showed no evidence of renal rickets. The child's condition deteriorated 36 hours after admission and she developed a large retinal haemorrhage in the right eye.

Treatment. Although the prognosis appeared to be hopeless and because a definite diagnosis of the cause of the hypertension could not be made, it was decided to attempt a combined Smithwick operation and suprarenalectomy as a last resort. This was performed by Mr. Lee McGregor in two stages at an interval of 10 days. The right side was done first and following this the blood pressure dropped to 170/150 mm.Hg, and the child's condition improved; and soon after the second stage she improved rapidly and the blood pressure stabilized at about 120/90 mm.Hg and was easily controlled by replacement cortisone therapy. Her general condition improved daily and 2 weeks after the second stage the fundi had resolved entirely, the urine was free of albumin and the patient was able to leave hospital.

Pathological report on kidney biopsies showed nephrosclerosis of the hypertensive type.

23 September 1953. Enjoying good health on a maintenance dose of cortisone, 5 mg. twice a day. Tonsils successfully removed in August 1953. Recovered from whooping cough and measles without complications. Fundi normal. Blood pressure 120/88 mm.Hg. Urine concentration test, specific gravity 1020. Urine dilution test, specific gravity 1006.

30 September 1957, when aged 14 years. On maintenance cortisone, 5 mg. twice a day, and keeping very well. Healthy looking, but a little undersized. Normally developed. Fundi within normal limits. Blood pressure 140/90 mm.Hg. Pubic hair scanty. Heart not enlarged on screen examination. ECG within normal limits. Urine contained ? trace of albumin. Blood urea 28 mg.%. Electrolyte studies within normal limits. Intravenous pyelogram showed a compensatory hypertrophy of the right kidney and a small left kidney with some blunting of the calyces; the renal parenchyma was narrowed, measuring about 1 inch, and the appearance was suggestive of an old pyelonephritic kidney.

25 April 1958. Very well on cortisone, 5 mg. twice a day. Blood pressure 135/90 mm.Hg. Fundi within normal limits. Urine contained a trace of albumin. Nephrogram (Dr. M. Denny) showed a contracted left kidney with poor arterial supply. Normal right kidney. Selective ureteral catheterization (Mr. D. Venn): right kidney normal; left kidney low urea excretion, low volume, albumin ++, and no excretion of dye on the p.s.b. test. Left-sided nephrectomy advised.

2 September 1958. Left-sided nephrectomy (Mr. N. G. C. Gane, Salisbury). Pathological report: generally contracted chronic pyelonephritic kidney.

January 1959. Well-proportioned, normal pubic and axillary hair, no acne, menstruating regularly and normally, blood pressure 135/90 mm.Hg, urine albumin-free. Marked pigmentation of the nipples; no other pigmentation. Leads a full life, plays all games and is popular with the boys and a keen exponent of 'jive' and 'rock-and-roll'.

Review of case. The cause of the original hypertension is considered to have been a left-sided pyelonephritis.

Case 2

Mr. R.W.F., aged 59 years, postmaster. Seen on 6 December 1954, this patient complained of severe headaches for the previous 7 months, with progressive tiredness and attacks of irritability, and for the last 2 months a sudden progressive deterioration in vision.

Past health (record obtained from his family doctor). Had always been a healthy subject; served in World War II, but was boarded out on return from Abyssinia as a case of ? peptic ulcer. 1952, healthy (blood pressure 128/78 mm.Hg). 1953, blood pressure 160/90 mm.Hg. May 1954, blood pressure 190/110 mm.Hg. October 1954, seen by an eye specialist, who reported retinal haemorrhages.

On examination. A well-built man. Fundi showed grade-4 eye changes. Blood pressure when lying, 250/120 mm.Hg. Peripheral vessels not thickened; peripheral pulses all present and equal. Heart was not enlarged clinically. Heart sounds closed with a split second sound at the base. ECG showed left ventricular strain. Urine: Albumin +++; centrifuged urine showed moderate numbers of red cells, leucocytes, and hyaline and granular casts. Blood urea 36 mg.%. Blood count normal. Intravenous pyelogram showed poor function of the left kidney which was small, with some narrowing of the cortex. Aortogram within normal limits, but the nephrogram on the left side revealed poor vascularization, especially of the upper pole.

Treatment. 20 December, left-sided nephrectomy (Mr. C. Thompson). This was followed by an immediate drop in blood pressure to 130/90 mm.Hg. Three weeks after operation: blood pressure 150/90 mm.Hg, lying down; urine concentration test, specific gravity 1030; urine dilution test, specific gravity 1012; blood urea 40 mg.%.

Pathological report. Chronic pyelonephritic kidney.

Follow-up, June 1959. Has remained normotensive and is in normal health.

Case 3

Mr. H.M., aged 46 years. Seen on 29 December 1955, this patient had enjoyed good health and been normotensive until 9 months previously, when he began complaining of early morning headaches and progressive tiredness. His family doctor reported a sudden rise of blood pressure to about 180/110 mm.Hg, which 3 months later rose to about 200/140 mm.Hg and the patient was found to have albumin in the urine.

On examination. A well-built man. Fundi showed grade-1 eye changes. Peripheral vessels doubtfully thickened; all the peripheral pulses present and equal. Heart not enlarged clinically or on screen examination. Heart sounds closed and of good character. ECG within normal limits, except for tall T waves in the chest leads. Urine: Albumin ++++; centrifuged specimen showed 2 red cells per high-power field, together with epithelial cells and scanty leucocytes, and numerous hyaline casts and scanty granular casts; culture yielded a growth of *Staphylococcus albus*. Blood urea 42 mg.%. Blood creatinine 1·5 mg.%. Blood count within normal limits. Intravenous pyelogram: Right kidney shadow was faintly visualized, longitudinal measurement 3¼ inches; left kidney showed good dye excretion, longitudinal measurement 4¼ inches. Retrograde pyelogram: The shadow of pelvis and calyces of the right kidney were relatively small and disproportionately large in relation to the size of the renal outline, indicative of atrophy of the renal parenchyma; the left kidney was within normal limits.

The sudden onset of hypertension with albuminuria, a poorly secreting right kidney on intravenous pyelography, and the retrograde pyelogram findings, were considered to be consistent with the diagnosis of a right-sided chronic pyelonephritis and a nephrectomy was recommended.

4 January 1956. Right-sided nephrectomy (Mr. C. Thompson). Blood pressure on the morning of operation under preoperative sedation was 230/135 mm.Hg and immediately after operation it dropped to 115/80 mm.Hg. Three weeks later: Blood pressure 110/80 mm.Hg; urine—albumin ++, centrifuged specimen showed an occasional pus cell and an occasional hyaline cast but no red cells.

Pathological report. The kidney was reduced in size, the surface was finely granular, and the capsule was adherent. On section the cortex showed irregular atrophy. The kidney showed the histological features of chronic pyelonephritis.

3 months after nephrectomy. In good health. Blood pressure about 135/90 mm.Hg. Urine free from albumin.

October 1957. Not so well. Complained of renewed tiredness. Normotensive. Urine clear.

2 February 1958. Referred for reassessment. Complained of progressive tiredness, but no effort dyspnoea or effort angina.

TABLE I, CASES 1-6

Age, Sex	*BP*	*Presenting symptoms*	*IV Pyelogram*	*Aortogram*	*Lesion*	*Treatment*	*Follow-up*
9 yrs female	250/180	18 months—early morning headaches, nausea, vomiting 1 week—convulsions	L kidney—small, contracted	L kidney—small, poor blood supply	L kidney—chronic pyelonephritis	20.8.53—bilateral thoracolumbar sympathectomy 2.9.58—L nephrectomy	7 yrs: BP 130/90 mm. Hg, excellent health on cortisone 5 mg. *b.d.*
59 yrs male	250/120	7 months—severe headaches, deterioration in vision	L kidney—small poor function	L kidney—poor vascularization, especially the upper pole	L kidney—chronic pyelonephritis	L nephrectomy	5 yrs: BP 150/90 mm.Hg, normal health
46 yrs male 29.12.55	200/140	9 months—early morning headaches progressive tiredness	R kidney—poor concentration, smaller than left	nil	R kidney—chronic pyelonephritis	4.1.56—R nephrectomy	1½ yrs: BP 135/90 mm.Hg. good health
2.2.58	140/90	Progressive tiredness	nil	Aorta atherosclerotic L common iliac and R superficial femoral occluded	Atherosclerosis	Diet, anticoagulants, nicotinic acid	4 yrs: Normotensive, improved
54 yrs male	200/120	Sudden vertigo, temporary paralysis of L arm and L leg for 10 minutes	Normal	R nephrogram—normal L nephrogram—faint filling Aorta atherosclerotic	Atherosclerosis. ?? atherosclerotic stricture of left renal artery	Diet, anticoagulants, nicotinic acid	1 yr: BP 160/100 mm.Hg, improved
49 yrs female	230/150	6 months—severe headaches with vomiting Progressive loss of energy	R kidney—normal L kidney—ptosed, poor function	R kidney—renal artery, small saccular aneurysm L kidney—poor visualization	Polycythaemia vera with renal thrombotic incidents	Radio-active phosphorus	1½ yrs: Normotensive, good health, blood count normal
32 yrs male	130/85	Examination for old inactive bilharzia	R kidney—normal L kidney—?? right upper calyx	nil	Treated bilharzia	nil	5 yrs: Normal BP, good health
38 yrs	180/90	Headaches, tiredness	L kidney—one large cyst	nil	Congenital cysts (4)	Surgical removal	6 yrs: Normotensive
41 yrs	240/160	Ill, severe headaches	Both kidneys—polycystic disease	nil	Congenital polycystic disease	nil	9 yrs: Grave hypertensive renal disease

On clinical examination: Fundi within normal limits; in right arm radial pulse absent and blood pressure not recordable; left arm blood pressure 135/90 mm.Hg; heart not clinically enlarged; heart sounds closed and of good character; resting ECG within normal limits; effort ECG no change; pulses present in the right leg, but left femoral and left pedal pulses absent. *Aortogram:* Abdominal aorta grossly atherosclerotic and the outline irregular; marked narrowing of the origin of the right common iliac; left common iliac completely occluded from its origin to the commencement of the superficial femoral artery; good collateral circulation to the left leg by way of the gluteal vessels and the lumbar collaterals; right femoral artery occluded from the origin of the profunda femoris to the commencement of the adductor canal, and collateral circulation carried *via* the muscular branches of the profunda femoris and the re-formation of the femoral artery below the adductor canal; the popliteal vessels and the terminal vessels of the lower limbs filled normally. *Lipoprotein partition:* Total cholesterol 250 mg.%; beta lipoprotein cholesterol percentage 83; cholesterol phospholipid ratio 1·01.

Diagnosis: The patient was considered to be atherosclerotic with occlusive vascular disease of both legs.

Progress report. 21 February 1959. On a low-fat, low-cholesterol diet, supplemented with vitamin A, and with anticoagulant treatment, the patient has been keeping well and symptom-free.

Final review. This patient is considered to have been initially a case of unilateral chronic pyelonephritis causing hypertension, which was relieved by nephrectomy; and with later development of atherosclerotic disease with occlusive thrombotic disease in several sites and symptomatic improvement on a low-fat, low-cholesterol diet and anticoagulant treatment.

Case 4

Mr. K.M.F., aged 54 years, electrical engineer. Seen on 3 June 1958, this man had always enjoyed good health until 3 weeks previously, when he experienced a sudden attack of giddiness and paralysis of the left arm and left leg, which passed off in about 10 minutes. He was attended by his family doctor, who found that his systolic blood pressure was 220 mm.Hg, and venesected about 1 pint of blood, but the systolic blood pressure remained raised at about 210 mm. After this he continued working and appeared to be reasonably well, but walking fast for 200 yards brought on a cramp in the left leg which was immediately relieved by rest.

On examination. A muscular well-built man. Fundi were within normal limits. Peripheral vessels thickened, but not tortuous. In left leg femoral and pedal pulses absent. Blood pressure 200/120 mm.Hg. Heart not enlarged on screen examination. Heart sounds closed and of good character. ECG within normal limits. Urine: Free of albumin and sugar; centrifuged urine shows nothing of note. Blood count was within normal limits. Lipoprotein partition studies: Total cholesterol 336 mg.%; beta lipoprotein cholesterol percentage 81; cholesterol phospholipid ratio 1·11. Intravenous pyelogram showed normal function and no abnormality of note. Biological and chemical tests for catecholamines negative.

Aortogram: Right nephrogram was within normal limits. In spite of three injections, only faint filling of the left renal artery was obtained, and at no time was an adequate nephrographic phase shown. The lower aorta was arteriosclerotic and narrowed, and the origin of both common iliacs showed a similar change, the left being more marked than the right. The left internal iliac artery was occluded. The presence of atherosclerotic disease involving the aorta, and particularly the left common iliac artery, suggested a strong possibility that this process had extended up the left lateral wall of the aorta and was possibly involving the left renal orifice. In view of this, it was decided to have differential ureteric excretion studies undertaken.

Differential ureteric excretion. Right kidney: Volume 13·1 ml., urea 1·04 g.%, sodium 1·05 mEq. Left kidney: Volume 16·7 ml., urea 0·94 g.%, sodium 1·30 mEq. These studies showed no significant abnormality of function of either kidney.

Treatment. The patient was put on to a low-fat, low-cholesterol diet, supplemented with vitamin A (10,000 units

daily), intermittent heparin therapy (12,500 units a day intramuscularly for 8 weeks), and after this, oral anticoagulant treatment and nicotinic acid (1,000 mg. *t.d.s., p.c.*).

Present condition. The patient is doing a normal day's work. He is symptom-free, and has an average blood pressure of 160/100 mm.Hg. He is now due for a reassessment of his case.

General review. This patient is considered to be a case of atherosclerosis with aortographic evidence of atheroma involving the lower aorta, the vessels supplying the lower limbs, and possibly the left renal artery, the condition of which might have been clarified by selective catheterization. General symptomatic improvement, with a drop in blood pressure followed on medical treatment.

Case 5

Mrs. F.M.S., aged 49 years. This patient was first seen on 9 May 1958. She complained of recurring attacks of severe headache for some months, either frontal or occipital, and on occasions vomiting without relief of the headache; and of progressive loss of energy for 6 months. Before this illness she had enjoyed excellent health.

On examination. An anxious subject. Fundi within normal limits. Blood pressure about 230/150 mm.Hg. Peripheral vessels not thickened. Peripheral pulses present and equal. Heart not enlarged. Heart sounds closed and of good character. ECG within normal limits. Some tenderness over the right renal angle. Blood urea 30 mg.%. Chemical test for catecholamines negative. Blood count: Haemoglobin 19 g.%, PCV 59%; the blood picture was thought to be consistent with the diagnosis of commencing polycythaemia vera or a compensatory polycythaemia. Urine: Albumin, trace; centrifuged deposit showed presence of very scanty leucocytes; no epithelial cells, bacteria, red cells or casts. Urea clearance test, 40% of normal function. Intravenous pyelogram: Right kidney within normal limits; left kidney thought to be atrophied, with poor function.

Aortogram and nephrogram. Report: 'The right renal artery showed well-marked tortuosity, and a saccular aneurysm was seen arising from the lower division of the main renal artery just at its origin. In the left kidney at nephrographic stage a well-marked cortical blush was present, demonstrating the cortex of the left kidney, and at 10 minutes no excretion was seen from the left kidney. The findings were thought to be consistent with an aneurysm involving the right system, and possibly evidence of some atrophy of the left kidney as the result of chronic pyelonephritis'.

Progress. The patient was further observed and treated with hypotensive drugs, in particular serpasil and ansolysen by mouth, with very little progress. On 2 June 1958 the spleen became definitely palpable and a repeat blood count and blood-volume studies confirmed the diagnosis of polycythaemia vera. She was then treated with radio-active phosphorus and after a month's interval showed progressive improvement. In November 1958 she was symptom-free, blood pressure being about 140/100 mm.Hg, and the hypotensive drugs were discontinued. She continued to make progress and remained symptom-free, and has since been normotensive (average blood pressure 130/80 mm.Hg).

April 1959: Repeat intravenous pyelogram. Report: 'The spleen is much enlarged, and displaces the left kidney downwards. The calyceal outlines are not clearly demonstrated on the left side, but the concentration is sufficient to produce a dense shadow in the renal pelvis and upper part of the left ureter. The right kidney shows normal appearance.' At this time the haemoglobin was 13·1 g.% and the haematocrit 45%.

Summary and review. It is considered that this patient is a case of polycythaemia vera of insidious onset; and that before this became clinically obvious she developed renal thrombotic incidents, resulting in the Goldblatt phenomenon with a high blood pressure, which responded poorly to medical treatment until ultimately she became symptomatically well and clinically improved on therapy with radio-active phosphorus.

Case 6

Mr. R.W.C., aged 32 years, accountant. This patient was seen on 15 June 1950 when he was re-examined for bilharzia, which he had contracted when he was 14 years old and for which he had received 4 courses of treatment. At this re-examination a cystoscopy showed scattered bilharzial nodules indicative of old inactive bilharzia. At that time the intravenous pyelogram was normal except for one of the upper calyces in the left kidney, which was a little broadened and suggested the possibility of a slight abnormality in this kidney.

The patient remained well from July 1950 until early 1956, when he was referred as a case of high blood pressure of possible kidney origin, and in September 1956 a repeat intravenous pyelogram showed a large round tumour involving the upper calyces of the left kidney. At an exploratory operation of the left kidney a large cyst was found occupying most of the upper pole, and 3 smaller cysts lower down. The large cyst was enucleated and the small cysts were uprooted, leaving a healthy-looking kidney. After this operation the patient remained well and normotensive until February 1959, when he was reported to have a severe hypertension of 182/152 mm.Hg. A repeat intravenous pyelogram then showed evidence of a superficial mass projecting from the lower pole of the right kidney and minor changes in the pyelographic appearance of that kidney raised the suspicion of further small cysts within the renal substance. The left kidney showed evidence of generalized stretching and distortion of the calyces and a small crescentic calcification in the middle third of the kidney, and the renal pelvis filled poorly. Both kidneys showed evidence of some lobulation of the renal contours. The findings were considered to be consistent with bilateral polycystic disease of the kidneys.

Summary and review. This patient is considered to be a case of polycystic disease of the kidneys. This was difficult to diagnose clinically or on intravenous pyelogram in 1950, but 6 years later the intravenous pyelogram showed evidence of cysts involving the left kidney, and the hypertension was relieved by removal of the cysts, thus demonstrating the Goldblatt phenomenon. After a further interval of 3 years the hypertensive state returned, and the intravenous pyelogram then gave evidence of bilateral polycystic disease of the kidneys.

COMMENT

We have found that a careful history is helpful in selecting the cases which require detailed investigation, and the history of a known normotensive developing a sudden hypertension is useful. Most help has been obtained from aortography and by the differential kidney-function tests described by Howard and others. A successful outcome can only be decided by the result of surgery, which may consist of unilateral nephrectomy, endarterectomy of a renal artery, or a bypass graft to improve the renal circulation. These cases form a small fraction of all hypertensive patients, but in our experience they are definitely commoner than those due to phaeochromocytoma.

SUMMARY

The close association between unilateral renal ischaemia and hypertension, as reported by Goldblatt and others in 1934, and its diagnosis and treatment, are discussed. Stress is laid on the significance of sudden hypertension in a known normotensive subject.

Six case histories of hypertensive patients showing this feature are presented, 3 of them being cases of unilateral pyelonephritis successfully treated by nephrectomy. The other 3 cases are respectively (1) aortic atherosclerosis with a strong possibility of involvement of the left renal artery, (2) polycythaemia vera with renal complications, and (3) polycystic disease of the kidneys first involving the left kidney.

A tabular statement is also submitted of the chief features of these 6 cases.

Our thanks are due to colleagues who referred cases and who helped in the investigation of these cases. We are indebted to Dr. F. P. Reid for the notes on case 5 and to Messrs. H. C. Brayshaw and P. S. Olivier for the notes on case 6.

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