survival rate is approximately 1 in 5. This rate is markedly dependent on the extent of anatomical involvement of the lymphoma at presentation in this series, but in the previously reported series of Clifford (1966) this is not so. A possible reason for this discrepancy is sought in the different dosages of drugs used in the two centres.

This investigation represents a continuation and extension of the work initiated by Mr. D. Burkitt. We are grateful to Mr. Burkitt and Mr. S. Kyalwazi, who between them treated nearly all the cases reported in this series, for permission to follow up their patients. Special acknowledgement is due to both the past and the present pathologists at Makerere Medical School, and in particular to Dr. D. H. Wright, for carrying out the microscopy of the cases considered in this paper. We would like to thank Mrs. Barbara Wright (registrar, B.E.C.C.-financed Kampala Cancer Registry) for the assistance she gave us in collecting details of the patients. We gratefully acknowledge the financial support of the U.S. National Cancer Institute (N.I.H.) under their Contract No. PH-43-67-47, and of the British Empire Cancer Campaign for Research.

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Hypokalaemic Alkalosis and Hyperplasia of the Juxtaglomerular Apparatus without Hypertension or Oedema*

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Brit. med. J., 1967, **4**, 327–331

Hyperaldosteronism is commonly associated with either hypertension and hypokalaemic alkalosis as in renal vascular hypertension, malignant hypertension, and primary aldosteronism, or with oedema as in cirrhosis with ascites, the nephrotic syndrome, and chronic congestive heart failure. A dissociation between the hypertensive, potassium-losing, and continued sodium-retaining effects of aldosterone has previously been noted (Nelson and August, 1959) in patients who become oedematous. Three patients have been described with hypokalaemia, hyperaldosteronism, and hyperplasia of the juxtaglomerular apparatus but without hypertension or oedema (Bartter *et al.*, 1962; Boucher *et al.*, 1964; Ames *et al.*, 1965). The present case, like the other similar cases, apparently represents another instance in which aldosterone in excess has not resulted in hypertension. The cause of the syndrome described in these cases is unknown, but it has been suggested that it represents an important aberration from the normal relations between the renin-angiotensin-aldosterone system and blood pressure control.

Case Report

The patient was an 18-year-old negro male whose illness dates from infancy. He was delivered after a seven-month pregnancy and was well until 4 months of age, when he stopped growing. At 7 months he was admitted to the Children's Medical Center in Dallas, Texas, after two weeks' incessant vomiting. He was found to be grossly dehydrated, wasted, and drowsy. He was in opisthotonos, but Trousseau and Chvostek signs were negative. The testes were undescended and he had a first-degree hypospadias. Investigations at that time revealed a serum bicarbonate of 46 mEq/l., serum chloride 74.6 mEq/l., serum sodium 123 mEq/l., serum potassium 4.8 mEq/l., and a blood *p*H of 7.65. Idiopathic alkalosis was diagnosed, and he was initially treated with ammonium chloride, sodium chloride, and deoxycortisone without effect. He eventually recovered after subcutaneous fluids and oral feeding, and was discharged clinically well but with persistent hypochloraemic alkalosis.

He grew slowly, walked at 15 months, but at 4 years was noted to have polyuria and polydipsia. He was then reinvestigated and found to have one testis in the scrotum and a bone age of 3½ years. Hypochloraemic alkalosis was present with serum chloride of 87 mEq/l., serum bicarbonate 41 mEq/l., a blood *p*H of 7.59, and a creatinine clearance of 116.6 ml./min. He was given no further treatment at that time.

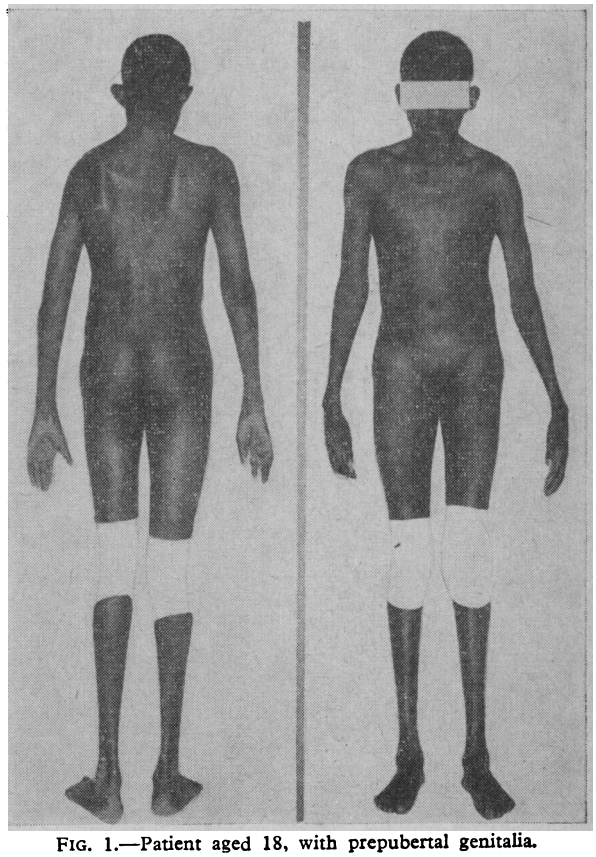

FIG. 1.—Patient aged 18, with prepubertal genitalia.

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He attended school, requiring special classes, and received no further medical treatment until the age of 17, when he suffered from recurrent dislocation of the patella. At 18 he presented to the endocrine clinic at the Los Angeles County General Hospital complaining of delayed puberty. His only other symptoms were constipation and diminished energy. He denied ingestion of drugs or having diarrhoea, and had no attacks of paralysis or tetany.

Physical examination showed him to be alert, with a eunochoid build and prepubertal genitalia (Fig. 1). The right testis was in the inguinal canal and was 2 cm. long; the left was in the scrotum and was 3 cm. in length. His height was 159 cm. in the 10–25th percentile and exceeded his span by 2 cm. His weight was 34 kg., which was below the third percentile for his age and height. Blood pressure ranged from 108/60 to 90/50 with no significant postural changes.

Preliminary investigations revealed a serum potassium of 2.6 mEq/l., serum bicarbonate 29 mEq/l., serum chloride 85 mEq/l., serum sodium 134 mEq/l., and blood urea 10 mg./100 ml.; alkaline phosphatase 6.5 and 6 Bodanski units/ml.; and serum magnesium 1.6 mg./100 ml. Serum cholesterol was raised on two occasions to 301 and 256 mg./100 ml. Serum bilirubin was 0.7 mg./100 ml. and bromsulphalein retention less than 5% in 45 minutes. Haemoglobin was 11–13.5 g./100 ml., with a normal film and a W.B.C. of 7,100, and normal differential. Bone age estimated by skeletal *x*-rays was 15–17 years. Skull and chest *x*-ray examination showed nothing abnormal. E.E.G. was normal. Buccal smear was negative for Barr bodies. E.C.G. showed changes of hypokalaemia with inverted and biphasic T waves and a prolonged Q–U interval. Urinanalysis showed a normal sediment with less than 100 mg. protein per 24 hours and no glucose (tested with Clinistix). Urine cultures were sterile. Creatinine clearance was 98 ml./min. Urine concentrating ability was impaired as shown by urine osmolality of 378 mOsm/l. after four hours' intravenous infusion of pitressin at a rate of 4 microunits/min. Urinary acidification was slightly impaired, the urine reaching a minimum *p*H of 5.9 after an oral ammonium chloride load of 0.5 g./kg. body weight (Wrong and Davies, 1959). Twenty-four-hour urine amino-acids totalled 179 and 490 mg./100 ml. with no distinctive pattern. An intravenous pylogram suggested some irregularity of the calices of the right kidney but was otherwise normal. Renal aortogram and ^{131}I hippuran renal scan were normal. Twenty-four-hour urine 17-ketogenic steroids and 17-ketosteroids were normal and there was a normal response to intravenous A.C.T.H. and metyrapone. Urinary gonadotropins were less than 6 units and 6–16 units/24 hours on two occasions. P.B.I. was 6.8 μg./100 ml. and a ^{131}I uptake was 14% in 24 hours, rising to 40% after thyroid-stimulating hormone.

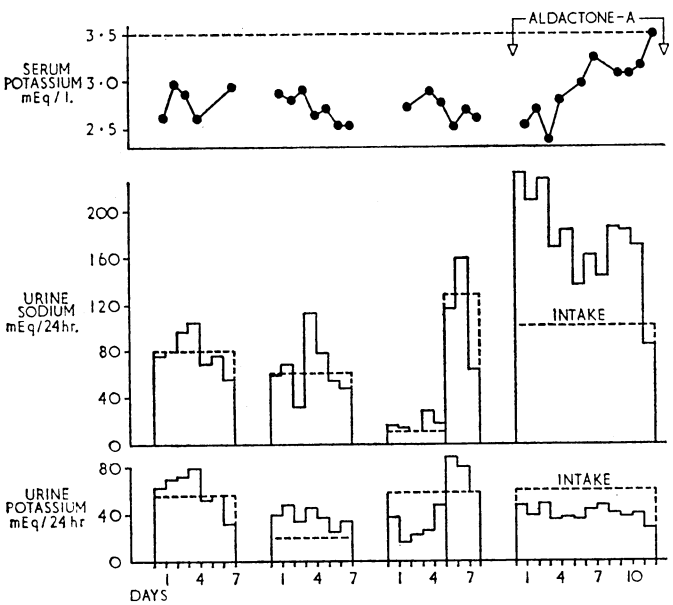

FIG. 2.—Upper graph: Solid line represents serum potassium; the horizontal dotted line indicates the lower limit of normal serum potassium. Middle graph: Solid blocks represent 24-hour urine sodium excretion; the horizontal dotted lines indicate daily sodium intake. Lower graph: Solid blocks represent 24-hour urine potassium excretion; the horizontal dotted lines indicate daily potassium intake. Four main balance periods are shown from left to right. The first two demonstrate a tendency to renal potassium loss in the presence of hypokalaemia. The third period shows potassium retention on a low sodium intake and increased potassium loss when the sodium intake was increased. The fourth period shows potassium retention and natriuresis induced by Aldactone-A (spironolactone) and a rise in serum potassium towards normal.

Balance Studies

Metabolic balance studies carried out in the metabolic unit of the Los Angeles County General Hospital were designed to determine the site and cause of potassium loss. Results are shown in Fig. 2. On a potassium intake of 60 mEq/day excretion of urinary potassium exceeded intake, and this urinary potassium loss persisted when dietary potassium was reduced to 20 mEq/day. Total exchangeable potassium at this time was 1,172 mEq compared with an expected normal of 2,000 mEq for a person of his size. When dietary sodium was reduced to 10 mEq/day, urinary potassium excretion fell below intake. Increasing the dietary sodium to 120–150 mEq/day then caused an immediate rise in urine sodium and return of negative potassium balance. These results were interpreted as evidence that the hypokalaemia resulted from renal potassium loss due to hyperaldosteronism, the potassium retention induced by the low sodium diet being due to insufficient sodium reaching the distal tubule to exchange with potassium under the influence of aldosterone. When spironolactone, 200 mg. daily, was administered, urine sodium increased to 140 to 230 mEq/day, potassium was retained, and for the first time the serum potassium rose toward normal (Fig. 2).

Twenty-four-hour urine aldosterone excretion was measured on several different occasions. Table I shows that there was only one elevated value during the initial hospital admission, but that two weeks after cessation of two months' spironolactone therapy values were raised. Aldosterone secretion rate was 231 μg./100 ml., which was a high normal value for adults on 100 mEq sodium diet and is probably high for a prepubertal individual weighing 34 kg.

TABLE I.—*24-Hour Urinary Aldosterone Levels*

Diet	Lab. A (N 3–32 mg.)	Lab. B (N 5–19 mg.)
60 mEq K	22	—
80 mEq Na	11	—
20 mEq K	6	17·4
	7	14·1
58 mEq Na	57	27·7
	2	—
2 weeks after 2 months of spironolactone	—	44
	—	45

Plasma Renin Activity

Plasma renin activity was measured[1] by a modified method of Boucher. The patient was studied initially while taking a low-sodium diet, a high-sodium diet, and after a period of spironolactone therapy. Samples were withdrawn after the patient was supine, after an overnight sleep, and again after four hours' standing. Table II shows that the values obtained in the patient were extremely high compared with normal subjects or patients with essential hypertension, renal hypertension, or primary aldosteronism. (Values given were ranges for each group.) Values for the patient were significantly higher on a low-sodium diet but showed no change after the period on aldactone.

TABLE II.—*Plasma Renin Activity (Dr. M. Crane). (mμg. Angiotensin II per 100 ml. Plasma)*

		Patient	Normals	Hypertensives
Low sodium diet	Supine	9,120	150–705 (9)	176–904 (19)
	Standing	12,200	574–1240	258–3440
High sodium diet	Supine	6,000	36–236 (11)	46–1120 (19)
	Standing	6,400	237–825	
Normal diet and spironolactone	Supine	5,400		
	Standing	4,310		

Values for the normals and hypertensives are ranges. The numbers of subjects are indicated in parentheses.

[1] Measured in the laboratory of Dr. Milton Crane.

Renal Biopsy

A percutaneous renal biopsy of the right kidney was examined by light microscopy.[1] Haematoxylin and eosin stains revealed cellular proliferation in a few glomerular loops in a small number of the glomeruli. The juxtaglomerular cells that make up the walls of the preglomerular portion of the afferent arteriole were greatly increased in number, causing a distinct cellularity of the vascular pole of many of the glomeruli.

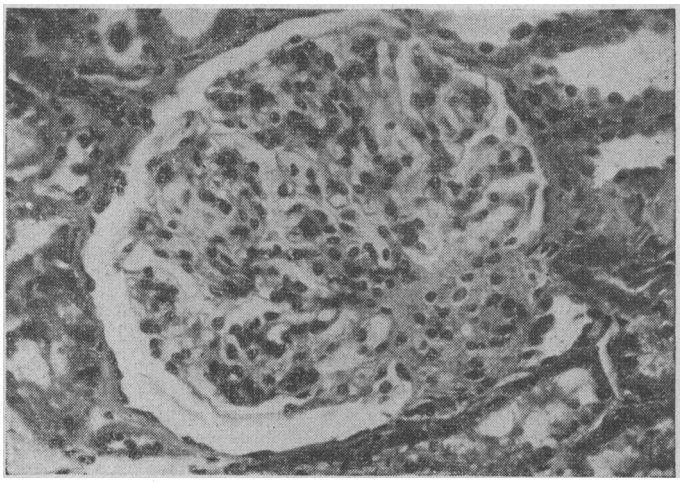

FIG. 3.—A glomerulus stained with haematoxylin and eosin showing increased cellularity of the vascular pole and cellular proliferation in a few capillary loops.

Periodic acid Schiff stained sections revealed P.A.S.-positive granules in the cytoplasm of the granular juxtaglomerular cells. The average number of granular juxtaglomerular cells per vascular pole was between 17 and 20, compared with four to six in normal individuals. Hence there was unquestioned hyperplasia of the granular juxtaglomerular cells. The granularity of each cell was more difficult to quantitate since human kidneys lack significant numbers of granules even in situations where the cells are known to have increased secretory activity.

Angiotensin Infusion Test

Synthetic angiotensin II amide was infused intravenously in a solution of 0.9% saline by means of an infusion pump to deliver

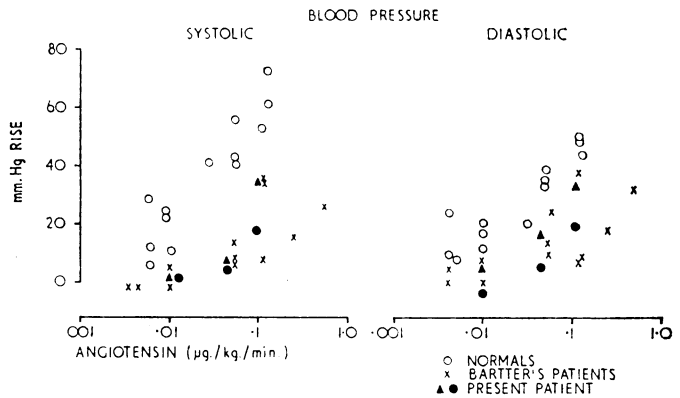

FIG. 4.—Effect of infusion of angiotensin II amide on blood pressure. Results in present case are superimposed on the data of Bartter *et al.* (1962). The latter are reproduced by kind permission of the *American Journal of Medicine*.

doses of 0.01, 0.05, and 0.2 μg./kg. body weight/minute. Blood pressures were recorded by sphygmomanometry during a basal period of 30 minutes and then at 60-second intervals for 8 to 10 minutes during each dose rate of angiotensin infusion. The mean systolic and diastolic blood pressures for each infusion rate were calculated and the increments in blood pressure above baseline levels were estimated. The experiment was performed on two occasions when the serum potassium was below 3 mEq/l. Fig. 4 shows these results superimposed on data obtained from the patients studied by Bartter *et al.* (1962). It can be seen that our patient shows grossly diminished pressor sensitivity to angiotensin compared with the normal subjects and similar sensitivity to that seen in the other two patients. Expressed according to the criteria of Kaplan and Silah (1964), our patient required 70–100 mμg. of angiotensin per kg. body weight per minute to obtain a 20 mm. Hg rise in diastolic blood pressure. This compared with 2.6–7.6 mμg. for normal subjects (Hocken and Kark, 1965). The normal pressor activity of the angiotensin used in our patient was confirmed in normal subjects.

The pressor response to noradrenaline was also studied by infusing noradrenaline at rates of 0.6, 1.2, and 3.0 μg./kg. body weight/minute. Average increments of 1.5, 7.2, and 24 mm. Hg in diastolic pressures were obtained for the respective doses of noradrenaline.

Discussion

The patient showed several features which suggested that he suffered from the same disease as the patients described by Pronove *et al.* (1960), Bartter *et al.* (1962), and Bryan *et al.* 1966—namely, hypokalaemic alkalosis, evidence of hyperaldosteronism, hyperplasia of the juxtaglomerular apparatus, increased renin secretion, and presumably increased angiotensin formation, normal blood pressure, absence of oedema, and relative insensitivity to pressor effects of exogenous angiotensin.

The variably elevated urinary aldosterone levels in our patient were thought to be due to the inhibiting effect that hypokalaemia itself has on aldosterone secretion (Cannon *et al.*, 1964 ; Gann *et al.*, 1964). The balance studies appear to provide good evidence that the hypokalaemia was due to an aldosterone effect and values were raised after a period of potassium repletion. It is noted that preliminary values of aldosterone excretion reported by Bryan *et al.* (1966) were also normal in their patient.

Plasma renin activity was grossly raised in our patient (Table II). Despite the high levels on the sodium intake of 120 mEq/day, it appeared that the kidneys were still capable of producing more renin in response to a low sodium intake.

The increased granularity of the juxtaglomerular apparatus (Fig. 3) demonstrated by light microscopy was similar to that seen in the other patients (Bartter *et al.*, 1962 ; Bryan *et al.*, 1966) and provided further evidence of increased renin production.

If is is reasonable to assume that the increased renin production resulted in increased angiotensin formation and consequently hyperaldosteronism and hypokalaemia in these patients, then two outstanding questions are posed. Firstly, what is the fundamental cause of the increased activity of the juxtaglomerular apparatus ? Secondly, why is the blood pressure normal in the presence of increased angiotensin and aldosterone production ? It has been suggested (Bartter *et al.*, 1962 ; Bryan *et al.*, 1966) that the prime abnormality is a decreased vascular sensitivity to angiotensin as demonstrated by the infusion of exogenous angiotensin—the implication being that renin production is increased to produce angiotensin, which directly maintains vasomotor tone. The hyperaldosteronism would then be an unwanted side-effect of this compensatory mechanism. This theory assumes that angiotensin is of physiological importance as a peripheral vasoconstrictor. The situation is not clear in this respect, but it is apparent that any physiological pressor effect is modified and mediated at least in part by electrolyte changes and aldosterone (Peart, 1965).

Ames *et al.* (1965) showed that prolonged angiotensin infusions in normal subjects were associated with increasing pressor sensitivity coincident with increased aldosterone production and sodium retention. On the other hand, a diminished

[1] Kindly reported by Dr. H. Demopoulos

pressor response to angiotensin has been described in both normotensive and hypertensive states where increased endogenous angiotensin circulates. For example, in the secondary hyperaldosteronism of cirrhotics (Ames *et al.*, 1965) and in some cases of malignant and renal hypertension (Kaplan and Silah, 1964), it has been suggested that the diminished pressor response to exogenous angiotensin in those conditions may be due to tachyphylaxis to the prolonged high levels of circulating angiotensin. Such tachyphylaxis might account for the pressor insensitivity seen in our patient. The absence of a simple alternative explanation for the increased renin production and decreased pressor response to angiotensin in these patients is presumably due to lack of knowledge of the precise mode of activation and functions of the renin-angiotensin system. It is therefore pertinent to examine two situations in which the renin-angiotensin-aldosterone system is known to be stimulated—namely, local abnormalities of renal blood flow and sodium depletion. Stemming from the experiments of Goldblatt *et al.* (1934), it has been shown that local disorders of renal blood flow may give rise to increased renin production and hypertension. A local abnormality of renal blood flow seems unlikely in our patient since hypertension was absent and the renal vasculature was grossly normal on aortography.

Sodium depletion also causes increased renin (Genest *et al.*, 1964 ; Brown *et al.*, 1964), angiotensin (Veyrat *et al.*, 1964), and aldosterone (Luetscher, 1956) production but without the development of either hypertension or oedema. It is therefore tempting to speculate that there is a tendency towards sodium depletion and thus extracellular fluid volume depletion in these patients. Abnormalities in extracellular fluid volume are not easy to establish, but if such a deficit were the stimulus to excess renin production then expansion of extracellular fluid or plasma volume should depress plasma renin activity, whereas if some other overriding stimulus were responsible then volume expansion would be less likely to affect renin output materially.

This theory was examined by measuring plasma renin levels before and after infusions of 0.9% saline and 6% dextran in 0.9% saline. The studies were carried out while the patient was on a constant sodium intake of 100 mEq/day. The two infusions were given four days apart and were each for four hours between 10 a.m. and 2 p.m. with the patient supine and fasted but without prior fluid restriction. Urine output and body weight were recorded every 20 to 30 minutes during the infusions. Plasma renin activity was measured immediately before and after each infusion. Plasma volume was estimated before and after the dextran infusion, using radioiodinated human serum albumin. Table III shows these results along with the volumes of fluid infused, the maximum weight increase during infusion, and the change in plasma volume after dextran. It can be seen that even when allowance was made for haemodilution plasma renin levels fell markedly with both saline and dextran infusions. The fact that at the end of the infusions renin activity, though drastically reduced, was still higher than normal may be due to the relatively short periods of volume

TABLE III.—*Effect of Fluid Volume Expansion of Plasma Renin Activity*

Infusion:	0·9% Saline	6% Dextran in 0·9% Saline
Plasma renin activity { Before ..	10,700 units	4,870 units
{ After ..	918 „	654 „
Volume infused	3,400 ml.	700 ml.
Maximum weight increase during infusion	1·2 kg.	0·5 kg.
Plasma Volume { Before		1·81 l.
{ After		2·36 „

expansion compared with the biological half-life of renin. Data on the latter are scanty. In the dog it has been estimated as 15 minutes (Fasciolo *et al.*, 1964) and is probably longer in man. With a half-life for renin of say 30 minutes and volume expansion for three of the four hours of infusion, initial values of 10,700 and 4,870 units should fall to 446 and 203 units, compared with the observed values of 918 and 654 units. If the infusions had been continued longer renin activity might well have fallen more.

It seems likely that the inhibition of renin activity with both infusions was mediated by changes in plasma volume rather than changes in extravascular volume. A total of 700 ml. of 6% dextran in saline caused plasma renin activity to fall to a similar extent to 3,700 ml. of saline, and whereas a rise in plasma volume is anticipated with both infusions, and a rise in interstitial fluid volume is expected with the saline alone, the observation that 550 of the 700 ml. of 6% dextran in saline could be accounted for by the rise in plasma volume (Table III) suggests that this infusion caused little interstitial volume expansion.

The demonstration that excess renin production could be inhibited by plasma volume expansion is compatible with the suggestion of an abnormality of fluid volume control in these patients, the increase in renin, angiotensin, and aldosterone acting in concert to retain sodium and to restore extracellular fluid volume to normal. Neither hypertension nor oedema would be expected in such a situation, and if fluid volume correction were inadequate blood pressure would tend to be low, as in these patients. A mechanism leading to subnormal extracellular or plasma volume still requires explanation. Impaired proximal tubular sodium reabsorption seems possible, as suggested by Laragh and Kelly (1964), in which case increased delivery of sodium to the distal tubule and exchange there of sodium for potassium would lead to the hypokalaemia observed.

Other features of this disorder which remain unexplained are the growth retardation and the glomerular damage. Growth retardation has been attributed to chronic hypokalaemia (Bryan *et al.*, 1966), and no better explanation is offered. Glomerular damage was widespread in the renal biopsies obtained in three of the patients. Light microscopy of the kidney in our patient showed capillary basement membrane thickening similar to that seen in many forms of glomerulonephritis. Despite this, there was no significant proteinuria. It is not clear whether these glomerular changes are related causally to the disease process or result from it. However, it has been shown that renin injections cause glomerular damage and proteinuria (Pickering and Prinzmetal, 1940), and it is possible that prolonged excessive release of endogenous renin might have a similar though less marked effect.

Management of our patient has been on the basis of spironolactone therapy to increase the serum potassium with attempts to administer up to 400 mg. a day, and though the drug is taken irregularly the serum potassium remains at 3.2–3.5 mEq/l., which is higher than the levels originally observed (Bartter *et al.*, 1962). The blood pressure has not changed significantly despite the initial saluresis caused by spironolactone. The patient appears to be more alert and active but he is receiving testosterone injections, which may influence this impression. No surgery is contemplated, since two of the three other patients had subtotal bilateral adrenalectomy with no immediate or long-term effect on their hypokalaemia and no symptomatic improvement.

Summary

A disorder characterized by hypokalaemia, evidence of hyperaldosteronism, increased plasma renin activity, increased granularity of the juxtaglomerular apparatus, glomerular abnormalities, normal blood pressure, and decreased pressor response to exogenous angiotensin is described in a non-oedematous 18-year-old prepubertal negro youth and is compared with a similar condition described in three other patients (Pronove *et al.*, 1960 ; Bartter *et al.*, 1962 ; Bryan *et al.*, 1966).

The pathogenesis of the syndrome is discussed and evidence for an abnormality of extracellular fluid volume control is presented.

We wish to acknowledge the invaluable assistance of the Metabolic Unit Staff at the Los Angeles County General Hospital in studies of this patient.

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Total Parathyroidectomy in Treatment of Secondary (Renal) Hyperparathyroidism

C. S. OGG,* M.B., B.SC., M.R.C.P.

Brit. med. J., 1967, **4**, 331–334

Recent improvements in the care of patients with chronic renal failure have revealed that symptoms attributable to renal glomerular (uraemic) osteodystrophy develop in a considerable number of cases.

The various components of this bone disease include rickets or osteomalacia, osteitis fibrosa, and osteosclerosis. These may occur in almost pure forms but more commonly appear in varying combinations in different patients.

Treatment with large doses of vitamin D (Liu and Chu, 1943 ; Dent *et al.*, 1961 ; Stanbury and Lumb, 1962) is often successful in promoting healing of the skeleton, but is dangerous because of the ease with which it is possible to produce hypercalcaemia and metastatic calcification. This danger is greatest in those patients who have osteitis fibrosa as the dominant skeletal lesion, and who, unlike the majority of patients with renal failure who have low plasma calcium levels (de Wesselow, 1923), have plasma calcium levels that are either within the normal range (Stanbury and Lumb, 1966) or even frankly raised (Hubbard and Wentworth, 1921 ; Smyth and Goldman, 1934 ; Shelling and Remsen, 1935).

Stanbury *et al.* (1960) were the first to embark deliberately on a policy of subtotal parathyroidectomy in a patient with severe secondary hyperparathyroidism, and they were subsequently able to heal the bone lesions with vitamin D without producing hypercalcaemia. Since then further reports have appeared in this country (Findley *et al.*, 1961), in South Africa (Stables *et al.*, 1964), and in the United States of America (Anderson *et al.*, 1963 ; Fordham and Williams, 1963 ; Golden *et al.*, 1965 ; Felts *et al.*, 1965 ; Wilson *et al.*, 1965). These reports include a total of only 12 cases, and it is considered worth while to report four more.

Case 1

A 43-year-old man presented in 1961 with severe chronic renal failure (plasma urea 210 mg./100 ml.). No cause for this was found, and he was treated with a high fluid intake, a low protein diet, and sodium bicarbonate 2 g. daily.

During the following year he developed radiological changes of hyperparathyroidism in his hands, but this caused no symptoms until 1964, when he felt severe pain in both shoulders. His plasma calcium had risen to 10.5 mg./100 ml. (Fig. 1), with an ionized fraction of 5.0 mg./100 ml. and a complexed fraction of 1.3 mg./100 ml. (normal range 4.9–5.8 and 0.0–0.7 mg./100 ml. respectively (L. Watson, personal communication, 1967). *X*-ray films showed progression of the subperiosteal erosions of the phalanges and the appearance of erosions of the lateral ends of both clavicles.

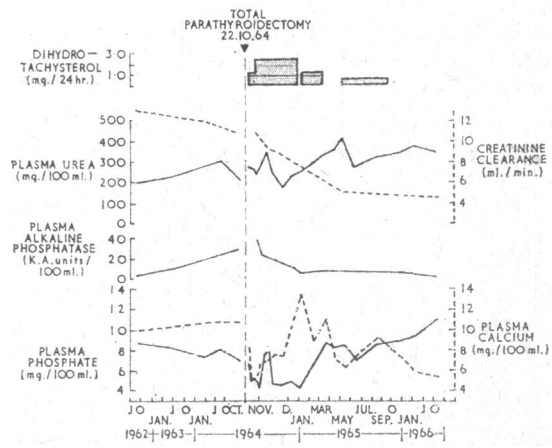

FIG. 1.—Case 1. Biochemical progress. Note changing time scale.

It was also noted that there was a considerable amount of calcification in the internal iliac arteries. Because of the metastatic calcification total parathyroidectomy was recommended, and four hyperplastic glands were removed. Preoperative and postoperative findings are shown in Tables I and II.

The plasma calcium fell to a minimum of 4.9 mg./100 ml. on the fifth postoperative day, and symptoms of tetany were controlled with intravenous calcium gluconate and oral dihydrotachysterol. Within a month he had lost his bone pain and was discharged

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