Thus increased osmolality and low oxygen tension, both of which are factors favourable to sickle-cell formation, obtain in the renal medulla.

In view of the possibility of further bleeding from the opposite kidney, conservative measures should be adopted in the management of such patients. Immergut and Stevenson (1965) believe that bleeding from the necrotic areas in the papillae may be perpetuated by excessive fibrinolysis of the clot. They treated three patients with massive haematuria due to the sickle-cell trait with epsilon-amino-caproic-acid to inhibit this excessive fibrinolytic activity. In all three patients haematuria ceased within three to seven days of starting therapy, and in one patient in whom bleeding recurred one week later a second course of the drug was again effective. Certainly in our own case the correction of a "prothrombin" deficiency by the administration of vitamin K, the administration of epsilon-amino-caproic-acid, and blood transfusion were followed by a cessation of bleeding.

In a personal series of 27 cases presented by Lucas and Bullock (1960), 23 were managed conservatively. In 22 of these bleeding ceased with bed-rest alone. Nephrectomy was undertaken on four patients on account of recurrent and persistent haematuria. These authors consider that the indications for nephrectomy in this condition are continued severe haematuria, transfusion reactions, and strong suggestions of malignancy.

Dees (1965) favours renal pelvic tamponade as a means of controlling haematuria. Two of the patients whom he reports had sickle-cell disease, and their bleeding ceased when the ureter was completely obstructed for 24 to 48 hours by an indwelling tapered ureteral bougie. One of these patients had a recurrence of bleeding a week later. In view of the possible role of anoxia as a cause of this condition, he was placed in a hyperbaric oxygenation chamber at two-atmospheres pressure for one hour. Bleeding ceased within two hours.

It is felt that the diagnosis in our own case might have been made at an earlier stage had the haematologists and pathologists concerned known that the patient was an immigrant West Indian. In view of the large numbers of such patients presenting it is suggested that this fact should be recorded in the details sent by the clinician to the laboratory.

The severity of the haematuria in this condition makes it likely that such patients will be referred to surgeons. We therefore have no hesitation in advising that this diagnosis should be considered early in the investigation of massive haematuria in a susceptible patient.

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Renal Potassium Wasting in Hypercalcaemia

Brit. med. J., 1967, **1**, 679–680

The adverse effects of hypercalcaemia on renal function are well known. Urea retention, accompanied by a failure to form concentrated urine, has been described in hypercalcaemia due to hyperparathyroidism (Cohen *et al.*, 1957), calciferol intoxication (Danowski *et al.*, 1945), sarcoidosis (Dent *et al.*, 1953), osteolytic bone metastases (Sanderson, 1959), and milk-alkali syndrome (Wenger *et al.*, 1957). Attention has also been drawn to an impaired renal excretion of acid and ammonia in hypercalcaemia (Fourman *et al.*, 1960 ; Ferris *et al.*, 1961). In addition, renal potassium wasting may occur, somewhat rarely, in such cases (Ferris *et al.*, 1961). This paper describes a case of hyperparathyroidism which was complicated by excessive renal potassium loss and severe hypokalaemia.

CASE REPORT

A man of 28 sustained a fracture of the left femoral shaft. He was admitted to hospital, and after reduction of the fracture an intramedullary nail was inserted. The operation was followed by persistent vomiting. Nine days after admission he complained of pain at the fracture site and the leg was found to be shortened and externally rotated. *X*-ray examination showed disintegration of the bone at the fracture site and partial extrusion of the intramedullary nail. Metabolic bone disease was suspected and the serum calcium was found to be 14.9 mg./100 ml.

At this stage further questioning revealed the following additional symptoms: loss of weight (20 lb. (9 kg.) in nine months), increasing lassitude and fatigue for nine months, thirst and polyuria for six months, anorexia and occasional vomiting for six months, and vague

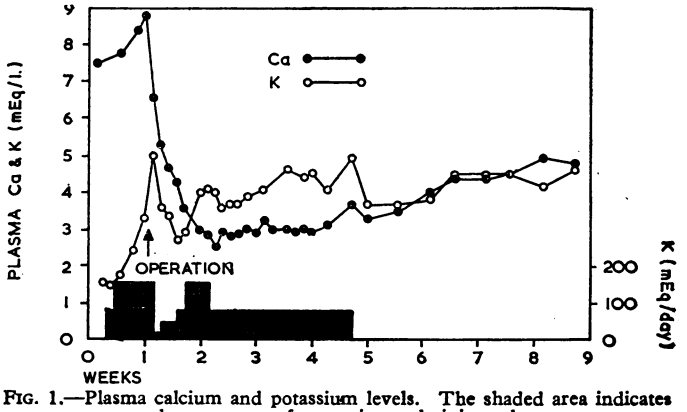

FIG. 1.—Plasma calcium and potassium levels. The shaded area indicates the amounts of potassium administered.

intermittent pains in the limbs for an indefinite period. *X*-ray examination of the abdomen showed scattered small opacities in the renal areas, but these did not suggest the presence of calculi in the renal pelvis. *X*-ray films of hands, skull, and pelvis showed the typical changes of hyperparathyroidism, and exploration of the

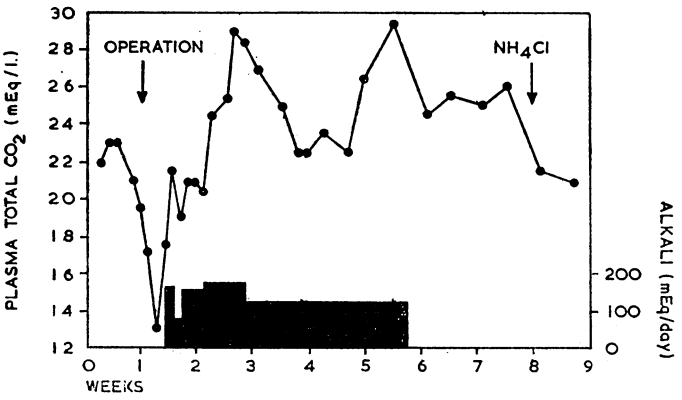

FIG. 2.—Plasma total CO_2 levels. The shaded area indicates the amount of alkali administered.

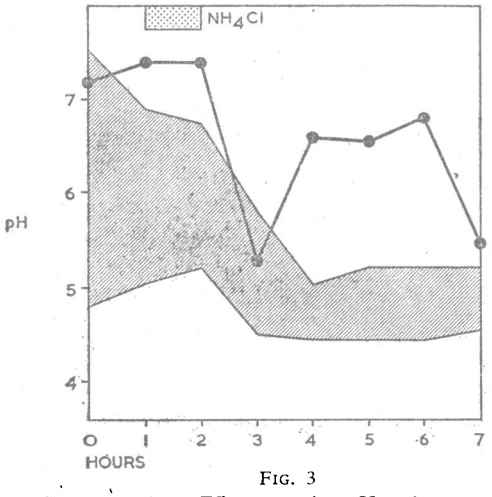

FIG. 3

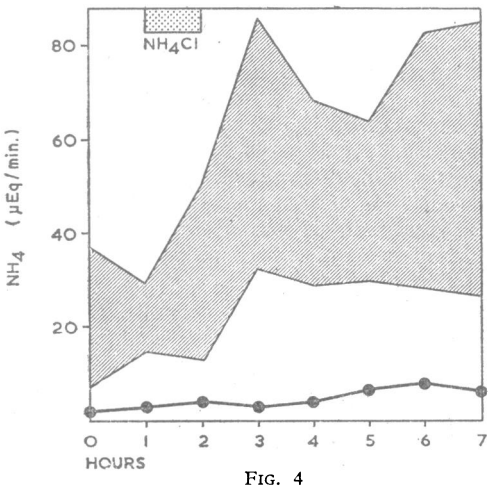

FIG. 4

FIGS. 3 and 4.—Effect on urine *p*H and ammonia excretion of a test dose of ammonium chloride. The shaded areas indicate the limits of normal response (Wrong and Davies, 1959).

neck (Mr. K. Owen) revealed a large solitary parathyroid adenoma measuring 2.4 by 1.8 by 1.8 cm. Removal of the adenoma led to a precipitous fall in serum calcium and ultimately to satisfactory union of the fracture.

Plasma and Urine Potassium.—The plasma electrolytes and urea before corrective treatment were as follows: sodium 127, potassium 1.6, chloride 86.5, and total CO_2 22 (all mEq/l.); urea 77 mg./100 ml. Two days later, after 240 mEq of potassium had been given, the plasma potassium was still only 1.8 mEq/l., but the 24-hour urine output of potassium was 36 mEq; next day, with the plasma level at 2.5 mEq/l., the urine output was 77 mEq. These figures indicate gross derangement of the renal potassium-regulating mechanism. Because of persistently low serum levels, potassium replacement was continued for four weeks after operation. The patient now appeared to be able to regulate his plasma potassium level satisfactorily without potassium supplements. Fig. 1 shows the changes in plasma potassium and calcium and the amount of potassium supplement given.

Acidosis.—Two days after removal of the parathyroid adenoma the plasma total CO_2 had fallen to 13 mEq/l. No plasma *p*H measurements were made at this stage, but there was no hyperventilation, and it seems legitimate to infer an acidosis. The urine *p*H was not measured at this point. Alkali supplementation was given as shown in Fig. 2. Seven weeks after removal of the adenoma a standard ammonium chloride loading test (Wrong and Davies, 1959) was performed, the results being shown in Figs. 3 and 4.

DISCUSSION

The severe potassium depletion which developed in this patient was at least partly due to a failure of the kidneys to conserve potassium. No doubt the antecedent anorexia and vomiting also contributed by curtailing potassium intake. The combination of renal potassium wasting together with azotaemia, loss of urine-concentrating power, and failure to excrete acid and ammonia forms a picture which strikingly resembles that described by Ferris *et al.* (1961) in a boy suffering from vitamin-D intoxication. Their suggestion that this spectrum of disturbances of renal function may be a rare consequence of hypercalcaemia from any cause is supported by the present case. The opacities seen in the *x*-ray films of the renal areas suggest that nephrocalcinosis and calcific lesions, of the type illustrated in the paper by Ferris *et al.* (1961), may well have been present.

Ferris *et al.* (1962) showed that the normal renal conservation of potassium which occurs after feeding a potassium-deficient diet to rats is interfered with if the animals are made hypercalcaemic by injections of vitamin D. They also showed that the effect was not abolished by prior adrenalectomy. In the present case the urinary sodium/potassium ratio at the height of the potassium depletion was between 2 and 3, and it seems unlikely that excessive aldosterone secretion could have caused the loss of potassium. Specific interference with the renal tubular functions studied, caused by the hypercalcaemia, seems to be the likeliest explanation.

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