showed that paralytic attacks in the familial form were associated with a low level of potassium in the serum. Hypopotaemia has also been described in attacks of thyrotoxic paralysis, but both in the familial form and in thyrotoxic paralysis attacks may occur with a normal level of serum potassium. This does not, however, exclude the possibility that a fall in the serum potassium level from a normal to a low normal level has taken place before the paralysis has become manifest. Grob, Liljestrand, and Johns (1957) and Zierler and Andres (1957) have shown that attacks of familial periodic paralysis are associated with a shift of potassium from serum into muscle, and that during recovery there is a leakage of potassium from muscle. Therefore it has been suggested that the attacks are initiated by an abnormal uptake of potassium by muscle, thus causing an increase in the ratio of intracellular to extracellular concentration of this ion, with consequent hyperpolarization of the muscle membrane and reduced responsiveness to nerve stimulation (Grob, Johns, and Liljestrand, 1957).

We believe that a similar mechanism may be at work in thyrotoxic paralysis. The patient always recovered from attacks without administration of potassium and with a rise of the serum potassium level to normal value. This would indicate a redistribution of body potassium.

The electrocardiographic changes showed typical signs of hypopotaemia during paralytic attacks, with concomitantly low levels of potassium in the serum. However, when the patient was up and walking about the next day, the level of serum potassium was normal, whereas the electrocardiogram still showed changes suggesting hypopotaemia (Figs. 1 and 2). This shows that the electrocardiogram may be misleading in evaluating the level of serum potassium. Van Buchem (1957) has suggested that the shift of potassium into muscle in familial periodic paralysis might give rise to a withdrawal from the myocardium. A relatively slow restoration of intracellular cardiac potassium may then offer an explanation of the persistence of hypopotaemic signs in the electrocardiogram in spite of a normal serum level.

In familial periodic paralysis attacks may be brought on by giving insulin, glucose, or adrenaline. In the present case there was a normal response to a glucose-tolerance test, and the comparatively large amount of glucose ingested did not bring on any paralysis, nor was there any association between the patient's dietary habits and the frequency of attacks.

We have the impression that the patient benefited from the treatment, but the observation period is, of course, too short to permit any definite conclusions.

Summary

A case of hyperthyroidism associated with attacks of flaccid paralysis is reported. During the paralytic attacks the level of serum potassium was found to be below normal, and the electrocardiogram showed signs of hypopotaemia. The patient recovered spontaneously from the attacks, the serum potassium level reaching normal values.

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Medical Memoranda

Syndrome of Nutritional Siderosis in an Indian in Durban

A liver-biopsy survey of 200 adult male Africans in the King Edward VIII Hospital, Durban, revealed that hepatic siderosis was present in 88% (Gillman *et al.*, 1957, 1959). In the patients with advanced siderosis the following clinical features were common: (1) *Grey-black pigmentation*, particularly of the skin of the face. We have called this appearance the "coal-heavers' look." (2) *Alopecia* of the outer third of the eyebrows and of the axillae and pubis, the latter resembling the female escutcheon. (3) *Testicular atrophy*, varying from a distinct loss of tension of the gland to an almost complete absence of testicular tissue. The degree of involvement was not necessarily the same in both testes. This atrophy was often associated with impotence. (4) *Hepatomegaly*, the liver edge being firm, and in the presence of frank cirrhosis also irregular. (5) *History of poor diet*, consisting primarily of maize products with only occasional supplements of meat, vegetables, and dairy products. These supplements were available not more than once a week. (6) *Consumption of appreciable quantities of "kaffir-beer"* (the traditional brew of the African). This liquor has been shown to have a high iron content (Walker and Arvidsson, 1953).

The above clinical picture has been encountered in this hospital almost exclusively in African patients as distinct from Indians. The present case is therefore being reported because the patient, an Indian male, showed certain of the clinical features which we have so far encountered only in African siderotics. A diagnosis of nutritional hepatic siderosis was made on clinical grounds and was subsequently confirmed by liver biopsy.

CASE REPORT

An Indian man aged 28 was referred to one of us in the out-patient department of King Edward VIII Hospital in May, 1958. He had exhibited psychotic symptoms, and the doctor in charge of the medical services of the sugar company for which the patient worked described him as "incoherent and forgetful." He had been referred to the hospital for the exclusion of any organic disorder prior to certification.

His wife described a similar episode occurring in 1956 at roughly the same time of the year (March—that is, autumn). She had five children, the youngest being 5 months old. After the birth of the last child the patient had not resumed sexual relations; this the wife described as "unusual."

Examination revealed a well-built co-operative Indian man. His expression was somewhat facile. He had the facial pigmentation characteristic of siderosis in Africans ("coal-heavers' look"), and this was associated with a mild facial dyssebacea. Hair distribution was normal; the testes were within normal limits. His liver was palpable two fingerbreadths below the right costal margin, and the edge was firm and smooth. The diet was primarily maize, with bread and rice as occasional supplements, instead of the traditional rice diet of the South African Indians. He ate meat only once a week, and his food was cooked in *aluminium* pots. He regularly drank "kaffir-beer" (supplied by the sugar company) in appreciable quantities, but did not touch other alcoholic liquors.

It was recommended that he be confined to bed and his diet rearranged. He was warded for three weeks and was given double hospital rations. A month after his first visit to this hospital he returned and appeared mentally normal. The dyssebacea of his face had cleared and he volunteered that he felt a sense of well-being. Two months later he reported back. He was well and was subjected to liver biopsy. In addition his plasma iron levels were determined on three separate occasions.

Laboratory Findings.—Haemoglobin, 17.2 g./100 ml.; P.C.V., 53%; M.C.H.C., 32%; white-cell count, 12,000 per c.mm. with normal differential; serum proteins, 7.2 g./100 ml. (albumin 3.2 g., globulin 4 g.); serum alkaline phosphatase, 2 King-Armstrong units; zinc turbidity, 13 units; thymol turbidity, 4 units; fasting blood sugar, 85

mg./100 ml.; Wassermann reaction negative. A radiograph of the chest was negative.

Plasma Iron.—Plasma iron/total iron-binding capacity: 120/391, 195/399, 200/397 µg./100 ml.

Liver Biopsy Findings.—A good core of tissue showed large amounts of iron-containing pigment as fine granules along the biliary poles of most of the liver cells in all lobules (Fig. 1). Coarser granules of iron were also present, especially in the periportal liver cells (Fig. 2) and in many of the portal phagocytes (Figs. 1 and 2). Only small amounts of iron were present in the Kupffer cells (Fig. 2). The portal tracts were distinctly fibrotic (Fig. 3), and, although there was some linking of the portal tracts by fibrous tissue, there was no architectural distortion. Conclusion: diffuse hepatocellular and portal siderosis with moderate portal fibrosis but no cirrhosis.

Analysis of Diet.—Samples of the patient's food and a specimen of the "kaffir-beer" were subjected to chemical analysis; the findings were as follows:

	Iron	Calcium	Phosphate
Diet (mg./100 g.)	2	11	16.2
Kaffir-beer (mg./100 ml.)	4.8	4.5	6.7

COMMENT

The clinical picture in this case corresponded closely to that which we have encountered in our African siderotics. It confirmed for us the usefulness of these physical signs in a patient of a racial group in whom hepatic siderosis is thought to be uncommon.

An analysis of the diet revealed a low iron content in the food but a higher iron content of the kaffir-beer. It is of interest also that this patient's food was cooked in aluminium pots. We are coming round to the view that the source of the excess iron absorbed from the diet is the kaffir-beer, and if taken in conjunction with a poor diet would result in hepatic siderosis. We have found that advanced siderosis in our 200 male Africans was more common in kaffir-beer drinkers. In this connexion it is of interest to note that the third plasma iron value (200 µg. per 100 ml.), which was the highest of the three determinations, was obtained only a week after a heavy bout of kaffir-beer drinking. The raised T.I.B.C. is also consistent with our findings in African siderotics (Gillman *et al.*, 1957).

The fact that the patient's diet was closely similar to that of the African may therefore be significant in pointing to the nutritional basis of this condition. This possibility is in conformity with our experimental findings that the composition of the diet as a whole, apart from its iron content, plays an important part in determining iron absorption and its deposition in body tissues (Gillman *et al.*, 1958).

We record our gratitude to Dr. H. M. Fyvie for his co-operation in this case; we thank Dr. P. A. S. Canham for the chemical determinations, and Professor Adams and Dr. S. Disler for permission to publish.

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FIG. 1

FIG. 2

FIG. 3