quote from the records of patients under their charge, and to Dr. J. B. Randell, Dr. H. F. Osmond, and Dr. L. Ford for their help.

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FAMILIAL PERIODIC PARALYSIS

REPORT OF A CASE

BY

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Familial periodic paralysis presents a well-defined clinical picture. It was first described by Cavaré (1853), whose findings have been supplemented by studies of the electrocardiogram (Janota and Weber, 1928), of the electroencephalogram (Watson, 1946), and of rare features such as non-familial incidence, protracted seizures, visceral involvement, and fatal complications (Smith, 1939). Precipitation of attacks has been ascribed to physical exertion and mental strain, indulgence in carbohydrates or alcohol, alimentary disturbance such as overeating, hunger, diarrhoea, or constipation, and exposure to cold or damp. Postulated relationships between this and other conditions—e.g., endocrinopathy (Shinosaki, 1926), migraine (MacLachlan, 1932), muscular dystrophy (Biemond and Daniels, 1934), spinal vascular spasm (Vernon, 1936), chemical defect of the neuraxis (Pudenz, McIntosh, and McEachern, 1938)—are inconstant and indefinite.

The search for an adequate pathology was initiated by suspicion that muscular metabolism was deranged by some transitory autotoxaemia, which was suggested by arrest of gastric digestion and motility during the palsy (Mitchell, Flexner, and Edsall, 1902), and by transient urinary anomalies—e.g., acetonuria (Pastine, 1918), albuminuria (Shinosaki, 1926), and creatinuria (Ferrebee, Atchley, and Loeb, 1938). Recent studies of the blood chemistry of this syndrome have led to a fundamental advance in the state of knowledge as it existed between the review of the literature made by Singer and Goodbody (1901) and that by Schoenthal (1934).

From a casual observation that the potassium content of the blood serum was lowered during the palsy (Biemond and Daniels, 1934) there developed a new line of inquiry (Walker, 1935). A low serum potassium was found in attacks induced by administration of glucose in large quantities and insulin, separately and together (Aitken, Allott, Castleden, and Walker, 1937), and by adrenaline injection (Allott and McArdle, 1938). Attention has been drawn to alterations in the phosphorus balance of the blood (Milhorat, 1937; Allott and McArdle, 1938), variations in electrolyte features of the blood (Ferrebee *et al.*, 1938), and experimental evidence suggestive of a central controlling factor (Pudenz *et al.*, 1938). The pathogenesis of this disease presumably depends upon an excessive call for potassium in the muscles, its consequent withdrawal from the blood and retention in the body, with parallel changes in phosphorus metabolism. Undue lability of the serum potassium and phosphorus in relation to sugar mobilization perhaps conditions some motor dysfunction, such as interference with transmission of nerve impulses or inhibition of the muscular contractile response.

It has long been recognized that this palsy may be averted or arrested by the ingestion of various potassium salts—e.g., the acetate (Singer and Goodbody, 1901), the carbonate, the chlorate (Mitchell *et al.*, 1902), the bromide (Holtzapfel, 1905), the chloride (Aitken *et al.*, 1937), the citrate (Herrington, 1937), and the phosphate (Allott and McArdle, 1938). The success of prophylactic potassium medication at night (Allott and McArdle, 1938) is probably related to nocturnal lowering of serum potassium in this malady (Gammon, Austin, Blithe, and Reid, 1939). Symptoms have been controlled also by choline esters (Pudenz *et al.*, 1938), pilocarpine (Guttmann, 1931), and pancreatic extract (Vernon, 1936), and averted by cod-liver oil (G.D.H., 1948).

The following case, which illustrates typical familial and clinical features of this rare and remarkable malady, shows a prominent association between muscular exertion and paralytic seizures, a striking hemiplegic distribution of symptoms, and a prompt response to potassium therapy.

Case Report

A South Indian male, aged 24, of a rice-eating race, serving in a labour corps in Ceylon, was referred for neuropsychiatric examination with a history of transient flaccid hemiplegic seizures occurring early in the morning after a hard day's work. A provisional diagnosis of hysteria had been suggested by the relation of these attacks to heavy work, their transitory duration, apparent response to bromides, irregular recurrence, and increasing frequency.

Personal History.—Symptoms had originated at the age of 17 with transient weakness of the lower limbs after strenuous work. As increasing frequency and severity of the disability began to interfere with his work as a cultivator, he had sought *ayurvedhic* treatment (herbals and massage), with apparent relief for several months. After resuming work, however, several relapses compelled him to abandon manual labour and obtain light work in a rural grocery store. This was followed by freedom from the disability. Enlistment in the Indian Army, six months before the time of examination, had been followed by another recurrence of symptoms, this time ranging in intensity from fleeting muscular weakness to profound loss of power lasting 2½ days. The frequency of attacks had increased from one in six to eight weeks to one in two to three weeks, having almost always affected both limbs of the right side, rarely having spread to the opposite side, and never to the trunk, neck, or head. The palsy evidently used to come on insidiously during sleep at night, as it would become apparent on awaking next morning without any premonitory warning. Spontaneous recovery had been the invariable termination of these seizures. Other past illnesses comprised two attacks of intermittent fever suggestive of malaria, and a few diarrhoeic episodes.

Family History.—Six other members of the patient's family, who were of coolie or labourer stock, had been afflicted by disabilities similar to his own, as indicated in the accompanying family tree. A maternal uncle, U, had suffered from occasional paralysis of his legs until his death, by accident, at the age of about 40. A maternal aunt, A, had had intermittent loss of power of her legs, but had gradually become free of her disability after about 25 years. This woman's son, C, was subject to paralytic attacks in his right arm and leg. The patient's elder sister, S, used to suffer from intermittent loss of power of her upper limbs, and one of her daughters, N, had a similar weakness of her legs. The patient's younger brother, B, had died in childhood during a para-

Members of family affected by periodic paralysis are shown in black.

lytic seizure of all four limbs and body. The patient, P, had been married for about three years. His only child had died of convulsions eight days after birth, but had exhibited no manifestations of paralysis. No information was available regarding the patient's maternal grandparents. No paralytic disability was known to have occurred in his father's family.

Investigations.—Observation in hospital for a fortnight revealed no physical abnormality; the urine, faeces, blood, and spinal fluid were normal. Psychiatric investigation disclosed no evidence of a psychogenic basis for the palsy.

As the classical features of familial periodic paralysis were recognizable in the history, attempts were made to induce symptoms. Four ounces (114 ml.) of glucose was given by mouth at 1 p.m. and again at 7 p.m. Weakness of the right arm and leg was reported the next day at 5 a.m., and of the left arm and leg at 6.15 a.m. By 10 a.m. all four limbs were in a state of flaccid paralysis with extinction of reflexes. Paralysis was profound on the right side, but weak movements of the left hand and foot indicated incomplete involvement of the distal muscles of the limbs on that side. Faradic reaction was abolished on the right side and feeble on the left. There was no motor disturbance of the trunk, neck, or head. The cranial nerves, abdominal reflexes, sphincter control, and sensory functions were normal. Urine was voided normally during the paralysis, but the bowel was not evacuated. Transient, mild glycosuria (0.02%), high acidity, and excess of urates were observed during the palsy. Results of systemic examination were otherwise negative. After a static period of about 26 hours, gradual return of power began distally and progressed to complete recovery in about 11 hours. Reflexes were restored later, but remained sluggish for about 18 hours, with coexistent aching and tenderness of the limbs. Four days later an attempt was made, without success, to provoke the palsy by subcutaneous injection of 10 min. (0.6 ml.) of adrenaline, repeated half-hourly from 9 a.m. to 3 p.m.

A spontaneous attack developed a week later, following exercise, the limbs of the right side only being affected. When paralysis was maximal 1 dr. (3.5 ml.) of potassium citrate in peppermint water was given by mouth. Symptoms passed off rapidly, with complete recovery in 3½ hours, leaving slight post-paralytic myalgia for about two hours.

As the patient's seizures were invariably nocturnal in incidence, a dose of ½ dr. (1.75 ml.) of potassium citrate was ordered as a routine prophylactic measure every night. No further recurrences were reported thereafter.

It was unfortunate that wartime conditions precluded biochemical study of this case—e.g., estimations of potassium, phosphorus, and sugar in the blood. I am not aware of a previous record of periodic paralysis affecting several members of an Indian family. The first such instance reported in a family in Britain is accredited to Farquhar Buzzard (1901). One of his three patients has recently described his symptoms, mentioning a persistent attack which had crippled him for three weeks (G. D. H., 1948).

I am grateful to Dr. C. Worster-Drought for his helpful comments and suggestions regarding this paper. He has kindly sent me an interesting note about one of three members of a family afflicted by this palsy who invariably develops attacks after bowling three or four overs at cricket, but never after batting or fielding. I am obliged to my colleague, Captain Oliver Medonza, for assistance in searching the literature available in Ceylon for references to this interesting syndrome.

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Medical Memoranda

Some Presenting Symptoms of Carcinoma of the Bronchus

Carcinoma of the bronchus may give rise to a variety of presenting symptoms due either to the primary growth or to secondary deposits. The table shows the mode of presentation in 17 cases seen during a six months' appointment as house-physician at the Essex County Hospital. In these unselected cases from the medical and surgical wards of a general hospital nearly half presented with symptoms caused by metastases.

Presenting Symptoms

(i) Caused by Primary Growth	No. of Cases
Cough, haemoptysis, blood-stained sputum, pain in chest, dysphagia, superior vena caval obstruction	9
(ii) Caused by Primary Growth but simulating Pleuro-Pulmonary Infection ..	2
Fever and symptoms and signs suggestive of unresolved pneumonia or empyema.	
(iii) Caused by Secondary Growth	
Paraplegia	3
Cerebral tumour	1
Pain in abdomen (ileum)	1
Gross enlargement of liver	1
Total	17

In cases in which carcinoma of the bronchus was diagnosed before secondary growth had caused symptoms death occurred within three months of the onset. Death supervened rapidly in this small series of cases, but some patients survive for two or three years after diagnosis. One case is worth recording in detail because of the unusual mode of presentation.

CASE REPORT

Up to six weeks before admission this woman, aged 39, had been perfectly well. Her first complaint was of swelling of the face and upper part of the trunk. She also had some shortness of breath at that time, but no other symptoms.

On admission, on Sept. 9, 1947, her most distressing symptom was a massive, painful enlargement of both breasts. They were enormous and intensely tender; pitting oedema and peau d'orange was manifest. The right breast was larger than the left, and while she was sitting in bed this breast rested on her thigh. There was no discharge from the nipple; no localized inflammatory or other mass could be detected.

She also had a severe persistent cough, with brown blood-stained sputum. Her temperature was 104° F. (40° C.), pulse 200, and respiration rate 48. She was pale, cyanosed, very dyspnoeic, and obviously moribund. There was oedema and puffiness of her face and both upper limbs. The neck veins were engorged on the right side, but not on the left. Enlarged tender lymphatic glands were palpable in both axillae and in