Footwear is important. Protection against wet and cold is desired, on the other hand footwear that increases sweating is a hazard (rubber soled sneakers are the worst offenders). The substitution of rubber for leather soles in present day army footwear is a matter which should be watched. Constriction or pressure on the toes or around the heel must be avoided. British army boots proved superior to American military boots in the Second World War. The latter were smart but too well fitted and tended to cause constriction. Trench Foot was rife in the American army but practically unknown in the British.

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Dr A L LEIGH SILVER

HYPOKALAEMIC PERIODIC PARALYSIS COMPLICATING THYROTOXICOSIS IN A NEPALI

Periodic paralysis has been reported to occur in 8.9 per cent of Japanese males¹ and 13 per cent of Chinese males² with thyrotoxicosis. The corresponding figures for females were 0.4 per cent and 0.17 per cent. McFadzean and Yeung² commented that the high incidence of periodic paralysis among thyrotoxic mongoloids suggests that the basic defect may be genetically determined but that if this is so it is strange that the complication had not been reported among other Mongoloids. This is thought to be the first reported case in a Nepali who was ethnically of a mongoloid tribe (Limbu).

Case report
The patient was a 28 year old Nepali male who had ten days previously been diagnosed as a thyrotoxic and started on treatment with thyroxine 0.05 mg daily and carbimazole 15 mg to be taken three times a day (tds). One day prior to his admission on this occasion he complained of weakness in his legs, and a sore throat for which he received penicillin. On the morning of admission he awoke unable to get out of bed and had to be helped to the toilet; that afternoon he was admitted by stretcher to hospital.

Examination revealed a patient so moulded to the bed that his ear on the dependant side was bent forwards the patient being unable to raise his head in order to be more comfortable. He had normal facial movements, phonation and respiration, and was able to flutter his hands from the wrist as well as wriggle his feet and toes; the cranial nerves apart from both spinal accessories were intact. The triceps jerks were present on both sides but all other tendon reflexes were absent. The abdominal and plantar reflexes were normal. He complained of tingling in his hands and feet as well as ankles and knees. Whilst clinical evidence of thyrotoxi-
cosis did not seem present, the thyroid function tests from this episode were still abnormal. Serum thyroxine 247 nmol/l (normal range 44-161), T₃ percentage uptake 76 per cent (normal range 92-117), Serum T₄ (Radio-immune Assay) 5.42 nmol/l (normal range 1.54-3.85), Free Thyroxine Index 325 (normal range 44-161).

A diagnosis of hypokalaemic paralysis in association with thyrotoxicosis was made and confirmed by a Serum Potassium of 2.2 mmol/l. The ECG showed an increased rate of 100 beats/min a prolonged Q1T interval of 440 msec and no ‘P’ or ‘T’ waves.

He was treated with a single oral dose of 5 g (65 mEq) potassium chloride and within ninety minutes there was considerable improvement in proximal muscle function. The ECG gradually returned to normal and after fifty minutes the rate was 100 beats/min, Q-T interval 260 msec and configuration normal apart from one millimetre depression of ST segment. After four hours the ECG had returned to normal. The serum potassium one hour after treatment was 3.1 mmol/l and after eight hours 4.0 mmol/l.

The patient was discharged after 48 hours and continued carbimazole 15 mg tds and thyroxine 0.05 mg daily, plus slow potassium (1200 mg twice daily). A further episode of weakness occurred ten days later on awakening at 0600 hours. This attack resolved in less than eight hours, and by admission physical and electrolyte examinations were within normal limits. When last reviewed the patient had had no further attacks and was clinically and biochemically euthyroid though still on potassium supplements.

Discussion

This case illustrates several of the points brought out by McFadzean and Yeung². The attacks appeared before the thyrotoxicosis was controlled, they were within the quoted Seasonal Range of May to October, during which time the climate is at its most oppressive with a combination of high temperature and high humidity leading to profuse sweating and possible potassium loss. On both occasions the onset was whilst in bed and the individual concerned had well developed muscles.

Ali¹ and McFadzean and Yeung² reported that the condition remits spontaneously and that further relapse is prevented by controlling the thyrotoxicosis. Potassium supplements may therefore not be necessary. The actual mechanism of paralysis is as yet unexplained. Periodic paralysis complicating thyrotoxicosis cannot be entirely explained by a fall in the serum potassium level². Abnormalities of muscle calcium pump activity have been found in association with paralysis⁴ but do not account for the occurrence of hypokalaemia and its relationship to the paralysis.

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