foot, flexing the knee (to relax the biceps) and applying pressure. Open reduction is occasionally required, especially in the less common posterior dislocation when the common peroneal nerve may be damaged, as it was in the case reported by Dennis & Rutledge (1958).

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References

The myocardium in periodic paralysis

N. K. CONI
M.R.C.P., F.R.C.P.(C.)
Major, R.A.M.C.,
British Military Hospital,
Singapore

A case of familial periodic paralysis is reported in order to present the electrocardiographic changes observed during a series of artificially precipitated attacks. These changes draw attention to the present ignorance of the metabolic disturbances underlying this disorder and of the precise significance of the hypokalaemic electrocardiogram.

Case report
A 23-year-old Malay soldier was admitted to
hospital one afternoon having awoken that morning with profound weakness of his limbs which had persisted since. This was the fifth such attack, the first one having occurred at the age of 19, and the second a year later when he was admitted to hospital. The impression there was of an hysterical illness, but in retrospect such a conclusion was unwarranted because the serum potassium the day after admission was 3-2 mEq/l and the electrocardiogram showed the 'severe-classic' features of hypokalaemia described by Fletcher, Hurst & Schlant (1967) with ST segment depression, flattened T waves and prominent U waves. The next year he was afflicted by two further paroxysms which were again regarded as hysterical.

On each occasion he had awoken paralysed in the morning and there had been a gradual spontaneous recovery of power during the next 24 hr. No history could be elicited of any departure from his normal habits of diet or activity on the day before these episodes, nor was there any family history.

*Examination* during the presenting and subsequent paroxysms revealed a flaccid paralysis involving all four limbs and the hip-girdle and anterior abdominal muscles. The deltoids and posterior tibial muscles were comparatively unaffected and there was complete sparing of respiratory and bulbar musculature. The right biceps and patellar reflexes could not be elicited and this abnormality has persisted after recovery.

His serum potassium on admission was 2·5 mEq/l and the electrocardiogram was grossly hypokalaemic with PR prolongation (0·26 sec), T wave flattening throughout and U waves of greater amplitude than the T waves. These changes were most marked in leads V2 and 3 as Surawicz (1967) has noted.

The attack was terminated by the oral administration of 100 mEq of potassium so investigations were delayed for a week to allow him to settle down biochemically. Over an initial 5-day period on his customary diet his daily urinary potassium excretion averaged 28·6 mEq (range 20–42) with a fairly constant serum level of 4·0 mEq/l, so that there was no question of a renal leak of potassium which was, if anything, being conserved. A gastro-intestinal loss of the ion was considered very improbable as there...
was never any bowel disturbance and a barium enema was normal. Other investigations included normal \(^{131}I\) uptake, glucose tolerance test and urine chromatography.

During his time in hospital he sustained a further spontaneous episode, and on three occasions the paralysis was provoked by the intravenous administration of glucose and insulin (after the procedure had been explained to the patient and his cooperation enlisted). Although further urine electrolyte studies were not particularly revealing, they did suggest the possibility that these artificially induced attacks might not accurately reproduce the metabolic situation of the spontaneous attack. The spontaneous attack was followed the next day by a considerable sodium diuresis of 500 mEq, in contrast to his daily excretion of less than 200 mEq on all other days, and this sodium diuresis on the day after a paroxysm was described by Streeten (1963) and by de Graeff & Lameijer (1965). The sodium output on the day after an induced attack, however, was only 35 mEq. The point is made because the serial electrocardiographic changes recorded during a provoked attack were also dissimilar to those described in his second and fifth episodes.

The illustrations show lead V2 of his electrocardiogram when he was well, and in various phases during three induced attacks of paralysis. During the last of these episodes, his serum sodium and potassium reached a nadir of 120 and 1.48 mEq/l, respectively (a low serum sodium was not a constant feature of his attacks). It can be seen that the prolonged AV conduction, depressed ST segments, and flat T waves initially seen were not repeated. The striking changes are the reduction in T wave amplitude and the gross U waves, both of which bear very little relationship to the level of the serum potassium. There is, for instance a well marked U wave with a serum potassium of 4.8 mEq/l (Fig. 1c) and the changes are most obvious in the record taken at a level of 2.5 (Fig. 1b), while the second of the two tracings taken at a level of 1.48 (Fig. 2b) shows that the abnormalities are beginning to regress.

**Discussion**

Of the varieties of periodic paralysis, the familial
hypokalaemic form is that most frequently encountered. Whether the present patient was a sporadic case or whether closer scrutiny of his relatives would have revealed a family history is unknown. As the name suggests, the disease is associated with a fall in the potassium ion concentration of the extracellular fluid during the paroxysms, although the basic nature of the metabolic anomaly remains totally obscure. As in the present subject, there is no urinary potassium wastage nor excessive loss of gastro-intestinal secretions, and the total body exchangeable potassium is normal or only slightly low between attacks (Talso et al., 1963). The inference, therefore, is that there is a migration of potassium ions from the extracellular fluid into the intracellular compartment during the attacks. This is supported by the observations of Grob, Johns & Liljestrand (1957) who noted a marked arteriovenous potassium differential during an attack, and also by the precipitation of such attacks by glucose and insulin. More direct evidence is provided by the work of Vastola & Bertrand (1956), who measured intracellular ion concentrations in muscle biopsy specimens during an attack and found an increase in the potassium when compared with the interictal level.

For obvious reasons, there is no such information available concerning any electrolyte shifts affecting cardiac muscle in this condition. Evidence from the electrocardiograph is indirect and indeed the controversy about the relative roles of extracellular and intracellular potassiums in the genesis of the hypokalaemic cardiogram remains unresolved. Soloff, Kanosky & Boutwell (1960) found that the electrocardiographic manifestations of potassium depletion were associated with a reduced potassium concentration within the red cells whether the serum potassium was low, normal or high. More recently Swales (1964) has argued cogently that changes in the T waves and U waves reflected intracellular potassium depletion, the T wave pattern being restored pari passu with restoration of intracellular potassium. The striking lack of correlation between the configuration of the TU complex and the serum potassium in the present case lends support to the view that the extracellular concentration of the ion may not be the prime factor influencing the electrocardiographic appearances, which are probably due to intracellular changes instead. This would indicate that the myocardium is deficient in intracellular potassium, and that in this respect the behaviour of cardiac muscle in this disorder is totally unlike that of skeletal muscle. The explanation may have been provided by the speculation of Van Buchem (1957) that the myocardium sacrifices its potassium to be donated to the skeletal muscle.

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