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

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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 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

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.
Case reports

FIG. 2. (a) Third induced attack—40 min after glucose and insulin: serum potassium 1.48 mEq/l. (b) Third induced attack—55 min after glucose and insulin: serum potassium 1.48 mEq/l. (c) Third induced attack—80 min after glucose and insulin: serum potassium 1.7 mEq/l. (d) Third induced attack—100 min after glucose and insulin: serum potassium 2.15 mEq/l.

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
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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