MEDICAL MEMORANDA

Periodic Hypothermia

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Periodic hypothermia is a rarely recognized condition. Incorrect technique of taking temperatures and the infrequent use of low-reading thermometers may belie its true incidence.

Case Report

A 39-year-old mother of four children was referred to medical out-patients two years ago by her family doctor with the history that for 18 months she had slowed down and had started to neglect her family and home. For two months she had been having major convulsions without an aura at fortnightly intervals, and had been given sodium phenytoin 100 mg three times daily. There was no previous personal or family history of fits. Her periods had been scanty for two years and totally absent for seven months. The births of her children had been normal and she had breast-fed each of them. In the preceding 12 months she had been admitted urgently to a psychiatric hospital on three occasions and to a general hospital once. Each time the clinical picture was similar: she was depressed and withdrawn with some abnormal movements, spontaneously recovering after a few weeks. Her discharge summaries gave the diagnosis as depression, conversion hysteria, catatonic schizophrenia, and atypical liver failure.

On examination she was lethargic, uncommunicative, and depressed. Her skin was dry and coarse and there was early frontal balding (fig. 1). Her speech was slurred and there was a mild ataxia of cerebellar type with a resting tremor and a superimposed flap. The flap closely resembled that seen in liver failure. All reflexes were slow to relax. There was a bradycardia of 40 beats per minute. Her temperature failed to register on the scale of an ordinary thermometer and proved to be 31°C on a low-reading type. She remained hypothermic for four weeks (fig. 2). In the mornings she usually had attacks of profuse sweating which lowered her temperature still further, then there was a gradual rise during the day. Throughout this period her bodily thermostat seemed to be set at a level between 31 and 32°C. On attempting to warm her with an electric blanket she would feel hot, become vasodilated, and start to sweat, thereby maintaining her temperature below 32°C. Towards the end of her illness the paroxysms of sweating diminished and she complained more of shivering attacks. Over five days her temperature returned to normal. She was no longer depressed or ataxic.

Blood sugar estimations during sweating were consistently normal, as was catecholamine and 5-hydroxyindole-acetic acid excretion. There was no postural hypotension and the cardiovascular responses to Valsalva's manoeuvre were normal. Protein-bound iodine and serum thyroxine were in the normal range on several occasions. Basal metabolic rate was minus 14. Insulin stimulation test showed a normal growth hormone response to hypoglycaemia. Serum cortisols and gonadotrophins were within the normal range. Liver function tests showed abnormalities, the alkaline phosphatase being 20 to 25 K.A. units and the 5-nucleotidase was 30 to 40 units. Bilirubin, serum transaminases, proteins, and electrophoretic strip were normal. During her previous medical admission the alkaline phosphatase had been 80 K.A. units, the SGOT 160 U/ml, and the bilirubin 2.3 mg/100 ml with a normal blood ammonia. Liver biopsy was unremarkable. The electrocardiograph showed bradycardia with prolonged P-R and Q-T

FIG. 1—Appearances of early frontal balding.

FIG. 2—Temperature and pulse chart during typical period of hypothermia showing tendency for lowest figures to be found in mornings and gradual rise to normality as patient recovered.

intervals. Electroencephalograms showed slow background activity in the theta range at 5 to 6 Hz with runs of delta wave bilaterally. Occasional spikes were seen especially on photic stimulation. The E.E.G. remained abnormal after normothermia had returned. Skull radiographs showed no signs of abnormality, nor did a brain scan. Lumbar air encephalogram showed nothing abnormal, there was no suggestion of agenesis of the corpus callosum. The protein content of the cerebrospinal fluid was 35 mg/100 ml.

She was maintained on phenobarbitone 30 mg three times daily as an outpatient, and her fits were well controlled. However, she has had ten further attacks of hypothermia necessitating readmission. Each attack remitted spontaneously after one to four weeks. There was no seasonal pattern and they did not occur more frequently in cold weather. Some months ago she stopped taking phenobarbitone. There was no change in the frequency of attacks, but she had more fits. In some remissions menstruation returned. She requested and received tubal ligation. During one admission she developed a urinary infection and her temperature rose to 35°C. After administering an appropriate antimicrobial it returned to 31°C. Part of her temperature chart during her most recent admission when she developed an influenza-like illness that was affecting other patients on the ward is shown in fig. 3. In between attacks her temperature was between 35.5 and 36°C. There was a good correlation between her temperature and clinical state. Unfortunately, both during and between her attacks she was unco-operative, which rendered further detailed physiological investigation impossible.

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FIG. 3—Temperature and pulse chart showing effect of influenza-like illness, during which time patient felt hot and sweated profusely until temperature returned to previous baseline.

Comment

Hypothermia, as a terminal event, is not uncommon with hypothalamic tumours (Davison and Selby, 1935; Bauer, 1954). It also occurs in other forms of intracranial disease (Hockaday *et al.*, 1962; Fox *et al.*, 1970) but only four patients with a similar periodic temperature disturbance to our patient have been reported on previously (Duff *et al.*, 1961; Shapiro *et al.*, 1969; Guihard *et al.*, 1971). The girl reported by Duff *et al.* (1961) was essentially the same 15 years after hypothermia was first diagnosed (V. M. Leveaux, personal communication, 1972). Like the present patient there was no abnormality on the lumbar air encephalogram, but the two women reported on by Shapiro *et al.* (1969) and the boy by Guihard *et al.* (1971) had agenesis of the corpus callosum with an enlarged third ventricle extending upwards between widely separated lateral ventricles.

Hoffman and Pobirs (1942) suggested that sweating attacks were a form of focal autonomic epilepsy. Our patient was certainly epileptic and has had major convulsions when both hypothermic and normothermic. Her fits were not preceded by an aura, they did not seem to be related to her sweating attacks, and while the former were well controlled by anticonvulsants the latter were not. The ataxia that occurs in hypothermia has been described before (Duff *et al.*, 1961) but to our knowledge the hypothermic flap has not. Her ataxia was not due to phenytoin as it occurred when she was not on drugs.

It seems highly probable that her previous admissions to psychiatric hospitals might have been due to hypothermia, but with a clinical thermometer of the usual type hypothermia can easily be missed. The notch at the base of the column of a clinical thermometer is designed to prevent the mercury retreating down the scale after removal from the patient. It is essential, therefore, for the mercury column to be shaken down below the temperature scale before a recording is attempted. In some cases this takes much time and effort. Twenty ordinary thermometers in ward use were tested. One required 25 vigorous shakes and three others more than a dozen before this was achieved. The two low-reading thermometers available on the ward both needed more than 30 shakes to push the column below the scale. How many overworked nurses are obsessional enough to bother with every patient, especially when detection of pyrexia is their usual aim? Are there other patients intermittently attending hospitals with undiagnosed periodic hypothermia?

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An Unusual Neurological Manifestation of Tuberculosis

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The clinical presentation of tuberculosis in immigrant groups in this country may differ from that observed in the indigenous population. We have recently seen two immigrant patients with tuberculosis presenting with loss of use in an upper limb due to

an asymmetrical cervical radiculopathy. In neither case were there symptoms of spinal cord dysfunction and the radiographs of the spine showed no abnormality.

Case 1

A 32-year-old West Indian man who had lived in England for 18 years gave a six-month history of intermittent pains in the interscapular region. He felt generally unwell and lethargic with a 20-lb (9-kg) weight loss and night sweats. Six weeks before admission he had developed several soft painless lumps around the ribs on the left side. Seven days before admission he noticed weakness of the right arm, which progressed over three or four days to complete loss of power in the shoulder, elbow, and wrist, although he could still move his fingers.

On admission his temperature was 38.4°C and he looked unwell. Neck movements were limited by pain, though there was no neck rigidity and straight leg raising was full. There was a localized swelling, exquisitely tender, just to the left of the spinous processes of the L2 and L3 vertebrae. There were four soft fluctuant non-tender round lumps about 2 cm in diameter between the skin and bone over the left side of the thorax, lying between the second and eighth rib and between the anterior axillary line and the sternum.

Examination of the central nervous system showed a complete flaccid paralysis of all muscles at the right shoulder and elbow, with incomplete flaccid paralysis of the right wrist extensors and long

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