The physiological response to hypothermia is controlled by the hypothalamus, involving peripheral vasoconstriction and shivering. In hypothalamic hypothermia these systems fail with loss of reactive peripheral vasoconstriction to reduce heat loss and loss of the shivering response to produce heat. It is the failure of these systems that contributes to the hypothermia and also produces diagnostic difficulty, with the patient feeling warm to the touch and not shivering. The ECG showing the pathological Q waves, with absence of shiver waves mirrored the hypothalamic cause of the hypothermia.

This is the first description of hypothermia by multiple sclerosis with a proved hypothalamic plaque and no other identifiable cause for hypothermia.

We thank David Hughes for the preparation of the histopathological photographs.

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Lyme neuroborreliosis presenting with propranolol myoclonus

A 60 year old white woman presented with an arthralgia of the right knee which was diagnosed to be due to a tick bite on the right thigh on 11 July 1995. On 2 October 1995, she complained of a lumbar pain which radiated to the right thigh. She received dextropropoxyphene, paracetamol, thiocolchicoside, and lidocaine and had an lowered normal blood pressure which was additionally treated with codeine, chloromazanone, and tenoxicam. Despite this, the pains, which prevented sleep, rapidly radiated bilaterally to the back and leg in the abdomen. On 11 October 1995 she presented with flexor non-rhythmic symmetrical jerks of the trunk, the abdomen, both hips, and knees evident both sitting and standing. The myoclonic jerks and photophobia were treated by oral prednisolone and later by intravenous prednisolone. On 13 October 1995, she was transferred to the intensive care unit. The painful jerks were flexor, simultaneous in all the muscles, and spontaneous or induced consistently by flexion of the neck, with intermittent jerks of the upper limbs. The intervals between the jerks became so short that the paroxysms gave the impression of being attacks of sustained truncal flexion. An EEG during jerking was unremarkable. Finally, the patient was anaesthetised and ventilated artificially. The treatment was propofol, fentanyl, and muscle relaxant pancuronium. Ceftriaxone (2 g intravenously daily) was given for 14 days. The CSF contained 398 mononuclear cells/μl, numerous atypical cytological features, normal glucose and chloride ratios, increased protein content (1/2 g), intrathecal synthesis of IgG and IgM, and three oligoclonal bands were detected. The titre of antibodies to Borrelia burgdorferi was raised in the CSF (1/64: normal < 1/4) by indirect immunofluorescence with IgG (1/16) and IgM (1/16 to 1/322) (normal < 1/10) by enzyme linked immunosorbent assay (ELISA) (ImmunoWell borrelia Lyme—BMD); their detection in serum was negative three weeks later. On 22 October the patient was extubated. The jerks had totally disappeared and the pains dramatically improved. At this time, EMG failed to detect any myoclonic jerks. Recording of peroneal nerve somatosensory evoked potentials and MRI of the spine were unremarkable. On 24 October, the patient was free of pain and then recovered full strength and normal tendon reflexes.

The clinical features of pain resistant to analgesic agents, meningoradicularitis with a history of tick bite, and erythema migrans strongly evokes a Lyme neuroborreliosis confirmed by the presence and detection of antibodies to Borrelia burgdorferi. However, the most dramatic feature was the myoclonic jerks which support the clinical diagnosis of propranolol myoclonus characterised by the new appearance of myoclonic jerks of the neck, trunk, both hips, and knees. Sometimes attacks of sustained truncal flexion are generated by paroxysmal bouts of axial jerks.1 In this type of myoclonus, the discharge arises from a limited segment of the spinal cord and then spreads slowly up and down by the involvement of the long propriospinal pathways.2 The jerks had disappeared two weeks later. An EMG investigation of the patient’s EMG failed to detect any remaining myoclonus in our patient. Accordingly, we could not ascertain the possible origin in the throracic segment of the spinal cord, corresponding to the abdominal and lumbar muscles, which were painful throughout the course of the disease and constantly affected by the jerks. To our knowledge, no case of Lyme neuroborreliosis has been associated with a propranolol myoclonus. Another patient had stiffness, painful cramps, and spasmodic jerks confined to the left leg,3 which suggest a localised myelitis of the spinal interneuron. Our case strongly evokes the involvement of many spinal segments. Apart from the myoclonus, no other evidence of spinal cord disease was apparent. The treatment with propranolol relieved the pain and dramatically suppressed the myoclonus.

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Metamorphosis and visual hallucinations restricted to the right visual hemifield after a left putaminal haemorrhage

Metamorphosis is a rare neurological phenomenon in which objects appear distorted in form. Many reports have attributed the responsible lesion to the occipitoparietal cortex and its related structures.1 2 We report a case of left putaminal haemorrhage followed by metamorphosis and visual hallucinations restricted to the right visual hemifield. The origin of this patient’s symptoms was considered to be the left optic radiation.

A 63 year old right handed man with a previous history of hypertension was admitted to the hospital with acute right hemiparesis. On admission, his visual field examination showed a right homonymous hemianopia. There was also a right inferior hemianopia, facial palsy and a right hemiparesis without sensory involvement. The right homonymous hemianopia disappeared on the third day. On the fourth day, he complained that the doctor’s left hand had seemed to have been scraped, that the doctor’s left hand seemed tortuous, and that some of the fingers of the hand seemed to be missing. He drew a picture of what he saw (fig1A). Visual field examination by confrontation was immediately performed but no abnormalities were found, later confirmed by using Goldmann’s perimeter. On the next day, he complained, ‘The right half of the curtain in front of me suddenly transforms into an animal’s face. It rotates there for a while and finally flows to the right, and then disappears. At the next moment, another face springs up at the very portion’. He then drew a picture to illustrate his experience (fig1B). These phenomena lasted three to four days and then disappeared. One month later, he was able to walk without assistance and was discharged from hospital.

The laboratory analysis of blood and urine was within the normal range. Cranial CT on admission showed a left putaminal haemorrhage without ventricular extension

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Lyme neuroborreliosis presenting with propriospinal myoclonus.

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*J Neurol Neurosurg Psychiatry* 1996 61: 420
doi: 10.1136/jnnp.61.4.420

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