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 pathognomonic J waves, with absence of shiver waves mirrored the hypothalamic cause of the hypothermia.

This is the first description of hypothermia in a patient with 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 propriospinal myoclonus

A 60 year old white woman presented with an erythema migrans after 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 tetrazepam and then was additionally treated with codeine, chlormezanone, and tenoxicam. Despite this, the pains, which prevented sleep, rapidly radiated bilaterally to the low back and the abdomen. On 11October 1995 she presented with flexor non-rhythmic symmetric jerks of the trunk, the abdomen, both hips, and knees evident both sitting and standing, increasing when lying, without suppression by an effort of will or during voluntary movements. Although the pains and jerks were atypical, the patient was diagnosed as having a herniated disc; an epidural infiltration of dexamethasone (10 mg) gave a transient relief of the pains and jerks. A second infiltration was not effective. On 13 October the patient was admitted to hospital. The myoclonic jerks had reinforced, occurring sometimes in bursts, occasionally involving the neck and the shoulders but never the face. The patient was agitated and exhausted, and cried on account of the continuous distressing pains. She was free of headache and fever. Walking was difficult, although she felt better when standing. She had a mild motor deficit in the proximal part of the lower limbs, patellar tendon reflexes abolished on the right, diminished on the left, normal ankle reflexes, plantar reflexes flexor, and axial muscles and neck were not rigid. Results from routine laboratory investigations were normal. Ketoprofene, haloperidol, clorazepate, and then tiaprid, paracetamol, and buprenorphine were tried with negligible relief. On 19 October, 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, without involvement of the face and superior limbs. The intervals between the jerks became so short that the paroxyms 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/l), 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, both for IgM (1/16) and IgG (1/16), 1·352 (normal < 0·16) 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,[1] meningoradiculitis with a history of tick bite, and erythema migrans strongly evokes a Lyme neuroborreliosis confirmed by the CSF findings and detection of antibodies to *Borrelia burgdorferi*.[2] However, the most dramatic feature was the myoclonic jerks which support the clinical diagnosis of propriospinal myoclonus characterised by repetitive, non-rhythmic jerks of the neck, trunk, both hips, and knees.[3] Sometimes attacks of sustained truncal flexion are generated by paroxysmal bouts of axial jerks.[4] 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.[5] The jerks had disappeared at the time of the EMG investigation in our patient. Accordingly, we could not ascertain the possible origin in the thoracic 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 propriospinal myoclonus. Another patient had stiffness, painful cramps, and spasmodic jerks confined to the left leg,[5] which suggest a localised myelitis of the spinal interneurons. Our own strongly evokes the involvement of many spinal segments. Apart from the myoclonus, no other evidence of spinal cord disease was apparent. The treatment of the Borrelia rapidly relieved the pain and dramatically suppressed the myoclonus.

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

Metamorphopsia 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.[1–4] We report a case of left putaminal haemorrhage followed by metamorphopsia 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 facial palsy and a right hemiparesis without sensory involvement. The right homonymous hemianopsia disappeared on the third day. On the fourth day, he complained that the doctor's left cheek 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 and . . ." He then drew a picture to illustrate his experience (fig 1B). 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