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, and also the the tinctorial changes with loss of the myoclonus. A EEG was tried in June. The patient was admitted 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 increasing in the lower or superior 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/l), intrathecal synthesis of IgM and IgG, 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 for IgM (1/16) and IgG (1/32: normal <1/10) by enzyme linked immunosorbent assay (ELISA) (Immunowell borrelia Lyme—BMD); their detection in serum was negative three weeks after the onset. On 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 recovered full strength and normal tendon reflexes.

Lyme neuroborreliosis presenting with proopriospinal myoclonus

A 60 year old white woman presented with anemia, fatigue and intermittent headaches 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, tiaprid and tiaprid, with no effect. The pain was treated with codeine, chlorpromazine, and tenoxicam. Despite this, the pains, which prevented sleep, rapidly radiated bilaterally to the back and legs. On 5 October 1995, she presented with flexor non-rhythmic symmetric jerks of the trunk, the abdomen, both hips, and knees evident both sitting and standing. When lying, the patient was also treated with codeine, chlorpromazine, and tenoxicam. Despite this, she was unable to stand, and the pains and jerks were atypical, the patient was diagnosed as having a herniated disc; an epidural infiltration of dexamethasone (10 ml) 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 the con-

The clinical features of pain resistant to analgesic agents, meningoradiculitis with a history of tick bite, and erythema migrans strongly evokes a Lyme neuroborreliosis confirmed by EMG and detection of antibodies to Borrelia burgdorferi. However, the most dramatic feature was the myoclonic jerks which support the clinical diagnosis of proopriospinal myoclonus characterised by non-rhythmic jerks of the neck, trunk, both hips, and knees. 1

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 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 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 seemed to have been scraped, that the doctor’s left hand seemed to have been scraped, and that some of the fingers of the hand seemed to be missing. He drew a picture of what he saw (fig 1A). 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.

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Lyme neuroborreliosis presenting with propriospinal myoclonus.

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