The physiological response to hypothermia is controlled by the hypothalamus, involving peripheral vasodilatation and shivering. In hypothermic hypothermia these systems fail with loss of reactive peripheral vasodilatation 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 hypothermic cause of the hypothermia.

This is the first description of hypothermia in multiple sclerosis with a proved hypothermic 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 migration 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, thiochlorophen, and acetaminophen when lying, and thio 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 resection of dexamethasone (10 mg) gave a transient relief of the pains and jerks. A second infiltration was not effective. On 13 October 1995 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 continuous distressing pains. She was free of headache and fever. Walking was difficult, although she felt better when standing. She had a mildest kinetic tremor, normal proximal power 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 of neurological investiga-

tions were normal. Ketoprofen, haloperidol, clorazepate, and then tiaprid, paracetamol, and buprenorphine were tried with negligible response. On 26 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 proximal and 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 prescribed 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 <1/10)) by enzyme linked immunosorbent assay (ELISA) (Immunowell borrelia Lyme— BMD); their detection in serum was negative three weeks later. On 28 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, meningoarachnitis with a history of tick bite, and erythema migrans strongly evokes a Lyme neuroborreliosis confirmed by ELISA and determination of antibodies to Borrelia burgdorferi. However, the most dramatic feature was the myoclonic jerks which support the clinical diagnosis of propranolol myoclonus characterized by sustained non-kinetic jerks of the neck, trunk, both hips, and knees. Sometimes attacks of sustained truncal flexion are generated by paroxysmal bouts of axial jerks. 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 propranolol pathways. The jerks had disappeared by repeat tests of non-EMG investigations in our patient. Accordingly, we could not ascertain the possible origin in the thoricacic section of the spinal cord, corre-

sponding 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, which suggest a localised myelitis of the spinal interneuron of unknown origin strongly evokes the involvement of many spinal seg-

ments. Apart from the myoclonus, no other evidence of spinal cord disease was apparent, and the treatment to relieve 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

Metamorphos is a rare neurological phe- nomenon 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 hallucina-

tions 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 hemi-

parasis. On admission, his visual field examination showed a right homonymous hemianopia. The patient also had a right inferior temporal facial palsy and a right hemiparesis without sensory involvement. The right homony-

mous hemianopia 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 (fig 1A). Visual field examination by confrontation was immediately per formed 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 phena-

omena 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.
We studied 10 patients with panic disorder (mean age 29.5 years [range 23-35 years]) and 10 age matched healthy controls (mean age 27.5 years [range 24-34 years]). Controls were recruited from the sleep laboratory technicians. Patients satisfied criteria for a diagnosis of panic disorder according to DSM-IV. Protocol exclusion criteria included: (a) a history of major medical or neurological illness; (b) a history of sleep panic attacks; (c) current or past evidence of affective disorders; (d) history of drug abuse or dependence; and (e) taking benzodiazepines or other medications that might influence sleep or psychiatric status in the two weeks before the study. All subjects underwent a 48 hour ambulatory polysomnography (Oxford Medilog 9200). The ECG signal was recorded using a 12 lead tape and digitised at 128 Hz with 8 bit resolution using a specific option of the Medilog system. The R-R intervals were measured by means of a derive-threshold algorithm; the accuracy of the components, and the variability, was improved by fitting each RSR complex by a second order polynomial function.

Cardiac autonomic regulation during sleep in panic disorder

Panic disorder is thought to be associated with a dysfunction of the autonomic nervous system. Power spectrum analysis has been used recently to quantify spontaneous variability in heart rate in humans. Some authors have described alterations in patterns of cardiovascular response in panic disorder that can be interpreted in favour of sympathetic over-activity or cholinergic underactivity. These studies were performed during wakefulness and the result may reflect states of increased anxiety. During sleep there are repetitive modifications of the autonomic nervous system that are constant and not influenced by cognitive factors. In the present study, we used power spectrum analysis of the heart rate variability during sleep in patients with panic disorder to verify a possible intrinsic deficit in the autonomic regulation in this disorder.

Figure 1 (A) Drawing of the referring doctor's face. His left cheek seemed to have been scraped and some of his left fingers were missing. (B) A drawing of the curtain lace. A fold of the lace seemed to have been transformed into an animal's face and it seemed to flow to the right.

or mass effect in either the occipital or pari-
tetal lobes (fig 2). In the pattern shift visual evoked potential (VEP), the latency of the P100 during right visual hemifield stimulation was 112-4 ms, which was moderately delayed compared with 98-0 ms, the latency recorded during stimulation of the left visual hemifield, indicating the involvement of the left visual pathway posterior to the optic chiasm.

Many reports have attributed the lesion of metamorphopsia to the occipitoparietal cor-
tex and its related structures. However, others have reported that chiasmatic or retro-
splenial lesions could elicit this symp-
tom. This patient had a common putaminal haemorrhage without involvement of the parietal and occipital lobes, as confirmed by cranial CT. The results of the VEPs disclosed a lesion in the left visual pathway pos-
terior to the optic chiasm. Thus the lesion responsible for his visual symptoms is in the left optic radiation. This is the first report that such a lesion could cause use metamorph-
opsia. Based on the above mentioned reports and our own patient, we propose that any lesion along the visual pathway, from the retina to the occipitoparietal cortex, can cause metamorphopsia. Retinal lesions elicit ipsilateral monocular metamorphopsia, chias-
amatic lesions give rise to bitemporal meta-
morphopsia, and occipitoparietal lesions cause contralateral homonymous metamor-
phopsia. This patient with injury in the left optic radiation complained of contralateral homonymous metamorphopsia. The distribu-
tional pattern of metamorphopsia seems to correspond to the part of the visual pathway affected.

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Figure 2 Cranial CT on admission, showing a high density area in the left putamen.

Concerning sleep architecture, no differ-
ence was found in the percentages of all sleep stages between patients with panic dis-
order and controls (values are mean (SD)): stage 1 non-REM sleep 4-5 (2) v 3-9 (2-7); stage 2 non-REM sleep 49-8 (6-2) v 51-7 (7-4); stages 3-4 non-REM sleep 20-6 (8) v 20-1 (6-8); REM sleep 25-1 (6-8) v 25-5 (3-6). No difference was found in the num-
ber of analysed segments in each sleep stage between the two groups.

Mean R-R showed, though, in patients with panic disorder and controls, a trend towards an increase in all sleep stages compared with wakefulness before sleep. No difference was found in R-R mean and variance between the two groups. The LF component (sympathetic activity) decreased during sleep with minimal values during stages 3-4 non-REM sleep, whereas the HF component (parasympathetic activity) displayed a reciprocal
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