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 with multiple sclerotic foci with a proved hypothalamic plaque and no other identifiable cause for hypothermia.

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Lyme neuroborreliosis presenting with proopriospinal myoclonus

A 60 year old white woman presented with an ataxia mimicking 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 tetrazeptam and then was additionally treated with codeine, chlorzamene, and tenoxicam. Despite this, the pains, which prevented sleep, rapidly radiated bilaterally to the back and down the legs. 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, 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 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 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 right hemiparesis and pathological plantar responses. Palpation 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 neurodiagnostic investigations were normal. Propofol, haloperidol, clorazepate, and then tiaprid, paracetamol, and buprenorphine were tried with negligible effect. On 22 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 impulses from the lower limbs. The intervals between the jerks became so short that the paroxysms gave the impression of being attacks of sustained trunca 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 IgG (1/16) and IgG (1/16), 1-352 (normal < 0.16) by enzyme linked immunosorbent assay (ELISA) (Immunowell borreli Lyme—BMD); their detection in serum was negative three weeks later. On 9 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 meningoaracditis with a history of tick bite, and erythema migrans strongly evokes a Lyme neuroborreliosis confirmed by the presence of antibodies to Borrelia burgdorferi. However, the most dramatic feature was the myoclonic jerks which support the clinical diagnosis of proopriospinal myoclonus characterised by repetitive, non-rhythmic jerks of the neck, trunk, both hips, and knees.2 Sometimes attacks of sustained trunca flexion are generated by paroxysmal bouts of axial jerks.3 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.4 The jerks had disappeared at the time of the EMG investiga- tion in our patient. Accordingly, we could not ascertain the possible origin in the thoroacic segment 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 proopriospinal 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 of their own strongly evokes the involvement of many spinal seg- ments. Apart from the myoclonus, no other evidence of spinal cord disease was apparent. The patient dramatically 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 phe- nomenon, which objects appear distorted in form. Many reports have attributed the responsible lesion to the occipitoparietal cor- tex and its related structures.6 We report a case of left putaminal haemorrhage followed by metamorphopsia 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- paresis. On admission, his visual field examination showed a right homonymous hemianopia. There was also a right inferior quad- rant palysy and a right hemiparesis without sensory involvement. The right homony- mous 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 pic- ture of what he saw (fig 1A). Visual field examination by confrontation was immedi- ately 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 ...”. Then he drew a picture to illustrate his experience (fig 1B). These phe- nomena 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 illness, including antidepressant medication; and (d) use of psychoactive drugs in the two weeks before the study. All subjects underwent a 48 hour ambulatory polysomnography (Oxford Medilog 9200). The ECG was recorded using high fidelity tape and digitised at 128 Hz with 8 bit resolution using a specific option of the Medilog system. The R-R intervals were detected by means of a derive-threshold algorithm; the accuracy of the R wave detection on ECG tracing was improved by fitting each QRS complex by a second order polynomial function. The fiducial point on the ECG was taken as the maximum of the fitting parabola to reduce the error due to the low sampling rate. The heart rate variability signal was processed using an autoregressive algorithm. All the spectral calculations were performed on all the successive 300 second segments of ECG recordings of the second night. The analysed time intervals were chosen from: (a) awake state at the beginning of the night; (b) stage 2 non-REM sleep; (c) stages 3-4 non-REM sleep; (d) REM sleep.

We focused on two regions of interest in the spectrum: (1) the low frequency (LF) component 0.05-0.15 Hz: an increase of the power in this band is commonly associated with sympathetic activity; (2) the high frequency (HF) component 0.2-0.4 Hz, mainly expression of parasympathetic control. The following variables were evaluated: the R-R mean and variance, the power of LF and HF components, and the sympathovagal balance (LF/HF ratio). We analysed the normalised spectral component (ratio between the power density of each spectral component and the total spectral density minus the power in the band 0.05-0.5 Hz) as better measures of the autonomic activity in respect to the absolute numbers; in this way it is possible to remove the effects of the large variability in the HRV among the several subjects. We applied ANOVA to determine the changes within each group through the different conditions. Differences between the two groups were evaluated by unpaired two tailed Student’s t test.

Concerning sleep architecture, no difference was found in the percentages of all sleep stages between patients with panic disorder 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 24.1 [6.8]; REM sleep 25.1 [6.8] v 25.5 [3.6]. No difference was found in the number of analysed segments in each sleep stage between the two groups.

Mean HR-R showed, in both 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 wakefulness and all sleep stages in both groups.

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 hypothesised patterns of cardiovascular responsivity 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 variation during sleep in patients with panic disorder to verify a possible intrinsic defect in the autonomic regulation in this disorder.

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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 parietal 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 is 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-

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