nerve fibres which originate in the visual association field go to the ipsilateral motor association field via the ipsilateral parieto-occipital area. The other half of the nerve fibres, which originate in the same visual association field, go to the contralateral motor association field via the ipsilateral parieto-occipital area and thereafter via the posterior part of the corpus callous. A lesion at the parieto-occipital area might interrupt the connection between the visual association and motor association fields. If the lesion is small, one of the two pathways which go to the ipsi- or contralateral motor association field can be disturbed. It will be difficult therefore for the patient to grasp an object in the contralateral homonymous visual field with a unilateral, either right or left, hand. If at least one pathway is large, it will be difficult to grasp an object in the contralateral homonymous visual field with bilateral hands because of damage to both pathways.

Symptoms similar to optic ataxia can be seen in patients with motor disturbance, cerebellar symptoms, somatosensory disorders, visuo-spatial agnosia, apraxia, or visual field defects. Rondot et al maintained that these symptoms can be excluded in a disease with unspecific oculomotor ataxia, but most of the reported cases of optic ataxia have had some of these symptoms. Our report of the existence of pure optic ataxia also reported by Piccirilli et al, Hirose et al implies that optic ataxia can exist as a symptom independent of those symptoms we describe previously. Our CT findings and those by Hirose et al related to the lesion is located at the junction between the parietal and occipital lobes. In Piccirilli's case, the patient had difficulty in grasping an object in his hemivisual field using either hand. Hirose et al excluded a nystagmus optic ataxia, but most of the reported cases of optic ataxia have had one of these other symptoms.

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We have recently successfully treated a case of severe chronic palatal myoclonus. A 72 year old right handed woman presented with vomiting, "shaking" of the tongue and jaw, a choking sensation during sleep and slurred speech. Her condition had started three years previously with vomiting which occurred mainly at night. This was initially mild and intermittent and was not related to food. She denied any nausea, abdominal pain or dysphagia. She lost 22 kg in three years. One year before admission she developed what she described as a constant shaking of the tongue and mouth which persisted during sleep and appeared to wake her up during the night with a choking sensation. To prevent this she slept propped up with six pillows. Six months later she developed intermittent jerky movements of the left arm.

She had partial bowel resection for ileo-caecal tuberculosis many years before. She had no relevant family or social history and she was taking paracetamol and lorazepam 2.5 mg at night. She had not received treatment with neuroleptic drugs.

On examination she had continuous rapid myoclonic involuntary movements of the lips, soft palate and tongue. These were not affected by voluntary activity, including during sleep. Occasionally she also had brief jerky movements of the left hand. The rest of the neurological and general physical examination was normal.

Her full blood count, urea and electrolytes, liver function tests and brain computerised tomography scan were normal. Barium studies of the upper gastro-intestinal tract were also normal.

A diagnosis of palatal myoclonus was made and treatment with clonazepam 0.5 mg three times a day was started. One week later all her symptoms resolved completely. Palatal myoclonus is a rare condition characterised by rhythmic involuntary contractions of the oro-pharangeo-palatine muscles and the pharynx at a nystagmoid rate (120-180/min). Persistence of these movements during sleep distinguishes palatal from other forms of myoclonus and indeed from all involuntary movements. In addition to the classical features of palatal myoclonus, our patient had vomiting which occurred mainly when lying flat and improved when she was sitting up. This is probably due to mechanical stimulation of the pharangeal receptors by the soft palate.

Palatal myoclonus is due to a lesion interrupting the central tegmental tract,1 the olivo-dentate pathways or the contralateral dentate nucleus2 and leading to secondary dentate nuclear degeneration and pseudohypertrophy of the inferior olives. These lesions are usually due to brainstem infarction or idiopathic degeneration. Other causes include tumours, head injury and rarely neurosyphilis, multiple sclerosis, syringobulbia, and amyotrophic lateral sclerosis.

Until recently there was no effective treatment for palatal myoclonus. Phenobarbitone and sodium valproate were tried with limited success. Reduced brain serotonin metabolism has been reported in palatal myoclonus and 5-hydroxytryptophan (in addition to carbidopa) was used successfully in the treatment of one case. We used small doses of clonazepam to a patient with severe palatal myoclonus, with excellent response. We suggest that clonazepam is worth considering in the treatment of palatal myoclonus.

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Body building and rhabdomyolysis

In 1988 we reported three cases of myoglobinuria following the first session of body building.1 Our theory was that body building might be a frequent cause of exertional muscle necrosis. Since then, four other cases of this kind during body building have been referred to us.

Case 1 was a 20 year old student with no history of neuromuscular diseases. The day after a 30 minute session of body building, he complained of diffuse myalgia and of passing dark urine. Four days later his serum creatine kinase (CK) was 62 380 U/l (normal up to 170). One month after neurological examination, the serum CK and electromyography were normal. Open muscle biopsy, performed on the quadriceps muscle and processed as previously reported,1 did not show abnormalities.

Case 2 was a 20 year old student who regularly practised tennis, skiing, and body building. He interrupted his sports activities to have surgery for recurrent shoulder subluxation. Six months later it was recommenced and thereafter body building became his main activity. This patient was admitted to the first session of the serum CK revealed a marked increase of the enzyme (16 000 U/l). The patient was asymptomatic. Neurological examination was normal. Serum CK values returned to normal one week later. Electromyography and muscle biopsy performed one month later were normal.

Case 3 was a 25 year old housewife, who had practised competitive athletics from the age of 12 and 18 years. After the first session of body building she complained of diffuse myalgia. Five days later serum CK was 3500 U/l, but returned to normal in a few days. Neurological examination was normal.

Case 4 was a 19 year old student who complained of diffuse myalgia without myoglobinuria after the first session of body building. Three days later the serum CK was 11 000 U/l and returned to normal in one week. Neurological examination was normal.

All four patients denied using steroid hormones or other drugs. The four cases confirm the occurrence of rhabdomyolysis with and without myoglobinuria19 after body building and suggest
that this sport may be a frequent cause of exertional muscle necrosis. These cases suggest two other considerations: 1) as all the patients were young and athletic, the occurrence of rhabdomyolysis appears related to the specific characters of body building, which uses muscles less involved in other physical activities; 2) rhabdomyolysis is probably much more frequent than observed.

In conclusion, body building must be included among the causes of exertional rhabdomyolysis and caution must be recommended in practising this sport.

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