

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 callosum. 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 the lesion 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 should be excluded in diagnosing optic ataxia, but most of the reported cases of optic ataxia have had some of these other symptoms.^{1,2,4}

Our report on the existence of pure optic ataxia, also reported by Piccirilli *et al*,³ Hirose *et al*,⁴ 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* revealed that 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* reported a case similar to that of Piccirilli's. In our case, the patient could not grasp an object in his left homonymous visual field using his left hand. These three cases strongly support the anatomical explanation of optic ataxia offered by Rondot *et al*.

We conclude that optic ataxia is an important symptom which indicates the existence of a parieto-occipital lesion.

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Palatal myoclonus successfully treated with clonazepam

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 ileocaecal tuberculosis many years before. She had no relevant family or social history and she was taking paracetamol and lorazepam 2.5 mgs at night. She had not received treatment with neuroleptic drugs.

On examination she had continuous rapid rhythmical involuntary movements of the lips, soft palate and tongue. These were not affected by voluntary action and persisted 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 mgs three times a day was started. One week later all her symptoms resolved completely. Palatal myoclonus is a rare condition characterised by rhythmical involuntary contractions of the oro-pharyngo-palatine muscles and the diaphragm at a nystagmoid rate (120–180/minute). 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 in the upright position. This is probably due to mechanical stimulation of the pharyngeal 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 vacuolar degeneration and pseudohypertrophy of the inferior olive. 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.^{3,4} Phenobarbitone and sodium valproate were tried with limited success. Reduced brain serotonin metabolites have been reported in palatal myoclonus and 5 hydroxytryptophan (in addition to carbidopa) was used successfully in the treatment of one case.⁴ We used small doses of clonazepam, a drug which increases brain 5 hydroxytryptamine,⁵ 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 rhabdomyolysis following 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 50 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 recommended that he take up body building. The day after the first session, 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 between 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.

These four cases confirm the occurrence of rhabdomyolysis with and without myoglobinuria^{1–3} after body building and suggest

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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 rhabdomyolysis⁴ and caution must be recommended in practising this sport.

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