dyssaphria but no dysphasia. Vision and extraocular movements were normal. The corneal reflex was diminished on the left. There was mild weakness of the left side of the face. Taste was preserved. The uvula deviated to the left and the gag reflex was diminished on the right. The tongue had no wasting but deviated to the right upon protrusion. There was no evidence of Horner’s syndrome. There was pronator drift of the left upper extremity and mild weakness of the left leg. A left hemisensory deficit for pinprick, touch, and proprioception involved the left arm, leg, and trunk. Muscle stretch reflexes were more active on the left and plantar responses were flexor. There was no ataxia.

Initial CT scan of the head was normal. At this point the diagnosis of medullary infarction on the right was considered but the left lower facial weakness and absent left corneal reflex could not be explained. A repeat CT scan of the head four days later revealed a right parietal infarction. Duplex ultrasound of the carotids revealed an occlusion of the distal right internal carotid artery. Magnetic resonance imaging ten days after presentation demonstrated a dramatic enlargement of the diameter of the right internal carotid artery, beginning several centimeters above the bifurcation and extending to the base of the skull (fig). The signal was compatible with intramural haemorrhage. There was no flow void signal. Tapering occlusion of the right internal carotid artery was demonstrated by angiography. Heparin was started and then changed to warfarin. One year later the patient has improved ability to swallow but is otherwise unchanged. Repeat duplex ultrasound has shown no evidence of recanalisation.

Hypoglossal palsy in association with internal carotid artery dissection has been reported infrequently, occurring in 5% of cases.2 The occurrence of both tenth and twelfth nerve palsies in this situation is rare.3 Hypoglossal paralysis with contralateral motor and sensory symptoms simulating a crossed hemiplegia has been reported only once.4 That patient experienced two transient ischaemic attacks in association with a persistent hypoglossal paralysis. Our patient is unique in that the lower cranial nerve palsies began simultaneously with the contralateral motor and sensory signs and the deficits persisted. The presentation mimicked that of medullary infarction. The clues of mild left facial weakness and a diminished left corneal reflex cautioned against this localisation.

MRI showed the enlarged internal carotid artery with intramural haemorrhage and loss of the flow void signal indicating occlusion. Angiography confirmed the presence of a tapering occlusion of the internal carotid artery. The close proximity of the pharyngeal branches of the vagus, glossopharyngeal and hypoglossal nerves to the internal carotid artery in the neck makes them susceptible to compression from an expanding internal carotid artery.5 However, other factors such as ischaemia may be involved in the pathogenesis of cranial nerve palsy. MRI is superior to angiography in that the vessel wall is visualised and not just the lumen.6 Internal carotid artery dissection with ipsilateral lower cranial nerve palsies and contralateral hemiplegia should be added to the better known false localising signs.7

Pure optic ataxia associated with a right parieto-occipital tumour

Optic ataxia is defined as a disorder of hand movement when grasping an object in the peripheral visual field. Twenty or more cases of optic ataxia have been reported since 1967 when Garvin et al8 originally introduced the idea of optic ataxia. Rondot et al9,10 colleagues of Garvin, established that a disturbance of visuo-motor coordination is responsible for optic ataxia. They insisted that motor, cerebellar, and somatosensory disturbances, visuo-spatial agnosia, apraxia, and visual field defects should be excluded in diagnosing optic ataxia. However, we have found that, among reported cases, pure optic ataxia without these symptoms has been confirmed in only two cases.11 We report a case of optic ataxia in a pure form. Our case supports Rondot’s concept that optic ataxia is a specific entity independent of other neurological disorders.

In November 1983 a 47 year old right handed man with no previous disease noticed difficulties in grasping objects, errors in estimating distances, and misty vision. On 23 January 1984, when these symptoms became slightly aggravated, the patient was admitted to our hospital. On admission, he was alert and well-oriented. His verbal functions and memory were normal. Visual acuity was normal in each eye. External ocular movements were completely normal in every direction, and no diplopia was detected. Optic fundi were clear bilaterally. With the visual line fixed to the centre of the visual field, it was impossible for him to grasp an object at the periphery of the left homonymous visual field with the left hand, while easily possible with the right hand. Grasping an object in the right homonymous visual field with the left or right hand was performed smoothly. The patient did not exhibit visual inattention. Disturbances of the cranial nerves were not found. Muscle strength of the four extremities was symmetrically normal. Deep sensation such as joint sense, vibration sense, and deep pain was not disturbed. Two point discrimination, graphesthesia, stereognosis, and double simultaneous stimulation were all normal. Superficial sensation was also normally preserved. There were no disorders of cerebellar functions. In the neuropsychological examination, there was no aphasia, apraxia, or visuo-spatial agnosia. On the basis of these neurological findings, it was diagnosed that the patient was suffering from optic ataxia.

Enhanced CT revealed a high density lesion in the right parieto-occipital region (fig). Subtotal removal of the tumour was performed, and it was histopathologically shown to be a malignant lymphoma. Systemic examinations revealed no malignant lymphoma elsewhere. We concluded that the tumour was a primary malignant lymphoma in the right parieto-occipital area. The residual tumour completely disappeared after whole brain irradiation of a total of 50 Gy.

In 1967 Garvin et al reported the case of a patient who had difficulty in grasping objects at the periphery of the visual field, when the patient’s visual line was fixed. They named this neurological disorder “optic ataxia (ataxie optique)”.11 It has been revealed on CT and necropsy that optic ataxia is caused by a lesion at the junction of the parietal and occipital lobes.12 Rondot et al13 have established the concept that optic ataxia is a kind of disconnection syndrome which occurs due to interruption of nerve fibres connecting the visual association field (area 19) and the motor association field (area 6) which is responsible for identifying targets in space, for choosing a course of action, and for programming movement.14 It is thought that one half of the

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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 internal capsule. 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 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 would be included in diagnosing optic ataxia, but most of the reported cases of optic ataxia have had some of these other symptoms.

Our report of the existence of pure optic ataxia also reported by Piccirilli et al,1 Hirose et al,2 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 excluded the possibility of optic ataxia, but most of the reported cases of optic ataxia have had some of these other symptoms.

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

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Body building and rhabdomyolysis

In 1988 we reported three cases of myoglobinuria following the first session of body building. Our theory was that body building might be a frequent cause of exertional muscle necrosis. Since then, four other cases of rhabdomyolysis 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 50 minute session of body building, he complained of diffuse myalgia and of passing dark urine. Four days later his serum creatinine 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, 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 at a level which he considered to be light. 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 pursued competitive athletics for many years. At 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 after body building and suggest...
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