compared with the first, whereas the prolactin response was unaltered. We now report the endocrine effects of repeated intravenous infusions of RX77368 in MND.

Seven patients (six male, one female, age range 43–67) with MND were given six intravenous infusions of RX77368 on Mondays and Thursdays of three successive weeks, starting at 9.30 am on each occasion. The dose of RX77368 was 0.2 mg/kg, given over two hours. If side effects were unacceptable, the dose was reduced on subsequent infusions. Two patients failed to complete all the infusions, one having five, the other four. Of the other patients, three had dose reductions, to 0.15 or 0.1 mg/kg because of side effects. Blood samples were taken for measurement of T4, T3, TSH and prolactin before, after one hour and at the end of the infusion, then at four and six hours after the infusion, and every morning at 9 am until the next infusion.

All hormones were measured as previously described, except that prolactin standards were obtained from the Division of Molecular Endocrinology, Hammersmith Hospital, London. The detection limit for prolactin was 40 μU/l. Results were analysed using analysis of variance with Duncan’s test for multiple comparisons. Correlations between groups were described using Spearman’s test.

The study was approved by the local ethics committee.

Each point represents blood samples taken from up to seven patients. Samples were taken before each infusion, one hour through, immediately after, then four and six hours after each infusion. Further samples were then taken each day until the next infusion. There was a three to four day interval between infusions. Error bars represent standard error of the mean. Cross-hatched bars represent RX77368 infusions, 0.1–0.2 mg/kg over two hours. Bars in the TSH are suppressed between infusions compared with pre-treatment (p < 0.05), and peak TSH is greater after the first infusion than after subsequent infusions (p < 0.05). Peak values of T1 and T2 after the infusion follow the same pattern as TSH, being greatest after the first infusion (p < 0.05 in each case).

Carotid dissection: a new false localising sign

Spontaneous internal carotid artery dissection typically presents with ipsilateral head and neck pain, oculosympathetic paresis, transient ischaemic attacks and stroke. The patient was admitted to hospital for investigation.

We describe a carotid dissection with tenth and twelfth cranial nerve involvement and hemiplegia which clinically mimicked medullary infarction.

A hypertensive 41 year old man developed right frontal and orbital pain. There was no history of trauma but there was a history of alcohol abuse. Three days later on awakening, the patient was unable to articulate. He noticed weakness, numbness, and clumsiness of the left hand and leg. On physical examination his blood pressure was 140/90. There were no head, neck or orbital bruits. Mental status examination was normal. There was no alteration of consciousness. MRI of the brain showed infarction of the right middle cerebral artery. There was no evidence of atherosclerotic disease. Carotid Doppler ultrasound revealed a dissection extending from the carotid bulb to the distal intracranial artery. The patient subsequently developed hemiparesis which resolved over several weeks.
dysarthria but no dysphasia. Vision and extraocular movements were normal. The corneal reflex was diminished on the left. There was mild weakness of the left lower facial muscles. 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, pain and proprioception involved the 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.3 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 nerve to the internal carotid artery in the neck makes them susceptible to compression from an expanding internal carotid artery.4 However, other factors such as ischaemia may be involved in the pathogenesis of cranial nerve palsies. MRI is superior to angiography in that the vessel wall is visualised and not just the lumen.5 Internal carotid artery dissection with ipsilateral lower cranial nerve palsies and contralateral hemiplegia should be added to the better known false localising signs.5

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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 al6 originally introduced the idea of optic ataxia. Rondot et al7 established the concept of optic ataxia in a pure form. Our case reports 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 noted 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. Optical 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 were 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”). 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.8,9 Rondot et al10 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.11 It is thought that one half of the