Letters to the Editor

myopathy and superficial peroneal sensory responses and low amplitude right posterior tibial and peroneal motor responses.

The patient had hypothensive treatment in the intensive care unit but dissection extended to the aortic bifurcation causing haemopericardium and the patient died four weeks later. A necropsy was not performed.

Our patient probably had an acute ischaemic plexopathy. The sudden beginning of the clinical picture “like an apoplexy” was in favour of a vascular aetiology. The diagnosis of more distal neuritis was not suspected as an angiogram of the lower limbs showed that flow was strictly normal. Ischaemic neuritides are divided into those occurring with diseases affecting large arteries and those occurring with dissection as that affect small arteries. Acute ischaemia of peripheral nerves generally results from occlusion of a main proximal limb artery or from occlusion of many distal arteries. The rareness of such a case can be explained by the relative resistance of peripheral nerves to ischaemia. It results from their slight metabolic needs (oxygen requirement of human nerve = 0.9 ml/100 g) and from the afferent vessels that provide a generous blood flow (43 ml/100 g/min).2 The nutrient arteries that supply peripheral nerves enter the epineurium and form a collateral vascular network of interfascicular arteries along the length of the nerve, so that it is difficult to infract a peripheral nerve. The intensity of nerve damage varies with the number of nutrient vessels, the availability of the collateral circulation, age of the nerve and the ratio of the relative and particular duration of arterial occlusion.

The different fibres that constitute peripheral nerves seem to be affected selectively by ischaemia. In some studies, anoxia seems to affect the small fibres before the large ones and Schwann cells seem to be more resistant to anoxia than axons. Nevertheless, opposite data are found in other studies, which suggest a greater vulnerability of large fibres.

Clinical features of ischaemic neuritis are generally stereotyped.2 Sensory deficits are more frequent than motor deficits. In particular, cutaneous sensory loss, deep burning pain and neurological examination usually shows impairment of all sensory modalities or pattern of sensory loss that spares the large fibres. There are no signs of muscle necrosis. An EMG usually shows abnormalities consistent with axonal damage. Conduction blocks are unusual.

Such ischaemic lumbosacral plexopathies have been reported after aortobifemoral graft and aortofemoral bypass graft by resection of nutrient arteries, after use of an intra-aortic balloon, and after vasospasm caused by an injection of drugs into an infrapopliteal artery. Only one case of painful ischaemic lumbosacral plexopathy due to an aortic dissection has been reported.1

Anatomically, the lumbosacral plexus is supplied by five lumbar arteries, which originate from the aortic bifurcation. The deep circumflex iliac artery, a branch of the external iliac artery, and the ilioilumbar and glutal branches of the internal iliac artery.11 In our patient, the median of the lumbosacral plexus could be due to interruption of blood flow through the lumbar segmental arteries and branches of the iliac arteries secondary to the dissection of the aortic wall and of the most proximal iliac artery walls just below the aortic bifurcation.

The clinical picture in our patient was also characterised by the absence of pain whereas causalgia like burning is generally common in acute and chronic ischaemic neuritides.

Absence of pain has rarely been reported in ischaemic plexopathies. Clinical and pathological studies of nerves in painful neuritides have been limited but this occurrence of pain is usually related to a selective damage of unmyelinated and myelinated small fibres. In the same way, painless neuritides seem to be related to a selective damage of large fibres, as in our patient. On the other hand, a selective loss of fibres has not been confirmed by other studies, in which the ratio between large and small fibres was not modulated.1

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Electroconvulsive therapy for the physical signs of Parkinson's disease without depressive disorder

Faber and Trimble1 have reviewed the reports on electroconvulsive therapy (ECT) in Parkinson's disease. We conducted a prospective open trial to evaluate the short term benefit of ECT on the physical signs of Parkinson’s disease without major depressive disorder. This is a repeated measures time series design where all patients received a single baseline assessment before and two assessments after ECT, and were their own control group. Four sessions of ECT were given unilaterally to the non-dominant hemisphere over eight days. Approval for the study was given by the ethics committee, Royal Hobart Hospital and all patients gave written informed consent.

Fifteen patients (12 men) participated. Five failed to comply with all assessments and were therefore eliminated from the analyses. The remaining population (seven men and three women) had an average age of 64.8 years. The time since initial diagnosis of Parkinson’s disease ranged from two months to 16 years. Most were severely

affected and a minority were mildly affected. Two patients were not receiving medication; the others were taking antiparkinsonian medication that was continued at the same dose throughout the study. Psychiatric assessment before the first treatment excluded major depressive disorder.

Neurological assessments were performed two weeks before the first and 24 hours and two weeks after the last therapy. These were scales or parts thereof, including unified Parkinson’s disease rating scale (subscales for the evaluation of akinesia, tremor, and rigidity), North Western University disability scale (subscales for the evaluation of walking, dressing, hygiene, eating, and speech), and Hoehn and Yahr staging system (the whole scale was used in assessing the stage of the disease).

Repeated measures analyses of variance with the rejection criterion set at the 0.05 probability level were used to assess the differences between each assessment period for each subscale. Significant differences between the assessment before ECT and the two assessments after ECT were present for akinesia \( F(2,9) = 11.377, P = 0.0022 \), tremor \( F(2,9) = 9.947, P = 0.0030 \), rigidity \( F(2,9) = 13.941, P = 0.0055 \), feeding \( F(2,9) = 6.789, P = 0.0206 \), and speech \( F(2,9) = 5.516, P = 0.0215 \), and the Hoehn and Yahr score \( F(2,9) = 7.744, P = 0.0065 \).

Significant improvements in akinesia, tremor, rigidity, and feeding were found when the pretreatment was compared with the assessment 24 hours after ECT. These improvements were sustained, and there were also significant improvements in speech and the Hoehn and Yahr staging, when the pretreatment assessment was compared with the assessment two weeks after ECT. This pattern of improvement has not been previously reported. Walking, dressing, hygiene, and eating did not significantly improve.

Psychiatric monitoring throughout and formal assessment at 24 hours and two weeks after ECT found no serious side effects. Four patients developed transient confusion after ECT, which was not sufficient to cause withdrawal from the study. One patient developed dyskinesia, which resolved with reduction of medication.

We concluded that four ECT treatments unilaterally to the non-dominant hemisphere over eight days was useful in the management of Parkinson’s disease, at least in the short term.

We are unable to report the long term effects of ECT on Parkinson’s disease as our research team disbanded. Some patients are known, however, to have maintained improvement for at least 16 weeks. The medical literature contains reports of beneficial effects lasting from several weeks to years. Given the favourable results reported here, studies with longer follow up periods are indicated.

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