they may relate to the high frequency of the 11 778 mutant allele in the retinal cell layer and optic nerve, and to the relative rarity of peroneal and peroneal motor responses. The patient had hypotensive treatment in the intensive care unit but dissection extended to the aorta, causing haemopericardium and the patient died four weeks later. A necropsy was not performed.

Our patient probably had an acute ischaemicplexopathy. The sudden beginning of the clinical picture “like an apoplexy” was in favour of a vascular aetiology. The diagnosis of more distal neurpathies was not suspected as an angiogram of the lower limbs was strictly normal. Ischaemic neuropathies are divided into those occurring with diseases affecting large arteries and those occurring with diabetes, which 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 cases can be explained by the relative resistance of peripheral nerves to ischaemia. It results from the slight metabolic needs (oxygen requirement of human nerve = 0.3-0.5 ml/100 g) and from the presence of a collateral network, which provides a generous blood flow (43 ml/100 g/min). The nutrient arteries that supply peripheral nerves enter the epineurium and form a collateral vascular network of interconnecting arteries along the length of the nerve, so that it is difficult to infarct a peripheral nerve. The intensity of nerve damage varies with the number of nutrient vessels, the availability of the collateral circulation, age of the patient and between the different arterial occlusion.

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

Clinical features of ischaemic neuropathy are generally stereotyped. Sensory deficits are more frequent than motor deficits. In particular, spontaneous pain and neurological examination usually shows impairment of all sensory modalities or pattern of sensory loss that spares the large fibres. These are no signs of muscle necrosis. An EMG diagnosis of these abnormalities consistent with axonal damage. Conduction blocks are unusual. Such ischaemic lumbosacral plexopathies have been reported after aortobieminal graft and aortofemoral bypass graft by resection of nutrient arteries, after use of an intral-aortic balloon, and after vasospasm caused by an injection of drugs into an infe-

Letter to the Editor

Letters to the Editor


Painless ischaemic lumbosacral plexopathy and aortic dissection

There is only one case report dealing with ischaemic lumbosacral plexopathy without simultaneous signs of limb ischaemia.1 We present a similar case characterised by the total absence of pain. A 69 year old man suddenly developed a weakness and numbness of the right leg. He also complained of vague abdominal discomfort but no pain was felt in the leg. His risk factors included arterial hypotension and angina pectoris. He was not diabetic. On examination there was a pronounced global weakness (grade 0-2/5) of the right lower limb, including the hip flexors and the gluteal muscles. The knee jerk and the ankle jerk were abolished. Sensory disturbances were found below the L2 level on the right side. Light touch and proprioception were completely impaired whereas sensation for pain and temperature was less diminished. The real muscle function was noted and defecation was normal. Distal pulses were normal and there were no signs of muscle or skin necrosis. His CSF was normal. Aortic angiography showed a typical subacute aortic dissection extending from the origin of the descending thoracic aorta down to the bifurcation (type 3). The angiogram of iliac and femoral arteries showed distal arteries of lower limbs was normal.

An EMG taken two weeks later showed almost no voluntary motor unit potentials in the iliopectos, quadriceps, glutei and thigh adductors muscles. Recruitment of motor unit was much reduced in other muscles of the right lower limb. Fibrillations and positive sharp waves were absent. Nerve conduction studies showed absent right sural and superficial peroneal sensory responses and low amplitude right posterior tibial and peroneal motor responses.

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 neurpathies. Absence of pain has rarely been reported in ischaemic plexopathies. Clinical and pathological studies of nerves in painful neurpathies have been limited but the occurrence of pain is usually related to a selective damage of unmethylated and myelinated small fibres. In the same way, painless neuropathies 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 modified.1

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

Faber and Trimble 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 blinded, 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 diagno-

disease of Parkinson's disease ranged from two months to 16 years. Most were severely
Painless ischaemic lumbosacral plexopathy and aortic dissection.

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