Rapid recovery after delayed myelopathy from electrical burns

Sir: We report a 34 year old man who was transferred to the Burns Unit, Concord Hospital, Sydney, Australia after accidentally flying his hang-glider into 22,000 volt power lines from which he had remained suspended for twenty minutes. On arrival he was noted to have sustained full thickness burns to the medial and ventral aspects of both forearms as well as the lateral aspect of the left thigh. Apart from weakness of right wrist extension, on initial examination power in all muscle groups was normal. He underwent extensive emergency fasciotomies of both arms and the left thigh. During his first few days in hospital he developed rhabdomyolysis (creatinine 132,000 units/l), myoglobinuria and mild renal impairment (serum creatinine 0·15 mm/l).

Two weeks after his admission, when his medical condition had improved, he complained of bilateral leg weakness and numbness of the left buttock. Neurological examination revealed signs of a myelopathy with bilateral lower limb spasticity and grade 4/5 weakness in a pyramidal distribution in both legs. There was also grade 4/5 to 4+5/5 weakness of the intrinsic muscles of both hands and wrist extension and shoulder abduction of the right arm. The right supinator jerk was reduced. All lower limb reflexes were pathologically brisk and remaining reflexes were normal. Plantar responses were bilaterally extensor. Sensory examination revealed diminished sensation to pin and light touch over the median nerve territory of the right hand. There was no evidence of a sensory level and joint position sense was normal. Bowel and bladder function was unaffected. The patient's gait was markedly spastic and he was unable to walk unaided. Magnetic resonance imaging of the cervical and thoracic spinal cord was normal. Nerve conduction studies were not possible because of the burns to both forearms. However, somatosensory evoked potentials from both posterior tibial nerves revealed delayed peripheral as well as an absent cerebral (N/P37) response bilaterally (fig). In addition median nerve somatosensory evoked responses showed an absence at Erbs point, as well as low amplitude prolonged cervico-medullary P/N13 (17·4 ms: normal < 15·4 ms) and cerebral N19 (22·6 ms: normal < 21·9 ms) on the right side and on the left a prolonged low amplitude Erbs point potential (12·6 ms: normal 12 ms) with low amplitude cervico-medullary and cerebral responses of normal latency.

Most of the 40 cases of myelopathy following electrical injury were described before World War II.1 In a more recent series in one major US Army trauma centre myelopathy was found in only two of 111 patients presenting with electrical burns.2 A delay between electrical injury and development of the myelopathy is characteristic. The reason for this is not known but it is speculated that as the electrical current passes through the spinal cord it damages its blood supply leading to a delayed vasculopathy.3,4 As with our patient, most cases have little sensory and no autonomic disturbance.3,5 Our patient's right median nerve sensory loss was probably related to carpal tunnel compression by oedema occurring after his burns, or to direct current injury. Somatosensory evoked potentials revealed upper and lower limb peripheral conduction abnormality as well as central conduction delay. This not only reflects damage to the spinal cord but also involvement of nerve roots and peripheral nerves lying in the path of the electrical current.6 Recovery, if it occurs, has been said to be incomplete, not infrequently extending over years.7 Several more recent reports attest to the fact that, as with our patient, a relatively rapid and almost complete recovery can occur from this unusual myelopathy.3,5

Intradural spinal lipoma with enlarged intervertebral foramen.

Sir: Spinal lipomas are benign tumours, usually divided into extradural and intradural types.4 Spinal lipoma with spina bifida (lipomyelomeningocele) is distinguished from spinal lipoma without spina bifida.1 Spinal lipoma without spina bifida is a rare benign tumour comprising about 1% of all spinal tumours.1 Spinal hourglass tumours (dumb-bell tumour) occurs simultaneously both inside and outside the dura mater, or may develop an extratubular extension through one or more intervertebral foramen,2 and is a neurinoma in the majority of cases. Dumb-bell extension has rarely been reported in cases of lipomyelo-meningocele and extradural spinal lipoma.3,5 To our knowledge, there are no case reports of intradural spinal lipoma in the absence of spina bifida, which extend through the intervertebral foramen and cause radiologically-proven widening of the intervertebral foramen. We report such a case of intradural spinal lipoma with an enlarged intervertebral foramen.

An 18 year old Japanese man was admitted to our hospital in August 1985, because of a gait disturbance. His foot had an equinovarus from childhood. He complained of back pain at the age of 15 years. In August 1984 he noticed weakness in the right and left leg. In December 1984 he visited another University Hospital and was diagnosed as having "famillial spastic paraplegia".

Fig. Posterior tibial somatosensory evoked potentials from right and left lower limbs demonstrating bilateral delay of the lumbar potential (LVI-IC) (marked 2) at 26·4 and 26·0 ms respectively (normal less than 24·0 ms). A cerebral response (CZ-FZ) was absent bilaterally.

References
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