SHORT REPORT

Pathogenesis and recovery of tetraplegia after electrical injury

J Thaventhiran, M J O'Leary, J H Coakley, M Rose, K Nagendran, R Greenwood

Abstract

The site of neurological damage causing paralysis after electrical trauma remains to be clarified. A patient is described who developed a flaccid tetraplegia after a high voltage electrical injury. The findings on initial examination and neurophysiological investigation showed a very severe generalised sensory-motor polyneuropathy. His subsequent follow up over 60 months showed a remarkable degree of reinnervation and the unmasking of a myelopathy. The degree of reinnervation noted suggests an axonopathy that left the other elements of the peripheral nerves relatively spared. These findings provide the most convincing evidence to date that a generalised polyneuropathy can follow electrical injury and that it results from non-thermal mechanisms such as electroporation.

(*J Neurol Neurosurg Psychiatry* 2001;71:535-537)

Keywords: electrical injury; tetraplegia after electrical injury; recovery after electrical injury

Various neurological syndromes have been described after electrical injury.¹ The brain, spinal cord, or peripheral nerves may be involved and presentation may be immediate or delayed.²⁻³ Involvement of the peripheral nervous system⁴ is well recognised to cause various mononeuropathies, which may be multiple,⁵⁻⁶ sometimes as a result of plexus involvement.⁷⁻⁸ A symmetric generalised polyneuropathy, which may be transient, is thought to occur,⁹ but remains to be convincingly documented.⁴ This paper describes a patient rendered tetraplegic after electrical injury, and the remarkable recovery that occurred over a 5 year follow up period. Our clinical and electrophysiological findings provide the most convincing evidence to date that electrical injury can cause a generalised polyneuropathy, outside the area of electrically induced thermal injury, as a result of non-thermal mechanisms including electroporation.

Case report

A 24 year old man with a history of affective disorder deliberately made contact with a 20 000 voltage AC supply. The current entered via the right arm and exited via the left elbow,

resulting in 30% full thickness burns to the right arm, left elbow, and abdomen, and flash burns to the face and neck. On initial assessment in accident and emergency it was noted that he was conscious and talking. He was unable to move his left arm and both legs. Tendon reflexes were not commented on, but both plantars were noted to be flexor. The right arm was non-viable and required amputation at the shoulder. Elsewhere, immediate escharotomies were followed by successive extensive debridement of necrotic tissue, in places down to the bone. Skin and musculocutaneous grafts were required to cover the resulting tissue deficits. He required mechanical ventilation, initially with propofol and subsequently midazolam sedation. At day 18, he required minimal sedation and it was clear that he was completely paralysed below the neck. At day 38 he had made no progress weaning from ventilation and formal neurological assessment was performed.

He was cognitively intact. There were cortical cataracts with corrected visual acuities of 6/24 on the right and 6/9 on the left. The cranial nerves were normal and the jaw jerk brisk. There was marked left periscapular and arm muscle wasting with flaccid tone and no movement in the arm or both legs. Left shoulder shrug was preserved. All tendon reflexes and the plantar responses were absent. There was distal sensory impairment to light touch and pain below the left elbow and distally in the legs. Proprioception was impaired in the left fingers, left wrist, and in the toes and ankles bilaterally. The results of neurophysiological testing showed evidence of some preserved innervation to proximal left shoulder muscles but otherwise complete denervation in the arm and leg muscles (table 1). Quadriceps muscle biopsy showed angulated fibres with no necrosis and appearances consistent with acute denervation. Magnetic resonance imaging of the spine with and without gadolinium enhancement performed 2 months after the injury showed no abnormality of the cord or roots.

Re-examination 5 months after injury showed minimal voluntary flexion of the fingers but no other movement in the left arm. Tone was increased in the legs with power against gravity for hip flexion and extension but there was still no movement distally. Knee and ankle reflexes were obtainable. The sensory

Department of
Neurology, Homerton
Hospital, Homerton
Row, London E9 6SR,
UK

J Thaventhiran
R Greenwood

Department of
Intensive Care, The St
George Hospital,
Kogarah, New South
Wales, Australia
M J O'Leary

Department of
Intensive Care, St
Bartholomew's
Hospital, West
Smithfield, London
EC1A 7BE, UK
J H Coakley

Department of Clinical
Neurophysiology
K Nagendran

King's Neurosciences
Centre, King's College
Hospital, Denmark
Hill, London SE5 9RS,
UK
M Rose

Correspondence to:
Dr R Greenwood
richard.greenwood@
homerton-hospital.thenhs.com

Received 8 September 2000
and in revised form
18 April 2001
Accepted 23 April 2001

findings remained unchanged. Signs of reinnervation in the proximal muscles of the left arm and right leg were found on neurophysiological testing.

Repeat MRI of the spinal cord 1 year after the injury again showed no abnormality. Two years after the electrical injury the patient was able to bear weight with aid, abduct the left shoulder, and flex the elbow and fingers; weaker movements were possible for elbow and finger extension. There was no movement of intrinsic hand muscles. He still required an indwelling urinary catheter. Sensory action potentials remained absent, but motor studies showed conduction in the median nerve even distally. Electromyography showed reinnervation in the left forearm and abductor pollicis brevis but not below the knee: This appeared at 33 months after injury when further reinnervation of the small hand muscles had occurred.

Five years after injury there was further improvement. The patient was able to walk unassisted 10 m with splints, flex his hips against resistance, and flex and extend his knees against gravity. Active movement at the ankle was just possible. Tone was increased bilaterally in his legs, the knee and ankle jerks were brisk, and the plantars were extensor bilaterally. Sensory testing showed that in the legs superficial sensation was reduced to the

ankles, vibration to the knees, and proprioception in the toes, and in the left hand there was an ulnar sensory deficit; he remained doubly incontinent. Further improvement was noted on neurophysiological testing with a return of the sensory action potentials in the median nerve.

Discussion

This case documents convincingly, for the first time, a severe generalised sensory-motor polyneuropathy after electrical injury and records a remarkable degree of clinical and electrophysiological recovery over a 5 year period. At one month after injury the patient presented with a flaccid tetraplegia, impaired superficial and deep sensation distally in the limbs, normal cranial nerves and cognition, and double incontinence, the only initial evidence of cord involvement. Neurophysiological assessment at this stage showed complete absence of motor and sensory potentials in the limbs and EMG showed complete denervation in muscles supplied by all the major nerves in the left arm and both legs. As his limbs had been completely paralysed on admission, there was no good reason to think that the paralysis was the result of the Guillain-Barré syndrome, or a critical illness neuromyopathy or necrotising myopathy, and muscle

Table 1 Results of electrophysiological investigations at intervals during 5 years after injury

	Electromyography				Nerve conduction studies			
	Spontaneous activity	Motor units	Interference pattern		DML (ms)	MCV (M/s)	CMAP (mV)	SNAP (Amplitude)
1 Month:								
Left biceps brachii, right tib ant, biceps femoris and rectus femoris	Profuse fibrillations and PSW	No motor units seen		Left brachial plexus	Muscle twitches from pectorals and deltoid			
Left pectoralis	Some fibrillations	Infrequent burst of normal motor units		Left median	No response			Absent
Left deltoid	Some fibrillations	Occasional motor units		Left radial	No response			Absent
Left APB and FDIO	Profuse fibrillations and PSW	No motor units seen		Right lat pop	No response			
5 Months:								
Left EDC, left and right tib ant	Profuse fibrillations and PSW	No motor units seen		Left median	No response			Absent
Right rectus femoris	Few Fibrillations	Highly complex motor units		Left radial	No response			Absent
				Right and left lat pop				
				Right and left sural				Absent
24 Months								
Left EDC	Nil.	High firing and large units	Reduced 3 mV	Left brachial plexus- biceps	8.0			
Left APB	Few fibrillations	Polyphasia and complex units	Reduced 1 mV	Left median	4.5	32 (E-W)	0.9	Absent
Left FDIO	Some fibrillations and PSW	No motor units seen		Left ulnar and radial				Absent
Right rectus femoris	Nil.	High firing and large units	Reduced 3 mV					
Left tib ant	Fibrillations	No motor units seen						
33 Months:								
Left FDIO	Few fibrillations	A single unit	Discrete	Left median	4.4		3.8	Absent
Right tib ant	Nil	Few complex units	Reduced 1–2 mV	Left ulnar	5.1		0.08	Absent
				Right post tib	8.5			
				Right lat pop	3.8		0.4	
60 Months:								
Left tib ant	Patchy areas of fibrillations and PSW	High firing polyphasic units	Reduced 3–4 mV	Left median	3.8	30 (E-W)	4.7	6 μ V
				Left sural				Absent
				Left lat pop	4.6		1.3	

APB=Abductor pollicis brevis; FDIO=first dorsal interosseous; Tib ant=tibialis anterior; EDC=extensor digitorum communis; PSW=positive sharp waves; lat Pop=lateral popliteal nerve; Post tib=posterior tibial.

biopsy and EMG sampling showed no evidence of a myopathy. The neurological damage seemed, therefore, to be solely the result of electrical injury and prognosis initially was guarded. With time, a remarkable degree of reinnervation occurred and a myelopathy not seen on MRI but previously reported at postmortem,¹⁰ was unmasked; this now causes most of the disability including the double incontinence.

Peripheral nerve dysfunction of various types is well recognised after high voltage electrical shock and may occur in 30%–50% of patients admitted to hospital after this type of injury.^{6–11} Thermal damage typically occurs to peripheral nerve fibres within the zone of local injury; often recovery is poor in these cases.^{4–11} However, peripheral nerve dysfunction may occur outside the zone of local injury and in the absence of significant damage to other tissues. Mononeuropathies, which may be multiple,^{5–6} or a brachial plexopathy,^{7–8} usually result. Recovery may be good despite initial complete denervation and absence of motor and sensory action potentials on electrophysiological testing.⁵ The occurrence of a generalised polyneuropathy is less well documented. It is clearly not the cause of an initial and rapidly resolving paraplegia, keraunoparalysis, typically occurring after lightning strike and well described by Critchley,¹⁰ and convincing electrophysiological evidence to confirm the presence of a more established polyneuropathy has never been presented. As Wilbourn⁴ has noted, whereas the patient reported by Hawkes and Thorpe⁹ clearly had multiple mononeuropathies in the upper limbs, evidence that the initial flaccid tetraparesis had a significant peripheral component was confined to pseudomyotonic discharges in the leg muscles and denervation in the tibialis anterior on sampling at 6 and 18 months after injury; motor and sensory nerve conduction studies in the legs were normal at 4 months after injury and earlier electrophysiological investigation was not performed. By contrast, our patient showed a very severe and extensive sensory-motor polyneuropathy clearly affecting the lower limbs, which were outside the observed zones of local tissue injury, as well as the upper limbs. It was initially not clearly the result of an axonopathy, and potentially recoverable, rather than a neuronopathy, resulting in a permanent tetraplegia. The subsequent remarkable degree of regeneration and recovery of the peripheral nerves in both legs as well as the left arm is entirely consistent with a generalised axonopathy, relative sparing of anterior horn cells, and regeneration after acute wallerian degeneration.

Tissue damage after electrical injury is mediated either thermally or electrically.¹² When electricity passes through a solid conductor, heat is generated in proportion to the current strength, the duration of the current

flow, and the resistance of the conductor. The greatest resistance encountered by the flow of current through the body is across the skin; this accounts for the burns and local coagulative tissue injury often seen at the points of electrical entry and exit. When electrical contact is brief, thermal injury and burns may be minimal but non-thermal injury may still cause damage by direct electrical effects causing electroconformational changes in membrane proteins, and the formation of pores in the cell membrane—electroporation.¹² The vulnerability of a cell to non-thermal electrical damage is particularly related to its length in the direction of the electrical field,^{13–14} larger transmembrane potentials being induced in longer cells. Skeletal muscle cells and, particularly, nerve axons are thus especially susceptible to this type of non-thermal damage, which may disrupt peripheral nerve axons in isolation and in the absence of significant damage to surrounding tissue.¹² The remarkable degree of peripheral nerve regeneration and recovery seen in this patient suggests that the axons were selectively injured, leaving the surrounding tissue including the Schwann cells intact to enable subsequent regeneration. This pattern of injury is consistent with acute non-thermal electrical injury. More detailed electrophysiological investigation of patients early after electrical injury may show that a generalised axonal polyneuropathy occurs more often after this type of injury than has previously been documented, and that it contributes, for example, to the tetraparesis seen early on in other cases reported in the literature.⁹

- 1 Grube BJ, Heimbach DM. Acute and delayed neurological sequelae of electrical injury. In: Lee RC, Cravalho EG, Burke JF, eds. *Electrical trauma*. Cambridge: Cambridge University Press, 1992:133–52.
- 2 Farrell DF, Starr A. Delayed neurological sequelae of electrical injuries. *Neurology* 1968;18:601–5.
- 3 Petty PG, Parkin GP. Electrical injury to the central nervous system. *Neurosurgery* 1986;19:282–4.
- 4 Wilbourn AJ. Peripheral nerve disorders in electrical and lightning injuries. *Semin Neurol* 1996;15:241–55.
- 5 Esses SI, Peters WJ. Electrical burns: pathophysiology and complications. *Can J Surg* 1981;24:11–14.
- 6 Haberal HA, Guerer S, Akman N, et al. Persistent peripheral nerve pathologies in patients with electric burns. *J Burn Care Rehabil* 1996;17:147–9.
- 7 Suematsu N, Matsura J, Atsuta Y. Brachial plexus injury caused by electrical current through the ulnar nerve. *Arch Orthop Trauma Surg* 1989;108:400–2.
- 8 Still JM, Law EJ, Duncan JW, et al. Long thoracic nerve injury due to an electric burn. *J Burn Care Rehabil* 1996;17:562–4.
- 9 Hawkes CH, Thorpe JW. Acute polyneuropathy due to lightning injury. *J Neurol Neurosurg Psychiatry* 1992;55:388–90.
- 10 Critchley M. Neurologic effects of lightning and electricity. *Lancet* 1934;4:68–72.
- 11 Ferrero I, Melendez J, Regalado J, et al. Factors influencing the sequelae of high tension electrical injury. *Burns* 1998;24:649–52.
- 12 Lee RC, Aarsvold JN, Chen W, et al. Biophysical mechanisms of cell membrane damage in electrical shock. *Semin Neurol* 1995;15:367–74.
- 13 Abramov GS, Bier M, Capelli-Schellpfeiffer M, et al. Alteration in sensory nerve function following electrical shock. *Burns* 1996;8:602–6.
- 14 Lee RC. Injury by electrical forces: pathophysiology, manifestations and therapy. *Curr Probl Surg* 1997;34:677–765.