Comment

These two cases indicate that in some cases tennis elbow may have a neurological aetiology. The first major review of the condition suggested that the main predisposing factor was repeated pronation and supination movements of the elbow leading to tears of the extensor tendon from the underlying bone.¹ This view is supported in the largest survey of tennis elbow to date,³ but other possible aetiopathogenetic mechanisms have been suggested.⁴ The repetitive movements occurring in our patients support the theory of continued minor trauma resulting in small tears of the common extensor origin.

Tennis elbow is usually a self limiting condition and treatment is by conservative means including rest, splintage, non-steroidal anti-inflammatory drugs, and local steroid injection. An appreciable minority of patients have a more chronic disability and a few of these require surgical intervention. Our two cases show that reducing repetitive movements may be of therapeutic benefit and that in a few cases this may be possible by treating an underlying neurological lesion.

DMR is grateful to the Arthritis and Rheumatism Council for continued support.

¹ Cyriax JH. The pathology and treatment of tennis elbow. *J Bone Joint Surg* 1936;18:921-40.

² Price T. Lateral epicondylitis presenting as jailer's elbow. *Br Med J* 1982; 285:1775.

³ Conrad RW, Hooper WR. Tennis elbow: its course, natural history, conservative and surgical management. *J Bone Joint Surg (Am)* 1973; 55:1177-82.

⁴ Boyd HB, McLeod AC. Tennis elbow. *J Bone Joint Surg (Am)* 1973; 55:1183-7.

(Accepted 11 March 1983)

Department of Medical Neurology, Northern General Hospital, Edinburgh

J D MITCHELL, MB, MRCP, senior registrar

Rheumatic Diseases Unit, Northern General Hospital, and Department of Medicine, Western General Hospital, University of Edinburgh

D M REID, MB, MRCP, lecturer

Correspondence to: Dr J D Mitchell.

Acute central cervical cord injury due to disco dancing

Acute central cervical cord injury is usually caused by sudden extension or occasionally by flexion movements in patients with cervical spondylosis. We report a case in a patient with this injury due to headbanging—a form of dancing involving rapid flexion-extension movements of the neck.

Case report

A 15 year old boy was admitted complaining of weakness of all four limbs. On the evening before admission he had been headbanging at a discotheque and after about five minutes of this activity he suddenly developed neck pain and tingling radiating first down his left arm and then down his right arm. Both arms suddenly became weak. He stopped dancing and over the next two hours developed mild weakness of his legs and a burning sensation over both shoulders.

On admission he had a flaccid quadriplegia with severe weakness of the right arm, no movement of the left arm, and mild weakness of the legs. All reflexes were absent in the left arm and only the biceps jerk was present in the right. The knee jerks were reduced and the ankle jerks were absent with bilateral extensor plantar responses. There was a loss of pain and temperature sensation from C4-L1 on the right and from C4-T1 on the left side. There was no sphincter disturbance. A diagnosis of acute central cervical cord injury was made. Cervical spine x ray films were normal and a myelogram showed mild enlargement of the lower cervical cord which (in width) measured 55% of the interpedicular distance at the C5 level. There was no evidence of cord compression or disc prolapse.

He was treated with dexamethasone and over the next two weeks normal power returned to his legs. Eight weeks after admission there was still severe weakness of the left arm with only slight finger flexion and extension.

There was moderate wasting of the intrinsic muscles of both hands. Power was normal in the right arm except for moderate weakness of the finger extensors and intrinsic muscles of the hand. Reflexes were normal in the right arm but the left biceps jerk remained absent with an inverted supinator jerk. Both plantar responses were flexor and dissociated sensory loss persisted on the right side from C6-C8 and from T2-T9. Pain and temperature sensation had returned to normal on the left side.

Comment

After a period of rapid flexion and extension movements of his neck this patient developed an acute central cervical cord injury characterised by weakness of all four limbs—particularly the arms—a dissociated sensory loss, and hyperalgesia of his upper chest. This occurred in the absence of any bony injury, spondylosis, or disc prolapse and in a cervical spinal canal of normal dimension. These neurological signs could result only from a central lesion in the lower cervical cord. Such a lesion would affect the corticospinal fibres, the lower cervical anterior horn cells, the spinothalamic fibres crossing the cord centrally, and also the lateral spinothalamic tracts, particularly on the left side in this patient.

The syndrome of acute central cervical cord injury was first described by Schneider,¹ who reported development of the syndrome in 15 patients after hyperextension injuries of the neck. Many of these patients had cervical spondylosis and the remainder had compression fractures or fracture dislocation of the cervical vertebrae. The lesion was thought to be due to acute anterior compression of the cervical cord resulting in central haemorrhagic necrosis.

The pattern of predominant upper limb symptoms has been attributed to the lamination of the corticospinal tracts with the fibres to the cervical segments lying medially in the cord at this level.³ This proposed lamination has been disputed,⁴ and Hopkins and Rudge⁵ suggested that corticospinal fibres were affected as they passed medially to their termination in the lower cervical cord. Injury to these fibres with damage to the anterior horn cells would explain the phenomenon of severe upper limb weakness.

In the absence of disc prolapse, spondylosis, or canal stenosis it is difficult to explain the pathogenesis of this boy's cervical cord injury. The rapid repeated flexion and extension movement of the neck with associated lengthening and shortening of the cervical cord may possibly have resulted in a shearing injury to the vascular supply and ischaemic necrosis of the central cord.

¹ Schneider RC, Cherry G, Pantek H. The syndrome of acute central cervical spinal cord injury. *J Neurosurg* 1954;11:546-77.

² Schneider RC, Thompson JM, Bebin J. The syndrome of acute central cervical spinal cord injury. *J Neurol Neurosurg Psychiatry* 1958;21:216-27.

³ Foerster O. Sympathologie der erkrankungen des rückenmarks und seiner wurzeln. In: Bumke O, Foerster O, eds. *Handbuch der neurologie*. Vol 5. Berlin: Springer, 1936:1-403.

⁴ Nathan PW, Smith MC. Long descending tracts in man. 1. Review of present knowledge. *Brain* 1955;78:248-303.

⁵ Hopkins A, Rudge P. Hyperpathia in the central cervical cord syndrome. *J Neurol Neurosurg Psychiatry* 1973;36:637-42.

(Accepted 9 March 1983)

Department of Neurology, St Vincent's Hospital, Dublin, Ireland

J REDMOND, M.D., senior house officer

A THOMPSON, MB, MRCP, registrar

M HUTCHINSON, MB, MRCP, consultant

Correspondence to: Dr A Thompson.

Correction

Infant chlamydial pneumonia

Errors occurred in the table in this short report by Dr Jane Braithwaite *et al* (30 April, p 1394-5). The correct table is given below.

Serological results (reciprocal titres in microimmunofluorescence test)

Antigen	Infant		Mother	
	IgG	IgM	IgG	IgM
<i>C trachomatis</i> A-C	64	0	256	0
<i>C trachomatis</i> D-K	256	8	64 000	0
Lymphogranuloma venereum 1-3	128	8	256	0
Herpes simplex virus 1	64	0	256	0
Herpes simplex virus 2	0	0	64	0