

## SHORT REPORT

## Reversible motor and sensory neurophysiological abnormalities in cauda equina claudication

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### Abstract

**A case of cauda equina claudication with canal stenosis is presented. Neurophysiological studies show reversible changes during symptomatic and asymptomatic phases. The somatosensory evoked potential from the tibial nerve was reduced in amplitude. Central motor conduction time (CMCT) after transcranial magnetic stimulation of the brain was reversibly prolonged in the symptomatic phase. Reversible CMCT changes have not been previously shown. The findings are discussed in the light of the pathophysiology of ischaemic nerve.**

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Nerves and nerve roots respond to insult by slowing conduction, conduction block, and a change in membrane excitability leading to mechanical sensitivity or the spontaneous generation of ectopic impulses. We describe here a 50 year old man in whom such changes were seen and in whom they were exercise related and reversible.

### Case report

The patient was involved in a road traffic accident in 1967, and sustained fracture dislocations of C4/5 and C5/6 with paralysis of the right arm and an incomplete paraplegia. The paraplegia rapidly improved and his right arm slowly returned to normal but he developed Lhermitte's sign with paraesthesia over the abdomen, perineum, and buttocks on neck flexion. He has remained impotent since the accident but has retained full sphincter control.

Two oil based myelograms three years apart showed slight backward displacement of the contrast column at the level of C5 but no evidence of progression between them.

Four years after the accident he developed progressive weakness of the right hand and both legs. A third myelogram showed narrowing of the cervical canal from C5-C7, and the next year he underwent decompressive cervical laminectomies at C5-7. He remained well until January 1991 when he developed sensory symptoms that only occurred when

standing. He described the sudden onset of dull pain in the midline of his lower back followed by a sensation of pins and needles from the waist down, together with a sensation of hot and cold water over the same area. This evolved to a sensation of urinary and faecal incontinence but he remained continent. He had difficulty controlling his legs and he developed an erection. These sensations resolved after sitting for a few minutes.

Clinical examination showed evidence of a residual paraparesis with increased reflexes, more pronounced on the right. There was minimal weakness of right wrist extensors and elbow flexion and extension. Sensory examination showed normal vibration and joint position sense with a band of pronounced hypersensitivity to pin prick and touch from T7 to T11. His peripheral pulses and circulation were normal.

A haematological and biochemical screen was normal. Cervical MRI showed evidence of the previous trauma and surgery but there was no cord compression. The thoracolumbar spine from T8 to S2 showed a progressively more severe stenosis preceding caudally below L2-3. Globules of myodil were shown in the lumbosacral sac and there was clumping of the nerve roots, particularly below L3, confirming adhesive arachnoiditis (fig 1).

Cutaneous vasomotor reflexes were measured with a laser Doppler flow meter. These provided evidence of normal pulsatility with a response to deep inspiration, from both thumbs, but there was no response from either big toes. The absence from the toes was consistent with either autonomic dysfunction or sympathetic neuropathy.

Somatosensory evoked potentials were recorded after posterior tibial nerve stimulation on three occasions, while asymptomatic, after standing for 10 minutes when the patient had become symptomatic, and after resolution of the symptoms by lying flat. These showed no change in latencies but a reduction in amplitude and the left P40 wave disappeared during the symptomatic phase with recovery afterwards (fig 2). Superimposition of two potentials in each situation confirms the constancy of the response.

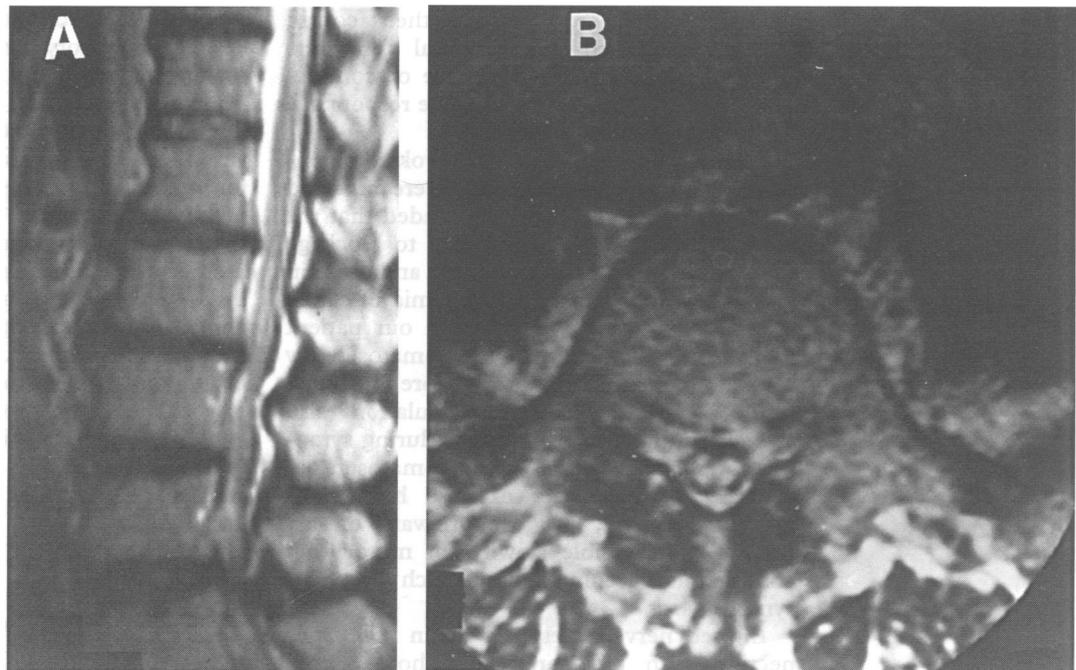
Magnetic stimulation was undertaken with a Magstim 200 with a figure of eight double 70 mm coil. Surface EMG signals were

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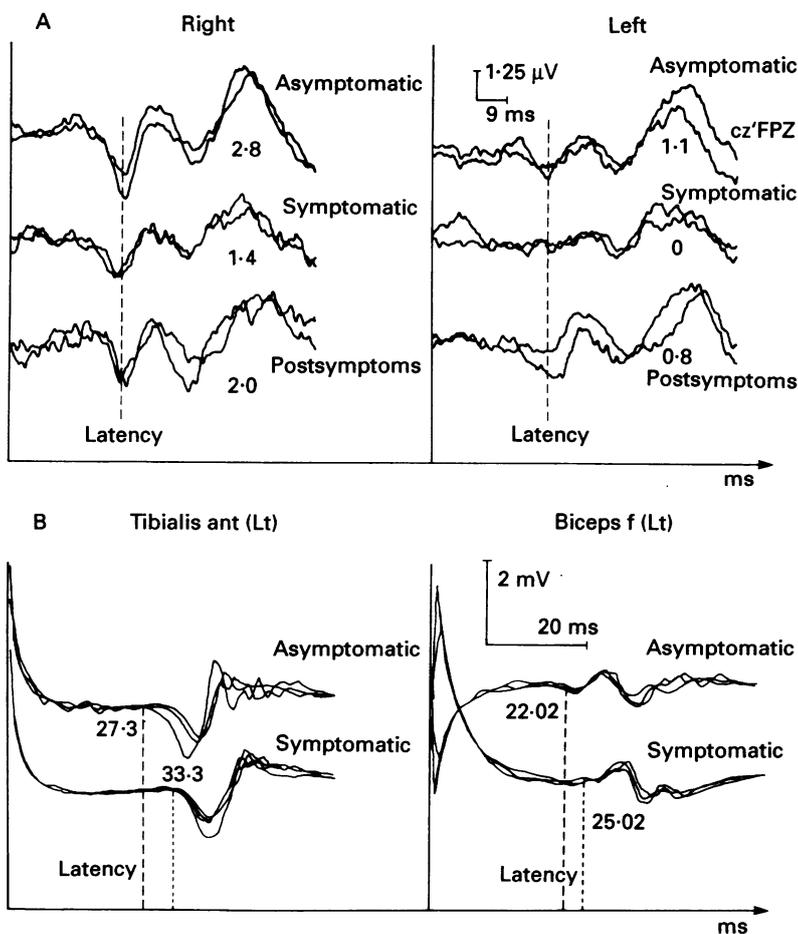
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**Figure 1** (A) Sagittal T2W image from T8 to S2, showing progressive stenosis due to facet hypertrophy. There is a small central L5/S1 disc protrusion. (B) Axial T2W images at the L4, L5 level showing globules of myxoid with clumping of the nerve roots, confirming adhesive arachnoiditis.



recorded with silver/silver chloride electrode pairs placed on five muscles: the biceps femoris, rectus femoris, tibialis anterior, lateral gastrocnemius, and adductor hallucis.



**Figure 2** (A) Posterior tibial somatosensory evoked potential recording showed absence of the left posterior tibial P40 during symptoms, and reduction in amplitude on the right with recovery afterwards. Bold figures give N33-P40 amplitude in  $\mu\text{V}$ . (B) Recording of compound muscle action potentials from cortical magnetic stimulation showing increased central motor conduction time when the patient was symptomatic.

Magnetic stimulation was applied transcranially on Cz and Fz and the compound muscle action potentials recorded simultaneously from the five muscles, which were held slightly contracted. The latency of the muscle twitch represents the total motor conduction time (cortex to muscle). The lumbosacral roots were stimulated magnetically by applying the coil over L3-S1 roots while the patient lay relaxed. This gave the peripheral motor conduction time from intervertebral foramen to the muscle.<sup>1</sup> Four responses were obtained and the latencies measured. Peripheral motor conduction time (root to muscle) was subtracted from total motor conduction time (cortex to muscle) to give central motor conduction time (CMCT cortex to intervertebral foramen).<sup>1</sup> The recordings were performed in two symptomatic and asymptomatic situations and showed increased latency in total motor conduction time and CMCT by 3–6.2 ms in all five muscles during the symptomatic phase. The peripheral conduction times were unchanged.

**Discussion**

The patient has three pathological conditions, spinal canal stenosis, arachnoiditis, and an old cervical cord injury. The typical clinical picture of lumbar spinal stenosis is intermittent claudication that includes pain, tingling, and paraesthesia in the distribution of the lower lumbar and sacral dermatomes, which appears on walking and is relieved by rest.<sup>2</sup> Similar symptomatology is occasionally seen due to prolapsed intervertebral discs.

Unusual symptoms may occur including penile erections and urinary and faecal incontinence in cauda equina claudication but they have not been described in arachnoiditis.<sup>3-7</sup> It has been postulated that increased parasympathetic tone may be responsible for these phenomena, the exertion causing stimulation

of spinal nerve roots along their course through narrowed intervertebral foramina. Foster *et al* considered blockage of conduction in the S2–4 motor roots to be responsible for these symptoms.<sup>7</sup>

Changes in somatosensory evoked potentials before and after exercise were reported by Kondo *et al*,<sup>8</sup> and they concluded that the reduction in amplitude was due to blockage of impulses, temporal dispersion, and a rise in thresholds due to relative ischaemia of cauda equina roots during exercise. In our patient there was a reduction in the somatosensory evoked potential amplitude of more than 75% after exercise. Magnetic stimulation also showed an increase in latencies during symptoms. Although changes in somatosensory evoked potential after exercise have been described before,<sup>8</sup> we are unaware of any reports of reversible slowing of motor root conduction accompanying such unusual symptoms.

Injured nerve fibres that retain their connection with the perikaryon show altered function by (a) slowed conduction, (b) conduction block in the injured segment, (c) spontaneous generation of ectopic impulses, and (d) developing mechanical sensitivity. This patient showed all four manifestations and they were reversible.

The afferent fibres show evidence of conduction block as judged by the reduction in amplitude of the somatosensory evoked potential. They also show ectopic impulse generation and mechanical sensitivity as evidenced by the paraesthesia and other sensa-

tions experienced by the patient. Slowed conduction in sensory pathways was not found.

The efferent fibres showed slowed conduction as indicated by the central motor conduction times. Ectopic impulses in the vasomotor fibres must have been responsible for his erections; these are remarkable in a man with 25 years of impotence after a spinal injury. Microneuronography would be the method to study ectopic impulses directly but this could not be done. There was no evidence of ectopic impulse generation in the motor fibres which could have generated fasciculations, spasms, or cramps. His sensation of weakness and poor control of the legs could be a manifestation of motor conduction block. It is thought that his cauda equina claudication is due to canal stenosis with arachnoiditis.

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