Focal conduction block in compression neuropathy of the proximal sciatic nerve

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1. Figure SN = at stimulation A

Abstract
Direct percutaneous stimulation of the proximal sciatic nerve with a high voltage electrical stimulator was given to a patient with acute sciatic compression neuropathy. Results clearly showed a focal conduction block at the sciatic notch, which could not be precisely localised and quantified by conventional electrodiagnostic methods.

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Sciatic neuropathies caused by external compression usually occur when persons are bedridden for a long period or sit for long periods with their buttocks pressed against a sharp edge.1,3 With conventional methods for the study of nerve conduction, it is difficult to locate the compression site on the proximal sciatic nerve because the nerve lies so deep that a long needle electrode is required to stimulate it directly.4 Moreover, needle electrode stimulation is invasive and is not acceptable for the short segment stimulation needed to detect focal conduction abnormalities.

We applied high voltage percutaneous electrical stimulation as a supramaximal shock (which cannot be given by a conventional electrical stimulator) to the proximal sciatic nerve, to detect the compression site of the sciatic neuropathy.

Case report
A 26 year old man experienced weakness of his left leg and mild sensory loss in the lateral calf when he awakened after sleeping eight hours with his left buttock compressed by the edge of a chair constructed of iron pipes. Neurological examination showed that he could not flex his left ankle or toes. Eversion and dorsiflexion of his left foot and toe extension were moderately impaired. Mild hypeaesthesia and dysesthesia were present in his left lateral calf and foot. The left ankle tendon reflex had disappeared but the patellar tendon reflex was preserved. All right leg functions were normal.

Methods
The first electrophysiological investigation was done 10 days after the onset of the symptoms. Motor nerve conduction studies were performed on both legs. The compound muscle action potentials (CMAPs) were recorded on the abductor hallucis and the extensor digitorum brevis muscles from surface electrodes in the standard belly tendon arrangement. Nerve conduction between the ankle and popliteal fossa was evaluated by a conventional method. Twenty consecutive supramaximal electrical shocks were applied to the posterior tibial and deep peroneal nerves at the ankle and the persistence of the F response was examined. The proximal sciatic nerve was stimulated with a high voltage electrical stimulator (the Digitimer D180,
In our patient, segment stimulation was given at 6 cm intervals between the sciatic notch and sacral nerve root to determine the site of the conduction abnormalities.

Nerve action potentials (NAPs) of the left sural nerve were recorded antidromically. Needle electromyography (EMG) was performed in the left anterior tibial and medial head of the gastrocnemius muscles.

During the examinations, the skin temperature was maintained above 32°C. The instrument used for the recordings and analyses was a Counterpoint (Dantec Co Ltd) electromyograph with a 30 Hz-5 kHz frequency band.

**Results**

In normal controls, the % reductions in p-p amplitude and in the p area between the sciatic notch and sacral nerve root were 8-4 (SD 6-9)% and 5-6 (8-3)% in the tibial nerve, and 6-1 (12-4)% and 0-03 (8-4)% in the peroneal nerve. None of the controls showed more than a 30% reduction in the p-p amplitude and p area of their CMAPs in this segment.

The patient’s motor nerve conductions were normal between the ankle and popliteal fossa in the tibial and peroneal nerves on both sides. Figure 1 shows results of the sciatic nerve conduction study and the F responses for the patients. The persistence of the F responses was greatly decreased in the left tibial nerve and moderately decreased in the left peroneal nerve. There were abnormal reductions in p-p amplitude and p area between the left sciatic notch and sacral nerve root. In this segment, the respective reductions in p-p amplitude and p area were 96-8% and 94-6% in the tibial nerve, and 55-0% and 50-2% in the peroneal nerve. With short segment stimulations, we located the focal conduction block as being between the sciatic notch and 6 cm proximal to it (fig 1, arrow head). There were no conduction abnormalities on the unaffected (right) side. Amplitude and conduction velocity of the NAP in the left sural nerve were normal. A needle EMG showed a severely reduced interference pattern at maximal effort with normal motor unit potentials (MUPs) in the left gastrocnemius muscle. The tibialis anterior muscles showed a mildly reduced interference pattern with normal MUPs. Spontaneous activities at rest were not found in either muscle.

Results of nerve conduction studies done two months after the onset of weakness in the patient’s left leg showed that there had been considerable improvement (fig 2 and table). The % reductions of p-p amplitude and p-
area of his CMAPs at the site of the conduction block had improved to 33.2% and 33.3%, and there was no conduction block in the left peroneal nerve. The persistence of the F response had also improved in both nerves.

Percutaneous high voltage stimulation produced only moderate discomfort and was well tolerated by the patient. No adverse side effects were seen.

**Discussion**

Abnormal motor and sensory conduction below the knee⁷ and abnormal F responses⁸ have been reported in sciatic neuropathy. For evaluating compression neuropathy, however, it is important to detect focal conduction abnormalities such as conduction block or conduction delay in the affected segment.

The percutaneous high voltage electrical stimulator developed by Merton and Morton⁶ can stimulate deeply lying nerve structures.¹⁰ We have shown that it is capable of stimulating the sciatic nerve between the knee and the sciatic notch.¹¹ We have also used it to show the presence of a focal conduction block between the sciatic notch and spinal nerve root.

The normal controls showed up to 30% reduction of p-p amplitude and −p area of CMAP between the sciatic notch and the spinal nerve root of S1. Although it is difficult to exclude the possibility that the supramaximal stimulation could not be achieved over the spinous process of S1, we think that the reduction was caused mainly by temporal dispersion and interphase cancellation of motor action potentials. The advantages of our method are its safety and non-invasiveness for short segment supramaximal stimulations. These advantages allowed repeated examinations to confirm the reproducibility of the site of conduction block and improvement in the block. Generally, greater vulnerability of the peroneal division occurs in sciatic neuropathy.¹² In our patient, however, the clinical symptoms and electrophysiological investigation showed that the tibial division was much more affected than the peroneal division. Although the reason why the tibial division was affected severely in our patient is uncertain, the tibial nerve was reported to be affected dominantly in 10% of the patients.¹³

We think that this is the first report of direct electrodiagnostic evidence of focal conduction block in sciatic nerve compression neuropathy. Percutaneous high voltage stimulation should be of great assistance in evaluating the localisation of lesions of the sciatic nerve.

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