

Peripheral nerve injuries mimicking lumbar disc herniations

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A lumbar disc herniation often gives rise to a distinctive clinical picture of pain, altered sensation, muscle weakness or atrophy and diminished reflexes; yet this "classic" picture may occur following a peripheral nerve injury to mimic a disc lesion and confuse the clinician. The authors emphasize the importance of an electrodiagnostic assessment in problem back patients and present two case histories.

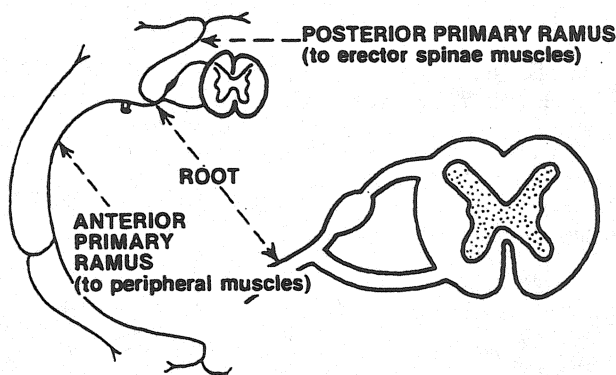


Figure 1. A segmental nerve. Compression at the root involves both anterior and posterior primary rami.

A lumbar disc herniation often gives rise to a distinctive clinical picture of pain, altered sensation, muscle weakness or atrophy and diminished reflexes; yet this "classic" picture may occur following a peripheral nerve injury to mimic a disc lesion and confuse the clinician. The clue to identification then is that pathology at root level affects the spinal nerve before it divides into its two divisions and therefore must involve both the anterior and posterior primary rami (Figure 1). When there is doubt, a complete electromyographic examination^{2,3,4} investigating muscles

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belonging to both rami (Figure 1) or even the "tender motor points procedure"¹ may resolve the issue. In neuropathy, denervation supersensitivity develops^{5,6} and a muscle may be frankly painful or the pain may be latent until induced by pressure, namely tenderness. Muscle tenderness when elicited is usually maximal at the motor point where nociceptor concentration is highest. In root lesions, tenderness is present in the muscles of the entire myotome supplied by both anterior and posterior rami.

The following two patients defied correct diagnosis until electromyography was performed. In both, contrast myelography did not reveal any indentation of the dye column. In one, surgery was averted and, in the other, surgery did not reveal any root lesion.

Although every surgeon may be aware of this problem, a *Medline* search of the literature did not reveal any previous reports.

The cases

Case 1. Initial diagnosis: Prolapsed intervertebral disc between L3 & L4 or L4 & L5.

Final diagnosis: Iatrogenic femoral nerve palsy following inguinal herniorrhaphy and postoperative infection.

This patient, a 48-year-old retort man, was injured on February 26, 1974 while attempting to move a long, heavy piece of pipe. He lost his balance and twisted his low back. He had moderate low back pain but subsequently developed weakness and atrophy of his right quadriceps muscles associated with paresthesia and partial loss of sensation over the front of his thigh. The knee jerk was depressed. The initial diagnosis by the attending physician, supported by the orthopedic surgeon, was a prolapsed lumbar disc between L3 and 4 or L4 and 5 vertebrae. Traction in bed was prescribed and the back pain resolved but the wasting of the quadriceps remained. On July 2, 1974, a lumbar myelogram was done but no abnormality was detected. An electromyographic examination on July 9 (investigating only the quadriceps muscle) suggested a right femoral nerve lesion of obscure etiology. Diabetes was ruled out and there was no history of alcoholism.

The patient was admitted to this Clinic on August 22 when, on examination, straight leg-raising was bilaterally 90°, with Lasegue's test negative. The obvious wasting in the right quadriceps amounted to 2.5 cm. at 10 cm. above the patella, compared to the left side. The right knee jerk was depressed but the right ankle jerk was normal. There was a partial loss of sensation to touch and pinprick noted over the femoral distribution on the front of the thigh. Ely's test was negative. A repeat electromyographic examination performed at the Clinic showed signs of neuropathy in the right quadriceps muscles (an increase in insertion activity, no fibrillations or fasciculations at rest; on volition, amplitude and frequency of action potentials were severely diminished accompanied by many polyphasic complexes), but the presence of normal action potentials and a full interference pattern in the erector spinae muscles (supplied by the posterior primary rami) excluded the possibility of root injury. A scar was then noted in the right inguinal region and on questioning, the patient reported that in April of 1974 he had had a right inguinal herniorrhaphy. The conclusion was that the femoral nerve was somehow damaged during this operation. (No details of operation available.)

In February 1975, the patient complained of a lump over the site of the herniorrhaphy and this subsequently gave rise to a purulent discharge. The draining eventually subsided but atrophy and weakness of the quadriceps remained. In May 1975, the inguinal region was explored and several silk sutures removed. The patient subsequently made an uneventful though incomplete recovery.

Case 2. Initial diagnosis: Prolapsed intervertebral disc protrusion between L5 & S1.

Final diagnosis: Sciatic nerve palsy following contusion at lower buttock.

This patient, a 47-year-old male mechanic, was injured on April 15, 1975 following a slip and fall onto his bottom. He developed moderate intermittent low back pain with constant left lower extremity pain and numbness. He complained of an uncomfortable aching sensation involving his left lower leg ranging from calf region into the sole associated with a burning sensation over the small toes. On prolonged walking, his left foot tended to become floppy. Roentgenograms showed some degenerative findings related to L3 and L4 vertebrae but there was no disc space narrowing.

When first seen at this Clinic on August 20, 1975, there was a good range of lumbar spine movements without back spasm, Trendelenburg's test was negative. SLR was bilaterally 90° but on the left side Lasegue's test gave rise to some sciatic nerve discomfort in the upper posterior thigh. The left ankle jerk was depressed but there were no changes in sensation. The motor points of the left short head of biceps femoris, left medial gastrocnemius and soleus were acutely tender, but there were no tender motor points in the glutei or erector spinae muscles.

Electromyography demonstrated findings of neuropathy in the left side distal muscles supplied by the sciatic nerve (insertion activity was increased; there were no signs of denervation but on volition there was a fall of action potential amplitude and frequency with many polyphasic complexes), but these findings were not noted in the gluteus maximus (not supplied by the sciatic nerve) (Figure 2) or erector spinae muscles. The indication was that there was an injury to the sciatic nerve at the level of the lower buttock where he landed striking a sharp edge and there was no evidence of pathology at root level.

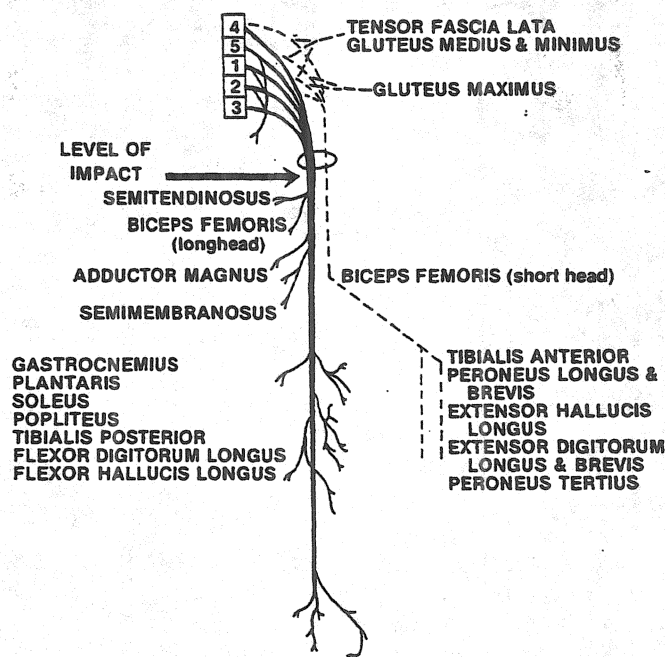


Figure 2. Diagram of sciatic nerve showing traumatic level (case 2) and distal muscles affected but sparing the glutei muscles.

Contrast myelography on November 27, 1975 was negative.

When reviewed by a neurologist on February 27, 1976, straight leg-raising was restricted on the left side, there was some reduction in calf muscle bulk and the deep tendon ankle jerk was absent. There was also some weakness of the great toe extensor. Because of these findings, the patient underwent L5 left side laminectomy on March 22, 1976 but no disc protrusion was found. Electromyography repeated on August 3, 1976 showed mild residual neuropathy in the calf muscles.

Discussion

An injury to a peripheral nerve is readily recognized when there is an obvious history or trauma, such as a penetrating injury, a fracture or contusion associated with signs of soft tissue injury, but may be overlooked when the history is non-contributory. These two patients presented with many "classic" clinical features of a lumbar disc lesion and were mistaken as having one until electromyography or the "tender motor points procedure" was performed on the muscles of both primary rami of the spinal nerve.

In electromyography the electrical activity arising from needles at rest and associated with muscle activity are detected by needle electrodes inserted into skeletal muscle. The electrical activity is displayed on a cathode-ray oscilloscope and played on a loud-speaker for simultaneous visual and auditory analysis. Normal muscle at rest shows no action potentials in contrast to the varieties of spontaneous activity which may be detected in pathological conditions. Abnormal spontaneous activity include fibrillation potentials (characteristic of denervated muscle), positive potentials (characteristic of denervation), fasciculation potentials (seen in denervated muscles and in progressive lesions of the anterior horn cells) and high frequency discharges (seen in myotonic conditions).

Contracting muscle gives rise to action potentials: on minimal volitional effort, small detectable potentials are derived from single motor units and greater voluntary effort results in more motor units discharging, until on maximum volition, numerous action potentials merge to form an "interference pattern". Abnormal potentials on volition may include polyphasic potentials which are characteristic of reinnervating muscle or shorter duration polyphasic potentials characteristic of myopathic disorders.

Although the electromyogram does not give a specific clinical diagnosis, its information integrated with results of other tests and clinical features aid in arriving at a final diagnosis. It is particularly useful in the diagnosis of lower motor neuron disease, in the detection of defects in transmission at neuromuscular junction and in the diagnosis of primary muscle disease. In a nerve root lesion, denervation potentials will be found in the muscles of that myotome. With a basic knowledge of the neuro-anatomy referable to motor nerves and muscles which they innervate, it is a straightforward procedure to determine the level of a lesion. Since the nerve root, upon leaving the spinal canal, divides into posterior and anterior rami, pathology at nerve root level must logically include findings in the muscles of both rami.

Electromyography is also of some value when the primary complaint is one of pain. If the pain arises from root or trunk involvement, it is highly unlikely for the pain fibres to be selectively involved, sparing the motor fibres, as these fibres run in the same cable. Electromyography is entirely objective as it is impossible for the patient to fake a fibrillation potential or a positive sharp wave. The electromyograph, however, cannot

make a diagnosis in regard to etiology. It does not supplant a good history, physical examination and neurological evaluation but should be regarded as an additional microphysiological physical finding when it has value in the differential diagnosis of complicated disabilities. Some authorities feel that no patient should be operated on for a herniated disc without having had an electromyogram prior to surgery. Electromyography can give a prognosis as to whether a lesion is complete or partial and it reports on the presence of denervation long before clinical signs appear. In the management of a low back patient, serial electromyographic examinations can monitor progress closely and it would seem logical, even imperative, for problematic patients to have an electrodiagnostic examination.

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