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Electrodiagnostic approach to the patient with suspected radiculopathy

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Radiculopathy is one of the most common causes for referral to the electromyography (EMG) laboratory. However, the value of electrodiagnosis in the assessment of possible radiculopathy is extremely variable. Depending on issues of patient selection, segmental level of involvement, and the electrodiagnostic (EDX) modalities used, reports have suggested both high and low correlation between EDX testing and either neuroimaging or surgical localization [1–3]. Many patients referred to the laboratory have nonspecific symptoms that represent nonneurologic disorders caused by musculoskeletal disease. Among patients with true radiculopathy, most have only radicular pain and sensory symptoms, which do not have electrophysiologic correlates measurable with standard nerve conduction studies and needle electrode examination (NEE).

EDX testing is most valuable in patients with motor or other focal neurologic deficits, such as muscle stretch reflex asymmetry. In this setting, EDX testing can aid in the segmental localization of the lesion and can provide information regarding the physiology (axon loss or conduction block), age, activity, and severity of the process. EDX testing can aid in the exclusion of other disorders masquerading as radiculopathy and may be of value in the assessment of patients with post surgical deficits, multi-segmental neurologic deficits, or multilevel intraspinal structural changes.

The approach to the patient with suspected radiculopathy should incorporate data from various sources: clinical history, general and neurologic examination, and imaging studies. In the patient with classic localizable symptoms of radiculopathy, focal neurologic deficits, and appropriately positioned structural abnormalities on neuroimaging studies, clinical decisions can be made without the confirmatory findings provided by the EMG examination. Unfortunately, the medical picture is usually not completely

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clear, especially when pain hampers the reliability of muscle strength testing at the bedside. This chapter explores the principles of electrodiagnosis in radiculopathy and discusses various testing procedures and their relative value.

Anatomy and pathophysiology

There are 31 pairs of spinal nerve roots: eight cervical, twelve thoracic, five lumbar, five sacral, and one coccygeal. Each spinal nerve root is composed of a dorsal (somatic-sensory) root and a ventral (somatic-motor) root, which join in the intraspinal region, just proximal to the neural (intervertebral) foramen (Fig. 1). In the extraspinal region, just distal to the neural foramen, the nerve root divides in two parts: a small posterior primary ramus, which innervates the paraspinal muscles and skin of the neck and trunk; and a large anterior primary ramus, which innervates the limbs and trunk, including intercostal and abdominal wall muscles. Neural foramina are formed between each pair of vertebral bodies and are bounded superiorly and inferiorly by pedicles, anteriorly by intervertebral disks and vertebral bodies, and posteriorly by facet joints (Fig. 1). The spinal nerve roots, recurrent meningeal nerves, and radicular blood vessels pass through the neural foramina. Cervical roots C1–7 enter the neural foramen above the vertebral body of the same number, such that the C3 root exits the spinal canal via the C2–3 neural foramen. Because there are only seven cervical vertebrae, the C8 root exits through the C7–T1 neural foramen. As a result,

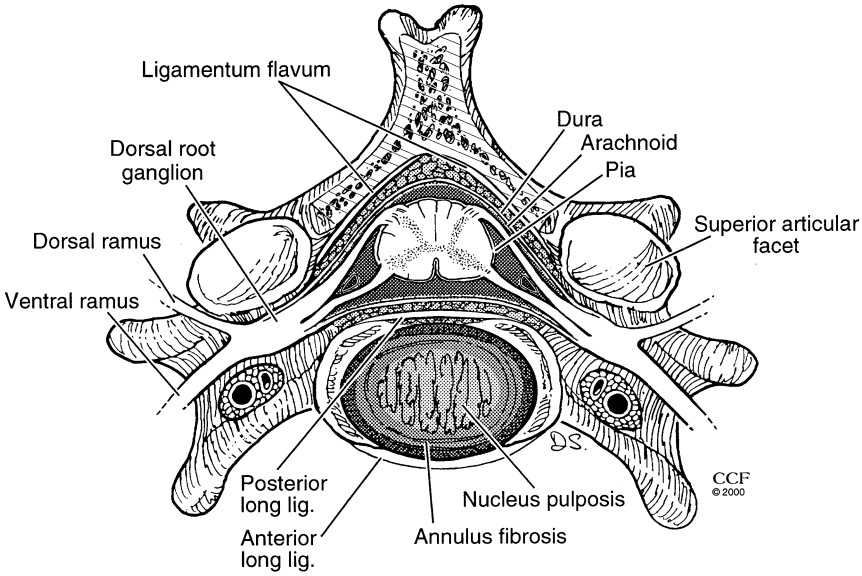


Fig. 1. Section of the cervical spine.

all thoracic, lumbar, and sacral roots exit below the vertebral body of the same number.

A capillary network derived from the radicular arteries provides the blood supply to spinal nerve roots. In the transitional region between the peripheral and central nervous system (the root entry zone) of the rat, blood vessels are positioned on the surface of rootlets and in inter-radicular spaces, but not in rootlets themselves. The density of capillaries is very high in the ventral nerve root entry zone [4]. Distal to the rootlets in rats, at the proximal and distal root levels, ventral root capillary density is higher than at the dorsal roots [5].

Cell bodies of the motor nerve fibers reside in the anterior horns of the spinal cord, whereas those of the sensory nerve fibers reside in the dorsal root ganglia (DRG). DRG are, in general, located in a protected position within the neural foramina and are therefore not intraspinal. However, at the lumbar and sacral levels, there is a tendency for DRG to reside proximal to the neural foramina, in intraspinal locations. About 3% of L3 and L4 DRG are intraspinal, about 11% to 38% of L5 DRG are intraspinal, and about 71% of S1 DRG are intraspinal, according to recent cadaver, radiographic, and magnetic resonance imaging studies [6,7]. In the cervical region, the C5 and C6 DRG also have a tendency to reside in relative intraspinal locations [8]. When DRG are in intraspinal positions, they are more exposed and therefore vulnerable to injury. With disk or bony compression of DRG, or with disruption of sensory axons distal to DRG, sensory axons degenerate and sensory nerve action potential (SNAP) amplitude loss is seen during nerve conduction studies.

Nerve root fibers are vulnerable to the same types of injury as other peripheral nerves: entrapment, compression, infiltration, ischemia, and transection. The likelihood of nerve root compression by disk rupture at lumbosacral levels may be increased by the presence of extrathecal dural and foraminal ligaments, which anchor nerve roots and reduce their plasticity [9]. Mild injury may result in focal demyelination, leading to conduction block or conduction velocity slowing along nerve root fibers. Axon loss at the root level results in Wallerian degeneration along the whole course of affected nerve fibers. Conduction block and axon loss produce symptoms and neurologic deficits if a sufficient number of nerve fibers are affected. Conduction velocity slowing alone is insufficient to produce weakness or significant sensory loss, although sensory modalities that require timed volleys of impulse transmission along their pathways, such as vibration and proprioception, can be altered.

Nerve conduction studies

Many factors reduce the sensitivity of nerve conduction studies in the diagnosis of radiculopathy. First, most radiculopathies are caused by compression from disk protrusion or spondylosis, and result in damage to only a

fraction of nerve root fibers, producing limited motor and sensory deficits. Second, in the acute setting, radiculopathy manifests itself most commonly by symptoms of pain and alteration of sensory perception. Sensory radiculopathy can only rarely be reliably localized segmentally by EDX techniques for the following reasons: symptoms of pain and paresthesia are primarily mediated through C-type sensory fibers, which are too small to be studied by routine EDX techniques; the peripheral processes of sensory root fibers remain intact with intraspinal lesions, so SNAPs remain normal; and the intraspinal location of most lesions makes it impossible to perform direct nerve conduction studies on the nerve root proximal to the damaged segment, preventing the diagnosis of conduction block or focal conduction velocity slowing along the damaged segment of the root.

NCS are an important part of the routine EDX work-up for radiculopathy as a means to exclude other disorders that may coexist with radiculopathy or may clinically masquerade as radiculopathy. Such disorders include focal mononeuropathies and polyneuropathy. The tibial H-reflex is one nerve conduction study that is useful in supporting the diagnosis of S1 radiculopathy [53].

Routine studies

Sensory NCS performed along peripheral nerve trunks are characteristically normal in radiculopathy. The SNAP amplitude, distal latency, and nerve conduction velocity should not be affected in radiculopathy. The SNAP amplitude may be abnormal if DRG are affected in the pathologic process. In pathologic processes that infiltrate or extend from the intraspinal space into the neural foramen, such as malignancy, infection, or meningioma, DRG are damaged and Wallerian degeneration along sensory axons occurs, resulting in SNAP amplitude loss. When DRG reside in an intraspinal location, as mentioned previously, they become vulnerable to compression by disk protrusion and spondylosis. Therefore, L5 radiculopathy can uncommonly be associated with loss of the superficial peroneal SNAP [10]. However, S1 radiculopathy is almost never associated with sural SNAP amplitude loss. Although S1 DRG are even more commonly intraspinal than L5 DRG, intraspinal location is caudal to the L5-S1 disk space where most compressive S1 radiculopathies occur. When nerve root damage occurs distal to the neural foramen, SNAP amplitude will be affected [10,11].

Motor nerve conduction studies are relatively insensitive in the diagnosis of motor radiculopathy for several reasons. First, most radiculopathies interrupt only a fraction of the total number of motor root fibers, whereas loss of close to 50% of motor axons in a nerve trunk is required to reliably establish a significant reduction in the compound muscle action potential (CMAP) amplitude, compared with the same response on the uninvolved side [12]. Second, to identify an abnormality of CMAP amplitude in a motor radiculopathy, the muscle belly from which the CMAP is generated must be in the myotome of the injured root. For example, a severe C8 radiculopathy

would be expected to produce some change in the ulnar CMAP amplitude, recording from either the abductor digiti minimi or the first dorsal interosseus. In the C5 myotome, the musculocutaneous and axiliary nerve trunks can be stimulated to assess CMAPs from the biceps and deltoid muscles, respectively. However, muscles in the C6 and C7 myotomes are not spatially isolated from muscles of other myotomes, and therefore CMAPs derived from them are unreliable. Table 1 outlines the screening nerve conduction studies for nonspecific arm and leg symptoms.

Late responses

Late responses are electrical stimulus evoked motor potentials that can be used to measure the travel time of propagated nerve action potentials from a distal point of electrical stimulation along a peripheral nerve trunk, proximally to the spinal cord, and then back down the limb to a muscle belly innervated by the same peripheral nerve trunk. Theoretically, they make possible the assessment of conduction through the damaged segment of a nerve root, but there are a number of limitations. First, the sensitivity is low because even severe slowing over a short segment will not usually prolong the total latency enough to be significant. Second, as long as a few nerve fibers conduct normally through a damaged segment, a normal shortest latency will be recorded, even in the presence of severe nerve root damage. Finally, late responses such as F-waves are of limited value in the diagnosis of radiculopathy because they are not recorded along sensory nerve fibers and are therefore useless in the assessment of sensory symptoms.

The F-wave was first described by McDougal and Magladery in 1950, so named because it was originally recorded from foot muscles. The F-wave is a motor response often recorded from a muscle belly after stimulation of the peripheral nerve trunk innervating the muscle. It is thought to arise from

Table 1
Screening nerve conduction studies for arm and leg pain

Arm pain		Leg pain	
Sensory	Motor	Sensory	Motor
Distal amplitude and latency	Distal latency, distal and proximal amplitudes, conduction velocity, and F latency	Distal amplitude and latency	Distal latency, distal and proximal amplitudes, conduction velocity, and F latency
Median	Median (recording from thenar eminence)	Sural	Tibial (recording from abductor hallucis)
Ulnar	Ulnar (recording from hypothenar eminence)	Tibial H reflex	Peroneal (recording from extensor digitorum brevis)
Radial			

the backfiring of motor neurons as impulses arrive antidromically from a peripheral site of nerve trunk stimulation. The F-wave occurs after the CMAP, but as the point of nerve trunk stimulation is moved more proximally, the CMAP latency lengthens and the F-wave latency shortens, indicating that the impulse eliciting the F-wave travels away from the recording electrodes toward the spinal cord before returning to activate distal muscles. Traditionally, minimal latency of at least eight consecutive discharges is measured. The absence of an F-wave response from stimulation of the median, ulnar, or tibial nerve in the presence of normal evoked CMAPs from the same muscle suggests conduction block or very recent (<5–8 d) axon loss somewhere along the nerve trunk proximal to the point of nerve stimulation. This is most often encountered in the setting of acute demyelinating polyneuropathy, but could conceivably be a feature of isolated radiculopathy when occurring in a single myotomal distribution. Peroneal F-responses are not reliably recorded in healthy individuals.

Some studies have suggested that other F-wave measurements may be more sensitive than minimal latency, including F-wave duration, mean F-latency, and chronodispersion (the interval between the shortest and longest F-latency in a consecutive series of stimuli) [13]. Several studies suggested that using these methods increases the sensitivity of F-wave analysis in L5/S1 radiculopathy to a level close to the sensitivity of the NEE [14,15]. One study reported that F-wave chronodispersion increased in patients with lumbar canal stenosis and L5/S1 root lesions after 3 minutes of standing [16]. However, F-wave changes can be seen in a number different peripheral neuropathic disorders, and therefore cannot themselves support radiculopathy.

Hoffmann first described the H-reflex in 1918. Traditionally, this response has been considered the electrophysiologic equivalent of the Achilles' tendon muscle stretch reflex. Although the contention that the H-reflex represents conduction through a monosynaptic pathway is likely to be overly simplistic, it is clear that the electrical stimulus travels orthodromically along Ia afferents to the spinal cord, where the motor neuron in the same segment is activated, producing a motor response peripherally [17,18]. When elicited from the tibial nerve with stimulation at the popliteal fossa, a motor response in the soleus-gastrocnemius muscle complex occurs. In some healthy individuals, there is discordance between the ability to elicit the H-reflex and the presence of the ankle muscle stretch reflex [19].

The H-reflex can be elicited from other nerve trunks. With corticospinal tract disease, H-reflexes can be elicited from many nerve trunks, as a result of loss of the normal central inhibitory influences on motor neuron pools. Under normal circumstances, aside from the tibial H-reflex, the H-reflex can be elicited reliably only from the median nerve, recording over the flexor carpi radialis. Abnormalities of the median H-reflex have been found in patients with C6-7 radiculopathy. One study identified 11 of 25 patients with an absence of the median H-reflex, whereas 6 of the remaining 14 patients had prolonged H-reflex latency [20]. The upper limit for the median H-reflex

has been reported as 20 milliseconds, but nomograms taking into account the effect of arm length allow more precision in diagnosis [20].

Only the tibial H-reflex is routinely used in clinical practice, where it is an extremely sensitive test for the assessment of the integrity of the tibial/S1 sensory pathway, including the intraspinal course of the S1 root. In one study, the H-reflex was absent or low in amplitude in over 80% of surgically proven cases of S1 radiculopathy [21]. It is markedly reduced in amplitude or absent in axon loss lesions affecting the S1 root and the tibial nerve at or proximal to the popliteal fossa. Reports have explored the sensitivity of the H-reflex latency compared with the H-reflex amplitude [22]. The upper limit of normal for the tibial H-reflex latency is often 34 to 35 milliseconds, but normal latency values vary depending on age, limb length, and height. Use of nomograms can narrow the normal range and potentially improve diagnostic precision [23]. Still, the most direct and reliable measurement appears to be the assessment of the side-to-side difference in H-amplitude. A report of side-to-side differences in healthy individuals suggested that an H-amplitude ratio (abnormal H-amplitude divided by the contralateral H-amplitude) of less than 0.4 is likely to be abnormal, although 1 of 47 individuals had a ratio of 0.33 [24]. In our laboratory, an additional criterion for abnormality is amplitude of less than 1 mV in individuals aged younger than 60 years.

The H-reflex is likely to show an abnormality with any disturbance of conduction through the tibial/S1 pathway. Although sensitive, the H-reflex has reduced specificity, resulting in a number of clinical limitations. First, the response is not reliably present in healthy individuals over the age of 60, although normal responses have been identified at all ages. Second, although unilateral absence of the H-reflex is abnormal at any age, bilateral absence of H-responses is often of uncertain clinical significance. Technical factors and generalized neuropathic processes can affect the H-reflex. Possible causes include obesity and inadequate penetration of the stimulus in the popliteal fossa, prior lumbar spine surgery, and peripheral polyneuropathy, especially in individuals with diabetes. Bilateral absence of the H-reflex may be the earliest EDX feature of acute demyelinating peripheral polyneuropathy (Guillain-Barré syndrome). Abnormalities along the tibial/S1 sensory or motor pathway will alter the H-response, including posterior tibial mononeuropathies proximal to the branch point of the nerve to the soleus and gastrocnemius muscles. Thus, an H-reflex abnormality is insufficient by itself to confirm the presence of an S1 radiculopathy.

Somatosensory evoked responses

Theoretically, somatosensory evoked potentials (SEPs) should be a valuable tool in the assessment of conduction abnormalities along sensory fibers at the root level. Electrical stimuli are delivered on the skin surface to a mixed sensory and motor nerve trunk, a sensory nerve trunk, or the skin

in a specific dermatomal distribution. Responses are recorded over the spine and scalp, and latencies are measured to assess the conduction time along large diameter sensory fibers across various segments of the peripheral and central conduction pathways primarily subserving proprioception and vibratory sense.

Unfortunately, several limitations diminish the value of this technique. First, amplitude measurements are too variable in healthy individuals to have clinical significance, thus the assessment of partial axon loss lesions and partial conduction block is not reliable. Second, focal slowing in the root segment is diluted by normal conduction along the rest of the sensory pathway. Third, nerve trunk stimulation often simultaneously activates nerve fibers belonging to more than one root segment, masking the abnormality in the abnormal root in question [25]. SEPs assess conduction along primarily large fiber sensory pathways that subserve proprioceptive and vibratory perception functions, not the pain and cutaneous sensation pathways that are more likely to be affected in radiculopathy. Fourth, the procedure is time consuming and subject to technical artifacts.

Given the previously mentioned limitations, SEPs obtained from nerve trunk stimulation have been shown to add little diagnostic value [26,27]. Likewise, SEPs derived from L5/S1 dermatomal stimulation have not been found to be as useful as standard EDX techniques [26,28].

Cutaneous sensory nerves have more specific and isolated root innervations, and thus SEPs derived from cutaneous nerve stimulation have a potential diagnostic advantage. Studies have been performed on the saphenous, sural, and superficial peroneal sensory nerves. Scalp-recorded cutaneous SEPs were abnormal in 57% of 28 cases of cervical and lumbosacral radiculopathy in one report, based on findings of abnormal amplitude and waveform configuration [29]. Using the same technique, Seyal, Sandhu, and Mack [30] found 20% of patients had abnormal scalp-recorded recordings; however, abnormal cases increased to approximately 50% when spine-recorded SEP latency, or response size was measured. In spite of these results, the overall correlation has not been optimal between the SEP abnormality and the clinical localization of the sensory radicular symptoms [31].

In summary, SEPs do not have either the specificity or sensitivity of other EDX techniques, such as the NEE, to recommend them for routine radiculopathy diagnosis.

Other conduction studies

A few studies have explored the value of spinal nerve root stimulation, performed at the level of the vertebral lamina. Studies assessed latency and amplitude asymmetry, and appeared to have greater reliability at the cervical levels than at the lumbosacral levels [32,33]. Two factors decrease the potential value of this technique. First, it is not clear at what site the root is being stimulated. Stimulation of the root at or distal to the neural fora-

men would not include the likely site of nerve compression for most cases of radiculopathy. Second, the procedure is uncomfortable, because it produces contraction of paraspinal muscles and proximal muscles in the shoulder or hip girdles.

Studies have also explored the value of magnetic stimulation at the spinal root level. Opinions differ regarding whether the exact site of root stimulation occurs at or distal to the neural foramen [34,35]. For measuring latency and conduction times at cortical and spinal stimulation sites, reports have suggested a correlation with clinical patterns of weakness and the ability to discriminate medially versus laterally located disc herniations that produce nerve root compression [36,37].

Needle electrode examination

General concepts

Although the needle electrode examination (NEE) assesses the motor component of radiculopathy, it is the most specific and sensitive of the EDX tests for the identification of axon loss radiculopathy. In many cases, the NEE provides information about the root level of involvement, the degree of axon loss present, the degree of ongoing motor axon loss, and the chronicity of the process. In most laboratories, patients with arm or leg pain receive a general NEE survey that samples all major root and nerve trunk distributions in the limb. If abnormalities are identified, the examination is modified to focus on the cause for the abnormality. If a symptom is in a specific region of the limb (eg, the shoulder girdle or posterior thigh), muscles in that region are examined. Tables 2 and 3 outline the screening NEE for nonspecific arm and leg symptoms, respectively.

The localization of a nerve root lesion requires the identification of neurogenic abnormalities in a distribution of muscles that shares the same root innervation but involves more than one peripheral nerve distribution. The abnormalities may include one or more of the following abnormalities: increased insertional activity in the form of positive waves or sharp spikes;

Table 2
Screening needle electrode survey for arm pain

Muscle	Root level	Nerve trunk
First dorsal interosseus	C8	Ulnar
Flexor pollicis longus	C8	Anterior interosseus (median)
Extensor indicis proprius	C8	Posterior interosseus (radial)
Pronator teres	C6–7	Median
Triceps	C6–7	Radial
Biceps	C5–6	Musculocutaneous
Deltoid	C5–6	Axillary
C7 paraspinal	Overlap	—

Table 3
Screening needle electrode survey for leg pain

Muscle	Root level	Nerve trunk
Abductor hallucis	S1	Posterior tibial
Medial gastrocnemius	S1	Posterior tibial
Biceps femoris (short head)	S1	Peroneal
Extensor digitorum brevis	L5 (S1)	Peroneal
Tibialis anterior	L5 (L4)	Peroneal
Tibialis posterior	L5	Posterior tibial
Gluteus medius	L5	Superior gluteal
Rectus femoris	L2,3,4	Femoral
S1 paraspinal	Overlap	—

abnormal spontaneous activity in the form of fibrillation potentials; reduced (neurogenic) recruitment of motor-unit firing; and features of chronic motor unit action potential (MUAP) reinnervation, such as increased duration, increased amplitude, and polyphasia.

The timing of the NEE is important. In acute radiculopathy, fibrillation potentials are the abnormality most likely to confirm the presence of a motor radiculopathy. Fibrillation potentials seldom develop before 2 weeks have elapsed from the onset of weakness and, in some patients, may not appear for 4 to 6 weeks. The most efficient use of the EMG is to delay the performance of the NEE for at least 3 weeks after the onset of motor symptoms.

Root localization by NEE

The choice of muscles for the NEE must be tailored to the specific clinical questions and symptoms, but must be comprehensive enough to maximize diagnostic certainty. The particular muscles showing neurogenic changes in the myotome in question will vary from case to case because most root lesions are partial, and not all muscles in the myotome will be affected equally. During the NEE, the more muscles identified as abnormal in the myotome, the more secure the electrodiagnosis. To make a reliable diagnosis of a single-root lesion, at least two muscles in that myotome should be found with neurogenic changes, and they should not share the same peripheral nerve innervation. In myotomes where it is possible, involvement of proximal and distal muscles should be sought to increase the certainty of the diagnosis and exclude peripheral mononeuropathy as the cause for the abnormalities. To complete the NEE in an individual with an identified single-root lesion, muscles in the myotomes framing the involved root level should be examined to verify that those myotomes are normal. For example, the biceps and first dorsal interosseus muscles should be normal in a patient with a C7 radiculopathy.

Paraspinal muscle involvement should always be sought, as it adds important support for the diagnosis of an intraspinal lesion, and rules out plexopathy and peripheral mononeuropathy as the cause of extremity mus-

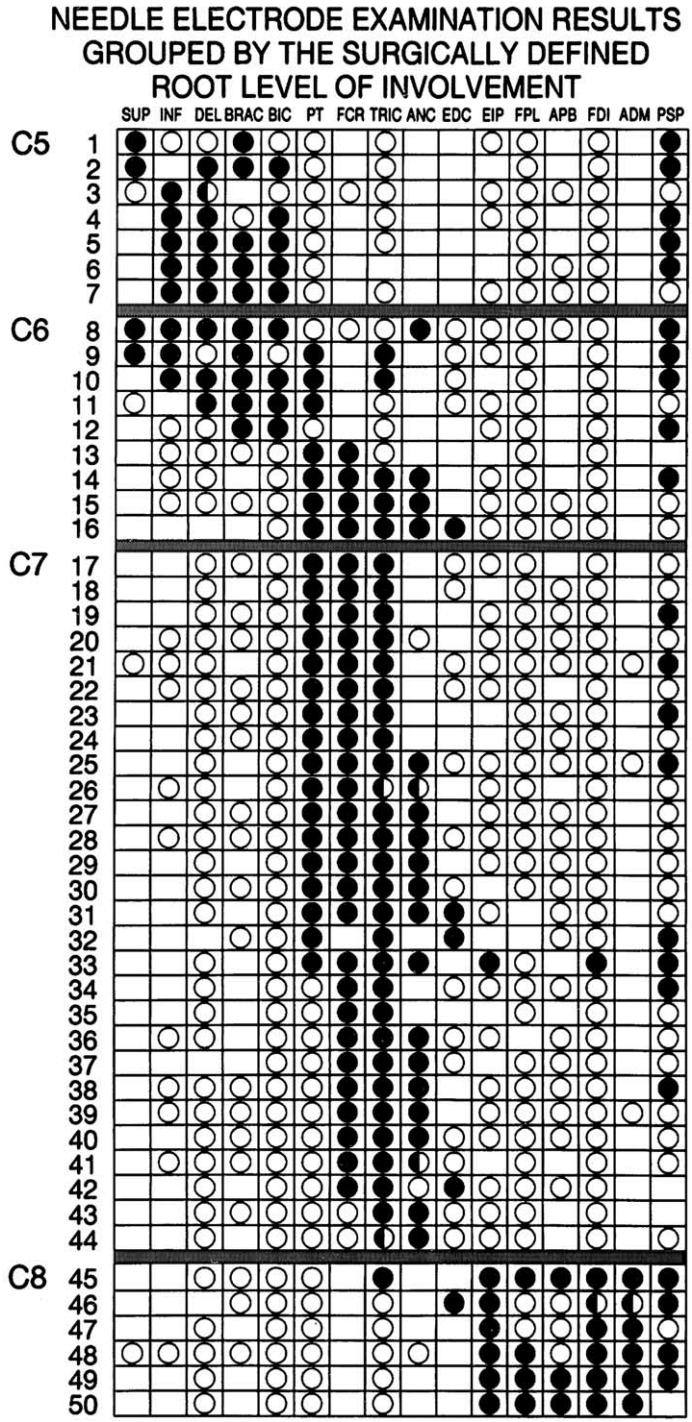
cle involvement. However, the following factors reduce their value. First, paraspinal muscle fibrillation can be seen in disorders of the root, processes affecting anterior horn cells, and muscle disorders such as necrotizing myopathy. Second, paraspinal muscle involvement cannot precisely localize the segmental level of root damage because segmental innervation of paraspinal muscles can overlap as much as four to six segments [38]. Third, clear evidence of paraspinal denervation with cervical and lumbosacral radiculopathies is seen only in approximately 50% of cases [21,39]. Causes include overlapping segmental innervation of paraspinal muscles and the tendency of muscles in close proximity to the nerve lesion site to reinnervate sooner and more completely than muscles further from the point where nerve regeneration must begin. Fourth, in paraspinal muscles that are close to a prior laminectomy site, fibrillation might persist indefinitely because of iatrogenic denervation. In routine practice, we do not examine paraspinal muscles in areas of prior surgery.

Anatomic, clinical, and EMG myotomal charts are used to correlate the pattern of EMG abnormalities in a limb with a specific root level. Tracing root and peripheral nerve innervations of muscles from cadaver studies has been used to create anatomic charts. Clinical charts have been derived by correlating the distribution of clinical muscle weakness in patients with specific traumatic lesions. Although these charts are useful, they are not entirely applicable to the NEE. Muscles are chosen for the NEE because of specific attributes of root innervation and accessibility. Some muscles, such as the anconeus, pronator teres, and brachioradialis, are not easily isolated in the clinical examination, but are easily isolated by the NEE, and are important in root localization. Thus, EMG-derived myotomal charts are useful in the electrodiagnosis of radiculopathy [21,39]. Figs. 2 and 3 are EMG-derived myotomal charts.

Defining an acute radiculopathy

In axon loss radiculopathy, determining the age of the lesion requires combining information about the duration of the symptoms with NEE attributes of both active and chronic motor axon loss. When MUAPs are of normal configuration and size, the presence of abnormal insertional or spontaneous activity in the form of trains of brief sharp spikes or positive waves indicates recent motor axon loss. Abnormal insertional activity alone suggests that the process may be only several weeks old. The presence of spontaneous activity in the form of fibrillation potentials indicates a process of at least 3 weeks duration.

Although EDX testing for radiculopathy is most valuable when significant axon loss has occurred, testing may also uncover evidence of a prominent conduction block lesion at the root level as the cause for weakness. When examining a muscle whose CMAP is of normal amplitude, the presence of a reduced recruitment pattern of MUAP activation in the absence of


NEEDLE ELECTRODE EXAMINATION RESULTS
GROUPED BY THE SURGICALLY DEFINED
ROOT LEVEL OF INVOLVEMENT
SUP INF DEL BRAC BIC PT FCR TRIC ANC EDC EIP FPL APB FDI ADM PSP
C5
C6
C7
C8
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 32 33 34 35 36 37 38 39 40 41 42 43 44 45 46 47 48 49 50

fibrillation potentials suggests conduction block. If this pattern is seen in multiple muscles of a specific myotome, a diagnosis of radiculopathy can be made. This strategy is not reliable for the diagnosis of conduction block if the onset of weakness occurs less than 4 weeks prior to the EDX study, since an acute axon loss lesion may not clearly manifest fibrillation potentials for 3 or more weeks after onset of symptoms.

Defining a chronic radiculopathy

The diagnosis of a chronic/active or a chronic/remote root lesion is based on the observation of neurogenic MUAP changes, in the presence or absence of evidence of fibrillation potentials, respectively. In the early stages of reinnervation of denervated muscle fibers, between 6 to 26 weeks after nerve root injury, collateral sprouting from surviving nerve fiber terminals gives rise to MUAPs of increased serration or polyphasia. These MUAPs may also demonstrate instability (moment-to-moment variation in configuration). As more time elapses and reinnervation becomes more complete, MUAPs lose their instability and develop the characteristic features of a chronic lesion; increased duration and amplitude. An NEE demonstrating these chronic neurogenic MUAP changes without fibrillation potentials indicates the residuals of a remote lesion. These MUAP changes are permanent, reflecting the histopathologic changes in the reinnervated muscle, and will remain unchanged unless the motor unit is injured again. After a significant motor axon loss process has occurred, MUAPs never return to their preinjury morphology.

Chronic lesions can be classified into a chronic/active category if there are both fibrillation potentials and chronic neurogenic MUAPs. In root distributions where the myotome includes muscles in both distal and proximal regions of a limb (especially the L5 and S1, and perhaps the C5-6, root distributions), the presence of a chronic and ongoing axon loss process can be more clearly defined when fibrillation potentials are seen in both distal and proximal muscles in the root distribution. In lesions where fibrillation potentials are seen in distal muscles only, the diagnosis of an ongoing axon loss process is less certain. Some inactive but severe axon loss processes never fully reinnervate, especially in muscles farthest from the injury site, leaving some muscle fibers denervated indefinitely. The NEE findings at progressive stages of axon loss radiculopathy are summarized in Table 4 .

Fig. 2. Needle electrode examination results in 50 patients with cervical radiculopathies. Closed circle: positive waves or fibrillation potentials, with or without neurogenic recruitment and motor unit changes; half-closed circle: neurogenic recruitment changes only; open circle: normal examination. Abbreviations: SUP, supraspinatus; INF, infraspinatus; DEL, deltoid; BRAC, brachioradialis; BIC, biceps; PT, pronator teres; FCR, flexor carpi radialis; TRIC, triceps; ANC, anconeus; EDC, extensor digitorum communis; EIP, extensor indicis proprius; FPL, flexor pollicis longus; APB, abductor pollicis brevis; FDI, first dorsal interosseus; ADM, abductor digiti minimi; PSP, paraspinal muscle. (*From* Levin KH, MaggianoHJ, Wilbourn AJ. Cervical radiculopathies: comparison of surgical and EMG localizaton of single –foot lesions. Neurology 1996; 46:1022-25; with permission.)

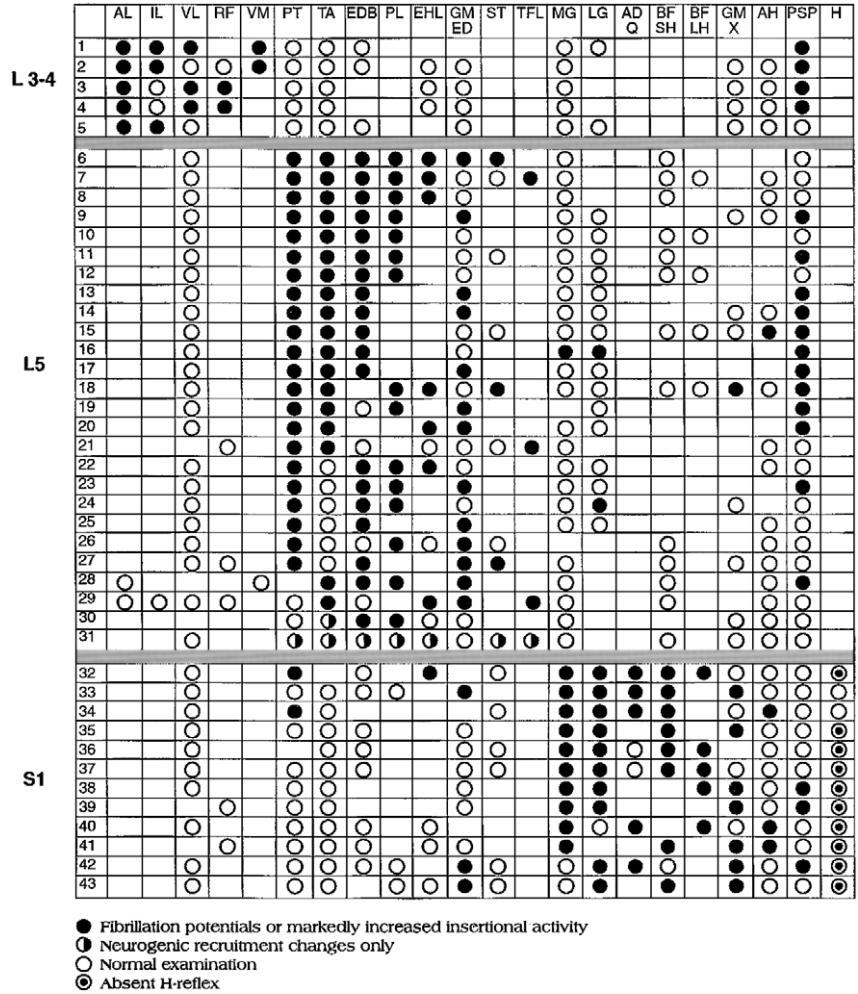


Fig. 3. Needle electrode examination results grouped by the surgically defined root level of involvement in 43 patients with lumbrosacral radiculopathies. Abbreviations: AL, adductor longus; IL, iliacus; VL, vastus lateralis; RF, rectus femoris; VM, vastus medialis; FDL, Flexor digitorum longus; PT, posterior tibialis; TA, tibialis anterior; EDB, extensor digitorum brevis; PL, peroneus longus; EHL, extensor hallucis longus; GMED, gluteus medius; ST, semitendinosus; TFL, tensor fascia lata; MG, medial gastrocnemius; LG, lateral gastrocnemius; ADQ, abductor digiti quinti; BFSH, biceps femoris (short head); GMX, gluteus maximus; AH, abductor hallucis; PSP, paraspinal; H, H-reflex.

Defining the severity of a radiculopathy

The severity of an axon loss process can be graded during the NEE by assessing the degree of motor unit loss in the root distribution. This is determined by a subjective measurement of the degree of reduced recruitment of

Table 4
Findings in the needle electrode examination at progressive stages of axon loss radiculopathy

	Recruit	Insertion	PSP	Fib	Poly/var	Neur	MTP/CRD
<3 wk	++	+/++	+	—	—	—	—
3–6 wk	++	++	++	+++	—	—	—
6–26 wk	++	+	+/–	++	+++	—	—
Chronic/active	++	—	+/–	+	++	++	—
Chronic/remote	+/++	—	—	—	—	+++	+

Abbreviations: Fib, fibrillation potentials in myotomal muscles; Insertion, abnormal insertional activity in myotomal muscles; MTP/CRD, myotonic discharges/complex repetitive discharges; Neur, neurogenic motor-unit potential changes (increased duration and amplitude); Poly/var, polyphasic motor-unit potential changes/motor-unit potential variation; PSP, paraspinal fibrillation; Recruit, neurogenic recruitment of myotomal motor units; +/–, equivocal amount; +, mild amount; ++, moderate amount; +++, greatest amount.

(*From* Levin KH. Cervical radiculopathies. In: Katirji B, Kaminski HJ, Preston DC, Ruff RL, Shapiro EB, editors. Neuromuscular disorders in clinical practice. Boston: Butterworth-Heinemann, 2002.)

motor-unit potential activation. Although there is a correlation between the degree of reduced recruitment of motor units in a neurogenic process and the degree of weakness, reduced recruitment is not necessarily due to axon loss unless the CMAP elicited from the same muscle is also reduced in amplitude. Thus, defining the severity of an axon loss radiculopathy requires evaluation of both the CMAPs in the myotome in question (when possible) and the degree of reduced recruitment of MUAP activation. Measuring the number of fibrillation potentials in a muscle is highly subjective and does not correlate as well with the degree of axon loss.

Cervical radiculopathies

The most complete clinical study of specific cervical root lesions was carried out by Yoss, Corbin, MacCarty, and Love [40]. In that study, clinical and radiographic evidence of radiculopathy was found to occur at the C7, C6, C8, and C5 levels in 70%, 19%–25%, 4%–10%, and 2% of the time, respectively. The following NEE data on individual cervical radiculopathies come from a study of isolated single-root lesions based on confirmed surgical localization [39] (Fig. 2):

- *C5 radiculopathy* produces a rather stereotyped pattern of muscle involvement, affecting the spinati, biceps, deltoid, and brachioradialis with about equal frequency, but not all of them together in any one patient. The pronator teres is never involved in C5 radiculopathy. Because the rhomboid major muscle is said to have prominent C5 innervation, it should be examined in unclear cases. The upper trapezius, with its prominent C4 innervation, is spared in C5 radiculopathy. Nerve conduction studies are not likely to be helpful, although severe lesions may be associated with axiliary and musculocutaneous CMAP amplitude loss.

- *C7 radiculopathy* produces a rather stereotyped pattern of muscle involvement, affecting particularly the triceps, but also the anconeus, flexor carpi radialis, and pronator teres. The triceps muscle is affected in essentially all cases of C7 radiculopathy. Because the extensor carpi radialis is not reliably affected in most C7 radiculopathies, it is not usually part of the routine NEE survey for radiculopathy. An important part of the clinical diagnosis of C7 radiculopathy rests upon the finding of a diminished triceps deep tendon reflex, but several studies have shown that the reflex is abnormal in less than 70% of patients. [39,40] There are no reliably performed motor nerve conduction studies that can be used to generate CMAPs from C7 innervated muscles.
- With *C6 radiculopathy*, there is no single characteristic pattern of muscle involvement. Rather, two patterns are discernible: the first is very similar to the C5 pattern, with additional involvement of triceps and pronator teres in some; and the second is similar to the C7 pattern. The pronator teres is abnormal in 80% of patients with C6 radiculopathy, but is also abnormal in 60% of the cases of C7 radiculopathy. The triceps is abnormal in over half the cases of C6 radiculopathy. Thus, significant EMG overlap occurs between C5 and C6 radiculopathy, and between C6 and C7 radiculopathy. There are no reliably performed motor nerve conduction studies that can be used to generate CMAPs from C6 innervated muscles.
- *C8 radiculopathy* produces a stereotyped pattern of muscle involvement, including the ulnar innervated muscles, extensor indicis proprius, and flexor pollicis longus. Abductor pollicis brevis is involved less often, and to a lesser degree than other muscles. Of all the root lesions, C8 radiculopathy is the most clearly identified by NEE because of the limited myotomal overlap. Nerve conduction studies are not likely to be helpful, although severe lesions may be associated with ulnar (recording from the abductor digiti minimi or first dorsal interosseus) CMAP amplitude loss.
- *T1 radiculopathy* is the least common isolated root lesion affecting the arm. Although all C8 muscles of the hand are said to have T1 contributions, the abductor pollicis brevis muscle appears to be the only muscle with predominately T1 innervation [41,42]. In a single case of T1 radiculopathy with neuroimaging and intraoperative confirmation, the EMG showed chronic and active denervation limited to the abductor pollicis brevis [42].

Lumbosacral radiculopathies

According to one large study, lumbar disk herniations leading to EMG-determined motor radiculopathy occurs at the L4–5, L5–S1, and L3–4 levels in 55%, 43%, and 2% of the time, respectively [43]. At lumbosacral levels, the anatomic localization of the site of root injury, the identification of single-root

lesions, and the accuracy of electrodiagnosis are all less successful than at the cervical levels.

First, there is the issue of the longer intraspinal course of most lumbosacral roots. All lumbar and sacral spinal nerve roots are constituted at the T12–L1 vertebral level, where the spinal cord ends as the conus medullaris. The roots then course down the canal as the cauda equina, until they exit at their respective neural foramina. Depending upon the nature and location of intraspinal compression, roots may be injured at any disk level, from the L1–L2 level to the level of their exit into the neural foramen. For example, the L5 root can be compressed by a central disk protrusion at the L2–3 or L3–L4 levels, a lateral disk protrusion at the L4–L5 level, or foraminal stenosis at the L5–S1 level. Thus, the EDX localization of a specific root lesion does not specify the vertebral level of damage. Second, because of the presence of multiple spinal nerve roots in the cauda equina, the likelihood of multiple, bilateral radiculopathies increases. This occurrence reduces EDX accuracy and introduces possible confusion with other disorders, such as peripheral polyneuropathy and motor neuron disease. Thus, the identification of a lumbosacral radiculopathy requires at least a limited evaluation of the contralateral side for evidence of concurrent lesions. The following NEE data on individual lumbosacral radiculopathies come from a study of isolated single-root lesions with active axon loss, confirmed by surgical localization [21] (Fig. 3):

- With *S1 radiculopathy*, there was a stereotyped pattern of muscle involvement, including the gastrocnemius muscles, the short and long heads of the biceps femoris, and the abductor hallucis. The biceps femoris short head, biceps femoris long head, and medial gastrocnemius were found exclusively innervated by the S1 root, although other studies have described significant L5 root innervation of these muscles [44–46]. Muscles involved in over 80% of these patients included the gastrocnemius (both medial and lateral heads) and the biceps femoris (both short and long heads). Paraspinal denervation was seen in only 25% of patients, because of the significant overlap of paraspinal segmental innervation. The gastrocnemius muscles are often difficult to voluntarily activate, making the assessment of MUAP recruitment and morphologic changes incomplete. Therefore, the identification of abnormalities in proximal muscles, such as the biceps femoris short head and long head and the gluteus maximus, is crucial for the confirmation of an S1 radiculopathy, eliminating the possibility of more distal peripheral mononeuropathies. The biceps femoris short head (BFSH) was never involved in any L5 root lesion, although some reports have described significant L5 innervation of that muscle [44].
- With *L5 radiculopathy* the NEE showed involvement of the peroneus longus and tensor fascia lata in essentially all patients, and in the flexor digitorum longus/tibialis posterior, and tibialis anterior muscles in over

75% of the patients. In this study, the L5 root exclusively innervated the tibialis anterior, although other studies have described significant L4 root innervation of that muscle [45,47,48]. About 50% of patients with L5 radiculopathy demonstrated paraspinal fibrillation potentials. NEE of the posterior tibialis or flexor digitorum longus is critical, as these are the only L5 innervated muscles below the knee not innervated by the peroneal nerve. Abnormalities in either of these muscles exclude the diagnosis of peroneal mononeuropathy. To verify the presence of an L5 radiculopathy, abnormalities should be sought in proximal L5 muscles, such as the tensor fascia lata and gluteus medius, in order to eliminate the diagnoses of sciatic and peroneal mononeuropathies. This is especially true in elderly individuals whose superficial peroneal sensory responses may be absent because of age, and in whom peroneal and sciatic mononeuropathy may not be as easily excluded.

- L2–L4 *radiculopathies* are not reliably distinguished from each other because of the overlap of innervation of the anterior thigh muscles. The problem in reliable localization is compounded by the absence of proximal and distal muscles to examine, and the low incidence of L2–L4 radiculopathies, which has prevented definitive analysis. We routinely examine the rectus femoris, vastus lateralis, iliacus, and adductor longus in patients with a question of upper lumbar radiculopathy. All these muscles appear to be equally likely to be involved at these levels, but they are seldom all involved in any single root lesion. As the adductor longus is the only muscle not innervated by the femoral nerve, its evaluation is critical for the differentiation of femoral mononeuropathy and L2–L4 radiculopathy. Paraspinal fibrillation potentials are commonly seen in patients with active axon loss radiculopathies at these segmental levels, but the paraspinal fibrillation potentials are often seen at the L5, S1, or S2 vertebral levels.

Other radicular disorders

Extraspinal radiculopathies

Extraspinal radiculopathy (focal damage to anterior primary rami) constitutes an unusual group of disorders that is difficult to diagnose. In the cervical region, two such disorders have traditionally been categorized as types of brachial plexopathy, but EDX evidence suggests that they are more likely to represent damage to extraspinal root fibers traveling in the anterior primary rami. First, *neurogenic thoracic outlet syndrome*, long considered a type of lower trunk brachial plexopathy, produces most severe axon loss in the abductor pollicis brevis muscle and the medial antebrachial cutaneous SNAP distribution, both sharing principally T1 root innervation [41]. In most cases, lower trunk/C8 structures are affected to a much lesser extent. Second, *median sternotomy brachial plexopathy*, an iatrogenic disorder that

can result from rib cage retraction during open heart surgery, is manifested by most severe axon loss in the ulnar SNAP and C8 root distribution, with little involvement of T1 innervated structures.

These two lesions show distributions of involvement that, in their purest forms, may be mutually exclusive: the abductor pollicis brevis and the medial antebrachial cutaneous response with neurogenic thoracic outlet syndrome, and C8 muscles and the ulnar sensory response with median sternotomy brachial plexopathy. However, the nerve fibers innervating all these structures travel together in the lower trunk of the brachial plexus. Therefore, neurogenic thoracic outlet syndrome and median sternotomy brachial plexopathy more likely represent, respectively, extraspinal T1 and C8 root lesions proximal to the formation of the lower trunk (Figs. 4 and 5).

Polyradiculopathies

The term polyradiculopathy indicates damage to multiple root segments simultaneously or in progressive order, occurring in a single limb or, more frequently, bilaterally, and sometimes diffusely. Its causes are diverse and not always clear. Some neurologic disorders, such as polyradiculopathy, coexist with lesions in distal peripheral nerves or lesions in the central nervous system. Following is a brief description of the most prominent causes of polyradiculopathy, and Table 5 lists causes of polyradiculopathy and their differential diagnosis.

Compressive polyradiculopathies

Spondylosis of the spine is often multifocal, and multiple roots may suffer compressive damage concurrently. This is especially true at the lumbosacral level, where spondylosis causes lumbar canal stenosis and multilevel neural foraminal stenosis. In our laboratory, we see few elderly patients with single lumbosacral root lesions, but many more with multiple simultaneous radiculopathies, often show a combination of active and more chronic features. Lumbar canal stenosis exerts compressive effects on the cauda equina, resulting in the potential for multiple root involvement. It may present clinically with weakness in a single root distribution, in several distributions, or as chronic progressive weakness of the legs in a diffuse distribution. Alternatively, lumbar canal stenosis may present as intermittent progressive fatigability and aching of the legs elicited by walking or exercise, a symptom complex known as intermittent neurogenic claudication. The EDX picture of lumbar canal stenosis is extremely variable; some patients demonstrate no changes, while others show bilateral, multilevel motor axon loss.

Regardless of the cause of lumbosacral polyradiculopathy, EDX specificity is hampered when NEE abnormalities are bilateral and confluent. In the chronic state, the NEE changes are usually most prominent in distal muscles of the myotome, shading to normal in more proximal muscles. When chronic motor axon loss spans the L5 and S1 distributions symmetrically, the

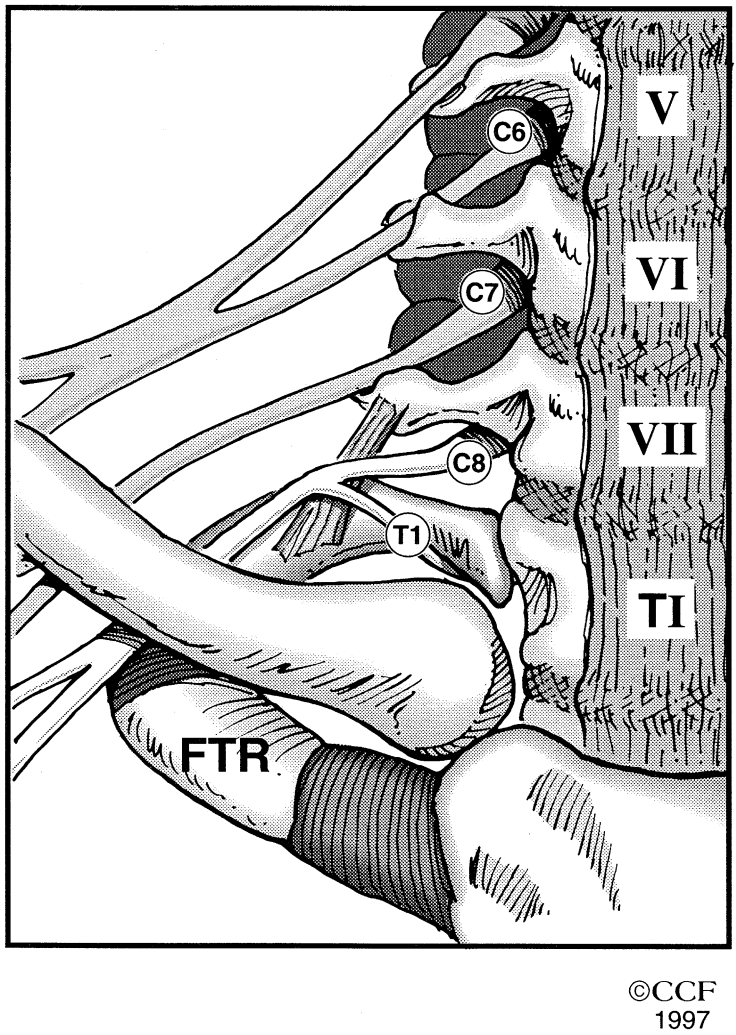


Fig. 4. Diagram depicts the likely anatomic relationship between the T1 and C8 nerve roots and the offending ligamentous band in neurogenic thoracic outlet syndrome, and shows entrapment of the T1 and C8 nerve trunks. Roman numerals indicate vertebral body levels; circled numbers indicate root levels. FTR = first thoracic rib.

electrical picture resembles the confluent changes seen in peripheral polyneuropathy. This is especially true in elderly individuals, when physiologic loss of sural and superficial peroneal sensory responses can prevent the clear distinction between axon loss peripheral polyneuropathy and a chronic or active pattern of bilateral L5 and S1 radiculopathies.

When the process is chronic and active, the EMG pattern may be difficult to distinguish from early to midstage progressive motor neuron disease

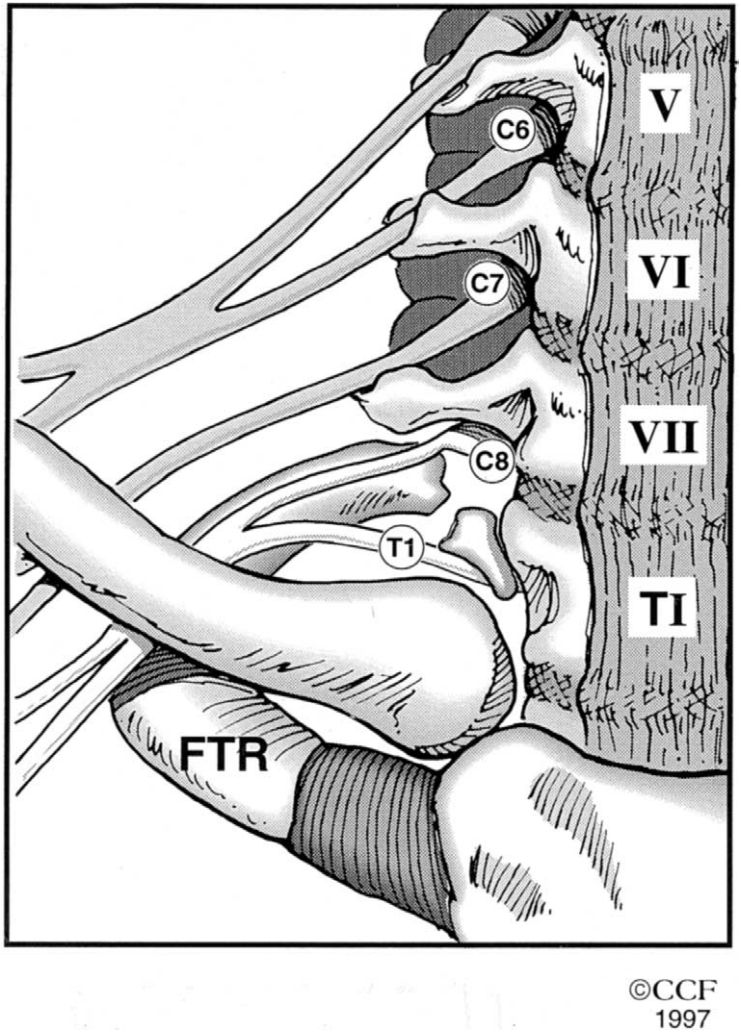


Fig. 5. Diagram depicts the anatomic relationship between the C8 nerve root and fracture of the first rib near the costotransverse articulation in a patient who has undergone median sternotomy. FTR = first thoracic rib.

(ALS) or progressive necrotizing myelopathy. In ALS, contiguous muscles of the same root or adjoining roots are more likely to show a similar degree neurogenic damage. Early to midstage ALS is also more likely to show a significant distal to proximal gradient of muscle involvement in a limb.

Diabetic polyradiculopathies

Radiculopathies caused by diabetes can occur at the thoracic, lumbar, and sacral levels, but have been rarely reported at cervical levels [50]. Approximately

Table 5
Differential diagnosis of polyradiculopathies

	Polyradiculopathy	Polyneuropathy	Myelopathy
Disorders with true root involvement			
Arachnoiditis	+		
Inflammatory polyneuropathy	+	+	
Diabetes	+	+	
HNPP	+	+	
Adrenal insufficiency	+	+	
Procainamide polyradiculoneuropathy [49]	+	+	
Spondylosis	+		+
Radiation	+		+
Vascular malformation (conus medullaris)	+		+
Malignant invasion	+	+	+
Sarcoidosis	+	+	+
Lyme disease	+	+	+
Viral infection (HZ, CMV, HSV, EBV)	+	+	+
Mycoplasma infection	+	+	+
Vasculitis	+	+	+
Angiotropic lymphoma	+	+	+
Disorders mimicking root involvement			
Porphyric polyneuropathy		+	
Alpha lipoprotein deficiency		+	+
X-linked bulbospinal neuronopathy		+	+
Motor neuron disease			+
Juvenile monomelic amyotrophy			+
Spinal cord infarction			+
Multiple sclerosis			+
Syringomyelia			+

Abbreviations: CMV, cytomegalovirus; EBV, Epstein Barr virus; HNPP, hereditary neuropathy with tendency to pressure palsy; HSV, herpes simplex virus; HZ, herpes zoster.

25% of diabetic polyradiculopathies occur in the absence of underlying peripheral polyneuropathy [51].

Thoracic radiculopathies occur either unilaterally or bilaterally. They are clinically characterized by cutaneous pain and dysesthesia in the posterior and anterior aspects of the torso in the distributions of the involved roots, and there may be weakness and bulging of the abdominal wall from denervation of rectus abdominus muscles. Thoracic radiculopathies can be confused clinically with intra-abdominal disorders. The NEE shows evidence of denervation in thoracic paraspinal muscles as well as in associated rectus abdominis muscles.

Diabetic lumbosacral radiculopathies may occur at any segmental level, but the L3–L4 levels are especially vulnerable. In one study, 15 of 16 cases

of diabetic lumbosacral radiculopathy included the L3–L4 level, and 5 of the 15 were limited to that distribution [52]. L5 root involvement occurred in 10 cases. S1 root involvement occurred in seven of these cases and all but one exhibited L5 root involvement. In only one case did L5 and S1 root involvement occur in the absence of L3–L4 root involvement. Bilateral involvement occurred in 11 cases. These data support the clinical observation that diabetic lumbosacral radiculopathy usually begins at the L3–L4 level, often increases over weeks and months to involve contiguous root levels, and eventually may involve the contralateral side.

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