ABSTRACT: The anatomy and pathophysiology of radiculopathies are reviewed, and the electrodiagnostic approaches used in evaluating patients with suspected root lesions are discussed. Such electrophysiologic procedures include motor and sensory nerve conduction studies, late-response studies, somatosensory and motor evoked potentials, nerve root stimulation, and needle electromyography. The value and limitations of these different procedures are considered. At the present time, needle electromyography is the single most useful approach. The findings in patients with radiculopathies at different levels are summarized.

AAEM MINIMONOGRAPH 32: THE ELECTRODIAGNOSTIC EXAMINATION IN PATIENTS WITH RADICULOPATHIES

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Radiculopathies, typically caused by root compression, are one of the most common causes of patient referrals to electrodiagnostic (EDX) laboratories, often exceeding even carpal tunnel syndrome. 113 Because they occur so frequently in adults, it is often forgotten that the concept of root compromise as a common cause of neck, back, and limb symptoms is relatively new. Lumbosacral radiculopathies were first described by Mixter and Barr in 1934, 87 and cervical radiculopathies by Semmes and Murphy in 1943, 106 but until the 1950s many physicians, such as Walsh, 126 were reluctant to attribute symptoms to root involvement from intervertebral disk disease. The EDX examination has been used in the assessment of radiculopathies for nearly 50 years. 12,109 In this review, both the benefits and the limitations of the various electrophysiologic procedures used in assessing radiculopathies and the typical results obtained with them will be discussed.

ANATOMY

Thirty-one pairs of spinal nerves are attached to the spinal cord by ventral and dorsal roots: 8 cervical, 12 thoracic, 5 lumbar, 5 sacral, and 1 coccygeal. Most of the axons composing the ventral roots originate from cells in the anterior and lateral gray columns of the spinal cord, whereas those composing the dorsal roots originate in the spinal, or dorsal, root ganglia (DRG). 17 The DRG are located very distally along the dorsal roots, near where the latter join the ventral roots to form the mixed spinal nerves. Hence, the DRG usually are within the ostium of the bony intervertebral foramina. 18 Immediately after they exit the intervertebral foramina, the spinal nerves terminate by dividing into posterior and anterior primary rami (also known as dorsal and ventral primary divisions). The posterior primary rami supply the skin and intrinsic or “deep” muscles of the posterior neck and trunk; depending upon the level, the anterior primary rami supply either the anterolateral aspect of the trunk or, through intervening plexuses, the limb muscles. The roots and spinal nerves vary in size. The largest are attached to the cervical and lumbar swellings of the spinal cord, and contain axons
that are distributed principally to the upper and lower extremities.17

Excluding the C8 root, the cervical roots exit superi-orly to the vertebrae with which they share a numerical designation. Thus, the C5 root traverses the intervertebral foramen between the C4 and C5 vertebrae. There are eight cervical roots, however, but only seven cervical vertebrae. Consequently, the C8 root leaves the intraspinal canal between the C7 and T1 vertebrae. Inferior to that level, each root exits caudal to the corresponding numbered vertebra; for example, the L5 root passes between the L5 and S1 vertebrae.17 The more caudal spinal roots must descend beside and then beyond the most inferior portion of the spinal cord, the conus medul-laris, to reach their exit foramina. Collectively, these roots constitute the cauda equina. The sensory fibers composing the cauda equina are “preganglionic,” since they are situated between the spinal cord and the DRG.17,134

Root and vertebra designations can be a source of confusion with radiculopathy assessments. Referring physicians frequently discuss root involvement in terms of disk levels (e.g., a C6–C7 lesion), whereas EDX physicians are concerned with the specific root affected (e.g., a C7 root lesion). Such miscommuni-cations can be a particular problem with lumbar ra-diculopathies, since roots derived from more than one segment can be compromised at a single disk level, depending on the location and size of the compressive lesion. Thus, L4, L5, and S1 radiculopathies can all occur at the L4–L5 level.134

All the muscles that share innervation from the same spinal cord segment (ventral root) compose a myotome. Almost all muscles are constituents of more than one myotome, however, because they receive innervation from two, and sometimes more, contiguous ventral roots, although usually not to the same extent. As a result, contiguous myotomes overlap, in that they have some muscles in common.142

For many years, the diagnosis of a radiculopathy in the EDX laboratory was based entirely upon finding abnormalities in a myotome distribution on needle electrode examination (NEE).54

The region of skin receiving sensory innervation from a single dorsal root constitutes a dermatome. The territories of contiguous dermatomes overlap considerably.17,142 Some of the more recently introduced electrophysiologic procedures used for radiculopathy evaluation assess the sensory component of the root; one of them does so by dermatomal stimu-lation.1,2,6,100,134

PATHOPHYSIOLOGY OF RADICULOPATHIES

Most radiculopathies ultimately are attributable to root compression, resulting either from interverte-bral disk protrusions or ruptures (generally in pa-tients less than 40–50 years of age) or from more complex degenerative changes of the spinal column, involving osseoligamentous hypertrophy (typically in patients over 50 years of age).134 Compromise of the same root produces clinical and EDX presentations that can vary among patients, suggesting that there are differences at the compression site in both the particular fibers affected and the nature of their pathologic involvement.

The clinical presentations of radiculopathies, in decreasing order of frequency, suggest injury of sensory root fibers alone; simultaneous involvement of both sensory and motor fibers; and isolated motor fiber dysfunction.44 The focal pathology and result-ing pathophysiology that occur at the lesion site de-pend on the nature of the underlying disorder and, with compressive radiculopathies, on the amount of pressure sustained by the individual fibers. When severe enough, axon loss ensues. One of the electrical manifestations of this process when it involves the extrafusal motor fibers is fibrillation potentials in an appropriate myotomal distribution. Such spontaneous activity, however, cannot be detected in some patients with radiculopathies. With some chronic root lesions in which fibrillation potentials are lacking, motor unit action potential (MUAP) changes indicate that both motor axon degeneration and subsequent regeneration have occurred.136 With many other radiculopathies, however, even acute ones studied within a few weeks of onset, no evidence of motor axon loss is detectable. This suggests that another pathophysiologic process, focal demyelination, is operative.121,140 At times, such demyelination is severe enough to produce conduction block;22 when this occurs along motor fibers, it is manifested as prominent, but usually short-lived, weakness.136 This process has been confirmed both inferentially and directly in the EDX laboratory.8,139 Lesser degrees of focal demyelination may result in conduction slowing at the injury site. The slowing may affect all axons to the same degree (synchronized slowing) or alter the speed of conduction along different axons to different degrees (desynchronized or differential slowing). The latter is the presumed mechanism whenever deep tendon reflexes are lost without accompanying clinical weakness or fixed sensory deficits.41,12,136

The clinical and EDX features of isolated compres-sive radiculopathies usually are much less severe than those seen with single root avulsions, indicating
that subtotal root involvement is characteristic of the former.\textsuperscript{140} Furthermore, the roots generally are affected more distally with typical compressive lesions than with avulsion injuries, although with both the site of injury along sensory fibers is proximal to the DRG.

**ELECTROPHYSIOLOGIC PROCEDURES USED IN DIAGNOSING RADICULOPATHIES**

**Conventional Motor and Sensory Nerve Conduction Studies.** Motor and sensory nerve conduction studies (NCS) usually are normal with single radiculopathies, for both anatomic and pathophysiologic reasons. The sensory nerve action potentials (SNAPs) rarely are affected, regardless of whether focal demyelination or axon degeneration has occurred, and even when there is a fixed sensory deficit on clinical examination. This is because of the proximal location of the lesions; even if axon degeneration ensues, both the DRG and the peripheral processes arising from them are spared, since degeneration proceeds centrally rather than peripherally.\textsuperscript{1,7,17,73}

The sole component of the motor NCS that may be affected substantially is the amplitude of the compound muscle action potential (CMAP). This occurs, however, only if the radiculopathy is causing axon degeneration, because the conduction properties of the axons distal to the root lesion—the segment that is evaluated by routine motor NCS—remain normal with focal demyelinating lesions; hence, the amplitudes, distal latencies, and conduction velocities are all unaffected. By contrast, with axon degeneration, especially of recent onset, CMAP amplitudes may be reduced because they reflect the number of viable motor axons supplying the muscle from which the recording is made.\textsuperscript{136} Thus, a C8 radiculopathy sometimes causes low-amplitude ulnar CMAPs, and an L5 radiculopathy may alter the peroneal CMAP similarly. Nonetheless, such CMAP alterations are seen infrequently with single root lesions because the muscle from which the recording is made characteristically sustains modest, rather than severe, denervation. There are two reasons for this. First, the root compromise generally is incomplete, so that it is rare for the majority of motor fibers within the root to degenerate. Second, one or more additional roots also innervate the affected muscle, and their fibers are not involved. Consequently, the CMAP amplitudes are likely to be reduced with radiculopathies only when axon loss is exceptionally severe or when more than one root supplying the muscle is compromised. These circumstances are seldom encountered with cervical radiculopathies and isolated lumbosacral radiculopathies, but are relatively frequent with cauda equina lesions caused by lumbar canal stenosis or large central disk protrusions, which may result in simultaneous compromise of L5 and S1 roots.\textsuperscript{136,140} Even when root lesions produce considerable axon loss, the motor distal latencies and conduction velocities are not slowed because they reflect conduction along surviving fibers that are conducting at their normal rate.

**Late Responses.** \textit{H Waves.} The H wave is one of the so-called late responses. It is a monosynaptic spinal reflex named after Hoffmann who first described it in 1918.\textsuperscript{27} It has been used with technical modifications to evaluate most of the lumbosacral roots that sustain compressive radiculopathies,\textsuperscript{130} and a few reports have described the utility of assessing the C6 and C7 roots by recording an H response from the flexor carpi radialis muscle while stimulating the median nerve at the elbow.\textsuperscript{89,98,102,130} Nevertheless, the only H-reflex study that enjoys wide popularity is that assessing the S1 fibers; this involves stimulating the tibial nerve while recording from the gastrocnemius–soleus muscle group.\textsuperscript{1,24,26,134}

There is almost universal agreement that H-wave studies, because of their sensitivity, may be helpful with radiculopathy assessment. There is disagreement, however, among various investigators regarding which component of the H wave, its latency or its amplitude, is most useful.\textsuperscript{89,134} Side-to-side latency differences of greater than 1.0–1.8 ms, or latencies that exceed those predicted by a nomogram, have been used to diagnose S1 radiculopathies,\textsuperscript{26,90,104,108,121} as have side-to-side amplitude differences of 50% or greater, or unelicitable responses.\textsuperscript{50,99,102,129,130,140} In the authors’ experience, reduction in the H-wave amplitude has been the most useful H-wave abnormality for detecting root lesions.

H-wave measurements offer several theoretical advantages over the traditional method of diagnosing radiculopathies, i.e., detecting fibrillation potentials in a myotomal distribution. They can become abnormal with the onset of root compression and remain abnormal until the compression ceases. Furthermore, they evaluate the function of sensory root fibers, including the segment proximal to the DRG. This is pertinent because sensory complaints are more common than motor complaints in patients with radiculopathies, suggesting more frequent or more severe compromise of sensory fibers (at least unmyelinated or small myelinated ones) than motor fibers.\textsuperscript{117}

Unfortunately, the advantages of H-wave studies are offset by certain limitations. First, in most EDX
laboratories only the S1 roots can be evaluated consistently. Second, H waves are sometimes normal with proven radiculopathies, presumably because of incomplete root involvement with sparing of the fibers that mediate the reflex. Third, an abnormal H wave is not synonymous with a radiculopathy because the reflex is mediated over a long pathway that includes peripheral nerves, plexuses, and various segments of the spinal cord, as well as the sensory and motor roots; lesions at any of these levels can cause identical H-wave abnormalities. Fourth, once H waves become unelicitable, at least because of S1 compromise, they often remain so indefinitely. Consequently, the test offers very little assistance in assessing patients with prior S1 radiculopathies, particularly those who have undergone lumbar laminectomies. Finally, H responses often are unelicitable bilaterally, not only in patients with polyneuropathies—there loss is a very sensitive, early finding in these generalized disorders—but also in otherwise normal persons over the age of 60 years.90,121,140

F Waves. F waves also are late responses. They are elicited by antidromic activation of motor neurons following peripheral nerve stimulation. Their name derives from the fact that they were first recorded from foot muscles.82 F waves vary in size, shape, and latency, and are generally less than 5% of the size of the direct muscle response.37,91 Different investigators consider different aspects of the F-wave study to be helpful. The most widely used component is the “minimal latency”: the latency of the shortest reproducible response. This is assumed to represent conduction time along one of the largest diameter motor fibers in the stimulated nerve.30,57 Other parameters that have been used are the “mean latency,” the extent of scatter (“chronodispersion”),31 and the size and persistence of the F waves.27,37

F waves have been used for almost two decades in the evaluation of radiculopathies.29,37,38,119 Initially, it was thought that they would be valuable in detecting lesions involving proximal portions of the peripheral nervous system (PNS), such as radiculopathies, that are inaccessible to the more conventional NCS. In support of this view, high yields were claimed in early reports. Unfortunately, subsequent experience has shown F-wave studies to be disappointing in patients with clinically unequivocal cervical and lumbosacral radiculopathies; frequently they are normal, and even when abnormal they often are redundant because NEE abnormalities are present as well.1,3,26,31,92,116

Several factors probably account for the low sensitivity of F waves. First, they assess the functional integrity only of motor fibers. Second, for F-wave studies to be considered abnormal, it is generally necessary for conduction slowing to be present along at least some of the fibers studied, and this may not have occurred. Third, even if such focal slowing is present, the affected portion of the motor pathway is so small compared with the total pathway being assessed that this conduction abnormality may be obscured. Fourth, F waves are elicited by stimulating the peripheral nerve innervating the muscle from which the response is recorded, and that peripheral nerve contains motor axons derived from more than one root. Hence, conduction slowing caused by an isolated lesion of one root may readily be masked by the normal conduction along fibers traversing the other, unaffected root(s). Moreover, even if an F-wave abnormality is present as a result of a root lesion, it is not possible to determine on this finding alone which one of the roots supplying the affected muscle is involved. Fifth, similar to H waves, F waves are mediated over a long peripheral segment, extending from the recorded muscle to the spinal cord. Consequently, if an F-wave abnormality is present, it is not diagnostic of a radiculopathy because the lesion may be located at any level (peripheral nerve, plexus, root, cell body in the spinal cord) along the motor pathway being tested.134 For the above reasons, the authors do not consider abnormal F waves, either alone or combined with NEE abnormalities limited to the paraspinal muscles, to be sufficient for the diagnosis of a radiculopathy, including one involving the L5 or S1 root.101

Somatosensory Evoked Potentials. Somatosensory evoked potentials (SEPs) have been used increasingly in recent years to evaluate the function of peripheral sensory pathways. They are elicited by electrical stimulation of an accessible mixed or cutaneous nerve, or the skin in the territory of a particular nerve or nerve root. Responses are recorded with either surface or needle electrodes over the scalp, over the spine, and over peripheral nerve fibers in the limb under study. Full technical details are provided elsewhere.3 In assessing the response, the latencies of the individual components and the intervals between different components (i.e., the interpeak latencies) are examined and related to height or limb length. In addition, the presence or absence of individual components and their amplitudes are studied. Changes in morphology and in the degree of dispersion of the response may also reflect a lesion of the somatosensory pathways, but defining the boundaries of normality is difficult.

SEP studies theoretically should be of help in
evaluating patients with suspected root lesions because they are the only simple means (excluding H-wave studies) of studying sensory function in proximal portions of the PNS. There are, however, several general limitations to their use in this context. First, any focal slowing of conduction due to compression of nerve fibers traversing a particular nerve root may be asked by the long distance between the site of peripheral stimulation and the site at which the responses are generated. Second, focal conduction block in some fibers may not lead to any obvious abnormality in the SEP because conduction is unaffected in the remaining fibers within the root or nerve that is stimulated. Third, there is normally some interside and intersubject variation in amplitude of SEPs so that only an extreme change—or loss of the response—reliably indicates the presence of an underlying lesion. Fourth, although a SEP abnormality may indicate a lesion in the somatosensory pathways proximal to the limb plexus, any further localizing information is very limited. Finally, SEP abnormalities provide no clue to the nature or age of a lesion in the sensory pathways.3

**Nerve Trunk Stimulation.** SEPs elicited by nerve trunk stimulation have been used to evaluate patients with suspected cervical spondylosis. In general, patients who have pain and paresthesias but lack neurologic signs have normal median, ulnar, or radial SEPs. When there are radicular signs, however, SEP abnormalities may be present, regardless of whether there is an accompanying myelopathy.32,39 Nonetheless, only rarely among patients with objective evidence of root compression is the SEP abnormal when the NEE is normal, whereas the reverse commonly occurs.145

In patients with isolated compressive lumbosacral root lesions, the peroneal-derived SEP is always normal in the authors’ experience.5,6 as might have been anticipated, because the nerve contains fibers from several different segments. Although others have reported a high incidence of abnormalities in peroneal SEPs in these situations, their failure to provide any detailed account of their criteria for abnormality36 makes interpretation of their findings difficult.

**Cutaneous Nerve Stimulation.** SEPs elicited by cutaneous nerve stimulation are more segmentally specific than those elicited by mixed nerve stimulation. Eisen and coworkers found that 16 (57%) of 28 patients studied by this technique had abnormal scalp-recorded responses, but NEE had the best diagnostic yield (75%).28 The most common SEP abnormalities were reduced amplitude and poor morphology; latency abnormalities were uncommon. Using a similar technique, Perlik and associates evaluated the L4, L5, and S1 segments of 27 patients with low-back pain, unilateral radicular symptoms, and abnormal computerized tomography (CT) scans. Radiographic changes and SEP abnormalities consistent with focal root dysfunction were found in 21 patients. Only 6 of the 21 patients had other electrophysiologic changes (e.g., abnormalities on NEE or late response testing), and they were the only patients with clinical signs. These findings, then, suggested that the SEP elicited by cutaneous nerve stimulation was very sensitive to compressive lesions of the appropriate nerve root.94 In a subsequent study that Fisher, the senior investigator in the earlier study, undertook on a larger group of 59 patients with clinical features of a lumbosacral radiculopathy, the SEP findings were compared with the operative findings and the results of other diagnostic studies. It was again found that the SEP had a higher diagnostic yield than NEE; thus, of the 38 patients with abnormal postmyelogram CT scans, 32 had abnormal SEPs, but only 11 had NEE abnormalities. This study therefore supported a useful role for this type of SEP even when conventional EDX studies are normal.124

By contrast, Seyal and coworkers found the scalp-recorded SEP elicited by cutaneous nerve stimulation to be abnormal in only 20% of patients with a lumbosacral radiculopathy and appropriate radiologic abnormalities.107 Moreover, Tans and Vrede-veld could find no significant difference between NEE and SEPs in the incidence of abnormal findings in their patients,117 while Dumitru and Dreyfuss reported that the technique had high specificity but low sensitivity.25 The findings of these latter investigators, and those from the original study by Eisen and his colleagues,28 raise doubt about the utility of SEPs elicited by cutaneous nerve stimulation in assessing patients with suspected lumbosacral radiculopathy.

**Dermatomal Stimulation.** Dermatomal SEPs have also been used to evaluate patients with isolated compressive radiculopathies; they have intuitive appeal for this purpose since the sensory fibers being assessed are derived from a single root.43,79 Some investigators have reported a very high diagnostic yield for dermatomal SEPs in the diagnosis of lumbosacral root entrapment,100 but the absence of data from normal subjects and the apparently arbitrary criteria for abnormality used by these workers engender a certain skepticism as to the value of the technique. Saal and colleagues concluded that dermatomal SEP studies are helpful in evaluating patients with upper-lumbar radiculopathies, but the published report of their experience is difficult to evalu-
predict the operative findings. Their “gold standard” for establishing the diagnosis of radiculopathy is not clear, few clinical details are furnished, there is no account of the NEE or the findings obtained thereby, the extent of the anatomic abnormalities detected by imaging studies in individual patients is not stated, and normative SEP data are not provided. Moreover, it is likely that some of the patients studied had spinal stenosis rather than isolated radiculopathies. It is also noteworthy that the SEPs were sometimes abnormal when imaging studies showed no abnormality; for uncertain reasons, Saal and colleagues attributed this to a “chemically mediated radicular syndrome” as opposed to a false-positive test finding.

Aminoff and coworkers compared the diagnostic utility of dermatomal SEPs with more conventional electrophysiologic techniques in 28 patients with clinically unequivocal L5 or S1 compressive root lesions and found that in only 7 (25%) was the final diagnosis confirmed by the SEP findings; the responses were lost or grossly attenuated in 6 patients and prolonged in latency in 1 patient. Although in 2 patients the SEPs were abnormal when other electrophysiologic studies were normal, NEE had the greatest yield, revealing denervation in a myotomal pattern in 21 (75%).

Somewhat surprisingly, Katifi and Sedgwick reported a much higher yield for dermatomal SEPs, but there are a number of problems that confound interpretation of their results. In particular, they studied patients who had diffuse rather than restricted lumbosacral disease, and they used more generous criteria for defining abnormality. The published data of Katifi and Sedgwick were reviewed by Aminoff and Goodin in light of the operative findings used to establish a definite diagnosis. It was found not only that the dermatomal SEPs incorrectly suggested abnormalities in 12 roots that were unaffected at operation, but also that in only 4 of the 21 patients did the SEPs accurately predict the operative findings.

Others also have reported that dermatomal SEP studies add little to the diagnosis of radiculopathy, and that they are less sensitive than the NEE. Du-nimtru and Dreyfuss have recently examined the clinical utility of dermatomal SEPs in 20 patients with unilateral involvement of either the L5 or S1 nerve root (but not both roots) as determined by the history, physical findings, results of imaging studies, and presence of EDX abnormalities. They compared their findings with those in a control population and found that dermatomal SEPs were of inadequate sensitivity and specificity to justify their use for diagnostic purposes. Snowden and his colleagues have also used dermatomal SEPs to evaluate root function. They have suggested that in patients with spinal stenosis, in whom lesions are likely to extend for a greater distance along the length of the roots, the findings may be important in indicating the extent of radicular involvement, but a formal comparison with the results of NEE has not yet been published. In any case, they concur with the view that this technique is not helpful in detecting isolated root involvement.

In summary, SEPs evoked by nerve trunk stimulation are diagnostically unhelpful, while both cutaneous nerve and dermatomal SEPs are relatively insensitive in patients with clinically unequivocal root lesions, making it unlikely that they will be of any clinical utility when the diagnosis is less clear.

**Motor Evoked Potentials.** The responses that can be recorded from muscle after stimulation of the central nervous system or proximal nerve roots are designated motor evoked potentials (MEPs). Either electrical or magnetic stimulation can be used for this purpose. A procedure for assessing cervical radiculopathies depends on stimulating the motor roots electrically via a monopolar needle electrode inserted into the paraspinal muscles near them, while recording from selected muscles representative of the various myotomes in the upper extremities. Nerve root stimulation studies are performed bilaterally, seeking amplitude and minimal latency asymmetries of the CMAPs. It has been claimed that this procedure is more sensitive than other techniques for detecting compressive radiculopathy, but this awaits further confirmation, particularly since one report suggests that with this technique the motor root fibers are being stimulated “at their exit from the spinal canal” and, hence, distal rather than proximal to the lesion site. A similar technique for lumbosacral root stimulation was originally described several years ago (although for lumbosacral plexus assessment), and is still used occasionally. A more invasive procedure has also been described in which needle stimulating and recording electrodes are used, with the former inserted into the spinal canal (extrathecally). In an early report, Tabaraud and colleagues recorded the responses from the tibialis anterior and soleus muscles using a somewhat different approach, namely after percutaneous electrical stimulation over the lumbar spine, in 45 patients with radiologically verified L5 or S1 disk disease that was causing unilateral symptoms; they compared the findings on the two sides and also to the findings elicited in 25 normal control subjects. The MEP latency on the symptomatic side...
was prolonged by more than three standard deviations from the normal mean interside latency difference in 72% and 66% of those with L5 and S1 lesions, respectively. This approach, however, has largely been discarded in favor of magnetic stimulation, which causes less discomfort to patients.

Magnetic stimulation has been used to excite the cervical or lumbosacral nerve roots. When used in the cervical region, Evans and coworkers found no difference in the mean amplitude or area of the MEPs compared to the responses elicited by electrical stimulation with a monopolar needle electrode. However, the precise site of stimulation with the magnetic coil stimulator is uncertain; some investigators believe excitation may occur more distally than with electrical stimulation; others have suggested that root excitation occurs preferentially at the neural foramen. In regard to lumbosacral root assessment, Chokroverty and colleagues used magnetic stimulation over the lumbar spine and found the MEPs recorded from muscles innervated by affected roots were delayed in latency and attenuated in amplitude. Despite such encouraging preliminary reports, there has been little follow-up on this type of assessment, although two groups have found a disappointingly low yield from MEPs elicited by magnetic stimulation in patients with lumbosacral radiculopathy, and it is difficult to stimulate the nerve roots supramaximally by this means. Consequently, the approach remains experimental, and its clinical relevance is uncertain.

Needle Electrode Examination. Although the NEE is by far the oldest electrophysiologic method used to evaluate patients with suspected radiculopathies, it still is the single most useful procedure, having a considerably higher diagnostic yield than other techniques. With the NEE, only motor root fibers are assessed and then principally for the occurrence of axon loss. Recognition and localization by this procedure entail detecting abnormalities, most often fibrillation potentials, in the muscles of a particular myotome (i.e., in a segmental distribution), while demonstrating the absence of similar abnormalities in adjacent myotomes. The NEE with suspected radiculopathies must be fairly extensive; at the very least, several muscles must be examined. An adequate “radiculopathy screen” requires assessment of five to seven muscles (at least), including the paraspinals; however, there is no consensus regarding exactly which muscles should be included in the survey. Nonetheless, muscles are generally sampled from all the myotomes of the limb, with emphasis on those of the myotome in question and those overlapping it. A study is considered positive if abnormalities are present in two or more muscles that receive innervation from the same root, preferably via different peripheral nerves, but are not detected in muscles innervated by the normal roots adjacent to the involved one; for example, an L5 radiculopathy may produce abnormalities in the tibialis anterior (L4 and L5 root innervation via the common peroneal nerve) and tibialis posterior (L5 and S1 innervation via the tibial nerve), but not in the vastus lateralis (L2, L3, and L4 innervation via the femoral nerve) or the medial gastrocnemius (S1 and S2 innervation via the tibial nerve). Thus, the only innervation common to the affected muscles is that derived from the L5 spinal cord segment, L5 root, sacral plexus, and sciatic nerve. The latter two possibilities are eliminated if similar abnormalities also are found in the lumbosacral paraspinal muscles, and a sciatic nerve lesion also is excluded if muscles innervated by the gluteal nerves (glutei; tensor fascia lata) are abnormal. (It is impractical, with both L5 and the S1 radiculopathies, to require completely separate peripheral nerve innervation for the involved muscles because almost all the limb muscles innervated by these two roots share intermediate peripheral nerve innervation via the sciatic nerve.)

Myotomal Maps. Many myotomal maps have been published, derived from various sources, including autopsies, clinical neuroimaging, and electrophysiologic (some intraoperative) studies. Nonetheless, the root (or primary root) innervation of many muscles remains debatable. The myotomal charts used in this article (Figs. 1 and 2) represent the consensus views of the authors. All myotomal maps are, however, best considered as “approximate guides” only; there is considerable individual variation in the roots innervating a particular muscle and in those roots providing its principal innervation.

Paraspinal Muscles. Performing the NEE on the paraspinal muscles is necessary for proper assessment of radiculopathy. Fibrillation potentials in paraspinal muscles indicate axonal lesions in the posterior primary ramus and thus within or near the intraspinal canal (i.e., proximal to the plexus). This important localizing point was first discussed in 1951 by Woods and Shea. The multifidus is the deepest paraspinal muscle and is the only one considered to have monosegmental innervation. Because of overlapping innervation of most paraspinal muscles, fibrillation potentials are not segmentally specific, often occurring caudal to the presumed anatomic level of a root lesion. Consequently, ad-
equate NEE of the paraspinal muscles usually requires sampling both at and inferior to the level of the lesion.

Paraspinal muscle assessment in patients with suspected radiculopathies has varied in importance in different series. Thus, the occurrence of paraspinal fibrillation potentials or insertional positive sharp waves as the sole NEE abnormality has ranged between less than 5% and over 40% of patients studied in different series. Such variation may be due to study methodology. For example, one study involved NEEs on patients with symptoms present for less than 14 days, a time period that favors the occurrence of paraspinal abnormalities. In the authors’ experience, if the NEE is performed no sooner than 3 weeks after symptom onset, it is rare to find fibrillation potentials in the paraspinal but not the limb muscles.

Although paraspinal muscle evaluation is unquestionably important in radiculopathy assessment, it has limitations. First, the paraspinal muscles may not show fibrillation potentials with

![FIGURE 1. Myotomal chart of the upper extremity (see text for details).](image1)

![FIGURE 2. Myotomal chart of the lower extremity (see text for details).](image2)
proven radiculopathies, either because the axons supplying them were spared or because the affected muscles have been reinnervated. Second, paraspinal fibrillation potentials and insertional positive sharp waves may be seen in asymptomatic persons, at least in the lumbosacral region. Fibrillation potentials and insertional positive sharp waves occur in lumbar paraspinal muscles in between 14.5 and 48% of normal persons studied. \(^{22,88}\) These abnormalities are age related; most are seen in persons over 40 years old and may reflect degenerative changes resulting from normal aging. \(^{22,88}\) Third, paraspinal fibrillation potentials are not pathognomonic of radiculopathy; they may be found in motor neuron disease, localized paraspinal muscle trauma (as can occur with epidural blocks and lumbar punctures), metastases to the posterior primary rami and paraspinal muscles, various generalized (especially inflammatory and toxic) myopathies, and particularly, diabetes mellitus. \(^{1,2,26,65,135,136}\) Fibrillation potentials, or at least insertional positive sharp waves, may occur throughout the paraspinal muscles in diabetics, but are typically most abundant in the lumbar region. \(^{135}\)

Fourth, NEE of the paraspinal muscles may be suboptimal because satisfactory muscle relaxation is not achieved, particularly in the thoracic region. (Weddell and colleagues, in 1944, reported that satisfactory assessment of cervical paraspinal muscles was impossible for this reason. \(^{128}\) ) Fifth, the extensive overlap of the root innervation of the paraspinal muscles makes it difficult to determine the specific root involved by the distribution of fibrillation potentials. Finally, NEE of the paraspinal muscles is of questionable value in the setting of posterior spinal surgery because any abnormalities may relate to perioperative damage to the posterior primary rami. \(^{105}\) Therefore, a radiculopathy cannot be excluded by the absence of paraspinal fibrillation potentials, or diagnosed solely by their presence.

**NEE Abnormalities with Radiculopathies.** The presence of fibrillation potentials in a myotomal distribution is reliable evidence of a radiculopathy. \(^{1,26,41,68,77}\) Other abnormalities may occur in a root distribution, but have different significance. All myotomal abnormalities can be divided into spontaneous activity, abnormal insertional activity, and MUAP changes.

**Fibrillation Potentials.** Fibrillations in a myotomal distribution may be the only electrophysiologic abnormality in radiculopathies because so few motor fibers have degenerated that MUAP changes cannot be detected. \(^{56,112,134}\) They usually occur in a proximal-to-distal sequence. Following onset of an acute lesion, they may be found after 6 or 7 days in the paraspinal muscles, but may not appear in limb muscles for 5–6 weeks after onset of the lesion. \(^{1,26,68}\) Many EDX physicians once required fibrillation to be present in all muscles of the myotome before radiculopathy could be diagnosed, \(^{84}\) but total myotomal involvement is rare. First, individual variations in muscle innervation occur and may exclude a particular muscle from a myotome to which it customarily belongs. Second, root compromise usually is incomplete and, typically, only a minority of the affected axons are injured so severely that they degenerate. Therefore, the nerve fibers supplying some of the muscles in the myotome are spared. In the authors’ experience, fibrillation potentials are more likely to be found in the distal than proximal muscles. Third, the NEE often is performed several months after the onset of root compression, and muscle fibers in the more proximal muscles of the myotome have had time in the interim to become reinnervated, usually by collateral sprouting. As a result, fibrillation potentials are found in their widest distribution early in the course of radiculopathies; with time, fewer muscles of the myotome, most often the more distal ones, contain them. \(^{64,77,140}\)

**Other Spontaneous Activity.** Fasciculation potentials and complex repetitive discharges occurring in a myotomal distribution are rare but satisfy criteria for diagnosing a root lesion. \(^{26,27}\) Fasciculation potentials appear in a segmental distribution so seldom as to be of no practical value. \(^{16,136}\) Whenever they are the sole type of spontaneous activity found in muscles innervated by the same root, a more extensive NEE assessment of multiple limbs is needed, because they may represent generalized benign fasciculations or amyotrophic lateral sclerosis (ALS). \(^{140}\) Moreover, fasciculation potentials occur in the normal abductor hallucis muscle, so their presence in that muscle is not helpful in the diagnosis of S1 radiculopathies. \(^{134}\) Complex repetitive discharges are also rare with radiculopathies and—when present—indicate chronicity. Furthermore, they typically are then very restricted in distribution, often being present in just one or two muscles of the myotome (e.g., the paraspinal muscles with chronic cervical radiculopathies). Also, complex repetitive discharges may occur in the iliacus muscle, so their presence in that muscle is not helpful in the diagnosis of L2 and L3 radiculopathies. \(^{59,140}\)

**Abnormal Insertional Activity.** Although the amount of insertional activity may be deemed abnormal soon after onset of symptoms, this is a nonspecific finding that must be interpreted with caution. Insertional positive sharp-wave activity may be encountered in a myotomal distribution before spon-
taneous fibrillation potentials become apparent, but other insertional activity may be mistaken for it, such as brief myotonic potentials and a benign type of nonspecific increased insertional activity.\textsuperscript{84,131,141,144} Moreover, the interval before it appears after onset of the lesion is variable, as is its distribution within the muscles of the myotome. Accordingly, NEEs should not be performed on patients so soon after onset of symptoms, and caution is advised in relying on changes in insertional activity alone for diagnosing a radiculopathy.

\textit{Abnormal MUAP Recruitment.} Single motor units usually fire at 5–10 Hz. In neurogenic lesions there are fewer units firing, but they do so at faster rates, during activity.\textsuperscript{69} Firing rate abnormalities occur at the onset of clinical symptoms. Therefore, detecting such a firing pattern in a myotomal distribution has been recommended as one method of diagnosing radiculopathies early in their course, before fibrillation potentials have had the opportunity to develop.\textsuperscript{16,27,53,59} There are several limitations of this approach. First, a single radiculopathy seldom compromises enough of the motor axons supplying a particular muscle to produce this MUAP firing pattern. Second, not all myotomal muscles are affected. For example, the tibialis anterior muscle may be the only muscle affected in an L5 radiculopathy. Therefore, the changes are too limited for diagnosis. Lastly, recognizing changes in MUAP firing pattern requires considerable experience. Nonetheless, this method is important for distinguishing a lower motor neuron MUAP firing pattern from upper motor neuron patterns or submaximal voluntary effort.

\textit{Chronic Neurogenic MUAP Changes.} Abnormalities of MUAP configuration are frequent in chronic radiculopathies. The MUAPs are of increased duration, reflecting both denervation and subsequent reinnervation via collateral sprouting. They usually are polyphasic as well, but the latter change is irrelevant for diagnostic purposes. Chronic neurogenic MUAP changes may be the only NEE abnormality in chronic radiculopathies. Because they may represent static axonal loss, they cannot be considered evidence of ongoing active root damage.\textsuperscript{140} Just as with fibrillation potentials, chronic neurogenic MUAP changes seen with L5 and S1 radiculopathies often are limited to, or more prominent in, the more distal limb muscles. MUAPs of markedly increased amplitude (e.g., exceeding 8 mV) occur rather infrequently with compressive root lesions. They are seen more often with cervical than with lumbosacral radiculopathies, particularly those associated with spondylosis.\textsuperscript{68,112} Patients in whom MUAP amplitude exceeds 8 mV in a myotomal distribution often have a past history of previous poliomyelitis that may account for the electrophysiologic finding.

\textit{Abnormal Polyphasic MUAPs.} Those MUAPs that have more than four phases are designated polyphasic MUAPs. These are found in several different settings with radiculopathies; some are very controversial. Uncontested is that whenever muscle reinnervation is occurring, MUAPs can be seen that are not only highly polyphasic but are also of increased duration, often of low amplitude, and sometimes unstable on repetitive firing. Such “reinnervational” MUAPs can occur with radiculopathies, but more often they are encountered with plexus and peripheral nerve lesions, in which the denervation of a single muscle has been more substantial. Equally unchallenged is that almost all chronic neurogenic MUAPs are polyphasic as well. The relevance to the diagnosis of radiculopathies of polyphasic MUAPs of normal duration and amplitude and occurring in a myotomal distribution is controversial. Some EDX physicians believe that they can develop soon after symptom onset, with lesions of less than 7 days’ duration. These early appearing polyphasic MUAPs have been attributed both to slowing along the small terminal nerve fibers, caused by demyelination, and to ephaptic transmission at the site of root compression, resulting in two MUAPs firing nearly simultaneously, thereby generating a “pseudopolyphasic MUAP.”\textsuperscript{16,19} Others have shown that polyphasic MUAPs in a myotomal distribution may be the sole NEE findings with chronic radiculopathies, particularly chronic cervical radiculopathies of greater than 6 months’ duration, in which fibrillation potentials seldom are seen.\textsuperscript{16,20,48,67,127}

There are several serious problems with these approaches. First, the pathophysiologic mechanisms that would cause increased polyphasias in these situations without also altering the external configuration of MUAPs are unknown. Second, unlike fibrillation potentials and chronic neurogenic MUAPs, polyphasic MUAPs are not abnormal as isolated findings; furthermore, between 10 and 20% of MUAPs may be polyphasic in any normal muscle. Accordingly, to demonstrate excessive numbers of polyphasic MUAPs in a segmental distribution, quantitative MUAP analysis is required in several muscles.\textsuperscript{136} Finally, the high incidence of false-positive NEE studies that invariably results when excess MUAP polyphasias alone is used as a diagnostic criterion of neurogenic lesions, particularly when the examiner is inexperienced, is unacceptable to many EDX physicians.\textsuperscript{1,2,26,60,68,84,140}
Duration of Radiculopathy. The specific NEE abnormalities seen with a radiculopathy are partly a function of the duration of the lesion. Myotomal fibrillation potentials unaccompanied by abnormalities of MUAP configuration suggest the radiculopathy is of recent onset. Conversely, chronic neurogenic MUAP changes alone in a root distribution are probably due to a very slowly progressive or remote static lesion. Finally, the combination of both myotomal fibrillation potentials and chronic neurogenic MUAP changes are most consistent with either a chronic but ongoing active radiculopathy or a more acute root lesion superimposed upon a longstanding one. The amplitude, firing characteristics, and distribution of fibrillation potentials are also important. Kraft showed that the mean amplitude of fibrillation potentials decreases progressively with time, perhaps because of progressive atrophy of the denervated muscle fibers. The authors have observed that whenever fibrillation potentials fire in an irregular manner, the lesion is usually less than 6–8 weeks in duration. Also, whenever fibrillation potentials are present in the more proximal muscles of the myotome, including the paraspinal muscles, the radiculopathy is usually of recent onset or, at least, ongoing. With chronic L5 and S1 root lesions, for example, fibrillation potentials typically are not found in the hamstrings or glutei. Finally, complex repetitive discharges may be seen, usually in a minority of the myotomal muscles, with chronic radiculopathies, i.e., those of greater than 6 months’ duration.

ELECTROMYOGRAPHIC ABNORMALITIES IN RADICULOPATHY: REGIONAL CONSIDERATIONS

Cervical Radiculopathies. The cervical region is the second most common spinal area afflicted with root disorders. They constitute 5–36% of all radiculopathies encountered, in the authors’ experience, they account for approximately 5–10%. Most cervical disk and osseoligamentous disorders affect the lower four disk regions, i.e., C4–C7, and thus typically compress the lower four cervical roots. Although reports vary regarding the incidence of involvement of each root, most agree that C7 radiculopathies account for up to 70% of all cervical radiculopathies. The incidence of other cervical radiculopathies was as follows: C6: 19–25%; C8: 4–10%; and C5: 2%. In a recent publication concerned with 50 cervical radiculopathies, C7 root lesions were the most common, although their incidence (56%) was somewhat less, whereas that of C5 lesions was greater (14%).

C5 Radiculopathies. Lesions of this root (typically due to involvement of the C4 disk) may produce abnormalities in the rhomboids, spinati, deltoid, biceps, brachialis, and brachioradialis. Most of the muscles that show NEE abnormalities with C5 radiculopathies do so with C6 radiculopathies as well; because of myotomal overlap, distinguishing one from the other may be impossible. However, if changes are found in the rhomboids, the C5 root probably is the one affected. Conversely, if the pronator teres, the flexor carpi radialis, or both, are involved, the compromised root probably is C6. In many patients, however, the NEE localization to C5 as opposed to C6 remains uncertain. In these instances, many EDX physicians refer to the changes seen as those due to a “C5–C6 radiculopathy.” An H-wave study, recording from flexor carpi radialis, would point toward a C5 root lesion if abnormal.

C6 Radiculopathies. A recent report has indicated that lesions of this root (C5 disk region) have two distinct NEE presentations, one mimicking C5 radiculopathies, as already noted, and the other simulating C7 radiculopathies. With the latter, which appears to represent nearly 50% of C6 radiculopathies, abnormalities are seen in the triceps, anconeus, pronator teres, and flexor carpi radialis muscles.

C7 Radiculopathies. With C7 radiculopathies, changes typically are found in the triceps, anconeus, flexor carpi radialis, and somewhat less often, the pronator teres. One common error made with suspected C7 root lesions is to base the diagnosis on changes found solely in limb muscles that receive their intermediate innervation via the radial nerve. In addition to the NEE abnormalities, the H reflex recorded from the flexor carpi radialis muscle may be abnormal with C7 root lesions, but this is unhelpful in separating C6 from C7 lesions.

C8 Radiculopathies. Lesions of the C8 root (C7 disk region) can result in abnormalities in all of the muscles supplied by the ulnar nerve, as well as in some supplied by the radial nerve (e.g., extensor indicis proprius and extensor pollicis brevis) and some by the median nerve (e.g., flexor pollicis longus and sometimes the abductor pollicis brevis). Abductor pollicis brevis is unaffected with many C8 radiculopathies, however, presumably because it receives its predominant innervation from the T1 root.

Cervical Radiculopathies—Caveats. Both the clinical and NEE findings with cervical radiculopathies can closely mimic those found with partial axon-loss brachial plexopathies; specifically, lesions of the C5 and C6 root resemble those of the upper trunk and
lesions of the C8 root resemble those of the lower trunk. Two procedures, however, are of benefit: (1) examination of the cervical and high-thoracic paraspinal muscles, where the presence of fibrillation potentials suggests radiculopathy; and (2) evaluation of the appropriate SNAPs, because low-amplitude or unelicitable responses suggest plexopathy. Unfortunately, both of these approaches have limitations. Paraspinal fibrillation potentials are not detected with many cervical radiculopathies, despite a thorough search, so their absence never excludes a root lesion. Similarly, with mild axon-loss brachial plexopathies, not enough sensory axons degenerate to affect SNAP amplitudes. Hence, if neither paraspinal fibrillation potentials nor SNAP amplitude changes are present, the EDX localization remains indefinite.133,138 C5 and C6 radiculopathies also are mistaken for neuralgic amyotrophy, since the latter often involves one or more peripheral nerves derived principally from these roots, e.g., the suprascapular and axillary nerves. Because of the particular nerve fibers affected, the EDX abnormalities often are limited to the NEE, which is helpful in determining both the severity and distribution of the disorder.1,133

C6 and particularly C7 radiculopathies commonly are confused clinically with carpal tunnel syndrome, but are readily distinguished by EDX examination. In contrast, C8 radiculopathies frequently are mistaken for ulnar mononeuropathies, and these sometimes can be difficult to distinguish from one another, because the NEE abnormalities in any or all of the ulnar-innervated muscles are common to both. The presence or absence of abnormalities of the ulnar SNAP amplitudes, of ulnar motor NCS along the elbow segment, and in C8 root/median and radial nerve-innervated muscles is critical for accurate localization.

Myelopathies involving the anterior horn cells or their existing fibers can be mistaken for root lesions. ALS often involves the anterior cervical segments of the spinal cord. The myotomal changes that result may be identical to those seen with radiculopathies. The distinction is particularly difficult whenever ALS begins in a monosegmental, unilateral manner. A lesion involving the anterior horn cells, rather than the roots, should be suspected whenever: (1) there is no history of interscapular pain or neck stiffness, and limb pain and paresthesias are absent; (2) the denervation is exceptionally severe, as judged by the density of fibrillation potentials and amplitudes of the CMAPs recorded from affected muscles; and (3) multiple (often bilateral) involvement is found on NEE. If two or more of these features are present simultaneously (e.g., painless, severe axon loss in the distribution of more than one cervical segment), ALS is the more likely diagnosis. Occasionally, however, cervical canal stenosis in an elderly patient presents in this manner. In any event, a more extensive NEE is required.

The majority of cervical radiculopathies are solitary lesions. Nonetheless, whenever one is detected, it is prudent to examine at least a single muscle in the contralateral limb, preferably homologous to a muscle that showed abnormalities on the symptomatic side. When multisegmental abnormalities are found in an upper extremity, NEE of the contralateral limb is mandatory. Very often, similar changes are seen in that upper extremity also, frequently in multiple root distributions as well. These findings are more consistent with cervical canal stenosis or a spinal cord lesion than with a typical compressive radiculopathy. Also, whenever multilevel lesions are found bilaterally, at least a limited NEE of a lower extremity is indicated; the presence of similar changes in one or both lower extremities raises the possibility of ALS.

**Thoracic Radiculopathies.** Lesions of the thoracic roots are rare, accounting for less than 2% of all radiculopathies.62,85 EDX assessment is difficult because thoracic myotomes contain few muscles, and those muscles often are difficult to examine. To obtain adequate relaxation usually is a tedious, sometimes impossible, task, and the action potentials generated by the respiratory muscles may be confused with fibrillation potentials. The intercostal and abdominal muscles, particularly in obese patients, are studied with trepidation, if at all, because of concerns regarding the inadvertent entering of body cavities. Even when fibrillation potentials are found in the abdominal muscles, localization cannot be made to a specific root.134

Patients with clinical and EDX changes suggestive of thoracic radiculopathies are often diabetic and appear to have two or even more of their mid- or lower-thoracic roots affected, sometimes bilaterally; thus, they appear to have diabetic thoracic radiculopathy, a subdivision of diabetic polyradiculopathy. Unfortunately, in such patients there is no electrophysiologic means of distinguishing between a compressive, metabolic, or ischemic root lesion.134,135

**Lumbosacral Radiculopathies.** The lumbosacral region is the most common spinal area afflicted with root disease. Depending upon the study, lumbosacral radiculopathies comprise 62-90% of all radicu-
Lesions involving the same lumbar disk may compress different roots, depending upon the direction and size of herniation, so that the anatomic level of compromise is not reliably indicated by the affected root. The majority of disk herniations between the L4 and L5 vertebrae, for example, are posterolateral and compromise the L5 root; however, far lateral herniation may injure the L4 root, whereas central herniations may compress the S1–S4 roots. Because the L5 and S1 roots have extensive myotomal representation, extending from the paraspinal muscles to the intrinsic foot muscles, the fact that fibrillation potentials disappear from the myotome centrifugally with reinnervation is often especially obvious on NEE with L5 and S1 root lesions. In fact, fibrillation potentials may still be found in the more distal limb muscles innervated by these roots some 12–18 months after onset of a nonprogressive lesion, whereas with cervical radiculopathies they usually have disappeared by 6 months.

L2, L3, and L4 Radiculopathies. Lesions of L2, L3, and L4 are best considered collectively because they have such extensive myotomal overlap. Consequently, it is frequently impossible to distinguish isolated lesions involving one of them. Abnormalities typically are found in the various quadriceps muscles, the thigh adductors, and the iliacus. In addition, with L4 radiculopathies similar changes may be found in the tibialis anterior muscle, but their absence never excludes a lesion of that root.

EDX assessment is often difficult for several reasons. First, the L2, L3, and L4 myotomes have limited limb representation. Second, almost all the muscles comprising the L2–L4 myotomes are situated proximally in the lower extremity and, hence, tend to reinnervate sooner than muscles located more distally. Third, the majority of the muscles in these myotomes receive their intermediate innervation from the femoral nerve, making it difficult at times to distinguish a root from a peripheral nerve disorder. Fourth, no reliable sensory NCS are available for evaluating the L2–L4 fibers. Consequently, unlike the situation with L5 and S1 radiculopathies, SNAP amplitudes cannot be used to distinguish a plexopathy from a radiculopathy. The only means of making this distinction is thus by detecting paraspinous fibrillation potentials, which are not seen with many radiculopathies. As a result of these limitations, the NEE with L2–L4 radiculopathies is more likely to be falsely negative than with L5 and S1 radiculopathies; moreover, when NEE abnormalities are found, it is sometimes impossible to determine from their distribution whether they are due to a radiculopathy, a lumbar plexopathy, or even a femoral neuropathy.

L5 Radiculopathies. Most studies show that L5 radiculopathy is the most common single radiculopathy encountered. Abnormalities may be found in the tibialis anterior, extensor hallucis, peroneus longus, and extensor digitorum brevis (all of which are innervated by the common peroneal nerve), as well as the flexor digitorum longus, tibialis posterior, tensor fascia lata, and gluteus medius and maximus (particularly the former).

EDX physicians disagree regarding which hamstring muscles most often show NEE abnormalities with L5 radiculopathies; in the authors’ experience, the internal hamstrings (semitendinosus; semimembranosus) are likely to do so, but other EDX physicians have made the same claim for the biceps femoris, particularly the short head. Regarding the NEE with L5 radiculopathies, two points are noteworthy. First, of the L5 innervated muscles located proximal to the knee, the tensor fascia lata, in the authors’ experience, is more likely to show abnormalities than either the internal hamstrings or the glutei muscles. Moreover, it is one of the few muscles in the lower extremity innervated by the L5 root that does not receive its intermediate innervation from the sciatic nerve (the other muscles being the glutei). Second, the L5 myotomal muscles situated distal to the knee are the ones most likely to show abnormalities with L5 radiculopathies, particularly with chronic disorders. Nearly all of these muscles receive their intermediate innervation from the common peroneal nerve except the tibialis posterior and flexor digitorum longus; hence, one or both of the latter two muscles should always be assessed whenever an L5 radiculopathy is suspected, although some have questioned whether the tibialis posterior can be reliably assessed using the NEE.

S1 Radiculopathies. Lesions of the S1 root are the second most common radiculopathy encountered. Abnormalities may be seen in the abductor hallucis, abductor digiti quinti pedis, soleus, gastrocnemii (particularly the medial), and the glutei (es-
especially the maximus); they are sometimes found in
the extensor digitorum brevis and flexor digitorum
longus as well. As with L5 radiculopathies, there is
disagreement concerning the hamstring muscles
most likely to show abnormalities with S1 radiculopa-
thies. In the authors’ experience, the most likely is
the biceps femoris, both the long and short heads. As
with L5 radiculopathies, the NEE changes may be
restricted to muscles distal to the knee with some
acute and many chronic S1 root lesions. In addition
to the NEE, the H-wave study often is abnormal with
S1 radiculopathies.

Of the various root lesions studied in the EDX
laboratory, those involving the S1 root are most of-
ten bilateral, because the sacral fibers are more me-
dially situated in the cauda equina and, therefore,
are especially vulnerable to midline compression.125
For this reason, NEE of the contralateral limb is
mandatory whenever an S1 radiculopathy is found,
with emphasis on the S1 myotome. Moreover, be-
cause some of the S1 myotomal muscles are in the S2
myotome as well, e.g., the abductor hallucis, abduc-
tor digiti quinti pedis, soleus, and medial gastrocne-
mius, these limb muscles are the ones most likely to
be abnormal in patients with sphincter disturbances.
Consequently, they should be assessed, along with
the external anal sphincter muscle, in all patients
who have bowel and bladder disturbances on a sus-
pected lower motor neuron basis.

S2, S3, and S4 Radiculopathies. Lesions of the S2,
S3, and S4 roots often occur together and bilaterally,
usually from cauda equina injury. When due to mid-
line compression, they usually coexist with lesions,
usually bilateral, of other lumbosacral roots. Isolated
lesions of the S2, S3, and S4 roots, in the authors’
experience, are seldom due to compression. Instead,
they are most often iatrogenic, the result of inadver-
tent injury during caudal or epidural anesthesia.154
Only limited abnormalities are seen with these le-
sions during the basic EDX examination. Fibrillation
potentials, often sparse, may be found in the rela-
tively few limb muscles that are innervated by the S2
root; frequently they are limited to the abductor hal-
lucis and abductor digiti quinti pedis. The H re-
sponse frequently is normal; this can be confusing
because it is abnormal so commonly with S1 radicu-
lopathies. NEE of the anal sphincter muscle, both its
right and left portions, typically reveals severe MUAP
dropout; depending upon the duration of the lesion,
this is accompanied by fibrillation potentials,
chronic neurogenic MUAP changes, or both.

Multiple Lumbosacral Radiculopathies. Simulta-
neous involvement of more than one root is far more
likely to occur with lumbosacral lesions than with
cervical or thoracic ones. The majority of such les-
ions are seen in elderly patients. Although they can
be caused by single lateral disk protrusions,62 they
are due more often to compromise of the cauda
equina by either central disk herniations or lumbar
canal stenosis.140 Polyradicular lumbosacral lesions
typically are bilateral, although often asymmetric,
and usually involve the lower lumbosacral roots, es-
pecially S1. These are the only compressive root le-
sions that frequently are associated with abnormali-
ties on routine NCS. Low-amplitude or unelicitable
CMAPs often are found on the basic peroneal and
posterior tibial NCS, especially the latter, because of
severe axon loss affecting both roots innervating
the muscle from which the recording is made. With
these lesions the H waves characteristically are un-
elicitable on either side because of bilateral S1 root
compromise, whereas the lower extremity SNAPs
(sural, superficial peroneal) are unaffected. How-
ever, the majority of bilateral, multilevel lumbosacral
radiculopathies occur in patients who are over the
age of 60 years, and the lower-extremity SNAPs may
then be unelicitable bilaterally because of age alone.

To determine the extent of denervation present, it
often is very helpful to record from muscles prox-
imal to the ankle. With relatively acute lesions, the
CMAP amplitude of the direct motor response (M
wave) recorded during the H-wave test is an excel-
 lent indicator of the degree of S1 denervation,
whereas the CMAP amplitude recorded from the
tibialis anterior muscle, while stimulating the com-
mon peroneal nerve, often reflects the extent of L5
root denervation. With chronic lesions, the CMAP
amplitudes may not accurately mirror the severity of
motor axon loss because extensive reinnervation by
collateral sprouting may have occurred in these
muscles.122

Lumbar Canal Stenosis. The clinical hallmark of
lumbar canal stenosis is neurogenic intermittent
claudication.123 Pain, sometimes associated with
weakness or other sensory complaints, develops in
the hips, thighs, or legs after the patient walks a
certain distance and is relieved by sitting or by flex-

ing the lumbar spine. EDX changes are variable. Bi-
lateral, multiple lumbosacral radiculopathies (cauda
equina lesions) are found, in the authors’ experi-
ence, in approximately half of patients with lumbar
canal stenosis. Frequently, these are of quite pro-
longed duration; chronic neurogenic MUAP
changes tend to be prominent, whereas fibrillation
potentials often are restricted to the more distal
muscles of the myotomes. In the remaining patients
with lumbar canal stenosis, a variety of findings are
obtained. In some, two distinct lumbosacral radicu-
lopathies, most often manifested as a single root lesion in each lower extremity, occur either symmetrically (e.g., bilateral S1) or asymmetrically (e.g., left L5 and right S1). In others, an isolated radiculopathy, almost always either an L5 or an S1 lesion, is found. In some patients, limited but nondiagnostic abnormalities are encountered. One common scenario is bilaterally absent H waves associated with normal sural SNAPs and a normal NEE of the lower extremities; another is fibrillation potentials in a single limb muscle, most often one innervated by the S1 root. Finally, in the remaining patients, extensive EDX examinations are normal.

Because lumbar canal stenosis affects the cauda equina so frequently, H-wave studies and NEE should be performed bilaterally whenever this entity is suspected.

Lumbosacral Radiculopathies—Caveats. The utility of the EDX examination in individual patients depends on the particular portion of the PNS affected, the underlying pathophysiology, and the age of the patient. ALS may be mistaken for one or more lumbosacral radiculopathies when: (1) only one or a few lumbosacral segments are involved; (2) fasciculation potentials have not yet developed; (3) the process has not extended to the upper extremity; and (4) incidental back pain or a history of remote lumbar disk disease is present. Certain findings on the EDX examinations of patients with suspected lumbosacral radiculopathy should raise the possibility of ALS, and thereby encourage more extensive assessments: (1) when the patient has few or no sensory complaints, but motor axon loss in one or more myotomes is severe (e.g., fibrillation potentials and MUAP dropout are prominent and the appropriate CMAP amplitudes are low); (2) when substantial axon loss is present in an S1 myotome, and yet the H response is intact; and (3) whenever fasciculation potentials as well as fibrillation potentials are present, especially when the former are widespread.

As already noted, it is sometimes impossible to distinguish an L2, L3, or L4 radiculopathy from a lumbar plexopathy or even, occasionally, a femoral neuropathy. L5 and S1 radiculopathies can sometimes be confused with nontraumatic sacral plexopathies and sciatic mononeuropathies. The most helpful EDX procedures for distinguishing these lesions from radiculopathies in such circumstances are sensory NCS of the superficial peroneal and sural nerves. Depending upon the particular fibers affected, the SNAPs from one or both of these nerves will be of low amplitude or unelicitable with axon-loss lesions involving the sacral plexus and sciatic nerve because the sensory fibers are injured distal to their DRG. Unfortunately, many patients with these lesions, particularly those with sacral plexopathies, are over the age of 60 and may thus have bilaterally unelicitable lower extremity SNAPs because of age alone.

L5 radiculopathies frequently are confused with common peroneal neuropathies at the fibular head (CPN-FH) when they cause clinical footdrop by producing severe MUAP dropout in the tibialis anterior muscle from axons loss or, less often, demyelinating conduction block. They usually are distinguished by the EDX findings. First, the peroneal motor NCS, recording from the tibialis anterior muscle, distinguish those CPN-FH caused by conduction block from other lesions. Second, the superficial peroneal SNAPs are normal (at least in patients under the age of 60 years) with L5 compressive radiculopathies and with those CPN-FH manifested solely as demyelinating conduction block; in contrast, they are usually unelicitable, or at least of low amplitude, with CPN-FH causing axon loss. Third, with L5 radiculopathies, fibrillation potentials typically are found in myotomal muscles that do not receive their intermediate innervation via the common peroneal nerve. S1 radiculopathies sometimes are confused with distal tibial mononeuropathies (tarsal tunnel syndrome). The most helpful differentiating points are the presence with S1 radiculopathies of changes on the H-wave test and abnormalities on NEE in S1-innervated muscles proximal to the ankle.

The EDX changes seen with mild/moderate axon-loss peripheral polyneuropathies sometimes are similar to those of chronic bilateral S1 radiculopathies. In patients over the age of 60, the following combination of findings, encountered frequently, is equally consistent with both disorders: (1) bilaterally unelicitable H waves (due to the bilateral S1 radiculopathy, a polyneuropathy, or advanced age); (2) bilaterally unelicitable lower extremity SNAPs (due to a polyneuropathy or advanced age); (3) fibrillation potentials essentially limited to the intrinsic foot muscles; (4) borderline slow lower extremity motor conduction velocities; and (5) normal EDX examination of an upper extremity; with more severe axon-loss polyneuropathies, the upper-extremity SNAPs typically are low in amplitude or unelicitable, whereas with more active S1 radiculopathies definite NEE abnormalities usually can be seen in other S1-innervated muscles.

Needle Electrode Examination—Limitations. Although the NEE is the single best EDX procedure for detecting radiculopathies, it has many limitations. These, in turn, are the major limiting factors
for the EDX examination as a whole in the recognition of root lesions. First, the findings on NEE are dependent on loss of motor axons. Sensory root compromise, however substantial, has no effect on the NEE findings. Moreover, demyelinating conduction slowing and modest or lesser amounts of demyelinating conduction block along motor axons are not detected with the NEE. Substantial demyelinating conduction block alters the MUAP firing pattern, but such pathophysiology is rarely widespread enough at the root level to allow for identification of a radiculopathy.

Second, even when axon loss has affected some of the motor root fibers, it may not involve enough fibers, or the appropriate fibers, for a myotomal distribution of abnormalities to be discerned.

Third, because fibrillation potentials are the cardinal sign of denervation, the NEE is very time dependent. Studies may be falsely negative if they are performed either too early or too late in the course of a radiculopathy, after reinnervation has occurred. In this regard, it is important to appreciate that, following a single episode of root compression, fibrillation potentials not only develop in the muscles of the myotome in a proximodistal sequence, but also soon begin to disappear from the myotome in the same sequence because of collateral sprouting in the partially denervated muscles. With static radiculopathies, this reparative process occurs regardless of whether the root is decompressed. Hence, the NEE frequently “normalizes” with time, even when clinical symptoms and signs (especially sensory) persist. This is probably the reason that false-negative studies are found so frequently with cervical radiculopathies of greater than 6 months’ duration and lumbosacral radiculopathies of more than 12-18 months’ duration. As a corollary, fibrillation potentials will persist indefinitely only if additional motor axons are continually being compromised, i.e., if the lesion is progressive rather than static.

Fourth, although fibrillation potentials in a myotome distribution are highly suggestive of a proximal axon-loss lesion, they reveal neither the etiology of the lesion nor whether it is involving the root, rather than the appropriate spinal cord segment. Because of this, the indefinite designation “intraspinal canal lesion” is often more appropriate than “radiculopathy” when abnormalities are found in one or, particularly, more myotomal distributions. Thus, bilateral changes in an L5 distribution can be described as “a lumbar intraspinal canal lesion involving the L5 segment/root bilaterally.”

Fifth, even if fibrillation potentials are present in several muscles of the myotome, the affected root may not be accurately localized by the NEE because of root innervation anomalies, myotomal overlap, or both. This is particularly a problem when only a few muscles of the myotome show definite NEE changes. Finally, definite myotomal changes permit the affected segmental or radicular level to be determined, but not the anatomic site of the lesion in regard to disk levels. This, limitation, as already noted, is more significant with lumbosacral radiculopathies than with cervical or thoracic ones.

**COMPARATIVE DIAGNOSTIC SENSITIVITY**

The EDX examination and neuroimaging studies focus on different aspects of the nerve roots. The EDX examination detects functional abnormalities, whereas neuroimaging studies detect structural abnormalities. Studies of relative diagnostic sensitivity are difficult, because there is no widely accepted “gold standard” against which tests can be judged. The operative findings are often used for this purpose, even though, as Tullberg and associates note, this is not ideal “because different surgeons tend to have different subjective evaluations of the degree of nerve root entrapment.” Moreover, typically just one or two roots are explored.

Early studies compared the EDX examination to myelography. The EDX examination was positive in 73–94% of cases, whereas myelography was positive in 75–84%; in four of five studies, the EDX examination had a higher yield. More recently, the EDX examination has been compared to CT, with essentially the same results. The authors are unaware of similar comparative studies with magnetic resonance imaging (MRI). Regardless, many physicians routinely refer patients for imaging studies and only occasionally consider the value of an electrophysiologic appraisal for nerve root function. This is unfortunate because, as for myelography, both CT scans and MRI are so sensitive for detecting structural abnormalities that their diagnostic specificity is impaired. For the lumbar spine, myelography may reveal abnormalities in 24% of asymptomatic subjects, CT scans in 36%, and MRI in between 28 and 64%. Similarly, cervical MRI is abnormal in 19% of asymptomatic subjects. Indeed, because of the high incidence of asymptomatic abnormalities in the spinal MRI, EDX examination is often necessary to determine whether structural abnormalities are of functional significance. Such studies are particularly important whenever the clinical symptoms and the neuroimaging studies are discordant.
VALUE AND LIMITATIONS OF THE EDX EXAMINATION

There are several limiting factors related to the different techniques in use, as already discussed, and other more general concerns. First, the EDX examination does not detect all compressive radiculopathies. The EDX findings can therefore never be used to exclude a radiculopathy. This critical fact concerning the EDX examination should be included in the formal EDX report, whenever appropriate, to avoid misleading the referring physician. A second limiting factor of the EDX examination is that even when the findings suggest a radiculopathy, the etiology of the responsible process cannot be determined. Third, even when a specific root appears to be involved, various anatomic factors may lead to inaccurate localization, so that the root actually affected is not recognized (it is usually adjacent to the one identified); moreover, the site of the lesion within the intraspinal canal cannot be accurately determined, at least with lumbosacral radiculopathies. Finally, the presence of a number of "confounding factors," many interrelated and the majority occurring principally in the elderly, can produce changes on the NCS, H-wave studies, and NEE that may compromise the ability of the EDX physician to recognize a radiculopathy. These include advanced patient age, diabetes mellitus, a generalized polyneuropathy, and remote poliomyelitis.

The EDX examination provides several distinct advantages in the assessment of patients with suspected root disorders. First, it is the only laboratory study in widespread use that directly assesses the physiologic integrity of the roots, thereby providing information of both diagnostic and prognostic relevance. Second, it may reveal changes consistent with a root lesion in the presence of a clinical examination that is normal or unsatisfactory because of a host of complicating factors, including poor voluntary effort (hysteria; malingering; pain on activation) and an upper motor neuron lesion. Third, the EDX evaluation may be abnormal when all other laboratory procedures, including neuroimaging studies, are unrevealing. Such imaging studies typically are of no benefit in patients with noncompressive radiculopathies; moreover, with compressive root lesions imaging studies cannot determine the severity of axon loss and thereby aid in determining the prognosis. Fourth, the EDX examination generally can identify extraforaminal lesions, e.g., plexopathies, mononeuropathies, and polyneuropathies, the symptoms of which are often attributed incorrectly to radiculopathies. Fifth, when performed by experienced physicians, EDX studies are rarely false-positive. In contrast, incidental neuroimaging abnormalities are very common, particularly in middle-aged or elderly patients without related symptoms. Consequently, performing EDX studies on these patients may be helpful for determining whether the reported anatomic abnormalities are of clinical significance. Finally, the EDX examination has a low morbidity, can be performed on outpatients, and can be used to follow radiculopathies over time.

REFERENCES


