Electrodiagnostic Assessment of Radiculopathies

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KEYWORDS

• Radiculopathy • Electromyography • Electrodiagnosis • Neuromuscular

KEY POINTS

- Nerve conduction studies are limited in their utility to diagnose radiculopathy. They should be used primarily to exclude alternative diagnoses.
- A needle examination root screen approach should include a minimum of 6 muscles covering all common root levels.
- On needle electrode examination, spontaneous activity and/or motor unit action potential morphologic changes in 2 different muscles of the same myotome, but with different peripheral nerve innervation, supports an electrodiagnosis of motor radiculopathy.

INTRODUCTION

One of the most common referrals to the electrodiagnostic laboratory is for evaluation of clinically suspected radiculopathy, a pathologic process involving the nerve root. In 1950, Shea and colleagues[1] first described how electrodiagnosis (EDX) could identify fibrillation potentials in a specific myotome, thereby supporting a diagnosis of compressive radiculopathy. Despite being a common referral indication, EDX confirmation of a radiculopathy is challenging due to several limitations of testing. A study of 1000 patients referred for electromyography (EMG) evaluation of radiculopathy found 49.8% with a normal study and only 7% confirming radiculopathy.[2] Sensory symptoms and pain are the most common complaints,[3] but because small unmyelinated pain fibers or preganglionic sensory fibers cannot be assessed by routine nerve conduction studies (NCSs), patients often have a normal study even if the pathology truly involves the root. Despite the limitations of EDX testing in the diagnosis of radiculopathy, EMG plays an important role. When EMG is combined with a clinical history, examination, and other testing, it can support the diagnosis of radiculopathy and exclude mimicking disorders.[4]

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REVIEW OF ANATOMY

There are 31 nerve root pairs: 8 cervical, 12 thoracic, 5 lumbar, 5 sacral, and 1 coccygeal. Each root is formed from a ventral motor axon whose cell body originates in the anterior horn cell of the ventral spinal cord and a dorsal sensory axon whose cell body originates from the dorsal root ganglion (DRG). The DRG typically is located along the dorsal root at the entrance of the intervertebral foramen, and thus is not truly intraspinal. Approximately 3% to 9% of L3 and L4 DRGs, 11% to 38% of L5 DRGs, and 71% to 77% of S1 DRGs are intraspinal.[5–7] Similarly, in the cervical region, there has been suggestion that the C5 and C6 DRGs can be intraspinal.[8]

The ventral motor and postganglionic dorsal sensory axons come together within the central canal to form the nerve root, which then exits the canal via the intervertebral foramen (**Fig. 1**). Upon exiting laterally from the intervertebral foramen, the nerve root divides into small posterior primary ramus, supplying the neck and paraspinal muscles, and a larger anterior primary ramus supplies the limbs and anterior trunk, including the abdominal and intercostal muscles.

From C1 to C7, the nerve root exits above the corresponding vertebral body (eg, the C7 root exits from the C6-7 intervertebral foramen). The C8 nerve root is a transition point and exits between the C7 and T1 vertebral bodies and all subsequent roots exit below their corresponding vertebral body. The cervical, thoracic, and high lumbar segments the root exit laterally essentially along a horizontal plane. Because the spinal cord ends in the adult at approximately the L2 vertebral body, however, the nerve roots representing segments below this level must travel caudally within the central canal to reach their exiting intervertebral foramen. This collective group of nerve roots forms the cauda equina. Because they travel together, pathology at the L3 level

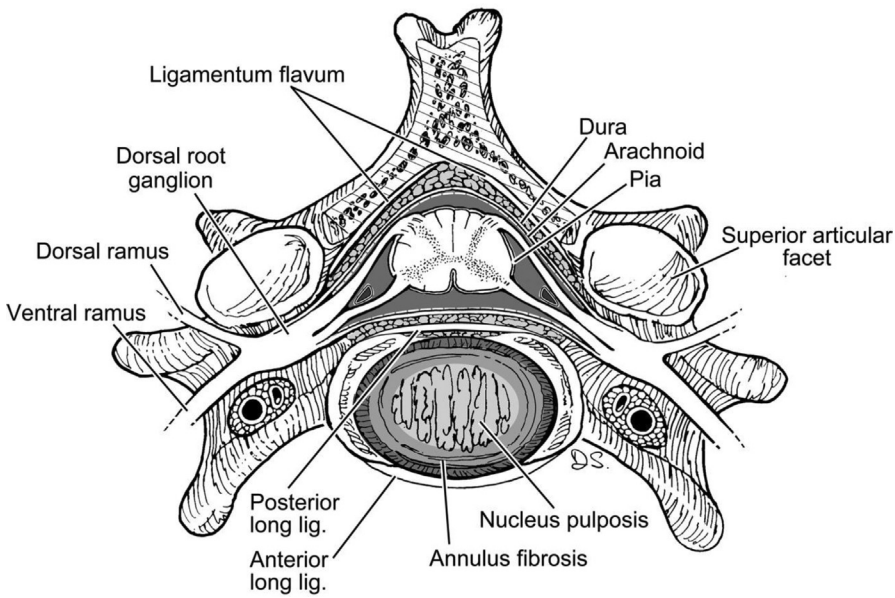

Fig. 1. Anatomy of the spine. The ventral and dorsal roots are intraspinal, meaning located within the central canal. The dorsal root ganglion is located at the entrance of the intervertebral foramen, and thus is not truly intraspinal. long, longitudinal; lig, ligament. (*Reprinted with permission*, Cleveland Clinic Center for Medical Art & Photography ©2021. All Rights Reserved).

potentially could have an impact on not only the L3 nerve root but also the descending nerve roots traveling as part of the cauda equina.

Almost all muscles receive innervation from more than 1 nerve root, and the degree to which each nerve root segment contributes innervation is unpredictable and can vary among individuals.[9]

NERVE CONDUCTION STUDIES

An EDX study evaluating for potential radiculopathy starts with NCSs. Nerves selected for testing are guided by clinical history, examination, and the requisition from the ordering provider. Commonly used NCSs are listed in **Table 1** for the upper limb and **Table 2** for the lower limb. Normative values based on age, gender, and height have been published.[10,11]

Several factors limit the utility of NCSs in the diagnosis of radiculopathy. Disc protrusion and spondylosis are among the most common causes of compressive radiculopathy but often result in damage to only a small number of traversing nerve fibers, producing limited motor and sensory symptoms. Paresthesias and pain, which often are the predominant complaints in radiculopathy, are transmitted via unmyelinated C-type sensory fibers that are not evaluated using routine NCS techniques. Furthermore, focal compression of a nerve root could cause focal conduction velocity slowing and/or conduction block along the compressed segment. These are not identifiable, however, because intraspinal location of most radicular lesions render direct NCSs on the nerve root proximal to the compressed area impossible.

Sensory Nerve Conduction Studies

Sensory NCSs are of limited value in the diagnosis of radiculopathy. The sensory nerve action potential (SNAP) responses are not affected in radiculopathy because most radicular lesions are located within the central canal and proximal neural foramen.

Table 1
Common nerve conduction studies performed in the upper limb

Nerve	Recording Site	Root Distribution
Sensory conduction responses		
Median	Digit 1	C6
	Digit 2	C6-7
	Digit 3	C7
Ulnar	Digit 4–5	C8 (T1)
Radial	Dorsal hand	C6 (7)
LAC	Forearm	C6
MAC	Forearm	T1
Motor conduction responses		
Median	APB	(C8) T1
Ulnar	ADM	C8 (T1)
Radial	EDC	C8
Musculocutaneous	Biceps	C5-6
Axillary	Deltoid	C5-6

Abbreviations: EDC, extensor digitorum communis; LAC, lateral antebrachial cutaneous; MAC, medial antebrachial cutaneous.

Table 2
Common nerve conduction studies performed in the lower limb

Nerve	Recording Site	Root Distribution
Sensory conduction responses		
Sural	Lateral ankle	S1
Superficial peroneal	Dorsum of foot	L5
Saphenous	Medial foreleg	L3-4
Motor conduction responses		
Tibial	Abductor hallucis	S1
Peroneal	EDB	L5 (S1)
Peroneal	Tibialis anterior	(L4) L5
Femoral	Rectus femoris	L3-4

Because most DRGs reside distal to the area of nerve root compression, the SNAPs remain normal. If the lesion is noncompressive (ie, infiltrative) or extraspinal (distal to the neural foramen), the DRG can be damaged leading to wallerian degeneration and SNAP amplitude reduction or loss. Examples of this include malignancy and infection.

One exception is the L5 DRG, which sometimes can reside within the central canal where it is vulnerable to intraspinal compression.[12] This can result in an absent or low amplitude superficial fibular (peroneal) sensory response. The S1 DRG also can reside within the central canal; it typically rests below the L5-S1 disc space where most compressive pathology occurs; thus, the sural SNAP remains unaffected.[13] The value of sensory NCSs primarily is to assess for other lesions, such as mononeuropathy and plexopathy, because their clinical presentation can mimic radiculopathy.[14]

Testing the median, ulnar, and superficial radial SNAPs usually is adequate to screen for common mimics, such as carpal tunnel syndrome, ulnar mononeuropathy, or peripheral neuropathy affecting the arm (see **Table 1**). If a C5-6 root lesion is in question, adding a lateral antebrachial cutaneous SNAP may be appropriate to rule out an upper trunk brachial plexopathy. Similarly, a medial antebrachial cutaneous sensory response could help rule out a lower trunk brachial plexopathy mimicking a T1 radiculopathy.

For patients presenting with a suspected lumbosacral radiculopathy, the superficial fibular and sural nerves are most useful (see **Table 2**). These responses can be obtained reliably in most individuals; although, after the ages of 50 and 75, respectively, their absence may be a normal finding.[15,16] Normal sensory responses help to rule out mimics, such as peripheral neuropathy, peroneal mononeuropathy, sciatic mononeuropathy, and sacral plexopathy. Saphenous and lateral femoral cutaneous SNAPs also can be performed but are technically challenging. Asymmetric amplitude reduction or absence may be useful to exclude a femoral mononeuropathy or lumbar plexopathy mimicking an L3 or L4 root lesion.

Motor Nerve Conduction Studies

Motor NCSs can be useful in radiculopathy assessment but have limitations and frequently are normal.[17] Typically, compressive intraspinal lesions damage a limited number of motor fibers of the traversing nerve root. The recorded compound muscle action potential (CMAP) reflects the summation of all underlying muscle fiber action potentials within the recording electrode field. At least 50% of motor axons must be

lost to produce a reliably abnormal CMAP difference compared with the contralateral side.[18] Timing between the onset of the lesion and the study also is important, because the CMAP may not decrease until sufficient time has passed for wallerian degeneration to take place (at least 5 days post-transection). Similarly, reinnervation can result in normalization of previously reduced CMAP amplitudes.

Reliable CMAPs can be obtained from C5-6, C8, and T1 myotomes (see **Table 1**). The median CMAP response recording abductor pollicis brevis (APB) reflects T1 root/segment innervation, whereas the musculocutaneous CMAP recording over biceps and axillary CMAP recording over deltoid reflect the C5-6 roots/segments. In a C8 lesion, the ulnar CMAP recording abductor digiti minimi (ADM) could be reduced, and an amplitude less than 10.2 mV was shown to have sensitivity and specificity of 0.86 and 0.74, respectively, in NEE-confirmed active radiculopathy.[19] The C7 myotome has no reliable CMAP because the muscles are not spatially isolated from muscles of other myotomes.[20]

In the leg, L5 and S1 myotomes are well represented with routine studies (see **Table 2**). The fibular CMAP (recording extensor digitorum brevis [EDB] and tibialis anterior) reflects predominantly the L5 myotome. The tibial CMAP reflects predominantly the S1 myotome. For NEE-confirmed active L5 radiculopathy, a fibular CMAP amplitude less than 3.6 mV was shown to have sensitivity and specificity of 0.92 and 0.60, respectively.[19] Femoral motor NCS recording rectus femoris can show axon loss at the L3 and L4 root levels.

Routine motor NCSs obtained in the arm as part of a screening assessment include the median (recording APB) and the ulnar (recording ADM). These studies, however, evaluate the C8 and T1 roots, which are not the most common levels involved in cervical radiculopathy.[21] In the lower limb, routine motor NCSs obtained include the fibular (EDB) and the tibial response recording abductor hallucis, which assess the commonly involved L5 and S1 root levels.

If an abnormality is noted on NCS, it is good practice to obtain the same response on the contralateral side for comparison. It also is important to note any other technical factors, such as peripheral edema and obesity, which could contribute to spuriously reduced CMAP amplitudes.[22]

LATE RESPONSES

Late responses include both the F wave and H reflex. In radiculopathy, both can be of potential value because the evoked motor potential travels to the spinal cord and back down through the nerve root, theoretically allowing for assessment of the nerve root itself.

F Wave

An F response is a pure motor arc that measures the time it takes for an evoked antidromic motor nerve action potential to travel proximally up the peripheral nerve from the point of stimulation and reactivate a small number of anterior horn cells' axon hillocks, triggering a backfire that sends the action potential orthodromically back down the motor peripheral nerve to the recording electrode. The most common measurement for assessment is the earliest latency. In root compression, the very narrow segment of slowing is diluted by the longer segment of normal conduction, thus reducing sensitivity of the response. Other parameters, such as chronodispersion and persistence, have been studied but are less reliable.[23]

Commonly obtained F responses include the ulnar response (ADM) and median (APB) in the arm and the fibular (EDB) and tibial (abductor hallucis) in the leg, which

all are highly reproducible.[24,25] Using the motor CMAP and F response together can be beneficial in interpretation, but studies have concluded F responses have relatively low sensitivity in this regard.[26] In a recent study of 142 patients with unilateral L5 (n = 67) or S1 (n = 76) radiculopathy in whom magnetic resonance imaging and NEE correlated, abnormal fibular and tibial F responses were found only 50.7% and 36.0% of the time, respectively.[23] Because F waves represent a pure motor arc, they are normal in patients with only sensory complaints.

The radial F wave technique (recording form anconeus and extensor indicis proprius) has been validated with normative values published.[27,28] In theory, an F wave can be obtained from any peripheral motor nerve but the clinical utility of other responses is not clear.

H Reflex

The tibial H reflex is a true root reflex arc that involves both the sensory and motor roots and is a highly sensitive measure of S1 root pathology (up to 80% in surgically proved cases).[29] A recent study also suggests that the lateral or medial gastrocnemius also could be recorded without reduced sensitivity.[30]

An abnormal H reflex with a normal tibial CMAP is suggestive of more proximal disease, which could be anywhere along the reflex arc, including the S1 root, sciatic nerve, or proximal tibial nerve; thus, specificity as an isolated abnormality is low. Also, limb length, temperature, and age have an impact on the response. Therefore, a side-to-side comparison of the H amplitude is felt to be of highest clinical utility, with an H-amplitude ratio (abnormal H amplitude divided by contralateral H amplitude) of less than 0.4 being abnormal.[31] The absence of an H reflex on 1 side when present on the other always is abnormal, whereas bilateral absence could be technical in nature, particularly in large individuals. An H amplitude of less than 1 mV or the absence of an H reflex in a person above age 60 is considered a possible normal finding.

NEEDLE ELECTRODE EXAMINATION

The needle electrode examination (NEE) is more valuable than NCSs in the assessment of radiculopathy. The sensitivity of the NEE has been reported to range from 49% to 86% for lumbosacral to 50% to 71% for cervical motor radiculopathies.[32] Specificity has been found to range from 87% to 100%, depending on the abnormalities identified in lumbosacral radiculopathy.[33,34] Specificity was 100% if fibrillations and positive waves (PWs) were seen in 2 limb muscles with or without the corresponding paraspinal muscle, or in 1 limb and its corresponding paraspinal muscle. If greater than 30% polyphasia was utilized as the abnormal finding, the specificity dropped to the lower end of the range. In light of the lag in generation of fibrillation potentials after acute nerve transection, delaying NEE for at least 3 weeks after the onset of motor symptoms is recommended.

One of the reasons for low sensitivity of NEE relates to the need for motor axon loss or significant motor root demyelination to occur for changes to be seen on NEE. If a lesion affects only the sensory root fibers, NEE is normal. Therefore, a normal NEE does not rule out radiculopathy as a cause for the clinical symptoms if sensory symptoms are the main complaint.

The American Association of Neuromuscular & Electrodiagnostic Medicine recommends a root screen approach to the design of the NEE study to ensure that screening of the most common root levels involved in radiculopathy is performed.[35] In the arm, this includes C5-8 and in the leg L3-S1. If an abnormality is found, additional muscles in the same myotome are studied. The more muscles in a myotome showing

consistent change, the more reliable the diagnosis. A minimum of 2 muscles in the same myotome with different peripheral nerve innervation is minimum criterion for EDX of motor radiculopathy.[9] Identifying both distal and proximal muscle involvement further supports the diagnosis and rules out peripheral causes like polyneuropathy or mononeuropathy.

Recently, a 6-muscle screen has been recommended for both cervical and lumbosacral radiculopathies.[32] This was based predominantly on prior studies looking at a root screen with or without paraspinal muscles in the cervical and lumbosacral regions. The sensitivity difference among 5-muscle, 6-muscle, and 7-muscle screening algorithms was compared along with various NEE parameters.[36,37] PWs, fibrillations, complex repetitive discharges, high-amplitude, long-duration motor unit action potentials (MUAPs), and reduced recruitment were analyzed. Radiculopathy was considered confirmed when 2 or more muscles from the same root but different peripheral nerves showed any of these findings or when the paraspinal muscles demonstrated fibrillation potentials, PWs, or complex repetitive discharges. The 6-muscle screens were 94% to 99% sensitive in detecting radiculopathy, with the higher range being when paraspinal muscles were included in the screen. Screens can serve as valid initial work-ups, but additional muscles should be examined to confirm the diagnosis.

Paraspinal muscle involvement can be a useful tool to support intraspinal disease and rule out extraspinal causes of motor symptoms, such as plexopathy, but there are limitations of paraspinal muscle NEE examination. Paraspinal abnormalities occur in other disorders, such as motor neuron disease and necrotizing myopathies. Fibrillation potentials or PWs rarely can occur in normal individuals, particularly in the lumbosacral region.[38] The segmental innervation to these muscles can overlap by up to 6 segments in some cases, and thus abnormalities at the C7 vertebral level may not correlate to C7 root pathology.[38,39] Iatrogenic injury during spinal surgery can result in permanent denervation, and fibrillation potentials may persist indefinitely rendering them unreliable when assessing acute or subacute symptoms. In routine clinical practice, paraspinal muscles are not sampled if prior surgery has been performed near the root level of interest.

ACUTE VERSUS CHRONIC RADICULOPATHY

MUAP morphology and the presence or absence of fibrillation potentials help define the age of a radiculopathy. Each of these changes takes time to develop, and the changes seen during NEE need to be correlated with the time of symptom onset. When interpreting the NEE changes, the wording used to describe them also matters and has been a source of debate.[40] In a 2014 study, various terminology was used to describe whether a radiculopathy occurred recently (days to weeks) or in the more distant past (months to years) and if there was evidence of an active or ongoing lesion. Referring providers were asked to interpret this terminology with variable results. Describing the age of a root lesion with words like “acute” to mean days to weeks and “chronic” to mean months to years yielded reliable understanding and is recommended. To indicate whether the lesion still is occurring at the time of NEE, it is recommended to use a qualifier, such as “active” or “inactive.” Without these qualifiers, non–EMG-trained physicians had confusion correctly interpreting the EMG report.

In an axonal lesion of less than 3 weeks, insufficient time may have elapsed for the development of abnormal spontaneous activity and MUAP morphology is normal. Occasionally, abnormal insertional activity in the form of very brief trains of PWs may be seen in myotome-specific muscles that could suggest very recent motor axon loss.

After 3 weeks, fibrillation potentials and PWs develop and their presence suggests an active lesion. If the MUAP morphology is normal, this is qualified as an acute, active radiculopathy.

Although axonal lesions produce the most reliable NEE changes, demyelinating lesions also can occur. A demyelinating root lesion with prominent conduction block in the absence of axon loss change (ie, fibrillation potentials) may be suspected when clinical weakness is present; reduced recruitment of normal MUAPs is seen in a muscle whose distal CMAP is normal.

In more chronic root lesions with axonal loss, surviving motor axons attempt to reinnervate denervated muscle fibers via collateral sprouting. This typically occurs between 6 weeks and 26 weeks after the initial root injury. During initial reinnervation, the morphologic appearance of MUAPs is polyphasic and they may be unstable due to immaturity of newly formed neuromuscular junctions. As new neuromuscular junctions stabilize and reinnervation becomes complete, MUAPs take on their final chronic appearance of increased duration and amplitude with a reduced recruitment pattern. When these MUAPs are seen, the lesion is qualified as chronic. Abnormal spontaneous activity seen with chronic MUAPs is indicative of a chronic, active lesion. Alternatively, the absence of abnormal spontaneous activity indicates the lesion is chronic, inactive. Sometimes, very distal muscles never fully reinnervate following root injury. This chronic muscle fiber denervation can result in persistent fibrillation potentials and does not necessarily indicate an active lesion. In these circumstances, it may not be possible to reliably determine if a lesion is active or inactive. If there is further uncertainty, stating that "it is unclear whether there is ongoing nerve root injury" is appropriate.[40]

Single-fiber EMG has been used to study the course of reinnervation of chronic radiculopathy.[41–43] One study evaluated 32 patients with EMG-confirmed chronic radiculopathy based on increased amplitude and duration MUAPs with decreased recruitment. Jitter analysis was performed on the most severely affected muscle, with the most commonly studied muscles in descending order being tibialis anterior, triceps, medial gastrocnemius, vastus lateralis, and rectus femoris. Abnormal mean jitter values were found in 75% of patients with chronic MUAPs on conventional EMG and in 100% of patients where fibrillation potentials were identified. It was concluded, however, that increased jitter was not a reliable measurement and should be avoided in chronic denervated muscles.

CERVICAL RADICULOPATHY

Most muscles have more than 1 root innervation. Although the C5-T1 motor nerve roots are assessed easily on NEE, segmental innervation of a muscle may vary between individuals and important anatomic variations specific to the brachial plexus occur in up to half of all individuals.[44] In those with a prefixed brachial plexus, C4 contributes to traditionally C5-innervated muscles, and, in a postfixed brachial plexus, T2 nerve roots contribute to T1-innervated muscles.

A typical root screen for cervical radiculopathy might include first dorsal interosseous, flexor pollicis longus, extensor indicis proprius, pronator teres, triceps, biceps, and deltoid, effectively covering C5-8. If a radicular distribution of fibrillation potentials is noted during the root screen, the paraspinal muscles also is evaluated. Depending on the root level in question, NCSs and muscles could be added to the root screen study (**Table 3**). In a recent study of 114 patients with an infraspinatus muscle weakness, 16 were found to have C4/5/6 structural (disc herniation or spondylosis) radiculopathy as the cause. They found that in these patients, deltoid was

Table 3
Needle electromyography and nerve conduction abnormalities in cervical radiculopathies

Root	Commonly Affected	Nerve Conduction Study Considerations
C5	Deltoid Biceps Infraspinatus Brachioradialis Rhomboid major	Axillary or musculocutaneous CMAP amplitudes
C6	Deltoid Biceps Infraspinatus Brachioradialis Pronator teres Triceps	A normal amplitude lateral antebrachial cutaneous SNAP reflects an intact C6 DRG, from which it typically is derived.
C7	Triceps > pronator teres Anconeus Flexor carpi radialis Extensor carpi radialis	No reliable studies
C8	First dorsal interosseous Extensor indicis proprius Flexor pollicis longus	Ulnar SNAP is characteristically normal and helps rule out ulnar mononeuropathy and lower trunk/medial cord brachial plexopathy. Medial antebrachial cutaneous SNAP can be obtained to rule out a lower trunk plexopathy and true neurogenic thoracic outlet syndrome.

affected most severely, followed by infraspinatus and then biceps.[45] This underscores the importance of including infraspinatus if a C5/6 radiculopathy is suspected, which increases diagnostic yield because biceps may be normal on NEE in more than 50% of patients.

LUMBOSACRAL RADICULOPATHY

At the lumbosacral root levels, the L5 and S1 root distributions are the most common patterns on NEE. Due to the long length of descent for root fibers in the cauda equina through the central canal for multiple spinal segments, however, the correlation with the spinal level of root damage is not discernible by NEE. For example, a lateral disc herniation at the L2-3 spinal level can produce an L2 or L3 radiculopathy, whereas a central herniation at the same level can produce an L4, L5, or S1 radiculopathy. A study of 14 patients demonstrated that upper lumbar stenosis from L1-L4 resulted most commonly in abnormalities in L5 and S1 myotomes.[46]

A lumbosacral radiculopathy root screen may include tibialis posterior or flexor digitorum longus, medial gastrocnemius, tibialis anterior, rectus femoris or vastus lateralis, and gluteus medius or tensor fascia lata. If peroneal and tibial CMAP amplitudes are reduced, EDB and abductor hallucis may be added to evaluate for distal-proximal gradient of motor axon loss when polyneuropathy is in the differential diagnosis. Additional muscles may be examined when needle abnormalities are found to establish a diagnosis more confidently (**Table 4**). Identification of abnormalities in proximal muscles helps support a diagnosis and exclude mimics such as sciatic and peroneal mononeuropathy. This can be important particularly in elderly patients in whom absent sensory responses might be a normal finding. It is important to recall that a low or absent superficial peroneal SNAP does not reliably exclude an L5

Table 4
Needle electromyography and nerve conduction abnormalities in lumbosacral radiculopathies

Root	Commonly Affected	Nerve Conduction Study Considerations
L2-3	Rectus femoris Vastus medialis Vastus lateralis Iliacus Adductor longus	Saphenous and lateral femoral cutaneous SNAPs can be performed to exclude lumbar plexopathy. Femoral CMAP (rectus femoris) amplitudes may be low.
L4	Rectus Femoris Adductor Longus (tibialis anterior)	Femoral CMAP (rectus femoris) amplitudes may be low.
L5	EDB Tibialis posterior Tibialis anterior Gluteus medius Peroneus longus Extensor hallucis longus Semitendinosus/semimembranosus	Fibular motor (EDB and tibialis anterior) recording
S1	Medial gastrocnemius Short head biceps femoris Gluteus maximus Abductor hallucis	

radiculopathy given the common intraspinal involvement of the L5 DRG.[12] Fibrillation potentials in a myotomal distribution should trigger paraspinal muscle examination.

THORACIC RADICULOPATHY

Thoracic radiculopathy is uncommon.[47] From an EDX standpoint, it is difficult to confirm with a high degree of confidence due to the overlapping multisegment innervation of the paraspinal muscles and rectus abdominus muscles. The NEE approach should include both thoracic paraspinal muscles and relevant levels of the rectus abdominis muscles. The paraspinal examination often is hampered by poor relaxation, precluding reliable assessment of spontaneous activity. Studying the upper, mid, and lower rectus abdominis muscles often is more productive, assessing for both spontaneous activity and MUAP changes.

T1 radiculopathy can be assessed by examination of APB. Flexor pollicis longus also can have significant T1 innervation, but both muscles also can have C8 contributions.[48,49] With T1 radiculopathy, APB shows significant chronic and/or active motor axon loss in the absence of features, suggesting carpal tunnel syndrome. Normal medial antebrachial cutaneous sensory response excludes true neurogenic thoracic outlet syndrome.[50,51]

SUMMARY

EMG is an important tool used to assist in the diagnosis of radiculopathy. NEE is the most sensitive and specific portion of the study in this regard. Finding spontaneous activity and/or MUAP morphologic changes in 2 different muscles of the same myotome, but with different peripheral nerve innervation, supports an EDX of motor radiculopathy. A 6-muscle NEE root screen approach is optimal. Understanding the limitations is important, including the inability to assess sensory-only symptoms. Thus, a normal EMG does not preclude the presence of radiculopathy.

Finally, clear and precise wording of the diagnostic interpretation is required to convey meaning, minimize confusing terminology, and provide proper direction for subsequent patient care.

CLINICS CARE POINTS

- When performing NEE in suspected radiculopathy and neurogenic MUAPs are seen, sample additional muscles innervated by the same root but a different peripheral nerve when possible.
- When radiculopathy is suspected but sensory conduction responses are reduced in amplitude or absent, consider alternative diagnoses.
- Avoid NEE of the paraspinal muscles if posterior spine surgery has been performed in the area of interest.
- A normal EMG study does not exclude radiculopathy as the cause of clinical symptoms, particularly in sensory predominant cases.

DISCLOSURE

The authors have nothing to disclose.

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