29 Radiculopathy

Radiculopathy is one of the most common diagnoses referred to any electromyography (EMG) laboratory. Even with the widespread use of magnetic resonance imaging, EMG continues to play an important role in the evaluation of radiculopathy. Although imaging studies usually are diagnostic in the more common radiculopathies caused by structural lesions, they often are unrevealing in radiculopathy caused by infection, infiltration, demyelination, or infarction. Whereas imaging studies do well in visualizing the spinal cord and nerve roots and their relationship to the vertebrae and intervertebral discs, they yield no information about how the nerve is functioning. In this regard, EMG complements magnetic resonance imaging with its ability not only to localize the lesion but also to functionally assess the nerve. However, every electromyographer should be aware that EMG has several significant limitations in assessing radiculopathy that can result in false-negative studies.

CLINICAL

The clinical hallmark of radiculopathy includes pain and paresthesias radiating in the distribution of a nerve root, often associated with sensory loss and paraspinal muscle spasm. Motor dysfunction may also be present. Radiculopathy caused by degenerative bone and disc disease most often affects the cervical (C3–C8) and lower lumbosacral (L3–S1) segments, resulting in well-recognized clinical syndromes (Tables 29–1 and 29–2). Associated paraspinal muscle spasm commonly limits the range of motion, and movement of the neck or back may exacerbate symptoms.

The particular sensory and motor symptoms associated with a radiculopathy depend on which nerve root or roots are involved. Each nerve root supplies cutaneous sensation to a specific area of skin, known as a *dermatome* (Figures 29–1 and 29–2), and motor innervation to certain muscles, known as a *myotome* (Tables 29–3 and 29–4). Each dermatome overlaps widely with adjacent dermatomes. *Consequently, it is very unusual for a patient with an isolated radiculopathy to develop a severe or dense sensory disturbance*. Dense numbness usually is more indicative of a peripheral nerve lesion than a radiculopathy. *In a patient with radiculopathy, sensory loss more often is vague, poorly defined, or absent, despite the presence of paresthesias*.

Just as with dermatomes, there is a wide overlap of myotomes. Indeed, nearly every muscle is innervated by at least two if not three myotomes (i.e., nerve roots). For instance, the triceps brachii muscle, predominantly a C7-innervated muscle, also receives some innervation from the C6 and C8 nerve roots. C*onsequently, paralysis of a muscle is very unusual in an isolated radiculopathy*. Even in the case of a severe or complete C7 radiculopathy, the triceps brachii will become weak but not paralyzed, retaining some strength from its partial C6 and C8 innervation.

The deep tendon reflexes may be abnormal in a radiculopathy, depending on the root innervation to the muscle tendon being tested. The biceps and brachioradialis reflexes may be depressed in a lesion of the C5 or C6 nerve roots. The triceps reflex typically is most depressed with a lesion of the C7 nerve root but, because of its significant partial C6 innervation, may be abnormal with a lesion of that root as well. There is no routine reflex to check for a lesion of C8 or T1. In the lower extremities, the knee and ankle reflexes are commonly checked. The knee jerk may be reduced with a lesion of the L3 or L4 (rarely L2) nerve roots and the ankle jerk with a lesion of the S1 nerve root. Again, there is no useful routine reflex to assess the L5 root. Occasionally, a tibialis posterior or medial hamstring reflex can be elicited and, if asymmetric, suggests an L5 radiculopathy. However, both reflexes often are unobtainable in normal individuals.

ETIOLOGY

There are a vast number of causes of radiculopathy. The most common are structural lesions, including herniated discs, bony impingement from spondylosis, and mass lesions such as epidural abscesses and metastatic tumors to the spine.

Less well appreciated is that radiculopathy can occur on a microscopic level without evidence of a mass lesion. The cause can be infiltration by tumor (carcinomatous or lymphomatous meningitis), infiltration by granulomatous tissue (e.g., sarcoid), or infection (e.g., Lyme disease, herpes zoster, cytomegalovirus, herpes simplex). Rarely, cases of pure radiculopathy or polyradiculopathy may be due to acquired demyelinating neuropathy (e.g., early Guillain–Barré syndrome). In addition, radiculopathy can be seen as a result of infarction of the nerve root, which may occur in

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Table 29–1. Common Cervical Root Syndromes

Root	Pain Location	Sensory Disturbance	Weakness	Reflex Change
C3–4	Paraspinal muscles, superior shoulder	Neck	Diaphragm, nuchal muscles, strap muscles	None
C5	Neck, shoulder, anterior arm	Shoulder	Deltoid, supraspinatus, infraspinatus, rhomboids, biceps, brachioradialis	Biceps, brachioradialis
C6	Neck, shoulder, anterior upper arm extending to antecubital fossa	Thumb, index finger, radial forearm	Deltoid, supraspinatus, infraspinatus, rhomboids, biceps, brachioradialis, pronator teres, flexor carpi radialis, extensor carpi radialis	Biceps, brachioradialis
C7	Neck, shoulder, dorsum of forearm	Middle finger	Triceps, latissimus dorsi, pronator teres, flexor carpi radialis, extensor carpi radialis	Triceps
C8	Neck, shoulder, ulnar forearm	Ring, little fingers, hypothenar eminence	Intrinsic hand muscles, finger extensors, finger flexors	None
T1	Neck, shoulder, ulnar arm	Ulnar forearm	Intrinsic hand muscles (Horner's syndrome)	None

Adapted from Geckle, D.S., Hlavin, M.L., 1995. Spondylosis and disc disease. In: Samuels, M.A., Feske, S. (Eds.), Office practice of neurology. Churchill Livingstone, New York, NY.

Table 29–2. Common Lumbar Root Syndromes

Root	Pain Location	Sensory Disturbance	Weakness	Reflex Change
L3	Anterior thigh, groin	Anterior thigh	Iliopsoas, adductors, quadriceps	(Knee)
L4	Anterior thigh	Medial calf, medial foot	Quadriceps, adductors, (iliopsoas)	Knee
L5	Posterolateral thigh and calf, extending into great toe and dorsum of foot	Dorsum of foot, great toe, lateral calf	Tibialis anterior, tibialis posterior, extensor hallucis longus, peronei, gluteus medius, tensor fascia latae	None
S1	Posterolateral thigh and calf, extending into lateral toes and heel	Lateral foot, posterior calf, sole of foot	Gastroc-soleus, hamstrings, gluteus maximus	Ankle

Adapted from Geckle, D.S., Hlavin, M.L., 1995. Spondylosis and disc disease. In: Samuels, M.A., Feske, S., (Eds.), Office practice of neurology. Churchill Livingstone, New York, NY.

vasculitic neuropathy and presumably occurs commonly in diabetic polyradiculopathy. These nonstructural etiologies illustrate how a patient may have a clinical radiculopathy with completely normal imaging studies. It is in such cases that EMG is especially useful in demonstrating a physiologic radiculopathy.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of pain and radiating paresthesias includes not only radiculopathy but also proximal neuropathy, plexopathy and entrapment neuropathy. Although plexopathies are much less common than radiculopathies, separating plexopathy from radiculopathy on clinical grounds can be quite difficult. In addition, some entrapment neuropathies may be mistaken for radiculopathy, especially when the symptoms are mild. Because an entrapped nerve can cause referred pain and paresthesias, it is possible for distal entrapment to cause symptoms in more proximal segments. For instance, in ulnar neuropathy at the elbow, pain radiating into the upper arm or shoulder is not unusual. Some cases of carpal tunnel syndrome (CTS) are associated with pain in the forearm, the arm, and rarely the shoulder. The presence of referred pain along with distal paresthesias from entrapment neuropathies may suggest radiculopathy. However, pain in the neck or back and exacerbation of symptoms with neck or back movement do not occur in the common entrapment neuropathies and thus provide an important clinical clue pointing to radiculopathy.

Besides plexopathy, proximal neuropathy, and entrapment neuropathy, the major differential diagnosis of radiculopathy includes local orthopedic problems that result in pain and secondary muscle spasm. Often the key task in the EMG laboratory is to try to separate pain due to muscle spasm alone from pain due to true nerve root dysfunction.

ELECTROPHYSIOLOGIC EVALUATION

Nerve Conduction Studies

In patients with radiculopathy, nerve conduction studies typically are normal, and the electrodiagnosis is established

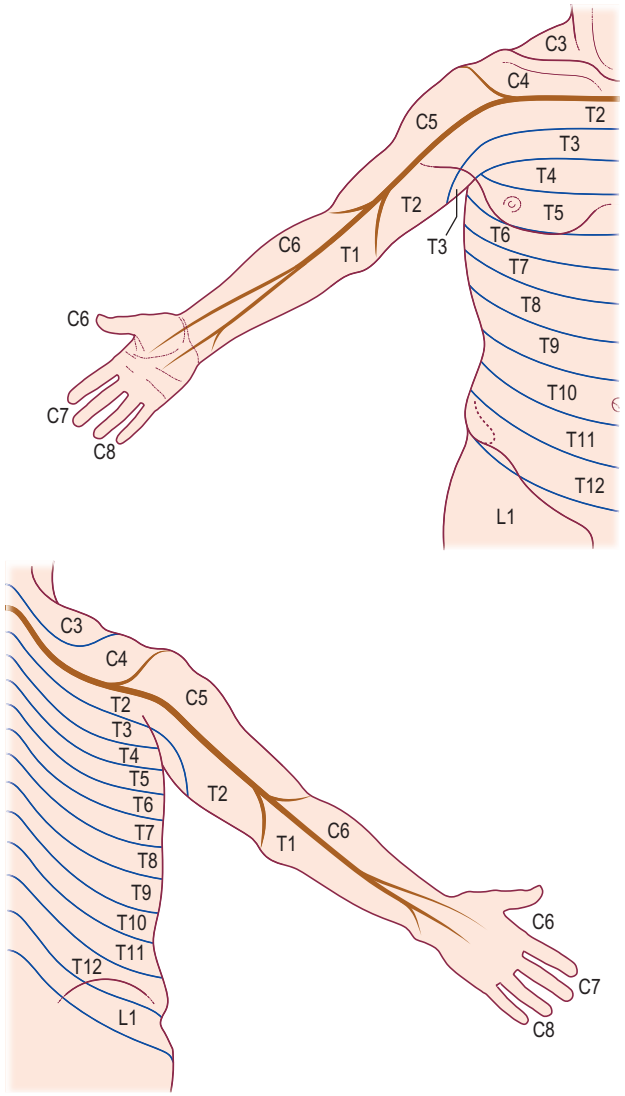

FIGURE 29–1 Cervical and thoracic dermatomes.
(From Aids to the examination of the peripheral nervous system. London: Baillière Tindall. With permission, 1986.)

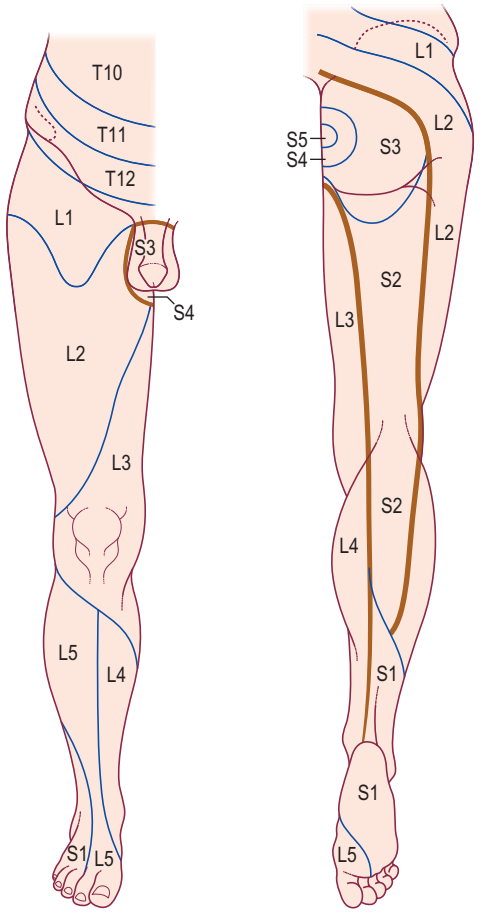

FIGURE 29–2 Lower thoracic and lumbosacral dermatomes.
(From Aids to the examination of the peripheral nervous system. London: Baillière Tindall. With permission, 1986.)

with needle EMG (Box 29–1). Although some motor abnormalities are occasionally seen in radiculopathy, the more important reason to perform nerve conduction studies is to exclude other conditions that may mimic radiculopathy, especially entrapment neuropathy and plexopathy. In cases of upper extremity lesions, ulnar neuropathy at the elbow and CTS must be excluded. Ulnar neuropathy and C8 radiculopathy both can present with pain in the arm associated with numbness of the little and ring fingers. Likewise, pain in the arm with paresthesias involving the thumb, index, and middle fingers may be seen in C6–C7 radiculopathy and CTS. In the case of lower extremity symptoms, one must exclude peroneal neuropathy at the fibular neck. Both peroneal palsy and L5 radiculopathy may present with pain in the leg, accompanied by footdrop and paresthesias over the dorsum of the foot and lateral calf. In more severe cases, the clinical differentiation between a radiculopathy and a common entrapment usually is straightforward. In mild or early cases, however, the distinction often is more difficult, and nerve conduction studies are useful to either demonstrate or exclude an entrapment neuropathy.

Depending on the underlying pathophysiology and the level of the lesion, abnormalities occasionally may be seen on routine motor conduction and F response studies in radiculopathy. If the pathophysiology is predominantly demyelinating, the underlying axons remain intact. In that case, any motor study, stimulating and recording distally, will show a normal latency, conduction velocity, and compound muscle action potential (CMAP) amplitude. The only possible abnormality will be in the F responses. Because the F responses assess conduction both distally and proximally, abnormal F responses with normal distal conduction studies suggest a proximal lesion, either in the proximal nerve, plexus or roots. Of course, F waves will be abnormal only if the recorded muscle is innervated by the affected nerve roots.

Table 29–3. Root Innervation of Major Upper Extremity Muscles

Root	Muscle	Nerve
C4 5	Rhomboids	Dorsal scapular
C5 6	Supraspinatus	Suprascapular
C5 6	Infraspinatus	Suprascapular
C5 6	Deltoid	Axillary
C5 6	Biceps brachii	Musculocutaneous
C5 6	Brachioradialis	Radial
C5 6 7	Serratus anterior	Long thoracic
C5 6 7	Pectoralis major: Clavicular	Lateral pectoral
C6 7 8 T1	Pectoralis major: Sternal	Medial pectoral
C6 7	Flexor carpi radialis	Median
C6 7	Pronator teres	Median
C6 7	Extensor carpi radialis longus	Radial
C6 7 8	Latissimus dorsi	Thoracodorsal
C6 7 8	Triceps brachii	Radial
C6 7 8	Anconeus	Radial
C7 8	Extensor digitorum communis	Radial
C7 8	Flexor digitorum sublimis	Median
C7 8	Extensor indicis proprius	Radial
C7 8	Extensor carpi ulnaris	Radial
C7 8 T1	Flexor pollicis longus	Median
C7 8 T1	Flexor digitorum profundus	Median/Ulnar
C8 T1	Flexor carpi ulnaris*	Ulnar
C8 T1	First dorsal interosseus	Ulnar
C8 T1	Abductor digiti minimi	Ulnar
C8 T1	Abductor pollicis brevis	Median

*In some individuals, the flexor carpi ulnaris may have a C7 contribution.
Note: Underlining indicates predominant root innervation.

Table 29–4. Root Innervation of Major Lower Extremity Muscles

Root	Muscle	Nerve
L2 3 4	Iliacus	Femoral
L2 3 4	Rectus femoris	Femoral
L2 3 4	Vastus lateralis and medialis	Femoral
L2 3 4	Adductors	Obturator
L4 5	Tibialis anterior	Deep peroneal
L4 5	Extensor digitorum longus	Deep peroneal
L4 5 S1	Extensor hallucis longus	Deep peroneal
L4 5 S1	Extensor digitorum brevis	Deep peroneal
L4 5 S1	Medial hamstrings	Sciatic
L4 5 S1	Gluteus medius	Superior gluteal
L4 5 S1	Tensor fascia latae	Superior gluteal
L5 S1	Tibialis posterior	Tibial
L5 S1	Flexor digitorum longus	Tibial
L5 S1	Peronei	Superficial peroneal
L5 S1	Lateral hamstrings (biceps femoris)	Sciatic
L5 S1 2	Gastrocnemius – lateral	Tibial
L5 S1 2	Gluteus maximus	Inferior gluteal
L5 S1 2	Abductor hallucis brevis	Tibial–medial plantar
S1 2	Abductor digiti quinti pedis	Tibial–lateral plantar
S1 2	Gastrocnemius – medial	Tibial
S1 2	Soleus	Tibial

Note: Underlining indicates predominant root innervation.

In the upper extremity, F waves are routinely recorded only for the median and ulnar nerves, which are C8–T1 innervated. Thus, median and ulnar F-wave abnormalities may be seen in C8–T1 radiculopathy; however, these roots are infrequently affected by disc or bone impingement, the most common causes of radiculopathy. A radiculopathy at C5, C6, or C7, which are more common sites of root impingement, will not be reflected in the median or ulnar F responses. The situation is different in the lower extremities. The distally recorded peroneal and tibial muscles (extensor digitorum brevis, abductor hallucis brevis) are innervated predominantly by the L5 and S1 nerve roots, respectively. These levels are often affected by radiculopathy. Thus, in L5–S1 radiculopathies, peroneal and tibial F responses may be prolonged, especially in comparison with the contralateral side.

The H reflex occasionally is helpful in evaluating lower extremity radiculopathy. However, the H reflex, recorded from the soleus, can be used to evaluate only a possible S1 radiculopathy and is most useful when the symptomatic side is compared with the asymptomatic side. The H reflex is the electrical correlate of the ankle reflex; accordingly, it may be delayed or absent in any lesion that depresses the ankle jerk, including polyneuropathy, sciatic neuropathy, lumbosacral plexopathy, and S1 radiculopathy. Unfortunately, the combination of normal distal motor nerve conduction studies and an abnormal H reflex cannot help differentiate between plexopathy and radiculopathy, but can only suggest a proximal lesion.

Box 29–1. Recommended Nerve Conduction Study Protocol for Radiculopathy

Upper Extremity

Motor studies:

- Perform median and ulnar motor conduction studies, recording abductor pollicis brevis and abductor digiti minimi, respectively. Be sure to exclude carpal tunnel syndrome in suspected C6–C7 radiculopathy and ulnar neuropathy at the elbow in suspected C8 radiculopathy. Ideally, studies should be performed bilaterally if CMAP distal latency, amplitude, or conduction velocity is abnormal or borderline.

Sensory/mixed studies:

- Perform at least one sensory study, ideally in the distribution of the suspected radiculopathy (see Table 29–6). It is best to perform the sensory studies bilaterally if the amplitude on the symptomatic side is low or borderline.
- In suspected C6–C7 radiculopathy (paresthesias into thumb, index, and middle fingers), perform at least one median versus ulnar internal comparison study (e.g., median versus ulnar palm-to-wrist mixed studies), as a sensitive internal control, to definitely exclude electrophysiologic evidence of median neuropathy across the wrist.

Late responses:

- Perform median and ulnar F responses. In suspected C8 radiculopathy, these should be performed bilaterally if the results are abnormal or borderline on the symptomatic side.

Lower extremity

Motor studies:

- Perform peroneal and tibial motor conduction studies, recording extensor digitorum brevis and abductor hallucis brevis, respectively. Be sure to exclude peroneal palsy at the fibular neck, especially in suspected L5 radiculopathy. Ideally, studies should be performed bilaterally if CMAP distal latency, amplitude or conduction velocity is abnormal or borderline.

Sensory studies:

- Perform at least one sensory study, ideally in the distribution of the suspected radiculopathy (see Table 29–6). It is best to perform these studies bilaterally if the amplitude on the symptomatic side is low or borderline.

Late responses:

- Perform tibial and peroneal F responses. It is best to perform these studies bilaterally if the results are abnormal or borderline on the symptomatic side.
- Perform H reflexes to soleus bilaterally, especially when considering S1 radiculopathy.

CMAP, compound muscle action potential.

If the pathophysiology also involves axonal loss, nerve conduction abnormalities may be seen in the motor conduction studies. Here again, abnormalities are seen only if the recorded muscle is innervated by the affected nerve root. Axonal loss may result in a decreased CMAP amplitude, with some slowing of conduction velocity and distal latency, especially if the largest fibers are involved. For instance, in an L5–S1 radiculopathy associated with axonal loss, the ipsilateral peroneal and tibial motor responses may have slightly slowed conduction velocities, slightly prolonged distal latencies, and reduced CMAP amplitudes, especially in comparison with the contralateral side. The distal latency prolongation and conduction velocity slowing, however, should never drop into the demyelinating range.

Sensory studies are the most important part of the nerve conduction studies in the assessment of radiculopathy. The sensory nerve action potential (SNAP) remains normal in lesions proximal to the dorsal root ganglion (Figure 29–3). Nearly all radiculopathies, including those caused by compression from herniated discs and spondylosis, damage the root proximal to the dorsal root ganglion (Figure 29–4). Conversely, lesions at or distal to the dorsal root ganglion result in decreased SNAP amplitudes if they are associated with axonal loss. Thus, lesions of the plexus and peripheral nerve (proximal and distal nerve) are associated with abnormal SNAPs, whereas lesions of the nerve root result in normal SNAPs.

It is always imperative to check the SNAP that is in the distribution of the sensory symptoms (Table 29–5). For instance, if a patient has pain down the arm with tingling and paresthesias of the middle finger, the median sensory response to the middle finger should be checked. In such a case, if the lesion is at or distal to the dorsal root ganglion (e.g., in the brachial plexus or median nerve) and there is axonal loss, the SNAP amplitude will be abnormal, if enough time has passed that wallerian degeneration has taken place. On the other hand, if the lesion is proximal to the dorsal root ganglion (e.g., C7 radiculopathy), the SNAP amplitude will be normal. The presence of a normal SNAP yields important diagnostic information. A normal SNAP in the same distribution as sensory symptoms and signs should always suggest a lesion proximal to the dorsal root ganglion (although a proximal demyelinating or acute peripheral nerve lesion also can result in a normal SNAP). One important rare exception to his rule is discussed below.

Superficial Peroneal SNAP and L5 Radiculopathy: the Rare Exception

If one follows the important tenet of EDX testing that SNAPs are normal in radiculopathy (or any lesion proximal to the dorsal root ganglia), and abnormal in disorders of the peripheral nerve associated with axonal loss (at or distal to the dorsal root ganglia), one will be correct over 99% of the time. However, there is one important exception that deserves comment: *in some rare cases of L5 radiculopathy, the superficial peroneal SNAP may be abnormal (abnormal defined as absent, or reduced in amplitude either in an absolute sense or being 50% or less of the contralateral*

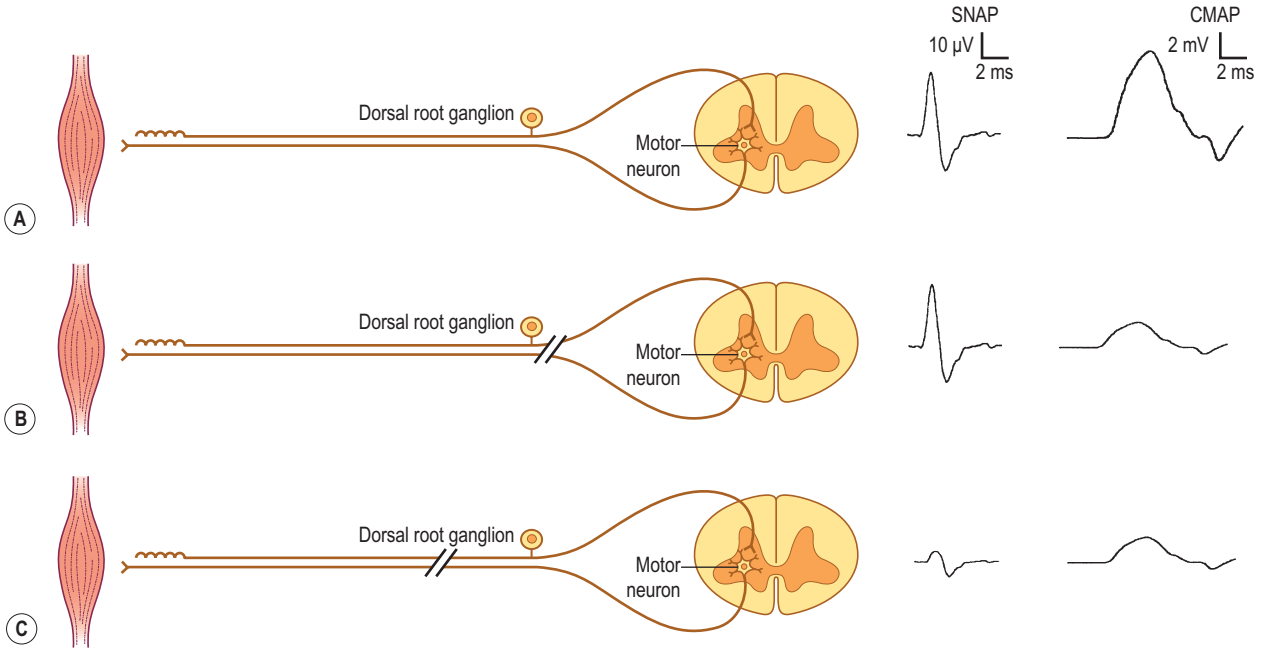

FIGURE 29–3 Sensory and motor potentials in axonal loss lesions distal and proximal to the dorsal root ganglion. **A:** Normal. **B:** Lesion proximal to the dorsal root ganglion. **C:** Lesion at or distal to the dorsal root ganglion. In axonal loss lesions, both proximal and distal to the dorsal root ganglion, degeneration of motor fibers results in decreased compound motor action potential amplitudes. If larger motor fibers are lost, conduction velocities and distal latencies also may slow slightly. The situation is different for sensory fibers. Lesions proximal to the dorsal root ganglion result only in degeneration of sensory fibers proximally into the spinal cord. Because the dorsal root ganglion is a bipolar cell, it remains in continuity with the distal sensory fibers. Therefore, sensory nerve action potentials (SNAPs), when stimulated and recorded distally, remain normal. In axonal loss lesions at or distal to the dorsal root ganglion, distal sensory fibers degenerate, as do the motor fibers. Accordingly, SNAPs are reduced in lesions of the plexus and peripheral nerve but are normal in radiculopathies and other lesions proximal to the dorsal root ganglion.

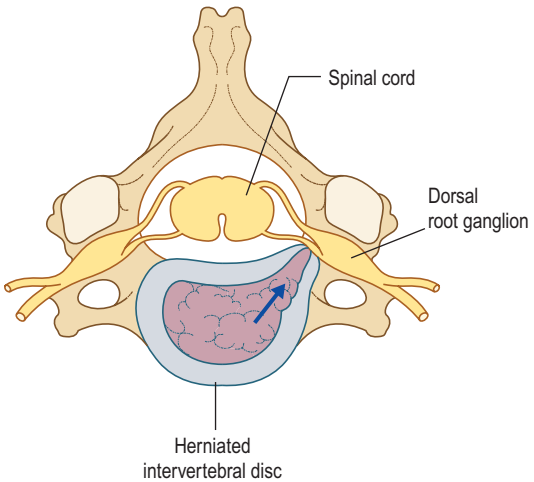

FIGURE 29–4 Radiculopathy and sparing of the dorsal root ganglion. Herniated intervertebral discs are a common cause of cervical and lumbosacral radiculopathy. Herniated discs are most often lateral and posterior, with the dorsal root ganglion located distal to the herniation. In disc herniations, this anatomic relationship results in injury to the roots but sparing of the dorsal root ganglion and the peripheral sensory nerves. Consequently, sensory conduction studies remain normal in radiculopathy.
(From Wilbourn, A.J., 1993 Radiculopathies. In: Brown, W.F., Bolton, C.F. (Eds.), Clinical electromyography, 2nd ed. Butterworth, Boston. With permission.)

superficial peroneal SNAP). The reason behind this finding is not completely understood. In cadaver and other anatomic studies, the L5 dorsal root ganglion is actually located proximal to the intervertebral foramen in 10–40% of individuals, where it is theoretically susceptible to external intraspinal compression (e.g., from a disc). However, some S1 dorsal root ganglia are similarly located, but abnormalities of the sural sensory nerve are never seen in S1 radiculopathies. This discrepancy may be explained by the fact that in cadaver studies, some L5 dorsal root ganglia are found indented by the superior facet. The superior facet frames part of the intervertebral foramen. In contrast, there is no facet joint that frames the intervertebral foramen for the S1 root.

Regardless of the underlying etiology, this finding of an abnormal superficial peroneal SNAP can be seen, although very rarely, in L5 radiculopathy. *The take-home message is the following: in an EDX study wherein all the clinical and electrophysiologic findings are consistent with an L5 radiculopathy, with the exception of an abnormal superficial peroneal SNAP, one can form an EDX impression of an L5 radiculopathy, with the important proviso in the report that these findings could also represent a lumbosacral plexus lesion.* Conversely, if the EDX impression is that of a lumbosacral plexopathy, and the only abnormal sensory response

Table 29–5. Sensory Potentials to Check in Radiculopathy

SNAP	Root
Lateral antebrachial cutaneous	C5–C6
Radial to the thumb	C6
Median to the thumb	C6
Radial to the snuffbox	C6–C7
Median to the index finger	C6–C7
Median to the middle finger	C7
Median to the ring finger	C7–C8
Ulnar to the ring finger	C7–C8
Ulnar to the little finger	C8
Dorsal ulnar cutaneous	C8
Medial antebrachial cutaneous	T1
Saphenous	L4
Superficial peroneal sensory	L5
Sural	S1

SNAP, sensory nerve action potential.

Note: SNAPs are normal in lesions proximal to the dorsal root ganglion, including lesions resulting in radiculopathies. When evaluating a possible radiculopathy, one should examine at least one SNAP in the distribution of the suspected radiculopathy. For example, the ulnar SNAP to the little finger should be normal in a C8 radiculopathy. If it is abnormal, the lesion likely is not at the root, unless there is another reason for the SNAP to be abnormal, such as a superimposed ulnar neuropathy at the elbow.

Box 29–2. Recommended Electromyographic Protocol for Radiculopathy

1. Examine the relevant myotome first. If possible, sample at least two muscles in each of the following areas: paraspinal, proximal and distal limb. In each limb area, try to use muscles with similar root innervation but different peripheral nerve innervation.
2. If abnormalities are found, examine muscles in adjacent myotomes, above and below the suspected lesion level, to exclude a more widespread or diffuse lesion.
3. If findings are mild or equivocal, compare with a contralateral asymptomatic muscle.
4. In the post-spinal surgery setting, fibrillation potentials in the paraspinal muscles do not necessarily have diagnostic significance; thus, they are not helpful to sample.

is the superficial peroneal SNAP, it is likewise essential to put a proviso in the report that the findings could also represent an L5 radiculopathy with the very unusual variant that the superficial peroneal sensory fibers are involved.

Electromyographic Approach

The needle EMG strategy in radiculopathy is straightforward. Distal, proximal, and paraspinal muscles in the symptomatic extremity are sampled, looking for abnormalities in a myotomal pattern that are beyond the distribution of any one nerve (Box 29–2). It is important to exclude a mononeuropathy, polyneuropathy, or more diffuse process that might account for the signs and symptoms.

1. *Muscles innervated by the same myotome but by different nerves must be sampled to exclude a mononeuropathy*. For example, the finding of fibrillation potentials and decreased recruitment of motor unit action potentials (MUAPs) in the triceps brachii (C6–**C7**–C8), extensor carpi radialis (**C6–C7**), and extensor carpi ulnaris (**C7–C8**) could indicate an acute, predominantly C7 radiculopathy, since they all share this nerve root. However, because each of these muscles is also innervated by the radial nerve, one could not differentiate between a radial neuropathy and a C7 radiculopathy by sampling only these muscles. If, however, the flexor carpi radialis (**C6–C7**) or pronator teres (**C6–C7**) were also sampled and showed fibrillation potentials with reduced recruitment of MUAPs, the pattern of abnormalities could no longer be explained by a single nerve lesion (radial neuropathy) because the last two muscles are both innervated by the median nerve. Since all of these muscles have C7 innervation in common, despite different peripheral nerve innervation, this pattern of abnormalities points toward a radiculopathy as the lesion. Note that while nearly all muscles are innervated by multiple myotomes, certain muscles are predominantly innervated by one myotome, and these muscles are the most useful in the electrodiagnosis of radiculopathy (Tables 29–6 and 29–7).
2. *Proximal and distal muscles that are innervated by the same myotome should be sampled to exclude a distal-to-proximal pattern of abnormalities such as occurs in polyneuropathy*. For example, the finding of fibrillation potentials with reduced recruitment of MUAPs in the extensor hallucis longus (**L5**–S1), medial gastrocnemius (**S1**–S2), and peroneus longus (**L5–S1**) muscles would be consistent with an L5–S1 radiculopathy. However, because these are all distal muscles, one could not exclude a typical distal polyneuropathy, especially if the sural sensory potential is borderline low. On the other hand, if more proximal S1 muscles, such as the gluteus maximus (L5–**S1**–S2), also show similar abnormalities, a distal-to-proximal gradient would be excluded, making radiculopathy the more likely diagnosis.
3. *Muscles innervated by myotomes above and below the suspected lesion level must be sampled to exclude a more widespread or diffuse process*. For example, if a C7 radiculopathy is suspected, muscles predominantly innervated by the C5–C6 and C8–T1 nerve roots also should be sampled.
4. *The paraspinal muscles should always be examined*. Examination of the paraspinal muscles is crucial in the evaluation of radiculopathy. The paraspinal muscles are innervated by the dorsal rami, which arise directly from the spinal nerves. Neuropathic abnormalities in these muscles nearly always imply a lesion at or

Table 29–6. Electromyography in Upper Extremity Radiculopathy: Most Useful Muscles to Sample

	C5	C6	C7	C8	T1
Dorsal scapular nerve					
Rhomboid major/minor					
Suprascapular nerve					
Supraspinatus					
Infraspinatus					
Axillary nerve					
Deltoid					
Musculocutaneous nerve					
Biceps brachii					
Median nerve					
Pronator teres					
Flexor carpi radialis					
Flexor pollicis longus					
Abductor pollicis brevis					
Ulnar nerve					
Flexor carpi ulnaris					
Flexor digitorum profundus (V)					
Abductor digiti minimi					
First dorsal interosseous					
Radial nerve					
Triceps					
Brachioradialis					
Extensor carpi radialis					
Extensor digitorum communis					
Extensor carpi ulnaris					
Extensor indicis proprius					

Note: Green squares indicate "marker" muscles that are most often abnormal for that root in an isolated radiculopathy. Blue squares indicate muscles that may be involved, but are abnormal less frequently. This chart shows those muscles that are most helpful in making the electrodiagnosis of radiculopathy but does not indicate the entire myotomal representation of the individual muscle (see Table 29–3).

From Wilbourn, A.J., 1993. Radiculopathies. In: Brown, W.F., Bolton, C.F. (Eds.), Clinical electromyography, 2nd ed. Butterworth, Boston, with permission.

proximal to the nerve roots. Other than the presence of normal sensory nerve conduction studies, abnormalities in the paraspinal muscles are the only other finding that can conclusively differentiate radiculopathy from plexopathy. Unfortunately, the paraspinal muscles are affected in only about 50% of cases of radiculopathy. *Thus, the absence of paraspinal abnormalities cannot exclude a radiculopathy; however, the presence of paraspinal abnormalities clearly localizes the lesion to the root or anterior horn cell level.* Note that if the patient has had previous neck or back surgery, the paraspinal muscles in the area of previous surgery may remain abnormal for years after the surgery, and any abnormal findings in these muscles would not help differentiate a new lesion from a remote effect of previous surgery. Thus, paraspinal muscles in the area of previous surgery are generally not sampled (see below).

TIME COURSE IN RADICULOPATHY

To interpret an electrodiagnostic study properly, the electromyographer must fully understand the time-related

Table 29–7. Electromyography in Lower Extremity Radiculopathy: Most Useful Muscles to Sample

	L2	L3	L4	L5	S1	S2
Inferior gluteal nerve						
Gluteus maximus						
Superior gluteal nerve						
Gluteus medius						
Tensor fascia latae						
Obturator nerve						
Adductor longus						
Femoral nerve						
Iliopsoas						
Rectus femoris						
Vastus lateralis/medialis						
Sciatic nerve						
Medial hamstrings						
Lateral hamstrings						
Deep peroneal nerve						
Tibialis anterior						
Extensor hallucis longus						
Superficial peroneal nerve						
Peroneus longus						
Tibial nerve						
Medial gastrocnemius						
Soleus						
Flexor digitorum longus						
Tibialis posterior						
Abductor hallucis brevis						
Abductor digiti minimi pedis						

Note: Green squares indicate "marker" muscles that are most often abnormal for that root in an isolated radiculopathy. Blue squares indicate muscles that may be involved, but are abnormal less frequently. This chart shows those muscles that are most helpful in making the electrodiagnosis of radiculopathy but does not indicate the entire myotomal representation of the individual muscle (see Table 29–4).

From Wilbourn, A.J., 1993. Radiculopathies. In: Brown, W.F., Bolton, C.F. (Eds.), Clinical electromyography, 2nd ed. Butterworth, Boston, with permission.

changes that occur in radiculopathy. In all neuropathic lesions resulting in axonal loss, the time that elapses before a muscle begins to show fibrillation potentials (i.e., denervation) is dependent on the intervening distance between the lesion and the muscle. As the normal process of reinnervation then follows, it usually is quite slow and prolonged. Consider the following example. A patient who is otherwise well lifts a heavy box today, herniating the L4–L5 disc, resulting in severe compression of the L5 nerve root. He develops immediate pain in the back that radiates down the buttock and into the leg, along with numbness over the dorsum of the foot and weakness of hip abduction and ankle dorsiflexion.

In the EMG laboratory, the only abnormality that is seen in the acute phase is decreased recruitment of MUAPs in clinically weak muscles – in this case, the weak L5-innervated muscles. Decreased recruitment occurs because some of the L5 motor units have been blocked or lost. MUAP morphology remains normal during the acute phase. The next change occurs at approximately day 10 to 14, when fibrillation potentials and positive waves (i.e., denervating potentials) may develop in the paraspinal muscles (i.e., those

muscles most proximal to the lesion). This is followed at 2 to 3 weeks by similar changes in the proximal L5-innervated muscles (e.g., tensor fascia latae, gluteus medius, etc.). It is not until week 3 or 4 that fibrillation potentials develop in the lower leg L5-innervated muscles (e.g., tibialis anterior), and it can take until week 5 or 6 for the most distally innervated L5 muscles to develop denervating potentials. Throughout this time, the MUAPs remain normal in morphology, with decreased recruitment, just as they were on day 1. After denervation, reinnervation then begins to occur, with the development first of polyphasic MUAPs and later of long-duration, large-amplitude, polyphasic MUAPs. Like denervation, reinnervation occurs first in the most proximal muscles. As months pass, reinnervation is more successful, and the fibrillation potentials diminish, leaving large reinnervated MUAPs with decreased recruitment.

Thus, by examining the combination of spontaneous activity, MUAP morphology, and recruitment pattern, one can approximate the time course of any neuropathic lesion, including radiculopathy.

LIMITATIONS OF THE NEEDLE ELECTROMYOGRAPHIC STUDY IN RADICULOPATHY

Although the EMG study is very sensitive to the presence and approximate localization of a radiculopathy, equivocal or false-negative studies are not uncommon in patients with true radiculopathy. The limitations of nerve conduction studies and needle EMG must be appreciated by both the electromyographer performing the study and the physician ordering and using the results of the study to treat a patient with suspected radiculopathy. The following points should be kept in mind.

It may be Difficult to Localize a Radiculopathy to a Single Root Level

Although the EMG study is a sensitive test for identifying radiculopathy, it may still be difficult to identify the specific segmental level because most muscles are innervated by more than one myotome. For instance, the finding of fibrillation potentials with decreased recruitment of MUAPs in the biceps, deltoid, infraspinatus, and mid-cervical paraspinal muscles is consistent with a C5–C6 myotomal pattern. In such a case, one can be certain that the lesion is not due to a single peripheral nerve injury because abnormalities are present in muscles innervated by the dorsal rami as well as by the musculocutaneous, axillary, and suprascapular nerves. However, in such a case, it is more challenging and sometimes impossible to differentiate between a C5 and C6 radiculopathy.

In such a case, one would next sample muscles that belong to one but not the other myotome. For example, it would be helpful to sample muscles with partial C5 but without C6 innervation, as well as muscles with partial C6 but without C5 innervation. For instance, if the rhomboids (C4–C5) were sampled and found to be normal while the pronator teres (C6–C7) showed fibrillation potentials, then a C6 lesion would be more likely than a C5 lesion. The same approach is used to identify radiculopathies at other levels. One can see that it is just as important to identify which muscles are normal as which are abnormal to try to identify the specific root level involved. Often multiple muscles must be sampled to try to define the level of the involved myotome.

In studies of patients who had a surgically defined single-level radiculopathy, the correct level often could be deduced from extensive needle EMG studies (Figures 29–5 and 29–6). However, not infrequently there was significant overlap between adjacent segments, making a single root localization difficult. The most difficult levels to differentiate were C6 from C7.

If the Lesion is Acute, the Electromyographic Study may be Normal

As noted earlier, during the first 10 to 14 days after the onset of an acute radiculopathy, there are no needle EMG abnormalities except for decreased recruitment of MUAPs in weak muscles. Because it is unusual to find significant weakness in radiculopathy, the EMG study often is completely normal in the acute setting. Fibrillation potentials take several weeks to develop in the more distal limb muscles; therefore, it often is best to wait several weeks before sending a patient for an EMG study, unless one is willing to repeat a normal study after several weeks to look for new changes.

If the Radiculopathy is Purely Demyelinating, the Electromyographic Study will be Normal

If the nerve root is compressed, resulting in demyelination without axonal loss, the needle EMG study may be completely normal. Diagnosing radiculopathy with EMG usually rests on the identification of denervation and reinnervation, signs of axonal loss. If there is no axonal loss, the study usually is normal. Only if demyelination results in significant conduction block, with accompanying weakness, will MUAP recruitment be diminished. This situation, however, is rarely seen in radiculopathy.

If the Sensory Nerve Root is Predominantly Affected, the Electromyographic Study will be Normal

Most patients with radiculopathy have prominent sensory symptoms, including pain and paresthesias, indicating dysfunction of the sensory nerve root. If the sensory nerve root is preferentially affected and the motor nerve root is spared, the EMG study will be normal. Unfortunately, there is no good way to assess the proximal sensory segments using routine nerve conduction studies. Somatosensory evoked potentials are often used to assess the proximal segments,

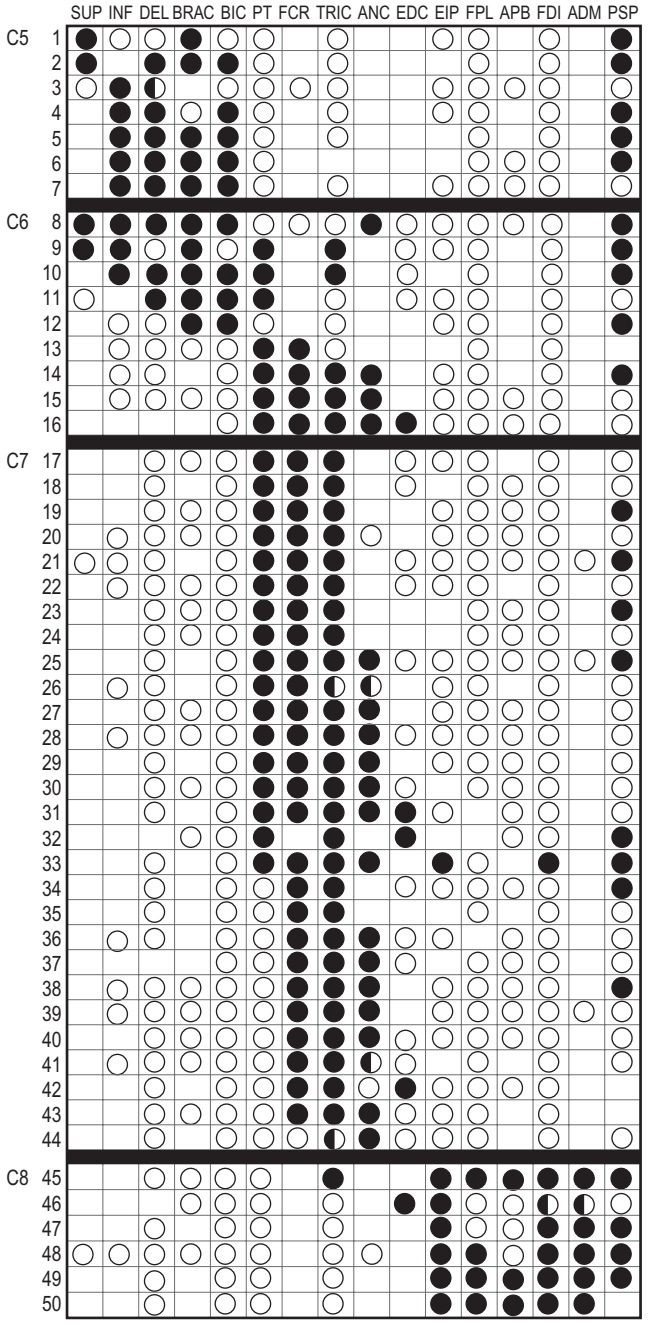

FIGURE 29–5 Cervical radiculopathy: needle electromyographic results in 50 patients grouped by surgically defined root level of involvement. Closed circles represent positive waves or fibrillation potentials, with or without neurogenic recruitment and motor unit action potential changes. Half-closed circles represent neurogenic recruitment changes only. Open circles represent normal examination. ADM, abductor digiti minimi; ANC, anconeus; APB, abductor pollicis brevis; BIC, biceps; BRAC, brachioradialis; DEL, deltoid; EDC, extensor digitorum communis; EIP, extensor indicis proprius; FCR, flexor carpi radialis; FDI, first dorsal interosseous; FPL, flexor pollicis longus; INF, infraspinatus; PSP, paraspinal muscle; PT, pronator teres; SUP, supraspinatus; TRIC, triceps.
(From Levin, K.H., Maggiano, H.J., Wilbourn, A.J., 1996. Cervical radiculopathies: comparison of surgical and EMG localization of single-root lesions. Neurology 46, 1022–1025. With permission.)

but they share many of the limitations of the F response. Most areas of skin are innervated by multiple dermatomes. Accordingly, although a single spinal nerve root may be severely damaged with resultant slowing, the somatosensory evoked latencies may be normal because the adjacent nerve roots (and the overlapping dermatomes they innervate) are not affected.

Different Fascicles may be Preferentially Affected or Spared

Just as in other entrapment syndromes, it is not unusual for some fascicles within a myotome to be affected while others are spared. In fact, some muscles of a particular myotome may be markedly involved, whereas others are affected only minimally or not at all. For example, in some C7 radiculopathies, the triceps may show fibrillation potentials and reduced recruitment of MUAPs, whereas the flexor carpi radialis is essentially normal, although both receive substantial C7 innervation. Thus, the yield of abnormal EMG findings in a radiculopathy clearly increases as more muscles are sampled. As always, however, the electromyographer must balance patient comfort, the length of the test, and the goal of obtaining as much useful information as possible.

The Paraspinal Muscles may be Normal

One expects the paraspinal muscles to be abnormal in radiculopathy, and they often are (Figures 29–5 and 29–6). In some cases, however, they are normal. This may be due to fascicular sparing of fibers to the dorsal rami or may simply be due to sampling error. In addition, some patients have difficulty tolerating the paraspinal examination and consequently may not be able to relax those muscles. The paraspinal needle examination is best done with the patient lying on his or her side in the fetal position, with the side to be studied facing up. This position often will relax the paraspinal muscles. If relaxation is incomplete, however, it may be difficult or impossible to exclude denervation. This situation is encountered most often when studying the thoracic paraspinal muscles.

In addition, reinnervation, like denervation, occurs first in the most proximal muscles. Accordingly, the paraspinal muscles are the first to be reinnervated, often resulting in a pattern of denervation in the limb muscles with sparing of the paraspinals, a pattern equally consistent with plexopathy. In such a case, the finding of normal SNAPs in the distribution of sensory complaints can help to differentiate plexopathy from radiculopathy. One also can look for reinnervated MUAPs in the paraspinal muscles, although it is sometimes difficult for patients to activate these muscles.

Abnormal Paraspinal Muscles are Useful in Identifying a Radiculopathy but Not the Segmental Level of the Lesion

The paraspinal muscles (also known as the erector spinae muscles) run along the spine from the occipital bone in the

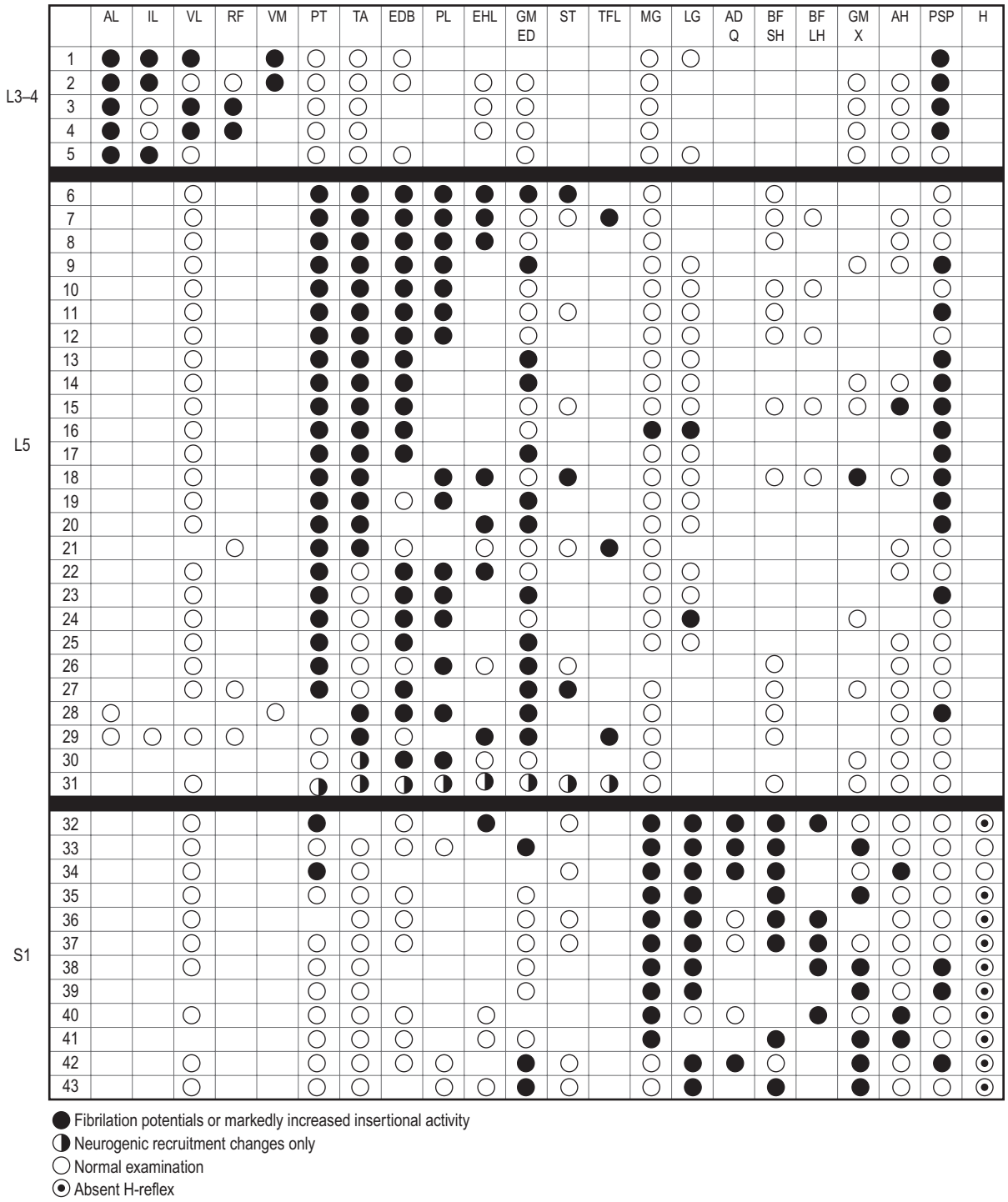

FIGURE 29–6 Lumbosacral radiculopathy: needle electromyographic results in 43 patients grouped by surgically defined root level of involvement. Closed circles represent positive waves or fibrillation potentials, with or without neurogenic recruitment and motor unit changes. Half-closed circles represent neurogenic recruitment changes only. Open circles represent normal examination. ADQ, abductor digiti quinti; AH, abductor hallucis; AL, adductor longus; BFLH, biceps femoris, long head; BFSH, biceps femoris, short head; EDB, extensor digitorum brevis; EHL, extensor hallucis longus; GMED, gluteus medius; GMX, gluteus maximus; H, H-reflex; IL, iliopsoas; LG, lateral gastrocnemius; MG, medial gastrocnemius; PL, peroneus longus; PSP, paraspinal muscles; PT, posterior tibialis; RF, rectus femoris; ST, semitendinous; TA, tibialis anterior; TFL, tensor fascia latae; VL, vastus lateralis.

(From Levin, K.H., 2002. Electrodiagnostic approach to the patient with suspected radiculopathy. Neurol Clin 20, 397–421. With permission.)

skull down to the sacrum. Functionally, they are divided into three groups: (1) the iliocostalis (superficial, lateral); (2) the longissimus (superificial, medial); and (3) the multifidus (deep, adjacent to the spinous process and lamina). In the superficial layers of the iliocostalis and longissimus, which are most often sampled during needle EMG, there is marked overlap in the innervation. Thus, a radiculopathy at one level, determined by examination of the limb muscles, may show denervation in the paraspinal muscles not only at the involved level but also at one or more levels above and below. *Therefore, EMG abnormalities in these paraspinal muscles can be used only to mark the lesion as at or proximal to the nerve root level, but cannot be used to identify which specific nerve root level is involved.*

In contrast, the deep layer of paraspinal muscles, the multifidus, are invariably innervated by only a single nerve root – the one below the spinous process from which they originate. Thus, if these muscles are sampled and are abnormal, the abnormalities are specific to the root (and level) supplying that muscle. The multifidus muscles laterally flex and rotate the spine to the opposite side. The following technique to identify and sample the multifidus has been described:

- The spinous process is first identified and marked.
- The needle insertion point is located 2.5 cm lateral and 1.0 cm rostral to the spinous process (Figure 29–7).
- The needle is directed medially at a 45 degree angle and inserted no more than 3.5 cm.
- When bone is reached, the needle is slightly withdrawn.
- If no bone is encountered, the needle is pulled back and redirected at 60 degrees but no deeper than 5 cm.

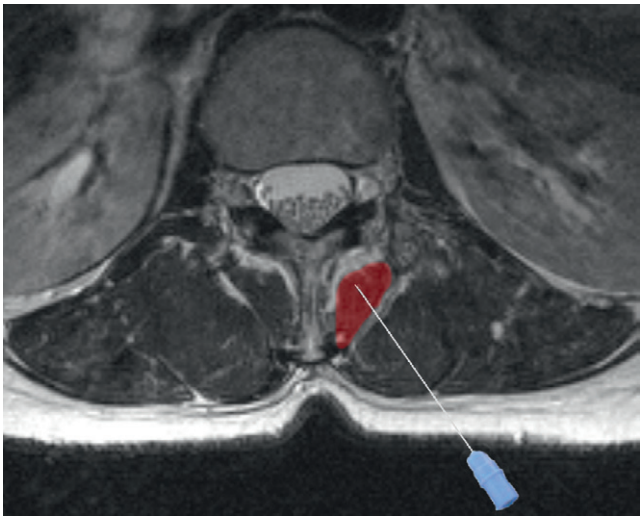

FIGURE 29–7 Needle EMG sampling of the multifidus muscle. The paraspinal muscles consist of three groups: the iliocostalis which is superficial and lateral, the longissimus which is superficial and medial, and the multifidus which is deep. Multiple root levels innervate the superficial iliocostalis and longissimus muscles. However, the multifidus is only supplied by a single nerve root. Techniques to sample the multifidus have been described (see text). In this MRI illustration of the lumbar spine, the EMG needle is in the multifidus muscle (red). Note that the multifidus is deep and adjacent to the spinous process and lamina.

Using this technique, one samples the multifidus muscle above the spinous process that was originally marked. For example, if one identifies the L4 spinous process, this technique would then sample the L3 multifidus muscle, innervated by the L3 root.

Although this technique is appealing because it tries to determine the level of the radiculopathy based on the paraspinal exam, in practice it has several limitations. In cadaver studies, the accuracy of sampling the correct multifidus was approximately 80% (i.e., 20% false-positive rate). Second, it relies on identifying the spinous processes and their correct corresponding levels on the basis of anatomic landmarks – either counting up from the lowest spinous process (L5), and/or identifying the L4 spinous process as being at the highest level of the iliac crests. In many individuals, these landmarks can be difficult to determine; in overweight patients, it is nearly impossible. *Thus, with these limitations in mind, it is prudent to use abnormalities in the paraspinal muscles as a marker that the lesion is at or proximal to the root level, but leave the determination of the actual root level to the pattern of abnormalities seen in the limb muscles.*

There is no Difference between a Radiculopathy/Polyradiculopathy and Focal/Diffuse Motor Neuron Disease on the Electromyographic Study

This is a very important concept often overlooked by electromyographers. Based on EMG results alone, an abnormality of the nerve root cannot be distinguished from an abnormality of the motor neurons supplying that root. The EMG and nerve conduction studies are identical in both conditions. First, all sensory nerve conduction studies are normal in both conditions. In radiculopathy, the sensory roots may be involved, but the SNAPs remain normal because the lesion is proximal to the dorsal root ganglion. In motor neuron disease, the sensory nerves are not involved. Second, motor conduction studies are normal in both radiculopathy and motor neuron disease, unless the muscles being recorded are innervated by the involved roots or anterior horn cells. If there is axonal loss from nerve root or anterior horn cell damage, the CMAP amplitudes may be decreased, with slight prolongation of distal latency and slight slowing of conduction velocity in both situations. Third, in both conditions the EMG shows denervation, reinnervation, or both in the involved myotomes and paraspinal muscles. Consequently, there is no electrophysiologic difference between a polyradiculopathy and diffuse motor neuron disease, or between an isolated radiculopathy and motor neuron disease affecting a single segment.

Although EMG cannot differentiate between a disorder of the nerve roots and a disorder of the motor neurons, there are clear and unequivocal *clinical* differences that allow the distinction to be made. For example, there likely would be no difference between the EMG of a patient with motor neuron disease, such as amyotrophic lateral sclerosis (ALS), and that of a patient with malignant lymphoma diffusely infiltrating multiple nerve roots and cranial nerves. The F responses might be abnormal in polyradiculopathy, in contrast to motor neuron disease, but otherwise the studies could be identical (normal SNAPs, diffuse denervation and reinnervation). However, the clinical presentation and neurologic examination certainly would be markedly different. In motor neuron disease, there are no sensory signs or symptoms. In contrast, pain and paresthesias are prominent in polyradiculopathy. Deep tendon reflexes usually are depressed or absent in polyradiculopathy, whereas they are increased or present in ALS (although they may be depressed in the progressive muscular atrophy variant of ALS). These points once again underscore that EMG and nerve conduction studies can be properly interpreted only in the context of the clinical history and physical examination.

Fibrillation Potentials may Persist in the Paraspinal Muscles after Spinal Surgery

Patients with recurrent or persistent pain after disc surgery often are referred to the EMG laboratory. However, the interpretation of fibrillation potentials in the paraspinal muscles of such patients is not straightforward. Patients who have undergone successful disc surgery and no longer have symptoms or signs of radiculopathy have been demonstrated to have persistent fibrillation potentials in the paraspinal muscles, often for several years. It is not clear why this occurs, but it may be related to the surgical scar through the paraspinal muscles. For this reason, the paraspinal EMG examination no longer assumes the same diagnostic importance in postsurgical patients, and it is questionable whether sampling the paraspinals is worthwhile in such patients (i.e., the absence of denervation cannot exclude a radiculopathy, and the presence of denervation may be a "normal" finding many years after spinal surgery and is of no clinical significance).

Only the Distal Muscles may be Abnormal in Radiculopathy

The diagnosis of radiculopathy is based on demonstrating neuropathic changes in distal, proximal, and paraspinal muscles in a myotomal pattern. Reinnervation, like denervation, occurs in proximal before distal muscles. In addition, the more proximal the denervation, the more successful the reinnervation. Thus, if the proximal limb and paraspinal muscles have been successfully reinnervated, chronic radiculopathies may show denervation only in the distal muscles. Once this reinnervation has occurred in proximal muscles, it may not be possible to differentiate a radiculopathy from a plexopathy or distal neuropathy by the needle EMG examination alone.

There may be Few or no Electromyographic Abnormalities in Spinal Stenosis

Lumbosacral spinal stenosis is a common condition, especially in the elderly. Patients often develop neurogenic claudication (pain and paresthesias in the legs with standing and walking, relieved by sitting). This condition results from *intermittent* compression of the lumbosacral nerve roots. Because the symptoms are intermittent and occur only when the nerve roots are compromised in the upright posture, fixed EMG changes seldom occur. More often, the EMG findings in spinal stenosis, especially in mild to moderate cases, are normal or, at most, equivocal.

Fibrillation Potentials in the Paraspinal Muscles do not Necessarily Imply Radiculopathy

Too often, the presence of fibrillation potentials in the paraspinal muscles is automatically interpreted as evidence of radiculopathy. Although fibrillation potentials in the paraspinal muscles are an important finding in radiculopathy, they are frequently seen in other conditions as well. Most important among those conditions are (1) proximal myopathies with inflammatory or necrotic features (e.g., polymyositis), (2) disorders of the motor neurons (e.g., ALS, intrinsic spinal cord disease), (3) botulism, and (4) neuropathies affecting the dorsal rami (e.g., diabetic polyneuropathy). As in any electrophysiologic study, the final impression should never be based on a single finding but rather on a combination of all nerve conduction and EMG data in conjunction with the clinical information.

In addition, sparse fibrillation potentials and especially positive sharp waves are occasionally seen in the paraspinal muscles in normal individuals. Although this is rare in patients younger than 40 years of age, in one study of normal individuals, approximately 40% of older individuals had brief runs of fibrillation potentials or positive sharp waves in the lower lumbosacral paraspinal muscles. In this study, the denervating potentials were counted if they lasted longer than 0.5 seconds. Remember, traditionally a muscle is graded as +1 fibrillation potentials if you see fibrillation potentials and/or positive waves that last more than 3 seconds, at two or more locations. *The take home message is the following: increased insertional activity or a few brief runs of fibrillation potentials or positive sharp waves limited to the paraspinal muscles, especially in an older individual, may not have any clinical significance, and needs to be interpreted with caution.* Again, remember one of the cardinal rules of EMG: when in doubt, do not overcall a diagnosis. You do not want to make a type I error (i.e., make a diagnosis of an abnormality when one is not really present).

In the Elderly, it may not be Possible to Differentiate a Mild Chronic Distal Polyneuropathy from Mild Chronic Bilateral L5–S1 Radiculopathies

Normally, conduction velocities decrease slightly and sensory amplitudes fall with advancing age. In addition, both polyneuropathy and degenerative disc disease of the lumbosacral spine are common conditions in the elderly. Consider the following pattern in an elderly patient:

- Sural and superficial peroneal SNAPs are just at the lower limits of normal in amplitude
- Peroneal and tibial CMAP amplitudes are slightly reduced, with mildly slowed conduction velocities although still in the range of axonal loss
- Peroneal and tibial F responses and H reflexes are slightly prolonged
- Denervation/reinnervation changes are present in the distal leg muscles
- Nerve conduction study and EMG findings are normal in the upper extremities

If this pattern is present bilaterally in an elderly patient, a definite conclusion cannot be reached. There may be bilateral L5–S1 radiculopathies with successful reinnervation in the paraspinal and proximal muscles, and the borderline SNAPs may be related to advanced age. However, this pattern is also consistent with a distal polyneuropathy that is mild enough that it reduces only the lower extremity SNAPs into the borderline range and spares the upper extremities.

EXAMPLE CASES

Case 29–1

History and Physical Examination

A 50-year-old woman was referred for tingling in the middle and index fingers of the right hand for several months. She also reported associated diffuse aching of the arm for the past 3 to 4 weeks. Examination showed normal strength and deep tendon reflexes. Sensory examination showed a patchy area of decreased sensation over the finger pads of the second and third digits of the right hand. There was no Tinel's sign at the wrist, and a Phalen's maneuver did not increase symptoms.

Summary

The clinical presentation is pain in the arm associated with paresthesias of the middle and index fingers with no other localizing findings by history or physical examination. Because sensation to the index and middle fingers is mediated via the median nerve, brachial plexus, and C6–C7 nerve roots, the most likely diagnoses include CTS and cervical radiculopathy. Other less likely possibilities include a brachial plexus lesion or a high median neuropathy at either the pronator teres or the ligament of Struthers. Unfortunately, there are no other localizing findings to help sort out the clinical differential diagnosis. There is no history of neck pain or paraspinal muscle spasm suggesting radiculopathy and no local findings such as a Tinel's sign or positive Phalen's maneuver suggesting a median neuropathy at the wrist. The motor

CASE 29–1. Nerve Conduction Studies

Nerve Stimulated	Stimulation Site	Recording Site	Amplitude Motor = mV; Sensory = μV			Latency (ms)			Conduction Velocity (m/s)			F-wave Latency (ms)		
			RT	LT	NL	RT	LT	NL	RT	LT	NL	RT	LT	NL
Median (m)	Wrist	APB	8.0		≥4	3.6		≤4.4				24		≤31
	Antecubital fossa	APB	7.8			7.6			57		≥49			
Ulnar (m)	Wrist	ADM	9.8		≥6	3.2		≤3.3						≤32
	Below elbow	ADM	8.8			7.5			54		≥49	26		
	Above elbow	ADM	8.6			8.8			59		≥49			
Median (s)	Wrist	Index finger	57		≥20	3.2		≤3.5	62		≥50			
Ulnar (s)	Wrist	Little finger	48		≥17	2.9		≤3.1	58		≥50			
Radial (s)	Forearm	Snuffbox	37		≥15	2.2		≤2.9	62		≥50			
Median (mixed study)	Palm	Wrist	126		≥50	1.7		≤2.2	58		≥50			
Ulnar (mixed study)	Palm	Wrist	38		≥12	1.8		≤2.2	56		≥50			
Mixed difference						–0.1		≤0.3						

m = motor study; s = sensory study; RT = right; LT = left; NL = normal; APB = abductor pollicis brevis; ADM = abductor digiti minimi.

Note: All sensory and mixed latencies are peak latencies. All sensory and mixed nerve conduction velocities are calculated using onset latencies. The reported F-wave latency represents the minimum F-wave latency.

CASE 29–1. Electromyography

Muscle	Insertional Activity	Spontaneous Activity		Voluntary Motor Unit Action Potentials				
		Fibrillation Potentials	Fasciculations	Activation	Recruitment	Configuration		
						Duration	Amplitude	Polyphasia
Right biceps brachii	NL	0	0	NL	NL	NL	NL	NL
Right pronator teres	↑	+2	0	NL	↓	NL	NL	NL
Right triceps brachii	↑	+1	0	NL	↓	+1	NL	NL
Right extensor digitorum communis	↑	+1	0	NL	NL	NL/+1	NL	NL
Right medial deltoid	NL	0	0	NL	NL	NL	NL	NL
Right first dorsal interosseous	NL	0	0	NL	NL	NL	NL	NL
Right abductor pollicis brevis	NL	0	0	NL	NL	NL	NL	NL
Right extensor indicis proprius	NL	0	0	NL	NL	NL	NL	NL
Right flexor carpi ulnaris	NL	0	0	NL	NL	NL	NL	NL
Right C7 paraspinal	↑	0	0	NL	NL	NL	NL	NL
Right C6 paraspinal	↑	+1	0	NL	NL	+1	NL	+1

↑ = increased; ↓ = slightly reduced; NL = normal.

examination is normal, so there is no pattern of weakness suggesting either a median nerve or cervical root problem.

This situation of diffuse non-localizing pain associated with paresthesias is commonly seen in the outpatient setting. It is in such cases that the electrophysiologic examination often plays a key role in localizing the lesion. Before proceeding to the nerve conduction and EMG study, we must consider all of the diagnoses mentioned previously. Of greatest interest will be the SNAPs in the index and middle fingers. If either of these SNAPs is reduced, the lesion must be at or distal to the dorsal root ganglion, either in the median nerve or brachial plexus. Conversely, normal SNAPs in these two fingers suggest a more proximal lesion at the root level.

Continuing on to the nerve conduction studies, the median and ulnar motor studies are normal, including the F responses. Likewise, the median and ulnar sensory potentials are normal and very robust. When potentials are borderline or just slightly above the upper limit of normal, it is important to compare them with the contralateral side to look for a significant asymmetry. In this case, there is no need to do so because the median SNAP amplitude is 57 μV in the index finger on the involved side. With such a high normal amplitude on the involved side, we are very unlikely to find the amplitude on the other side to be twice as high, the level required for a significant asymmetry. The radial nerve sensory study also is performed and is normal.

Finally, the median and ulnar palm-to-wrist mixed nerve studies are performed, comparing their two respective latencies. Both are normal with no significant difference in latency. The median and ulnar palmar mixed nerve studies are essential to exclude the possibility of a median neuropathy at the wrist. In fact, approximately 10 to 20% of patients with median neuropathy at the wrist will go undiagnosed in the EMG laboratory if only the median motor and sensory studies are performed without an additional median-versus-ulnar comparison study, such as the palmar mixed nerve studies.

Moving next to the EMG study, particular attention is focused on muscles innervated by the C6 and C7 nerve roots because the patient's paresthesias are in that distribution. Fibrillation potentials and decreased recruitment of MUAPs are found in the pronator teres, triceps brachii, and extensor digitorum communis muscles. In addition, the MUAPs in the triceps brachii and extensor digitorum communis muscles are slightly reinnervated. Muscles innervated by the C5–C6 nerve roots (biceps brachii, medial deltoid) are normal, as are muscles innervated by the C8–T1 nerve roots (abductor pollicis brevis, first dorsal interosseous). Finally, the C6 and C7 paraspinal muscles are sampled, which show increased insertional activity and fibrillation potentials at the C6 paraspinal level.

At this point, we are ready to formulate our electrophysiologic impression.

IMPRESSION: *The electrophysiologic findings are consistent with a right C7 radiculopathy.*

Despite the non-localizing clinical symptoms, the EMG study clearly demonstrates that the lesion is at the cervical root level. A median nerve lesion (at the carpal tunnel or proximally) is essentially excluded by the combination of normal median motor, sensory, and palmar mixed nerve studies. Despite the presence of a cervical radiculopathy, the F responses are normal. This is not surprising because the median and ulnar F responses travel via the C8–T1 nerve roots. Any radiculopathy affecting the C5, C6, or C7 nerve roots will always result in normal median and ulnar F waves.

Several important questions can be addressed at this point.

Can the Lesion be Localized to an Isolated Nerve Root?

The needle EMG study shows clear neuropathic abnormalities in the triceps brachii, extensor digitorum communis, and pronator teres. The abnormalities are in the distribution of two different nerves: the radial and median. Thus, the EMG abnormalities cannot be explained by an isolated peripheral nerve lesion. The normal SNAPs also suggest that the lesion is proximal to the dorsal root ganglion. The best way to assess the segmental localization is to analyze the pattern of abnormalities in the limb muscles. Why is the C7 nerve root the most likely? First, the three abnormal muscles (pronator teres, triceps brachii, extensor digitorum communis) all have C7 nerve root innervation in common, although they also each receive some C6 innervation, and the latter two receive some C8 innervation. How does one exclude a C6 or C8 radiculopathy? Muscles innervated by the C6 nerve root but without any C7 innervation (i.e., biceps brachii, medial deltoid) are normal. Similarly, muscles innervated by the C8 nerve root but without any C7 innervation (i.e., abductor pollicis brevis, first dorsal interosseous) are normal. To summarize, although the abnormal muscles have the C6 and C7 nerve roots in common, other muscles with C6 or C8 innervation, but without a C7 contribution, are normal. Therefore, a C6 radiculopathy is less likely, given the normal findings in other C6-innervated muscles. A C8 radiculopathy is unlikely for similar reasons and also would not explain the abnormalities in the pronator teres, which has no C8 innervation. Therefore, determination of the segmental level of a radiculopathy relies on the pattern of muscles that are abnormal as well as those that are normal.

How are the Paraspinal Muscles Helpful in the Diagnosis of Radiculopathy?

In the case discussed here, the cervical paraspinal muscles show clear neuropathic changes. The paraspinal muscles are especially important to examine because abnormalities there imply a lesion at or proximal to the roots. Note that the fibrillation potentials in this case were more prominent at the C6 paraspinal level, yet the electrophysiologic impression was that of a C7 radiculopathy. Because of the wide overlap in innervation of the paraspinal muscles, the level of the radiculopathy should not be based on findings in the paraspinal muscles. Abnormalities in the paraspinal muscles are best used to indicate that the lesion is at or proximal to the roots, whereas the pattern of abnormalities in the limb muscles is used to identify the segmental level.

Case 29–2

History and Physical Examination

A 36-year-old man was referred to the EMG laboratory for evaluation of a possible lower extremity radiculopathy. Eight weeks ago, he had bent down to lift a chair and developed acute pain in the right back and buttock with radiating paresthesias into the calf and lateral foot. His past medical history was notable for several episodes of low back pain over the last several years.

Neurologic examination demonstrated normal muscle bulk and tone in the lower extremities. Straight-leg raising elicited pain and paresthesias into the right leg at 45 degrees. Muscle strength was normal in the left leg. Strength testing in muscles around the right hip was difficult because of pain. In the distal right leg, ankle dorsiflexion appeared normal and plantar flexion appeared to be slightly weak. When asked to stand on tiptoe, the patient was able to do so on the left side but not on the right. Sensory examination demonstrated a subtle sensory loss on the right sole and lateral foot. All deep tendon reflexes were normal and symmetric except for the right ankle reflex, which was absent.

Summary

In contrast to the previous case, the history and examination here both are quite suggestive of a radiculopathy. The several-year history of low back pain, with a recent exacerbation consisting of radiating pain and paresthesias into the calf and lateral foot, is very characteristic of a lumbosacral radiculopathy due to disc disease. On examination, there are clear mechanical signs: straight-leg raise elicits pain and paresthesias in the right leg at 45 degrees. When a nerve root is entrapped by spondylosis or disc herniation, symptoms are often provoked when the nerve is stretched during a straight-leg raise maneuver.

On examination, there is difficulty assessing proximal strength because of pain. This is not an uncommon situation in patients with radiculopathy or other painful conditions. However, when distal muscle strength is tested using maneuvers that do not disturb the proximal hip girdle, there is a suggestion of ankle plantar flexion weakness. That weakness is brought out when the patient is asked to stand on tiptoe. He can do so on the left but not the right, suggesting weakness of the gastroc–soleus muscle. In addition, there is evidence of a subtle sensory loss along the right sole and lateral foot. This sensory disturbance is in the distribution of the S1 dermatome,

CASE 29–2. Nerve conduction studies

Nerve Stimulated	Stimulation Site	Recording Site	Amplitude Motor = mV; Sensory = μV			Latency (ms)			Conduction Velocity (m/s)			F-wave latency (ms)		
			RT	LT	NL	RT	LT	NL	RT	LT	NL	RT	LT	NL
Tibial (m)	Ankle	AHB	3.2	5.3	≥4	5.3	4.8	≤5.8				58	52	≤56
	Popliteal fossa	AHB	2.8	4.8		13.6	13.0		40	46	≥41			
Peroneal (m)	Ankle	EDB	4.4	4.8	≥2	5.8	5.7	≤6.5				52	47	≤56
	Below fibula	EDB	4.0	4.8		10.6	10.4		45	46	≥44			
	Lateral popliteal fossa	EDB	3.9	4.6		13.5	13.3		47	48	≥44			
Sural (s)	Calf	Posterior ankle	13	12	≥6	3.7	3.6	≤4.4	48	47	≥40			
Peroneal (s)	Lateral calf	Lateral ankle	11	10	≥6	3.6	3.8	≤4.4	49	47	≥40			
H reflex	Popliteal fossa	Soleus				NR	32	≤34						

m = motor study; s = sensory study; RT = right; LT = left; NL = normal; NR = no response; AHB = abductor hallucis brevis; EDB = extensor digitorum brevis.
Note: All sensory latencies are peak latencies. All sensory conduction velocities are calculated using onset latencies. The reported F-wave latency represents the minimum F-wave latency.

CASE 29–2. Electromyography

Muscle	Insertional Activity	Spontaneous Activity		Voluntary Motor Unit Action Potentials				
		Fibrillation Potentials	Fasciculations	Activation	Recruitment	Configuration		
						Duration	Amplitude	Polyphasia
Right tibialis anterior	NL	0	0	NL	NL	NL	NL	NL
Right medial gastrocnemius	↑	+3	0	NL	↓↓	NL	NL	NL
Right extensor hallucis longus	↑	+1	0	NL	↓	NL	NL	NL
Right peroneus longus	↑	+1	0	NL	↓	NL	NL	NL
Right biceps femoris	↑	+1	0	NL	↓	NL	NL	NL
Right gluteus maximus	↑	+2	0	NL	↓	NL	NL	NL
Right gluteus medius	↑	+1	0	NL	NL	NL	NL	NL
Right tensor fascia latae	↑	0	0	NL	NL	NL	NL	NL
Right vastus lateralis	NL	0	0	NL	NL	NL	NL	NL
Right iliacus	NL	0	0	NL	NL	NL	NL	NL
Right S1 paraspinal	NL	0	0	NL	NL	NL	NL	NL
Right L5 paraspinal	NL	0	0	NL	NL	NL	NL	NL
Right L4 paraspinal	NL	0	0	NL	NL	NL	NL	NL
Right L3 paraspinal	NL	0	0	NL	NL	NL	NL	NL

↑= increased; ↓= slightly reduced; ↓↓ = moderately reduced; NL = normal.

as well as the distribution of the sciatic, sural, and plantar nerves. Sensory loss secondary to radiculopathy usually is subtle or vague because there is so much overlap between adjacent dermatomes. Finally, all of the deep tendon reflexes are normal, with the notable exception of an absent right ankle jerk. The ankle jerk is mediated via the tibial nerve, sciatic nerve, lumbosacral plexus, and S1 nerve root.

Altogether, there are abnormalities in muscle strength (ankle plantar flexion), sensation (lateral foot and sole), and deep tendon reflexes, along with back pain and radiating paresthesias. Clinically, the most likely diagnosis is

an S1 radiculopathy. The only other possible diagnosis is a lesion of the proximal sciatic nerve or the lower lumbosacral plexus, although back pain would not typically be associated with either of these. Finally, before proceeding to the nerve conduction and EMG studies, we must remember that the patient's acute pain developed 8 weeks ago.

When the tibial and peroneal motor conduction studies are performed, the tibial motor amplitudes on the involved right side are found to be low. For this reason, the amplitudes are compared with the contralateral, asymptomatic side. A clear asymmetry is found in the tibial motor studies but not in the peroneal. Along with the relatively decreased tibial CMAP amplitudes, there is slight slowing of conduction velocity and slight prolongation of the F responses. These findings are consistent with mild axonal loss in the tibial motor fibers. The localization of the axonal loss is not clear, however, and at this point could be due to a lesion in the tibial nerve, sciatic nerve, lumbosacral plexus, or lumbosacral nerve roots. Moving next to the sural and superficial peroneal sensory studies, the amplitudes are normal, and there are no significant asymmetries compared with the contralateral side. The sural response is especially important because that is the distribution where the patient has sensory symptoms and subtle sensory loss. The normal sural response after an 8-week course strongly suggests that the lesion is proximal to the dorsal root ganglion.

Finally, because an S1 radiculopathy is a strong possibility, H reflex studies are performed. The latency is normal on the asymptomatic side, but the response is absent on the involved side. The absent H reflex correlates with the clinically absent ankle jerk. If the ankle jerk is present clinically, the H response must be present. If the ankle jerk is absent clinically, however, an H reflex may occasionally be present. It is important to compare the latency of the H reflex on the asymptomatic side with that on the symptomatic side, if present. Any prolongation of the H reflex latency suggests a proximal lesion, provided the distal tibial conductions are normal. Thus, after the nerve conduction studies are completed, there is evidence of axonal loss affecting the tibial motor fibers, with a normal sural sensory response and an absent H reflex. The normal sural SNAP suggests that the lesion is proximal to the dorsal root ganglion.

Moving next to the EMG study, distal and proximal muscles innervated by different nerve roots are sampled in the leg. Fibrillation potentials with decreased recruitment of MUAPs are found in several muscles, including the medial gastrocnemius, extensor hallucis longus, peroneus longus, biceps femoris, gluteus maximus, and gluteus medius. These findings are most prominent in the medial gastrocnemius. In addition, the MUAP morphology in these muscles is normal. Several muscles studied are completely normal, including the tibialis anterior, vastus lateralis, and iliacus. Finally, the lumbosacral paraspinal muscles from the L3 to S1 level also are normal.

At this point, we are ready to formulate our electrophysiologic impression.

IMPRESSION: *The electrophysiologic findings are most consistent with a subacute right S1 radiculopathy. Although much less likely, this study cannot completely exclude a lower lumbosacral plexopathy.*

Several important questions can be addressed at this point.

How is the Level of the Radiculopathy Localized to S1?

The nerve conduction and EMG findings in this case are classic for an S1 radiculopathy, with the notable exception of the normal paraspinal muscles. In radiculopathy associated with significant axonal loss, an axonal loss pattern of abnormalities may be seen in the distal motor nerve conduction studies. If an L5 or S1 radiculopathy is associated with axonal loss, signs of axonal loss may be seen in the peroneal and tibial nerve conduction studies, respectively. In the case discussed here, evidence of axonal loss was present in the right tibial motor conduction study. Because the lesion in radiculopathy is proximal to the dorsal root ganglion, the SNAP remains normal even though the patient has sensory complaints in that distribution. The EMG study in this case showed prominent fibrillation potentials in muscles innervated by the L5 and S1 myotomes. The normal tibialis anterior (L4–L5) makes an L5 radiculopathy less likely. In addition, the medial gastrocnemius (S1–S2), which is the most abnormal muscle, has no L5 innervation. Muscles with S1 innervation, both distally and proximally, are abnormal. Thus, there is no distal-to-proximal gradient, as might occur in a polyneuropathy. In addition, because the SNAPs are normal, a neuropathy or plexopathy is less likely. In this context, the absent H reflex also points toward an S1 radiculopathy.

Why are the Paraspinal Muscles Normal if this is a Radiculopathy?

On EMG, although there are fibrillation potentials in several muscles, the MUAP morphology is completely normal. This implies that enough time has passed in the distal muscles for denervation to occur but not enough time for reinnervation. The only part of the study that is surprising for a radiculopathy is the absence of denervation in the paraspinal muscles. In some cases of radiculopathy, abnormalities are not seen in the paraspinal muscles; this can occur for several reasons. First, not all fascicles are equally affected in radiculopathy. It would be ideal if in a radiculopathy all of the muscles in the involved myotome were equally affected, but often that is not the case. In some cases, the fascicles to the dorsal rami are normal, thus sparing the paraspinal muscles. Second, there may be some sampling error in the examination of any muscle. Third and most important, reinnervation occurs first in the paraspinal muscles, and is usually most successful there. If denervation is mild and

reinnervation is successful, the changes in MUAP morphology may be very mild and difficult or impossible to detect.

Can a Lumbosacral Plexopathy be Excluded?

The EMG abnormalities alone are equally consistent with a lesion of the lower lumbosacral plexus. The only strong evidence for radiculopathy and against plexopathy is preservation of the SNAPs. Because the patient has sensory symptoms in the distribution of a normal SNAP (i.e., the sural SNAP), and the lesion is subacute with axonal loss and enough time has passed for wallerian degeneration to have taken place, it is much more likely that the abnormalities represent a lesion of the nerve root and not of the plexus. Because of the absence of abnormalities in the paraspinal muscles, however, it is important to state in the impression that a lumbosacral plexopathy, although less likely, cannot be completely excluded based on this study, although the normal SNAPs argue against this possibility.

Suggested Readings

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