

Radial Neuropathy

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In the electromyography (EMG) laboratory, the radial nerve is studied less frequently than the median and ulnar nerves and their respective well-known lesions. Nevertheless, entrapment of the radial nerve does occur, often affecting the main radial nerve either in the upper arm or axilla. Isolated lesions of its terminal divisions in the forearm, the posterior interosseous and superficial radial sensory nerves, also occur. Although radial motor nerve conduction studies are technically demanding, the electrophysiologic evaluation of radial neuropathy usually is able to localize the lesion, assess the underlying pathophysiology, and provide useful information regarding severity and subsequent prognosis.

ANATOMY

The radial nerve receives innervation from all three trunks of the brachial plexus and, correspondingly, a contribution from each of the C5–T1 nerve roots (Figures 21–1 and 21–2). After each trunk divides into an anterior and posterior division, the posterior divisions from all three trunks unite to form the posterior cord. The *posterior cord* gives off the *axillary*, *thoracodorsal*, and *subscapular nerves* before becoming the radial nerve. In the high arm, the radial nerve first gives off the *posterior cutaneous nerve of the arm*, the *lower lateral cutaneous nerve of the arm*, and the *posterior cutaneous nerve of the forearm* (Figure 21–3), followed by muscular branches to the three heads of the triceps brachii (medial, long, and lateral) and the anconeus. The anconeus is a small muscle in the proximal forearm that effectively is an extension of the medial head of the triceps brachii. After giving off these muscular branches, the radial nerve wraps around the posterior humerus in the *spiral groove*. Descending into the region of the elbow, muscular branches are then given off to the brachioradialis and the long head of the extensor carpi radialis. Next, three to four cm distal to the lateral epicondyle, the radial nerve bifurcates into two separate nerves: one superficial and the other deep. The superficial branch, known as the *superficial radial sensory nerve*, descends distally into the forearm over the radial bone to supply sensation over the lateral dorsum of the hand as well as part of the thumb and the dorsal proximal phalanges of the index, middle, and ring fingers (Figure 21–4). Distally, the nerve is quite superficial, running over the extensor tendons to the thumb, where it can easily be palpated (Figure 21–5).

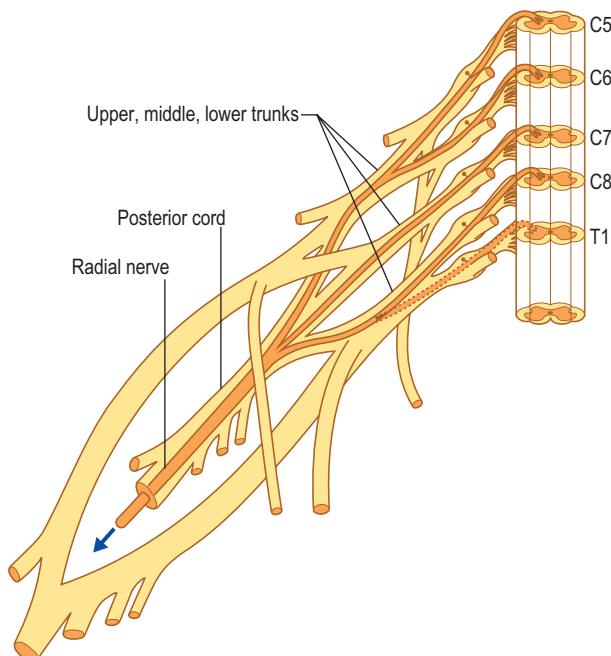


FIGURE 21–1 Anatomy of the radial nerve. The radial nerve receives innervation from all three trunks of the brachial plexus and, correspondingly, a contribution from each of the C5–T1 nerve roots. (Adapted with permission from Haymaker, W., Woodhall, B., 1953. Peripheral nerve injuries. WB Saunders, Philadelphia.)

The deep branch, known as the *deep radial motor branch*, first supplies the extensor carpi radialis brevis and the supinator muscles before it enters the supinator muscle under the Arcade of Frohse (Figure 21–6). The Arcade of Frohse is the proximal border of the supinator and in some individuals is quite tendinous. After the nerve enters the supinator, it is known as the *posterior interosseous nerve*, which then supplies the remaining extensors of the wrist, thumb, and fingers (extensor digitorum communis, extensor carpi ulnaris, abductor pollicis longus, extensor indicis proprius [EIP], extensor pollicis longus, and extensor pollicis brevis). Although the posterior interosseous nerve is thought of as a pure motor nerve (supplying no cutaneous sensation), it does contain sensory fibers that supply deep sensation to the interosseous membrane and joints between the radial and ulna bones.

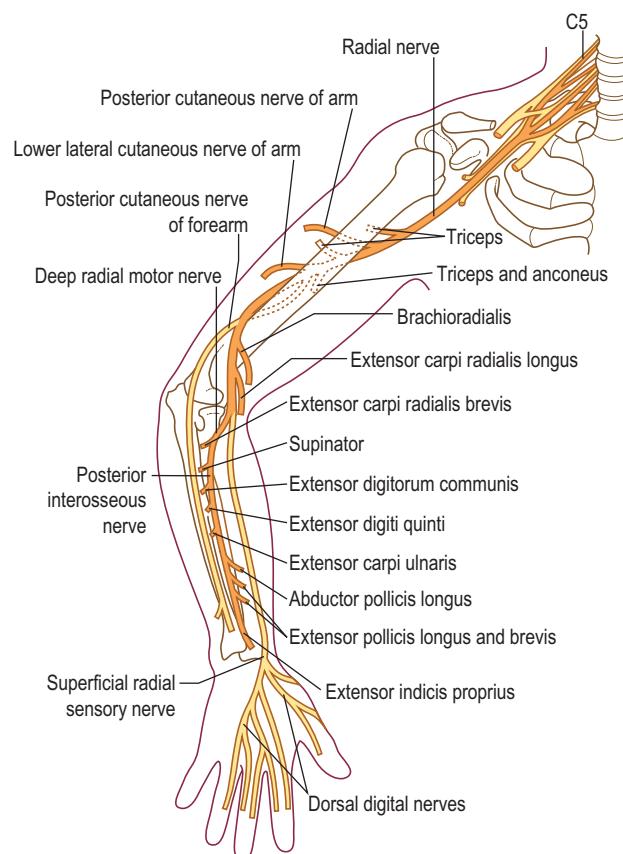


FIGURE 21–2 Anatomy of the radial nerve. The radial nerve is derived from the posterior cord of the brachial plexus. In the high arm, the radial nerve first gives off the posterior cutaneous nerve of the arm, the lower lateral cutaneous nerve of the arm, and the posterior cutaneous nerve of the forearm, followed by muscular branches to the triceps brachii and anconeus. The radial nerve then wraps around the humerus, descending into the region of the elbow, where muscular branches are given to the brachioradialis and long head of the extensor carpi radialis. The nerve then bifurcates into the superficial radial sensory and deep motor branch of the radial nerve. The deep motor branch supplies the extensor carpi radialis brevis (in most cases) and the supinator muscle before continuing on as the posterior interosseous nerve. The posterior interosseous nerve supplies the remainder of the wrist and finger extensors, as well as the abductor pollicis longus.

(Adapted with permission from Haymaker, W., Woodhall, B., 1953. Peripheral nerve injuries. WB Saunders, Philadelphia.)

Nomenclature of the Branches of the Radial Nerve near the Elbow

One of the more confusing aspects of radial nerve anatomy is the inconsistency regarding the nomenclature of the branches of the radial nerve near the elbow used in various anatomic texts and clinical reports (Figure 21–7). The following points should help the electromyographer when dealing with potential lesions of the radial nerve in this area:

Radial Nerve between the Spiral Groove and the Bifurcation near the Elbow

- Distal to the spiral groove but before the elbow, the main radial nerve always supplies two muscles: the

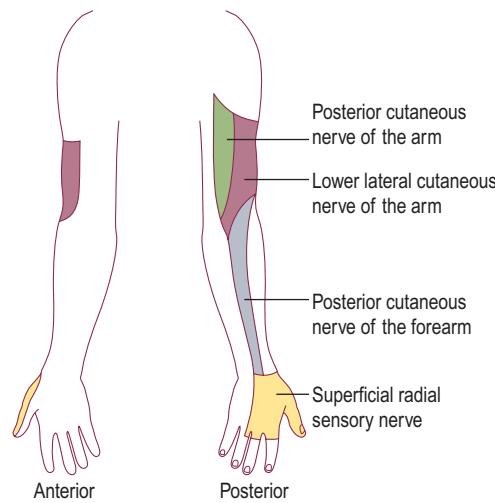


FIGURE 21–3 Sensory territories supplied by the radial nerve. (Adapted with permission from Haymaker, W., Woodhall, B., 1953. Peripheral nerve injuries. WB Saunders, Philadelphia.)

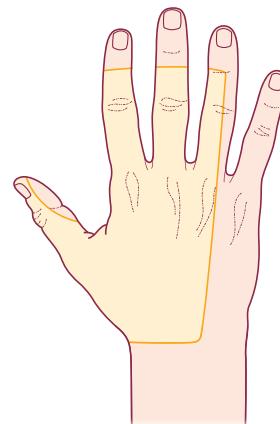


FIGURE 21–4 Sensory territory of the superficial radial sensory nerve. The superficial radial sensory nerve supplies sensation over the lateral dorsum of the hand, as well as part of the thumb and dorsal proximal phalanges of the index, middle, and ring fingers.

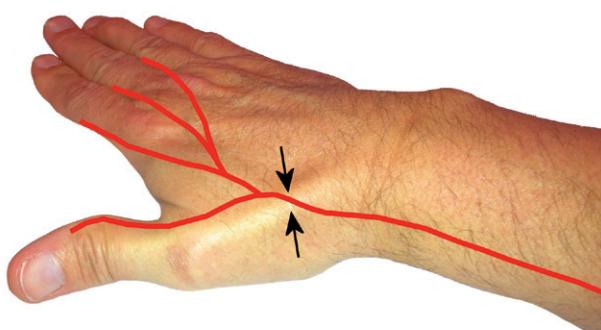


FIGURE 21–5 Superficial radial sensory nerve. The superficial radial nerve runs distally in the forearm over the radial bone to supply sensation over the lateral dorsum of the hand as well as part of the thumb and dorsal proximal phalanges of the index, middle, and ring fingers. It runs over the extensor tendons to the thumb (arrows), where it can easily be palpated.

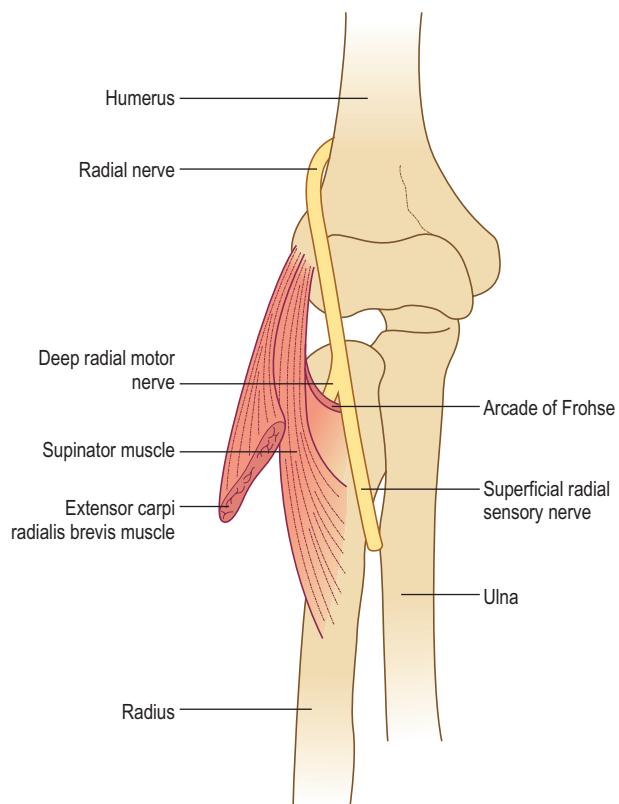


FIGURE 21–6 Anatomy of the radial nerve at the elbow. Distal to the elbow, the radial nerve bifurcates into the superficial radial sensory and deep radial motor branch. The deep radial motor branch enters the supinator muscle under the Arcade of Frohse where it is then known as the posterior interosseous nerve which supplies the remaining extensors of the wrist, thumb, and fingers.

(Adapted with permission from Wilbourn, A.J., 1992. Electrodiagnosis with entrapment neuropathies. AAEM plenary session I: entrapment neuropathies. Charleston, South Carolina.)

brachioradialis and the extensor carpi radialis longus (also known as the long head of the extensor carpi radialis).

- In some individuals, the main radial nerve will also supply a third muscle, the extensor carpi radialis brevis muscle*.

The Bifurcation near the Elbow

- The main radial nerve always bifurcates into superficial and deep branches just distal to the elbow.

Superficial Branch

- The superficial branch continues as a pure cutaneous sensory branch (the *superficial radial sensory branch*).
- However, in a small number of individuals, there is an anatomic variation wherein the superficial branch near its origin will supply one muscle, the extensor carpi radialis brevis*.

*Thus, the innervation to the extensor carpi radialis brevis has several normal variations: from the main radial nerve, the superficial radial nerve, and the deep radial motor branch of the radial nerve.

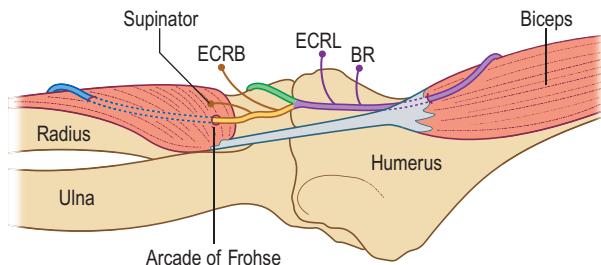


FIGURE 21–7 Anatomy and nomenclature of the radial nerve around the elbow. As the main radial nerve enters the region of the elbow (purple), it supplies the brachioradialis (BR) and extensor carpi radialis longus (ECRL) muscles. It then divides into a *superficial radial sensory branch* (green) and a *deep radial motor branch* (yellow). The deep radial motor branch typically innervates the extensor carpi radialis brevis (ECRB) and supinator muscles before entering into the substance of the supinator muscle at the Arcade of Frohse. Past the Arcade of Frohse, the continuation of the deep radial motor branch is known as the *posterior interosseous nerve* (blue). However, please note that some anatomic texts define the posterior interosseous nerve as originating at the bifurcation of the main radial nerve, and thus use the terms *deep radial motor branch* and *posterior interosseous nerve* interchangeably. If this definition is used, then both the ECRB and the supinator muscle would both be supplied by the posterior interosseous nerve.

(Adapted with permission from Thomas, S.J., Yakin, D.E., Parry, B.R., et al., 2000. The anatomical relationship between the posterior interosseous nerve and the supinator muscle. J Hand Surg Am 25 (5), 936–941.)

Deep Branch

- The *deep radial motor branch* first supplies the extensor carpi radialis brevis muscle in some individuals*.
- It then supplies one or more branches to the supinator muscle before entering the supinator muscle proper.
- The *deep radial motor branch* then runs under the Arcade of Frohse (the proximal border of the supinator) and through the supinator muscle.
- After leaving the supinator muscle, branches are given off that supply the extensor muscles to the thumb and fingers as well as the abductor pollicis longus and extensor carpi ulnaris. The inconsistency in the nomenclature regarding these nerve branches involves where the posterior interosseous nerve begins, and whether the posterior interosseous nerve and the deep radial motor branch are one and the same nerve:
 - In some textbooks and many clinical reports, the entire *deep radial motor branch* is known as the *posterior interosseous nerve*, with the two names used interchangeably. Thus, using this anatomic definition, a complete posterior interosseous neuropathy would include the supinator and the extensor carpi radialis brevis muscles, as well as the extensors to the thumb and fingers, and the abductor pollicis longus and extensor carpi ulnaris.
 - In most anatomic texts, however, only the segment of the deep branch between the bifurcation of the main radial nerve at the elbow to

where the nerve enters the supinator muscle at the Arcade of Frohse is known as the *deep radial motor branch*. The *posterior interosseous nerve* is then the continuation of the deep radial motor branch *after it enters the supinator*. In the remainder of this text, we will use this latter anatomic definition. Thus, with this anatomic definition, a complete posterior interosseous neuropathy would spare the supinator and the extensor carpi radialis brevis muscles. As the most common entrapment site of the posterior interosseous nerve is at the Arcade of Frohse, the use of this anatomic convention fits the common clinical syndromes most appropriately as well.

CLINICAL

Radial neuropathies can be divided into those caused by lesions at the spiral groove, lesions in the axilla, and isolated lesions of the posterior interosseous and superficial radial sensory nerves. These lesions usually can be differentiated by clinical findings.

Radial Neuropathy at the Spiral Groove

The most common radial neuropathy occurs at the spiral groove. Here, the nerve lies juxtaposed to the humerus and is quite susceptible to compression, especially following prolonged immobilization (Figure 21–8). One of the times this characteristically occurs is when a person has draped an arm over a chair or bench during a deep sleep or while intoxicated ('Saturday night palsy'). The subsequent prolonged immobility results in compression and demyelination of the radial nerve. Other cases may occur after strenuous muscular effort, fracture of the humerus, or

infarction from vasculitis. Clinically, marked wrist drop and finger drop develop (due to weakness of the EIP, extensor digitorum communis, extensor carpi ulnaris, and long head of the extensor carpi radialis), along with mild weakness of supination (due to weakness of the supinator muscle) and elbow flexion (due to weakness of the brachioradialis). Notably, elbow extension (triceps brachii) is spared. Sensory disturbance is present in the distribution of the superficial radial sensory nerve, consisting of altered sensation over the lateral dorsum of the hand, part of the thumb, and the dorsal proximal phalanges of the index, middle, and ring fingers.

In isolated radial neuropathy at the spiral groove, median- and ulnar-innervated muscles are normal. However, tested in a wrist drop and finger drop posture, finger abduction may appear weak, giving the mistaken impression of ulnar nerve dysfunction. To prevent this error, one should test the patient's finger abduction (ulnar-innervated function) with the fingers and wrist passively extended to a neutral wrist position. This often can be accomplished by placing the hand on a flat surface.

Radial Neuropathy in the Axilla

Radial neuropathy may occur in the axilla from prolonged compression. For instance, this is often seen in patients on crutches who use them inappropriately, applying prolonged pressure to the axilla. The clinical deficit is similar to that seen in radial neuropathy at the spiral groove, with the notable exception of additional weakness of arm extension (triceps brachii) and sensory disturbance extending into the posterior forearm and arm (posterior cutaneous nerves of the forearm and arm). Radial neuropathy in the axilla is differentiated from even more proximal posterior cord lesions by normal strength of the deltoid (axillary nerve) and latissimus dorsi (thoracodorsal nerve).

Posterior Interosseous Neuropathy

Posterior interosseous neuropathy (PIN) clinically resembles entrapment of the radial nerve at the spiral groove at first glance. In both conditions, patients present with wrist drop and finger drop with sparing of elbow extension. However, with closer inspection, several important differences easily separate the two. In PIN, there is sparing of radial-innervated muscles above the takeoff of the posterior interosseous nerve (i.e., brachioradialis, long and short heads of the extensor carpi radialis, triceps). Thus, a patient with PIN still may be able to extend the wrist, but weakly, with a radial deviation. This is due to the relative preservation of the extensor carpi radialis longus and brevis that arise proximal to the posterior interosseous nerve, with a weak extensor carpi ulnaris. In addition, of course, are the sensory findings. In PIN, there is no cutaneous sensory loss. However, there may be pain in the forearm from involvement of the deep sensory fibers of the posterior interosseous nerve that supply the interosseous membrane and joint capsules.

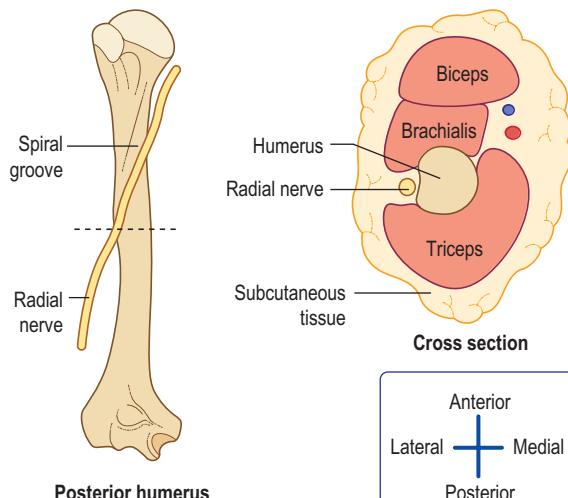


FIGURE 21–8 Radial nerve and the spiral groove. The most common radial neuropathy occurs at the spiral groove on the posterior side of the humerus. Here, the nerve lies juxtaposed to bone and is susceptible to external compression.

PIN usually occurs as an entrapment neuropathy under the tendinous Arcade of Frohse. Rarely, other mass lesions (e.g., ganglion cysts, tumors) result in PIN.

Radial Tunnel Syndrome

In radial tunnel syndrome, patients are reported to have isolated pain and tenderness in the extensor forearm, not unlike persistent tennis elbow, thought to result from compression of the posterior interosseous nerve near its origin. However, this is one of the more controversial and disputed nerve entrapment syndromes. As opposed to patients with a true posterior interosseous neuropathy (see above), these patients typically have no objective neurologic signs on examination, and accordingly have normal EDX studies. They are said to have increased pain with maneuvers that contract the extensor carpi radialis or the supinator (e.g., resisted extension of the middle finger or resisted supination, respectively). However, there is no compelling evidence that this chronic pain syndrome is caused by any nerve entrapment. Nevertheless, this syndrome is important to know of, as it is not unusual for a patient to be referred to the EMG laboratory for evaluation of “radial tunnel syndrome.” In such cases, the focus of the EDX is to look for any objective evidence of a posterior interosseous neuropathy, although in the absence of any weakness or other neurological signs, the EDX study is almost always normal.

Superficial Radial Sensory Neuropathy

The superficial radial sensory nerve is derived from the main radial nerve in the region of the elbow. In the distal third of the forearm, it runs subcutaneously next to the radius. Its superficial location next to bone makes it extremely susceptible to compression, a syndrome coined “Cheiralgia Paresthetica” which translates from the Greek

as *cheir* + *algos*, meaning pain in the hand. Tight-fitting bands, watches, or bracelets may result in compression of the superficial radial nerve. Handcuffs, especially when excessively tight, also characteristically result in a superficial radial neuropathy. Because the superficial radial sensory nerve is purely sensory, no weakness develops. A characteristic patch of altered sensation develops over the lateral dorsum of the hand, part of the thumb, and the dorsal proximal phalanges of the index, middle, and ring fingers.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of wrist drop, aside from a radial neuropathy at the spiral groove, axilla, and PIN, includes unusual presentations of C7–C8 radiculopathy, brachial plexus lesions, and central causes (Box 21–1). Because most muscles that extend the wrist and fingers are innervated by the C7 nerve root, C7 radiculopathy may rarely present solely with a wrist drop and finger drop, with relative sparing of non-radial C7-innervated muscles. However, several key clinical features help differentiate a C7 radiculopathy from a radial neuropathy, PIN, brachial plexopathy, or central lesion (Table 21–1). Radial neuropathy at the

Box 21–1. Wrist Drop: Possible Anatomic Localizations

- Posterior interosseous nerve
- Radial nerve at the spiral groove
- Radial nerve in the axilla
- Posterior cord of the brachial plexus
- C7 root
- Central nervous system

Table 21–1. Clinical Differentiating Factors in Wrist Drop

	Posterior Interosseous Neuropathy	Radial Nerve: Spiral Groove	Radial nerve: Axilla	Posterior Cord	C7
Wrist drop or finger drop	X	X	X	X	X
Radial deviation on wrist extension	X				
Weakness of supination (mild)		X	X	X	
Weakness of elbow flexion (mild)		X	X	X	
Diminished brachioradialis tendon reflex		X	X	X	
Weakness of elbow extension			X	X	X
Diminished triceps tendon reflex			X	X	X
Weakness of shoulder abduction				X	
Sensory loss in lateral dorsal hand		X	X	X	X (equivocal)
Sensory loss in posterior arm or forearm			X	X	X (equivocal)
Weakness of wrist flexion					X

X, may be present.

Table 21–2. Electromyographic and Nerve Conduction Abnormalities Localizing the Lesion Site in Wrist Drop

	Posterior Interosseous Neuropathy	Radial Nerve: Spiral Groove	Radial Nerve: Axilla	Posterior Cord	C7
EMG Findings					
Extensor indicis proprius	X	X	X	X	X
Extensor digitorum communis	X	X	X	X	X
Extensor carpi ulnaris	X	X	X	X	X
Extensor carpi radialis-long head		X	X	X	X
Brachioradialis		X	X	X	
Supinator		X	X	X	
Anconeus			X	X	X
Triceps			X	X	X
Deltoid				X	
Latissimus dorsi				X	X
Flexor carpi radialis, pronator teres					X
Cervical paraspinal muscles					X
Nerve Conduction Study Findings					
Abnormal radial SNAP (if axonal)		X	X	X	
Low radial CMAP (if axonal)	X	X	X	X	X
Conduction block at spiral groove (if demyelinating)		X			
Conduction block between forearm and elbow (if demyelinating)	X				

X, may be abnormal; CMAP, compound muscle action potential; SNAP, sensory nerve action potential.

spiral groove or axilla should result in weakness of the brachioradialis, a C5–C6-innervated muscle, which should not be weak in a lesion of the C7 nerve root. On the other hand, radial neuropathy at the spiral groove and PIN should spare the triceps, which would be expected to be weak in a C7 radiculopathy. If a C7 radiculopathy is severe enough to cause muscle weakness, other non-radial C7-innervated muscles also should be weak (e.g., pronator teres, flexor carpi radialis), leading to weakness of arm pronation and wrist flexion. However, in rare situations, non-radial C7-innervated muscles may be relatively spared, making the clinical differentiation quite difficult.

Although lesions of the posterior cord of the brachial plexus result in weakness of radial-innervated muscles, the deltoid (axillary nerve) and latissimus dorsi (thoracodorsal nerve) should also be weak. Central lesions may result in a wrist drop and finger drop. The typical upper motor neuron posture results in flexion of the wrist and fingers, which in the acute phase or when the lesion is mild may superficially resemble a radial neuropathy. Central lesions are identified by increased muscle tone and deep tendon reflexes (unless acute), slowness of movement, associated findings in the lower face and leg, and altered sensation beyond the radial distribution.

ELECTROPHYSIOLOGIC EVALUATION

In the evaluation of a patient with a wrist drop, the role of nerve conduction studies and EMG is to identify a potential

radial neuropathy, assess its location and severity, and, by defining the underlying pathophysiology, establish a prognosis (Table 21–2).

Nerve Conduction Studies

The most important nerve conduction study in assessing a wrist drop is the radial motor study (Box 21–2). A radial compound muscle action potential (CMAP) can be recorded over the EIP muscle, placing the active electrode two fingerbreadths proximal to the ulnar styloid with a reference electrode placed over the ulnar styloid (Figure 21–9). The radial nerve can be stimulated in the forearm, at the elbow (in the groove between the biceps and brachioradialis muscles), and below and above the spiral groove. The normal CMAP recorded from the EIP typically is 2 to 5 mV. Comparing the CMAP amplitude to that on the contralateral asymptomatic side is always important. Any axonal loss will result in a decreased distal CMAP amplitude after 3 to 5 days, when Wallerian degeneration for motor fibers has occurred. In fact, the best way to assess the degree of axonal loss is to compare the CMAP amplitudes between the involved side and the contralateral side.

Several significant technical considerations must be taken into account when performing radial motor studies. First, placement of the active recording electrode over the EIP almost always results in a CMAP with an initial positive deflection. This occurs because volume-conducted potentials from other nearby radial-innervated muscles (e.g., extensor pollicis brevis and longus) contaminate the CMAP response, resulting in an initial positive deflection.

Box 21–2. Recommended Nerve Conduction Study Protocol for Radial Neuropathy

Routine studies:

1. Radial motor study recording extensor indicis proprius, stimulating forearm, elbow, below spiral groove, and above spiral groove; bilateral studies
2. Ulnar motor study recording abductor digiti minimi, stimulating wrist, below groove, and above groove in the flexed elbow position
3. Median motor study recording abductor pollicis brevis, stimulating wrist and antecubital fossa
4. Median and ulnar F responses
5. Superficial radial sensory study recording over the extensor tendons to thumb, stimulating forearm; bilateral studies
6. Ulnar sensory study recording digit 5, stimulating wrist
7. Median sensory study recording digit 2 or 3, stimulating wrist

The following patterns may result:

- *Posterior interosseous neuropathy (axonal loss lesion):* Normal superficial radial SNAP, low amplitude distal radial CMAP.
- *Posterior interosseous neuropathy (demyelinating lesion):* Normal superficial radial SNAP, normal amplitude distal radial CMAP with motor conduction block between forearm and elbow.
- *Posterior interosseous neuropathy (mixed axonal loss and demyelinating lesion):* Normal superficial radial SNAP, low amplitude distal radial CMAP with motor conduction block between forearm and elbow.
- *Radial neuropathy at the spiral groove (axonal loss lesion):* Reduced superficial radial SNAP, low-amplitude distal radial CMAP. No conduction block across spiral groove.
- *Radial neuropathy at the spiral groove (demyelinating lesion):* Normal superficial radial SNAP, normal amplitude distal radial CMAP with conduction block across spiral groove.
- *Radial neuropathy at the spiral groove (mixed axonal loss and demyelinating lesion):* Reduced superficial radial SNAP, low amplitude distal radial CMAP with conduction block across spiral groove.
- *Radial neuropathy at the axilla (axonal loss lesion):* Reduced superficial radial SNAP, low amplitude distal radial CMAP.
- *Radial neuropathy at the axilla (demyelinating lesion):* Normal superficial radial SNAP, normal amplitude distal radial CMAP with normal motor study to above spiral groove.
- *Superficial radial sensory neuropathy:* Reduced superficial radial SNAP, normal radial motor study.

CMAP, compound muscle action potential; SNAP, sensory nerve action potential.

Second, it may be difficult to make accurate surface distance measurements. Because the radial nerve winds around the humerus and takes a somewhat circuitous course through the forearm, surface distance measurements often are inaccurate. Measuring distance with obstetric calipers, especially between the elbow and arm, reduces some of this error. However, the combination of difficulty

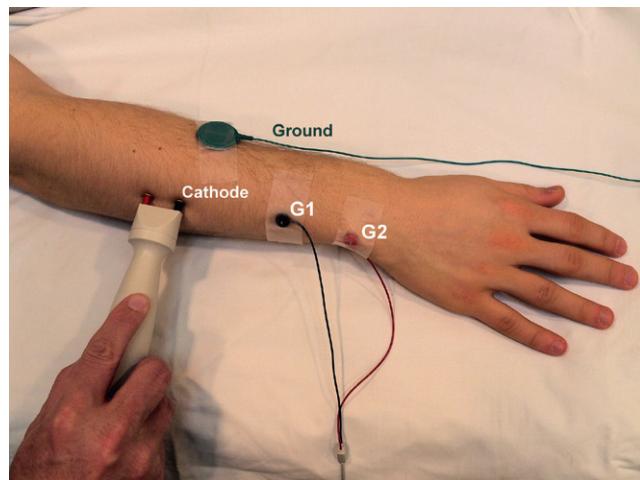


FIGURE 21–9 Radial motor study. The active electrode is placed over the extensor indicis proprius, 2 cm proximal to the ulnar styloid, with the reference electrode over the ulnar styloid. The radial nerve can be stimulated in the forearm, at the elbow, and below and above the spiral groove.

measuring the true nerve length and the initial positive deflection CMAP can lead to considerable potential inaccuracies in measuring true conduction velocities. Radial conduction velocities sometimes are calculated as factitiously fast (>75 m/s). The value of performing radial motor studies usually lies not in the measurement of conduction velocities but in looking for a focal conduction block between the proximal and distal sites and determining the relative CMAP amplitude to assess axonal loss (Figure 21–10).

In cases of radial neuropathy at the spiral groove, CMAPs recorded with stimulation at the forearm, elbow, and below the spiral groove can be completely normal if the lesion is purely demyelinating. However, stimulation above the spiral groove will result in electrophysiologic evidence of a conduction block, i.e., a marked decrease of amplitude and area. The relative drop in distal to proximal CMAP amplitude will give some indication of the proportion of fibers blocked.

Rarely, in cases of PIN, there may be conduction block between the forearm and elbow sites. However, most cases of PIN are pure axonal loss lesions (akin to ulnar neuropathy at the elbow), and no conduction block is demonstrable. In these cases, the distal radial CMAP amplitude will be decreased in proportion to the amount of axonal loss.

In contrast to radial motor studies, the superficial radial sensory nerve is easy to stimulate and record (Figures 21–11 and 21–12). The active electrode is placed over the extensor tendons to the thumb, with the reference electrode placed 3 to 4 cm distally. The nerve is easily stimulated 10 cm proximally, over the radius. If there has been secondary axonal loss, the response will be diminished in amplitude. Similar to motor studies, it is often useful to compare the response with the contralateral asymptomatic

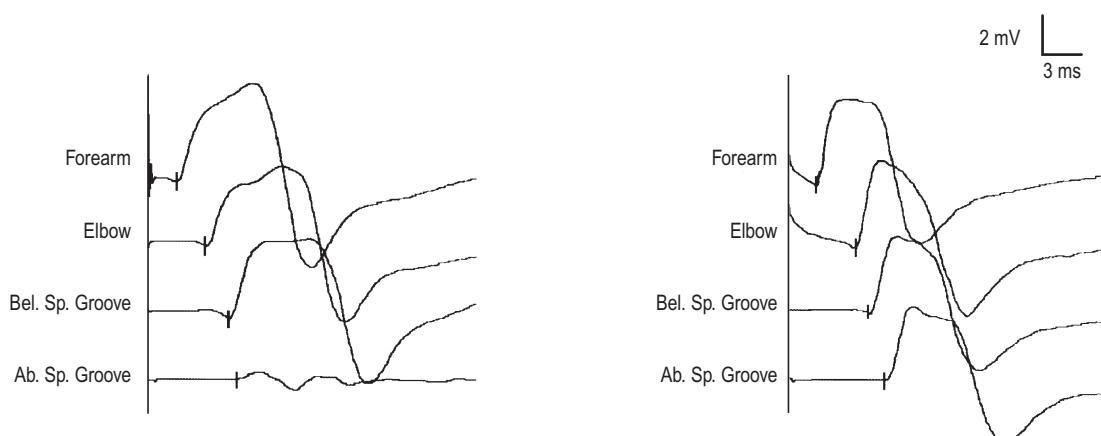


FIGURE 21-10 Radial motor studies for radial neuropathy at the spiral groove. **Left:** Symptomatic arm. **Right:** Contralateral asymptomatic arm. Recording extensor indicis proprius and stimulating the forearm, elbow, below spiral groove, and above spiral groove. Note the marked drop in amplitude and area across the spiral groove on the left (conduction block) and the symmetric distal compound motor action potential amplitudes from side to side. Taken together, these findings imply a predominantly demyelinating lesion at the spiral groove.

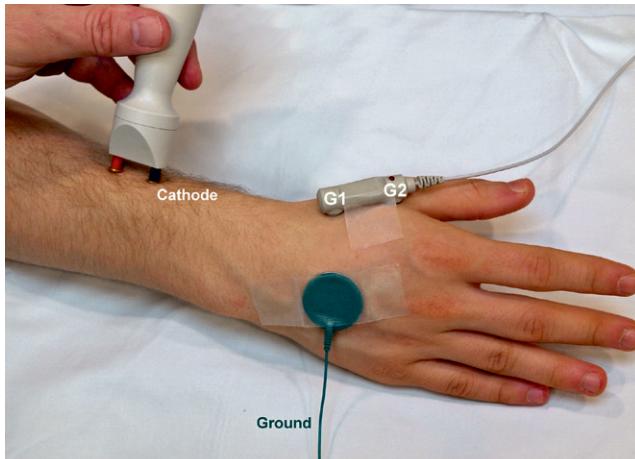


FIGURE 21-11 Radial sensory study. The superficial radial sensory nerve is easy to palpate over the extensor tendons. The active electrode is placed over the nerve with the reference electrode placed 3 to 4 cm distally. The superficial radial nerve is stimulated 10 cm proximal to G1 over the radial bone.

side. If the pathology is one of pure or predominant proximal demyelination, a very interesting phenomenon occurs. Although the patient reports marked numbness in the distribution of the superficial radial sensory nerve, the superficial radial sensory nerve action potential (SNAP) will be normal, even comparing side to side. This unusual finding (a normal sensory response in the distribution of cutaneous numbness) can occur in only one of three situations: (1) a hyperacute axonal loss lesion (before Wallerian degeneration has occurred), (2) a lesion proximal to the dorsal root ganglion, or (3) a lesion caused by proximal demyelination. Thus, in cases of radial neuropathy at the spiral groove or axilla, a pure proximal demyelinating lesion will result in a normal superficial radial sensory potential, despite

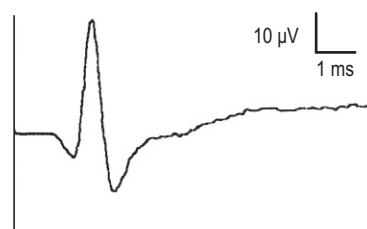


FIGURE 21-12 Radial sensory nerve action potential. The radial sensory nerve action potential is easy to record and typically has a triphasic morphology. It is expected to be normal in all posterior interosseous neuropathy lesions, as well as in other higher radial neuropathies that are purely demyelinating.

Box 21-3. Causes of Wrist Drop and a Normal Superficial Radial Sensory Nerve Action Potential

- Posterior interosseous neuropathy
- Demyelinating radial neuropathy at the spiral groove or axilla
- C7 radiculopathy
- Central nervous system lesion
- Hyperacute axonal loss injury of the main radial nerve (<4 days old)

sensory loss on clinical exam. A normal superficial radial sensory response is also seen in PIN, as expected, as the nerve carries no cutaneous sensory fibers. One can see that if a patient presents with a wrist drop and the superficial radial SNAP is normal, the differential diagnosis is quite limited (Box 21-3).

Note that if the clinical examination suggests weakness beyond the radial distribution, investigation for a more widespread neuropathy is indicated, especially a search for conduction blocks along other motor nerves, which may

Box 21–4. Recommended Electromyographic Protocol for Radial Neuropathy

Routine muscles:

1. At least two posterior interosseous-innervated muscles (e.g., extensor indicis proprius, extensor carpi ulnaris, extensor digitorum communis)
2. At least one radial-innervated muscle proximal to the bifurcation of the main radial nerve near the elbow but distal to the spiral groove (e.g., brachioradialis, long head of extensor carpi radialis)
3. At least one radial-innervated muscle proximal to the spiral groove (e.g., triceps brachii, anconeus)
4. At least one non-radial posterior cord-innervated muscle (e.g., deltoid, latissimus dorsi)
5. At least two non-radial C7-innervated muscles (e.g., flexor carpi radialis, pronator teres, flexor digitorum sublimis, cervical paraspinal muscles)

Special considerations:

- The only electromyographic abnormality in purely demyelinating lesions with conduction block will be decreased recruitment of MUAPs in weak muscles.
- The supinator muscle is best avoided. It is deep and difficult to localize and often is spared in posterior interosseous neuropathy.

MUAP, motor unit action potential.

indicate multifocal motor neuropathy with conduction block (see Chapter 26).

Electromyographic Approach

The EMG approach is straightforward in suspected radial neuropathy (Box 21–4). In a patient with wrist drop and finger drop, the EMG must differentiate among PIN, radial neuropathy at the spiral groove, radial neuropathy in the axilla, a posterior cord lesion, a C7 radiculopathy, and a central lesion. In PIN, abnormalities will be limited to those muscles innervated by the posterior interosseous nerve (among them, the EIP, extensor digitorum communis, and extensor carpi ulnaris), notably sparing the brachioradialis, long head of the extensor carpi radialis, and triceps. In radial neuropathy at the spiral groove, the brachioradialis, long head of the extensor carpi radialis, and supinator will be abnormal, in addition to PIN-innervated muscles, with notable sparing of the triceps. If the lesion is at the axilla, the above muscles, as well as the triceps and anconeus, will be involved. A proximal lesion of the posterior cord will show additional abnormalities, including the deltoid (axillary nerve) and latissimus dorsi (thoracodorsal nerve). A C7 radiculopathy will show abnormalities of the cervical paraspinal muscles and radial-innervated C7 muscles (e.g., triceps, extensor digitorum communis) as well as non-radial-innervated C7 muscles (e.g., pronator teres, flexor carpi radialis). Finally, in central lesions, motor unit action potential (MUAP) configuration and recruitment will be normal in weak muscles, but decreased *activation* of normal configuration MUAPs will be seen.

Anatomic Considerations of Some Radial-Innervated Muscles on Needle EMG

The EMG evaluation of radial neuropathy is very orderly, as there are many muscles innervated by the radial nerve, including several below and above each potential entrapment site. However, there are unique characteristics and limitations of certain muscles, including:

- **Anconeus.** The anconeus is a unique muscle because it is the only muscle in the forearm proper that is supplied by the radial nerve *above the spiral groove*. The anconeus can essentially be thought of as an extension of the medial head of the triceps. Thus, in severe or complete radial neuropathies at the spiral groove, every radial-innervated muscle in the forearm (which includes every wrist and finger extensor), as well as the supinator and brachioradialis, may be completely denervated, and only the anconeus will be normal.
- **Supinator.** There are four muscles that come off between the radial nerve at the spiral groove and the origin of the posterior interosseous nerve at the Arcade of Frohse: the brachioradialis, the long and short heads of the extensor carpi radialis, and the supinator. Thus, these muscles are very helpful in determining if the lesion is at the level of the posterior interosseous nerve, or above it, in the region of the elbow. However, the supinator has several significant limitations. First, it is very deep (essentially in the center of the forearm) and, hence, placing the EMG needle correctly is quite problematic. Second, much of supination is subserved by the biceps muscle (the primary function of the biceps is elbow flexion; its secondary function is forearm supination). Thus, weakness of supination may be difficult to elicit in radial neuropathy. Third, the supinator and its relationship to the radial nerve are somewhat akin to that of the pronator teres and the median nerve: the deep branch of the radial nerve runs through the supinator muscle at which point it is known as the posterior interosseous nerve. However, the branch or branches supplying the supinator originate from the deep radial motor branch *before* it enters under the Arcade of Frohse. Lesions at that location may or may not affect the innervation to the supinator (again, akin to the pronator teres being spared in some cases of pronator syndrome). Because of these limitations, the supinator is best avoided, especially since there are other muscles (especially the brachioradialis and long head of the extensor carpi radialis) that can be more easily sampled that are below the spiral groove but proximal to the posterior interosseous nerve.
- **Extensor carpi radialis – long head.** As noted above, several muscles come off between the main radial nerve at the spiral groove and the origin of the posterior interosseous nerve at the Arcade of Frohse, including the long head of the extensor carpi radialis and the brachioradialis. Thus, these muscles are very

helpful in determining if the lesion is at the level of the posterior interosseous nerve, or proximal to it, in the main radial nerve in the region of the elbow. However, in the case of the long head of the extensor carpi radialis, it is located anatomically just proximal to the short head of the extensor carpi radialis. Thus, in order to place the EMG needle correctly in the long head of the extensor carpi radialis, one has to be quite exact. This is especially important because if the needle is mistakenly placed in the short head of the extensor carpi radialis (also known as the extensor carpi radialis brevis), and found to be abnormal, the mistaken impression may arise of a lesion in the main radial nerve at or proximal to the elbow, whereas the lesion may actually be more distal, in the deep radial motor branch. This is because the short head of the extensor carpi radialis has several common anatomic variants: it can arise from the main radial nerve in the elbow as well as from the deep radial motor branch, and rarely from the proximal superficial radial nerve. One can see that if the short head of the extensor carpi radialis in this case is supplied by the deep radial motor branch rather than the main radial nerve, the mistaken impression of a lesion of the main radial nerve could be made. Because of the anatomic variations of the nerve supply to the extensor carpi radialis brevis, abnormalities in this muscle cannot differentiate between lesions of the main radial nerve in the elbow and the deep radial motor branch.

Thus, although the long head of the extensor carpi radialis can be routinely sampled, of the available muscles that can be sampled which are below the spiral groove but proximal to the bifurcation of the radial nerve just distal to the elbow, *the brachioradialis is the easiest and has the fewest potential problems.*



EXAMPLE CASES

Case 21–1

History and Physical Examination

A 42-year-old man was referred for persistent left wrist drop. The patient reported that he was well until approximately 3 weeks ago, when he awoke with a nearly complete left wrist drop and finger drop. Although there was no pain, he did notice an area of abnormal sensation on the back side of his hand between the thumb and index finger. The patient, initially concerned about a stroke, presented to his local emergency room, where no specific diagnosis was made. During the subsequent 3 weeks, no improvement occurred.

On physical examination, the patient was a well-appearing man with a prominent left wrist drop and finger drop. There was near paralysis of wrist and finger extension (MRC grade 1/5). Finger abduction initially appeared weak, but strength was much better when the hand was passively extended to the neutral position. Wrist and finger flexion were intact. Elbow flexion and

extension were normal. Shoulder abduction was normal. On sensory examination, there was a well-demarcated area of numbness over the lateral dorsum of the left hand between the thumb and index fingers extending into the proximal phalanges of the index, middle, and ring fingers. Otherwise, sensation was intact. Reflexes were normal and symmetric at the biceps and triceps. The left brachioradialis reflex was absent, whereas the right was normal. In the lower extremities, the knee reflexes were normal, but both ankle reflexes were difficult to elicit.

Summary

In this case, the patient presented with the acute onset of marked wrist drop and finger drop. The differential diagnosis includes PIN, radial neuropathy at the spiral groove or in the axilla, a posterior cord lesion of the brachial plexus, an unusual C7 radiculopathy, or a central lesion. The pattern of weakness on the physical examination suggests radial neuropathy at the spiral groove as the most likely localization. Clinically, a PIN is excluded because of (1) the presence of abnormal sensation in the superficial radial distribution (superficial radial sensory nerve) and (2) the abnormal brachioradialis reflex (radial nerve above the elbow). A radial neuropathy in the axilla remains possible but is less likely in the absence of any sensory abnormality in the distribution of the posterior cutaneous nerve of the forearm and arm and especially in the presence of the intact triceps muscle strength and reflex. A lesion of the posterior cord of the brachial plexus is unlikely for the same reasons and also because of the normal strength of the deltoid and latissimus dorsi, which would be expected to be abnormal if the lesion affected the posterior cord. The clinical presentation of a C7 radiculopathy occasionally can mimic a radial neuropathy. However, in such a case the triceps strength and reflex would be expected to be abnormal, as well as the median-innervated C7 muscles (e.g., pronator teres, flexor carpi radialis). Finally, a central lesion appears very unlikely, both because the motor and sensory deficits fit the distribution of a peripheral nerve (i.e., radial nerve) and because no increased reflexes, spasticity, or other signs that accompany an upper motor neuron lesion are present.

Nerve conduction studies begin with the radial motor studies. On the involved left side, a normal radial CMAP is recorded over the EIP muscle, with the forearm, elbow, and below the spiral groove stimulated. When stimulating above the spiral groove, there is a marked drop in amplitude (4.6 mV below the spiral groove, 0.7 mV above). This finding (conduction block) is a clear indication of demyelination across the spiral groove. When the contralateral radial motor nerve is studied, no drop in amplitude with proximal stimulation is noted. Significantly, the distal CMAPs on the involved and uninvolved sides are nearly identical (the involved side actually is slightly higher than the uninvolved side). Because the lesion is 3 weeks old, sufficient time has passed that any Wallerian degeneration that will occur has already occurred in the motor nerves (i.e., 3–5 days). Comparing

CASE 21-1. Nerve Conduction Studies

Nerve Stimulated	Stimulation Site	Recording Site	Amplitude Motor = mV; Sensory = μ V			Latency (ms)			Conduction Velocity (m/s)			F Wave Latency (ms)		
			RT	LT	NL	RT	LT	NL	RT	LT	NL	RT	LT	NL
Radial (m)	Forearm	EIP	5.0	5.7	\geq 2	3.1	3.1	\leq 3.3	57 55 \geq 49			31 \leq 31		
	Elbow	EIP	5.0	4.6		6.6	6.7							
	Below spiral groove	EIP	4.5	4.6		9.4	9.3					60 63 \geq 49		
	Above spiral groove	EIP	4.3	0.7		11.0	11.7					65 45 \geq 49		
Median (m)	Wrist	APB	8.0 6.9		\geq 4	4.3 8.2		\leq 4.4	51			31 \leq 31		
	Antecubital fossa	APB												
Ulnar (m)	Wrist	ADM	7.1 6.7 5.7		\geq 6	2.9 6.5 8.5		\leq 3.3	55 50 \geq 49			31 \leq 32		
	Below elbow	ADM												
	Above elbow	ADM												
Radial (s)	Forearm	Snuffbox	21	10	\geq 15	2.2	2.6	\leq 2.9	63	55	\geq 50			
Median (s)	Wrist	Index finger	12	11	\geq 20	3.6	3.7	\leq 3.5	48	46	\geq 50			
Ulnar (s)	Wrist	Little finger	11	12	\geq 17	2.9	3.2	\leq 3.1	44	46	\geq 50			
Sural (s)	Calf	Posterior ankle	2		\geq 6	4.3		\leq 4.4	45					

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

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 21-1. Electromyography

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

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

the CMAP amplitude on the involved side with that on the asymptomatic side is the best way to assess the amount of axonal loss. Thus, there are two pieces of evidence pointing to demyelination as the predominant pathophysiology in this radial neuropathy: conduction block at the spiral groove and intact distal CMAP amplitude. The median and ulnar motor conduction studies are then performed to exclude a more widespread lesion of the brachial plexus. The results of both motor studies are normal.

Next, the sensory studies are performed. The median sensory amplitudes are reduced, with mild prolongation of peak latency bilaterally. However, these potentials are relatively symmetric between the involved and unininvolved sides. Similar findings are discovered in the ulnar sensory studies. When the radial sensory potentials are obtained, however, there is a clear asymmetry: the involved left side is significantly lower in amplitude than the asymptomatic right side.

At this point in the study, there is definite evidence of a left radial neuropathy across the spiral groove that is predominantly demyelinating. The low superficial radial sensory amplitude implies an axonal loss component as well. In addition, there are reduced median and ulnar sensory potentials bilaterally. An ipsilateral brachial plexopathy cannot account for these reduced sensory potentials because the contralateral side shows similar changes in the median and ulnar sensory nerves. This suggests the possibility of a superimposed polyneuropathy. To investigate this idea further, the sural sensory potential is obtained, and it is found to be low in amplitude as well. Thus, the nerve conduction studies have provided additional evidence of an underlying mild polyneuropathy.

Moving onto EMG, three muscles innervated by the posterior interosseous nerve (EIP, extensor digitorum communis, extensor carpi ulnaris) are checked first. Each of them shows fibrillation potentials and markedly reduced recruitment of MUAPs with normal morphology. This is the classic pattern of a subacute lesion. Enough time has occurred so that fibrillation potentials are present (2–3 weeks), but there has not been sufficient time for reinnervation to occur (months). This is the typical pattern that occurs following acute trauma, compression, or nerve infarction. Note that this pattern always indicates that something acute has occurred within the last several weeks, and is not seen with the typical polyneuropathy, which is usually slowly progressive.

Moving onto muscles innervated above the posterior interosseous nerve, both the brachioradialis and extensor carpi radialis-long head show similar findings to the distal radial (PIN) muscles. When checking the extensor carpi radialis, sampling the long head is important. The long head is always innervated by the radial nerve above the bifurcation near the elbow, whereas the short head may be innervated by either the deep motor branch of the radial nerve or the main radial nerve in the elbow. Next, the triceps brachii and medial deltoid are sampled and are found to be normal. Since these two muscles are

normal, this makes a radial lesion above the spiral groove, in the axilla, or a lesion in the posterior cord of the brachial plexus much less likely. Next, two non-radial-innervated distal muscles (i.e., abductor pollicis brevis and first dorsal interosseous) are sampled. They show only borderline enlarged polyphasic MUAPs without fibrillation potentials. These findings are much less dramatic than those seen in the radial-innervated muscles, and, because the muscles are distal, the findings may be consistent with a polyneuropathy, as suggested by the nerve conduction studies. Finally, proximal, non-radial-innervated C6 and C7 muscles (pronator teres, biceps brachii) are sampled and found to be normal.

At this point, we are ready to form an electrophysiologic impression.

IMPRESSION: *There is electrophysiologic evidence of a subacute, predominantly demyelinating radial neuropathy across the spiral groove with a superimposed mild axonal sensorimotor polyneuropathy.*

Several questions can be considered.

Could the Radial Neuropathy and Sensorimotor Polyneuropathy have a Common Etiology?

After the EMG, the patient was questioned regarding possible alcohol use. He described moderately heavy use of alcohol for the past 10 years and excessive drinking the night before he awakened with the wrist drop. Thus, there may be a good explanation for the underlying polyneuropathy (alcohol-induced), along with a reasonable answer to why the patient awoke with an acute compressive radial neuropathy at the spiral groove. Prolonged immobilization from a deep sleep or after intoxication is the most common cause of this type of radial neuropathy.

Do the Nerve Conduction Studies and EMG Correlate Well?

The nerve conduction studies and EMG findings correlate quite closely. Nerve conduction abnormalities point to a definite demyelinating lesion across the spiral groove, and the EMG findings show subacute changes only in radial-innervated muscles below the spiral groove. Both nerve conduction studies and EMG studies localize the lesion to the same location. In addition, the results can help to assess the severity and underlying pathophysiology. Clearly, the severity is fairly pronounced. There is markedly reduced recruitment of MUAPs in the weak muscles, signifying that most of the motor axons have been blocked. However, despite the severity of the lesion, given that the distal radial CMAP amplitude is normal, and the lesion is predominantly demyelinating, the prognosis is quite good.

If the Lesion is Predominantly Demyelinating, Why are so Many Fibrillation Potentials Seen?

One can be fairly certain that the primary pathophysiology is demyelination. Demyelination is demonstrated by

preservation of the distal radial CMAP amplitude and the clear finding of conduction block across the spiral groove. One then may ask why are there so many fibrillation potentials if the primary pathophysiology is demyelination? Almost all demyelinating lesions are associated with some secondary axonal loss and, accordingly, with fibrillation potentials. Many studies have shown that the number of fibrillation potentials correlates quite poorly with the amount of axonal loss. Indeed, prominent fibrillation potentials are common even with a small amount of axonal loss. Loss of CMAP amplitude much more accurately approximates the amount of axonal loss, especially with acute lesions, but after enough time has elapsed that Wallerian degeneration has occurred. Therefore, although this study shows both a demyelinating and an axonal loss component to the radial neuropathy, the primary problem here is demyelination. This fact has direct implications for prognosis because the prognosis for demyelination usually is very good. It is likely that this patient will recover completely, probably over the next several weeks to months. If, on the other hand, the distal CMAP amplitude had been very low or unobtainable, implying axonal loss, the prognosis would be much more guarded. In that case, nerve regrowth would have to occur from the distal stump, at a rate of 1 mm/day. Axonal regrowth down the length of the arm could easily take months to years and likely would be incomplete.

Case 21–2

History and Physical Examination

An 18-year-old man was referred for right-hand weakness of 2 months' duration. There were no sensory symptoms.

Examination showed marked weakness of finger extension. Wrist extension also was weak, and a radial deviation was noted. Finger and wrist flexion were normal, as was intrinsic hand function. Reflexes and sensation were normal.

Summary

The history and physical examination in this case are primarily indicative of wrist drop and finger drop. The differential diagnosis again includes a lesion of the posterior interosseous nerve, a radial neuropathy at the spiral groove or at the axilla, a posterior cord lesion, a C7 radiculopathy, and a central lesion. The physical examination provides several clues that help limit the differential diagnosis. Normal sensation favors a lesion of the posterior interosseous nerve as opposed to the main radial nerve. Of course, sensory loss may be vague or ill-defined in a radiculopathy, and sensation could also be normal in a central lesion. With voluntary wrist extension, there is a radial deviation, suggesting that the long head of the extensor carpi radialis is relatively preserved compared to the extensor carpi ulnaris. Such a pattern is commonly seen in an isolated lesion of the posterior interosseous nerve.

When the radial motor study is performed on the involved side, there is a very low CMAP amplitude with forearm stimulation when recording the EIP. No potential can be elicited when stimulating at the elbow or above. In contrast, the contralateral side shows a normal distal CMAP amplitude, and no drop is seen with proximal stimulation. Therefore, we can be certain that there has been severe axonal loss of the right radial motor fibers. One might question the possibility of a conduction block

CASE 21–2. Nerve Conduction Studies

Nerve Stimulated	Stimulation Site	Recording Site	Amplitude Motor = mV; Sensory = μ V			Latency (ms)			Conduction Velocity (m/s)			F Wave Latency (ms)										
			RT	LT	NL	RT	LT	NL	RT	LT	NL	RT	LT	NL								
Radial (m)	Forearm	EIP	0.2	7.8	\geq 2	2.4	1.7	\leq 2.9														
	Elbow	EIP	NR	7.7	\geq 2	NR	4.7							67	\geq 49							
	Above spiral groove	EIP	NR	7.7	\geq 2	NR	8.9							64	\geq 49							
Median (m)	Wrist	APB	5.4	\geq 4		3.6	\leq 4.4								27	\leq 31						
	Antecubital fossa	APB	5.3	\geq 4		7.0	59															
Ulnar (m)	Wrist	ADM	9.8	\geq 6		2.7	\leq 3.3								25	\leq 32						
	Below elbow	ADM	9.6	\geq 6		6.0	61									\geq 49						
	Above elbow	ADM	9.0	\geq 6		7.6	63									\geq 49						
Radial (s)	Forearm	Snuffbox	31	30	\geq 15	1.9	1.7	\leq 2.9	66	68	\geq 50											
Median (s)	Wrist	Index finger	50	\geq 20		2.6	\leq 3.5		69	\geq 50												
Ulnar (s)	Wrist	Little finger	33	\geq 17		2.2	\leq 3.1		65	\geq 50												

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

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 21–2. Electromyography									
Muscle	Insertional Activity	Spontaneous Activity		Voluntary Motor Unit Action Potentials				Configuration	
		Fibrillation Potentials	Fasciculations	Activation	Recruitment	Duration	Amplitude	Polyphasia	
Right extensor indicis proprius	↑	+3	0	NL	↓↓↓	NL/-1	NL/-1	+2	
Right extensor digitorum communis	↑	+2	0	NL	↓↓↓	NL	NL	NL	
Right extensor carpi ulnaris	MK	+1	0	NL	↓	+1	+1	+2	
Right extensor carpi radialis-long head	NL	0	0	NL	NL	NL	NL	NL	
Right brachioradialis	NL	0	0	NL	NL	NL	NL	NL	
Right triceps brachii	NL	0	0	NL	NL	NL	NL	NL	
Right anconeus	NL	0	0	NL	NL	NL	NL	NL	
Right first dorsal interosseous	NL	0	0	NL	NL	NL	NL	NL	
Right pronator teres	NL	0	0	NL	NL	NL	NL	NL	
Right biceps brachii	NL	0	0	NL	NL	NL	NL	NL	
Right medial deltoid	NL	0	0	NL	NL	NL	NL	NL	

↑= increased; ↓= slightly reduced; ↓↓↓ = markedly reduced; NL = normal; MK = myokymic discharges.

between the forearm and elbow sites on the involved site, but with such a low distal potential, the drop in proximal amplitude would be of dubious significance. The median and ulnar motor and sensory studies are subsequently performed to ensure that there is not a more widespread lesion. These studies are normal. The superficial radial sensory potential on the involved side is normal and, when compared with the contralateral side, is symmetric. Thus, a normal superficial radial sensory potential accompanies the extremely abnormal radial motor amplitude. This pattern is consistent with either a pure motor lesion or a lesion proximal to the dorsal root ganglion (i.e., nerve root or anterior horn cell). A lesion of the main radial nerve severely affecting motor fibers but sparing sensory fibers would be very unlikely. This pattern is also consistent with a lesion of the posterior interosseous nerve, which is primarily a motor nerve that supplies no cutaneous sensation.

The EMG study shows florid fibrillation potentials in the EIP with markedly reduced recruitment of small, short, very polyphasic MUAPs. Fibrillation potentials and decreased recruitment are also seen in the extensor digitorum communis and extensor carpi ulnaris. All three of these muscles are innervated by the posterior interosseous nerve. In addition, myokymic discharges are present in the extensor carpi ulnaris, along with the fibrillation potentials. When radial muscles innervated proximal to the posterior interosseous nerve are sampled (long head of the extensor carpi radialis, brachioradialis, triceps brachii, anconeus), they are all normal, as are non-radial

C5 through T1-innervated muscles in the upper extremity.

At this point, we can form an electrophysiologic impression.

IMPRESSION: *There is electrophysiologic evidence of a severe predominantly axonal lesion of the posterior interosseous nerve.*

The history, physical examination, and subsequent electrophysiologic evaluation are all consistent with a PIN. In PIN, the radial sensory potential is not involved because the superficial radial sensory nerve separates from the main radial nerve in the proximal forearm, before the take-off to the PIN. This explains why the patient has no sensory complaints and why the radial sensory potential is normal and symmetric in comparison with the asymptomatic side. Rarely, conduction block may be seen on radial motor studies in PIN between the forearm and elbow, but usually the lesion is one of axonal loss.

The EMG probably is the most important test in localizing a lesion to the posterior interosseous nerve, showing abnormalities in muscles innervated by that nerve alone. Once abnormalities are found in muscles innervated by the posterior interosseous nerve, the key muscles to check are those innervated by the radial nerve proximal to the posterior interosseous nerve (i.e., long head of extensor carpi radialis, brachioradialis, anconeus, triceps).

Several questions can be addressed.

What is the Significance of the Myokymic Discharges?

There are several interesting findings in this case. First is the presence of myokymic discharges in the extensor carpi ulnaris. Myokymia is spontaneous activity consisting of grouped repetitive discharges of MUAPs. The generator in myokymia is an abnormal motor nerve, and the pathophysiology is thought to be demyelinating. Myokymia is classically seen in radiation injury, Guillain-Barré syndrome, multiple sclerosis, and brainstem tumors, but it may also be seen in some entrapment neuropathies. Indeed, myokymia rarely is seen in the abductor pollicis brevis muscle in patients with carpal tunnel syndrome. In the case discussed here, myokymic discharges are seen in one of the posterior interosseous-innervated muscles, likely caused by entrapment of the posterior interosseous nerve, with some element of demyelination.

What is the Significance of the Small, Short, Polyphasic Motor Unit Action Potentials?

Small, short, polyphasic MUAPs in the EIP denote that individual motor units have a lower than normal number of muscle fibers. Such loss typically is associated with myopathy or severe disorders of the neuromuscular junction in which individual muscle fibers have been blocked. Therefore, one may ask if there is a coexistent myopathy or neuromuscular junction disorder here as well. The answer is unequivocally no. The other situation in which small, short, polyphasic MUAPs can be seen is in the setting of nascent motor units. In that situation, following severe denervation, the only way muscle fibers can be reinnervated is by regrowth of the axon from the terminal stump, because there are no nearby motor units to reinnervate the denervated muscle fibers by way of collateral sprouting. As such regrowth occurs, there will be a time early in reinnervation when the axon is connected to only a few muscle fibers (i.e., a "nascent motor unit"). Accordingly, the nascent motor unit potentials seen on EMG will be small, short, and polyphasic. How, then, can one distinguish a nascent from a myopathic MUAP? In myopathy, the number of MUAPs firing is normal for the level of activation; therefore, the recruitment is normal or

sometimes even early. The converse is true with nascent motor units, which occur following severe denervation. In this situation, recruitment is always moderately to markedly reduced, often in association with prominent fibrillation potentials. Reviewing again the EMG findings in the EIP, we find more than sufficient evidence to suggest the presence of nascent motor unit potentials. Along with the small, short, polyphasic MUAPs, there are marked fibrillation potentials, and, more importantly, recruitment is markedly reduced.

After the electrophysiologic study, the patient underwent surgical exploration of the posterior interosseous nerve. Compression was identified and relieved at the Arcade of Frohse. Subsequently, the patient had complete recovery of his wrist drop and finger drop, although recovery required 12 months, signifying again that the predominant underlying pathology was axonal loss.

Suggested Readings

- Branovacki, G., Hanson, M., Cash, R., et al., 1998. The innervation pattern of the radial nerve at the elbow and in the forearm. *J Hand Surg (British and European Volume)* 23 (2), 167–169.
- Brown, W.F., Watson, B.V., 1993. AAEM case report no. 27: acute retrohumeral radial neuropathies. American Association of Electrodiagnostic Medicine, Rochester, MN.
- Dawson, D.M., Hallet, M., Millender, L.H., 1983. Entrapment neuropathies. Brown, Boston: Little.
- Rosenbaum, R., 1999. Disputed radial tunnel syndrome. *Muscle Nerve* 22, 960–967.
- Schnall, S.B., Wongworawat, M.D., 2002. Apparent inconsistency regarding the nomenclature of the branches of the radial nerve near the elbow. *J Hand Surg Am* 27 (5), 916–917.
- Sprockin, B.E., 1954. Cheiralgia Paresthetica – Wartenberg's disease. *Neurology* 4 (11), 857.
- Sunderland, S., 1945. Traumatic injuries of the peripheral nerves: simple compression injuries of the radial nerve. *Brain* 68, 56.
- Thomas, S.J., Yakin, D.E., Parry, B.R., et al., 2000. The anatomical relationship between the posterior interosseous nerve and the supinator muscle. *J Hand Surg Am* 25 (5), 936–941.