

## 19

## Ulnar Neuropathy at the Elbow

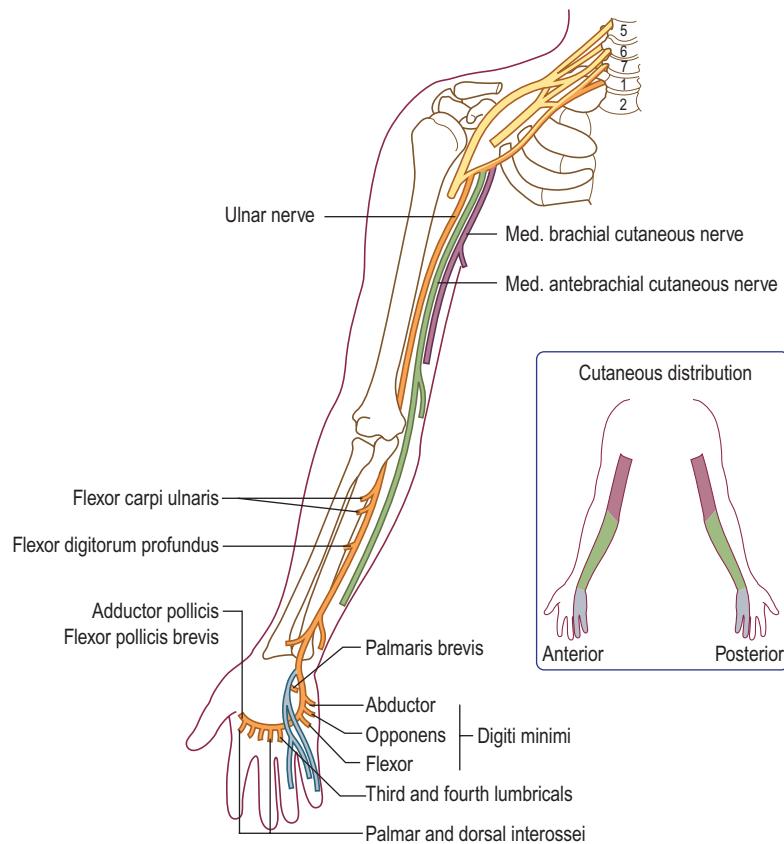
Ulnar neuropathy at the elbow (UNE) is second only to median nerve entrapment at the wrist (i.e., carpal tunnel syndrome [CTS]) as the most common entrapment neuropathy affecting the upper extremity. In contrast to CTS, localizing the site of the lesion by electrodiagnostic (EDX) studies often is much more difficult in patients with ulnar neuropathy. Indeed, the diagnosis of a non-localizable ulnar neuropathy is not infrequently the best that can be accomplished in the electromyography (EMG) lab. Although the elbow is the most common site of compression, the ulnar nerve is susceptible to entrapment at other sites, especially at the wrist. In addition, lesions of the lower brachial plexus or C8–T1 roots may result in symptoms similar to UNE. It is the role of the electromyographer to identify the ulnar nerve lesion, localize it as accurately as possible, and exclude other disorders that may mimic it.

**FIGURE 19–1** Ulnar nerve anatomy. The ulnar nerve, along with the medial brachial and medial anterbrachial cutaneous nerves, is derived from the medial cord of the brachial plexus. **Inset:** Cutaneous distributions of the ulnar, medial anterbrachial, and medial brachial cutaneous nerves.

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

## ANATOMY

The ulnar nerve is essentially derived from the C8 and T1 roots (Figure 19–1), although some anatomic dissections have also demonstrated a minor component from C7. Accordingly, nearly all ulnar fibers travel through the *lower trunk of the brachial plexus* and then continue into the medial cord. The terminal extension of the medial cord becomes the ulnar nerve. The *medial brachial and medial anterbrachial cutaneous sensory nerves and a large contribution to the median nerve* are derived from the medial cord as well. As the ulnar nerve descends through the medial arm, it does so without giving off any muscular branches. The ulnar nerve pierces the medial intermuscular septum in the mid-arm and then passes through the arcade of Struthers, which is composed of deep fascia, muscle fibers



from the medial head of the triceps, and the internal brachial ligament. The ulnar nerve then travels medially and distally toward the elbow.

At the elbow, the nerve enters the ulnar groove formed between the medial epicondyle and the olecranon process. Slightly distal to the groove in the proximal forearm, the ulnar nerve travels under the tendinous arch of the two heads of the flexor carpi ulnaris (FCU) muscle, known as the humeral-ulnar aponeurosis (HUA) or cubital tunnel. Muscular branches to the FCU and the medial division (fourth and fifth digits) of the flexor digitorum profundus (FDP) are then given off.

The nerve then descends through the medial forearm, giving off no further muscular branches until after the wrist. Five to eight centimeters proximal to the wrist, the *dorsal ulnar cutaneous sensory branch* exits to supply sensation to the dorsal medial hand and the dorsal fifth and medial fourth digits. At the level of the ulnar styloid, the *palmar cutaneous sensory branch* originates to supply sensation to the proximal medial palm.

The nerve next enters the medial wrist through Guyon's canal to supply sensation to the volar fifth and medial fourth digits and muscular innervation to the hypothenar muscles, the palmar and dorsal interossei, the third and fourth lumbricals, and two muscles in the thenar eminence, the adductor pollicis and the deep head of the flexor pollicis brevis.

## DETAILED ANATOMY AT THE ELBOW

As the nerve approaches the ulnar groove, it becomes quite superficial (Figure 19–2). The ulnar nerve normally runs in the groove formed by the medial epicondyle of the humerus and the olecranon process of the ulna. In some individuals, fully flexing the elbow may allow the ulnar nerve to sublux out of the groove medially over the medial epicondyle. In

a small number of individuals, a dense fibrotendinous band or an accessory epitrochleoanconeus muscle (or both) may be present between the medial epicondyle and the olecranon process. Just distal to the groove is the HUA (cubital tunnel).

Studies have shown that the distance from the medial epicondyle to the cubital tunnel distally varies between 3 and 20 mm in cadaver dissections and from 0 to 22 mm in surgical specimens. *This variation underscores the importance of stimulating the below-elbow site at least 3 cm distal to the elbow, in routine ulnar motor studies, to ensure that the stimulation is distal to the cubital tunnel, a common site of entrapment.* In the cubital tunnel, the ulnar nerve then continues under the FCU to exit between the deep fascia separating the FCU and FDP. The location of this exit from the cubital tunnel varies from 3 to 7 cm distal to the ulnar groove, according to cadaver studies. The muscular branch to the FCU usually arises distal to the cubital tunnel in 93–95% of cadaver dissections, and always follows the same course as the main ulnar nerve.

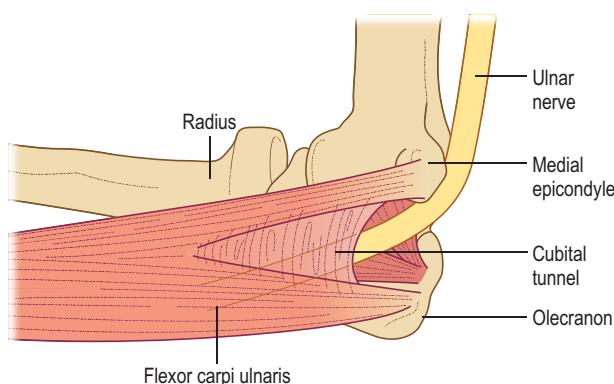
## ETIOLOGY

UNE usually occurs as a result of chronic mechanical compression or stretch, either at the groove or at the cubital tunnel. Although rare cases of ulnar neuropathy at the groove are caused by ganglia, tumors, fibrous bands, or accessory muscles, most are caused by external compression and repeated trauma. Elbow fracture, often sustained years earlier, and subsequent arthritic change of the elbow joint may result in so-called *tardy ulnar palsy*. In addition, chronic minor trauma and compression (including leaning on the elbow) can either exacerbate or cause ulnar neuropathy at the groove. Ulnar neuropathy at the groove also is common in patients who have been immobilized because of surgery or who sustain compression during anesthesia or coma. More controversial is the possibility that repeated subluxation of the ulnar nerve out of the groove (during elbow flexion) also leads to ulnar neuropathy.

Distal to the groove is the cubital tunnel, the other major site of compression of the ulnar nerve in the region of the elbow. Although some use the term *cubital tunnel syndrome* to refer to all lesions of the ulnar nerve around the elbow, it more properly denotes compression of the ulnar nerve under the HUA. Some individuals have congenitally tight cubital tunnels that predispose them to compression. Repeated and persistent flexion stretches the ulnar nerve and increases the pressure in the cubital tunnel, leading to subsequent ulnar neuropathy.

## CLINICAL

UNE caused by compression at the groove or at the cubital tunnel may present in a similar manner. In contrast to CTS, in which sensory symptoms predominate, motor symptoms are more common in ulnar neuropathy, especially in chronic cases. In some patients, insidious motor



**FIGURE 19–2** Detailed ulnar nerve anatomy at the elbow. Entrapment of the ulnar nerve occurs both at the groove (between the medial epicondyle and the olecranon) or distally at the cubital tunnel.  
(Reprinted with permission from Kincaid, J.C., 1988. AAEE minimonograph no. 31: the electrodiagnosis of ulnar neuropathy at the elbow. Muscle Nerve 11, 1005.)

loss may occur without sensory symptoms, particularly in those with slowly worsening mechanical compression. Because most of the intrinsic hand muscles are ulnar innervated, weakness of these muscles leads to loss of dexterity and decreased grip and pinch strength. These are often the complaints that bring the patient to medical attention. There may be atrophy of both the hypothenar and thenar eminences (the ulnar-innervated adductor pollicis and deep head of the flexor pollicis brevis are in the thenar eminence). However, thumb abduction is spared (median and radial innervated).

In moderate or advanced cases, examination often shows the classic hand postures that occur with ulnar muscle weakness. The most recognized is the *Benediction posture* (Figure 19–3). The ring and little fingers are clawed, with the metacarpophalangeal joints hyperextended and the proximal and distal interphalangeal joints flexed (from third and fourth lumbrical weakness), while the fingers and thumb are held slightly abducted (from interossei and adductor pollicis weakness). The *Wartenberg's sign* is recognized as a passively abducted little finger due to weakness of the third palmar interosseous muscle (Figure 19–4). The clinical correlate to this sign is that the patient reports



**FIGURE 19–3** Benediction posture. The deformity results from a combination of finger adduction weakness (interossei) and clawing of digits 4 and 5 (extension at the metacarpophalangeal joints and flexion of the distal and proximal interphalangeal joints, from weakness of the third and fourth lumbricals).



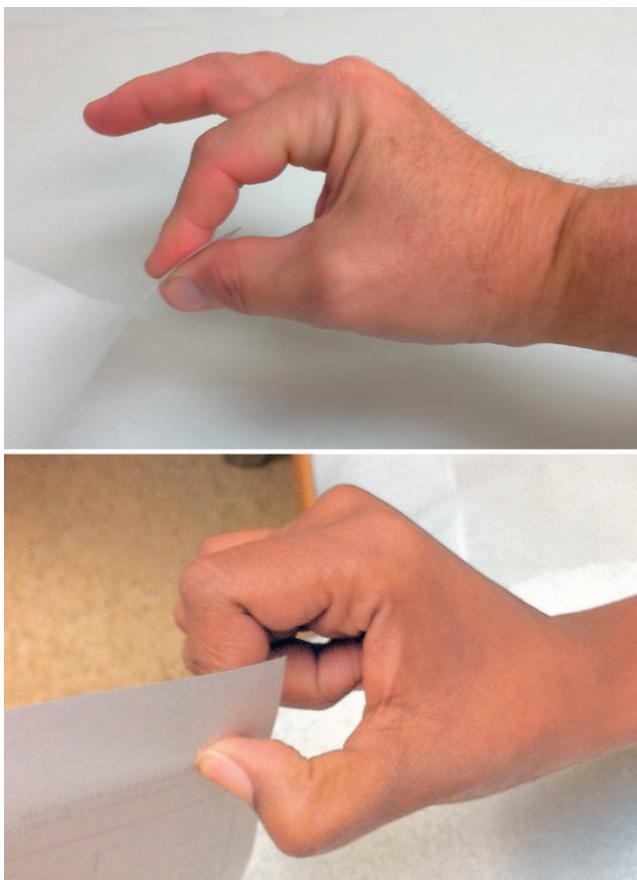
**FIGURE 19–4** Wartenberg's sign. The sign results from difficulty abducting the fifth digit because of preferential weakness of the third palmar interosseous muscle. In the photo, the patient was asked to hold her fingers together. Note that the patient's left fifth finger is held abducted.

getting the little finger caught when trying to put their hand in their pocket. The *Froment's sign* occurs when the patient attempts to pinch an object or a piece of paper (Figure 19–5). To compensate for intrinsic ulnar hand weakness, the long flexors to the thumb and index finger (median innervated) are used, creating a flexed thumb and index finger posture.

Examination of the patient's grip often reveals it to be abnormal. Weakness of the ulnar-innervated FDP will result in the inability to flex the joints of the ring and little fingers. This often can be demonstrated just by having the patient make a fist (Figure 19–6). Patients with ulnar neuropathy may not be able to flex the distal fourth and fifth fingers completely when making a grip; in contrast, the median-innervated second and third distal digits flex normally.

In UNE, sensory disturbance, when present, involves the volar and dorsal fifth and medial fourth digits and the medial hand (Figure 19–7). The sensory disturbance does not extend proximally much beyond the wrist crease. Sensory involvement extending into the medial forearm implies a higher lesion in the plexus or nerve roots (i.e., this is the territory of the medial antebrachial cutaneous sensory nerve, which arises directly from the medial cord of the brachial plexus). Another important skin territory to check is the dorsal medial hand. Sensory abnormalities here are important because they indicate that the dorsal ulnar cutaneous sensory nerve territory also is involved. This finding excludes an ulnar neuropathy at the wrist as the dorsal ulnar cutaneous sensory nerve arises proximal to the wrist.

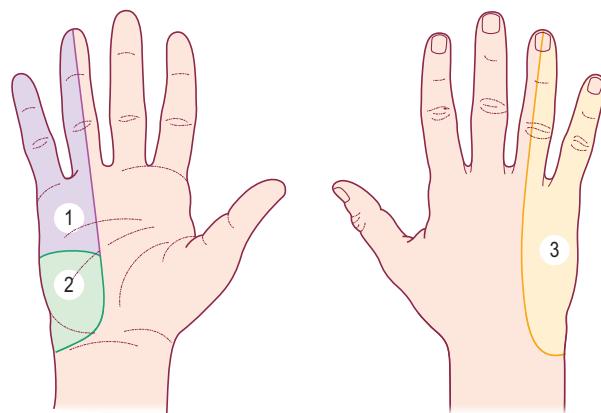
Pain, when present, may localize to the elbow or radiate down to the medial forearm and wrist. Paresthesias may be reproduced by placing the elbow in a flexed position or by



**FIGURE 19–5** Froment's sign. **Top:** Normally to pinch a piece of paper, the finger pads of the thumb and index finger are brought together by the action of the ulnar-innervated adductor pollicis and first dorsal interosseous, respectively. **Bottom:** In ulnar neuropathy, weakness of these muscles results in a characteristic posture, known as the Froment's sign. To compensate, the median-innervated flexor pollicis longus and flexor digitorum profundus (digit 2) have to contract, resulting in marked flexion of the interphalangeal joints of the thumb and index finger.



**FIGURE 19–6** Weakness of ulnar flexor digitorum profundus. In ulnar neuropathy at the elbow, making a fist may result in the inability to completely flex the distal phalanx of the fourth and fifth digits due to weakness of the flexor digitorum profundus to digits 4 and 5. The median-innervated flexor digitorum profundus to digits 2 and 3 is normal (affected hand shown is the right hand – left side of the photo).



**FIGURE 19–7** Sensory loss in ulnar neuropathy. The ulnar nerve contains three sensory branches: (1) ulnar digital sensory branches supply the volar fifth and medial fourth fingers; (2) palmar cutaneous branch supplies the proximal volar medial hand, arising 1 to 2 cm proximal to the wrist; and (3) dorsal ulnar cutaneous sensory branch arises 5 to 7 cm proximal to the wrist and supplies the dorsal medial hand and the dorsal medial fourth and fifth fingers. Lesions at the elbow may be associated with abnormalities in all three territories; lesions at the wrist never involve the dorsal ulnar territory (3) or the proximal volar ulnar palm (2).

applying pressure to the groove behind the medial epicondyle. The ulnar nerve may be palpably enlarged and tender. The nerve may also be palpably taut with decreased mobility, especially in patients with ulnar neuropathy at the cubital tunnel.

## DIFFERENTIAL DIAGNOSIS

The differential diagnosis in a patient suspected of having UNE (Table 19–1) principally includes C8-T1 radiculopathy, lower trunk or medial cord brachial plexopathy, and ulnar neuropathy at the wrist. Very rare cases of ulnar nerve entrapment in the proximal arm and more distally in the forearm have also been reported.

A cervical radiculopathy at the C8-T1 level, although seen less frequently than radiculopathy at the C6 and C7 root levels (which are more commonly affected in cervical disc disease or spondylosis), may be difficult to differentiate clinically from ulnar neuropathy. Neck pain and radiation into the arm, sensory disturbance extending into the forearm, and weakness involving the median and radially innervated C8-T1 muscles are the major differentiating features. Of course, weakness often is minimal and sensory loss often vague in radiculopathy, making the differentiation between a mild C8-T1 radiculopathy and an ulnar neuropathy demanding, if based on clinical findings alone.

Lower trunk/medial cord brachial plexopathies are uncommon. Entrapment of the lower trunk by a fibrous band or hypertrophied muscle results in *neurogenic thoracic outlet syndrome* (see Chapter 30). Lower trunk plexopathies may also result from infiltration by neoplasm, prior radiation, or self-limited inflammatory processes (e.g.,

**Table 19–1.** Clinical Differentiating Factors in Suspected Ulnar Neuropathy

	UNW*	UNE	Medial Cord	Lower Trunk	C8-T1
Weakness of the interossei	X	X	X	X	X
Weakness of the hypothenar muscles	X	X	X	X	X
Weakness of the third and fourth lumbricals	X	X	X	X	X
Weakness of distal finger flexion of the little and ring fingers		X	X	X	X
Weakness of thumb abduction			X	X	X
Weakness of thumb flexion			X	X	X
Weakness of index finger extension				X	X
Sensory loss of the volar medial hand, volar little finger, and volar medial ring finger	X	X	X	X	X
Sensory loss of the dorsal medial hand, dorsal little finger, and dorsal medial ring finger		X	X	X	X
Sensory loss of the medial forearm			X	X	X
Tinel's sign at the elbow			X		
Neck pain					X

\*Assumes both motor and sensory branches are involved; some cases of UNW may spare the hypothenar muscles and/or the sensory branch (for details, see Chapter 20).

X, may be present; UNE, ulnar neuropathy across the elbow; UNW, ulnar neuropathy at the wrist.

neuralgic amyotrophy). Like C8-T1 radiculopathy, lower trunk plexopathies may demonstrate weakness of non-ulnar-innervated C8-T1 muscles and sensory disturbance that extends into the medial forearm.

Other than in the region of the elbow, entrapment of the ulnar nerve in the arm or forearm is rare. In the arm proper, entrapment under the arcade of Struthers has been reported. In the forearm, infrequent cases of ulnar neuropathy occur at the exit of the cubital tunnel. The entrapping structure is the deep fascia between the FCU and FDP. Unusual cases of ulnar neuropathy in the distal forearm have also been reported due to a fibrovascular band supplying blood to a hypertrophied FCU muscle. Clinical differentiation of these unusual cases from typical UNE is difficult. They are usually discovered either by careful electrophysiologic examination, at the time of surgery, or at the time of a second surgery after a failed ulnar surgery at the elbow.

## ELECTROPHYSIOLOGIC EVALUATION

Like other mononeuropathies, the goal of nerve conduction studies and electromyography (EMG) is to demonstrate abnormalities that are limited to one nerve, in this case the ulnar nerve. Although in most cases the lesion is at the elbow, entrapment at the wrist, at the medial cord or lower trunk of the brachial plexus, or at the C8-T1 nerve roots can mimic a UNE clinically. Patterns of nerve conduction and EMG abnormalities often can be used to differentiate these possibilities (Table 19–2). If the ulnar nerve lesion is

demyelinating, nerve conduction studies may demonstrate conduction velocity slowing, conduction block, or both at the lesion site. Unfortunately, in many cases of UNE, the pathophysiology is that of axonal loss, and nerve conduction studies demonstrate only a non-localizable ulnar neuropathy. The EMG study, if abnormal, then can be used to localize the lesion only at or proximal to the takeoff to the most proximal muscle affected on EMG. Because there are no ulnar-innervated muscles above the elbow, the electrophysiologic impression often is one of an ulnar neuropathy at or proximal to the FCU muscle (the most proximal ulnar-innervated muscle).

## Nerve Conduction Studies

The goal of nerve conduction studies in patients with UNE is to demonstrate, when possible, focal demyelination across the elbow (Box 19–1). Focal demyelinating lesions may manifest as slowing of conduction velocity or conduction block between proximal and distal stimulation sites (Figure 19–8). As for focal slowing, one needs to consider how much slowing is abnormal. In general, conduction velocities of more proximal nerve segments are the same as, or more often faster than those of distal segments. This is due to a combination of (1) larger nerve fiber diameter and less tapering of the nerve more proximally (the reason that conduction velocities are faster in the upper than in the lower extremity) and (2) warmer temperatures in the proximal limb compared to the distal limb. In ulnar motor nerve conduction studies, however, this relationship may not hold true unless the position of the elbow is controlled.

**Table 19–2.** Electromyographic and Nerve Conduction Study Abnormalities Localizing the Lesion Site in Ulnar Neuropathy

	UNW	UNE	Medial Cord	Lower Trunk	C8-T1
<b>Electromyographic Findings</b>					
First dorsal interosseous	X	X	X	X	X
Abductor digiti minimi	X	X	X	X	X
Flexor digitorum profundus (digits 4, 5)		X	X	X	X
Flexor carpi ulnaris		X	X	X	X
Abductor pollicis brevis			X	X	X
Flexor pollicis longus			X	X	X
Extensor indicis proprius				X	X
Cervical paraspinal muscles					X
<b>Nerve Conduction Study Findings</b>					
Abnormal ulnar digit 5 SNAP (if axonal)	X	X	X	X	
Abnormal dorsal ulnar cutaneous SNAP (if axonal)		X	X	X	
Abnormal medial antebrachial cutaneous SNAP (if axonal)			X	X	
Low ulnar CMAP (if axonal)	X	X	X	X	X
Low median CMAP (if axonal)			X	X	X
Conduction block/slowing of ulnar nerve across elbow (if demyelinating)		X			

X, may be abnormal; UNW, ulnar neuropathy at the wrist; UNE, ulnar neuropathy across the elbow; SNAP, sensory nerve action potential; CMAP, compound muscle action potential.

Note: The table indicates classic patterns; other patterns may be seen (see text for details).

### Box 19–1. Recommended Nerve Conduction Study Protocol for Ulnar Neuropathy at the Elbow

#### Routine studies:

1. Ulnar motor study recording abductor digiti minimi, stimulating wrist, below elbow, and above elbow in the flexed elbow position (note: the optimal site for stimulating at the below-elbow site is 3 cm distal to the medial epicondyle)
2. Median motor study recording abductor pollicis brevis, stimulating wrist and antecubital fossa
3. Median and ulnar F responses
4. Ulnar sensory response, recording digit 5, stimulating wrist
5. Median sensory response, recording digit 2 or 3, stimulating wrist
6. Radial sensory response, recording snuffbox, stimulating lateral forearm

#### The following patterns may result:

Ulnar neuropathy at the elbow with demyelinating and axonal features:

- Low ulnar SNAP.
- Normal or low-amplitude ulnar CMAP with normal or slightly prolonged distal latency.
- Unequivocal evidence of demyelination at the elbow (conduction block and/or slowing >10–11 m/s across the elbow compared with the forearm segment, in the flexed elbow position).

Ulnar neuropathy at the elbow with pure demyelinating features:

- Normal distal ulnar SNAP and CMAP amplitudes and latencies.
- Unequivocal evidence of demyelination at the elbow (conduction block and/or slowing >10–11 m/s across the elbow compared with the forearm segment, in the flexed elbow position).

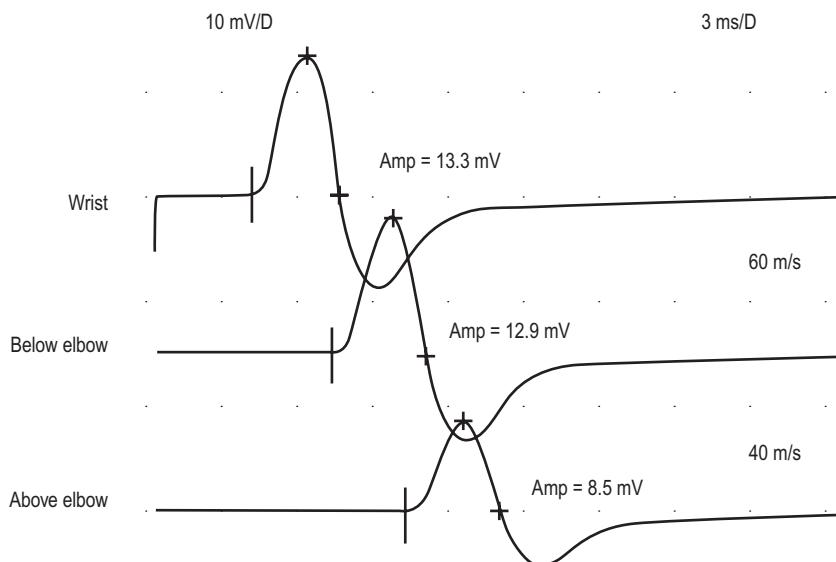
Non-localizable ulnar neuropathy (axonal features alone):

- Low ulnar SNAP.
- Normal or low-amplitude CMAP with normal or slightly prolonged distal latency.
- No focal slowing or conduction block across the elbow.

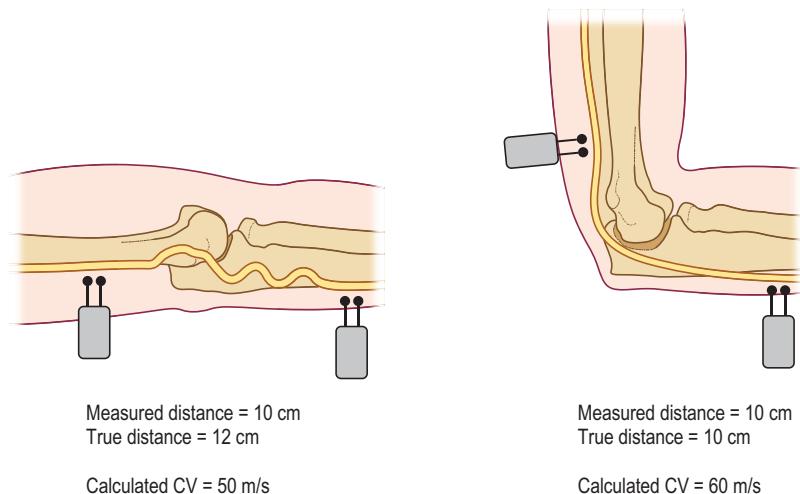
If the ulnar neuropathy is non-localizable, the following studies should be considered:

- Repeat motor studies recording the first dorsal interosseous.
- Inching studies across the elbow.
- Sensory or mixed nerve studies across the elbow.
- Recording the dorsal ulnar cutaneous SNAP (bilateral studies) (remember that the dorsal ulnar cutaneous SNAP can be normal in some patients with ulnar neuropathy across the elbow).
- Recording the medial antebrachial cutaneous SNAP (bilateral studies) if sensory loss extends above the wrist on clinical examination or there is a suggestion of lower brachial plexus lesion by history.

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



**FIGURE 19–8** Focal slowing and conduction block at the elbow. The ulnar compound muscle action potential amplitude is normal at the wrist and below the elbow. Stimulation above the elbow results in a marked drop in amplitude and focal slowing between the above-elbow and below-elbow sites (40 m/s) compared to the forearm segment (60 m/s). These are the electrophysiologic markers of focal demyelination, which allow for definitive localization of ulnar neuropathy at the elbow.



**FIGURE 19–9** Extended versus straight elbow technique and measurement error. **Left:** Ulnar conduction studies performed in the extended elbow position often show artifactual slowing of conduction velocity across the elbow due to underestimation of the true nerve length. In the extended elbow position, the ulnar nerve is slack with some redundancy. **Right:** With the elbow flexed, the true length of the ulnar nerve is more accurately measured, and the resultant conduction velocity is more valid. To prevent this error, the flexed elbow is the preferred position when studying the ulnar nerve.

#### Differential Slowing: Flexed versus Extended Elbow Conduction Techniques

One of the more complicating factors in ulnar conduction studies is the position of the elbow and its effect on the calculated conduction velocity across the elbow. It has been well established in many studies that the position of the elbow during ulnar conduction studies strongly influences the calculated conduction velocity. Ulnar conduction studies performed in the extended (i.e., straight) elbow position often show artifactual slowing of conduction velocity across the elbow due to underestimation of the true nerve length (Figure 19–9). This is because in the extended

elbow position, the ulnar nerve is slack with some redundancy. In normal subjects, this results in ulnar conduction velocities being slower in the across-the-elbow segment than in the segment above or below it, if the study is performed with the elbow in the extended position. Autopsy studies have confirmed that the length of the ulnar nerve across the elbow is measured more accurately with the elbow flexed (i.e., bent).

In several studies of normal controls, the mean differential slowing comparing the across-the-elbow conduction velocity to forearm conduction velocity in the flexed elbow position (90–135 degrees) was 0 m/s, with an upper limit

of normal of 10 to 11 m/s. In contrast, in the extended elbow position, mean slowing was 10 to 11 m/s, with an upper limit of normal in the range of 25 to 30 m/s (to reemphasize, in normal controls!). This extent of factitious conduction velocity slowing across the elbow, in the extended elbow position, is poorly appreciated in some EMG laboratories. Some laboratories arbitrarily use a value of 10 m/s differential slowing across the elbow, in the extended elbow position, to localize an ulnar neuropathy to the elbow. However, appreciation of the large range of variability in normal subjects, with the elbow in an extended position, is crucial to avoid erroneously diagnosing UNE in the normal population. An arbitrary cutoff value of 10 m/s differential slowing between the forearm and across elbow segments, in the extended elbow position, will result in many false-positive diagnoses of UNE. A patient with sensory loss in the little finger from a C8 radiculopathy would not be pleased to undergo ulnar nerve surgery simply based on a conduction velocity slowing of 10 m/s across the elbow compared to the forearm segment, if tested in the extended elbow position (as this is a normal finding in the extended elbow position).

Similar considerations apply to the absolute conduction velocity across the elbow in normal controls. The lower limit of normal for absolute conduction velocity across the elbow is 38 m/s in the extended elbow position, but never drops below 49 m/s in the flexed elbow position. Some have found that the absolute conduction velocity across the elbow is a better measure than differential conduction velocity slowing for detecting abnormalities in patients with ulnar neuropathy. Although absolute conduction velocity across the elbow may be considered a sensitive indicator of ulnar neuropathy, it does not localize the lesion. In any patient with significant axonal loss and dropout of the largest conducting fibers, conduction velocity will decrease across all nerve segments. An ulnar conduction velocity across the elbow segment of 40 m/s has little localizing value if the forearm conduction velocity is also 40 m/s.

In studies comparing the relative usefulness of the flexed versus extended elbow position in demonstrating focal slowing across the elbow, in those patients who had localizing electrophysiology, the flexed elbow position has been found to be more sensitive than the extended position. The difference in the yield between the flexed and extended positions likely is related to the greater range and variability found in normal subjects for differential and absolute conduction velocities across the elbow when tested in the extended elbow position, leading to lower cutoff values.

*Thus, the flexed elbow position is considered the preferred technique when performing ulnar nerve conduction studies across the elbow.* However, the flexed elbow position is more demanding in terms of measuring the curved anatomic course of the ulnar nerve around the elbow. In addition, the flexed position has the drawback of undercalling patients with UNE and subluxable ulnar nerves, which might lead to an overestimation of the true nerve length (see below). Nevertheless, it is far better to undercall UNE in this uncommon patient group, using the flexed

technique, than to erroneously diagnose UNE in normal patients, using the extended position, with inappropriate low cutoff values.

### Conduction Block

In addition to focal slowing, the other electrophysiologic marker of demyelination is conduction block (Figure 19–8). There is some controversy regarding how much the amplitude or area must drop between distal and proximal sites to be considered conduction block (see Chapter 3). Ulnar motor conduction studies in normal subjects have shown a maximum drop in compound muscle action potential (CMAP) amplitude of 10% comparing below and above elbow and 20 to 25% comparing wrist and above-elbow sites. Accordingly, any drop in amplitude of more than 10% between below and above the elbow, especially if associated with a very small change in stimulating electrode position (see the following section) or an abrupt drop in conduction velocity, likely represents true demyelination and is of localizing value.

The other issue that must be considered in the proper interpretation of a conduction block is to not confuse a Martin–Gruber anastomosis (MGA) with a conduction block. Almost always, an MGA is recognized on routine ulnar motor nerve conduction studies as a drop in amplitude and area between the wrist and below-elbow stimulation sites (i.e., mimicking a conduction block in the forearm). The site of the MGA is typically between 3 and 10 cm distal to the medial epicondyle, a location that is not thought to interfere with electrodiagnostic evaluation of UNE. However, there are rare reports of very proximal MGAs wherein the drop in amplitude and area occurs between the below-elbow and above-elbow stimulation sites – that is, across the elbow. Thus, in all cases of ulnar conduction block across the elbow, it is prudent also to check for an MGA (by stimulating the median nerve at the wrist and antecubital fossa, and recording the ulnar muscle) (see more below in the Nerve Conduction Pitfalls section).

### Short Segment Incremental Studies (“Inching”)

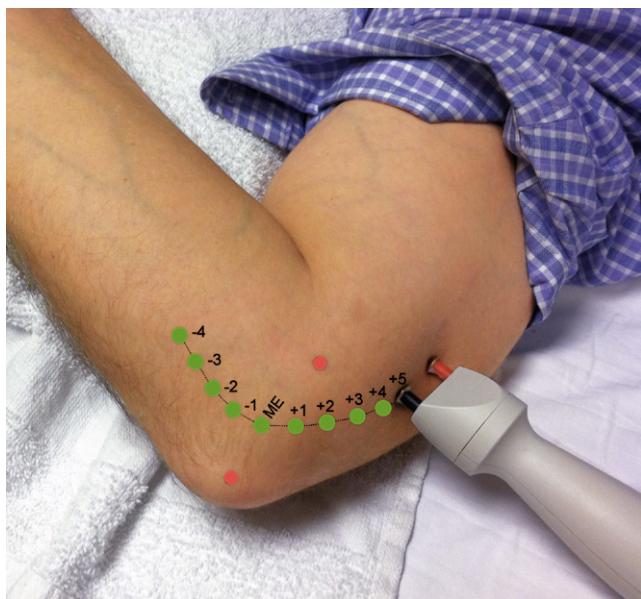
In a technique similar to that used for CTS, short segment incremental studies (SSIS), also known as “inching,” can be performed effectively on the ulnar nerve across the elbow to try to localize the lesion, looking for an abrupt change in either latency or amplitude. The technique is performed as follows:

1. Either the abductor digiti minimi (ADM) or first dorsal interosseous (FDI) muscle is recorded. A mark is first placed halfway between the medial epicondyle and the olecranon to mark the ulnar groove. The location of the ulnar nerve is then mapped out. This process is basically identical to that of ensuring that the stimulator is directly over the nerve, as described in Chapter 3. This is accomplished by using a submaximal current (10–25% supramaximal), and stimulating medial to and lateral to the suspected nerve location in successive sites across the elbow. Several locations are tested sequentially from the

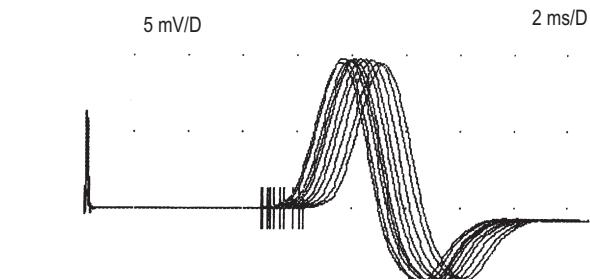
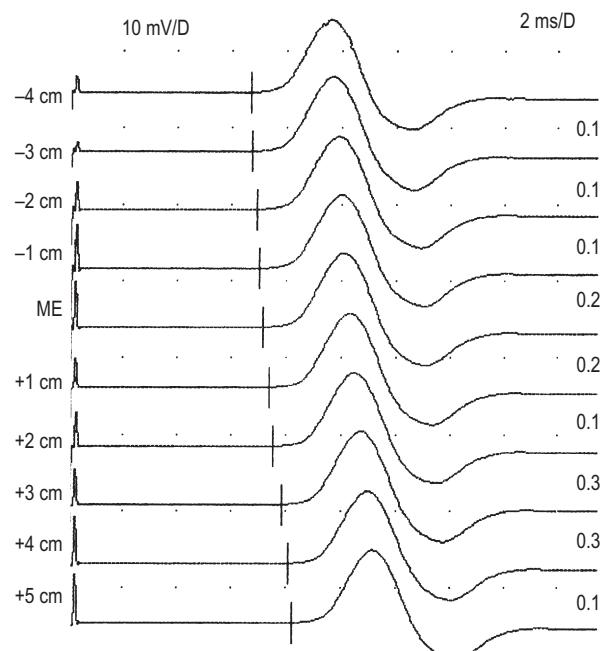
below-elbow to above-elbow sites. At each site, the location that gives the highest CMAP amplitude is the one that is closest to the nerve, and is marked with a marker pen. A line is then drawn across the elbow “connecting all the dots” to mark exactly where the nerve lies.

2. The spot between the medial epicondyle and olecranon is marked as the “zero” point along the line that was drawn across the elbow, and denotes the spot adjacent to the medial epicondyle. Next, 1 cm increments are carefully marked off, along the line that was drawn, from 4 cm below the “zero” point (medial epicondyle) to 4 or 6 cm above.
3. The ulnar nerve is stimulated supramaximally at each location at successive 1 cm intervals from below to above the medial epicondyle (Figure 19–10).

Any abrupt increase in latency or drop in amplitude between successive stimulation sites implies focal demyelination. In normal individuals, the latency between two successive 1 cm stimulation sites usually is 0.1 to 0.3 ms and rarely 0.4 ms (Figure 19–11). Any greater latency shift (i.e.,  $\geq 0.5$  ms) suggests focal slowing and demyelination (Figure 19–12). The inching technique is very sensitive but



**FIGURE 19–10** Short segment incremental studies. To perform these studies, a mark is first placed halfway between the medial epicondyle (ME) and the olecranon to mark the ulnar groove (the red circles are over the ME and olecranon). The location of the ulnar nerve is then mapped using a submaximal current and stimulating from the below-elbow to above-elbow sites, stimulating medial to and lateral to the suspected nerve location in successive sites across the elbow. The location that gives the highest compound muscle action potential amplitude is the one that is closest to the nerve. Several locations are tested from the below-elbow to above-elbow sites to mark exactly where the nerve lies. Then 1 cm increments are carefully marked off from 4 cm below the elbow to 4 or 6 cm above. The ulnar nerve then is stimulated supramaximally at each location at successive 1 cm intervals from below to above the elbow looking for any abrupt change in latency or drop in amplitude.

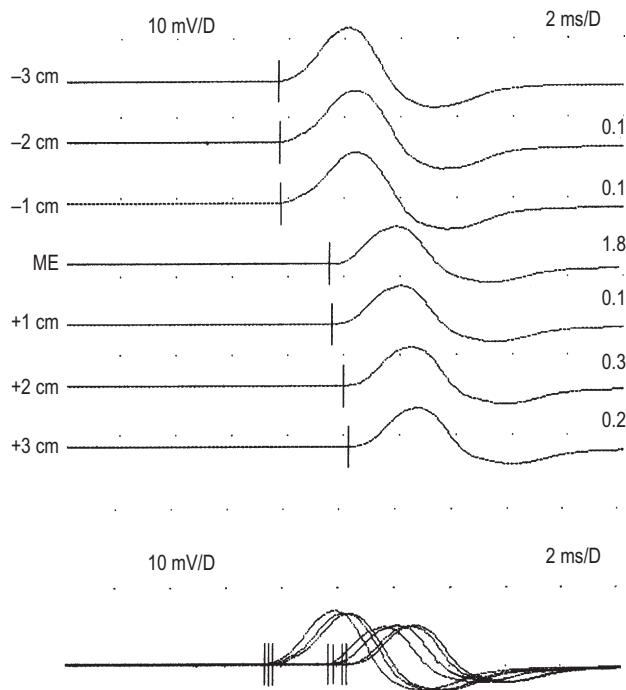


**FIGURE 19–11** Inchng around the elbow – normal. Ten successive traces in 1 cm increments from 4 cm below the medial epicondyle (ME) to 5 cm above. Superimposed traces are at the bottom. The numbers on the right are the latency differences in milliseconds between successive traces. Note: In normals, the amplitude of the waveform stays constant, and the latency difference between successive traces is 0.1 to 0.3 ms.

technically demanding. Any error in measurement is magnified when such short distances are used. The technique has the advantage of potentially being able to directly locate the lesion either at the groove or at the cubital tunnel. This may be of more than just academic interest, because it may be of some help in deciding the best surgical technique to use (e.g., a lesion of the cubital tunnel may be best handled by a simple release rather than a transposition).

#### Recording the First Dorsal Interosseous

In entrapment neuropathies, it is well known that nerve fibers to certain muscles may be preferentially affected whereas others are preferentially spared. Within a nerve, bundles of nerve fibers to different muscles run in separate fascicles separated by connective tissue. External compression may preferentially affect the fibers within the fascicle

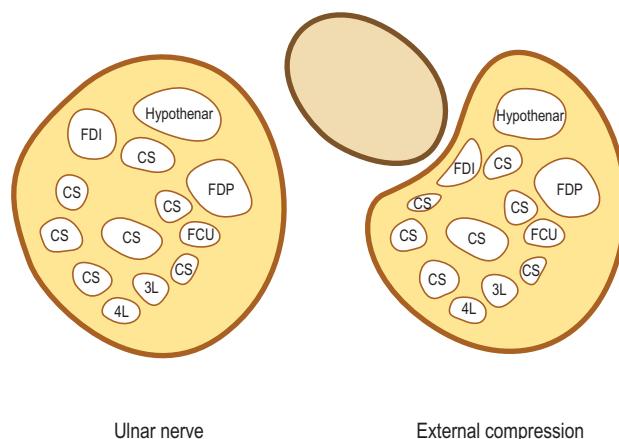


**FIGURE 19–12** Inchng around the elbow – cubital tunnel syndrome. Seven successive traces in 1 cm increments from 3 cm below the medial epicondyle (ME) to 3 cm above. Superimposed traces are at the bottom. The numbers on the right are the latency differences in milliseconds between successive traces. Note: Between the 1 cm below-elbow site and the ME, there is an abrupt change in latency (1.8 ms) and a drop in amplitude. In this case, inching studies not only have confirmed an ulnar neuropathy at the elbow but have precisely located the lesion at the cubital tunnel.

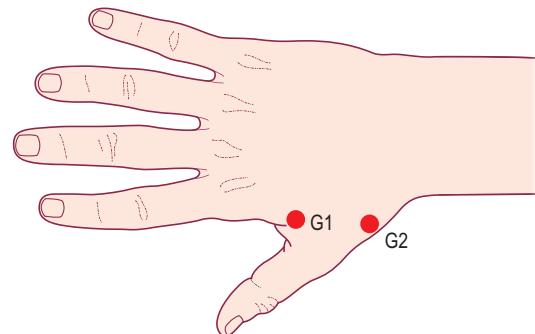
nearest to the compression, thereby preferentially affecting the muscle that those fibers innervate (Figure 19–13). Thus, recording from different muscles sometimes can increase the yield of demonstrating either focal slowing or conduction block. Some studies have shown that recording the first dorsal interosseous (FDI) may be a slightly more sensitive technique than recording the ADM in UNE. When recording the FDI, it is best to place the active electrode over the muscle belly and the reference over the metacarpophalangeal joint of the thumb (Figure 19–14). If the reference electrode is placed on the metacarpophalangeal joint of the index finger, an initial positive deflection often will be seen, which complicates latency measurements (Figure 19–15).

#### Mixed and Sensory Nerve Conductions

Mixed and sensory ulnar nerve conductions across the elbow may increase the yield of identifying focal slowing in patients with UNE. Sensory conductions can be performed antidromically or orthodromically from the fifth digit using the wrist, below-elbow, and above-elbow sites for either stimulating or recording, respectively. Likewise, a mixed nerve potential can be recorded below and above the elbow, stimulating the mixed nerve at the wrist.

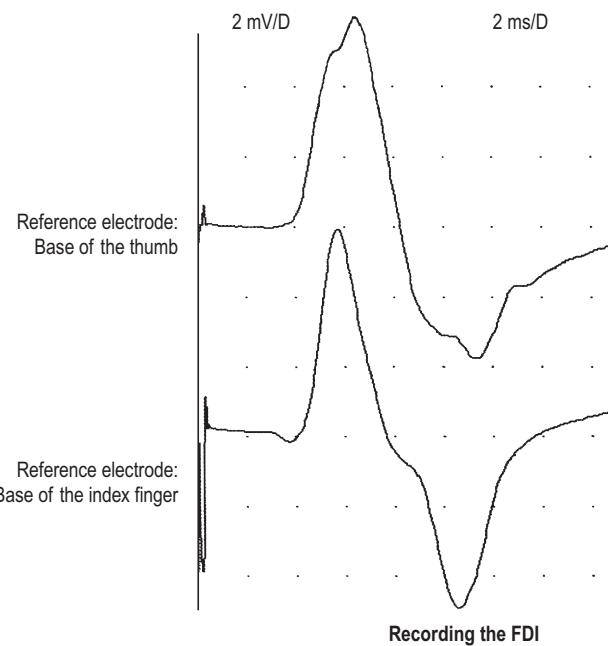


**FIGURE 19–13** Fascicular involvement and sparing in entrapment neuropathies. Within a nerve, bundles of nerve fibers to different muscles run in separate fascicles separated by connective tissue. External compression may preferentially affect the fibers within the fascicle nearest to the compression, thereby preferentially affecting the muscle that those fibers innervate. Thus, recording from different muscles sometimes can increase the yield of demonstrating either focal slowing or conduction block. In this example, the fibers that supply the first dorsal interosseous (FDI) run in a fascicle closest to the site of compression. Accordingly, studying the FDI may have a higher yield of demonstrating abnormalities. 3L, third lumbrical; 4L, fourth lumbrical; FCU, flexor carpi ulnaris; FDP, flexor digitorum profundus; CS, cutaneous sensory.



**FIGURE 19–14** Recording the first dorsal interosseous muscle. The active electrode (G1) is placed over the muscle belly, and the reference electrode (G2) is placed over the metacarpophalangeal joint of the thumb. Recording the first dorsal interosseous is helpful in ulnar neuropathy at the wrist and in some cases of ulnar neuropathy at the elbow.

Although these studies may have increased sensitivity for identifying UNE, they involve significant technical difficulties. Both sensory and mixed nerve potentials dramatically decrease in amplitude when recorded at greater distances because of the normal phenomena of temporal dispersion and phase cancellation (Figure 19–16). For instance, a normal antidromic ulnar sensory nerve action potential (SNAP) amplitude may be 20 µV when stimulated at the wrist; however, stimulating at the below-elbow site, the amplitude may fall to 5 µV and above the elbow to 2 µV. In patients with ulnar neuropathy, these potentials are often low if axonal loss has occurred. In such cases, the potential at the below-elbow and above-elbow sites may be

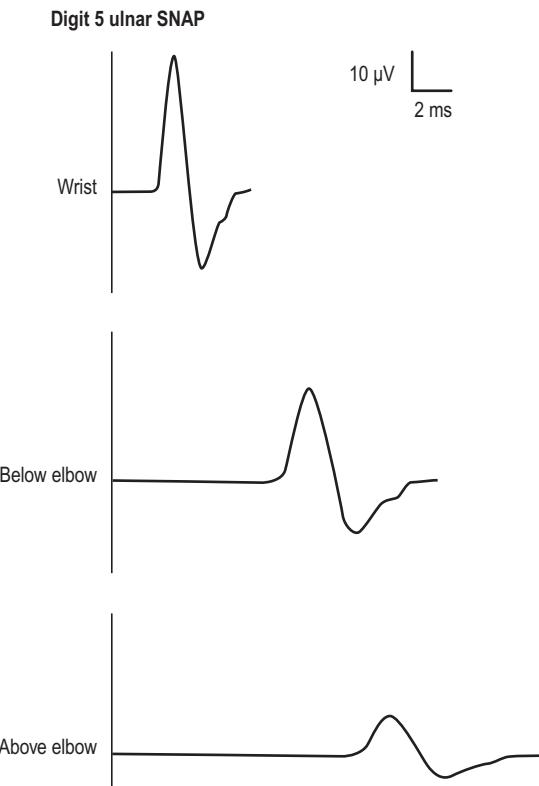


**FIGURE 19–15** First dorsal interosseous muscle – compound muscle action potential morphology and placement of the reference electrode. When recording the first dorsal interosseous, it is best to place the active electrode over the muscle belly and the reference electrode over the metacarpophalangeal joint of the thumb. If the reference electrode is placed on the metacarpophalangeal joint of the index finger, an initial positive deflection often will be seen, which complicates latency measurements.

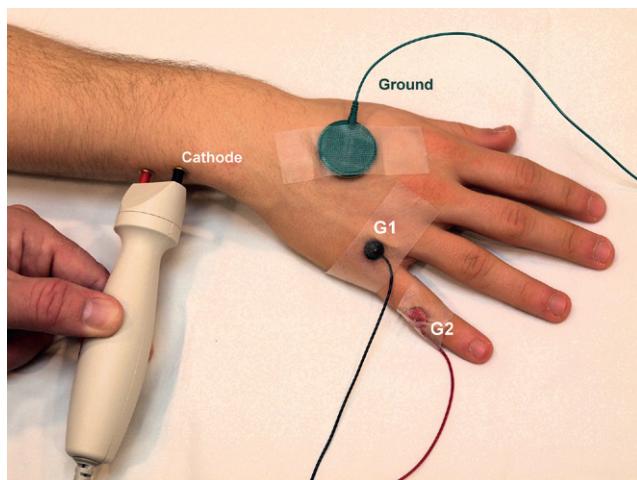
very low or absent. Averaging is frequently required, and identification of the onset latency of these very small potentials is often difficult. These techniques may be best suited for mild cases of UNE, in which the distal sensory and mixed nerve potentials are of relatively normal amplitude. It is important to emphasize that when performing these studies, one is looking for focal slowing across the elbow and not for conduction block. Because of the normal drop in amplitude of sensory and mixed nerve fibers over distance, separating conduction block from normal temporal dispersion and phase cancellation may be very difficult (unless the stimulation sites are very close, such as the 1 cm increments used in inching across the carpal tunnel).

#### Dorsal Ulnar Cutaneous Sensory Study

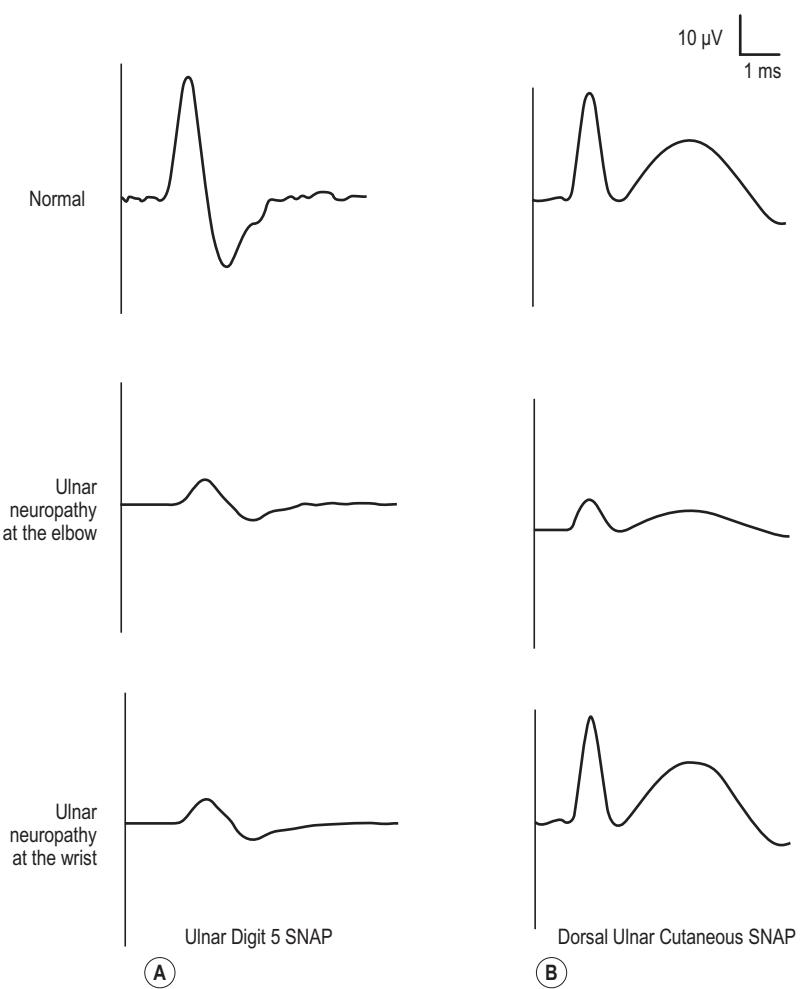
Recording the dorsal ulnar cutaneous sensory nerve can be a useful technique to perform in patients with suspected ulnar neuropathy. The dorsal ulnar cutaneous sensory nerve arises 5 to 8 cm proximal to the wrist to supply sensation over the dorsal medial hand, as well as the dorsal fifth and medial fourth digits. The dorsal ulnar SNAP can be recorded by placing recording electrodes in the dorsal web space between the fifth and fourth digits and stimulating 8 to 10 cm proximally just below the ulnar styloid with the hand placed in a pronated position (Figure 19–17). Usually, the potential can be recorded using a small stimulating current (e.g., 5–15 mA). A normal antidromic response is



**FIGURE 19–16** Ulnar sensory conduction studies: normal responses. An antidromic ulnar sensory nerve action potential can be recorded over the fifth digit, stimulating the wrist and below and above the elbow. Normal sensory responses decrease markedly in amplitude and area, whereas their duration increases at more proximal stimulation sites because of the normal processes of temporal dispersion and phase cancellation. For this reason, proximal demyelinating lesions in sensory fibers can be detected only by conduction velocity slowing and not by drop in amplitude or area.



**FIGURE 19–17** Dorsal ulnar cutaneous sensory study. With the hand in a pronated position, recording electrodes are placed in the dorsal web space between the fourth and fifth fingers. The stimulation site is just below the ulnar styloid, 8 to 10 cm proximal to the recording electrodes.



**FIGURE 19–18** Ulnar sensory response patterns. The typical patterns of the routine ulnar digit 5 (**A**) and the dorsal ulnar cutaneous sensory nerve action potential (SNAP) responses (**B**) in ulnar neuropathy at the elbow and at the wrist. Both assume lesions that involve axonal loss and that there is no anomalous innervation of the dorsal ulnar cutaneous sensory nerve. However, significant exceptions to these classic patterns may occur: (1) if the ulnar neuropathy at the elbow is purely demyelinating or (2) if there is an ulnar neuropathy at the wrist that spares the sensory branch. In both of these situations, both ulnar SNAPs may be normal. In addition, some cases of mild axonal loss ulnar neuropathy at the elbow may spare the dorsal ulnar cutaneous sensory response and thus display the pattern common to ulnar neuropathy at the wrist, sparing the dorsal ulnar cutaneous SNAP.

greater than 8  $\mu$ V, but, as in other uncommonly performed sensory nerve conduction studies, comparison with the contralateral asymptomatic side frequently is helpful. Any potential that is less than 50% that of the contralateral asymptomatic side likely is abnormal.\*

With knowledge of the anatomy of both the routine digit 5 ulnar SNAP and the dorsal ulnar SNAP, one can predict the expected patterns of abnormalities in lesions of the

ulnar nerve at the elbow and at the wrist (Figure 19–18). In patients with UNE, one would expect both SNAPs to be abnormal, provided there has been axonal loss. If the lesion is pure demyelination at the elbow, both distal sensory responses will be normal. Conversely, the presence of a normal dorsal ulnar SNAP with an abnormal digit 5 ulnar sensory response suggests that the lesion is at the wrist (the dorsal ulnar branch arises proximal to the wrist). Nevertheless, this latter pattern does not completely exclude the possibility of UNE. In some patients with definite UNE and axonal loss, the dorsal ulnar cutaneous SNAP is spared. This finding is thought to be due to preferential fascicular sparing of the dorsal ulnar cutaneous sensory fibers at the elbow. Studies of the microanatomy of the fascicle that forms the dorsal ulnar cutaneous sensory branch have shown that it commonly separates from the main ulnar trunk above the elbow and effectively travels as

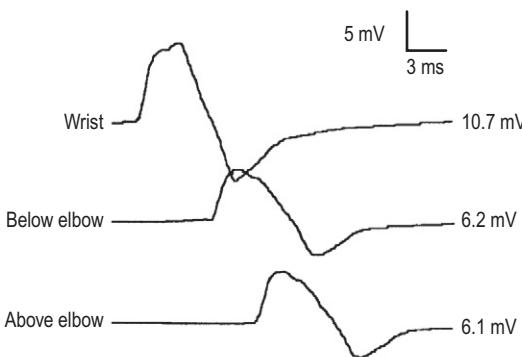
\*Remember that very rarely there is an anomalous innervation wherein the superficial radial sensory nerve supplies the entire dorsum of the hand, including the usual territory of the dorsal ulnar cutaneous sensory nerve. Thus, in cases where the dorsal ulnar cutaneous sensory response is absent, it is prudent to stimulate the superficial radial sensory nerve along the lateral radius with the recording electrodes in place for the dorsal ulnar cutaneous sensory study to ensure that this very rare anomalous innervation is not present.

a separate nerve next to the ulnar nerve in the forearm. Therefore, care must be taken in interpreting the findings of an abnormal digit 5 ulnar SNAP and a normal dorsal ulnar cutaneous SNAP. This pattern has limitations as a diagnostic marker and cannot be used alone to reliably localize the site of the lesion to the wrist. Electrophysiologic measurement of the dorsal ulnar SNAP is useful, but only in those cases where it is abnormal, implying localization of the lesion proximal to the wrist. *To summarize, although an abnormal dorsal ulnar cutaneous SNAP indicates that the lesion is proximal to the wrist, the converse is not necessarily true.*

### Nerve Conduction Study Pitfalls

When performing routine ulnar nerve conduction studies, one must keep in mind several important technical factors:

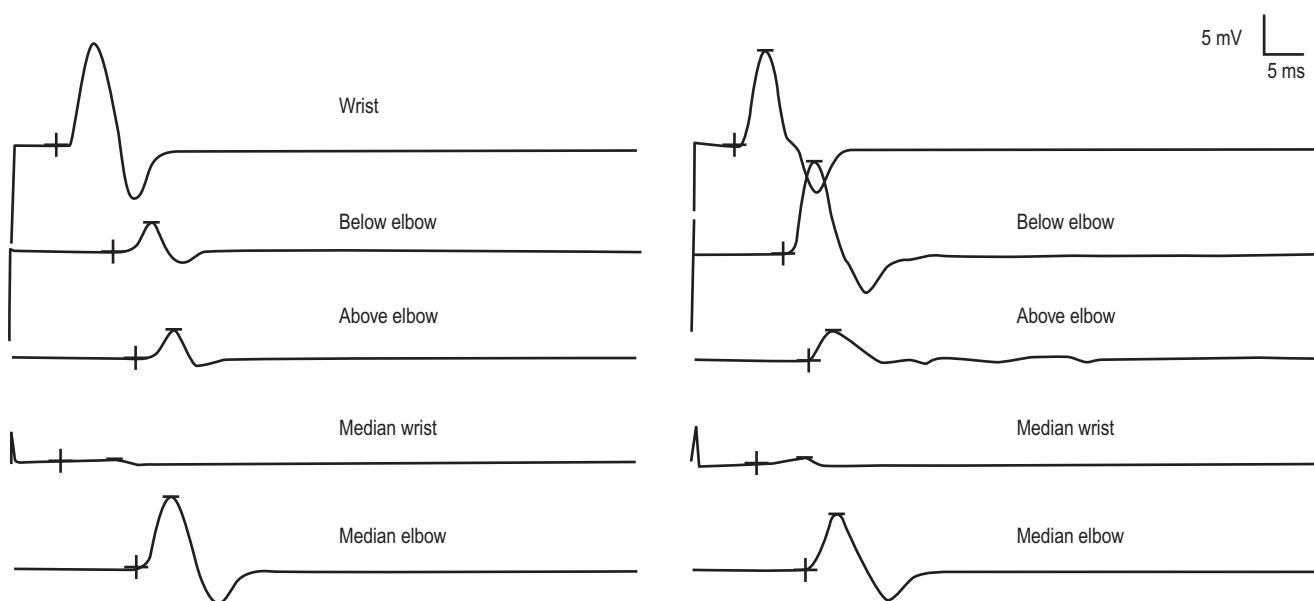
1. When stimulating at the below-elbow site, the stimulator must be located 3 cm distal to the groove to ensure that the stimulation point is distal to the cubital tunnel.
2. Care must be taken not to stimulate too distally at the below-elbow site. The mistaken impression of UNE may occur if the below-elbow stimulation site is too distal and the patient has a coexistent high Martin–Gruber anastomosis (see Chapter 7). In this situation, one could see a drop in amplitude between the below-elbow and above-elbow stimulation sites. Thus, the optimal position to stimulate the below-elbow site is 3 cm distal to the medial epicondyle, not less, not more. In addition, as the nerve runs deep under the FCU, higher current is required at the below-elbow site, and supramaximal stimulation may be difficult to achieve.
3. The distance from the below-elbow site to the above-elbow site ideally should be 10 cm. If shorter distances are used, slight errors in measurement may create large differences in calculated conduction velocities. If longer distances are used, a longer length of normal nerve may dilute any conduction velocity slowing across the elbow, yielding normal or equivocal results.
4. The examiner should be cautious of any apparent conduction block between the wrist and below-elbow sites (Figure 19–19). Although there are very rare ulnar lesions in the forearm (e.g., exit of the cubital tunnel, fibrovascular bands in the forearm), this finding usually indicates a Martin–Gruber anastomosis, which is a normal finding. In these situations, stimulating the median nerve at the wrist and antecubital fossa, while recording the ulnar muscle (ADM or FDI), is essential to demonstrate that an anastomosis is present.
5. As noted above, a Martin–Gruber anastomosis is usually recognized on routine ulnar motor nerve conduction studies as a drop of amplitude and area between the wrist and below-elbow stimulation sites. However, there are rare reports of a very proximal



**FIGURE 19–19** Martin–Gruber anastomosis mimicking conduction block of the ulnar nerve in the forearm. The most common cause of drop in amplitude between the wrist and below-elbow sites during routine ulnar motor conduction studies is not true conduction block but a Martin–Gruber anastomosis. Conduction block of the ulnar nerve in the forearm should never be diagnosed unless the median nerve has been stimulated at the wrist and antecubital fossa to exclude a Martin–Gruber anastomosis.

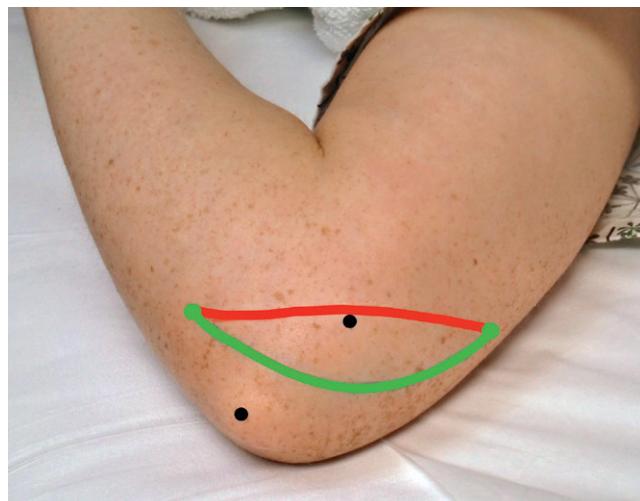
MGA wherein the drop in amplitude and area occurs between the below-elbow and above-elbow stimulation sites (i.e., across the elbow), mimicking a conduction block across the elbow. The optimal site for the below-elbow stimulation site is 3 cm distal to the medial epicondyle. It should be more than 2 cm in order to be distal to the cubital tunnel, and less than 4 cm to avoid the nerve being so deep that it is difficult to stimulate. Although almost all MGAs occur in the forearm and do not interfere with electrodiagnostic determination of conduction block at the elbow, some reports have found an MGA to be as proximal as 3 cm distal to the medial epicondyle (Figure 19–20). Thus, in this very unusual situation, one could confuse an MGA (a normal finding) with an ulnar conduction block across the elbow. This underscores that the correct site to stimulate the ulnar nerve below the elbow is 3 cm distal to the medial epicondyle. In all cases where an ulnar conduction block is found across the elbow, it is prudent to check for an MGA (by stimulating the median nerve at the wrist and antecubital fossa, and recording the ulnar muscle).

6. Rarely, flexing the elbow causes the ulnar nerve to sublux out of the groove medially over the medial epicondyle. Recurrent subluxation of the ulnar nerve has been a suggested cause of repetitive ulnar trauma and UNE. Thus, in a situation where a patient does have UNE and a subluxed nerve when in the elbow flexed position, the measured distance around the groove will actually overestimate the length of the nerve, resulting in a spuriously fast conduction velocity across the elbow (see Figure 19–21). In one recent study of normal individuals with a subluxed ulnar nerve at the elbow, the average change in distance across the elbow segment was overestimated by 1.6 cm (range, 0.6 to 2.5 cm), which equated to



**FIGURE 19-20** Martin–Gruber anastomosis: “conduction block” patterns in the forearm and across the elbow. **Left:** Recording from the first dorsal interosseous, there is a marked drop in amplitude between the wrist and below-elbow stimulation sites, suggesting a conduction block in the forearm. However, stimulating the median nerve at the wrist and antecubital fossa (elbow) while recording from the FDI confirmed an MGA, as a much larger response was present at the antecubital fossa than at the wrist. This is the common presentation of an MGA during routine ulnar motor conduction studies. **Right:** Recording from the first dorsal interosseous, there is a marked drop in amplitude between the below-elbow and above-elbow stimulation sites, which was interpreted as a conduction block across the elbow (i.e., UNE). However, stimulating the median nerve at the wrist and antecubital fossa (elbow) while recording from the FDI also demonstrated an MGA in this case. By short segment “inching studies,” the site of the MGA was found to be 3 cm distal to the retrocondylar groove. This finding of an MGA mimicking an ulnar conduction block across the elbow is exceedingly rare.

(With permission from Leis, A.A., Stetkarova, I., Wells, K.J., 2010. Martin–Gruber anastomosis with anomalous superficial radial innervation to ulnar dorsum of hand: a pitfall when common variants coexist. *Muscle Nerve* 41, 313–317.)



**FIGURE 19-21** Ulnar nerve location at the elbow. In the vast majority of individuals, the ulnar nerve runs in a curved pattern (green line) through the ulnar groove between the medial epicondyle (upper black dot) and the olecranon (lower black dot). However, in some individuals, when the elbow is flexed the ulnar nerve will sublux out of the groove medially over the medial epicondyle (red line). In these cases, the measured distance (green line) will overestimate the true distance (red line), which then results in an overestimate of the ulnar conduction velocity across the elbow (i.e., the conduction velocity will be spuriously fast). Accordingly, in some cases of true UNE, this could result in a false-negative study.

an overestimated ulnar conduction velocity across the elbow an average of 7.9 m/s (range, 3.0 to 13.0 m/s). One can see how this could result in a false-negative study in a person with UNE, if the conduction velocity across the elbow were overestimated, and did not meet the cutoff criteria for absolute and differential focal slowing across the elbow. It is important to emphasize that a subluxed ulnar nerve interferes with determination of the true conduction velocity across the elbow, but not conduction block across the elbow. These cases emphasize the value of SSIS (“inching”) in suspected UNE, as this technique maps out the exact location of the ulnar nerve, and is able to detect the precise area of slowing.

### Electromyographic Approach

The strategy in the EMG examination of UNE is directed toward identifying denervation or reinnervation limited to the ulnar-innervated muscles of the hand and forearm (**Box 19-2**). Useful muscles to check are the FDI, FDP (to digit 4 or 5), and FCU. Of the intrinsic ulnar hand muscles, testing of the FDI is usually tolerated best by patients. Testing of the ADM often is perceived as more painful, similar to the abductor pollicis brevis (APB). Median- and radial-innervated C8 muscles are screened to rule out

**Box 19–2. Recommended Electromyographic Protocol for Ulnar Neuropathy at the Elbow**
*Routine muscles:*

1. Ulnar muscle distal to the wrist (first dorsal interosseous or abductor digiti minimi)
2. Ulnar muscles in the forearm (FDP 5 and flexor carpi ulnaris)

*If any of the ulnar muscles are abnormal, test the following additional muscles:*

1. At least two non-ulnar lower trunk/C8–T1-innervated muscles (e.g., abductor pollicis brevis, flexor pollicis longus, extensor indicis proprius) to exclude a lower brachial plexopathy, polyneuropathy, or C8–T1 radiculopathy
2. C8 and T1 paraspinals

*Special considerations:*

- If the ulnar neuropathy is superimposed on another condition (e.g., polyneuropathy, plexopathy, radiculopathy), a more detailed electromyographic examination will be required.
- The abductor digiti minimi frequently is painful and more difficult for some patients to tolerate than the first dorsal interosseous.
- In ulnar neuropathy at the elbow, the flexor carpi ulnaris may be spared even when the FDP 5 is abnormal.
- If no evidence of ulnar neuropathy is found on nerve conduction studies, electromyographic study should focus on evaluation for lower trunk brachial plexopathy or C8–T1 radiculopathy if clinically indicated.

FDP, flexor digitorum profundus.

evidence of a C8 radiculopathy or lower trunk brachial plexopathy. Useful muscles to check are the APB, flexor pollicis longus, and extensor indicis proprius. If a cervical radiculopathy is suggested based on clinical history, then sampling the cervical paraspinal muscles is also indicated.

Of interest, the FCU is either normal or minimally affected in many surgically proven cases of UNE. Overall, involvement of the FCU correlates with the severity of the ulnar neuropathy both clinically and electrically. The finding of FCU involvement is slightly more common in lesions at the groove than at the cubital tunnel. Although sparing of the FCU was classically thought to be due to the muscular branch to the FCU arising proximal to the groove, cadaver anatomic studies have found this not to be the case. The true etiology of FCU sparing likely is related to either *differential fascicular susceptibility* (i.e., different fascicles are involved or spared depending on their position within the nerve trunk in relationship to the compression site) or the “dying back” concept of nerve lesions (i.e., the most distal muscles are maximally affected and the proximal ones are relatively preserved). This EMG pattern of an abnormal FDI and FDP to digits 4 and 5, but a spared FCU, is important to recognize in patients with UNE.

Unfortunately, there is no ulnar-innervated muscle above the elbow. If all ulnar-innervated muscles, including the FCU, are abnormal and the nerve conduction studies do not identify any focal slowing or conduction block around the elbow, the best electrophysiologic diagnosis that can be

reached is one of an ulnar neuropathy that can only be localized at or proximal to the takeoff to the FCU muscle. Although most such cases will in fact be cases of UNE, the electrophysiologic examination cannot exclude an unusual ulnar neuropathy in the proximal arm or a lower brachial plexopathy that selectively involves ulnar fibers. Examination of the medial antebrachial cutaneous sensory nerve, which comes directly off the medial cord of the brachial plexus, may help identify a lower brachial plexus lesion.



## EXAMPLE CASES

### Case 19–1

#### History and Physical Examination

A 44-year-old man was referred for numbness and pain in his right arm and hand. The patient described several months of numbness involving his right fourth and fifth digits, accompanied by pain in the right elbow, shoulder, and neck. Examination showed hypesthesia in the right ulnar distribution. There was slight weakness of all intrinsic hand muscles. Reflexes were normal. There was no tenderness of the ulnar nerve in the groove.

#### Summary

The clinical history and examination suggest dysfunction in the ulnar nerve distribution. The fourth and fifth fingers are innervated by the ulnar nerve, as are most of the intrinsic hand muscles, which are described as slightly weak. There is nothing further in the history or examination to suggest a more specific localization. Indeed, the patient has some pain in the elbow, shoulder, and neck but no tenderness in the ulnar groove. This complicates the case further because the differential diagnosis of dysfunction of the ulnar nerve includes ulnar neuropathy at the wrist, UNE, a lower brachial plexopathy, and a lesion of the C8–T1 nerve roots. Occasionally, patients with UNE have pain that radiates more proximally into the arm and shoulder but not into the neck. Pain into the neck associated with more distal numbness and weakness usually suggests a cervical radiculopathy.

The nerve conduction studies begin with a routine median motor conduction study recording the APB. Although the CMAP amplitude is normal with a normal conduction velocity, the distal motor latency is slightly prolonged. This mild prolongation of distal latency may be due to a median nerve lesion at the wrist. However, it may also simply suggest loss of the fastest conducting axons from any axonal loss lesion involving the median-innervated C8–T1 fibers from the anterior horn cells in the spinal cord on down. Further studies will be required to make this distinction. The routine ulnar motor conduction study recording the ADM muscle shows a marked decrease in motor amplitude but with a normal distal latency. The conduction velocity in the forearm is normal, but the conduction around the elbow is markedly reduced at 34 m/s. Correspondingly, the F responses are prolonged, compared both with normal controls and with

CASE 19–1. Nerve Conduction Studies														
Nerve Stimulated	Stimulation Site	Recording Site	Amplitude Motor = mV; Sensory = $\mu$ V			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	6.4		$\geq 4$	4.6		$\leq 4.4$				31		$\leq 31$
Median (m)	Antecubital fossa	APB	6.0			8.3			55		$\geq 49$			
Ulnar (m)	Wrist	ADM	2.9		$\geq 6$	3.2		$\leq 3.3$				34		$\leq 32$
Ulnar (m)	Below elbow	ADM	2.7			6.5			58		$\geq 49$			
Ulnar (m)	Above elbow	ADM	2.4			9.4			34		$\geq 49$			
Median (s)	Wrist	Index finger	27		$\geq 20$	3.2		$\leq 3.5$	58		$\geq 50$			
Ulnar (s)	Wrist	Little finger	7	22	$\geq 17$	3.1	3.0	$\leq 3.1$	49	53	$\geq 50$			
Radial (s)	Forearm	Snuffbox	28		$\geq 15$	2.5		$\leq 2.9$	55		$\geq 50$			
Median (mixed study)	Palm	Wrist	50		$\geq 50$	2.2		$\leq 2.2$	50		$\geq 50$			
Ulnar (mixed study)	Palm	Wrist	4		$\geq 12$	2.3		$\leq 2.2$	49		$\geq 50$			
Mixed difference						-0.1		$\leq 0.3$						

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 19–1. Electromyography									
Muscle	Insertional Activity	Spontaneous Activity			Voluntary Motor Unit Action Potentials				
		Fibrillation potentials	Fasciculations	Activation	Recruitment	Duration	Amplitude	Configuration	Polyphasia
Right FDI	↑	+3	0	NL	↓↓	+2	+2		+2
Right APB	NL	0	0	NL	↓	+1	+1		NL
Right extensor indicis proprius	↑	0	0	NL	↓↓	+1	+1		+1
Right FCU	NL	0	0	NL	↓	+1	+1		NL
Right FDP 5	NL	0	0	NL	↓	+1	+1		NL
Right biceps brachii	NL	0	0	NL	NL	NL	NL		NL
Right pronator teres	NL	0	0	NL	NL	NL	NL		NL
Right triceps brachii	NL	0	0	NL	↓	NL	NL		NL/+1
Right C7 paraspinal	↑	0	0	NL	NL	NL	NL		NL
Right C8 paraspinal	↑	+2	0	NL	NL	NL	+1		+1
Right T1 paraspinal	NL	0	0	NL	NL	NL	NL		NL

↑ = increased; ↓ = slightly reduced; ↓↓ = moderately reduced; NL = normal; FDI = first dorsal interosseous; APB = abductor pollicis brevis; FCU = flexor carpi ulnaris; FDP = flexor digitorum profundus.

the median nerve. This study of the ulnar nerve clearly demonstrates a UNE: there is a demyelinating conduction velocity in the segment across the elbow. There is also clear differential slowing (24 m/s) when the forearm segment is compared with the across-elbow segment. Any slowing of more than 10 to 11 m/s in the flexed

elbow position denotes focal slowing. The ulnar F response is prolonged; the reason is similar to why the median F response is prolonged in cases of CTS. The F response must travel antidromically through the ulnar lesion at the elbow, then to the anterior horn cells, and then back down the arm, again through the ulnar lesion,

and finally distally to the ADM muscle. Because the F response must travel twice through the area of demyelination, prolonged, dispersed, or absent F responses are not unusual in UNE.

Next, the routine median sensory study is performed, which is normal including the distal peak latency of 3.2 ms. Thus, there is not good evidence at this time that the prolonged median distal motor latency is due to a median neuropathy at the wrist. Finding a normal median sensory latency with slowed median motor fibers is extraordinarily unusual in CTS. The routine ulnar study shows a low SNAP on the involved right side, with a normal contralateral potential. There is already a good explanation for the abnormal ulnar SNAP: the motor studies have already clearly defined a UNE. The low ulnar sensory amplitude signifies that there has been secondary axonal loss in the ulnar sensory fibers, as was also found in the ulnar motor fibers. The normal radial sensory response reinforces the evidence that there is not a more widespread polyneuropathy or brachial plexopathy. Finally, the median and ulnar palm-to-wrist mixed nerve studies are performed, showing peak latencies of 2.2 and 2.3 ms, respectively, with a low ulnar amplitude. Although the ulnar latency is slightly prolonged compared to normal, there is no significant asymmetry between the two latencies, and there is no electrophysiologic evidence for a median neuropathy across the wrist. The prolonged median distal motor latency to the APB still has not been adequately explained. With normal median sensory and palm-to-wrist mixed nerve responses, a median neuropathy at the wrist is unlikely.

The EMG shows prominent denervation and reinnervation with decreased recruitment in the FDI, consistent with the previously defined UNE. Although the APB does not show any denervation, it does show reduced recruitment with some reinnervation of motor unit action potentials (MUAPs). This finding correlates well with the mild prolongation of the median distal motor latency, suggesting that there has been some loss of the fastest conducting axons. One might ask why the median motor CMAP amplitude is normal when the EMG study shows evidence of axonal loss. CMAP amplitudes can remain in the normal range for one of two reasons. First, if reinnervation has been sufficient, the CMAP amplitude may remain normal. Second, the CMAP amplitude is only in the “normal range.” There is a wide range of normal values. In this particular case, the patient’s CMAP amplitude may have been greater initially and then decreased, still remaining within the “normal range.”

The reason for the reinnervation in the APB remains unclear. There must be a second problem beyond the UNE. Moving on, the extensor indicis proprius (also a C8-innervated muscle) not only shows increased insertion activity, but also dramatic changes in recruitment and MUAP size. At this point, the additional abnormalities in the APB and extensor indicis proprius suggest that there is either a lesion distally (because both are distal muscles), such as a polyneuropathy, or that there is a

superimposed lower brachial plexus lesion or cervical radiculopathy at the C8 level. The normal median and radial sensory amplitudes from the nerve conduction studies effectively exclude a polyneuropathy. These EMG abnormalities possibly represent a superimposed lesion of the lower brachial plexus or C8 nerve root. Because the extensor indicis proprius (a radial, posterior cord-innervated muscle) is abnormal, a medial cord lesion is excluded. The additional lesion must be at the level of the lower trunk of the brachial plexus or located more proximally. Further EMG demonstrates that both the FCU and the FDP to digit 5 show decreased recruitment and mild reinnervation, consistent with both UNE (as demonstrated from the nerve conduction studies) and the superimposed lesion that is being investigated. Examination of the biceps and pronator teres shows normal results, suggesting that there is not a more widespread lesion affecting the C6–C7 nerve roots or the upper and middle trunks of the brachial plexus. The triceps brachii also is slightly abnormal, but the triceps, a C6–C7–C8-innervated muscle, also runs through the middle and lower trunks of the brachial plexus. Finally, the paraspinal muscles are extremely informative, showing frank fibrillation potentials at the C8 paraspinals. This finding unequivocally demonstrates that the additional lesion is at or proximal to the root level, although the myotomal level of the lesion is best determined by the limb muscles.

Therefore, at this time we can form an electrophysiologic impression.

**IMPRESSION:** *There is electrophysiologic evidence of an ulnar neuropathy at the elbow with a superimposed C8 radiculopathy.*

Several questions deserve consideration.

#### *Does the Clinical–Electromyographic Correlation Make Sense?*

The important findings in this case are the unequivocal nerve conduction abnormalities of the ulnar nerve around the elbow, demonstrating a lesion at that site, and the presence on EMG of clear neuropathic changes in several C8-innervated muscles beyond the ulnar-innervated muscles, including the paraspinal muscles. The finding of paraspinal denervation signifies that the lesion is at or proximal to the root level (root or motor neuron). Thinking back to the clinical history and examination, we now have a better explanation for the patient’s pain not only in the shoulder and elbow, which may be seen with UNE, but also in the neck, which is a consequence of the radiculopathy. The radiculopathy also explains the slight weakness of all intrinsic hand muscles, some of which are median and some of which are ulnar innervated. Some would call this a *double crush syndrome*, in that ulnar fibers have been affected at two lesion sites. Whether or not these two lesions are causally related remains controversial. The important point is that detailed nerve conduction and EMG studies are required to sort out that

there are, in fact, two superimposed lesions. One can easily see that if the nerve conduction studies had been limited to ulnar nerve conductions and the EMG studies had been limited to ulnar-innervated muscles and the APB, the cervical radiculopathy could easily have been missed, as one might have diagnosed only a UNE and possibly (inaccurately) a superimposed median neuropathy at the wrist. As noted earlier, however, the normal median sensory response and normal median palmar mixed nerve response effectively exclude the diagnosis of median neuropathy at the wrist.

#### *Why is the Median Distal Motor Latency Prolonged if there is no Carpal Tunnel Syndrome?*

The mild prolongation of the distal median motor latency is consistent with dropout of the fastest conducting axons. Once the study is completed, there is an adequate explanation for this finding. Some of the fastest conducting axons to the APB muscle have been lost because of the C8 radiculopathy. The mild distal latency prolongation does not indicate unequivocal demyelination but is only in the range of axonal loss. The needle EMG shows clear evidence of reinnervation in the APB, indicating some prior axonal loss.

## Case 19–2

### *History and Physical Examination*

A 53-year-old right-handed man developed numbness over the fourth and fifth digits of the right hand. There was no history of elbow trauma. Subsequently, he developed weakness of grip and loss of dexterity.

Examination demonstrated a Tinel's sign over the right elbow and mild wasting of the ulnar intrinsic hand muscles, along with weakness of distal flexion of the little finger. Sensory loss was present in the fifth and splitting the fourth finger, and it extended just proximal to the distal wrist crease.

### **Summary**

The history and examination in this case are similar to those in Case 19–1. There appear to be clear abnormalities in the distribution of the ulnar nerve. Again, the numbness of the fourth and fifth fingers is in the distribution of the ulnar nerve, along with weakness of grip and loss of dexterity, which can easily be explained by weakness of ulnar-innervated intrinsic hand muscles. The examination demonstrates a Tinel's sign over the right elbow, suggesting that the lesion may be at that site. One must always be cautious in interpreting Tinel's signs, however, because they occur in some normal subjects. The sensory loss appears to be quite consistent with ulnar neuropathy.

The first nerve conductions demonstrate normal median CMAP amplitudes, latencies, conduction velocities, and F responses. When the ulnar study is performed, however, a low CMAP amplitude is seen on the involved right side, with a normal amplitude on the contralateral side. The distal latency on the involved side is just at the upper limit of normal. Notably, there is slight slowing around the elbow compared to the forearm velocity, but the conduction velocity does not slow by 10 to 11 m/s compared to the forearm segment, nor is it slowed in an

CASE 19–2. Nerve Conduction Studies

Nerve Stimulated	Stimulation Site	Recording Site	Amplitude Motor = mV; Sensory = $\mu$ V						Conduction Velocity (m/s)			F Wave Latency (ms)		
			RT	LT	NL	RT	LT	NL	RT	LT	NL	RT	LT	NL
Median (m)	Wrist	APB	5.8	8.2	≥4	3.3	3.3	≤4.4				25	26	≤31
Median (m)	Antecubital fossa	APB	5.4	7.1		7.2	7.0		51	53	≥49			
Ulnar (m)	Wrist	ADM	3.2	8.2	≥6	3.3	3.1	≤3.3				31	28	≤32
Ulnar (m)	Below elbow	ADM	2.9	8.1		6.4	6.0		65	68	≥49			
Ulnar (m)	Above elbow	ADM	2.8	8.1		8.0	7.5		61	66	≥49			
Median (s)	Wrist	Index finger	21	23	≥20	3.1	3.1	≤3.5	54	56	≥50			
Ulnar (s)	Wrist	Little finger	NR	20	≥17	NR	2.7	≤3.1	NR	55	≥50			
Dorsal ulnar (s)	Wrist	Dorsal medial hand	NR	10	≥8	NR	3.0	≤2.8	NR	57	≥50			
Median (mixed study)	Palm	Wrist	52		≥50	2.0		≤2.2	53		≥50			
Ulnar (mixed study)	Palm	Wrist	NR		≥12	NR		≤2.2	NR		≥50			

m = motor study; s = sensory study; RT = right; LT = left; NL = normal; NR = no response; 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 19–2. Electromyography									
Muscle	Insertional Activity	Spontaneous Activity		Voluntary Motor Unit Action Potentials					
		Fibrillation Potentials	Fasciculations	Activation	Recruitment	Duration	Amplitude	Polyphasia	
Right FDI	↑	+2	0	NL	↓↓	1+	NL	NL	
Right ADM	NL	+1	0	NL	↓	1+	+1	+1	
Right APB	NL	0	0	NL	NL	NL	NL	NL	
Right extensor indicis proprius	NL	0	0	NL	NL	NL	NL	NL	
Right FCU	NL	0	0	NL	NL	NL	NL	NL	
Right FDP 4,5	↑	+1	0	NL	NL	+1	+1	+1	
Right biceps brachii	NL	0	0	NL	NL	NL	NL	NL	
Right flexor pollicis longus	NL	0	0	NL	NL	NL	NL	NL	
Right triceps brachii	NL	0	0	NL	NL	NL	NL	NL	
Right medial deltoid	NL	0	0	NL	NL	NL	NL	NL	
Right C7 paraspinal	NL	0	0	NL	NL	NL	NL	NL	
Right C8 paraspinal	NL	0	0	NL	NL	NL	NL	NL	
Right T1 paraspinal	NL	0	0	NL	NL	NL	NL	NL	

↑= increased; ↓ = slightly reduced; ↓↓= moderately reduced; NL = normal; FDI = first dorsal interosseous; ADM = abductor digiti minimi; APB = abductor pollicis brevis; FCU = flexor carpi ulnaris; FDP = flexor digitorum profundus.

absolute sense in the demyelinating range (<35 m/s). In addition, there is no evidence of conduction block across the elbow. The ulnar F response, however, is prolonged in comparison to the median F response.

The sensory studies are performed next. The median sensory amplitudes are normal bilaterally, as are the latencies and conduction velocities. The ulnar sensory study demonstrates an absent response on the involved right side and a normal contralateral response. Therefore, at this point in the study, it is clear that there is involvement of ulnar motor and sensory fibers. *However, there is no localizing electrophysiology.* The only localizing information comes from the absence of the sensory response, which denotes a postganglionic lesion. This finding is not consistent with a cervical radiculopathy.

To look for evidence of ulnar neuropathy at the wrist, some additional studies are performed. The dorsal ulnar cutaneous study is performed, and the response is absent on the involved side, whereas a normal response is found on the contralateral side. Because the dorsal ulnar cutaneous sensory nerve originates several centimeters proximal to the wrist, an ulnar nerve lesion at the wrist has effectively been ruled out. The lesion affecting the ulnar nerve remains non-localizable, but it must be above the level of the wrist. The EMG study will be needed to localize the lesion further.

The EMG study shows clear denervation and reinnervation in the FDI, ADM, and FDP to digits 4 and 5, all ulnar-innervated muscles. The FCU is normal. Next,

non-ulnar C8-T1-innervated muscles must be sampled. The APB, flexor pollicis longus, and extensor indicis proprius are sampled and are normal. The lack of abnormalities in these muscles makes a lesion of the C8-T1 nerve roots or lower trunk of the brachial plexus much less likely. Finally, the lower cervical paraspinal muscles are sampled and are normal.

At this time we are ready to form our electrophysiologic impression.

**IMPRESSION:** *There is electrophysiologic evidence consistent with an ulnar neuropathy at or above the takeoff to the FDP muscle.*

Several questions should be considered.

#### *Can the Ulnar Neuropathy be More Precisely Localized?*

One may ask whether the ulnar nerve lesion can be localized further using the information at hand. For instance, the FDP is abnormal whereas the FCU is normal. Does not that localize the lesion to between those two sites? Unfortunately, the answer is no. In nerve lesions of any kind, the distal muscles tend to be the most affected. In addition, especially in entrapment neuropathy, certain fascicles are often relatively spared while others are preferentially involved.

Demonstrating an ulnar neuropathy (which most likely is localized at the elbow) whose electrophysiology does

not clearly localize it at the elbow often is frustrating for the electromyographer. In this case, a lesion at the wrist has been excluded by the abnormal dorsal ulnar cutaneous sensory response. Unusual lesions in the distal forearm have also been excluded because the FDP is abnormal. The lesion must be at or above that site. Often, in UNE, the branch to the FCU is spared. This finding can be seen in ulnar neuropathy at both the groove and the cubital tunnel.

#### *What Other Studies Might Localize the Lesion Further?*

One might consider performing inching studies across the elbow. The procedure is technically demanding, but it clearly increases the diagnostic sensitivity of the electrophysiologic examination. Stimulating the ulnar nerve at the elbow in 1 cm increments from above to below the elbow (looking for either an abrupt decrease in amplitude or an abrupt increase in latency) may be very useful in localizing the lesion at the elbow.

Another possibility would be to repeat the ulnar motor study but recording from the FDI muscle. Sometimes, focal slowing or conduction block may be identified when recording the FDI, even in cases when recording the ADM muscle is normal. One also might consider performing either sensory or mixed-nerve conduction studies across the elbow. Although sensory and mixed-nerve conduction studies are more sensitive than motor conduction studies, when the sensory nerve action potential is low or absent with wrist stimulation, as in this case, usually one can expect absent potentials at the more proximal sites above and below the elbow. These studies are best reserved for the patient who clinically demonstrates a clear ulnar neuropathy and whose distal sensory potentials are relatively intact.

To localize a lesion by nerve conduction studies requires demonstrating demyelination, either focal slowing or conduction block. Unfortunately, the pathophysiology of UNE is often axonal loss alone. In that case, the routine as well as the additional nerve conduction studies often fail to localize the lesion.

The abnormal ulnar SNAP signifies that the lesion is at or distal to the dorsal root ganglion. The abnormal EMG findings in the flexor digitorum profundus signify that the lesion is at or proximal to that muscle. The lesion is somewhere between those two sites but unfortunately cannot be localized further based on the present study. The electrophysiologic study cannot completely exclude the possibility that the ulnar nerve lesion is in the higher arm in an unusual location or in the lower part of the brachial plexus, either the lower trunk or the medial cord. The medial antebrachial cutaneous sensory nerve could be studied if there is a clinical suspicion of a lower brachial plexopathy.

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# Ulnar Neuropathy at the Wrist

# 20

Ulnar neuropathy at the wrist (UNW) is a rare condition that sometimes is confused with ulnar neuropathy at the elbow (UNE) or, more often, with early motor neuron disease. Knowledge of the detailed anatomy of the ulnar nerve at the wrist is necessary to understand the several unique clinical and electrophysiologic patterns that can occur with UNW (Figure 20-1).

## ANATOMY

At the wrist, the ulnar nerve enters *Guyon's canal* at the level of the distal wrist crease. The canal is formed proximally by the pisiform bone and distally by the hook of the hamate. The floor is formed by a combination of the thick transverse carpal ligament and the adjacent hamate and triquetrum bones. The roof is loosely formed. In contrast, there is a thick band at the outlet that runs from the hook of the hamate to the pisiform bone, the pisohamate hiatus. In the canal, the nerve divides into *superficial* and *deep* branches. Before exiting through the pisohamate hiatus, motor fibers are given off the deep branch (also known as the *deep palmar motor branch*) to three of the four hypothenar muscles (abductor digiti minimi [ADM], flexor digiti minimi and opponens digiti minimi). After the hiatus, the superficial branch supplies sensation to the volar fifth and medial fourth digits, and also supplies motor innervation to the one remaining hypothenar muscle, the palmaris brevis. The deep palmar motor branch goes on to innervate the third and fourth lumbricals, the four dorsal and three palmar interossei, the adductor pollicis, and the deep head of the flexor pollicis brevis.

## CLINICAL

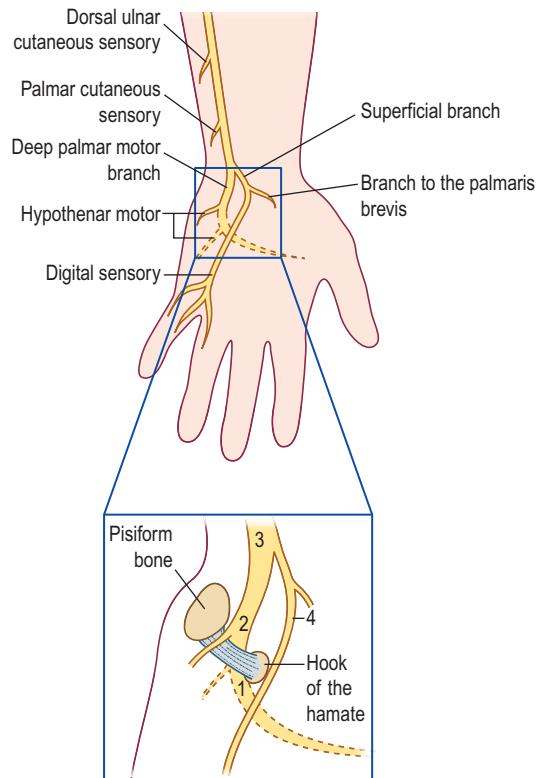
Several subtypes of UNW occur, depending on the exact location of the lesion and which fibers are affected (Table 20-1 and Box 20-1). The following lesions have been described:

- *Distal deep palmar motor lesion:* Affects all muscles supplied by the deep palmar motor branch except the hypothenar muscles; the superficial branch containing the sensory fibers and motor innervation to the palmaris brevis is not affected.

- *Proximal deep palmar motor lesion:* Affects all ulnar-innervated hand muscles, including the hypothenar muscles, with the exception of the palmaris brevis; the superficial branch containing the sensory fibers and motor innervation to the palmaris brevis is not affected.
- *Proximal canal lesion:* Affects all branches of the ulnar nerve, including the proximal and distal deep palmar motor and the superficial branches which contain the sensory fibers and motor innervation to the palmaris brevis.
- *Superficial branch lesion:* Affects only the superficial branch, which is primarily sensory. Note that while the palmaris brevis muscle is affected, this is not clinically apparent.

The first two patterns are the most common, accounting for more than 75% of all cases of UNW. In both, the superficial branch is not affected; thus, there are no sensory symptoms or sensory loss. Patients present with painless weakness and atrophy of ulnar intrinsic hand muscles. Because the ulnar-innervated adductor pollicis and deep head of the flexor pollicis brevis are in the thenar eminence, both the hypothenar and thenar eminences may be wasted, if the lesion is in the proximal deep palmar motor branch. Similar to UNE, the Benediction hand posture, Froment's sign, and Wartenberg's sign may be present in advanced cases. In addition, another somewhat obscure sign, known as the "palmaris brevis sign," may be seen in severe lesions of the deep palmar motor branch. Remember that the palmar brevis is the only muscle supplied by the superficial branch, and is therefore spared in lesions of the deep branch. When the palmaris brevis contracts, it results in puckering of the skin along the proximal medial border of the hand. Because the other intrinsic hand muscles are wasted, prominent contraction (and possibly hypertrophy) of the palmaris brevis may be seen when the fifth digit is forcibly contracted in the more common lesions of the deep palmar motor branch of the ulnar nerve at the wrist (the "palmaris brevis sign," Figure 20-2).

In more proximal lesions, the superficial branch will also be affected, leading to sensory disturbance of the volar fifth and medial fourth digits. *The dorsal medial aspect of the*



**FIGURE 20–1** Detailed anatomy of the ulnar nerve at the wrist. Entrapment of the ulnar nerve at the wrist can take on several patterns: (1) pure motor affecting only the distal deep palmar motor branch, distal to the hypothenar muscles, (2) pure motor affecting the proximal deep palmar branch including the hypothenar motor branches, (3) motor and sensory (proximal canal lesion), and rarely (4) sensory affecting the superficial branch involving the sensory fibers to the volar fourth and fifth fingers. The superficial branch does supply one muscle, the palmaris brevis. This muscle, however, is difficult to assess clinically or by EMG, although there are reports of the “palmaris brevis sign” wherein this muscle is spared or prominent in lesions of the deep palmar motor branch (see Figure 20–2).

(Adapted with permission from Olney, R.K., Hanson, M., 1988. AAEE case report no. 15: ulnar neuropathy at or distal to the wrist. Muscle Nerve 11, 828.)

#### Box 20–1. Clinical and Electrophysiologic Abnormalities not Consistent with Ulnar Neuropathy at the Wrist

##### Clinical

- Weakness of thumb abduction (*abductor pollicis brevis* – median innervated)
- Weakness of the finger flexors of digits 4 and 5 (*flexor digitorum profundus* – ulnar innervated in the forearm)
- Weakness of index finger extension (*extensor indicis proprius* – radial innervated)
- Sensory symptoms/signs in the dorsal medial hand/dorsal fifth and fourth fingers (territory of the dorsal ulnar cutaneous sensory branch)
- Sensory symptoms/signs in the medial forearm (territory of the medial antebrachial cutaneous sensory nerve)

##### Nerve conduction studies

- Abnormal median motor study (unless there is a coexistent carpal tunnel syndrome)
- Abnormal dorsal ulnar cutaneous sensory study
- Focal slowing or conduction block of the ulnar nerve at the elbow

##### Needle electromyography

- Abnormalities in the proximal ulnar-innervated muscles (*flexor carpi ulnaris* and *flexor digitorum profundus* to digits 4 and 5)
- Abnormalities in non-ulnar C8-innervated muscles (*abductor pollicis brevis*, *flexor pollicis brevis*, *extensor indicis proprius*)

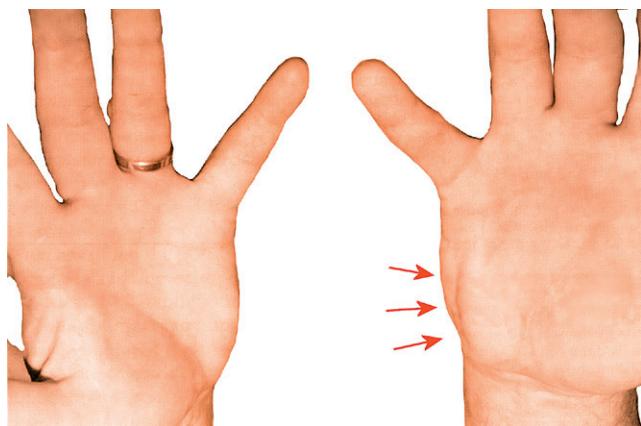
**Table 20–1.** Clinical and Electrophysiologic Differentiating Factors in Variants of Ulnar Neuropathy at the Wrist

	Deep Palmar Motor Branch Distal	Deep Palmar Motor Branch Proximal	Proximal Canal	Superficial Branch <sup>†</sup>
Weakness – interossei and 3rd/4th lumbricals	X		X	
Weakness – hypothenar muscles (ADM, ODM, FDM)		X		X
Sensory loss – volar medial hand and little finger, medial half ring finger			X	X
Reduced CMAP at FDI	X		X	
Reduced CMAP at ADM		X		X
Prolonged FDI latency	X		X	
Prolonged ADM latency		X		X
Reduced SNAP to digit 5			X	X
Prolonged latency comparing INT to 2nd LUM	X		X	
Conduction block at the wrist	X		X	
CV slowing at the wrist	X		X	
EMG abnormalities in FDI	X		X	
EMG abnormalities in ADM		X	X	

X = abnormalities may be present; INT = interossei; LUM = lumbrical; ADM = abductor digiti minimi; ODM = opponens digiti minimi; FDM = flexor digiti minimi; CV = conduction velocity; CMAP = compound muscle action potential.

<sup>†</sup>The superficial branch is often thought of as a “sensory branch.” However, it does supply one muscle, the palmaris brevis.

hand and fingers will be spared because they are innervated by the dorsal ulnar cutaneous sensory branch, which arises several centimeters proximal to the wrist. This is an important clinical point to remember when trying to discern if the ulnar nerve lesion is at the wrist or more proximal. In addition, the proximal volar medial hand should be spared because the palmar cutaneous branch also arises just proximal to the wrist.



**FIGURE 20-2** Palmaris brevis sign. When the palmaris brevis contracts, it results in puckering of the skin along the proximal medial border of the hand. As the palmar brevis is the only muscle supplied by the superficial branch of the ulnar nerve at the wrist, it will be spared in lesions of the deep branch. Thus, prominent contraction of the palmaris brevis may be seen when trying to abduct digit 5 in the more common lesions of the deep palmar motor branch of the ulnar nerve at the wrist. Note the prominent contraction of the palmaris brevis and wrinkling of the skin on the right hand (arrows) compared to the normal left hand.

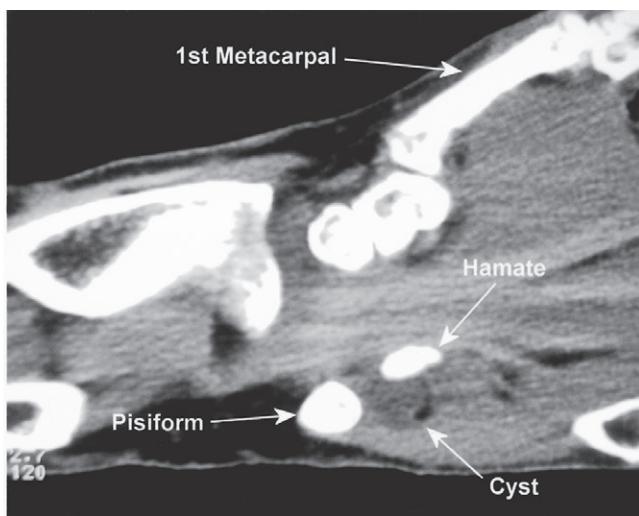
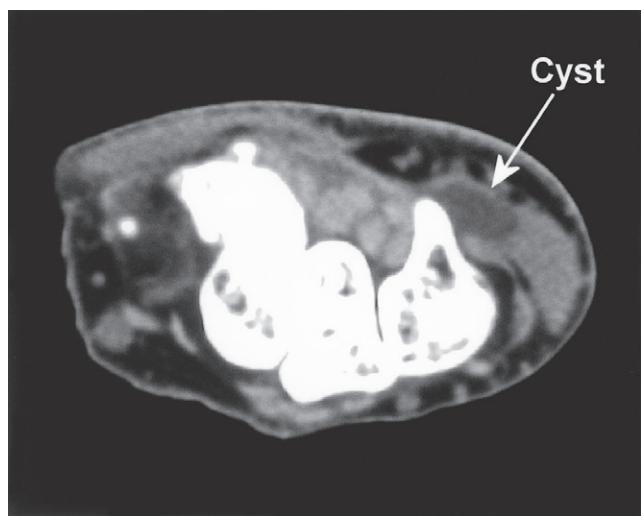
(Adapted with permission from Iyer, V.G., 1998. Palmaris brevis sign in ulnar neuropathy. *Muscle Nerve* 21, 675–677.)

## ETIOLOGY

Entrapment of the ulnar nerve at the wrist is far less common than at the more usual sites at the elbow. It has been described in association with trauma and wrist fracture. However, more common is a ganglion cyst within Guyon's canal that compresses the ulnar nerve (Figure 20-3). Rarely, an anomalous muscle or other mass lesions have been reported, including ulnar artery aneurysms, lipomas and other tumors. In addition, certain occupations or activities that involve repetitive movement or pressure against the ulnar wrist predispose to lesions at this location. This is especially true for bikers or laborers who use the same hand tools repetitively, which results in pressure on the hypothenar eminence (Figure 20-4). In such patients, the hypothenar area may be calloused at the compression site.

## DIFFERENTIAL DIAGNOSIS

In lesions where the superficial branch containing the sensory fibers is not affected, UNW is most often confused with early motor neuron disease. Motor neuron disease is well known to present with painless atrophy and weakness of a distal limb, a pattern essentially identical to distal UNW lesions. *The key differentiating finding on physical examination in UNW is the intact strength and bulk of the abductor pollicis brevis muscle, supplied by the median nerve.* In motor neuron disease, one would expect all C8–T1-innervated muscles to be equally affected. In UNW, there is a marked difference between ulnar C8–T1-innervated muscles (which are weak and wasted) and median C8–T1-innervated muscles (which are spared). However, this difference in ulnar versus median innervated muscles



**FIGURE 20-3** Ganglion cyst in Guyon's canal. One of the more common causes of ulnar neuropathy at the wrist is compression of the ulnar nerve by a ganglion cyst. Computed tomographic scan of the wrist. **Left:** Axial scan, volar side up. Note the cyst medial to the hook of the hamate. **Right:** Coronal scan, lateral hand up. Note the cyst between the pisiform and the hamate. (From Preston, D.C., Shapiro, B.E., Schecht, H.M., 2001. Ganglion cyst at Guyon's canal: electrophysiology and pathology. *J Clin Neuromusc Dis* 3, 89–91.)



**FIGURE 20-4** Occupational and activity risk factors for ulnar neuropathy at the wrist. Occupations that require repetitive use of hand tools can result in pressure on the hypothenar eminence (upper arrow). In addition, certain activities, especially prolonged cycling, can similarly result in ulnar neuropathy at the wrist (bottom arrow).

can also be seen in some atypical motor neuron disorders, such as multifocal motor neuropathy with conduction block, a rare autoimmune mediated motor neuropathy that preferentially affects distal muscles in a non-myotomal pattern of weakness (see Chapter 26).

In proximal lesions at the wrist where the superficial branch (and hence sensory fibers) is affected, the differential diagnosis is similar to that of UNE. Indeed, in UNW with sensory involvement, the most important diagnosis to exclude is UNE. Unequivocal sensory loss over the medial dorsal aspect of the hand and fingers and/or weakness of the distal flexors of the ring and little fingers are consistent with a lesion at the elbow, not at the wrist. However, in mild or early cases of UNE, these signs may not be present. In addition to UNE, one must keep in mind the possibilities of C8–T1 radiculopathy, lower trunk or medial cord brachial plexopathy, and rare cases of ulnar nerve entrapment in the arm or forearm, which can present with similar symptoms and signs.

## ELECTROPHYSIOLOGIC EVALUATION

### Nerve Conduction Studies

The findings on nerve conduction studies in UNW depend on (1) whether the superficial sensory branch is involved and (2) if the deep motor branch is involved, whether it is affected proximal or distal to the hypothenar muscles. If the lesion is distal, affecting only the deep palmar motor branch after the take-off to the hypothenar muscles, then the routine ulnar sensory study, recording the fifth digit, and the routine ulnar motor conduction study, recording the ADM, will be normal. In suspected UNW, additional nerve conduction studies must always be performed in order to detect abnormalities that may not be present on routine ulnar motor and sensory studies (Box 20-2).

In addition to routine ulnar motor studies recording ADM and sensory studies recording digit 5, the following studies often are helpful.

#### *Ulnar Motor Studies Recording the First Dorsal Interosseous*

*In all cases of suspected UNW, it is imperative to perform ulnar motor studies recording the first dorsal interosseous (FDI). In lesions of the distal deep palmar motor branch, the latency to the FDI may be prolonged with a decreased compound muscle action potential (CMAP) amplitude. Comparison with the contralateral asymptomatic side often is helpful as well. In cases where the lesion is more proximal, affecting the hypothenar branches, the distal motor latency (DML) to the ADM also may be prolonged, with a decreased CMAP amplitude. However, one of the patterns highly suggestive of UNW is preferential involvement of the distal deep palmar motor branch, whereby the ulnar motor study recording the FDI is affected out of proportion to the ulnar motor study recording the ADM. Comparison of their relative distal motor latencies often can be helpful:*

#### Normal Values:

DML to FDI:	$\leq 4.5$ ms
DML comparing FDI to ADM:	$\leq 2.0$ ms difference
DML comparing symptomatic FDI to contralateral FDI:	$\leq 1.3$ ms difference

#### *Dorsal Ulnar Cutaneous Sensory Study*

In cases of suspected UNW where the routine ulnar sensory conduction to digit 5 is abnormal, it is important to study the dorsal ulnar cutaneous sensory nerve. As the dorsal ulnar cutaneous sensory nerve arises 5 to 8 cm proximal to the wrist, it is expected to be normal in all cases of UNW. A normal antidromic response is greater than  $8 \mu\text{V}$ , but, as in other uncommonly performed sensory nerve conduction studies, comparison with the contralateral asymptomatic side frequently is helpful. Any potential that is less than 50% of the amplitude of the contralateral asymptomatic

### Box 20–2. Recommended Nerve Conduction Study Protocol for Ulnar Neuropathy at the Wrist

#### Routine studies:

1. Ulnar motor study recording abductor digiti minimi, stimulating wrist, below groove, and above groove in the flexed elbow position
2. Ulnar motor study recording first dorsal interosseous, stimulating wrist, below groove, and above groove in the flexed elbow position
3. Ulnar motor study recording first dorsal interosseous, stimulating the wrist (3 cm proximal to the distal wrist crease) and palm (4 cm distal to the distal wrist crease)
4. Median motor study recording abductor pollicis brevis, stimulating wrist and antecubital fossa
5. Median and ulnar F responses
6. Ulnar sensory response, recording digit 5, stimulating wrist (bilateral studies)
7. Median sensory response, recording digit 2 or 3, stimulating wrist
8. Dorsal ulnar cutaneous sensory response (bilateral studies)

#### Additional studies to consider:

9. Ulnar motor study recording the contralateral first dorsal interosseous, stimulating the wrist (in order to compare distal latencies and amplitudes side to side)
10. Lumbrical–interossei distal latency comparison study
11. Ulnar motor study recording the first dorsal interosseous, inching across the wrist in 1 cm increments

*The following patterns are consistent with ulnar neuropathy at the wrist:*

- DML to FDI: >4.5 ms (provided CMAP amplitude is not markedly reduced)
- DML comparing FDI to ADM: >2.0 ms difference
- DML comparing symptomatic FDI to contralateral FDI: >1.3 ms difference
- DML comparing ulnar INT to second lumbrical: >0.4 ms difference

SNAP, sensory nerve action potential; CMAP, compound muscle action potential; DML, distal motor latency; UNE, ulnar neuropathy at the elbow; ADM, abductor digiti minimi; FDI, first dorsal interosseous; INT, interossei.

*The following patterns denote ulnar neuropathy at the wrist with certainty:*

DML to FDI in the demyelinating range: >130% upper limit of normal (i.e., any DML to the FDI >6.0 ms)

Focal slowing across the wrist during inching studies: ≥0.5 ms over a 1 cm increment, recording FDI

Conduction block, comparing palm and wrist stimulations, recording FDI

Conduction velocity slowing across the wrist recording FDI

#### Special considerations:

- If the superficial sensory branch is affected, the SNAP amplitude will be low or absent, with a normal dorsal ulnar cutaneous SNAP. (Caution must be taken in interpreting this pattern, which also can occur in patients with UNE.)
- Occasional false-positive results occur when using the DML to FDI or ADM; comparing DML to FDI versus ADM; and the lumbrical–interossei study, especially in cases of moderate or severe UNE with axonal loss. Wrist versus palmar stimulation studies, or inching studies across the wrist should be done to demonstrate UNW with certainty.
- If the dorsal ulnar cutaneous sensory study is performed and is absent, it is prudent to stimulate the superficial radial sensory nerve along the lateral radius with the recording electrodes in place for the dorsal ulnar cutaneous sensory study to ensure that an anomalous innervation is not present (recall there is a very rare anomalous innervation wherein the superficial radial sensory nerve supplies the entire dorsum of the hand, including the usual territory of the dorsal ulnar cutaneous sensory nerve).

side likely is abnormal as well, even if the absolute amplitude is greater than 8  $\mu$ V\*.

Although the dorsal ulnar cutaneous sensory study often is helpful in establishing the level of the lesion, there are significant limitations of which every electromyographer must be aware. Although a *normal* dorsal ulnar cutaneous sensory study in the context of an *abnormal* digit 5 ulnar sensory study certainly suggests a diagnosis of UNW, this is not always the case. This pattern does not necessarily exclude the possibility of UNE (see Chapter 19). In some

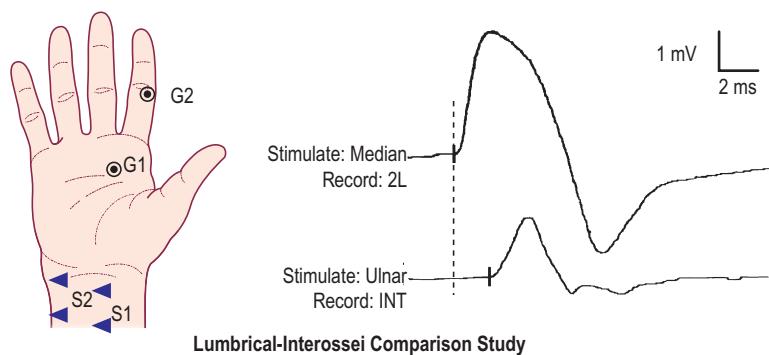
patients with definite UNE with axonal loss (although usually mild), the dorsal ulnar cutaneous sensory potential is spared. This is thought to be due to preferential fascicular sparing of the dorsal ulnar cutaneous sensory fibers. Therefore, care must be taken when interpreting the findings of a patient with a normal dorsal ulnar cutaneous SNAP and an abnormal digit 5 ulnar sensory response, especially if there is no conduction block or focal conduction velocity slowing across the elbow. These findings must be interpreted in light of findings on the ulnar motor studies and the needle electromyographic (EMG) study. Only when the dorsal ulnar cutaneous sensory study is abnormal is one assured that the lesion is above the level of the wrist; the converse is not always true.

#### Median Second Lumbrical versus Ulnar Interossei Distal Motor Latencies

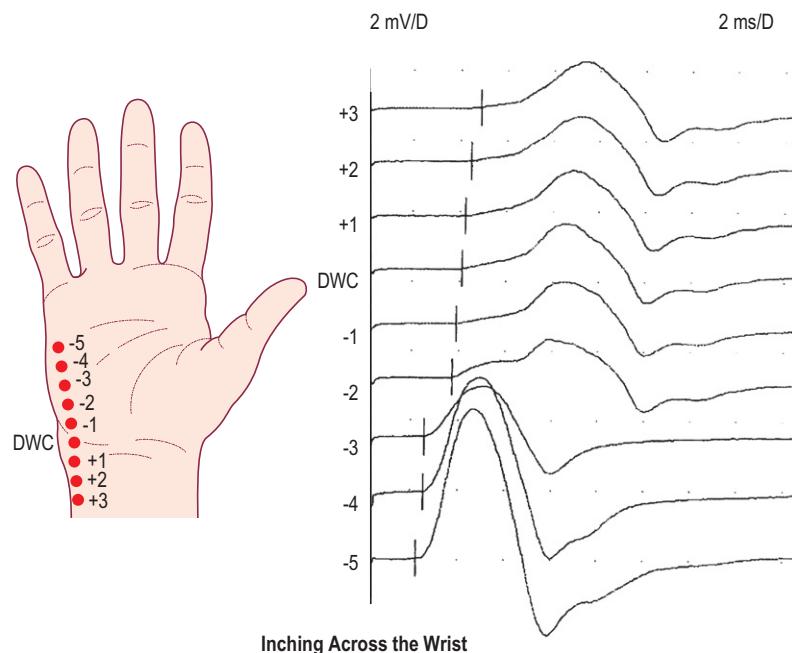
The lumbrical–interossei distal latency comparison often is performed as a sensitive, internal comparison study to demonstrate median nerve slowing across the carpal tunnel (see Chapter 17, Figure 17–9). However, this study can

\*Very rarely there is an anomalous innervation wherein the superficial radial sensory nerve supplies the entire dorsum of the hand, including the usual territory of the dorsal ulnar cutaneous sensory nerve. Thus, in cases where the dorsal ulnar cutaneous sensory response is absent, it is prudent to stimulate the superficial radial sensory nerve along the lateral radius, with the recording electrodes in place for the dorsal ulnar cutaneous sensory study, to ensure that this very rare anomalous innervation is not present (see Figure 7–12 from Chapter 7).

**FIGURE 20–5** Lumbral-interossei comparison study. This study is used most often in the diagnosis of carpal tunnel syndrome but can be equally helpful in the diagnosis of ulnar neuropathy at the wrist. The median nerve is stimulated at the wrist while the second lumbral muscle is recorded (**right top trace**); the ulnar nerve is stimulated at the wrist, using the same distance, while the interossei muscles are recorded (**right bottom trace**). In normal controls, latencies are similar. In a patient with ulnar neuropathy at the wrist, the interossei latency is prolonged compared with the second lumbral.



**FIGURE 20–6** Short segment incremental study of the ulnar nerve across the wrist. **Left:** Recording the first dorsal interosseous, the ulnar nerve is stimulated in successive 1 cm increments across the wrist. **Right:** Note the abrupt increase in amplitude, shift in latency, and change in morphology of the compound muscle action potential between 2 and 3 cm distal to the distal wrist crease (DWC). Inching studies allow for exact localization of the lesion.  
(From Preston, D.C., Shapiro, B.E., Schecht, H.M., 2001. Ganglion cyst at Guyon's canal: electrophysiology and pathology. *J Clin Neuromusc Dis* 3, 89–91.)



be used just as effectively to demonstrate UNW (Figure 20–5), looking for significant slowing of ulnar compared with median fibers across the wrist. Because the interossei are innervated by the distal deep palmar motor branch of the ulnar nerve and the second lumbral is innervated by the median nerve, this comparison test can be very useful in identifying ulnar slowing at the wrist. A DML difference of greater than 0.4 ms comparing the ulnar interossei with the median second lumbral, stimulating the nerves at the same distance, suggests focal slowing of the ulnar nerve across the wrist.

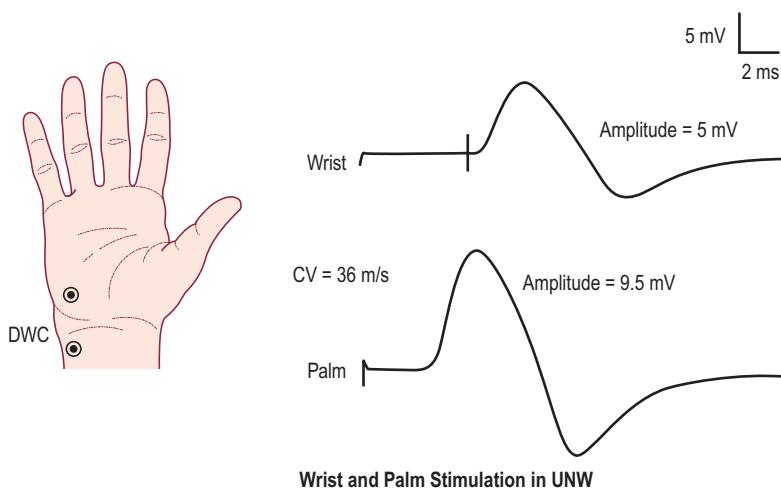
This study is reliable and easy to perform. However, two limitations must be kept in mind. First, if an ulnar neuropathy has a moderate or severe amount of axonal loss, be it at the wrist or higher, one should expect some mild slowing across the wrist simply on the basis of loss of some of the fastest conducting axons. Second, the lumbral-interossei study will fail in cases of UNW if there is a coexistent

median neuropathy at the wrist. This is generally not an issue when using this comparison study for median neuropathy at the wrist, as UNW is so rare. However, when looking for a UNW, an incidental median neuropathy at the wrist may not be that uncommon.

#### Short Segment Incremental Studies

Using a technique identical to that used for ulnar nerve lesions at the elbow, short segment incremental studies (SSIS) or “inching” can be effectively performed at the wrist, recording the FDI, looking for an abrupt change in either latency or amplitude (Figure 20–6). One centimeter increments are carefully marked off from 2 to 4 cm above the distal wrist crease to 4 to 5 cm below. The ulnar nerve is then stimulated supramaximally at each location at successive 1 cm intervals, from below to above the wrist. Any abrupt increase in latency or drop in amplitude between successive stimulation sites implies focal demyelination. In

**FIGURE 20–7** Wrist and palm stimulation in ulnar neuropathy at the wrist. As an alternative to inching studies, a compound muscle action potential can be recorded at the first dorsal interosseous with stimulation at the wrist and palm, looking for conduction block and/or conduction velocity slowing across the ulnar wrist. Note that in this case of ulnar neuropathy at the wrist, there is a large decrease in amplitude with stimulation at the wrist compared to the palm, signifying conduction block, and a slowed conduction velocity. Both findings localize the ulnar neuropathy to the wrist.



normal individuals, the latency between two successive 1 cm stimulation sites usually is 0.1 to 0.3 ms and rarely 0.4 ms. Any latency shift  $\geq 0.5$  ms suggests focal slowing.

#### Wrist and Palmar Stimulation

Comparing the CMAP amplitudes stimulating at the wrist and palm can be technically easier than inching across the wrist, and yield similar information (Figure 20–7). To perform this study, the ulnar nerve is stimulated 3 cm above the wrist and 4 cm distal to the distal wrist crease in the palm, recording the FDI. Whereas inching requires multiple stimulations at 1 cm increments, this study only requires single palm and wrist stimulations. UNW can be localized either by finding a conduction block between the wrist and palm stimulation sites or by finding conduction velocity slowing across the wrist. Similar to all routine motor studies, if a nerve is stimulated at two sites, a conduction velocity can be calculated. *In UNW, any conduction velocity less than 37 m/s is considered abnormal and is of localizing value.* In UNW, the demonstration of conduction block or conduction velocity slowing is most helpful in definitively localizing the lesion. However, additional information is gained about prognosis, as demyelinating lesions have a far better prognosis than those associated with axonal loss.

#### Comparison of the Various Electrophysiologic Tests in Ulnar Neuropathy at the Wrist

There has been little data comparing the relative usefulness of the various studies outlined earlier, because UNW is relatively uncommon. Most reports of UNW have been single case reports or reports of a small number of patients. One large study of 20 consecutive patients with clinically defined UNW was performed prospectively, comparing the following studies: (1) wrist and palmar stimulation studies, recording FDI, looking for conduction block across the wrist; (2) wrist and palmar stimulation studies, recording FDI, looking for conduction velocity slowing across

the wrist; (3) lumbrical–interossei study, comparing ulnar versus median distal latencies; and (4) routine ulnar motor studies, recording FDI and ADM, comparing their respective DMLs. In five patients, inching studies across the wrist also were performed. Importantly, these studies were also compared in 30 asymptomatic normal control subjects and in 20 consecutive disease control patients with definite UNE.

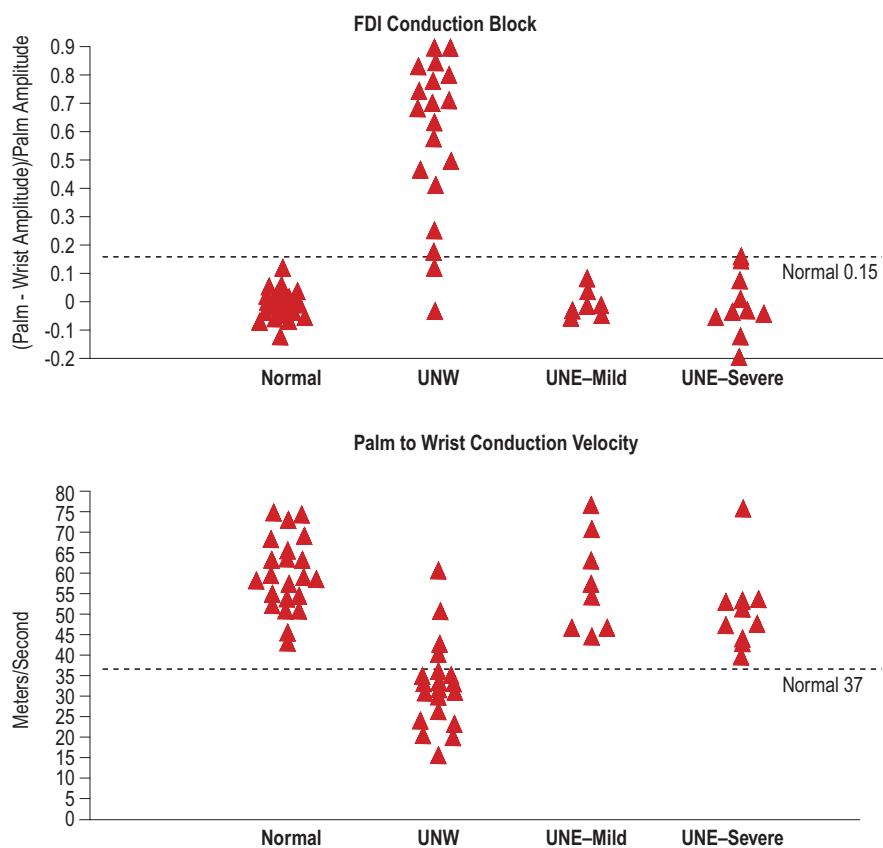
*The most sensitive and specific studies for localizing the lesion to the wrist were conduction block across the wrist and a slowed wrist-to-palm conduction velocity recording the FDI.* Conduction block was found in 70% and a slowed wrist-to-palm conduction velocity in 80% of patients with UNW, using wrist and palmar stimulation (Figure 20–8). Overall, 95% of the patients with UNW had either conduction block or a slowed conduction velocity. These findings were 100% specific. Neither conduction block nor conduction velocity slowing across the wrist was found in any of the control patients with UNE. Of the five patients in whom inching was performed, all showed focal slowing and conduction block.

The lumbrical–interossei comparison study had a sensitivity of 60% (Figure 20–9). However, one patient with a severe UNE had an abnormal study (latency difference of 0.6 ms). One reason for the lower than expected sensitivity for this study was the presence of coexistent median neuropathy at the wrist in 25% of patients.

A prolonged DML to the FDI or ADM also had a lower sensitivity, in the range of 55 to 60% (Figure 20–9). More importantly, prolonged distal latencies to these muscles were also less specific than the previously described studies. A prolonged DML to the FDI was found in one patient with mild UNE and in 40% of patients with severe UNE. Similarly, a prolonged DML to the ADM was found in 40% of patients with severe UNE. The prolonged DMLs in patients with UNE presumably were the result of axonal loss and dropout of some of the faster conducting fibers.

**FIGURE 20-8** Conduction block and focal slowing across the wrist. **Top:** Change in first dorsal interosseous (FDI) compound muscle action potential (CMAP) amplitude with the ulnar nerve stimulated above and below the wrist is plotted for normals, patients with ulnar neuropathy at the wrist (UNW), and patients with ulnar neuropathy at the elbow (UNE) (mild and severe). Conduction block is calculated as (Palmar–Wrist CMAP Amplitude)/(Palmar CMAP Amplitude). **Bottom:** Conduction velocity across the ulnar wrist, recording the FDI, is plotted for normals, patients with UNW, and patients with UNE (mild and severe). Normal limits are shown as dotted lines.

(From Cowdery, S.R., Preston, D.C., Herrmann, D.N., et al., 2002. Electrodiagnosis of ulnar neuropathy at the wrist: conduction block versus traditional tests. *Neurology* 59, 420–427.)



The least sensitive study for UNW was the comparison of DMLs to the FDI versus ADM, being abnormal in only 15% of patients with UNW. However, one patient with a mild UNE also had a relatively prolonged DML to FDI compared with ADM.

*The important points to take away from this study are as follows:*

1. By performing an additional stimulation at the palm, while recording the FDI, conduction block or focal slowing across the wrist can be demonstrated in 95% of patients with clinically definite UNW. This finding was 100% specific; it was not seen in any control patient with UNE.
2. Inchng studies across the wrist also are very sensitive and specific. However, these studies are more time consuming and technically demanding than simply stimulating at one additional site in the palm.
3. The lumbrical–interossei study is a sensitive and helpful test, with one important exception. Its usefulness is greatly diminished if there is a coexistent median neuropathy at the wrist. Rarely, a false-positive result can occur if a patient has a severe UNE. Increasing the cutoff value to 0.7 ms or above may eliminate this problem.
4. Prolongation of the DML to FDI or ADM is much less sensitive than conduction block or slowing across the wrist, recording the FDI. In addition, it is also

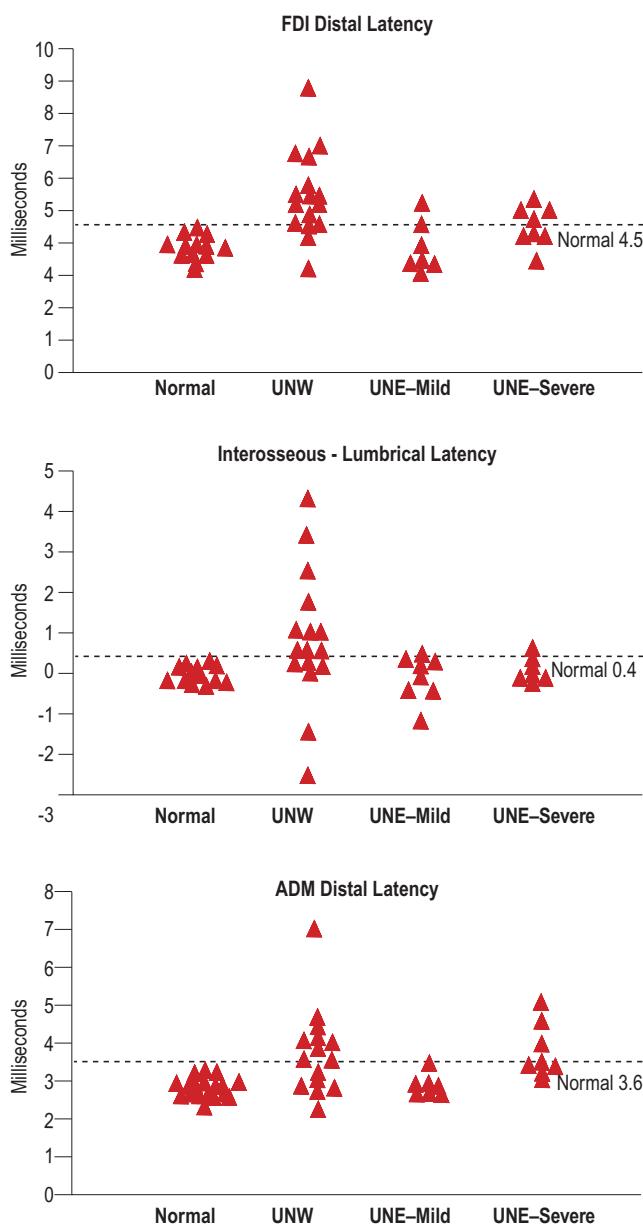
much less specific, being present in some cases of UNE.

5. Comparing the DML to FDI versus ADM is only infrequently helpful, being fairly insensitive to ulnar neuropathy at the wrist.

## Electromyographic Approach

The needle EMG examination in suspected UNW is straightforward (Box 20-3). The FDI and ADM must be sampled, with the electromyographer looking for involvement of the distal and proximal deep palmar motor branches, respectively. The flexor digitorum profundus (FDP) 5 and flexor carpi ulnaris (FCU) must be sampled to exclude an ulnar neuropathy proximal to the wrist. Finally, median- and radial-innervated C8 muscles (e.g., abductor pollicis brevis, flexor pollicis longus, extensor indicis proprius) and the lower cervical paraspinal muscles must be sampled to exclude a cervical root or motor neuron lesion.

As in UNE, the lesion in UNW can be purely axonal, indicated by low CMAP amplitudes at ADM and FDI with normal or only mild slowing of distal latency. In these cases, it can be difficult to differentiate a lesion of the deep palmar motor branch from a lesion proximal to the dorsal root ganglion (cervical root or motor neuron). The EMG is helpful in this regard. The electromyographer can confirm



**FIGURE 20-9** Distal motor latency studies in ulnar neuropathy at the wrist (UNW). Distal motor latencies to the first dorsal interosseous (**top**), difference in distal motor latencies between the interosseous and lumbrical (**middle**), and distal motor latencies to the abductor digiti minimi (**bottom**) are plotted for normals, patients with UNW, and patients with ulnar neuropathy at the elbow (UNE) (mild and severe). Normal limits are shown as dotted lines. Note the false-positive results that occur in some cases of UNE. (From Cowdery, S.R., Preston, D.C., Herrmann, D.N., et al., 2002. Electodiagnosis of ulnar neuropathy at the wrist: conduction block versus traditional tests. *Neurology* 59, 420–427.)

that the abnormalities are limited to ulnar-innervated muscles distal to the wrist by also sampling proximal ulnar-innervated and non-ulnar C8-T1-innervated muscles. Again, however, early motor neuron disease may be difficult to exclude. In these cases, the clinical presentation and serial follow-up remain important.

### Box 20-3. Recommended Electromyographic Protocol for Ulnar Neuropathy at the Wrist

#### Routine studies:

1. Distal deep palmar motor ulnar-innervated muscle (first dorsal interosseous)
2. Proximal deep palmar motor ulnar-innervated branches to hypothenar muscles (abductor digiti minimi)
3. Forearm ulnar-innervated muscles (flexor carpi ulnaris and flexor digitorum profundus 5)

*If any of the ulnar-innervated muscles are abnormal, test the following additional muscles:*

4. At least two non-ulnar lower trunk/C8 innervated muscles (e.g., abductor pollicis brevis, flexor pollicis longus, extensor indicis proprius) to exclude a lower trunk brachial plexopathy, polyneuropathy, C8-T1 radiculopathy, or motor neuron disease
5. C8 and T1 paraspinal muscles

*Special consideration:* If the pathology at the wrist is purely axonal and spares sensory fibers, it is difficult to completely exclude a lesion proximal to the dorsal root ganglion (i.e., root or motor neuron).



### EXAMPLE CASE

#### Case 20-1

##### History and Physical Examination

A 36-year-old right-handed man complained of numbness and paresthesias over digits 4 and 5, as well as right arm pain for 6 months. The sensory disturbance had become worse over the past few weeks. He worked in a library stacking books and denied any history of trauma. He had vague complaints of right elbow pain.

On examination, there was mild atrophy of the intrinsic hand muscles. There was mild weakness of the ADM and interossei muscles on the right. The long flexors of digits 4 and 5 were strong. There was no Tinel's sign at the elbow. Sensation to pin and light touch was normal.

##### Summary

The history and physical examination both are suggestive of ulnar neuropathy. Despite the normal sensory examination, the patient noted paresthesias and numbness in the ulnar-innervated fourth and fifth digits. In addition, the motor examination showed atrophy and weakness of the right ADM and the interossei. Thus, the patient has clear symptoms of ulnar sensory and motor dysfunction. The suggestion of pain at the right elbow leads one to seriously consider the possibility of ulnar neuropathy in the region of the elbow. However, at this point there are no other signs to help localize the lesion. The fact that the long flexors to digits 4 and 5 (ulnar portion of the FDP) are normal suggests that the ulnar neuropathy either is mild and has not affected these more proximal muscles or is more distal.

The clinical approach to this case is similar to that used in other cases of ulnar nerve dysfunction. The differential diagnosis includes UNW, UNE, lower trunk/

**CASE 20–1.** Nerve Conduction Studies

Nerve Stimulated	Stimulation Site	Recording Site	Amplitude Motor = mV; Sensory = $\mu$ V			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	12.5		$\geq$ 4	4.2		$\leq$ 4.4				28		$\leq$ 31
Median (m)	Antecubital fossa	APB	12.2			8.3			50		$\geq$ 49			
Ulnar (m)	Wrist	ADM	4.2		$\geq$ 6	4.1		$\leq$ 3.3				31		$\leq$ 32
Ulnar (m)	Below elbow	ADM	4.1			7.4			60		$\geq$ 49			
Ulnar (m)	Above elbow	ADM	4.1			9.2			57		$\geq$ 49			
Median (s)	Wrist	Index finger	48		$\geq$ 20	3.2		$\leq$ 3.5	58			$\geq$ 50		
Ulnar (s)	Wrist	Little finger	10	23	$\geq$ 17	3.4	3.2	$\leq$ 3.1	42	52		$\geq$ 50		

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

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 20–1.** Additional Nerve Conduction Studies

Nerve Stimulated	Stimulation Site	Recording Site	Amplitude Motor = mV; Sensory = $\mu$ V			Latency (ms)			Conduction Velocity (m/s)			F-wave Latency (ms)		
			RT	LT	NL	RT	LT	NL	RT	LT	NL	RT	LT	NL
Dorsal ulnar (s)	Lateral wrist	Dorsal medial hand	24	26	$\geq$ 8	2.1	2.2	$\leq$ 2.8	50	51	$\geq$ 50			
Median (m)	Wrist	Second lumbral	4.1	3.8	$\geq$ 1.0	4.4	4.4							
Ulnar (m)	Wrist	Interosseous	5.5	6.2	$\geq$ 2.5	5.5	4.4							
Lum-int diff.						1.1	0.0	$\leq$ 0.4						
Ulnar (m)	Wrist	FDI		3.6	$\geq$ 7	5.2	4.4	$\leq$ 4.5						
Ulnar (m)	Below elbow	FDI		3.4			7.6		55		$\geq$ 50			
Ulnar (m)	Above elbow	FDI		3.4			8.9		57		$\geq$ 50			
Ulnar (m)	Palm	FDI		8.0			3.2		35		$\geq$ 50			

**CASE 20–1.** Electromyography

Muscle	Insertional Activity	Spontaneous Activity			Voluntary Motor Unit Action Potentials						
		Fibrillation Potentials	Fasciculations	Activation	Recruitment	Configuration			Duration	Amplitude	Polyphasia
Right FDI	↑	+1	0	NL	↓				+1	+1	+1
Right ADM	↑	+1	0	NL	↓				+1	+1	+1
Right APB	NL	0	0	NL	NL				NL	NL	NL
Right EIP	NL	0	0	NL	NL				NL	NL	NL
Right FCU	NL	0	0	NL	NL				NL	NL	NL
Right FDP 5	NL	0	0	NL	NL				NL	NL	NL

↑ = increased; ↓ = slightly reduced; NL = normal; FDI = first dorsal interosseous; ADM = abductor digiti minimi; APB = abductor pollicis brevis; EIP = extensor indicis proprius; FCU = flexor carpi ulnaris; FDP = flexor digitorum profundus.

medial cord lesions of the brachial plexus, or a C8-T1 radiculopathy.

The nerve conduction studies include, first, a normal median motor conduction study recording the abductor pollicis brevis muscle. However, the ulnar motor conduction study shows a mildly low CMAP amplitude recording the ADM with a moderately prolonged distal latency but a normal conduction velocity in the forearm and across-elbow segments. There is no conduction block or significant differential slowing of the ulnar nerve across the elbow ( $>10-11$  m/s) to substantiate the possibility of UNE. Median and ulnar routine sensory studies are then performed. The median study is completely normal, but the ulnar study shows a decreased amplitude on the right with a normal amplitude on the left. Therefore, at this point in the study, one can be fairly certain that the patient has an ulnar neuropathy because both the ulnar motor and sensory studies are abnormal. The normal median motor and sensory studies exclude a more generalized process such as a polyneuropathy to explain the abnormal ulnar motor and sensory findings. Although a lower trunk brachial plexopathy is still a consideration, one would expect to also see a low median CMAP amplitude in this case. At this point in the study, we are confronted with a common problem, that of a non-localizable ulnar neuropathy. There is no focal slowing or conduction block to suggest an ulnar neuropathy at the elbow.

Several questions should be addressed.

### **What is the Significance of the Prolonged Ulnar Distal Motor Latency?**

The only unusual abnormality is the moderately prolonged distal latency to the ADM muscle (4.1 ms). This value is more than 125% of the upper limit of normal and suggests the possibility of a demyelinating lesion at the wrist. Recall from the history that the patient uses his hands to stack books repetitively, which may be a risk factor for entrapment of the ulnar nerve at the wrist. Further studies of the ulnar nerve at the wrist are indicated.

### **What Other Tests can be Used to Help Localize the Lesion?**

Because the routine ulnar conduction studies typically are normal or equivocal in UNW, additional nerve conduction studies are required to localize the lesion to the wrist (Box 20.2). In UNW, the dorsal ulnar cutaneous sensory response is expected to be normal, whereas the sensory potential to the fifth digit may be abnormal. When the dorsal ulnar cutaneous sensory response is checked and compared with the contralateral side, it is normal and symmetric bilaterally. The presence of a normal dorsal ulnar cutaneous response with an abnormal digit 5 ulnar response is consistent with UNW, although, as already noted, this pattern occasionally can be seen in mild cases of UNE.

Next, the lumbrical-interossei comparison study is performed using identical distances. On the left

(asymptomatic) side, an identical distal latency of 4.4 ms to both the lumbrical and interossei is found. On the involved right side, however, there is a clear asymmetry: the ulnar latency is 1.1 ms longer than the median latency. Any difference of more than 0.4 ms suggests focal slowing across the wrist.

Lastly, the ulnar motor study is repeated but recording the FDI. There is no focal slowing or conduction block across the elbow. However, the FDI distal latency on the involved right side is moderately prolonged at 5.2 ms, with a normal value of 4.4 ms on the contralateral side. In addition, the CMAP amplitude is reduced on the right compared with the left. Comparing the distal latency to the FDI to that of the ADM, there is a difference of 1.1 ms, which is in the range of normal ( $\leq 2.0$  ms). When an additional stimulation is given in the palm while recording the FDI, the amplitude markedly increases to 8.0 mV, signifying a conduction block between the palm and wrist. In addition, the calculated velocity across the wrist is in the demyelinating range, being less than 37 m/s.

Proceeding to the needle EMG study, particular attention must be paid to the ulnar-innervated muscles above the level of the wrist, which would be expected to be normal in cases of UNW. The EMG study shows active denervation and reinnervation in the FDI (innervated by the distal deep palmar motor branch of the ulnar nerve). The right ADM yields similar findings, indicating that the branch to the hypothenar muscles is also affected. The right abductor pollicis brevis is normal, as is the right extensor indicis proprius. The normal findings in these two non-ulnar C8-innervated muscles again signify that the problem likely is limited to the ulnar nerve. Finally, both proximal ulnar muscles, the FCU and FDP 5, are sampled and are normal.

Therefore, with EMG and nerve conduction studies completed, we are ready to form an electrophysiologic impression.

**IMPRESSION:** *There is electrophysiologic evidence of an ulnar neuropathy at the wrist.*

From the pattern of the nerve conduction and EMG data, we can conclude that the patient has an ulnar nerve lesion at the wrist affecting the superficial sensory branch and the proximal deep palmar branch. This pattern is one variant of UNW. In this case, the patient's history, examination, and electrophysiologic results all correlate well. The atrophy and weakness of the intrinsic hand muscles correlate with the reduced ulnar CMAP amplitudes seen on nerve conduction studies and with the denervation and reinnervation with reduced recruitment of MUAPs revealed by the needle EMG findings. The findings that, taken together, tend to localize the lesion at the wrist include not only the EMG abnormalities that are limited to ulnar muscles distal to the wrist but also the intact dorsal ulnar cutaneous sensory response and the

prolonged ulnar latency on the lumbral–interossei study. *However, the study that unequivocally localizes the ulnar neuropathy to the wrist is the palmar stimulation compared to the wrist stimulation, while recording the FDI.* The finding of focal demyelination across the wrist (conduction block and/or conduction velocity slowing) is the key finding.

One can easily see that if additional studies had not been performed (i.e., the dorsal ulnar cutaneous sensory study, motor conduction study to the FDI including palmar stimulation, and lumbral–interossei distal latency comparison study), the erroneous diagnosis of a non-localizable ulnar neuropathy might have been made. The initial clue to this diagnosis on the nerve conduction studies was the relatively prolonged distal latency to the ADM muscle in conjunction with only a mildly reduced CMAP amplitude.

In this case, sensory symptoms and abnormalities on nerve conduction studies indicated an ulnar nerve lesion. However, one should remember that in other cases of UNW, in which the lesion affects the deep palmar motor branch in isolation, only the motor fibers are affected; the sensory fibers are spared. In such cases, excluding a lesion proximal to the dorsal root ganglion (either nerve root or anterior horn cell) may be very difficult. If the pathology is axonal loss alone and there is no focal slowing or conduction block of ulnar motor fibers across the wrist, making that differentiation is impossible. In those unusual cases, the EMG report must be considered indeterminate. Although EMG abnormalities may be limited to ulnar-innervated muscles, the possibility that those muscles are the first to be affected in a lesion of the nerve root or anterior horn cells cannot be completely excluded. Indeed, there are cases of focal motor neuron disease that mimic UNW on initial presentation, preferentially affecting the deep palmar motor branch. Clinical history and often follow-up electrophysiologic studies are required to make the differentiation.

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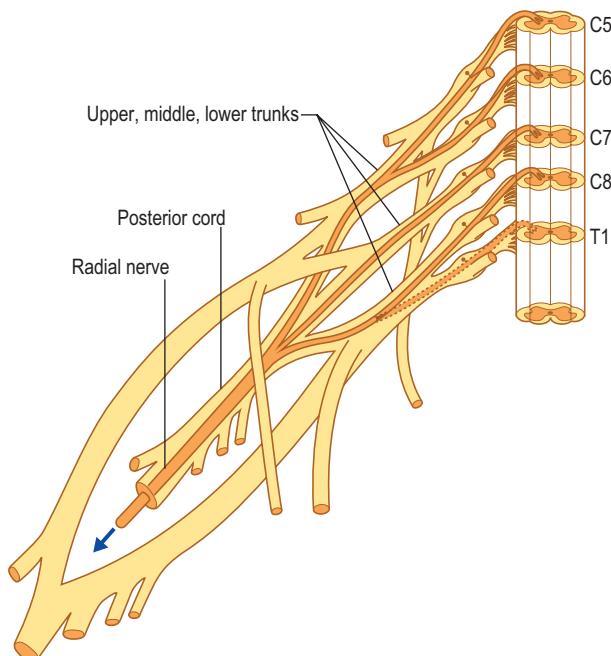
# Radial Neuropathy

# 21

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.

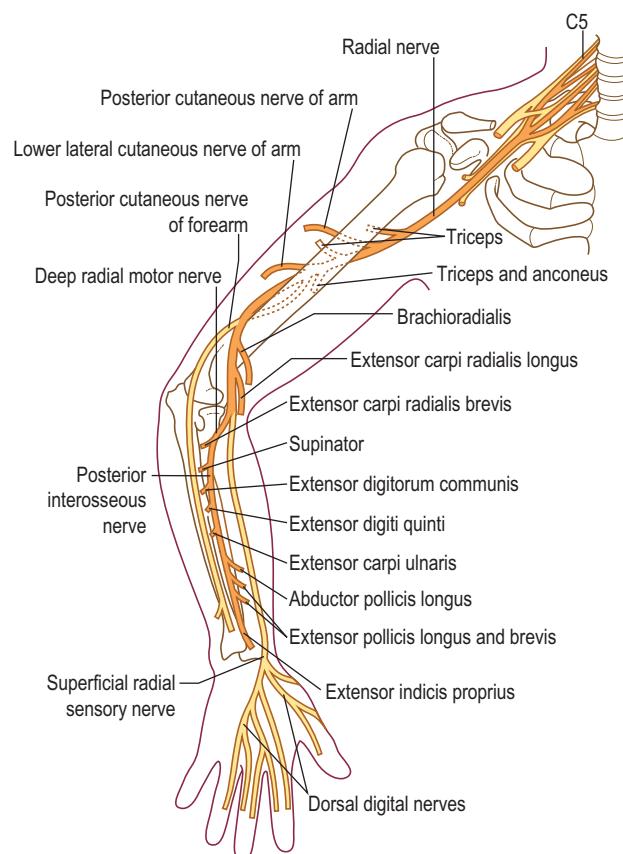
## ANATOMY

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.

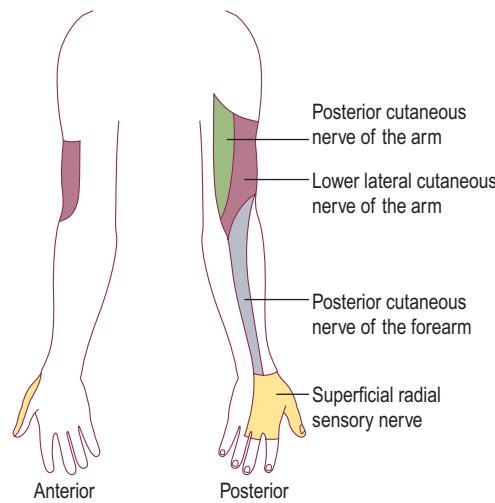
(Adapted with permission from Haymaker, W., Woodhall, B., 1953. Peripheral nerve injuries. WB Saunders, Philadelphia.)

### Nomenclature of the Branches of the Radial Nerve near the Elbow

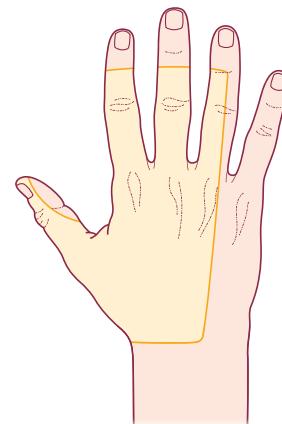
One of the more confusing aspects of radial nerve anatomy is the inconsistency regarding the nomenclature of the branches of the radial nerve near the elbow used in various anatomic texts and clinical reports (Figure 21–7). The following points should help the electromyographer when dealing with potential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

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

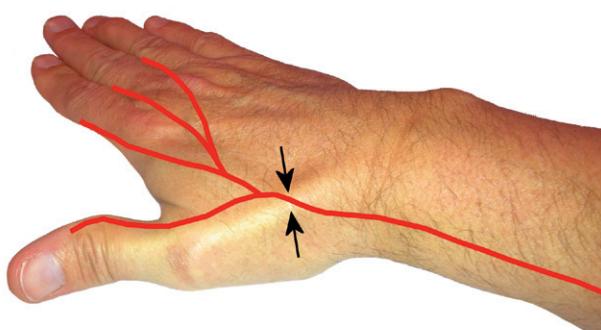
- Distal to the spiral groove but before the elbow, the main radial nerve always supplies two muscles: the



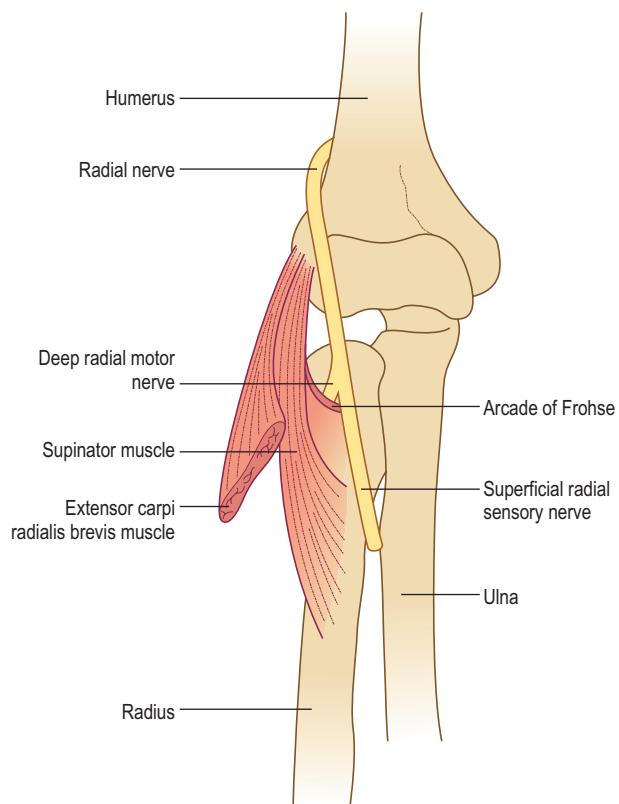
**FIGURE 21–3** Sensory territories supplied by the radial nerve. (Adapted with permission from Haymaker, W., Woodhall, B., 1953. Peripheral nerve injuries. WB Saunders, Philadelphia.)



**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 the 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\*.

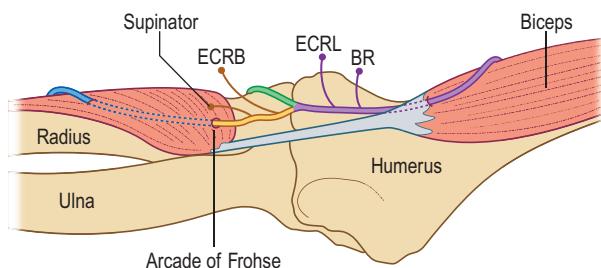
#### The Bifurcation near the Elbow

- 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.

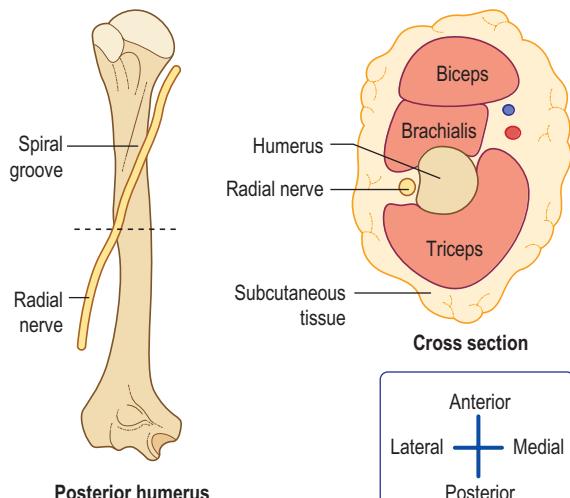
In isolated radial neuropathy at the spiral groove, median- and ulnar-innervated muscles are normal. However, tested in a wrist drop and finger drop posture, finger abduction may appear weak, giving the mistaken impression of ulnar nerve dysfunction. To prevent this error, one should test the patient's 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
<b>EMG Findings</b>					
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
<b>Nerve Conduction Study Findings</b>					
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:*

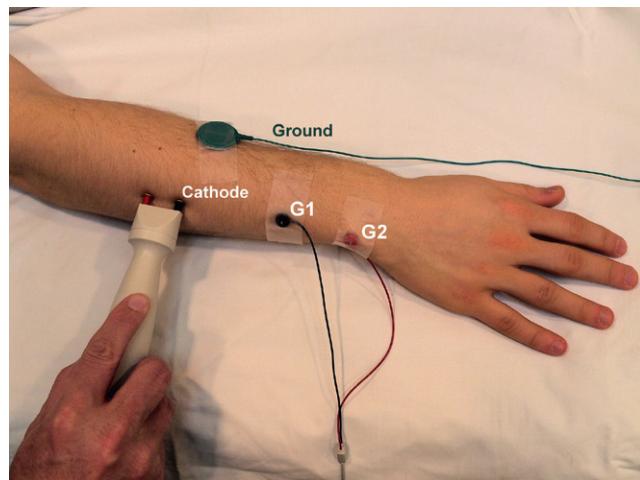
1. Radial motor study recording extensor indicis proprius, stimulating forearm, elbow, below spiral groove, and above spiral groove; bilateral studies
2. Ulnar motor study recording abductor digiti minimi, stimulating wrist, below groove, and above groove in the flexed elbow position
3. Median motor 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



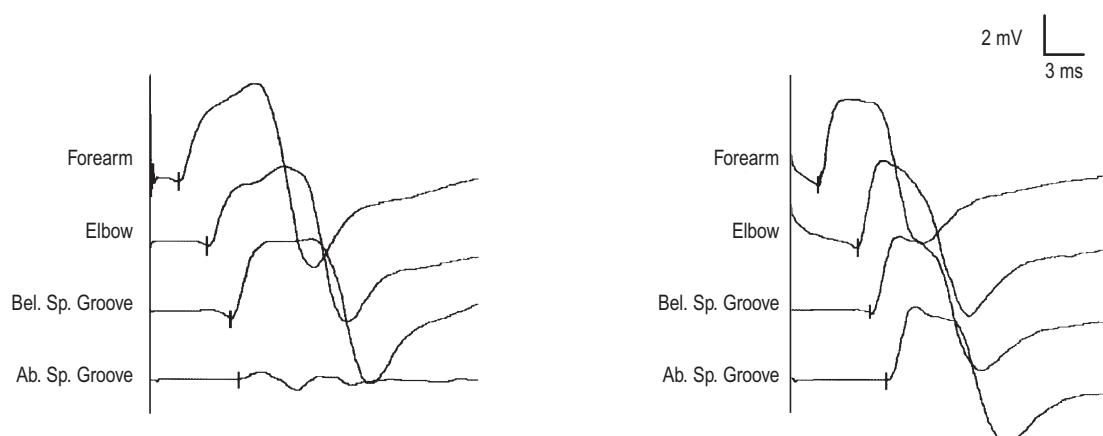
**FIGURE 21–9** Radial motor study. The active electrode is placed over the extensor indicis proprius, 2 cm proximal to the ulnar styloid, with the reference electrode over the ulnar styloid. The radial nerve can be stimulated in the forearm, at the elbow, and below and above the spiral groove.

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).

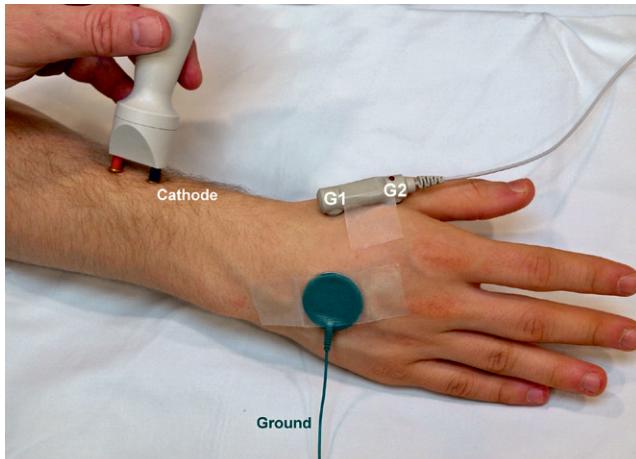
In cases of radial neuropathy at the spiral groove, CMAPs recorded with stimulation at the forearm, elbow, and below the spiral groove can be completely normal if the lesion is purely demyelinating. However, stimulation above the spiral groove will result in electrophysiologic evidence of a conduction 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

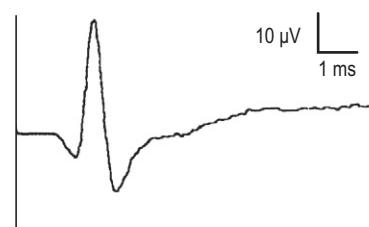


**FIGURE 21-10** Radial motor studies for radial neuropathy at the spiral groove. **Left:** Symptomatic arm. **Right:** Contralateral asymptomatic arm. Recording extensor indicis proprius and stimulating the forearm, elbow, below spiral groove, and above spiral groove. Note the marked drop in amplitude 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 = $\mu$ 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 $\geq 49$			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 $\geq 49$			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	Configuration			Duration	Amplitude	Polyphasia
						NL	↓↓↓	NL			
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 = $\mu$ 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			
	Above spiral groove	EIP	NR	7.7	$\geq$ 2	NR	8.9				64			
Median (m)	Wrist	APB	5.4	$\geq$ 4		3.6	$\leq$ 4.4							
	Antecubital fossa	APB	5.3	$\geq$ 4		7.0	59				$\geq$ 49			
Ulnar (m)	Wrist	ADM	9.8	$\geq$ 6		2.7	$\leq$ 3.3							
	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**

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