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Electrodiagnostic approach to the patient with suspected mononeuropathy of the upper extremity

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Upper extremity mononeuropathies are common. Their diagnosis depends on a working knowledge of neuroanatomy, a detailed history and clinical examination, and electrodiagnostic studies. The electrodiagnostic examination, being an extension of the clinical evaluation, confirms the clinical diagnostic hypothesis, excludes competing diagnoses, and assesses severity, chronicity, and activity of the mononeuropathy. Electrodiagnostic studies are of most value when they are focused by a comprehensive clinical evaluation.

This article considers an electrodiagnostic approach to upper extremity mononeuropathies, according to the presenting complaint as follows:

- "Weak or painful shoulder"
- "Weakness about the elbow"
- "Wrist or finger drop"
- "Pain and weakness/numbness in the forearm or wrist and hand"
- "A numb or weak hand"

A working knowledge of upper extremity neuroanatomy and of the clinical features of upper extremity entrapment syndromes is assumed. For each neuropathy, the authors provide their opinion on the most effective electrodiagnostic strategy. The terms *entrapment* and *compression* are used interchangeably, although it should be recognized that a true "entrapment neuropathy" implies compression of neural structures in a fibro-osseus canal.

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General principles

The electrodiagnostic consultant should address a number of questions that ultimately guide management decisions (see Box 1). The authors believe the electromyographer is best served by a conservative approach. Conclusions based on a single measure of nerve function or on equivocal data are prone to error and may lead to inappropriate care [1,2]. Timing of electrodiagnostic studies is critical. In the setting of acute focal peripheral nerve lesions, the authors generally recommend that electrodiagnostic studies be performed 2 to 3 weeks after onset of symptoms to maximize information gained regarding the degree of axon loss.

"Weak or painful shoulder"

Long thoracic neuropathies

Long thoracic neuropathies are traumatic or nontraumatic in origin. Traumatic causes include acute direct injury (eg, radical mastectomies), repetitive stretch or traction injury (eg, weight training), or external compression (eg, "rucksack palsy"). Most nontraumatic cases form a subgroup of neuralgic amyotrophy with selective involvement of the long thoracic nerve [3]. Patients complain of shoulder pain, weakness, and reduced range of motion at the shoulder. Occasionally, scapula winging is reported. Physical examination discloses winging of the scapula that is accentuated by protraction of the arm against resistance.

Electrodiagnostic approach

Needle examination is the mainstay of electrodiagnosis (see Box 2 on next page). The long thoracic nerve has a purely motor supply to the serratus

Box 1
Goals of the electrodiagnostic examination

What is the localization of the lesion?
Are motor or sensory fibers involved or both?
What is the physiologic basis of the lesion (eg, axon loss, demyelination)?
What is the severity of the lesion?
  Degree of axon loss?
  Is axonal continuity present?
What is the chronicity of the lesion?
  Is there evidence of reinnervation of evidence or ongoing axon loss?
What is the prognosis?

Box 2
Suggested electrodiagnostic approach to long thoracic neuropathies

Nerve conduction studies (NCS) (ipsilateral)
Median and ulnar sensory NCS (digits 2 and 5)
Median and ulnar motor NCS and an ulnar F response

Needle examination
Serratus anterior, infraspinatus, deltoid, biceps, triceps, C5--7 paraspinal muscles

anterior. Electrodiagnosis focuses on the demonstration of isolated involvement of the serratus anterior muscle. Other C5–7-innervated muscles are sampled to exclude a cervical radiculopathy, brachial plexopathy, or a myopathy (eg, facioscapulohumeral dystrophy). Nerve conduction studies (NCS) should be performed in the upper extremity to exclude coexistent compression neuropathies (eg, hereditary neuropathy with liability to pressure palsies (HNPP) and to evaluate for evidence of a brachial plexopathy. The authors do not routinely perform long thoracic NCS [4–6]; their opinion is that these studies are technically unreliable and add little to the needle examination findings in what is typically an axon loss lesion [7].

Suprascapular neuropathies

The suprascapular nerve innervates the supraspinatus and infraspinatus muscles, and provides sensation to the glenohumeral and acromioclavicular joints. It originates from the proximal upper trunk of the brachial plexus, with predominantly C5 nerve root innervation. Suprascapular nerve entrapment occurs primarily at two sites: the suprascapular notch and the spinoglenoid notch [8]. Proximal entrapment of the suprascapular nerve at the suprascapular notch results in shoulder pain and weakness of the supraspinatus and infraspinatus muscles. More distal entrapment at the spinoglenoid notch produces isolated, usually painless weakness and atrophy of the infraspinatus muscle. The suprascapular nerve is also prone to trauma at other sites along its course including in the posterior triangle of the neck.

Electrodiagnostic approach

Evaluation of putative suprascapular nerve palsy relies largely on needle electromyography (see Box 3 on next page). The supraspinatus and infraspinatus muscles are sampled to determine the presence of suprascapular nerve involvement, the likely site of susprascapular nerve injury, and the severity and acuity thereof.

At a minimum, other C5/6-innervated limb muscles (deltoid, biceps, ±Rhomboideus) and C5/6 cervical paraspinal muscles should be sampled

Box 3
Suggested electrodiagnostic approach to suprascapular neuropathies

Nerve conduction studies (ipsilateral)
  Antidromic median and ulnar sensory responses (digits 2 and 5)
  Ulnar and median motor responses, forearm conduction velocities and F responses

Needle examination
  Supraspinatus, infraspinatus, deltoid, biceps, C5/6 paraspinal muscles

Optional
Rhomboids, serratus anterior, triceps, flexor carpi radialis, flexor pollicis longus, first dorsal interosseous

to exclude a C5/6 radiculopathy or an upper trunk brachial plexopathy. If the diagnosis of brachial neuritis is a possibility (suprascapular palsies can be a dominant feature of brachial neuritis), then it may be necessary to sample other muscles (eg, serratus anterior and flexor pollicis longus (FPL)).

The authors obtain upper extremity NCS to exclude a coexistent brachial plexopathy or evidence of HNPP (slowed distal sensory and motor conduction velocities). NCS techniques are available for the suprascapular nerves. The suprascapular nerves may be studied bilaterally by stimulating over Erb's point, with monopolar needle recordings from the infraspinatus and the supraspinatus muscles. A side-to-side delay, or segmental prolongation of latency, may be of localizing value (eg, a prolonged latency with recording from the infraspinatus muscle, but not the supraspinatus, suggests entrapment at the spinoglenoid notch) [8]. The authors do not routinely perform suprascapular NCS, as they add little to a careful needle electromyography (EMG) examination and, in their opinion, should not be relied on for localization of a suprascapular neuropathy.

Pitfalls

Care needs to be taken to avoid pneumothorax when performing needle EMG of the supraspinatus and serratus anterior muscles [9]. Also, because infraspinatus lies deep to the lower trapezius muscle, needle examination generally requires deep insertion of the electrode down to the periosteum of the scapula bone to ensure that EMG activity arises from the infraspinatus muscle [9].

Axillary neuropathies

The axillary nerve consists of sensory and motor fibers that derive from C5 and C6 roots. It is one of the terminal branches of the posterior cord of

the brachial plexus and provides innervation to the deltoid and teres minor muscles [10]. The axillary nerve supplies cutaneous sensation to a patch of skin over the upper lateral arm. Axillary mononeuropathies are characterized by weakness of shoulder abduction, typically with localized sensory loss over the upper lateral arm. Axillary mononeuropathies may result from shoulder trauma (eg, dislocations of the glenohumeral joint) or occasionally as a manifestation of brachial neuritis [3,11].

Electrodiagnostic approach

Needle examination forms the basis of the electrodiagnostic assessment (see Box 4 on next page). The objective is to demonstrate selective denervation or reinnervation of the deltoid and teres minor muscles. The authors study all three components of the deltoid muscle (posterior, middle, and anterior) and teres minor because selective patterns of reinnervation may be seen.

Other causes of shoulder abduction weakness (eg, a C5/6 radiculopathy, upper trunk brachial plexopathy, and suprascapular nerve palsy) should be excluded. The authors study the triceps muscle to exclude a posterior cord lesion. Axillary motor NCS are easily and reliably performed, stimulating percutaneously at Erb's point and recording over the mid-deltoid with surface electrodes [12]. Side-to-side comparison of deltoid compound muscle action potential (CMAP) amplitudes is helpful in estimating the degree of axon loss. There are no nerve conduction techniques to directly assess the sensory branch of the axillary nerve.

"Weakness about the elbow"

Musculocutaneous neuropathies

The musculocutaneous nerve originates from the lateral cord and is composed of sensory and motor fibers that traverse C5–7 nerve roots and the upper trunk of the brachial plexus [10]. It innervates the coracobrachialis, brachialis, and biceps brachii muscles. It also provides sensation to the lateral arm and forearm through the lateral cutaneous nerves of the arm and forearm; the latter nerve represents the terminal extension of the musculocutaneous nerve [10]. The musculocutaneous nerve is prone to injury at the level of the coracobrachialis, and the distal sensory branch is vulnerable as it pierces fascia to enter the forearm because its position is relatively fixed at both sites [7]. Proximal injury results in weakness of elbow flexion and shoulder adduction, an absent or reduced biceps reflex, and reduced sensation over the lateral aspect of the volar forearm. Distal injury at the elbow produces a pure sensory syndrome with pain and tenderness in the cubital fossa, and sensory loss over the lateral volar forearm to the level of the wrist [7,10].

Electrodiagnostic approach

The goal of electrodiagnostic testing is to demonstrate selective involvement of musculocutaneous-innervated muscles on needle EMG and selective

Box 4
Suggested electrodiagnostic approach to axillary neuropathies

Nerve conduction studies
- Median or radial sensory responses (record from the thumb with side-to-side comparison)
- Median motor responses and F waves
- Axillary motor responses (stimulate at Erb's point)—perform bilaterally

Needle examination
- Deltoid (anterior, middle, posterior), teres minor, infraspinatus, biceps, triceps, C5/6 paraspinals

abnormalities of the lateral cutaneous nerve of the forearm on sensory NCS (see Box 5). A C5/6 radiculopathy or a predominantly upper trunk brachial plexopathy should be excluded as they may mimic the pattern seen with musculocutaneous nerve lesions.

The authors perform lateral antebrachial cutaneous (LAC) sensory NCS, with side-to-side comparison of amplitudes [13]. The authors also obtain radial and median sensory responses (recording over the thumb with side-to-side comparison) to exclude involvement of other sensory fibers that traverse the upper trunk or lateral cords of the brachial plexus.

Bilateral musculocutaneous motor NCS can be performed by stimulating at Erb's point and at the axilla, and recording the biceps CMAPs with surface electrodes. Side-to-side comparison of amplitudes is essential. Latency comparisons are unreliable [14]. The authors do not routinely obtain musculocutaneous motor conduction studies because similar information can generally be obtained on needle examination. However, in cases where conduction block (eg, multifocal motor neuropathy) is suspected, musculocutaneous motor NCS may be helpful.

Needle examination is essential. The authors study the biceps brachii muscle to assess for active denervation, axonal continuity, and the degree of reinnervation. Although the brachialis muscle is easily studied, it provides little additional information and may receive dual innervation from the radial nerve [7,15]. The authors do not routinely examine the coracobrachialis muscle because it is more difficult to access and is typically spared in proximal musculocutaneous nerve palsies [7].

The authors do study other C5–7-innervated muscles (infraspinatus, deltoid, triceps, and cervical paraspinals) to exclude a cervical radiculopathy or a predominantly upper trunk brachial plexopathy.

Distal musculocutaneous nerve lesions at the elbow manifest as isolated abnormalities on LAC nerve testing in the distal forearm. A >50% reduction in the LAC sensory nerve action potential (SNAP) amplitude on the affected

Box 5
Suggested electrodiagnostic approach to musculocutaneous neuropathies

Nerve conduction studies
- Lateral antebrachial cutaneous nerve responses bilaterally
- Median (± radial) sensory responses recording from the thumb bilaterally
- Musculocutaneous motor conduction studies bilaterally (optional)

Needle examination
- Biceps, deltoid, triceps, cervical paraspinals (C5/6)
- Coracobrachialis (optional)

side is considered abnormal [16]. LAC nerve responses may be difficult to elicit (even on the "normal side") in obese or older patients.

"Wrist or finger drop"

Radial neuropathies

The radial nerve is composed of motor fibers from C5–8 (infrequently T1) nerve roots and sensory fibers that arise in the C5–8 dorsal root ganglia. Nerve fibers destined to form the radial nerve traverse all three trunks of the brachial plexus and the posterior cord. From an electrodiagnostic standpoint, it is useful to consider radial neuropathies as occurring at four anatomic levels: (1) axilla/upper arm above the spiral groove, (2) at or just distal to the spiral groove, (3) posterior interosseous neuropathies, and (4) superficial radial neuropathies [17–19].

The radial nerve—a terminal branch of the posterior cord—innervates the long and medial heads of the triceps in the axilla. It supplies the lateral head of the triceps—the anconeus muscle—and gives off the posterior cutaneous nerves of the arm and forearm in the upper arm above the spiral groove. Between the spiral groove and the elbow, it innervates brachialis (partial), brachioradialis, extensor carpi radialis longus and, in some individuals, extensor carpi radialis brevis. At the elbow, the radial nerve divides into its two terminal branches: the posterior interosseous nerve (PIN) and the superficial radial nerve (SRN). The PIN passes under the fibrous arcade of Frohse and enters the supinator. It contains purely motor fibers and innervates extensor carpi radialis brevis, extensor digitorum communis, extensor digiti minimi, extensor carpi ulnaris, abductor pollicis longus, and extensor pollicis longus and brevis. Finally it innervates extensor indicis proprius (EIP) [20].

The SRN travels deep to the brachioradialis in the anterolateral forearm. In the distal third of the forearm, it passes to the posterior aspect of the radial forearm and then over the tendons of the anatomic snuffbox where it can be easily palpated. It supplies cutaneous sensation to the posterolateral hand and the proximal portions of the dorsum of the thumb and digits 2 to 4 [15].

High radial neuropathies ("above spiral groove") present with weakness of elbow extension (triceps), mild weakness of elbow flexion (brachioradialis, weakness that may be difficult to elicit, but by inspection is easily seen not to contract), and wrist and finger extension. If the lesion is as proximal as the posterior cord, then there is weakness of the deltoid and latissimus muscles as well. There may be variable loss of sensation over the posterior arm, forearm, and hand. The triceps and brachioradialis reflexes are lost or reduced. The more common spiral groove lesion is similar but, importantly, spares the triceps. PIN neuropathies are purely motor, and manifest as finger drop, with variable weakness of wrist extension and radial deviation of the extended wrist. Elbow extension, and flexion are normal [17]. SRN neuropathies (cheiralgia paresthetica) manifest with isolated pain, numbness, and paresthesias over the dorsolateral aspect of the hand and the dorsum of the proximal thumb and digits 2 to 4 [19].

Electrodiagnostic approach

Electrodiagnostic testing demonstrates abnormalities confined to the radial nerve territory (see Box 6 on page 460). The electromyographer localizes the level, severity, and chronicity of the radial nerve lesion, and assesses whether the neuropathy is primarily demyelinative (conduction block) or axon loss in nature [21]. It is necessary to exclude other lesions that may mimic a radial neuropathy, including a C7 radiculopathy, posterior cord lesion, multifocal motor neuropathy, or lead neuropathy, and a central nervous system process (eg, stroke) [20].

The authors obtain radial sensory responses (forearm) recording over the superficial radial nerve at the anatomic snuffbox. If responses are in the low-normal range or abnormal, the authors test the contralateral side, accepting a >50% amplitude difference side-to-side as being abnormal (on the side of the lower amplitude) [16]. In young adults, it is particularly important to obtain side-to-side superficial radial studies because a response amplitude on the affected side that is in the low-normal range may, in fact, be low for someone who otherwise has responses in the upper-normal range at baseline.

The authors obtain radial motor NCS bilaterally. The authors use surface recording over the EIP muscles, and stimulate the radial nerve in the mid-posterolateral forearm, antecubital fossa (lateral to the biceps tendon) and in the lateral arm above and below the spiral groove (Fig. 1). In cases where radial nerve compression is suspected in the axilla, the authors may also stimulate more proximally at the level of Erb's point to demonstrate conduction block across the axilla. Side-to-side comparison of the radial motor distal CMAP amplitude provides a useful index of the degree of axon loss,

as early as 1 week following the onset of the neuropathy. Motor conduction studies may be helpful in localizing the lesion, as in the commonly encountered conduction block at the level of the spiral groove (see Fig. 1) or, uncommonly, the conduction block seen (between the forearm and elbow) with PIN lesions. Measurement of segmental conduction velocity is usually not helpful due to problems with distance measurement.

Needle examination is important in the evaluation of radial neuropathies. It serves to localize the level of the lesion, exclude C6/7 radiculopathies and posterior cord plexopathies, and provides information regarding the severity, activity, and chronicity of the lesion.

The authors study the triceps brachii, brachioradialis, extensor digitorum communis, and EIP muscles to confirm involvement of radial-innervated muscles, and to localize the level of the lesion to above the spiral groove, at or below the spiral groove, or to the PIN. The authors also assess nonradial-innervated C6/7 muscles (eg, flexor carpi radialis) to exclude a radiculopathy, and the deltoid to evaluate the posterior cord.

In the case of radial nerve lesions above the spiral groove, abnormality is expected on needle examination within the triceps muscle and in more distal

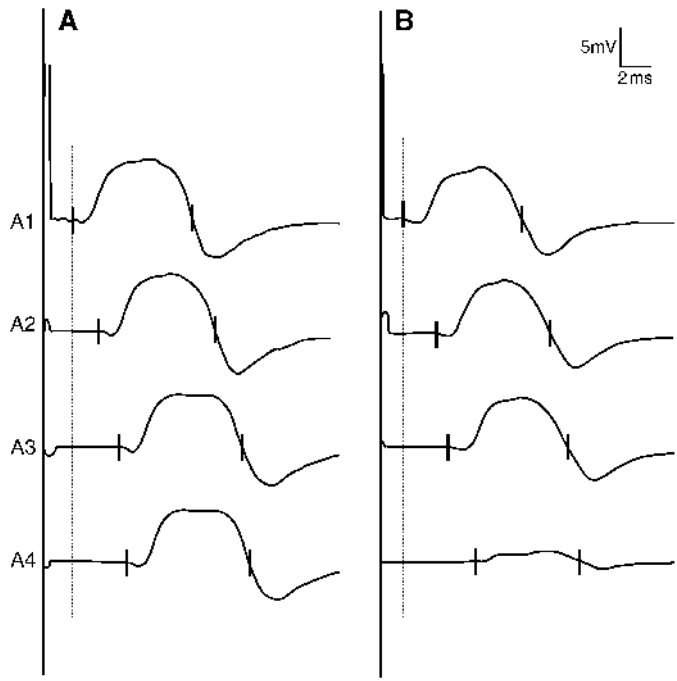


Fig. 1. (A) A normal radial motor NCS (surface recording from the EIP), with stimulation in the forearm (A1), elbow (A2), below the spiral groove (A3), and above the spiral groove (A4). (B) A radial neuropathy with motor conduction block across the spiral groove. The distal radial CMAP amplitude (A1) is similar to the unaffected side (A), suggesting a primarily demyelinative lesion, and a good prognosis.

radial-innervated muscles. The superficial radial response is usually reduced in amplitude, except in very acute lesions (within the first 10 days) or, in purely demyelinating lesions, where conduction block may be present in sensory fibers, producing radial sensory loss, yet a normal distally recorded superficial radial response. Lesions at the spiral groove spare the triceps, but involve more distal radial-innervated muscles including brachioradialis, which receives its innervation from the radial nerve just distal to the spiral groove. The SRN is affected, as in high radial neuropathies. Radial neuropathies at the spiral groove are frequently demyelinative (neuropraxic), thus, with the distal CMAP amplitude on the symptomatic side being comparable to the asymptomatic side. Radial neuropathies below the level of the spiral groove, but above the elbow, spare the brachioradialis muscle but affect distal radial and PIN-innervated muscles and the SRN.

PIN neuropathy with entrapment at the arcade of Frohse is usually an axon loss lesion, and spares the triceps and brachioradialis muscles, with variable involvement of wrist extensors (eg, extensor carpi ulnaris). Extensor digitorum communis and EIP should show abnormality with acute denervation and/or reduced recruitment and, in more chronic lesions, signs of reinnervation. The superficial radial response is normal. This lesion is infrequently demyelinative.

Box 6
Suggested electrodiagnostic approach to radial neuropathies

Sensory nerve conduction studies (NCS)
- Superficial radial nerve (bilaterally) (stimulate forearm, record snuffbox)
- Posterior cutaneous nerve of forearm (rarely required)

Motor NCS
- Radial motor NCS bilaterally (record from extensor indicis proprius EIP, stimulate forearm, antecubital fossa, and arm above and below spiral groove
- Ulnar and other motor NCS (if a brachial plexopathy, lead neuropathy, multifocal motor neuropathy, multifocal acquired demyelinating sensory and motor neuropathy, etc. suspected)

Needle examination
- Triceps, brachioradialis, extensor digitorum communis, EIP (to localize radial nerve/posterior interosseous nerve involvement)

Flexor carpi radialis, first dorsal interosseous, cervical paraspinals to exclude a C7,8 radiculopathy; deltoid to exclude a posterior cord lesion

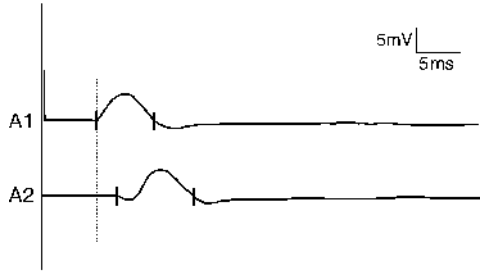
"Pain and weakness/numbness in the forearm/wrist/hand"

Median neuropathy in the arm/forearm

Median nerve fibers derive from C6–T1 nerve roots, and traverse all three trunks and the medial and lateral cords of the brachial plexus. Median sensory fibers (C6/7 dorsal root ganglia, upper and middle trunks, lateral cord) that provide cutaneous innervation in most individuals to the thumb, digits 2 and 3, and the lateral half of digit 4, pass through the carpal tunnel. The palmar cutaneous branch arises from the median nerve just proximal to the wrist, and travels anterior to the carpal tunnel to provide sensation to the thenar eminence [15].

Median motor fibers originate from nerve roots C6–T1, all three trunks, and the medial and lateral cords of the brachial plexus. The first major motor branches supply pronator teres. Subsequent branches innervate flexor digitorum superficialis and flexor carpi radialis and, finally, the large purely motor branch, the anterior interosseous nerve (AION) in the proximal forearm. The AION innervates flexor pollicis longus, flexor digitorum profundus (FDP) subserving digits 2 and 3, and pronator quadratus. The median nerve then passes through the carpal tunnel, and innervates abductor pollicis brevis (APB), opponens pollicis, the superficial head of flexor pollicis brevis, and the first and second lumbricals [15].

Anatomic variants (eg, median to ulnar crossovers in the forearm: Martin–Gruber anastomoses) and median-to-ulnar crossovers in the hand (Riche Cannieu anastomoses) occasionally complicate the clinical and electrodiagnostic picture in median neuropathies (Fig. 2) [22].



Stimulus Site	Lat1 ms	Amp mV	CV m/s
A1: Wrist	6.0	5.325	
A2: Elbow	8.3	5.534	87

Fig. 2. A Martin–Gruber anastomosis in a patient with a MNW. The higher median CMAP amplitude at the elbow (A2) compared with the wrist (A1) stimulation site suggests a crossover of median-to-ulnar nerve fibers in the forearm. The initial positive dip seen with median stimulation at the elbow (but not at the wrist) results from innervation of thenar muscles by crossover fibers that are not slowed in the carpal tunnel. The crossover fibers and initial positive dip in median CMAP (elbow stimulation) cause a spuriously fast median motor conduction velocity in the forearm.

In the proximal arm, the median nerve may be subject to external compression (eg, crutch palsy in the axilla/upper arm) or to trauma from humeral fractures [15]. Proximal entrapments occur at four sites [23,24]: (1) the Ligament of Struthers in the distal arm (rarest); (2) the Lacertum fibrosis (fibrous entrapment of the median nerve in the antecubital fossa); (3) between the hypertrophied heads of pronator teres (the most common entrapment site for proximal median neuropathies); and (4) at the level of the flexor digitorum superficialis. The AION may be entrapped at tendinous origins of the deep head of the pronator teres or of the flexor digitorum superficialis. The AION may also be entrapped at an accessory head of the flexor pollicis longus. Distal median nerve entrapment invariably occurs within the carpal tunnel (described later).

Proximal median neuropathies are variable in their manifestations. In the mildest cases, pain and paresthesias predominate [25]. Median neuropathies in the arm present with pain in the distal arm and forearm, a sensory disturbance involving median-innervated digits and the thenar eminence, and weakness of both AION and median nerve-innervated muscles including pronator teres. Median nerve entrapment in the forearm, either at the level pronator teres or flexor digitorum superficialis, presents similarly, although the pronator teres muscle is always spared in flexor digitorum superficialis syndromes and classically, but not always, spared in the pronator syndrome [27,28]. AION neuropathy, a purely motor disorder, manifests with volar forearm discomfort and flexor weakness of the terminal phalanx of the thumb [27].

Electrodiagnostic approach

NCS techniques have been described for selective evaluation of the AION and for the pronator syndrome [28–30]. The authors do not generally employ these techniques in their laboratory because the techniques add little to the clinical evaluation and are subject to significant technical limitations. The authors initially perform standard ulnar and median motor and sensory conduction studies (see Box 7). The results are frequently normal in proximal median neuropathies, but serve to exclude entrapment at more distal sites (eg, the carpal tunnel) or the presence of Martin–Gruber anastomoses [31]. The needle examination is most helpful in confirming and localizing proximal median neuropathies and assessing the degree of axon loss [26,32]. The authors study the pronator teres, flexor carpi radialis, flexor pollicis longus, and APB muscles to confirm involvement of median-innervated muscles, and to assess the likely site of the lesion. If an AION syndrome is suspected, the authors also assess pronator quadratus. The authors study nonmedian nerve-innervated C6–T1 muscles (first dorsal interosseous [FDI], triceps, biceps, and cervical paraspinal) to exclude cervical radiculopathy or brachial plexopathy. Because brachial neuritis may present with relatively selective involvement of the AION, the authors typically evaluate

Box 7
Suggested electrodiagnostic approach to proximal median neuropathies

Sensory nerve conduction studies (NCS)
Median and ulnar antidromic sensory responses (digits 2 and 5)

Motor NCS
Median motor NCS (record from abductor pollicis brevis (APB)), ulnar motor NCS (record from abductor digiti quinti (ADQ))

Needle examination
Pronator teres, flexor carpi radialis, flexor pollicis longus, APB, and pronator quadratus (if anterior interosseous nerve syn drome suspected)
Study: triceps, biceps, first dorsal interosseous, lower cervical paraspinal muscles to evaluate for C6–8 radiculopathy or brachial plexopathy

muscles commonly involved in brachial neuritis (eg, spinatii and serratus anterior) in patients with AION syndromes [33].

Median neuropathy at the wrist (MNW)

MNW is the most common entrapment neuropathy. A suggested algorithm for the evaluation of MNW is detailed later in this section.

Several points deserve special consideration:

- In studies from the Mayo Clinic, median motor distal latencies were prolonged in just 51% and median sensory peak latencies were abnormal in only 64% of subjects with carpal tunnel syndrome [34]. The authors thus perform one or more additional internal comparison studies between the median and ulnar/radial sensory nerves when carpal tunnel syndrome is suspected.
- For each individual NCS, 2.5% of the normal population will be misclassified as abnormal when using commonly employed mean ±2 SD reference ranges [2]. Studies designed to increase diagnostic sensitivity (eg, use of short nerve segments and comparison of more than one nerve) increase the likelihood of technical errors. The authors thus require abnormalities on *two separate tests* of median nerve function that localize to the carpal tunnel to make a diagnosis of MNW.
- The fascicular arrangement of the median nerve within the carpal tunnel is such that individual fascicles may be variably affected. It is important to individualize testing according to the patient's symptoms. It is appro-

priate, for instance, to assess sensory responses from digit 3 (rather than digit 2) if this is the most symptomatic digit.

- The concept of "double crush" as it applies to median neuropathy and the risk of coexistent cervical radiculopathy is controversial [35]. Nonetheless, when median nerve abnormalities across the wrist are minimal, when symptoms are atypical of carpal tunnel syndrome, or when clinical features suggest a coexistent cervical radiculopathy, the authors perform additional needle studies to examine this possibility.
- "Motor only" carpal tunnel syndrome is uncommon (incidence of 3.5% in the Mayo Clinic series) [34]. In this situation, C8/T1 radiculopathy or focal onset motor neuron disease deserve consideration.
- Martin–Gruber anastomoses and carpal tunnel syndrome may co-exist (see Fig. 2). The finding of an initial positive "dip" in the median CMAP (APB), present with stimulation at the elbow (but not with stimulation at the wrist) implies a MNW, in the setting of a Martin–Gruber anastomosis. The initial positive dip reflects median-to-ulnar crossover fibers that pass through the ulnar rather than the carpal tunnel. They reach their target thenar muscle fibers (e.g., adductor pollicis, flexor pollicis brevis) before median fibers—focally slowed in the carpal tunnel—reach their APB target. The presence of an initial positive dip gives rise to a spuriously fast "median" forearm conduction velocity [22].
- MNW and polyneuropathy frequently coexist [36]. Thus, depending on the clinical circumstances, electrodiagnostic screening for underlying polyneuropathy may be warranted (see later discussion). Conversely, the identification of MNW may be difficult in subjects with polyneuropathies (in particular, diabetes) [34]. In instances of severe sensory polyneuropathy, where sensory responses are absent in the upper extremities, a comparison between median and ulnar motor distal latencies and between the lumbrical–interosseous distal latencies may be helpful to demonstrate segmental slowing across the carpal tunnel (see later discussion) [37].
- The severity of the MNW, as determined by electrodiagnostic studies, is often used to guide therapy. Various empiric severity grading scales have been suggested [34]. Sensory and motor latencies are reported to correlate poorly with the degree of clinical symptomatology [34]. Historically, sensory and motor axon loss (reduced SNAP and CMAP amplitudes, and signs of denervation on needle examination) and clinical evidence of median nerve sensory or motor deficits suggested the need for surgical intervention [34]. The authors empirically grade MNW as *mild* when median sensory or motor slowing occurs without evidence of sensory or motor axon loss; as *mild-to-moderate* when median sensory or motor slowing is accompanied by mildly reduced median SNAP amplitudes or mild chronic reinnervation; as *moderate* when median sensory or motor slowing occurs with moderate sensory or motor axon loss (eg, moderate reductions in median SNAP or CMAP amplitudes, or

moderate chronic partial denervation/reinnervation); and *severe* when the median SNAP (at wrist or palm) is unobtainable or when a severe reduction of median CMAP amplitude is present with active denervation or severe chronic denervation/reinnervation.

- Electrodiagnostic studies are often requested on subjects who have undergone carpal tunnel release surgery and in subjects whose symptoms have persisted, become worse, incompletely resolved, or recurred. Median NCS results generally improve after surgery and relate to a decrease in symptoms. This improvement in NCS results usually occurs within 6 weeks after surgery [34]. However, because nerve conduction abnormalities may not fully resolve after surgery (despite relief of symptoms), the electromyographer should be conservative in interpreting residual postsurgical abnormalities. In this situation, the authors try to obtain the preoperative NCS for comparison. If the MNW has shown interval deterioration both clinically and electrodiagnostically, then recurrent carpal tunnel syndrome is likely. If NCS results are normal or improved from the preoperative study, the authors consider other possible causes for the symptoms (e.g., cervical radiculopathy). In cases where NCS results are unchanged or preoperative conduction studies are not available and the median nerve abnormalities are of a mild nature, follow-up studies may be of value if symptoms progress [34].

Specific techniques in the evaluation of MNW

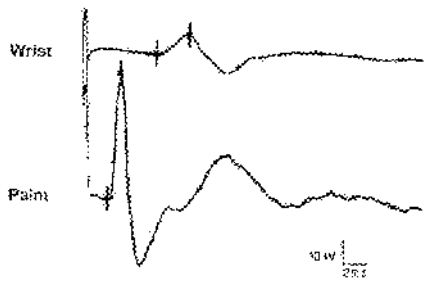
NCS performed in all patients. The authors obtain median and ulnar CMAPs, conduction velocities, and F responses in the symptomatic limb or limbs (see Box 8 on page 468). The authors perform antidromic median sensory studies (recording from digit 2 or the most symptomatic digit), with stimulation of the median nerve at the wrist (proximal to the carpal tunnel) and in the palm distal to the carpal tunnel. The authors also obtain ulnar sensory responses, recording from digit 5, with stimulation at the wrist. Criteria for abnormality of the median sensory onset or peak latency should be established by each electrodiagnostic laboratory, controlling for temperature, age, body mass index, and distance [38–40]. Stimulation of median sensory fibers at the wrist and in the palm permits localization of slowing of median sensory conduction velocities to the carpal tunnel segment. In our laboratory, slowing of conduction velocities in the wrist to palm, relative to the palm-to-digit segment, of 13 m/s or more is considered abnormal [37,41]. This technique requires digital averaging of responses, uniform warming of digits and the hand, careful measurement of stimulation distances, and an even baseline without significant shock artifact. It is not uncommon for symptomatic subjects with ''normal'' wrist-to-digit sensory latencies to have significant slowing across the wrist segment. In addition, carpal tunnel syndrome patients with presumptive axon loss based on a low amplitude wrist-to-digit sensory response are often found to have a demyelinative lesion when the palm-to-digit potential shows a normal

amplitude response (Fig. 3). Conversely, segmental stimulation may demonstrate that a prolonged median sensory peak latency is due to diffuse slowing, as in an axonopathy.

The authors perform one or more of the following internal comparison studies if criteria for MNW are not met on two of the previously mentioned studies:

Comparison of median sensory latencies to ulnar or radial sensory latencies. Comparisons of antidromic median and ulnar sensory latencies to digit 4 permit use of a single placement of recording electrodes [42]. The median and ulnar nerves each are stimulated separately at the wrist using the same stimulation distance. A median sensory latency that exceeds the ulnar latency to digit 4 by ≥0.5 milliseconds is considered abnormal. The median latency to digit 2 can similarly be compared with the ulnar response recorded from digit 5 if the same stimulation distances are used for each nerve. If an ulnar neuropathy is present, the median sensory latency recorded from the thumb can be compared with the superficial radial sensory latency recorded from the thumb [43].

Comparison of median and ulnar mixed nerve (midpalmar) latencies. Comparison of the latency difference (peak or onset) between the mixed median



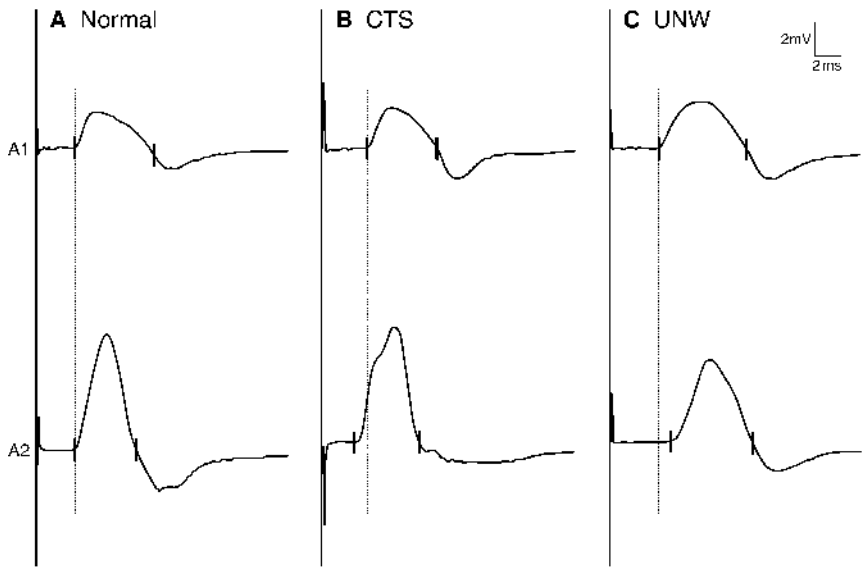
Stimulus Site	Latency (ms)	Amplitude (uV)
Wrist	4.5	5.25
Palm	1.7	34.97

Segment	Distance (mm)	Conduction Velocity (m/s)
Digit 2- Wrist	160	35
Digit 2 - Palm	80	47
Wrist - Palm	80	29

Fig. 3. Antidromic stimulation of median sensory fibers at the wrist and in the palm (recording from digit 2) shows a dramatically reduced median SNAP amplitude with wrist stimulation, relative to a normal median SNAP amplitude with palm stimulation, in a patient with carpal tunnel syndrome. This finding suggests focal demyelination of median sensory fibers across the carpal tunnel without significant axon loss. Note also that there is a segmental drop of median conduction velocity in the palm-to-wrist versus palm-to-digit nerve segments.

and ulnar nerves across the carpal tunnel increases the diagnostic yield of standard median motor and sensory studies by about 21% [34]. Focal slowing of the median nerve within the carpal tunnel is more evident using this technique because of the shorter stimulation distance (8 cm). The median and ulnar nerves are stimulated separately, in the midpalm, and bar recording electrodes are placed over the respective nerves 8 cm proximal to the site of stimulation (just proximal to the distal wrist crease). In their laboratory, the authors consider a ≥0.4-millisecond latency difference (ie, longer for the median nerve) as abnormal. This is somewhat controversial because various laboratories accept anywhere between a 0.3- and 0.5-millisecond difference as significant [34].

Comparison of lumbrical (median) and interosseous latencies (ulnar) (Fig. 4). Through placement of surface electrodes (active just radial to the middle of the third metacarpal, and reference over the proximal interphalangeal joint), one may record an interosseous CMAP if the ulnar nerve is stimulated at the wrist and a lumbrical CMAP if the median nerve is stimulated at the wrist. If a standard stimulation distance of 8 to 10 cm is used for separate stimulation of the median and ulnar nerves, a lumbrical distal



Stimulus Site	Lat1 ms	Amp mV
A1: Wrist-median	3.1	2.22
A2: Wrist-ulnar	3.0	7.02

Stimulus Site	Lat1 ms	Amp mV
A1: Wrist-median	3.5	2.568
A2: Wrist-ulnar	2.5	7.198

Stimulus Site	Lat1 ms	Amp mV
A1: Wrist-median	3.9	2.88
A2: Wrist-ulnar	4.8	5.10

Fig. 4. (A) A normal lumbrical–interosseous study. The median (A1) and ulnar nerves (A2) are stimulated at the wrist using identical stimulation distances (8–10 cm), with the active recording electrode just radial to the middle of the third metacarpal. The lumbrical and interosseous distal latencies are comparable. (B) CTS: the lumbrical (median) distal latency exceeds the interosseous (ulnar) distal latency by 1 millisecond. (C) UNW: the interosseous (ulnar) distal latency is 0.9 milliseconds longer than the lumbrical distal latency.

latency of ≥0.6 milliseconds longer than the interosseous latency is indicative of a MNW. It is important that the recording electrode be adjusted such that the lumbrical potential has a short rise time. This technique is particularly useful in severe median neuropathies at the wrist, where median and ulnar sensory responses and the median motor response recording from APB may be absent [37,44,45].

Studies with low sensitivity or specificity. The terminal latency index and residual latency has a low sensitivity, and the authors do not calculate this in

Box 8
Suggested electrodiagnostic approach to median neuropathy at the wrist (MNW)

Routine
- Antidromic median sensory responses (digit 2 or 3): stimulate wrist and palm, compute amplitude, onset, and peak latency, and conduction velocity across wrist segment. A >10 m/s drop in conduction velocity across the wrist is abnormal.
- Median motor nerve conduction studies (NCS) and F response (record abductor pollicis brevis (APB), stimulate wrist and elbow): distal latency >4.4 milliseconds is abnormal under age 60 (stimulation distance 7 cm)
- Ulnar motor (record abductor digiti quinti) and sensory NCS (record digit 5)

If above studies are normal or if only one piece of data supports MNW, the authors perform one or more internal comparison studies:
- Median-to-ulnar palmar comparison (abnormal if median latency ≥0.4 milliseconds longer)
- Comparison of median-to-ulnar antidromic sensory responses recorded from digit 4 using the same stimulation distance (11–14 cm) for each nerve (abnormal if median latency ≥0.5 milliseconds longer)

Needle examination
- APB, if abnormal assess first dorsal interosseous; the authors also examine flexor carpi radialis (FCR) and triceps. If coexistent radiculopathy is suspected, a more detailed examination is done including cervical paraspinals. If FCR abnormal, triceps normal, more detailed examination of other proximal median-innervated muscles is done to exclude median neuropathy at the elbow.

their laboratory [2]. The authors perform F responses to exclude a more proximal lesion or underlying demyelinating polyneuropathy. Minimal F wave latencies may be prolonged in proportion to the prolongation in median motor distal latencies, however they are of no localizing value. Median motor conduction velocities may be mildly slowed in a minority of subjects with MNW, presumably due to conduction block or axon loss of the fastest median motor fibers at the wrist. This finding does not imply a proximal median neuropathy.

Needle examination. The authors routinely perform a needle examination in the evaluation of possible MNW. Needle examination of APB serves to assess the severity, activity, and chronicity of the median neuropathy. A C6–C8 radiculopathy, proximal median neuropathy, or brachial plexopathy should be excluded in subjects with hand numbness, tingling, or weakness when MNW is not evident on NCS results or when the finding of mild MNW is insufficient to explain the clinical presentation.

Ulnar neuropathy at the elbow (UNE)

UNE is the second most common focal mononeuropathy [46]. The ulnar nerve consists of motor and sensory fibers that arise in C8–T1 roots and associated dorsal root ganglia, and travels in the lower trunk and medial cord of the brachial plexus [15]. The ulnar nerve provides sensation to digit 5, the medial half of digit 4, the hypothenar eminence (superficial and palmar cutaneous branches that arise just proximal to Guyons canal), and the dorsomedial aspect of the hand (dorsal cutaneous nerve that arises above the wrist). The motor branch to flexor carpi ulnaris arises at or above the level of the cubital tunnel (humeroulnar arcade) and the flexor digitorum profundi (digits 4/5) arises in the humeroulnar arcade. In the hand, it innervates the hypothenar muscle group (eg, abductor digiti quinti; ADQ), and a deep motor branch that arises in Guyon's canal, innervates lumbricals 3/4, palmar and dorsal interossei, flexor pollicis brevis (deep head), and adductor pollicis brevis.

Manifestations of UNE range from elbow pain and intermittent paresthesias of the medial hand to marked sensory loss, wasting and weakness, and a claw hand. Sensory loss over the dorsum of the hand and weakness of FDP 4/5 localize the ulnar neuropathy to above the wrist. In UNE, compression typically occurs at either the retroepicondylar groove (located 0–2 cm above the medial epicondyle), or the humeroulnar arcade, typically located 0 to 3 cm below the medial epicondyle [47,48]. Entrapment just above the elbow at the arcade of Struthers or more distally at the deep flexor-pronator aponeurosis is less common [1]. Compression at each of these sites produces an indistinguishable clinical syndrome.

Most cases of UNE are chronic, and manifest electrophysiologically as a primarily demyelinating lesion (segmental conduction slowing across the

elbow), an axon loss lesion, or a combination of the two. About 6% of patients with UNE have acute motor conduction block across the elbow [48]. Such cases usually have an acute or subacute presentation [49]. The differential diagnosis of UNE includes ulnar neuropathy at the wrist (UNW), a lower trunk or medial cord brachial plexopathy, C8/T1 radiculopathy, or early motor neuron disease. UNE may be a clue to an underlying polyneuropathy (eg, diabetes mellitus, Hereditary neuropathy with liability to pressure palsy).

Electrodiagnostic approach

Routine NCS. The authors study antidromic ulnar (digit 5) and median (digit 2) sensory responses in all subjects (see Box 9). The authors obtain ulnar motor NCS, recording from the ADQ, with stimulation at the wrist below elbow and above elbow. The authors perform ulnar motor NCS with the elbow flexed 70 to 90°, and with a 10-cm stimulation distance between above and below elbow sites. In this position, measured distances between the recording and stimulating electrodes better approximate the length of the ulnar nerve by reducing slack in the nerve that occurs with elbow in the extended position. Consequently, in the extended position, underestimation of the length of the ulnar nerve may result in spuriously low conduction velocities across the elbow [50].

Ulnar SNAP amplitudes reduced below 10 μV, CMAPs reduced to below 7 mV, or absolute ulnar motor conduction velocities in the above-to-below elbow segment of <50 m/s suggest ulnar nerve involvement, but do not localize the site of the lesion [51].

Ulnar motor NCS is the most important study for localization of UNE. With the elbow in the flexed position, ≥11 m/s slowing in conduction velocities across the elbow relative to the forearm is considered significant [50]. If recording from the ADQ is nonlocalizing, the authors record from the FDI because selective fascicular involvement may occur [52]. In one study [52], slowing of ulnar motor conduction velocities across the elbow relative to the forearm (elbow flexed 70–90°) has been found in 71% of UNE patients when recording from the ADQ, and in 83% of cases when recording from the FDI.

Secondary criteria on routine ulnar motor NCS that localize UNE include a >20% drop in ulnar CMAP amplitude from the below elbow to the above stimulation sites (assumes a 10-cm stimulation distance) [51]. This finding likely indicates focal demyelination [51]. A drop in ulnar CMAP amplitude across the elbow of >50% (or area reduction of >40%, with <30% increase in duration) is unequivocal evidence of conduction block [53,54].

Routine motor NCS more often fail to localize UNE that is purely sensory or axon loss in type and where substantial axonal degeneration (loss of large myelinated fibers) leads to slowing in the wrist-to-elbow segment of the ulnar motor nerve [1]. In these circumstances, several alternative studies may localize UNE:

- Short-segment incremental stimulation studies (1-cm segments; across the elbow) allow precise localization of the UNE to the retroepicondylar groove or to the Humeroulnar arcade (Fig. 5) [47]. Focal slowing on short-segment incremental stimulation may be evident when routine ulnar motor NCS results are normal.
- In predominantly sensory UNE, mixed nerve stimulation studies may be helpful [52,55]. With stimulation of the ulnar nerve orthodromically at the wrist, mixed nerve responses can be recorded from the ulnar nerve above and below the elbow, and the mixed nerve conduction velocity across the elbow compared with the conduction velocity below the elbow. The authors perform this study in their laboratory with elbow in the straight position, and accept as abnormal >22 m/s slowing across the

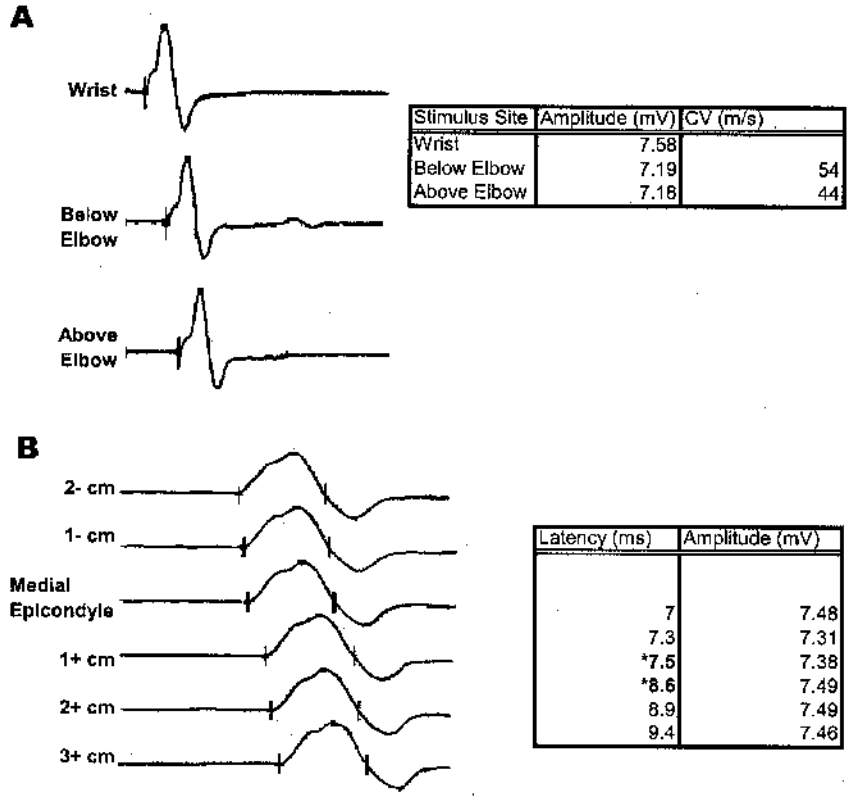


Fig. 5. UNE. (A) Standard ulnar motor NCS (recording from ADQ), with stimulation at the wrist, below elbow, and above the elbow reveal a borderline abnormal (10 m/s) drop in ulnar motor conduction velocity in the below–above elbow segment relative to the forearm. (B) Short-segment incremental stimulation of the ulnar nerve across the elbow confirms UNE with focal slowing, localized 0–1 cm above the medial epicondyle. In this segment, there is a latency shift of 1.1 millisecond, much larger than the 0.2- to 0.5-millisecond shifts seen across the other 1-cm segments.

elbow segment [52]. Mixed nerve responses are usually not obtainable when the ulnar SNAP is absent.

- The authors evaluate the dorsal ulnar cutaneous nerve bilaterally in cases where distinction between UNW and elbow remain unclear after

Box 9
Suggested electrodiagnostic approach to ulnar neuropathy at the elbow

Routine
- Antidromic ulnar and median sensory nerve conduction studies (NCS)
- Ulnar motor NCS and F responses (record from abductor digiti quinti, stimulate at the wrist, below elbow and above the elbow). A ≥11 m/s drop in conduction velocity (elbow flexed 90°), in the below-to-above elbow segment relative to the forearm is abnormal.
- Median motor NCS and F responses

If the ulnar NCS results are nonlocalizing (with normal median NCS), and the index of suspicion is high, the authors perform one or more of the following studies to localize suspected ulnar neuropathy elbow:
- Ulnar motor NCS recording from first dorsal interosseous (FDI; stimulate at the wrist, below elbow and above the elbow)
- Short-segment incremental stimulation of the ulnar motor nerve across the elbow (see Fig. 5)
- Mixed ulnar nerve stimulation (stimulate at the wrist, double channel recording below and above the elbow)
- Dorsal unlar cutaneous sensory responses (optional)

Needle examination
- FDI, flexor digitorum profundus 4, abductor pollicis brevis (APB), extensor indicis proprius (EIP) to localize ulnar involvement, and exclude a C8/T1 radiculopathy or lower trunk brachial plexopathy

If the ulnar sensory nerve action potential is normal (with side-to-side comparison), and the median compound muscle action potential low, or needle electromygraphy abnormalities are present in the APB or EIP, the authors perform the following:
- Medial antebrachial cutaneous nerve studies bilaterally, and assess low cervical/upper thoracic paraspinal muscles to distinguish a lower trunk or medial cord brachial plexopathy from a C8/T1 radiculopathy
- Median motor NCS are performed to exclude a lower trunk plexopathy or more diffuse process.

routine evaluation. An asymmetrically absent or low (<50% the amplitude of the asymptomatic side) dorsal ulnar cutaneous response localizes the lesion to the ulnar nerve above the wrist (or the medial cord or lower trunk of the brachial plexus). However, a normal dorsal cutaneous response does not exclude UNE because it is unaffected in 25% of cases of UNE due to selective fascicular involvement [56].

There are several caveats: first, as in the case of MNW, the authors require two pieces of concordant data to confirm UNE [1]. Second, short-segment incremental stimulation and mixed ulnar nerves studies have more technical limitations than routine ulnar NCS, and should only be performed by electromyographers who have an appreciation of the technical pitfalls of these studies [50]. In our opinion, they should not be used as the sole piece of data supporting UNE.

Needle examination. Needle examination aids in localization of UNE, and helps exclude lower trunk brachial plexopathy or C8/T1 radiculopathy. It provides an important measure of the severity, chronicity, and degree of axon loss. The authors routinely study FDI and FDP subserving digits 4 or 5 (FDP4 or FDP5). The finding of fibrillation potentials or reinnervation in the FDP4 muscle, in the setting of an ulnar mononeuropathy, localizes the lesion to at or above the elbow. The FDP4/FDP5 is spared in about 50% of cases of UNE. The authors rarely study the flexor carpi ulnaris; it is less often involved in UNE because the branches supplying this muscle are frequently given off above the elbow. The FDI is the most commonly involved muscle in UNE; however, abnormality in this muscle has no localizing value. The authors also study the APB and EIP muscles to exclude a lower trunk brachial plexopathy or C8/T1 radiculopathy.

"Weakness and/or numbness in the hand"

Ulnar Neuropathy at the Wrist (UNW)

UNW is suspected in the patient with isolated focal hand weakness involving ulnar-innervated muscles. Compression usually occurs at one of 5 sites [56,57]:

1. Proximal portion of Guyon's canal: involving deep (motor) and the superficial (sensory) branches of the ulnar nerve. All ulnar-innervated hand muscles are affected. There is a sensory disturbance involving the medial half of the fourth finger and the fifth finger.
2. Lesion of the superficial branch in Guyon's canal: a purely sensory disturbance involving the fifth digit and medial half of the fourth digit, sparing the dorsal ulnar cutaneous distribution.
3. A proximal lesion of the deep motor branch in Guyon's canal: pure motor disturbance with weakness of all ulnar nerve-innervated hand muscles.

4. A more distal lesion of the deep motor branch (in the region of the Hamate): weakness of the interossei, with sparing of the hypothenar muscles and superficial sensory branch.
5. A very distal lesion of the deep motor branch: isolated weakness of the FDI and adductor pollicis muscles.

Electrodiagnostic approach

Precise localization of UNW can be difficult because of the various possible sites of compression. Diagnosis and localization requires a combination of NCS and a detailed needle examination (see Box 10). UNW should be distinguished from UNE, lower trunk brachial plexopathy, a C8 radiculopathy and, in the case of a purely motor presentation, focal onset motor neuron disease. The authors obtain routine ulnar sensory and motor (recording from both ADQ and FDI) NCS. The authors also perform median motor and sensory NCS to confirm that findings are limited to the ulnar nerve territory. In UNW types 1 and 2, the ulnar SNAP (recorded from digit 5) is of low amplitude or of prolonged distal latency [58]. In UNW types 3 to 5, the ulnar SNAP is normal. Ulnar motor studies may demonstrate reduced- or normal-amplitude CMAP, with a prolonged distal latency. In UNW types 1 and 3, these abnormalities are present both with recording from the ADQ and the FDI. There should be no focal slowing of ulnar motor conduction velocity across the elbow. In UNW types 4

Box 10
Suggested electrodiagnostic approach to ulnar neuropathy at the wrist

Routine
- Ulnar and median sensory nerve conduction studies (NCS)
- Ulnar motor NCS and F responses (record from both first dorsal interosseous (FDI) and abductor digiti quinti (ADQ), stimulate at the wrist, below elbow and above elbow)
- Lumbrical-interosseous study
- Median motor NCS and F responses

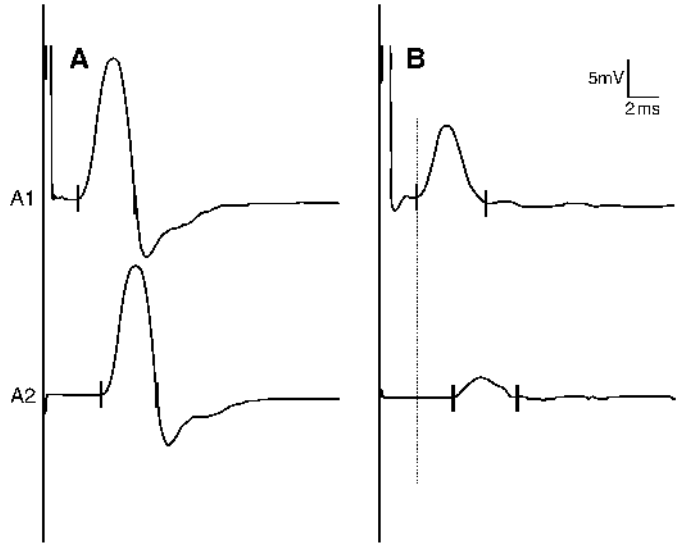
Needle examination
- FDI, ADQ, flexor digitorum profundus 4, abductor pollicis brevis, extensor indicis proprius

Optional
- Dorsal ulnar cutaneous sensory responses
- Median-to-ulnar midpalmar comparison studies
- Short-segment incremental stimulation recording from FDI (useful if conduction block is suspected)

and 5, ulnar motor responses recorded from ADQ are normal, but those from FDI are often reduced in amplitude and prolonged in latency. A side-to-side ulnar motor distal latency (recording from FDI) difference of 1.3 milliseconds or a 2-millisecond difference between the FDI and ADQ ulnar motor distal latencies is supportive of distal UNW (types 4 or 5) [60].

In addition to ulnar sensory and motor latency criteria, the authors find the lumbrical-interosseous study described above for the evaluation of MNW also to be quite useful for UNW [61]. In normal subjects, the lumbrical distal latency (median nerve stimulation at the wrist) is equal to the interosseous distal latency (ulnar nerve stimulation at the wrist). An interosseous latency >0.4 milliseconds longer than the lumbrical recording suggests UNW (see Fig. 4) [61].

The needle EMG examination is very helpful in the evaluation of possible UNW. It aids in localization of the lesion to the ulnar nerve, and in the separation of UNW types 1 and 3 from 4 and 5. The authors use the needle examination to exclude a coexistent lower trunk brachial plexopathy, a C8 radiculopathy, and focal onset motor neuron disease.



Stimulus Site	Lat1 ms	Amp mV
A1: Palm	2.3	18.41
A2: Wrist	3.9	16.91

Stimulus Site	Lat1 ms	Amp mV
A1: Palm	2.5	9.53
A2: Wrist	4.9	2.62

Fig. 6. (A) Normal ulnar motor NCS (surface recording from FDI), with stimulation in the palm (A1) and at the wrist (A2). (B) Ulnar neuropathy at the wrist (UNW). Ulnar motor NCS (recording from FDI) demonstrates a 73% drop in ulnar CMAP amplitude at the wrist (A1) relative to the palmar stimulation site (A2), indicative of partial conduction block and focal demyelination. The distal ulnar CMAP amplitude is approximately 50% lower than the unaffected side (A), suggesting associated axon loss.

The authors study the FDI, ADQ, and FDP4. In UNW, the FDP 4 is normal. The FDI is affected in UNW types 1, 3, 4, and 5, whereas the ADQ is abnormal in types 1 and 3 but spared in types 4 and 5. The authors also examine APB and EIP to exclude a C8 radiculopathy or a lower trunk brachial plexopathy.

Finally, in patients in whom the ulnar motor CMAP amplitudes to the FDI or ADQ are reduced and the needle examination is suggestive of partial conduction block (decreased recruitment with little active denervation or reinnervation), the authors perform short-segment incremental stimulation studies (recording from FDI) across the wrist to confirm conduction block and localize the site of the lesion (Fig. 6) [62].

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