

Clinical Features and Electrodiagnosis of Ulnar Neuropathies

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KEYWORDS

• Ulnar neuropathy • Electrodiagnosis • EMG • Sensitivity • Specificity

KEY POINTS

- The most common locations for ulnar mononeuropathy are at the retroepicondylar (RTC) groove and the humeroulnar arcade.
- Precise localization of the ulnar nerve below and above the elbow with submaximal stimulations improves accuracy of the distance measurement.
- The factors that can lead to spuriously low nerve conduction velocity (NCV) across the elbow include a cold elbow and falsely low distance measurements.
- Multiple internally consistent abnormalities should be present to ensure accurate diagnosis of ulnar neuropathy at the elbow.
- In the setting of ulnar mononeuropathies, when routine electrodiagnostic studies fail to demonstrate focal slowing of NCV across the elbow, short-segment techniques should be done.
- The electromyographer should ascertain the specific point of abnormality (ie, the RTC groove, humeroulnar arcade, or other location) prior to surgical referrals.

INTRODUCTION

The ulnar nerve may be compressed at several sites. Ulnar neuropathy at the elbow (UNE) is the second most frequent upper extremity compression neuropathy. There are 4 different sites of potential compression in the region of the elbow. The nerve may also sustain focal injury in the wrist and hand and even less frequently in the axilla, upper arm, or forearm. Distinguishing between these different compression sites is not

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always straightforward. In most cases, the earliest electrodiagnostic findings are demyelinating. Early diagnosis and management can prevent secondary axonal damage and permanent disability.

RELEVANT ANATOMY

Anatomic details are important in understanding focal ulnar mononeuropathies.^{1,2} The ulnar nerve branches from the medial cord of the brachial plexus and courses in the forearm just medial to the brachial artery. It passes between the medial intermuscular septum (MIS) and the medial head of the triceps prior to reaching the medial epicondyle. The existence of the arcade of Struthers between the MIS and medial head of the triceps is debatable.³ The nerve then passes just dorsal to the ME, and into the ulnar groove, ventral to the olecranon process (OP). It subsequently passes beneath the humeroulnar aponeurotic arcade (HUA), a dense aponeurosis between the tendinous attachments of the flexor carpi ulnaris (FCU) muscle typically 1.0 cm to 2.5 cm distal to the ME.² The nerve then runs through the belly of the FCU and exits from the muscle through the deep flexor pronator aponeurosis.

In elbow extension, the medial epicondyle and OP are juxtaposed, with the HUA slack and the nerve lying loosely in the groove. With elbow flexion, the OP moves forward and away from the ME. The humeral head of the FCU, attached to the ME, and the ulnar head, attached to the OP, are pulled apart, progressively tightening the HUA across the nerve, resulting in pressure increases up to 19 mm Hg in the ulnar groove.⁴ In addition, with elbow flexion, the ulnar collateral ligament bulges into the floor of the groove and the medial head of the triceps may be pulled into the groove from behind.¹ In extension, the ulnar groove is smooth, round, and capacious, but in flexion the nerve finds itself in inhospitable surroundings, in a flattened, tortuous, and narrow canal with the HUA pulled tightly across it. In full flexion, the nerve partially or completely subluxes out of its groove in many normal individuals.⁵

The only motor branches in the forearm are those to the FCU and flexor digitorum profundus (FDP). The palmar ulnar cutaneous branch (PUC) separates from the main trunk in the mid to distal forearm, and enters the hand superficial to the Guyon canal, supplying sensation to the skin of the hypothenar region. The dorsal ulnar cutaneous (DUC) branch leaves the main trunk 5 cm to 10 cm proximal to the wrist, arcs around the ulna, and innervates the dorsal skin of the medial hand and fingers. The ulnar nerve then enters the hand through the Guyon canal.

The transverse carpal ligament, which arches over and forms the roof of the carpal tunnel, dips downward to form the floor of the Guyon canal. The roof, lateral, and medial boundaries of the canal are formed by the volar carpal ligament and the thin palmaris brevis muscle, hook of the hamate, and the pisiform bone, respectively. Just beyond the transverse carpal ligament, the pisohamate ligament runs from the pisiform bone to the hook of the hamate and forms the distal part of the floor of the canal. The nerve exits the Guyon canal by passing beneath the pisohamate ligament.

In the hand, the nerve bifurcates into the superficial terminal division and the deep palmar division. The superficial terminal portion supplies sensation to the small finger and ulnar half of the ring finger. The deep palmar branch subserves no cutaneous sensation but innervates all of the hypothenar muscles, the third and fourth lumbricals, all of the palmar and dorsal interossei, the adductor pollicis, the deep head of the flexor pollicis brevis, and the first dorsal interosseous (FDI). There are frequent anatomic variations.

Anatomic factors account for much of the susceptibility of the ulnar nerve to injury at the elbow. The lack of protective covering over the nerve in its course through the ulnar

groove accounts for its susceptibility to external pressure. Repetitive elbow flexion and extension may predispose to UNE because of the dynamic changes in the nerve's passageway with motion. With elbow joint derangement due to trauma or arthritic changes, the nerve's vulnerability increases even further. Valgus deformities increase the stretch on the nerve with elbow flexion, and osteophytic overgrowth further narrows an often already narrow passageway. Most ulnar neuropathies occur at the level of the RTC groove. The nerve may also be entrapped at the HUA or at the point of exit from the FCU.⁶⁻¹⁰

The internal architecture of the ulnar nerve, particularly the fascicular arrangement, has an important impact on the clinical and electrodiagnostic findings.¹¹ The fibers destined for the FCU, the PUC, and DUC lie in individual fascicles at the elbow and in a deep dorsolateral position, rendering them less susceptible to damage with UNE. This can create difficulty differentiating UNE from ulnar neuropathy at the wrist (UNW).

CLINICAL FEATURES

In the majority of patients with UNE, the initial symptoms are typically intermittent numbness and tingling in the ulnar nerve distribution, often associated with elbow flexion, particularly at night. These intermittent symptoms may occur over months or years, although in patients with more severe entrapment, permanent symptoms may develop more rapidly. The amount of pain and paresthesia varies, and for some patients the sensory loss is not bothersome. In contrast to carpal tunnel syndrome, where pain is usually a prominent feature, UNE tends to cause numbness and paresthesias and pain is less prominent, often absent. Vanderpool and colleagues¹ state that subjective motor loss may not be noted for months or years, depending on the degree of compression. In contrast, pain and dysesthesias are more frequent components with acute injury to the elbow, pain and dysesthesias are more frequent components. Elbow pain is rare except in acute focal injury.

The sensory abnormalities in ulnar neuropathies do not always conform to the expected distribution due to anatomic variations. Splitting sensory symptoms of the ring finger is highly specific for ulnar neuropathy. C8 radiculopathy and brachial plexopathy are more likely to affect the entire ring finger or spare it completely. In UNE, parasthesias typically involve the digits to a greater extent than the dorsal and palmar aspects of the medial hand, due to relative sparing of the DUC and PUC.¹¹ The cutaneous field of the ulnar nerve does not extend more than a few centimeters proximal to the wrist crease. Sensory abnormalities in the forearm should raise the suspicion for plexus or nerve root lesions.

The motor disability from ulnar nerve palsy is related to 4 components¹: strength of pinch between the thumb and adjacent digits,² coordination of thumb and digits in tasks requiring precision,³ synchrony of digital flexion during grasp, and⁴ strength of power grasp. Wrist flexion weakness is rarely significant due to normal function of the flexor carpi radialis.

Froment sign is due to weakness of the adductor pollicis and FDI, with compensation provided by the flexor pollicis longus. The lumbricals flex the metacarpophalangeal joints and extend the interphalangeal joints. In ulnar lesions, unopposed extensor tone at the fourth and fifth metacarpophalangeal joints and unopposed flexor tone at the interphalangeal joints produces the ulnar griffe or claw deformity. Clawing varies, depending on the amount of muscle weakness. A distal ulnar lesion that spares the FDP induces more clawing than more proximal lesions due to greater flexion of the interphalangeal joints of the fourth and fifth digits. The palmaris brevis (PB) sign

is a wrinkling of the skin over the hypothenar eminence during 5th digit abduction. This is due to contraction of the PB which is spared with UNW. The elbow flexion test is analogous to the carpal compression test and the Phalen test seeking to elicit ulnar paresthesias on forcefully flexing the elbow and applying pressure over the ulnar groove. Tinel sign is sometimes useful. But some patients have generally mechano-sensitive nerves, and only a disproportionately active Tinel sign over the suspect ulnar nerve has any significance. Both have a high incidence of false positives.

The forearm muscles, FCU and FDP, are frequently spared in UNE, so the lack of clinical or electromyographic abnormality in these muscles in no way excludes a lesion at the elbow.^{11,12} Abnormalities of these muscles are more common in lesions in the ulnar groove than compression at the HUA. Sparing seems related to either the redundant innervation via several branchlets from the main ulnar trunk or relative differences in fascicular vulnerability. Branches to the FCU do not arise from the ulnar nerve proximal to the elbow.²

One of the earliest signs of UNE is weakness of the third palmar interosseous, sometimes manifested by an abducted posture of the small finger (Wartenberg sign). The FDI is easily observed, and the bulk can be palpated and compared with the opposite side. It is particularly useful to test small hand muscles against the strength of an examiner's like muscles, after the methods described by Wolf.¹³ Demonstrating weakness in muscles outside the ulnar nerve distribution is vital for recognizing lower brachial plexopathies, C8 radiculopathies, and motor neuron diseases.

Ulnar nerve lesions in the wrist and hand can cause a confusing array of clinical findings, ranging from a pure sensory deficit to pure motor syndromes with weakness, which may or may not involve the hypothenar muscles. Of the different lesions of the ulnar nerve near the wrist, the most common and extensively reported is a compression of the deep palmar branch. In their now classic article, Shea and McClain¹⁴ classified ulnar compression syndromes of the wrist and hand into 3 types. In type I, the lesion is proximal to or within Guyon canal, involves both the superficial and deep branches, and causes a mixed motor and sensory deficit, with weakness involving all the ulnar hand muscles. In type II, the lesion is within Guyon canal or at the pisohamate hiatus, involves the deep branch, and causes a pure motor deficit with a variable pattern of weakness depending on the precise site of compression. A type III lesion is in Guyon canal or in the palmaris brevis, involves the superficial branch only and causes a purely sensory deficit. In the type I and III lesions, sensory loss should spare the dorsum of the hand, innervated by the DUC branch, and should also largely spare the hypothenar eminence because its innervation is via the palmar cutaneous branch, which arises proximal to the wrist. Other proposed UNW classification schemes add nominal value.

TERMINOLOGY

Careless use of terms, such as tardy ulnar palsy and cubital tunnel syndrome, has resulted in a nosologic quagmire. In 1878, Panas first described UNE developing long after an elbow injury, and the term, *tardy ulnar palsy*, was later applied to UNE after remote elbow trauma, generally after an old fracture or dislocation.¹⁵ The term soon degenerated into a nonspecific, generic term for any UNE, on the weak presumption that trauma must have occurred but patients simply could not recall it. The HUA as a site of ulnar compression was first described in 1916 by the British neurologist Dr F. Buzzard¹⁶ and his surgical colleague, Mr P. Sargent. The observation was lost until the 1950s when Osborne, Fiendel, and Stratford rediscovered it.

Osborne referred to the condition as spontaneous ulnar paresis.¹⁷ The HUA is sometimes referred to as Osborne band.

Feindel and Stratford¹⁰ introduced the term, *cubital tunnel syndrome*, to refer to patients with compression of the nerve by the HUA. They were attempting to define a subgroup of patients who suffered from a focal entrapment at the origin of the FCU and who could be spared a transposition procedure and managed with simple release of the aponeurotic arcade. The title of their article is illuminating, *The Role of the Cubital Tunnel in Tardy Ulnar Palsy*. As with tardy ulnar palsy, the term, cubital tunnel syndrome, soon degenerated into a useless, nonspecific, generic label for any UNE. Most physicians believed *cubital tunnel* refers to the nerve's subcutaneous passage through the ulnar groove and that cubital tunnel syndrome is synonymous with UNE, a serious misperception of the original intent. The authors restrict the use of the term to cases due to constriction by the HUA.

ETIOLOGY

There are 4 locations in the region of the elbow where the ulnar nerve may suffer compression: at the MIS, in the RTC groove, at the HUA, and at the point of exit from the FCU. Lesions in the RTC groove account for the vast majority of cases, but HUA compression is also common. In the 2 studies that have convincingly addressed the issue, there is remarkable concordance in the incidence of RTC abnormalities (69% and 62%) and HUA abnormalities (ie, cubital tunnel syndrome [23% and 28%]) and changes in both the RTC and HUA (8% and 10%).^{18,19} Other investigators disagree. Kline and colleagues²¹ reported 460 cases of ulnar entrapment at the elbow localized with intraoperative NAP inching. Conduction abnormalities always lay just proximal to and through the ulnar groove; there were only 8 cases of HUA entrapment.^{20,21} The exit compression syndrome is infrequent but turns up regularly if examiners are sensitive to its existence.^{6,9} The nerve can rarely be compressed by the MIS or arcade of Struthers proximal to the elbow.

Lesions occur in the RTC groove for several reasons, including acute or chronic external pressure, bony or scar impingement, anomalous muscles or bands, chronic stretch, particularly in the presence of a valgus deformity, and, rarely, mass lesions. In 30% to 50% of cases, no specific cause is discovered in spite of careful investigation, including surgical exposure.²²

Causes of UNW include extrinsic compressive neuropathy, fractures of the wrist, thrombosis of the ulnar artery secondary to trauma, and masses within Guyon canal, such as a ganglion.

RECURRENT SUBLUXATION OF THE ULNAR NERVE

Subluxation is often listed as a cause of UNE, but its role is far from clear. Childress⁵ examined 1000 normal, asymptomatic people and found an incidence of ulnar nerve subluxation of 16%. All these patients were asymptomatic, and the majority had the condition bilaterally. The incidence of subluxation in patients with UNE and how it compares with that in the general population is unknown. It is not clear by any means that subluxation predisposes to UNE and could help prevent UNE by allowing the nerve to escape from a narrow groove during flexion. If subluxation does predispose to UNE, it could be a result of the repetitive rubbing of the nerve across the epicondyle causing a RTC neuropathy (friction neuritis). Just as plausibly, subluxation could cause angulation of the nerve under the HUA during elbow flexion and result in HUA compression.

ELECTROPHYSIOLOGIC EVALUATION

Electrodiagnosis can play several roles in the evaluation of ulnar neuropathies. It can document the presence of a mononeuropathy; localize the lesion to any of several locations in the wrist, forearm, or elbow; and distinguish a mononeuropathy from a plexopathy, radiculopathy, polyneuropathy, or motor neuron disease. An abnormality can be confirmed prior to surgery and can be used to quantitate recovery following treatment. There are, however, limited data relating quantitative results of studies with prognosis after surgery. Electrodiagnosis of the ulnar nerve at the elbow is not nearly as straightforward as that of the median nerve at the wrist. The diagnostic yield is less and the interpretations of the data often more difficult. There are many possible techniques to use, and several studies have suggested useful approaches that are not commonly used in EMG laboratories.

NERVE CONDUCTION STUDIES

Ulnar Neuropathy at the Elbow

There have been several problem areas in the electrodiagnosis of UNE. These include controversy over the best elbow position, the ideal length of the across-elbow segment, and the value of absolute slowing in the above elbow (AE) to below elbow (BE) segment in contrast to a relative slowing of the AE-BE segment compared with the BE-wrist segment.

Technical errors are a major source of misdiagnosis. Care should be taken to insure supramaximal stimulation, especially at the BE site where the ulnar nerve lies deep in the FCU distal to the HUA. A common error is to stimulate too far posteriorly for the AE site, which can significantly alter the latency. The nerve curves acutely around the elbow and moves quickly toward the biceps, not the triceps. To minimize error, the AE and BE nerve locations can be mapped with submaximal stimulations before carrying out the conduction studies. It is frequently difficult to accurately measure around the curved elbow with a standard flat tape measure. A useful trick is to use a more flexible electrode wire lead to measure, marking the distance from the end of the wire placed at the AE site down to the point of BE stimulation, then laying the wire atop a tape measure to get the distance. The elbow should be in the same position used to obtain the reference values, and no change in position should be permitted between stimulation and measurement.

The difficulties with elbow position relate to the discrepancies between true nerve length and measured skin distance in different elbow positions. In extension, the nerve has redundancy, which progressively plays out with flexion. In extreme flexion, the nerve begins to stretch and slide distally and may partially or completely sublux. In extension, skin distance is falsely short compared with true nerve length, causing spurious and artifactual conduction slowing. In extreme flexion, if subluxation occurs, the skin distance is falsely long, causing spurious quickening.

Checkles and colleagues,²³ in a now classic article, first pointed out the remarkable difference in CV between an extended and a flexed position. Absolute AE-BE NCVs in the range of 35 m/s to 38 m/s and differences in the range of 20 m/s to 30 m/s between the AE-BE segment compared with the BE-wrist segment have been regularly reported in controls studied with the elbow extended.¹⁰ This position-related, artifactual slowing likely explains the high incidence of subclinical UNE reported by some investigators.²⁴ It is not clear that there is any difference in the relative diagnostic sensitivity of the different elbow positions in detecting abnormalities in patients with clinically defined UNE, as long as appropriate reference values are used. It is clear that consistency is paramount. A standard position must be used for stimulation as

well as for the measurement of distance, and this must be the same position used for obtaining the reference values. The American Association of Neuromuscular and Electrodiagnostic Medicine (AANEM) practice parameter on the electrodiagnosis of UNE concluded the most logical elbow position for ulnar conduction studies was moderate flexion, 70° to 90° from horizontal (**Box 1**).⁸

The ADM or the FDI for recording may be used; the latter is also useful for identifying lesions of the deep palmar branch. There is value in doing NCV while recording from both the FDI and ADM in order to detect lesions causing selective damage.^{25,26} Although the ADM is more commonly studied, the motor fibers to the FDI are more likely to be abnormal in a lesion at the elbow, and conduction studies are more likely to show conduction block (CB).¹⁰

After Maynard and Stolov's²⁷ influential article on experimental error, a minimum 10 cm across-elbow distance became standard. This article showed specifically how errors in measurement of latency and distance affect calculation of NCV, with errors in latency measurement accounting for 89% of the error, and error from distance measurement accounting for only 11%. A repeat of the same study using computer-automated equipment demonstrated an improvement in latency measurements errors.²⁸ This improvement in latency measurement, however, did not offset the significantly worsened error, resulting from distance measurements across a nonlinear surface.²⁹ The distance measurement error for the curved across-elbow segment can be 3 times higher than for a straight-line segment of comparable length. A decrease of greater than 10 m/s between the distal and proximal segments can occur from distance measurement error alone.

Although accepted that longer distance measurements are used to lessen experimental error and improve specificity for diagnosis, focal nerve injuries typically cause

Box 1

Synopsis of the recommendations of the AAEM practice parameter on ulnar neuropathy at the elbow

1. When using moderate-elbow flexion (70°–90° from horizontal), a 10-cm across-elbow distance, and surface stimulation and recording, the following abnormalities suggest a focal lesion involving the ulnar nerve at the elbow:
 - a. Absolute motor NCV from AE to BE of less than 50 m/s
 - b. An AE to BE segment greater than 10 m/s slower than the BE-wrist segment
 - c. A decrease in compound muscle action potential (CMAP) negative peak amplitude from BE to AE greater than 20%
 - d. A significant change in CMAP configuration at the AE site compared with the BE site
 - e. Multiple internally consistent abnormalities
2. If routine motor studies are inconclusive, the following procedures may be of benefit:
 - a. NCS recorded from the FDI muscle
 - b. An inching study
3. Needle examination should include the FDI, the most frequently abnormal muscle, and ulnar innervated forearm flexors. If ulnar innervated muscles are abnormal, the examination should be extended to include nonulnar C8/medial cord/lower trunk muscles to exclude brachial plexopathy, and the cervical paraspinals to exclude radiculopathy

Data from Campbell WW, Carroll DJ, Greenberg MK, et al. Literature review of the usefulness of nerve conduction studies and electromyography in the evaluation of patients with ulnar neuropathy at the elbow. Muscle Nerve 1999;22(Suppl 8):S175–205.

abnormalities of nerve conduction over a 1-cm segment.¹⁸ Studying long nerve segments may mask focal slowing by including lengths of normally conducting nerve. Thus, shorter distances are necessary to improve sensitivity. Two independently conducted studies, that equally weighted sensitivity and specificity, concluded that optimal distance to detect focal lesions is approximately 5 cm to 6 cm.^{30,31}

The technical and biologic factors that affect determination of ulnar forearm and across-elbow NCV are different. Technically, the distance measurement for the across-elbow segment is nonlinear and shorter than the forearm segment. Two important biologic variables are body mass index (BMI) and temperature. Each affects NCV determination, but the effects on the across-elbow and forearm segment are different. The across-elbow segment NCV directly correlates with BMI, but the forearm segment does not.^{32,33} As BMI increases, the distance measurement increases and dissociates from the actual nerve distance. Thus, demonstrating a difference in the NCV between the 2 segments is more difficult in those with high BMIs (possible false-negative study) and is easier in those with low BMIs (possible false-positive study).

Ambient temperatures also affect ulnar forearm and across-elbow NCV differently. Low skin temperature causes no appreciable change in forearm NCV but significantly lower across-elbow NCV.^{34,35} The deep location of the forearm segment presumably insulates this segment from surface temperature fluctuations, whereas the superficial location of the across-elbow segment makes it more susceptible to temperature effects. This discrepancy can be seen when there are no other indications from other nerve conduction studies of cool temperature effects (eg, prolonged peak latencies of sensory potentials). Failure to maintain adequate temperature of the across-elbow segment may, therefore, lead to false-positive studies and should be warned particularly when there are no other clinical or electrodiagnostic findings that support the diagnosis of UNE (cold elbow syndrome). The continued use of the forearm NCV as an internal control variable for diagnosis of UNE should be reconsidered.

All or most cases of UNE demonstrate demyelinating abnormalities. Focal demyelination at the elbow leads to an excessive dispersal of conduction velocities of the motor axons, which produces a low amplitude, long duration, fragmented CMAP on stimulation proximal to the elbow compared with stimulation distal to the elbow. A decline in total area under the CMAP curve correlates better with true CB, but temporal dispersion (TD) is just as suggestive of focal demyelination.^{36,37} A reduction in amplitude of more than 20% or a significant change in CMAP configuration between the BE and AE sites is suggestive of UNE. A reduction in amplitude of more than 25% was the best criterion for localization in one study.³⁸

Some patients with UNE have no or minimally detectable conduction velocity abnormalities, the so-called pure axon loss ulnar neuropathy.³⁹ Sensory and motor studies demonstrate decreased amplitudes and slowing of NCV consistent with axonal loss but no differences between across-elbow and forearm NCVs. In nearly all cases, however, if other muscles are used to measure NCV (ie, FCU or FDI) or short-segment studies are performed, focal demyelination can be disclosed.¹⁸

The distal sensory nerve action potential (NAP) is a sensitive indicator of ulnar nerve function. Most patients with UNE have a low amplitude or absent NAP, although it is a nonspecific, nonlocalizing, finding.^{7,24} A lesion at the elbow can sometimes be identified by sensory studies using needle electrodes to record possible focal slowing and NAP dispersion at the elbow, especially in patients with only sensory symptoms. Such NAP studies have significant pitfalls and limitations and should only be used if the examiner is fully aware of the technical details and the applicable literature.⁸

Motor conduction studies in patients have shown localizing abnormalities in symptomatic elbows with a sensitivity of 37% to 100%.^{38,40-45} Eisen²⁴ demonstrated 53%

sensitivity in severe cases and 27% in mild cases. In general, change in the absolute CV is a more sensitive indicator of abnormality than is abnormality of the relative CV. The results of various studies are reviewed in detail by Campbell and colleagues.⁸

An ancillary technique measures ulnar nerve conduction to the FCU.^{46,47} Benecke and Conrad⁴⁷ found the technique equally sensitive to motor conduction to the ADM.⁴⁸ Payan was able to localize the lesion to the elbow in another 10 of his 50 cases with this method.⁴⁰ The technique is limited by the nerve fibers to the FCU tending to be spared in UNE.

Ulnar Neuropathy at the Wrist

Assessment of conduction to the FDI muscle, in addition to the routine motor latencies to the ADM, is integral to the evaluation of distal ulnar neuropathies.⁴⁸ Olney and Wilbourn⁴⁹ studied conduction to the FDI and ADM in 373 nerves, determining absolute distal motor latency (DML) to the FDI as well as differences between the latency to FDI and the ADM on the same side and differences in the side-to-side FDI latencies.⁴⁹ With stimulation at the proximal wrist crease, 5.5 cm to 6.5 cm proximal to the ADM recording site, the upper limit of normal for DML was 3.4 ms to the ADM and 4.5 ms to the FDI. There was an increase of approximately 0.5 ms per decade in the DML to each muscle, but advancing age did not significantly alter the side-to-side latency difference or ipsilateral muscle-to-muscle difference. According to this study, the side-to-side difference in DML should not exceed 1.0 ms for the ADM or 1.3 ms for the FDI. The ipsilateral difference in DML to the ADM versus FDI should not exceed 2.0 ms. A CMAP amplitude less than 6 mV for the FDI and less than 5 mV for the ADM was considered abnormal.

A lesion of the deep palmar branch, beyond the branches to the hypothenar muscles, causes prolongation of the DML to the FDI in the face of a normal motor latency to the ADM and normal sensory studies. Even if the DML is not prolonged, the CMAP may demonstrate fragmentation, dispersion, or CB. Needle electrode examination (NEE) typically shows denervation in all the ulnar intrinsic hand muscles except those of the hypothenar eminence. A sequential assessment of the first through the fourth dorsal interossei can sometimes provide precise localization. When the lesion involves the volar sensory branch alone, only the distal sensory action potentials are abnormal.

As with carpal tunnel syndrome, some ulnar lesions at the wrist cause mild secondary slowing of motor conduction velocity in the forearm segment. Care must be taken in the final assessment to determine the site of most significant slowing, and the final electrophysiologic diagnosis should reflect the perspective of the entire picture.

SHORT-SEGMENT STUDIES

Precise localization of demyelinating ulnar neuropathies at the elbow or wrist region can often be achieved by inching or short-segment incremental studies (SSISs)—monitoring the CMAP while moving the stimulator in discrete, small steps.^{7,18–20,50,51} When there is definitive CB or TD, precise measurements between the stimuli are not necessary. Movement of the stimulator along the course of the nerve discloses the exact location of demyelinating injury with a sudden change in amplitude or configuration of the CMAP. There are at least 6 reported short-segment techniques for evaluation of the ulnar nerve, 5 for the elbow and 1 for the wrist region. Although these techniques have not been systematically compared with more routine techniques, it is possible that the use of short distances between stimulation points increases sensitivity for detection.

Irrespective of which technique is chosen, limiting technical error is of utmost importance. The elbow should be maintained in a fixed position throughout the study. Submaximal stimulations should be applied to the ulnar nerve to accurately determine its location prior to making any measurements. The nerve location is defined by where a submaximal stimulation produces the largest CMAP response. This is particularly important for detecting partial or complete subluxation of the nerve. When attempting to detect focal slowing without CB/TD, precise measurements are necessary. The authors recommend using calipers preset at 1 cm or 2 cm to determine stimulation points and minimize experimental error. During testing of each segment, maximal, but not excessively supramaximal, stimulations should be given to avoid inadvertent stimulus spread more distally.

Short-segment studies can be time consuming when meticulously performed. There are, however, multiple situations when SSISs can be useful. SSISs can differentiate lesions at the HUA from the RTC groove and potentially have an impact on determining which surgical technique is most suitable for an individual patient. The AANEM practice parameter recommended that multiple internally consistent abnormalities be present to make a diagnosis of UNE.⁸ In patients with an isolated abnormality of decreased across-elbow NCV on routine NCS, SSISs can provide another abnormality to ensure accurate diagnosis. In cases of pure axon loss UNE, SSISs can demonstrate focal slowing, confirming localization in the elbow region. A completely normal SSISs in this situation suggests the possibility of a lesion elsewhere. In patients with persistent symptoms after ulnar nerve transposition, a modified short-segment study can be performed.⁵²

The use of SSISs in mildly symptomatic patients to detect subtle abnormalities otherwise not seen on routine conduction studies is debatable. The results will not likely change conservative management. The mere diagnosis, however, may prevent ordering other unnecessary diagnostic tests or incorrectly attributing symptomatology to a different cause (eg, a cervical radiculopathy).

The CMAP can be recorded from any muscle with SSISs. FDI might be expected to yield the highest results followed by ADM and then, lastly, FCU. One study, however, demonstrated that recording over FCU was more sensitive than ADM for detecting focal slowing.⁵³

ELECTROMYOGRAPHY

Although NEE is not as sensitive as nerve conduction studies for detecting UNE, denervation of ulnar-innervated hand muscles is commonly seen.^{11,26,39} The FDI is the most frequently involved muscle, followed by the ADM, FDP, and FCU, respectively.¹¹ NEE can localize a lesion to the elbow only if ulnar-innervated forearm muscles are also involved, but they are spared in many patients (*vida supra*). Pickett and Coleman³⁸ found NEE abnormalities in two-thirds of their UNE patients, but the abnormalities localized the lesion to the elbow in only 1 of 5. Kimura⁴¹ found NEE abnormalities were most frequent in patients with absent SNAPs; Eisen²⁴ found the incidence of NEE abnormalities correlated with the severity of motor conduction slowing. NEE is necessary in UNE diagnosis to exclude abnormalities in nonulnar innervated muscles.

AN APPROACH TO THE PATIENT

There are several regularly recurrent problem scenarios in dealing with suspected UNE:

1. Isolated slowing of NCV in the across-elbow segment of the ulnar nerve: When there are no other corroborating electrodiagnostic abnormalities, and the finding

does not correlate well with the clinical assessment, it is possibly artifactual, although some investigators have argued this represents subclinical ulnar neuropathy. The authors suspect that in many instances, this finding represents a technical error or the effects of cold temperatures.^{27,33,35} This situation requires utmost care. The dictum, “never underestimate the ability of the EMG report to bring the knife down on the patient,” applies.

2. Lesions of the ulnar nerve not at the elbow: An ulnar nerve lesion at the wrist can be identified by more significant slowing of nerve conduction across the wrist than across the elbow and by lack of denervation in the forearm. The NAP of the dorsal cutaneous branch is normal, with lesions at the wrist even if the NAP of the PUC or digital sensory branches is reduced, whereas all are usually abnormal with lesions at the elbow.^{54,55} Rarely, ulnar entrapment occurs proximal to the elbow or in the forearm segment. These localizations should always be considered when no definitive slowing of conduction is noted in the elbow segment.
3. The pure axon loss lesion at the elbow: In some patients with UNE, it is difficult to demonstrate focal slowing across the elbow. When the forearm flexors are involved, probable localization to the elbow is still possible by NEE, although it is impossible to exclude a more proximal lesion in the axilla or upper arm. Helpful ancillary techniques in this situation include recording from the FDI, which may demonstrate focal slowing or CB even though fibers to the ADM do not, and/or SSISs, which can often demonstrate focal slowing missed when studying longer segments. Abnormality of the DUC can at least place the lesion proximal to its takeoff, but the DUC can occasionally be spared in elbow lesions. Many cases of pure axon loss UNE are likely due to lesions in unusual locations.
4. Patients with purely sensory symptoms and normal routine motor studies. Such patients usually have a mild or early UNE, and an adequate evaluation may well be to exclude other pathology, such as brachial plexopathy or cervical radiculopathy, and manage patients conservatively. To localize the lesion more confidently, FDI recording or SSISs may be useful. NAP recording, either surface or near nerve, across the elbow is technically difficult and the results should be interpreted cautiously.
5. Patients with forearm sparing: When conduction studies place the lesion at the elbow, sparing of the forearm muscles should be no deterrent to localization.
6. Wallerian degeneration with confusing distal abnormalities: When severe UNE causes major axon loss distal to the lesion, there may be secondary slowing of the entire distal ulnar nerve and prolongation of the DML, usually with an absent sensory potential and abundant denervation. In lesions of this severity, NEE abnormalities are usually present in the forearm muscles and FDI recording or SSISs often place the lesion at the elbow, even if routine studies are equivocal. An occasional error is to diagnose a second lesion at the wrist. If the CMAPs measured at the ADM or FDI are so small as to hinder accurate NCV determinations, recording over the FCU can be performed (the only situation in which the authors use this particular methodology).
7. Failed ulnar nerve surgery. Unfortunately, patients present with persistent or recurrent symptoms after an unsuccessful operation on the ulnar nerve. Sometimes there are no preoperative studies for comparison, in which case the electromyographer is reduced to guesswork. The first order of business should be to establish with certainty that no other process, such as plexopathy or radiculopathy, was responsible for the symptoms initially. One patient with persistent symptoms following two decompression surgeries for UNE was eventually found to have Ewing sarcoma in the axilla. Then the course of the nerve should be mapped to

determine whether or not transposition was done. This procedure alone may sometimes establish, by showing abrupt changes in nerve course, that kinking has occurred due to inadequate distal (more rarely proximal) release. After mapping the course of the nerve, an SSISs study can establish whether there is persistent focal compression or fibrosis, potentially amenable to reoperation, or whether there has been devascularization of a long nerve segment, an essentially end-stage condition.

SURGERY

The electrophysiologic contribution to the decision whether or not to operate is to document quantitatively the clinical state. For patients with mild sensory symptoms who are not believed surgical candidates, the studies should confirm only minimal abnormalities of sensory conduction.⁵⁶ For patients with mild to moderate motor and sensory symptoms who are considered for operation, studies are useful to demonstrate the degree of abnormality, to confirm the localization of injury, and to serve as a baseline for postoperative evaluation. Marked prolongation of conduction across the elbow suggests a poorer prognosis. In addition, deterioration can be determined objectively in those cases being followed.^{42,54} For patients with severe muscle atrophy who are believed inappropriate candidates for surgery, EMG can document the irreversible loss of muscle.

After successful ulnar nerve surgery there usually is electrophysiologic improvement.⁵⁷ The NCV in the elbow segment improves, but remyelination with short, thin internodes may prevent a return to normality despite a good clinical outcome. The motor NCV can improve, however, even with a poor result.⁵⁸ Subsequently the amplitude of the NAP recovers, accompanied by clinical improvement.⁵⁷

OTHER DIAGNOSTIC METHODS

High-definition ultrasound and MRI are becoming increasingly more useful adjuncts in the diagnosis of UNE. In a small study of 4 patients with symptoms of UNE and normal electrodiagnosis, ultrasound demonstrated enlargement near the elbow as defined by cross-sectional area (CSA).⁵⁹ The extent of the electrodiagnostic testing was not provided but nonetheless showed how promising this technique could be. There are several advantages over electrodiagnosis, to include patient tolerability, real time observation of structure, and time requirements. Ultrasound can distinguish focal enlargements of the nerve as typically seen in compression mononeuropathies from abnormal masses. Ultrasound does not provide any functional information regarding nerve conduction.

Parameters of ultrasound evaluation include nerve echogenicity, diameter, and CSA. Normally the ulnar nerve is echogenic with parallel internal linear echoes.⁶⁰ At the level of the ME, the nerve is more hypoechoic. Due to the quantitative nature, diameter and CSA are usually used for diagnosis. One complex study assessed multiple methods for quantitating nerve echogenicity and concluded that this parameter could distinguish UNE subjects from a healthy control group.⁶¹

Diameter is measured on longitudinal imaging and CSA on transverse imaging. Much of the ongoing research attempts to define reference values for each. One difficulty is determining the optimal control for comparison. An internal control uses comparison to the homologous region on the contralateral limb or to a predefined segment of the ulnar nerve away from the elbow. Alternatively, values derived from a normal control population can be used. With elbow flexion at 90°, the ulnar nerve changes shape and the CSA decreases. A critical review suggested that the maximum

diameter and CSA of the ulnar nerve at the elbow are 2.4 mm and 8 mm² to 10 mm², respectively. The sensitivity for detecting enlargement of the ulnar nerve using these parameters was approximately 80%.⁶⁰ This is approximately the same sensitivity as for routine conduction studies.

There are also few data about false-positive results using ultrasound. For example, a professional bowler found to have an enlarged nerve on MRI performed for medial elbow pain had medial epicondylitis clinically, with no evidence of UNE. Electrodiagnostic studies, including SSISs and FDI recording, were completely normal. The AANEM practice parameter emphasized the importance of multiple, internally consistent abnormalities in the electrodiagnosis of UNE. The same should be said about the clinical, ultrasound, and MRI aspects.

The findings on ultrasound have been correlated to the abnormalities seen in electrodiagnosis. Volpe and colleagues⁶² demonstrated that the maximum CSA was 14.6 mm² ± 5.0 mm² in UNE subjects versus 7.1 mm² ± 2.1 mm² in controls. The upper limit of normal based on mean plus 2 SDs in this study is 11.3 mm², significantly higher than in Beekman and colleagues'⁶⁰ critical review. The severity of the electrodiagnostic findings were predefined as mild, moderate, or severe. The severe UNE cases had a mean CSA of 18.3 mm ± 5.1 mm compared with 11.1 mm ± 3.4 mm in the milder group. The investigators concluded that ultrasound can have a role in severity stratification in addition to diagnosis.

Won and colleagues⁶³ used ultrasound to demonstrate ulnar nerve subluxation in 9 patients with clinical UNE but a paucity of electrophysiological evidence. They proposed that subluxation resulted in spuriously high distance measurements between stimulation points of the ulnar nerve above and below the elbow. The length of the ulnar nerve segment was remeasured under ultrasound guidance. The newly attained smaller distances resulted in a mean decrease of the ulnar NCV across the elbow by 7.9 m/s from the initial studies; subjects now had abnormalities that met criteria for UNE. Spuriously fast conduction velocities due to subluxation have been recognized and commented in previous studies.⁸ Mapping the nerve by using submaximal stimuli can also readily detect subluxation and avoid this error.

MRI is also useful in the evaluation of UNE. MRI is reliable in detecting structural abnormalities around the nerve as well as intrinsic abnormalities within the nerve. Vucic and colleagues⁶⁴ demonstrated a higher specificity of MRI in detecting UNE than conventional electrodiagnostic studies. The most frequent MRI changes included high signal intensity within the nerve, nerve enlargement, a combination of both, or nerve compression. MRI had a 90% sensitivity, whereas electrodiagnosis had a 65% sensitivity. The investigators used a distance of 13 cm between AE and BE stimulation points for motor nerve conduction studies, limiting the diagnostic sensitivity for focal demyelination. Furthermore, it was not disclosed whether SSISs or motor studies to the FDI were performed to potentially increase the diagnostic yield. Nonetheless, the MRI was particularly sensitive, without any false-positive errors. There were 15 control subjects with normal-appearing nerves. Additionally, there were 19 subjects with abnormal electrodiagnostic studies that did not definitively localize to the elbow region. In 16 of these subjects, MRI detected the abnormality at the elbow.

Bäumer and colleagues⁶⁵ assessed the role of magnetic resonance neurography in UNE. They demonstrated an increased nerve T2 signal as measured by a T2-weighted contrast-to-noise ratio in subjects with UNE compared to controls, with a sensitivity and specificity of 83% and 85%, respectively. This is a more realistic specificity than the 100% claimed by Vucic and colleagues.⁶⁴ Furthermore, they were able to distinguish mild cases of UNE from severe ones via nerve caliber measurements.

Researchers are also evaluating the potential use of diffusion-weighted MRI in UNE diagnosis.⁶⁶ In all 3 of these referenced studies, the RTC groove was incorrectly called *cubital tunnel*.

There is a trend in research, with many studies assessing the utility of MRI or ultrasound in UNE, to attempt to demonstrate superiority of the newer modalities over conventional electrodiagnostic studies. Each technique has specific advantages over electrodiagnosis, and it could be speculated that either may ultimately replace electrodiagnosis as the primary tool in the evaluation of ulnar neuropathies. Further studies that develop well-defined, universal parameters of high specificity will be essential to avoid overdiagnoses and unnecessary treatment interventions.

INTRAOPERATIVE ELECTRONEUROGRAPHY

Intraoperative electroneurography (IE) can help elucidate the precise point of abnormality and guide the type of surgical procedure that is performed. For instance, it may demonstrate focal slowing at the level of the HUA, suggesting a simple release of this structure will suffice. Alternatively, IE may demonstrate an abnormality at the level of the RTC groove supporting the need for either anterior transposition or medical epicondylectomy. Intraoperative studies are not technically difficult or especially time consuming.^{67,68}

In performing IE, it is important to minimize dissection to minimize complications. The authors' technique involves exposing the nerve via a curvilinear incision passing anterior to the ME; then, releasing the HUA (required for both transposition and simple decompression); then, with a minimum of further dissection, performing direct epineural stimulation over successive 1-cm segments while recording the M wave from the ADM.⁶⁸ Latency changes in excess of 0.45 ms over a 1-cm distance are considered abnormal, assuming an otherwise normal nerve. In patients with focal accentuation of a generalized neuropathy, judgment is required to best identify the pathologic segments. If the maximal abnormalities center about the site of the HUA (now divided), the procedure is terminated as a simple decompression. If maximal abnormalities center about the ME, further mobilization is carried out and the nerve transposed. If no pathologic segments are identified, the incision is extended and electroneurography repeated. In one such instance, a novel site of entrapment was uncovered.⁶ Direct epineural recording of NAPs requires some isolation and mobilization of the nerve to place electrodes. Because the authors prefer to minimize dissection, they have not used this technique. In summary, an operation for UNE could be tailored to the specific pathology present and transposition done only when truly necessary. IE can help guide the choice of procedure. The recent surgical literature increasingly favors simple decompression over transposition as the initial procedure, making precise localization less important.⁶⁹ IE may still have a role to play in those patients who fail the initial procedure.

SUMMARY

UNE results from focal compression of the ulnar nerve, primarily at the RTC groove or the humeroulnar arcade. In nearly all cases, focal slowing of nerve conduction can be demonstrated in the ulnar nerve segment across the elbow. When focal slowing cannot be demonstrated, other localizations for ulnar nerve compression must be considered. Electrodiagnosis is currently the primary tool for diagnosis. False-positive and false-negative errors occur, however, and are highly dependent on operator technique. False-positive errors are perhaps more damaging, because they can lead to unnecessary surgery. Among the many important procedural steps,

the electromyographer needs to precisely measure the distance between stimulation points above and below the elbow. Additionally, the elbow region should be warmed in cases of isolated slowing of NCV across the elbow, particularly in patients with low BMI or with little subcutaneous tissue in the elbow region. In cases referred for surgical intervention, the electromyographer should ascertain the specific point of abnormality (ie, the RTC or HUA or other). Ideally, the type of surgery performed is dictated by this determination.

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