

ELECTRODIAGNOSTIC EVALUATION OF CARPAL TUNNEL SYNDROME

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ABSTRACT: Carpal tunnel syndrome (CTS) is the most common nerve entrapment. Electrodiagnostic (EDX) studies are a valid and reliable means of confirming the diagnosis. This monograph addresses the various EDX techniques used to evaluate the median nerve at the wrist. It also demonstrates the limitations of EDX studies with a focus on the sensitivity and specificity of EDX testing for CTS. The need to use reference values for populations such as diabetics and active workers, where normative values differ from conventional cutoffs used to confirm suspected CTS, is presented. The value of needle electromyography (EMG) is examined.

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Carpal tunnel syndrome (CTS) is a clinical syndrome of numbness, tingling, burning, and/or pain associated with localized compression of the median nerve at the wrist. It is the most commonly reported nerve compression syndrome, accounting for 0.2% of all U.S. ambulatory care visits in 2006¹ and over 500,000 carpal tunnel releases in 2006.² The impairment of the median nerve within the carpal tunnel is secondary to compression of the median nerve, resulting in mechanical compression and/or local ischemia. However, the symptoms associated with CTS are frequently reported in areas outside the distribution of the median nerve.

Clinical CTS can be confirmed using electrodiagnostic (EDX) techniques that document abnormalities of the median nerve fibers within the carpal tunnel. Numerous studies have reported that comparison of sensory nerve responses is more effective than the use of an absolute median nerve latency in documenting the median nerve abnormalities consistent with CTS.³ Sensory fibers

have a larger proportion of large myelinated fibers, which have a higher energy requirement, and thus are more susceptible to ischemic damage.⁴ Focal compression results in both ischemia and mechanical damage to the nerve fibers due to dysfunction of the myelin and disruption at the nodes of Ranvier.^{4,5} Together this results in slowed conduction velocity, which allows the EDX physician to confirm a focal abnormality of the median nerve within the carpal tunnel. The comparison of median sensory latency to the radial, ulnar, or median (segments outside the carpal tunnel) sensory latencies allows the greatest accuracy for confirming the clinical diagnosis.³ Use of a comparison latency, as opposed to an absolute latency, controls for confounding factors of age, temperature, disease state (i.e., diabetes), gender, and hand size.⁶

GENERAL APPROACH TO NERVE CONDUCTION STUDIES

Uncomplicated CTS. In all cases, median sensory nerve distal latencies should be measured and compared to either the ulnar or radial sensory latencies in the same hand. There are multiple techniques to accomplish this, most of which are described herein. Using two comparison techniques that agree (either normal or abnormal) lowers the risk of a false positive or false negative.⁷ Thus, two comparison techniques that clearly agree (either normal or abnormal) should be adequate to confirm the diagnosis. In cases where the testing is borderline, extra testing and/or the combined summary index (CSI), described in what follows, can help clarify the diagnosis.^{8,9} If the median sensory response is absent, the use of median motor latency in comparison to the ulnar motor latency can help localize the problem. When comparison nerve conduction studies (NCSs) are abnormal for CTS in one limb and that limb is the only symptomatic limb, the American Association of Neuromuscular and Electrodiagnostic Medicine (AANEM) guidelines do not recommend performing NCSs on the opposite hand.¹⁰ If the symptoms are bilateral, or more diffuse, then NCSs on the opposite side are useful. If initial comparison NCSs on the symptomatic side are normal, further NCSs are rarely needed. NCSs in

Abbreviations: AANEM, American Association of Neuromuscular and Electrodiagnostic Medicine; AAOS, American Academy of Orthopedic Surgeons; APB, abductor pollicis brevis; CMAP, compound muscle action potential; CSI, combined summary index; CTS, carpal tunnel syndrome; EDX, electrodiagnostic; EMG, electromyography; inching, median sensory short segment stimulation across the wrist; MGA, Martin–Gruber anastomosis; MMW, median mononeuropathy at the wrist; NAP, nerve action potential; NCS, nerve conduction studies; palmdiff, median–ulnar mixed nerve palm latency difference from palm to wrist; ringdiff, median–ulnar sensory latency difference to the ring finger; SNAP, sensory nerve action potential; thumbdiff, median–radial latency difference to the thumb

Key words: carpal tunnel syndrome; electromyography; median nerve; nerve entrapment; nerve injury

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Table 1. AANEM practice recommendations for CTS.¹⁰

1. **Standard:** Sensory conduction studies across the wrist of the median nerve, and if the results are abnormal, of one other sensory nerve in the symptomatic limb.
2. **Standard:** If the initial median sensory nerve conduction study across the wrist has a conduction distance greater than 8 cm and the results are normal, additional studies as follows:
 - A. Comparison of median sensory nerve conduction across the wrist over a short (7–8 cm) conduction distance; or:
 - B. Comparison of median sensory conduction across the wrist with radial or ulnar sensory conduction across the wrist in the same limb; or:
 - C. Comparison of median sensory or mixed nerve conduction through the carpal tunnel to sensory or mixed nerve conduction studies of proximal (forearm) or distal (digit) segments of the median nerve in the same limb.
3. **Guideline:** Motor conduction studies of the median nerve recording from the thenar muscle and of one other nerve in the symptomatic limb to include measurement of distal latency.
4. **Option:** Electromyography of a sample of muscles innervated by the C5 to T1 spinal roots, including a thenar muscle innervated by the median nerve of the symptomatic limb.
5. **Option:** Comparison of the median motor nerve distal latency (second lumbrical) to the ulnar motor nerve distal latency (interossei).

the asymptomatic or minimally symptomatic limb are based on clinical utility, but they are not required to diagnose CTS. If a systemic problem like polyneuropathy is suspected, more widespread testing will likely help. Practice parameters for an EDX examination in CTS have been published by the AANEM¹⁰ (Table 1). These also have been incorporated by the American Academy of Orthopedic Surgeons (AAOS) evidence-based guidelines.¹¹

MEDIAN SENSORY NERVE CONDUCTION STUDIES

Most laboratories use antidromic sensory NCSs, because they have the advantage of producing larger amplitude sensory nerve action potentials (SNAPs) compared with orthodromic stimulation.^{12,13} In addition, peak sensory latencies are preferred over onset latencies in most laboratories, because the peaks are easier to identify in many waveforms, especially when the SNAP amplitude is small and the baseline is variable. Peak latencies have a higher inter- and intrarater reliability compared with the onset latencies.¹⁴ Commonly, these latencies are measured at 14 cm from the digits (Fig. 1).

Comparing the median sensory distal latency to the ulnar sensory distal latency can improve sensitivity and specificity and helps to control for other confounding variables such as temperature, age, height, and other patient-specific variability.^{6,10} SNAP amplitude has not been reported as reliable in the diagnosis of CTS. The sensory amplitude has considerable variability; therefore, the lower

limit of normal is relatively small. In the absence of a prolonged distal latency, a low SNAP amplitude has limited localizing value, unless a normal amplitude can be obtained when stimulation is distal to the carpal tunnel (suggesting conduction block in the carpal tunnel). A prolonged finger-to-wrist latency can also be the result of a focal lesion distal to the carpal tunnel, such as a digital neuropathy.¹⁵ Mild slowing of median forearm sensory nerve conduction velocities has been reported in 3–9% of CTS patients.^{16–18}

Measurement of Latency from Wrist to Palm and Palm to Wrist. The use of comparison studies and shorter segments has resulted in improved diagnostic sensitivity of NCSs for CTS.³ Because the segments of nerve outside the carpal tunnel conduct relatively normally, their inclusion in longer segments “dilutes” the sensitivity of routine methods, and a more practical method using antidromic stimulation has been used to measure the short segment across the palm (Fig. 2).^{19,20}

The use of the distal segments of the median nerve helps distinguish CTS from peripheral neuropathy. In CTS the maximal slowing is across the wrist, whereas in peripheral neuropathy the distal segment is more abnormal. This technique also

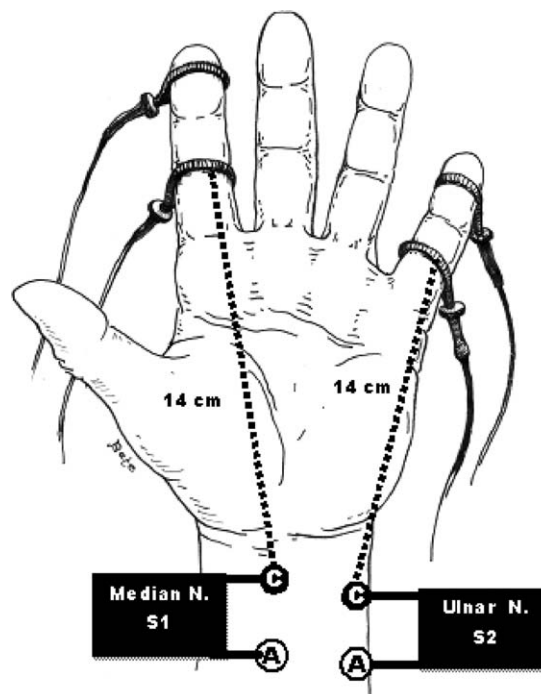
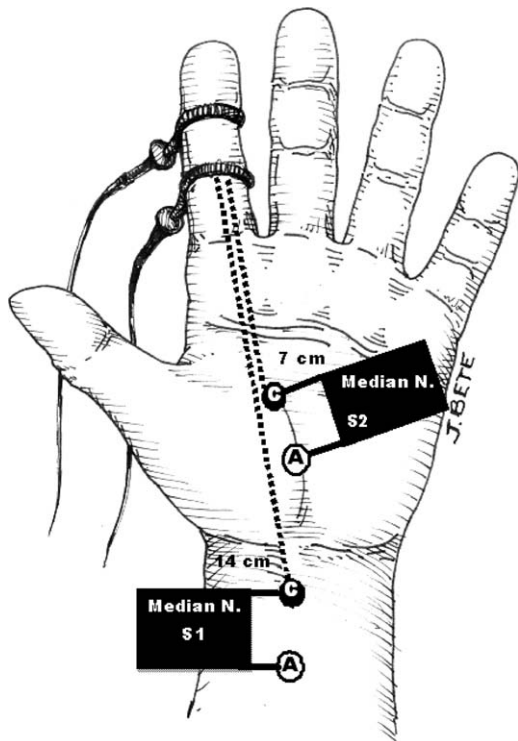


FIGURE 1. Sensory antidromic recording of the median nerve to digit 2 (S1) and the ulnar nerve to digit 5 (S2). The stimulation is at the wrist 14 cm proximal to the recording electrode (G1) usually placed on the index finger or middle finger. The reference electrode is a ring electrode on the distal finger. The ulnar stimulation is also 14 cm at the wrist with recording from the little finger and similar electrode placement as the index finger.



Antidromic Median Sensory Study

FIGURE 2. Antidromic stimulation in the palm with recording at the index finger or middle finger. Stimulation is 7 or 8 cm from the wrist stimulation, usually between the second and third metacarpals.

allows the EDX physician to recognize conduction block, which may be seen in acute CTS.²¹ Determining the exact criteria for conduction block in sensory studies is difficult, but an increase in the palm SNAP amplitude of $\geq 50\%$ compared with wrist stimulation has been reported to suggest conduction block.^{17,18,22} This technique may clarify the meaning of small SNAP amplitudes at the wrist. If both stimulation sites have small amplitudes, there may be axonal loss. Neurapraxia is more likely if the amplitude of the distal response (midpalm to digit) is significantly larger than the proximal wrist SNAP amplitude.

Orthodromic Palmar Stimulation. The median and ulnar nerves are stimulated in the midpalm at a point 8 cm distal to the wrist recording electrodes.²³ This is referred to as palmdiff, or the median–ulnar mixed nerve palm latency difference from palm to wrist (Fig. 3).

Median palmar stimulation evokes a mixed nerve action potential (NAP) from motor fibers innervating the second lumbrical muscle and the sensory digital nerve afferents from the index and middle fingers. However, the NAP recorded at the wrist is primarily generated by sensory fibers.²³ A difference of ≥ 0.3 ms or ≥ 0.4 ms is considered abnormal in most laboratories (Table 2). One dis-

advantage of this technique is the greater shock artifact, occasionally making it more difficult to obtain a good response, especially from the ulnar nerve.

Median–Ulnar Sensory Latency Difference to the Ring Finger. Known as ringdiff, this is usually an antidromic technique to stimulate the median and ulnar nerve at the wrist and record 14 cm from the ring finger using ring electrodes. The ring finger has median and ulnar innervation, and thus comparison of these latencies can be an efficient method of establishing a relative slowing of the median nerve compared with the ulnar across the wrist (Fig. 4).

A difference between the median and ulnar nerve latencies of ≥ 0.4 ms or ≥ 0.5 ms is considered significant (Table 1). The median nerve fibers to the ring finger may be more subject to compression due to the position of ring finger fibers in the outer margin of the median nerve beneath the transverse carpal ligament. Because the SNAP amplitudes are small, the median response may be absent in more severe cases of CTS.

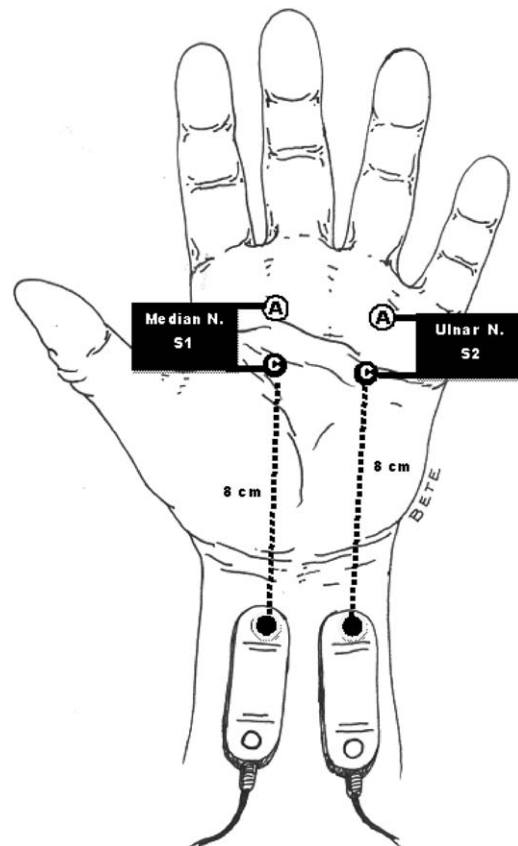


FIGURE 3. Median–ulnar mixed nerve palm latency difference from palm to wrist (also known as palmdiff). Stimulation is in the palm over the median (S1) or ulnar (S2) nerve, 8 cm distal to the active recording electrode. Recording is made with a bar electrode at the wrist over the course of the median and ulnar nerves, respectively.²³

Table 2. Review of existing studies and their suggested upper limits of normal..

Comparison	Technique	Investigator	Threshold defining abnormal
Sensory nerve responses			
Median-median	Short segment, 1 cm, antidromic (inching)	Kimura, 1979 ⁶⁸ Andary et al., 1992 ⁶⁹ Nathan et al., 1988 ²⁷	≥0.5 ms ≥0.4 ms
Median-ulnar	Digit 2 to digit 5, 14 cm, antidromic	Felsenthal and Spindler, 1979 ¹⁹	≥0.5 ms
Median-ulnar(normalized for active workers)	Digit 2 to digit 5, 14 cm, antidromic	Salerno et al., 1998 ¹⁴	≥0.8 ms
Median-ulnar (normalized for mild diabetics)	Digit 2 to digit 5, 14 cm, antidromic	Albers et al., 1996 ⁵²	≥1.0 ms
Median-ulnar (ringdiff)	Digit 4, 14 cm, antidromic	Johnson et al., 1981 ⁷⁰ Jackson and Clifford, 1989 ⁷¹ Robinson et al., 1998 ⁸ Uncini et al., 1989 (13 cm) ⁷² Andary et al., 1992 ⁶⁹	≥0.4 ms ≥0.5 ms
Median-ulnar (palmdiff)	Midpalmar, 8 cm, orthodromic	Stevens et al., 1987 ¹⁷ Robinson et al., 1998 ⁸ Andary et al., 1992 ⁶⁹ Jackson and Clifford, 1989 ⁷¹ Redmond and Rivner, 1988 ⁷³	≥0.2 ms ≥0.3 ms ≥0.4 ms 0.5 ms
Median-radial (thumbdiff)	Digit 1, 10 cm, antidromic or orthodromic	Johnson et al., 1987 ⁷⁴ Carroll et al., 1987 ²⁴ Robinson et al., 1998 ⁸ Jackson and Clifford, 1989 ⁷¹ Andary et al., 1992 ⁶⁹	≥0.5 ms ≥0.4 ms
Motor nerve responses			
Median-ulnar	Median-to-thenar compared with ulnar-to-hypothenar	Kimura, 1983 ²⁰	≥1.5 ms
Median-ulnar	Median-to-lumbricals compared with ulnar-to-intrinsics	Preston and Andary, 1992 ³²	>0.4 ms
Median-ulnar	Median-to-thenar compared with ulnar-to-thenar	Sander et al., 1999 ³⁰	>0.8 ms

Median-Radial Latency Difference to the Thumb. Known as thumbdiff, these SNAPs are acquired similar to the ringdiff technique described previously with ring electrodes on the thumb (digit 1) but typically using only a 10-cm distance from stimulation to recording (Fig. 5).

Thumb position can alter this measurement, and the thumb should be extended. The cutoff for the median-radial latency difference using antidromic stimulation and a 10-cm distance for each nerve is ≥ 0.4 or ≥ 0.5 ms.^{24,25} The radial nerve is less prone to injury, and therefore comparing the medial to the radial sensory segments is useful in patients with concomitant ulnar nerve injury.

Median Sensory Short Segment Stimulation across the Wrist. Using this technique, called inching, the median nerve is stimulated in serial 1-cm increments across the carpal tunnel with antidromic recording from the index or middle fingers (Fig. 6).^{26,27}

For routine use, eight or even fewer stimulation sites are sufficient, and a segmental peak latency difference of ≥ 0.5 ms is considered abnormal.²⁷ A drop in amplitude can be used to document conduction block. Although inching is a reliable method, it has the disadvantages of being time-consuming and

uncomfortable, because multiple sites are stimulated. In addition, this technique can be difficult to record because of stimulation artifact. In those patients with thick skin or a thick transverse ligament between the stimulation point and median nerve, the strong stimulus intensity required to adequately stimulate the nerve makes the exact site of nerve depolarization uncertain.^{17,18} The short segments also accentuate any measurement error that can occur with marking the segments or change in position of the stimulator when placed upon the skin surface.

CSI. Robinson and colleagues have used the sum of the three comparison tests just described to improve the reliability as well as the sensitivity and specificity of NCS to diagnose CTS. CSI = thumbdiff + ringdiff + palmdiff. Abnormal is defined as ≥ 0.9 ms and has a sensitivity of 83% and specificity of 95%. If the cutoff was raised to 1.1 ms, the sensitivity dropped to 82%, but specificity was found to improve up to 100% in one data set.⁸ Subsequent studies have demonstrated that if the first comparison (regardless of which of the possible three comparisons is chosen) has a significant latency difference, then it is not necessary to perform all three comparisons, because, when the first comparison is

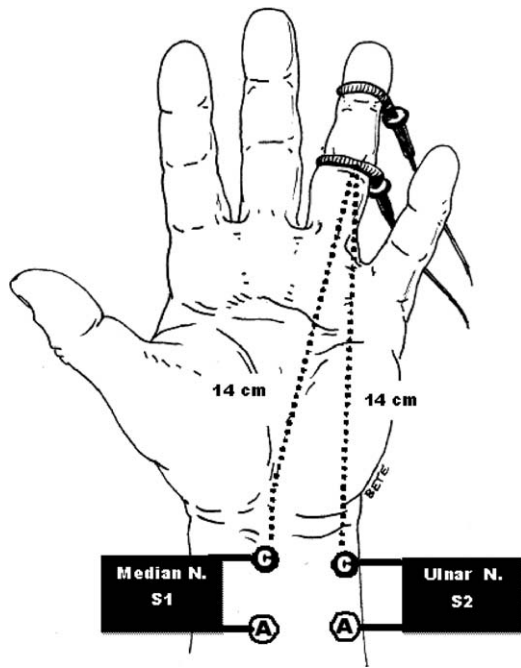


FIGURE 4. Median–ulnar sensory latency difference to the ring finger (also known as ringdiff). Stimulation is 14 cm proximal to the G1 ring electrode, over the median (S1) or ulnar (S2) nerves at the wrist. Recording is made with ring electrodes on the ring finger.²³

clearly abnormal, there is a 98% likelihood that the CSI will also be abnormal.⁹ If the first comparison does not meet the critical threshold, it is very useful to use the full CSI. In addition, a retrospective trial

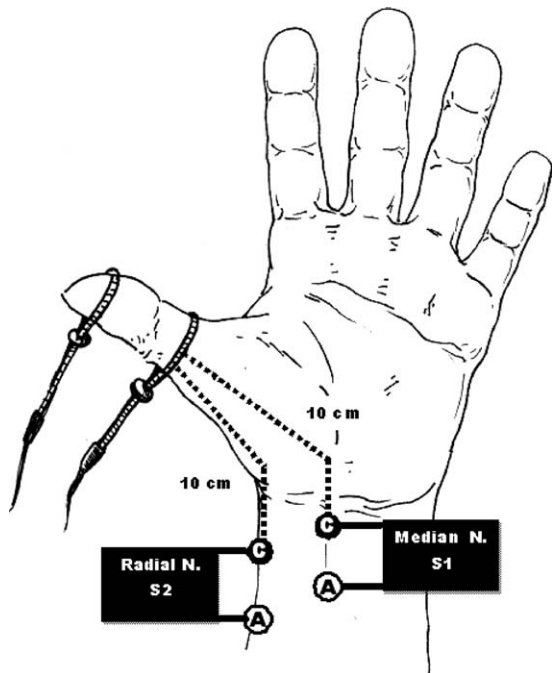
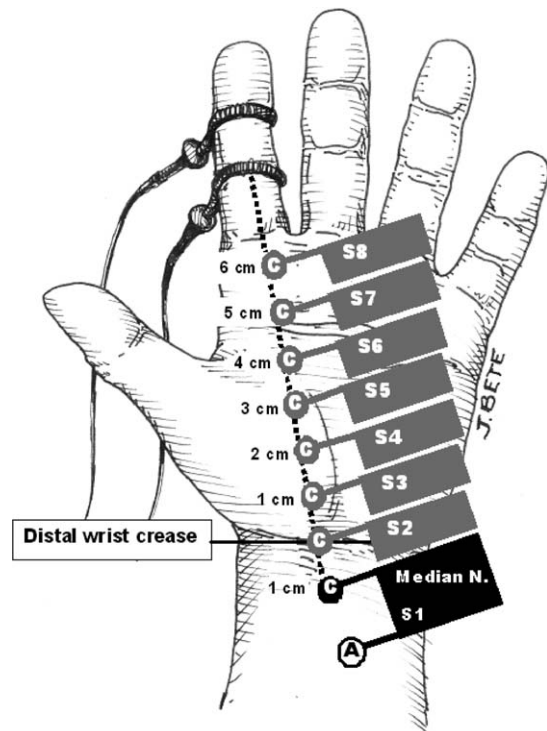


FIGURE 5. Median–radial sensory latency difference to the thumb (also known as thumbdiff). Stimulation is over the median (S1) and radial (S2) nerves at the wrist, 10 cm proximal to ring electrodes. Recording is made with ring electrodes on the thumb.



Median Sensory Short Segment Study

FIGURE 6. Median–sensory short segment stimulation (also known as inching). Stimulation starts 1 cm proximal to the distal wrist crease and continues segmentally in 1-cm increments to 6 cm distal to the wrist crease for a total of eight stimulations of the median nerve (S1–S8). Recording is made with ring electrodes on the index finger or middle finger.

demonstrated that patients with a CSI between 2.5 and 4.6 had somewhat better surgical outcomes.²⁸

The use of multiple comparisons for the evaluation of a focal entrapment may increase the risk of a type 1 error (false-positive result).²⁹ Alternatively, the finding of more than one abnormality when evaluating the median nerve across the wrist would lower the possibility of a type 1 error.⁷ Any attempt to increase the sensitivity of a testing paradigm will have the impact of lowering the specificity of the testing.

MEDIAN MOTOR NERVE CONDUCTION STUDIES

Although less sensitive than the other diagnostic methods just described, motor nerve stimulation still plays an important role in the documentation of motor fiber involvement and can be used to localize the lesion when the median sensory potentials are absent. The median motor distal latency is obtained by recording over the abductor pollicis brevis (APB) with stimulation at the wrist (Fig. 7).

This latency can be compared with the ulnar motor distal latency to the hypothenar eminence. There is some controversy regarding the appropriate cutoff. Most laboratories define a prolonged median motor latency if the relative latency is ≥ 1.2 – 1.8 ms.^{30,31} The use of shorter cutoff values likely will increase the rate of false positives.

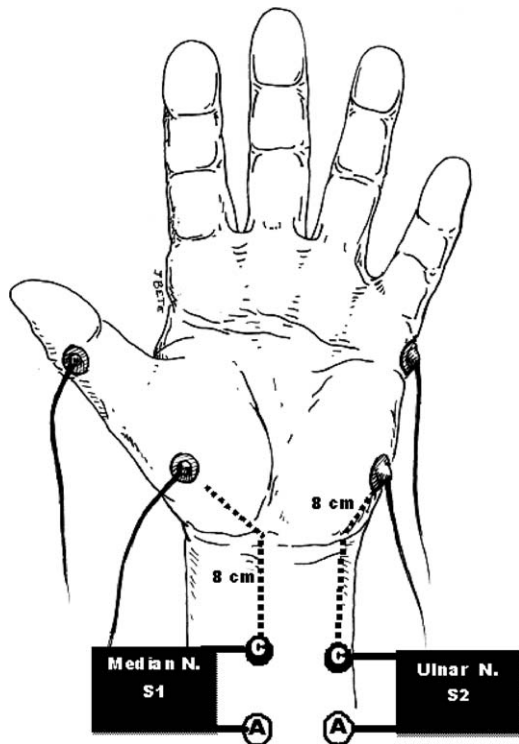


FIGURE 7. Electrode placement for evoking the median–thenar (S1) and ulnar–hypothenar (S2) compound muscle action potentials.

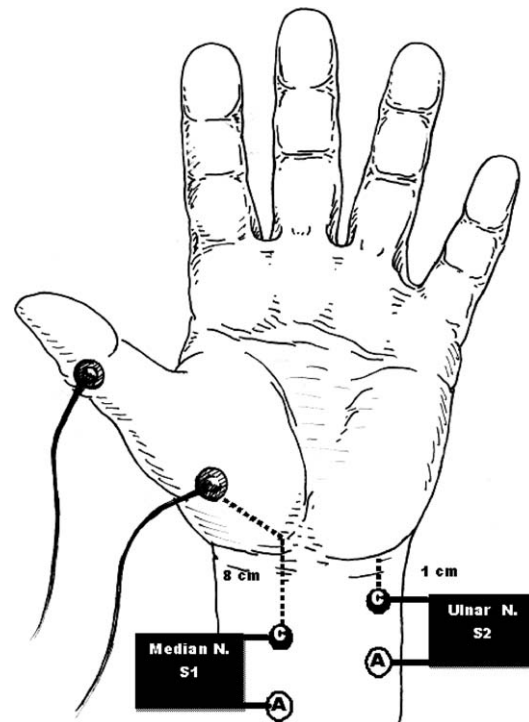


FIGURE 8. Electrode placement for evoking the median–thenar (S1) to ulnar–thenar (S2) latency difference (TTLD). The second stimulation is over the ulnar nerve, and the recording is over the thenar muscles.

Median–Thenar to Ulnar–Thenar Latency Difference. This is a simple motor comparison technique in which the first step is the standard median motor technique just described. The ulnar nerve can be stimulated 1 cm proximal to the distal wrist crease, and a compound muscle action potential (CMAP) can be recorded with volume conduction from other ulnar-innervated thenar area muscles (adductor pollicis and flexor pollicis brevis). This technique is useful in patients with Riche–Cannieu anastomosis, where the opponens and APB may also have some ulnar innervation (Fig. 8).

The ulnar nerve waveform usually has an initial positive deflection, and the latency should be taken from this point. Some investigators reported an abnormal value of >0.8 ms.³⁰ They cautioned against the use of >0.8 ms, as this would increase the false positives due to finding median–ulnar motor latency differences up to 1.4 ms in patients who may not have CTS. This may be related to the difficulty in determining the exact “take off” for the CMAP.³²

Lumbrical and Interossei Recording. Another relatively simple motor comparison technique records a median CMAP latency from the second lumbrical and an ulnar CMAP latency from the interossei (Fig. 9).^{33–35}

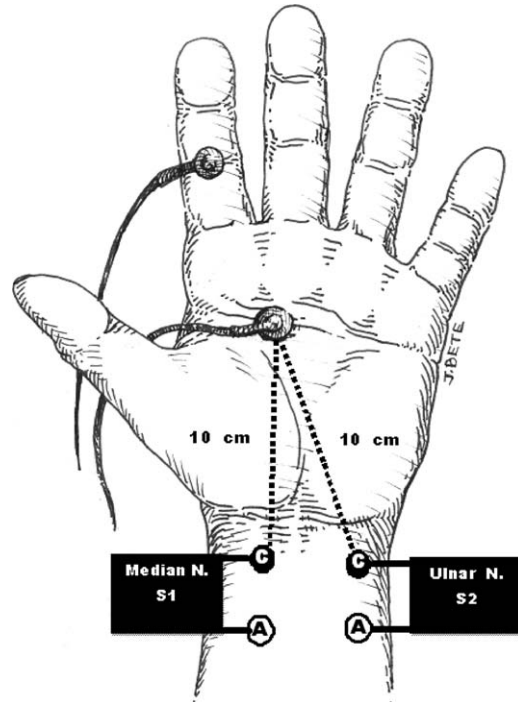


FIGURE 9. Median–ulnar motor latency difference to intrinsic muscles. Stimulation is at the wrist, with the same distance (usually 9–10 cm) proximal to G1 along the course of the median (S1) and ulnar (S2) nerves. Recording is at G1 between the second and third metacarpals in the distal third of metacarpals. Median stimulation records from the second lumbrical, while ulnar stimulation records primarily from the interossei. The G2 is over the proximal interphalangeal joint of the index finger.

A difference of >0.4 ms between the median and ulnar latencies has been reported as significant.³² In that study, the investigators cautioned the possibility of false positives using this number, as they have noted larger differences in those who were believed to be normal. In addition, because the median fibers to the second lumbrical are relatively spared compared with fibers to the thenar muscles, this technique may help with diagnosis of severe CTS when a lumbrical response may be obtained when other responses are absent (i.e., the median CMAP when recorded over the APB).^{18,36,37}

Midpalm Motor Stimulation. Information about the presence or absence of conduction block at the carpal tunnel can be obtained by recording the change in amplitude after stimulation of motor fibers in the palm and at the wrist.³⁸ This technique must be used with caution, because the distal stimulation in the palm can also stimulate ulnar-innervated fibers to the thenar muscles or result in direct muscle stimulation, thus demonstrating a false-positive conduction block. Care should be used to look for a positive dip when performing the palm stimulation, which would suggest stimulation of ulnar-innervated muscles in the thenar eminence.

Martin-Gruber Anastomosis. The Martin-Gruber anastomosis (MGA) is an anatomic anomaly observed in 7–34% of the population. Motor fibers usually cross from the anterior interosseous nerve (a branch of the median nerve) to the ulnar nerve and enter the hand with the ulnar nerve going through the Guyon canal and bypassing the carpal tunnel. Many muscles in the hand can be innervated by these fibers, but the first dorsal interosseous is the most commonly reported.³⁹ The MGA usually is clinically irrelevant; however, it can confuse the interpretation of the motor NCS, thus knowledge of the patterns is helpful. Because MGA fibers are only motor, sensory studies are not affected. Findings that are seen include: (1) larger amplitude of the median CMAP at the elbow than the CMAP at the wrist; (2) initial positive (downward) deflection of the CMAP at the elbow; and (3) spurious and falsely fast median nerve conduction velocity between elbow and wrist. The positive deflection from the elbow usually occurs when there is both CTS and MGA, because the MGA fibers that pass through the Guyon canal are faster than the slowed median fibers going through the carpal tunnel. This initial positive deflection with median elbow stimulation comes from the distant ulnar-innervated muscles in the thenar eminence and not from the APB. In severe CTS the velocity can be calculated as >100 m/s, and in extreme cases the latency from the elbow to the thenar

CMAP can be faster than the median latency from the wrist to thenar.³⁹

We have not found any published cases in which the motor latency is abnormal and the comparison sensitive sensory studies described are normal. There are cases of recurrent branch median motor neuropathy at the wrist, but this is not the same as CTS. If the distal motor latencies are abnormal when sensory nerve latencies are normal, extra care is required to exclude other causes, such as a C8–T1 radiculopathy, anterior horn cell disease, recurrent branch median neuropathy, or multifocal motor neuropathy, by reviewing the clinical findings and by performing additional NCSs and needle electrode examinations. Usually, isolated abnormalities of median motor nerve conduction with a normal median sensory NCS are not due to CTS.

A summary of studies and the suggested cutoff values for common sensory and motor comparison studies are presented in Table 2.

Conduction Velocity in the Forearm. Some patients with more severe CTS have slowing in the forearm proximal to the carpal tunnel. The etiology for this is unclear. The two major theories are: (1) retrograde degeneration of the median nerve proximal to the wrist; and (2) conduction block of the fastest conducting fibers at the carpal tunnel.^{18,39} The finding of slowed conduction velocity in the forearm does not rule out the diagnosis of CTS. The EDX physician must be aware that this finding is not rare in patients with a strong clinical impression of CTS.

Other Methods. Median nerve latencies across the wrist may increase in cases of borderline CTS after 2–5 minutes of wrist flexion^{40,41} or after 10–15 minutes of intensive hand activity. Unfortunately, the latencies also increase in normal subjects to a similar extent.⁴² The current AANEM guidelines do not recommend inclusion of these methods in the routine evaluation of patients suspected to have CTS.¹⁰

Comparison of Different Techniques. Although sensory comparison techniques have been shown to be more sensitive and specific than absolute sensory distal latencies, no one comparison technique has been clearly shown to be better than another.^{3,43}

Sensitivity and Specificity of NCSs for CTS. A consensus conference employed to refine the epidemiologic case definition of CTS concluded that there is no “gold standard” for the diagnosis of CTS, and therefore attempts at defining sensitivity and specificity are estimates at best. There are multiple problems in methodology including variable reference standards and spectrum bias in case-

control studies.^{43,44} Rempel and colleagues⁴⁴ concluded that the combination of EDX study findings and characteristic symptoms was the most accurate method to establish the diagnosis. They recognized that EDX studies could not be used as a gold standard for establishing the diagnosis. Approximately 10–15% of subjects with clinical CTS will have normal NCSs, which reflects a sensitivity of 85–90%. There are patients with intermittent symptoms that did not result in demyelination or axon loss as measured using NCSs. These symptoms are most likely related to acute ischemia with associated conduction block and are rapidly reversible (both symptoms and EDX findings). This has been demonstrated in experimental use of external compression of the median nerve in the carpal tunnel.⁴⁵ Many EDX physicians want to improve the sensitivity of their evaluation of patients suspected of having CTS, but this will influence the specificity of the test. What is frequently overlooked by EDX physicians is the finding that the specificity of EDX testing is only 82–85% in certain populations (i.e., there is a 15–18% chance of a false positive).^{3,46–48} This can be a major issue when evaluating workers with hand-intensive jobs who have vague hand symptoms but have a higher false-positive rate for a median mononeuropathy at the wrist (MMW).^{46,48}

Predictive Value of an Abnormal Median Nerve Latency across the Wrist in Asymptomatic Subjects. The meaning of an abnormal median NCS across the wrist in someone with no symptoms suggestive of CTS has been questioned. It is reasonable to ask if these subjects were preclinical cases of CTS. A well-designed prospective case–control study compared asymptomatic workers, with and without an MMW, to determine if they would proceed to develop symptoms consistent with CTS. Cases were defined as asymptomatic workers with prolongation of ≥ 0.5 ms of the median sensory distal latency compared with the ipsilateral ulnar sensory latency (14 cm, antidromic). Control subjects were age- and gender-matched asymptomatic workers from the same work site with a median-to-ulnar sensory peak difference of ≤ 0.2 ms and with both the median and ulnar absolute latencies within normal limits. After 2 years, asymptomatic workers with a prolonged median SNAP were no more likely to develop symptoms consistent with CTS than workers with a normal median sensory nerve latency. When these workers were subsequently followed for a total of 6 years, an increased risk of developing CTS symptoms was found for subjects who had an earlier abnormal median sensory nerve latency.^{49,50} These findings are very similar to those of another trial with an 11-year follow-up of industrial workers.⁵¹ In

each study, approximately 25% of asymptomatic workers with an abnormal median NCS across the wrist developed symptoms of CTS. However, 75% of workers with an abnormal median sensory latency at baseline did not develop symptoms of CTS within 7–11 years after the initial evaluation.

Defining CTS in Special Populations. A few studies using large reference populations have proposed different cutoff values for defining CTS in specific non-normal populations (e.g., diabetics and assembly-line workers). Among mild diabetics, without symptoms suggestive of CTS, a large multicenter trial demonstrated that the upper limit of normal (2 SD above the mean) was 1.0 ms when comparing median and ulnar latency (digits 2 and 5, 14 cm, antidromic).⁵² If a typical cutoff of 0.5 ms is used for diabetic patients, it would result in a large number of false positives, because patients with a relative latency of 0.5–1.0 ms are actually normal for this reference population. The diagnosis of CTS in diabetics is more difficult, and values obtained from normal populations can be misleading. These investigators suggested using comparison techniques with larger differences than those used in normal populations (e.g., ≥ 1.0 ms for diabetics). Antidromic midpalm sensory responses to the index finger may show selective slowing in the carpal tunnel when compared with the distal palm, as discussed earlier. Similarly, in a large cohort of >300 active assembly and clerical workers (asymptomatic workers—subjects with diabetes, rheumatoid arthritis, and thyroid conditions—were excluded, as were workers with highly hand-intensive jobs), the upper limit of normal for this reference population was 0.8 ms (comparison of the median and ulnar, digits 2 and 5, 14 cm, antidromic).¹⁴ This is a much larger cohort of normal subjects compared with most normative data collected for a specific laboratory, and caution should be used in defining an abnormality when evaluating an active worker with moderate slowing of the median nerve relative to the ulnar (i.e., only 0.5–0.7 ms). The diagnosis still is based on the clinical presentation. However, if the patient has vague hand symptoms that do not clearly define CTS and they have a borderline slowing of the median nerve (i.e., a relative latency of 0.5–0.7 ms), this should not be defined as CTS. Because half of the patients in the USA diagnosed with CTS have a surgical release of the carpal tunnel for management of the condition, EDX physicians should be pay particular attention what they write in the EDX report. If a patient has a confirmed diagnosis of a peripheral neuropathy, the use of a more conservative cutoff should be considered similar to the situation described earlier in diabetic patients. CTS

still can be diagnosed if the median motor and sensory distal latencies are disproportionately prolonged compared with the ulnar or radial latencies. Unfortunately, firm guidelines for interpretation of NCSs using exact numbers in the setting of peripheral neuropathy and other diseases are not available.

NCSs after Surgery. NCSs performed after surgery usually normalize but may not become completely normal. The exact timing of the improvement is not documented clearly, but it appears that some patients improve immediately^{53–55}; however, improvement in NCSs can take as long as 42 weeks for sensory latencies.⁵⁶ These slowed, but improved, distal latencies persist for at least 1 year and probably forever, even if symptoms are resolved.^{18,57} It has been suggested that the almost immediate relief of symptoms after surgery is the result of a reduction in spontaneous activity generated by the compressed nerve segment rather than by recovery from the conduction block.⁵⁸

NEEDLE ELECTRODE EXAMINATION

The decision whether to perform needle electromyography (EMG) in patients with CTS is not agreed upon universally and is listed as an option in the AANEM practice parameter.^{10,59} If the EDX consultant is not concerned about possible cervical radiculopathy, plexopathy, or other focal mononeuropathies and the possibility of axon loss will not alter management, then needle EMG is not necessary. If the median nerve conduction across the wrist is normal, there is a very low yield for an abnormal finding on needle examination of the APB.⁶⁰ Abnormal median NCSs do not predict abnormal needle EMG findings.⁵⁹ Nevertheless, there are many situations in which needle EMG may add useful information to the test. Needle EMG can help and is usually necessary to diagnose cervical radiculopathy, proximal median neuropathy, or polyneuropathy in addition to documenting axon loss in CTS. These diagnoses may be in addition to, or instead of, CTS. Patients with weakness of the APB, atrophy of the thenar eminence, or reduced amplitude of the median CMAP would benefit from needle examination to document axonal loss. In some situations, the only way to document axonal loss is with needle EMG of the median-innervated thenar muscles. The findings may indicate axon degeneration, including fibrillation potentials, large motor unit potentials, or excessive polyphasic motor unit potentials.

STANDARDS FOR DETERMINING THE DIAGNOSIS AND SEVERITY OF CTS

EDX findings are commonly used to grade the severity of CTS but lack scientific rigor. The corre-

lation between severity of NCSs and symptoms is not well established.^{61,62} However, the degree of abnormality can partially predict the outcome of surgery, with normal studies and very severe studies being worse after surgery.⁶³ The use of these classification scales have been discouraged for several reasons, primarily because treatment may be driven by the NCS when symptoms and other factors should be more important.⁶⁴

Despite these problems, the scale proposed in what follows has been used and works within the more complicated six-level scale proposed by Bland.^{18,63}

Mild CTS—Prolonged (relative or absolute) sensory latencies with normal motor studies. No evidence for axon loss.

Moderate CTS—Abnormal median sensory latencies as noted for mild CTS, and (relative or absolute) prolongation of median motor distal latency. No evidence of axon loss.

Severe CTS—Any of the aforementioned NCS abnormalities with evidence of axon loss as defined by either: (1) an absent or low-amplitude SNAP or mixed NAP; (2) a low-amplitude or absent thenar CMAP; or (3) a needle EMG with fibrillation potentials or motor unit potential changes (large-amplitude, long-duration motor unit potentials, or excessive polyphasics). The NCS amplitude abnormalities may be due to severe conduction block, which is not axonotmesis. This can be assessed by antidromic midpalm sensory conduction to the digits. If the SNAP at the wrist is absent or very low and the midpalm amplitude is much greater, this suggests conduction block and not axon loss. Midpalm motor conduction must be cautiously approached, because midpalm motor stimulation can easily stimulate the deep ulnar nerve fibers in the palm or Riche–Cannieu fibers that will indicate apparent motor neurapraxia. Sensory fibers are much more reliable for midpalm stimulation.

INTERPRETATION OF EDX FINDINGS

After the EDX data are collected, interpretation of the test findings is critical. There is variability among physicians about how to interpret and differentiate between normal, abnormal, or borderline studies. Age, limb temperature, skin thickness, coexisting medical conditions, and multiple other issues confound these decisions. Separation of the EDX abnormalities into the specific diagnosis of CTS distinct from other diagnoses—such as polyneuropathy, proximal median neuropathy, recurrent median neuropathy, or radiculopathy—is another aspect of the interpretation. The categorization of the median nerve injury (i.e., demyelinating or axonal) is important because some physicians will make a clinical management decision

based on the specific type of injury documented. The AAOS guidelines¹¹ list early surgery without conservative treatment as an option in patients with axon loss. Thus, treatment may be altered based on whether patients have demyelination or axonal loss.

Differentiating “carpal tunnel syndrome” from “median mononeuropathy at the wrist” (MMW) is a significant issue. Most definitions of CTS require symptoms that are related to median nerve dysfunction in the carpal tunnel. Thus, patients with symptoms of CTS and supportive EDX testing can be defined as confirmed CTS. Patients with abnormal NCSs but no symptoms probably should not be labeled as CTS but rather as MMW cases. This is especially important when evaluating patients with a high body mass index. There is a strong association between obesity and MMW (but not CTS). The reason for this association is not fully understood, but the relationship has been confirmed in several studies and can confound the diagnosis of CTS.^{65–67} If a patient has vague hand and/or wrist symptoms that do not seem related to the median nerve dysfunction at the wrist, physicians should be cautious in labeling these patients with CTS even if MMW is present. Calling this CTS may be misleading and lead to ineffective treatments. On occasion, the EDX test is normal in patients with classical CTS symptoms. In these cases, the test is likely a false negative and the patient still has CTS that has not been confirmed with EDX testing. This can be explained in the report.

Some EDX physicians formulate treatment recommendations as part of their EDX consultation, whereas others do not. Making treatment recommendations requires physicians to use their skills as a physician and utilize clinical information specific to that patient that will affect treatment decisions. These include but are not limited to disability, previous treatments, patient preferences, severity of symptoms, coexisting medical and psychological conditions, financial considerations, and quality of life. Noting that some or all of the symptoms are due not only to CTS but also to another problem, such as arthritis, tendonitis, or radiculopathy, can be part of the consultation. Treatment recommendations are usually beyond the direct EDX data collection and interpretation but are frequently included in the medical consultation. The emphasis of the report may differ depending upon the target audience. A hand surgeon is likely to want different information than a primary care doctor.

CONCLUSIONS

In the majority of cases, a careful history and physical examination are sufficient to make a clinical diagnosis of CTS and make initial treatment

decisions. However, many patients present with multiple problems and/or their presentation is atypical. An EDX examination can confirm the clinical impression of CTS, which is reassuring for both the patient and physician. Other patients may have coexisting or confounding diagnoses that EDX testing and consultation can help sort out. Performing, interpreting, and reporting high-quality and evidence-based EDX testing can potentially lead to improved outcomes in many patients.

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