Electrodiagnosis of Carpal Tunnel Syndrome

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KEYWORDS

- Carpal tunnel syndrome
- Electrodiagnosis
- Combined Sensory Index
- Nerve conduction study

KEY POINTS

- Carpal tunnel syndrome (CTS) will likely continue to be a common problem encountered by the electrodiagnostician, given recent evidence suggesting an increasing incidence of the condition.
- New modalities, such as MRI and ultrasound, are being applied to CTS and can potentially add important information about the anatomy and morphology of the carpal tunnel, median nerve, and surrounding tissue.
- Although CTS is a condition that is relatively easy to recognize, investigations to improve early CTS detection and treatment outcomes are warranted.

INTRODUCTION

Carpal tunnel syndrome (CTS) is the most common and the best studied of all focal neuropathies.\(^1,2\) CTS has provided an unmatched teaching and learning experience for generations of students, physicians, and scholars involved in patient care and electrodiagnostic testing (EDX). The EDX methodology for evaluating CTS has served as an example for the study of all focal neuropathies.\(^3,4\)

It is estimated that one in five patients presenting with upper limb pain, numbness, tingling, and weakness have the diagnosis of CTS. CTS also accounts for 90% of all known compression or entrapment neuropathies.\(^5,6\) A search on PubMed using the phrase “carpal tunnel syndrome” yields more than 7000 articles published in the English language literature alone in the new millennium, 2000 of which have been published since the beginning of this decade. This ongoing scholarly interest in CTS holds promise for further refinements to the diagnosis and treatment of the
disorder and, it is hoped, will lead to effective ways to prevent this common human ailment.

Recent epidemiologic studies suggest the number of people afflicted by CTS is substantial in US and European populations. The prevalence of self-reported CTS in the US adult population has been estimated to be as high as 5%. New demographic data indicate an increasing trend.

CTS was initially defined as a clinical disorder diagnosed by pattern recognition of patients presenting with similar symptoms and careful clinical examination. Rudimentary EDX for CTS began in the mid-1950s and continued to develop throughout subsequent decades. EDX has been regarded as an objective, reliable, and valid test for CTS. More recent advancements in technology have brought EDX to a level of performance and reliability unavailable to earlier clinicians. There have also been improvements in the speed in which the study can be conducted and comfort afforded to the patient.

This article discusses the historical aspects related to the understanding of CTS and its diagnosis, highlighting observations about this disease that have yet to be challenged. This is followed by a discussion regarding the use of EDX as a diagnostic tool for CTS, as well as the author’s approach to making the diagnosis of CTS. Finally, conclusions about future directions in the diagnosis, treatment and research of this disorder are presented.

HISTORICAL ASPECTS OF CTS

Descriptions of CTS by surgeons treating traumatic upper limb injuries date back to at least the mid-1800s. Brain’s 1947 landmark publication in the Lancet reported spontaneous CTS and summarized earlier published cases from 1909 to 1945. The term acroparesthesias, coined by Ormerod in 1833 to describe paresthesias in the extremities, was later used to refer to the nighttime burning and pins-and-needles sensation in the fingers of middle-aged women. Acroparesthesias in the median nerve distribution continue to be useful in evaluating CTS. Another feature noted in the early history of CTS was that of thenar eminence atrophy and hand weakness. However, an understanding that the sensory and motor symptoms were caused by a single median nerve lesion at the wrist was not appreciated until many years after the first clinical descriptions of the disease. Eventually the hypotheses that thenar neuritis, brachial plexus lesions, or an extra cervical rib could cause CTS were refuted, and focal median nerve compression at the carpal tunnel (CT) was identified as the sole cause for both the sensory and motor deficits.

The term carpal tunnel syndrome was said to appear in print for the first time in a paper published by Kremer and colleagues in 1933. George Phalen, an American orthopedic surgeon at the Cleveland Clinic, was credited with popularizing the use of the contemporary term beginning in the 1950s. Besides using the Tinel test to help support the diagnosis of CTS, Phalen also gave us the wrist flexion test that carries his name. Both the Tinel and Phalen tests have been widely used in clinical practice and for research. However, the lack of an agreed on standard method contribute to the variable sensitivity and specificity of the tests.

Many risk factors for CTS have been recognized early on such as pregnancy, space-occupying tumor, trauma (especially crush injuries), and work activities. The long list of known CTS risk factors includes age, rheumatoid arthritis, diabetes, hypothyroidism, obesity, square-shaped wrist, and many others. These risks can be divided into intrinsic (ie, genetic, biologic) and extrinsic (ie, environmental, activity-related) factors.
The present day understanding of the diagnosis and treatment of CTS incorporates contributions by many medical and surgical specialties, including orthopedics, psychiatry, neurology, rheumatology, neurosurgery, plastic surgery, chiropractic, osteopathy, and others. This frequently encountered condition typically presents with a constellation of signs and symptoms. The underlying pathogenesis has been well proven and accepted. It is now known that focal compression of the median nerve at the CT results in the symptoms of CTS as a consequence of injury to the sensory and motor nerves. Use of EDX medicine has proved to be indispensable in confirming the diagnosis of CTS.

ANATOMY OF THE CT

The CT is a dumb-bell-shaped passage, with an estimated width of 25 mm at its entrance and exit and 20 mm at its narrowest. The pathophysiology of CTS is closely related to the anatomy of the CT. The median nerve, which is derived from the lateral and the medial cord of the brachial plexus and C6 to T1 nerve roots, follows a path at the elbow close to the brachial artery. As the median nerve approaches the distal crease of the wrist, before entering the cephalic margin of the CT, it splits off the palmar cutaneous branch to provide sensation to the palmar skin over the thenar eminence. Inside the CT, the median nerve is accompanied by nine long flexor tendons, including the four tendons of the flexor digitorum superficialis, the four tendons of the flexor digitorum profundus, and the flexor pollicis longus tendon.

The walls of the CT are made of fibro-osseous tissue, which is inelastic and unyielding to pressure (Fig. 1). The dorsal and lateral aspects of the tunnel are formed by four carpal bone prominences, including the proximal lateral scaphoid, the proximal medial pisiform, the distal medial hook of the hamate, and the tubercle of the trapezium. The floor is formed by the lunate and capitate bones, and the volar side is formed by the fibrous transverse carpal ligament. The narrow space enclosed by

![Fig. 1. The anatomy of the CT. (From Cobb TK, Cooney WP, An K. Pressure dynamics of the carpal tunnel and flexor compartment of the forearm. J Hand Surg Am 1995;20:193–8; with permission.)](image)
the rigid wall and the crowded content make this segment of median nerve inside the CT particularly vulnerable to compression.

Intra-CT pressure plays a role in the pathophysiology of CTS. A recent study of fluid dynamics in CTS subjects demonstrated that intracarpal pressure is elevated and dissipation of this pressure is abnormally slow relative to controls.\textsuperscript{25,26} CT pressure measured in CTS subjects had a mean of 32 mm Hg, compared with 2.5 mm Hg in the healthy control group.\textsuperscript{27,28} Large myelinated nerve fibers in the CT are most susceptible to mechanical and ischemic damage. In CTS, microscopy has shown that disruption of myelin and the nodes of Ranvier results in conduction dysfunction and, when severe enough, axonal death.\textsuperscript{29}

The severity and the rate of neuropathic changes correlate not only with the degree and duration of the compression, but also with the acuity of pressure elevation. The natural history of most spontaneous CTS is that of gradual onset and slow progression, often over months if not years. Acute CTS is less common and usually associated with traumatic injuries and bony fractures. Anticoagulation-related hemorrhages into the CT and prolonged mechanical drilling have been reported as causes of acute CTS. It is not entirely clear how much the mechanical force and ischemia from microvascular compromise contributes to the pathogenesis of CTS. Morphologically, the median nerve observed during CT surgery often has a flattened, pale, and edematous appearance. Pulsatile blood flow in the median nerve is restored within minutes after the release of the transverse carpal ligament. Symptomatic pain relief reported by patients also happens quickly, often within days after the surgery. The recovery of the median neuropathy may take weeks to months and is often incomplete, supporting the view that the cause of the symptoms in the patient with CTS is more complex than just demyelination and axonal loss of the median nerve.

**CLINICAL EVALUATION OF CTS**

A directed history and physical examination is essential for the diagnosis of CTS. Pain, tingling, numbness, relief with hand shaking, and the dropping of objects are common complaints. Thumb abduction weakness and atrophy of the thenar eminence are positive predictors of CTS. The self-performed Katz Hand Diagram drawing is useful in assessing some patients for CTS, though it has a relatively low sensitivity of 0.64 and specificity of 0.73 according to one study.\textsuperscript{30} Numbness is typically appreciated in the thumb, index, and middle fingers but has been reported to be present in all fingers. Testing of light touch and pinprick sensation at the bedside typically demonstrates alterations in a median nerve distribution. Two-point discrimination or Semmes Weinstein monofilament testing is often used as well to help further define sensory deficits. Knowledge of the classic median nerve terminal branches serves to guide the physical examination. However, one should be aware that many variations and anomalies exist.\textsuperscript{31} As mentioned previously, the Tinel and Phalen tests may also be of value in assessing for CTS. The flick sign or arm shaking is worthwhile mentioning because it reportedly has the best sensitivity and specificity.\textsuperscript{32} In more severe cases of CTS, atrophy of the thenar eminence and thumb abduction and opposition weakness can be appreciated.

**ELECTRODIAGNOSIS OF CTS**

In essence, electrodiagnosis is an extension of the history and physical examination. The key finding for CTS is that of conduction slowing localized to the segment of the median nerve passing through the CT. Nerve conduction study (NCS) is more valuable than needle electromyography (EMG) study in general because of the underlying
pathophysiology of focal demyelination in CTS. Both the distal location and relative ease of CTS study contribute to the reliable nature of EDX in the assessment of this disorder. Meticulous attention to electrode placement, distance measurements, stimulation intensity, skin temperature, and many others factors are important to prevent misdiagnosis of CTS. It is recommended that the temperature of the upper limb be maintained at 32°C for NCS. There is no uniform agreement on the best location for the temperature probe.

**Sensory Conduction Studies**

In early and mild CTS, mild sensory nerve conduction slowing across the CT is often the only abnormal finding. The peak latency of the median sensory nerve action potential (SNAP) is typically delayed or prolonged. The use of peak latency for detecting CTS, instead of the onset latency, has become increasingly embraced by many electrodiagnosticians because of the frequent challenge in identifying the onset of the SNAP in the presence of a large stimulus artifact. Sensory amplitudes vary greatly among individuals and offer limited diagnostic value until the amplitude becomes smaller than the range of normal reference values. As CTS progresses, sensory peak latency is progressively delayed and the amplitude becomes smaller. In more advanced CTS, there is often no recordable SNAP even with signal averaging and enhancement.

There are many different protocols for median nerve sensory conduction study to help diagnose CTS. Using antidromic techniques with stimulation applied to the wrist, median SNAPs can be recorded at the thumb, the index, the long, and the ring fingers. Median sensory fiber electrical signals can also be recorded in the long finger with the application of two stimuli, one at the wrist and one at the palm. When present, relative sensory nerve conduction slowing in the proximal segment can reveal focal median sensory nerve injury localized to the CT. Conduction block is suspected when the SNAP elicited with proximal stimulation is 50% or less in amplitude than that recorded with stimulation applied distal to the wrist. Proximal segmental conduction slowing relative to a more distal segment helps in differentiating CTS from length-dependent peripheral neuropathy. The method of sensory “inching” along the median nerve between the wrist and palm is not regularly used, but could potentially help localize conduction slowing to a short segment of the median nerve.

**Motor Conduction Studies**

Median motor nerve conduction is usually obtained recording over the abductor pollicis brevis (APB) muscle. Delay of the distal motor latency (DML) supports the diagnosis of CTS. However, by itself, mild prolongation of the median DML does not absolutely suggest focal demyelination. The preferential loss of the large fast-conducting myelinated nerve fibers could also result in slower nerve conduction and delayed DML. It has been shown that mild proximal conduction slowing can occur in CTS and, therefore, a decreased median motor nerve conduction velocity in the forearm does not exclude the diagnosis of CTS. Comparing the conduction velocity and DML of the median nerve to that of the ulnar nerve, which travels outside the CT, helps to confirm CTS and differentiate it from length-dependent peripheral polyneuropathy. One should also be aware of the possibility of anatomic variants such as a Martin-Gruber anastomosis in the forearm, but its presence generally does not cause confusion in the diagnosis of CTS in contrast to EDX for ulnar neuropathy at the elbow.

Short segment studies testing the recurrent median motor branch distal to the compression site have the potential for more precisely localizing a median neuropathy...
to the CT. In performing such testing, distal motor conduction block can sometimes be demonstrated as consequence of significant focal motor nerve demyelination. However in practice, the short distance between stimulation and recording sites and the close proximity of the recurrent branch of the median motor nerve to the nearby ulnar motor nerve make such short segment testing challenging.

**Needle EMG Studies**

Needle EMG is often helpful in further characterizing the neuropathic insult, especially when the compound muscle action potential amplitude is reduced because this can be a consequence of either distal demyelination or denervation. Needle EMG can provide evidence of denervation by the presence of membrane instability, such as fibrillation potentials and positive sharp waves, and altered motor unit morphology. With severe CTS, electrodiagnostic evidence of axonal loss of motor nerves and subsequent motor unit reorganization can be seen in the intrinsic hand muscles innervated by the median nerve, including the APB, the superficial head of the flexor pollicis brevis, opponens pollicis, and the first and second lumbricals. However, axon loss of motor nerves is generally uncommon in CTS. Also, fibrillation potentials and positive sharp waves cannot be used to quantify the extent of motor axon loss. For most CTS cases, the use of needle EMG is debated.

**Latency Differences in CTS**

There are two methods to decide whether the median nerve latency is normal or prolonged, both of which require normal reference values obtained from healthy individuals without CTS. The first is the absolute latency method. Using this method, the median sensory or motor latency is compared against the normal reference value. Conduction slowing is determined if the latency exceeds the upper limit of normal. This absolute latency method has several drawbacks, especially for sensory conduction because the skin temperature effects and other factors. The recommendation is that each EDX laboratory establishes its own normal data. Unfortunately, this is not always achievable.

An increasing awareness of variation among normal reference values from laboratory to laboratory has led to the comparison reference value method. This newer method has been well described and was recommended for use in diagnosing CTS in a recent 2011 American Association of Neuromuscular and Electrodiagnostic Medicine (AANEM) monograph. In this method, conduction study of the median nerve is compared with that of another nerve, usually in the same limb and typically the ulnar. Comparison reference values from different investigators show less variation, because of better built-in controls for differences in subjects, techniques, and equipments.

For instance, for the assessment of CTS, latency measurements can be compared using median–thenar and ulnar–hypothenar pairs. With this protocol, a DML difference of greater than 1.2 to 1.8 ms is said to be significant. This technique is especially useful when studying patients who have generalized peripheral polyneuropathy, most commonly from diabetes. As supporting evidence for probable CTS, this author uses a difference of greater than 2.0 ms comparing the median DML with the ipsilateral ulnar DML. When there are no sensory responses in the upper extremities of a diabetic patient who has additional unilateral hand paresthesias and weakness, such a motor comparison can be quite helpful to demonstrate evidence for CTS in the presence of diabetic peripheral neuropathy. Another useful technique in this setting of possible peripheral neuropathy and superimposed CTS is a comparison of the difference in DML between the median-innervated second lumbrical muscle and the
The Combined Sensory Index

Though the best electrodiagnostic test for CTS has yet to be determined, many studies for CTS have been designed over the last few years. For instance, using the absolute method, one can assess for CTS by obtaining median sensory latency recording from the thumb, and the index, long, and ring fingers. Using the comparison method, CTS can be diagnosed by determining median-radial sensory latency differences to the thumb, median-ulnar sensory differences to the ring finger, and transpalmar median-ulnar latency differences. An important question is whether one normal test is sensitive enough to refute the diagnosis of CTS. Because CT compression can affect some median nerve fascicles earlier or preferentially, one electrodiagnostic test may demonstrate CTS when another does not. Performing limited studies could reduce the sensitivity of EDX considerably, causing a false negative error. However, increasing the number of tests also likely increases the probability of reducing the specificity of testing, causing a false positive error in proportion to the number of tests performed. Although a false negative error may limit early detection and treatment, a false positive error could result in overtreatment, which has the potential to cause harm to the patient.

The combined sensory index (CSI) is an attempt to maximize sensitivity for detecting CTS without reducing specificity by using a single score derived from multiple sensory tests. The CSI, sometimes called the Robinson index, is easy to obtain. The CSI is the sum of comparisons of sensory latencies collected with three established sensory tests for the study of CTS using the following formula: CSI = ringdiff + thumbdiff + palmdiff. In this formula, ringdiff is the peak latency difference of the median and ulnar antidromic sensory nerve conduction to the ring finger stimulating 14 cm proximally, the thumbdiff is the peak latency difference of the median and radial antidromic sensory nerve conduction to the thumb stimulating 10 cm proximally, and the palmdiff is the transpalmar peak latency difference of the median and ulnar orthodromic conduction using a distance of 8 cm. For example, if the latencies are 3.8 ms for median nerve conduction to the ring finger and 3.4 ms for ulnar nerve conduction to the ring finger, then the ringdiff is 0.4. If the median latency is 3.4 ms and the ulnar latency is 3.8 ms, then the ringdiff would be a negative number (−0.4). Using negative numbers helps to cancel random errors such as distance measurement and account for ulnar or radial sensory neuropathy if present.

The CSI has a high specificity (ie, few false positives), a high sensitivity (ie, few false negatives), and excellent test-retest reliability. When the upper limit of the normal CSI is set at 0.9 ms, the test sensitivity is 0.83 and the specificity 0.95. If the normal upper limit for the CSI is raised from 0.9 ms to 1.1 ms, the sensitivity remains essentially the same at 0.82; however, the specificity increases to 1.00 indicating very low if any probability of false positive error. Generally, a CSI of 1.0 ms or greater would be consistent with CTS. The test and retest reliability for the CSI has been shown to be superior to that of other techniques in identifying CTS because of improved control for variables such as hand size, height, age, and temperature.

Study Approaches and Reporting of CTS

In the author’s laboratory, CTS is considered with any clinical presentation of pain, numbness, paresthesias, and weakness in the upper extremities. Other potential diagnoses, such as arthritis, tendinopathy, polyneuropathy, brachial plexopathy, or cervical radiculopathy, are also entertained. Clinical impression of the likelihood of
CTS from history and physical examination is formed before EDX study. The author usually begins with median motor nerve conduction, followed by ulnar motor nerve conduction, and then the three sensory tests that form the CSI study. When bilateral CTS is suspected, the more symptomatic hand is studied first. If the median nerve is normal, the less symptomatic side is not always studied. The author does not perform NCS in the asymptomatic limb for the purpose of contralateral comparison of the median nerves given the significant incidence of bilateral EDX abnormalities in some asymptomatic population. This is in accordance with AANEM guidelines.

For most patients referred to our EDX laboratory with possible CTS, the author performs all three CSI sensory tests and reports the calculated CSI. The added time, cost, and discomfort to the patient of these studies are quite reasonable. The benefit of obtaining a complete CSI is its excellent reproducibility. Repeat CTS referrals on the same patients are fairly common in the author’s institution. These patients have recurrent CTS after a period of satisfactory symptom relief using conservative treatments. Comparison of two CSI scores could provide information in regard to progression of CTS and is potentially of help to the patient and the treating physician in making treatment choices. A change of the CSI in such repeat studies of greater than 0.3 ms is considered to be statistically significant. The author is occasionally asked to study the patient who has persistent hand pain and numbness after CT release surgery. The comparison of two CSI scores obtained before and after the operation, when available, is useful in differentiating residual median neuropathy versus incomplete release or recurrent CTS, which is rare.

For nerve conduction studies, the author prefers warming the upper limb rather than extrapolating using a latency or conduction velocity corrected for temperature. Individual thermostats in the study rooms help regulate room temperature to minimize skin temperature drop during the study, which is usually between 1°C and 2°C in our laboratory. Warming pads are used to maintain skin temperature, though a heat lamp or a hand-held hair dryer can also work. The author does not routinely perform needle EMG for CTS evaluation.

Needle EMG is performed if there is concern for the possibility of ulnar neuropathy, brachial plexopathy, cervical radiculopathy, or if there is a history of trauma. For example, non–median innervated C8 muscles, such as the first dorsal interosseus, abductor digiti minimus, or extensor indicis proprius, are examined in addition to the APB muscle if C8 radiculopathy is suspected. Needle EMG study of median-innervated forearm muscles, such as the pronator quadratus and pronator teres, help to differentiate CTS from less common median nerve entrapment syndromes that occur proximal to the wrist.

When providing the patient with CTS and the referring clinician with our EDX impression, the author confirms the localization of a median neuropathy at the wrist and comments on the electrodiagnostic evidence or sensory nerve involvement only or if there is focal demyelination of the motor nerve. The severity of the demyelination is also commented on in reference to the degree of conduction slowing and the presence or absence of motor conduction block. Denervation from CTS is rare, but when present, the extent and chronicity are also discussed in the report.

No matter the EDX protocol used, some patients with the clinical symptoms of CTS have negative findings. Conservative treatment (see later discussion) is a satisfactory approach for many of these patients. Conversely, EDX CTS in patients without clinical CTS symptoms should be reported with caution to referring clinicians to avoid the potential risk of overtreatment. A prevalence study of the Swedish general population found 18.4% of a control group to have abnormal EDX findings characteristic for CTS.
TREATMENT OPTIONS FOR CTS

Acute CTS associated with trauma and bone fractures often requires timely open exploration and decompression, and EDX could be performed at the time of surgery or after the postoperative follow-up. For most subacute and chronic CTS, initial treatments are designed to relieve the uncomfortable and often disabling symptoms using nonsurgical methods. Early and mild symptoms of CTS are frequently self-limiting and often resolve within weeks. Simple and noninvasive treatments can provide significant symptomatic relief and protect the median nerve from further injury. Pain in CTS can be alleviated with oral medications, such as Tylenol, nonsteroidal antiinflammatory medications, and gabapentin. Oral steroids are less commonly used. Treatment should be considered for underlying conditions that could predispose to CTS, such as diuretics for fluid retention, thyroid supplementation for hypothyroidism, insulin for diabetes, and immune modulating agents for rheumatoid arthritis. A nighttime wrist brace that reduces nocturnal wrist flexion is often of benefit. A recent prospective study showed that local steroid injections into the CT can be effective in treating CTS44,45 and should be considered as an option. Educating the patient about avoiding overuse and activity modification is essential to prevent recurrence.

When pain persists and interferes with sleep and daily activities, the surgical option should be considered and supported. In reference to the CSI, outcomes of surgical release have been carefully examined in a few recent studies. Interestingly, patients with a CSI between 2.5 and 4.6 gain the most from carpal tunnel release (CTR).46 However, improvement following CTR is also reported by many patients with a normal CSI and similarly in some patients with advanced chronic CTS. A decision to recommend CTR should be based not just on the electrodiagnostic abnormalities but also other physical and psychosocial and vocational factors that could play a role in the patient’s functional recovery from such surgery. For instance, a recently published article reports that before surgery, whether or not and the duration of worker’s compensation is a strong positive predictor for long-term disability related to CTS.47

SUMMARY

CTS will likely continue to be a common problem encountered by the electrodiagnostician, given recent evidence suggesting an increasing incidence of the condition. New modalities, such as MRI and ultrasound are being applied to CTS evaluation and can potentially add important information about the anatomy and morphology of the CT, median nerve, and nearby tissue.48–50 With further refinement and higher resolution of imaging techniques, important information regarding median nerve, edema, inflammation, as well as the milieu of the surrounding CT, will be, it is hoped, provided, supplementing the EDX and clinical findings. Although CTS is a condition that is relatively easy to recognize, investigations aim for early detection, effective treatment including outcomes are warranted with the ultimate goals to alleviate suffering and disability.

REFERENCES

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