17 Median Neuropathy at the Wrist

Median nerve entrapment at the wrist is the most common of all entrapment neuropathies and, consequently, is one of the most frequent reasons for referral for an electrodiagnostic (EDX) study. In nearly all patients, the usual site of compression occurs in the carpal tunnel and results in a constellation of symptoms and signs known as the *carpal tunnel syndrome* (CTS). Lesions of the C6–C7 nerve roots or, less often, the brachial plexus and the proximal median nerve may be confused clinically with median neuropathy at the wrist, especially in early or mild cases.

For an electromyographer, familiarity with the various nerve conduction and electromyographic patterns associated with CTS is essential. It has long been recognized that in any individual patient with CTS, there may be little correlation between the degree or frequency of clinical symptoms or signs and the abnormalities seen on nerve conduction studies. For example, an occasional patient will have only mild or trivial clinical symptoms yet will have clear signs on physical examination (e.g., dense numbness, wasting of thenar muscles) and evidence of severe axonal loss on nerve conduction and needle electromyography (EMG) studies. On the other hand, there are patients whose clinical history clearly indicates CTS but who show few or no abnormalities on neurologic examination or on routine median motor and sensory nerve conduction studies. It is in these latter patients with early or electrically mild CTS that additional more sensitive nerve conduction studies must be performed in order to demonstrate median nerve slowing at the wrist. By appropriately applying the various electrophysiologic techniques available to study the median nerve, a definite diagnosis can usually be reached, and lesions of the nerve roots, proximal median nerve, or brachial plexus can be excluded.

ANATOMY

Understanding the anatomy of the median nerve is the first step toward being able to differentiate entrapment of the median nerve at the wrist from lesions of the proximal median nerve, brachial plexus, and cervical nerve roots, on both clinical and electrophysiologic grounds. The median nerve is formed by a combination of the *lateral and medial cords* of the brachial plexus (Table 17–1, Figure 17–1). The lateral cord is made up of C6–C7 fibers and supplies median sensory fibers to the thenar eminence, thumb, index, and middle fingers, and motor fibers to the proximal median forearm muscles. The medial cord, composed of C8–T1

Table 17–1. Median Nerve Innervation

	Median Branch	Cord	Trunk	Root
Muscle				
Pronator teres	(Main median nerve)	Lateral	Upper/middle	C6–C7
Flexor carpi radialis	(Main median nerve)	Lateral	Upper/middle	C6–C7
Flexor digitorum sublimis	(Main median nerve)	Lateral/medial	Middle/lower	C7–C8
Flexor digitorum profundus (2,3)	Anterior interosseous	Lateral/medial	Middle/lower	C7–C8
Flexor pollicis longus	Anterior interosseous	Lateral/medial	Middle/lower	C7–C8–T1
Pronator quadratus	Anterior interosseous	Lateral/medial	Middle/lower	C7–C8–T1
Abductor pollicis brevis	Recurrent thenar	Medial	Lower	C8–T1
Opponens pollicis	Recurrent thenar	Medial	Lower	C8–T1
Flexor pollicis brevis (superficial head)	Recurrent thenar	Medial	Lower	C8–T1
Sensory area				
Thenar eminence	Palmar cutaneous	Lateral	Upper	C6
Medial thumb	Digital branch	Lateral	Upper	C6
Index finger	Digital branches	Lateral	Upper/middle	C6–C7
Middle finger	Digital branches	Lateral	Middle	C7
Lateral ring finger	Digital branch	Lateral/medial	Middle/lower	C7–C8

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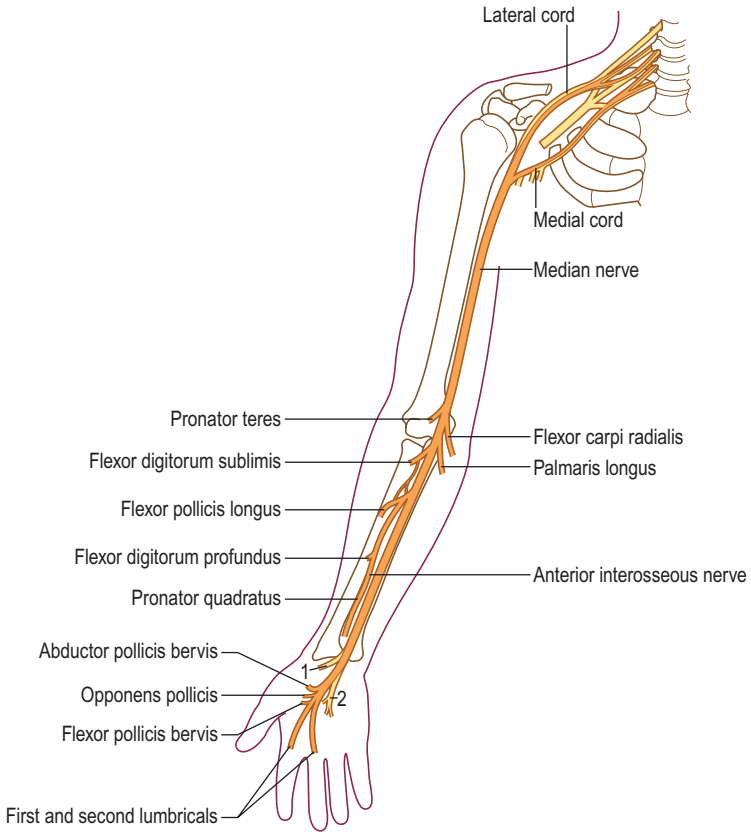

FIGURE 17–1 Anatomy of the median nerve. The median nerve is derived from a combination of the lateral and medial cords of the brachial plexus. Motor innervation is supplied to forearm muscles and to muscles of the thenar eminence. Sensation is supplied to the thenar eminence by the palmar cutaneous sensory branch (1) and to the first three and one-half digits by several digital sensory branches (2). (Adapted with permission from Haymaker, W., Woodhall, B., 1953. Peripheral nerve injuries. WB Saunders, Philadelphia.)

fibers, supplies motor fibers to the median muscles of the distal forearm and hand, as well as sensory fibers to the lateral half of the ring finger.

The median nerve descends in the upper arm, giving off no muscular branches. In the antecubital fossa, the nerve lies adjacent to the brachial artery. As it passes into the forearm, the median nerve runs between the two heads of the pronator teres (PT) before giving off muscular branches to the PT, flexor carpi radialis (FCR), flexor digitorum sublimis (FDS), and, in some individuals, the palmaris longus muscles. The *anterior interosseous nerve* is given off next in the proximal forearm, innervating the flexor pollicis longus (FPL), the medial head of the flexor digitorum profundus (FDP) to the index and middle fingers, and the pronator quadratus (PQ) muscles. The anterior interosseous nerve is considered a pure motor nerve clinically because it carries no cutaneous sensory fibers. However, deep sensory fibers are carried in the anterior interosseous nerve, supplying the wrist joint and interosseous membrane.

Just proximal to the wrist and carpal tunnel, the *palmar cutaneous sensory branch* arises next, running subcutaneously to supply sensation over the thenar eminence. The median nerve then enters the wrist through the carpal tunnel. Carpal bones make up the floor and sides of the carpal tunnel, and the *thick transverse carpal ligament* forms the roof (Figure 17–2). In addition to the median nerve, nine flexor tendons traverse the carpal tunnel as

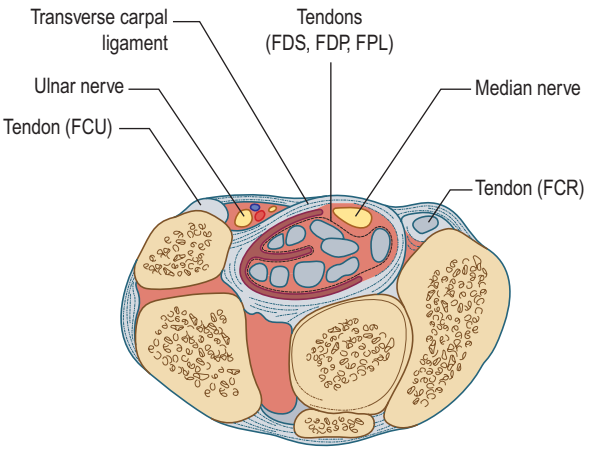

FIGURE 17–2 Anatomy of the median nerve at the carpal tunnel. At the wrist, the median nerve runs through the carpal tunnel, along with nine flexor tendons. Carpal bones form the floor and sides of the carpal tunnel; the thick transverse carpal ligament forms the roof. FCR, flexor carpi radialis; FCU, flexor carpi ulnaris; FDP, flexor digitorum profundus; FDS, flexor digitorum sublimis; FPL, flexor pollicis longus. (Reprinted with permission from Pecina, M.M., Krmpotic, Nemanic, J., Markiewitz, A.D., 1991. Tunnel syndromes. CRC Press, Boca Raton, FL.)

well (FDP: four tendons; FDS: four tendons; FPL: one tendon). In the palm, the median nerve divides into motor and sensory divisions. The motor division travels distally into the palm, supplying the first and second lumbricals (1L, 2L). In addition, the *recurrent thenar motor branch* is given off. This branch turns around (hence, recurrent) to supply muscular branches to most of the thenar eminence, including the opponens pollicis (OP), abductor pollicis brevis (APB), and superficial head of the flexor pollicis brevis (FPB). The sensory fibers of the median nerve that course though the carpal tunnel supply the medial thumb, index finger, middle finger, and lateral half of the ring finger. The index and middle fingers are each supplied by two digital branches (one lateral and one medial); the thumb and ring fingers receive only one branch each (Figure 17–3).

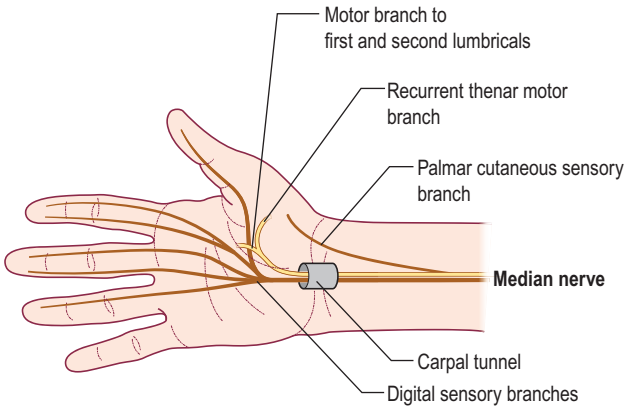

FIGURE 17–3 Distal motor and sensory branches of the median nerve. Proximal to the carpal tunnel, the palmar cutaneous sensory branch arises to supply sensation to the thenar eminence. Distal to the carpal tunnel, the median nerve divides into sensory and motor branches. Digital sensory branches supply the index and middle fingers and part of the thumb and fourth finger. Motor fibers supply the first and second lumbricals, while the recurrent thenar motor branch innervates most muscles of the thenar eminence.

CLINICAL

Patients with CTS may present with a variety of symptoms and signs (Table 17–2). Women are affected more often than men. Although CTS usually is bilateral both clinically and electrically, the dominant hand usually is more severely affected, especially in idiopathic cases. Patients complain of wrist and arm pain associated with paresthesias in the hand. The pain may be localized to the wrist or may radiate to the forearm, arm, or, rarely, the shoulder; *the neck is not affected*. Some patients may describe a diffuse, poorly localized ache involving the entire arm. Paresthesias are frequently present in the median nerve distribution (medial thumb, index, middle, and lateral ring fingers). Although many patients report that the entire hand falls asleep, if asked directly about little finger involvement, most will subsequently note that the little finger is spared.

Symptoms often are provoked when either a flexed or extended wrist posture is assumed. Most commonly, this occurs during ordinary activities, such as driving a car or holding a phone, book, or newspaper. *Nocturnal paresthesias are particularly common*. During sleep, persistent wrist flexion or extension leads to increased carpal tunnel pressure, nerve ischemia, and subsequent paresthesias. Patients frequently will awaken from sleep and shake or wring their hands out or hold them under warm running water.

Sensory fibers are involved early in the majority of patients. Pain and paresthesias usually bring patients to medical attention. Motor fibers may become involved in more advanced cases. Weakness of thumb abduction and opposition may develop, followed by frank atrophy of the thenar eminence. Some patients describe difficulty buttoning shirts, opening jars, or turning doorknobs. However, development of significant functional impairment from loss of median motor function in the hand is unusual.

The sensory examination may disclose hypesthesia in the median distribution. Comparing sensation over the lateral ring finger (median innervated) to that over the medial

Table 17–2. Clinical Symptoms and Signs

Highly Suggestive of Carpal Tunnel Syndrome	Possible Carpal Tunnel Syndrome	Inconsistent with Carpal Tunnel Syndrome
Nocturnal paresthesias awakening patient from sleep	Hand, wrist, forearm, arm, and/or shoulder pain	Neck pain
Shaking or ringing the hands		
Pain/paresthesias associated with driving or holding a phone, book, or newspaper	Perception of paresthesias involving all five digits	Paresthesias radiating from neck and shoulder down the arm
Sensory disturbance of digits 1,2, 3, and 4, splitting the fourth digit	No fixed sensory disturbance, or sensory disturbance of digits 1, 2, 3, and/or 4	Unequivocal numbness over the thenar eminence
Weakness/wasting of thenar eminence	Decreased hand dexterity	Weakness/wasting of hypothenar muscles, thumb flexion (interphalangeal joint), arm pronation, and/or elbow flexion/extension
Phalen's maneuver reproduces symptoms	Tinel's sign over the median nerve at the wrist	Reduced biceps or triceps reflexes

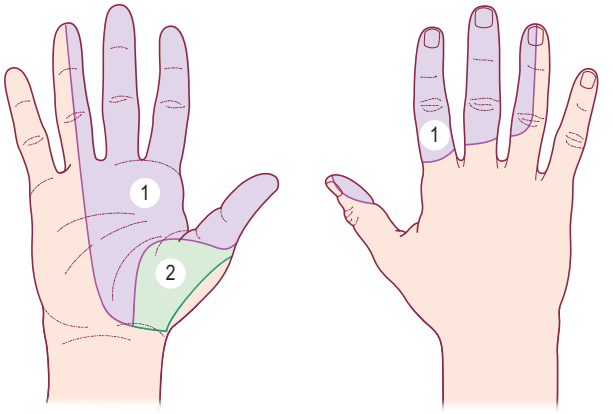

FIGURE 17–4 Typical median sensory territory. The median sensory territory is innervated by the palmar digital sensory branches (1) and the palmar cutaneous sensory branch (2). In most individuals, digit 4 is innervated by median and ulnar nerves; rarely, digit 4 may be all median or all ulnar. Only the digital sensory branches travel through the carpal tunnel resulting in the pattern of sensory loss seen in carpal tunnel syndrome (1). In contrast, sensation over the thenar area is normal in carpal tunnel syndrome (2).

FIGURE 17–5 Provocative test for carpal tunnel syndrome: Tinel's sign. The Tinel's sign is elicited by tapping over the median nerve in the center of the wrist. If abnormal, the patient will report paresthesias radiating into one or more median-innervated digits.

FIGURE 17–6 Provocative test for carpal tunnel syndrome: Phalen's maneuver. The Phalen's maneuver is performed by placing the wrist in a flexed posture **(top).** This position increases pressure within the carpal tunnel and may provoke paresthesias radiating into median-innervated digits (especially digit 3) in patients with carpal tunnel syndrome. The pressure also increases and median paresthesias may result if the wrist is placed in an extended posture **(bottom),** sometimes known as the "reverse Phalen's maneuver."

ring finger (ulnar innervated) is often helpful. *Sensation over the thenar area is spared because this area is innervated by the palmar cutaneous sensory branch, which arises proximal to the carpal tunnel* (Figure 17–4). The *Tinel's sign* is often present when tapping over the median nerve at the wrist, which results in paresthesias in the median-innervated fingers (Figure 17–5). The *Phalen's maneuver*, whereby the wrist is held passively flexed, may also provoke symptoms (Figure 17–6, top). A wide range of sensitivities and specificities for the Tinel's sign and Phalen's maneuver have been reported in the literature. A Tinel's sign is present in more than half of CTS cases; however, false-positive Tinel's signs are common in the general population. A Phalen's maneuver usually produces paresthesias within 30 seconds to 2 minutes in CTS; it is more sensitive than the Tinel's sign and has fewer false-positive results. Most commonly, the Phalen's maneuver will produce paresthesias in the middle or index fingers. It should be noted, however, that because the Phalen's maneuver often is performed with the elbow flexed as well (a provocative maneuver for ulnar neuropathy at the cubital tunnel), this position occasionally may produce ulnar paresthesias in patients with ulnar neuropathy.

The motor examination involves inspection of the hand, looking for wasting of the thenar eminence (severe cases), and testing the strength of thumb abduction and opposition (Figure 17–7). Isolating the actions of the APB and OP (median-innervated muscles distal to the carpal tunnel) may be difficult because thumb abduction is also served by the abductor pollicis longus (radial nerve) and thumb opposition by a combination of the deep head of the FPB

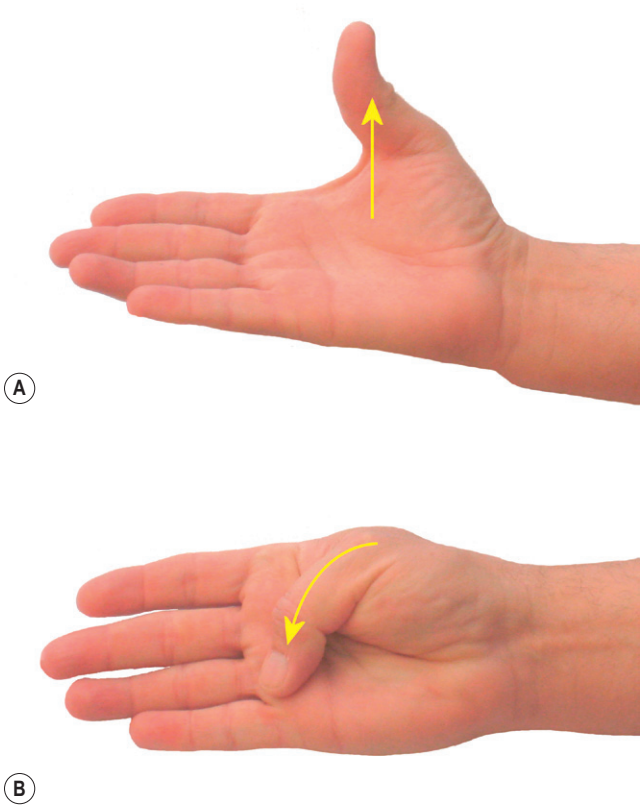

FIGURE 17–7 Muscle testing in carpal tunnel syndrome. Thumb abduction **(A)** and opposition **(B)** may be weak in more advanced cases of carpal tunnel syndrome.

(innervated by the ulnar nerve) and the FPL (innervated by the anterior interosseous nerve).

It is important to emphasize that CTS is a clinical diagnosis. It represents a constellation of clinical symptoms and signs caused by compression and slowing of the median nerve at the wrist. However, there are patients who have median nerve slowing at the wrist on nerve conductions but who have no clinical signs or symptoms. Such patients do not have CTS *per se* and do not need directed therapy. This situation is encountered most often in patients with an underlying polyneuropathy in whom preferential slowing at common sites of compression is not unusual. Often, patients with an underlying polyneuropathy may be found to have incidental slowing at several entrapment sites, including the median nerve at the wrist, ulnar nerve at the elbow, and peroneal nerve at the fibular neck. For example, a patient with numbness and tingling of both feet from a mild alcohol-induced or diabetic polyneuropathy may have relative slowing of the median nerve across the wrist on nerve conduction studies yet may have no complaints of pain, paresthesias, or weakness in the hands. According to the EDX studies, such a patient has a median neuropathy at the wrist superimposed on an underlying polyneuropathy, but the patient does not have CTS. This distinction is important, because in this case treatment with splinting, injection, or surgery is not appropriate. The point is again underscored that *nerve conduction and EMG studies can be properly performed and interpreted only with knowledge of the clinical history and physical examination.*

Box 17–1. Conditions Associated with Carpal Tunnel Syndrome

- Idiopathic disorders
  - Repetitive stress
  - Occupational
- Endocrine disorders
  - Hypothyroidism
  - Acromegaly
  - Diabetes
- Connective tissue disease
  - Rheumatoid arthritis
- Tumors
  - Ganglia
  - Lipoma
  - Schwannoma
  - Neurofibroma
  - Hemangioma
- Congenital disorders
  - Persistent median artery
  - Congenital small carpal tunnel
  - Anomalous muscles (palmaris longus, flexor digitorum sublimis)
- Infectious/inflammatory
  - Sarcoid
  - Histoplasmosis
  - Septic arthritis
  - Lyme
  - Tuberculosis
- Trauma
  - Fractures (especially Colles' fracture)
  - Hemorrhage (including anticoagulation)
- Other
  - Spasticity (persistent wrist flexion)
  - Hemodialysis
  - Amyloidosis (familial and acquired)
  - Pregnancy
  - Any condition that increases edema or total body fluid

ETIOLOGY

The reported causes of CTS are numerous (Box 17–1). *Despite this exhaustive list, most cases are idiopathic.* Indeed, idiopathic cases present with the same signs and symptoms as CTS caused by the other conditions listed in Box 17–1. Although the etiology of idiopathic cases was long considered to be tenosynovitis of the transverse carpal ligament, pathologic evaluation typically shows little evidence of inflammation. In most cases, edema, vascular sclerosis, and fibrosis are seen, findings consistent with repeated stress to connective tissue. Compression results in symptoms by way of ischemia and demyelination and, if it is severe enough, wallerian degeneration and axonal loss.

Occupations or activities that involve repetitive hand use clearly increase the risk of CTS (e.g., typists, data entry workers, mechanics, and carpenters). From the exhaustive list given in Box 17–1, the conditions most often associated with CTS, other than idiopathic, are diabetes, hypothyroidism, rheumatoid arthritis, amyloidosis, and pregnancy.

One important clue to an underlying cause, other than idiopathic, is the presence of CTS in the non-dominant hand. In idiopathic cases, the dominant hand is nearly always the affected hand; if symptoms are bilateral, then the dominant hand is more affected than the contralateral hand. CTS that is significantly worse in the non-dominant hand should raise a red flag to a specific underlying cause other than idiopathic CTS.

DIFFERENTIAL DIAGNOSIS

There are several peripheral as well as central nervous system (CNS) lesions that may result in symptoms similar to CTS. The peripheral lesions that enter into the differential diagnosis include median neuropathy in the region of the elbow, brachial plexopathy, and cervical radiculopathy. The most common among the disorders that may be confused with CTS is cervical radiculopathy, especially lesions of the C6 or C7 root, which may cause both pain in the arm and paresthesias similar to those that characterize CTS. The important clinical clues that suggest radiculopathy rather than CTS are pain in the neck, radiation from the neck to the shoulder and arm, and exacerbation of symptoms by neck motion. Key points in the physical examination that suggest radiculopathy are abnormalities of the C6–C7 reflexes (biceps, brachioradialis, triceps), diminished power in proximal muscles (especially elbow flexion, elbow extension, arm pronation), and sensory abnormalities in the palm or forearm, which are beyond the distribution of sensory loss found in CTS.

Median neuropathy at the elbow and brachial plexopathy are very uncommon, especially in comparison to the incidence of CTS. If present, however, they may easily lead to clinical confusion. Important clues on physical examination that suggest a more proximal lesion of the median nerve are sensory disturbance over the thenar eminence and weakness of median innervated muscles proximal to the carpal tunnel, especially distal thumb flexion (FPL), arm pronation (PT and PQ), and wrist flexion (FCR). In brachial plexus lesions, the neurologic examination may reveal abnormalities similar to those noted in cervical radiculopathy, although the distribution of reflex abnormalities, weakness, and sensory loss may be more widespread, beyond the distribution of one spinal segment.

As for CNS disorders, transient paresthesias may be seen in patients with focal seizures, migraine, and transient ischemic attacks and occasionally are misinterpreted as symptoms of CTS. In exceptional cases, patients referred to the EMG laboratory for suspicion of CTS will be found to have a small lacunar infarct involving the lateral thalamus and internal capsule, causing hand clumsiness and sensory disturbance predominantly affecting the median-innervated digits. In addition to the presence of other evidence of CNS dysfunction, such as limb spasticity and brisk reflexes, the major differentiating factor is the lack of pain. One should always question the diagnosis of CTS in the absence of pain.

ELECTROPHYSIOLOGIC EVALUATION

The electrophysiologic evaluation of a patient suspected of having CTS is directed toward the following:

1. Demonstrating focal slowing or conduction block of median nerve fibers across the carpal tunnel
2. Excluding median neuropathy in the region of the elbow
3. Excluding brachial plexopathy predominantly affecting the median nerve fibers
4. Excluding cervical radiculopathy, especially C6 and C7
5. If a coexistent polyneuropathy is present, ensuring that any median slowing at the wrist is out of proportion to slowing expected from the polyneuropathy alone

Nerve Conduction Studies

The nerve conduction strategy for evaluating possible CTS is outlined in Box 17–2. The pathophysiology of CTS typically is demyelination, which, depending on the severity, may be associated with secondary axonal loss. In moderate to advanced cases, the electrodiagnosis usually is straightforward. On routine median studies, a demyelinating lesion at the carpal tunnel results in slowing of the distal motor and sensory latencies. If there is either demyelination with conduction block or axonal loss, the distal compound muscle action potential (CMAP) and sensory nerve action potential (SNAP) amplitudes, stimulating the median nerve at the wrist, will be decreased as well.

In patients with typical CTS, the median distal motor and sensory latencies, and minimum F wave latencies, are moderately to markedly prolonged. However, there are a group of patients with clinical symptoms and signs of CTS in whom these routine studies are normal (approximately 10–25% of CTS patients). In such patients, the electrodiagnosis of CTS will be missed unless further testing is performed using more sensitive nerve conduction studies. Those studies usually involve a comparison of the median nerve to another nerve in the same hand. The ulnar nerve is the nerve most commonly used for comparison; less often the radial nerve is used.

The common median-versus-ulnar comparison tests are (1) median-versus-ulnar palm-to-wrist mixed nerve latencies, (2) median-versus-ulnar wrist-to-digit 4 sensory latencies, and (3) median (second lumbrical)-versus-ulnar (interossei [INT]) distal motor latencies. In each of the comparison studies, identical distances between the stimulator and recording electrodes are used for the median and ulnar nerves. These techniques create an ideal internal control in which several variables that are known to affect conduction time are held constant, including distance, temperature, age, and nerve size. Ideally, the only factor that varies in these paired median-versus-ulnar comparison studies is that the median nerve traverses the carpal tunnel, whereas the ulnar nerve does not. Thus, any

Box 17–2. Recommended Nerve Conduction Study Protocol for Carpal Tunnel Syndrome

Routine studies

1. Median motor study recording abductor pollicis brevis, stimulating wrist and antecubital fossa
2. Ulnar motor study recording abductor digiti minimi, stimulating wrist, below groove, and above groove
3. Median and ulnar F responses
4. Median sensory response, recording digit 2 or 3, stimulating wrist
5. Ulnar sensory response, recording digit 5, stimulating wrist
6. Radial sensory response, recording snuffbox, stimulating over the lateral radius

The study is highly suggestive of isolated carpal tunnel syndrome if

The median studies are abnormal, showing marked slowing across the wrist (prolonged distal motor and sensory latencies), and prolonged minimum F wave latencies. The median compound muscle action potential (CMAP) and sensory nerve action potential (SNAP) amplitudes may be diminished if there is secondary axonal loss or if demyelination has led to conduction block at the wrist

and

The ulnar motor, sensory, and F wave studies are normal and the radial sensory response is normal (making a brachial plexopathy or polyneuropathy unlikely)

No further nerve conductions are necessary, proceed to electromyography (EMG).

If the median studies are completely normal or equivocal, proceed with the median-versus-ulnar comparison tests, the median-versus-radial comparison test, or the median segmental sensory study.

Median-versus-ulnar comparison studies

1. Comparison of the median and ulnar mixed palm-to-wrist peak latencies, stimulating the median and ulnar palm one at a time 8 cm from the recording electrodes over the median and ulnar wrist, respectively
2. Comparison of the median lumbrical and ulnar interossei distal motor latencies, stimulating the median and ulnar wrist one at a time at identical distances (8–10 cm), recording with the same electrode over the 2L/interossei
3. Comparison of the median and ulnar digit 4 sensory latencies, stimulating the median and ulnar wrist one at a time at identical distances (11–13 cm) and recording digit 4

Median-versus-radial comparison study

1. Comparison of the median and radial digit 1 sensory latencies, stimulating the median nerve at the wrist and the superficial radial sensory nerve at the forearm one at a time at identical distances (10–12 cm) and recording digit 1

Median segmental sensory study

1. While recording digit 3, stimulate the median nerve at the wrist and in the palm (with the palm-to-digit distance being one-half of the wrist-to-digit distance). Then calculate the wrist-to-palm conduction velocity and compare it to the palm-to-digit conduction velocity

If two or more of the above studies are abnormal, there is a high likelihood of carpal tunnel syndrome. Proceed to EMG. If these studies are normal, consider alternative diagnoses, especially cervical radiculopathy (note: a small number of patients with CTS can have normal NCSs).

Other important considerations:

1. If there is a co-existent polyneuropathy, the case will be more challenging. The question will be: is the median nerve slowing out of proportion to the slowing associated with the polyneuropathy. It is possible that all the motor and sensory latencies may be prolonged from the polyneuropathy itself. In addition, it would not be uncommon that the sensory and mixed studies may be absent, in which case the palmar mixed, digit 4, and digit 1 comparison studies cannot be used. In this situation, the lumbrical – interosseous comparison is often the most useful internal comparison study, as these motor responses usually remain present in a polyneuropathy.
2. In the unusual situation wherein there is a co-existent ulnar neuropathy at the wrist, all of the median versus ulnar internal comparison studies may be unhelpful, as both the median and ulnar latencies may be prolonged. In this situation, the median versus radial internal comparison study or the median segmental sensory study would be most useful.
3. If there is a co-existent ulnar neuropathy at the elbow (which would not be uncommon), the ulnar mixed and sensory responses may be absent, in which case the palmar mixed and digit 4 studies cannot be used. In this situation, the median versus radial internal comparison study, the median segmental sensory study, or the lumbrical – interosseous comparison would be most useful.
4. If the distal median motor or median sensory amplitudes are low, this may denote either axonal loss or distal conduction block. The only way to differentiate between these two is to stimulate the median nerve in the palm and compare the amplitudes with wrist stimulation. Any palm/wrist ratio >1.6 for sensory and >1.2 for motor amplitudes denotes some conduction block.

preferential slowing of the median nerve compared with the ulnar nerve can be attributed to conduction slowing through the carpal tunnel. The diagnostic yield increases from approximately 75% using routine motor and sensory studies to approximately 95% using these more sensitive techniques.

These sensitive median-versus-ulnar comparison studies are considered abnormal if very small differences between the median and ulnar latencies are found (typically 0.4–0.5 ms). *Therefore, meticulous attention must be paid to all technical factors, especially distance measurement, stimulus artifact, supramaximal stimulation, and electrode placement, to obtain reliable and reproducible data.* Furthermore, it is essential to avoid overstimulation, which can cause unintentional stimulus spread to an adjacent nerve. In the three studies outlined in the following section, overstimulation with unintentional spread of current to the adjacent nerve may yield a waveform that appears perfectly normal yet obscures the true latency difference between the median and ulnar potentials.

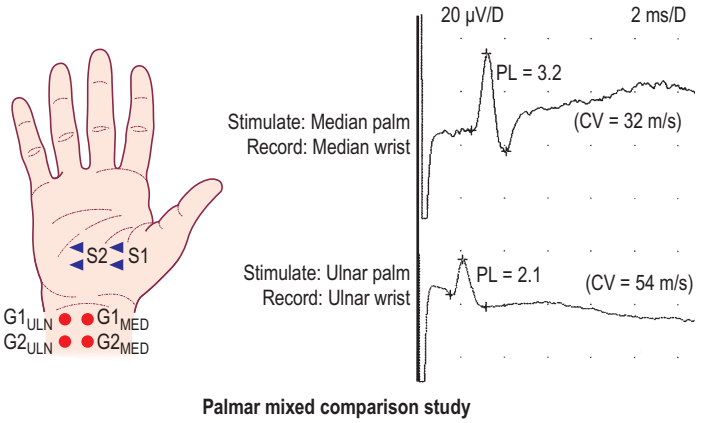

FIGURE 17–8 Palmar mixed comparison study. In this study, the median mixed nerve latency across the palm is compared to the adjacent ulnar mixed nerve latency, using identical distances between stimulation and recording sites. **Left:** G1, active recording electrode; G2, reference recording electrode; S1, median stimulation point; S2, ulnar stimulation point. In normals, there is no significant difference between the two latencies. **Right:** In carpal tunnel syndrome, the median palmar peak latency (PL) is prolonged both in an absolute sense (>2.2 ms) and in comparison to the ulnar palmar peak latency (≥0.4 ms difference).

Median-versus-Ulnar Comparison Studies

Median-versus-Ulnar Palm-to-Wrist Mixed Nerve Studies

This technique takes advantage of measuring the mixed nerve potential. Mixed nerve potentials consist of both motor and sensory fibers. The sensory fibers in the mixed nerve potential carry both cutaneous sensory fibers, which are measured in routine sensory studies, as well as muscle sensory fibers, which are not measured in routine sensory studies. This is important because the muscle sensory fibers include the Ia afferents from muscle spindles, which are the largest and fastest-conducting fibers and hence have the greatest quantity of myelin sheath. These fibers are very susceptible to demyelination, the primary pathology in CTS. The mixed nerve study also takes advantage of conducting over a very short distance of 8 cm. Because such a short distance is used, most of the conduction time is computed over the area of pathology. Only a short length of normal nerve is included that potentially could dilute any slowing present across the carpal tunnel.

The technique is performed by stimulating the median nerve in the palm, recording the median nerve at the wrist, and comparing it with the ulnar nerve stimulated in the palm and recorded over the ulnar nerve at the wrist (Figure 17–8). Each nerve is stimulated supramaximally in the palm at a distance of 8 cm from its respective recording electrodes. The median nerve is stimulated in the palm on a line connecting the median nerve in the middle of the wrist to the web space between the index and middle fingers. The ulnar nerve is stimulated in the palm on a line connecting the ulnar nerve at the medial wrist (lateral to the flexor carpi ulnaris tendon) to the web space between the ring and little fingers. Supramaximal responses are obtained for each nerve, and the difference between the onset or peak latencies is calculated.

Median-versus-Ulnar Digit 4 Sensory Latencies

The technique of comparing median-versus-ulnar digit 4 sensory latencies takes advantage of the fact that, in most individuals, the sensory innervation to the fourth digit (ring finger) is split, with the lateral half innervated by the median nerve and medial half innervated by the ulnar nerve (Figure 17–9). Thus, if identical distances are used, the latencies stimulating each nerve can be directly compared. The antidromic technique is performed by stimulating the median and ulnar nerves at the wrist, one at a time, with recording ring electrodes placed over digit 4 (G1 over the metacarpophalangeal joint and G2 over the distal interphalangeal joint). Identical distances must be used for both (range 11–13 cm). Supramaximal responses are obtained and the difference between median and ulnar onset or peak latencies recorded. The study also can be done orthodromically, stimulating with the ring electrodes over digit 4 as just described and recording the median and ulnar nerves at the wrist at identical distances. We do not recommend the latter method because with orthodromic stimulation at digit 4, co-stimulation of the median and ulnar nerves cannot be avoided, and spread of the potential from the adjacent nerve may contaminate the recorded SNAP at the wrist.

Median Second Lumbrical-versus-Ulnar Interossei Distal Motor Latencies

The technique of comparing the second lumbrical (2L)-versus-interosseous (INT) distal motor latencies takes advantage of two facts: (1) motor fibers are easy to record and more resistant to compression than sensory fibers, and (2) the median 2L muscle lies just above the ulnar INT. In some cases of generalized polyneuropathy with superimposed CTS, the SNAPs and mixed nerve potentials may be absent. In severe cases, the routine median CMAP recording the APB may also be absent, whereas the motor fibers to the second lumbrical and ulnar INT are still recordable.

CMAPs from both the median-innervated 2L and the ulnar-innervated INT can easily be recorded by placing an active electrode (G1) slightly lateral and distal to the midpoint of the third metacarpal, with the reference electrode over the proximal interphalangeal joint of the second digit, and stimulating the median and ulnar nerves at the wrist, respectively (Figure 17–10). The motor point to the 2L is

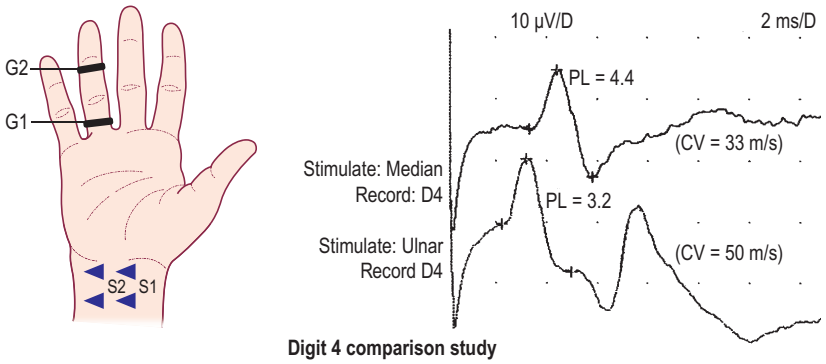

FIGURE 17–9 Digit 4 comparison study. In this study, the median sensory latency recording digit 4 is compared to the ulnar sensory latency recording digit 4, using identical distances between stimulation and recording sites. **Left:** G1, active recording electrode; G2, reference recording electrode; S1, median stimulation point; S2, ulnar stimulation point. This study takes advantage of the anatomic fact that digit 4 has a split innervation, half median and half ulnar, in most individuals. In normals, there is no significant difference between the two latencies. **Right:** In carpal tunnel syndrome, the median sensory peak latency (PL) is prolonged both in an absolute sense (>3.5 ms) and in comparison to the ulnar sensory peak latency (≥0.5 ms difference).

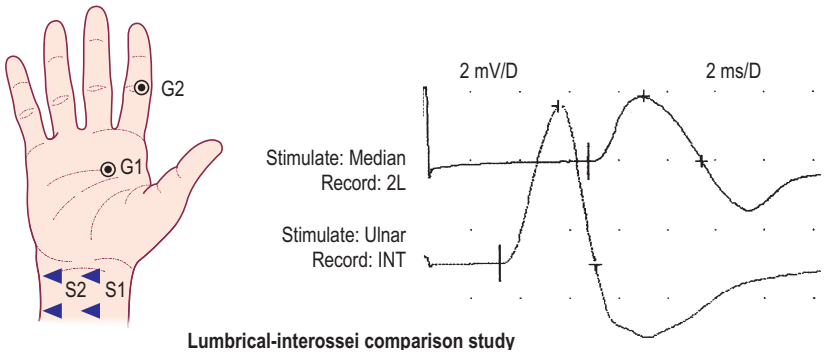

FIGURE 17–10 Lumbrical–interossei comparison study. In this study, the median motor latency recording the second lumbrical is compared to the ulnar motor latency recording the interossei, using identical distances between stimulation and recording sites. **Left:** G1, active recording electrode; G2, reference recording electrode; S1, median stimulation point; S2, ulnar stimulation point. This study takes advantage of the anatomic fact that the second lumbrical (median innervated) lies on top on the first palmar interosseous (ulnar innervated). In normals, there is no significant difference between the two latencies. **Right:** In carpal tunnel syndrome, the median motor latency is prolonged compared to the ulnar motor latency. In this case, the latency difference is about 2 ms. Any difference ≥0.5 ms is considered

identified when the active recording electrode has been placed such that stimulation of the median nerve at the wrist elicits a waveform with the fastest rise time and an initial negative deflection. Because the 2L cannot be seen or palpated, moving the active electrode slightly may be necessary to ensure the electrode is optimally placed. In some individuals, if the sensitivity is increased, a small mixed nerve potential will be seen slightly before the onset of the 2L CMAP. This is a normal finding, especially in younger patients. If this small mixed nerve potential is present, the latency should be measured from the onset of the 2L CMAP, not from the onset of the mixed nerve potential. The ulnar nerve then is stimulated supramaximally at the wrist, at the same distance, *leaving the recording electrodes in place*. A CMAP from the underlying ulnar INT muscles will be easily elicited. The ulnar CMAP is generally larger than the median CMAP. Identical distances (range 8–10 cm) must be used to compare the difference between the distal latencies.

The normal values for the three median-versus-ulnar comparison studies are given in Table 17–3. In our laboratory, the palmar mixed nerve peak latency difference is the most sensitive study, followed closely by the digit 4 sensory and 2L-INT motor studies. However, there is a very high degree of correlation among the results of the three studies. In one comparison study, two of the three studies yielded abnormal results in 97% of all patients with mild CTS. In a patient in whom only one of the median-versus-ulnar comparison studies is abnormal, one should be hesitant to make a definite electrodiagnosis of CTS (see Chapter 9).

Other Useful Studies

Inching across the Wrist and Palmar Stimulation

Another technique useful in demonstrating CTS, first described by Kimura and later by others, involves segmental stimulation ("inching") of the median nerve across the carpal tunnel (Figure 17–11). One looks for an abrupt change in latency or increase in amplitude above normal control values, recording either a median CMAP at the APB or a median digital SNAP at the index or middle finger.

Kimura's method begins at 4 cm proximal to the distal wrist crease and continues to 6 cm distal to the wrist crease, with segmental stimulation at 1 cm increments. For each 1 cm increment, latency usually increases 0.2 to 0.3 ms.

Table 17–3. Median–Ulnar Comparison Studies

Study	Nerve	Stimulate	Record	Distance (cm)	Significant Difference (ms)
Palmar mixed	Median	Median palm	Median nerve at wrist	8	≥0.4
	Ulnar	Ulnar palm	Ulnar nerve at wrist	8	
Digit 4 Sensory	Median	Median nerve at wrist	Digit 4	11–13*	≥0.5
	Ulnar	Ulnar nerve at wrist	Digit 4	11–13	
Lumbrical – interossei	Median	Median nerve at wrist	Lateral to the mid third metacarpal (over the second lumbrical and interossei)	8–10*	≥0.5
	Ulnar	Ulnar nerve at wrist	Lateral to the mid third metacarpal (over the second lumbrical and interossei)	8–10	

*Must use the identical distance for median and ulnar nerve stimulation.

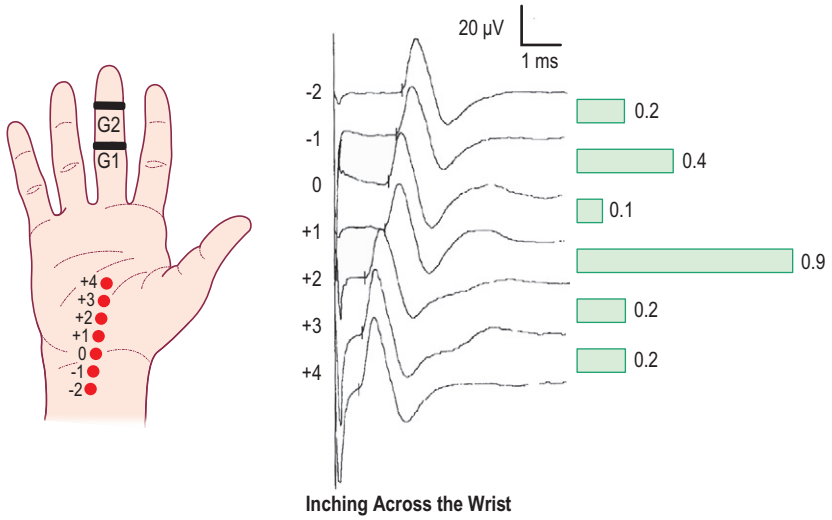

FIGURE 17–11 Inching across the wrist. **Left:** Stimulating the median nerve at 1 cm increments from 2 cm proximal to the wrist crease to 4 cm distal into the wrist crease, recording the digit 3 sensory nerve action potential. **Right:** Actual waveforms and relative change in latency between stimulation points is plotted. Note the abrupt change in latency of the sensory nerve action potential between +1 and +2 cm distal to the wrist crease, signifying the area of focal slowing.

Any abrupt change in latency is highly suggestive of focal demyelination. Although the inching technique has the advantage of showing the exact site of the lesion, its effectiveness often is limited by difficulty stimulating the nerve at the sites just distal to the wrist crease. The technique is particularly difficult to perform recording the median CMAP because stimulation of motor fibers at 1 cm increments following the course of the recurrent thenar branch of the median nerve can be quite difficult. Furthermore, stimulation in the palm often requires rotation of the anode to prevent excessive stimulus artifact (Figure 17–12).

Rather than measuring a change in latency, comparing the CMAP or SNAP *amplitudes* stimulating at the wrist and palm can be technically easier and can yield additional information about the underlying pathophysiology (Figure 17–13). Wrist and palmar stimulation can be performed for either median motor or sensory studies. Only single palm and wrist stimulations are required, whereas inching requires stimulation at multiple 1 cm increments. Several technical factors must be taken into account. First, as noted earlier for motor studies, the anatomy of the recurrent thenar motor branch is such that for stimulating the motor branch in the palm, the stimulator often must be placed beyond the thenar eminence with the anode rotated distally to prevent excessive stimulus artifact (Figure 17–12). Second, the examiner must be aware of normal values when comparing amplitudes proximal and distal to the carpal tunnel. There is always some drop in amplitude proximally compared with distally due to greater temporal dispersion and phase cancellation with proximal stimulation. The effects of normal temporal dispersion and phase cancellation are always greater for sensory fibers than for motor fibers. In normal median nerves, the ratio of the distal to proximal CMAP amplitude does not exceed 1.2, whereas the distal to proximal SNAP amplitude ratio does not exceed 1.6. Larger ratios suggest some element of conduction block (Figure 17–14). This assumption presumes that both stimulations are supramaximal, that there is no co-stimulation of adjacent nerves, and that the baseline is not obscured by shock artifact or noise that precludes an accurate amplitude measurement.

In CTS, if wrist stimulation yields a low CMAP or SNAP amplitude, there are two possible explanations: (1) there is conduction block secondary to demyelination across the

carpal tunnel with the underlying axon intact, or (2) there has been secondary axonal loss (Figure 17–15). Comparison of the amplitudes obtained with wrist and palmar stimulation can easily sort out these two possibilities. Take the following example:

	Case A	Case B
CMAP (stimulate wrist, record APB)	2 mV	2 mV
CMAP (stimulate palm, record APB)	6 mV	2 mV

In both cases, when the median nerve is stimulated at the wrist, the recorded CMAP is low (normal value >4.0 mV). When the palm is stimulated in case A, however, the CMAP amplitude increases by 200%; the distal to proximal ratio is 3.0, signifying conduction block. In contrast, there is no change in amplitude in case B, signifying that the low amplitude is secondary to axonal loss.

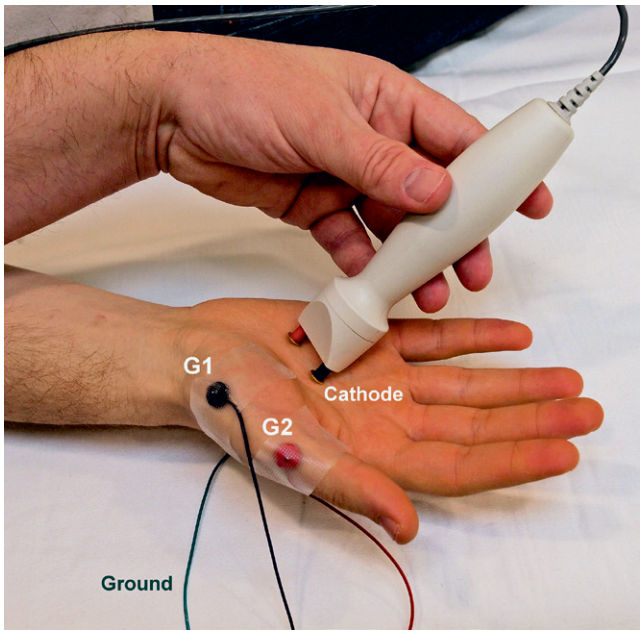

FIGURE 17–12 Stimulating the recurrent thenar motor branch in the palm. Stimulating the median nerve in the palm often is technically difficult. In many instances, the anode of the stimulator must be rotated to reduce stimulus artifact.

Median-versus-Radial Digit 1 Sensory Latencies

Comparison of the median-versus-radial digit 1 sensory latencies takes advantage of the fact that, in most individuals, digit 1 (the thumb) is innervated by both the median and radial nerves (Figure 17–16). The basic concept is the same as in the median-versus-ulnar digit 4 sensory study: the median and radial nerves are stimulated at the wrist, using identical distances, with recording ring electrodes over digit 1 (G1 over the metacarpophalangeal joint and G2 over the interphalangeal joint). The radial nerve is stimulated at the wrist along the lateral border of the radial bone. Using the same distance, the median nerve is stimulated at the wrist in the usual location. Supramaximal responses are obtained at each stimulation site, and the onset or peak latencies are compared. Although this technique is popular in some laboratories, stimulating the nerves at identical distances may be difficult because the median nerve travels to the thumb at an angle, which can hinder measurement of its true distance. Any difference between the median and radial latencies greater than or equal to 0.5 ms is considered abnormal.

Wrist-to-Palm versus Palm-to-Digit Sensory Conduction Velocity (Segmental Sensory Conduction Studies across the Wrist)

This technique compares the sensory conduction velocity along the median nerve at two segments of identical distance: the wrist-to-palm segment and the palm-to-digit segment. Digit 3 is the preferred finger to record from due to its longer length. The recording electrodes (G1, G2) are placed at the proximal and the distal interphalangeal joints, respectively. The median nerve is then stimulated at the wrist at a fixed distance to G1. The median nerve then is stimulated at the palm, with the recording ring electrodes left in place, at half the wrist-to-digit distance (Figure 17–17). Although any distances could be used for this study, making the palm-to-digit distance half that of the wrist-to-digit distance greatly simplifies the mathematical

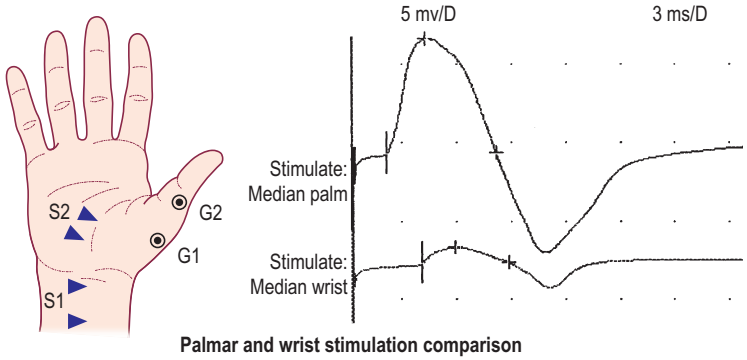

FIGURE 17–13 Palmar and wrist stimulation comparison. The median nerve is stimulated at the wrist and palm while recording the abductor pollicis brevis muscle. **Left:** G1, active recording electrode; G2, reference recording electrode; S1, stimulation at the wrist; S2, stimulation in the palm. **Right:** A significantly larger amplitude stimulating in the palm compared to the wrist signifies conduction block (i.e., demyelination) across the wrist.

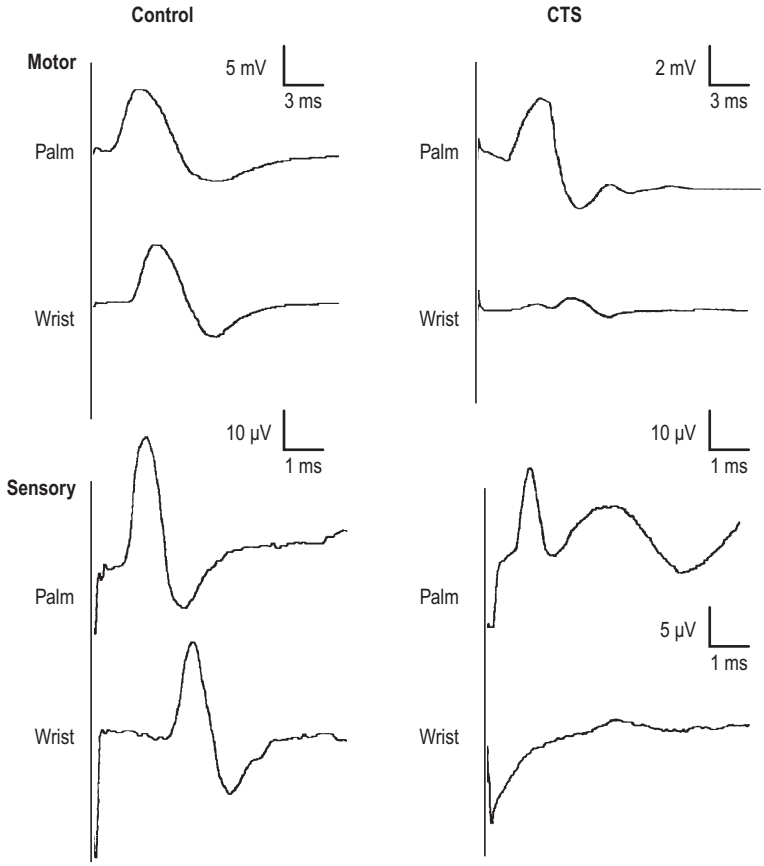

FIGURE 17–14 Change in compound muscle action potential (CMAP) and sensory nerve action potential (SNAP) amplitude across the carpal tunnel. To assess possible conduction block across the carpal tunnel, either the median CMAP or SNAP can be recorded with stimulation of the wrist and palm. Note that in normal controls, there is only a slight increase in amplitude between wrist and palm stimulation sites. A large difference in amplitude between wrist and palm sites in patients with carpal tunnel syndrome signifies conduction block. For motor studies, a normal palm to wrist amplitude ratio is ≤1.2 and for sensory studies it is ≤1.6.
(Adapted with permission from Lesser, E.A., Venkatesh, S., Preston, D.C., et al., 1995. Stimulation distal to the lesion in patients with carpal tunnel syndrome. Muscle Nerve 18, 503.)

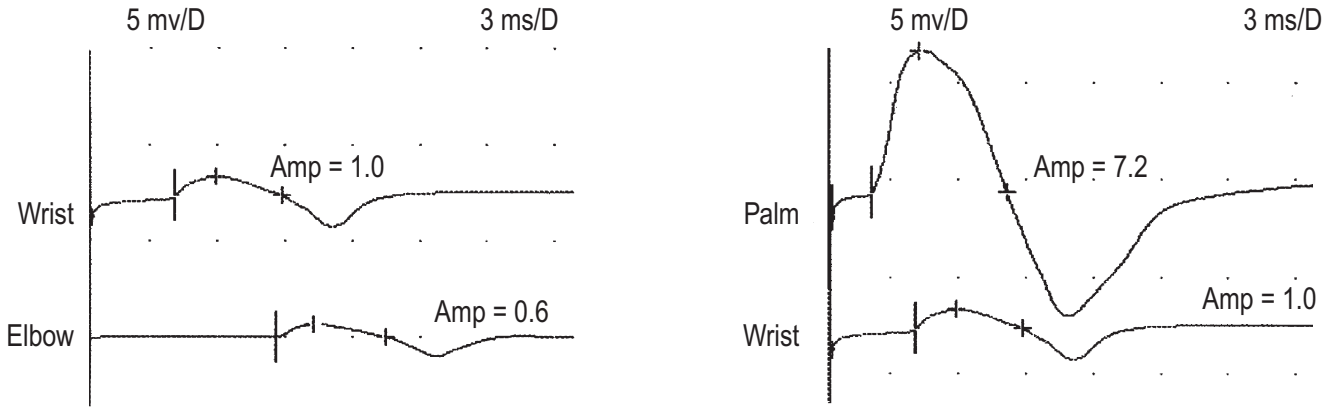

FIGURE 17–15 Distal conduction block mimicking axonal loss. Low distal amplitudes usually are attributed to axonal loss. However, if conduction block is present distal to the typical distal stimulation site, it can mimic the pattern of axonal loss. Such is often the case in carpal tunnel syndrome (CTS), where the lesion is distal to the usual distal stimulation site. **Left:** Median motor study, stimulating the wrist and antecubital fossa. Note that this appears to be a typical axonal loss pattern. **Right:** Median motor study, stimulating the palm and wrist. In this patient with CTS, a markedly higher-amplitude CMAP is evoked stimulating the palm, signifying conduction block. The identification of conduction block not only localizes the lesion, but it also denotes a much better prognosis than axonal loss. The clinical clue to the presence of conduction block in a patient with CTS is a weak thumb abduction and relatively intact muscle bulk (i.e., no atrophy) of the abductor pollicis brevis muscle, with a low median CMAP stimulating at the wrist.

equation. The wrist-to-palm conduction velocity is then computed by multiplying the palm-to-digit conduction velocity by the wrist-to-digit conduction velocity, and then dividing it by the quantity of the palm-to-digit conduction velocity times two minus the wrist-to-digit conduction velocity (Figure 17–18). In normal subjects, the wrist-to-palm segment (i.e., the segment across the carpal tunnel) is equal to or faster than the distal segment (palm-to-digit) because proximal nerve normally conducts faster than distal segments, secondary to larger nerve diameter and warmer temperatures. In CTS, there is a reversal of this normal pattern; the proximal segment (wrist-to-palm)

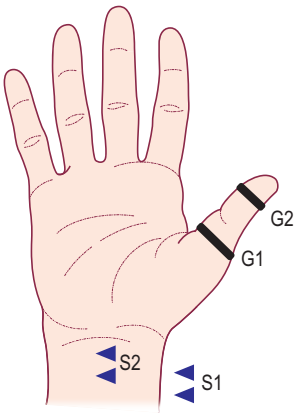

FIGURE 17–16 Median-radial sensory comparison study. In most individuals, the thumb is innervated by both the superficial radial and median sensory nerves. Using identical distances, the median and radial sensory latencies to the thumb can be compared in patients with suspected carpal tunnel syndrome, looking for preferential slowing of the median sensory fibers. G1, active recording electrode; G2, reference recording electrode; S1, radial stimulation point; S2, median stimulation point. Any difference between the median and radial latencies ≥0.5 ms is considered abnormal.

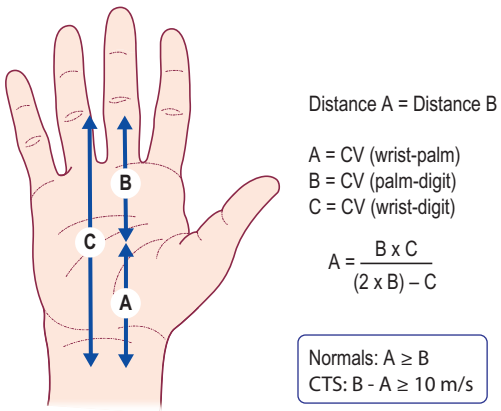

FIGURE 17–18 Calculation of the wrist-to-palm velocity in segmental sensory studies. There is no direct way to stimulate the median cutaneous sensory fibers at the wrist and record them at the palm. The wrist-to-palm conduction velocity (CV) can be calculated from knowledge of the wrist-to-digit and palm-to-digit CVs, both of which can be directly measured. If the palm-to-digit distance is half the wrist-to-digit distance, the calculation is simplified. In normal nerves, one expects the proximal segments to conduct at the same velocity or faster than the distal segments, due to larger nerve diameters and warmer temperatures (see Chapter 8). In carpal tunnel syndrome, there is a reversal of this pattern: the wrist-to-palm CV (across the carpal tunnel) is slower than the palm-to-digit CV. Any slowing ≥10 m/s is considered abnormal.

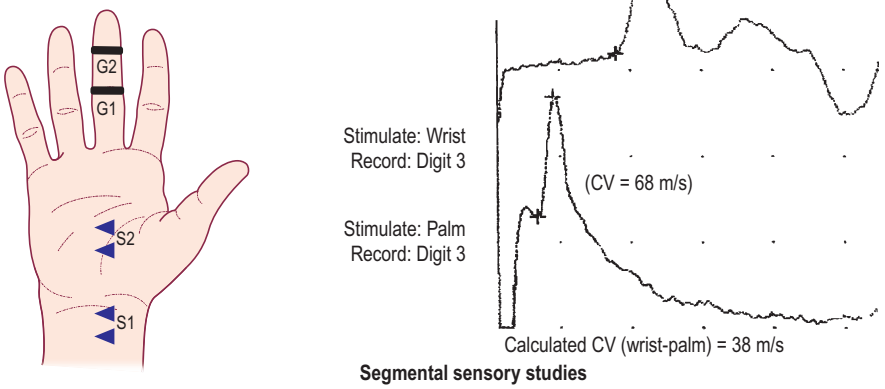

FIGURE 17–17 Segmental sensory conduction studies across the wrist. Using this technique, sensory conduction velocities (CVs) can be obtained for the wrist-to-digit and palm-to-digit segments, and then the wrist-to-palm CV can be calculated (see Figure 17–18). **Left:** The median nerve is stimulated at the wrist at a fixed distance and at the palm at half that distance, recording the median sensory nerve action potential with ring electrodes over digit 3. G1, active recording electrode; G2, reference recording electrode; S1, median stimulation point at the wrist; S2, median stimulation point in the palm. Placing the recording electrodes more distally on the finger helps reduce stimulus artifact when stimulating in the palm. **Right:** In patients with carpal tunnel syndrome, the calculated CV from wrist to palm (38 m/s) is slower than the CV from palm to digit (68 m/s).

conducts more slowly than the distal palm-to-digit segment. In general, any slowing of more than 10 m/s is considered abnormal.

Median-versus-Ulnar Minimum F Wave Latencies

This technique compares the minimum F wave latency stimulating the median and ulnar nerves at the wrist, recording the APB and abductor digiti minimi muscles, respectively. In normal individuals, the minimum F wave latency from the median nerve is approximately 1 to 2 ms shorter than the minimum F wave latency from the ulnar nerve. A reversal of this pattern is considered abnormal (Figure 17–19). This test is nonspecific, however, because the F wave measures conduction along the entire length of nerve, from the recording electrode to the spinal cord. Although this study can confirm a problem with the median nerve, it cannot localize the lesion to the wrist. It is generally used only as confirmatory evidence for a diagnosis of CTS, in conjunction with abnormalities noted using more sensitive techniques.

Electromyographic Approach

The recommended EMG approach to a patient with CTS is outlined in Box 17–3. The EMG strategy is designed with the clinical differential diagnosis in mind (i.e., proximal median neuropathy, brachial plexopathy, C6–C7 radiculopathy). The key muscle to check is the APB. In mild or early cases of CTS, the APB often is normal. In later or more severe cases, EMG may reveal secondary axonal loss resulting in denervation and reinnervation. In general, the hand muscles are best approached with a smaller-gauge needle. Because examination of the APB often is painful for patients to tolerate, it is best to begin the study with a different C8–T1 innervated muscle, such as the first dorsal interosseous (FDI). The APB can be examined next. Although some electromyographers may prefer to study the APB toward the end of the examination, there is the potential problem that the patient may quit the study before this key muscle can be studied, especially if the patient is generally intolerant of the EMG examination.

If the APB is abnormal, proximal median muscles and at least two other non-median C8–T1/lower trunk-innervated muscles should be sampled. In addition, C6–C7-innervated muscles should be sampled to exclude a cervical radiculopathy. The PT and FCR are very helpful muscles to sample because they can be used both as proximal median and C6–C7-innervated muscles. Some electromyographers have difficulty with the notion that the C6–C7-innervated muscles are important to sample, because the distal median hand muscles are innervated by the C8–T1 roots. One must remember that the distribution of numbness (not the weakness) in CTS may be very similar to the numbness noted in C6–C7 radiculopathies. Of course, because each case is different, the electromyographer must always be willing to modify each study throughout the testing, based on abnormalities noted as the study progresses.

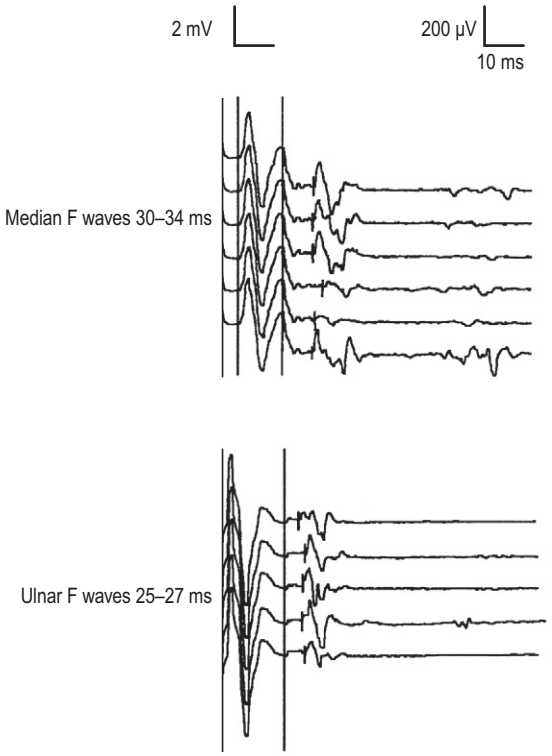

FIGURE 17–19 Inverted F waves in carpal tunnel syndrome. In normal subjects, the minimum F wave latency of the median nerve is approximately 1 to 2 ms shorter than that of the ulnar nerve. In carpal tunnel syndrome, the median F waves often are prolonged compared with the ulnar F waves, providing a useful measure to confirm median neuropathy.

Box 17–3. Recommended Electromyographic Protocol for Carpal Tunnel Syndrome

1. Abductor pollicis brevis (APB)
2. At least two C6–C7-innervated muscles (e.g., pronator teres, flexor carpi radialis, triceps brachii, extensor digitorum communis) to exclude a cervical radiculopathy

If APB is abnormal, the following additional muscles should be sampled:

1. At least one proximal median-innervated muscle (e.g., flexor carpi radialis, pronator teres, flexor pollicis longus) to exclude a proximal median neuropathy (note: the pronator teres may be spared in pronator syndrome)
2. At least two other non-median, lower trunk/C8–T1-innervated muscles (e.g., first dorsal interosseous, extensor indicis proprius) to exclude a lower trunk brachial plexopathy, polyneuropathy, or C8–T1 radiculopathy

Note: If the carpal tunnel syndrome is superimposed on another condition (e.g., polyneuropathy, plexopathy, radiculopathy), a more detailed electromyographic examination will be required.

The APB study frequently is painful and difficult for some patients to tolerate. It is best not studied first, but also best not left for the end of the electromyographic study in case the patient is unable to tolerate the entire examination.

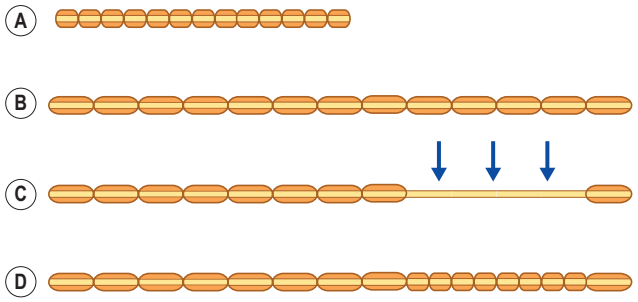

FIGURE 17–20 Persistent "slowing" following demyelination and remyelination. **A:** The process of myelination occurs at approximately age three. **B:** Between childhood and adulthood, the limb grows in length; however, the number of internodes does not change. **C:** Demyelination occurs at the site of compression (blue arrows). **D:** After the compression is successfully released, remyelination occurs. However, the new internodes are short, the same distance apart they were originally laid down as a child. Therefore more nodes are required to remyelinate the original site of compression. The greater the number of nodes of Ranvier, the more depolarizations, and hence the longer total time of depolarization. Thus, conduction velocity across the remyelinated area of compression will be slower than normal, because of the increase in number of nodes.

Special Situation: EDX Studies after Carpal Tunnel Release

It is not uncommon for a patient who has previously undergone carpal tunnel release surgery to be referred for EDX studies. The patient will either have recently undergone surgery with no clinical improvement, or will have developed recurrent symptoms a long period of time after successful carpal tunnel decompression. In some cases, the patient will not have had a pre-operative EDX study to confirm the diagnosis of CTS, which further complicates the issue. Thus, every electromyographer should be aware of what happens to nerve conduction study abnormalities after successful carpal tunnel release surgery. In general, the distal latencies and amplitudes improve both for median motor and sensory studies. However, this may take many weeks to months, and in some studies, improvement continues up to a year after surgery. However, some slowing may persist indefinitely. In the authors' experience:

1. Median distal motor latencies improve and usually return to the "normal" range. Never do distal latencies remain in the demyelinating range (i.e., >130% the upper limit of normal) after successful carpal tunnel release.
2. Median sensory latencies improve and usually return to the "normal" range. Never do conduction velocities remain in the demyelinating range (i.e., <75% the lower limit of normal) after successful carpal tunnel release.
3. Median motor amplitudes improve and return to the normal range.
4. Median sensory amplitudes may or may not improve. Many remain in a slightly reduced or borderline normal range.
5. The sensitive internal comparison studies (i.e., palmar mixed studies, digit 4 study, digit 1 study, lumbrical–interosseous study, and segmental sensory study) remain abnormal indefinitely, showing some slowing of median conduction across the carpal tunnel.

Although these findings are seen most often after carpal tunnel release surgery, similar findings are seen for other entrapments. This begs the question: after successful release surgery, why do the median conductions not return completely back to normal? The answer involves knowledge of normal myelination, demyelination, and then remyelination (Figure 17–20). As noted in Chapter 2, the process of myelination begins in utero, and full myelination of peripheral nerves does not occur until approximately age 3. Thus, by age 3, all the myelin and all the internodes have been laid down (Figure 17–20A). However, between childhood and adulthood, while the limb grows in length, resulting in longer internodes, the number of internodes does not change (Figure 17–20B). In entrapment neuropathies, such as carpal tunnel syndrome, demyelination occurs at the site of compression, resulting in interruption of the internodes at the site of compression (Figure 17–20C). When the compression is successfully released, remyelination can then occur. However, the new internodes are short, the same distance apart that they were when originally laid down as a child (Figure 17–20D). Therefore more nodes are required to remyelinate the original site of compression. When remyelination is completed, nerve impulses can once again travel successfully up and down the nerve. However, remember that the time of conduction (and hence conduction velocity) is completely dependent on the depolarization time at the nodes of Ranvier. The greater the number of nodes of Ranvier, the more depolarizations, and hence the longer total time of depolarization. Thus, conduction velocity across the remyelinated area of compression will be slower than normal, because of the increase in number of nodes. In any situation where there has been demyelination and then remyelination, sensitive techniques will always demonstrate a slightly slower conduction time across the remyelinated segment. Accordingly, one must always be cautious when interpreting any mild "slowing" on nerve conduction studies in patients who have undergone carpal tunnel release.

EXAMPLE CASES

Case 17–1

History and Examination

A 67-year-old woman was referred for clumsiness, tingling, and pain in both hands of several months' duration. Symptoms were most prominent at night, often awakening her from sleep, or during hand use such as driving. Examination showed slight wasting of both thenar eminences. Reflexes were normal. Thumb abduction was weak bilaterally. Sensation was slightly reduced over the finger pads of the thumb, index, middle, and ring fingers. There was no Tinel's sign at the wrist on either side. A Phalen's maneuver elicited tingling in the middle finger bilaterally after 30 seconds.

Summary

The history of pain and paresthesias in both hands, which was worse at night and provoked by driving, is characteristic of CTS. In addition, the examination suggests median neuropathy. Weak thumb abduction suggests dysfunction of the APB, a distally innervated median muscle. Sensation is reduced over the median-innervated digits. Although a Tinel's sign is not present at the wrist, a Phalen's maneuver causes paresthesias in the third digit. A Phalen's maneuver is thought to reproduce the situation that occurs at night when the patient is asleep and the wrist commonly assumes a flexed posture. Note that nothing in the physical examination or history suggests a radiculopathy (i.e., there is no neck pain or weakness in the C6 or C7 muscles, and the reflexes are normal). One would assume that there is a high likelihood of bilateral CTS in this patient even before proceeding to the nerve conduction and EMG studies.

Both the nerve conduction studies and EMG findings are abnormal. The median motor study on the right shows a low CMAP amplitude with a markedly prolonged distal motor latency, moderately slow conduction velocity in the forearm, and absent F responses. The left median nerve also is abnormal but not as severely as the right, with a normal CMAP amplitude, moderately prolonged distal motor latency, borderline slow conduction velocity in the forearm, and prolonged F responses. The ulnar motor study is completely normal, an important finding that indicates that the median motor abnormalities are not secondary to a more widespread polyneuropathy. The sensory studies demonstrate a similar pattern of abnormalities. The median sensory response to digit 2 is absent on the right but present on the left, with a low-amplitude, prolonged peak latency, and a correspondingly markedly slow conduction velocity. The right ulnar sensory response is completely normal. Because the median mixed potential is absent on the right, the ulnar mixed nerve study is not performed on that side; there would be nothing to compare it with. The median mixed nerve study on the left demonstrates a markedly prolonged peak latency. Furthermore, not only is the median mixed latency on the left markedly prolonged (3.8 ms) in an absolute sense, but it is clearly prolonged out of proportion to the ulnar mixed peak latency (1.7 ms), which is normal.

After completion of the nerve conduction studies, one can be fairly certain of the diagnosis of bilateral median neuropathy at the wrist, affecting both motor and sensory fibers. The localization of the lesion at the wrist, rather than more proximally, is determined by the markedly prolonged latencies with wrist stimulation. These markedly prolonged latencies signify demyelination across the wrist. There is no suggestion of a superimposed

CASE 17–1. Nerve Conduction Studies

Nerve Stimulated	Stimulation Site	Recording Site	Amplitude Motor = mV; Sensory = µV			Latency (ms)			Conduction Velocity (m/s)			F-Wave Latency (ms)		
			RT	LT	NL	RT	LT	NL	RT	LT	NL	RT	LT	NL
Median (m)	Wrist	APB	3.4	8.6	≥4	10.9	6.4	≤4.4				NR	33	≤31
	Antecubital fossa	APB	3.0	8.4		15.8	10.6		41	49	≥49			
Ulnar (m)	Wrist	ADM	11.2		≥6	3.0		≤3.3				25		≤32
	Below elbow	ADM	11.2			6.3			60		≥49			
	Above elbow	ADM	11.1			8.0			61		≥49			
Median (s)	Wrist	Index finger	NR	8	≥20	NR	4.9	≤3.5	NR	32	≥50			
Ulnar (s)	Wrist	Little finger	24		≥17	2.9		≤3.1	62		≥50			
Median (mixed study)	Palm	Wrist	NR	8	≥50	NR	3.8	≤2.2	NR	27	≥50			
Ulnar (mixed study)	Palm	Wrist		16	≥15		1.7	≤2.2		61	≥50			
Mixed difference							1.1	≤0.3						

m = motor study; s = sensory study; RT = right; LT = left; NL = normal; NR = no response; APB = abductor pollicis brevis; ADM = abductor digiti minimi.
Note: All sensory and mixed latencies are peak latencies. All sensory and mixed-nerve conduction velocities are calculated using onset latencies. The reported F-wave latency represents the minimum F-wave latency.

CASE 17–1. Electromyography

Muscle	Insertional Activity	Spontaneous Activity		Voluntary Motor Unit Action Potentials				
		Fibrillation Potentials	Fasciculation Potentials	Activation	Recruitment	Configuration		
						Duration	Amplitude	Polyphasia
Right APB	↑	+1	0	NL	↓↓	+2	+2	+2
Right FDI	NL	0	0	NL	NL	NL	NL	NL
Right PT	NL	0	0	NL	NL	NL	NL	NL
Right triceps brachii	NL	0	0	NL	NL	NL	NL	NL
Right FCR	NL	0	0	NL	NL	NL	NL	NL
Right C8 paraspinal muscles	NL	0	0	NL	NL	NL	NL	NL
Left APB	NL	0	0	NL	NL	NL	NL	NL

NL = normal; ↑ = increased; ↓↓ = moderately reduced; APB = abductor pollicis brevis; FDI = first dorsal interosseous; PT = pronator teres; FCR = flexor carpi radialis.

polyneuropathy because the ulnar motor, sensory, and F wave studies are completely normal.

The EMG study shows increased insertional activity and fibrillation potentials in the right APB, with decreased recruitment of long, large, polyphasic motor unit action potentials. Because the APB is abnormal on the right, the FDI and C8 paraspinal muscles are sampled to rule out the possibility of a coexistent C8–T1 radiculopathy. Note that if the clinical examination or history strongly suggests the possibility of a superimposed C8–T1 radiculopathy (e.g., weakness of other intrinsic hand muscles or pain radiating from the neck to the medial forearm), further sampling of other C8–T1-innervated muscles should be done. In addition, because the APB is abnormal, proximal median muscles (PT, FCR) must be sampled to ensure that the abnormalities seen in the APB are not secondary to a high median neuropathy. Sampling the PT muscle alone may not be sufficient because that muscle may be spared in the pronator syndrome, wherein compression of the median nerve occurs after the takeoff to the branch to the PT (see Chapter 18). Had there been a high clinical suspicion of a proximal median neuropathy, additional median-innervated proximal muscles should have been sampled.

Sampling both the PT and the FCR serves a dual purpose in that they are both proximal median and C6–C7-innervated muscles. The fact that they are normal makes the diagnosis of a superimposed C6–C7 radiculopathy, or brachial plexopathy, unlikely. The triceps brachii often is useful in this situation as well because it is very strongly innervated by C7 and typically is abnormal in C7 radiculopathy. Again, if the clinical examination or history suggests a superimposed C6–C7 radiculopathy (e.g., weakness of elbow or wrist extension, absent biceps or triceps reflex), more extensive sampling of muscles in those myotomes would have been warranted. Finally, because the symptoms are bilateral and the nerve conduction studies are abnormal bilaterally, the left APB is sampled to assess the severity of the median nerve lesion on that side. Because the APB is normal on the left and there is no clinical suspicion of a superimposed proximal median neuropathy, plexopathy, or radiculopathy, no further needle examination is needed on that side. At this point, an electrodiagnostic impression can be formed.

***IMPRESSION**: There is electrophysiologic evidence of bilateral, moderately severe (right more severe than left) median neuropathies at the wrist.*

Several questions deserve consideration.

Does the Clinical – Electromyographic Correlation Make Sense?

The answer in this case clearly is yes. The patient's history and physical examination are highly suggestive of CTS. There is nothing to suggest a superimposed radiculopathy, plexopathy, or polyneuropathy. Nerve conduction studies and EMG both confirm the clinical impression. All the electrodiagnostic abnormalities are limited to the median nerve. In addition, the markedly prolonged distal motor and sensory latencies are consistent with demyelination of the median nerve across the carpal tunnel. All findings are more severe on the right than on the left. This is the common situation in idiopathic CTS; the dominant hand is most affected. Any clearcut case of CTS in which the non-dominant hand is more severely affected should raise a red flag that there may be an unusual etiology, such as a mass lesion. In such a situation, one must go back to the clinical history and examination to look for unusual features (e.g., a palpable mass on examination). In some individuals, imaging studies of the wrist should be considered.

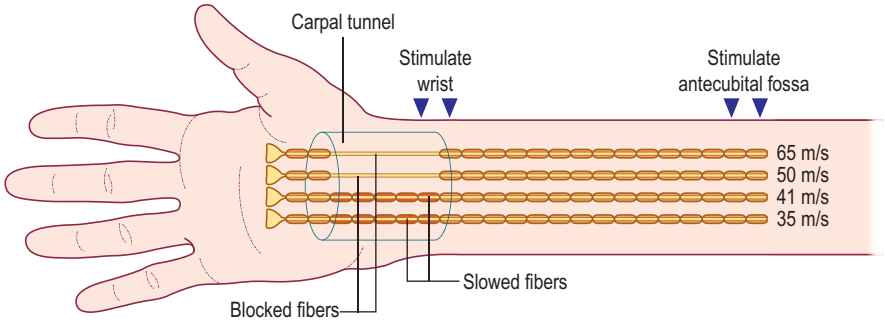

FIGURE 17–21 Slowing of forearm conduction velocity in carpal tunnel syndrome. The normal median nerve has fast-, medium-, and slow-conducting myelinated fibers. Normally, the distal latency and conduction velocity represent only the fastest fibers. In severe carpal tunnel syndrome, if the fastest fibers are blocked at the wrist or have undergone wallerian degeneration, they cannot be measured recording the median compound muscle action potential in the hand; only the normal, slower-conducting myelinated fibers can be measured, producing a spuriously low forearm conduction velocity.

Is the Lesion Demyelinating or Axonal?

In this case, there are both demyelinating and axonal features. Both distal motor latencies are markedly prolonged. The right distal motor latency (10.9 ms) is approximately 250% the upper limit of normal, and the left (6.4 ms) is approximately 145%. Any distal latency greater than approximately 130% the upper limit of normal cannot be attributed to axonal loss or dropout of the fastest fibers alone. These markedly prolonged distal latencies signify demyelination between the recording and stimulating sites (i.e., between the wrist and the APB muscle). Second, although the median sensory response is absent on the right, it is present on the left, with a demyelinating conduction velocity. The velocity of 32 m/s is less than 75% the lower limit of normal, which cannot be explained by the dropout of the fastest-conducting fibers. The lesion must be demyelinating. However, there also are axonal changes. Note that the CMAP amplitude on the right is slightly low (3.4 mV); this may be the result of either distal conduction block or axonal loss. On EMG, there are fibrillation potentials in the right APB along with long-duration, large-amplitude, polyphasic motor unit action potentials. These are EMG signs of denervation and reinnervation that signify active and chronic axonal loss. Therefore, one can say with confidence that on the right side the lesion is both demyelinating and axonal. On the left side, the EMG is normal. Thus, there is no definite evidence of axonal loss by EMG on that side.

The EMG abnormalities of denervation and reinnervation signify a more severe lesion. There is ongoing axonal loss occurring on the right side. Simple conservative treatment measures, such as a neutral wrist splint or steroid injection, likely would not be successful on the right side. This patient likely requires surgical decompression.

If the Lesion is at the Carpal Tunnel, Why is the Forearm Median Motor Conduction Velocity Slow?

The right median motor conduction velocity is slowed in the forearm segment (41 m/s). Because this value represents the speed of the median motor fibers in the forearm between the elbow and the wrist (i.e., proximal to the carpal tunnel), one might consider the possibility of an additional median nerve problem in the forearm segment. However, the finding of a slowed conduction velocity in the forearm segment is quite common in CTS, especially in severe cases. It may occur for two reasons. First, in cases of severe CTS with secondary axonal loss and wallerian degeneration, the wallerian degeneration may proceed proximally. If some of the fastest fibers are lost, then these fibers no longer contribute to the calculated conduction velocity. Second, forearm slowing may occur simply as a byproduct of the method by which the motor conduction velocity is computed (Figure 17–21). In severe CTS, demyelination may result in conduction block of the fastest and largest fibers, the fibers most prone to compression. Even though these fibers are present and their underlying axons intact, demyelination at the carpal tunnel may result in complete block. Because the blocked fibers cannot carry their impulses distally, they do not contribute to the median CMAP. As a result, the conduction velocity of these blocked fibers is not included in the calculated conduction velocity. The calculated conduction velocity will be slowed, based on the speed of the fastest of the remaining normal slower-conducting fibers. In theory, if the median motor fibers in the forearm could be selectively stimulated at the antecubital fossa and recorded at the wrist, before the conduction block at the carpal tunnel, the conduction velocity would be normal. Therefore, a slowed forearm median motor conduction velocity in a patient with severe CTS is not unusual and does not imply an additional proximal lesion.

If the Lesion is at the Carpal Tunnel, Why are the F Responses Absent or Prolonged?

In this case, both median F responses are abnormal (absent on the right, prolonged on the left), especially compared to the ulnar F responses, which typically are 1 to 2 ms longer than the median. One usually thinks of the F responses as checking the proximal nerve

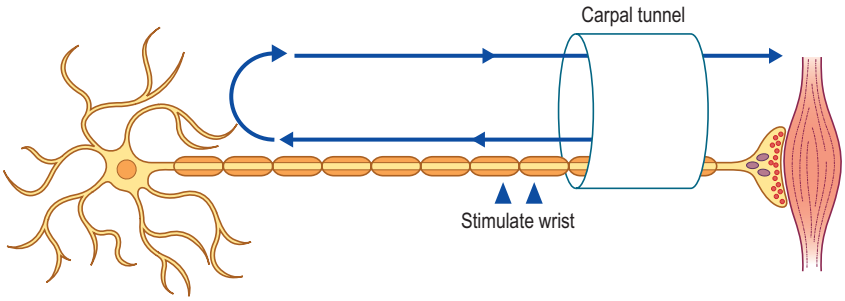

FIGURE 17–22 F wave slowing in carpal tunnel syndrome. The F wave travels antidromically to the anterior horn cell, back down to the point of stimulation, then distally through the carpal tunnel to the muscle. In the carpal tunnel, the F wave may be slowed or blocked. Prolonged or absent F waves are not unusual in carpal tunnel syndrome.

segments, and of prolonged or absent F responses as indicative of a proximal lesion. However, the F response travels the entire course of the axon. When the F response study is performed, the impulse follows a course initially up the nerve antidromically to the anterior horn cell, followed by retrograde travel down the motor nerve to the point of stimulation, and then past the point of stimulation to the distal nerve segment, across the neuromuscular junction, and into the muscle (Figure 17–22). The F response is actually a small motor response, representing approximately 5% of the motor fibers. Therefore, conduction slowing anywhere along the length of the F response circuitry will result in prolonged or absent F responses. In CTS, when the median F response is elicited stimulating at the wrist, the impulse travels antidromically up to the spinal cord and back down to the wrist and then through the carpal tunnel to the muscle, where it slows or is blocked. Prolonged or absent F responses are not unusual and should be expected in severe CTS.

Case 17–2

History and Examination

A 44-year-old woman who was diagnosed with rheumatoid arthritis 6 months previously was referred for a second opinion concerning right hand and wrist pain, paresthesias, and an abnormal cervical magnetic resonance imaging (MRI) scan. The symptoms had developed over the preceding 2 months and were associated with diffuse aching of the right arm. The patient stated that she would awaken from sleep one or two times nightly with pain and tingling in the hand. She would arise from bed and shake her right hand for several minutes or put it under running water. During the day, driving or holding a book, newspaper, or telephone would particularly exacerbate the symptoms. The symptoms slowly worsened over 2 months until nearly all activities caused pain, paresthesias, and considerable distress.

The patient initially had been referred to an outside hospital for an EMG and nerve conduction study, with a question of CTS. Bilateral median and ulnar motor, sensory, and F wave studies were normal. Needle EMG of both APB muscles was normal. The impression was that the study was normal, with no evidence of CTS.

In light of the continued symptoms and the normal nerve conduction and EMG studies, cervical radiculopathy was considered as an alternative diagnosis. A cervical MRI scan was reported to demonstrate an increased T2 signal in the center of the cervical spinal cord, consistent with a syrinx. The patient was referred for further evaluation and management of her syrinx and upper extremity symptoms.

On examination, mental state and cranial nerves were normal. Motor examination revealed normal bulk and strength testing throughout. Reflexes were normal and symmetric. Sensory examination demonstrated a patchy area of decreased light touch sensation over the finger pads of the index and middle fingers of the right hand. There was no Tinel's sign at the wrist. Phalen's maneuver after 60 seconds of wrist flexion caused paresthesias in the finger pads of the right middle finger.

Summary

In many ways, the clinical history in Case 17–2 is similar to that in Case 17–1. The history of pain and paresthesias, which awakened the patient from sleep and were exacerbated by driving or holding a book, is very characteristic of CTS. In addition, the patient has a history of rheumatoid arthritis, a condition commonly associated with CTS. Rheumatoid arthritis is associated with several other peripheral nerve disorders as well, including a distal symmetric sensorimotor polyneuropathy, a vasculitic neuropathy resulting in mononeuritis multiplex, and radiculopathies. In this case, there are no symptoms or signs suggesting any of those diagnoses.

The examination also suggests the possibility of CTS. There is a patchy decrease of light-touch sensation in the index and middle fingers (median-innervated digits). Although there is no Tinel's sign at the wrist, a Phalen's maneuver, which is more sensitive and specific for median neuropathy at the wrist, does cause paresthesias in a median-innervated digit.

Based on the history and physical examination, the suspicion of CTS should be strong. We then are confronted with the prior nerve conduction studies showing normal median and ulnar motor, sensory, and F responses, along with normal needle EMG findings of both APB muscles. This information was initially used to rule out the presence of CTS and unfortunately led to diagnostic confusion. Further investigations included a cervical MRI

CASE 17–2. Nerve Conduction Studies

Nerve Stimulated	Stimulation Site	Recording Site	Amplitude motor = mV; sensory = μV			Latency (ms)			Conduction Velocity (m/s)			F Wave Latency (ms)		
			RT	LT	NL	RT	LT	NL	RT	LT	NL	RT	LT	NL
Median (m)	Wrist	APB	6.2		≥4	4.2		≤4.4				29		≤31
	Antecubital fossa	APB	6.0			7.9			54		≥49			
Ulnar (m)	Wrist	ADM	9.0		≥6	2.9		≤3.3				28		≤32
	Below elbow	ADM	8.9			6.4			57		≥49			
Median (s)	Wrist	Index finger	24		≥20	3.4		≤3.5	56		≥50			
Ulnar (s)	Wrist	Little finger	22		≥17	2.9		≤3.1	62		≥50			
Median (mixed study)	Palm	Wrist	30		≥50	2.4		≤2.2	40		≥50			
Ulnar (mixed study)	Palm	Wrist	15		≥12	1.8		≤2.2	62		≥50			
Mixed difference						0.6		≤0.3						
Median (m)	Wrist	Second lumbrical	1.4		≥1.0	3.7								
Ulnar (m)	Wrist	Interosseous	4.5		≥2.5	2.9								
Lum – int difference						0.8		≤0.4						
Median (s)	Wrist	Ring finger	21		≥10	3.4			40		≥50			
Ulnar (s)	Wrist	Ring finger	23		≥10	2.8			50		≥50			
Digit 4 difference						0.6		≤0.4						

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

Note: All sensory and mixed latencies are peak latencies. All sensory and mixed-nerve conduction velocities are calculated using onset latencies. The reported F wave latency represents the minimum F wave latency.

CASE 17–2. Electromyography

Muscle	Insertional Activity	Spontaneous Activity		Voluntary Motor Unit Action Potentials				
		Fibrillation Potentials	Fasciculations	Activation	Recruitment	Configuration		
						Duration	Amplitude	Polyphasia
Right APB	NL	0	0	NL	NL	NL	NL	NL
Right FDI	NL	0	0	NL	NL	NL	NL	NL
Right triceps brachii	NL	0	0	NL	NL	NL	NL	NL
Right FCR	NL	0	0	NL	NL	NL	NL	NL
Right PT	NL	0	0	NL	NL	NL	NL	NL

NL = normal; APB = abductor pollicis brevis; FDI = first dorsal interosseous; FCR = flexor carpi radialis; PT = pronator teres.

scan, which demonstrated an increased T2 signal in the center of the cervical spinal cord. With this new information, the patient's symptoms and signs were attributed to a syrinx.

At this point, what is the next logical step? When there is a discrepancy among the clinical history, examination, and electrophysiologic findings, one should always go back to the patient's history and physical examination. The history and physical examination clearly suggest CTS. There is nothing in the history or the examination to suggest a syrinx, despite the MRI scan result. A syrinx in the cervical cord usually is associated with a suspended, dissociated loss of pain and temperature sensation over the shoulders due to early involvement of the crossing spinothalamic fibers in the spinal cord, which lie adjacent to the central canal. In addition, there usually is asymmetric wasting and weakness of selected upper extremity muscles with reflex changes, depending on the spinal segments involved. Thus, what does one make of the initial nerve conduction and EMG studies? Despite the normal initial test results, the diagnosis of CTS should not be abandoned. There certainly is a group of patients with a history and examination very suggestive of CTS in whom the routine median motor and sensory conduction studies are normal. In this group, the more sensitive median-versus-ulnar comparison studies often are needed to make an electrodiagnosis of median neuropathy at the wrist.

Nerve conduction studies are repeated, which show normal routine median and ulnar motor and sensory

conduction studies, as were reported in the initial study. The F responses are normal in an absolute sense, although the right median F response is 1 ms longer than the ulnar F. When the three median-versus-ulnar comparison studies are performed, however, each is abnormal: (1) the median-versus-ulnar palm-to-wrist latency difference, 0.6 ms, is clearly above the upper range of normal; (2) the distal motor latencies to the median second lumbrical-versus-ulnar INT muscles at an identical distance reveal a latency difference of 0.8 ms, again clearly above the normal cutoff; and, finally, (3) the antidromic median and ulnar sensory responses recording the ring finger at identical distances reveal a peak latency difference of 0.6 ms, again above the upper limit of normal. The EMG study of the right upper extremity reveals no active denervation or reinnervation in the APB, the FDI, or in more proximal median- or C7-innervated muscles.

At this point, an electrodiagnostic impression can be formed.

IMPRESSION: *There is electrophysiologic evidence of a mild right median neuropathy at the wrist.*

What is one to make of the abnormal MRI scan showing a syrinx? In this case, a repeat MRI scan disclosed no evidence of a syrinx, and the abnormality on the original MRI scan was interpreted as an artifact from the magnetic coil.

Several questions should be considered.

Does the Clinical – Electromyographic Correlation Make Sense?

The answer clearly is yes in the case of the second set of studies performed on this patient. The patient's clinical history and physical examination were very suggestive of CTS, and she had a clear predisposing factor, rheumatoid arthritis. The important point here is that although the routine median motor and sensory conduction studies are normal, the more sensitive median-versus-ulnar comparison studies all are abnormal, demonstrating preferential median slowing across the wrist when compared with ulnar conduction across the wrist. In cases of mild CTS, abnormalities in these three comparison studies usually are closely correlated with one another. One should be hesitant to make any diagnosis based on a single abnormality. It is easy to imagine that one distance or latency measurement may be slightly in error; one would be remiss to make a diagnosis based on a single piece of abnormal data. In this case, all three median-versus-ulnar comparison studies are abnormal. Collaborating clinical evidence that supports an electrical diagnosis is always desirable.

The initial clinical–electromyographic correlation did not make sense: that of a patient with intermittent paresthesias of the second and third digits provoked by sleeping or driving, with no other neurologic signs, caused by a cervical syrinx, and with normal electrophysiologic results. This case reinforces the notion that CTS is a clinical diagnosis. Rarely, there will be a patient with clinical CTS in whom all electrodiagnostic tests are normal, even when the sensitive comparison studies are done (i.e., a false negative). In these patients, there is no demyelination or axonal loss; presumably, the symptoms are caused by intermittent compression resulting in transient ischemia. This case also reinforces the fact that incidental or erroneous test results with no clinical or electrophysiologic correlate, in this case the supposed "syrinx" seen in the cervical cord on the original MRI scan, should not take on undue meaning.

If this Patient has Carpal Tunnel Syndrome, Why are the Median Motor and Sensory Distal Latencies Normal?

This situation is not uncommon. Patient's results are commonly compared with population normal values. For example, in this patient, the distal median motor latency is 4.2 ms, which is within the normal range. However, the word range must be emphasized. There is a wide range of normal values. For instance, 1 year ago, before the onset of rheumatoid arthritis and CTS, the patient may well have had a distal median motor latency of 3.5 ms. When her distal motor latency increased from 3.5 to 4.2 ms, it became markedly prolonged in relationship to its own baseline normal. However, the value still falls within the "population normal range." It is in these cases that the median-versus-ulnar comparison studies are of greatest value because they rely on the patient's own nerves, rather than population normal values, as a control. Variables such as temperature, nerve length, size, age, and coexistent polyneuropathy all are controlled.

When the median motor and sensory latencies are normal in patients with CTS, the values often are near the upper limit of the normal range. Values near the upper limit of the normal range should be a clue that there may be an underlying abnormality. In the present case, the median distal motor latency of 4.2 ms is very close to the upper limit of normal (4.4 ms), and the median sensory peak latency of 3.4 ms is very close to the upper limit of normal (3.5 ms).

Should Electromyography and Nerve Conduction Studies be Used to Rule Out Carpal Tunnel Syndrome?

The answer is no. The value of EMG and nerve conduction studies lies in confirming the clinical impression, assessing the severity of the neuropathy, and looking for possible coexisting conditions. As already noted, patients with mild disease may have normal routine median motor and sensory studies, and rare patients will have completely normal studies, including the more sensitive median-versus-ulnar comparison studies. No laboratory test is 100% sensitive and 100% specific. CTS remains a clinical diagnosis. Once again, EDX tests can be interpreted properly only with knowledge of the clinical history and examination.

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