Carpal Tunnel Syndrome

The non-idiopathic causes of carpal tunnel syndrome (CTS) involve intrinsic and extrinsic conditions responsible for nerve compression. To establish a work-related association, there should be a history of excessive or unusual hand use of a nature known to be associated with CTS prior to the onset of symptoms.

By Ron Gorsché, MD, MMedSc (Occupational Health), CCFP

Carpal tunnel syndrome (CTS) is responsible for the most time lost in the workplace, yet there is little consensus regarding work as a causative factor of the syndrome. Little is known about CTS and it is among the most controversial of disorders. This article focuses on how recent literature has contributed to the theories of pathophysiology and pathogenesis of CTS, and provides clinicians with a more scientific approach to causative factors and treatment.

Historical Perspectives

In 1860, Paget reported the first cases of median nerve compression of the wrist — one case attributed the disorder to a tight band wrapped around the wrist and the other cited complications associated with a fractured distal radius.¹ In 1941,
Woltman first postulated the possibility of nerve compression within the carpal tunnel as a cause of “median neuritis,” after reporting 12 cases associated with acromegaly. Phalen, who reported his extensive clinical experience between 1950 and 1972, supported active hand use as a factor in symptom aggravation, but cast doubt on work as the sole etiology. Phalen raised one of the primary issues concerning the definition of work-related aspects of CTS. He noted the distinction between factors that aggravate symptoms of CTS and factors responsible for the development of the condition.

Defining Carpal Tunnel Syndrome

CTS can be defined simply as a complex of symptoms resulting from the compression of the median nerve at the carpal tunnel. Median nerve entrapment is the pathological process that causes symptoms of CTS. This approach works well for the clinician attempting to explain the syndrome to a patient, but requires further classification for epidemiological study. It also is effective when considering treatment options.

Although there is no gold standard case definition for epidemiological study, to simply use “arm pain” as a definition, as does the Department of Labor in the United States, grossly overestimates the prevalence. A combination of median nerve-specific symptoms and electrodiagnostic signs provides the most accurate diagnostic information (Table 1). To rely solely on positive nerve conduction studies has a poor predictive value, since, in some populations, as few as 22% of individuals found positive by Nerve Conduction Study (NCS) actually had CTS symptoms. After 17 months of follow-up with a matched cohort, this finding did not change. Furthermore, these workers were not found to be at risk for future development of CTS.

In the presence of electrodiagnostic data, physical findings aside from thenar muscle wasting add...
little to the accuracy of diagnosis. All other provocative diagnostic tests, such as Tinel’s, Phalen’s, wrist compression, and two-point discrimination, are subjective and, therefore, are not sensitive. In the absence of electrodiagnostic testing, however, adding one positive provocative physical finding to the case definition is most accurate.

Prevalence and Incidence
The prevalence of CTS in the general population has been estimated to be 5% for women and only 0.6% for men.5 The surgical incidence among the working population in a Montreal study has been calculated to be 0.9 per 1,000 adults.6 In studies of specific groups of workers, the incidence in computer users is actually no different than in the general population, whereas in meat packers it is reported as high as 11 per 100 person years.7,8

Anatomy
- This fibro-osseous U-shaped canal is made up of a bony floor and walls and a roof of fibrous flexor retinaculum. The proximal edge of the canal is near the distal wrist crease at the level of the pisiform carpal bone. The canal then

### Table 1

<table>
<thead>
<tr>
<th>Category</th>
<th>Symptoms</th>
<th>Electrodiagnostics(EDS)</th>
<th>Ordinal Likelihood of CTS</th>
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<tbody>
<tr>
<td>Classic/Probable</td>
<td>Nocturnal symptoms, tingling, burning or pain in at least 2 digits of 1,2, or 3. Palm pain, wrist pain or radiation proximal to wrist.</td>
<td>Positive</td>
<td>+++</td>
</tr>
<tr>
<td>Classic/Probable</td>
<td>As above</td>
<td>Negative</td>
<td>+/-</td>
</tr>
<tr>
<td>Possible</td>
<td>Tingling, numbness, burning or pain in at least 1 of the first 3 digits 1,2, or 3</td>
<td>Positive</td>
<td>+ +</td>
</tr>
<tr>
<td>Unlikely</td>
<td>No symptoms in digits 1,2,3,4</td>
<td>Positive</td>
<td>—</td>
</tr>
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*In the absence of electrodiagnostic studies, a combination of symptoms and one positive provocative sign, such as Tinel’s, Phalen’s, wrist compression test or 3 point discrimination gives the best diagnostic information.

*Thenar muscle wasting is the only truly objective physical sign of CTS and increases the likelihood of CTS in all categories.
extends distally about 2.5 cm to the level of the hook of hamate. The normal contents of the canal or tunnel include the flexor digitorum profundus and flexor digitorum superficialis tendons of digits II-V, the flexor pollicis longus and the median nerve as shown in Figure 1.

- Tendon sheaths cover the tendons within the carpal tunnel. The tendon sheath for the flexor pollicis longus, also called the radial bursa, begins proximal to the carpal tunnel and extends to the base of the distal phalanx of the thumb. The ulnar bursa is larger and covers the superficial and deep tendons of digits II-V. It also begins proximal to the carpal tunnel, extending to the base of the distal phalanx of digit V and to the middle of the metacarpals.

- In many individuals, the proximal ends of the lumbrical muscles that originate on the flexor digitorum profundus tendon enter the distal end of the carpal tunnel during flexion.9 At the opposite, or proximal end, of the carpal tunnel, the flexor muscle bellies will pass by the pisiform carpal bone at the entrance to the canal when the wrist is extended greater than 30 degrees and intra canal pressures would exceed 30 mmHg, if fingers were extended to 0 degrees or flexed greater than 45 degrees.10

- Although the median nerve innervates the thumb, index, middle and one-half of the ring fingers, aberrant or cross-over innervation from the ulnar nerve has been found in 30% of hands. The motor branch of the median nerve to the thumb can exit prior to the flexor retinaculum, in the middle, or distal to it.

Peripheral Nerve Structure and Function

The neuron consists of a cell body located in the anterior horn (motor) or dorsal root ganglia (sen-
sory) and an axon extending into the periphery, and made up of myelinated and non-myelinated fibers. These fibers are arranged in bundles, called fascicles, surrounded by membrane, called a perineurium. The fascicles are organized into groups held together by a loose connective tissue, called the epinerium. The endoneurium is the connective tissue that separates the individual nerve fibers from their basement membrane. There are a few important properties of peripheral nerve physiology that help explain the changes that occur during periods of increased pressure or injury.

- The peripheral nerve has a well-developed microvascular system that supplies the energy for axonal transport of nutrients. Disturbances in this transport mechanism may be involved in the development of diabetic neuropathy, also rendering the nerve more vulnerable to injury, such as compression at the carpal tunnel.
- The vessels have a coiled configuration, allowing for uncompromised circulation during normal gliding of the nerve.
- There is a blood-nerve barrier protecting the endoneurial space, and the tissue pressure within the fascicle is slightly positive.
- There are no lymphatics within the endoneurial space. Any edema that develops, therefore, may increase pressure within the fascicle rapidly and interfere with the microcirculation.
- The median nerve can move an average of 9.6 mm during wrist flexion and slightly less so during extension.

### Pathophysiology

CTS occurs as a result of an increase in pressure transmitted to the median nerve within the canal. Two theories have been proposed to explain the effect this increase in pressure has upon the median nerve. Both theories attempt to explain the nerves response to pressure, but not the cause.

**Microvascular insufficiency.** The acute symptoms of pain, paraesthesia and night numbness are thought to be secondary to ischemia in the median nerve. These cases are characterized by rapid, reversible nerve conduction changes and symptomatic improvement following carpal

<table>
<thead>
<tr>
<th>Effects of Compression on the Peripheral Nerve</th>
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<tbody>
<tr>
<td>• Pressures of 80 mmHg (10.7 kPa) interrupts all intraneural blood flow.</td>
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<tr>
<td>• Pressures above 30 mmHg (4.0 kPa) inhibit all antegrade and retrograde axonal transport.</td>
</tr>
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<td>• When extraneural pressure fluctuates rapidly, the effects on nerve function are associated with the mean value of the pressure waveform.</td>
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<tr>
<td>• There is an increased tolerance for high pressures among those with hypertension. The critical extraneural pressure threshold above which nerve function is blocked is 30 mmHg (4.0 kPa) below the diastolic pressure. CTS often manifests itself after treatment for hypertension.</td>
</tr>
<tr>
<td>• Compression of 30 mmHg (4.0 kPa) led to an elevated intraneurial pressure that persisted for 24 hours. These effects are likely due to the increased vascular permeability of the epineurial and endoneurial vessels producing edema after compression.</td>
</tr>
<tr>
<td>• A pressure of 30 mmHg applied to the median nerve appears to be the critical threshold for injury.</td>
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</table>
tunnel release. One study found that up to 98% of CTS cases examined had vascular sclerosis.12

**Mechanical compression.** Persistent nerve conduction impairment is believed to be due to the effects of nerve compression. A “tadpole” lesion, characterized by thinning of the myelin, develops because of a shearing-type of pressure phenomenon at the end of the internodal segment with a bulbous-like swelling at the facing edge.

### The Effects of Nerve Compression

A dose-response curve has been established, following experiments with intra canal pressure transducers.13,14 The outcomes of such studies are important to the clinician who is faced with decisions in determining causation and prescribing modifications and appropriate splints. Table 2 summarizes the outcomes of pressure studies involving peripheral nerves. In general, axonal demyelination and degeneration is rarely seen when nerves are subjected to pressures of less than 30 mmHg.

### Carpal Tunnel Pressures Associated with Wrist and Hand Position

Measuring carpal tunnel pressure during various hand and wrist positions allows physicians to determine optimal positioning for splints and in prescribing work restrictions. As shown in Table 3, intra canal pressures follow a U-shaped parabolic curve, with only extremes of wrist and finger flexion and extension producing pressures above the 30 mmHg threshold for injury. A wrist at neutral position, with the metacarpo-phalangeal joints at 45 degrees, results in the lowest pressure and is most suitable for splinting.15

### The Effect of Force or Loading on Carpal Tunnel Pressures

Researchers have studied the effects of various hand activities and forces on the carpal tunnel pressures (Table 4).16 Highest pressures occur with forceful grasping, and the lowest with finger-tapping. Epidemiological studies have also confirmed the low incidence of CTS and protective effect of keyboarding.17,18 Activities involving forceful, repetitive and prolonged grasping or vibration, therefore, will increase pressures, whereas finger work or light repetitive grasping
Pathogenesis

The non-idiopathic causes of CTS involve intrinsic and extrinsic conditions responsible for nerve compression (Table 5). To establish a work-related association, there should be a history of excessive or unusual hand use of a nature known to be associated with CTS prior to the onset of symptoms. The carpal-canal-pressure studies outlined above provide a guide to those activities that put patients at risk of having nerve compression. The evidence suggests that neither cumulative trauma disorder (CTD) nor repetitive hand use without forceful or prolonged grasping, are hazardous. The literature is not able to settle the controversy over whether work is causative or purely an aggravation to an underlying condition. A number of personal factors have been found to be associated with CTS.\(^{19}\) There appears to be a marked hormonal influence in women (Table 6), whereas, in men, CTS is associated with morphology, such as short stature and obesity.\(^{20}\) Work-related activities found to be associated with the onset of CTS, include the following:

- Prolonged grasping;
- Forceful grasping;
- The use of handtools (especially vibrating);
- The use of the wrist in extreme flexion or extension;
- Activities that compress the palm;
- Repetitive impact to the base of the palm; and
- Shoulder rotation with arm elevated.\(^ {21}\)

Diagnosis
Signs and symptoms. CTS, by definition, is a clinical syndrome and the clinician must decide which patients are the best candidates for confirmatory electrodiagnostic study. A study examining the various clinical criteria necessary to achieve high-specificity (low false-positive) arrived at the consensus outlined in Table 1. Except for the presence of thenar muscle wasting, the addition of physical examination findings offers little to the accuracy of diagnosis. There are no sensitive, objective, provocative physical examination techniques available to the clinician. A simple hand diagram has been validated for identifying median nerve symptoms (Figure 2).22 This hand diagram is especially useful in populations where language is a concern. The use of a simple pinwheel is helpful when mapping out areas of numbness or differences between hands. There has been a significant association between bilateral CTS, present in up to 30% of cases, and cervical radiculopathy, often referred to as double crush syndrome.23 If symptoms are purely sensory, the theory of interruption to axonic flow, causing increased peripheral nerve susceptibility, is less likely, since flow from the nerve root to the spine and periphery is separate and independent.24

Electrodiagnostic study. The electrodiagnostic study (EDS) consists of two components — the nerve conduction study (NCS) and the electromyographic examination (EMG). These studies can be administered safely in patients with pacemakers and cardiac arrhythmias. Patients with human immunodeficiency virus (HIV), hepatitis or Cruetzfeld-Jacob disease can be tested with disposable needles. Patients can be reassured that electric shocks of varying intensity are slightly uncomfortable, but harmless. A neurologist performs the examination, with the limb
warmed to at least 32 degrees, and will follow the guidelines set out by the American Association for Electrodiagnostic Medicine.

The Electrodiagnostic Glossary of Terms

**The nerve conduction study (NCS).** The purpose of the NCS is to determine if there is a time delay, a change in intensity, or a reduction in velocity of a nerve impulse between one section of a peripheral nerve and another. In the case of CTS, any delay of motor and sensory impulses traveling across the carpal tunnel is of interest.

- **The Sensory Axon.**
  - Sensory nerve action potential (SNAP): When sensory axons are examined, the SNAP is recorded traveling to the distal limb (antidromically or against the normal direction) and returning from the distal limb (orthodromically or the direction normally taken).
  - Distal sensory latency, or onset latency, is the length of time it takes for a stimulus to travel a set distance to initiate a SNAP.
  - Sensory amplitude: The amplitude of the SNAP is not determined by the number of axons, but by the cross-sectional density of the conducting sensory axons within the nerve.

- **The Motor Axons** are assessed by placing an electrode on the skin overlying a muscle innervated by the median nerve, such as the thenar eminence.

### Table 6

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<tr>
<th>Risk Factor</th>
<th>OR</th>
<th>95% CI</th>
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<tbody>
<tr>
<td>Prior musculoskeletal complaint for which consultation sought</td>
<td>1.98</td>
<td>1.61 to 2.42</td>
</tr>
<tr>
<td>Obesity</td>
<td>1.68</td>
<td>1.29 to 2.18</td>
</tr>
<tr>
<td>Prior oral contraceptive use (but not current use) in women over 40 years old</td>
<td>1.38</td>
<td>1.08 to 1.76</td>
</tr>
<tr>
<td>History of consultation for any menstrual disorder</td>
<td>1.36</td>
<td>1.11 to 1.66</td>
</tr>
<tr>
<td>Lower socioeconomic class</td>
<td>1.23</td>
<td>1.02 to 1.50</td>
</tr>
<tr>
<td>Current use of hormone replacement therapy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Respiratory complaints</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pregnancy</td>
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</table>

Males: Obesity, age, short stature and history of stenosing flexor tenosynovitis.
- Compound muscle action potential (CMAP): The summated depolarization of the muscle fibers underlying an electrode produces a waveform, called the compound muscle action potential.
- Distal motor latency (DML): The length of time between stimulation at a standard site and the onset of the CMAP is termed the distal motor latency.
- Motor conduction velocity: By stimulating the nerve at two different sites, the motor nerve conduction velocity can be calculated.

The distal motor latency and motor nerve conduction velocity are mainly dependent on the integrity of the myelin sheath, whereas the amplitude and shape of the CMAP offer information about the number of functioning axons supplying the muscle. In demyelinating disorders, therefore, there will be abnormally prolonged distal latencies and reduced conduction velocities, due to slowed impulse transmission. If the entrapment continues, axonal degeneration occurs, and the recorded SNAP and CMAP will be absent or abnormally small. This is due to a loss of impulse conduction or a conduction block. The reference laboratory should determine the normal ranges for any electrodiagnostic study. The normal range has traditionally been established as the mean +/- 2 standard deviations.

The electromyogram (EMG). A needle electrode inserted into muscle can be used to assess the electrical stability of the muscle membrane and the electrical properties of a muscle undergoing voluntary contraction. Pathological changes in nerves and muscle, plus many metabolic abnormalities, may be detected. The needle electrode examination begins by recording the muscle at rest, searching for spontaneous potentials, such as:
- **Fibrillation potentials** and **positive sharp waves**. These are assessed by recording the muscle at rest, while looking for spontaneous potentials from a single muscle fiber. These findings indicate underlying muscle denervation. Whole motor units can be assessed next by looking for the presence of:
  - **Fasciculations**. Whole motor units can be evaluated, regardless of the presence of denervation, because fasciculations may originate in the spinal cord or anywhere along the length of the motor axon.
  - **The motor unit action potential (MUAP)** is a summated, large single waveform, which results from the near simultaneous discharge of the muscle fibers supplied by an anterior horn cell, stimulated during a voluntary muscle contraction. Disease processes affecting the motor unit can be detected more readily with this measurement. A neurologist may decide that an electromyogram (EMG) is unnecessary in a straightforward case of CTS. If, however, the symptoms are atypical or a concomitant cervical lesion is present, an EMG may be
helpful. The clinician must remember that there are at least six regions proximal to the carpal tunnel where the median nerve may be entrapped (Figure 3).

**Laboratory Investigations**

All cases of CTS that fail to improve with conservative treatment should undergo a baseline investigation to rule out other associated diseases. This should include tests for s-thyroid stimulating hormone (s-TSH), fasting blood sugar, protein electrophoresis, B12, rheumatoid factor, antinuclear antibody (ANA) and erythrocyte sedimentation rate (ESR). If there is a history of previous trauma to the wrist or hand an x-ray should be ordered.26

**The Stages of CTS**

With the foregoing knowledge of pathophysiology, three stages of CTS have been proposed (Table 7). Treatment is based on the severity determined by both clinical and electrodiagnostic assessment.

**Treatment**

The goal of treatment is the resolution of symptoms and preservation of hand function. A significant number of CTS patients will improve with no treatment. This is especially true for young workers with severe initial impairment.27 This helps explain why many unproven, alternative therapies claim success. For those patients who fail to resolve spontaneously, early intervention is the key to successful outcomes.

**Conservative: 50% to 75% improvement. Work Restrictions.** Patients should be instructed to avoid activities associated with CTS, both at work and at home. Such activities include using hand tools, forceful grasping, prolonged grasping, placing pressure against the palm and awkward positions of extreme flexion or extension.

**Night splints.** A custom, molded night splint should be fashioned by an occupational therapist (Figure 4). Using carpal canal pressure principles, as outlined previously, the splint should maintain the wrist in a near-neutral position with a few degrees of ulnar deviation and with metacarpophalangeal (MP) joints at 45 degrees. Night splints prevent the incursion of the lumbrical muscles into the distal carpal tunnel, which occurs with wrist flexion during sleep. Daytime working splints are of little value. Although one recent article found improvement in one aspect of sensory conduction, there was

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**Table 7**

<table>
<thead>
<tr>
<th>Classification of the Severity of the Electromyogram</th>
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<tbody>
<tr>
<td>Mild</td>
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<tr>
<td>------</td>
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<tr>
<td>Motor Latency</td>
</tr>
<tr>
<td>Sensory Latency</td>
</tr>
<tr>
<td>Sensory Amplitude</td>
</tr>
<tr>
<td>Denervation</td>
</tr>
</tbody>
</table>

no improvement in symptoms.\textsuperscript{28}

\textbf{Medications.}

\begin{itemize}
  \item Nonsteroidal anti-inflammatories (NSAIDs) improve pain, but have not been shown to be effective treatment for long-term resolution.
  \item Prednisone (1 mg/kg) orally often provides symptomatic relief in an acute onset.
  \item Steroid injection into the carpal tunnel produces short-term relief of symptoms, but risks include median-nerve damage. There is one randomized controlled trial reporting symptomatic improvement with injection of 40 mg of methylprednisolone into the volar forearm, 4 cm proximal to the wrist crease.\textsuperscript{29}
  \item There is no evidence to date of an association between CTS and vitamin B deficiency.
  \item Vitamin C has not been shown to be effective for this purpose. Be aware that supplementing with vitamin C in the presence of vitamin B6, or ingesting more than 500 mg per day of vitamin B6, may injure the nerve.\textsuperscript{30}
\end{itemize}

\textbf{Alternative therapies.} There is insufficient evidence to support the use of physiotherapy, ultrasound, magnets, lasers or manipulation in the treatment of CTS.

\textbf{Surgical treatment (90\% improvement).} Surgical treatment should only be considered in the presence of ongoing symptoms or signs of thenar wasting, together with electrodiagnostic confirmation. The use of the short 2 cm to 3 cm palmar incision to release the flexor retinaculum is the method preferred by most reports in the literature. Complications include injury to the thenar motor branch, injury to the median nerve itself, infection, bleeding and failure to resolve symptoms. A tender surgical scar is the post-operative complication that most often hampers return to work. Endoscopic surgical release, however, has been largely abandoned because of a higher surgical complication rate.

\textbf{Post-operative return to work.} The most successful return-to-work outcomes have occurred when the wrist is not splinted post-operatively and the patient is instructed in a graduated program of early mobilization.\textsuperscript{31} Most patients can return to light-hand use following the removal of sutures, but may not tolerate the use of hand tools for an average of six to eight weeks. All cases of surgical failure should undergo a complete examination and repeat electrodiagnostic assessment to rule out other, less common, causes of peripheral neuropathy.

\textbf{Conclusion}

In summary, compression of the median nerve within the carpal tunnel at the wrist is responsible for the symptoms and electrodiagnostic findings in CTS. Modifications in hand use, as well as night splints to reduce intra-canal pressure, are the most effective conservative interventions, and, failing that, surgery is more than 90\% successful.

\textbf{References}

7. Stevens JC, Witt JC, Smith BE, et al: The frequency of carpal tunnel syndrome in computer users at a medical
Carpal Tunnel Syndrome


Suggested Readings

Put Your Knowledge to the Test

Answer the questions in our quiz found on page 263 and send the response card to the University of Calgary for CME credits.