WorkCoverSA

A clinician's guide to carpal tunnel syndrome incorporating workers compensation aspects

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Acknowledgement and participation

This material has been written for health care professionals and others to assist them in managing this specific condition. The executive summary and recommendations in this document are supported by recently published material and current best practice guidelines applying evidence-based medicine principles.

In addition the following organisations have supported its content:

- Royal Australasian College of Physicians
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Contents

Executive summary and recommendations

Carpal tunnel syndrome (CTS) is a common condition in both the general community and in the work environment.

- The loss to the South Australian community, both social and economic, through the workers compensation system is significant and potentially, in part, preventable. (See Preamble, page 5.)
- CTS is a clinical diagnosis. This is supported, when appropriate, by electrophysiological tests and increasingly by imaging, especially ultrasound. (See Clinical assessment – Diagnosis, page 8 and Neurophysiology, page 14.)
- Nerve conduction studies (NCS) are helpful in determining the severity and the response to treatment of CTS. Normal NCS should prompt the clinician to consider alternative diagnoses. (See Clinical assessment – Differential diagnosis, page 11.)
- Early identification and appropriate management can potentially reduce the disability and improve health outcomes and return to work rates. Practitioners should also consider the implications of psychosocial and workplace issues (yellow flags) when considering return to work management. (See Non-surgical management, page 21.)
- Ergonomic approaches to job design can reduce the incidence of the condition or aggravating factors in the work environment. (See the Occupational environment, page 19 and Non-surgical management, page 21.)
- There is a role for conservative treatment of CTS but surgical intervention should be considered in significant cases. (See Management – Non-surgical management, page 21 and Surgical management, page 22.)
- Given the significant incidence of persistent symptoms following surgery in the occupational environment, a baseline NCS prior to surgery is recommended. (See the Utility of nerve conduction testing, page 17.)
- Early return to work following intervention is often achievable and current evidence supports the notion that it can result in superior outcomes and reduced community costs. This includes certification for work, when indicated, remembering that certification should incorporate any appropriate restrictions to either time or of duties. (See The occupational environment, page 19.)
- When difficulties arise in managing the return to work, consideration should be giving to early referral to specialists to facilitate this process. The considerations should also include the recognition of any psychosocial and/or workplace issues. The specialists can include:
  - rehabilitation and occupational medicine physicians
  - psychologists or physical therapists with expertise in work places.

You should also consider the involvement and assistance of vocational rehabilitation consultants and the case manager.

Preamble

Carpal tunnel syndrome (CTS) is a significant problem in the South Australian working environment. The number of workers compensation claims lodged for the financial years 2000-05 and the related cost to the South Australian Workers Compensation Scheme is shown in table 1 below. The claims data quoted relates only to employers registered with WorkCover, it does not include claims data for self-insurers. It is estimated that self-insurers account for approximately 40% of the SA workforce.

Table 1 – Number of CTS claims and costs in South Australia, as at 10 July 2008

Year	Number of claims	Total costs	Average cost per claim	Median cost per claim
2000-01	297	$7,844,818	$26,414	$3,934
2001-02	298	$8,486,669	$28,479	$3,954
2002-03	289	$10,348,530	$35,808	$5,777
2003-04	238	$7,275,638	$30,570	$6,327
2004-05	241	$5,472,989	$22,709	$5,038

While the number of claims for CTS has reduced by about 19% for this period, the number of all claims lodged over the same period has also reduced by 14% (WorkCoverSA annual reports).

For the financial year 2002-03 the outcomes relating to CTS are shown in Table 2 below. The data is at the 10 July 2008 which allowed a minimum of five years development.

Table 2 – Outcomes of CTS claims in South Australia for 2002–03 as at 10 July 2008

	Number claims	Total cost	Average cost per claim	Average no. of days that weekly comp is paid	Claims with a return to work	Claims without a return to work	Claims with liability redeemed
Time lost*	169	$9,949,475	$58,872	380	132	17	20
No time lost**	120	$483,600	$4,030				

**   Based on WorkCover having made a payment of weekly compensation.*

***   In SA the employer is responsible for first two weeks of compensation payments.*

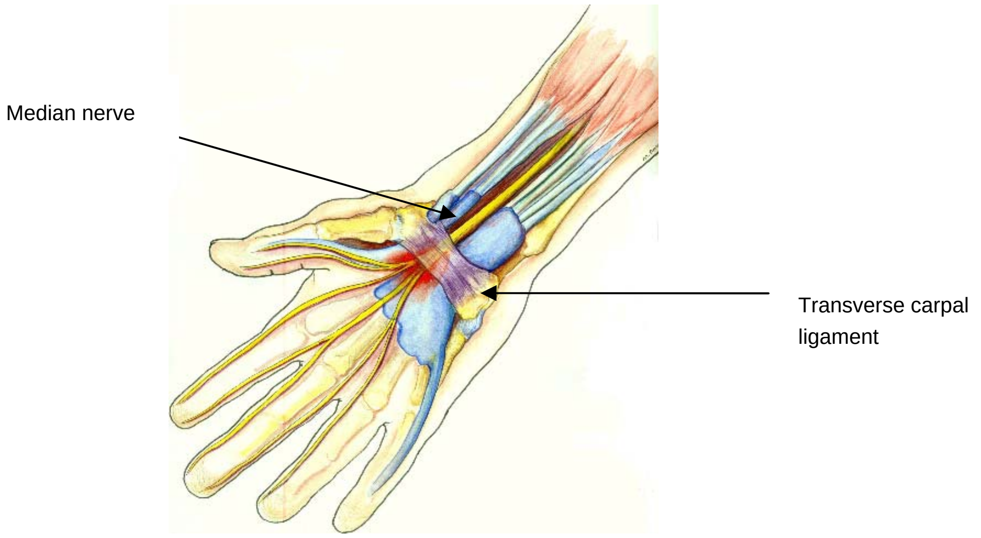
While approximately 57% of workers who lodged claims for CTS claims had time off work, of those, 10% were still off work five years after claims were lodged. All of the claimants without a return to work (RTW), as at July 2008, had surgical intervention for carpal tunnel release. These outcomes are in contrast to non-compensable CTS injuries where usually there is less than six weeks time off work post-surgery and where most activities are continued after treatment. The delayed and poorer health outcomes for compensation patients are well recognised, although not fully explainable[1]. Psychosocial, personal, family and employment issues may partly explain this phenomenon.

Introduction

The term carpal tunnel syndrome came into use in the 1950s. The syndrome, consisting of intermittent paraesthesiae in the hands, was recognised in the 19th century and was known by a variety of names including acroparaesthesia syndrome, median neuritis, median thenar neuritis and in the more chronic cases, tardy median palsy[2]. It was not until the 1930s that it was clearly demonstrated that the syndrome was due to compression of the median nerve at the wrist and that surgical decompression (by dividing the transverse carpal ligament) might provide symptomatic relief and preserve median nerve function[3].

The syndrome is defined by its clinical features[4]. These may be summarised as the presence of intermittent paraesthesiae in the affected hand often associated with pain from the hand which radiates proximally to the forearm and elbow and occasionally to the shoulder. The symptoms are often prominent at night or on waking in the morning. More severe cases demonstrate evidence of motor and sensory deficits consistent with a median neuropathy occurring at the wrist.

Carpal tunnel syndrome occurs as a result of raised pressure in the carpal tunnel, implying that structures in the tunnel have an increased volume or that the tunnel dimensions have decreased.[5]. The pathology of idiopathic carpal tunnel syndrome is a non-inflammatory fibrosis of the sub-synovial connective tissue surrounding the flexor tendons[5]. Secondary causes such as pregnancy, obesity, hypothyroidism, synovial cysts, rheumatic conditions and proximal extension of the lumbrical muscles are other potential causes of increased volume of the contents of the carpal tunnel. Arthritis of the carpal joints and deformity from previous wrist fracture may diminish the dimensions of the canal.



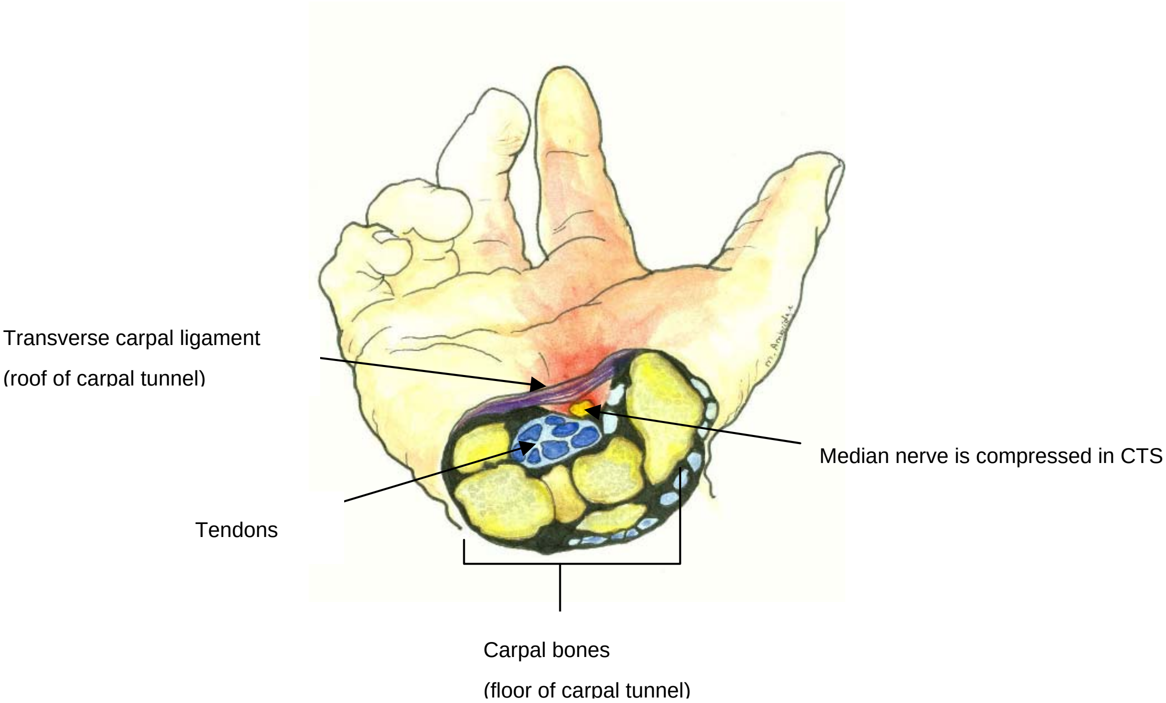


Figure 1 – Wrist and hand showing internal structures

Epidemiology

The prevalence of electrophysiologically confirmed, symptomatic carpal tunnel syndrome is about 3% among women and 2% among men, with peak prevalence in women older than 55 years of age[6], making it the most common of the entrapment neuropathies. In approximately one third of cases, CTS is associated with other conditions including inflammatory arthritis, amyloidosis, diabetes, hypothyroidism, acromegaly and Colles' fracture. Indeed CTS may be the presenting complaint of these conditions on some occasions. In addition, pregnancy or the use of corticosteroids or oestrogens may be associated with the condition. CTS is also associated with repetitive activities of the hand and wrist, particularly with a combination of forceful and repetitive activities[7 8]. A number of occupations are associated with a high incidence of carpal tunnel syndrome including food processing, manufacturing, logging and construction work.

Clinical assessment

Diagnosis

The diagnosis of carpal tunnel syndrome is usually straight forward, however in a small number of cases that is not so. There is no accepted 'gold standard' diagnostic tool for CTS. Potential gold standards include expert clinical diagnosis, positive response to carpal tunnel release/injections and electro-diagnosis and imaging studies. Each of these approaches is subject to limitations and potential misclassification[9]. Clinical history and examination can be supported by the use of hand diagrams of patient-reported symptoms[10]. Diagnostic scales based on self-reported symptoms have also been developed[11] as have standardised questionnaires that are reported to have reasonable sensitivity and specificity. The synthesis of a combination of a well-constructed history and examination supplemented when appropriate with nerve conduction studies or imaging techniques will provide the greatest diagnostic certainty[4 9.]

Symptomatology

Carpal tunnel syndrome characteristically presents with sensory symptoms namely complaints of intermittent feelings of pins and needles, numbness and a sensation of swelling affecting the lateral (radial) three and a half digits. Many individuals report that the paraesthesiae affect the whole hand and some describe it as affecting much of the upper limb. In these instances, education and closer observation may lead the subject to identify that the little finger is spared although some authors[12] state that the paraesthesiae (but not a sensory deficit) can involve the little finger although this is rare.

Similarly, on anatomical grounds, the sensory symptoms should spare the dorsum of the hand and fingers, supplied by the radial and ulnar nerves, and the palm of the hand, which is supplied by the palmar branch of the median nerve which arises proximal to the wrist and passes superficial to the transverse carpal ligament.

Typically the symptoms disturb sleep at night or are present on waking in the morning. Morning stiffness with difficulty in fully opening or closing the hand is common, as is reduced grip strength and a tendency to drop objects. These symptoms may reflect pressure effects on the tendon sheaths rather than nerve compression. Paraesthesiae often emerge with activities which involve sustained wrist position such as talking on a telephone, driving a car, reading a book or using certain tools. When repetitive activities exacerbate symptoms these commonly occur at rest rather than while performing the task. Pain may be felt in the hand and wrist but commonly is referred to the forearm and elbow and occasionally higher. Individuals may report that the sensory symptoms improve with repeatedly flicking the wrist and hand or by repeated flexion and extension of the fingers or, when in bed, by hanging the hand out of bed.

Patients may complain of persistent feelings of numbness in the fingertips, particularly affecting the index and middle fingers but seldom observe or complain of weakness or wasting in the median supply of thenar muscles.

Provocative tests:

A number of provocative tests have been evaluated and help improve diagnostic certainty when present.

1) Phalen's sign

As originally described by Phalen[13] the wrists are dropped by gravity assistance into flexion and a test is regarded as positive if after 30 to 60 seconds the patients sensory symptoms are reproduced. Wrist extension (reverse Phalen's sign) appears to be less sensitive than wrist flexion. A recent systematic review suggested Phalen's test has a sensitivity of 68%, and an average specificity of 73%[14].

2) Carpal compression test

The examiner compresses the median nerve by pressing over the proximal margin of the transverse carpal ligament which corresponds with the proximal wrist crease with his/her thumbs with the wrist held in a neutral position. This is positive if paraesthesiae develop in the median nerve distribution. (Sensitivity 64%, specificity 83%)[14] .

3) Tinel's sign

The test is positive if tapping over the median nerve just proximal to the flexor retinaculum produces a sensation of paraesthesiae or electric shock-like feelings in the median nerve distribution. (Sensitivity 50%, specificity 77%)[14]

A number of other provocative tests have been described, mostly of lesser sensitivity or specificity than those included here.

Neurological examination

The majority of patients presenting with carpal tunnel syndrome will have a normal neurological examination. When compression has been more longstanding or severe and the nerve has sustained axonal injury, motor and sensory deficits compatible with median nerve pathology at the wrists will be evident.

Atrophy of the median supplied thenar muscles produces a flattening of the normally convex contour of the thenar muscles. In unilateral cases, this is often best appreciated by directly comparing the bulk on one side to the other. Often weakness of the abductor pollicis brevis can be detected before wasting is evident. This should be tested with the thumb held at 90 degrees to the plane of the hand to eliminate activity of the long extensors.

Figure 2 – Bilateral wasting of the abductor pollicis brevis, greater on the right

In all but severe cases, the sensory deficits will be mild and generally confined to the pads of the terminal phalanges of the affected digits. Frequently this will be evident in only one or two digits and may consist of an alteration in the feeling of light touch or a diminution in the sensation of pin prick when compared to the uninvolved little finger. Demonstrating a qualitative difference in sensation between the lateral and medial halves of the ring finger is a particularly useful sign but one which is typically only evident in more advanced cases.

Static two-point discrimination has a sensitivity of 24% and a specificity of 95%.[14]

Touch thresholds can be measured using Semmes-Weinstein monofilaments (SWMF) but skill is involved in both administration and interpretation of SWMF tests.

The patient's ability to perceive a vibratory stimulus threshold can be a useful diagnostic tool but is technically demanding and usually reserved for the research setting.

Differential diagnosis

When the presenting problem is one of intermittent paraesthesiae which appears to affect all or most of the fingers and hand, median nerve compression at the wrist is by far the most likely diagnosis. The differential diagnosis includes compromise of other nerves at a number of anatomical sites in the upper limb, as well as pressure on the median nerve at site proximal to the carpal tunnel (Table 3).

Table 3 – The differential diagnosis of carpal tunnel syndrome

Diagnosis	Signs and symptoms
Ulnar nerve compression/injury at elbow or wrist	Sensory symptoms at the ulnar border of the hand, little finger +/- ulnar border of ring finger Possible weakness and wasting of the abductor digiti minimi, adductor pollicis and interossei and if located at the elbow flexor carpi ulnaris and deep flexors of the ring and little finger.
Median nerve entrapment at the head of pronator teres (pronator syndrome)	Pain in the upper part of the flexor compartment of the forearm during activity +/- median nerve distribution paraesthesiae +/- weakness in the median nerve supplied flexor pollicis longus and the deep flexors of the index and middle finger in addition to the weakness which may appear in the thenar muscles. Tends not to produce recurrent nocturnal paraesthesiae.
C6/C7 radiculopathy	Pain radiates down the arm to the hand and may be made worse by neck movement and proximal upper limb movement. C6 - the biceps and brachioradialis reflexes reduced or absent. A C7 radiculopathy - weakness of the triceps muscle and

	depressed or absent triceps jerk. Potential paraesthesiae in C6 (lateral forearm, thumb and index finger) or C7 (dorsal forearm and hand, index and middle finger) distribution.
Brachial plexus injury	More widely distributed neurological deficit with or without significant proximal pain.
Neurological thoracic outlet syndrome (as seen with cervical rid or band)	Arm pain often associated with traction on the extended upper limb (eg, carrying shopping bags) and focal atrophy of lateral portion of the thenar muscles. Sensory loss and paraesthesiae mainly in the medial border of the forearm +/-the ulnar border of the hand and little finger. The radial pulse may disappear with elevation of the arm and this may be preceded by a subclavian bruit with auscultation over the clavicle.
Local musculoskeletal injury/pathology	Pain in the absence of paraesthesiae is seldom related to median nerve compression.

Nearly as common as paraesthesiae in the median nerve distribution are intermittent sensory symptoms related to **ulnar nerve disturbance** which most commonly occurs at the elbow but occasionally occurs at the wrist or in other locations. Ulnar nerve sensory symptoms are almost always accurately identified as involving the ulnar border of the hand and the little finger and observant patients may also note the involvement of the ulnar half of the ring finger. In ulnar nerve pathology the symptoms are frequently related to specific elbow position and often occur when resting the affected limb on the elbow or proximal forearm, with repeated flexion/extension of the elbow and during the night with sustained flexion of the elbow. When a persistent sensory disturbance occurs it is clearly confined to the ulnar nerve distribution and the presence of sensory loss on the ulnar border of the dorsum of the hand implies that the pathology is proximal to the wrist. Weakness and wasting of the abductor digiti minimi and interossei will be evident as the neuropathy progresses and an ulnar neuropathy at the elbow may also show evidence of weakness of the flexor carpi ulnaris and the deep flexors of the little and ring finger on the affected side.

Median nerve entrapment can occasionally occur at the elbow as it passes between the heads of pronator teres **(pronator syndrome).** Typically this presents with pain in the upper part of the flexor compartment of the forearm during activity and median nerve distribution paraesthesiae may occur at that time. Weakness may occur in the median nerve supplied muscles in the forearm including the flexor pollicis longus and the deep flexors of the index and middle finger in addition to the weakness which may appear in the thenar muscles. The pronator syndrome does not tend to produce recurrent nocturnal paraesthesiae. Neurophysiological investigation will reveal normal median nerve conduction through the wrist and typically slowing of nerve conduction in the forearm.

Radiculopathy particularly affecting the **C6 and C7** nerve roots can produce a distribution of paraesthesiae which might be confused with a median nerve pattern. Nerve root compression is typically associated with significant pain which is felt to radiate down the arm to the hand. The pain is often made worse by neck movement and often also with proximal upper limb movement such as shoulder abduction or extension. The paraesthesiae are generally felt to radiate down the arm into the hand. With a C6 radiculopathy, patients commonly demonstrate the paraesthesiae radiating into the lateral forearm and the dorso-lateral aspects of thumb and index finger and with C7 radiculopathy, patients often demonstrate the paraesthesiae radiating down the dorsal aspect of the forearm into the dorsal aspect of the index and middle finger. It may be difficult to demonstrate specific muscle weakness in a C6 radiculopathy, unless it is severe, but generally the biceps and brachioradialis reflexes will be reduced or absent. A C7 radiculopathy will frequently demonstrate weakness of the triceps muscle and an absent or depressed triceps jerk.

More diffuse feelings of upper limb aching, heaviness and complaints of ‘numbness’ or a ‘dead feeling’ in the upper limb, seldom have any demonstrable neurogenic basis but may occur as a **referred phenomenon** from the **lower cervical and upper thoracic spine**.

Pathology in the **brachial plexus** generally present with a neurological deficit with or without significant proximal pain, which will be quite easily distinguished from the intermittent symptoms associated with carpal tunnel syndrome. A thoracic outlet syndrome may occasionally cause confusion. There are two distinct varieties of thoracic outlet syndrome. The neurogenic form is usually associated with a cervical rib or band which elevates and distorts the lower trunk of the brachial plexus. This may produce arm pain and focal atrophy of the lateral portion of the thenar muscles which may resemble the thenar muscle atrophy seen in median nerve compression. Sensory loss and paraesthesiae however are felt mainly in the medial border of the forearm and possibly into the ulnar border of the hand and little finger. This syndrome is quite rare whereas compression of the vascular structures with elevation of the arm is quite common and this should be looked for in patients who complain of aching, colour change or paraesthesiae when working with their arms elevated. The radial pulse is typically felt to disappear (Adson test) with elevation of the arm and this may be preceded by a subclavian bruit with simultaneous auscultation over the clavicle. It should be noted, however, that these findings are also common in asymptomatic individuals. Patients with downward sloping shoulders may also traction the neurovascular bundle when carrying loads with their arms at their sides.

Other causes of thenar, wrist and forearm pain may give rise to concern of median nerve compression. In general these musculoskeletal problems **produce pain with movement and activity**, which may persist at rest and be associated with morning stiffness. Pain in the absence of paraesthesiae is seldom related to median nerve compression and this possibility only warrants consideration if the pattern of the pain occurrence is typical of carpal tunnel syndrome.

Neurophysiology

Electrodiagnostic tests were introduced soon after the description of carpal tunnel syndrome [15] as a means of demonstrating impairment of median nerve conduction at the level of the wrist. Over the last half-century this has proven to be an invaluable tool which has helped identify the characteristics of the syndrome.

Electrodiagnostic studies contribute the following regarding CTS:

(1) Information regarding the severity of the median neuropathy
(2) A baseline to evaluate the progression or responsiveness to treatment
(3) The possibility of alternative or associated diagnoses.

The neurophysiological assessment of the peripheral nervous system generally involves performing nerve conduction studies (NCS), which examine the function of the motor and sensory components of peripheral nerves and electromyography (EMG) in which muscle activity is examined using a concentric needle electrode. EMG is not commonly required in the assessment of routine cases of carpal tunnel syndrome.

Nerve conduction studies assess the function of the large diameter myelinated motor and sensory fibres, and routine techniques do not assess the function in small myelinated and unmyelinated nerve fibres. Nerve compression results in demyelination particularly in the region of the nodes of Ranvier. This effects saltatory nerve conduction producing focal slowing of conduction velocity. The demonstration of localised median nerve compression at the level of the wrist relies on the finding of slowing of sensory and/or motor conduction in this segment of the nerve. As the effects of compression increase, conduction may become more dispersed or may fail completely (conduction block) and axonal injury and loss also occurs. This leads to a reduction in the amplitude of the sensory responses (sensory nerve action potentials or SNAPs) and the motor responses (compound muscle action potentials or CMAPs).

In carpal tunnel syndrome, nerve conduction studies define and quantify the extent of impairment of nerve function and confirm the location of the disturbance[16].

Nerve conduction testing – methodology

Motor conduction studies

In routine practice, the motor conduction study is performed by placing recording electrodes over the median supplied thenar muscle, the abductor pollicis brevis (APB). The nerve is then stimulated with short duration electrical pulses which are increased in intensity until the maximal response is obtained from the APB. The latency to onset of the response is known as the distal motor latency and this is typically prolonged when median nerve compression is present. The amplitude of the response is also recorded. This will be typically within the normal range with mild-to-moderate median nerve compression but will be diminished with more severe compression. The median nerve is then stimulated at the elbow and the stimulation intensity is increased until the same response recorded at the wrist is achieved. The distance between the two sites of stimulation is measured and the difference in latency between the response at the wrist and the response of the elbow allows the forearm conduction velocity to be calculated. In carpal tunnel syndrome this should be normal.

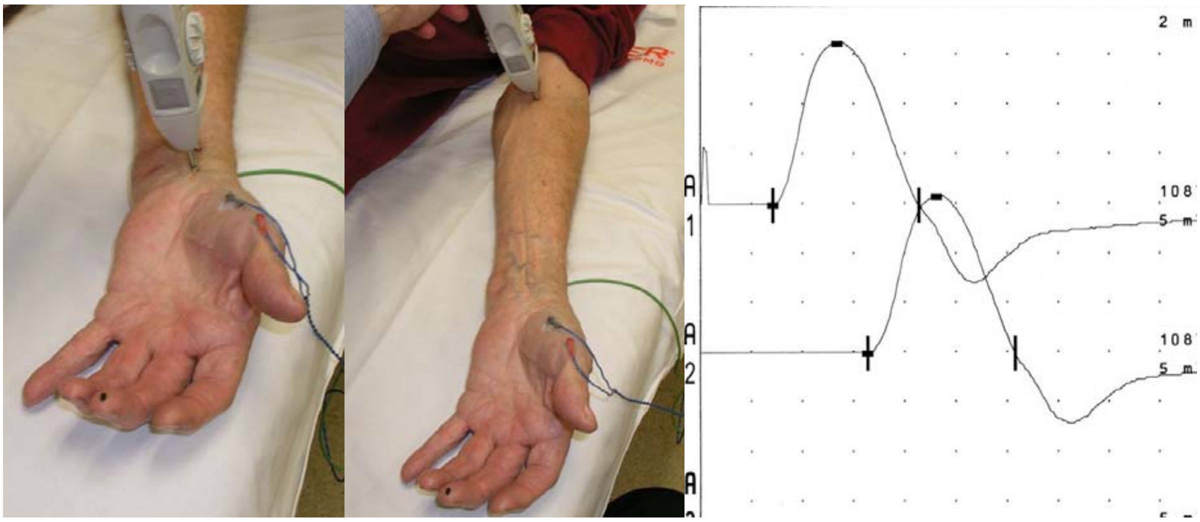


Figure 3 - Photo showing electrode placement and showing typical findings in CTS.

Overall reported sensitivity for motor nerve conduction studies varies from 43-80% and specificity from 95-100%[9].

An ulnar nerve motor conduction study is usually performed as a control to ensure that subclinical generalised neuropathy or unappreciated cool limb temperatures are not responsible for abnormal findings in the median nerve.

Sensory conduction studies

The purely sensory digital nerves are stimulated with ring electrodes around the finger or thumb. This evokes a sensory nerve action potential (SNAP) which is recorded at the wrist. The latency to the onset of the nerve action potential is measured and compared to a normal range for a set distance or the distance is also measured and converted to a conduction velocity.

The amplitude of the SNAP is also compared to the normal range and also to unaffected nerves in the same upper limb or the contralateral limb. It is often helpful to demonstrate slowing of sensory conduction from more than one digit to confirm an abnormal finding. The median SNAPs can also be compared to another nerve from the same digit. For example, the median SNAP and radial SNAP can be recorded from the thumb and the latencies compared and the median SNAP and ulnar SNAP can be recorded from stimulation of the ring finger and the latencies compared. It is sometimes also helpful to record conduction through short segments of the nerve by stimulating the mixed motor and sensory nerve in the palm of the hand and recording the response from the wrist and this may be particularly helpful in demonstrating focal slowing, although small errors in measuring distance are amplified by this technique. In reporting these findings, it is important to appreciate that increasing sensitivity may trade off specificity[17]. Overall reported sensitivity for sensory nerve conduction studies varies from 44-80% and specificity from 95-100%[9].

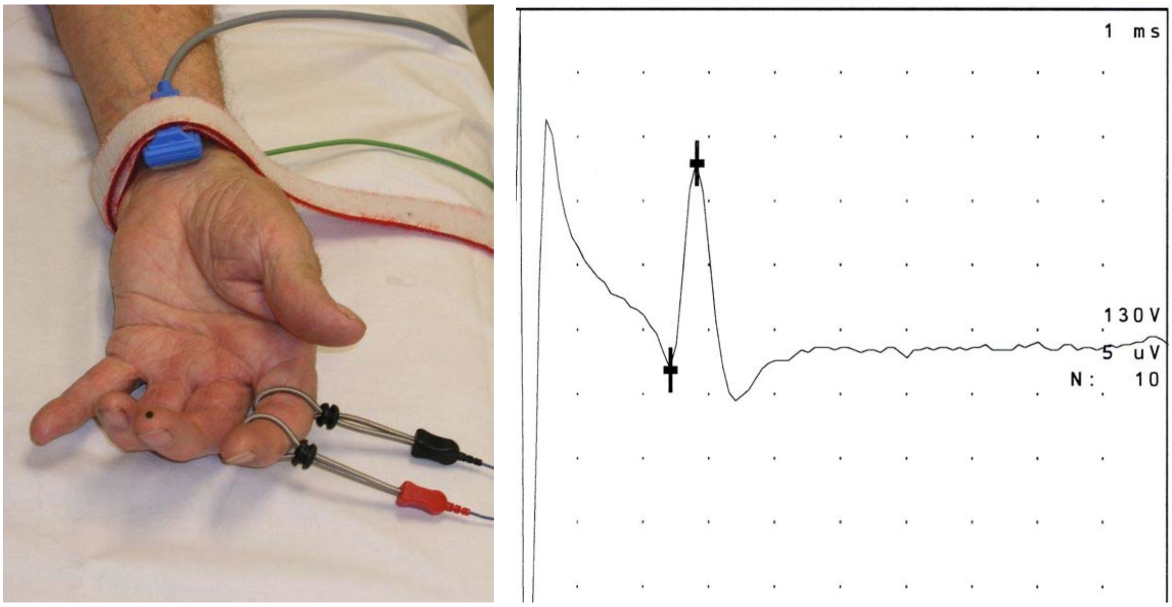


Figure 4 - Sensory nerve conduction tests showing usual position of electrodes and typical results.

The utility of nerve conduction testing

Nerve conduction testing provides a functional assessment of the extent to which the median nerve is being affected by compression.

The findings need to be interpreted in the knowledge that a proportion of individuals with clinically determined carpal tunnel syndrome show no disturbance of nerve conduction. One recent study suggested a false negative rate of 10-25%[18]. It is also possible for electrophysiological features of median nerve compression to be present in individuals with no relevant symptoms[17] and it is not uncommon to find abnormalities in the apparently asymptomatic contralateral hand. The false positive rate in truly asymptomatic individuals is not known.

The diagnosis of carpal tunnel syndrome, which is defined by its clinical features, does not, therefore, depend on an abnormal nerve conduction study. A positive study provides confirmation of the diagnosis in a suitable clinical context. The study also provides a functional assessment of the median nerve by indicating the degree to which the nerve is being affected and possibly damaged by the effects of compression. This may be useful in selecting cases in which early referral for decompression should be considered. On the other hand, a normal nerve conduction study warrants careful reconsideration of diagnosis and treatment options prior to embarking on surgical decompression. Nerve conduction studies may suggest an alternative diagnosis such as a more proximal entrapment of the median nerve or indicate the presence of an underlying generalised neuropathy with or without focal accentuation at sites of compression.

When patients return following surgical decompression of the carpal tunnel but have ongoing symptoms, the absence of a baseline assessment makes it almost impossible to determine whether the surgery has been adequate. Decompression will commonly lead to improvement in nerve conduction studies but frequently the nerve conduction studies will not return to the normal range particularly with more severe findings preoperatively. Given the significant incidence of persistent symptomatology following surgery in occupationally related carpal tunnel syndrome, a baseline nerve conduction study prior to surgery is recommended.

The role of imaging in carpal tunnel syndrome

Imaging in carpal tunnel syndrome can be used both to confirm compression of the median nerve within the carpal tunnel and to diagnose a causative pathology such as a ganglion within the carpal tunnel or flexor tenosynovitis.

Plain radiographs can be used to look for bony deformity and/or mal-alignment at the wrist, bone lesions, arthropathy and soft tissue calcifications.

Ultrasound and magnetic resonance imaging can both be used to exclude a lesion within the carpal tunnel which may be resulting in elevated pressure within the tunnel (eg, ganglion, flexor tenosynovitis, accessory muscles) and to aid in diagnosis. Enlargement of the median nerve at the proximal aspect of the carpal tunnel can be used to diagnose carpal tunnel syndrome[19 20]. The cross-sectional area of the nerve can be accurately measured with ultrasound and MRI with some studies demonstrating comparable sensitivity and specificity to nerve conduction studies[5]. Decreased nerve echogenicity (ultrasound), nerve hypervacularization (ultrasound), increased signal (MRI with T2 weighting), and bowing of the flexor retinaculum may also be evident.

Volar wrist

Carpal T

Median N

Trans

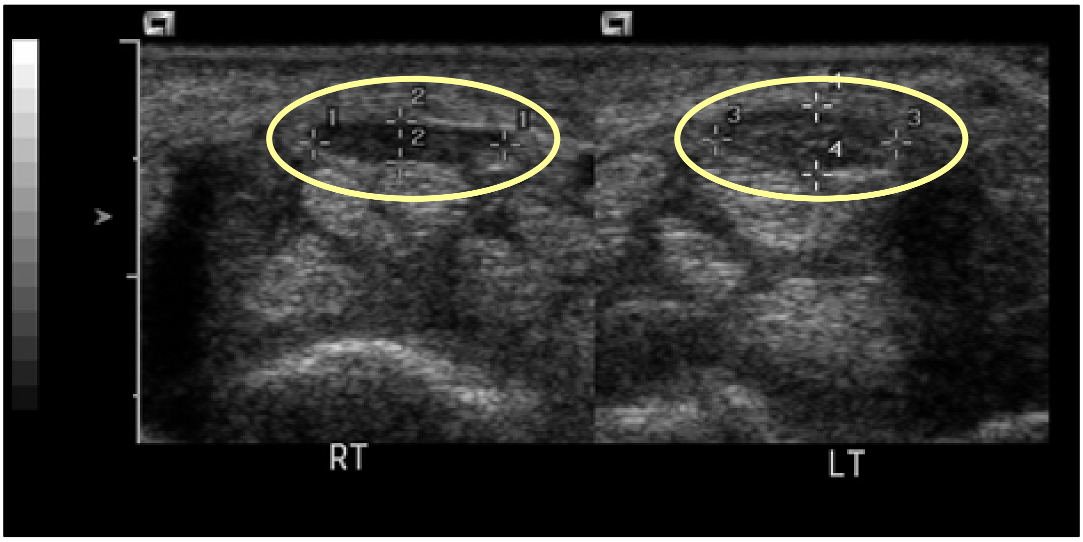


Figure 5 - Ultrasound – Idiopathic carpal tunnel – enlarged left median nerve transverse view

Dynamic assessment with ultrasound can be used to assess median nerve motion with finger flexion and extension (nerve motion may be reduced in CTS) and to assess the degree of lumbrical muscle incursion (which may be a contributing factor in some cases). Dynamic assessment is also valuable post-operatively to assess for incomplete release of the carpal tunnel and tethering of the median nerve to scar tissue. It also enables accurate steroid injection of the carpal tunnel with real-time image guidance.

While ultrasound is operator-dependant it is generally easier for the patient and less expensive than MRI and is the preferred imaging modality in most cases. Recent articles[21 22] suggest that ultrasound may be a cost-effective initial diagnostic alternative to nerve conduction studies in patients with suspected carpal tunnel syndrome. A large-scale, randomized controlled trial is, however, yet to be performed. Ultrasound examination may also be useful in patients with history and examination findings suggesting a possible lesion within the carpal tunnel, or in patients with equivocal or normal nerve conduction studies.

The occupational environment

The clinical signs and symptoms of CTS attributed to work are identical to the clinical signs and symptoms of the condition in the non-occupational environment. One caveat to this is that CTS may present earlier in the occupational environment than in the general population[23].

The difference lies in the recognition of a likely occupational exposure promoting the condition. Although there is some debate in the literature on this point, most authorities agree that certain activities in the workplace can precipitate the development of carpal tunnel syndrome in a predisposed individual. In particular the identification of ergonomic factors such as repetitious activity involving prolonged flexion or twisting in the affected wrist (such as the use of screwdrivers in the flexed wrist position) may be found[7]. Other activities such as those involving exposure to vibration and/or percussion may also predispose to CTS. In a review of a large number of epidemiological studies, the US National Institute for Occupational Health and Safety concluded that there was evidence of an association with highly repetitive work, forceful work, and hand/wrist vibration and the evidence was especially strong for a combination of factors such as force/repetition and force/posture[24]. However in the most recent systemic literature review on the role of occupation in carpal tunnel syndrome by Palmer et al found that the regular use of hand-held vibrating tools increased the risk of CTS two-fold[25]. The authors also found that occupations requiring high-repetitive flexion and extension at the wrist and also forceful grip increased the risk of CTS. The authors did not find any association between CTS and keyboard or computer work.

Recently Kao published a series of questions derived from other studies that may be of assistance to practitioners attempting to determine the work-relatedness of an individual's carpal tunnel syndrome[26]. These questions are intended to be used after the diagnosis of CTS has been confirmed in order to weigh the likelihood of the condition being work-related.

Table 4 – Quick guide to estimating risk of work-related CTS

Ask the patient, "In your current occupation..."

1. Is this the hand you primarily use to perform your current job?
2. Do you bend the wrist up and down or from side to side repeatedly more than twice a minute (wrist flexion/extension, ulnar/radial deviation) or twist/rotate the wrist with palm facing up and then down more than twice a minute (wrist rotation)?
3. Do you have repeated finger-tapping movement more than twice a minute?
4. Do you spend more than four hours per day moving your hand/wrist in the same fashion?
5. Do you grip or hold any object in the palm with a force greater than 6kg while performing the activities listed in questions 2, 3, or 4?
6. Do you hold tools that vibrate during most of your workday?

Scoring: the number of 'Yes' answers is directly proportional to the degree of risk: 0 to 2, low risk for occupational CTS; 3 to 4, moderate risk; 5 to 6, high risk.

Occupational categories with the highest prevalence rates (about 2-3%) are delivery of mail, health-care work, food processing and assembly work in factories, logging and construction work[27].

In contrast to ergonomic work factors, the influences of psychological and psychosocial factors in the development of CTS are relatively poorly studied. The role of job control, and 'just in time' production systems as well as psychological well-being have been reported to have an effect on the prevalence of CTS in some studies[28]. A history of improvement when away from work or when involved in other work activities may be identified, at least in the early stages of the condition. To this end, job rotation may be part of an appropriate management strategy.

The South Australian workers compensation system

The *South Australian Workers Rehabilitation and Compensation Act 1986* (the Act) has broad eligibility criteria under which a person can claim compensation for an injury/disability.

In South Australia, CTS is compensable only in circumstances where work can be shown to be the cause or if work was a contributing factor.

Section 30 of the Act lists that criterion as:

- arising from employment ie, it arises out of or in the course of employment.

If however the disability is an aggravation, acceleration, exacerbation, deterioration or recurrence of a prior disability then the criteria changes to either:

- arising from employment or
- arising in the course of employment and employment contributed.

No maximum or minimum work contribution has been defined in the Act and a decision is based on the balance of probality.

Once eligibility has been established then entitlements follow, this includes compensation for weekly payments, lump sums for permanent disability and for medical investigations, treatment and any other medical services in relation to the injury/disability. The particular service must however, be reasonable in the circumstances (and in accordance with accepted treatment modalities and standards of care) and the costs of the service are determined by regulated fees which are based upon Medicare item numbers.

Management

The management of CTS depends on the severity, chronicity and aetiology of the syndrome in the individual.

Non-surgical management

The following approaches may be considered in the non-surgical management of CTS.

(1) Identify and treat any underlying medical conditions. Diabetes, pregnancy, hypothyroidism, inflammatory arthritis, amyloidosis, Colles' fracture and acromegaly are some of the conditions that may predispose to CTS. Up to one-third of cases of CTS occur in association with such medical conditions. Appropriate management of these conditions may be central to the management of CTS in certain individuals.

(2) Avoid aggravating activities. Intuitively, advising patients to avoid aggravating activities appears prudent, although there have been few studies to confirm this approach. In the work environment this may entail job rotation and/or appropriate rest breaks. In certain situations (with more severe or prolonged symptoms or in work environments where job rotations are not possible) time off work may be required. Naturally, advice regarding the avoidance of aggravating activities extends to the non-occupational home environment and recreational activities.

(3) Splinting. More than 80% of patients with CTS report that a wrist splint alleviates symptoms, generally within days[29]. Splints are more effective if they maintain the wrist in the neutral position rather than in extension. Commercially available splints are acceptable as long as they maintain the wrist in the neutral position. Splints that are light weight and comfortable generally assist compliance. A recent Cochrane review concluded that a hand brace improved symptoms after four weeks[30].

(4) Medication. Non steroidal anti-inflammatory drugs, diuretics, and pyridoxine (B6) have each been studied in small randomised trials, with *no* evidence of efficacy and so are not routinely recommended. In one small study, oral prednisolone at a dose of 20mg per day appeared superior to placebo in short-term symptom reduction[4 30].

(5) Injection therapy. A number of randomised trials have been performed comparing surgical decompression therapy versus injection of steroid into the carpal tunnel[31]. Several of these studies show significant short-to-medium term benefit from the injection treatment[32 33]. At least one study has shown similar outcomes from injection therapy at one year versus surgical decompression[34]. The number of injections considered safe has not been studied. Risks from the injection therapy such as nerve damage or tendon rupture appear low, but have not been formally studied[4].

(6) Other therapies. In one trial involving 51 people, yoga significantly reduced pain after eight weeks compared with wrist splinting. Carpal bone mobilization showed symptomatic improvement in one small trial compared with placebo. One trial showed improvement with ultrasonic treatment though two other trials did not. Trials of magnet therapy, laser acupuncture, exercise, and chiropractic therapy have not shown significant benefit over control[30].

(7) Yellow flags (see the TREAT[35] section of the WorkCover website, www.workcover.com > Health providers > TREAT) or psychosocial, workplace and other factors that increase the risk of developing or perpetuating long term disability also need to be considered. Action is warranted if the clinical situation, including the ability to perform suitable duties, is deteriorating. Similarly, action is required when there is a delay in expected improvement. These actions are implemented to prevent the dire circumstances that occur with poorer than expected health outcomes and the resultant disability.

Surgical management

Surgical decompression remains an important treatment option for CTS, especially for more severe cases. In the South Australian workers compensation system it would be usual to seek collaborating specialist opinion on the diagnosis and the likely effectiveness of any surgery, particularly if NCS do not confirm the diagnosis. In this context, severe cases are considered to be those which have:

- rapidly progressive neurological signs
- significant symptoms
- functionality limitations
- muscle weakness
- failing conservative therapy.

Certainly if there are clinical signs and symptoms to suggest axonal loss (constant numbness, symptoms for more that one year, loss of sensibility, thenar muscle atrophy or weakness) or evidence of axonal loss on NCS, then surgery should be seriously considered. Some guidelines suggest an attempt at conservative treatment for two to six weeks and then specialist referral for consideration of surgical decompression if the condition is not settling.[36]

In a recent Cochrane review, the relative merits of endoscopic versus open surgery were compared. The review concluded that with the possible exception of quicker recovery after endoscopic surgery, there was no significant difference in outcome between the two approaches. Overall, more than 70% of patients report being completely satisfied or very satisfied with the results of carpal tunnel surgery[37] and therefore post-surgical investigations are not routinely required.

Surgical decompression results in a 2-15% complication rate. Post-operative problems include failure to relieve symptoms, incomplete release, recurrence due to the repair of the transverse carpal ligament, injury to the median nerve or its branches, persistent pain in the proximal palm, hypersensitivity, neuroma formation weak grip, tendon adhesions, finger and joint stiffness, volar bowstringing of the flexor tendons, and reflex sympathetic dystrophy (complex regional pain syndrome)[38].

Rehabilitation following surgical management

One potential advantage to endoscopic release versus open decompression is the potential for earlier return to work. Following carpal tunnel release, pain relief occurs within days, but hand strength does not reach preoperative levels for several months[39]. Tenderness of the surgical scar may persist for up to a year after open release[37]. Patients with better general functional status and mental health have more favourable outcomes after carpal tunnel release[40].

In the workers compensation environment, legal representation is associated with poorer patient outcomes but this may simply reflect the complex variety of reasons that legal advice is sought[40]. Paradoxically, those workers with less strikingly abnormal electro-diagnostic studies tend to have worse outcomes[41]. At least one study has reported that supportive work organizations have an impact on the successful medical rehabilitation of workers following carpal tunnel release[42]. Poor self-efficacy and depression are also negative predictors of a successful return to work[43]. Accurate and earlier diagnosis had a positive effect on long-term outcome in one follow-up study[44].

With appropriate management the expectation for the long-term outlook for CTS is complete recovery in the majority of cases. For uncomplicated CTS, follow-up studies show a return to work rate of 64-90% following treatment, with the majority returning to their normal duties[45 46]. The results do, however, demonstrate that a significant proportion of cases do not return to work and this represents a particularly disappointing outcome from what is generally perceived to be a minor procedure for a readily treatable condition.

Time to return to work following surgery varies widely, with the most significant factor determining return to work being the surgeon's recommendations[47]. In one recent study, over 70% of people returned to work within 30 days of surgery, despite only 16% having regained full pre-operative function with regard to grip strength at that time. In another recent survey of patient recovery from open carpal tunnel release return to driving took nine days on average, and return to work took 17 days on average. There was no difference between those patients who has unilateral versus bilateral releases[48]. Some studies also report patients exposed to higher levels of bending and twisting of their hands and wrists were slower to return to work after carpal tunnel release surgery. It is reasonable to note that no negative effects of work on recovery from carpal tunnel release have been found.[36]

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