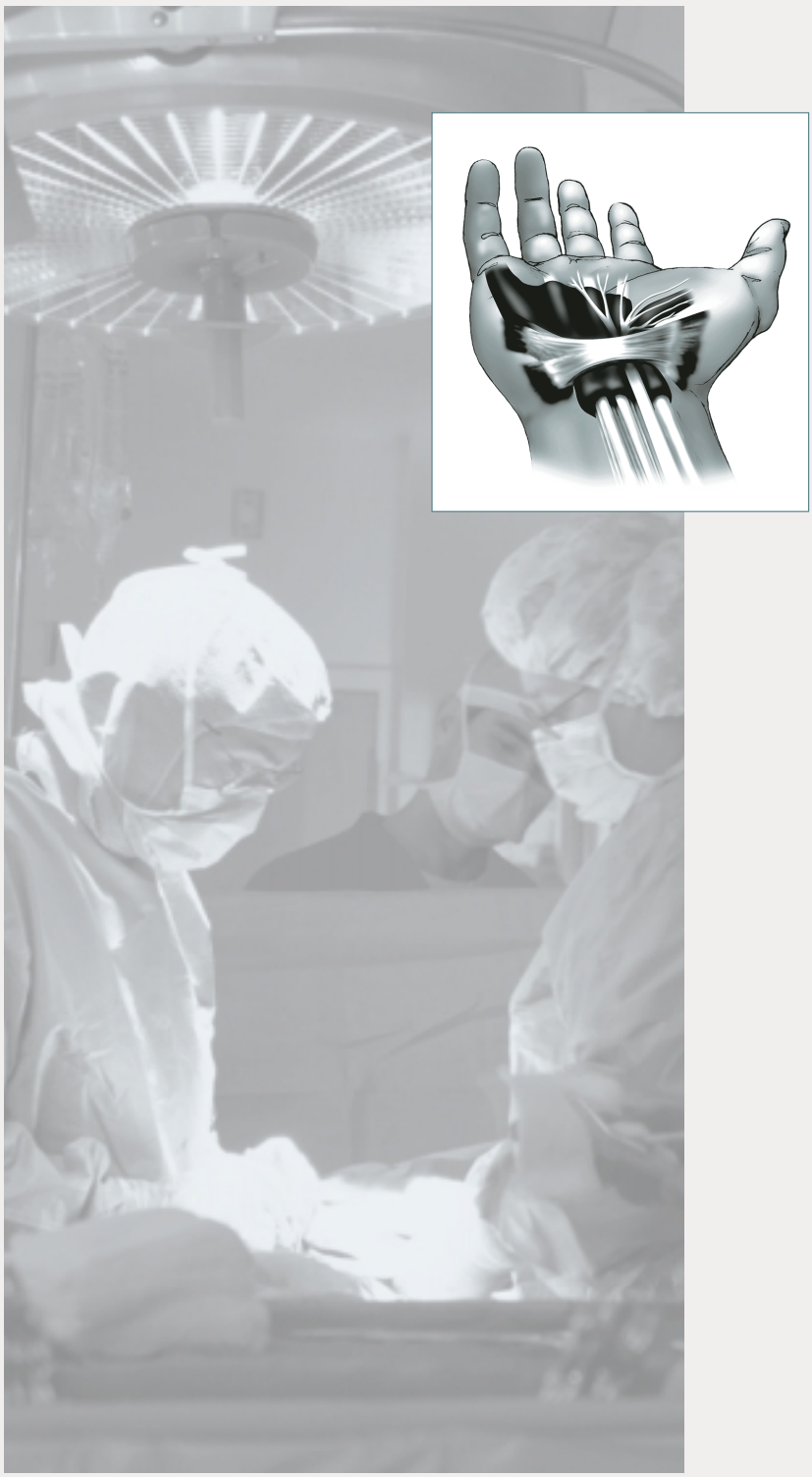
A CLINICAL MONOGRAPH SERIES

Carpal Tunnel Syndrome

written by JAMES W. STRICKLAND, MD



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ABOUT THE AUTHOR

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Dr. Strickland is one of the world's leading authorities in hand and orthopaedic surgery. He is Clinical Professor of Orthopaedic Surgery, Indiana University School of Medicine. He has served as the Chairman of the Department of Hand Surgery at St. Vincent Hospital, and he founded The Indiana Hand Center, a national leader in upper extremity care. Dr. Strickland has served as President of the American Society for Surgery of the Hand and as President of the American Academy of Orthopaedic Surgeons. Dr. Strickland has also served on the executive board of the Academic Orthopaedic Society, the board of directors of the American Foundation for Surgery of the Hand, and is a member of the American Orthopaedic Association.

Dr. Strickland received his medical degree from the Indiana University School of Medicine and completed his orthopaedic surgery residency after serving as a captain in the Air Force. He completed a fellowship in hand surgery at Northwestern University in Chicago. He specializes in surgery of the hand and upper extremity with particular interests in nerve compression disorders, cerebral palsy and head injury, Dupuytren's disease, tendon repair and reconstruction, and restoration following arthritic conditions.

Dr. Strickland has written or edited nine hand surgery books, published over 170 scientific articles, book chapters and commentary and has given over 650 papers and presentations at local, national and international courses and conferences. He has been a visiting professor for 60 different medical schools or training programs and has given over 50 named or distinguished lectureships.

INTRODUCTION

Carpal tunnel syndrome (CTS) is a large and growing problem in the United States. Data from the National Center for Health Statistics indicates that 849,000 new problem visits were made to physicians in office-based practice in 1994 because of median nerve pathology at the wrist. Women are more frequently affected with median neuropathy in the carpal tunnel than are men and the mean age of diagnosis is about 50 years. Approximately 260,000 carpal tunnel release operations are being performed each year.[1]

In recent years, the condition has become a leading cause of on-the-job injuries. The Bureau of Labor Statistics indicates that in 1994, carpal tunnel syndrome accounted for 1.7 percent of workplace-related conditions in private industry that resulted in lost work and the disorder results in the highest median number of days of work lost (30 days) among all major work-related injury or illness categories. The common task related factors that seem to contribute to the development of carpal tunnel syndrome include repetitiveness, force, mechanical stress, posture, vibration and temperature but these factors are variable and inconsistent and the mechanisms by which they produce neuropathy are not well appreciated.

ANATOMY

The carpal tunnel is a space in the proximal palm, (Fig. 1). A concave arch of carpal bones that are covered by the extrinsic palmar wrist ligaments forms the floor. The roof is made up of the transverse carpal ligament, which attaches radially to the scaphoid tuberosity and the crest of the trapezium and ulnarly to the pisiform and the hook of the hamate. It is a conduit for the median nerve and nine digital flexor tendons from the forearm into the palm, (Fig. 2).

Although not a closed compartment, the carpal tunnel has been called a closed space.[2] Because of the anatomy, any pathological process that reduces capacity or increases the volume tends to increase interstitial pressure within the carpal canal. This, in turn, can lead to compression of the median nerve.[3] In addition, the anterior position of the median nerve as it passes directly under the rigid transverse carpal ligament renders it vulnerable to direct pressure from the flexor tendons.[4]

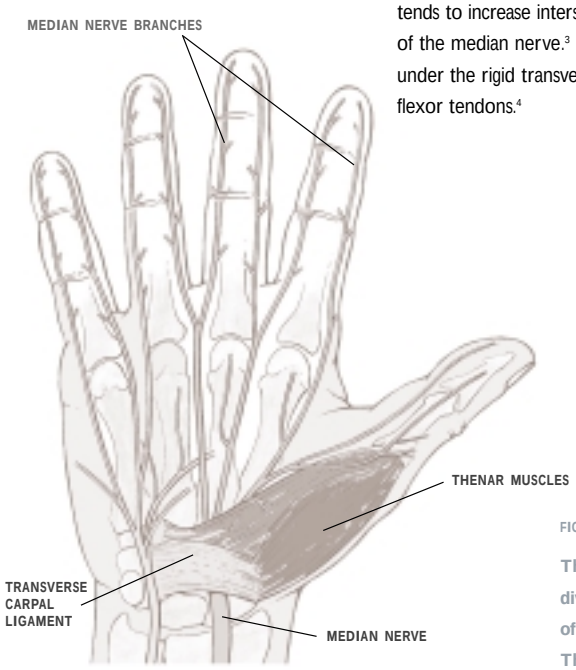


FIGURE 1: ANATOMIC VIEW OF THE ANATOMY OF THE PALMAR WRIST, PALM AND DIGITS.

The median nerve passes beneath the transverse carpal ligament and divides into branches that will provide sensation to the palmar surface of the thumb, and the index, middle, and radial one-half of the ring fingers. The motor branch curves to enter and innervate the thenar muscles.

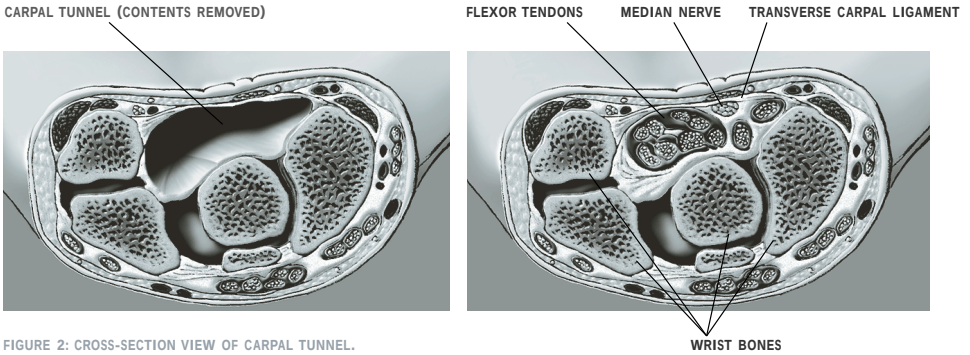


FIGURE 2: CROSS-SECTION VIEW OF CARPAL TUNNEL.

The passageway is bounded by the carpal bones on three sides and the transverse carpal ligament on the palmar side. Its contents include nine flexor tendons; two each to the index, middle, ring and small fingers and one to the thumb. The tendons are covered by synovium which provides nourishment and facilitates gliding. The median nerve is directly beneath the ligament and is compressed against it when there is an increased tissue volume within the passageway.

SYMPTOMS

Compression of the median nerve at the wrist results in irritation that is known as the clinical disorder carpal tunnel syndrome.[5] Major symptoms include pain in the wrist and hand that radiate to the forearm and paresthesias in the thumb, index, middle and radial half of the ring fingers. Nocturnal symptoms may be particularly severe and it is common for affected patients to awaken with numbness or tingling in their fingers that requires shaking the hand or other positional changes in order to gain a measure of relief. Discomfort, numbness or tingling in the median nerve distribution may also occur with repetitious activities such as using a keyboard or assembly line work or with prolonged flexion or extension of the wrist such as driving an automobile. Advanced stages of median nerve compression can result in thenar muscle weakness,[6,7] and patients may tend to drop objects as the weakness becomes more pronounced. Atrophy of the thenar muscles and demonstrable sensory changes are seen in severe, long-standing median nerve compression.

CAUSES

At its most basic level, any process that reduces the capacity or increases the contents of the carpal canal can lead to higher interstitial pressure and compression of the median nerve.[2] Since the anterior portion of the median nerve lies directly under the transverse carpal ligament, there is also an increased vulnerability to direct pressure from the flexor tendons.[3]

IDIOPATHIC CTS

Kerwin and colleagues suggested classifying the causes of CTS as idiopathic, intrinsic or extrinsic.[8] Idiopathic CTS (ICTS) has a different clinical presentation than other forms. As noted by Phalen, it occurs in healthy adults, more frequently in women, has an older onset (40-60 years of age) and may be bilateral.[9] Early studies indicated that chronic tenosynovitis might be responsible for increased volume in the carpal tunnel. Newer research, however, has called that into question. Fuchs took tenosynovial biopsy specimens from 177 wrists undergoing carpal tunnel release procedures and a control group of 19. They found that inflammation was present in only 10% of the patient specimens. Thus, true tenosynovitis is "uncommon" in those with idiopathic CTS undergoing carpal release surgery.[10]

Nakamichi and Tachibana found similar results. Histology of the transverse carpal ligament and flexor tenosynovium was investigated in 166 wrists from 130 patients with idiopathic CTS. Nine control wrists were used for comparison. Consistent with Fuchs, the tenosynovium showed inflammation in 10.2% of the cases with 65% showing no histological changes.

They also noted that 73.5% of the ligaments showed normal morphology and histology. They concluded that in ICTS both the ligament and tenosynovium are usually normal and that there were no typical changes that could be associated with the syndrome.[11]

It has also been noted that variations in the tunnel's diameter occur in the normal population. Studies by Dekel and Papaioannou have used computed tomography (CT) studies to demonstrate that patients with CTS have smaller carpal canals than the normal population. Their findings may account for increased prevalence of CTS in women, although the role, if any, of this anatomic phenomenon in CTS is still not clear.[12]

INTRINSIC CTS

Intrinsic CTS (InCTS) was defined by Kerwin as being from "factors that increase the volume of the contents of the carpal tunnel…" and lead to increases in interstitial pressure and a typical series of pathophysiologic events that leads to the clinical symptoms of CTS. For example, increased retention of body fluids has been implicated in the development of the syndrome during pregnancy as the increased volume within the passageway compresses the median nerve. The majority of patients become symptomatic during the third trimester and symptoms often resolve following delivery.[13,3]

Chronic hemodialysis patients also have a high incidence of median nerve related neuropathy. Hirasawa and Ogura examined 110 patients with CTS and chronic renal failure requiring dialysis. They found a significant relationship between the incidence of CTS and duration of dialysis treatment.[14] Suggested mechanisms for the development of CTS in hemodialysis patients vary from elevations in canal pressure secondary to increases in body water to distal stenosis or a vascular steal phenomenon related to the required vascular shunt.[15] Longer-term, the higher incidence of median nerve compression has been attributed to a microglobulin amyloid deposition that increases as renal function is lost.[16]

CTS is also seen in patients with hypothyroidism. The etiology is thought to be the accumulation of myxedemateous tissue under the transverse carpal ligament.[17] Inflammatory conditions such as rheumatoid arthritis (RA) and gout have also been tied to increased incidence of CTS. Solomon and colleagues published a case-controlled study of New Jersey Medicare or Medicaid enrollees looking at risk factors for carpal tunnel release procedures. Inflammatory arthritis was strongly associated with release surgery with an odds ratio of 2.9.[18] Hypertrophic flexor tenosynovitis distending the parathenon-covered flexor tendons may be responsible for compression of the median nerve. Radiocarpal subluxation and joint deformity may also play a part in the development of carpal tunnel syndrome.[19,3]

EXTRINSIC CTS

One of the extrinsic factors (ECTS), according to Kerwin, is change in the dimensions of the carpal arch or canal that may result in increased interstitial pressure since the volume of the contents are unchanged. For example, CTS has been described in association with wrist fractures, scaphoid non-union, rotary subluxation of the scaphoid, or wrist arthritis.[20] Diabetes, hemophilia and myeloma have also been linked to CTS. Solomon found a weak but significant association between diabetes and carpal tunnel release in his study of New Jersey Medicare and Medicaid patients (OR 1.7).[17] Dell and colleagues found that about 17% of patients with CTS also had a history of diabetes.[18] As of now, the underlying pathology is not well understood. The causes for increased CTS found among those with hemophilia is similarly unsettled. Nerve compression from a surrounding hematoma, intramuscular hemorrhage or ischemia from hemorrhage within the nerve itself, have all been suggested.[21]

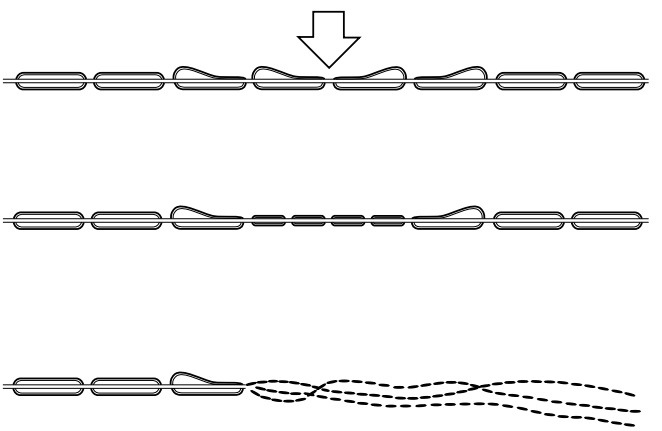
OCCUPATIONAL FACTORS

To say that occupational causes of CTS are controversial is almost an understatement. Some studies show that workers engaged in repetitive flexion and extension of the wrist, strong grip or exposure to vibration are at greatest risk.

Tanaka, et al estimated the prevalence of self-reported CTS among workers using the Occupational Health Supplement of the 1988 National Health Interview Survey (NHIS). Among the 127 million people who had worked during the 12 months prior to the survey, 1.87 million self-reported CTS and an additional 675,000 stated prolonged hand discomfort that had been called CTS by a medical person. The risk factor in this group most strongly associated with medically confirmed CTS was exposure to repetitive bending/twisting of hands or wrists at work (OR=5.2).[22]

Others have argued that the condition would be seen more frequently if it were truly an occupational disease. Nathan and colleagues looked at 471 employees from 27 occupations in four industries. They evaluated the role of occupational hand activity as a risk factor for CTS using a slowing of sensory conduction of the median nerve at the carpal tunnel. They found no consistent association between the type and level of hand activity and the prevalence or severity of the slowing.[23] Their follow-up study over the years from 1985 to 1989 noted that slowing was still highly correlated to increasing age and that slowing was no longer correlated to occupational hand use in any fashion.[24]

FIGURE 3: PATHOLOGY OF CHRONIC MEDIAN NERVE ENTRAPMENT AT THE WRIST.



(A) Myelin internodes are damaged and distorted proximal and distal to the site of compression.

(B) With progressive demyelination, the axon becomes exposed. Schwann cells reform myelin internodes in an effort to repair the lesion.

(C) In advanced cases, the axon undergoes Wallerian degeneration and regeneration of new axons may be initiated from growth cones above the site of the injury.

PATHOPHYSIOLOGY

The pathophysiological mechanism of CTS is the same irrespective of the duration or severity of the symptoms. In normal hands, the average interstitial pressure within the tunnel is 2.5 mm Hg with maximum pressure elevations in wrist extension or flexion well below the 32 mm Hg average capillary refill pressures.[25] Any increase in pressure within the tunnel may result in mechanical distortion of the myelin sheath or ischemia of the median nerve.

One of the hallmarks of chronic compressive neuropathies such as CTS, is demyelination. The loss of myelin, which is critical to normal conduction of neural impulses, appears to result primarily from mechanical disruption of the internodal segments. The myelin segments form ovoid shapes that are distorted and damaged proximal and distal to the site of compression.[26] Notably, if the compressive insult is relieved, then Schwann cells will remyelinate the axon and may restore near normal conduction. Extensive demyelination and persistent compression eventually result in direct axonal damage and Wallerian degeneration distal to the site of injury.[27] Under these circumstances, recovery of function requires the more complex and lengthy process of axon regeneration, (Fig. 3).

Obstruction of venous return in the epineural or perineural vascular plexuses appears to result in anoxia and endoneural edema of the nerve.[28] The magnitude of edema formation and subsequent nerve conduction blockage is related to the amount and duration of the compression.[29] It can also lead to venous congestion, hyperemia and circulatory slowing.[30]

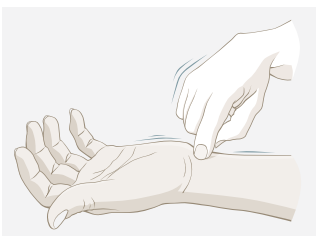
As the pressure become higher and/or more sustained, swelling of the nerve bundles can occur within the endoneurium related to the accumulation of exudates and edema. In addition, endoneural edema itself interferes with nerve function due to alterations in the local ionic environment of the axons.[31] There are also data indicating that increased canal interstitial pressure has a direct mechanical effect on axonal transport. Experimental outcomes suggest that persistent compression at 20 mm Hg results in a reduction of orthograde fast axonal transport with reductions in orthograde slow transport at 30 mm Hg.[32] The longer the pressure increases are allowed to continue, the more likely that the disturbances in blood flow and axonal transport will lead to permanent changes.

Destruction of the epineurium and endoneurium with a dense, fibrous scar tissue may be the final result of prolonged compression.[33]

DIAGNOSIS

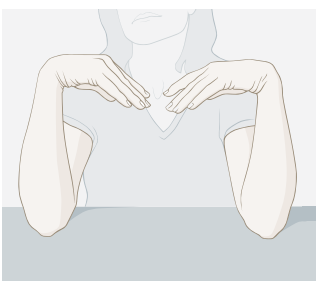
CLINICAL SYMPTOMS AND PHYSICAL FINDINGS

FIGURE 4: TINEL'S SIGN



A Tinel's tapping sign or Tinel's test is elicited by tapping directly over the median nerve in the midline of the wrist. Tingling or "electric shocks" radiating into the fingers is interpreted as being positive for median nerve embarrassment.

FIGURE 5: PHALEN'S SIGN



The patient is asked to place their elbows on the examination table and allow the wrists to fall into full flexion with the fingers relaxed. Numbness or tingling experienced into the median nerve distribution within one minute is considered a positive test.

Diagnosis of CTS is based on a combination of clinical signs, symptoms and abnormal nerve conduction and electromyographical studies. The condition must be differentiated from more proximal embarrassment of the median nerve such as cervical radiculopathy or median nerve compression at the elbow or in the proximal forearm, a condition known as "pronator syndrome".

The classical symptoms of CTS are pain, numbness or paresthesia in the thumb, index, middle and radial one-half of the ring fingers. The palmar cutaneous branch of the median nerve is spared since it branches off several centimeters proximal to the carpal tunnel. Wrist pain, digital weakness, inability to pinch strongly and frequent dropping of objects are also common complaints particularly when the condition has been present for many months. Aching in the forearm or even the proximal arm and shoulder are frequently reported in association with digital numbness and tingling. Typically, the symptoms are worse at night and are aggravated by repetitive tasks, as well as by wrist extension or flexion.

Two clinical maneuvers that have been widely used during physical examination are Phalen's test and Tinel's sign. The simplest such test is referred to as "Tinel's tapping test" or "Tinel's sign". It involves tapping lightly with a rubber mallet or the examiner's index finger directly over the median nerve proximal to the middle of the wrist crease. A tingling sensation radiating to the tips of the thumb or any of the first three fingers indicates the possibility of embarrassment of the median nerve in the carpal tunnel, (Fig. 4). Phalen's wrist flexion test is also commonly employed in making a diagnosis of CTS. For this provocative maneuver, the patient allows the wrists to fall into full flexion letting the fingers dangle downward. If a tingling sensation in the median nerve distribution starts in less than a minute, it is considered a positive sign for the presence of carpal tunnel syndrome, (Fig. 5).

Although these test are commonly used by most physicians diagnosing CTS, a review of the literature indicates that there are very wide reported differences in their specificity and sensitivity for the accurate detection of CTS. Gerr and Letz, in their review article, found that estimates of sensitivity for Phalen's test range from 10% to 88% and of Tinel's sign from 26% to 79%. Similar variations were found in the assessments of specificity for both tests.[34] Basing diagnostic decisions solely on these signs and symptoms can lead to confusion with other common disorders that have similar presentations, such as tendonitis and cervical radiculopathy.[35] However, Herbert and his group note that clinical symptoms and these two tests may "have some limited utility for improvement of the positive predictive value of clinical evaluation when electrodiagnostic studies are not available".[36]

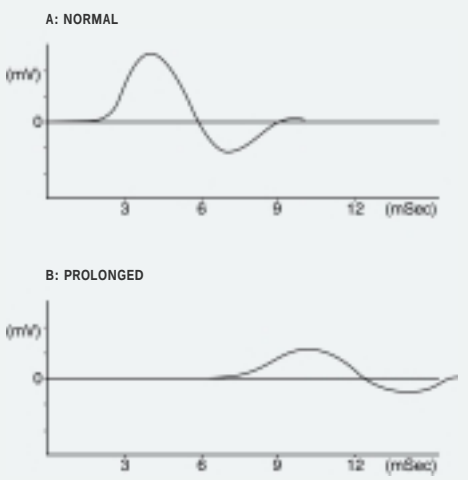
NEURODIAGNOSTIC METHODS

Although there remains some controversy over which electrodiagnostic methods are the "gold standard" for diagnosis of CTS, nerve conduction studies are widely regarded as the principle objective tool and can be useful in clinical staging. Simpson was the first to show that focal slowing of the median nerve at the wrist was associated with CTS in 1956.[37] Nerve conduction studies involve stimulating the peripheral nerves and recording the evoked response from the muscle (motor conduction) or nerve (sensory conduction). Measurements of conduction times, amplitude, duration and configuration of the compound motor action potential (CMAP) or sensory nerve action potential (SNAP) are clinically valuable in the assessment of peripheral nerve function.[38] It has also been shown that there are many factors that need to be taken into consideration when conducting and interpreting electrodiagnostic tests. Age, height, skin temperature, and methodological variations have all been found to impact on normative values.[39]

In motor conduction studies, nerves are stimulated supramaximally, causing the simultaneous depolarization of all the large fiber, myelinated axons. The resultant action potential travels orthodromically down the nerve, evokes synaptic transmission at the neuromuscular junction and results in a CMAP or M-wave. The time (in milliseconds) it takes for the impulse to travel from stimulation point to the motor point (region of muscle innervated by nerve) is defined as the distal motor latency (DML). The stimulation impulse simultaneously propagates antidromically, induces back-firing in a subset of motor neurons within the anterior horn of the spinal cord, which then generates a second impulse that travels back to the innervated muscle resulting in an F-wave. F-waves are low amplitude signals that occur much later than M-waves; they provide useful information about the proximal segments of the nerve, including the nerve roots.

While motor latency measurements are generally considered the most important metric, the shape of the M-wave can also provide valuable information. The area and amplitude of the motor action potential is correlated with the number of functioning fibers. Atrophy of muscle fibers or degeneration of nerve fibers will result in a lower amplitude M wave. In addition, increased duration of the M wave indicates an expansion in the range of conduction velocities, (Fig 6).

FIGURE 6: MEDIAN NERVE COMPOUND MOTOR ACTION POTENTIALS.



Distal motor latency provides objective evidence to differentiate normal median nerve conduction (A) from median nerve compression at the wrist (B). Note that prolonged duration of the CMAP (B) reflects an expansion in the range of conduction velocities frequently associated with severe entrapment.

Sensory nerve conduction may be measured by stimulating a mixed nerve proximally (e.g. wrist) and recording at a distal site (e.g. a digit) where only sensory axons are present (antidromic). It can also be measured by stimulating a distal site (e.g. digit or palm) and recording the results proximally (orthodromic). The resulting SNAP is the sum of thousands of action potentials of the myelinated sensory fibers within the nerve. Sensory latency is measured as the transmission along a predetermined length of nerve and may be reported as a latency or conduction velocity.

Electromyography (EMG), involves the insertion of a needle electrode into a muscle to record its electrical activity. It is useful in identifying muscle membrane instability, changes in amplitude, duration or shape of the motor unit action potential and changes in numbers of rates and voluntary recruitment of those motor units.[40] It may be useful in defining the severity of a lesion, distinguishing CTS from proximal median nerve entrapment and cervical radiculopathies. In straight forward carpal tunnel syndrome with classic symptoms, clinical findings, and positive nerve conduction studies, EMG tests may not be warranted because they are painful, expensive and unlikely to appreciably contribute to the diagnosis.

NEURODIAGNOSTICS AND CTS

Electrodiagnostic tests have many uses in the evaluation and management of CTS patients. They have been suggested for use in diagnosis, staging and, more recently, as a possible method of predicting surgical outcomes. There are also studies ongoing to address methods that are useful in screening populations with higher risks of CTS. Stevens, in his 1997 review of electrodiagnosis of CTS suggested median and ulnar studies should be performed on all patients. The ulnar motor tests will help sort out those patients with polyneuropathies. He also advocates comparison of the median and ulnar orthodromic latencies. When these tests are abnormal in one limb or the symptoms are bilateral, median sensory tests should be done on the opposite side, with median motor follow-up considered if CTS is found again.[41]

Sanders and colleagues described two methods for median-to-ulnar motor conduction comparison in the diagnosis of CTS. They looked at the median-thenar to ulnar-thenar latency difference (TTLD) and the median-thenar to ulnar-hypothenar latency difference (THLD). In patients with clinically defined CTS, the diagnostic sensitivities were 95-98% and 85-88% respectively. These tests are sensitive, easily performed and can be added to current routines with few problems.[42]

You and coworkers examined the severity of symptoms in relation to nerve conduction measures of the median nerve. They evaluated 64 hands in 45 patients with CTS. Using a symptom severity questionnaire, six typical symptoms (pain, weakness, clumsiness, numbness, tingling and nocturnal symptoms) were assessed for magnitude, frequency or duration of the episode. Their analysis found that the symptoms could be classified as primary (numbness, tingling and nocturnal symptoms) and secondary (pain, weakness and clumsiness). Primary symptoms are considered to be more specific for nerve injury and secondary more commonly found in soft-tissue injuries. There were also significant relationships between the overall symptom scale and median motor and sensory nerve conduction measurements. In addition, there were indications that the severity scale for primary symptoms was more closely related to the nerve conduction measures than were the secondary ones.[43]

OUTCOMES STUDIES

Various nerve conduction studies have also been shown to have use in correlating pre-operative studies with surgical treatment and outcomes. Harris looked retrospectively at 124 hands (101 patients) with CTS confirmed by nerve conduction studies who went on to carpal release and were then followed for a minimum of six months after surgery. They found that those with greater prolongation of nerve conduction time had better results than those with less severe changes. When post-operative nerve conduction results were available, it was noted in every instance that, even where there was rapid subjective improvement, a lag in resolving of abnormal conduction studies suggests much slower actual repair of nerve damage.[44]

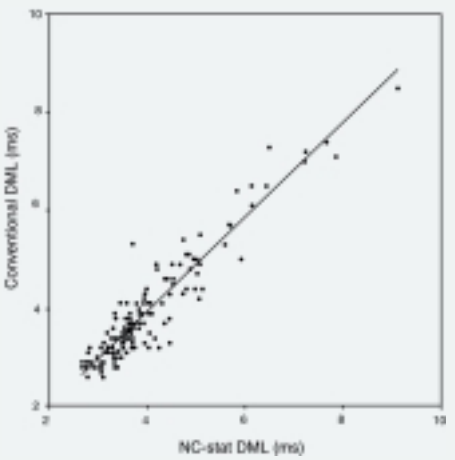
More recently, Higgs and colleagues enrolled 93 workers having undergone carpal tunnel surgery. They were followed from 16 to 100 months. Significant differences were found in pre-operative nerve conduction values between groups reporting poor results and those reporting good results. Their data indicated that those with terminal latencies of 1 ms greater than the norm for that testing facility or with sensory conduction velocities 10 m/s less than the facility norm were more likely to benefit from surgery. They suggested caution in performing surgery on those with normal or near normal nerve-conduction studies.[45]

Research is beginning to accumulate on the use of various nerve conduction studies in predicting the future development of CTS. Nathan and his group completed a follow-up of their initial study that began in 1984 of 942 hands in 471 randomly selected workers. This cohort was visited again in 1989, and 1994-95. The last group included 578 hands, about 92% of the original. The authors excluded those who had undergone release surgery since the procedure interfered with the natural history of the disease process. They found that the overall trend was for the mean sensory latency and prevalence of slowing to increase, the prevalence of symptoms to decrease and the prevalence of CTS to remain unchanged over the period. There also was a strong, direct linear correlation between initial slowing and development of CTS.

However, most of those workers who developed de novo slowing did not go on to develop CTS or show symptoms. They concluded that the changes in conduction in the median nerve were normal for increasing age and did not necessarily lead to symptoms or CTS.[46]

Continuing results from a long-term study by Werner and others lend credence to the possibility of using latency studies as a screening predictor for the development of CTS. They prospectively involved 77 workers who were asymptomatic but had electrodiagnostic findings consistent with median mononeuropathy. They were compared to an age- and sex-matched control group. Follow-up was completed an average of 70 months later with a rate of 70%. Among subjects with abnormal median sensory latencies, 23% went on to develop CTS during the observation period, compared with only 6% in the control group. After about 6 years, there was an increased risk of CTS if the worker had an abnormal early finding.[47]

FIGURE 7: SCATTER PLOT COMPARING CONVENTIONAL NERVE CONDUCTION TECHNIQUES AND THE NC-STAT METHOD.



The correlation between the median nerve DML determined by traditional techniques and the DML determined by NC-stat method was 0.94 (P<0.001).

OFFICE BASED NEURODIAGNOSTIC METHODS

Electrodiagnostic studies have demonstrated their value in detecting CTS and are emerging as useful methods for work place screening. They are however inconvenient and uncomfortable for patients and, when multiple nerves and extremity locations are tested, there can be considerable expense to the patient and health care payor. It can be reasonably argued that accurate and robust motor or sensory conduction evaluation of the median nerve at the wrist is adequate for the majority of patients with classic carpal tunnel syndrome symptoms and clinical findings. Ideally the tests should be non-invasive, available at the initial point of care, and modestly priced. Such systems would save patients the inconvenience of multiple appointments in physician offices and electrodiagnostic facilities, lessen or eliminate the discomfort of the testing, and reduce the overall cost for diagnosis and treatment.

One of the new techniques that may address these issues is an automated electrodiagnostic device (AEND), the NC-stat® system from NeuroMetrix. The NC-stat system consists of a battery operated hand-held monitor, a preconfigured disposable biosensor and a docking station that connects to an information management system to generate a highly informative hard copy report. The device automatically identifies the maximal stimulus intensity, delivers a series of stimuli and calculates the DML and F-wave latency. The test can be performed quickly by a nurse or technician, and the report is returned by fax within 10 minutes.

To assess the reliability of the AEND, Leffler studied two groups of 75 consecutive patients each (an initial group and a validation group) who were referred to an academic electrodiagnostic laboratory (Massachusetts General Hospital) for evaluation of upper extremity complaints. The research standard for diagnosis of median neuropathy at the wrist was the neurologist's diagnosis after formal clinical and electrodiagnostic evaluation with the diagnostician being blinded to the results of the AEND studies. In the validation group, the AEND yielded a DML in 97% of the hands with a conventional motor response and the correlation of the AEND DML with the conventional DML was 0.94, significant at $p<0.001$, (Fig.7).

The sensitivity at 90% specificity improved from 40% using the clinical model to 86% for the model that also included the AEND DML. Using receiver operating characteristic curve (ROC) analysis, the diagnostic accuracy of an evaluation based on the NC-stat method was determined to be 0.96. Compared with a model based solely on clinical variables, an algorithm including symptoms plus the AEND DML had an odds ratio of correct diagnostic classification of 6.3. The authors concluded that there was a significant improvement in diagnosis using the AEND.[48]

CONSERVATIVE MANAGEMENT

Although rarely mentioned in the literature, rest may be enough for a select group of patients with a recent onset of symptoms or for those whose symptoms tend to be transitory. As was noted by Futami and colleagues, approximately one of every three patients has resolution of their symptoms within five months, even without treatment.[49] Splinting remains the first line conservative treatment in CTS. It is most effective if applied quickly, usually within three months of symptom onset. Splinting the wrist in a neutral position serves to maximize space in the tunnel and minimize compression on the median nerve.[50] Kuo used ultrasound to determine the wrist angle that produces the least compression to the median nerve. They studied 17 wrists of 17 healthy volunteers who received dynamic, high-frequency (8 MHz), high-resolution sonography with the wrist splinted at 15 degrees of flexion, neutral position, and 15 degrees and 30 degrees of extension. The neutral position caused significantly lower compression of the median nerve.[51]

Walker and colleagues recruited 21 outpatients (30 hands) with untreated CTS from a Veterans' Administration Medical Center electrodiagnostic laboratory. They were given custom-molded neutral wrist splints and randomized to wearing them either full time or only at night. Despite compliance issues in both groups leading to a tendency for treatment crossovers, those assigned to the full time group still showed superior distal latency improvement in both motor and sensory nerve conduction. These outcomes lead the researchers to conclude that there was support for both neutral wrist splints and their use full time in treating CTS.[52]

Steroid injection has also been found to have some efficacy in the non-surgical treatment of CTS. Recently Dammers et al conducted a randomized, double blind, placebo controlled trial to assess the effect of 40 mg of methylprednisone injected proximal to the carpal tunnel. Participants were given either 10 mg of lidocaine or the same dose of lidocaine combined with 40 mg of methyl-prednisone. Non-responders to lidocaine only received the combined treatment in an open-study that followed. At one-month, 20% of 30 patients in the control group had improved compared with 77% of 30 in the intervention group. At one year, 2 of 6 improved patients in the control group did not require additional treatment, compared with 15 of 23 in the intervention group. Of the 28 who initially did not respond, 24 (86%) improved after methylprednisone treatment. The authors concluded that a single injection of steroids close to the carpal tunnel may result in long term improvement and should be considered prior to surgery.[53]

Chang and others studied the effectiveness of diuretics, non-steroidal anti-inflammatory drugs (NSAIDs) or steroids in the treatment of mild to moderate CTS. In a prospective, randomized, double-blind and placebo-controlled method, they evaluated patients with clinical signs and symptoms of CTS, confirmed with electrodiagnosis. Using the Global Symptom Scale (GSS), they found no significant reduction from baseline at either two or four weeks post therapy in the placebo, NSAID and diuretic groups. The mean GSS at four weeks in the steroid group decreased significantly indicating that corticosteroids are of greater benefit in this group.[54]

Vitamin B6 (pyridoxine) deficiency is seen in some patients with CTS. However the link between the deficiency and the disorder is controversial, as is the impact of pyridoxine on treatment. The review by Jacobson and co-workers concluded that the "literature at this time does not give convincing evidence for use of pyridoxine as the sole treatment when confronted with a patient with idiopathic CTS."[55]

Yoga has also been considered as a possible conservative treatment for CTS. A randomized, single-blind controlled trial of 42 individuals with CTS had subjects assigned to either receiving an intervention of 11 yoga postures or a wrist splint in addition to previous treatment. The results were spotty with those in the yoga group showing significant improvements in grip strength, pain reduction and Phalen's sign. However, there were no significant improvements in sleep disturbances, Tinel's sign and median nerve conduction times.[56]

Tendon and nerve gliding exercises are another non-surgical modality that has been considered. Rozmaryn and others studied 197 patients (240 hands). They were divided into two groups.

Both received standard conservative methods with the experimental group also treated with a program of nerve and tendon gliding exercises. Seventy-one percent of those in the control group underwent surgery compared with 43% of those who were prescribed the exercise regimen. Of those in the experimental group who did not undergo surgery, 70.2% reported good results at an average follow-up time of 23 months. A significant number of patients who would have otherwise undergone release surgery were spared the surgical morbidity.[57]

SURGICAL MANAGEMENT

Generally speaking, surgical management of CTS is suggested when the symptoms are not responsive to more conservative treatments after two or three months. The goal for surgery is either to reduce the volume of the carpal tunnel contents or enlarge the passageway itself. Some have favored synovectomy as a method of decreasing the content of the canal however the predictability and long-term benefits of the procedure have not been well documented. Surgical options include either open division of the transverse carpal ligament or endoscopic carpal tunnel release. Both procedures increase the capacity of the carpal tunnel and therefore relieve the pressure on the median nerve.

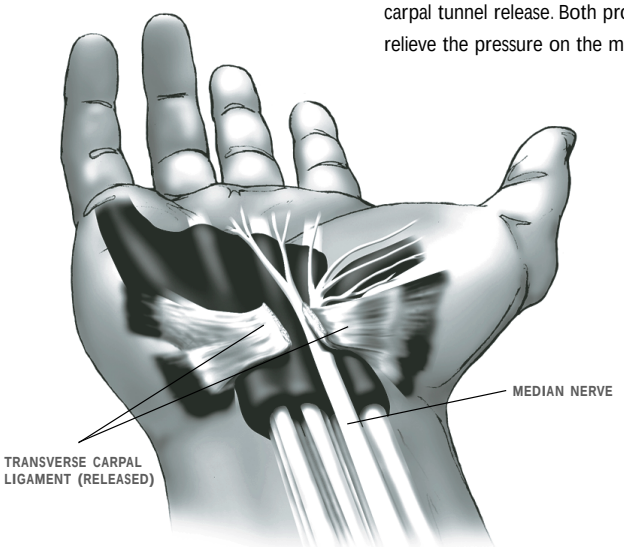


FIGURE 8: SURGICAL TRANSECTION AFTER CARPAL TUNNEL RELEASE SURGERY.

Transection of the transverse carpal ligament allows the passageway to widen and reduces or eliminates the compression of the median nerve.

There have been many different methods for open release of the transverse carpal ligament (OCTR). The procedure is usually performed on an out-patient basis and local, regional or general anesthetic may be used. Incisions vary according to the preference of the surgeon but are usually longitudinal in the proximal palm with or without extensions across the wrist. The surgeon carries out dissection until all or part of the transverse carpal ligament has been exposed and will then carefully transect the entire ligament to decompress the nerve. (Fig. 8.)

Limited or short palmar incisions have been favored by many surgeons because these methods result in reduced morbidity versus more extensive approaches that violate all tissue levels over a greater distance. Different instruments and methods for completing the ligament division using short incisions have been devised and the techniques may benefit short-term recovery although there is probably minimal long-term difference between the long and short incision methods. Recovery is usually fairly rapid following open carpal tunnel decompression but return to demanding hand and wrist use may require several months.

In recent years endoscopic carpal tunnel release (ECTR) has gained some popularity as a minimally invasive method of reducing pressure within the carpal tunnel. The advantages are felt to be the reduced damage to wrist and palmar tissues and, therefore, a more rapid recovery following surgery. The instruments used are inserted through one or two small holes in the wrist to the synovial sheath of the tunnel and directed along the axis of the ring finger. The ligament is seen using a small telescope that provides a magnified image on a television screen that the surgeon watches during the procedure.

Jimenez, Gibbs and Clapper undertook a ten-year (1987-1997) review on endoscopic release of TCL in the management of patients with CTS. A total of 52 studies on six endoscopic techniques comprising 8068 cases were found and analyzed for this review. The overall success rate in those articles reviewed was 96.52% with a complication rate of 2.67% and a failure rate of 2.61%. The authors concluded, "this review indicates that the success, complication and failure rates of ECTR are comparable to those of OCTR procedures. However, as with OCTR techniques, there is significant variability in ECTR procedures."[58]

Among the positives touted for open release are that the larger incisions may make a complete release easier for the surgeon. ECTR, on the other hand, is said to lessen the problems with a high rate of scar tenderness and a delay in returning to normal activities of daily living that are seen with open surgery.[59] In addition, complication rates of between 10% and 20% have been seen in the past with open surgery.[60,61] However, some newer reports show similar frequency and severity in complications with both open and ECTR.[62]

Palmer et al used questionnaire responses from members of the American Society for Surgery of the Hand to compare the surgically treated complications of endoscopic and open carpal tunnel release over a five-year period. There were 455 major complications reported by 708 respondents. The complications included 100 median nerve lacerations, 88 ulnar nerve lacerations, 77 digital nerve lacerations, 121 vessel lacerations, and 69 tendon lacerations. The complications were fairly equally divided between the two methods. They concluded that carpal tunnel release by either open or endoscopic means is not always a safe and simple procedure.[63]

Currently, the trend among surgeons dealing with surgical decompression of the median nerve in the carpal tunnel appears to be away from ECTR and more towards methods of limited incision OCTR. Instruments for the safe division of the transverse carpal ligament with some degree of direct visualization of the carpal tunnel contents are proving to accomplish the same morbidity, thus reducing advantages of ECTR without the disadvantages and complications posed by the endoscopic methods.[64] The reliability and good visualization of open carpal tunnel release methods reinforces their role as the preferred techniques for decompressing the median nerve.

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