

**CONSERVATIVE TREATMENT OF CARPAL TUNNEL SYNDROME**  
**A LITERATURE REVIEW**

**SENIOR RESEARCH ADVISOR: CHRISTOPHER STANDRING, D.C.**

**LAWRENCE H. PAYTON B.A.**

**JUNE 25, 1997**

## **ABSTRACT**

Carpal tunnel syndrome[CTS]and median nerve entrapment syndrome of the upper extremity are often treated as the same syndrome. Misdiagnosis and an early choice of surgical release often leaves the patient dissatisfied due to recurring symptoms. An aggressive personalized conservative treatment plan of chiropractic adjustments, trigger point therapy, myofascial release technique, nutrition, and exercise can be an effective alternative to surgery.

### **Key Words:**

Carpal tunnel syndrome[CTS], Median nerve entrapment syndromes, CTS-diagnosis, CTS-treatment, CTS-chiropractic care

## Introduction

Carpal tunnel syndrome(CTS)is defined as a peripheral entrapment neuropathy involving the median nerve where it passes below the transverse carpal ligament of the anterior wrist. This syndrome was first described in the literature in 1854, and currently is considered the most common entrapment neuropathy of the upper extremity.[1]

Because of its compactness and rigid walls, the carpal tunnel is unable to accommodate any swelling, pus infiltration, or pathological growth without compressing the structures, especially the median nerve, that traverse it. The condition known as the "carpal tunnel syndrome" is usually characterized by numbness of the fingers supplied by the median nerve, and in severe cases by muscular paralysis marked by loss of thumb opposition.[2]

Cumulative trauma usually of a compressive nature is associated with repetitive motion activities of the hands and fingers, combined with application of forceful pressure from work surfaces.[3]

Traditionally, patients have been hastily, and often incorrectly, diagnosed with carpal tunnel syndrome. Conservative care consisted of rest, cryotherapy, splints, and anti-inflammatory drugs. If these measures failed to

relieve symptoms, patients were presented for surgery to reduce to pressure on the median nerve. The results of surgery had low success rates in work-related disorders. Disability was often prolonged. It was concluded that surgery should only be performed for clear diagnosis and clear indications after adequate non-operative treatment, with careful consideration of the job to which the worker will return.[4]

The purpose of this paper is to present a thorough understanding of CTS, its presenting features, methods of diagnosis, differential diagnosis, and comprehensive plan of patient care in order to restore function, reduce symptoms, and return the patient to work fitness without surgical trauma.

### ANATOMY OF THE CARPAL TUNNEL

At the wrist, the antebrachial fascia thickens to form the palmar carpal ligament. This fibrous band is anchored to the styloid process of the radius and ulna. Anteriorly, this ligament has some attachment to the flexor retinaculum, which lies deep to the palmar carpal ligament. The flexor retinaculum is a strong, dense, fibrous, band that stretches across the wrist from the scaphoid and trapezium bones laterally to the pisiform and hook of the hamate bones medially. Together, with the anterior concave surfaces of the carpal bones, the flexor retinaculum completes the osteofibrous canal, the carpal tunnel.

Through the carpal tunnel, and into the palm of the hand passes the median nerve, the four tendons of the flexor digitorum profundus, the four tendons of the flexor digitorum superficialis, and the tendon of the flexor pollicis longus. The tendon of the flexor carpi radialis pierces the lateral side of the retinaculum in its own compartment[not the carpal tunnel] as it crosses the wrist. Note that the ulnar nerve and artery enter the palm superficial to the flexor retinaculum but deep to the palmar carpal ligament. One common synovial sheath encloses the tendons of the flexor digitorum profundus and the flexor digitorum superficialis. The flexor pollicis longus tendon is enclosed by a separate synovial sheath.

The median nerve arises from the fusion of the lateral cord[C5,C6,C7]and the medial cord[C8,T1] of the brachial plexus. The median nerve, like the ulnar nerve, has no muscular branches in the arm. However, it is not uncommon to find communicating twigs between it and the musculocutaneous nerve. The median nerve then passes distally through the cubital fossa lying medial to the brachial artery and medial and anterior to the ulnar artery. As it leaves the fossa the median nerve passes through the pronator teres muscle, splitting that muscle into superficial and deep heads with respect to the median nerve. The median nerve then runs almost vertically through the forearm into the hand. In the distal third, it is covered superficially by only the tendon of the palmaris longus muscle. Consider that 12% of the population have a congenitally absent palmaris longus muscle. In such cases the median nerve is hazardously superficial.[2]

Muscles innervated by the median nerve include:

Pronator teres

Palmaris longus[absent in 12% of the population]

Flexor carpi radialis

Flexor digitorum superficialis

Flexor pollicis longus

Flexor digitorum profundus[lateral portion]

Pronator quadratus[deep surface]

Articulations innervated by the median nerve include:

- Elbow joint
- Proximal radio-ulnar joint
- Distal radio-carpal joint
- Radio-carpal joint
- Carpal joints

In the hand, the median nerve innervates the:

- Flexor pollicis brevis
- Abductor pollicis
- Opponens pollicis
- First dorsal interosseous
- First, second, and often third lumbrical

Cutaneous innervation of the palmar surface of the hand by the median nerve includes the lateral three and one half digits.[2][5]

#### **CLINICAL PRESENTATION OF CARPAL TUNNEL SYNDROME**

Carpal tunnel syndrome[CTS]is a nerve entrapment syndrome caused by compression of the median nerve within the carpal tunnel. A variety of factors can result in an acute or insidious onset of symptoms. The dominant hand is more often involved, but symptoms can occur bilaterally.[6]

The most common signs of CTS are pain and paresthesias in the thumb and the second and third fingers. Patients present with decreased sensation to pinprick in the first three digits along with an electric shock sensation produced by percussing the carpal tunnel[Tinel's sign]. Acute flexion of the wrist will produce tingling in the same three digits[Phalen's sign].[7]

Symptoms are usually worse in the morning, frequently causing the patient to awaken from sleep to shake their hands to relieve the numbness and pain. This nocturnal pain is considered pathognomonic of CTS. In the early stages of CTS, symptoms may be non-specific and diffuse. Referred pain from median nerve entrapment within the carpal tunnel manifests symptoms similar to those seen in the inflammatory disorders of tendonitis or bursitis. The areas of pain referral are the shoulder, elbow, and forearm. Diagnosis is often delayed for this reason. Late symptoms of CTS include muscle weakness and atrophy. The thenar eminence is most commonly involved. This weakness causes reduced grip and pinch strength. Patients present with impaired ability to perform standard tasks of daily living. What was once perceived as unexplained clumsiness now causes the patient heightened concern.[8]

Repetitive motion injuries have been associated with CTS. The British Commonwealth countries use the term repetitive

stress injury[RSI], and The United States uses cumulative trauma disorder[CTD]to label repetitive motion injuries. In the past two or three decades, increasing attention has been turned toward the role of motion or repetitive activity in the development or exacerbation of compression neuropathies of the upper extremities. Enormous costs associated with CTDs has kept both medical and governmental attention focused on these disorders. One survey found that 47% of 7214 cases of CTS were occupationally related.[9]

The compressive neuropathies and non-specific complaints of numbness, tingling, and upper extremity discomfort mimic CTS symptoms. Current research points to multilevel problems, including posturally induced muscular imbalance as probable causes. Conservative approaches to successfully treat many individuals have lessened the indication for surgical intervention. Ergonomic changes at work stations, postural changes, muscle stretching and strengthening, and muscle group balancing have proven effective approaches to conservative treatment.[10]

#### **DIFFERENTIAL DIAGNOSIS**

Vague pain and numbness of the upper extremity can be caused by several conditions other than CTS. Therefore, misdiagnosis of CTS is a common error in practice. Examples of conditions that can mimic the symptoms of CTS are:

**Table 1**

**Degenerative Conditions:** multiple sclerosis and syringomyelia

**Hereditary Conditions:** Recklinghausen's disease, tuberous sclerosis

**Idiopathic-Central Causes:** psychosis[hysterical numbness, clenched fist syndrome]and seizures

**Idiopathic-Peripheral Causes:** angina, atypical TOS, brachial plexus injury, Buerger's disease, cervical radiculopathy, pronator syndrome, Raynaud's phenomenon, reflex sympathetic dystrophy, work related fatigue, and static strain

**Infectious Conditions:** brain abscess and deep palmar abscess

**Inflammatory Conditions:** bursitis, carpometacarpal joint arthritis, cervical spine arthritis, myofascial dysfunction, tendonitis

**Neoplastic Conditions:** extracranial or intracranial lesion, spinal cord lesion

**Patient Characteristics:** aging, lack of aerobic exercise, malingering, obesity

**Traumatic Conditions:** head injury, radiation neuritis, subdural hematoma

**Vascular Conditions:** aneurysms, A-V malformations, migraines, stroke, TIA, vasculitis, White-finger syndrome  
Vitamin B6 or B12 deficiency.[8]

Organic causes of CTS symptoms may include congenital anomalies, recent or poorly healed fractures, space

occupying lesions, synovitis of the flexor tendon sheaths or carpal joints, pregnancy[edema in soft tissues], and obesity. Systemic causes of CTS symptoms are rheumatoid arthritis and rheumatic conditions such as inflammatory tenosynovitis, myxedema[hypothyroidism], sarcoidosis, amyloidosis, tissue infiltration leukemia, acromegaly, hyperparathyroidism, hypocalcemia, and diabetes mellitus.[11, 12, 13]

Myofascial dysfunction, trigger points in the musculature, muscle fatigue, static muscle strain in the upper extremity and neck can mimic CTS. Cervical spondylosis and disc protrusions[C7-T1 nerve roots], as well as thoracic outlet syndrome[TOS]and apical lung tumors, can also be added to the differential diagnosis. Rarely, cord level lesions may present with symptoms of neuropathy. Spinal tumors, neurofibromas or syringomyelia are among these lesions. They present with additional symptoms of reflex changes or spasticity. Systemic neurologic disorders such as amyotrophic lateral sclerosis[ALS or Lou Gerhig's Disease] may cause muscle weakness and atrophy, symptoms common to CTS, but rarely present with sensory symptoms.

The numbness and paresthesia of the radial three and one half digits can be caused by nerve compression of the median nerve at several levels along its course. Entrapment occurs most commonly at the thoracic outlet as the nerve passes

through the costo-clavicular area, at the level of the subscapularis in the axilla, at the ligament of Struthers just proximal and medial to the elbow, between the superficial and deep heads of the pronator teres muscle, under the anterior interosseous membrane in the forearm, and most distally as it passes through the carpal tunnel into the wrist. Although entrapment can occur anywhere along the course of the median nerve, the most common site is between the two bellies of the pronator teres.[14]

#### **DIAGNOSTIC EVALUATION OF CARPAL TUNNEL SYNDROME**

Clinical diagnosis of CTS can be made by assessment of patient symptoms. Three types of physical findings are typical. These include:

1. Decreased sensibility
2. Patient response to provocative Ortho/Neuro tests
3. Decreased strength

#### **DECREASED SENSIBILITY**

The assessment of nerve fiber sensibility [threshold testing] is achieved primarily by three methods; Semmes-Weinstein monofilament test, vibratory sensibility test, and the Weber two point discrimination test. The Semmes-Weinstein monofilament test has only slightly less

sensitivity than the vibratory test, and is much more cost effective. The Weber two point discrimination test presents clouded neurological findings in that overlapping peripheral nerve receptors are measured as opposed to the threshold test which assess single nerve fibers.[14]

#### **PATIENT RESPONSE TO PROVOCATIVE ORTHO/NEURO TESTS**

Evans describes the procedure for Phalen's Sign[Phalen's Sign, Prayer Sign]as follows:

The patient flexes the patient's wrists maximally and holds this position for 1 minute by pushing both the wrists together. A positive test is indicated by a tingling sensation that radiates into the thumb, the index finger, and the middle and lateral half of the ring finger. The presence of this sensation indicates carpal tunnel syndrome caused by pressure on the median nerve.

Evans further explains:

Phalen's sign duplicates the wrist flexion/extension maneuvers that irritate the median nerve. The presence of Phalen's sign is a good indicator that wrist splints will be useful in the management of the carpal tunnel syndrome. As a screening test, a reverse Phalen's maneuver can be performed. The patient is asked to press the hands together in a vertical plane and raise the elbows until they are horizontal. Loss of any dorsiflexion should be obvious. The most common cause of lost dorsiflexion is stiffness after a Colles' fracture.[15]

The procedure for Tinel's sign at the wrist is:

The patient's elbow is flexed and the forearm supinated. The wrist and hand are slightly dorsiflexed by the examiner. The examiner percusses the volar surface of the wrist over the carpal tunnel with a reflex hammer or tuning fork. Tingling that is along the median nerve distribution and distal to the point of percussion indicates regeneration of the nerve. Pain following the same distribution, above and below the point of percussion, indicates neural inflammation and degeneration. Percussion at the Tunnel of Guyon reveals the condition of the ulnar nerve as it passes into the hand.

Tinel's sign is extremely useful in identifying (1) the most proximal point of nerve regeneration or (2) the most distal point of nerve degeneration. These points are one and the same. Tinel's is most evidenced at the Valleix points [tender points] along the course of the peripheral nerve [as in a neuralgia/neuritis]. The examiner also may slide the tip of the index finger across the palm, noting frictional resistance and temperature. Increased thenar resistance from lack of sweating and temperature rise [vasodilation] may occur with median involvement. [15]

The Wringing test procedure for diagnosis of CTS: The patient, using both hands, wrings a towel. Maximum effort is applied. The test will localize the discomfort to the primary site of origin. If the test elicits pain at the elbow, epicondylitis is suspected. If the discomfort is felt at the wrist, arthropathy or carpal derangement is suspected. Paresthesia in the hand indicates carpal tunnel syndrome.

The wringing test is useful to determine the area for primary investigation. The patient also may be asked to hold both wrists in a fully flexed position for 1 to 2 minutes. The appearance or exacerbation of paresthesia suggests carpal tunnel syndrome. This test is the most sensitive clinical test for carpal tunnel syndrome. Advanced carpal tunnel syndrome can produce thenar atrophy and distal phalangeal acroasphyxia. The wringing test is particularly useful in eliciting responses in more subtle afflictions of the median nerve. [15]

A more recent provocative test is the carpal compression test. The procedure for this test involves application of direct pressure over the carpal tunnel. The examiner uses both thumbs along the long axis of the median nerve at the carpal tunnel. Steady pressure is held for 30 seconds.

Provocation of symptoms is a positive test. [16]

Research varies on the sensitivity and specificity of these tests. Carpal compression, Wringing test, and Phalen's sign have higher sensitivity than Tinel's sign. The Tourniquet

test(occlusion of the extremity arteries with a blood pressure cuff)has the lowest sensitivity rating.[17]

#### **DECREASED STRENGTH**

Decreased muscle strength is a somewhat subjective matter for the clinician. It is based on observation of thenar atrophy and muscle testing of the abductor pollicis brevis, opponens pollicis, flexor pollicis longus, flexor pollicis brevis, and palmaris longus.[18]

#### **ELECTRODIAGNOSIS OF CARPAL TUNNEL SYNDROME**

Nerve conduction studies[NCSs]and electromyography[EMG] are considered the definitive tests for diagnosis of CTS. NCS's are judged to be the most sensitive for the detection of median nerve sensory and motor conduction deficits.[19] However, statistical analysis demonstrated no significant differences in final symptomatic outcome after surgical carpal tunnel release when comparing patients who had positive, negative, or no electrodiagnostic testing prior to surgical release.[20]

#### **ADVANCED IMAGING**

Plain film radiograph seldom offer findings that contribute to diagnosis of CTS. Use of magnetic resonance imaging[MRI]is indicated only after conservative treatment has failed, nerve conduction studies are definitive, or

after physical examination reveals a mass or fullness at the carpal tunnel.[21]

When MRI studies are considered necessary for diagnosis, coronal and axial images should be done in the determination of median nerve damage or deficit in CTS. The following changes to the median nerve are present in any etiology of CTS:

1. Diffuse swelling or segmental enlargement of the median nerve, best evaluated at the level of the pisiform.
2. Flattening of the median nerve, best demonstrated at the level of the hamate.
3. Palmer bowing of the flexor retinaculum, assessed at the level of the hamate.
4. Increased signal intensity within the median nerve at any level. This finding is well demonstrated on axial T2- and T2\* - weighted and inversion recovery[STIR]images.[21]

#### **TREATMENT OF CARPAL TUNNEL SYNDROME**

Patients with carpal tunnel syndrome should be told that it is a progressive condition. And without treatment it will worsen with time.[22] Medical treatment has traditionally involved anti-inflammatory drugs, oral prednisone,

corticosteroid injections, physical therapy, and when symptoms persist, surgery is recommended.[23, 24]

A conservative, non-surgical approach to treatment of CTS is in the best interest of the patient. Surgery should only be performed for clear diagnosis and clear indications after adequate non-operative treatment, with careful consideration of the job to which the worker will return.[4]

The object of conservative treatment is to provide the patient with every chance to recover from CTS without the trauma and disability associated with surgical intervention. Rest, cryotherapy, electrical stimulation, and pulsed ultrasound to the involved area[s] is the initial treatment plan. Reduction of pain, inflammation, and edema are the immediate goals.

If the patient presents with work related CTS, identification of occupational repetitive motions and/or postures should be made. The patient should avoid these tasks during the acute phase of treatment. As the patient is able to return to work status, they should be instructed in stretching exercises to be done during break periods. If possible, ergonomic changes should be made at work stations. If the employer is unable or unwilling to make such changes, the employee should be allowed job rotation to interrupt the repetitive motions that are producing CTS symptoms. Employer

education as to the cost effectiveness of these changes can be of benefit to both employer and employee.[25]

### **NUTRITION AND CTS**

Vitamin B6 [pyridoxine] deficiency has been found to cause the symptoms of CTS.[26] Another study found, "In all patients taking pyridoxine, decreases in motor latency and increases in the conduction of the velocity of the median nerve were observed." [27]

Clinical deficiency of vitamin B6 is virtually unknown. However, minor metabolic stressors can lead to deficiency. Alcohol consumption and increased protein intake increase B6 requirements.[28]

Correct dosage must be given. Overdosage can actually cause the symptoms of CTS. A dosage of 100 mg/day for twelve weeks can correct B6 deficiency related CTS.[29] In a study by Kasdan and Janes,[30] it was found that with conservative care alone 14.3% of the patients[n1075]were satisfied with the results. These patients were treated with wrist splints, job changes, anti-inflammatory agents, and steroid injections. When 100 mg/2x daily dosage of B6 was given alone or with wrist splints and/or job changes as treatment, 68% of trial patients had alleviation of symptoms. After symptoms were alleviated the dosage of B6 was reduced to 50-100 mg/day.

### TRIGGER POINT THERAPY

Myofascial trigger points are hyperirritable points located within taught bands of skeletal muscle, connective tissue, and/or skin. These points are capable of producing symptoms that mimic CTS. They may be present along with CTS. This tends to confuse the diagnosis and delay appropriate treatment. Conservative treatment of CTS must include examination of the upper extremity for the presence of trigger points. Trigger points are described by the following grading system:

Grade 1-palpable, no tenderness

Grade 2-palpable, exquisite local tenderness

Grade 3-palpable, exquisite local tenderness with referral  
pain pattern within 10-15 seconds

Grade 4-active symptomatology; activation of trigger points  
reproduces symptoms and referral pattern.[31]

Common areas of treatment related to CTS symptoms are:

1. Levator scapulae
2. Rhomboids
3. Posterior deltoids
4. Infraspinatous
5. Teres minor
6. Pectoralis minor

7. Scalenes
8. Extensor carpi radialis
9. Extensor digitorum communis
10. Pronator teres
11. Flexor carpi radialis
12. Flexor digitorum
13. Flexor carpi ulnaris
14. Thenar eminence
15. Hypothenar eminence[31]

#### **MYOFASCIAL RELEASE TECHNIQUE AND CARPAL TUNNEL SYNDROME**

Myofascial adhesions causing median nerve entrapment exhibit symptoms common to CTS. Treatment of the areas of adhesion that are causing the entrapment symptoms involves the use of myofascial release technique[MRT]as described by Leahy and Mock[32]. MTR is a method of soft tissue manipulation that is specifically for manually removing fibrous adhesions in order to restore function and proper biomechanics. The ability to manually test muscles properly and instruct the patient how to actively lengthen or shorten the muscle while the doctor performs the treatment are mandatory skills.

The basic premise of MRT is to locate impediments to normal function through palpation and muscle testing and then to remove these impediments using specific contact and active motion by the patient, sometimes using added resistance. MRT works best using a hand lotion as a lubricant to reduce

tissue resistance. The doctor's contact should be as broad and flat as possible. Use of the thumb or palm reinforced thumb is the preferred contact.

MRT has four levels of procedure. Level 1 and 2 are passive on the part of the patient. Level 1 is a longitudinal massage of the relaxed tissue. Levels 3 and 4 involve movement of the tissue being treated. Level 3 treatment has the doctor move the tissue from its shortest to longest length. Level 4 consists of the patient actively moving the same tissue from its shortest to longest length. Treatment should only be used only 3-4 times and then reevaluate the results.[32]

#### **CHIROPRACTIC CARE AND CARPAL TUNNEL SYNDROME**

Because the wrist structures are innervated primarily from C6-C7 nerve roots, lesions affecting structures of similar nerve derivation may refer pain to the wrist and vice versa.[33]

Studies have determined that low amplitude, short lever, low force, high velocity type adjustments to the cervical spine, shoulder, elbow, and wrist made a demonstrable difference in CTS. Symptoms dissipated, grip strength increased, and motor and sensory latencies were normalized.[34, 35, 36, 37]

Restoration of joint function in all joints of the upper extremity are of importance in the treatment of CTS. Of particular importance are the joints of the wrist.

Evaluation through palpation, knowledge of biomechanics, and adjustive techniques must be thoroughly mastered by the practitioner treating CTS.[33]

#### **COLD LAZER THERAPY FOR CARPAL TUNNEL SYNDROME**

Lazer therapy using helium/neon low dose lazer instruments has been found to be effective in reducing fibrosis in nerve entrapment syndromes and fibromyalgia cases. Thirty second bursts at entrapment sites are the standard treatments.[38,39]

#### **SURGERY FOR CARPAL TUNNEL SYNDROME.**

If aggressive conservative treatment does not significantly reduce the symptoms of CTS, then surgical intervention must be considered. The patient should be informed of the various types of surgical release techniques that are available to them. These include open release, closed endoscopy, and microsurgery. Some techniques are available on an outpatient basis. Pain symptoms are usually alleviated by surgery, but numbness and tingling often persists. The type of job that the patient is returning to often influences the long term success of surgical release.[40,41]

## **CONCLUSION**

Carpal tunnel syndrome has no unique cure. Practitioners must be prepared to manage each case with a personalized treatment plan. Logical elimination of probable causes and treatment of actual causes of CTS symptoms is an achievable goal. The myriad of symptom sources makes the conservative treatment of CTS a formidable challenge to both the practitioner and the patient. An active role by the patient in both treatment and recognition of causes can lead to a successful non-surgical outcome.

## REFERENCES

1. Paget J. Lectures on surgical pathology. Philadelphia: Lindsay & Blakiston, 1854.
2. Jacobs John J, ed. Shearer's Manual of Human Dissection 7th ed. 1989. McGraw-Hill Information Services Company Health Profession Division. St. Louis. 199-229.
3. Feldman RG, Goldman R, Keyserling WM. Peripheral nerve entrapment syndromes and ergonomic factors. Am J Ind Med 1983; 4: 661-81.
4. Terrono AL, Millender IH. Management of work-related upper extremity nerve entrapments. Orthop Clin Am 1996; 27(4): 783-93.
5. Netter FH, Atlas of Human Anatomy. 6th ed. Summit, New Jersey: Ciba-Geigy, 1993: 411-444.
6. Diamond MR. Carpal tunnel syndrome: A review. Chiropractic Sports Med 1989; 3(2): 46-53.
7. Mengel MB, Schwiebert LP, eds. Ambulatory Medicine 2nd ed. 1996. Appleton & Lange. Stamford. 263-267.
8. Kasdan LM, Lane C, Merrit WH, Nathan PA. Carpal tunnel syndrome: the workup. Patient Care 1993; April 15: 97-107.
9. Cummings K, Maizlish N, Rudolph L, et al. Occupational disease surveillance: Carpal tunnel syndrome. MMWR 1989; 38(28): 486-489.
10. Higgs PE, Mackinnon SE. Repetitive motion injuries. Annu. Rev. Med. 1995; 46: 1-16.
11. Tierney LM, McPhee SJ, Papadakis MA. Current medical diagnosis and treatment 33rd ed. 1994. pp 679-80.
12. Vessey MP, Villard-Mackintosh L, Yeats D. Epidemiology of carpal tunnel syndrome in women of childbearing age: findings in a large cohort study. Int. J Epidemiology 1990; Sept. 19(3): 655-659.
13. Allen CW Jr. Weight of evidence links obesity, fitness to carpal tunnel syndrome. Occup Health Safety 1993; Nov. 62(11): 51-52.
14. Leahy PM, Mock LE. Myofascial release technique and mechanical compromise of peripheral nerves of the

- upper extremity. Chiro sports Med 1992; 16(4): 139-150.
15. Evans RC. Illustrated essentials in orthopedic physical assessment. 1st ed. St. Louis: Mosby, 1994: 161-207.
  16. Durkan JA. A new diagnostic test for carpal tunnel syndrome. J Bone Joint Surg 1991; 73-A(4): 535-538.
  17. Kuschner SH, Ebrahimzaadeh E, Johnson D, Brien WW, Sherman R. Tinel's sign and Phalen's test in carpal tunnel syndrome. Clin Ortho 1992; 15(11): 1297-1302.
  18. Kendall FP, McCreary EK, Provance PG. Muscles testing and function. 4th ed. Baltimore: Williams & Wilkins, 1993: 235-263.
  19. Jablecki CK, Andary MT, So YT, Wilkens DE, Williams FH. Literature review of the usefulness of nerve conduction studies and electromyography for the evaluation of patients with carpal tunnel syndrome. Muscle Review 1993; Dec. 16(12). 1392-1414.
  20. Glowacki KA, Breen CJ, Sachar K, Weiss AP. Electrodiagnostic testing and carpal tunnel release outcome. J Hand Surg[Am]1996; Jan. 21(1). 117-121.
  21. Yochum TR, Rowe LJ. Essentials of Skeletal Radiology 2nd ed. Baltimore: Williams & Wilkins, 1996: 471.
  22. Kulick RG. Carpal tunnel syndrome. Orthop Clin North Am 1996; Apr. 27(2). 345-354.
  23. Herskovitz S, Berger AR, Lipton RB. Low dose, short-term oral prednisone in the treatment of carpal tunnel syndrome. Neurology 1995; Oct. 45(10). 1923-1925.
  24. Irwin LR, Beckett R, Suman RK. Steroid injection for carpal tunnel syndrome. J Hand Surg[Br]1996; Jun. 21(3). 355-357.
  25. Thomas RE, Butterfield RK, Hool JN, Herrick, RT. Effects of exercise on carpal tunnel syndrome symptoms. Applied Ergonomics 1993; 24(2). 101-108.
  26. Ellis JM, et al. Survey and new data on treatment with pyrioxine of patients having a clinical syndrome including the carpal tunnel and other defects. Res Comm Chem Pathol Pharmacol 1976; 17(1).
  27. Wolaniuk A, Vadhanavikit S, Folkers K. Electromyographic data differentiate patients with the carpal tunnel syndrome when double blindly treated with pyrioxine and placebo. Res Comm Chem Pathol Pharmacol 1983; 41(3).

28. Ellis JM, Folkers K, Levy M, et al. Response to vitamin B6 deficiency and carpal tunnel syndrome to pyrioxine. Proc. Nat. Acad Sci 1982; 79: 7494-7498.
29. Walther DS. Applied Kinesiology Synopsis 1st ed. Pueblo: Systems DC, 1988: p.448.
30. Kasdan ML, Janes C. Carpal tunnel syndrome and vitamin B6. Plastic Reconstruc Surg 1987; 79(3).
31. Travell JG, Simons DG. Myofascial Pain and Dysfunction. The Trigger Point Manual. The Upper Extremities. Baltimore: Williams & Wilkins, 1983: 12.
32. Leahy PM, Mock LE. Myofascial release technique and mechanical compromise of peripheral nerves of the upper extremity. Chiro Sports Med 1992; 6(4). 139-150.
33. Bergmann TF, Peterson DH, Lawrence DJ. Chiropractic Technique Principles and Procedures. New York: Churchill Livingstone, 1993: 543-633.
34. Valente R, Gibson H. Chiropractic manipulation in carpal tunnel syndrome. J Manipulative Physiol Ther 1994; 17(4). 246-249.
35. Bonebrake AR, Fernandez JE, Dahalan JB, Marley RJ. A treatment for carpal tunnel syndrome: results of a follow-up study. J Manipulative Physiol Ther 1993; 16(3). 125-139.
36. Bonebrake AR, Fernandez JE, Marley RJ, Dalahan JB, Kilmer KJ. A treatment for carpal tunnel syndrome: evaluation of the objective and subjective measures. J Manipulative Physiol Ther 1990; 13(9). 507-520.
37. Mariano KA, McDougale MA, Tanksley GW. Double crush syndrome: chiropractic care of entrapment neuropathy. J Manipulative Physiol Ther 1991; 14(4). 262-265.
38. Basford J. Laser therapy: scientific basis and clinical role. Orhtopedics 1993; 16. 541-547.
39. Rochkind S, et al. Stimulatory effect of He-Ne low dose laser on injured sciatic nerves of rats. Neurosurgery 1987; 20. 843.
40. Mirza MA, King FT Jr. Newer techniques in carpal tunnel release. Orthrop Clin North Am 1996; Apr. 27(2). 355-371.
41. Shapiro S. Microsurgical carpal tunnel release. Neurosurgery 1995; Jul. 37(1). 66-70.