ELECTROCEUTICAL TREATMENT AND THE RESOLUTION OF COMPRESSION / ENTRAPMENT NEUROPATHY

Cumulative Trauma Disorders: Carpal Tunnel Syndrome

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Abstract

Compression of the median nerve within the carpal tunnel is the most common entrapment neuropathy and represents nearly 50% of all occupational injuries. At present, the definitive treatment for this condition is decompression of the nerve by surgical division of the transverse carpal ligament. Specific-parameter bioelectric treatment was utilized in 133 patients presenting with moderate-to-severe Cumulative Trauma Disorders (CTD), specifically Carpal Tunnel Syndrome (CTS). Complete or partial resolution of symptoms was achieved in 79% (105) of the patients without surgical intervention.

Introduction

The median nerve contains contributions from C5-T1 spinal nerve roots and is formed from the lower trunk of the brachial plexus. It lies medially in the upper arm, crosses the elbow anteriorly and in the upper forearm usually passes between the two heads of the pronator teres, although its relationship to this muscle is variable.[1] At the wrist, the median nerve passes, along with the flexor tendons, under the transverse carpal ligament, which forms the roof of the carpal tunnel. In the hand, the median nerve innervates four intrinsic hand muscles - the abductor pollicis brevis, opponens pollicis, and the first lumbrical muscles.

Compression of the median nerve within the carpal tunnel is the most common entrapment neuropathy. The dominant hand is the first and most severely affected but CTS is often bilateral and typically affects more women than men,[2] CTS is caused by compression of the median nerve as it passes through the wrist. This occurs when the area of the carpal tunnel is reduced or narrowed by wrist flexion or swelling of the flexor tendons of the fingers, increasing the susceptibility of the nerve to pressure. Occasionally, there is no apparent cause and these patients may have congenitally narrow carpal tunnels with compression developing from normal degenerative changes in the synovial membranes and other structures within the tunnel.[3] Improved technology in imaging with diagnostic ultrasound and MRI should increase the number of differentiated, identifiable causes in patients with otherwise idiopathic CTS.[4]

According to the Bureau of Labor Statistics and the National Institute of Occupational Safety and Health, CTS has long been associated with occupations involving repetitive movements of the hands and fingers. Specific biomechanical studies have shown that pressures within the carpal tunnel are increased substantially during wrist flexion, especially when the fingers were flexed simultaneously in a pinching-type movement.[5, 6, 7] CTS frequently occurs in occupations that offer little relief from repetitive motion movement or job that requires a force exceeding 8-12 pounds It is postulated that it is repetitive motion without adequate recovery time that causes the inflammation and tunnel area reduction. A research study from the University of Michigan found that the use of more than 8 or 9 repetitions per minute did not allow the wrists sufficient time to produce enough lubricating fluid (Barrier, 1991).

CTS is characterized by bouts of pain and paresthesia in the wrist and hand, often occurring during sleep or on waking and symptoms are usually worsened by using the hands. Pain often spreads to the fingers, arm, or the shoulder, but the paresthesias is localized to the palmar aspect of the fingers.[8, 9, 10] CTS is considered a class 2 type nerve injury, characterized by axonal interruption, with intact connective tissue framework maintained. The physiological features of CTS (chronic compression / entrapment neuropathy) are still not clearly understood, but it is believed that demyelination initially occurs and, if this condition persists, axonal destruction may follow

Table 1 depicts the types of nerve injury and their corresponding anatomic and clinical features.

TABLE 1

Type	Anatomic Lesion	Clinical Features	Prognosis
1	Either (A) transient conduction block due to ischemia or (B) demylelination.	(A) mild sensory loss and weakness (ischemic) from transient abnormal posture.	(A) Rapid and complete recovery.
2	Axonal interuption; connective tissue intact.	(B) Prolonged compression with paralysis and moderate sensory loss below site of lesion	(B) Gradual usually complete recovery.
3	Transection of axons.	Closed, crushed and percussion injury: loss of motor, sensory, and autonomic-function below site of lesion.	Very slow recovery Prognosis best with distal lesions.

Differential Diagnosis

As with any medical disorder; the diagnosis begins with the history and physical examination. Because the etiology of Cumulative Trauma Disorder (CTD) can be so varied, the history should encompass both occupational and non-occupational factors. The date of onset, character, extent, duration and frequency of symptoms, causation, effect on function, and social implications of the injury should be ascertained as well.

The physical examination should emphasize soft tissue. This includes a complete assessment of muscle strength, spasm or contracture, sensory intactness for pin prick and proprioception, range of motion (ROM) with the determination of articular abnormality kinesiologic function and restriction, ligamentous laxity and resilence, tendon and synovial palpitation, and vasomotor/sudomotor changes relating to skin temperature, color; swelling, and sweating.

The examination should not be confined to the area of presenting complaint, since the pathology may be referred from another location. An example would be subjective pain and numbness in the first three digits of the hand secondary to first dorsal interosseous myofacial syndrome, radiocaipal ligamentous strain, carpal tunnel syndrome, extensor carpi radialis, longus tendonitis, or C-6 radiculopathy.

Sensory symptoms in the fingers innervated by the median nerve and proximal radiation of pain can be caused by compression of the C-6 or C-7 nerve roots. However; the symptoms of cervical raiculopathy are rarely bilateral, are not usually worse at night, and are sometimes precipitated by neck movements. Also, the sensory symptoms and signs in a cervical radiculopathy are usually in the dorsal as well as palmar aspects of the thumb (C6) or the third digits (C7); there is often weakness of the muscles of the arm and forearm supplied by nerves other than the median, the tendon reflexes in the arm are often reduced. Nerve conduction studies are normal, but EMG usually shows abnormalities in muscles innervated by C6 or C7.

Wasting of the hand muscles, particularly those of the thenar eminence, can be caused by compression of the lower trunk of the brachial plexus by a plexus by a cervical rib or band- the true neurogenic thoracic outlet syndrome. However, the sensory deficits are usually in the C8 and T1 dermatomes - the fourth and fifth digits and the ulnar border of the hand and forearm. Nerve conduction and EMG studies usually clearly distinguish other entrapment neuropathies form CTS.[11, 12]

Discussion

Depending on the reference cited, CTDs account for 30% to 47% of workplace injuries. Part of the problem in classifying the exact percentage is that they are referenced by many different names. They have been referred to as repetitive use injuries, overuse syndromes, repetitive strain syndromes, and cerviobrachial injuries. Among the more commonly reported diagnosis are Cubital and Carpal Tunnel Syndrome (CTS), radial nerve entrapment and lateral epicondylitis, DeQuervain's disease and tendosynovitis, and a variety of synovitis and myositis abnormalities.

CTD injuries are disorders of the soft tissue, which are caused, aggravated, or precipitate from repeated mechanical stresses, exertions, and movements of the body. Both occupational and non-occupational factors are thought to contribute to their development. Occupational factors include forceful, repeated exertions, especially in combination with neutral arm and hand postures and static muscle loads. Other occupational factors reported in the literature include the use of certain hand tools, awkward gloves, machine pacing, vibration, and a cold work environment.

Identified non-occupational factors are extensive. Some are related to lifelong or temporary medical conditions others are related to lifestyle. While there is a consensus that human mechanical predisposition toward the development of CTDs exists, there is a growing perception that certain types of medical conditions are more important than once thought. A large percentage of medical conditions relate in some manner to abnormalities of tissue blood flow.

It is known that the sympathetic component of the autonomic nervous system innervates tissues of mesodermal and ectoderma] origin. It travels in close association with small, unmyelinated C fiber. The relationship of sympathetically mediated pain syndromes and how these factors impact on CTDs merits further study.

It appears that the incidence of Carpal Tunnel Syndrome (CTS) continues to grow at such a rapid rate due to the changing industrial environment, the demand for more repetitive tasks for the worker, increased patient awareness of cumulative trauma disorders and changes in overall work ethics. The shear costs involved with the work-time lost, decreased productivity, medical treatment, rehabilitation and management of CTS has caused a significant amount of attention to be focused by employers, medical treatment, rehabilitation and management of CTS has caused a significant amount of attention to be focused by employers, medical professionals, and insurance companies to aggressively work towards reducing CTS cases.

The National Institute of Occupational Safety and Health estimates that CTS occurs in more than 25,000 workers per year, costing approximately $4,000 in benefits and rehabilitation per patient and if surgical intervention is required, the costs rise to more than $20,000 per patient. These figures do not include costs associated with undesired pharmacological side effects or surgical complications reported later by the patient.

In conjunction with the patient's intake examination, exploration of the individual daily experiences should be made. Once CTS is determined in differential diagnosis, the treatment approach requires potent analgesic measures along with a well-planned rehabilitation program. This can be accomplished by surgical or electroceutical methods. Treatment methods or therapy regiments include the following:

1. **Electroceutical treatment**
2. **B-Complex vitamin therapy**
3. **Rest from repetitive tasks**
4. **NSAID therapy program**
5. **Flexibility exercise program**
6. **Cold (ice) therapy**
7. **Application of splint (mild CTS)**
8. **Medical Microwave Resonance (MMR)**

The electroceutical methods were evaluated for the most consistent clinical results and it was found that ***"specific-parameter electroceutical treatment"*** produced consistently better patient results that any other therapy or therapy regimen. Overall results indicate an asymptomatic patient response in 79% of the patients with no appreciable incidence of reoccurrence at a 6-month follow-up.

Materials and Methods

To assist in creating an average CTS patient profile, 150 patients from different geographical areas whose history and examination findings were consistent with carpal tunnel syndrome were studied prospectively. After a 3 day treatment trial, 17 patients were dropped from the programmed study because they refused to discontinue pain medication, could not guarantee future-treatment compliance (transportation problems), or reported an already-attained asymptomatic status (mild CTS). Of the remaining 133 patients entering the 30-day regimen, 78 were women and 55 were men with occupations primarily in the motor vehicle industry, data processing business, general manufacturing industry and from meat-packaging or food processing plants. Both hands were involved in 87 patients and all reported moderate-to-severe pain, paresthesia or numbness in all digits or in the median nerve distribution. The duration of symptoms ranged from 5 weeks to 10 years and most patients noted easy fatigability with fine motor activities, such as writing or buttoning clothes. Thirty-six (36) patients (27%) had co-existing cervical arthritis, diabetes mellitus, or hypothyroidism.

All patients underwent formal diagnostic testing of the median curve which included the asymptomatic extremities of the unbilateral CTS patients. Abnormal values consisted of distal motor latencies of 4.3 msec or greater and distal sensory latencies of 3.6 msec or greater. Terminal motor latencies over carpal tunnel were determined by stimulation of median nerve 3 cm proximal to the distal flexion crease of the wrist. [8, 9]

After the initial trial, a detailed treatment program was administered to all patients. Specific-parameter bioelectric treatment was applied via special 6-field CTS anatomical electrodes - 20 minutes daily for 5 consecutive days and then every other day for 3 weeks (14 total treatments). Dosage was initially set at just sensory threshold and, later in the course of treatment, increased to just below motor threshold - each patient was dosed individually but ***never*** to an uncomfortable level. A special electroceutical medical device was supplied and programmed with specific treatment parameters provided by the Clinical Electromedical Research Academy (CERA).[13] These electroceutical treatment parameters were specified for producing the following necessary physiological effects in the treatment of CTD/CTS: *diffusion of metabolic toxins, anti-inflammatory action, increased circulation for trophic improvement, counter-irritation and neuropeptide release for potent analgesia.*

Although NSAIDs were discontinued for this study, it was found that when NSAIDs were given in the initial acute pain phase, they produced a favorable influence in most all patients *provided* the dosage was *reduced* to approximately *one-half* of the standard prescribed dose. Normally-prescribed dosage appears to negatively influence or reduce the electroceutical treatment activity-even though no unusual side effects were noted.[14]

The *one-half* dosage drug interaction with simultaneous electroceutical treatment warrants further investigation because there appears to be no loss in drug potency while reducing possible standard-dose toxicity or side effects.

Results, Summary and Conclusion

At the end of the 30-day treatment program, 79% (105) of the patients reported substantial, acceptable partial pain relief or total pain relief (asymptomatic) of CTD/CTS symptoms. In addition to pain relief, other improvements were noted - these included; increased active movements of extremities, restored sensory function of extremities, restored sensory function of affected extremities, restored sensory function of affected extremities, improved sleep and overall improved health, mental status, and quality of life. Twenty-eight (28) patients (21%) reported *unacceptable* pain relief and elected surgical intervention. Of the 21% of the patients considered *treatment failures*, most were considered to have congenitally narrow carpal tunnels and possibly predisposed to CTS. In these cases, surgery would probably be the *only* definitive option.

Although surgical intervention is still considered the definitive treatment for compression/entrapment neuropathies like CTS, our study and summarized data indicates that a *electoceutical regimen* of specific-parameter bioelectric treatment is an extremely effective alternative for eliminating permanent CTS disability.

Based upon our research and other numerous, well documented reports covering non-invasive approaches to CTD/CTS, it is or opinion that ***more than 75% of all CTS surgeries could be avoided,*** especially by administering approximately 15 treatments (most cases) of cost-effective, specific-parameter electroceutical treatment.

Compared with the escalating costs associated with a surgical approach to CTS and the subsequent rehabilitation time that is typically required, the electroceutical approach should be considered in the *first line approach* to this condition.

References

1. Solnitzky, O.: Pronator syndrome: compression neuropathy of the median nerve at the level of the pronator teres muscle. Geoigetown Med. Bull., *13*::232, 1960.
2. Dyck, P., Thomas, P: Peripheral Neurnpath~Median nerve anatomy Pg. 963-3rd Ed. 1993.
3. Dekel, S., Papaionnou, T., Rushworth, 0., and Coates, R.: Idiopathic carpal tunnel syndrome caused by carpal stenosis. Br Med. 3., *1*:1297, 1980.
4. Middleton, W.D., Kneeland, J.B., Keilman, G.M., et al.: MR imaging of the carpal tunnel: normal anatomy and preliminary findings in the carpal tunnel syndrome.A.J.R., *148*:307,1987.
5. Fletcher, M.: Cumulative trauma disorders; repetitive motion cases cost billions annually *Business Insurance*, September 1990.
6. Burt, S.: "Carpal tunnel syndrome among employees at a window hardware manufacturing plant.", *Health Hazard Series*, 39(12): 576-5771991.
7. Birbeck, M.Q.: Occupation in relation to the carpal tunnel syndrome, Rheumatology Rehabilitation, *14*:218, 1975.
8. Pryse-Phillips, W.: Validation of a diagnostic sign in carpal tunnel syndrome. J. Neurology Neurosurg. Psychiatry, *47*: 870, 1984
9. Golding, D.N., Rose, D.M., and Selvarajah, K.: Clinical tests for carpal tunnel syndrome: an evaluation. Br J. Rheumatol., *25*: 388, 1986.
10. Stewart, J.D., and Eisen, A.: Tinel's sign and the carpal tunnel syndrome. Br. J., *2*:1125,1987.
11. Bleeker, ML.: Medical surveillance for carpal tunnel syndrome in workers. 3. Hand Sug., *124*: 845, 1987.
12. Dyck, P., Thomas, P, Griffin, J., Low, P., Poduslo, J.: Carpal Tunnel Syndrome – Differential Diagnosis; Peripheral Neuropathy, 3rd Ed. *50*: 964, 1993.
13. Sorgnard, R., Savery, F., Schwartz, R., Hansjürgens, A., May, H.; Electroceutical classifications guideline according to induced physiological mechanisms of action; CERA Summarization, 1993.
14. Silberstein, S., and Merriam, G.: Journal of Pain and Symptom Management,Vol. 8: 2: 98,1993.