

# Improved Treatments for Carpal Tunnel and Related Syndromes

P. Michael Leahy, D.C., C.C.S.P.

**Abstract:** *This article summarizes a case load of cumulative trauma disorder (CTD) patients seen during 1993 and 1994, outlines a suggested protocol for the case management of CTD cases, and proposes a new model or “law” of repetitive motion. The actual techniques have been described previously and must be developed by the provider through personal hands-on instruction and careful application over a period of 1 to 2 years.*

**Key Words:** Carpal tunnel disorder, repetitive motion, Active Release Techniques , law.

In recent years, the treatment of carpal tunnel syndrome has not been an area where successful outcomes were the norm. In speaking with surgeons, employers, and insurance adjusters, the estimates of “success rate” have been placed anywhere from 70% to as low as 3%. In lay publications, Blue Cross Blue Shield of California estimated that the average total cost of a “carpal tunnel” case is \$100,000. We in the health care establishment are sometimes sheltered from the realities of this problem (1). It has become one of the major sources of lost productivity and financial drains in this country. This article will briefly describe the problem, an appropriate protocol using during the period from January 1993 to April 1994, and a synopsis of over 200 of these cases that were treated over a period of several months. These cases were of the full range of severity and chronicity and were all seen within the workmen’s compensation system. No effort has been made to identify risk factors other than those cited. Although over 60% of those seen were women, the jobs involved were performed by women more than men and might not be a meaningful statistic.

The actual techniques have been previously described by Leahy and Mock (2-4). One must understand that it takes from 1 to 2 years to become adequately proficient with the techniques to produce these results. It is hoped that this information will spark interest and critical thought in those responsible for the treatment of these problems.

## **PROBLEM 1: NOT THE CARPAL TUNNEL**

Previous errors have included an overemphasis of the role that the carpal tunnel plays in peripheral nerve entrapment (5). Some researchers indicate that it is very difficult to have an increase in pressure over the median nerve in the carpal tunnel (6). In over 500 cases of upper extremity peripheral nerve entrapment and repetitive motion disorders that were supervised by this author over a period of 1 year, only two involved the carpal tunnel to any significant extent. These cases did not represent the total of cases for any one company or group but rather the total referred to the author’s group for treatment. The most common site of peripheral nerve entrapment has been at the pronator teres. In order of prevalence, the common sites of peripheral nerve entrapment were as follows (see Table 1).

**Table 1**  
**Most common peripheral nerve entrapment sites**

<b>Median nerve</b>	<b>Pronator teres</b>
<b>Radial nerve</b>	<b>Wrist extensors</b>
<b>Ulnar nerve</b>	<b>Medial edge of triceps and lig't of Struthers</b>
<b>Ulnar nerve</b>	<b>Wrist flexors</b>
<b>Ulnar nerve (medical chord)</b>	<b>Subscapularis</b>
<b>Multiple nerves</b>	<b>Subscapularis</b>
<b>Brachial plexus</b>	<b>Scalenes (trapezius, levator, serratus post)</b>
<b>Axillary nerve</b>	<b>Quadrangular space</b>

Nerve roots were seldom involved to any great extent. This was evident by physical examination because the numbness and paresthesias were of peripheral nerve patterns and not nerve root. The carpal tunnel/median nerve were involved in two cases. There were several postcarpal tunnel surgery cases in which the median nerve was entrapped at the wrist and hand by scar tissue formed as a result of the surgery. Electromyography (EMG) and nerve conduction velocity (NCV) studies do not always follow the clinical picture, and although they are still the standard test, they are not relied on as a foolproof method of examination (5, 7). Several of the cases had documented EMG or NCV alterations from normal but failed to include sufficient data to accurately locate the source of the entrapment.

All cases cited here had at least one of the following physical findings:

1. Pain in the upper extremity that could be duplicated by pressure over the appropriate tissues.
2. Numbness or paresthesias that could be mapped to a peripheral nerve pattern by light touch, pinwheel, or direct pressure examination.
3. Strength degradation that could be measured by hand dynamometer. Strength degradation evidenced by history of dropping objects or inability to accomplish previously performed tasks. Strength deficit by manual muscle testing, compared with the contralateral side and graded 1 to 5.
4. Positive electrodiagnostic studies.
5. Limited range of motion by examination.
6. Altered soft tissue texture by manual examination.
7. Altered joint or soft tissue motion by manual examination.

## **PROBLEM 2: MULTIPLE CAUSES**

Another error was the tendency to treat the case as if there was always a single site causing all of the problems. This in fact is rare. In the same 500 cases, it was extremely rare to find a situation where only one location was involved. Because the peripheral nerves are composed of single cells extending from the spinal cord, pressure in one area makes the entire nerve susceptible to even smaller pressures in other areas along its course (2). This "whole nerve syndrome" often exists before the case presents in the office.

### PROBLEM 3: INEFFECTIVE TREATMENT

Errors in treatment often contribute to the chronicity and severity of the case. Many of the case histories seemed to indicate that massage was effective at reducing muscle tension but usually failed to address the underlying cause. Stretches were poorly designed and in some cases caused more problems. Electrical muscle stimulation and iontophoresis provided symptom relief in some cases but also failed to provide a lasting solution. As more information is becoming available, bracing could well be shown to be the most troublesome therapy. The case histories show that bracing may have provided several individuals with short-term relief. Over the long term (over 1 week), bracing also led to an increase in muscle tension as a result of the musculature to “fight” the brace while attempting to perform the work task. The same statistics show that *the cases that are braced get worse*. Surgical intervention seemed to be the only logical alternative as many cases tracked through the above sequence while getting progressively worse. The problem was that, because the cases almost never involved only one site, the odds of solving the case with surgery on a single site was very low (7). Given that there is now a protocol with an extremely high success rate, one might consider this option in advance of surgical intervention.

A basic understanding of the underlying principles involved in cumulative trauma disorders (CTD) is necessary. For simplicity “CTD” will be taken to include all cases of peripheral nerve entrapment and repetitive motion disorders.

The following is a theoretical explanation and analysis of the CTD situation based on the author’s experience and efforts to define the factors involved. Only when the factors and their relations are defined may answers to the problem be acquired.

Repetitive tasks have been changed over the years in subtle ways. Force and number of repetitions have usually been considered in analyses of CTD (1). The amplitude of motion has slowly been reduced, whereas the number of repetitions has slowly gone up. Because the tasks involved less power, more repetitions could be done. A professional cyclist is required to make millions of repetitions in his sport, but CTD is not a prevalent problem. The keyboard operator also makes millions of repetitions, but CTD is common. One difference is amplitude of motion. There is an inverse relationship between amplitude and number of repetitions. If total insult to the tissues is defined as “**I**,” number of repetitions is defined as “**N**,” and amplitude of motion is defined as “**A**,” then as a model, we may define a simplified law as:

Law of repetitive motion:  $I = N/A$

As “**N**” is increased, the total insult goes up (4). As “**A**” is increased, the total insult goes down. It is quite likely that the older mechanical typewriters, for example, actually caused less of a problem because the keys had a longer throw and therefore increased amplitude. In treating a world-class cycling team over a period of 3 years, repetitive motion injuries rarely developed in a manner such as on a keyboard. This is thought to be a result of the increased amplitude of motion involved.

A more complete law must include additional factors, e.g., the force exerted for each repetition “**F**” and the relaxation period between repetitions “**R**”. A high-force requirement is difficult for the tissues to sustain and a minimal or nonexistent relaxation time does not allow sufficient circulation and cellular respiration to occur. Relaxation of the tissues involves not only a lack of

muscular contraction but also joint position. This is why the term “rest” is not used here. Total insult is directly proportional to force and inversely proportional to relaxation time.

Expanded law of repetitive motion:  $I = NF/AR$

Vibration is a motion with a very high N, a low A, and a low R and therefore is a severe insult to working tissues.

Following this model, the only way to decrease the incidence of “carpal tunnel syndrome” or CTD is to manipulate these four factors and thereby reduce the total insult to the tissues. There are four options.

- Decrease the number of repetitions
- Decrease the force required for each repetition.
- Increase the amplitude of each repetition.
- Increase the relaxation time between repetitions.

The insult to tissues has several effects. First, a hypertension develops in the musculature. This is relatively continuous and restricts circulation and lymphatic flow. This complicates the fatigue of the musculature and requires increased effort on the part of the subject. For these reasons, the situation can be self-perpetuating. Tissue hypoxia may set in, with resultant decreases in cell function and waste removal by lysosomation (8).

As a result of cellular breakdown, tissues can become fibrous (3,4). This can be palpated in the muscle itself. Adhesions may develop between tissues. As the tense muscle rubs against a sliding nerve, eventually, an injury occurs. This heals with inflammation and scar tissue formation. The resultant adhesion and lack of relative motion may also be palpated. The same may occur between muscle and muscle or any two tissues where relative motion occurs.

## **TREATMENT PROTOCOL**

Active Release Techniques , which were used by the author and the two other treating doctors in all of the cases cited, are designed to accomplish three unique objectives.

Restore free and unimpeded motion of all soft tissues.

Release entrapped nerves, vasculature, and lymphatics.

Reestablish optimal texture, resilience, and function of soft tissues.

Location of lesions requires a thorough understanding of the relative motions between tissues and the normal textures of tissues by palpation. A careful history will usually lead to likely sites, as will a thorough “orthoneuro” examination. Nothing replaces the last step, which is palpation of the lesion itself. This is a skill that must be developed by the practitioner. The learning curve can be shortened by training with one who has long-term experience and with subjects who have the lesions, but even in this situation, it requires 1 to 2 years for a doctor with good tactile skills.

**If the doctor or therapist insists on treating the case without proper training, aggravation of the condition is likely. For this reason, attempting this protocol without proper training must be considered inappropriate on the part of the treating professional and a potential risk to the patient.**

The treatment method used for these cases is quite simple in concept. The affected tissue is trapped while the body part is moved, taking the tissue from its shortened to elongated position. Relative motion between tissues is introduced in order to restore gliding between those tissues. **Eight critical guidelines are followed** (2). These ensure maximum results, minimize discomfort, and avoid complications. Each treatment site is unique and requires specific alterations in technique. If these are not followed, results are compromised and the chance of inducing complications is strong.

The treatment protocol has been suggested as follows:

It is suggested that this protocol be completed before surgical intervention is attempted.

Treatment should be initiated at the onset of symptoms or at the earliest available time.

Symptoms include:

Muscles tighten excessively during activity

Excessive fatigue during normal tasks

Paresthesias develop

Numbness

Muscle atrophy/weakening, dropping objects

Pain, lymphatic edema

Symptoms usually start in the forearm or hand and progress through the entire upper extremity to the paraspinal area last.

Inflammation should be reduced by standard methods (rest, ice, pulsed ultrasound, etc.) before manual treatment begins in earnest.

Bracing is removed as quickly as possible unless necessary to accomplish a short-term task. This prevents hypertonic muscles from “fighting” the brace.

The workstation should be evaluated. Shoulder support is much more critical than wrist support. Back support should be firm and not spring supported. Check relaxation of the trapezius, levator scapulae and serratus posterior muscles during work activity.

Active Release Techniques is used. These use levels 3 and 4 active methods, previously described by Leahy and Mock (2-4). Symptom and objective improvements should be evident in the first to the third treatments. A 50% improvement within this timeframe is common and should be expected.

Specific stretching is taught and should be accomplished at least every waking hour by the patient in order to minimize or reduce tissue tension. Patient instruction sheets must be given to ensure the most accurate reproduction of stretching moves. It is the experience of the author that this is essential to the successful resolution of the case. Many patients forget to stretch or cannot remember the stretches without a visual aid.

Soreness after treatment should be minimal. Treatment intensity and frequency are adjusted to accommodate for tissue recovery. Normal protocol is treatment on alternate days.

If the case is not resolved by the sixth visit, a reevaluation is made and appropriate changes in treatment are initiated. Very often, slow progress is caused by outside influences such as sleeping on the extremity or recreational activity that exacerbates the condition. Careful patient consultation is required.

Active exercise to include sports activity is usually begun after the second visit as long as inflammation is not a problem.

If the case is not resolved in 12 visits, a reevaluation with a second doctor is done. If the case requires more than 12 visits, a reason or complicating factor must be identified to explain the slow response. If this cannot be found a choice between MMI, alternate or combined treatment protocol, and referral for other procedures should be considered. If treatment is continued, a definitive estimate of length of treatment and outcome must be available.

After release from treatment, the stretching program should be continued. Although the physical problem is solved, there is every reason to believe that reinjury is possible by the same mechanisms that caused the first injury. It has been the experience of the author that, with proper case management, over 90% of the cases do not recur within a 2-year follow-up. If reinjury occurs within the first year after treatment and all guidelines have been followed, the patient should be put on a preventive program that includes treatment at a frequency of approximately once every 3 months. A centralized RSD, if present, must be treated with an altered protocol and odds of resolution drop.

Over a period of several months, 223 cases were tracked. The results are summarized below. A successful resolution is defined as one that returns to full work capacity with little or no discomfort and requires no maintenance treatment (see Table 2).

**Table 2**  
**Results of case load**

<b>Total Cases</b>	<b>Successful</b>	<b>Average No. of Treatments</b>	<b>Success Rate (%)</b>	<b>Average \$ Per Case</b>
<b>223</b>	<b>215</b>	<b>6</b>	<b>96.4</b>	<b>490</b>

There were several recurrences. These occurred in 4% of the cases and were during the first year after treatment. One-half of the recurrences were with individuals who did not continue the stretching regimen. Treatment after recurrence involved an average of two visits.

New cases are now being seen at a rate of over 70 per month. The success rates and other statistics stated above are holding true. Within a year, a statistical analysis on over 1,000 cases should be available. An analog pain and interference scale will be added to each case for the evaluation of treatment effectiveness. A controlled study is now being implemented with the state of Colorado and a major university.

**References**

Gerr F, Letz R, Landrigan PJ. Upper-extremity musculoskeletal disorders of occupational origin. *Annu Rev Publ Health* 1991; 12:543-566.

Leahy PM, Mock LE. Myofascial release technique and mechanical compromise of peripheral nerves of the upper extremity. *Chiropract Sports Med* 1992; 6:139-150.

Leahy PM, Mock LE. Altered biomechanics of the shoulder and the subscapularis. *Chiropract Sports Med* 1991; 5:62-66.

Leahy PM, Mock LW. Synoviochondrometaplasia of the shoulder: a case report. *Chiropract Sports Med* 1992; 6:5-8.

Parachuri R, Adams M. Entrapment neuropathies. *Postgrad Med* 1993; 94:0.

Hunter JM. Recurrent carpal tunnel syndrome, epineural fibrous fixation, and traction neuropathy. *Hand Clin* 1991; 7:491-504.

Hartz CR, et al. The pronator teres syndrome: compressive neuropathy of the median nerve. *J Bone Joint Surg* 1981; 63A:885-890.

Scarpelli DG, Chiga M. Cell injury and errors of metabolism. In: Anderson WAD, Kissane JM, eds. *Pathology*. 7<sup>th</sup> ed. St. Louis: C.V. Mosby, 1977:90-147.