

## The Neurology of Carpal Tunnel Syndrome

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Carpal tunnel syndrome (CTS) is one of the more common neurological diagnoses we are likely to encounter in our office. As physicians, we can all provide the same answers in regard to the clinical questions and pathophysiology of CTS: It is a median nerve entrapment at the flexor aspect of the wrist; it produces pain, numbness and weakness that might be excruciating at the thumb, forefinger and sometimes the middle finger of the affected hand; and surgical decompression of the carpal tunnel generally might be the recommended treatment of choice. Let's take a closer look at CTS and hopefully gain a better understanding of what is really going on with some of these cases.

Entrapment can and does occur at the carpal tunnel, just not with the frequency with which it's diagnosed. The neurophysiological reality is that we live in a very flexor-based world. Most of us tend to use our flexor muscles more than our extensor muscles, such as in the acts of eating, typing, using a computer mouse, playing piano or doing massage. Not many of us can play the piano well with our extensor muscles. The flexor muscles, due to bulk and attachments, are much stronger and thus bear the brunt of hand and forearm work. With this extended activity, there can very well be hypertrophy of the flexor musculature such that compression of the median nerve at the carpal tunnel occurs and symptoms manifest.

Having treated many of these cases, I will tell you most people do not have Popeye-like forearms in addition to their nerve entrapment. Due to the fact we are using our flexor muscles more than our extensor muscles, there is greater likelihood that disynaptic-postsynaptic inhibition of the extensor muscles will occur at the level of the spinal cord, producing a physiological weakness of the extensor group and increasing the likelihood of flexor compression.

This is a normal process that happens to allow fluidity of movement. If my triceps and biceps contract at the same time, I do not get good flexion or extension of the arm. When I contract my biceps, the triceps must be inhibited at the level of the spinal cord to allow fluid movement to occur. If we are using our flexor muscles over and over, that same inhibition is occurring at the level of the cord such that our extensor muscles also undergo inhibition.

If this process occurs continuously throughout the day, it's understandable how an increased flexor response can produce a muscle contraction in the flexor group that is now facilitated, and how inhibition of the extensor group allows further facilitation of the flexor group to occur. Over time (and perhaps not too long a time, depending on the previous stability of that extensor group), we can develop such an unopposed facilitation of the flexor group that compression occurs.

My treatment for these patients is to inhibit the flexor group with a fast stretch of their muscle fibers, promoting direct inhibition of the muscles. Most importantly, they are given extensor exercises to allow re-strengthening of the opposite muscles, which will allow the flexor area of the wrist to be decompressed naturally. These exercises are as simple as the lifting of a 3- to 5-pound weight for three sets of 20-30 repetitions. We do not need the muscles to be exceedingly strong. The application of the exercises is to promote facilitation of the extensor group to the extent we will inhibit the flexor group and thus minimize the likelihood of compression. Coupled with proper muscle work and adjusting, the symptoms resolve rather quickly and stability is promoted long-

term. I recommend the patients do the exercises at least two times a week once they improve to maintain good function of the affected areas and decrease the likelihood irritation to the nerve will occur with continued use.

The next matter to be discussed is in regard to whether decompression is even necessary. When a peripheral nerve is compressed, a myriad of signs and symptoms must be present. It amazes me how many patients I see who come with a special diagnosis of "this or that nerve being pinched," but upon examination, no signs or symptoms of actual compression are present.

When a nerve is compressed, the first thing compromised are large-diameter afferent pathways of the "A" fibers of the nerve. These control fine, discriminating touch and motor function. The reality is that if a patient has had long-standing compression of a nerve, there must be muscle atrophy and loss of vibration and/or two-point discrimination.

If these signs and symptoms are not present, the likelihood of a true compression existing is greatly diminished. If it is a relatively new symptom, it might take two to three weeks before signs of atrophy and weakness are noticeable, but even then, a needle EMG will show signs of de-nerivation. I would expect, with true CTS, to see some loss of sensation along the thumb and forefinger, as well as some weakness of the muscles thereof. In the absence of these signs and symptoms, I would start to question the diagnosis.

Differentials can include a median nerve irritation at the elbow, a brachial plexus irritation (these previous sites of compression were sometimes known as the "double and triple crush" areas), or even vascular reasons for compression. If the compression is bilateral, I would start to look to possible systemic causes of inflammation such as a nutritional deficiency (often B6) or a problem with blood perfusion. Regardless, to have true CTS, one must find the real signs of compression or, in their absence, consider an alternate diagnosis.

There are great treatment protocols for CTS including cold-laser treatment, friction massage, etc. In the end, I think everything is good and effective, but the neurological state must be balanced if a patient is expected to achieve long-standing relief and eliminate the need for surgery. Finally, I am not a big advocate of the CTS brace commonly prescribed by both the medical and chiropractic professions. They can provide symptomatic relief due to the forced extended state the wrist is placed in, but the neurological consequence of a soft, static stretch on any tissue is an increased facilitation of that stretched muscle. In other words, we actually will increase the tension of the flexor group in question when wearing the brace for an extended period of time, which more likely than not will impede long-term stability.

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