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Effects of Prolonged Wrist Flexion on Transmission of Sensory Information in Carpal Tunnel Syndrome

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Abstract: Carpal tunnel syndrome presents a constellation of symptoms which include discomfort (eg, pain, paraesthesia) and diminished sense of touch. This exploratory study simultaneously measured changes in tactile threshold and discomfort ratings during prolonged wrist flexion in symptomatic patients from a rehabilitation clinic and from a control population. Prolonged (15 min) wrist flexion significantly increased tactile threshold and discomfort ratings above baseline levels in both symptomatic and control populations. Sixty-two percent of the symptomatic sample was found to have abnormal conduction latency. Tactile threshold in symptomatic subjects with normal conduction latency (n = 13) did not differ significantly from control subjects (n = 36) at baseline but showed significant elevation during wrist flexion. In contrast, subjects with abnormal conduction latency (n = 21) exhibited significant elevation relative to control subjects at baseline and throughout wrist flexion as well as a slower recovery after flexion. Conduction latency correlated with baseline (r = .52, *P* < .0001) and 15-min (r = .67, *P* < .0001) tactile threshold for the entire subject population, as well as 15-min threshold (r = .53, *P* = .013) for the subpopulation with abnormal conduction latency. At 2.5 min after flexion, correlation was significant for whole (r = .64, *P* < .0001) and abnormal conduction latency (r = .58, *P* = .0063) samples. Regression slope of tactile threshold versus conduction latency was significantly greater than zero and did not differ significantly from linearity. The study demonstrates that increases in mechanosensory threshold and discomfort ratings during prolonged wrist flexion are more profound (and recovery less rapid) in patients with electrophysiologic evidence of injury.

***Perspective:** This study demonstrates a provocative procedure that enhances the symptoms of carpal tunnel syndrome. This measure may help clinicians discriminate median nerve compression from other types of peripheral nerve injury and help researchers investigate the impact of mechanical stress, tissue compression, and vascular stasis on compression-related neuropathy.*

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***Key words:** Nerve entrapment, mechanoreceptor, tactile, conduction latency.*

Carpal tunnel syndrome results from median nerve entrapment at the level of the wrist and is the most frequently encountered compressive neuropathy in clinical practice. When confirmed by electrophysiologic assessment, its prevalence is about 3% among women and 2% among men.[2]

Symptomatic carpal tunnel syndrome can be defined on the basis of "primary" and "secondary" symptoms,[48,64] with primary symptoms being numbness, tingling, and nocturnal complaints and secondary symptoms being pain, weakness, and clumsiness. Primary symptoms are considered more specific to nerve injury, and secondary symptoms are more reflective of soft tissue and other musculoskeletal disorders. Generally, carpal tunnel syndrome worsens with time and can be correlated to histopathologic and pathophysiologic degrees of nerve injury.[21,55,36,8,44,43,47] First-degree injury involves nerve slowing due to conduction block and possi-

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bly focal demyelination (without axonal injury) which leads to full recovery. The majority of carpal tunnel patients fall into this category.[43] Second-degree injury involves axonal injury, thus requiring some degree of nerve regeneration with the potential for full recovery. In third-degree injury, nerve function is permanently diminished and degree of recovery is dependent on the amount of scar tissue formation. This would correspond to severe carpal tunnel syndrome. Fourth- and fifth-degree injuries involve severe scar tissue formation or transection, respectively, with expectation of irreversible dysfunction.

Magnetic resonance imaging has increased our understanding of carpal tunnel configuration during wrist movement.[8,36] In flexion, the tunnel's cross-sectional area decreases (ie, flattens) and the distance between the proximal edge of the transverse carpal ligament and the distal radius decreases.[47] In addition, because the median nerve is located between the flexor tendons and carpal ligament, increase in tendon tension is thought to increase pressure on the nerve during flexion.[14] Decreased cross-sectional area increases interstitial pressure. For example, with the wrist in neutral position, normal carpal tunnel pressure is approximately 2.5 mm Hg, and it increases to about 30 mm Hg with maximum flexion.[20] At this pressure epineural blood flow decreases.[53] Above 30 mm Hg, axon transport is impaired and subjects report mild paresthesias.[16] In carpal tunnel patients, average pressure with the wrist in neutral position is about 32 mm Hg.[20] At sustained pressures, the effects on median nerve function become more deleterious, including epineural edema, axon transport block, intraneural ischemia, and endoneural edema.[23,54]

Elevated carpal tunnel pressure increases sensory threshold of fingertips innervated by the median nerve via suppression of action potential transmission.[22,56] Increased threshold is likely due to conduction blockade in the carpal tunnel[27] or to increased action potential dispersion (due to conduction slowing) which reduces postsynaptic potential amplitude and thereby efficacy of synaptic transmission. Because most arteries that perfuse the palm and fingers bypass the carpal tunnel, an acute increase in carpal tunnel pressure is unlikely to alter cutaneous receptor perfusion and therefore mechanoreceptor response is likely to remain normal.

Basing carpal tunnel syndrome diagnosis solely on signs and symptoms can lower reliability, because other disorders such as tendonitis and cervical radiculopathy may present similarly. Quantitative electrophysiologic or sensory testing can confirm the clinical diagnosis; however, psychophysical evaluation with the wrist in a neutral position is not specific for carpal tunnel syndrome, because nerve injury could be due to varied causes (eg, diabetes or cervical radiculopathy). In contrast, differentially comparing threshold before and during a provocation that increases carpal tunnel pressure has been hypothesized to increase sensory evaluation specificity.[6,24]

Our experimental approach was to explore in greater detail than previous studies change in discomfort and tactile threshold during prolonged wrist flexion by comparing a symptomatic carpal tunnel syndrome sample to a nonsymptomatic control. Our hypothesis was that the symptomatic sample would show significantly greater shift in threshold and discomfort from baseline level than the control. During analysis, the symptomatic population was parsed into subsets with normal and abnormal conduction latency to analyze the relationship between baseline latency and shifts in tactile threshold and discomfort during provocation. In addition, tactile threshold on the middle finger (median nerve distribution) was compared to the little finger (ulnar distribution) and to the palmar branch of the median nerve in control and patient populations.

Materials and Methods

This study was approved by the Institutional Review Board at the University of Utah, and an informed consent form was provided for each subject. Subjects had to read and sign the consent form before participating in the experiment. A control sample was recruited from students at the University of Utah. All but 1 control subject had negative nerve conduction latency (see below). All had no history of peripheral neuropathy and no prior injury to the hand or wrist. Patients entering a rehabilitation clinic were selected based on physician assessment of their presenting symptoms as well as medical and work histories. Conduction velocity measurement was not used for inclusion in the patient population. Therefore, the basis for inclusion was presentation with wrist-related symptomatology.

Data were collected in a standardized sequence: questionnaire, clinical signs (Phalen and Tinel), nerve conduction latency, and tactile threshold before, during, and after prolonged wrist flexion. The questionnaire included anthropometric and demographic data such as gender, age, height, weight, and hand dominance, as well as ratings of past and present discomfort while awake and asleep. Subjects estimated their hand discomfort on a 0 to 10 visual analog scale (VAS, 0 = no discomfort, 10 = maximum imaginable discomfort). Phalen[9,39] and Tinel[9,18,39] tests were conducted for all subjects using the 0 to 10 discomfort rating scale. Nerve conduction latency (NCL) was measured (NervePace model 200-VS; NeuMed, Pennington, NJ) from a point just proximal to the wrist to the digital nerve of the middle finger[42] (conduction distance, 14 cm). To minimize temperature-related changes in conduction latency,[15,37] skin temperature was maintained above 29°C. The criterion for a positive NCL test was nerve conduction latency greater than 310 μs, as defined by the manufacturer.

The vibrometer probe (flat, 1.0 mm diameter) was connected to a DC spring-mounted motor (model V2, beryllium copper suspension; Gearing & Watson, East Sussex, UK). A probe-mounted LED radiated light (.1 mm diameter beam) onto a position detector (.1 μm resolution, model S-3931-01; Hamamatsu, Bridgewater, NJ) to produce a voltage proportional to probe position. Probe excursion was controlled with DC negative feedback circuitry, and displacement was independently calibrated

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