

## **PROLONGED PROVOCATIVE WRIST FLEXION AS AN IMPROVED METHOD FOR CARPAL TUNNEL SYNDROME SCREENING**

Richard F. Seseck, University of Utah  
Robert P. Tuckett, University of Utah  
Donald S. Blowski, University of Utah  
Mehdi Khalighi, University of Utah

[r.seseck@m.cc.utah.edu](mailto:r.seseck@m.cc.utah.edu)

### **ABSTRACT**

The purpose of this study was to determine if changes in sensory threshold associated with prolonged, provocative wrist flexion could be used to enhance screening methods for carpal tunnel syndrome. Provocative wrist flexion was used help distinguish subjects with carpal tunnel symptoms from those without symptoms. Compression of the median nerve in the carpal tunnel can decrease the sensitivity of the fingertips. This study analyzed the effects of prolonged wrist flexion on the transmission of tactile sensory information (50 Hz vibration) and subjective discomfort ratings (visual analog scale, 0 – 10). Significant differences were found between symptomatic and asymptomatic populations. Information on the differences between symptomatic and asymptomatic subjects can be used to improve carpal tunnel screening methods that use sensory threshold as a component.

### **INTRODUCTION**

Carpal tunnel syndrome is often initiated by inflammation-related swelling of the wrist flexor tendons as they pass through the carpal tunnel. Swelling can be induced or exacerbated by repetitive motions, forceful exertions, awkward postures, and inadequate work-rest schedules. Force, posture, and repetition have been linked to carpal tunnel syndrome (Moore and Garg, 1995; Silverstein, Fine, and Armstrong, 1987). Some of these risk factors can have an immediate effect on the median nerve in the carpal tunnel. The purpose of this study was to determine if these effects could be used to enhance screening methods for carpal tunnel syndrome. Specifically, provocative wrist flexion was used help distinguish subjects with carpal tunnel symptoms from those without symptoms.

Carpal tunnel pressure increases during mechanical stress associated with hand, wrist, and finger posture and loading (Keir, Bach, and Rempel, 1998; Keir, Bach, and Rempel, 1998; Rempel, et al., 1998; Werner et al., 1997). The resulting inflammation can create ischemic conditions of reduced oxygen, metabolite build up, and changes in pH levels, which can compromise metabolically active structures such as the median nerve fibers. Compression of the median nerve in the carpal tunnel can decrease the sensitivity of the fingertips. Fingertip sensitivity can

be evaluated by shifts in the mechanosensory threshold of the skin to vibration (Gelberman et al., 1983; Szabo et al. 1984).

Prolonged wrist flexion temporarily decreases blood flow, increases carpal tunnel pressure, and has a measurable effect on finger tip sensitivity as measured using a vibrating source (Borg and Lindblom, 1988; Gerr et al., 1995). This study analyzed the effects of prolonged wrist flexion on the transmission of tactile sensory information (50 Hz vibration) and subjective discomfort ratings. Baseline tactile thresholds were measured with the wrist in a neutral posture and then every 2.5 minutes for 15 minutes of wrist flexion. A post flexion “recovery” threshold was taken with the wrist again in a neutral posture. The hypotheses studied were:

- H1: Prolonged wrist flexion will result in decreased fingertip sensitivity and, therefore, increased sensory thresholds
- H2: The effect on the sensory thresholds of subjects with carpal tunnel symptoms will be greater than that experienced by the symptom-free, control subjects.
- H3: Subjective discomfort will be greater for symptomatic subjects than for symptom-free, control subjects.

Information on the differences between symptomatic and asymptomatic subjects can be used to improve carpal tunnel screening methods that use sensory threshold as a component.

## **MATERIALS AND METHODS**

The Institutional Review Board (IRB) at the University of Utah approved this study, and each subject signed an informed consent form. Nerve conduction latency (NCL) was measured using a NervePace Model 200-VS (NeuMed, Inc.). NCL was measured from a point just proximal to the wrist to the digital nerve of the middle finger. Skin temperature was maintained above 29°C to minimize temperature-related effects on sensory measurements (Klinenberg, So, and Rempel, 1996). Nerve conduction latency greater than 310  $\mu$ s was considered a positive NCL test (criterion in Operating Manual for Model 200-VS, NeuMed, Inc., 1998).

Subjects were recruited from an occupational medicine clinic and from the student body at the University of Utah. Subjects were divided into two major groups: controls from the University of Utah and symptomatic subjects from the occupational medicine clinic who were being treated for carpal tunnel syndrome symptoms. None of the University students were symptomatic or seeking treatment for hand or wrist discomfort. The symptomatic subjects were subdivided into two groups: NCL positive (NCL+) and NCL negative (NCL-) based on the 310  $\mu$ s criterion.

A questionnaire was administered to all subjects before conducting the tests. The questionnaire included simple anthropometric and demographic data such height, weight, age, and gender as well as ratings of past and present discomfort. Discomfort was estimated using a 0 to 10 visual analog scale (0 = no discomfort and 10 = maximum imaginable discomfort). Subjects also rated discomfort on this visual analog scale with respect to Phalen’s test and Tinel’s sign.

A vibrometer with a 1.0 mm diameter probe and a vertical oscillation resolution of 0.1  $\mu$ m with respect to the finger rest surface was used to obtain sensory threshold at the tip of the middle

finger (Sesek et al., submitted 2003). Sensory threshold was measured using a simple up-down staircase procedure (Garcia, 1998;Kaernbach, 1991; Linschoten, et al., 2001). The stimulus was given at randomized times between 4 and 7 seconds. Failure to detect the stimulus within 2 seconds or false positive detection of the stimulus increased the amplitude of the next stimulus. A correctly identified stimulus was followed by a stimulus of lower intensity. Each threshold test ended after the stimulus amplitude decreased below and increased above the subject's threshold for two complete cycles. Threshold was recorded as the two-cycle average of reversal points. Measurement of sensory threshold typically required between 60 and 90 seconds.

Median nerve sensory latency was measured before beginning the sensory stimulus trial. The stimulus trial was in three phases: a baseline sensory threshold with the wrist in a neutral posture (Figure 1), 15-minute provocative flexion (Figure 2) with thresholds measured every 2.5 minutes, and a post-test recovery threshold taken with the wrist again in a neutral posture. Subjects positioned their wrists in a maximum voluntary, unforced flexion position with their finger on the stimulus probe for the 15-minute flexion trial. After the last flexion threshold was measured (time  $t=15$ ), the subject was allowed one minute to relax, massage, or move their wrist as they saw fit prior to a post-test measurement (neutral wrist posture as in Figure 1). Therefore, post-test measurement occurred at approximately  $t=17.5$  since the threshold measurement for  $t=15$  minutes typically took between 1 and 1.5 minutes prior to the wrist and recovery minute. At each sensory threshold measurement, subjects were asked to rate their discomfort using the 0 – 10 visual analog scale.



Figure 1. Vibrotactile Testing with Neutral Wrist Posture: Baseline and Post-test



Figure 2. Vibrotactile Testing with Provocative Wrist Posture: 15-minute flexion trial

Control subjects had no history of wrist trauma, upper-extremity cumulative trauma disorders, or peripheral sensory neuropathy. Control subjects were evaluated on their non-dominant hands. Symptomatic subjects were evaluated on the hand for which they were reporting symptoms. Two subjects had bilateral symptoms and were therefore evaluated on both hands. Since there was an age difference between symptomatic and asymptomatic subjects, an age correction was employed. This study used a probe design and stimulus algorithm very similar to previous studies and these data, collected on group of 105 asymptomatic subjects of varying ages, were regressed to create an equation for expected threshold for each subject based on their age (Hardy, et al., 1992; Horch, et al., 1992; Jimenez, et al., 1993; Ward and Tuckett, 1991). The expected threshold was subtracted from each subject's threshold to allow age-corrected comparisons between groups.

## RESULTS

This study included 68 adult subjects (70 individual hands). There were 36 asymptomatic control subjects (36 subjects, 36 hands – mean age 26.3 years, 18 – 52 years) with no history of carpal tunnel syndrome symptoms or severe injury to the hand or wrist. All controls except one had negative nerve conduction latency. There were 34 symptomatic subjects (mean age 37.0 years, 19 – 60 years) who were patients at an occupational medicine clinic and were in various stages of treatment for carpal tunnel syndrome symptoms. The symptomatic subjects were subdivided into NCL+ and NCL- subpopulations based on their nerve conduction latencies. There were 21 NCL+ hands in 20 subjects. There were 13 NCL- hands in 13 subjects. One clinic patient was NCL+ bilaterally and another was NCL+ on one hand and NCL- on the other hand. Data were gathered specifically for each hand on these two subjects (i.e., thresholds and subjective discomfort levels).

### Wrist Flexion Induced Changes in Tactile Threshold

For the control population, multiple comparisons of age-corrected tactile thresholds (see Methods) differed significantly ( $P < 0.0001$ , repeated measures ANOVA). Figure 3 shows the increase in sensory threshold over time. Post-test recovery threshold was  $2.0 \mu\text{m}$  ( $\pm 0.7 \mu\text{m}$  SEM) greater than pre-test baseline sensory threshold ( $p=0.008$ ).

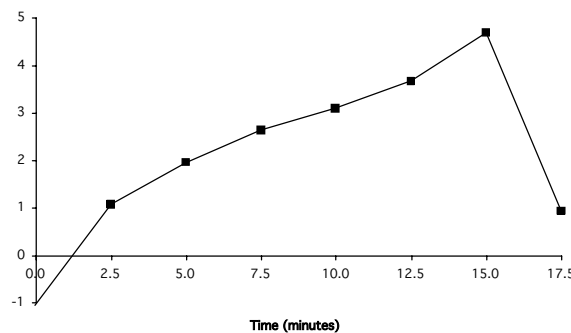


Figure 3: Control Subjects' Sensory Threshold vs. Time

For the symptomatic population, multiple comparisons of age-corrected tactile thresholds (see Methods) also differed significantly ( $P < 0.0001$ , repeated measures ANOVA). Figure 4 shows the increase in sensory threshold over time. Thresholds increased more dramatically than for control subjects and post-test recovery thresholds also remained significantly higher ( $6.2 \mu\text{m} \pm 1.1 \mu\text{m}$ , SEM) than pre-test baseline threshold ( $P < 0.0001$ ).

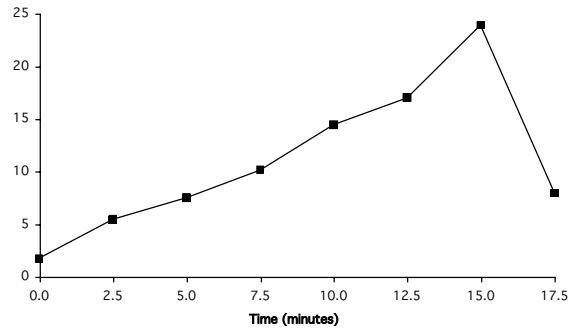


Figure 4: Symptomatic Subjects' Sensory Threshold vs. Time

For both control and symptomatic populations, tactile thresholds were significantly greater than baseline for all test flexion time intervals. These data are summarized in table 1. The first null hypothesis, that there would be no decrease in fingertip sensitivity (increase in tactile threshold) over time, was rejected. Therefore, the hypothesis that sensitivity will decrease over time was supported.

Table 1. Change in tactile sensitivity relative to baseline during prolonged wrist flexion

Time (min)	Control		Symptomatic	
	Difference ( $\mu\text{m} \pm \text{SEM}$ )	P	Difference ( $\mu\text{m} \pm \text{SEM}$ )	P
2.5	$2.1 \pm 0.4$	<0.0001	$3.7 \pm 0.9$	=0.0003
5.0	$3.0 \pm 0.5$	<0.0001	$5.9 \pm 1.0$	<0.0001
7.5	$3.7 \pm 0.7$	<0.0001	$8.3 \pm 1.4$	<0.0001
10.0	$4.2 \pm 0.8$	<0.0001	$12.3 \pm 2.4$	<0.0001
12.5	$4.7 \pm 0.8$	<0.0001	$15.3 \pm 2.8$	<0.0001
15.0	$5.8 \pm 1.2$	<0.0001	$22.2 \pm 4.6$	<0.0001

Comparing the difference in thresholds at each time interval, demonstrated significant differences between the symptomatic and control populations. These data are summarized in Table 2. The second null hypothesis, that the effect on sensory threshold would not differ between symptomatic and control populations was also rejected. Populations differed significantly at each time interval and the difference between groups increased steadily over time. Therefore, the second hypothesis, that the effects of prolonged flexion would be greater on symptomatic than control populations was also supported.

Table 2. Differences in tactile threshold between sample populations

Symptomatic minus Control		
Time (min)	Difference ( $\mu\text{m} \pm \text{SEM}$ )	P
0	2.8 $\pm$ 0.8	=0.0014
2.5	4.4 $\pm$ 1.3	=0.0011
5.0	5.6 $\pm$ 1.5	=0.0003
7.5	7.6 $\pm$ 1.9	=0.0002
10.0	11.4 $\pm$ 2.9	=0.0002
12.5	13.4 $\pm$ 3.2	<0.0001
15.0	19.3 $\pm$ 4.9	=0.0002

The symptomatic subjects were sub-divided into NCL-, or “at risk” for carpal tunnel syndrome, and NCL+ or “high risk” for carpal tunnel syndrome subgroups. These symptomatic subgroups are shown in Figures 5 and 6.

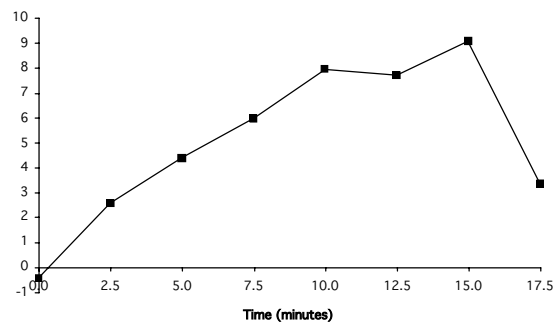


Figure 5: At Risk (NCL-) Symptomatic Subjects' Sensory Threshold vs. Time

At risk (NCL-) subjects differed significantly from control subjects' measures between 5 minutes and 12.5 minutes, but were not significantly different at baseline, the beginning or end of the provocative flexion trial, or at post-test recovery. High-risk (NCL+) subjects differed significantly from controls at all time intervals and from at risk (NCL-) subjects at baseline and from 12.5 minutes through post-test recovery. Table 3 compares the sensory thresholds of controls, at risk, and high-risk populations at each time interval.

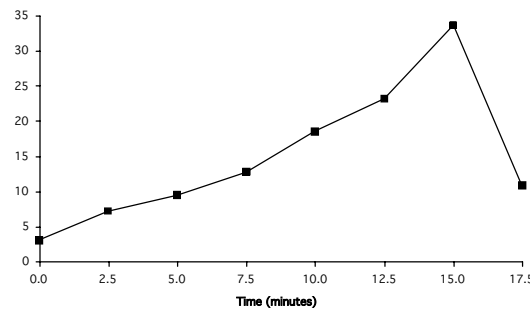


Figure 6: High Risk (NCL+) Symptomatic Subjects' Sensory Threshold vs. Time

Table 3: Differences in tactile threshold between sample populations

Time (min)	High Risk - Control		At Risk - Control		High Risk – At Risk	
	Difference (µm, m ± SEM)	P	Difference (µm, m ± SEM)	P	Difference (µm, m ± SEM)	P
<b>Baseline</b>	4.1 ± 1.0	<0.0001	0.6 ± 0.6	ns	3.5 ± 1.6	=0.0321
<b>2.5</b>	6.2 ± 1.5	<0.0001	1.5 ± 1.0	ns	4.7 ± 2.5	ns
<b>5.0</b>	7.5 ± 1.7	<0.0001	2.4 ± 1.2	=0.0462	5.1 ± 2.8	ns
<b>7.5</b>	10.2 ± 2.2	<0.0001	3.3 ± 1.6	=0.0404	6.8 ± 3.7	ns
<b>10.0</b>	15.4 ± 3.4	<0.0001	4.9 ± 1.8	=0.0105	10.5 ± 5.7	ns
<b>12.5</b>	19.5 ± 3.6	<0.0001	4.0 ± 1.8	=0.0272	15.4 ± 6.1	=0.0162
<b>15.0</b>	28.9 ± 5.6	<0.0001	4.4 ± 2.3	ns	24.6 ± 9.3	=0.0129
<b>Recovery (17.5)</b>	9.9 ± 2.0	<0.0001	2.4 ± 1.5	ns	7.5 ± 3.1	=0.0197

Figure 7 shows sensory thresholds over time for control, at-risk, and high-risk populations. At 12.5 minutes and 15 minutes, high-risk subjects have an abrupt increase in threshold that the other groups do not. They also do not recover as close to pre-test levels as the other groups. Table 4 shows the comparison of baseline to post-test recovery threshold for these groups.

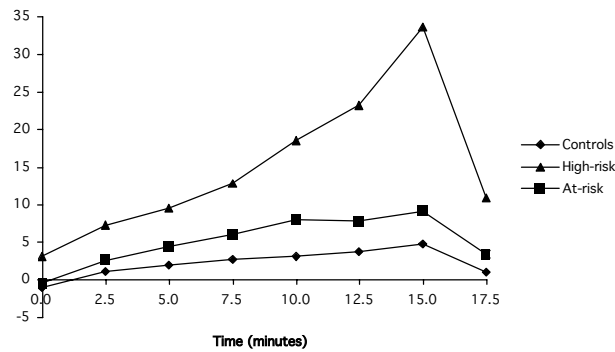


Figure 7: Controls, At Risk, and High Risk Subjects' Sensory Threshold vs. Time

Table 4. Comparison of Pre-test Baseline and Post-test Recovery Thresholds

	Middle finger µm (SEM)		
	Baseline	Recovery	Difference
<b>Control</b>	-1.1 (0.3)	0.9 (0.7)	2.0 P=0.008
<b>High-risk</b>	3.0 (1.2)	10.8 (2.2)	7.8 P=0.0002
<b>At-risk</b>	-0.4 (0.5)	3.3 (1.4)	3.7 P=0.0032

## Subjective Discomfort

Subjective discomfort during provocation through post-test recovery is compared in Figure 8. Control subjects were significantly different from symptomatic subjects at all time intervals ( $p < 0.0001$ ). High-risk and at-risk subjects differed significantly from 12.5 minutes through post-test recovery.

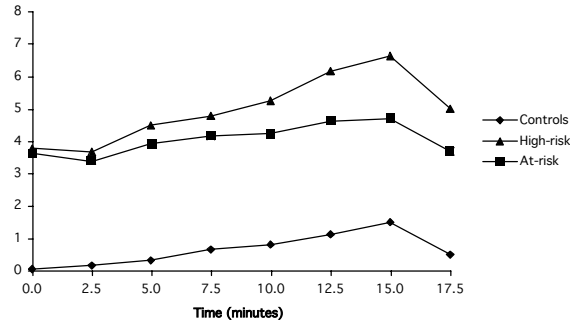


Figure 8: Controls, At Risk, and High Risk Subjects' Discomfort vs. Time

Table 5 summarizes differences in subjective discomfort between groups. The null hypothesis, that subjective discomfort will not differ between control and asymptomatic populations, was rejected. Therefore, it seems that discomfort during provocation can be used to distinguish controls from symptomatic subjects.

Table 5: Differences in discomfort ratings between sample populations

Time (min)	High-risk - Control		At-risk - Control		High-risk - At-risk	
	Difference (units, $m \pm SEM$ )	P	Difference (units, $m \pm SEM$ )	P	Difference (units, $m \pm SEM$ )	P
<b>0 (Baseline)</b>	$3.7 \pm 0.3$	<0.0001	$3.6 \pm 0.2$	<0.0001	$0.1 \pm 0.6$	ns
<b>2.5</b>	$3.5 \pm 0.3$	<0.0001	$3.2 \pm 0.3$	<0.0001	$0.3 \pm 0.6$	ns
<b>5.0</b>	$4.2 \pm 0.3$	<0.0001	$3.6 \pm 0.3$	<0.0001	$0.6 \pm 0.6$	ns
<b>7.5</b>	$4.1 \pm 0.3$	<0.0001	$3.5 \pm 0.3$	<0.0001	$0.6 \pm 0.6$	ns
<b>10.0</b>	$4.4 \pm 0.4$	<0.0001	$3.4 \pm 0.4$	<0.0001	$1.0 \pm 0.6$	ns
<b>12.5</b>	$5.0 \pm 0.4$	<0.0001	$3.5 \pm 0.4$	<0.0001	$1.5 \pm 0.6$	<0.0155
<b>15.0</b>	$5.1 \pm 0.5$	<0.0001	$3.2 \pm 0.6$	<0.0001	$1.9 \pm 0.7$	<0.0081
<b>17.5 (Recovery)</b>	$4.5 \pm 0.4$	<0.0001	$3.2 \pm 0.4$	<0.0001	$1.3 \pm 0.6$	<0.0409

## **DISCUSSION**

While significant differences existed between high-risk symptomatic subjects and the other populations at baseline, prolonged flexion increased the differences between groups, particularly after 10 minutes of flexion. Also, high-risk subjects did not recover to pre-test baseline thresholds as quickly as the other groups. This may indicate both greater damage to the median nerve and a lessened ability to recover from mechanical insult, putting these persons at potentially higher risk of carpal tunnel syndrome particularly if there is inadequate recovery time. It would appear that discomfort during provocation can be used to distinguish controls from symptomatic subjects and that sensory threshold used to further distinguish persons at the highest risk for carpal tunnel syndrome from those that are symptomatic, but not yet NCL+. Further research into the effects of provocative flexion as well as other ergonomic risk factors on sensory threshold may lead to enhanced carpal tunnel syndrome screening methods. The relatively low cost and non-invasive nature of vibratory sensory threshold make it a promising tool for workplace ergonomic surveillance programs. Currently, research is underway to study other risk factors such as finger loading, direct pressure, and restriction of blood flow (via venous blood occlusion).

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