

MEASURING THE EFFECTS OF ERGONOMIC RISK FACTORS ON TACTILE SENSATION

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ABSTRACT

Pressure on the median nerve at the wrist can lead to decreased tactile sensitivity in the fingertips. People with carpal tunnel syndrome may often experience numbness, tingling, and decreased sensitivity in their finger tips. Compared to a control group, subjects symptomatic of carpal tunnel syndrome had a greater mean shift (decrease) in tactile sensitivity than the control group when exposed to certain ergonomic risk factors. These factors include wrist flexion, direct pressure on the transverse carpal ligament area of the wrist, and tendon loading. Additionally, the effect of slight venous occlusion in the forearm was studied. Inability to show significance between the groups and provocations at this time is likely due to high variance and low sample size in the symptomatic group. There is an increase in threshold during the recovery period after each provocation.

INTRODUCTION

Carpal tunnel syndrome (CTS) is caused by compression of the median nerve within the carpal tunnel. It is much more common (three times) in women than in men. It has been attributed to many conditions including anatomical anomalies, fractures, repetitive action, induced trauma, nerve sheath tumors, ganglions, circulatory disturbances, and others (Pećina et al., 2001). Due to its prevalence in occupations requiring repetitive motion, especially at high force or in awkward wrist postures, it is of interest to those studying ergonomics.

If diagnosed and treated early, permanent damage to the nerve may be avoided. Treatment may include immobilizing the wrist with a splint, discontinuing repetitive motion, use of anti-inflammatory medication, and corticosteroid injections. If symptoms continue, the transverse carpal ligament may be sectioned to allow for more space (hence less pressure) within the carpal tunnel (Pećina et al., 2001).

Several common diagnostic procedures exist. Tapping on transverse carpal ligament (Tinel's sign), placing the wrist in flexion (Phalen's sign), or use of a tourniquet may cause paresthesia within a subject (Gilliat, 1953). Direct pressure on the carpal tunnel has also been suggested (Durkan, 1991).

Additionally, nerve conduction studies are often used in the diagnosis of CTS. These require electrically stimulating nerves or muscles and using surface or imbedded electrodes to monitor nerve or muscle response. Conduction velocity is either sensory or motor. Sensory studies seek to find the velocity of conduction of the compound action potential within the nerve, while motor studies seek to measure the recruitment of muscle fibers to a given stimulus (a twitch in the muscle). In diagnosing CTS, sensory studies are preferable (Pećina et al. 2001, 6).

The purpose of the current research is to assess the effect of several ergonomic risk factors applied to the wrist. The effect is measured using the tactile sensitivity of the finger tips, as sensory deficit is an indicator of nerve compression. These mechanosensory threshold studies test the difference between the minimum distinguishable amplitude of vibration in the absence of the risk factors and the amplitude when these factors are present. Also, we hope to show a significant difference between how normal nerves react as opposed to those subjected to CTS and other peripheral neuropathys.

The current research compares four modes of provocation: prolonged wrist flexion, prolonged wrist flexion with tendon loading on the index and ring fingers, prolonged wrist flexion with direct pressure on the carpal tunnel region, and prolonged wrist flexion with venous occlusion at the forearm.

Improvements over nerve conduction studies may include improved accuracy (statistical sensitivity, selectivity, specificity) while causing less discomfort (physical and psychological) and the ability to monitor changes in the sensory transmission of the median nerve over time. Vibrotactile studies may also require less expertise to perform, though variance in results between examiners is beyond the scope of this study.

PROCEDURE AND RESULTS

Subjects were recruited by word of mouth and through flyers posted at medical clinics. Most of the test subjects to date were recruited by word of mouth. The control group consisted of 4 males and 6 females with a mean age of 29 years and a range of 21 to 60 years. Six symptomatic subjects have been recruited with a mean age of 45 and a range of 28 to 62. No subjects were excluded from the study, and none discontinued participation voluntarily. The study was approved by the University of Utah Institutional Review Board (IRB), and subjects read and signed a consent form.

Subject Screening

Common diagnostic procedures. Each subject completed a questionnaire. This requested information regarding current CTS symptoms, risk factors, and related injuries. They were also tested using Phalen's sign (maximum wrist flexion for 60 seconds) and Tinel's sign (gently tapping on the transverse carpal ligament area of the wrist/hand). None of the control subjects tested positive to either Phalen's sign or Tinel's. Four of the symptomatic subjects (67%) tested Phalen's sign positive, while two (33%) tested Tinel's sign positive.

Nerve conduction studies. Antidromic sensory nerve conduction latency was tested on each of the subjects using surface electrodes (Brevio manufactured by NeuMed, Inc., Pennington, NJ). This nerve conduction instrument reports whether the latency is within the normal range, but requires a 14 cm distance between the active electrode (placed on the middle finger) and the stimulator cathode. Since some subjects have longer hands, a longer distance was used and the instrument's report was not used. Instead, sensory nerve conduction velocity (sNCV) was found by dividing the distance by the latency. This value was compared to 41.26 m/sec (Oh, 1993).

Several factors can affect conduction velocity including age and temperature. It is suggested that a 2 m/sec per decade over 60 years allowance be given for subjects over 60 years old (Oh, 1993). However, the signal amplitude for the only subject over 60 (symptomatic) was not high enough to record the latency, so no age correction was used.

Some of the subjects, despite washing in warm water, had less than the recommended (31 to 34 °C) skin temperature. For these people, the nerve conduction velocity was corrected using a correlation suggested by DeJesus: $V_{\text{corrected}} = V_{\text{measured}} e^{0.0419\Delta T}$, where ΔT is the difference between the desired skin temperature and the temperature at the time of the measurement (Oh, 1993).

Two of the control group subjects had low temperature and low conduction velocities, but exceeded the 41.26 m/sec limit when temperature was corrected to 32 °C. The rest of the control group had velocities in the normal range. Two of the six symptomatic subjects exceeded this limit; one without correction, and one correction to 32 °C. Thus all control subjects and two symptomatic subjects were sNCV negative.

Test hand selection. The test hand for the control group was the least symptomatic (or non-dominant if both were equally asymptomatic). For the symptomatic group, the most symptomatic (or dominant if both were equally symptomatic) hand was chosen unless there were previous injuries on this hand unrelated to CTS.

Vibrotactile Studies

Mechanical sensitivity of the middle finger is measured using a computer-controlled vibrometer. The timing of probe vibration, amplitude, and duration between stimuli (50 Hz) were controlled by the computer. The subject pressed a button when a stimulus was sensed. The amplitude was decreased to find the smallest vibration sensed. This smallest vibration is the vibrotactile threshold.

The vibrotactile threshold was tested during four separate sessions with at least 24 hours between visits. At each session, a baseline threshold was found with the wrist in neutral posture (see Fig. 1). Then the threshold measurement was begun at the start of flexion (time 0) and at each 2.5 min interval while the wrist was placed in one of four provocations for 15 minutes. Following the measurement at 15 min, the subject was instructed to massage, shake, or otherwise relieve pressure and numbness for one minute. Three recovery measurements were then taken at 2.5 min intervals with the wrist in a neutral posture.



Figure 1. Baseline measurement with wrist in neutral posture



Figure 2. Test A: Wrist flexion

The four provocations, presented to the subject in randomized order, were A) wrist flexion (Fig. 2), B) wrist flexion with direct pressure on the carpal tunnel (Fig. 3), C) wrist flexion with tendon loading (Fig. 5), and D) wrist flexion with venous occlusion (Fig. 6).



Figure 3. Test B: Wrist flexion with direct pressure



Figure 4. Durkan Gauge in fixture



Figure 5. Test C: Wrist flexion with tendon loading



Figure 6. Test D: Wrist flexion with venous occlusion

Test A: Wrist flexion. One of the provocations was simply placing the wrist in flexion. The vibrometer was elevated and the elbow was supported by a foam pad (see Fig. 2). This has been studied in previous research (Khalighi, 2001; Sesek et al., submitted). Flexion of the wrist increases the pressure within the carpal tunnel, especially in the region of swelling in CTS subjects (Gelberman, 1981; Phalen, 1972). The result is decreased sensation within the region of the hand innervated by the median nerve, often in the tip of the middle finger. Hence, Phalen suggested wrist flexion as a diagnostic procedure (Phalen, 1972).

Test B: Wrist flexion with direct pressure. Another provocation combined wrist flexion with the application of pressure directly on the carpal tunnel region (see Fig. 3). Pressure was applied with a rounded (approx. 2cm diameter) probe on a Durkan Gauge (Gorge Medical; Hood River, OR; see Fig. 4). A gauge reading of 9 psi (this assumes a certain surface contact area; a 9 psi gauge reading was found to correspond to about 16.8 N or 3.8 lbf) was maintained throughout the 15 min of testing. Direct pressure is hypothesized to increase the interstitial pressure on the median nerve above that of flexion alone, much as edema.

Test C: Wrist flexion with tendon loading. Kempe studied the effect of tendon loading on fingertip sensory deficit (Kempe, 2002). In this test, however, tendon loading was coupled with wrist flexion. Loops were placed on the middle segment of the index and ring fingers. These loops were connected by a system of strings and pulleys to weights (see Fig. 5). The force was intended to increase tension on the tendons running through the carpal tunnel, thereby increasing the pressure on the median nerve.

Test D: Wrist flexion with venous occlusion. Understanding the effect of decreased blood flow on nerves is important, and is thought to be part of the reason that people with CTS experience pain at night (Sesek, submitted). It may be important also in distinguishing between CTS and other peripheral neuropathies such as that caused by diabetes. In this test, the wrist was placed in flexion as before and a pressure cuff was placed on the forearm. The pressure was raised to 15 mm Hg to slightly occlude the veins (see Fig. 6), causing hypoxia.

Results

Figure 7 shows the trend in the data with error bars representing the standard error of the mean at each time. A generally increasing trend was seen among both groups during the fifteen minutes of provocation. This was followed by a reduction at the first recovery point then another general increase in threshold over the last two recovery points.

None of the control group thresholds exceeded 39 μm on any test. However, several in the symptomatic group exceeded the limit of the machine (over 400 μm). This contributed to large variance in the symptomatic group data. While this prevents demonstrating statistical significance, it shows the expected trend.

The data were adjusted such that thresholds above 50 μm were set equal to 50. This accounts for 21 observations, all among symptomatic subjects. The data plots (see Fig. 8) show the same trend, but the variance in the symptomatic group is reduced.

Some threshold measurements took much longer than 2.5 min to run. When this occurred, the next measurement was skipped so that the following one could be started on time. This occurred twice at the 12.5 min period among symptomatic subjects (with particularly high thresholds) during Test B. Exclusion of these points caused the mean threshold to drop drastically between the 10 and 15 min means, so a valley appeared on the plot at 12.5min. Because of this, the Test B data for these two subjects were excluded from the plots.

The data were compared to see if the differences in the means at each time were significant. Repeated measures ANOVA showed that there was significant difference in the normal data (for A, C, and D $p < 0.0001$, for B $p = 0.0036$). The symptomatic data were compared using a nonparametric ANOVA because of the significant differences in variance among the groups. The difference between times in each test was once again significant ($p < 0.03$ for each).

Further, for each test, the mean threshold at each time was compared to the baseline. The values of these comparisons are shown in Tables 1-4. Then the mean threshold at each time was compared to the threshold for the previous time. Because all groups of data (for each test at each time within each study group) passed normality tests, paired t-tests were performed when making comparisons within study groups.

Comparing the mean threshold at each time to the baseline showed significant difference at all times for each test except for test B at 0 ($p = 0.2889$), 2.5 ($p = 0.0969$), 7.5 ($p = 0.051$), and 12.5 min ($p = 0.1048$) for the control group. In the symptomatic group, few comparisons were significant.

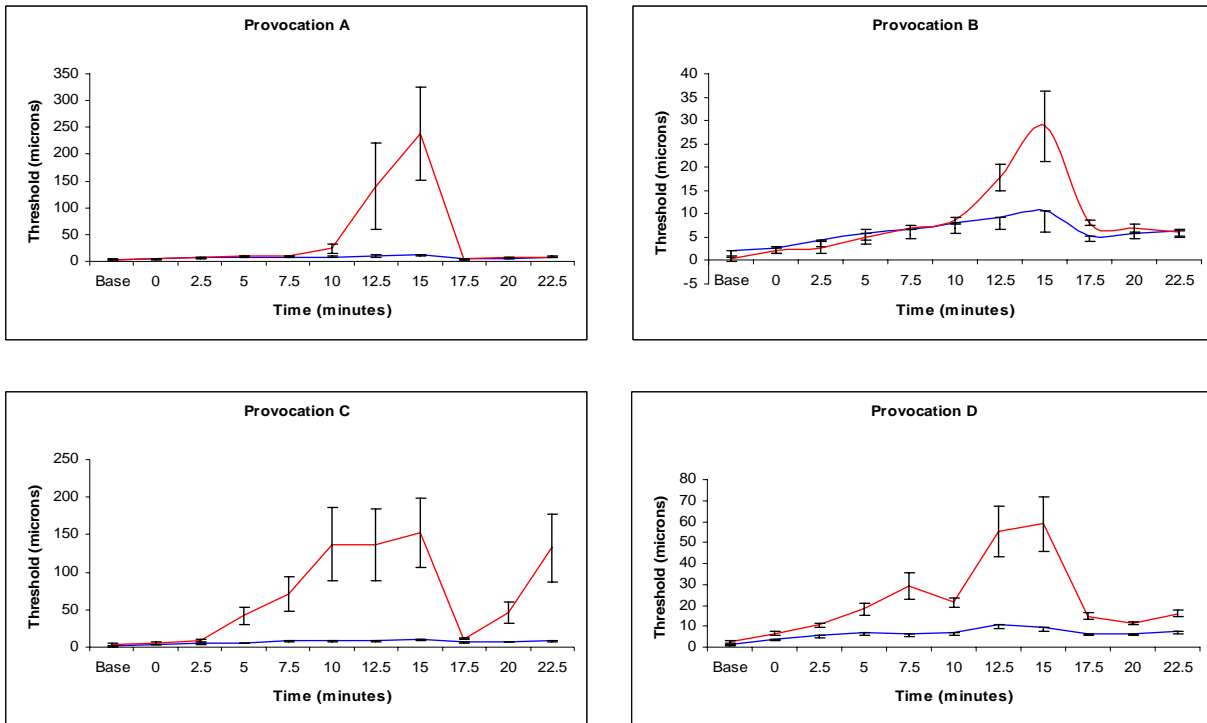


Figure 7. Threshold vs. time for each provocation. Symptomatic data are represented by the top line.

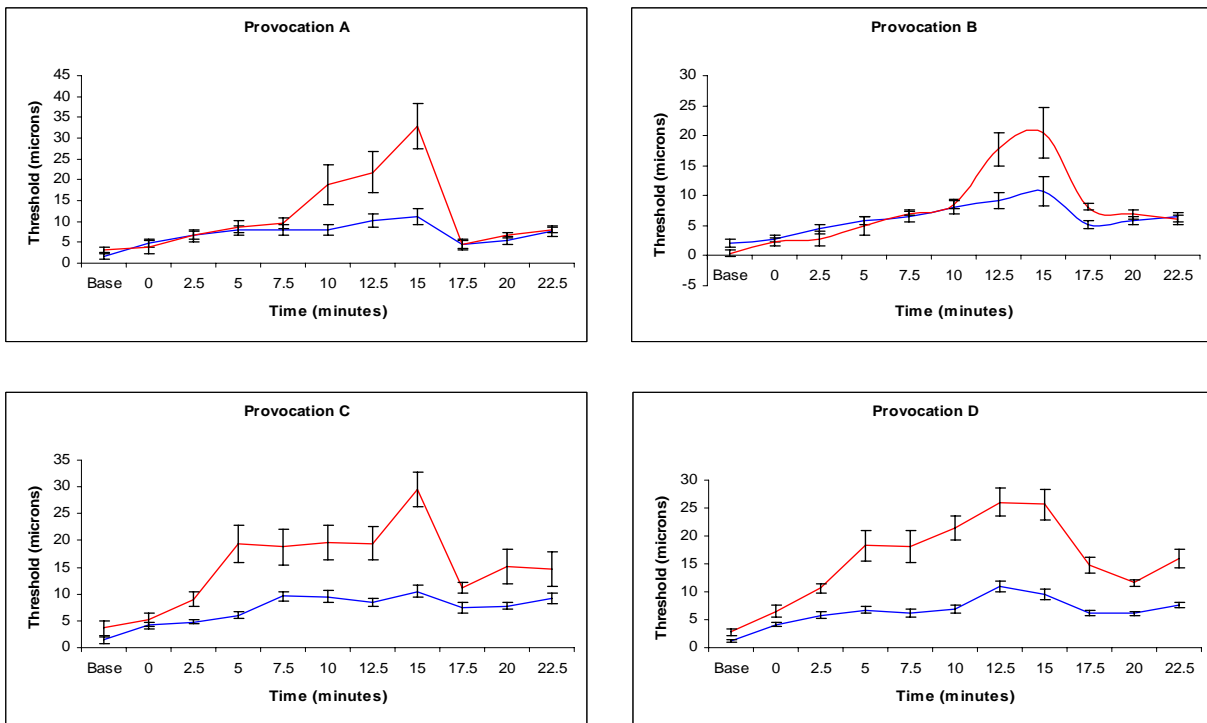


Figure 8. Adjusted data. Values over 50 were assigned a value of 50. The top line in each represents the symptomatic group.

Table 1. Threshold relative to baseline: Flexion.

Time (min)	Control Group		Symptomatic Group	
	Difference ($\mu\text{m} \pm \text{SEM}$)	P - Value	Difference ($\mu\text{m} \pm \text{SEM}$)	P - Value
0.0	3.19 \pm 1.11	0.0184	0.7167 \pm 1.44	0.6404
2.5	5.22 \pm 0.71	<.0001	3.45 \pm 1.51	0.0709
5.0	6.3 \pm 0.85	<.0001	5.6 \pm 1.31	0.0078
7.5	6.44 \pm 1.02	0.0001	6.55 \pm 1.62	0.01
10.0	6.58 \pm 1.49	0.0017	20.95 \pm 11.29	0.1226
12.5	8.67 \pm 1.65	0.0005	136.78 \pm 124.16	0.3324
15.0	9.71 \pm 2.23	0.0018	235.75 \pm 126.55	0.1215
17.5	2.93 \pm 1.09	0.0251	1.417 \pm 1.93	0.4959
20.0	3.86 \pm 0.96	0.003	3.6 \pm 1.42	0.0519
22.5	6.08 \pm 1.4	0.0019	4.75 \pm 1.12	0.0082

Table 2. Threshold relative to baseline: Flexion and direct pressure.

Time (min)	Control Group		Symptomatic Group	
	Difference ($\mu\text{m} \pm \text{SEM}$)	P - Value	Difference ($\mu\text{m} \pm \text{SEM}$)	P - Value
0.0	0.74 \pm 0.66	0.2889	3.75 \pm 3.82	0.3709
2.5	2.37 \pm 1.28	0.0969	3.317 \pm 4.35	0.4776
5.0	3.72 \pm 1.01	0.005	7.333 \pm 4.88	0.1932
7.5	3.35 \pm 1.49	0.051	13.367 \pm 6.07	0.0787
10.0	6.05 \pm 1.47	0.0026	123.6 \pm 101.66	0.2783
12.5	4.29 \pm 2.38	0.1048	17.375 \pm 6.54	0.0743
15.0	8.65 \pm 3.5	0.0356	132.82 \pm 99.88	0.241
17.5	3.11 \pm 0.72	0.002	8.35 \pm 2.52	0.0211
20.0	3.85 \pm 0.85	0.0014	5.833 \pm 1.53	0.0124
22.5	4.36 \pm 1.28	0.0077	6.4 \pm 2.67	0.062

Table 3. Threshold relative to baseline: Flexion and tendon loading.

Time (min)	Control Group		Symptomatic Group	
	Difference ($\mu\text{m} \pm \text{SEM}$)	P - Value	Difference ($\mu\text{m} \pm \text{SEM}$)	P - Value
0.0	2.77 \pm 0.95	0.0168	1.52 \pm 1.51	0.3716
2.5	3.44 \pm 1.09	0.0118	5.34 \pm 2.43	0.093
5.0	4.7 \pm 0.67	<.0001	38.2 \pm 27.74	0.2405
7.5	8.18 \pm 1.84	0.0016	67.38 \pm 57.54	0.3066
10.0	8.14 \pm 2.07	0.0035	133.4 \pm 122.57	0.3376
12.5	7.09 \pm 1.56	0.0014	133.28 \pm 122.59	0.3381
15.0	9.15 \pm 2.01	0.0014	148.98 \pm 119.16	0.2793
17.5	6.04 \pm 1.4	0.0019	7.58 \pm 0.4	< 0.0001
20.0	6.38 \pm 0.6	<.0001	41.84 \pm 36.52	0.3159
22.5	7.83 \pm 1.54	0.0007	128.58 \pm 123.76	0.3575

Table 4. Threshold relative to baseline: Flexion and venous occlusion.

Time (min)	Control Group		Symptomatic Group	
	Difference ($\mu\text{m} \pm \text{SEM}$)	P - Value	Difference ($\mu\text{m} \pm \text{SEM}$)	P - Value
0.0	2.99 \pm 0.62	0.001	3.76 \pm 3.73	0.3705
2.5	3.60 \pm 1.37	0.0271	7.88 \pm 3.36	0.0791
5.0	5.57 \pm 1.11	0.0007	15.46 \pm 8.53	0.1442
7.5	5.11 \pm 1.35	0.0043	26.28 \pm 19.44	0.2478
10.0	5.84 \pm 1.48	0.0033	18.58 \pm 6.56	0.0472
12.5	8.28 \pm 2.58	0.0107	52.42 \pm 35.84	0.2174
15.0	8.45 \pm 2.19	0.0039	56.06 \pm 40.08	0.2344
17.5	5.15 \pm 0.84	0.0002	12.02 \pm 3.73	0.0323
20.0	5.00 \pm 0.59	<.0001	8.78 \pm 2.61	0.0281
22.5	6.53 \pm 1.04	0.0001	13.28 \pm 5.89	0.0872

When mean thresholds at each time were compared to the threshold at the time before, significant difference was seen in test A between 0 and 2.5 min and 15 and 17.5 min and in test C between 5 and 7.5 five min for the control group. For the symptomatic group, significant difference was seen in test C between 0 and 2.5 min and in test D between 0 and 2.5 min.

Between each test (provocation), the mean thresholds were compared at each time. Significant difference was seen between tests A and C at 2.5 and 20 min, and between B and C at 7.5 min. This does not statistically demonstrate that adding other risk factors to flexion causes a substantial change in the effect on the median nerve.

Control data for each provocation were compared to symptomatic data at each time interval. Comparisons were made using unpaired t-tests, allowing for unequal variance. The results are tabulated in Table 5. Though there are large differences between the groups at many of the time intervals during provocation, statistical tests do not show significance in these differences. This may also be attributed to the large variance in symptomatic data.

As discussed above, comparing within subject groups (symptomatic or control), there was not a significant difference between provocations at each time. Likewise, there is not a significant difference when comparing mean thresholds between groups for each provocation at each time, potentially because of the small sample size and large variance in symptomatic data. However, there does appear to be a difference between tests when comparing symptomatic mean thresholds to control thresholds for each test (see Table 5). For instance, at the 5 min measurement, the mean difference between symptomatic and control (symptomatic minus control) is 0.9003 μm for Test A, 3.504 μm for Test B, 35.778 μm for Test C, and 11.558 μm for Test D. This indicates that adding risk factors to flexion causes a greater separation between symptomatic and control data. We hope that increasing the symptomatic sample size will allow this to be statistically demonstrated.

Table 5. Threshold Comparisons: Symptomatic minus control

Time (min)	Flexion		Flexion + Direct Pressure		Flexion + Tendon Loading		Flexion + Venous Occlusion	
	Difference (μm)	P - Value	Difference (μm)	P - Value	Difference (μm)	P - Value	Difference (μm)	P - Value
Baseline	1.6	0.1082	-0.1097	0.9602	2.278	0.5216	1.668	0.4121
0.0	-0.873	0.6906	2.9	0.3984	1.028	0.7594	2.438	0.4494
2.5	-0.1697	0.9291	3.54	0.467	4.178	0.2738	4.851	0.1419
5.0	0.9003	0.6815	3.504	0.4609	35.778	0.3037	11.558	0.2365
7.5	1.71	0.4602	8.749	0.2178	61.478	0.3645	22.838	0.2976
10.0	15.97	0.23	117.44	0.3073	127.54	0.3659	14.408	0.0968
12.5	129.43	0.3585	8.523	0.3171	128.47	0.3628	44.191	0.2819
15.0	227.64	0.1335	124.06	0.2767	142.11	0.3084	49.278	0.2828
17.5	0.087	0.972	5.13	0.0507	3.818	0.2926	8.538	0.1272
20.0	1.34	0.4538	1.874	0.3126	37.738	0.3894	5.448	0.0469
22.5	0.2703	0.8936	1.93	0.4903	123.03	0.3853	8.418	0.1888

An unexpected but noteworthy trend occurred during recovery. The 17.5 min interval data show a decrease in tactile threshold from the 15 min threshold, though not statistically significant. However, the 20 and 22.5 data show a trend of increasing thresholds, and the mean thresholds at these times are significantly different from baseline in each test for the control group and for tests A at 22.5 min, B at 20 min, and D at 20 min for the symptomatic group. This may be caused by reactive hyperemia, and may have importance when considering work/rest cycles.

CONCLUSIONS

While the data show the expected trend during provocation—that the tactile threshold gradually increases but more for symptomatic subjects—statistical significance cannot be shown between the study groups because of extreme variance among the symptomatic subjects. With an increased sample size, better comparisons may be made.

It appears from the data collected thus far that the most effective risk factor in compromising tactile sensitivity in the fingertips is wrist flexion. This may be concluded because of lack of significance in most comparisons between each provocation at each time. Perhaps the data will show significance between provocations when more symptomatic subjects are tested.

The trend of increasing tactile thresholds during recovery may be due to reactive effects. It is more pronounced in some of the provocation, but is common to both control and symptomatic data. Without extending recovery measurements, it is unknown how long the trend will continue before the thresholds again approach the baseline.

We hope that testing more symptomatic subjects will also show which combination of risk factors will cause the greatest difference in tactile threshold shift between study groups.

SUGGESTIONS FOR FURTHER RESEARCH

Because of the unexpected trend in recovery data, future tests will extend the recovery threshold measurements until the threshold approaches baseline or level off. This data may help us understand the effects of the various ergonomic risk factors beyond during the period of provocation or exposure.

As part of this study, subjects with diabetic neuropathy are also being tested to see if these ergonomic risk factors affect these subjects differently than CTS-symptomatic subjects and the normal subjects. To date, only two subjects with diabetic neuropathy have been tested.

Wrist extension has been shown to have greater effect on carpal tunnel pressure than extension, but may not be as effective at provoking symptoms of CTS as flexion (Gelberman, 1981; Phalen, 1972). To understand the effect of wrist posture on the median nerve, extension should also be studied, though this posture will require a different vibrometer configuration.

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