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ORIGINAL ARTICLE

The effect of different warming methods on sensory nerve conduction velocity in shipyard workers occupationally exposed to hand–arm vibration

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Abstract
Objectives Segmental sensory nerve conduction velocity (SNCV) was measured from the wrists to the hands and digits in a population of 134 (126 men and 8 women) vibration-exposed shipyard workers following systemic warming using a bicycle ergometer. Results were compared to earlier nerve conduction tests, identical in execution, except that the warming process was segmental and cutaneous. The study was designed to investigate whether SNCVs, which were selectively slow in the fingers after segmental cutaneous (skin surface) warming, would be affected differently by systemic warming.
Methods Wrist–palm, palm–proximal digit, and digital sensory nerve segments were assessed antidromically by stimulating at the wrist with recording electrodes placed distally. The same subjects were cutaneously warmed in 2001 to ≥31°C and were systemically warmed 28 months later in 2004 by ramped sustained exercise to 100 W for 12 min. Skin temperatures were measured by traditional thermistry and by infrared thermal images taken over the hand and wrist surfaces.
Results When systemic warming was compared to segmental cutaneous warming, SNCVs were increased by 15.1% in the third digit and 20.4% in the fifth digit of the dominant hand. Respective increases in the non-dominant hand were 11.0% and 19.4%. A strong association between increased surface skin temperature and faster SNCV, which had been observed after segmental cutaneous warming, was largely eliminated for both digit and palmar anatomic segments after systemic warming. Significant differences in SNCV between vibration-exposed and non-exposed workers, which had been observed after segmental cutaneous warming, were eliminated after systemic warming. Systemic warming had only a small effect on the wrist–palm (transcarpal) segmental SNCVs.
Conclusions Reduced SNCV in the digits was observed in vibration-exposed and non-exposed workers. Substituting exercise-induced systemic warming for segmental cutaneous warming significantly increased SNCV in the digits and appeared to reduce differences in SNCV between vibration-exposed and non-exposed workers. These findings persisted despite a substantial time interval between tests, during which the subjects continued to work. There may be more general implications for diagnosing clinical conditions in industrial workers, such as the carpal tunnel syndrome and the hand–arm vibration syndrome.

Keywords Sensory nerve conduction velocity · Exercise-induced warming · Vibration exposure · Thermography

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Introduction

Abnormalities of sensory nerve conduction velocity (SNCV) that appear to be independent of clinical entrapment neuropathy have long been recognized in workers exposed to hand–arm vibration (HAV; Seppalainen 1972; Chatterjee et al. 1982). Several investigators have also described a distal pattern of delayed sensory nerve conduction velocity (SNCV), localized to the digits (Sakakibara et al. 1994, 1998; Hirata et al. 2002). The methodology and our results from a baseline study of segmental SNCV in vibration-exposed shipyard workers were the first phase of an international exposure–response study on HAV, and were previously reported in this journal (Cherniack et al. 2004a) and seemed to confirm the earlier reports. Significant digital SNCV reduction was observed. The well-known positive association between skin temperature and increased nerve conduction velocity (Tashjian et al. 1987; Denys 1991) was also replicated, but with a notable twist: each sensory nerve segment had a characteristic velocity/temperature pattern, with the slopes increasing in the more distal segments. The relationship was more pronounced for the fifth digit than the third, and there was little effect of temperature on SNCV in the proximal wrist segments. Since an acceptable surface temperature (≥31°C) had been maintained throughout testing (Halar et al. 1983), such a strong variation in the dependence of SNCV on skin temperature between adjacent hand segments was unexpected. Simple reapplication of the nerve conduction protocol would not differentiate between particulars of the test method and a physiologically based explanation that might arise out of the HAV disease process itself.

The members of the HAVIC consortium[1] concluded that retesting with the baseline warming technique required reconsideration, given the unexplained variability of the segmental velocity–temperature association. That, alone, compromised future repeated measures comparisons of SNCV at the individual level, which was a fundamental pre-condition for a longitudinal exposure–response study. The possibility that vascular dysfunction due to HAV was a potentially powerful covariate of segmental SNCV also merited further investigation. Scandinavian investigators, confronted with the problem of maintaining stable hand temperatures during cold weather months without prolonged acclimatization, were able to induce stable temperatures from systemic warming, using a ramped exercise protocol (Wallin 2002; Sanden et al. 2005). Stable and prolonged hand and finger warming had, in their studies, lasted throughout nerve conduction tests for more than 30 min. Accordingly, body segment heating was replaced by exercise-based whole body warming, following the methodology of Wallin (2002). Before field introduction, the systemic warming approach was pre-tested in a laboratory environment where it was compared to conventional surface warming (Croteau 2004). There was a serious trade-off. Protocol alteration meant the potential loss of meaningful longitudinal comparison in the largest single cohort, shipyard workers. There was, however, an opportunity to compare SNCV using two different warming techniques in a relatively large and diverse cohort, having exposure to vibration. Since 28 months had elapsed between the baseline and follow-up SNCV tests, the principal time-related factors, age and added exposure, need to be considered in a comparison of two testing protocols. These factors were, however, expected to act against detection of an exercise-induced warming effect. They would thus introduce a conservative bias into the study.

[1] The HAVIC (Hand–Arm Vibration International Consortium) is a multinational research group, organized to better define exposure–response relationships from segmental vibration by using longitudinal study design.

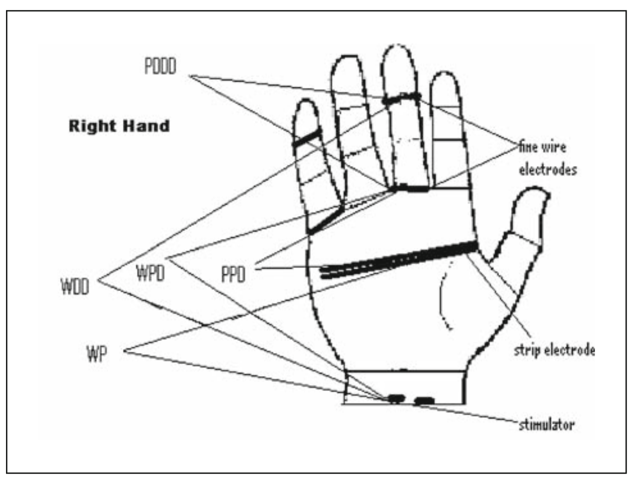
Methods, subjects, and design

Nerve conduction measurements

The measurements of nerve conduction reported here follow a test–retest design, where results from two different testing protocols are compared at the individual level. SNCVs were measured for the distal median and ulnar nerves in both the upper extremities. Motor nerve conduction velocity (MNCV) was assessed for the median nerve (abductor pollicis brevis) in both theupper extremities. All studies were performed on a Nicolet Viking Quest ® device. SNCV testing followed the approach of Sakakibara et al. (1994, 1998). The following median and ulnar nerve sensory segments were assessed:

1. proximal digit–distal digit (pddd),
2. palm–proximal digit (ppd),
3. wrist–palm (wp),
4. wrist–distal digit (wdd),
5. wrist–proximal digit (wpd).

The anatomic segments and the position of the recording electrodes are shown in Fig. 1.



PDDD	Proximal digit – distal digit	WP	Wrist-palm
PPD	Palm – proximal digit	WDD	Wrist-distal digit
WPD	Wrist-proximal digit		

Fig. 1 SNCV segments of the median nerve

Baseline protocol (2001)

All nerve conduction tests were preceded by at least 30 min of acclimatization at 20–22°C and by hand warming using electrical warming mittens and hydroculation pads. Studies did not proceed until the temperature at the base of the second digit was ≥31°C. Temperatures were recorded just prior to nerve stimulation. For SNCVs on the digit innervated by the median nerve (third digit) and the ulnar nerve (fifth digit), paired wire-looped ring electrodes were positioned at the proximal and distal sites. The proximal digital electrode was located at the metacarpo-phalangeal (MCP) crease, and the distal digital electrode bisected the distal interphalangeal (DIP) joint crease. For detection of the stimulus at the palm (wrist–palm segment), disposable gelled ring electrodes were cut and contoured along the width of the palm and extended on the distal margin of the mid-palmar crease to a boundary, defined medially by a line drawn from the intersection of digits two and three and laterally to the edge of the ulnar palm. Segment distances were measured for each subject based on common anatomical landmarks in order to accommodate variations in hand anthropometry (short palm, short finger) that would influence disparities between a standard distance and the actual anatomic segment. There was a common site of stimulation for the median and ulnar nerves at the first wrist crease. Motor nerve conduction velocity (MNCV) was determined in a conventional way (Kimura 1989). Because of our interest in assessing carpal tunnel syndrome (CTS), a transpalmar SNCV (from wrist to mid-palm) was also measured for both median and ulnar nerves. All tests were performed bilaterally.

All results are reported as velocities (in m/s), determined from the time of stimulation to the onset of the action potential. The choice of onset latency reflects an interest in the fastest conducting sensory fibers. The conversion to velocity provides a translatable value for segments that lack normal values for latency. A more detailed description of the technique can be found in Cherniack et al. (2004a).

Revised protocol (2004)

The revised protocol used an identical nerve conduction method, but took a different approach to warming and temperature measurement. Before nerve stimulation, sub-maximal exercise took place on a bicycle ergometer (12 min, ramped at 50–100 W; Wallin 2002), followed by testing within a temperature-controlled environment. Consistent with the Wallin protocol, ramping was linked to heart rate (continuously monitored) and was curtailed by extremity discomfort. Subjects underwent a physician review prior to exercise. There was a low threshold for exclusion, subjects being directed to cutaneous warming or to limited exercise warming (50 W) in the presence of musculoskeletal or cardiovascular symptoms, identified disease or heart rate slowing medication.

The room temperature was thermostatically controlled at 22°C with an acceptable error range of ±1°C. All testing was performed in the sitting position. Subjects were draped with a thermal blanket during nerve conduction testing to maintain core temperature. The skin temperature was measured by thermistry as in 2001, and also by infrared digital thermography, using a thermal camera and dedicated software (ISI Snapshot v. 2.1/Plymouth, MN, USA). The skin temperature was measured on the volar surfaces of both the hands. There were three images: (1) prior to exercise, (2) post-exercise, prior to stimulation, and (3) post- stimulation. Mean temperatures and their variance were translated from thermal images to linear averages for each of the five interelectrode segments. Temperature regions were defined from stimulator to electrode, consistent with anatomic landmarks. Thermistry was maintained to maximize the test similarity. The use of thermal imaging reflected our appreciation that skin temperatures might be specific to nerve segments, therefore invalidating velocity/temperature relationships defined by a single-point temperature measurement.

Subject related issues and repeated measures

In October–December of 2001, 217 members of a shipyard workforce (202 men and 15 women) participated in a baseline assessment that included SNCV testing and surface warming. In February–April of 2004, 134 participants (126 men and 8 women) were retested (0.63 retest rate) following a revised warming method. Reasons for failure to return were documented for 55 participants. These included: long-term travel or re-assignment to other shipyards ($n = 12$); withdrawal for specified reasons, such as long-term offsite assignment to a military base ($n = 12$); termination ($n = 11$); disability and medical leave ($n = 10$); problems of documentation within the shipyard ($n = 9$) and retirement ($n = 1$). The low retirement number reflects compliance with a study request that workers anticipating retirement within 2 years of study onset should not participate. A total of 28 participants who were evaluated in 2001 remained on employment rosters, but did not provide a reason for failure to follow-up. All participation was voluntary and increased production schedules forced significant rescheduling and work conflicts. There was no mechanism to recapture the non-retested group, but demographic and SNCV results from 2001 are available for this group and comparisons with ongoing participants are presented in the "Results" section.

Working populations tempered by shipyard employment will usually present with more clinically detectable abnormality than an external control group, whether determined by symptom prevalence or by more quantitative metrics (Jang et al. 2002; Bovenzi 1998; Letz et al. 1993). The comparative over-representation of disease may invite spurious assignment of causation due to confounding factors that contribute to morbidity. When the prevalent abnormality nears 50%, as occurred in this working group in 2001, external referent comparisons are likely to be to obscure particular to sub-groups within exposed cohorts (Cherniack et al. 2004a). Accordingly, in 2001 a small internal non-exposed comparison group ($n = 28$) was selected from the larger cohort. There were three inclusion criteria: (1) no current exposure to vibratory tools, (2) absence of CTS on physical examination, and (3) a Stockholm Workshop Scale of 0 or 1 for sensorineural symptoms (Brammer et al. 1987a).

Questionnaire and physical examination

Participants completed a self-administered questionnaire, directed to vibration-specific and more general musculoskeletal disorders in both 2001 and 2004. Exposure-specific questions were developed to profile each set of work tasks. The questionnaire has been previously described (Cherniack et al. 2004b). Symptom questions also included staging with the Stockholm Workshop Scale, a consensus instrument used for classifying sensorineural and vascular symptoms of HAVS (Gemne et al. 1987; Brammer et al. 1987a). Questionnaire components pertinent to this study include estimates of vibration exposure and work history, neurological and vascular symptoms, and demographics.

A standardized upper extremity clinical examination instrument was developed, incorporating proximal and distal evaluation. Each clinical test and detailed procedure was reviewed for consistency with other standardized examinations (Nilsson 2002; Sluiter et al. 2001; Viikari-Juntura 1996). Components used in the following analyses include finger circumference, hand dimensions, height and weight.

Statistical analysis

Statistical analyses utilized SAS 10.0 for Windows. Summary statistics on basic demographics, electrophysiological variables, hand and wrist distances and skin temperatures were calculated for a vibration exposed and control group as well as for the whole cohort. As a first order of comparison, an internal control group was assembled for shipyard workers. Difference between groups was measured using *t* tests.

Linear models were constructed for SNCV. Residual analyses was routinely carried out to check model assumptions. Wilk-Shapiro's test and quantile–normal plots were used to check normality, and Brown and Forsythe's test and plots of residuals versus fitted values were used for homogeneity of variance.

The relationship between the log-transformed nerve conduction velocity and age, gender, finger circumference, height, body mass index (BMI) and hand temperature was investigated by multiple linear regressions. Covariates were included in the model if the regression coefficients had a *P* value less than 0.05. The contribution of the partial R^2 was not an inclusion criterion.

Simple linear regression models were also built to study the change of nerve conduction velocity by unit change of hand temperature ($\Delta V/\Delta t$). Differences in conduction velocity between the positive and negative groups for sensory numbness or CTS were assessed using ANOVA. The relationship of explanatory covariates to SNCV was rarely linear. Log transformations of the SNCV were employed in multivariate regression models. Although fit was sometimes optimized with high order models, the slight improvement in fit was outweighed by incomparability of coefficients.

To assess the extent to which increased SNCV is a temperature-, rather than a technique-related effect, 2001 temperatures were normalized to 2004 and the 2001 change in SNCV with temperature ($\Delta V/\Delta t$) for surface warming was applied to segmental velocities. The adjustment is conservative, since it presumes that the 2004 temperatures could have been reached in 2001 by surface warming and that change in SNCV with temperature is unaffected by the warming method.

Results

Overall comparisons

In Table 1, results from subjects restudied in 2004 were compared with their 2001 baselines. The restudied shipyard workforce was 28 months older with an additional 1.15 years per person of vibration exposure among the 117 workers, who continued to use power tools. In spite of increased age and greater cumulative exposure, SNCVs were higher in 2004, after systemic warming, most dramatically in the digital segments (ppdd). In the wrist–palm (wp) segment, these changes were modest, under 3% for both the median and ulnar nerves. In the digital segments of the median nerve, there were SNCV increases of 15.1% in the dominant hand third digit and 11.0% in the non-dominant hand third digit; the respective increases in SNCV in the dominant hand and non-dominant hand fifth digits were 20.4% and 19.5%. The longer track SNCV results (wpd and wdd), which most closely replicate conventional distal nerve conduction distances were also increased, particularly for the ulnar nerve. Differences are driven, however, by two non-independent factors. They are, (1) the proportional contribution of the distal segment (pddd and ppd) to

Table 1 Follow-up of shipyard workforce, 2001–2004 (n = 134)

Variable	2001			2004		
	Mean	SD	Minimum–maximum	Mean	SD	Minimum–maximum
Age (years)***	48.27	6.46	22–60	50.62	6.33	24–62
Height (cm)**	176.73	8.50	158–196	174.59	8.62	152–194
Weight (kg)	93.53	21.90	51.75–164.25	93.83	22.16	53.55–162.00
Third finger circumference (mm)	72.21	5.42	60–86	72.61	5.59	58–85
Fifth finger circumference (mm)	62.31	4.94	50–76	61.98	5.90	47–76
Years worked at the shipyard**	21.87	6.77	0.5–39.6	23.95	6.78	2.5–42
Years exposed to vibration	18.29	8.84	0–33	19.44	9.16	0–33
Vibration h/week	9.20	9.56	0–40	9.25	11.45	0–64
Nerve conduction velocity (m/s)						
Median nerve dominant						
Proximal digit–distal digit***	43.05	9.35	23–79	49.55	7.50	27–71
Palm–proximal digit**	51.45	10.73	29–96	54.67	8.27	35–79
Wrist– palm	41.59	8.08	24–70	42.71	7.14	26–60
Wrist–distal digit**	44.73	7.44	29–85	46.73	5.68	29–59
Wrist–proximal digit	45.14	6.66	27–62	46.11	5.90	30–58
Ulnar nerve dominant						
Proximal digit–distal digit***	43.24	14.05	12–100	52.04	8.92	33–89
Palm–proximal digit	55.48	14.96	20–100	57.86	8.92	34–83
Wrist–palm	50.24	7.84	23–75	51.64	5.90	41–70
Wrist–distal digit***	49.45	6.36	29–63	52.75	4.31	44–65
Wrist–proximal digit***	51.24	6.04	34–63	53.26	4.69	43–68
Median nerve non-dominant						
Proximal digit–distal digit***	45.49	11.21	22–90	50.50	7.98	34–78
Palm–proximal digit	52.79	9.92	21–90	54.39	7.35	27–71
Wrist–palm	42.09	8.58	25–65	43.52	7.81	28–63
Wrist–distal digit**	45.43	6.65	29–63	47.22	5.92	32–64
Wrist–proximal digit	45.60	7.46	28–65	46.56	6.43	31–63
Ulnar nerve non-dominant						
Proximal digit–distal digit***	43.34	11.21	21–86	51.80	7.94	29–75
Palm–proximal digit**	53.52	14.41	13–97	56.63	9.01	17–87
Wrist–palm	49.37	8.56	27–79	50.61	7.93	19–70
Wrist–distal digit***	48.63	5.94	26–65	52.01	5.70	31–66
Wrist–proximal digit**	50.31	7.56	28–74	52.26	6.47	26–71
Motor latency						
Dominant**	4.09	0.83	2.9–8.3	3.83	0.93	2.3–9.9
Non-dominant*	4.02	0.81	2.6–9.0?	3.83	0.73	2.6–7.0

P value for mean difference = 0: *** < 0.01; ** < 0.05; * < 0.1

wrist–digit SNCV, and (2) a trend towards decreased variance with longer (averaged) distances. For point of reference, median nerve motor latencies are also included; they were reduced by 6.4% on the dominant hand and 4.7% on the non-dominant hand in 2004 from the values recorded in 2001.

Another characteristic of the 2004 SNCV studies, compared to 2001, is reduced variance in all segments, when nerve conduction studies were performed after exercise-induced warming. This is most evident in the more distal segments: palm–proximal digit, and proximal digit–distal digit, particularly for the ulnar nerve.

Internal comparisons

There were 15 subjects from the internal unexposed group (IUG) available for retesting in 2004, who met the study criteria in both testing intervals (see Table 2). Nine subjects were no longer available for restudy, and four did not meet selection requirements due to a change in exposure and/or

Table 2 Demographic and SNCV comparison of dropouts and continuing participants, 2001 comparisons

Variable	Internal unexposed group		Variable	Exposed group	
	Dropout[a] *n* = 9	Retested[b] *n* = 15		Dropout[a] *n* = 76	Retested[b] *n* = 119
Age (years)**	51.56 (5.83)	44.2 (8.7)	Age (years)*	46.87 (6.69)	48.36 (5.99)
Height (cm)*	171.67 (12.8)	180.6 (8.68)	Height (cm)	175.37 (6.72)	176.24 (8.39)
Weight (kg)*	82.4 (21.21)	95.25 (22.19)	Weight (kg)	92.28 (16.52)	93.31 (21.95)
Third finger circumference (mm)	70.22 (6.7)	70.13 (4.14)	Third finger circumference (mm)	72.32 (5.34)	72.47 (5.52)
Fifth finger circumference (mm)	59 (6.18)	61.53 (5.64)	Fifth finger circumference (mm)	62.89 (5.09)	62.4 (4.87)
Years worked at shipyard*	23.94 (7.05)	18.49 (8)	Years worked at shipyard*	23.29 (7.74)	22.27 (6.53)
Years exposed to vibration	9.22 (9.77)	7.93 (9.16)	Years exposed to vibration**	21.63 (9.36)	19.6 (7.92)
Vibration h/week	0 (0)	0 (0)	Vibration h/week	10.59 (11)	10.39 (9.53)
Nerve conduction velocity (m/s)					
Median nerve dominant					
Proximal digit–distal digit	46.33 (8.87)	45.47 (6.86)	Proximal–distal digit	41.53 (8.64)	42.7 (9.63)
Wrist–palm	42.75 (7.54)	45.07 (6.54)	Wrist–palm	42.06 (7.61)	41.12 (8.18)
Wrist–distal digit**	44.67 (6.93)	48.27 (4.04)	Wrist–distal digit	45.41 (6.64)	44.22 (7.69)
Wrist–proximal digit**	44.33 (7.11)	49.4 (4.4)	Wrist–proximal digit	45.41 (6.38)	44.57 (6.71)
Median nerve non-dominant					
Proximal–distal digit	44.22 (7.5)	48.6 (6.92)	Proximal–distal digit	43.76 (9.83)	45.07 (11.62)
Wrist–palm	43.11 (5.35)	46.47 (8.31)	Wrist–palm	43.19 (7.91)	41.52 (8.49)
Wrist–distal digit	47.56 (5.08)	49.2 (5.83)	Wrist–distal digit	45.72 (6.15)	44.92 (6.62)
Wrist–proximal digit	48.78 (4.97)	49.6 (6.93)	Wrist–proximal digit	46.36 (6.83)	45.08 (7.39)

P value for mean difference = 0: ** < 0.05; * < 0.10

[a] Dropout is a subject tested in 2001, but not retested in 2004

[b] Retested is a subject tested in 2001 and in 2004

symptom status. There were two participants who met IUG criteria in 2004, but were ineligible in 2001.

In 2001, the IUG and the larger exposed group (EG) were demographically similar in age and years of employment (Cherniack et al. 2004a). However, as Table 2 demonstrates, the nine subjects from the IUG who were tested in 2001, but had dropped out in 2004 (column 2), were significantly older by 7.36 years than those who were tested in 2001 and retested in 2004 (column 3). They also had accrued an additional 5½ years of employment in 2001, compared to their 15 retested peers. Differences between retested and non-retested subjects were less pronounced for the 119 exposed participants who were retested in 2004 and the 76 who were not retested. Nevertheless, in 2001, non-retested members of the EG were both younger than those who continued, and had more years of exposure to vibration (21.63 ± 9.36) versus (19.60 ± 7.92). In addition to subject information, Table 2 also includes a comparison of selected 2001 SNCV results. In 2001, there was a significant difference between the IUG and EG participants when specific segmental velocities were compared. However, results in 2001 from the nine IUG subjects who were not retested in 2004 (column 2) showed markedly slower SNCVs in all but the ppd segment of the median nerve than for those who returned for retesting. These differences approached statistical significance despite the small sample. In the EG, the differences between participants retested in 2004 and those dropping out were less marked. No within-group comparisons approached significance, but there was a slight trend towards slower SNCV in the retested 2004 participants, except for the pddd segment. The effect on between-group comparisons, from limiting the sample to retested subjects only, was a slight enhancement of mean differences in SNCV between the IUG and EG in 2001.

Within-group and between-groups differences for the retested IUG and EG participants, based on the 2001 and 2004 comparisons, are presented in Table 3. To simplify, results are presented only for the dominant hand. Differences between groups for the non-dominant hand were smaller and are summarized in the text. To clarify the table, significant differences *between* groups are noted in the year column, and significant differences *within* groups between test years are indicated in the rows labeled T_2–T_1. For the IUG, in 2004 the pddd segment of the median nerve had a borderline significant increase, but other segmental differences were not statistically significant. Although there was actually a greater

Table 3 Comparison of exposed and unexposed shipyard workers

Nerve	Year	Internal unexposed group (IUG), $n = 15$		Exposed group (EG), $n = 119$	
		Mean (SD)	Range	Mean (SD)	Range
Median nerve dominant					
Proximal digit–distal digit	2001	45.47 (6.86)	32–54	42.70 (9.63)	23–79
	2004	49.62 (5.90)	38–59	49.54 (7.73)	27–71
	T_2–T_1	4.15 (6.44)		6.83 (8.8)***	
Palm–proximal digit	2001**	55.47 (7.33)	45–68	50.91 (11.02)	29–96
	2004	53.15 (7.43)	38–69	54.89 (8.39)	35–79
	T_2–T_1	−2.31 (7.37)		3.98 (9.93)***	
Wrist–palm	2001**	45.07 (6.54)	31–54	41.12 (8.18)	24–70
	2004*	46.23 (6.18)	37–55	42.22 (7.15)	26–60
	T_2–T_1	1.16 (6.38)		1.10 (7.73)	
Wrist–distal digit	2001***	48.27 (4.04)	41–54	44.22 (7.69)	29–85
	2004	48.77 (5.34)	41–56	46.44 (5.7)	29–59
	T_2–T_1	0.50 (4.69)		2.22 (6.84)**	
Wrist–proximal digit	2001***	49.40 (4.4)	41–56	44.57 (6.71)	27–62
	2004	48.38 (5.44)	41–57	45.78 (5.92)	30–58
	T_2–T_1	−1.02 (4.91)		1.21 (6.37)	
Ulnar nerve dominant					
Proximal digit–distal digit	2001*	50.00 (17.76)	31–100	42.33 (13.32)	12–82
	2004	55.07 (10.82)	45–88	51.6 (8.59)	33–89
	T_2–T_1	5.07 (14.7)		9.27 (11.29) **	
Palm–proximal digit	2001	53.92 (15.04)	23–74	55.68 (15.02)	20–100
	2004	57.36 (5.23)	50–67	57.94 (9.35)	34–83
	T_2–T_1	3.43 (11.08)		2.25 (12.52)	
Wrist–palm	2001	51.86 (7.32)	38–64	50.02 (7.92)	23–75
	2004	53.50 (5.02)	43–64	51.37 (5.99)	41–70
	T_2–T_1	1.64 (6.27)		1.35 (7.05)	
Wrist–distal digit	2001**	52.64 (5.3)	42–58	49.03 (6.39)	29–63
	2004 *	54.43 (4.03)	46–63	52.51 (4.32)	44–65
	T_2–T_1	1.79 (4.71)		3.48 (5.5)***	
Wrist–proximal digit	2001	53 (4.74)	45–61	51.01 (6.17)	34–63
	2004 *	54.57 (3.82)	46–63	53.07 (4.79)	43–68
	T_2–T_1	1.57 (4.3)		2.06 (5.56)***	

Difference (1–2) is for the interval 2004–2001. Significant intergroup differences for either test are noted by the year. T_2–T_1 within-group change from 2001–2004 in the SNCV

P value for mean difference = 0: *** < 0.01; ** < 0.05; * < 0.1

difference between means for ulnar SNCV across the fifth digit, greater variance made the comparison less statistically significant, although not necessarily less meaningful. For the transcarpal or wp segment, there were modest inter-test differences in the IUG. For the EG, SNCV increases for the pddd segment of the median nerve [↑6.83 m/s ± (8.78)] and the ulnar nerve [↑9.27 m/s ± (11.76)] were even greater than in the IUG internal controls. The faster velocities in long track measurements are driven by these digital segments as transcarpal SNCV increases (wp) were small. In 2004, between-group comparisons demonstrate an overall narrowing of the differences observed in 2001. SNCV for the ppd segment of the median nerve discriminated between IUG and EG participants in 2001, but results were essentially identical in 2004. This reduction of difference was ascribable to relatively greater increases in SNCV in the EG. For both IUG and EG, transcarpal segmental velocities were only modestly higher in 2004 and the degree of increase was also comparable between groups.

For SNCV in the non-dominant hand, there was very little change between 2001 and 2004 except in the median nerve pddd segment where there was an average increase of 2.59 m/s (±6.45) in the IUG and 5.32 m/s (±10.75) in the EG. Between 2001 and 2004, there was a slight decrease in the wrist–palm segment of the median nerve in the IUG and a corresponding slight increase in the same segment for the EG. As a result, the small difference between the IUG and EG in the wp segment in 2001 had disappeared in 2004.

Association of skin temperature with SNCV

In 2001, the relationship between skin temperature and SNCV, represented by the non-transformed slope of $\Delta V/\Delta t$, was distinctive. Key features, along with the 2004 retest results are presented in Table 4. Dominant and non-dominant hands are combined, but median and ulnar nerves are treated separately. The reason that there are fewer hands ($n = 220$) than would come from a doubling of subjects reflects a conservative decision to only count hands when all five segmental measurements were accepted, and to normalize the difference between dominant and non-dominant hands. There are three comparison groups. In 2001, hand temperature was recorded in a conventional manner at the base of the second digit. In 2004, hand temperatures were represented by the same single-point measurement, but were also averaged over each discrete segment using thermal imaging. In columns marked 2001[2] and 2004[3], the velocity/temperature relationship is determined using the single-point measurement. In the column marked 2004[4], the relationship is specific to the thermally imaged segment. The $\Delta V/\Delta t$ slopes for the pddd segment in 2001–2.00 (SD ± 0.43) for the median nerve and 2.09 (0.53) for the ulnar nerve, and for the ppd segment 1.17 (SD ± 0.43) for the median and 1.61 (SD ± 0.64) for the ulnar nerve were no longer significantly different from zero in 2004 after systemic warming. Of the three shorter segments (pddd, ppd, wp), in 2004, both the ulnar wrist–palm and proximal digit–distal digit segment had a slope that differed significantly from zero with both local and systemic warming.

When temperatures were based on the segment-specific temperatures determined by thermal image, there was a tendency towards somewhat greater slopes compared to the point temperature measurement in some segments. Systemic warming eliminated the positive SNCV–temperature relationship in the pppd segment that had been observed with body segment warming in 2001

In 2001, we had regressed SNCV by segment on age, gender, exposure, test digit circumference, height, BMI (body mass index) and skin temperature. Only age and skin temperature were significantly predictive. In Table 5, the inter-test difference in SNCV between 2001 and 2004 (ΔSNCV) was regressed on key explanatory variables. The analysis also provides an estimate of key confounders, such as change in age and body mass, which might be influential due to the inter-test time interval. Only the absolute inter-test difference in skin surface temperature predicted a positive ΔSNCV. Aging by 28 months did not significantly influence ΔSNCV. It is also notable that although many of the models are significant, more than 20% of the variance is attributable to the explanatory variables in only two seg-

Table 4 Segmental nerve conduction velocity and temperature relationship[a] compared to slope $\Delta V/\Delta t = 0$

Nerve segment	2001-point temperature[b]		2004-point temperature[c]			2004-segmental temperature[d]		
	$\Delta V/\Delta t \pm$ SD	*P* value[e]	$\Delta V/\Delta t \pm$ SD	*P* value[e]	*P* value[f]	$\Delta V/\Delta t \pm$ SD	*P* value[e]	*P* value[f]
Median nerve								
Proximal digit–distal digit	2.00 (0.43)	<0.001	−0.01(0.44)	0.976	0.006	0.58(0.42)	0.173	0.019
Palm–proximal digit	1.17 (0.43)	0.008	−0.01(0.45)	0.975	0.091	−0.01(0.45)	0.979	0.059
Wrist–palm	−0.38 (0.35)	0.287	0.53(0.43)	0.215	0.098	0.71(0.45)	0.120	0.056
Wrist–distal digit	0.33 (0.31)	0.291	0.29(0.33)	0.380	0.933	0.29(0.33)	0.285	0.936
Wrist–proximal digit	0.36 (0.30)	0.228	0.32(0.35)	0.360	0.985	0.54(0.41)	0.188	0.717
Ulnar nerve								
Proximal digit–distal digit	2.09 (0.53)	<0.001	0.92(0.42)	0.0298	0.054	0.92(0.37)	0.014	0.071
Palm–proximal digit	1.61 (0.64)	0.013	0.40(0.45)	0.3749	0.214	0.40(0.45)	0.375	0.122
Wrist–palm	0.52 (0.36)	0.147	0.97(0.35)	0.0060	0.729	1.18(0.38)	0.002	0.203
Wrist–distal digit	1.21 (0.25)	<0.001	0.77(0.25)	0.0020	0.107	0.93(0.26)	<0.001	0.006
Wrist–proximal digit	0.70 (0.29)	0.017	0.76(0.28)	0.0068	0.852	0.84(0.29)	0.004	0.725

There were no significant differences between the temperature-velocity relationship for point and segmental determinations of temperature

[a] Combined left and right hands $n = 220$

[b] Point temperature at digit base surface temperature (°C). Median nerve: mean (SD) = 31.59 (1.49), range = 27.30–35.20; ulnar nerve: mean (SD) = 31.55 (1.47), range = 27.30–35.20

[c] Point temperature at digit base surface temperature (°C). Median nerve: mean (SD) = 33.50 (1.18), range = 28.48–39.70; ulnar nerve: mean (SD) = 33.38 (1.32), range = 28.67–36.73

[d] Segmentally determined temperature: median and ulnar nerve temperatures are averaged over each independent segment

[e] *P* value for difference between sample and slope = 0

[f] *P* value for difference between slope for 2004 compared with 2001

Table 5 Regression of SNCV difference (2004–2001)[a] for all subjects ($n = 134$)

Segment	Age	Finger circumference	BMI	CTS	Temperature difference	RSQ	Pr>F
Median nerve dominant							
Proximal digit–distal digit						0.143	0.119
Palm–proximal digit						0.041	0.887
Wrist–palm					0.509	0.148	0.093
Wrist–distal digit					0.445	0.259	0.003
Wrist–proximal digit						0.137	0.120
Ulnar nerve dominant							
Proximal digit–distal digit				8.333		0.126	0.226
Palm–proximal digit						0.088	0.514
Wrist–palm			−0.241		0.923	0.251	0.002
Wrist–distal digit		0.284		3.404		0.165	0.037
Wrist–proximal digit		−0.480		.	2.965	0.209	0.009
Median nerve non-dominant							
Proximal digit–distal digit						0.084	0.456
Palm–proximal digit					1.223	0.233	0.003
Wrist–palm						0.1523	0.064
Wrist–distal digit					2.067	0.111	0.245
Wrist–proximal digit					2.256	0.107	0.295
Ulnar nerve non-dominant							
Proximal digit–distal digit					0.887	0.093	0.365
Palm–proximal digit					0.961	0.139	0.089
Wrist–palm						0.122	0.127
Wrist–distal digit	−0.013				−0.077	0.277	<0.001
Wrist–proximal digit						0.143	0.119

[a] Coefficients significant at 5%

ments, the ulnar nerve wdd in the dominant hand and the median nerve wdd in the non-dominant hand.

In addition, even the effect of higher skin temperature must be regarded with caution. Table 4 documents that in 2004, SNCV tests were performed on skin that was on average 1.9°C warmer for the median nerve and 1.8°C warmer for the ulnar nerve than in 2001. Means, standard deviations and ranges of skin temperature are indicated in footnotes #2 and #3 of Table 4. As observed in Table 6, this raw difference in point skin surface temperature is insufficient to explain the inter-test difference.

In Table 6, temperatures are normalized to 2004, and 2001 $\Delta V/\Delta t$ values have been applied to segmental velocities. In column 2 (SNCV differences), segmental SNCVs are first compared directly (2004–2001), then in a corrected form [2004–2001(Temp-adj], where the 2001 temperature is normalized to its 2004 equivalent. This is a very conservative correction since it presumes that surface warming and systemic warming affect skin temperature through an equivalent mechanism of heat transfer. Differences between 2001 and 2004 SNCVs (ΔSNCV) are reduced, particularly in the wpd segment. However, digital SNCVs in both median and ulnar distributions for both dominant and non-dominant hands remain elevated in 2004 compared to 2001. Even under conditions of the most conservative correction, systemic warming appears to increase SNCV, apart from the absolute skin temperature measured on the hand.

Discussion

The hand–arm vibration syndrome consists of neurological and vascular components. It has long been recognized that sensorineural deficits and vascular dysfunction, characterized as occupational Raynaud's phenomenon, can occur independently (Virokanas 1992), and there is a compelling logic, as articulated in international consensus, to separate the two major presentations for purposes of epidemiologic study (Gemne et al. 1987). It has also been recognized that electrophysiological studies performed on symptomatic workers with HAV exposure differ in substantial ways from non-vibration-exposed workers with more conventional nerve entrapment (Stromberg et al. 1997). Sakakibara et al. (1998) and our own group (2004a) appeared to demonstrate that a distinctive pattern of SNCV slowing in vibration-exposed workers could be ascribed to reduced nerve conduction velocity in the digital segments. Our analyses of surface-warmed hands in 2001 at first appeared to confirm

Table 6 Adjusted 2001 SNCV to equal skin temperatures for all subjects (n = 134)

Nerve segment	SNCV difference	Mean (SD)	P value	Nerve segment	SNCV difference	Mean (SD)	P value
Median nerve dominant				Median nerve non-dominant			
Proximal digit–distal digit	2004–2001	6.66 (10.6)	<0.001	Proximal digit–distal digit	2004–2001	5.96 (12.73)	<0.001
	2004–2001(Temp.-adj.)	2.66 (10.57)	0.017		2004–2001(Temp.-adj.)	2.45 (12.11)	0.044
Palm–proximal digit	2004–2001	3.5 (13.14)	0.008	Palm–proximal digit	2004–2001	0.99 (11.13)	0.357
	2004–2001(Temp.-adj.)	1.3 (13.04)	0.323		2004–2001(Temp.-adj.)	−1.23 (11.04)	0.263
Wrist–palm	2004–2001	0.87 (5.56)	0.114	Wrist–palm	2004–2001	1.28 (6.24)	0.036
	2004–2001(Temp.-adj.)	NA			2004–2001(Temp.-adj.)	NA	
Wrist–distal digit	2004–2001	1.6 (5.82)	0.008	Wrist–distal digit	2004–2001	1.79 (4.55)	<0.001
	2004–2001(Temp.-adj.)	NA			2004–2001(Temp.-adj.)	NA	.
Wrist–proximal digit	2004–2001	0.6 (4.44)	0.172	Wrist–proximal digit	2004–2001	0.54 (4.3)	0.197
	2004–2001(Temp.-adj.)	NA			2004–2001(Temp.-adj.)	NA	
Ulnar nerve dominant				Ulnar nerve–non-dominant			
Proximal digit–distal digit	2004–2001	7.69 (17.32)	<0.001	Proximal digit–distal digit	2004–2001	8.33 (12.74)	<0.001
	2004–2001(Temp.-adj.)	3.46 (15.91)	0.034		2004–2001(Temp.-adj.)	4.95 (12.35)	<0.001
Palm–proximal digit	2004–2001	4.38 (14.83)	0.005	Palm–proximal digit	2004–2001	3.31 (15.29)	0.030
	2004–2001(Temp.-adj.)	0.66 (14.6)	0.667		2004–2001(Temp.-adj.)	0.37 (15.02)	0.809
Wrist–palm	2004–2001	1.54 (7.8)	0.056	Wrist–palm	2004–2001	1.08 (9.44)	0.2393
	2004–2001(Temp.-adj.)	NA			2004–2001(Temp.-adj.)	NA	
Wrist–distal digit	2004–2001	3.38 (6.78)	<0.001	Wrist–distal digit	2004–2001	3.53 (6.07)	<0.001
	2004–2001(Temp.-adj.)	0.73 (6.34)	0.256		2004–2001(Temp.-adj.)	1.24 (5.96)	0.038
Wrist–proximal digit	2004–2001	2.46 (5.79)	<0.001	Wrist–proximal digit	2004–2001	1.67 (7.82)	0.027
	2004–2001(Temp.-adj.)	0.85 (5.78)	0.140		2004–2001(Temp.-adj.)	0.23 (7.74)	0.762

Differences between 2004–2001 SNCV adjusted in 2001 by $\Delta V/\Delta t$ coefficients

NA indicates a non-significant velocity/temperature coefficient the conclusions of Sakakibara et al. (1994, 1998) that digital nerve segments in a vibration-exposed population were slower than proximal palm–digit base segments. Unlike their Japanese counterparts, the North American shipyard workers also had slowing in the wrist–palm segment of the median nerve. The 2001 tests also duplicated Japanese observations that paresthesias (Stockholm Scale SN I-III) coupled to current exposure were associated with reduced median nerve SNCV in the wrist and digit. The 2004 retest results placed a set of restrictive conditions on that observation. Digital segments of the median nerve resembled the more proximal segments. For the ulnar nerve, inter-segment differences between wrist–palm, palm–proximal digit, and digit segments were reduced. The results also suggested that altered digital perfusion, indirectly measured as skin temperature, may explain the unusual pattern of SNCV slowing in vibration-exposed workers.

Had we set out to measure differential effects of warming methods on SNCV, a longitudinal study with substantial inter-test intervals would not have been the preferred study design. The interval between baseline testing and retesting, dictated by the timelines from a larger study, complicate the basic premise that there is a difference between the two warming methods on nerve conduction velocity. However, unexpected results are an important aspect of cohort studies. Putting the reservations over design aside for a moment, there are notable implications for better understanding the hand–arm vibration syndrome.

1. Systemic warming seems to generate significantly faster SNCVs in the distal median and ulnar nerves than cutaneous warming.
2. The relationship of faster velocity to warming method appears to persist after conservative normalization.
3. Faster SNCV occurs mostly in the digit and across the palm, whereas the transcarpal segment is less affected.
4. The well recognized relationship between skin surface temperature and SNCV was observed in distal segments with surface warming, but it was largely eliminated after systemic warming, particularly for the median nerve distal segments.
5. While small numbers compromise inferences drawn from comparison of unexposed and exposed groups, it does appear that significant between-group differences seen after cutaneous warming disappeared after systemic warming.

The surprising results from the cross-sectional study in 2001 generated several explanatory hypotheses: (1) vibratory exposure or heavy industrial work might cause a fixed distal SN demyelinating injury or adaptation; (2) single-point thermistor measurements at the digit base might misrepresent actual digital temperature, leading to an erroneous velocity/temperature association; (3) blood flow variation (vascular flux) of the hand in vibration-exposed workers might differentially influence near nerve (NN) temperatures and depolarization; or (4) more general physiological characteristics, such as myelin tapering, might contribute to reduced distal velocities. In the last hypothesis, SNCV distal slowing would have been a normal observation that resulted from a sensitive technique.

The comparability of SNCVs recorded in the exposed and unexposed populations with systemic warming refutes the premise that SNCV deficits are fixed injuries or otherwise based on a static phenomenon. Differences in temperature measurement technique are also an unlikely explanation of the findings. With the exception of pddd segments for the median nerve, Table 4 shows that substituting the averaged segment temperature (infrared measurement of the entire hand surface) for a point measurement did not significantly alter the velocity/temperature slope. However, this can only be construed for 2004, since there were no thermal images in 2001, and systemic warming was only done in 2004. It remains a possibility that, had we measured cutaneous temperature in 2001 with an infrared camera, we would have uncovered cooler digital temperatures, thus explaining slower velocities on the basis of reduced skin temperature. However, as Table 6 demonstrates, even when 2004 distal temperatures, derived from thermal images, are applied to the velocity/temperature coefficients of 2001, SNCVs are still elevated in 2004. Moreover, the presumed interaction is probably unrealistically large, since the effect of temperature on velocity tends to flatten with higher temperatures. It also seems likely that the increase in digital SNCVs with systemic warming discounts a purely anatomic explanation for comparative SNCV slowing in the digits, such as physical tapering of myelin in digital nerves. With perfusion from systemic warming, “slow” nerve segmental velocity appeared to be dynamic and responsive to temperature change.

While reasons for the association between vibratory exposure and reduced digital nerve conduction in 2001 have yet to be explained, there are inferences. It appears that systemic warming may eliminate the differential in digital SNCV between exposed and non-exposed subjects. This would seem to suggest a baseline difference, which is not affected by surface warming. Hence, the differences between exposed subjects and internal controls for SNCV with surface warming that were eliminated with systemic warming may well be the result of an unspecified neurovascular mechanism that is insufficiently captured by our measurements.

Losses to follow-up and inter-test interval

We were concerned with the repeatability of a test rather than the outcome, and the loss of a substantial part of the study population to follow-up injects the potential for a significant selection bias. While there may be factors that associate failure to be re-tested with the dependence of SNCV on method of warming, such a cohort effect, however unlikely, would only emphasize the more general point that conclusions based on this particular group of 134 workers should be extrapolated with caution. Table 2 suggests a general uniformity in 2001 for demographics and SNCVs between the retested and non-retested groups. Another type of independent confirmation may be derived from cold challenge plethysmography. In 2001, 25% of the full test group was abnormal and 22% were abnormal in 2004, again suggesting comparability of the follow-up and non-retested populations.

The elapse of 28 months between tests presents an apparent problem, even if the effects would likely be conservative. There are reasons to suspect, however, that an effect on SNCV due to aging <2.5 years would be minimally significant. Rivner et al. (2001) reviewed data on nerve conduction and aging and found little age effect in the upper extremity on velocity between the ages of 20 and 55, an age distribution that overlaps this population. The follow-up studies by Nathan et al. (1988, 1998) on an active workforce demonstrate a small decrease in SNCV over 11 years, but suggest minimal change within an interval equivalent to this study. Tong et al. (2004) restudied an employed population with an inter-test period that averaged 5.4 years. The distal median nerve measurement declined by 1.1 m/s and the distal ulnar measurement by 0.7 m/s. These changes would translate into <0.5 m/s for the shipyard population, given the shorter inter-test interval. Furthermore, in 2001 the effect of age on SNCV, while significant, was quite small (correlation coefficient <−0.01 in all cases), and never positive for the proximal digit–distal digit and palm–proximal digit segments. From 2001–2004, as Table 5 demonstrates, there was no age effect on SNCV. While contributions from new onset morbidity or exposure may have introduced an unrecognized temporal effect at follow-up testing, the available evidence supports the conclusion that nerve conduction tests in 2004 can be viewed both as a retest, as well as a follow-up sample. In any event, any effect of aging, including exposure, would tend to reduce SNCVs in 2004 and so introduce a conservative bias.

Technical issues

In our earlier paper, we addressed pertinent issues around segmental nerve conduction studies (Cherniack et al. 2004a). Perhaps the most problematic is obtaining reliable SNCV measurements over short segments. It has been argued that a minimal distance of 4 cm per segment is required to avoid physiologic phase cancellation due to insufficient inter-electrode distance (Kimura 1998; Dumitru and Walsh 1988). The problem of limited space between stimulator and electrode was not a concern in this study because the stimulator was held at a constant location with the electrodes applied to fixed distal points. Moreover, more recent reports on short segment stimulation, particularly for the ulnar nerve, have confirmed the reliability of these measurements (Azrielli et al. 2003; Visser et al. 2005). In our earlier study, we investigated potential measurement error with short segments by internal comparisons of means and of variance within segments, and found no differences. Thus, there is no observational reason for discounting these measurements of SNCV in the digits.

Significance of the study

CTS has commonly been reported in vibration-exposed workers (Farkkila et al. 1988; Hagberg et al. 1991; Wieslander et al. 1989) and comparatively slowed nerve conduction has been described in industrial cohorts, compared with non-industrial workers (Stetson et al. 1993). Bingham et al. (1996) localized slowing of the median nerve, noting that 17.5% of industrial job applicants had distal slowing at the time of hire. There is, moreover, evidence that industrial work and particularly vibration exposure may affect SNCV in the digital segments (Brammer and Pyykkö 1987b). Cutaneous nerve biopsies from industrial workers, chronically exposed to vibration, have shown digital nerve demyelisation and fibre loss (Takeuchi et al. 1988). The current study both clarifies and complicates these observations. With both methods of warming, the wrist–palm segment remained slower for the median nerve than the two more distal segments in both the dominant and non-dominant hands. However, systemic warming tended to increase the slope of the temperature–velocity relationship ($\Delta V/\Delta t$) in the median nerve wrist–palm segment, as demonstrated in Table 4, although the effect was of borderline statistical significance. Even with conservative adjustment for skin temperature differences (Table 6), the increase in SNCV in the median nerve wrist–palm segment approached statistical significance for the dominant hand ($P < 0.11$) and was significant for the non-dominant hand ($P < 0.03$). In summary, the method of warming had less effect across the wrist than in the finger, but there was an observable effect.

These increases in more distal hand and finger segments may seem limited in their clinical significance given the stability in the longest single segment (wrist–palm), particularly since the transcarpal or transpalmar nerve conduction segment figures prominently in the electrophysiological definition of median nerve mononeuropathy. However, default reliance on a standardized cut-off for an electrophysiological CTS diagnosis would produce a dramatic change in the number of positive tests in this shipyard population, because nerve conduction velocities are generally low and would be centrally distributed ± 1 SD. around an algorithmic cut-off value (Cherniack et al. 2004). In this earlier paper, we had used SNCV reference values for median nerve abnormality from Salerno et al. (1998) that were based on studies from 324 industrial workers. There are differences in the two methods, however. They used a standard 14 cm wrist–digit interval for the median nerve and presented their data for dominant and non-dominant hands. We relied on individually measured distances between anatomic landmarks (the volar wrist crease and the distal phalangeal joint). Accordingly, inter-segment distances were longer: 17.3 cm (SD ± 8.1) for the dominant hand and 17.4 cm (SD ± 9.5) for the non-dominant hand. For purposes of comparison, we converted their published latencies to velocities based on constant distance. Consistent with the authors' recommendation, a 95th percentile lower limit non-parametric cut-off was also used. In 2001, using a SNCV lower limit of 43.7 m/s in the dominant hand and 45.2 m/s in the non-dominant hand, 41.7% of dominant hands and 49.5% of non-dominant hands were abnormal across the wdd segment among the 134 subjects who were restudied. Using the same cut-off on the same subjects in 2004, 32.7% of the dominant hands and 36.2% of the non-dominant hands were abnormal. Thus, electrophysiological results consistent with CTS were decreased by 22% on the dominant hand and by 27% on the non-dominant hand with a change in the warming technique. On a group basis using the 2004 protocol, the median SNCV was ∼5% faster than in 2001. This difference would have produced only a small effect on diagnosis in a population that resembled the Salerno et al. (1998) reference cohort; in our "abnormal" cohort, there was a large distributive effect on the diagnostic threshold.

The suggestion that in a vibration-exposed cohort, the method of warming may eliminate slowed SNCV in the digits raises another consideration. The differences between methods of warming may be as much a measure of pathology as a measure of choice of technique. If, for example, vibratory exposure reduces SNCV in the fingers because of a neurovascular injury, then elimination of the effect by an aggressive warming method implies something different than simple imposition of normalization conditions.

While the comparisons between 2001 and 2004 may suggest a strong influence of the choice of warming technique, there are other possible explanations. For one, since hand–arm vibration is associated with neurovascular injury and Raynaud's phenomenon, it might be posited that an increase in SNCV is influenced by underlying vascular pathology and an exercise-related perfusion effect. We observed more subjects with lower skin temperature in 2001 compared to 2004, thus introducing the confounding consideration, that ↑SNCV is at least in part a reflection of increased skin surface temperature, independent of the selection of the warming technique. Finally, there is the less plausible, but not irrelevant, possibility that decreased vibratory exposures over the 28 month inter-test interval may have resulted in a true improvement in sensory nerve function.

It is also unknown if the same type of effect from exercise-induced warming could be achieved by other means, such as longer acclimatization in a "hot room", or a non-surface warming method, such as infrared arm warming. On the other hand, the problem of over-diagnosis of CTS based on delayed SNCV seems to be somewhat obviated by looking only at the transcarpal segment where the median nerve is most embedded and less subject to surface temperature flux and changes in blood flow. Even here, there are differences, which might best be addressed with the well-known caveat that nerve conduction studies are not a gold standard for diagnosing nerve entrapment.

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References

Azrieli Y, Weimer L, Lovelace R, Gooch C (2003) The utility of segmental nerve conduction studies in ulnar mononeuropathy at the elbow. Muscle Nerve 27:46–50

Bingham RC, Rosecrance JC, Cook TM (1996) Prevalence of abnormal median nerve conduction in applicants for industrial jobs. Am J Ind Med 30(3):355–361

Brammer AJ, Taylor W, Lundborg G (1987a) Sensorineural stages of the hand–arm vibration syndrome. Scand J Work Environ Health 13:279–283

Brammer AJ, Pyykko I (1987b) Vibration-induced neuropathy: detection by nerve conduction measurements. Scand J Work Environ Health 13:317–322

Bovenzi M (1998) Vibration-induced white finger and cold response of digital arterial vessels in occupational groups with various patterns of exposure to hand-transmitted vibration. Scand J Work Environ Health 24(2):138–144

Chatterjee DS, Barrwick DD, Pertrie A (1982) Exploratory electromyography in the study of vibration-induced white fingers in rock driller. Brit J Indus Med 39:89–97

Cherniack M, Brammer AJ, Lundstrom R et al (2004a) Segmental nerve conduction velocity in vibration exposed shipyard workers. Int Arch Occup Environ Health 77(3):159–176

Cherniack M, Morse TF, Brammer AJ et al (2004b) Vibration exposure, workplace modification, and disease in a shipyard: a 13-year revisit. Am J Indus Med 45:500–512

Croteau M (2004) Establishment of nerve conduction techniques in hand arm vibration syndrome. Master's thesis, UCONN Health Center

Denys EH (1991) AAEM Minimonograph #14: the influence of temperature in clinical neurophysiology. Muscle Nerve 14:795–811

Dumitru D, Walsh NE (1988) Practical instrumentation and common sources of error. Am J Phys Med Rehabil 67:55–65

Farkilla M, Pyykko I, Jantti V et al (1988) Forestry workers exposed to vibration: a neurological study. Brit J Indus Med 45:188–192

Gemne G (1987) (ed) Stockholm Workshop 1986. Symptomatology and diagnostic methods in the hand–arm vibration syndrome. Scand J Work Environ Health 13S:265–388

Hagberg M, NystrÖm A, Zetterlund B (1991) Recovery from symptoms after carpal tunnel syndrome surgery in males in relation to vibration exposure. J Hand Surg 16:66–71

Halar EM, DeLisa JA, Soine TL (1983) Nerve conduction studies in upper extremities: skin temperature corrections. Arch Phys Med Rehabil 64:412–416

Hirata M, Sakakibara H, Abe M (2002) Reduced sensory nerve conduction velocity of the distal part of the radial nerve among patients with vibration syndrome. Electromyogr Clin Neurophysiol 42(2):113–118

Jang JY, Kim S, Park SK et al (2002) Quantitative exposure assessment for shipyard workers exposed to hand-transmitted vibration from a variety of vibration tools. AIHA J 63(3):305–310

Kimura J (1989) Electrodiagnosis. In diseases of nerve and muscle: principles and practices. FA Davis, Philadelphia

Kimura J (1998) Kugelberg lecture: principles and pitfalls of nerve conduction studies. Electroencephalogr Clin Neurophysiol 106:470–476

Letz R, Cherniack M, Gerr F et al (1993) A cross-sectional epidemiologic survey of shipyard workers exposed to hand–arm vibration. Br J Ind Med 49:53–62

Nathan PA, Meadows KD, Doyle LS (1988) Relationship of age and sex to sensory conduction of the median nerve at the carpal tunnel and association of slowed conduction with symptoms. Muscle Nerve 11:1149–1153

Nathan PA, Keniston RC, Myers LD et al (1998) Natural history of median nerve sensory conduction in industry: relationship to symptoms and carpal tunnel syndrome in 558 hands over 11 years. Muscle Nerve 21:711–721

Nilsson T (2002) Neurological diagnosis: aspects of bedside and electrodiagnostic examinations in relation to hand–arm vibration syndrome. Int Arch Environ Health 75:55–67

Rivner MH, Swift TR, Malik K (2001) Influence of age and height on nerve conduction. Muscle Nerve 24:1134–1141

Sakakibara H, Kondo T-A, Miyao M, Yamada S (1994) Digital nerve conduction velocity as a sensitive indication of peripheral neuropathy in vibration syndrome. Am J Indus Med 26:359–366

Sakakibara H, Hirata M, Hashiguchi T et al (1998) Affected segments of the median nerve detected by fractionated nerve conduction measurement in vibration-induced neuropathy. Indus Health 36:155–159

Salerno DF, Franzblau A, Armstrong TJ et al (1998) Nerve conduction studies among workers: normative values. Muscle Nerve 21:999–1005

Sanden H, Edblom HM, Hagberg M, Wallin BG (2005) Bicycle ergometer test to obtain adequate skin temperature when measuring nerve conduction velocity. Clin Neurophysiol 116:25–27

Seppalainen A-M (1972) Peripheral neuropathy in forest workers. A field study. Work Environ Health 9:106–111

Sluiter JK, Rest KM, Frings-Dresen MH (2001) Criteria document for evaluating the work-relatedness of upper-extremity musculoskeletal disorders. Scand J Work Environ Health 27(1):1–102

Stetson DS, Silverstein BA, Keyserling WM et al (1993) Median sensory distal amplitude and latency: comparisons between nonexposed managerial/professional employees and industrial workers. Am J Ind Med 24(2):175–189

Stromberg T, Dahlin LB, Brun A, Lundborg G (1997) Structural nerve changes at wrist level in workers exposed to vibration. Occup Environ Med 54:307–311

Tashjian EA, Ellenberg MR, Cross N et al (1987) Temperature effect on antidromic and orthodromic sensory nerve action potential latency and amplitude. Arch Phys Med Rehabil 68:549–552

Takeuchi T, Takeyama M, Imanishi H (1988) Ulstrastructural changes in peripheral nerves of the fingers of three vibration-exposed persons with Raynaud's phenomenon. Scand J Work Environ Health 14:31–35

Tong HC, Werner RA, Franzblau A (2004) Effects of aging on sensory nerve conduction parameters. Muscle Nerve 29:716–720

Viikari-Juntura E, Rauas S, Martikainen R et al (1996) Validity of self-reported physical work load in epidemiologic studies on musculoskeletal disorders. Scand J Work Environ Health 22:251–259

Virokanas H (1992) Dose–response relation between exposure to two types of hand–arm vibration and sensorineural perception of vibration. Occup Environ Med 52:332–336

Visser LH, Beekman R, Franssen H (2005) Short-segment nerve conduction studies in ulnar neuorpathy at the elbow. Muscle Nerve 31:331–338

Wallin G (2002) A method to reach adequate skin temperature for nerve conduction velocity testing. Rappot från Yrkes–0ch Miljömedicin 93:28–31

Wieslander G, Norbäck D, Göthe CJ, Juhlin L (1989) Carpal tunnel syndrome (CTS) and exposure to vibration, repetitive wrist movements, and heavy manual work: a case-referent study. Br J Indus Med 46:43–47