Associations among measures of median, ulnar, and sural nerve conduction and age, skin temperature, sex, and anthropometric factors were evaluated in a population of 105 healthy, asymptomatic adults without occupational exposure to highly repetitive or forceful hand exertions. Height was negatively associated with sensory amplitude in all nerves tested ($P < 0.001$), and positively associated with median and ulnar sensory distal latencies ($P < 0.01$) and sural latency ($P < 0.001$). Index finger circumference was negatively associated with median and ulnar sensory amplitudes ($P < 0.05$). Sex, in isolation from highly correlated anthropometric factors such as height, was not found to be a significant predictor of median or ulnar nerve conduction measures. Equations using age, height, and finger circumference for prediction of normal values are presented. Failure to adjust normal nerve conduction values for these factors decreases the diagnostic specificity and sensitivity of the described measures, and may result in misclassification of individuals.
Key words: nerve conduction studies • normal values • age • height • anthropometry

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EFFECTS OF AGE, SEX, AND ANTHROPOMETRIC FACTORS ON NERVE CONDUCTION MEASURES

DIANA S. STETSON, PhD, JAMES W. ALBERS, MD, PhD, BARBARA A. SILVERSTEIN, PhD, and ROBERT A. WOLFE, PhD

Normal values for distal nerve conduction measures are needed for clinical evaluation of individual patients and as control data for epidemiologic studies of work-related peripheral nerve compression disorders such as carpal tunnel syndrome. Although many control populations have been described,[5,6,11,17,24,28,29,38] variations in methodology, unspecified interelectrode distances, choice of digits, and type (or lack) of skin temperature control, as well as small sample sizes, limit their usefulness. Furthermore, while some reports specified that control subjects had no peripheral nerve disease,[17,28,29] none fully described the occupations of their subjects or specified a population with low exposure to known occupational risk factors for carpal tunnel syndrome such as force, repetitiveness, and vibration.[8,34,39]

This study describes the distribution of median, ulnar, and sural nerve conduction measures in a population of healthy, working adults without exposure to forceful or repetitive hand exertions, or segmental vibration. The hypotheses of no association between each nerve conduction measure and independent variables known or hypothesized to affect nerve conduction were tested. These factors included age,[4,19,26,38] skin temperature,[10,22,31] height,[7,21,32,35] and finger circumference.[3] A square-shaped wrist has been negatively associated with distal median nerve conduction.[18] Factors for which associations with specific nerve conduction measures were found to be statistically significant, biologically plausible, and clinically important were used to develop formulas which predict normal values.

MATERIALS AND METHODS

Population. Participants were salaried employees in a large corporation. Prior to age- and sex-strat-

From the Department of Environmental and Industrial Health, School of Public Health (Dr. Stetson), and Department of Neurology, Medical School (Dr. Albers), The University of Michigan, Ann Arbor, Michigan; *Safety & Health Assessment Research Program*, Washington Department of Labor and Industries, Olympia, Washington (Dr. Silverstein); and Department of Biostatistics, School of Public Health, The University of Michigan, Ann Arbor, Michigan (Dr. Wolfe)

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Address reprint requests to Dr. J. W. Albers, Department of Neurology, 1C 325 UH, Box 0032, The University of Michigan Medical Center, 1500 E. Medical Center Drive, Ann Arbor, MI 48109.

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ified random selection, employees on jobs thought to have repetitive or forceful hand exertions were excluded (e.g., secretaries, word processors). Of 271 randomly selected employees,[37] 249 were available for participation (Table 1). Of these, 75 refused to participate and 69 were excluded, leaving 105 study participants. Exclusion criteria were neurological symptoms, a self-reported history of a systemic illness or disorder affecting the central or peripheral nervous systems, or occupational exposure to forceful or repetitive hand exertions. Participants returned a self-administered questionnaire which included information on age, height, medical history, occupational history, and symptoms in the hand/wrist during the preceding year.

Physical Evaluation. All subjects had a screening neurologic examination performed by a neurologist (J. A.) and an additional examination of the hand and wrist performed by a nurse (B. S.). Index finger circumference was measured at the middle of the proximal phalanx with a narrow cloth measuring tape. The wrist anterior–posterior and medial–lateral diameters were measured with engineering calipers at the distal wrist crease.[18] Wrist ratio was calculated as the anterior–posterior diameter divided by the medial–lateral diameter.

Table 1. Subject selection criteria.

Consent form and questionnaire sent to randomly selected employees	n = 271
Less:	
• Unavailable for participation (n = 22)	n = 249
• Refused to participate (n = 75)	n = 174
Excluded (n = 69):	
• Medical exclusion* (n = 28)	
• >21 oz. ethanol/week or self-described problem (n = 2)	
• Numbness or paresthesia in hand occurring >3 times or lasting >1 week or occurring within prior week (n = 18)	
• Pain in volar hand/wrist (n = 2)	
• Current job has ≥4 h/d with repetitive/forceful hand exertions (n = 14)	
• Prior job, within previous 2.5 years, with repetitive/forceful hand exertions (n = 5)	
Study population	n = 105

*Medical reasons for exclusion were: diabetes mellitus, rheumatoid arthritis, abnormal thyroid function within previous 10 years, carpal tunnel syndrome, cervical disk disease (or symptoms of paresthesias in hands radiating from the neck), pregnancy, renal disease, hemophilia, prior radial mononeuropathy, prior cerebral infarction, chemotherapy, or use of anticonvulsant medication.

Nerve Conduction Studies. Nerve conduction studies were performed on the dominant hand of all subjects by the same certified electromyography technician. Using a TECA TD-5 electromyography machine and standard TECA surface electrodes, standard techniques of supramaximal percutaneous nerve stimulation and surface recording were employed.[9] Anatomic landmarks and standardized stimulation to recording electrode distances are shown in Table 2. Skin temperatures were monitored at the proximal crease of digits II and V and at the midcalf. Temperature was maintained above 32°C in the hand, and above 30°C in the calf, with an electric heating pad.

Sensory nerve action potential and compound muscle action potential amplitudes were measured from the baseline to the negative peak. Sensory latencies were measured to the onset of the negative deflection and the negative peak. Motor latencies were measured to the onset of the initial negative deflection. Median motor conduction velocity for the forearm was calculated using proximal and distal onset latencies. Terminal sensory conduction velocities were calculated by dividing the distal distance by the onset latency. F-wave latencies were measured as the minimal latency in a series of five responses.

Statistics. Student's t test was used to compare the difference between means for quantitative variables. Least squares regression was used for model building and hypothesis testing. The Pearson product moment was calculated for correlations of continuous variables. A P-value of <0.05 was used to define statistical significance. For model building, criteria for including an additional independent variable were that its coefficient have a P-value <0.05 and the partial R^2 associated with adding the variable be >0.05. Data for many of the nerve conduction measures did not follow a normal distribution. Prior to hypothesis testing, several transformations (natural log, square root, and inverse) were evaluated. Transformations resulting in a more normal distribution (Kolmogorov D-statistic and visual inspection of histograms/box plots) were used in the regression models. A model building approach was used for hypothesis testing. The independent variables were added in an order based on the strength of previously reported evidence for biologically plausible and significant associations: age, skin temperature, height, dominant index finger circumference, dominant wrist ratio, and sex.

Table 2. Description of nerve conduction measurements (n = 105): mean ± standard deviation (range).

Nerve	Stimulate	Recording electrodes (interelectrode distance)	Amplitude (μV or mV)	Latency (ms)	Conduction velocity (m/s)
Median sensory	Wrist	Digit II, proximal and distal phalanxes; 30–40 mm apart (140 mm)	32.7 ± 11.4 (14–68)	3.0 ± 0.2 (2.6–3.8)	60.2 ± 4.9 (47–70)
	Wrist	Digit IV, proximal and distal phalanxes; 30–40 mm apart (140 mm)	17.7 ± 8.6 (5–50)	3.0 ± 0.3 (2.5–4.1)	
	Midpalm, midthenar crease	Wrist, between PL* and FCR tendons (80 mm)	115 ± 41.5 (40–225)	1.8 ± 0.2 (1.4–2.4)	
Median motor	Wrist	Thenar muscle (70 mm)	12.5 ± 3.1 (4–19)	3.2 ± 0.4 (2.4–4.2)	
	Antecubital fossa, over brachial pulse	Thenar muscle (measured)			56.7 ± 2.9 (50–64)
Median F response	Wrist; cathode proximal	Thenar muscle		27.1 ± 2.2 (21.6–32.6)	
Ulnar sensory	Wrist	Digit V, proximal and distal phalanxes; 30–40 mm apart (140 mm)	28.9 ± 11.6 (9–60)	2.8 ± 0.2 (2.5–3.7)	63.0 ± 4.1 (48–74)
	Wrist	Digit IV, proximal distal phalanxes; 30–40 mm apart (140 mm)	19.0 ± 7.3 (6–38)	2.9 ± 0.2 (2.4–3.9)	
	Medial midpalm	Medial wrist (80 mm)	20.8 ± 8.4 (8–52)	1.7 ± 0.1 (1.5–2.2)	
Sural sensory	Midcalf	Lateral malleolus (140 mm)	17.5 ± 7.7 (6–48)	3.4 ± 0.3 (2.9–4.9)	52.2 ± 5.3 (36–64)

*PL = palmaris longus; FCR = flexor carpi radialis.

RESULTS

Population Description. The mean (±SD) age for men was 42.7 (±12.6) years and for women it was 37.8 (±10.0) years ($P < 0.05$); age and sex distributions are reported elsewhere.[37] Distributions of height, dominant hand index finger circumference, and dominant hand wrist ratio, stratified by sex, are summarized in Table 3. As a group, women were shorter and had a smaller finger circumference than men, but there was considerable overlap between the two sexes. Skin temperature at the index finger had a range of 32.0 to 35.5°C with a mean of 34.0 ± 0.82. For the fifth digit, the range was 32.5 to 36.0°C (34.1 ± 0.76), and at midcalf the range was 30.0 to 37.0°C (33.6 ± 1.3).

Table 3. Population distribution of height, dominant hand index finger circumference, and wrist ratio, by sex.

Measure	Female (n = 43)	Male (n = 62)	P-value
Height (cm)	163.8 ± 7.1 (150–180)	180.1 ± 6.4 (168–193)	0.0001
Dominant index finger Circumference (cm)	6.2 ± 0.4 (5.2–7.0)	7.2 ± 0.4 (6.4–8.1)	0.0001
Dominant wrist ratio	0.68 ± 0.03 (0.62–0.76)	0.68 ± 0.03 (0.61–0.78)	NS

Values expressed in mean ± SD (range).

Nerve Conduction Measures. Nerve conduction measurement distributions are shown in Table 2 with results for the median to ulnar comparison measures in Table 4. A description of the model building process is reported elsewhere[37]; results are shown in Tables 5 and 6. Age was significantly associated with all sensory amplitude measures ($P < 0.001$) and all conduction velocity and latency measures ($P < 0.05$) except sural conduction velocity. There was no consistent pattern of association between digit temperature and median or ulnar nerve conduction, likely because of the narrow range of temperatures maintained as part of the study protocol. There was a strong association between temperature and sural conduction

Table 4. Description of median to ulnar comparison measurements (n = 105).

Measurement	Mean ± SD	Percentile		Range
		95th	99th	
Median *minus* ulnar distal sensory latency (digit II vs. V)	0.1 ± 0.20	0.4	0.8	−0.3–0.8
Median minus ulnar distal sensory latency (digit IV)	0.1 ± 0.25	0.5	0.9	−0.4–1.2
Median minus ulnar sensory latency: midpalm (wrist)	0.1 ± 0.17	0.4	0.6	−0.2–0.6
Median to ulnar amplitude ratio (digit II/digit V)	1.2 ± 0.34	1.8	2.0	0.7–3.0

velocity ($P < 0.001$), probably related to the wider range of temperatures at the midcalf.

Height was negatively associated with sensory amplitude in all nerves tested ($P < 0.05$), and positively associated with median and ulnar sensory distal latencies ($P < 0.01$) and sural latency ($P < 0.001$). With age and skin temperature in the regression model, height explained an additional 25% of the variability in wrist–digit II median sensory amplitude. Index finger circumference was negatively associated with median and ulnar sensory amplitudes ($P < 0.05$). Adding finger circumference to regression models containing age, skin temperature, and height explained an additional 10% of the amplitude data variability. The association between median motor latency and wrist ratio ($P < 0.001$) explained an additional 12% of the variation.

Sex was highly correlated with height (Pearson correlation coefficient = −0.77, $P = 0.0001$) and dominant index finger circumference (Pearson correlation coefficient = −0.74, $P = 0.0001$). For the upper extremity distal conduction measures, sex was not important when these anthropometric characteristics were included in the models. For the sural nerve, there was a significant association between both amplitude (smaller in females, $P < 0.05$) and latency (longer in females, $P < 0.01$).

To assess the relative importance of each independent variable included in the regression models, the standardized regression coefficient (SRC) is listed below each coefficient (Tables 5 and 6), where:

$$\text{SRC} = \frac{\text{regression coefficient}}{} * \left(\frac{\text{SD of independent variable}}{\text{SD of dependent variable}}\right)$$

The SRC represents the change in the dependent nerve conduction measure (in units of its standard deviation) per unit change in the independent variable (in units of its standard deviation), and thus, is dimensionless. Although the magnitude of an SRC may not always reflect the importance of the independent variable, it is reasonable to use this measure to look for patterns of relative importance.

Overall, for upper extremity sensory amplitudes, age and index finger circumference appeared to be the most important independent variables. As an example of the range of amplitudes associated with a change in finger circumference, the prediction equation (Table 5) was used to calculate predicted values for wrist–digit II amplitude, using constant mean values for age and height. For a finger circumference of 8.0 cm (mean + 2 SD) the predicted amplitude is 25.0 μV, while for a circumference of 5.5 cm (mean − 2 SD) predicted amplitude is 39.5 μV. These results demonstrate the magnitude of effect that finger circumference can have on sensory amplitude.

For median and ulnar distal sensory conduction velocities and latencies, age and height appeared to be the most important variables. Because the conduction velocity data were never transformed, the magnitude of change in nerve conduction caused by a change in age or height can be assessed by looking at the regression functions for these measures (Tables 5 and 6). For the upper extremity conduction velocities shown, there was about a 1 m/s decrease in conduction velocity per decade increase in age. For the wrist–digit measures, there was about a 0.2 m/s decrease in conduction velocity for each centimeter increase in height (0.5 m/s per inch). Over a narrow range of age and/or height the magnitude of change was relatively minor, but large differences could have a substantial impact on the normal range of these measures. For median sensory conduction velocity and latencies, wrist ratio appeared to be the least important variable. A ±2 SD change in age produced a much greater change in the predicted value for wrist–digit IV latency (2.8 to 3.3 ms) than did a ±2 SD variation in wrist ratio (2.9 to 3.1 ms).[37]

Table 5. Summary of model building and prediction equations: upper extremity (prediction equations which are different from summary models are shown in italics).

Dependent variable	Constant	Regression coefficients (standardized regression coefficients) Age (yr)	Height (cm)	Index finger circumference (cm)	Wrist ratio*	Model R^2	$2\times(MSE)^{1/2}$
Median sensory							
Wrist–digit II							
Square root of amplitude	14.0	−0.035§ (0.42)	−0.020† (0.21)	−0.51‡ (0.32)		0.51	1.392
Conduction velocity	107.6	−0.13§ (0.32)	−0.14§ (0.30)		−25.4 (0.18)	0.22	—
Conduction velocity	*89.5*	*−0.12‡*	*−0.14†*			*0.17*	*8.858*
Negative inverse of latency	−0.58	0.00083§ (0.38)	0.00068‡ (0.27)		0.14† (0.18)	0.24	—
Negative inverse of latency	*−0.48*	*0.00078§*	*0.00066‡*			*0.21*	*0.046*
Wrist–Digit IV							
Natural log of amplitude	6.8	−0.017§ (0.42)	−0.0089† (0.19)	−0.27§ (0.37)		0.54	0.642
Negative inverse of latency	−0.67	0.0012§ (0.44)	0.00079‡ (0.26)		0.22‡ (0.23)	0.29	—
Negative inverse of latency	*−0.51*	*0.0011§*	*0.00076‡*			*0.24*	*0.056*
Midpalm–wrist							
Natural log of amplitude	5.24	−0.014§ (0.43)				0.19	0.684
Negative inverse of latency	−0.88	0.0018§ (0.36)			0.35† (0.20)	0.15	—
Negative inverse of latency	*−0.64*	*0.0016§*				*0.11*	*0.110*
Median motor							
Amplitude: no statistically significant association with the tested independent variables							
Conduction velocity	60.0	−0.081§ (0.33)				0.11	5.572
Negative inverse of latency	−0.85	0.00057† (0.19)	0.0017§ (0.48)		0.32§ (0.30)	0.34	—
Negative inverse of latency	*−0.61*	*0.00044*	*0.0016§*			*0.25*	*0.064*
Median F response							
Latency	−3.33	0.04§ (0.22)	0.17§ (0.77)			0.66	2.644
Ulnar sensory							
Wrist–digit V							
Natural log of amplitude	6.5	−0.015§ (0.42)	−0.0054 (0.14)	−0.25§ (0.38)		0.51	0.582
Conduction velocity	98.1	−0.099§ (0.29)	−0.18§ (0.46)			0.31	6.876
Negative inverse of latency	−0.53	0.00071§ (0.37)	0.00083§ (0.38)			0.29	0.038
Wrist–Digit IV							
Natural log of amplitude	5.96	−0.015§ (0.45)	−0.0088† (0.24)	−0.14† (0.22)		0.45	0.588
Negative inverse of latency	−0.55	0.00080§ (0.36)	0.00097§ (0.38)			0.29	0.046
Midpalm–wrist							
Square root of amplitude	10.5	−0.034§ (0.45)	−0.026§ (0.31)			0.31	1.510
Negative inverse of latency	−0.78	0.00071† (0.22)	0.00090‡ (0.25)			0.12	0.070

Wrist ratio = anterior–posterior diameter/medial–lateral diameter.
†$P < 0.05$; ‡$P < 0.01$; §$P < 0.001$.

Table 6. Prediction equations: lower extremity.

Dependent variable	Constant	Regression coefficients (standardized regression coefficients) Age (yr)	Height (cm)	Midcalf temperature (°C)	Sex*	Model R^2	$2\times(MSE)^{1/2}$
Sural sensory							
Natural log of amplitude	7.6	−0.019§ (0.52)	−0.023§ (0.55)		−0.29† (0.33)	0.37	0.694
Conduction velocity	30.7	−0.051 (0.12)	−0.27§ (0.53)	2.1§ (0.52)	−2.6 (0.24)	0.47	7.986
Negative inverse of latency	−0.30	0.00052‡ (0.23)	0.0018§ (0.66)	−0.0099§ (0.47)	0.023‡ (0.41)	0.48	0.042

*Sex coded as male = 0; female = 1.
†$P < 0.05$; ‡$P < 0.01$; §$P < 0.001$.

DISCUSSION

The results describe the statistical significance of associations. Final model development also included a review of whether: (a) the association was biologically plausible; (b) the magnitude of association was large enough to be of clinical importance; (c) the association was supported by previous studies; and (d) the associated independent variable was intrinsic or could be controlled through standardized testing procedures.

Age. The decrease in nerve conduction velocity and sensory amplitude associated with increasing age has been well documented,[4,6,19,25–27,38] and attributed to a decreased number of nerve fibers,[25] a reduction in fiber diameter,[19,25] and changes in the fiber membrane.[4,23] The 1.3 m/s decrease in median sensory distal conduction velocity and the 0.8 m/s decrease in motor conduction velocity per decade of aging found in this study are comparable to previous reports.[6,38] Our data showed an average loss of about 5 μV per decade for wrist–digit II sensory amplitude. This was somewhat larger than the 1.5 μV per decade loss in digit III–wrist sensory amplitude reported by Buchthal et al.[6] or the 3.8 μV per decade loss in wrist–digit III sensory amplitude reported by Tackmann.[38] Although the magnitude of change is relatively small within a narrow age range, it does affect predicted normal values. A prolonged latency in a young age group will be missed if normal values based on an older age group are used.[6]

Temperature. Over relatively wide temperature ranges (e.g., 21 to 36°C[4] or 29 to 38°C[15]), motor and sensory nerve conduction velocities have a positive linear relationship with body temperature.[10,20] With cooling, motor and sensory amplitudes increase and conduction values decrease.[10,16,22,31] Cooling is thought to affect muscle and nerve membrane function, particularly the sodium ion channel.[10,20,31]

Our ability to detect true associations was limited by the relatively narrow temperature range in the upper extremity (32 to 36°C), as well as sources of error such as the difference between surface temperature and the near nerve temperature.[10] For the sural nerve, where there was a 7° temperature range (30 to 37°C), relatively strong associations between surface temperature and both conduction velocity and latency were found (partial R^2 from 0.29 to 0.35). The slope of 2.3 m/s per °C corresponds to previous reports.[4,36] Unlike individual characteristics such as age and height, temperature is an extrinsic factor which can be controlled. We recommend use of a standard protocol for warming the extremities, as proposed by others.[10,20]

Height. The negative relationship between height and sensory amplitudes was a consistent finding in all three sensory nerves tested. While controlling for age and surface temperature, height explained much of the sensory amplitude variation (partial R^2 in 0.20 to 0.25 range). Our results duplicated those of others who have found a strong negative correlation between height and either sural or peroneal conduction velocity.[7,21,33,35] Controlling for age and temperature, the 0.17 m/s decrease in sural conduction velocity per centimeter increase in height (0.44 m/s per inch) was somewhat larger than previously reported.[32,35] Also, controlling for

sex, there was a 0.27 m/s per centimeter decrease (0.68 m/s per inch) (Table 6). We also found strong associations between height and both median and ulnar sensory terminal conduction velocities and latencies (average sensory terminal conduction velocity decrease of 0.16 m/s per centimeter). This was consistent with Lang's[21] report of a positive correlation between height and radial sensory conduction velocity. While differing from the lack of correlation between height and median sensory conduction velocity reported by Soudmand,[35] our larger population, and its wider age range, may have given more power to detect a true association. In addition, the associations were found only for distal measures (e.g., wrist–digit or midpalm–wrist), whereas Soudmand reported forearm conduction velocity.

A negative correlation between distal fiber diameter and height may best explain both decreased conduction velocity and amplitude. Campbell[7] proposed that a decrease in diameter occurs abruptly at a given distance from the cell body. In mature rabbit nerves, Williams[40] found that peripheral motor axon diameter was about half that of ventral spinal nerve root fibers and, despite an increase in myelin sheath thickness, there was an overall decrease in total fiber diameter. Buchthal and Rosenfalck[4] reported histologic characteristics of median nerve fibers from 2 men and found no evidence of nerve fiber tapering between the wrist and axilla, but the digital nerve mode diameter was about 3 μm smaller than that found proximally. Our results, which showed an association with median sensory distal conduction velocities, are consistent with the contention that tapering occurs distal to the wrist. The association between height and ulnar, but not median, midpalm–wrist sensory measures may have an anatomical basis, e.g., more proximal tapering in the ulnar than median nerve, or may be an artifact.

There is strong evidence for including height in models used to predict normal values for distal lower extremity measures[7,21,32,35] and F-wave latencies.[33] There is increasing evidence that height is an important predictor of distal sensory upper extremity nerve conduction.[21,32] Use of "normal" values based on a control population is inappropriate, because they will be influenced by the population's height distribution.[33]

Finger Circumference. Bolton[3] has reported a negative correlation between finger circumference and both median and ulnar antidromic sensory amplitudes. Although we inferred digit V circumference from that measured for digit II, our results replicated his findings. With age and height held constant, finger circumference had a substantial effect on distal sensory amplitude. Differences in finger circumference are due primarily to differences in bone mass and subcutaneous tissue. Bone mass is presumed to be positively correlated with height, which was controlled for in our analysis. Therefore, the association between finger circumference and amplitude is probably related to subcutaneous tissue depth, which is the major determinant of the distance between the digital nerves and the recording (ring) electrode. Since amplitude decreases as the distance between nerve and electrode increases,[4] our results were in the expected direction.

Wrist Ratio. A positive association between wrist ratio and median sensory latency has been reported in a mixed population of patients and control subjects[18] and, for median motor latency, in a population with symptoms compatible with carpal tunnel syndrome.[13] We found weak positive associations between wrist ratio and both median sensory and motor latencies which were absent when wrist ratio was tested in single factor models. No biological hypothesis for the relationship between wrist ratio and median nerve latency has been proposed, and the observed association seems counterintuitive. In the biomechanical model of the wrist proposed by Armstrong,[1] a small radius of tendon curvature is predicted to increase the risk of tendon and synovial trauma within the carpal tunnel area; subsequent synovitis and synovial membrane thickening cause median nerve compression.[2] In contrast, a large wrist ratio (more "box-shaped" wrist) would be associated with a large radius of tendon curvature. In addition, Richman[30] has pointed out that measurements based on bony landmarks cannot be used to estimate the distal third of the carpal tunnel, a potential area of nerve compression, and Winn[41] has shown that carpal tunnel area itself is not a risk factor for carpal tunnel syndrome. Because of the relatively weak associations and small magnitudes of effect in this study, and the lack of a biologically plausible model, we do not recommend controlling for wrist ratio. However, the association is intriguing and subsequent biological explanations may increase our understanding of functional anatomy within the carpal tunnel.

Sex. Like others who have reported that differences in conduction velocity or amplitude initially

attributed to sex disappeared once anatomical factors were taken into account,[3,7,35] we found no association between sex and median or ulnar nerve conduction measures that could not be attributed to the correlation between sex and height or sex and dominant finger circumference. For the sural nerve, using a model which also included age, midcalf temperature, and height, women were found to have a significantly smaller amplitude and slower conduction velocity then men. The most plausible hypothesis is that both sex and sural nerve conduction and amplitude are correlated with an anatomical or physiological factor which we did not measure. Although some have recommended that normal values be segregated by sex, as a surrogate measure for anatomical differences,[3] we feel it is more appropriate to incorporate age, height, and finger circumference into regression models used to predict normal values because there is considerable overlap between the female and male distributions of these factors (Table 3).

SUMMARY OF RECOMMENDATIONS

Adjusting normal nerve conduction values for known anatomical or physiological determinants such as age, height, and finger circumference increases the diagnostic sensitivity of these measures.[3,21,35] In Table 7, the predicted normal ranges for 2 subjects with different characteristics are shown and compared to the conventional abnormal value (mean ±3 SD) used in our laboratory. The predicted values and confidence intervals were calculated from the regression models shown in Table 5. The values of the independent variables used in each model (e.g., age = 25) are shown for each subject. Using the laboratory criteria, a value within the 99% confidence interval of normal results for the tall, older individual could mistakenly be interpreted as abnormal (e.g., wrist–digit II sensory amplitude greater than 7.3 μV and less than 15 μV) and might be used to validate unnecessary surgery. Conversely, values beyond the 99% confidence interval (i.e., abnormal) for the relatively short 25-year-old could be interpreted as normal and result in a failure to diagnose and treat. This is a predictable occurrence whenever the normal range is based on values found for the control population as a whole.

Predicting normal values in the context of pertinent individual characteristics will increase sensitivity, and possibly specificity, of electrodiagnostic procedures. Although rarely used in the electrodiagnostic field, prediction formulae developed in the spirometric lung function field have consistently included height, sex, age, and ethnic group—factors known to affect lung function.[12] While there is continuing debate about the best statistical method to use,[12,14] computer technology makes the development of an algorithm which

Table 7. Comparison of predicted normal range derived from equations with laboratory definition of an abnormal value.

Measure	Individual characteristics	Predicted value	99% confidence interval	Abnormal per lab.
W-digII sensory amplitude				
	Age 25, 163 cm, 5.8-cm index finger circumference	47.6 μV	(25.7–76.1)	
				<15 μV
	Age 55, 183 cm, 7.6-cm index finger circumference	20.4 μV	(7.27–40.2)	
W-digII sensory latency				
	Age 25, 163 cm	2.8 ms	(2.4–3.4)	
				>3.9 ms
	Age 55, 183 cm	3.1 ms	(2.6–3.9)	
Midpalm-W sensory latency				
	Age 25	1.7 ms	(1.4–2.2)	
				>2.5 ms
	Age 55	1.8 ms	(1.4–2.5)	
Median motor latency				
	Age 25, 163 cm	2.9 ms	(2.4–3.9)	
				>4.5 ms
	Age 55, 183 cm	3.4 ms	(2.7–4.7)	

predicts the normal range of values for each individual in the context of pertinent characteristics a relatively simple task.

Predicted normal values can be calculated from the regression equations given in Tables 5 and 6, or the 95% prediction interval can be estimated from the following equation:

$$95\% \text{ prediction interval} = Y \pm 2*[(\text{MSE})^{1/2}]$$

where Y = the predicted value, and MSE = the mean square for error. The values for $2*[(\text{MSE})^{1/2}]$ are given in Tables 5 and 6. An abnormal value can be defined as one which is outside the 95% prediction interval.

None of the independent variables tested helped predict the outcome for median motor amplitude or the median-to-ulnar comparison measures. The latter measures utilized either the ratio or the difference between a median and an ulnar nerve measure in the same hand in the same person. It is therefore expected that comparison measures need not be corrected for age, height, or skin temperature. The standard deviations for median motor amplitude and the comparison measures are shown in Tables 2 and 4. Comparison measures data did not follow a normal distribution; percentile and range may give more appropriate descriptive information than increments of the standard deviation.

In summary, in randomly selected adults without occupational exposure to high force or repetitive hand exertions, age, height, and index finger circumference were found to be important predictors of median, ulnar, and sural nerve conduction measures. These factors were incorporated into equations used to predict normal values for these measures.

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