Increased weight and, more recently, body mass index (BMI), have been suggested as risk factors for carpal tunnel syndrome (CTS). In an effort to determine the relative risk (RR) of obesity in the development of CTS, 949 patients who had an evaluation of the right upper extremity that included motor and sensory conduction studies of the median and ulnar nerves were reviewed. Of these patients, 261 were diagnosed with a median mononeuropathy at the wrist. Those individuals who were classified as obese (BMI > 29) were 2.5 times more likely than slender individuals (BMI < 20) to be diagnosed with CTS. Forty-three percent of obese women and 32% of obese men had the diagnosis of CTS compared to 21% of slender women and 0% of slender men.

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**THE RELATIONSHIP BETWEEN BODY MASS INDEX AND THE DIAGNOSIS OF CARPAL TUNNEL SYNDROME**

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There are many factors thought to place an individual at risk for the development of carpal tunnel syndrome (CTS). Numerous personal cofactors (see Table 1) have been reported but very few have been quantified as to the strength of the association.2,4,5 Several investigators have reported that individuals with CTS were heavier and shorter than the general population.3,5,6,11,16,17 Cannon3 noted that 27% of individuals (8 of 30) with CTS were obese compared to 12% (11 of 90) in a control population; this difference did not reach statistical significance. Falck and Aarnio6 described 17 butchers, most of whom were obese, and found that 53% had CTS. There was no difference in the body mass index (BMI) between butchers with or without CTS. Dieck and Kelsey5 found an increased prevalence of CTS, within an adult female population, among individuals with short stature, greater weight and recent weight gain. The BMI was significantly higher in the CTS group (27 kg/m² vs. 25 kg/m², \( P = 0.01 \)). Vessey et al.,15 found that the risk for CTS among obese women was double that of slender women.

Within an industrial population, Nathan et al.,11 demonstrated that a higher BMI was associated with a higher prevalence of median mononeuropathy. They found a relative risk (RR) of 4.1 for obese individuals compared to slender individuals. This relationship was more pronounced in men (RR = 5.1) than in women (RR = 2.7). This study did have a number of methodological flaws of which the most prominent was an analysis by hand instead of by person.

Most prior studies were relatively small and primarily addressed the relationship between CTS and obesity among women. Nathan's study was larger and included both genders but surveyed an industrial-based population. The purpose of this study was to determine the risk of obese individuals for the development of CTS in a large nonindustrial population.

**METHODS**

A cross-sectional study design was employed to study the relative risk for CTS related to body size. Of approximately 6000 patients seen in the elec-
Table 1. Personal cofactors related to the occurrence of carpal tunnel syndrome.

<table>
<thead>
<tr>
<th>Medical conditions</th>
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<tbody>
<tr>
<td>Diabetes</td>
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<tr>
<td>Thyroid disease</td>
</tr>
<tr>
<td>Connective tissue disorders</td>
</tr>
<tr>
<td>Amyloidosis</td>
</tr>
<tr>
<td>Acromegalia</td>
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<tr>
<td>Vitamin B&lt;sub&gt;6&lt;/sub&gt; deficiency (?)</td>
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<tr>
<td>Age</td>
</tr>
<tr>
<td>Gender</td>
</tr>
<tr>
<td>Oral contraceptive use</td>
</tr>
<tr>
<td>Oophorectomy</td>
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<tr>
<td>Wrist dimension</td>
</tr>
<tr>
<td>Weight</td>
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<tr>
<td>Stature</td>
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<td>Body mass index (BMI)</td>
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The electrodiagnostic laboratory during 1991 and 1992, 949 patients were identified who were: (1) referred for evaluation of the right upper extremity; (2) completed an evaluation of the median and ulnar, motor and sensory, nerves; and (3) reported height, weight, and age. The diagnosis of a median mononeuropathy was based on the relative difference in peak sensory latency between the median and ulnar responses (14 cm, antidromic stimulation) using a threshold of abnormality of 0.5 ms or longer. The standard within our laboratory dictated that if there was a clinical suspicion and the standard sensory evoked responses were borderline, median and ulnar midpalmar responses were obtained; a difference of 0.3 ms or greater was considered diagnostic of a median mononeuropathy. If the median sensory responses were absent, median motor latency was compared to ulnar motor latency with a 1.7-ms difference being defined as abnormal. All subjects had midpalm temperatures recorded and were warmed if the temperature was below 32.0°C. The combination of clinical symptoms and a median mononeuropathy was defined as CTS. All further reference to CTS among subjects in this study will use this case definition.

The BMI (kg/m<sup>2</sup>) was calculated for each subject and subgroups were created based upon the BMI distribution reported for the state of Michigan: slender, BMI < 20; normal, BMI 20–25; heavy, BMI 25–29; and obese, BMI > 29.1 Age was also divided into three age groups: (1) less than 45; (2) 45–64; and (3) 65 or older.

STATISTICS

Statistical analysis was done using STATA.15 Preliminary analysis consisted of 2 × K tables of diagnosis (CTS and “other”) by body size (slender, normal, heavy, and obese), age group, and gender. Pearson chi-squared statistics were used to determine the variation from expected values. Risk ratios were determined for body size, age group, and gender. Mantel-Haenszel statistics were used to evaluate for interactions between the independent variables. Logistic regressions were performed using the diagnosis of CTS versus “other” as the dependent variable and age, gender, and BMI as the independent variables. Age was analyzed as a continuous as well as an ordinal variable as was BMI. Interactions as well as second and third order variables were tested in the model. Finally, multiple linear regressions were developed with the difference between the median and ulnar sensory latencies as the dependent variable and age, gender, and BMI as the independent variables. The same statistical analyses were performed using a subset of the population; individuals with CTS (but no other codiagnosis) were compared to individuals with a normal study. All results are reported as a mean and 1 standard deviation [mean (1 SD)]. All risk ratios or odds ratios are presented with the 95% confidence interval following in parentheses. Any interval that includes the number 1.0 is not significant at the P = 0.05 level.

RESULTS

Of the 949 subjects, the average age was 46.1 (14.7) with 56% being female. The mean BMI was 26.8 (6.4) with 28.6% being classified as obese, 27.8% as heavy, 35.6% as normal, and 8% as slender. Two hundred sixty-one subjects (27.5%) were diagnosed with CTS, while 342 (36%) were characterized as normal, with the remaining 36.5% of subjects having some other electrodiagnostic abnormality (i.e., cervical radiculopathy, brachial plexopathy, ulnar neuropathy, peripheral polyneuropathy, etc.).

Figure 1 demonstrates the relationship between body size and the percent of patients diagnosed with CTS. Only 16% of slender individuals were diagnosed with CTS compared to 39% of obese subjects. The relative risk for obese individuals compared to slender individuals, without controlling for other variables, is 2.5 (95% CI = 1.4, 4.2). This means obese individuals are 2.5 times as likely to have the diagnosis of CTS compared to a group of slender individuals. The mean BMI for those individuals with CTS was 28.9 (6.8) and 26.2 (6.0) in the remainder of the population. The distribution of BMI for each diagnostic group is presented.
FIGURE 1. The percent of patients with carpal tunnel syndrome by body type: slender (BMI < 20), normal (BMI 20–25), heavy (BMI 25–29), obese (BMI > 29).
with the exception that gender was not a significant factor in the analysis.

DISCUSSION

The findings of this study support the hypothesis that individuals with a higher BMI are at increased risk for CTS. The pathophysiology that would explain this relationship is not well understood. These observations support the findings of Nathan et al., as well as Vessey et al., but still explain only a small portion of the variance related to the diagnosis of CTS or electrodiagnostic abnormalities involving the median nerve. This study also reaffirms that the highest prevalence of CTS is in the age group 45–65 years and that women are at a higher risk than men for CTS.

If a causal relationship between obesity and a slowing of median conduction across the wrist exists, it may relate to increased fatty tissue within the carpal canal or to increased hydrostatic pressure throughout the carpal canal in obese individuals compared to normal or slender individuals. Magnetic resonance imaging has demonstrated increased fatty tissue in the carpal canal being associated with CTS in 1 patient, but there has not been a systematic study of the intracarpal canal anatomy in obese compared to slender individuals. Likewise, intracarpal canal pressures have been demonstrated to be higher in individuals with CTS, but the relationship between body size and intracarpal canal pressure has not been explored.

The finding that BMI is correlated with the median but not ulnar sensory latencies as the dependent variable demonstrated that BMI, age, and gender were significant but only accounted for 8% of the total variance. Within this model, BMI was the most influential variable accounting for 5% of the variance; age and gender accounted for the other 2.6% explained in the model. Second order variables and interactions were not significant.

A secondary analysis was done on a subset of the population that had a diagnosis of CTS but no other electrodiagnostic abnormality and compared them to individuals classified as normal. The findings were very similar to the population as a whole with the exception that gender was not a significant factor in the analysis.

FIGURE 2. The distribution of body mass index (BMI) among patients with a diagnosis of carpal tunnel syndrome versus patients with arm/hand complaints and no evidence of CTS on nerve conduction studies ("other"). (*) represents the mean and 1 standard deviation. The scale on the right border represents the number of patients represented in the histogram.

2.1 times the risk for getting CTS compared to men. The apparent relationship between gender and slender body size was not significant. This most likely reflects the small number of the slender men in the population \( (n = 18) \). There was a trend suggesting that slender men were protected against CTS, but the sample size of this subgroup was small and the finding was not statistically significant.

The multiple linear regression model with the difference between median and ulnar sensory latencies as the dependent variable demonstrated that BMI, age, and gender were significant but only accounted for 8% of the total variance. Within this model, BMI was the most influential variable accounting for 5% of the variance; age and gender accounted for the other 2.6% explained in the model. Second order variables and interactions were not significant.

The finding that women were more likely to have a higher prevalence of CTS than men supports other population based studies but differs from the worker compensation–based data on CTS reported by Franklin. In the workplace, the risk for women is only 10–20% higher than men as
opposed to 300% reported in population-based studies.7,11

The population reported here, from a hospital-based EMG laboratory, does not represent the general population and is substantially different from the industrial-based survey studied by Nathan.11

The fact that BMI was a significant risk factor in both studies strengthens its importance as a risk factor for CTS. Also this is not an association that is unique to women. Despite the prevalence of CTS being higher in women, obesity seems to be a more significant risk factor for men in our study as well as Nathan's. Conversely, being slender may be a protective factor with this being most striking in men. This was a retrospective study and was not able to factor in the role of exposure to cumulative trauma or to define the influence of the concurrent medical diagnoses on the prevalence of CTS. The possible association between obesity and the development of type II diabetes may be a confounder that we are unable to assess in this study.

Additionally, caution must be used in applying this information in the clinical setting. As demonstrated by Figure 1, not all obese individuals develop CTS. Sixty-five percent of the obese individuals in this study did not have CTS. Conversely, 20% of thin and normal individuals did develop CTS. The mechanism underlying this relationship is not understood and causation cannot be determined.

REFERENCES