Archives of Rehabilitation Research and Clinical Translation (2022) 4, 100238

ACRM
AMERICAN CONGRESS OF REHABILITATION MEDICINE

Archives of Rehabilitation Research and Clinical Translation

Archives of Rehabilitation Research and Clinical Translation 2022;4:100238

Available online at www.sciencedirect.com

Original Research

Longitudinal Median Nerve Ultrasound Changes in Individuals With Spinal Cord Injury and an Age- and Sex-Matched Nondisabled Cohort

Minh Quan T. Le, MD [a], Elizabeth R. Felix, PhD [c,d], Robert Irwin, MD [c], Diana D. Cardenas, MD [c], Rachel E. Cowan, PhD [b,e]

[a] *Department of Physical Medicine and Rehabilitation, University of Miami Miller School of Medicine, Miami, FL*
[b] *Department of Physical Medicine & Rehabilitation, University of Alabama at Birmingham, Birmingham, AL*
[c] *South Florida SCI Model System, Department of Physical Medicine & Rehabilitation, University of Miami Miller School of Medicine, Miami, FL*
[d] *Department of Physical Medicine and Rehabilitation, Miami Veterans Administration Medical Center, Miami, FL*
[e] *The Miami Project to Cure Paralysis, Department of Neurosurgery, University of Miami Miller School of Medicine, Miami, FL*

KEYWORDS
Ultrasonography;
Spinal cord Injuries;
Carpal tunnel syndrome;
Median nerve;
Rehabilitation

Abstract *Objectives:* To assess the natural history for development of carpal tunnel syndrome (CTS) in persons with acute spinal cord injury (SCI) at 1 year postdischarge from initial rehabilitation and to assess baseline median nerve (MN) cross-sectional area (CSA) above/below 10 mm^2 correlates with any longitudinal changes in quantitative ultrasound (US) of the MN.
Design: A prospective cohort study of persons with acute SCI evaluated for CTS using quantitative US and compared to a group without SCI (non-SCI).
Setting: Academic medical center.
Participants: N=69 total (N=34 SCI, N=35 non-SCI). The average age in both groups was 28 and the SCI group included 30 males and 2 females and the non-SCI group included 30 males and 3 females.
Interventions: Not applicable.

List of abbreviations: BMI, body mass index; CSA, cross-sectional area; EDS, electromyography studies; CTS, carpal tunnel syndrome; GS, grayscale; MN, median nerve; PE, physical exam; SCI, spinal cord injury; US, ultrasound.

Funded by grant the National Institute on Disability, Independent Living, and Rehabilitation Research (NIDILRR), Award # 90SI5001.
Disclosures: None.
Cite this article as: Arch Rehabil Res Clin Transl. 2022;4:100238

https://doi.org/10.1016/j.arrct.2022.100238

Main Outcome Measures: The primary outcome was the change in quantitative US parameters of the MN, including CSA and grayscale, from baseline to 1-year follow-up in those with SCI and those without SCI. CTS symptomatology and physical exam sum score and US measures for dominant and nondominant arms were considered secondary outcomes.
Results: The SCI had darker nerves at baseline (P=.036, nondominant), greater CTS symptoms at follow-up ($P \leq .036$, bilateral), and no differences in all change scores (all $P \geq .056$). Individuals with smaller nerves at baseline had larger increases in nerve size (P=.029, nondominant) vs those with larger nerves. Change in CTS symptoms CSA (nondominant) and nerve echogenicity (dominant) were inversely associated with their respective baseline values (all $P \leq .045$).
Conclusions: We observed few differences between the SCI group and the non-SCI control group and between those with smaller vs larger MN. In general, MN pathology changes (CTS symptoms and US variables) over 1 year were more common in the nondominant arm and appear to be a function of MN pathology at enrollment. Individuals with SCI may experience increased CTS symptoms as soon as 1 year after injury.

Currently in the United States, there are 249,000 to 363,000 persons living with a spinal cord injury (SCI), and since 2015, 39.5% of new SCIs have resulted in paraplegia (complete and incomplete).[1,2] Compared to those without SCI, persons with paraplegia rely more on the upper extremities for functional mobility, including locomotion, transfers, and pressure relief, as well as activities of daily living.[3-7] Repetitive activities, such as manual wheelchair manipulation, increase the likelihood of developing carpal tunnel syndrome (CTS).[3,8-10]

CTS is the most common upper extremity entrapment neuropathy, accounting for 90% of all such neuropathies in able-bodied individuals.[11-13] CTS prevalence is estimated at 49%-73% among people with SCI who use wheelchairs[7,9,14-16] vs 3%-4% in the general population.[17,18] In the general population, risk factors for CTS include occupation, alcoholism, advanced age, diabetes, obesity, hypothyroidism, pregnancy, and rheumatoid arthritis.[13,19,20] For people with SCI, wheelchair usage over time is a risk factor for both prevalence and disease severity.[4,9] In individuals with paraplegia, who rely on upper extremities for mobility and daily activities, CTS can be especially detrimental to quality of life.[6] Early diagnosis and intervention are crucial in preventing functional decline and long-term sequelae.[6,9]

Patients with CTS commonly experience paresthesia and pain in median nerve (MN) distribution and develop thumb abduction and opposition weakness with disease progression.[10,13] CTS results from MN compression and traction, which cause microcirculation nerve injury, synovial hypertrophy, and increased pressure in the carpal tunnel.[18] These symptoms are evident in individuals who use wheelchairs; for example, the MN swelling occurring in wheelchair basketball players after acute propulsion.[10] CTS history and physical examination have varied sensitivity and specificity for diagnosing CTS.[13] Therefore, nerve conduction and electromyography studies (EDS) are used to confirm CTS diagnosis and prognosis.[21] Despite their high sensitivity and specificity for diagnosis of CTS (85% and 95%, respectively), EDS is invasive and offers no anatomic information about the cross-sectional area (CSA) of the MN or its surrounding structures.[22,23]

Ultrasonography (US) is a popular diagnostic tool for CTS because it is readily available in clinics as a quick point of contact assessment.[21] Patients prefer the less expensive, noninvasive US to EDS.[12,24] US allows for the visualization of carpal tunnel anatomy and real-time assessment of dynamic changes within.[5] Ultrasonographic parameters such as hypervascularity, hypoechogenicity, and MN-to−carpal tunnel size ratio have been studied in evaluating CTS.[10,25] A systematic review found that using CSA to diagnose CTS yielded a sensitivity of 65%-97%, specificity of 73%-98%, and accuracy of 79%-97%.[26] Normal MN CSA is between 7.2 and 9.8 mm^2; CSA greater than 10 mm^2 is suggestive of CTS[12,27]; CSA greater than 14 mm^2 can rule in CTS, and CSA less than 8 mm^2 can rule it out.

Our objectives were to describe changes in CTS symptoms and MN US parameters occurring between discharge from initial rehabilitation and 1 year postdischarge in individuals with SCI, determine whether these longitudinal changes are unique to individuals with SCI compared to those without SCI, and assess what factors are associated with CTS symptoms and MN US parameters at discharge and change over 1 year. We hypothesized that MN baseline US measures and longitudinal changes in MN US measures would differ between participants with paraplegia due to traumatic SCI and a control group without SCI.

Methods

Study participants

Participants were recruited from patients who (1) were admitted to our institution's inpatient rehabilitation unit after experiencing a traumatic SCI; (2) were enrolled in the SCI Model System national database; (3) were within 6 months of initiating injury; (4) had an International Standards for Neurological Classification of SCI classification of A, B, C, or D at admission to inpatient rehabilitation; and (5) were at least 20 years old. Nondisabled control participants were recruited via posted flyers and word of mouth. Control participants were screened for sex and age to match the age (±5 years) and sex of an enrolled participant with SCI before being invited to participate. Potential participants, both those with SCI and controls, were screened out if they

reported a history of rotator cuff tears, fractures, or surgery on both shoulders. Non-SCI control participants were also excluded if they used an assistive device for ambulation. All subjects underwent informed consent processes as approved by the University of Miami Institutional Review Board.

Sample size justification

Sample size was determined by the parent study. The parent study was an observational study powered to detect differences in the proportion of individuals with and without SCI who had a ≥5% increase in supraspinatus tendon thickness (ie, worsening pathology) over the course of the first year postinjury. A sample of N=34 (N=17 with SCI, N=17 without SCI) provided 80% power at α=.05 to detect a difference of 45% in the proportion of individuals who experienced worsening tendon pathology.

Testing protocol

Study activities were completed in a 3-hour visit at enrollment (SCI=near discharge) and 1 year later. We documented demographics, physical exam (PE) sum score, and quantitative ultrasound metrics.

Physical exam

A physiatrist (R.W.I.) performed a bilateral physical exam following the Collaboration on Upper Limb Pain in SCI study guidelines.[3] Higher scores represent greater clinically graded median neuropathy (range, 0-14).

Ultrasound imaging

Following published procedures,[21] US images were collected on both the dominant and nondominant wrist using BK Medical's Mini focus 1402a,[a] with a 5-12 MHz 50-mm linear transducer. Images were taken using the MN border at the level of the pisiform bone as a reference. The first image was unmarked (unlabeled image), and in the second, the MN boundary was roughly traced (labeled image).

Data extraction and analysis

Following procedures described by Impink et al,[21] 2 MN variables, CSA and grayscale (GS), were extracted from each unlabeled image using a custom MATLAB script.[b] Two investigators, blinded to participant, SCI status (yes/no), and image time (baseline/follow-up), processed each unlabeled image twice, with the median of all 4 processing trials used for analysis. The median was selected rather than the average to reduce the effect of variance across the 4 trials. For each image, the MATLAB script presented the labeled and unlabeled images side by side and prompted the investigator to trace the boundary of the MN (excluding the hyperechoic epineurium). CSA (mm^2) was computed as the total number of pixels inside of the boundary trace (19 pixels per millimeter). GS was computed as the average value of all pixels inside the boundary trace. One-year change scores were computed as follow-up minus baseline.

Participants' characteristic variables (days postinjury of the baseline assessment, days between visits, age, height, weight, body mass index [BMI]), PE scores, and US parameters (CSA and GS) at baseline, follow-up, and change were assessed for a normal distribution (Kolmogorov-Smirnov test). Because the majority of variables were nonnormally distributed, nonparametric (Spearman rho, Mann-Whitney U) tests and measures of central tendency (median) and variance were computed. To determine whether there were differences between (a) SCI and non-SCI groups and (b) baseline CSA above/below 10.0 mm^2 with regards to demographics and PE, chi-square and Mann-Whitney U tests were used. To determine whether there were baseline and 1-year follow-up change differences between (a) SCI and non-SCI groups and (b) baseline CSA above/below 10.0 mm^2, we used the Mann-Whitney U test. To determine what factors were associated with US parameter baseline and change scores, Spearman rho and point biserial correlations were used. For all tests, US parameters were assessed separately for each arm (dominant, nondominant). Significance was set a priori at $P \leq .05$. False discovery rate was used to control for type I error across the 36 total comparisons of the SCI/non-SCI and baseline CSA above/below 10.0 mm^2 groups.[28]

Results

Descriptives

Seventy individuals were enrolled; 69 (34 SCI/35 non-SCI) completed baseline testing, and 55 (24 SCI/31 non-SCI) completed 1-year follow-up testing. One individual was administratively withdrawn post consent secondary to an inability to visualize the MN on US. Demographic characteristics of the SCI/non-SCI and baseline CSA below/above 10 mm^2 groups are summarized in tables 1 and 2, respectively.

At baseline, individuals with SCI were heavier (P=.03) than individuals in the control group (table 1). The median age in both groups was 28. Chi-square tests indicated that race/ethnicity and education level were associated with inclusion in the SCI or non-SCI group. Inspection of table 1 suggests that a higher proportion of individuals with SCI identified as African American (56% vs 6%) and a higher proportion in the non-SCI group identified as Hispanic or White (80% vs 38%). Though the proportion of African Americans is higher than the reported range from national statistics (4.2%-39.8%), the demographic supports an increasing trend (14.2% in 1972-1979 to 24.5% in 2015-2019).[2] A higher proportion in the non-SCI group reported education beyond high school (83% vs 26%). Although not statistically significant, fewer individuals with SCI completed the follow-up testing (70% vs 86%, P=.06).

Across the entire sample (N=69 individuals, N=138 arms), at baseline, 39% of dominant arms and 46% of nondominant arms had a CSA≥10.1 mm^2 (table 2). There were no differences in participant characteristics between arms with a baseline CSA below/above 10 mm^2 (all $P \geq .153$).

Baseline comparison

At baseline, individuals with SCI had hypoechoic MNs (ie, greater GS) in the nondominant arm ($P \leq .036$, table 3).

Table 1 Differences in participant characteristics between persons with SCI and without SCI

Variable	N	SCI	Non-SCI	*P* Value
N with baseline ultrasound	69	34	35	—
N with 1-y ultrasound	55	24	31	.063
Right/left/unknown dominant	62/7/4	29/3/2	29/4/2	1.00
Paraplegia/tetraplegia	—	28/3	—	—
Motor complete/incomplete	—	22/9	—	—
Sex (M/F)	60/5	30/2	30/3	.667
Days since injury Median (25th-75th percentile)	—	39 (28-52)	—	—
Days between visits Median (25th-75th percentile)	—	376 (365-435)	366 (355-394)	.082
Age Median (25th-75th percentile)	—	28 (23-35)	28 (23-33)	.966
BMI (kg/m^2) Median (25th-75th percentile)	—	26.1 (24.9-29.8)	25.7 (24.1-27.0)	.111
Weight (kg) Median (25th-75th percentile)	—	80.4 (73.7-91.5)	75.9 (67.5-82.3)	.029
Race/ethnicity	—			
Hispanic	20	8	12	<.001
White	23	6	17	
African American	23	20	3	
Asian/Pacific Islander	2	0	2	
Other	1	0	1	
Education				
Less than high school	10	10	0	<.001
High school/GED	21	15	6	
Associate's	8	3	5	
Bachelor's	14	3	11	
Master's	7	1	6	
Doctorate	7	0	7	
Other	2	2	0	

No other SCI/non-SCI differences were observed in either arm at baseline ($P \geq .061$, table 3). There were no baseline differences between CSA groups (all $P \geq .052$, table 4).

One-year follow-up comparison

In both arms, individuals with SCI had a greater PE score at 1 year ($P \leq .036$, table 3). No other SCI/non-SCI differences at 1-

Table 2 Differences in participant characteristics between arms with baseline CSA below/above 10 mm^2

		Dominant			Nondominant		
	N Arms (Total)	<10.0 mm^2	≥10.1 mm^2	*P* Value	<10.0 mm^2	≥10.1 mm^2	*P* Value
N arms (total)	138	42	27	—	37	32	—
N with baseline ultrasound	138	42	27	—	37	32	—
N with 1-y ultrasound	110	32	23	—	29	26	—
N non-SCI/SCI	70/68	20/22	15/12	.346	17/20	18/14	.270
N paraplegia/tetraplegia	62/6	21/1	10/2	.279	19/1	12/2	.365
N motor complete/incomplete	48/18	16/5	8/4	.421	15/5	9/4	.509
N sex (M/F)	122/16	36/6	25/2	.384	32/5	29/3	.592
Days between visits Median (25th-75th percentile)	—	371 (357-420)	375 (364-392)	.932	376 (357-416)	368 (358-391)	.413
Age Median (25th-75th percentile)	—	27 (23-35)	29 (26-36)	.153	27 (23-34)	28 (25-36)	.191
BMI (kg/m^2) Median (25th-75th percentile)	—	25.7 (24.9-28.6)	25.0 (22.9-28.0)	.273	25.8 (24.1-28.3)	25.7 (24.3-28.1)	.636
Weight (kg) Median (25th-75th percentile)	—	79.8 (74.8-89.8)	78.5 (68.0-85.2)	.314	79.4 (71.7-87.1)	178 (80.7-88.9)	.583

NOTE. Except for paraplegia/tetraplegia and motor complete/incomplete, all variables are based on the entire sample (N=69 participants, N=138 arms).

Table 3 Baseline, follow-up, and change values for individuals with and without SCI

		Dominant			Nondominant		
		SCI	Non-SCI	*P* Value	SCI	Non-SCI	*P* Value
CSA (mm^2)	Baseline	8.9 (7.4-11.0)	9.8 (7.8-12.0)	.319	9.2 (8.2-10.8)	10.5 (7.9-13.4)	.319
	1-y follow-up	10.6 (7.9-12.0)	9.7 (7.9-13.1)	.773	11.1 (9.3-14.2)	11.0 (9.1-12.4)	.766
	1-y change	0.46 (−0.84 to 2.25)	0.66 (−0.42 to 2.95)	.939	1.16 (−0.28 to 5.00)	0.94 (−0.38 to 1.82)	.118
GS	Baseline	10.0 (7.4-15.0)	7.9 (5.9-13.3)	.017	10.6 (8.7-14.4)	7.9 (5.9-13.3)	.006
	1-y follow-up	9.8 (7.7-13.1)	9.2 (6.4-12.6)	.351	9.9 (7.7-13.1)	8.8 (5.7-10.4)	.118
	1-y change	0.40 (−1.86 to 1.91)	0.33 (−2.2 to 2.6)	.703	−1.2 (−6.8 to 2.1)	0.63 (−3.5 to 1.85)	.401
PE (count)	Baseline	1 (0-3)	0 (0-1)	.072	1 (0-2.5)	0 (0-1)	.086
	1-y follow-up	1 (0-4)	0 (0-1)	.006	3 (1-5.25)	0 (0-1.25)	<.001
	1-y change	0 (−1 to 2)	0 (−1 to 0)	.460	1 (−0.25 to 3.5)	0 (−1 to 0)	.017

NOTE. All data are median (25th-75th percentile).

year follow-up were observed in either arm ($P \geq .265$, table 3). When comparing persons with baseline CSA above/below 10 mm^2, follow-up CSA remained larger in both arms in persons with baseline CSA$\geq$10.0 mm^2 (both $P \leq .03$, table 4).

One-year follow-up change comparisons

No SCI/non-SCI differences in change scores were observed in either arm ($P \geq .056$, table 3). Persons with CSA<10 mm^2 at baseline had greater CSA change scores in the nondominant arm only ($P = .029$, table 4).

Associations between baseline descriptives and baseline US parameters and PE scores

Weight was related to baseline GS in both arms (dominant $P = .009$; nondominant $P = .004$, table 4) and baseline PE score in the nondominant arm only ($P = .029$, table 5). For individuals with SCI, days since injury was related to baseline GS ($P = .001$) and PE score ($P = .047$) in the nondominant arm only (table 5). No other associations among baseline descriptives, baseline US parameters, or baseline PE score were observed in either arm (all $P \geq .088$, table 5).

Associations between baseline descriptives and US parameter and PE change scores

Change in CSA, GS, and PE score were unrelated to baseline personal characteristics in both arms (all $P \geq .106$, table 6).

Associations among baseline scores and change scores for US parameters and PE score

Baseline CSA, GS, and PE score were each inversely associated with their respective change values in the nondominant arm ($P = .008$, $P < .001$, $P = .045$, respectively, table 6). Baseline GS and PE score were also inversely associated with their respective change scores in the dominant arm ($P < .001$, $P = .001$, respectively, table 6).

Discussion

We evaluated MN US characteristics and CTS symptomology of individuals with SCI at discharge from initial rehabilitation and 1 year later and in an age-matched non-SCI group. Individuals with SCI had darker MN at baseline and had greater CTS symptoms at follow-up compared to the non-SCI group with MN baseline CSA$\leq$10 mm^2. In contrast, those with baseline CSA$\leq$10 mm^2 experienced an increase in CSA over 1 year compared to no change among those with CSA>10 mm^2. For sonographic variables and symptomology, change over 1 year was related to baseline levels and not related to personal characteristics. In general, significant associations and differences between groups were more frequent in the nondominant arm.

In our sample, individuals with SCI identified as African American at a higher proportion than individuals in the non-SCI group (56% vs 6%) and higher than proportions reported from the National Spinal Cord Injury Statistical Center (4.2%-39.8%).[29] SCI participants also weighed more ($P = .03$) than non-SCI participants. Greater weight correlates with increased nerve size, and in individuals with SCI, higher weight has been shown to increase the risk of developing CTS because of greater strain during transfer and wheelchair propulsion.[30,31] The average age of our participants was 28, well below the peak age for CTS incidence (40-60).[32] The age of our participants may affect how our results compare to other studies, where wheelchair users' mean age is often approximately 40 years.[3,33]

One of the differences we observed at baseline (but not at follow-up) was that individuals with SCI had a greater GS (hypoechoic) in the nondominant arm when compared to age-matched non-SCI participants. The greater GS is possibly an acute response suggestive of increased MN edema in individuals with SCI undergoing acute rehabilitation training. Compression of the carpal tunnel arises during wheelchair transfer training, because extreme wrist angles cause decreases vascular flow and venous congestion, resulting in edema.[5,18] These changes are expected to persist at follow-up, given increased wheelchair usage and transfers; however, at 1-year follow-up comparison, more hypoechoic MNs (ie, greater GS) were no longer present. Over time, decreased activity level compared to acute rehabilitation and better transfer technique may attenuate the greater GS level seen at discharge from rehabilitation.[5]

At baseline, CSA in both arms was unrelated to personal characteristics such as height, age, BMI, or weight ($P \geq .160$). Similarly, Impink et al evaluated wheelchair athletes after sporting events and found no relationship with respect to

Table 4 Baseline, follow-up, and change values for baseline CSI above/below 10 mm^2

		Dominant			Nondominant		
		Baseline CSA<10.0 mm^2	Baseline CSA≥10.1 mm^2	*P* Value	Baseline CSA<10.0 mm^2	Baseline CSA≥10.1 mm^2	*P* Value
CSA (mm^2)	Baseline	8.0 (7.3-9.2)	11.6 (11.0-13.1)	<.001	8.3 (7.5-9.1)	12.1 (10.9-14.5)	<.001
	1-y follow-up	8.5 (7.6-10.9)	12.8 (10.3-16.1)	<.001	10.5 (8.7-11.5)	12.3 (10.0-13.8)	.007
	1-y change	0.86 (−0.18 to 2.10)	−0.12 (−1.77 to 3.23)	.533	1.82 (0.29-4.0)	0.77 (−2.31 to 1.46)	.004
GS	Baseline	9.1 (5.8-14.3)	8.6 (6.8-13.2)	.754	10.7 (7.6-13.9)	8.8 (6.9-11.8)	.224
	1-y follow-up	9.1 (6.7-13.0)	9.8 (7.6-11.9)	.695	8.9 (6.6-12.7)	8.9 (7.2-11.1)	.711
	1-y change	−0.85 (−5.03 to 1.49)	1.25 (−2.86 to 2.59)	.844	−0.85 (−5.0 to 1.49)	0.84 (−2.27 to 2.29)	.191
PE (count)	Baseline	0 (0-1)	1 (0-4)	.013	1 (0-2)	1 (0-1)	.429
	1-y follow-up	0 (0-1)	1 (0-2)	.807	1 (0-3)	1 (0-2.5)	.469
	1-y change	0 (−0.5 to 1)	−0.5 (−1 to 0)	.104	0.5 (−0.75 to 2)	0 (−1 to 0)	.184

NOTE. All data are median (25th-75th percentile).

baseline characteristics and MN CSA changes.[33] We did observe that baseline GS in both arms ($P \leq .009$) was related to weight in both groups. Additionally, baseline PE score was related to weight for the nondominant arm only ($P \geq .014$, table 4). Greater weight has been found to be associated with MN dysfunction and increases the risk of CTS in patients with paraplegia.[3,30,31] Individuals with paraplegia with increased body weight may have greater nerve CSA at the pisiform as a result of higher force exerted on the MN during transfer or wheelchair propulsion.[5,30] In the general population, individuals with overweight have a 1.5-fold increase risk of developing CTS.[34] The lack of correlation in our study with other baseline characteristics may be because of the small sample size and the young age of our participants. Longer follow-up in our study population may have elicited greater MN changes and stronger associations with baseline variables because duration of SCI may increase the incidence of CTS and severity.[9,35,36]

At 1-year follow-up, individuals (SCI and non-SCI) with baseline CSA<10 mm^2 had a statistically significant increase in CSA in the nondominant hand from 8.3 mm^2 (range, 7.5-9.1) to 10.5 mm^2 (range, 8.7-11.5; P=.004). However, there were no statistically significant CSA changes in individuals with baseline CSA≥10 mm^2 in either hand. There were no differences in CTS symptomology change (both $P \geq .104$) between CSA groups in either arm. In contrast to our findings, Betancourt et al[37] observed CSA changes to acute stress in larger nerves (>10 mm^2) and no changes in smaller nerves (<10 mm^2) MN, with pre-exercise CSA>10 mm^2 becoming smaller after exercise. The difference in results may be due to study design, because we examined the MN pre-post with 1-year difference and Betancourt et al[37] examined the MN pre-post a single exercise bout in a 2-hour period. The MN response to stress may depend on MN prestress size and the duration and magnitude of the stress. These results and those of Betancourt et al[37] suggest that the response of the MN to chronic and acute loading may be related to preloading size, with larger nerves (>10.0 mm^2) less responsive to chronic stress because they have already permanently enlarged in response to prior stress. Our observations are supported by Kim et al, who found an increased in MN CSA after exercise in wheelchair basketball athletes as a result of acute loading.[10] In individuals without SCI, the larger CSA change may be the result of repetitive forceful movements or increased usage to protect the dominant hand or to balance out the dominant hand in activities.[38]

We observed more CTS symptoms for individuals with SCI for both dominant and nondominant arms at 1-year follow-up. In the general population, CTS commonly involves the dominant hand.[39] Our findings contrast with other studies that demonstrated that MN changes commonly occur bilaterally or just in the dominant hand for individuals with SCI.[3,4] The nondominant hand may be more susceptible to injury in individuals with SCI 1 year post injury due to increased propulsion and transfers.[37] This increased recruitment results in greater stress of the MN on the nondominant side.[40,41]

Study limitations

There were several key limitations. The small sample size (N=69 total, N=34 age-matched pairs) increases type II error risk. However, our study enables planning for a sample size

Table 5 Factors associated with baseline US and CTS symptoms

	CSA		GS		PE	
	Dom	Nondominant	Dominant	Nondominant	Dominant	Nondominant
Age	0.216	0.152	−0.073	0.037	−0.043	0.009
Sex*	−0.078	−0.025	−0.038	−0.060	−0.164	−0.174
Height	−0.043	0.071	0.222	0.114	0.016	0.193
Weight	−0.160	0.109	0.333‡	0.364‡	0.112	0.354†
BMI	−0.147	0.013	0.135	0.154	0.091	0.211
Days since injury	−0.233	−0.246	−0.318	0.545‡	0.404‡	0.473†

NOTE. Except for days since injury, all correlations are based on the entire sample (non-SCI+SCI).
* Indicates point biserial correlation. All other correlations are Spearman rho.
† $P<.05$.
‡ $P<.01$.

Table 6 Factors associated with baseline to 1-year follow-up change in US and CTS symptoms

	Δ CSA		Δ GS		Δ PE	
	Dom	Nondominant	Dom	Nondominant	Dom	Nondominant
Age	−0.042	0.107	0.159	0.049	0.179	0.253
Sex*	−0.123	−0.146	−0.085	−0.197	0.029	0.267
Height	−0.080	−0.096	0.098	0.228	−0.124	−0.032
Weight	−0.039	0.066	−0.123	−0.064	−0.021	0.067
BMI	−0.039	0.136	−0.210	−0.127	0.036	0.032
Days since injury	−0.135	−0.054	−0.312	−0.295	−0.051	−0.103
Days between visits	−0.010	−0.078	−0.093	−0.064	0.196	0.067
Baseline CSA	−0.098	−0.404‡	−0.044	0.100	−0.061	−0.106
Baseline GS	−0.148	0.120	−0.600§	−0.574§	−0.019	0.059
Baseline PE	−0.209	−0.143	−0.022	0.194	−0.513‡	−0.310†

NOTE. Except for days since injury, all correlations are based on the entire sample (non-SCI+SCI).
* Indicates point biserial correlation. All other correlations are Spearman rho.
† $P<.05$.
‡ $P<.01$.
§ $P<.001$. Exact P values are provided in the text for all statistically significant correlations.

large enough to detect a statistical difference. Another limitation is that our sample's average age (28 years) is much younger than the average age of CTS onset (40-60 years).[3,37] Time since injury is also an important factor to consider because duration of upper extremity use is associated with CTS.[9] Future studies should follow individuals longitudinally for a greater period of time to define the natural history of MN pathology in individuals with SCI. Lastly, although investigators who traced the MNs were blinded to assessment time, participant ID, and participant SCI/non-SCI classification and traced the images in a randomized order, learning effects, fatigue effects, and boundary identification errors may have occurred. Other studies had radiologists identify and trace the MN and did not use blinded persons for the tracing of the nerves.[42]

Conclusions

We found few MN ultrasound characteristics and PE differences between individuals without SCI and newly injured individuals with SCI during the first year postinjury. Observed differences at baseline and in change scores were primarily identified in the nondominant arm and appear to be related to MN anatomy at injury. Furthermore, individuals with SCI had greater symptomology at 1 year, which warrants closer monitoring at follow-up. Our study provides a unique analysis with an age-matched group of individuals without SCI and individuals with SCI. Future studies with a longer follow-up period and increased study population age may yield greater differences.

Suppliers

a. Minifocus 1402a with a 5- to 12-MHz 50-mm linear transducer; BK Medical.
b. MATLAB; The MathWorks, Inc.

Corresponding author

Minh Quan Le, MD, Jackson Memorial Hospital, 10140 Ramblewood Dr, Coral Springs, FL 33071. *E-mail address:* Quanle0105@gmail.com.

References

1. Lasfargues JE, Custis D, Morrone F, Carswell J, Nguyen T. A model for estimating spinal cord injury prevalence in the United States. Paraplegia 1995;33:62–8.
2. National Spinal Cord Injury Statistical Center. Spinal cord injury facts and figures at a glance; 2020. Available at: https://www.nscisc.uab.edu/Public/Facts%20and%20Figures%202020.pdf. Accessed November 16, 2022.
3. Yang J, Boninger ML, Leath JD, Fitzgerald SG, Dyson-Hudson TA, Chang MW. Carpal tunnel syndrome in manual wheelchair users with spinal cord injury: a cross-sectional multicenter study. Am J Phys Med Rehabil 2009;88:1007–16.
4. Asheghan M, Hollisaz MT, Taheri T, Kazemi H, Aghda AK. The prevalence of carpal tunnel syndrome among long-term manual wheelchair users with spinal cord injury: a cross-sectional study. J Spinal Cord Med 2016;39:265–71.
5. Hogaboom NS, Diehl JA, Oyster ML, Koontz AM, Boninger ML. Ultrasonographic median nerve changes after repeated wheelchair transfers in persons with paraplegia: relationship with subject characteristics and transfer skills. PM R 2016;8:305–13.
6. Dalyan M, Cardenas DD, Gerard B. Upper extremity pain after spinal cord injury. Spinal Cord 1999;37:191–5.
7. Akbar M, Penzkofer S, Weber MA, Bruckner T, Winterstein M, Jung M. Prevalence of carpal tunnel syndrome and wrist osteoarthritis in long-term paraplegic patients compared with controls. J Hand Surg Eur Vol 2014;39:132–8.
8. Goodman CM, Steadman AK, Meade RA, Bodenheimer C, Thornby J, Netscher DT. Comparison of carpal canal pressure in paraplegic and nonparaplegic subjects: clinical implications. Plast Reconstr Surg 2001;107:1464–71; discussion 1472.
9. Aljure J, Eltorai I, Bradley WE, Lin JE, Johnson B. Carpal tunnel syndrome in paraplegic patients. Paraplegia 1985;23:182–6.
10. Kim DK, Kim BS, Kim MJ, Kim KH, Park BK, Kim DH. Electrophysiologic and ultrasonographic assessment of carpal tunnel syndrome in wheelchair basketball athletes. Ann Rehabil Med 2017;41:58–65.
11. Impink BG, Collinger JL, Boninger ML. The effect of symptoms of carpal tunnel syndrome on ultrasonographic median nerve measures before and after wheelchair propulsion. PM R 2011;3:803–10.
12. Vahed LK, Arianpur A, Gharedaghi M, Rezaei H. Ultrasound as a diagnostic tool in the investigation of patients with carpal tunnel syndrome. Eur J Transl Myol 2018;28:7380.
13. Wipperman J, Goerl K. Carpal tunnel syndrome: diagnosis and management. Am Fam Physician 2016;94:993–9.
14. Burnham RS, Steadward RD. Upper extremity peripheral nerve entrapments among wheelchair athletes: prevalence, location, and risk factors. Arch Phys Med Rehabil 1994;75:519–24.
15. Davidoff G, Werner R, Waring W. Compressive mononeuropathies of the upper extremity in chronic paraplegia. Paraplegia 1991;29:17–24.
16. Tun CG, Upton J. The paraplegic hand: electrodiagnostic studies and clinical findings. J Hand Surg Am 1988;13:716–9.
17. Atroshi I, Gummesson C, Johnsson R, Ornstein E, Ranstam J, Rosén I. Prevalence of carpal tunnel syndrome in a general population. JAMA 1999;282:153–8.
18. Aboonq MS. Pathophysiology of carpal tunnel syndrome. Neurosciences (Riyadh) 2015;20:4–9.
19. Nathan PA, Keniston RC, Lockwood RS, Meadows KD. Tobacco, caffeine, alcohol, and carpal tunnel syndrome in American industry. A cross-sectional study of 1464 workers. J Occup Environ Med 1996;38:290–8.
20. Gorsche RG, Wiley JP, Renger RF, Brant RF, Gemer TY, Sasyniuk TM. Prevalence and incidence of carpal tunnel syndrome in a meat packing plant. Occup Environ Med 1999;56:417–22.
21. Impink BG, Gagnon D, Collinger JL, Boninger ML. Repeatability of ultrasonographic median nerve measures. Muscle Nerve 2010;41:767–73.
22. Chen YT, Williams L, Zak MJ, Fredericson M. Review of ultrasonography in the diagnosis of carpal tunnel syndrome and a proposed scanning protocol. J Ultrasound Med 2016;35:2311–24.
23. Practice parameter for electrodiagnostic studies in carpal tunnel syndrome: summary statement. Muscle Nerve 2002;25:918–22.
24. Visser LH, Smidt MH, Lee ML. High-resolution sonography versus EMG in the diagnosis of carpal tunnel syndrome. J Neurol Neurosurg Psychiatry 2008;79:63–7.
25. Rahmani M, Ghasemi Esfe AR, Vaziri-Bozorg SM, Mazloumi M, Khalilzadeh O, Kahnouji H. The ultrasonographic correlates of carpal tunnel syndrome in patients with normal electrodiagnostic tests. Radiol Med 2011;116:489–96.
26. Cartwright MS, Hobson-Webb LD, Boon AJ, et al. Evidence-based guideline: neuromuscular ultrasound for the diagnosis of carpal tunnel syndrome. Muscle Nerve 2012;46:287–93.
27. Atroshi I, Gummesson C, Johnsson R, Ornstein E, Ranstam J, Rosén I. [Prevalence for clinically proved carpal tunnel syndrome is 4 percent]. Lakartidningen 2000;97:1668–70.
28. Boca SM, Leek JT. A direct approach to estimating false discovery rates conditional on covariates. PeerJ 2018;6:e6035.
29. The 2020 annual statistical report complete public version for the Spinal Cord Injury Model Systems. University of Alabama. Available at: https://www.nscisc.uab.edu/Public/Facts%20and%20Figures%202020.pdf. Published 2020. Accessed November 16, 2020.
30. Boninger ML, Cooper RA, Baldwin MA, Shimada SD, Koontz A. Wheelchair pushrim kinetics: body weight and median nerve function. Arch Phys Med Rehabil 1999;80:910–5.
31. Cartwright MS, Shin HW, Passmore LV, Walker FO. Ultrasonographic reference values for assessing the normal median nerve in adults. J Neuroimaging 2009;19:47–51.
32. Petrover D, Richette P. Treatment of carpal tunnel syndrome: from ultrasonography to ultrasound guided carpal tunnel release. Joint Bone Spine 2018;85:545–52.
33. Impink BG, Boninger ML, Walker H, Collinger JL, Niyonkuru C. Ultrasonographic median nerve changes after a wheelchair sporting event. Arch Phys Med Rehabil 2009;90:1489–94.
34. Shiri R, Pourmemari MH, Falah-Hassani K, Viikari-Juntura E. The effect of excess body mass on the risk of carpal tunnel syndrome: a meta-analysis of 58 studies. Obes Rev 2015;16:1094–104.
35. Gellman H, Sie I, Waters RL. Late complications of the weight-bearing upper extremity in the paraplegic patient. Clin Orthop Relat Res 1988 Aug: 132–5.
36. Vives Alvarado JR, Felix ER, Gater Jr. DR. Upper extremity overuse injuries and obesity after spinal cord injury. Top Spinal Cord Inj Rehabil 2021;27:68–74.
37. Betancourt L, Cowan RE, Chang A, Irwin R. Case-control study of ultrasound evaluation of acute median nerve response to upper extremity circuit training in spinal cord injury. Arch Phys Med Rehabil 2020;101:1898–905.
38. Zambelis T, Tsivgoulis G, Karandreas N. Carpal tunnel syndrome: associations between risk factors and laterality. Eur Neurol 2010;63:43–7.
39. Reinstein L. Hand dominance in carpal tunnel syndrome. Arch Phys Med Rehabil 1981;62:202–3.
40. Falck B, Aarnio P. Left-sided carpal tunnel syndrome in butchers. Scand J Work Environ Health 1983;9:291–7.
41. Ferrante MA. The relationship between sustained gripping and the development of carpal tunnel syndrome. Fed Pract 2016;33:10–5.
42. Mallouhi A, Pülzl P, Trieb T, Piza H, Bodner G. Predictors of carpal tunnel syndrome: accuracy of gray-scale and color Doppler sonography. AJR Am J Roentgenol 2006;186:1240–5.