

Abnormal Somatosensory Temporal Discrimination Is Not Related to Impaired Fine Motor Skills in Carpal Tunnel Syndrome

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Cite this article as: Aliş C, Ayas S, Zileli I, Uzun Adatepe N, Karaali Savrun F, Gündüz A. Abnormal somatosensory temporal discrimination is not related to impaired fine motor skills in carpal tunnel syndrome. *Cerrahpaşa Med J.* 2023;47(1):72-76.

Abstract

Objective: Carpal tunnel syndrome has been shown to cause hand clumsiness, impairment in pinch grip force, and fine motor skills. We hypothesized that impaired fine motor skills in carpal tunnel syndrome may be related to maladaptive central somatosensory changes. This study aimed to investigate the relationship between the changes in central sensory pathways and fine motor activities in patients with carpal tunnel syndrome by examining the somatosensory temporal discrimination threshold.

Methods: This study included 37 patients with idiopathic carpal tunnel syndrome and 19 healthy subjects. After recording routine nerve conduction studies, sensory threshold and somatosensory temporal discrimination threshold on volar faces of dominant second (2f) and fifth fingers (5f) were determined. All patients performed 9-hole peg test. We also questioned the presence of dropping objects.

Results: Object dropping was more frequent in patients with idiopathic carpal tunnel syndrome in comparison to healthy subjects ($P = .000$). Duration to complete 9-hole peg test was also higher in patients with dropping objects. Although sensory threshold-2f and somatosensory temporal discrimination threshold-2f were higher in patients with carpal tunnel syndrome compared to healthy subjects, somatosensory temporal discrimination threshold-2f was not related to dropping objects. The duration of 9-hole peg test did not correlate with somatosensory temporal discrimination threshold-2f.

Conclusion: We showed that there was a maladaptive reorganization process in primary somatosensory cortex of 2f represented by somatosensory temporal discrimination threshold in patients with carpal tunnel syndrome. Fine motor skills may be due to the disruption of superficial touch sensation or motor dysfunction secondary to carpal tunnel syndrome, but it is not related to maladaptive changes in somatosensory cortex-basal ganglia network represented by somatosensory temporal discrimination threshold.

Keywords: Fine motor skills, somatosensory temporal discrimination threshold, carpal tunnel syndrome

Introduction

Carpal tunnel syndrome (CTS) is a peripheral nervous system disease that causes pain, paresthesia, and weakness involving median nerve innervated territory distal to the wrist. However, involvement in CTS is not limited to the hand. Pain sometimes may involve more proximal regions. There is a diffusion of sensory symptoms toward the ulnar nerve hand territory.¹ Furthermore, the disability caused by CTS is beyond these symptoms. It leads to hand clumsiness, impairment in pinch grip force, fine motor skills, and handwriting.²⁻⁴ These deficits were not related to the severity of electrophysiological findings, and bilateral deficits in fine motor control were even found in patients with unilateral CTS.^{2,5} Impaired handwriting developed in patients with poorer sensation with excessive force exertion of the digits and pen tip.³ Hand clumsiness in CTS was related to the severity of sensory symptoms and to the clinical-neurographic signs of motor but not to the sensory nerve damage.⁶ These previous findings suggested that

impaired fine motor skills in CTS could have been related to central somatosensory changes. A recent study indicated inefficient object manipulation by CTS patients which may help to explain why CTS patients tend to drop objects. Additionally, it was shown that there is an alteration in the central somatosensory pathways in CTS patients. This can be seen by changes in neural activity at multiple locations, such as the somatosensory system in the cortex, brainstem, or spinal cord.⁷ Furthermore, an abnormal change in the contralateral primary somatosensory cortex (S1) has been linked to worse symptoms, decreased fine motor skills, and poor accuracy in detecting sensations in the median innervated digits.⁸

Therefore, this study aimed to investigate the relationship between the changes in the central sensory pathways and fine motor skills in patients with CTS. To evaluate central sensory pathways, we used somatosensory temporal discrimination threshold (STDT), which is the shortest time interval required to detect a pair of tactile stimuli separately. It is an electrophysiological response indicating temporal discrimination and the major area mediating STDT is S1.

Material and Method

We included all consecutive patients who presented with complaints such as numbness, tingling, pain, and loss of strength in the first 3 fingers of the hand between October 2020 and

Received: October 5, 2022 Accepted: November 1, 2022

Publication Date: March 23, 2023

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DOI: 10.5152/cjm.2023.21089



March 2021 and in whom diagnosis of CTS was confirmed by routine electrophysiological tests according to the standardized methods.⁹

Patients were grouped as mild, moderate, and severe CTS according to the severity of carpal tunnel syndrome. The patients who had prolonged median sensory peak latency were classified in "mild CTS" group, the patients who had prolonged median sensory peak latency with low amplitude or prolonged median motor distal latency were classified in "moderate CTS" group, and the patients who had no sensory response or motor response with reduced amplitude were classified in "severe CTS" group.¹⁰ A control group constituted 19 healthy subjects who had similar age and sex.

Exclusion criteria were the presence of polyneuropathy, tremor, ulnar neuropathy in the same extremity, other systemic or neurological disorders, painful conditions, history of orthopedic operation, and pregnancy.

The study was approved by the ethical committee of İstanbul University-Cerrahpaşa (Date: October 6, 2020, approval number is 83045809-604.01.02). All participants provided informed consent.

Clinical Evaluations

Following routine neurological and electrophysiological examinations, the presence of dropping objects was questioned. Furthermore, symptoms were grouped as sensory symptoms such as paresthesia and hypoesthesia and pain and symptoms not in hand (such as arm pain). All participants performed 9-hole peg test (NHPT) according to the previous reports.^{11,12} The duration for placement (NHPT I) and total completion (placement+assembly, NHPT II) were recorded in each participant.

Electrophysiological Investigations

Sensory threshold (ST) and somatosensory temporal discrimination threshold (STDT) were determined. All electrophysiological examinations were performed using a Neuropack Sigma MEB-5504k (Nihon Kohden Medical, Tokyo, Japan). Silver-silver chloride surface recording electrodes were used. When determining the ST, the single electrical stimulus was given to the volar faces of the dominant hand second finger (2f) and the fifth finger (5f) at an intensity of 1 mA which was gradually increased by 0.2 mA. Then, the stimulus at the intensity of ST was given in pairs. The interval between the 2 stimuli was gradually increased from the 10 ms interval till the interval at which subjects discriminated the 2 different stimuli for the first time which was defined as STDT.¹³

Data and Statistical Analysis

First, we determined the frequency of dropping objects in each group. We compared NHPT I and II, ST-2f, ST-5f, STDT-2f, STDT-5f between patients dropping objects, patients not dropping objects, and healthy subjects. Kruskal-Wallis test was used and post hoc comparisons were done by Mann-Whitney *U* test. We also performed a multifactorial analysis using variables, dropping objects, and symptoms of CTS.

Frequency of dropping objects, NHPT I and II, ST-2f, ST-5f, STDT-2f, STDT-5f was also compared between groups according to the severity (mild, moderate, and severe). Kruskal-Wallis test was used and post hoc comparisons were done by Mann-Whitney *U* test.

Correlation analysis was performed to evaluate correlation between sensory latency, motor latency, NHPT I and II, ST-2f, ST-5f, STDT-2f, STDT-5f using Spearman's correlation test.

Statistical analysis was done using Statistical Package for Social Sciences (IBM SPSS Corp., Armonk, NY, USA) 20.0. *P*-value <.05 was deemed significant.

Results

In the study period, we identified 37 patients with idiopathic CTS who fulfilled the inclusion criteria and there were 19 healthy subjects of similar age and sex (Table 1). Thirty-nine patients with CTS were excluded due to accompanying disorders. There were

Table 1. Demographic and Clinical Findings in Patients with Carpal Tunnel Syndrome and Healthy Subjects

	Patients with CTS, n = 37	Healthy Subjects, n = 19	<i>P</i>
Age, mean ± SD, year	49.7 ± 10.4	45.9 ± 9.9	.195
Sex, M/F, n	5/32	5/14	.205
Dropping objects, n (%)	16 (43.2)	0	.000
Pain, n (%)	5 (13.5)	-	-
Numbness, n (%)	1 (2.7)	-	-
Paresthesia, n (%)	29 (78.4)	-	-

CTS, carpal tunnel syndrome; F, female; M, male; SD, standard deviation.

Table 2. Clinical and Electrophysiological Findings in Patient Groups and Healthy Subjects

	Patients Dropping Objects, n = 16	Patients Not Dropping Objects, n = 21	Healthy Subjects, n = 19	<i>P</i>
Age, mean ± SD, year	51.8 ± 10.8	48.0 ± 9.9	45.9 ± 9.9	.221
Sex, M/F, n	1/15	4/17	5/14	.299
NHPT-I	15.0 ± 2.0	13.0 ± 1.8	13.8 ± 2.3	.021*
NHPT-II	22.0 ± 2.1	19.6 ± 2.7	20.6 ± 3.2	.048*
ST-2f, mA	4.8 ± 2.1	4.2 ± 0.9	3.3 ± 1.0	.028**
ST-5f, mA	2.7 ± 1.3	2.7 ± 1.0	2.7 ± 0.8	.978
STDT-2f, ms	139.4 ± 84.4	133.8 ± 58.2	64.7 ± 52.3	.001***
STDT-5f, ms	103.7 ± 56.8	108.6 ± 54.1	75.7 ± 60.2	.203

P* < .005 (Kruskal-Wallis) post hoc analysis showed NHPT-I and II were significantly longer in patients dropping objects compared to patients who do not drop (*P* = .002 and *P* = .008, respectively, Mann-Whitney *U*); *P* < .005 (Kruskal-Wallis) post hoc analysis showed ST was significantly higher in both groups of patients compared to healthy subjects (*P* = .012 for both comparisons, Mann-Whitney *U*); ****P* < .001 (Kruskal-Wallis) post hoc analysis showed STDT was significantly longer in both groups of patients compared to healthy subjects (*P* = .000 and *P* = .003, Mann-Whitney *U*).

2f, second finger; 5f, fifth finger; F, female; M, male; NHPT-I, duration for placement of 9-hole peg test; NHPT-II, total completion time (placement+assembly) of 9-hole peg test; SD, standard deviation; ST, sensory threshold; STDT, somatosensory temporal discrimination threshold.

Table 3. Comparisons Between Patients with Mild, Moderate, and Severe Carpal Tunnel Syndrome

	Mild CTS, n = 24	Moderate CTS, n = 7	Severe CTS, n = 6	P
Age, mean ± SD, year	48.1 ± 11.7	54.1 ± 6.8	50.5 ± 6.3	.215
Sex, M/F, n	4/20	0/7	1/5	.164
NHPT-I	13.38 ± 1.9	14.8 ± 1.9	15.9 ± 2.6	.083
NHPT-II	19.9 ± 2.5	21.5 ± 2.1	22.9 ± 2.9	.091
ST-2f, mA	3.9 ± 1	5.1 ± 1.6	5.5 ± 2.6	.009
ST-5f, mA	2.6 ± 1	2.7 ± 1.2	3.1 ± 1.3	.147
STDT-2f, ms	137.9 ± 76.5	133.3 ± 61.8	132.8 ± 58.7	.025
STDT-5f, ms	116.2 ± 59.6	97.1 ± 25.6	78.3 ± 51.9	.158

2f, second finger; 5f, fifth finger; CTS, carpal tunnel syndrome; F, female; M, male; NHPT-I, duration for placement of 9-hole peg test; NHPT-II, total completion time (placement+ assembly) of 9-hole peg test; SD, standard deviation; ST, sensory threshold; STDT, somatosensory temporal discrimination threshold.

24 patients (64.8%) in mild CTS group, 7 patients (18.9%) in moderate CTS group, and finally 6 patients (16.2%) in severe CTS group. Sensory potentials could not be evoked in 3 of 6 patients with severe CTS.

Sixteen patients with CTS had history of dropping objects. Comparisons between patients dropping objects, patients with no history of dropping objects and healthy subjects, the age and sex were still similar. However, the durations of NHPT test (NHPT-I and II) were longer in patients dropping objects compared to healthy subjects and patients not dropping objects (Table 2). Sensory threshold -2f was significantly higher and STDT-2f was significantly longer in both patient groups compared to healthy subjects. Sensory threshold-5f and STDT-5f were similar between groups (Table 2).

None of the patients had thenar muscle atrophy or weakness even in the severe CTS group. However, dropping objects was more

common in patients with severe and moderate CTS ($P = .035$). Although ST-2f was higher in patients with severe CTS compared to those with moderate and mild ones ($P = .009$), STDT-2f was longer in patients with mild and moderate CTS compared to patients with severe CTS ($P = .025$). Although not significant, NHPT I and II were longer in patients with severe CTS (Table 3).

Sensory latency positively correlated with NHPT I and II ($P = .010$ and $P = .022$, correlation coefficients: 0.434 and 0.391, respectively, Figure 1). Motor latency also positively correlated with NHPT I and II ($P = .007$ and $P = .009$, correlation coefficients: 0.810 and 0.434, respectively, Figure 2). But there was no correlation between STDT-2f and motor or sensory latency. Furthermore, there was no correlation between NHPT and STDT-2f ($P = .752$ and $P = .853$). We also found that ST-2f positively correlated with both NHPT I and NHPT II ($P = .040$ and $P = .017$, correlation coefficients: 0.339 and 0.390, respectively).

Nine-hole peg test and electrophysiological findings were similar in patients with different symptoms (presence of paresthesia, pain, or no hand symptom).

Discussion

The most important findings of our study are as follows: (i) patients with CTS had difficulty in fine motor skills; (ii) difficulty in fine motor skills in CTS was related to the severity of CTS; (iii) STDT-2f was abnormal in CTS, especially in mild ones; and (iv) abnormal STDT-2f was not related to abnormal fine motor skills in CTS.

The major area mediating STDT is assumed to be S1 because transcranial continuous theta burst stimulation (cTBS) studies showed that cTBS applied on S1 prolonged STDT, by probably decreasing GABAergic activity of inhibitory interneurons in this region.^{14,15} However, cTBS applied on association areas (S2) had no effect on STDT. The other regions mediating STDT are the supplementary area and basal ganglia since it was found abnormal in movement disorders such as dystonia or Parkinson’s disease.^{16,17} Therefore, longer STDT in our study suggested reorganization in central somatosensory network including S1 and basal ganglia in CTS. Previous studies using somatosensory evoked potentials (SEPs), magnetoencephalography, or functional magnetic resonance imaging (MRI) demonstrated maladaptive reorganization in S1. Patients with CTS had significantly reduced gray matter volume

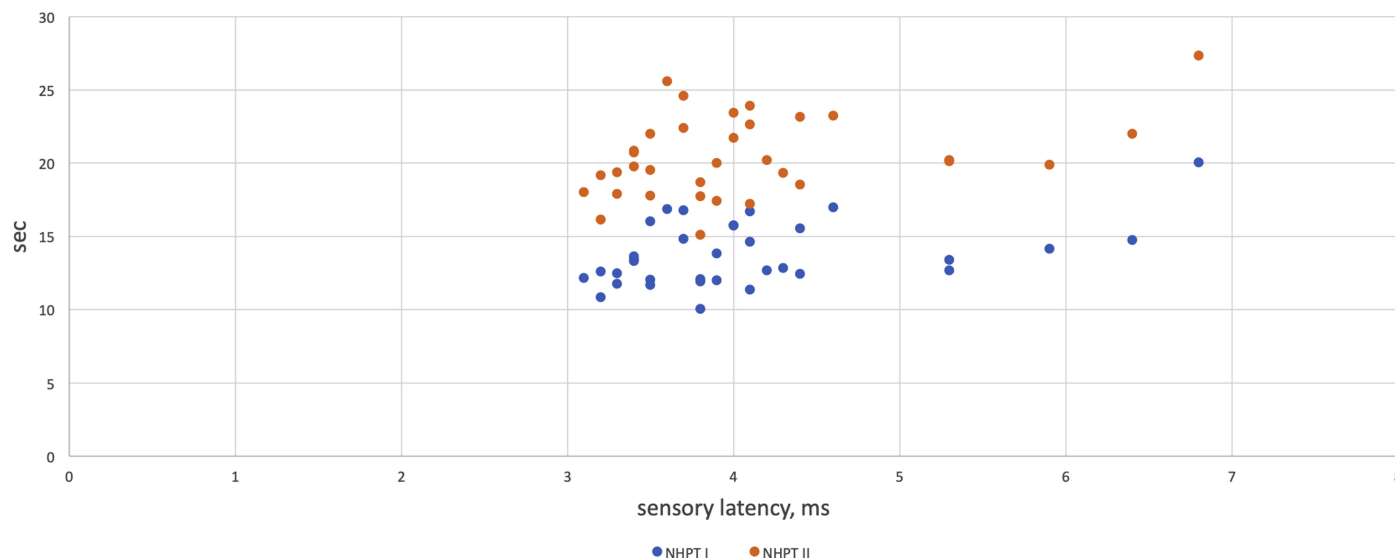


Figure 1. Graphical representation of correlation between sensory latency and 9-hole peg test (NHPT) I and II.

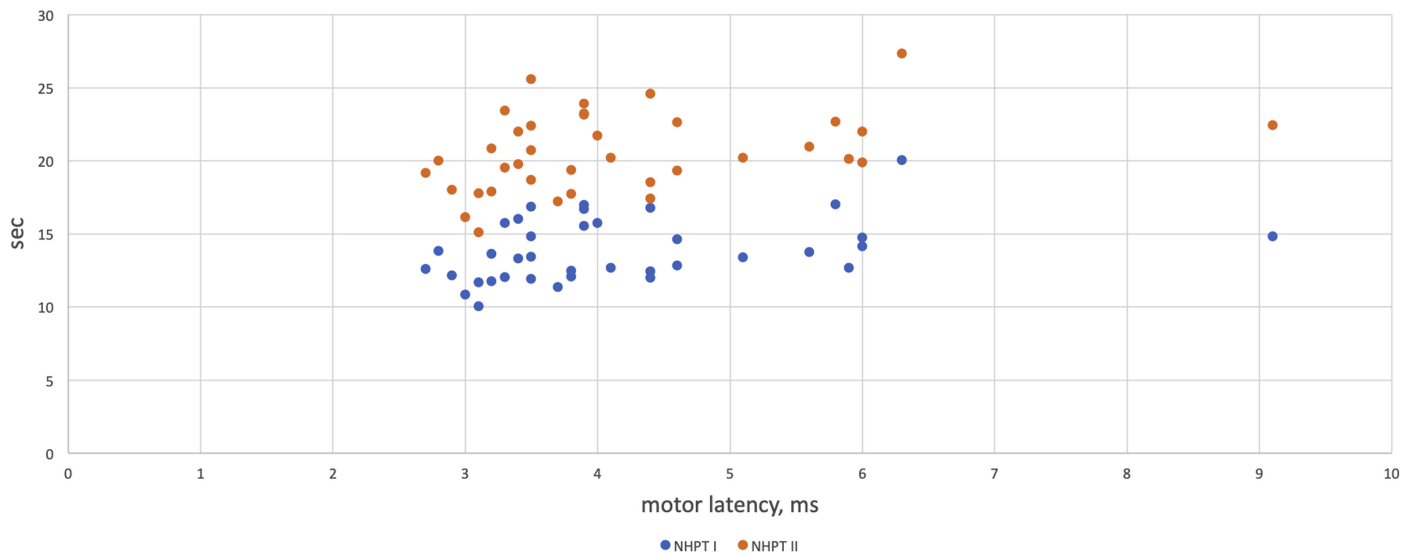


Figure 2. Graphical representation of correlation between motor latency and 9-hole peg test (NHPT) I and II.

in contralesional primary somatosensory area (S1, hand-area) and pulvinar and frontal pole^{18,19} and reduced volume in contralesional S1 correlated with median nerve conduction velocity.¹⁸ Cortical representation of the injured median nerve was reduced in size and presumably invaded by sensory zone of index finger or thumb.¹⁹ Studies using recovery function of SEPs showed shorter N20 latency and higher percentage recovery in patients with CTS suggesting a greater number of activated neurons in the S1 or afferent fibers to S1 than in that of the healthy subjects.²⁰ In addition to disinhibition created by CTS, the authors also showed destruction of somatotopic organization of S1. Functional MRI studies replicated similar results. Sensory complaints in fingers that were innervated by the median nerve caused a continuous synchronous stimulation on S1, leading to maladaptive somatosensory reorganization and, subsequently, deterioration in the 2-point discrimination and fine motor skills.⁸ Similarly, we found maladaptive changes involving second finger measurements but not ulnar nerve innervated 5f and our results revealed malfunctioning in the relevant central sensory network. Interestingly, abnormal somatosensory functioning was more pronounced in patients with mild CTS rather than in patients with severe CTS according to our results. However, the grading was done according to the electrophysiological findings but not to the clinical severity. Although we did not quantitatively analyze the symptoms, there was no difference in STDT according to the presence of different symptoms. However, we should emphasize that most patients had paresthesia. Another limitation was the inability to quantify the exact duration of symptoms. In an animal study, the cerebral reorganization was directly related to the time window and was not observed at 2-month evaluation. Therefore, abnormal STDT may be more closely related to the duration rather than severity.²¹

Fine motor skills require well-coordinated sensorimotor control of fingers. In CTS, due to axonal loss and reinnervation of median nerve, this control mechanism might disrupt and cause impairment of fine motor skills.³ Despite the presence of plastic changes in the somatosensory network or abnormal fine motor skills in patients included in this study, they did not exhibit any association between abnormal STDT and fine motor skills. In healthy subjects, there is activation of somatosensory network after passive movements.²² Sensation contributes to the precision of movements. In CTS, loss of sensation is clearly associated with hand clumsiness.

However, abnormal fine motor movements in CTS are not originated in the S1-basal ganglia network represented by STDT.

To perform movements smoothly, sensorimotor integration is required. Sensorimotor modulation contributes to monitor corticospinal activity and function in both active and surrounding muscles.²³ The best-known disorder, in which there is a dysfunctional sensorimotor integration, is dystonia. The STDT is abnormally long in patients with dystonia¹⁹ and is closely associated with movement execution. For example, it increases STDT in healthy subjects and this finding is abnormal in dystonia.¹⁸ We do not ignore the role of sensory system in movement production and association between STDT and movement. However, network represented by STDT does not have a major role in hand clumsiness in CTS.

Our results disclosed fine motor skills may be due to disruption of superficial touch sensation or motor dysfunction secondary to CTS, but it is not related to maladaptive changes in S1-basal ganglia network represented by STDT.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of İstanbul University-Cerrahpaşa (Date: October 6, 2020, number: 83045809-604.01.02).

Informed Consent: Informed consent was obtained from all participants who participated in this study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – C.A., S.A., İ.Z., A.G.; Design – C.A., S.A., İ.Z., A.G.; Supervision – A.G., N.U.A., F.K.S.; Materials – İ.Z.; Data Collection and Processing – C.A., S.A., A.G.; Analysis and Interpretation – C.A., A.G.; Literature Review – C.A., S.A., A.G.; Writing – C.A., S.A., N.U.A., F.K.S., A.G.

Declaration of Interests: The authors have no conflicts of interest to declare.

Funding: The authors declared that this study has received no financial support.

References

- Ginanneschi F, Mondelli M, Rossi A. Sensory neuropathy may cause central neuronal reorganization but does not respect perceptual

- quality or localization of sensation. *Clin J Pain*. 2012;28(8):653-657. [\[CrossRef\]](#)
2. de la Llave-Rincón AI, Fernández-de-Las-Peñas C, Pérez-de-Heredia-Torres M, Martínez-Perez A, Valenza MC, Pareja JA. Bilateral deficits in fine motor control and pinch grip force are not associated with electrodiagnostic findings in women with carpal tunnel syndrome. *Am J Phys Med Rehabil*. 2011;90(6):443-451. [\[CrossRef\]](#)
 3. Kuo LC, Hsu HM, Wu PT, Lin SC, Hsu HY, Jou IM. Impact of distal median neuropathy on handwriting performance for patients with carpal tunnel syndrome in office and administrative support occupations. *J Occup Rehabil*. 2014;24(2):332-343. [\[CrossRef\]](#)
 4. Gehrman S, Tang J, Kaufmann RA, Goitz RJ, Windolf J, Li ZM. Variability of precision pinch movements caused by carpal tunnel syndrome. *J Hand Surg Am*. 2008;33(7):1069-1075. [\[CrossRef\]](#)
 5. Fernández-de-Las-Peñas C, Pérez-de-Heredia-Torres M, Martínez-Piédrola R, de la Llave-Rincón AI, Cleland JA. Bilateral deficits in fine motor control and pinch grip force in patients with unilateral carpal tunnel syndrome. *Exp Brain Res*. 2009;194(1):29-37. [\[CrossRef\]](#)
 6. Tamburin S, Cacciatori C, Marani S, Zanette G. Pain and motor function in carpal tunnel syndrome: a clinical, neurophysiological and psychophysical study. *J Neurol*. 2008;255(11):1636-1643. [\[CrossRef\]](#)
 7. Tinazzi M, Zanette G, Volpato D, et al. Neurophysiological evidence of neuroplasticity at multiple levels of the somatosensory system in patients with carpal tunnel syndrome. *Brain*. 1998;121(9):1785-1794. [\[CrossRef\]](#)
 8. Maeda Y, Kettner N, Holden J, et al. Functional deficits in carpal tunnel syndrome reflect reorganization of primary somatosensory cortex. *Brain*. 2014;137(6):1741-1752. [\[CrossRef\]](#)
 9. Kimura J. *Electrodiagnosis in Diseases of Nerve and Muscle: Principles and Practice*. 4th ed. Oxford: Oxford University Press; 2013.
 10. Watson JC, Daube JR. Compound muscle action potentials. In: Daube JR, Rubin DI, eds. *Clinical Neurophysiology*. 3rd ed. Oxford: Oxford University Press; 2009.
 11. Niu HX, Wang RH, Xu HL, et al. Nine-Hole Peg Test and ten-meter walk test for evaluating functional loss in Chinese Charcot-Marie-Tooth disease. *Chin Med J (Engl)*. 2017;130(15):1773-1778. [\[CrossRef\]](#)
 12. Hervault M, Balto JM, Hubbard EA, Motl RW. Reliability, precision, and clinically important change of the nine-Hole Peg Test in individuals with multiple sclerosis. *Int J Rehabil Res*. 2017;40(1):91-93. [\[CrossRef\]](#)
 13. Conte A, Rocchi L, Ferrazzano G, et al. Primary somatosensory cortical plasticity and tactile temporal discrimination in focal hand dystonia. *Clin Neurophysiol*. 2014;125(3):537-543. [\[CrossRef\]](#)
 14. Rocchi L, Casula E, Tocco P, Berardelli A, Rothwell J. Somatosensory temporal discrimination threshold involves inhibitory mechanisms in the primary somatosensory area. *J Neurosci*. 2016;36(2):325-335. [\[CrossRef\]](#)
 15. Conte A, Rocchi L, Nardella A, et al. Theta-burst stimulation-induced plasticity over primary somatosensory cortex changes somatosensory temporal discrimination in healthy humans. *PLoS One*. 2012;7(3):e32979. [\[CrossRef\]](#)
 16. Conte A, Ferrazzano G, Belvisi D, et al. Somatosensory temporal discrimination in Parkinson's disease, dystonia and essential tremor: pathophysiological and clinical implications. *Clin Neurophysiol*. 2018;129(9):1849-1853. [\[CrossRef\]](#)
 17. Scontrini A, Conte A, Defazio G, et al. Somatosensory temporal discrimination in patients with primary focal dystonia. *J Neurol Neurosurg Psychiatry*. 2009;80(12):1315-1319. [\[CrossRef\]](#)
 18. Maeda Y, Kettner N, Sheehan J, et al. Altered brain morphometry in carpal tunnel syndrome is associated with median nerve pathology. *NeuroImage Clin*. 2013;2:313-319. [\[CrossRef\]](#)
 19. Druschky K, Kaltenhäuser M, Hummel C, et al. Alteration of the somatosensory cortical map in peripheral mononeuropathy due to carpal tunnel syndrome. *NeuroReport*. 2000;11(17):3925-3930. [\[CrossRef\]](#)
 20. Iwatsuki K, Yoshida A, Shinohara T, et al. Recovery function of somatosensory evoked brain response in patients with carpal tunnel syndrome: a magnetoencephalographic study. *Clin Neurophysiol*. 2016;127(8):2733-2738. [\[CrossRef\]](#)
 21. Bao BB, Qu DQ, Zhu HY, Gao T, Zheng XY. Brain remodeling after chronic median nerve compression in a rat model. *Neural Regen Res*. 2018;13(4):704-708. [\[CrossRef\]](#)
 22. Sasaki R, Tsuiki S, Miyaguchi S, et al. Repetitive passive finger movement modulates primary somatosensory cortex excitability. *Front Hum Neurosci*. 2018;12:332. [\[CrossRef\]](#)
 23. Cho HJ, Panyakaew P, Thirugnanasambandam N, Wu T, Hallett M. Dynamic modulation of corticospinal excitability and short-latency afferent inhibition during onset and maintenance phase of selective finger movement. *Clin Neurophysiol*. 2016;127(6):2343-2349. [\[CrossRef\]](#)