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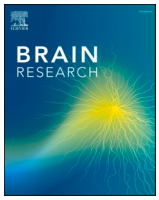

Research paper

Frequent object dropping in carpal tunnel syndrome: a consequence of impaired sensorimotor integration?

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ABSTRACT

Frequent object dropping is a common complaint in patients with carpal tunnel syndrome (CTS), suggesting potential disruptions in sensorimotor integration. This study investigated the electrophysiological characteristics of sensorimotor integration in CTS patients with and without this symptom. We enrolled twelve CTS patients with frequent object dropping (dCTS), ten CTS patients without noticeable clumsiness (ndCTS), and sixteen healthy controls. All participants underwent clinical evaluation, the Boston Carpal Tunnel Questionnaire, and the Purdue Pegboard Test. To assess sensorimotor integration, electrical stimulation was applied to the median and ulnar nerves, followed by transcranial magnetic stimulation over the contralateral motor cortex at interstimulus intervals (ISIs) of 20, 35, 50, 65, 80, 100, and 200 ms. Motor-evoked potentials (MEPs) were recorded from the thenar and hypothenar muscles. Key findings revealed distinct sensorimotor integration patterns on the dominant side. In the dCTS group, median nerve stimulation at a 35 ms ISI resulted in significantly higher MEP amplitude ratios in the thenar muscles compared to controls. Conversely, in the ndCTS group, ulnar nerve stimulation at ISIs of 20, 80, and 100 ms produced greater MEP ratios in the same muscles. These results demonstrate topographically divergent cortical sensory processing between dCTS and ndCTS patients. One interpretation of these findings is that altered sensorimotor integration from the median nerve underlies the clumsiness in dCTS, while patients without this symptom (ndCTS) may successfully compensate by utilizing sensory input from the ulnar nerve.

1. Introduction

Carpal tunnel syndrome (CTS), the most prevalent focal peripheral neuropathy, involves median nerve entrapment at the wrist (Atroshi et al., 2011). Beyond classic sensory symptoms, many patients report motor complaints like impaired dexterity and frequent object dropping—a symptom affecting nearly half of patients, particularly women, older individuals, and those with greater functional impairment (Pazzaglia et al., 2010; Isoardo et al., 2023; Matur et al., 2024). However, these motor symptoms often dissociate from severe weakness or atrophy (Padua et al., 1997; Tamburin et al., 2008; Pazzaglia et al., 2010), suggesting that small sensory fiber impairment can drive clumsiness without major motor fiber loss (Tamburin et al., 2008; Isoardo et al., 2023). Given the crucial role of proprioceptive and cutaneous

Abbreviations: BCTQ, Boston carpal tunnel questionnaire; dCTS, carpal tunnel syndrome patients with frequent object dropping; ISI, interstimulus interval; LAI, long-latency afferent inhibition; ndCTS, carpal tunnel syndrome patients without frequent object dropping; SAI, short-latency afferent inhibition; TMS, transcranial magnetic stimulation.

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feedback in motor control (Toth et al., 2022; Isoardo et al., 2023), this functional sensory loss likely disrupts motor performance directly.

In a substantial subset of CTS patients, symptoms extending beyond the median nerve territory imply central sensitization (Fernández-de-Las-Peñas et al., 2009a, Fernández-de-las-Peñas et al., 2009b; Zanette et al., 2010). Consistent with this, peripheral nerve lesions trigger central nervous system changes, including somatosensory cortex alterations in CTS such as: (1) digit map reorganization (Napadow et al., 2006; Dhond et al., 2012; Maeda et al., 2014; Iwatsuki et al., 2016); (2) gray matter thinning (Maeda et al., 2013, Maeda et al., 2016); and (3) modified brain networks (Xing et al., 2021; Osborne et al., 2022). This cortical maladaptive plasticity likely impairs sensorimotor integration, thereby underlying motor dysfunction in CTS.

Sensorimotor integration refers to the process by which peripheral sensory inputs modulate motor output (Ridding and Rothwell, 1999; Tamburin et al., 2001). In humans, combined transcranial magnetic stimulation (TMS) and peripheral nerve stimulation can assess sensorimotor integration by eliciting short-latency [SAI, 20–30 ms interstimulus intervals (ISI)] and long-latency (LAI: ~100 ms ISI) afferent inhibition, establishing a gold-standard paradigm (Tinazzi et al., 1998; Tokimura et al., 2000; Turco et al., 2018). This technique has documented sensorimotor integration deficits in various neurologic conditions including aging (Degardin et al., 2011), Parkinson's disease (Sailer et al., 2003), and dystonia (McDonnell et al., 2007), supporting its utility for investigating possible central perturbations related to CTS.

In this study, we aimed to examine sensorimotor integration differences between CTS patients with and without frequent object dropping by hypothesizing that object dropping in CTS results from impaired sensorimotor integration due to abnormal central plasticity driven by abnormal flow of peripheral sensory inputs.

2. Methods

2.1. Ethical approval and recruitment of subjects

The study was approved by the Institutional Ethics Committee of Istanbul University Istanbul Faculty of Medicine (Approval No: 1793-793). Written informed consent was obtained from all participants prior to enrollment.

A power analysis was conducted to determine the minimum sample size required for this study. Based on a large effect size (Cohen's *d* = 1.36) observed in a previous study on restless legs syndrome (Bocquillon et al., 2017) that used a similar sensorimotor integration paradigm, the analysis was performed using G*Power software (Version 3.1.9.7). The parameters were set for a two-tailed independent samples *t*-test, with an alpha level (α) of 0.05 and a statistical power (1-β) of 0.80. This analysis indicated that a minimum of 8 participants per group would be sufficient to detect a statistically significant effect of comparable magnitude. Therefore, this was established as the lower bound for our sample size target.

The study included 22 patients with CTS referred for electrophysiological evaluation who volunteered to participate. In our prior study (Matur et al., 2023), object dropping was initially assessed retrospectively by asking subjects if they had experienced any incidents without an external cause. However, pilot tests revealed that participants had significant difficulty accurately recalling the frequency of such events over a three-month period. To enhance data accuracy, we therefore implemented a prospective diary method, wherein participants were instructed to record each object-dropping incident as soon as possible after it occurred. The total number of incidents was reported at the end of a three-month monitoring period. Based on our previous findings, which indicated that a frequency of "equal to or more than once a week" was consistently perceived as troublesome by patients, this threshold was defined as "frequent object dropping" for the purpose of the study. Participants were divided into two groups based on symptom profiles:

dCTS group (N = 12): Patients reporting frequent object dropping (≥1 episode/week) described as a clinically distressing symptom.

ndCTS group (N = 10): Patients without complaints of frequent object dropping or notable hand clumsiness.

Sixteen healthy persons from the study team and from their family members were volunteered to be included in the control group. None of the patients and control cases have any neurological or systemic disease (e.g. diabetes) or were using drugs that have the potential of affecting the central and peripheral nervous system.

2.2. Clinical evaluation

Age (years), height (cm), and weight (kg) were recorded and body mass index (BMI, weight/height^2) was calculated for each participant. Handedness was determined by using the Edinburgh Handedness Inventory-10 (EHI-10, Oldfield, 1971), validated in Turkish by Atasavun Uysal et al. (Atasavun Uysal et al., 2019). Participants indicated their hand preference for 10 activities (like writing, brushing teeth, using scissors), choosing from always left-handed (−10 points) to always right-handed (10 points). Scores over 40 classified participants as right-handed, −40 to 40 as ambidextrous, and below −40 as left-handed. All cases, except one left-handed subject in the control group were found to be right-handed.

We recorded the duration of symptoms, along with the localization, severity, and diurnal characteristics of paresthesia, pain, other sensory symptoms, and feelings of weakness for all patients. The neurological examination included a detailed sensory, motor and reflex examination aiming for the identification of single nerve or nerve root lesions affecting the upper extremities as well as for the exclusion of a co-existing polyneuropathy. The cases were regarded as having CTS if they had one or more of the following symptoms and signs (Amirjani et al., 2011): (1) numbness and paresthesia in the hand, with or without pain in the upper extremity; (2) symptoms aggravated by repetitive hand movements or malposition, and relieved by resting, moving or shaking the hand; (3) nocturnal awakening and sleep disturbance by the symptoms, (4) hypo/dysesthesia in the median nerve distribution in the hand, (5) induction of the paresthesia by percussion/compression of the median nerve and (6) weakness in thumb abduction (with or without thenar atrophy), combined with electrophysiological confirmation of median nerve conduction abnormality across the wrist (see below).

Patient oriented data were obtained by applying a validated Turkish version of the Boston Carpal Tunnel Questionnaire (BCTQ) comprising of two parts, the Symptom Severity Scale (SSS) with 11 questions on pain, numbness, tingling, and weakness, and the Functional Status Scale (FSS) with 8 questions on daily activities, like writing and opening jars. Participants rated the symptoms from 1 (mild) to 5 (severe). Higher scores indicate more severe CTS (Levine et al., 1993; Sezgin et al., 2006).

The hand dexterity of the subjects was examined by using Purdue Pegboard test which was shown to be a valid and reliable tool to quantify functional impairment in CTS (Amirjani et al., 2011). This test was designed to assess fine motor hand function using three common objects, pegs, washers, and collars, to be inserted on a special pegboard (Model 32020; Lafayette Instrument Co., Lafayette, Indiana, USA). The test consisted of four subsets. In the first three subsets, subjects had 30 s to fill the holes with pegs initially with the dominant hand, then with the non-dominant hand, and finally with both hands simultaneously. In the last subset, subjects had 1 min to assemble in sequence a peg, a washer, a collar, and finally another washer with alternating hands. Each subset was repeated three times to obtain an average (Amirjani et al., 2011).

2.3. Electrophysiological tests

Electrophysiological studies were performed by using a 5 channel Nicolet Viking Select electromyography system (version 11.1) (Natus Medical Incorporated, CA 94070 USA) and a Magstim 200 magnetic stimulator connected to a figure-of-eight coil with the outside diameter of 7 cm for each ring (Magstim Company LTD, Whitland, UK).

2.3.1. Nerve conduction studies (NCSs)

The following studies were performed: 1) bilateral orthodromic sensory NCSs of the median and ulnar nerves, with stimulation of the 2nd and 5th fingers and recording at the wrist; 2) bilateral motor NCSs of the median and ulnar nerves, with recording from the thenar and hypothenar eminences, respectively; 3) comparison of ulnar and median sensory conduction with stimulation of the 4th finger and recording at the wrist if any abnormality in median nerve conduction was not detected in steps 1 and 2; 4) antidromic sural sensory and fibular motor NCSs to exclude concomitant polyneuropathy

Median nerve conduction abnormality at the wrist was defined according to the normal limits of our laboratory. The cases without such an abnormality were excluded from the study even though they had symptoms suggestive of the CTS. The severity of the electrophysiological findings were classified, according to the method described by Padua et al, as follows: extreme (absence of motor and sensory responses); severe (absence of sensory response and abnormal distal motor latency); moderate (abnormal digit/wrist sensory nerve conduction velocity and abnormal distal motor latency; mild (abnormal digit/wrist sensory nerve conduction velocity and normal distal motor latency) and minimal (abnormal segmental and comparative tests only) (Padua et al., 1997).

2.3.2. Sensorimotor integration

It was studied by pairs of electrical and magnetic stimuli consisting of submaximal electrical stimulation of the median and ulnar nerves at the wrist on the dominant and non-dominant sides, which were followed by TMS over the contralateral motor cortex. Motor responses were recorded simultaneously from the thenar and hypothenar muscles on the 1st and 2nd channels of the electromyograph connected to the surface electrodes placed according to the belly-tendon recording technique.

2.3.2.1. Electrical stimulation. A saddle shaped surface stimulator with the inter-electrode distance of 2.5 cm was placed cathode proximally over the median or ulnar nerves at the wrist in the following order: median nerve on the dominant side, ulnar nerve on the dominant side, median nerve on the non-dominant side and ulnar nerve on the non-dominant side. Submaximal electrical stimulation of each nerve was performed by manually activating the electrical stimulator to give stimuli of 100 ms duration at a frequency slower than once every 5 s with an intensity just eliciting a small twitch of the target muscle. The intensity of the electrical stimulation was also adjusted so as to induce a small motor response recorded from the electrodes placed on the hand muscles innervated by the stimulated nerve (electrode pair on the thenar eminence for median nerve stimulation and those on the hypothenar region for ulnar nerve stimulation) but not from those innervated by the other nerve. Switching for the electrical stimulation also initiated the magnetic stimulator after a certain ISIs.

2.3.2.2. Measuring motor thresholds. The figure-of-eight magnetic coil was placed tangentially over the motor cortex contralateral to the stimulated peripheral nerve (median or ulnar) with the handle pointing to the postero-lateral direction, 45° away from the midline and the point at which the TMS could evoke the highest amplitude motor responses from the thenar muscles with moderate stimulus intensities (usually 50 % of maximal stimulator output while the subject was performing a submaximal contraction effort) was determined (hot spot). This position was marked on the swimming cap worn by the subject, and the position of the coil was fixed with the aid of a coil holder. Then, the resting motor threshold (rMT) was measured by having the subject keep the thenar muscles fully relaxed. Single TMS pulses were applied to the motor cortex, and the intensity is gradually adjusted to determine the lowest level that consistently elicited a motor evoked potential (MEP) of >50 μV amplitude in at least 5/10 trials. Active motor threshold (aMT) was measured by having the subject contract the thenar muscles mildly, typically at 10–20 % of maximum voluntary contraction. Single TMS pulses were delivered over the motor cortex, and the intensity was gradually adjusted to determine the lowest level that consistently elicited a MEP of >200 μV amplitude in at least 5/10 trials in the active muscle. The 1 mV motor threshold was determined by adjusting the TMS intensity in 2–3 % increments to find the lowest stimulator output that consistently elicited motor responses with an amplitude of ≥1 mV in the fully relaxed thenar muscle, as monitored on the EMG screen and audio, in at least 5 out of 10 stimulations.

2.3.2.3. Sensorimotor integration. Magnetic stimulation intensity was kept constant at 1 mV motor threshold level throughout the study performed with stimulations over the same hemisphere. MEPs were recorded simultaneously from surface electrodes over the thenar and hypothenar regions. The sensorimotor integration study was performed by applying pairs of stimuli (electrical and magnetic) with ISIs of 20, 35, 50, 65, 80, 100 ve 200 ms. These ISI values were corrected for the somatosensory evoked potential (SEP) N20 latency recorded in each patient [The latency of N20 potential nearly equals to the conduction time from the stimulation point at the wrist to the somatosensory cortex. This potential was recorded in each case before the sensorimotor integration experiment by stimulating the median nerve at the wrist and recording from the contralateral cortex (C3 or C4 referenced to Fz)]. Eight stimulations for each ISI were performed in a pseudo-randomized order, and the recordings were made with 500 ms sweep speed and 0.2–2 mV/division sensitivity. The ratios of the mean peak to peak amplitudes and areas of the MEPs obtained at each ISI to those of the 8 MEPs elicited by single magnetic test stimuli were calculated (MEP ratio) to draw the graphs delineating the sensorimotor integration course.

2.4. Statistical Methods

All analyses were performed using SPSS (version 25.0; IBM Corp., Armonk, NY, USA). The significance threshold was set at $p < 0.05$. Different statistical tests were applied based on the nature and distribution of the data. Descriptive statistics for continuous variables were expressed as mean ± standard deviation (SD) and median (interquartile range, IQR), while categorical variables were reported as count (%).

Numerical demographic and clinical data were compared using the Kruskal-Wallis test for differences among the three groups. When a significant difference was detected, pairwise comparisons were conducted using the Mann-Whitney *U* test. To control for Type I error inflation due to multiple comparisons, the significance level for these post-hoc tests was adjusted using the Bonferroni correction, and the adjusted p-values were reported. Categorical variables were compared using Pearson's chi-square test.

The effects of ISI on sensorimotor integration in controls were assessed using Friedman's two-way ANOVA by ranks. Multivariate analyses compared (a) CTS patients versus controls across ISIs, and (b) dCTS, ndCTS, and control groups (ISIs as dependent variables; group as fixed factor), with post-hoc analyses performed with the Bonferroni correction to account for multiple comparisons. The results section presents the adjusted F-values, Bonferroni-corrected p-values, and partial eta squared (η2) values derived from this procedure. All reported p-values for these multi-comparison analyses reflect this adjusted p values.

3. Results

3.1. Clinical findings

The mean age of the subjects included in the study was 44.7 years (range: 23–65). The ndCTS group was significantly older than the control group ($p = 0.004$), despite the dCTS and ndCTS groups having comparable ages ($p = 1.000$). The dCTS group had the highest average body weight, with significant differences in both body weight and BMI were found between the dCTS and control groups ($p = 0.002$). All

patients with CTS were right-handed, while one participant in the control group was left-handed (Table 1).

The CTS-related properties and BCTQ scores of the case groups are detailed in Table 2. The electrophysiological severity of CTS was greater on the dominant side of the patients; however, no significant difference in CTS severity was found between the dCTS and ndCTS groups. The mean symptom duration was 14.08 ± 4.3 months in the dCTS group and 15.3 ± 2.3 months in the ndCTS group (p = 0.441). There was no significant difference in the duration of CTS symptoms and the clinical characteristics of CTS between the dCTS and ndCTS groups (Table 2). The BCTQ SSS and FSS scores were higher in the dCTS group compared to the ndCTS group. A significant difference was found between the CTS groups for the total SSS score (Table 2). The Purdue Pegboard Test results showed that motor skills, manual dexterity, and bimanual coordination were poorer in dCTS patients than in controls. (Table 3, Supplementary Table 1).

3.2. Electrophysiological results

3.2.1. NCS results

There were no significant differences between dCTS and ndCTS groups with respect to the severity of median nerve conduction abnormalities found in NCS. Minimal to mild median nerve conduction abnormalities were found in 11 dCTS and 10 ndCTS hands, whereas moderate degree abnormalities were present in 10 dCTS and 8 ndCTS hands and there was severe abnormality in only one ndCTS hand (Table 2, Supplementary Table 1).

3.2.2. Sensorimotor integration studies

Mean motor thresholds (% maximum stimulator output) were: aMT (dominant: 38.9 % ± 6.2; non-dominant: 39.8 % ± 4.8), rMT (dominant: 48.6 % ± 7.5; non-dominant: 49.8 % ± 6.4), and 1 mV MT (dominant: 62.0 % ± 9.2; non-dominant: 62.7 % ± 8.0). No side- or group-related differences reached significance (p > 0.05 in all comparisons).

Mean + SD values of the amplitude ratios at *different ISIs in dCTS, ndCTS and control groups* are presented in the Supplementary Table 2. Since the amplitude and area ratios were found to be very similar throughout the study, only the amplitude ratio values are provided for simplicity.

The ISI showed a statistically significant effect on sensorimotor integration values in the control group (p < 0.001). Pairwise comparisons with Bonferroni correction revealed significant differences between (a) ISI 20 vs. 35/50/65/200 ms, (b) ISI 80 vs. 35/50/65/200 ms, and (c) ISI 100 vs. 35/50/65/200 ms. SAI was observed at approximately 20 ms and LAI at 80–100 ms. The median nerve-thenar muscle protocol revealed maximal SAI/LAI, particularly following dominant median nerve stimulation (Fig. 1). The patients with CTS exhibited significantly reduced SAI in response to right median nerve stimulation. This was accompanied by a shift towards enhanced afferent facilitation, which peaked at approximately 35 ms (F = 7.049, p = 0.012, η^2 = 0.164) (Fig. 1A). Furthermore, analysis of the left ulnar nerve pathway revealed that LAI at an ISI of 200 ms was present in CTS patients but absent in healthy controls (F = 7.077, p = 0.012, η^2 = 0.172) (Fig. 1D).

Sensorimotor integration profiles demonstrated distinct, side-specific abnormalities in CTS patients. Following dominant-side stimulation, significant between-group differences emerged. Median nerve stimulation recorded from thenar muscles revealed enhanced afferent facilitation at the 35 ms ISI in the dCTS group compared to controls (F = 4.929, p = 0.013, η^2 = 0.220; post hoc p = 0.010; Fig. 2A). Ulnar nerve stimulation showed complex modulation: thenar recordings were significant at 20, 80, and 100 ms ISIs, with the ndCTS group exhibiting higher ratios than the dCTS group at 20 ms and surpassing controls at 80 and 100 ms (F = 4.239–5.125, all p < 0.05; Fig. 2B). Furthermore, hypothenar recordings revealed pronounced long-latency afferent inhibition (LAI) specifically in the dCTS group at 200 ms ISI (F = 6.585, p = 0.004, η^2 = 0.273; post hoc p ≤ 0.031 vs. other groups; Fig. 2B).

In contrast, non-dominant side stimulation yielded less pronounced but significant findings. Median nerve stimulation recorded from

Table 2
Clinical characteristics of patients with carpal tunnel syndrome.

		dCTS (N = 12)	ndCTS (N = 10)	P value *
CTS side	Right Bilateral	210	28	0.840
CTS severity**				
Dominant side	Minimal/ Mild/Moderate/ Severe	6/ 1/5/0	1/ 4/4/1	0.096
Non-dominant side	Negative/ Minimal/Mild/ Moderate	3/ 3/1/5	2/ 2/3/3	0.660
Duration of symptoms (months)		14.08 ± 4.3	15.3 ± 2.3	0.441
Nocturnal paraesthesia/pain		11	9	0.892
Daytime paraesthesia/pain		12	9	0.262
Relief of symptoms by shaking hand		10	9	0.650
Sensory deficit		7	2	0.069
Motor weakness		3	2	0.781
Thenar atrophy		0	0	–
Tinel's/Phalen's sign		11	7	0.050
Boston Carpal Tunnel Questionnaire				
Symptoms Severity Scale		3.6 (2.5)	2.2 (1.7)	**0.003**
Functional Status Scale		3.5 (3.7)	2.4 (1.9)	0.059

CTS, carpal tunnel syndrome; dCTS, CTS with frequent object dropping; N, number of patients; ndCTS, CTS patients without frequent object dropping.
Categorical variables are presented as number and numerical variables are presented as mean ± standard deviation or median (interquartile range).
* Comparisons between groups were made using Pearson Chi-Square Test for categorical variables and Mann-Whitney *U* test for numerical variables.
** Severity of CTS reflected by the electrophysiological parameters.

Table 1
Demographic characteristics of the groups.

	dCTS (N=12)	ndCTS (N=10)	Control (N=16)	*Test Statistic	P values			
					All	dCTS vs ndCTS	dCTS vs control	ndCTS vs control
Age (years)	45.3 ± 6.7	52.2 ± 7.1	39.6 ± 10	7.076	**0.006**	0.303	0.366	**0.004**
Height (m)	1.6 ± 0.1	1.6 ± 0.1	1.6 ± 0.1	0.443	0.844			
Weight (kg)	84.3 ± 19.1	74.1 ± 16.2	57.9 ± 8	10.164	**0.002**	1.000	**0.002**	0.068
BMI (kg/m^2)	31.8 ± 7.2	27.4 ± 3.3	22.4 ± 3.1	11.4	**0.002**	1.000	**0.002**	0.076
Sex (men/women)	2/10	2/8	2/14	0.270	0.874			
Handedness(right/left)	12/0	10/0	15/1	0.270	0.874			

BMI, body mass index; CTS, carpal tunnel syndrome; dCTS, CTS patients with frequent object dropping; N, number of patients; ndCTS, CTS patients without frequent object dropping.
Categorical variables are presented as number and numerical variables are presented as mean ± standard deviation.
Statistically significant differences are highlighted by bold numbers.
* Comparisons between groups were made using Pearson Chi-Square Test for categorical variables and Kruskal-Wallis test for numerical variables. Significance values have been adjusted by the Bonferroni correction for multiple tests.

Table 3
Purdue Pegboard Test scores across study groups.

Tasks	dCTS	ndCTS	Control	*p-values	**Pairwise comparisons, p-values		
	median (IQR)	median (IQR)	median (IQR)		dCTS vs. ndCTS	dCTS vs. Controls	ndCTS vs. Controls
Dominant hand task	13.33 (2)	15.83 (2.33)	17 (2.67)	<0.001	0.095	<0.001	0.581
Non-dominant hand task	12.50 (1.33)	14.67 (2.58)	16.33 (2)	<0.001	0.025	<0.001	0.531
Both hands task	20 (1.83)	22.33 (4.84)	27 (3.34)	<0.001	0.132	<0.001	0.242
Assembly task	22.66 (2.59)	27.33 (9.84)	33.66 (12)	0.001	0.095	0.001	0.581

CTS, carpal tunnel syndrome; dCTS, CTS patients with frequent object dropping; IQR, interquartile range; ndCTS, CTS patients without frequent object dropping.
* Comparisons between groups were made using Kruskal-Wallis Test.
** Pairwise comparisons were performed using the Mann-Whitney *U* test with Bonferroni correction. Significance values have been adjusted by the Bonferroni correction for multiple tests.

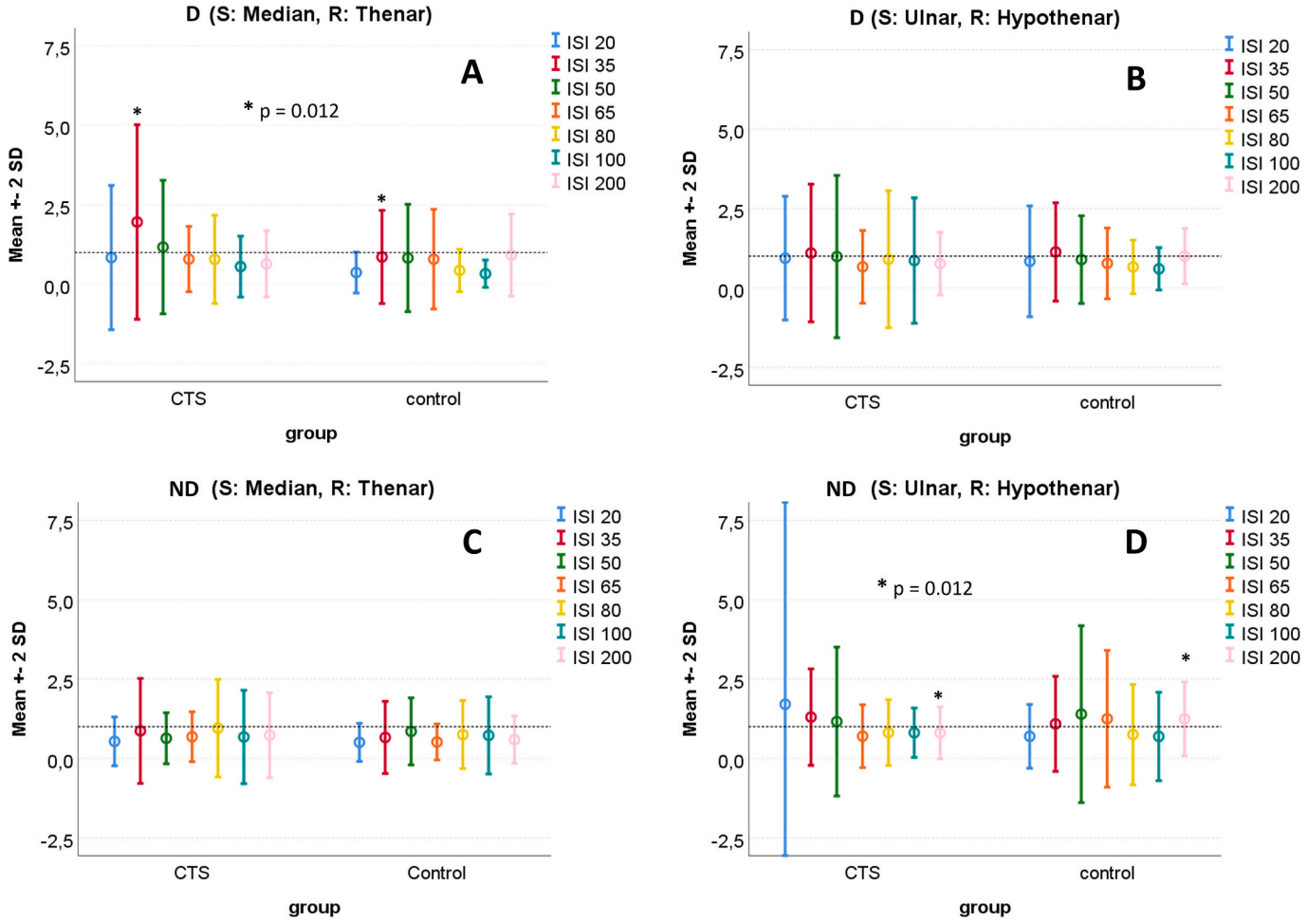

Fig. 1. Sensorimotor integration profiles in healthy controls and CTS patients. Amplitude ratios of sensorimotor integration assessments are shown for (A) dominant and (C) non-dominant median nerve stimulation (recorded from thenar muscles), and for (B) dominant and (D) non-dominant ulnar nerve stimulation (recorded from hypothenar muscles). The median nerve-thenar muscle protocol revealed maximal reduction in short- and long-latency afferent inhibition (SAI/LAI), particularly following dominant median nerve stimulation. The patients with CTS exhibited significantly reduced SAI in response to dominant median nerve stimulation. This was accompanied by a shift towards enhanced afferent facilitation, which peaked at approximately 35 ms ($F = 7.049$, $p = 0.012$, $\eta^2 = 0.164$) Furthermore, analysis of the left ulnar nerve pathway revealed that an LAI at an ISI of 200 ms was present in CTS patients but absent in healthy controls ($F = 7.077$, $p = 0.012$, $\eta^2 = 0.172$). CTS, carpal tunnel syndrome; D, dominant; ISI, interstimulus interval; ND, non-dominant; R, recording site; S, stimulating site; SD, standard deviation.

hypothenar muscles showed facilitation in the dCTS group at 65 ms ISI ($F = 4.442$, $p = 0.020$, $\eta^2 = 0.212$; post hoc $p = 0.029$; Fig. 3B). Ulnar nerve stimulation, however, revealed consistent LAI at 200 ms ISI in the dCTS group across both thenar and hypothenar recordings (Thenar: $F = 4.234$, $p = 0.023$, $\eta^2 = 0.204$; Hypothenar: $F = 4.086$, $p = 0.026$, $\eta^2 = 0.198$; post hoc for both $p = 0.024$ vs. controls; Fig. 3C, D).

4. Discussion

Our study on frequent object dropping in CTS revealed that dCTS patients exhibited: (1) a non-significant trend toward more sensory deficits and Tinel signs; (2) worse scores on the BCTQ-SSS; and (3) distinct sensorimotor integration patterns, indicating an altered influence of sensory inputs on motor outputs. These findings suggest that clumsiness in CTS may stem from maladaptive CNS reorganization rather than from peripheral dysfunction alone.

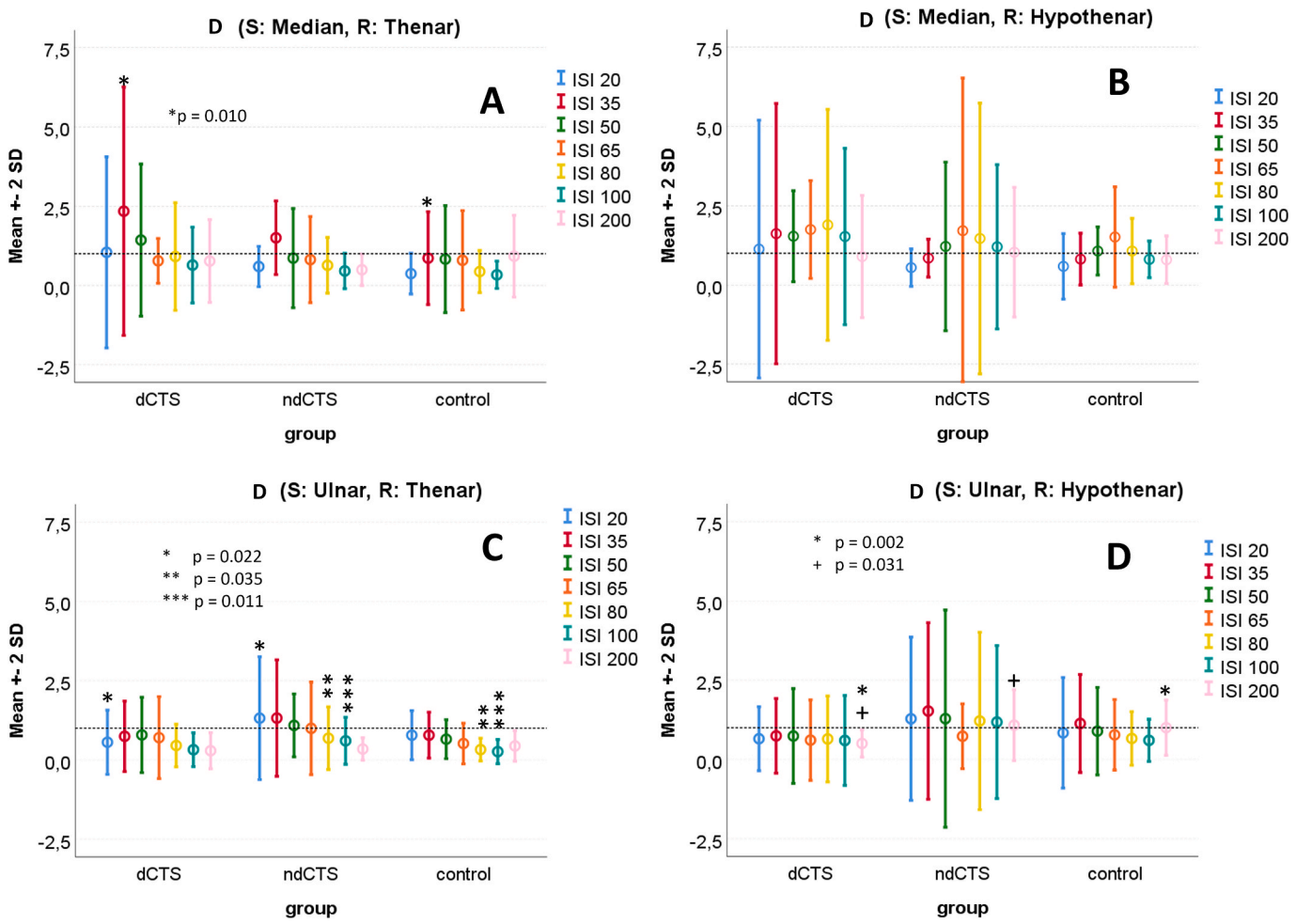

Fig. 2. Sensorimotor integration profiles following dominant-side nerve stimulation. Amplitude ratios are shown for median (A, B) and ulnar (C, D) nerve stimulation on the dominant side, recorded from thenar (A, C) and hypothenar (B, D) muscles. A) Following median nerve stimulation, thenar recordings in the dCTS group showed significant afferent facilitation at the 35 ms ISI compared to controls ($F = 4.929$, $p = 0.013$, $\eta^2 = 0.220$; post hoc $p = 0.010$). C) Ulnar nerve stimulation revealed complex modulation on thenar recordings, with significant effects at 20, 80, and 100 ms ISIs ($F = 4.239$, $p = 0.022$, $\eta^2 = 0.195$; $F = 3.549$, $p = 0.035$, $\eta^2 = 0.169$; $F = 5.125$, $p = 0.011$, $\eta^2 = 0.227$). Post hoc tests indicated that the ndCTS group had higher ratios than the dCTS group at ISI 20 ms, and ndCTS group had higher ratios than controls at ISI 80 and 100 ms. D) In hypothenar recordings following ulnar nerve stimulation, a significant LAI was observed specifically in the dCTS group at the 200 ms ISI ($F = 6.585$, $p = 0.004$, $\eta^2 = 0.273$; post hoc $p \leq 0.031$ vs. other groups). CTS, carpal tunnel syndrome; D, dominant; dCTS, CTS patients with frequent object dropping; ISI, interstimulus interval; ndCTS, CTS patients without frequent object dropping; R, recording site; S, stimulation site; SD, standard deviation.

Peripheral nerve injuries induce well-documented maladaptive CNS plasticity, demonstrated in animal models by multilevel changes from disinhibition to dendritic growth (Jain et al., 1998; Han et al., 2013; Krishnan et al., 2022). In humans, this is evidenced by cortical reorganization and gray matter thinning that correlate with clinical deficits and contribute to incomplete recovery (Hansson and Brismar, 2003; Lundborg and Rosén, 2007; Taylor et al., 2009;). CTS serves as a key clinical model for these changes, where neurophysiological and neuroimaging studies reveal altered somatosensory organization, structural brain changes, and abnormal cortical processing that correlate with symptom profiles (Tinazzi et al., 1998; Tecchio et al., 2002; Napadow et al., 2006; Dhond et al., 2012; Maeda et al., 2013; Maeda et al., 2014).

Fine motor deficits are well-documented in CTS (Fernández-de-Las-Peñas et al., 2009a; Pazzaglia et al., 2010; Li et al., 2015), with frequent object dropping representing a particularly distressing manifestation of impaired motor control. While object dropping is associated with more severe CTS (Pazzaglia et al., 2010), the correlation between dexterity measures and symptom scores remains weak (Amirjani et al., 2011), and neurographic parameters poorly predict motor symptoms like clumsiness (Tamburin et al., 2008). This discrepancy has been attributed to C-fiber-mediated pain that modulates motor function at central levels (Tamburin et al., 2008; Fernández-de-Las-Peñas et al., 2009a), leading us to hypothesize that object dropping may specifically reflect deficient sensorimotor integration, which can be assessed through electrophysiological studies.

Consistent with established sensorimotor integration protocols, we stimulated nerve trunks to elicit robust SAI (Ridding and Rothwell, 1999; Tamburin et al., 2001; Tokimura et al., 2000), optimizing detection of sensory input's effect on motor outputs. Our results revealed topographically distinct patterns: on the dominant side, dCTS patients showed reduced SAI from the affected median nerve, suggesting a maladaptive cortical strategy linked to object dropping. Conversely, ndCTS patients showed reduced SAI from the unaffected ulnar nerve, indicating a potential compensatory mechanism that preserves motor function. Sensorimotor integration patterns were less distinct on the non-dominant side, likely reflecting less severe median nerve pathology.

A key observation was the dissociation between electrophysiological and clinical measures. While the severity of median nerve injury was similar between dCTS and ndCTS groups, BCTQ symptom scores were significantly higher in the dCTS group. This suggests that their

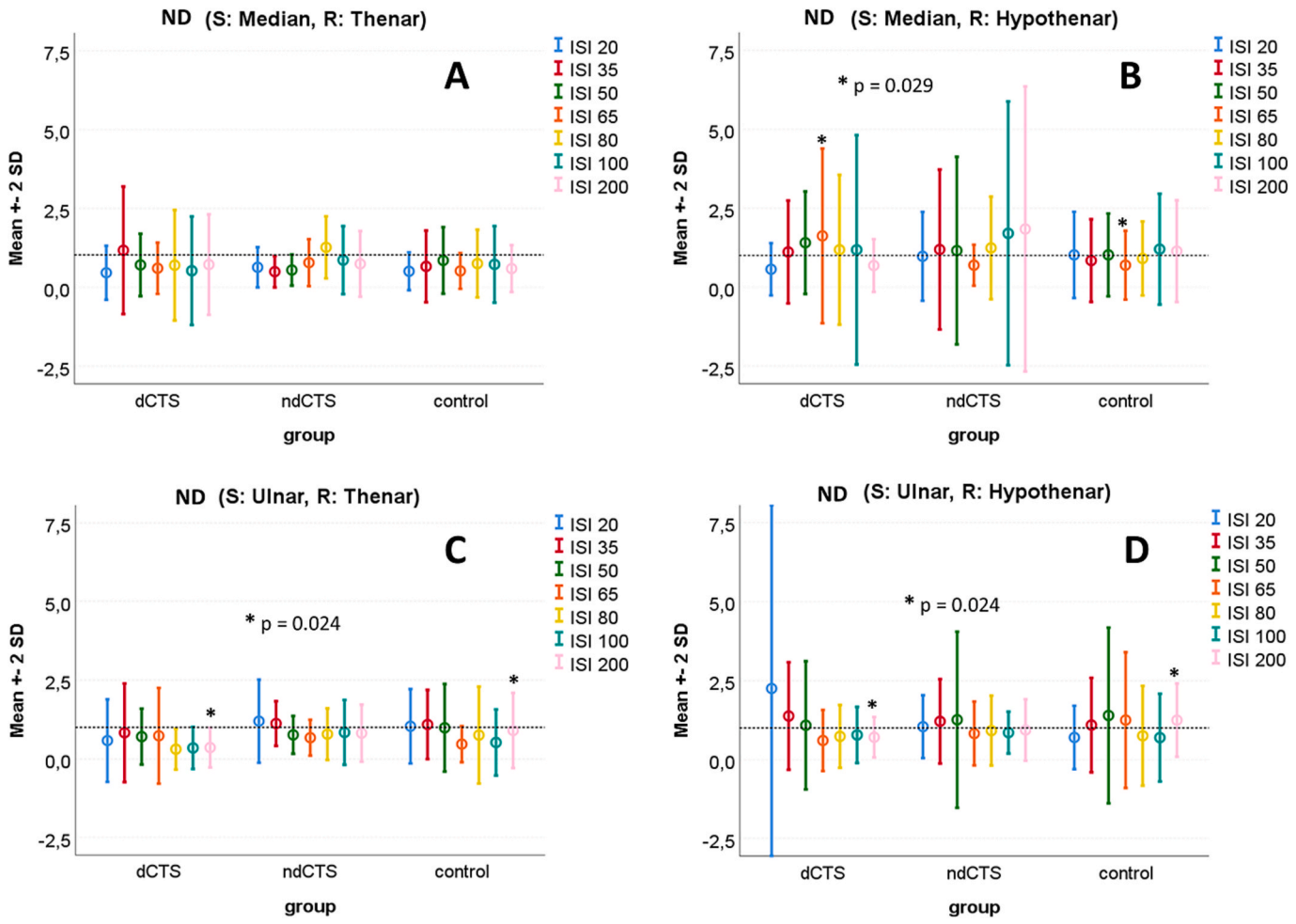

Fig. 3. Sensorimotor integration profiles following non-dominant side nerve stimulation. Amplitude ratios are shown for median (A, B) and ulnar (C, D) nerve stimulation on the non-dominant side, recorded from thenar (A, C) and hypothenar (B, D) muscles. Compared to dominant-side stimulation, between-group differences were less pronounced. Key findings include: B) Significant facilitation in hypothenar recordings at 65 ms ISI following median nerve stimulation in dCTS compared to controls ($F = 4.442$, $p = 0.020$, $\eta^2 = 0.212$; post hoc $p = 0.029$). C-D) Ulnar nerve stimulation revealed significant long-latency inhibition at 200 ms ISI in dCTS patients across both thenar ($F = 4.234$, $p = 0.023$, $\eta^2 = 0.204$; post hoc $p = 0.024$) and hypothenar recordings ($F = 4.086$, $p = 0.026$, $\eta^2 = 0.198$; post hoc $p = 0.024$) compared to controls. CTS, carpal tunnel syndrome; dCTS, CTS patients with frequent object dropping; ISI, interstimulus interval; ND, non-dominant; ndCTS, CTS patients without frequent object dropping; R, recording site; S, stimulation site; SD, standard deviation.

pronounced clumsiness and object dropping may not be solely due to peripheral nerve compromise. This is supported by the Purdue Pegboard Test, which showed no significant intergroup differences. The discrepancy between robust self-reported symptoms and objective performance implies this complaint is not rooted purely in manual dexterity deficits. Collectively, these dissociations point towards a potential role for altered central sensorimotor integration.

The maladaptive sensorimotor patterns in dCTS—featuring reduced inhibition—provide a neurophysiological correlate for this. This suggests clumsy hand function may stem from differences in central sensory processing. Two non-exclusive mechanisms may explain this: 1) Sensory-driven maladaptation: More intense paresthesia/pain in dCTS may bombard the cortex, disrupting integration. 2) Plasticity-driven symptoms: Inherent differences in cortical plasticity could amplify sensory perception and impair motor control. Conversely, the ndCTS group may employ a more effective compensatory strategy, preserving dexterity despite similar peripheral compromise.

Evidence suggests that severe sensory symptoms in CTS drive motor dysfunction via maladaptive central mechanisms (Tamburin et al., 2008; Fernández-de-Las-Peñas et al., 2009a). Neuroimaging reveals that these symptoms directly shape cortical reorganization. MEG studies show enlarged hand representations during paresthesia, suggesting a "central magnification" to counter peripheral loss (Tecchio et al., 2002), while other work indicates chronic paresthesia renders sensorimotor cortices "busy," reducing processing capacity and correlating with poorer psychomotor performance (Napadow et al., 2006; Dhond et al., 2012; Maeda et al., 2013, Maeda et al., 2014).

Collectively, this establishes that aberrant sensory input disrupts motor output. However, prior studies—by not stratifying patients by clumsiness—have conflated distinct neural strategies. We hypothesize that ulnar nerve facilitation in ndCTS represents an effective compensatory strategy, contrasting with the maladaptive "busy brain" in dCTS. Systematically comparing these subgroups could resolve contradictions in the literature regarding median versus ulnar nerve responses (Tinazzi et al., 1998; Tecchio et al., 2002; Napadow et al., 2006) and clarify the neural determinants of motor impairment.

Our study has several limitations that should be considered. First, the sample size, while sufficient to detect the primary neurophysiological differences, was inadequate to investigate the complex potential interactions between some factors such as hand dominance, CTS severity and bilaterality. Second, the absence of neuroimaging data represents a significant constraint. While our sensorimotor integration findings reveal distinct functional profiles, correlative structural or network-level data from other techniques like MRI or MEG could have provided a more

comprehensive understanding of the neural substrates differentiating dCTS from ndCTS patients. This limitation is compounded by the fact that previous neuroimaging studies in CTS have not stratified participants based on the presence of object dropping, meaning the existing literature likely conflates distinct cortical adaptation strategies.

In conclusion, this study provides comprehensive neurophysiological evidence that carpal tunnel syndrome is associated with distinct and side-specific alterations in sensorimotor integration, extending beyond mere peripheral nerve dysfunction. Our key finding is that the functional impairment of the dominant hand in dCTS is reflected in a maladaptive central nervous system response, characterized by a shift from inhibitory towards facilitatory processing in median nerve pathways, alongside a paradoxical emergence of inhibitory effects in non-afflicted ulnar nerve pathways. These findings suggest that chronic compression of the median nerve triggers complex cortical reorganization, the pattern of which is heavily influenced by hand dominance and the functional demands placed on the sensorimotor system. The demonstrated sensorimotor integration abnormalities offer a novel neurophysiological correlate for the clinical symptoms of manual clumsiness and object dropping reported by the patients. Therefore, assessing sensorimotor integration profiles could serve as a valuable biomarker for evaluating central plastic changes in CTS. Future longitudinal studies incorporating neuroimaging are warranted to elucidate the causal trajectory of these adaptations and to explore their potential reversibility following therapeutic interventions.

5. Authors' contribution and agreement

All authors have reviewed and approved the final version of the submitted manuscript. The authors warrant that this work is original, has not been published previously, and is not under consideration for publication elsewhere.

CRediT authorship contribution statement

Zeliha Matur: Writing – original draft, Validation, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Nimet Dörtcan:** Writing – original draft, Methodology, Investigation, Data curation, Conceptualization. **Sertaç İmişçi:** Visualization, Investigation, Data curation. **Melis Süner:** Visualization, Investigation, Data curation. **Zeynep Acar:** Visualization, Investigation, Data curation. **Nejla Sözer:** Visualization, Methodology, Conceptualization. **Ali Emre Öge:** Writing – review & editing, Supervision, Methodology, Investigation, Formal analysis, Data curation, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.brainres.2025.150029.

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