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Comparative effects of tensioning and sliding neural mobilization on peripheral and autonomic nervous system function: A randomized controlled trial

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Background: Although different types of neural mobilization (NM) exercises induce different amounts of longitudinal nerve excursion and strain, the question whether the increased longitudinal stress and nerve excursion from sliding or tensioning intervention may subtly affect the neural functions has not been answered yet.
Objective: To compare the effects of tensioning NM versus sliding NM of the median nerve on peripheral and autonomic nervous system function.
Methods: In this randomized controlled trial, 90 participants were randomly assigned to tensioning NM, sliding NM, or sham NM. The neurophysiological outcome measures included peak-to-peak amplitude of the dermatomal somatosensory evoked potential (DSSEP) for dermatomes C6, C7, C8, and T1. Secondary outcome measures included amplitude and latency of skin sympathetic response. All outcome measures were assessed pretreatment, immediately after the two weeks of treatment and one week after the last session of the treatment.
Results: A 2-way repeated measures ANOVA revealed significant differences between the three groups. The *post hoc* analysis indicated that tensioning NM significantly decreased the dermatomal amplitude for C6, C7, C8, and T1 ($p < 0.005$). Sympathetic skin responses in the gliding NM group showed lower amplitudes and

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prolonged latencies post-treatment when compared to tensioning NM group ($p < 0.05$). In contrast, no significant changes were observed in the DSSEPs and skin sympathetic responses for participants in the sham treatment group ($p > 0.05$).
Conclusions: A tensioning NM on the median nerve had a possible adverse effect on the neurophysiology variables of the nerves involved in the neural mobilization. Thus, tensioning NM with the current parameters that place increased stress and strain on the peripheral nervous system should be avoided.

Keywords: Median nerve; neural; autonomic nervous system; evoked potentials; randomized controlled trial.

1. Introduction

Neural mobilization (NM) is a movement-based intervention aimed at integrating structural, biomechanical, and functional aspects of the nervous system.[1] It targets restoration of homeostasis in and around the neural tissues.[2] NM is delivered using "sliding/gliding" and "tensioning" techniques. Though both techniques are performed to recover the normal mechanics of the peripheral neural structures and to augment the optimal neural function,[3–6] the longitudinal excursion and strain exerted by them are different.[7] Accordingly, their impact on neural function will vary.

The suggested benefits of such techniques (e.g., improved nerve gliding, decreased nerve adherence, increased neural vascularity, improved flow of axoplasm, and dispersal of noxious substances)[1] potentially promote the optimum neural physiologic functions. However, the comparisons and subsequent conclusions about the effectiveness of NM techniques in the majority of the previous studies were primarily focused on biomechanical factors such as range of motion, flexibility, and muscle strength. Moreover, those studies were of heterogeneous nature with each one including participants suffering from different pathologies and employing different types of NM.[8–12]

Neurodynamic concept refers to the integration of biomechanical, functional, and morphological characteristics of the nervous system[1]; this has made direct quantitative analysis of physiological neural function more important. In this regard, very limited studies have investigated the neurophysiological parameters. All the previous studies[13–15] testing the physiological effects of NM, have not clarified exactly what impact the NM has on nerve root which is considered as the most sensitive structure to longitudinal stresses and strain owing to the absence of the perineurium.[16] In terms of neurophysiological outcomes, we used dermatomal somatosensory evoked potential (DSSEP) to mitigate the issues arising from mixed nerve stimulation associated with the F wave or mixed nerve somatosensory-evoked potentials. Moreover, DSSEP provides reliable outcomes of segmental nerve root function that correlates well to clinical features compared to the other electrophysiological parameters.[19,20]

Although limited in the literature, other potential effects of NM on autonomic nervous system function have been reported.[17] The hypothesized autonomic benefits include increased neural vascularity and decreased skin temperature. Currently, there are no randomized controlled clinical trials investigating the effects of different NM techniques on nerve root function and autonomic nervous system. Therefore, our study was aimed at comparing the effects of sliding and tensioning NM techniques targeting the neurophysiological parameters of the median nerve and autonomic neural function. As the findings of previous studies clearly demonstrate that different types of NM have largely different mechanical effects on the peripheral nervous system producing markedly different amounts of strain and longitudinal excursion,[18–20] we hypothesized that the tensioning and sliding NM techniques would differently affect the neural functions.

2. Methods

A prospective, parallel, randomized controlled trial was conducted at a research laboratory of the University of Sharjah, UAE. The patients were enrolled in the study from December 2020 to January 2021 for a 2-week intervention with a 1-week follow-up. The trial has been registered with the ClinicalTrials.gov NCT04690959 where the full

Table 1. Baseline participants' demographics, clinical and anthropometric information.

	G1 ($N = 30$)	G2 ($N = 30$)	G3 ($N = 30$)
Age (y)	20 ± 2.5	21 ± 2	20 ± 3
Weight (kg)	68 ± 5	68 ± 4	66 ± 7
Gender (%)			
Male	5 (16.7%)	4 (13.3%)	5 (16.7%)
Female	25 (83.3%)	26 (86.7%)	25 (83.3%)
Smoking status			
Nonsmoker	20	19	22
Smoker	10	11	8
Tampa Scale of Kinesiophobia - General Population	22.6 ± 3.6	22.4 ± 3.5	22.7 ± 3.5
State-Trait Anxiety Inventory	32.7 ± 5.5	32.1 ± 7.5	31.6 ± 5

protocol can be accessed. Ethical approval was obtained from the institutional research ethics committee (REC-19-10-31-03-S) and informed consents were received from all participants before data collection in accordance with relevant guidelines and regulations.

Participants were recruited using printed adverts and through social media. These advertisements were targeted at the students, alumni, and employees of the University.

3. Participants

Ninety participants participated in this study. Healthy young individuals from both genders were included in this study if they were aged between 18 and 30 years and had no current or previous

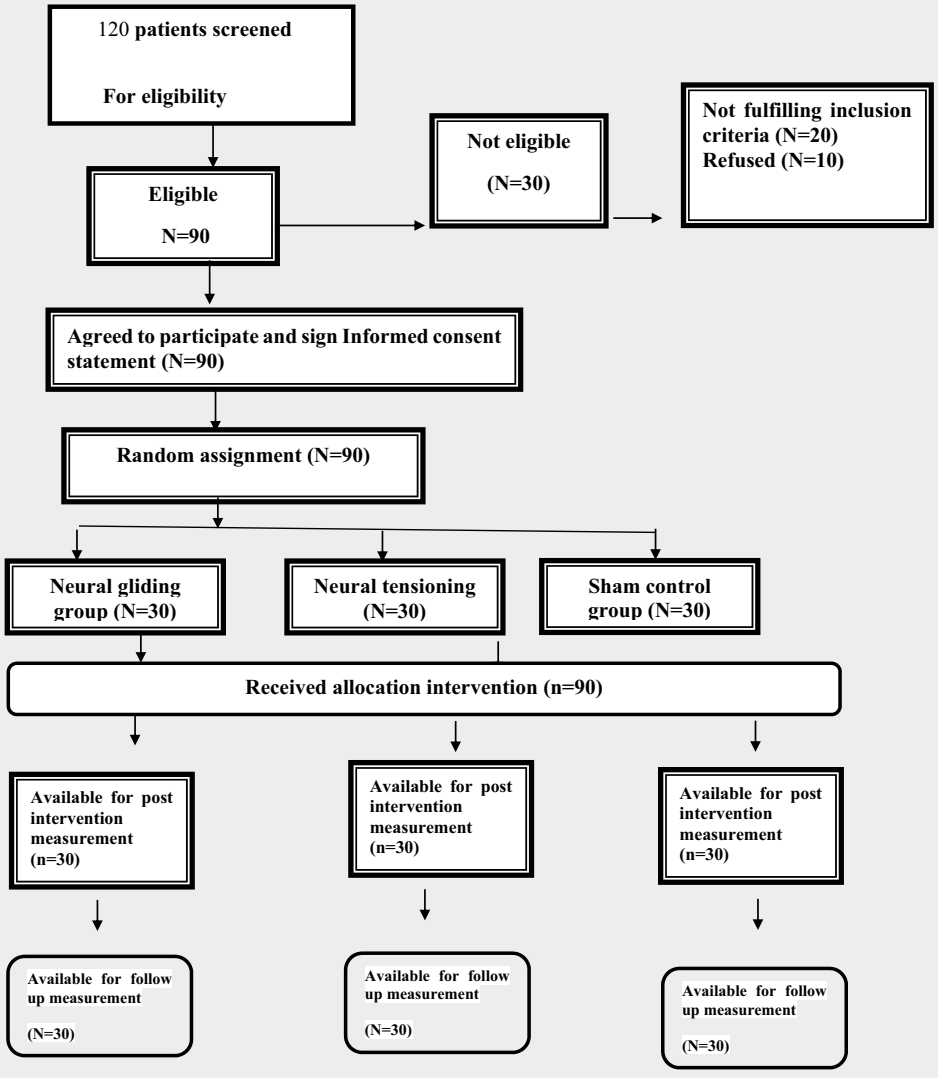

Fig. 1. Flow chart.

complaints of neck or dominant upper limb pain within the last year (Table 1).

Potential participants were screened for the following exclusion criteria: (i) Inflammatory joint disease or other systemic pathologies; (ii) prior history of overt injury and surgery relating to the musculoskeletal system, or disorder related to the spine and extremities (Fig. 1).

Initially, two self-report questionnaires were administered to know if the groups differed on important variables that might alter neurophysiological findings. These questionnaires were as follows:

(1) The Tampa Scale of Kinesiophobia-General Population (TSK-G): The TSK-G is a 12-item self-report checklist based on a 4-point Likert scale. A high score indicates an increasing likelihood for kinesiophobia. This is a valid and reliable version for assessing the fear of movement among the general population.[21]
(2) The State-Trait Anxiety Inventory: This is a psychological inventory that uses a 4-point Likert scale and consists of 20-item with high scores indicating increased levels of anxiety.[22]

The participants were randomly allocated to one of the three groups: (1) Tensioning NM group, (2) Sliding NM group, and (3) Control group (that received sham treatment). The randomization process was based on permuted blocks of variable sizes. Each random permuted block, created randomly by a number generator, was added to sequentially numbered, opaque, sealed envelopes which were kept in a locked cabinet until needed. Once a participant was included in the study, the next envelope in the sequence was opened by the researcher in the presence of the participant who would be assigned to a group according to the number found in the envelope (where the participant would still be blinded to the group they were assigned to).

4. Interventions

Neural tensioning mobilization: For this technique, joint movements were simultaneously performed to increase the length of the nerve bed, while allowing tensile loading to the median nerve. With the participant in supine lying, shoulder girdle was depressed, the glenohumeral joint was abducted (110°) and laterally rotated, the wrist and fingers were extended, the forearm was supinated and then the elbow was extended.[3] The terminal test position was considered as the end or the extent of joint movement (amplitude) that provoked pain, paresthesia or numbness. In case of pain, a reduction of 5°–10° of elbow extension was allowed for symptoms to vanish and from this new terminal position the therapist performed 10 elbow flexion/extension movements while sustaining the test position for rest of the joints. Four series of 10 tensioning movements at a rhythm of ∼6 s per cycle and 1 min rest between each series were performed. After each cycle of 10 repetitions, the position was held for 10 s. Participants were not aware of which NM technique they received; they were informed that they would receive one of two different NM techniques (sliding or tensioning maneuvers). Six sessions were given every other day for 2 weeks.[18,23]

4.1. *Neural sliding mobilization*

This technique was aimed at increasing the nerve excursion. This technique comprised of two active movements performed simultaneously. (1) Elbow extension of the elbow for loading the median nerve distally and (2) Ipsilateral lateral flexion of the cervical spine for unloading the nervous system proximally. In the supine position, the shoulder was abducted ∼90° and the elbow was flexed to 90°, with the wrist and head/neck in a neutral position. Then, participants actively and simultaneously extended the elbow (to 45°) and side flexed the neck ipsilaterally (to ∼45°) and then returned the elbow to 90° flexion and the neck to 45° of contralateral side flexion while maintaining 90° shoulder abduction. A combination of these movements has been reported to allow the greatest sliding (10.2 mm) of the median nerve.[19] Four series of 10 sliding movements at a rhythm of ∼6 s per cycle and 1 min rest between each series were performed.[18,23]

4.2. *Control group (sham neural mobilization)*

The control group participants received maneuvers mimicking NM in the supine lying position but without any stress on the neural tissues to match therapist contact, participant expectations and the differences in attention time compared to other

groups.[24–28] Accordingly, the head was passively positioned in the neutral position, with the shoulder abducted (∼45°) and externally rotated (∼45°) and elbow flexed (∼45°) with forearm pronation. This was immediately followed by 10 passive wrist flexion/extension movements at a rate of ∼6 s per cycle. At the end of the 10th cycle, a static hold of wrist flexion was sustained for 10 s. The participants in the sham group received the same regimen, six sessions were given every other day for two weeks.

The intervention program was carried out individually by a physiotherapist, with a clinical experience of eight years, who was trained in these techniques. The same therapist was employed for all groups to minimize between-therapist variability, enhance fidelity and maintain the same patient physiotherapist relationship.

Although the treating therapist was not blinded to the treatment method, the participants and outcome assessor were blinded to the random allocation of participants in different groups. Further, neither participants nor the outcome assessor was allowed to discuss about the treatment the participants received during outcome measurement.

4.3. *Outcome measures*

Assessments of the outcome measurements happened at three time points: Pretreatment, immediately after the two weeks of treatment and one week after the last session of the treatment. The third assessment of the outcome measures was to determine the presence of any short-term carryover effects within one-week following the intervention period. The primary outcome measure used to determine the treatment's effect was DSSEPs.

4.4. *DSSEPs*

Neurophysiological findings (peak-to-peak amplitude of DSSEPs for the C6, C7, C8, and T1 levels) were measured in this study. For neurophysiological assessment, an electromyography device (Neuropack S1 MEB-9400K, Nihon Koden, Japan) was used to measure these variables.

DSSEPs were elicited by repetitive, square wave (0.5 ms) electrical pulses (at 3 Hz) delivered by standard surface gel electrodes (20 mm) placed over cervical dermatomes. DSSEPs were recorded at a stimulus intensity that was above the perception threshold of each participant. All participants were lying supine, with eyes closed, and asked to remain quiet and relaxed during the testing procedure. Following preparation of the scalp with Nuprep gel and Ag–AgCl disc recording electrodes (10 mm with 60-in lead wires) were fixed with Elefix paste (Nihon Kohden, Tokyo, Japan). The ground strap electrode was placed around the forearm with an even contact for all recordings. The impedance of the electrodes was kept at $< 5\,k\Omega$. Two complete recording runs were done during each session for all the dermatomes stimulated (C6–T1) with an average of 250–1200 cortical responses from the recording electrodes (C3–C4 in a 10–20 electrode configuration) of the contralateral scalp.[20]

The secondary outcome measures included skin sympathetic response (SSR).

4.5. *SSRs*

Participants were asked to refrain from using medications and cosmetics (on the arms and hands), avoid physical activity on the day of data collection, and also avoid smoking, eating, and drinking coffee at least two hours prior to the recordings. All the participants spent 20 min in a room at a temperature of 22–24°C to acclimatize them to the experimental environment before the measurements.

An electromyography (EMG) equipment was used for measuring the SSR. The skin temperature was maintained at 32°C[29–34] by sustaining the room temperature stable around 26°C, asking subjects to stay in that room for 20 min before the examination and to prevent local warm-up for extremities. Active surface electrodes were placed on the volar side of the hand, and the references were placed on the dorsal side. The stimulus was given at the wrist contralateral to the recording limb; however, measurements were obtained from both sides. An intensity of 20–30 mA with an irregular interval > 1 min was chosen to prevent habituation. If habituation occurred, then stimulation was delayed for approximately three or four minutes. The latency (period from the beginning of stimulus to the onset of response from the baseline) and peak-to-peak amplitude parameters in SSR (recorded waves) were measured manually by the cursors of EMG.

4.6. *Sample size determination*

To estimate the number of participants needed for the trial, estimates of mean and standard

Table 2. 2-way repeated ANOVA table (DSSEPs).

					P		
		Pre	Post	Follow	G	T	G Vs T
C6	Gliding	2.9 ± 0.6	3.1 ± 0.5	3.1 ± 0.5	< 0.001*	< 0.001*	< 0.001*
	Tensioning	3 ± 0.7	2.2 ± 0.4	2.2 ± 0.5			
	Sham Control	2.8 ± 0.8	2.9 ± 0.5	2.9 ± 0.5			
	p-value	0.3	< 0.001*	< 0.001*			
C7	Gliding	2.02 ± 0.5	2.9 ± 0.7	2.8 ± 0.7	< 0.001*	< 0.001*	< 0.001*
	Tensioning	2.2 ± 0.3	1.6 ± 0.4	1.7 ± 0.5			
	Sham Control	2 ± 0.6	2.12 ± 0.5	2.1 ± 0.5			
	p-value	0.4	< 0.001*	< 0.001*			
C8	Gliding	2.2 ± 0.3	2.8 ± 0.6	2.7 ± 0.7	< 0.001*	< 0.001*	< 0.001*
	Tensioning	2.4 ± 0.5	1.7 ± 0.3	2.1 ± 0.4			
	Sham Control	2.4 ± 0.4	2.4 ± 0.3	2.3 ± 0.8			
	p-value	0.1	< 0.001*	< 0.001*			
T1	Gliding	2.4 ± 0.3	3.04 ± 0.9	2.9 ± 0.5	< 0.001*	< 0.001*	< 0.001*
	Tensioning	2.5 ± 0.5	2.08 ± 0.6	2.2 ± 0.6			
	Sham Control	2.7 ± 0.4	2.68 ± 0.5	2.6 ± 0.7			
	p-value	0.3	< 0.001*	< 0.001*			

Notes: Data is expressed as mean ± standard deviation. * = statistically significant difference; G = group; T = time; Pre = baseline value; Post = after 2 weeks; Follow = one week after.

deviations were collected from a pilot trial consisting of 10 participants who received the same program. The mean differences and standard deviation of the peak-to-peak amplitude of DSSEPS for different levels C6, C7, C8, and T1 were registered. The mean differences (±SD) for C6, C7, C8, and T1 were −0.28 (±0.5), −0.1 (±0.7), −0.2 (±0.7), and −0.3 (±0.5), respectively.

These values were used to estimate the sample size separately for each primary outcome by applying a Bonferroni adjustment to the significance level. The largest value was then used as the optimal sample size for the study. Accordingly, at least 25 participants in each group, at a statistical power of 80%, were needed in this study. The sample size was increased by 20% to account for potential dropouts.

4.7. *Data analysis*

Normality of distribution for all data collected was analyzed with the Kolmogorov–Smirnov test. Equality of variance was assessed with the Levene's test. For attaining a 95% confidence level, *p*-value set was < 0.05. The baseline between-group differences in data *b* for the continuous and categorical variables were assessed using one-way ANOVA and $\chi 2$ test, respectively.

A2-way repeated-measures analysis of covariance was used to compare the outcome measures between groups (Table 2). The model included one independent factor (three groups), one repeated measure (three time-points: Pre-intervention, post-intervention at two weeks and follow-up at three weeks), and an interaction factor (group × time). A *post hoc* analysis using Bonferroni correction was performed if significant interactions were found ($p < 0.05$). The baseline values of DSSEPs and SSRs were used as covariates to assess the between-group differences, to center the baseline covariates, everyone's score value was subtracted from the overall mean.

5. Results

A flow chart of participant's randomization and retention throughout the study is shown in Fig. 1. The initial sample included 120 volunteers. 20 participants (16.6% of the total initial sample) did not meet the inclusion criteria or had one of the exclusion criteria, while 10 participants (8%) refused to participate. Ninety participants met the study's inclusion criteria. In total, 90 (100%) participants completed the entire study. All three groups were similar at the baseline for all variables compared without any significant differences (Table 1).

5.1. *Compliance*

Participants in the NM sliding group and the NM tensioning group attended 98% and 96% of the sessions, respectively. Participants in the sham NM group attended 95% of the sessions. The missed sessions were due to an illness of them (51%) or their family member (49%). None of the participants reported any adverse effects with the NM techniques used.

5.2. *Neurophysiological outcomes*

A 2-way repeated measures ANOVA revealed a significant group x time effect on C6, C7, C8, and T1 for DSSEPs and SSRs ($p < 0.001$) (Tables 2 and 3). The *post hoc* analysis indicated that tensioning NM significantly decreased the DSSEPs amplitudes for C6, C7, and C8, and T1 by 27%, 27%, 29%, and 16%, respectively ($p < 0.05$) from the baseline measurements to the post intervention measurements. For the same time interval, neural sliding significantly increased the DSSEPs amplitudes for C6, C7, and C8, and T1 by 7%, 44%, 27%, and 27%, respectively. The control group had an increase in DSSEPs for C6, C7, C8, and T1 of 4%, 6%, 0%, and a decrease of 1%, respectively (Tables 2–4) (Figs. 2 and 4). From the first intervention to the follow-up measurements, the tensioning group showed a decrease in the amplitude of DSSEPs for C6, C7, C8, and T1 of 0%, 3%, 4%, and 5%, respectively. While the sliding group showed an increase of 0%, 6%, 24%, and 6%, respectively, for the same time interval. The control group decrease in amplitude ranged between 0% and 4%.

Sympathetic skin responses in the gliding NM group showed lower amplitudes and prolonged latencies post-treatment when compared to tensioning NM group ($p < 0.05$) (Table 3). The gliding group had a 25% decrease in the SSR amplitude from the baseline to post-treatment measurements and an increase of 6% from the post-treatment to the follow-up measurements. The latency increased by 30% from the baseline to the post-treatment measurement and decreased by 6% from the post-treatment to the follow-up measurement. In the tensioning group, the

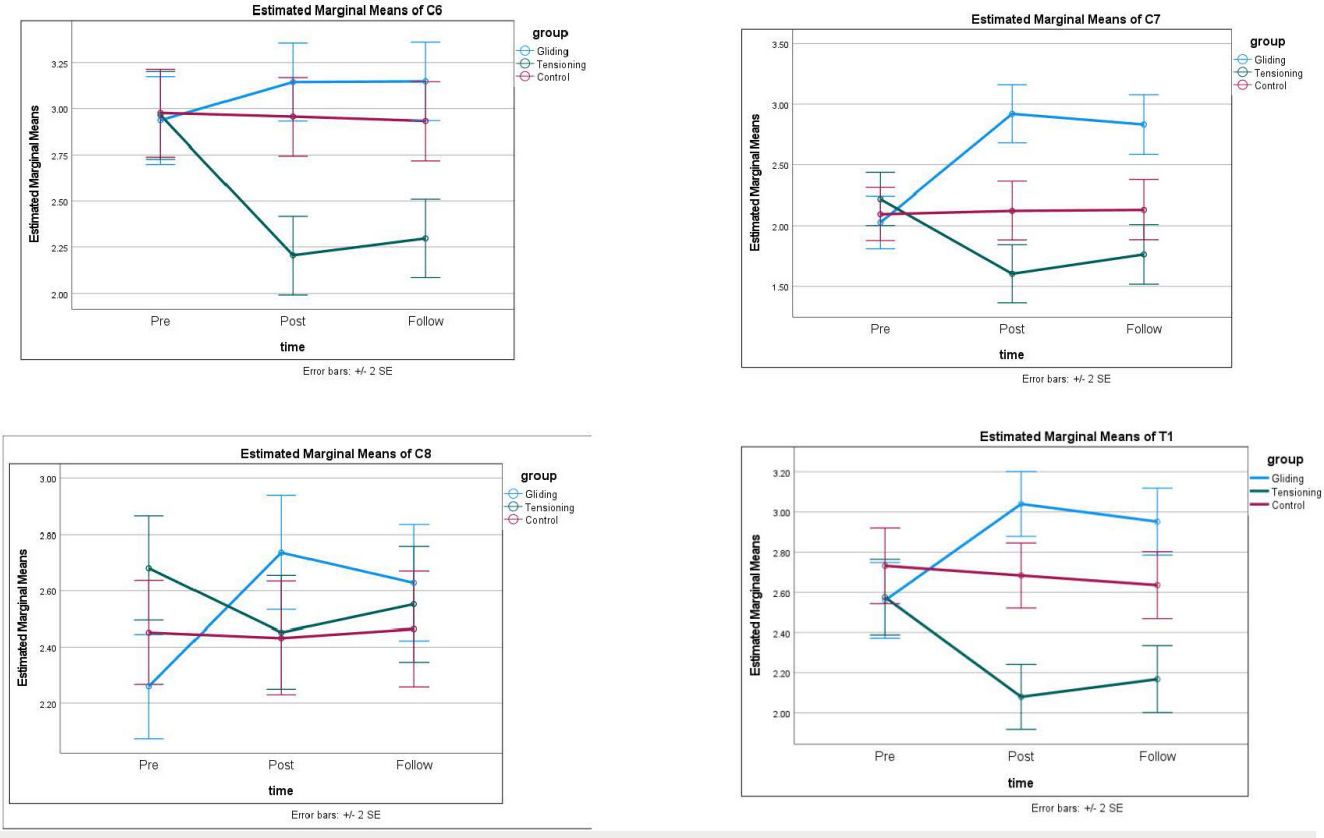

Fig. 2. Estimated marginal means of DSSEP for the dermatomes C6, C7, C8, and T1. DSSEP: Dermatomal Somatosensory Evoked Potential.

Table 3. 2-way repeated ANOVA table (SSR).

					P-value		
		Pre	Post	Follow	G	T	G vs. T
SSR amplitude	Gliding	2.4 ± 0.3	1.8 ± 0.5	1.9 ± 0.6	< 0.001*	< 0.001*	< 0.001*
	Tensioning	2.1 ± 0.4	2.9 ± 0.3	2.9 ± 0.5			
	Sham Control	2.09 ± 0.4	2.2 ± 0.7	2.1 ± 0.6			
	p-value	0.4	< 0.001*	< 0.001*			
SSR latency	Gliding	1.3 ± 0.3	1.7 ± 0.3	1.6 ± 0.4	< 0.001*	< 0.001*	< 0.001*
	Tensioning	1.4 ± 0.3	0.9 ± 0.4	1.01 ± 0.2			
	Sham Control	1.2 ± 0.5	1.2 ± 0.4	1.2 ± 0.3			
	p-value	0.3	< 0.001*	< 0.001*			

Notes: Data is expressed as mean ± standard deviation. * = statistically significant difference; G = group; T = time; Pre = baseline value; Post = after 2 weeks; Follow = one week after. SSR: Skin sympathetic response.

Table 4. Post hoc analysis matrix

						95% Confidence interval	
(I) groups		(J) groups	Mean difference (I-J)	Std. error	Sig.	Lower Bound	Upper Bound
C6	Control	Gliding	−0.32*	0.15	0.002	−0.50	0.25
		Tensioning	0.46*	0.14	0.010	0.08	0.84
C7	Control	Gliding	−0.47*	0.14	0.005	−0.83	−0.11
		Tensioning	0.70*	0.18	0.001	0.25	1.15
C8	Control	Gliding	−0.62*	0.12	0.000	−0.93	-0.31
		Tensioning	0.45*	0.12	0.002	0.14	0.76
T1	Control	Gliding	−0.16	0.10	0.4	−0.45	0.11
		Tensioning	0.40*	0.11	0.002	0.12	0.69
SSR Amp	Control	Gliding	0.58*	0.11	0.000	0.3244	0.8516
		Tensioning	−0.52*	0.10	0.000	−0.7849	−0.2578
SSR Lat	Control	Gliding	−0.32*	0.07	0.001	−0.52	−0.11
		Tensioning	0.44*	0.08	0.000	0.24	0.64

amplitude increased by 38% from the baseline to the post treatment measurement, with no change at the follow-up. The latency decreased by 35% from the baseline to the post-treatment and increased by 12% from post-treatment to the follow up measurements. In contrast, no significant changes were observed in SSRs for participants in the sham treatment group; the changes ranged between 0% and 5% ($p > 0.05$) (Tables 3 and 4) (Fig. 3).

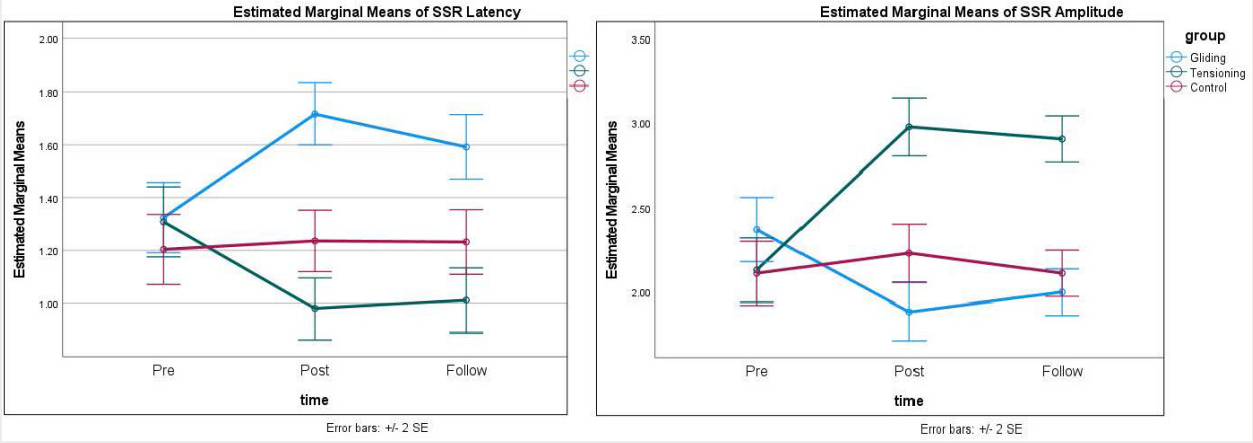

Fig. 3. Latency and peak-to-peak amplitude of SSR. SSR: Skin Sympathetic Response.

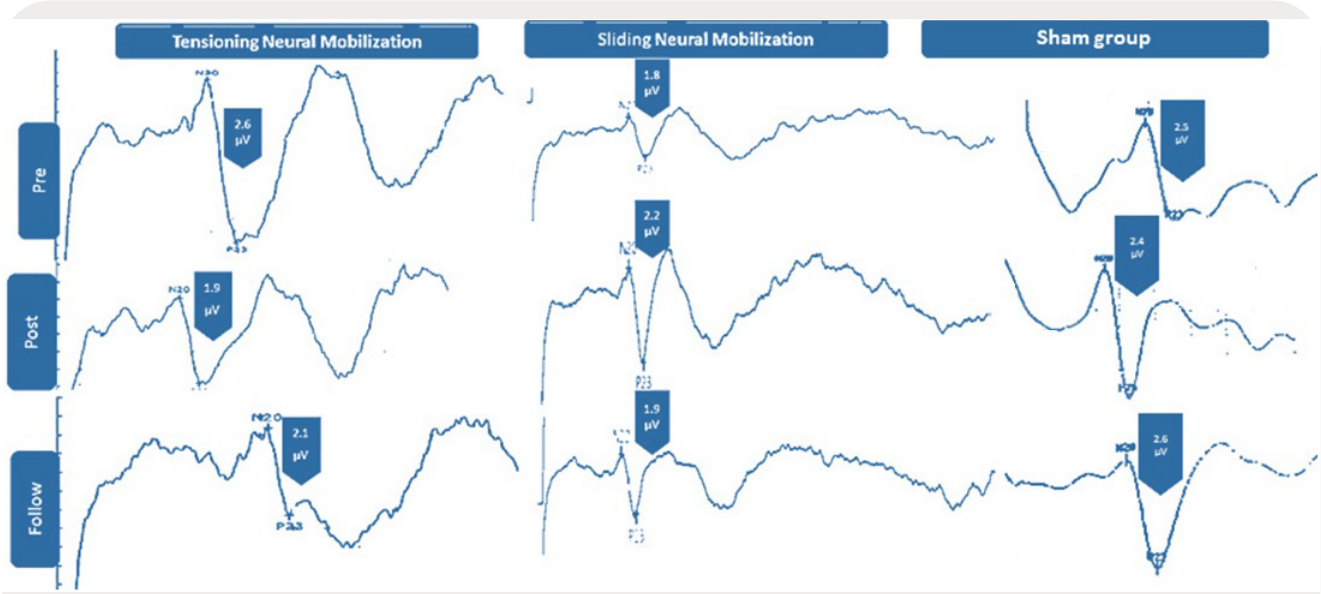

Fig. 4. Illustrative example for DSSEPs after neural mobilization.

6. Discussion

The differences between groups indicate that different NM techniques had a different effect on DSSEPs. Thus, our study's primary hypothesis is confirmed by these findings. With regard to the neurophysiological parameters, gliding NM showed greater improvement in the peak-to-peak amplitudes of C6, 7, 8, and T1 when compared to neural tensioning in asymptomatic participants. Furthermore, gliding NM positively affects SSRs and these positive impacts lasted for at least one week.

Based on our results, gliding mobilization was more effective compared to tensioning with respect to the neural function represented by the evoked potentials (DSSEPs) and autonomic nervous function (SSRs). To our knowledge, this study is the first of its kind to present objective evidence on two different NM techniques' effect on these specific neurophysiological parameters.

In this study, we have chosen the DSSEPs as it has been found to be a better indicator of segmental nerve root function and associated clinical symptoms compared to other electrophysiological assessments.[15,16] As impaired conduction is always manifested by amplitude reduction,[35] the peak-to-peak amplitude was selected as reflection of the nerve root function in this study. Specially, more recent studies have indicated that the amplitude of single root DSSEPs may provide useful information about rootlet and root dysfunction.[36,37] Regarding the autonomic function assessment, there is a lack of consensus in the research studies investigating SSR about using qualitative or quantitative analysis. However, the majority of the previous studies support the quantitative evaluation where prolonged latency and decreased amplitude of SSR reflect the sympathetic dysfunction.[38]

There is a dearth of studies comparing the effects of neural gliding and neural tensioning techniques. This might be because neural gliding is a much more recent technique than neural tensioning.[39] Moreover, the majority of these comparisons were limited to measurement of pain, thermal, and mechanical sensitivity.[40–43]

Accordingly, this study cannot be directly compared to the results of others due to the differences in methodology, populations, and outcomes that were measured. Still, the novel result of this study is that tensioning NM significantly decreases the peak-to-peak amplitude of the DSSEPs tested in addition to increase the amplitude of the SSRs. An increase in latency and a decrease in amplitude of SSRs after gliding technique indicate that neural sliding has a sympathetic inhibitory effect.

One of the primary mechanical mechanisms that could lead to a neurologic dysfunction is an increase in longitudinal tension in the nerve root. Especially, for the nerve root, the maximal elastic limit and failure strain occurred at lower loads compared to the peripheral nerve.[44]

The strain or stress that occurs along the course of the nerve as a result of upper extremity joint

motion may also be considered as an etiological reason for neural dysfunction. This tension within the nerve can affect intraneural blood flow and nerve function.[45,46]

Lundborg and Rydevik.[47] determined that even the lower limits of strain 5(–10%) demonstrated the first signs of changes in blood flow in the epineural and perineurial vessels. The axoplasmic flow may also be considered when utilizing NM (gliding or tensioning) techniques. It has been found that neural axoplasm has thixo tropic properties, which indicates that lack of motion or increased sustained strain will increase the viscosity of fluid flow and may slow or impair neural transport mechanisms.

This idea of relating the dynamic forces applied on the neural tissue to the impairment seen in the neural function agrees with the concept described by Brieg.[48] He proposed that the neural tissue after being subjected to longitudinal tensile stresses, the nerve root sleeves unfold and become strained, and the blood supply is decreased with a reduced lumen cross-sectional area.

The current autonomic related findings contrast with earlier study of Kornberg and McCarthy, which showed that neural stretching technique has a sympathetic inhibitory effect.[39] This study cannot be directly compared to the results of Kornberg and McCarthy because of the differences in methodology and populations that were tested. This study investigated sympathetic outflow to the lower limbs. Moreover, it was concerned with the immediate effect of NM after holding the stretching position for only 7 s.

7. Study Limitations

We propose several limitations of this study pointing to future works. First, our project only included a short-term follow-up of one week after the intervention was terminated; it is unclear how long the identified neurophysiological changes would remain. Second, because our study population was composed of young and asymptomatic healthy adults, it is unknown how our study results might be related to musculo-skeletal pain populations. However, rehabilitation programs guided by patho-anatomy and functional disturbances would theoretically benefit and perhaps have greater success in the treatment of symptomatic cases if nerve root and autonomic function may be improved by adding neural gliding to the clinical armamentarium of treatment methodology.

8. Conclusions

This study showed that sliding NM was optimal in achieving neurophysiological benefits and minimizing the negative effects on the neural function of the involved nerve roots and autonomic nervous function. A tensioning NM had a negative effect on the neurophysiology variables of the nerves involved in the NM. Although these results cannot be generalized to the general population, considering the healthy asymptomatic participants included in this study, these results should assist in selecting the optimal NM technique in order to achieve the well-established goals from NM, while avoiding any negative effects on neural function.

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Conflict of Interest

The authors declare that they have no competing interests.

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Author Contributions

All the authors contributed to the conception and design of study and approval for the submission to publication. Budour Yousif and Ibrahim Moustafa contributed to the data analysis and interpretation. Ashokan Arumugam and Amal Ahbouch made substantial contributions to the drafting of the paper as well as implementation, analysis, and interpretation of data.

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