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ORIGINAL ARTICLE/ARTICLE ORIGINAL

Therapeutic effects of peripheral magnetic stimulation on traumatic brachial plexopathy: Clinical and neurophysiological study

Étude clinique et neurophysiologique des effets thérapeutiques de la stimulation magnétique périphérique en cas de plexopathie brachiale traumatique

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KEYWORDS

Therapeutic magnetic stimulation; Brachial plexopathy; Pain

Summary

Objective. – To evaluate the therapeutic effects of peripheral repetitive magnetic stimulation (rMS) on recovery of traumatic brachial plexopathy.

Patients and methods. – Thirty-four patients with traumatic brachial plexopathy were studied. Strength of different muscles of upper limbs was evaluated neurologically. Nerve conduction studies (NCS), upper limb F-waves and visual analogue scales (VAS) for shoulder pain were obtained for all patients. These were randomly assigned into two groups with a ratio of 2:1; each patient received conventional physical therapy modalities and active exercises as well as real or sham rMS applied over the superior trapezius muscle of the affected limb daily for 10 sessions. Patients were reassessed with the same parameters after the 5th and the 10th session, and 1 month after rMS treatment.

Results. – No significant between-group differences were recorded at baseline assessment. Significant improvement was observed (time X groups) after real rMS in comparison to the sham group (P = 0.0001 for muscle strength and 0.01 for VAS of shoulder pain). These improvements were still present at 1 month after the end of treatment. In accordance with the clinical improvement, a significant improvement was recorded in the neurophysiological parameters in the real vs the sham group.

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Conclusions. — We demonstrate that peripheral rMS for 10 sessions may have positive therapeutic effects on motor recovery and pain relief in patients with traumatic brachial plexopathy. Therefore, it is a useful adjuvant in the therapy of these patients. © 2011 Elsevier Masson SAS. All rights reserved.

Résumé

But de l'étude. – Évaluer les effets de la stimulation magnétique répétitive (SMR) périphérique sur la récupération d'une plexopathie brachiale traumatique.

Patients et méthodes. – L'étude porte sur 34 patients atteints de plexopathie brachiale traumatique. La force de différents muscles des membres inférieurs a été mesurée cliniquement. Nous avons obtenu, chez tous les patients, une mesure des conductions nerveuses incluant celle des ondes F des membres supérieurs ainsi qu'une échelle visuelle analogique (EVA) des scapulalgies. Les patients ont été aléatoirement distribués en deux groupes selon une proportion 2:1; chaque patient a bénéficié d'une prise en charge physiothérapeutique conventionnelle incluant des exercices de mobilisation active et a suivi dix sessions au cours desquels une SMR réelle ou fantôme était appliquée sur le muscle trapèze du membre atteint. Les mêmes paramètres ont été évalués chez les patients après la cinquième et la dixième session et un mois après la SMR. Résultats. – La ligne de base ne différait pas entre les deux groupes. Une amélioration significative fut observée après la SMR réelle par comparaison à la SMR fantôme (p = 0,0001 pour la force musculaire et 0,01 pour l'EVA). Cette amélioration était toujours manifeste un mois après le traitement. Parallèlement à l'amélioration clinique, une amélioration significative des paramètres neurophysiologiques fut observé après SMR réelle par opposition à la SMR fantôme. Conclusions. – Dix sessions de SMR périphérique peuvent avoir un effet favorable sur la récupération motrice et l'atténuation de la douleur en cas de plexopathie brachiale traumatique. La SMR périphérique peut, dès lors, constituer une thérapie adjuvante utile chez ces patients.

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MOTS CLÉS Stimulation

magnétique thérapeutique ; Plexopathie brachiale ; Douleur

Introduction

Brachial plexopathy is a common complication of traffic accidents. It is characterized by brachial neuralgia and upper limb weakness. One approach to treatment of the peripheral pain consists of repetitive electrical stimulation of peripheral nerve; however, deep structures are difficult to activate due to local discomfort at the site of stimulation. Single and repetitive pulse magnetic coil stimulation (rMS) can activate deeper neural structures without causing irritation and has been successfully applied to reduce musculoskeletal pain for several days [14]. The mechanism of action is unclear, although it may be similar to transcutaneous electrical nerve stimulation (TENS) with actions at both peripheral and/or central levels of the nervous system. For example, it has been proposed that TENS could cause slowing of conduction in both small and large afferent nerve fibers [19,23]. Kaelin-Lang et al. [6] concluded that TENS elicits focal increase of cortico-motorneuronal excitability outlasting the stimulation period and probably occurring at cortical sites.

Struppler et al. [21,22] found that rMS could reduce spasticity and improve perception of joint position in stroke patients. Heldmann et al. [5] found that prolonged peripheral rMS could modulate the response of primary and secondary somatosensory cortices to afferent input. Recent studies on healthy subjects demonstrated that somatosensory input produced by peripheral nerve stimulation or muscle stretch can produce a lasting increase in cortico-motorneuronal excitability of the stimulated body parts [15]. Thus, peripheral mixed nerve stimulation may evoke conjoint activity of somatosensory afferents and intrinsic motor cortical circuits. Such combination seems particularly effective in modulating motor output, as shown by the fact that median nerve stimulation paired with transcranial magnetic stimulation can lead to lasting changes in excitability of motor cortex [20].

The aim of this study was to evaluate the therapeutic effects of peripheral rMS on pain relief and motor recovery as an adjuvant therapy in patients with traumatic brachial plexopathy.

Patients and methods

Neurophysiological measurements

Ulnar and median nerve motor conduction velocities, distal latencies and compound muscle action potentials (CMAP) amplitudes were measured with standard surface stimulating and recording electrodes in both affected and unaffected arms. For the axillary and suprascapular nerves, the technique described by Gassel [4] was used for measuring motor nerve conduction time (latency) to the deltoid and suprascapular muscles, respectively, using a concentric needle as the recording electrode. A concentric needle electrode was placed in the middle of the biceps, deltoid, and supraspinatus muscles.

The brachial plexus was stimulated with bipolar surface electrodes at Erb's point (a few centimeters above the clavicle in the angle between the posterior border of the sternomastoid muscle and the clavicle at the level of the 6th cervical vertebra). Latency values obtained with anodal and cathodal stimulation were averaged to calculate the final value. The normal limits of motor conduction velocities, distal latencies and conduction times were set at +2 SD from the mean values of the healthy arm of the same group of patients. The CMAP was considered abnormal if the peakto-peak amplitude was below the lowest value found in the healthy arm.

F-waves from both upper limbs were recorded to median and ulnar nerves supramaximal stimulation at the wrist using surface electrode at thenar and hypothenar eminences, respectively. The ground electrode was placed at the forearm. Twenty trials for each nerve were recorded; the mean F-wave latency was measured. A 1.5-ms Fwave latency difference between both arms was considered abnormal.

Skin temperature was controlled. Recordings were performed with a Nihon Kohden equipment (model 7102), with the following parameters: sweep time 8 ms/D, sensitivity 0.5 mV/D, low frequency 2 Hz, high frequency 10 Hz, stimulation duration 0.1 ms, stimulation frequency 1 Hz.

Magnetic stimulation

Resting motor threshold of the 1st dorsal interosseous muscle (FDI) of the unaffected limb was measured for each patient using a Magstim Super Rapid (Magstim, Whitland, UK) stimulator connected to a 90-mm outer diameter figure-of-eight coil. First, we determined the optimal scalp location by moving the figure-of-eight coil systematically in 1-cm steps in order to determine the scalp position from which transcranial magnetic stimulation (TMS) (constant suprathreshold intensity) evoked motor potentials of maximum peak-to-peak amplitude in the target muscle. The coil was positioned tangentially to the scalp and oriented so that the induced electrical currents would flow approximately perpendicular to the central sulcus, at a 45° angle from the mid-sagittal line. Single-pulse TMS was then delivered to the optimal location, starting at suprathreshold intensity and decreasing in steps of 2% of the stimulator output. Relaxation and EMG signals were monitored for 20 ms prior to stimulation. Resting motor thresholds was defined as the minimum output of the stimulator that induced reliable MEPs (amplitude of $50 \,\mu V$ and $200 \,\mu V$ at rest or during weak voluntary contraction, respectively) in at least five of 10 consecutive trials in the FDI muscle.

Patients

We conducted this study in the Department of Neurology in collaboration with the Department of Rheumatology and Rehabilitation at Assiut University Hospital, Assiut, Egypt. The study included 40 consecutive patients (28 males) with traumatic weakness of one upper limb. These were recruited from the outpatient clinics of Rehabilitation and Orthopedic, during the period from March 2005 to December 2010. The mean age was 37.2 ± 14.1 years (range: 16 to 59 years). All patients had been treated after trauma with conventional physical therapy, muscle strengthening exercises and medications, including anticonvulsants, narcotic or non-narcotic analgesics, without any satisfactory pain control or improvement in muscle strength for at least one and half month. Exclusion criteria were: patients with open injury, fractures, dislocation, head trauma, tendon tears of shoulder joint, severe limb paralysis and wasting of the muscles, in which no evoked potential could be recorded. Four cases were excluded due to the presence of associated tendon tear and fracture in the shoulder (Fig. 1: flow chart). Out of the remaining 36 patients, 20 presented with right and 16 with left brachial plexopathies. Traffic accident (16 patients) was the most common cause followed by lifting heavy objects on the shoulder (10 patients), direct trauma, object striking shoulder or falling from a height (six patients), and postoperative arm traction during general anesthesia (four patients). Mean illness duration was 7.8 ± 2.1 weeks (range: 6 to 12 weeks). This study was approved by the local ethical committee of Assiut University Hospital. Written informed consent was obtained from all of the subjects.

The strength of different upper limb muscles was neurologically assessed in each patient using the Medical Research Council Scale [12] and each patient was asked to score shoulder pain using a visual analogue scale (VAS) [13].

The patients were randomly classified into one of the two groups with a 2:1 ratio, using closed envelopes. The first group (24 patients) received both physical therapy (electrical stimulation, ultrasound, heat therapy and therapeutic as well as active exercises) and real rMS. Physical therapy aimed at alleviating pain, maintaining range of motion (ROM), and optimizing motor-function recovery at the time of muscle reinervation. Therapeutic exercises gradually progressed from passive to active ROM, as tolerated. The second group (12 patients) received the same physical therapy with sham rMS.

Two types of real rMS were used. The first one (''7 Trains'') was designed to relieve shoulder pain: stimulation at motor threshold, 15 Hz, 10 seconds per train with an 20second inter-train interval for a total of 1050 pulses. The second one ("50 Trains") was designed to increase strength: stimulation at 70% of the motor output, sufficient to give rise to arm contraction, 3 Hz, 10 seconds per train with a 30-second inter-train interval, for a total of 1500 pulses. A 10-minute rest period was observed between both types of stimulation. Both were daily applied over the superior trapezius muscle (10 sessions, five sessions/week). The same parameters were used for sham rMS and real rMS but the coil was elevated away from the trapezius muscle (not touching the patient). The magnetic stimulator (Magstim model 200; Magstim, Whitland, UK) was connected to a 70-mm outer diameter figure-of-eight coil, which resulted in a maximal output of 2.2 Tesla. Two patients in the real group were lost to follow-up, so that 34 patients completed the study (Fig. 1).

Patients were evaluated before rMS, after the 5th session and the 10th session, and at 1 month, using VAS for assessment of shoulder pain and rating scale for strength of power. Neurophysiological assessment was performed both before and after the series of sessions. Patients were not aware of the type of stimulation. The investigator who was responsible for clinical and neurophysiological follow-up was blind to the type of treatment. However, the investigator who assessed muscle strength was aware of the type of stimulation.





Data analysis

Pain level and muscle strength at baseline, 5th, 10th and after 1 month were evaluated using a 2-factor analysis of variance (Anova) ("time factor" as the main factor pre, 5th, 10th, 1 month) and "session type" (real vs sham), for both rating scores (muscle strength and VAS). Paired *t* test was used to evaluate the pre- vs post-changes in neurophysiological parameters. The Greenhouse–Geisser correction of

degrees of freedom was used whenever necessary to correct non-sphericity. Spearman's correlation between the degree of improvement in muscle strength and VAS (pre-rMS-1 month after) were calculated for the different muscles.

Results

Table 1 shows the demographic and clinical data of the patients at admission. Weakness was more pronounced in

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Table 1	Demographic and clinical	data of the studied pat	tients (that complete the f	follow-up) at baseline assessment.

Variable	Real group n = 22	Sham group n = 12	P value
Age mean (SD) (years)	33.9 (11.1)	30.9 (11.70)	0.197
Sex (Male/Female)	18/4	10/2	NS
Duration of illness (weeks) mean (SD)	7.8 (2.2)	8.4 (2.5)	0.611
Affected arm (Right/Left)	12/10	6/6	NS
Causes			
Motor car accident	10	6	
Lifting heavy objects	5	3	NS
Postoperative traction to the limb	3	1	
Direct trauma	4	2	
Strength of the muscles mean (SD)			
Hand grip strength	3.9 (0.9)	3.6 (1.2)	NS
Elbow flexor (biceps muscle)	3.6 (0.9)	3.9 (0.9)	NS
Elbow extensor (triceps muscle)	3.9 (0.9)	3.8 (1.0)	NS
Deltoid muscle	2.5 (1.4)	2.3 (1.1)	NS
Supraspinatus muscle	2.6 (1.3)	2.3 (1.1)	NS
NS: non-significant.	2.0 (1.3)	2.5 (1.1)	113

 Table 2
 Neurophysiological data of the studied patients (34 patients) at base line assessment.

	Normal arm (34 arms)	Affected arm	Affected arm	P value Between both groups
		Real group (22 arms)	Sham group (12 arms)	
Median nerve (mean \pm SD)				
Distal latency (ms)	3.7 ± 0.5	$\textbf{3.7}\pm\textbf{0.6}$	3.6 ± 0.5	0.93
NCV (m/s)	56.5 ± 6.8	53.1 ± 6.2	$\textbf{56.4} \pm \textbf{5.8}$	0.95
CMAP amplitude (μV)	10.1 ± 4.3	$\textbf{6.8} \pm \textbf{4.6}$	$\textbf{6.1} \pm \textbf{3.1}$	0.23
F-wave latency (ms)	$\textbf{27.5} \pm \textbf{2.9}$	$\textbf{29.6} \pm \textbf{3.7}$	$\textbf{28.7} \pm \textbf{2.6}$	0.36
Ulnar nerve (mean \pm SD)				
Distal latency (ms)	$\textbf{2.6} \pm \textbf{0.6}$	$\textbf{2.9} \pm \textbf{0.6}$	2.5 ± 0.4	0.2
NCV (m/s)	56 ± 6.6	$\textbf{54.8} \pm \textbf{9.3}$	$\textbf{54.8} \pm \textbf{13.5}$	0.27
CMAP amplitude (μ V)	8.9 ± 3.7	$\textbf{6.8}\pm\textbf{3.7}$	$\textbf{8.7} \pm \textbf{8.2}$	0.09
F-wave latency (ms)	$\textbf{28.8} \pm \textbf{2.7}$	$\textbf{29.8} \pm \textbf{3.0}$	$\textbf{34.5} \pm \textbf{12.8}$	0.53
Axillary nerve (mean \pm SD)				
Conduction time (ms)	3.5 ± 0.7	$\textbf{5.5} \pm \textbf{2.9}$	5.9 ± 3.4	0.3
CMAP amplitude (μ V)	$\textbf{8.3}\pm\textbf{6.1}$	$\textbf{2.5}\pm\textbf{3.6}$	$\textbf{2.4} \pm \textbf{4.4}$	0.4
Musculocutaneous nerve (mean \pm SD)				
Conduction time (ms)	4.1 ± 0.8	6.1 ± 2.8	5.1 ± 1.8	0.4
CMAP amplitude (μ V)	$\textbf{8.2}\pm\textbf{5.3}$	$\textbf{3.9} \pm \textbf{5.1}$	$\textbf{3.3}\pm\textbf{3.2}$	0.1
Suprascapular nerve (mean \pm SD)				
Conduction time (ms)	$\textbf{2.7} \pm \textbf{0.5}$	$\textbf{4.7} \pm \textbf{0.8}$	$\textbf{4.7} \pm \textbf{1.5}$	0.74
CMAP amplitude (µV)	$\textbf{6.9} \pm \textbf{4.9}$	$\textbf{2.1} \pm \textbf{4.6}$	$\textbf{2.9} \pm \textbf{1.9}$	0.1

muscles supplied by the upper trunk of the brachial plexus (deltoid, supraspinatus and biceps muscles) followed by triceps and hand grip muscles. Table 2 confirms that the neurophysiological abnormalities were more severe in axillary, suprascapular and musculocutaneous nerves (upper trunk of brachial plexus) followed by median nerve and the ulnar nerve as the least affected one. Peripheral rMS was well-tolerated by all patients, without any adverse effects.

Effect of treatments on muscle strength

The mean rating score of muscle strength increased over the four times of assessment in the real rMS group, for all



Figure 2 Changes in mean power rating scores of hand grip (A), elbow flexion (B), and deltoid (C) at the four assessment points (pre, 5th, 10th, 1 month). The first assessment was performed immediately prior to a 10-session repetitive magnetic stimulation (rMS) treatment, the 2nd and 3rd assessments immediately after the 5th and 10th sessions of transcranial magnetic stimulation of the brain (rTMS), respectively, and the last assessment one month after the end of sessions. There were no significant between-group differences in muscle strength at baseline assessment, while a significant improvement was observed over the course of the real repetitive magnetic stimulation (rMS) treatment as compared to the sham group (P=0.03, for hand grip, and 0.0001, for elbow flexor and deltoid muscles).

	Real group (nine cases)		Sham group (six cases)			
	Pre-sessions	Post-sessions	P value	Pre-sessions	Post-sessions	Paired t test
Median nerve (mean \pm SD)						
Distal latency (ms)	$\textbf{3.5} \pm \textbf{0.33}$	$\textbf{2.85} \pm \textbf{0.01}$	0.002	3.6 ± 0.5	$\textbf{3.65} \pm \textbf{0.01}$	0.85
NCV (m/s)	59.5 ± 5.5	$\textbf{57.3} \pm \textbf{2.8}$	0.13	$\textbf{53.8} \pm \textbf{4.9}$	54.3 ± 3.3	0.83
CMAP amplitude (μ V)	7.2 ± 3.0	14.5 ± 2.5	0.04	$\textbf{6.6} \pm \textbf{3.7}$	$\textbf{6.56} \pm \textbf{3.3}$	0.92
F-wave latency (ms)	$\textbf{27.3} \pm \textbf{0.8}$	$\textbf{25.4} \pm \textbf{1.6}$	0.006	$\textbf{29.7} \pm \textbf{0.8}$	$\textbf{29.0} \pm \textbf{2.1}$	0.21
Ulnar nerve (mean \pm SD)						
Distal latency (ms)	$\textbf{2.6} \pm \textbf{0.34}$	2.2 ± 1.8	0.08	$\textbf{2.5} \pm \textbf{0.5}$	2.5 ± 1.8	0.63
NCV (m/s)	$\textbf{56.9} \pm \textbf{9.4}$	$\textbf{58.2} \pm \textbf{9.3}$	0.109	$\textbf{56.9} \pm \textbf{9.4}$	$\textbf{58.2} \pm \textbf{9.3}$	0.08
CMAP amplitude (μ V)	13.4 ± 8.6	15.5 ± 8.9	0.123	5.3 ± 2.6	5.2 ± 2.7	0.64
F-wave latency	$\textbf{29.4} \pm \textbf{4.2}$	$\textbf{26.2} \pm \textbf{2.8}$	0.58	$\textbf{30.7} \pm \textbf{3.6}$	$\textbf{29.9} \pm \textbf{3.2}$	0.03
Axillary nerve (mean \pm SD)						
Conduction time (ms)	$\textbf{7.98} \pm \textbf{4.1}$	$\textbf{4.1} \pm \textbf{0.78}$	0.028	6.1 ± 3.8	6.3 ± 4.5	0.51
CMAP amplitude (μV)	$\textbf{0.48} \pm \textbf{0.11}$	$\textbf{1.6} \pm \textbf{0.88}$	0.009	$\textbf{0.5}\pm\textbf{0.12}$	$\textbf{1.1} \pm \textbf{0.85}$	0.06
Musculocutaneous nerve (mean \pm SD)						
Conduction time (ms)	5.2 ± 1.6	$\textbf{3.8}\pm\textbf{0.4}$	0.04	5.6 ± 1.6	5.4 ± 1.6	0.29
CMAP amplitude (μV)	$\textbf{4.08} \pm \textbf{3.5}$	$\textbf{4.9} \pm \textbf{0.95}$	0.16	4.08 ± 3.5	$\textbf{4.9} \pm \textbf{0.95}$	0.88

Table 3 Neurophysiological parameters (pre- and post-sessions) of nine cases of real and six cases of sham groups.

CMAP: compound muscle action potentials; NCV: nerve conduction velocity.

muscles. This was evident for the deltoid: P < 0.0001, F = 91, df = 1.6 (33), supraspinatus, P < 0.0001, F = 102, df = 2.1 (44), elbow flexors P < 0.0001, F = 45, df = 1.5 (31), elbow extensors P < 0.0001, F = 39, df = 1.3 (27.7), and hand grip muscles P < 0.0001, F = 44.1, df = 1.3 (27.6). In the sham group,

there was a significant improvement in hand grip only, while the other muscles showed a non-significant improvement: P < 0.01, F = 5.8, df = 1.5 (18) for hand grip, P = 0.10, 0.51, 0.06 and 0.6 for elbow flexor, extensor, deltoid, and supraspinatus muscles respectively. Fig. 2A, B and C



Figure 3 Changes in mean pain rating scores (Visual Analogue Scale [VAS]) over the course of the treatment. The Visual Analogue Scale improvement is significantly more marked in the real vs sham repetitive magnetic stimulation (rMS) group (P = 0.0001).

illustrate changes in the mean scores of hand grip, elbow flexion, and deltoid muscles at each assessment time (pre, 5th, 10th, 1 month).

Improvements were statistically significant at the second and third assessment points (pre vs 5th and 5th vs 10th) P=0.001, and 0.0001 respectively for all muscles except the elbow extensor (P=0.15 5th vs 10th). At 1 month, the improvements were stationary with no significant changes (10th vs 1 month) in any muscle except the deltoid muscle (P = 0.04).

Effect of treatment on neurophysiological parameters

Nine cases in the real group and six cases in the sham group were neurophysiologically re-evaluated after the last session. Paired t test showed that the mean value of the neurophysiological parameters in each nerve was significantly improved after real rMS especially for the axillary, median and musclucutaneous nerves. No similar changes were recorded in the sham group except for a small improvement in ulnar nerve parameters. Details are provided in Table 3.

Pain assessment

A significant improvement in VAS was noticed during the course of treatment: P < 0.0001, F = 27 and df = 1 (21) for both the real and the sham group: P < 0.01, F=6.1 and df = 1.6 (24). However, a two-way Anova time X groups showed that the improvement was significantly greater in the real group in comparison to the sham group: P < 0.0001, F = 25.9 and df = 2.1 (66). This improvement also persisted until 1 month (Fig. 3). There was a significant correlation between the degree of improvement in muscle strength and VAS (pre-rMS-1 month after) (P=0.001 for all muscles) in the real group.

Discussion

This preliminary study demonstrates that 2 weeks of real rMS combined with conventional physical therapy can improve pain and muscle strength in cases of brachial plexopathy. Noteworthy, these changes started during the peripheral rMS therapy and improvement continued during the following month. Pain improvement paralleled the progressive increase in muscle strength.

The advantage of rMS over conventional therapeutic electrical stimulation lies in its ability to stimulate deep structures without the local discomfort that is produced by high intensities of electric stimulation [1]. That is to say that brachial plexus activation was readily obtained in all patients.

The mechanisms of the response to rMS are still uncertain, and, objective effects could occur at both spinal and supraspinal levels. There could be a direct effect on pain, with a secondary increase in muscle strength as patients start to increase their use of the affected limb. Conversely, there could be a direct effect on strength with a secondary effect on pain. Finally, there could even be a direct effect of rMS on both pain and strength.

Muscle strength

In this study, we applied a mixture of sub-motor threshold stimulation, in order to activate only sensory afferents, together with supra-motor threshold stimulation, in order to evoke clear muscle contraction through stimulation of the efferent nerve. As noted by Struppler et al. [21], the latter will activate sensory afferent fibers by direct depolarization as well as indirectly via the muscular contractions evoked by stimulation of efferent motor fibers. This will produce a large input to the CNS and a strong sensation of contraction and movement. Pain could be influenced by activation of large diameter afferent fibers that may excite inhibitory neurons in the spinal dorsal horn and suppress the neurons in laminae I, II, and V, which ordinarily fire in response to noxious stimuli [8]. They also may activate supraspinal inhibitory systems acting on nociceptive spinal neurons [18].

Several factors could explain the improvement in muscle strength. However, it is important to note that even though rMS induces muscle contraction, it is unlikely that the increase in muscle strength observed was due to a direct effect on the muscle. Indeed, a direct effect would take several weeks to occur, whereas the increase in strength was evident after the first 5 days of stimulation. Therefore, it is likely to be due to increased volitional drive to the remaining peripheral connections. Reduced pain could be one factor that would increase volitional output. In addition, Ridding et al. [15] noted that prolonged peripheral input can increase corticospinal excitability, which would also tend to increase voluntary strength. Both hypotheses are consistent with a PET study of stroke patients by Spiegel et al. [18] who found that rMS of the upper extremity increased activation of fronto-parietal circuits.

Another possibility is that magnetic stimulation of the nerve trunk of the brachial plexus increases the number of the endoneurial vessels, thereby improving the ischemic state of damaged nerve and promoting axonal regeneration. Such effects might contribute to the gradual increases in strength that are observed after cessation of treatment. This is in keeping with the significant improvement in neurophysiological parameters that was recorded in this study. An additional mechanism might be that rMS enhances the effect of physiotherapy at some central site.

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Pain

Although several previous studies underlined the usefulness of transcranial magnetic stimulation of the brain (rTMS) to treat pain syndromes [2,3,7,9–11], only a few studies dealt with the effects of peripheral magnetic stimulation. In keeping with the studies of Smania et al. [16,17] on myofascial pain syndromes and Pujol et al. [14] on musculoskeletal pain, our study indicates that rMS might be an effective tool to reduce peripheral pain.

Despite some of its limitations (small sample size, short follow-up, incomplete blinding), this preliminary study should encourage further research aimed at establishing the clinical usefulness of this new procedure in the treatment of brachial plexopathies.

Conclusion and recommendations

Even if the mechanisms of improvement need further investigations, peripheral rMS for 10 sessions might enhance motor recovery and pain relief in patients with traumatic brachial plexopathy.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

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