Central neuroplasticity and functional outcome of swinging upper limbs following repetitive locomotor training of lower limbs in stroke patients

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Aims
The aim of the study was to investigate the effect of long-term repetitive locomotor training on a treadmill with partial body weight support (TTPBWS) on motor performance of the swinging and supported paretic upper limb and to explore the neurophysiological mechanism underlying this improvement.

Materials and Methods
Thirty ambulatory chronic hemiparetic stroke patients were assigned randomly to either one of two experimental conditions while being trained for 20 min on a treadmill with PBWS 6 days a week for 8 weeks. Patients under condition 1 received verbal cueing to perform bilateral upper limb swinging. In condition 2, patients were instructed to support both upper limbs by holding the treadmill handrails. Fugel–Meyer upper extremity motor performance test (FMUE) and motor evoked potentials (MEPs) of the paretic middle deltoid (D), biceps brachii (BB), and abductor pollicis brevis muscles were assessed before rehabilitation (A-begin), immediately at its end (A-end), and 3 months later (A-3m). Changes in the FMUE scores and MEP variables were used for comparisons among groups.

Results
Both rehabilitation conditions resulted in a greater than 10% increase in the mean FMUE score. Group I showed a significant improvement in MEP variables (lower resting threshold, shorter central motor conduction time, and higher amplitude) in the three tested muscles. Group II showed a significant improvement in all the MEP variables of abductor pollicis brevis muscle and an increase in the MEP amplitude of only the BB muscle. Changes in the MEP threshold and amplitude of D and BB muscles were significantly higher in the patients in group I than those in group II.

Conclusion
Active bilateral upper limb swinging during treadmill training is more effective in improving paretic upper limb motor performance than training with supported upper limbs on treadmill handrails. Central neural plasticity may be underlying this recovery. Task-dependent neuronal coupling between lower and upper limb muscles during walking could be beneficial in stroke rehabilitation.

Keywords:
locomotor training, motor evoked potential, neuroplasticity, treadmill with partial body weight support

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Introduction

Upper extremity (UE) weakness after stroke is prevalent in acute and chronic stages of recovery, with up to 40% of patients never regaining functional use of their paretic UE in daily activities [1]. A multitude of different problems (including weakness, spasticity, and decreased aerobic capacity) may interfere with the accuracy of UE movement and motor performance (MP). These deficits may limit the implementation and success of rehabilitation programs that target UE use [2]. As the central nervous system has plastic neural networks amenable to reorganization, motor learning-based rehabilitation therapies that target the use of the hemiparetic limb may improve motor control and induce neural plasticity [3].

In chronic stroke patients, treadmill training with partial body weight support (TTPBWS), as a task-oriented approach that stimulates repetitive and rhythmic stepping, was effective in restoring locomotor function, and therefore, has increasingly been used in clinical practice. Besides, it was found that TTPBWS also had an effect on the hemiplegic UE [4]. Ploughman *et al.* [4] found that a single session of TTPBWS improved the arm motor skill. They attributed this improvement to some possible central and/or psychological changes. They recommended further studies to examine the effect of TTPBWS with and without arm support on the UE MP and to explore the etiology and duration of this enhancing effect. Investigation of the effect of repetitive TTPBWS on UE MP seems interesting as TTPBWS could be a possible alternative rehabilitation

strategy for improving MP of both the upper and the lower extremities in chronic stroke patients. We hypothesize that long-term repetitive TTPBWS may improve UE MP because of central changes or neuroplasticity. Therefore, the aim of this study was to investigate the effect of long-term repetitive locomotor TTPBWS on MP and on motor evoked potential (MEP) of the paretic UE in patients with chronic stroke. Studying the MEP might reflect any possible central changes associated with changes in MP [5].

Materials and Methods

Participants

Thirty patients with chronic hemiparetic stroke participated in this study. Inclusion criteria were as follows: age between 35 and 65 years, first ever-unilateral stroke, disease duration more than 6 months after stroke onset, able to walk with or without a cane, having residual UE weakness, muscle spasticity (at shoulder adductors, elbow flexors, wrist flexors, and hand flexors of the affected UE) classified as level 2 according to the Modified Ashworth Spasticity Scale (MASS) [6], ability to complete a 6-min walk test without cardiopulmonary distress, and received previous physical rehabilitation in the form of therapeutic exercises and parallel bar gait training not during the last 3 months before participation in the study. Exclusion criteria were as follows: complete loss of volitional movement of the UE involved (to avoid having many patients with unobtainable MEP and to ensure that patients will be able to follow therapist instructions during therapy), unsatisfactory general condition, distressing pulmonary diseases, history of myocardial infarction or myocardial ischemia, clinical signs of heart failure (New York Heart Association) [7], lower extremity vascular insufficiency, other neurological or orthopedic diseases compromising walking ability or UE function (e.g. neuropathy, joint stiffness, arthritis, or pain in the upper or the lower limb joints), insufficient communication, defective cognitive function, previous experience with TTPBWS, and contraindications for transcranial magnetic stimulation (TMS) (e.g. seizure, metallic implant in the head or neck, pacemaker).

A preliminary treadmill exercise test was performed. Patients who were able to walk for at least 6 min (at a minimum of 0.1 m/s) without signs of cardiopulmonary distress, myocardial ischemia, or treadmill exercise intolerance were enrolled. All patients provided their written informed consent for participation in the study, which was approved by the local ethics committee.

Training protocol

Over-ground self-selected comfortable walking speed along a 10-m walkway was determined, and then participants were allocated to either one of two experimental groups by a computer-generated randomization code. Each patient received a 20-min session of TTPBWS per day 6 days a week for 8 successive weeks. Patients of group I received verbal cueing to swing both UE (alternate with the ipsilateral leg motion as in normal gait) during each session of TTPBWS, whereas patients of group II were instructed to hold the treadmill handrails with both hands and not to swing their UE during TTPBWS. During TTPBWS, patients walked on a motor-driven treadmill while suspended by a modified parachute harness to an overhead suspension system. Training started with 30% body weight support and was decreased progressively as the patients were able to carry the remaining load on the paretic lower limb throughout the stance phase. Treadmill speed was adjusted below over-ground walking speed for a comfortable cadence and stride length of each patient that allowed gait correction. The mean treadmill speed was 0.4 m/s (range 0.2–0.6 m/s). Patients were assisted manually by two therapists to correct gait deviation and encourage a symmetrical gait pattern. One therapist facilitated swinging of the paretic lower limb, determined initial heel contact, and secured it during the stance phase. The other therapist stabilized the trunk, facilitated hip extension, and instructed the patient on the UE activity according to individual requirements [8].

Assessment

Assessment was performed in both groups immediately before the start of rehabilitation (A-begin), immediately at the end of the eight-week rehabilitation period (A-end), and at 3 months after the end of the study (A-3m). In each assessment, the following outcome measures were performed:

(1) *Fugl–Meyer upper extremity (FMUE) motor performance test*: This was done for the affected UE. This test was chosen as it has been described to be a simple and a reliable quantitative test [9] (66 points; each point is scored 0–2). It assesses motor impairments and recovery from hemiplegic stroke. Its motor domain includes items measuring volitional movements (flexor synergy, extensor synergy, movement combining synergies, and movement out of synergy), coordination/speed, and reflex action about the shoulder, elbow forearm, wrist, and hand [9].
(2) *Percutaneous TMS to elicit MEP*: MEP was recorded from the affected UE following TMS of the contralateral cortical motor area. During TMS stimulation, the stimulating coil was

positioned tangentially over the skull with the center of the coil placed over the vertex (which is corresponding to Cz), with the handle parallel to the sagittal plane. MEP were recorded from paretic middle deltoid (D), biceps brachii (BB), and abductor policisbrevis (APB) muscles using surface recording disc electrodes (1 cm diameters) connected to a conventional electrophysiological apparatus (Neuropack 2; Nihon Kohden, Tokyo, Japan). Filter was set to 3 Hz–3 kHz. Gain was varied according to the MEP amplitude (200–20 μV/division). Time base was set at 5 ms/division. Magnetic stimulation was performed using a Magstim 200 single pulse stimulator (Magstim Company, Whitland, Wales, UK) equipped with a high-power 90 mm circular coil, which generates 2 T maximum field intensity. The testing protocol was carried out according to the International Federation of Clinical Neurophysiology criteria for magnetic stimulation of the brain [10]. MEP was considered unobtainable if 10 successive discharges failed to elicit a response from the target muscle at the maximum output (100%) intensity. Resting threshold intensity, MEP maximum peak to peak amplitude (mV), and the shortest MEP cortical latency (CL) in ms were the recorded MEP variables. Patients were assessed while in a relaxed supine position, and TMS testing lasted from 35 to 45 min for each patient.

Central motor conduction time (CMCT) was calculated for D and BB muscles using the following formula: CMCT = CL (m)–RL (ms) (RL = root latency). RL was recorded by centering the stimulating coil over the C7 spinous process and recording compound muscle potential from the same site as during TMS. For the APB muscle, CMCT was calculated by subtracting peripheral latency (PL) from the CL. The PL for the APB was calculated using the following formula:

$$\mathrm{PL(ms)} = \frac{[\text{minimal F-wave latency(ms)} + \text{M-wave latency(ms)} - 1\text{ms}]}{2}.$$

The F-wave and M-wave were recorded following supramaximal stimulation of the median nerve at the wrist. The subtracted 1 ms in the formula is the estimated turnaround time of the antidromic volley at the anterior horn cell [11]. The amplitude of MEP was expressed as the ratio of M-wave amplitude of the corresponding muscle.

Data analysis

The percent changes in FMUE scores and MEP parameters (at A-end and at A-3m) were calculated for each patient and used for comparison between the two groups (rather than the absolute difference between prerehabilitation and postrehabilitation values). This was done to control for rehabilitation effects. The percent change was calculated according to the following formulae:

$$\text{Change 1} = \frac{[(\text{A-end}) \text{ value} - (\text{A-begin}) \text{ value}]}{(\text{A-begin}) \text{ value}} \times 100,$$

$$\text{Change 2} = \frac{[(\text{A-3m}) \text{ value} - (\text{A-end}) \text{ value}]}{(\text{A-end}) \text{ value}} \times 100.$$

Descriptive statistics (as means ± SD) were used to compare baseline characteristics, FMUE scores, and MEP variables of both groups. Skewness of the measured variables was assessed to determine the normality of distribution at baseline assessment. Statistical differences in FMUE scores and MEP parameters at each assessment as well as changes in these measures were compared between the two groups using a nonparametric Mann–Whitney test. A nonparametric Wilcoxon test was used for intragroup comparisons. Significance was set at P value of 0.05 or less for all analyses. The statistical package SPSS, version 17 (Inc., Chicago, Illinois, USA) was used for statistical analyses.

Results

All patients completed the training sessions. There were no dropouts throughout the study or adverse events. The groups studied were comparable, without a significant difference between them in age, sex, height, arm length, weight, disease duration, side affected, preintervention FMUE scores, and preintervention MEP variables (Table 1).

MEP was unobtainable (from the three tested muscles) before and after rehabilitation in only one patient of group I. However, MEP was obtainable before and after rehabilitation in all patients of group II.

The FMUE scores and MEP variables before and after rehabilitation in patients of both groups are shown in Table 2. There was a significant increase in FMUE scores within each group at A-end (compared with A-begin) and at A-3m (compared with A-begin and A-end) (Table 2). However, there was no significant difference between the two groups in the change in FMUE scores (Table 3). The postrehabilitation increase in the FMUE scores was greater than 10% in both groups.

In group I, all MEP variables (of the three tested muscles) improved (lower mean resting threshold, shorter mean CMCT, and higher mean amplitude

Table 1 Baseline demographic, clinical characteristics, FMUE scores, and MEP variables in patients of group I and group II

	Minimum–maximum (mean ± SD)		
	Group I	Group II	*P*
Age of patients (years)	35–62 (50.6 ± 9.7)	40–65 (53.5 ± 6.2)	0.493
Sex (male/female)	7/8	8/7	0.278
Weight (kg)	63–87 (76.6 ± 5.2)	64–86 (75.9 ± 7.3)	0.122
Height (cm)	154–178 (165.3 ± .23)	154–179 (166.7 ± 6.5)	0.139
Arm length (cm)	64–75 (69 ± 2.3)	64–72 (68.3 ± 2.7)	0.296
Disease duration (months)	6–72 (24.8 ± 21.3)	6–72 (26.2 ± 29.2)	0.917
Affected side (right/left)	9/6	8/7	0.321
FMUE scores	33–52 (43.3 ± 5.2)	34–50 (42.9 ± 4.5)	0.771
APB threshold (%)	30–90 (55.3 ± 16.2) (*n* = 14)	35–82 (55.8 ± 15.8) (*n* = 15)	0.982
APB CMCT (ms)	5.1–15.9 (9.3 ± 2.6)	5.4–12.2 (8.3 ± 1.9)	0.356
APB amplitude (mV)	0.01–0.60 (0.22 ± 0.1)	0.03–0.43 (0.19 ± 0.13)	0.967
D threshold (%)	43–88 (61.0 ± 13.4) (*n* = 14)	34–85 (53.6 ± 15.1) (*n* = 15)	0.277
D CMCT (ms)	5.3–8.6 (6.8 ± 1.0)	5.1–7.4 (6.2 ± 0.79)	0.128
D amplitude (mV)	0.04–0.32 (0.14 ± 0.09)	0.07–0.41 (0.22 ± 0.11)	0.081
BB threshold (%)	44–86 (60.92 ± 11.9) (*n* = 14)	35–83 (54.6 ± 14.5) (*n* = 15)	0.332
BB CMCT (ms)	5.2–8.4 (7.0 ± 0.8)	5.1–8.3 (6.8 ± 1.0)	0.533
BB amplitude (mV)	0.03–0.53 (0.18 ± 0.18)	0.05–0.38 (0.22 ± 0.10)	0.290

Except when indicated otherwise, values are the minimum–maximum (mean ± SD). APB, abductor pollicis brevis; BB, biceps brachii; CMCT, central motor conduction time; D, deltoid; MEP, motor evoked potential; FMUE, Fugl–Meyer upper extremity motor performance test. $P \leq 0.05$, significant.

Table 2 FMUE scores and MEP variables before and after rehabilitation in the two groups studied

	Group I						Group II					
	A-begin Mean ± SD	A-end Mean ± SD	A-3m Mean ± SD	*P*1	*P*2	*P*3	A-begin Mean ± SD	A-end Mean ± SD	A-3m Mean ± SD	*P*1	*P*2	*P*3
FMUE scores	43.33 ± 5.2	45.3 ± 5.1	47.0 ± 5.3	0.002*	0.001*	0.001*	42.9 ± 4.5	44.2 ± 4.4	45.6 ± 4.2	0.024*	0.001*	0.001*
APB threshold (%)	55.3 ± 16.2	56.0 ± 19.8	54.3 ± 19.5	0.005*	0.007*	0.003*	55.8 ± 15.8	53.4 ± 15.2	52.0 ± 12.9	0.004*	0.058	0.006*
APB CMCT (ms)	9.3 ± 2.6	8.5 ± 2.0	8.3 ± 1.8	0.003*	0.022*	0.006*	8.3 ± 1.9	8.1 ± 1.9	7.9 ± 1.8	0.001*	0.081	0.002*
APB amplitude (mV)	0.22 ± 0.1	0.24 ± 0.18	0.25 ± 0.19	0.003*	0.163	0.003*	0.19 ± 0.13	0.21 ± 0.13	0.22 ± 0.12	0.02*	0.046*	0.008*
D threshold (%)	61.0 ± 13.4	59.9 ± 16.9	59.1 ± 16.3	0.002*	0.23	0.004*	53.6 ± 15.1	54.6 ± 13.9	54.3 ± 15.0	0.67	0.54	0.94
D CMCT (ms)	6.8 ± 1.0	6.6 ± 1.0	6.4 ± 1.0	0.002*	0.085	0.003*	6.2 ± 0.79	6.0 ± 0.7	5.8 ± 0.6	0.146	0.02*	0.01*
D amplitude (mV)	0.14 ± 0.09	0.17 ± 0.10	0.18 ± 0.11	0.003*	0.05*	0.001*	0.22 ± 0.11	0.22 ± 0.12	0.24 ± 0.11	0.40	0.09	0.16
BB threshold (%)	60.9 ± 11.9	61.2 ± 16.0	60.5 ± 16.0	0.009*	0.05*	0.006*	54.6 ± 14.5	54.2 ± 13.3	53.9 ± 12.3	0.61	0.87	0.62
BB CMCT (ms)	7.0 ± 0.8	6.7 ± 0.8	6.6 ± 0.6	0.019*	0.085	0.019*	6.8 ± 1.0	5.7 ± 0.7	5.7 ± 0.7	0.001*	0.948	0.002*
BB amplitude (mV)	0.18 ± 0.18	0.22 ± 0.19	0.25 ± 0.19	0.003*	0.053	0.001*	0.22 ± 0.10	0.25 ± 0.10	0.25 ± 0.10	0.003*	0.635	0.011*

MEP variables were taken from those with obtainable MEP. A, assessment; other abbreviations as in Table 1. *$P \leq 0.05$, significant. *P*1: Probability for A-begin vs. A-end. *P*2: Probability for A-end vs. A-3m. *P*3: Probability for A-begin vs. A-3m.

ratio) at A-end and at A-3m compared with A-begin. However, in patients of group II, there was a significant postrehabilitation improvement in only some MEP variables (Table 2). Comparison between the two groups indicated that change 1 of the D MEP threshold and amplitude were significantly higher in patients of group I. Nevertheless, there were no other significant changes in MEP variables between the two groups (Table 3).

Discussion

This study investigated the change in the paretic UE MP in chronic stroke patients following 8 weeks of TTPBWS under two experimental conditions: one with UE swinging (group I) and the other with the hands holding the treadmill handrails (group II). The paretic UE MP improved significantly under both training conditions, as determined by the significant increase in FMUE scores following TTPBWS. Although the improvement in the UE MP was partial (>10%), it represents a clinically meaningful improvement as this advances patients to the next stage of motor recovery.

Ploughman *et al.* [4] reported that a single 20-min session of TTPBWS enhanced UE motor skills in 72 patients with chronic stroke [4]. Therefore, they recommended further studies to examine the etiology and longevity of this effect of exercise. Lindquist *et al.* [8] studied the effects of combined TTPBWS and functional electrical stimulation (for 27 sessions) on gait in eight chronic stroke patients. They [8] reported that the paretic UE motor activities improved

Table 3 Changes in FMUE scores and MEP variables in patients of group I and group II

	Mean ± SD		
	Group I	Group II	*P*
Change 1[a] FMUE scores	4.7 ± 3.5	3.2 ± 4.7	0.158
Change 2[b] FMUE scores	3.8 ± 2.1	3.2 ± 2.2	0.389
Change 1[a] APB threshold	−4.9 ± 4.2	−4.0 ± 4.6	0.407
Change 1[a] APB CMCT	−8.4 ± 8.0	−3.2 ± 2.4	0.197
Change 1[a] APB amplitude	54.5 ± 136.4	25.5 ± 42.9	0.800
Change 1[a] D threshold	−6.9 ± 5.1	3.3 ± 12.8	0.002*
Change 1[a] D CMCT	−5.2 ± 4.4	−2.5 ± 6.1	0.093
Change 1[a] D amplitude	34.6 ± 33.2	2.3 ± 25.8	0.017*
Change 1[a] BB threshold	0.06 ± 6.2	0.04 ± 5.2	0.259
Change 1[a] BB CMCT	−5.1 ± 8.3	−14.6 ± 11.0	0.982
Change 1[a] BB amplitude	34.5 ± 44.2	22.7 ± 22.7	0.927
Change 2[b] APB threshold	−2.8 ± 2.6	−1.6 ± 5.1	0.895
Change 2[b] APB CMCT	−2.2 ± 3.9	−1.6 ± 3.4	0.382
Change 2[b] APB amplitude	15.0 ± 28.4	7.0 ± 11.9	0.948
Change 2[b] D threshold	−0.95 ± 5.1	−1.0 ± 6.8	0.896
Change 2[b] D CMCT	−1.6 ± 3.2	−2.9 ± 4.7	0.205
Change 2[b] D amplitude	8.6 ± 14.3	10.7 ± 18.8	0.541
Change 1[b] BB threshold	−1.1 ± 2.0	0.01 ± 4.5	0.273
Change 1[b] BB CMCT	−1.9 ± 5.0	−0.03 ± 3.7	0.116
Change 1[b] BB amplitude	1.6 ± 15.8	1.2 ± 18.4	0.258

[a]The percent changes at the end of rehabilitation. [b]The percent changes at 3 months after rehabilitation; other abbreviations as in Table 1. *$P \leq 0.05$, significant.

in two patients, although the UE did not undergo specific training. They [8] reported that gait is a full-body activity that may account for UE improvement, and that hand control could have been influenced by the training because the patients were encouraged to hold onto the horizontal bars attached to the sides of the treadmill.

Although previous studies [4,8] used no specific upper limb exercises, in the present study, both groups performed locomotor training combined with bilateral UE tasks, which could have influenced motor performance by drawing the patient's attention to the UE. In group II, patients trained while holding treadmill handrails (close hand from fully opened position and vice versa). This might have influenced hand control and/or could have relaxed tone in the arm and hand by performing rhythmic movement of the trunk on fixed arms while walking as suggested previously [4]. In group I of the present study, the motor task was closest to normal walking and according to the specificity of learning hypothesis and this is expected to provide greater functional benefit.

UE performance could be improved during gait training because of the existence of a common neural control of movements of the upper and lower limbs and related muscle activities. Interlimb coordination has been well documented during locomotion and has been attributed to neural linkages connecting cervical and lumbosacral networks in the spinal cord [12,13]. Juvin *et al.* [13] reported the dominance of locomotor drive from the lumbar central pattern generators over the cervical counter parts in an isolated spinal cord preparation. This inherent interlimb neural coupling was suggested to be mediated by an ascending caudorostral propriospinal excitability gradient. Humans coordinate upper and lower limb movements during locomotion through the spinal reflex pathway, which becomes facilitated rhythmically by activity of central pattern generators during gait. Dietz *et al.* [14]showed that there was a close relationship between leg and arm muscle electromyographic responses and that arm muscle responses were most pronounced during normal gait. A pilot study examined the effects of aerobic exercise on upper extremity function in chronic stroke using an upper and lower body reciprocal trainer [15]. After 8 weeks of exercise, the time to complete the upper limbs-specific tasks of the Wolf motor function test showed significant decreases and these changes were maintained for 4 weeks.

The results of the current study indicated that exercise incorporating both upper and lower extremities improved MP of the UE after stroke. The continued improvement in FMUE scores for 3 months after the end of the study in the two groups is evidence of the long-term benefit of TTPBWS on seemingly unrelated upper limb performance. This could be because of neuroplasticity as evidenced from the MEP changes obtained. Significant postrehabilitation improvement in MEP variables was observed in the proximal and distal UE muscles in patients of group I, whereas in group II, postrehabilitation improvement was mainly in the APB MEP variables. Also, the changes in the D MEP threshold and amplitude were significantly better in group I compared with the changes in group II. These findings suggest that active arm swinging (which necessitates active repetitive UE proximal muscle movement, unlike holding the treadmill handrails) during TTPBWS was more effective in improving MEP variables of the UE proximal muscles. This could be because of potentiation of cortical motor areas, which in turn modify the excitability of specific motor neurons through synaptic plasticity in the motor cortex [16]. In group II, supporting UE on handrails might have hindered significant improvement of MEP variables because of less activation of arm muscles and consequently less activation of cortical motor areas. This does not contradict the Dietz *et al.* [14] study, which reported a strong reduction of arm muscle responses and background electromyography when arm movements became restricted during locomotion.

Sensory information from the auditory verbal cues provides intrinsic attention feedback to the patient for the movement goal, which is known to promote motor learning. It is plausible that repetition and cueing contributed toward motor learning in hemiparetic patients [17]. Also, neurophysiological variables obtained from the paretic upper limb muscles could be improved as a result of not only being actively integrated in the training conditions but also because of the activity of the unaffected upper limb. Practicing bilateral UE movement results in contralateral activity in homologous muscles during voluntary contraction by an interhemispheric facilitation effect from the nonlesional hemisphere to the lesional one by a decrease in inhibition along the intercallosal connections between the primary and the supplementary motor areas. Again, the existence of an interlimb coordination between arms during a great variety of manipulative tasks is well documented [18,19].

Furthermore, exercise training can improve MP through several other mechanisms. Exercise training was found to increase cerebral blood volume and angiogenesis in areas crucial for task performance [20]. Another mechanism is that exercise upregulates neurotrophins such as brain-derived neurotrophic factor and insulin-like growth factor I [21], which support dendritic branching and synaptic plasticity in the adult brain. Also, a near-infrared spectroscopic topography study showed activation in arm areas of the primary sensorimotor cortex during gait activity [22]. Besides, exercise improves mood and alleviates depression [23], which may also affect assessment of MP [4].

Conclusion

This study showed that although TTPBWS enhanced the UE MP in chronic stroke patients, active bilateral UE swinging during TTPBWS yielded more significant improvement in the paretic UE MP than training with the UE supported on treadmill handrails. MEP findings suggested that central neural plasticity could be the underlying mechanism of this improvement. Therefore, task-dependent neuronal coupling between lower and upper limb muscles during walking could be beneficial in stroke rehabilitation.

Acknowledgements

Conflicts of interest

None declared.

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