Gait Training–Induced Change in Corticomotor Excitability in Patients With Chronic Stroke

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Background. Numerous studies have reported the effects of gait training on motor performance after stroke. However, there is limited information on treatment-induced changes in corticomotor excitability. *Objectives.* The purpose of the study was to investigate the effects of additional gait training on motor performance and corticomotor excitability and to demonstrate the relationship between motor improvement and corticomotor excitability change in patients with chronic stroke. *Methods.* Fourteen patients were randomly assigned to the experimental or control group. Participants in both groups participated in general physical therapy. Those in the experimental group received additional body weight–supported treadmill training for 4 weeks. All participants received baseline and posttreatment assessments. The outcome measures included assessment of the Berg Balance Scale (BBS) and gait parameters. Focal transcranial magnetic stimulation was used to measure the motor threshold, map size, and location of the amplitude-weighted center of gravity of the motor map for the tibialis anterior (TA) and abductor hallucis (AH) muscles. *Results.* After general physical therapy, we noted that the patients showed an improvement only in walking speed and cadence, and there were no significant changes in corticomotor excitability. After additional gait training, participants improved significantly on BBS score, walking speed, and step length. Moreover, the motor threshold for TA decreased significantly in the unaffected hemisphere. The map size for TA was increased in both hemispheres, whereas that for AH was increased only in the affected hemisphere. There were significant differences between the change scores of the groups in terms of walking speed, step length, and motor threshold for TA in the unaffected hemisphere and map size for AH in the affected hemisphere. Additionally, the changes in corticomotor excitability correlated with functional improvement. *Conclusions.* Additional gait training may improve balance and gait performance and may induce changes in corticomotor excitability.

Key Words: *Body weight–supported treadmill training—Balance and gait—Transcranial magnetic stimulation—Corticomotor excitability—Stroke.*

After stroke, there are marked deficits in balance and gait. Decreases in gait velocity, cadence, and step length are hallmark features of gait in patients with stroke.[1,2] Gait deviations such as hip-hiking, hip circumduction, or drop foot are commonly noted.[3] The ability to walk independently with efficient velocity and endurance is one of the major goals for rehabilitation after stroke. Positive effects of treadmill training on motor performance have been demonstrated in previous studies.[3-9] Furthermore, the effects of treadmill training in combination with body weight support (BWS) were superior to those without BWS or overground walking for stroke patients at different stages.[10-17] However, the neuroplasticity induced by body weight–supported treadmill training (BWSTT) remains unclear.

Cortical reorganization occurs after central nervous system lesions. It consists of lesion- and treatment-induced cortical reorganization.[18,19] Several studies have revealed that improvement in motor performance is correlated with neurophysiologic measures.[19-22] Improvement in motor performance is likely related to brain changes. It has been indicated that cortical reorganization occurs after stroke.[22-25] Most studies have focused on the recovery of the paretic upper extremity after stroke. However, few studies investigated exercise-induced lower extremity neural plasticity. Using transcranial magnetic stimulation (TMS), Perez et al[26] reported that motor skill training involving ankle movement induced an increase in the excitability of the leg cortical area in healthy adults. Using functional magnetic resonance imaging, Dobkin

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et al[18] demonstrated changes in several cortical areas over the course of BWSTT in 4 patients with stroke. Miyai et al[27] used near infrared spectroscopy during actual treadmill walking to demonstrate that multiple motor areas might play important roles in gait restoration in patients with stroke. More recently, Forrester and colleagues also reported that treadmill training for 3 months may alter the excitability of the lower-extremity central motor pathways in patients with stroke.[28] Although corticomotor reorganization following lower extremity training has been demonstrated in these previous studies, the relationship between motor improvement and corticomotor excitability change after gait training remains unknown.

Our previous results showed that ankle impairment affected gait performance significantly in stroke patients.[29] Dorsiflexor strength of the affected side was the most important factor in determining gait velocity and temporal symmetry.[29] The corticospinal projections to motor neurons of the tibialis anterior (TA) are more pronounced than those to the motor neurons of the other muscles of the lower extremity.[30] Moreover, some evidence suggested that the abductor hallucis (AH) receives a disproportionately large output from the motor cortex.[31] Recruitment of AH by the TMS is relatively easy compared to that in the case of other lower-limb muscles in awake humans.[32] Considering this, it is reasonable to assume that the TA and AH play a prominent role in cortically controlled voluntary movements.

The purpose of the present study was to investigate the effects of additional BWSTT on motor performance and corticomotor excitability in patients with chronic stroke and to clarify the relationship between motor improvement and corticomotor excitability change. In this study, we used TMS to measure the cortical outcome and selected TA and AH as the target muscles.

METHODS

Participants

Individuals with stroke were recruited from Taipei Veterans General Hospital. The criteria for inclusion were (1) unilateral stroke resulting in unilateral hemiparesis, (2) a minimum 6-month time lapse since stroke onset, (3) ability to walk at least 10 m with or without assistance, (4) sufficient cognition, and (5) stable medical condition. The exclusion criteria were (1) any neurological or orthopedic diseases that might interfere with the study, (2) past history with seizure, (3) heart pacemaker, (4) severe cardiac problems, (5) metallic implant materials in the head, (6) walk with a normal gait pattern, and (7) inability to walk before stroke.

Procedure

The study protocol was approved by the Institutional Review Board of Taipei Veterans General Hospital. After explaining the experimental protocol to the participants, their informed consent was obtained. Participants were randomly assigned to either the experimental or control group by an independent person who selected 1 of the sealed envelopes 30 minutes before the start of the intervention. All participants were evaluated prior to the commencement of training (baseline) and at the end of the 4-week training period (posttreatment). Since we had limited resources, this study did not use a blinded assessor. However, we attempted to minimize bias by using standardized testing instructions and protocols. All participants underwent a 50-minute general physical therapy session (2 to 5 sessions a week) over a period of 4 weeks. These sessions involved stretching, muscle strengthening, balance, and overground walking training. Furthermore, the participants in the experimental group received 12 additional sessions of BWSTT (30-min/session, 3 sessions a week) over a 4-week period.

Training Principles for BWSTT

BWSTT involved treadmill training (EN-MILL, Bonte Zwolle BV, the Netherlands) using a body weight–supported system (Biodex, Shirley, NY). A BWS of less than 40% of the body weight was provided and was decreased to the maximum extent possible.[14] The criterion for decreasing the amount of BWS was the patient's ability to carry the remaining load on the paretic leg with less than 15 degrees of knee flexion during the single-support phase.[12,15] The treadmill speed was determined according to the patient's ability. If the patient's ability increased with training, BWS was initially decreased and the speed was then increased.

The subject was trained with the assistance of 1 or 2 physical therapists. The main purpose of this training was to normalize the gait pattern of individuals in terms of maintaining a neutral position of the ankle joint during the swing phase and knee extension during the stance phase to the maximum possible extent. The participants were not permitted to use a lower-extremity orthosis during the training phase, were instructed to refrain from holding a handrail if possible, and were encouraged to use reciprocal arm swing.[9,14]

Outcome Measures

The outcome measures that included motor performance and corticomotor activity were evaluated at baseline and posttreatment for participants in both groups.

The motor performance consisted of balance and gait performance. The Berg Balance Scale (BBS) was used to evaluate static and dynamic balance performance. To evaluate gait performance, the GAITRite system (CIR Systems, Inc, Havertown, PA) was used. The participants were instructed to ambulate along the GAITRite walkway at their fastest speed in the 3 trials conducted. The results from these 3 trials were averaged. Walking speed, cadence, and step length were evaluated.

TMS was used to evaluate corticomotor activity. It was delivered via MagStim 200 (MagStim, Carmarthenshire, Wales, UK) electromagnetic stimulation by using a figure-of-eight coil. The motor-evoked potentials (MEPs) were recorded using an electromyographic machine (Neuropack 8, Nihon Kohden, Tokyo, Japan). Bilateral TA and AH muscles were studied. While stimulating over the affected (or unaffected) hemisphere, MEPs were recorded over the muscles contralateral to the affected (or unaffected) hemisphere. The participants were seated comfortably with their hip and knee maintained in 90-degree flexion. A coordination system (distance of 1 cm) was marked on a fitting cap worn by the participant. The participants were instructed to completely relax their leg during the evaluation period. The handle of the coil pointed outward during MEP testing for TA and AH.[33] Three parameters were used to analyze the neurophysiologic data. The first was the resting motor threshold. It was determined at rest as the minimum percentage of the stimulation output that induces MEPs greater than 0.05 mV peak-to-peak amplitude in at least 5 out of 10 trials over the motor cortex.[34] The second parameter was map size, and this was expressed as the number of scalp positions with stimulations that evoked MEPs more than 0.05 mV in at least 1 out of 4 trials.[22] The third parameter was the location of the amplitude-weighted center of gravity (CoG) of the motor map. This was derived from the distribution of MEP amplitudes within the motor output area and calculated using a single *x-y* coordinate.[35] Prior to mapping, the optimal spot to elicit MEP in each target muscle was determined. At this position, the resting motor threshold was evaluated. During mapping, the stimulus intensity was applied with 110% of the motor threshold.[36] The coil was then moved systematically over the skull in 1-cm steps to identify all scalp positions whose stimulations produced a MEP. Each scalp position was defined in relation to the vertex. This made it possible to repeatedly locate each position during subsequent mapping procedures. The same intensity as that used in the pretreatment session was then applied for the posttreatment session.[22]

Data Analysis

All data were analyzed by the SPSS 10.0 software (SPSS Inc, Carey, NC). To understand the intragroup training effect, the Wilcoxon signed-rank test was used. To analyze the intergroup improvement, the change scores were analyzed by Mann-Whitney *U* test. Change scores were calculated by subtracting the baseline data from the posttreatment data. Furthermore, Spearman test was used to analyze the correlation between the results of the change of each motor performance and those of the corticomotor excitability changes. Statistical significance was set at $P < .05$.

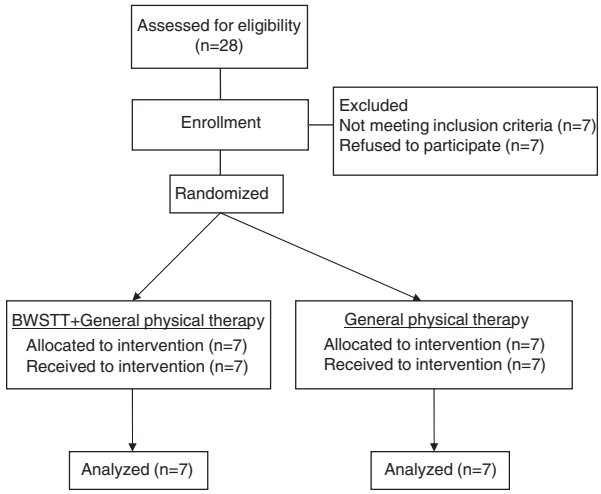

Figure 1. Flow diagram of the study. BWSTT = body weight–supported treadmill training.

RESULTS

Participant Flow

For this study, 28 individuals were identified as potential participants (Figure 1). Of these, 7 were excluded because they failed to fulfill the inclusion criteria and 7 did not sign the informed consent. Thus, of the original pool, 14 participants met the eligibility criteria and provided their written informed consent. These participants were randomized to the control group (n = 7) or experimental group (n = 7). The demographic and clinical characteristics of participants in both groups are listed in Table 1. There were no significant differences in the demographic and clinical characteristics between the groups. All participants successfully completed the study protocol. In the experimental group, the attendance rate was 100% for the 4-week training program.

Percentage of BWS and Treadmill Speed

Initially, the mean percentage of the BWS used was 16.43% ± 7.48%. Finally, 6 out of 7 participants

Table 1. Demographic and Clinical Features of Patients in Both Groups

	Experimental Group (n = 7)	Control Group (n = 7)	*P* Value
Age (years)[a]	57.30 ± 16.44 (36-77.7)	56.05 ± 12.69 (37.7-77)	.85
Gender[b]			.09
Male	3 (42.86%)	6 (85.71%)	
Female	4 (57.14%)	1 (14.29%)	
Height (cm)[a]	161.86 ± 7.97 (150-172)	168.21 ± 9.72 (152-184)	.18
Weight (kg)[a]	64.43 ± 12.33 (48-83)	70.03 ± 7.39 (56-78)	.25
Time since stroke (years)[a]	1.97 ± 0.61 (1.5-2.9)	1.96 ± 2.42 (0.5-7.1)	.14
Affected side[b]			.28
Right	5 (71.43%)	3 (42.86%)	
Left	2 (28.57%)	4 (57.14%)	
Stroke type[b]			.58
Infraction	4 (57.14%)	5 (71.43%)	
Hemorrhage	3 (42.86%)	2 (28.57%)	
Brunstrom stage of lower extremity[a]	4 ± 0 (4-4)	4 ± 0.38 (4-5)	.32
Frequency of physical therapy (time/week)[a]	2.43 ± 0.54 (2-3)	2.43 ± 1.27 (1-5)	.17

Values are mean ± standard deviation (range)[a] or frequency (percentage)[b].

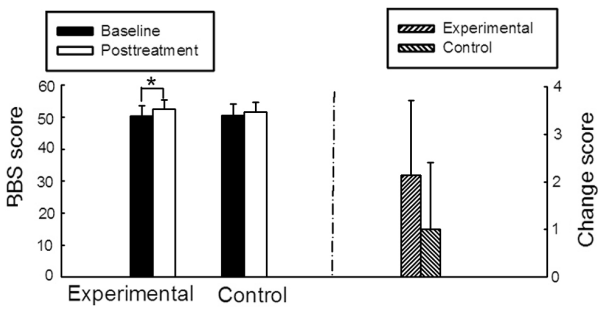

Figure 2. Berg Balance Scale (BBS) score was increased in the experimental group (*$P < .05$). No significant difference was found in the control group (left). The improvement of BBS score was similar between the experimental group and control group (right). Values are mean ± standard deviation.

(85.71%) were trained without BWS; 3 participants were trained without the BWS from the third week, and another set of 3 from the fourth week. The mean BWS was significantly decreased ($P = .01$) from the first through the final session (1.43% ± 3.78%). The mean treadmill speed was increased significantly ($P = .01$) from 1.32 ± 0.42 to 2.16 ± 0.62 kmph through the 4-week treatment period.

Motor Performance

The BBS data are presented in Figure 2. After general physical therapy, the BBS score did not change significantly (from 50.57 ± 3.55 to 51.57 ± 3.1, $P = .102$) for participants in the control group (Figure 2, left). Following additional BWSTT, the BBS score significantly increased from 50.29 ± 3.25 to 52.43 ± 2.88 ($P = .016$) for participants in the experimental group (Figure 2, left). The change score of BBS did not show any difference ($P = .211$) in the intergroup comparison (Figure 2, right).

The detailed results of the gait parameters are presented in Table 2. In intragroup comparisons, the participants in the control group showed a significant improvement in their walking speed ($P = .018$) and cadence ($P = .042$) after general physical therapy. However, there was no improvement in the step length bilaterally ($P = .091$ for both limbs). The participants in the experimental group significantly improved their walking speed ($P = .018$) and step length of both limbs ($P = .018$ for both limbs) after additional BWSTT. However, the experimental group did not show a significant difference in cadence ($P = .176$) in the intragroup comparison. In the intergroup comparisons, the change scores of walking speed ($P = .004$) and step length ($P = .048$ for both limbs) in the experimental group were higher than those in the control group. There were no significant differences in the change scores of cadence ($P = .749$) between the groups.

Corticomotor Activity

Motor threshold. In the unaffected hemisphere, the MEPs for TA and AH could be elicited in all the participants at the baseline and posttreatment assessments.

Table 2. Gait Parameters in the Intragroup and Intergroup Comparisons

	Experimental Group (n = 7)		Control Group (n = 7)		Change Score	
	Baseline	Post	Baseline	Post	Experimental	Control
Speed (cm/s)	68.66 ± 32.86	92.24 ± 32.34*	78.44 ± 43.49	86.79 ± 43.00*	23.59 ± 10.69†	8.34 ± 2.58
Cadence (step/min)	97.31 ± 27.69	102.84 ± 20.30	97.37 ± 22.23	103.06 ± 18.58*	5.53 ± 12.14	5.69 ± 5.61
Step length (cm)						
Affected	44.88 ± 19.93	56.53 ± 15.83*	48.25 ± 18.87	50.87 ± 17.22	11.64 ± 13.20†	2.62 ± 3.81
Nonaffected	37.73 ± 17.93	49.09 ± 14.80*	41.40 ± 20.74	45.42 ± 22.63	11.35 ± 8.06†	4.03 ± 4.91

Values are mean ± standard deviation.
Post: posttreatment assessment.
*$P < .05$ for the intragroup comparison.
†$P < .05$ for the intergroup comparison.

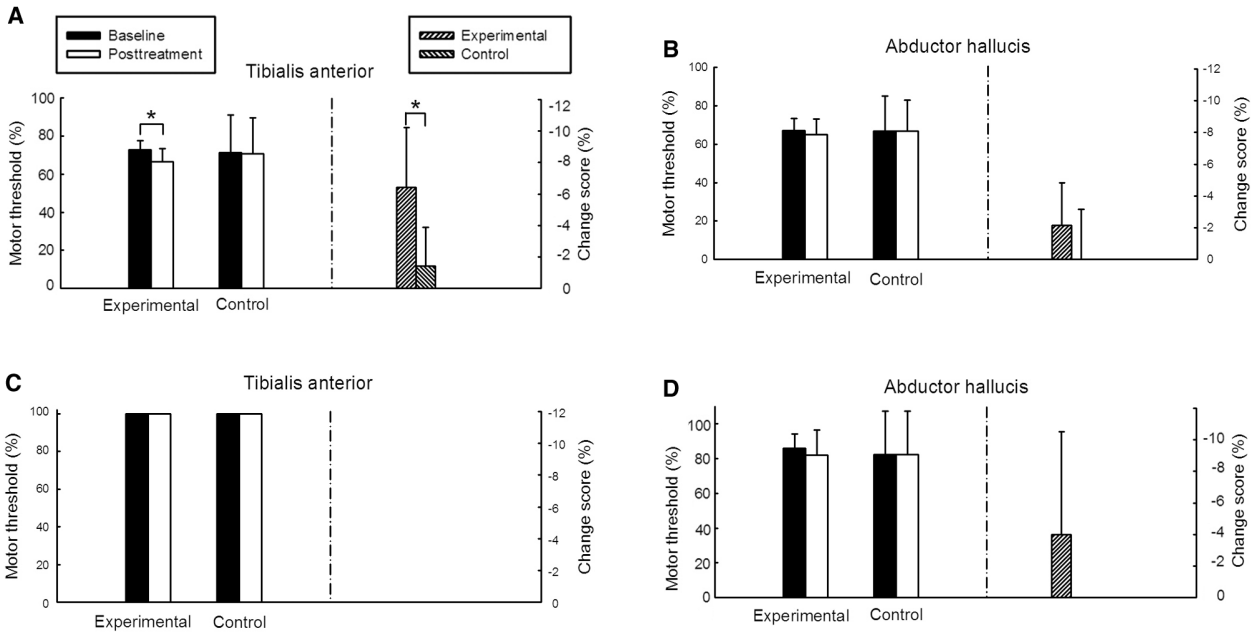

Figure 3. The motor threshold in unaffected (A, B) and affected (C, D) hemisphere in the intragroup (left) and intergroup (right) comparisons. The motor threshold for tibialis anterior muscle in the unaffected hemisphere (A, left) was decreased in the experimental group (*$P < .05$). The decrease of motor threshold for tibialis anterior muscle (A, right) was larger in the experimental than that in the control group (*$P < .05$). Values are mean ± standard deviation.

The motor threshold for TA in the control group did not show a significant difference (from 71.43 ± 19.52 to 70.75 ± 18.93, $P = .157$) in the intragroup comparison (Figure 3A, left). The motor threshold for TA in the experimental group showed a significant decrease (from 72.86 ± 4.88 to 66.43 ± 6.9, $P = .024$) in the intragroup comparison (Figure 3A, left). There was a significant difference in the change score of the motor threshold for TA ($P = .02$) between the groups (Figure 3A, right). The motor threshold for AH showed no significant changes in the intra- and intergroup comparisons (Figure 3B, left and right, respectively).

In the affected hemisphere, only 1 participant (14.3%) in the experimental group and 2 participants (28.6%) in the control group could induce MEPs for TA at the baseline. However, 5 participants (71.4%) in the experimental group and 2 participants (28.6%) in the control group could induce MEPs for TA at posttreatment assessment. The motor threshold for TA in both groups was 100% of the maximum stimulator power output at baseline and posttreatment assessments (Figure 3C, left). In addition, 5 participants (71.4%) in the experimental group and 2 participants (28.6%) in the control group could induce MEPs for AH at baseline and at posttreatment assessments. The motor threshold for AH did not show significant differences in the intra- and intergroup comparisons ($P = .157$ in both cases; Figure 3D, left and right, respectively).

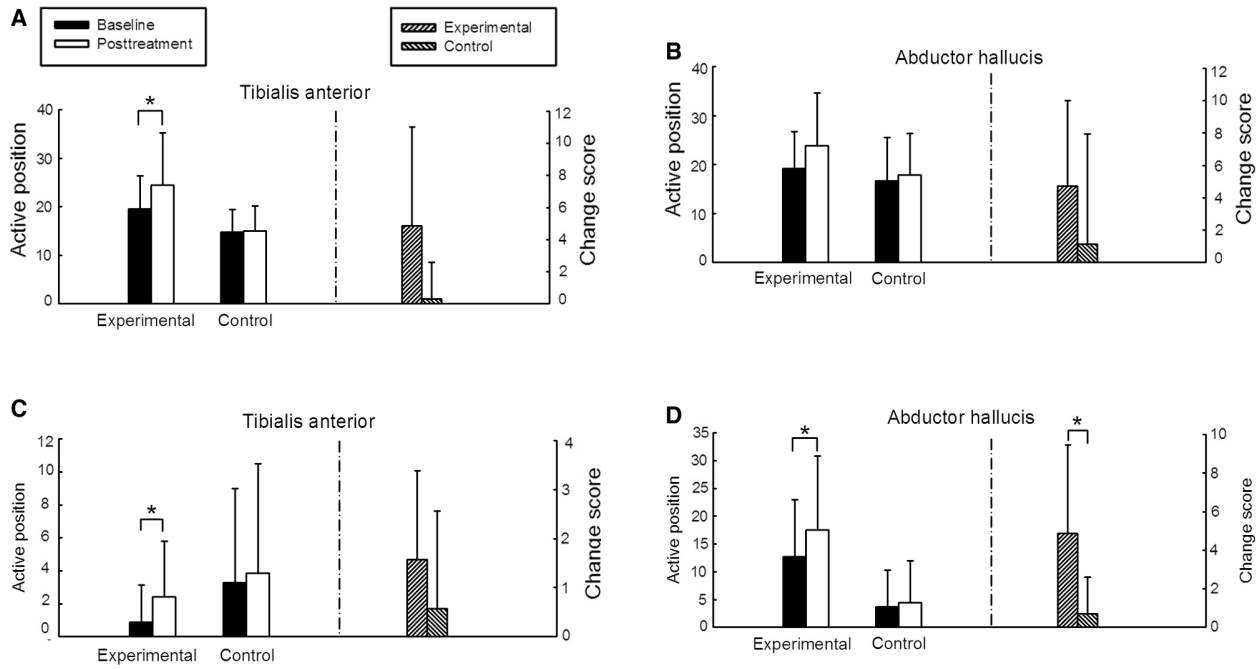

Figure 4. Size of the cortical motor output area in unaffected (A, B) and affected (C, D) hemisphere in the intragroup (left) and intergroup (right) comparisons. The map size for tibialis anterior muscle in the unaffected hemisphere was increased in the experimental group (*$P < .05$) (A, left). The map size for tibialis anterior (C, left) and abductor hallucis (D, left) muscles in the affected hemisphere was enlarged in the experimental group (*$P < .05$). The enlargement of map size for the abductor hallucis muscle (D, right) was larger in the experimental group than that in the control group (*$P < .05$). Values are mean ± standard deviation.

Map size. In the unaffected hemisphere, the map size for TA in the control group did not show a significant difference (from 14.71 ± 4.68 to 15.0 ± 5.16, $P = .608$) in the intragroup comparison (Figure 4A, left). The map size for TA in the experimental group showed a significant increase (from 19.57 ± 6.75 to 24.43 ± 10.75, $P = .034$) in the intragroup comparison (Figure 4A, left). There was no significant difference in the change score of the map size for TA ($P = .091$) between the groups (Figure 4A, right). The map size for AH in the control group did not show a significant difference (from 16.71 ± 8.79 to 17.86 ± 8.51, $P = .5$) in the intragroup comparison (Figure 4B, left). In the experimental group, the map size for AH (from 19.14 ± 7.6 to 23.86 ± 10.79, $P = .058$) showed an increasing trend (Figure 4B, left). There was no significant difference in the change score of the map size for AH ($P = .304$) between the groups (Figure 4B, right).

In the affected hemisphere, the map size for TA in the control group did not show a significant difference (from 3.29 ± 5.68 to 3.86 ± 6.64, $P = .655$) in the intragroup comparison (Figure 4C, left). In the experimental group, this value was significantly higher (from 0.86 ± 2.27 to 2.43 ± 3.36, $P = .039$) in the intragroup comparison (Figure 4C, left). There was no significant difference in the change score of the map size for TA ($P = .074$) between the groups (Figure 4C, right). The map size for AH in the control group did not show a significant difference (from 3.71 ± 6.58 to 4.43 ± 7.57, $P = .317$) in the intragroup comparison (Figure 4D, left). In the experimental group, this value showed a significant increase (from 12.71 ± 10.23 to 17.57 ± 13.21, $P = .042$) in the intragroup comparison (Figure 4D, left). There was a significant difference in the change score of the map size for AH ($P = .047$) between the groups (Figure 4D, right).

CoGs. The CoGs showed almost similar results in both hemispheres between the groups. Previous studies indicated that minor shifts (mean 3 to 4 mm) could be due to technical limitations.[25] The average variability of the CoGs in both hemispheres was 0.68 cm for the normal participants.[37] The normal variability of the CoGs in stroke patients was 0.99 ± 0.26 cm in the affected side and 0.55 ± 0.36 cm in the unaffected hemisphere.[22] Therefore, our results were within the limits of normal variability.

Correlation Between Motor Performance and Corticomotor Activity

Improvement in the motor threshold for TA in the unaffected hemisphere positively correlated with the increase in the BBS score ($r = .839$, $P = .018$). The increase in the map size for TA in the unaffected

hemisphere also correlated significantly with the improvement in the step length of the unaffected limb ($r = .757$, $P = .049$).

DISCUSSION

This is the first study to demonstrate that the additional BWSTT could improve motor performance and induce changes in corticomotor excitability. In the present study, walking performance could be improved in both groups (Table 2). However, improvements in the walking speed and step length in the experimental group were greater than those in the control group (Table 2). No significant changes in corticomotor excitability were observed in the control group (Figures 3 and 4). In contrast, the motor threshold for TA in the unaffected hemisphere, map size for TA in both hemispheres, and map size for AH in the affected hemisphere were significantly improved in the experimental group (Figures 3 and 4). Moreover, there were significant differences between the groups with regard to the change scores of the motor threshold for TA in the unaffected hemisphere and map size for AH in the affected hemisphere. Based on these findings, it appears that BWSTT in combination with general physical therapy is more effective in improving motor performance and inducing changes in corticomotor excitability as compared to general physical therapy alone. Furthermore, our results showed that the improvement in motor performance may be related to the change in corticomotor excitability in patients with chronic stroke. Our findings suggest that stroke duration may not be a strong limiting factor. Similar results were reported in a study by Carey et al.[25]

The participants in the experimental group showed improved motor performance. Previous studies also reported positive results.[11-14,16] However, the improvements in our study were less than those in previous studies probably due to the fact that the participants had better balance and gait ability at the baseline level in our study. Thus, it would have been difficult to observe a greater degree of improvement in our study. Furthermore, both groups showed improvement in speed, but different strategies may be used between the 2 groups. While participants in the experimental group may increase the step length for greater speed, those in the control group may increase the cadence to enhance their speed.

There is limited information regarding the change of motor threshold and map size following lower-extremity training in this regard. Our results showed that in the unaffected hemisphere, the motor threshold was significantly decreased (Figure 3A, left), and the map size was significantly increased (Figure 4A, left) for the TA muscle in the experimental group. In contrast, those for AH did not show any differences between baseline and posttreatment assessments. The discrepancies in the results from TA and AH may be explained in 3 ways: first, there may be greater activation of the corticospinal input to the TA as compared to the gastrocnemius when walking on a treadmill;[18] second, there may be greater emphasis on normal gait patterns such as 0-degree dorsiflexion on the swing phase during BWSTT in the present study; and third, activation of AH barely contributes to ambulation.[38]

After a stroke, brain excitability in the affected hemisphere is decreased.[21] This is probably due to the injury itself and disuse.[23] In the affected hemisphere, our results revealed that the motor threshold for the AH muscles remained unchanged after training (Figure 3D). In previous studies, the motor threshold of the affected hemisphere also remained unchanged in studies using constraint-induced movement therapy (CIMT).[36,39-41] However, the motor threshold of the affected hemisphere for a hand muscle was significantly decreased at the last training session in a study by Koski et al.[22] Recruitment of TA is more difficult than that of other muscles in the lower extremity.[38] Our results showed that only 1 participant in the experimental group could induce the MEPs of TA with 100% of the maximum stimulator power output at the baseline level. Although we could not observe a change in the motor threshold for TA after training (Figure 3C), 4 additional participants in the experimental group elicited MEPs with 100% maximum stimulator power output at posttreatment. This may indicate an improvement in the motor threshold for TA after additional BWSTT in the present study.

Additionally, our results showed that the map sizes for the TA and AH muscles in the affected hemisphere were increased from baseline to posttreatment after additional BWSTT (Figure 4 C,D). The change score of the map size for AH was higher in the experimental group than that in the control group. Immediate and long-term positive effects have also been shown in previous studies.[23,36,39] Although our results corresponded to those obtained in previous studies, the percentage of increase appeared to be lesser in our study. This may be attributable to the difference in the training intensity. In CIMT, the participants are instructed to wear a resting splint for a target duration of 90% of their waking hours. Furthermore, the participants also received "shaping" training 6 hours per day for using the affected arm.[36] Therefore, the training intensity of CIMT is likely greater than that of the intervention used in the present study. This may explain the lesser enlargement obtained in our study.

Although recruitment of TA is difficult, we found an improvement in the motor threshold and map size for TA in both hemispheres after additional BWSTT. These results may indicate enhanced neuronal excitability in

both hemispheres, perhaps because BWSTT is an intervention for bilateral lower extremities. In contrast, the present results showed an increase in the AH map size in the affected hemisphere, while the contralateral cortical representation area remained unchanged; the motor thresholds remained identical. This discrepancy between the changes in map size and motor threshold indicates that the main increases in excitability occurred at the border rather than the center of the representation area. Moreover, we observed that the map size for AH in the affected hemisphere was smaller than that in the unaffected side before training. However, the map size for AH in the affected hemisphere was similar to that in the unaffected side after BWSTT. Bilateral ambulation training is probably an important factor to achieve symmetrical excitability of both hemispheres.

The CoGs did not shift in the present study. However, the map size was increased. These results were consistent with those of a previous study by Liepert et al.[23] The authors explained that the motor map may expand in all directions to a similar degree.[23] Byrnes et al[42] indicated that shifts in the mediolateral axis may suggest reorganization within the primary motor cortex, while anteroposterior shifts were present in keeping with the recruitment of the premotor or postcentral cortex. Therefore, these results suggest the recruitment of a new area over the affected hemisphere. However, the results of the motor map in the present study must be interpreted with caution. If the patient's motor threshold decreases after training, the stimulus intensity relative to the baseline threshold increases posttreatment. Therefore, the increases in the motor map's values after training possibly occurred in part due to the same stimulus intensity being used at baseline and posttreatment. Further studies are required to discern the changes in the motor map values.

In the present study, we could not differentiate between cortical and subcortical changes that occurred following additional gait training. However, training-induced changes in cortical[18,27] and subcortical[43] excitability have been demonstrated. It has been proposed that motor learning possibly relays on integrated activity at multiple sites in the cortex and subcortical structures.[26,43] Therefore, it is possible that changes in both cortical and subcortical excitability occurred following additional gait training.

In this study, we attempted to clarify whether some of the physiological changes in the corticomotor pathways would accompany functional improvement after stroke. According to our results, interesting correlations were observed between the improvements in TMS measures for TA in the unaffected hemisphere and functional improvement. These results implied that functional improvement may be partially due to the increase in excitability over the unaffected hemisphere. On the other hand, Koski et al[22] found that the amount of change in the affected side measures correlated with the amount of improvement in hand/arm function. In contrast, we could not detect any correlation between the change in TMS measures of the affected hemisphere and functional improvement in the present study. This may be attributable to the fact that only a small number of patients could induce MEPs for the target muscles in the affected hemisphere. However, TMS measures were derived from some specific muscles, whereas motor performance was contributed by many muscles.[22] Further studies are required to clarify these issues.

This study has several limitations. First, the sample size used in this trial was small, which implies that caution should be exercised when interpreting the results. Second, since we had limited resources, this study did not use a blinded assessor. Bias in study results was minimized by the use of standard instructions during testing. Third, there was limited information on the time the participants spent out of therapy for improvement. Fourth, we did not use a control for the additional training. The participants in the control group did not receive any "placebo" intervention. Also this study lacks follow-up data. We have not determined whether the participants maintained changes found in our study. We suggest that future studies control these potential sources of bias.

CONCLUSION

Our results showed that additional BWSTT may demonstrate effects on motor performance and induce changes in corticomotor excitability in patients with chronic stroke. Improvement in motor performance may be related to the change in corticomotor excitability. Furthermore, additional BWSTT may be recommended for chronic stroke patients with abnormal gait patterns and insufficient balance ability.

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