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THE RELATIONSHIPS BETWEEN TRUNK FUNCTION AND THE FINDINGS OF TRANSCRANIAL MAGNETIC STIMULATION AMONG PATIENTS WITH STROKE

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In this study, we investigated the relationship between the motor evoked potentials obtained from trunk muscles and the clinical function of trunk muscle. Twenty patients with unilateral hemispheric stroke and 11 healthy adults were examined. The responses of the bilateral external oblique muscles and the erector spinae muscles to the magnetic stimulation of multiple sites over both cortical hemispheres were recorded. Trunk muscle performance was assessed using the Trunk Control Test and Stroke Impairment Assessment Set. In the stroke group, stimulation of the affected hemisphere resulted in a motor evoked potential in only one patient, while the other 19 stroke patients produced no response to stimulation of the affected hemisphere. Stimulation of the unaffected hemisphere evoked bilateral responses in 19 patients. Further, stimulation of the unaffected hemisphere in the stroke group produced larger motor evoked potentials in the ipsilateral muscles than the motor evoked potentials recorded in the ipsilateral muscles of the control group. The clinical assessment scores of trunk function (i.e. Trunk Control Test and trunk items of Stroke Impairment Assessment Set) were correlated with the amplitudes of the motor evoked potentials of the ipsilateral external oblique muscle that were evoked by stimulation of the unaffected hemisphere. Our results suggest that the recovery of trunk function after stroke is associated with an increase in ipsilateral motor evoked potentials in the external oblique muscle upon stimulation of the unaffected hemisphere, suggesting a role for compensatory activation of uncrossed pathways in recovery of trunk function.

Key words: transcranial magnetic stimulation, stroke, trunk muscle.

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INTRODUCTION

A stroke or other injury to the motor cortex results in weakness and paralysis in the contralateral limbs and axial musculature. Often, however, a gradual return of some motor abilities occurs in weeks and months after injury. The severity of trunk impairment is usually less than more distal musculature. Poor recovery of trunk muscle performance results in a severe disability and a reduction in the activities of daily living (ADL) (1). In stroke rehabilitation, trunk muscle performance is an important factor in predicting the functional outcome (1–3). An improvement in trunk function is not always matched by an improvement in the degree of hemiparesis (4).

Few studies have investigated the impairment of trunk muscle performance. However, there is considerable literature addressing limb muscle performance after stroke (5–7). Some studies have assessed trunk muscle performance as the ability to control balance in sitting and standing, trunk movement, and trunk muscle strength (1, 2, 8–12). Trunk muscle performance is considered to be less affected after stroke than the performance of the upper and lower extremities because trunk muscles are innervated by both hemispheres (13). However, the role of the unaffected hemisphere in the recovery of trunk muscle performance has not been clarified.

The transcranial magnetic stimulation (TMS) technique has been used to elicit responses in arm and leg muscle under various conditions (14–18). This technique may offer new insights into the actual contribution of contra- and ipsilateral descending motor pathways to residual motor functions after unilateral brain damage (15, 16). Few studies have analyzed the response of trunk muscles in patients after a hemispheric lesion. The aim of this study is to examine the relationship between the motor evoked potentials (MEPs) obtained from trunk muscles and the clinical performance of trunk muscle.

MATERIAL AND METHODS

Subjects

The clinical details of the stroke patients are shown in Table I. Twenty patients, exhibiting symptoms of hemiparesis caused by cerebrovascular disease, and 11 healthy adults (9 males and 2 females) participated in this study. The mean age of the stroke patients was 63.9 years (range 47–73 years) and the mean age of the healthy adults was 44.2 years (range 28–62 years). Patients who were admitted for post stroke rehabilitation at our rehabilitation center were recruited. The mean time from stroke onset was 3.1 months (range 1–8 months). All recruited patients satisfied the inclusion criteria of (1) being capable of giving informed consent, (2) having no other neurological disease, and (3) having no contraindications to undergoing TMS (19). The site and type of strokes were identified before the study in all patients using magnetic resonance imaging (MRI). Twelve patients had an involved left hemisphere and 8

Table I. *Clinical details of subjects*

Patient/sex/age	Lesion	SIAS-U	SIAS-L	Vert	Abd	TCT	Time (month)
1 / M / 69	Rt sub	1	5	1	1	24	4
2 / M / 68	Lt sub	3	6	3	2	61	3
3 / M / 68	Lt sub	0	6	3	2	74	3
4 / M / 59	Rt cort	1	5	2	1	24	8
5 / M / 59	Lt sub	1	5	3	2	74	4
6 / M / 64	Rt sub	0	0	0	0	0	1
7 / M / 72	Lt sub	7	7	3	2	74	3
8 / M / 56	Lt sub	10	15	3	3	100	2
9 / M / 59	Rt cort	1	5	3	2	74	4
10 / M / 56	Lt sub	1	4	3	2	62	2
11 / M / 65	Rt sub	1	6	3	2	74	3
12 / M / 69	Rt sub	0	2	2	2	48	3
13 / M / 47	Rt sub	3	6	3	3	87	2
14 / M / 73	Lt sub	8	14	3	3	87	2
15 / M / 69	Lt cort	0	0	1	0	12	1.5
16 / M / 71	Lt cort	0	0	2	2	61	8
17 / F / 71	Lt sub	0	0	0	0	0	1
18 / M / 53	Lt cort	3	7	3	2	74	4
19 / M / 66	Lt cort	1	4	3	2	37	2.5
20 / M / 64	Rt sub	0	0	1	0	12	2

Rt = right; Lt = left; Sub = subcortical lesion; Cort = cortical lesion; SIAS-U and SIAS-L = the score of SIAS motor score of upper extremity and lower extremity, respectively; Vert = the score of SIAS verticality item; Abd = the score of SIAS abdominal muscle strength item; TCT = the score of Trunk Control Test; Time = time from stroke onset.

patients had an involved right hemisphere. Fourteen patients had a subcortical lesion and 6 patients had a cortical lesion.

The study was approved by the local ethics committee and a full explanation of the experiment was given to subjects and/or close relatives, and written consent was obtained.

Transcranial magnetic stimulation (TMS)

Electromyographic activities were recorded from the external oblique and erector spinae muscles on both sides using Ag-AgCl surface electrodes placed with their centers 20 mm apart over the muscle bellies. Surface electrodes of the external oblique muscle were placed on the upper lateral quadrant of the abdomen below the costal margin and parallel with the external oblique muscle fibers, with one electrode on each side of the mid-clavicular line (20). Electrodes of the erector spinae muscle were placed 3.5 cm lateral to the 3rd–4th lumbar vertebra with a cranio-caudal montage of recording electrodes (13). Plassman & Gandevia (20) recorded the potentials of external oblique muscle with surface electrodes as we did. They also recorded the potentials of external oblique muscle using pairs of bipolar hook-wire electrodes. Responses of similar latencies were recorded with the two types of electrodes. Given the orientation of the electrodes, their proximity to the external oblique muscle, and the results of intramuscular recordings, it is likely that these surface electrodes recorded the potential predominantly from the external oblique with a contribution from the internal oblique.

Magnetic stimulation of different sites on the motor cortex was performed using a Dantec MagLite with a figure of 8 shaped coil. The stimulator possessed a maximum output voltage of 1.8 KV and a peak magnetic field of 1.9 T, according to the manufacturer's specifications. Stimulus intensity was 100% of the maximum output of the stimulator in all subjects. The stimulator coil was positioned tangential to the scalp with the handle in an antero-posterior orientation and the center of the figure eight over the site to be stimulated. We determined vertex (Cz) according to the international 10/20 system. Subjects wore swimming caps marked lateral, anterior, and posterior to Cz on the scalp in 2 cm steps on both hemispheres. Three to four stimuli were applied at each spot. Twenty scalp sites for each hemisphere were stimulated.

We measured peak-to-peak amplitude and the latency of the first initial negative or positive response of the largest response. When no response to stimulation was obtained, a zero amplitude value was recorded. MEPs were recorded with a Neuropack 8 (Nihon Koden Co.) amplified with a band pass filter set at 2 Hz–3 KHz. The responses were stored on a hard disk and later measured and plotted. All recordings were made while subjects were sitting on a comfortable semi-reclining chair.

In addition to cortical stimulation, nerve root stimulation to the target muscles was performed by positioning the coil over the spinous process of Th9–L1, where the maximal response of target muscles can be obtained (20). During the root stimulation, subjects were sitting without a backrest. Patients who were not able to maintain posture without a backrest were provided manual assistance. We defined the amplitude of the MEP obtained by root stimulation as the root MEP. Each amplitude of MEP, gained by TMS, was described as a percentage of the root MEP (MEP ratio) (Fig. 1).

TMS was performed on both the affected and unaffected hemisphere. We defined the MEPs of the ipsilateral side muscles from the stimulation site as ipsilateral MEP and those of contralateral side muscles as contralateral MEP. All 20 patients were studied within the first week of admission (T1). Ten of the patients underwent a second study within two weeks before discharge (T2). The mean study interval was 3 months.

Construction of topographical mapping

The scalp sites of stimulation where represented the maximal ipsilateral and contralateral MEPs of target muscles were plotted. We made the scalp mapping of the distribution of maximal ipsilateral and contralateral MEP among controls and stroke group. The area was described as the sum of the number of scalp grids where elicited MEPs of the target muscle. We counted the area of ipsilateral and contralateral external oblique muscle among control and stroke group.

Clinical assessment

Trunk muscle performance was assessed using the Trunk Control Test (TCT) (1) and trunk control portions of the Stroke Impairment Assessment Set (SIAS) (9). The inter-rater reliability and the validity of TCT and SIAS have already been reported (1, 3, 9, 21–23). TCT assesses axial movement by rolling from a supine position to the weak side and to the strong side, sitting up from a lying down position, and sitting in a balanced position on the edge of the bed with feet off the floor for 30 seconds. The scoring is as follows: 0, unable to perform movement without assistance; 12, able to perform movement but in an abnormal manner; and 25, able to complete movement normally. The

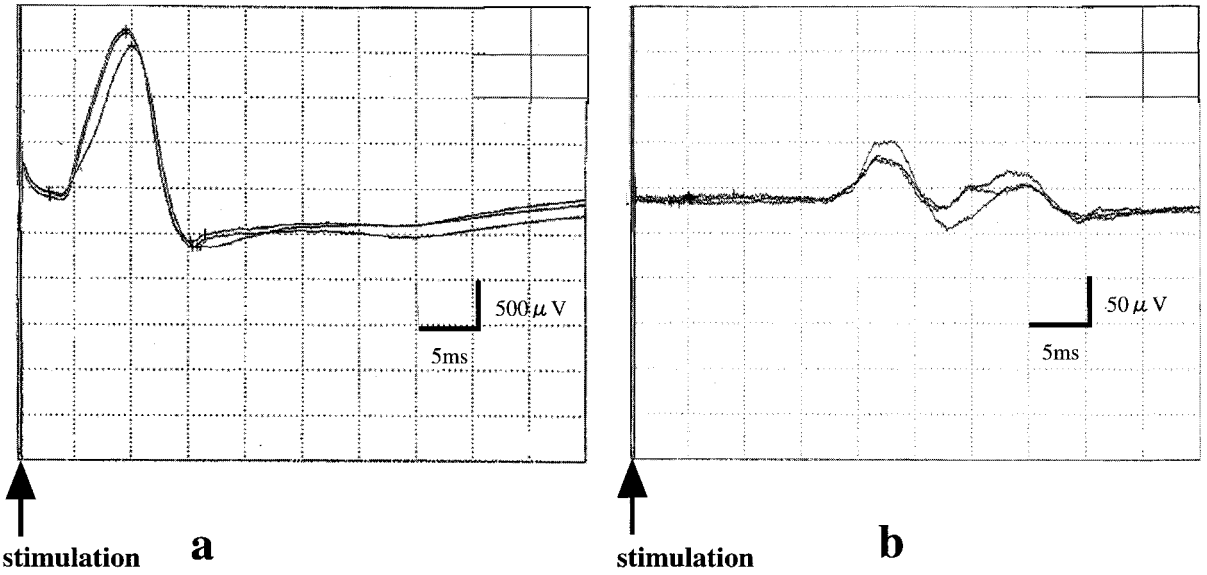

Fig. 1. Electromyographic responses for one control subject recorded with surface electrodes from the external oblique muscle following stimulation at the level of 10th thoracic spinous process (a) and transcranial stimulation (b).

TCT score is the sum of the scores obtained on the four tests (range 0 to 100).

The trunk control portion of the SIAS consists of vertical balance and abdominal muscle strength. Each category was scored 0 to 3. In the vertical balance test, a score of 0 is given if the patients cannot maintain a sitting position. When a sitting position can only be maintained while tilting to one side and the patient is unable to correct the posture to the erect position, the patient is assigned a score of 1. A score of 2 indicates that the patient can sit vertically when reminded to do so. If the patient can sit vertically in a normal manner, this is scored as 3. The abdominal muscle strength was evaluated with the patient resting in the 45° semireclining position in either a wheelchair or a high-back chair. The patient is asked to raise the shoulders off the back of the chair and assume a sitting position. If the patient is unable to sit up, the score is 0. A score of 1 indicates that the patient can sit up provided there is no resistance to the movement. If the patient can come to the sitting position despite pressure on the sternum by the examiner, a score of 2 is given. A score of 3 means that the patient has good strength in the abdominal muscles and is able to sit up against considerable resistance.

Motor function of the affected extremities was assessed using the SIAS motor function test. The patients were examined by the same researcher in the first (T1) and the second (T2) clinical examination.

Statistical analysis

Differences between ipsilateral and contralateral MEPs were tested for statistical significance using the two-tailed Student *t*-test. We also compared between the stroke group and the control group using the unpaired two-tailed Student *t*-test. The correlation between MEP parameters and the clinical assessments was calculated with the Spearman's rank correlation. A *p*-value of less than 0.05 was considered statistically significant.

RESULTS

Control group

Contralateral MEPs in the external oblique muscle were recorded in all subjects. Right hemisphere stimulation evoked bilateral MEPs in the external oblique muscle in 8 of 11 subjects. Left hemisphere stimulation evoked bilateral MEPs in the external oblique muscle in 5 of 11 subjects.

The mean ipsilateral MEP ratio of the external oblique muscle was 9.09 ± 12.19, and the mean contralateral MEP ratio was 17.65 ± 12.92. The difference in the MEP ratio between the ipsilateral and the contralateral MEPs was statistically significant ($p < 0.05$). The mean latency of the ipsilateral and the contralateral MEPs was 17.06 ± 3.57 and 15.68 ± 2.75 ms, respectively. However, this difference was not statistically significant.

Contralateral MEPs in the erector spinae muscle were recorded in all muscles. Right hemisphere stimulation evoked bilateral MEPs in the erector spinae muscle in 6 of 11 subjects. Left hemisphere stimulation evoked bilateral MEPs in the erector spinae muscle in 5 of 11 subjects. The mean MEP ratio of the ipsilateral erector spinae muscle was 5.21 ± 11.22, and that of the contralateral erector spinae muscle was 8.41 ± 11.7. The difference in the MEP ratio between the ipsilateral and the contralateral MEPs was not significant. The mean latency of the ipsilateral and the contralateral MEPs was 15.19 ± 2.85 and 15.93 ± 4.86 ms, respectively. This difference was not statistically significant.

Subject age showed no correlation with the MEP ratio or the latency of both muscles among the control group.

Stroke group

At T1, TMS of the affected hemisphere failed to evoke a response in either the ipsilateral or contralateral external oblique and erector spinae muscles, except in one patient. However, stimulation of the unaffected hemisphere evoked a bilateral response in 19 patients. TMS of the affected hemisphere evoked a response in both the ipsilateral and contralateral external oblique and erector spinae muscles in one patient, whose trunk control test was a full score.

The mean MEP ratio of the ipsilateral external oblique muscle

Table II. *The mean motor evoked potential (MEP) ratio of the control group and the stroke group (mean ± S.D.). The MEP ratios of stroke group were obtained by the stimulation of the non-affected hemisphere. Statistical analysis was performed by using paired two-tailed Student* t*-test (ipsilateral vs contralateral) and unpaired two-tailed Student* t*-test (control vs stroke)*

	Ipsilateral MEP ratio	Contralateral MEP ratio	
External oblique			
Control ($n = 11$)	9.09 ± 12.19	17.65 ± 12.92	$p < 0.05$
Stroke ($n = 20$)	34.82 ± 23.99	64.97 ± 33.00	$p < 0.01$
	$p < 0.01$	$p < 0.01$	
Erector spinae			
Control ($n = 11$)	5.21 ± 11.22	8.41 ± 11.70	n.s
Stroke ($n = 20$)	13.63 ± 14.07	25.68 ± 20.93	$p < 0.05$
	$p < 0.05$	$p < 0.01$	

n.s. = not significant.

was 34.82 ± 23.99, and the mean MEP ratio of the contralateral external oblique muscle was 64.97 ± 33. The difference in the MEP ratios between the ipsilateral and the contralateral MEP was statistically significant ($p < 0.01$). The mean latency of ipsilateral MEPs was 19.45 ± 2.85 ms, while the mean latency of contralateral MEPs was 16.61 ± 3.16 ms. The difference in latency was statistically significant ($p < 0.01$).

The mean MEP ratio of the ipsilateral erector spinae muscle was 13.63 ± 14.07, and the mean MEP ratio of the contralateral erector spinae muscle was 25.68 ± 20.93. The difference was statistically significant ($p < 0.05$). The mean latency of the ipsilateral and the contralateral MEPs were 17.75 ± 3.92 ms and 15.72 ± 3.80 ms, respectively. The difference in latency was not statistically significant.

A comparison between the ipsilateral MEP ratio of the stroke group and the control group was shown in Table II. The ipsilateral and contralateral MEP ratio resulting from stimulation of the unaffected hemisphere were significantly increased compared with the control group in both the external oblique ($p < 0.01$) and the erector spinae muscle ($p < 0.05$).

Subject age showed no correlation with the MEP ratio or the latency of both muscles among the stroke group.

Relationships between MEPs and the clinical assessments

At T1, the MEP ratio of the ipsilateral external oblique muscle, with non-involved hemisphere stimulation, was correlated with the TCT score, the SIAS abdominal muscle strength, and the SIAS verticality (Figs 2 and 3). The MEP ratio of the ipsilateral erector spinae muscle was not statistically correlated with the clinical assessment. In addition, no correlation was observed between latency and clinical scores.

Time course of clinical assessments and MEPs

Ten patients were examined at T1 and T2. At T1, no MEPs were recorded from stimulation of the affected hemisphere. At T2, stimulation of the affected hemisphere evoked MEPs in only one patient. The other patients displayed no detectable change in the MEP representation of the affected hemisphere. The change in the ipsilateral MEP ratio of the external oblique muscle and the TCT score was shown in Table III. The MEP ratio of the ipsilateral external oblique muscle and the TCT score at T2 were significantly increased compared with those of T1 ($p < 0.01$) (Table III). In addition, the MEP ratio of the ipsilateral external oblique muscle at T2 was statistically correlated with the TCT score at T2 ($p < 0.01$). Spearmann's rank correlation efficient was 0.77.

Topographical mapping

Fig. 4 showed the distribution of the scalp site elicited the maximal MEP of external oblique muscle. In both control and stroke group, most of the maximal MEPs of contralateral and ipsilateral external oblique muscles were evoked within 2–4 cm lateral and 0–4 cm anterior from Cz.

The mean areas of ipsilateral external oblique muscle of controls, stroke group at T1 and T2 were 2.1 ± 1.7, 3.1 ± 2.1 and 3.4 ± 1.5. The difference between control and stroke group was not significant. In the stroke group, the difference between T1 and T2 was not significant. The mean area of contralateral external oblique muscle of control, stroke group at T1 and T2 were 4.1 ± 1.6, 4.1 ± 1.9 and 4.0 ± 1.4, respectively. We found no significant difference among those three groups.

DISCUSSION

Some studies have examined the cortical representation of trunk muscles in healthy adults (13, 20), but few studies have examined these parameters in stroke patients. Previous studies of the human cortical representation of trunk muscles, using either TMS or transcranial electrical stimulation, have shown that the cortical pathways to the trunk muscles were represented bilaterally in the hemispheres, although contralateral pathways were considered dominant (13, 20). In stroke patients, the cortical representation of trunk muscles has not been established clearly, and the objective assessment of trunk muscle impairment had not been performed. Further, the factors that determine the recovery of trunk impairment remain unclear. In this study,

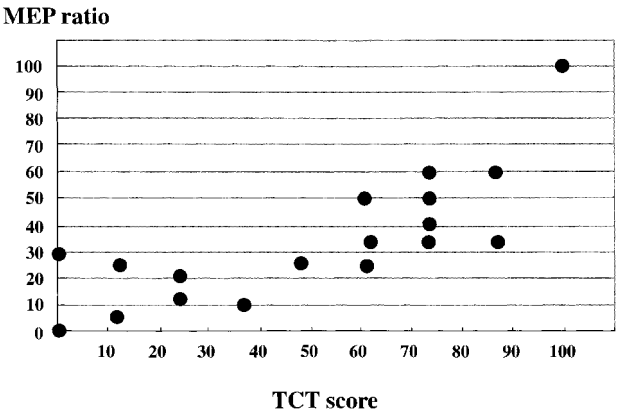

Fig. 2. The relationship between the motor evoked potential (MEP) amplitude of the ipsilateral external oblique muscle with stimulation of the non-affected hemisphere and trunk control test (TCT). The MEP ratio of the ipsilateral external oblique muscle with stimulation of the non-affected hemisphere was statistically correlated with the score of TCT using Spearman's rank correlation ($R = 0.82$, $p < 0.01$).

we compared the MEPs of trunk muscles between a healthy control group and a stroke patient group. In order to clarify the possible role of the intact hemisphere in motor recovery of the trunk muscles, we examined the relationship between the MEPs obtained from trunk muscles and their clinical assessed trunk muscle function.

In healthy adults, we found ipsilateral MEPs in both the external oblique and the erector spinae muscles. The appearance of the ipsilateral MEPs was less frequent than that of the contralateral MEPs. We presume that an ipsilateral projection to trunk muscles exists, however, the trunk muscles are innervated predominantly by projections from the contralateral hemisphere, as reported previously (13). The innervation is most probably mediated by similar rapid conduction cortico-spinal pathways, as described in previous studies of trunk muscles (13, 19).

In most of the patients, stimulation of the affected hemisphere failed to evoke MEPs in trunk muscles, although stimulation of the unaffected hemisphere evoked a bilateral response in trunk muscles. The ipsilateral MEPs, resulting from stimulation of the non-affected hemisphere, had an increased MEP ratio in comparison with the ipsilateral MEPs of the control group. The latency of ipsilateral MEPs of the external oblique muscle was statistically longer than the contralateral latency among the stroke patients. The latency of ipsilateral MEPs was approximately 2 ms longer than the latency of the contralateral MEPs. This result may support the observation that the ipsilateral projection is mediated by the polysynaptic tract, rather than the direct corticospinal tract. These various pathways most probably exhibit different conduction times, giving rise to ipsilateral responses with variable latencies. The longer latency of ipsilateral response in stroke patients may be mediated by either an ipsilateral cortico-reticulospinal or other polysynaptic pathways to compensate for the affected tract (15).

Our results reveal that the MEP ratio of the ipsilateral external oblique muscle resulting from stimulation of the non-affected hemisphere correlates with the clinical assessment. Further, the MEP ratio increased as the clinical assessment scores improved. The cortical projection from the affected hemisphere, however, did not change. These results suggest that the recovery of trunk performance resulted from the non-affected cortical projection.

Clinical observations, TMS, and positron emission tomography have suggested a role for pathways to the ipsilateral muscles in the recovery from hemiparesis due to a central lesion (15, 16, 24–26). However, some studies have suggested that these mechanisms do not help in motor recovery of the upper extremity, as the presence of ipsilateral responses after TMS are not correlated with clinical improvement (27). The relative paucity of preexisting ipsilateral projections to the limb muscles may account for these observations.

In this study, both ipsilateral and contralateral MEP ratios of stroke group were greater than those MEP ratios of control. The maximal MEPs of ipsilateral external oblique muscle were evoked by the stimulation at the sites of 2–4 cm lateral and 0–4 cm anterior from Cz among the control and stroke group. These results suggested that the recovery of trunk function might

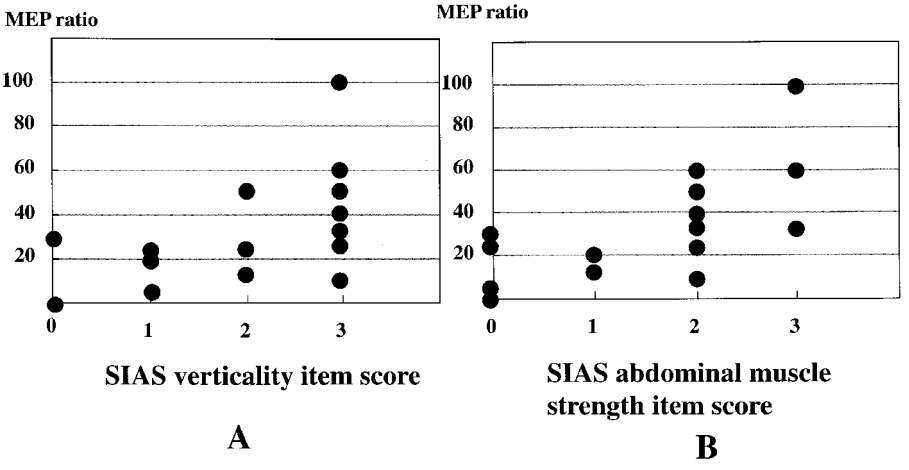

Fig. 3. The relationship between the score of the stroke impairment assessment set (SIAS) verticality item (A) and abdominal muscle strength item (B) and the amplitude of the ipsilateral motor evoked potential (MEP) in the external oblique muscle with stimulation of the non-affected hemisphere. The score of the SIAS verticality item was statistically correlated with the MEP ratio of the ipsilateral external oblique muscle with stimulation of the non-affected hemisphere ($R = 0.62$, $p < 0.01$ and $R = 0.69$, $p < 0.01$, respectively).

Table III. *The mean ipsilateral motor evoked potential (MEP) ratio of external oblique muscle and the mean score of trunk control test (TCT) at T1 and T2* (n = *10). Statistical analysis was performed by using paired two-tailed Student* t-*test (T1 vs T2)*

	T1	T2	
Ipsilateral MEP ratio	40.40 ± 15.52	67.00 ± 23.94	$p < 0.01$
TCT score	61.70 ± 28.11	83.10 ± 12.33	$p < 0.01$

be due to compensatory activation of preexisting uncrossed pathways but not cortical reorganization.

Our results predict that recovery of trunk function, after a restricted mono-hemispheric lesion, is possible without the recovery of the cortical motor projections from the affected hemisphere. The intact hemisphere is responsible for restoration of trunk function, most likely by potentiating the effects of preexisting uncrossed motor pathways. One potential mechanism is that preexisting uncrossed pathways are unmasked by a decreased intracortical inhibition (28). In addition, reorganization at the spinal level may contribute to these results. Further research is required regarding this issue.

Our results suggest no relationship between the MEP ratio of the erector spinae muscle and the TCT score. This finding may be due to the contents of test item of TCT. During rolling and sitting up, the motions were more dependent on external oblique muscle function, while erector spinae muscle activity does not act as a prime mover. In addition, the erector spinae muscles contribute little to the maintenance of erect posture. Electromyographic studies have shown that the erector spinae muscles are relaxed in ordinary sitting and standing unless a deliberate effort is made to extend the thoracic spine more completely (29).

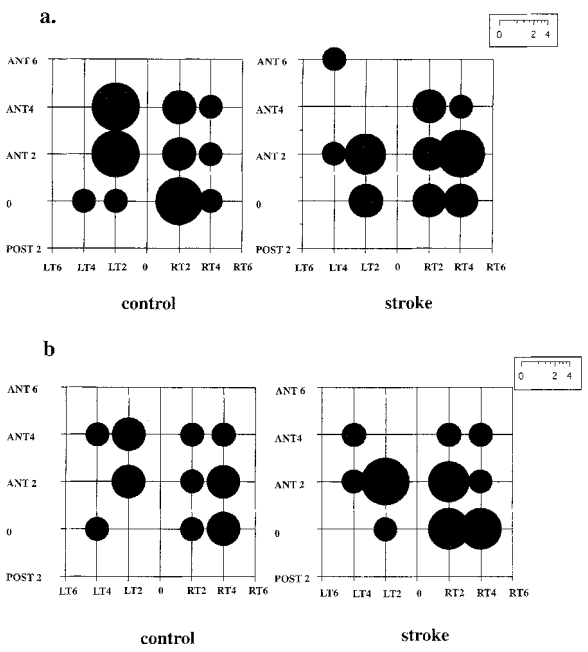

Fig. 4. The distribution of the scalp sites of maximal motor evoked potential (MEP) of external oblique muscle. The size of circle indicated the number of subjects who represented the maximal MEP at a given scalp position. The distribution of maximal MEPs of contralateral external oblique (a) and ipsilateral external oblique muscle (b) are shown. ANT 6; 6 cm anterior from vertex, POST 2; 2 cm posterior from vertex, RT 6; 6 cm lateral from vertex in right hemisphere, LT 6; 6 cm lateral from vertex in left hemisphere.

In conclusion, our results support the hypothesis that ipsilateral MEPs of the external oblique muscle, obtained by non-affected hemisphere TMS, represent a useful objective evaluation of trunk motor function in stroke patients. The improvement of clinical manifestations correlated with the ipsilateral trunk MEPs resulting from stimulation of the non-affected hemisphere. However, the cortical representation of the affected hemisphere did not change.

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