The Loss of Dexterity in the Bilateral Lower Extremities in Patients With Stroke

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The aim of this study was to examine the dexterity of both lower extremities in patients with stroke. Twenty patients with stroke and 20 age-matched control subjects participated in this study. To determine the dexterity of the lower extremities, we examined the ability to control muscle force during submaximal contractions in the knee extensor muscles using a force tracking task. The root mean square errors were calculated from the difference between the target and response force. The root mean square error was significantly greater in the affected limb of patients with stroke compared with those of the unaffected limb and the control subjects, and in the unaffected limb compared with that of the control subjects. Furthermore, the root mean square error of the affected limb was related significantly to motor function as determined by Fugl-Myer assessment. These results demonstrate impairment of the dexterity of both the affected and the unaffected lower extremities in patients with stroke.

Keywords: stroke, force control, motor skill, isometric

Motor deficits in patients with stroke may be evident in the extremities contralateral to the brain damage. The major contributor to physical disability is considered to be a loss of strength and dexterity. The loss of muscle strength of the lower extremities (LE) and upper extremities (UE) is an important predictor of functional disability, and is usually assessed during rehabilitation (Adams et al., 1990; Andrews & Bohannon, 2000; Bertrand et al., 2007). Previous studies have reported that the knee extensor (Flansbjer et al., 2006; Lindmark & Hamrin, 1995), ankle plantar flexor and hip flexor muscles (Hsu et al., 2003; Kim & Eng, 2003) of the affected LE are required to achieve fast walking speeds in patients with stroke. The relationships between arm function, elbow flexor force (Canning et al., 2004) and finger grip force (Boissy et al., 1999) have also been examined in patients with stroke.

A number of studies have examined the dexterity in the elbow, handgrip and finger-pinch in patients with stroke (Canning et al., 2000; Kriz et al., 1995). The results of these studies indicated that, in patients with stroke, the condition affected their grip force control, similar to muscular weakness (Hermsdorfer et al., 2003). The poor control of the grip force during lifting the manipulandum using the affected hand, correlated with UE function (McDonnell et al., 2006).

Little is known of the dexterity of the LE in patients with stroke (Wirth et al., 2008). Similar to the functional disability of the UE, injury to the central nervous system leads to a loss of dexterity in the affected LE. Dexterity can be defined as the ability to coordinate muscle activities to meet environmental demands, and is not restricted to manual tasks. To examine the dexterity of the LE, several studies examined the ability to control muscle force during submaximal contractions by using a force-tracking task (Christou & Carlton, 2002; Tracy et al., 2005). In the force-tracking task, the subject applies the force according to the visual feedback on the target and the actual response. The force tracking tasks, which are focused on both spatial accuracy in the force scaling and on temporal accuracy, can greatly increase the precision and objectivity in the assessment of skilled motor function. Studies using the force-tracking task have demonstrated that force control error and force variability during the isometric task increase with age (Christou & Carlton, 2001; Tracy et al., 2005). To our knowledge, no studies have examined the force control ability of the LE in patients with stroke.

The strength of the unaffected LE may be reduced in patients with stroke due to disuse (Hachisuka et al., 1997). The reduction of muscle strength by disuse is related to neural changes, such as a reduction in the number of motor units, a decrease in the maximal firing rate of the motor units, as well as muscle atrophy (Duchateau &
The loss of dexterity and strength may stem from the same underlying neural mechanisms. With respect to disuse, few studies have reported increased force fluctuation during submaximal isometric contraction following an unweighted condition (Clark et al., 2007; Shinohara et al., 2003). However, the loss of dexterity in the unaffected LE has not been clarified. The aims of this study were to determine whether there is a significant difference in the force control ability in the affected limb when compared with the unaffected limb and control subjects, to determine whether there is a correlation between the force control ability and the severity of the affected limb of patients with stroke, and to determine whether loss of force control ability in the LE becomes evident with increasing task difficulty.

Methods

Subjects

Twenty patients with stroke (age range 51–75 years, mean 63.9 ± 7.3 years) and 20 age-matched control subjects without a history of stroke (age range 50–77 years, mean 65.8 ± 7.2 years) participated in this study (Table 1). Patients with stroke were recruited from the outpatients and inpatients of a local hospital in Kagoshima, Japan. The control subjects were recruited from the community as volunteers. For hemiparetic patients, inclusion criteria were patients with a first time unilateral stroke and the ability to follow 3-step verbal commands to confirm the patient’s language understanding. The patients with stroke were excluded if they had major medical problems (active cardiac, systemic, or progressive neurological disease), a musculoskeletal disorder of the limbs, visual deficits, or hemi-neglect. Nineteen patients with stroke could walk 50 m without physical assistance. In our pilot study, a patient with very low maximum voluntary contraction (MVC) force of the affected knee extensor had difficulty completing the force-tracking task. Thus patients who could not produce at least 80 N of MVC force on the affected limb were excluded from participation. Sensorimotor function on the affected side was assessed using the LE portion of the Fugl-Myer stroke assessment instrument.

Control subjects with a history of LE musculoskeletal problems, cardiovascular illness, or arthritis in the LE joints were excluded. The demographic and clinical characteristic of the subjects are presented in Table 1. Before the investigation, all participants provided written informed consent for participation in the study. This study was approved by the Clinical Research Review Board of the Faculty of Medicine, Kagoshima University.

Instrumentation

During testing, subjects were seated on a chair in front of a computer screen. A stabilization strap was placed over the pelvis to eliminate extraneous movement, and participants were instructed to relax their arms on their thigh. A hand held dynamometer sensor (EG-230, SAKAI Inc., Tokyo, Japan) was used to measure muscle force during isometric MVC and force tracking tasks (Figure 1). The hand-held dynamometer sensor, which was firmly fixed to the chair, was attached just above the lateral malleolus during the test. The MVC force and force control ability were assessed in the knee extensor muscle at 90 degrees of knee flexion. The knee extension force was selected for its importance in functional task performance in patients with stroke (Flansbjer et al., 2006; Lindmark & Hamrin, 1995). The data acquisition system consisted of a laptop computer (WS3100xp, SOTEC Inc., Tokyo, Japan) with Microsoft Visual Basic 6.5 and Excel 2003 (Microsoft Inc., WA, USA). The output from the dynamometer transmitted to the computer through RS232C, and was sampled at 20 Hz.

Table 1  Subject characteristics, mean (SD)

<table>
<thead>
<tr>
<th></th>
<th>Unaffected</th>
<th>Affected</th>
<th>Control</th>
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<tbody>
<tr>
<td></td>
<td>(n = 20)</td>
<td>(n = 17)</td>
<td>(n = 20)</td>
</tr>
<tr>
<td>Age (years)</td>
<td>63.9 ± 7.3</td>
<td>65.0 ± 6.9</td>
<td>66.2 ± 6.9</td>
</tr>
<tr>
<td>Gender, male/female</td>
<td>14/6</td>
<td>13/4</td>
<td>14/6</td>
</tr>
<tr>
<td>MVC force (N/kg)</td>
<td>4.6 ± 1.2*</td>
<td>3.0 ± 1.3†</td>
<td>5.4 ± 1.5</td>
</tr>
<tr>
<td>Lesion type, ischemic/hemorrhagic</td>
<td>9/11</td>
<td>8/9</td>
<td></td>
</tr>
<tr>
<td>Affected side, left/right</td>
<td>6/14</td>
<td>5/12</td>
<td></td>
</tr>
<tr>
<td>Years since stroke (years)</td>
<td>2.0 ± 3.4</td>
<td>2.1 ± 3.6</td>
<td></td>
</tr>
<tr>
<td>FMA motor score</td>
<td>26.1 ± 6.9</td>
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<tr>
<td>FMA sensory score</td>
<td>7.5 ± 2.7</td>
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Note. MVC force, maximum voluntary contraction force; FMA score, Fugl-Meyer stroke assessment scores.
* p < .05, significant difference between affected.
† p < .05, significant difference between control subjects.
Procedure

The knee extension forces in the affected and unaffected limb were measured in the patients with stroke, and the same limb as the unaffected limb of the patients was measured in the control subjects. The MVC force was obtained before the tracking task. Three 5 s maximal contractions were recorded with a 30 s rest between each contraction. The peak trial was recorded and used for deciding the target force in the force-tracking task. To compare subjects, we calculated a strength-to-weight ratio by dividing the MVC force by body weight.

Force control ability was assessed using a force-tracking task with the data acquisition system providing real time visual feedback. The subjects adjusted their force output to match the moving black target line on the monitor and an on-line feedback of their response was reported by a red line. The target line corresponded to 0–20% of the MVC force allowing subjects to control force continually. The target force varied over a period of 60 s at frequencies of 0.3 Hz and 0.5 Hz. A previous study reported that the duration of the sit-to-stand movement was about 2.1 s in older adults and 2.6–3.1 s in patients with stroke (Cheng et al., 2004; Janssen et al., 2008; Lecours et al., 2008). The wavelength of the target force was therefore set to 2.0 and 3.3 s in this study. Figure 2 shows the representative data of the affected limb during the 0.3 Hz task and the 0.5 Hz task executed by a stroke patient. One or two practice sessions and three trials were completed for each task condition. Practice sessions were chosen to minimize the effects of learning for the tracking task and to reduce the opportunity for fatigue to develop. To minimize fatigue, participants were given a 90 s rest between each force-tracking task.

Data Analysis

The initial 10 s and final 10 s of force data from each trial were removed before analysis to avoid the initial force stabilization and/or premature cessation of force production (Sosnoff & Newell, 2006). We assessed the knee extensor force control ability by root mean square of error (RMS error) between the target force and response force. The RMS error was normalized by the mean target force to allow comparison between the results obtained from different subjects (Kurillo et al., 2004). A lower value of RMS error suggests increased force control. To quantify timing error, the time delay of force response to

Figure 1 — Schematic drawing of the force-tracking task. Force control ability was assessed using a data acquisition system providing real time visual feedback. Estimation of the force control ability was performed by analyzing the difference between the target and measured response forces.

Figure 2 — Representative data of the force tracking task during the 0.3 Hz (A) and the 0.5 Hz (B) task on the affected limb in patients with stroke.
the target force was calculated using the cross-correlation technique (Mello et al., 2007). A minimum of three force-tracking tasks were used to calculate each subject’s force control ability.

**Statistical Analyses**

One-way analysis of variance (ANOVA) was used to compare the MVC force among the affected and the unaffected limbs of patients with stroke and the control subjects. The effect of limb (affected limb versus unaffected limb versus control subjects) and the effect of task condition (0.3 Hz task versus 0.5 Hz task) on RMS error and timing error were determined using a two-way ANOVA with repeated measure. If an interaction (limb × task condition) was observed, a one-way ANOVA was applied to each task condition and differences between task conditions were assessed by Wilcoxon signed rank test for each limb. Each ANOVA analysis was followed by Tukey’s post hoc test. In addition, to investigate the relationship between RMS error and sensorimotor function in the affected limb, the Spearman test was used. All statistical tests were performed using SPSS 17.0 statistical software (SPSS Inc, Chicago, IL, USA). For all analyses, the level of significance was set at $p < .05$.

**Results**

Characteristics of the MVC force during knee extension are shown in Table 1. With respect to MVC force, the one-way ANOVA was significant ($F = 15.4, p < .001$). Post hoc analysis indicated that the MVC force of the affected limb of patients with stroke was reduced significantly compared with that of the unaffected limb ($p = .002$) and the control subjects ($p < .001$). The difference between the unaffected limb of patients with stroke and the control subjects was not significant ($p = .137$).

The results of the RMS error during the tracking task are shown in Figure 3. Two-way ANOVA revealed a significant effect of limb ($F(2, 54) = 11.5, p < .001$) and task condition ($F(1, 54) = 30.9, p < .001$), and no significant interaction between limb and task condition ($F(2, 54) = 0.6, p = .554$). Post hoc analysis indicated that the RMS error was significantly greater in the affected limb of patients with stroke over the unaffected limb ($p = .037$) and the control subjects ($p < .001$), and was significantly greater in the unaffected limb over the control subjects ($p = .047$). In comparison of task conditions, the RMS error of the 0.5 Hz task was greater than that of the 0.3 Hz task. The RMS error of the unaffected limb for the 0.3 Hz task was similar to that of the control subjects for the 0.5 Hz task. In addition, the RMS error of the affected limb for the 0.3 Hz task was similar to that of the unaffected limb for the 0.5 Hz task.

The results of the timing error during the tracking task are shown in Figure 4. Two-way ANOVA revealed a significant interaction between limb and task condition ($F(2, 54) = 5.5, p = .007$). The one-way ANOVA revealed a significant effect of limb ($F(2, 54) = 3.4, p = .041$) in the 0.5 Hz task on the timing error, but no effect of limb in the 0.3 Hz task ($F(2, 54) = 0.5, p = .589$) on the timing error. Post hoc analysis indicated significant differences between the control subjects and the affected limb ($p = .032$) in the 0.5 Hz task. The difference between the task was significant in the affected limb ($p = .008$), but was not significant in the unaffected limb ($p = .102$) and the control subjects ($p = .468$).

The motor function, as measured by Fugl-Meyer stroke assessment of the LE, was significantly associated with RMS error in the 0.5 Hz task ($r_s = -0.55, p = .021$), but was not significantly associated with RMS error in the 0.3 Hz task ($r_s = -0.47, p = .059$).

The sensory function was not associated significantly with RMS error in the 0.3 Hz task ($r_s = -0.08, p = .755$) or the 0.5 Hz task ($r_s = -0.13, p = .666$).

![Figure 3](image-url) — RMS error of the three limbs (affected, unaffected, and control). The chart shows the mean value with standard deviation. There was a significant difference between all three limbs ($p < .05$), and between the two task conditions ($p = .001$). No significant interaction was observed between limb and task condition ($p = .554$).
Discussion

The aim of this study was to assess the dexterity of the affected and unaffected limbs in patients with stroke by using a force tracking task. The results from comparison between the affected limb and unaffected limb of patients with stroke and the control subjects suggest the presence of deficits of force control ability in the both LE after stroke. Furthermore, force control ability in the 0.5 Hz task was shown to be related to motor function in patients with stroke.

The affected knee extensor MVC force of patients with stroke was reported previously to be approximately 45% of that of the knee extensor MVC force of control subjects (Andrews & Bohannon, 2000; Gerrits et al., 2009), which is lower than the results of the current study where the affected knee extensor MVC force of the patients with stroke was 55.5% of the strength of control subjects. This may be due to the exclusion of the patients with stroke that exhibited less than 80 N of MVC forces of the affected knee extensor. While this loss of muscle strength in the affected LE musculature is a well-described phenomenon in patients with stroke, loss of dexterity has been reported only in the affected UE.

The RMS error was larger in the affected limb than in the unaffected limb, and the RMS error correlated with motor function. These results indicate that force control ability depends on the severity of neuromotor deficits. Motor deficits after stroke are due to damage to the corticospinal system. This damage results in a reduced ability to voluntarily activate spinal motoneurons. Thus, the selective activation of many motoneurons at a variety of frequencies is impaired in the affected limb, and may relate to the loss of force control ability. In the UE, many studies have reported motor unit degeneration. Kallenberg and Hermens (2009) demonstrated a lower mean frequency of the power spectrum of the motor unit action potentials, indicating an increased contribution of low-threshold motor units, related to the degeneration of high-threshold motor units. Hara et al. (2004) reported motor unit loss due to trans-synaptic degeneration that occurs secondarily to an upper motor neuron lesion. Other authors have suggested that the remaining motor units enlarge following stroke due to collateral sprouting and branching of motor neurons (Chang, 1998; Cruz Martínez et al., 1982). Because the neural mechanism is the same, these would be related to loss of force control in the LE, as well.

Other factors related to the reduction of force control ability after stroke include co-contraction and abnormal synergies. Excessive co-contraction has been reported in the knee musculature in patients with stroke during sub-maximal isometric conditions (Neckel et al., 2006) and gait (Higginson et al., 2006). Following stroke, patients lose independent control over select muscle groups, resulting in coupled joint movements that are known as abnormal synergy (Welmer et al., 2006). The excessive co-contraction and abnormal synergies disturb the isolated knee muscle contraction during the force-tracking task.

The RMS error was larger in the 0.5 Hz task than in the 0.3 Hz task in the three limbs. These results indicate that force control ability is influenced by the speed of the task, consistent with previous results suggesting that, in elderly people, movement control is reduced with an increase in speed and complexity of movement (Christou & Carlton, 2001; Yan, 2000). In addition, the timing error was affected by limb during the 0.5 Hz task, and an increase in the timing error was observed in the affected limb during the 0.5 Hz task. The patients with stroke were not able to selectively activate and deactivate muscles with appropriate timing and force scaling in the affected limb in the faster task condition. A study using EMG during cycling of lower limb movements reported timing abnormalities in patients with stroke, and that the
Timing change related to a consequence of a central lesion and secondary changes such as those in muscle fiber and connective tissue properties following stroke (Kautz & Brown, 1998). Other authors reported abnormalities in the initiation and termination of muscle activity (Fellows et al., 1994; Chae et al., 2002; Hammond et al., 1988).

Although damage to proprioceptive afferents may influence force control ability, there was no relationship between the sensory function and the RMS error. One reason for this result was that patients with stroke that participated in this study showed minor sensory function deficits. In addition, visual control of the knee extensor force was allowed during the whole experiment, and reduced proprioception did not negatively affect force control in the present experimental protocol.

The unaffected LE of the patients with stroke showed a significantly larger RMS error than the control subjects during the force-tracking task, indicating a deficit in dexterity in the unaffected limb. These results support those of a previous study using trajectory tracking (Kawahira et al., 2005) and target movement tasks (Kim et al., 2003). A significant difference was not observed between the unaffected limb and the control subjects in the MVC force, but the unaffected limb showed 16% lower MVC force than the control subjects. These deficits may reflect the functional decline of the unaffected limb by disuse. Disuse is a major mechanism responsible for muscle atrophy in stroke patients, because muscle atrophy correlates with daily physical activity (Hachisuka et al., 1997). Few studies have examined the adaptations in force control ability following disuse, but human motor control seems to be negatively affected by the chronic removal of gravitational forces. Muscle atrophy and a reduction in the number of motor units related to age correlates with the decline of force control (Christou & Carlton, 2001). Similarly, these changes were induced by disuse, and related to this finding (Fuglsang-Frederiksen & Scheel, 1978). Previous reports on changes in neuro-motor function with disuse showed an increased variability in submaximal force control in LE musculature, suggesting that alterations in muscle activation strategies may lead to increased force fluctuations. Shinohara et al. (2003) reported that the strength decrease for the knee extensor was 16% of the MVC force, while the variability increased 22% in steady submaximal isometric contraction after 20 days of bed rest. Similarly, Clark et al. (2007) reported increased fluctuation in knee extensor and plantar flexors, and change in spinal excitability after 4 weeks of an unweighted condition. Thus, in addition to muscle atrophy, it is likely that some neural adaptations are induced by disuse. These neural adaptations may explain the declines of force control ability.

Damage to the corticospinal axons also may explain the findings observed in the current study. The corticospinal axons that remain ipsilateral to the stroke lesion causes muscle atrophy (Andrews & Bohannon, 2003). In addition, functional imaging studies showed that complex motor tasks requiring motor planning, an interaction of sensorimotor information and attention to sequencing are associated with bihemispheric activity (Kuhtz-Buschbeck et al., 2008; Ward & Frackowiak, 2003). Damage to corticofugal pathways and bihemispheric activity may interfere with the normal control of the limb ipsilateral to the brain damage (Kim et al., 2003). However, the subjects were, on average, 2 year post stroke patients in this study. Therefore, both the decreased MVC force and loss of dexterity may be more related to disuse.

We acknowledge that there are limitations to our study. Since force control abilities may be affected by joint motion and muscle contraction types, the relation between force control ability during isometric contraction and functional ability is unclear. Further study is needed to investigate how the loss of force control abilities in the LE affects the activities of daily living in patients with stroke.

In conclusion, we investigated the knee extension force control abilities in both affected and unaffected limbs of patients with stroke and in the control subjects. Our results indicate a loss of knee extension force control abilities not only in the affected limb, but also in the unaffected limb of patients with stroke. Furthermore, force control ability during the 0.5 Hz task was related to the motor function of the affected limb in the patients with stroke.

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