Characteristics of the Fastest Isometric Knee Extension in Patients with Spinocerebellar Degenerations

MASAAKI FUJITA and RYUICHI NAKAMURA

Institute of Rehabilitation Medicine, Tohoku University
School of Medicine, Narugo, Miyagi 989-68

Fujita, M. and Nakamura, R. Characteristics of the Fastest Isometric Knee Extension in Patients with Spinocerebellar Degenerations. Tohoku J. Exp. Med., 1989, 157 (1), 13-17 —— The force output of the knee extensor muscles and its time course were measured in nine patients with spinocerebellar degenerations and age-matched 10 normal subjects during the fastest and strongest isometric contraction. The time from the rise of tension to its maximum, $FT_{\text{max}}$, was definitely long in the SCD compared to the normal group, whereas the peak tension was not different between the two groups. $FT_{\text{max}}$ of the SCD group was not related to the rate of tension development and the maximum tension nor to the degree of ataxia, suggesting that the prolongation of $FT_{\text{max}}$ was a characteristic feature of cerebellar lesions. ——— spinocerebellar degenerations; fast isometric contraction; knee extension

The slowness of the affected limb in starting the action and in developing maximum force has been clinically well known in patients with cerebellar lesions since Holmes’ classic studies (Holmes 1917, 1939). Some of the kinesiological and physiological studies of motor functions in patients with spinocerebellar degenerations (SCD) have quantitatively analyzed this phenomenon; for instance, prolonged electromyographic reaction time (EMG-RT) of the biceps brachii muscle for elbow flexion and forearm supination (Nakamura and Taniguchi 1980), and prolongation of the initial biceps EMG bursts in ballistic elbow flexion task (Hallett et al. 1975). Reduced velocity or decreased rate of tension development in fast muscular contraction has commonly found in other neurological disorders such as spastic hemiparesis (Angel 1975; Tsuji and Nakamura 1987), and amyotrophic lateral sclerosis (Hallett 1979). Reduced force output for the fast contraction is coupled not only with the prolongation of initial agonist burst (Angel 1975) but also with the prolonged time from the rise of tension to its maximum (Tsuji and Nakamura 1987) in spastic muscles, which would be a part of compensatory mechanism for muscle weakness to generate sufficient forces to

Received October 14, 1988; revision accepted for publication November 22, 1988.
Correspondence to: Masaaki Fujita, M.D., Institute of Rehabilitation Medicine, Tohoku University School of Medicine, Narugo, Miyagi 989-68, Japan.
accomplish motor tasks (Hallett 1979). There remains a question how the slowness in tension development for fast contraction in patients with SCD is related to prolonged rise time of muscular tension. In the present study, we analyzed the peak tension and its time course during the fastest isometric knee extension in patients with SCD and age matched normal subjects.

METHOD

Nine patients with SCD and age-matched 10 normal subjects as control group participated in the experiment (Table 1). Strength of the knee extensor muscles examined by manual muscle testing (Daniels and Worthingham 1980) was normal in all patients, and spasticity of the lower extremities, i.e., exaggerated velocity dependent stretch reflex, was observed in two patients. Unsteadiness of station referred to as dysequilibrium clinically rated on a four point scale from 0 (none) to 3 (severe) by observing the patients' standing with the feet together and the head upright. One patient could not stand and walk alone due to axial dysequilibrium. Also the grade of incoordination such as dysmetria, decomposition of movement and kinetic tremor of the lower extremity during heel to knee test was classified into a four point scale.

Force output and its time course during the fastest and strongest isometric contraction of the left knee extensors, and EMG activities of the left rectus femoris muscle were examined, using the previously reported apparatus and method of measurement (Nagasaki and Nakamura 1985). The subject sat on a specially designed chair with the trunk upright, the hip and the knee at 90 degrees flexion. At the distal end of the left leg, about 35 cm apart from the medial joint space of the knee, a strap was fixed with two ropes which were connected posteriorly to a tension meter (U3B1, NMB Inc., Tokyo) and anteriorly to 2 kg weight through a pulley. The subject was asked to extend the left knee as rapidly and forcefully as possible responding to a sound stimulus (1 kH, 50 msec duration) presented 2 sec after a warning signal. Each subject was given to a preliminary training session before the experimental run, and then 10 trials were performed with trial-intervals of more than 30 sec. Output of the tension meter and surface EMG of the rectus femoris were fed to a computer (PC-9801F, NEC, Tokyo) via A/D converter. The force output and its time course, and the onset of EMG activities were measured in the computer with a msec scale.

Table 1. Summary of subject data of TLT, FT_{max}, F_{max} and RTD

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>SCD without spasticity</th>
<th>SCD with spasticity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>10</td>
<td>7</td>
<td>2</td>
</tr>
<tr>
<td>Age (year)</td>
<td>52.2 (7.4)</td>
<td>58.6 (5.3)</td>
<td>49.5 (0.5)</td>
</tr>
<tr>
<td>Sex (M/F)</td>
<td>6/4</td>
<td>3/4</td>
<td>0/2</td>
</tr>
<tr>
<td>Duration (yr) of illness</td>
<td>—</td>
<td>4.7 (3.2)</td>
<td>8.5 (3.5)</td>
</tr>
<tr>
<td>TLT (msec)</td>
<td>30.3 (4.3)</td>
<td>31.3 (2.5)</td>
<td>57.0 (1.2)</td>
</tr>
<tr>
<td>FT_{max} (msec)</td>
<td>132.0 (25.2)</td>
<td>256.0 (49.6)</td>
<td>309.3 (29.7)</td>
</tr>
<tr>
<td>F_{max} (kg)</td>
<td>18.1 (6.2)</td>
<td>20.1 (8.7)</td>
<td>9.7 (5.3)</td>
</tr>
<tr>
<td>RTD (kg/msec)</td>
<td>0.14 (0.06)</td>
<td>0.08 (0.03)</td>
<td>0.03 (0.02)</td>
</tr>
</tbody>
</table>

Values of TLT, FT_{max} and, F_{max} and RTD are presented by mean and s.d. in parentheses
Fast Isometric Contraction in SCD

RESULTS

Table 1 presents the means and SDs of TLT, FT\text{max} and F\text{max} of the control and the SCD groups. Since TLT and FT\text{max} prolonged and F\text{max} decreased in spastic muscles (Tsuji and Nakamura 1987), the patients were divided into two groups according to the presence or absence of spasticity. There was no significant difference in the degree of ataxia, dysequilibrium and incoordination, between the two groups.

One way analysis of variance of TLT indicated a significant main effect of group ($F(2, 16) = 43.63, \ p < 0.01$). Post hoc analysis using Tukey’s method showed that TLT of the SCD group with spasticity was significantly longer than that of the control and the SCD group without spasticity ($p < 0.01$, respectively), and TLT of the SCD without spasticity was not significantly different from that of the control. These results indicated that the prolonged TLT was related to spasticity but not to ataxia.

There was a significant difference of FT\text{max} among the three groups ($F(2, 16) = 28.99, \ p < 0.01$). Tukey’s post hoc analysis yielded a significant difference of FT\text{max} between the control and the two SCD groups ($p < 0.01$, respectively), but no
significant difference of $FT_{\text{max}}$ between the two SCD groups. Thus, we combined $FT_{\text{max}}$ of the SCD groups into one. $FT_{\text{max}}$ of the combined SCD group, 268.9 msec, was about twice as long as that of the control, 132.0 msec.

$F_{\text{max}}$s were not significantly different among the three groups ($F (2,16) = 1.40$). $F_{\text{max}}$ of the combined SCD group, 18.2 kg, was not different from that of the control, 18.1 kg.

RTDs of the three groups were significantly different ($F (2,16) = 4.48, p < 0.05$). Tukey’s analysis indicated a significant difference of RTD between the control and the two SCD groups ($p < 0.01$, respectively), but no significant difference between the two SCD groups.

Table 2 presents the correlation coefficients between age, duration of illness, incoordination, dysequilibrium, TLT, $FT_{\text{max}}$, $F_{\text{max}}$ and RTD of the patients. $FT_{\text{max}}$ was not related significantly to any variables examined. There was significant correlation between $F_{\text{max}}$ and RTD ($p < 0.01$), i.e., the higher the RTD, the larger the $F_{\text{max}}$.

**DISCUSSION**

Although there was no significant difference of the maximum tension, $F_{\text{max}}$, during the fastest and strongest isometric knee extension between the control and the SCD groups, the time to attain peak tension, $FT_{\text{max}}$, of the SCD was definitely long as compared with that of the control.

Prolonged $FT_{\text{max}}$ during the fastest isometric contraction of the knee extensor muscles was observed also in patients with other neurological disorders such as spinal cord injuries (Sajiki and Nakamura 1987) and hemiparesis due to stroke (Nakamura and Tsuji 1986). According to Tsuji and Nakamura (1987), $FT_{\text{max}}$s of the affected side of hemiparetic patients were widely distributed and the prolongation of $FT_{\text{max}}$ was coupled with the reduction of RTD. Hallett (1979), examining EMG activities of the biceps and the triceps brachii during fast elbow flexion in
patients with amyotrophic lateral sclerosis, reported that prolonged EMG activities of the agonist muscle was a compensation for reduced force output.

FT\textsubscript{max} of the patients in the present study remained within a narrow range and were not related to RTD or F\textsubscript{max}, while their neurological signs such as dysequilibrium and incoordination were so variable. These results indicate that the prolongation of time to attain peak tension is a specific feature of cerebellar lesions during the fastest and strongest isometric muscular contraction.

References