Deficits in task-specific modulation of anticipatory postural adjustments in individuals with spastic diplegic cerebral palsy

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Tomita H, Fukaya Y, Ueda T, Honma S, Yamashita E, Yamamoto Y, Mori E, Shionoya K. Deficits in task-specific modulation of anticipatory postural adjustments in individuals with spastic diplegic cerebral palsy. J Neurophysiol 105: 2157–2168, 2011. First published February 23, 2011; doi:10.1152/jn.00569.2010.—We examined whether individuals with spastic diplegic cerebral palsy (SDCP) have the ability to utilize lower leg muscles in anticipatory postural adjustments (APAs) associated with voluntary arm movement while standing, as well as the ability to modulate APAs with changes in the degree of postural perturbation caused by arm movement. Seven individuals with spastic diplegia (SDCP group, 12–22 yr of age) and seven age- and sex-matched individuals without disability (control group) participated in this study. Participants flexed both shoulders and lifted a load under two different load conditions, during which electromyographic activities of focal and postural muscles were recorded. Although the timing of anticipatory activation of the erector spinae and medial hamstring (MH) muscles was similar in the two participant groups, that of the gastrocnemius (GcM) muscle was significantly later in the SDCP group than in the control group. An increase in anticipatory postural muscle activity with an increase in load was observed in MH and GcM in the control group but not in GcM in the SDCP group. The degree of modulation in MH was significantly smaller in the SDCP group than in the control group. An additional experiment confirmed that these differences in APAs between the two participant groups were unlikely to be attributable to their differences in initial standing posture before load lift. The present findings suggest that lower leg muscles play a minor role in APAs in individuals with spastic diplegia. In addition, it is likely that these individuals have difficulty modulating anticipatory postural muscle activity with changes in the degree of postural perturbation.

load-lifting task; electromyogram; center of pressure

SPASTIC DIPLEGIA IS A FORM OF CEREBRAL PALSY (CP), which affects the lower limbs more than the upper limbs (Du Plessis 2004). Although many individuals with spastic diplegia have the ability to stand and walk independently with or without the help of assistive devices (Badell-Ribera 1985), these individuals have problems with postural control while standing (de Graaf-Peters et al. 2007; Woollacott and Shumway-Cook 2005). Because postural control is a prerequisite for daily activities, it is important to examine stance postural control in individuals with spastic diplegia. This will enhance understanding of their postural deficits and thereby facilitate therapeutic development.

The central nervous system (CNS) utilizes two major types of postural control to compensate for postural disturbances.
more affected than thigh and trunk muscles in individuals with spastic diplegia (Du Plessis 2004). In addition, previous studies on CPAs have reported that the onset latencies of lower leg muscles in response to external perturbations are delayed in individuals with spastic diplegia (Burtner et al. 1998; Nasher et al. 1983; Woollacott et al. 2005). Therefore, individuals with spastic diplegia may also exhibit a delayed onset of activity of lower leg muscles in APAs. In the above-mentioned study, Tomita et al. (2010b) reported that individuals with spastic diplegia hardly exhibited anticipatory activation of lower leg muscles before bilateral arm flexion. However, anticipatory activation of lower leg muscles was also scarcely observed in individuals without disability in that study (Tomita et al. 2010b). This is probably because a bilateral arm-flexion task in the range from 0° to 90° requires a lesser degree of activity of the lower leg muscles than those of the thigh and trunk muscles (Fujiwara et al. 2003; Mochizuki et al. 2004). It is thus unclear whether individuals with spastic diplegia have the ability to use the lower leg muscles in APAs. Second, Roncesvalles et al. (2002) reported that the primary impairment of CPAs in individuals with spastic diplegia is an inability to modulate postural muscle activity with changes in the degree of external postural perturbation. However, it remains to be determined whether this impairment is also observed in APAs in individuals with spastic diplegia.

Previous studies using a load-lifting task while standing have revealed that, in addition to thigh and trunk muscles on the dorsal side of the body, anticipatory activation of the gastrocnemius (GcM) muscle is observed before load lift in individuals without disability (Fujiwara et al. 2009; Maeda and Fujiwara 2007; Shen et al. 2009). In addition, APAs associated with arm movement in individuals without disability are reportedly modulated by changes in the degree of postural perturbation caused by arm movement (Kasai and Taga 1992; Mochizuki et al. 2004). In the load-lifting task, the degree of postural perturbation can be easily manipulated by changing the load. Therefore, the two questions listed above regarding APAs in individuals with spastic diplegia could be examined with the use of the load-lifting task while standing.

The present study aimed to determine whether individuals with spastic diplegia have the ability to utilize the lower leg muscles in APAs associated with voluntary arm movement, as well as the ability to modulate anticipatory postural muscle activities with changes in the degree of postural perturbation caused by the arm movement. We assumed that, in regard to APAs, both of these abilities are impaired in individuals with spastic diplegia.

METHODS

Participants. Seven individuals with spastic diplegic CP (SDCP group, 3 females and 4 males, 12–22 yr of age) and seven age- and sex-matched individuals without disability (control group, 12–22 yr) participated in this study. A previous study on the development of APAs associated with voluntary arm movement reported that typically developing children younger than 11 yr of age exhibit large background body sway and inconsistent timing of anticipatory shift of center of pressure (COP) (Riach and Hayes 1990). To minimize the effects of developmental changes in APAs, only participants who were 12 yr of age and older were included in this study. All participants in the SDCP group underwent multiple simultaneous surgeries including lengthening of lower limb muscles and osteotomies of the femurs and tibias.

The inclusion criteria for the SDCP group were as follows: level II or III on the Gross Motor Function Classification System (GMFCS; Palisano et al. 1997), no surgical procedures within 2 yr before participation, no history of any genetic or neurological disorder other than spastic diplegic CP, and no flexion contracture of the hip or knee joint or plantar flexion contracture of the ankle joint. All participants with spastic diplegia could stand with their entire soles in contact with the floor without support for 3 min or more. They could also walk indoors independently without any assistive device. No participants in the control group had any history of neurological or orthopedic impairment.

In the SDCP group, mean age, height, weight, and foot length (FL) were 17.1 yr [standard deviation (SD) = 3.5], 151.9 cm (SD = 7.0), 42.9 kg (SD = 9.6), and 22.2 cm (SD = 1.7), respectively. In the control group, these measurements were 17.6 yr (SD = 4.0), 151.8 cm (SD = 5.8), 43.2 kg (SD = 5.6), and 22.2 cm (SD = 2.0), respectively. No significant difference between the two groups was found for any parameter.

Following an explanation of the experimental protocols, all participants and their parents (in the case of participants aged 20 yr or younger) provided written informed consent in accordance with the Declaration of Helsinki. This study was approved by the Ethics Committee at Toyohashi SOZO University.

Apparatus and data recording. Basically, we used the same method as that used in previous studies that examined APAs in the load-lifting task for individuals without disability (Fujiwara et al. 2009; Maeda and Fujiwara 2007; Shen et al. 2009). The experimental setup in the present study is illustrated in Fig. 1. All measurements were performed with participants standing barefoot on a force platform (G-6100; Anima, Tokyo, Japan). The force platform was used to measure the COP position in the mediolateral and anteroposterior directions (CoPx and CoPy, respectively). Arm acceleration was recorded using a miniature unidirectional accelerometer (AS-10GB; Kyowa, Tokyo, Japan) taped to the dorsal surface of the dominant wrist joint so that the axis of sensitivity was along the sagittal plane.

In the load-lifting task, participants held wooden grips attached beneath a wooden board (17 cm × 35 cm × 1.5 cm, weight: 0.6 kg), and had their hands fixed to the wooden board by acrylic belts, with both elbows flexed to 60°, so that both hands were 10 cm below the shoulder joints. The board was suspended from a metal frame by nonextensible metal wires, and a load was attached to the center of the board. To monitor the force applied to the board by the participant, a load cell (LUB-50KB-P, Kyowa) was attached to the connection between the frame and the wire (Fig. 1). The force was displayed on a computer (AT971W7C2; Epson, Nagano, Japan), and trials in which a clear change in the force applied to the board (beyond ±2.5% of the force during quiet standing posture) was observed before load lift were excluded. The total weight of the board and load was set at 2% of body weight for women and 3% of body weight for men under the light condition. Under the heavy condition, the weight was set at 4% of body weight for women and 6% of body weight for men.

Electromyograms (EMGs) were recorded using bipolar surface electrodes placed over the following muscles on both sides of the body. The anterior deltoid (AD) muscle was recorded as the focal muscle of the load lift. The rectus abdominis (RA), ES, rectus femoris (RF), MH, tibialis anterior (TA), medial head of GcM, and soleus (Sol) muscles were recorded as postural muscles. Electrodes were placed on the midportion of the muscle belly. The electrodes were aligned along the long axis of the muscle with an interelectrode distance of about 2 cm. Electrode input impedance was below 5 kΩ. EMG signals from the electrodes were amplified (×2,000) and bandpass filtered (10–1,000 Hz) using an EMG amplifier (MEG-6116; Nihon Kohden, Tokyo, Japan). Electrical signals of CoP, arm acceleration, load cell, and EMGs were recorded using a computer (FMVC310; Fujitsu, Kanagawa, Japan) via an A/D converter (ADA16–32/2(CB)F; Contec, Osaka, Japan) with a sampling frequency of 2,000
Hz and 16-bit resolution using BIMUTAS II-R software (Kissei Comtec, Nagano, Japan).

Motion of the upper and lower limbs and the trunk during load lift was recorded using an eight-camera motion analysis system (VICON Motion System, Oxford, UK) with a sampling frequency of 120 Hz. The standard Plug-in-Gait marker protocol (with 35 reflective markers) was used. Plug-in-Gait model processing was applied to reprocess all kinematic data using VICON Nexus 1.3 software (VICON Motion System). A trigger signal was also recorded to synchronize motion data with electrical signals of CoP, arm acceleration, load cell, and EMGs.

Procedure. Participants stood on the force platform at a stance width of 5–10 cm between the heels. Initially, both hands were strapped to the wooden board, and participants were instructed to keep their shoulder muscles as relaxed as possible without leaning toward the board. CoPx and CoPy positions were then measured for 10 s while participants maintained a quiet standing posture. Five measurements were taken with intermittent 30-s periods of seated rest. The mean of the five measurements was used as the participant’s representative CoPx and CoPy positions during quiet standing.

The load-lifting task then commenced. Under each condition (light or heavy), the load lift was repeated 15 times after 10 practice trials. It has been reported that the timing and amplitude of anticipatory postural muscle activity are influenced by CoP position just before lifting (Benvenuti et al. 1997; Fujisawa et al. 2003). To minimize intertrial differences in APAs attributable to differences in CoP position before the load lift, a buzzing sound, which was generated by a computer (PP21L; Dell, Round Rock, TX), was connected to the force platform and was used to inform participants whether they were maintaining CoPx and CoPy positions within a range of ±1.5 cm of the quiet standing position. In each trial, participants maintained the CoPx and CoPy positions within the range for at least 3 s while hearing the buzzing sound. Within 3 s after cessation of the buzzing sound, participants started to flex both shoulders and lifted the load by about 10 cm at their own timing. Participants were told to lift the load at their maximum speed and to maintain the 10-cm lifted position for about 3 s before returning to the starting position. Participants were given seated rest periods of 5 min after every five trials. The order of conditions was randomized for each participant.

Data analysis. All data analyses were performed offline using Matlab software version R2009b (MathWorks, Natick, MA). Mean CoPy position was calculated for the period from −300 ms to −150 ms with respect to burst onset of AP and was defined as the initial CoPy position. Initial CoPy position was represented by a percentage of the distance from the heels in relation to the participant’s FL (%FL). The difference between the initial CoPy position and the most anterior CoPy position during the load lift was then calculated and defined as the peak CoPy displacement.

To exclude electrocardiogram and movement artifacts, EMGs were high-pass filtered (20 Hz) using the third-order zero-phase Butterworth method and then full-wave rectified. The time course of the EMG burst of focal and postural muscles in each trial was then analyzed. Because the background activity of AP before burst onset was extremely small, burst onset of AP on the dominant side was identified by visual inspection. For postural muscles, the mean EMG amplitude over the period from −300 ms to −150 ms with respect to burst onset of AP was defined as background activity. EMG bursts of postural muscles that continued for at least 50 ms were determined within the period from −150 ms to +50 ms with respect to burst onset of AP was defined as background activity. EMG bursts of postural muscles that continued for at least 50 ms were determined within the period from −150 ms to +50 ms with respect to burst onset of AP. This period was selected on the basis of previous findings that suggested that the quickest monosynaptic reflexes could be observed after a perturbation in the time interval that is usually longer than +50 ms (Friedli et al. 1984; Latash et al. 1995). The burst onset of postural muscles was defined as the time at which the rectified EMG deviation was extremly small, burst onset of AP on the dominant side was identified by visual inspection. For postural muscles, the mean EMG amplitude over the period from −300 ms to −150 ms with respect to burst onset of AP defined as the start time of postural muscles and presented as a negative value when the burst onset was earlier in postural muscles than in AP.

Further analysis of EMGs was performed to examine the amplitude of anticipatory postural muscle activity with respect to the background activity. First, for each postural muscle, EMGs from −300 ms to +50 ms with respect to AD onset were averaged. This averaging was performed separately for each participant and for each load condition. Next, the mean amplitude of a given postural muscle in the back-
ground range was calculated for the period from $-300$ ms to $-150$ ms. The mean amplitude of the postural muscle in the anticipatory range was then calculated by subtracting the mean amplitude of the postural muscle in the background range from the mean amplitude in the period from $-150$ ms to $+50$ ms. Finally, to allow interparticipant comparison, the ratio of mean EMG amplitude in the anticipatory range to that in the background range was calculated and defined as the normalized anticipatory EMG amplitude of the postural muscle.

To examine differences in initial postural alignment before load lift between the SDCP group and control group, angles of the left and right hip, knee, and ankle joints were analyzed. Mean joint angle was calculated over the period from $-300$ ms to $-150$ ms with respect to burst onset of AD separately for each joint listed above and was defined as the initial joint angle. Positive value meant flexion at the hip and knee joints and dorsiflexion at the ankle joint.

No obvious laterality was observed in the start time or normalized anticipatory EMG amplitude of postural muscles or the initial joint angles in either participant group. Therefore, the EMG measurements and initial joint angles recorded from the left and right side of the body were pooled.

**Statistical analysis.** Mean values of the peak value and time of arm acceleration, initial CoPy position, peak CoPy displacement, initial joint angles, and start times of postural muscles for 15 trials were calculated separately for each load condition (light or heavy) and used as representative values for each participant.

Two-way mixed-design ANOVA was used to assess the effects of participant group (SDCP or control) and load condition (light or heavy) on each parameter. When significant main effects of participant group and load condition were found with a significant interaction between the two factors, post hoc analyses were performed to examine differences suggested by ANOVA. Differences between the two participant groups were assessed by post hoc t-tests or Welch's test with Bonferroni correction depending on whether a significant difference in variance was observed or not, respectively. Differences between the two load conditions were assessed by post hoc paired t-test with Bonferroni correction.

Welch’s test was used to compare the degree of increase in the peak CoPy displacement and the normalized anticipatory EMG amplitude of MH under the heavy condition with respect to that under the light condition between the SDCP group and control group.

α-Level was set at $P < 0.05$. All statistical analyses were performed using IBM SPSS Statistics 18 software (SPSS, Chicago, IL).

**RESULTS**

**Kinematic and kinetic measurements.** Table 1 shows the kinematic and kinetic measurements under each load condition for each participant group. The percentage of trials in which the EMG burst was observed in the anticipatory range is also shown in Table 1. No significant main effect of participant group or load condition was found for the peak value or time of arm acceleration, and there was no significant interaction between the two factors (Table 1).

A significant main effect of participant group was found for the initial angles of the knee and ankle joints (knee: $F_{1,12} = 8.8, P < 0.05$; ankle: $F_{1,12} = 7.6, P < 0.05$); the knee joint was significantly more flexed and the ankle joint was more dorsiflexed in the SDCP group than in the control group under both load conditions (Table 1). No significant main effect of load condition or significant interaction between the two factors was found for the initial angles of the knee or ankle joint. No significant main effect of participant group or load condition was found for the initial hip joint angle, and there was no significant interaction between the two factors (Table 1).

A significant main effect of participant group was found for the initial CoPy position ($F_{1,12} = 18.0, P < 0.001$); the initial CoPy position was significantly more anterior in the SDCP group than in the control group under both load conditions (Table 1). No significant main effect of load condition or significant interaction between the two factors was found for the initial CoPy position.

Significant main effects of participant group ($F_{1,12} = 14.7, P < 0.01$) and load condition ($F_{1,12} = 82.4, P < 0.001$) were found for the peak CoPy displacement with a significant interaction between the two factors ($F_{1,12} = 12.0, P < 0.01$). Post hoc analyses revealed that the peak CoPy displacement was significantly larger in the SDCP group than in the control group under both load conditions (light: $t_{12} = 2.5, P < 0.05$; heavy: $t_{12} = 4.2, P < 0.01$, with t-test with Bonferroni correction) and was significantly larger under the heavy condition than under the light condition in both participant groups.

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**Table 1. Kinematic and kinetic measurements and percentage of trials with anticipatory EMG activity under each load condition for each participant group**

<table>
<thead>
<tr>
<th></th>
<th>Light Condition</th>
<th>Heavy Condition</th>
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<tr>
<td></td>
<td>SDCP Group</td>
<td>Control Group</td>
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<tr>
<td>Peak arm acceleration, m/s²</td>
<td>16.5 (10.9)</td>
<td>18.4 (9.5)</td>
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<tr>
<td>Time of peak arm acceleration, ms</td>
<td>215.5 (88.2)</td>
<td>154.7 (42.4)</td>
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<tr>
<td>Initial CoPy position, %FL</td>
<td>54.3 (5.1)</td>
<td>42.8 (4.6)</td>
</tr>
<tr>
<td>Peak CoPy displacement, %FL</td>
<td>12.2 (2.3)</td>
<td>9.2 (2.2)</td>
</tr>
<tr>
<td>Initial hip joint angle, degrees</td>
<td>17.4 (11.1)</td>
<td>10.7 (2.1)</td>
</tr>
<tr>
<td>Initial knee joint angle, degrees</td>
<td>14.5 (10.6)</td>
<td>1.9 (4.0)</td>
</tr>
<tr>
<td>Initial ankle joint angle, degrees</td>
<td>20.3 (11.5)</td>
<td>6.3 (5.7)</td>
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<tr>
<td>Burst percentage of postural muscles, %</td>
<td>11.1 (8.8)</td>
<td>14.8 (9.5)</td>
</tr>
<tr>
<td>Erector spinae</td>
<td>76.7 (17.8)</td>
<td>94.0 (8.7)</td>
</tr>
<tr>
<td>Rectus femoris</td>
<td>9.5 (9.1)</td>
<td>8.8 (6.7)</td>
</tr>
<tr>
<td>Medial hamstrings</td>
<td>74.4 (28.7)</td>
<td>95.4 (8.6)</td>
</tr>
<tr>
<td>Tibialis anterior</td>
<td>16.4 (6.4)</td>
<td>11.5 (5.0)</td>
</tr>
<tr>
<td>Gastrocnemius</td>
<td>55.0 (29.9)</td>
<td>84.1 (15.2)</td>
</tr>
<tr>
<td>Soleus</td>
<td>39.3 (37.9)</td>
<td>47.1 (34.5)</td>
</tr>
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</table>

Values are means (SD). Measurements with significant main effect of participant group, that of load condition, and significant interaction between the two factors are indicated. EMG, electromyogram; SDCP, spastic diplegic cerebral palsy; CoPy, center of pressure in the anteroposterior direction; FL, foot length.
(SDCP group: $t_6 = 6.6, P < 0.01$; control group: $t_6 = 8.3, P < 0.001$, with paired t-test with Bonferroni correction) (Table 1).

Increase in the peak CoPy displacement under the heavy condition vs. that under the light condition was significantly larger in the SDCP group than in the control group [SDCP group: 8.2%FL (SD = 3.3); control group: 3.7%FL (SD = 1.2), $t_{12} = 3.5, P < 0.01$].

**EMG measurements.** Overall, the percentage of trials with postural muscle activity in the anticipatory range was much higher in the dorsal muscles (ES, MH, and GcM), with the exception of Sol, than in the ventral muscles (RA, RF, and TA) (Table 1). We therefore focused our analysis of EMG measurements on ES, MH, and GcM. Figure 2 shows the percentages of trials with anticipatory activity in these dorsal postural muscles for each participant under each load condition. Although the percentages for these muscles tended to be lower in the SDCP group than in the control group, individual differences were fairly large in the SDCP group. Statistical analyses revealed that, in GcM alone, a significant main effect of participant group was found for the percentage ($F_{1,12} = 5.7, P < 0.05$); the percentage in GcM was significantly lower in the SDCP group than in the control group under both load conditions (Table 1). No significant main effect of load condition or significant interaction between the two factors was found for the percentage in GcM.

Figure 3 shows representative EMG data for ES, MH, and GcM during a trial under each load condition in a participant with spastic diplegia and a participant without disability. In the participant without disability, these three postural muscles were activated in advance of AD, whereas anticipatory activation with respect to AD was not observed in GcM in the participant with spastic diplegia. In addition, an increase in
EMG amplitudes of these postural muscles under the heavy condition compared with those under the light condition was apparent in the participant without disability but not in the participant with spastic diplegia.

Figure 4 shows the means and SDs of start times of ES, MH, and GeM with respect to AD in each participant group (SDCP group or control group) under each load condition (light or heavy). The start times in the SDCP group are shown with filled symbols, whereas those in the control group are shown with open symbols. **P < 0.01.

EMG amplitudes of these postural muscles under the heavy condition compared with those under the light condition was apparent in the participant without disability but not in the participant with spastic diplegia.

Figure 4 shows the means and SDs of start times of ES, MH, and GeM with respect to AD, under each load condition for each participant group. No significant main effect of participant group or load condition was found for the start time of ES or MH, and there was no significant interaction between the two factors. A significant main effect of participant group was found for the start time of GeM ($F_{1,12} = 14.9, P < 0.01$); the start time of GeM was significantly later in the SDCP group than in the control group under both load conditions. No significant main effect of load condition or significant interaction between the two factors was found for the start time of GeM.

Figure 5 shows the means and SDs of normalized anticipatory EMG amplitudes of ES, MH, and GeM, with respect to background activities, under each load condition for each participant group. A significant main effect of participant group was found for the normalized anticipatory EMG amplitude of ES ($F_{1,12} = 10.1, P < 0.01$); the normalized anticipatory EMG amplitude in ES was significantly smaller in the SDCP group than in the control group under both load conditions. No significant main effect of load condition or significant interaction between the two factors was found for the normalized anticipatory EMG amplitude of GeM.

Figure 5 shows the means and SDs of normalized anticipatory EMG amplitudes of ES, MH, and GeM, with respect to background activities, under each load condition for each participant group. A significant main effect of participant group was found for the normalized anticipatory EMG amplitude of ES ($F_{1,12} = 10.1, P < 0.01$); the normalized anticipatory EMG amplitude in ES was significantly smaller in the SDCP group than in the control group under both load conditions. No significant main effect of load condition or significant interaction between the two factors was found for the normalized anticipatory EMG amplitude of GeM.

Significant main effects of participant group (MH: $F_{1,12} = 9.3, P < 0.01$; GeM: $F_{1,12} = 22.0, P < 0.001$) and load condition (MH: $F_{1,12} = 12.2, P < 0.01$; GeM: $F_{1,12} = 14.7, P < 0.01$) were found for the normalized anticipatory EMG amplitudes of MH and GeM, with significant interactions between the two factors (MH: $F_{1,12} = 10.0, P < 0.01$; GeM: $F_{1,12} = 12.1, P < 0.001$). Post hoc analyses revealed the following findings. The normalized
anticipatory EMG amplitudes in MH and GcM were significantly smaller in the SDCP group than in the control group under both load conditions (light: $t_{12} > 3.0, P < 0.05$; heavy: $t_{12} > 3.1, P < 0.05$, with $t$-test with Bonferroni correction). In the control group, the normalized anticipatory EMG amplitudes in MH and GcM were significantly larger under the heavy condition than under the light condition (MH: $t_6 = 3.3, P < 0.05$; GcM: $t_6 = 3.7, P < 0.05$, with paired $t$-test with Bonferroni correction). In the SDCP group, although a similar result was obtained in MH ($t_6 = 2.5, P < 0.05$, with paired $t$-test with Bonferroni correction), load condition had no significant effect on the normalized anticipatory EMG amplitude of GcM.

Although participants in both the SDCP group and control group exhibited larger normalized anticipatory EMG amplitudes of MH under the heavy condition than the light condition, the degree of increase was significantly smaller in the SDCP group than in the control group ($t_{12} = 7.3, P < 0.01$) (Fig. 6).

Additional experiment. Our results revealed several differences in APAs between the SDCP group and control group. However, participants with spastic diplegia exhibited the crouch posture, a typical standing posture characterized by excessive knee flexion during standing (Table 1). It has been reported that differences in CPAs in response to external postural perturbation between children with spastic diplegia and typically developing children result, at least in part, from differences in their postural alignment (Burtner et al. 1998). In addition, in the present study, initial CoPy position was more anterior in the SDCP group than in the control group. This difference is also a potential reason for the differences in APAs between the two participant groups because anticipatory postural muscle activity appears to be influenced by initial CoPy position before voluntary arm movement (Benvenuti et al. 1997; Fujiwara et al. 2003). To determine the effects of difference in initial standing posture before the load lift between the SDCP group and control group on the differences in APAs between the two participant groups, we conducted an additional experiment in which participants without disability performed the load-lifting task in upright and crouch postures.

The participants were seven university students without disability (3 females and 4 males). Mean age, height, weight and foot length were 21.3 yr (SD = 0.8), 166.8 cm (SD = 6.7), 57.1 kg (SD = 7.2), and 25.0 cm (SD = 1.6) respectively. Written informed consent was obtained from all participants.

Basically, we used the same method as in the initial experiment. Participants performed the load-lifting task in two different initial postures (upright and crouch) under two different load conditions (light and heavy). In the upright condition, participants maintained their normal upright standing posture with their CoPy position within the range of ±1.5 cm of the mean CoPy position in the control group in the initial experiment (42.9%FL), while hearing the buzzing sound. In the crouch condition, they maintained the CoPy position within the range of ±1.5 cm of the mean CoPy position in the SDCP group in the initial experiment (54.1%FL). In this condition, they also flexed their knee joints by about 15°, which was almost the same as the mean initial angle of the knee joints in the SDCP group in the initial experiment (see Table 1). Angles of the left and right knee joints were monitored using an electrogoniometer system (Biometrics, Gwent, UK). Within 3 s after cessation of the buzzing sound, participants lifted load by about 10 cm at their own timing. In each condition, the load lift was repeated 10 times after 10 practice trials. The order of conditions was randomized for each participant.

Two-way repeated-measures ANOVA was used to assess the effects of initial standing posture (upright or crouch) and load condition (light or heavy) on each parameter.

Results of the additional experiment are shown in Table 2, which shows the kinematic and kinetic measurements in each initial standing posture under each load condition. The percentages of trials with anticipatory activity in ES, MH, and GcM are also shown in Table 2. Overall, the initial angles of the hip, knee, and ankle joints and the initial CoPy position in the crouch condition were similar to those in the SDCP group in the initial experiment (see also Table 1). A significant main effect of load condition was found for the peak CoPy displacement ($F_{1,6} = 74.3, P < 0.001$); the peak CoPy displacement was significantly larger in the heavy condition than in the light condition in both initial standing postures (Table 2). However, no significant main effect of initial standing posture or significant interaction between the two factors was found for the peak CoPy displacement.

The percentages of trials with anticipatory activity in the dorsal muscles (ES, MH, and GcM) in the upright condition in the additional experiment (Table 2) were almost the same as those in the control group in the initial experiment (Table 1). Although the percentages in ES and MH were similar between the crouch and upright conditions, the percentage in GcM in the crouch condition was to some extent lower than that in the upright condition. However, the percentage in GcM in the crouch condition was somewhat higher than that in the SDCP group in the initial experiment (see also Table 1).
In the initial experiment, significant main effects of participant group and/or load condition were found for the start time of GcM and the normalized anticipatory EMG amplitudes of MH and GcM (see Figs. 4 and 5). We therefore focused our analysis of EMGs in the additional experiment on these measurements. Figure 7 shows the means and SDs of start time of GcM with respect to AD (Fig. 7A) and those of normalized anticipatory EMG amplitudes of MH and GcM (Fig. 7B) in each initial standing posture (upright or crouch) under each load condition (light or heavy). The start times under the light condition are shown with circles, whereas those under the heavy condition are shown with triangles. The start times in the upright condition are shown with filled symbols, whereas those under the crouch condition are shown with open symbols. The normalized anticipatory EMG amplitudes in the upright condition are shown with filled bars, whereas those in the crouch condition are shown with open bars. *P < 0.05.

### Table 2. Kinematic and kinetic measurements, and percentage of trials with anticipatory EMG activity in each initial standing posture under each load condition

<table>
<thead>
<tr>
<th></th>
<th>Light Condition</th>
<th>Heavy Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Upright</td>
<td>Crouch</td>
</tr>
<tr>
<td>Peak arm acceleration, m/s²</td>
<td>23.5 (9.4)</td>
<td>22.0 (10.2)</td>
</tr>
<tr>
<td>Time of peak arm acceleration, ms</td>
<td>141.6 (33.9)</td>
<td>133.5 (29.2)</td>
</tr>
<tr>
<td>Initial CoPy position, %FLa</td>
<td>42.6 (2.2)</td>
<td>54.8 (2.2)</td>
</tr>
<tr>
<td>Peak CoPy displacement, %FLb</td>
<td>15.5 (7.4)</td>
<td>13.6 (5.6)</td>
</tr>
<tr>
<td>Initial hip joint angle, degreesa</td>
<td>12.2 (10.7)</td>
<td>18.3 (9.8)</td>
</tr>
<tr>
<td>Initial knee joint angle, degreesa</td>
<td>–2.3 (2.1)</td>
<td>15.2 (3.4)</td>
</tr>
<tr>
<td>Initial ankle joint angle, degreesa</td>
<td>4.1 (2.3)</td>
<td>16.3 (1.6)</td>
</tr>
<tr>
<td>Burst percentage of postural muscles, %</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Erector spinae</td>
<td>100.0 (0.0)</td>
<td>97.9 (3.9)</td>
</tr>
<tr>
<td>Medial hamstrings</td>
<td>100.0 (0.0)</td>
<td>96.4 (6.3)</td>
</tr>
<tr>
<td>Gastrocnemius</td>
<td>90.0 (8.7)</td>
<td>71.4 (10.3)</td>
</tr>
</tbody>
</table>

Values are means (SD). Measurements with *significant main effect of initial standing posture and that of #load condition are indicated.
each initial standing posture under each load condition. Although the start time of GcM in the crouch condition was slightly later than that in the upright condition, no significant main effect of initial standing posture was found for the start time of GcM (Fig. 7A). In addition, no significant main effect of load condition was found for the start time, and there was no significant interaction between the two factors.

A significant main effect of load condition was found for the normalized anticipatory EMG amplitudes of MH and GcM (MH: $F_{1,6} = 6.4, P < 0.05$; GcM: $F_{1,6} = 12.4, P < 0.05$); the normalized anticipatory EMG amplitudes in MH and GcM were significantly larger in the heavy condition than in the light condition in both initial standing postures (Fig. 7B). However, no significant main effect of initial standing posture or significant interaction between the two factors was found for the normalized anticipatory EMG amplitude of MH or GcM.

Overall, the results of the additional experiment suggested that the anticipatory postural muscle activity and the disturbance of postural equilibrium during load lift are less influenced by difference in initial standing posture.

**DISCUSSION**

The main differences in APAs, obtained in the initial experiment, between participants in the SDCP group and those in the control group, can be summarized as follows: 1) although participants with spastic diplegia exhibited anticipatory activation of dorsal postural muscles (ES, MH, and GcM), the percentage of trials with anticipatory activity in GcM was lower in participants with spastic diplegia than in those without disability; 2) although the start times of ES and MH with respect to AD were similar in the two participant groups, that of GcM was later in participants with spastic diplegia; 3) the anticipatory activities of the dorsal postural muscles with respect to background activities were smaller in participants with spastic diplegia; 4) the increase in anticipatory activities of dorsal postural muscles (MH and GcM) with increase in load was smaller in participants with spastic diplegia; and 5) the disturbance of postural equilibrium during load lift, which was quantified by peak CoPy displacement, was larger in participants with spastic diplegia.

However, initial postural alignment and CoPy position before the load lift differed between the SDCP group and control group. These differences have been reported as potential causes of differences in stance postural control (Benvenuti et al. 1997; Burtner et al. 1998; Fujiwara et al. 2003). To address this issue, in the additional experiment, participants without disability lifted the load in two different initial standing postures to mimic those in the SDCP group and control group in the initial experiment. As a result, the anticipatory activities of the dorsal postural muscles and the disturbance of postural equilibrium during load-lifting task were less influenced by the difference in initial standing posture, suggesting that the difference in initial standing posture between the SDCP group and control group in the initial experiment was not the primary cause of the differences in APAs between the two participant groups. Therefore, the implications and potential causes, other than initial standing posture, of the findings obtained in the initial experiment listed above are discussed below.

**Anticipatory postural muscle activity in individuals with spastic diplegia.** Anticipatory postural muscle activity associated with voluntary arm movement is reportedly influenced by peak arm acceleration during this movement (Mochizuki et al. 2004). However, in the present study, no significant difference in the peak value or time of arm acceleration during load lift was found between the SDCP group and control group under either load condition. It is thus unlikely that differences in APAs during load lift between the two participant groups were attributable to the differences in kinematics of arm movement.

Participants with spastic diplegia, as well as those without disability, exhibited anticipatory activation of ES and MH in advance of AD under both load conditions. In addition, anticipatory activation of postural muscles on the ventral side of the body (RA, RF, and TA) was scarcely observed under either load condition or in either participant group. However, the anticipatory muscle activities of ES, MH, and GcM, with respect to background activities, were significantly smaller in the SDCP group than in the control group. Similar results have been reported for individuals with spastic diplegia in a previous study on APAs associated with bilateral arm flexion in the range from 0° to 90° while standing (Tomita et al. 2010b). These findings suggest that, although individuals with spastic diplegia have the ability to anticipate the effects of perturbation caused by arm movement on posture and equilibrium and retain the ability to generate direction-specific anticipatory postural muscle activity, the anticipatory activity is smaller in individuals with spastic diplegia. In addition, it is likely that antagonistic coactivation, which has been observed in individuals with spastic diplegia in CPAs in response to support surface translation (Burtner et al. 1998; Nasher et al. 1983; Woollacott et al. 2005), is only minimally exhibited in APAs associated with voluntary arm movement in individuals with spastic diplegia. This assumption is also supported by the previous finding that children with spastic diplegia scarcely used the coactivation strategy during voluntary forward reach while sitting (van der Heide et al. 2004).

Anticipatory activation of GcM was observed in the control group, consistent with previous findings of APAs in the load-lifting task in individuals without disability (Fujiwara et al. 2009; Maeda and Fukushima 2007; Shen et al. 2009), suggesting that GcM plays an important role in APAs in this task. However, the percentage of trials with anticipatory activation of GcM was significantly lower in the SDCP group than in the control group. In addition, the start time of GcM in the trials with anticipatory activation was significantly later in the SDCP group than in the control group. These results support our first hypothesis that individuals with spastic diplegia have difficulty utilizing their lower leg muscles in APAs associated with voluntary arm movements while standing.

In the control group, increases in anticipatory postural muscle activity with the increase in load were observed in MH and GcM. In the SDCP group, such modulation of anticipatory postural muscle activity was observed in MH, but not in GcM. This finding also supports our notion that, in individuals with spastic diplegia, lower leg muscles play a minor role in APAs associated with voluntary arm movement while standing. In addition, the degree of increase in anticipatory activation of MH with increase in load, which was observed in both participant groups, was significantly smaller in the SDCP group than in the control group. These results support our second hypothesis that individuals with spastic diplegia have difficulty modulating anticipatory postural muscle activities with changes in the degree of postural perturbation caused by arm movement.
A delayed onset of activity of lower leg muscles in individuals with spastic diplegia has also been reported in previous research on APAs in response to support surface translation (Burtner et al. 1998; Nashner et al. 1983; Woollacott et al. 2005). In addition, deficits in modulation of postural muscle activity to fit task conditions in these individuals are reported in CPAs in response to external postural perturbation (Ronc-esvalles et al. 2002) and voluntary changes in standing posture in the anteroposterior direction (Tomita et al. 2010a). It is thus likely that inabilities to utilize their lower leg muscles and to modulate postural muscle activity are common characteristics of stance postural control, including APAs, in individuals with spastic diplegia.

Postural equilibrium during arm movement in individuals with spastic diplegia. The stability limit in the forward direction is reportedly more posterior in individuals with spastic diplegia than in those without disability (Tomita et al. 2010a). In the present study, the initial CoPy position before load lift in the SDCP group was more anterior than that in the control group. This means that the stability margin in the forward direction is smaller in participants with spastic diplegia than in those without disability and, thus, that the need for compensation for postural disturbance in the forward direction caused by the load lift should be higher in the SDCP group than in the control group.

However, the peak CoPy displacement during the load lift was significantly larger in the SDCP group than in the control group under both load conditions, which is in accordance with a previous finding on APAs in individuals with spastic diplegia (Tomita et al. 2010b). Furthermore, the increase in peak CoPy displacement under the heavy condition, compared with that under the light condition, was significantly larger in the SDCP group than in the control group. These results suggest that postural disturbance caused by arm movement is less compensated in individuals with spastic diplegia than in those without disability. In addition, when postural perturbation caused by arm movement becomes larger, postural equilibrium during the arm movement in individuals with spastic diplegia is likely to be disrupted to a greater extent than that in individuals without disability. It might be suspected that the degree of disturbance of postural equilibrium during load lift is influenced by a difference in postural perturbation attributable to variations in kinematics of the arm movement and/or the participant’s anthropometric characteristics. However, no significant differences between the SDCP group and control group were found in the peak value or time of arm acceleration or any anthropometric measurement. Instead, anticipatory activation of postural muscles reportedly plays an important role in decreasing CoP displacement after postural perturbation (Santos et al. 2010). It is thus likely that the smaller anticipatory activation of postural muscles and the smaller degree of increase in anticipatory postural muscle activities with increase in load in the SDCP group influenced the larger disruption of postural equilibrium during the load lift.

Why do individuals with spastic diplegia have difficulty modulating APAs to fit task conditions? APAs are believed to be preprogrammed in the CNS because postural muscle activity is observed before postural perturbations caused by voluntary movements (Friedli et al. 1984; Horak et al. 1984). Consistent with our previous finding (Tomita et al. 2010b), in the present study, anticipatory muscle activities were observed in several dorsal postural muscles in participants with spastic diplegia, suggesting that their damaged CNS causing spastic diplegic CP can produce APAs to compensate for forthcoming postural disturbances. However, the present findings reveal that participants with spastic diplegia exhibited deficits in adequately modulating their APAs in accordance with the degree of effects of postural perturbation. Although no direct evidence was obtained from this study, previous findings on individuals with spastic diplegia raise several possibilities regarding this deficit.

First, it has been suggested that the ability to generate adequate APAs is not innate, but rather acquired with motor development (van der Heide et al. 2003). Experience and learning are believed to be essential in the development of APAs (Kazennikov et al. 2008; Schmitz and Assaiante 2002). In addition, it is likely that proprioceptive information, which is used to build up internal representations of the effects of voluntary movements on posture and equilibrium (Massion 1998; Schmitz et al. 2002), is essential for the development of APAs (Palluel et al. 2008). However, it has been reported that proprioceptive deficits and/or poor motor experience attributable to motor disabilities.

In this study, anticipatory activities in the dorsal postural muscles with respect to background activities were much smaller in participants with spastic diplegia than in those without disability. This finding raises another possibility that, although individuals with spastic diplegia have the ability to correctly estimate the effects of forthcoming perturbation on posture and equilibrium, they cannot adequately increase anticipatory postural muscle activity because of primary and/or secondary impairments in muscle contraction caused by SDCP. Individuals with spastic CP appear to exhibit impaired control of muscle contraction because of spasticity (Becher et al. 1998) and/or muscle weakness (Wiley and Damiano 1998). In addition, it has been reported that the capacity to rapidly contract lower limb muscles is reduced in individuals with spastic diplegia (Downing et al. 2009). These impairments in individuals with spastic diplegia may limit their ability to increase postural muscle activity in the anticipatory range. Furthermore, because selective voluntary motor control of the lower limbs is reportedly impaired in individuals with spastic diplegia, with a proximal-to-distal gradient (Fowler et al. 2010), they may be unable to simultaneously activate extensor (e.g., GcM) and flexor (e.g., MH) postural muscles during voluntary movements while standing.

However, it is difficult to determine which, if any, of these possibilities is correct on the basis of the present findings alone. Furthermore, the findings for the percentages of trials with anticipatory activity of the dorsal postural muscles suggest that individual differences in APAs were fairly large in participants with spastic diplegia, possibly because severity was not uniform across them (i.e., level II or III on GMFCS). However, the relatively small number of participants in this
study prevented further analyses regarding individual differences in APAs in the SDCP group. Further testing with greater numbers of individuals with spastic diplegia, including those who are 12 yr of age or younger, is needed to determine why individuals with spastic diplegia exhibit deficits in APAs and to incorporate our findings into therapeutic intervention aimed at improving these deficits.

Conclusions. The results of this study support the previous findings that, although individuals with spastic diplegia exhibit anticipatory postural muscle activity during voluntary arm movement, their anticipatory activities in postural muscles are insufficiently small (Tomita et al. 2010b). In addition, the present study suggests the following novel findings providing insight into characteristics of APAs while standing in individuals with spastic diplegia: 1) lower leg muscles play a minor role in APAs in individuals with spastic diplegia; 2) individuals with spastic diplegia have difficulty modulating anticipatory postural muscle activity with changes in the degree of postural disturbance caused by voluntary movement; and 3) these deficits in APAs in individuals with spastic diplegia are likely to be related to the larger disturbance of postural equilibrium during voluntary arm movement.

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DISCLOSURES

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