Deficits in anticipatory inhibition of postural muscle activity associated with load release while standing in individuals with spastic diplegic cerebral palsy

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Running head: Deficits in inhibitory APAs in individuals with SDCP

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ABSTRACT

This study aimed to determine whether individuals with spastic diplegic cerebral palsy (SDCP) have deficits in anticipatory inhibition of postural muscle activity. Nine individuals with SDCP (SDCP group, 3 females and 6 males, 13–24 years of age) and 9 age- and gender-matched individuals without disability (control group) participated in this study. Participants stood on a force platform, which was used to measure the position of the center of pressure (CoP), while holding a light or heavy load in front of their bodies. They then released the load by abducting both shoulders. Surface electromyograms were recorded from the rectus abdominis, erector spinae (ES), rectus femoris (RF), medial hamstring (MH), tibialis anterior (TA), and gastrocnemius (GcM) muscles. In the control group, anticipatory inhibition before load release and load-related modulation of the inhibition were observed in all the dorsal muscles recorded (ES, MH, and GcM). In the SDCP group, similar results were obtained in the trunk muscle (ES), but not in the lower limb muscles (MH and GcM), although individual differences were seen, especially in MH. Anticipatory activation of the ventral lower limb muscles (RF and TA) and load-related modulation of the activation were observed in both participant groups. CoP path length during load release was longer in the SDCP group than in the control group. The present findings suggest that individuals with SDCP exhibit deficits in anticipatory inhibition of postural muscles at the dorsal part of the lower limbs, which is likely to result in a larger disturbance of postural equilibrium.

Keywords: anticipatory postural adjustment, spastic diplegic cerebral palsy, inhibition, electromyography, center of pressure
INTRODUCTION

Spastic diplegia is a form of cerebral palsy, which bilaterally affects the lower limbs more than the upper limbs (du Plessis 2004). Although many individuals with spastic diplegic cerebral palsy (SDCP) have the ability to stand and walk independently with or without 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 SDCP. This will enhance understanding of their postural deficits and thereby facilitate therapeutic development.

When standing humans voluntarily move their arm, the postural muscles of the lower limbs and trunk that control standing posture are activated in advance of the focal muscles that move the arm rapidly (Belen'kiĭ et al. 1967). This type of postural control, known as anticipatory postural adjustment (APA), is believed to reduce the effects of forthcoming perturbations caused by voluntary movement on posture and equilibrium (Bouisset and Zattara 1981; Friedli et al. 1984; Horak et al. 1984). APAs thus probably play an important role in adequately performing various voluntary movements while standing (Boiusset and Do 2008; Massion 1992). Recently, we examined APAs associated with voluntary arm movement while standing in individuals with SDCP (Tomita et al. 2010, 2011). These studies demonstrated that although anticipatory activation of postural muscles is observed in individuals with SDCP, these individuals exhibit several deficits in APAs, including a delayed onset of activation of lower leg muscles, insufficiently smaller anticipatory activation of postural muscles, and a lack of adequate modulation of anticipatory postural muscle activity with changes in the degree
of postural perturbation. These deficits in APAs in individuals with SDCP are likely to be related to a larger disturbance of postural equilibrium during voluntary arm movement (Tomita et al. 2010, 2011). However, certain issues related to APAs in individuals with SDCP remain unclear. Many previous studies have reported that anticipatory inhibition, as well as anticipatory activation of postural muscles plays an important role in maintaining postural equilibrium during voluntary movement while standing (Massion 1992). For example, when a person is standing and holding a load with extended arms, then releases the load, postural muscle activities in the lower limbs and trunk decrease in advance of the load release to reduce the effects of unloading on posture and equilibrium (Aruin and Latash 1995, 1996; Aruin et al. 1996). Although it has been reported that anticipatory inhibition of postural muscle activity is impaired in individuals with neurological diseases, such as stroke (Slijper et al. 2002) and Parkinson’s disease (Aruin et al. 1996), no previous studies have examined inhibitory APAs in individuals with SDCP. However, previous studies examining isometric voluntary force production and relaxation have demonstrated that the capacity to rapidly relax lower limb muscles is reduced in individuals with SDCP (Downing et al. 2009; Tammik et al. 2008). These findings raise a possibility that individuals with SDCP exhibit deficits in inhibitory APAs.

The present study was designed to determine whether individuals with SDCP are able to organize anticipatory inhibition of postural muscle activities, by using a load-release task while standing. We hypothesized that inhibitory APAs may be impaired in individuals with SDCP.

**METHODS**
Participants

Nine individuals with SDCP (SDCP group, 3 females and 6 males, 13–24 years of age) and nine age- and gender-matched individuals without disability (control group, 13–24 years) participated in this study. 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 years prior to participation; no history of any genetic or neurological disorder other than SDCP; and no flexion contracture of the hip or knee joint or plantar flexion contracture of the ankle joint. All participants with SDCP 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 were 17.2 years [standard deviation (SD) = 3.6], 152.3 cm (SD = 7.1), 42.0 kg (SD = 7.4), and 22.4 cm (SD = 1.4), respectively. In the control group, these measurements were 17.0 years (SD = 3.4), 155.0 cm (SD = 7.4), 45.7 kg (SD = 6.5), and 23.2 cm (SD = 1.3), respectively. There were no significant differences between the two groups in any of these parameters.

Following an explanation of the experimental protocols, all participants and their parents (in the case of participants aged 20 years 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
The experimental setup in this study is shown in figure 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 positions of the center of pressure in the mediolateral and anteroposterior directions (CoPx and CoPy, respectively). Arm acceleration during the load-release task 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 horizontal plane.

In the load-release task, participants held wooden grips (10 cm × 17 cm × 1.5 cm) attached beneath a wooden board (17 cm × 35 cm × 1.5 cm) in front of their bodies with both shoulders flexed so that the arms were parallel to the floor (Fig. 1). A load was fixed on the board. After the load was released, the board was caught on metal wires loosely attached to a metal frame placed in front of the participant. The total weight of the board, grip and load was set at 2% of body weight (%BW) for women and 3%BW for men under the light condition. Under the heavy condition, the weight was set at 4%BW for women and 6%BW for men. Since muscle strength of the upper and lower limbs is generally lower in women than in men, many previous studies using a load-lifting task have adopted different percentage of BW based on the participant’s sex (Fujiwara et al. 2009; Maeda and Fujiwara 2007; Tomita et al. 2011). We therefore used a similar method in this study to examine APAs associated with load release.

Electromyograms (EMGs) were recorded using bipolar surface electrodes placed over the following postural muscles on both sides of the body: the rectus abdominis (RA), erector spinae (ES), rectus femoris (RF), medial hamstring (MH), tibialis anterior (TA), and medial head of gastrocnemius (GcM) muscles. Electrodes were placed on the
midportion of the muscle belly. The electrodes were aligned along the long axis of the muscle with an inter-electrode distance of about 2 cm. Electrode input impedance was below 5 kΩ. EMG signals from the electrodes were amplified (×2000) and band-pass-filtered (10–1000 Hz) using an EMG amplifier (MEG-6116, Nihon Kohden, Tokyo, Japan).

Electrical signals of CoP, arm acceleration, and EMGs were recorded to a computer (FMV-C310, Fujitsu, Kanagawa, Japan) via an A/D converter (ADA16-32/2(CB)F, Contec, Osaka, Japan) with a sampling frequency of 2000 Hz and 16-bit resolution using BIMUTAS®II-R software (Kissei Comtec, Nagano, Japan).

Motions of the upper and lower limbs and the trunk during load release were recorded using an 8-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, Oxford, UK). A trigger signal was also recorded to synchronize motion data with electrical signals of CoP, arm acceleration, and EMGs.

Procedure

Participants stood on the force platform at a stance width of 10–15 cm between the heels. Initially, the CoPx and CoPy positions were measured for 10 s while participants maintained a quiet standing posture with their arms by their sides. Five measurements were taken with intermittent 30-s periods of seated rest. The mean of the 5 measurements was used as the participant’s representative CoPx and CoPy positions during quiet standing.
The load-release task then commenced. It has been reported that APAs are modulated by CoP position just before voluntary movement (Benvenuti et al. 1997; Fujiwara et al. 2003). To minimize inter-trial differences in APAs due to differences in CoP position before the load release, a buzzing sound, which was generated by a computer (PP21L, Dell, Round Rock, TX, USA) connected to the force platform, was used to inform participants if they were maintaining CoPx and CoPy positions within a range of ±1.5 cm of the quiet standing position. In each trial, participants were instructed to hold the load in front of their bodies and maintain the CoPx and CoPy positions within the range for at least 3 s while hearing the buzzing sound (Fig. 1A). Within 3 s after cessation of the buzzing sound, participants started to abduct both shoulders by about 15 cm to release the load by their own timing (Fig. 1B). Participants were told to release the load at their maximum speed and to maintain the 15-cm abducted position for about 3 s. Under each condition (light or heavy), the load release was repeated 15 times after 10 practice trials. Participants were given seated rest periods of 5 min after every 5 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, USA). Since no obvious laterality of the CoP position before load release or lateral CoP deviation after load release was observed in either participant group, CoPx data were excluded from analysis. In addition, since no obvious laterality was observed in the kinematic or EMG data in either participant group, these measurements recorded from the left and right side of the body were pooled.
The onset of the rise in the accelerometer signal (i.e., onset of the load release) was identified by visual inspection and was defined as $T_0$. Peak value and time of arm acceleration were calculated (Fig. 2).

To examine differences in shoulder abduction and initial postural alignment between the SDCP group and control group, motion data during load release were analyzed. Peak displacement of the marker taped to the radial styloid process during load release was identified and defined as peak shoulder abduction. The value and time of peak shoulder abduction were then calculated (Fig. 2). Next, angles of the left and right hip, knee, and ankle joints were analyzed. The mean joint angle in the background range was calculated for the period from $-500$ ms to $-250$ ms with respect to $T_0$, and was defined as the initial joint angle. A positive value indicated flexion at the hip and knee joints and dorsiflexion at the ankle joint.

CoPy position was normalized by calculating a percentage of the distance from the heels in relation to the participant’s foot length (%FL). Mean CoPy position was then calculated for the period from $-500$ ms to $-250$ ms with respect to $T_0$ and was defined as the initial CoPy position. The difference between the initial CoPy position and CoPy position at $+50$ ms with respect to $T_0$ was calculated and defined as the anticipatory CoPy displacement (Fig. 2). A positive value indicated anticipatory forward displacement of the CoPy position. In addition, to examine the disturbance in postural equilibrium caused by load release, postural sway in the sagittal plane was quantified by calculating the path length of the CoPy position in the period from $T_0$ to $+1000$ ms and was defined as the CoPy path length (Fig. 2).

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 mean amplitude of a given postural muscle in the background range and that in the anticipatory range was calculated for the period from –500 ms to –250 ms and that from –150 ms to +50 ms with respect to T₀, respectively (Fig. 2). Anticipatory change in EMG amplitude was then calculated by subtracting the mean amplitude of the postural muscle in the background range from that in the anticipatory range. To allow inter-participant comparison, the anticipatory change in EMG amplitude was normalized by calculating a percentage of the change in relation to the mean EMG amplitude in the background range and defined as the normalized anticipatory change in EMG amplitude of the postural muscle [percent background activity (%BA)]. Positive and negative values indicated anticipatory increase and decrease in EMG amplitude with respect to background activity, respectively (i.e., anticipatory activation and inhibition).

To examine percentages of trials with anticipatory activation and inhibition, cases in which EMG amplitude in the anticipatory range increased more than 20% of that in the background range were considered to be anticipatory activation. Cases in which EMG amplitude in the anticipatory range decreased more than 20% of that in the background range were considered to be anticipatory inhibition. In addition, cases in which anticipatory activations were observed in antagonistic postural muscles in the trunk (RA and ES), thigh (RF and MH), and lower leg (TA and GcM) in a given trial were defined as antagonistic coactivation.

Statistical analysis

Mean values of the peak value and time of arm acceleration, peak value and time of shoulder abduction, initial CoPy position, anticipatory CoPy displacement, CoPy path
length, initial joint angles, and normalized anticipatory change in EMG amplitude for
15 trials were calculated separately for each load condition (light or heavy) and used as
representative values for each participant.

A single group \( t \)-test was used to confirm that the normalized anticipatory change in
EMG amplitude of a given postural muscle was significantly different from zero (i.e.,
background activity of the postural muscle). Two-way mixed-design analyses of
variance (ANOVA) were used to assess the effects of participant group (SDCP or
control) and load condition (light or heavy) on each parameter. When a significant
interaction between the two factors was found, post hoc analyses were performed to
examine differences suggested by ANOVA. Differences between the two participant
groups were assessed using post hoc \( t \)-test 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 using a post
hoc paired \( t \)-test with Bonferroni correction. Pearson correlations were used to evaluate
the magnitude of correlation between parameters.

The significance level was set at alpha = 0.05. All statistical analyses were
performed using IBM SPSS Statistics 18 software (SPSS, Chicago, IL, USA).

RESULTS

No participants in either participant group lost their balance or took an extra step
after load release.

Kinematic and kinetic measurements

Table 1 shows the means and SDs of kinematic and kinetic measurements under
each load condition for each participant group. 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). Also, no
significant main effect of participant group or load condition was found for the peak
value or time of shoulder abduction, 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: $F_{1,16} = 9.1, p < 0.01$; ankle: $F_{1,16} = 8.7, p < 0.01$); 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 angle 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
($F_{1,16} = 9.1, p < 0.01$); the initial CoPy position was significantly more anterior 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 initial CoPy position (Table 1).

Significant main effects of participant group ($F_{1,16} = 21.6, p < 0.001$) and load
condition ($F_{1,16} = 17.4, p < 0.01$) were found for the anticipatory CoPy displacement
without a significant interaction between the two factors. The anticipatory displacement
of the CoPy position was significantly more anterior in the control group than in the
SDCP group under both load conditions, and was significantly more anterior under the
Significant main effects of participant group ($F_{1,16} = 11.6, p < 0.01$) and load condition ($F_{1,16} = 58.2, p < 0.001$) were found for the CoPy path length without a significant interaction between the two factors. The CoPy path length was significantly longer in the SDCP group than in the control group under both load conditions, and was significantly longer under the heavy condition than under the light condition for both participant groups.

**Background EMG activity**

Table 2 shows the means and SDs of background EMG amplitudes of ventral and dorsal postural muscles for each participant group under each load condition. Normalized values of the amplitudes were not available in this study, which prevented statistical analyses of these measurements. However, because variability in the amplitude in a given postural muscle between participants was relatively small in each participant group, we compared the background activities between the two participant groups. The mean background EMG amplitudes of RA, ES, and MH were similar between the two participant groups. Those of RF, TA, and GcM tended to be larger in the SDCP group than in the control group.

**Anticipatory EMG activity**

Figure 3 shows group-averaged data of EMGs under each load condition for each participant group. The means and SDs of the normalized anticipatory change in EMG amplitude of postural muscles are shown in figure 4. In the control group, a significant anticipatory decrease in EMG amplitude with
respect to the background activity was observed in all the dorsal postural muscles recorded, i.e., ES (light: $t_8 = 20.4, p < 0.001$; heavy: $t_8 = 18.3, p < 0.001$), MH (light: $t_8 = 11.5, p < 0.001$; heavy: $t_8 = 16.6, p < 0.001$), and GcM (light: $t_8 = 5.0, p < 0.001$; heavy: $t_8 = 6.6, p < 0.001$). In the SDCP group, although such anticipatory inhibition was observed in ES (light: $t_8 = 5.3, p < 0.001$; heavy: $t_8 = 7.5, p < 0.001$), no anticipatory changes in EMG amplitude were observed in MH and GcM.

A significant main effect of load condition was found for the normalized anticipatory change in EMG amplitude of ES ($F_{1,16} = 10.6, p < 0.01$); anticipatory inhibition of ES was significantly larger under the heavy condition than under the light condition for both participant groups. No significant main effect of participant group or significant interaction between the two factors was found for the normalized anticipatory change in ES activity. Significant interactions between participant group and load condition were found for the normalized anticipatory changes in MH and GcM activities (MH: $F_{1,16} = 9.1, p < 0.01$; GcM: $F_{1,16} = 5.7, p < 0.05$). Post hoc analyses revealed the following results. Anticipatory changes in EMG amplitudes of MH (light condition: $t_{16} = 4.2, p < 0.01$, with $t$-test with Bonferroni correction; heavy condition: $t_{9.7} = 3.5, p < 0.05$, with Welch’s test with Bonferroni correction) and GcM (light condition: $t_{16} = 4.2, p < 0.01$; heavy condition: $t_{16} = 5.1, p < 0.001$, with $t$-test with Bonferroni correction) were significantly larger in the control group than in the SDCP group under both load conditions. Although anticipatory inhibition of MH and GcM was significantly larger under the heavy condition than under the light condition in the control group (MH: $t_8 = 7.4, p < 0.001$; GcM: $t_8 = 3.6, p < 0.05$, with paired $t$-test with Bonferroni correction), such load-related modulation was not observed in the SDCP group.
In contrast to the dorsal postural muscles, an anticipatory increase in EMG amplitude with respect to the background activity was observed in RF and TA under both load conditions for both participant groups ($t_8 > 2.3, p < 0.05$) (Figs. 3 and 4). Significant main effects of load condition were found for the normalized anticipatory changes in EMG amplitudes of RF and TA (RF: $F_{1,16} = 9.3, p < 0.01$; TA: $F_{1,16} = 8.2, p < 0.05$); anticipatory increases of RF and TA activities were both significantly larger under the heavy condition than under the light condition in each of the participant groups. No significant main effect of participant group or significant interaction between the two factors was found for the normalized anticipatory change in RF or TA activity. No significant main effect of participant group or load condition was found for the normalized anticipatory change in RA activity, and there was no significant interaction between the two factors.

**Individual differences in anticipatory EMG activity**

Although results from the group-averaged data revealed that a lack of anticipatory inhibition of postural muscles in the dorsal part of the lower limbs (MH and GcM) in the SDCP group was the primary difference between the two participant groups, this result could not rule out the possibility that individual differences existed in dorsal postural muscle activities in the anticipatory range. Thus, we examined this possibility by analyzing percentages of trials with anticipatory activation and inhibition in the dorsal postural muscles (ES, MH, and GcM) for each participant (Fig. 5). In the control group, although the percentages of trials with anticipatory inhibition of the dorsal postural muscles decreased somewhat with a proximal-to-distal gradient, the percentages of trials with anticipatory activation in these muscles were extremely low.
In the SDCP group, although a similar tendency was observed in ES, large variations were found in the lower limb muscles (MH and GcM). In MH, some SDCP participants showed anticipatory inhibition in a relatively higher percentage of trials, whereas some others showed anticipatory activation. In GcM, although few SDCP participants showed anticipatory inhibition, some participants showed anticipatory activation in a relatively higher percentage of trials.

**Antagonistic coactivation**

Figure 6 shows the percentages of trials with anticipatory antagonistic coactivation in the thigh and lower leg. A significant main effect of participant group was found for the percentage of trials with anticipatory coactivation in the lower leg ($F_{1,16} = 6.8, p < 0.05$); the percentage was significantly higher in the SDCP group than in the control group. Similar tendency was found in the thigh, but these data did not reach statistical significance ($F_{1,16} = 3.5, p = 0.08$). However, although some SDCP participants showed anticipatory antagonistic coactivation in a relatively higher percentage of trials (especially in the lower leg), the mean percentages in the thigh and lower leg were less than 30% under both load conditions. No significant main effect of load condition or significant interaction between the two factors was found for the percentage of trials with anticipatory coactivation in the lower leg or thigh.

**Relationships between APAs and initial postural state**

Although deficits in anticipatory inhibition of lower limb muscles (MH and GcM) were observed in the SDCP group, the initial knee and ankle joint angles and the initial CoPy position were different between the two participant groups. To examine whether
the lack of inhibitory APAs in participants with SDCP was due to their initial postural
state, correlations between the initial postural alignment and CoPy position and the
EMG measurements in the SDCP group were calculated under each load condition. No
significant correlations were found between the initial knee joint angle and the
normalized anticipatory change in EMG amplitude of MH (light: $r = 0.18$, $p = 0.64$;
heavy: $r = 0.30$, $p = 0.43$) or GcM (light: $r = 0.48$, $p = 0.18$; heavy: $r = 0.24$, $p = 0.53$).
Similarly, no significant correlations were found between the initial ankle joint angle
and the normalized anticipatory change in MH or GcM ($r < 0.29$, $p > 0.45$), or between
the initial CoPy position and the normalized anticipatory change in MH or GcM ($r <
0.41$, $p > 0.27$).

DISCUSSION

The primary goal of this study was to determine whether individuals with SDCP
exhibit deficits in inhibitory APAs. Our main results can be summarized as follows: (1)
in the control group, anticipatory inhibition of EMG activity was observed in all the
dorsal postural muscles recorded (i.e., ES, MH, and GcM), and the inhibition was larger
under the heavy condition than under the light condition. In the SDCP group,
anticipatory inhibition of dorsal muscle activities and load-related modulation of
inhibitory APAs were observed in the trunk muscle (ES), but not in the thigh and lower
leg muscles (MH and GcM, respectively). Analysis of each SDCP participant’s data
revealed that the lack of anticipatory inhibitory of the lower limb muscle activity was
not present in all cases, but some SDCP participants could inhibit their lower limb
muscle activity, especially in the thigh muscle (MH); (2) anticipatory activation of the
ventral postural muscles in the lower limbs (RF and TA) and load-related modulation of
the anticipatory activation of these muscles were observed in both participant groups. The degree of anticipatory activation of these muscles was similar in the two participant groups; and (3) anticipatory CoPy displacement was smaller in the SDCP group than in the control group. In addition, the disturbance in postural equilibrium during load release, which was quantified by the CoPy path length, was larger in the SDCP group than in the control group under both load conditions.

**APAs associated with load release in individuals without disability**

When a person is standing and holding a load in front of the body, an increase in background activity of dorsal postural muscles is required to counteract an increase in rotational momentum in the forward direction (Aruin and Latash 1995). Release of the load induces disturbance in postural equilibrium in the backward direction, which needs to be counterbalanced to prevent a fall. Anticipatory inhibition of dorsal muscle activities (ES, MH, and GcM) and anticipatory activation of ventral muscle activities (RA and TA) observed in the control group appear to minimize the effects of the forthcoming backward perturbation caused by the load release on posture and equilibrium, which is in agreement with the previous findings in individuals without disability in the load-release task while standing (Aruin and Latash 1996; Slijper et al. 2002). In addition, in the control group, the degree of inhibition and activation of the postural muscle activities was larger under the heavy condition than under the light condition. This finding suggests that individuals without disability adopt a strategy combining modulations of inhibition and activation of antagonistic postural muscles to adapt changes in the degree of postural perturbation caused by load release.
Anticipatory activation of ventral postural muscles in individuals with SDCP

Anticipatory activation of ventral postural muscles in the lower limbs (RF and TA) and load-related modulation of the anticipatory activation were also observed in the SDCP group, resulting in a lack of significant difference in the normalized anticipatory changes in the ventral muscle activities between the two participant groups. This finding suggests that individuals with SDCP have the ability to modulate anticipatory activation of ventral postural muscles with changes in the degree of perturbation. This is a surprising finding, since our previous study on APAs in the load-lifting task revealed that insufficient modulation of anticipatory activation of postural muscles is a primary constraint on APAs while standing in individuals with SDCP (Tomita et al. 2011). In addition, an inability to modulate postural muscle activities to fit task conditions in these individuals has been reported for voluntary forward reach while sitting (van der Heide et al. 2004) and compensatory postural adjustments (CPAs) to backward translation of a support surface while standing (Roncesvalles et al. 2002). Since internally or externally induced postural perturbations in these previous studies are in the forward direction, activation of dorsal postural muscles is needed to maintain postural equilibrium. Thus, the present findings, taken together with the findings of the previous studies (Roncesvalles et al. 2002; Tomita et al. 2011; van der Heide et al. 2004), raise the possibility that individuals with SDCP have the ability to modulate activation of postural muscles in the ventral part, but not in the dorsal part, of the lower limbs.

Previous findings of development of postural adjustments in children without disability suggest that differences exist in developmental patterns between dorsal extensor synergy and ventral flexor synergy. Hadders-Algra and colleagues suggest a larger flexibility in the flexor synergy than in the extensor synergy, which in turn may
indicate a higher degree of supraspinal modulation of the flexor synergy (Hadders-Algra et al. 1998). Recent studies have revealed that the human sensorimotor system in the central nervous system (CNS) shows developmental plasticity after pre- and perinatal brain lesions (Staudt 2010). Although, to our knowledge, no previous studies have examined developmental differences in APAs between the dorsal and ventral postural muscles in individuals with SDCP, brain damage causing SDCP may affect the dorsal and extensor synergies differently, resulting in fewer deficits in anticipatory activation of ventral postural muscles. Alternatively, individuals with SDCP may be able to modulate their postural muscle activities in certain postural tasks, possibly depending on task difficulty. Further studies are needed to test these possibilities.

Anticipatory inhibition of dorsal postural muscles in individuals with SDCP

Group-averaged data showed that in the SDCP group, anticipatory inhibition of dorsal muscle activity and load-related modulation of the inhibition were observed in ES, but not in MH and GcM. This finding suggests that individuals with SDCP have deficits in inhibitory APAs that are manifested in lower limb muscles. There are several potential causes of the differences in inhibitory APAs between the two participant groups, which are related to our experimental setup. First, it has been reported that anticipatory inhibition of postural muscle activity associated with load release is influenced by characteristics of motor action, especially by shoulder abduction speed (Shiratori and Aruin 2007). However, in this study, the peak value and time of arm acceleration and those of shoulder abduction were similar between the two participant groups.

Second, APAs are reportedly modulated by CoP position before voluntary
movement (Benvenuti et al. 1997; Fujiwara et al. 2003). In the present study, the initial CoPy position was significantly more anterior in the SDCP group than in the control group, suggesting a larger stability margin in the backward direction in the participants with SDCP. In addition, it has been reported that initial postural alignment also influences APAs associated with voluntary movement (Aruin 2003). In the present study, the initial angle of the knee joint was significantly more flexed and that of the ankle joint was more dorsiflexed in the SDCP group than in the control group. Therefore, the differences in initial postural state are a potential cause of the differences in inhibitory APAs between the two participant groups. However, no significant correlations were found between the initial postural state (i.e., joint angles or CoPy position) and the normalized anticipatory change in EMG amplitude of dorsal lower limb muscles (MH or GcM), suggesting that the initial postural state was not the primary cause of the lack of inhibitory APAs in the SDCP group. This is in agreement with our previous finding regarding APAs associated with voluntary arm movement in individuals with SDCP (Tomita et al. 2010, 2011). However, Burtner et al. (1998) found that differences in some characteristics of postural muscle activities in CPAs while standing between children with SDCP and those without disability result from differences in initial postural alignment, i.e., crouch or upright. Thus, the present findings, taken together with the findings of the previous studies (Burtner et al. 1998; Tomita et al. 2010, 2011), raise the possibility that initial postural state is not the primary cause of differences in dynamic stance postural control between individuals with SDCP and those without disability, but influences, at least in part, their postural muscle activities to counteract internal or external perturbations.

Last, since we used the percent change in EMG amplitude in the anticipatory range
with respect to that in the background range to quantify the anticipatory changes in postural muscle activities, the absence of anticipatory inhibition of lower limb muscles in participants with SDCP may be due to a difference in background activities between the SDCP group and control group. In particular, if background activities in the lower limb muscles are quite small in individuals with SDCP, a strategy of anticipatory inhibition would not be expected. However, the EMG amplitudes of MH and GcM in the background range were not smaller in the SDCP group than in the control group, although these values were not normalized. A previous study reported that individuals with SDCP exhibit greater background activity before voluntary arm movement than individuals without disability (Girolami et al. 2011). It is thus unlikely that the lack of anticipatory inhibition of dorsal lower limb muscles in the SDCP group was due to muscle activities in the background range. Therefore, the differences in APAs during load release between the SDCP group and control group were probably not due to characteristics of motor action, initial postural state, or background EMG activities.

Additional data analyses of the percentages of trials with anticipatory activation and inhibition for each participant also suggest that some participants with SDCP did not exhibit a lack of inhibitory APAs in the lower limbs, and could inhibit their dorsal lower limb muscle activities, especially in thigh muscle (MH). In this study, ambulation ability in the SDCP group was not uniform (level II or III on the GMFCS), suggesting that differences in severity of gross motor function may be related to differences in the degree of deficits in inhibitory APAs in individuals with SDCP. In addition, individuals with SDCP are well known to have limitations resulting from multiple sensorimotor impairments, e.g., spasticity, muscle weakness, diminished selective motor control, and sensory-perceptual deficits. All of these impairments could influence APAs (Horak
 Differences in these neural and musculoskeletal limitations among participants with SDCP may result in individual differences in deficits in inhibitory APAs.

Antagonistic coactivation in individuals with SDCP

Many previous studies on CPAs to external perturbations (e.g., support surface translation) have revealed that a higher degree of antagonistic coactivation of ventral and dorsal postural muscles is a primary characteristic in individuals with SDCP (Burtner et al. 1998; Nashner et al. 1983; Woollacott et al. 2005). In the present study, the percentages of trials with anticipatory antagonistic coactivation in the thigh and lower leg tended to be higher in the SDCP group than in the control group. However, although some participants with SDCP showed coactivation in a relatively higher percentage of trials (especially in the lower leg), the mean percentages in the thigh and lower leg were less than 30%, suggesting that the damaged CNS in individuals with SDCP does not necessarily select a coactivation strategy in APAs to counteract postural perturbations while standing.

This assumption is also supported by previous findings on APAs (Tomita et al. 2010, 2011) and CPAs (Roncesvalles et al. 2002) in individuals with SDCP. In addition, van der Heide et al. (2004) examined postural adjustments during voluntary forward reach while sitting and proposed that antagonistic coactivation in individuals with SDCP is probably task-specific. Our findings of individual differences regarding anticipatory antagonistic coactivation in the SDCP group also suggest that the severity of gross motor function may influence whether individuals with SDCP adopt a coactivation strategy.
Postural equilibrium during load release in individuals with SDCP

Anticipatory changes in the level of postural muscle activities reportedly result in displacement of CoP position before load release (Aruin and Latash 1995). In the present study, anticipatory CoPy displacement was smaller in the SDCP group than in the control group. This is probably because participants with SDCP exhibited deficits in anticipatory inhibition of lower limb muscles. APAs appear to play an important role in reducing negative effects of voluntary movements on postural equilibrium (Santos et al. 2010). In the present study, the CoPy path length during load release was significantly longer in the SDCP group than in the control group under both load conditions, suggesting that postural disturbance caused by the load release was less compensated in the SDCP group. Anticipatory decreases in MH and GcM activities were less in the SDCP group. In addition, although anticipatory increases in RF and TA activities and an anticipatory decrease in ES activity were apparent in these individuals, the degree of the increases and decrease was not larger than that in the control group. These findings suggest that although anticipatory activation of the ventral lower limb muscles and anticipatory inhibition of the dorsal trunk muscle in advance of the load release were observed in individuals with SDCP, the anticipatory activation and inhibition were insufficient to compensate for the lack of anticipatory inhibition of the dorsal lower limb muscles, resulting in larger disturbances of postural equilibrium during load release while standing.

Possible neural factors relating to deficits in inhibitory APAs in individuals with SDCP

Although the present findings clearly indicate that individuals with SDCP exhibit deficits in anticipatory inhibition of dorsal postural muscle activities in the lower limbs,
the reason why inhibitory APAs are impaired in these individuals cannot be determined based on the present findings alone. However, previous findings on individuals with SDCP raise several possibilities regarding this deficit. First, a loss of selective motor control, which is a common motor impairment in individuals with SDCP (Sanger et al. 2006), may result in deficits in inhibitory APAs during the load-release task. Selective motor control of the lower limbs is reportedly impaired in individuals with SDCP, with a proximal-to-distal gradient (Fowler et al. 2010). A loss of the selectivity in these individuals is likely to be related to abnormal kinematic patterns during the swing phase of gait, such as difficulty extending the knee while the hip is flexed to prepare for the next step (Fowler and Goldberg 2009). Due to impairments in selective motor control, individuals with SDCP may be unable to activate ventral postural muscles (e.g., RF and TA) and inhibit dorsal postural muscles (e.g., MH and GcM) simultaneously, especially in the lower limbs, during voluntary movement while standing.

Second, disturbances in inhibitory mechanisms in the CNS may be related to deficits in inhibitory APAs in individuals with SDCP. It has been reported that inhibitory reflex mechanisms in spinal neuronal networks (e.g., reciprocal inhibition) that are subject to several supraspinal as well as segmental modulatory mechanisms (Jankowska 1992) are impaired in individuals with SDCP (Achache et al. 2010; Leonard et al. 2006). However, APAs are believed to be preprogrammed in the CNS since increases and/or decreases in postural muscle activity are observed before postural perturbations caused by voluntary movements (Bouisset and Zattara 1981; Friedli et al. 1984; Horak et al. 1984). It is thus unlikely that the lack of anticipatory inhibition of dorsal postural muscle activities in the lower limbs in individuals with SDCP is due to dysfunction in inhibitory reflex mechanisms in spinal neuronal networks. Motor dysfunction in individuals with SDCP
is probably due to not only injury to the corticospinal tract, but also to reduction in volume of the cortical gray matter and diminished cortical connectivity within motor cortical areas (Lee et al. 2011). In addition, previous studies using transcranial magnetic stimulation have revealed that cortical inhibitory functions quantified by transcallosal inhibition (Heinen et al. 1999) and postexcitatory silent period (Vry et al. 2008) are impaired in individuals with SDCP. Impairments in inhibitory mechanisms in the CNS, including cortical motor areas, in individuals with SDCP may result in deficits in inhibitory APAs.

Limitations of this study

This is the first study to demonstrate that individuals with SDCP exhibit deficits in inhibitory APAs. However, there are several limitations in this study: (1) since the number of participants was relatively small, it is unclear if other individuals with SDCP, including those who are rated at GMFCS levels other than level II and III, will show characteristics that are similar to those described in this study. In addition, the small number of participants also prevented detailed analyses regarding individual differences in APAs in the SDCP group; (2) several sensorimotor impairments, such as spasticity, muscle weakness, diminished selective motor control, and sensory-perceptual deficits were not tested in this study. It is thus unclear what impairments were related to deficits in inhibitory APAs in participants with SDCP; (3) the participants’ age ranged from 13 to 24 years in this study. Therefore, the present study could not examine developmental changes in inhibitory APAs in individuals with SDCP. Further testing with a greater number of individuals with SDCP is needed to substantiate our findings and to determine their deficits in inhibitory APAs in more detail. It is also clear that future
work is required to identify the neural and musculoskeletal mechanisms involved in deficits in inhibitory APAs in individuals with SDCP.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.
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Figure legends

**Fig 1.** Experimental setup. Participants held wooden grips attached to a wooden board, on which a load was fixed, in front of their bodies (A). Participants then abducted both shoulders and released the load (B). (a) Metal frame. (b) Infrared camera. (c) Metal wires. (d) Accelerometer. (e) Load. (f) Surface electrode. (g) Reflective marker. (h) Force platform.

**Fig 2.** Representative data of arm acceleration (arm acc.), shoulder abduction (shoulder abd.), CoPy position, and EMG activities in the rectus femoris (RF activity) and medial hamstring (MH activity) during a trial with a heavy load in a participant without disability. $T_0$ (i.e., onset of the load release identified by the accelerometer) is indicated with a thick solid line. Note that anticipatory forward displacement of the CoPy position, anticipatory activation of RF, and anticipatory inhibition of MH were observed in this trial.

**Fig 3.** Group-averaged electromyographic data for the activity in the rectus abdominis (RA), erector spinae (ES), rectus femoris (RF), medial hamstring (MH), tibialis anterior (TA), and medial head of gastrocnemius (GcM) muscles under each load condition (light or heavy) in participants with spastic diplegic cerebral palsy (SDCP group) and those without disability (control group). Onsets of the load release are indicated as solid lines. The EMG data in each postural muscle is normalized by dividing by the mean EMG amplitude in the background range (−500 ms to −250 ms with respect to onset of the load release) in the light condition. Note that anticipatory inhibition of EMG activity
in MH and GcM was observed in the control group, but not in the SDCP group.

Fig 4. Means and standard deviations of normalized anticipatory changes in EMG amplitudes with respect to the background activities [percent background activity (%BA)] in each participant group (SDCP or control) under each load condition (light or heavy). Positive values indicate an increase in EMG amplitude in the anticipatory range (−150 ms to +50 ms with respect to onset of the load release), whereas negative values indicate a decrease in EMG amplitude. The normalized anticipatory changes in EMG amplitudes in the SDCP group are shown with filled bars, whereas those in the control group are shown with open bars. Values with a significant difference from zero (i.e., significant changes in EMG amplitudes with respect to the background activities) are indicated with daggers († \( p < 0.05 \), ††† \( p < 0.001 \)). Significant differences in the normalized anticipatory changes in EMG amplitudes between two participant groups and those between two load conditions are indicated with asterisks (* \( p < 0.05 \), ** \( p < 0.01 \), *** \( p < 0.001 \)).

Fig 5. Means and standard deviations of the percentages of trials with anticipatory activation (acti., filled circles) and inhibition (inhi., open circles) in dorsal postural muscles (ES, MH, and GcM) in each participant group (SDCP or control) under each load condition (light or heavy). The percentages for each participant are also shown.

Fig 6. Means and standard deviations of the percentage of trials with anticipatory antagonistic coactivation in the thigh and lower leg in each participant group (SDCP or control) under each load condition (light or heavy). The mean percentages in the SDCP
group are shown with filled circles, whereas those in the control group are shown with open circles. The percentages for each participant are also shown. The percentages for individual participants with SDCP are shown with filled triangles, whereas those for individual participants without disability are shown with open triangles. * $p < 0.05$. 
Ventral postural muscles

Dorsal postural muscles

RA

ES

RF

MH

TA

GcM

Normalized anticipatory change in EMG amplitude (%RA)
SDCP group

Control group

ES

Percentage of trials (%)

MH

Percentage of trials (%)

GcM

Percentage of trials (%)


<table>
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<th></th>
<th>Heavy condition</th>
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<td>Control group</td>
<td>SDCP group</td>
<td>Control group</td>
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<td>Peak arm acceleration (m/s^2)</td>
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<td>26.5 (13.0)</td>
<td>22.5 (9.7)</td>
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<td>Time of peak arm acceleration (ms)</td>
<td>143.3 (30.8)</td>
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<td>16.5 (3.4)</td>
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<td>15.7 (2.9)</td>
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<tr>
<td>Time of peak shoulder abduction (ms)</td>
<td>395.8 (103.9)</td>
<td>364.0 (74.9)</td>
<td>406.4 (82.3)</td>
<td>373.5 (64.1)</td>
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<td>Initial hip joint angle (degree)</td>
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<td>Initial knee joint angle (degree)</td>
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<td>Initial ankle joint angle (degree)</td>
<td>14.6 (12.5)</td>
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<td>Initial CoP path position (%FL)</td>
<td>52.5 (8.1)</td>
<td>41.9 (6.7)</td>
<td>53.3 (9.4)</td>
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<td>Anticipatory CoP displacement (%FL)</td>
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<td>CoP path length (%FL)</td>
<td>15.9 (5.6)</td>
<td>10.0 (1.6)</td>
<td>22.1 (5.9)</td>
<td>14.8 (2.8)</td>
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Mean (SD). Measurements with significant main effect of participant group (a) and that of load condition (b) are indicated with superscripts.
<table>
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<th>Muscles</th>
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<th>Heavy condition</th>
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<tr>
<td></td>
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<td>Control group</td>
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<tr>
<td>Rectus abdominis (μV)</td>
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<td>Gastrocnemius (μV)</td>
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<td>4.9 (2.0)</td>
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Mean (SD).