Published in final edited form as:

Clin Neurophysiol. 2008 June ; 119(6): 1431–1442.

Changes in the activity of the cerebral cortex relate to postural response modification when warned of a perturbation

Jesse V. Jacobs¹, Katsuo Fujiwara², Hidehito Tomita², Naoe Furune², Kenji Kunita³, and Fay B. Horak¹

¹Neurological Sciences Institute, Oregon Health and Science University, Beaverton, OR

²Dept. of Human Movement and Health, Kanazawa University, Kanazawa, Japan

³Research Center for Urban Health and Sports, Osaka City University, Osaka, Japan

Abstract

Objective—To determine whether the cerebral cortex contributes to modifying upcoming postural responses to external perturbations when provided with prior warning of the perturbation.

Methods—Electroencephalographic (EEG) potentials were recorded from 12 healthy human subjects (21–32 years of age) before perturbing their balance with backward translations of a platform under their feet. The subjects responded with and without a visual cue that warned them 2 seconds before the perturbation (the Cue and No Cue conditions, respectively).

Results—Contingent negative variation (CNV) was evident before perturbation onset in only the Cue condition. In the Cue condition, the subjects also produced smaller center of pressure (CoP) displacements than in the No Cue condition. The cue-related difference in the subjects' CNV potentials correlated with the cue-related difference in their CoP displacements. No significant associations existed among the CNV potentials and any cue-related postural adjustments made before the perturbation.

Conclusions—Cortical activity before an externally triggered perturbation associates with modifications of the ensuing postural response.

Significance—This is the first study to demonstrate a cortical correlate for changes in central postural set that modify externally triggered postural responses based on anticipation.

Keywords

EEG; adaptation; posture; balance; cortex; contingent negative variation

Introduction

Injury and death due to falling represent a significant public health concern and become more prevalent with age and neurological disorders (Myers et al., 1996). Thus, it is essential to understand the neural mechanisms that underlie postural responses for the recovery of standing

Contact: Jesse V Jacobs, University of Vermont, 305 Rowell Building, 106 Carrigan Drive, Burlington, VT 05405; E-mail: JJacobs@uvm.edu; Phone: 802-656-8647; Fax: 802-656-6586.

Disclosure: The authors have no conflicts of interest to disclose.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

balance. Postural responses to an external perturbation represent a unique set of behaviors because (1) while they are voluntarily modifiable, they cannot be completely suppressed, and (2) their onset is earlier than a cued voluntary movement but later than that of a segmental spinal reflex (Chan et al., 1979; Diener et al., 1984; Ackermann et al., 1991). Thus, postural responses exhibit characteristics between voluntary and reflexive movement. Because the cerebral cortex is known to be involved in generating voluntary movement, with reflexive movement relegated to the brainstem and spinal cord, these unique qualities of the postural response render the influence of the cerebral cortex enigmatic.

The cerebral cortex can potentially contribute to postural responses either prior to a perturbation, through anticipatory control, or after the onset of a perturbation through cortical response loops. It had been long debated whether the cerebral cortex contributes to generating a postural response via trans-cortical response loops, and the literature suggests that the cerebral cortex does participate in the late phases of the response (Jacobs and Horak, 2007a; Maki and McIlroy, 2007). Regarding anticipatory activation of the cerebral cortex, however, studies utilizing peripheral measures of motion, ground reaction forces, and muscle activity have demonstrated that several high-level cognitive processes, such as expectation (Horak et al., 1989; Ackermann et al., 1991; Maki and Whitelaw, 1993), intention (Gottlieb and Agarwal, 1980; McIlroy and Maki, 1993; Burleigh and Horak, 1996; Grin et al., 2007; Jacobs and Horak, 2007b), and anxiety (Carpenter et al., 2004), influence how an individual responds to a perturbation that threatens balance. In anticipation of a sudden postural perturbation, these changes in “central set” modify postural responses for an upcoming situation (Horak and Macpherson, 1996). As described by Prochazka (1989), central set is a state of readiness to receive a stimulus or make a movement, represented by task-dependent preparatory neural discharge within the central nervous system. Despite the known influence of central set on postural responses, the contribution of the cerebral cortex remains unclear because changes in cortical activity prior to a postural perturbation have never been shown to relate to the modification of postural responses based on changes in central set.

Research attempting to characterize the role of the cerebral cortex on triggered postural responses has focused on electroencephalogram (EEG) potentials that occur *after* a perturbation (known as perturbation-evoked potentials; Dietz et al., 1985; Ackermann et al., 1986; Dimitrov et al., 1996; Quant et al., 2004), rather than recording EEG potentials *prior* to a perturbation. These studies have demonstrated that perturbation-evoked potentials become altered with changes in central set, such as with changes in the predictability of a perturbation (Dietz et al., 1985; Quintern et al., 1985; Adkin et al., 2006) or with a secondary motor task (Quant et al., 2004). These perturbation-evoked potentials occur after the perturbation, however, and are thought to represent error- or conflict-related cortical signals of sensory-motor processing related to the balance disturbance (Dietz et al., 1985; Adkin et al., 2006). Thus, changes in perturbation-evoked potentials represent the consequence of changes in central set, not the process of altered central set itself.

As an alternative to studying perturbation-evoked potentials, pre-movement potentials may provide insight into cortical neurophysiology related to the control of postural responses. The most-commonly studied pre-movement potentials are the Bereitschaftspotential (BP; also called the readiness potential), and the contingent negative variation (CNV). The BP is a slow, negative shift in EEG amplitude that occurs prior to self-initiated voluntary movement (Kornhuber and Deecke, 1965), whereas the CNV potential is a slow, negative shift in EEG amplitude that occurs prior to voluntary movements performed in response to a ‘go’ stimulus that was preceded by a warning stimulus (Walter et al., 1964). Although postural responses to an external perturbation better resemble cued movements accompanied by CNV potentials than self-initiated movements accompanied by BPs, the extent to which automatic postural responses to external perturbations resemble the voluntary reaction-time movements that are

accompanied by CNV potentials remains uncertain. Thus, recording CNV potentials prior to postural responses may provide insight into how the cerebral cortex contributes to modifying postural responses based on changes in central set.

CNV potentials consist of an initial positive deflection that represents a visual orienting response to a warning stimulus (Kok, 1978), followed by a slow negative potential at approximately one second prior to a second stimulus that cues movement onset. This negative potential then peaks around movement onset with its maximum located over the scalp's vertex. The CNV potential represents both non-motor processes related to anticipation of the second stimulus and (similar to the BP) motor processes related to movement preparation (van Boxtel and Brunia, 1994). The neural generators of the CNV potential's motor components have been shown to include the supplementary motor and primary sensory-motor cortex (Lamarche et al. 1995; Hamano et al., 1997; Bares et al., 2007). Therefore, CNV potentials may provide a neural correlate of central set that originates in the cerebral cortex. Although CNV potentials have been shown to precede voluntary postural tasks, such as gait initiation and voluntary body sway (Yazawa et al., 1997; Slobounov et al., 2005), we are not aware of any reports demonstrating the existence of CNV potentials prior to an externally triggered postural perturbation. Thus, unlike previous studies that relate CNV potentials of the cerebral cortex to voluntary movement, we hypothesized that the cerebral cortex would also exhibit CNV potentials that prepare the body for expected external perturbations of balance to modify externally triggered postural responses.

To test this hypothesis, we recorded the EEG signals of healthy human subjects prior to perturbing their balance with backward translations of the support surface under the subjects' feet, with and without a visual warning cue that turned on 2 seconds prior to the perturbation. We predicted that the warning cue would alter the subjects' pre-movement EEG potentials and modify their postural responses. Further, we predicted that these cue-related changes in EEG activity would correlate with cue-related modifications of the postural response, thereby supporting the hypothesis that the cerebral cortex associates with the modification of postural responses based on changes in central set that occur prior to a perturbation. Because changes in central set may also manifest as voluntary changes in initial postural orientation or muscle activation before perturbation onset, we further examined whether cue-related changes in EEG activity correlated with cue-related postural modifications that occur before the perturbation.

Methods

Subjects

According to the Helsinki agreement, 12 healthy human subjects (4 males and 8 females) without neurological or neuromuscular impairment gave informed consent to participate in the protocol that was approved by Kanazawa University, Kanazawa, Japan. On average (range), the subjects were 27 (21–32) years of age, 163 (154–174) cm tall, and weighed 55 (45–62) kg.

Protocol

The task was to maintain balance without taking a step in response to fast, backward movements of the support surface, with and without a visual warning cue. Prior to the task, subjects stood on a moving force plate, with their eyes fixed on a 2×2-cm warning light positioned 1.1 m high and 2.5 m in front of them. Electrodes were attached for EEG, electromyographic (EMG), and electrooculographic (EOG) recordings. Infrared emitting diodes were also taped on the body's joints for kinematic recordings. The subjects then stood with their arms crossed in front of their torso, and they stood with a consistent stance width of 16.5 cm between the heel centers. The positions of the subjects' feet were outlined by tape and checked between each trial to ensure consistent foot placement across trials and conditions.

Before responding to perturbations, the subjects performed five 10-s trials of quiet stance in order to record the average position of the center of pressure (CoP) exerted on the force plate by their feet. For the ensuing perturbation conditions, the subjects were told to maintain this initial position, and we monitored their CoP position by oscilloscope to ensure they complied with this instruction; we discarded any trials in which the subjects exceeded a threshold of ± 1 cm from their average CoP position recorded during quiet stance. The postural perturbations consisted of 18-cm backward translations of the force plate under the subjects' feet, with durations of 548 ms, peak ramp velocities of 35 cm/s, and average initial accelerations of 9 m/s^2 . Perturbations of this speed and magnitude naturally elicit a stepping response when subjects respond to the perturbation without any instruction (Mille et al., 2003), and we chose this perturbation based on personal observations that subjects step to the perturbation but could generally maintain balance without stepping when challenged to do so. Therefore, we chose our perturbation to ensure that we challenged the subjects' stability while still allowing for modifications of the response.

Without any practice trials, the subjects responded to the perturbations in two blocked conditions that were presented in counterbalanced order across subjects: the Cue and No Cue conditions. In the No Cue condition, the subjects attempted to keep their balance without taking a step when the force plate was moved backward, without warning, and at a variable time of 4–8 seconds after having been told to be ready (that is, to maintain their initial stance position with their eyes fixed, without blinking, on the warning light that did not turn on in this condition). In the Cue condition, the subjects also attempted to keep their balance without taking a step in response to the perturbation, but the warning light turned on to indicate that the platform was going to move backward 2 seconds later. Thus, in the Cue condition, a predictable warning stimulus allowed comparison of the effect of predictability on both the subjects' CNV potentials (McAdam et al., 1969) and measures of their postural responses. To ensure changes in the CNV potentials weren't simply due to a visual orienting response to the cue light, in a separate session, we recorded EEG potentials from the same 12 subjects during a No Perturbation condition. In the No Perturbation condition, the subjects were instructed to stand upright and attend to the visual cue being presented twice, two seconds apart, without any platform movement.

Subjects were presented with as many trials as necessary to record 40 trials per condition without any eye-movement artifacts in the EEG signal or without any pre-trial CoP displacements greater than 1 cm beyond the subjects' average quiet stance position. To prevent fatigue, the subjects were allowed to rest at their request, and they rested, at minimum, after every 20 trials.

EEG And EMG Data Collection And Analysis

To extract the subjects' CNV potentials, scalp EEGs were recorded from 8-mm diameter, silver/silver-chloride electrodes at Cz, Pz, Fz, F3, and F4 sites defined by the 10/20 system of electrode placement (Jasper, 1958) and referred to linked electrodes affixed to the earlobes. A BA-1008 amplifier (TEAC Instruments, Japan) amplified the EEG signals by 20000, and the signals were band-pass filtered from 0.05 to 60 Hz. To identify eye movement artifacts, EOGs were monitored by a pair of electrodes placed above and below the left orbit. The EOG signals were amplified by 4000 and band-pass filtered from 0.05 to 30 Hz. The EMG electrodes were placed as a bipolar montage along the length of the contracted muscle with inter-electrode distance of 2 cm over the right: (1) tibialis anterior, (2) medial gastrocnemius, (3) rectus abdominus at the level of the umbilicus, and (4) erector spinae at the level of the third lumbar segment. The EMG signals were amplified by 2000 and band-pass filtered from 10 to 400 Hz. Electrode impedance was kept below $5 \text{ k}\Omega$ for the EEG, EMG, and EOG electrodes. All signals

were sampled at one kHz with 12-bit resolution from 3 seconds before the perturbation to 3 seconds after the perturbation.

For analysis, we discarded trials with eye movement artifacts or unacceptable initial CoP positions (on average $\pm$ SD, 13 ± 10 trials in the Cue condition and 10 ± 8 trials in the No Cue condition; $T = 1.00$, $P = 0.34$) before averaging each subject's EEG waveforms by condition with EPLYZER II software (Kissei Comtec, Japan). Offline, the subjects' average EEG waveforms for each condition were analyzed with Matlab software (MathWorks, USA). The subjects' average EEG waveforms from each condition were zeroed to their average baseline activity during the first 500 ms of recording. For EEG waveforms recorded in the No Cue condition, subtracting the voltage signal from this time interval may not actually represent a subtraction of baseline activity because, in any given trial, subjects may anticipate the perturbation at different moments within the unpredictable inter-trial period. Thus, our measure assumes that motor preparation and anticipation occurring immediately before the perturbation contributes more to the subjects' postural responses than any preparation or anticipation occurring more than 3 seconds before the perturbation.

In order to evaluate the existence of CNV potentials in each condition, we averaged the subjects' EEG waveforms into 500-ms intervals for each condition. We also determined the peak amplitudes of the subjects' CNV potentials in each condition, defined as the maximum negative shift of the EEG signal over the 3000 ms that immediately preceded the perturbation. To determine cue-related differences in the subjects' CNV potentials, we defined cortical modulation as the peak amplitude of a subject's average CNV potential in the No Cue condition, minus the peak amplitude of the subject's average CNV potential in the Cue condition.

To determine whether there were any cue-related anticipatory changes in the subjects' muscle activity preceding the onset of the perturbation, the EMG signals were rectified and integrated by six sequential 500-ms intervals during the 3000 ms that immediately preceded perturbation onset for each condition. To evaluate the subjects' EMG responses to the perturbation, we identified muscle onset latencies as the time following perturbation onset when the rectified EMG signals reached 3 standard deviations above baseline activity, which was defined as the average EMG amplitude exhibited during the 100 ms that immediately preceded perturbation onset. In addition, EMG response amplitude was calculated from the integrated EMG of four sequential 100-ms intervals starting at muscle onset.

The Center of Pressure and Joint Angles

We recorded the subjects' CoP displacements as a primary outcome measure of the postural response. A subject's CoP position was calculated from the ground reaction forces recorded by the force plate under the subjects' feet as previously reported by Fujiwara and colleagues (2003). The data from the force plate were sampled at one kHz with 12-bit resolution. The CoP data were then low-pass filtered offline at 10 Hz. The displacement of the CoP in response to the perturbation was normalized for each subject as the maximum forward position of the foot, minus the maximum forward position of the CoP induced by the perturbation. For each subject, CoP displacements were calculated for each trial, and then averaged by condition. As a measure of response modification due to cueing, we calculated the subjects' mean CoP displacements in the No Cue condition, minus those in the Cue condition.

To evaluate any possible changes in postural orientation before perturbation onset, we calculated the subjects' hip, knee, and ankle angles from the positions of infrared emitting diodes placed on the left 5th metatarsal, lateral maleolus, lateral femoral condyle, greater trochanter, and acromion. The subjects' average initial hip, knee, and ankle angles were calculated from sequential 500-ms intervals over the 3000 ms that immediately preceded perturbation onset. As secondary measures of the subjects' postural responses, we also

calculated the onset times, peak velocities, and peak amplitudes of hip flexion and ankle extension in response to the perturbations in order to quantify modifications in the subjects' hip and ankle response strategies (Horak and Nashner, 1986) between the Cue and No Cue conditions.

Statistical Analysis

For all analyses, decisions to use parametric versus non-parametric statistical tests were based on whether the data satisfied the assumption of normality, as determined by Shapiro-Wilks tests of normality. The only measure to fail the assumption of normality was the number of unintentional steps taken in response to the perturbations. We therefore performed a Wilcoxon t-test to compare the number of steps taken between the Cue and No Cue conditions.

Because postural responses represent a unique behavior, we first sought to determine the existence of a CNV potential prior to the perturbations. Within each condition, a CNV potential was defined as a significant decrease from the average baseline EEG amplitude of zero μV among the five subsequent 500-ms intervals recorded prior to the perturbation. To verify that the CNV potentials exhibited a spatial distribution that was consistent with previous reports on voluntary movement (see Yazawa et al., 1997 as an example), we initially performed a 3-factor, repeated-measures ANOVA that tested for changes in EEG amplitude across electrode sites, time intervals, and conditions. Because CNV amplitudes were maximal at the Cz electrode (such as with voluntary movement), subsequent analysis then focused on the CNV potentials recorded from the Cz electrode. Thus, we then performed a 2-factor, repeated-measures ANOVA that tested for interaction effects on the EEG signal recorded from the Cz electrode among the Cue and No Cue conditions and the 5 interval times that followed the baseline interval. As a secondary analysis, the EEG potentials of the Cue condition were compared to those of the No Perturbation condition with a two-factor, repeated-measures ANOVA that evaluated differences across the 2 conditions and 5 time intervals. The ANOVA statistics were adjusted by Greenhouse-Geisser epsilon (ϵ) corrections to remedy any violations of the assumption of sphericity.

A two-tailed paired t-test was performed to compare the subjects' peak CNV amplitudes between the Cue and No Cue conditions. A two-tailed paired t-test was also used to compare the subjects' mean CoP displacements between the Cue and No Cue conditions. Two-tailed Pearson correlation coefficients determined whether cue-related modulation of the CNV potential was associated with a subject's cue-related modification of their CoP displacements. Because the perturbation occasionally induced unintentional steps in some of the subjects, CNV potentials and CoP displacements were also analyzed after discarding the trials with unintentional steps in order to isolate the feet-in-place postural responses.

To determine whether the subjects exhibited any voluntary modifications to their posture or muscle activity prior to perturbation onset and to determine whether these modifications were different between the Cue and No Cue conditions, we performed 2-factor, repeated-measures ANOVA that compared the subjects' initial CoP positions, and their initial hip, knee, and ankle angles, as well as the integrated EMG amplitudes of their tibialis, gastrocnemius, rectus abdominus, and erector spinae muscles between the Cue and No Cue conditions and across the six 500-ms, pre-perturbation intervals. Any significant cue-related differences in the subjects' initial posture or muscle activity prior to the postural perturbation (that is, any measures with significant condition, or condition-by-interval differences) were then correlated by Pearson coefficients with the subjects' CNV potentials and CoP displacements to determine whether these cue-related adjustments associated with cue-related modulations of cortical activity prior to perturbation onset or with cue-related modifications of the postural response.

Paired t-tests were performed to analyze differences between the Cue and No Cue conditions in the onset times of the subjects' joint angle displacements and muscle activations, as well as in the peak amplitudes and velocities of their joint angle displacements in response to the perturbations. A 2-factor, repeated-measures ANOVA was performed to compare integrated EMG amplitudes in response to the perturbations between the Cue and No Cue conditions and across the four 100-ms intervals of integrated EMG.

Secondary analysis was also performed to test for the effects of condition order and trial-related experience on the subjects' CNV potentials and CoP displacements in response to the perturbations. To test for the effects of condition order on the subjects' CNV potentials and CoP displacements, a 2-sample t-test was performed to compare each of these measures between the subjects who performed the Cue condition before the No Cue condition and the subjects who performed the Cue condition after the No Cue condition. Because cue-related changes in the CoP displacements may also be confounded by learning with repeated trials, rather than representing an immediate improvement due to changes in central set, the subjects' CoP displacements across trials were compared by a 2-way, repeated-measures ANOVA that examined differences between the Cue and No Cue conditions and across trials. For this analysis, the subjects' CoP displacements were averaged by eight 5-trial bins according to trial order.

Results

Changes in EEG Potentials with a Warning Cue

Briefly, our initial analysis on the spatial distribution of CNV potentials demonstrated significant negative shifts of the EEG signal at the Cz and Pz electrodes, but not at the Fz, F3, or F4 electrodes. These CNV potentials were maximal over Cz, consistent with previous reports of CNV potentials prior voluntary postural movement (e.g., Yazawa et al., 1997). In more detail, our primary analysis then demonstrated significant negative shifts in the EEG signal (CNV potentials) were evident at the Cz electrode prior to postural perturbations in the Cue condition, but not in the No Cue condition, during the 1000 ms immediately preceding perturbation onset (intervals 5 and 6), thereby producing a significant interaction effect between conditions and intervals [$F_{4,44} = 21.77$, $\epsilon = 0.453$, $P < 0.00005$]. A significant main effect for interval was also evident [$F_{4,44} = 11.18$, $\epsilon = 0.641$, $P = 0.0001$], but a significant main effect of condition was not [$F_{1,11} = 3.73$, $\epsilon = 1.0$, $P < 0.08$].

A significant positive EEG deflection was evident within 500 ms of when the visual cue was presented in both the Cue and No Perturbation conditions, but the significant negative deflection (the CNV potential) that occurred in the Cue condition was not evident in the No Perturbation condition [main effect of condition: $F_{1,11} = 4.24$, $\epsilon = 1.0$, $P < 0.064$; main effect of interval: $F_{4,44} = 25.72$, $\epsilon = 0.516$, $P = 0.000001$; interaction effect: $F_{4,44} = 10.27$, $\epsilon = 0.623$, $P < 0.0005$]. Thus, visual orienting responses were evident in both conditions but significant CNV potentials were only evident in the Cue condition. Figures 1A and 1B illustrate these results for all three conditions.

The peak amplitudes of the subjects' CNV potentials were, on average, about 300% larger in the Cue condition than in the No Cue condition [$T = 3.81$, $P < 0.005$] (Fig. 1C). Even after removing the trials with steps in the Cue and No Cue conditions, the peak amplitudes of the subjects' average CNV potentials remained larger in the Cue condition than in the No Cue condition [$T = 3.11$, $P < 0.01$] (Fig. 1D). The order in which the subjects performed the Cue and No Cue conditions did not significantly affect peak CNV amplitudes [$T = 1.07$, $P = 0.31$].

Changes in Postural Responses with a Warning Cue

The distance between the subjects' CoP displacements and the front edge of their feet was significantly smaller in the No Cue condition than in the Cue condition [$T = 3.81$, $P < 0.005$] (Fig. 2A and 2B). This effect remained significant after excluding trials with steps from the analysis [$T = 3.79$, $P < 0.005$] (Fig. 2C).

Eight of the 12 subjects stepped less often in the Cue condition than in the No Cue condition, resulting in a trend for fewer trials with unintended steps in the Cue condition [Wilcoxon $T = 1.90$; $P < 0.06$] (Fig. 2D). Only one subject stepped more often in the Cue condition than in the No Cue condition, and 3 subjects did not step in either condition.

Although we counterbalanced the order of the conditions across the subjects, we performed analyses to ensure that the order of the conditions did not contribute to the difference in CoP displacements between the Cue and No Cue conditions. Cue-related differences in the subjects' CoP displacements were not significantly different between the subjects who performed the Cue condition before the No Cue condition and those who performed the Cue condition after the No Cue condition [$T = 1.00$, $P = 0.34$]. The subjects' CoP displacements were immediately affected by the presence of a warning cue and did not change throughout the 40 trials performed in each condition [main effect of condition: $F_{1,11} = 15.47$, $\epsilon = 1.0$, $P < 0.005$; main effect of trial: $F_{7,77} = 1.06$, $\epsilon = 0.330$, $P = 0.37$; interaction effect between trial and condition: $F_{7,77} = 0.57$, $\epsilon = 0.458$, $P = 0.65$] (Fig. 3).

Contrary to the results observed for CoP responses, the presence of a cue did not consistently affect the onset times or response amplitudes of the subjects' joint angles or EMG activations. In fact, there was considerable inter-individual variation in how each subject altered their responses between the Cue and No Cue conditions. Table 1 summarizes the differences between the Cue and No Cue conditions for each subject's CoP displacements, as well as the velocities and onset times of their hip flexions and ankle extensions, demonstrating how the cue-related change in CoP displacements varied in amplitude across subjects, whereas the subjects' kinematic profiles varied in both amplitude and direction.

Relationship Between Cortical Modulation and Response Modification

The difference in the amplitudes of the subjects' peak CNV potentials between the Cue and No Cue conditions significantly correlated with the difference in their CoP displacements between the Cue and No Cue conditions [Pearson $r = 0.59$; $P < 0.05$] (Fig. 4A). With the exception of one outlying subject, the correlation strengthened after removing trials with unintentional steps from the analysis [Pearson $r = 0.83$; $P < 0.005$] (Fig. 4B). The subjects' cue-related modifications in CoP displacements did not correlate with the subjects' age, height, weight, or foot length [range of Pearson $r = 0.14$ to 0.37 ; range of P -values = 0.26 to 0.68].

Modifications in Posture and Muscle Activity Before the Perturbation

No significant main effects of condition were evident for any measure of initial muscle activity or joint angle [range of $F_{1,11} = 0.00$ to 1.97 , range of $\epsilon = 1.0$; range of $P = 0.19$ to 0.98]. In both conditions, a shift in CoP position and increased EMG activity in all four of the recorded muscles were evident during the final 500 ms before perturbation onset compared to earlier intervals [main effect of interval: range of $F_{5,55} = 5.74$ to 8.23 , range of $\epsilon = 0.235$ to 0.334 ; range of $P < 0.05$ to 0.005], but a significant change in initial hip, knee or ankle angle was not evident prior to the perturbations [main effect of interval: range of $F_{5,55} = 1.04$ to 2.75 , range of $\epsilon = 0.227$ to 0.435 ; range of $P = 0.09$ to 0.34]. Changes across time intervals in initial CoP position and initial tibialis muscle activity were greater in the Cue condition than in the No Cue condition [interaction effect for CoP position: $F_{5,55} = 4.09$, $\epsilon = 0.379$, $P < 0.05$; interaction effect for tibialis activity: $F_{5,55} = 5.05$, $\epsilon = 0.313$, $P < 0.05$]. Significant interactions between

conditions and time intervals were not evident for the EMG activity of other muscles or for initial joint angles [interaction effects: range of $F_{5,55} = 1.16$ to 2.87 , range of $\epsilon = 0.224$ to 0.493 ; range of $P = 0.10$ to 0.33]. Although initial CoP positions changed more in the Cue condition than in the No Cue condition over the 3000 ms that preceded the perturbation, during the final 500 ms of this epoch, the subjects' initial CoP positions were similar between the Cue and No Cue conditions: the subjects' initial CoP positions (relative to the position of their heels) were, on average ($\pm$ SD), held at 10.41 ± 0.83 cm in the Cue condition and at 10.38 ± 1.48 cm in the No Cue condition [$T = 0.11$, $P = 0.91$]. Unlike the subjects' cue-related modifications in their CoP responses to the perturbations, the cue-related modifications in the subjects' initial CoP positions and tibialis activity before the perturbations did not significantly correlate with the cue-related changes in their CNV potentials [for initial tibialis activity: Pearson $r = 0.38$, $P = 0.23$; for initial CoP position: Pearson $r = 0.21$, $P = 0.50$] or with the CoP displacements that followed the perturbations [for initial tibialis activity: Pearson $r = 0.04$, $P = 0.89$; for initial CoP position: Pearson $r = 0.41$, $P = 0.18$]. Table 2 summarizes the group's average initial CoP positions, EMG activity, and joint angles by condition and time interval prior to the perturbations.

Discussion

This study demonstrated for the first time that cortical CNV potentials were evident prior to postural perturbations and that modulation of these potentials with changes in central set correlates with modifications of the ensuing postural responses (but not with the anticipatory modifications of initial posture observed prior to the perturbation).

Although CNV potentials appeared not to be present in the No Cue condition, we speculate that this does not represent a complete lack of anticipation and preparation for the postural response, rather that the lack of average potential represents an inconsistently timed anticipation and preparation for the response. That is, we suspect that the subjects always attempted to anticipate the onset of the perturbation, but without the benefit of the predictably timed visual warning cue in the No Cue condition, the subjects were unable to temporally couple their cortical preparation with the perturbation. Thus, over separate trials in the No Cue condition, the subjects' CNV potentials likely occurred at different times prior to the perturbation and, over repeated trials, these potentials would progressively offset each other in the average EEG waveform. This speculation is consistent with previous studies demonstrating that decreased potential amplitudes correspond to an increased difficulty in predicting response timing when testing subjects under different preparatory periods (McAdam et al., 1969; Maeda and Fujiwara, 2007). In contrast, during the Cue condition, the subjects could consistently couple their response preparation with the perturbation and, consequently, their average EEG potentials progressively increased with repeated trials.

The presence of the warning cue not only affected cortical activity, but also affected the magnitude of the subjects' postural responses: the warning cue resulted in smaller peak CoP excursions that remained farther from the front edge of foot support, as well as fewer unintended steps. These cue-related effects were evident regardless of the order in which the conditions were presented, suggesting that the cue itself – not the order in which the conditions occurred – mediated the subjects' modifications of their postural responses. Despite the consistent effect of cueing on the subjects' CoP displacements, no significant differences in joint displacements or EMG activations were evident between the Cue and No Cue conditions. The lack of a significant difference in the subjects' kinematics and EMG activations seemed to reflect the inter-subject variability in the response strategy used to decrease CoP displacements. This variability in postural strategy is not surprising given that our instructional set was simply to ask the subjects to maintain standing balance with their feet in place, without suggesting a type of strategy to accomplish this goal. Unlike specific joint motions or EMG

activations, the reduced CoP excursion represents a generalized measure that reflected the general goal to maintain balance without stepping.

CNV potentials were not evident in the No Perturbation condition, confirming the interpretation that these potentials represented anticipation and motor preparation for the postural response, not solely a visual orienting response. Further, CoP displacements remained farther from the front edge of the subjects' feet in the Cue condition than in the No Cue condition throughout all 40 trials of each condition, without any evidence of trial-related learning. Thus, the warning cue effectively altered postural performance without the need for practice.

The subjects' cue-related modifications of their postural responses were related to the cue-related changes in their pre-perturbation cerebral activity. Further, the change in magnitude of the CNV potentials appeared to specifically relate to the modification of the feet-in-place postural response, not the stepping response, because the relationship between response modification and cortical modulation strengthened when removing trials with unintentional steps from the analysis. One outlying subject exhibited an unusually large CNV potential in the No Cue condition after removing trials with steps, and this large potential was likely because the subject was only able to perform 6 of the 40 trials without stepping in the No Cue condition. Thus, when removing trials with unintended steps, this subject's average CNV potential was not based on enough trials to sufficiently increase the signal-to-noise ratio in order to accurately record the amplitude of the CNV potential.

Although the subjects modified their muscle activity and CoP positions before perturbation onset, most of these modifications were evident in both the Cue and No Cue conditions. For those changes that were different among the Cue and No Cue conditions, these cue-related modifications in initial posture were not related to the subjects' cue-related modulation of CNV potentials or to the cue-related modification of their CoP responses to the perturbations. Thus, the subjects' cortical activity prior to the perturbations appears to be related to modifying the postural response directly, rather than indirectly through modifications in initial posture or muscle activity.

It remains unclear whether the pre-perturbation cortical activity modified the postural responses by changing the response strategy or simply by priming and augmenting existing synergies. Previous studies examining the predictability of perturbation onset on postural responses suggest that the response strategy isn't changed, but simply primed for an earlier response latency (Ackermann et al., 1991). The original study of CNV potentials (Walter et al., 1964) also suggested a similar role for the CNV potential, stating that the CNV represents an "electrical sign of cortical priming whereby responses to associated stimuli are economically accelerated and synchronized", and a recent report on voluntary postural control in response to startle stimuli seems to confirm this interpretation (MacKinnon et al., 2007). Further study, however, is required to more directly test the contribution of the CNV potential to changes in an intended response strategy, particularly given the constraints we imposed on the subjects' ability to step or to use their arms in response to the perturbation.

The cerebral cortex may only relate to postural responses when actively attending to postural preparation because both CNV potentials and postural responses depend on a subject's attention to the movement task (Tecce, 1972; Norrie et al., 2002). Because CNV potentials prior to cued responses are dependent on attention to the cue and include a component that is specifically related to a visual orientation to the warning cue (Tecce, 1972; Kok, 1978), the results suggest that the activation of cortical circuits to modify postural responses may only occur in situations where a loss of balance is anticipated. Such a contribution, however, would still be essential to human behavior outside the laboratory because individuals can often

anticipate potential perturbations based on environmental cues, such as when approaching obstacles or slippery surfaces, when riding public transit, or when participating in sports.

In addition, our study only examined responses to a single type of perturbation, and unpredictable perturbation characteristics may hinder the effectiveness of anticipatory cortical activity to modify a postural response in a context-appropriate manner. Behavioral evidence, however, suggests that healthy subjects are capable of modifying their postural responses through pre-planning based on prior intention or the existence of environmental obstacles, even when responding to perturbations of unpredictable direction, timing, and amplitude (Zettel et al., 2005; Jacobs and Horak, 2007b). Nevertheless, further study is required to understand the role of the cerebral cortex in shaping reactive postural responses to unexpected or unpredictable perturbations.

Because a high-density electrode set was unavailable for source analysis in this study, we cannot confirm the cortical generators responsible for modifying postural responses based on changes in central set. However, subdural recordings of CNV potentials during voluntary finger extensions have demonstrated that the neural generators of the CNV potential comprise an executive circuit that includes the prefrontal and temporal cortex, and a motor circuit that includes the supplementary motor area and primary sensory-motor cortex (Lamarche et al., 1995; Hamano et al., 1997; Bares et al., 2007). Thus, given the similarity among the CNV potentials observed in our study and those observed with voluntary movements, we speculate that these neural networks were also involved in modifying the postural responses of the subjects in this study.

This speculation that the observed cortical activity contributes to the modification of ensuing postural responses also insinuates a causal relationship among the pre-perturbation CNV activity and the observed modifications of the postural response. This study, however, only satisfies some of requirements for a causal association: the changes in cortical activity correlate with the changes in the postural response, the cortical activity occurs prior to the postural response, and the potential confound or modifier of pre-perturbation changes in initial posture did not likely explain this relationship. Nevertheless, this study did not examine all the variables with potential to confound the correlation, nor does it demonstrate that changes in cortical activity are necessary for the modification of postural responses with changes in central set.

In summary, our study demonstrated that activity of the cerebral cortex associates with the modification of postural responses to external perturbations through changes in anticipatory central set. Thus, this study adds to a growing literature, demonstrating that postural responses may be influenced by the activity of the cerebral cortex, such as for the identification and sensory-motor processing of postural instability (Slobounov et al., 2005; Adkin et al., 2006), modification of postural responses through cortical response loops (Jacobs and Horak, 2007a; Maki and McIlroy, 2007), and now through the priming of postural responses before an anticipated perturbation. This literature suggests that movements once considered automatic might be susceptible to voluntary control. Thus, techniques that are used to train voluntary movements (e.g., repetitive training and visualization techniques) may also be useful to train postural responses because the voluntary motor networks of the cerebral cortex access the automated postural networks activated during postural responses. Therefore, individuals with impaired balance may benefit from cognitive training of their postural responses to improve balance control (Rogers et al., 2003; Jobges et al., 2004; Maffiuletti et al., 2005; Woollacott et al., 2005).

Acknowledgements

We thank our research subjects for their participation, Andrew Owings for assisting with hardware setup, Sandra Oster, PhD for editorial assistance, and members of the Human Movement and Health Laboratory of Kanazawa University

for their help with subject preparation and data collection. This research was supported by grant number F31NS048800 from the National Institute of Neurological Disorders and Stroke (Jacobs), grant number 15500436 from the Ministry of Education, Science, Sports, and Culture of Japan (Fujiwara), and grant number AG06457 from the National Institute on Aging (Horak).

References

- Ackermann H, Dichgans J, Guschlbauer B. Influence of an acoustic preparatory signal on postural reflexes of the distal leg muscles in humans. *Neurosci Lett* 1991;127:242–246. [PubMed: 1881636]
- Ackermann H, Diener HC, Dichgans J. Mechanically evoked cerebral potentials and long-latency muscle responses in the evaluation of afferent and efferent long-loop pathways in humans. *Neurosci Lett* 1986;66:233–238. [PubMed: 3725188]
- Adkin AL, Quant S, Maki BE, McIlroy WE. Cortical responses associated with predictable and unpredictable compensatory balance reactions. *Exp Brain Res* 2006;172:85–93. [PubMed: 16418848]
- Bares M, Nestržil I, Rektor I. The effect of response type (motor output versus mental counting) on the intracerebral distribution of the slow cortical potentials in an externally cued (CNV) paradigm. *Brain Res Bull* 2007;71:428–435. [PubMed: 17208661]
- Burleigh A, Horak F. Influence of instruction, prediction, and afferent sensory information on the postural organization of step initiation. *J Neurophysiol* 1996;75:1619–1628. [PubMed: 8727400]
- Carpenter MG, Frank JS, Adkin AL, Paton A, Allum JH. Influence of postural anxiety on postural reactions to multi-directional surface rotations. *J Neurophysiol* 2004;92:3255–3265. [PubMed: 15295016]
- Chan CW, Jones GM, Kearney RE, Watt DG. The 'late' electromyographic response to limb displacement in man. I. Evidence for supraspinal contribution. *Electroencephalogr Clin Neurophysiol* 1979;46:173–181. [PubMed: 86424]
- Diener HC, Dichgans J, Bootz F, Bacher M. Early stabilization of human posture after a sudden disturbance: influence of rate and amplitude of displacement. *Exp Brain Res* 1984;56:126–134. [PubMed: 6468561]
- Dietz V, Quintern J, Berger W, Schenck E. Cerebral potentials and leg muscle e.m.g. responses associated with stance perturbation. *Exp Brain Res* 1985;57:354–384.
- Dimitrov B, Gavrilenko T, Gatev P. Mechanically evoked cerebral potentials to sudden ankle dorsiflexion in human subjects during standing. *Neurosci Lett* 1996;208:199–202. [PubMed: 8733304]
- Fujiwara K, Maeda K, Toyama H. Influences of illusionary position perception on anticipatory postural control associated with arm flexion. *J Electromyogr Kinesiol* 2003;13:509–517. [PubMed: 14573366]
- Gottlieb GL, Agarwal GC. Response to sudden torques about ankle in man. II. Postmyotatic reactions. *J Neurophysiol* 1980;43:86–101. [PubMed: 7351552]
- Grin L, Frank J, Allum JH. The effect of voluntary arm abduction on balance recovery following multidirectional stance perturbations. *Exp Brain Res* 2007;178:62–78. [PubMed: 17051384]
- Hamano T, Luders HO, Ikeda A, Collura TF, Comair YG, Shibasaki H. The cortical generators of the contingent negative variation in humans: a study with subdural electrodes. *Electroencephalogr Clin Neurophysiol* 1997;104:257–268. [PubMed: 9186240]
- Horak FB, Diener HC, Nashner LM. Influence of central set on human postural responses. *J Neurophysiol* 1989;62:841–853. [PubMed: 2809706]
- Horak, FB.; Macpherson, JM. Postural orientation and equilibrium. In: Rowell, LB.; Shepherd, JT., editors. *Handbook of Physiology. Exercise: Regulation and Integration of Multiple Systems. Neural Control of Movement*. New York, NY: Oxford University Press; 1996. p. 255-292.
- Horak FB, Nashner LM. Central programming of postural movements: adaptation to altered support-surface configurations. *J Neurophysiol* 1986;55:1369–1381. [PubMed: 3734861]
- Jacobs JV, Horak FB. Cortical control of postural responses. *J Neural Transm*. 2007electronic publication ahead of print, March 29
- Jacobs JV, Horak FB. External postural perturbations induce multiple anticipatory postural adjustments when subjects cannot pre-select their stepping foot. *Exp Brain Res* 2007;179:29–42. [PubMed: 17091288]

- Jasper HH. The Ten Twenty Electrode System of the International Federation. *Clin Neurophysiol* 1958;10:371–375.
- Jobges M, Heuschkel G, Pretzel C, Illhardt C, Renner C, Hummelsheim H. Repetitive training of compensatory steps: a therapeutic approach for postural instability in Parkinson's disease. *J Neurol Neurosurg Psychiatry* 2004;75:1682–1687. [PubMed: 15548482]
- Kok A. The effect of warning stimulus novelty on the P300 and components of the contingent negative variation. *Biol Psychol* 1978;6:219–233. [PubMed: 667245]
- Kornhuber HH, Deecke L. Hirnpotentialänderungen beim Menschen vor und nach Willkürbewegungen, dargestellt mit Magnetbandspeicherung und Rückwärtsanalyse. *Pflügers Arch Eur J Physiologie* 1964;281:52.
- Lamarche M, Louvel J, Buser P, Rektor I. Intracerebral recordings of slow potentials in a contingent negative variation paradigm: an exploration in epileptic patients. *Electroencephalogr Clin Neurophysiol* 1995;95:268–276. [PubMed: 8529558]
- MacKinnon, CD.; Shiratori, T.; Rogers, MW. Movement-related cortical potentials reflect the progressive preparation of movement sequences. Program No. 292.2. 2007 Neuroscience Meeting Planner; San Diego, CA: Society for Neuroscience; 2007.
- Maeda K, Fujiwara K. Effects of preparatory period on anticipatory postural control and contingent negative variation associated with rapid arm movement in standing posture. *Gait Posture* 2007;25:78–85. [PubMed: 16504512]
- Maffiuletti NA, Agosti F, Proietti M, Riva D, Resnik M, Lafortuna CL, Sartorio A. Postural instability of extremely obese individuals improves after a body weight reduction program entailing specific balance training. *J Endocrinol Invest* 2005;28:2–7. [PubMed: 15816363]
- Maki BE, McIlroy WE. Cognitive demands and cortical control of human balance-recovery reactions. *J Neural Transm.* 2007electronic publication ahead of print, June 8
- Maki BE, Whitelaw RS. Influence of expectation and arousal on center-of-pressure responses to transient postural perturbations. *J Vestib Res* 1993;3:25–39. [PubMed: 8275241]
- McAdam DW, Knott JR, Rebert CS. Cortical slow potential changes in man related to interstimulus interval and to pre-trial prediction of interstimulus interval. *Psychophysiology* 1969;5:349–358. [PubMed: 5399373]
- McIlroy WE, Maki BE. Changes in early 'automatic' postural responses associated with the prior-planning and execution of a compensatory step. *Brain Res* 1993;631:203–211. [PubMed: 8131048]
- Mille ML, Rogers MW, Martinez K, Hedman LD, Johnson ME, Lord SR, Fitzpatrick RC. Thresholds for inducing protective stepping responses to external perturbations of human standing. *J Neurophysiol* 2003;90:666–674. [PubMed: 12711707]
- Myers AH, Young Y, Langlois JA. Prevention of falls in the elderly. *Bone* 1996;18:87S–101S. [PubMed: 8717552]
- Norrie RG, Maki BE, Staines WR, McIlroy WE. The time course of attention shifts following perturbation of upright stance. *Exp Brain Res* 2002;146:315–321. [PubMed: 12232688]
- Prochazka A. Sensorimotor gain control: a basic strategy of motor systems? *Prog Neurobiol* 1989;33:281–307. [PubMed: 2682784]
- Quant S, Adkin AL, Staines WR, Maki BE, McIlroy WE. The effect of a concurrent cognitive task on cortical potentials evoked by unpredictable balance perturbations. *BMC Neurosci* 2004;5:18. [PubMed: 15147586]
- Quintern J, Berger W, Dietz V. Compensatory reactions to gait perturbations in man: short- and long-term effects of neuronal adaptation. *Neurosci Lett* 1985;62:371–376. [PubMed: 4094724]
- Rogers MW, Johnson ME, Martinez KM, Mille ML, Hedman LD. Step training improves the speed of voluntary step initiation in aging. *J Gerontol A Biol Sci Med Sci* 2003;58:46–51. [PubMed: 12560410]
- Slobounov S, Hallett M, Stanhope S, Shibasaki H. Role of cerebral cortex in human postural control: an EEG study. *Clin Neurophysiol* 2005;116:315–323. [PubMed: 15661110]
- Tecce JJ. Contingent negative variation (CNV) and psychological processes in man. *Psychol Bull* 1972;77:73–108. [PubMed: 4621420]
- van Boxtel GJ, Brunia CH. Motor and non-motor aspects of slow brain potentials. *Biol Psychol* 1994;38:37–51. [PubMed: 7999929]

- Walter WG, Cooper R, Aldridge VJ, McCallum W, Winter AL. Contingent negative variation: an electric sign of sensori-motor association and expectancy in man. *Nature* 1964;203:380–384. [PubMed: 14197376]
- Woollacott M, Shumway-Cook A, Hutchinson S, Ciol M, Price R, Kartin D. Effect of balance training on muscle activity used in recovery of stability in children with cerebral palsy: a pilot study. *Dev Med Child Neurol* 2005;47:455–461. [PubMed: 15991865]
- Yazawa S, Shibasaki H, Ikeda A, Terada K, Nagamine T, Honda M. Cortical mechanism underlying externally cued gait initiation studied by contingent negative variation. *Electroencephalogr. Clin Neurophysiol* 1997;105:390–399. [PubMed: 9363005]
- Zettel JL, Holbeche A, McIlroy WE, Maki BE. Redirection of gaze and switching of attention during rapid stepping reactions evoked by unpredictable postural perturbation. *Exp Brain Res* 2005;165:392–401. [PubMed: 15883802]

Fig. 1.

CNV potentials prior to postural perturbations. (A) A subject's average CNV potential at the Cz electrode prior to perturbations from 40 trials in the Cue (thick black line), No Cue (thin gray line), and No Perturbation (thin black line) conditions. By convention, negative shifts in the EEG signal are oriented upward. The vertical dashed line represents the onset of the cue for the Cue condition. The horizontal dashed line represents the zeroed baseline activity. (B) Group mean (SD) amplitudes of the subjects' average EEG potentials, taken over successive 500-ms intervals prior to the perturbation in the Cue (thick black line with filled circles), No Cue (thin gray line with filled circles), and No Perturbation (thin black line with open circles) conditions. An asterisk represents a significant deviation (p -value < 0.05) for that sampling period. (C) The peak amplitudes of the subjects' CNV potentials in the Cue and No Cue conditions. Lines represent average peak amplitudes of individual subjects, and the symbols represent the group mean (SD) peak amplitudes in the Cue (filled circles) and No Cue (open circles) conditions. (D) The peak amplitudes of the subjects' CNV potentials in the Cue and No Cue conditions after removing trials with unintentional steps.

Fig. 2.

Effects of cueing on the postural response. (A) A subject's CoP displacement in response to the perturbation from representative trials in the Cue (thick black line) and No Cue (thin gray line) conditions. The vertical dashed line illustrates the onset of the perturbation. The horizontal dashed line represents the front edge of the subject's foot. The black brackets illustrate the normalized CoP displacement from the front edge of the foot, demonstrating a smaller distance between the CoP and the foot's edge in the No Cue condition than in the Cue condition. (B) For the Cue and No Cue conditions, the subjects' average normalized CoP displacements in response to the perturbations. Lines represent the averages of individual subjects, and the circles represent the group mean (SD) CoP displacements in the Cue (filled circles) and No Cue (open circles) conditions. (C) For the Cue and No Cue conditions, the subjects' average CoP displacements in response to the perturbations, after removing trials with unintentional steps from the analysis. (D) For the Cue and No Cue conditions, the percent of trials in which subjects took a step to maintain balance in response to the perturbations.

Fig. 3.

Trial-related changes in the CoP response. Lines represent the group's mean amplitude (SD) of the subjects' average CoP displacements (normalized to represent the distance in centimeters of the peak CoP displacement from the front edge of the feet) within successive 5-trial intervals when responding to perturbations in the Cue (solid line and filled circles) and No Cue (dashed line and open circles) conditions.

Fig. 4.

The relationship among cortical modulation and postural response modification. (A) The cue-related difference in the subjects' average normalized CoP displacements (postural response modification) compared to the cue-related difference in the peak amplitude of the subjects' average CNV potentials at Cz (cortical modulation). The circles represent subject averages, and the diagonal line represents the best-fit line. (B) The subjects' postural response modification compared to their cortical modulation, after removing trials with steps from the analysis. The circles represent subject averages, and the diagonal line represents the best-fit line, excluding one outlier (represented by the triangle) who was only able to perform 6 trials without steps in the No Cue condition.

TABLE 1
Inter-Subject Variability in Cue-Related Response Modifications

Subj #	Average Response Modification (Cue minus No Cue)					
	CoP Displacement (cm)	Hip Flexion Velocity (deg/s)	Ankle Extension Velocity (deg/s)	Hip Flexion Onset (ms)	Ankle Extension Onset (ms)	
1	0.80	-2	1	-24	-5	
2	0.54	-21	0	1	-20	
3	0.53	-50	4	-27	6	
4	0.53	-71	32	27	-30	
5	0.29	12	32	-14	-13	
6	0.26	86	-1	2	4	
7	0.16	-47	1	-11	-24	
8	0.10	7	4	-8	2	
9	0.10	17	-7	18	11	
10	0.10	-20	-16	-10	3	
11	0.08	-7	16	-34	-57	
12	-0.07	22	6	20	-1	
Range	-0.07 to 0.80	-50 to 86	-16 to 32	-34 to 27	-57 to 11	

TABLE 2
Mean Initial CoP position, EMG Activity, and Joint Angles Prior to the Postural Perturbations

Measure	Condition	500-ms Interval Prior to the Postural Perturbation					
		1	2	3	4	5	6
CoP Position (cm) ^{*A}	Cue	10.25	10.28	10.30	10.32	10.38	10.41
	No Cue	10.30	10.32	10.34	10.34	10.36	10.38
TIB EMG (μ Vs) ^{*A*}	Cue	1.43	1.49	1.57	1.70	1.76	2.11
	No Cue	1.39	1.41	1.47	1.54	1.51	1.54
GAS EMG (μ Vs) ^{**}	Cue	6.42	6.43	6.47	6.61	7.11	7.37
	No Cue	6.21	6.52	6.54	6.72	6.89	7.06
ABD EMG (μ Vs) ^{**}	Cue	11.67	11.91	12.04	12.16	12.79	13.48
	No Cue	11.61	11.83	11.92	11.97	12.13	12.27
ESP EMG (μ Vs) ^{**}	Cue	4.10	4.19	4.14	4.28	4.36	4.45
	No Cue	4.13	4.24	4.26	4.28	4.36	4.40
Hip Angle (deg)	Cue	185.81	185.82	186.02	185.69	185.73	185.56
	No Cue	185.93	185.92	185.90	185.87	185.85	185.81
Knee Angle (deg)	Cue	182.16	182.17	182.02	182.53	182.43	182.40
	No Cue	182.79	182.78	182.77	182.77	182.75	182.76
Ankle Angle (cm)	Cue	107.05	106.98	106.93	106.37	106.32	106.47
	No Cue	107.55	107.54	107.52	107.53	107.52	107.51

* significant main effect of interval
^A significant condition-by-interval interaction; no significant main effects of condition were evident
Abbreviations: TIB = tibialis; GAS = gastrocnemius; ABD = rectus abdominus; ESP = erector spinae