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Worse balance is associated with larger perturbation-evoked cortical responses in healthy young adults

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Abstract

Background: Reactive balance recovery evokes a negative peak of cortical electroencephalography (EEG) activity (N1) that is simultaneous to brainstem-mediated automatic balance-correcting muscle activity. This study follows up on an observation from a previous study, in which N1 responses were larger in individuals who seemed to have greater difficulty responding to support-surface perturbations.

Research Question: We hypothesized that people engage more cortical activity when balance recovery is more challenging. We predicted that people with lower balance ability would exhibit larger cortical N1 responses during balance perturbations.

Methods: In 20 healthy young adults (11 female, ages 19–38) we measured the amplitude of the cortical N1 response evoked by 48 backward translational support-surface perturbations of unpredictable timing and amplitude. Perturbations included a Small (8 cm) perturbation that was identical across participants, as well as Medium (13–15 cm) and Large (18–22 cm) perturbations scaled to participant height to control for height-related differences in perturbation difficulty. To assess individual differences in balance ability, we measured the distance traversed on a narrow (0.5-inch wide) 12-foot beam across 6 trials. We tested whether the cortical N1 response amplitude was correlated to balance ability across participants.

Results: Cortical N1 amplitudes in response to standing balance perturbations (54 ± 18 μV) were inversely correlated to the distance traveled in the difficult beam-walking task (R^2 = 0.20, p = 0.029). Further, there was a significant interaction between performance on the beam-walking task and the effect of perturbation magnitude on the cortical N1 response amplitude,

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Data statement

A labeled MATLAB dataset with all the filtered single trial EEG data at several electrodes is available at https://doi.org/10.5061/dryad.qz612jm9x.

Declaration of Competing Interest

No authors have any financial or personal relationship with other people or organizations that could inappropriately influence or bias this work.

whereby individuals who performed worse on the beam-walking task had greater increases in N1 amplitudes with increases in perturbation magnitude.

Significance: Cortical N1 response amplitudes may reflect greater cortical involvement in balance recovery when challenged. This increased cortical involvement may reflect cognitive processes such as greater perceived threat or attention to balance, which have the potential to influence subsequent motor control.

Keywords

Posture; motor skill; EEG; N1; Walking balance

1. Introduction

Reactive balance recovery invokes hierarchical sensorimotor control, but the role of cortical activity in balance recovery is unclear. Reactive balance recovery begins with automatic brainstem-mediated balance-correcting muscle responses at ~100 ms latency, with the potential for cortically mediated control at longer latencies [1]. Cortical electroencephalography (EEG) recordings during balance recovery reveal a negative peak of activity (cortical N1) localized to the supplementary motor area 100–200 ms after perturbation [2,3].The timing and scalp localization of the cortical N1 are appropriate for a potential cortical role in balance recovery, but the factors influencing the cortical N1 remain unclear. Prior studies have shown that the cortical N1 is impacted by perceived threat [4,5], attention [6,7], and predictability [4,5,8,9]. Here, we investigate an influence of difficulty recovering balance on the cortical N1.

This study assesses a relationship between the cortical N1 and difficulty recovering balance that was suggested, but not explicitly tested, in a prior study [10]. We previously tested the effect of sensory input on the cortical N1 amplitude, finding only a weak effect of sensory input that was limited to a fraction of participants, indicating that the cortical N1 amplitude is not strongly or consistently modulated by sensory input [10]. In contrast to the small within-subjects effect of sensory input, we observed much larger between-subjects differences in N1 amplitudes that were associated with inability to maintain balance without stepping [10]. Specifically, those with larger cortical N1s more frequently required compensatory steps, despite instructions to recover balance without moving the feet [10]. Although these findings suggested that N1 amplitudes are larger when people are more challenged by balance perturbations, difficulty performing the task and larger N1 amplitudes were both associated with shorter participant height [10]. The confound of participant height may have resulted from the larger angular acceleration of shorter bodies in response to identical movements of the support-surface [10], in contrast to studies that define perturbation magnitudes relative to body measurements [11,12]. Now using perturbations that are proportional to body height, we test whether remaining between-subjects differences in N1 amplitudes are associated with balance ability, as an indicator of difficulty recovering balance.

To measure differences in intrinsic balance ability, we used a challenging beam-walking task that has been validated in healthy young adults [13]. While dynamic balance performance

is not a direct measure of reactive balance ability [14], the beam-walking task involves frequent near-falls, and thus those with better reactive balance control perform better in this task. Because perturbations did not push everyone to a point of failure, performance measures from kinematics in perturbations do not reflect the limits of balance ability, and are also impacted by factors such as ankle stiffness and flexibility [15]. In contrast, everyone reached a point of failure on the beam-walking task, yielding an objective performance measure (distance traversed) at the limits of their ability.

We hypothesized that people engage more cortical activity when balance is challenged. Specifically, we predicted that young adults who performed worse on the beam [13] would have larger cortical N1 responses to support-surface perturbations. Based on the prior effect of participant height on the cortical N1 [10], we now use perturbations that are proportional to participant height. We additionally use much larger perturbations with greater differences between magnitudes compared to our prior study to present a greater challenge.

2. Methods

2.1. Participants

We recruited 20 young adults (11 female, ages 19–38) for a research study approved by Emory University's Institutional Review Board. Participants reported no significant history of neurological or musculoskeletal disorders. Participants signed written informed consent before participation. Participants were 26 ± 5 years old (mean ± standard deviation), 168 ± 8 cm tall (range 156–185 cm), and 70 ± 14 kg.

2.2. Perturbations

Participants were given 48 backward translational support-surface perturbations (custom platform, Factory Automation Systems, Atlanta, GA) of unpredictable amplitude and onset timing while barefoot (Fig. 1A). Perturbations were evenly divided between three magnitudes to vary difficulty and to maintain unpredictability of magnitude. The Small perturbation (7.7 cm, 16.0 cm/s, 0.23 g) was identical across participants. To control for height-related differences in perturbation difficulty [10], the Medium (12.6–15.0 cm, 26.6–31.5 cm/s, 0.38–0.45g) and Large (18.4–21.9 cm, 38.7–42.3 cm/s, 0.54–0.64 g) perturbations were linearly scaled to participant height. The temporal characteristics (i.e. the shape) of the acceleration, velocity, and displacement waveforms were identical across perturbation magnitudes for the first 500 ms after perturbation onset (Fig. 2).

Participants were asked to execute a stepping response on half of perturbations and to resist stepping on the other half. Stepping and non-stepping instructions were altered between blocks of six trials in a block-randomized order. Trials containing stepping and non-stepping responses are combined for analyses, except where explicitly stated (below).

2.3. Electroencephalography (EEG) collection

EEG data were collected during the perturbation series, and recording equipment was removed before the beam-walking task. Thirty-two active EEG electrodes (ActiCAP, Brain Products, Germany) were placed on the scalp according to the international 10–20 system.

Electrodes TP9 and TP10 were placed on the skin over the mastoid bones behind the ears for offline re-referencing. After the wired electrode cap was placed on the participant, the active electrode sites were prepared with conductive electrode gel (SuperVisc HighViscosity Electrolyte-Gel for active electrodes, Brain Products) using a blunt-tipped needle, which was simultaneously used to rub the scalp to improve electrode impedance. Mastoid sites were additionally scrubbed with an alcohol swab prior to placement. Impedances below 10 kOhm were obtained for Cz and mastoid electrodes before the start of data collection. Focusing on the Cz electrode is for consistency with prior studies, although the N1 amplitude is similar at adjacent electrodes [16].

To enable subtraction of eye movement and blink artifacts, electrooculography (EOG) data were collected with bipolar passive electrodes (E220x, Brain Products) vertically bisecting the right pupil with a reference electrode on the forehead. Before electrode placement, the skin was scrubbed with an alcohol swab, and electrodes were prepared with high-chloride abrasive gel (ABRALYT HiCl, High-chloride-10 % abrasive electrolyte gel, Brain Products). EEG and EOG data were amplified on an ActiCHamp amplifier (Brain Products) sampling at 1000 Hz, with a 24-bit A/D converter and an online 20 kHz anti-aliasing low-pass filter.

2.4. EEG data preprocessing

Raw EEG data were high-pass filtered offline at 1 Hz with a third-order zero-lag Butterworth filter, mean-subtracted within channels, and then low-pass filtered at 25 Hz. Cz data were re-referenced to mastoids and epoched into 2.4 s segments beginning 400 ms before perturbation onset. Vertical EOG data were similarly filtered and segmented without re-referencing. Blinks and vertical eye movement artifacts were subtracted from the epoched data at Cz using the algorithm developed by Gratton and Coles [17], as described in Payne et al. [10]. Single-trial epochs of Cz data were then baseline-corrected by subtracting the mean between 50–150 ms prior to perturbation onset.

2.5. EEG quantification

Epoched cortical responses were averaged within each participant across trials, both within and across perturbation magnitudes. Cortical N1 peak response amplitudes (μV) and latencies (ms) were then measured between 100–200 ms after perturbation onset in the averaged cortical responses.

2.6. Center of mass (CoM) position and trunk angle

A 10-camera Vicon Nexus 2 motion capture system recorded body motion at 100 Hz during perturbations. Participants wore a reflective 25-marker set used by Vicon's Plug-in Gait model to estimate CoM position. CoM positions were baseline subtracted (−50 to −150 ms baseline) to obtain CoM displacements. Trunk angles relative to the vertical were calculated using a vector from the average position of hip markers to the average position of shoulder markers. One participant was excluded from CoM position and trunk angle calculations due to a missing marker that prevented calculation of one of the body segments (N = 19).

2.7. Quantification of CoM displacement

CoM displacement along the axis of platform motion was averaged across non-stepping responses to Small perturbations and quantified as peak amplitude between 1–2 s after perturbation.

2.8. Quantification of trunk angle

Trunk angles were averaged across non-stepping responses to Large perturbations and quantified as the peak between 0.5–1 s after perturbation.

2.9. Beam-walking task

Balance ability was assessed by beam-walking performance after completion of the perturbation series. Participants were given 6 attempts to walk across a narrow beam (12 feet long, 0.5 inch wide, 1 inch high) while wearing standardized shoes and keeping their arms crossed [13] (Fig. 1B). Participants were not given instructions regarding speed or step length. Each trial ended when the participant (1) reached the end of the beam, (2) stepped off the beam, or (3) uncrossed their arms. Distance traversed was measured as the parallel distance from the start of the beam to the back of the heel on the forward foot when the trial ended. Balance ability is reported as the normalized distance traveled, with a maximum possible score of 1 if the end of the beam was reached on all 6 trials.

2.10. Statistical analyses

Univariate linear regressions tested for correlations between cortical N1 amplitudes or latencies and potential confounding variables, including subject height, age, or kinematic measures, including peak CoM displacement, peak trunk angle, or initial platform acceleration. These regressions did not identify any confounding variables that needed to be accounted for when testing the association between the cortical N1 and balance ability. No variables included in regression tests showed significant deviations from normal distributions based on Kolmogorov-Smirnov tests ($p > 0.05$).

Univariate linear regressions were then used to test for correlations between cortical N1 amplitudes or latencies and beam-walking performance within and across perturbation magnitudes. To demonstrate that the combination of data across stepping and non-stepping reactions did not impact our outcomes, we repeated these regressions separately in stepping and non-stepping data.

Mixed model ANOVAs assessed the within-subjects fixed effect of perturbation magnitude on cortical N1 amplitudes and latencies, accounting for large between-subjects differences as a random effect. N1 amplitudes and latencies met ANOVA assumptions of normality (Kolmogorov-Smirnov) and homogeneity (Levene, O’Brien, and Brown-Forsythe tests).

An additional ANOVA tested the interaction between beam-walking performance and perturbation magnitude on N1 amplitudes. In this analysis, beam-walking performance was represented by a categorical variable, labeling participants by quartiles of beam-walking performance scores. Least square means were used for post-hoc comparisons of N1 amplitudes between perturbation magnitudes within each quartile. All analyses were

performed in Statistical Analysis Systems (SAS) software (SAS Institute, SAS University Edition 2.8 9.4 M6).

3. Results

Participant-averaged cortical N1 response amplitudes of 54 ± 18 μV were observed at 141 ± 14 ms across participants (Fig. 3A). N1 response amplitudes and latencies were not associated with participant height, age, peak CoM displacement, trunk angle, or platform acceleration (all $p > 0.05$). Thus, we did not identify any confounding variables that needed to be controlled for in subsequent between-subjects associations.

As perturbation magnitude increased, cortical N1 response amplitudes increased ($F(2,38) = 20.0$, $p < 0.0001$) and latencies decreased ($F(2,38) = 10.7$, $p = 0.0002$) within participants (Fig. 3B). Cortical N1 response amplitudes increased by 5 or 6 μV with each increase in perturbation magnitude (Fig. 3C, all $p < 0.05$), and cortical N1 response latencies in Large perturbations were ~4 ms shorter than in Medium perturbations, and ~8 ms shorter than in Small perturbations (Fig. 3D, $p < 0.05$). There was no difference in cortical N1 response latency between Small and Medium perturbations ($p > 0.05$).

The difficulty of the beam-walking task was evident in that most participants were unable to reach the end of the beam. The average normalized beam distance travelled was 0.40 ± 0.21 (range 0.15–0.84 across participants). The end of the beam was reached on only 10 trials (8% of trials) by 4 participants (20 % of participants), indicating any ceiling effects were small. Performance on the beam-walking task was not correlated with participant height or age (all $p > 0.05$).

Worse beam-walking performance was associated with larger cortical N1 responses to perturbations during standing. Distance travelled on the beam was inversely correlated with the cortical N1 response amplitude across perturbation magnitudes ($R^2 = 0.20$, $p = 0.029$), with the strongest correlation in Large perturbations (Fig. 4A, $R^2 = 0.24$, $p = 0.016$). This correlation was also observed in Medium perturbations (Fig. 4B, $R^2 = 0.21$, $p = 0.025$), but not in Small perturbations (Fig. 4C, $p = 0.09$). Outcomes were unchanged when considering only stepping responses (Fig. 4, dotted regression lines, Large: $R^2 = 0.24$, $p = 0.017$, Medium: $R^2 = 0.23$, $p = 0.018$, Small: $p = 0.061$) or only non-stepping responses (Fig. 4, dashed regression lines, Large: $R^2 = 0.22$, $p = 0.026$, Medium: $R^2 = 0.17$, $p = 0.039$, Small: $p = 0.12$). Beam-walking performance was not correlated to cortical N1 response latency, within or across perturbation magnitudes (all $p > 0.05$). Beam-walking performance was also not associated with performance measures in perturbations, including CoM displacement or trunk angle ($p > 0.05$).

Worse beam-walking performance was associated with a greater effect of perturbation magnitude on cortical N1 response amplitudes. There was a significant interaction between beam-walking performance and the effect of perturbation magnitude on cortical N1 amplitudes ($F(11,32) = 23.1$, $p < 0.0001$), driven by a greater effect of perturbation magnitude in those with worse beam-walking performance (Fig. 5). In the lowest quartile of beam-walking performance, N1 amplitudes increased with each increase in perturbation

magnitude (Fig. 5 Q1, $p < 0.05$). In the intermediate quartiles, N1 amplitudes increased from Small to Large perturbations (Fig. 5 Q2 and Q3, $p < 0.05$), but not from Small to Medium perturbations ($p > 0.05$). In the highest quartile of beam-walking performance, there was no effect of perturbation magnitude on N1 amplitudes (Fig. 5 Q4, $p > 0.05$).

4. Discussion

Our results support the hypothesis that people engage more cortical activity when balance is challenged. Cortical N1 amplitudes were larger when people had greater difficulty recovering balance, either due to more difficult perturbations or due to lower intrinsic balance ability. The relationship to balance ability was driven by the Large and Medium perturbations, whereas the Small perturbation may have been easy for everyone. Additionally, the effect of perturbation magnitude was greatest in those with the lowest balance ability, while those with the best balance may not have been sufficiently challenged by the larger perturbations. The interaction with balance ability may explain why prior studies have found weak and inconsistent effects of perturbation magnitude on the cortical N1 [5,10,18], as this relationship may be limited to those who have greater difficulty with the larger perturbations. Larger cortical N1s in individuals with worse balance may be related to greater perceived threat [4,5,19] or attention to balance [6,7]. It remains unclear whether this increased cortical activity might compensate for worse balance or even interfere with automatic balance control.

Greater cortical engagement with difficulty recovering balance may reflect greater perceived threat, attention to balance, or cortically-mediated balance control. Our previous study suggesting a relationship between the cortical N1 and difficulty recovering balance was confounded by an association with participant height [10]. Addressing this confound with height-adjusted perturbations, we show that cortical N1 amplitudes are larger in those with worse balance. This association was most apparent in the larger, more challenging perturbations, supporting the idea of increased cortical involvement when balance is challenged. This increased cortical activation could reflect greater perceived threat [4,5,19] in those with lower balance ability. Additionally, the increased cortical activity could reflect greater attention to balance, consistent with the reduction in N1 amplitudes when attention is divided by dual-task performance [6,7], and greater attention to balance in older adults with balance problems [20]. In either case, whether the cortical N1 is involved in cortically-mediated balance-correcting behavior remains to be established.

The interaction between balance ability and perturbation magnitude may explain the small and inconsistent effects of perturbation magnitude on the cortical N1 in prior studies [5,10,18]. Those with the best balance ability exhibited little to no scaling of cortical N1 amplitudes across perturbation magnitudes, suggesting they were not sufficiently challenged. In contrast, those with the lowest balance ability increased N1 amplitudes with each increase in perturbation magnitude. Increases in cortical N1 amplitudes with perturbation magnitude may therefore be secondary to an increase in difficulty, rather than a direct consequence of increasing sensory and motor activity. Additionally, whereas one prior study speculated a ceiling effect, whereby the N1 may be maximally activated by the smallest perturbation in

individuals who appear insensitive to perturbation magnitude [18], our data instead suggest a floor effect, requiring larger perturbations to challenge people with better balance (Fig. 5).

Increasing cortical N1 amplitudes with perturbation difficulty supports a previously suggested relationship to theta frequency (4–7 Hz) brain activity. Time-frequency analyses have been used to suggest the cortical N1 reflects a transient synchronization of theta frequency brain activity [12,21]. Such observations must be interpreted cautiously, as such analyses cannot distinguish synchronization of oscillatory components from individual component peaks [22]. However, more difficult continuous balancing task elicit larger amplitude theta frequency oscillatory brain activity [23,24], similar to increases in cortical N1 amplitudes with difficulty, lending some support to a relationship between cortical N1 responses and theta oscillations.

This study is limited by the small sample size and the lack of trial-by-trial balance performance measures. While our between-subjects measures suggest the cortical N1 amplitude could be a biomarker for difficulty recovering balance, we are unable to clarify the underlying mechanisms. Trial-by-trial balance performance measures in a larger population could test for a causal relationship between the cortical N1 and subsequent balance recovery behavior. While the cortical N1 may represent a purely cognitive event, like a shift of attention [6,7] or threat perception [4,5], such a cognitive event could still impact subsequent motor control. This possibility is supported by the localization of the cortical N1 to the supplementary motor area [2,3], which is anterior to the motor cortex, and posterior to the frontal lobe where executive functions are typically attributed [25]. The supplementary motor area is thought to help translate intention into action by mediating interactions between motor cortex and areas in the frontal cortex [26], thereby mediating interactions between cognitive and motor processes. Further study of the cortical N1 could therefore provide insight into the unexplained association between balance and cognitive problems in older adults [27,28]. Further, by virtue of its cortical location, the supplementary motor area is accessible to noninvasive brain stimulation techniques [29] that are used in motor rehabilitation in other contexts [30].

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Role of the funding sources

Sponsors had no involvement in the study design; collection, analysis, or interpretation of data; writing or decision of whether or where to submit the manuscript.

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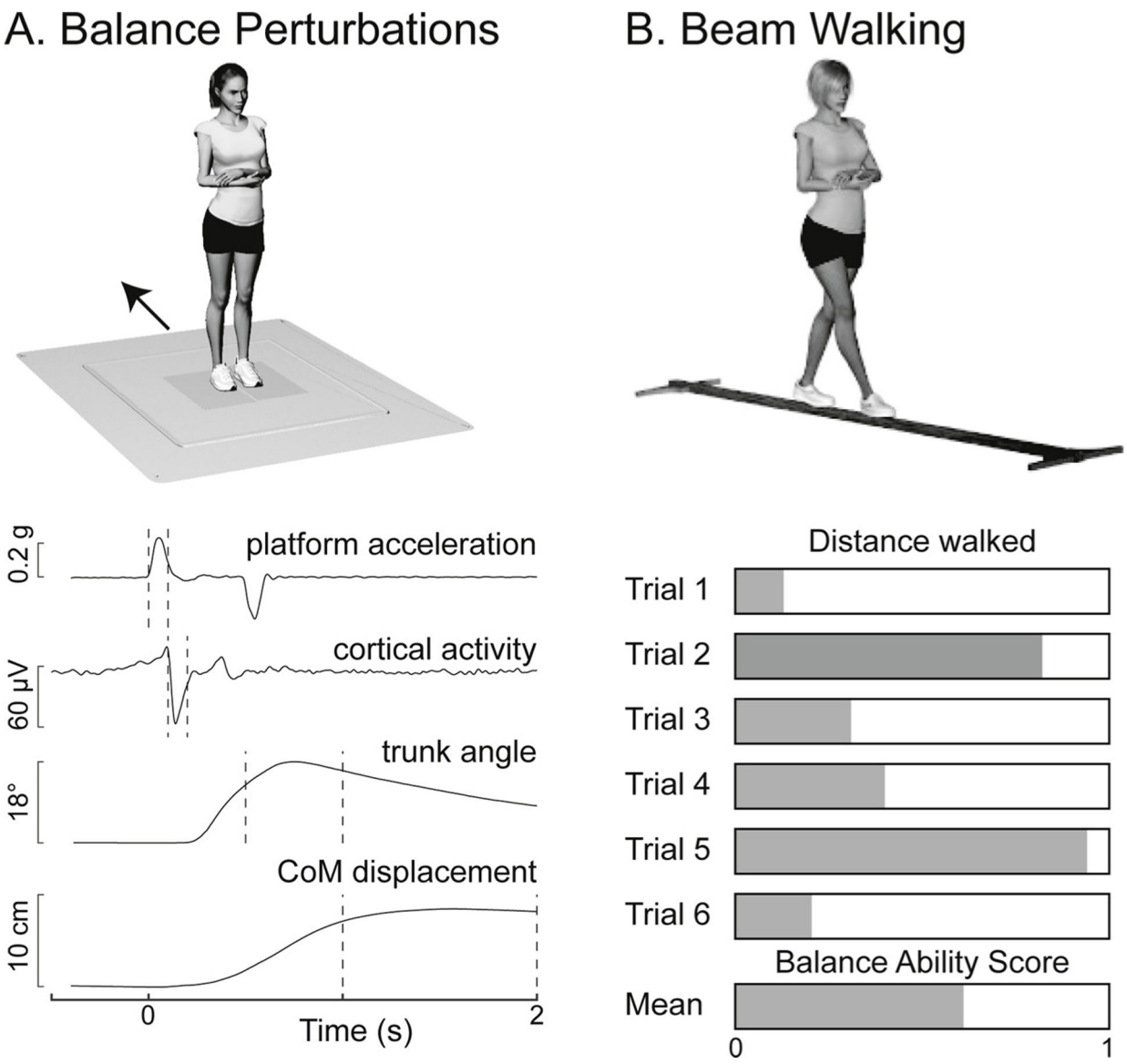

Fig. 1.

Balance tasks. (A) Support-surface perturbations were used to evoke cortical N1 responses. The time window of interest is indicated by vertical dashed lines for platform acceleration (0–100 ms), evoked cortical responses (100–200 ms), trunk angle (0.5–1 s), and center of mass displacement (1–2 s) in data from a single example participant averaged across multiple non-stepping responses to Small perturbations, except trunk angle which is shown averaged across non-stepping responses to Large perturbations. (B) A narrow balance beam was used to assess balance ability. Balance ability scores were calculated as the normalized average distance travelled across six trials. Shoes are depicted in both panels, but shoes were only worn during the beam-walking task. CoM: center of mass; g: units of gravity.

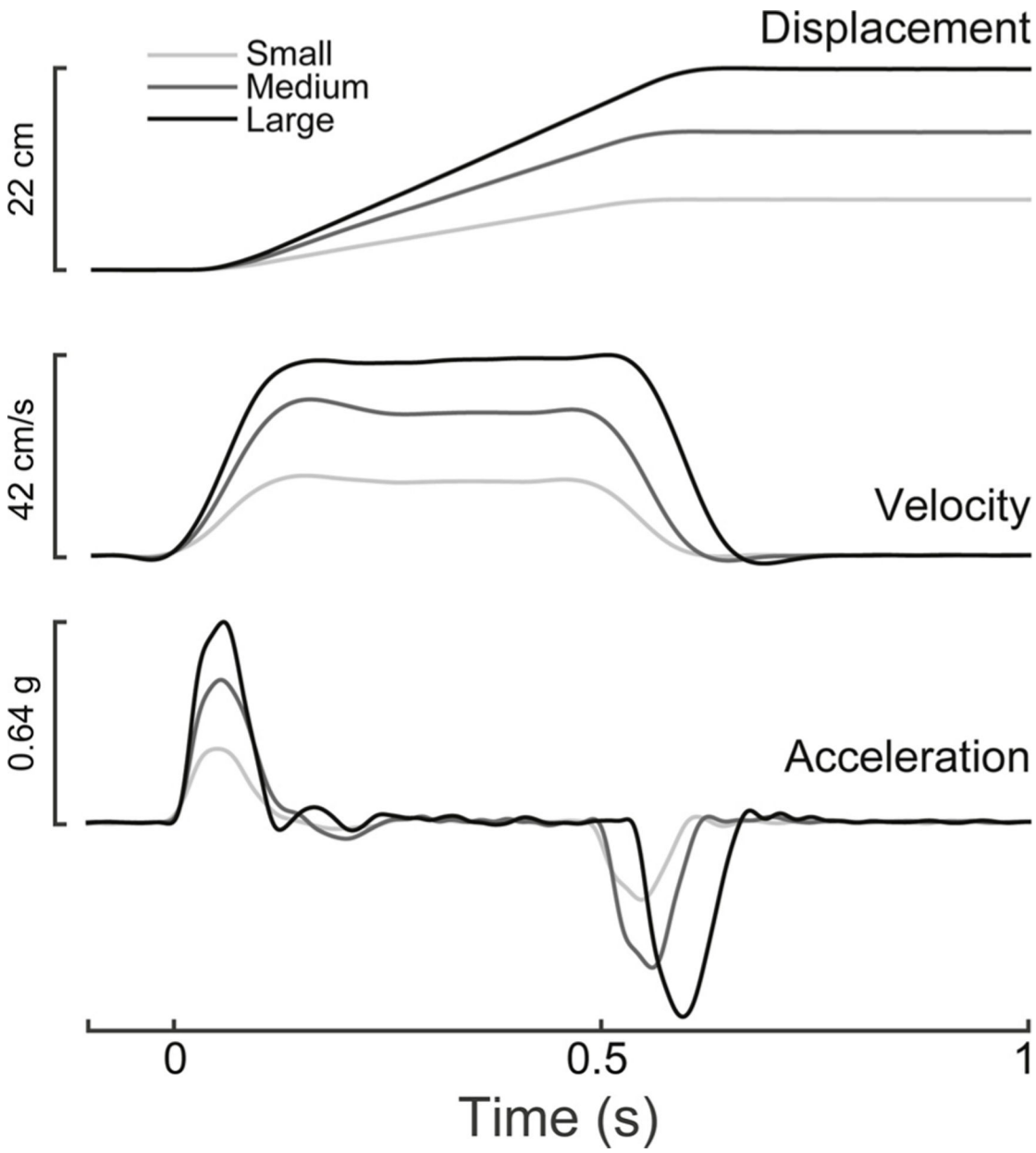

Fig. 2.

Perturbation kinematics for the perturbation magnitudes delivered to the tallest participant. The lightest gray lines correspond to the Small perturbation magnitude, which was identical across participants (7.7 cm, 16.0 cm/s, 0.23 g). The darker two colors correspond to the Medium (12.6–15.0 cm, 26.6–31.5 cm/s, 0.38–0.45 g) and Large (18.4–21.9 cm, 38.7–42.3 cm/s, 0.54–0.64 g) perturbation magnitudes, which were scaled by participant height.

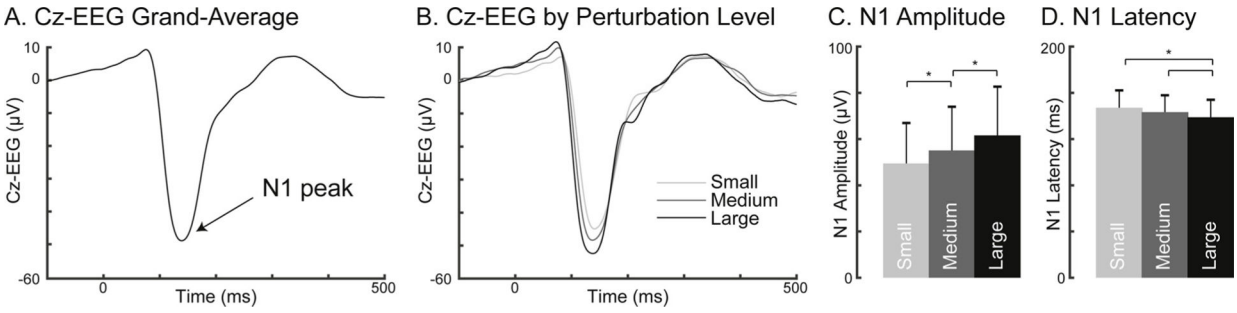

Fig. 3.

Cortical responses across perturbation magnitude. (A) The grand-average cortical response at Cz is shown with a peak N1 response of 54 ± 18 μV occurring 141 ± 14 ms after perturbation onset. (B) Averaged cortical responses are shown by perturbation magnitude, with darker lines for larger perturbation magnitudes. Bar plots display cortical N1 response peak amplitude (C) and latency (D) as the mean and standard deviation across participants. Asterisks (*) indicate significant differences identified in post-hoc Tukey comparisons ($p < 0.05$). In all panels, N = 20 (11 female).

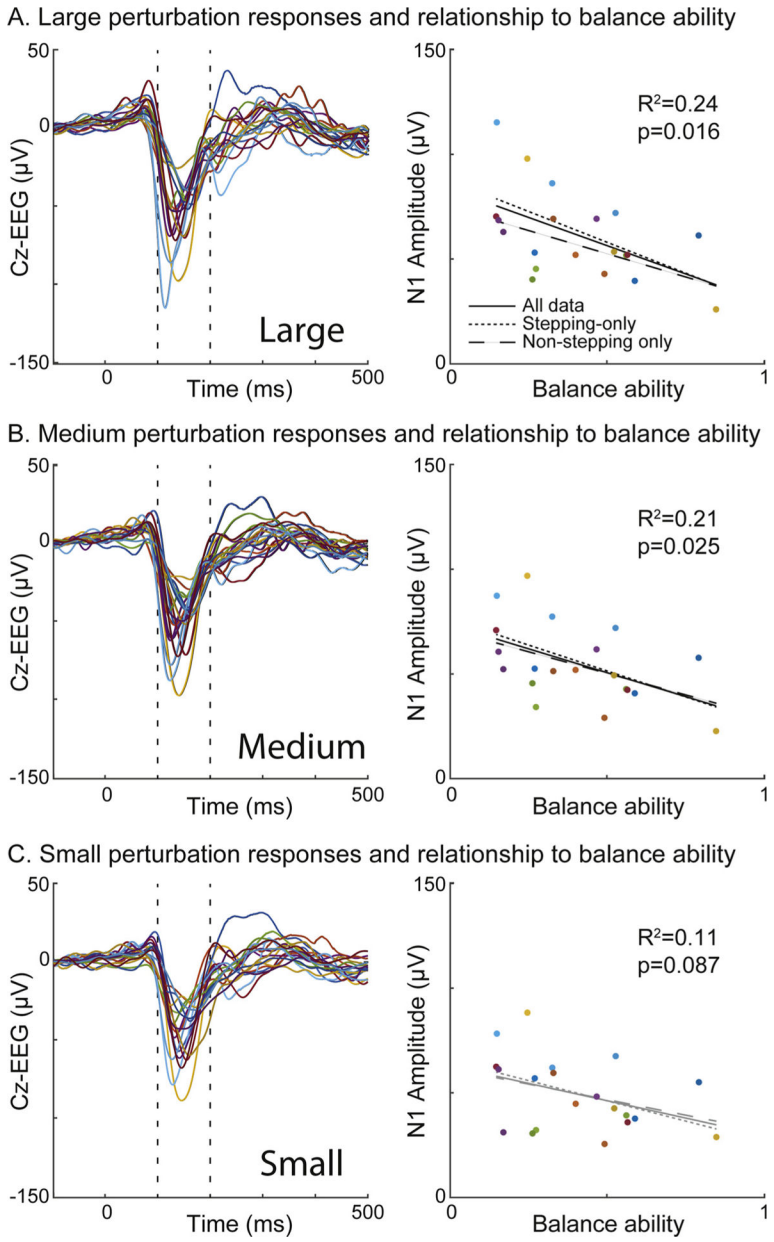

Fig. 4.

Inverse correlation of cortical response amplitudes to balance ability. (A) Participant-averaged cortical responses to Large perturbations are shown on the left. On the right, cortical N1 response peak amplitudes from Large perturbations are plotted on the vertical axis against the normalized distance travelled in the narrow beam-walking task on the horizontal axis. The same are shown for Medium perturbations in (B) and Small perturbations in (C). Different color lines and dots correspond to different participants. The solid regression lines correspond to the data shown in the scatter plots. The dotted and dashed regression lines correspond to stepping reactions (dotted) or non-stepping reactions (dashed) to demonstrate that the combination of stepping and non-stepping data did not influence the outcomes. In all panels, N = 20 (11 female).

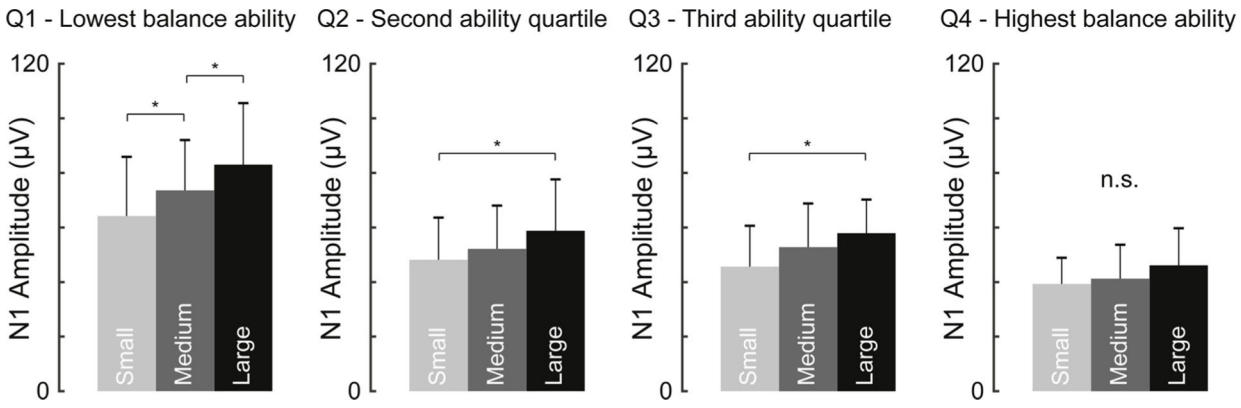

Fig. 5.

The effect of perturbation magnitude differed across balance ability levels. Bar plots illustrate the post-hoc comparisons for the interaction between balance ability and perturbation magnitude on the cortical N1 response amplitude. Participants with the lowest balance ability (Q1) showed significant increases in N1 amplitude with each increase in perturbation magnitude. Participants in the middle two quartiles (Q2 and Q3) showed increases in N1 amplitudes between Small and Large perturbation magnitudes. Participants in the highest balance ability quartile (Q4) showed no effect of perturbation magnitude on N1 amplitudes. N = 5 in each panel.