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RESEARCH ARTICLE | *Control of Movement*

Balance perturbation-evoked cortical N1 responses are larger when stepping and not influenced by motor planning

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Payne AM, Ting LH. Balance perturbation-evoked cortical N1 responses are larger when stepping and not influenced by motor planning. *J Neurophysiol* 124: 1875–1884, 2020. First published October 14, 2020; doi:10.1152/jn.00341.2020.—The cortical N1 response to balance perturbation is observed in electroencephalography recordings simultaneous to automatic balance-correcting muscle activity. We recently observed larger cortical N1s in individuals who had greater difficulty resisting compensatory steps, suggesting the N1 may be influenced by stepping or changes in response strategy. Here, we test whether the cortical N1 response is influenced by stepping (planned steps versus feet-in-place) or prior planning (planned vs. unplanned steps). We hypothesized that prior planning of a step would reduce the amplitude of the cortical N1 response to balance perturbations. In 19 healthy young adults (ages 19–38; 8 men and 11 women), we measured the cortical N1 amplitude evoked by 48 backward translational support-surface perturbations of unpredictable timing and amplitude in a single experimental session. Participants were asked to plan a stepping reaction on half of perturbations, but to resist stepping otherwise. Perturbations included an easy (8 cm, 16 cm/s) perturbation that was identical across participants and did not naturally elicit compensatory steps, and a height-adjusted difficult (18–22 cm, 38–42 cm/s) perturbation that frequently elicited compensatory steps despite instructions to resist stepping. In contrast to our hypothesis, cortical N1 response amplitudes did not differ between planned and unplanned stepping reactions, but cortical responses were 11% larger with the execution of planned compensatory steps compared with nonstepping responses to difficult perturbations. These results suggest a possible role for the cortical N1 in the execution of compensatory steps for balance recovery, and this role is not influenced by whether the compensatory step was planned before the perturbation.

NEW & NOTEWORTHY The cortical N1 response to balance perturbation is larger when executing compensatory steps, suggesting a relationship between the cortical N1 and subsequent motor behavior. Additionally, the cortical N1 response is not impacted by prior planning of the stepping reaction, suggesting that predictability of the motor outcome does not impact the N1 in the same way as predictability of the perturbation stimulus.

biomechanics; EEG; electromyography; kinematics; posture

INTRODUCTION

Subcortically mediated involuntary balance-correcting motor reactions are affected by intention, expectation, and arousal in ways that may depend on descending influences from cortical processes. Reactive balance recovery behavior begins with a subcortically mediated automatic postural response at ~100 ms (Carpenter et al. 1999) that can be followed by voluntary corrections at longer latencies >150 ms (Jacobs and Horak 2007a). The earliest involuntary reactions to balance perturbations can be influenced by instructed motor goals, such as whether to resist or give in to perturbations (Weerdesteyn et al. 2008), or whether or not to take a step in response to perturbations (Burleigh and Horak 1996; Burleigh et al. 1994; McIlroy and Maki 1993). These involuntary reactions are also reduced in amplitude with experience (Horak et al. 1989; Maki and Whitelaw 1993; Welch and Ting 2014) and predictability (Horak et al. 1989), and enhanced with perceived threat (Carpenter et al. 2004). Such changes in involuntary balance recovery reactions are often attributed to changes in "central set" (Prochazka 1989), referring to the ability of the central nervous system to preselect the gain of stimulus-evoked responses in consideration of motor goals, environmental context, prior experience, and arousal. Similar goal-dependent modulation of perturbation-evoked involuntary muscle activity has also been observed in the upper limbs (Crevecoeur and Kurtzer 2018; Dimitriou et al. 2012; Marsden et al. 1981; Nashed et al. 2012; Rothwell et al. 1980; Weiler et al. 2015). Although it is unclear how such changes in central set occur, the influence of motor goals suggests involvement of higher cortical areas in modulation of the automatic motor responses that are mediated by subcortical circuits.

Cortical N1 responses evoked by balance perturbations are also influenced by expectation and arousal, but the extent to which they are affected by motor intention remains unclear. The cortical N1 response has been localized to the supplementary motor area (Marlin et al. 2014; Mierau et al. 2015) and is simultaneous to the involuntary balance-correcting motor response (Payne et al. 2019). Although the function of the cortical N1 response is unknown, it is reduced in amplitude with prior experience (Mierau et al. 2015; Payne et al. 2019; Quintern et al. 1985) and predictability of perturbations (Adkin et al. 2006; Dietz et al. 1985a; Mochizuki et al. 2009b; Mochizuki et al. 2008) and enhanced with perceived threat (Adkin et al. 2008), much like the evoked motor responses. When perturbations are entirely predictable, the cortical N1 response is absent (Adkin et al. 2006, 2008), but in these cases, a slow and sustained negativity can be observed leading up to perturbation onset (Jacobs and

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Horak 2007b; Mochizuki et al. 2008, 2009b, 2010). These observations seem to suggest that the changes in central set that influence the evoked motor reactions may similarly influence the cortical N1 response. Additionally, much like how the motor responses to perturbation can be evoked through various task-relevant sensory modalities, the cortical N1 response does not appear to depend on a particular sensory modality. For example, in support-surface perturbations, slow peripheral conduction velocities lead to similar delays between the cortical N1 response and the evoked muscle activation, while being unaffected by bilateral loss of vestibular function (Dietz et al. 1985a), suggesting a somatosensory rather than vestibular origin of the cortical N1 in support-surface perturbations, consistent with our prior observation of limited head motion until after the cortical N1 response in our paradigm (Payne et al. 2019). In contrast, in seated whole body perturbations that present a natural vestibular stimulus while all of the joint angles remain unchanged, an N1 response can be observed in neurotypical individuals that is entirely absent in individuals with bilateral loss of vestibular function (Hood and Kayan 1985), suggesting a vestibular rather than somatosensory origin in this type of perturbation. Therefore, the cortical N1 response appears to be evoked nonspecifically by task-relevant sensory information that indicates an unexpected disturbance to body posture, in a manner that is further modified by expectations and context in which the disturbance occurs.

Furthermore, we recently observed larger cortical N1 responses in participants who had greater difficulty resisting compensatory steps (Payne et al. 2019), suggesting a possible relationship to online changes in response strategy or execution of compensatory steps. Whereas prior studies have suggested that the cortical N1 depends on the extent that a perturbation stimulus differs from expectations in terms of sensory inputs (Adkin et al. 2006; Dietz et al. 1985a; Mochizuki et al. 2008, 2009b), it is also possible that the cortical N1 response depends on the extent that the motor response outcome differs from expectations. As such, the cortical N1 response could be involved in incorporating unexpected information during the perturbation into the upcoming motor reaction. Indeed, the supplementary motor area has been widely implicated in the transformation of intention into action through a variety of direct pathways to cortical areas and spinal motor neurons, as well as indirect pathways via basal ganglia-thalamocortical loops [see Goldberg (1985) for an extensive review].

We hypothesized that prior planning of a compensatory stepping reaction would reduce the amplitude of the cortical N1 response to unpredictable balance perturbations. Healthy young adults were given a series of translational support-surface balance perturbations that were unpredictable in timing and amplitude but predictable in direction. We tested the effect of prior planning by comparing cortical N1 responses between planned stepping reactions and unplanned stepping reactions. We tested the effect of stepping by comparing cortical N1 responses between planned stepping reactions and planned nonstepping reactions. Participants were asked to recover balance without taking a compensatory step on half of trials, and on the other half of trials participants were asked to plan and prepare to take a single compensatory step to recover balance in response to the upcoming perturbation. Perturbations varied in magnitude, including a very easy low-magnitude perturbation that did not naturally elicit stepping reactions and a very difficult high-magnitude perturbation that often elicited stepping reactions despite instructions to resist stepping.

METHODS

Participants

Nineteen healthy young adults (8 men, 11 women, ages 19–38 yr) were recruited from Emory University and the surrounding population to participate in the experiment. The protocol was approved by the Emory University Institutional Review Board, and all participants signed written informed consent before participation. Different analyses from these same participants were reported previously (Payne and Ting 2020). Participants were 26 ± 5 yr old (mean ± SD), 168 ± 8 cm tall, and 70 ± 14 kg.

Experimental Protocol

The experimental protocol was adapted from McIlroy and Maki (1993). To test the effects of execution and planning of compensatory steps, we presented participants with a series of ramp-and-hold perturbations in which the floor was displaced backward during quiet standing while participants were instructed whether or not to step (Fig. 1). When instructed to step, participants were told, "When the platform moves, recover your balance by taking a single step forward with your right [or left] leg." When instructed not to step, participants were told, "Do your best to recover balance without taking a step. If you must take a step, please try to do so with your right [or left] leg." Stepping leg was predetermined on the basis of "the leg used to kick a ball."

Each participant was exposed to three levels of perturbations, which will be referred to as easy, moderate, and difficult. As in McIlroy and Maki (1993), the easy perturbation was designed to be so easy that it would not naturally elicit stepping reactions in most participants, while the difficult perturbation was designed to be so difficult that most participants would be unable to resist stepping, even when asked not to do so. The moderate perturbation was included to further reduce the predictability of perturbation magnitude, as described by McIlroy and Maki (1993). The easy perturbation (7.7 cm, 16.0 cm/s, 0.23 g) was the same across participants and could be easily resisted without stepping. To account for the previously observed effect of participant height on evoked cortical responses (Payne et al. 2019), the moderate (12.6–15.0 cm, 26.6–31.5 cm/s, 0.38–0.45 g) and difficult (18.4–21.9 cm, 38.7–42.3 cm/s, 0.54–0.64 g) perturbations were scaled linearly with participant height. This linear relationship was defined such that a participant who is 20% taller than another participant would receive perturbations that were 20% larger in acceleration, velocity, and displacement.

Forty-eight perturbations were delivered to each participant, divided evenly between the three perturbation magnitudes and divided evenly between the two instructed conditions. No practice trials were given before the start of the perturbation series. To prevent any effects of trial order from impacting our between-condition comparisons, the perturbation magnitude and condition that was presented first were balanced

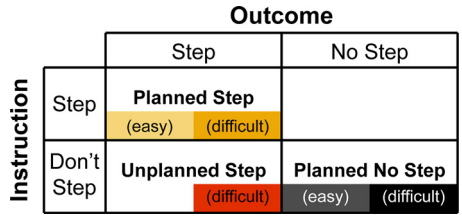


Fig. 1. Experimental design. Participants were instructed whether or not to plan to step in response to upcoming perturbations. In some cases, participants failed to resist taking a compensatory step, resulting in an unplanned step that was not intended before the perturbation. We test the effect of stepping by comparing stepping reactions (planned step) to nonstepping reactions (planned no step), and we test the effect of planning by comparing planned steps to unplanned steps.

across participants. Perturbations were randomized into eight blocks of six trials, each containing two replicates of each perturbation magnitude in random order. Each block was randomly assigned with instructions to step or not to step. Two different randomized block orders were used across participants, and each of the block orders were presented in two different versions in which the instructions were reversed within each block to control for any effect of trial or instruction order. Perturbations were unpredictable in timing and amplitude, but participants were told that all perturbations would be backward movements of the floor so that the forward direction of the compensatory step could be expected and planned for. Perturbations were delivered using a custom perturbation platform (Factory Automation Systems, Atlanta, GA), and perturbation onsets were defined as the beginning of platform acceleration.

To prevent fatigue, a 5-min break was enforced during the perturbation series when the total duration was expected to take longer than 16 min. Not counting these breaks, the average total duration of the perturbation series was 17.4 ± 1.6 min. As described in Payne et al. (2019), to reduce the potential for recording artifacts, perturbations were manually initiated only when electroencephalography (EEG) and electrooculography (EOG) activity was relatively quiescent, based on visual inspection of a live monitor displaying the online EEG data. Intertrial-intervals, measured from perturbation onset to perturbation onset, were intended to be ~20 s, with enough jitter to maintain unpredictability of the timing of the upcoming perturbation, but were extended as long as necessary to allow a return to a stable EEG baseline, and to ensure 5–15 s of quiet standing after recovering upright posture. Excluding the 5-min rest breaks, intertrial-intervals were 22 ± 13 s (means ± SD across all trials, with a minimum of 14 s and a median of 20 s).

Regardless of stepping instructions, participants were asked to cross their arms across their chest, so that they rely on their legs rather than swinging out their arms to recover balance, and to stare at a central location in a poster of a mountain landscape on a wall 4.5 m in front of them. Participants were reminded to relax and look forward whenever electromyography (EMG) activity or eye movements were apparent in the live EEG data. Participants were allowed to blink freely. Participants wore a climbing harness that was attached to the ceiling throughout perturbations for safety in case they were unable to recover their balance. This harness was slack and did not provide any weight support during perturbations, even when bending forward at the hips. The harness would allow a participant's torso to drop more than a foot vertically before suddenly catching them, but no participants lost their balance to the extent of being caught by the harness during the perturbation series.

Ground Reaction Forces

Platform-mounted force plates (AMTI OR6-6) collected ground reaction forces under each foot during perturbations. Ground reaction forces were sampled at 1,000 Hz after a 500-Hz low-pass analog antialias filter.

Quantification of Foot-Off Latency

Single trial recordings of left and right vertical ground reaction forces were relabeled in association with the stance or swing limb based on the instructed stepping leg for each participant. The presence and timing of a stepping reaction was defined as a reduction in the vertical load force under either limb to a value below 5 N within the first 1,000 ms after perturbation. On the basis of these events, stance and swing labels were corrected for any trial in which participants stepped with the opposite leg. Stepping with the wrong leg occurred on 2% of all trials, all but one of which was an accidental step, with the other instance occurring in the medium perturbation magnitude. Latencies to foot-off were then averaged across trial replicates in each condition of interest for each participant.

Quantification of Anticipatory Postural Adjustments

To assess whether the instruction to plan a compensatory step was associated with an anticipatory lateral weight shift before perturbation onset, vertical load forces under the stance and swing limbs were averaged across all trials for each participant within each instruction condition. Specifically, the preperturbation vertical load forces were averaged across all trials in which a participant was instructed to *1*) plan a compensatory step, or *2*) plan a feet-in-place reaction, regardless of the upcoming perturbation magnitude or stepping outcome. The averaged vertical load force data were then quantified as the mean amplitude between 50 and 150 ms before perturbation onset for each instruction condition within each participant under the stance and swing limbs.

Electroencephalography Collection

Thirty-two active EEG electrodes (ActiCAP, Brain Products, Germany) were placed on the scalp, according to the international 10–20 system of electrode placement, with the exception of electrodes TP9 and TP10, which were placed on the mastoid bones for offline rereferencing. Active electrode sites were prepared by applying a conductive electrode gel (SuperVisc 100 gr. HighViscosity Electrolyte-Gel for active electrodes, Brain Products) using a blunt-tip needle, which was simultaneously used to rub the scalp to improve electrode impedance. Mastoid sites were additionally prepared with an alcohol swab before electrode placement. Impedances for Cz and mastoid electrodes were below 10 kΩ before the start of data collection.

Electrooculography (EOG) data were used to collect vertical and horizontal eye movements using a bipolar pair of passive electrodes (E220x, Brain Products) that vertically bisected the pupil of the right eye and another pair of passive electrodes that horizontally bisected the pupils of both eyes. An EOG reference was placed on the forehead. Before electrode placement, the skin was prepared with an alcohol swab, and electrodes were filled with high-chloride abrasive gel (ABRALYT HiCl 250 g, high-chloride-10% abrasive electrolyte gel, Brain Products). EEG and EOG data were amplified on an ActiCHamp amplifier (Brain Products) sampling at 1,000 Hz, with a 24-bit analog-to-digital (A/D) converter and an online 20-kHz low-pass filter. Although both vertical and horizontal eye movements were monitored online to ensure that participants had returned their gaze to the central target between perturbations, only vertical eye movements were used to correct blink and eye movement artifacts (described under *EEG Data Preprocessing*) because the effects of symmetric horizontal eye movements cancel out at midline EEG electrodes (Semlitsch et al. 1986).

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 each channel, and then low-pass filtered at 25 Hz. Cz data were rereferenced to linked mastoids and epoched into 2.4-s segments beginning 400 ms before perturbation onset. Vertical EOG data was filtered and segmented following the same steps without rereferencing. Blinks and vertical eye movement artifacts were subtracted from the epoched data at Cz using the serial regression algorithm developed by Gratton and Coles (Gratton et al. 1983), as described by Payne et al. (2019). Single-trial epochs of Cz data were then baseline-corrected by subtracting the mean voltage between 50 and 150 ms before perturbation onset.

Quantification of EEG

Cortical event-related potentials (ERPs) were created by averaging EEG data at the Cz electrode across like trials within each participant. The cortical N1 response was then quantified as the peak amplitude between 100 and 200 ms after perturbation onset within the ERPs.

Electromyography Collection

Surface electromyographs (EMGs) (Motion Analysis Systems) were collected from tibialis anterior (TA), medial gastrocnemius (MG), and sternocleidomastoid (SC) muscles bilaterally. Electromyography (EMG) signals were anti-alias filtered with a 500-Hz low-pass filter and sampled

at 1,000 Hz. MG and TA were selected on the basis of their roles as primary agonist and antagonist muscles in response to backward translations, and SC was selected as an indicator of startle-related muscle activity (Brown et al. 1991; Campbell et al. 2013; Nonnekes et al. 2015). Skin was shaved and scrubbed with an alcohol pad before electrode placement. Bipolar silver silver-chloride electrodes were used (Nortrode 20, Myotronics, Kent, WA).

EMG Data Preprocessing

Raw EMG signals were segmented into 2.4-s epochs starting 400 ms before the onset of platform motion. Segmented EMG signals were high-pass filtered at 35 Hz offline with a third-order zero-lag Butterworth filter. EMG signals were then mean-subtracted and half-wave rectified. Rectified EMG signals were then low-pass filtered at 40 Hz with a similar Butterworth filter.

Quantification of EMG

Single-trial EMG recordings were normalized, so that each muscle, across all trials within each participant, had a maximum value of 1 between 100 and 200 ms after perturbation onset. Because all perturbations were in the backward direction, i.e., the direction in which the TA is an antagonist, this normalization may make TA-EMG antagonist activity appear unusually large in figures, but this does not impact the within-subjects comparisons across conditions. Left and right MG-EMG and TA-EMG were relabeled in association with the stance or swing limb as described for the ground reaction forces. SC-EMG was averaged across left and right sides. EMG signals were then averaged across replicates of like trials within each participant. EMG data were then quantified as the peak amplitudes observed in early (100–200 ms) and late (200–300 ms) time windows, and as the mean during a baseline (−150 to −50 ms) time window. The early (100–200 ms) time window was selected to contain the automatic portion of the EMG response that has previously been shown to be influenced by changes in central set, without the potential to contain causal influences from the simultaneously occurring cortical N1 response. In contrast, the later (200–300 ms) time window was selected for the potential to receive influences from the cortical N1, in addition to the ongoing automatic response.

Electrodermal Response Collection

Electrodermal responses (EDRs) were collected as a measure of arousal (Sibley et al. 2008, 2010) to assess whether the surprise of an unplanned step increases autonomic responsivity, which would influence the interpretation of any changes in the N1 amplitude. EDRs were collected from the thenar and hypothenar eminences of the right hand using a galvanic skin conductance sensor (Brain Products). Electrodermal responses were amplified on the actiCHamp amplifier and sampled at 1,000 Hz with a 24-bit A/D converter and an online 20-kHz low-pass filter.

Quantification of EDRs

EDRs were averaged across like trials within participants, baseline-subtracted between 50 and 150 ms before perturbation, and quantified as the peak amplitude between 2 and 6 s after perturbation.

Statistical Analyses

Anticipatory postural adjustments. A paired two-tailed t test was used to test for differences in vertical load forces under the stance limb before perturbation onset between the two instruction conditions. All statistical analyses were performed in SAS statistical software, with a significance threshold of $\alpha = 0.05$.

Latencies to foot-off. Paired two-tailed t tests were used to test for differences in the latency to foot-off between planned compensatory steps in easy compared with difficult perturbations and between planned compared with unplanned compensatory steps in difficult perturbations.

Effect of executing a compensatory step. Paired two-tailed t-tests were used to test for differences in muscle, cortical, and electrodermal response amplitudes between (planned) stepping compared with (planned) nonstepping reactions within easy and difficult perturbations. This compares the stepping and nonstepping reactions when both reactions are congruent with the behavior that participants were explicitly asked to execute in response to the perturbation.

Effect of planning a compensatory step. Paired two-tailed t-tests were used to test for differences in muscle, cortical, and electrodermal response amplitudes between planned stepping compared with unplanned stepping reactions in difficult perturbations. This compares the same stepping behavior across conditions that differ in whether that behavior was planned before the perturbation.

Interaction between stepping and perturbation magnitude. Because of the different outcomes of the effect of executing a compensatory step on the N1 amplitude between the easy and difficult perturbation magnitudes, we performed an additional analysis on single-trial data across all three perturbation magnitudes to confirm our findings. We used a generalized linear model, including factors subject, planning (1: plan to step or 2: plan not to step), perturbation magnitude, and the interaction between perturbation magnitude and stepping (1: step was taken, 2: step was not taken). The interaction effect was further broken down by using least-squares means to compare stepping and nonstepping conditions (at $\alpha = 0.05$) within each perturbation magnitude using the "slice" option in SAS.

Associations between anticipatory postural adjustments and evoked responses. To determine whether changes in initial sensory inputs, rather than changes in central set, could explain differences in evoked responses across conditions, we tested for associations between the anticipatory postural adjustments and other variables across conditions. To do this, we created a new variable consisting of the difference in vertical load forces in the stance leg between the instruction sets, as well as variables consisting of the difference in each evoked-response variable (i.e., EMG in early and late time bins in each muscle, N1, and EDR) across each outcome: planned steps versus nonstepping reactions in *1*) easy perturbations and *2*) difficult perturbations; and planned versus unplanned steps in difficult perturbations. Because many of these

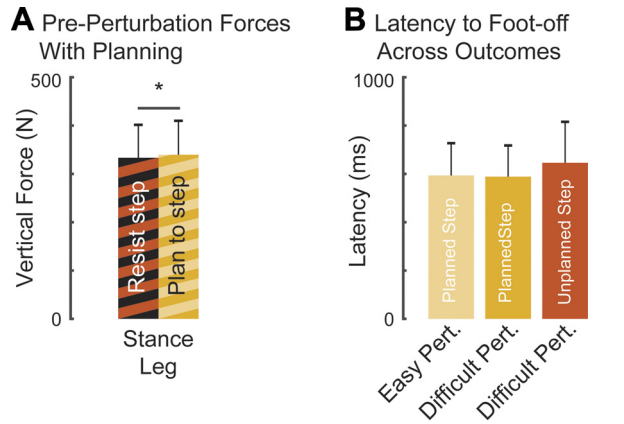
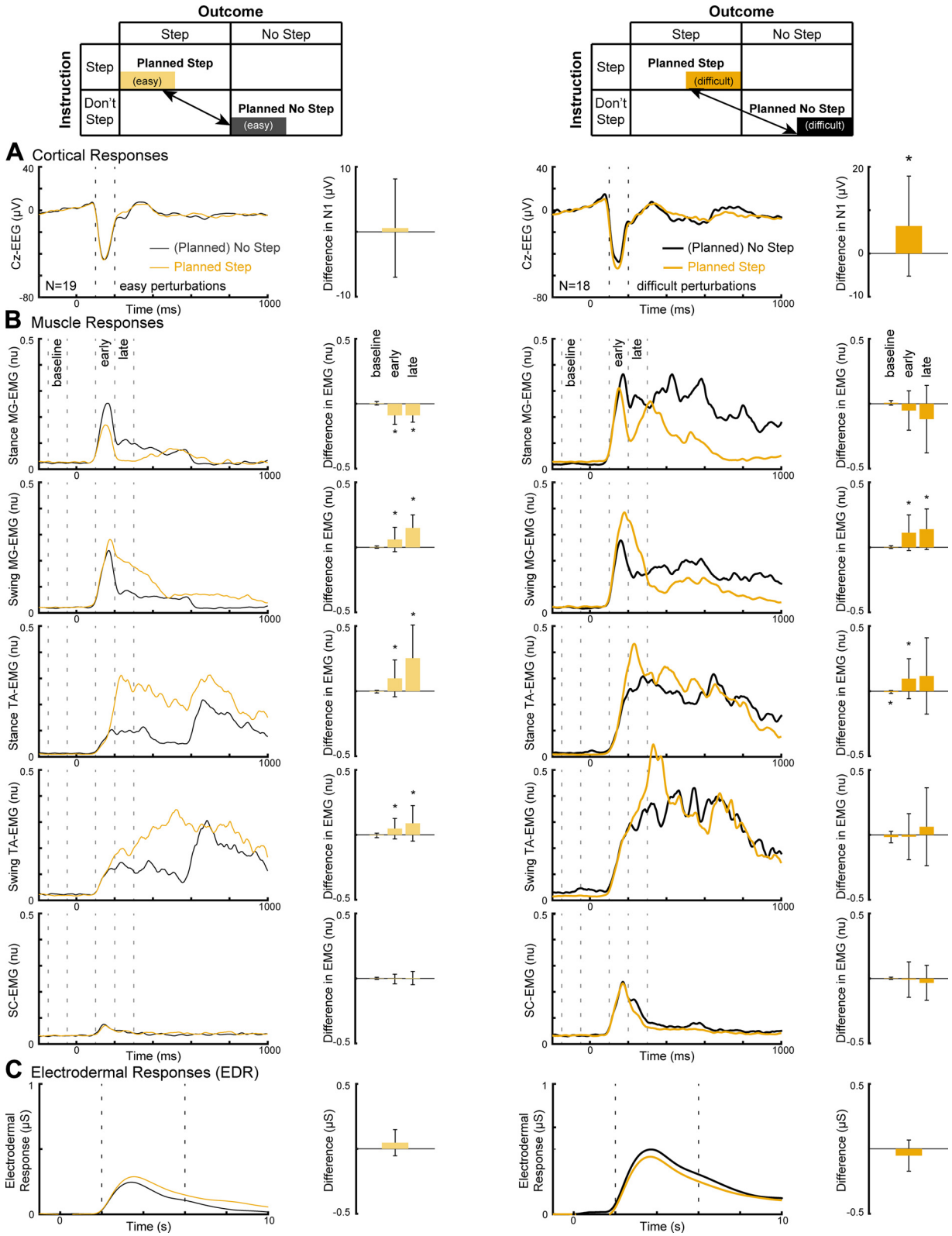


Fig. 2. Instruction to step induced a lateral weight shift but did not influence step latency. *A*: bar plot shows preperturbation (50–150 ms before perturbation onset) vertical load forces under the stance leg. Forces are shown for trials in which participants were asked to step (denoted by shades of yellow), and for trials in which participants were asked to resist stepping (denoted by black and red). *Significant difference, $\alpha = 0.05$. *B*: latency to foot-off is shown for planned steps to easy perturbations (denoted in light yellow), latency to planned steps to difficult perturbations is denoted in dark yellow. Latency to foot-off is shown in red for unplanned steps to difficult perturbations. Latencies to foot-off did not differ between conditions at $\alpha = 0.05$. There is no black bar corresponding to the nonstepping condition in *B* because there was no foot-off in this condition, but this nonstepping condition is included in *A* because this represents the bulk of the trials in which participants were asked not to step.

variables were not normally distributed (Shapiro-Wilk; $P < 0.05$), we used Spearman Rank correlations to test for associations with a significance level of $\alpha = 0.05$. Because our intention was to identify possible confounds, not correcting the significance level for multiple comparison is the more conservative approach. That is, any significant associations would be a cause for concern when attributing between-condition differences to changes in central set.

Associations between changes in N1 amplitudes and changes in subsequent balance-correcting motor responses. It is possible that changes in N1 amplitudes could be associated with subsequent (200–



300 ms) balance-correcting EMG activity. We used Spearman Rank correlations to test for associations between cortical N1 responses and subsequent EMG activity using the between-condition difference variables described in the previous paragraph at the $\alpha = 0.05$. Correcting for multiple comparisons ($\alpha = 0.05/3$) had no impact on the outcomes.

RESULTS

Behavior and Biomechanics

When asked to recover balance without stepping, participants were able to successfully resist stepping on 99 ± 4% (means ± SD) of easy perturbations but only 61 ± 30% of difficult perturbations. In contrast participants were 100% successful at executing planned steps in difficult perturbations, and 97 ± 8% successful at executing planned steps in easy perturbations. Rare failures to execute planned steps in easy perturbations consisted of cases in which participants either "forgot to step" or executed steps too late (foot-off >1,000 ms after perturbation); these trials were excluded from analysis. In easy perturbations, participants had 7.9 ± 0.3 (means ± SD deviation, range: 7–8) successful nonstepping trials and 7.7 ± 0.7 (range: 5–8) planned stepping trials included for analyses. In difficult perturbations, participants had 5.0 ± 2.5 (range 0–9) successful nonstepping trials, 7.9 ± 0.2 (range 7–8) planned stepping trials, and 3.1 ± 2.4 (range 0–8) unplanned stepping trials included for analyses. Three participants did not take any unplanned steps and were excluded from paired comparisons of unplanned versus planned stepping reactions. One of these participants was given one additional difficult perturbation at the end of the series as one more chance to elicit an accidental step, resulting in the upper bound of 9 successful nonstepping trials in the difficult magnitude. Additionally, one participant did not have nonstepping reactions to difficult perturbations and was excluded from paired comparisons of nonstepping versus planned stepping reactions in difficult perturbations.

Instructions to plan a stepping reaction resulted in a shift in weightbearing toward the stance leg (Fig. 2*A*). When instructed to plan stepping reactions, participants slightly loaded the stance leg by <10 N, or <1-kg force ($P = 0.024$).

Perturbation magnitude and instruction to step did not alter the latency to step (Fig. 2*B*). The latency of foot-off in planned stepping reactions did not differ between easy perturbations (600 ± 132 ms) and difficult perturbations (600 ± 122 ms, $P = 0.86$, $n = 19$). The latency to foot-off also did not differ between planned (632 ± 104 ms) and unplanned stepping reactions (662 ± 162 ms, $P = 0.33$, $n = 16$). The different number of participants and latencies for the planned stepping reactions between these two comparisons is due to three fast steppers (432 ± 49 ms in difficult perturbations) who never executed unplanned steps and could not be included in the corresponding paired comparison.

Step Execution

Whereas muscle responses differed with step execution in both easy and difficult perturbations, cortical responses increased with step execution only in difficult perturbations. In easy perturbations, the execution of a planned step was accompanied by a reduction in early and late stance-MG (Fig. 3*B*, *left*, $n = 19$, early, $P = <0.0001$; late; $P < 0.0001$), and increases in early and late swing-MG (early, $P = 0.012$; late, $P < 0.0001$), stance-TA (early, $P = 0.0071$; late, $P = 0.0003$), and swing-TA (early, $P = 0.017$; late, $P = 0.011$). In difficult perturbations, the execution of a planned step was accompanied with increases in early and late swing-MG (Fig. 3*B*, *right*, $n = 18$, early, $P = 0.0029$; late, $P = 0.0014$), as well as a reduction in baseline stance-TA ($P = 0.043$) and an increase in early stance-TA ($P = 0.017$). Cortical N1 responses increased by 6.3 μV with the execution of a compensatory step in difficult perturbations (Fig. 3*A*, *right*, $n = 18$, $P = 0.033$) but did not differ with the execution of a compensatory step in easy perturbations (Fig. 3*A*, *left*, $n = 19$; $P = 0.74$). Electrodermal response amplitudes did not differ with the execution of planned steps in easy (Fig. 3*C*, *left*, $n = 19$; $P = 0.058$) or difficult (Fig. 3*C*, *right*, $n = 18$; $P = 0.082$) perturbations. SC activity did not differ with the execution of planned stepping reactions in easy or difficult perturbations (Fig. 3*B*, $P > 0.05$).

Step Planning

Despite differences in muscle responses, cortical responses did not differ between planned and unplanned stepping reactions. Relative to planned steps, unplanned steps were associated with decreases in early stance-TA (Fig. 4*B*, $n = 16$; $P = 0.0093$) and late swing-MG ($P = 0.0046$), and increases in early and late stance-MG (early, $P = 0.046$; late, $P = 0.017$) and early and late SC activity (early, $P = 0.024$; late, $P = 0.0034$). In contrast, cortical N1 responses did not differ between planned and unplanned steps (Fig. 4*A*, $n = 16$, $P = 0.83$). Moreover, electrodermal response amplitudes did not differ between planned and unplanned steps (Fig. 4*C*, $n = 16$; $P = 0.92$).

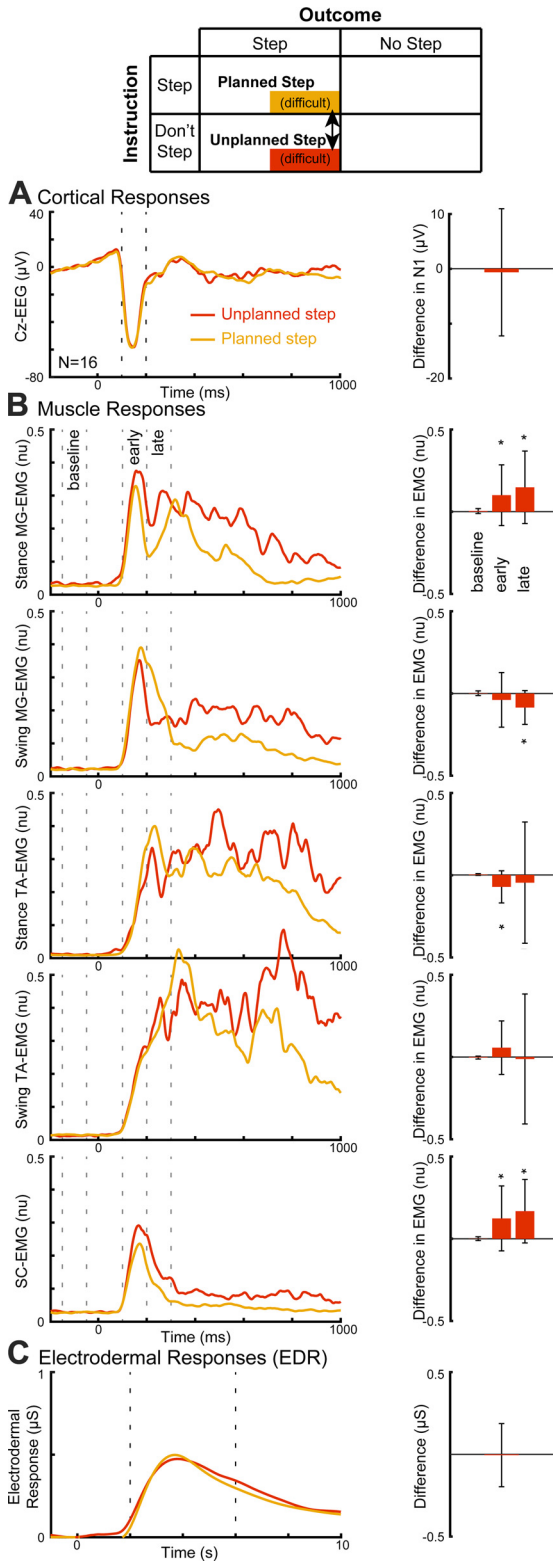
Interaction Between Stepping and Perturbation Magnitude

A generalized linear model in single-trial data across all three perturbation magnitudes further supports our primary analyses. N1 amplitude did not differ between planning conditions ($P = 0.73$). The effect of stepping on the N1 amplitude had a significant interaction with perturbation magnitude ($P = 0.023$). This interaction was characterized by greater increases in N1 amplitudes across perturbation magnitudes for stepping compared with nonstepping behaviors. Looking within each perturbation magnitude, there was a significant increase in N1 amplitude when stepping within difficult perturbations ($P = 0.010$), but not within moderate ($P = 0.69$) or easy ($P = 0.76$) perturbations.

Fig. 3. Changes in evoked responses with step execution between behaviors that are congruent with the task goal. *A*: group-averaged cortical responses are shown for nonstepping (black) and planned stepping reactions (yellow) in easy perturbations (*left*) and difficult perturbations (*right*). Vertical dashed bars indicate the time window of 100–200 ms. The bar plots show the mean and standard deviation of the difference in N1 response amplitude between the corresponding conditions across participants (planned step – no step). *B*: group-averaged electromyograph (EMG) responses are shown for each muscle for the same conditions shown in *A*. Vertical dashed bars indicate the baseline (−150 to −50 ms), early (100–200 ms), and late (200–300 ms) time windows. Bar plots show the mean and standard deviation of the difference in EMG activity between corresponding conditions in each time window across participants. *C*: group-averaged electrodermal responses are shown for the same conditions shown in *A*. Vertical dashed bars indicate the time window of 2–6 s. The bar plots show the mean and SD of the difference in electrodermal response amplitude between corresponding conditions. *Significant differences in paired *t* tests between response conditions within the corresponding time window, $\alpha = 0.05$ (i.e., any bar marked with an asterisk is significantly different from 0). MG, medial gastrocnemius; SC, sternocleidomastoid; TA, tibialis anterior.

Anticipatory Postural Adjustments Cannot Explain Changes in Evoked Response Variables

Anticipatory postural adjustments when planning a step were not associated with the changes in EMG activity, N1 amplitude, or EDR amplitude between planned stepping and successful feet in place responses at either perturbation magnitude or between planned and accidental steps in difficult perturbations (all $P > 0.05$). P values for all comparisons can be seen in Table 1.



N1 Response Amplitudes Were not Related to Subsequent Balance-Correcting Muscle Activity

Changes in cortical N1 response amplitudes when planning or executing steps were not associated with changes in subsequent balance-correcting EMG activity (all $P > 0.05$). P values for all comparisons can be seen in Table 2.

DISCUSSION

Our study suggests a possible role for the cortical N1 response in the execution—but not planning—of compensatory stepping reactions. Consistent with planning-related changes in central set, we observed differences in automatic muscle responses to perturbations when planning a step (Burleigh and Horak 1996; Burleigh et al. 1994; McIlroy and Maki 1993; Prochazka 1989) that could not be attributed to anticipatory postural adjustments preceding perturbations. These automatic responses are simultaneous to the cortical N1 response and before the execution of compensatory steps. However, we did not find an effect of prior planning on cortical N1 responses, rejecting our hypothesis that the cortical N1 response would be altered with prior planning. Instead, the cortical N1 response was larger in trials where compensatory steps were executed in response to difficult perturbations, with no effect of prior planning on the N1 amplitude. Prior work has demonstrated that the cortical N1 is influenced by factors preceding the N1 response, such as prior experience (Mierau et al. 2015; Payne et al. 2019; Quintern et al. 1985), predictability of perturbations (Adkin et al. 2006; Dietz et al. 1985a; Mochizuki et al. 2008, 2009b), attention to balance recovery (Little and Woollacott 2015; Quant et al. 2004b), and perceived threat (Adkin et al. 2008). We believe this is the first demonstration of a relationship between the cortical N1 and subsequent behavior. If the cortical N1 reflects a role of the cortex in compensatory balance-recovery behavior, it may reflect a role of the cortex in motor compensation, relevant to motor learning and rehabilitation.

We successfully replicated prior work showing planning-related changes in the evoked muscle responses, validating our ability to test whether this effect of planning extends to the evoked cortical responses. Our results are consistent with

Fig. 4. Changes in evoked responses with prior planning of executed steps. *A*: group-averaged cortical responses are shown for planned steps (yellow) and unplanned steps (red) in difficult perturbations. Vertical dashed bars indicate the time window of 100–200 ms. The bar plot shows the mean and SD of the difference in N1 response amplitude between conditions across participants (unplanned step – planned step). *B*: group-averaged EMG responses are shown for each muscle for the same conditions shown in *A*. Vertical dashed bars indicate the baseline (−150 to −50 ms), early (100–200 ms), and late (200–300 ms) time windows. Bar plots show the mean and SD of the difference in EMG activity between conditions in each time window across participants. *C*: group-averaged electrodermal responses are shown for the same conditions shown in *A*. Vertical dashed bars indicate the time window of 2–6 s. The bar plot shows the mean and standard deviation of the difference in electrodermal response amplitude between conditions. *Significant differences in paired t tests between response conditions within the corresponding time window, $\alpha = 0.05$ (i.e., any bar marked with an asterisk is significantly different from 0). MG, medial gastrocnemius; SC, sternocleidomastoid; TA, tibialis anterior.

Table 1. *Changes in evoked responses with planning and execution of compensatory steps were not associated with anticipatory postural adjustments between conditions across participants*

			Easy Pertubation Planned Steps Versus Nonstepping	Difficult Perturbation Planned Steps Versus Nonstepping	Difficult Perturbation Planned Steps Versus Accidental Steps
N1			0.81	0.55	0.96
MG	stance	early	0.79	0.87	0.7
		late	0.5	0.77	0.81
	swing	early	0.57	0.35	0.51
		late	0.34	0.17	0.84
TA	stance	early	0.19	0.89	0.26
		late	0.97	0.67	0.67
	swing	early	0.34	0.73	0.28
		late	0.69	0.7	0.92
SC		early	0.28	0.92	0.37
		late	0.18	0.9	0.068
EDR			0.84	0.84	0.076

Each row contains one evoked response variable, each column represents the two conditions in which each response variable is to be compared with anticipatory postural adjustments. Numerical values represent *P* -values for Spearman rank correlations testing for associations in between-condition changes between evoked response variables and anticipatory postural adjustments. EDR, electrodermal response; MG, medial gastrocnemius; SC, sternocleidomastoid; TA, tibialis anterior.

previous work demonstrating planning-related changes in the central sensitivity of the automatic brain stem-mediated motor responses to balance perturbations. When planning a step, early gastrocnemius muscle responses decreased in the stance leg and increased in the swing leg, consistent with changes in central set shown by Burleigh et al. (1994); however, unlike Burleigh and Horak (1996), this reduction did not require perturbation magnitude to be predictable. We also found planning-related changes in tibialis anterior muscle responses similar to those observed by McIlroy and Maki (1993), but in the opposite direction, due to the fact that we used backward perturbations, versus the forward perturbations in their study. Although it is theoretically possible that anticipatory changes in posture could have altered the sensory inputs that trigger the early muscle responses, rather than these changes being centrally mediated, none of the changes that we observed in the automatic motor responses were associated with anticipatory postural adjustments before perturbations. Taken together, these results demonstrate a change in the central nervous system due to motor planning, which affected the perturbation-evoked balance-correcting muscle activity.

Surprisingly, the cortical N1 response was neither influenced by prior planning, nor by errors in the motor outcome with respect to prior planning. In our prior work, we observed larger N1 responses in people who frequently took unplanned steps (Payne et al. 2019), which we surmised to be due to the unexpected error with respect to the goal of maintaining the feet in place. Whereas prior studies have demonstrated that the cortical N1 depends on the extent that the perturbation stimulus differs from expectations (Dietz et al. 1985a; Mochizuki et al. 2008, 2009b), we speculated that the cortical N1 magnitude might also depend on the extent that the motor response outcome differs from expectations. This possibility was also consistent with the comparison of the cortical N1 to the cognitive error-related negativity (Marlin et al. 2014; Payne et al. 2019b), which is enhanced by unexpected errors with respect to the intended motor goal. Therefore, we hypothesized that prior planning of a step would reduce the amplitude of the cortical N1 response by making step execution both expected and congruent with the prior motor goal. Instead, we observed a perturbation magnitude dependent increase in the cortical N1 response when executing steps versus feet-in-place responses, with no effect of prior planning on the N1 amplitude. Therefore, the increase in the cortical N1 in our experiment appears to be more related to the subsequent act of stepping rather than the prior planning of a step. Furthermore, the cortical N1 does not appear to be sensitive to unexpected errors with respect to the intended motor goal, in contrast to the error-related negativity (Payne et al. 2019b).

To our knowledge, this is the first evidence of a relationship between the cortical N1 and subsequent behavior, although this relationship may not be fully captured by the binary classification of stepping versus not stepping. The group-level increase that we observed in the cortical N1 when stepping was small and limited to the difficult perturbation magnitude. Although the small effect size could explain the lack of an effect of stepping in the easy perturbation, several participants described these steps as awkward or unnatural, suggesting that the

Table 2. *Changes in in cortical N1 response amplitudes with planning and execution of compensatory steps were not associated with changes in subsequent balance-correcting motor responses*

			Easy Perturbation Planned steps Versus nonstepping	Difficult Perturbation Planned Steps Versus Nonstepping	Difficult Perturbation Planned Steps Versus Accidental steps
MG	stance	late	0.42	0.93	0.20
	swing	late	0.77	0.62	0.078
TA	stance	late	0.73	0.47	0.58
	swing	late	0.19	0.89	0.16

Each row contains one evoked-response variable, and each column represents the two conditions in which each response variable is to be compared with cortical N1 responses. Numerical values represent *P* values for Spearman rank correlations testing for associations in between-condition changes between cortical N1 responses and subsequent balance-correcting motor responses. MG, medial gastrocnemius; TA, tibialis anterior.

unwarranted steps in the easy perturbation may not be comparable to the helpful compensatory steps in the difficult perturbation. In fact, stepping in the easy perturbation may be more comparable to a dual-task condition, which is known to reduce the N1 amplitude (Little and Woollacott 2015; Quant et al. 2004b), and, therefore, could have confounded stepping in easy perturbations. However, even in difficult perturbations, the changes in the N1 responses when stepping were highly variable across individuals, with some individuals showing smaller N1s when stepping. As the current study was specifically designed to test the effect of planning on the N1, our current data limit our ability to probe other factors that may better explain the increased N1 responses when stepping. For example, the N1 could be increased by greater attention to balance recovery (Little and Woollacott 2015; Quant et al. 2004b) if people tended to pay more attention to balance recovery when stepping, particularly in the difficult perturbations. Such changes in attention could serve to facilitate or coordinate, rather than strictly initiate or execute, compensatory behaviors such as stepping. The increase in N1 amplitudes when stepping in the difficult perturbations does not appear to be related to changes in startle reactions or autonomic arousal based on the lack of differences in the sternocleidomastoid responses or electrodermal responses between these conditions (Fig. 3). Although we recorded a minimal set of muscles necessary to replicate prior studies showing changes in central set, recording from more muscles would have enabled us to assess relationships between the cortical N1 and subsequent muscle activation more thoroughly. It is also possible that other muscles could have displayed stretch reflexes, which could have indicated changes in sensory gain that were not observed in the automatic balance-correcting motor response but could have impacted the cortical N1 response. Nevertheless, we believe this is the first evidence of a relationship between the cortical N1 amplitude and subsequent behavior.

A potential role of the cortical N1 in subsequent behavior could provide an explanation of the various factors preceding the N1 that influence its magnitude and could contribute to our understanding of motor compensation, learning, and rehabilitation. On the basis of the observation of larger N1s in young adults with lower balance ability, we recently proposed that the cortical N1 reflects the allocation of cortical resources, such as attention, to cortically mediated compensatory balance control to compensate for greater postural instability (Payne and Ting 2020). The potential role of N1 activity in subsequent behavior is further supported by the present finding of larger N1s when executing compensatory steps in difficult perturbations, which represents one of many potential ways such cortical control could be manifested. Although there is no direct evidence linking the cortical N1 to subsequent muscle activation, the N1 amplitude is reduced during dual-task performance (Little and Woollacott 2015; Quant et al. 2004b), which has separately been shown to reduce balance-correcting muscle activity at longer latencies than the N1 response (Rankin et al. 2000). Furthermore, dual-task conditions worsen balance-performance more in older adults with a history of falls (Shumway-Cook et al. 1997), who may rely more heavily on attention-dependent compensatory balance control (Woollacott and Shumway-Cook 2002). Additionally, the effect of perceived threat, which enhances the N1 amplitude in young adults when standing on an elevated surface (Adkin et al. 2008) could also be due to the reported shift of attention toward balance control when standing at the elevated surface heights (Huffman et al. 2009). Furthermore, the reduction in the N1 with prior experience (Mierau et al. 2015; Payne et al. 2019; Quintern et al. 1985) could be explained by a reduced reliance on attention-based compensatory balance control as people adapt their automatic balance-correcting motor responses with training (Welch and Ting 2014). Because the N1 is larger in individuals with worse balance (Payne and Ting 2020) and becomes smaller with training (Mierau et al. 2015; Payne et al. 2019; Quintern et al. 1985), it could be a useful biomarker for predicting and tracking whether balance control becomes less difficult, or more automatic, with rehabilitation (Petzinger et al. 2013). Finally, if the N1 plays a causal role in compensatory balance recovery behavior, it may be possible to target the neural substrates of the N1 with noninvasive stimulation to impact rehabilitation outcomes, which could be tested by future studies.

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DISCLOSURES

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

AUTHOR CONTRIBUTIONS

A.M.P. and L.H.T. conceived and designed research; A.M.P. performed experiments; A.M.P. analyzed data; A.M.P. and L.H.T. interpreted results of experiments; A.M.P. prepared figures; A.M.P. drafted manuscript; A.M.P. and L.H.T. edited and revised manuscript; A.M.P. and L.H.T. approved final version of manuscript.

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