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RESEARCH ARTICLE

Visual feedback is not necessary for recalibrating the vestibular contribution to the dynamic phase of a perturbation recovery response

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Abstract
Our recent work demonstrated that vision can recalibrate the vestibular signal used to re-establish equilibrium following a platform perturbation. Here, we investigate whether vision provided during a platform perturbation can recalibrate the use of vestibular reafference during the dynamic phase of the perturbation response. Dynamic postural responses were examined during a series of five forward perturbations to the body, while galvanic vestibular stimulation (GVS) selectively altered vestibular feedback and LCD occlusion spectacles controlled visual availability. Responses with and without vision were compared. The presence of GVS caused 1.78 ± 0.19 cm of medio-lateral (ML) body motion toward the anode during the initial 3 s of the dynamic postural response across perturbations. This dynamic ML response was attenuated across perturbations 1–3 independent of visual availability, resulting in a significant reduction of ML center of mass and pressure deviations ($p < 0.01$, $\eta^2 = 0.27$). That is, the vestibular influence on the ML perturbation response could be altered but vision was not necessary for this adaptation. After removing GVS, the ML response component reversed in direction toward the cathode with a magnitude that was not significantly different to the amount of response attenuation seen when GVS was present (-0.95 ± 0.19 cm; $p = 0.99$, $\eta^2 = 0.00$). This suggested that the use of a GVS-altered vestibular signal during dynamic perturbation responses could be recalibrated, but that visual feedback was likely not responsible. Alternative mechanisms to explain the recalibration process are discussed.

Keywords Vision · Vestibular · Dynamic · Recalibration · Somatosensory · Perturbation response

Introduction

The ability to maintain balance when standing and moving about can be largely attributed to the sensory feedback we receive from our visual, vestibular and somatosensory systems (Nashner 1972; Lacour et al. 1997; Berencsi et al. 2005). While each sensory system on its own provides useful information for maintaining balance, it is only when the brain integrates feedback from multiple sensory sources that we achieve a well-defined percept of our environment and where we are within it (Peterka 2002). The brain, however, cannot simply rely on integrating this feedback in the same fashion when sensory feedback from one modality is reduced, or altered, as an inappropriate bias might develop in the resulting percept (Ernst and di Luca 2011). One method by which the brain can cope with a reduction in the quality or quantity of the information from a sensory modality is by recalibration.

Previous work has shown that visual information can recalibrate the interpretation of a vestibular signal during a navigation task (Sturnieks et al. 2005). Given the importance of the vestibular system during the recovery from sudden perturbations to balance (Inglis et al. 1995; Horak and Hlavacka 2002) a common occurrence in daily living, we more recently investigated and demonstrated that visual feedback could recalibrate the use of vestibular reafference

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for re-establishing postural equilibrium during a perturbation recovery task (Toth et al. 2017). In that study, despite the fact that the prior visual–vestibular recalibration affected mediolateral (ML) equilibrium displacements of the center of mass (CoM) and center of pressure (CoP), the vestibular contribution to the dynamic phase (the 3 s window immediately following a platform perturbation) of the recovery response remained unaltered. One explanation for this is that vision was only provided during GVS when participants stood static prior to the perturbation rather than during the perturbation itself. In support of this explanation, Sturnieks et al.(2005) found that visual recalibration of vestibular feedback for navigation only manifested when vision was provided, while participants actively participated in a dynamic walking task compared to when they were passively transported in a wheelchair. Therefore, providing visual feedback during the dynamic phase of a perturbation response may be necessary for the body to recalibrate how it uses an altered vestibular signal during this period of perturbation recovery.

The purpose of the current study was to examine whether visual input provided during the dynamic phase of a platform perturbation response could recalibrate how vestibular feedback is used to control postural recovery during this same period. To do this, we followed previous work to isolate the contribution of vestibular feedback on the dynamic phase a perturbation recovery response (Inglis et al. 1995; Toth et al. 2017) where we paired anterior–posterior (AP) platform perturbations with mediolateral (ML) vestibular perturbations created by GVS. We first hypothesized that when vision was provided during platform perturbations, it would be used to reduce the GVS-induced ML CoM and CoP deviations seen during the dynamic phase of the recovery. An attenuation of ML CoM and CoP deviations independent of visual availability would suggest that other sources of sensory feedback may be responsible for adapting the vestibular contribution to the dynamic recovery response; whereas, a lack of reduction of the ML CoM and CoP deviations in the presence of vision would suggest that the dynamic phase of the response could not be adapted. Second, we hypothesized that after removing vision, the ML postural deviations during the dynamic recovery response would remain attenuated. This would provide evidence that visual feedback could recalibrate how vestibular feedback was used during the dynamic phase of the perturbation recovery response. If the response did not remain attenuated following the removal of vision, it would suggest that visual information was more heavily weighted for controlling dynamic recovery responses and was not responsible for recalibrating vestibular feedback. Finally, we hypothesized that following the visual recalibration of the GVS-altered vestibular signal and well after the removal of GVS and restoration of baseline vestibular feedback, postural deviations of the ML CoM and CoP would be reversed (become cathode directed) during the dynamic recovery phase. This would further support the role of vision in recalibrating how vestibular feedback is used to control the dynamic phase of perturbation recovery. Responses that attenuated across multiple perturbations in the absence of vision and reversed following GVS removal would suggest that vision were not necessary and alternative sensory sources might be responsible for recalibrating the use of vestibular feedback to control the dynamic phase of perturbation recovery responses.

Methods

Nine male and three female participants (age 24.50 ± 4.40 years; height 1.77 ± 0.07 m; mass 71.10 ± 12.75 kg; mean $\pm$ SD) with no history of neuromuscular disorders provided written informed consent prior to participating in the study. The experimental protocol was approved by the University of Guelph Research Ethics board, which conforms to the standards set by the Declaration of Helsinki.

Equipment and setup

Participants stood (inter-metatarsal distance 4 cm) on a force plate (model 9281B, Kistler Instruments AG, Winterthur, Switzerland) made level with the surface of a hexapod motion platform (Mikrolar Inc., NH, USA). A safety harness with enough slack to allow movement about the platform (platform diameter: 2.1 m) yet enough to prevent a fall (no falls were recorded) was worn by each participant. To control visual availability, participants wore PLATO™ LCD spectacles (Translucent Technologies, Toronto, Canada) that contained lenses that could be remotely controlled to become opaque during data collection. Participants wore fitted clothing to minimize the movement of passive kinematic markers that were placed on the following anatomical locations: left and right temporal-mandibular joints (TMJs), left and right acromia, left and right anterior–superior iliac spines (ASISs), and left and right first metatarsals. One additional marker was placed on the motion platform. A 12-camera Optitrak system was used to collect 3D kinematic data (100 Hz).

Vestibular perturbations

To perturb the vestibular system, bipolar binaural GVS (Linear Stimulus Isolator A395, World Precision Instruments Inc., FL, USA) was applied via two Ag/AgCl electrodes placed bilaterally on the skin overlying each participant's mastoid processes. Electrode gel was applied to the skin behind the ears to minimize impedance and lower the voltage necessary to maintain a constant current. In an attempt to make the voltage associated with the vestibular stimulus

less perceptually noticeable, we applied a 4 × threshold constant stimulus (for thresholds below 0.25 mA, a minimum 1-mA testing current was applied) with a stimulus rise time of 4 s (threshold: smallest amount of current required to elicit a noticeable postural response; range 0.20–0.80 mA; Bent et al. 2000). A rise time of 4 s was chosen based on pilot work and on the recognition that an immediate transition from zero to maximal stimulation is potentially un-physiological and poorly tolerated by some individuals (Rosengren 2002). Stimulation was either anode-L/cathode-R or anode-R/cathode-L randomized across trials.

Platform perturbations

To evoke anterior postural perturbations, the mechanical platform upon which participants stood was translated 9 cm backwards with a trapezoidal velocity profile (peak velocity 14.0 cm/s) and a peak acceleration of approximately 1.8 m/s^2. After each perturbation, the platform was held in place for 6 s before it was slowly moved back to its starting position at a peak velocity and acceleration of approximately 4.0 cm/s and 0.5 m/s^2, respectively, where it was held a further 3 s before the next perturbation. Conditions with left, right, and posterior postural perturbations in the absence of GVS were included in an attempt to keep testing conditions unpredictable.

Protocol

Prior to starting the experimental protocol, participants were exposed to the testing level of GVS as well as platform perturbations in each direction to prevent first-trial effects (Keshner et al. 1987; Allum et al. 2002). The experimental trials were conducted in two separate blocks. In the first block of trials (NoVISION condition), participants were instructed to maintain a relaxed upright standing posture and to do their best to recover their posture in response to the platform perturbations without waving their arms and while keeping their feet in place. In the second block of trials (VISION condition), participants were additionally instructed to use vision, when available, to aid the maintenance and realignment of their vertical posture. Participants were first exposed to all trials in the first block (NoVISION). After a short break, trials of the second (VISION) block were conducted. This specific ordering ensured that instructions pertaining to trials of the VISION condition did not confound responses during trials in the NoVISION condition.

In total, 35 trials were collected per participant; 20 GVS trials (five anode right and five anode left trials for both NoVISION and VISION blocks), ten NoGVS trials (five NoVISION and five VISION) and five catch trials that included at least one forward, one leftward and one rightward platform perturbation.

VISION block

During all trials in the VISION block, participants began by standing quietly on the force plate for 3 s prior to visual occlusion. Three seconds after visual occlusion, participants either received anode left GVS, anode right GVS, or no GVS. GVS type was randomized across trials. Eight seconds after GVS was turned on (4 s ramp and 4 s constant current), visual feedback was restored to normalize each participant's starting posture prior the platform perturbations. Three seconds later, participants experienced the first of a series of four posterior platform perturbations (P1–P4). Perturbations P1–P3 were administered while vision was available; whereas, vision was occluded 3 s prior to P4.

Three seconds following the recovery of the platform from the 4th perturbation (P4), the GVS current was ramped off over 4 s (or in the case of the NoGVS condition, participants continued to stand quietly). Four seconds following the complete removal of the GVS, vision was restored for 4 s. Three seconds after vision was re-occluded, participants were exposed to a 5th and final platform perturbation (P5) (Fig. 1). The adaptation times following a change in visual availability were chosen based on previous pilot work in our lab and this amount of time was sufficient for recalibrating equilibrium responses following recovery to a platform perturbation (Toth et al. 2017).

NoVISION block

As was the case in the VISION block, participants were randomly exposed to trials involving anode left GVS, anode right GVS or no GVS. GVS and platform perturbation magnitudes and timings were identical to those in the VISION block. However, unlike the VISION block, vision was occluded during all perturbations (Fig. 1).

Data processing

Data processing was performed using Visual 3D software (c-motion Inc. MD, USA). Kinematic data from each marker were interpolated using a second-order polynomial across a maximum gap of three frames (30 ms) and then filtered using a second-order dual low-pass Butterworth filter with a cut-off frequency of 5 Hz. Right and left metatarsal marker data were each subtracted from the platform marker motion. If the calculated difference was greater than 1 cm for any perturbation it was assumed that a step had occurred and the trial was removed from further analyses (this process resulted in the removal of less than 15% of all trials). A visual 3D reduced whole-body Center of Mass (CoM) model,

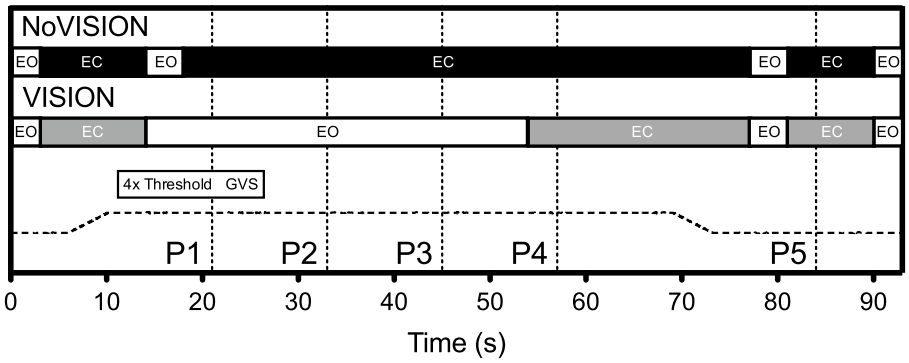

Fig. 1 Experimental protocol for both NoVISION and VISION conditions. Black 'EC' ('eyes closed') and white 'EO' ('eyes open') bars illustrate periods of time when vision was occluded and available respectively in the NoVISION condition. Gray 'EC' ('eyes closed') and white 'EO' ('eyes open') bars illustrate periods of time when vision was occluded and available respectively in the VISION condition. The horizontal dotted line illustrates the timing and magnitude of the GVS current used and the vertical dotted lines indicate the onset of each platform perturbation (P1–P5) in both the NoVISION and VISION conditions

composed of head (TMJ and acromia markers) and trunk (acromia and ASIS markers) segments, was constructed and used to estimate whole-body postural sway in the mediolateral (ML) and Anterior–Posterior (AP) directions (Toth et al. 2017). Platform marker data were subtracted from the CoM data to reference body motion relative to the support surface (platform). Force and moment data collected from the force plate were filtered in the same manner as kinematic data. The filtered force and moment data were then used to calculate AP and ML CoP displacements for each trial.

AP and ML displacements of both the CoM and CoP were referenced to their average positions during the 500-ms quiet stance period immediately prior to the onset of GVS. ML CoM and CoP traces for NoVISION and VISION conditions were calculated using the average of the anode right data with the inverse of the anode left data [(Anode-R) + (-Anode-L)]/2. Averaging the ML data in this way removed any spurious effects due solely to the backward platform perturbation (common to anode L and R trials) and allowed us to examine vestibular influences on lateral sway during each condition. This technique has been previously implemented by others for the same purpose (Inglis et al. 1995).

Dependent variables

To quantify responses to the onset and offset of GVS in the NoVISION and VISION conditions, the average ML CoM and CoP positions over 50 ms following the 4 s ramp phase of GVS onset (ON) and offset (OFF) were calculated relative to the average position (50 ms) immediately prior to GVS onset and offset, respectively.

Examination of the CoP responses revealed that they were biphasic containing (1) the traditional dynamic perturbation responses (R1, R2, R3, R4 and R5) (Toth et al. 2017), and (2) an earlier smaller and oppositely directed ML component within the first 600 ms after perturbation onset. A similar perturbation response has been described previously in the EMG signal of postural muscles (Horak and Hlavacka 2002). This early ML CoP response has also been described previously in response to the onset and offset of GVS (Smetanin and Popov 1990) but not in response to a platform perturbation while GVS was already ongoing. Therefore, in the current work, we distinguish an early component of the dynamic perturbation responses specifically for biphasic ML CoP perturbation responses and in addition to the R1–R5 responses. For the biphasic ML CoP perturbation response, the early responses (*e*Rs) were defined as the peak displacement within the 600-ms window following perturbation onset, whereas Rs were defined as the peak displacement from 600 ms to 3 s following perturbation onset. Both peaks were calculated relative to the average position prior to each perturbation (defined as the average position in the 500 ms window immediately prior to perturbation onset). For ML CoM, AP CoM and AP CoP dynamic responses, R magnitudes were defined as the peak displacement within a 3 s window after perturbation onset relative to the average position prior to each perturbation.

If the dynamic response to the fifth perturbation (R5) (after removing GVS) was reversed compared to the four prior dynamic responses during GVS and were to have a magnitude similar to the response reduction between P1 and P4 (R1–R4) in the VISION condition, then this would show that vision could recalibrate how vestibular feedback is used to control the dynamic phase of a perturbation recovery response. To establish the magnitude of the ML response reduction between P1 and P4 in both the NoVISION and VISION conditions, we subtracted the NoVISION R1 from each condition's R4 (see Eqs. 1 and 2) and compared the resulting difference to the magnitude of R5 (Fig. 3).

$$\text{NoVISION (R4 – R1)} = \text{NoVISION R4} - \text{NoVISION R1} \quad (1)$$

$$\text{VISION (R4 – R1)} = \text{VISION R4} - \text{NoVISION R1} \quad (2)$$

It should be noted that the R1 in the NoVISION condition was used to calculate R4−R1 for both the NoVISION and VISION conditions because, unlike the R1 in the VISION condition, the NoVISION R1 could not have been altered by visual feedback. As a result, the R4−R1 difference for the VISION condition better reflects the capacity of visual feedback to modulate the R's across perturbations P1–P4. Additionally, the difference in early response between perturbations 1 and 4 (*e*R4—*e*R1) was similarly calculated specifically for ML CoP and compared to *e*R5 for both NoVISION and VISION conditions. This again was to determine whether the magnitude of the early component of the biphasic dynamic CoP response following GVS removal could also be explained by a prior recalibration of the GVS-altered vestibular signal.

Statistics

Statistical analyses were conducted using Prism GraphPad software (version 5.0, GraphPad Software, Inc. CA, USA) and statistical tests were performed on AP and ML CoM and CoP data unless otherwise stated. All data were expressed as means ± 95% Confidence Interval (CI) and significance was set at $p \leq 0.05$. Data normality was checked using Shapiro–Wilk and histogram analyses.

Paired *t* tests were used to determine whether ON and OFF responses were different between NoVISION and VISION conditions for AP and ML CoM and CoP data. This was done to establish whether perturbation responses between the visual conditions might be explained by a difference in the level of GVS experienced by participants. AP CoM ON responses in the NoVISION and VISION conditions, which violated normality assumptions, were compared using a Wilcoxon matched pairs signed rank test.

To examine the influence of visual availability on AP perturbation responses, a two-way ANOVA (VISION × PERTURBATION) was used to test for differences between Rs for the NoVISION and VISION conditions; Sidak multiple comparison corrections (Abdi 2006) were made for AP CoM and CoP data. Two-way ANOVAs (VISION × PERTURBATION) were also conducted to examine whether ML CoM and CoP R magnitudes differed between VISION and NoVISION conditions or between the multiple platform perturbations. Finally, two-way ANOVAs (VISION × PERTURBATION) were also used to compare ML CoP *e*R magnitudes between VISION and NoVISION conditions, and between perturbations. ANOVAs were conducted on ML CoM and CoP data with a priori comparisons between Rs and *e*Rs to perturbations P1 and P2, P1 and P3, and P1 and P4 for both the NoVISION and VISION conditions.

To compare responses to the fifth perturbation (following the removal of GVS) with the prior response change across perturbations P1 and P4 during GVS, paired *t* tests were used to test for differences between R4−R1 and R5 as well as between *e*R4 − *e*R1 and *e*R5 within both NoVISION and VISION conditions.

Results

Mediolateral center of mass and center of pressure displacements

Participants demonstrated a consistent CoM (Fig. 2b; left) and CoP postural shift (1.2 cm and 1.5 cm, respectively) toward the anode electrode in response to GVS onset (ON) that did not differ between the visual conditions (CoM $p = 0.18$, $\eta^2 = 0.02$ CoP $p = 0.33$, $\eta^2 = 0.01$). When GVS was turned off, large CoM (Fig. 2b; right) and CoP (data not shown) postural deviations of 1.6 cm and 2.1 cm, respectively, were observed in the direction of the cathode. OFF responses were not significantly different between NoVISION and VISION conditions.

CoM (Fig. 3a) and CoP (not shown) responses (Rs) were shifted towards the anode for perturbations P1–P4 for both NoVISION and VISION conditions. In contrast, R5, which occurred 9 s after GVS had been turned off, deviated in the direction of the cathode (Fig. 3a). A two-way ANOVA revealed a significant interaction between visual condition and perturbation for CoM ($F_{(5,18)} = 6.89$, $p = 0.05$) and CoP ($F_{(5,18)} = 6.79$, $p = 0.04$) responses. Simple effects revealed that within the NoVISION condition, R1 was significantly larger than R2 (CoM $p < 0.01$, $\eta^2 = 0.10$; CoP $p < 0.01$, $\eta^2 = 0.18$), R3 (CoM $p < 0.01$, $\eta^2 = 0.26$; CoP $p < 0.01$, $\eta^2 = 0.33$) and R4 (CoM $p = 0.01$, $\eta^2 = 0.19$; CoP $p < 0.01$, $\eta^2 = 0.27$). Likewise in the VISION condition, CoM R1 was larger than R2 ($p = 0.04$, $\eta^2 = 0.07$) (Fig. 3b) and CoP R1 was larger than both R2 ($p = 0.04$, $\eta^2 = 0.09$) and R3 ($p = 0.05$, $\eta^2 = 0.05$). However, R1 was not significantly different from R4 (CoM $p = 0.46$, $\eta^2 = 0.00$; CoP $p = 0.42$, $\eta^2 = 0.00$).

When comparing perturbation responses between vision conditions, R1 and R2 were significantly larger in the NoVISION compared to the VISION condition (R1: CoM $p = 0.04$, $\eta^2 = 0.09$; CoP $p = 0.03$, $\eta^2 = 0.14$; R2: CoM $p = 0.05$, $\eta^2 = 0.08$; CoP $p = 0.02$, $\eta^2 = 0.15$) (Fig. 3b left; CoP R data not shown). Following the removal of GVS, R5s were not different between NoVISION and VISION conditions (CoM $p = 0.62$, $\eta^2 = 0.01$; CoP $p = 0.42$, $\eta^2 = 0.00$).

Importantly, for determining whether vision is necessary for recalibration, the response decline in the VISION

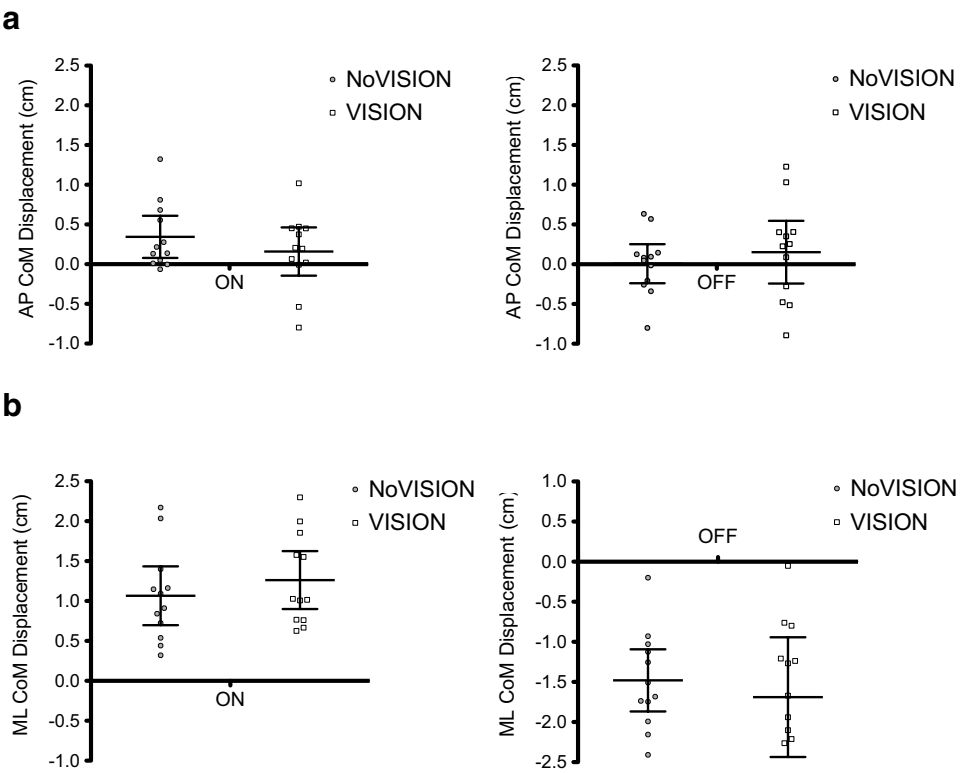

Fig. 2 Average (black line ± 95% CIs) and individual AP (**a**) and ML (**b**) CoM displacements (cm) in response to the onset (ON; left) and offset (OFF; right) of GVS in both the NoVISION (gray circles) and VISION (white squares) conditions. No significant differences were found between ON or OFF responses in both VISION and NoVISION conditions

and NoVISION conditions was compared. There was no difference between the progressive decline in the response magnitude from P1 to P4, while GVS was on (R4–R1) and the response after GVS was switched off (R5) within either the NoVISION (CoM $p = 0.98$, $\eta^2 = 0.00$; CoP $p = 0.99$, $\eta^2 = 0.00$) or VISION (CoM $p = 0.64$, $\eta^2 = 0.01$; CoP $p = 0.63$, $\eta^2 = 0.01$) conditions (Fig. 3b; right). These data suggest that the modification of the postural response seen at P5 can occur without vision.

The early component (*e*R; 0–600 ms) of the biphasic ML CoP perturbation responses was directed toward the cathode for perturbations P1–P4, and reversed in direction in response to the fifth perturbation during both NoVISION and VISION conditions (Fig. 4a). A 2-way ANOVA revealed a main effect for perturbation ($F_{(3,20)} = 3.90$, $p = 0.02$). There was no difference between early responses in both the NoVISION and VISION conditions for any perturbation (*e*R1 $p = 0.34$, $\eta^2 = 0.02$; *e*R2 $p = 0.24$, $\eta^2 = 0.03$; *e*R3 $p = 0.98$, $\eta^2 = 0.00$; *e*R4 $p = 0.34$, $\eta^2 = 0.02$; *e*R5 $p = 0.24$ $\eta^2 = 0.03$).

In the NoVISION condition, *e*R1 was significantly larger than *e*R3 ($p = 0.02$, $\eta^2 = 0.17$) and *e*R4 ($p = 0.04$, $\eta^2 = 0.06$) (Fig. 4b; left). In the VISION condition, *e*R1 was significantly larger than *e*R4 ($p = 0.05$, $\eta^2 = 0.08$) (Fig. 4b; left). The change in early CoP response magnitudes across P1–P4 with GVS ongoing (*e*R4–*e*R1) was not different from the response after GVS was switched off (*e*R5) for either the NoVISION ($p = 0.20$, $\eta^2 = 0.03$) or VISION ($p = 0.71$, $\eta^2 = 0.01$) conditions (Fig. 4b; right). ML CoM and CoP response magnitudes and the response difference between P1 and P4 during NoVISION and VISION conditions are shown in Table 1.

Anteroposterior center of mass and center of pressure displacements

In contrast to the ML results, there were limited systematic changes observed in the anterior–posterior (AP) responses. Firstly, CoM and CoP positions did not deviate with GVS onset (ON) (Fig. 2a; left) or termination (OFF) (Fig. 2a; right) and were not different between NoVISION and VISION conditions (ON: CoM $p = 0.15$, $\eta^2 = 0.02$; CoP $p = 0.19$, $\eta^2 = 0.03$) (OFF: CoM $p = 0.3$, $\eta^2 = 0.01$; CoP $p = 0.32$, $\eta^2 = 0.01$).

A two-way ANOVA revealed an interaction between the visual condition and the perturbation number for both CoM and CoP responses (CoM; $F_{(4,19)} = 1.38$, $p = 0.04$) (CoP; $F_{(4,19)} = 8.11$, $p < 0.01$). Post hoc analyses revealed that the availability of visual feedback did not consistently modulate the pattern of postural responses. For CoP data, a difference between visual conditions was observed only for R1 ($p < 0.01$, $\eta^2 = 0.28$), R2 ($p < 0.01$, $\eta^2 = 0.19$) and R5 ($p = 0.04$, $\eta^2 = 0.11$) (Fig. 5). For CoM data, R2 ($p = 0.02$, $\eta^2 = 0.13$) and R3 ($p < 0.01$, $\eta^2 = 0.22$) differed between visual conditions. In the NoVISION condition, the first CoP response was larger than all others ($p < 0.01$). Despite these CoP changes, NoVISION CoM responses did not differ

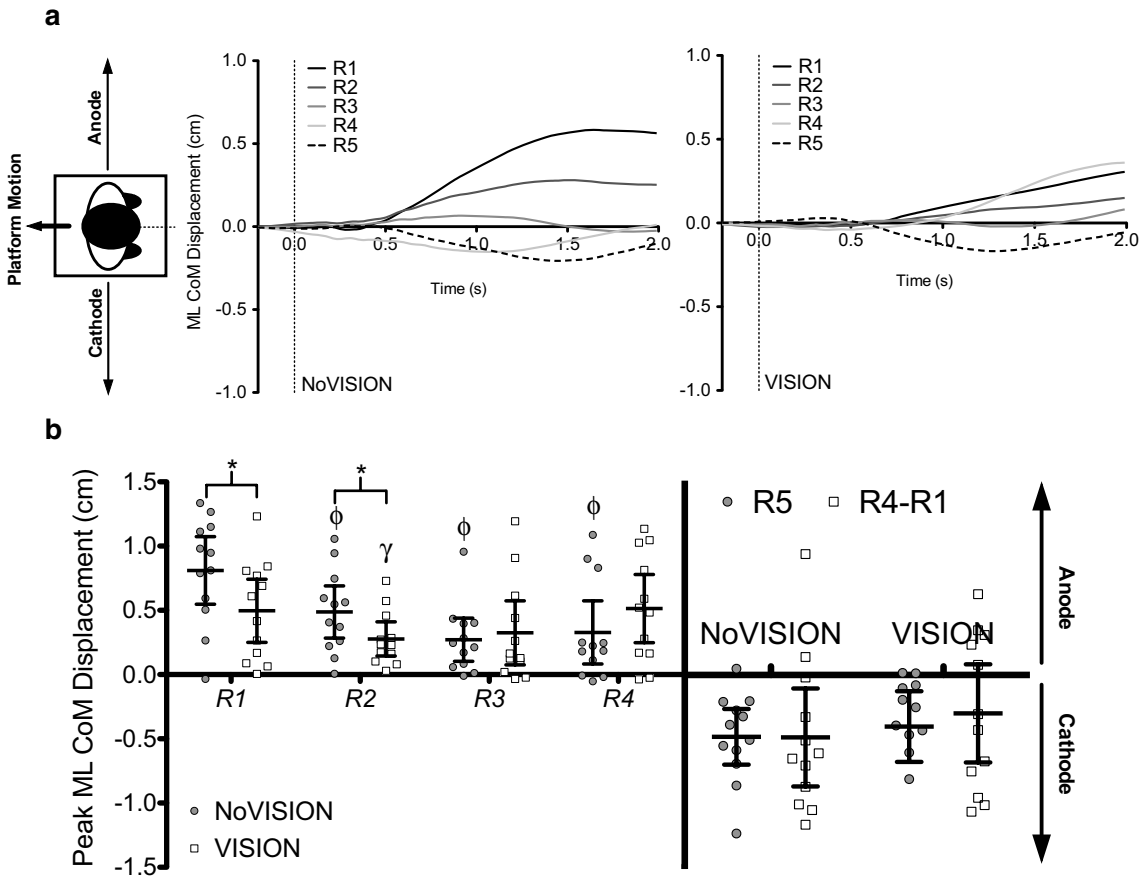

Fig. 3 **a** Average ML CoM responses of all participants to platform perturbations (P1–P5) for both NoVISION (left) and VISION (right) conditions. Traces are aligned to the onset of platform perturbation (vertical dotted line) and expressed relative to the ML position immediately before the perturbation. A superior view of a participant standing on the platform is provided to illustrate lateral motion toward the anode (+) and cathode (-) during posterior platform motions. **b** (left): Average (black line ± 95% CIs) and individual peak ML CoM responses to perturbations P1–P4 for both NoVISION (gray circles) and VISION (white squares). **b** (right): Average (black line ± 95% CIs) and individual peak ML CoP R5 (gray circles) for NoVISION and VISION conditions as well as the predicted R5, measured as the difference in magnitude between R1 and R4 (white squares). Asterisk denotes a significant difference between NoVISION and VISION conditions for a given perturbation. ϕ denotes a significant difference from NoVISION R1 and γ denotes a significant difference from VISION R1

from each other ($p > 0.13$). In the VISION condition, while CoP responses did not differ ($p > 0.17$), the CoM response to the first perturbation (R1) was significantly smaller than R2 ($p < 0.01$, $\eta^2 = 0.18$) and R3 ($p < 0.01$, $\eta^2 = 0.20$) (see Table 2).

Discussion

The current study aimed to determine whether visual feedback provided during a platform perturbation could be used to recalibrate how altered vestibular reafference (from GVS) was interpreted during the dynamic phase of a perturbation response. Contrary to our hypothesis, we found that vision was not necessary for recalibrating the vestibular feedback used during initial perturbation recovery responses. Firstly, although participants attenuated their GVS-biased dynamic mediolateral response magnitudes over the course of multiple platform perturbations, they were able to do so regardless of visual availability. However, vision did appear to facilitate the initial adaptation process, as responses to the first two perturbations were smaller in the presence of vision (Figs. 3 and 4). Interestingly, well after removing GVS in both the NoVISION and VISION conditions, the dynamic response to the final perturbation (R5 and eR5) was reversed relative to the responses elicited during the first four perturbations when GVS was ongoing (P1–P4). Moreover, the magnitude of this response reversal was not different from the reduction of GVS bias on the dynamic response

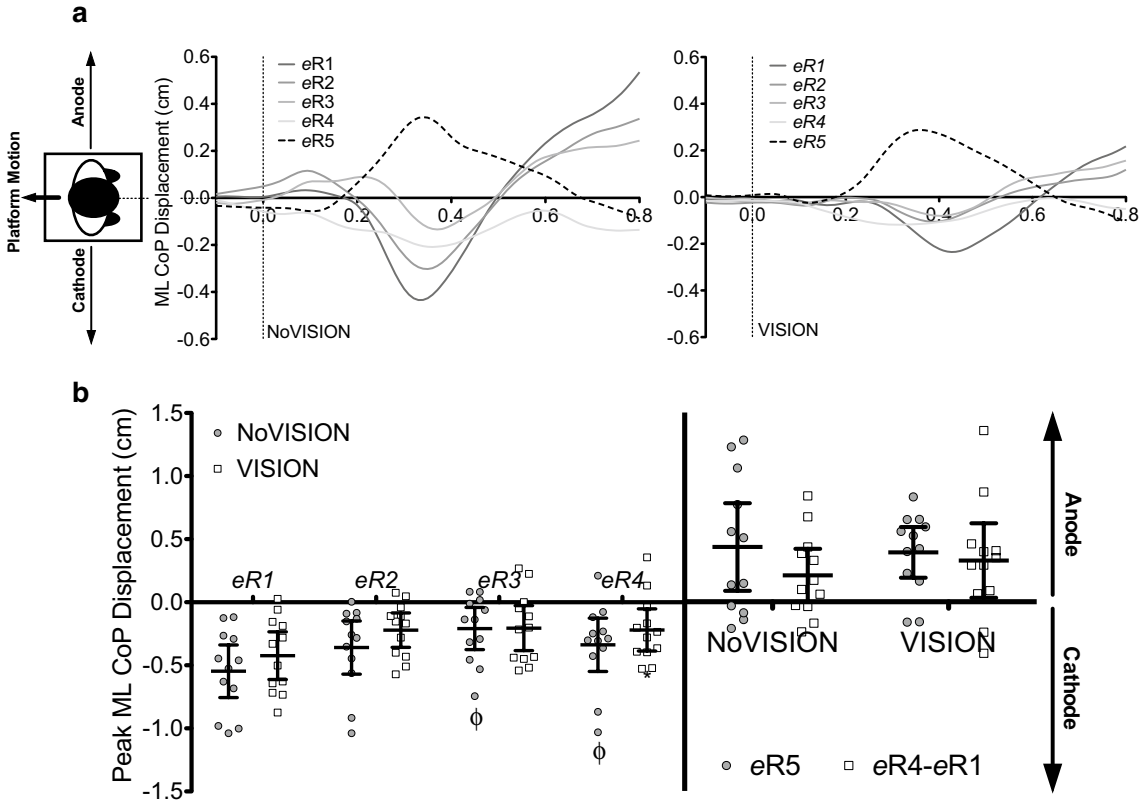

Fig. 4 **a** Average ML CoP early responses (eR) of all participants for both NoVISION (left) and VISION (right) conditions. Traces are aligned to the onset of each platform perturbation and expressed relative to the ML position immediately before the perturbation. A superior view of a participant standing on the platform is provided to illustrate lateral motion toward the anode (+) and cathode (−) during posterior platform motions. **b** (left): Average (black line ± 95% CIs) and individual peak ML CoP eR's to perturbations P1–P4 for both NoVISION (gray circles) and VISION (white squares). **b** (right): Average (black line ± 95% CIs) and individual peak ML CoP eR5's (gray circles) for NoVISION and VISION conditions as well as the predicted eR5, measured by the response reduction between eR1 and eR4 (white squares). ϕ and asterisk denote a significant difference from NoVISION eR1 and VISION eR1 respectively

Table 1 ML CoM and CoP perturbation responses (R's), CoP early responses (*e*R's) and the decline between the first and fourth perturbations for each variable are given in cm for the NoVISION and VISION conditions

CoM			CoP					
Response	NoVISION	VISION	Response	NoVISION	VISION	Response	NoVISION	VISION
R1	0.81 ± 0.24	0.50 ± 0.22	R1	1.78 ± 0.37	1.12 ± 0.40	*e*R1	−0.55 ± 0.20	−0.42 ± 0.18
R2	0.49 ± 0.20	0.28 ± 0.12	R2	1.10 ± 0.26	0.65 ± 0.22	*e*R2	−0.36 ± 0.20	−0.22 ± 0.12
R3	0.27 ± 0.16	0.32 ± 0.22	R3	0.73 ± 0.28	0.71 ± 0.40	*e*R3	−0.21 ± 0.16	−0.21 ± 0.16
R4	0.33 ± 0.22	0.51 ± 0.24	R4	0.83 ± 0.33	1.05 ± 0.40	*e*R4	−0.34 ± 0.20	−0.22 ± 0.16
R5	−0.48 ± 0.20	−0.40 ± 0.24	R5	−0.95 ± 0.37	−0.91 ± 0.51	*e*R5	0.43 ± 0.32	0.39 ± 0.18
R4-R1	−0.48 ± 0.33	−0.30 ± 0.33	R4–R1	−0.94 ± 0.50	−0.72 ± 0.55	*e*R4–*e*R1	0.21 ± 0.20	0.33 ± 0.26

Values are expressed as mean ± 95% CIs

between perturbations P1 and P4. It is the reversal of the response, well after the restoration of baseline vestibular feedback, which provides evidence that the initial response adaptation occurring while GVS was ongoing was actually a recalibration of how the brain was using the altered vestibular signal to organize an appropriate postural response as opposed to simply a down-weighting of erroneous vestibular information. However, the fact that this occurred regardless of visual availability raises the question, if not vision, what is the brain using to calibrate the GVS-biased feedback?

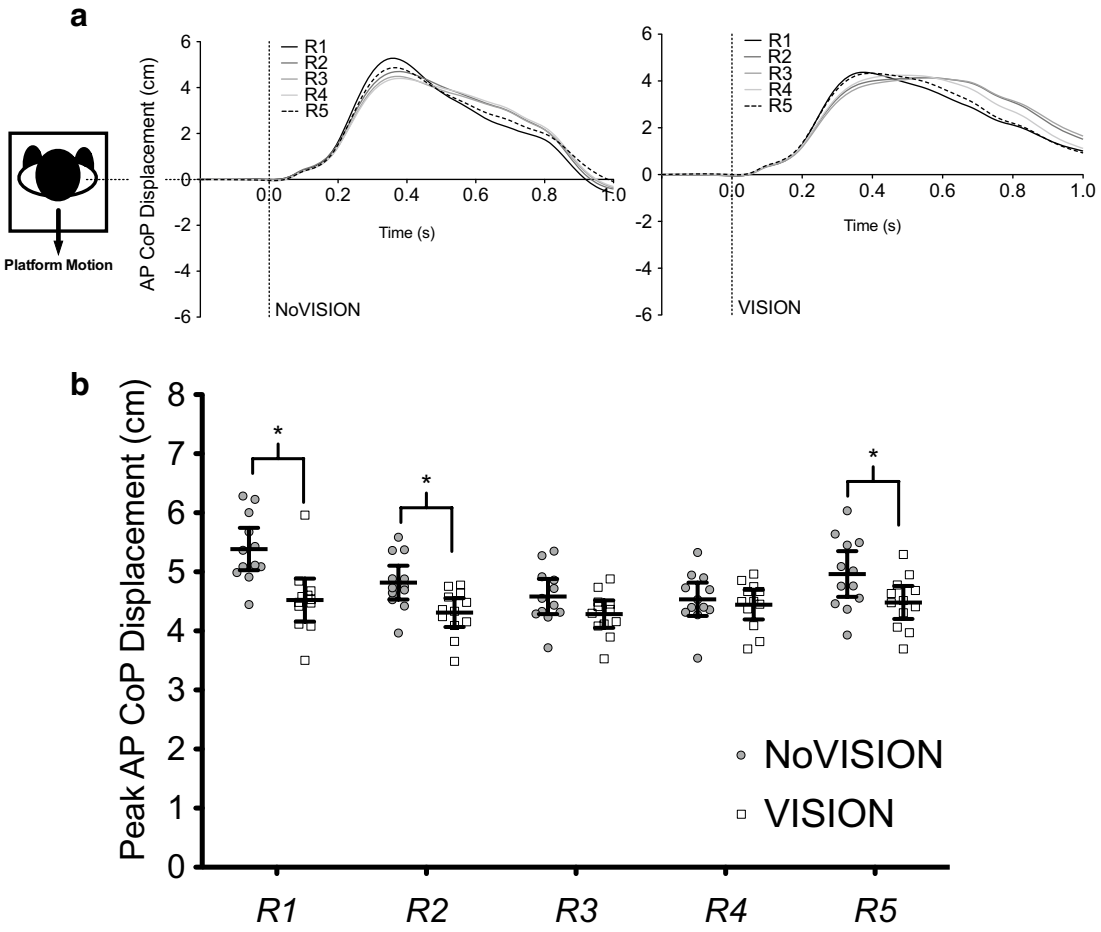

Fig. 5 **a** Average AP CoP displacements of all participants in response to platform perturbations P1–P5 for both NoVISION (left) and VISION (right) conditions. Traces are aligned to the onset of each platform perturbation (Time = 0 s) and expressed relative to the AP position immediately before the perturbation. A superior view of a participant standing on the platform is provided to illustrate anterior (+) and posterior (−) trace magnitudes in response to the posterior platform motion. **b** Average (black line ± 95% CIs) and individual peak AP CoP response magnitudes (R1–R5) in response to platform perturbations (P1–P5) for both NoVISION (gray circles) and VISION (white squares) conditions. Asterisk denotes a significant difference between responses in the NoVISION and VISION conditions during a given perturbation

Table 2 AP CoP and CoM peak perturbation response magnitudes (mean ± 95% CIs) for NoVISION and VISION conditions

CoP	NoVISION		VISION		CoM	NoVISION		VISION	
R1	5.38 ± 0.32		4.52 ± 0.33		R1	5.93 ± 0.48		6.42 ± 0.37	
R2	4.82 ± 0.26	*	4.31 ± 0.22	NS	R2	6.38 ± 0.53	NS	7.17 ± 0.35	*
R3	4.58 ± 0.26	*	4.28 ± 0.20	NS	R3	6.29 ± 0.44	NS	7.19 ± 0.30	*
R4	4.53 ± 0.26	*	4.44 ± 0.22	NS	R4	6.36 ± 0.33	NS	6.87 ± 0.46	NS
R5	4.96 ± 0.35	*	4.48 ± 0.26	NS	R5	6.12 ± 0.46	NS	6.51 ± 0.48	NS

* and NS represent significant and non-significant differences, respectively, relative to dR1 for the NoVISION and VISION conditions. All values are in cm

Vestibular recalibration by vision

The magnitude of the ML center of mass (CoM) and center of pressure (CoP) dynamic responses (R and *e*R) were reduced over perturbations P1–P4 in the NoVISION condition (Figs. 3 and 4). In the VISION condition, participants were able to use the available visual feedback provided to significantly attenuate further their ML R1 and R2 magnitudes, compared to NoVISION. However, the reduction of their R3 magnitudes did not differ based on visual

availability. This suggested that vision was able to facilitate the adaptation of the dynamic ML response but that an alternate mechanism was effective by P3.

Sturnieks et al. (2005) demonstrated that when vision was used to recalibrate the vestibular reafference indicating 'straight ahead', the newly adapted behavior was maintained even after removing vision. This led us to similarly hypothesize that vision would be used to recalibrate the vestibular reafference responsible for organizing appropriate perturbation responses if, in the VISION condition, the newly adapted ML response observed across perturbations 1–3 was maintained in R4 following the removal of vision. Overall, we found that R4 was attenuated in both the NoVISION and VISION conditions relative to the NoVISION R1. This suggests that vision is not necessary and potentially not used at all to recalibrate the influence of vestibular feedback on the dynamic phase of the perturbation response. Moreover, vision is not necessary for the adaptation (as distinct from recalibration) of the response across perturbations 1–3 as this adaptation occurs without any visual availability. Instead, vision appears to assist the adaptation of ML body motion during the dynamic phase of perturbation recovery in the presence of GVS. While this corroborates previous work, which demonstrates the involvement of vision for reducing vestibular responses during navigation (Carlsen et al. 2005) and postural control (Day and Cole 2002; Day and Guerraz 2007), we show that with only two additional perturbation exposures, alternative mechanisms are appropriate for adapting dynamic perturbation recovery responses in the presence of altered vestibular feedback.

Is habituation responsible?

If vision were solely responsible for recalibrating the vestibular reafference used during the dynamic phase of perturbation recovery responses, then occluding vision during all of the perturbations in the NoVISION condition would hinder participants' ability to attenuate the ML component of their dynamic recovery response across the multiple perturbations. In contrast to this hypothesis, we observed a significant attenuation of the dynamic ML response across perturbations P1–P4 in both the VISION and NoVISION conditions with no significant effect of visual availability on the magnitude of this attenuation by P4. One possible explanation is that participants were habituating to the vestibular stimulus leading to a reduction in their postural deviations. Previous work using long-duration GVS (~ 76 s) demonstrated that the perception of rotation created by GVS can indeed subside over time (St George et al. 2011). However, if participants were simply habituating the ongoing GVS across perturbations P1–P3, removing GVS would have produced little alteration to the response to the last perturbation (R5). Instead, well after removing GVS and restoring baseline vestibular feedback, participants demonstrated a significant reversal of their ML responses (R5) such that their responses consisted of an incorrect and oppositely directed shift of their CoM and CoP trajectories (Figs. 3 and 4). This result suggests that the brain, rather than habituating to GVS, had recalibrated its interpretation of the previously altered vestibular feedback such that baseline vestibular feedback following GVS removal was no longer appropriate for controlling the dynamic ML response.

Is vestibular recalibration mediated by somatosensory feedback?

Somatosensory feedback may have been involved in attenuating ML sway during GVS across platform perturbations (Maurer et al. 2000; Creath et al. 2002; Maaswinkel et al. 2013). Day and Cole (2002) showed that in patient I.W., who has no available somatosensory feedback from below the neck, vestibular responses were an order of magnitude larger in the absence of vision. This suggested that in healthy individuals with intact somatosensory feedback, this feedback can suppress GVS-induced postural responses in the absence of vision (Day and Cole 2002). Therefore, somatosensory feedback may contribute to the attenuation of the ML component of the dynamic response across perturbations 1–4 in our NoVISION condition (Figs. 3 and 4). Importantly, if participants were simply weighting somatosensory over vestibular feedback over the course of the multiple perturbations, the dynamic ML component of the postural response would be expected to remain attenuated in response to both the fourth and fifth perturbations. However, concurrently increasing reliance on salient somatosensory feedback and decreasing reliance on vestibular feedback would result in a diminished influence of vestibular feedback on lateral body sway by the fifth platform perturbation. Instead, participants exhibited a reversal of their ML R5 magnitude, which was not significantly different from the attenuation in the dynamic response magnitude observed between perturbations P1 and P4 (Fig. 3). A similar reversal of vestibular influence following the removal of GVS has been observed following the recalibration of vestibular feedback during walking (Sturnieks et al. 2005), quiet standing (Héroux et al. 2015) and during the reestablishment of equilibrium following a platform perturbation (Toth et al. 2017) in which CoM and CoP equilibrium positions reversed well after the restoration of baseline vestibular feedback following GVS removal. Wright et al. (2014) pose that once a motor pattern has been adapted, evidence of sensory recalibration is shown when a reversal of the normal motor pattern is observed upon restoring baseline sensory conditions. Therefore, based on our data, we conclude that the vestibular influence on the dynamic phase of the perturbation recovery response can be recalibrated; however, vision does not appear necessary

for this recalibration. Instead, similar to the conclusions of Héroux et al. (2015), it may be that somatosensory feedback from the feet and ankles is contributing to the observed recalibration.

Vestibular recalibration during the early center of pressure response

Upon examination of the dynamic ML CoP perturbation responses, we noticed that in addition to the anode-directed response in perturbations P1–P4 during GVS, an earlier and oppositely directed component of this dynamic response (towards the cathode; *e*R) existed, making the dynamic CoP response biphasic. This early phase of the response shows similarities to the "K1" response described by Smetanin and Popov (1990) in that the two are similar in latency (initiated approximately 200 ms after the onset of GVS), similarly directed (both are in the direction of the cathode during GVS), and both appear to eventually move the CoM in the direction of the anode.

However, the early responses observed in the current study are unique in that they are not caused purely by the onset of GVS. Rather, these responses occurred in response to the platform perturbations while GVS was ongoing. Horak and Hlavacka (2002) identified vestibular contributions to early EMG responses in postural muscles in response to platform perturbations paired with GVS onset. However, they mention that appropriate resolution may not exist in CoP variables to identify a vestibular contribution to early postural responses. In the current study, the platform and vestibular perturbations occurred in orthogonal planes (AP and ML), and because any direct ML responses to the perturbation were removed (see "Methods"), we are able to observe a vestibular contribution to the early phase of the dynamic perturbation recovery responses.

Similar to CoP Rs, participants were able to attenuate the magnitude of their ML CoP *e*Rs across perturbations P1–P4 in the presence of GVS in both the NoVISION and VISION conditions. However, unlike what was observed for the CoP Rs where the response adaptation appeared to be accentuated by vision, vision did not seem to facilitate the attenuation of CoP *e*Rs across platform perturbations relative to those responses in the NoVISION condition (although the small size of the early response may have masked such an effect). Following GVS removal, the *e*R5 also reversed in direction with a magnitude that did not significantly differ from the magnitude of response reduction between *e*R1 and *e*R4. These findings further substantiate the earlier claim that the influence of vestibular feedback on the dynamic phase of perturbation recovery responses can be recalibrated, but that vision is not necessary for this recalibration. Work by Smetanin and Popov (1990) supports the lack of visual influence on the early component of the vestibular response. These authors demonstrated that vision did not exert a great influence toward modifying the early K1 response to GVS. As mentioned earlier, we suggest that rather than vision, alternative sources of intact and reliable feedback, such as somatosensory feedback from skin or muscle spindles, are perhaps contributing to the observed recalibration of vestibular feedback here. However, further work is required to determine the specific mechanisms involved.

Anteroposterior CoM and CoP perturbation responses

Could alterations in the perturbation response along the orthogonal AP axis explain the effects we observe in the ML response? The posterior platform perturbations induced large AP CoM and CoP responses which were more rapidly reduced in the presence of vision (VISION R1 and R2 responses are significantly attenuated compared to the same responses in the NoVISON condition; Fig. 5). This corroborates previous findings that the availability of vision can modify platform perturbation responses (Soechting and Berthoz 1979; Jilk et al. 2013). However, we do not observe the same consistent response modulation in the AP direction that is evident in the ML direction. This is particularly evident when contrasting responses to P1 and P5 for AP and ML variables. For example, when specifically comparing the responses between P1 and P5 for both CoM and CoP and in NoVISION and VISION conditions, we observe that although the AP CoP R5 in the NoVISION condition was significantly smaller than R1, the final perturbation response (R5) was not different in direction or magnitude compared to the R1 response for the VISION AP CoP as well as AP CoM in both visual conditions (see Table 2). This contrasts with the observed pattern of ML response between perturbations 1 and 5, specifically with the R5 response oriented in the opposite direction and not significantly different in magnitude when compared to the response attenuation across perturbations P1–P4 (Fig. 3). These findings suggest that separate mechanisms may be controlling AP and ML responses. This notion is supported by Winter et al. (1996), who show evidence of separate mechanisms for the control of AP and ML balance. They modeled AP balance using an inverted pendulum model that pivots about the ankle joint and modeled ML balance using a hip load/unload model. Furthermore, Mian and Day (2014) show that lightly touching a stable reference during GVS while the head is turned 45° relative to the feet only stabilizes the ML component of the sway response and not the AP component, further supporting a dissociation between AP and ML postural control mechanisms.

Conclusion

We have shown that the vestibular influence on the dynamic phase of the response to a perturbation can be recalibrated. Importantly, during the initial response adaptation, we see that vision may facilitate this process but does not appear to be necessary. As a result, we question extent of the involvement of vision in the recalibration process and instead, propose that alternate mechanisms may be involved. Specifically, we suggest that somatosensory feedback may be being used to drive the observed vestibular recalibration. Furthermore, we have identified, for the first time, a vestibular contribution to the early dynamic CoP component of the whole-body response to platform perturbation and demonstrated that this component too can be recalibrated. Overall, this work further illuminates the complexity behind the recovery response to a sudden physical threat to stability and how sensory feedback may be differentially integrated to control different phases of the response.

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