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Cortical facilitation of somatosensory inputs using gravity-related tactile information in humans with vestibular hypofunction

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Marie Fabre¹, Laura Beullier¹, Chloé Sutter¹, Amirreza Kebritchi², Pascale Chavet⁴, Martin Simoneau^{2,3}, Michel Toupet^{5,6}, Jean Blouin¹, Laurence Mouchnino^{1,7*}

¹ Aix Marseille Univ. CNRS, Laboratoire de Neurosciences Cognitives, FR 3C, Marseille, France

² Centre Interdisciplinaire de Recherche en Réadaptation et Intégration Sociale (CIRRIS) du CIUSSS de la Capitale Nationale, Québec, Canada

³ Département de kinésiologie, Faculté de médecine, Université Laval, Québec, QC G1V 0A6, Canada

⁴ Aix Marseille Univ, CNRS, Institut des Sciences du Mouvement, Marseille, France

⁵ IRON, Institut de Recherche en Oto-Neurologie, Paris, France

⁶ Centre d'Explorations Fonctionnelles Oto-Neurologiques, Paris, France

⁷ Institut Universitaire de France

*Corresponding author: Laurence Mouchnino

Aix-Marseille Université, CNRS, Laboratoire de Neurosciences Cognitives, FR 3C

3 place Victor Hugo, 13331 Marseille, France

Phone : 33 4 13 55 09 47

E-mail : laurence.mouchnino@univ-amu.fr

Running head: Tactile-based compensation of vestibular hypofunction

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Abstract

A few years after their bilateral vestibular loss, patients usually show a motor repertoire that is almost back to normal. This recovery is thought to involve an up-regulation of the visual and proprioceptive information that compensates for the lack of vestibular information. Here, we investigated whether plantar tactile inputs, which provide body information relative to the ground and to the Earth-vertical, contribute to this compensation. More specifically, we tested the hypothesis that somatosensory cortex response to electric stimulation of the plantar sole in standing adults will be greater in humans ($n = 10$) with bilateral vestibular hypofunction (VH) than in an age-matched healthy group ($n = 10$). Showing significantly greater somatosensory evoked potentials (i.e., P_1N_1) in VH than in controls, the electroencephalographic recordings supported this hypothesis. Furthermore, we found evidence that increasing the differential pressure between both feet, by adding a 1 kg mass at each pendant wrist, enhanced the internal representation of body orientation and motion relative to a gravitational reference frame. The large decrease in alpha power in the right posterior parietal cortex (and not in the left) is in line with this assumption. Finally, behavioral analyses showed that trunk oscillations were smaller than head oscillations in VH and showed a reverse pattern for healthy participants. These findings are consistent with a tactile-based postural control strategy in the absence of vestibular input, and a vestibular-based control strategy in healthy participants where the head serves as a reference for balance control.

Introduction

When standing upright, balance can be jeopardized by gravitational force if not properly controlled. This control is thought to involve feedforward processes, and to rely on the use and the quality of internal representations of body orientation and motion in space (Borel et al., 2008; Massion, 1992; Pfeiffer et al., 2014 for reviews). Observations in humans with major vestibular hypofunction (VH) suggest that the gravitational reference frame is crucial for encoding body-related information. The vestibular system is paramount for constructing the geocentric frame of reference as it senses linear and angular head (body) accelerations (linked to earth gravitational force). Alteration of this reference frame would be responsible for the trunk deviation relative to the earth-vertical which is commonly observed in VH humans standing on a moving surface (Horak et al., 2002). Similar body deviations were also reported in cats with bilateral labyrinthectomy during comparable stance perturbations (Macpherson et al., 2007). With impaired otoliths-based gravitational reference (see Day & Fitzpatrick, 2005 for a review), the nervous system would detect body motion relative to the supporting surface (through the processing of somatosensory inputs) but would be impaired when determining how the body is moving relative to the earth-vertical (see Horak et al., 2002). Other cat studies found delayed postural responses following translations of the supporting surface when somatosensory inputs were removed (with a large dose of Pyridoxine, Stapley et al., 2002), but not after bilateral labyrinthectomy (Inglis & Macpherson, 1995). Therefore, the greater incidence of falls and the reduced quality of life in humans with VH (Herdman et al., 2000; Ward et al., 2013; Zingler et al., 2008; Pavlou et al., 2006) even years after the onset of their first symptoms, cannot solely be attributed to a deficit in postural reactions. Rather, vestibular disorder complications could be linked to an impaired representation of body orientation and motion relative to the external world (i.e., earth gravity vertical).

It is well known that sensory loss or decrease in the reliability of sensory information can alter the processing of the spared sensory modalities. In such cases, intact sensory inputs are upregulated, and are given more weight by the central nervous system (Angelaki & Laurens, 2020; Bronstein, 2016; Lopez et al., 2007; Toupet et al., 2017). Surprisingly, despite the fact that the plantar mechanoreceptors provide relevant information about body position and velocity relative to the ground and to the earth-vertical (e.g., Carriot et al., 2004; Morasso & Schieppati, 1999; Mouchnino & Blouin, 2013), it has yet to be determined whether cutaneous inputs are enhanced in absence of vestibular information. Upregulation of cutaneous inputs in VH humans would support studies showing that the brain can selectively increase the functional

gain of task-relevant sensory inputs, including those from somatosensory systems during challenging equilibrium tasks (Duysens et al., 1995; Mouchnino et al., 2015; Saradjian et al., 2013; 2019).

The primary aim of the present study was to explore whether the response of the somatosensory cortex to foot cutaneous stimulation increases in humans with bilateral VH. We tested this hypothesis by measuring the amplitude of the P₁N₁ cortical response, extracted from the electroencephalogram, following the electric stimulation of the plantar sole (i.e., somatosensory-evoked potential (SEP) technique). We reasoned that the amplitude of the P₁N₁ should inform about how VH individuals and healthy controls weighted foot cutaneous inputs during upright standing.

Importantly, whether or not foot cutaneous inputs are upregulated in VH individuals, balance-related issues are still reported in individuals with vestibular disorders (Herdman et al., 2000; Ward et al., 2013; Zingler et al., 2008). This suggests that, if present, the sensory compensation may not suffice to create a reliable internal gravitational reference frame. Our second aim was to test if increasing the differential forces between both feet would enhance the internal representation of body orientation and motion relative to a gravitational reference frame in humans with VH. When standing still, the foot sole undergoes pressure variations due to postural sways: the pressure exerted under the loading foot increases while the pressure exerted under the unloading foot simultaneously decreases (Winter et al., 1996). This loading/unloading provides gravity-related tactile information as slow adapting afferent fibers (e.g., Merkel and Ruffini) from the plantar sole increase their firing rate resulting from changes in force applied to the skin (Vallbo & Johansson, 1984; see Macefield, 2005 for a review). Therefore, the impulse discharge pattern alternates between the feet depending on the loading or unloading. This phenomenon is reminiscent of the effect of body tilts on vestibular neurons in healthy humans. The neurons located in the labyrinth ipsilateral to the tilt increase their discharge rates while neurons of the contralateral labyrinth concomitantly decrease theirs (Fernandez & Goldberg, 1976). Shaped by this push-pull like organization of the vestibular system, vestibular signals provide key information on the body's orientation relative to gravity (Angelaki et al., 2004; Merfeld & Zupan, 2002).

The differential forces between both feet of standing participants (VH and Controls) were increased by adding a 1 kg mass to each of their wrist. We hypothesized that adding a weight to both wrists should enhance the detection of body sways through plantar sole mechanoreceptors due to improved sensorimotor integration (larger weight assigned to lower limb somatosensory inputs), resulting in more accurate encoding of body sways within the

gravitational field in the absence of vestibular information. The right posterior parietal cortex (rPPC) is a key region for integrating tactile information into body representation (Tsakiris et al., 2008, Wang et al., 2016), for perceiving upright through different sensory modalities (Kheradmand et al.2015), and for processing body motion relative to the gravitational field (see Pfeiffer et al., 2014, for a review). Observing an increased activity of the rPPC when humans with VH wore the added mass would support our hypothesis. The approach we used to test this hypothesis was based on the current consensus that functional processing of sensory inputs is associated with the modulation of band-specific neural oscillation power (Haegens et al., 2011; Lebar et al., 2017; Van Ede et al., 2012; Zumer et al., 2014; Pfurtscheller & Lopes da Silva, 1999). We predicted that with the added mass, this modulation would specifically target PPC alpha and beta power bands, which have been associated with the processing of tactile information (Van Ede et al. 2012), notably relative to external frames of reference (Ruzzoli & Soto-Faraco, 2014).

Participants and methods

Participants

Ten participants (including 3 women) with bilateral idiopathic vestibular hypofunction took part in the study. Their clinical evaluations are presented in Table 1. They were aged between 44 and 71 (mean 61 years) and their body mass index ranged from 22 and 33 (mean 26, i.e., healthy weight) and their mean height was 1.73 m). For all VH participants, the vestibular pathology started at least 5 years prior to the study. The bilateral vestibular hypofunction was confirmed by a major deficit in the four caloric vestibular tests (the speed of the nystagmus' slow phase was below 6°/s for all four caloric tests, Bárány 1906). The canal function was assessed by measuring the ocular response to low frequency rotations (0.05 Hz, period 20 s), the saccular and the utricular function were tested by measuring vestibular-evoked myogenic potentials (VEMP, respectively on the sternocleidomastoid muscle and on the inferior eyelid). Most VH participants showed no ocular or muscular response to the rotatory and VEMP tests for utricular function, but they showed myogenic responses to the VEMP for saccular function. All additional pathologies (e.g., hearing loss) and/or other vestibular pathologies led to the patient's exclusion from the study. A control group (Controls) was composed of 10 self-declared healthy participants (including 3 women). They were aged between 52 and 64 (mean 58 years) and their body mass index (BMI) ranged between 22 and 31 (mean 25, healthy weight, mean height 1.71 m).

All protocols and procedures were conducted in accordance with the ethical standards set out in the Declaration of Helsinki except for registration in a data base. All participants gave their written informed consent and procedures were approved by the local ethics committee of the Centre d'Explorations Fonctionnelles Oto-neurologiques Falguière (CEFON).

Task and stimulation procedure

Participants were asked to stand still in a comfortable position with their arms alongside their body and eyes open. They were instructed to prevent any voluntary movements (e.g., eyes, head, arm). Particular attention was paid to maintain constant the self-selected foot position before each trial to avoid potential effect of stance width on balance control (Day et al., 1993). Before the experimental sessions, participants were asked to self-select a comfortable side-by-side foot position and small sticker were positioned in front of each big toe. This allowed us to verify that the feet position remained similar (with an accuracy of a few mm) throughout the experiment.

Participants were asked to hold their gaze on a small cross fixed on a wall, 3m ahead at eye level. This distance was chosen to avoid vergence eye movements which can be impaired in humans with VH leading to instability in the mediolateral direction (Kapoula et al., 2013). For the main experiment, we recorded cortical activity, and pelvis and head accelerations (see below) of all participants (i.e., VH and Controls) in 16 trials of 15 s. Eight trials were performed while participants wore a 1kg weight bracelet (Fig. 1A) wrapped around each wrist (Weight condition) and 8 trials were performed without mass (No-weight condition). To avoid potential post-effects of carrying extra mass (e.g., on the frame of reference used for perceiving Earth-vertical), the No-weight condition (i.e., condition closest to everyday life) was always tested first. The participants were given several ~2 min rest periods during the experimental session, which lasted 25 minutes. During these rest periods, the participants walked on the spot to release skin compression and reactivate mechanoreceptors. This procedure was used to avoid sustained compression of the foot sole which can decrease the cortical potentials evoked by electric stimulations (see Fabre et al., 2021).

We chose to strap a 1 kg (i.e., 10 N) around each wrist to increase reaction force under each foot. Previous studies have shown that such load does not cause disequilibrium while standing upright but influences balance control (e.g., Aruin & Latash, 1995; Mouchnino et al., 2012; Simoneau et al., 2003). Obviously, the added weight increased the body gravitational force. According to basic laws of physics, compared to adding weights at the ankle joints, body

sways with weight at both wrists should also augment the shear forces under each foot. Despite being small relative to the weight force during natural quiet standing, the shear forces that are generated at the foot-ground interface, are readily detectable by the tactile receptors (Morasso & Schieppati, 1999; Knellwolf et al., 2018). The current study was conducted in a clinical setting which was not equipped with force platforms. Consequently, we developed a biomechanical model to verify 1) that adding a weight of 20 N alters the shear reaction forces under both feet during natural body sways and, 2) whether the shear forces increased more by adding the weight at the wrists (i.e., 10N) rather than at the ankles.

For the model, which was based on Peterka et al. (2018), we simulated a double inverted pendulum model (mass = 75 kg, height = 171 cm) with stiffness (K) and damping (B) at the ankle and hip joints (Fig. 1A). The biomechanical model only swayed along the frontal plane with an amplitude of 10 mm (i.e., amplitude of center of mass displacement commonly recorded during quiet standing, e.g., Lafond et al., 2004) with frequencies of 0.25, 0.5 or 0.75 Hz. Compared to simulations without the added weight, the outcomes of the model showed that the mediolateral (ML) shear reaction forces were altered during body oscillations (Fig. 1B). Importantly the forces increased under the loaded foot when the added mass was located at the wrists and decreased when located at the ankles location. The increase in shear force (wrist location) likely enhanced the stimulation of the plantar sole mechanoreceptors. As expected, during postural oscillations results of the model also confirmed that adding weights at both wrists increased the vertical force under one foot while it decreased under the other foot.

In the present study, all participants self-declared being right-handed. We stimulated the left foot for all participants because it is considered as the “postural” foot in most right-handed humans (Coren, 1993). Electric stimulation of the foot was delivered via a set of 2 Carbon with metal screening foam electrodes (5 × 9 cm) using an isolated bipolar constant current stimulator (DS5 Digitimer, Welwyn Garden City, UK). Using the same technique as in previous studies (Fabre et al., 2020; Mouchnino et al., 2015), the cathode was located under the metatarsal region and the anode underneath the heel (Fig. 2A). Cutaneous afferents from plantar sole are distributed across the foot sole (with some regional variation, Strzalkowski et al., 2018). Note that due to their low intensity, the electric stimulations might have more specifically stimulated the medium cutaneous afferents (i.e., A β , Williams et al., 1998), which mainly innervate low-threshold mechanoreceptors. Each stimulation consisted of a single rectangular 10-ms pulse which proved to be effective in generating salient SEPs over the somatosensory cortex (e.g., Lhomond et al., 2016; Mouchnino et al., 2015; Fabre et al., 2021). Since the inter-participants differences can be a factor that influences the SEP amplitude, all participants were tested

relative to their perceptual threshold as commonly used for measuring and monitoring somatosensory functions (see for examples Duysens et al., 1995; Sayenko et al., 2009; Cybulska-Klosowicz et al., 2011). This threshold was estimated prior to the experimental session using a forced-choice adaptive method (Ehrenstein & Ehrenstein, 1999), while the participants were standing without added mass and were gazing at the fixation cross. For each participant, stimulation intensity was set 25% above the tactile perceptual threshold (i.e., below the motor threshold) and was not painful.

The mean stimulation intensities used for VH and Controls groups are reported in the Results section. All stimuli were clearly detectable but did not create illusion of body motion, as mechanical stimulation of the foot can do (Kavounoudias et al., 1998). Moreover, a previous study using stimulation intensities similar to those employed here showed that such stimulations are not strong enough to evoke reflex-triggered postural response which could alter normal body sway (Mouchnino et al., 2015).

Fourteen sets of stimuli were delivered in each 15 s trial, giving a total of 98 stimulations for each condition (i.e., No-weight and Weight). We chose inter-stimulus intervals of 1000 ms \pm 20%. These stimulus intervals were chosen on the basis that regular intervals are known to boost the cortical response to somatosensory stimulation compared to irregular intervals (e.g., \pm 20% jitter in Mujunen et al., 2021). In this light, the use of a \pm 20% jitter appeared as a suitable conservative method for assessing the cortical sensitivity to somatosensory inputs during the standing task. Moreover, because the inter-stimulus intervals were $>$ 800 ms, the evoked response was unlikely influenced by the previous stimulation. Indeed, when the inter-stimulus intervals is $<$ 500 ms, a decrease in the evoked response is commonly observed due to the previous stimulation (Burke & Gandevia, 1988).

Electrophysiological recordings and analyses

As described previously (Fabre et al., 2021), electroencephalographic (EEG) activity was recorded continuously from 64 Ag/AgCl surface electrodes embedded in an elastic cap (10-20 system, BioSemi ActiveTwo system: BioSemi, Netherlands). Specific to the BioSemi system, “ground” electrodes were replaced by Common Mode Sense active and Driven Right Leg passive electrodes. The signal was pre-amplified at the electrode sites and post-amplified with DC amplifiers, digitized at a sampling rate of 1024 Hz (Actiview acquisition program, BioSemi). The signals of each electrode were referenced to the mean signal of all the 64 electrodes. Three external Ag/AgCl electrodes (positioned next to the right and left external canthus and under the left eye orbit) and one from the cap (FP1) allowed controlling for ocular

movements and blinks. After artefact rejection based on visual inspection, 87% and 83% of the trials were included in the analyses for VH and Controls groups, respectively.

As in previous studies (Duysens et al., 1995, Fabre et al., 2021; Staines et al. 2000), the largest responses to foot stimulations were observed at electrode Cz (vertex). The SEPs were recorded at this electrode which overlays the sensorimotor feet regions located on the inner surface of the longitudinal fissure. These SEPs were obtained by averaging, for each participant and condition (i.e., No-weight and Weight), all synchronized epochs relative to the electrical stimulus (Fig. 2A). We searched, from 25 ms post-stimulus onward (i.e., after stimulation artifact), for the earliest discernible positive and negative peaks. Overall (groups, mass conditions), the latencies of P₁ and N₁ were respectively of 59 ± 9 ms and 97 ± 16 ms and were like those reported in previous studies using similar methods (e.g., Fabre et al., 2020, Duysens et al., 1995). Amplitudes of the P₁N₁ were measured peak-to-peak between these positive and negative deflections (Fig. 2A).

Time frequency analyses were performed in EEG source space. The neural sources were estimated with dynamical Statistical Parametric Mapping (dSPM, Dale et al., 2000) implemented in the Brainstorm software (Tadel et al., 2011). A three-shell (i.e., scalp, outer skull, and inner skull) sphere boundary element model (BEM) was used to compute the forward model on the anatomical MRI brain template from the Montreal Neurological Institute (MNI Colin27). The data were transformed into the time-frequency domain using Morlet wavelet transforms. Due to the Gaussian shape of the frequency response, Morlet wavelets produce smooth-looking time-frequency plots which can be easily interpreted (see Fig. 3B, where activities in distinct frequency bands can be identified). We used a 1Hz central frequency (full width at half maximum (FWHM), $t_c=3$ sec) that offered a good compromise between temporal and spectral resolutions (Allen & MacKinnon, 2010). We computed alpha (8–12 Hz) and beta (15–25 Hz) event related desynchronization/synchronization (ERD/ERS) using a baseline window (-500 to -50 ms). We purposely selected a 300 ms time window for the ERS/ERD computation [-100; +200 ms relative to the stimulation] to remove the edge effects for the following extraction. We extracted the power average of alpha and beta event-related ERS/ERD from 50 to 100 ms (i.e., interval encompassing the P₁N₁ SEP). These computations were performed in regions of interest (ROIs) from the right and left PPC (see Fig. 3A). These ROIs were defined based on the Destrieux cortical atlas (Destrieux et al., 2010) and had similar numbers of vertices. We chose to analyze alpha and beta power in this region as for the VH participants, the added mass should enhance the internal representation of body orientation and motion relative to a gravitational reference frame. Consequently, we predicted that the VH

participants' right PPC should show greater alpha and beta ERDs in the Weight condition than in the No-weight condition. The left PPC, which is not considered to be involved in integrating tactile information into body representation, served as a control ROI. Due to extensive artifact in the beta band, data of 2 participants of the control group were excluded from the time-frequency analysis of the left PPC (i.e., control ROI).

Behavioral recordings and analyses

Upright standing was assessed by measuring head and pelvis accelerations. Head acceleration was measured with a triaxial accelerometer (Model 4630: Measurement Specialties, USA) placed on the participants' chin. The accelerometer was securely fixed on a chin-cup (Fig. 1A). This chin-cup reduced the possibility to move the jaw. Pelvis acceleration was estimated using a triaxial accelerometer (Model ADXL335: Analog Devices, USA) located on the sacrum (i.e., an approximation of the body center of mass, Thirunarayan et al. 1996). Signals from these sensors were recorded at a sampling rate of 1000 Hz and low-pass filtered (Butterworth 4th order, 10 Hz cutoff frequency). Head and pelvis accelerations were analyzed in both anteroposterior (AP) and mediolateral (ML) axes. We computed, for each trial, the integrals of the ML and AP absolute values over time (15 sec) to estimate the amount of acceleration in each direction and calculated the mean ML and AP accelerations for each condition and group. These analyses provided an estimate of head and pelvis acceleration in ML and AP directions for both groups (VH and Controls) and both experimental conditions (i.e., No-Weight and Weight).

Statistical Analyses

The behavioral and neurophysiological dependent variables were submitted to separate analyses of variance (ANOVA, Statistica software) as the data had a normal distribution (Kolmogorov-Smirnov test). The homogeneity of variance was assessed using Levene's Test for Equality of Variances. Since the sample size of the groups was small, partial Eta-Squared values (η^2) for the effect size were given for the EEG data. Effect size higher than 0.14 were considered as large effect size (Cohen, 1988). For SEP amplitude and latency and alpha/beta ERS/ERD, mixed-design ANOVAs were used for mean comparisons with Condition (No-weight, Weight) as a within-subject factor and Group (VH, Controls) as a categorical predictor. For head and pelvis accelerations, we used 2 (Group: VH, Controls) x (Condition: No-weight, Weight) x 2 (Direction: AP and ML) ANOVAs with repeated measures on the last two factors. For post-hoc analyses, we used Newman-Keuls comparisons. When main effects could be

solely explained by a higher order interaction, only the break-down of the interaction will be reported. Significance threshold was set at $p < 0.05$ for all analyses.

Results

Foot stimulation perceptual threshold

The mean (\pm standard deviation) thresholds for perceiving foot stimulations were 6.95 ± 2.3 mA for the VH and 6.83 ± 1.3 mA for Control groups. The result of a t-test indicated that perceptual thresholds did not differ between both groups ($t = -0.23$; $p = 0.81$). This result suggests that if the SEP differed between the VH and Control groups, this difference would unlikely result from difference in stimulation intensities.

SEP response to foot tactile stimulation

Consistent with an upregulation of plantar cutaneous inputs for the VH group, the evoked response to the foot cutaneous stimulation was larger in VH than in Control group (Fig. 2A-B). This was confirmed by the ANOVA showing that P_1N_1 amplitude was greater in VH than in Control groups (Figure 2B; $F_{1, 18} = 5.84$; $p = 0.02$, $\eta_p^2 = 0.44$). Adding a 2 kg mass did not change the SEP amplitude, either as a main effect ($F_{1, 18} = 2.19$; $p = 0.15$) or as an interaction between Weight and Condition ($F_{1, 18} = 0.36$; $p = 0.55$).

For the SEP P1 latency, the ANOVA did not reveal a significant effect of Group ($p = 0.82$). However, the main effect of Condition fell just short of the conventional 0.05 cut-off value for statistical significance ($F_{1, 18} = 4.16$; $p = 0.07$; Weight condition: $58 \text{ ms} \pm 5$, No-weight condition: $61 \text{ ms} \pm 8$). No Group by Condition interaction was observed ($F_{1, 18} = 0.36$; $p = 0.55$).

Following these results, we tested a group of 10 younger healthy participants (mean age of 39 ± 8) using the same procedures as in the main experiment, to determine if the VH's SEPs were more alike those of younger healthy participants' SEPs than to those of healthy age-matched participants. Because perceptual threshold is likely much smaller in young adults (~ 5 mA in the 25 year-old individuals in Lhomond et al., 2016) than in our older VH and Control participants (~ 7 mA), younger adults were not tested. Results of the ANOVA revealed that the mean stimulation intensity at perceptual threshold did not significantly differ between the 3 groups of participants (39 years and 58 years Control groups, and VH group) ($F_{2, 27} = 0.34$; $p = 0.71$, $\eta_p^2 = 0.24$).

The ANOVA and post-hoc analyses revealed that the P₁N₁ amplitudes for the VH and Younger groups were not significantly different ($p = 0.53$; overall mean: -2.01 , Fig. 2C), but were greater in these groups than in the older Control group (Fig. 2C, mean: $-1.04 \text{ mV} \pm 0.45$) ($F_{2, 27} = 4.21$; $p = 0.025$; Fig. 2C). The SEP's latency did not significantly differ between the groups (overall mean = $56 \text{ ms} \pm 8$, $F_{2, 27} = 1.67$; $p = 0.2$, $\eta_p^2 = 0.11$).

EEG spectral analyses

Consistent with an increased activity of the right PPC when adding weight in VH (Fig. 3B-C), the alpha power desynchronization increased for the VH group whereas for the Control group, adding weight to the wrists did not have any significant effect (significant Group by Condition interaction, $F_{1, 15} = 6.01$; $p = 0.02$, $\eta_p^2 = 0.28$). A trend for decreasing was observed for the beta band frequency without reaching the significant value for statistical significance ($F_{1, 15} = 4.1$; $p = 0.062$). No significant effect of Condition was observed in the left PPC for both groups, neither for alpha, nor for beta frequency bands ($F_{1, 15} = 1.17$; $p = 0.29$ and $F_{1, 16} = 3.68$; $p = 0.072$, respectively for alpha and beta).

Behavioral results

Variables related to balance control were analyzed to compare body sways between VH and Control groups, and to verify whether the added mass altered body sways. For pelvis accelerations (Fig. 4A), the ANOVA revealed a main effect of Group; pelvis acceleration integral being greater in the Control than in the VH groups ($F_{1, 17} = 79$; $p < 0.001$). For both groups, the pelvis acceleration was greater in the Weight compared to the Non-Weight conditions (main effect of Condition: $F_{1, 17} = 8$; $p = 0.01$). The analyses also revealed a significant interaction Group x Direction ($F_{1, 17} = 27$; $p < 0.001$). The breakdown of this interaction revealed that pelvis acceleration was greater in the AP direction than in ML for the Control group ($p < 0.001$), but did not differ between AP and ML directions for the VH group ($p = 0.89$).

Interestingly, for the head accelerations (Fig. 4B), the analyses yielded different results; the head acceleration varied across groups and directions (Group by Direction interaction: $F_{1, 17} = 5.4$; $p < 0.032$). For the Control group, contrary to what was observed for the pelvis, the head accelerations were similar along the AP and ML directions ($p = 0.37$). The mean head acceleration of the Control group was not different to the threshold for vestibular detection (0.048 m.s^{-2} ; Gianna et al. 1996) [t -test for means against a value; $t_8 = 1.09$, $p = 0.30$ and $t_8 = 1.03$ $p = 0.33$ respectively for Weight ($0.055 \pm 0.02 \text{ m.s}^{-2}$) and No-weight ($0.052 \pm 0.01 \text{ m.s}^{-2}$)

conditions]. Conversely, for VH group, the head accelerations were different between AP and ML directions; head acceleration being significantly greater in AP direction ($p = 0.0031$). No main effect of Group ($F_{1, 17} = 0.003$; $p = 0.95$) or Condition ($F_{1, 17} = 0.38$; $p = 0.54$) was observed.

Discussion

Due to the properties of its sensors and their afferents (e.g., otolith neurons) which cannot be turned off (Fernandez & Goldberg, 1976), the vestibular system is a valuable source of information used by the brain to preserve balance during bipedal stance. Being sensitive to linear and angular head accelerations, it provides key information for determining the Earth vertical axis and for estimating how the body deviates from it (Day & Fitzpatrick, 2005). Here, we found evidence that this function could be supported, at least in part, by foot cutaneous inputs in humans with bilateral vestibular hypofunction. Our results provide valuable insights into the mechanisms underpinning this sensory substitution.

We found that the response of the sensorimotor cortex to foot cutaneous stimulations was much greater in VH participants than in age-matched healthy humans. Although the present experimental protocol does not account for the origin of this finding, the increased SEP in VH unlikely resulted from a lower perceptual threshold of the plantar sole tactile receptors. Indeed, our results showing similar detection thresholds for foot cutaneous stimulation between VH and Control groups provides evidence against this possibility. Distinct mechanisms could explain the larger SEP in the VH group. The multimodal cells, where vestibular and somatosensory inputs converge, could have augmented their responsiveness to cutaneous stimulations following reduced vestibular inputs. Such cells are present, for example, in the somatosensory cortex (Zarzecki et al., 1983; Guldin and Grüsser, 1998) and in the thalamus (Lopez and Blanke, 2011). Enhancing the weight of somatosensory inputs was shown by Bolton et al. (2011) in standing participants touching lightly with a finger, a surface that provides a stable reference frame to maintain equilibrium. Such effect of light finger touch was also associated with decrease of vestibular responsiveness in healthy human standing (Mian & Day, 2014). These findings obtained from healthy participants are in line with a strong interdependence between the vestibular and somatosensory inputs.

Another possible mechanism responsible for the larger SEP in VH may involve the thalamus. This principal sensory relay station sends potentiated inputs to the brain through the effect of the reticular nucleus which intercepts and modulates all corticothalamic (including from prefrontal cortex) and thalamocortical communications (Guillery et al., 1998; Zikopoulos

& Barbas, 2006). Because cells that respond exclusively to vestibular stimulations have not yet been discovered in the brain, it is unlikely that greater SEP in participants with VH resulted from cross-modal plasticity. This phenomenon allows, for example, cells which respond to visual stimulation in sighted humans to be sensitive to cutaneous inputs in individuals with visual impairment (Bavelier and Neville, 2002; Sathian & Stilla, 2010).

Interestingly, the P1N1 amplitudes shown by the VH (mean age 61 years) and Younger control (mean: 39 years old) groups were not significantly different, but were greater in these groups than in the Older control group (mean: 58 years old). These results are important as they suggest that, through compensatory mechanisms, the response of the somatosensory cortex to plantar cutaneous inputs increased in our VH group to the level exhibited by healthy younger participants. This increased sensory sensitiveness likely developed through extensive reliance on tactile inputs in the absence of vestibular system function. These findings are compatible with the cortical plasticity observed in highly trained dancers/slackliners leading to a down regulation of vestibular inputs (which are potentially detrimental for balance control during vertiginous activities) and an enhancement of visual inputs processing (Hüfner et al., 2011).

Our behavioral data showed further evidence of somatosensory compensation for vestibular loss. Indeed, we observed that pelvis accelerations were small along both directions (i.e., AP and ML) for the VH group, while for the Control group, data revealed small head acceleration along both directions (see Fig. 3). The reduced head accelerations in the Control group could suggest that in healthy humans, the head serves as a reference frame for controlling upright standing. Note that, along the frontal plane, the mean head acceleration for the control participants was not significantly different than the acceleration threshold for vestibular detection (Gianna et al., 1996). Functionally, such near-vestibular threshold head acceleration puts the vestibular system in an optimal state for being stimulated in case of unexpected loss of balance.

Using the head as a frame of reference is thought to require intact vestibular information (Bent et al., 2004; Blouin et al., 2007; Horak et al., 2002; Pozzo et al., 1991). In this light, the greater pelvis stabilisation showed by VH participants suggests that, in absence of vestibular information, the brain could determine body sways amplitude and direction with respect to the base of support (i.e., new reference frame) through lower limb somatosensory and plantar sole afferents signals. This change in reference frame likely requires a tight control of the pelvis. In particular, the body sways can be regulated within a small area by reducing the amplitude of the centre of pressure displacement (not recorded in the present study) and increasing its frequency. Such tactile exploration, by changing the pressure onto the ground with the feet,

likely contributes to improve the signal-to-noise ratio of sensory cues and to determine the limit of stability (Fabre et al., 2021; Latash et al., 2003; Murnaghan et al., 2011). The increase in plantar sole afferents through this strategy and the upregulation of cutaneous inputs (evidenced here by greater SEP in VH than in Control groups) could be particularly important in the absence of vestibular information. Attentional processes, which are known to increase the excitability to sensory input (Eimer & Forster, 2003; Sambo & Forster, 2011; Bolton & Staines, 2012) could also contribute to the observed sensory upregulation in VH participants.

Perhaps more surprisingly, we found that the pelvis oscillations shown by VH in the AP direction were of similar amplitude to their oscillations in the ML direction. This behavior differs drastically from that normally shown by healthy humans who instead exhibit -as did our control group- much larger oscillations observed at the foot-ground interface in the AP direction than in the ML direction (Collins & de Luca, 1993; Lord et al., 1999; Johnson-Hilliard et al., 2008; Winter et al., 1996). For the VH group, the tight control of the pelvis acceleration in the AP direction may reflect a stronger awareness to the consequence of balance instability. A similar strategy was observed when the perceived risk of injury was increased by having healthy humans stand on a high surface (Adkin et al., 2000; Carpenter et al., 1999; Lim et al., 2017) and in patients with postural phobic vertigo (Krafczyk et al., 1999).

Our second hypothesis claimed that enhancing the differential pressure between both feet during standing should contribute in restoring the internal representation of the VH participants' body orientation and motion relative to a gravitational reference frame hosted in the right PPC (Pérennou et al., 2008; Dai et al., 2021a,b; Pfeiffer et al., 2014). In the present study, to enhance foot pressure during standing, we added a 1 kg mass to participants' both wrists. Results from the biomechanical model revealed that this procedure magnified the differential pressure between both feet, and more specifically the shear forces. The analyses of the cortical alpha and beta power are consistent with an enhanced internal presentation of the body when VH participants stood upright with the added mass. Indeed, enhancing the seesaw effect on the feet's cutaneous receptors with the bilateral mass addition resulted in a significant right-lateralized decrease of the PPC alpha power in the VH (beta band ERD, $p = 0.062$) but not in the Control group. The powers of the alpha and beta oscillations are inversely related to cortical excitability and to the reliance on sensory processing, respectively (Neuper & Pfurtscheller, 2001; Romei et al., 2008; Sauseng et al., 2009). For the VH participants, the decreased in alpha power observed in the right PPC might entail integration of cutaneous inputs to improve body orientation representation relative to the gravitational field. There is a large body of evidence, notably from clinical studies, suggesting that determining self-orientation

with respect to gravity involves the right PPC (Pérennou et al., 2008; Dai et al., 2021a, b; Pfeiffer et al., 2014). The increased activity (i.e., increase alpha desynchronization in VH), which was specifically found in the right PPC is therefore in line with an enhancement of the internal representation of body orientation and motion relative to a gravitational reference frame.

The PPC is known to receive thalamic projections of cutaneous cues through either direct (Padberg et al. 2009; Impieri et al. 2018) or indirect (i.e., through SII/parietal ventral, Cavada and Goldman-Rakic 1989; Disbrow et al. 2003) pathways. In VH participants, the contribution of the PPC to balance control could have been enhanced likely by increasing tactile afferents in the Weight condition. This would be consistent with findings suggesting that the reliance on internal representations for controlling balance increases in contexts with impaired sensory inputs (Blouin et al., 2007; Fabre et al., 2020). The importance of cutaneous inputs in building a representation of the body orientation relative to a gravitational reference frame was also reported in a study with a deafferented patient (Day & Cole, 2002). The patient (IW), who suffered from a rare lack of cutaneous and proprioceptive inputs (see Cole & Waterman, 1995), experienced a “floating sensation” and a “loss of contact with the world” when receiving galvanic vestibular stimulations (GVS) while seated. This unusual effect suggests an inability to create an accurate spatial representation of the body orientation in the absence of cutaneous inputs, even when vestibular information is preserved.

Some limitations in the present study should be considered. The protocol that we used does not allow determining whether the sensory upregulation observed in VH only concerned tactile inputs from the plantar sole or whether it is generalized to other somatosensory inputs (e.g., lower limb proprioception). Further studies are necessary to elucidate this issue and also to determine whether sensory upregulation only concerned plantar receptors. It should also be noted that VH did not show greater body stabilization when holding bilateral weights. This could be due to the small weights of the bracelets (i.e., 1 kg) and to the choice of a quiet standing task. Further experiments are required to determine if carrying bracelets improves balance in more challenging balance tasks (e.g., step initiation, obstacle avoidance task).

In conclusion, we found that plantar cutaneous inputs are upregulated in humans with vestibular information processing deficits. Such sensory upregulation points to an increased role of cutaneous input in balance control in these participants. The greater reliance on foot tactile information was also suggested by the change in balance control and postural organization of the VH, which led to a greater stabilization of the pelvis than of the head. Finally, we found that increasing the load/unload response under both feet of the VH

participants (with bilateral weights) changed the dynamics of the right PPC neural activity (i.e., increased alpha ERD). This observation supports our hypothesis suggesting that an increased in processing plantar sole tactile information enable building up an internal representation of body orientation relative to a gravitational reference frame.

References

- Adkin AL, Frank JS, Carpenter MG & Peysar GW (2000). Postural control is scaled to level of postural threat. *Gait Posture* 12, 87-93.
- Allen DP & MacKinnon CD (2010). Time–frequency analysis of movement-related spectral power in EEG during repetitive movements: A comparison of methods. *Journal of neuroscience methods*, 186(1), 107-115.
- Angelaki DE & Laurens J (2020). Time course of sensory substitution for gravity sensing in visual vertical orientation perception following complete vestibular loss. *ENeuro* 0-36. <https://doi.org/10.1523/ENEURO.0021-20.2020>
- Angelaki DE, Shaikh AG, Green AM & Dickman JD (2004). Neurons compute internal models of the physical laws of motion. *Nature* 430, 560-564.
- Aruin AS & Latash ML (1995). The role of motor action in anticipatory postural adjustments studied with self-induced and externally triggered perturbations. *Experimental brain research*, 106(2), 291-300.
- Bavelier D & Neville HJ (2002). Cross-modal plasticity: where and how? *Nature Rev Neuroscience* 3, 443-52.
- Bárány R. (1906). Über die vom Ohrlabyrinth ausgelöste Gegenrollung der Augen bei Normalhörenden, Ohrenkranken und Taubstummen. *Archiv für Ohrenheilkunde*, 68(1-2), 1-30.
- Bent LR, Inglis JT & McFadyen BJ (2004). When is vestibular information important during walking? *Journal of Neurophysiology* 92, 1269-1275.
- Blouin J, Teasdale N, & Mouchnino L (2007). Vestibular signal processing in a subject with somatosensory deafferentation: The case of sitting posture. *BMC Neurology* 7: 25.
- Bolton DA, McIlroy WE & Staines WR (2011). The impact of light fingertip touch on haptic cortical processing during a standing balance task. *Experimental Brain Research* 212, 279–291.
- Bolton DA & Staines WR (2012). Age-related loss in attention-based modulation of tactile stimuli at early stages of somatosensory processing. *Neuropsychologia* 50,1502-13.
- Borel L, Lopez C, Péruch P & Lacour M (2008). Vestibular syndrome: A change in internal spatial representation. *Neurophysiologie Clinique* 38, 375–389.
- Bronstein AM (2016). Multisensory integration in balance control. *Handbook of Clinical Neurology* (1st ed., Vol. 137). Elsevier B.V. <https://doi.org/10.1016/B978-0-444-63437-5.00004-2>
- Buchholz VN, Jensen O, Medendorp WP (2014). Different roles of alpha and beta band

- oscillations in anticipatory sensorimotor gating. *Frontiers in Human Neuroscience* 8,446. doi: 10.3389/fnhum.2014.00446
- Burke D & Gandevia SC (1988). Interfering cutaneous stimulation and the muscle afferent contribution to cortical potentials. *Electroencephalography and Clinical Neurophysiology* 70, 118–125.
- Carpenter MG, Frank JS & Silcher CP (1999). Surface height effects on postural control: a hypothesis for a stiffness strategy for stance. *Journal of Vestibular Research* 9, 277–286.
- Carriot J, Bringoux L, Charles C, Mars F, Nougier V & Cian C (2004). Perceived body orientation in microgravity: Effects of prior experience and pressure under the feet. *Aviation Space and Environmental Medicine* 75, 795–799.
- Cavada C, & Goldman-Rakic PS (1989). Posterior parietal cortex in rhesus monkey: II. Evidence for segregated corticocortical networks linking sensory and limbic areas with the frontal lobe. *Journal of Comparative Neurology* 287:422–445
- Cohen J (1988) *Statistical power analysis for the behavioral sciences*. Lawrence Erlbaum Associates, Hillsdale
- Cole JD & Waterman I (1995). *Pride and a daily marathon*. MIT Press, Boston.
- Collins JJ & de Luca CJ (1993). Open-loop and closed-loop control of posture : A random-walk analysis of center-of-pressure trajectories. *Experimental Brain Research* 95, 308–318.
- Coren S (1993). The lateral preference inventory for measurement of handedness, footedness, eyedness, and earedness: Norms for young adults. *Bulletin of the Psychonomic Society* 31, 1-3.
- Cybulska-Klosowicz A, Meftah EM, Raby M, Lemieux ML & Chapman CE (2011). A critical speed for gating of tactile detection during voluntary movement. *Experimental brain research*, 210, 291-301.
- Dai S, Piscicelli C, Clarac E, Baciú M, Hommel M, Pérennou D (2021a). Balance, lateropulsion, and gait disorders in subacute stroke. *Neurology* 96:1-13.
- Dai S, Piscicelli C, Clarac E, Baciú M, Hommel M, Pérennou D (2021b) Lateropulsion after hemispheric stroke. A form of spatial neglect involving graviception. *Neurology* 96 (17).
- Dale AM, Liu AK, Fischl BR, Buckner RL, Belliveau JW, Lewine JD & Halgren E (2000). Dynamic statistical parametric mapping: combining fMRI and MEG for high-resolution imaging of cortical activity. *Neuron* 26, 55-67.
- Day BL & Cole J (2002). Vestibular-evoked postural responses in the absence of somatosensory information. *Brain* 125, 2081-8.
- Day BL & Fitzpatrick RC (2005) The vestibular system. *Current Biology* 15, R583-6.

- Day BL & Fitzpatrick RC (2005). The vestibular system. *Current biology*, 15(15), R583-R586.
- Day BL, Steiger MJ, Thompson PD & Marsden CD (1993). Effect of vision and stance width on human body motion when standing: implications for afferent control of lateral sway. *The Journal of physiology*, 469(1), 479-499.
- Destrieux C (2010). Automatic parcellation of human cortical gyri and sulci using standard anatomical nomenclature. *NeuroImage* 53, 1-15.
- Disbrow E, Litinas E, Recanzone GH, Padberg J, & Krubitzer L (2003). Cortical connections of the second somatosensory area and the parietal ventral area in macaque monkeys. *Journal of Comparative Neurology* 462: 382–399
- Duysens J, Tax AAM, Nawijn S, Berger W, Prokop T & Altenmuller E (1995). Gating of sensation and evoked-potentials following foot stimulation during human gait. *Experimental Brain Research* 105, 423–431.
- Ehrenstein WH & Ehrenstein A (1999). Psychophysical methods. *Modern techniques in Neuroscience research*. Windhorst U, Johansson H. (eds) Springer, Berlin, Heidelberg.
- Eimer M & Forster B (2003). Modulations of early somatosensory ERP components by transient and sustained spatial attention. *Experimental Brain Research* 151, 24–31.
- Fabre M, Chavet P, Fornerone T, Juan B, Abossolo O, Pardo F, Blouin J, Dany L & Mouchnino L (2020) Somatosensory cortical facilitation during step preparation restored by an improved body representation in obese patients. *Gait & Posture* 80: 246-252
- Fabre M, Antoine M, Robitaille MG, Ribot-Ciscar E, Ackerley R, Aimonetti JM, Chavet P, Blouin J, Simoneau M & Mouchnino L (2021). Large postural sways prevent foot tactile information from fading: neurophysiological evidence. *Cerebral Cortex Communications* 2, 1-10, <https://doi.org/10.1093/texcom/tgaa0942020>
- Fernandez C & Goldberg JM (1976). Physiology of peripheral neurons innervating otolith organs of the squirrel monkey. II. Directional selectivity and force response relations. *Journal of Neurophysiology* 39, 985–995.
- Gianna C, Heimbrand S & Gresty M. (1996). Thresholds for detection of motion direction during passive lateral whole-body acceleration in normal participants and patients with bilateral loss of labyrinthine function. *Brain Research Bulletin* 40, 443–447.
- Guillery RW, Feig SL & Lozsádi DA (1998). Paying attention to the thalamic reticular nucleus. *Trends in Neuroscience* 21, 28–32.
- Guldin WO (1998). Is there a vestibular cortex? *Trends in neurosciences* 21, 254 - 259.
- Haegens S, Händel BF & Jensen O (2011). Top-down controlled alpha band activity in somatosensory areas determines behavioral performance in a discrimination task. *Journal*

- of Neuroscience 31, 5197–5204.
- Herdman SJ, Blatt PJ & Schubert MC (2000). Vestibular rehabilitation of patients with vestibular hypofunction or with benign paroxysmal positional vertigo. *Current Opinion in Neurology* 13, 39–43.
- Horak FB, Buchanan J, Creath R & Jeka J (2002). Vestibulospinal control of posture. *Advances in Experimental Medicine and Biology* 508, 139–145.
- Hüfner K, Binetti C, Hamilton DA, Stephan T, Flanagan VL, Linn J., ... & Brandt T (2011). Structural and functional plasticity of the hippocampal formation in professional dancers and slackliners. *Hippocampus*, 21(8), 855-865.
- Impieri D, Gamberini M, Passarelli L, Rosa MGP, Galletti C (2018). Thalamo-cortical projections to the macaque superior parietal lobule areas PEc and PE. *Journal of Comparative Neurology* 526: 1041–1056.
- Inglis T & Macpherson JM. (1995). Bilateral labyrinthectomy in the cat: effects on the postural response to translation. *Journal of Neurophysiology* 73, 1181-91.
- Johnson-Hilliard M, Martinez KM, Janssen I, Edwards B, Mille ML, Zhang Y & Rogers MW. (2008). Lateral balance factors predict future falls in community-living older adults. *Archives of Physical Medical Rehabilitation* 89, 1708–1713.
- Kapoula Z, Gaertner C, Yang Q, Denise P & Toupet M (2013). Vergence and standing balance in subjects with idiopathic bilateral loss of vestibular function. *Plos One* (8)6, e66652.
- Kavounoudias A, Roll R & Roll JP (1998). The plantar sole is a ‘dynamometric map’ for human balance control. *NeuroReport* 9, 3247–3252.
- Kheradmand A, Lasker A & Zee DS (2015). Transcranial magnetic stimulation (TMS) of the supramarginal gyrus: a window to perception of upright. *Cerebral Cortex*, 25(3), 765-771.
- Knellwolf TP, Burton AR, Hammam E & Macefield VG (2018). Microneurography from the posterior tibial nerve: a novel method of recording activity from the foot in freely standing humans. *Journal of Neurophysiology*, 120(3), 953-959.
- Krafczyk S, Schlamp V, Dieterich M, Haberhauer P & Brandt T (1999). Increased body sway at 3.5–8 Hz in patients with phobic postural vertigo. *Neuroscience Letters* 259,149–15.
- Lafond, D., Duarte, M., & Prince, F. (2004). Comparison of three methods to estimate the center of mass during balance assessment. *Journal of biomechanics*, 37(9), 1421-1426.
- Latash ML, Ferreira SS, Wieczorek SA & Duarte M (2003). Movement sway: changes in postural sway during voluntary shifts of the center of pressure. *Experimental Brain Research* 150, 314-24.
- Lebar N, Danna J, Moré S, Mouchnino L & Blouin J (2017). On the neural basis of sensory

- weighting: Alpha, beta and gamma modulations during complex movements. *NeuroImage* 150, 200–212.
- Lim SB, Cleworth TW, Horslen BC, Blouin JS, Inglis JT, Carpenter MG (2017). Postural threat influences vestibular-evoked muscular responses *Journal of Neurophysiology* 117 , 604-611.
- Lhomond O, Teasdale N, Simoneau M & Mouchnino L (2016). Neural consequences of increasing body weight: evidence from somatosensory evoked potentials and the frequency-specificity of brain oscillations. *Frontiers in Human Neuroscience*, 10, 318.
- Lopez C & Blanke O (2011). The thalamocortical vestibular system in animals and humans. *Brain Research Reviews* 67, 119–146.
- Lopez C, Lacour M, Ahmadi A, El Magnan J & Borel L (2007). Changes of visual vertical perception: A long-term sign of unilateral and bilateral vestibular loss. *Neuropsychologia* 45, 2025–2037.
- Lord SR, Rogers MW, Howland A & Fitzpatrick R (1999). Lateral stability, sensorimotor function and falls in older people. *Journal of the American Geriatrics Society* 47, 1077–1081.
- Macefield VG (2005). Physiological characteristics of low-threshold mechanoreceptors in joints, muscle and skin in human subjects. *Clinical and experimental pharmacology & physiology* 32, 135 - 144.
- Macpherson JM, Everaert DG, Stapley PJ & Ting LH (2007). Bilateral vestibular loss in cats leads to active destabilization of balance during pitch and roll rotations of the support surface. *Journal of Neurophysiology* 97, 4357–4367.
- Massion J (1992). Movement, posture and equilibrium: interaction and coordination. *Progress in Neurobiology* 38, 35-56.
- Merfeld DM & Zupan LH (2002). Neural processing of gravito-inertial cues in humans. III. Modeling tilt and translation responses. *Journal of Neurophysiology* 87, 819–833.
- Mian OS & Day BL (2014). Violation of the craniocentricity principle for vestibularly evoked balance responses under conditions of anisotropic stability. *Journal of Neuroscience* 34, 7696-703.
- Morasso PG & Schieppati M (1999). Can muscle stiffness alone stabilize upright standing? *Journal of Neurophysiology* 82, 1622-1626.
- Mouchnino L & Blouin J (2013). When standing on a moving support, cutaneous inputs provide sufficient information to plan the anticipatory postural adjustments for gait initiation. *PLoS One* (8)2.

- Mouchnino L, Fontan A, Tandonnet C, Perrier J, Saradjian AH, Blouin J & Simoneau M (2015). Facilitation of cutaneous inputs during the planning phase of gait initiation. *Journal of Neurophysiology* 114, 301–308.
- Mouchnino L, Robert G, Ruget H, Blouin J & Simoneau M (2012). Online control of anticipated postural adjustments in step initiation: evidence from behavioral and computational approaches. *Gait & posture*, 35(4), 616-620.
- Mujunen T, Nurmi T, & Piitulainen H (2021). Corticokinematic coherence is stronger to regular than irregular proprioceptive stimulation of the hand. *Journal of Neurophysiology*, 126(2), 550-560.
- Murnaghan CD, Horslen BC, Inglis JT & Carpenter MG (2011). Exploratory behavior during stance persists with visual feedback. *Neuroscience* 195, 54-59.
- Neuper C, & Pfurtscheller G (2001). Event-related dynamics of cortical rhythms: frequency-specific features and functional correlates. *International Journal of Psychophysiology* 43:41-58.
- Padberg J, Cerkevich C, Engle J, Rajan AT, Recanzone G, Kaas J, & Krubitzer L (2009). Thalamocortical connections of parietal somatosensory cortical fields in macaque monkeys are highly divergent and convergent. *Cerebral Cortex*. 19(9):2038–2064.
- Pavlou M, Davies RA & Bronstein AM (2006). The assessment of increased sensitivity to visual stimuli in patients with chronic dizziness Marousa. *Journal of Vestibular Research* 16, 223–231.
- Pérennou DA, Mazibrada G, Chauvineau V, Greenwood R, Rothwell J, Gresty MA, Bronstein AM (2008). Lateropulsion, pushing and verticality perception in hemisphere stroke: a causal relationship? *Brain* 131: 2401–2413.
- Peterka RJ, Murchison CF, Parrington L, Fino PC & King LA (2018). Implementation of a central sensorimotor integration test for characterization of human balance control during stance. *Frontiers in neurology*, 9, 1045.
- Pfeiffer C, Serino A & Blanke O (2014). The vestibular system: A spatial reference for bodily self-consciousness. *Frontiers in Integrative Neuroscience* 8, 1–13.
- Pozzo T, Berthoz A, Vitte E & Lefort L (1991). Head stabilization during locomotion: perturbations induced by vestibular disorders. *Acta Oto-Laryngologica* 111, 322-327.
- Pfurtscheller G, Lopes da Silva FH (1999). Event-related EEG/MEG synchronization and desynchronization: basic principles. *Clinical Neurophysiology* 110, 1842-1857.
- Romei V, Rihs T, Brodbeck V & Thut G (2008). Resting electroencephalogram alpha-power over posterior sites indexes baseline visual cortex excitability. *Neuroreport* 19(2):203–

208.

- Ruzzoli M & Soto-Faraco S (2014). Alpha stimulation of the human parietal cortex attunes tactile perception to external space. *Current Biology* 24, 329–332.
- Sambo CF & Forster B (2011). Sustained Spatial Attention in touch: modality-specific and multimodal mechanisms. *The Scientific World Journal* 11, 199–213.
- Saradjian AH, Teasdale N, Blouin J & Mouchnino L (2019). Independent early and late sensory processes for proprioceptive integration when planning a step. *Cerebral Cortex* 29, 2353–2365.
- Saradjian AH, Tremblay L, Perrier J, Blouin J & Mouchnino L (2013). Cortical facilitation of proprioceptive inputs related to gravitational balance constraints during step preparation. *Journal of Neurophysiology* 110, 307-407.
- Sauseng P, Klimesch W, Gerloff C & Hummel FC (2009) Spontaneous locally restricted EEG alpha activity determines cortical excitability in the motor cortex. *Neuropsychologia* 47, 284-8.
- Sayenko DG, Vette AH, Obata H, Alekhina MI, Akai M & Nakazawa K (2009). Differential effects of plantar cutaneous afferent excitation on soleus stretch and H-reflex. *Muscle & Nerve: Official Journal of the American Association of Electrodiagnostic Medicine*, 39(6), 761-769.
- Staines WR, Brooke JD & McIlroy WE (2000). Task-relevant selective modulation of somatosensory afferent paths from the lower limb. *NeuroReport* 11, 1713-1719.
- Sathian K & Stilla R (2010). Cross-modal plasticity of tactile perception in blindness. *Restorative Neurology Neuroscience* 28, 271–281.
- Simoneau M, Tinker S, Hain T & Lee . (2003). Effects of predictive mechanisms on head stability during forward trunk perturbation. *Experimental brain research*, 148(3), 338-349.
- Stapley PJ, Ting LH, Hulliger M, Macpherson JM (2002). Automatic postural responses are delayed by pyridoxine-induced somatosensory loss. *Journal of Neuroscience* 22, 5803-7.
- Strzalkowski NDJ, Peters RM, Inglis JT & Bent LR (2018). Cutaneous afferent innervation of the human foot sole: What can we learn from single-unit recordings? *Journal of Neurophysiology* 120, 1233–1246.
- Tadel F, Baillet S, Mosher JC, Pantazis D, Leahy RM (2011). Brainstorm: a user-friendly application for MEG/EEG analysis. *Computational Intelligence Neuroscience* 879716.
- Thirunarayan MA, Kerrigan DC, Rabuffetti M, Della Croce U & Sainia M (1996). Comparison of three methods for estimating vertical displacement of center of mass during level walking in patients. *Gait & Posture* 4, 306-314.

- Toupet M, Van Nechel C, Hautefort C, Duquesne U, Heuschen S & Bozorg Grayeli A (2017). Subjective visual vertical in idiopathic bilateral vestibular hypofunction: enhanced role of vision, neck, and body proprioception. *Otology & Neurotology* 38, 1010-1016.
- Tsakiris M, Costantini M & Haggard P (2008). The role of the right temporo-parietal junction in maintaining a coherent sense of one's body. *Neuropsychologia* 46, 3014-3018.
- Vallbo AB & Johanson RS (1984). Properties of cutaneous mechanoreceptors in the human hand - related to touch sensation. *Human Neurobiology* 3, 3-14.
- van Ede F, Köster M & Maris E (2012). Beyond establishing involvement: Quantifying the contribution of anticipatory α - and β -band suppression to perceptual improvement with attention. *Journal of Neurophysiology*, 108, 2352–2362.
- Wang H, Callaghan E, Gooding-Williams G, McAllister C & Kessler K (2016). Rhythm makes the world go round: An MEG-TMS study on the role of right TPJ theta oscillations in embodied perspective taking. *Cortex* 75, 68 - 81.
- Ward BK, Agrawal Y, Hoffman HJ, Carey JP & Della Santina CC (2013). Prevalence and impact of bilateral vestibular hypofunction: Results from the 2008 US national health interview survey. *JAMA Otolaryngology - Head and Neck Surgery* 139, 803–810.
- Williams SR, Shenasa J & Chapman CE (1998). Time course and magnitude of movement-related gating of tactile detection in humans. I. Importance of stimulus location. *Journal of Neurophysiology*, 79(2), 947-963.
- Winter DA, Prince F, Frank JS, Powell C & Zabjek KL (1996). Unified theory regarding A/P and M/L balance in quiet stance. *J Neurophysiol* 75, 2334-2343.
- Zarzecki P, Blum PS, Bakker DA & Herman D (1983). Convergence of sensory inputs upon projection neurons of somatosensory cortex: vestibular, neck, head, and forelimb inputs. *Experimental brain research* 50-50)2-3. p.408.
- Zikopoulos B & Barbas H (2006). Prefrontal projections to the thalamic reticular nucleus form a unique circuit for attentional mechanisms. *Journal of Neuroscience*, 26(28):7348–7361
- Zingler VC, Weintz E, Jahn K, Mike A, Huppert D, Rettinger N, T Brandt & Strupp M (2008). Follow-up of vestibular function in bilateral vestibulopathy. *Journal of Neurology, Neurosurgery and Psychiatry* 79, 284–288.
- Zumer JM, Scheeringa R, Schoffelen JM, Norris DG & Jensen O (2014). Occipital Alpha Activity during Stimulus Processing Gates the Information Flow to Object-Selective Cortex. *PLoS Biology* 12(10). <https://doi.org/10.1371/journal.pbio.1001965>

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Competing Interests

None of the authors has any conflicts of interests on the submission form

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Figure legends

Figure 1: A. Biomechanical model. The model represents a double inverted pendulum with movement at the ankle and hip joints. Illustration of the weight bracelet wrapped around each wrist (bottom panel). **B.** Upper panel depicts maximal shear forces amplitude exerted by the left foot along the mediolateral axis and the lower panel presents the maximal value of the vertical forces under one foot. Simulations are for the three center of mass movement frequencies (i.e., 0.25, 0.5 and 0.75 Hz) and for three weight conditions (i.e., without weight, and with a 1 kg weight added at the wrist or at the ankle joints). The effects of frequencies and of weight positions on the modeled shear and vertical forces were the same for the other foot (not shown).

Figure 2: A. Grand average SEP traces for VH (n = 10) and Control (n= 10) participants in the No weight condition. The signals were aligned at stimulation onset (i.e., vertical dashed line). The upper and lower edges of the stimulation-induced artifact were cut. Inset: Position of the stimulating electrodes underneath the left foot. **B.** Averaged Weight and No weight conditions SEP amplitude of each participant of the VH and Control groups. The values are ranked in ascending order for each group. Group (10 Controls and 10 VH) means amplitude of the P₁N₁ SEP evoked by the electric stimulation for the No Weight and Weight conditions (error bars depict standard error of the means). **C.** Healthy group means amplitude of the P₁N₁ SEP evoked by the electric stimulation during the second experimental session for a younger group (39 years old) solely tested in the No weight condition (error bars depict standard deviation across participants).

Figure 3: A. Location of the regions of interest (ROIs) on the anatomical MRI Colin 27 brain template used to compute cortical activations. Similar ROIs were defined for the left and right Parietal Posterior Cortex (rPPC). **B.** Time-frequency power (ERS/ERD) of the signals by means of a complex Morlet's wavelet transform applied on the ROIs for each trial of each VH participant then averaged across VH participants in the Weight and No weight conditions (left panel). Cooler colors indicate ERD (event-related desynchronization) while warmer colors indicate ERS (event-related synchronization). Mean difference between Weight and No weight conditions (right panel). Frequency bands up to 90 Hz are illustrated to present changes in brain electrocortical activities for delta, theta, alpha, beta and gamma bands. The artifact caused by

the electric stimulation is present at stimulation onset. We epoched the data from -100 to 200 ms to emphasize changes in brain electrocortical activities (ERS/ERD) near plantar sole electrical stimulation. **C.** Group means power for the Alpha (8-12 Hz) and Beta (15-29 Hz) frequency bands computed during [50; 100 ms] time window for the No weight and Weight conditions (error bars depict standard error of the mean) over the left and right PPC.

Figure 4: **A.** Upper panel) Pelvis and head acceleration time-series, along the mediolateral (ML) and antero-posterior (AP) axes, for one representative participant in each of the Control and VH groups for the No weight condition. Bottom panel) mean of the integration of the pelvis acceleration along the mediolateral (ML) and antero-posterior (AP) directions for each participant for the No weight and the Weight conditions (error bars depict standard error of the mean). **B.** Pelvis and head accelerations for all participants in ML and AP directions. **C.** Group means integration of head acceleration along the mediolateral (ML) and anteroposterior (AP) directions for the No weight and Weight conditions (error bars depict standard error of the mean)

Table 1: VH participants' characteristics. Negative values are for the maximal slow phase velocity when nystagmus are beating on opposite sides as expected. Ocular (o) and cervical (c) Vestibular Evoked Myogenic Potential (VEMP) Tests (A absent and N normal). The Tullio phenomenon is defined as vertigo that occurs as a result of extremely high acoustic stimuli. The diagnostic of vestibular migraine was made according to the International Classification of Headache Disorders. The test were administered at the clinic one year before the experimental session.

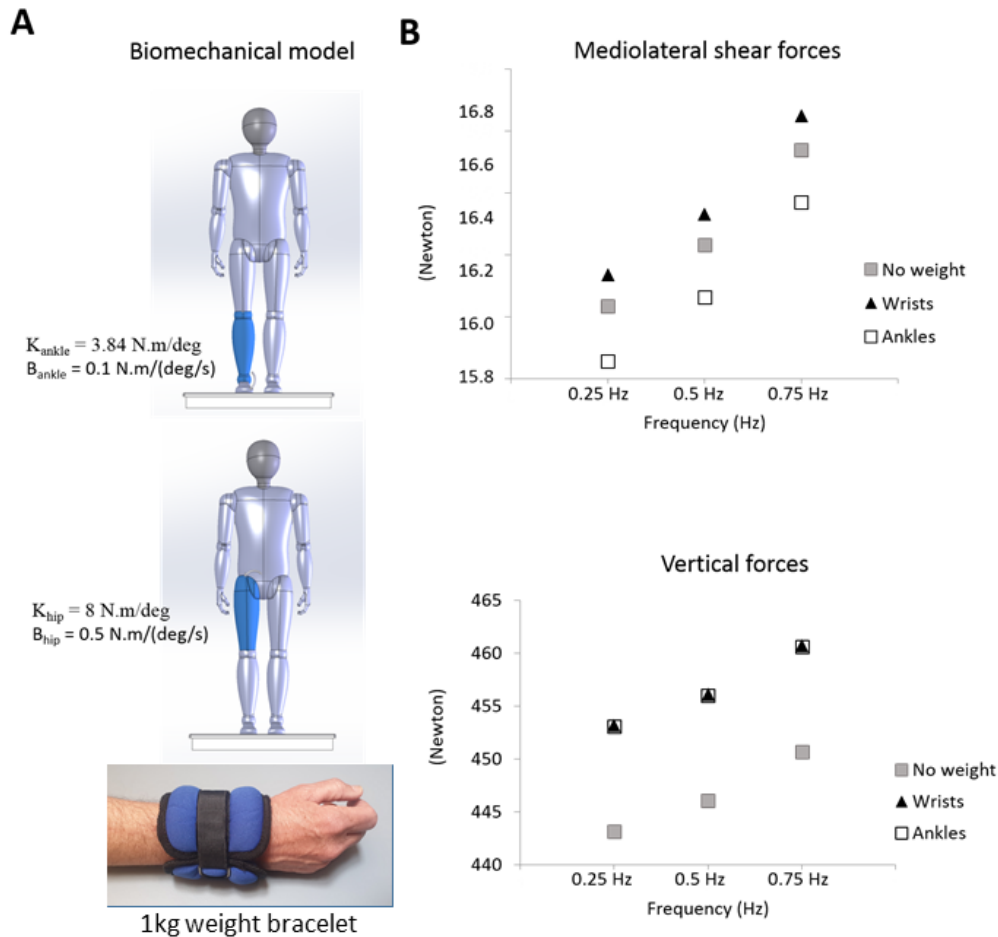


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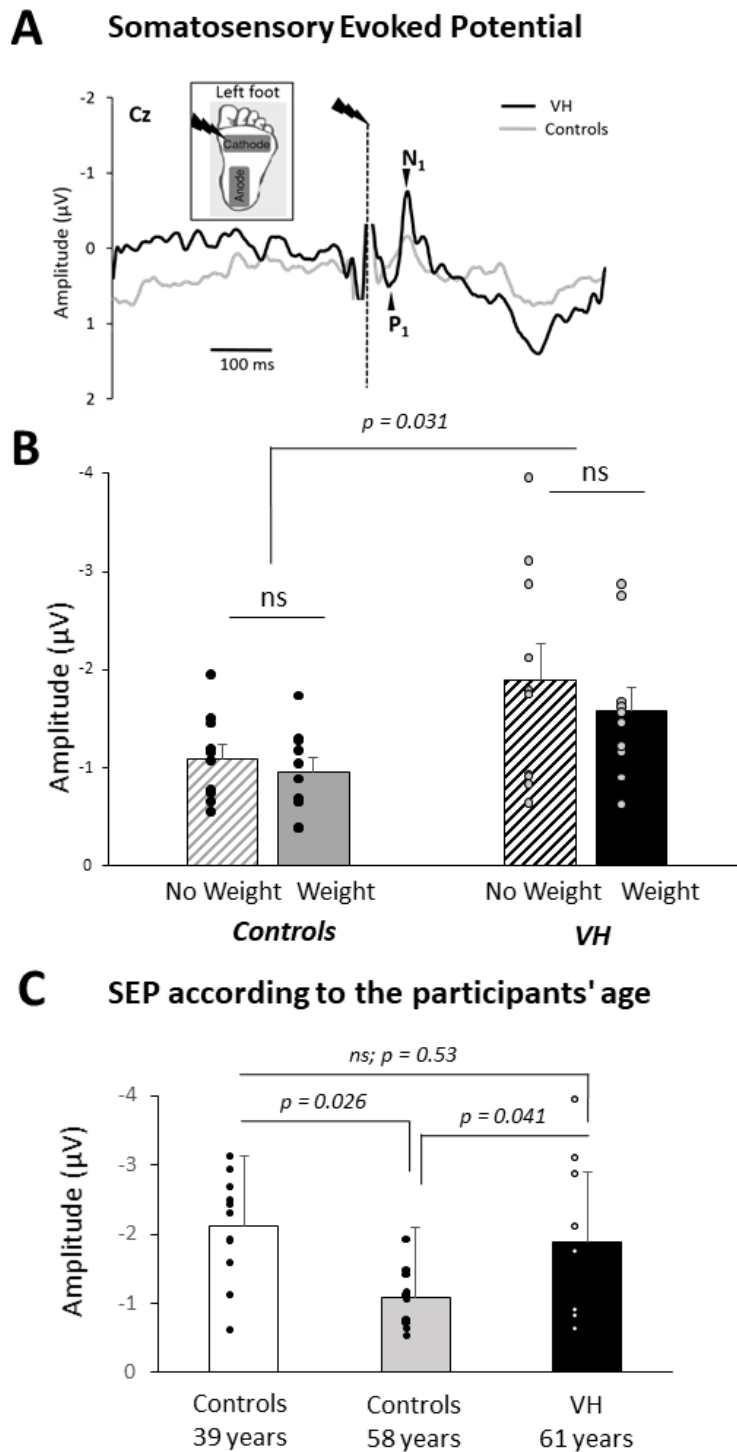


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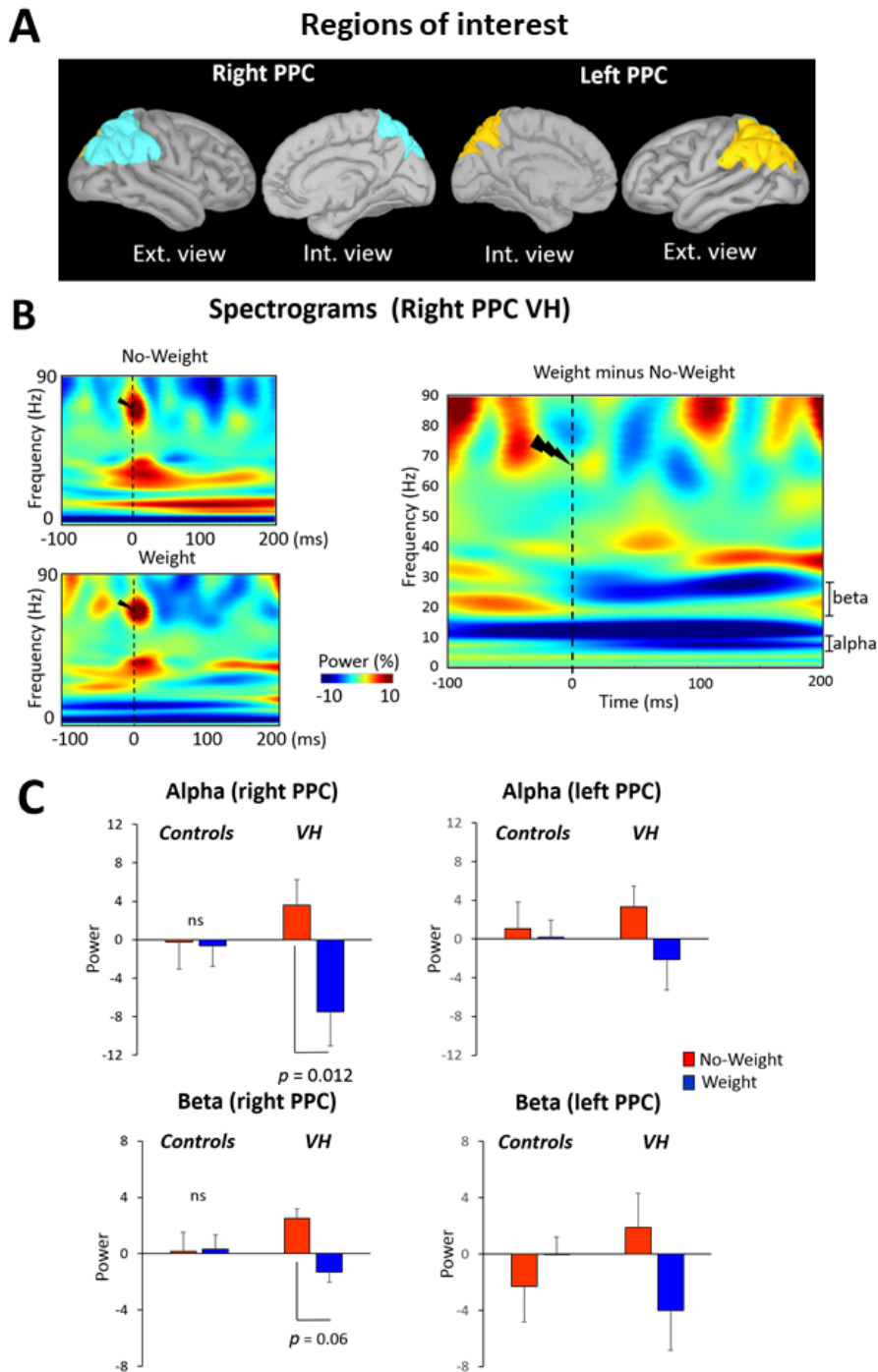


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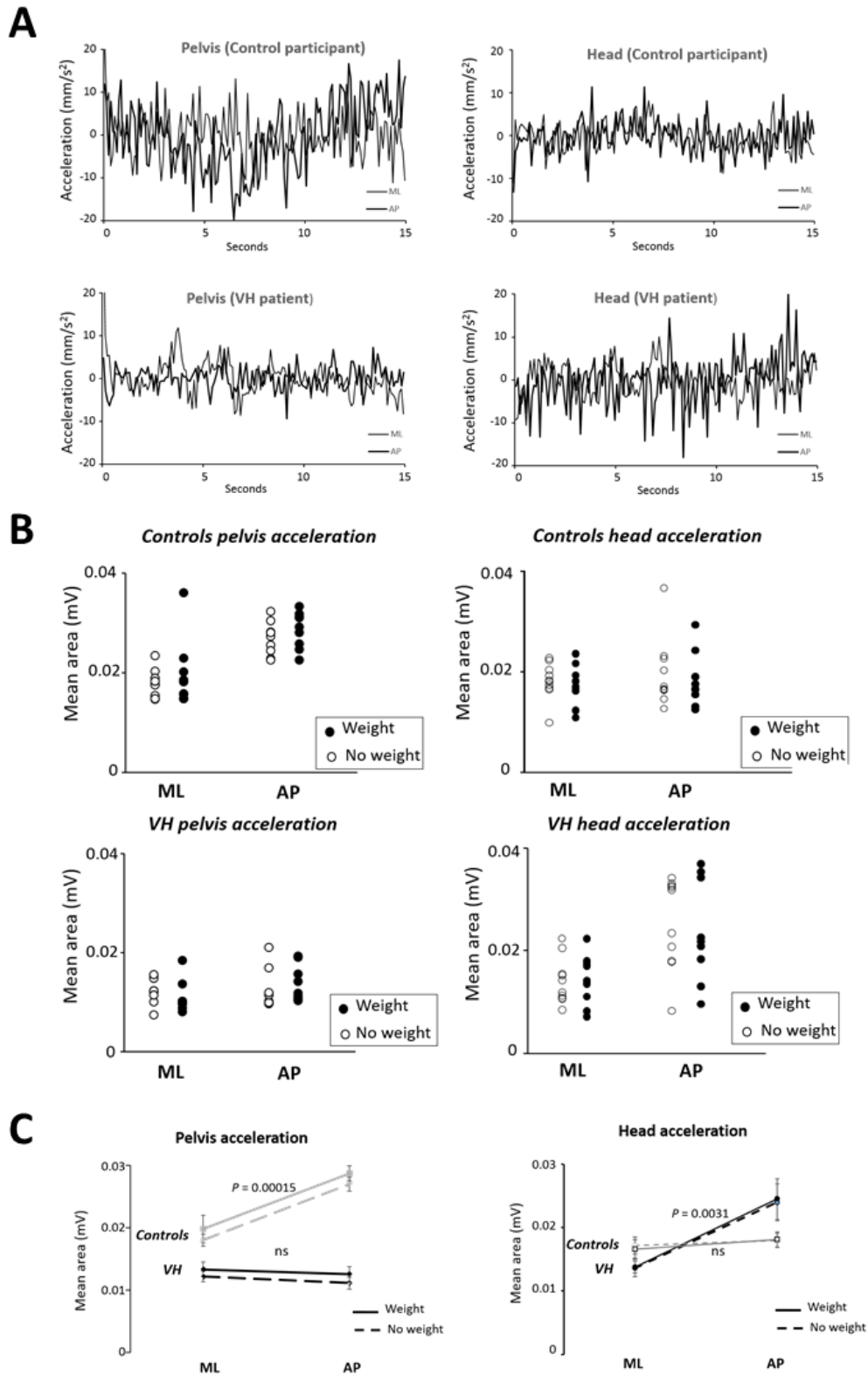


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VH	Age	Gender	Installation mode	Years of disease	Tullio	Migraines	vestibular caloric testing						Vestibulo-ocular reflex (VOR) gain						oVEMP		cVEMP	
							maximal slow phase velocity of post caloric nystagmus		sum		Right		Left		Right		Left		Right	Left	Right	Left
							Left 44°C	Right 44°C	Left 30°C	Right 30°C	Left	Right	Sup	Horiz	Post	Sup	Horiz	Post	Right	Left	Right	Left
1	62	M	Acute	9	0	0	0	2	1	-1	1	1	0.38	0.26	0.33	0.72	0.49	0.18	A	N	N	A
2	56	M	subacute after surgery	20	0	0	3	0	0	3	3	3	0.1	0.1	0.1	0.1	0.1	0.1	A	A	A	A
3	48	M	progressive	7	0	1	0	0	0	0	0	0	0.1	0.4	0.1	0.1	0.5	0.5	N	A	N	A
4	71	M	progressive	10	1	1	-3	3	6	-4	0	-1	0.42	0.48	0.59	1	0.9	0.55	A	N	N	N
5	56	M	subacute righth then left	2	0	0	0	0	0	0	0	0	0.12	0.04	0.16	0.08	0.23	0.16	A	A	A	A
6	82	F	progressive	7	0	0	0	0	0	0	0	0	0.24	0.12	0.31	0.13	0.07	0.18	N	N	N	N
7	59	M	progressive	20	0	0	0	0	0	0	0	0	1	0.86	0.89	1	0.2	0.52	A	N	N	N
8	45	M	Acute	4	0	0	3	3	3	3	6	6	0.69	0.6	0.24	0.53	0.57	0.51	N	N	N	N
9	79	F	progressive	7	0	0	0	0	0	0	0	0	0.64	0.19	0.14	0	0.32	0.44	A	A	N	N
10	48	F	Acute	7	0	0	0	0	0	0	0	0	0.9	0.9	0.9	0.62	0.55	0.7	A	A	N	N

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