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Body sway during postural perturbations is mediated by the degree of vestibulo-cortical dominance

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Dear Editor,

Maintenance of posture requires the integration of mutlisensory (vestibular, visual and proprioceptive) cues [1,2]. Contributions to postural control were historically noted to primarily implicate subcortical, spinal and cerebellar structures. More recent work, predominantly using electroencephalography (EEG), has unequivocally demonstrated the role of the cerebral cortex [3,4].

We have recently demonstrated that during increasingly demanding postural tasks, alpha power decreases proportionately with the subjective rating of task difficulty in central and parietal regions. Furthermore, the reduction in alpha power that was observed revealed an asymmetric distribution, such that it was greater in the right hemisphere. This asymmetric distribution implies involvement of vestibular cortical processing areas in the non-dominant hemisphere during active postural control [5]. However, to date the exact contribution that the lateralisation of the vestibular cortex plays upon maintaining postural control remains to be elucidated.

Accordingly, here we investigated the relationship between vestibulo-cortical dominance and body sway measures during postural perturbations. To address this, we implemented our biomarker of vestibulo-cortical hemispheric dominance, reflected by the degree of vestibular nystagmus suppression following transcranial direct current stimulation (tDCS) over the left PPC (cathodal stimulation) [6] and related this measure to changes in body sway (pre/post tDCS).

We recruited 24 right-handed participants (16 males, mean age: 21.6 years), that had no history or active neurological, ophthalmological, otological and psychiatric disorder. No subject had any brain-stimulation contraindications. All subjects provided written informed consent.

tDCS (neuroConn GMBH, Ilmenau, Germany) was applied using a current of 1.5mA for 15min. Two stimulation conditions were applied, either cathodal (test condition) or anodal (control) stimulation over the left PPC (P3; 10–20 international EEG classification; electrode placement area 25cm^2; reference electrode placed on the ipsilateral shoulder). The current had a ramp-up time of 10s at which point a constant current (1.5mA) was applied for 15 minutes. Current ramped down in a 10s fade-out period [6–8].

To assess postural control, participants stood blindfolded on a linear sled [9] perpendicular to the direction of motion (Fig. 1A). The stimulus consisted of a combination of 4 harmonically unrelated sine waves of different frequencies (0.18, 0.37, 0.69 and 0.9 Hz) delivering leftward and rightward oscillations for 30 s in a fixed pseudo-random sequence (Fig. 1B). Objective measures of body sway included left and right upper trunk displacement with respect to the platform, and hip level right and left angular velocities (Fig. 1A).

To establish vestibulo-cortical dominance participants underwent caloric stimulation of both the right and left ear separately with cold water (i.e. baseline measure of vestibular nystagmus). They then received either cathodal or anodal tDCS over the left PPC, randomised across participants. Following tDCS, the caloric irrigations were repeated to assess the mean percentage change in slow phase eye velocity induced by the tDCS. The degree of suppression of vestibular nystagmus induced by cortical inhibition of the left PPC reflects the degree of vestibulo-cortical hemispheric dominance [6], quantified by calculating the nystagmus suppression index using the following formula: subtracting the peak post-tDCS SPV from the peak pre-tDCS SPV divided by the peak pre-tDCS SPV $\times$ 100. Note, we only expected to observe a marked nystagmus suppression following cathodal but not anodal stimulation, with the latter specifically implemented as an active control for any non-specific effects associated with electrical stimulation [6–8].

We observed a significant suppression of the slow phase eye velocity during left cathodal stimulation of the PPC (mean reduction 51.71%, s.d. 18.91) when compared to left anodal stimulation (mean reduction 11.03%, s.d. 16.82) (F:11.73, $p < 0.01$; repeated measures ANOVA). Regarding the sway analysis during postural perturbations, we observed no differences between the pre/post tDCS sway path or velocity measures between both conditions (F:0.55, p:0.46 for sway path and F:1.34, p:0.253 for sway velocity, repeated measures ANOVA).

To specifically assess whether an individual's body sway was related to the degree of vestibulo-cortical dominance, we correlated the NSI, with measures of sway path and velocity change after tDCS. We observed a significant negative relationship between the degree of hemispheric dominance and the overall sway path (r: 0.721, $p < 0.01$) and velocity (r: 0.65, $p < 0.05$) only in the cathodal group. To split the analysis separately for right and left translations, we took the initial position of the subject for each trace as “centre”, and the traces were then divided into either rightward or leftward

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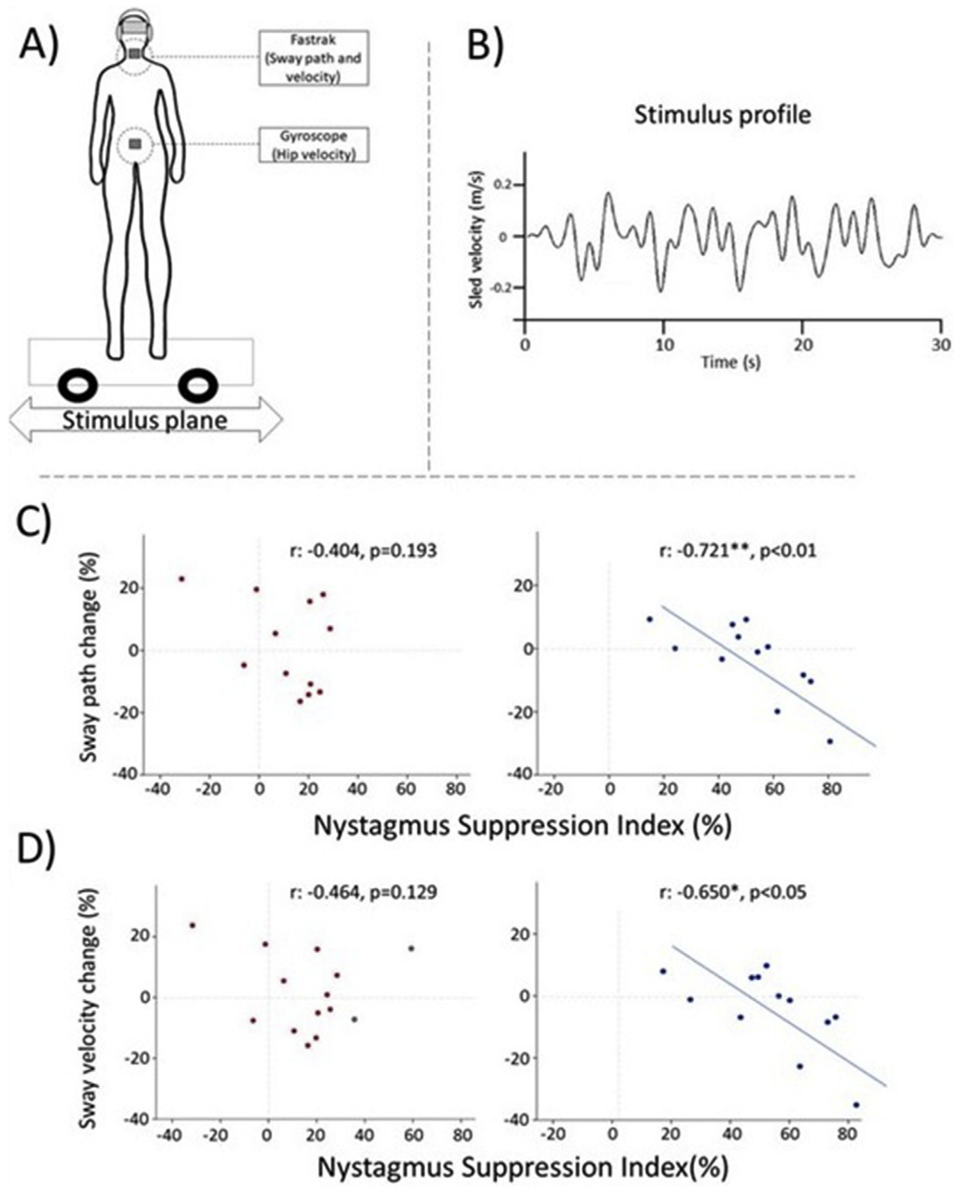

Fig. 1. 1A) Platform task; Subjects stood sideways to the sled displacement and had two movement sensors placed in order to record body movement. One sensor was placed on the upper trunk and the other at the hip. Subjects were blindfolded and wore earmuffs to avoid visual or auditory cues. 1B) Stimulus profile used to drive the sled movement depicted as a velocity profile. The stimulus consisted of a combination of 4 harmonically unrelated sinewaves of different frequencies (0.18, 0.37, 0.69 and 0.9 Hz). The first and last second of the stimulus were tapered to zero to avoid sudden onsets and stops. This stimulus-induced left and right oscillations for 30 s in a fixed pseudo-random sequence. Finally, the waveform was amplified using a linear potentiometer at 3 times the original waveform amplitude to generate trials of differing velocities (range 0–0.2 m/s). 1C and D) Correlation between the NSI and the overall (left and right) sway path(1C) and velocity (1D) percentage change in both the anodal (red dots) and cathodal groups (blue dots). A significantly negative correlation was observed only in the cathodal group, illustrating that in individuals with a greater nystagmus suppression index swayed less (1C) and with a slower velocity (1D) after tDCS.

movements from centre. The same correlation was present for both rightward (r: 0.79, $p < 0.01$ for sway path and r: 0.66, $p < 0.05$ for sway velocity) and leftward (r: 0.59, $p < 0.05$ for sway path and r: 0.62, $p < 0.05$ for sway velocity) perturbations respectively. No significant correlations were observed in the anodal stimulation group (r: 0.40, p:0.193 for sway path and r: 0.46, p:0.13 for sway velocity) (Fig. 1C and D).

Our findings illustrate a reduction in sway path and velocity, after cathodal stimulation over the left PPC, which was correlated with an individual's degree of vestibulo-cortical hemispheric dominance. Accordingly, those individuals with a greater right hemispheric cortical representation for gravito-inertial processing swayed less and with a reduced velocity.

Specifically, we propose that rightward body perturbations result in a leftward sway, shifting spatial attention leftwards mediated by the right hemisphere. Since left hemisphere cathodal stimulation inhibits the left PPC, individuals who are more right-hemisphere dominant will be able to better compensate for such inhibition. For leftward platform perturbations, the body sways rightward and shifts spatial attention rightwards mediated by the left hemisphere. Thus, inhibition of the left hemisphere had a greater un-stabilising effect upon postural control in less right hemisphere dominant individuals. An alternative explanation is that participants with a higher right hemispheric vestibulo-cortical dominance favour the use of gravito-inertial cues, hence sway less, a notion supported by previous lesion data [10].

To conclude, our results illustrate how vestibular-cortical dominance impacts upon postural control. These findings have potential clinical implications particularly for neurological patients with balance and gait disorders in whom their impairment could be influenced by the degree of vestibular cortical dominance and the localization of any cortical lesion.

Conflicts of interest statement

We wish to confirm that there are no known, either financial or personal with other people or organisations, conflicts of interest associated with this publication that could have influenced the outcome of this study.

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