JNeurosci
THE JOURNAL OF NEUROSCIENCE

Research Articles | Behavioral/Cognitive

A “conscious” loss of balance: Directing attention to movement can impair the cortical response to postural perturbations.

https://doi.org/10.1523/JNEUROSCI.0810-24.2024

Received: 23 April 2024
Revised: 20 August 2024
Accepted: 26 September 2024

Copyright © 2024 Parr et al.
This is an open-access article distributed under the terms of the Creative Commons Attribution 4.0 International license, which permits unrestricted use, distribution and reproduction in any medium provided that the original work is properly attributed.

This Early Release article has been peer reviewed and accepted, but has not been through the composition and copyediting processes.The final version may differ slightly in style or formatting and will contain links to any extended data.

Alerts: Sign up at www.jneurosci.org/alerts to receive customized email alerts when the fully formatted version of this article is published.

A “conscious” loss of balance: Directing attention to movement can impair the cortical response to postural perturbations.

Johnny V. V. Parr[1], Richard Mills[1], Elmar Kal[2], Adolfo M. Bronstein[3] & Toby J. Ellmers[3].

[1]Manchester Metropolitan University Institute of Sport, Manchester, United Kingdom.

[2]Centre for Cognitive and Clinical Neuroscience, Department of Health Sciences, College of Health, Medicine, and Life Sciences, Brunel University London, Uxbridge, UK.

[3]Centre for Vestibular Neurology, Department of Brain Sciences, Imperial College London, United Kingdom.

Short title: Conscious balance control impairs the cortical N1.

Co-corresponding authors:

Dr Johnny V. V. Parr (johnnyvvparr@gmail.com), Manchester Metropolitan University Institute of Sport, Manchester, United Kingdom.

Dr Toby J. Ellmers (t.ellmers@imperial.ac.uk), Centre for Vestibular Neurology, Department of Brain Sciences, Imperial College London, United Kingdom.

Manuscript info: *24 pages, 7 figures*

Word count: abstract = 241, introduction = 642, discussion = 1341

Conflict of interest: none

Acknowledgements: none.

JNeurosci Accepted Manuscript

Abstract

‘Trying too hard’ is known to interfere with skilled movement, such as sports and music playing. Postural control can similarly suffer when conscious attention is directed towards it (termed ‘conscious movement processing’; CMP). However, the neural mechanisms through which CMP influences balance remain poorly understood. We explored the effects of CMP on electroencephalographic (EEG) perturbation-evoked cortical responses and subsequent balance performance. Twenty healthy young adults (age=25.1±5 years; 10 males and 10 females) stood on a force plate-embedded moveable platform whilst mobile EEG was recorded. Participants completed two blocks of 50 discrete perturbations, containing an even mix of slower (186 mm/s peak velocity) and faster (225 mm/s peak velocity) perturbations. One block was performed under conditions of CMP (i.e., instructions to consciously control balance), whilst the other was performed under ‘Control’ conditions with no additional instructions. For both slow and fast perturbations, CMP resulted in significantly smaller cortical N1 signals (a perturbation-evoked potential localised to the supplementary motor area), and lower sensorimotor beta EEG activity 200–400 ms post-perturbation. Significantly greater peak velocities of the centre of pressure (i.e., greater postural instability) were also observed during the CMP condition. Our findings provide the first evidence that disruptions to postural control during CMP may be a consequence of insufficient cortical activation relevant for balance (i.e., insufficient cortical N1 responses followed by enhanced beta suppression). We propose that conscious attempts to minimise postural instability through CMP acts as a cognitive dual-task that dampens the sensitivity of the sensorimotor system for future losses of balance.

Keywords: balance, perturbation, EEG, N1, posture, kinetics, conscious control.

JNeurosci Accepted Manuscript

48 **Significance statement:**

49 'Trying too hard' is known to interfere with skilled movement, such as sports and
50 music playing. Postural control can also paradoxically worsen when individuals direct
51 conscious attention towards maintaining balance. Yet, the brain mechanisms
52 underpinning the counterproductive effects of such conscious movement processing
53 (CMP) remain unclear. Here, we show that impaired postural control when engaging
54 in CMP is expressed by a reduction in the evoked cortical signal following a
55 perturbation to balance. These findings imply that conscious attempts to minimise
56 postural instability may act as a cognitive dual-task that dampens the sensitivity of
57 the sensorimotor system for future losses of balance.

58

JNeurosci Accepted Manuscript

Introduction

When our movements fail us – or when we worry that they might – motor control can become a conscious, effortful process (Masters and Maxwell 2008). In sport and music instrument playing this is usually referred as 'trying too hard'. This is especially true for balance, where movement failure can have catastrophic consequences to health. Whilst engaging in such conscious movement processing (CMP) can occasionally be adaptive (Clark 2015), the control of balance – and of motor skills more generally (Baumeister 1984; Parr, Gallicchio, and Wood 2023) – typically suffers if too much conscious attention is directed towards it (Boisgontier et al. 2017; Kal, Young, and Ellmers 2022; Uiga et al. 2020). However, the neural mechanisms underpinning the maladaptive effects of CMP upon balance remain unclear.

Following an external balance perturbation, the central nervous system triggers rapid (~100 ms) brainstem-mediated postural responses (Horak 2006; Jacobs and Horak 2007; Welch and Ting 2008). This is followed by a negative electroencephalographic (EEG) cortical response (the 'N1' evoked potential) across the supplementary motor area ~100-200 ms after perturbation onset (Marlin et al. 2014; Varghese, McIlroy, and Barnett-Cowan 2017). The N1 is greater when facing larger perturbations (Payne, Hajcak, and Ting 2019), when a corrective step is required to avoid falling (Payne and Ting 2020a; Solis-Escalante et al. 2021; Zaback et al. 2023), when a perturbation is unexpected (Adkin et al. 2006), and in individuals with poorer balance abilities (Payne and Ting 2020b). Researchers have therefore proposed that the N1 acts as an error detection mechanism that is "primed" for (i) detecting centre of mass movements that approach one's limits of stability and (ii)

JNeurosci Accepted Manuscript

predicting the need for compensatory (i.e., stepping) behavioural responses (Payne and Ting 2020a; Solis-Escalante et al. 2021; Zaback et al. 2023).

The N1 can be influenced by “cognitive processes such as greater perceived threat or *attention to balance*, which have the potential to influence subsequent motor control” (Payne and Ting 2020b). Indeed, decreased cortical N1 amplitudes occur when attention is directed *away* from balance via a cognitive dual-task (Little and Woollacott 2015; Quant et al. 2004). In contrast, greater cortical N1 amplitudes occur when stance is perturbed during conditions which are known to increase attention *towards* balance (e.g. postural threat (Adkin et al. 2008; Zaback et al. 2023). However, these changes in CMP have co-occurred with increases in physiological arousal and/or cognitive loading, making it difficult to isolate the neural mechanisms through which CMP disrupts postural performance. The primary aim of this study is to therefore explore the direct effects of increased CMP on the cortical N1 response and subsequent postural control performance.

Engaging in CMP is thought to increase the general sensitivity of the sensorimotor system (or ‘vigilance’) to balance (Ellmers and Kal 2023; Ellmers, Kal, and Young 2021; Harris, Wilkinson, and Ellmers 2023), and may therefore influence pre- and post-perturbation cortical activities beyond the N1. For example, CMP could drive changes in EEG beta activity, given evidence that lower pre-perturbation beta supports perceptual sensitivity towards somatosensory signals (Mirdamadi, Ting, and Borich 2024; Shin et al. 2017), and that higher post-perturbation beta activity may reflect increased cortical engagement towards balance recovery following the N1 response (Ghosn et al. 2020; Palmer et al. 2021). Engaging in CMP can also evoke heightened EEG alpha activity across the visual cortex (Parr et al. 2023; Sherman et al. 2021), which may support the vigilance towards somatosensory

JNeurosci Accepted Manuscript

processing by down-weighting visual processing (Gallicchio and Ring 2020; Jensen and Mazaheri 2010). Despite these findings, the specific role of CMP upon beta and alpha activity remains unknown.

We hypothesised that under conditions of increased CMP we would observe greater cortical N1 amplitudes, lower pre-perturbation beta power, and greater pre-perturbation occipital alpha power, when compared to control conditions where no specific attentional instructions are provided. As directing conscious attention to movement is known to disrupt postural control in healthy young adults (Boisgontier et al. 2017), we also predicted that balance would become impaired during conditions of CMP.

Materials and methods

Participants

Twenty neurotypical young adults participated in the experiment (10 females, 10 males; *M*±*SD* age = 25.1±*5.0* yrs; height = 173.30±*11.17* cm; weight = 74.30±*10.81* kg). Sample size estimates were based on the medium (d = 0.71) to large effects (d = 0.82) reported upon the cortical N1 under conditions that indirectly manipulate CMP (e.g., heightened postural threat (Adkin et al. 2008) and divided attention (Little and Woollacott 2015). Assuming a medium-to-large effect size (d = 0.71), a minimum sample size of 18 participants was required to yield 80% power with an alpha level of p = 0.05 when comparing mean differences between two related groups (calculated using G*POWER software 3.1; Henrich University Dusseldorf, Germany). All participants were free from any neurological disease and had no prior experience of dizziness or balance problems. The experiment was approved by the Manchester Metropolitan University institutional ethics committee (project ID #56055).

JNeurosci Accepted Manuscript

Protocol

Perturbations were delivered via a bespoke moveable platform (80 x 60 cm with an embedded force plate recording at 1000 Hz; Type 9281B, Kistler Instrument Corp., Winterthur, Switzerland). The platform was driven by an electromagnetic actuator and controlled through custom written software (Labview v19 SP1, National Instruments, Austin, Texas) via DAQ card (USB-6210, National Instruments). Participants stood on the force plate, with their feet shoulder-width apart and their hands on their hips. Foot positioning was marked to ensure consistency between trials and conditions (i.e., participants could return to the same position between trial blocks, or in the event a step was taken as response to the perturbation). During the trials, participants were instructed to fixate on a cross marked on the wall at eye level, four metres away.

Participants experienced two blocks of 50 discrete sine-wave perturbations (7-15s random delay between each perturbation) consisting of an initial forward translation of the support surface (maximum forward displacement = 70 mm) before reversing direction and completing the sinewave to return to original position. Each 10-min block consisted of 50 perturbations: 25 fast (0.5 Hz, peak acceleration = 1883 mm/s^2, peak acceleration latency = 60 ms) and 25 slow (0.3 Hz, peak acceleration = 1277 mm/s^2, peak acceleration latency = 60 ms), presented in a pseudo-random order. For the purpose of this study, we focused only on the initial forward portion of the perturbation (see Figure 1) to not risk contamination of EEG data with the return of the sine-wave perturbation. Perturbations were therefore predictable in amplitude (70 mm max forward displacement) and direction (i.e., forwards), but unpredictable in terms of both speed and timing, as perturbations were delivered every 7-15 seconds. To further maximise the unpredictability of stimulus presentation,

JNeurosci Accepted Manuscript

participants wore noise-isolating headphones to minimise any anticipatory audio cues. Both perturbation stimuli (fast and slow) were designed to challenge postural stability but small enough to not necessitate a correcting stepping response. To prevent fatigue, participants received a 5–10-minute break after each block of trials. To define the onset of platform perturbations, we recorded the kinematics of a reflective marker placed on the platform at a frequency of 100 Hz using a 10-camera motion analysis system (Qualisys v2021.1, Gothenburg, Sweden). The "findpeaks" function in MATLAB was used to identify the forward peaks (i.e., peak forward displacement) in the platform's forward-backward position vector. We then utilised the “ischange” function in MATLAB to identify the moment at which an abrupt change in the vector’s acceleration profile first occurred in the 1-second of data prior to each peak.

Attentional focus manipulation

As we sought to explore how CMP affects the neural control of balance when stance is perturbed, one block (of 50 trials) was performed under conditions designed to induce CMP; whilst the other block was performed under ‘control’ conditions (no other instructions provided aside from the general task instructions). For the CMP condition, participants were instructed to consciously monitor their postural stability between each perturbation (“focus your attention towards how the weight is distributed beneath your feet”) and minimise any movement in their ankles. These instructions were based on qualitative research that has explored what participants direct their attention towards when CMP (spontaneously) occurs during postural control (Zaback, Carpenter, and Adkin 2016). Prompts and reminders were delivered to ensure that participants maintained this focus of attention throughout the block of

JNeurosci Accepted Manuscript

trials. The presentation order of conditions (CMP vs. Control) was counterbalanced across participants.

After each block of trials, participants completed a 4-item questionnaire that assessed the extent to which they directed conscious attention towards their balance during the previous set of trials (e.g., “I am always trying to think about my balance when I am doing this task”; 1 = strongly disagree; 6 = strongly agree; Ellmers et al. 2021; Ellmers and Young 2018). This questionnaire served as a manipulation check. Scores from the four separate items were summed to produce a total score of state CMP. To assess any carry-over effects (i.e., order effects) of performing the CMP condition first, we performed post-hoc independent t-tests to compare state CMP between participants who performed either the Control or CMP condition first. Results showed no difference between groups for the Control condition ($t(18) = .518$, $p = .611$), the CMP condition ($t(18) = .767$, $p = .453$), or the change scores between conditions ($t(18) = .446$, $p = .661$). After each condition of trials, participants also completed a visual analogue scale that ranged from 0 (“not at all anxious”) to 10 (“the most anxious I have ever felt”) to rate the level of state anxiety that they felt during the preceding trials (Castro et al. 2019). Higher scores therefore indicate greater state anxiety. These self-reported assessments were used to confirm that the CMP manipulation led to the intended increase in state CMP, whilst verifying that any results observed were not confounded by any between-condition differences in state anxiety.

EEG recording and analyses

The EEG signals were recorded at 1000 Hz from 29 active shielded AgCl electrodes embedded in a stretchable fabric cap (eego sports, ANT Neuro, Hengelo,

JNeurosci Accepted Manuscript

Netherlands) positioned according to the extended 10–20 international system (Jurcak, Tsuzuki, and Dan 2007). Electrodes in sites CPz and AFz were used as reference and ground, respectively. Nasion, Inion, and preauricular points were used as anatomical landmarks to position the EEG cap. Conductive gel for electrophysiological measurements was used (Signa gel, Parker), and impedance was kept below 20 kΩ. The EEG and forceplate (see below) signals were synchronized through a square-wave trigger upon the initiation of an experimental recording.

EEG signals were band-pass filtered using the EEGLAB “basic FIR filter (new)” (1–45Hz, 3300 filter order, −6 dB cutoff frequency, 1 Hz transition bandwidth) prior to being cut into epochs ranging from −1 to +2 s relative to perturbation onset and re-referenced to the average of all scalp electrodes. These epochs were visually inspected for large EEG contamination from muscular artifacts, but no trials were discarded. No bad EEG channels were identified. Independent component analysis (ICA) weights were obtained separately for each condition through the RunICA infomax algorithm (Jung et al. 1998) running on EEG signals. ICA weights that presented obvious non neural activity upon visual inspection (e.g., eyeblinks, line noise, muscular artifact) were manually rejected. On average, we retained 25.9 ± 1.1 and 25.9 ± 1.7 components across the CMP and Control conditions, respectively. Following visual inspection, we then identified the brain component that gave rise to a distinct cortical N1. Consistent with other studies, N1 components were localised across the supplementary motor area (Marlin et al. 2014; Varghese et al. 2017), with a midfrontal topography consistent across all participants and across the two experimental conditions (Control and CMP; Figure 2). For visualisation purposes only, cortical N1 sources were further mapped onto a standard MNI template and

JNeurosci Accepted Manuscript

estimated using the DIPFIT plugin (coarse fit; Klug and Gramann 2021; Oostenveld and Oostendorp 2002). Estimated cortical locations and percentage of power accounted for by the cortical N1 components can be found in Extended Data Table 2-1. To assess spectral characteristics of the selected cortical N1 component and EEG channel data, we performed time-frequency decomposition via trial-by-trial convolution with complex Morlet wavelets. We used 44 frequencies linearly spaced between 2 and 45 Hz, with wavelets logarithmically spaced from 5 to 8 cycles. All processing steps were performed using EEGLAB (v2020.0) functions (Delorme and Makeig 2004) for MATLAB.

Pre-perturbation EEG measures. For pre-perturbation activity, decomposed power spectra of the selected cortical N1 component and EEG channel-level data were averaged from -1000 to -50 ms relative to perturbation onset. The FOOOF (Fitting Oscillations & One-Over-F) algorithm (Donoghue et al. 2020) was then used to decompose the averaged power spectra into aperiodic (1/f) and periodic components (activity above 1/f) from 4 to 30 Hz using the following parameters: max number of peaks = 4, minimum peak height = 0.1, peak threshold = 2, aperiodic module = fixed). Peak periodic beta (15 – 30 Hz) and peak periodic alpha (8 – 12 Hz) were extracted from the fitted spectra. If more than one peak was detected, values were averaged across the peaks. Since the width of periodic peaks can vary, we also extracted the area under the spectral curve (AUC; see Ref. (Mirdamadi et al. 2024). As pre-perturbation beta and alpha oscillatory activities were calculated *prior* to the perturbation onset, values were averaged across both fast and slow trials within a given condition (CMP versus Control) to increase statistical power. Changes in broadband 1/f activity of the cortical N1 component were also assessed by extracting the aperiodic slope and aperiodic offset using the FOOOF algorithm.

JNeurosci Accepted Manuscript

Post-perturbation EEG analyses. To assess the cortical N1 response, we extracted single trial N1 amplitudes from the selected N1 component (see Figure 2). However, given that analytical approaches vary across the literature (with some studies analysing the N1 component (e.g., Mirdamadi et al. 2024; Solis-Escalante et al. 2021) and others focusing only on channel Cz (e.g., Payne and Ting 2020b; Varghese et al. 2017; Zaback et al. 2023), we also performed parallel N1 analyses on channel Cz to confirm whether our findings were robust across component versus channel level analyses. Time series data were baseline subtracted (-150 to -50 ms before perturbation onset) for each participant, and the N1 was quantified as the largest negative peak occurring 50-200 ms after perturbation onset. For each participant, N1 amplitudes were subsequently averaged across fast and slow perturbations separately for both the CMP and Control conditions. We also calculated event-related spectral power (ERSP) of both the cortical N1 component and EEG channel level data by dividing decomposed time-frequency data by the average activity from -1000 to -500 ms prior to perturbation across all conditions and trials (i.e., neutral baseline across conditions) before performing a 10*log10 transformation (i.e., decibel change). We then extracted the average beta activity (15 – 30 Hz) between 200 to 400 ms post-perturbation from the selected cortical N1 component as an index of cortical engagement in balance recovery *following* the cortical N1 response (Ghosn et al. 2020; Palmer et al. 2021). We again performed parallel analyses of post-perturbation beta activity on channel Cz to confirm whether our findings were robust across component versus channel level analyses. For the purpose of visualisation, grand average ERSP of channel Cz are presented in Figure 3.

JNeurosci Accepted Manuscript

Postural control analyses.

We used custom MATLAB scripts to determine the peak velocity of centre of pressure (COP) data in response to the initial forward portion of the perturbation. As we used a forwards-moving perturbation, we restricted analysis to the anterior-posterior (AP) direction. Peak backwards COP velocity was selected as our outcome variable as it is a direction-specific response to the initial forward perturbation; greater backwards CoP velocity generally indicates greater instability and higher risk of falling (Hewson et al. 2010; Masani et al. 2014). First, for each event we selected and low-pass filtered (5 Hz, 2nd order bidirectional Butterworth filter) a 3-second AP-COP trace that spanned 2000 ms pre-perturbation and 1000 ms post-perturbation. We then corrected this trace for offset using the estimated average AP COP displacement during the 'baseline' period (based on the 1100-100 ms pre-perturbation window). Peak velocity of the postural response to the perturbation was then identified as the first negative peak in the derivative of the AP-COP trace in the initial forward portion of the perturbation (Figure 1). By default, the initial negative peak was selected unless a subsequent peak was of >50% greater magnitude than the earlier peak. The mean latency to peak velocity (termed 'peak latency') for slow perturbations were 219 ms (*SD* = 29, range = 166-278) and 217 ms (*SD* = 27, range = 164-271) for Control and CMP conditions, respectively. The mean peak latencies for fast perturbations were 213 ms (*SD* = 23, range = 172-260) and 212 ms (*SD* = 21, range = 173-258) for Control and CMP conditions, respectively.

Statistical analyses.

The Gaussian distribution of data were checked via Shapiro-Wilk test of normality. Paired samples t-tests were therefore used to determine differences between

JNeurosci Accepted Manuscript

attention conditions (CMP vs Control) for self-reported conscious processing, self-reported anxiety, pre-perturbation peak beta and beta AUC, aperiodic exponent, and aperiodic offset. For the N1 amplitude, post-perturbation beta activity, and for peak AP COP velocity, we performed a two-way repeated measures analysis of variance (ANOVA) with perturbation speed (slow vs fast) and condition (CMP vs Control) as within-subject factors. However, as data for peak AP-COP velocity during the control condition were significantly non-normally distributed (p = .035), we first performed a log-transformation of AP velocity data prior to ANOVA. Pearson's correlations were then performed to determine any association between N1 amplitude and AP velocity. To explore topographical differences between conditions in pre-perturbation beta and alpha AUC, we performed channel-wise paired samples t-tests (i.e., one t-test for each channel pair). The multiple comparisons problem (i.e., one test per channel/pixel) was solved by applying the false discovery rate (FDR) to obtained p-values. ANOVA effect sizes were reported using partial eta squared (ηp^2), common indicative thresholds for which are small (0.01), medium (0.06) and large (0.14; (Field 2013). All statistical analyses were performed using IBM SPSS statistics (version 26) with an alpha level of 0.05.

Results

Attentional focus manipulation checks

Participants reported directing significantly greater conscious attention towards their balance in the CMP (M = 14.50, SD = 4.02) compared to Control condition (M = 11.80, SD = 5.45, t = -4.61, p <.001, d = 0.56), confirming the effectiveness of the CMP manipulation. There was no difference in state anxiety between conditions, with

JNeurosci Accepted Manuscript

low levels of anxiety experienced for both (Control, M = 1.95, SD = 1.76; CMP, M = 1.95, SD = 1.32, Z = -0.36, p = .971, r = 0.018).

N1 amplitude

Analysis of the cortical N1 component showed a significant main effect of perturbation speed, $F(1, 19) = 28.86$, $p < .001$, $\eta p^2 = .603$, with larger N1 amplitudes observed during fast compared to slow perturbations (irrespective of attentional focus condition). There was also a significant main effect of Attention condition, $F(1, 19) = 6.11$, $p = .023$, $\eta p^2 = .243$, with smaller N1 amplitudes observed in CMP compared to the Control condition (irrespective of the perturbation speed). On average, N1 amplitudes during the CMP condition were 8% smaller for fast perturbations and 10% smaller for slow perturbations, compared to Control. There was no Attention x Speed interaction, $F(1, 19) = 0.12$, $p = .737$, $\eta p^2 = .006$ (Figure 4). Consistent findings were observed when analyses were performed on channel Cz (rather than the N1 component). However, N1 amplitudes for channel Cz were approximately three times larger than the amplitudes of the N1 component (see Extended Data Figure 4-1). Individual N1 amplitudes from both the component and channel Cz analyses were also highly correlated ($rs > .92$), confirming the robustness of the results across component- and channel-level analyses (see Extended Data Figure 4-2). A detailed comparison of descriptive and inferential statistics from the component and channel Cz analyses is provided in Extended Data Table 4-1 and 4-2.

Postural control

There was a significant main effect of perturbation Speed ($F(19) = 274.683$, $p < .001$, $\eta p^2 = .935$), with greater peak AP velocities observed for fast compared to slow

JNeurosci Accepted Manuscript

perturbations. There was also a significant main effect of Attention condition (F(1, 19) = 7.915, p = .011, ηp^2 = .294) and a significant interaction between Attention and perturbation Speed (F(1, 19) = 9.109, p = .007, ηp^2 = .324). Post-hoc comparisons showed peak AP velocities to be significantly greater during the CMP condition compared to the Control condition for both fast (p = .047) and slow (p = .004) perturbations, with this effect more pronounced for the slow perturbations (Figure 4). For fast perturbations, Pearson's correlations also revealed a significant negative correlation between peak AP velocity and N1 amplitude for both the CMP (r = -.51, p = .022) and Control conditions (r = -.57, p = .008), whereby greater velocities were associated with smaller N1 amplitudes. The same relationship was observed for slow perturbations during both the CMP (r = -.64, p = .002) and Control (r = -.52, p = .016) conditions (see Figure 5).

Pre- and post-perturbation cortical activity*.* Paired t-tests revealed no difference in the cortical N1 component's pre-perturbation peak beta (t(19) = 0.62, p = .539, d = .14), beta AUC (t(19) = 0.67, p = .513, d = .14), aperiodic exponent (t(19) = 0.04, p = .970, d = .01), or aperiodic offset, t(19) = 0.89, p = .391, d = .20) between CMP and Control conditions. For EEG channel-level analyses, topographical analyses of pre-perturbation periodic beta and alpha activity revealed no channel-wise differences between conditions in peak or AUC values (Figure 6). For post-perturbation beta activity of the N1 component, the ANOVA revealed no main effect of Condition, F(1, 19) = 2.31, p = .144, np2 = .109, no main effect of perturbation Speed, F(1, 19) = 3.71, p = .069, np2 = .163, and no Condition x Speed interaction, F(1, 19) = 0.01, p = .976, np2 = .000. However, for post-perturbation beta activity of channel Cz, the ANOVA showed a significant main effect of Condition, F(1, 19) = 4.45, p = .048, np2 = .190, with lower beta activity during the CMP condition compared to the Control

JNeurosci Accepted Manuscript

condition, particularly for the slower perturbations (Figure 7). There was neither a significant main effect of perturbation Speed, $F(1, 19) = 1.44$, $p = .244$, np2 = .071, nor Condition x Speed interaction, $F(1, 19) = 3.68$, $p = .070$, np2 = .162.

Discussion

We explored how directing conscious attention towards balance affects the cortical control of posture during discrete perturbations to quiet stance. Our findings revealed that the cortical N1 – a neural signal involved in monitoring postural instability and mobilising compensatory balance-correcting responses (Payne and Ting 2020a; Solis-Escalante et al. 2021; Zaback et al. 2023) – was significantly smaller during conditions of experimentally-induced CMP. Behaviourally, this was coupled with greater peak COP velocity during the CMP condition, indicating greater postural instability. Although effective postural control requires some degree of attentional resources (Boisgontier et al. 2017; Woollacott and Shumway-Cook 2002), directing *too much* attention towards balance can disrupt postural control – much like how athletic performance breaks-down when experts adopt a self-focus (Baumeister 1984; Parr et al. 2023; Smith et al. 2003). The present findings provide the first evidence that such maladaptive effects of CMP on postural control appear to be expressed by insufficient activation at the cortex relevant for postural control.

Previous work has reported larger N1 signals during conditions of increased postural threat (Adkin et al. 2008; Zaback et al. 2023), and reduced N1s when performing a cognitive dual-task (Little and Woollacott 2015; Quant et al. 2004). Although not a direct manipulation of CMP, individuals will reliably direct greater conscious attention towards movement when their balance is threatened and they become anxious/fearful about falling (Ellmers et al. 2023; Huffman et al. 2009;

INeurosci Accepted Manuscript

Zaback et al. 2016). Conversely, individuals will direct *less* attention towards balance during conditions of dual-task (Ellmers et al. 2021; Johnson et al. 2020). We therefore expected larger N1 amplitudes under conditions of CMP that was induced *independent* of postural threat, and that this result would reflect an increased sensitivity of the sensorimotor system for responding to postural disturbances during self-focused attention (Harris et al. 2023). Self-report data confirmed that our manipulation was successful at isolating CMP from perceived threat/anxiety. However, contrary to our prediction, we observed significantly *smaller* N1 responses under conditions of CMP. This reduction in N1 amplitudes (average reduction of ~9%) is akin to reductions previously reported during conditions of cognitive dual-task (between ~5-20% reduction; Little and Woollacott 2015; Quant et al. 2004). This suggests that the larger N1 amplitudes observed previously during conditions of increased postural threat (which is known to induce CMP) may instead reflect threat-related increases in mental vigilance or arousal, rather than changes in attention to movement (Zaback et al. 2023). Indeed, emotional arousal has also been shown to modulate the amplitude of the N1 in non-motor (i.e. cognitive) tasks (Luna et al. 2023).

Researchers have proposed that the N1 – which is localised to the supplementary motor area – acts as an instability and/or error detection mechanism that is "primed" for (i) detecting centre of mass movements that approach one's limits of stability and (ii) mobilising compensatory stepping responses (Payne and Ting 2020a; Solis-Escalante et al. 2021; Zaback et al. 2023). Supporting this stance, the present findings showed that the cortical N1 scales with perturbation intensity, with greater N1 amplitudes observed during the fast (compared to slow) perturbation. Our findings also revealed larger N1 amplitudes in individuals with poorer within-task

JNeurosci Accepted Manuscript

balance performance (i.e., greater peak COP velocity; Figure 5), which aligns with previous work showing larger N1 responses in individuals with poorer *generalised* balance ability (Payne and Ting 2020b). Collectively, these findings support the notion that the cortical N1 amplitude reflects the allocation of cognitive resources towards compensatory balance-correcting responses (Payne and Ting 2020a). The reduction in N1 amplitudes observed during conditions of CMP therefore likely reflects a maladaptive process. Indeed, on group level, these reductions were accompanied by disruptions in postural performance (increased peak COP velocity – and hence greater disturbance – in response to the perturbation). We are unable to draw causal inferences between the reduction in N1 and the subsequently disrupted postural control in the present work. However, as the N1 occurred on average 68 ms (*SD* = 24 ms) *before* peak instability (see grand averages presented in Figure 4), the neural processes underpinning the N1 response may have directly influenced subsequent balance performance.

CMP, by definition, is a 'conscious' process, meaning that it requires attentional resources (Ellmers and Young 2018). Engaging in this form of motor control can therefore act like a cognitive dual-task and limit the resources available for processing other tasks or information (Parr et al. 2023; Uiga et al. 2018). During the CMP condition, participants were instructed to consciously monitor their postural stability and minimise ankle movement during the pre-perturbation period. We suggest therefore that individuals were so focused on consciously minimising instability during the pre-perturbation period that they became less able to flexibly shift attentional resources towards processing the perturbation itself, resulting in a maladaptively smaller N1 and disrupted postural response. In other words, conscious attempts to maximise stability *prior* to a loss of balance acts like a

JNeurosci Accepted Manuscript

cognitive dual-task that reduces the attentional resources available for processing the instability and then behaviourally responding once the loss of balance itself occurs (Little and Woollacott 2015; Quant et al. 2004). We therefore propose that conscious attempts to minimise postural instability in a given moment serves to dampen the sensitivity of the sensorimotor system for *future* losses of balance, via disruptions to the 'central set' (the nervous system's ability to prepare itself for upcoming sensory information and movement (Horak, Diener, and Nashner 1989)).

However, the effect of CMP upon the cortical N1 may differ across balance-impaired populations for whom CMP reflects a compensatory strategy to overcome poorer (and less 'automatic') balance (Boisgontier et al. 2017; Clark 2015; Kal et al. 2022). For instance, it is possible that older adults with fear of falling may instead use CMP proactively in a way that enhances, rather than disturbs, the central set (see Ellmers et al. 2023). Future work should therefore look to extend these findings beyond healthy young adults. Nonetheless, these findings provide the evidence that, in neurotypical young adults with relatively good balance control, CMP may disrupt postural control via insufficient compensatory activation at the cortex in response to perturbations.

Contrary to our prediction, the CMP manipulation had no effect on pre-perturbation oscillatory alpha or beta activity. Within the context of balance, lower pre-perturbation beta EEG activity of the cortical N1 component is associated with *enhanced* perception of the subsequent perturbation to balance (Mirdamadi et al. 2024), suggesting that lower beta activity may reflect a more sensitive sensory processing system. Given that CMP is proposed to increase perceptual sensitivity for postural disturbances (Ellmers et al. 2021; Harris et al. 2023), we had expected CMP would thus lower pre-perturbation beta. In line with previous research (Parr et al.

JNeurosci Accepted Manuscript

2023; Sherman et al. 2021), we had also expected CMP to promote elevated alpha activity across the visual cortex, possibly reflecting a mechanism that supports vigilance to somatosensory processing by down-weighting visual processing through regional inhibition (Jensen and Mazaheri 2010). However, no differences in pre-perturbation alpha or beta activity were observed, which suggests that our CMP manipulation did not alter ongoing perceptual sensitivity prior to postural disturbances. Previous research has also reported higher post-N1 beta activity in individuals with poorer balance (Palmer et al. 2021), and when experiencing larger perturbations (Ghosn et al. 2020), suggesting a (conscious) compensatory role for such neural activity. However, we instead observed significantly *larger* reductions in post-perturbation beta activity during CMP irrespective of perturbation size. Whilst the functional role of sensorimotor beta oscillations is still not fully understood (Barone and Rossiter 2021; Spitzer and Haegens 2017), researchers have proposed that reductions in beta activity during an ongoing action may reflect a "decrease in somatosensory responsiveness for the efficient unfolding of the movement" (p. 22, Kilavik et al. 2013). The reduced beta activity we observed during the late recovery phase of the perturbation could therefore reflect a continued dampening of the sensorimotor system (i.e. beyond the initial cortical N1 response) when engaging in CMP. Previous researchers have consistently proposed CMP to enhance, rather than dampen, sensorimotor sensitivity during postural control (Ellmers et al. 2021; Harris et al. 2023), but our findings question this interpretation of CMP. It is also important to note that this finding was restricted to the channel-level (i.e., Cz) analyses, suggesting these post-perturbation features were not captured by the single component that contributes to the cortical N1. Future research should look to

JNeurosci Accepted Manuscript

504 further scrutinise the specific mechanisms through which CMP alters post-
505 perturbation beta activity.

506 ***Conclusions***

507 Our findings revealed that directing conscious attention towards balance
508 significantly reduced the size of the cortical N1. As this was coupled with poorer
509 postural control, this reduced cortical response is likely maladaptive in nature. We
510 therefore provide evidence that the maladaptive effects of CMP upon balance may
511 be driven by insufficient activation at the cortex relevant for postural control. We
512 propose that conscious attempts to minimise postural instability in a given moment
513 acts as a cognitive dual-task that serves to dampen the sensitivity of the
514 sensorimotor system for *future* losses of balance. These findings provide novel
515 insight into the neural mechanisms underpinning the maladaptive behavioural effects
516 of 'trying too hard' during motor performance.

517

JNeurosci Accepted Manuscript

Reference list

Adkin, Allan L., Adam D. Campbell, Romeo Chua, and Mark G. Carpenter. 2008. 'The Influence of Postural Threat on the Cortical Response to Unpredictable and Predictable Postural Perturbations'. *Neuroscience Letters* 435(2):120–25. doi: 10.1016/j.neulet.2008.02.018.

Adkin, Allan L., Sylvia Quant, Brian E. Maki, and William E. McIlroy. 2006. 'Cortical Responses Associated with Predictable and Unpredictable Compensatory Balance Reactions'. *Experimental Brain Research* 172(1):85–93. doi: 10.1007/s00221-005-0310-9.

Barone, Jacopo, and Holly E. Rossiter. 2021. 'Understanding the Role of Sensorimotor Beta Oscillations'. *Frontiers in Systems Neuroscience* 15. doi: 10.3389/fnsys.2021.655886.

Baumeister, Roy F. 1984. 'Choking under Pressure: Self-Consciousness and Paradoxical Effects of Incentives on Skillful Performance'. *Journal of Personality and Social Psychology* 46(3):610–20. doi: 10.1037/0022-3514.46.3.610.

Boisgontier, Matthieu P., Boris Cheval, Sima Chalavi, Peter van Ruitenbeek, Inge Leunissen, Oron Levin, Alice Nieuwboer, and Stephan P. Swinnen. 2017. 'Individual Differences in Brainstem and Basal Ganglia Structure Predict Postural Control and Balance Loss in Young and Older Adults'. *Neurobiology of Aging* 50:47–59. doi: 10.1016/j.neurobiolaging.2016.10.024.

Castro, Patricia, Diego Kaski, Marco Schieppati, Michael Furman, Qadeer Arshad, and Adolfo Bronstein. 2019. 'Subjective Stability Perception Is Related to Postural Anxiety in Older Subjects'. *Gait and Posture* 68:538–44. doi: 10.1016/j.gaitpost.2018.12.043.

Clark, David J. 2015. 'Automaticity of Walking: Functional Significance, Mechanisms, Measurement and Rehabilitation Strategies'. *Frontiers in Human Neuroscience* 9.

Delorme, Arnaud, and Scott Makeig. 2004. 'EEGLAB: An Open Source Toolbox for Analysis of Single-Trial EEG Dynamics Including Independent Component Analysis'. *Journal of Neuroscience Methods* 134(1):9–21. doi: 10.1016/j.jneumeth.2003.10.009.

Donoghue, Thomas, Matar Haller, Erik J. Peterson, Paroma Varma, Priyadarshini Sebastian, Richard Gao, Torben Noto, Antonio H. Lara, Joni D. Wallis, Robert T. Knight, Avgusta Shestyuk, and Bradley Voytek. 2020. 'Parameterizing Neural Power Spectra into Periodic and Aperiodic Components'. *Nature Neuroscience* 23(12):1655–65. doi: 10.1038/s41593-020-00744-x.

Ellmers, Toby J., and Elmar C. Kal. 2023. 'Exploring the Role of Attention towards Balance in Chronic Dizziness: Development of the Balance Vigilance Questionnaire'. *European Journal of Neurology*. doi: 10.1111/ene.16148.

Ellmers, Toby J., Elmar C. Kal, and William R. Young. 2021. 'Consciously Processing Balance Leads to Distorted Perceptions of Instability in Older Adults'. *Journal of Neurology* 268(4):1374–84. doi: 10.1007/s00415-020-10288-6.

Ellmers, Toby J., Mark R. Wilson, Elmar C. Kal, and William R. Young. 2023. 'The Perceived Control Model of Falling: Developing a Unified Framework to Understand and Assess

INeuroSci Accepted Manuscript

559 Maladaptive Fear of Falling'. *Age and Ageing* 52(7):afad093. doi:
560 10.1093/ageing/afad093.

561 Ellmers, Toby J., and William R. Young. 2018. 'Conscious Motor Control Impairs Attentional
562 Processing Efficiency during Precision Stepping'. *Gait & Posture* 63:58–62. doi:
563 10.1016/j.gaitpost.2018.04.033.

564 Field, Andy. 2013. *Discovering Statistics Using IBM SPSS Statistics*. SAGE.

565 Gallicchio, Germano, and Christopher Ring. 2020. 'The Quiet Eye Effect: A Test of the
566 Visual and Postural-Kinematic Hypotheses'. *Sport, Exercise, and Performance*
567 *Psychology* 9(1):143–59. doi: 10.1037/spy0000162.

568 Ghosn, Nina J., Jacqueline A. Palmer, Michael R. Borich, Lena H. Ting, and Aiden M.
569 Payne. 2020. 'Cortical Beta Oscillatory Activity Evoked during Reactive Balance
570 Recovery Scales with Perturbation Difficulty and Individual Balance Ability'. *Brain*
571 *Sciences* 10(11):860. doi: 10.3390/brainsci10110860.

572 Harris, D. J., S. Wilkinson, and T. J. Ellmers. 2023. 'From Fear of Falling to Choking under
573 Pressure: A Predictive Processing Perspective of Disrupted Motor Control under
574 Anxiety'. *Neuroscience & Biobehavioral Reviews* 148:105115. doi:
575 10.1016/j.neubiorev.2023.105115.

576 Hewson, David J., Neeraj K. Singh, Hichem Snoussi, and Jacques Duchêne. 2010.
577 'Classification of Elderly as Fallers and Non-Fallers Using Centre of Pressure
578 Velocity'. Pp. 3678–81 in *2010 Annual International Conference of the IEEE*
579 *Engineering in Medicine and Biology*.

580 Horak, F. B., H. C. Diener, and L. M. Nashner. 1989. 'Influence of Central Set on Human
581 Postural Responses'. *Journal of Neurophysiology* 62(4):841–53. doi:
582 10.1152/jn.1989.62.4.841.

583 Horak, Fay B. 2006. 'Postural Orientation and Equilibrium: What Do We Need to Know about
584 Neural Control of Balance to Prevent Falls?' *Age and Ageing* 35(suppl_2):ii7–11. doi:
585 10.1093/ageing/afl077.

586 Huffman, J. L., B. C. Horslen, M. G. Carpenter, and A. L. Adkin. 2009. 'Does Increased
587 Postural Threat Lead to More Conscious Control of Posture?' *Gait & Posture*
588 30(4):528–32. doi: 10.1016/j.gaitpost.2009.08.001.

589 Jacobs, J. V., and F. B. Horak. 2007. 'Cortical Control of Postural Responses'. *Journal of*
590 *Neural Transmission* 114(10):1339–48. doi: 10.1007/s00702-007-0657-0.

591 Jensen, Ole, and Ali Mazaheri. 2010. 'Shaping Functional Architecture by Oscillatory Alpha
592 Activity: Gating by Inhibition'. *Frontiers in Human Neuroscience* 4. doi:
593 10.3389/fnhum.2010.00186.

594 Johnson, Kyle J., Alexander M. Watson, Craig D. Tokuno, Mark G. Carpenter, and Allan L.
595 Adkin. 2020. 'The Effects of Distraction on Threat-Related Changes in Standing
596 Balance Control'. *Neuroscience Letters* 716:134635. doi:
597 10.1016/j.neulet.2019.134635.

598 Jung, Tzyy-Ping, Scott Makeig, Anthony J. Bell, and Terrence J. Sejnowski. 1998.
599 'Independent Component Analysis of Electroencephalographic and Event-Related

JNeurosci Accepted Manuscript

Potential Data'. Pp. 189–97 in *Central Auditory Processing and Neural Modeling*, edited by P. W. F. Poon and J. F. Brugge. Boston, MA: Springer US.

Jurcak, Valer, Daisuke Tsuzuki, and Ippeita Dan. 2007. '10/20, 10/10, and 10/5 Systems Revisited: Their Validity as Relative Head-Surface-Based Positioning Systems'. *NeuroImage* 34(4):1600–1611. doi: 10.1016/j.neuroimage.2006.09.024.

Kal, Elmar C., William R. Young, and Toby J. Ellmers. 2022. 'Balance Capacity Influences the Effects of Conscious Movement Processing on Postural Control in Older Adults'. *Human Movement Science* 82:102933. doi: 10.1016/j.humov.2022.102933.

Kilavik, Bjørg Elisabeth, Manuel Zaepffel, Andrea Brovelli, William A. MacKay, and Alexa Riehle. 2013. 'The Ups and Downs of Beta Oscillations in Sensorimotor Cortex'. *Experimental Neurology* 245:15–26. doi: 10.1016/j.expneurol.2012.09.014.

Klug, Marius, and Klaus Gramann. 2021. 'Identifying Key Factors for Improving ICA-Based Decomposition of EEG Data in Mobile and Stationary Experiments'. *European Journal of Neuroscience* 54(12):8406–20. doi: 10.1111/ejn.14992.

Little, C. Elaine, and Marjorie Woollacott. 2015. 'EEG Measures Reveal Dual-Task Interference in Postural Performance in Young Adults'. *Experimental Brain Research* 233(1):27–37. doi: 10.1007/s00221-014-4111-x.

Luna, Fernando Gabriel, María Julieta Aguirre, Elisa Martín-Arévalo, Agustín Ibáñez, Juan Lupiáñez, and Pablo Barttfeld. 2023. 'Event-Related Potentials Associated with Attentional Networks Evidence Changes in Executive and Arousal Vigilance'. *Psychophysiology* 60(8):e14272. doi: 10.1111/psyp.14272.

Marlin, Amanda, George Mochizuki, William R. Staines, and William E. McIlroy. 2014. 'Localizing Evoked Cortical Activity Associated with Balance Reactions: Does the Anterior Cingulate Play a Role?' *Journal of Neurophysiology* 111(12):2634–43. doi: 10.1152/jn.00511.2013.

Masani, Kei, Albert H. Vette, Masaki O. Abe, and Kimitaka Nakazawa. 2014. 'Center of Pressure Velocity Reflects Body Acceleration Rather than Body Velocity during Quiet Standing'. *Gait & Posture* 39(3):946–52. doi: 10.1016/j.gaitpost.2013.12.008.

Masters, Rich, and Jon Maxwell. 2008. 'The Theory of Reinvestment'. *International Review of Sport and Exercise Psychology* 1(2):160–83. doi: 10.1080/17509840802287218.

Mirdamadi, Jasmine L., Lena H. Ting, and Michael R. Borich. 2024. 'Distinct Cortical Correlates of Perception and Motor Function in Balance Control'. *Journal of Neuroscience* 44(15). doi: 10.1523/JNEUROSCI.1520-23.2024.

Oostenveld, Robert, and Thom F. Oostendorp. 2002. 'Validating the Boundary Element Method for Forward and Inverse EEG Computations in the Presence of a Hole in the Skull'. *Human Brain Mapping* 17(3):179–92. doi: 10.1002/hbm.10061.

Palmer, Jacqueline A., Aiden M. Payne, Lena H. Ting, and Michael R. Borich. 2021. 'Cortical Engagement Metrics During Reactive Balance Are Associated With Distinct Aspects of Balance Behavior in Older Adults'. *Frontiers in Aging Neuroscience* 13. doi: 10.3389/fnagi.2021.684743.

Parr, Johnny V. V., Germano Gallicchio, Andrés Canales-Johnson, Liis Uiga, and Greg Wood. 2023. 'Cortical, Muscular, and Kinetic Activity Underpinning Attentional Focus

JNeurosci Accepted Manuscript

642 Strategies during Visuomotor Control'. *Psychophysiology* 60(6):e14249. doi:
643 10.1111/psyp.14249.

644 Parr, Johnny V.V., Germano Gallicchio, and Greg Wood. 2023. 'EEG Correlates of Verbal
645 and Conscious Processing of Motor Control in Sport and Human Movement: A
646 Systematic Review'. *International Review of Sport and Exercise Psychology*
647 16(1):396–427. doi: 10.1080/1750984X.2021.1878548.

648 Payne, Aiden M., Greg Hajcak, and Lena H. Ting. 2019. 'Dissociation of Muscle and Cortical
649 Response Scaling to Balance Perturbation Acceleration'. *Journal of Neurophysiology*
650 121(3):867–80. doi: 10.1152/jn.00237.2018.

651 Payne, Aiden M., and Lena H. Ting. 2020a. 'Balance Perturbation-Evoked Cortical N1
652 Responses Are Larger When Stepping and Not Influenced by Motor Planning'.
653 *Journal of Neurophysiology* 124(6):1875–84. doi: 10.1152/jn.00341.2020.

654 Payne, Aiden M., and Lena H. Ting. 2020b. 'Worse Balance Is Associated with Larger
655 Perturbation-Evoked Cortical Responses in Healthy Young Adults'. *Gait & Posture*
656 80:324–30. doi: 10.1016/j.gaitpost.2020.06.018.

657 Quant, Sylvia, Allan L. Adkin, W. Richard Staines, Brian E. Maki, and William E. McIlroy.
658 2004. 'The Effect of a Concurrent Cognitive Task on Cortical Potentials Evoked by
659 Unpredictable Balance Perturbations'. *BMC Neuroscience* 5(1):18. doi:
660 10.1186/1471-2202-5-18.

661 Sherman, David A., Tim Lehmann, Jochen Baumeister, Alli Gokeler, Luke Donovan, and
662 Grant E. Norte. 2021. 'External Focus of Attention Influences Cortical Activity
663 Associated With Single Limb Balance Performance'. *Physical Therapy*
664 101(12):pzab223. doi: 10.1093/ptj/pzab223.

665 Shin, Hyeyoung, Robert Law, Shawn Tsutsui, Christopher I. Moore, and Stephanie R. Jones.
666 2017. 'The Rate of Transient Beta Frequency Events Predicts Behavior across Tasks
667 and Species' edited by Y. Kajikawa. *eLife* 6:e29086. doi: 10.7554/eLife.29086.

668 Smith, Aynsley M., Charles H. Adler, Debbie Crews, Robert E. Wharen, Edward R.
669 Laskowski, Kelly Barnes, Carolyn Valone Bell, Dave Pelz, Ruth D. Brennan, Jay
670 Smith, Matthew C. Sorenson, and Kenton R. Kaufman. 2003. 'The "Yips" in Golf'.
671 *Sports Medicine* 33(1):13–31. doi: 10.2165/00007256-200333010-00002.

672 Solis-Escalante, Teodoro, Mitchel Stokkermans, Michael X. Cohen, and Vivian
673 Weerdesteyn. 2021. 'Cortical Responses to Whole-Body Balance Perturbations
674 Index Perturbation Magnitude and Predict Reactive Stepping Behavior'. *European*
675 *Journal of Neuroscience* 54(12):8120–38. doi: 10.1111/ejn.14972.

676 Spitzer, Bernhard, and Saskia Haegens. 2017. 'Beyond the Status Quo: A Role for Beta
677 Oscillations in Endogenous Content (Re)Activation'. *eNeuro* 4(4). doi:
678 10.1523/ENEURO.0170-17.2017.

679 Uiga, Liis, Catherine M. Capio, Donghyun Ryu, Mark R. Wilson, and Rich S. W. Masters.
680 2018. 'The Role of Conscious Control in Maintaining Stable Posture'. *Human*
681 *Movement Science* 57:442–50. doi: 10.1016/j.humov.2017.10.008.

682 Uiga, Liis, Catherine M. Capio, Donghyun Ryu, William R. Young, Mark R. Wilson, Thomson
683 W. L. Wong, Andy C. Y. Tse, and Rich S. W. Masters. 2020. 'The Role of Movement-

INeuroSci Accepted Manuscript

684 Specific Reinvestment in Visuomotor Control of Walking by Older Adults'. *The*
685 *Journals of Gerontology: Series B* 75(2):282–92. doi: 10.1093/geronb/gby078.

686 Varghese, Jessy Parokaran, Robert E. McIlroy, and Michael Barnett-Cowan. 2017.
687 'Perturbation-Evoked Potentials: Significance and Application in Balance Control
688 Research'. *Neuroscience & Biobehavioral Reviews* 83:267–80. doi:
689 10.1016/j.neubiorev.2017.10.022.

690 Welch, Torrence D. J., and Lena H. Ting. 2008. 'A Feedback Model Reproduces Muscle
691 Activity During Human Postural Responses to Support-Surface Translations'. *Journal*
692 *of Neurophysiology* 99(2):1032–38. doi: 10.1152/jn.01110.2007.

693 Woollacott, Marjorie, and Anne Shumway-Cook. 2002. 'Attention and the Control of Posture
694 and Gait: A Review of an Emerging Area of Research'. *Gait & Posture* 16(1):1–14.
695 doi: 10.1016/S0966-6362(01)00156-4.

696 Zaback, Martin, Mark G. Carpenter, and Allan L. Adkin. 2016. 'Threat-Induced Changes in
697 Attention during Tests of Static and Anticipatory Postural Control'. *Gait & Posture*
698 45:19–24. doi: 10.1016/j.gaitpost.2015.12.033.

699 Zaback, Martin, Kyle J. Missen, Allan L. Adkin, Romeo Chua, J. Timothy Inglis, and Mark G.
700 Carpenter. 2023. 'Cortical Potentials Time-Locked to Discrete Postural Events during
701 Quiet Standing Are Facilitated during Postural Threat Exposure'. *The Journal of*
702 *Physiology* 601(12):2473–92. doi: 10.1113/JP284055.

703

704

JNeurosci Accepted Manuscript

Figure Legends

Figure 1. (Left) Visual representation of the experimental task. Participants stood with eyes open and feet shoulder width apart on a moveable platform whilst wearing a mobile EEG system on their back. The platform would translate in the forward direction at two speeds with a consistent displacement. (Right) Line plots displaying the displacement, velocity, and acceleration of the initial forward platform translation for each perturbation speed (recorded via motion-capture marker and accelerometer placed on the platform).

Figure 2. (Top) Participant-specific scalp topographies of cortical N1 components for both the Control (top left) and CMP (top right) conditions. (Bottom) Cortical N1 sources mapped onto a standard Montreal Neurological Institute (MNI) template and estimated using the DIPFIT plugin. Estimated cortical locations and percentage of power accounted for by the cortical N1 components can be found in Extended Data Table 2-1.

Figure 3. Grand average event related spectral power of channel Cz across each experimental condition for both slow (A) and fast (B) perturbations.

Figure 4. Summary results for the N1 component's ERP and AP velocity for the slow (4A; top four panels) and fast perturbations (4B; bottom four panels). For each figure, separately presented are: Top left: Group-level perturbation evoked potentials, with the thick solid lines and shaded region of the ERP denoting mean and standard deviation, respectively; Top Right: N1 amplitudes for both the CMP and Control conditions, with the bars denoting group mean values and points denoting individual participant mean values; Bottom Left: Group-level AP velocity traces for both the

JNeurosci Accepted Manuscript

CMP and Control conditions, with thick solid lines and shaded region denoting mean and standard deviation, respectively; and Bottom Right: AP peak amplitudes, with the bars denoting group mean values and points denoting individual participant mean values. For all panels on the right, lines connect the mean values for each participant from the CMP to the Control condition. Asterisks denote a pairwise significant difference at the $p < .05$* and $p < .01$** levels. A detailed comparison of descriptive and inferential statistics of the cortical N1 amplitude derived from the component and channel Cz analyses is provided in Extended Figure 4-1 and 4-2, and in Extended Data Table 4-1 and 4-2.

Figure 5. Scatter plots denoting the Pearson's correlation between the amplitude of the N1 component and peak AP velocity for both slow (top row) and fast (bottom row) perturbations.

Figure 6. Scalp maps denoting the group mean values of pre-perturbation beta peak (top row) and alpha peak (bottom row) for the Control and CMP conditions, presented as normalised area under the spectral curve. The scalp maps furthest right denote the t-scores obtained through channel-wise paired comparisons, with red regions indicating greater power in the CMP compared to Control condition, and blue regions indicating greater power in the Control compared to CMP condition.

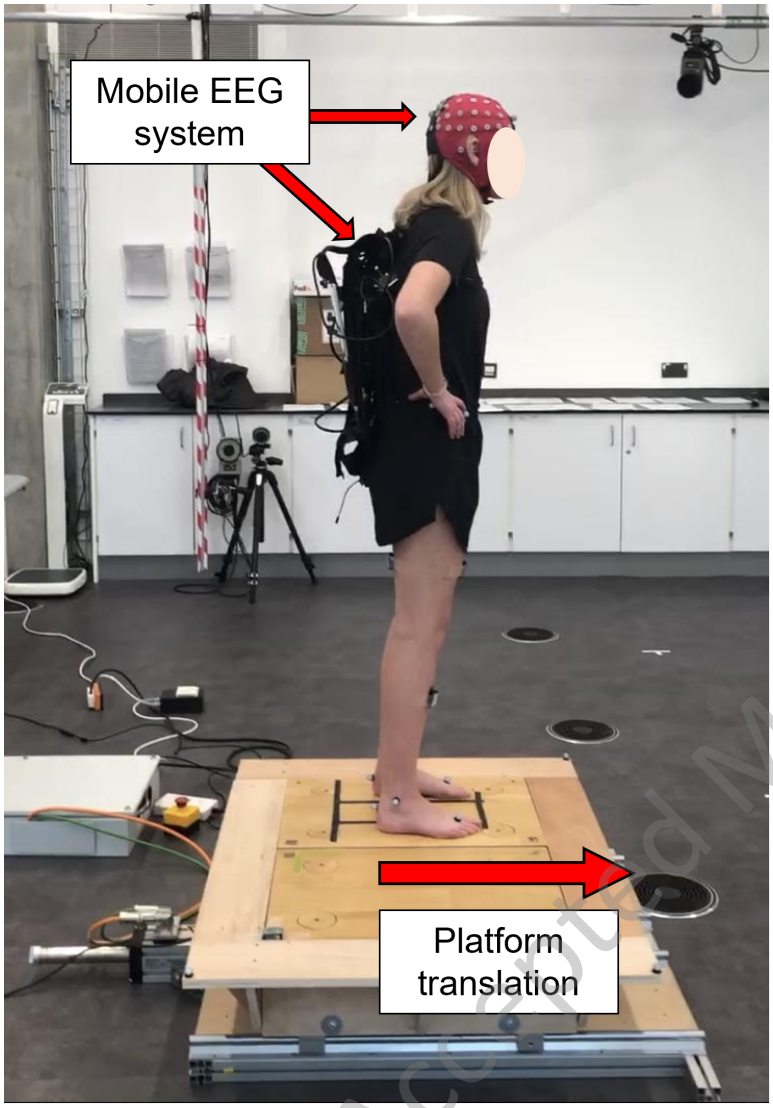
Figure 7. Scalp maps denoting the group-mean post-perturbation beta activity (decibels) across conditions for both the Slow (top row) and Fast (bottom row) perturbations. The scalp maps further right denote the t-scores obtained through channel-wise comparisons, with red regions indicating higher beta activity in the CMP compared to Control condition, and blue regions indicating lower beta activity in

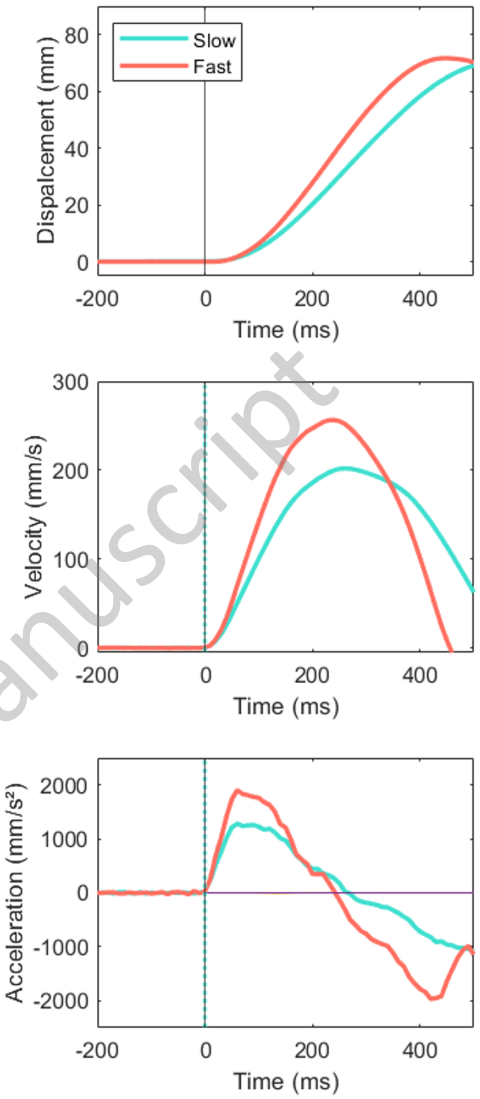
JNeurosci Accepted Manuscript

753 the CMP compared to the Control condition. Channel Cz is indicated by the white
754 dot, as this channel was the focus of these particular analyses.

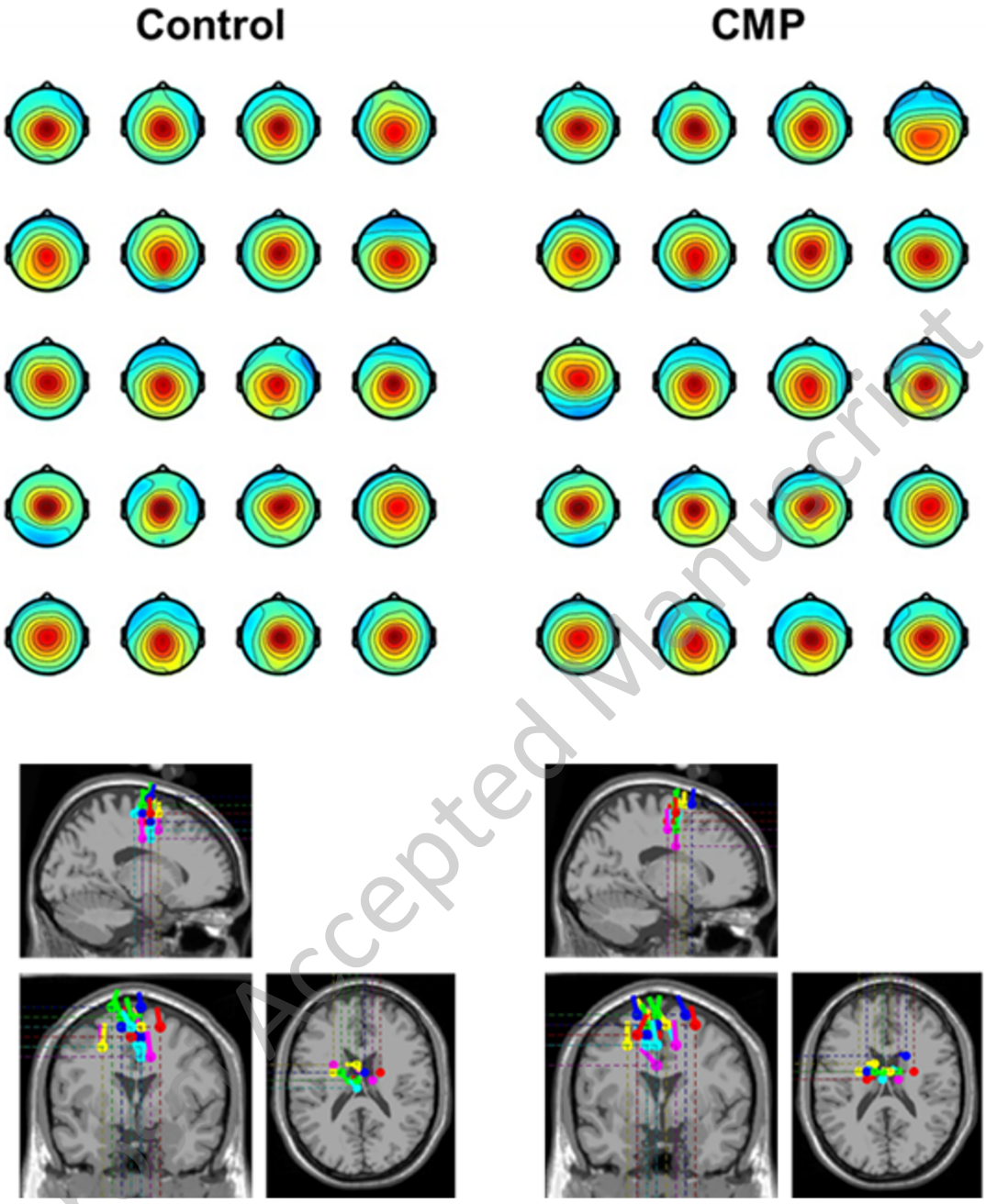
755

JNeurosci Accepted Manuscript

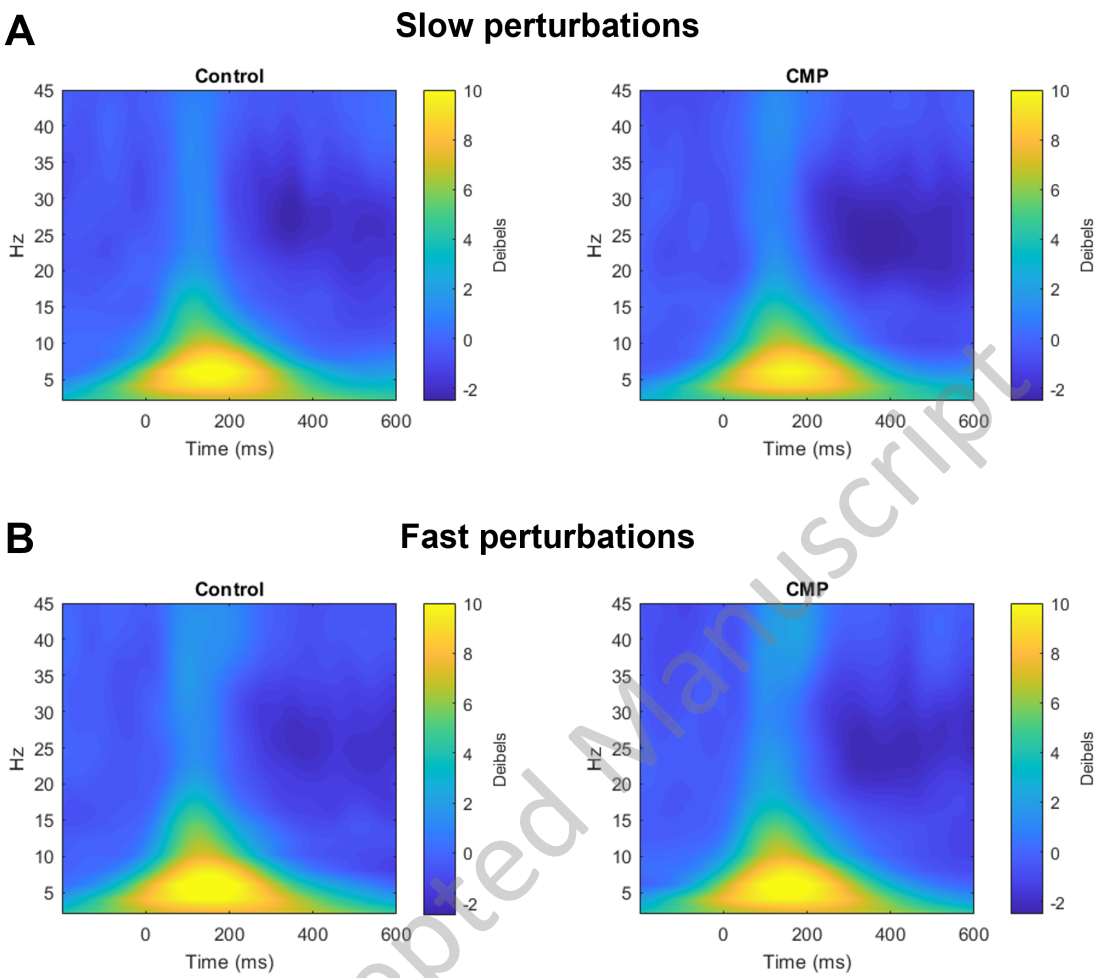




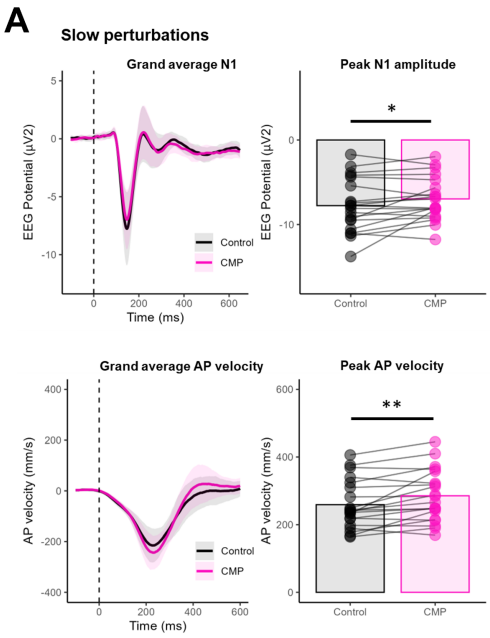
JNeurosci Accepted Manuscript



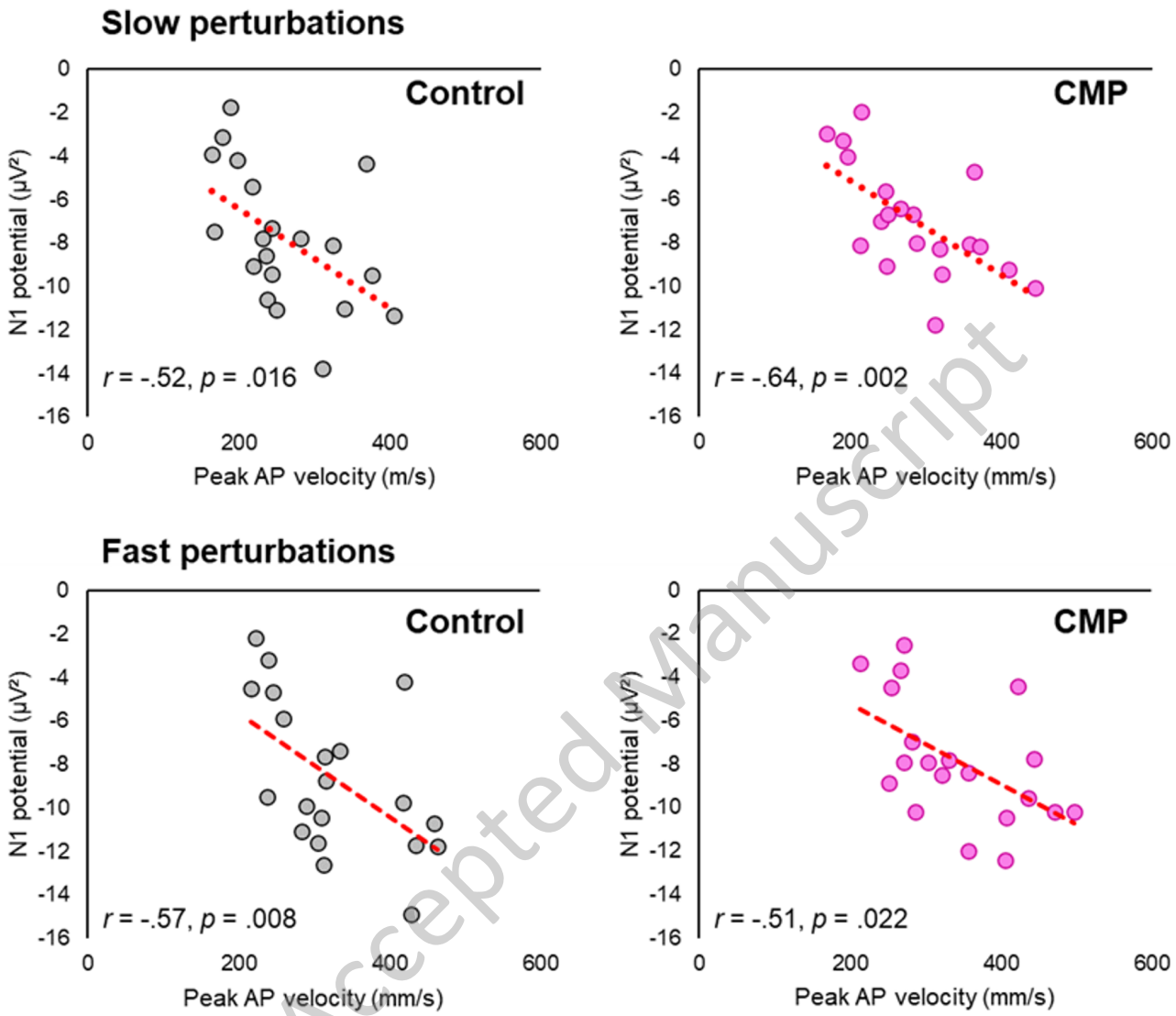
JNeurosci Accepted Manuscript



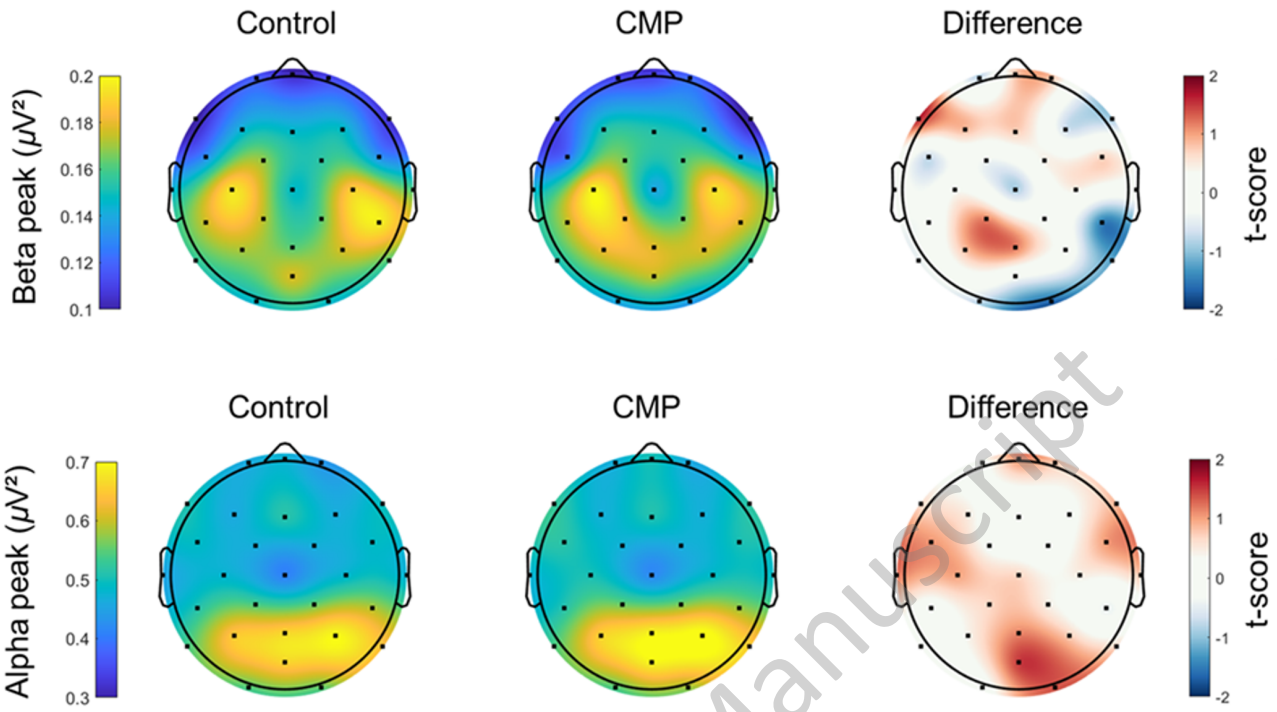
JNeurosci Accepted Manuscript



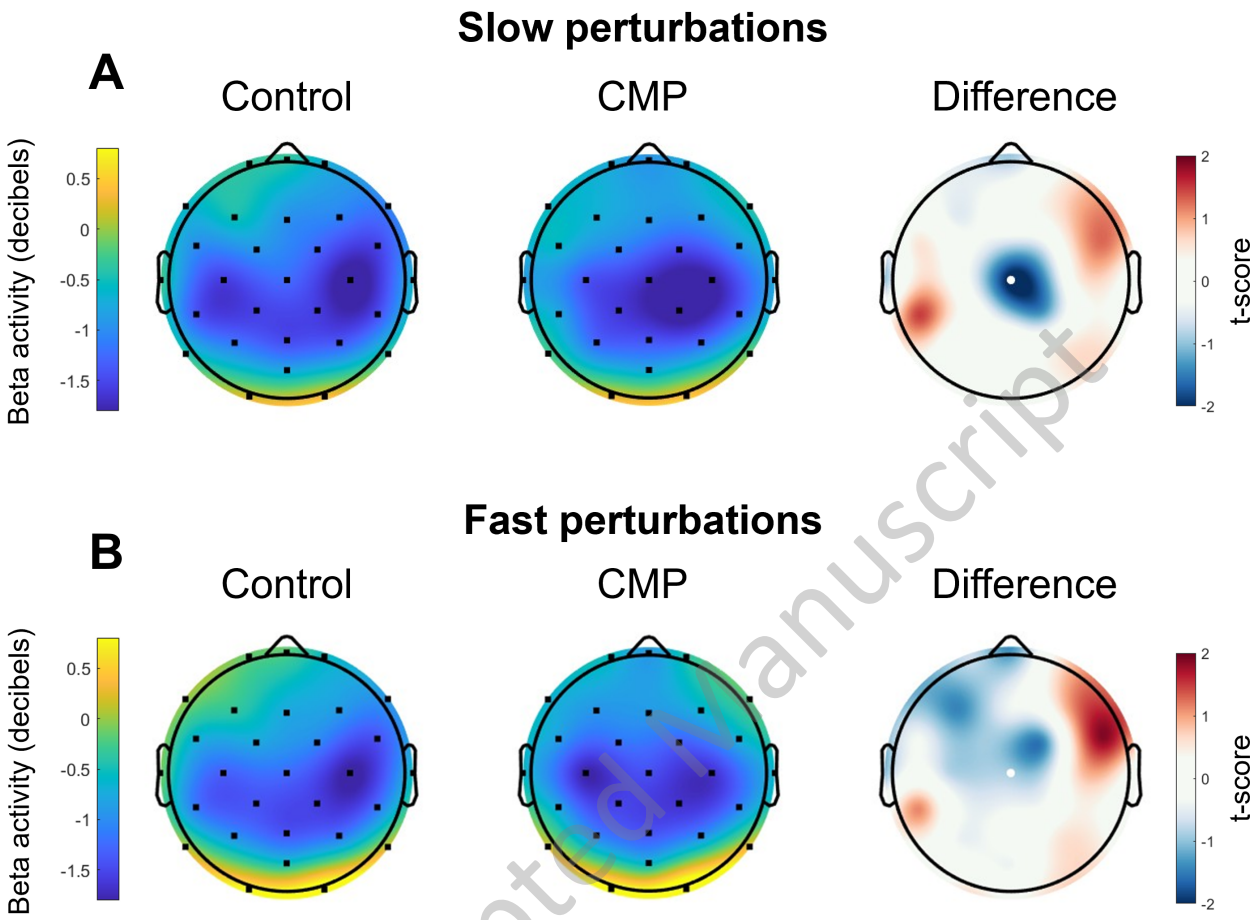
JNeurosci Accepted Manuscript



JNeurosci Accepted Manuscript



JNeurosci Accepted Manuscript



JNeurosci Accepted Manuscript