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

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11 **Short title: Conscious balance control impairs the cortical N1.**

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21

22

23 **Abstract**

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

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

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

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59 **Introduction**

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

71 Following an external balance perturbation, the central nervous system triggers
72 rapid (~100 ms) brainstem-mediated postural responses (Horak 2006; Jacobs and
73 Horak 2007; Welch and Ting 2008). This is followed by a negative
74 electroencephalographic (EEG) cortical response (the ‘N1’ evoked potential) across
75 the supplementary motor area ~100-200 ms after perturbation onset (Marlin et al.
76 2014; Varghese, McIlroy, and Barnett-Cowan 2017). The N1 is greater when facing
77 larger perturbations (Payne, Hajcak, and Ting 2019), when a corrective step is
78 required to avoid falling (Payne and Ting 2020a; Solis-Escalante et al. 2021; Zaback
79 et al. 2023), when a perturbation is unexpected (Adkin et al. 2006), and in individuals
80 with poorer balance abilities (Payne and Ting 2020b). Researchers have therefore
81 proposed that the N1 acts as an error detection mechanism that is “primed” for (i)
82 detecting centre of mass movements that approach one’s limits of stability and (ii)

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

85 The N1 can be influenced by “cognitive processes such as greater perceived
86 threat or *attention to balance*, which have the potential to influence subsequent
87 motor control” (Payne and Ting 2020b). Indeed, decreased cortical N1 amplitudes
88 occur when attention is directed *away* from balance via a cognitive dual-task (Little
89 and Woollacott 2015; Quant et al. 2004). In contrast, greater cortical N1 amplitudes
90 occur when stance is perturbed during conditions which are known to increase
91 attention *towards* balance (e.g. postural threat (Adkin et al. 2008; Zaback et al.
92 2023). However, these changes in CMP have co-occurred with increases in
93 physiological arousal and/or cognitive loading, making it difficult to isolate the neural
94 mechanisms through which CMP disrupts postural performance. The primary aim of
95 this study is to therefore explore the direct effects of increased CMP on the cortical
96 N1 response and subsequent postural control performance.

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

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

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

118 **Materials and methods**

119 *Participants*

120 Twenty neurotypical young adults participated in the experiment (10 females, 10
121 males; $M \pm SD$ age = 25.1 ± 5.0 yrs; height = 173.30 ± 11.17 cm; weight = 74.30 ± 10.81
122 kg). Sample size estimates were based on the medium ($d = 0.71$) to large effects (d
123 = 0.82) reported upon the cortical N1 under conditions that indirectly manipulate
124 CMP (e.g., heightened postural threat (Adkin et al. 2008) and divided attention (Little
125 and Woollacott 2015). Assuming a medium-to-large effect size ($d = 0.71$), a minimum
126 sample size of 18 participants was required to yield 80% power with an alpha level of
127 $p = 0.05$ when comparing mean differences between two related groups (calculated
128 using G*POWER software 3.1; Henrich University Dusseldorf, Germany). All
129 participants were free from any neurological disease and had no prior experience of
130 dizziness or balance problems. The experiment was approved by the Manchester
131 Metropolitan University institutional ethics committee (project ID #56055).

132 *Protocol*

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

144 Participants experienced two blocks of 50 discrete sine-wave perturbations (7-
145 15s random delay between each perturbation) consisting of an initial forward
146 translation of the support surface (maximum forward displacement = 70 mm) before
147 reversing direction and completing the sinewave to return to original position. Each
148 10-min block consisted of 50 perturbations: 25 fast (0.5 Hz, peak acceleration = 1883
149 mm/s², peak acceleration latency = 60 ms) and 25 slow (0.3 Hz, peak acceleration =
150 1277 mm/s², peak acceleration latency = 60 ms), presented in a pseudo-random
151 order. For the purpose of this study, we focused only on the initial forward portion of
152 the perturbation (see Figure 1) to not risk contamination of EEG data with the return
153 of the sine-wave perturbation. Perturbations were therefore predictable in amplitude
154 (70 mm max forward displacement) and direction (i.e., forwards), but unpredictable
155 in terms of both speed and timing, as perturbations were delivered every 7-15
156 seconds. To further maximise the unpredictability of stimulus presentation,

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

169 *Attentional focus manipulation*

170
171 As we sought to explore how CMP affects the neural control of balance when stance
172 is perturbed, one block (of 50 trials) was performed under conditions designed to
173 induce CMP; whilst the other block was performed under 'control' conditions (no
174 other instructions provided aside from the general task instructions). For the CMP
175 condition, participants were instructed to consciously monitor their postural stability
176 between each perturbation ("focus your attention towards how the weight is
177 distributed beneath your feet") and minimise any movement in their ankles. These
178 instructions were based on qualitative research that has explored what participants
179 direct their attention towards when CMP (spontaneously) occurs during postural
180 control (Zaback, Carpenter, and Adkin 2016). Prompts and reminders were delivered
181 to ensure that participants maintained this focus of attention throughout the block of

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

184 After each block of trials, participants completed a 4-item questionnaire that
185 assessed the extent to which they directed conscious attention towards their balance
186 during the previous set of trials (e.g., “I am always trying to think about my balance
187 when I am doing this task”; 1 = strongly disagree; 6 = strongly agree; Ellmers et al.
188 2021; Ellmers and Young 2018). This questionnaire served as a manipulation check.
189 Scores from the four separate items were summed to produce a total score of state
190 CMP. To assess any carry-over effects (i.e., order effects) of performing the CMP
191 condition first, we performed post-hoc independent t-tests to compare state CMP
192 between participants who performed either the Control or CMP condition first.
193 Results showed no difference between groups for the Control condition ($t(18) = .518$,
194 $p = .611$), the CMP condition ($t(18) = .767$, $p = .453$), or the change scores between
195 conditions ($t(18) = .446$, $p = .661$). After each condition of trials, participants also
196 completed a visual analogue scale that ranged from 0 (“not at all anxious”) to 10
197 (“the most anxious I have ever felt”) to rate the level of state anxiety that they felt
198 during the preceding trials (Castro et al. 2019). Higher scores therefore indicate
199 greater state anxiety. These self-reported assessments were used to confirm that the
200 CMP manipulation led to the intended increase in state CMP, whilst verifying that
201 any results observed were not confounded by any between-condition differences in
202 state anxiety.

203

204 *EEG recording and analyses*

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

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

215 EEG signals were band-pass filtered using the EEGLAB “basic FIR filter (new)” (1–
216 45Hz, 3300 filter order, –6 dB cutoff frequency, 1 Hz transition bandwidth) prior to
217 being cut into epochs ranging from –1 to +2 s relative to perturbation onset and re-
218 referenced to the average of all scalp electrodes. These epochs were visually
219 inspected for large EEG contamination from muscular artifacts, but no trials were
220 discarded. No bad EEG channels were identified. Independent component analysis
221 (ICA) weights were obtained separately for each condition through the RunICA
222 infomax algorithm (Jung et al. 1998) running on EEG signals. ICA weights that
223 presented obvious non neural activity upon visual inspection (e.g., eyeblinks, line
224 noise, muscular artifact) were manually rejected. On average, we retained 25.9 ± 1.1
225 and 25.9 ± 1.7 components across the CMP and Control conditions, respectively.

226 Following visual inspection, we then identified the brain component that gave rise to
227 a distinct cortical N1. Consistent with other studies, N1 components were localised
228 across the supplementary motor area (Marlin et al. 2014; Varghese et al. 2017), with
229 a midfrontal topography consistent across all participants and across the two
230 experimental conditions (Control and CMP; Figure 2). For visualisation purposes
231 only, cortical N1 sources were further mapped onto a standard MNI template and

232 estimated using the DIPFIT plugin (coarse fit; Klug and Gramann 2021; Oostenveld
233 and Oostendorp 2002). Estimated cortical locations and percentage of power
234 accounted for by the cortical N1 components can be found in Extended Data Table
235 2-1. To assess spectral characteristics of the selected cortical N1 component and
236 EEG channel data, we performed time-frequency decomposition via trial-by-trial
237 convolution with complex Morlet wavelets. We used 44 frequencies linearly spaced
238 between 2 and 45 Hz, with wavelets logarithmically spaced from 5 to 8 cycles. All
239 processing steps were performed using EEGLAB (v2020.0) functions (Delorme and
240 Makeig 2004) for MATLAB.

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

257 **Post-perturbation EEG analyses.** To assess the cortical N1 response, we
258 extracted single trial N1 amplitudes from the selected N1 component (see Figure 2).
259 However, given that analytical approaches vary across the literature (with some
260 studies analysing the N1 component (e.g., Mirdamadi et al. 2024; Solis-Escalante et
261 al. 2021) and others focusing only on channel Cz (e.g., Payne and Ting 2020b;
262 Varghese et al. 2017; Zaback et al. 2023), we also performed parallel N1 analyses
263 on channel Cz to confirm whether our findings were robust across component versus
264 channel level analyses. Time series data were baseline subtracted (-150 to -50 ms
265 before perturbation onset) for each participant, and the N1 was quantified as the
266 largest negative peak occurring 50-200 ms after perturbation onset. For each
267 participant, N1 amplitudes were subsequently averaged across fast and slow
268 perturbations separately for both the CMP and Control conditions. We also
269 calculated event-related spectral power (ERSP) of both the cortical N1 component
270 and EEG channel level data by dividing decomposed time-frequency data by the
271 average activity from -1000 to -500 ms prior to perturbation across all conditions and
272 trials (i.e., neutral baseline across conditions) before performing a $10 \cdot \log_{10}$
273 transformation (i.e., decibel change). We then extracted the average beta activity (15
274 – 30 Hz) between 200 to 400 ms post-perturbation from the selected cortical N1
275 component as an index of cortical engagement in balance recovery *following* the
276 cortical N1 response (Ghosn et al. 2020; Palmer et al. 2021). We again performed
277 parallel analyses of post-perturbation beta activity on channel Cz to confirm whether
278 our findings were robust across component versus channel level analyses. For the
279 purpose of visualisation, grand average ERSP of channel Cz are presented in Figure
280 3.

281

282 *Postural control analyses.*

283 We used custom MATLAB scripts to determine the peak velocity of centre of
284 pressure (COP) data in response to the initial forward portion of the perturbation. As
285 we used a forwards-moving perturbation, we restricted analysis to the anterior-
286 posterior (AP) direction. Peak backwards COP velocity was selected as our outcome
287 variable as it is a direction-specific response to the initial forward perturbation;
288 greater backwards CoP velocity generally indicates greater instability and higher risk
289 of falling (Hewson et al. 2010; Masani et al. 2014). First, for each event we selected
290 and low-pass filtered (5 Hz, 2nd order bidirectional Butterworth filter) a 3-second AP-
291 COP trace that spanned 2000 ms pre-perturbation and 1000 ms post-perturbation.
292 We then corrected this trace for offset using the estimated average AP COP
293 displacement during the 'baseline' period (based on the 1100-100 ms pre-
294 perturbation window). Peak velocity of the postural response to the perturbation was
295 then identified as the first negative peak in the derivative of the AP-COP trace in the
296 initial forward portion of the perturbation (Figure 1). By default, the initial negative
297 peak was selected unless a subsequent peak was of >50% greater magnitude than
298 the earlier peak. The mean latency to peak velocity (termed 'peak latency') for slow
299 perturbations were 219 ms ($SD = 29$, range = 166-278) and 217 ms ($SD = 27$, range
300 = 164-271) for Control and CMP conditions, respectively. The mean peak latencies
301 for fast perturbations were 213 ms ($SD = 23$, range = 172-260) and 212 ms ($SD =$
302 21, range = 173-258) for Control and CMP conditions, respectively.

303

304 *Statistical analyses.*

305 The Gaussian distribution of data were checked via Shapiro-Wilk test of normality.

306 Paired samples t-tests were therefore used to determine differences between

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

324

325 **Results**

326 *Attentional focus manipulation checks*

327 Participants reported directing significantly greater conscious attention towards their
328 balance in the CMP ($M = 14.50$, $SD = 4.02$) compared to Control condition ($M =$
329 11.80 , $SD = 5.45$, $t = -4.61$, $p < .001$, $d = 0.56$), confirming the effectiveness of the
330 CMP manipulation. There was no difference in state anxiety between conditions, with

331 low levels of anxiety experienced for both (Control, $M = 1.95$, $SD = 1.76$; CMP, $M =$
332 1.95 , $SD = 1.32$, $Z = -0.36$, $p = .971$, $r = 0.018$).

333

334 *N1 amplitude*

335 Analysis of the cortical N1 component showed a significant main effect of
336 perturbation speed, $F(1, 19) = 28.86$, $p < .001$, $\eta^2 = .603$, with larger N1 amplitudes
337 observed during fast compared to slow perturbations (irrespective of attentional
338 focus condition). There was also a significant main effect of Attention condition, $F(1,$
339 $19) = 6.11$, $p = .023$, $\eta^2 = .243$, with smaller N1 amplitudes observed in CMP
340 compared to the Control condition (irrespective of the perturbation speed). On
341 average, N1 amplitudes during the CMP condition were 8% smaller for fast
342 perturbations and 10% smaller for slow perturbations, compared to Control. There
343 was no Attention x Speed interaction, $F(1, 19) = 0.12$, $p = .737$, $\eta^2 = .006$ (Figure 4).
344 Consistent findings were observed when analyses were performed on channel Cz
345 (rather than the N1 component). However, N1 amplitudes for channel Cz were
346 approximately three times larger than the amplitudes of the N1 component (see
347 Extended Data Figure 4-1). Individual N1 amplitudes from both the component and
348 channel Cz analyses were also highly correlated ($r_s > .92$), confirming the
349 robustness of the results across component- and channel-level analyses (see
350 Extended Data Figure 4-2). A detailed comparison of descriptive and inferential
351 statistics from the component and channel Cz analyses is provided in Extended Data
352 Table 4-1 and 4-2.

353 *Postural control*

354 There was a significant main effect of perturbation Speed ($F(19) = 274.683$, $p < .001$,
355 $\eta^2 = .935$), with greater peak AP velocities observed for fast compared to slow

356 perturbations. There was also a significant main effect of Attention condition ($F(1,$
357 $19) = 7.915, p = .011, \eta^2 = .294$) and a significant interaction between Attention and
358 perturbation Speed ($F(1, 19) = 9.109, p = .007, \eta^2 = .324$). Post-hoc comparisons
359 showed peak AP velocities to be significantly greater during the CMP condition
360 compared to the Control condition for both fast ($p = .047$) and slow ($p = .004$)
361 perturbations, with this effect more pronounced for the slow perturbations (Figure 4).
362 For fast perturbations, Pearson's correlations also revealed a significant negative
363 correlation between peak AP velocity and N1 amplitude for both the CMP ($r = -.51, p$
364 $= .022$) and Control conditions ($r = -.57, p = .008$), whereby greater velocities were
365 associated with smaller N1 amplitudes. The same relationship was observed for slow
366 perturbations during both the CMP ($r = -.64, p = .002$) and Control ($r = -.52, p = .016$)
367 conditions (see Figure 5).

368 ***Pre- and post-perturbation cortical activity.*** Paired t-tests revealed no difference
369 in the cortical N1 component's pre-perturbation peak beta ($t(19) = 0.62, p = .539, d =$
370 $.14$), beta AUC ($t(19) = 0.67, p = .513, d = .14$), aperiodic exponent ($t(19) = 0.04, p =$
371 $.970, d = .01$), or aperiodic offset, $t(19) = 0.89, p = .391, d = .20$) between CMP and
372 Control conditions. For EEG channel-level analyses, topographical analyses of pre-
373 perturbation periodic beta and alpha activity revealed no channel-wise differences
374 between conditions in peak or AUC values (Figure 6). For post-perturbation beta
375 activity of the N1 component, the ANOVA revealed no main effect of Condition, $F(1,$
376 $19) = 2.31, p = .144, \eta^2 = .109$, no main effect of perturbation Speed, $F(1, 19) =$
377 $3.71, p = .069, \eta^2 = .163$, and no Condition x Speed interaction, $F(1, 19) = 0.01, p =$
378 $.976, \eta^2 = .000$. However, for post-perturbation beta activity of channel Cz, the
379 ANOVA showed a significant main effect of Condition, $F(1, 19) = 4.45, p = .048, \eta^2$
380 $= .190$, with lower beta activity during the CMP condition compared to the Control

381 condition, particularly for the slower perturbations (Figure 7). There was neither a
382 significant main effect of perturbation Speed, $F(1, 19) = 1.44$, $p = .244$, $\eta^2 = .071$,
383 nor Condition x Speed interaction, $F(1, 19) = 3.68$, $p = .070$, $\eta^2 = .162$.

384 Discussion

385 We explored how directing conscious attention towards balance affects the
386 cortical control of posture during discrete perturbations to quiet stance. Our findings
387 revealed that the cortical N1 – a neural signal involved in monitoring postural
388 instability and mobilising compensatory balance-correcting responses (Payne and
389 Ting 2020a; Solis-Escalante et al. 2021; Zaback et al. 2023) – was significantly
390 smaller during conditions of experimentally-induced CMP. Behaviourally, this was
391 coupled with greater peak COP velocity during the CMP condition, indicating greater
392 postural instability. Although effective postural control requires some degree of
393 attentional resources (Boisgontier et al. 2017; Woollacott and Shumway-Cook 2002),
394 directing *too much* attention towards balance can disrupt postural control – much like
395 how athletic performance breaks-down when experts adopt a self-focus (Baumeister
396 1984; Parr et al. 2023; Smith et al. 2003). The present findings provide the first
397 evidence that such maladaptive effects of CMP on postural control appear to be
398 expressed by insufficient activation at the cortex relevant for postural control.

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

405 Zaback et al. 2016). Conversely, individuals will direct *less* attention towards balance
406 during conditions of dual-task (Ellmers et al. 2021; Johnson et al. 2020). We
407 therefore expected larger N1 amplitudes under conditions of CMP that was induced
408 *independent* of postural threat, and that this result would reflect an increased
409 sensitivity of the sensorimotor system for responding to postural disturbances during
410 self-focused attention (Harris et al. 2023). Self-report data confirmed that our
411 manipulation was successful at isolating CMP from perceived threat/anxiety.
412 However, contrary to our prediction, we observed significantly *smaller* N1 responses
413 under conditions of CMP. This reduction in N1 amplitudes (average reduction of
414 ~9%) is akin to reductions previously reported during conditions of cognitive dual-
415 task (between ~5-20% reduction; Little and Woollacott 2015; Quant et al. 2004). This
416 suggests that the larger N1 amplitudes observed previously during conditions of
417 increased postural threat (which is known to induce CMP) may instead reflect threat-
418 related increases in mental vigilance or arousal, rather than changes in attention to
419 movement (Zaback et al. 2023). Indeed, emotional arousal has also been shown to
420 modulate the amplitude of the N1 in non-motor (i.e. cognitive) tasks (Luna et al.
421 2023).

422 Researchers have proposed that the N1 – which is localised to the
423 supplementary motor area – acts as an instability and/or error detection mechanism
424 that is “primed” for (i) detecting centre of mass movements that approach one’s limits
425 of stability and (ii) mobilising compensatory stepping responses (Payne and Ting
426 2020a; Solis-Escalante et al. 2021; Zaback et al. 2023). Supporting this stance, the
427 present findings showed that the cortical N1 scales with perturbation intensity, with
428 greater N1 amplitudes observed during the fast (compared to slow) perturbation. Our
429 findings also revealed larger N1 amplitudes in individuals with poorer within-task

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

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

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

462 However, the effect of CMP upon the cortical N1 may differ across balance-
463 impaired populations for whom CMP reflects a compensatory strategy to overcome
464 poorer (and less 'automatic') balance (Boisgontier et al. 2017; Clark 2015; Kal et al.
465 2022). For instance, it is possible that older adults with fear of falling may instead
466 use CMP proactively in a way that enhances, rather than disturbs, the central set
467 (see Ellmers et al. 2023). Future work should therefore look to extend these findings
468 beyond healthy young adults. Nonetheless, these findings provide the evidence that,
469 in neurotypical young adults with relatively good balance control, CMP may disrupt
470 postural control via insufficient compensatory activation at the cortex in response to
471 perturbations.

472 Contrary to our prediction, the CMP manipulation had no effect on pre-
473 perturbation oscillatory alpha or beta activity. Within the context of balance, lower
474 pre-perturbation beta EEG activity of the cortical N1 component is associated with
475 *enhanced* perception of the subsequent perturbation to balance (Mirdamadi et al.
476 2024), suggesting that lower beta activity may reflect a more sensitive sensory
477 processing system. Given that CMP is proposed to increase perceptual sensitivity for
478 postural disturbances (Ellmers et al. 2021; Harris et al. 2023), we had expected CMP
479 would thus lower pre-perturbation beta. In line with previous research (Parr et al.

480 2023; Sherman et al. 2021), we had also expected CMP to promote elevated alpha
481 activity across the visual cortex, possibly reflecting a mechanism that supports
482 vigilance to somatosensory processing by down-weighting visual processing through
483 regional inhibition (Jensen and Mazaheri 2010). However, no differences in pre-
484 perturbation alpha or beta activity were observed, which suggests that our CMP
485 manipulation did not alter ongoing perceptual sensitivity prior to postural
486 disturbances. Previous research has also reported higher post-N1 beta activity in
487 individuals with poorer balance (Palmer et al. 2021), and when experiencing larger
488 perturbations (Ghosn et al. 2020), suggesting a (conscious) compensatory role for
489 such neural activity. However, we instead observed significantly *larger* reductions in
490 post-perturbation beta activity during CMP irrespective of perturbation size. Whilst
491 the functional role of sensorimotor beta oscillations is still not fully understood
492 (Barone and Rossiter 2021; Spitzer and Haegens 2017), researchers have proposed
493 that reductions in beta activity during an ongoing action may reflect a “decrease in
494 somatosensory responsiveness for the efficient unfolding of the movement” (p. 22,
495 Kilavik et al. 2013). The reduced beta activity we observed during the late recovery
496 phase of the perturbation could therefore reflect a continued dampening of the
497 sensorimotor system (i.e. beyond the initial cortical N1 response) when engaging in
498 CMP. Previous researchers have consistently proposed CMP to enhance, rather
499 than dampen, sensorimotor sensitivity during postural control (Ellmers et al. 2021;
500 Harris et al. 2023), but our findings question this interpretation of CMP. It is also
501 important to note that this finding was restricted to the channel-level (i.e., Cz)
502 analyses, suggesting these post-perturbation features were not captured by the
503 single component that contributes to the cortical N1. Future research should look to

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

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703

704

705 **Figure Legends**

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

713

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

720

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

723 **Figure 4.** Summary results for the N1 component's ERP and AP velocity for the slow
724 (4A; top four panels) and fast perturbations (4B; bottom four panels). For each figure,
725 separately presented are: Top left: Group-level perturbation evoked potentials, with
726 the thick solid lines and shaded region of the ERP denoting mean and standard
727 deviation, respectively; Top Right: N1 amplitudes for both the CMP and Control
728 conditions, with the bars denoting group mean values and points denoting individual
729 participant mean values; Bottom Left: Group-level AP velocity traces for both the

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

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

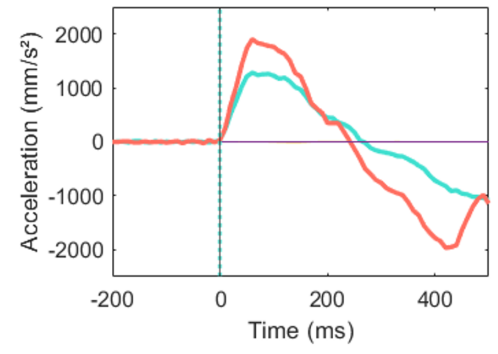
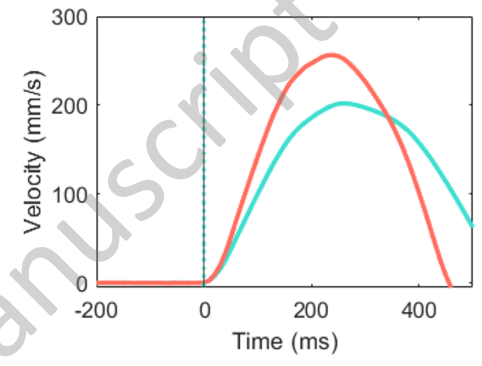
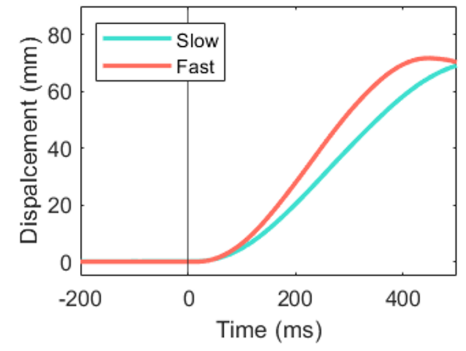
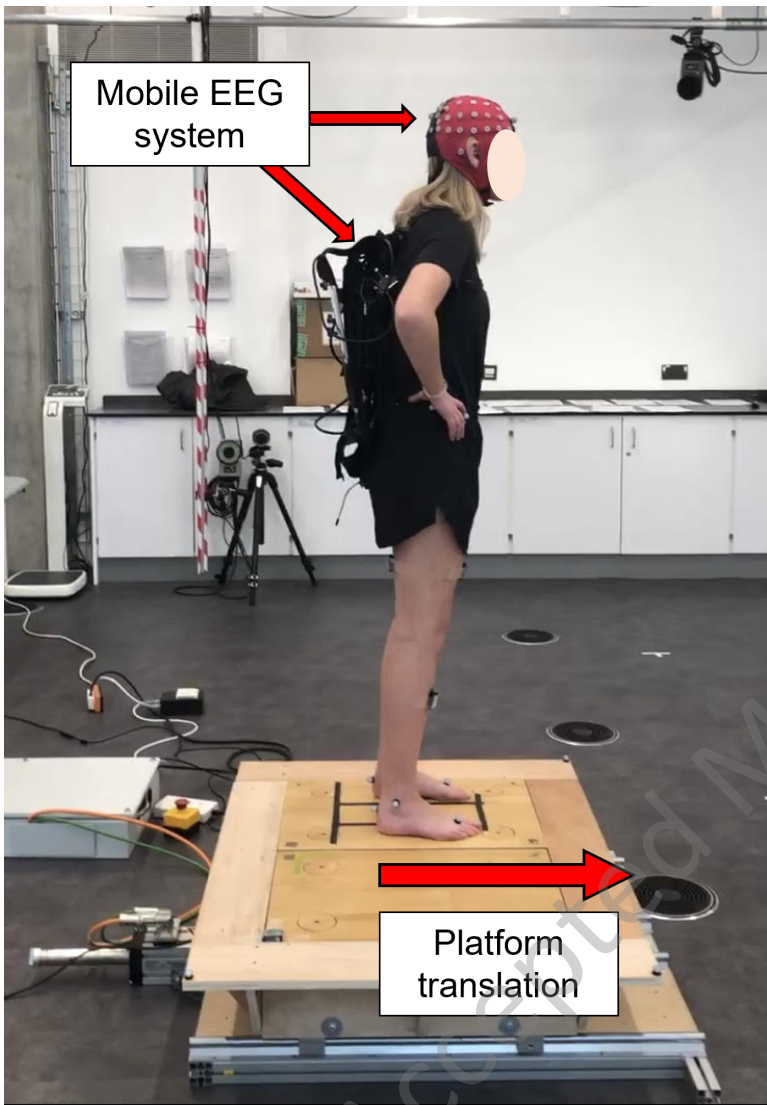
742 **Figure 6.** Scalp maps denoting the group mean values of pre-perturbation beta peak
743 (top row) and alpha peak (bottom row) for the Control and CMP conditions,
744 presented as normalised area under the spectral curve. The scalp maps furthest
745 right denote the t-scores obtained through channel-wise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

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

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

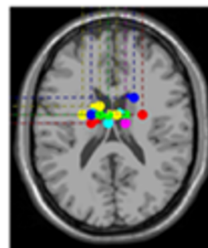
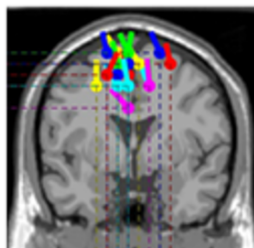
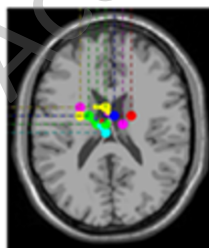
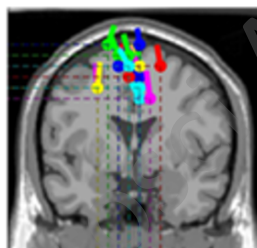
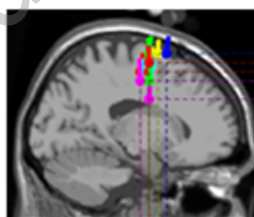
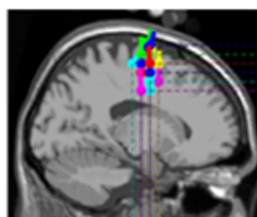
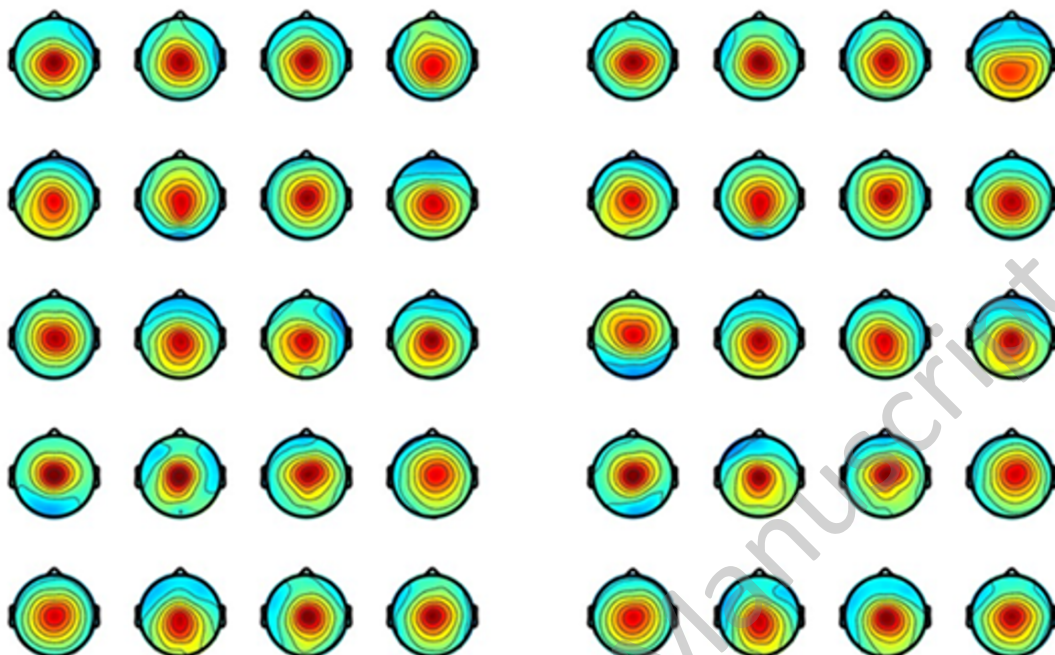
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Control

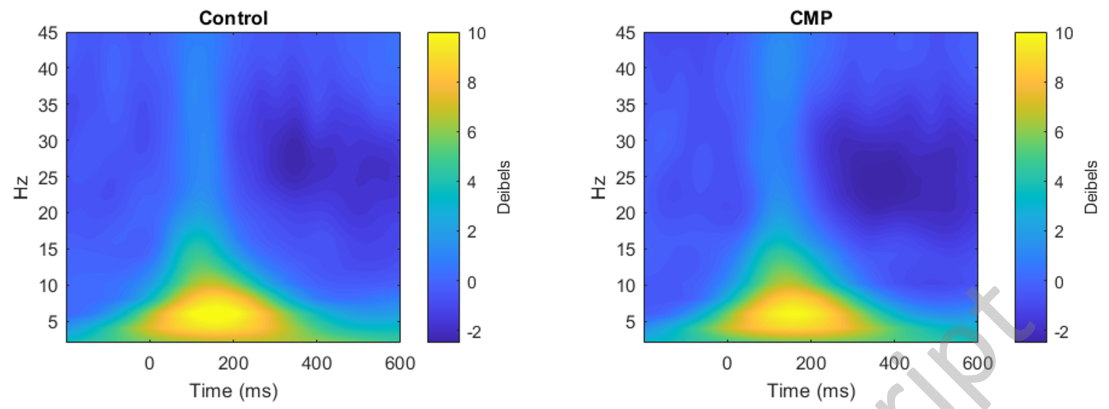
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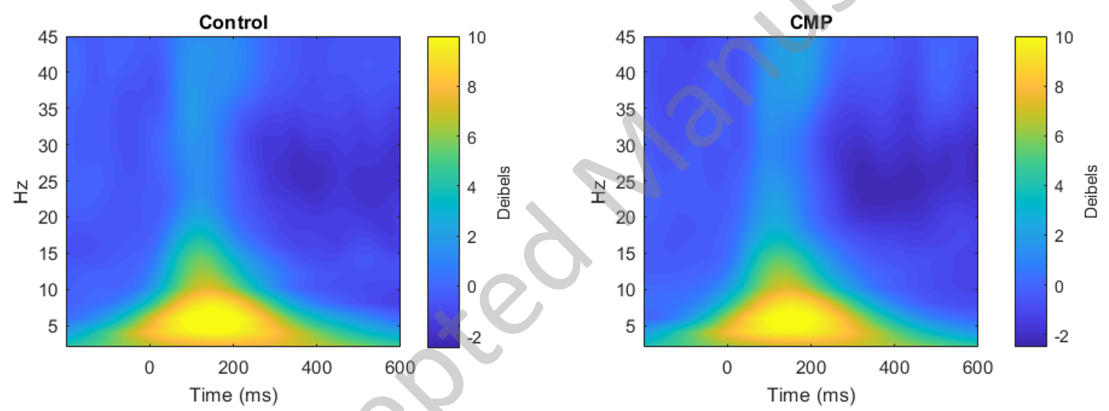
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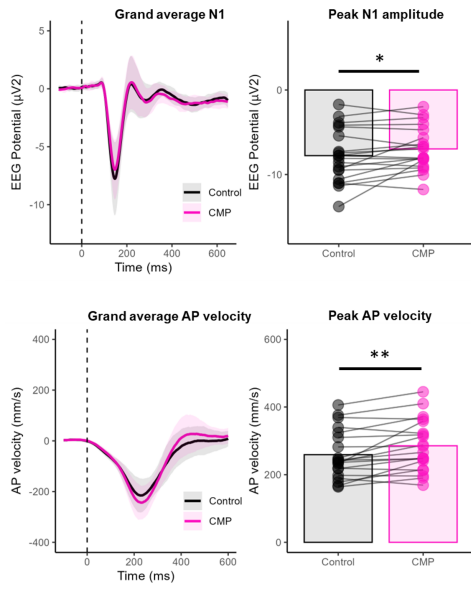
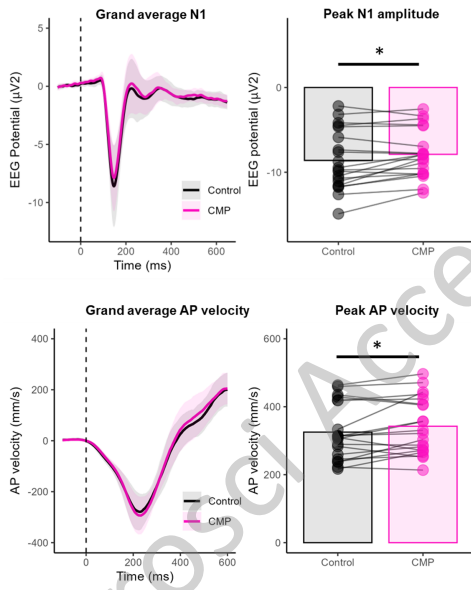
A Slow perturbations



B Fast perturbations

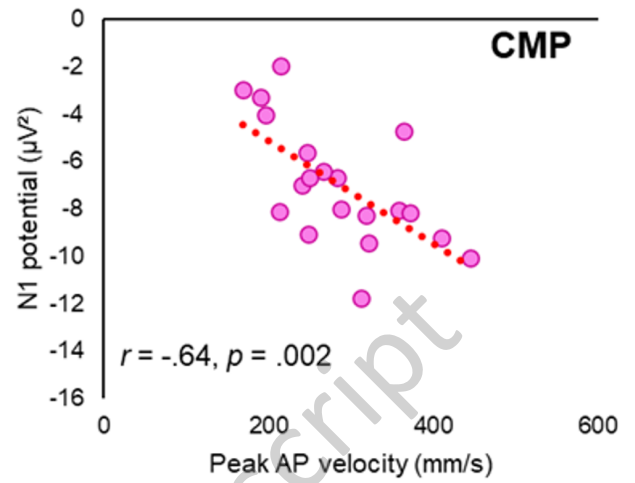
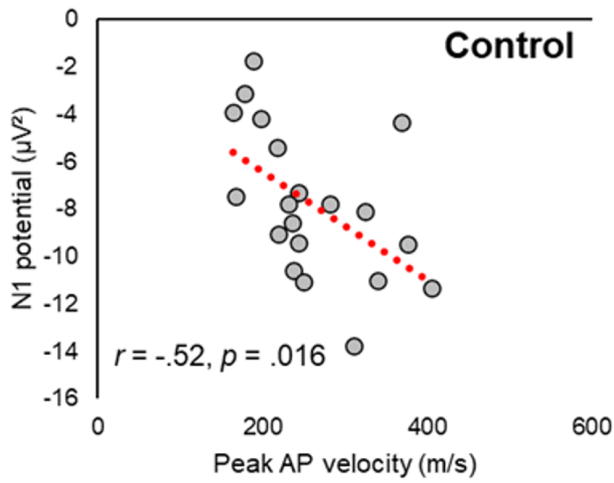


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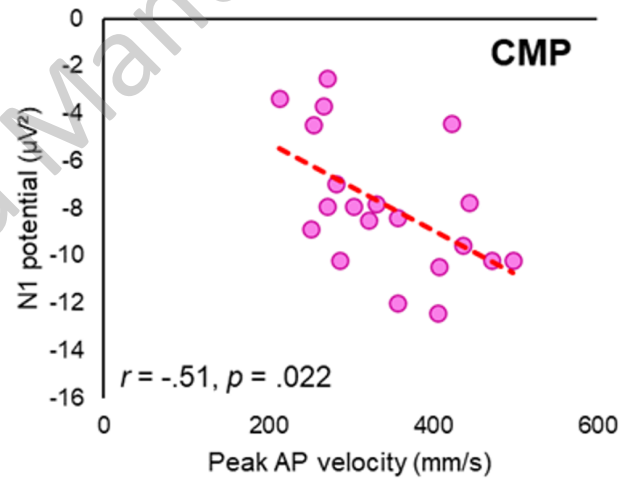
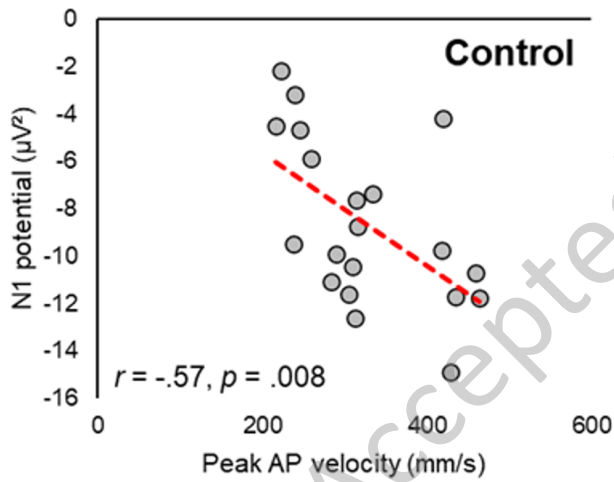
A**Slow perturbations****B****Fast perturbations**

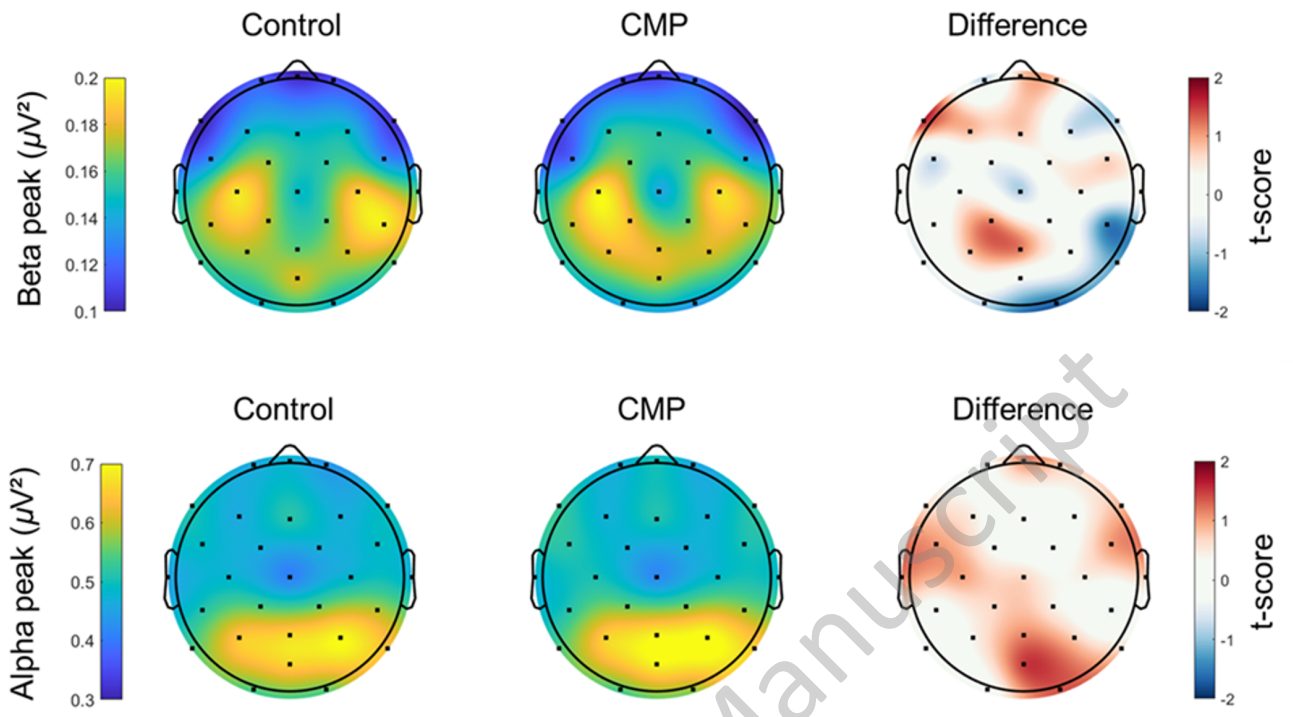
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Slow perturbations

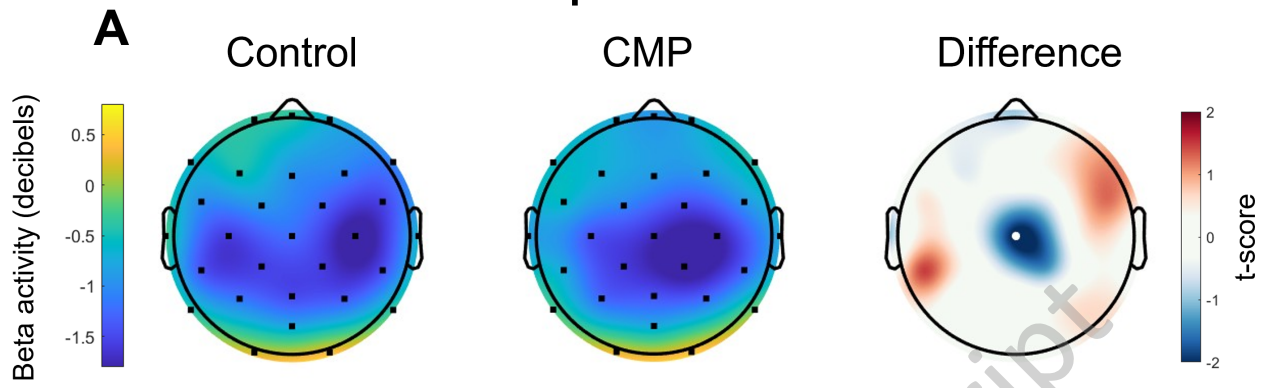


Fast perturbations

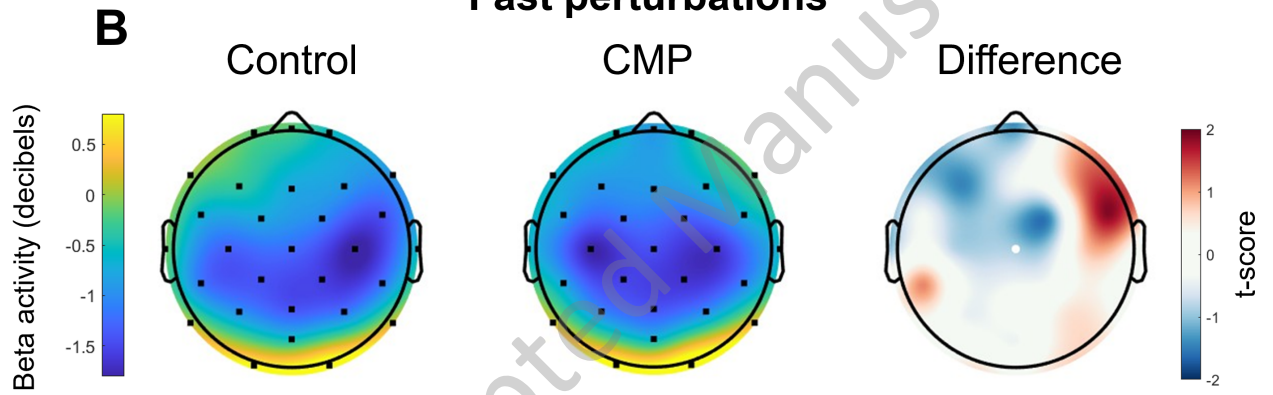




Slow perturbations



Fast perturbations



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