Perceiving Instability: How Expectations Bias Sensorimotor Processing in Balance Control

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Classification:

Biological Science (Neuroscience)

Keywords:

Predictive processing; Predictive coding; Posture; Perception; EEG

Funding:

This research was supported by a Wellcome Trust Sir Henry Wellcome Postdoctoral Fellowship awarded to T.J.E. (Grant Number: 222747/Z/21/Z).

Conflicts of interest:

The authors declare no competing interest.

Data availability:

All data and analytical code are freely available via Open Science Framework (https://osf.io/yv2a9/).

Significance Statement

Perception is often explained through predictive coding, a framework in which the brain compares incoming sensory input with internal expectations and updates its models based on mismatches, or prediction errors. Whether this process extends to rapid motor systems like balance, however, remains unclear. We tested this question in the context of human postural control—a fast, ecologically relevant sensorimotor system. Using EEG and behavioural measures, we show that top-down expectations shape both the perception and early cortical processing of imbalance, even when sensory input contradicts those expectations. Crucially, these cortical markers were not sensitive to prediction errors. This challenges the universality of prediction error in perception and calls for a rethinking of how predictive perceptual processes operate in rapid sensorimotor systems. This has implications for both predictive coding theories and clinical conditions marked by distorted bodily awareness.

Abstract

Maintaining balance requires rapid integration of sensory input with top-down sensorimotor predictions. Predictive coding frameworks posit that mismatches between the two ("prediction errors") drive perceptual inference. Although these frameworks have helped explain perception in unisensory domains (e.g., vision, touch), it remains unclear whether such mechanisms govern perception in fast-acting sensorimotor systems like human balance. Using electroencephalography (EEG) and discrete postural perturbations, we independently manipulated expectations and sensory input. Participants were primed to expect small or large perturbations, with occasional violations (i.e., delivering smaller or larger-than-expected perturbations). Subjective perception of instability was shaped by expectation alone, regardless of actual sensory input (i.e., perturbation magnitude). Similarly, both pre-perturbation beta-band suppression and post-perturbation gamma increases were sensitive to *expected* perturbation magnitude. In contrast, both the balance N1 (a well-established stimulus-evoked cortical potential) and the objective postural responses of instability tracked actual perturbation magnitude alone. Crucially, neither perception nor early neural or behavioural markers encoded prediction errors. These findings point to two distinct systems: one shaped by top-down expectations and another driven solely by sensory input, independent of mismatch or prediction error. This challenges the central role of prediction error in fast sensorimotor systems like postural control and may inform conditions involving distorted bodily awareness.

Introduction

Recent advances in neuroscience have revealed that the brain operates as a ‘prediction machine’ (1). Rather than passively receiving sensory input from the periphery, the brain actively shapes perception by attempting to match incoming information to top-down expectations (2). This is useful, because it allows us to efficiently process and filter the vast amount of (often “noisy”) sensory information we encounter. But such processing shortcuts mean that perception can be inaccurately biased towards expectation, especially during scenarios that have thus far been highly predictable and/or when sensory input is ambiguous (with any resulting prediction errors thus explained away as “noise” rather than being used to update perceptual inference). Such expectation-based perceptual biases have been reported across sensory domains, including vision (3,4), audition (5,6), and pain (7–9).

It remains unknown, however, if the same expectation-based perceptual biases similarly affect sensory systems that are fast, automatic, and tightly coupled to motor output, such as balance control. Balance relies on rapid subcortical feedback loops (10,11) and continuous multisensory integration (12,13) to respond to postural disturbances. Although most of these disturbances (and associated postural adjustments) occur outside of conscious awareness (14,15), the balance system nonetheless gives rise to vivid subjective perceptions of (in)stability (16,17). Whilst balance behaviour itself is strongly driven by predictive processes (e.g., locomotor adaptation (18,19)), the extent to which top-down predictions biases balance perception and sensorimotor processing remains unclear. Individuals frequently report feeling imbalanced even when postural stability remains high (16,20) and, in some cases, these feelings persist despite clear sensory evidence to the contrary (21). This would suggest a dominant role for expectations and limited involvement of prediction errors. These findings raise a key question: in balance, does prediction error drive perception and sensorimotor processing, as predictive coding would suggest, or are expectations dominant?

The present work therefore tested how sensory signals interact with expectations (and violated expectations, i.e., prediction errors) during postural imbalance. Using electroencephalography (EEG), we measured neural responses during discrete postural perturbations whilst manipulating expectation about the size of the upcoming disturbance. Our results revealed that subjective perceptions of instability were strongly shaped by expectation: participants felt more unstable when anticipating large perturbations, regardless of actual perturbation size received. We identified two temporally and spatially distinct neural markers that were modulated by expectation: anticipatory beta-band activity and post-perturbation gamma power. In contrast, the balance N1, a well-established evoked cortical response to instability (22,23), and postural responses themselves were driven solely by perturbation size (i.e., sensory input) and unaffected by expectation. Crucially, none of these signals were sensitive to prediction errors.

Our findings provide the first direct evidence that top-down expectations bias how sensory signals are interpreted at both the perceptual and neural level during balance control. The lack of clear cortical markers that tracked prediction error also challenges the universality of

prediction error in perceptual inference. Instead, whilst top-down expectations appear to dominate perceptual inference in fast-acting motor systems like balance, short-latency mechanisms (i.e., N1) that are insensitive to both expectation and mismatch persist. These systems likely ensure that behaviour remains flexibly tuned to rapidly changing contexts, minimising the risk of a fall occurring even when faulty predictions occur. By identifying neural markers of expectation-driven distortion in balance perception, our results offer new insight how predictive coding drives perception during fast-acting sensorimotor processes and offers novel avenues for mechanistic research into clinical conditions marked by subjective imbalance.

Results

Twenty-one neurotypical young adults experienced a series of anterior-posterior (AP) discrete balance perturbations. Perturbations were delivered under two blocks that manipulated expectation. In the Small-Expectation (SE) block of trials, participants were informed that they would encounter small perturbations (see Figure 1A) throughout the block of 60 trials. However, 15 of these perturbations violated expectations (i.e., they were 'big'). These expectancy-violating perturbations were introduced on average every 2-3 trials and were never presented in succession. The Big-Expectation (BE) block of trials followed the same structure but with the perturbation sizes reversed. After certain expectation-confirming and -violating trials (see Methods section), participants also self-reported their levels of perceived instability and state anxiety during the preceding trial (17). Behavioural responses to each perturbation were operationalised as directionally-specific (i.e., backwards) centre of pressure (COP) peak velocity.

We assessed EEG throughout the experiment. Specifically, we analysed pre-perturbation beta activity, given that lower pre-stimulus beta activity is reliably linked to enhanced sensory sensitivity (24,25) and might thus be sensitive to top-down expectation. We then also analysed perturbation-evoked responses: the balance N1 (a negative response occurring across the supplementary motor area ~150 ms after an external balance perturbation (26,27)) and post-perturbation gamma activity. Whilst balance N1 amplitude typically scales with perturbation magnitude (26,28), findings suggest that the N1 is attenuated when perturbations are temporally expected (22). Likewise, there is extensive research describing cortical gamma (>30 Hz) as a robust prediction error signal across tasks and sensory modalities (29,30). These outcomes will therefore provide insight into how prediction errors (e.g., trials that violate expectations) shape cortical processing of balance.

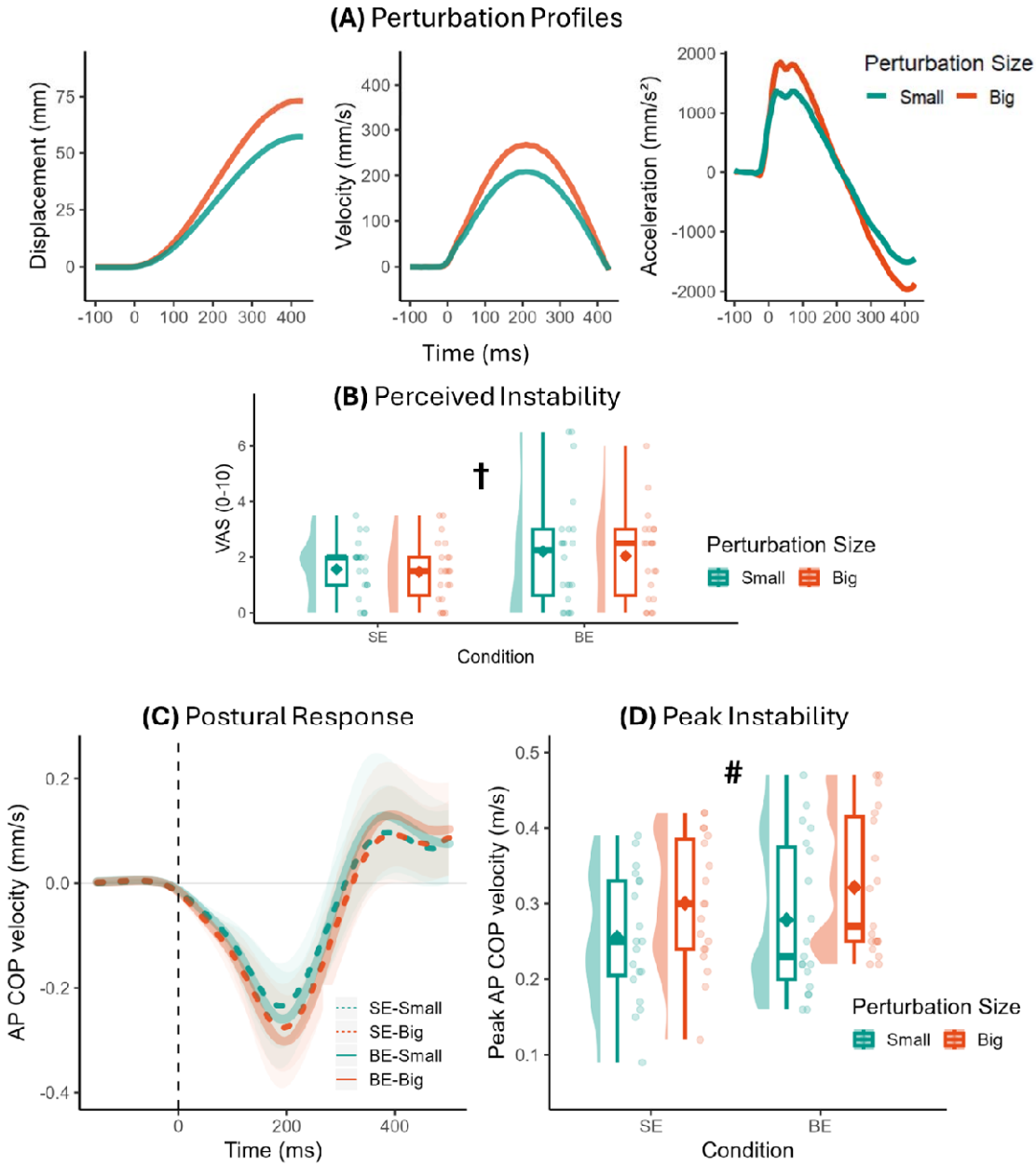
Subjective and objective balance response

Subjective balance experience. We first assessed how expectations and perturbation intensity influenced how unstable and anxious participants felt during perturbations. We used separate generalized estimating equations (GEE) analyses to explore the effects of Expectation and Perturbation Size on both self-reported outcomes (perceived instability and anxiety). There was a significant main effect of Expectation for both perceived instability (χ^2 =

6.05, $p = 0.014$) and anxiety ($\chi^2 = 10.38$, $p < 0.001$). There was no effect of Perturbation Size (*p*s > 0.260), nor a significant Expectation x Perturbation Size interaction (*p*s > 0.550). This indicates that both perceived instability and self-reported anxiety were shaped by expectation alone, with significantly greater perceived instability and anxiety levels experienced during Big-Expectation trials, irrespective of the size of the perturbation received (Figure 1B).

Objective balance response. Next, we used a GEE to assess *objective* balance responses to the perturbation. In contrast to the self-reported outcomes, there was a main effect of Perturbation Size only with respect to peak backwards COP velocity ($\chi^2 = 164.02$, $p < 0.001$), but no effect of either Expectation ($\chi^2 = 2.35$, $p = 0.126$) nor an interaction effect ($\chi^2 = 0.04$, $p = 0.835$). Consequently, the behavioural response to the perturbation was shaped by perturbation size alone, with significantly greater peak backwards COP velocity when receiving Big versus Small perturbations, irrespective of expectation (Figure 1C and 1D).

Figure 1. *Displacement, velocity, and acceleration of the initial forward platform translation for each perturbation size (A). Subjective balance experience (B; perceived instability). Grand averages (±SD) of centre of pressure (COP) velocity (C) and objective balance outcomes of peak instability (peak anterior-posterior [AP] centre of pressure COP velocity) across the four trial types (D). Note, SE reflects Small-Expectation trials whilst BE reflects Big-Expectation trials (e.g., 'SE-Small' indicates that participants expected a small perturbation and received this). Condition means for boxplots on 1B and 1D are denoted with ◊. Significant main effects of Expectation (†) and Perturbation Size (#) are denoted on the Figures where relevant.*



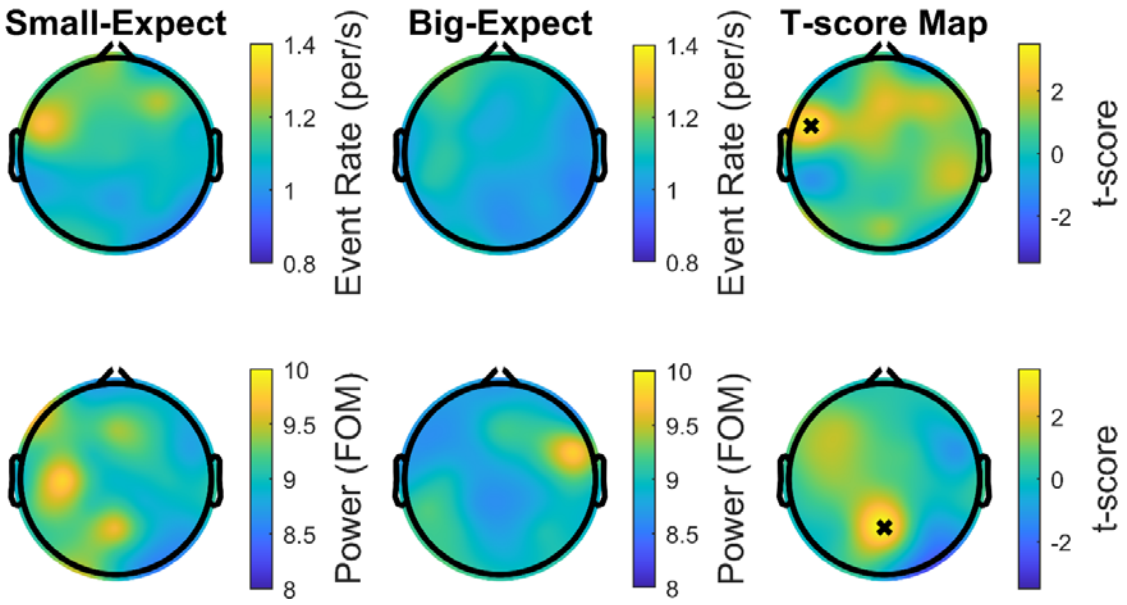
Pre-perturbation anticipatory beta activity

With respect to the EEG analysis, we first explored how expectations shaped anticipatory (pre-perturbation) EEG activity in the 1s window prior to perturbation onset. Due to the transient (or, “burst-like” (31)) nature of beta activity, we analysed the characteristics of individual beta burst-like events (15-29 Hz), rather than averaged pre-stimulus activity. Specifically, we analysed beta event rate (per/s), normalised power of beta events (normalised in factors of median (FOM)), and timing of the most recent beta event (with respect to perturbation onset; with more negative values indicating that the last beta event occurred further from perturbation onset) (24,25). Channel-wise comparisons revealed statistically fewer pre-perturbation beta events during Big-Expectation trials across the

frontal cortex – with this difference reaching statistical significance for electrode FC5 (t = 3.42, p = 0.0027; Figure 2, top row). The mean power of pre-perturbation beta events was also significantly reduced during Big-Expectation trials across electrode Pz (t = 3.63, p = 0.0017; Figure 2, bottom row). Both these differences remained statistically significant when corrected for multiple comparisons ($p < 0.05$). As illustrated in Supplementary Figure 1, the timing of the final beta event (prior to perturbation onset) tended to also be earlier (i.e., further from perturbation onset) during Big-Expectation trials—specifically for frontal (F4 (t = 2.19, p = 0.040) and FC5 (t = 2.79, p = 0.011)) and central electrodes (C4; t = 2.20, p = 0.040). However, these differences did not survive statistical correction for multiple comparisons.

In summary, expectation led to clear modulations in anticipatory EEG activity, with reduced beta activity (both event rate and power of events) observed in trials when expecting bigger perturbations.

Figure 2. *Topographical representation of beta event rate (per/s; top row) and beta power (bottom row) during the one-second prior to perturbation onset. Beta power is presented normalised in factors of median (FOM). X-markers indicate statistically significant differences (p<0.05, corrected for multiple comparisons).*

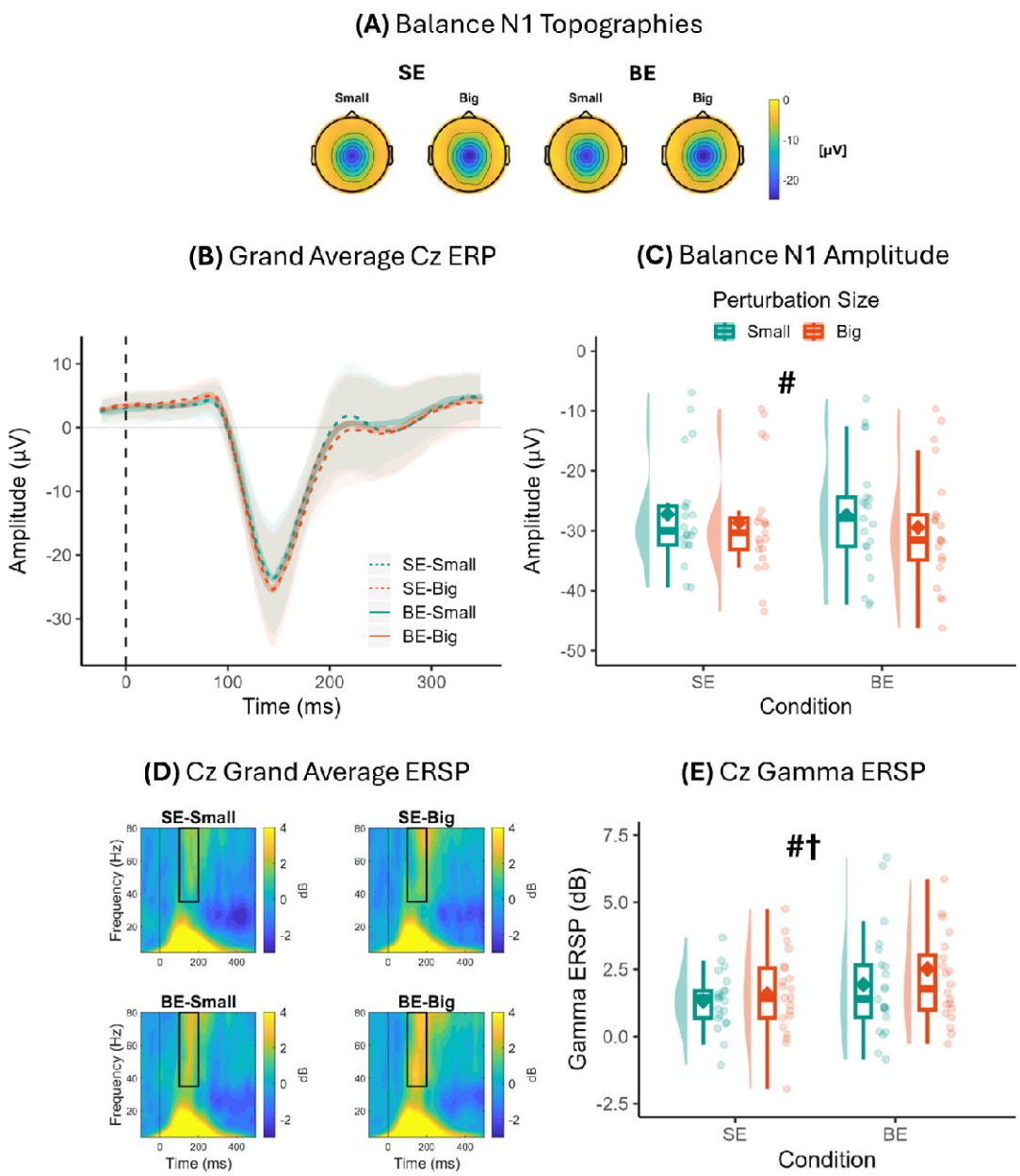


Post-perturbation cortical outcomes

We next analysed post-perturbation cortical activity. Our analyses revealed that the balance N1 scaled to Perturbation Size (χ^2 = 44.39, p < 0.001), but not Expectation (main effect: χ^2 = 0.35, p = 0.553, interaction: χ^2 = 0.857, p = 0.354). As illustrated in Figure 3C, the balance N1 response was significantly larger (i.e. greater negativity) when receiving Big versus Small perturbations, irrespective of expectation. This is therefore in line with the objective balance

outcomes, suggesting that the balance N1 is shaped by the received, rather than expected, sensory signals.

Figure 3. *Post-perturbation cortical responses during the four trial types, including grand average balance N1 scalp topographies (A), grand average (±SD) ERP of channel Cz (B) boxplots, distributions, and individual-participant jitter points of balance N1 amplitudes (C), grand average ERSP plots of channel Cz across each trial type (D), mean Cz gamma (35-80 Hz) ERSP (E). Note, SE reflects Small-Expectation trials whilst BE reflects Big-Expectation trials (e.g., 'SE-Small' indicates expecting a small perturbation and receiving this). Timepoint zero on 3B and 3D indicates perturbation onset. Condition means for boxplots on 3C and 3E are denoted with ◊. Significant main effects of Expectation (†) and Perturbation Size (#) are denoted on the Figures where relevant.*



We next analysed evoked sensorimotor (channel Cz) gamma activity, focusing on a time-window 100-200 ms after perturbation onset (solid black box, Figure 3D). In contrast to the balance N1 which scaled to perturbation size only, post-perturbation gamma activity independently scaled to both Expectation ($\chi^2 = 5.28$, $p = 0.022$) and Perturbation Size ($\chi^2 = 6.77$, $p = 0.009$). The interaction effect was not significant ($\chi^2 = 1.41$, $p = 0.235$). As illustrated in Figure 3E, post-perturbation gamma activity was both higher during Big-Expectation compared to Small-Expectation trials (irrespective of the size of the subequent perturbation), and when receiving Big versus Small perturbations (independent of expectation).

In summary, our results revealed that post-perturbation evoked EEG activity is independently shaped by both expectation and sensory signals. Whilst the balance N1 scaled to the size of the perturbation alone (with no influence of expectation), post-perturbation gamma activity was influenced independently by both expectation and perturbation size. Contrary to our predictions, there was no evidence of either response being shaped by prediction errors, as no significant interaction effects were observed.

Discussion

Our findings provide the first direct evidence that top-down expectations may bias how sensory signals are interpreted at both the perceptual and neural level during balance control. Participants reported feeling more unstable when expecting greater postural instability—even when sensory input (and actual behavioural responses) remained constant. These perceptual changes were accompanied by distinct EEG signatures: expecting greater imbalance reduced anticipatory beta-band activity and increased post-perturbation gamma responses. In contrast, the balance N1 evoked response, as well as behavioural responses themselves, scaled only with perturbation magnitude, unaffected by expectations. Notably, none of the a priori EEG measures reflected prediction error, suggesting that mismatches between expected and actual perturbations do not drive the dominant cortical responses observed during balance control. Instead, it appears that two distinct systems exist: one that is sensitive to top-down expectations and another that is driven by sensory input, independent of expectation or mismatches. We propose that such dual system allows for perceptual stability whilst ensuring that behaviour can be flexibly tuned to rapidly changing environmental contexts.

Top-Down Modulation of Beta and Gamma Activity

Our results highlight two distinct, expectation-sensitive EEG markers: anticipatory beta and post-perturbation gamma. Reduced beta power in sensory regions prior to perturbation onset was observed when participants expected greater instability. This mirrors findings from pain research, where high-expectancy of painful stimuli leads to reduced pre-stimulus beta and increased sensory sensitivity (8). Beta oscillations are commonly interpreted as reflecting cortical inhibition (24,25,32); thus, reductions may reflect a hypervigilant state that primes the system for heightened responsiveness to expected input (33). In the context of balance, this may result in amplified processing of even mild disturbances, which could explain the exaggerated perceptions of instability reported by our participants.

The inhibitory role of cortical beta is not restricted to sensory processing as it also plays a key role in *motor* inhibition, particularly in frontal regions. For example, successful inhibition of a planned reaching action has been linked to increased frontal beta events (34). The demands of the present study required participants to rapidly initiate rather than *inhibit* a motor action to contend with the postural perturbation. As we observed significant reductions in frontal beta events when participants expected the upcoming perturbation to be more destabilising, it is likely that this top-down strategy was employed to facilitate rapid motor

initiation. Together, reductions in sensory and frontal beta suggest distinct yet convergent mechanisms by which expectations prime the sensorimotor system for anticipated imbalance.

Post-perturbation sensorimotor gamma activity (100–200 ms) increased both when participants expected greater instability, and when they experienced it. Although we initially hypothesised that gamma would track prediction error (see (29)), our findings suggest that post-stimulus gamma responses may instead reflect perceived sensory salience more broadly: stimuli that demand greater processing (or which are expected to) elicited stronger gamma activity. This aligns with evidence that gamma oscillations support sensory gain and perceptual amplification (35,36). In our study, gamma responses were shaped by expectation and closely mirrored subjective reports of instability, suggesting that gamma may contribute to the top-down construction of balance perception.

Prediction errors: Hard to predict?

Contrary to our hypotheses, no a priori EEG markers, including the balance N1, tracked the mismatch between expected and received perturbations (i.e., prediction error). Rather, the N1 scaled to the size of the perturbation received, supporting previous work describing larger balance N1 responses during more destabilising events (26,37). Although some have proposed that the balance N1 reflects an ‘error detection’ mechanism (38), our findings support the alternative view that the N1 reflects a cortical (but subconscious) mechanism employed to detect and behaviourally respond to instability rather than signalling error per se. Supporting this interpretation are findings that larger N1 responses are recorded during trials in which participants must take a compensatory step to regain balance following a perturbation (37,39). While prediction errors (likely originating from subcortical areas such as the cerebellum (40)) are known to drive balance behaviour (18,19), our findings suggest these exert little influence over cortical sensorimotor processing or balance perception.

Given the latencies of the cortical pathways associated with reactive balance responses (41), we focused our analyses on early sensorimotor cortical responses (i.e., <200ms). However, it is possible that other neural markers outside this temporal window might instead reflect error processing during balance. For instance, Jalilpour and Muller-Putz (42) found that balance perturbations characterised by a prediction error (participants tilted in opposite direction as expected) were only distinguished from trials that matched expectations by cortical rhythms occurring following the N1. Specifically, they identified post-N1 alpha suppression to be a key marker of error processing (a response often termed ‘error-related alpha suppression’ (42)). In-line with this, our exploratory whole-scalp analyses (see Supplementary File Figure 2) revealed a similar suppression of alpha activity (200–400 ms) over parietal-occipital electrodes following unexpectedly *large* perturbations (i.e., big perturbations during the Small-Expect block), but not following unexpectedly *small* ones. These findings suggest the potential of post-N1 alpha suppression as a neural marker of balance error processing, warranting further investigation in future work.

Implications for Balance Disorders and Predictive Coding

That expectations alone can both distort balance perception and modulate cortical activity has important implications for clinical conditions marked by subjective instability, including functional dizziness (43) and age-related balance disorders (21,44)). Identifying EEG markers of expectation-driven perceptual distortion, particularly anticipatory beta and post-perturbation gamma may offer new diagnostic or monitoring tools for these conditions. Currently, there are none.

More broadly, our findings extend perceptual predictive coding frameworks into the motor domain of balance regulation. Unlike more traditional sensory systems, balance involves complex multisensory integration and rapid subcortical feedback mechanisms (10,11). Yet even here, top-down expectations shape perception and modulate cortical responses. Importantly, unlike pure unisensory processes (4,45,46), these effects do not appear to rely on prediction errors, but rather on direct modulation of neural activity linked to motor preparation and sensory salience.

Conclusions

In sum, our results demonstrate that early cortical processing of imbalance integrates both sensory input and top-down expectations, but not their mismatch. Notably, subjective balance perception was shaped by expectations alone. These findings identify anticipatory beta and post-perturbation gamma as specific EEG markers that may mediate perceptual distortions in balance and motor control more broadly. This work not only advances predictive coding frameworks beyond purely sensory domains but also points to potential neural targets for diagnosing or treating balance disorders rooted in expectation-based distortions (47).

Methods

Participants

Twenty-one neurotypical adults participated in the experiment (11 females, 10 males; M ± SD age = 25.0 ± 4.9 years; height = 173.0 ± 11.0 cm; weight = 73.7 ± 10.9 kg). Participants were free from neurological disease and had no prior experience of dizziness or balance problems. The experiment was approved by the Manchester Metropolitan University ethics committee. Sample size estimates were based on the self-report measure of perceived instability, our primary indicator of expectation manipulation. Prior studies have reported large to very large effects for similar manipulations (48,49). Power analysis for a repeated-measures, within-subjects design ($f = 0.40$, $\alpha = 0.05$, power = 0.95, $r = 0.70$) indicated that 16 participants were sufficient to detect a large effect.

Protocol

Perturbations were delivered by a moveable platform (80 x 60 cm with an embedded force plate recording at 1,000 Hz; Type 9281B, Kistler Instrument). The platform was driven by an electromagnetic actuator and controlled through custom written software (LabVIEW v19 SP1, National Instruments) via DAQ card (USB-6210, National Instruments). Participants

stood on the force plate, with feet shoulder width apart and hands on hips. Foot positioning was marked to ensure consistency. During perturbations, participants were instructed to fixate on a cross marked on the wall at eye level, 4 m away. Participants wore noise-cancelling headphones to minimise any anticipatory audio cues.

Expectation manipulation

Participants experienced two blocks of 60 discrete sine wave perturbations (7-15 s random delay between each perturbation), each consisting of an initial forward translation of the support surface before reversing in direction and completing the sine wave to return to the original position. Both blocks contained a combination of “Small” (maximum forward displacement = 57.0 mm; peak acceleration = 1,472 mm/s^2; peak acceleration latency = 70 ms; see Figure 1) and “Big” perturbations (maximum forward displacement = 73.3 mm, peak acceleration = 1,965 mm/s^2; peak acceleration latency = 60 ms; see Figure 1A). Prior to completing the two experimental blocks of trials (described below), participants received 5 small and 5 big perturbations (order randomised across participants) to enhance the formation of their expectations.

In the Small-Expectation (SE) block of trials, participants were informed they would encounter small perturbations throughout the block of 60 trials. This block commenced with ten consecutive small perturbations, reinforcing their expectation. The remaining 50 perturbations comprised a predetermined sequence of 35 small and 15 big (expectation-violating) perturbations. These expectancy-violating perturbations were introduced on average every third trial and were never presented in succession. The Big-Expectation (BE) block of trials followed the same structure but with the perturbation sizes reversed. The sequence of perturbations was fixed and identical for all participants, ensuring consistency across the cohort, and the presentation of the two blocks of trials was counterbalanced across participants. For analyses, we compared 15 Small perturbations from the Small-Expectation block to 15 Small perturbations from the Big-Expectation block, and 15 Big perturbations from the Big-Expectation block to 15 Big perturbations from the Small-Expectation block. To ensure a consistent and unbiased selection, the 15 trials chosen from the expectancy-aligned conditions (out of the available 45) were those immediately preceding a trial that violated expectancy. This selection criterion was applied for two key reasons: (a) the first 10 trials of each block were excluded to avoid confounding effects of early adaptation, and (b) the selected trials were always maximally distant from a preceding expectancy-violating perturbation, reducing potential carryover effects.

Both perturbation sizes (small and big) were designed to challenge postural stability without necessitating a corrective stepping response. To prevent fatigue, participants received a 5–10 minute break between each block of 60 perturbations.

Self-reported outcomes

After certain trials, participants completed self-reported questionnaires assessing their perceived instability and anxiety during the preceding trial (17). For perceived instability,

participants were asked “how unstable did you feel during the trial?” and responded via a visual analogue scale (0 = “completely steady”, 10 = “so unsteady that I would fall”). Participants were then asked to rate the level of anxiety they experienced in the preceding trial via a visual analogue scale that ranged from 0 “not at all anxious” to 10 “extremely anxious” (17,26). These questionnaires were completed after two random big and small perturbations, in each expectation block.

EEG recording and analyses

EEG signals were recorded at 1000 Hz from 29 active electrodes (eego sports, ANT Neuro, Hengelo, Netherlands) positioned according to the extended 10–20 international system. Signals were band-pass filtered (1–80 Hz), epoched (−2 to +2 s relative to perturbation onset) and averaged referenced. Independent component analysis (ICA; RunICA informax) were then conduced to manually reject non-neural activity (average of 23.5 ± 1.9 components retained per participant).

Pre-perturbation cortical beta (15 – 29 Hz) activity was analysed across the 1 s pre-perturbation window, given its established role in modulating subsequent stimulus perception (24,25,31) and behavioural response (50,51). Following the methods of Shin et al. (25), we measured three key outcome measures using the open-source ‘SpectralEvents Toolbox’ within MATLAB (52): beta event rate (per/s), normalised power of beta events (normalised in factors of median (FOM)), and timing of the most recent beta event (with respect to perturbation onset; ranging from -1 to 0 s). Please see Supplementary Materials for further details about the analyses employed.

For post-perturbation data, we calculated the ‘balance N1’ for each trial as the largest negative peak occurring at electrode Cz 50-200ms after perturbation (following baseline correction [-1000 to -250 ms]; 27,28). We also calculated Event-Related Spectral Power (ERSP) for electrode Cz, using complex Morlet wavelets. We used 77 frequencies linearly spaced between 4 and 80 Hz, with wavelets logarithmically spaced from 3 to 8 cycles. Data were baseline normalised to average activity from -500 to -200 ms prior to perturbation onset. We chose to focus on gamma based on the well-documented role of cortical gamma as a robust prediction error signal across tasks and sensory modalities (29,30). We limited our analysis to 35-80Hz (to avoid also capturing any beta activity) 100-200 ms post-perturbation onset. This window of interest was selected based on condition-average inspection that revealed gamma-band activity to be strongest within this boundary (53).

Postural response to perturbations

We used custom MATLAB scripts to determine the peak velocity of anterior-posterior (AP) centre of pressure (COP) data in response to the initial forward portion of the perturbation. 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 (54,55). First, 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 (26).

Statistical analyses

Subjective and objective balance outcomes. Subjective (perceived instability and anxiety) and objective balance outcomes (AP COP velocity) were analysed using separate generalized estimating equations (GEE) analyses to explore the effects of Expectation (SE and BE) and Perturbation Size (Small and Big). We chose an exchangeable working correlation matrix to define dependency amongst measurements. Significant main and interaction effects were followed up with Bonferroni-corrected post-hoc pairwise comparisons. Threshold for significance was set at 0.05.

Pre-perturbation analyses. To assess the influence of expectation on preparatory cortical activity, trial-level beta outcomes were averaged across all trials within an expectation block. This approach enabled us to specifically examine how Expectation (i.e., Small-Expectation vs. Big-Expectation) modulated preparatory neural activity prior to perturbation onset. Given this activity occurred *before* the delivery of an expectancy-violating trial, averaging within blocks provided the most appropriate means of capturing expectation-driven cortical dynamics (and served to maximise analytical power). To explore expectation-driven topographical differences between experimental blocks in pre-perturbation beta activity, we performed channel-wise paired samples t-tests. To control for inflated Type I error due to multiple comparisons across EEG channels, we employed a non-parametric permutation-based "maximum statistic" method (53).

Post-perturbation outcomes. We used generalized estimating equations (GEE) analyses to explore the effects of Expectation (SE and BE) and Perturbation Size (Small and Big) on both the balance N1 and post-perturbation gamma. This approach was deemed the most appropriate for our time-frequency analyses given the difficulties associated with applying permutation testing to factorial designs as in the present study (e.g., Expectation x Perturbation Size; see (53)). As with the balance outcomes described above, we chose an exchangeable working correlation matrix. Significant main and interaction effects were followed up with Bonferroni-corrected post-hoc pairwise comparisons. Threshold for significance was set at 0.05.

References

1. Clark A. Whatever next? Predictive brains, situated agents, and the future of cognitive science. Behav Brain Sci. 2013 Jun;36(3):181–204.

2. de Lange FP, Heilbron M, Kok P. How Do Expectations Shape Perception? Trends Cogn Sci. 2018 Sep 1;22(9):764–79.

3. Kok P, Jehee JFM, de Lange FP. Less Is More: Expectation Sharpens Representations in the Primary Visual Cortex. Neuron. 2012 Jul 26;75(2):265–70.

4. Rao RPN, Ballard DH. Predictive coding in the visual cortex: a functional interpretation of some extra-classical receptive-field effects. Nat Neurosci. 1999 Jan;2(1):79–87.

5. Wacongne C, Labyt E, van Wassenhove V, Bekinschtein T, Naccache L, Dehaene S. Evidence for a hierarchy of predictions and prediction errors in human cortex. Proc Natl Acad Sci. 2011 Dec 20;108(51):20754–9.

6. Bendixen A, SanMiguel I, Schröger E. Early electrophysiological indicators for predictive processing in audition: A review. Int J Psychophysiol. 2012 Feb 1;83(2):120–31.

7. Strube A, Rose M, Fazeli S, Büchel C. The temporal and spectral characteristics of expectations and prediction errors in pain and thermoception. Kok P, de Lange FP, Schulz E, editors. eLife. 2021 Feb 17;10:e62809.

8. Nickel MM, Tiemann L, Hohn VD, May ES, Gil Ávila C, Eippert F, et al. Temporal–spectral signaling of sensory information and expectations in the cerebral processing of pain. Proc Natl Acad Sci. 2022 Jan 4;119(1):e2116616119.

9. Wittkamp CA, Wolf MI, Rose M. The neural dynamics of positive and negative expectations of pain. Biurrun Manresa J, Behrens TE, editors. eLife. 2024 Dec 2;13:RP97793.

10. Welch TDJ, Ting LH. A Feedback Model Reproduces Muscle Activity During Human Postural Responses to Support-Surface Translations. J Neurophysiol. 2008 Feb;99(2):1032–8.

11. Welch TDJ, Ting LH. Mechanisms of Motor Adaptation in Reactive Balance Control. PLOS ONE. 2014 May 8;9(5):e96440.

12. Peterka RJ. Sensorimotor Integration in Human Postural Control. J Neurophysiol. 2002 Sep;88(3):1097–118.

13. Bronstein AM. Chapter 4 - Multisensory integration in balance control. In: Furman JM, Lempert T, editors. Handbook of Clinical Neurology [Internet]. Elsevier; 2016 [cited 2025 Jul 25]. p. 57–66. (Neuro-Otology; vol. 137). Available from: https://www.sciencedirect.com/science/article/pii/B9780444634375000042

14. Fitzpatrick RC, Taylor JL, McCloskey DI. Ankle stiffness of standing humans in response to imperceptible perturbation: reflex and task-dependent components. J Physiol. 1992;454(1):533–47.

15. Luu BL, Inglis JT, Huryn TP, Van der Loos HFM, Croft EA, Blouin JS. Human standing is modified by an unconscious integration of congruent sensory and motor signals. J Physiol. 2012;590(22):5783–94.

16. Cleworth TW, Carpenter MG. Postural threat influences conscious perception of postural sway. Neurosci Lett. 2016 May 4;620:127–31.

17. Castro P, Kaski D, Schieppati M, Furman M, Arshad Q, Bronstein A. Subjective stability perception is related to postural anxiety in older subjects. Gait Posture. 2019 Feb;68:538–44.

18. Torres-Oviedo G, Vasudevan E, Malone L, Bastian AJ. Chapter 4 - Locomotor adaptation. In: Green AM, Chapman CE, Kalaska JF, Lepore F, editors. Progress in Brain Research [Internet]. Elsevier; 2011 [cited 2025 Aug 8]. p. 65–74. (Enhancing Performance for Action and Perception; vol. 191). Available from: https://www.sciencedirect.com/science/article/pii/B9780444537522000138

19. Bronstein AM, Bunday KL, Reynolds R. What the “Broken Escalator” Phenomenon Teaches Us about Balance. Ann N Y Acad Sci. 2009;1164(1):82–8.

20. Ellmers TJ, Kal EC, Young WR. Consciously processing balance leads to distorted perceptions of instability in older adults. J Neurol. 2021 Apr;268(4):1374–84.

21. Castro P, Ibitoye R, Ellmers T, Kaski D, Arshad Q, Bronstein AM. Towards an explanation for ‘unexplained’ dizziness in older people. Age Ageing. 2024 Jul 2;53(7):afae137.

22. Adkin AL, Quant S, Maki BE, McIlroy WE. Cortical responses associated with predictable and unpredictable compensatory balance reactions. Exp Brain Res. 2006 Jun 1;172(1):85–93.

23. Mochizuki G, Sibley KM, Cheung HJ, Camilleri JM, McIlroy WE. Generalizability of perturbation-evoked cortical potentials: Independence from sensory, motor and overall postural state. Neurosci Lett. 2009 Feb 13;451(1):40–4.

24. Mirdamadi JL, Ting LH, Borich MR. Distinct Cortical Correlates of Perception and Motor Function in Balance Control. J Neurosci [Internet]. 2024 Apr 10 [cited 2025 Apr 2];44(15). Available from: https://www.jneurosci.org/content/44/15/e1520232024

25. Shin H, Law R, Tsutsui S, Moore CI, Jones SR. The rate of transient beta frequency events predicts behavior across tasks and species. Kajikawa Y, editor. eLife. 2017 Nov 6;6:e29086.

26. Parr JVV, Mills R, Kal E, Bronstein AM, Ellmers TJ. A “Conscious” Loss of Balance: Directing Attention to Movement Can Impair the Cortical Response to Postural Perturbations. J Neurosci [Internet]. 2024 Nov 27 [cited 2025 Apr 2];44(48). Available from: https://www.jneurosci.org/content/44/48/e0810242024

27. Varghese JP, McIlroy RE, Barnett-Cowan M. Perturbation-evoked potentials: Significance and application in balance control research. Neurosci Biobehav Rev. 2017 Dec 1;83:267–80.

28. Payne AM, Ting LH. Worse balance is associated with larger perturbation-evoked cortical responses in healthy young adults. Gait Posture. 2020 Jul 1;80:324–30.

29. Arnal LH, Giraud AL. Cortical oscillations and sensory predictions. Trends Cogn Sci. 2012 Jul 1;16(7):390–8.

30. van Pelt S, Heil L, Kwisthout J, Ondobaka S, van Rooij I, Bekkering H. Beta- and gamma-band activity reflect predictive coding in the processing of causal events. Soc Cogn Affect Neurosci. 2016 Jun;11(6):973–80.

31. Lundqvist M, Miller EK, Nordmark J, Liljefors J, Herman P. Beta: bursts of cognition. Trends Cogn Sci. 2024 Jul 1;28(7):662–76.

32. Forster C, Stephani T, Grund M, Panagoulas E, Al E, Hofmann SM, et al. Pre-stimulus beta power mediates explicit and implicit perceptual biases in distinct cortical areas. Commun Psychol. 2025 Jun 21;3(1):93.

33. Ellmers TJ, Kal EC. Exploring the role of attention towards balance in chronic dizziness: Development of the Balance Vigilance Questionnaire. Eur J Neurol. 2024;31(3):e16148.

34. Jana S, Hannah R, Muralidharan V, Aron AR. Temporal cascade of frontal, motor and muscle processes underlying human action-stopping. van den Wildenberg W, Ivry RB, van den Wildenberg W, Huster R, Bissett PG, editors. eLife. 2020 Mar 18;9:e50371.

35. Merker B. Cortical gamma oscillations: the functional key is activation, not cognition. Neurosci Biobehav Rev. 2013 Mar 1;37(3):401–17.

36. Pritchett DL, Siegle JH, Deister CA, Moore CI. For things needing your attention: the role of neocortical gamma in sensory perception. Curr Opin Neurobiol. 2015 Apr 1;31:254–63.

37. Solis-Escalante T, Stokkermans M, Cohen MX, Weerdesteyn V. Cortical responses to whole-body balance perturbations index perturbation magnitude and predict reactive stepping behavior. Eur J Neurosci. 2021;54(12):8120–38.

38. Payne AM, Ting LH, Hajcak G. Do sensorimotor perturbations to standing balance elicit an error-related negativity? Psychophysiology. 2019;56(7):e13359.

39. Payne AM, Ting LH. Balance perturbation-evoked cortical N1 responses are larger when stepping and not influenced by motor planning. J Neurophysiol. 2020 Dec 1;124(6):1875–84.

40. Morton SM, Bastian AJ. Cerebellar Contributions to Locomotor Adaptations during Splitbelt Treadmill Walking. J Neurosci. 2006 Sep 6;26(36):9107–16.

41. Boebinger S, Payne A, Martino G, Kerr K, Mirdamadi J, McKay JL, et al. Precise cortical contributions to sensorimotor feedback control during reactive balance. PLOS Comput Biol. 2024 Apr 17;20(4):e1011562.

42. Jalilpour S, Müller-Putz G. Balance perturbation and error processing elicit distinct brain dynamics. J Neural Eng. 2023 Mar;20(2):026026.

43. San Pedro Murillo E, Bancroft MJ, Koohi N, Castro P, Kaski D. Postural misperception: a biomarker for persistent postural perceptual dizziness. J Neurol Neurosurg Psychiatry. 2023 Feb;94(2):165–6.

44. Ellmers TJ, Ibitoye R, Castro P, Kal EC, Kaski D, Bronstein AM. Chronic dizziness in older adults: Disrupted sensorimotor EEG beta oscillations during postural instability. Clin Neurophysiol. 2025 Jun 1;174:31–6.

45. Rust NC, Cohen MR. Priority coding in the visual system. Nat Rev Neurosci. 2022 Jun;23(6):376–88.

46. Furutachi S, Franklin AD, Aldea AM, Mrsic-Flogel TD, Hofer SB. Cooperative thalamocortical circuit mechanism for sensory prediction errors. Nature. 2024 Sep;633(8029):398–406.

47. Harris DJ, Wilkinson S, Ellmers TJ. From fear of falling to choking under pressure: A predictive processing perspective of disrupted motor control under anxiety. Neurosci Biobehav Rev. 2023 May;148:105115.

48. Russell K, Duncan M, Price M, Mosewich A, Ellmers T, Hill M. A comparison of placebo and nocebo effects on objective and subjective postural stability: a double-edged sword? Front Hum Neurosci. 2022;16:967722.

49. Castro P, Papoutselou E, Mahmoud S, Hussain S, Bassaletti CF, Kaski D, et al. Priming overconfidence in belief systems reveals negative return on postural control mechanisms. Gait Posture. 2022 May 1;94:1–8.

50. Wessel JR. β-Bursts Reveal the Trial-to-Trial Dynamics of Movement Initiation and Cancellation. J Neurosci. 2020 Jan 8;40(2):411–23.

51. Little S, Bonaiuto J, Barnes G, Bestmann S. Human motor cortical beta bursts relate to movement planning and response errors. PLOS Biol. 2019 Oct 4;17(10):e3000479.

52. jonescompneurolab/SpectralEvents [Internet]. jonescompneurolab; 2025 [cited 2025 Jun 24]. Available from: https://github.com/jonescompneurolab/SpectralEvents

53. Cohen MX. Analyzing Neural Time Series Data: Theory and Practice. MIT Press; 2014. 615 p.

54. Masani K, Vette AH, Abe MO, Nakazawa K. Center of pressure velocity reflects body acceleration rather than body velocity during quiet standing. Gait Posture. 2014 Mar 1;39(3):946–52.

55. Hewson DJ, Singh NK, Snoussi H, Duchêne J. Classification of elderly as fallers and non-fallers using Centre of Pressure velocity. In: 2010 Annual International Conference of the IEEE Engineering in Medicine and Biology [Internet]. 2010 [cited 2025 Jun 24]. p. 3678–81. Available from: https://ieeexplore.ieee.org/abstract/document/5627649