

Tracking Neural Activity Underlying Postural Control Dysfunction in a VR-Induced System: Demonstration Using BioVRSea

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Abstract—Disrupted postural control (PC), a hallmark of neurological conditions such as Parkinson’s disease (PD) and mild traumatic brain injury (mTBI), is associated with altered neural oscillatory dynamics measured using electroencephalography (EEG). However, few studies have employed ecologically valid paradigms to assess these dynamics. In this study, we utilized BioVRSea, a multimodal virtual reality (VR) platform combining realistic postural perturbations with real-time EEG to examine cortical responses to multisensory stimulation. We tested whether EEG time–frequency representations could differentiate pathological PC responses in PD and mTBI from normative function in matched healthy controls (HC). Motion sickness susceptibility and symptom changes were evaluated using the Motion Sickness Susceptibility Questionnaire (MSSQ) and the Motion Sickness Symptoms Scale. In HC, only the older subgroup exhibited a significant increase in low-frequency (delta–theta, $\approx 1\text{--}7$ Hz) power at 50% perturbation intensity and during the post-perturbation phase (cluster-level $p < 0.05$). Compared with matched controls, PD participants showed increased spectral power in both the delta–theta ($\approx 1\text{--}7$ Hz) and alpha–beta ($\approx 10\text{--}30$ Hz) ranges (cluster-level $p < 0.05$), particularly at higher perturbation intensities, suggesting impaired sensorimotor

integration. In contrast, no significant EEG differences were observed between the mTBI group and matched controls ($p > 0.05$). These findings demonstrate the feasibility of synchronized VR–EEG paradigms for capturing challenge-dependent neural signatures of postural control dysfunction. Further validation is warranted to determine their potential clinical utility in neurological assessment.

Index Terms—Virtual reality (VR), electroencephalography (EEG), postural control, neurorehabilitation, motion sickness, parkinson’s disease, traumatic brain injury, sensorimotor integration.

I. INTRODUCTION

THE incorporation of virtual reality (VR) technology into biosignal systems has opened new avenues for investigating human physiological responses in ecologically valid, simulated environments [9], [10]. Initially introduced to explore the mechanisms of postural control (PC) and motion sickness (MS), the development of BioVRSea represents a major advancement by integrating EEG and other biosignals within an immersive VR platform [11]. PC reflects a complex interaction among visual, vestibular, proprioceptive, somatosensory, musculoskeletal, autonomic, and central systems that collectively maintain stability and spatial orientation [12], [13]. Dysfunction in PC is frequently observed in early neurodegenerative and neuropsychiatric disorders such as PD and mTBI. Evidence indicates that their pathophysiology often involves disruption or hyperactivation of postural mechanisms [1], [3], [7]. Such disturbances—whether caused by neurological pathology or excessive sensory stimulation—can manifest as MS.

Advanced paradigms employing complex PC tasks have provided mechanistic insight and diagnostic potential [14], [15]. The BioVRSea paradigm provides an ecologically valid framework for investigating human PC by combining immersive VR with platform-induced biomechanical perturbations and multimodal physiological monitoring. By manipulating visual and somatosensory inputs, it elicits coordinated central responses measurable through EEG, electromyography (EMG), heart rate variability (HRV), and center-of-pressure (CoP) metrics. Rather than treating MS as a separate phenomenon, this approach considers it a boundary condition of postural stability, where multisensory integration and motor

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prediction limits are tested. In this way, BioVRSea unifies the study of balance regulation and sensory conflict, offering a systems-level view of embodied control.

Aubonnet et al. [16] demonstrated that multimodal responses form distinct neurophysiological signatures of postural adaptation, confirming BioVRSea's reliability for probing sensorimotor dynamics. Subsequent studies extended its scope to population variability: in early-stage PD, Jacob et al. [7] reported impaired neural modulation and altered sway patterns, revealing quantifiable sensory-integration deficits. This sensitivity allows BioVRSea to differentiate pathological and healthy control profiles using data-driven approaches [14]. Lifestyle-related differences have also been identified—biometric responses differ significantly between sedentary and physically active individuals—highlighting the interplay between physiological resilience and balance performance [15], [17].

Within this broader framework, MS emerges as an expression of compromised postural adaptation under sensory stress. Rooted in conflicts between visual and vestibular signals or prolonged instability, MS reflects the nervous system's failure to reconcile mismatched sensory cues or to execute timely compensatory adjustments [5], [18]. The BioVRSea platform exposes participants to such perturbations, oscillating between stable and destabilizing phases—creating a continuum linking balance maintenance and motion sensitivity. Studies show that individuals with greater sway amplitude or delayed neuromuscular responses are more prone to MS symptoms, supporting the postural-instability theory of motion sickness [19], [20], [21]. Physiological markers derived from BioVRSea and related studies help identify the threshold where adaptive control deteriorates into symptom onset.

In addition to characterizing cortical dynamics of postural control, the present study examines motion sickness symptoms as a behavioral correlate of multisensory conflict and postural adaptation during graded perturbations. Taken together, these findings motivate a hypothesis-driven evaluation of frequency-specific cortical dynamics during graded multisensory perturbations.

EEG spectral changes correlate with MS severity [22], [23], [24]; electrogastrography (EGG) reflects nausea-related gastric dysrhythmias [25], [26]; and electro-oculography (EOG) tracks visually induced instability [27], [28]. Autonomic measures—skin conductance, heart rate, blood pressure, and body temperature—are also sensitive to motion-induced stress [24]. Functional MRI reveals altered activity in brainstem and vestibular regions during MS episodes [8], [29], [30], while postural instability measured via CoP predicts MS susceptibility [31]. EEG remains central among these modalities for characterizing neural dynamics in PC and MS [8], [32]. For instance, cortical activity during balance involves decreased alpha-band connectivity between occipital and parietal regions during visual perturbations and increased theta-band connectivity around the supplementary motor area during mechanical perturbations [16]. EEG has also revealed correlations between motor responses, sensory processing, and oscillatory patterns, contributing to biomarker discovery in PD and mTBI [5], [7], [23], [24].

Recent work using controlled VR visual motion has demonstrated persistent postural control deficits following mTBI, highlighting that visual-flow perturbations can unmask impairments not evident under static conditions [33]. Similarly, studies in PD report reduced flexibility of postural strategies during visual perturbations, consistent with altered sensorimotor integration under sensory conflict [34]. In parallel, research on visually induced motion sickness (VIMS) shows that immersive VR stimulation produces systematic neurophysiological changes measurable with EEG, supporting the use of task-based neural markers beyond resting-state traits [9], [35]. However, many of these paradigms rely primarily on visual perturbations alone.

BioVRSea extends prior VR-based postural paradigms by integrating synchronized multisensory perturbations, combining immersive visual stimulation with reproducible platform motion to enable graded and standardized balance challenges during concurrent EEG acquisition (with EMG/HRV/CoP) [5], [11], [36]. In contrast to earlier VR approaches that primarily assessed postural stability before and after immersion or relied predominantly on visual perturbations alone [4], this synchronized visual-mechanical framework permits controlled investigation of cortical dynamics under multisensory conflict. Because cortical contributions to balance are most evident during perturbation responses and sensory reweighting rather than during quiet stance [3], [13], such a design is particularly suited to probe functional sensorimotor integration mechanisms relevant to PD and mTBI. Prior BioVRSea investigations further demonstrate that this multimodal paradigm elicits measurable neurophysiological responses and reveals population-specific alterations across clinical cohorts [7], [11], [16].

The main contributions of this work are:

- A synchronized VR-mechanical perturbation paradigm with graded intensity (PRE, 25%, 50%, 75%, POST), enabling systematic manipulation of multisensory load during balance tasks.
- Time-frequency
- EEG characterization of cortical dynamics during ecologically valid postural perturbations.
- A cross-group evaluation (HC, early-stage PD, mTBI) demonstrating the feasibility of identifying challenge-dependent cortical signatures associated with postural control dysfunction.

Building on this literature, the present study investigates cortical oscillatory dynamics during BioVRSea-induced postural perturbations across healthy, mTBI, and PD groups using time-frequency analysis of EEG data. These hypotheses are grounded in prior evidence that postural perturbations modulate cortical oscillations associated with attentional engagement and sensorimotor control. In particular, theta activity has been linked to cognitive-attentional demands during balance tasks, beta oscillations to sensorimotor integration and motor control processes, and alpha dynamics to sensory gating and adaptation during multisensory conflict. Aging, PD, and mTBI have been shown to differentially alter these oscillatory mechanisms during balance-challenging

conditions. Given this framework, we examined whether graded and synchronized visual-mechanical perturbations would elicit frequency-specific cortical responses that differ across healthy and clinical populations.

Specifically, we aim to:

1. Characterize event-related brain oscillations across five BioVRSea phases with varying levels of visual and physical perturbation.
We hypothesize that healthy individuals will exhibit increased theta and beta modulation during visuo-motor phases (25–75% movement).
2. Compare EEG responses in early-stage PD patients and age-matched healthy controls.
We expect that PD participants will exhibit altered beta modulation and reduced alpha suppression relative to controls.
3. Evaluate oscillatory patterns in mTBI participants relative to matched controls.
We hypothesize that mTBI individuals will display altered theta and alpha modulation, particularly during visual and high-load movement phases, consistent with impaired multisensory integration.
4. Track within-group changes across phases to examine dynamic adaptation during graded perturbations.
We predict that group differences will be most pronounced during the highest perturbation intensity (75%) and the post-movement phase, when sensory reweighting and balance stabilization demands are maximal.

II. METHOD

A. Participants

1) **Healthy Controls:** A total of 355 healthy, right-handed, and educated adults (169 females, aged 20–73) participated in the study. All participants had normal or corrected-to-normal vision and no diagnosed or reported hearing impairments. Individuals with a history of neurological or psychiatric disorders, medication use, or related symptoms were excluded from this cohort. Recruitment of the healthy participants was conducted via social-media advertisements at Reykjavík University and the University of Iceland, as well as announcements in Icelandic newspapers.

From this cohort, a subset of healthy participants was selected for analysis based on age-related grouping and clinical-group matching. To examine age-related differences in neural oscillatory dynamics, 100 individuals were assigned to one of two age-based subgroups: a young group ($N = 49$, 23 females, aged 18–30) and an old group ($N = 51$, 21 females, aged 50–73). These groups were selected to reflect distinct age distributions while minimizing overlap with middle-aged participants.

For patient comparisons, two additional control subgroups were drawn from the healthy cohort. One control group ($N = 49$, all female, aged 21–40) was matched to the mTBI group based on age and sex, and another control group ($N = 35$, 16 females, aged 49–73) was matched to the PD group. Matching criteria included age, sex, and sample size, allowing for balanced group-level comparisons of oscillatory dynamics between clinical and non-clinical populations.

2) **Early-Stage Parkinson's Disease:** Individuals diagnosed with early-stage PD were evaluated based on the Hoehn and Yahr criteria [37]. Within this cohort, 42 individuals classified as Hoehn and Yahr stage I–III were initially screened; however, those with cognitive impairment or advanced disease stages were excluded. A total of 22 early-stage PD participants (8 females, aged 56–82 years) were included. Some participants exhibited mild tremors or early signs of postural instability, indicative of early-stage PD. All individuals in this group were undergoing Levodopa treatment as part of their standard care regimen.

3) **Sports-Related mTBI:** A total of 36 female athletes with a history of sports-related mTBI were recruited using snowball sampling through the mTBI research group at Reykjavík University. These individuals were aged 18–40 years and were either active or retired athletes who had participated in high-risk contact sports. The mTBI history of participants was confirmed using a self-reported online questionnaire (SCAT5) [38]. The SCAT5 is a standardized tool for assessing suspected mTBI in individuals aged 13 and older. It includes an on-field assessment for red-flag symptoms, memory testing, GCS evaluation, and cervical-spine checks, along with an off-field evaluation covering history, symptoms, cognitive and neurological screening, and delayed recall.

4) **Ethical Approval and Study Environment:** The study was conducted at the Institute of Biomedical and Neural Engineering at Reykjavík University, in the Motion Sickness and Postural Control Lab. All participants voluntarily took part and provided written informed consent. The study received ethical approval from the Reykjavík University Ethics Committee (Tilv.: VSNb2020050009/03.01), ensuring that all procedures complied with institutional and legal standards. Each participant attended a single experimental session and was fully briefed before participation; comfort and safety were prioritized throughout.

B. Task Design

1) Behavioral Measurements:

a) **Motion sickness susceptibility questionnaire (MSSQ):** Before participating in the study, all participants completed the Motion Sickness Susceptibility Questionnaire (MSSQ) as proposed by Golding [6]. The MSSQ evaluates an individual's susceptibility to motion sickness across multiple modes of transport, including cars, buses, trains, airplanes, small boats, ships, swings, ferris wheels, and amusement-park rides. Participants rated their experience of motion sickness on a scale from 0 (never) to 3 (frequently) for each category. The total MSSQ score was computed following the procedure described by Golding [6], with a maximum possible score of 27. This score was used to quantify each participant's baseline susceptibility to motion sickness.

b) **Motion sickness symptoms (MSS) scale:** To assess the impact of the BioVRSea paradigm on motion sickness, participants completed a symptom questionnaire immediately before and after the experiment. The ten-item symptom set (general discomfort, fatigue, headache, eye strain, difficulty focusing or concentrating, increased salivation, sweating, nausea,

blurred vision, dizziness/vertigo) was adapted from motion-sickness symptom frameworks in the MSSQ lineage [6], while adopting a pre/post symptom-rating approach consistent with established simulator/VR methodology [4], [9], [10]. Each symptom was rated on a 0–3 scale (0 = none; 3 = severe). The final MSS score for each participant was computed as the post- minus pre-experiment average symptom rating, yielding a single value (range 0–3) indexing the net change in motion-sickness symptom severity attributable to BioVRSea exposure.

2) VR-EEG Framework: The BioVRSea platform was developed to simulate a realistic open-sea environment in which participants experienced motion analogous to being on a boat in a laboratory setting. The setup includes a six-degree-of-freedom (DoF) simulation platform developed by Motion-Systems, integrated with a VR system and two fixed force plates from Virtualis to simulate wave motion and measure ground-reaction forces. The moving platform was designed to simulate waves according to the virtual environment. The operator adjusted the wave frequency between 0.5 and 3 Hz and the amplitude from 0 to 2, with platform-movement intensity ranging between 0 % and 100 %. The platform was capable of executing rapid, customized movements at angles of 0°–45°–90°–135°–180°–225°–270°–315°–360°, allowing for synchronized multimodal physiological acquisition using an ANT Neuro 64-channel EEG, a Polar heart-rate monitor, Kiso wireless EMG sensors, and motion-tracking systems [5], [7], [11], [14], [15], [17].

As illustrated in Fig. 1, participants stood on a motion platform while wearing a VR headset that displays a seascape from the perspective of a virtual boat. The platform reproduces oscillatory movements synchronized with the VR visuals, creating a realistic and dynamic balance challenge. The simulation is specifically designed to destabilize the participant and elicit continuous postural adjustments. The platform's oscillations increase in intensity over time, with the final phase representing the most demanding balance condition. This graded approach allows for the systematic examination of postural responses under varying sensory and motor demands [4].

3) Wave Frequency Selection: The selection of wave frequencies was guided by two considerations. (1) *Theoretical models of motion sickness.* Sensory-conflict accounts posit that discrepancies across visual, vestibular, and proprioceptive inputs drive discomfort, whereas the postural-instability theory (PIT) emphasizes that instability in body sway—especially at low oscillation rates—precedes symptom onset. Accordingly, very low frequencies (<0.5–1 Hz) were avoided because they promote prolonged instability and facilitate motion-sickness onset [39], [40], [41]. (2) *Safety–challenge balance.* A 1 Hz protocol provided a milder condition to reduce fall risk, whereas 3 Hz introduced a more demanding dynamic challenge to elicit robust vestibulo-postural responses [42].

5) Task Execution: Participants began the protocol by standing still for 120 s with their arms relaxed at their sides while viewing a static mountain landscape in VR. This period included 45 s for sensor stabilization, followed by 60 s serving

as the resting baseline phase. A 15-s white-screen transition was then used to shift into the dynamic sea simulation.

In the *pre-movement (PRE)* phase, participants were exposed to wave-like visual motion through the VR headset, simulating sea conditions without any physical platform movement, to evoke visually induced postural responses.

The movement (MOV) phase followed, during which the platform oscillated in synchrony with the VR waves at three increasing intensity levels—25%, 50%, and 75%—each constituting a distinct sub-phase. The visual scene and platform-motion profile were identical for all participants, and perturbation intensities were applied in a fixed, progressive order (25% → 50% → 75%), without pseudo-randomization. This standardized progression ensured consistent multisensory stimulation and comparability across participants while modeling gradually increasing balance load. Participants held a support bar while experiencing multisensory perturbations engaging vestibular, visual, and proprioceptive systems.

In the *post-movement (POST)* phase, the platform came to an abrupt stop while the VR sea scene continued. Participants were instructed to release the support bar and maintain balance using visual cues alone. Before beginning the experimental sequence, all participants received standardized instructions regarding posture, hand placement, and task execution to ensure procedural consistency.

All participants received standardized verbal instructions prior to the experimental sequence and before each phase transition. During the baseline phase, participants were instructed to stand upright with arms alongside the body, remain as still as possible, and maintain gaze on the VR scene. During the PRE (visual-only) phase, they were instructed to maintain an upright posture and continue focusing on the visual environment without physical movement. During the MOV phases (25%, 50%, and 75%), participants were instructed to hold the support bars, maintain upright posture, and allow natural postural adjustments while following the visual scene. During the POST phase, participants were instructed to release the support bars and maintain balance using visual information only. Brief verbal cues were provided at each phase transition to ensure compliance and consistency across participants.

Handrail use was directly monitored by the experimenters throughout the protocol. Participants were explicitly instructed when to hold the support bars (MOV phases) and when to avoid using them (PRE and POST phases). During the POST phase, release of the support bars was visually confirmed by the experimenter. Trials in which participants continued to use the handrails during the POST phase were documented and excluded from subsequent EEG and behavioral analyses to ensure consistency of stabilization measurements.

6) EEG Acquisition and Preprocessing: EEG data were recorded with a 64-channel portable system (ANT Neuro) at 4096 Hz using *ego* software, with CPz as the acquisition reference electrode and AFz as the ground electrode, and synchronized with the VR platform and EMG sensors via MacroRecorder and TeamViewer.

Preprocessing was performed using *MNE-Python* [43] following a multistep procedure [44]. Data were first band-pass filtered (1–100 Hz) and down-sampled to 1024 Hz.

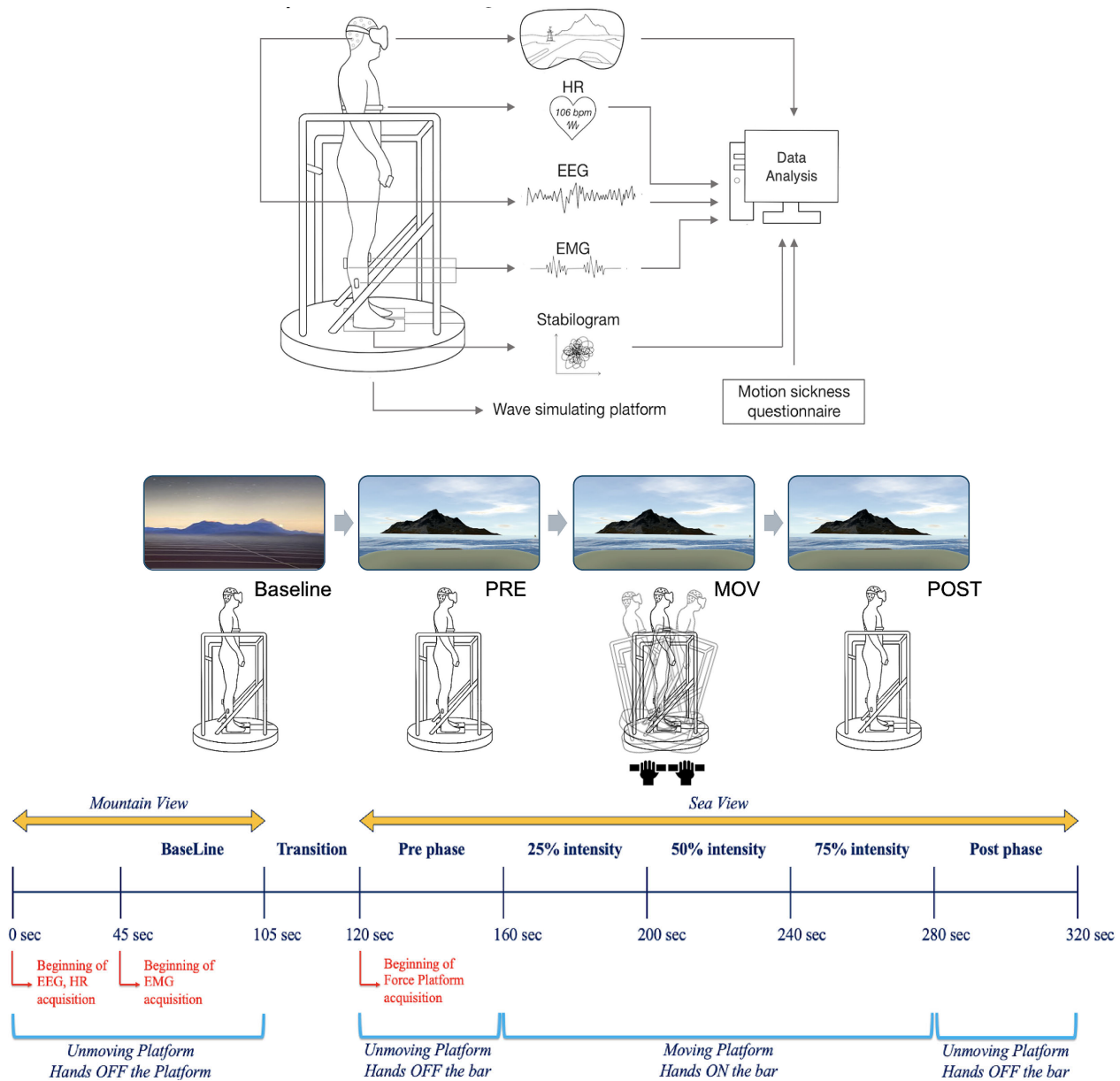


Fig. 1. BioVRSea Paradigm and experimental protocol. The BioVRSea system integrates multimodal recordings during simulated postural perturbations in a VR environment. Participants stood on a wave-simulating platform while EEG is recorded. The experiment consists of six consecutive phases designed to manipulate visual and visuo-motor input during postural control: Baseline (45–105 s; participants stood still with their arms by their sides on a static platform while viewing a non-moving mountain landscape in VR), PRE (Phase 1; 120–160 s; the scene transitioned to a dynamic sea view, introducing visual wave motion without physical platform movement to elicit visually driven postural responses), MOV (Phase 2–4; consisting of three sub-phases with increasing motion intensity as 25% (160–200 s), 50% (200–240 s), and 75% (240–280 s); during these phases, participants held onto support bars while the platform oscillated in synchrony with the VR waves, creating a progressively challenging visuo-motor condition), POST (Phase 5; 280–320 s; the platform returned to a static state while the VR sea scene continued, requiring participants to maintain balance without using the bars and relying primarily on visual input). The timeline below shows the sequence of experimental phases and recording onsets.

Noisy-channel detection was performed using the *pyprep* algorithm [45], [46], followed by channel interpolation. The data were then re-referenced to the common average reference. Independent Component Analysis (ICA) with *ICLabel* classification was applied to identify and remove motion-related, ocular, and cardiac artifacts [47]. In addition to automated artifact correction, all recordings were visually inspected to identify and exclude segments with excessive noise. A second band-pass filter (1–45 Hz) was applied to remove high-frequency noise and attenuate the 50 Hz mains

component [48]. Continuous-signal analysis was employed to preserve temporal integrity across experimental phases.

Of 449 initial EEG recordings, 433 were valid; 242 were retained for final analysis based on cohort inclusion and data completeness (49 HC-Young, 51 HC-Old, 35 HC matched to PD, 22 PD, 49 HC matched to mTBI, and 36 mTBI).

7) EEG Analysis: Time–frequency analysis was performed using the Morlet wavelet transform implemented in MNE-Python (*tfr_array_morlet*). Frequencies from 1–30 Hz were analyzed in 0.05 Hz steps at an effective sampling rate

TABLE I
DESCRIPTIVE INFORMATION OF GROUPS

Group	Male	Female	Mean Age	SD
HC-Young (N=49)	26	23	24.0	2.74
HC-Old (N=51)	30	21	58.0	6.49
HC (N=35)	19	16	57.3	8.59
PD (N=22)	14	8	63.5	7.89
HC (N=49)	0	49	26.0	5.26
mTBI (N=36)	0	36	27.9	5.94

of 125 Hz. The number of cycles increased as a function of frequency ($\text{cycles} = 5 \times (\text{freq} / 2)$), providing adaptive time–frequency resolution across the spectrum. Power estimates were computed over the 0–200 s experimental window and expressed in decibels (dB). This frequency-dependent scaling follows standard recommendations for balancing temporal and spectral precision in EEG time–frequency analysis [48].

8) Statistical Analysis: Behavioral data were analyzed using paired and independent t-tests following Shapiro–Wilk normality checks. A significance level of $\alpha = 0.05$ was adopted. Behavioral between- and within-group comparisons were restricted to a priori, hypothesis-driven contrasts and corrected using Bonferroni adjustment to control the family-wise error rate. Effect sizes were reported as Cohen’s d [49].

EEG data were analyzed using cluster-based permutation analysis (CBPA) to control for multiple comparisons in the time–frequency domain [39], [49]. Clusters of contiguous time–frequency points were identified and tested using 1,000 random permutations, with significance determined at a cluster-level threshold of $p < 0.05$. This non-parametric framework provides control over multiple comparisons in high-dimensional datasets and is well suited for repeated-measures EEG designs.

III. RESULT

A. Participants

The final sample included 49 young HC (mean $\pm$ SD: 24 ± 2.7 years), 51 older HC (58 ± 6.4 years), 36 mTBI

participants (27.9 ± 5.9 years), and 22 early-stage PD participants (63.5 ± 7.8 years). Age- and sex-matched HC subgroups were used for comparisons with the mTBI ($N = 49$; all female; mean $\pm$ SD = 26 ± 5.2 years) and PD ($N = 35$; 16 females; mean $\pm$ SD = 57.3 ± 8.5 years).

Demographic and matching information is summarized in Table I.

B. Behavioral Results

1) Motion Sickness Susceptibility Questionnaire (MSSQ): MSSQ comparisons between groups are summarized in Table II. After applying the Bonferroni correction for 27 comparisons (corrected $\alpha = 0.00185$), no significant differences were found between the HC-Young and HC-Old subgroups for any transportation mode. Similarly, the HC group matched with PD participants and the PD group itself did not differ significantly across any transportation modes. In the comparison between the HC group matched with the mTBI cohort and the mTBI group, no transportation mode reached the corrected significance threshold.

2) Motion Sickness Symptoms (MSS) Scale: Table III summarizes the MSS results, showing mean symptom differences (post–pre) for both between- and within-subject analyses after Bonferroni correction. For between-subject comparisons (corrected $\alpha = 0.00167$), HC-Old participants reported greater symptom severity than HC-Young for fatigue ($p < 0.001$, $d = 0.71$), increased salivation ($p < 0.001$, $d = 0.80$), and nausea ($p < 0.001$, $d = 0.86$). No HC vs PD differences survived correction. In the HC vs mTBI comparison, the mTBI group showed higher severity for sweating ($p = 0.001$, $d = -0.73$), blurred vision ($p = 0.001$, $d = -0.48$), and dizziness/vertigo ($p = 0.001$, $d = 0.67$).

For within-subject pre–post comparisons (corrected $\alpha = 0.00833$ per item), HC-Young participants exhibited significant increases in general discomfort ($p < 0.001$, $d = -0.59$), fatigue ($p < 0.001$, $d = 0.90$), and nausea ($p = 0.001$, $d = 0.27$) following exposure. HC-Old participants showed significant increases in general discomfort ($p = 0.001$, $d = -0.47$), sweating ($p < 0.001$, $d = -0.40$), and nausea ($p < 0.001$, $d = -0.49$). The HC group matched with PD demonstrated an increase in general discomfort ($p < 0.001$, $d = -0.61$).

In the mTBI group, significant pre–post increases were observed across multiple symptoms, including general discomfort ($p < 0.001$, $d = -0.67$), sweating ($p < 0.001$, $d = -0.82$), nausea ($p < 0.001$, $d = -0.62$), blurred vision ($p < 0.001$, $d = -0.52$), and dizziness/vertigo ($p < 0.001$, $d = -0.81$), indicating a broader and more severe post-exposure symptom profile compared with other groups. These findings are consistent with prior studies describing increased postural and sensory symptom severity in visually induced motion sickness [10], [23], [24], [40], [41].

C. EEG Results

Time–frequency representations (TFRs) of brain activity are shown in Figs. 2–4, illustrating spectral power changes across five stimulus-intensity phases during the postural

TABLE II
MSSQ T-TEST STATISTICS

Groups	Car	Bus	Train	Aircraft	Funfair	Small boat	Ship	Swing	Roundabouts
HC-Young M (SD)	0.755 (0.947)	0.592 (0.864)	0.114 (0.443)	0.306 (0.619)	0.556 (0.813)	0.957 (0.999)	0.804 (0.859)	0.130 (0.499)	0.533 (0.869)
HC-Old M (SD)	0.725 (0.940)	0.725 (0.896)	0.213 (0.508)	0.431 (0.781)	0.850 (1.190)	1.130 (1.170)	1.310 (1.040)	0.370 (0.645)	0.786 (1.060)
<i>p-value</i>	<i>0.876</i>	<i>0.450</i>	<i>0.325</i>	<i>0.378</i>	<i>0.182</i>	<i>0.451</i>	<i>0.013</i>	<i>0.050</i>	<i>0.239</i>
<i>Cohen's d</i>	<i>-0.031</i>	<i>0.152</i>	<i>0.208</i>	<i>0.177</i>	<i>0.292</i>	<i>0.156</i>	<i>0.523</i>	<i>0.415</i>	<i>0.256</i>
HC M (SD)	0.667 (0.956)	0.750 (0.967)	0.265 (0.666)	0.444 (0.809)	0.800 (1.240)	1.030 (1.150)	1.340 (1.060)	0.394 (0.747)	0.667 (1.030)
PD M (SD)	0.545 (0.671)	0.667 (0.966)	0.050 (0.224)	0.364 (0.581)	0.500 (0.707)	0.619 (0.669)	0.905 (0.831)	0.286 (0.561)	0.762 (0.944)
<i>p-value</i>	<i>0.605</i>	<i>0.755</i>	<i>0.170</i>	<i>0.685</i>	<i>0.355</i>	<i>0.144</i>	<i>0.111</i>	<i>0.572</i>	<i>0.738</i>
<i>Cohen's d</i>	<i>0.141</i>	<i>0.086</i>	<i>0.392</i>	<i>0.110</i>	<i>0.279</i>	<i>0.417</i>	<i>0.448</i>	<i>0.159</i>	<i>-0.096</i>
HC M (SD)	1.200 (0.960)	0.843 (0.925)	0.234 (0.476)	0.240 (0.517)	0.630 (0.951)	1.230 (1.090)	1.040 (1.060)	0.279 (0.701)	0.783 (1.130)
mTBI M (SD)	1.260 (1.070)	1.140 (1.030)	0.500 (0.663)	0.343 (0.591)	0.968 (0.983)	1.210 (1.090)	1.380 (1.070)	0.343 (0.838)	1.000 (1.120)
<i>p-value</i>	<i>0.782</i>	<i>0.163</i>	<i>0.039</i>	<i>0.398</i>	<i>0.136</i>	<i>0.909</i>	<i>0.177</i>	<i>0.715</i>	<i>0.400</i>
<i>Cohen's d</i>	<i>-0.061</i>	<i>-0.309</i>	<i>-0.474</i>	<i>-0.187</i>	<i>-0.350</i>	<i>-0.026</i>	<i>-0.312</i>	<i>-0.083</i>	<i>-0.193</i>

Note: Bonferroni correction applied for 27 comparisons; significance threshold set at $p < 0.00185$.

control paradigm. Power variations (in dB) are displayed as a function of time (seconds) and frequency (Hz). The color scale represents power intensity, with warmer colors (reds, oranges) indicating increased oscillatory power and cooler colors (blues) reflecting decreases. Black contours mark statistically significant clusters in neural activity, determined through cluster-based permutation analysis (CBPA) [39].

Time–frequency representations shown in Figs. 2–4 correspond to whole-scalp averaged spectral power (averaged across all EEG channels) for each participant and then averaged at the group level.

Figs. 2–4 depict the five stimulus phases analyzed: Phase 1 (PRE), Phases 2–4 (MOV at 25 %, 50 %, and 75 % intensity), and Phase 5 (POST). Together, these phases represent the temporal evolution from pre-perturbation to recovery.

1) Within-Group Comparisons: TFRs across Phases 1–5 were analyzed separately for each group using CBPA (1000 permutations; $p < 0.05$). No statistically significant phase-to-phase clusters were identified in any group (all $p > 0.05$). Healthy young and older adults displayed stable spectral power across phases, with no evidence of intra-group modulation. Similarly, clinical groups—including mTBI and PD—showed comparable spectral profiles, and no clusters reached statistical significance. The matched HC groups demonstrated consistent

time–frequency patterns, mirroring the absence of significant within-group effects.

2) Between-Group Comparisons:

a) *HC-Young vs HC-Old:* The TFR for this comparison is shown in Fig. 2. The HC-Young group exhibits a relatively stable pattern of spectral power, primarily concentrated in the low-frequency range (1–5 Hz). In contrast, the HC-Old group demonstrates a more pronounced low-frequency power increase, with more persistent oscillatory activity across multiple frequencies. Spectral power differences between the HC-Young and HC-Old groups were assessed across Phases 1–5 using a CBPA (1000 permutations; cluster-forming threshold $p < 0.05$). No significant clusters were detected for Phases 1–4 ($p > 0.05$). A single cluster at the end of Phase 5 ($t > 30$ s) showed a significant between-group difference ($p < 0.05$). No additional clusters survived correction for multiple comparisons.

The significant cluster observed at 30–40 s corresponds to the late POST recovery phase, during which participants re-stabilize following termination of platform motion while visual stimulation persists. This interval is consistent with the time course of sensory reweighting processes described in human postural control [50], and likely reflects ongoing cortical engagement required for balance recalibration after mechanical perturbation. Prior EEG studies

TABLE III
MSS T-TEST STATISTICS

Items	HC-Young MeanDiff ± SD	HC-Old MeanDiff ± SD	p-value	Cohen's d	HC MeanDiff ± SD	PD MeanDiff ± SD	p-value	Cohen's d	HC MeanDiff ± SD	mTBI MeanDiff ± SD	p-value	Cohen's d
General discomfort	-0.44 ± 0.61	0.37 ± 0.40	0.053	0.391	-0.59 ± 0.79	0.10 ± 0.11	0.010	-0.724	-0.35 ± 0.21	-0.54 ± 0.32	0.371	0.197
<i>post-pre (p, d)</i>	(<.001*, -0.587)	(0.001*, -0.471)			(<.001*, -0.607)	(0.633, 0.101)			(0.005, -0.407)	(<.001*, -0.674)		
Fatigue	0.48 ± 0.41	0.06 ± 0.05	<.001*	-0.708	0.00 ± 0.14	0.05 ± 0.13	0.927	-0.025	0.29 ± 0.19	0.23 ± 0.12	0.912	-0.025
<i>post-pre (p, d)</i>	(<.001*, 0.897)	(0.464, 0.102)			(0.991, 0.002)	(0.760, 0.064)			(0.004, 0.419)	(0.094, 0.287)		
Headache	-0.12 ± 0.04	-0.10 ± 0.13	0.324	-0.199	-0.14 ± 0.17	0.18 ± 0.49	0.024	-0.628	-0.07 ± 0.21	-0.29 ± 0.32	0.076	0.394
<i>post-pre (p, d)</i>	(0.799, -0.037)	(0.021, -0.330)			(0.030, -0.372)	(0.089, 0.371)			(0.280, -0.153)	(0.013, -0.434)		
Eye strain	0.08 ± 0.21	-0.16 ± 0.28	0.076	-0.359	-0.22 ± 0.35	0.13 ± 0.10	0.114	-0.435	0.00 ± 0.12	-0.40 ± 0.22	0.820	0.051
<i>post-pre (p, d)</i>	(1.000, 0.000)	(0.028, -0.314)			(0.021, -0.396)	(0.356, 0.197)			(0.159, -0.200)	(0.003, -0.524)		
Difficulty in concentrating	0.19 ± 0.41	-0.18 ± 0.19	0.024	-0.456	-0.20 ± 0.19	0.13 ± 0.14	0.027	-0.614	0.04 ± 0.04	-0.23 ± 0.09	0.234	0.263
<i>post-pre (p, d)</i>	(0.146, 0.211)	(0.016, -0.344)			(0.076, -0.300)	(0.357, 0.090)			(0.719, 0.051)	(0.026, -0.388)		
Increased salivation	0.12 ± 0.45	-0.23 ± 0.55	<.001*	-0.796	-0.19 ± 0.46	0.00 ± -0.13	0.151	-0.394	-0.12 ± 0.20	-0.14 ± 0.23	0.022	0.511
<i>post-pre (p, d)</i>	(0.011, 0.379)	(0.005, -0.431)			(0.021, -0.410)	(1.000, 0.000)			(0.057, -0.273)	(0.002, -0.546)		
Sweating	0.10 ± 0.38	-0.22 ± 0.30	0.002	-0.631	-0.25 ± 0.43	-0.18 ± -0.21	0.004	-0.809	-0.19 ± 0.22	-0.74 ± 0.42	0.001*	0.733
<i>post-pre (p, d)</i>	(0.088, 0.249)	(<.001*, -0.403)			(0.032, -0.368)	(0.213, 0.267)			(0.032, -0.310)	(<.001*, -0.821)		
Nausea	0.21 ± 0.36	-0.38 ± 0.61	<.001*	-0.856	-0.25 ± 0.70	-0.14 ± 0.44	0.306	-0.280	-0.36 ± 0.43	-0.66 ± 0.58	0.142	0.325
<i>post-pre (p, d)</i>	(0.067, 0.267)	(<.001*, -0.486)			(0.005, -0.486)	(0.356, -0.197)			(0.004, -0.418)	(<.001*, -0.616)		
Blurred vision	0.14 ± 0.03	-0.18 ± 0.35	0.002	-0.088	-0.24 ± 0.49	0.05 ± 0.15	0.004	-0.342	-0.21 ± 0.41	-0.34 ± 0.52	0.001*	0.079
<i>post-pre (p, d)</i>	(0.164, -0.202)	(0.016, -0.344)			(0.011, -0.441)	(0.736, -0.071)			(<.001*, -0.519)	(0.004, -0.509)		
Dizziness/vertigo	0.02 ± 0.15	-0.43 ± 0.75	0.002	-0.563	-0.52 ± 0.89	0.00 ± 0.01	0.004	-0.633	-0.59 ± 0.53	-0.60 ± 0.52	0.001*	-0.670

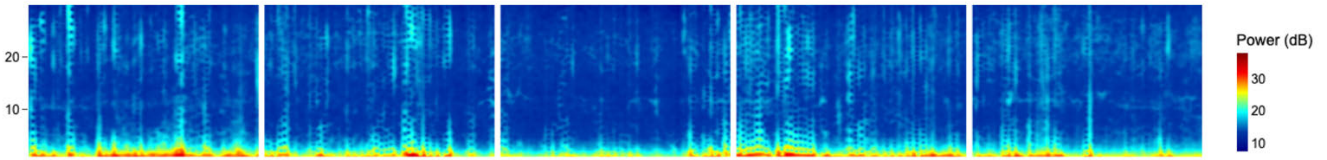
TABLE III
(Continued.) MSS T-TEST STATISTICS

<i>post-pre</i> (<i>p</i> , <i>d</i>)	(0.830, 0.031)	(<.001*, -0.577)	(<.001*, -0.613)	(1.000, 0.000)	(<.001*, - 0.810)	(<.001*, 0.666)
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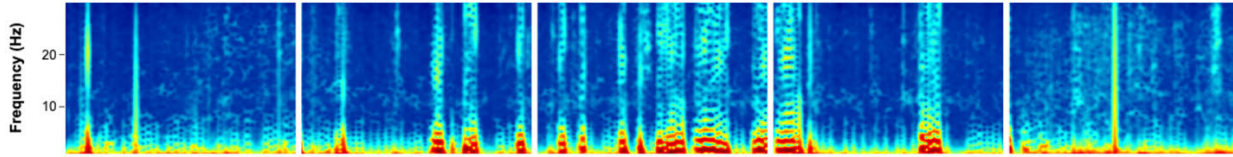
Note:

1. MeanDiff indicates 'post-pre'.
2. 'post-pre (p-value, d)' rows represent the within-group pre-post comparison.
3. Asterisks (*) denote statistical significance after Bonferroni correction. For between-subject analyses (30 comparisons), the corrected threshold was $p < 0.00167$; for within-subject analyses (6 comparisons per item), the threshold was $p < 0.00833$.

a. HC - Young



b. HC - Old



c. Young-Old

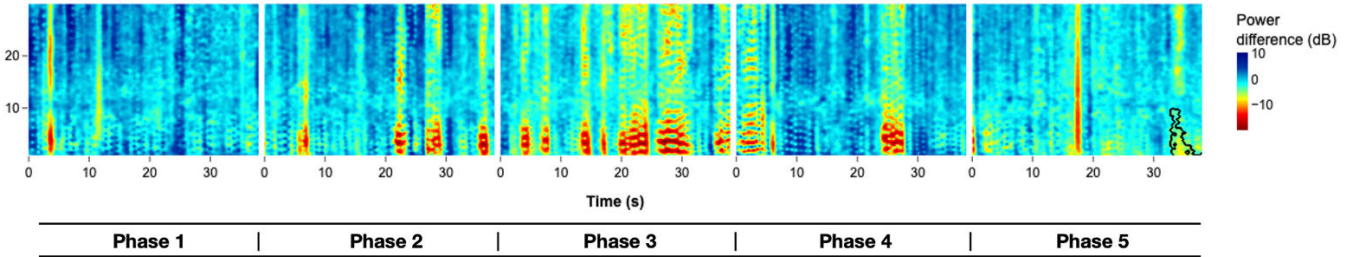


Fig. 2. Healthy Young vs Healthy Old ($p > 0.05$). (a) Average TFR of the young HC-Young subgroup of 49 participants (23 females, 26 males) aged 18–30; (b) average TFR of the older HC-Old subgroup of 51 participants (23 females, 28 males) aged 55–72 from the total healthy control group; (c) difference in TFR between HC-Young and HC-Old groups. Black contours represent statistically significant clusters identified through cluster-based permutation analysis ($p < 0.05$).

of postural recovery report enhanced late-phase cortical activity during reactive balance responses, particularly in older adults and under unpredictable perturbation conditions [51], [52], supporting the interpretation that this time window reflects compensatory sensorimotor processing during stabilization.

b) HC vs PD: The TFR for this comparison is shown in Fig. 3. The HC group exhibited relatively stable spectral power across phases. In contrast, the PD group demonstrated increased spectral power in both the delta–theta (≈ 1 –7 Hz) and alpha–beta (≈ 10 –30 Hz) frequency ranges. Cluster-based permutation analysis revealed significant group differences (cluster-level $p < 0.05$) across all five phases, with clusters predominantly localized within these low- and mid-frequency bands.

c) HC vs mTBI: The TFR for this comparison is shown in Fig. 4. Both groups exhibit dominant low-frequency power

(1–5 Hz) throughout all phases, with no visually striking differences in the spectral power distribution. Analysis revealed no statistically significant spectral differences between the HC and mTBI groups across the five phases. Both groups exhibited consistent low-frequency power (< 5 Hz) throughout the task, with no clusters reaching significance across time or frequency, with no localized deviations exceeding the cluster-corrected threshold ($p > 0.05$).

IV. DISCUSSION

This study investigated cortical oscillatory dynamics during postural control tasks using the BioVRSea paradigm in three populations: healthy adults, early-stage PD patients, and individuals with a history of mTBI. By integrating VR-based visual perturbations with platform-induced motion, and recording EEG in real time, we captured neural responses under ecologically valid balance challenges. Time–frequency

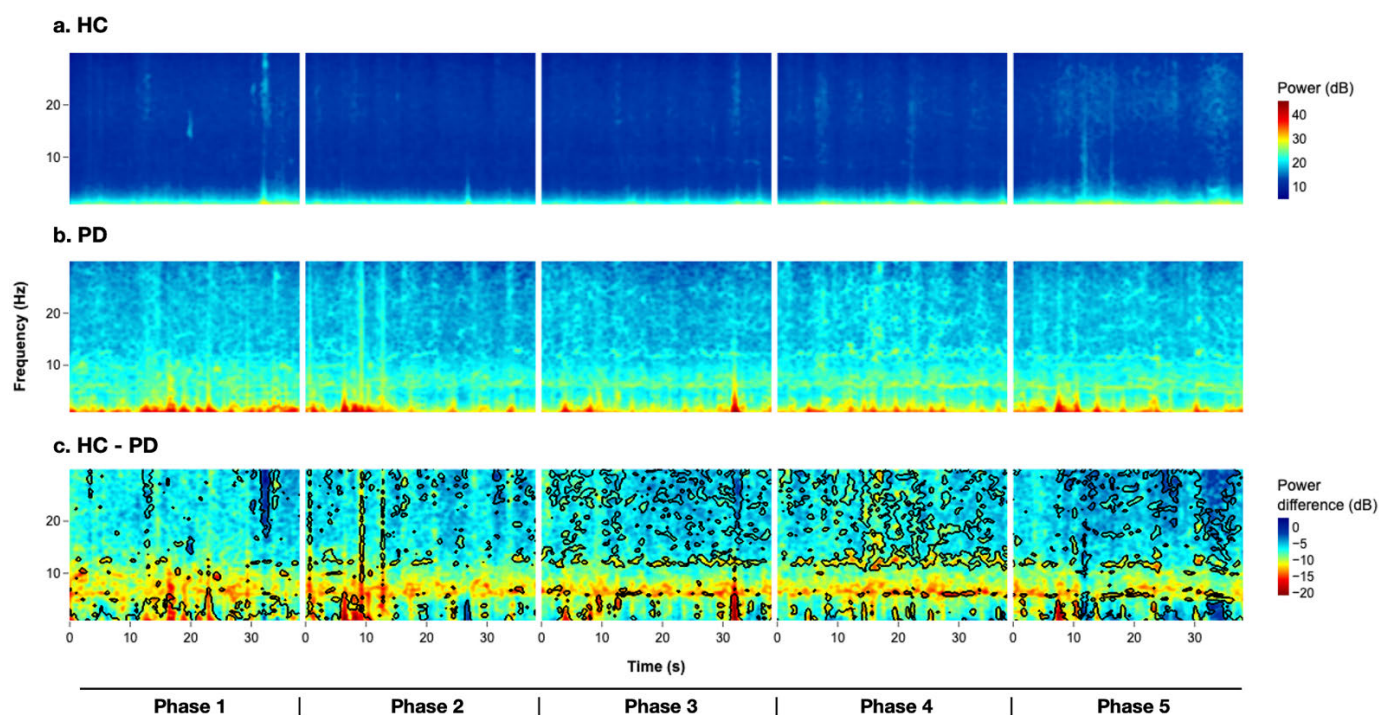


Fig. 3. Healthy vs Parkinson (PD) ($p < 0.05$). (a) Average TFR of 35 healthy participants (16 females, 19 males; aged 55–73); (b) average TFR of 22 PD patients (8 females, 14 males; aged 55–83); (c) difference in TFR between the HC and PD groups. Black contours represent statistically significant clusters identified through cluster-based permutation testing ($p < 0.05$).

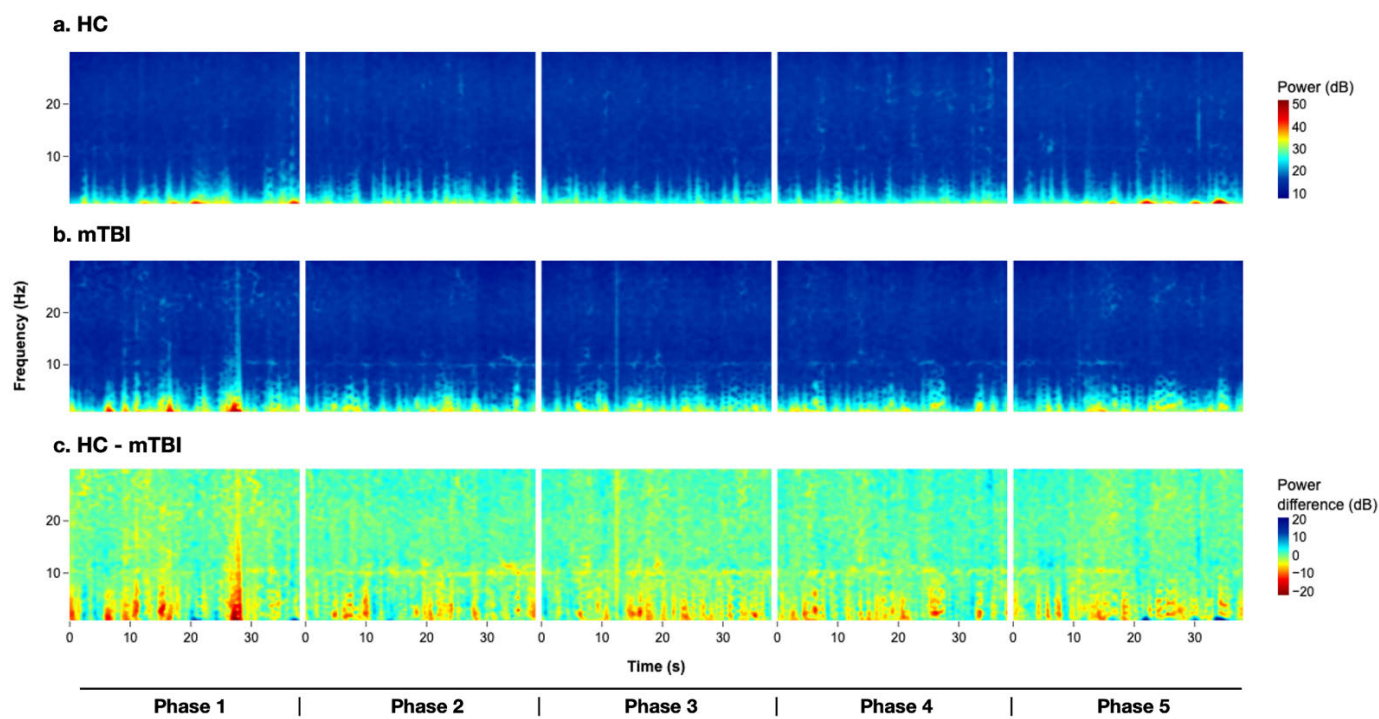


Fig. 4. Healthy vs mTBI ($p > 0.05$). (a) Average TFR of 49 healthy participants (49 females, 0 males; aged 18–40); (b) average TFR of 36 participants (36 females, 0 males; aged 19–40) with a history of mTBI; (c) difference in TFR between the HC and mTBI groups ($p > 0.05$).

analysis revealed condition- and group-specific spectral modulations.

Recent EEG studies using the BioVRSea paradigm have demonstrated that such perturbations elicit robust changes in spectral power and connectivity across multiple cortical

regions and frequency bands. Specifically, healthy participants show increased theta power during early movement phases, likely reflecting heightened cognitive load, attentional allocation, and motor planning [42]. Concurrently, alpha desynchronization over parietal and occipital areas has been

linked to sensory reweighting and increased visual processing demands [16], [43]. During high-intensity platform motion, beta power tends to increase over central areas, consistent with sensorimotor engagement and balance control [36], [43].

In some cases, gamma-band activity has also been observed, potentially indexing complex integrative processes at peak perturbation phases. Notably, these oscillatory dynamics vary by age: older adults exhibit attenuated alpha modulation and reduced theta flexibility, suggesting diminished neural adaptability to balance demands [2], [53]. In clinical contexts, the BioVRSea protocol has revealed impaired modulation of these rhythms. For example, individuals with mTBI demonstrate reduced posterior alpha and altered frontoparietal theta during visual-only and movement phases, indicating difficulties in sensory filtering and attentional control [11]. Likewise, early-stage PD patients show attenuated beta power and less distinct phase-dependent changes, pointing to deficits in sensorimotor integration and preparatory motor activity [36].

Although resting-state EEG differences among healthy individuals, patients with PD, and individuals with mTBI are well documented, such recordings primarily reflect baseline neural traits and provide limited insight into the functional integrity of postural and multisensory control mechanisms. In contrast, clinically relevant impairments in these populations often become evident under conditions of sensory conflict and balance perturbation rather than during quiet stance. Task-based paradigms that integrate visual and mechanical perturbations therefore enable direct evaluation of sensorimotor integration, adaptive postural control, and cortical engagement under ecologically valid instability. Within this framework, the BioVRSea paradigm facilitates characterization of challenge-dependent neural dynamics associated with functional balance performance and motion-related symptoms, extending beyond static EEG markers [8], [13].

Compared with prior VR-only visual perturbation studies in mTBI [33] and visual-perturbation balance studies in PD [34], the BioVRSea paradigm integrates synchronized visual flow with graded mechanical perturbations and continuous EEG time–frequency analysis. This combined design enables controlled manipulation of multisensory conflict and progressive balance load, allowing assessment of cortical dynamics during active sensory reweighting and postural adaptation rather than isolated visual processing alone.

Recent VR-based perturbation studies in older adults report modulation of sensorimotor and beta-band cortical activity during balance-challenging and immersive conditions, along with altered sensory reweighting strategies during postural control [54], [46], [56]. Our findings are consistent with this literature in demonstrating perturbation-related cortical engagement; however, the present study extends prior work by quantifying whole-scalp time–frequency EEG dynamics during graded and synchronized visual–mechanical perturbations rather than focusing primarily on training effects, corticomuscular coupling, or behavioral outcomes. In contrast to VR feasibility or symptom-based investigations in mTBI and PD [57], [58], [59], this work provides electrophysiological characterization of multisensory postural processing under progressively increasing perturbation load. Furthermore, recent

EEG studies of cybersickness and visually induced motion sickness have shown consistent frequency-domain alterations during immersive VR exposure, supporting the validity of time–frequency markers in such contexts. The observed age-related differences during late stabilization in the present study are also consistent with reports linking altered beta-band modulation to postural instability in older adults [60], reinforcing the relevance of multimodal VR perturbation paradigms for probing functional balance-related neural dynamics.

Building on this framework, we employed combined visual and vestibular stimulation to more accurately capture EEG power changes in VR environments, compared to single-modality approaches. Our findings support the broader literature on the reorganization of postural control systems in aging and neurological populations [61], [63], [64].

Subjects with early-stage PD exhibited greater increases in autonomic and cognitive symptoms—such as headache, difficulty concentrating, sweating, and dizziness—compared to HC, indicating heightened susceptibility to motion-related discomfort. This may be explained by symptoms commonly arising from impaired autonomic regulation and disrupted multisensory integration in PD, which are likely exacerbated by motion-related stimulation in VR environments [7]. Notably, although overall susceptibility scores did not differ significantly between groups, PD participants demonstrated consistent symptom increases across experimental phases (Phase 1 to Phase 5), revealing a sensitivity not fully captured by group-level comparisons.

The mTBI group also reported significantly greater increases in symptoms—such as sweating, dizziness, increased salivation, and blurred vision—compared to HC, aligning with evidence that mTBI disrupts central autonomic regulation and vestibular processing, leading to exaggerated responses to sensory conflict [65], [66], [67]. Reports of increased cognitive and autonomic symptoms in the mTBI group support the utility of VR-based paradigms in probing post-injury dysfunctions that may evade conventional clinical assessments [68], [69], [70].

Older participants showed significantly greater increases in symptoms such as nausea and salivation relative to younger participants, consistent with evidence that aging impairs vestibular compensation and sensory adaptation [2], [53]. This finding aligns with a growing body of evidence indicating that aging is associated with alterations in the multisensory and neuromuscular systems underlying postural stability. It has been shown that physiological complexity within postural sway diminishes with age, reflecting reduced adaptability and impaired integration of sensory feedback [70]. Such declines in the dynamic coordination of sensory and motor inputs are thought to constrain the system's ability to respond flexibly to perturbations, contributing to both instability and motion-related discomfort. It has also been reported that older adults increasingly rely on cortical control to maintain balance, representing a compensatory shift from automatic subcortical pathways toward cognitively demanding cortical mechanisms [1], [2]. While this cortical recruitment aids balance maintenance, it reduces efficiency and adaptability, particularly under dual-task or unpredictable conditions. Evidence further

indicates that age-related degradation in visual, proprioceptive, and vestibular processing slows corrective responses and amplifies sensory conflict, resulting in greater instability and higher fall risk [71], [72], [73]. Together, these findings suggest that the amplified physiological and perceptual responses observed in our older cohort during the BioVRSea paradigm—particularly the heightened nausea and salivation during the post-perturbation phase—reflect not only peripheral vestibular decline but also a broader cortical and systemic reorganization that diminishes sensory reweighting flexibility. In this context, the BioVRSea paradigm appears to reveal reduced dynamic reserve of the aging postural control system, revealing how limited adaptability and excessive cortical engagement may jointly underlie increased motion susceptibility in older adults.

A. Key Findings

Age-related differences in neural oscillations emerged during postural control, with older adults exhibiting significantly increased low-frequency (delta–theta, ≈ 1 –7 Hz) EEG power at the end of the task (Phase 5, $t = 40$ s, $p < 0.05$), while younger adults maintained a stable oscillatory profile across all phases. These spectral shifts were accompanied by greater self-reported post-exposure symptoms in the older group—including fatigue, nausea, salivation, and dizziness—suggesting that aging is associated with both altered cortical dynamics and increased physiological susceptibility during sensorimotor tasks.

Participants with early-stage PD showed significant increases in spectral power across both delta–theta (≈ 1 –7 Hz) and alpha–beta (≈ 10 –30 Hz) frequency bands across stimulus phases compared to matched controls, with statistically significant clusters ($p < 0.05$) consistent with altered cortical excitability or impaired neural gating. These neurophysiological alterations co-occurred with elevated motion sickness symptoms (e.g., headache, difficulty concentrating, and sweating), supporting the interpretation that PD may involve both central oscillatory dysregulation and compromised multisensory integration.

Despite the absence of statistically significant EEG differences between mTBI participants and their matched controls, the mTBI group reported significantly greater post-exposure symptoms (e.g., salivation, sweating, dizziness), along with higher susceptibility to motion-related discomfort in train travel. This dissociation between subjective symptomatology and group-level EEG findings suggests that subtle or diffuse functional disruptions following mTBI may not be captured by traditional time–frequency power analyses alone, highlighting the need for more sensitive or multimodal assessment strategies.

EEG-based within-group analyses (CBPA, $p > 0.05$) revealed no significant spectral modulation across stimulus phases for any group, indicating that neither healthy nor clinical cohorts exhibited strong phase-dependent dynamic shifts in power during the postural control task. This temporal stability in oscillatory responses suggests that observed between-group differences are likely trait-related neural

signatures rather than transient state-based adaptations during sensorimotor challenge.

Across all groups, oscillatory dynamics and behavioral symptom profiles demonstrated condition-specific interactions: aging amplified both cortical low-frequency engagement and physical symptoms, PD was marked by diffuse oscillatory increases and multisensory distress, while mTBI presented with behavioral hypersensitivity in the absence of overt electrophysiological anomalies. These findings support prior evidence suggesting that neurological and age-associated sensory deficits may exacerbate motion sensitivity in VR environments [22], [68], [74], and support the utility of the BioVRSea paradigm as a tool for characterizing population-specific neural and perceptual responses during ecologically valid postural perturbations.

B. Study Limitations and Future Directions

This study has several limitations. First, the mTBI group was identified based on self-reported history, which may introduce recall bias. Although the SCAT5 was administered to aid classification, the absence of formal neuropsychological assessment constitutes a methodological limitation. Second, the relatively small sample sizes in both the PD and mTBI groups limit the generalizability of the findings. Third, the absence of comprehensive cognitive assessment limits the ability to directly relate oscillatory findings to specific cognitive domains. Furthermore, the single-session design precludes insights into longitudinal changes, underscoring the need for follow-up assessments to evaluate progression or recovery over time. Finally, future research could benefit from extending the BioVRSea paradigm to additional neurological populations, such as individuals with vestibular dysfunction.

Many studies conventionally define “older” or “elderly” adults as individuals aged ≥ 65 years; therefore, the older cohort in the present study—representing mid-to-late adulthood—should not be interpreted as an advanced-aging population, and the findings may not generalize to individuals over 65 years of age [75], [76]. However, balance-related and cortical oscillatory changes have been reported in adults beginning around 60 years of age, suggesting that the observed effects likely reflect early age-associated alterations in postural control [77]. Future studies including participants ≥ 65 years will be necessary to determine whether these neural dynamics are amplified or qualitatively altered in later-life aging.

In addition to chronological age classification, musculoskeletal comorbidities (e.g., knee osteoarthritis, chronic low back pain, or osteoporosis-related postural changes) were not systematically screened in the present study. Such conditions are known to influence balance performance and postural sway in older adults and clinical populations [78], [79], [80], [81], and may therefore have contributed to variability in stabilization-related behavioral responses. However, because the primary outcomes focused on cortical oscillatory dynamics rather than purely biomechanical measures, it is unlikely that peripheral musculoskeletal factors alone account for the observed group-specific neural patterns. Future work incorporating structured musculoskeletal screening and covariates

(e.g., pain scores, mobility indices) will help further dissociate peripheral biomechanical influences from central sensorimotor mechanisms [82], [83].

Although linear mixed-effects models could provide a complementary framework to explicitly model subject-level variability and phase-by-group interactions within a unified statistical structure, the present study employed pairwise and cluster-based permutation approaches to test condition- and group-level effects. While cluster-based correction mitigates the risk of inflated Type I error across time–frequency comparisons, mixed-effects modeling may offer improved sensitivity to hierarchical and longitudinal effects across phases. Future studies incorporating mixed-model frameworks would therefore further strengthen inference regarding within- and between-group dynamics.

V. CONCLUSION

This study offers novel insights into the neural dynamics of postural control across healthy individuals, patients with PD, and individuals with a history of mTBI. Using the BioVRSea framework, we demonstrate the feasibility of synchronized VR–EEG for investigating electrophysiological mechanisms underlying postural control impairments.

Although resting-state EEG differences in PD, mTBI, and aging are well documented, postural perturbation paradigms specifically probe reactive and adaptive balance control under conditions of multisensory conflict. The present findings demonstrate that synchronized visual–mechanical perturbations reveal challenge-dependent cortical dynamics—particularly during late-phase stabilization—that are not fully captured by baseline spectral traits alone [50], [84], [85], [86]. By eliciting condition-specific oscillatory patterns alongside symptom responses across cohorts, the BioVRSea framework highlights the added value of task-based EEG assessment for characterizing functional postural control mechanisms. These findings motivate the integration of ecologically valid perturbation paradigms in future neurorehabilitation and clinical research aimed at understanding balance-related neural dysfunction.

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