

Temporal dynamics of EEG microstates during postural control in Parkinson's disease

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ABSTRACT

Parkinson's disease (PD) is a progressive neurodegenerative disorder characterised by motor impairments extending to balance and postural regulation. Although EEG abnormalities in oscillatory activity and functional connectivity are well documented in PD, large-scale brain dynamics during tasks directly engaging postural control remain poorly understood. To address this gap, we examined EEG microstate organisation during the BioVRSea virtual-reality postural-control task in early-stage PD patients ($n = 30$) and matched healthy controls (HC; $n = 26$). EEG microstates, brief quasi-stable scalp topographies representing global neural states, provide a robust framework for characterising the rapid temporal structure of whole-brain activity. Although task-based microstate approaches exist, applications to PD remain limited and largely confined to resting-state research. Topographical analyses revealed pronounced between-group differences in microstates D and E, whose group-averaged maps in PD diverged markedly from canonical configurations. Because these maps were not topographically equivalent between groups, comparisons of temporal parameters were restricted to the comparable microstates A–C. Across all task phases, PD patients showed increased duration and coverage of microstates A and B. Transition-probability analysis, likewise restricted to A–C, indicated different trajectories across phases in PD and HC. A single significant Group $\times$ Phase interaction emerged for A→C: the largest between-group difference occurred during the POST-movement phase, when PD showed a higher probability than HC. For the remaining transitions, no phase-dependent differences emerged within PD. Because patients were assessed ON medication and clinical or behavioural correlates were unavailable, these findings represent candidate task-state EEG microstate alterations requiring validation, rather than established disease-specific markers.

1. Introduction

Parkinson's disease (PD) is a progressive neurodegenerative disorder marked by complex motor and non-motor dysfunctions [1]. Current diagnostic approaches rely heavily on clinical observation of motor

symptoms that emerge only after substantial neurodegeneration has occurred. There is a critical need for objective, accessible biomarkers that support earlier diagnosis, track disease progression, and assess therapeutic responses. Electroencephalography (EEG) has attracted increasing interest as a method for characterising neural dysfunctions

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associated with PD [2], including abnormalities in oscillatory activity [3], functional connectivity [4,5], and temporal network dynamics, such as EEG microstates [6]. Microstate analysis has emerged as a promising tool for studying global brain function, building on empirical evidence that scalp electric field distributions recorded in spontaneous EEG, as captured by topographic voltage maps, remain briefly stable before transitioning to another configuration (i.e., a state) [7,8]. These quasi-stable patterns reflect transient global neuronal activity states, with changes in scalp potential topography indicating shifts in the brain's large-scale network coordination over time. This approach has shown promise in identifying and characterising neurophysiological indicators associated with neural alterations underlying neuropsychiatric conditions, including PD [9]. To date, most EEG microstate studies in PD have been limited to resting-state recordings, potentially missing functionally relevant alterations that emerge during active cognitive-motor challenges (for a comprehensive review, see Li et al. [6]). The large body of EEG research in PD shows cortical slowing—elevated theta and reduced alpha, beta, and gamma power—both at rest and during motor tasks, indicating reduced neural efficiency and impaired sensorimotor integration [10]. Connectivity analyses further reveal abnormal coupling in motor and cognitive networks, consistent with disrupted cortico-basal ganglia-thalamic loop mechanisms [11]. EEG microstate analysis has recently proven effective for probing sub-second brain dynamics in PD, primarily reflecting specific disruptions in motor and cognitive networks. Studies reporting associations with motor and cognitive symptoms (e.g., MoCA, MDS-UPDRS) highlight the potential of microstates as electrophysiological markers [6]: compared to healthy controls (HC), PD patients exhibit changes in microstate topographies and altered temporal parameters—especially increased duration and coverage of certain microstates, alongside reductions in occurrence [6]. This indicates a brain state that is less responsive to environmental demands and might give rise to the apparent slowing in thinking [12]. Altered dynamics are also observed with sensory functional states and might reflect disability and cognitive fatigue [13]. Our recent findings from a large study of healthy participants during the BioVRSea, a novel paradigm to measure postural control responses using virtual reality and a moving platform, demonstrated the feasibility of applying microstate analysis in a complex, dynamic environment, revealing that a five-map solution has strong topographical correspondence with canonical microstate classes [14]. Our hypothesis, based on the known involvement of large-scale network dynamics in PD, is that patients may show microstate organisation that differs from that of matched controls during the BioVRSea task, including differences in microstate temporal parameters.

In this study, we apply EEG microstate analysis during a dynamic, multisensory experiment, the BioVRSea task [15], which provides an ecologically valid context for assessing group-related neural differences, using postural control as a functional framework. We seek to quantify whether EEG microstate temporal dynamics differ between subjects with PD and matched HC. A specific aim will be to look at the following temporal parameters [8]: (1) average duration that a given microstate remains stable, (2) frequency of occurrence for each microstate independent of its individual duration (3) coverage (i.e. the fraction of total recording time), for which a given microstate is dominant. This research introduces a novel application of EEG microstate analysis in a dynamic, multisensory task context. By moving beyond resting-state recordings, we offer a more comprehensive assessment of PD-related brain dynamics, potentially uncovering group-level differences that might otherwise go undetected. In this way, the study will provide foundational data supporting task-based EEG microstate analysis as a candidate approach for PD, with results that may guide future longitudinal efforts to track disease progression and evaluate therapeutic responses. By characterising differences in microstate parameters, we aim to identify candidate, objective indicators of group-level EEG differences in PD that warrant validation.

2. Materials and methods

2.1. Participants

Thirty early-stage PD patients (mean age, 67.96 ± 7.7 years) were included in the study. All participants had a diagnosis of PD established according to the Unified Parkinson's Disease Rating Scale (UPDRS). Only patients classified as stages 1-3 on the Hoehn and Yahr scale were included. Additional inclusion criteria included: stable antiparkinsonian medication before enrollment, the ability to comply with study procedures, and the absence of other neurological or psychiatric disorders that could interfere with the assessments. In addition, 30 age- and gender-matched HC participants were recruited as a control group; 26 (mean age, 64.15 ± 6.5) were included in the full analysis, and 4 were excluded due to data quality issues. A detailed description of the sample is provided in the Supplementary Materials (Supplementary Table 2). Informed consent was obtained in written form from all individual participants included in the study. The research was conducted in accordance with the principles outlined in the Declaration of Helsinki and in adherence to the Icelandic statutory requirements. The study protocol was approved by the Icelandic National Bioethics Committee (17-183-S1).

2.2. Acquisition protocol

Participants underwent a single session of the BioVRSea protocol, a multimodal postural control paradigm that combines immersive virtual reality (VR) with a motion platform capable of moving in synchrony with the VR environment, presented through VR goggles (see Fig. 1). This setup allows the induction of realistic sensorimotor perturbations while monitoring balance and adaptive responses. The task is performed on a moving platform that simulates sea waves, which are also displayed through an integrated virtual reality (VR) system. The task combines congruent and incongruent dynamic visual-motor stimulation to systematically challenge both motor and cognitive processing. The experiment lasts about 5 min and is divided into four phases: a Baseline period (BL) with a motionless platform and non-sea visual scene, lasting 120 s; a PRE phase (PRE) with dynamic sea-visual stimulation only, lasting 40 s; a Movement (MOV) phase, lasting 120 s, where the platform moves concurrently with sea visuals, with the wave frequency progressively goes from 0.5 Hz to 3 Hz, and the wave amplitude from 0 to 2 m, during which participants were asked to hold the safety bar; and a POST (POST) phase returning to visual stimulation only (stationary platform with dynamic sea visuals), lasting 40 s. Throughout each phase of the experiment, participants were instructed to maintain their balance as steadily as possible. During the experiment, EEG signals were continuously acquired using a 64-channel system (ANT Neuro, The Netherlands), with electrodes positioned according to the 10–10 system. The EEG amplifiers, connected to the cap, were secured in a lightweight backpack worn by the participants to allow free movement during platform oscillations. Data were recorded at a sampling rate of 4096 Hz, and all electrode impedances were maintained below 5 k Ω .

2.3. EEG preprocessing

The *EEGLAB MATLAB* Toolbox was used to preprocess the EEG data. EEG was downsampled to 512 Hz and then bandpass-filtered between 2 and 20 Hz (2 Hz high-pass and 20 Hz low-pass cutoffs). The choice of the filter's cutoff was based on previous literature on EEG microstates analysis [6,11]. Furthermore, this cutoff was chosen to remove potential high-frequency noise, given the task's dynamic nature. Channels mainly contaminated by muscular artefacts were systematically removed, resulting in a reduced dataset of 52 channels (see Supplementary Fig. 1 in the Supplementary Materials for a detailed description of the EEG montage used in this study). This step was applied to minimise persistent contamination from neck and trunk muscle activity expected in the

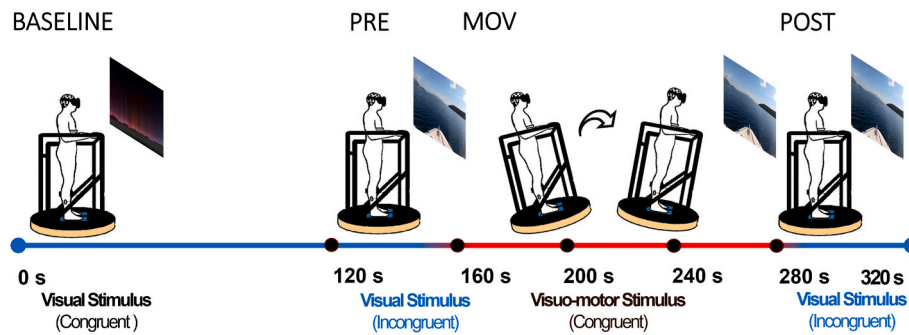


Fig. 1. Schematic Overview of the BioVRSea Experimental Paradigm. The BioVRSea paradigm combines immersive VR with synchronized motion platform perturbations. The 5-min protocol consists of four sequential phases: (1) BASELINE (0–120s): stationary platform with non-sea visual scene; (2) PRE (120–160s): stationary platform with dynamic sea visual stimulation; (3) MOV (160–280s): synchronized platform movement with sea visuals, with wave parameters progressively increasing from 0.5 to 3 Hz frequency and 0–2m amplitude; and (4) POST (280–320s): return to stationary platform with continued dynamic sea visual stimulation. Participants wore VR goggles and a 64-channel EEG cap with amplifiers secured in a backpack, allowing continuous neural recording during platform oscillations. Visual stimulus conditions are indicated as congruent (matching platform state) or incongruent (mismatched platform state) below the timeline.

standing, perturbed postural task. Additional outlier channels were identified based on abnormal spectral properties (threshold = 2 SD) and subsequently interpolated using spherical splines. Bad data segments were not removed. The data were then re-referenced to the common average. Independent component analysis (ICA) was performed using the Picard algorithm, with dimensionality reduction (PCA) set to the effective data rank. Components were classified with ICLabel, and those reflecting artefacts (eye, muscle, heart, or line noise) with probabilities $\geq 70\%$ were rejected. Furthermore, manual rejection of ICs was performed for each EEG recording, resulting in the retention of 32.05 ± 11.30 components per recording (range = 10–47) from the initial 52 components. The retained components were predominantly classified as brain-related (mean proportion: $83.1\% \pm 14.7\%$). In contrast, retained components classified as muscle, eye, heart, channel noise, and line noise were minimal (muscle: $1.0\% \pm 1.8\%$; eye: $0.3\% \pm 0.8\%$; heart: $0.2\% \pm 0.6\%$; channel noise: $1.2\% \pm 2.4\%$; line noise: 0%). These values indicate that the reconstructed EEG signals were dominated by brain-related activity, with only minimal residual contribution from artefact-related components after preprocessing. The cleaned EEG signals were then reconstructed for further analysis.

2.4. EEG microstate analysis

The preprocessed data were then analysed using the *MICRO-STATELAB Toolbox* [16], employing a modified k-means clustering method. The number of clusters was constrained to 4–7, and the k-means algorithm was repeated 100 times to obtain a stable solution. To determine the optimal number of clusters, we computed the spatial similarity with previously published templates [16]. We further confirm the selection, using a meta-criterion composed of 9 optimisation metrics (see the Supplementary Materials, section 2.2 for details), which identified the most appropriate clustering solution among those tested. Clustering was performed on global field power (GFP) peaks, treating maps of opposite polarity as equivalent, and all EEG topographies were normalised before clustering. To assess the robustness of GFP-peak extraction in this movement-rich paradigm, GFP peak counts were also quantified for each subject and phase and compared across Group and Phase (see Supplementary Materials, section 3). For backfitting, cluster solutions ranging from 4 to 7 microstates were considered. Backfitting was performed on GFP peaks only, using a 30-ms smoothing window and a smoothness penalty of 0.3 to promote temporal continuity. In this step, each EEG time point was assigned to the grand-mean microstate map with which it showed the highest spatial correlation. This procedure produced a continuous sequence of microstates over time, from which standard temporal characteristics—mean duration (i.e. the average time each microstate class persists before transitioning to

another state), frequency of occurrence (i.e. how often a microstate class appears per unit time), and relative coverage (i.e. the proportion of total recording time occupied by a given microstate class)—were extracted for each microstate class across the four experimental phases. Additionally, transition probabilities among the five microstate classes were computed as adjusted transition probabilities – i.e., the relative preference or resistance to transition from a specific microstate to another, relative to the chance level.

2.5. Statistical analysis

Statistical analyses were performed using R. Demographic differences between groups were assessed at the subject level; age was compared using a two-sample *t*-test following assessment of equality of variances, and gender distribution was evaluated using a chi-square test of independence (results are detailed in the [Supplementary Materials, Tables 3 and 4](#)). Linear mixed-effects models were fitted using the *lme4* package to assess the effects of task phase, microstate class, and group, including all two- and three-way interactions. To account for the non-independence of repeated measurements within participants, the *subject ID* was included as a random intercept. Dependent variables included the standard temporal parameters of microstate dynamics: mean duration, frequency of occurrence, and relative coverage. Additionally, although the groups did not differ significantly in age ($p = 0.058$), we included age as a covariate. The model was formulated as follows:

$$\text{Parameter} = 1 + \text{Group} * \text{Phase} * \text{Microstate} + \text{Age} + (1|\text{Subject})$$

Significance testing for the fixed main effects and interactions was performed using a Type III analysis of variance, and effect sizes were reported as partial eta-squared (η_p^2). Post-hoc pairwise comparisons were performed with *p*-values corrected for multiple testing using the false discovery rate (FDR) method, and standardised effect sizes (ES) were calculated using Cohen's *d* and were computed from the estimated marginal mean contrasts using the *emmeans* function *eff.size*. Thus, for each contrast, Cohen's *d* was calculated as the estimated contrast divided by the residual standard deviation of the fitted model ($d = \hat{\Delta} / \hat{\sigma}$), where $\hat{\Delta}$ denotes the estimated contrast and $\hat{\sigma}$ the residual standard deviation. The sign of *d* indicates the direction of the contrast, whereas its absolute value reflects the magnitude of the effect. Results are reported with 95% confidence intervals. Model assumptions were verified through inspection of residuals and variance inflation factors (VIF). Residual diagnostics, including Q–Q plots, are provided in section 3 of the Supplementary Materials ([Supplementary Figs. 8–10](#)). To examine group differences and phase-related changes in microstate transition probabilities, a series of linear mixed models (LMMs) was fitted for each

of the 20 adjusted transition probabilities. Each model included Group, experimental phase (BL, PRE, MOV, POST) and their interaction as fixed effects. The *subject ID* was modelled as a random intercept to account for repeated measurements across phases. To control for multiple comparisons across the 20 transition models, p-values for each effect (Group, Phase, Group $\times$ Phase) were corrected using the FDR method. Post-hoc comparisons for significant interactions were performed using estimated marginal means (*emmeans* package), with pairwise contrasts between groups at each phase and between phases within each group. [Supplementary Fig. 2](#) in the Supplementary Materials presents the study's workflow schematically. As an additional quality-control analysis, GFP peak counts were also examined using a linear mixed-effects model with Group, Phase, and their interaction as fixed effects and Subject as a random intercept; the corresponding results are reported in the Supplementary Materials ([Supplementary Figs. 8–10](#)).

3. Results

3.1. A five-map microstate solution reveals marked group differences in microstate D and E topographies

We explored a range of clustering solutions, from 4 to 7 (see section 2.1 of the Supplementary Materials for further details on all computed solutions). A five-map solution demonstrated the greatest similarity to the meta-analytic microstate maps available in the *MICROSTATELAB Toolbox* [16]. Specifically, the spatial correlations with the canonical maps were very strong for maps A (94.3%) and B (94.0%), and remained robust for maps C (85.8%), D (80.4%), and E (70.8%). This choice was further supported by previous findings from a large healthy cohort of 266 participants, in which a five-map solution provided the best model fit and validated the robustness of this approach [14]. Notably, the meta-criterion confirmed that the optimal solution consisted of five clusters (see the Supplementary Materials, section 3 for detailed results). Nevertheless, HC and PD differed in their characteristic microstate

topographies (see [Fig. 2](#)), with the largest discrepancies observed for microstates D and E. While the similarity between the two groups was high for maps A (80.8%), B (83.4%), and C (94.3%), it dropped markedly for maps D (6.5%) and E (0.8%), indicating substantial alterations in these configurations in PD compared to HC.

3.2. Microstate temporal dynamics reveal disease-specific patterns

Linear mixed-effects modelling of the backfitted microstate temporal parameters demonstrated robust main effects of microstate class on duration ($F(2, 591.02) = 8.2271, p < 0.0001, \eta_p^2 = 0.03$), occurrence ($F(2590.94) = 12.8812, p < 0.0001, \eta_p^2 = 0.04$) and coverage ($F(2, 591.11) = 14.3047, p < 0.0001, \eta_p^2 = 0.05$). Significant main effects of group were observed for duration ($F(1, 53.14) = 20.1899, p < 0.0001, \eta_p^2 = 0.28$) and coverage ($F(1, 53.47) = 18.3344, p < 0.0001, \eta_p^2 = 0.26$), reflecting differences between patients with PD and HC, while no group effect was detected for occurrence ($F(1, 53.14) = 1.4813, p = 0.22894, \eta_p^2 = 0.03$). Critically, significant group $\times$ microstate interactions emerged across all three parameters - duration: ($F(2, 591.02) = 18.5459, p < 0.0001, \eta_p^2 = 0.06$), occurrence ($F(2, 590.94) = 15.0270, p < 0.0001, \eta_p^2 = 0.05$), and coverage ($F(2, 591.11) = 20.0603, p < 0.0001, \eta_p^2 = 0.06$), indicating that the PD vs. HC differences varied systematically as a function of microstate class. By contrast, neither the main effects of phase nor the higher-order interactions involving phase reached significance, suggesting that microstate temporal dynamics remained stable across experimental phases. Age, included as a covariate, was a significant predictor of occurrence ($F(1, 53.58) = 5.4763, p = 0.023$) and coverage ($F(1, 54.25) = 9.6209, p = 0.003$), but not duration ($F(1, 53.39) = 0.8941, p = 0.349$); group effects are therefore reported adjusted for age. A complete breakdown of these results is detailed in [Table 1](#). Collectively, these findings highlight microstate class-specific alterations in PD relative to HC, independent of phase-related modulations. Because microstates D and E differed in

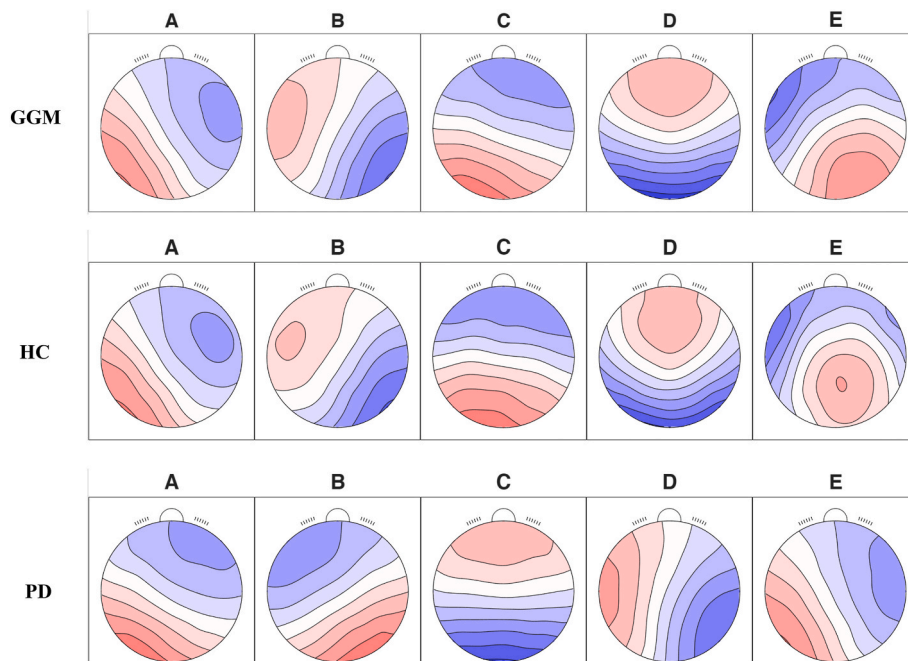


Fig. 2. Grand Mean and Mean Groups Microstates Maps. Scalp topographies of the five microstate classes (A-E) derived from grand average clustering analysis (GGM, top row panel) and for the single groups, shown separately for healthy controls (HC, middle row panel) and Parkinson's disease patients (PD, bottom row panel). Maps display voltage distributions with red indicating positive voltages and blue indicating negative voltages. Contour lines represent isopotential lines. The GGM solution showed optimal correspondence with canonical microstate templates, achieving spatial correlations of 94.3% for map A, 95% for map B, 85.8% for map C, 80.4% for map D, and 70.8% for map E. Spatial correlations between HC and PD topographies were 80.8% for map A, 83.4% for map B, 94.3% for map C, 6.5% for map D, and 0.8% for map E. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

Table 1**Summary of Main Effects and Interactions** - Type III Analysis of Variance tables with Satterthwaite's method for (A) duration, (B) occurrence, and (C) coverage.

A. Duration							
	Sum Sq	Mean Sq	DF (Num Den)		F-value	η_p^2	P-value
Phase	114.10	38.03	3	591.22	1.7100	0.008	0.1637579
Microstate	365.97	182.99	2	591.02	8.2271	0.03	0.0002991 ***
Group	449.06	449.06	1	53.14	20.1899	0.28	< 0.0001 ***
Age	19.89	19.89	1	53.39	0.8941	0.02	0.3486240
Phase*Microstate	68.89	11.48	6	591.02	0.5162	0.005	0.7962184
Phase*Group	85.48	28.49	3	591.22	1.2811	0.006	0.2799378
Microstate*Group	824.99	412.49	2	591.02	18.5459	0.06	< 0.0001 ***
Phase*Microstate*Group	41.78	6.96	6	591.02	0.3131	0.003	0.9302404
B. Occurrence							
	Sum Sq	Mean Sq	DF (Num Den)		F-value	η_p^2	P-value
Phase	0.1353	0.0451	3	591.28	0.1170	0.0004	0.95010
Microstate	9.9295	4.9647	2	590.94	12.8812	0.04	< 0.0001 ***
Group	0.5709	0.5709	1	53.14	1.4813	0.03	0.22894
Age	2.1107	2.1107	1	53.58	5.4763	0.09	0.02303 *
Phase*Microstate	0.8422	0.1404	6	590.94	0.3642	0.003	0.90157
Phase*Group	0.8341	0.2780	3	591.28	0.7214	0.003	0.53948
Microstate*Group	11.5835	5.7918	2	590.94	15.0270	0.05	< 0.0001 ***
Phase*Microstate*Group	0.5479	0.0913	6	590.94	0.2369	0.002	0.96435
C. Coverage							
	Sum Sq	Mean Sq	DF (Num Den)		F-value	η_p^2	P-value
Phase	8.35	2.78	3	591.73	0.1376	0.0006	0.937569
Microstate	579.22	289.61	2	591.11	14.3047	0.05	< 0.0001 ***
Group	371.19	371.19	1	53.47	18.3344	0.26	< 0.0001 ***
Age	194.78	194.78	1	54.25	9.6209	0.15	0.003051 **
Phase*Microstate	43.36	7.23	6	591.11	0.3570	0.003	0.905880
Phase*Group	7.49	2.50	3	591.73	0.1233	0.000	0.946335
Microstate*Group	812.27	406.14	2	591.11	20.0603	0.06	< 0.0001 ***
Phase*Microstate*Group	21.38	3.56	6	591.11	0.1760	0.003	0.983285

topography between groups (Section 3.1), their back-fitted temporal parameters index spatially non-equivalent configurations and cannot be compared across groups without confounding spatial and temporal differences. Between-group temporal comparisons and transition probability analyses were therefore restricted to microstates A, B and C, whose topographies were comparable across groups; the corresponding description of the full five-map results is reported in Supplementary Materials (Supplementary Table 5).

3.2.1. Duration

Post hoc contrasts for microstate duration revealed consistent and robust increases in PD relative to HC for microstates A and B across all experimental phases (see Fig. 3, upper panel). For microstate A, PD patients exhibited significantly longer durations than HC at baseline ($\Delta = 6.412$, $p = 0.0015$, $ES = 1.36$), pre-movement ($\Delta = 6.524$, $p = 0.0013$, $ES = 1.383$), movement ($\Delta = 8.61$, $p < 0.0001$, $ES = 1.825$), and post-movement ($\Delta = 8.498$, $p < 0.0001$, $ES = 1.802$). A comparable pattern was observed for microstate B, with longer durations in PD at baseline ($\Delta = 6.978$, $p = 0.0007$, $ES = 1.480$), pre-movement ($\Delta = 5.675$, $p = 0.0050$, $ES = 1.203$), movement ($\Delta = 8.512$, $p < 0.0001$, $ES = 1.805$), and post-movement ($\Delta = 7.125$, $p = 0.0006$, $ES = 1.511$). By contrast, group differences did not reach significance for microstate C (all $p > 0.10$).

3.2.2. Occurrence

Post hoc contrasts of microstate occurrence revealed no significant between-group differences for any microstate (all FDR-corrected $p > 0.10$; see Fig. 3, middle panel). Although occurrence tended to be lower in PD than HC for microstates A and B — most notably during the pre-movement phase (microstate A: $\Delta = 0.479$, $p = 0.129$, $ES = 0.77$; microstate B: $\Delta = 0.480$, $p = 0.129$, $ES = 0.77$) — these differences did not survive correction for multiple comparisons.

3.2.3. Coverage

Post hoc contrasts of microstate coverage revealed microstate- and phase-specific alterations in PD relative to HC (see Fig. 3, bottom panel). For microstate A, coverage was significantly higher in PD at baseline ($\Delta = 3.994$, $p = 0.0113$, $ES = 0.888$), pre-movement ($\Delta = 4.981$, $p = 0.0006$, $ES = 0.78391$), movement ($\Delta = 4.888$, $p = 0.0024$, $ES = 1.086$), and post-movement ($\Delta = 4.221$, $p = 0.0085$, $ES = 0.938$). Microstate B also showed increased coverage in PD at baseline ($\Delta = 3.742$, $p = 0.0188$, $ES = 0.832$), pre-movement ($\Delta = 4.430$, $p = 0.0054$, $ES = 0.985$), movement ($\Delta = 4.815$, $p = 0.0026$, $ES = 1.070$), and post-movement ($\Delta = 3.665$, $p = 0.0224$, $ES = 0.815$). In contrast, microstate C showed no significant PD–HC differences across phases. These results demonstrate microstate- and phase-specific alterations in coverage in PD, with increased engagement of microstates A and B.

3.2.4. Transition probabilities

Transition probabilities among the topographically group-stable microstates A, B and C were analysed with age-adjusted linear mixed models, and p-values were FDR-corrected across the six transitions. No transition showed a significant main effect of Group after correction (all $p > 0.05$), indicating that overall transition preferences among A, B and C did not differ between PD and HC. However, a significant Group $\times$ Phase interaction emerged for the A→C transition ($F = 4.96$, $p = 0.015$, $\eta_p^2 = 0.08$), indicating that the two groups followed different phase trajectories for this transition. Age-adjusted post-hoc contrasts revealed the source of this divergence: HC showed significant within-group A→C modulation, with the transition probability increasing from baseline to the PRE-movement phase (estimate = 13.74, $SE = 3.68$, $p = 0.0015$, $d = 1.05$) and from baseline to the POST-movement phase (estimate = 12.87, $SE = 3.72$, $p = 0.0021$, $d = 0.99$). In contrast, PD patients showed no significant within-group phase contrasts for A→C (all $p > 0.25$), indicating a stable transition probability across phases.

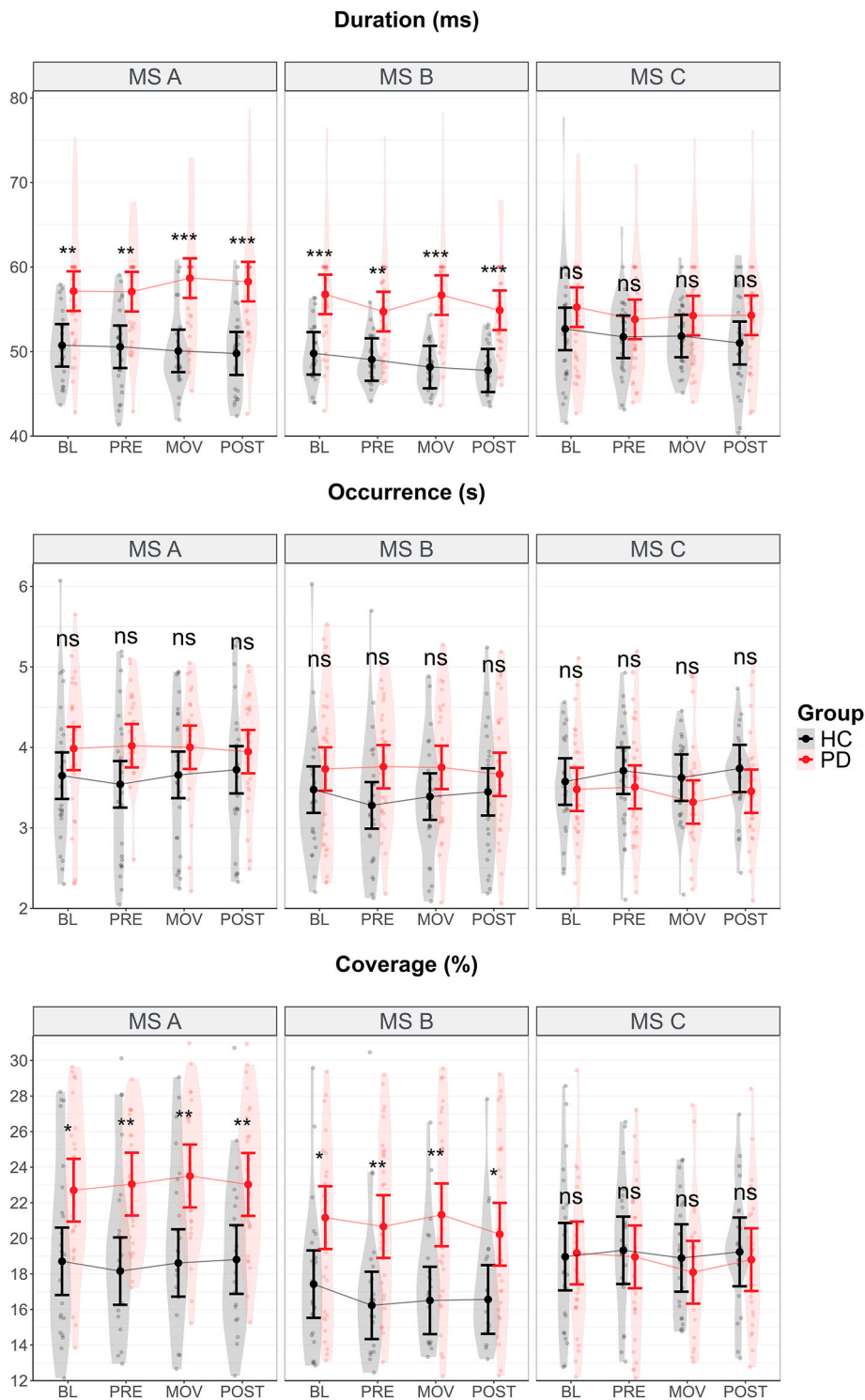


Fig. 3. Estimated Marginal Means of Microstate Metrics Across Groups and Phases. Estimated marginal means of microstates duration (ms), occurrence (s), and coverage (%) for microstates A through E across different BioVRSea experimental phases: Baseline (BL), pre-movement (PRE), movement (MOV), and post-movement (POST). Data are shown for healthy controls (HC, black) and Parkinson's disease patients (PD, red). Individual data points are overlaid on violin plots, with error bars representing 95% confidence intervals. Significant group differences are indicated by asterisks (*p < 0.05, **p < 0.01, ***p < 0.001; ns = not significant). (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

The groups diverged most strongly in the POST-movement phase, where PD patients showed a markedly higher A→C probability than HC ($\Delta = 11.77$, $p = 0.005$, $ES = 0.903$), with a similar trend during the pre-movement phase ($\Delta = 7.75$, $p = 0.058$, $ES = 0.595$); the groups did not differ at baseline or during movement (both $p > 0.28$). Detailed post-hoc contrasts for the A→C transition are reported in [Supplementary](#)

Table 10. A significant main effect of Phase, independent of Group, was observed for the B→C transition ($F = 5.05$, $p = 0.014$, $\eta p^2 = 0.08$), and a significant effect of age was observed for C→B ($F = 8.98$, $p = 0.024$, $\eta p^2 = 0.13$). A complete breakdown of all 6 transitions is provided in [Supplementary Table 8](#) for description; moreover, for descriptive purposes, the full 20 transitions are reported in

Supplementary Table 9, including those involving microstates D and E.

4. Discussion

To our knowledge, this is the first study to investigate EEG microstate dynamics during immersive VR-based postural perturbations in individuals with Parkinson's disease (PD). Our findings align with and extend previous resting-state microstate research in PD. Chu and colleagues [13], employing a 4-cluster solution, demonstrated that PD patients exhibited variant spatial topography in specific microstate classes compared to healthy controls, with abnormal microstate features corresponding to class F, as named by Tarailis and colleagues [9]. Consistent with our results, they found that PD patients showed longer durations, greater occurrence, and greater coverage of microstates A and B than healthy controls. Similarly, Guo and colleagues [17] reported significant topological changes in microstate D in PD patients compared to controls, along with altered temporal features of microstates C and D. These convergent findings across different methodological approaches strengthen the evidence for microstate-specific alterations in PD. At the same time, our study extends this literature by showing that such between-group differences are also detectable during the execution of an active postural control task. Convergent connectivity-based findings using the same paradigm also report PD-related reductions in network engagement during equivalent task phases [18]. A critical finding of our research is that while HC maintained microstate topographies closely resembling canonical maps, PD patients exhibited profound spatial distortions, particularly in microstates D and E. This represents a pronounced between-group difference in the spatial organisation of microstates D and E. The current design—without source localisation, behavioural, neuropsychological, or clinical metrics—cannot attribute this difference to particular network-level mechanisms. Instead, we describe it as a topographic group difference rather than as proof of network dysfunction. Moreover, because all patients were assessed ON medication, the design cannot disentangle disease-related effects from dopaminergic medication effects or from compensatory processes; the observed differences should be read as group-level differences in microstate organisation under the tested conditions. OFF-medication testing during immersive postural perturbation was not deemed appropriate for safety reasons.

4.1. Restriction of temporal dynamics to topographically equivalent maps

Between-group comparisons of microstate temporal parameters are valid only when a microstate label denotes equivalent topography across groups. While separate per-group templates inflate Type I error, a shared template applied to topographically divergent maps confounds spatial and temporal effects; topographic group differences should therefore be tested before temporal parameters are compared [19]. We accordingly assessed the PD–HC spatial correlation for each group-level map and restricted the analysis accordingly: maps A, B, and C were highly correlated across groups, whereas maps D and E were not (6.5% and 0.8%, respectively). All between-group comparisons of temporal dynamics and transition probabilities were thus restricted to maps A–C—transitions involving D or E were excluded, as both endpoints of a transition must be topographically matched—while the topographic divergence of maps D and E is reported as a finding rather than carried into the temporal analysis.

4.2. Microstate dynamics did not differ across experimental phases

A central result of this study is the largely absent Phase $\times$ Microstate $\times$ Group interaction: within the present VR-based postural paradigm, PD participants and healthy controls did not differ detectably in phase-dependent modulation of microstate temporal parameters for most microstates. This was broadly corroborated by the transition probability analysis, in which Group $\times$ Phase interactions did not

survive FDR correction for five of the six A–C transitions; the exception was the A→C transition, which remained significant after correction ($p_{\text{fdr}} = 0.015$), indicating a phase-dependent group difference in the sequencing between microstates A and C. The group-level differences identified in this study were thus largely, though not entirely, unaccompanied by phase-dependent modulation: they were present throughout the task for most microstates and transitions, consistent with a predominantly stable, persistent difference in microstate organisation in PD rather than one broadly reconfigured across phases, alongside a more circumscribed phase-dependent component localised to the A→C transition. This largely phase-invariant pattern should be read in light of both the paradigm and the prior literature. BioVRSea is not a graded task in which postural challenge escalates across phases; it imposes substantial postural and sensorimotor demand from the outset, so that even the earliest phase is a demanding postural-control condition rather than a low-demand baseline. A predominantly stable group difference is therefore expected under more than one account: it may be largely trait-like and independent of the specific manipulation, or it may be sensitive to postural demand yet already largely expressed from time 0 and thus show limited further graded change across subsequent phases. Such persistence is also what prior work would predict, as neuroimaging of postural control has repeatedly shown altered cortical and cerebellar activity in PD relative to matched controls [20–22], present even in medicated patients [20] and therefore unlikely to reflect a transient pharmacological state. The present design, however, cannot distinguish a demand-independent difference from one elicited at task onset and sustained thereafter, and the largely null pattern of interactions may also reflect limited power to resolve three-way effects, particularly for the isolated A→C exception. The data therefore support predominantly stable, phase-invariant group-level differences in microstate organisation under the conditions tested, with one transition (A→C) showing evidence of phase-dependent group divergence, and do not license broader inferences regarding phase-specific or demand-graded modulation beyond this transition.

4.3. Temporal differences in microstates a and B

Microstates A and B showed greater duration and coverage in the PD group than in healthy controls across all experimental phases. In the broader microstate literature, microstate A has been associated with phonological processing networks and microstate B with visual networks [8,9], and longer microstate durations have been interpreted as reflecting the stability of transiently synchronized large-scale networks [23]. At the parameter level, the present results are consistent with those of Chu and colleagues [13], who reported increased duration, occurrence, and coverage of microstates A and B in PD relative to controls. Prolonged durations have also been described in other basal ganglia-related conditions; Kong and colleagues [24] reported increased durations across four microstate classes in stroke patients following basal ganglia injury. The functional associations summarised above derive from independent datasets and are reported here only as context. Because the present study did not include source localisation or behavioural, neuropsychological, or clinical measures, the increased duration and coverage of microstates A and B are reported as group-level differences in microstate temporal organisation under the tested conditions, without inference regarding their neural sources or functional significance.

4.4. Implications of microstates D and E disruptions

In the PD group, the topographies of microstates D and E departed markedly from the canonical microstate maps, with spatial correlations of only 6.5% and 0.8%, respectively. Microstates D and E also showed marked topographic divergence between groups; because the maps were not equivalent across groups, between-group comparisons of their temporal parameters were interpreted with caution. In the canonical

microstate literature, microstate D has been associated with frontoparietal and dorsal attention networks and microstate E with the default mode network and internally directed cognition [8,9,25]. At the parameter level, Pal and colleagues [26] reported reduced occurrence of microstate D in PD. These functional associations refer to the canonical maps derived from independent datasets and are included only for context; given the topographic divergence observed here, they cannot be assumed to apply to the present microstate maps D and E. The present design did not include source localisation or behavioural, neuropsychological, or clinical measures. Accordingly, the topographic differences in microstates D and E are reported as group-level differences in microstate organisation under the tested conditions, without inference regarding the affected networks or their functional or clinical significance.

4.5. Microstate transitions show limited, A→C-Specific group differences

The analysis of transition probabilities revealed a more circumscribed pattern of group differences than the temporal dynamics measures. Among transitions between the microstates A, B and C, no overall difference in transition preference distinguished PD from HC after FDR correction. The groups differed only in how the A→C transition was modulated across the protocol: HC showed significant A→C modulation across phases, with increases from BL to PRE- and POST-movement phases; PD patients maintained a stable A→C transition profile across all phases, resulting in maximal between-group divergence at the POST-movement phase. Because this effect rested on a single transition and emerged only as a Group × Phase interaction, it should be interpreted with caution. Confirmatory analysis was restricted to the A→C transitions reported above (Supplementary Table 8); transitions involving microstates D and E lack topographic equivalence across groups and are reported for descriptive purposes only in the full transition matrix (Supplementary Table 9); their interpretation is further limited by the topographic non-equivalence of D and E across groups. Overall, the transition results indicate that group differences in microstate dynamics during the BioVRSea paradigm were expressed more in the temporal parameters of individual states than in the probabilistic structure of transitions between them.

5. Limitations

We recognise several limitations in our study. First, the lack of behavioural, clinical and neuropsychological data hinders the ability to correlate EEG microstate dynamics with observable motor and cognitive functions, such as balance ability or motor symptom severity. In addition, the absence of detailed clinical and demographic measures, including motor severity, disease duration, cognitive status, postural impairment, and years of education, limits the interpretability of the observed EEG microstate alterations and precludes a formal assessment of between-group comparability on these variables. Because the present study was conceived as a pilot case-control investigation and included a modest sample, it was not designed or powered for robust correlation or subgroup analyses. As a result, the observed group-level differences in microstate organisation should be regarded as hypothesis-generating rather than confirmatory, and the present data warrant no functional, cognitive, or network-level interpretation of the individual microstates. Moreover, the lack of longitudinal data limits our ability to assess how microstate dynamics evolve over time in PD and to correlate them with long-term clinical outcomes. Additionally, increasing the sample size would strengthen the robustness of the findings by allowing more detailed subgroup analyses and a more precise characterisation of how these factors relate to microstate temporal dynamics. The PD cohort comprised early-stage patients assessed ON medication; the present findings therefore apply to treated, early-stage disease and should not be generalised to advanced PD or to the OFF-medication/unmedicated state, which future studies should address. A larger cohort would

enable the examination of specific PD subtypes and the stratification of participants by age, sex, and disease severity stage. The narrow 2–20 Hz cut-off used in the preprocessing may reduce sensitivity to higher-frequency beta/gamma activity, which is particularly relevant in PD, and may therefore influence the resulting microstate maps and temporal parameters. In addition, the duration of the experimental phases may have been insufficient to capture adaptive or habituated transitions in microstate dynamics. This may have reduced the paradigm's sensitivity to detect phase-dependent modulation of large-scale network activity. Finally, in relation to the applied methodology, as highlighted in the body of EEG microstates literature [27], when two groups exhibit substantial differences in microstate topographies, strong conclusions regarding differences (or the absence of differences) in the derived microstate parameters should be avoided, as this was the case for microstates D and E.

6. Conclusions

The combination of the BioVRSea protocol with EEG microstate analysis revealed group-related differences in large-scale brain dynamics during an ecologically valid postural-control task. Marked topographical differences were observed, particularly for microstates D and E, where maps in participants with Parkinson's disease (PD) showed limited correspondence with canonical configurations. At the temporal level, between-group comparisons were restricted to the topographically equivalent microstates A–C; on these, PD patients showed increased duration and coverage of microstates A and B across all phases, whereas the temporal parameters of microstates D and E—whose maps were not topographically equivalent across groups—are reported as descriptive only (Supplementary Materials). The absence of significant phase-dependent effects indicates stable, group-level differences rather than task-phase-specific modulation. Because all patients were assessed ON medication and no behavioural, neuropsychological, or clinical correlates were available, the present design cannot disentangle disease-related effects from medication effects or compensatory processes, and does not support conclusions regarding underlying functional or network mechanisms. Given the exploratory nature of this pilot study, the absence of clinical correlations, and the ON-medication status of participants, these findings should be considered preliminary. Nevertheless, they provide a foundation for future studies integrating clinical, behavioural, and multimodal approaches to clarify the role of microstate alterations in PD.

CRedit authorship contribution statement

Carmine Gelormini: Writing – original draft, Visualization, Software, Methodology, Investigation, Formal analysis, Data curation. **Lorena Guerrini:** Writing – review & editing, Data curation. **Federica Pescaglia:** Writing – review & editing, Formal analysis. **Ida Maruotto:** Writing – review & editing, Data curation. **Romain Aubonnet:** Writing – review & editing, Software. **Halldór Jónsson:** Writing – review & editing, Supervision, Project administration. **Hannes Petersen:** Writing – review & editing, Supervision. **Gylfi Örn Þormar:** Writing – review & editing, Supervision, Project administration. **Giorgio Di Lorenzo:** Writing – review & editing, Supervision, Software, Methodology, Formal analysis. **Mahmoud Hassan:** Writing – review & editing, Supervision. **Paolo Gargiulo:** Writing – review & editing, Resources, Project administration, Methodology, Funding acquisition, Conceptualization.

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Declaration of competing interests

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Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.parkreldis.2026.108968>.

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