RESEARCH ARTICLE

Aberrant Beta-Band Network Alteration Preceding Freezing of Gait in Parkinson's Disease

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ABSTRACT: Background: Freezing of gait (FOG) is a debilitating motor symptom observed in the advanced stages of Parkinson's disease (PD), characterized by an abrupt inability to initiate or continue forward walking. Whole-brain functional connectivity analysis has shown promise in elucidating the underlying pathophysiology and identifying potential biomarkers in PD. However, the specific changes in local brain networks during the transition from normal gait to freezing remain unclear.
Objectives: This study aimed to investigate changes in brain network organization during the transition to FOG compared with the transition to voluntary stopping.
Methods: Eighteen PD patients with FOG performed walking tasks designed to trigger either freezing or voluntary stop events, while undergoing simultaneous ambulatory electroencephalography (aEEG) recording. Functional connectivity was estimated using phase-locking value (PLV) across multiple frequency bands, measuring the consistency of phase synchrony between brain regions, and examined network organization using graph modularity, an index of how strongly the brain segregates into functionally specialized subnetworks, focusing on the 2-second time windows preceding each event.
Results: Transitions to freezing were characterized by increased local beta-band connectivity within right frontoparietal, middle-frontal, parietal-occipital, visual, and bilateral insula regions, alongside reduced connectivity between frontal and posterior areas in lower-frequency bands.
Conclusions: Increased local beta segregation and reduced fronto-posterior connectivity may reflect network alterations that precedes freezing episodes. Such patterns could help identify neurophysiological markers for predicting and potentially preventing FOG in PD. © 2026 The Author(s). *Movement Disorders* published by Wiley Periodicals LLC on behalf of International Parkinson and Movement Disorder Society.

Key Words: electroencephalography; freezing of gait; functional connectivity; local network; Parkinson's disease

Freezing of gait (FOG) is a debilitating phenomenon characterized by the incapacitating, sudden, and involuntary cessation of gait, commonly observed in the advanced stages of Parkinson's disease (PD).[1] The emergence of FOG significantly impacts quality of life with a loss of independence and increased morbidity due to an increased risk of falls.[2] Although the underlying pathophysiology remains incompletely understood,[3,4] FOG has been suggested to arise from an overload in cortico-striatal circuitry (exacerbated by dopamine depletion) triggering excessive activity in the output nuclei of the basal ganglia inhibiting locomotor regions of the brainstem.[5,6]

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Although disruptions in basal ganglia circuits are well established in PD, emerging evidence from neuroimaging studies suggest that FOG is not solely explained by localized basal ganglia dysfunction but involves widespread network-level changes across cortical and subcortical regions.[7,8] Previous work has highlighted the role of impaired connectivity between motor, cognitive, sensory, and limbic networks in FOG, suggesting that deficits in sensorimotor integration and executive function may contribute to gait freezing.[7] Given this complexity, examining neural activity across distributed brain networks would appear critical to our understanding of FOG. Whilst functional neuroimaging has been helpful for localizing the neuroanatomical substrates of freezing,[7,8] its paroxysmal nature means that such approaches traditionally lack the temporal resolution required to capture dynamic changes within these networks relevant to FOG.

One approach for addressing the issue of temporal resolution at a network level has been the utilization of electrophysiological techniques (for overview, see Cui and Lewis[9]). Studies utilizing a virtual reality gait paradigm in PD patients undergoing deep brain stimulation surgery have recorded an increase in the pathological beta and theta rhythms within the subthalamic nucleus just prior to freezing onset.[10,11] Surface electroencephalography (EEG) has highlighted the relationship between impaired motor control and exaggerated beta-band activity in PD patients with FOG,[12] suggesting dysfunctional cortical–subcortical communication with increased coupling between the cerebral cortex and the basal ganglia.[7,13] However, relatively few studies have examined whether these neural network alterations can be detected during ambulatory surface EEG (aEEG), especially during the transition phase preceding the onset of a FOG episode. Previous research utilizing aEEG has shown that freezing episodes are associated with abnormal increases in the theta-band activity within the central and frontal areas.[14] Additionally, other studies have highlighted differences in the delta and low-beta power in the central area during FOG events compared with voluntary stopping (VS).[15] Another study using aEEG investigated turning-related freezing in PD patients and found that FOG during turning was associated with significant alterations in the high-beta and theta power over occipital and parietal regions.[16] However, existing studies have largely focused on regional power changes, and a network-level understanding of brain alteration during transitions remains limited.

To address this deficiency in the literature, we explored aEEG measures of functional connectivity during FOG and its transitions, which have been widely applied to examine frequency-specific signal correlations between brain networks.[17] Interregional neural coordination was quantified by the phase-locking value (PLV), which measures the consistently of phase synchrony between oscillatory signals.[18,19] Network organization was further examined using graph-theoretical modularity, indicating functional network segregation.[20,21] Altered modularity has been associated with impaired sensorimotor integration and executive-motor coupling, key processes implicated in FOG.[22] These measures provide insights into large-scale network dynamics underlying transition from normal walking to freezing and may inform future targeted interventions to reduce fall risk.

The current study used aEEG to examine network-level neural oscillation changes during the transition to FOG (trFOG) compared with voluntary stopping (trVS) in PD patients with FOG. Functional connectivity within and between predefined cortical networks was analyzed to identify potential markers for FOG prediction, and graph-theoretical modularity was calculated to assess differences in local network alteration between trFOG and trVS, finding a unique network signature preceding freezing.

Patients and Methods

Patients

We recruited 18 patients with PD and FOG from the Parkinson's Disease Research Clinic at the Brain and Mind Centre, University of Sydney (Table 1). All participants endorsed FOG on Item 3 of the self-reported Freezing of Gait questionnaire (FOG-Q)[23] and had FOG confirmed by a clinical specialist (S.J.G.L.). No participant had significant depression (Diagnostic and Statistical Manual of Mental Disorders, Fourth Editon [DSM-IV] criteria) or dementia.[24] Clinical assessments were conducted during the practically defined OFF-medication state (overnight withdrawal of dopaminergic therapy) and included the International Parkinson and Movement Disorder Society Unified Parkinson's Disease Rating Scale-Section III (MDS-UPDRS-III),[25]

TABLE 1 *Demographic and clinical characteristics of Parkinson's disease patients*

Characteristic	Value
Number	18
Age, years	69.9 ± 11.1
Disease duration, years	10 ± 2.8
Hoehn and Yahr, stage	2.8 ± 0.9
UPDRS-III	48.8 ± 13.7
FOG-Q total	10.6 ± 5.2
MoCA	26.8 ± 2.1

Abbreviations: UPDRS-III, Unified Parkinson's Disease Rating Scale-Section III; FOG-Q, Freezing of Gait Questionnaire; MoCA, Montreal Cognitive Assessment. Data are expressed as mean ± standard deviation of variables.

Montreal Cognitive Assessment (MoCA),[26] and FOG-Q. The University of Sydney Human Research and Ethics Committee approved the study and written informed consent was obtained (HREC 2013/945).

Experimental Design and Procedure

As described in Shine et al.,[27] participants performed a structured series of Time-Up and Go (TUG) tasks to provoke FOG (Fig. 1). Each trial started from a seated position and was video recorded for offline behavioral annotation.[15,28] Two experienced clinical researchers independently reviewed all video recordings to identify FOG episodes. Three 2-second epochs were extracted: (i) FOG, starting at freezing onset; (ii) transition-to-freezing (trFOG), immediately preceding the FOG onset; and (iii) normal walking (NW), during uninterrupted walking, which served as the baseline condition. The 2-second window length has been validated in prior studies.[15]

In the VS task, participants were instructed to stop voluntarily upon hearing an externally delivered verbal 'stop' command (Fig. 1), constituting a cued, reactive stopping paradigm, distinct from self-initiated VS. The 'stop' cue was delivered at unpredictable points while participants walked back toward the chair after completing the turn. A target box placed 10 m from the starting point served as a spatial reference to standardize walking distance and reduce cue anticipating. Upon hearing 'stop', participants were required to stop immediately and remain stationary for 5–10 seconds before resuming walking. Three 2-second epochs were extracted: (i) VS, beginning at physical cessation of walking; (ii) transition-to-voluntary stop (trVS), defined as the interval between the verbal 'stop' and the physical stopping (using a fixed 2-second time window despite natural variability in stopping latency); and (iii) normal walking (NW), during uninterrupted walking, serving as the baseline condition.

This design enabled direct comparison of involuntary FOG with reactive, externally cued VS, allowing isolation of neural dynamics associated with intentional movement cessation. To minimize order effects and habituation, TUG and VS trials were presented in a pseudo-random order.

Data Acquisition and Analysis

During tasks, aEEG was recorded using a wearable BioSemi Active-Two system with 32 Ag-AgCl electrodes according to the 10–20 International system (BioSemi Systems, Amsterdam, The Netherlands). Data were sampled at 500 Hz, down-sampled to 250 Hz, and band-pass filtered between 1 and 30 Hz. Artifact removal used line-noise mitigation (*pop_cleanline*), flat-line cleaning (*clean_flatlines*; 3 s threshold), and noisy channels detection (*clean_channels*). An average of 2.78 ± 3.42 (range: 0–13) EEG channels were identified as noisy and subsequently interpolated using spherical splines before being re-referenced to the average.[29] To further mitigate the movement-related artifacts, short segments with abnormal spectral power were removed (*pop_rejcont*; 0.5 s windows), followed by rejection of non-stationary artifact windows (*clean_windows*; >15% of channels deviation). These steps are

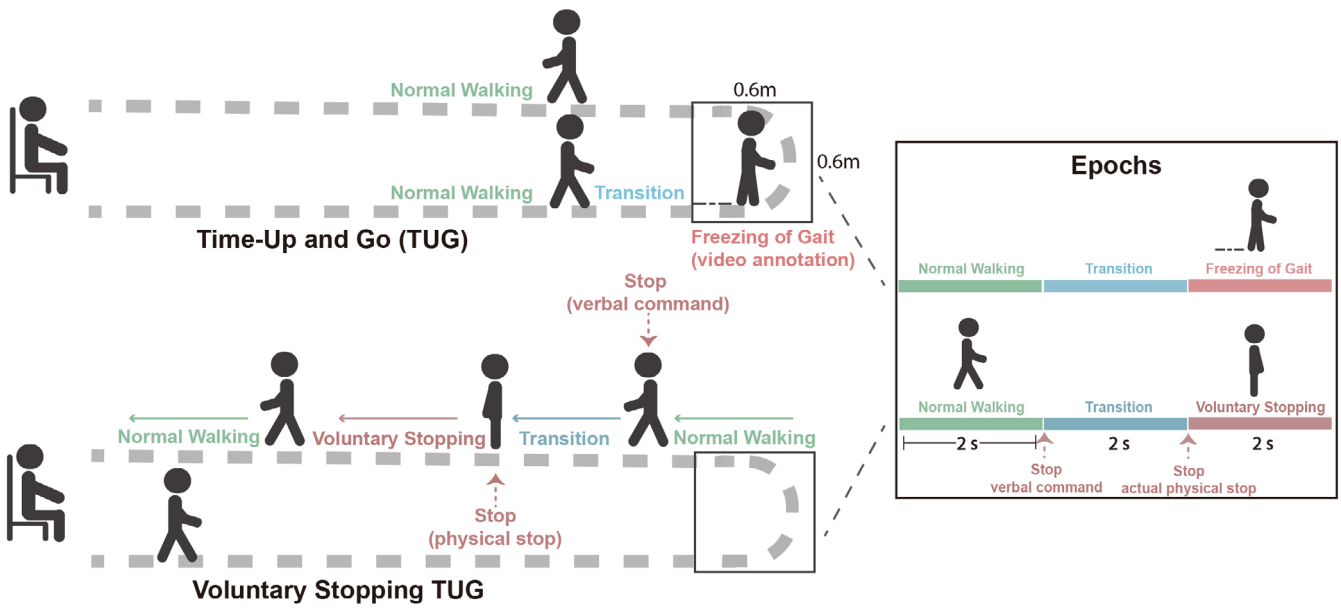

FIG. 1. Experimental protocol for the Time-Up and Go (TUG) and voluntary stopping (VS) task. Participants performed a series of TUG trials designed to provoke freezing of gait (FOG). In the VS task, participants were instructed to voluntarily stop walking upon hearing a verbal 'stop' command. Time-locked ambulatory electroencephalography (aEEG) and whole-body video recordings were required throughout both tasks to identify and analyze epochs, including normal walking (NW), transitions (trFOG/trVS), and either FOG or VS. [Color figure can be viewed at wileyonlinelibrary.com]

particularly important for aEEG to suppress step-related and motion-induced noise bursts. After preprocessing, an average of 29.22 ± 3.42 (range: 19–32) channels per participant remained for analysis.

Adaptive mixed independence component analysis (AMICA)[30] was then applied to decompose the EEG data into statistically independent components (ICs),[31,32] using ICLabel.[33] Only eye-related ICs (probability >0.5) were removed. No additional non-brain ICs were rejected, following recommendations for conservative ICA cleaning in mobile EEG to avoid misclassifying movement-related cortical activity.[34] On average, 24.33 ± 2.72 (range: 20–29) ICs per participant were retained for further analysis. All analyses were performed using custom-written scripts based on MATLAB 2022b (MathWorks Inc., Natick, MA, USA) with EEGLAB toolbox.[35,36]

A total of 277 FOG episodes (freezing duration >1 s) from both TUG tasks and 51 VS trials were included. Four FOG episodes occurred during VS trials but did not interrupt the VS task, with an average duration of 11.91 ± 4.15 seconds prior to the VS command. Although efforts were made to obtain an equal number of trials for trFOG and trVS conditions, participant well-being was prioritized, leading to variable trial numbers across participants and conditions. Sessions were discontinued if discomfort or fatigue was reported. Only those with valid data in both FOG and VS conditions were retained for within-subject, condition-based statistical analyses. Participants who experienced no FOG or no VS trials did not contribute to paired comparisons, resulting in an effective paired sample size of N = 8 for condition-level analyses (Table S1). Each participant completed a minimum of four turning trials with the remaining trials varying based on fatigue and safety considerations.

Functional Connectivity Analysis

Functional connectivity (FC) was analyzed using Brainstorm toolbox (version 01-Apr-2022).[29] Cortical regional time series were reconstructed using a dipole-based source localization approach[37] and parcellated according to the 68-region Desikan–Killiany atlas.[38] PLV was used to quantify FC by measuring the phase synchrony[18] across the entirety of brain nodes, with values ranging from 0 (no phase locking) and 1 (full synchrony). PLV was calculated for each 2-second epoch within five frequency bands (delta: 1–4 Hz; theta: 4–8 Hz; alpha: 8–12 Hz; beta: 12–30 Hz; broadband: 1–30 Hz) and averaged within condition. The highest 10% of PLV values were retained for the estimation of network properties to

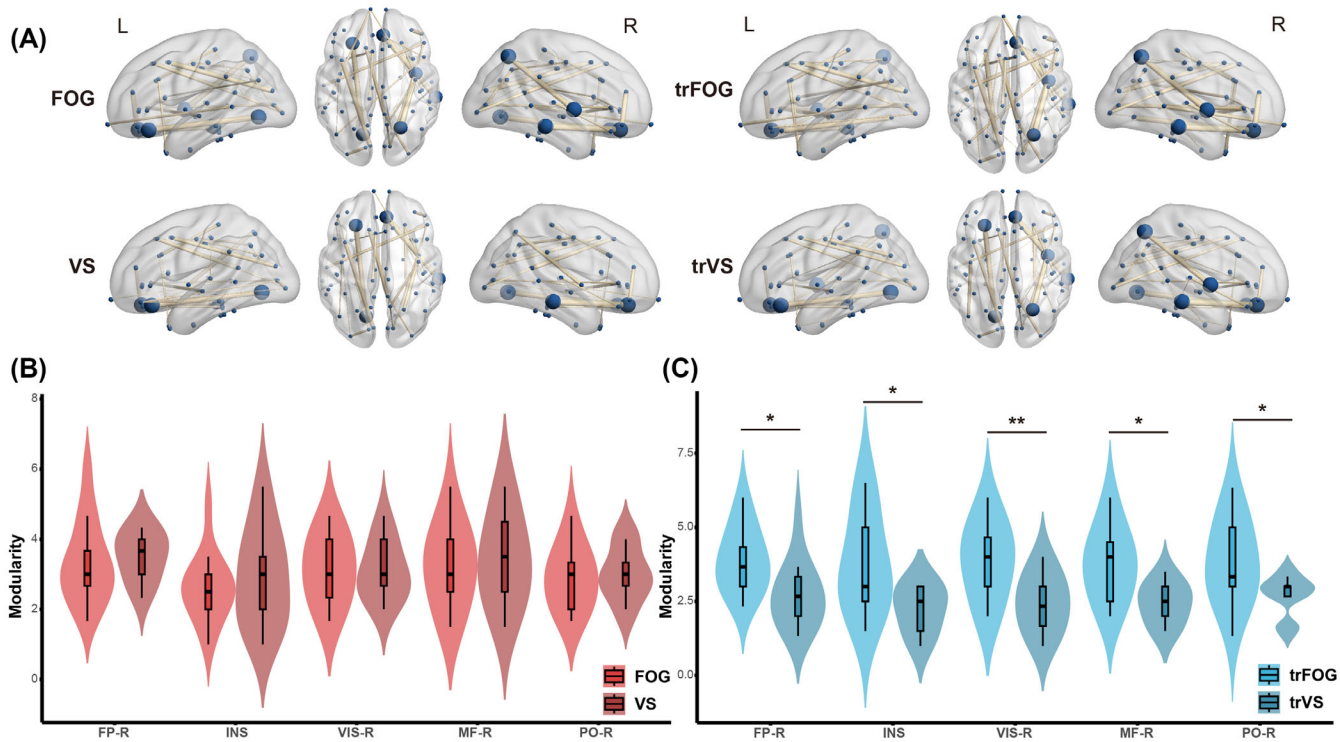

FIG. 2. Graph theory of modularity of the brain network in the beta band. (A) Nodes represent brain regions based on the 68-region Desikan–Killiany atlas. Edges indicate significant connections between nodes with edge size reflecting the connection strength. Conditions are labeled as follows: trFOG (transition to freezing of gait), FOG (freezing of gait), trVS (transition to voluntary stopping), and VS (voluntary stopping). Key networks highlighted include the right frontoparietal (FP-R), middle frontal (MF-R), parieto-occipital (PO-R), visual (VIS-R), and insula (INS) networks, visualized by the BrainNet Viewer.[37] (B) Graph properties of modularity in the beta band for FP-R, MF-R, PO-R, VIS-R, and INS networks. (*, ** indicate statistical significance at $P < 0.05$ and $P < 0.01$, respectively). [Color figure can be viewed at wileyonlinelibrary.com]

reduce spurious connections and ensure a consistent network density across participants,[39] with the remaining values set to zero. Additional details are provided in Supplementary Materials.

Network organization was characterized using graph theory by converting PLV matrices into weighted adjacency matrices. Functional segregation was estimated by modularity, which quantifies the degree of functional segregation, with higher modularity indicating more segregated, specialized network organization and lower values reflecting reduced segregation and potentially less efficient information flow.[40] Modularity was calculated by identifying the optimal community structure that maximized within-module connectivity.[20,22] Functional integration was assessed using participation coefficient and global efficiency. Participation coefficient quantified the distribution of each node's connections across modules,[41] and global efficiency was calculated as the average inverse shortest path length across the network.[42] Additional details are provided in Data S1.

Analyses were performed at the whole-brain level and within and between networks of interests. The 68 cortical regions were grouped into canonical brain networks: frontoparietal, middle-frontal, sensorimotor, parieto-occipital, temporal, occipital networks (each subdivided into left and right hemispheres), and bilateral insula (Fig. 3D).

Statistical Analysis

Statistical analyses were performed in R (RStudio Inc., Boston, MA, USA) using *lme4*, *lmerTest*, *emmeans*, and *dplyr*. Because not all participants contributed data to all conditions (FOG, trFOG, VS, trVS), the dataset contained missing observations by design. Connectivity was estimated within fixed 2-second epochs and then averaged across all episodes within each condition for each participant, yielding a single, stable subject-level estimate per condition and reducing within-subject noise while avoiding disproportionate weighting of

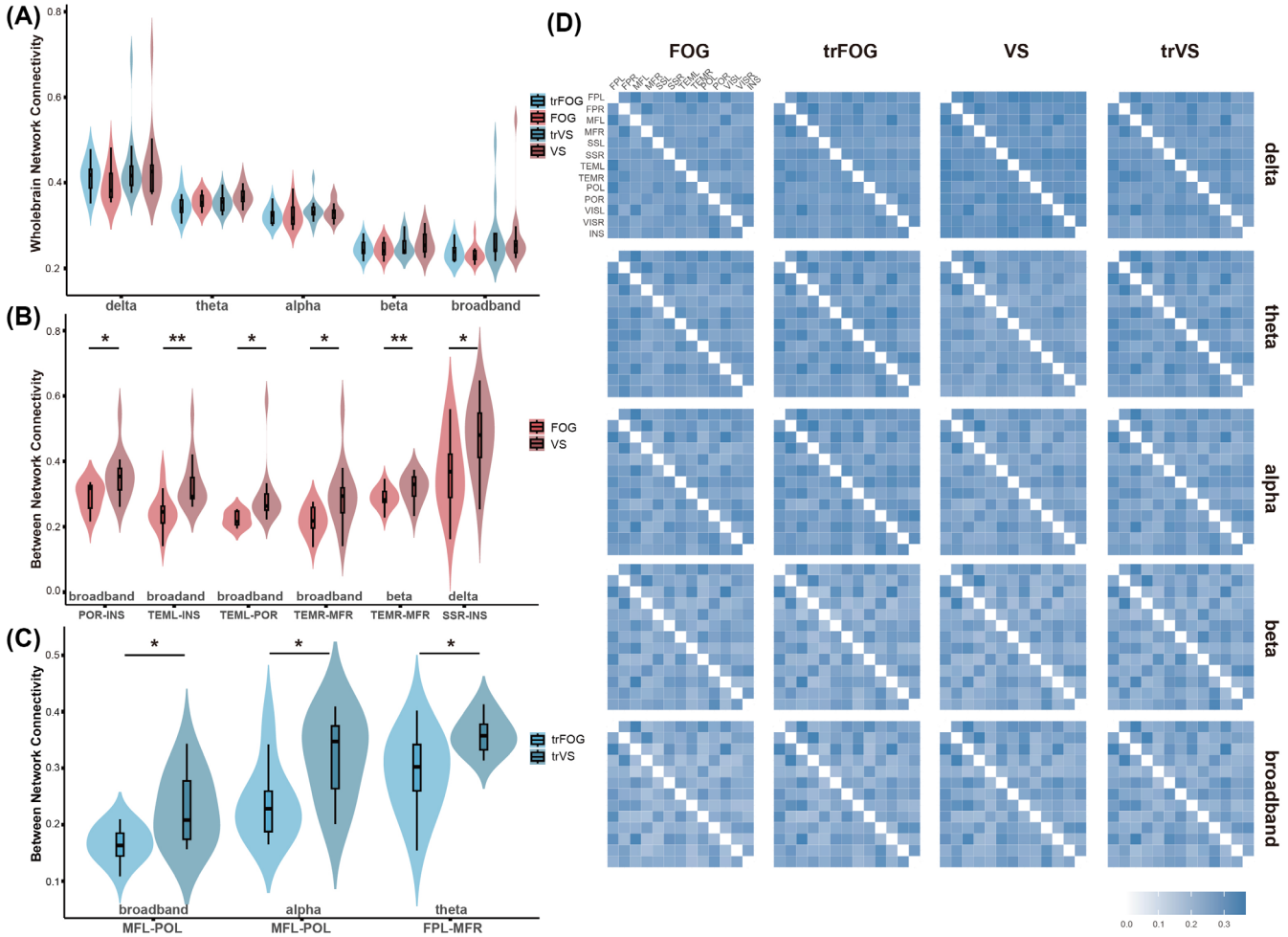

FIG. 3. Functional connectivity of the brain network. (A) Whole-brain networks connectivity across delta, theta, alpha, beta, and broadband frequency under the four conditions. (B) Between-network connectivity under the freezing of gait (FOG) and voluntary stopping (VS) conditions. (C) Between-network connectivity under the transition to FOG (trFOG) and transition to VS (trVS) conditions. (D) Synchronization matrix of whole-brain networks of interest. (*, ** indicate statistical significance at $P < 0.05$ and $P < 0.01$, respectively). [Color figure can be viewed at wileyonlinelibrary.com]

participants with a large number of episodes. To appropriately account for this unbalanced structure and to separate within-subject from between-subject variance, we used linear mixed-effects models (LMMs) with condition (FOG, trFOG, VS, trVS) as a fixed effect and subject as a random factor. Post hoc pairwise comparisons were Bonferroni-corrected. Degrees of freedom were estimated using Satterthwaite's approximation and therefore varied across network-specific models depending on the effective sample size and variance–covariance structure. Data were visualized using custom *ggplot2* scripts, with significance level (α) set at 0.05. Data supporting the findings can be made available upon request.

Results

Demographic and clinical characteristics of the PD cohort are reported in Table 1. The mean age of the sample was 69.9 ± 11.1 years, with an average MDS-UPDRS-III score of 48.8 ± 13.7 and a Hoehn and Yahr (H&Y) stage of 2.8 ± 0.9. The mean FOG-Q score was 10.5 ± 5.2 and the MoCA was 26.8 ± 2.1.

Altered Within-Network Configurations Characterize Transition to Freezing

Having identified between-network changes, we first explored the within-network ('local') changes that might characterize differences between the clinical conditions. Using LMMs with subject as a random factor, we observed a significant overall effect of condition on beta-band modularity within the right frontoparietal network (FP-R) ($F_{(3,48)} = 2.85$, $P = 0.047$), right visual (VIS-R) ($F_{(3,48)} = 4.338$, $P = 0.009$), right middle frontal (MF-R) ($F_{(3,48)} = 3.420$, $P = 0.024$), right parieto-occipital (PO-R) ($F_{(3,38)} = 3.446$, $P = 0.026$), and insula (INS) ($F_{(3,37)} = 4.056$, $P = 0.014$).

Post hoc Bonferroni-corrected pairwise comparisons revealed a significant difference, with the trFOG showing higher modularity compared with trVS in the beta band within the following networks (Fig. 2B). These within-subject contrasts are based on participants with valid data in both conditions (paired N = 8): FP-R (1.154 ± 0.410, t = 2.8815, $P = 0.0448$), VIS-R (1.4359 ± 0.402, $P = 0.006$), MF-R (1.346 ± 0.440, $P = 0.024$), PO-R (1.124 ± 0.381, $P = 0.032$), and INS (1.467 ± 0.458, $P = 0.016$).

To evaluate whether clinical factors influenced the results, we conducted additional LMMs analyses including disease duration, MDS-UPDRS-III, and FOG-Q as covariates. The main effects of condition remained significant, indicating that these factors did not alter the primary findings (Table S3). No significant differences were found in other frequency bands (delta, theta, alpha, and broadband) (Fig. S4). No significant differences across the clinical conditions were identified on secondary network measures such as participation coefficient and global efficiency (Figs S5, S6).

Reduced Between-Network Connectivity Precedes Freezing Episodes

Across conditions, LMMs identified significant differences in multiple network pairings. In the broadband signal, significant condition effects were observed for the following connections: TEM-L and INS ($F_{(3,39)} = 4.569$, $P = 0.008$), TEM-L and PO-R ($F_{(3,38)} = 5.902$, $P = 0.002$), TEM-R and MF-R ($F_{(3,38)} = 4.504$, $P = 0.008$), PO-R and INS ($F_{(3,39)} = 4.054$, $P = 0.013$), and MF-L and PO-L ($F_{(3,36)} = 5.329$, $P = 0.004$). A marginal effect was also observed in the delta band ($F_{(3,38)} = 4.569$, $P = 0.052$). Additional significant effects were observed in the beta band for TEM-R and MFR ($F_{(3,34)} = 4.718$, $P = 0.007$); and in the theta in the FP-L and MF-R ($F_{(3,35)} = 5.541$, $P = 0.003$) and in the alpha band for MF-L and PO-L ($F_{(3,48)} = 3.742$, $P = 0.017$) (Fig. 3).

Post hoc Bonferroni-corrected pairwise comparisons revealed a significant reduction in connectivity during trFOG compared with trVS in the following network pairs: MF-L and PO-L (broadband: $P = 0.004$; alpha: $P = 0.027$) and for FP-L and MF-R (theta: $P = 0.006$).

Additional significant differences were observed when comparing the FOG condition to VS, with lower connectivity in the following network pairs: TEM-L and INS (broadband: $P = 0.008$), TEM-L and PO-R (broadband: $P = 0.002$), TEM-R and MF-R (broadband: $P = 0.017$, beta: $P = 0.025$), PO-R and INS (broadband: $P = 0.024$), and SS-R and INS (delta: $P = 0.038$).

Discussion

Non-invasive markers with high temporal resolution are necessary to improve our understanding and identify early detection markers of FOG. To the best of our knowledge, this is the first study to investigate functional network connectivity changes associated with and preceding FOG using aEEG. Using a network-based approach, we identified altered FC during the transition to freezing (trFOG) compared with voluntary stopping (trVS), characterized by reduced between-network connectivity and increased beta-band modularity, reflecting greater functional segregation and local network alteration.

Increased Beta Segregation Within Networks

We observed increased modularity in the beta frequency band within the right prefrontal-parietal, middle frontal, parietal-occipital, visual, and bilateral insular networks during the transition phase preceding FOG, compared with transitions preceding VS. These networks are involved in cognitive control,[43] salience

detection,[44] and visuospatial processing,[45] all of which have been implicated in freezing.[46-48] The observed increase in modularity suggests a greater segregation within these systems, potentially reflecting disrupted inter-network communication and reduced coordination between networks. This may impair the brain's ability to flexibly shift between internal and external states, a function typically supported by the salience (attentional) and frontoparietal networks. In keeping with this, we found that the insular network, which plays a key role in switching between networks and orienting attention,[49] showed particularly strong beta segregation during trFOG. Increased network segregation and impaired switching during transitions may disrupt cognitive–motor integration thus compromising adaptive motor control and creating a vulnerable state that precipitates FOG.

Disrupted Between-Network Integration during trFOG

Reduced between-network connectivity during trFOG, particularly between middle frontal and parietal-occipital networks in the alpha and broad frequencies, may reflect impaired communication between regions involved in executive control and sensorimotor integration. Previous work in people with FOG highlighted the loss of automaticity of gait, and engagement of executive regions with a shift towards dependence on top-down regulation of adaptive gait control. Failure or vulnerability in the network supporting this top-down regulation may therefore lead to FOG. In patients with FOG, there is evidence of a compensatory shift from automatic to cognitively controlled gait even under normal walking conditions, reflected by increased engagement of frontoparietal regions during tasks requiring intentional movement cessation.[6,50] However, during trFOG, this compensatory mechanism appears to break down, resulting in reduced fronto-posterior connectivity and failure to maintain functional integration. In line with this, resting-state functional magnetic resonance imaging (fMRI) studies have highlighted decreased connectivity within executive and attention networks in PD patients with FOG.[51] Similarly, in our study, disrupted EEG connectivity across these canonical brain networks might signify a failure of engagement of compensatory mechanisms involving the executive and visual-attentional networks, with the latter underscoring the potential benefit of external visual cues as therapeutic aids. Consistent with this, we observed greater fronto-posterior connectivity during trVS compared with trFOG, which may reflect more coordinated engagement of networks involved in movement regulation during VS.

Associations Between FOG and Beta-Activity

Beta activity has previously been linked to freezing in PD, with studies reporting elevated beta synchrony in both cortical and subcortical regions during FOG episodes, particularly in the subthalamic nucleus and supplementary motor area.[52,53] These regions are involved in movement initiation and inhibition, and excessive beta activity is thought to interfere with adaptive motor control.[54] The prominence of beta abnormalities in our findings is consistent with this prior work. Physiologically, beta oscillations are associated with the maintenance of current motor or cognitive states.[55,56] While this may support stability, it can limit the flexibility required for dynamic processes like gait adaptation. Therefore, excessive segregation in the beta range may reflect a failure to shift or integrate across networks when flexible coordination is most needed.[13]

Neuromodulatory Contributions to Network Segregation

Cholinergic dysfunction has been previously linked to the occurrence of FOG[57] and may contribute to exaggerated local beta-band oscillatory synchrony.[58] Studies in rodent models have shown that basal forebrain cholinergic depletion increases cortical synchrony, and recent findings in PD patients further link basal forebrain cholinergic integrity with cognitive deficits.[59] Collectively, these findings suggest a plausible mechanism by which cholinergic dysfunction may drive elevated beta segregation, supporting the role of impaired cholinergic modulation in promoting less integrated and more rigid brain states.

In addition, the noradrenergic locus coeruleus, which also supports arousal and adaptive network reconfiguration, has also been implicated in FOG.[60-62] Impaired noradrenergic activity can promote over-segregation[63] and, along with cholinergic contributions, could also contribute to the observed over-segregation. These findings suggest that beta-band segregation in trFOG may reflect underlying neuromodulatory deficits involving both cholinergic and noradrenergic pathways, offering a potential avenue for reducing patient risk and preventing falls in PD.

Failure of Adaptive Network Coordination

No significant differences were observed in functional integration (as measured by participation coefficient and global efficiency) between the transition phase preceding FOG and that preceding VS, which might suggest that although local networks become more segregated during trFOG, there is no compensatory increase in communication between distant regions. This lack of compensatory global integration may also reflect noradrenergic deficits, which have been implicated in impaired network flexibility and reduced

capacity for dynamic reconfiguration during motor transitions in PD.

The fact that increases in modularity do not appear to facilitate the network-wide coordination needed for successful gait suggests that the brain may be attempting to compensate for impending gait freezing by reinforcing local processing. However, without adequate cross-network interaction, this strategy ultimately fails. Thus, in the absence of enhanced functional integration, increased beta segregation alone may represent a maladaptive neural signature that fails to support the dynamic coordination required for successful gait in PD.

Limitations and Future Directions

Although this study provides novel insights into network-level dysfunction in FOG, several limitations should be acknowledged. First, the use of a static functional connectivity approach with fixed 2-second epoch limits characterization of dynamic network state changes and may not consistently capture equivalent behavioral phases across trials given natural variability in gait movement. Second, the spatial resolution of aEEG constrains precise source localization, particularly in subcortical regions; multimodal high-density EEG with fMRI approaches may address this. Third, the relatively small sample size warrants validation in larger patient cohorts, including PD-nonFOG and healthy controls. Fourth, PLV is sensitive to zero-lag coupling arising from volume and source leakage. Although identical acquisition and preprocessing across conditions partially mitigated stationary leakage effects in within-subject contrasts, these effects cannot be fully eliminated, and estimation reliability may vary in participants with fewer episodes. Future work should therefore incorporate leakage-resistant connectivity metrics (eg, weighted phase lag index or imaginary PLV), and episode-level mixed-effects modeling to further assess robustness. Fifth, although both FOG and VS involve motor cessation, they likely engage overlapping but partially distinct neural systems, and most FOG occurred during turning, and so future work should investigate whether different FOG subtypes show distinct neural signatures. Finally, despite the use of LMMs to account for the imbalanced data and within-subject variability,[64,65] unbalanced across subjects number of data points may still influence estimate precision. Condition-level contrasts should therefore be interpreted cautiously, and episode-level hierarchical mixed-effect modeling represents an important direction for future work. Future work might also explore integrating these neurophysiological markers into real-time detection for adaptive therapeutic interventions.

Conclusions

In this study, we identified network- and frequency-specific differences in FC between the transition to FOG and VS. These changes could reflect the neurophysiological substrate underlying the pre-freezing period. The transition preceding FOG was characterized by increased beta-band segregation within local networks, including in frontoparietal, middle-frontal, parietal-occipital, visual and insular areas, alongside reduced fronto-posterior connectivity. These findings could be leveraged for development of targeted therapeutic intervention treatment incorporating biofeedback for PD patients with FOG.

Author Roles: (1) Research Project: A. Conception, B. Organization, C. Execution; (2) Statistical Analysis: A. Design, B. Execution, C. Review and Critique; (3) Manuscript Preparation: A. Writing of the First Draft, B. Review and Critique.
Y.T.: 1A, 1B, 1C, 2A, 2B, 3A.
E.M.: 1A, 1B, 2A, 2C, 3B.
S.B.: 3B.
S.J.G.L.: 1A, 1B, 2C, 3B.

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Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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Supporting Data

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