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Neural signatures of turn-induced freezing of gait in Parkinson's disease: insights from phase-specific cortico-subthalamic dynamics

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Freezing of gait (FOG), particularly during turning, is common in Parkinson's disease (PD), but its phase-specific neural mechanisms remain unclear. This study investigated cortico-subthalamic dynamics underlying turning-induced FOG and their modulation by dopaminergic medication. Local field potentials from primary motor cortex (M1), premotor cortex (PMC), bilateral subthalamic nucleus (STN), and kinematic data were recorded from 19 PD patients during timed up-and-go tasks in medication-off and medication-on states. Turns were segmented into four phases: TurnPre, TurnStart, TurnEnd, and TurnPost. During freezing episodes, alpha power in M1 and PMC significantly decreased in early turning phases. Enhanced PMC-STN coherence appeared during TurnPre in normal turning and TurnStart in freezing turning, with TurnPre alpha suppression and coherence predicting freezing duration. Medication normalized these abnormal oscillations and improved turning. These findings reveal phase-specific cortico-subthalamic disruptions in FOG and suggest novel electrophysiological biomarkers for intervention. Trial Registration: ChiCTR1900026601, registered October 15, 2019.

Freezing of gait (FOG), a debilitating motor phenomenon in Parkinson's disease (PD), manifests as abrupt, transient episodes of gait arrest despite preserved movement intention[1]. Affecting over 60% of advanced PD patients, FOG significantly elevates fall risk, restricts mobility, and impairs quality of life[2,3]. Among FOG subtypes, turning-induced freezing is particularly prevalent, constituting 48.4% of all episodes[4,5]. Although dopaminergic medications and deep brain stimulation (DBS) can offer relief from turn-related freezing, the benefits are often insufficient[6]. As turning-induced FOG imposes a disproportionate clinical burden and its etiology remains poorly understood, identifying its underlying neural mechanisms has become an urgent priority in advancing targeted interventions.

Turning maneuvers demand precise spatiotemporal integration of motor execution and cognitive control, governed by interconnected cortico-subcortical networks[7–12]. Among these circuits, the hyperdirect pathway connecting frontal cortical areas to the subthalamic nucleus(STN) plays a pivotal role in orchestrating movement selection, timing, and spatial scaling functions crucial for successful turning[13,14]. The STN plays a critical role, receiving input from the striatum via the indirect pathway and from frontal cortical regions via the hyperdirect pathway[15,16]. Concurrently, the primary motor cortex (M1) and premotor cortex (PMC) exhibit abnormal local field potential(LFP) dynamics, including exaggerated beta-band synchronization and reduced movement-related desynchronization in the alpha and beta bands[10,17,18], along with decreased blood oxygen level-dependent responses during freezing episodes, suggesting cortical-level disruptions in generating motor commands[19–21].

One previous study reported synchronized cortico-subthalamic activity during FOG[22], indicating that the cortex, STN, and their connections are all involved in the FOG process. However, prior investigations have largely failed to distinguish between freezing during straight-line walking and turning-induced freezing, and they have not examined the distinct phases of turning freeze—particularly across the pre-turn (TurnPre), onset (TurnStart), final (TurnEnd), and post-turn (TurnPost) segments, which remain poorly characterized.

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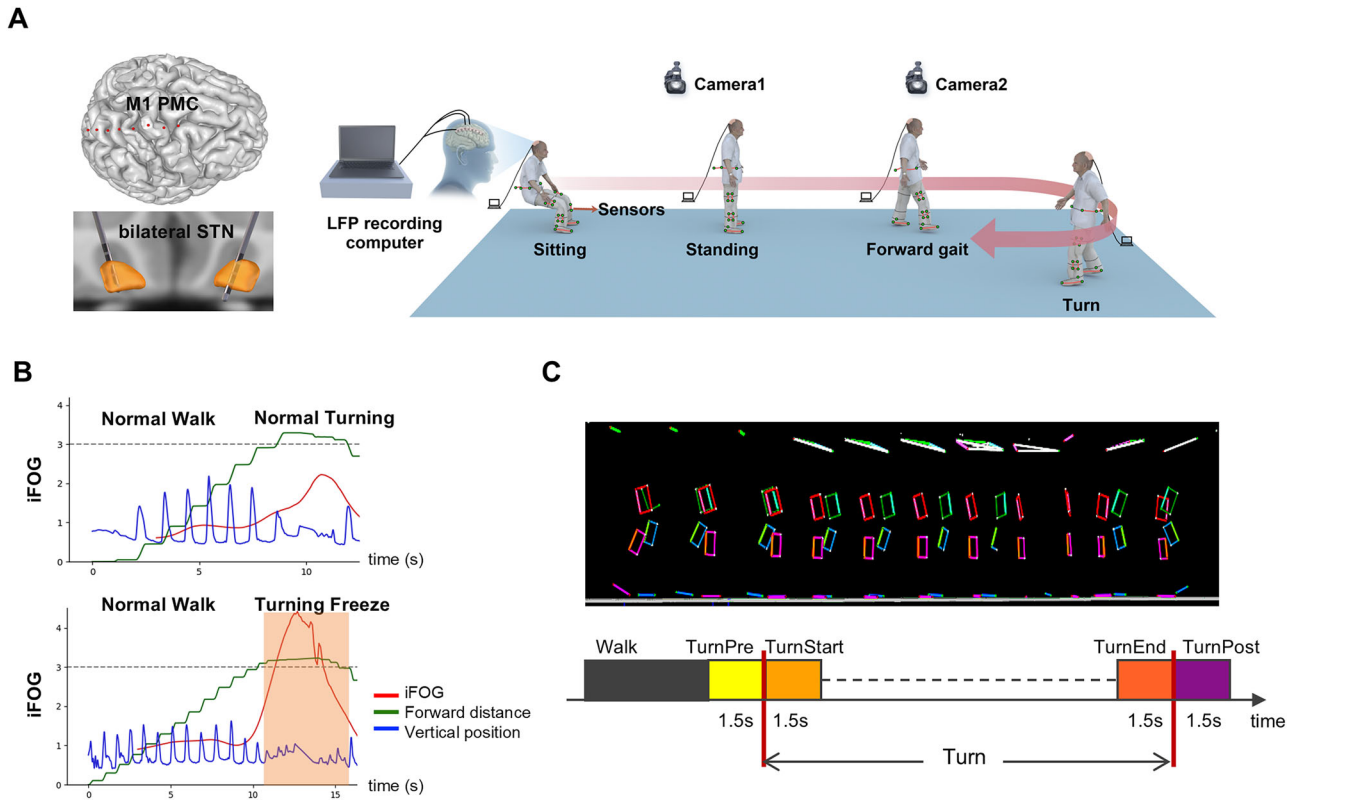

Fig. 1 | Experimental design, freezing quantification and phase-specific definitions of turning. A Representative illustration of electrode coverage and task setup. ECoG electrodes are positioned to cover the M1 and PMC regions, while DBS electrodes are placed in the bilateral STN. The schematic depicts participants performing the 5-meter Timed Up-and-Go (TUG) task, which includes straight walking, 180° right turns, and return walking under both Med-off and Med-on states. **B** Illustration of the Freezing Index (iFOG) within a trial. The blue lines indicate the vertical positions of the heel, the red line represents the iFOG, and the green line shows the forward distance. Turns are defined as freezing when the iFOG exceeds 3. **C** Overview of trial segmentation and body marker trajectories. The turning process is divided into four consecutive 1.5-second phases: TurnPre (before turn initiation), TurnStart (onset of rotation), TurnEnd (end of rotation), and TurnPost (after turn completion). A timeline beneath the schematic indicates the duration of each phase. Colored rectangles above represent the real-time 3D trajectories of reflective markers placed on the body, captured by the optoelectronic motion system. These visualizations illustrate changes in body orientation and limb dynamics during turning.

In this study, we aim to fill this gap by integrating electrocorticography (ECoG) recordings from the M1/PMC, bilateral STN-LFPs, and kinematic data from PD patients performing walking tasks under both medication-off (Med-off) and medication-on (Med-on) conditions (Fig. 1). Our objectives are twofold: (1) to identify the FOG-related phase-specific electrophysiological biomarkers that emerge during distinct turning segments, and (2) to evaluate how dopaminergic therapy modulates these biomarkers. By employing a dynamic, phase-resolved framework, this work aims to advance our understanding of FOG and inform novel strategies to mitigate turn-induced freezing.

Results

Patient characteristics

The study enrolled 19 participants (age: 65.2 ± 7.4 years; disease duration: 9.8 ± 3.1 years). Preoperative motor assessment revealed an Movement Disorder Society-Unified Parkinson's Disease Rating Scale part III (MDS-UPDRS III) score of 48.7 ± 12.4 in the Med-off state (≥12 h medication withdrawal), improving to 20.5 ± 8.0 in the Med-on state (1 h post-levodopa, 49.9% improvement). The mean freezing of gait questionnaire (FOGQ) score was 17.4 ± 4.2. Demographic and clinical characteristics are detailed in Table 1.

Turning performance and electrophysiological recording

Two independent video raters (trained researchers) independently reviewed synchronized video recordings to classify freezing episodes during turning tasks, achieving an inter-rater reliability of 90.2% (consensus on 275 trials). In the Med-off state, the trials included 87 turns with freezing episodes (62.6%, total duration: 1214.9 s) and 52 turns with no freezing episodes (37.4%, total duration: 216.7 s). In the Med-on state, performance improved, with 15 turns with freezing (10.7%, total duration: 582.2 s) and 125 turns without freezing (89.3%, total duration: 216.7 s). Electrophysiological data were obtained from the bilateral STN in all participants, from the PMC in 18 patients, and from the M1 in 17 patients.

Phase-specific neural oscillations during turning

In the Med-off state, the PSD analysis of the PMC, M1, and bilateral STN revealed phase-specific modulation of alpha and beta bands across five turning phases: Walk, TurnPre, TurnStart, TurnEnd, and TurnPost (Fig. 2A). All spectral values were normalized to the walking baseline within each condition, using the mean spectral power during the artifact-free straight-walking period of the same trial. This intra-trial normalization strategy helped minimize baseline variability across trials. Comparisons were conducted between each turning phase and the Walk phase within the same condition. During normal turning, a significant decrease in the alpha band power was observed in the STN during the TurnStart phase ($p = 0.012$). Additionally, there was a decrease in the alpha band power ($p < 0.001$) and an increase in the beta band power in the M1 ($p = 0.013$) during the TurnEnd phase (Fig. 2B). In the case of turning freeze, both the PMC and M1 showed significant suppression in the alpha band throughout the entire turning process, including the TurnPre ($p = 0.0042$ for PMC and $p < 0.001$ for M1), TurnStart ($p = 0.027$ for PMC and $p = 0.048$ for M1) and TurnEnd ($p = 0.0072$ for PMC and $p = 0.0016$ for M1) phases (Fig. 2C).

Table 1 | Demographics of the subjects

ID	Age/ Sex	DD (years)	LEDD (mg)	UPDRSIII		FOGQ	State[3]
				Moff[1]	Mon[2]		
sub01	72/M	10	675	47	28	10	off/on
sub02	60/F	7	750	47	24	20	off
sub03	52/F	15	1052	51	9	21	off
sub04	57/F	5	375	49	24	14	off
sub05	66/F	10	513	61	22	21	off/on
sub06	53/M	12	1100	79	25	24	off
sub07	70/M	12	688	70	37	17	on/off
sub08	73/F	9	1439	51	27	20	off/on
sub09	67/F	6	500	52	30	22	off/on
sub10	59/F	9	700	46	21	16	on/off
sub11	78/M	5	550	58	24	18	off
sub12	76/M	8	1351	41	11	13	off/on
sub13	66/F	15	669	55	8	13	on/off
sub14	61/M	7	1150	37	18	22	on/off
sub15	67/M	10	913	42	20	16	on/off
sub16	66/F	15	925	39	20	20	off/on
sub17	67/F	9	1000	27	5	15	off/on
sub18	71/F	11	1212	36	19	19	on/off
sub19	58/M	11	1263	37	18	9	on/off

DD disease duration, LEDD levodopa equivalent daily dose, *FOGQ* freezing of gait questionnaire. [1]MDS-UPDRS III off-medication score. [2]MDS-UPDRS III on-medication score. [3]Medication state order in the study. off: Medication 'off' state; on: Medication 'on' state.

Cortico-Subthalamic network dynamics during turning

To assess cortico-subthalamic network changes during turning in the Med-off state, we compared alpha and beta coherence between the PMC and STN, as well as between the M1 and STN, across the walking and turning phases. During normal turning, significantly higher PMC-STN alpha coherence was observed in the TurnPre phase ($p = 0.0045$, Fig. 3A). In contrast, during turning freeze, there was an increase in PMC-STN alpha coherence ($p = 0.048$) and a decrease in M1-STN alpha coherence ($p = 0.043$) during the TurnStart phase (Fig. 3B). In terms of beta coherence between the PMC-STN and M1-STN, no significant differences were found between the turning phases and walking in any of the regions analyzed.

Electrophysiological predictors of freezing severity

Building upon our previous findings of significant differences in local brain activity and inter-regional connectivity during turning freeze compared to walking, we sought to further investigate the relationship between electrophysiological changes and freezing behavior during turning in the Med-off state. We focused on indicators that showed significant changes during the TurnPre and TurnStart phases, specifically the alpha power in the PMC and M1, as well as the alpha coherence between the PMC-STN and M1-STN. For each trial, we calculated the percentage change of these electrophysiological indicators during the specific phase relative to the Walk phase. We then applied a linear mixed-effects model to determine whether these changes could predict the percentage of time spent freezing during the turning in that trial. Our analysis revealed that the decreases in the PMC alpha power ($\beta = 0.154$, $p = 0.035$) (Fig. 4A) and increases in PMC-STN alpha coherence ($\beta = 0.205$, $p = 0.006$) (Fig. 4B) during the TurnPre phase, but not the TurnStart phase, significantly predicted the freezing duration within the trial. In contrast, changes in the M1 alpha power and M1-STN alpha coherence in the TurnPre and TurnStart phase did not show significant predictive value (Fig. 4C, D), indicating a more localized role of PMC and its connectivity in freezing behavior.

Medication effects on neural dynamics and behavior

Medication significantly improved turning performance in patients, as evidenced by a substantial reduction in the percentage of time spent freezing ($p = 0.0033$) and a significant shortening of turning duration ($p < 0.001$, Fig. 5A). Due to the limited number of freezing trials under the Med-on condition, the analysis then focused solely on normal turning. Firstly, medication did not directly affect alpha power in the PMC and M1 during normal turning (Fig. 5B, Supplementary Fig. A), thereby reversing the abnormal electrophysiological indices observed under Med-off conditions. For PMC alpha, unlike in the Med-off condition during turn freezing, where significant reductions were observed in the TurnPre, TurnStart, and TurnEnd phases relative to walking (Fig. 2), no differences between the turning phases and walking were detected under Med-on conditions (Fig. 5C), mirroring the pattern seen in normal turning. With regard to PMC–STN alpha coherence, under Med-on conditions no significant differences between the turning phases and walking were found (Fig. 5D), whereas under Med-off conditions, increased coherence relative to walking was observed in some turning phases for both turn freezing and normal turning (Fig. 3). Additionally, under Med-on conditions, M1 alpha power showed a significant decrease during the TurnPre phase, and STN alpha power decreased during both the TurnPre and TurnStart phases (Supplementary Fig. C), while M1–STN alpha coherence did not differ significantly between the turning phases and walking (Supplementary Fig. B). These findings suggest that the pattern observed under Med-on conditions approximates that of normal turning under the Med-off state.

Discussion

This is the first study to investigate multi-site electrophysiological activity in the cortico-subthalamic network in freely walking PD patients to explore the mechanisms underlying turning freeze. We found that turning freeze is characterized by phase-specific biomarkers, which include both local activity changes and alterations in inter-regional coherence. Specifically, during the turning freeze, alpha power in the PMC and M1 significantly decreased across the TurnPre, TurnStart, and TurnEnd phases, with the magnitude of PMC alpha reduction in the TurnPre phase being predictive of freezing duration. Moreover, in turning freeze, there was an increase in PMC-STN alpha coherence and a decoupling of M1-STN alpha coherence during the TurnStart phase. Importantly, increases in the PMC-STN alpha coherence during the TurnPre phase significantly predicted the freezing duration within the trial. Pharmacological intervention significantly improved turning performance, partially rectifying the local abnormalities in the PMC and M1 and restoring the altered coherence patterns between the PMC-STN and M1-STN, thereby highlighting these phase-specific features as potential targets for intervention.

Turning is a complex motor task that requires precise spatiotemporal coordination and cognitive control, engaging distinct neural processes during its execution. One previous studies on turning have relied on fMRI, which necessitates the use of virtual reality simulations or stepping paradigms to mimic turning and walking[23]. These approaches are limited by the constraints of neuroimaging techniques in assessing naturalistic gait and by spatial resolution. In contrast, non-invasive EEG and fNIRS studies, though conducted during real walking, have typically analyzed turning as a single continuous segment, thereby hindering a comprehensive understanding of cortico-subthalamic interactions[10,13,24]. To our knowledge, the only previous investigation of phase-specific changes focused solely on freezing episodes[22], and no study has specifically examined phase-specific dynamics in turning-induced freezing. By subdividing the turning maneuver into discrete phases (TurnPre, TurnStart, TurnEnd, and TurnPost), we were able to capture the dynamic evolution of neural activity and identify specific time windows in which abnormal electrophysiological patterns emerge. The TurnPre phase, in particular, may reflect anticipatory processes critical for movement planning. This segmentation not only enhances the sensitivity of our analyses to detect subtle abnormalities in both local cortical activity and cortico-subthalamic connectivity but also provides a structured framework to pinpoint the temporal onset of turning-induced FOG episodes. Ultimately,

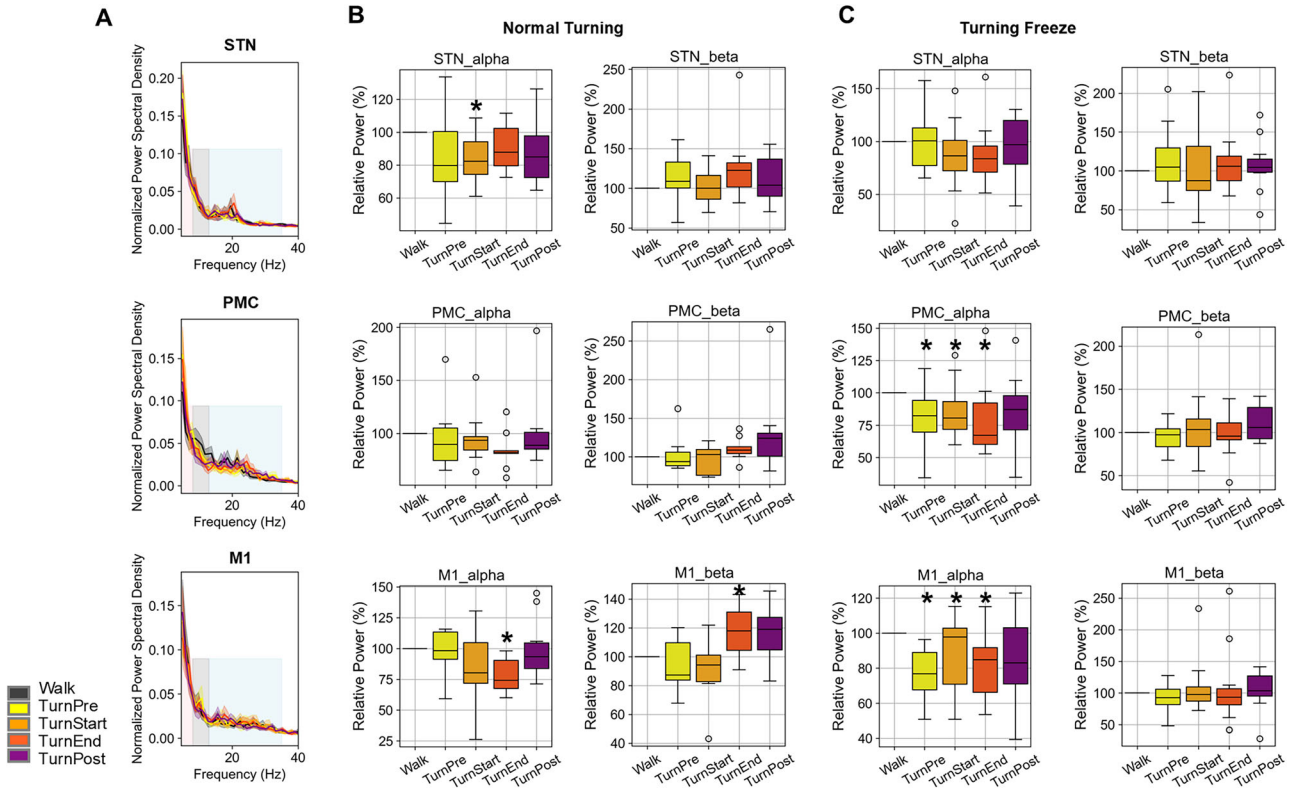

Fig. 2 | Phase-Specific Neural Oscillations During Turning. A Normalized power spectral density (PSD) for the STN, PMC, and M1 regions during the Walk, TurnPre, TurnStart, TurnEnd, and TurnPost phases. **B** Relative alpha and beta power during normal turning, normalized to the Walk condition. A significant reduction in STN alpha power was observed during TurnStart, while M1 alpha power decreased and M1 beta power increased during TurnEnd. No significant differences in other regions or frequency bands were observed compared to Walk. **C** Relative alpha and beta power during turning freeze, normalized to the Walk condition. Alpha power significantly decreases in the PMC and M1 during the TurnPre, TurnStart, and TurnEnd phases, with recovery observed in the TurnPost phase. All data shown were recorded in the Med-off state.

such a detailed temporal resolution is instrumental in designing targeted interventions aimed at mitigating freezing episodes.

Our study identifies a significant reduction in alpha power in the PMC and M1 during the TurnPre, TurnStart, and TurnEnd phases in turning freeze, a phenomenon not observed in normal turning. Notably, the magnitude of alpha suppression in the PMC during the TurnPre phase predicted the duration of freezing episodes, suggesting its potential as a neurophysiological marker of FOG severity. Alpha and beta desynchronization, or event-related desynchronization (ERD), is classically associated with motor preparation and cortical disinhibition in transitions from rest to movement[10,12,25,26]. In PD, and particularly in patients with FOG, this process is often attenuated, delayed, or spatially disorganized[17,18]. While prior ERD studies typically used rest as baseline, our study analyzed spectral dynamics relative to steady-state walking. Though this limits direct comparison with rest-to-movement transitions, it enables the detection of dynamic neural adaptations when switching from automated gait to cognitively demanding turning. Our finding that alpha power decreases during TurnPre extends this framework, suggesting that freezing may result from impaired anticipatory motor control rather than solely execution failure. The altered activity observed in the PMC during turning in PD patients is consistent with findings from previous fMRI studies[23,27]. These results align with fNIRS-based studies, which also report altered PMC and M1 activity during tasks that provoke freezing, such as turning and passing through doorways. Such changes likely reflect compensatory cortical recruitment in response to impaired automaticity in motor preparation[10,23,28]. However, our study offers a novel contribution by demonstrating that alpha power reduction in both the PMC and M1 occurs not only during the motor execution phase (TurnStart) but also during the preparatory phase (TurnPre), prior to the onset of freezing. This reduction in alpha power during TurnPre suggests that it may serve as an early indicator of motor preparation dysfunction, providing a window into the pathophysiology of freezing. Importantly, we found that the extent of the PMC alpha reduction during TurnPre is predictive of the duration of freezing, highlighting its potential as a biomarker for early detection of freezing episodes. Taken together, these findings reinforce the idea that freezing of gait involves not only execution deficits but also dysfunctional motor preparation. Alpha suppression in the PMC, especially during the preparatory phase, may reflect impaired feedforward control, and its association with freezing duration highlights its potential as an early neural marker of freezing vulnerability.

In addition to these local cortical changes, our findings reveal significant alterations in cortico-subthalamic network dynamics during freezing episodes. Specifically, we observed a decoupling of M1-STN alpha coherence during the TurnStart phase. These results suggest that the functional connectivity between cortical regions and the STN is disrupted during FOG, likely contributing to the motor deficits observed during turning. The STN, as a critical node in the cortico-basal ganglia-thalamo-cortical loop, is crucial for regulating movement execution, particularly during tasks involving high cognitive load or attention-demanding situations like turning[22,29,30]. We observed an increase in PMC-STN alpha coherence during TurnPre in normal turning, suggesting the role of PMC-STN connectivity in the initiation of the turning task. The increased coherence between the PMC and STN may reflect a compensatory mechanism to maintain motor control in the face of disrupted cortical signals; however, this abnormal coupling could also contribute to the rigidity and motor impairments characteristic of FOG[31,32]. On the other hand, the decoupling of M1-STN alpha coherence suggests that communication between the motor cortex and STN is impaired during freezing episodes, which might explain the transient loss of motor control seen in FOG[22]. This

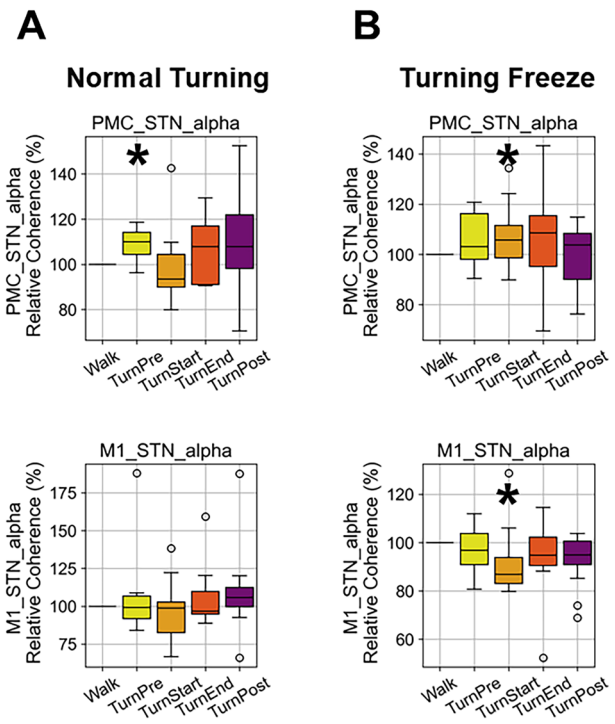

Fig. 3 | Phase-specific cortico-subthalamic network dynamics during turning. **A** Normal Turning: During the TurnPre phase, physiological motor preparation was characterized by a significant increase in PMC-STN alpha coherence. **B** Freezing of Turning: A delayed and exaggerated increase in PMC-STN alpha coherence and concurrent decoupling of M1-STN alpha coherence was observed during the TurnStart phase. All data shown were recorded in the Med-off state.

disruption in network connectivity is consistent with the notion that FOG is a multifactorial disorder involving both motor and cognitive dysfunctions[10]. Our findings also complement studies demonstrating that alterations in cortico-subthalamic communication, particularly in the beta band, are associated with FOG and other motor symptoms in PD[33–35]. Interestingly, while beta oscillations are often linked to motor inhibition and movement suppression, our study highlights that the alpha band may also play a crucial role in the modulation of cortico-subthalamic interactions during complex motor tasks such as turning.

Our study demonstrates that dopaminergic medication is associated with a substantial reduction in freezing time and turning duration, as well as a normalization of the altered neural oscillatory patterns in the PMC and M1. Specifically, medication restored the abnormal PMC-STN and M1-STN alpha coherence patterns observed during turning freeze, suggesting that dopaminergic treatment may help to re-establish functional cortico-subthalamic connectivity during turning. These findings are consistent with a growing body of literature indicating that dopaminergic therapy modulates both cortical and subcortical oscillatory activity in PD patients, thereby improving motor performance[12,36–38]. Moreover, the restoration of normal cortico-subthalamic connectivity in response to medication aligns with previous studies showing that dopaminergic treatment can reduce pathological synchronization between cortical and subthalamic regions, leading to improved motor function[39,40]. However, although medication partially corrects the abnormal alpha activity during turning freeze-shifting it toward the patterns seen in normal turning under the Med-off condition-the neural patterns do not completely normalize. This observation mirrors our clinical findings that while dopaminergic therapy can mitigate freezing episodes, it does not fully resolve them[6]. Such results underscore the need for more targeted treatments, such as neuromodulation approaches, which address the complex neural mechanisms contributing to FOG beyond dopaminergic modulation alone. Overall, our findings highlight the dynamic nature of FOG in the context of dopaminergic therapy. While medication can ameliorate certain abnormalities in neural oscillations and

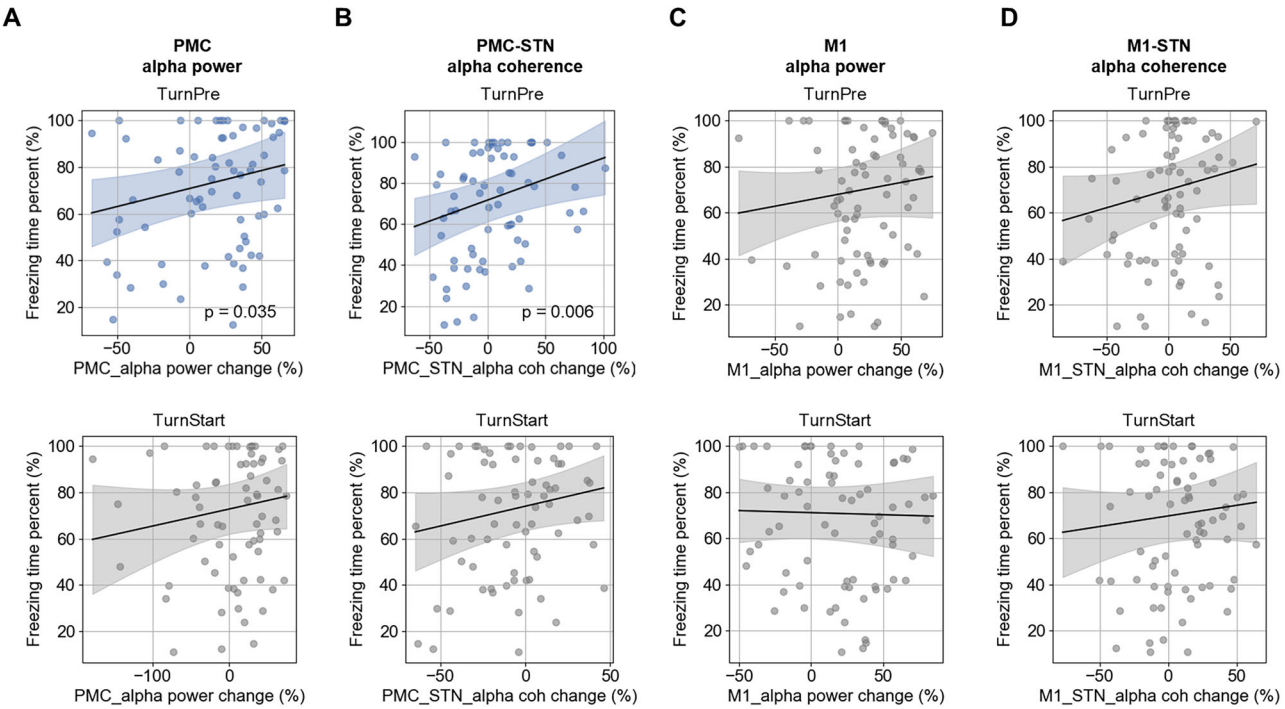

Fig. 4 | Electrophysiological predictors of freezing severity. **A** PMC alpha power reduction during the TurnPre phase (but not during the TurnStart phase) relative to walking positively correlated with freezing duration ($\beta = 0.154$, $p = 0.035$) **B** Increased PMC-STN alpha coherence during the TurnPre phase (but not during the TurnStart phase) relative to walking predicted shorter freezing episodes ($\beta = 0.205$, $p = 0.006$). **C** M1 alpha power changes and **D** M1-STN coherence changes during the TurnPre or TurnStart phase relative to walking do not significantly correlate with freezing duration. All data shown were recorded in the Med-off state.

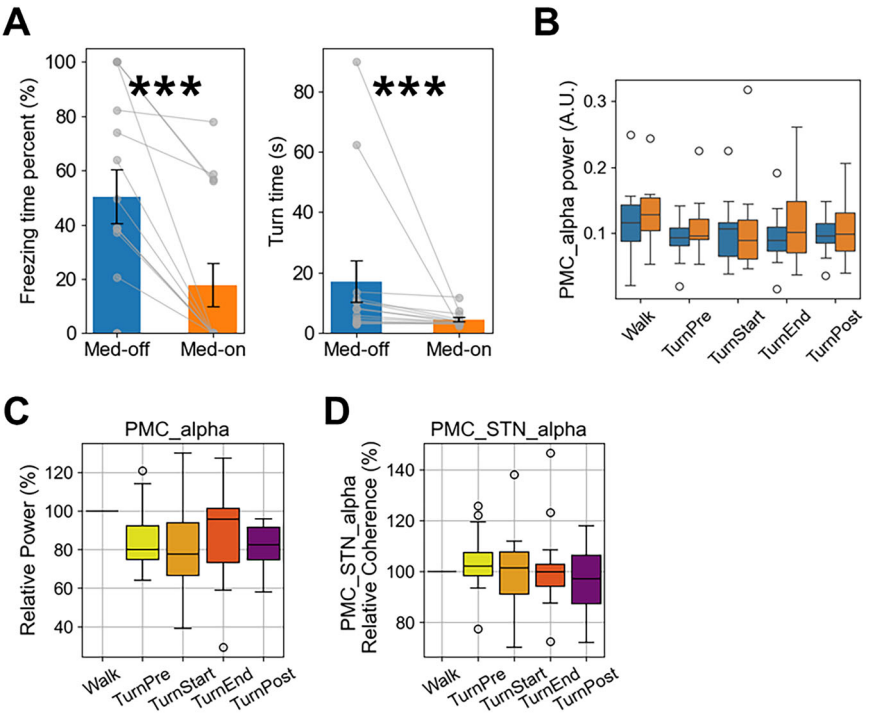

Fig. 5 | Medication effects on neural dynamics and behavior. A Medication significantly reduces freezing time and turning duration. **B** No significant differences were observed in PMC alpha power across walking and turning phases under both the Med-on and Med-off conditions. Under the Med-on condition, **C** PMC alpha power and **D** PMC-STN alpha coherence during each turning phase did not differ from that observed during walking.

improve turning performance, a comprehensive understanding of the phase-specific neural dysfunctions associated with FOG is essential for the development of more effective therapeutic strategies in the future.

Several limitations of our study should be acknowledged. First, the sample size for comparisons between freezing and normal turning was relatively small, which may limit the generalization of our findings. Future studies with larger cohorts are needed to validate and extend these results. Second, variability in electrode coverage among patients may have influenced our ability to detect significant changes in certain brain regions or networks. Standardizing electrode placement or utilizing advanced neuroimaging techniques could enhance the consistency and reliability of data in future research. Third, we adopted a block-based segmentation strategy to define discrete turning phases. This design, consistent with prior work by Pozzi et al.[22], allowed structured phase-wise comparisons but may have limited our ability to detect fine-grained transitions such as spectral shifts immediately preceding freezing onset. Lastly, our focus was limited to cortico-subthalamic dynamics. Other neuromodulatory systems, including the noradrenergic system and locus coeruleus, are known to contribute to freezing pathophysiology via arousal and cognitive control mechanisms[41,42], which were beyond the scope of the current setup.

Our study demonstrates that turning-induced freezing in PD is marked by phase-specific electrophysiological abnormalities. By segmenting the turning maneuver, we found a significant reduction in alpha power in the premotor cortex, with decreased alpha activity during the preparatory phase predicting freezing duration. Additionally, increased alpha coherence between the premotor cortex and subthalamic nucleus during this phase also predicted freezing duration. Although dopaminergic medication partially normalized these aberrant patterns, residual abnormalities remain, underscoring the need for targeted neuromodulation.

Methods

Participants and ethical approval

Nineteen patients with advanced PD and clinically significant FOG were prospectively recruited from Beijing Tiantan Hospital prior to scheduled bilateral STN-DBS surgery. Inclusion criteria were detailed in our previous work[12,19]. The study protocol complied with the Declaration of Helsinki, received approval from the Institutional Review Board of Beijing Tiantan Hospital (KY 2018-008-01). This sub-study was conducted during the externalized recording period prior to randomization and intervention in the main registered clinical trial (ChiCTR1900026601, registered on October 15, 2019). All participants provided written informed consent.

Clinical and neuropsychological assessments

Preoperative evaluations included comprehensive motor, cognitive, and neuroimaging assessments, as previously described[12,19]. Motor symptoms were quantified using the MDS-UPDRS III in both the Med-off (≥12 h withdrawal) and Med-on (1 h post-levodopa) states. Freezing severity was assessed using the FOGQ (score range: 0–24, with higher scores indicating more severe freezing) and video-annotated freezing episodes during TUG tasks.

Surgical procedures and electrophysiological setup

Bilateral four-contact STN-DBS electrodes (L301, PINS Medical, Beijing, China) were implanted stereotactically, guided by intraoperative microelectrode recordings. An 8-contact subdural strip electrode (8 mm diameter, 80 mm exposure, 10 mm intercontact spacing) was placed through the DBS burr hole to cover the M1 and PMC. STN electrode localization was performed by co-registering postoperative CT (0.625 mm) with preoperative T1-weighted MRI, followed by normalization to MNI space using Lead-DBS[43]. Electrode trajectories were automatically reconstructed and manually refined. For subdural electrodes, cortical parcellation was conducted on preoperative T1-weighted MRI using the FreeSurfer software to delineate motor-related cortical regions[44]. Electrode positions were projected onto the cortical surface and assigned to the primary motor cortex (M1) or premotor cortex (PMC) according to their anatomical locations. Externalized electrode extensions enabled for the synchronized recording of STN local field potentials (LFPs) and electrocorticography (ECoG) from the M1 and PMC.

Experimental design and data acquisition

Following surgical implantation, participants underwent experimental testing to assess neural and kinematic responses during turning tasks. All recordings were performed with DBS turned off to avoid stimulation-induced artifacts in STN-LFP signals. These experiments were conducted 3–5 days postoperatively in a gait laboratory. Participants performed

5-meter TUG tasks involving straight walking, 180°right turns, and return walking under both the Med-off and Med-on states, with task order randomization (except for sub02, sub03, sub04, sub06, and sub11, who only completed the Med-off condition). During these trials, participants walked at their natural pace or engaged in dual-task conditions, such as serial subtraction, object naming, or coin transfer from one hand to another. Kinematic data were captured at 200 Hz using a CODA optoelectronic system (Charnwood Dynamics Ltd, UK), which tracked the 3D joint trajectories of key body markers (Fig. 1A). Synchronized video recordings were used to annotate freezing episodes, with any inter-rater discrepancies being resolved through consensus.

Freezing quantification and phase-specific definitions during turning

Freezing episodes were quantified using a validated freezing index (iFOG), which is defined as the power ratio of the 3–8 Hz (freezing band) to the 0–3 Hz (locomotion band) within 6-second sliding windows, with a step size of 0.1 seconds. A threshold iFOG value greater than 3 was used to identify freezing events, as detailed in our previous studies (Fig. 1B)[12,19]. The freezing percentage was calculated as the proportion of the freezing duration relative to the total duration of the turn. The turning process was segmented into four phases: TurnPre (1.5 seconds before turn initiation), TurnStart (the first 1.5 seconds of rotation), TurnEnd (the last 1.5 seconds of stabilization), and TurnPost (1.5 seconds after completing the turn) (Fig. 1C).

Electrophysiological signal acquisition and processing

STN LFPs and ECoG signals were recorded at a sampling rate of 2000 Hz using a JE-212 amplifier (Nihon Kohden, Japan), synchronized with kinematic data via digital triggers. Preprocessing was performed offline using MNE-Python. Raw signals were bandpass filtered (1–80 Hz, Hamming-windowed FIR, transition bandwidth: 1 Hz) and notch filtered at 50 Hz (±1 Hz) to suppress line noise. Bipolar referencing between adjacent contacts was applied to reduce common-mode and volume-conducted artifacts. Data were segmented into 1.5-second non-overlapping windows corresponding to each turning phase (TurnPre, TurnStart, TurnEnd, and TurnPost) and straight-walking periods. Artifact screening was conducted by amplitude-based thresholding (±300 μV for ECoG, ±100 μV for STN-LFP) to detect transient movement or physiological artifacts. Electrode localization was performed using LeadDBS for STN contacts[43] and FreeSurfer-based cortical parcellation for M1 and PMC electrodes[44] (Fig. 1A). Turning phases were segmented based on 3D kinematic trajectories captured from body markers, which allowed precise identification of movement transitions across the TurnPre, TurnStart, TurnEnd, and TurnPost phases (Fig. 1C). A representative example of artifact-free M1, PMC, and STN signals during walking is presented in Supplementary Fig. S1.

Power spectrum density analysis

To analyze local oscillatory dynamics during turning compared to walking, we employed the multitaper method to compute the power spectral density (PSD) with the psd_array_multitaper function from the MNE-Python library. PSD calculations focused on two key frequency bands: the alpha band (8–13 Hz) and the beta band (13–35 Hz), given their crucial roles in motor control and their particular relevance to PD. To emphasize frequency-specific modulations and minimize the global signal fluctuations, we normalized power by dividing the band-specific power by the total power within the 1–80 Hz range for each segment. For baseline normalization, only artifact-free, non-freezing straight-walking segments within each trial were used. These segments typically lasted at least 8 seconds, covering a minimum of 7 gait cycles. They were divided into non-overlapping 1.5-second windows, consistent with the segmentation used for turning phases. PSD and cortico-subthalamic coherence were computed for each window and then averaged across all walking windows within the trial to generate the baseline. This within-trial, multi-cycle approach reduced inter-trial variability and baseline drift, while minimizing the influence of gait-phase-specific fluctuations.

Cortico-subthalamic coherence analysis

Functional connectivity between cortical regions (M1, PMC) and the bilateral STN was assessed by coherence analysis using the spectral_connectivity_time function from the MNE-Python connectivity package. Coherence was computed employing the continuous wavelet transform with Morlet wavelets. The frequencies of interest were specified, and the number of cycles for each frequency was set to n_cycles = freqs/2 to balance time and frequency resolution across the spectrum. For subsequent analysis, we focused on the real part of the coherence values to examine the magnitude of coupling between signals. The analyses specifically targeted phase-specific changes in the alpha and beta bands in comparison to walking.

Data normalization and comparison

To isolate turn-related neural modulations, the PSD and cortico-subthalamic coherence values during the turning phases (TurnPre, TurnStart, TurnEnd, and TurnPost) were normalized to the walking baseline within each trial:

$$\text{Normalized Value}\,(\%) = \frac{\text{Value}_{\text{Phase}}}{\text{Value}_{\text{Walk}}} \times 100 \quad (1)$$

This intra-trial normalization minimized inter-trial and inter-subject variability, thereby enhancing the sensitivity to detect phase-specific neural dynamics.

For condition-specific comparisons (turning freeze vs. normal turning; Med-off vs. Med-on), we analyzed the raw (non-normalized) values using paired tests. This approach preserved the absolute signal differences between conditions while controlling for inter-subject variability through within-participant contrasts.

Statistical analysis

For data distributions that were non-normal, as determined by the Shapiro-Wilk test, we utilized nonparametric methods for analysis. Phase-specific differences relative to walking were assessed using Wilcoxon signed-rank tests, with Bonferroni correction for multiple comparisons (TurnPre, TurnStart, TurnEnd, and TurnPost vs. Walk). A linear mixed-effects model was employed to assess whether phase-specific changes in alpha power (M1, PMC) and alpha coherence (M1-STN, PMC-STN) predicted the duration of freezing. Fixed effects included normalized electrophysiological changes during TurnPre and TurnStart, while random intercepts accounted for inter-subject variability. The fixed effects were defined as the normalized electrophysiological changes, calculated by:

$$\text{Percentage Change}\,(\%) = \frac{\text{Value}_{\text{Walk}} - \text{Value}_{\text{TurnPre or TurnStart}}}{\text{Value}_{\text{Walk}}} \times 100 \quad (2)$$

Random intercepts at the participant level were included in the model to account for individual variability. To assess neural-behavioral relationships influenced by medication-induced (Med-off vs. Med-on), Pearson correlations analyses were conducted. A significance threshold of $P < 0.05$ (two-tailed) was applied for these analyses. All analyses were conducted using Python 3.

Data availability

The data used to support this study's findings are available from the corresponding author upon reasonable request.

Code availability

The codes used to support this study's findings are available from the corresponding author upon reasonable request.

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Competing interests

The authors declare no competing interests.

Additional information

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