BRAIN ACTIVITY DURING MOVEMENT STOPPING: PARKINSON'S DISEASE VS. HEALTHY CONTROL PARTICIPANTS

by

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A THESIS

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This study explores the neural dynamics of inhibitory control in Parkinson's disease (PD) through the analysis of event-related potentials (ERPs) during a Continuous Movement Stop Task (CMST). Unlike classical inhibition paradigms such as the oddball or Go/No-Go tasks, which focus on internal suppression of responses that are never executed, the CMST required participants to initiate a motor action and then stop it in response to either a planned or unplanned cue—offering a more valid examination of planned and unplanned inhibition.

Electroencephalography (EEG) was used to measure key ERP components—P2, N2, and P3—across healthy controls (HC) and PD participants both on (PD ON) and off (PD OFF) dopamine medication. Notably, the P300 component was not visually detectable. One possible explanation would be that P300 may reflect internal movement suppression rather than externally cued action stopping. The N2, commonly associated with early-stage inhibition and conflict monitoring, showed high variability and was not significantly different across groups, suggesting its lack of participation in the halt of ongoing motor activity. In contrast, the P2 component was robust and revealed significant group differences, particularly between HC and PD ON participants, indicating its potential role in early attentional and perceptual processing during motor inhibition and sensitivity to dopamine concentrations.

These findings highlight the distinct neural mechanisms involved in stopping ongoing movement and offer novel insights into how inhibitory processes are altered in Parkinson's disease. Compared to traditional interpretations of ERP components from suppressed-movement paradigms, our results suggest that components like P2 may be a more salient marker of real-time motor inhibition.

Acknowledgements

I dedicate this work to Dr. Nicole Swann, my principal investigator, whose unwavering support and thoughtful mentorship have shaped my growth as a researcher. Your belief in me has made this journey possible.

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To my parents, Sonia Sanchez and Gabriel Muñiz—thank you for your endless love, encouragement, and strength. Though far away, your support has grounded me and given me the courage to move forward.

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Cinthia Muñiz

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PARKINSON'S DISEASE (PD)

Brief History

Parkinson's Disease (PD) was first comprehensively described by James Parkinson in his 1817 essay, "Essay on the Shaking Palsy" (Goetz, 2011). Parkinson astutely observed core motor symptoms such as rest tremor and festination, identifying a distinct neurological disorder. While early descriptions focused primarily on motor manifestations, the understanding of PD evolved significantly with advancements in neurology. The 19th and early 20th centuries saw formal classification differentiating PD from other neurodegenerative conditions (Obeso et al., 2017). The discovery of progressive degeneration of dopaminergic neurons in the substantia nigra and the presence of Lewy bodies (abnormal α-synuclein aggregates) cemented PD's definition as a synucleinopathy (Uhl et al., 1984; Spillantini et al., 1997). By the late 20th century, research expanded to recognize non-motor symptoms—including cognitive deficits in attention, executive function, and memory—now understood as integral to the disease spectrum due to reduced dopamine concentrations (Davis & Racette, 2016; Aarsland et al., 2021). This broader perspective is crucial for understanding the cognitive and inhibitory challenges faced by individuals with PD, which are central to the current project.

Epidemiology

Parkinson's Disease is a progressive neurodegenerative disorder characterized by the loss of dopaminergic neurons in the substantia nigra and the presence of alpha-synuclein-containing Lewy body aggregates (Armstrong & Okun, 2020). Its etiology is complex, involving a combination of genetic predispositions and environmental factors. Age is the most significant risk factor, with incidence rising sharply after 60 (Kalia & Lang, 2015).

A consistent epidemiological finding is that men have nearly twice the risk of developing PD compared to women, potentially due to neuroprotective effects of estrogen, though hormonal influences alone may not fully explain this disparity (Kalia & Lang, 2015). Geographic and racial variations in incidence rates are also observed, with North America and parts of Europe reporting higher rates compared to many Asian and African regions (Pringsheim et al., 2014). Genetic contributions are evident, particularly in familial forms linked to mutations in genes such as SNCA, LRRK2, and PARK2 (Kalia & Lang, 2015). Environmental factors, notably exposure to pesticides and herbicides, have also been implicated, as evidenced by cases of MPTP-induced parkinsonism (Ball et al., 2019). Given global aging populations, the prevalence of PD is projected to more than double by 2040 (Pringsheim et al., 2014), underscoring the growing public health challenge.

Pathophysiology

The neuronal degeneration in PD primarily affects pathways essential for movement initiation and regulation, disrupting the basal ganglia-thalamocortical circuits vital for purposeful motion. This degeneration leads to a functional imbalance: the direct pathway, which facilitates movement, is weakened by insufficient dopamine, while the indirect pathway, which suppresses movement, becomes overactive due to diminished dopaminergic inhibition (Albin et al., 1989). This results in excessive inhibitory output from the globus pallidus interna (GPi) to the thalamus, thereby reducing thalamic drive to the motor cortex. This explains hallmark motor symptoms like bradykinesia and akinesia (Hamani & Lozano, 2003).

The motor symptoms of PD—bradykinesia, rigidity, postural instability, and tremor—each stem from specific disruptions within these circuits. Bradykinesia is linked to weakened direct pathway activity, leading to hyper-inhibition of the thalamus and reduced cortical motor

output (Albin et al., 1989). Rigidity arises from an overactive indirect pathway, causing continuous contraction of opposing muscle groups (Berardelli et al., 1983; DeLong & Wichmann, 2007). Postural instability and gait disturbances are associated with degeneration in brainstem structures like the pedunculopontine nucleus (Pahapill & Lozano, 2000). Crucially for this project, this progressive dysfunction extends to cognitive and inhibitory control. Patients with PD frequently experience difficulties with both reactive and proactive stopping abilities, which are mediated by fronto-striatal and hyperdirect pathways (Sharif-Razi et al., 2019). These forms of inhibitory control rely on precise dopamine signaling and healthy cortical-subthalamic nucleus connections, circuits that are compromised by neurodegeneration in PD.

Motor inhibition is commonly studied through two related processes: proactive and reactive inhibition. Reactive inhibition refers to the sudden suppression of an ongoing behavior in response to an external signal, while proactive inhibition involves preparing to inhibit a response in anticipation of such a signal. This study explores how ERPs previously associated with Parkinson's disease manifest in different types of motor control. However, due to the novelty of our task design (discussed in detail later) and varying definitions in the literature, the terms "proactive" and "reactive" inhibition may not accurately capture the distinctions within our conditions. Therefore, throughout this paper, we refer to our conditions as *planned* and *unplanned* stops. Both involve some level of anticipatory (i.e., proactive) cognitive processing before the stop signal, but they diverge after the cue: planned stops allow for greater preparatory engagement and show ERP features consistent with proactive inhibition, whereas unplanned stops involve less precise temporal expectation and thus elicit more reactive neural responses.

Event-related Potentials

Event-related potentials (ERPs) provide a noninvasive method to explore the temporal dynamics of cognitive and motor processes. Essentially, ERPs are time-locked neuronal responses that occur before or after an event, and these responses are correlated with cognitive or motor processes (Luck, 2005, Chapter 1). In the context of PD, ERPs have been particularly valuable in explaining how dopaminergic dysfunction alters neural processing, especially during tasks requiring motor inhibition. Among these ERPs, the P200, N200, and P300 have emerged as central markers of attentional modulation, conflict monitoring, response inhibition, and motor preparation—processes all relevant to planned and unplanned stop conditions (Seer et la., 2016; Seer et al, 2017). Understanding these components and their alterations in PD allows us to probe the neural mechanisms of impaired inhibitory control and explore potential biomarkers for tracking disease progression or intervention efficacy.

The P200 component, a positive wave peaking around 150–250 ms post-stimulus, is linked to early sensory processing, attention allocation, and motor preparation. Studies have shown that P200 amplitude in oddball-like experiments is often attenuated and delayed in PD (Hansch et al., 1982), suggesting impaired integration of sensory inputs required for the preparation of motor output. Because a planned stop relies on early cue processing and attentional allocation to prepare a possible stop, alterations in the P200 during these types of tasks in PD may reflect deficits in setting up an internal inhibitory state before the imperative stimulus.

The N200 (N2), appearing between 200–350 ms post-stimulus, is widely associated with conflict monitoring, response selection, and early inhibitory processing (Folstein & Van Petten, 2008). It is thought to originate from the anterior cingulate cortex and prefrontal regions, both

heavily influenced by dopaminergic input from the basal ganglia. In PD, oddball studies have reported reduced N200 amplitudes and prolonged latencies (Xu et al., 2022), indicating diminished conflict detection and early inhibitory control. Given that reactive inhibition is triggered by a sudden need to stop an unplanned or surprise response, the N200's integrity might be critical to such mechanisms. This makes the N2 an ideal marker for assessing early reactive control deficits in PD.

The P300 component, particularly its subcomponents P3a and P3b, reflects attention, memory updating, and inhibitory control. Elicited around 300 ms post-stimulus, P3a is associated with novelty detection and attentional orienting, whereas P3b is linked to context updating and decision-making (Polich, 2007; Donchin, 1981). The P300 is frequently observed in oddball paradigms and Go/No-Go tasks, where its amplitude decreases and latency increases with greater cognitive load or pathology. In PD, oddball studies have consistently shown reduced P300 amplitudes and delayed peaks (Polich, 2009), with greater alterations correlating with disease severity and cognitive decline (Wang et al., 2020). P3 in oddball tasks could be closely tied to reactive inhibition, particularly the process of detecting a surprise response. The reduction in P3 amplitude and increased latency in PD suggest a compromised ability to engage in successful motor inhibition, which can be interpreted as an impulsive, reactive response. (Seer et al., 2016).

Critically, all these ERPs are modulated by neurotransmitter systems. P2 has not been specifically linked to any neurotransmitter. However, dopamine plays a central role, particularly in the N200 and P3a components, while norepinephrine is implicated in modulating P3b (Nieuwenhuis et al., 2005; Luck & Kappenman, 2012, Chapter 8). In PD, reduced dopamine

leads to fronto-striatal dysfunction, which impairs the generation and modulation of these ERPs, reflecting widespread deficits in both cognitive and motor control.

Together, the P200, N200, and P300 allow us to decompose the stages of inhibitory control into measurable neurophysiological events.Since a planned stop involves preparing to inhibit a response ahead of time, I hypothesized this mechanism is likely indexed by P200 and aspects of the P300. Unplanned stopping, on the other hand, might be better captured by N200 and P3NoGo responses, which reflect the detection of a stop signal and the immediate suppression of a response.

Given the use of a novel motor inhibition task, our assumptions are based on prior research, particularly oddball experiments and the Go/No-Go task, which align with our objectives. Nonetheless, the terminology for these components has predominantly been applied to those scenarios, thereby restricting our understanding of their behavior in different contexts, such as the cessation of movement. By comparing these ERP components across groups—specifically, PD patients both on and off medication, and healthy controls—and leveraging existing knowledge about their function in various settings and their neuromodulators, we can map how dopaminergic dysfunction alters each stage of inhibitory processing.

Within this framework, our study utilizes these ERP components to dissect how both planned and unplanned motor inhibition is compromised in PD. We analyze data from a continuous movement task that incorporates these paradigms. This allows us to distinguish group-level differences in ERP timing and amplitude, with the ultimate goal of identifying neurophysiological markers that reflect specific stages of motor control. This approach not only clarifies the mechanisms of inhibitory failure in PD but also offers insight into how dopaminergic modulation shapes behaviorally relevant brain activity. A deeper understanding of

these ERP differences will enhance our ability to design targeted interventions and track their effectiveness over time.

Despite extensive research into basal ganglia dysfunction in PD, significant gaps remain in understanding how these neural changes translate into specific types of motor suppression. While it is well-established that the degeneration of dopaminergic neurons in the basal ganglia contributes to motor symptoms like rigidity, less is known about how these changes affect cognitive domains such as attention and perception. Specifically, the relationship between basal ganglia pathology and alterations in the P2ERP component—widely recognized as a neural correlate of perceptual attention and stimulus evaluation—has not been fully elucidated.

This gap directly informs the central question of my research: How do previously studied ERPs in Parkinson's Disease correlate with evoked potentials in planned and unplanned motor inhibition? I hypothesize that, due to the diminished dopaminergic modulation inherent in PD, patients off medication will exhibit reduced P3, N2, and P2 amplitudes and increased latencies, if present. These changes, I believe, will reflect impaired perceptual attention, conflict monitoring, sensorial perception, and preparation for response, respectively. Similarly, I expect that dopaminergic treatment will partially restore the amplitudes and latencies of all ERPs, except for P2, aligning with anticipated improvements in cognitive function.

METHODS

Study Design

The experiment was based on a case-control design in which 27 white Parkinson's Disease (PD) patients, on dopamine medication only without exclusion of type of symptom, and 26 age-matched white healthy controls (HC) were recruited to participate in a 2-hour motor task. Participants performed a Continuous Movement Stop Task (CMST) while wearing an electroencephalography (EEG) device. CMST, originally proposed by Schultz et al. (2021), is a novel paradigm designed to better simulate real-life motor inhibition by distinguishing between planned and unplanned stopping.

The task involved a countdown displayed on a monitor, with the cues "READY," "SET," and "GO," after which the participant moved a cursor clockwise with their right hand within a circular track on the screen. To examine planned unplanned, the countdown was manipulated to either proceed linearly to zero or terminate abruptly. For example, an unplanned stop followed a sequence such as "6," "5," "4," "3," "STOP," while a planned stop proceeded with "6," ..., "2," "1," "STOP." Participants were instructed to begin moving at the "GO" cue and to cease movement at the "STOP" cue. Planned and unplanned stop trials were randomly interleaved and equally likely throughout the task. On average, each recording had a total of 82 planned conditions and 81 unplanned conditions.

To monitor electrophysiological responses, a total of 64 EEG electrodes were placed according to the international 10–20 system, along with eight EMG electrodes to detect artifacts from eye and limb movement. Specifically, one electrode was placed above and one below each eye, and one on each limb.

Data Collection

Data collection procedures differed slightly between PD participants and healthy controls. PD participants were instructed to withhold dopamine medication for at least 12 hours prior to testing to assess both medicated (ON) and unmedicated (OFF) states. Upon arrival, participants provided informed consent and underwent the Unified Parkinson's Disease Rating Scale (UPDRS) assessment to evaluate disease severity. They then completed the first 30-minute CMST session.

Following a 1-hour break, during which they resumed medication, participants completed the Montreal Cognitive Assessment (MoCA) and then repeated the CMST task under the ON-medication condition. Healthy controls completed the same CMST task (two 30-minute sessions with a break in between) but did not complete the UPDRS. Both tests were administered by trained lab personnel following the respective standardized protocols. All participants received monetary compensation for their time.

Demographics and Data Preprocessing

Demographics on the EEG data included for the final analysis can be found in Table 1. Some recordings were excluded due to excessive noise or data quality issues, and not all the PD participants for the ON-medication session were included in the OFF-medication data set.

Table 1. EEG Data Demographics.

Group	Count	Age (years)	Sex	Years of Education (years)	MoCA Score	UPDRS Score
HC	22	68.23 ± 11.29	6 male and 16 female	17.55 ± 3.00	26.18 ± 2.26	NA
PD OFF	21	69.57 ± 4.3	15 male and 6 female	17.24 ± 3.82	24 ± 3.46	10.48 ± 4.19

PD ON	20	67.00 ± 6.1	15 male and 5 female	17.53 ± 3.84	24.16 ± 3.5	7.79 ± 3.77

The primary objective was to analyze differences in the event-related potentials (ERPs) during planned and unplanned CMST stops. Data processing and analysis were performed using MNE (v1.5.1), Python (v3.11.5), and Excel (v16.96). EEG preprocessing included ICA-based artifact rejection, with only ocular components removed. No additional artifact rejection or outlier exclusion procedures were applied. Nonetheless, a FIR filter was applied to all data with a low-pass filter of 30 Hz and a high-pass filter of 0.1 Hz (sampling frequency = 1024 Hz). The average electrical activity for all channels was used for the EEG reference.

Data Analysis

EEG data were grouped into three categories: healthy controls (HC), Parkinson's disease patients ON medication (PD ON), and Parkinson's disease patients OFF medication (PD OFF). Based on event annotations, each subject's data was epoched separately for planned and unplanned stop trials (tmin = -1.0 s, tmax = 2.0 s). For each epoch, the ERP component was analyzed by extracting mean amplitude, peak amplitude, half-area latency, and peak latency (baseline = -200 to 0 ms) at electrodes Fz for N2, FCz for P2, and Pz for P300. All metrics were computed after averaging trials per subject. The logic behind the channels selected for each ERP was based on the specific scalp areas that elicited the strongest signals, thus minimizing the overlap of ERPs in our data analysis. Table 2 lists the windows selected for each ERP Analyzed in this study. Adjustments to the epochs windows based on previous research on the ERPs and visual examination of the plotted data had to be done to get the most and best amount of data possible. In addition to the ERP analysis, reaction times (RT) were gathered for a deeper understanding of the neuronal processes across time. All peak and mean values were gathered as

absolute values. Depending on the ERP of interest, only positive values were used for the analysis of P2 and P3, while negative values were used for the analysis of N2. Outliers for each metric in every condition were also excluded.

Statistics

All metrics were fit on subject-level averaged data. A one-way ANOVA test was conducted to evaluate the effects of group conditions on all ERP metrics, with separate analyses performed for each electrode of interest. Additionally, post-hoc tests were carried out to identify significant changes in ERP behavior across conditions for each group using Bonferroni correction.

RESULTS

Visual inspection of each component highlights key differences between the oddball and the Go/No-Go task ERPs. Primarily, the window selection did not seem consistent with the tasks mentioned. Due to this, the windows for each ERP study were arranged based on their location in the grand-averaged ERP to continue with wave component analysis. Table 2 lists the tmin and tmax used for each of these.

Table 2. Event-related Potentials Window Selection. T-min and t-max for each event-related potential window is shown in milliseconds. The P3 component was not distinguishable in the ERP plot for channel Pz (Figures 4 and 9), making the data collection window not applicable (NA).

ERP	tmin (ms)	tmax (ms)
P2	179.69	349.61
N2	279.29	419.92
P3	NA	NA

Component P300 was not detectable in any of the groups for both conditions (Figure 1 and 2). The grand-average ERP plot for the Pz channel during the unplanned stop condition (Figure 2) does show a positive peak at 1.5 µV for the PD ON group. Nonetheless, HC and PD OFF did not follow this trend. Consequently, no data analysis was performed for this ERP. Differences within-group per condition are also plotted in Figure 3, 4, and 5 for reference.

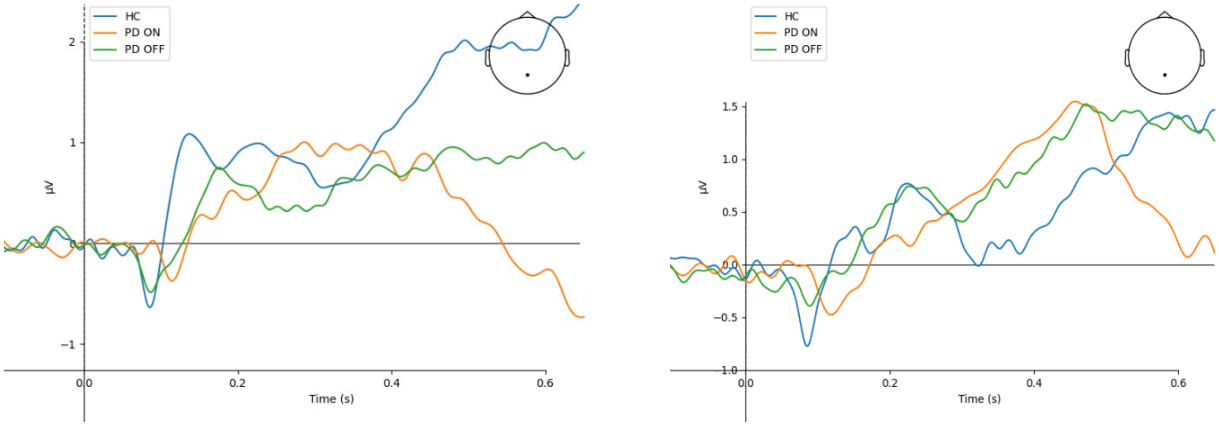
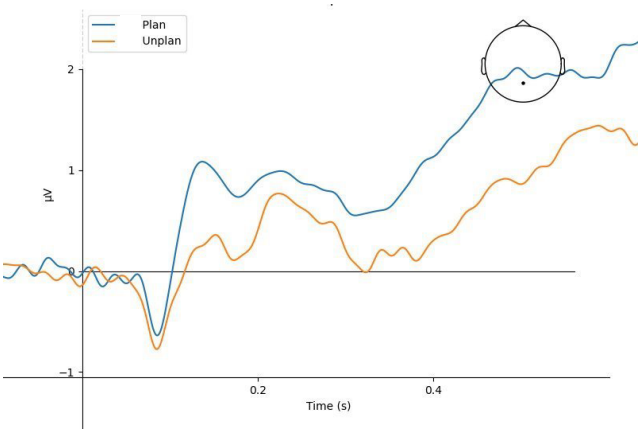


Figure 1. Pz channel grand-average ERP for the planned stop condition across all groups: HC (blue line), PD ON (orange line), and PD OFF (green line). Time zero represents the stop signal. The plot follows a typical Cartesian plane distribution for easier inspection. The top right corner depicts the area of the electrode being studied.

Figure 2. Pz channel grand-average ERP for the unplanned stop condition across all groups: HC (blue line), PD ON (orange line), and PD OFF (green line). Time zero represents the stop signal. The plot follows a typical Cartesian plane distribution for easier inspection. The top right corner depicts the area of the electrode being studied.



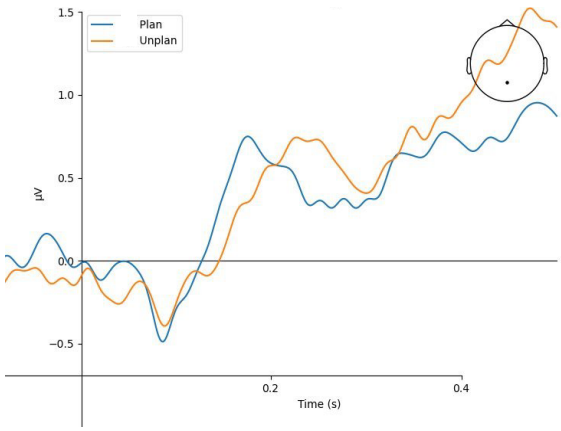


Figure 3. Pz channel grand-average ERP for HC during planned and unplanned stop conditions. Plan condition is depicted with an orange line while unplanned condition is blue. Time zero represents the stop signal. The plot follows a typical Cartesian plane distribution for easier inspection. The top right corner depicts the area of the electrode being studied.

Figure 4. Pz channel grand-average ERP for PD OFF during planned and unplanned stop conditions. Plan condition is depicted with an orange line while unplanned condition is blue. Time zero represents the stop signal. The plot follows a typical Cartesian plane distribution for easier inspection. The top right corner depicts the area of the electrode being studied.

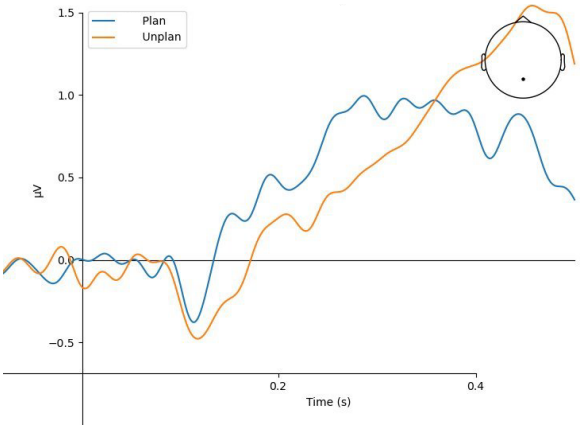


Figure 5. Pz channel grand-average ERP for PD ON during planned and unplanned stop conditions. Plan condition is depicted with an orange line while unplanned condition is blue. Time zero represents the stop signal. The plot follows a typical Cartesian plane distribution for easier inspection. The top right corner depicts the area of the electrode being studied.

Reaction Times

According to pre-liminary data collected from another member of the laboratory where the experiment took place, the average stop reaction time during the plan condition for the HC group was 393 ms, 443 ms for the PD OFF group, and 480 ms PD ON group. For the unplanned condition, the averages increased to 466 ms, 512 ms, and 549 ms, respectively. Thus, hinting at a trend of PD ON>PD OFF> HC for stop reaction time in the experiment.

P2 Component Analysis

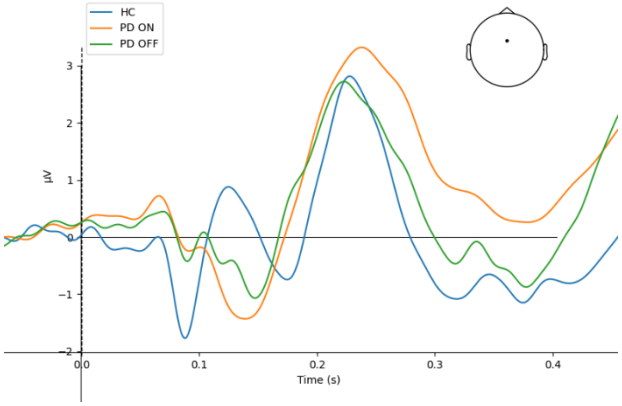


Figure 6. FCz channel grand-average ERP for the planned stop condition across all groups: HC (blue line), PD ON (orange line), and PD OFF (green line). Time zero represents the stop signal. The plot follows a typical Cartesian plane distribution for easier inspection. The top right corner depicts the area of the electrode being studied.

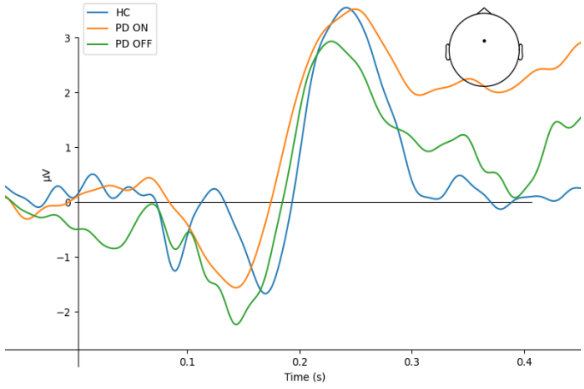


Figure 7. FCz channel grand-average ERP for the unplanned stop condition across all groups: HC (blue line), PD ON (orange line), and PD OFF (green line). Time zero represents the stop signal. The plot follows a typical Cartesian plane distribution for easier inspection. The top right corner depicts the area of the electrode being studied.

Figures 6 and 7 show the presence of P2 in the FCz channel during the task. The component was analyzed for peak amplitude, mean amplitude, peak latency, and half-area latency. Table 2 summarizes the mean ± standard deviation for each measure across groups and conditions. Figures 8, 9, and 10 show the differences between planned and unplanned conditions within the same group.

Table 3: P2 Component Descriptives (Mean ± SD)

Measure	Condition	HC	PD OFF	PD ON
Peak Amplitude (μV)	Plan	7.58 ± 3.76	7.40 ± 3.70	5.12 ± 3.06
	Unplan	6.78 ± 2.10	5.85 ± 1.75	4.81 ± 2.20
Mean Amplitude (μV)	Plan	3.84 ± 2.19	2.56 ± 2.25	2.32 ± 2.03
	Unplan	2.68 ± 1.54	1.86 ± 1.21	2.04 ± 1.55
Peak Latency (ms)	Plan	242.48 ± 36.85	250.41 ± 58.55	251.90 ± 46.31
	Unplan	275.95 ± 44.00	258.61 ± 54.67	268.96 ± 49.51
Half-Area Latency (ms)	Plan	267.38 ± 26.91	264.52 ± 31.81	255.70 ± 19.11
	Unplan	266.29 ± 23.10	256.37 ± 32.38	261.07 ± 20.75

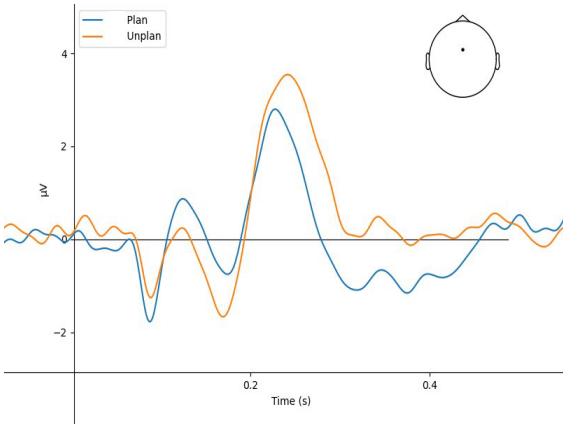


Figure 8. FCz channel grand-average ERP for HC during planned and unplanned stop conditions. Plan condition is depicted with an orange line while unplanned condition is blue. Time zero represents the stop signal. The plot follows a typical Cartesian plane distribution for easier inspection. The top right corner depicts the area of the electrode being studied.

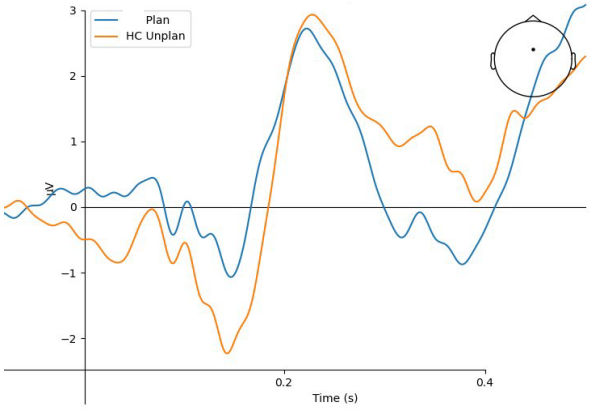


Figure 9. FCz channel grand-average ERP for PD OFF during planned and unplanned stop conditions. Plan condition is depicted with an orange line while unplanned condition is blue. Time zero represents the stop signal. The plot follows a typical Cartesian plane distribution for easier inspection. The top right corner depicts the area of the electrode being studied.

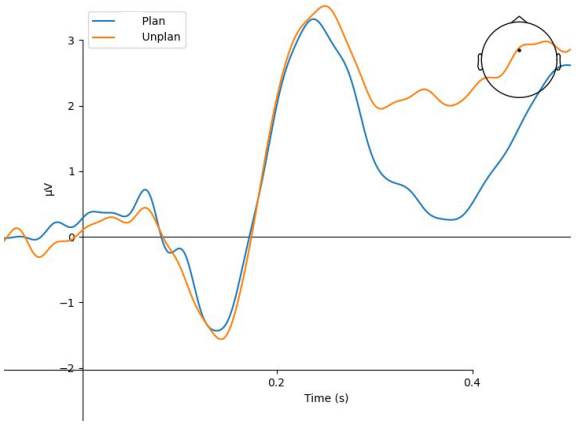


Figure 10. FCz channel grand-average ERP for PD ON during planned and unplanned stop conditions. Plan condition is depicted with an orange line while unplanned condition is blue. Time zero represents the stop signal. The plot follows a typical Cartesian plane distribution for easier inspection. The top right corner depicts the area of the electrode being studied.

Group Differences

Peak Amplitude: In the planned condition, there were a total of 10 HC, 17 PD ON subjects, and 12 PD OFF subjects' datasets remaining after applying the exclusion criteria. In the unplanned condition, there were 14 HC, 16 PD ON, and 9 PD OFF subjects left after exclusions. One-way ANOVA revealed no significant group differences in P2 peak amplitude during the plan condition ($F=2.27$, $p=0.12$, $\eta2=0.11$). However, a significant group effect was found in the unplan condition ($F=3.41$, $p=0.04$, $\eta2=0.16$). Post-hoc comparisons indicated that the HC group had significantly higher P2 peak amplitude than the PD ON group ($p=0.035$). No other significant pairwise differences were observed in either condition.

Mean Amplitude: Significant group differences in P2 mean amplitude were observed in both the plan ($F=6.34$, $p=0.002$, $\eta2=0.066$) and unplan ($F=6.84$, $p=0.001$, $\eta2=0.061$) conditions. Data for the plan condition was representative of 10 HC, 17 PD ON, and 12 PD OFF subjects, while for the unplanned conditions this was based on 15 HC, 19 PD ON, and 11 PD OFF

subjects after exclusion criteria. As depicted in Figures 5 and 6, in the plan condition, HC showed significantly greater mean amplitude than PD ON (p=0.002). In the unplan condition, HC differed significantly from both PD OFF (p=0.002) and PD ON (p=0.012).

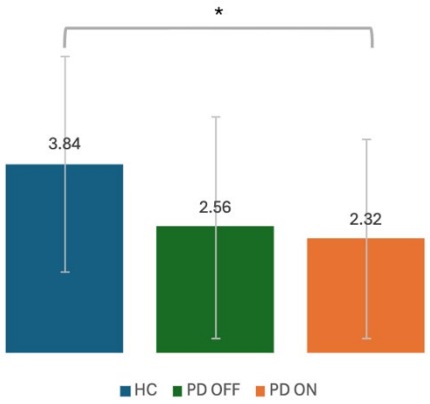


Figure 11. Mean Amplitude of P2 during the plan condition. HC (blue), PD ON (orange), and PD OFF (green). Data is portrayed as the mean across subjects and STD. "*" indicates significance at the 5% level.

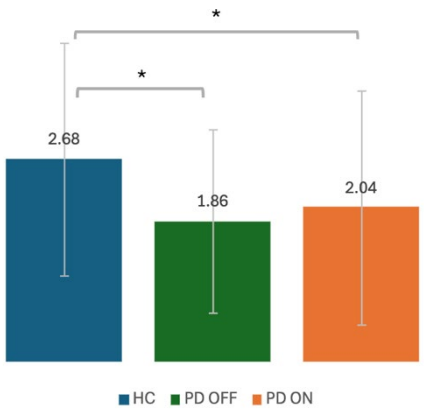


Figure 12. Mean Amplitude of P2 during the unplan condition. HC (blue), PD ON (orange), and PD OFF (green). Data is portrayed as the mean across subjects and STD. "*" indicates significance at the 5% level.

Latency Measures: Neither P2 peak latency (plan: F=0.12, p=0.88, η2=0.006; unplan: F=0.24, p=0.78, η2=0.012) nor half-area latency (plan: p=0.34; unplan: p=0.46) showed significant differences across groups in either condition.

Within-Group Condition Differences

Paired t-tests for significant differences between planned and unplanned conditions within each group revealed no significant changes for P2 peak amplitude, mean amplitude, peak latency, or half-area latency in any group (p>0.05 for all comparisons).

N2 peak amplitude at the Fz channel was not significantly different across groups per condition ($p>0.05$). Similarly, the peak amplitude of each group at both conditions was not statistically relevant.

N2 Component Analysis

Figures 13 and 14 show the electric activity in the Fz channels for the window analyzed. N2 appears weak in the plan condition and ambiguous in the unplanned condition. Nonetheless, the component was analyzed for peak amplitude, mean amplitude, peak latency, and half-area latency for possible significance. Table 3 summarizes the mean ± standard deviation for each measure across groups and conditions. Figures 15, 16, and 17 show within-group condition differences.

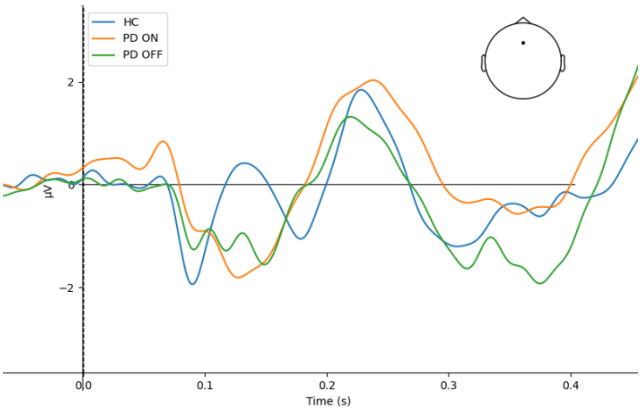


Figure 13. Fz channel grand-average ERP for the planned stop condition across all groups: HC (blue line), PD ON (orange line), and PD OFF (green line). Time zero represents the stop signal. The plot follows a typical Cartesian plane distribution for easier inspection. The top right corner depicts the area of the electrode being studied.

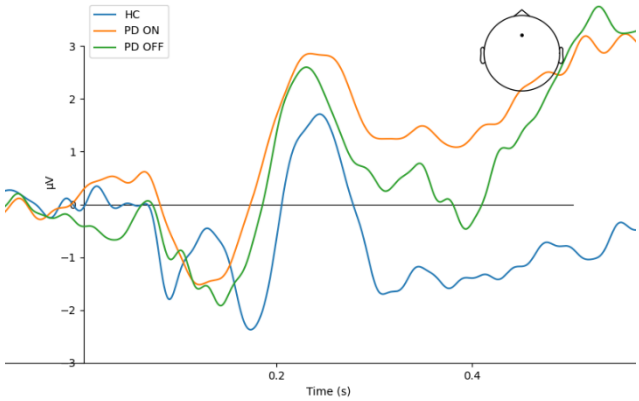


Figure 14. Fz channel grand-average ERP for the unplanned stop condition across all groups: HC (blue line), PD ON (orange line), and PD OFF (green line). Time zero represents the stop signal. The plot follows a typical Cartesian plane distribution for easier inspection. The top right corner depicts the area of the electrode being studied.

Table 4: N2 Component Descriptives (Mean ± SD)

Measure	Condition	HC	PD OFF	PD ON
Peak Amplitude (μV)	Plan	-5.12 ± 1.28	-5.93 ± 3.10	-4.31 ± 3.05
	Unplan	-4.52 ± 2.20	-5.50 ± 2.30	-3.40 ± 2.05
Mean Amplitude (μV)	Plan	-3.12 ± 1.80	-2.94 ± 2.36	-1.94 ± 1.43
	Unplan	-3.69 ± 3.02	-2.86 ± 2.18	-1.03 ± 0.72
Peak Latency (ms)	Plan	358.79 ± 57.48	362.79 ± 38.50	350.59 ± 31.25
	Unplan	359.13 ± 46.7±9	356 ± 48.40	355.96 ± 40.53
Half-Area Latency (ms)	Plan	351.44 ± 16.81	349.70 ± 20.95	358.45 ± 14.98
	Unplan	348.23 ± 18.80	357.24 ± 24.05	354.50 ± 19.97

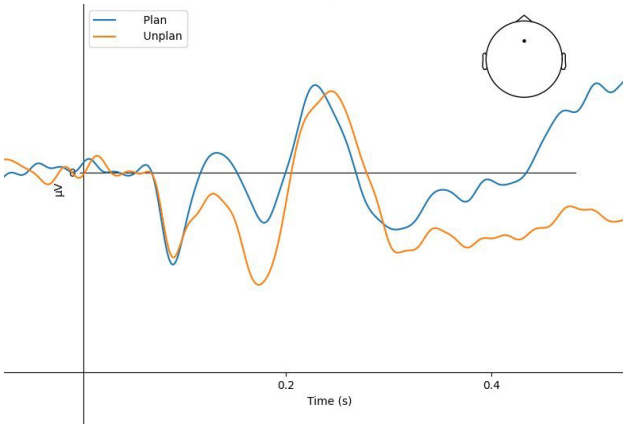


Figure 15. Fz channel grand-average ERP for HC during planned and unplanned stop conditions. Plan condition is depicted with an orange line while unplanned condition is blue. Time zero represents the stop signal. The plot follows a typical Cartesian plane distribution for easier inspection. The top right corner depicts the area of the electrode being studied.

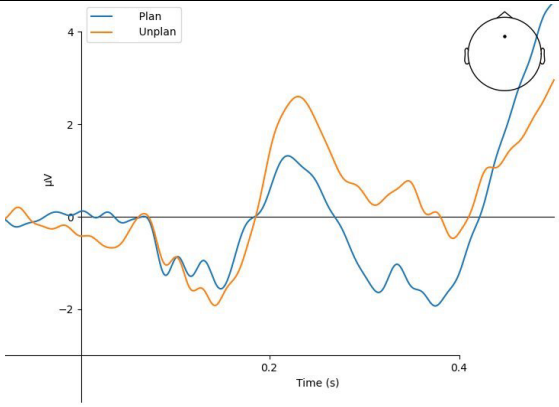


Figure 16. Fz channel grand-average ERP for PD OFF during planned and unplanned stop conditions. Plan condition is depicted with an orange line while unplanned condition is blue. Time zero represents the stop signal. The plot follows a typical Cartesian plane distribution for easier inspection. The top right corner depicts the area of the electrode being studied.

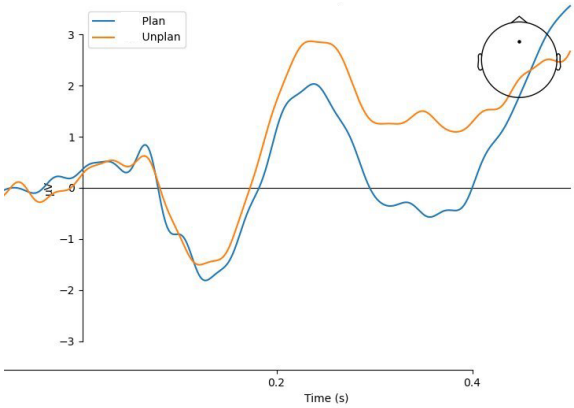


Figure 17. Fz channel grand-average ERP for PD ON during planned and unplanned stop conditions. Plan condition is depicted with an orange line while unplanned condition is blue. Time zero represents the stop signal. The plot follows a typical Cartesian plane distribution for easier inspection. The top right corner depicts the area of the electrode being studied.

Group Differences

Across all analyzed metrics of the N200 component—peak amplitude, mean amplitude, peak latency, and half-area latency—no significant differences were observed between HC, PD OFF, and PD ON groups in either the plan or unplan conditions. Specifically, peak latency showed no significant group differences in the plan condition ($F=0.21$, $p=0.81$, $\eta2=0.012$) or unplan condition ($F=0.018$, $p=0.98$, $\eta2=0.001$). Similarly, group differences for peak amplitude were not significant in either the plan ($F=1.10$, $p=0.34$, $\eta2=0.064$) or unplan condition ($F=1.79$, $p=0.18$, $\eta2=0.12$). Half-area latency also revealed no significant group differences in the plan condition ($F=1.38$, $p=0.25$, $\eta2=0.044$) or unplan condition ($F=1.03$, $p=0.36$, $\eta2=0.033$). Finally, the mean amplitude of the N200 component did not show any significant differences between groups in the plan ($F=1.19$, $p=0.31$, $\eta2=0.065$) or unplan condition ($F=3.19$, $p=0.06$, $\eta2=0.17$).

Within-Group Condition Differences

No significant differences were observed when comparing the plan and unplan conditions within any of the groups for peak latency, peak amplitude, mean amplitude, or half-area latency ($p>0.05$ for all comparisons).

DISCUSSION

This project aimed to refine our understanding of ERPs associated with planned and unplanned inhibition in a task where movement is not simply suppressed internally but actually executed and then stopped. Unlike traditional paradigms such as the oddball or Go/No-Go tasks—which primarily study suppression of action that never occurs (Huster et al., 2013; Wessel & Aron, 2015)—our Continuous Movement Stop Task (CMST) required participants to initiate movement and then halt it upon either a planned or an unplanned cue. This distinction is more reflective of real-life motor control demands and positions our study to contribute novel insights into the neural dynamics of inhibitory processing (Schultz et al., 2021).

One of the most striking deviations from previous literature was the absence of a clearly defined P300 component in our data, particularly at the Pz electrode. This contrasts sharply with classic ERP findings where the P300, especially NoGo-P3, is consistently elicited in response inhibition tasks and linked to attentional reallocation and context updating (Polich, 2007; Luck, 2014; Seer et al., 2016). Despite the cognitive demands of our task—such as sustained attention and working memory—no consistent P300 response emerged. This may suggest that the P300 is more sensitive to internal movement suppression rather than external movement halting. In Go/No-Go tasks, participants prepare a response but withhold it entirely; in our paradigm, they must stop an ongoing action. This could imply that P300 generation relies more on processes involved in evaluating the non-execution of movement rather than interruption of action already in progress. Thus, our findings suggest the possibility that P300 reflects a context evaluation and internal inhibition mechanism, not necessarily engaged when inhibition follows initiation (Wessel & Aron, 2017). Nevertheless, it is also possible that based on the baseline of the study,

which was set during movement, could have obscured the P3 findings, therefore, making it harder to see a peak in the grand-averaged ERPs.

Similar to that note, the N200 component, typically associated with early inhibitory control, conflict monitoring, and response selection (Folstein & Van Petten, 2008; Luck, 2014), showed high variability and no statistically significant differences between groups. During the unplanned condition, both PD groups showed a negative peak above 0 micro volts. This could be most likely attributed to the unstable baseline used for ERP arrangement. Our findings are partially consistent with literature showing diminished N2 amplitudes in PD during Go/No-Go tasks, often interpreted as reduced conflict monitoring or impaired early-stage inhibition (Bokura et al., 2005). However, in our executed inhibition paradigm, the N2 lacked clear definition in ERP plots—particularly among PD participants. This could be due to the additional temporal and cognitive complexity of stopping an active movement rather than canceling a prepotent response. It may also reflect the known variability in latency and amplitude of the N2 across individuals and contexts (Patel & Azzam, 2005), further complicated by dopamine-related fronto-striatal circuit disruptions in PD (Wang et al., 2020).

Moreover, the N2 variability in our results might speak to the differing roles planned and unplanned motor control play when action is already underway. It is plausible that reactive stopping in our unplanned condition did not engage in the same networks or temporal patterns as those traditionally observed in tasks where inhibition is preemptive or fully cognitive.

Our results still need to be replicated with a higher set of data since once exclusion criteria were applied, the total dataset decreased significantly, thus affecting the generalizability of our results. For example, Figures 11 and 12 hint at this same problem by depicting big error bars and showing a lack of correlation between them and the plotted ERPs.

Interestingly, the P2 component was the clearest and most statistically robust ERP across our data, especially in the FCz region. P2 is typically linked to early perceptual processing and attentional mechanisms (Luck & Kappenman, 2012), and its prominence in our results might suggest a stronger reliance on sensory-motor integration in tasks requiring movement termination.

It is worth noting that while peak and mean P2 amplitudes varied between groups, latency measures were not significantly different. This may indicate that the timing of early perceptual processing is preserved in PD, but the neural efficiency or resource allocation may differ. The same problems portrayed before with the sample count can be applied to P2 since our final results were gathered from a small pool.

One of the most unexpected yet compelling findings in this study was that the PD ON group—despite being on dopaminergic medication expected to restore deficits—showed significantly *lower* mean P2 amplitudes compared to the PD OFF group. This was particularly striking because the P2 component, associated with early sensory and attentional processing, was hypothesized to improve with medication. The fact that PD ON had significantly reduced mean amplitudes, especially when contrasted with healthy controls, challenges assumptions about dopaminergic normalization and instead supports the possibility of an *inverted-U-shaped* dopamine effect on cognition—where both too little and too much dopamine impair function (Cools & D'Esposito, 2011; van der Schaaf et al., 2013). Further aligning with this, reaction time data also indicated that the PD ON group had the slowest responses compared to PD OFF and healthy controls, suggesting a potential behavioral cost associated with overtreatment. These results underscore the need for more precise dopamine modulation in PD treatment, as excessive dopaminergic input may disrupt neural efficiency, particularly in circuits governing early

perceptual-motor integration. In this light, the reduced P2 amplitude in PD ON may reflect not a failure of medication per se, but rather an overcorrection for the loss of control driven by dopamine overdose (Frank et al., 2007).

Methodological Considerations

Component window misalignment across studies is a known issue in ERP research (Luck, 2005, Chapter 6), and the differences in ERP detection here could partially stem from this. We relied on literature-informed and visually adjusted time windows (Table 1), which might not precisely match the component timing in our novel task. However, given the substantial variability in ERP metrics across tasks and individuals, these inconsistencies are not unexpected and do not necessarily invalidate our findings.

The weak definition of N2 in the ERP waveform further suggests considerable inter-subject variability, which may be exacerbated in clinical populations like PD (Bokura et al., 2005; Hansch et al., 1982). Additionally, as mentioned before, reduced sample size and power after outlier exclusion could have significantly affected ERP and metrics clarity, especially in the more variable and less prominent components of N2.

Another potential confound is the gender imbalance across groups—our healthy control participants were primarily women, whereas PD participants were predominantly men. While gender effects on ERP components have been documented (Lee et al., 2017), they are generally small compared to the more robust effects of pathology or task differences. Nonetheless, it is worth considering this imbalance in future work and statistical modeling.

CONCLUSION AND FUTURE DIRECTIONS

This study advances the understanding of inhibitory control in tasks involving executed movement stopping, a condition more closely aligned with daily motor demands than traditional inhibition paradigms. The findings suggest that the P300, typically considered a key marker of motor inhibition, may not be consistently observed in scenarios requiring abrupt cessation of ongoing movement. Instead, components like the P2 might offer more informative insights in such contexts. The attenuated or absent P3 and N2 components, particularly in Parkinson's disease (PD) participants, may reflect impairments in planned and unplanned control mechanisms due to dopaminergic loss. Conversely, the prominent P2 suggests that early-stage attentional and perceptual processes are crucial when stopping executed movements and could serve as alternative biomarkers for inhibitory control in PD.

For future research, it is recommended to replicate these findings in larger and more demographically balanced cohorts. Employing source localization or time-frequency analyses could help to delineate the contributions of distinct neural circuits. Additionally, integrating neurochemical imaging or pharmacological manipulations could further clarify the role of dopamine in modulating these ERP components. As emphasized by Steven Luck, ERPs offer a powerful lens into cognition and behavior, but their interpretation must always consider the specific task and population. This study contributes to a more nuanced understanding, aiming to redefine the ERP correlates of inhibitory control within the context of real-time motor execution.

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