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Examining the Effectiveness of Infra-Low Frequency Neurofeedback on Cognitive and Clinical Components and Brain Signals in Patients with Parkinson's Disease

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A B S T R A C T

Objective: The aim of the present study was to examine the effectiveness of infra-low frequency neurofeedback on cognitive and clinical components and brain signals in patients with Parkinson's disease.
Methods and Materials: The study population consisted of patients with Parkinson's disease aged 50 to 95 years, with disease severity at stages 2 or 3 according to the Hoehn and Yahr criteria, who were referred to the neurology clinic of Ayatollah Rouhani Hospital in Babol. The final sample included 5 participants. During four phases of the study, participants completed computerized tests from the RehaCom software, the Unified Parkinson's Disease Rating Scale, and the Montreal Cognitive Assessment. After the second pretest phase, each patient received 20 sessions of 30-minute infra-low frequency neurofeedback intervention based on the Othmer protocol (2017). Brainwave activity was recorded at each phase of the study. Behavioral data were analyzed using the paired-samples t-test, while brain signal data were analyzed using analysis of variance (ANOVA) and post hoc tests.
Findings: The findings indicated improvements in motor and cognitive performance of participants at the follow-up stage compared to the pretest. Results from the RehaCom computerized tests demonstrated improvements in selective attention, working memory capacity, and logical reasoning. Additionally, the absolute power of gamma and beta brainwaves decreased, suggesting enhanced cognitive and emotional regulation in participants.
Conclusion: The findings of this study indicate that infra-low frequency neurofeedback is effective in improving motor and cognitive functioning in individuals with Parkinson's disease. Considering the results of the present study and the importance of non-invasive treatments in age-related disorders—particularly Alzheimer's and Parkinson's diseases—in enhancing cognitive and motor performance and slowing the progressive course of these disorders, greater attention to novel therapeutic approaches and further research is essential to address existing limitations and gaps in this field.
Keywords: *Parkinson's disease, infra-low frequency, neurofeedback, cognitive profile*

1. Introduction

Parkinson's disease is a progressive neurodegenerative disorder that imposes a substantial burden on affected individuals, families, and health systems because it compromises motor performance, cognition, affective functioning, autonomy, and quality of life over time (De Lau & Breteler, 2006; Raza & Anjum, 2019; Sveinbjornsdottir, 2016). Although Parkinson's disease is classically identified by bradykinesia, tremor, rigidity, and postural instability, its clinical presentation extends well beyond motor dysfunction to include deficits in attention, executive control, working memory, emotional regulation, autonomic function, and broader neuropsychiatric symptoms, all of which contribute to cumulative disability and reduced daily functioning (Riedel et al., 2010; Sveinbjornsdottir, 2016). This multidimensional profile complicates treatment planning because many patients experience incomplete response to standard pharmacological approaches, fluctuations in symptom severity, and persistent cognitive-behavioral disturbances even when overt motor symptoms are partially controlled (Raza & Anjum, 2019; Riedel et al., 2010). As a result, contemporary Parkinson's disease research increasingly emphasizes multimodal and non-invasive interventions capable of targeting not only overt motor impairment but also the neural dysregulation that underlies cognitive and behavioral deterioration (Badicu et al., 2025; Cook et al., 2021; Legarda et al., 2022).

The pathophysiology of Parkinson's disease is now widely understood as a disorder of distributed neural networks rather than a purely focal basal ganglia condition. Abnormal interactions among cortical, subcortical, thalamic, and brainstem systems lead to disturbances in motor initiation, inhibitory control, cognitive flexibility, and sensorimotor integration (Hammond et al., 2007; Raza & Anjum, 2019). Electrophysiological studies have shown that pathological synchronization is a central hallmark of Parkinsonian dysfunction, especially within beta-band activity, which has been associated with rigidity, bradykinesia, impaired motor updating, and the maintenance of an overly stable neural state that resists adaptive change (Engel & Fries, 2010; Hammond et al., 2007; Zaidel et al., 2010). From this perspective, beta oscillations may reflect an exaggerated neural tendency to preserve the status quo, thereby interfering with flexible motor and cognitive operations in Parkinson's disease (Engel & Fries, 2010). At the same time, gamma-band activity has been linked to cortical computation, motor control, and task-relevant information integration, and changes in gamma dynamics in the basal ganglia and cortex appear to be highly relevant to both compensatory functioning and disease-related dysregulation (Fries, 2009; Jenkinson et al., 2013). The clinical importance of oscillatory abnormalities is reinforced by findings showing that the spatial extent and expression of beta oscillations can predict therapeutic response to deep brain stimulation, underscoring the translational significance of abnormal rhythmic activity in Parkinson's disease (Zaidel et al., 2010).

Research on motor inhibition and action control has further clarified the relevance of abnormal oscillatory regulation in Parkinson's disease. The subthalamic nucleus has been shown to play a critical role in successful response inhibition, and electrophysiological evidence indicates that impaired stopping and inhibitory control in Parkinson's disease are closely linked to network-level oscillatory dysfunction (Alegre et al., 2013). More generally, effective corticospinal interaction depends on coherent neural communication across distributed systems, and disruptions in neuronal coherence can degrade the precision and adaptability of behavior (Schoffelen et al., 2005). In Parkinson's disease, these disruptions are not limited to movement execution but extend into executive control, decision processes, and cognitive monitoring, which helps explain why motor and cognitive impairments often co-occur and progress together (Alegre et al., 2013; Riedel et al., 2010). This convergence supports a therapeutic rationale focused on restoring rhythmic coordination and neural flexibility rather than treating symptoms in isolation.

An additional layer of complexity is introduced by slow and infra-slow neural dynamics. While much Parkinson's disease research has focused on beta and gamma activity, slower oscillatory processes are increasingly recognized as important regulators of large-scale brain organization and state-dependent neural responsiveness (Hiltunen et al., 2014; Mitra et al., 2018). Infra-slow EEG fluctuations are correlated with resting-state network dynamics observed in fMRI, suggesting that extremely slow neural processes may shape the temporal architecture of functional integration across the brain (Hiltunen et al., 2014). Resting-state fMRI studies have established that the brain is organized into distinct long-distance networks during rest, and such networks are highly relevant to cognition, self-monitoring, memory, attention, and disease expression (Buckner et al., 2008; De Luca et al., 2006). Because infra-slow activity appears to modulate or scaffold network-level organization, interventions targeting these fluctuations may offer a

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pathway for influencing broad neural systems rather than isolated cortical sites (Hiltunen et al., 2014; Mitra et al., 2018). This possibility is especially important in Parkinson's disease, where impairments arise from distributed network dysfunction spanning motor, cognitive, affective, and autonomic domains (Hammond et al., 2007; Sveinbjornsdottir, 2016).

Infra-low frequency neurofeedback has emerged within this theoretical context as a promising form of neuromodulation. Unlike conventional neurofeedback protocols that typically train activity in higher-frequency bands, infra-low frequency neurofeedback aims to influence very slow neural fluctuations presumed to support self-regulation, state stability, and large-scale network balance (Grin-Yatsenko et al., 2021; Schneider et al., 2021; Smith et al., 2014). The technical and clinical development of infra-slow or infra-low frequency training has suggested that these protocols may be particularly suitable for disorders characterized by unstable arousal regulation, inefficient network coordination, and persistent symptom clusters that are resistant to standard treatment (Schneider et al., 2021; Smith et al., 2014). Evidence indicates that infra-low frequency neurofeedback can modify infra-slow EEG fluctuations themselves, supporting the plausibility that this intervention exerts measurable effects on the neural substrates it targets (Grin-Yatsenko et al., 2021). Because Parkinson's disease involves impaired regulation of oscillatory activity across multiple temporal scales, infra-low frequency neurofeedback may be especially relevant as an intervention that addresses foundational neural timing mechanisms rather than only surface-level symptoms (Cook et al., 2021; Legarda et al., 2022).

The broader field of neuromodulation also lends support to investigating infra-low frequency neurofeedback in Parkinson's disease. Non-invasive brain-based interventions such as transcranial direct current stimulation have shown beneficial effects on cognitive, emotional, and performance-related outcomes in different populations, including children with ADHD, children with autism spectrum disorder, healthy adults, athletes, and older adults (Badicu et al., 2025; Jahedi Delivand et al., 2024; Shamsi Halasu et al., 2023; Taheri et al., 2025; Talebi & Hashemi Mad, 2025). Although these populations differ from Parkinson's disease, the cumulative evidence supports the broader principle that targeted neuromodulation can alter behavioral performance, psychological resilience, neural efficiency, and quality of life (Badicu et al., 2025; Taheri et al., 2025; Talebi & Hashemi Mad, 2025). Real-time neurofeedback studies using fMRI have likewise demonstrated that individuals can learn to regulate activity in specific regions or larger networks, with resulting improvements in anxiety and self-regulatory capacity (deCharms, 2007; Kim et al., 2024; Nicholson et al., 2023; Ruiz et al., 2014). These findings are important because they establish the general feasibility of training brain activity to achieve clinically meaningful outcomes, reinforcing the logic of applying neurofeedback approaches to disorders with network-level dysregulation (deCharms, 2007; Ruiz et al., 2014).

The relevance of neurofeedback to Parkinson's disease has been explored in a limited but growing body of literature. Theoretical and clinical discussions have proposed that neurofeedback may help reorganize dysfunctional neural networks in Parkinson's disease and improve both neurological and behavioral outcomes (Esmail & Linden, 2014). Initial feasibility work on sensorimotor rhythm neurofeedback has suggested that such training can be implemented in Parkinson's disease and may produce favorable changes in symptoms and self-regulation, although evidence remains preliminary and based on small samples (Cook et al., 2021). Infralow frequency neuromodulation has also been reported as a nonsurgical approach for managing intractable symptoms of Parkinson's disease, offering encouraging clinical observations for patients whose needs are not fully addressed by conventional interventions (Legarda et al., 2022). Furthermore, exercise-related neurofeedback research has shown that neurofeedback training may improve physical balance in Parkinson's disease, which is particularly relevant because balance impairment is among the most disabling and fall-related features of the disorder (Azarpaikan et al., 2014). Together, these studies provide a rationale for extending inquiry beyond general symptom reduction and toward a more integrated evaluation of clinical, cognitive, and electrophysiological outcomes.

The cognitive dimension is especially important. Cognitive dysfunction in Parkinson's disease often involves executive processes, attentional control, working memory, reasoning, and mental flexibility, all of which can decline even in the absence of overt dementia and meaningfully affect independence and treatment responsiveness (Riedel et al., 2010; Sveinbjornsdottir, 2016). Because successful motor behavior depends on intact attention, inhibitory control, and action monitoring, cognitive and motor deficits should not be conceptualized as entirely separate domains in Parkinson's disease (Alegre et al., 2013; Schoffelen et al., 2005). This is one reason that interventions capable of

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modulating neural networks at multiple levels may be more promising than symptom-specific approaches. If infra-low frequency neurofeedback can influence arousal regulation, network stability, inhibitory control, and oscillatory balance, then improvements may emerge simultaneously in motor symptoms, general cognitive status, and specific neuropsychological functions. Such a possibility is consistent with evidence that modulation of brain activity can alter both discrete regional processes and distributed functional systems (Buckner et al., 2008; De Luca et al., 2006; deCharms, 2007; Ruiz et al., 2014).

Another reason to focus on infra-low frequency dynamics is that Parkinson's disease symptoms are often state-sensitive and fluctuate across time. The temporal architecture of the brain matters clinically because pathological resonance and abnormal synchronization may be sustained or amplified under certain conditions, contributing to dyskinesia, rigidity, tremor, and inefficiency in cognitive-motor coordination (Alonso-Frech et al., 2006; Halje et al., 2012; Richter et al., 2013). Levodopa-induced dyskinesias, for example, have been associated with slow oscillatory activity and resonant cortical states, highlighting the extent to which Parkinsonian symptomatology is embedded in aberrant rhythmic organization (Alonso-Frech et al., 2006; Halje et al., 2012; Richter et al., 2013). This line of evidence suggests that treatments seeking to reshape oscillatory context rather than merely suppress isolated manifestations may yield broader and potentially more durable effects. Infra-low frequency neurofeedback is conceptually aligned with this objective because it targets very slow regulatory processes presumed to influence how higher-frequency rhythms are organized and expressed (Grin-Yatsenko et al., 2021; Mitra et al., 2018; Smith et al., 2014).

Methodologically, incorporating electrophysiological outcomes into intervention studies is especially valuable in this area. EEG provides direct temporal access to neural rhythms and is well suited for examining changes in oscillatory dynamics associated with neuromodulatory treatments. Advances in multimodal mapping between intracranial EEG and fMRI further strengthen the interpretive value of electrophysiological findings by situating rhythmic activity within broader functional network architecture (Carmichael et al., 2024). At the same time, systematic reviews of resting-state fMRI in psychiatric and neurological conditions underscore both the promise and the complexity of network-level biomarkers, emphasizing the need for carefully designed studies that link neural change to clinically meaningful outcomes (Zugman et al., 2023). In Parkinson's disease, where oscillatory abnormalities are among the most reproducible neural findings, combining behavioral and brain-signal indices in treatment research is particularly important for clarifying both mechanism and efficacy (Engel & Fries, 2010; Hammond et al., 2007; Jenkinson et al., 2013).

Despite the conceptual and clinical promise of infra-low frequency neurofeedback, the evidence base remains underdeveloped, especially for Parkinson's disease. Existing studies have often been exploratory, feasibility-oriented, or focused on restricted outcome domains such as motor symptoms or single electrophysiological markers (Cook et al., 2021; Legarda et al., 2022). There remains a clear need for research that simultaneously evaluates clinical symptoms, cognitive performance, and brainwave changes within the same protocol. Such an integrated design is necessary because an intervention may produce measurable neural shifts before or alongside overt behavioral improvements, or conversely may improve patient functioning through distributed mechanisms not fully captured by one behavioral scale alone (Carmichael et al., 2024; Grin-Yatsenko et al., 2021; Hiltunen et al., 2014). Moreover, because Parkinson's disease affects older adults who may also experience anxiety, reduced well-being, and performance-related decline, the growing literature on neuromodulatory enhancement of cognition, self-regulation, and psychological functioning in other groups provides a useful translational backdrop for Parkinson's intervention research (Badicu et al., 2025; Kim et al., 2024; Nicholson et al., 2023; Taheri et al., 2025; Talebi & Hashemi Mad, 2025).

Accordingly, the present study was designed to examine whether infra-low frequency neurofeedback can improve motor and cognitive outcomes while also producing measurable changes in brain signals in patients with Parkinson's disease, thereby addressing a clinically significant gap at the intersection of neurodegenerative disorder management, cognitive rehabilitation, and network-based neuromodulation (Cook et al., 2021; Esmail & Linden, 2014; Legarda et al., 2022). The study is grounded in converging evidence that Parkinson's disease is associated with pathological synchronization, abnormal beta and gamma dynamics, disrupted inhibitory control, and large-scale dysregulation of neural communication, all of which may be relevant targets for interventions that operate at the level of oscillatory self-regulation (Alegre et al., 2013; Engel & Fries, 2010; Fries, 2009; Hammond et al., 2007; Zaidel et al., 2010). It also draws on the emerging view that infra-slow

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and resting-state neural fluctuations are not peripheral phenomena but core organizing features of brain function that may be harnessed therapeutically (Buckner et al., 2008; De Luca et al., 2006; Hiltunen et al., 2014; Mitra et al., 2018). By evaluating clinical impairment, general cognition, specific cognitive components, and EEG indices in one framework, the study seeks to contribute to a more precise understanding of whether infra-low frequency neurofeedback can serve as a meaningful adjunctive intervention in Parkinson's disease (Grin-Yatsenko et al., 2021; Schneider et al., 2021; Smith et al., 2014).

The aim of the present study was to investigate the effectiveness of infra-low frequency neurofeedback on clinical symptoms, cognitive components, and brain signals in patients with Parkinson's disease.

2. Methods and Materials

2.1. Study Design and Participants

The present study employed a quasi-experimental design and, in terms of purpose, is categorized as an applied study. The study population consisted of patients with Parkinson's disease aged 50 to 95 years, with disease severity at stages 2 or 3 according to the Hoehn and Yahr criteria, who were referred to the neurology clinic of Ayatollah Rouhani Hospital in Babol. All patients aged 50 to 95 years with disease severity at stages 2 or 3 based on the Hoehn and Yahr scale who had visited the neurology clinic of Rouhani Hospital in Babol within the past year were considered as the target population. The final sample was selected from the target population using convenience sampling among patients who met the inclusion criteria. A total of five patients were selected for this study. Inclusion criteria were as follows: written informed consent to participate in the study, diagnosis of idiopathic Parkinson's disease based on standard criteria and the four cardinal symptoms, absence of dementia, absence of severe depression, minimum literacy in reading and writing, and absence of other parkinsonian disorders. Exclusion criteria included head trauma within the past year (Schretlen & Shapiro, 2009), substance dependence (Curran et al., 2016), and alcohol consumption (Weisburn & Duka, 2003). Withdrawal criteria included the participant's unwillingness to continue participation at any stage of the study.

2.2. Instruments

RehaCom software is an interactive program designed for training cognitive abilities. This system includes exercises aimed at improving attention, spatial processing, and executive functioning. Training sessions involve patient performance on tasks whose difficulty levels are predetermined by the therapist. The system calculates individual performance indices, including reaction time, individual results, and the number of errors (Fernández, 2017). Cognitive impairments were assessed using RehaCom tests, including divided attention, selective attention, working memory, verbal memory, spatial word search, visual field, visual scanning, and logical reasoning.

Motor impairments were assessed using the Unified Parkinson's Disease Rating Scale (UPDRS). This instrument is one of the standard measures for Parkinson's disease and objectively evaluates a patient's level of disability at a specific point in time. It was developed in 1987 and consists of four sections: (1) mentation, behavior, and mood; (2) speech, handwriting, and dressing; (3) motor skills (e.g., tremor, rigidity, posture, and gait); and (4) treatment-related complications (e.g., dyskinesia) and motor fluctuations (Fahn et al., 1986). Each score reflects the disease burden on the patient and is useful for describing disease progression and treatment response over time. The maximum possible score on the scale is 195, with higher scores indicating greater disability.

The Montreal Cognitive Assessment (MoCA) is a brief and simple screening tool used to evaluate overall cognitive status. This test assesses various cognitive domains, including episodic memory, language, attention, orientation, visuospatial abilities, and executive functions, and has been introduced as an alternative to the traditional Mini-Mental State Examination.

The MoCA has a maximum score of 30. Individuals scoring 25 or higher are considered cognitively normal, whereas scores below 25 are considered abnormal and indicative of mild cognitive impairment. The psychometric properties of this test have been examined in various studies, and its validity and reliability have been confirmed (Almeida et al., 1998). In a study conducted to examine the psychometric properties of the MoCA in patients with Parkinson's disease in Isfahan, results indicated a Cronbach's alpha of 0.77, concurrent validity of 0.79, sensitivity of 0.85, and specificity of 0.90. A cutoff score of less than 25 has been proposed for the diagnosis of mild cognitive impairment.

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Autonomic function was also assessed using the Parkinson's Disease Outcomes – Autonomic Dysfunction scale (SCOPA-AUT), the validity and reliability of which have been confirmed in previous studies. This self-report scale consists of 25 items assessing gastrointestinal (7 items), urinary (6 items), cardiovascular (3 items), thermoregulatory (4 items), pupillomotor (1 item), and sexual functioning (2 items). Each item is scored from 0 (never) to 3 (often) and refers to symptoms experienced over the past month, except for the item assessing syncope/fainting, which refers to the past 6 months. The total score ranges from 0 to 75, with higher scores indicating poorer autonomic functioning (Visser et al., 2004). If patients were taking medications related to the symptoms addressed in the questionnaire, they were allowed to report their symptoms concurrently with medication use, and discontinuation of medication was not required.

Figure 1

Description of the EEG 3840 electroencephalography device

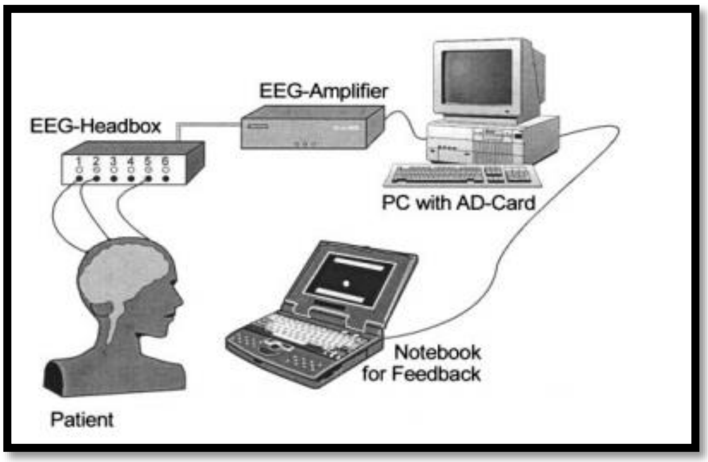
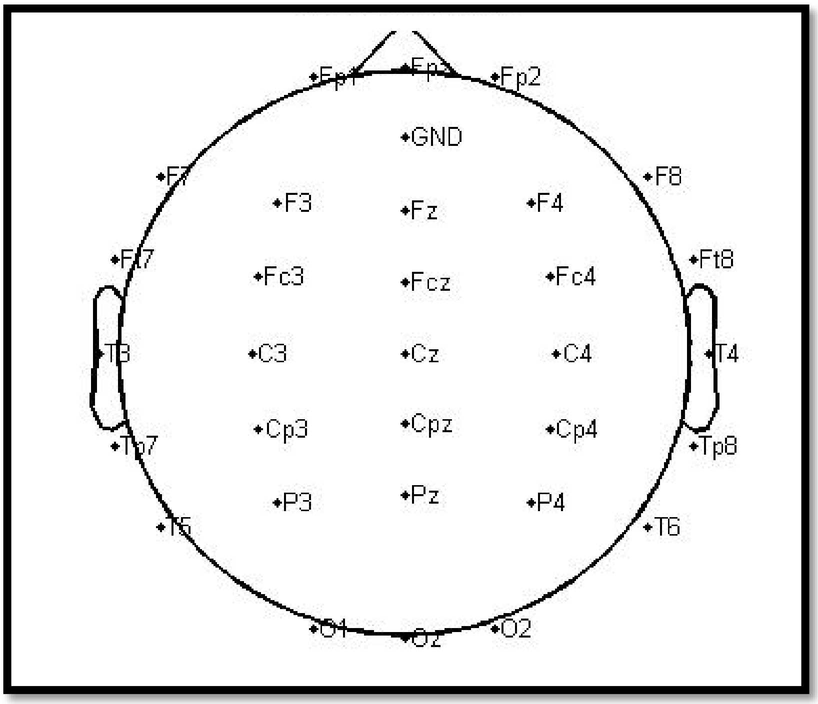


Figure 2

Placement of electroencephalography (EEG) channels



Electroencephalography (EEG) was used to examine hypotheses related to brainwave activity and to better understand the effectiveness of the intervention. Brain activity signals were measured using electrodes placed on the scalp to record the electrical activity of the brain. Electrodes were positioned to collect voltage from specific brain regions. Before electrode placement, a conductive gel was applied to reduce scalp resistance. The electrode outputs were connected to an amplifier input and then passed through high-pass and low-pass filters. Although EEG has relatively low spatial resolution, it has high temporal resolution, typically within a few milliseconds. Compared to methods such as functional magnetic resonance imaging (fMRI) and positron emission tomography (PET), EEG is more cost-effective and easier to use. Brainwave data in this study were collected using a 32-channel EEG 3840 system (Negar Andishgan Company, Iran). Electrode impedance was maintained below 5 kΩ. Signals were amplified using an EEG amplifier, filtered with a low-pass filter at 40 Hz and a time constant of 16 seconds, digitized at a sampling rate of 1024 Hz, and subsequently downsampled to 256 Hz for improved analysis.

The device included 32 channels, and electrode placement followed the international 10–20 system.

The infra-low frequency neurofeedback protocol was implemented using the Cygnet system (BEE Medic), consisting of NeuroAmp II hardware and Cygnet software integrated with Inner Tube video feedback. Various audiovisual elements changed dynamically in response to brainwave activity. The protocol was conducted using a Windows 10 operating system and a standard personal computer with a high-resolution monitor. The optimal reinforcement frequency for each participant was determined during the initial sessions based on the patient's subjective reports. Training was conducted using bipolar silver/silver chloride electrodes placed with Ten20 conductive paste at T4–P4 as active electrodes, with an electrode placed at Fpz as the reference and another at Cz as the ground, according to the standard 10–20 system. Each patient received 20 individual sessions of 30 minutes over a 5–6 week period. Electrode placement followed the neurofeedback approach developed by Othmer (2017), which involves frequency optimization based on the patient's clinical response. The T4–P4 and T3–T4 protocols are considered foundational protocols, each requiring at least 15 sessions either individually or in combination. The T4–P4 protocol is used for arousal regulation, where dysregulation may result from developmental trauma or developmental disorders. The T3–T4 protocol is used to regulate excitability, as imbalance between excitation and inhibition can lead to excessive irritability and neural instability. Simultaneous use of T4–P4 and T3–T4 protocols is recommended for individuals exhibiting both arousal dysregulation and excitability symptoms. The right posterior protocol (T4–P4) is applied to improve balance and coordination, reduce muscle tension and spasms, address hyperactivity and tremor associated with Parkinson's

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disease, and alleviate symptoms such as constipation and bruxism.

Frequency Optimization: At the outset, training began at a frequency of 0.1 mHz, and the frequency was then increased or decreased depending on the patient's reports and clinical observations.

2.3. Data analysis

Behavioral data were analyzed using the paired-samples t-test, while brain signal data were analyzed using analysis of variance (ANOVA) and post hoc tests via SPSS-28.

3. Findings and Results

Table 1 presents the descriptive characteristics of the participants in the variables of motor impairment (Unified Parkinson's Disease Rating Scale), general cognitive status (Montreal Cognitive Assessment), the cognitive components assessed by the RehaCom software, and the normality of score distributions for each variable (Shapiro–Wilk test).

Table 1

Demographic Characteristics of the Research Sample

Participant No.	Gender	Age	Marital Status	Education	Occupation	Duration of Parkinson's Disease	Dominant Hand
Patient No. 1	Male	58	Married	Primary school	Disabled / unable to work	7 years	Right
Patient No. 2	Male	59	Married	High school diploma	Retired	10 years	Right
Patient No. 3	Male	60	Married	Primary school	Retired	15 years	Right
Patient No. 4	Male	60	Married	Middle school	Retired	9 years	Right
Patient No. 5	Male	55	Married	Middle school	Disabled / unable to work	12 years	Right
Total / Mean	5 participants	58.4	—	—	—	10.6 years	—

Table 2 shows the mean and standard deviation of age and the study variables among the participants in the present study. Based on these findings, the status of the participants in motor impairment, cognitive status, and autonomic system functioning improved across the research phases. In addition, the data collected for all three aforementioned variables showed a normal distribution. Furthermore, the Z scores and percentile ranks of each participant in the cognitive assessment tests of the RehaCom software are presented in the continuation of Table 2.

Table 2

Descriptive Characteristics of Participants in the Research Variables

Variable / Instrument	Phase	n	Mean	SD	Normality
Unified Parkinson's Disease Rating Scale – Motor impairment	Pretest 1	5	20.33	8.11	0.895
	Pretest 2	5	20.22	7.08	0.824
	Posttest	5	12.40	4.56	0.918
	Follow-up	5	9.80	2.95	0.810
Montreal Cognitive Assessment – General cognitive status	Pretest 1	5	21.60	1.53	0.902
	Pretest 2	5	24.00	1.64	0.957
	Posttest	5	25.40	0.92	0.842
	Follow-up	5	25.80	1.32	0.831
Parkinson's Disease Outcomes – Autonomic Dysfunction Scale – Autonomic system assessment	Pretest 1	5	19.20	3.56	0.792
	Pretest 2	5	21.00	2.91	0.776
	Posttest	5	16.40	1.67	0.881
	Follow-up	5	16.68	1.51	0.894

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RehaCom Computerized Tests: Percentile Ranks and Z Scores

Computerized Test	Participant	Pre-1	Pre-2	Post	Follow-up
Alertness without signal	Patient No. 1	53% (Z = 0.07)	70% (Z = 0.54)	19% (Z = -0.89)	21% (Z = -0.80)
	Patient No. 2	82% (Z = 0.91)	85% (Z = 1.08)	85% (Z = 1.05)	58% (Z = 0.20)
	Patient No. 3	0.9% (Z = -2.35)	5% (Z = -1.58)	42% (Z = -0.21)	78% (Z = 0.76)
	Patient No. 4	77% (Z = 0.77)	48% (Z = -0.05)	0.01% (Z = 0.33)	68% (Z = 0.48)
	Patient No. 5	42% (Z = -0.19)	0.02% (Z = -2.80)	53% (Z = -4.79)	24% (Z = -0.71)
Alertness with signal	Patient No. 1	62% (Z = 0.30)	68% (Z = 0.46)	18% (Z = -0.90)	13% (Z = -1.12)
	Patient No. 2	68% (Z = 0.48)	82% (Z = 0.93)	84% (Z = 0.99)	56% (Z = 0.15)
	Patient No. 3	18% (Z = -0.90)	17% (Z = -0.94)	59% (Z = 0.22)	78% (Z = 0.77)
	Patient No. 4	81% (Z = 0.90)	26% (Z = -0.63)	50% (Z = 0.00)	42% (Z = -0.21)
	Patient No. 5	42% (Z = -0.21)	0.01% (Z = -3.02)	1.5% (Z = -2.17)	20% (Z = -0.84)
Selective attention (reaction speed)	Patient No. 1	67% (Z = 0.43)	36% (Z = -0.34)	40% (Z = -0.24)	60% (Z = 0.25)
	Patient No. 2	68% (Z = 0.47)	73% (Z = 0.63)	87% (Z = 1.11)	72% (Z = 0.59)
	Patient No. 3	46% (Z = -0.09)	42% (Z = -0.21)	62% (Z = 0.31)	68% (Z = 0.47)
	Patient No. 4	55% (Z = 0.14)	26% (Z = 0.64)	37% (Z = -0.32)	24% (Z = -0.71)
	Patient No. 5	25% (Z = -0.68)	0.04% (Z = -2.64)	11% (Z = -1.24)	19% (Z = -0.88)
Selective attention (response control)	Patient No. 1	9% (Z = -1.29)	9% (Z = -1.29)	0.04% (Z = -3.34)	74% (Z = 0.65)
	Patient No. 2	17% (Z = -0.96)	0.01% (Z = -4.51)	1.6% (Z = -2.14)	35% (Z = -0.37)
	Patient No. 3	63% (Z = 0.34)	81% (Z = 0.90)	22% (Z = -0.77)	42% (Z = -0.21)
	Patient No. 4	12% (Z = -1.18)	26% (Z = -0.64)	13% (Z = -1.10)	48% (Z = -0.05)
	Patient No. 5	2% (Z = -1.94)	5% (Z = 0.50)	9% (Z = -1.29)	42% (Z = -0.21)
Divided attention (auditory)	Patient No. 1	69% (Z = 0.50)	69% (Z = 0.50)	69% (Z = 0.50)	69% (Z = 0.50)
	Patient No. 2	72% (Z = 0.60)	72% (Z = 0.60)	72% (Z = 0.60)	10% (Z = -1.26)
	Patient No. 3	74% (Z = 0.65)	74% (Z = 0.65)	14% (Z = -1.06)	74% (Z = 0.65)
	Patient No. 4	75% (Z = 0.68)	45% (Z = -0.12)	46% (Z = -0.09)	75% (Z = 0.69)
	Patient No. 5	69% (Z = 0.50)	69% (Z = 0.50)	69% (Z = 0.50)	34% (Z = -0.42)
Divided attention (visual)	Patient No. 1	68% (Z = 0.48)	68% (Z = 0.48)	68% (Z = 0.48)	68% (Z = 0.48)
	Patient No. 2	18% (Z = -0.90)	73% (Z = 0.62)	44% (Z = -0.14)	44% (Z = -0.14)
	Patient No. 3	75% (Z = 0.69)	75% (Z = 0.69)	49% (Z = -0.02)	75% (Z = 0.69)
	Patient No. 4	77% (Z = 0.73)	77% (Z = 0.73)	77% (Z = 0.75)	77% (Z = 0.75)
	Patient No. 5	68% (Z = 0.48)	68% (Z = 0.48)	68% (Z = 0.48)	68% (Z = 0.48)
Spatial number search (processing speed)	Patient No. 1	0.01% (Z = -5.00)	0.01% (Z = -5.00)	0.01% (Z = -5.00)	0.01% (Z = -5.00)
	Patient No. 2	0.01% (Z = -5.00)	0.01% (Z = -5.00)	0.01% (Z = -5.00)	0.01% (Z = -5.00)
	Patient No. 3	0.01% (Z = -5.00)	0.01% (Z = -5.00)	0.01% (Z = -5.00)	0.01% (Z = -5.00)
	Patient No. 4	0.01% (Z = -5.00)	0.01% (Z = -5.00)	0.01% (Z = -3.84)	0.01% (Z = -2.96)
	Patient No. 5	0.01% (Z = -5.00)	0.01% (Z = -5.00)	0.01% (Z = -5.00)	0.01% (Z = -5.00)
Spatial number search (attention)	Patient No. 1	1.7% (Z = -2.11)	93% (Z = 1.48)	98% (Z = 2.28)	96% (Z = 1.83)
	Patient No. 2	99% (Z = 2.62)	57% (Z = 0.17)	97% (Z = 2.01)	99% (Z = 3.65)
	Patient No. 3	100% (Z = 5.00)	99% (Z = 3.35)	95% (Z = 1.69)	71% (Z = 0.55)
	Patient No. 4	100% (Z = 5.00)	99% (Z = 2.39)	97% (Z = 1.92)	6.1% (Z = -1.54)
	Patient No. 5	95% (Z = 1.70)	98% (Z = 2.28)	65% (Z = 0.40)	44% (Z = -0.16)
Spatial number search (neglect)	Patient No. 1	31% (Z = -0.48)	4.4% (Z = -1.70)	1.2% (Z = -2.25)	0.01% (Z = -5.00)
	Patient No. 2	87% (Z = 1.12)	46% (Z = -0.10)	29% (Z = -0.54)	63% (Z = 0.33)
	Patient No. 3	25% (Z = -0.68)	4% (Z = -1.69)	79% (Z = 0.81)	33% (Z = -0.44)
	Patient No. 4	33% (Z = -0.45)	0.6% (Z = -2.49)	2.6% (Z = -1.93)	77% (Z = 0.75)
	Patient No. 5	0.01% (Z = -5.00)	64% (Z = 0.36)	0.01% (Z = -4.30)	48% (Z = -0.06)
Logical reasoning	Patient No. 1	38% (Z = -0.31)	0.2% (Z = -2.86)	0.2% (Z = -2.86)	0.9% (Z = -2.35)
	Patient No. 2	0.3% (Z = -2.72)	1.2% (Z = -2.25)	0.3% (Z = -2.72)	1.2% (Z = -2.25)
	Patient No. 3	14% (Z = -1.06)	61% (Z = 0.27)	27% (Z = -0.61)	27% (Z = -0.61)
	Patient No. 4	19% (Z = -0.88)	32% (Z = -0.46)	5% (Z = -1.64)	21% (Z = -0.80)
	Patient No. 5	0.2% (Z = -2.86)	0.1% (Z = -3.37)	0.9% (Z = -2.35)	9.1% (Z = -1.33)
Working memory capacity	Patient No. 1	3.2% (Z = -1.85)	3.2% (Z = -1.85)	3.2% (Z = -1.85)	23% (Z = -0.72)
	Patient No. 2	33% (Z = -0.43)	0.1% (Z = -3.78)	33% (Z = -0.43)	75% (Z = 0.68)
	Patient No. 3	0.4% (Z = -2.60)	6.8% (Z = -1.49)	6.8% (Z = -1.49)	35% (Z = -0.38)
	Patient No. 4	35% (Z = -0.38)	0.4% (Z = -2.60)	35% (Z = -0.38)	77% (Z = 0.73)
	Patient No. 5	3.2% (Z = -1.85)	0.1% (Z = -2.98)	23% (Z = -0.72)	4% (Z = -1.74)
Visual field deficit (left)	Patient No. 1	19% (Z = -0.86)	35% (Z = -0.29)	50% (Z = 0.00)	50% (Z = 0.00)
	Patient No. 2	50% (Z = 0.00)	44% (Z = -0.14)	50% (Z = 0.00)	38% (Z = -0.29)
	Patient No. 3	33% (Z = -0.43)	33% (Z = -0.43)	44% (Z = -0.14)	44% (Z = -0.14)

	Patient No. 4	50% (Z = 0.00)	0.01% (Z = -4.14)	50% (Z = 0.00)	44% (Z = -0.14)
	Patient No. 5	50% (Z = 0.00)	50% (Z = 0.00)	19% (Z = -0.86)	9.8% (Z = -1.29)
Visual field deficit (right)	Patient No. 1	50% (Z = 0.00)	50% (Z = 0.00)	50% (Z = 0.00)	50% (Z = 0.00)
	Patient No. 2	50% (Z = 0.00)	50% (Z = 0.00)	50% (Z = 0.00)	50% (Z = 0.00)
	Patient No. 3	50% (Z = 0.00)	50% (Z = 0.00)	50% (Z = 0.00)	50% (Z = 0.00)
	Patient No. 4	50% (Z = 0.00)	38% (Z = -0.29)	38% (Z = -0.29)	38% (Z = -0.29)
	Patient No. 5	50% (Z = 0.00)	38% (Z = -0.29)	50% (Z = 0.00)	28% (Z = -0.57)
Visual field deficit (overall)	Patient No. 1	44% (Z = -0.14)	44% (Z = -0.14)	50% (Z = 0.00)	50% (Z = 0.00)
	Patient No. 2	50% (Z = 0.00)	50% (Z = 0.00)	50% (Z = 0.00)	50% (Z = 0.00)
	Patient No. 3	38% (Z = -0.29)	44% (Z = -0.14)	50% (Z = 0.00)	38% (Z = -0.29)
	Patient No. 4	50% (Z = 0.00)	33% (Z = -0.43)	38% (Z = -0.29)	50% (Z = 0.00)
	Patient No. 5	38% (Z = -0.29)	24% (Z = -0.71)	38% (Z = -0.29)	5.8% (Z = -1.57)

To facilitate a better understanding of the trend of change in each participant across the cognitive variables assessed by the RehaCom software, each of the evaluated indices is presented below in graphical form.

Table 3

Comparison of Unified Parkinson's Disease Rating Scale Results Across Research Phases

Instrument	Variable	Comparison	Test	Statistic	p	Effect Size
Unified Parkinson's Disease Rating Scale	Motor impairment	Pretest 1 vs. Pretest 2	Paired-samples t-test	2.44	0.071	—
		Pretest 2 vs. Posttest	Paired-samples t-test	2.38	0.075	—
		Posttest vs. Follow-up	Paired-samples t-test	1.39	0.238	—
		Pretest 1 vs. Follow-up	Paired-samples t-test	4.12	0.015*	d = 0.31

Based on the results in Table 3, although participants' scores on the Unified Parkinson's Disease Rating Scale decreased across the phases of the study, indicating improvement in their motor problems, no significant difference was observed between Pretest 2 and Posttest. At the follow-up stage, after completion of the intervention, the severity of the participants' motor problems had not returned to the pre-intervention level, and no significant difference was found between Posttest and Follow-up. Ultimately, the difference between Follow-up and Pretest 1 was statistically significant, indicating the positive effect of infra-low frequency neurofeedback on motor impairments in individuals with Parkinson's disease. The calculated effect size was 0.31. It appears that a longer period of time may be needed for the effects of the intervention to become apparent, and that these effects may not be immediately observable after the intervention, or that a longer course of treatment may be required to produce a substantial effect.

Table 4

Comparison of Montreal Cognitive Assessment Results Across Research Phases

Instrument	Variable	Comparison	Test	Statistic	p	Effect Size
Montreal Cognitive Assessment	Cognitive status	Pretest 1 vs. Pretest 2	Paired-samples t-test	-1.17	0.305	—
		Pretest 2 vs. Posttest	Paired-samples t-test	-0.953	0.395	—
		Posttest vs. Follow-up	Paired-samples t-test	-0.667	0.541	—
		Pretest 1 vs. Follow-up	Paired-samples t-test	-7.20	0.002**	d = -0.90

Based on the results in Table 4, although participants' scores on the Montreal Cognitive Assessment increased across the phases of the study, indicating improvement in their cognitive problems, no significant difference was observed between Pretest 2 and Posttest. At the follow-up stage, after completion of the intervention, the severity of the participants' cognitive problems had not returned to the pre-intervention level, and no significant difference was found between Posttest and Follow-up. Ultimately, the difference between Follow-up and Pretest 1 was statistically significant, indicating the positive effect of infra-low frequency neurofeedback on the cognitive problems of individuals with Parkinson's disease. The calculated effect size was -0.90, indicating a substantial intervention effect at follow-up compared with pretest. It appears that a longer period of time may be needed for the effects of the intervention to emerge, and that these effects may not be immediately observable after the intervention, or that a longer course of treatment may be required to produce a substantial effect.

This study analyzed brainwave patterns over time. Different scores at different time points were reported per second for ten brainwave rhythms. These values illustrate the changes in the participants' brainwaves associated with the effects of neurofeedback across the course of treatment. As stated in the research design, participants underwent brainwave recording on four occasions, and their brainwaves were recorded in the resting state. In the first stage, brainwave recording was performed two weeks before the start of treatment, and the next stage occurred immediately before the beginning of treatment. Using within-group t-tests, these two stages were compared. The t-tests conducted across channels and absolute power showed no significant differences between the two pretest phases. As predicted, no changes had occurred over time for the participants before treatment. In the next stage, Pretest 1 before the start of treatment was compared with the Posttest after completion of the intervention phase.

Table 5

Comparison of Pretest 2 and Posttest in Absolute Power Across Channels

Channel No.	Absolute Power Beta t	Absolute Power Beta p	Absolute Power Beta1 t	Absolute Power Beta1 p	Absolute Power Gamma t	Absolute Power Gamma p
P3	-3.08159	0.020426	-2.46809	0.057271	-2.81131	0.010726
P4	-3.10110	0.019857	-2.37777	0.065048	-3.45064	0.002803
C3	-3.19234	0.018719	-2.57425	0.051403	-3.17355	0.005060
C4	-2.41577	0.055088	-1.74792	0.148465	-3.34675	0.003953
F3	-1.97170	0.101397	-1.50936	0.199490	-3.15247	0.005538
F4	-2.77247	0.028433	-2.34037	0.063823	-3.16865	0.004975
FP1	-3.03060	0.022998	-2.57964	0.049704	-2.32910	0.032774
FP2	-2.82766	0.030327	-2.30192	0.071790	-3.01131	0.008388
Cz	-2.81703	0.026072	-2.29136	0.068814	-3.14008	0.005381
T3	-3.09564	0.022759	-2.51350	0.056386	-3.51751	0.002497
T4	-2.91211	0.023632	-2.32592	0.066649	-3.58936	0.002078
F7	-2.99707	0.024579	-2.50251	0.055469	-3.39850	0.003566
F8	-2.71821	0.030805	-2.24601	0.072723	-2.82200	0.011598
O1	-2.90575	0.025237	-2.39838	0.060748	-3.76044	0.001791
O2	-2.98566	0.021486	-2.39045	0.061214	-3.91744	0.001095
Fz	-2.94925	0.023063	-2.51600	0.051651	-3.62424	0.002139
T5	-3.16018	0.017870	-2.41045	0.061821	-3.17172	0.005481
T6	-3.08699	0.018637	-2.27651	0.073128	-3.24853	0.004536
Pz	-3.11305	0.018727	-2.57103	0.048937	-3.68438	0.002751

Table 5 examines the pretest–posttest results of neurofeedback treatment for the absolute power of beta and gamma waves in each channel. Overall, the results indicate that in many channels, the absolute power of beta and gamma waves decreased after neurofeedback treatment. For example, in channel P3, the t-score for absolute beta power was -3.08159 and for absolute gamma power was -2.81131, indicating that after neurofeedback treatment, the absolute power of beta and gamma waves in this channel decreased, and these changes were significant ($p < .05$). Similarly, significant changes in the absolute power of beta and gamma waves after neurofeedback treatment were observed in many other channels.

Therefore, this table indicates that neurofeedback treatment may reduce the absolute power of beta and gamma waves in many channels. Figure 3 also shows a comparison of changes in absolute beta wave power across different conditions, which may contribute to a better understanding of the findings. Overall, this table and figure may provide useful information for researchers and specialists in the field of neurofeedback and inhibitory brain control. These results also suggest that using brain activity analysis based on brain signals may be an effective method for studying the effect of neurofeedback treatment on the absolute power of beta and gamma waves. Figure below also presents a comparison of beta across different conditions.

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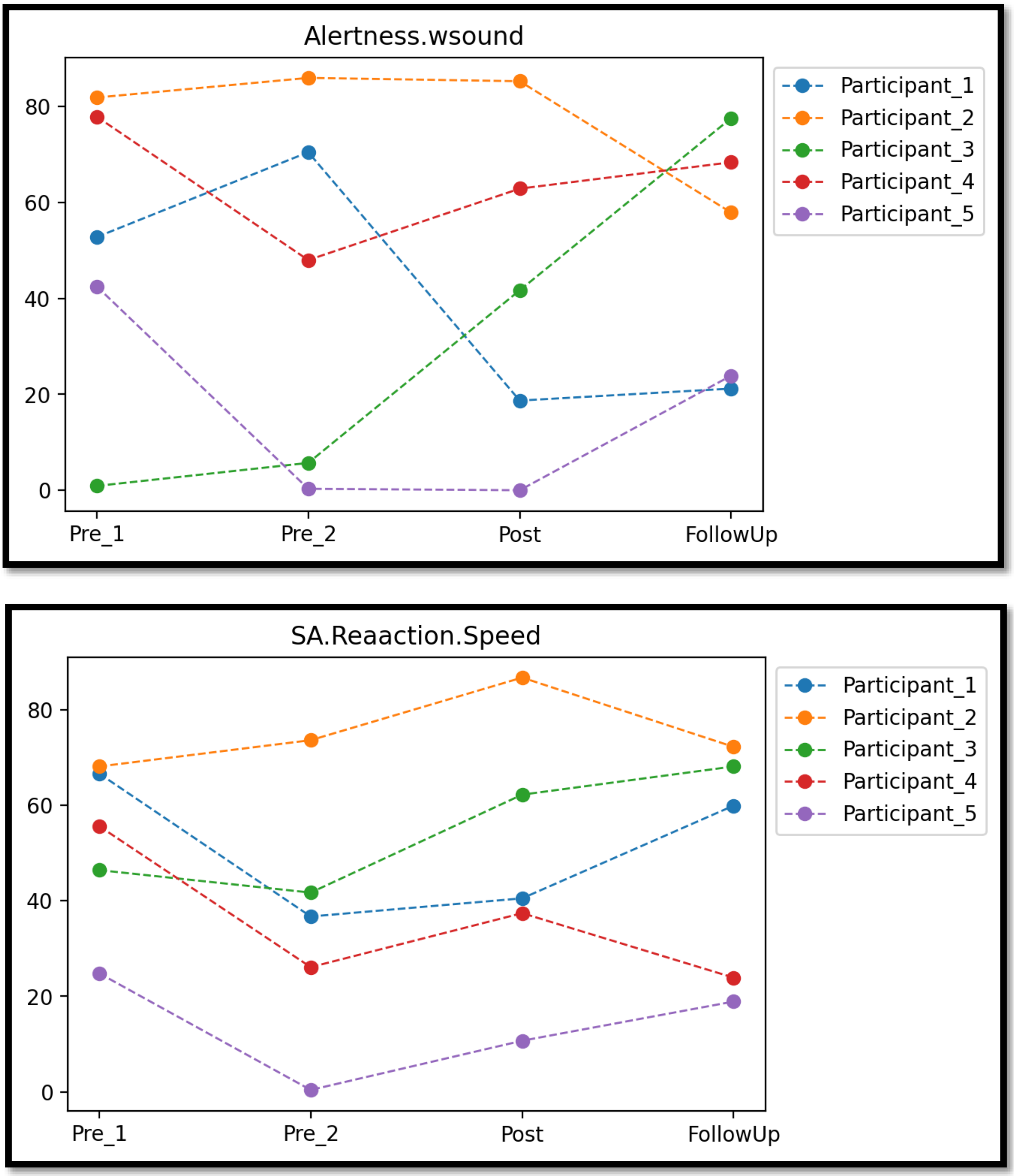
Figure 3

Trends of Changes and Comparison of changes in absolute beta wave power across different conditions.



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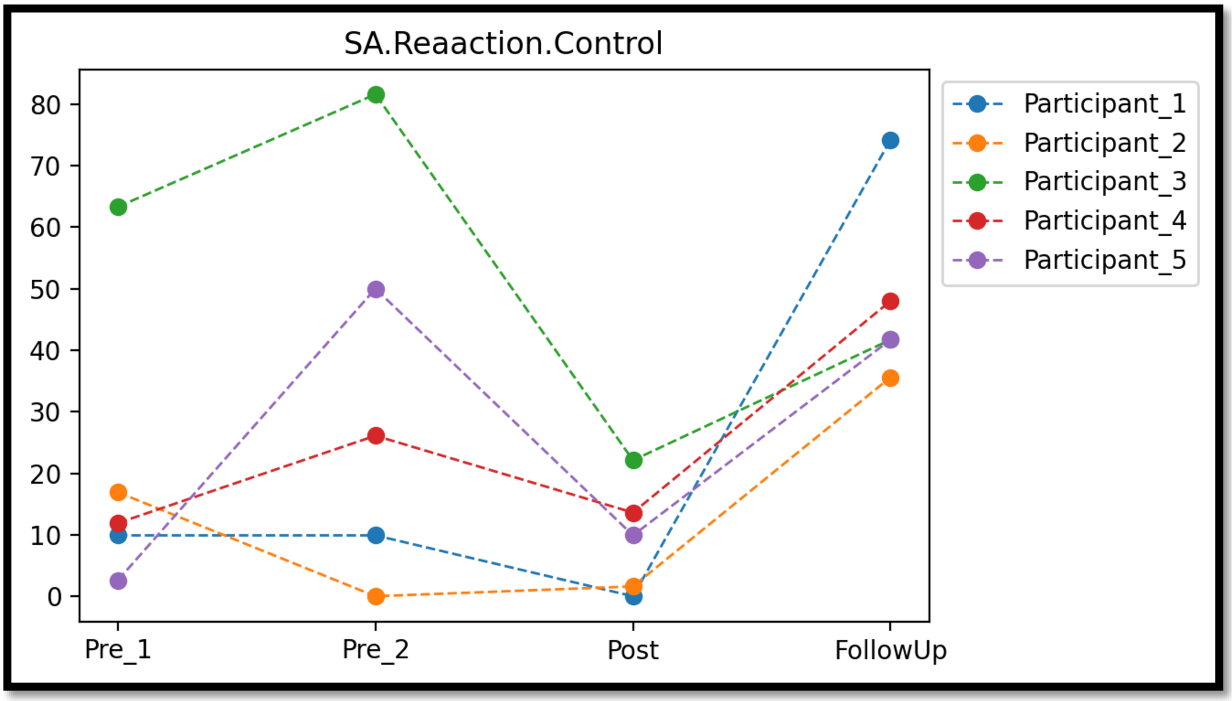
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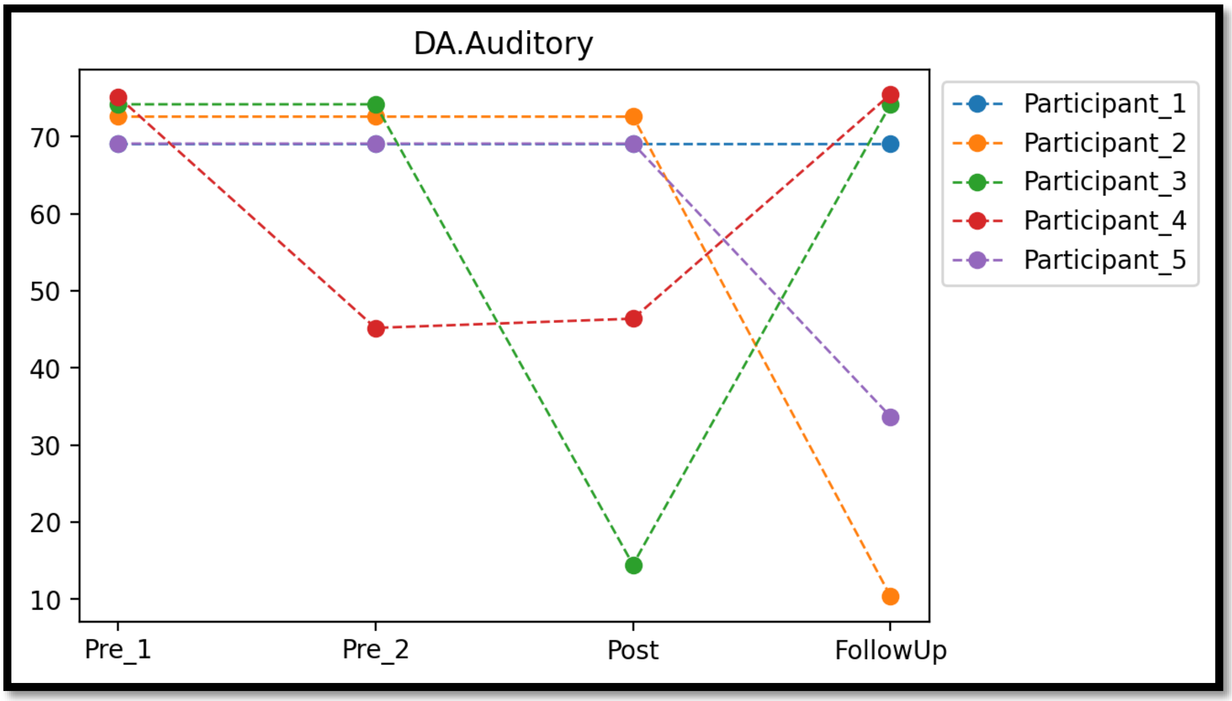
Participant_5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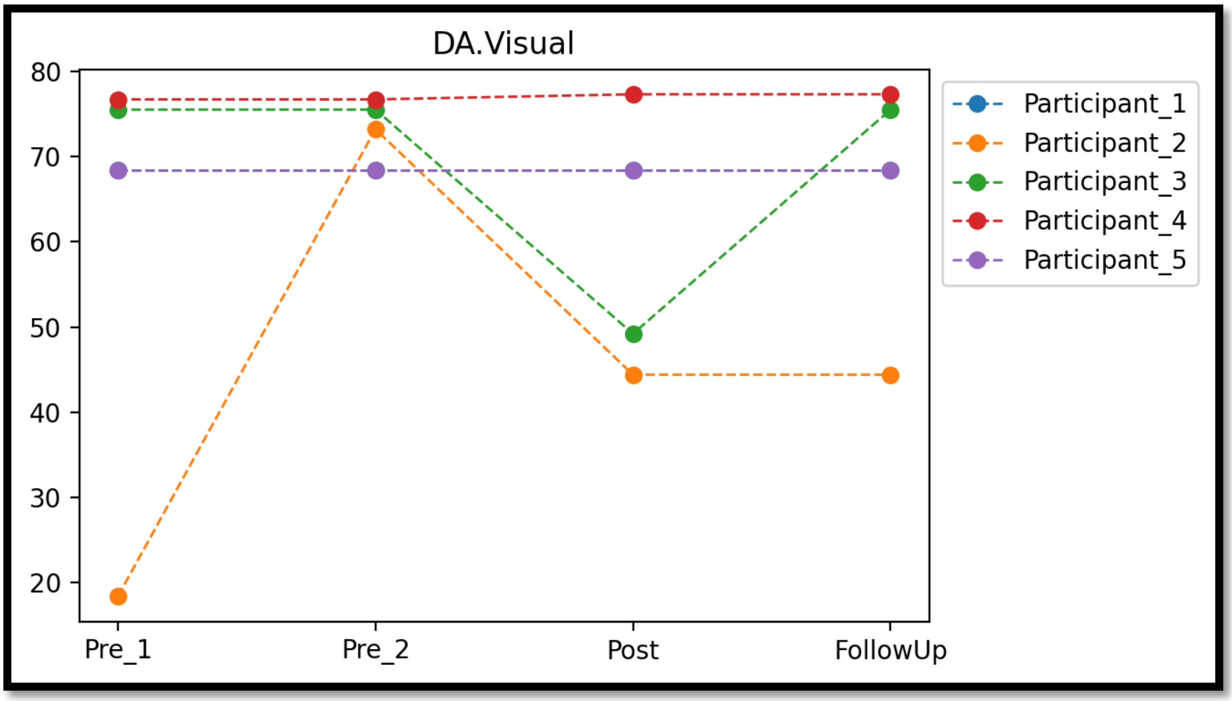
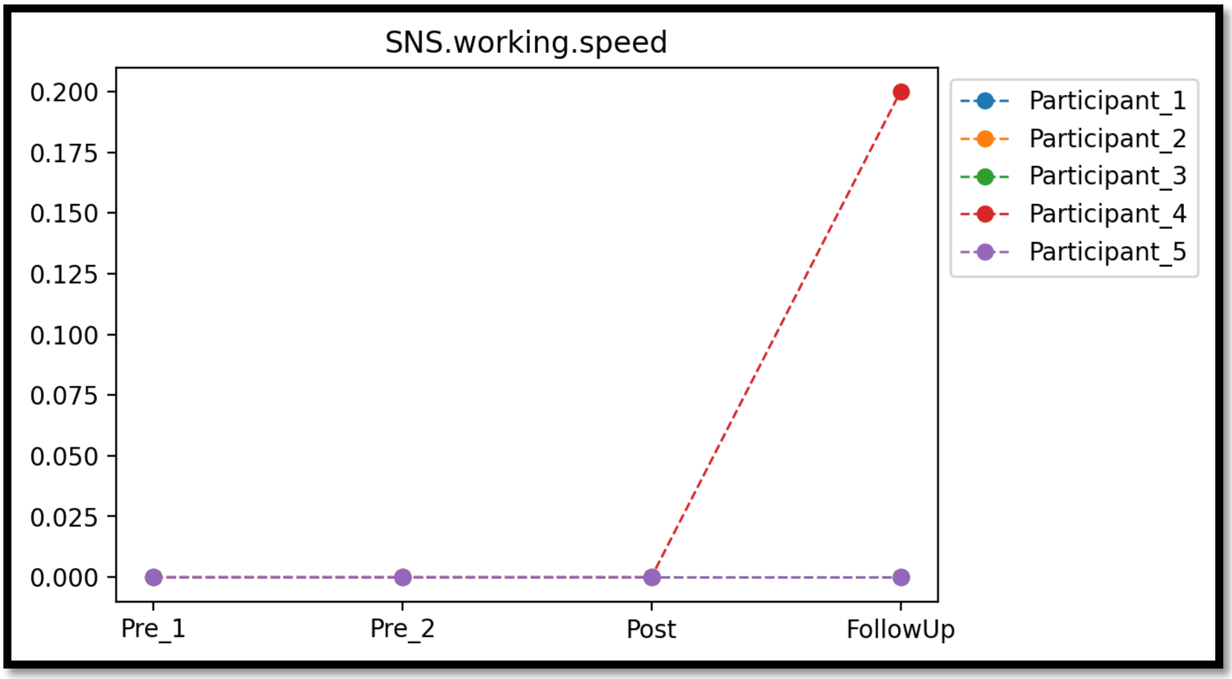
Participant_5





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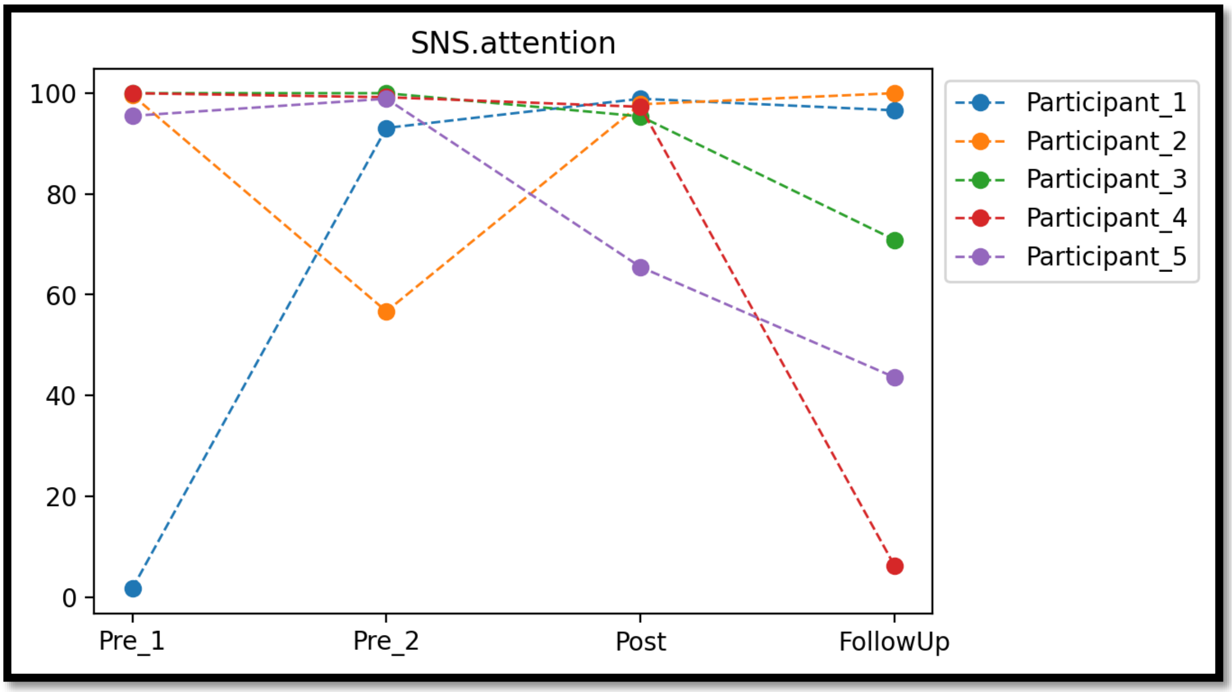

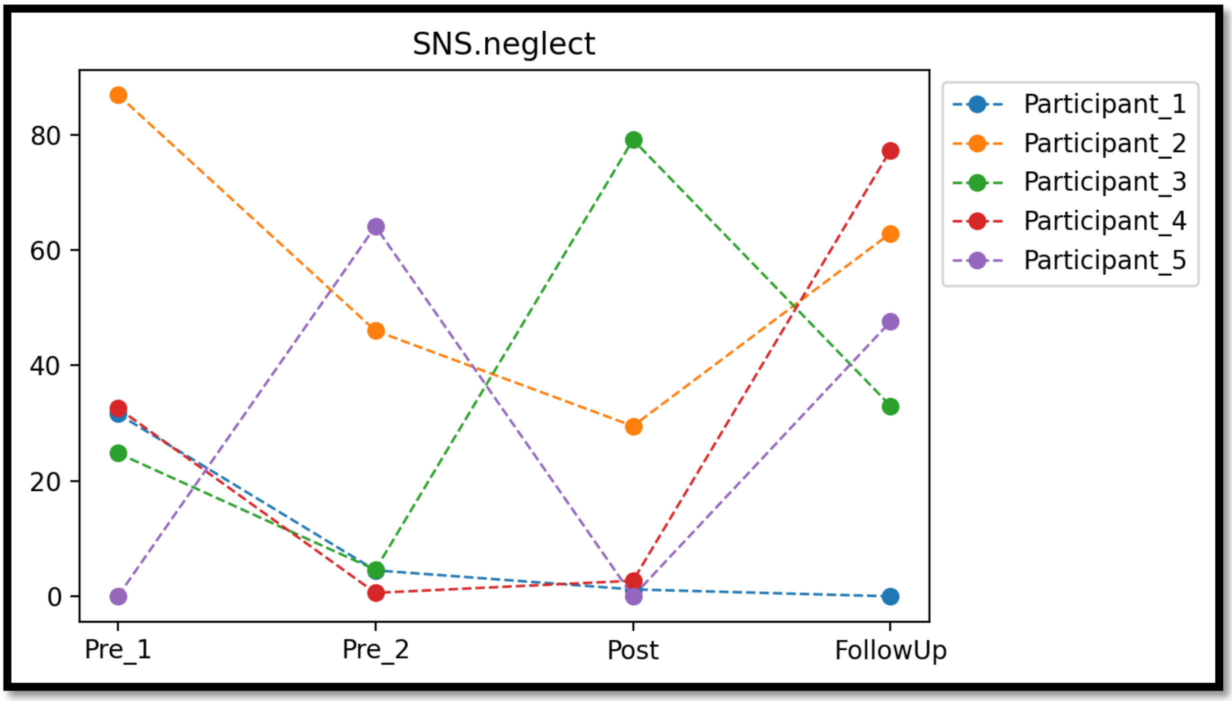

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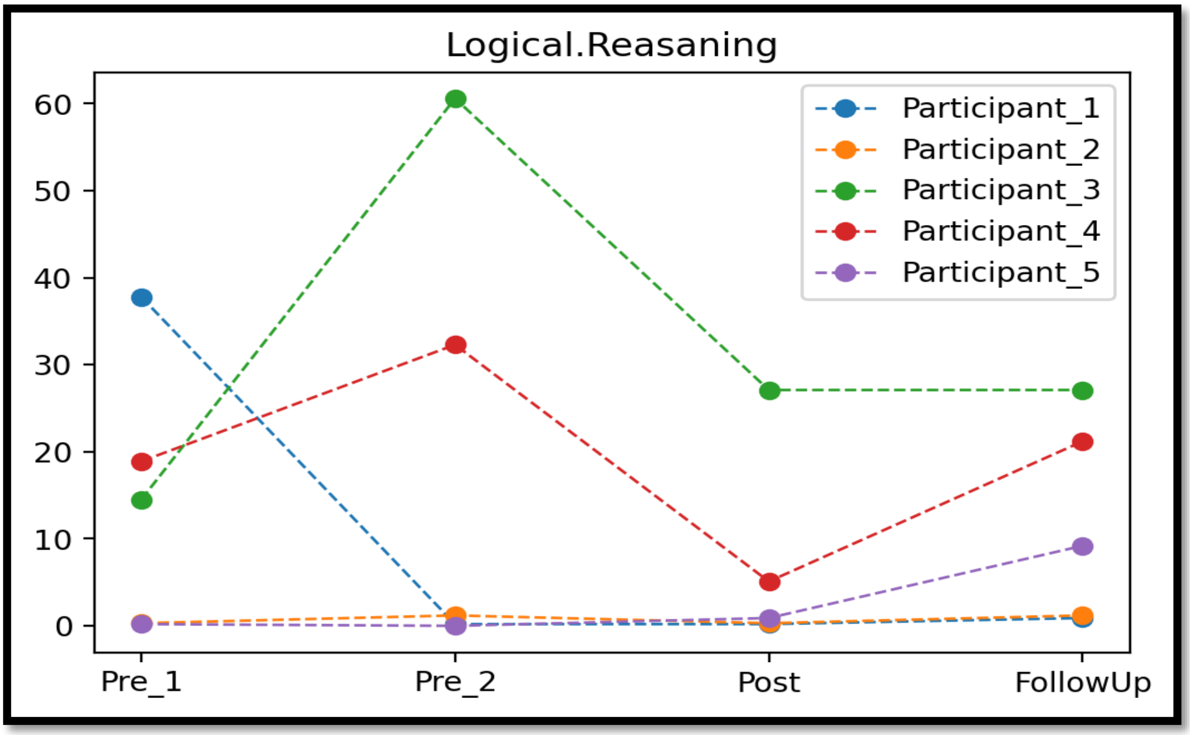
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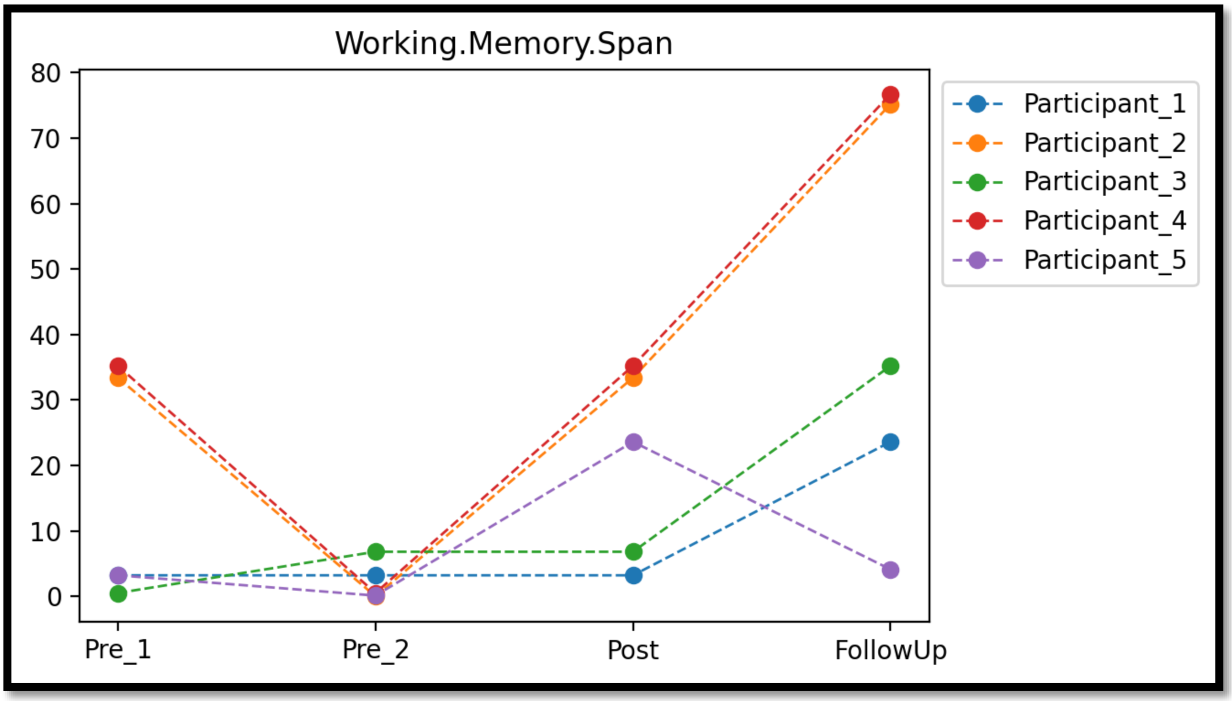
Participant_4
Participant_5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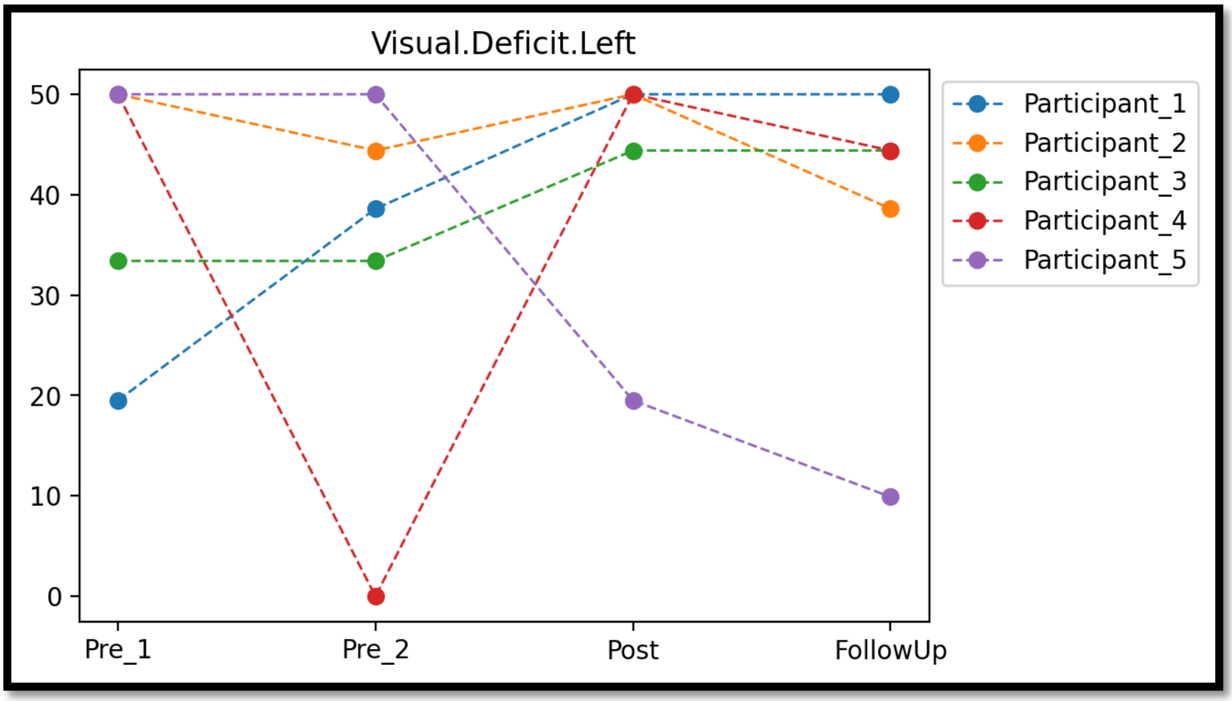
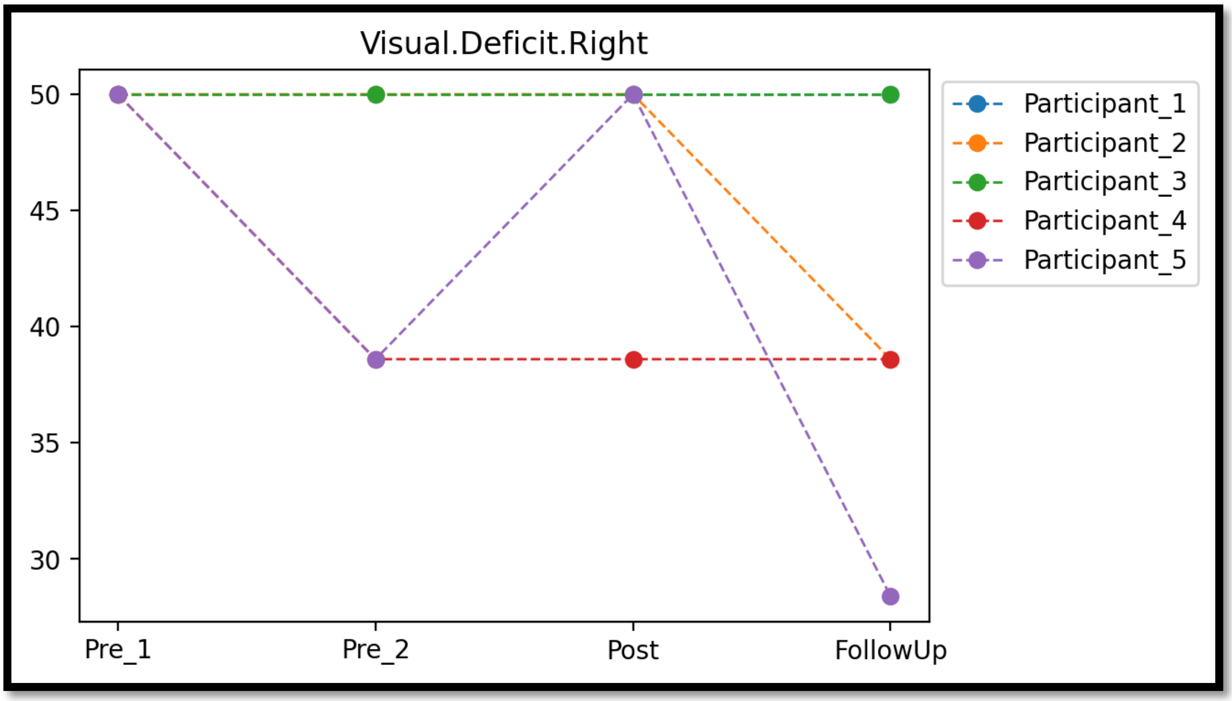
Participant_5





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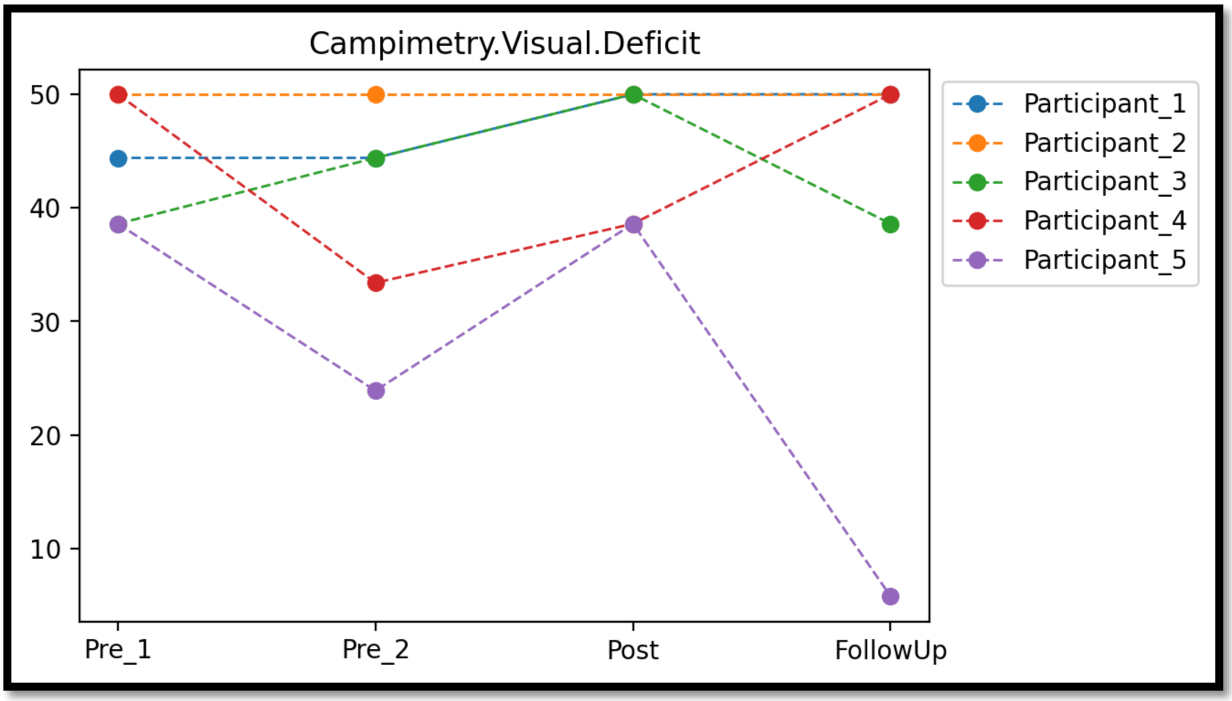

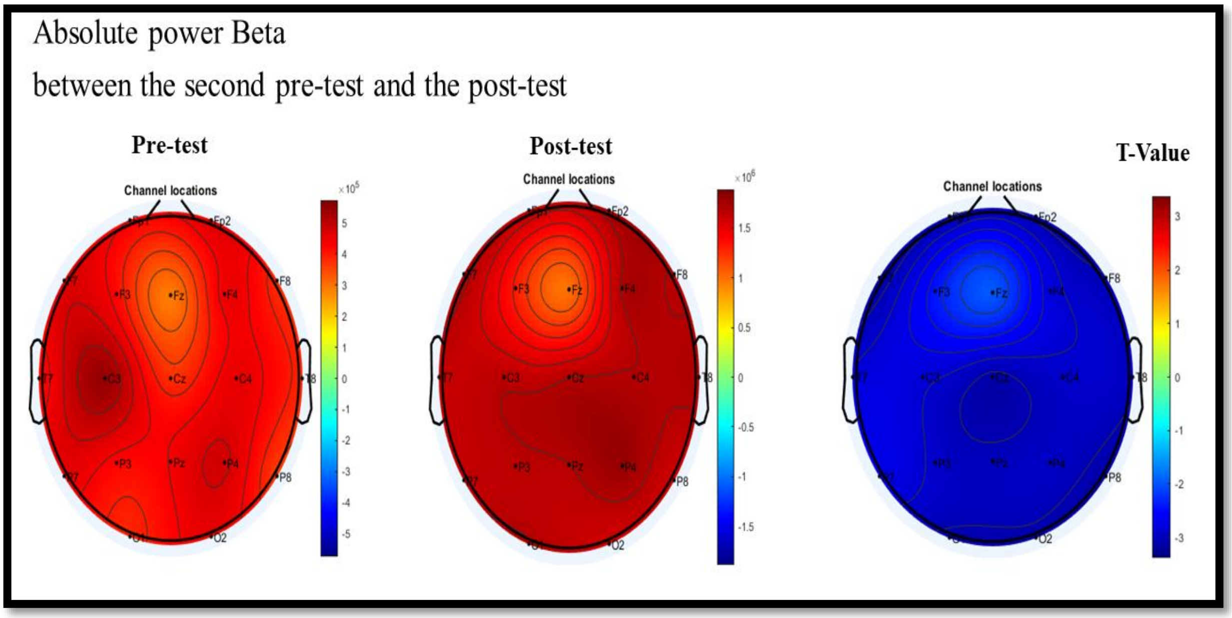

Figure 3 also presents a comparison of changes in absolute beta wave power across different conditions, which may facilitate better understanding. Overall, this table and figure may provide useful information for researchers and specialists in the field of neurofeedback and inhibitory brain control. Figure 4 shows a comparison of changes in absolute beta1 wave power across different conditions.

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Figure 4

Comparison of changes in absolute beta1 wave power across different conditions.

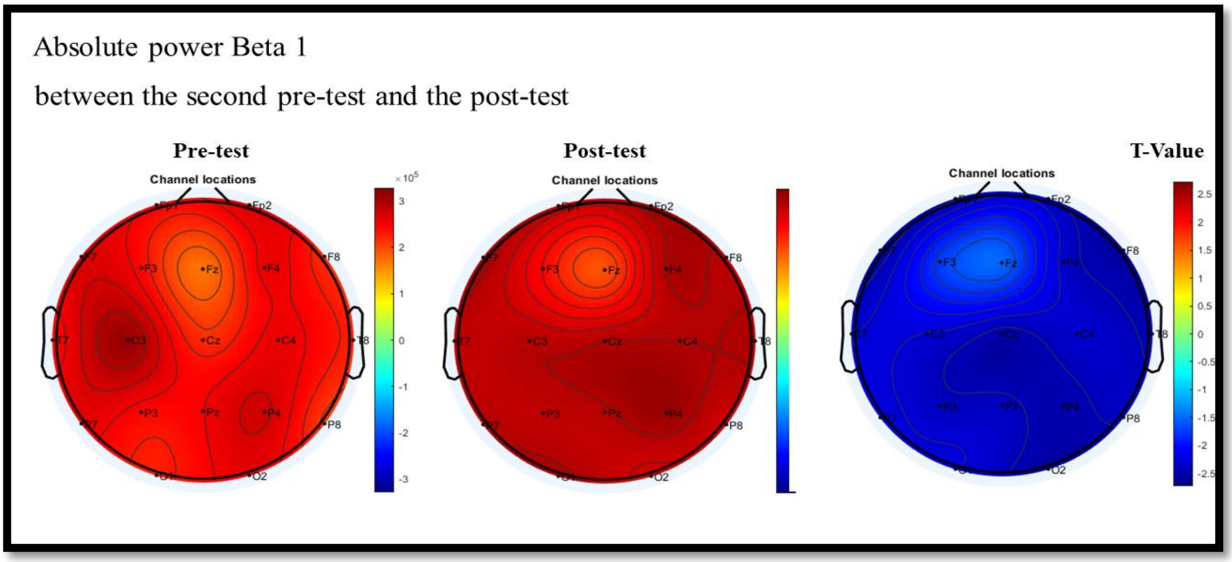

Figure 4 also shows a comparison of changes in absolute beta1 wave power across different conditions, which may facilitate better understanding. Overall, this table and figure may provide useful information for researchers and specialists in the field of neurofeedback and inhibitory brain control. Figure 5 shows a comparison of absolute gamma power across different conditions.

Figure 5

Comparison of absolute gamma wave power across different conditions.

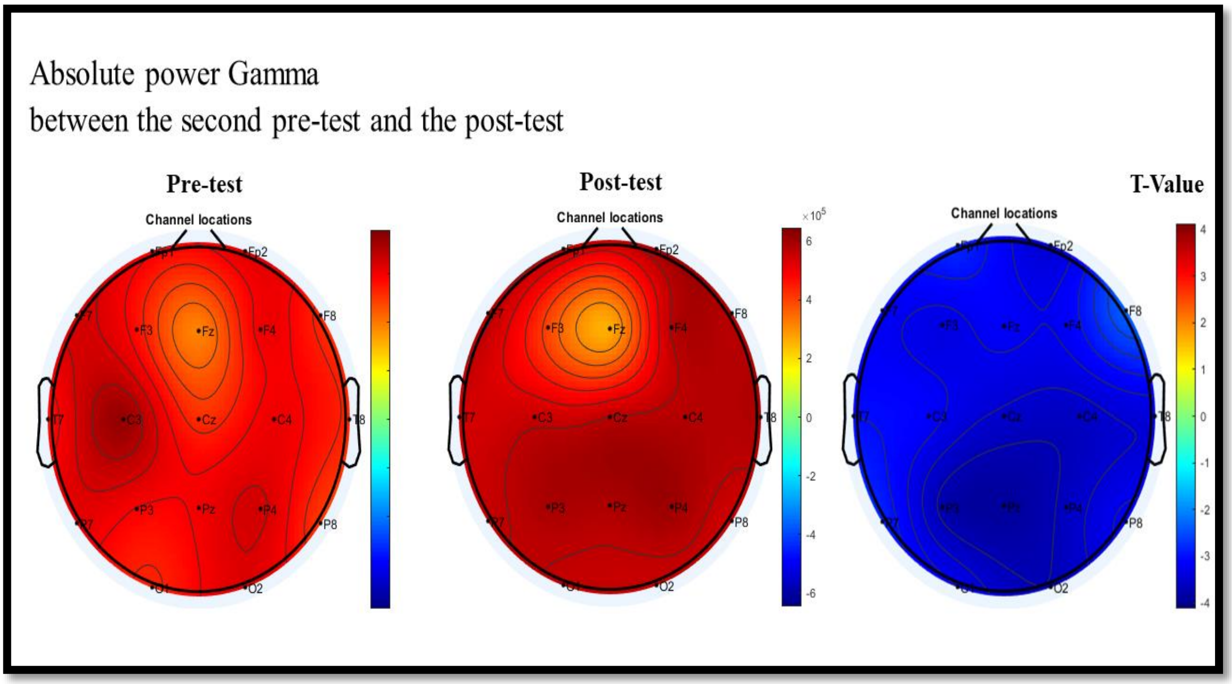

Figure 5 also shows a comparison of changes in absolute gamma wave power across different conditions, which may facilitate better understanding. Overall, this table and figure may provide useful information for researchers and specialists in the field of neurofeedback and inhibitory brain control.

In the next stage, the comparison between the posttest and follow-up phases was examined. Based on this comparison, no significant difference was observed between these two stages.

In the final stage, the comparison between pretest and follow-up was conducted, and the results are reported in Table 6.

Table 6

Within-Group t-Test Comparison Between Pretest 2 and Follow-up in Absolute Power Components

Channel No.	Absolute Power Beta t	Absolute Power Beta p	Absolute Power Gamma t	Absolute Power Gamma p
P4	-2.16751	0.045759	—	—
C4	-2.88855	0.008903	—	—
F3	-2.10984	0.046712	—	—
Cz	-2.36012	0.027828	-2.66364	0.014234
T3	-2.22110	0.036965	-2.05868	0.051726
O1	-2.73560	0.012160	-2.50868	0.020590
O2	-2.13199	0.044422	-3.03961	0.007078
Fz	—	—	-2.00082	0.057924
Pz	-2.45974	0.022747	—	—

Table 6 presents the within-group t-test comparison between Pretest 2 and Follow-up in the absolute power components. Based on this comparison, even two weeks after the end of the intervention, significant differences were still present in the specified regions.

This table shows that at the follow-up stage (two weeks after the end of the intervention), significant differences in the absolute power of beta and gamma waves were still present in some channels. Overall, in channels C4, F3, Cz, T3, O1, O2, and Pz, significant differences were observed in the absolute power of beta and gamma waves between Pretest 2 and Follow-up. For example, in channel C4, the t-score for absolute beta power was -2.88855 and the corresponding p-value was 0.008903, indicating a significant difference in the absolute power of beta waves between these two stages ($p < .05$). Likewise, in channel O2, a significant difference in absolute gamma power was observed between the two stages ($p < .01$).

Therefore, this table shows that even two weeks after the completion of the neurofeedback intervention, significant differences in the absolute power of beta and gamma waves were still present in some channels. These findings may provide useful information for researchers and specialists in the field of neurofeedback and inhibitory brain control and suggest that the effect of neurofeedback treatment on the absolute power of beta and gamma waves may remain stable for a longer period and warrants further follow-up. These findings also suggest that using brain activity analysis based on brain signals in neurofeedback studies may serve as an effective method for examining the effect of neurofeedback treatment on inhibitory brain control and improvement of individuals' cognitive and neurological functioning. Figure 6 shows a comparison of absolute beta power in Pretest 2 and Follow-up.

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Figure 6

Comparison of absolute beta wave power in Pretest 2 and Follow-up.

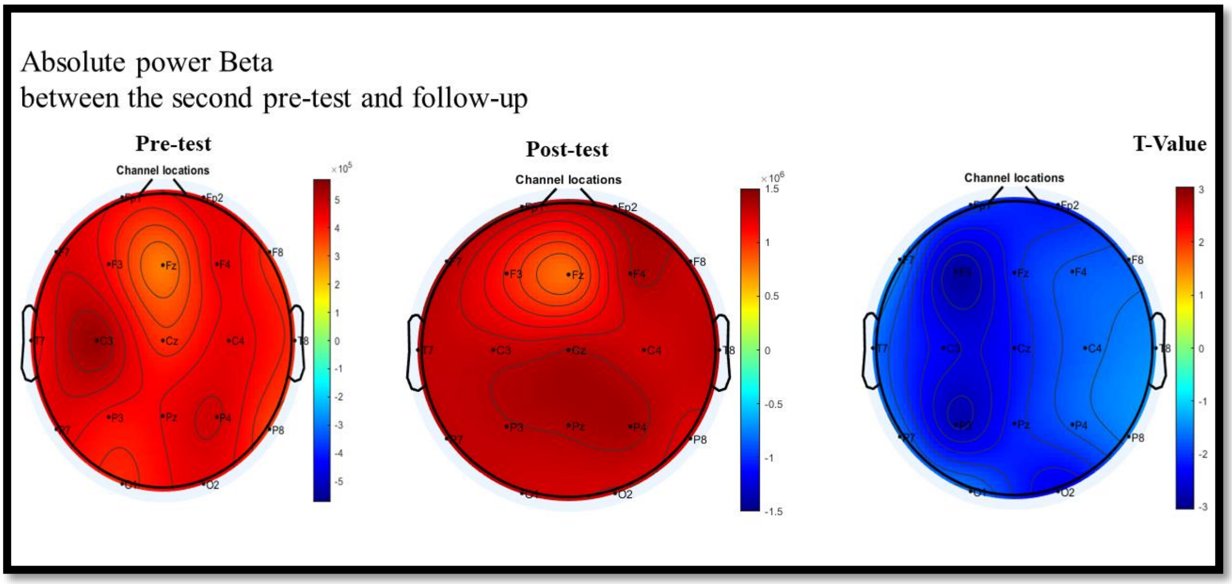

Figure 6 also shows a comparison of changes in absolute beta wave power in Pretest 2 and Follow-up, which may facilitate better understanding. Overall, this table and figure may provide useful information for researchers and specialists in the field of neurofeedback and inhibitory brain control. Figure 7 shows a comparison of absolute gamma power in Pretest 2 and Follow-up.

Figure 7

Comparison of absolute gamma wave power in Pretest 2 and Follow-up.

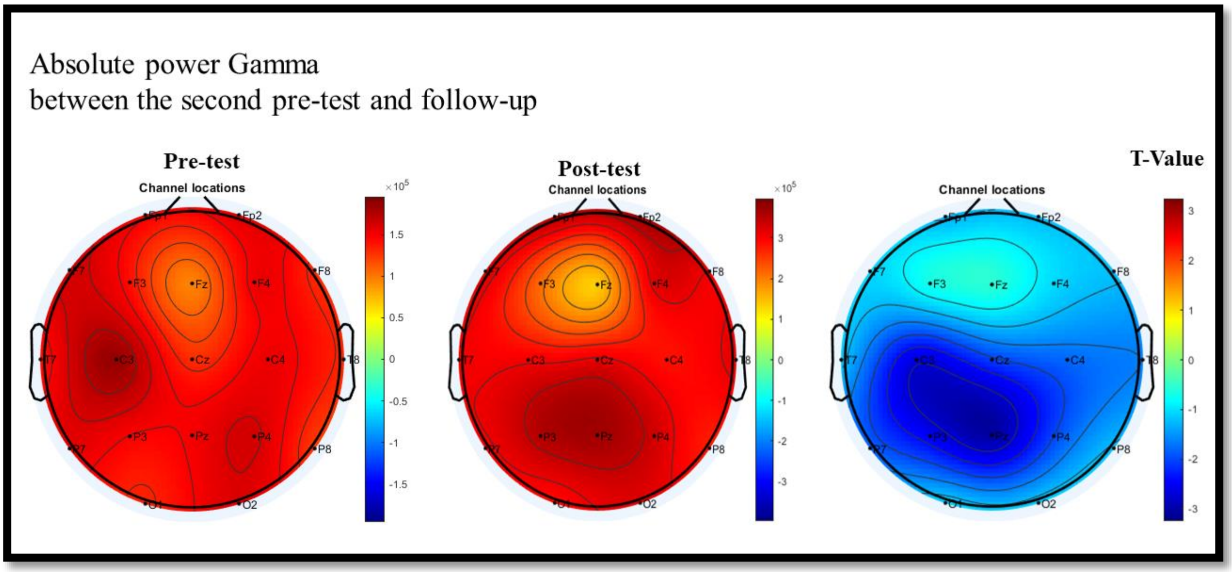

Figure 7 also shows a comparison of changes in absolute gamma wave power in Pretest 2 and Follow-up, which may facilitate better understanding. Overall, this table and figure may provide useful information for researchers and specialists in the field of neurofeedback and inhibitory brain control.

4. Discussion

The findings of the present study showed that infra-low frequency neurofeedback was associated with improvement in both the clinical and cognitive profile of patients with

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Parkinson's disease, although the temporal pattern of change differed across outcome domains. In the motor domain, the participants' scores on the Unified Parkinson's Disease Rating Scale decreased across the study phases, indicating a reduction in the severity of motor impairment, and the comparison between the first pretest and the follow-up phase reached statistical significance. This pattern suggests that the therapeutic effect on motor symptoms was not fully immediate but became more evident after the intervention had ended and patients had entered the follow-up period. A similar pattern was observed for global cognition, as Montreal Cognitive Assessment scores increased over time and the difference between the first pretest and follow-up was statistically significant, whereas the immediate pretest-to-posttest comparison was not. At the same time, the computerized RehaCom findings indicated improvement in several specific cognitive components, especially selective attention, working memory capacity, and logical reasoning. In parallel with these behavioral and neuropsychological gains, the electrophysiological findings demonstrated reductions in absolute beta and gamma power across multiple cortical channels after the intervention, with some of these changes persisting into follow-up. Taken together, these findings suggest that infra-low frequency neurofeedback may exert a gradual regulatory effect on dysfunctional neural activity in Parkinson's disease, with downstream benefits for motor control, general cognitive status, and selected higher-order cognitive processes.

The improvement in motor functioning is clinically meaningful because Parkinson's disease is fundamentally characterized by progressive disturbances in movement initiation, coordination, postural control, and motor adaptability (De Lau & Breteler, 2006; Raza & Anjum, 2019; Sveinbjornsdottir, 2016). The fact that significant motor improvement emerged most clearly at follow-up rather than immediately at posttest is theoretically consistent with the idea that neurofeedback is a learning-based intervention that may require time for consolidation before behavioral gains become fully observable. Unlike pharmacological interventions, which often produce direct short-latency symptomatic effects, neurofeedback may operate through progressive recalibration of self-regulatory networks, altered arousal control, and gradual reorganization of oscillatory dynamics (Grin-Yatsenko et al., 2021; Schneider et al., 2021; Smith et al., 2014). This interpretation aligns with previous work suggesting that infra-low frequency neuromodulation can be useful in the management of intractable Parkinsonian symptoms and may produce changes that are clinically relevant even when standard treatment avenues remain insufficient (Legarda et al., 2022). The present finding is also convergent with evidence that neurofeedback-based interventions can positively affect balance and physical performance in Parkinson's disease, supporting the broader idea that modulation of neural self-regulation can translate into functional motor benefit (Azarpaikan et al., 2014). More generally, research on neuromodulatory interventions in other populations has shown that non-invasive brain-based training can improve functional performance and neurobehavioral outcomes, reinforcing the plausibility of delayed but meaningful gains after repeated sessions (Badicu et al., 2025; Taheri et al., 2025; Talebi & Hashemi Mad, 2025).

The cognitive findings are equally important because Parkinson's disease is increasingly recognized as a disorder that affects cognition from relatively early stages, even in the absence of overt dementia (Riedel et al., 2010; Sveinbjornsdottir, 2016). In the present study, the increase in Montreal Cognitive Assessment scores and improvement in specific RehaCom indices suggest that infra-low frequency neurofeedback may support both global cognition and discrete cognitive operations. Improvement in selective attention and working memory is especially notable because these domains are central to executive regulation, dual-task performance, and adaptive motor behavior in Parkinson's disease. Likewise, gains in logical reasoning suggest that the intervention may have influenced broader executive and integrative processes rather than merely simple vigilance or transient alertness. These findings fit with the view that brain-based interventions can alter network efficiency and promote more adaptive cognitive control, especially when the targeted mechanisms involve large-scale regulatory systems rather than isolated cognitive modules (deCharms, 2007; Ruiz et al., 2014). Evidence from real-time neurofeedback and neuromodulation studies outside Parkinson's disease has shown that trained self-regulation of brain activity can improve emotional stability, anxiety, attention, and higher-order cognitive functioning, suggesting that the benefits observed here are plausible within a broader neuroplastic framework (Jahedi Delivand et al., 2024; Kim et al., 2024; Nicholson et al., 2023; Shamsi Halasu et al., 2023). The present results therefore support the proposition that infra-low frequency neurofeedback may not only reduce symptom burden but also enhance cognitive control processes that are central to independence and quality of life in Parkinson's disease.

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The electrophysiological findings provide an important mechanistic context for interpreting these behavioral changes. One of the clearest results of the study was the reduction in absolute beta and gamma power across several cortical regions after treatment, with persistence of some changes at follow-up. In Parkinson's disease, pathological synchronization and exaggerated beta-band activity have repeatedly been implicated in rigidity, bradykinesia, and impaired readiness for adaptive motor change (Engel & Fries, 2010; Hammond et al., 2007; Zaidel et al., 2010). From this standpoint, the reduction in beta power observed in the present study may reflect a weakening of maladaptive rhythmic rigidity and a shift toward more flexible cortical processing. The finding that this reduction was evident across multiple channels is compatible with the network-based nature of Parkinsonian dysfunction, in which abnormal synchronization is distributed rather than localized to a single cortical site (Cook et al., 2021; Hammond et al., 2007). Studies of the basal ganglia and subthalamic nucleus have similarly shown that oscillatory dynamics are directly relevant to inhibitory success, motor control, and treatment response, indicating that changes in rhythmic activity are not epiphenomenal but functionally significant (Alegre et al., 2013; Jenkinson et al., 2013; Zaidel et al., 2010). Accordingly, the electrophysiological shifts found in this study strengthen the interpretation that infra-low frequency neurofeedback acted on core neural mechanisms rather than producing only subjective or nonspecific improvements.

The reduction in gamma power also warrants careful interpretation. Gamma oscillations are often linked to cortical computation, information integration, and efficient neural communication, but in Parkinson's disease and related dysregulated states, altered gamma activity may reflect maladaptive or compensatory processes depending on context (Fries, 2009; Jenkinson et al., 2013). In the present study, decreases in gamma power were interpreted alongside clinical and cognitive improvement, suggesting that the observed gamma changes may represent normalization rather than simple suppression. This is consistent with the broader literature showing that pathological rhythmic states in Parkinson's disease are not limited to beta abnormalities; rather, disturbances in cross-frequency balance and resonance may contribute to dyskinesia, cognitive inefficiency, and unstable motor output (Alonso-Frech et al., 2006; Halje et al., 2012; Richter et al., 2013). If infra-low frequency neurofeedback helped recalibrate the general oscillatory milieu in which higher-frequency activity is embedded, then reductions in excessive beta and gamma activity may signal improved inhibitory control and more stable neural organization. The persistence of some of these differences at follow-up is especially important because it suggests that the intervention may have induced changes in regulatory set points rather than transient state effects alone.

The delayed emergence of significant differences between baseline and follow-up, together with the maintenance of some EEG changes after the intervention ended, supports the hypothesis that infra-low frequency neurofeedback operates through gradual modulation of infra-slow and resting-state processes. Infra-slow neural fluctuations are increasingly understood as foundational for large-scale network dynamics, including the coordination of resting-state systems and long-distance functional integration (De Luca et al., 2006; Hiltunen et al., 2014; Mitra et al., 2018). The brain's default network and related resting-state systems are relevant not only to cognition and self-referential processing but also to disease-related dysfunction when network stability is compromised (Buckner et al., 2008). Infra-low frequency neurofeedback has been proposed as a means of influencing this slow regulatory architecture, and empirical work has shown that such training can alter infra-slow EEG fluctuations themselves (Grin-Yatsenko et al., 2021; Smith et al., 2014). Therefore, the present findings are coherent with the notion that targeting infra-low activity may gradually influence large-scale neural regulation, which in turn affects higher-frequency oscillations, cognitive control, and motor behavior. This interpretation is also compatible with evidence from real-time fMRI work showing that self-regulation of neural systems can extend from single regions to network-level organization (deCharms, 2007; Ruiz et al., 2014). In this sense, the present study contributes to a growing literature suggesting that clinically meaningful change may occur when treatment addresses the temporal architecture of neural coordination, not merely its momentary output.

Another important aspect of the results is the convergence between general cognitive improvement and more specific changes in attentional and executive components. Parkinson's disease often compromises attentional filtering, inhibitory control, working memory updating, and executive monitoring, partly because these functions rely on the same distributed frontostriatal and corticobasal ganglia systems that are disrupted in motor control (Alegre et al., 2013; Riedel et al., 2010). The present improvements in selective attention and logical reasoning can therefore be interpreted

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as functionally related to the neural normalization reflected in the EEG data. If pathological synchronization was reduced, patients may have become more capable of shifting attentional focus, suppressing irrelevant information, and coordinating cognitive operations. Research on neuronal coherence indicates that effective interaction between cortical systems is critical for both motor and cognitive performance (Schoffelen et al., 2005). Likewise, the theoretical account of beta oscillations as signaling the status quo suggests that excessive beta activity may constrain not only movement but also cognitive flexibility (Engel & Fries, 2010). By reducing this excessive stability, infra-low frequency neurofeedback may have supported a more adaptive balance between persistence and flexibility in both behavior and cognition.

The present study also has implications for the broader literature on non-invasive neuromodulation. Across diverse populations, including athletes, children with neurodevelopmental conditions, healthy adults, and older adults, interventions such as neurofeedback and transcranial stimulation have been associated with improvements in performance, cognitive function, emotional regulation, and resilience (Badicu et al., 2025; Jahedi Delivand et al., 2024; Shamsi Halasu et al., 2023; Taheri et al., 2025; Talebi & Hashemi Mad, 2025). The present results extend this translational logic into a neurodegenerative context and suggest that Parkinson's disease may be an especially important target for such approaches because of its pronounced network-level dysregulation. While the study does not establish that infra-low frequency neurofeedback is superior to other neuromodulatory methods, it does support the clinical relevance of further investigating neurofeedback as a potentially accessible and non-invasive adjunct. In light of prior conceptual work on neural networks and neurofeedback in Parkinson's disease (Esmail & Linden, 2014), the current findings provide empirical support for the idea that the intervention may influence both symptom expression and underlying electrophysiological organization. They also complement feasibility evidence from sensorimotor rhythm neurofeedback by suggesting that very slow-frequency training may likewise be useful and perhaps particularly suited to widespread dysregulation across cortical systems (Cook et al., 2021).

At the same time, the findings should be interpreted with scientific caution. The observed pattern of improvement across motor, cognitive, and EEG outcomes is encouraging, but it remains possible that some of the gains were shaped by nonspecific factors such as repeated assessment, therapeutic contact, expectancy, or spontaneous fluctuation. Nonetheless, the parallel emergence of behavioral and electrophysiological changes, together with the lack of significant differences between the two pretest phases, argues against a purely artifactual explanation and supports the inference that the intervention contributed meaningfully to the changes observed. Moreover, the use of EEG-based indices strengthens the internal interpretability of the findings because the electrophysiological changes were directionally consistent with what is known about pathological oscillations in Parkinson's disease (Engel & Fries, 2010; Fries, 2009; Hammond et al., 2007). Advances in multimodal mapping between EEG and broader network signals suggest that such changes are increasingly interpretable within a systems neuroscience framework (Carmichael et al., 2024). In this regard, the present findings align with contemporary efforts to connect clinical change with network-level biomarkers, an issue of growing relevance across neuropsychiatric and neurological intervention research (Zugman et al., 2023).

5. Conclusion

One further interpretive point concerns the heterogeneity of response across specific RehaCom tasks and EEG channels. Not all indices changed equally, and some cognitive variables appeared more responsive than others. This is not surprising in Parkinson's disease, where symptom burden varies considerably across patients and where different neural systems may have different thresholds for plastic change (De Lau & Breteler, 2006; Sveinbjornsdottir, 2016). It is possible that infra-low frequency neurofeedback first affects foundational self-regulatory processes, which then generalize more readily to some domains, such as selective attention and working memory, than to others. Similarly, because infra-slow activity appears to support broad state regulation, region-specific EEG outcomes may depend on baseline network vulnerability and the degree to which local oscillatory abnormalities are embedded within broader dysfunctional circuits (Hiltunen et al., 2014; Mitra et al., 2018). Future work should therefore aim not only to confirm efficacy but also to identify which symptom profiles, electrophysiological signatures, and disease stages are most likely to benefit.

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6. Limitations & Suggestions

The main limitation of the present study was the very small sample size and the absence of a control or sham comparison group, which restricts the generalizability of the findings and limits causal certainty. The single-group quasi-experimental structure also makes it difficult to disentangle intervention effects from time effects, practice effects, or natural fluctuation in Parkinsonian symptoms. In addition, the follow-up period was relatively short, so the long-term stability of the observed gains remains unclear. Another limitation is that the sample consisted of patients within a restricted disease-severity range, which may limit the applicability of the findings to individuals at milder or more advanced stages. Finally, although EEG indices were included, the study did not incorporate broader multimodal neuroimaging or more extensive neuropsychological batteries that could have clarified the full mechanism and scope of change.

Future research should examine infra-low frequency neurofeedback in larger randomized controlled trials with sham or active comparison conditions and longer follow-up periods. It would also be valuable to compare infra-low frequency protocols with other neurofeedback approaches, cognitive rehabilitation methods, and non-invasive neuromodulation techniques to determine relative efficacy and differential indications. Further studies should include more detailed cognitive, emotional, and functional measures, as well as multimodal neural assessment, in order to clarify whether improvements are driven primarily by changes in arousal regulation, executive control, motor network dynamics, or broader resting-state reorganization. Researchers should also investigate individual differences in treatment response, including disease duration, dominant symptom pattern, medication status, baseline oscillatory profile, and comorbid cognitive or affective problems.

From a practical standpoint, the present findings suggest that infra-low frequency neurofeedback may be considered as a complementary, non-invasive intervention for patients with Parkinson's disease who experience combined motor and cognitive difficulties, particularly when conventional treatment does not fully address daily functional impairment. Clinicians should view this approach as potentially cumulative rather than immediately symptomatic, meaning that benefits may become more visible over time and may require structured follow-up. Integrating neurofeedback into multidisciplinary Parkinson's care may be especially useful when paired with cognitive rehabilitation, physical therapy, and careful neurological monitoring. Because the intervention appears capable of influencing both behavioral outcomes and electrophysiological regulation, it may offer added value in rehabilitation settings that prioritize long-term self-regulation, functional adaptation, and preservation of cognitive-motor capacity.

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Declaration of Interest

The authors of this article declared no conflict of interest.

Ethical Considerations

The study protocol adhered to the principles outlined in the Helsinki Declaration, which provides guidelines for ethical research involving human participants.

Transparency of Data

In accordance with the principles of transparency and open research, we declare that all data and materials used in this study are available upon request.

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Authors' Contributions

All authors equally contributed in this article.

Declaration

In order to correct and improve the academic writing of our paper, we have used the language model ChatGPT.

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