

Motor-imagery neurofeedback for beta downregulation in Parkinson's disease

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Introduction: Parkinson's disease (PD) is a neurodegenerative disease associated with motor and non-motor symptoms, with current treatments limited by eligibility criteria and side effects. Since 2002, neurofeedback (NF) has been explored as a complementary therapy, but evidence remains highly inconclusive, showing variability in NF metrics and motor outcomes [1]. One third of these studies have used EEG, often targeting generic neural activities unrelated to the mechanisms underlying PD [2]. Knowledge of PD pathophysiology has improved since, establishing alpha, beta and gamma bands as potential NF targets [1]. Yet, the pathological beta band has been targeted in only two studies [3, 4]. We aim to confirm the feasibility of a non-invasive beta downregulation NF protocol using motor imagery (MI) and explore its motor effects.

Material, Methods and Results: 14 PD patients on dopaminergic treatment performed an EEG-based NF session (64 electrodes). During NF trials, they used right-hand MI to reduce beta power (13-30 Hz) over the left motor cortex. Online feedback (FB) was provided by virtual hand movement amplitude, proportional to beta reduction relative to rest. The experiment included "MI" control trials, where patients performed MI without FB. Each patient completed 3-4 sequences of one MI trial followed by two NF trials. Every trial was immediately followed by a 7s-finger tapping task, measured via EMG and accelerometer.

Beta reduction (ie. NF performance) was greater in the NF condition than MI control for 13 out of 14 patients (Fig 1A). This reduction was associated with bilateral desynchronization, peaking over left central electrodes in the low beta band and on more central electrodes in the high beta band (Fig 1B, bottom row). In contrast, the MI control condition showed less pronounced and more widespread desynchronization, without clear focus on the targeted area (Fig 1B, top row). NF performance progressively increased along trials in the NF condition, while it remained stable in the MI condition. Eleven patients had shorter reaction times following NF than MI trials, and 10 of them showed an increased number of taps.

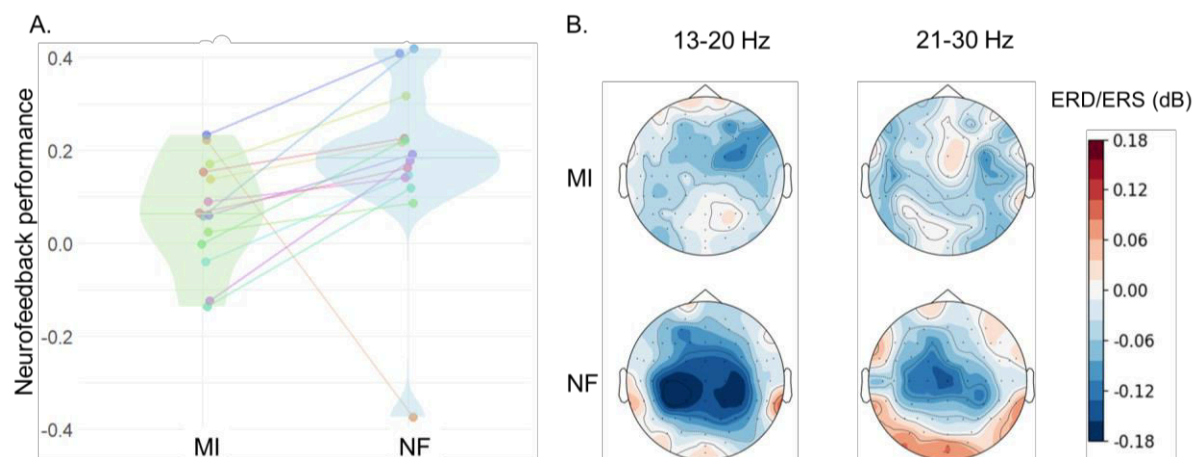


Figure 1: A. Individual neurofeedback performance during MI and NF trials for each subject (colored dots and lines), with the distribution of NF performance across subjects represented as a violin plot for each trial type. B. Grand average topographical maps of ERD/ERS in the low beta (13-20 Hz) and high beta (21-30 Hz) bands across the 14 participants during MI and NF trials.

Conclusion: ON-dopa PD patients can reduce successfully their beta activity in a single NF session. Providing FB induced a greater reduction of beta activity compared to performing MI alone. While the absence of a sham condition limits inferences about the specific impact of NF *per se*, MI was chosen because it is used in PD therapy and has been shown to improve motor function [5]. Our results suggest that enhancing beta desynchronization, here through NF, can be associated with improved motor function in PD, particularly the sequence effect of bradykinesia (i.e rapid decrement in amplitude and speed of repetitive movements). Future analyses will explore links between neurophysiological modulations and motor task performance.

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