Neuronal and Motor Activity in the Basal Ganglia During a Synchronization-Continuation Hand Tapping Task in Patients with Deep Brain Stimulation for Parkinson's Disease

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Abstract

Introduction: Deep brain stimulation (DBS) of the subthalamic nucleus (STN) is used for Parkinson's disease (PD) patients refractory to medications. One symptom that is not significantly improved by DBS is freezing of gait (FoG) which, is associated with a festination behaviour in the synchronization-continuation task (SCT). The SCT consists of three 30s phases: listening while still, tapping in time with an external cue (like a metronome), then continuing to tap without the external cue, requiring a shift from external to internal representations of rhythm. Studies have shown that patients with PD exhibit motor hastening in the continuation phase, with higher magnitude of hastening correlated to severity of FoG. To better understand the relationship between rhythmic control in the STN, medication, and DBS, 15 PD patients underwent the SCT while having both their motor behaviour and neural activity recorded.

Method: Patients with DBS leads implanted in the STN underwent testing in four different conditions: M1S1 (Med ON/ Stim ON), MOSO (Med OFF/Stim OFF), M1SO (Med ON/ Stim OFF), and MOS1 (Med OFF/Stim ON), with Med OFF defined as 12hr off anti-Parkinson medication. Motor data was recorded using a triaxial accelerometer or a piezoelectric force detection device. Neural activity in the form of local field potentials was recorded using the PerceptTM technology. Motor and neural local field potential (LFP) data were synchronized using Spike2 and analyzed using Python and R.

Results: Preliminary analysis of 3 patients showed motor hastening across all conditions, with greater magnitude in MOSO (0.20 Hz) than M1S1 (0.12 Hz). Beta power was elevated in OFF states and reduced by medication or stimulation, while peak frequencies remained stable. In MOSO, beta power was highest during the listening phase, suggesting desynchronization with motor initiation.

Discussion: Our findings align with prior reports of motor hastening in PD during the SCT. The greater hastening observed in MOSO compared to M1S1 suggests that medication and DBS may help stabilize rhythmic motor output, while future analyses of M1S0 and MOS1 will clarify their individual effects. Spectral results also showed elevated beta power in MOSO and suppression with medication or stimulation, consistent with existing literature.

Conclusions: The results thus far confirm that motor hastening in the SCT is common in patients with PD, and indicate that dopaminergic medication and DBS may help to reduce the degree of hastening. Additionally, the differences in beta oscillatory power across the different phases of the SCT, when combined with the behavioural results of the SCT, may help to better describe the role of the STN in rhythmic control, and how it is affected PD.

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