

A magnetoencephalographic study into the pathophysiological substrates of cognitive and motor symptoms and treatment effects in parkinsonian syndromes

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Institution

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A magnetoencephalographic study into the pathophysiological substrates of cognitive and motor symptoms and treatment effects in parkinsonian syndromes

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Parkinson's disease & PD-related disorders

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Research Abstract

Here I will test two hypotheses. First, that different clinical impairments in patients with Parkinson's Disease (PD) relate to distinct basal ganglia-cortical circuits identifiable through their preferred frequency of interaction and their topography. Second, that Deep Brain Stimulation (DBS) improves these impairments by modulating coupling across these circuits. To this end I will record local field potentials (LFPs) from electrodes implanted in the subthalamic nucleus (STN) of post-surgical PD patients whilst simultaneously performing magnetoencephalography (MEG). Using source reconstruction techniques I will reconstruct electrical activity from cortical areas and compute spectral coherence with electrical activity from the STN. I propose two experiments. In the first I will employ the Posner task in order to determine whether the previously defined alpha frequency STN-cortical network is involved in orienting attention to visuo-spatial cues and is dependent on dopamine. In the second experiment I will perform DBS at therapeutically effective and non-effective frequencies in order to determine specifically how STN-cortical networks are influenced by DBS. In order to facilitate this, we have developed a special amplifier that allows simultaneous stimulation and recording of LFP signals. Furthermore, we will also rely on a number of existing engineering methods to remove the stimulation artifact from the MEG channels prior to performing source reconstruction. Finally, by correlating motor and cognitive parameters with patterns of STN-cortical coupling I will gain valuable insight into: 1) the involvement of specific STN-cortical loops in mediating the symptoms of PD and 2) the therapeutic mechanisms of DBS.

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