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TRR 295 ReTune

Cortical network formation based on subthalamic beta bursts in Parkinson's disease.

Sure M, Vesper J, Schnitzler A & Florin E.
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Functional imaging of the resting brain consistently reveals broad motifs of correlated activity that engage cerebral regions from distinct functional systems. Importantly, these networks have been related to alterations in numerous neurological and psychiatric diseases. Yet, the neurophysiological processes underlying these organized large-scale fluctuations are not yet known.

Combined fMRI-local field potential (LFP) recordings in monkeys have indicated that certain cortical networks form around the occurrence of hippocampal ripples. In the present paper, we extended the neural event triggered approach to purely electrophysiological data and investigated network activity triggered by neural events such as burst-like activity. As a test case, we used combined magnetoencephalography (MEG)-LFP recordings in Parkinson patients. In previous studies, beta band activity in Parkinson patients has been identified as pathologically elevated in the subthalamic nucleus (STN) and appears in the form of transient bursts. Because of the event-like phasic burst nature of beta activity and its electrophysiological connection to cortical activity, we hypothesized that cortical activity patterns evolve time-locked to STN beta bursts.

To test this hypothesis as well as event-triggered analysis for human electrophysiological data, we simultaneously recorded in the post-operative phase STN LFPs from externalized deep brain stimulation electrodes and cortical activity using MEG of 26 Parkinson's disease patients in the medication OFF state. To study the spatio-temporal relationship between STN beta burst and cortical activity event-related magnetic fields were computed time-locked to STN beta bursts and subjected to source localization. The time before and after STN beta burst onsets was associated with significant changes in cortical activity compared to baseline. Beta bursts in the STN were associated with cortical activations in areas functionally belonging to the motor, limbic, and associative systems. Before the onset of a burst, altered activity was mainly observed in the somatosensory motor area.

Overall, our findings indicate that cortical activity can be linked to events originating in subcortical structures such as the STN. Neural event-triggered analysis allows to study whole-brain networks, while taking into account the relevance of subcortical activity. The identified connections between STN and cortex align with the basal-ganglia cortex loops connecting the STN with cortical areas. As we found increased and decreased cortical activity linked to STN beta bursts, such bursts are likely involved in two pathways of the basal-ganglia cortex loop: (1) STN beta busts are a result of the hyperdirect pathway from the cortex to the STN and (2) a cause of the indirect pathway from the STN to the cortex.

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Esther Florin is a Lichtenberg Professor at the Heinrich-Heine University Düsseldorf, funded by the Volkswagen foundation. In her research, she analyzes neural connectivity to understand cognition and behaviour in healthy subjects and neurological patients. A particular focus is on tracing out the electrophysiological mechanisms underlying the effectiveness of dopaminergic treatment and deep brain stimulation for Parkinson patients.

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