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Beta Bursting and Network Synchrony in Parkinson's Disease

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Introduction

Pathophysiological beta oscillations across cortico-subcortical structures are thought to be a central causative feature in Parkinson disease (PD) [1]. Furthermore, recent evidence suggests that features of beta oscillations, such as bursting [2], waveform shape [3] and phase-amplitude coupling [4], are abnormal in the dopamine depleted state. Here we show that cortical beta bursting is prolonged in PD and associated with increased interregional synchrony. Waveform shape of beta oscillations is distinct during beta bursts, contrasting with non-burst episodes, and bursts are accompanied by increases in local cortical phase-amplitude coupling.

Methods

We studied 32 patients undergoing deep brain stimulation (DBS) surgery for PD (22 patients) and essential tremor (10 patients). A subdural strip of 8 electrodes was advanced intracranially to lie rostro-caudally over sensorimotor cortices. Patients made cued opening and closing hand movements in blocks of 30s interpolated with 30s rest periods. MRI/CT fusion combined with SSEPs allowed for accurate identification of sensorimotor cortices.

Results

All cortices studied (S1, M1 and premotor) showed significantly increased higher amplitude beta oscillations (20-35Hz) in PD compared to ET patients. Both ET and PD patients showed similarly increased synchrony during beta bursting compared to non-bursting. Waveform analysis shows that burst episodes are associated with a more 'sawtooth' oscillation shape. Beta bursting was associated with increased beta to broadband gamma phase-amplitude coupling in motor cortex and prolongation of beta bursting shows a correlation with clinical bradykinesia and rigidity scores on the UPDRS.

Conclusions

Here we link together several strands of research to show that not only are beta bursts longer in duration in PD motor cortex but that they can be characterized as episodes of high interregional synchrony, phase-locking premotor and motor cortices. Furthermore, waveform shape of beta bursts is altered, potentially explaining why burst episodes show increased phase amplitude coupling between the beta band and broadband gamma.

Learning Objectives

Participants should be able to understand how various aspects of PD neurophysiology relate to one another and how this leads to expression of the symptoms of the disorder at the behavioral level.

References

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