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Cortical motor network excitability changes in Parkinson's disease

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Background: Bradykinesia in Parkinson's disease (PD) reflects changes in the basal ganglia-thalamo-cortical circuit converging on the primary motor cortex (M1) and supplementary motor area (SMA).

Objectives: To assess motor cortical network excitability in PD patients and their relation to dopaminergic status and bradykinesia.

Methods: We compared transcranial magnetic stimulation-evoked cortical potentials (TEPs) from the M1 and SMA between 15 PD patients tested off (OFF) and on (ON) medication and 12 healthy controls (HCs) and investigated possible correlations with bradykinesia tested clinically.

Results: OFF PD patients compared to HCs had smaller P30 responses from the M1s contralateral (M1+) and ipsilateral (M1-) to the most bradykinetic side, reduced N45 from the M1+, and increased N40 from the SMA. OFF PD patients showed a significant correlation between the amplitudes of the M1+ P30 and the SMA N40. Dopaminergic therapy normalized the amplitude of the M1+ and M1- P30 responses as well as the SMA N40. We found a positive correlation between M1+ P30 amplitude and bradykinesia in OFF PD patients.

Conclusions: Changes in M1 P30 and SMA N40 in PD suggest that M1 excitability is reduced on both sides while SMA excitability is increased. The effect of dopaminergic therapy and the clinical correlation suggest that these cortical changes may reflect abnormal basal ganglia-thalamocortical activity related to bradykinesia. The N45 reduction in PD patients suggests additional excitability changes in the most affected M1, which are dopamine independent and not directly correlated with bradykinesia. TMS-EEG provides a novel insight into motor cortical network changes related to the pathophysiology of PD.