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Aberrant brain plasticity in patients with Parkinson's disease and levodopa-induced dyskinesias

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Introduction: Several alterations of physiological synaptic plasticity have been documented in patients with Parkinson's disease (PD) and Levodopa-Induced Diskinesias (LIDs). These alterations, in particular the failure of synaptic depotentiation, play a key role in the pathophysiology of LIDs.

Objective: We performed this monocentric cross-sectional study aimed to identify the alterations of cortical plasticity at primary motor cortex (M1) in PD patients with and without LIDs and to correlate the neurophysiological alterations with the severity of LIDs.

Methods: Each patient underwent a preliminary visit of enrolment and administration of clinical scales (MMSE, UPDRS and UDyRS). The patients underwent two sessions of theta-burst stimulation (TBS) in different days. In the first paradigm ("potentiation session"), a train of continuous TBS (cTBS) 300 stimuli was delivered over M1 followed by the 1-min voluntary contraction of the target muscle (cTBSc0). In the second paradigm ("depotentiation session"), the potentiation session (cTBSc0) was followed by the delivery of a train of cTBS 150 stimuli (cTBS150), which reversed the long-term potentiation-like plasticity previously induced.

Results: We recruited 24 PD patients, 13 without LIDs and 11 with LIDs. We found a significant depotentiation in the group of non-dyskinetic patients ($F = 4.55$, $p = 0.002$) and a not significant depotentiation in the group of dyskinetic patients ($F = 0.49$, $p = 0.7$). A negative correlation was found between depotentiation (measured by the % of MEP amplitude variation following cTBS150) and score of the UDyRS part III ($r=-0.7$, $p=0.005$).

Conclusions: Our results showed that PD patients with LIDs fail to respond normally to a depotentiation protocol and that impairment in depotentiation correlates with dyskinesias severity, so cTBS may represent a potential biomarker of LIDs in PD patients.