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Neurophysiological changes of primary motor cortex in patients with Essential Tremor-Plus

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Introduction: Essential tremor-plus (ET-plus) represents a recently introduced entity indicating ET patients with additional neurological signs of uncertain significance, including rest tremor, bradykinesia and mild cognitive impairment (MCI). The concept of ET-plus, however, is still controversial and only few studies investigated the neurophysiological mechanisms underlying this condition.

Aims: To investigate possible neurophysiological changes of the primary motor cortex (M1) and their relationship with soft signs in patients with ET-plus.

Methods: Thirteen ET-plus patients were enrolled (5 females, 70±7.97 years). Most patients had rest tremor, subtle bradykinesia, MCI and only 3 of them had impaired tandem gait. Patients were evaluated by standardized clinical scales. Objective measurements of rest tremor and bradykinesia (during finger tapping) were obtained by kinematic analysis. M1 excitability was assessed by the recordings of resting motor thresholds (RMTs), input/output curve of the motor-evoked potentials (MEPs) and using a conditioning-test paradigm for the assessment of short-interval intracortical inhibition (SICI) and short-latency afferent inhibition (SAI). Plasticity-like mechanisms were indexed according to MEPs amplitude changes after intermittent theta-burst stimulation (iTBS). Data were compared to those from 16 healthy controls (HCs). Correlations between clinical, kinematic, and neurophysiological data were assessed in patients.

Results: Compared to HCs, ET-plus patients had higher RMTs (P=0.019), indicating a lower corticospinal excitability and a lower MEPs facilitation after iTBS (P=0.032), reflecting a lower cortical plasticity. ET patients were slower than HCs during finger tapping (P=0.03). No correlations, however, was found between neurophysiological, clinic and kinematic. In particular, there was no significant relationships between neurophysiological changes of M1 and the type or severity of soft signs in patients.

Conclusion: We here provided novel information on excitability and plasticity abnormalities of M1 in patients with ET-plus. The lack of correlation between clinical and neurophysiological data suggests that various ET-plus forms do not represent entities with a specific pathophysiological background.