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Pathophysiological mechanisms underlying sensory trick in cervical dystonia: an electroencephalogram- electromyography study

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Introduction: A characteristic and unique feature of focal dystonia is the partial or complete improvement of dystonic symptoms due to the execution of a voluntary manoeuvre, referred to as sensory trick (ST) [1]. Although previous studies showed that ST modifies sensorimotor cortex activity and connectivity [2-3], the underlying neurophysiological mechanisms remain unclear.

Objective: In this study, we investigated whether and how ST modulates cortical oscillations. Moreover, we aimed to verify whether the effectiveness of ST on dystonic symptoms reflects touch-related changes in oscillations over the sensorimotor cortex.

Methods: We recorded electroencephalography (EEG) activity before, during, and after ST execution in patients with cervical dystonia (CD) with effective ST. We also recorded electromyography (EMG) activity over the sternocleidomastoid (SCM) muscle bilaterally. Touch-related EEG spectral perturbation over sensorimotor areas was recorded in 8 CD patients with effective ST, defined as reduced EMG activity in the affected SCM muscle. Data were compared to those obtained in 9 CD patients with ineffective ST. Independent sample t-test comparisons were applied to test event-related spectral perturbation (ERSP) differences between patients with and without effective ST before, during, and after the ST touch. Bonferroni correction for multiple comparisons was used as a post hoc t-test.

Results: ERSP analysis in patients with effective ST showed bilateral event-related desynchronization (ERD) in the mu (8-13) band in sensorimotor cortical regions that was present only during the execution of ST ($p= 0.001$) and was directly related to reduced EMG activity in the dystonic muscle

Conclusions: Our results suggest that oscillatory activity changes in the mu band over the sensorimotor cortex are involved in the pathophysiology of ST. Our findings may also provide useful information regarding the role of abnormal sensorimotor integration in the pathophysiology of dystonia.

References:

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