P93

The role of the somatosensory system in cervical dystonic patients: a study with high-frequency oscillation and short-latency afferent inhibition

Alessandro Cruciani[1,2], A. Magliozzi[1,2], J. Lanzone[3], G. Anzini[1,2], V. Di Lazzaro[1,2], M. Marano[1,2]

[1]Department of Medicine, Research Unit of Neurology, Neurophysiology and Neurobiology, Università Campus Bio-Medico di Roma, Rome, Italy
[2]Fondazione Policlinico Universitario Campus Bio-Medico, Operative Research Unit of Neurology, Rome, Italy
[3]Neurorehabilitation Department, IRCCS Istituti Clinici Scientifici Salvatore Maugeri di Milano, Milan, Italy

Introduction: In the past few decades, researcher and clinicians have focused their attention on the sensory system's contribution to the pathogenesis of focal dystonia [1]. It is well known that patients suffering from cervical dystonia complain of sensory symptoms, such as pain in the neck or forearm, even before the clinical disease onset [2]. Moreover, other clinical evidence supports the idea that sensory abnormalities contribute to focal dystonia pathogenesis come from the sensory tricks phenomenon that is well described in particular in cervical dystonia [3]. Non-invasive brain stimulation (NIBS) and in particular High-frequency oscillation (HFOs) and short-latency intracortical inhibition (SAI) are two reliable neurophysiological parameters that give us the opportunity to study in vivo the status of the somatosensory system and the sensorimotor integration [4-5]. Thus, with the present study, we aim to investigate the role of the somatosensory system and sensorimotor integration in patients with cervical dystonia with the study of HFOs and SAI.

Objective: To investigate the role of the somatosensory system in Dystonia pathogenesis.

Methods: We consecutive enrolled 5 right -handed healty subjects (Mean age 45 ± 1,2) and 5 right-handed patients with cervical dystonia (Mean age 46 ± 12, mean TWSRS 15.8 ± 6.5) from movement disorders clinic of the Fondazione Campus Bio-Medico di Roma. Neurophysiological assessment includes bilateral registration of short-latency afferent inhibition (SAI) values and somatosensory evoked potentials (SEPs) elicited from the median nerve of the dominant hand. Then we applied a digital 400–800 Hz bandpass butterworth filter to extract HFOs from SEPs signal. Data were compared trought independent sample t-test or Mann-Whitney U test according to their distribution (Shapiro-Wilk test).

Results: Comparison between controls and dystonic patients shown no statistical differences in terms of SAI values in the dominant hemisphere ($p = 0,343$) and non-dominant hemisphere ($p = 0,343$). There was a statistically significant difference in terms of HFOs total area under the curve ($p = 0,03$) between patients and controls, reflecting higher values of early HFOs. Nonetheless, this difference does not reach a statistically significant difference ($p = 0,08$). No differences were found in the late HFOs area under the curve ($p = 0,144$).

Conclusions: Our study found that patient with cervical dystonia has HFOs values significantly higher than healthy controls, especially in the early component – i.e. the thalamo-cortical part - but without differences in terms of SAI. Accordingly, those evidence suggest that in dystonic patients there is a selective alteration of the somatosensory pathway rather than an implication of their integration with the motor cortex. Inoue et co-workers have tried to assess HFOs in cervical dystonia patients, found opposite results consisting in a significant decrement in the late HFOs [6]. Those results are not contrasting with our findings, but instead reinforce the concept that in cervical dystonia there is an

impairment in the sensory input signaling and processing that in neurophysiology studies could led to an alteration of the late or early HFOs components. Those preliminary data are part of a more extend study that also comprehend a second neurophysiological assessment after 30 days of treatment with Onabotolinum toxin A (Botox) in order to evaluate the central effects of the toxin and a biomarker to predict patients' degree of improvement with Botox.

References:

[1] Conte, A., Defazio, G., Hallett, M., Fabbrini, G. & Berardelli, A. The role of sensory information in the pathophysiology of focal dystonias. Nat. Rev. Neurol. 15, 224–233 (2019).

[2] Relja, M. & Miletić, V. When movement disorders hurt: Addressing pain in hyperkinetic disorders. Parkinsonism Relat. Disord. 44, 110–113 (2017).

[3] Ramos, V. F. M. L., Karp, B. I. & Hallett, M. Tricks in dystonia: ordering the complexity. J. Neurol. Neurosurg. Psychiatry 85, 987–993 (2014).

[4] Cruciani, A., Lanzone, J., Musumeci, G., Di Lazzaro, V. & Marano, M. Focal vibrations enhance somatosensory facilitation in healthy subjects: A pilot study on Equistasi® and high-frequency oscillations. Front. Neurol. 13, 1052989 (2022).

[5] Tokimura, H. et al. Short latency inhibition of human hand motor cortex by somatosensory input from the hand. J. Physiol. 523, 503–513 (2000).

[6] Inoue, K. et al. Disinhibition of the somatosensory cortex in cervical dystonia—decreased amplitudes of high-frequency oscillations. Clin. Neurophysiol. 115, 1624–1630 (2004).