Clinical Neurophysiology 130 (2019) 1025–1026

Contents lists available at ScienceDirect

Clinical Neurophysiology

journal homepage: www.elsevier.com/locate/clinph

Editorial

Need to find a signature of abnormal brain oscillations in task-specific focal dystonia

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Dystonia is a movement disorder characterized by uncontrolled muscle contractions, which have been associated to loss of inhibition, maladaptive neural plasticity and impaired sensory-motor integration (Neychev et al., 2011; Dubbioso et al., 2017). The lack of inhibition across multiple levels of the central nervous system may be responsible for excess of movement and overflow phenomena (Hallett, 2011), as well as for the occurrence of abnormal plasticity (Quartarone and Pisani, 2011).

Structural and functional neuroimaging studies have revealed a network model, where dystonia can emerge either from a single node dysfunction, from an impairment of multiple nodes or from an abnormal interplay among the nodes (Quartarone and Ruge, 2018). Resting state fMRI (rfMRI), for instance, showed reduced connectivity between the primary sensory-motor cortex of the affected body region and the dorsal premotor cortex and was able to differentiate types of task-specific focal dystonia by introducing specific patterns of impaired connectivity (Bianchi et al., 2019).

Among the not many studies exploring brain oscillations in dystonia, one study found a significant reduction of beta band connectivity within the sensorimotor area at rest and during finger tapping in patients with focal-hand dystonia (Jin et al., 2011). Within the same subset of patients, but using Magnetoencephalography, a gamma-band uncoupling emerged during a simple isometric contraction (Melgari et al., 2013) whereas two other studies, focusing on sensorimotor integration, documented a significant reduction of theta coherence between ipsilateral and contralateral sensory areas (Cheng et al., 2016) and an increase of cortico-muscular coherence in presence of a reduced primary motor cortex responsiveness to the inflow from the sensory regions (Tecchio et al., 2008). The beneficial effect of botulinum toxin injection therapy on aberrant sensorimotor integration has been explained by showing an increase of coherence mostly between left putamen and right superior parietal gyrus (Mahajan et al., 2017). Despite the growing scientific interest regarding dystonia, especially in last years, it is still difficult to identify a clear pattern that can explain a general oscillatory pathophysiology signature. This is even more evident for oscillatory properties peculiar to different task-specific focal dystonia, for example in spasmodic dysphonia (SD).

SD is characterized by irregular and uncontrolled voice breaks that interrupt normal speech flow. There are two general types: adductor SD (AdSD) with hyperadduction spasms disrupting phonation on vowels and abductor SD (AbSD) with prolonged voiceless consonants before vowel onsets (Murry, 2014).

SD is largely unknown and hence little advancement has been made in its diagnosis or treatment. To date only one EEG study investigated SD and identified cortical abnormalities in resting-state patterns (Devous et al., 1990). Now, a new contribution about AdSD during voice production is proposed by Khosravani et al., in the present issue of *Clinical Neurophysiology* (Khosravani et al., 2019). The study aimed at characterizing the dynamics of transient somatosensory-motor cortical processes associated with SD, using EEG to capture fast-cortical neuronal phenomena underlying the abnormal speech phonatory functioning. The new findings about SD include a reduced desynchronization in the alpha band over the left motor cortex and an increased coherence between left somatosensory and premotor cortical regions during early vocalization. During late vocalization instead, SD patients showed increased connectivity between left somatosensory and premotor cortical areas in the gamma band. The authors concluded that the pathophysiology of SD is characterized by an abnormally high synchronous activity within and across cortical neural networks involved in voice production, which might be linked to the impaired inhibitory processes. Still it remains unclear whether the findings are phenomena specific to SD or general properties of dystonia, since movement-related reductions of cortical desynchronization were already observed in cervical and segmental dystonia (Crowell et al., 2012; Miocinovic et al., 2015) as well as in writer's cramp (Toro et al., 2000). Furthermore, the neurophysiological findings would be clearer if the cortical activity of SD participants were not only evaluated in their symptomatic state, but also after the beneficial effect of botulinum toxin injection (Esposito et al., 2015). The study from Khosravani et al. can inspire future investigations to replicate and deepen the findings, since the low number of subjects and the methodological choice to focus on a limited number of EEG channels don't allow conclusive statements from the presented results.

These future studies should include both, methodologically advanced protocols and task specific experimental designs. For example, artefacts due to vocalization are hard to remove and there should be included a "sham" condition where word vocalization is mimicked. Or, the combination between EEG and techniques

https://doi.org/10.1016/j.clinph.2019.04.002

characterized by higher spatial resolution, such as fMRI and diffusion-MRI, could track the pathological networks underlying aberrant cortical oscillations. Finally, the comparison of several forms of task-specific focal dystonia can provide the answer about which impaired cortical synchronization is a specific signature of SD and which is a common pathophysiological mechanism of impaired inhibition underlying different dystonia disorders. This is probably the way to go for the development of more effective therapies for SD, for instance neuromodulation interventions capable to normalize aberrant cortical oscillations.

Conflict of interest statement

None of the authors have potential conflicts of interest to be disclosed.

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Accepted 2 April 2019

Available online 9 April 2019