DYSTONIA

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Editorial: Treatment options in dystonia

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Editorial on the Special Issue
Treatment options in dystonia

Our understanding of the pathophysiology of dystonia has significantly improved in the past two decades. A major turning point has been the shift from the historical view of dystonia as a basal ganglia disorder to a model of large-scale brain network dysfunction [1, 2]. In fact, shared and distinct patterns of altered structural, functional, and metabolic brain network organization across different forms of dystonia have been identified [1]. This knowledge, along with genetic and extrinsic/environmental risk factors, contributes to the disorder's multifactorial nature. However, our knowledge of the brain networks involved in dystonia does not translate into effective treatment options based on these novel findings. In fact, botulinum toxin injections, primarily address temporary symptom management. Off-label medications are prescribed on a trial-and-error basis, and invasive neuromodulation techniques only reach a small percentage of dystonia patients. In 2020, a workshop organized by the National Institute of Neurological Disorders and Stroke emphasized the need to develop novel, effective therapeutic interventions informed by evidence of network-level alterations in dystonia [3]. This Special Issue asked how close the field is to that goal. It contains nine contributions (five reviews, two original studies, a brief research report, and one opinion paper) that tackle the dystonic network through different endpoints: peripheral afferents, cortex, basal ganglia and thalamus, monoaminergic transmission, and cerebellum. Two reviews map the broader landscape of clinical trials and non-motor symptoms.

Konczak et al. performed a systematic review of the effectiveness of superficial vibrotactile stimulation (VTS) in focal dystonia. The technique is grounded in the notion that peripheral stimulation influences central circuits by reducing excessive synchronization over the sensorimotor cortex. The review of empirical data provided evidence of short-term symptom improvements in focal dystonia, particularly in cervical and focal-hand dystonias, with positive outcomes reported in 57%–85% of study participants. This research group also applied this technique to laryngeal dystonia and reported positive results. The effect of short applications (4 s–15 min) decays within minutes, whereas the effect of longer applications (20–45min) persists for hours or days. However, optimal dosing, long-term efficacy, and predictors of response remain unknown and need further investigation.

Kamo et al. reviewed rehabilitation strategies in cervical, task-specific, and functional dystonias. The greatest benefits were observed when rehabilitation was combined with botulinum toxin injection, particularly in cervical and focal hand dystonias, with home-based exercises necessary to maintain gains. Sensorimotor retraining for task-specific dystonia is

promising but rests on studies at high risk of bias. Conventional speech and behavioral therapies are not considered effective for laryngeal dystonia (LD), leaving botulinum toxin injections as the treatment gold standard for LD. Finally, in functional dystonia, emerging evidence supports the role of a multimodal approach, combining physical therapy with cognitive behavioral therapy or mind-body strategies.

At the cortical level, Simsek et al. conducted a meta-analysis of the effectiveness of transcranial magnetic stimulation (TMS) in reducing symptoms of focal hand dystonia. Nine papers were examined, yielding a mild-to-moderate improvement of symptoms in active stimulation vs. sham or pre- vs. post-TMS. Most studies used low-frequency or inhibitory protocols targeting the premotor cortex (six studies) and the motor and somatosensory cortices (two studies each). These results may support larger trials to define the optimal target, protocol, and durability of effects.

Two contributions address deep-brain stimulation (DBS). A commentary by O'Flynn et al. assessed the findings of a randomized crossover trial of DBS of the ventral intermediate thalamic nucleus (Vim) in patients with LD and co-occurring voice tremor [4]. The authors concluded that the outcomes of this clinical trial should be interpreted with caution, given the diagnostic uncertainties of participants and associated target choice, unblinding, the absence of a washout period, and outcome measures that cannot separate dystonic from tremor-related voice symptoms. The case report by Remz et al. described three patients with DYT-11 myoclonus-dystonia who responded suboptimally to DBS, in contrast to the brisk response typically reported in this condition. Two patients improved substantially only after lead revision, and one required advanced programming. These cases underscore the need to tailor target selection and programming to specific symptoms and brain circuits rather than to the complex diagnostic label alone.

Aziz et al. tested valbenazine, a vesicular monoamine transporter 2 (VMAT2) inhibitor approved for tardive dyskinesia and Huntington's disease chorea, as an add-on to botulinum toxin (BoNT) injections in patients with cervical dystonia. In this small open-label phase 1 study, the Toronto Western Spasmodic Torticollis Rating Scale total score and the pulling and jerking ratings improved relative to matched phases of the BoNT cycle without valbenazine, whereas sleep and neck disability did not. Fatigue was the most common adverse effect. The preliminary results are promising and warrant further study, including a placebo-controlled arm, a larger sample size, and multiple treatment cycles.

Dystonia is often associated with overrepresentation of psychiatric comorbidities and suicidal ideation [5], making mood a key domain in the systematic review of non-motor symptom treatment performed by Sugar and Pirio Richardson. Most interventions were aimed primarily at motor symptoms, with non-motor benefits observed and tested only in a few cases. The authors call for in-depth detection, characterization, and treatment of non-motor symptoms in dystonia to achieve comprehensive treatment for patients. Ly et al. complement this picture with a narrative review of published, completed, or ongoing clinical trials between 2020 and 2025, summarizing recent advances in therapeutic developments for dystonia, including oral medications, toxin injections, surgical interventions, gene-targeted therapies, and rehabilitation approaches.

Finally, Fitzgerald et al. provided a preclinical framework for vocal abnormalities in a cerebellum-specific mouse model of generalized dystonia, offering a testbed for circuit-targeted interventions in voice motor control dysfunction.

Collectively, these contributions show that, although several options can improve symptoms, few are grounded in the current understanding of dystonia as a heterogeneous group of network disorders. Four main priorities emerge. First, given the heterogeneity in responses to current treatment options, it is necessary to investigate system-level dysfunctions using a multimodal approach that can stratify patients before treatment. Second, investigations into target selection, dose, and the durability of effects are warranted for both invasive and non-invasive neurostimulation techniques. Third, motor and non-motor symptoms should be assessed and treated together, given the frequent psychiatric comorbidities in dystonia. Finally, pharmacological investigations remain extremely scarce, and research in this field should draw on our current understanding of the disorder's pathophysiology. Addressing these priorities will help the field move from symptom management to network-informed therapy.

Author contributions

All authors listed have made a substantial, direct, and intellectual contribution to the work and approved it for publication.

Conflict of interest

The author(s) declared that this work was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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