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LETTER

Therapeutic deep brain stimulation worsening dysprosody in Parkinson's disease – an unexplored entity

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Dear editor

We read this article, "Altered emotional recognition and expression in patients with Parkinson's disease" by Jin et al[1] with great interest and appreciate the novel information provided on altered emotional processing in pre-deep brain stimulation (DBS) Parkinson's disease (PD) patients and we would like to add our feedback on the role of DBS on nonmotor and emotional components of PD.

PD is considered a movement disorder primarily presenting with motor symptoms like resting tremors, bradykinesia, rigidity, and postural instability. However, several nonmotor symptoms such as cognitive impairment, dementia, depression, anxiety, and apathy are also noted. Nonmotor symptoms may even dominate the clinical picture as disease progresses.[2]

The subthalamic nucleus (STN), in particular basal ganglia and amygdala, has an important role in the recognition of basic facial emotions both from face and music. Results from both pathophysiological and neuroimaging studies suggested this, as well as the fact that its involvement in PD frequently impairs the recognition of disgust and fear emotions, followed by sadness and anger.[3]

DBS treats motor symptoms, such as tremor, rigidity and bradykinesia, but may worsen certain nonmotor symptoms, including articulation of words and expressing emotions, resulting in dysprosody in the patients. The clinical evaluation of speech after 1 and 3 years of STN-DBS in PD patients showed a significant deterioration of speech. Articulation rather than voice was most frequently affected, with a distinct dysarthria. The mechanisms by which DBS exacerbates dysarthria while improving other symptoms could be partly attributed to medially placed electrodes and high amplitude of stimulation, but this is still unclear.[4] However, due to obvious ethical and experimental boundaries in human studies, recent observations that hypokinetic or hyperkinetic dysarthria may be worsened by otherwise therapeutic DBS are largely unexplored.

King et al[5] presented a rodent model of Parkinsonian dysarthria that is worsened by DBS, thus providing a stepping stone for future research. They proposed this study using the existing 6-hydroxydopamine rodent model of Parkinsonism to observe hypokinetic dysarthria and its exacerbation by DBS. Stimulation of the STN aggravated these Parkinsonian vocalization symptoms. Vocalization complexity and rates, word durations, and individual sound bandwidths were all detrimentally compounded

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after DBS. The rats generated scant and shorter vocalizations that were less acoustically complex than those under the diseased condition.

This model will provide a framework for exploration into the electrophysiological mechanisms of Parkinsonian hypokinetic dysarthria and its exacerbation after DBS. Knowing how DBS therapy can intensify dysarthria may lead researchers to devise alternative approaches to treat motor and speech signs together. Such explorations are essential to minimize this side effect of DBS that worsens dysarthria, exacerbating Parkinsonian speech deficits that can greatly diminish patients' emotional recognition, expression, and quality of life.

Disclosure

The authors report no conflicts of interest in this communication.

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