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Review Article

Deep Brain Stimulation in Parkinson's Disease: Mechanisms, Targets, and Clinical Outcomes

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Abstract

Parkinson's disease (PD) is a progressive neurodegenerative disorder that primarily affects motor function due to the loss of function of the dopamine producing brain cells in substantia nigra. New neurosurgical interventions have emerged, including Deep Brain Stimulation, particularly when pharmacological treatments become less effective. This paper discusses the mechanism of DBS, brain structures involved, and the clinical results associated with its application in PD patients.

Keywords: *Parkinson's Disease (PD); Deep Brain Stimulation; Sub Thalamic Nucleus (STN); Globus Pallidus Internus (GPi); Ventral Intermediate Nucleus (VIM)*

Introduction

Parkinson's disease results from a progressive loss of dopaminergic neurons in the substantia nigra of basal ganglia, leading to inhibition of the nigrostriatal pathway. When pharmacological treatment fails, DBS can be used as an alternative [1]. DBS delivers high-frequency electrical impulses are delivered by DBS to important brain regions such as the sub thalamic nucleus (STN), the Globus pallidus internus (GPi), and the thalamic ventral intermediate nucleus (VIM) [2].

Mechanisms of deep brain stimulation

- Local inhibition of pathological firing: DBS can suppress abnormal neuronal firing, acting like a reversible lesion [3].
- Axonal activation: by stimulating nerve fibers affecting both local circuits and distant structures [4].
- Oscillatory modulation: by decreasing excessive beta-band oscillations and restoring gamma/theta wave activity linked to movement [5,6].
- Neurochemical effects: by altering dopamine, GABA, and glutamate release [4,7].

Target structures and symptom control

- Sub thalamic nucleus (STN): Improves tremor, rigidity, bradykinesia, and dyskinesia; enables significant medication reduction [2,5].
- Globus pallidus internus (GPi): Especially effective for dyskinesia and rigidity; may have milder effects on cognitive functions [1,8].
- Thalamus (VIM): Used mainly for tremor-dominant PD resistant to medications [2].

Clinical outcomes

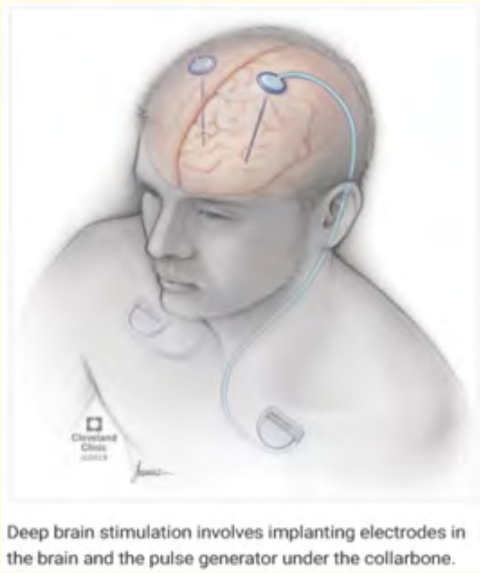
Both STN and GPi DBS significantly improve motor scores:

- 40 - 60% improvement in UPDRS motor scores [8].
- A ~50% levodopa medication dose reduction has been shown with STN-DBS [1,5].
- Dyskinesia relief by GPi-DBS [8].
- Long-term benefits can persist for up to 5 - 10 years, keeping in mind that non-dopaminergic symptoms may worsen [9,10].

Adverse effects: Cognitive and mood-related side effects have been documented. However temporary [9].

Conceptual framework

The concept of PD framework is dependent on direct and indirect pathways of basal ganglia where in PD there is an imbalance directed toward increased inhibition of thalamus [6,7]. This imbalance is reset by high frequency impulses sent through stimulation leads placed on a particular area of brain, while being attached to an implantable battery or a pulse generator [10].



Figure

Discussion

For most patients, the ability to have control over everyday activities makes DBS a life-changing option. However, DBS is not the solution for everything. One of the frequent questions is the possibility of cognitive and emotional harms, particularly when the sub thalamic nucleus (STN) is attacked. After surgery, several patients have trouble with verbal fluency or elevated impulsivity [13].

The timing of DBS is also a topic of fresh research. Traditionally, DBS was only available to patients in the diseases later stages. However, the findings from the EARLYSTIM trial showed that earlier intervention could result in improved long-term outcomes, particularly in terms of retaining independence and quality of life [11].

This means that physicians may have to reconsider when to introduce surgical options, as waiting too long could result in a lack of a window where the brain is most responsive.

So far, the results here are mixed [12], but it could be a promising field for future research.

Conclusion

DBS represents a powerful intervention in the treatment of advanced PD. By regulating abnormal neuronal activity and improving patient health outcome by providing symptomatic relief. With the ongoing rise in technology, newer interventions could be seen later.

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