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ELECTRICAL NEUROMODULATION IN THE SURGICAL MANAGEMENT OF PARKINSON'S DISEASE

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Abstract

Parkinson's disease is a progressive neurodegenerative disorder characterized by the degeneration of dopaminergic neurons and the accumulation of α-synuclein protein aggregates in the brain. It leads to a variety of motor symptoms, including tremor, rigidity, bradykinesia, and postural instability, as well as numerous non-motor manifestations. Pharmacological therapy, particularly treatment with levodopa, remains the main approach for symptomatic control; however, long-term drug therapy is frequently associated with the development of motor fluctuations and dyskinesia, which significantly complicate disease management in advanced stages. In recent decades, deep brain stimulation has emerged as one of the most important neurosurgical approaches for the treatment of advanced Parkinson's disease in patients whose symptoms cannot be adequately controlled with pharmacological therapy alone. This technique involves the delivery of controlled electrical impulses to specific brain structures, most commonly the subthalamic nucleus, in order to modulate abnormal neural activity within the basal ganglia–thalamocortical motor circuits. By altering pathological patterns of neuronal firing, deep brain stimulation can restore more physiologically organized neural signaling within motor pathways. Clinical studies have

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demonstrated that deep brain stimulation significantly improves motor function, reduces motor complications, increases "on" time without troublesome dyskinesia, and enhances patients' quality of life. Long-term follow-up investigations also show sustained therapeutic effects together with a reduction in antiparkinsonian medication requirements. The present article examines the epidemiology and pathophysiology of Parkinson's disease in the context of abnormal neuronal electrical activity and analyzes the application of deep brain stimulation as a modern neurosurgical method that utilizes electrical impulses to modulate pathological brain circuits in the treatment of Parkinson's disease.

Keywords: Parkinson's disease; deep brain stimulation; neuromodulation; levodopa; basal ganglia; subthalamic nucleus; motor fluctuations; dyskinesia; neurosurgical treatment.

Introduction

Parkinson's disease (PD) is a progressive neurodegenerative disorder clinically characterized by asymmetric bradykinesia, rigidity, tremor, and postural instability. Pathologically, it is associated with the degeneration of dopaminergic neurons in the substantia nigra pars compacta and the accumulation of misfolded α-synuclein aggregates (Lewy bodies) within the central and peripheral nervous systems. The global burden of PD continues to rise, with an estimated 6.1 million individuals affected worldwide in 2016—more than double the number reported in 1990.

Pharmacological management of PD primarily aims to alleviate motor symptoms and is largely based on evidence from clinical trials combined with clinical experience. Levodopa remains the most effective first-line therapy for motor manifestations, particularly bradykinesia and rigidity, although tremor may be less responsive in some patients. However, long-term levodopa therapy is frequently associated with dose-related adverse effects, including dyskinesia,

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hallucinations or behavioral disturbances, orthostatic hypotension, and nausea. In addition, the duration of symptomatic benefit ("on" time) typically shortens after several years of treatment, leading to motor fluctuations characterized by alternating "on" and "off" periods. These fluctuations are thought to result from the short half-life of levodopa, variable gastrointestinal absorption, and progressive dopaminergic neuronal degeneration (Tanner & Ostrem, 2024). Current clinical guidelines from the American Academy of Neurology and the National Institute for Health and Care Excellence recognize levodopa as the most effective symptomatic therapy for early PD, while noting that dopamine agonists may be considered as initial therapy in selected patients at higher risk of dyskinesia (Foltynie et al., 2024).

In contrast to pharmacological therapy, deep brain stimulation (DBS) has emerged as an effective surgical treatment for patients with advanced PD. DBS has been shown to significantly improve quality of life, reduce motor fluctuations, and increase daily "on" time by approximately 3–4 hours. It also improves motor performance during medication-off states, with reductions of 30–50% in the Unified Parkinson's Disease Rating Scale (UPDRS) part III scores. Furthermore, DBS allows a substantial reduction in antiparkinsonian medication doses, often by up to 50%, particularly when targeting the subthalamic nucleus and after optimization of stimulation parameters during the first 3–6 months following surgery (Tanner & Ostrem, 2024). Evidence from a recent randomized 12-week blinded trial evaluating long-term outcomes and safety of subthalamic nucleus DBS (STN-DBS) demonstrated sustained improvements in motor function and activities of daily living, as well as a stable reduction in antiparkinsonian medication over a 5-year follow-up period, despite the progressive nature of the disease (Starr et al., 2025).

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The aim of this article is to analyze the application of electrical impulses in the neurosurgical treatment of Parkinson's disease, with particular emphasis on deep brain stimulation as a method of electrical neuromodulation capable of modifying pathological neuronal activity within the basal ganglia–thalamocortical circuits and thereby improving motor function and quality of life in patients with Parkinson's disease.

Epidemiology

The global prevalence of Parkinson's disease (PD) has been rising rapidly; in 2016 an estimated 6.1 million individuals worldwide were affected, representing more than a twofold increase compared with 1990 (Armstrong & Okun, 2020). The incidence of Parkinson's disease has generally been reported to be higher among White persons than among Black or Asian persons; however, the prevalence of Lewy bodies identified at autopsy — a defining pathological hallmark of Parkinson's disease — has been found to be comparable between Black and White individuals (Tanner & Ostrem, 2024). In the United States, approximately 930,000 people were living with PD in 2020 (Armstrong & Okun, 2020). In Europe, prevalence estimates range from 108–257 per 100,000 population, with annual incidence rates of 11–19 per 100,000 (Balestrino & Schapira, 2020). Parkinson's disease is uncommon before the age of 50, and its prevalence increases progressively with advancing age, reaching a peak between 85 and 89 years. The condition is observed more frequently in men, with a male-to-female ratio of approximately 1.4:1 (Armstrong & Okun, 2020; Balestrino & Schapira, 2020).

Causes of Parkinson's disease

When examining the etiology of Parkinson's disease (PD), three principal factors are generally considered: genetic influences, environmental exposures, and the interactions between them. Genetic variants with substantial effect sizes have

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been identified in approximately 20% of individuals with Parkinson's disease, representing cases of monogenic Parkinson's disease. Autosomal dominant forms with incomplete penetrance include variants in LRRK2 — leucine-rich repeat kinase 2 — which are present in approximately 1–2% of all cases and in up to 40% of familial cases, as well as variants in GBA1, encoding glucocerebrosidase. Recessively inherited forms of Parkinson's disease include variants in PRKN, PINK1, and DJ1, which account for the majority of cases with early disease onset. Although each of these variants is relatively infrequent, in certain populations they represent the most common genetic causes of the disorder. Abnormal α-synuclein is detected in Parkinson's disease associated with SNCA (α-synuclein) or GBA1 and in approximately half of the cases associated with LRRK2, whereas it is rare in Parkinson's disease associated with recessive variants. In addition, recessively inherited Parkinson's disease is characterized by fewer nonmotor manifestations and more prominent dystonia compared with the typical clinical presentation of the disorder (Tanner & Ostrem, 2024).

Positioned conceptually between a causative genetic factor with markedly reduced penetrance and a strong genetic susceptibility factor, pathogenic GBA variants were identified in 8.5% of individuals with Parkinson's disease in a multi-ethnic cohort of more than 1100 participants. The phenotype associated with GBA-linked Parkinson's disease is characterized by an earlier age at onset and a more severe clinical course, particularly with respect to rapid cognitive decline. Furthermore, when considering genetic variants with typically much smaller individual effect sizes, the largest meta-analysis of genome-wide association studies identified 90 independent genome-wide statistically significant risk signals. Collectively, these signals account for approximately 16–36% of the heritable risk of Parkinson's disease (Bloem, Okun, & Klein, 2021).

In addition to genetic susceptibility, environmental toxicants are recognized as major contributors to the development of Parkinson's disease. Among the most significant of these are certain pesticides, industrial solvents such as

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trichloroethylene (TCE), and air pollution. Numerous epidemiological investigations and experimental studies in animal models support a causal association between exposure to specific pesticides and the development of Parkinson's disease. An important observation supporting this relationship emerged in the 1980s, when seven young adults developed severe parkinsonism subacutely after intravenous administration of the designer drug 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP). Neurodegeneration of nigrostriatal dopaminergic neurons occurs after MPTP is metabolized into 1-methyl-4-phenylpyridinium (MPP+), which induces dopaminergic neuronal death through inhibition of mitochondrial complex I. Notably, MPP+ demonstrates a striking structural similarity to paraquat, one of the most widely used herbicides worldwide, which similarly disrupts mitochondrial function.

Industrial solvents also represent significant environmental risk factors. Trichloroethylene (TCE), a well-established carcinogen, has been recognized for its toxic properties since at least 1932. Similar to many pesticides, TCE acts as a mitochondrial toxicant by inhibiting complex I of the respiratory chain. Recent experimental research has further demonstrated that, analogous to certain LRRK2 mutations, TCE can induce LRRK2 kinase activity in the brains of rats and lead to degeneration of nigrostriatal dopaminergic neurons. Supporting the potential relevance of this mechanism in humans, a study conducted by Gash and colleagues in 2008 reported that among 30 factory workers, three individuals developed Parkinson's disease after prolonged occupational exposure to TCE used for degreasing and cleaning metal gauges. These three workers were stationed closest to an open vat of TCE, whereas 14 of the remaining 27 workers who were positioned farther from the source exhibited multiple features of parkinsonism, including marked motor slowing (Dorsey & Bloem, 2024).

Airborne pollutants may also contribute to Parkinson's disease pathogenesis through mechanisms involving the olfactory system. The olfactory pathway represents a potential entry route for inhaled pollutants that bypasses the blood–

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brain barrier (BBB), and it has been proposed as a pathway for the transmission of pathogenic α-synuclein to the brain. Consequently, it is plausible that airborne pollutants may initiate α-synuclein neuropathology within the olfactory system, which may subsequently progress to Parkinson's disease in the context of aging-related vulnerability of the brain. In support of this hypothesis, α-synuclein aggregates have been identified in the olfactory bulbs of toddlers, children, adolescents, and young adults who died prematurely in Mexico City, an urban environment characterized by high levels of air pollution (Chen & Ritz, 2018).

Pathophysiology of Parkinson's Disease

Electrical impulse transmission between neurons enables rapid communication within the brain and forms the physiological basis for the regulation of behavior, cognition, emotion, and sensory perception. A key molecular component involved in synaptic transmission is the protein α-synuclein. The abnormal accumulation of misfolded α-synuclein within intracellular inclusions known as Lewy bodies represents a characteristic pathological feature and diagnostic hallmark of idiopathic Parkinson's disease, particularly within the nigrostriatal system (Rajput & Noyes, 2024).

α-Synuclein has long been recognized as the principal proteinaceous component of Lewy bodies (LB). Ultrastructural analyses have demonstrated that LBs are composed of fibrillar outer regions and granular cores containing proteins, as well as lipids and organelles with diverse reported morphologies. More recent investigations indicate that LBs resemble caged wheel–like structures in which negatively charged species of α-syn (pS129-α-syn) form the outer layer of the aggregate, whereas hydrophobic species of α-syn (C-terminal truncated) are located closer to the central region, which is composed predominantly of lipids.

Further studies have shown that this lipid-rich core consists primarily of mitochondrial and autophagosomal membranes. These findings suggest that α-

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syn toxicity may occur through dysregulation of these organelles, a hypothesis that has been supported by recent investigations using neuronal culture models.
In addition to the cellular mechanisms of α-synuclein aggregation, increasing attention has been directed toward the potential anatomical origins and propagation pathways of α-synuclein pathology. The enteric nervous system has been proposed as one possible initial site of pathological α-synuclein accumulation. This hypothesis is supported by several observations: constipation is a recognized prodromal symptom of Parkinson's disease; epidemiological associations have been reported between inflammatory bowel diseases and Parkinson's disease; and pathogenic α-synuclein aggregates have been identified within the enteric nervous system of affected individuals. According to the proposed propagation model, pathological α-synuclein may spread from the enteric nervous system through the vagus nerve to the dorsal motor nucleus of the vagus located in the brainstem. From this region, the pathology may progressively extend to other brainstem structures and eventually to higher brain regions in accordance with the Braak staging model of Parkinson's disease. Another potential site of early pathological initiation is the olfactory system. This possibility is supported by the high prevalence of anosmia among patients with Parkinson's disease and by observations that α-synuclein pathology in individuals corresponding to Braak stage 1 can involve the anterior olfactory nucleus. (Vázquez-Vélez & Zoghbi, 2021).

Pharmacological treatment of Parkinson's disease

The first and most significant therapeutic breakthrough in the management of Parkinson's disease occurred with the introduction of l-dopa in the 1960s. This treatment improves motor symptoms and, to a considerable extent, associated non-motor manifestations of the disease. Nevertheless, debate persists within the scientific community regarding the long-term use of l-dopa, primarily due to the

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development of fluctuations in motor behavior, potential acceleration of aging processes, and concerns regarding possible neurotoxic effects of l-dopa.

Multiple lines of evidence indicate that continuous delivery of dopamine-replacing medications represents an essential prerequisite for maintaining near-normal motor function in patients with Parkinson's disease. Consequently, pharmacological agents with longer half-lives, including delayed-release formulations, offer certain advantages. However, the compensatory capacity of the central nervous system to accommodate synaptic dopamine oscillations associated with persistent oral l-dopa administration gradually declines after an individually variable period of treatment. As a result, patients progressively develop so-called motor complications.

These complications are characterized by alternating phases of adequate motor function, recurrence of motor impairment (commonly referred to as the OFF state), and periods of excessive dopaminergic stimulation that lead to involuntary movements known as dyskinesia. Such fluctuations in motor activity are frequently accompanied by non-motor fluctuations. For example, apathy often occurs during OFF states, whereas dyskinesia may be associated with manic episodes. Importantly, patients generally tolerate OFF episodes less well than dyskinesia. Accordingly, the management of these fluctuations in both motor and non-motor manifestations remains a major focus of current pharmacological research in Parkinson's disease.

In response to these challenges, therapeutic innovation has concentrated on the development of new formulations of l-dopa, dopamine agonists, and amantadine with improved pharmacokinetic profiles. In addition, continuous drug delivery approaches, such as subcutaneous administration of apomorphine or intracerebral delivery of l-dopa via pump devices, have been shown to significantly reduce motor fluctuations.

At the same time, certain metabolic consequences associated with long-term therapy have been investigated. Chronic treatment with l-dopa in combination

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with dopa decarboxylase inhibitors (DDI) has been shown to reduce methylation capacity in patients with Parkinson's disease. This methylation deficit may contribute to the acceleration of aging processes, neuronal dysfunction, and increased generation of oxidative stress (Müller, 2025).

DBS as a contemporary treatment of Parkinson's disease

Several hypotheses have been proposed to explain the mechanisms underlying deep brain stimulation (DBS); however, its precise mode of action remains incompletely understood. One proposed mechanism suggests that DBS suppresses pathological beta-band oscillatory activity (13–30 Hz) observed in Parkinson's disease and promotes higher-frequency neuronal firing. The most commonly targeted structures include the subthalamic nucleus (STN), the globus pallidus internus (GPi), and the caudal zona incerta (cZi). Through this process, DBS may also activate adjacent fiber tracts that modulate the basal ganglia–thalamocortical network. Additionally, it has been proposed that DBS alters both the firing rate and the firing pattern of individual neurons within the basal ganglia. In this context, the abnormal and irregular neuronal activity characteristic of Parkinson's disease is replaced by a more regular stimulus-induced pattern, which limits the propagation of pathological firing and leads to improved processing of sensorimotor information and, consequently, alleviation of motor symptoms (Bratsos, Karponis, & Saleh, 2018).

Evidence supporting the clinical effectiveness of subthalamic nucleus deep brain stimulation has been provided by the INTREPID study, a multicenter, double-blind, randomized (3:1), sham-controlled clinical trial designed to evaluate both the short-term and long-term outcomes of DBS therapy in patients with Parkinson's disease. The trial consisted of an initial 12-week blinded controlled phase, followed by a long-term open-label extension that evaluated clinical outcomes over a period of up to five years. The results obtained during the blinded phase demonstrated significant improvements in "on" time without troublesome dyskinesia, motor scores measured during medication-off states, and patient-

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reported quality-of-life outcomes in individuals receiving active stimulation compared with the control group. Following completion of the blinded phase, participants entered the open-label follow-up stage, during which all individuals received active stimulation and were monitored for long-term safety and therapeutic effectiveness.

The study was conducted as a multicenter, parallel-design, double-blind, randomized, sham-controlled trial at 23 sites across the United States between May 2013 and December 2022. Approval for the study was obtained from the U.S. Food and Drug Administration and from the institutional review boards at each participating center. Written informed consent was obtained from all participants prior to enrollment, and all study data were collected by site personnel according to the established study protocol. Key inclusion criteria included age between 22 and 75 years, a diagnosis of bilateral idiopathic Parkinson's disease with more than 5 years of motor symptoms, more than 6 hours per day of poor motor function, stable antiparkinsonian medication use for at least 28 days prior to consent, a modified Hoehn and Yahr Scale score greater than 2, a Unified Parkinson's Disease Rating Scale (UPDRS-III) score of 30 or higher in the medication-off condition, and at least a 33% improvement in the UPDRS-III score during the medication-on state. The trial was conducted in accordance with ISO 14155 standards for clinical investigation of medical devices in human subjects and with 21 CFR Part 812 regulations. The study was also registered with ClinicalTrials.gov.

A total of 313 participants provided informed consent, 210 underwent screening procedures, and 193 participants were implanted with the Vercise DBS system (Boston Scientific). A detailed clinical protocol describing eligibility criteria, screening procedures, randomization, blinding, and evaluation of the primary endpoint had been previously published. Patients meeting the eligibility criteria were randomized and evaluated during the blinded controlled phase of the study. After completion of the 12-week blinded phase, 191 randomized participants

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transitioned to the open-label phase, during which individuals in the control group also received active stimulation. During this period, follow-up visits were scheduled at post-randomization weeks 20, 26, 48, and 52 (±14 days), as well as at week 78 (±28 days), and at years 2, 3, 4, and 5 (±28 days).

Clinical outcomes assessed over the 5-year follow-up period included the Unified Parkinson's Disease Rating Scale (UPDRS) parts I–IV, with UPDRS-II and UPDRS-III evaluated in both medication-on and medication-off states. Additional measures included changes in antiparkinsonian medication usage expressed as levodopa equivalent daily dose (LEDD), the Clinical Dyskinesia Rating Scale (CDRS), the Clinical Global Impression of Change (CGIC), the Parkinson's Disease Questionnaire-39 (PDQ-39), and the Treatment Satisfaction Questionnaire. Continuous variables were summarized using descriptive statistics, including the number of nonmissing observations, means, standard deviations (SDs), minimum and maximum values, and 95% confidence intervals (CIs). Categorical variables were presented as frequencies and percentages. Both primary and secondary outcomes were analyzed according to the intention-to-treat principle, including all randomly assigned participants. Individuals with major predefined protocol deviations were excluded from the per-protocol analysis, whereas the safety analysis included all enrolled patients.

For endpoints evaluating mean changes from screening to years 1 through 5, a linear mixed model for repeated measures with an autoregressive covariance structure was applied to compare results across time points while adjusting for study site. The model included fixed effects for visit and study site. Observed P values comparing screening versus year 1 and screening versus year 5 were reported for the following assessments: UPDRS (including part III motor subscores for tremor, bradykinesia, rigidity, and gait), PDQ-39, CDRS, and LEDD. Estimates and standard errors of the mean (SEM) were generated for each scheduled study visit using the repeated-measures model. Results were presented in box-and-whisker plots with tails representing the 5th to 95th percentiles. All

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statistical analyses were conducted using SAS version 9.4 (SAS Institute). Safety outcomes were evaluated by coding all adverse events according to MedDRA version 10.1 (International Council for Harmonisation), and a two-sided P value of less than .05 was considered statistically significant. UPDRS-III motor subcategories, including rest tremor, postural tremor, rigidity, bradykinesia, and gait, were also analyzed. Long-term outcomes stratified according to medication and stimulation status are described in Supplement 2. Safety parameters included the incidence of adverse events (AEs) and serious adverse events (SAEs) throughout the study period.

Results: A total of 193 participants were implanted with a deep brain stimulation implantable pulse generator (IPG) with bilateral leads placed in the subthalamic nucleus (STN). Among these participants, 191 were randomized to receive either active therapeutic stimulation or subtherapeutic stimulation settings (control group). Of the 191 randomized participants, 137 (72%) completed the long-term follow-up according to the study schedule. Forty-five participants withdrew from the study, most commonly for voluntary reasons (29 participants).

The study cohort included 139 men (73%) and 52 women (27%). Participants had a diagnosis of bilateral idiopathic Parkinson's disease with a mean (SD) disease duration of 10.3 (3.8) years and a mean (SD) Hoehn and Yahr score of 2.8 (0.7). At screening, the mean (SEM) Unified Parkinson's Disease Rating Scale part III (UPDRS-III) score in the medication-off state was 42.8 (0.68), indicating moderate disease severity. Compared with the medication-off condition, mean UPDRS-III scores improved by 57.5% (SEM, 0.58) in the medication-on state. As previously reported, the INTREPID study achieved its primary endpoint ($P < .001$), demonstrating a mean (SD) increase of 3.03 (4.52) hours in "on" time without troublesome dyskinesia between baseline and 3 months after randomization in the active stimulation group compared with the control group (Starr et al., 2025).

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Conclusion

Parkinson's disease remains a progressive neurodegenerative disorder characterized by complex pathological mechanisms involving dopaminergic neuronal degeneration and the accumulation of misfolded α-synuclein aggregates. These processes disrupt normal neuronal communication and contribute to the development of both motor and non-motor manifestations that define the clinical course of the disease.

Although pharmacological therapy, particularly treatment with levodopa, remains the primary method for symptomatic management of Parkinson's disease, long-term pharmacotherapy frequently leads to motor complications, including motor fluctuations and dyskinesia. As the disease progresses, these complications may become increasingly difficult to control through medication alone.

In this context, deep brain stimulation represents a highly effective neurosurgical intervention for patients with advanced Parkinson's disease who experience insufficient symptom control with pharmacological therapy. By delivering controlled electrical impulses to specific structures within the basal ganglia network, DBS modulates abnormal neuronal activity and restores more physiologically organized patterns of neural signaling.

Clinical evidence from long-term studies demonstrates that deep brain stimulation can significantly improve motor performance, increase "on" time without troublesome dyskinesia, reduce the severity of motor fluctuations, and decrease the required dosage of antiparkinsonian medications. These benefits contribute to substantial improvements in functional capacity and quality of life for patients with advanced Parkinson's disease.

Overall, the application of electrical impulses through neuromodulation techniques such as deep brain stimulation represents one of the most important advances in the neurosurgical treatment of Parkinson's disease. Continued research aimed at improving stimulation technologies, optimizing targeting strategies, and better understanding the neurophysiological mechanisms of DBS

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may further enhance its therapeutic effectiveness and expand its role in the management of this complex neurological disorder.

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