# AU InforMed

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***Key Inforbits***

- What is Parkinson's Disease?
- History of Parkinson's
- Deep Brain Stimulation
- New Pharmacological Advancements
- FDA Special Alert
- Role of Pharmacists


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## What is Parkinson's Disease?

Parkinson's Disease (PD) is a neurodegenerative disorder affecting the dopamine-producing ("dopaminergic") neurons in the brain.[1,2] These dopaminergic neurons weaken over time, becoming damaged and eventually dying **(Figure 1)**. This leads to movement-related ("motor") symptoms such as tremor, slowness and paucity of movement (bradykinesia and hypokinesia), limb stiffness (rigidity), and gait/balance problems (postural instability). Parkinson's symptoms may also be unrelated to movement ("non-motor"), including depression, anxiety, apathy, hallucinations, constipation, orthostatic hypotension, sleep disorders, loss of sense of smell, and a variety of cognitive impairments. The progression of symptoms can be different person to person, but most people with Parkinson's ultimately have difficulty walking, talking, or completing other simple tasks.

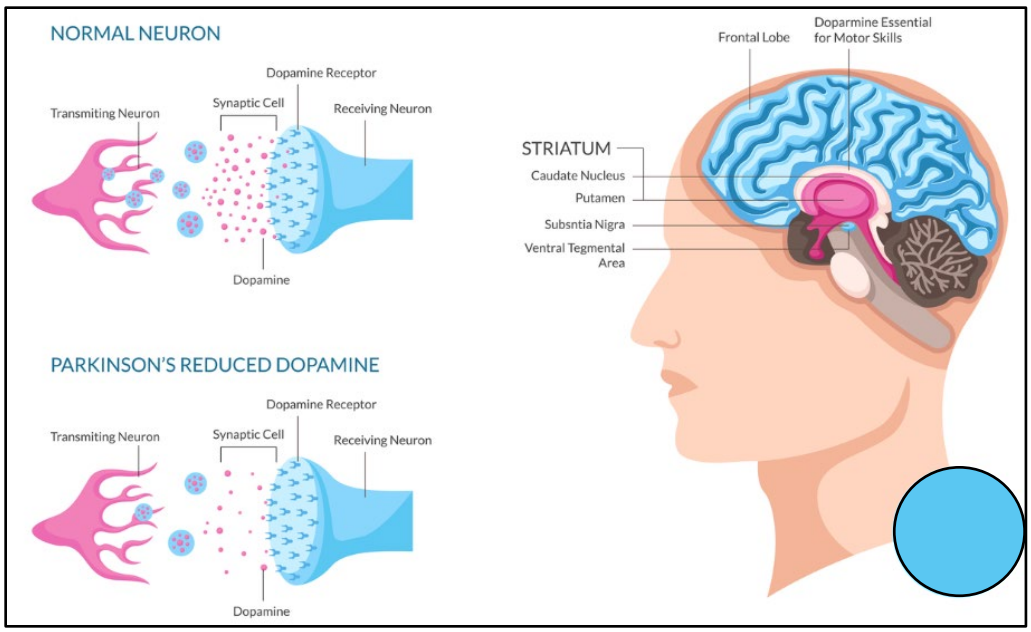


Figure 1. Parkinson's. Adapted from: https://www.fionawaring.com/recent-articles/parkinsons

## History of Parkinson's

### *Early Description and Diagnosis*

Parkinson's Disease has been recognized for centuries, with descriptions of Parkinson-like symptoms appearing in ancient medical texts from China and India.[3,4] However, the disease was first clearly defined in 1817 by James Parkinson as a neurological syndrome in "*An Essay on the Shaking Palsy*." In the late 1800s, Jean-Martin Charcot refined the clinical understanding of the disease by distinguishing it from other neurological disorders, identifying bradykinesia as a key feature, and formally establishing the term "Parkinson's disease." Diagnosis has historically been based on clinical observation of motor symptoms, and although diagnostic criteria have expanded to include non-motor features, it remains primarily a clinical diagnosis today.

### *Advances in Treatment*

Early treatment approaches in the 1800s and early 1900s were limited and often ineffective, including the use of anticholinergic agents, herbal remedies, and mechanical therapies.[3,4] A major scientific breakthrough occurred in the 1960s with the discovery that dopamine deficiency was associated with PD, leading to the development of levodopa. Levodopa remains the cornerstone of treatment, with additional therapies such as dopamine agonists and monoamine oxidase B (MAO-B) inhibitors developed to improve symptom control. Surgical advancements followed, most notably deep brain stimulation, which became widely used in the late 20th century for patients with advanced disease.

***Progress in Research***

Key discoveries include the identification of degeneration in the substantia nigra, the presence of Lewy bodies, and the role of dopamine in motor control.[3,4] More recent advances have focused on the role of alpha-synuclein, genetic mutations associated with familial PD, and the contribution of environmental factors. Emerging evidence suggests that disease progression may involve the spread of abnormal proteins through the nervous system. Ongoing research efforts are focused on identifying early biomarkers, improving diagnostic accuracy, developing disease-modifying therapies, and exploring innovative approaches such as gene therapy and immunotherapy. No cure currently exists and treatment remains focused on symptom management and improving quality of life.

## Deep Brain Stimulation

Deep brain stimulation (DBS) is an FDA-approved treatment for people with advanced Parkinson's disease whose symptoms are no longer well controlled with medication or who experience significant medication side effects.[5-7] The procedure involves implanting thin wires (electrodes) in specific brain regions—most commonly the globus pallidus internus (GPi), subthalamic nucleus (STN), or ventral intermediate nucleus (VIM)—which are connected to a small device placed under the skin near the collarbone that delivers electrical pulses to regulate abnormal brain signaling and improve communication between brain cells **(Figure 2)**. This can reduce motor symptoms such as tremor, stiffness, slowness, and dyskinesia, and may allow for lower medication doses. While DBS is generally safe and can provide long-lasting improvement in movement and quality of life, it is not a cure and does not stop disease progression; some symptoms like balance, gait, and cognitive changes may become less responsive over time, and it is not recommended for patients with dementia due to potential worsening of thinking or memory.

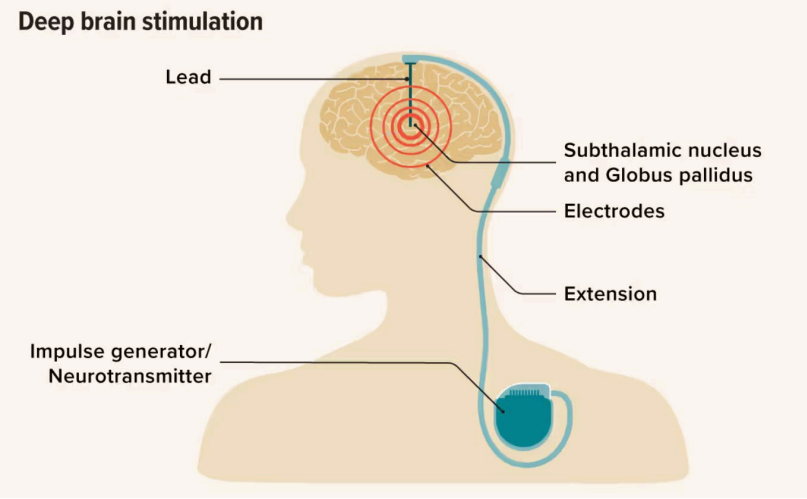


Figure 2. Deep brain stimulation: how it works. *Reproduced from Healthline*[8]

A nonrandomized clinical trial published in *JAMA Neurology* in September 2025 evaluated personalized adaptive deep brain stimulation (aDBS) in 68 patients with Parkinson's disease, a newer approach that adjusts stimulation in real time based on brain activity rather than delivering constant stimulation.[9] The study found that aDBS was effective in improving motor symptoms and maintaining symptom control over time, with sustained benefits observed during an average follow-up of about 17.5 months. In terms of safety, most adverse events were mild, stimulation-related, and occurred during the initial programming phase, with nearly all resolving after device adjustments; importantly, no unexpected serious device-related adverse events were reported, supporting a favorable safety profile.

## New Pharmacological Advancements

### Tavapadon

In September 2025, AbbVie submitted a New Drug Application for tavapadon, an oral, once-daily, selective D1/D5 agonist for the treatment of Parkinson's disease motor symptoms.[10] This medication underwent three, Phase 3 randomized clinical trials (TEMPO-1, TEMPO-2, and TEMPO-3) prior to submission.[11-13] All three trials were multicenter, double-blind, placebo-controlled trials, but TEMPO-1 had fixed-dosing and TEMPO-2 and TEMPO-3 had flexible dosing. TEMPO-1 (n=529) and TEMPO-2 (n=232) demonstrated the effectiveness of tavapadon at both 5 mg and 15 mg doses in patients with early Parkinson's disease, while TEMPO-3 (n-507) evaluated tavapadon as adjunctive therapy to levodopa, a common approach in current clinical practice. All three trials demonstrated the safety of the medication with main side effects including nausea, headache, and dizziness. TEMPO-3 did note cases of dyskinesia when used in conjunction with levodopa therapy. Most recently, a 58-week open-label trial of tavapadon in Parkinson's disease (TEMPO-4), completed in December 2025 with results pending, assessed the safety and efficacy of long-term administration of tavapadon in flexible doses (5 mg to 15 mg).[14] Overall, this medication shows promise in becoming a new therapy in Parkinson's disease treatment.

### Crexont (carbidopa/levodopa)

In August 2024, the FDA approved a new, extended-release capsule formulation of carbidopa and levodopa to improve time without dyskinesia.[15] This medication has similar levodopa plasma concentrations compared to immediate release formulations currently in clinical practice but maintains concentrations for a longer duration (approximately 1.6 hours longer).[16] Crexont can be used in both treatment- naive patients and patients initially on immediate-release (IR) formulations who planned to convert to the extended-release (ER) formulation. Conversions from IR to ER is not a 1:1 ratio, but conversion from Rytary ER capsules to Crexont ER capsules are interchangeable 1:1 **(Table 1)**.[17,18] Crexont may be used up to a maximum of carbidopa 525 mg/levodopa 2.1 grams per day, in up to 4 divided doses.[18] In the RISE-PD trial, patients

receiving Crexont had a 23% increase in waking up with good movement compared to a 10% increase in IR formulations ($p < 0.01$).[19] Additionally, at the start of the study more patients reported never having good movement upon waking compared to the end of the study when Crexont demonstrated a higher percent decrease compared to IR formulations (Crexont: 73% to 47%; IR: 71% to 59%; $p < 0.01$).[19] To note, dyskinesia, dizziness, nausea, sleep disturbances, and hallucinations led to discontinuation of the new therapy during IR to ER dose conversions.[17] Since many patients will have concurrent medication therapies, it is important to note that Crexont is contraindicated in patients currently taking a non-selective MAO inhibitor or have recently used within 2 weeks of initiation of therapy.[17]

| Total Daily Immediate-Release Levodopa Dosage | Most Frequent Immediate-Release Levodopa Single Dose | Recommended Starting CREXONT Dosage of Levodopa |
|---|---|---|
| Less than 500 mg daily | 100 mg | 280 mg twice daily |
| | 150 mg | 420 mg twice daily |
| | 200 mg | 560 mg twice daily |
| Equal to or greater than 500 mg daily | 100 mg | 280 mg three times daily |
| | 150 mg | 420 mg three times daily |
| | 200 mg | 560 mg three times daily |
| | Greater than 200 mg | 700 mg three times daily |

Table 1: Dose Conversion Table[17]

## New FDA Special Alert[20]

On March 20th, 2026, the FDA conducted a safety review of all carbidopa/levodopa medications and identified 14 cases of seizures linked to vitamin B6 deficiency. It is now recommended that healthcare providers evaluate a patient's vitamin B6 level prior to initiation of therapy and during treatment as levodopa/carbidopa can decrease vitamin B6 levels in the body. If a patient has vitamin B6 deficiency, the healthcare provider should supplement the vitamin B6 as necessary.

## YOUR role as a pharmacist[21]...


# Parkinson's Disease Resources!

### American Parkinson Disease Association

Provides support for all who are impacted by Parkinson's Disease

https://www.apdaparkinson.org/

### The Michael J. Fox Foundation

Heavily funds research for Parkinson's disease and promotes awareness of this condition

https://www.michaeljfox.org/

### Parkinson's Foundation

Supports research and provides educational resources globally

https://www.parkinson.org/

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**The last "dose"** …

## "I often say now I don't have any choice whether or not I have Parkinson's, but surrounding that non-choice is a million other choices that I can make"
## - Michael J. Fox

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