

Parkinson Disease

A plain-English guide for you and your family

WHAT IS HAPPENING

Think of the substantia nigra as a tiny factory deep in your brain that makes dopamine, a chemical messenger. That dopamine travels to another part called the striatum, which acts like a control panel for starting movement and keeping it smooth. When those factory cells start dying, clumps of a sticky protein called alpha-synuclein (Lewy bodies) build up inside them like rust in a machine. Without enough dopamine, your striatum cannot send clear start-and-go signals to your muscles. That is why you might see a hand tremor when it is resting, muscles that feel stiff and catch like a ratchet when someone bends your arm, slow movement overall, and trouble with balance. Your face may look less expressive, your handwriting shrinks, and your walk turns into a shuffle. Many people also lose their sense of smell years before the shaking starts, deal with constipation, feel sad or anxious, and notice their thinking is not as sharp. None of these problems are random—they all trace back to one upstream break: dying dopamine cells. Your body tries to cope by firing harder through the remaining pathways, but eventually the system cannot keep up. Treatment works by replacing the missing dopamine (levodopa), protecting the cells that are left (MAO-B inhibitors like selegiline or rasagiline), or even using a tiny brain pacemaker (deep brain stimulation) to override the broken signal. The key is catching it early and keeping you moving, because movement itself helps protect the brain.

What you might feel

- Shaking in one hand or arm while at rest (resting tremor)
- Moving slowly overall (bradykinesia)
- Stiff, rigid muscles
- Trouble with balance and walking (postural instability)
- Smaller handwriting that gets tinier as you write (micrographia)
- Soft, mumbled speech and a flat facial expression (hypomimia)
- Trouble rolling over in bed or getting up from a chair
- Falls in the first year, trouble moving the eyes up and down, or early problems controlling blood pressure and bladder

Call the doctor right away if...

- Falls or severe balance problems within the first year of symptoms
- Eye-movement trouble, especially looking up or down (vertical gaze palsy)
- Blood pressure that drops sharply when standing, severe constipation, or loss of bladder control early in the illness
- Symptoms that are exactly the same on both sides of the body from the start (true Parkinson usually begins on one side)
- No improvement at all after a fair trial of levodopa (the main Parkinson medicine)

What the care team will do

- Assess fall risk at every visit using the Timed Up and Go test, and check orthostatic vital signs (blood pressure and heart rate lying, sitting, and standing after 1 and 3 minutes).
- Give carbidopa-levodopa exactly on schedule, ideally 30 to 60 minutes before meals, and never hold or delay a dose without calling the provider.
- Monitor for sudden confusion, high fever, muscle rigidity, or inability to swallow, and escalate immediately if any appear.
- Encourage and document 30 to 60 minutes of aerobic exercise (fast walking, cycling, dancing, or boxing) at least three to five times per week.
- Assess swallowing safety before every meal by watching the patient drink 3 ounces of water, and refer to speech therapy if coughing, choking, or a wet voice occurs.
- Screen for depression, anxiety, and hallucinations using direct questions at every visit, such as 'Do you feel sad or hopeless most days?' and 'Do you see things or people that others do not see?'
- Teach patients to break complex movements into separate steps, use visual cues like floor tape lines to prevent freezing of gait, and count or hum rhythmically while walking.
- Monitor for impulse-control disorders by asking about new gambling, shopping, eating, or sexual behaviors, especially in patients taking dopamine agonists like pramipexole or ropinirole.

Medicines you may be given — and why

Carbidopa-levodopa

Levodopa is the building block the brain uses to make dopamine, the messenger that controls smooth movement. Parkinson's kills the cells that normally make dopamine, so we give the brain fresh raw material to rebuild its supply. Carbidopa blocks an enzyme in the gut that would destroy levodopa before it reaches the brain, so more levodopa gets through. Once

inside the brain, nerve cells turn levodopa into dopamine, filling the empty tank and letting muscles move smoothly again.

Pramipexole and ropinirole (dopamine agonists)

These drugs mimic dopamine by plugging directly into dopamine receptors (the docking stations on nerve cells that dopamine normally lands on). Even though the brain is not making enough real dopamine, these imposters fit the lock and turn on the same movement signals. They are often started first in younger patients (under 60–65) to delay the need for levodopa and reduce the early risk of motor fluctuations.

Rasagiline and selegiline (MAO-B inhibitors)

MAO-B is an enzyme inside brain cells that breaks down dopamine after it is used, like a cleanup crew. By blocking MAO-B, these drugs let each molecule of dopamine hang around longer and keep working, stretching the brain's dwindling dopamine supply. They provide mild symptom relief on their own in early disease and are often added to levodopa later to smooth out wearing-off spells.

Entacapone and tolcapone (COMT inhibitors)

COMT is another enzyme that breaks down levodopa, both in the bloodstream and in the brain. By blocking COMT, these drugs keep each dose of levodopa in the body longer, so the brain gets a steadier, longer-lasting supply of dopamine. They are always given together with carbidopa-levodopa (never alone) to extend the 'on' time and reduce the wearing-off that happens as the disease advances.

Amantadine

Amantadine blocks NMDA receptors (gates for glutamate, an excitatory brain messenger) in the striatum. In Parkinson's, when dopamine drops, glutamate signals become overactive and unbalanced, which worsens stiffness and contributes to levodopa-induced dyskinesias. By dialing down the glutamate noise, amantadine smooths movement and is especially helpful for treating the jerky, involuntary movements (dyskinesias) caused by long-term levodopa use.

Deep brain stimulation (DBS)

DBS is a brain pacemaker. A surgeon implants thin wires (electrodes) into specific deep brain targets—most commonly the subthalamic nucleus (STN) or the globus pallidus interna (GPi)—and connects them to a battery pack under the skin in the chest. The device sends steady electrical pulses that jam the abnormal, overactive signals caused by dopamine loss, restoring more normal movement patterns. It is reserved for patients whose motor symptoms (tremor, stiffness, slowness) and medication fluctuations severely limit daily life despite optimized drug therapy, and who do NOT have major thinking problems or unstable medical/psychiatric illness.

Where this information comes from

- Movement Disorder Society (MDS) Clinical Diagnostic Criteria for Parkinson Disease 2015
- American Academy of Neurology (AAN) Parkinson Disease Practice Guideline 2023
- National Institute of Neurological Disorders and Stroke (NINDS)
- Cochrane systematic reviews on Parkinson Disease

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