

Parkinsons Disease

Parkinson disease shows a one-sided resting tremor (shaking when the hand is still), stiffness that feels like turning a rusty gear, slow movement, and trouble balancing. Levodopa (a pill the brain turns into dopamine) is the main medicine that helps.

THE ONE-LINE TRUTH

The substantia nigra (a small dark spot deep in the brain that makes dopamine) loses its dopamine-making nerve cells, so the brain cannot send smooth movement signals anymore.

The chain — how it unfolds

1. Alpha-synuclein clumps into Lewy bodies inside dopamine cells in the substantia nigra and kills them. — why: The misfolded protein jams the cell's cleanup systems and poisons its energy factories, so the cell cannot survive.
2. Dopamine supply to the striatum drops, but you feel fine at first because the surviving cells work overtime. — why: The remaining cells make extra dopamine and sprout new connections to cover the gap, so movement stays smooth until 60–80% of cells are dead.
3. Once the loss crosses the threshold, motor symptoms appear—usually starting on one side: resting tremor (shaking when the hand is still), cogwheel rigidity (stiff muscles that give way in jerky steps, like turning a ratchet), bradykinesia (slow, small movements—shuffling walk, tiny handwriting, stiff face), and postural instability (trouble balancing). — why: Without enough dopamine, the striatum cannot properly time and smooth muscle commands; too much inhibitory signaling freezes movement, and loss of fine control allows tremor and balance loss.
4. Alpha-synuclein spreads along nerve pathways beyond the substantia nigra—into the brainstem (the lower brain that controls automatic functions), the olfactory bulb (the smell center), the gut nervous system, and eventually the cortex (the thinking layer of the brain). — why: The clumps move from cell to cell like a slow infection, killing nerve cells in each new region.

5. Non-motor symptoms appear years before or alongside the movement problems: REM sleep behavior disorder (acting out dreams—punching, kicking in sleep), loss of smell (anosmia), constipation, depression, anxiety, and later dementia (memory and thinking problems). — why: Alpha-synuclein kills cells in the brainstem that normally paralyze muscles during dreams, in the olfactory bulb that detects smells, in the gut nerves that move stool along, in mood-regulating centers (locus coeruleus, raphe nuclei), and in the cortex that handles memory and planning.
6. The body tries to adapt: muscles stiffen to fight the tremor, steps shorten and slow to avoid falls, breathing pauses to stabilize posture. — why: These are reflexes to maintain safety and balance, but they make movement even more awkward—so what you see is a mix of the disease (dopamine loss) and the body's clumsy attempts to cope.
7. As the disease advances, patients face falls (from balance loss and freezing of gait), choking and pneumonia (from weak swallowing muscles and silent aspiration), and autonomic crises (blood pressure swings, overheating, severe constipation). Stopping levodopa suddenly can trigger a life-threatening syndrome that looks like neuroleptic malignant syndrome (high fever, rigid muscles, confusion, kidney failure). — why: Dopamine loss spreads to circuits that control swallowing, blood pressure, temperature, and bowel function; the body depends on levodopa to keep these systems running, so abrupt withdrawal causes a dangerous crash.
8. Every symptom—tremor, stiffness, slow gait, loss of smell, acting out dreams, constipation, low mood, memory trouble—traces back to one root cause: alpha-synuclein killing dopamine and other nerve cells along connected pathways. — why: This is one disease spreading through the nervous system, not a random collection of problems; understanding the map (substantia nigra → striatum → movement; brainstem → sleep/smell/gut; cortex → memory) lets you predict what will happen next and explain it to families.

Spot it — what you see, and why

- Resting tremor (shaking when the limb is still): Dopamine (a brain messenger that smooths out movement) drops in the substantia nigra (a small area deep in the brain that controls motion), so circuits that normally quiet down tremors stay too active when you're at rest.
- Bradykinesia (movements become slow and small): The basal ganglia (clusters deep in the brain that plan and start movement) lose dopamine, so the brain takes longer to send 'go' signals to muscles, making every action—walking, writing, facial expressions—slower and smaller.
- Rigidity (muscles feel stiff and resist when someone bends your arm or leg): Without enough dopamine, the circuits that tell muscles to relax don't work well, so opposing muscle groups squeeze at the same time, creating a stiff, cogwheel feeling when the limb is moved.

- Postural instability (trouble balancing, shuffling gait, tendency to fall backward): The brain's automatic balance reflexes—controlled by the basal ganglia—slow down, so you can't adjust your posture quickly when you lean or get bumped, leading to shuffling steps and falls.
- Masked face (reduced facial expression, less blinking): The small muscles in the face move more slowly because dopamine loss affects fine motor control everywhere, so your face looks blank even when you feel emotion inside.
- Micrographia (handwriting gets tiny and cramped): The brain's ability to sustain smooth, repetitive movements fades as dopamine drops, so each pen stroke becomes smaller and weaker as you write across the page.

Red flags — act now

- Falls within the first year of symptoms (suggests a faster, broader brain disease rather than classic Parkinson's)
- Severe blood-pressure drops when standing, early bladder failure, or trouble swallowing within the first year (points to multiple system atrophy)
- Rapid progression with prominent dementia, hallucinations not caused by medication, or no response to levodopa (the main Parkinson's drug)
- Prominent downward-gaze palsy (can't look down) or backward falls early on (suggests progressive supranuclear palsy)
- Symptoms that begin symmetrically on both sides of the body at once (classic Parkinson's almost always starts on one side)

Treat it — in this order

1. Levodopa-carbidopa, started at 100/25 mg three times daily and slowly increased every few days to find the lowest effective dose → Levodopa crosses the blood-brain barrier and becomes dopamine, replacing what the dead substantia nigra cells cannot make anymore. This directly fixes the dopamine shortage that causes tremor, rigidity, and slow movement. Carbidopa blocks levodopa breakdown in the gut and bloodstream so more reaches the striatum (the movement hub).
2. Dopamine agonists (pramipexole 0.125–1.5 mg three times daily, or ropinirole 0.25–8 mg three times daily) in patients under 65 → These drugs plug directly into dopamine receptors in the striatum to mimic dopamine and keep movement smooth. Using them early, especially in patients under sixty-five, delays the need for levodopa and may lower the risk of wearing-off and dyskinesia problems.

3. MAO-B inhibitor (rasagiline 1 mg daily or selegiline 5 mg twice daily) → Blocks monoamine oxidase B (the enzyme that breaks down dopamine in the striatum), so whatever dopamine the brain still makes lasts longer and works harder. Can be used alone in early mild disease or added to levodopa later to smooth out wearing-off.
4. COMT inhibitor (entacapone 200 mg with each levodopa dose, or tolcapone 100–200 mg three times daily) added when wearing-off starts → Blocks catechol-O-methyltransferase (the enzyme that breaks down levodopa in the bloodstream), so each levodopa dose lasts longer and more reaches the striatum. Extends the on-time window and reduces the off-time between doses.
5. Physical therapy with LSVT BIG protocol, occupational therapy, and speech therapy with LSVT LOUD protocol → These teach the body new motor pathways to work around the broken nigrostriatal dopamine circuit — retraining balance, gait, voice volume, and hand coordination without relying on dopamine alone. LSVT programs use big exaggerated movements and loud voice to reprogram the brain's sense of normal effort.

Your top priorities at the bedside

1. Check orthostatic vital signs (blood pressure and heart rate lying, sitting, and standing after 1 and 3 minutes) every shift and before giving dopamine drugs. — why: Both Parkinsons and levodopa cause orthostatic low blood pressure (hypotension) (blood pressure drop when standing). A systolic drop greater than 20 mmHg or diastolic drop greater than 10 mmHg increases fall and fainting (syncope) (fainting) risk. Dopamine medications worsen this drop.
2. Assess swallowing safety before every meal by watching the patient drink 3 ounces of water and checking for coughing, throat clearing, or wet voice. — why: Parkinsons weakens the pharyngeal muscles (throat muscles) that protect the airway during swallowing. Silent aspiration (food or liquid entering the lungs without coughing) causes aspiration pneumonia, the leading cause of death in Parkinsons.
3. Give levodopa-carbidopa exactly 30 minutes before meals or 60 minutes after meals, never with high-protein food. — why: Levodopa competes with amino acids (protein building blocks) for absorption across the intestine and blood-brain barrier. Protein in the stomach reduces levodopa absorption by up to 50 percent, causing medication wearing-off and return of tremor and rigidity.