

Parkinsons Disease

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THE STORY IN ONE SENTENCE

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“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.”

Normal anatomy — the body's job

The substantia nigra (a small dark cluster of cells deep in the midbrain) makes dopamine and ships it along a highway of nerves to the striatum (the brain's movement-control center). The striatum uses that dopamine to fine-tune every movement—starting it, stopping it, and keeping it smooth and quick.

BODY'S JOB

Imagine the striatum is a busy switchboard that decides which muscles to turn on and which to turn off so you can walk, write, and gesture without thinking. Dopamine is the signal that keeps the switches flipping fast and smooth. Without it, the switchboard jams, and every movement becomes slow, stiff, and hard to start.

1. The substantia nigra makes dopamine inside its nerve cells.
2. Dopamine travels along long nerve fibers (the nigrostriatal pathway) to the striatum.
3. The striatum receives the dopamine signal and uses it to coordinate commands to your muscles.
4. Muscles get clear, timed instructions: start moving now, stop moving now, stay smooth.
5. You walk, reach, write, and turn without jerky pauses or extra shaking.

What breaks

A sticky, misfolded protein called alpha-synuclein clumps together inside dopamine-making cells in the substantia nigra and kills them. Once 60 to 80 percent of the cells are dead, the striatum runs out of dopamine, and the brain can no longer send smooth movement signals.

CLINICAL TERM

Alpha-synuclein aggregates (clumps of misfolded protein) form Lewy bodies inside neurons of the substantia nigra pars compacta, triggering cell death. Clinical motor signs appear when dopaminergic neuron loss exceeds 60–80% and striatal dopamine drops below the compensatory threshold.

How the body compensates

For years the surviving dopamine cells work overtime—making more dopamine per cell and sprouting extra connections—so you feel normal even though many cells are already dead. But once the loss crosses 60–80%, no amount of overtime can cover the gap, and motor symptoms suddenly appear.

1. The remaining dopamine cells increase their dopamine output per cell (upregulation of tyrosine hydroxylase, the enzyme that makes dopamine).
2. They sprout new nerve terminals to cover more of the striatum, trying to keep the dopamine level steady.
3. The striatum becomes extra-sensitive to dopamine (upregulation of dopamine receptors) to squeeze more signal out of each molecule.
4. These fixes keep movement smooth for years, so the disease is silent during this hidden phase.
5. Once cell loss crosses the 60–80% threshold, the remaining cells cannot compensate anymore, and motor symptoms break through.
6. Later, as dopamine cells keep dying, even high doses of levodopa cannot fully restore smooth movement, and motor fluctuations (on-off swings) and dyskinesias (involuntary wiggling) appear.

Domino effect

1. Alpha-synuclein clumps into Lewy bodies inside dopamine cells in the substantia nigra and kills them. — 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. — 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). — 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). — 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). — 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. — 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). — 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. — 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.

Symptoms · what the patient feels, 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.
- Soft, monotone voice (hypophonia): The muscles of the voice box and chest lose their coordinated push because the basal ganglia can't drive forceful, rhythmic breathing and vocal-cord tension, so speech comes out quiet and flat.
- Loss of smell (hyposmia or anosmia, often years before motor symptoms): Parkinson's disease deposits abnormal clumps of a protein called alpha-synuclein in the olfactory bulb (the smell center at the front of the brain) long before dopamine cells in the substantia nigra die, so smell fades early.
- REM sleep behavior disorder (acting out dreams—talking, thrashing, punching—during sleep): Alpha-synuclein builds up in the brainstem circuits that normally paralyze your muscles during REM sleep, so those muscles stay active and you physically move with your dreams.

- Early frequent falls, prominent autonomic problems (blood pressure drops when standing, severe constipation, urinary trouble), or symmetric motor symptoms from the start: These features suggest atypical parkinsonism (a different group of brain diseases—such as multiple system atrophy, progressive supranuclear palsy, or corticobasal degeneration—that damage broader brain regions and progress faster than classic Parkinson's), not typical Parkinson's disease.

Focused exam

- Resting tremor at 4–6 beats per second, worse on one side, stops when the patient reaches for something: This is the signature shake of Parkinson disease — it shows up when the hand is at rest, not during action. It means dopamine cells in the substantia nigra (the brain's dopamine factory for smooth movement) have died on one side, so that side cannot quiet the tremor circuit.
- Cogwheel rigidity — jerky stiffness when you gently bend and straighten the patient's arm, like rolling over bumps: Rigidity plus tremor together point strongly to Parkinson disease, not other causes of shaking. The muscles stay tight because dopamine is missing — dopamine normally tells muscles to relax between movements.
- Bradykinesia — slow shuffling walk, tiny cramped handwriting, blank face with no smile, quiet flat speech: Bradykinesia means the movement system is starving for dopamine signals. This is the most disabling motor symptom of Parkinson disease and the one that always gets worse over time.
- Postural instability — patient leans forward, has trouble stopping after a few steps, falls backward easily when gently pulled: Balance problems mean Parkinson disease is stealing dopamine from the brainstem circuits that keep you upright. Falls are the biggest danger in Parkinson disease because they lead to broken bones and loss of independence.
- Loss of smell (anosmia) — patient cannot name familiar scents like coffee or peppermint: Smell loss happens early in Parkinson disease because alpha-synuclein (the sticky clumping protein that kills cells) builds up in the nose nerves and spreads upward into the brain. It is one of the earliest clues, often years before the tremor starts.
- Symptoms that start on one side and stay mostly one-sided or spread slowly to the other side over months to years: Parkinson disease is usually one-sided at first because dopamine loss starts on one side of the substantia nigra. If both sides are affected equally from day one, think of other diagnoses like multiple system atrophy or progressive supranuclear palsy.

Diagnostics · every test answers a question

- Clinical diagnosis using the Movement Disorder Society criteria → Does the patient have bradykinesia plus at least one of resting tremor or rigidity, with one-sided start, gradual progression, and no red flags for other diseases?
- Brain MRI without contrast → Is there structural damage — stroke, tumor, hydrocephalus, or iron deposits — that could mimic Parkinson disease?
- DaTscan (ioflupane I-123 SPECT imaging of dopamine transporters) → Is dopamine transporter density reduced in the striatum (the brain's movement control hub)?
- Levodopa challenge test (100/25 mg levodopa-carbidopa given once, exam repeated after 60 minutes) → Do motor symptoms improve by at least 30 percent with one dose of levodopa?
- Autonomic function testing (tilt table, heart rate variability, thermoregulatory sweat test, bladder residual) → Is there severe autonomic failure — blood pressure dropping more than 30/15 mmHg when standing, absent sweating, or large bladder residual volume?
- Polysomnography (overnight sleep study) with video and EMG during REM sleep → Is the patient acting out dreams — punching, kicking, shouting, or running movements — during REM sleep?
- Serum ceruloplasmin and 24-hour urine copper (in patients under 50) → Is there copper buildup from Wilson disease mimicking Parkinson disease?
- Medication and toxin history review → Is the parkinsonism caused by drugs (antipsychotics, metoclopramide, valproate) or toxins (manganese, carbon monoxide, MPTP)?

Treatment logic

- 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).
- 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.

- 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.
- 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.
- 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.
- SSRI or SNRI (sertraline, citalopram, venlafaxine) for depression; rivastigmine for dementia; pimavanserin 34 mg daily for hallucinations → These address non-motor symptoms driven by loss of dopamine, serotonin, and acetylcholine in other brain circuits — treating depression, memory loss, and visual hallucinations separately. Pimavanserin is the only antipsychotic proven safe in Parkinson disease because it does not block dopamine receptors and worsen movement.
- Deep brain stimulation (DBS) targeting the subthalamic nucleus or globus pallidus interna with implanted electrodes → High-frequency electrical pulses to these deep motor-control nuclei bypass the broken dopamine circuit and restore smoother movement when drugs lose effectiveness or cause unbearable side effects. Reduces motor fluctuations, tremor, rigidity, and drug-induced dyskinesias.

Medications

Levodopa-carbidopa (Sinemet) · Dopamine precursor + dopa-decarboxylase inhibitor

Levodopa is the building block your brain uses to make dopamine (the chemical messenger that lets you move smoothly). In Parkinson's, the cells that make dopamine are dying, so you run low. Carbidopa stops an enzyme in your gut and blood from breaking down levodopa before it reaches your brain. Once levodopa crosses into the brain, the remaining dopamine cells turn it into dopamine, filling in what's missing so you can move smoothly again.

1. You swallow the pill. Carbidopa blocks the enzyme that would destroy levodopa in your gut and blood.

2. Levodopa survives the trip and crosses into your brain, entering the remaining dopamine nerve cells in the substantia nigra (the dopamine factory in the midbrain) and striatum (the movement switchboard).
3. Those cells use an enzyme called aromatic amino acid decarboxylase to turn levodopa into dopamine.
4. The new dopamine fills in for what's missing, so signals for smooth movement can flow again.
5. Your tremor, stiffness, and slowness improve.

SAFETY WATCH

Nausea and vomiting, especially at first. Taking it with a small snack helps, but avoid high-protein meals (like meat or cheese) that block levodopa from being absorbed. · Dizziness or feeling faint when you stand up (orthostatic low blood pressure (hypotension)), because dopamine affects how your blood vessels squeeze. · Dyskinesias (twisting, wiggling, jerky movements) after years of use, because the brain becomes overly sensitive to dopamine. · Motor fluctuations: the medicine wears off before the next dose, or you get sudden on-off switches between moving well and freezing. · Impulse-control problems (sudden urges to gamble, shop, eat, or have sex) in some people. · Hallucinations (seeing things that aren't there) or confusion, especially in older adults or people with memory problems. · Never stop suddenly—taper slowly with your doctor to avoid a dangerous withdrawal that looks like neuroleptic malignant syndrome (high fever, stiff muscles, confusion).

Pramipexole (Mirapex) or Ropinirole (Requip) · Dopamine agonist (non-ergot)

These drugs act like fake dopamine. They plug into dopamine receptors (the docking sites on nerve cells) and turn them on directly, so you don't need your own dying cells to make real dopamine. They're often started first in younger people to delay needing levodopa.

1. You take the pill. The drug enters your bloodstream and crosses into your brain.
2. It locks onto dopamine receptors on nerve cells in the striatum.
3. The receptors turn on as if real dopamine had arrived.
4. Movement signals flow more smoothly, reducing tremor, stiffness, and slowness.
5. Because you're not relying on dying cells to make dopamine, this buys time before you need levodopa.

SAFETY WATCH

Impulse-control problems (sudden strong urges to gamble, shop, eat, or have sex) are MORE common than with levodopa—warn every patient and family. · Daytime sleepiness and sudden sleep attacks (falling asleep without warning, even while driving). Do not drive if this happens. · Nausea, dizziness, and low blood pressure when you stand up. · Hallucinations and confusion, especially in older adults. · Leg swelling (peripheral swelling (edema)). · Worsening of existing heart-valve disease (rare; more common with older ergot agonists; these non-ergot drugs are safer).

Selegiline (Eldepryl, Zelapar) or Rasagiline (Azilect) · MAO-B inhibitor (monoamine oxidase type B inhibitor)

These drugs block an enzyme in your brain called MAO-B that normally breaks down dopamine. By stopping that breakdown, whatever dopamine your remaining cells make (or whatever levodopa becomes) lasts longer. Think of it as plugging a leak so your dopamine supply stretches further.

1. You take the pill. The drug enters your brain.
2. It locks onto and blocks the MAO-B enzyme.
3. Dopamine that would have been chopped up now stays active longer.
4. Your movement symptoms improve because dopamine signals last longer.
5. This can be used alone in very early disease or added to levodopa later to smooth out wearing-off periods.

SAFETY WATCH

Insomnia, especially if taken late in the day. · Dizziness and low blood pressure. · Nausea. · Headache. · Serotonin syndrome (confusion, agitation, muscle stiffness, fever) if combined with certain antidepressants (SSRIs like fluoxetine, SNRIs like duloxetine, tricyclics), tramadol, meperidine, dextromethorphan, or St. John's wort. At usual Parkinson doses, these drugs are generally safe with SSRIs, but tell all your doctors what you're taking. · Do NOT combine with MAO-A inhibitors or use non-selective MAOIs. · You do NOT need to avoid tyramine-rich foods (cheese, wine, aged meats) at usual doses because these drugs are selective for MAO-B, not MAO-A.

Entacapone (Comtan) or Tolcapone (Tasmar) · COMT inhibitor (catechol-O-methyltransferase inhibitor)

COMT is an enzyme that breaks down levodopa in your blood and brain. By blocking COMT, more levodopa reaches your brain and stays active longer. This is always given WITH levodopa-carbidopa, not alone. It helps when levodopa starts wearing off before the next dose.

1. You take this pill at the same time as your levodopa-carbidopa.
2. The COMT inhibitor blocks the enzyme that would destroy levodopa.
3. More levodopa enters your brain, and it lasts longer once it's there.
4. Your 'on' time (when the medicine works) gets longer, and 'off' time (when stiffness and slowness return) gets shorter.
5. Movement stays smoother between doses.

SAFETY WATCH

Diarrhea (very common with entacapone, often loose and orange). · Urine turns orange or brownish-red (harmless but can be alarming if you don't expect it). · Worsening dyskinesias (you may need to reduce your levodopa dose). · Nausea, dizziness, low blood pressure. · Tolcapone can cause severe liver damage (fatal cases reported). It's only used if entacapone doesn't work, and requires liver enzyme blood tests every 2–4 weeks for the first 6 months, then regularly after. · Hallucinations and confusion, especially in older adults. · Impulse-control problems (less common than with dopamine agonists but possible).

Amantadine (Gocovri, Osmolex ER) · NMDA receptor antagonist (also has dopamine-boosting effects)

Amantadine blocks a receptor in the brain called NMDA (a docking site for glutamate, an excitatory brain messenger) that becomes overactive when dopamine is low. By calming that overactivity, it helps smooth out movement. It also nudges your remaining dopamine cells to release a bit more dopamine and slows dopamine reuptake (the cleanup process). It's especially helpful for levodopa-induced dyskinesias (the involuntary jerky movements that appear after years of levodopa use).

1. You take the pill. Amantadine crosses into your brain.
2. It blocks NMDA receptors, which quiets the overexcited glutamate signals that worsen movement when dopamine is low.
3. It also helps dopamine nerve cells release more dopamine and keeps dopamine in the synapse a bit longer.
4. Your movements become less jerky (especially if you have dyskinesias), and tremor and stiffness may improve a little.

5. This is often added to levodopa therapy, not used alone.

SAFETY WATCH

Confusion, hallucinations, and vivid dreams, especially in older adults or people with memory problems. · Leg swelling with livedo reticularis (a lacy, purple skin mottling that is common and harmless). · Dizziness and low blood pressure. · Insomnia (take earlier in the day). · Dry mouth and constipation. · Impulse-control problems (less common than dopamine agonists). · Worsening of kidney function (dose must be adjusted if your kidneys are impaired). · Never stop suddenly after long-term use—taper slowly to avoid worsening parkinsonism or a withdrawal syndrome.

Rivastigmine (Exelon) · Cholinesterase inhibitor

Parkinson's disease can damage brain areas that use acetylcholine (a messenger for memory, attention, and thinking), leading to dementia. Rivastigmine blocks the enzyme acetylcholinesterase that breaks down acetylcholine, so more stays available. This helps with memory, attention, and daily thinking tasks. It does NOT help movement symptoms.

1. You take the pill or apply the skin patch. Rivastigmine enters your brain.
2. It blocks acetylcholinesterase, the enzyme that chops up acetylcholine.
3. Acetylcholine levels rise in the spaces between nerve cells.
4. Memory, attention, and daily function improve modestly.
5. This does NOT reverse dementia, but it can slow the decline for a while.

SAFETY WATCH

Nausea, vomiting, and loss of appetite (common, especially at the start; the patch causes less nausea than pills). · Diarrhea. · Dizziness and headache. · Weight loss. · Worsening of tremor in some people (rare). · Slow heart rate (bradycardia), especially if you have heart-conduction problems. · Worsening of asthma or COPD (because it boosts acetylcholine, which can tighten airways). · Increased stomach acid and risk of ulcers (use cautiously if you have a history of ulcers).

Pimavanserin (Nuplazid) · Selective serotonin inverse agonist (5-HT_{2A})

Hallucinations and delusions in Parkinson's are partly caused by too much signaling at serotonin 5-HT_{2A} receptors (docking sites for serotonin, a brain messenger) in areas that process reality. Pimavanserin blocks those receptors, which calms psychotic symptoms WITHOUT blocking dopamine receptors. This is crucial because typical antipsychotics block

dopamine and make Parkinson's movement symptoms much worse. Pimavanserin is the only antipsychotic specifically approved by the FDA for Parkinson's psychosis.

1. You take the pill. Pimavanserin enters your brain.
2. It locks onto and blocks 5-HT_{2A} serotonin receptors.
3. Abnormal perception signals quiet down.
4. Hallucinations (seeing or hearing things that aren't there) and delusions (false beliefs) lessen.
5. Because it does NOT block dopamine, your movement does NOT get worse.

SAFETY WATCH

Swelling in the legs (peripheral swelling (edema)). · Confusion. · Nausea. · Prolonged QT interval on the EKG (the heart's electrical tracing), which can lead to dangerous heart-rhythm problems. Do NOT use if you already have QT prolongation or take other drugs that lengthen QT. Get an EKG before starting. · Increased risk of death in elderly patients with dementia-related psychosis (this is a black-box FDA warning for all antipsychotics, including pimavanserin, though it's still considered safer than dopamine-blocking drugs in Parkinson's). · Hallucinations may worsen if the drug is stopped suddenly.

Quetiapine (Seroquel) — low dose · Atypical antipsychotic

Quetiapine blocks serotonin and dopamine receptors, which can calm hallucinations and delusions. At very low doses (25–50 mg), it has weak dopamine-blocking effects, so it's less likely to worsen Parkinson's movement symptoms than older antipsychotics. It's used off-label (not FDA-approved for this) when pimavanserin isn't available or tolerated, but the evidence is mixed.

1. You take a low-dose pill at bedtime. Quetiapine enters your brain.
2. It blocks serotonin receptors (which helps psychosis) and weakly blocks dopamine receptors.
3. Hallucinations and agitation lessen.
4. Sedation helps with sleep.
5. Movement symptoms are less likely to worsen than with stronger antipsychotics, but worsening IS still possible.

SAFETY WATCH

Sedation and drowsiness (common; often given at bedtime). · Dizziness and low blood pressure, especially when standing. · Weight gain and increased blood sugar (monitor for diabetes). · Worsening movement symptoms (stiffness, slowness, tremor) if the dose is too high or the person is very sensitive. · Prolonged QT interval (get an EKG). · Increased risk of death in elderly patients with dementia-related psychosis (black-box FDA warning). · Avoid higher doses—keep it under 100 mg/day in Parkinson's.

Red flags

- 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)

Nursing priorities

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. — 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. — 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. — 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.
4. Keep the bed in the lowest position with both side rails down and a bedside commode within 3 feet of the bed. — Parkinsons causes postural instability (poor balance), bradykinesia (slow movement), and freezing of gait (feet stuck to floor). Side rails create entrapment risk and force patients to climb over them. Short distance to the commode prevents freezing episodes during urgent toileting.
5. Monitor bowel movements daily and report if no stool for 3 days or hard, pellet-like stools. — Parkinsons damages the enteric nervous system (nerve network in the gut wall), slowing colonic transit time by 50 to 70 percent. Severe constipation increases aspiration risk during straining and can cause fecal impaction requiring manual disimpaction.
6. Screen for hallucinations or paranoia at every interaction by asking if the patient sees things others do not see or feels unsafe. — Levodopa and dopamine agonists cause psychosis in 40 percent of patients, usually visual hallucinations of people or animals. Untreated psychosis leads to nursing-home placement. Antipsychotics like haloperidol worsen Parkinsons, so only quetiapine or pimavanserin are safe.
7. Encourage the patient to stand and walk at least 50 feet four times per day, even if slow, and never use a wheelchair for short distances. — Bradykinesia and rigidity (muscle stiffness) worsen with immobility in a use-it-or-lose-it pattern. Deconditioning accelerates loss of dopamine neurons. Physical activity increases brain-derived neurotrophic factor (a brain growth protein) that protects remaining dopamine cells.
8. Assess mood and energy every shift using a two-question screen: Do you feel sad or hopeless? Have you lost interest in things you used to enjoy? — Depression occurs in 50 percent of Parkinsons patients due to dopamine and serotonin depletion in the limbic system (emotion-regulation brain area). Untreated depression worsens motor symptoms and cognitive decline. SSRIs like sertraline or citalopram are first-line treatment.

Sources

- AAN Practice Guideline: Parkinson Disease 2021
- Movement Disorder Society Evidence-Based Medicine Reviews
- NICE Guideline NG71: Parkinson's Disease in Adults 2017 (updated 2024)
- Cochrane Database of Systematic Reviews: Parkinson's Disease

Last reviewed: 2026-07-07