

Parkinsons Disease

A plain-English guide for you and your family

WHAT IS HAPPENING

Deep in your brain sits a tiny dark factory called the substantia nigra. Its job is to make dopamine, a messenger chemical that tells your muscles when and how to move smoothly—like the conductor of an orchestra keeping every instrument on time. In Parkinson disease, a sticky protein called alpha-synuclein clumps together inside these dopamine-making cells and kills them one by one. For years your brain hides the loss by pushing the surviving cells harder, so you feel fine. But once 60 to 80 percent of the cells are dead, the remaining few cannot keep up, and movement problems finally appear—usually on one side first. You might notice a hand that shakes when it rests in your lap, muscles that feel stiff and jerky (like a ratchet), a shuffling walk with tiny steps, handwriting that shrinks to a crawl, and trouble staying balanced. These are not separate problems; they are all one story: without enough dopamine, the brain's movement-control center (the basal ganglia) cannot smooth out muscle commands anymore. The clumps do not stay put—they spread along nerve highways to other brain regions and even down into the gut, which is why Parkinson also brings acting out dreams during sleep, loss of smell, constipation, low mood, and later memory trouble. None of this is random. Your body tries to cope—stiffening muscles to fight the tremor, slowing steps to avoid a fall—but these fixes only buy time. The real answer is replacing the missing dopamine with medicine (levodopa) and keeping you safe from falls, choking on food, and sudden crashes if the medicine is stopped too fast.

What you might feel

- Resting tremor (shaking when the limb is still)
- Bradykinesia (movements become slow and small)
- Rigidity (muscles feel stiff and resist when someone bends your arm or leg)
- Postural instability (trouble balancing, shuffling gait, tendency to fall backward)
- Masked face (reduced facial expression, less blinking)
- Micrographia (handwriting gets tiny and cramped)
- Soft, monotone voice (hypophonia)

- Loss of smell (hyposmia or anosmia, often years before motor symptoms)
- REM sleep behavior disorder (acting out dreams—talking, thrashing, punching—during sleep)
- Early frequent falls, prominent autonomic problems (blood pressure drops when standing, severe constipation, urinary trouble), or symmetric motor symptoms from the start

Call the doctor right away if...

- 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)

What the care team will do

- 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.
- Assess swallowing safety before every meal by watching the patient drink 3 ounces of water and checking for coughing, throat clearing, or wet voice.
- Give levodopa-carbidopa exactly 30 minutes before meals or 60 minutes after meals, never with high-protein food.
- Keep the bed in the lowest position with both side rails down and a bedside commode within 3 feet of the bed.
- Monitor bowel movements daily and report if no stool for 3 days or hard, pellet-like stools.
- Screen for hallucinations or paranoia at every interaction by asking if the patient sees things others do not see or feels unsafe.
- 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.
- 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?

Medicines you may be given — and why

Levodopa-carbidopa (Sinemet)

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.

Pramipexole (Mirapex) or Ropinirole (Requip)

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.

Selegiline (Eldepryl, Zelapar) or Rasagiline (Azilect)

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.

Entacapone (Comtan) or Tolcapone (Tasmar)

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.

Amantadine (Gocovri, Osmolex ER)

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).

Rivastigmine (Exelon)

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.

Pimavanserin (Nuplazid)

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.

Quetiapine (Seroquel) — low dose

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.

Where this information comes from

- 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

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