Parkinson Disease

Bradykinesia plus rest tremor or rigidity · levodopa on time, no dopamine blockers, and always check the standing blood pressure

Give levodopa
on time, every time

RECOGNIZE IT *the diagnosis is clinical, and the first test is the med list*

THE CARDINAL FEATURES

Bradykinesia is required: slowness with decrement on repetitive movement, showing up as micrographia, masked face, hypophonia. Plus **rest tremor** (4-6 Hz pill-rolling, absent in up to 25%, so no tremor does not exclude it) and/or **lead-pipe rigidity** with cogwheeling. **Asymmetric onset is typical.** Postural instability is a LATE feature.

RED FLAGS = NOT IDIOPATHIC PD

Early falls → PSP. Early severe autonomic failure → MSA. Dementia within 1 year of motor onset → DLB. Also symmetric onset, vertical gaze palsy, cerebellar signs, and **poor response to adequate-dose levodopa**. Why it matters: these syndromes respond poorly to levodopa and progress faster.

WORKUP LOGIC

Review the med list first: antipsychotics, metoclopramide and prochlorperazine cause a reversible, usually symmetric parkinsonism. MRI only for atypical pictures. **DaTscan only separates degenerative parkinsonism from essential tremor or drug effect**; it is abnormal in PD, MSA, PSP and DLB alike, so it cannot confirm PD.

MOTOR THERAPY *levodopa is the workhorse; match the rest to the patient*

Agent	How and why
Carbidopa-levodopa *most effective agent*	**25/100 mg, half to one tab TID**, titrated to function. Take **30-60 min before meals**: dietary protein competes for the large neutral amino acid transporter in gut and brain, so a protein-heavy meal visibly blunts a dose. **Poor response at adequate doses is itself a red flag for atypical disease**
Dopamine agonists *pramipexole, ropinirole, rotigotine patch*	Option in **younger patients** to delay levodopa motor complications. ⚠ **Ask directly about gambling, shopping and hypersexuality at every visit**: impulse-control disorders are common, hidden, and reversible on withdrawal. Sleep attacks and hallucinations; avoid in the elderly and cognitively impaired
MAO-B inhibitors *rasagiline 0.5-1 mg daily*	Modest benefit as early monotherapy or adjunct. **No meperidine** (serotonin syndrome); caution with serotonergic drugs
Wearing-off	Shorten the levodopa interval, or add **entacapone 200 mg WITH each levodopa dose** (COMT inhibition extends each dose; orange-brown urine is harmless), an MAO-B inhibitor, or an agonist
Dyskinesia	**Amantadine is the only evidence-based antidyskinetic**. Renally dose it; confusion and hallucinations in the elderly, livedo reticularis
Refractory	**DBS (STN or GPi)** for fluctuations or tremor in levodopa-responsive patients without dementia

THE HOSPITALIZED PATIENT *two order-set traps cause most of the harm*

Rule	Detail
⚠ **Levodopa ON TIME**	Order it at the patient's **home clock times, not ward med-pass times**. Late or missed doses cause rigidity, dysphagia, aspiration and falls within hours
⚠ **Never stop abruptly**	Withdrawal can precipitate **parkinsonism-hyperpyrexia syndrome**: an NMS-like emergency with fever, severe rigidity, autonomic instability and elevated CK. Treatment is **restarting the home regimen immediately** (NG if needed), cooling, IV fluids; dantrolene or bromocriptine if severe
NPO patient	**Levodopa tablets disperse in water via NG tube**, or convert to a **rotigotine transdermal patch (2-4 mg/24 h to start)**; SC apomorphine with specialist input. "Hold all PO meds" is never the answer for levodopa
⚠ **No dopamine blockers**	**Haloperidol, metoclopramide and prochlorperazine worsen parkinsonism** and can cause severe rigidity. Nausea → **ondansetron** or trimethobenzamide. Psychosis or agitation → **quetiapine 12.5-25 mg qHS or clozapine** (least D2 blockade), or **pimavanserin 34 mg**, approved for PD psychosis
Swallow surveillance	Screen early, meals upright, speech pathology involved: **aspiration pneumonia is a leading cause of death in PD**. Scheduled bowel regimen too, since constipation is near-universal and delays levodopa absorption

ORTHOSTATIC HYPOTENSION *the fall nobody explains, in 30-50% of patients*

DIAGNOSE IT

Sustained fall ≥20 mmHg systolic or ≥10 diastolic within 3 min of standing. Often silent, or disguised as fatigue, "brain fog," or a coat-hanger neck ache. **The neurogenic clue: the heart rate barely rises.** ΔHR/ΔSBP < 0.5 bpm/mmHg means the baroreflex itself is degenerated, so **fluids alone will not fix it**.

THE SUPINE HYPERTENSION TRADE-OFF

About half also have supine hypertension, and every pressor you add worsens it. Treat the position, not the number: never fully flat, head-up sleeping. If severe (≥160-180 systolic supine), a **short-acting bedtime agent** (losartan, or a nitroglycerin patch removed on waking) treats the night without sabotaging the morning stand.

Step	What and why
1 · Remove aggravators	**Alpha-blockers (tamsulosin), diuretics, tricyclics, excess antihypertensives**: deprescribe or move to bedtime. Agonists cause more orthostasis than levodopa, so sometimes the fix is a swap, not a new drug
2 · Nonpharmacologic	**Salt 6-10 g/day + fluids 2-2.5 L** (temper in HF/CKD). **500 mL rapid water bolus** raises SBP within 5-15 min for ~1 h: use before rising. **Abdominal binder** compresses the splanchnic bed, where the blood actually pools, so it beats thigh stockings. **Head-up sleeping 30-45°** cuts nocturnal pressure natriuresis (better mornings) AND treats supine hypertension. Leg-crossing and squatting buy time; rise in stages
3 · Midodrine	**2.5-10 mg TID**, alpha-1 pressor. **Last dose ≥4 h before bed, no lying flat after dosing** (supine HTN). Scalp tingling and piloerection mean it is working; watch urinary retention
3 · Droxidopa	**100-600 mg TID**, oral norepinephrine prodrug, FDA-approved for neurogenic OH. Titrate q24-48 h; same bedtime discipline
3 · Fludrocortisone / pyridostigmine	**Fludrocortisone 0.1-0.2 mg** expands volume: watch hypokalemia, edema, supine HTN; caution in HF and the frail. **Pyridostigmine 30-60 mg TID** boosts ganglionic transmission mainly while standing, so it has the **least supine hypertension**: best for mild OH with bad supine HTN

PEARLS & PITFALLS

- **The med list is the first diagnostic test**: metoclopramide and antipsychotic parkinsonism is the reversible mimic.
- **The morning fall has a mechanism**: supine hypertension drives overnight pressure natriuresis, so the patient wakes volume-depleted and the first stand of the day is the worst. Head-up sleeping fixes both halves.
- **In hospital the two traps are held levodopa and D2-blocking order sets** (metoclopramide, prochlorperazine, haloperidol). Reconcile home times at admission and flag the chart.
- **On an agonist, ask about gambling and hypersexuality directly**: patients do not volunteer impulse-control disorders, and they reverse on withdrawal.

References: MDS clinical diagnostic criteria for Parkinson disease · consensus panel recommendations on neurogenic orthostatic hypotension and supine hypertension (J Neurol 2017) · FDA labeling for midodrine, droxidopa and pimavanserin.

Educational aid; verify against current guidance, local formulary and patient factors.

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