

Beyond the Subluxation in Parkinson's

Visual-Vestibular Drivers of Postural Instability and Gait Dysfunction

A Chiropractic Neurology Approach

Presented by

naples brain center
unlocking potential

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Sponsored by



Poll:

- Frank is 72 years old and has been on levodopa for 6 years.
- His tremor is well controlled.
- He has had 3 falls in the last 3-4 months.
- He is becoming more stooped in posture.

- MD: “Medications are optimized”
- Family: “What else is there?”




disease
problem
life
medical
brain
ageing
illness
cerebral
tremors
clinic
mind
lose
dopamine
therapy
stage
healthcare
treat
confused
disability
Parkinson
mental
rehabilitation
condition
medicine
decay
functionality
old
stress
treatment
process
neurology
degenerative
change
weakness
prevention
shaking
elderly
function
dementia
damage
patient
rigidity
stiffness
nursing
health
senior
neuron
confusion
disorder
diagnostic

Introduction:

- Epidemiology
- Clinical Relevance



National/US Statistics



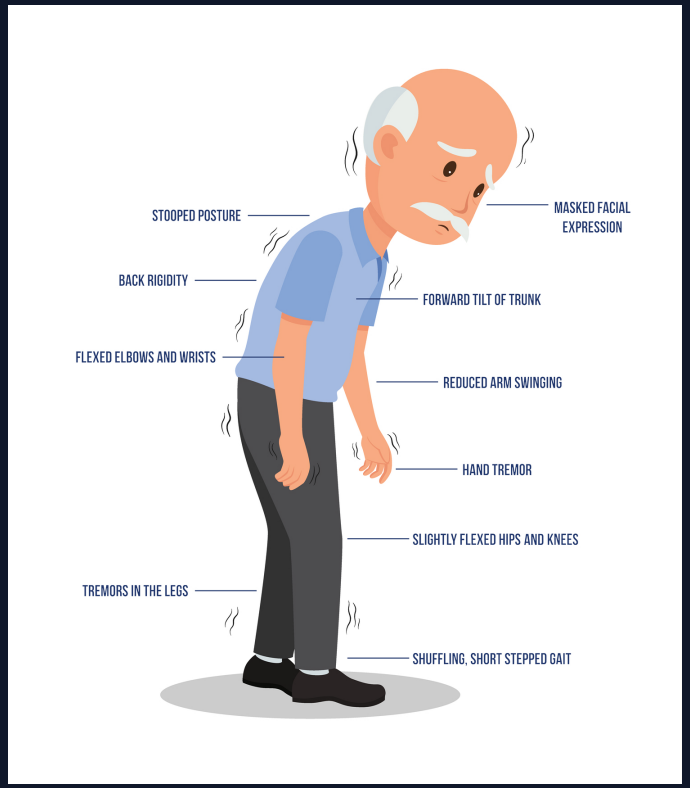
- **Prevalence**: ~1.1 million people living with PD in the US (expected to reach 1.2 million by 2030).
- **Incidence**: Nearly **90,000 new diagnoses per year** (50% higher than older estimates).
- **Demographics**: Risk increases sharply with age (most common after 60); men affected ~1.5x more than women. Second most common neurodegenerative disorder after Alzheimer's.
- **Etiology**: 90–98% sporadic/environmental (not purely genetic).
- **Mortality**: Age-adjusted death rates vary; Florida ranks notably high.

Florida-Specific Factors

- Aging population
- Golf course pesticides (glyphosate, paraquat)
- Mosquito control & environmental toxins
- 90-98% sporadic/ environmental etiology



PIGD Prevalence & Fall Risk in PD



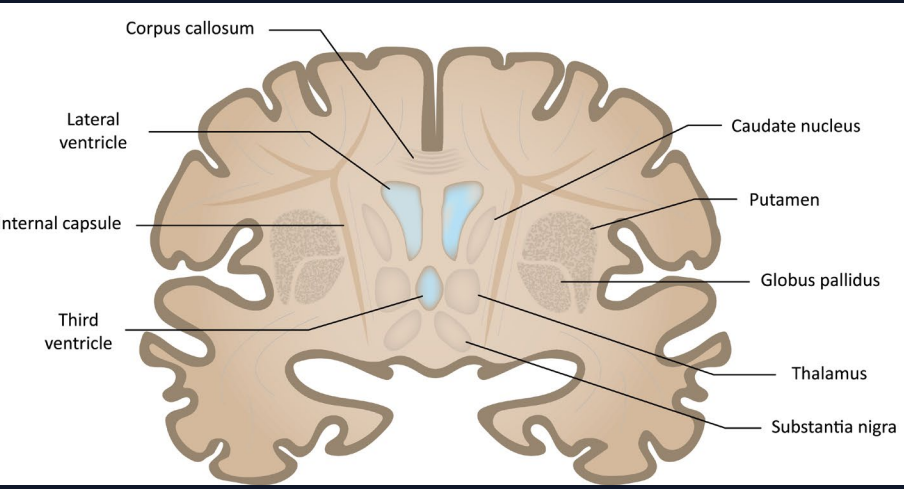
- **High prevalence of postural instability/gait disorder (PIGD)**:
  - Affects the majority of PD patients
  - Subtle gait changes may appear early; postural instability typically emerges later
- **Major fall risk, QoL impact, institutionalization, and mortality**:
  - Up to **60–80%** of PD patients experience falls annually
    - PIGD is a leading cause of hip fractures, nursing home placement, and reduced life expectancy.
    - It significantly impairs mobility, independence, and quality of life while driving higher healthcare costs and caregiver burden.
    - Relatively levodopa-resistant

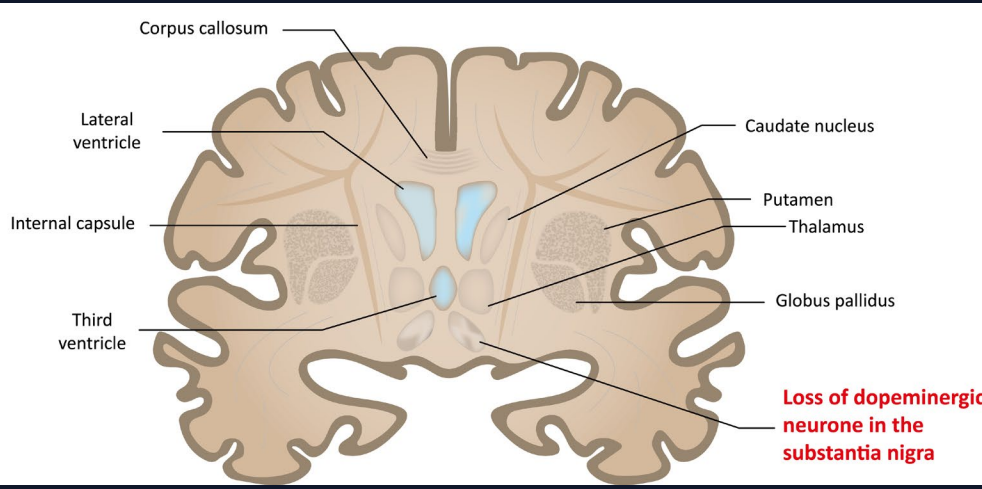
Neuroanatomy:

- The Basal Ganglia
- Direct and Indirect Pathways



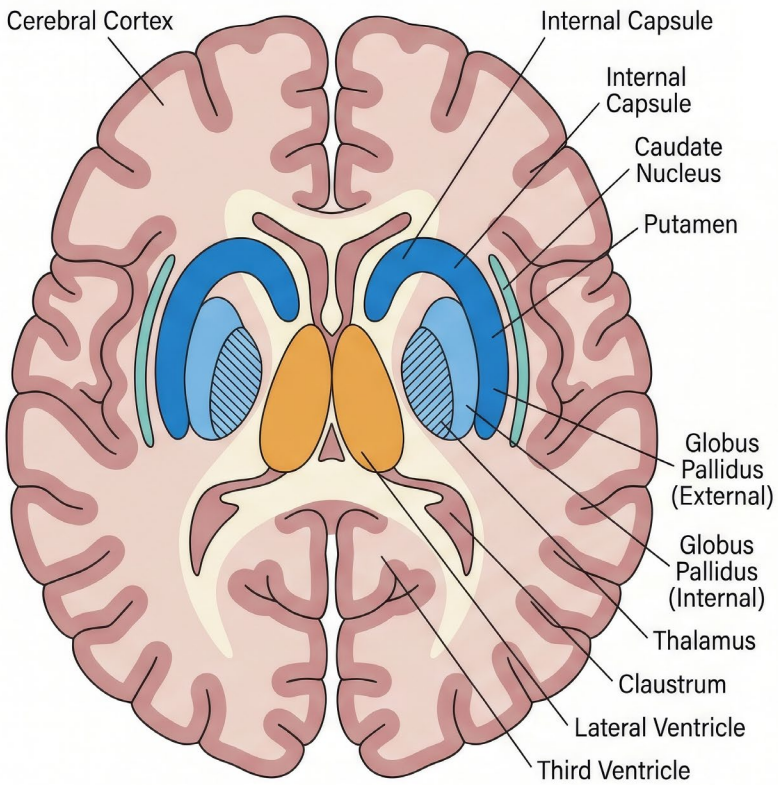
The Basal Ganglia = Your Brain's "Automatic Pilot" for Movement



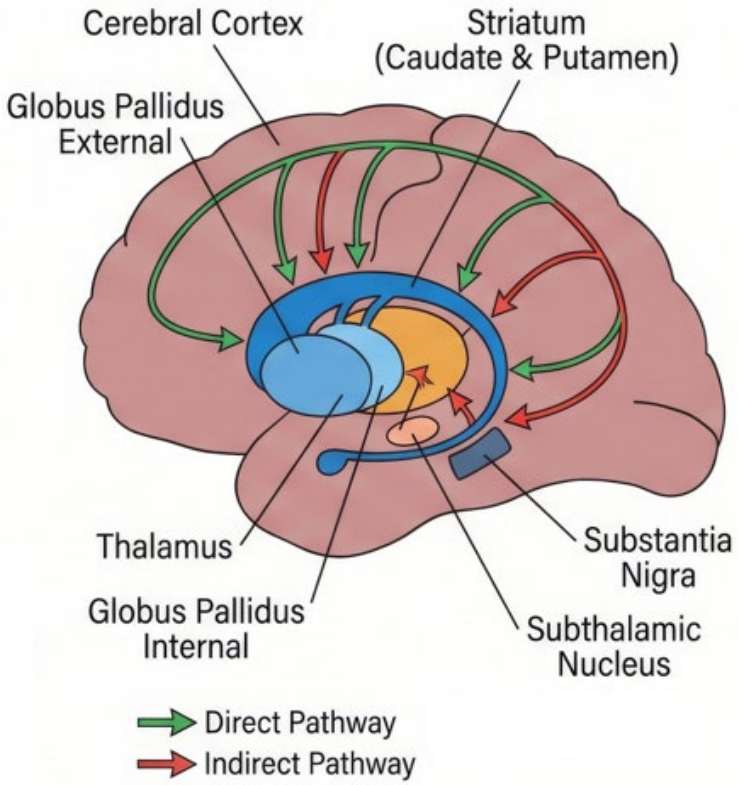


Think of the basal ganglia as the part of the brain that makes smooth, automatic movements happen without you having to think about every step (like walking, standing up straight, or swinging your arms while walking).

View (1) – Axial Section



View (2) – Lateral View Schematic



Caudate Nucleus: C-shaped structure involved in **cognitive and associative functions** (planning, learning, habit formation). Receives widespread cortical input and helps modulate goal-directed behavior.

Putamen: Primarily involved in **motor control**; works with the caudate as the main input nucleus of the striatum. Critical for automatic execution of learned movements and motor habit formation.

Globus Pallidus (externa & interna): GPi = major output nucleus (tonic inhibition of thalamus); GPe = relay in the indirect pathway. In PD: GPe is suppressed → STN overactive → GPi overactive → excessive braking of movement.

Substantia Nigra (pars compacta): Major source of **dopamine** to the striatum. Degeneration here in PD leads to the classic motor symptoms by disrupting the direct (facilitatory) and indirect (inhibitory) pathways.

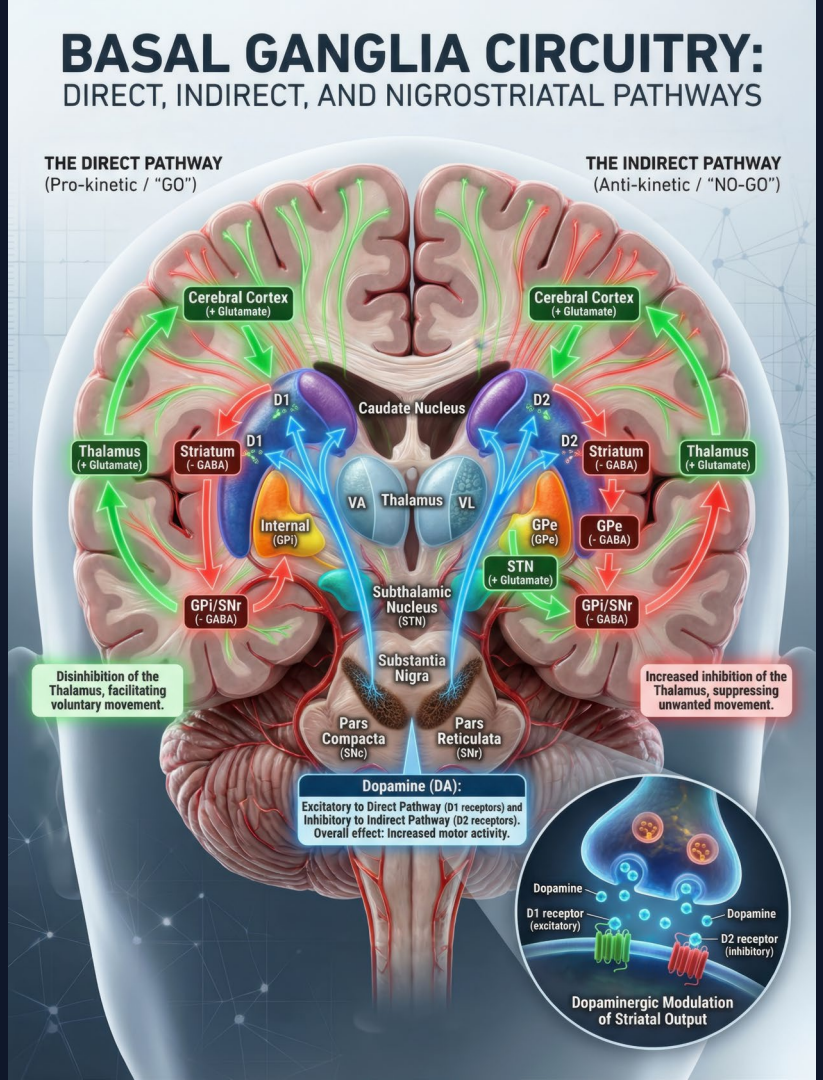
Subthalamic Nucleus (STN): Small but powerful excitatory nucleus. Part of the **indirect pathway**; provides glutamatergic drive that helps suppress unwanted movements. Hyperactivity in PD contributes to bradykinesia/rigidity (common DBS target).

Substantia Nigra pars reticulata (SNr): Output nucleus (along with GPi) that sends inhibitory (GABAergic) projections to the thalamus. Acts as a “brake” on movement; dopamine loss leads to excessive inhibition.

Nucleus Accumbens (part of ventral striatum): Involved in **reward, motivation, and limbic-motor integration**. Links emotion to action; relevant for non-motor PD symptoms like apathy and depression.

Striatum = The main “input station” Made up of the **Caudate** + **Putamen.** It receives messages from the thinking part of the brain (cortex) and decides “Should we move?”

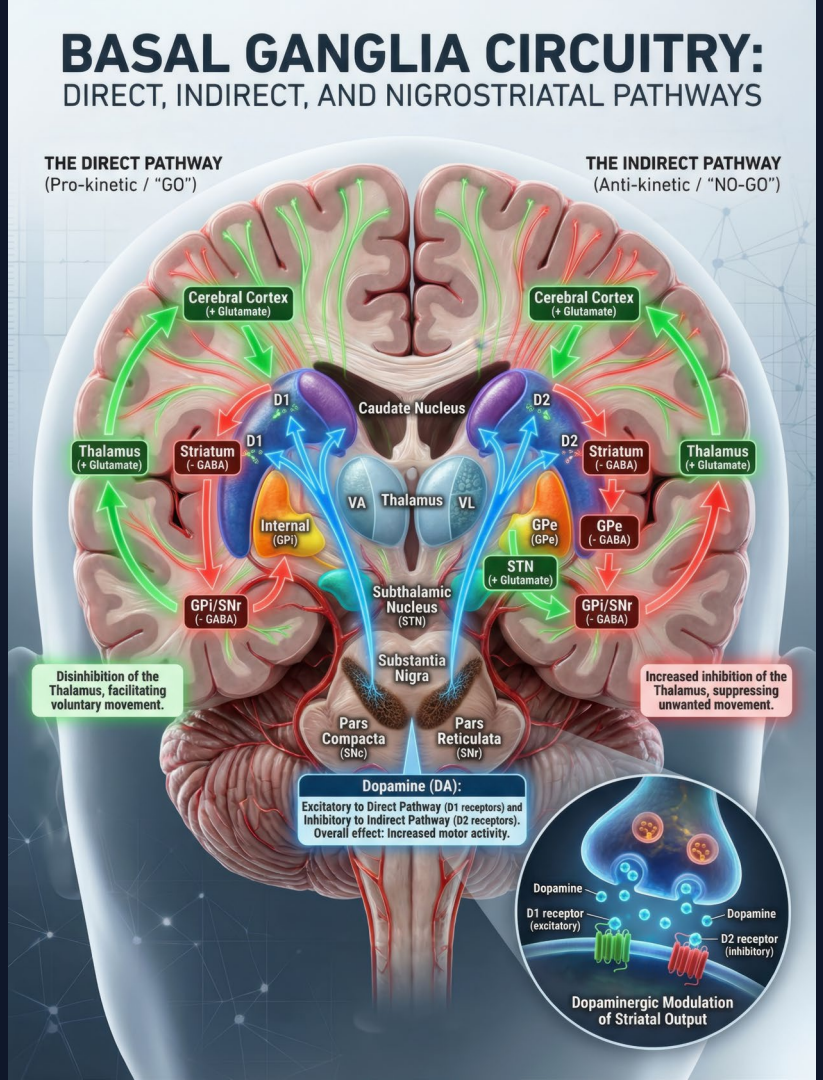
Globus Pallidus (GPi & GPe) = The “output brakes & gas” Acts like a volume control or gatekeeper. It tells the thalamus (relay station) whether to let movement signals through to the muscles.



Subthalamic Nucleus (STN) = The “amplifier” A small but loud structure that helps fine-tune and focus movements.

Substantia Nigra = The **Dopamine Factory** Produces dopamine (the “smooth operator” chemical). In Parkinson’s, this factory breaks down → less dopamine → movement becomes slow, stiff, and shaky.

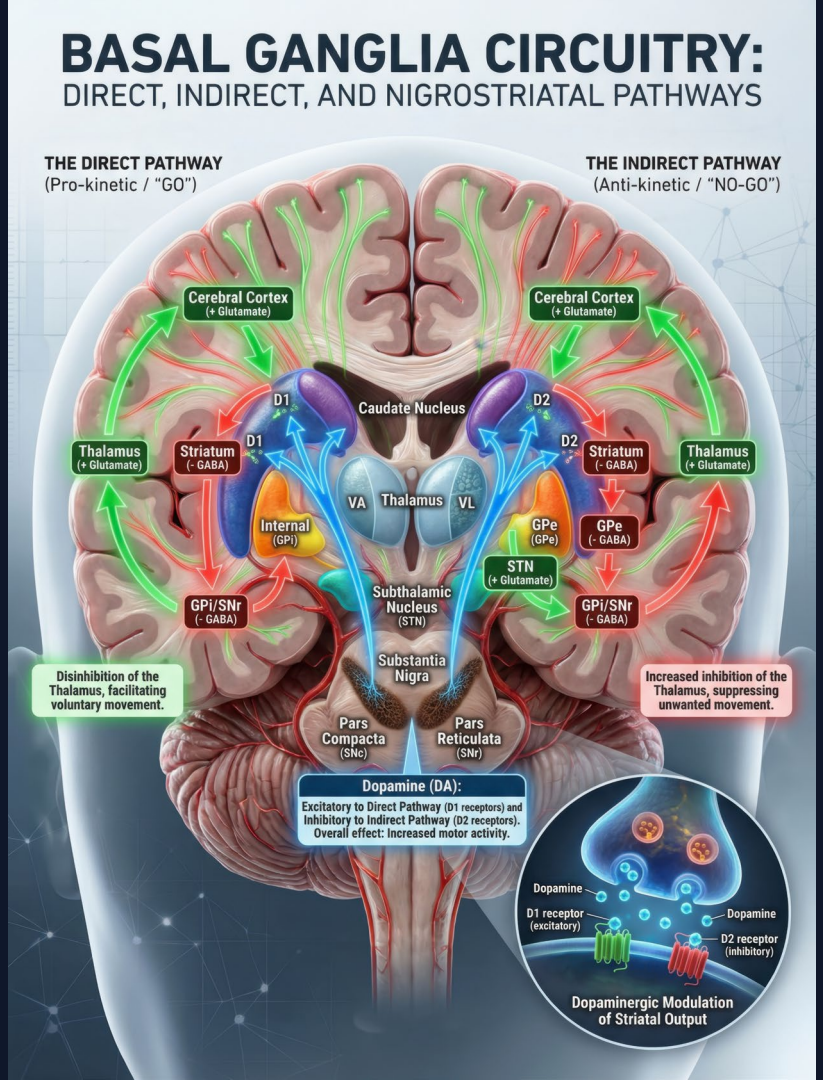
Direct Pathway (“GO!”): Pushes the gas pedal — helps **start and scale** movements (bigger steps, easier walking).



Indirect Pathway (“NO!”): Applies the brakes — stops unwanted or extra movements so your actions stay smooth and purposeful.

Key Question:

Explain why dopamine depletion causes a movement disorder?



In Parkinson's Disease

Dopamine depletion → **underactive direct** + **overactive indirect** pathways

Result: Excessive thalamic inhibition → bradykinesia, reduced amplitude, akinesia, and impaired automatic gait/postural control

Mesencephalic Locomotor Region (MLR)

The vital gateway connecting higher-order brain centers (like the basal ganglia and motor cortex) to the spinal central pattern generators (CPGs) in the spinal cord that produce rhythmic walking movements

The MLR is principally composed of two distinct nuclei:

- Cuneiform Nucleus (CnF): A dorsal cluster of glutamatergic (excitatory) neurons. It is responsible for initiating fast-paced locomotion and governing a broad range of speeds, including high-speed escape behaviors.
- Pedunculopontine Nucleus (PPN): A ventral, neurochemically mixed hub (containing glutamatergic, cholinergic, and GABAergic populations). It primarily regulates slower, exploratory walking gaits, adjusts posture, and connects directly with sleep-wake and arousal pathways.

Neural Circuitry & Pathway

- Upstream Inputs: The MLR receives commands and context-driven triggers from the motor cortex, superior colliculus (visual/orienting signals), and the basal ganglia. Under resting conditions, the basal ganglia send tonic inhibition to the MLR to prevent involuntary movements; this inhibition is paused to initiate gait.
- Downstream Relays: Rather than routing straight to muscles, the MLR activates reticulospinal neurons (RSNs) located in the medullary reticular formation of the hindbrain.
- Spinal Execution: The RSNs project downward through the spinal cord to trigger Central Pattern Generators (CPGs), which autonomously coordinate the alternating contraction of flexor and extensor leg muscles.

Therapies:

- Pharmacological
- Surgical
- Adjunct



Pharmaceutical Therapies & Limitations

Levodopa, dopamine agonists, etc.

- Do not halt progression or address α-synuclein
- Limited on PIGD, gait freezing, non-motor
- Opportunity for chiropractic neurology



Deep Brain Stimulation & Limitations

AI Adaptive DBS

- Do not halt progression or address α-synuclein
- Doesn’t rehab faulty sensory integration deficits
- Opportunity for chiropractic neurology



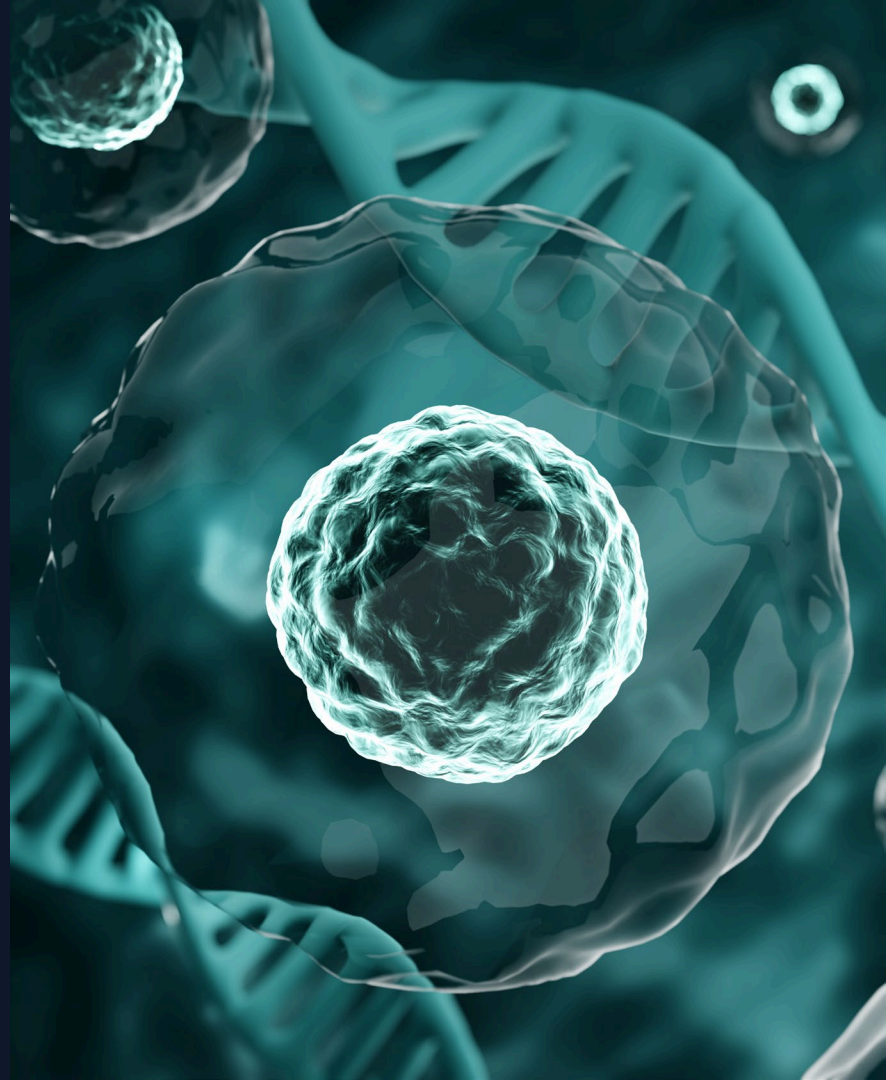
Stem Cell & Peptide Therapies

Stem Cell Therapies (Emerging)

- Promising potential to **replace lost dopamine-producing neurons** and restore function in the substantia nigra, going beyond symptom management.
- May offer **longer-lasting or disease-modifying benefits** with a single or limited intervention, reducing reliance on daily medications.

Peptides (Supportive Adjuncts)

- **Target mitochondrial function and neuroprotection** (e.g., MOTS-c), helping improve cellular energy, reduce oxidative stress, and support overall brain health.
- Enhance **recovery, inflammation control, and neuroplasticity**, complementing rehab drills for better gait, balance, and resilience.



Visual-Vestibular Drivers of Postural Instability and Gait Dysfunction



The Paradigm Shift
From Structure to Sensory Integration

Traditional Lens

- Posture = structural problem
- Stooped spine → adjust it
- Gait = joint mobility
- Result: short-lived change

Chiropractic Neurology Lens

- Posture = output of sensory integration
- Stooped spine → ask *why the brain chose it*
- Gait = timing, scaling & sensory weighting
- Result: train the system that produces the posture

The adjustment still matters. It's one input, not the whole plan.

Normal Function
(The ‘GPS + Camera’ System)

Your brain keeps you upright and moving smoothly by constantly combining three sensory systems:

- Vestibular system (inner ear): Acts like your internal GPS and gyroscope. It senses head position, movement, and gravity. It tells the brain “You’re tilting forward” or “You’re turning” so it can make automatic corrections.
- Visual system: Acts like a camera — provides information about where your body is in space relative to the environment (walls, floor, horizon).
- Proprioception: Muscle/joint sensors that tell the brain where the body is positioned.

These three systems are integrated in the brainstem, cerebellum, and cortex, with the basal ganglia helping scale and time the response. In healthy people, the brain automatically blends them so you stay balanced without thinking about it — especially during walking, turning, or recovering from a stumble.



PD damages more than just the dopamine system in the substantia nigra. It also disrupts sensory integration, particularly:

- Vestibular dysfunction (common in PD 44%): Reduced ability to accurately interpret head position and movement signals.
  - Impaired vestibulo-ocular reflex (VOR) → eyes don't stay steady when the head moves.
  - Poor vestibular-evoked postural reflexes (the automatic "catch yourself" reactions when you tilt or are pushed).

- Slower visual processing and visuospatial orientation.
  - Difficulty using visual cues for self-motion perception.
  - Patients often become over-reliant on vision because vestibular and proprioceptive signals are unreliable.

- Multisensory integration failure.
  - The brain has trouble combining visual + vestibular + proprioceptive information (especially when one input is unreliable, like in the dark or on uneven surfaces). This leads to sensory re-weighting problems — the brain doesn't properly prioritize the best available sense.







How Sensory Integration Deficits Contribute to the Classic PD Gait & Posture Problems

- Stooped posture → axial rigidity + an altered sense of “upright” + poor trunk proprioception
- Festination → loss of automatic step scaling + forward-shifted center of mass
- Freezing (turns, doorways, starts) → overloaded executive control + brainstem (PPN) loss; sensory demands spike during turns
- Falls & retropulsion → slowed automatic postural corrections when sensory input is inaccurate or late
- Worse in the dark or busy settings → over-reliance on vision fails when vision is poor

Key Question:
Why do motor symptoms persist despite levodopa/DBS?

- **Not purely dopaminergic. Gait and balance problems persist despite optimal dopamine treatment**

- **Widespread α-synuclein pathology**
  - ✓ **Cholinergic loss (PPN, brainstem)**
  - ✓ **Noradrenergic & serotonergic systems**
  - ✓ **Brainstem sensory-integration & vestibular areas**

- **Deconditioning + negative neuroplasticity. The brain learns inefficient movement patterns**

Neurological vs Orthopedic
3 Bedside Clues

1. **Lie them down**
   - ✓ Flexion corrects supine → **neurologic** (camptocormia)
   - ✓ Stays fixed → **orthopedic** (structural kyphosis)

2. **Cue them**
   - ✓ Gait improves with rhythm or floor lines → **neurologic**
   - ✓ No change → look at the joints

3. **Check the timing**
   - ✓ Worse when meds wear off (“OFF”) → **neurologic**
   - ✓ Same all day; pain-limited → **orthopedic**

Most patients have both. Treat the main driver first.

Not Every Shuffle Is Parkinson's: Suspect It? Refer It.

Common Mimickers

- PSP: early backward falls, limited vertical gaze
- MSA: early, severe autonomic failure; cerebellar signs
- Lewy body dementia: early cognitive change, hallucinations
- NPH: wide-based "magnetic" gait + urinary urgency + memory change
- Vascular parkinsonism: lower-body predominant, step-wise decline
- Drug-induced: symmetric; check for metoclopramide, antipsychotics

Red Flags → Refer

- ❑ Recurrent falls or rapid decline early in the disease
- ❑ Fainting or severe orthostatic hypotension
- ❑ Fall with head strike on a blood thinner → same day / ER
- ❑ New or rapidly worsening swallowing problems

Our Role: Co-Manage, Not Sole-Manage

Chiropractic:

- **Assessments**
  - ❑ Retropulsion/Pull Test
  - ❑ Timed Up & Go (dual-task)
  - ❑ Mini-BEST Test
  - ❑ Vestibular: VOR
  - ❑ Subjective Visual Vertical



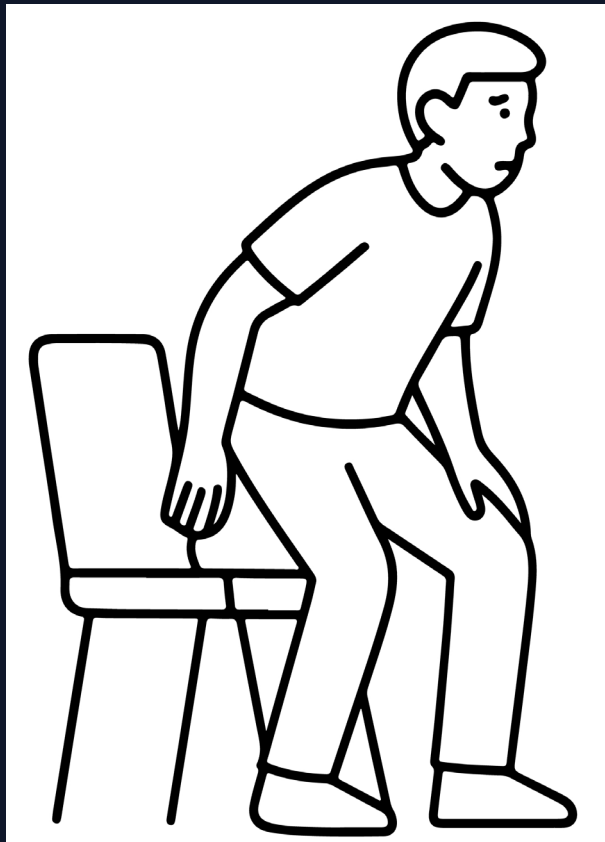
Retropulsion / Pull Test (MDS-UPDRS 3.12)

- What it tests: Reactive postural control, the automatic “catch yourself” response
- How: Patient stands, eyes open, feet comfortably apart. Stand behind, with a wall behind you. First pull = gentle demonstration (not scored). Second pull = quick, firm pull at the shoulders
- Scoring:▪ Normal: recovers in 1–2 steps▪ Abnormal: 3+ steps, or no protective step (would fall if not caught)
- Clinical Pearl: Combined with fall history, the strongest single predictor of falls. An abnormal pull test + prior falls = high risk



Timed Up & Go (TUG) – Especially Dual-Task Version

- **What it tests**: Functional mobility, gait speed, and divided attention (real-world cognitive-motor integration).
- **How to perform**: Rise from chair, walk 10 feet, turn, walk back, sit down.
  - Dual-task: Add counting backward or carrying a cup.
- **Interpretation in PD**: >11.5 seconds = increased fall risk.
  - Dual-task: compare to the standard TUG. A big slowdown, hesitation, or freezing on the turn reveals deficits the standard test can miss
- **Clinical Pearl**: Simple, quick, office-friendly test that mimics daily life challenges.



Mini-BESTest

- **What it tests**: Comprehensive dynamic balance (anticipatory, reactive, sensory orientation, gait).
- **How to perform**: 14 items (e.g., sit-to-stand, single-leg stance, tandem walk, pull test, eyes closed on foam).
  - Mini-Best Test Pdf
- **Scoring**: 0–28 (higher = better). Below ~19–20 suggests high fall risk in PD
- **Clinical Pearl**: Validated for identifying fallers in PD; breaks balance into 4 systems so you know what to train

ANTICIPATORY
1. Sit to stand
2. Rise on Toes
3. Stand on One Leg

REACTIVE POSTURAL CONTROL
4. Compensatory Stepping Correction – FWD
5. Compensatory Stepping Correction – BKW
6. Compensatory Stepping Correction – Lateral

SENSORY ORIENTATION
7. Stance (Feet Together); Eyes Open, Firm Surface
8. Stance (Feet Together); Eyes Closed, Foam Surface
9. Incline – Eyes Closed

DYNAMIC GAIT
10. Change in Gait Speed
11. Walk With Head Turns – Horizontal
12. Walk With Pivot Turns
13. Step Over Obstacles
14. Timed Up & Go With Dual Task

Vestibular: VOR (Vestibulo-Ocular Reflex) Testing

- **What it tests**: Ability to keep eyes stable on a target during head movement (key visual-vestibular integration).

- **How to perform** (bedside):
  - Head Thrust / Head Impulse Test: Quick, small head turns while the patient fixes on a target. Look for corrective saccades.
  - Dynamic Visual Acuity: Read an eye chart with head still vs. oscillating head.

- **Interpretation in PD: VOR dysfunction found in ~44% (vHIT). Corrective saccades = likely peripheral vestibular loss. DVA drop of 3+ lines = poor gaze stability**

- **Clinical Pearl: Poor gaze stability destabilizes walking with head turns and turning, both common triggers for falls and freezing**

Subjective Visual Vertical (SVV): The Bucket Test

- **What it tests: The patient's internal sense of "upright." It depends on the otoliths (utricle) and the brain's gravity-processing pathways**
- **How:**
  - **A straight line inside the bucket, a protractor on the outside**
  - **The patient looks into the bucket, with no other visual references**
  - **Rotate the bucket; the patient says when the line looks vertical**
  - **Do 6–10 trials, alternating starting direction, and average them**
- **Normal: within about ±2–2.5°**
- **Abnormal: a consistent tilt to one side**
- **In PD: Abnormal SVV has been reported in patients with lateral trunk lean (Pisa syndrome)**
- **Clinical Pearl: If they lean and their SVV is off, their sense of upright may be off, not just their muscles**

Chiropractic:

- **High-Yield Strategies**
  - ❑ Sit to Stand
  - ❑ Downhill Wedge
  - ❑ Rhythm & Gait
  - ❑ Supine Floating



How Cueing Works: Bypassing the Autopilot

- PD damages automatic (habitual) control: the basal ganglia “autopilot”
- Goal-directed control is relatively spared: attention, premotor cortex, cerebellum
- Cues switch walking from autopilot to manual, using circuits that still work
- Cue types:
  - ✓ Auditory: metronome, music
  - ✓ Visual: floor lines, laser cane or shoes
  - ✓ Tactile: vibrating wristband or ankle device
  - ✓ Internal / self-generated: singing, counting, “BIG steps,” vocalizing
- Benefits fade without practice → home program is essential (RESCUE trial)
- Cueing uses attention → train dual-tasking on purpose

No error: No Learning!

- **The brain learns from mistakes:** inferior olive → cerebellum; error signals drive motor adaptation
- **A still patient isn't learning:** firm floor + eyes open = no challenge, no error, no change
- **The cerebellum is relatively spared in PD** and appears to compensate for the damaged basal ganglia
- **Find the challenge point:** hard enough to create errors, safe enough to prevent falls
- **Progress one variable at a time:** surface • vision • speed • head movement • dual-task

Sit-to-Stand

- Why: Builds leg strength and power for gait initiation, one of the hardest daily tasks in PD
- Cue: Big breath in → loud “RAH!” or “UP!” on the way up (exhale with effort)
- How it works:▪ Cue: shifts control from autopilot to goal-directed▪ Arousal: brainstem/noradrenergic boost▪ StartReact: loud stimulus → reticulospinal pathway bypasses the basal ganglia▪ Effort recalibrates amplitude (LSVT principle)
- Progressions: Standard chair → lower seat → no hands → dual-task (count backward) → stand from floor

Downhill Wedge Drill (Visual-Vestibular Integration)

- Why: Decline pitches the body forward → posterior chain (calves, hamstrings, back & neck extensors) must work to stay upright. Challenges vestibular + proprioceptive input and gaze stability
- How: Stand on decline wedge (foam to progress). Add head turns, vertical & horizontal saccades, arm movements
- Cue: “Eyes on target, head steady”
- Progressions: Static → dynamic → walking on/off wedge → dual-task
- Safety: Gait belt / corner or counter; check orthostatic BP; build duration gradually

Rhythmic Vocal Cueing + Exaggerated “Soldier” Gait

- Why: Self-generated rhythm (voice) replaces lost internal timing; exaggerated amplitude fights bradykinesia and shuffling
- How: Walk while gargling/growling or saying “BIG STEPS” loudly with each step. Big arm swing, tall posture
- Cue: “Think BIG: chest up, arms swinging like a soldier!”
- Progressions: Metronome at own cadence → 110–120% → over obstacles → dual-task

Anti-Gravity / Supine Floating Reset

- Why: Removes postural demand so the patient can reduce axial rigidity and train extension (countering the flexed PD posture) without fighting gravity

- How: Supine on mat or table with knees bent or legs supported; encourage deep breathing + gentle rocking or head turns. Progress to bridging or “floating” limbs.

- Cue: “Let your body feel light — breathe and lengthen your spine.”

- Progressions: Supine → seated → standing weight shifts → dynamic gait.

Building the Session: Adjustment + Drills

Sample 30-minute visit:

- ❑ Re-test one marker (TUG or pull test): 3 min
- ❑ Adjustment / soft tissue for axial mobility: 5–8 min
- ❑ Supine reset → seated → standing drills: 10 min
- ❑ Gait with cueing → add dual-task: 7 min
- ❑ Re-test same marker + review home program: 3 min

Progress one variable at a time: surface • vision • speed • head movement • dual-task

Dual-task ladder: motor (carry a cup) → simple cognitive (count) → complex (serial 7s, naming)

Schedule during “ON” medication time

Safety Screen Before You Treat

- ❑ **Orthostatic BP:** supine → standing at 1 and 3 min. A drop of ≥20 systolic or ≥10 diastolic = orthostatic hypotension

- ❑ **Bone health:** higher osteoporosis and fracture risk, so modify thrust and force

- ❑ **DBS hardware:**
  - ✓ **No diathermy** (shortwave, microwave, therapeutic ultrasound) anywhere on the body
  - ✓ Know where the leads and wire run (behind the ear, down the neck to the chest)

- ❑ **Guarding:** gait belt for every standing drill; a wall or counter within reach

- ❑ **Swallowing:** caution with supine work after meals and with neck extension

The 4-Week Home Program

3 Times Daily, 15–20 minutes, during “ON” time, next to a counter or in a corner

Week 1: Foundation

Supine reset, 3 min
Sit-to-stand with “UP!” (2 × 8)
Walk with a metronome (2 × 3 min)

Week 2: Amplitude

“BIG” soldier gait with gargle or growl
Sit-to-stand from a lower chair
Weight shifts at the counter

Track it: daily checklist + falls/near-falls diary → re-test at week 4

The 4-Week Home Program

3 Times Daily, 15–20 minutes, during “ON” time, next to a counter or in a corner

Week 3: Sensory challenge

- Foam slant, 5–10 min blocks (caregiver nearby)
- Head turns while walking
- Feet together, eyes closed at the counter
- Turning practice: “wide U, not a pivot”

Week 4: Dual-task & real life

- Walk + count backward
- Carry an object while turning
- Doorway practice with floor tape

Track it: daily checklist + falls/near-falls diary → re-test at week 4

Exercise in Parkinson's: A Powerful, Safe Intervention

One of the most effective, accessible interventions for PD, improving motor symptoms, gait, balance, and quality of life, with a favorable safety profile when appropriately prescribed.

- ✓ Reduces falls: ~26% lower fall rate (Cochrane 2022)
- ✓ Increases BDNF: supports neuroplasticity; may support dopamine signaling
- ✓ Amplitude & rhythm programs work: LSVT BIG, PWR!Moves, boxing, dance
- ✓ Non-motor benefits: mood, cognition, sleep



Environmental & Lifestyle Support

Reduce ongoing exposures:

- ✓ Test private well water
- ✓ Ask about past and present pesticide and herbicide use, at home and at work
- ✓ Protective equipment, or better, stop using yard chemicals

Lifestyle factors with supportive evidence:

- ✓ Regular aerobic exercise
- ✓ Mediterranean / MIND-style diet (observational evidence)
- ✓ Sleep quality
- ✓ Constipation management

Be clear with patients: no “detox” protocol has been shown to slow PD

Clinical Pearls: Gut–Brain & Oculomotor Links

Gut–Brain

- ✓ Braak hypothesis: α-synuclein may begin in the gut or olfactory system and spread to the brainstem
- ✓ Constipation, loss of smell, and dream enactment (REM sleep behavior disorder) can come years before motor signs
- ✓ Ask every patient: bowels, smell, dreams
- ✓ Constipation slows levodopa absorption → less predictable “OFF” time

Oculomotor

- ✓ Hypometric (undershooting) saccades; jerky pursuit
- ✓ Convergence insufficiency → reading trouble, double vision
- ✓ Poor visual scanning → trips and doorway freezing
- ✓ Limited vertical gaze → think PSP and refer

What to Expect: Measuring Outcomes

Re-test at 4 weeks:

- TUG + dual-task TUG
- Mini-BESTest
- 10-meter walk (gait speed)
- Freezing questionnaire (NFOG-Q)
- Falls diary

What the evidence supports:

- ✓ Exercise: ~26% fewer falls in mild–moderate PD (Cochrane 2022)
- ✓ Cueing training improves gait and freezing; benefits fade without practice (RESCUE)
- ✓ Amplitude training (LSVT BIG) improves motor scores and TUG (Ebersbach 2010)
- ✓ Aquatic therapy: balance gains similar to land-based therapy, with better TUG (Carroll 2020)

Summary: 5 Take-Home Points

1. Postural instability in PD is only partly dopaminergic, so it persists despite levodopa and DBS
2. Suspect PD or a mimicker? Refer. We co-manage, not sole-manage
3. Test fall risk in minutes: pull test, TUG, Mini-BESTest, VOR, SVV
4. Cue around the broken autopilot, and progress the challenge: no error, no learning
5. Screen for safety first, start early, and send every patient home with a program

Thank You
Q&A

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