

PARKINSON'S DISEASE

Comprehensive Physiotherapy Guide
for Students & Practitioners

Chapter Overview

PART I: Disease Pathology

- Epidemiology & Etiology
- Pathophysiology & Stages

PART II: Clinical Presentation

- Motor & Nonmotor Symptoms
- Medical Diagnosis & Classification

PART III: PT Management

- Examination & Evaluation
- Intervention Strategies

Definition: Parkinson's Disease

Progressive CNS disorder with motor & nonmotor symptoms

CARDINAL MOTOR FEATURES (≥ 2 required for diagnosis):

- Rigidity: resistance to passive motion
- Bradykinesia: slowness of movement
- Tremor: involuntary shaking (rest tremor)
- Postural Instability: later stage feature

Insidious onset with slow progression

Variable rate of disease progression

Epidemiology & Incidence

PREVALENCE:

- 1 million Americans affected
- 7-10 million people worldwide
- 2% of people >65 years old
- Second only to Alzheimer's among neurodegenerative disorders

DEMOGRAPHICS:

- Average age of onset: 50-60 years
- Only 4-10% with early-onset PD (<60 years)
- Men affected 1.2-1.5× more than women
- Prevalence expected to increase with aging population

Types of Parkinsonism

IDIOPATHIC PARKINSON'S DISEASE (78% of cases):

- Unknown etiology; may be genetic or sporadic
- Late-onset: >40 years (sporadic)
- Early-onset: <40 years (often familial)
- Young-onset: 21-40 years
- Juvenile-onset: <21 years

SECONDARY PARKINSONISM:

- Viral, toxic, drug-induced, vascular causes

PARKINSON-PLUS SYNDROMES:

- MSA, PSP, CBD, DLBD - poor L-dopa response

Etiology: Genetic Factors

GENETIC INFLUENCES (<10% of cases):

Identified Gene Mutations:

- PARK1 (alpha-synuclein)
- PINK1
- LRRK2
- DJ-1
- Glucocerebrosidase

Two Gene Categories:

1. Causal genes: produce disease
2. Risk genes: increase susceptibility

ENVIRONMENTAL FACTORS:

Pesticides/Toxins:

- Permethrin, beta-HCH
- Paraquat, maneb
- Agent Orange

Industrial Chemicals:

- Manganese (occupational hazard)
- Carbon disulfide, monoxide

NOTE: Exposure alone insufficient

Secondary Causes of Parkinsonism

VIRAL: Encephalitis lethargica (1917-1926 epidemics)

TOXIC PARKINSONISM:

- MPTP (synthetic heroin contaminant)
- Manganese exposure (miners)

DRUG-INDUCED (reversible):

- Antipsychotics: chlorpromazine, haloperidol, risperidone
- Antidepressants: amitriptyline, amoxapine
- Antihypertensives: methyldopa, reserpine
- High doses particularly problematic in elderly
- Usually reversible with drug withdrawal

Parkinson-Plus Syndromes

Group of neurodegenerative diseases affecting basal ganglia

TYPES (more rapid progression, poor L-dopa response):

- Striatonigral degeneration (SND)
- Multiple system atrophy (MSA)
- Progressive supranuclear palsy (PSP)
- Cortico-basal ganglionic degeneration (CBGD)
- Lewy body disease (DLBD)
- Alzheimer's with parkinsonism

DIAGNOSTIC FEATURE: No improvement with L-dopa (apomorphine test)

Basal Ganglia Anatomy & Function

BASAL GANGLIA STRUCTURES:

- Caudate nucleus, putamen (striatum)
- Globus pallidus (internal/external)
- Subthalamic nucleus
- Substantia nigra (pars compacta/reticulata)

FUNCTIONS:

- Motor control & movement initiation
- Regulates thalamo-cortical activation
- Motor learning & procedural memory
- Cognitive & emotional functions

Basal Ganglia Motor Circuits

DIRECT LOOP (facilitates movement):

Cortex → Putamen → GPi → VL-Thalamus → SMA

Excitatory connection activates cortex

INDIRECT LOOP (inhibits movement):

Cortex → Striatum → Subthalamic nucleus → GPi → Superior colliculus

Decreases thalamocortical activation

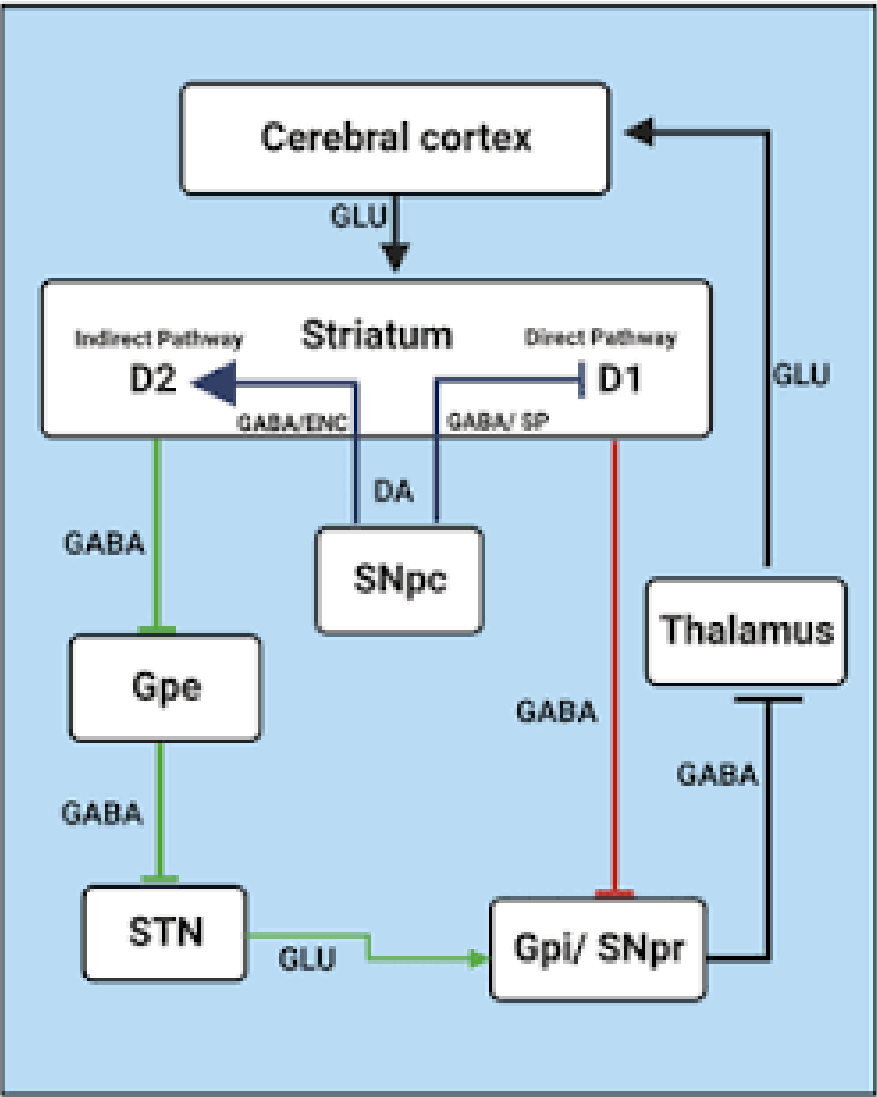
IN PARKINSON'S DISEASE:

↓ Dopamine in substantia nigra pars compacta

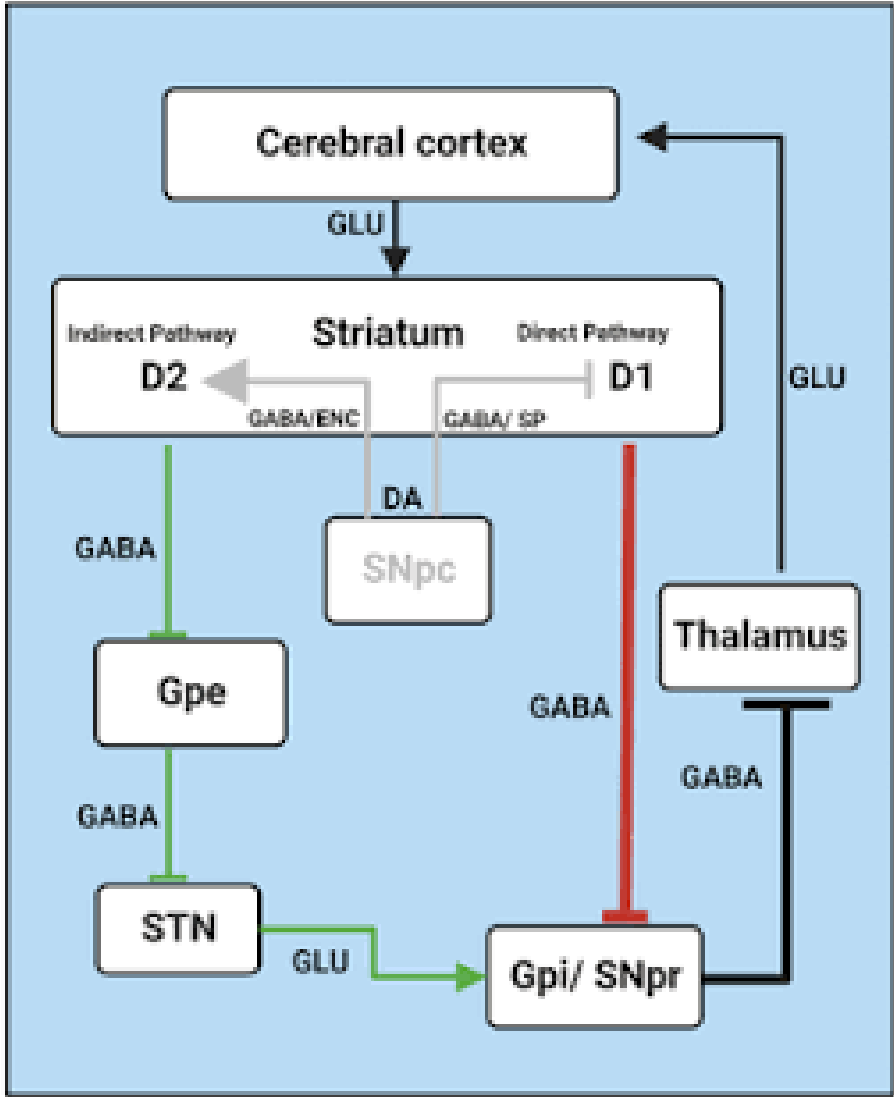
→ Imbalance of direct/indirect loops

→ Hypokinesia (reduced movement)

a)



b)



— Dopaminergic

— Direct Pathway

— Indirect Pathway

→ Excitatory

—| Inhibitory

Pathophysiology of PD

PRIMARY PATHOLOGY:

- Degeneration of dopaminergic neurons in substantia nigra
- Loss of dopamine production (25-50% loss by symptom onset)
- Formation of Lewy bodies (cytoplasmic inclusions)

CLINICAL CORRELATION:

- 30-60% neuronal degeneration before symptoms appear
- Explains late diagnosis despite long subclinical period
- Melanin-containing neurons die → pallor of substantia nigra

NONMOTOR INVOLVEMENT:

- Widespread CNS pathology beyond basal ganglia
- Affects brainstem, limbic system, cortex, PNS

Cardinal Feature #1: RIGIDITY

DEFINITION: Increased resistance to passive motion

CHARACTERISTICS:

- Felt uniformly in agonist & antagonist muscles
- Equal resistance in all directions
- Normal spinal reflexes (differentiates from spasticity)
- Often asymmetrical, especially early disease
- Varies with stress, time of day, medication cycle

DISTRIBUTION:

- First affects proximal muscles (shoulders, neck)
- Progresses to face and extremity muscles
- Becomes bilateral over time

Types of Rigidity

LEAD PIPE RIGIDITY:

- Sustained, uniform resistance
- No fluctuations
- No tremor co-exists
- Smooth, constant feel

COGWHEEL RIGIDITY:

- Jerky, ratchet-like resistance
- Alternating muscle tension/relaxation
- Occurs WITH tremor
- Feels like gears catching

Clinical Impact of Rigidity

FUNCTIONAL CONSEQUENCES:

- Loss of bed mobility
- Loss of reciprocal arm swing during gait
- Difficulty with fine motor tasks
- Postural deformity development

SECONDARY COMPLICATIONS:

- Contractures of hip/knee flexors, plantarflexors
- Shoulder adductors, internal rotators
- Decreased range of motion
- Increased energy expenditure → fatigue

ASSESSMENT: Passive ROM testing in relaxed position

Cardinal Feature #2: BRADYKINESIA

DEFINITION: Slowness of movement

CHARACTERISTICS:

- One of MOST DISABLING symptoms
- Due to insufficient force recruitment at movement initiation
- Patient UNDERSCALES movement commands (internally generated)

SUBTYPES:

- Akinesia: poverty of spontaneous movement
 - Hypomimia (masked face)
 - Loss of associated movements (arm swing)
- Hypokinesia: slowed, reduced-amplitude movements
 - Micrographia (handwriting shrinks)
 - Freezing of gait (FOG): sudden stops

Bradykinesia & Motor Learning

EXTERNAL CUEING HELPS:

- Vision, sound, tactile input can partially ameliorate
- Used therapeutically in treatment
- Bypasses insufficient internal dopaminergic cueing

BRADYPHRENIA:

- Slowed thinking also contributes to movement slowing

ASSESSMENT:

- Timed tests for rapid alternating movements (RAM)
- Observation of fine motor tasks (writing, dressing)
- Movement time and reaction time measurement
- Speed-accuracy trade-off: faster = less accurate

Cardinal Feature #3: TREMOR

DEFINITION: Involuntary shaking from opposing muscle contractions

CHARACTERISTICS (affects ~70% early disease):

- REST TREMOR: present at rest, suppressed by movement, absent in sleep
 - Most common in early PD
 - Mild initially, intermittent
- POSTURAL TREMOR: present against gravity (head, trunk)
- ACTION TREMOR: continues with movement (advanced disease)

FEATURES:

- Often unilateral initially
- Usually mild, intermittent
- Increases with stress, emotion, fatigue

Cardinal Feature #4: POSTURAL INSTABILITY (Part 1)

DEFINITION: Abnormal control of center of mass within base of support

CHARACTERISTICS:

- RARE in early disease (first 5 years)
- Becomes evident as disease progresses
- Late feature, marker of disease severity

UNDERLYING DEFICITS:

- Abnormal, inflexible postural responses
- Abnormal coactivation (rigid body response)
- Loss of normal postural synergies
- Impaired feed-forward, anticipatory adjustments
- Sensorimotor integration impairment

Postural Instability: Clinical Manifestations

EXACERBATING FACTORS:

- Narrowed base of support (tandem/single-limb stance)
- Divided attention (dual-tasking)
- Dynamic activities (reach, walk, turn)
- Perturbed balance challenges

POSTURAL DEFORMITIES (develop progressively):

- Flexed, stooped posture with kyphosis
- Forward head position
- Forward displacement of center of mass
- Contractures of hip/knee flexors, plantarflexors

CONSEQUENCES:

- High fall risk (70% fall within past year)
- ~25% develop hip fracture within 10 years

Secondary Motor Symptom #1: MUSCLE WEAKNESS

CHARACTERISTICS:

- Generalized weakness in all muscle groups
- Decreased torque production at all speeds
- Impaired motor unit recruitment (EMG findings)
- Delayed recruitment with asynchronous firing

DOPAMINE DEPENDENCE:

- Strength increases during 'ON' medication state
- Decreases during 'OFF' state

FATIGUE (common symptom):

- Difficulty sustaining activity
- Progressive weakness during day
- Repetitive tasks decrease in strength/amplitude
- May improve with L-dopa initially (honeymoon period)

Secondary Motor Symptom #2: GAIT DISTURBANCE

GAIT CHARACTERISTICS (13-33% have as initial symptom):

- Reduced arm swing (loss of reciprocal movement)
- Asymmetric movement patterns
- Stooped, flexed posture
- Short-stepped, shuffling pattern
- Reduced trunk rotation

FESTINATING GAIT:

- Progressive increase in speed
- Shortened stride to catch up with COM
- May break into run/trot

FREEZING OF GAIT (FOG):

- Sudden inability to generate stepping
- Triggered by narrow passages, turning, competing stimuli

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Secondary Motor Symptoms Summary

MUSCLE PERFORMANCE:

- Decreased torque production, fatigue, weakness

MOTOR PLANNING DEFICITS:

- Slow movement initiation (start hesitation)
- Difficulty with complex/sequential movements
- Speed-accuracy trade-off
- Prolonged movement preparation time

MOTOR LEARNING:

- Slower learning rates in advanced disease
- Deficits with random practice, complex tasks
- Better with blocked practice, external cues
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Nonmotor Symptoms: Cognitive & Behavior

COGNITIVE DYSFUNCTION:

- Bradyphrenia (slowed thinking)
- PD Dementia: 20-40% of patients
 - Executive function loss (planning, reasoning, judgment)
 - Visuospatial deficits
 - Memory changes, verbal fluency loss
- Risk increases with age (4.4× higher at 80+ years)

PSYCHIATRIC SYMPTOMS:

- Depression: 40% of patients
 - Often precedes motor symptoms
 - Suggests dopamine/serotonin deficiency
- Anxiety: up to 38% of patients

Nonmotor Symptoms: Autonomic Dysfunction

THERMOREGULATION:

- Excessive sweating (often 'ON' state associated)
- Abnormal heat/cold sensations
- Seborrhea (oily skin), seborrheic dermatitis

CARDIOVASCULAR:

- Orthostatic hypotension (OH): common in middle/late PD
 - Symptomatic: lightheadedness, dizziness, syncope
 - Increased fall risk
- Early sympathetic denervation of heart
- Diminished heart function → contributes to fatigue

GASTROINTESTINAL:

- Constipation: common early symptom
- Poor motility, appetite changes
- Sialorrhea (excessive drooling)

Nonmotor Symptoms: Sensory & Speech

SENSORY SYMPTOMS:

- No primary sensory loss
- Paresthesias: 50% experience (numbness, tingling, burning)
- Pain: can be central nociception-related
- Proprioceptive deficits: patients underscale movements

OLFACTORY DYSFUNCTION:

- Loss of smell (anosmia) in up to 100% of patients
- Often YEARS before motor symptoms
- Important for early diagnosis

- Impacts nutrition & diet quality

SLEEP DISORDERS:

- Excessive daytime somnolence
- Insomnia: difficulty falling/staying asleep
- REM sleep behavior disorder (50-60% affected)
 - Dream-enacting behaviors (talking, fighting, thrashing)

Nonmotor Symptoms: Speech & Swallow

SPEECH IMPAIRMENT (75-89% affected):

- Excessive drooling from ↑ production + ↓ swallowing

- Hypokinetic dysarthria:

- Decreased volume (hypophonia)

- Monotone/monopitch speech

- Imprecise articulation

- Uncontrolled rate

- Perceptual paradox: patient thinks normal loudness

- Social consequences: isolation, communication breakdown

- Socially embarrassing

DYSPHAGIA (95% affected):

- Impaired swallowing in all phases

- Abnormal tongue control, delayed swallow

Medical Diagnosis of PD

NO SINGLE DEFINITIVE TEST

DIAGNOSTIC CRITERIA:

- Clinical examination shows ≥ 2 of 4 cardinal features
- History and observation of clinical signs/symptoms

CLINICAL TOOLS:

- Handwriting samples (micrographia)
- Speech analysis (hypokinetic dysarthria)
- Interview focused on developing symptoms
- Physical examination

EMERGING DIAGNOSTIC TOOLS:

- Olfactory testing
- Cardiac sympathetic imaging
- Nonmotor symptom questionnaires

Clinical Course & Progression

DISEASE TIMELINE:

- Subclinical period: at least 5 years (no symptoms)
- Mean duration: ~13 years
- Variable progression rate

PROGNOSTIC FACTORS:

- BETTER prognosis: young age, tremor-predominant
- WORSE prognosis: postural instability/gait disturbance (PIGD)
 - More rapid deterioration
 - Cognitive/behavioral disturbances more common

WITH L-DOPA TREATMENT:

- Generally slower progression
- Improved mortality rates

Hoehn-Yahr Classification of Disability

STAGE I: Minimal disease, unilateral if present

STAGE II: Minimal bilateral/midline involvement, balance intact

STAGE III: Impaired righting reflexes, unsteady when turning/rising

Some activities restricted, but can live independently

STAGE IV: All symptoms present & severe

Standing/walking possible only with assistance

STAGE V: Confined to bed or wheelchair

Unified Parkinson's Disease Rating Scale (UPDRS/MDS-UPDRS)

ORIGINAL UPDRS (4 parts):

- Part I: Mentation, Behavior, Mood
- Part II: Activities of Daily Living
- Part III: Motor Examination
- Part IV: Complications of Therapy

REVISED MDS-UPDRS (improved):

- 48 items (originally 42)
- 4-point scale (0=normal, 4=severe)
- Parts I & II self-administered
- ~30 minutes administration time

ADVANTAGES:

- Better detects subtle changes in mild patients
- Increased focus on nonmotor symptoms
- 'Gold standard' for measuring PD progression

Goals of Pharmacological Management

PRIMARY AIMS:

- Slow disease progression (neuroprotection)
- Symptomatic treatment of motor symptoms
- Symptomatic treatment of nonmotor symptoms

CHALLENGES:

- Increasingly difficult management as disease progresses
- Stage III+ (Hoehn-Yahr) management more complex
- Long-term complications of therapy develop

PRINCIPLES:

- Medication on FIXED SCHEDULE (not PRN) essential
- Consistent timing optimizes effectiveness
- Timing of PT should correlate with medication cycle
- 61% of hospitalized patients NOT receiving meds on time had serious complications

Levodopa/Carbidopa (Sinemet): Gold Sta

MECHANISM:

- L-dopa: dopamine precursor converted to dopamine in brain
- Carbidopa: decarboxylase inhibitor → higher CNS L-dopa, lower peripheral metabolism
- Allows lower L-dopa doses with fewer side effects
- 4-6 year typical therapeutic window
- After: dyskinesias, motor fluctuations develop

FORMULATIONS:

- Immediate-release (IR): short half-life, multiple daily doses
- Controlled-release (CR): sustained-release, less frequent dosing

BENEFITS:

- Controls motor symptoms (bradykinesia, rigidity)
- ↑ movement velocity & strength
- DOES NOT prevent postural instability

HONEYMOON PERIOD:

- Initial dramatic improvement in function

Dopamine Agonists & Other Medications

DOPAMINE AGONISTS (ropinirole, pramipexole, bromocriptine):

- Directly stimulate postsynaptic dopamine receptors
- Used as monotherapy or with L-dopa (L-dopa sparing)
- Reduce rigidity, bradykinesia, motor fluctuations
- Side effects: nausea, hallucinations, impulse control disorders

ANTICHOLINERGICS (trihexyphenidyl, benztropine):

- Moderate tremor & dystonia
- Little effect on other symptoms

MAO-B INHIBITORS (selegiline, rasagiline):

- Slow dopamine degradation
- Modest disease-slowng effect in early PD

Deep Brain Stimulation (DBS)

PROCEDURE:

- Electrodes implanted in subthalamic nucleus (STN) or globus pallidus (GPi)
- High-frequency electrical stimulation
- Pacemaker-like device implanted subclavicularly
- Programmable by physician; patient control via remote

INDICATIONS:

- Advanced PD with medication-resistant motor fluctuations
- Dyskinesias refractory to pharmacological therapy

OUTCOMES:

- 90% achieve tremor suppression
- Substantially increases 'ON' time
- Improves ADL scores, reduces medication needs
- Variable improvement in akinesia, rigidity, gait

Nutritional Management

PROTEIN & L-DOPA INTERACTION:

- Dietary amino acids compete with L-dopa absorption
- HIGH protein impairs medication effectiveness
- Recommendation: $\leq 15\%$ calories from protein

TIMING STRATEGY:

- Shift daily protein intake to EVENING MEAL
- Maximizes L-dopa responsiveness during active hours

GENERAL DIETARY RECOMMENDATIONS:

- High-calorie diet to maintain energy
- Variety of foods with vitamin/mineral supplementation
- ↑ Water and fiber intake (constipation management)

OCCUPATIONAL THERAPY ROLE:

- Adaptive feeding devices for rigidity/bradykinesia
- Strategies to maintain nutritional intake

PT Examination: Components (Part 1)

PATIENT HISTORY:

- Age, sex, onset of symptoms
- Social history, family support, occupational status
- Medical/surgical history, current medications
- Functional status: premorbid & current

SYSTEMS REVIEW:

- Neuromuscular, musculoskeletal, cardiovascular/pulmonary
- Integumentary status

TESTS & MEASURES:

- Cognition: Mini-Mental State Exam (MMSE)
- Psychosocial: Geriatric Depression Scale, Beck Depression Inventory
- Autonomic: orthostatic hypotension, thermoregulation
- Cardiorespiratory: vital signs, aerobic capacity

PT Examination: Motor Function (Part 2)

tone & rigidity:

- Passive ROM testing in relaxed state
- Differentiate lead-pipe vs. cogwheel
- Document body segments affected, severity

Strength:

- Manual Muscle Testing (MMT)
- Isokinetic dynamometry for objective measures
- Assess rate of force development (often impaired)

Tremor:

- Location, persistence, amplitude (severity)
- Rest vs. postural vs. action tremor
- Effect on functional tasks (drinking, writing)
- Stress effect on tremor magnitude

PT Examination: Balance & Gait (Part 3)

BALANCE TESTS:

- Berg Balance Scale (BBS): correlates with UPDRS & fall risk
- Functional Reach Test (FRT)
- Timed Up and Go (TUG): measures mobility & fall risk
- Dynamic Gait Index (DGI): balance during walking

GAIT ANALYSIS:

- 10-Meter Walk Test: speed, stride, cadence
- Observe: arm swing, trunk rotation, posture, gait pattern
- Freezing of Gait Questionnaire (NFOG-Q)
- Complex gait patterns: cross-stepping, braiding

FALL RISK ASSESSMENT:

- Fall history documentation
- Fall risk diary: activity, medication timing, footwear, fatigue
- Cutoff scores for BBS/TUG to identify fallers

Motor Learning Strategies for PD

MOTOR LEARNING DEFICITS IN PD:

- Slower learning rates
- Context-specificity problems
- Deficits with complex/sequential tasks
- Degraded during 'OFF' medication state

PRACTICE PRINCIPLES:

1. BLOCKED practice order (same task repeatedly)
 - Reduces context interference
 - Better than random practice for PD patients
2. REPETITION: critical for procedural skill development
3. FOCUSED ATTENTION: full concentration on desired movement
4. AVOID dual-tasking during learning phase

External Cueing Strategies

RATIONALE:

- Bypasses diminished internal BG cueing
- Utilizes alternative brain pathways (premotor cortex)
- Shifts movement from automatic to conscious control

VISUAL CUES:

- Floor markings perpendicular to gait path (one step apart)
- Laser light signals (mounted on walker/harness)
- Improves stride length & velocity
- Reduces freezing episodes

AUDITORY CUES:

- Rhythmic auditory stimulation (RAS): metronome beat
- Set 25% faster than patient's preferred pace
- Verbal cues: 'Big step,' 'Walk fast,' 'Take large steps'
- More effective for temporal than spatial components

Exercise Training Programs

AMPLITUDE-BASED INTERVENTION (Lee Silverman Voice Treatment BIG):

- Repetitive HIGH-amplitude movements
- High intensity: 8/10 RPE scale
- 1 hour × 4×/week for 4 weeks
- Large, whole-body movements increasing in complexity
- Results: ↑UPDRS scores, TUG, 10-m walk

FLEXIBILITY EXERCISES:

- Static ROM, dynamic ROM, PNF techniques
- 2-3 days/week minimum, ideally 5-7 days
- 4 reps × 15-60 seconds per stretch

- Focus on common limited areas: hip/knee extension, trunk rotation

RELAXATION TECHNIQUES:

- Gentle rocking to reduce rigidity
- PNF rhythmic initiation (RI) technique
- Diaphragmatic breathing with movement

Functional Training: Bed Mobility & Trans

BED MOBILITY:

- Segmental rolling (upper/lower trunk rotation)
- Practice on firm then soft surfaces (home simulation)
- Bridging to improve hip extension & scooting
- Assistive devices: trapeze, satin sheets, bed blocks

SITTING:

- Pelvic mobility exercises on therapy ball
- Anterior/posterior tilts, side-to-side tilts, clock exercises
- Trunk rotations & reaching while maintaining posture

SIT-TO-STAND (often severely impaired):

- Forward trunk flexion (initial rocking enhances relaxation)
- Cueing strategies: counting, hand placement on shoulders
- Progression: firm raised seat → standard height
- Avoid standing directly in front (blocks initial attempts)
- Safety belt if needed

Functional Training: Standing & Balance (

STANDING ACTIVITIES:

- Weight-bearing symmetry over base of support
- Weight shifts: anterior/posterior, lateral
- Trunk rotation with reaching
- Step-ups: low platform, forward/lateral progression
- Backward stepping: strengthens hip/spinal extensors
- Elastic band resistance for challenge

GAIT TRAINING:

- VERTICAL POLES: improve upright posture, arm swing, trunk rotation
- VERBAL INSTRUCTIONAL SETS: 'Walk tall,' 'Walk fast,' 'Take large steps'
- VISUAL CUES: floor markings, laser signals
- AUDITORY CUES: rhythm, music, metronome

Balance Training: Center of Mass & Limits

COM & LOS EDUCATION:

- Teach how COM influences balance
- Practice expanding limits of stability
- Weight-bearing symmetry exercises

BALANCE CHALLENGES:

- Seated: inflatable disc or therapy ball
- Quadruped: contralateral limb lifts over ball
- Kneeling & half-kneeling: on BOSU disc with resistance
- Standing: disc challenges, foam surfaces

DYNAMIC STABILITY TASKS:

- Weight shifts, reaching, axial rotation
- Arm movements: out to side, overhead, across chest
- Vary foot positions: apart, together
- Add voluntary movements: clapping, head turns, leg raises

Locomotor Training & Gait Strategies

TREADMILL TRAINING:

- Motorized treadmill with safety harness
- High-intensity training with incremental speed increases
- Results: normalized gait, improved function, maintained at 4 months
- Acts as external pacemaker for gait rhythm
- Body weight support: 0-20% can be used

TASK-SPECIFIC TRAINING:

- Vary walking surfaces: tile, carpet, grass, ramps, stairs
- Community walking: stairs, curbs, obstacles

- Foot clearance over horizontal markers/lasers
- Complex confined areas (narrow doorways)

FREEZING OF GAIT MANAGEMENT:

- External cues: 'trick' movements (drop tissue to step over)
- Cueing strategies: counting, marching music
- Environmental modifications

THERAPY DOG ASSISTANCE:

- Provides balance support & external cueing of stepping

Specialized PT Interventions

PULMONARY REHABILITATION:

- Diaphragmatic breathing, air-shifting techniques
- Chest wall mobility exercises
- Upper body resistance training
- Results: improved VO_2 , VE, respiratory muscle strength

SPEECH THERAPY:

- Lee Silverman Voice Treatment (LSVT): intensive high-effort exercise
- Focuses on vocal loudness & self-perception recalibration
- Results: increased volume, improved facial expressions

WHOLE BODY VIBRATION (WBV):

- Preliminary evidence: improved strength, gait, balance, tremor
- Needs more high-quality research

SPINAL ORTHOTICS:

- Spinomed thoracolumbar orthosis:
 - Corrects posture, increases vital capacity
 - Biofeedback-induced proprioceptive effect

Patient & Family Education | Case Summary & Key Points

EDUCATION TOPICS:

- Disease process, prognosis, symptom management
- Medications: purpose, dosage, side effects
- Preventative measures for secondary complications
- Strategies for maintaining independence
- Energy conservation & activity pacing
- Fall prevention & recovery techniques
- Community resources & support groups

TEAM APPROACH:

- Multidisciplinary: PT, OT, SLP, psychologist, social worker
- Coordinate care across all disciplines
- Emphasize HOPE TEMPERED WITH REALISM

RESOURCES:

- National Parkinson Foundation (NPF): www.parkinson.org
- Parkinson's Disease Foundation (PDF): www.pdf.org
- Support groups for patients & caregivers

Thank you