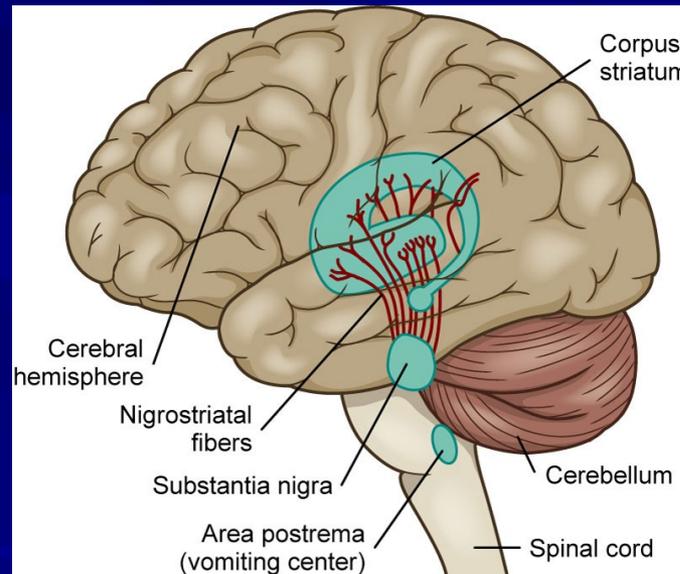


Chronic Neurologic Disorders

Parkinson's Disease



Sheryll Mae M. Coulombe, MSN, RN-BC



Parkinson's Disease (PD)

- Chronic, progressive neurodegenerative disease of the CNS
- Manifesting primarily in motor dysfunction
- Primarily of idiopathic origin
- 1.5x to 2x more common in males
- Begins between ages 40-70 years
 - 4% diagnosed before 50

PD: Etiology & Pathophysiology

- Exact cause of PD unknown
 - Possibly a result between environmental factors and person's genetic makeup
 - Family history in 15% of cases
 - Risk ↑ by well water, pesticides, herbicides, industrial chemicals, wood pulp mills, rural residence

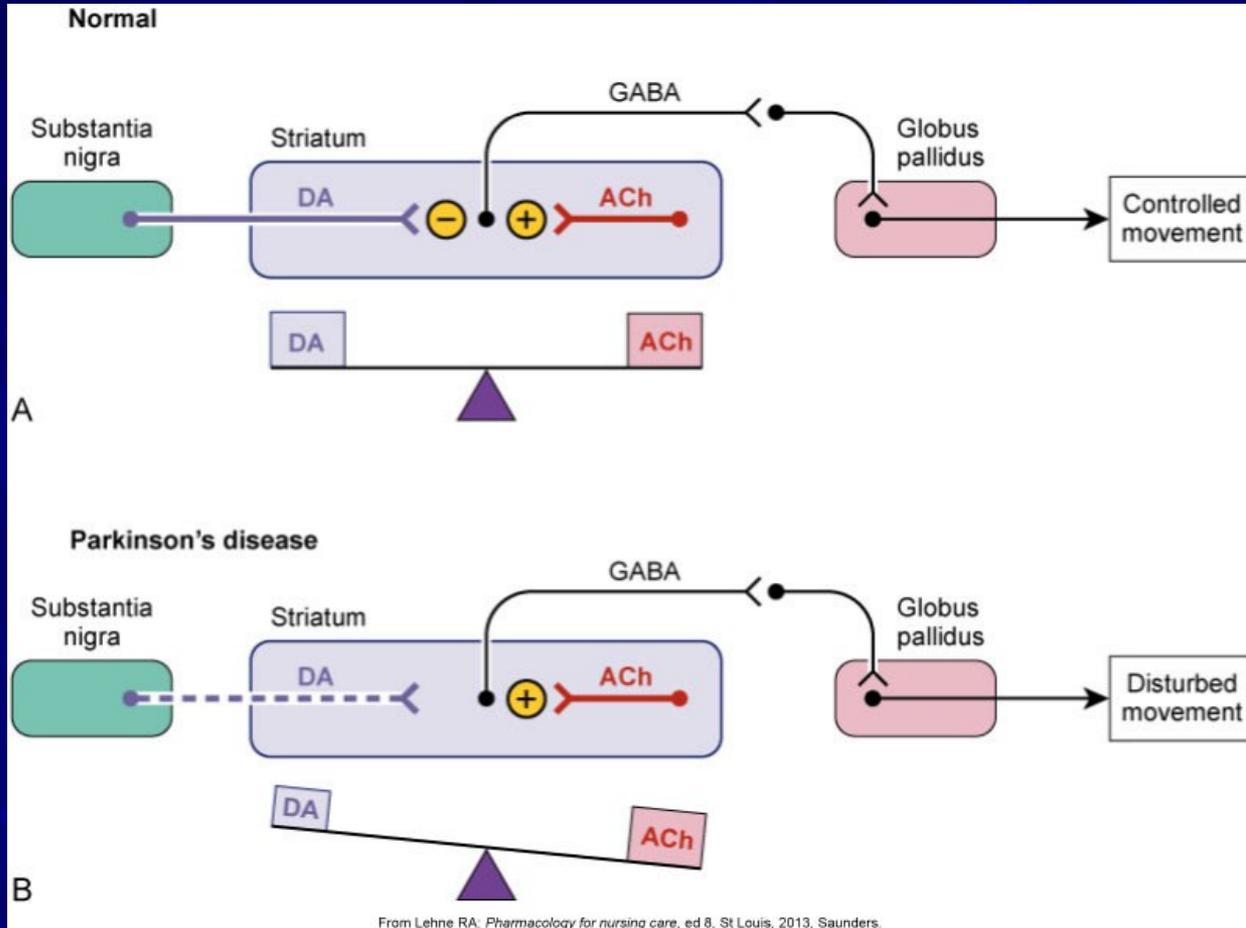
PD: Etiology & Pathophysiology

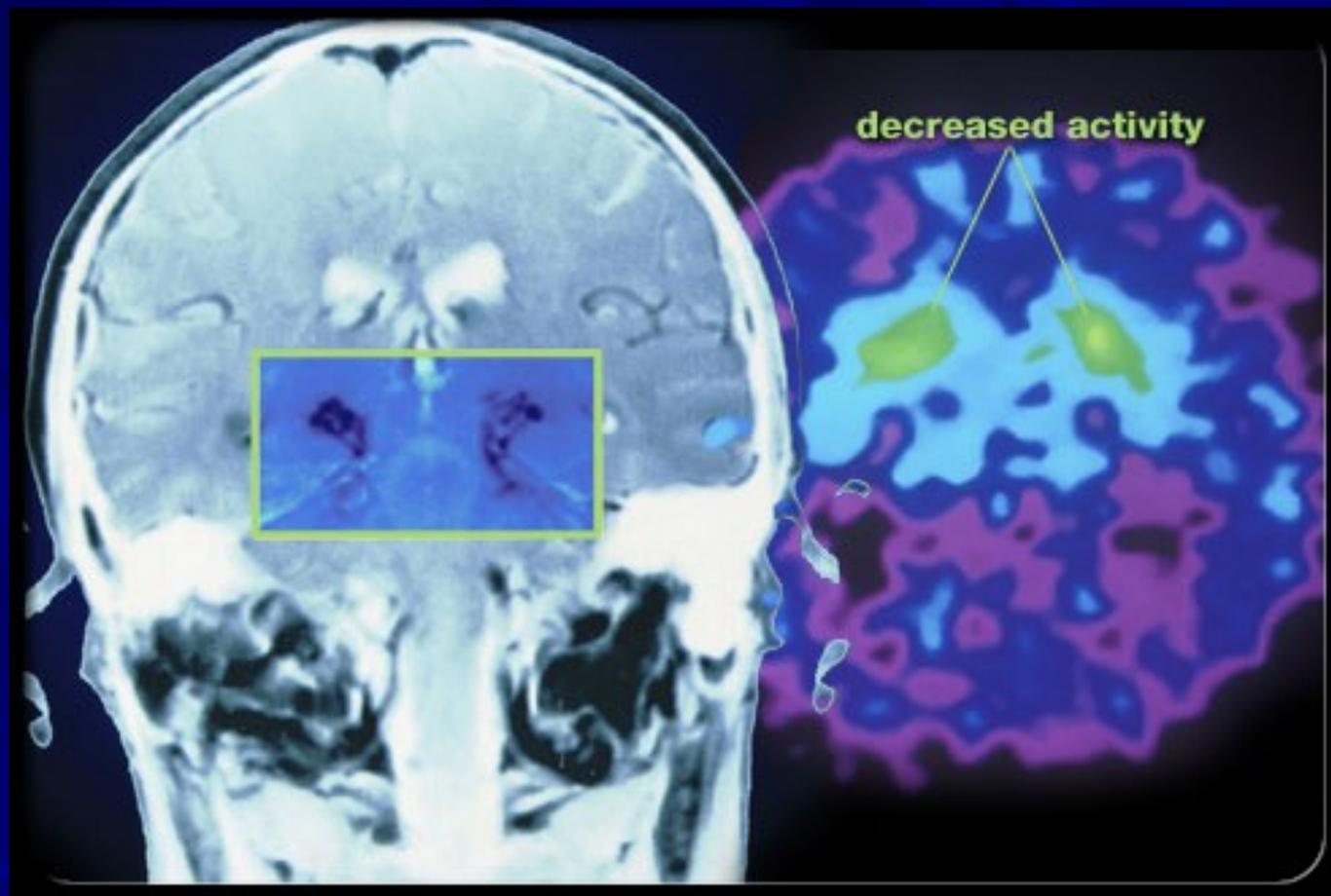
- Many forms of secondary/atypical parkinsonism exist
 - Exposure to chemicals
 - Drug-induced
 - Prescribed
 - Illicit
 - Others

PD: Pathophysiology

- Lack of dopamine (DA)
 - Degeneration of dopamine-producing neurons in substantia nigra of midbrain
 - Disrupts dopamine-acetylcholine balance in basal ganglia
 - Essential for normal functioning of extrapyramidal motor system
- Clinical symptoms appear with 60% neuron loss and 80% dopamine decrease

Dopamine Disorder in PD





PD: Clinical Manifestations

- Onset is gradual and insidious with ongoing progression
- **TRAP**
 - Tremor
 - Rigidity
 - Akinesia
 - Postural instability

PD: Clinical Manifestations

■ Beginning stages

- Mild tremor, slight limp, ↓ arm swing

■ Later stages

- Shuffling, propulsive gait with arms flexed, loss of postural reflexes

■ 90% experience hypokinetic dysarthria (speech abnormalities)

PD: Clinical Manifestations

■ Tremor

- Often first sign
- Initially minimal
- More prominent at rest
- Aggravated by
 - Emotional stress
 - ↑ Concentration

PD: Clinical Manifestations

- Pill rolling hand tremor
- Diaphragm, tongue, lips, jaw may be involved
- Essential tremor is not associated with PD
 - Occurs during voluntary movement, has more rapid frequency, is familial

PD: Clinical Manifestations

■ Rigidity

- ↑ Resistance to passive motion when limbs are moved through their ROM
- Cogwheel rigidity
 - Jerky quality
 - Like intermittent catches in passive movement of a joint
- Sustained muscle contraction
 - Complaints of soreness
 - Feeling tired and achy
 - Pain in the head, upper body, spine, or legs
- Slowness of movement

PD: Clinical Manifestations

■ Akinesia

- Absence or loss of control of voluntary muscle movements

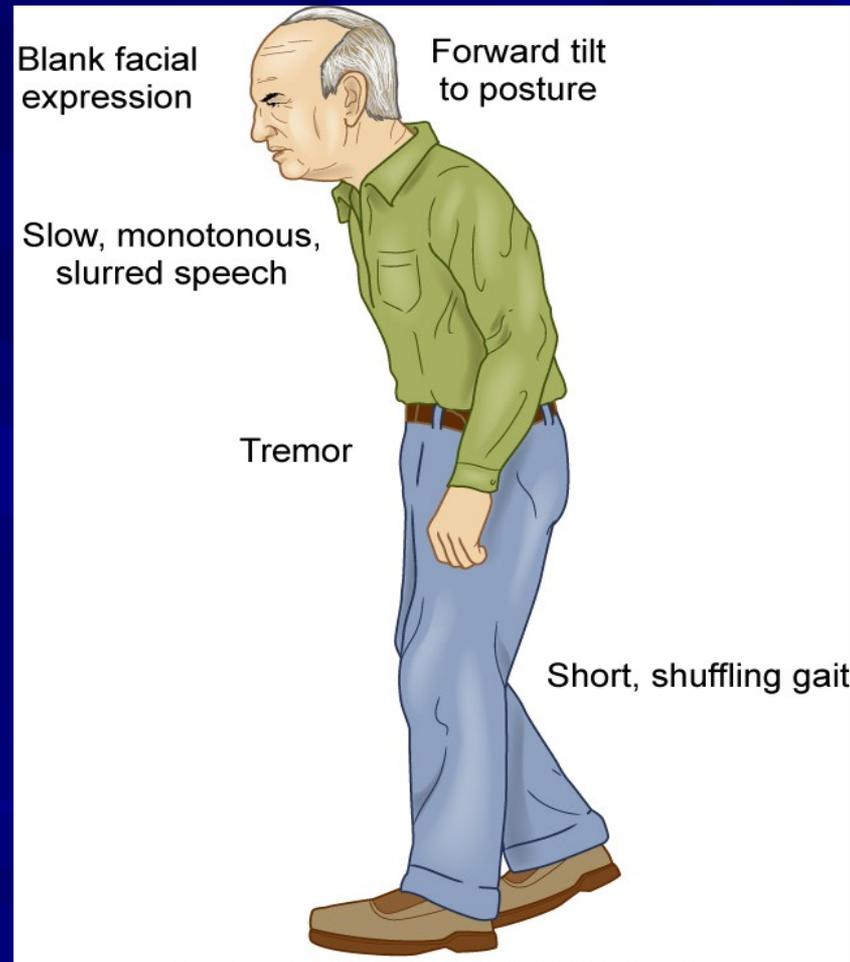
■ Bradykinesia

- Slowness of movement
- Particularly evident in the loss of automatic movements

PD: Clinical Manifestations

- Loss of automatic movements occur subconsciously and result in classic characteristics of a person with PD
 - Stooped posture
 - Masked face
 - Drooling
 - Festination (shuffling gait)

Appearance of Patient with PD



PD: Clinical Manifestations

■ Nonmotor symptoms

- Depression and anxiety
- Apathy
- Fatigue
- Pain
- Urinary retention and constipation
- Erectile dysfunction
- Memory changes

PD: Clinical Manifestations

■ Sleep problems are common

- Difficulty staying asleep
- Restless sleep
- Nightmares
- Drowsiness during the day
- REM behavior disorder
 - Violent dreams
 - Potentially dangerous motor activity during sleep

PD: Complications

- Complications ↑ as disease progresses
 - Motor symptoms
 - Weakness
 - Akinesia
 - Neurologic problems
 - Neuropsychiatric problems
- Dementia often results
 - Associated with ↑ mortality

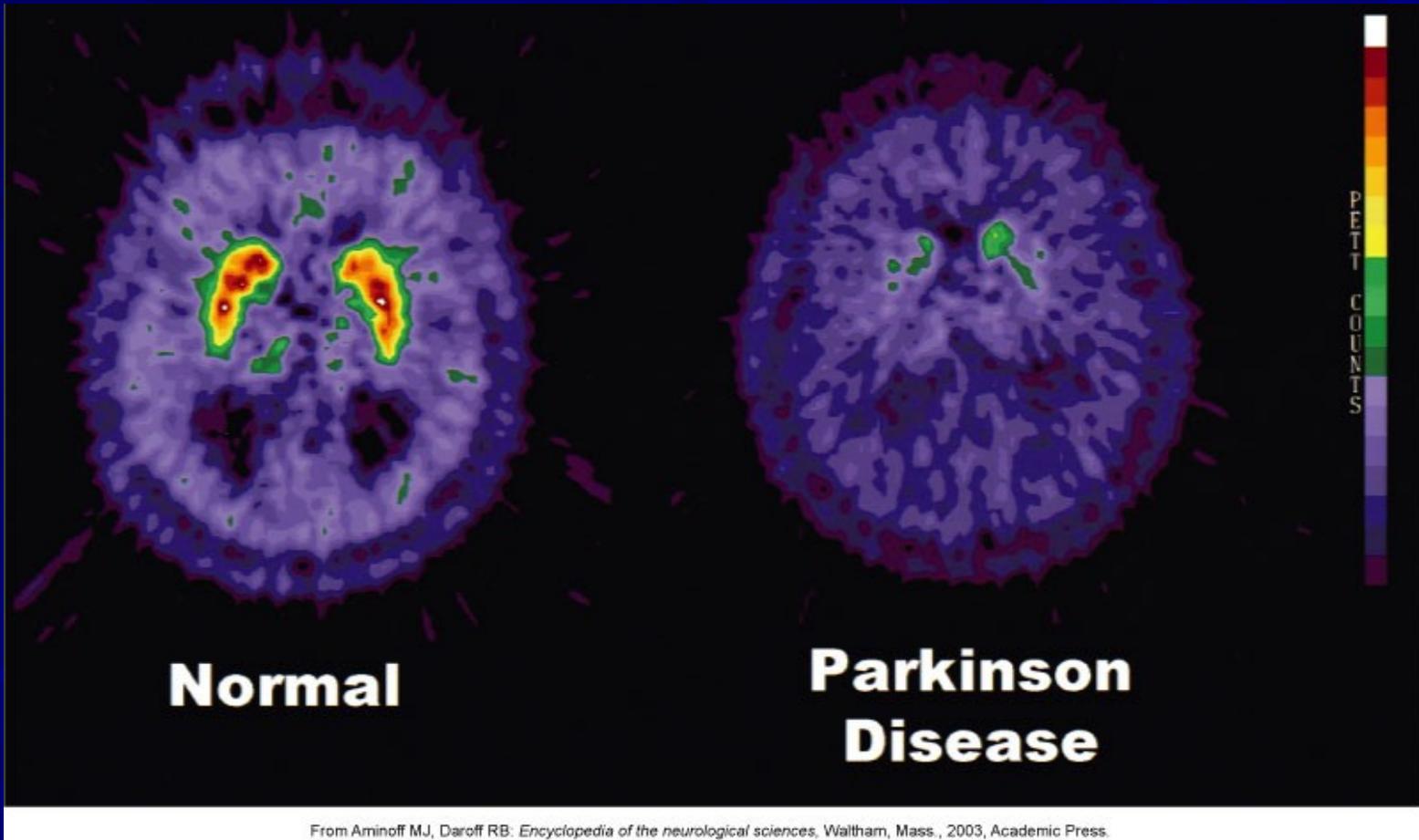
PD: Complications

- Dysphagia may result in malnutrition or aspiration
- General debilitation may lead
 - Pneumonia, UTIs, skin breakdown
- Orthostatic hypotension
 - ↑ risk for falls and injuries

PD: Diagnostic tests

- No definitive diagnostic procedures
- Patient history & clinical features
- Presence of 2 or more cardinal manifestations: TRAP
- Medical history, presenting symptoms, neurologic exam
- Positive response to antiparkinsonian drugs

PET Scan in PD



From Aminoff MJ, Daroff RB. *Encyclopedia of the neurological sciences*. Waltham, Mass., 2003, Academic Press.

Copyright © 2017, Elsevier Inc. All Rights Reserved.



www.alamy.com - FA43N6

PD: Drug Therapy

- Aimed at correcting imbalances of neurotransmitters within the CNS
- Antiparkinsonian drugs either
 - Enhance or release supply of dopamine
 - Antagonize or block the effects of overactive cholinergic neurons in the striatum

PD: Drug Therapy

■ Therapeutic goals

- Ideal treatment that reverses neuronal degeneration or prevents further degeneration does not exist
- Improve the patient's ability to carry out the activities of daily life
- Drug selection and dosages are determined by the extent to which PD interferes with work, dressing, eating, bathing, and other ADLs

PD: Drug Therapy

- Two Major categories
 - Dopaminergic agents
 - Most commonly used drugs for PD
 - Promote activation of dopamine receptors
 - Levodopa
 - Anticholinergic agents
 - Prevent activation of cholinergic receptors
 - Benztropine (Cogentin)

Levodopa

- Highly effective, but benefits diminish over time
- Orally administered; rapidly absorbed from small intestine
 - Food delays absorption
 - Neutral amino acids compete with levodopa for intestinal absorption & for transport across blood-brain barrier
 - High-protein foods reduce therapeutic effects

PD: Drug Therapy

- Levodopa/carbidopa (Sinemet)
- **MOA:** Levodopa is converted to dopamine in the CNS. Carbidopa prevents peripheral destruction of levodopa
- **Adverse Effects:** N/V, dyskinesias, postural hypotension, dysrhythmias, psychosis, impulse control, darkened sweat & urine, loss of effect

PD: Drug Therapy

■ Dopamine Agonists

- First drug of choice for mild or moderate symptoms
- Direct activation of dopamine receptors
 - Less effective than levodopa
 - Not dependent on enzymatic conversion to be active
 - Does not compete with dietary proteins
 - Lower incidence of response failure
 - Less likely to cause dyskinesias

Dopamine Agonists

■ Pramipexole (Mirapex)

- Used alone in early PD and with levodopa in advance PD
- Maximal benefits take several weeks
- **Adverse effects**
 - Monotherapy: SLEEP ATTACKS, nausea, dizziness, daytime somnolence, insomnia, constipation, weakness, hallucinations
 - Combined: orthostatic hypotension, dyskinesia, increased hallucinations
 - Rare instances of pathologic gambling & other compulsive self-rewarding behaviors

Anticholinergic

- Trihexiphenidyl & Benztropine
- **MOA:** blocks muscarinic receptors in the striatum. ↓ Activity of ACh
- **Adverse effects:** dry mouth, urinary retention, tachycardia, blurred vision, constipation, photophobia, confusion, hallucinations

COMT Inhibitors

- Inhibit metabolism of levodopa in the periphery
- No direct therapeutic effects of their own
- Two COMT inhibitors available
 - Entacapone (Comtan)
 - Tolcapone (Tasmar)

Entacapone (Comtan)

- Indicated for use with levodopa
- **MOA:** inhibit breakdown of levodopa in the periphery
- **Adverse Effects:** dyskinesia, orthostatic hypotension, N/V/D, hallucinations, sleep disturbances, impulse control disorders, & yellow-orange discoloration of urine

Tolcapone (Tasmar)

- Used only in conjunction with levodopa
- Only if safer agents are ineffective or inappropriate
- **MOA:** inhibits metabolism of levodopa in the periphery
- **Adverse effects:** Liver failure, dyskinesia, orthostatic hypotension, nausea, hallucinations, sleep disturbances, & yellow-orange urine

MAO-B Inhibitors

- Considered first-line drugs for PD
- Benefits are modest
- Combination with levodopa can reduce the wearing-off effect
- Two MAO-B Inhibitors
 - Selegiline
 - Rasagiline

Selegiline (Eldepryl)

- Monotherapy or used with levodopa
- **MOA:** Inhibits breakdown of dopamine
- **Adverse Effects:** insomnia, dry mouth, orthostatic hypotension, dizziness, hypertensive crisis, & GI symptoms

Rasagiline (Azilect)

- Approved for initial therapy & for combined use with levodopa
- Benefits derived from preserving dopamine in the brain
- **Adverse Effects:** insomnia, orthostatic hypotension, irritation of buccal mucosa, hypertensive crisis

PD: Drug Therapy

- Other agents used to manage PD:
 - Antihistamines with anticholinergic or β -adrenergic blockers are used to manage tremors.
 - Antiviral agent amantadine
 - Apomorphine for hypomobility

PD: Surgical Management

■ Surgical Therapy

- Used in patients unresponsive to drug therapy
- Have developed severe motor complications

■ Deep Brain Stimulation (DBS)

■ Ablation (Destruction)

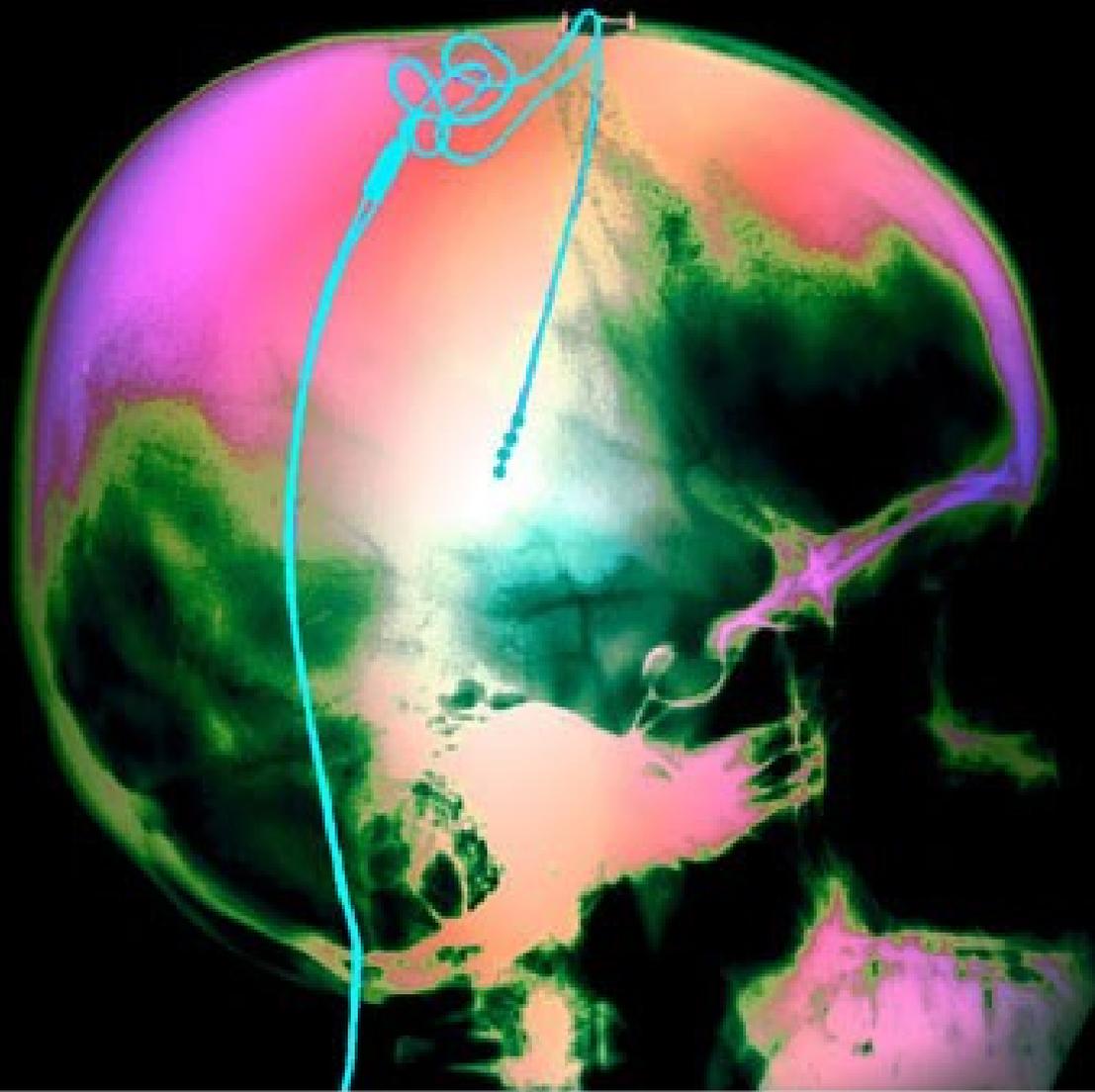
- Thalamotomy
- Pallidotomy
- Subthalamic nucleotomy

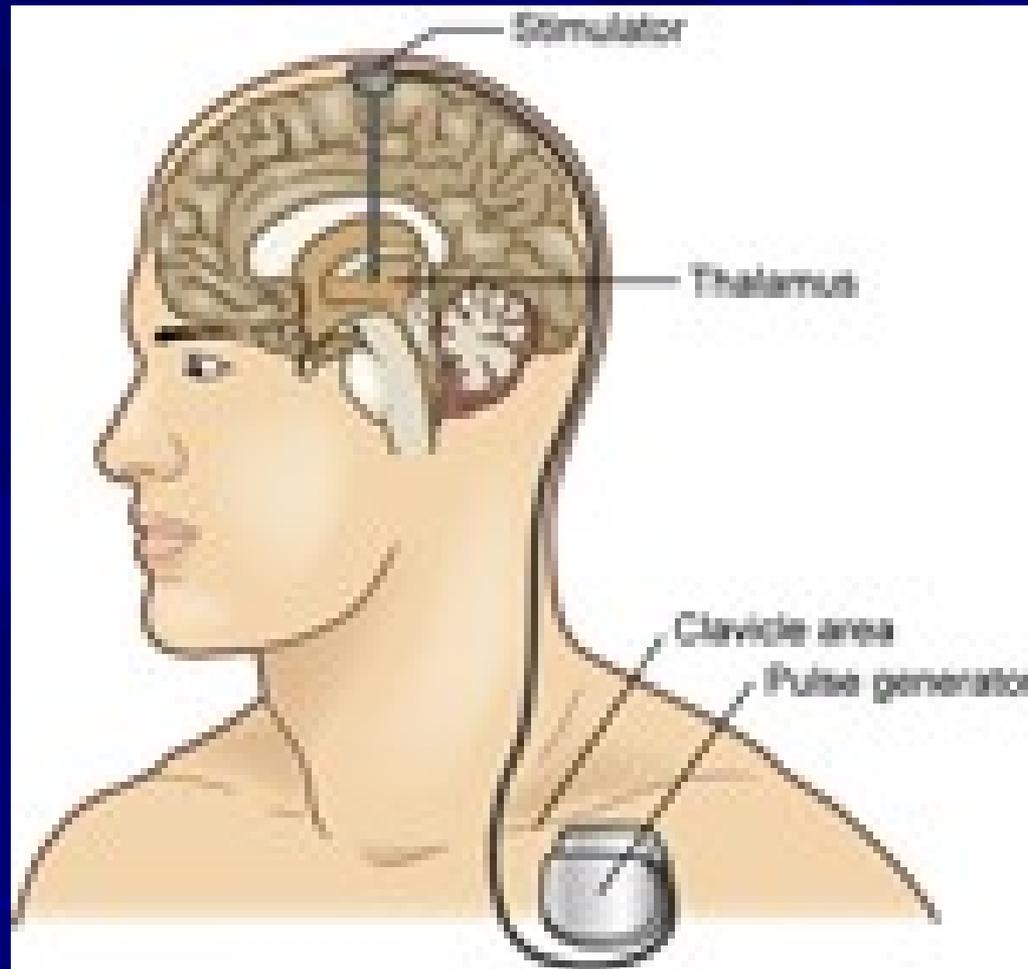
■ Transplantation

■ Pallidotomy

Deep Brain Stimulation

- Most common surgical treatment
- Reversible and programmable
- ↓ Increased neuronal activity produced by DA depletion
 - Improves motor function
 - Reduces dyskinesia and medications





Ablation Surgery

- Locate, target, destroy area of brain affected by PD
- Destroys tissue that produces abnormal chemical or electrical impulses leading to tremors or other symptoms

Transplantation

- Transplantation of fetal neural tissue into the basal ganglia
 - Provides DA-producing cells in the brain of patients
 - Research and clinical trials ongoing

Interprofessional Care

■ Nutritional Therapy

- Malnutrition and constipation can be serious consequences
- Patients with dysphagia and bradykinesia need food that is easily chewed & swallowed
- Adequate fiber
- Eating more numerous small meals
- Provide ample time

PD: Nursing Diagnoses

- Self-care deficit
- Chronic confusion
- Impaired physical mobility
- Impaired verbal communication
- Impaired swallowing
- Risk for imbalanced nutrition: Less than body requirements

PD: Nursing Management

- Maximize neurologic function.
- Maintain independence in activities of daily living (ADLs) for as long as possible.
- Optimize psychosocial well-being.
- Administer medications as prescribed
- Facilitate nutritional intake
- Interdisciplinary collaboration: PT, OT, Speech



www.alamy.com - D3BE5R



