
بسم الله الرحمن الرحیم

parkinsonism

ارائه توسط دکترمهدی شفیعی ثابت
عضو هیات علمی گروه پزشکی خانواده
متخصص نورولوژی

Clinical Dx

- History ----> understanding the nature of the patient's problem
- Ph/E --------> Etiologic hypothesis
- Para clinic --> Confirm etiology ----> treatment --------> Increased quality of life and health

Clinical DX in neurologic problems

History ------> understanding the nature of the patient's problem and **localization**

Neurologic exam ------> Confirm **localization**

Ph/E ------> Etiologic hypothesis

Paraclinic --------> confirm etiology

Physical examination

- Looking
- Touching
- Percussion
- Auscultation

Neurological Examination

- Mental Status and language
- Cranial nerves
- Motor system
- Sensory system
- Stance and gait

Motor system Examination

- Looking: abnormal movements(location, rhythm, frequency, amplitude, association with activity and posture and emotion)
- Touching: muscle bulk, muscle tonus, strength, coordination
- Percussion: fasciculation, reflexes
- Stance: posture, abnormal movements, Romberg test, pull-test
- Gait: rhythm, speed, coordination, arm swing, abnormal movements, tandem gait

Localization of Motor system dysfunction

- Upper motor neuron lesions
- Movement disorders (Extrapyramidal disorders)
- Ataxia (disequilibrium)
- Lower motor lesions

CNS lesions

- Cortex
- White matter
- Diencephalon
- Basal ganglia
- Cerebellum
- Spinal cord

Lower motor lesions

- Anterior horn cell
- Radicules
- Plexus
- Neurons
- Neuromuscular junctions
- muscles

Etiologic Dx

- Cerebrovascular diseases (ischemic or hemorrhagic, cerebrovascular thrombosis)
- Disturbances of CSF and its circulation (hydrocephalus, NPH, low-pressure syndrom)
- Neoplastic disorders (primary tumors or metastatic, paraneoplastic disorders)
- Infectious diseases (bacterial, viral, fungal, parasitic, prion)
- Inflammatory-immune diseases (demyelinative diseases, vasculitis)

- Toxic disorders (due to toxins, drugs and other chemical agents)
- Acquired metabolic disorders and nutritional deficiency
- Traumatic insults
- Congenital-Genetic diseases of nervous system (Developmental , Inherited metabolic diseases)
- Degenerative disorders (dementia, movement disorders)
- Functional disorers

Parkinsonism

- 1) Tremor at rest
- 2) Brady kinesia _ Hypo kinesia
- 3) Rigidity
- 4) Flexed posture
- 5) Loss of postural reflexes
- 6) Freezing

Definite Dx is clinical: At least two of six which should include at least one of "1 & 2"

Rest Tremor

- Frequency of 4 to 5 Hz
- Is present in: the limbs, lips, chin and tongue
- In the limbs: almost always distally; classic “pill-rolling”, disappears with action but reemerges as the limbs maintain a posture, in hands increases with walking (may be an early sign) , worsens with stress.

Bradykinesia

- The most common feature of parkinsonism
- Slowness of movement, difficulty initiating movement, and loss of automatic movement
- Can affect;

Face: masked facies or hypomimia, decreased frequency of blinking

Speech: hypophonia, monotonous tone of voice, aprosody(lack of inflection), dysarthria, tachyphemia(running the words together)

Swallow: drooling saliva

Hands: difficulty in shaving, brushing, combing hair, buttoning, micrographia(small and slow handwriting)

Trunk: difficulty rising from a chair

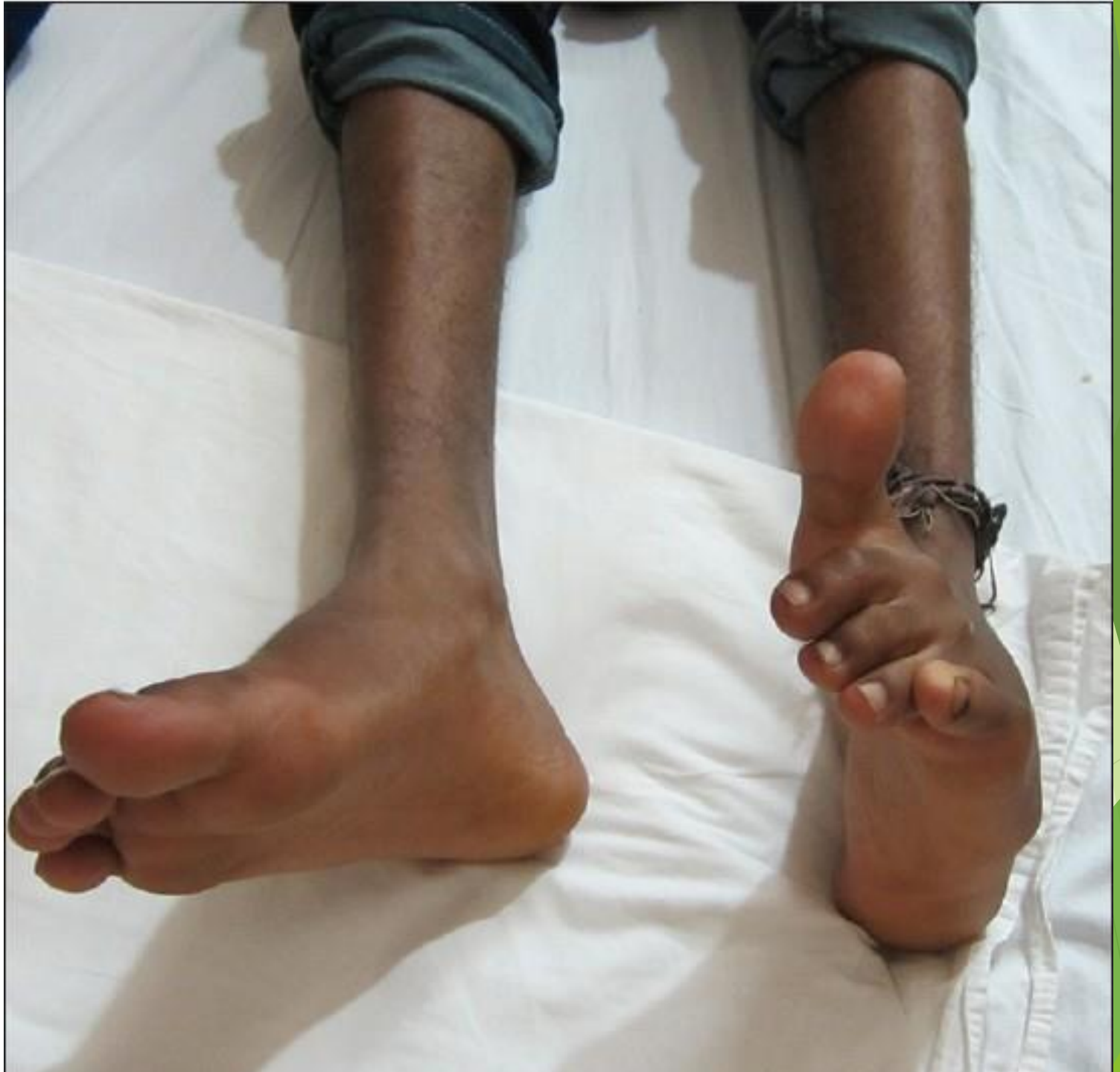
Gait: slow with a shortened stride lengh and a tendency to shuffle, decreased arm swing.

Rigidity

- An increased muscle tone in passive movement of patient's limbs, neck or trunk.
- In all direction
- Cogwheel
- Rigidity in limb increases when another limb is engaged in voluntary active movement.

Flexed posture

- Commonly begins in the arms, spreads to involve the entire body.
- Head is bowed, the trunk is bent forward, the back is kyphotic, the are are held in front, the elbows, hips, and knees are flexed
- Deformities of the hands include ulnar deviation of the hands, flextion of the metacarpophalangeal joints and extentention of the interphalangeal joints.
- Deformities of feet include inversion and dorsiflexed big toes (striatal toe).



Loss of postural reflexes

- Stance: sitting en bock, inability to stand unassisted
- Gait: festination, leads to falling

Freezing (motor block)

Suddenly transient inability to perform active movements, lasting usually no more than several seconds and includes:

Walking: occurs when patient begins to walk(start hesitation), attempts to turn while walking, approaches a destination(destination hesitation) and inability to deal with percieved barriers or time-restricted activities.

Eyelid: apraxia of lid openning or levator inhibition

Speaking: palilalia

writing

Biochemical pathology

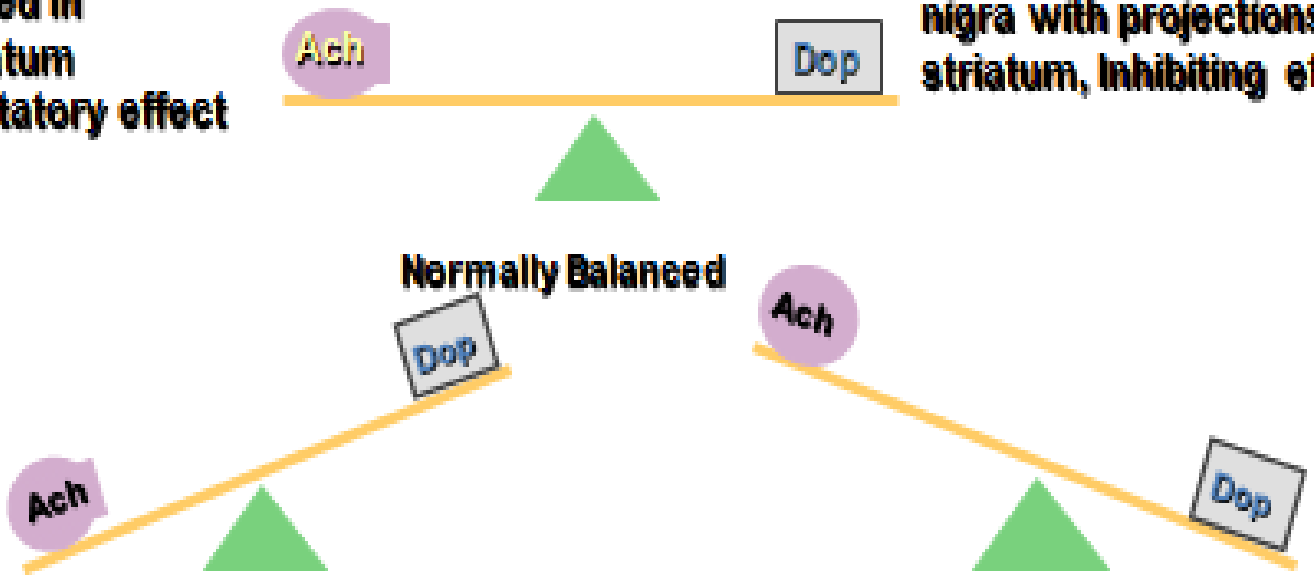
Core biochemical pathology is decreased dopaminergic neurotransmission in the basal nuclei ------> disinhibition of subthalamic nucleus and medial Globus pallidus-----> decreased VA/VL drive to cortex ---->less motor activity;

- 1] Degeneration of nigrostriatal dopamin system
- 2] Degeneration of the striatum with loss of dopamine receptor
- 3] Drug induced: a)blockade of dopamine receptor or , b)depletion of dopamine storage.

Balancing Acts.....

Acetylcholine:
Synthesized and stored in striatum
Excitatory effect

Dopamine:
Synthesized by sustantia nigra with projections in striatum, Inhibiting effect



Ach > Dop = Parkinson's

(Block Ach with anticholinergic drugs or Increase Dopamine)

Ach < Dop = Chorea

(Reduce dopamine or block dopamine receptors)

Classification of major parkinsonian syndromes

- Primary parkinsonism:

parkinson disease-sporadic and familial

- Secondary Parkinsonism:

Vascular;multi-infarct state, Hypoxia, Hemiatrophy-hemiparkinsonism, Hydrocephaus;NPH, Tumor, Infectious;postencephalitic, Toxin:Mn,CO,MPTP,cyanide, Metabolic;parathyroid dysfunction, Trauma

- **Parkinson-plus syndromes:**

Cortical-basal ganglionic degeneration, Dementia syndromes (AD, DLB dis, FTD), MSA syndromes (Striatonigral degeneration, Shy-drager syndrome, Sporadic olivopontocerebellar degeneration, motor neuron disease-parkinsonism), Progressive pallidal atrophy, PSP, Lytico-Bodig (Guamanian parkinsonism-dementia-ALS)

- **Heredodegenerative disease:**

Hallervorden-Spatz disease, Huntington disease, Lubag (X-linked dystonia-parkinsonism), Mitochondrial cytopathies with striatal necrosis, Neuroacanthocytosis, Wilson diseases

History of Parkinson´s Disease

- First described in 1817 by an English physician, James Parkinson, in "An Essay on the Shaking Palsy."
- The famous French neurologist, Charcot, further described the syndrome in the late 1800s.

James Parkinson, 1817

AN
ESSAY
ON THE
SHAKING PALSY.

CHAPTER I.

Dr. James Parkinson
1755-1824

Famous Faces of Parkinson

Michael J. Fox

Muhammad Ali

Katharine Hepburn

Pope John Paul II

Mao Tse Tung

Stages of PD

- Initially, symptoms are typically mild and on one side of the body
- (Hemi parkinsonism).
- Tremors are usually the first symptom to appear, but not all patients develop a tremor.
- Over time, the tremor usually becomes worse and patients develop bradykinesia (slowness of movement) and akinesia (lack of movement), rigidity, and difficulty with balance.

Stages of PD

- Symptoms eventually spread to the other side of the body, affecting both the UE and LE.
- Other symptoms can develop such as shuffling gait, freezing episodes, "poker face" (lack of facial expression), drooling, and muffled speech.
- Later stages may include a severe loss of mobility and independence.
- This can lead to the most serious risk of PD, bronchopneumonia, which is the leading cause of death.

Clinical features of PD

- Resting tremor: Most common first symptom, usually asymmetric and most evident in one hand with the arm at rest.

- Bradykinesia: Difficulty with daily activities such as writing, shaving, using a knife and fork, and opening buttons; decreased blinking, masked facies, slowed chewing and swallowing.

- Rigidity: Muscle tone increased in both flexor and extensor muscles providing a constant resistance to passive movements of the joints; stooped posture, anteroflexed head, and flexed knees and elbows.

Additional clinical features of PD

- Postural instability: Due to loss of postural reflexes.
- Dysfunction of the autonomic nervous system:
  . Impaired gastrointestinal motility
  . Bladder dysfunction
  . Sialorrhea
  . Excessive head and neck sweating
  . Orthostatic hypotension.
- Cognitive impairment
- Psychiatric Symptoms

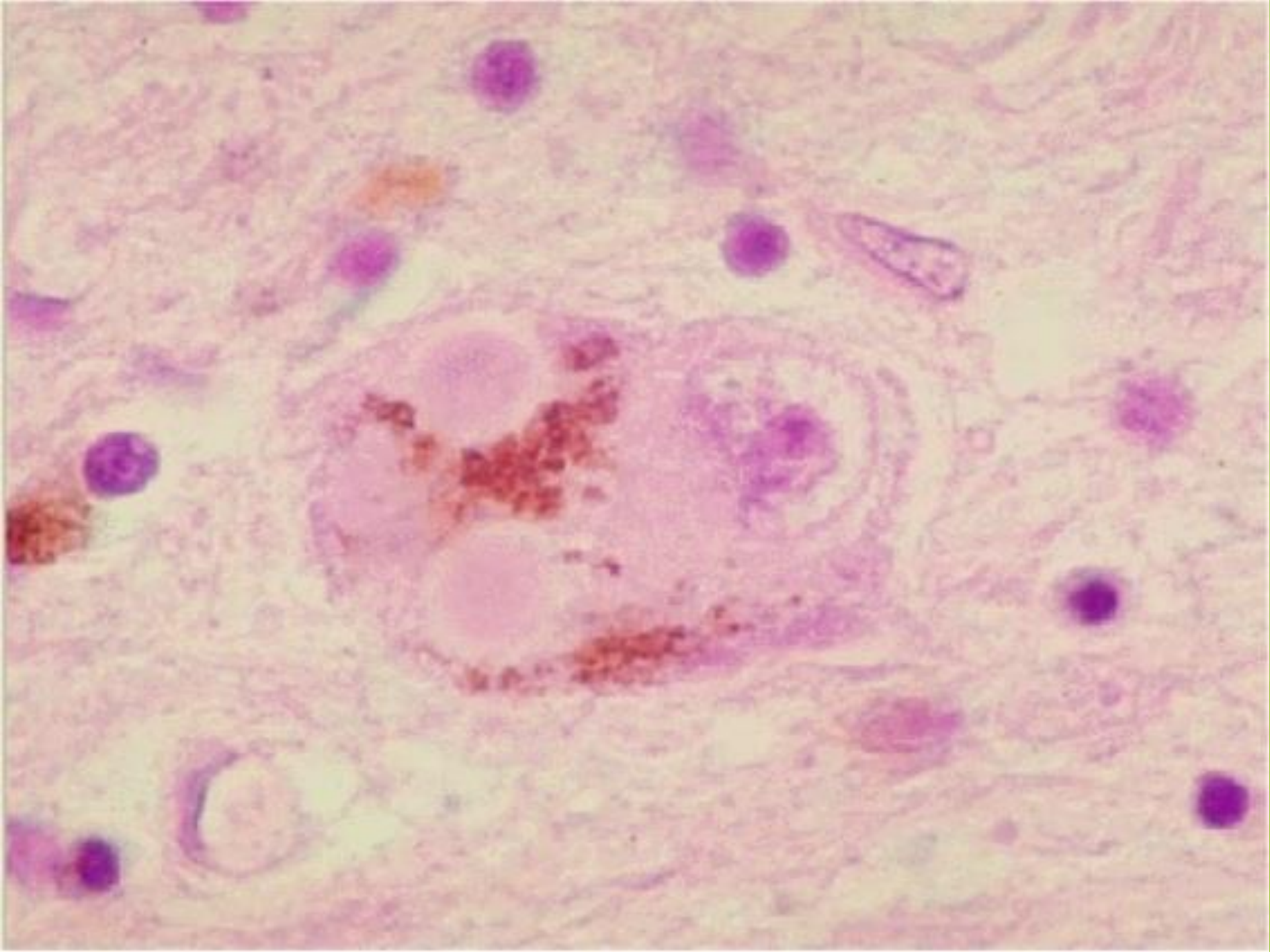
Neuropathology

- Degeneration of the neuromelanin containing neurons in the brain stem specially in yhe substantia nigra pars compacta and locus ceruleus
- Many of surviving neurons contains eosinophilic cytoplasmic proteinacious inclusions known as Lewy bodies the pathlogic hallmark of the disease.
- Lewy body First described in 1912 by a German neuropathologist, Friedrich Lewy.

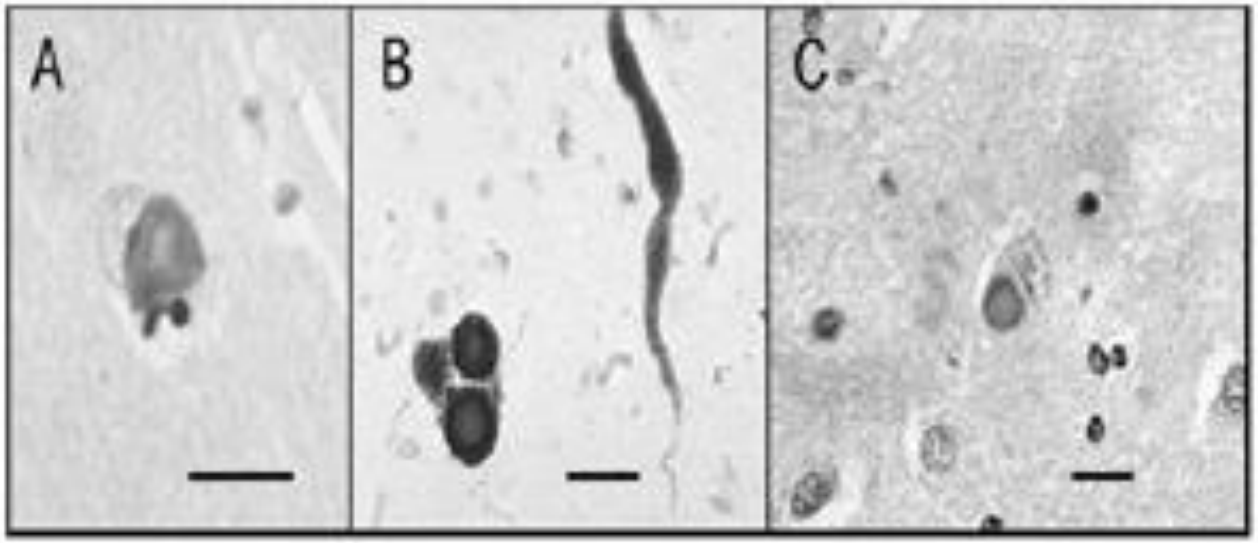
Neuropathology of PD:

Where are Lewy Bodies?

- Not limited to Substantia Nigra also in found in:
- Locus coeruleus
- Motor nucleus of the Vagus nerve
- Hypothalamus
- Nucleus basalis of Meynert
- Cerebral cortex,
- Olfactory bulb
- Autonomic nervous system.



Lewy Body



- **Confined largely to neurons; glial cells only rarely affected.**

F.H. Lewy (1885-1950)

Description des corps de Lewy : F.H. Lewy 1912

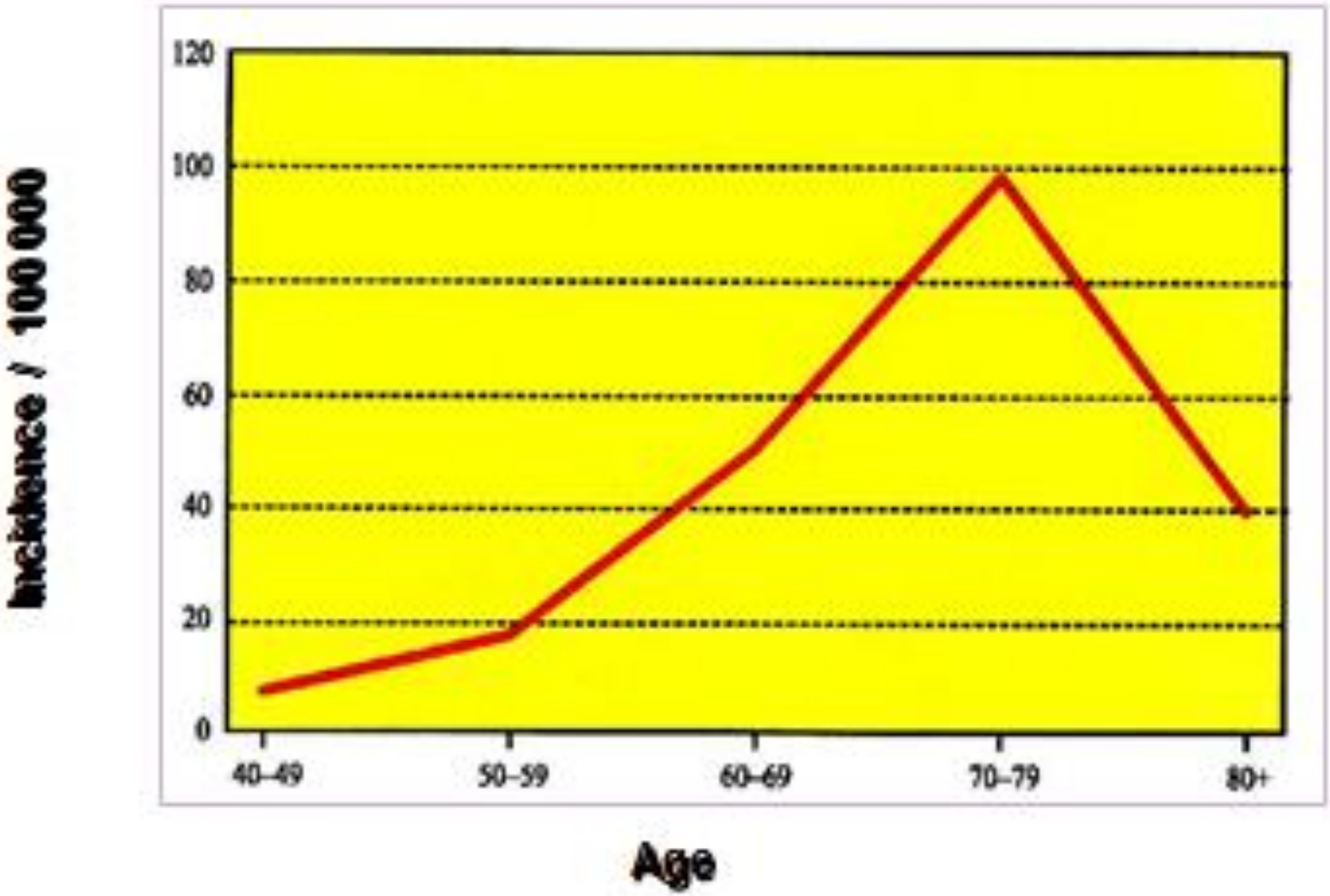
Epidemiology

- Prevalence rates in men are slightly higher than in women; reason unknown, though a role for estrogen has been debated.

- May be less prevalent in China and other Asian countries, and in African-Americans.

- Average age of onset is 60 y/o.

Incidence of PD



- Juvenile parkinsonism: onset at younger than 20 years; primary(usually familial) or heredodegenerative diseases(such as Huntington disease and wilson disease)
- Young onset PD: onset of primary parkinsonism between 20 and 40 years