

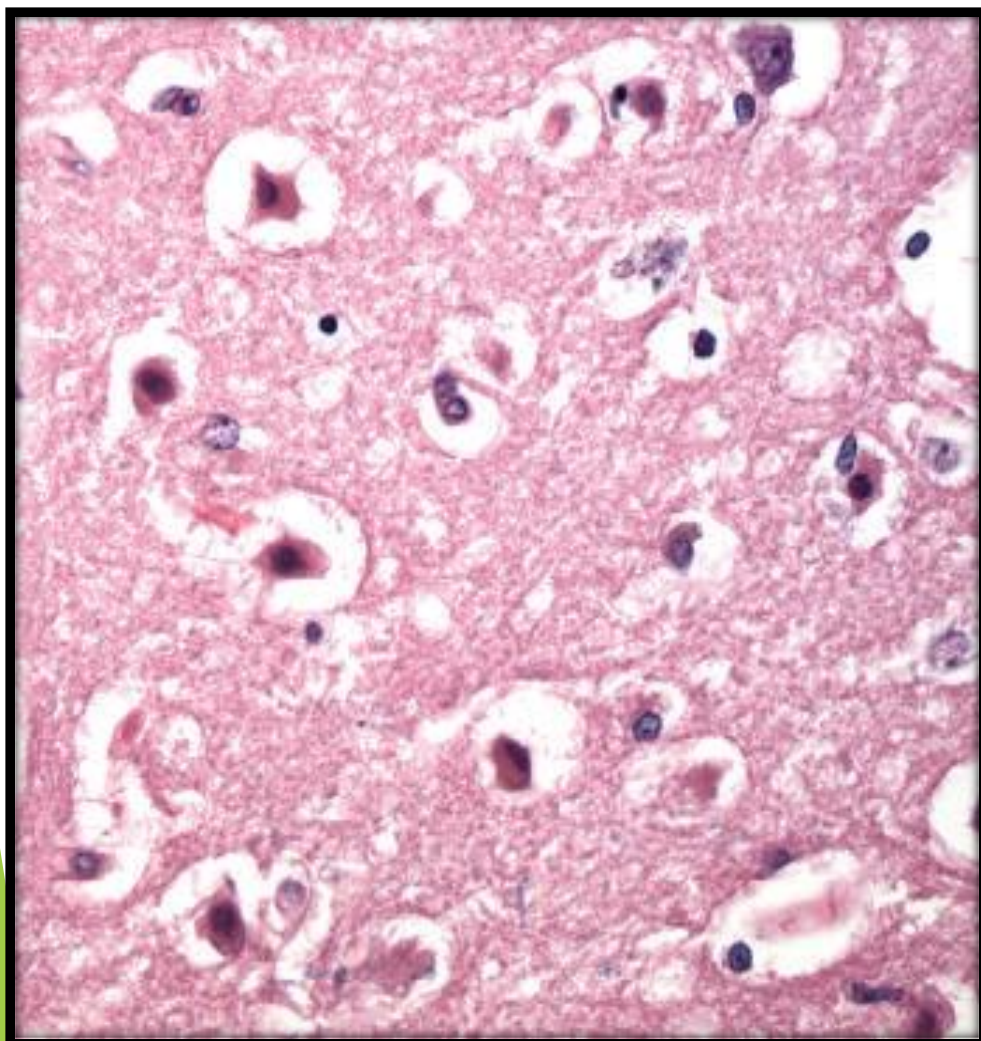
Neuroscience Pathology

Sura Al Rawabdeh MD

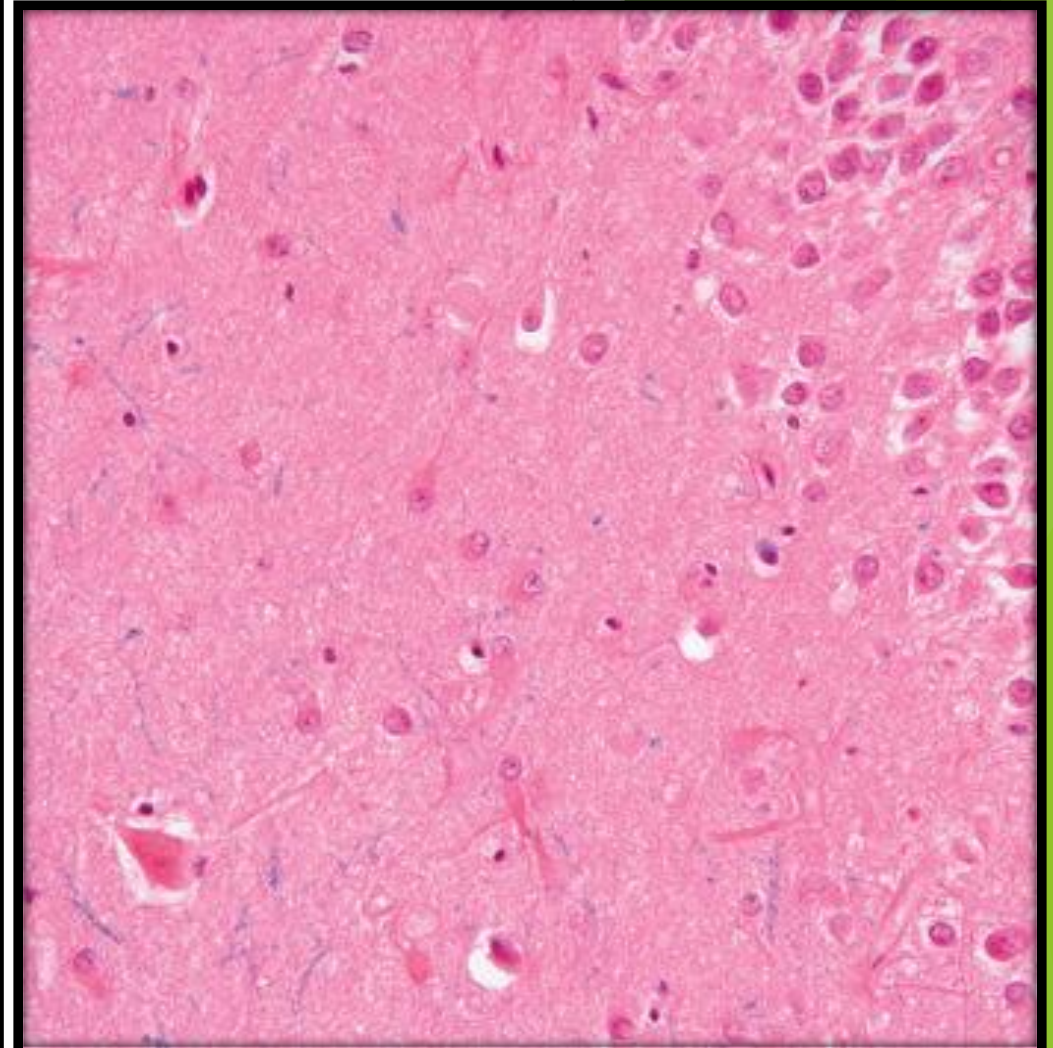
March 1 2022

Acute neuronal injury

Red neurone: Shrinkage of cytoplasm,
Pyknotic nuclei,
Disappearance of the nucleoli

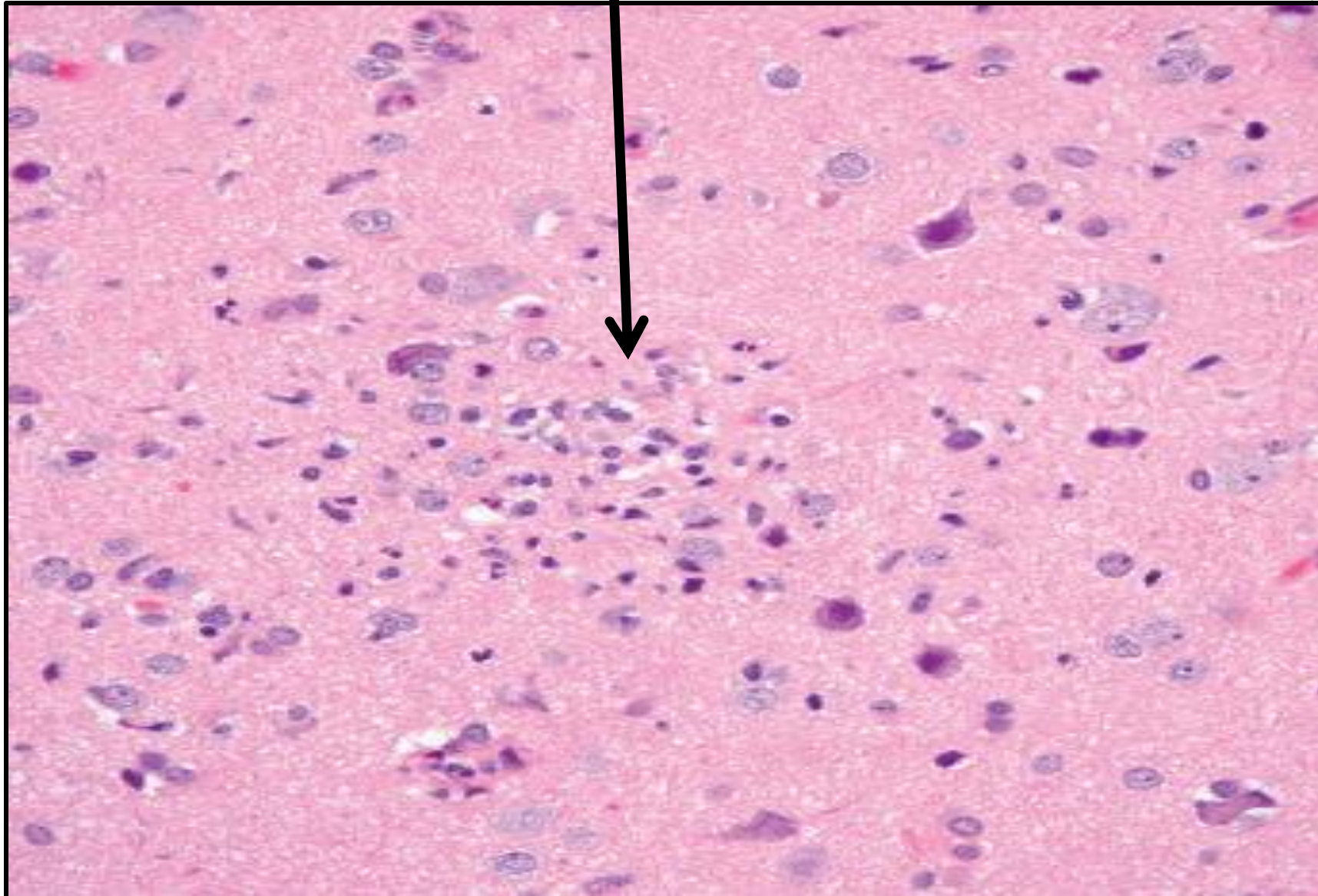


Red neurons; result from loss of
Nissl substance



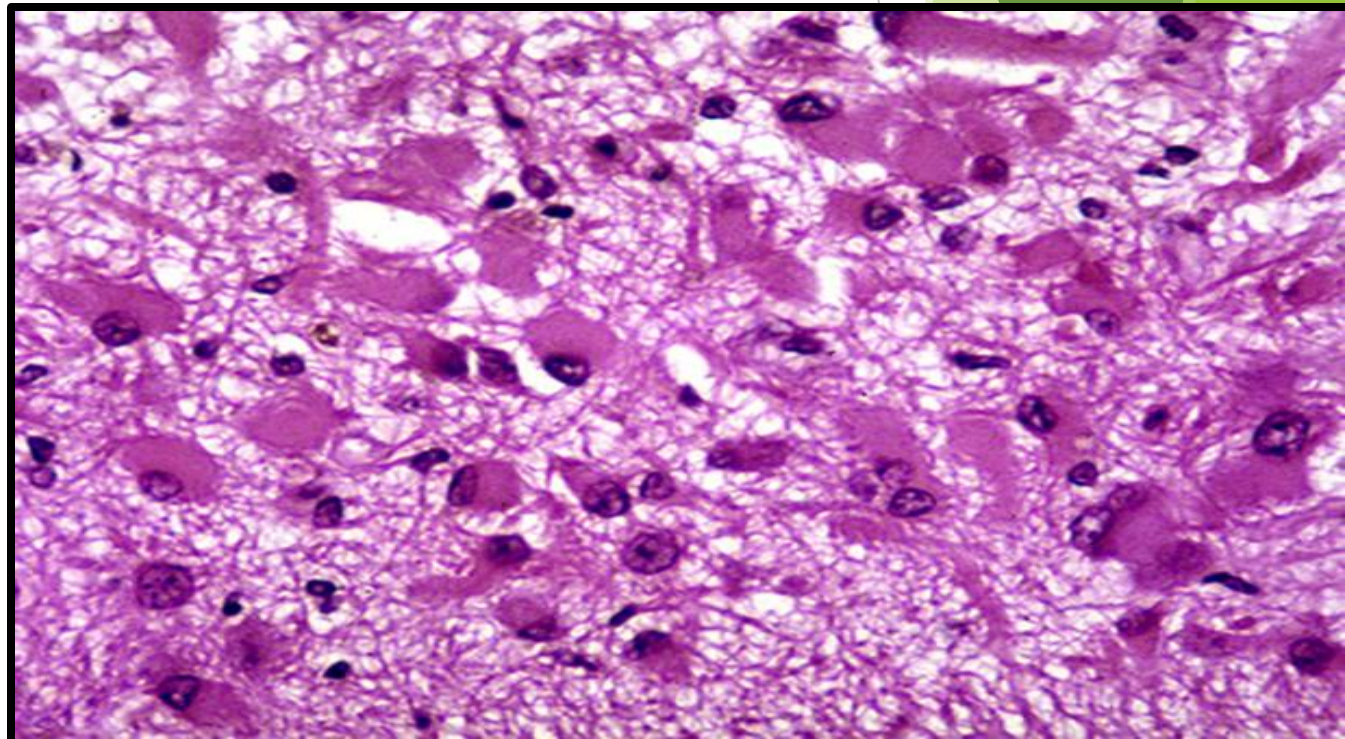
Acute neuronal injury

Area of gliosis



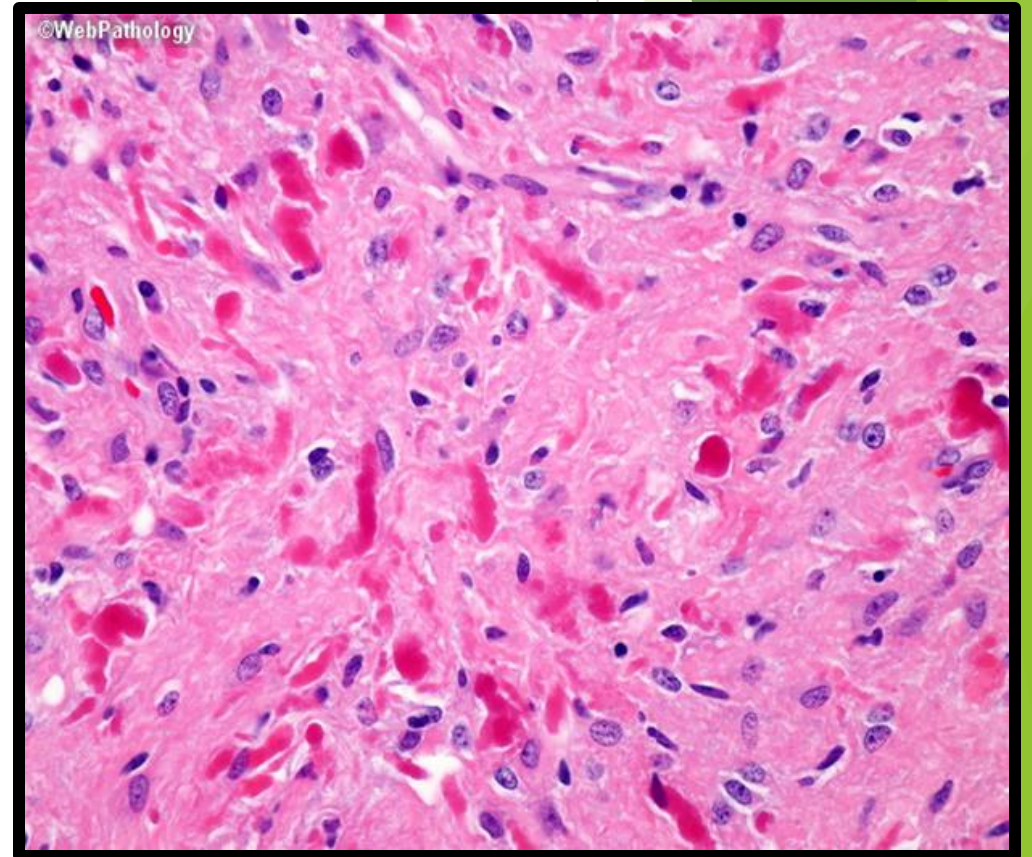
Astrocyte Injury and Repair

- ▶ The cytoplasm expands and takes on a bright pink hue, and the cell extends multiple stout, prominent nuclei, ramifying processes (called gemistocytic astrocyte),
- ▶ injured astrocytes become resembling ganglion cells of PNS



Astrocyte Injury and Repair

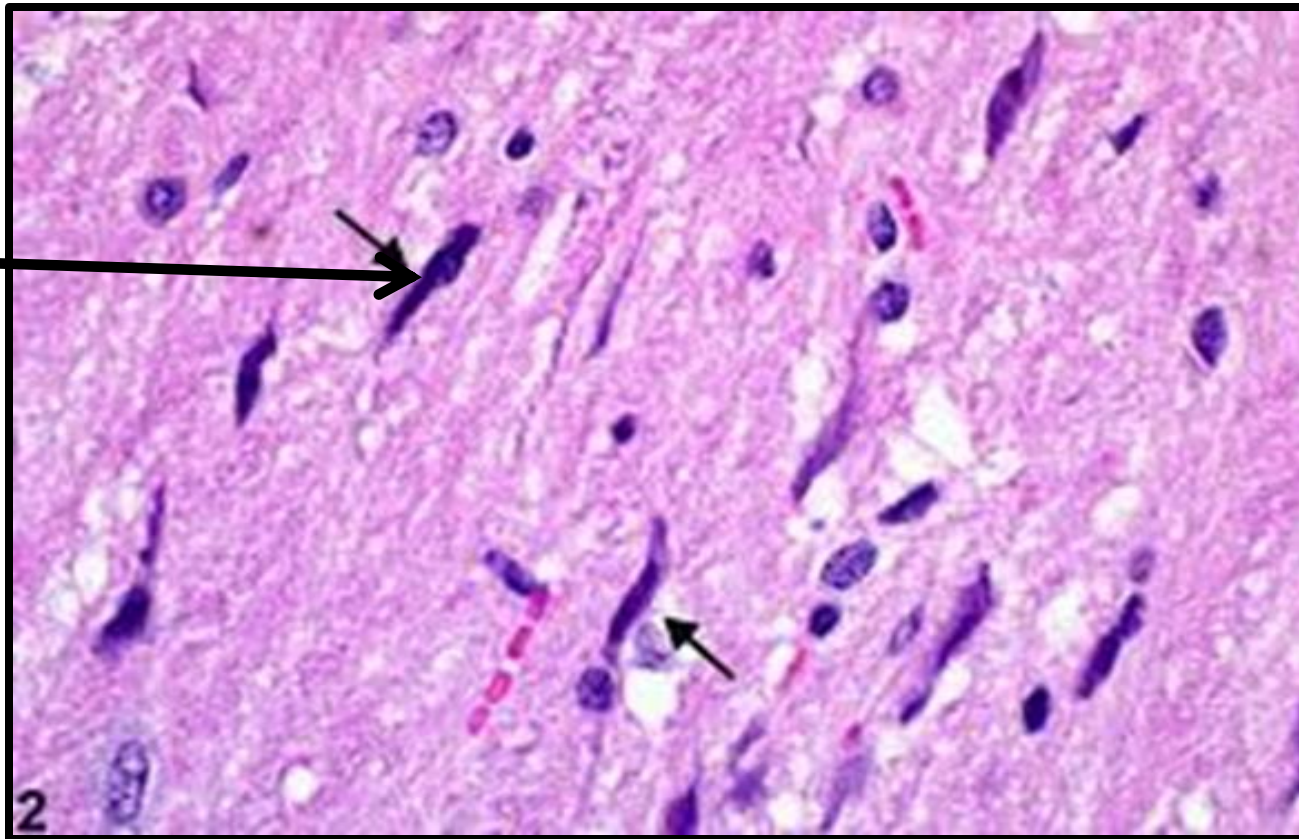
- ▶ Rosenthal fibers are thick, elongated, brightly eosinophilic protein aggregates found in astrocytic processes in chronic gliosis and in some (low-grade gliomas e.g → pilocytic astrocytoma).



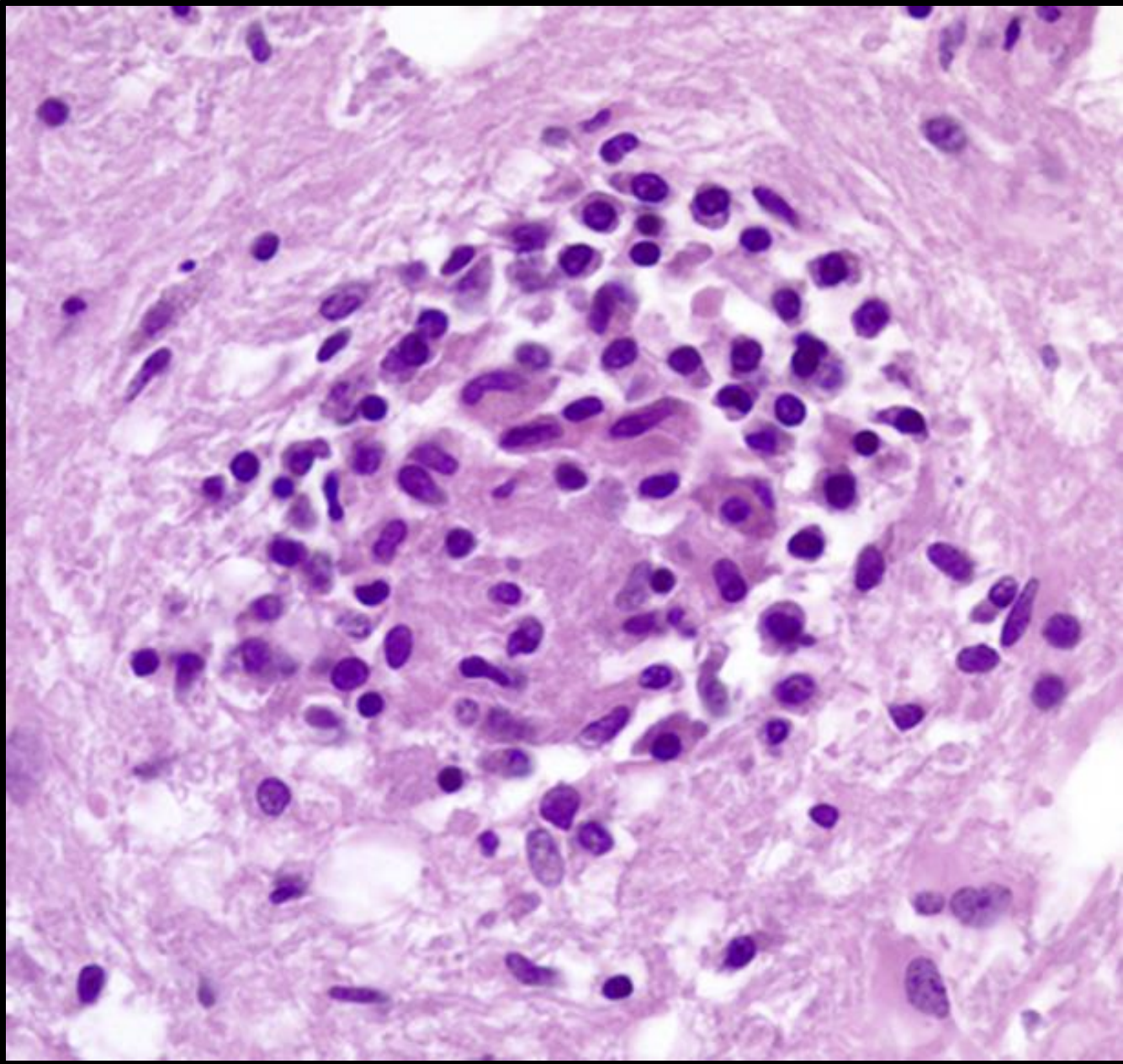
Astrocyte Injury and Repair

- ▶ Neuronophagia, which means when microglia engulf neurons.

Elongated
macrophages



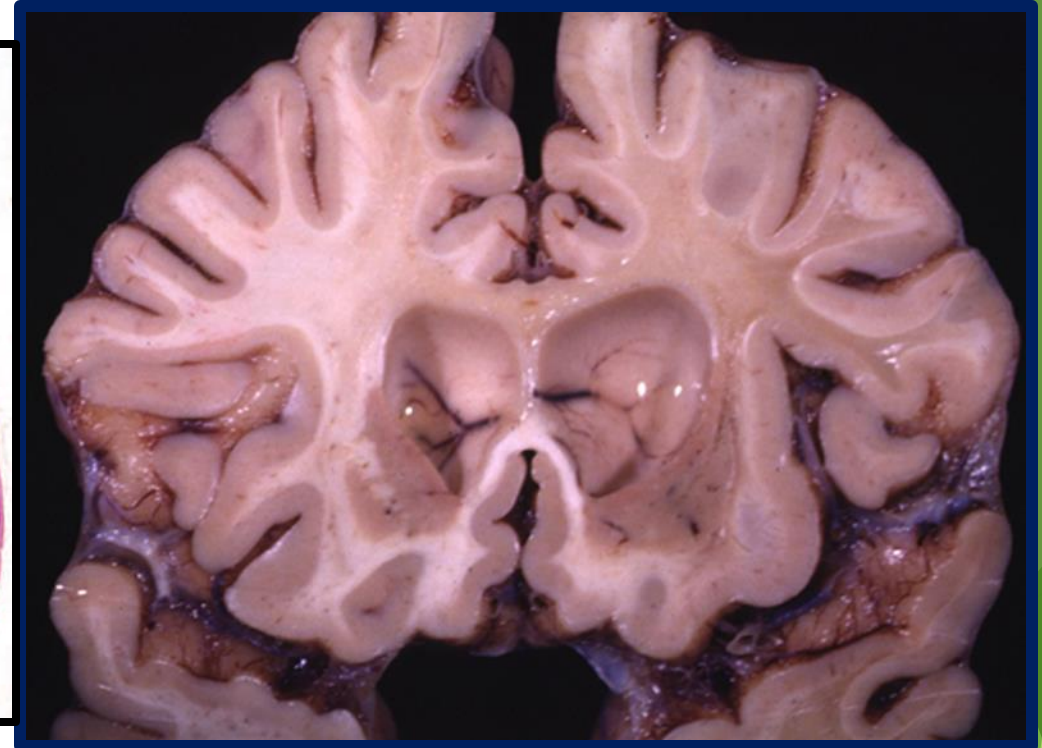
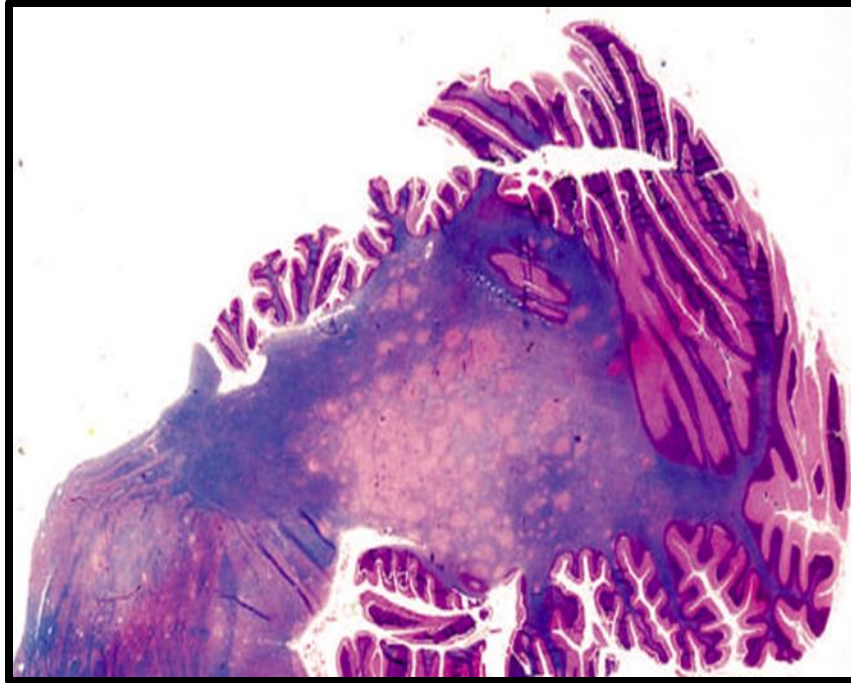
Astrocyte Injury and Repair



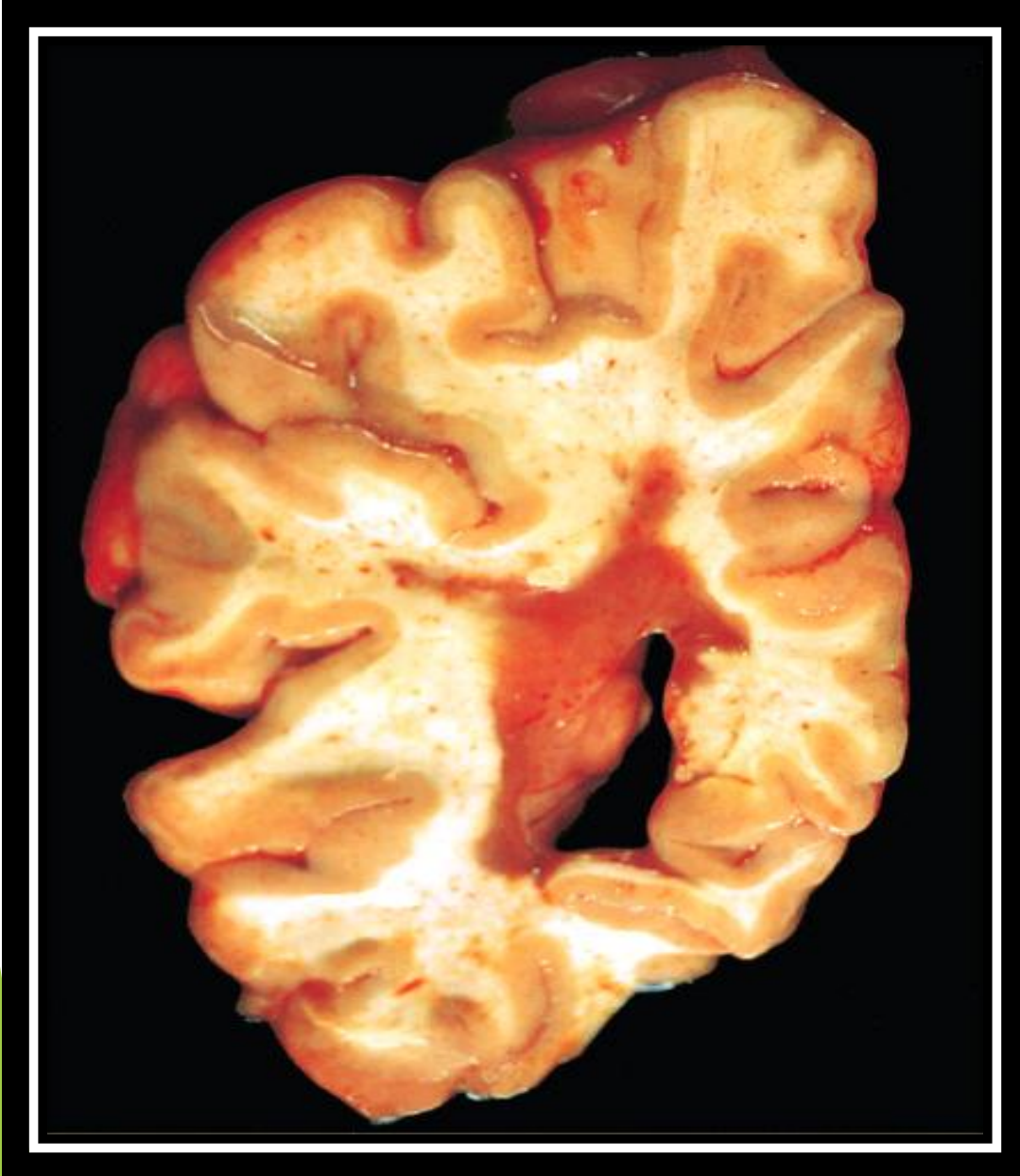
Microglial nodules
(Collection microglial cells)

Demyelinating diseases of CNS

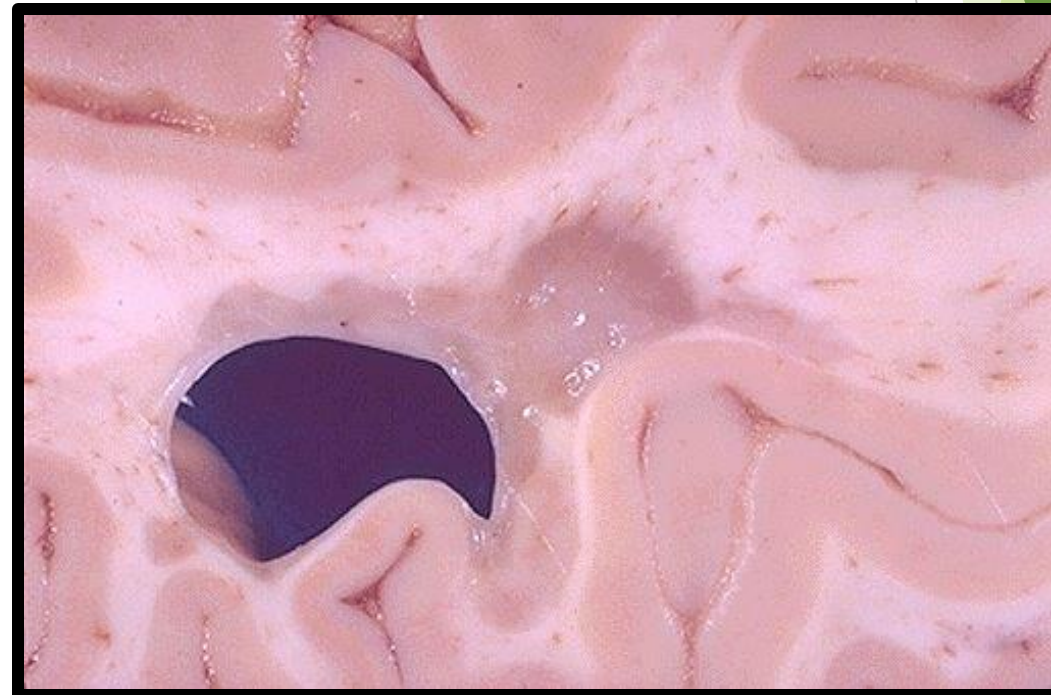
Demyelinating diseases of the CNS



Multiple sclerosis



A white matter disease (multiple sclerosis).
Lesions → plaques: discrete, slightly depressed,
glassy-appearing, and gray in color, and
commonly near the ventricles



Multiple Sclerosis (MS)

- ▶ Lesions are sharply defined microscopically:
- ▶ Active plaques (ongoing myelin breakdown): contain abundant macrophages stuffed with myelin debris (lipid), also perivascular cuffs of Lymphocytes.
- ▶ Inactive plaques (quiescent): inflammation mostly disappears, leaving little to no myelin, & gliosis.

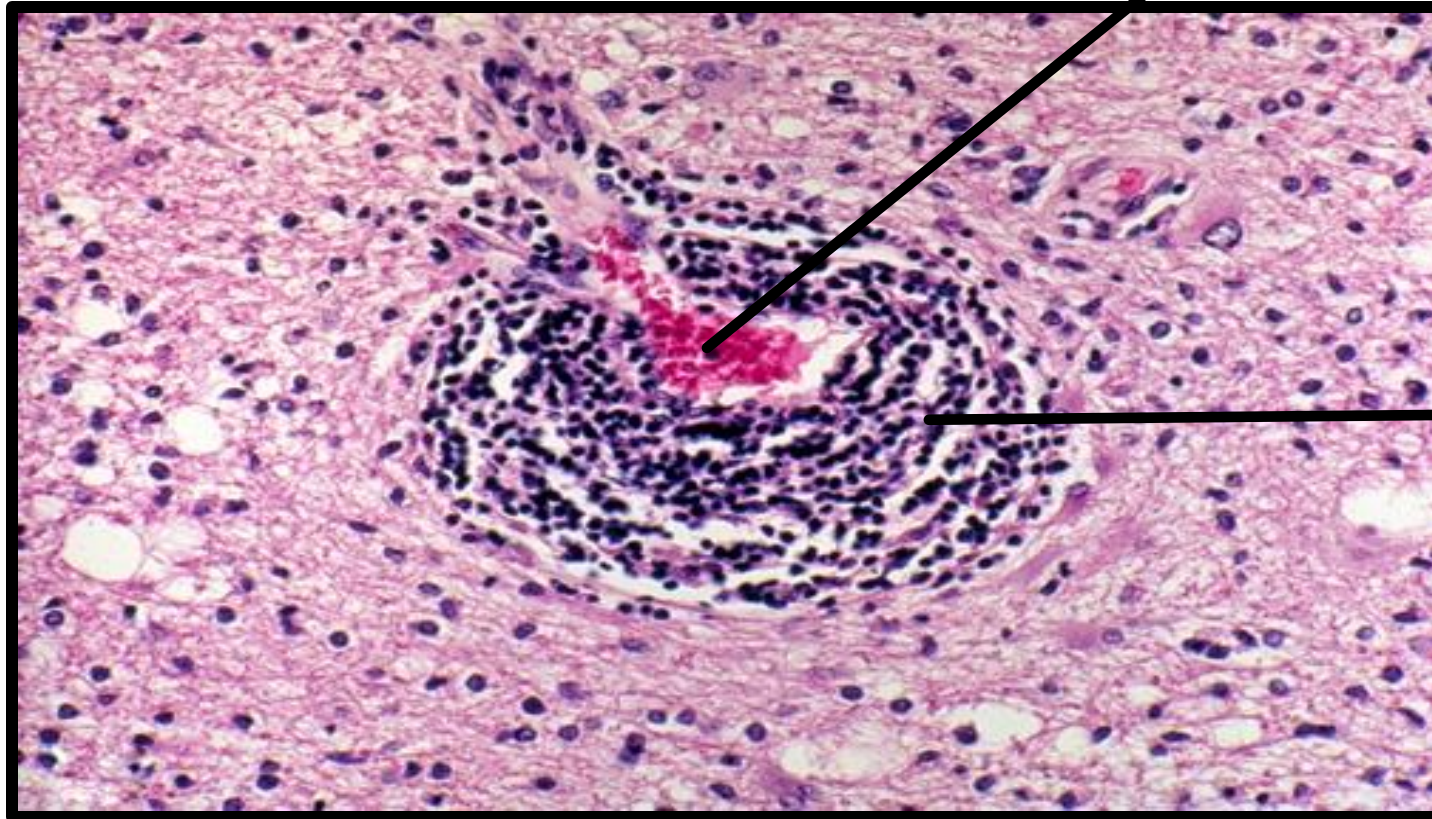


Cross section of the brain,
White plaques are affected
white matter

Multiple sclerosis

**Perivascular cuffs of Lymphocytes
(a sign of MS)**

B.V contain RBCs

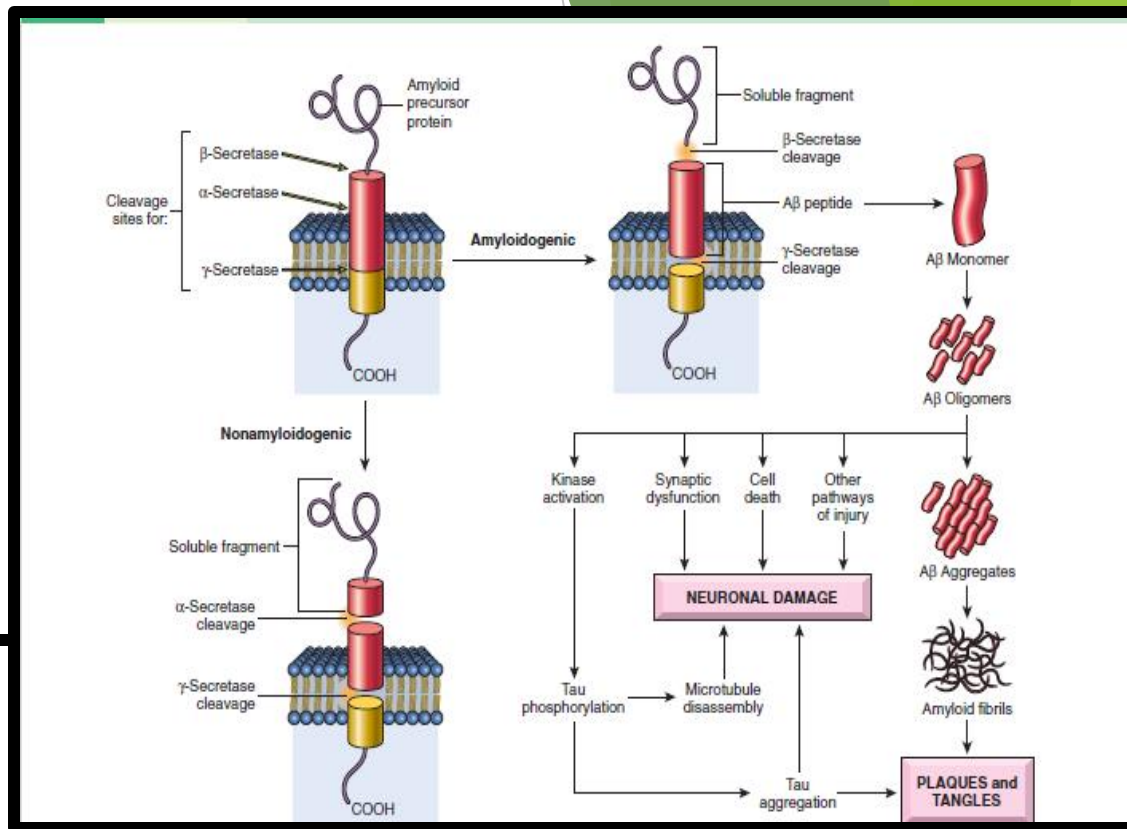


Lymphocytes

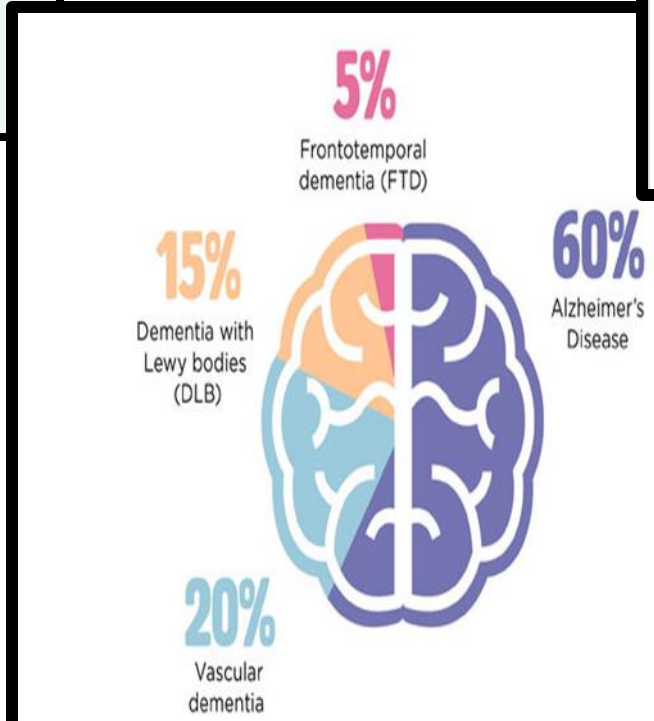
Dementia

- +/-** Poor or decreased judgement
- Head with question mark** Frequent memory loss that affects daily activities
- Abstract shapes** Problems with abstract thinking
- Wh?** Problems with language - e.g forgetting simple words
- Anchor** Loss of initiative
- Keys** Misplacing things or putting them in inappropriate places
- Sad face** Difficulty performing familiar tasks
- Angry face** Changes in personality
- Compass** Disorientation with time and place
- Sad face with frown** Changes in mood or behaviour

Formation of Beta Amyloid and Tau in Alzheimer D.



Irreversible dementia

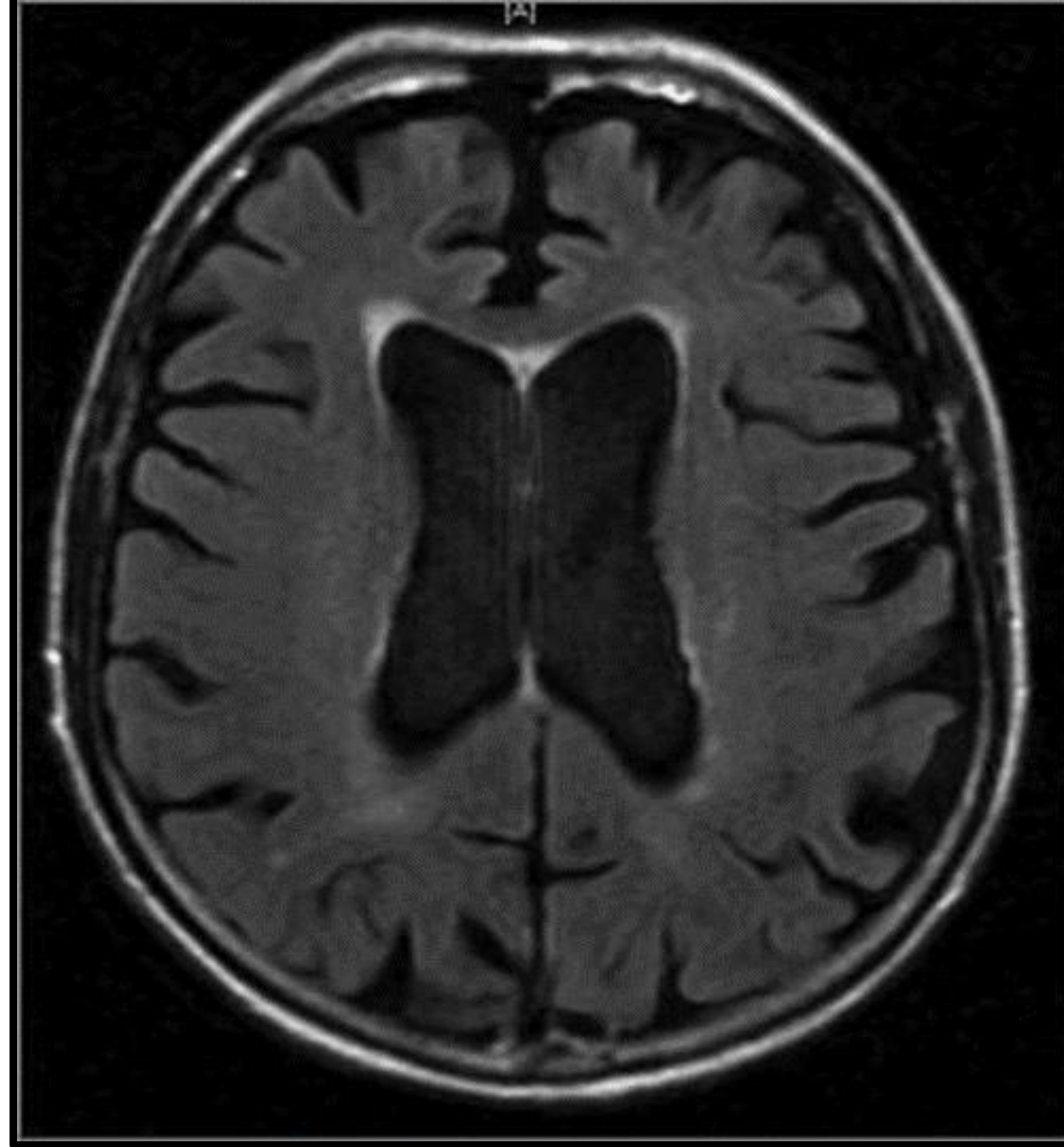
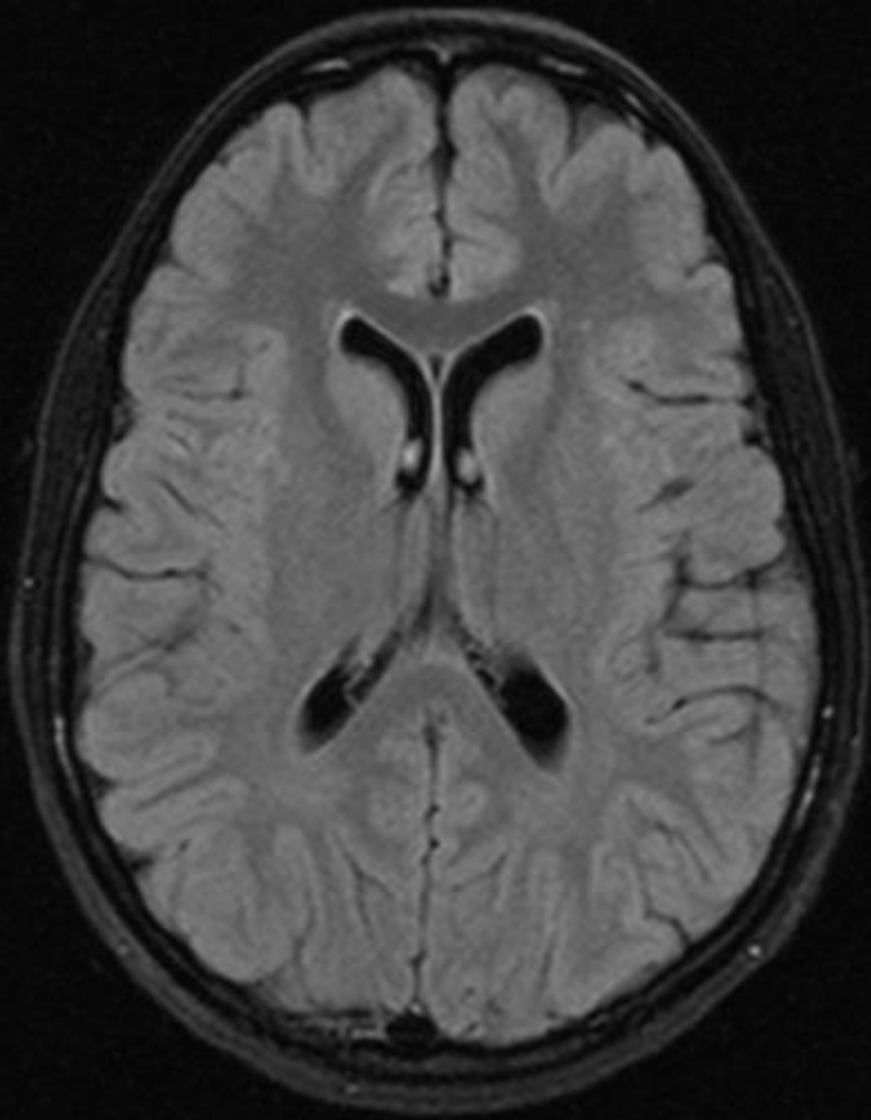


Alzheimer disease



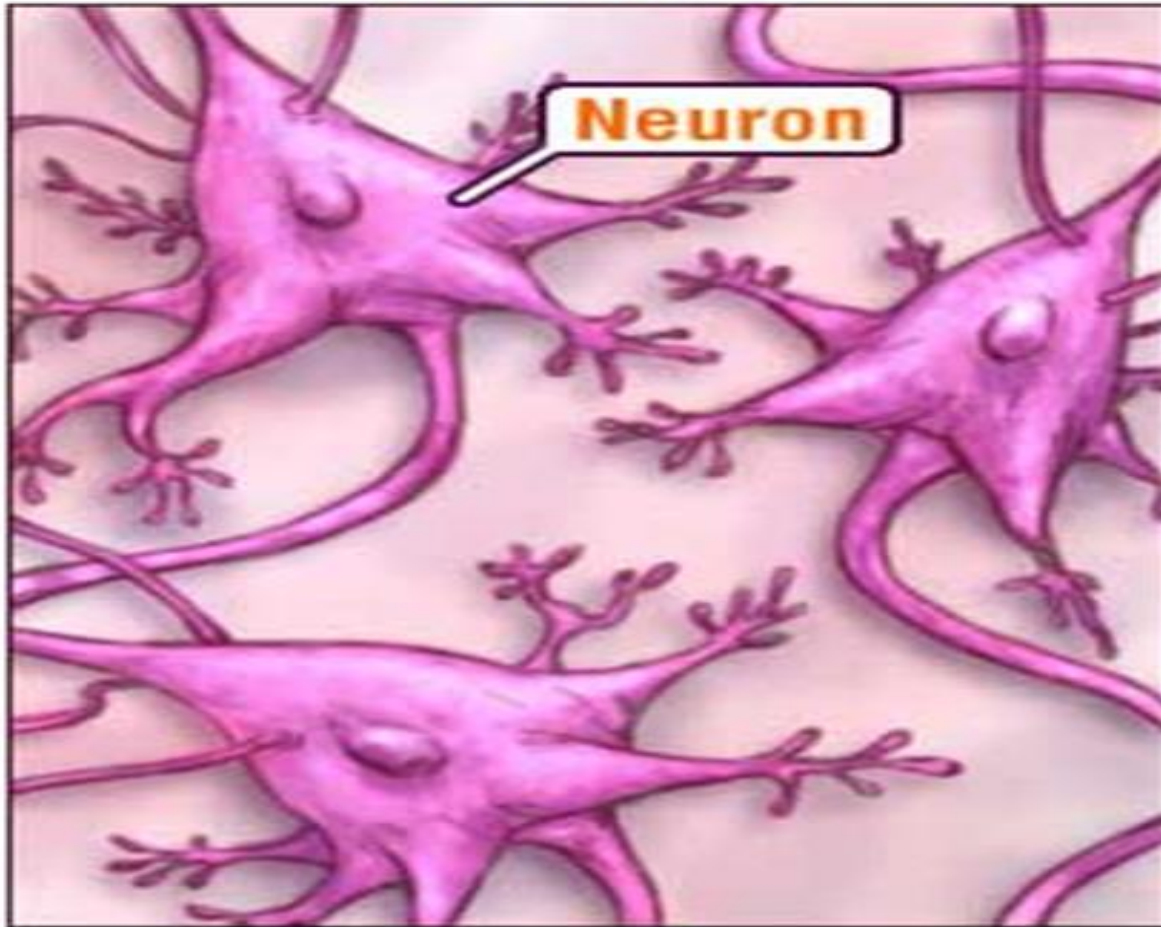
A variable degree of cortical atrophy, resulting in a widening of the cerebral sulci that is most pronounced in the frontal, temporal, and parietal lobes.

Alzheimer
disease

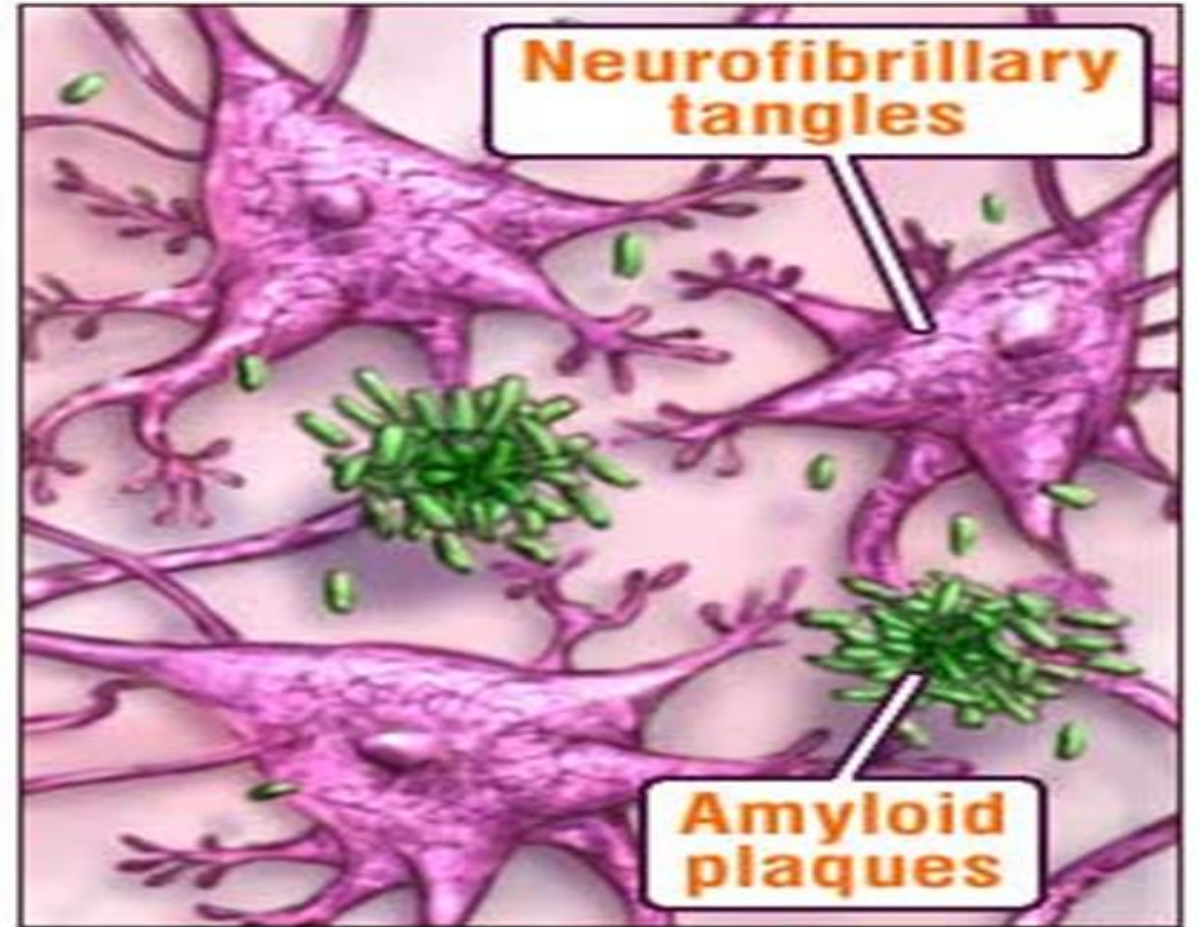


The atrophy produces a compensatory ventricular enlargement (hydrocephalus ex vacuo)

Normal

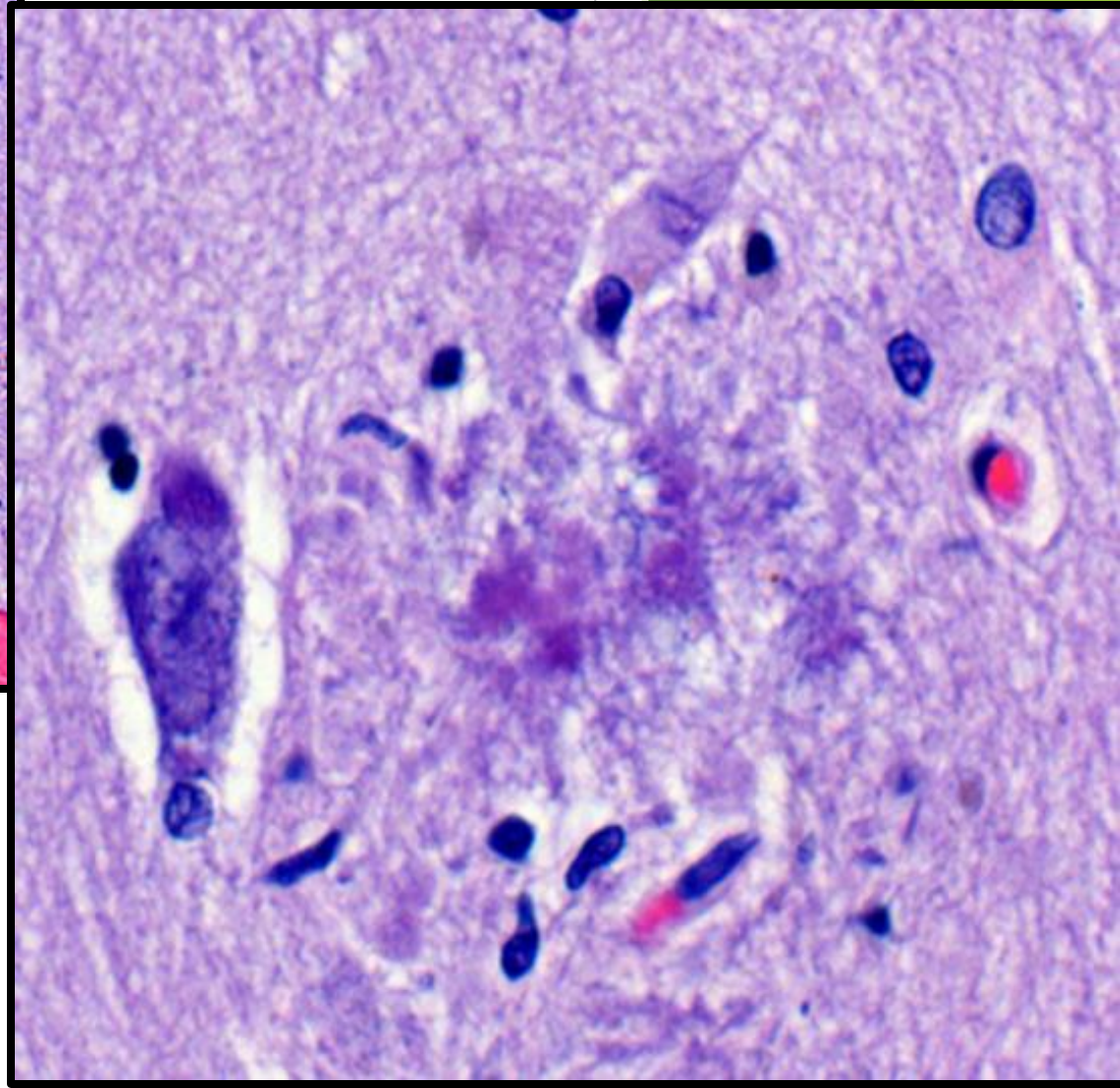
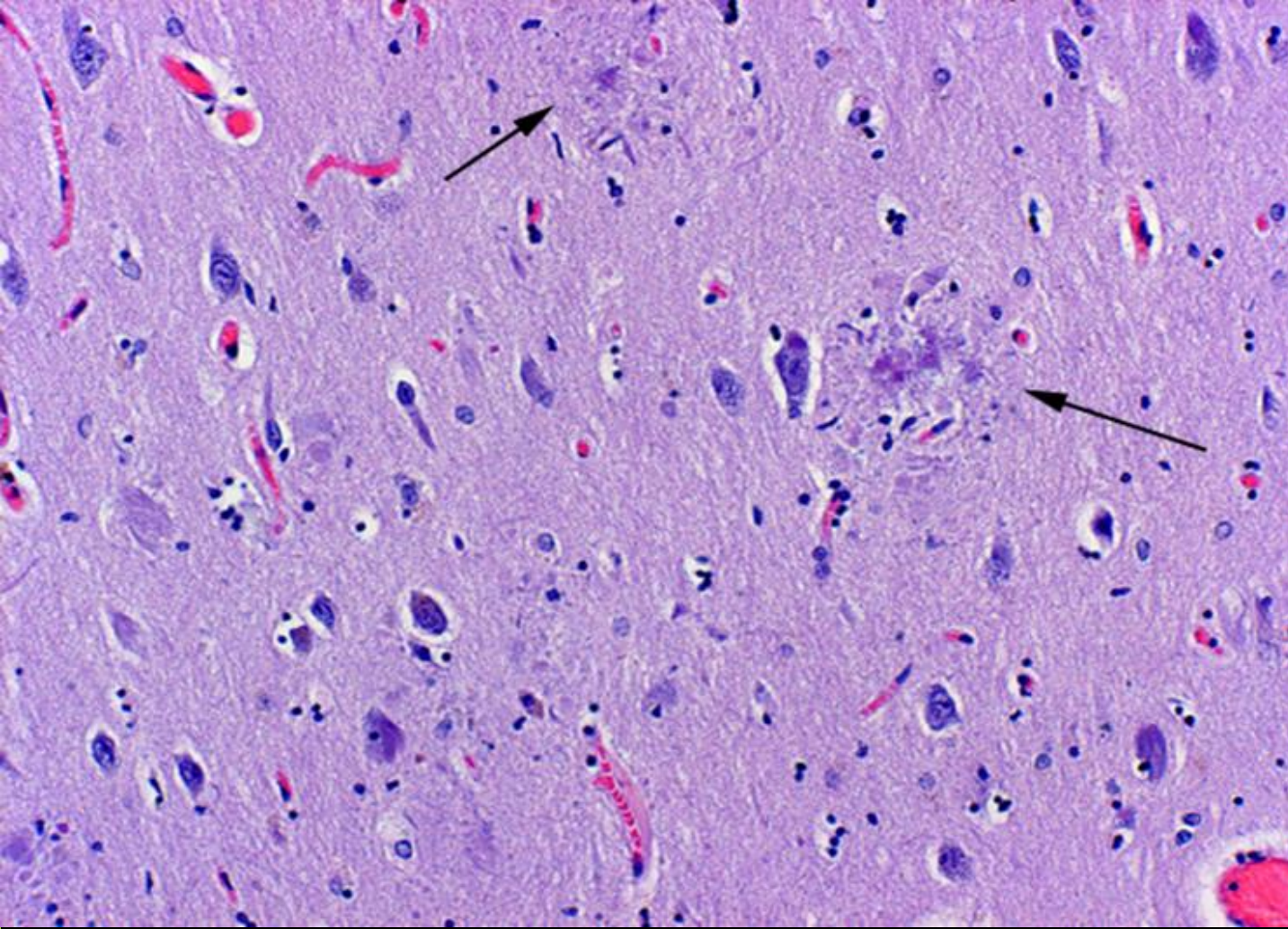


Alzheimer's



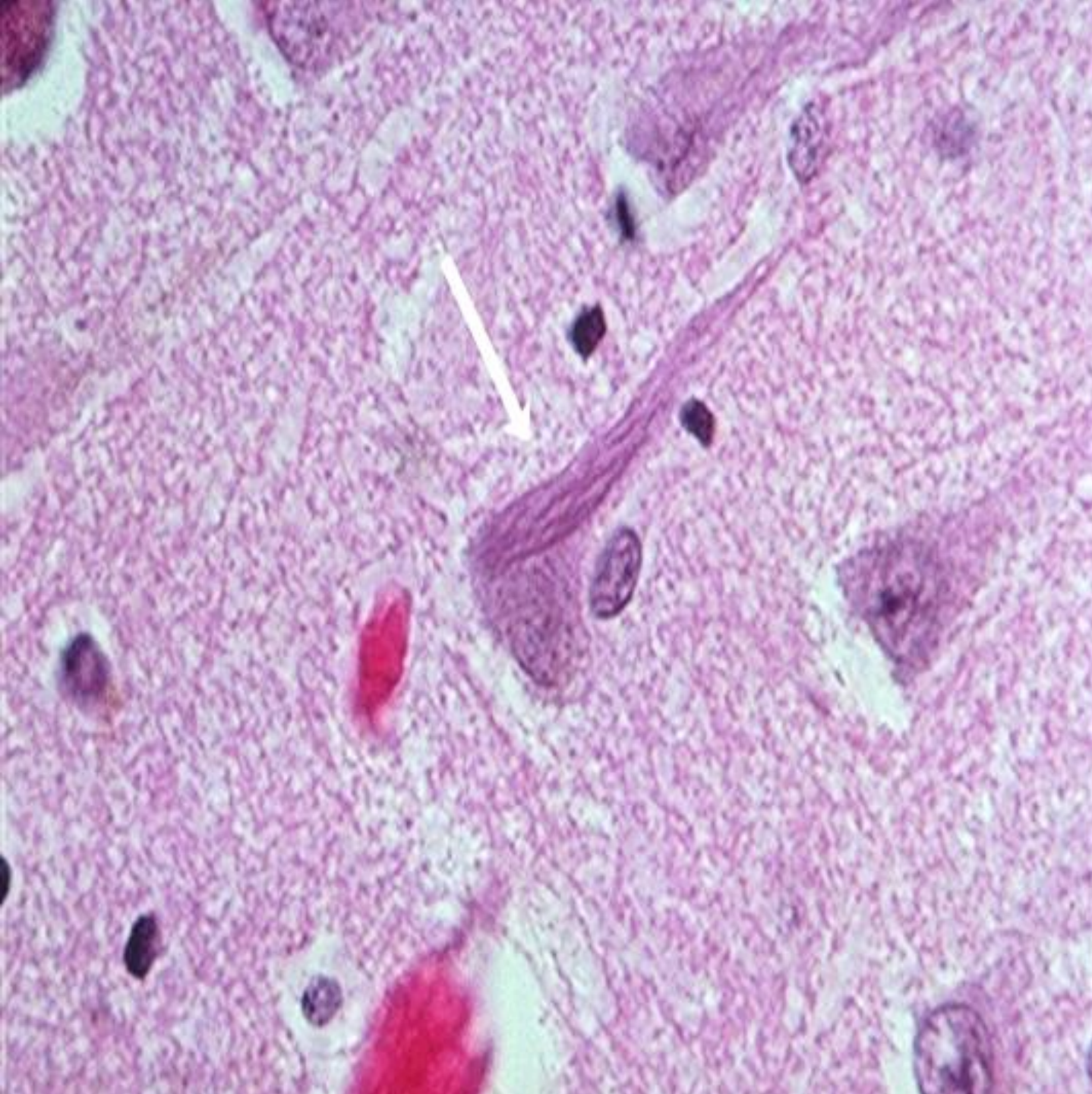
Microscopy: Amyloid plaques (extracellular - accumulation of A β amyloid) and neurofibrillary tangles (intracellular - Tau accumulation).

Alzheimer disease



Neuritic plaques (extracellular) are focal, spherical collections of dilated, tortuous, processes of dystrophic neurites around a central amyloid (A β) core. A β deposition without neurites termed diffuse plaques.

Alzheimer disease



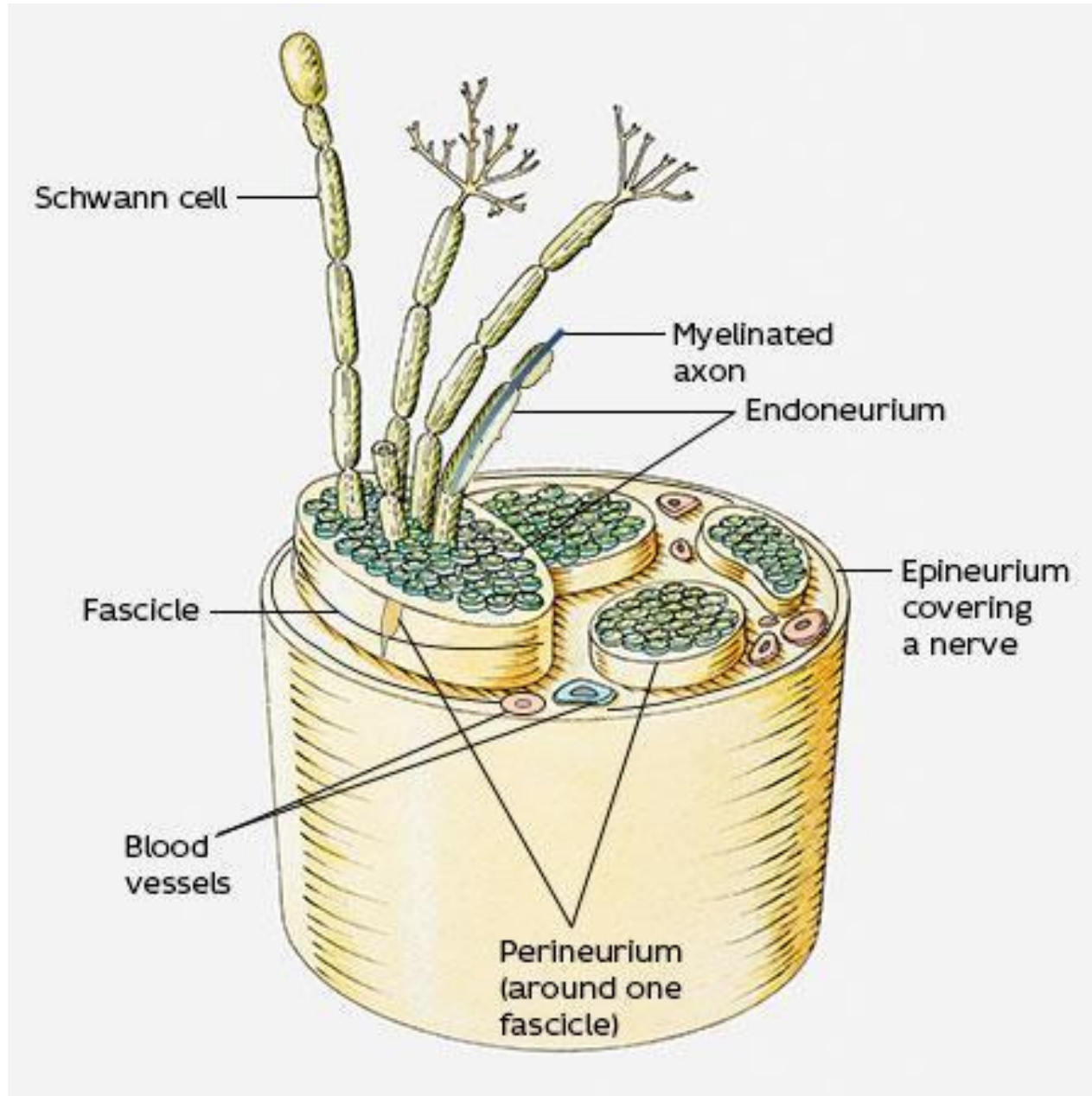
Neurofibrillary tangles: Tau containing bundles of filaments in neurons cytoplasm (encircle the nucleus), <flame shapes> Where ? cortical neurons (entorhinal cortex), & the pyramidal cells of hippocampus, amygdala, basal forebrain, the raphe nuclei.

Peripheral Nervous system Pathology



Sura Al Rawabdeh, MD
Mar 7th 2021

Anatomy of peripheral nerves

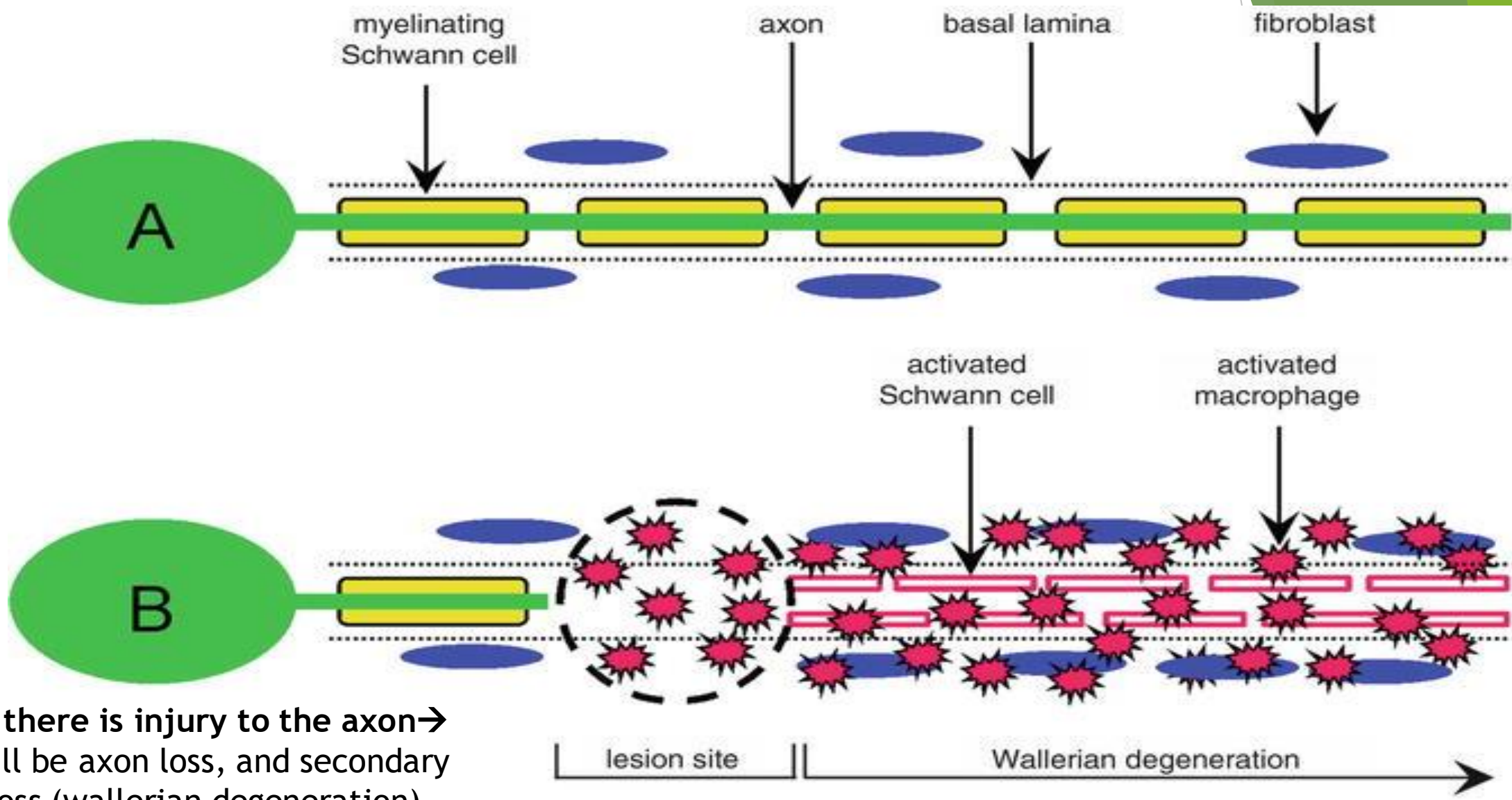


Epineurium: enclose the entire nerve.

Perineurium: groups subset of axons into fascicles.

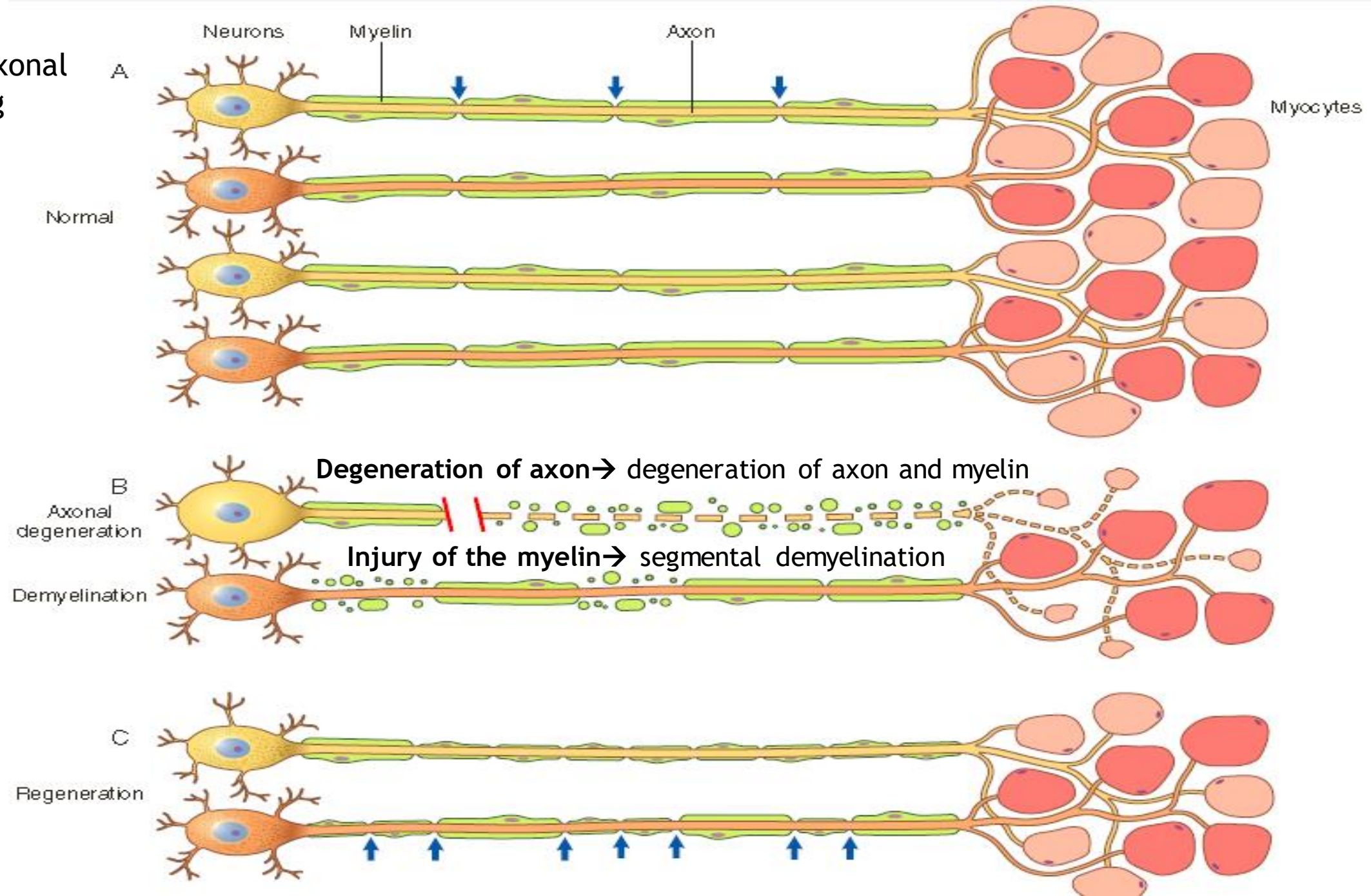
Endoneurium: surrounds individual nerve fibers.

Peripheral neuropathies → axonal neuropathies



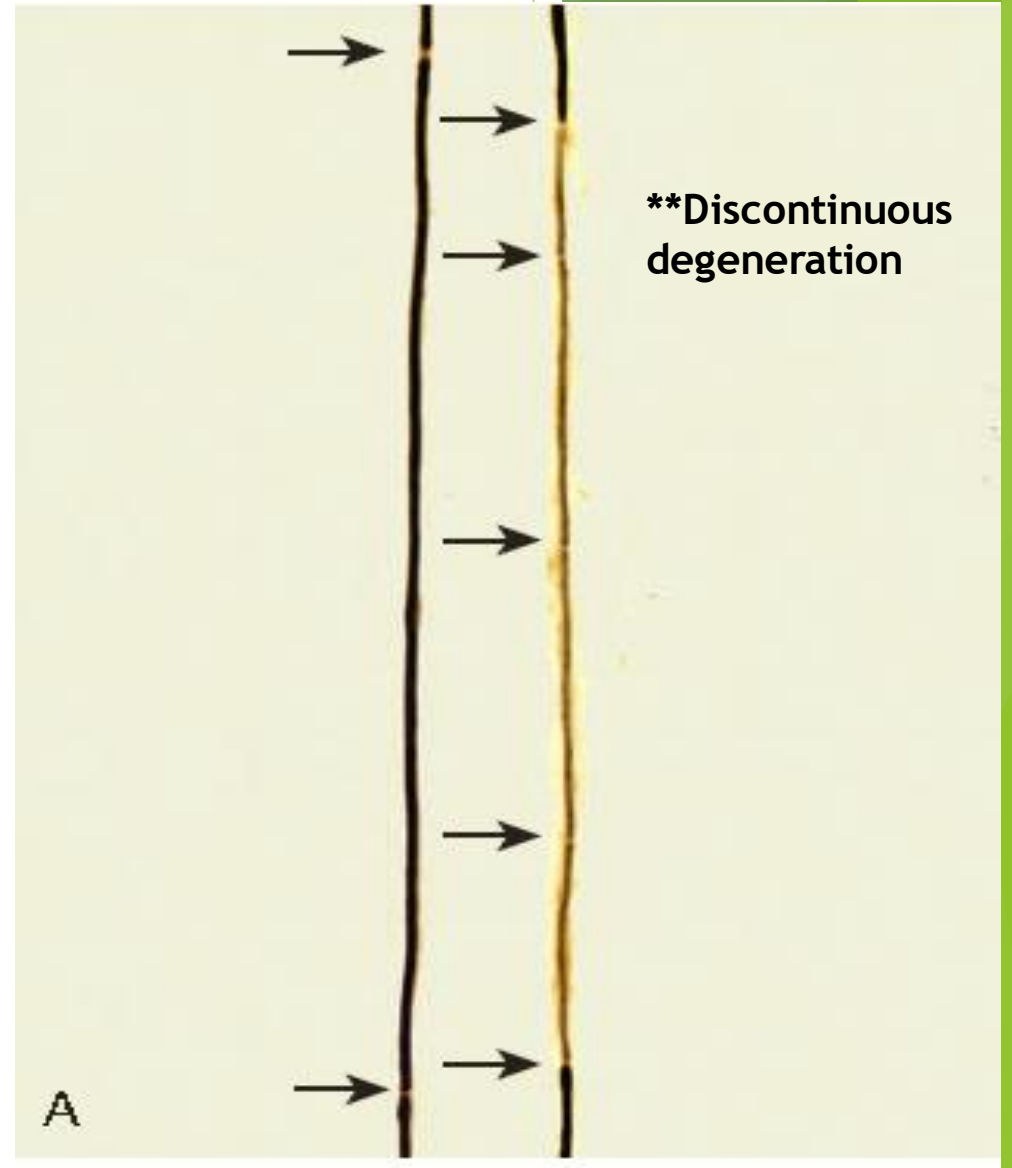
****When there is injury to the axon → there will be axon loss, and secondary myelin loss (wallerian degeneration)**

Peripheral neuropathies → Axonal and Demyelinating neuropathies



Demyelinating neuropathies

- Denuded axon provides a stimulus for remyelination
- Regeneration gives thinly myelinated internodes of uneven length (shorter).



Demyelinating polyneuropathy

GUILLAIN-BARRE' SYNDROME

RISK FACTORS:

- POSSIBLY AUTOIMMUNE
- MORE COMMON: 20 to 50-YEAR-OLDS
- ? ASSOCIATION WITH SWINE FLU IMMUNIZATIONS
- FREQUENTLY PRECEDED BY MILD RESPIRATORY OR INTESTINAL INFECTION

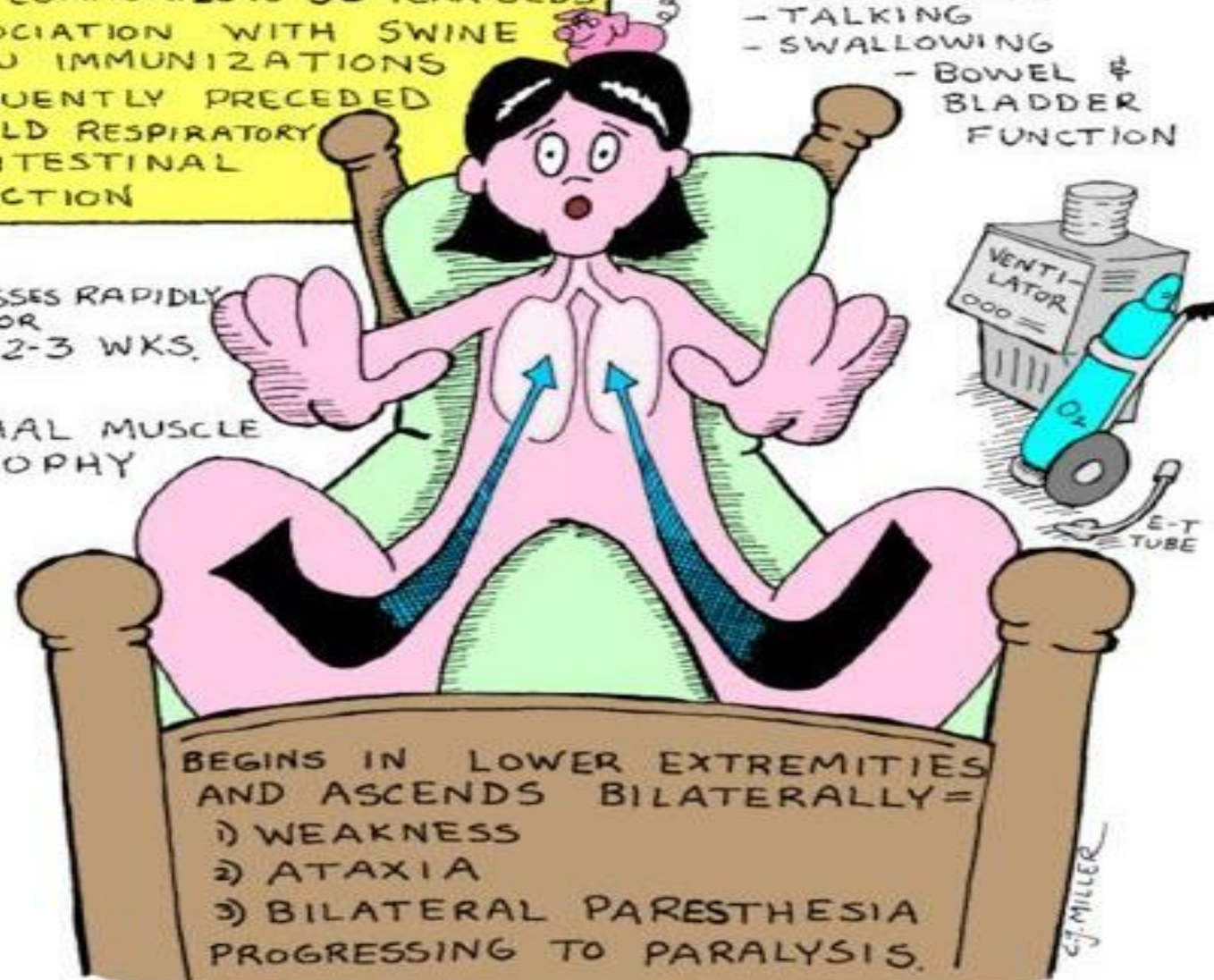
SYMMETRICAL PARALYSIS

CAUSES PROBLEMS WITH:

- RESPIRATION
- TALKING
- SWALLOWING
- BOWEL & BLADDER FUNCTION

- PROGRESSES RAPIDLY
OR
OVER 2-3 WKS.

- MINIMAL MUSCLE
ATROPHY



BEGINS IN LOWER EXTREMITIES
AND ASCENDS BILATERALLY =
1) WEAKNESS
2) ATAXIA
3) BILATERAL PARESTHESIA
PROGRESSING TO PARALYSIS.

G. MILLER

Chronic inflammatory demyelinating polyneuropathy (CIPD)

Electron
microscope
photo→



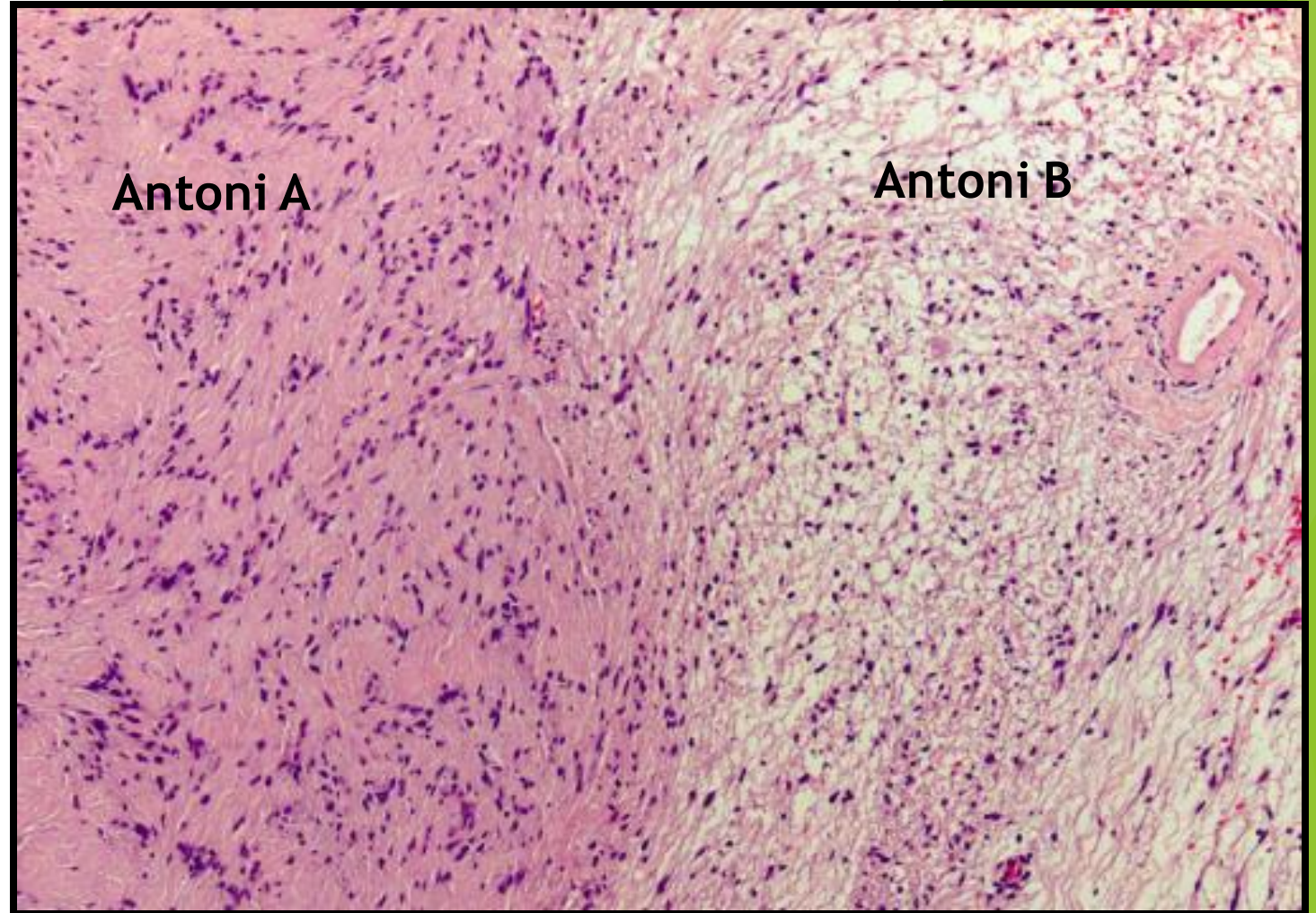
In long-standing cases, repeated activation and proliferation of Schwann cells result in the concentric arrangement of multiple Schwann cells around individual axons to produce multilayered structures → onion bulbs.

PERIPHERAL NERVE SHEATH TUMORS



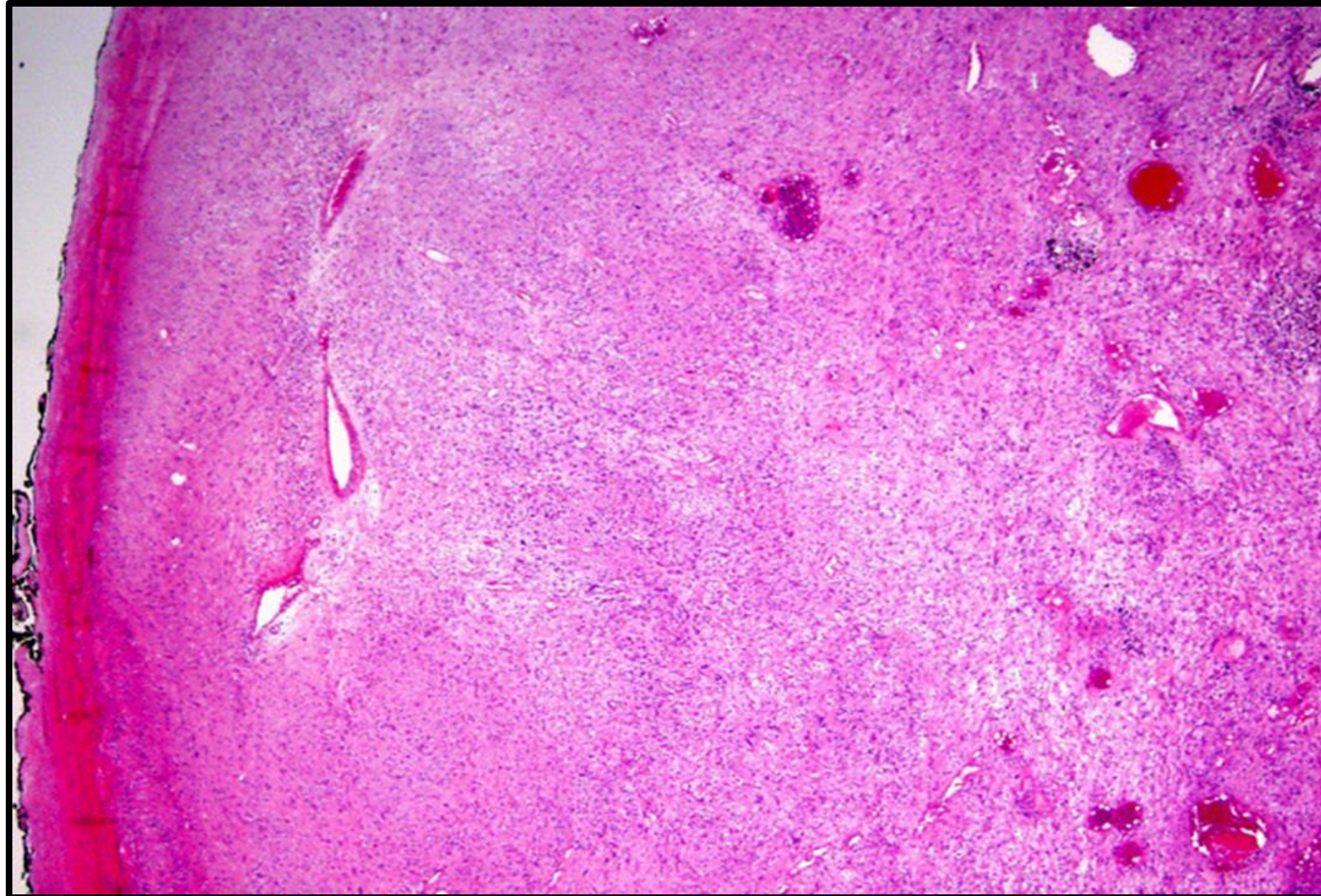
Schwannomas - Morphology

- Grossly:
Circumscribed masses abutting an adjacent nerve.
- Microscopically:
an admixture of dense & loose areas referred to as Antoni A and B, respectively.



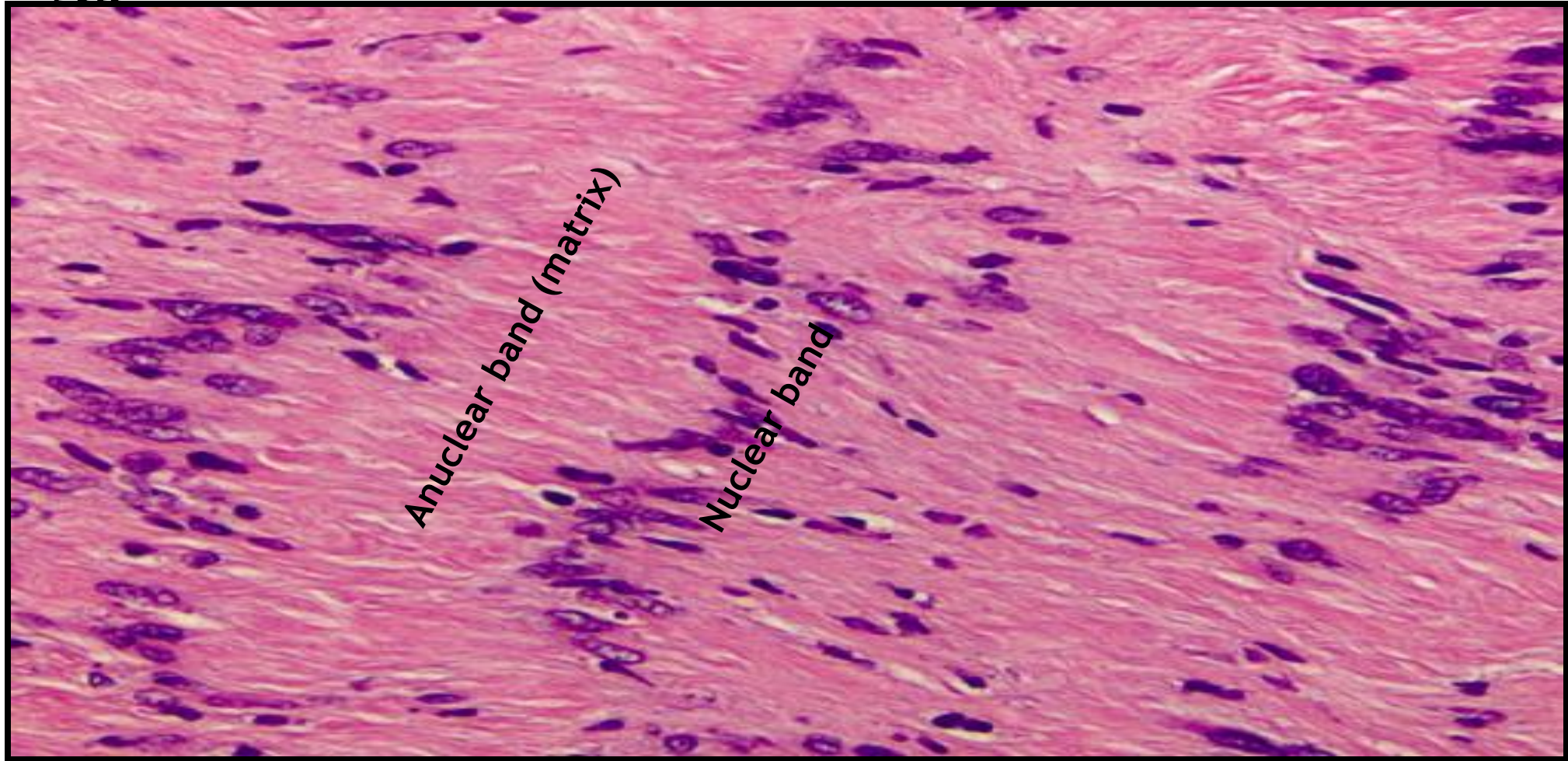
Schwannomas

Well circumscribed encapsulated schwannoma



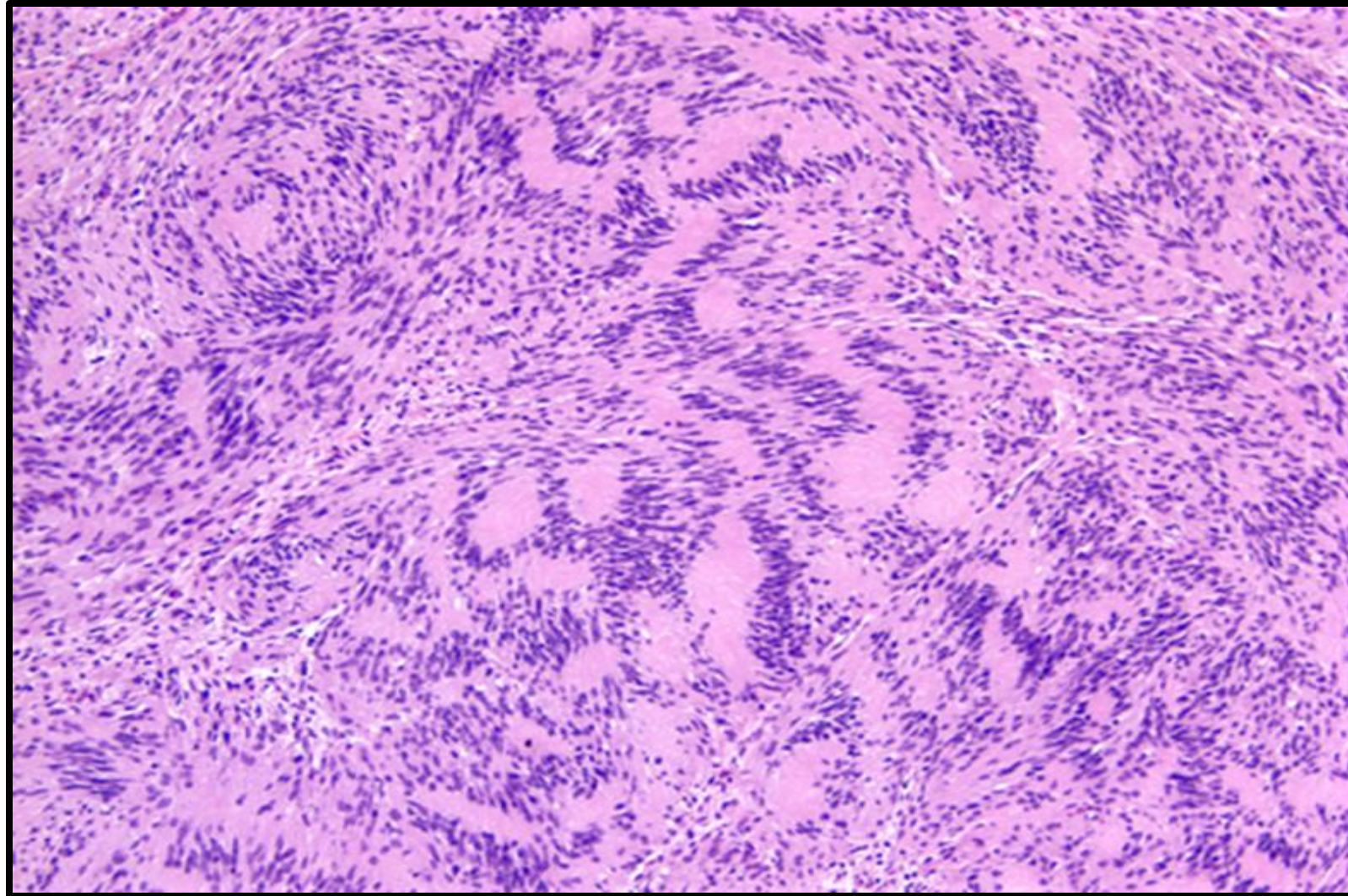
Schwannomas

Tumor cells aligned in palisading rows → Verocay bodies (Antoni A):



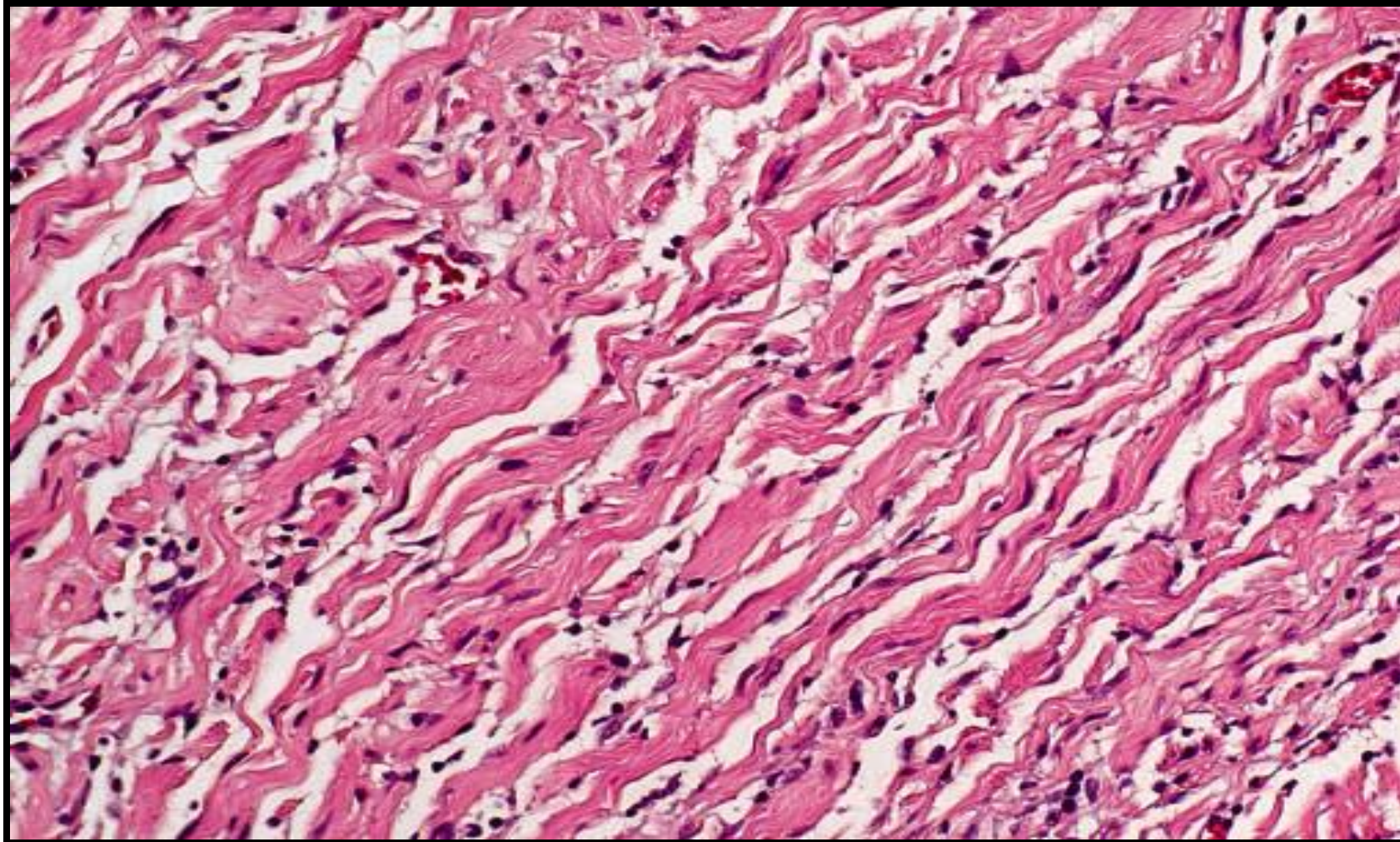
Schwannomas

Tumor cells aligned in palisading rows → Verocay bodies:



Neurofibromas

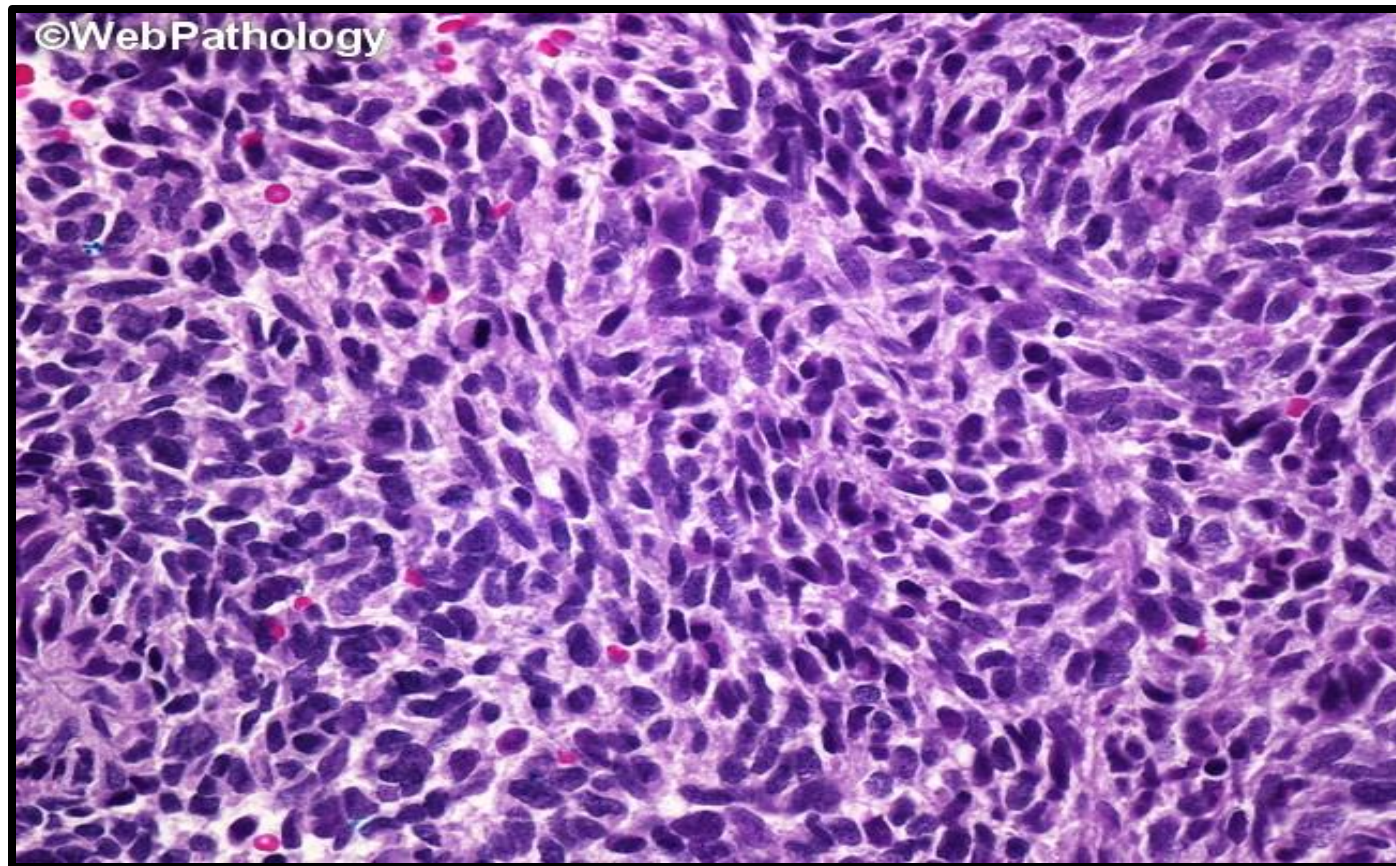
Schwann cells+ Fibroblast separated by thick collagen bundles



****Neural look→**

MPNST

Histologically, 1)highly cellular and exhibit features of overt malignancy; 2)anaplasia, 3)necrosis, 4)hyperchromasia, 5)infiltrative growth pattern, 6)not well circumscribed, 7)pleomorphism, 8)and high proliferative activity (mitoses).



Degenerative diseases of CNS (2)



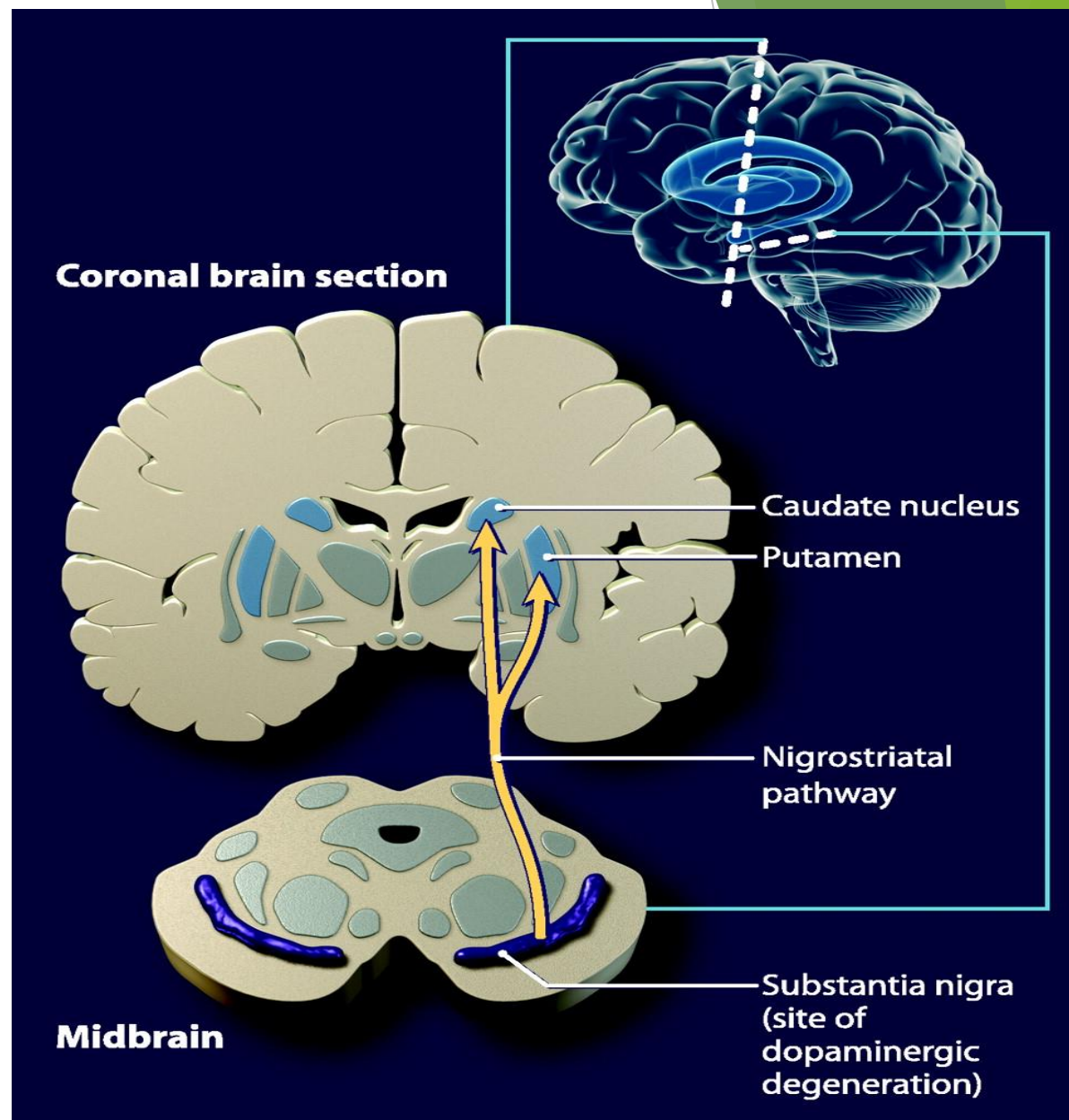
Ghadeer Hayel, MD
Mar 3rd 2021

Parkinson's Disease Symptoms



Parkinson Disease (PD)

Parkinsonism is seen in a range of diseases that damage dopaminergic neurons, which project from the substantia nigra to the striatum (nigrostriatal pathway)

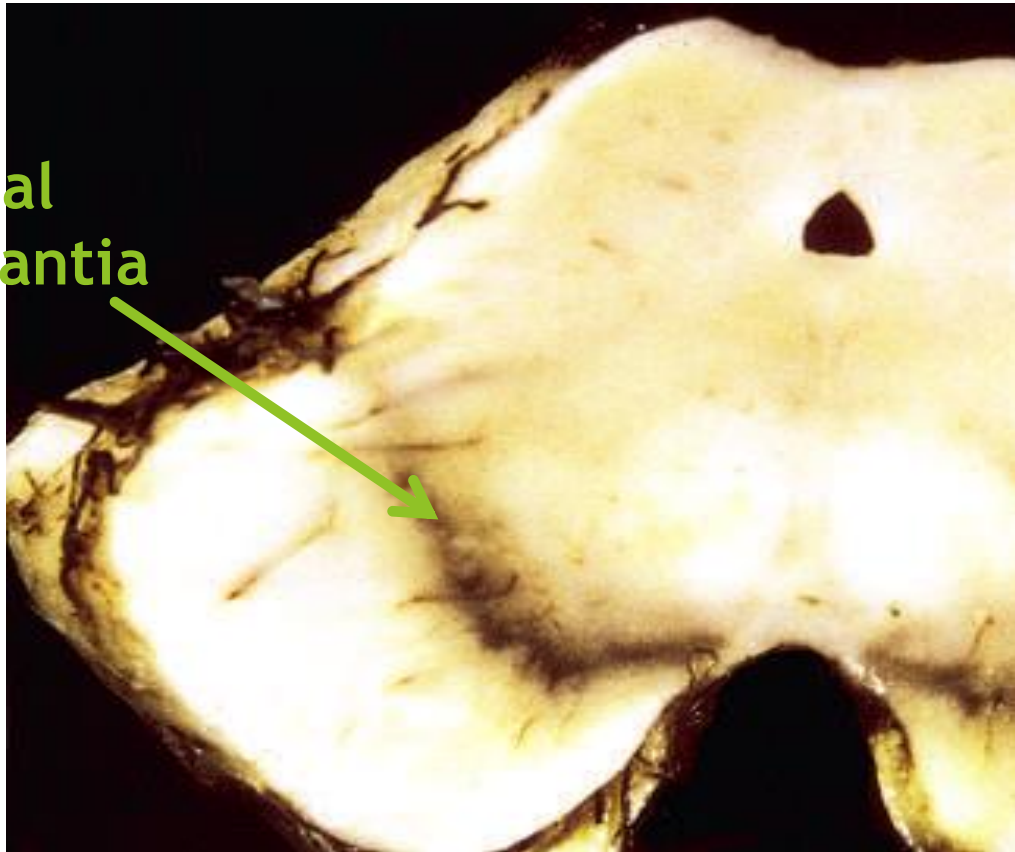


Parkinson disease

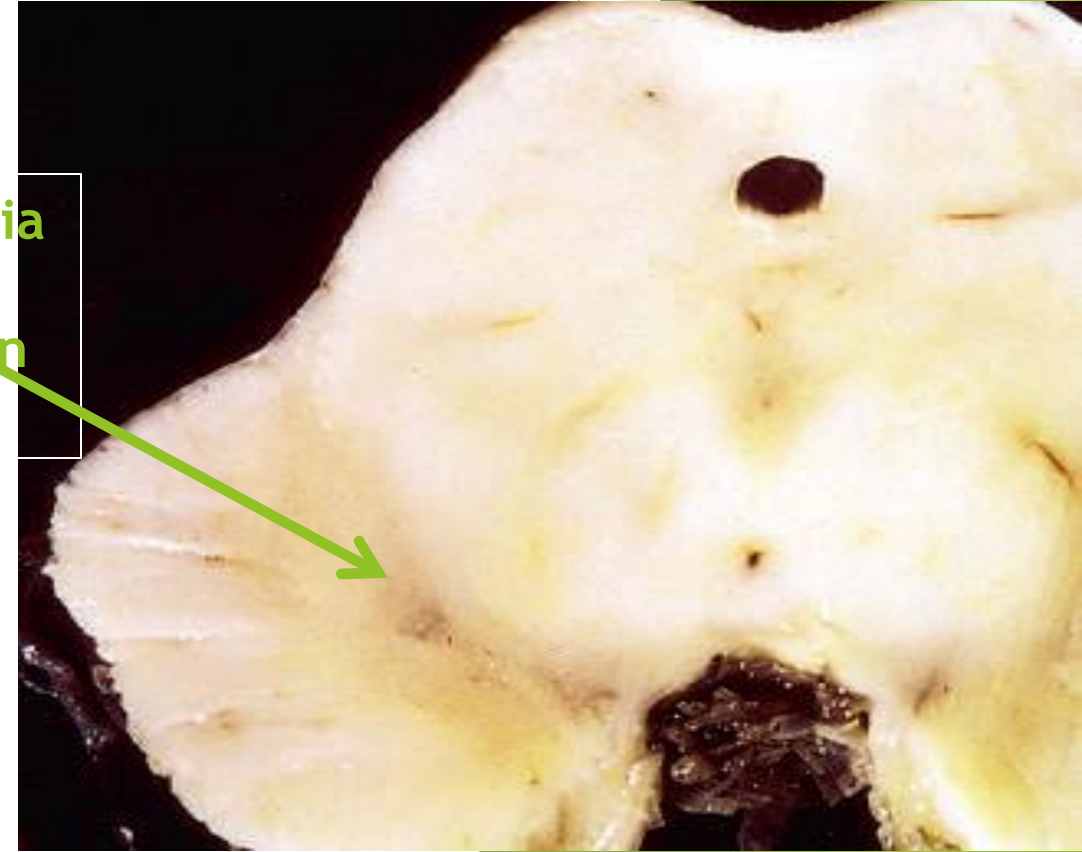


At autopsy is pallor of the substantia nigra and locus ceruleus, due loss of pigmented catecholaminergic neurons.

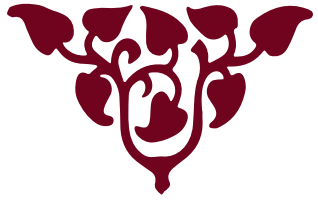
Normal substantia nigra



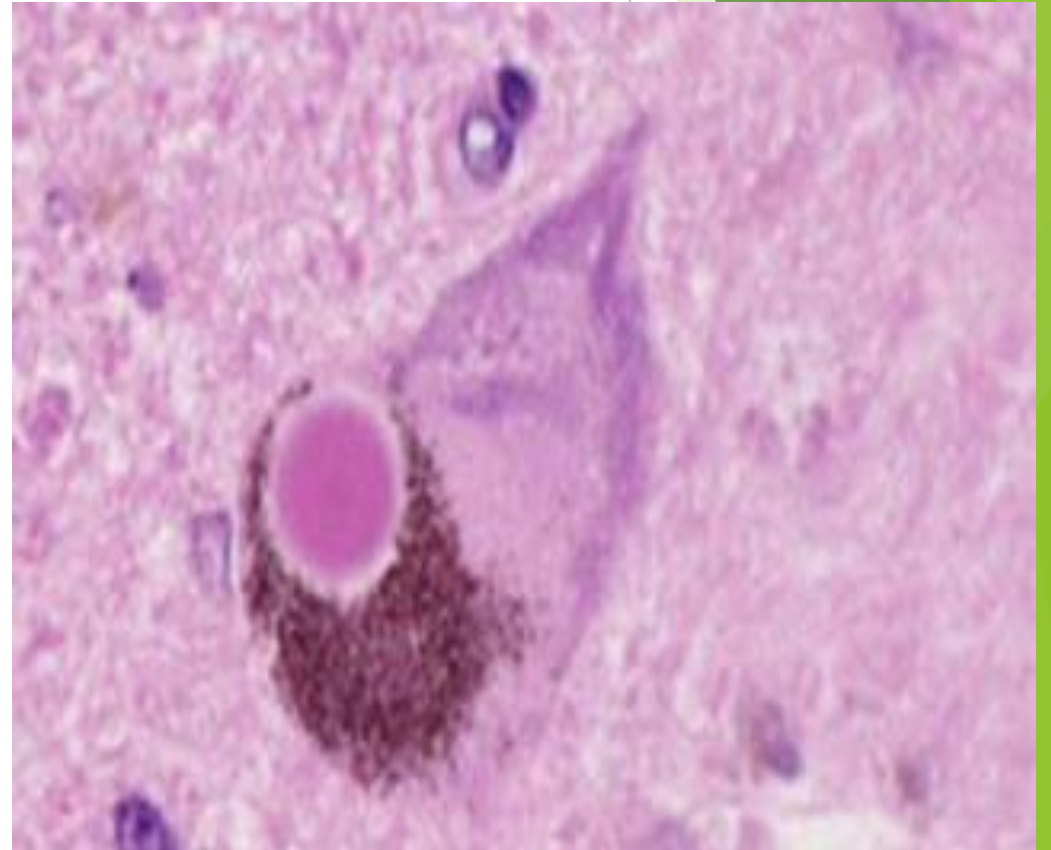
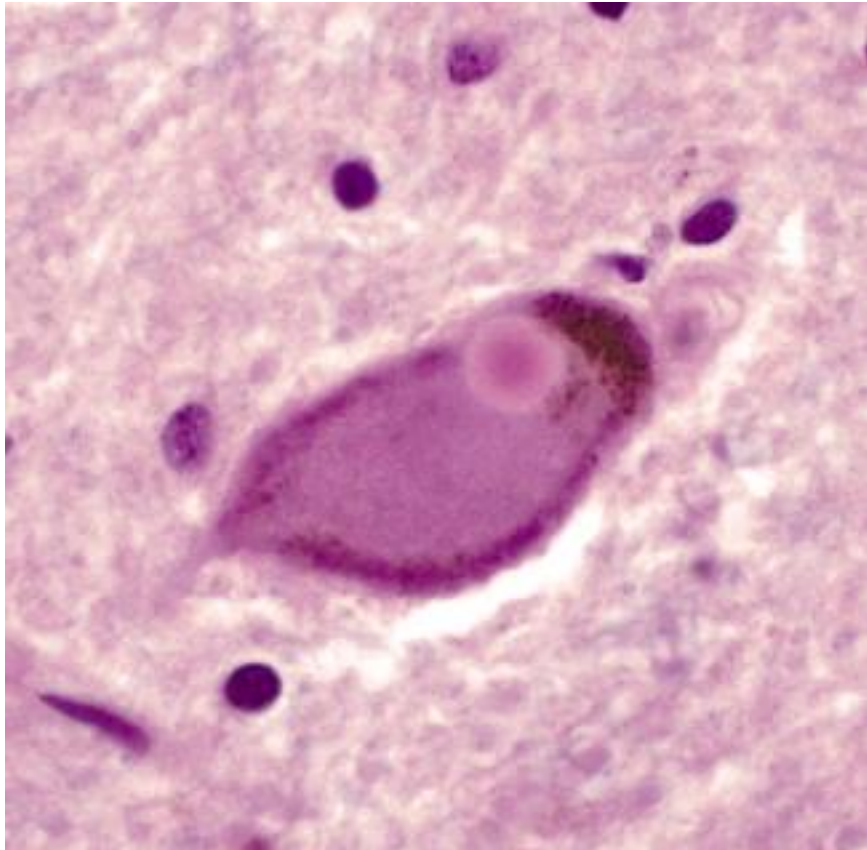
Substantia nigra in parkinson disease



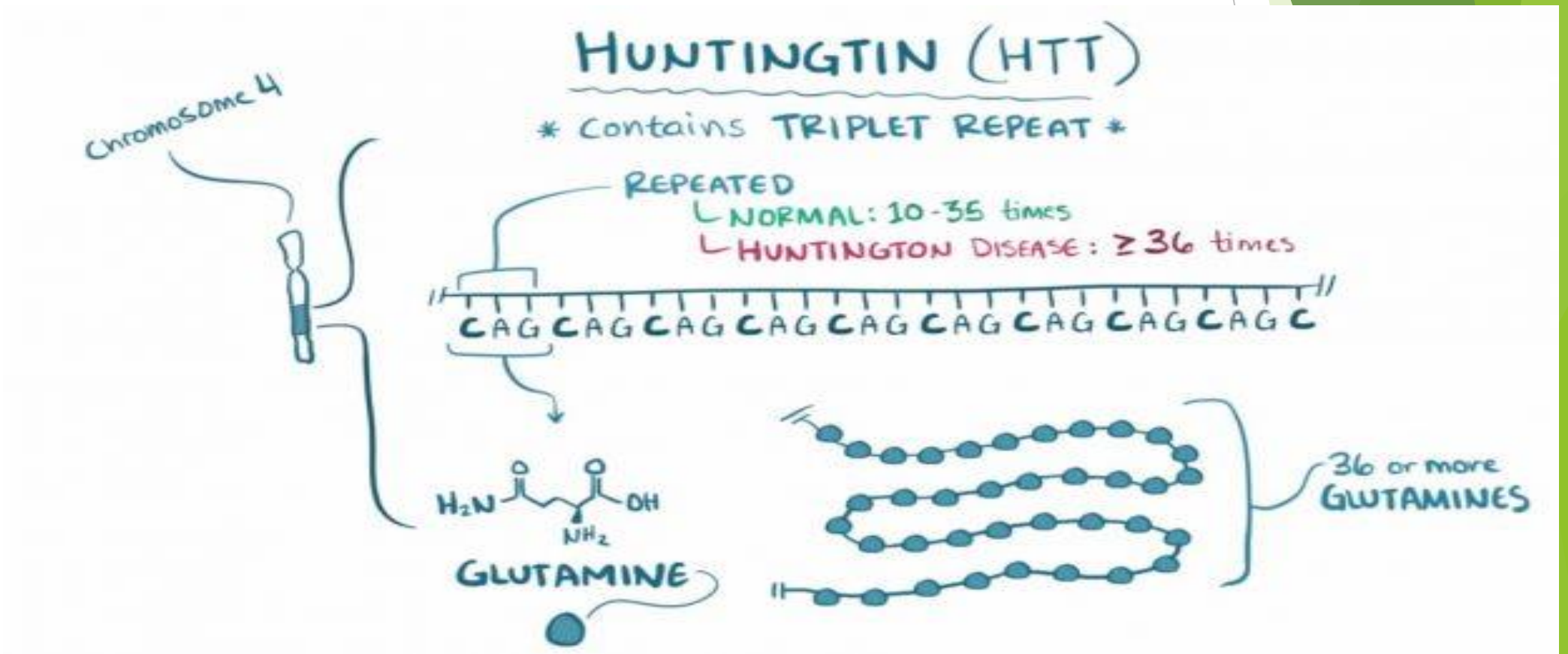
Parkinson disease



Areas of neuronal loss show gliosis. Lewy bodies found in those neurons that remain; single or multiple, cytoplasmic, eosinophilic, round inclusions (dense core with pale halo)



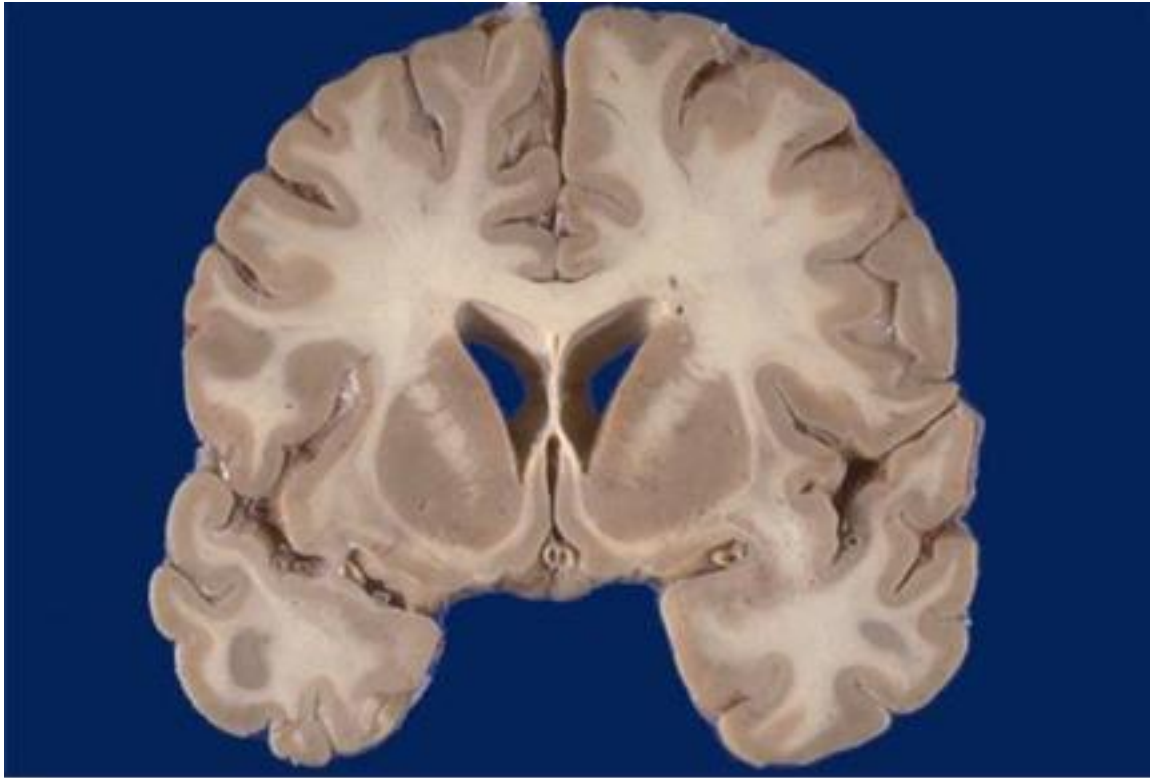
Huntington Disease (HD)



Huntington disease



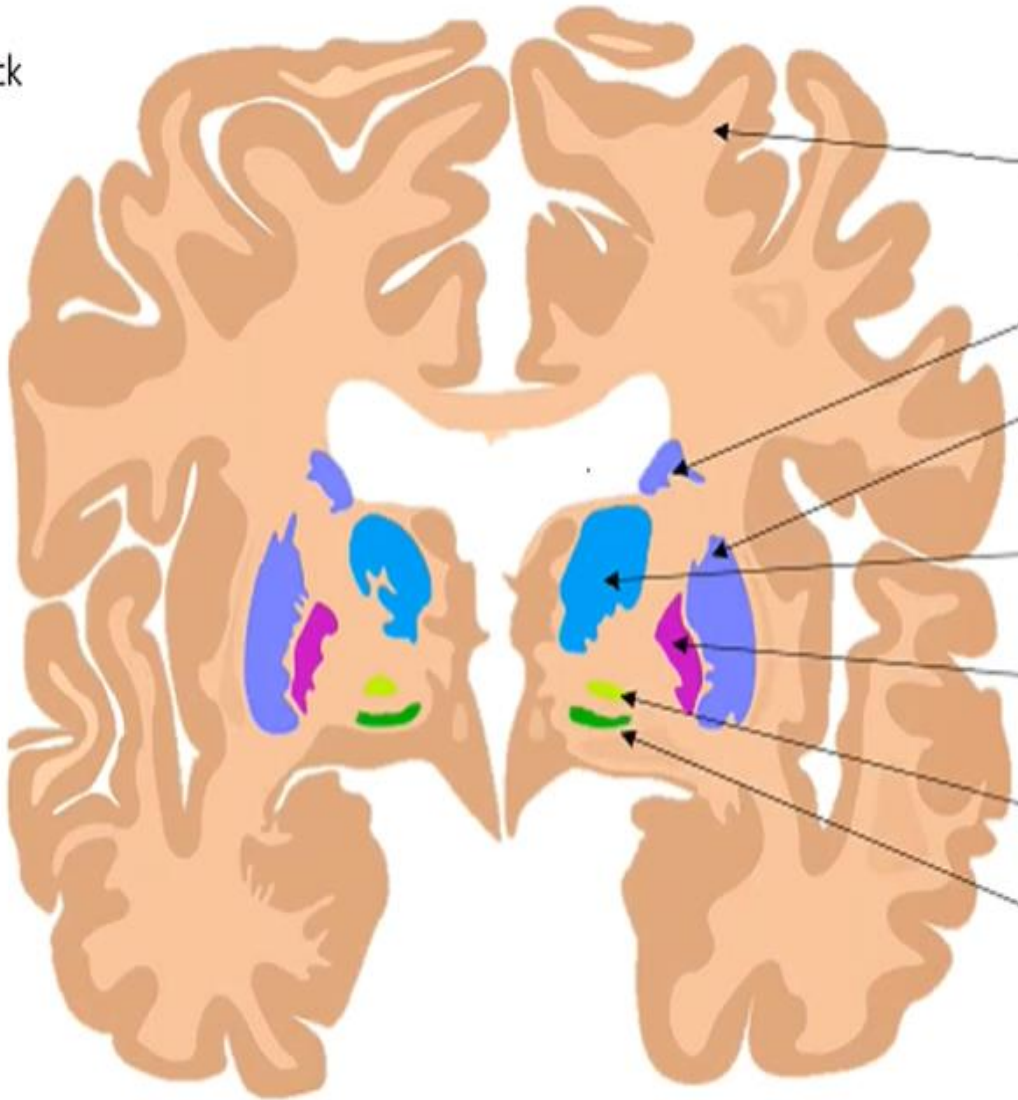
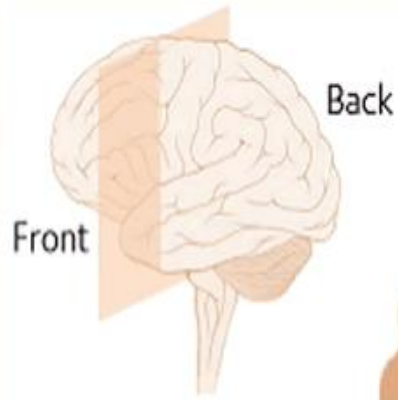
The brain is small and shows striking atrophy of the caudate nucleus and, sometimes, the putamen. The lateral and third ventricles are dilated.



WT



HD



Basal ganglia

Cerebral cortex

Caudate nucleus

Putamen

Striatum

For H.D

Thalamus

Globus pallidus

Subthalamic nucleus

Substantia nigra

For P.D

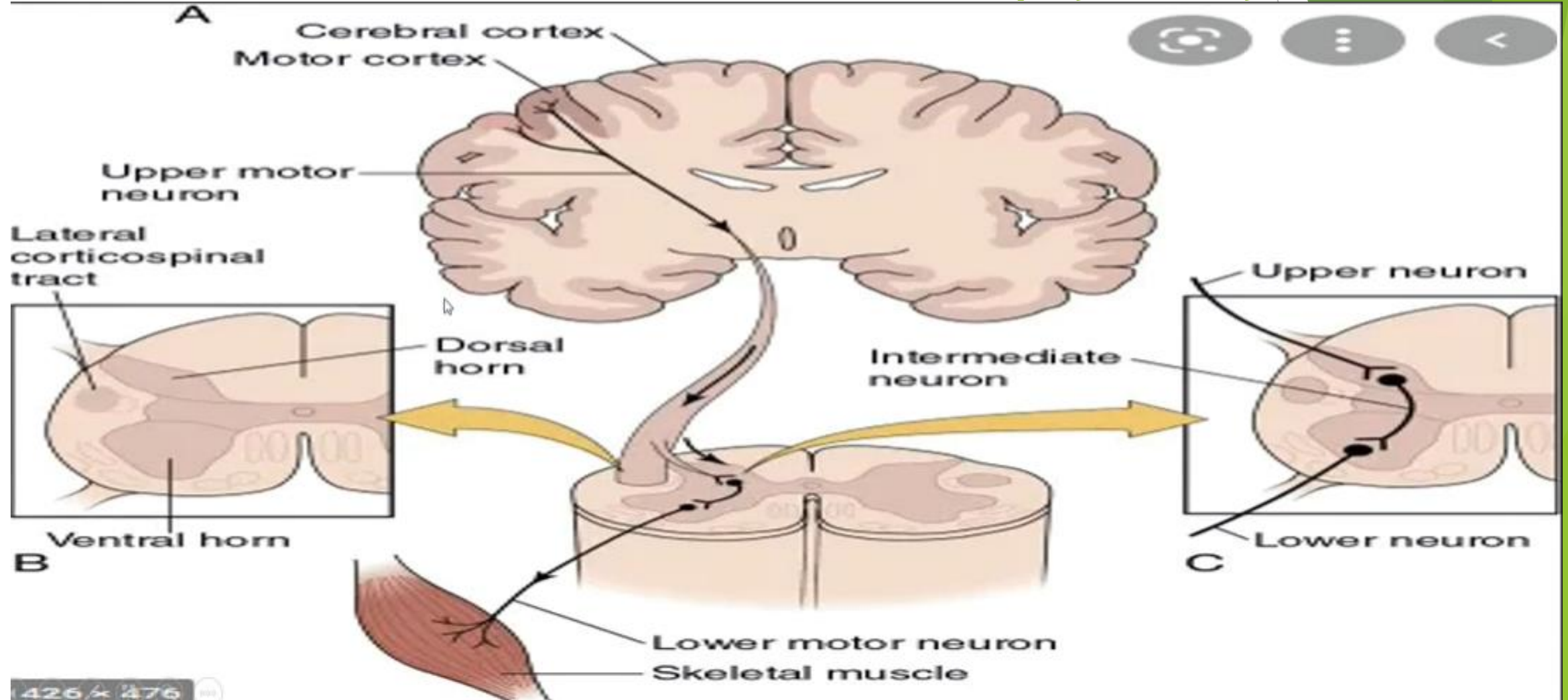
13



Amyotrophic Lateral Sclerosis (ALS)



Loss of UMN and LMN leads to muscle atrophy and dysfunction



Acquired metabolic diseases → Wernicke encephalopathy

Wernicke encephalopathy is characterized by foci of hemorrhage and necrosis in the mamillary bodies and the walls of the third and fourth ventricles.

Foci of hemorrhage

