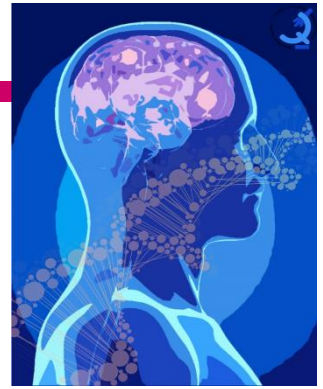

NEUROPATHOLOGY

INTRACRANIAL HEMORRHAGE, EDEMA, HERNIATION

HAJNALKA RAJNAI

Semmelweis Egyetem – I. Patológiai és Kísérleti Rákkutató Intézet

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INTRACRANIAL HEMORRHAGE

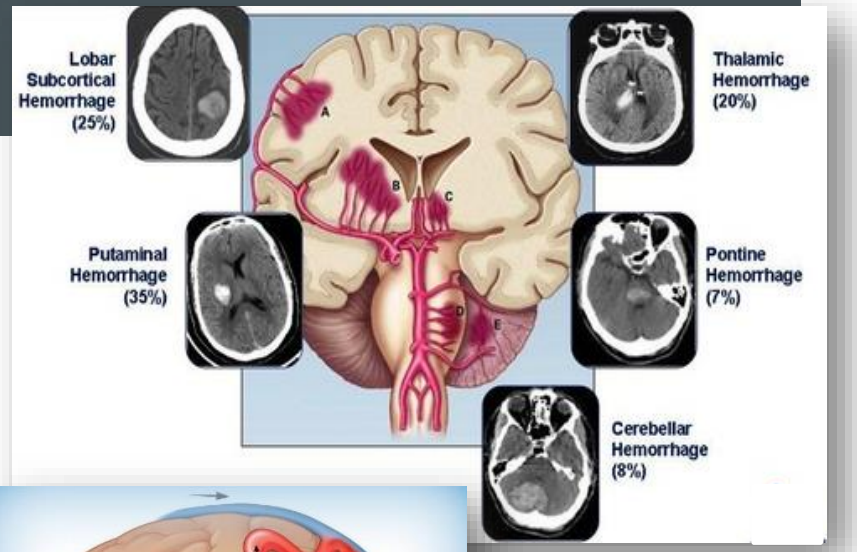
ETIOLOGY:

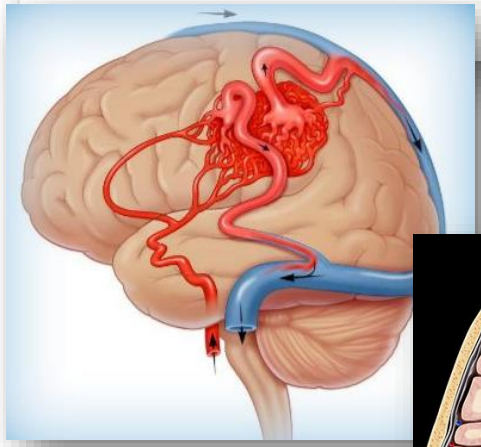
I. **Vascular wall injury**
   - Hypertension
   - Protein deposition
   - Trauma

II. **Primary structural lesions of the vessels**

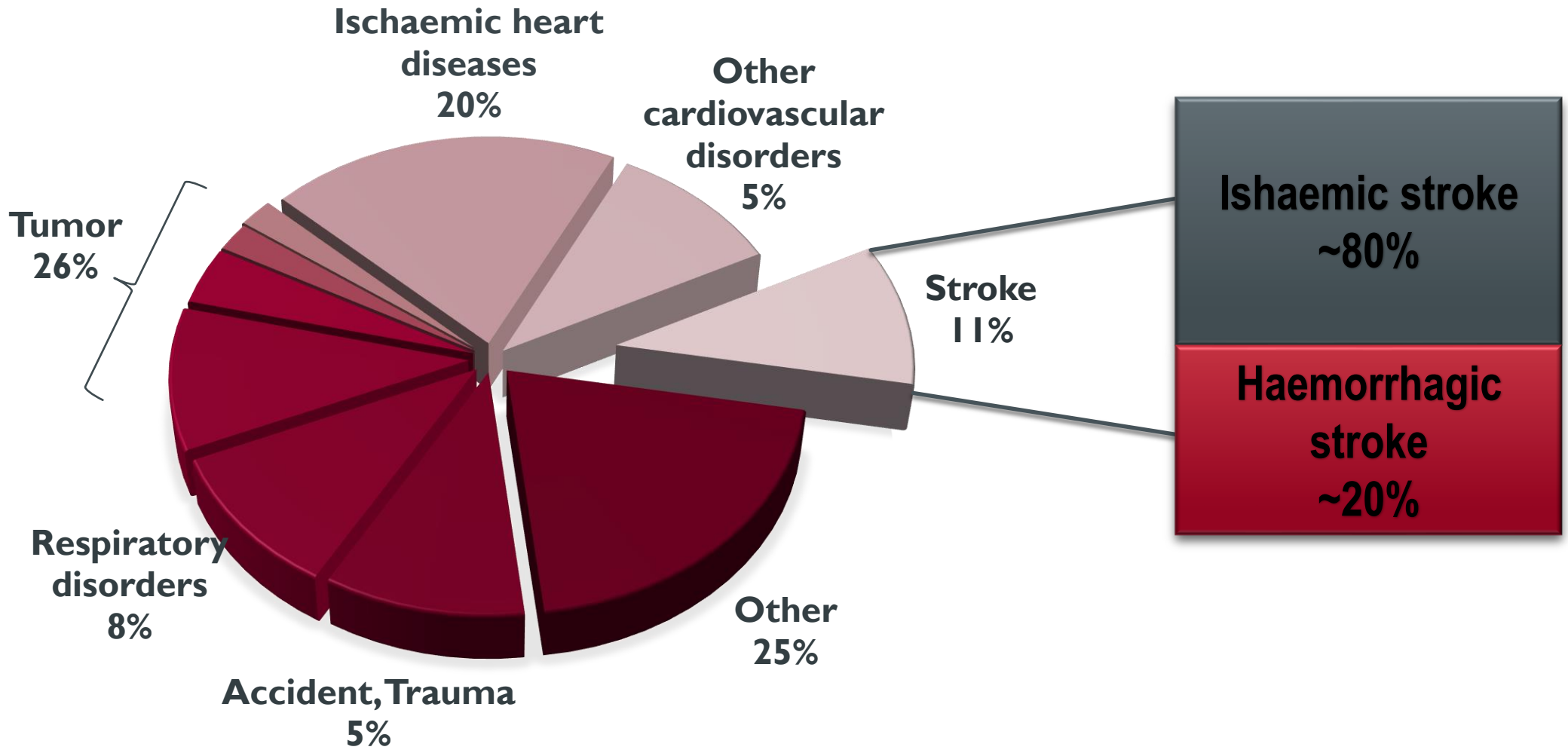
III. **Tumors**

IV. **Hematological disorders**





MORTALITY, EUROPEAN CARDIOVASCULAR DISEASE STATISTICS 2016



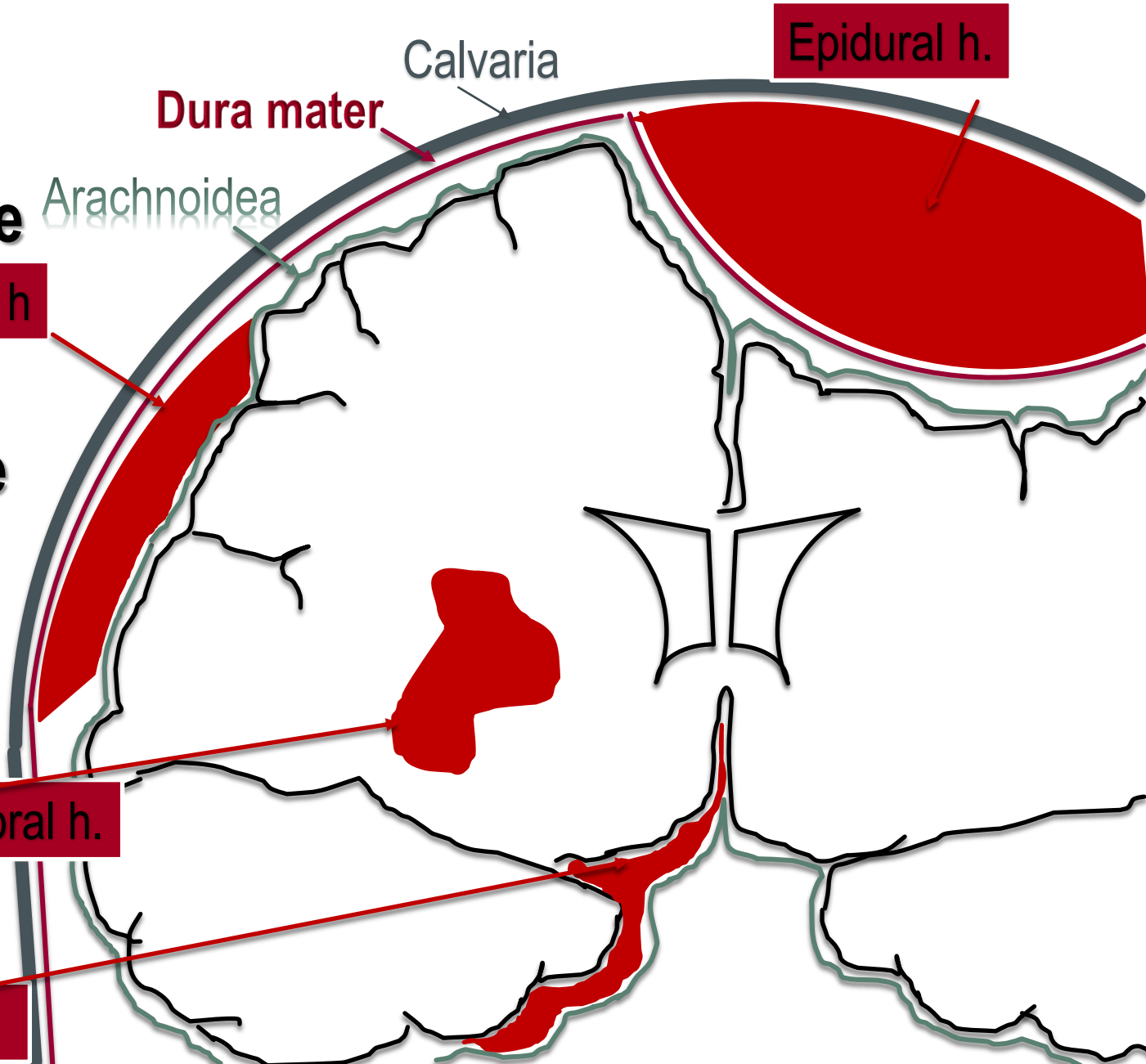
LOCATION:

I. **Intraparenchymal hemorrhage**
   - Hypertension
   - Tumor

II. **Subarachnoideal hemorrhage**
   - Aneurysma
   - Primary vascular lesion

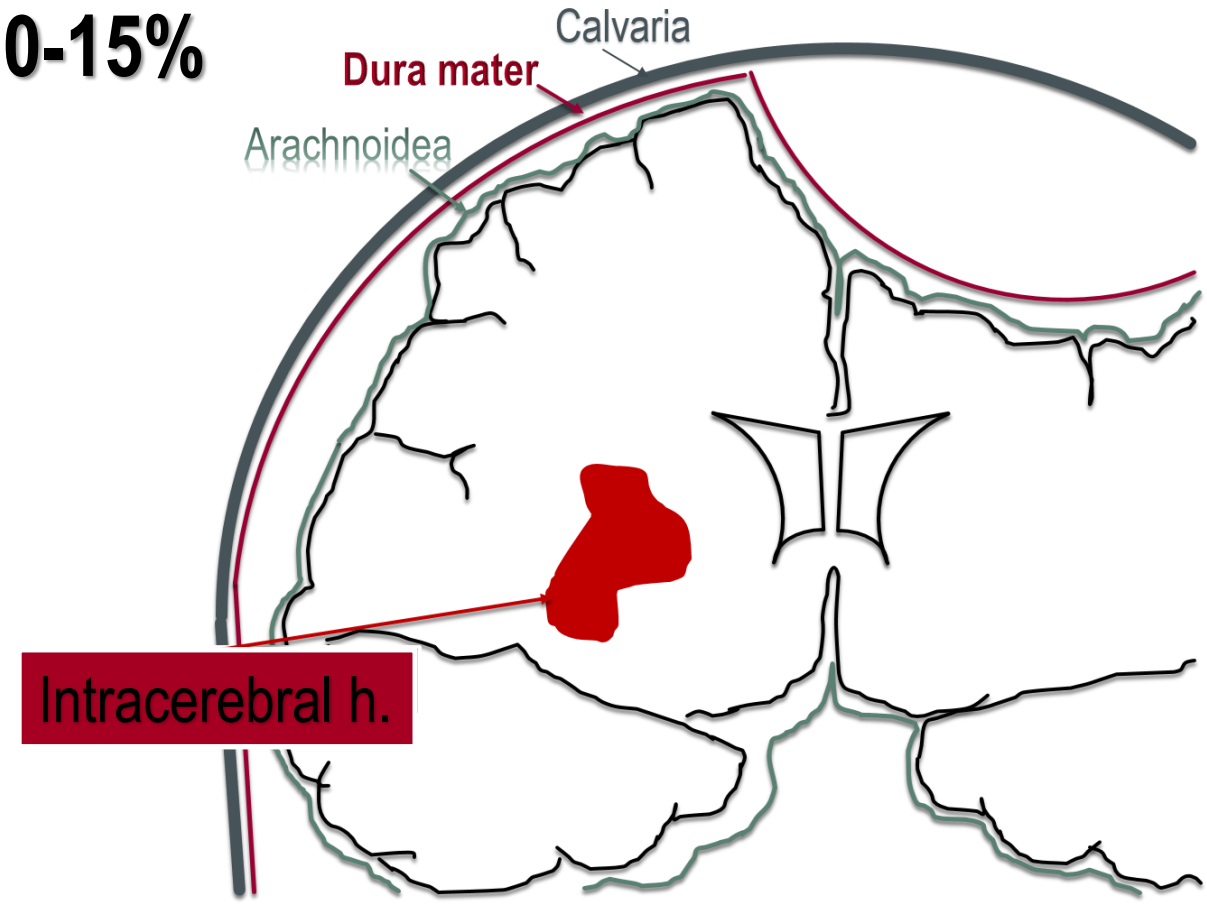
III. **Subdural hemorrhage**
   - Trauma

IV. **Epidural hemorrhage**
   - Trauma



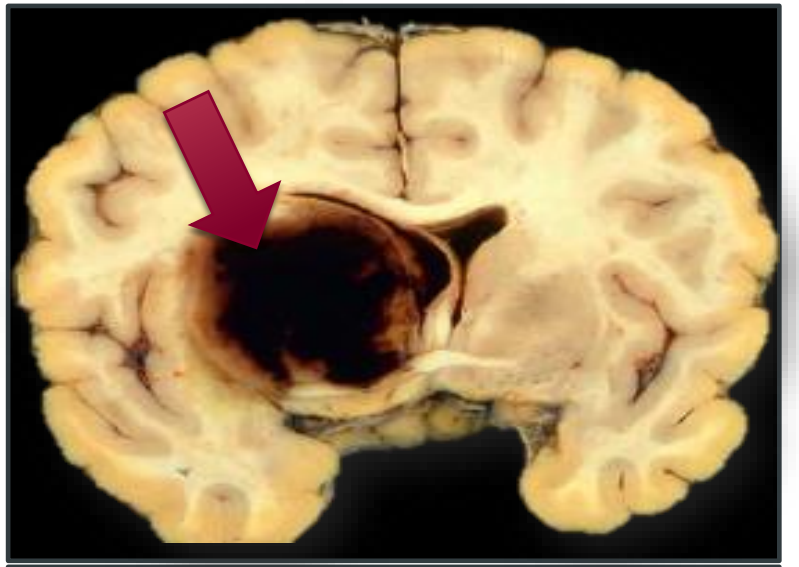
INTRAPARENCHYMAL BRAIN HEMORRHAGE

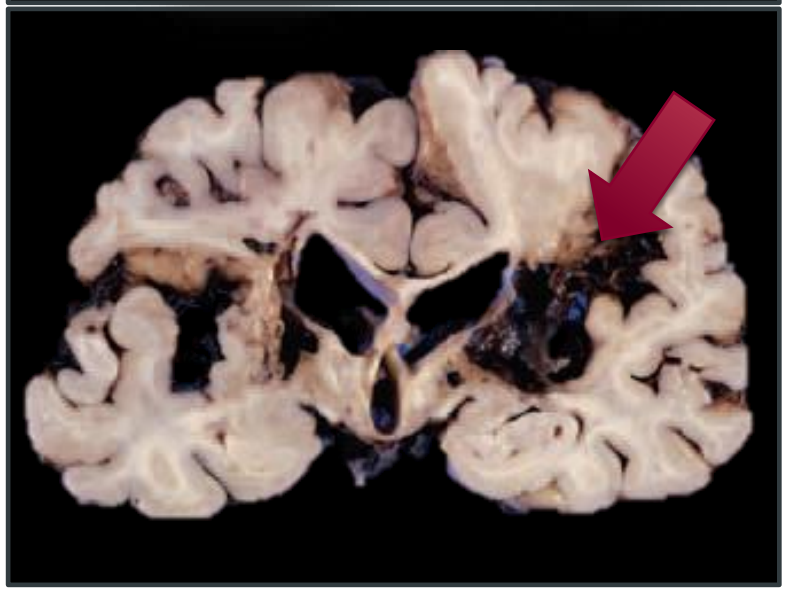
1. **Hypertension ~ 50%**
2. **Cerebral amyloid angiopathy ~10-15%**
3. **Tumor ~8-10%**
4. Vascular malformations ~5%
5. Trauma
6. Hematological disorders
7. Old age



Morphology

Time	Macroscopy	Microscopy
Seconds		Rupture
Minutes	Bright red hematoma	Extravasation
Hours	Perifocal edema, compression	Erythrocyte lysis edema, ischaemia
2-3 days - week	Brown hematoma	Hemosiderin-laden macrophages Astrocytosis
Weeks-Months	Friable brown hematoma	Organisation Phagocytosis of necrotic tissue
Months-1year	Cyst containing brownish liquid	Cyst lined with glial cells Residual hemosiderin Macrophages
Yers	Cyst with brownish wall containing clear liquid	Cyst lined with glial cells Residual hemosiderin Macrophages



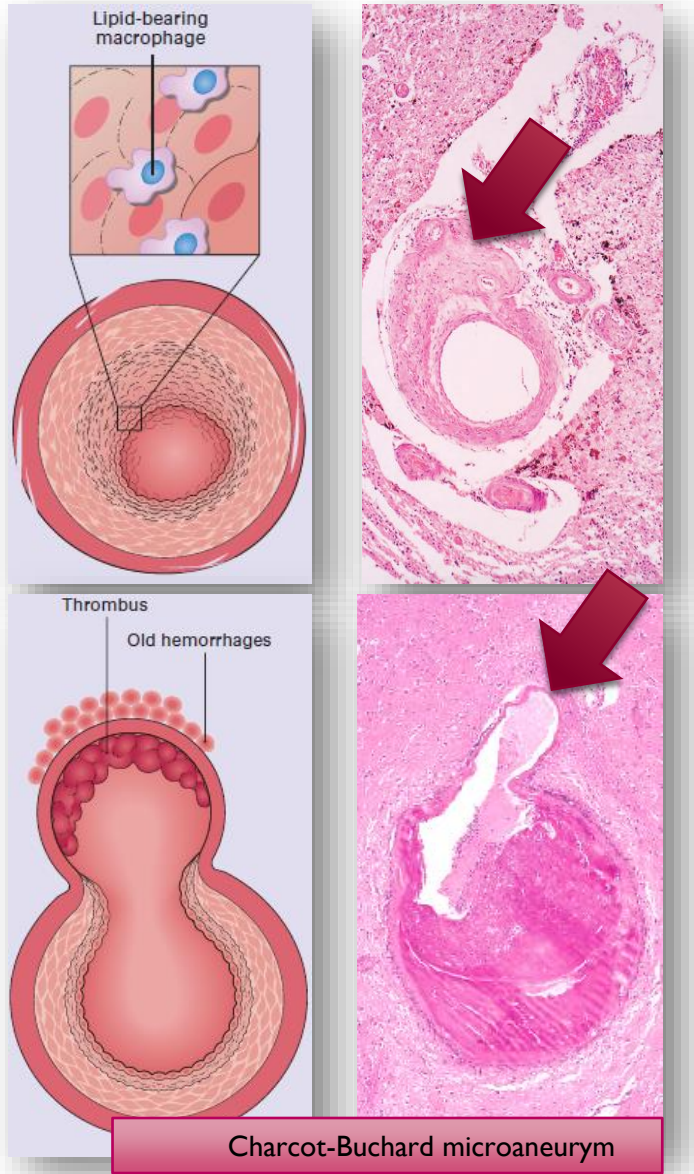


Hypertensive brain hemorrhage

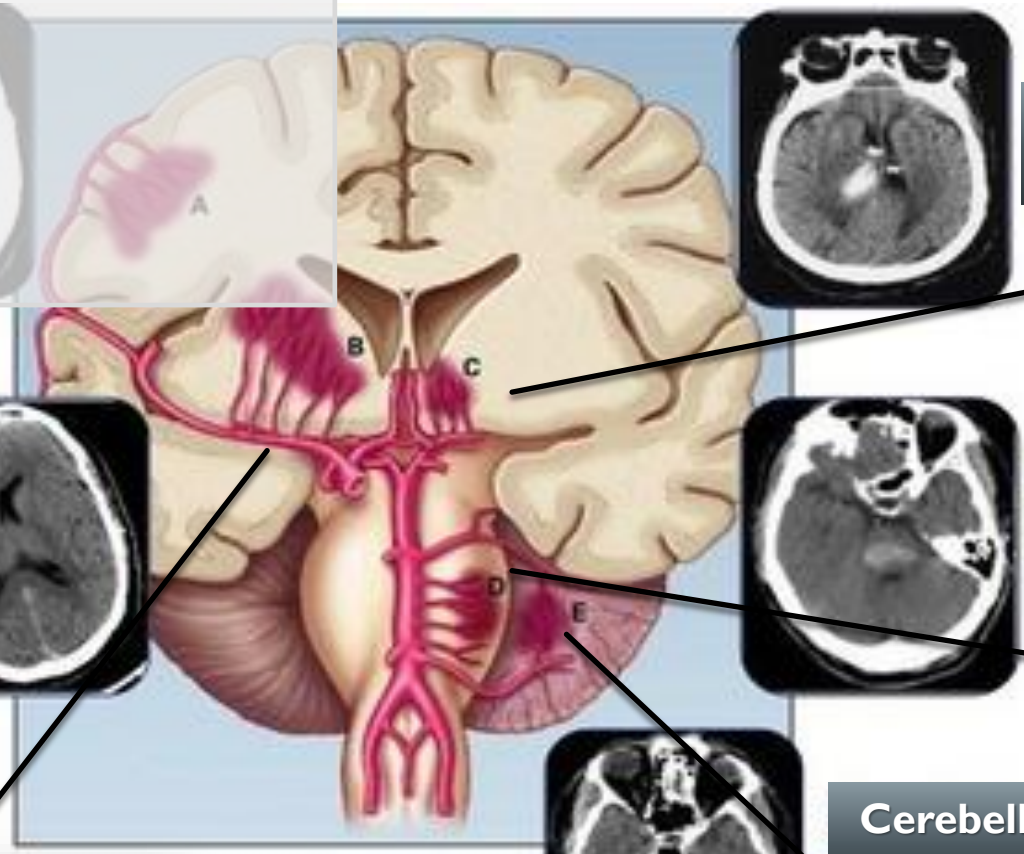
Intraparenchymal arteriolar walls are weakened

- Hyaline arteriolar sclerosis
- Fragmentation of elastica membrane

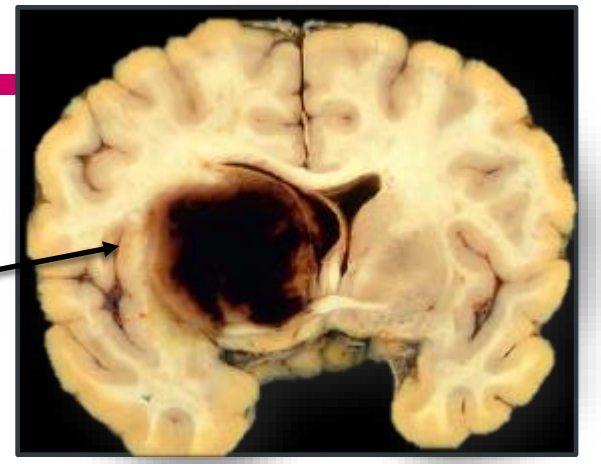
- Focal dilation of small vessels
  - Charcot-Buchard microaneuryms



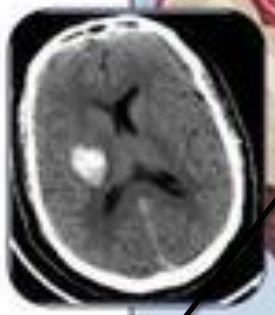
Lobaris Subcorticalis 25%

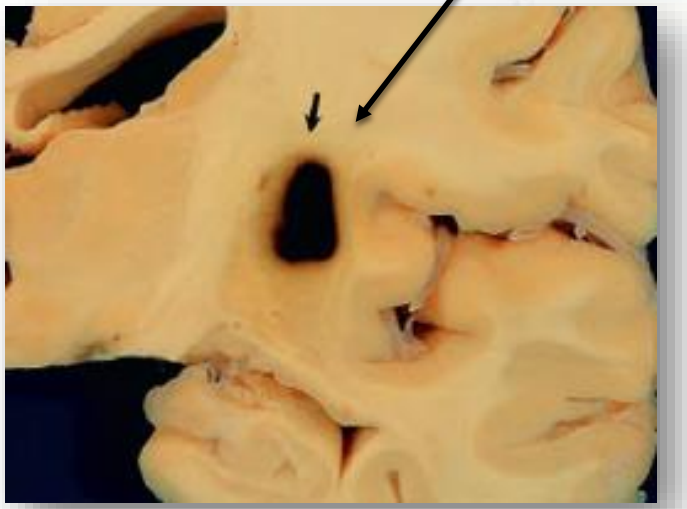


Thalamus 20%



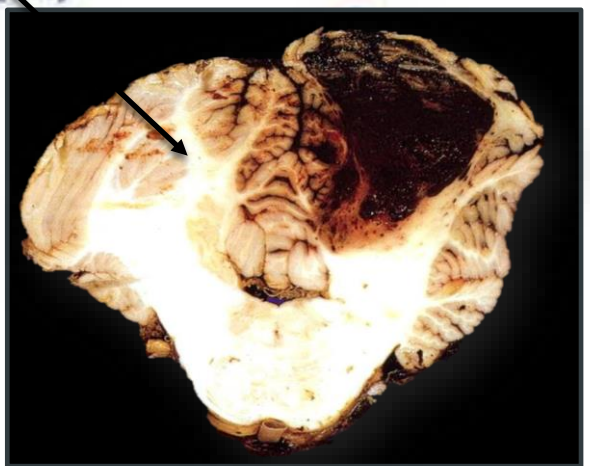
Putamen 35%





Pons 7%

Cerebellaris 8%



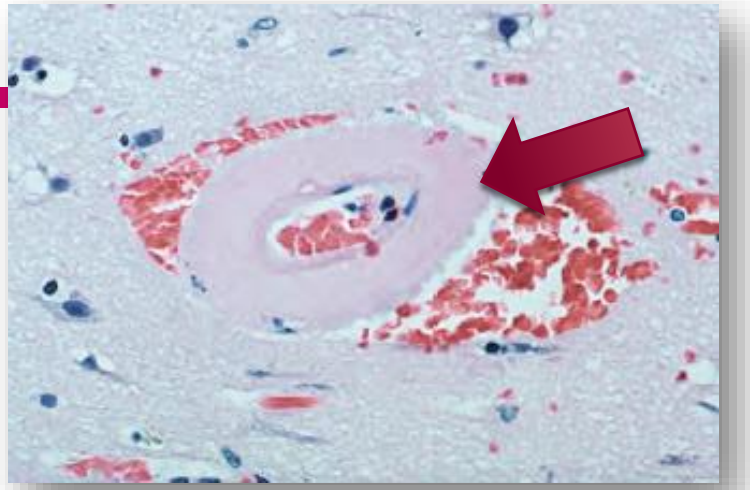
Cerebral amyloid angiopathy (CAA)

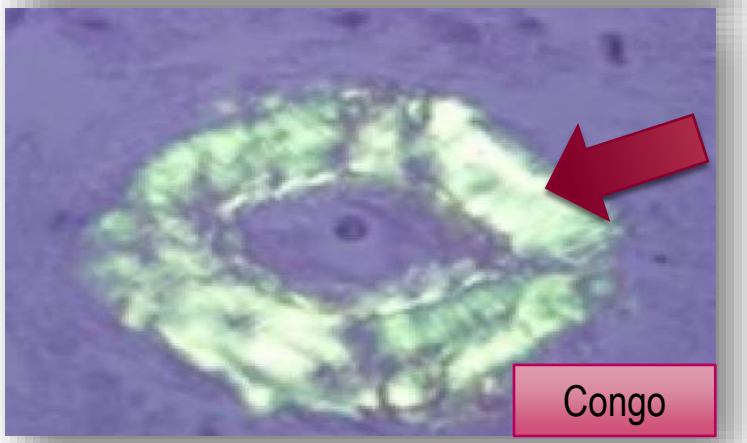
1. Sporadic/age-associated CAA

- Linked to Alzheimer's disease
- Aβ amyloid deposition
  - Disappearance of smooth muscle

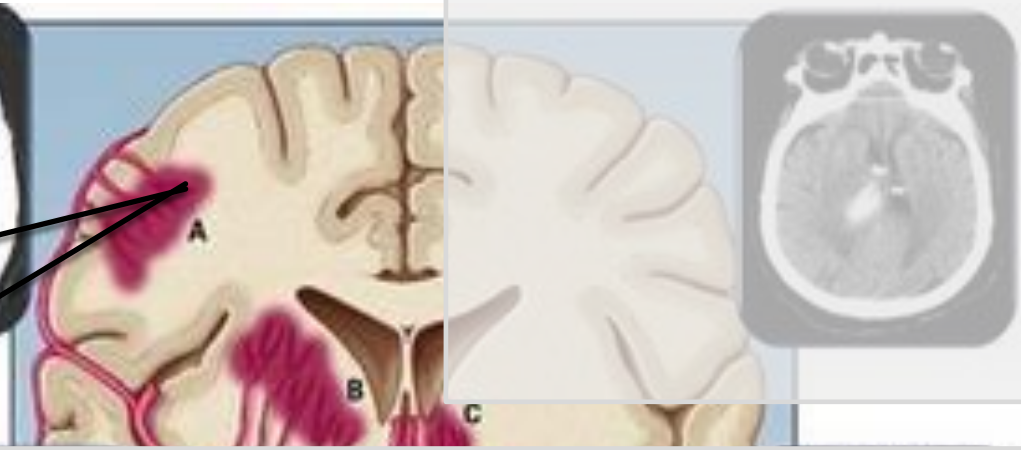
2. Familial CAA

- Younger age
- Autosomal dominant
- Amyloid precursor protein mutation

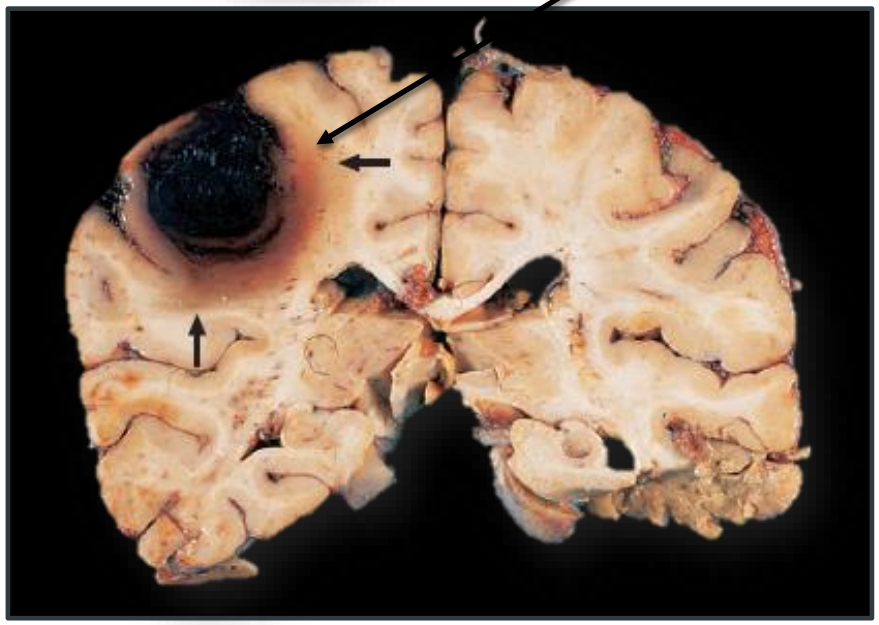


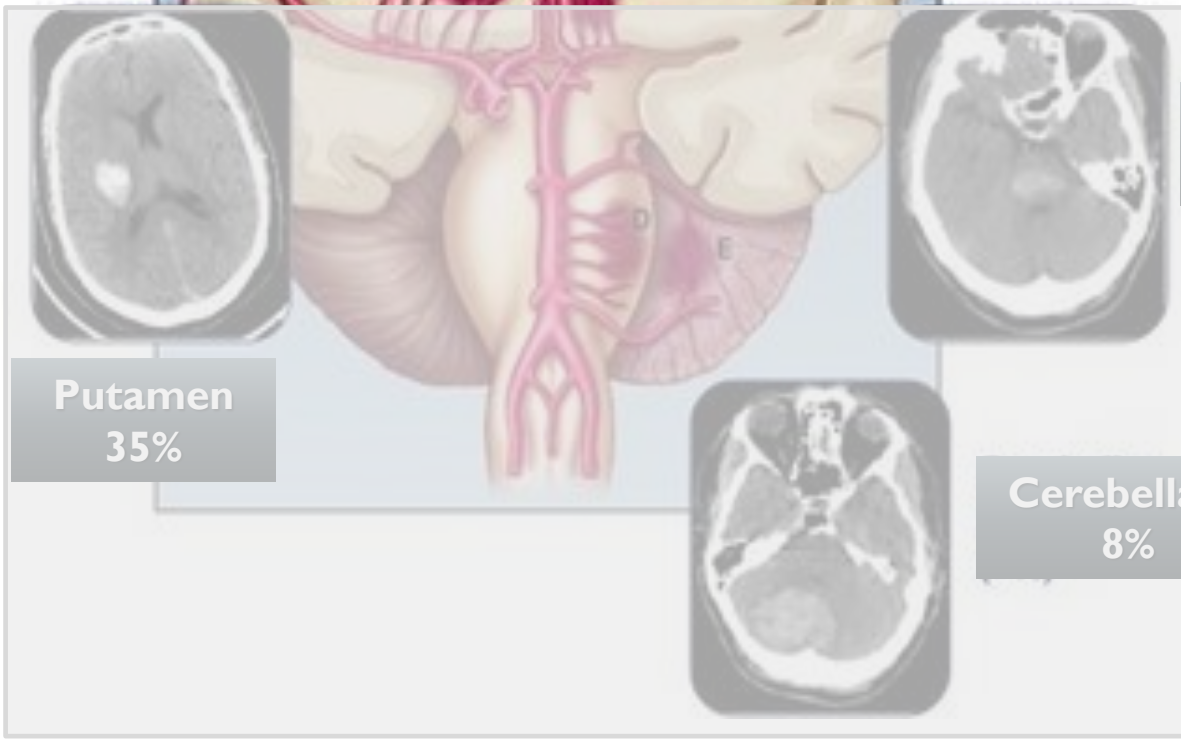


Lobar
Subcortical
25%



Thalamus
20%





Pons
7%

Putamen
35%

Cerebellaris
8%

Neoplastic lesions

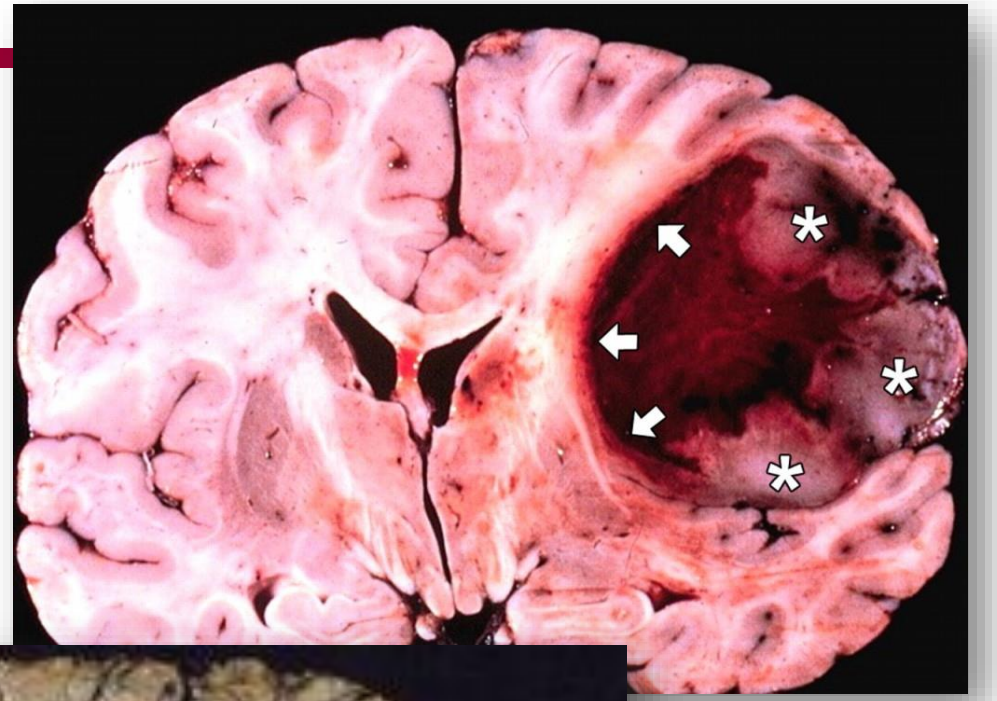
- No specific localisation
- Atypical, abnormal vessels

1. Primary CNS Tumors

- Glioblastoma
- Meningeoma
- Pituitary adenoma

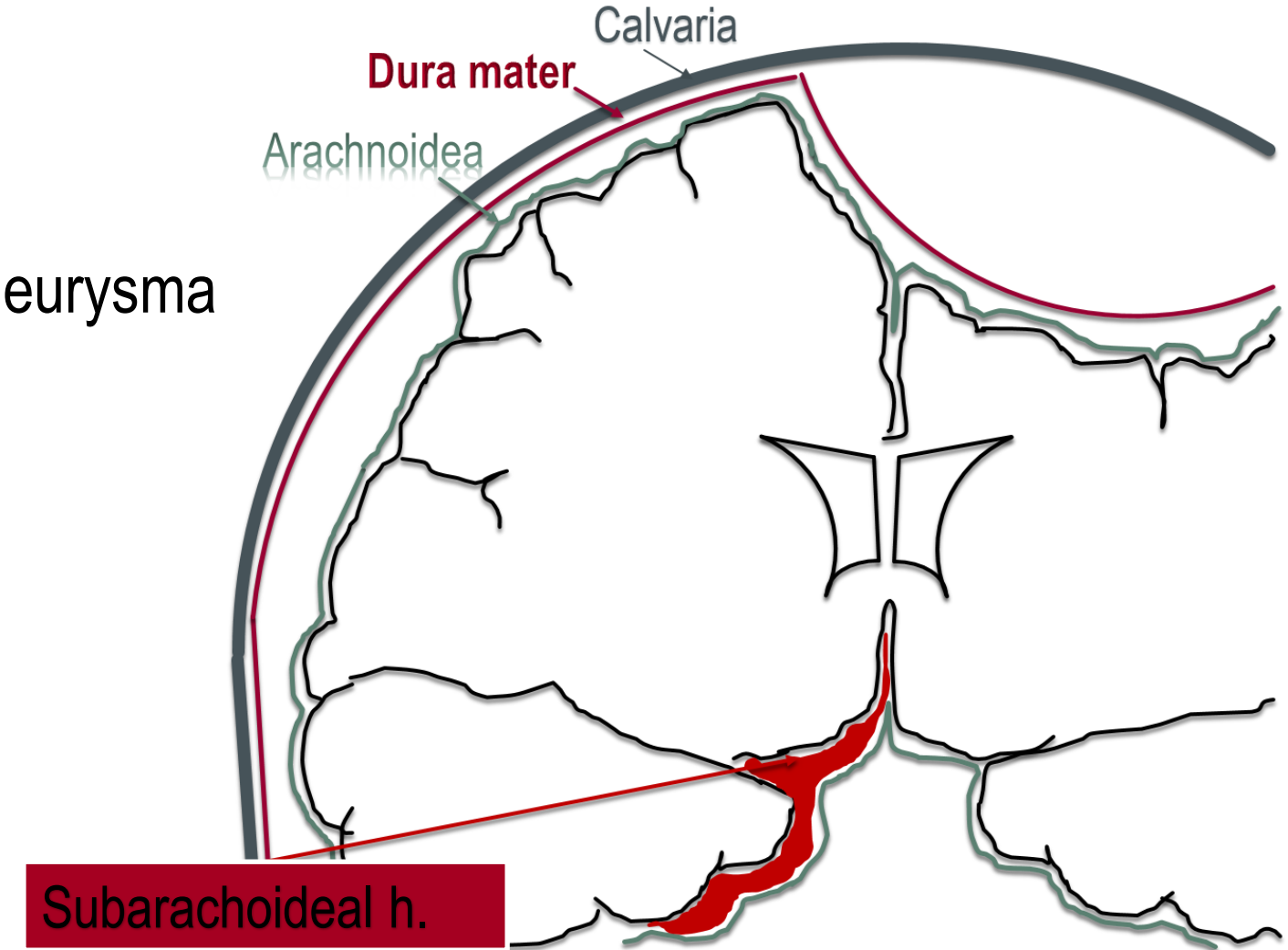
2. CNS metastasis

- Melanoma malignum
- Renal cell cancer



SUBARACHNOIDEAL HEMORRHAGE

1. **Aneurysm**
   - Saccular /berry/ aneurysma
   - Fusiform /atherosclerosis/ aneurysma
   - Infektív aneurysma
2. **Vascular malformations**
3. Trauma
4. Infections



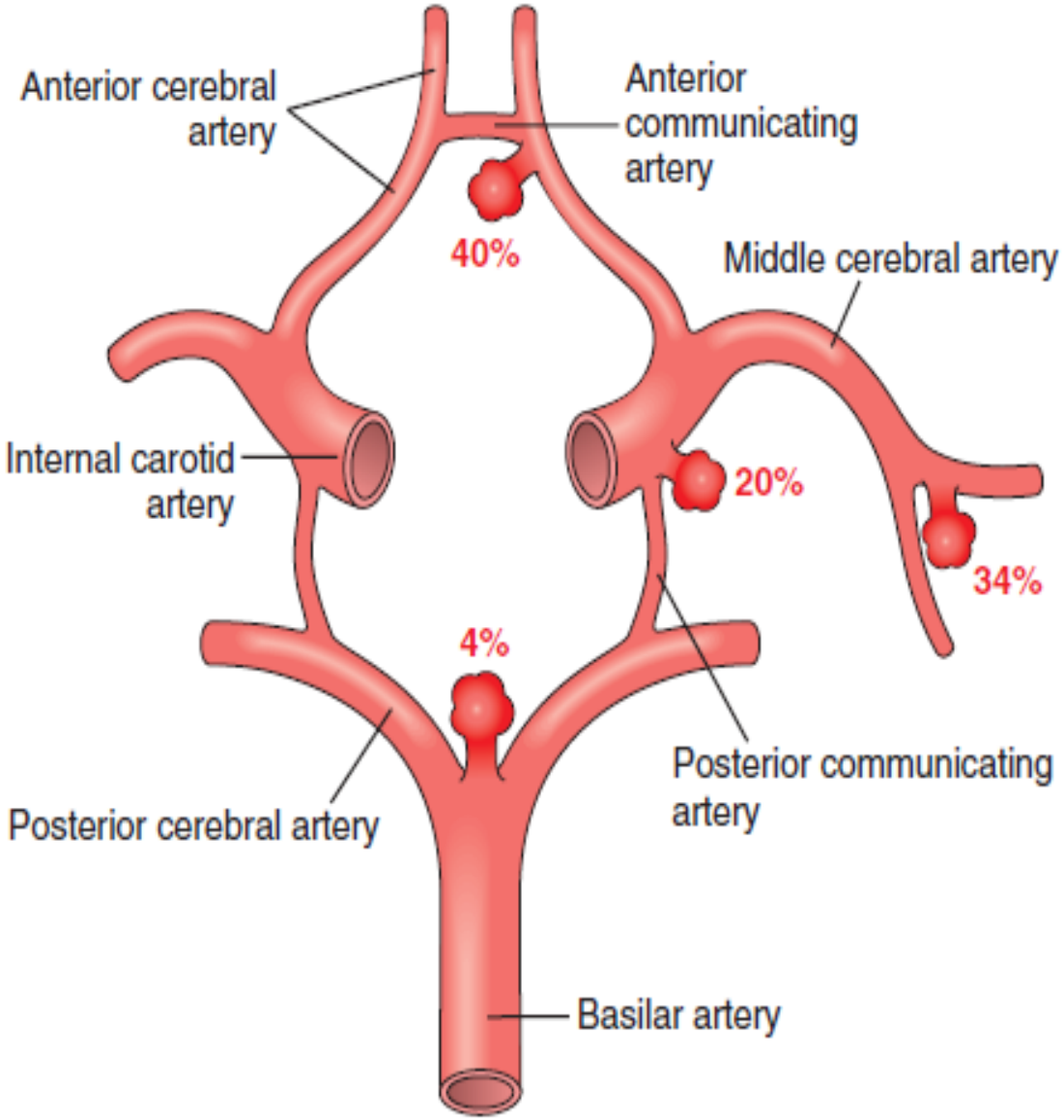
Subarachnoid Hemorrhage and Aneurysm

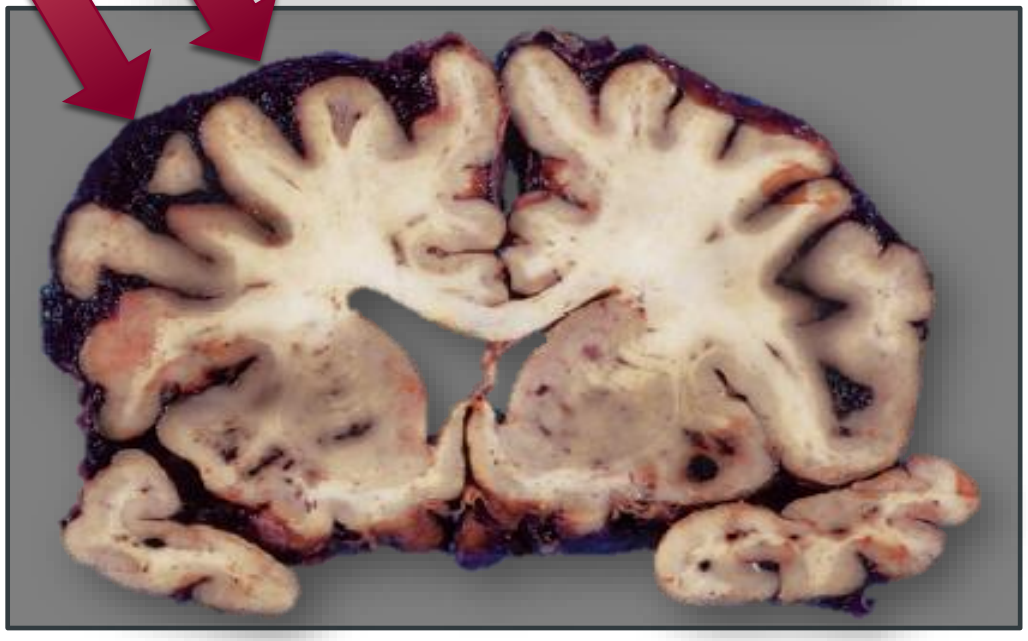
Saccular /Berry/ aneurysm

- Congenital – defects in the vessel media

- **Risk factors**
  - Hypertension
  - Systemic vasculopathy
  - Connective tissue diseases
  - Polycystic kidney disease

- Probability of rupture increasing with size


Anterior cerebral artery
Anterior communicating artery
40%
Middle cerebral artery
Internal carotid artery
20%
34%
4%
Posterior communicating artery
Posterior cerebral artery
Basilar artery

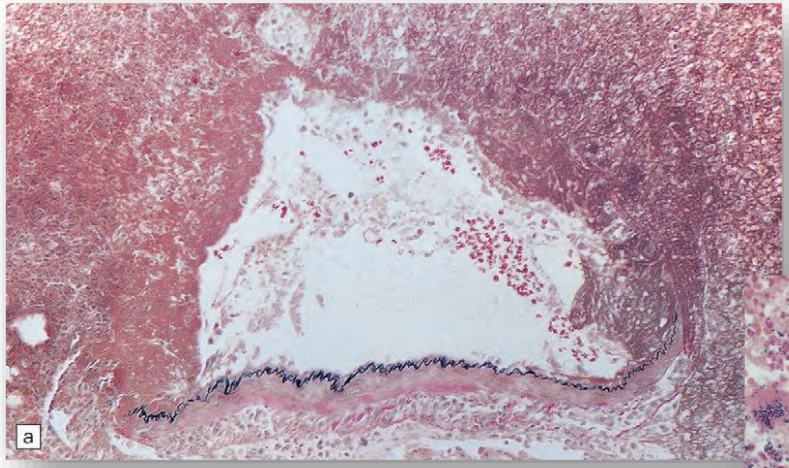


Fusiform aneurysm

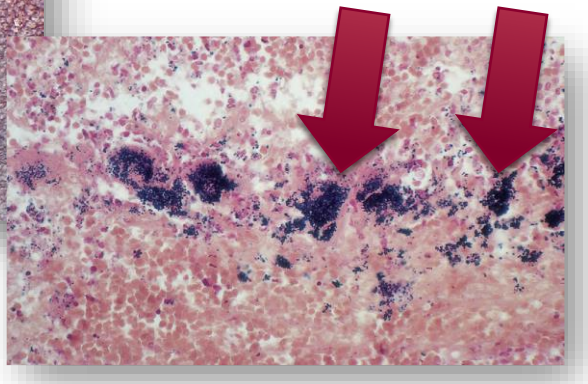
- Atherosclerotic origin
  - Most commonly involve basilar artery
- Complications
  - Cranial nerve compression
  - Occlusion
  - Rupture - Rare

Infective /mycotic/ aneurysm

- Inflammation weakens the vessel wall
- Bacterial, fungal infection
  - Hematogenous origin (Endocarditis)
  - Local infection (Meningitis)
- Distal branches of cerebral vessels

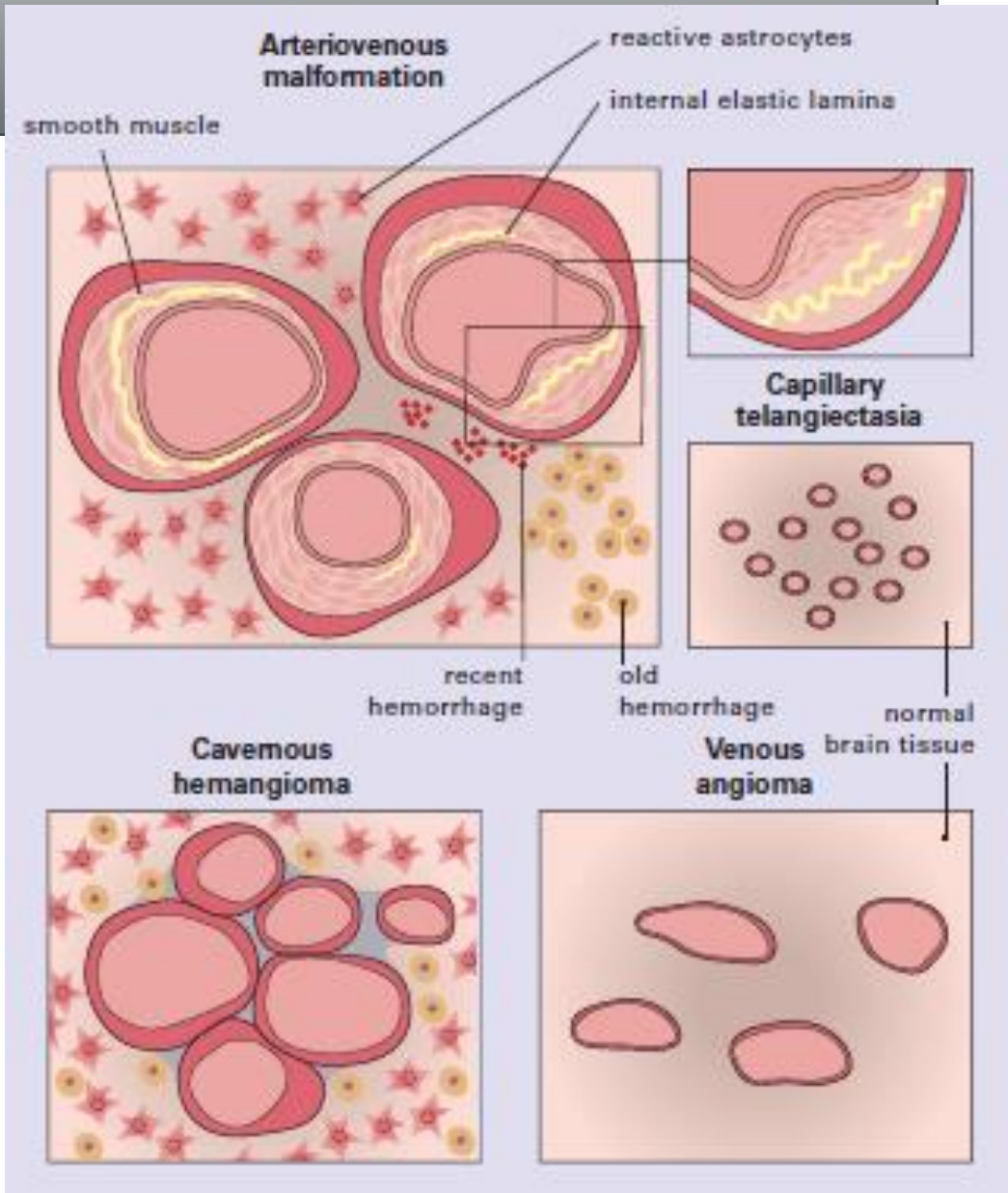


Bacteria



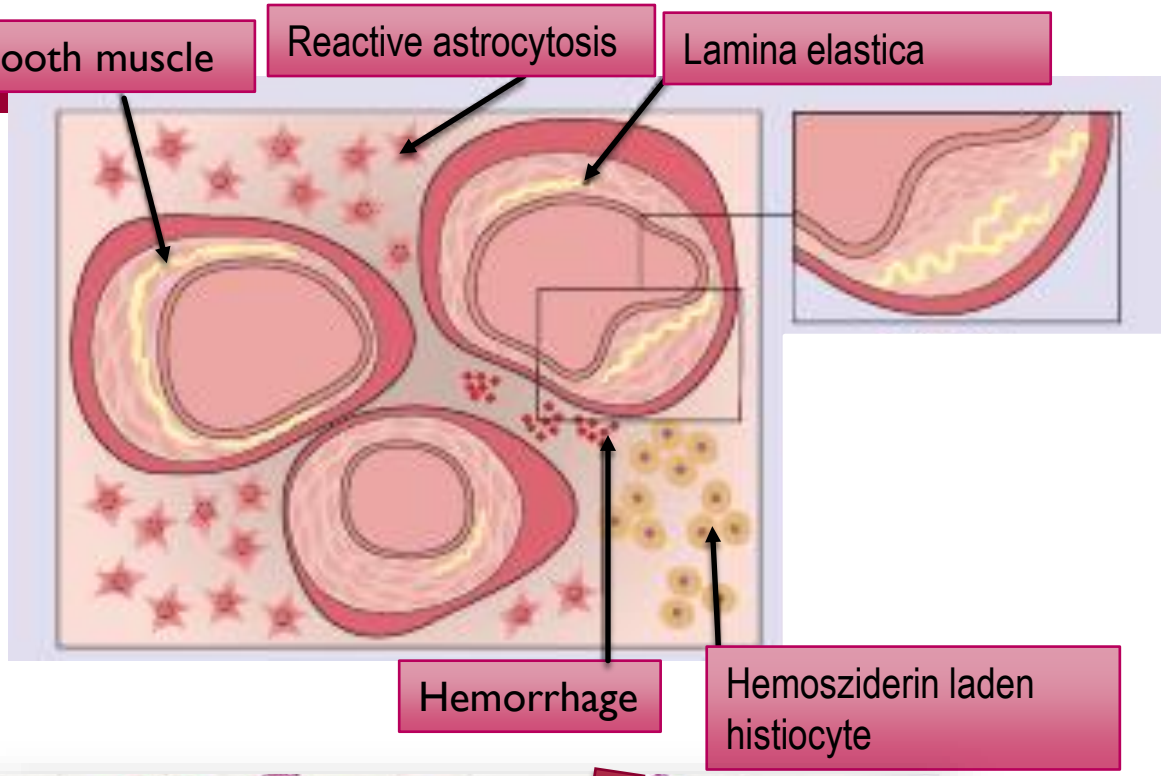
Vascular malformations

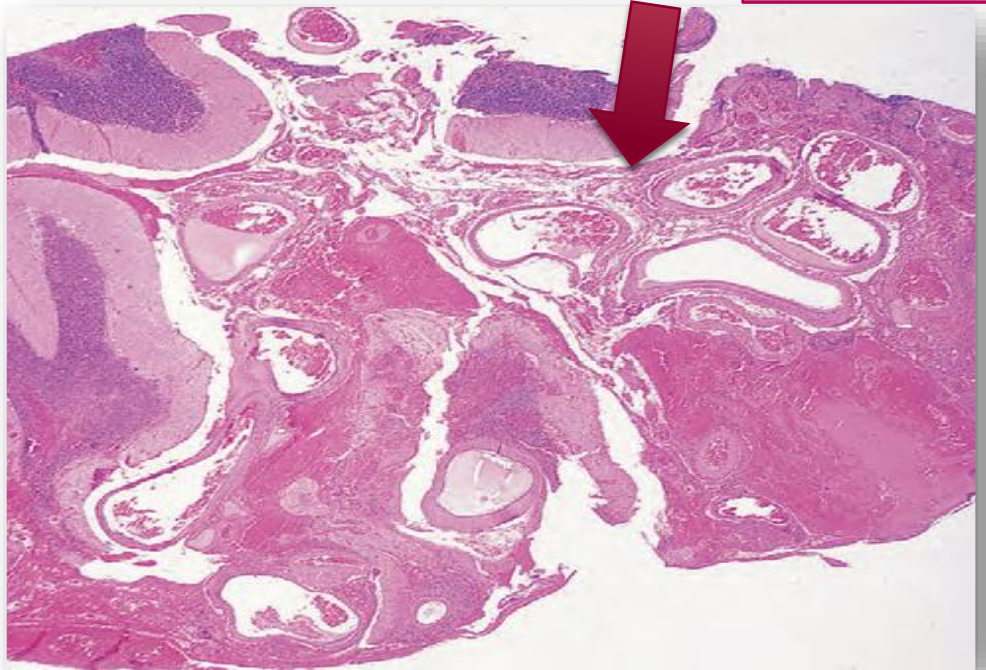
1. **Arteriovenous malformation (AVM)**
2. **Cavernous malformation**
3. Capillary telangiectasia
4. Venous angioma



Arteriovenous malformation

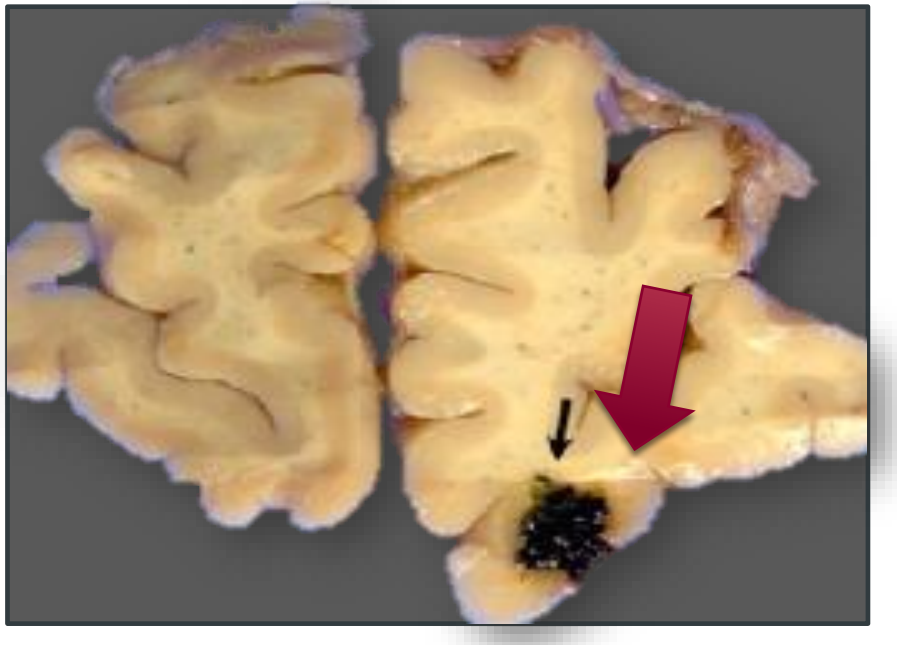
- Young adults, most commonly men
- Often associated with intracerebral hemorrhage
- Arteries, veins and abnormal vessels usually embedded within brain parenchyma

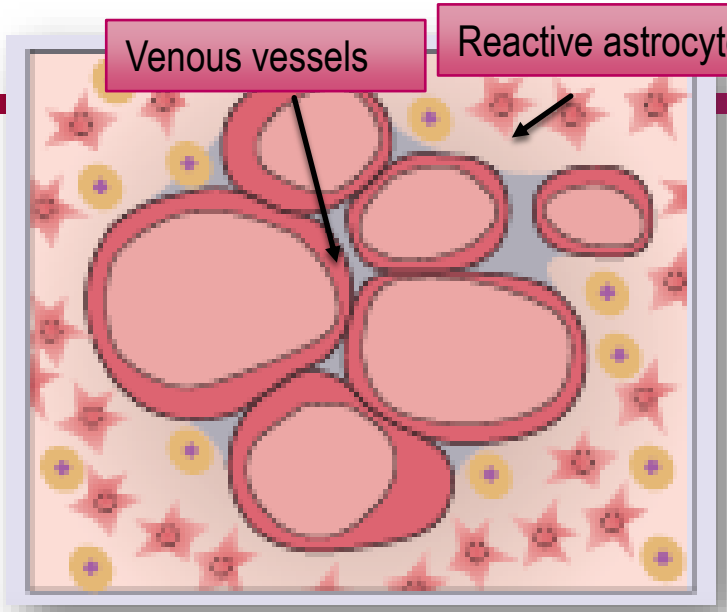


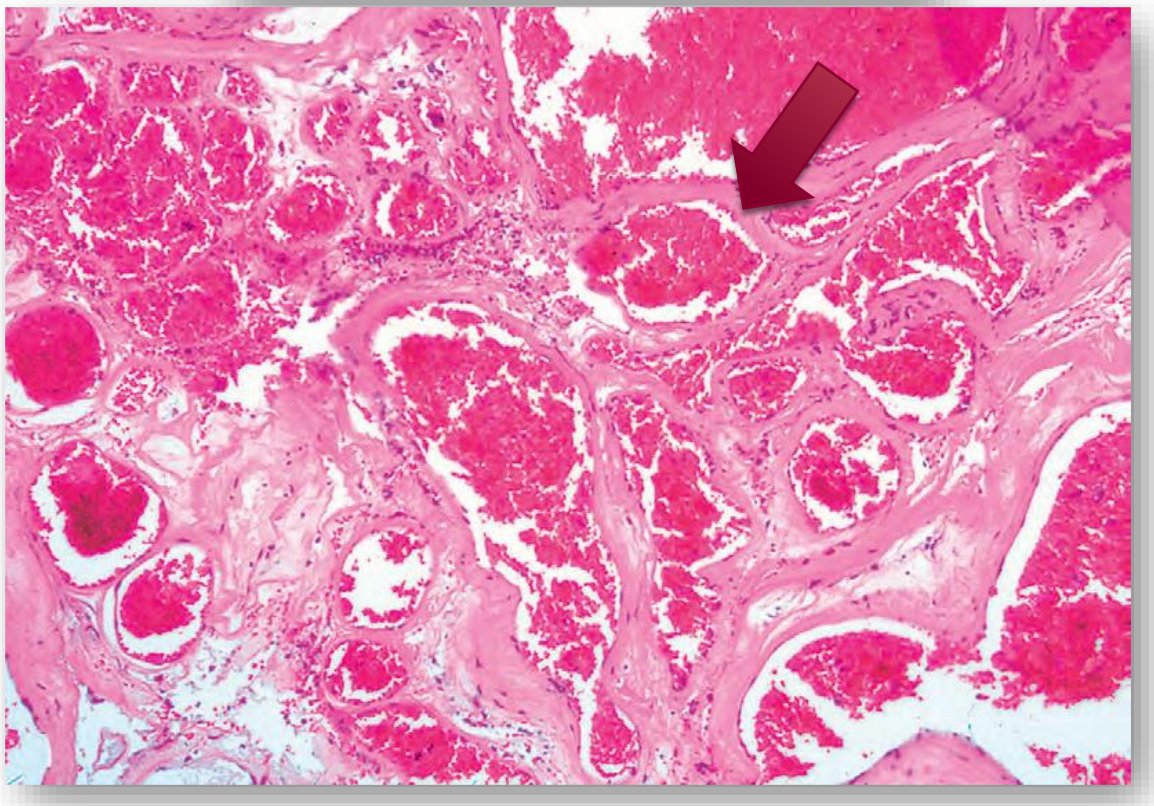


Cavernous hemangioma

- Familial disorder
- Often associated with intracerebral hemorrhage
- Tightly packed collection of hyalinized vessels
- Thin venous vessels

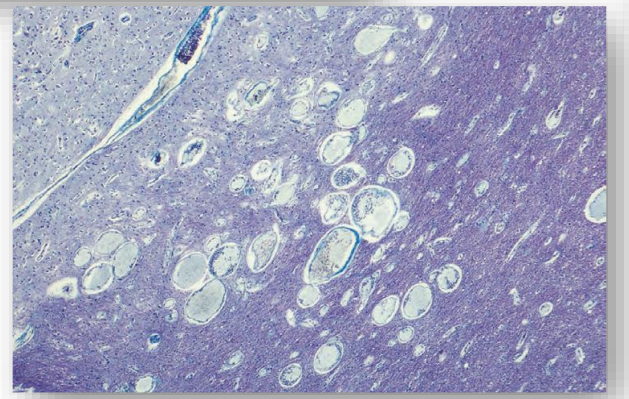


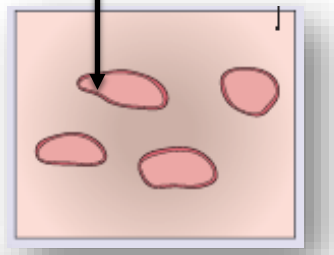




Venous angioma

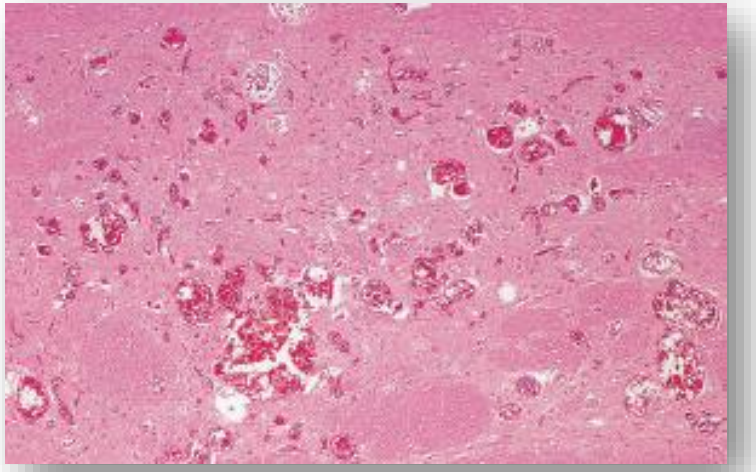
- Incidental – Rarely bleed
- Cerebellum
- Thin-walled, moderately dilated vascular channels within normal brain parenchyma

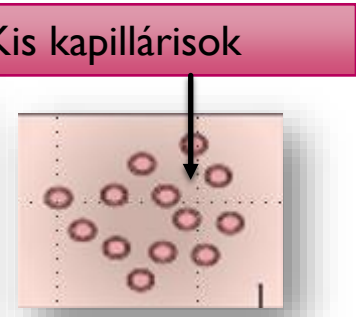




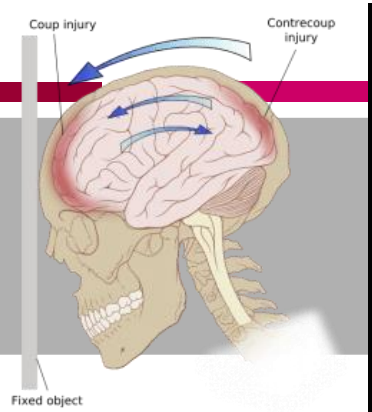
Capillary telangiectasia

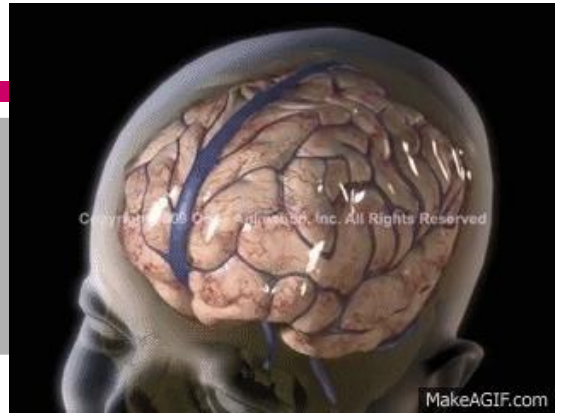
- Incidental – Rarely bleed
- Small-caliber thin-walled vascular channels surrounded by brain substance





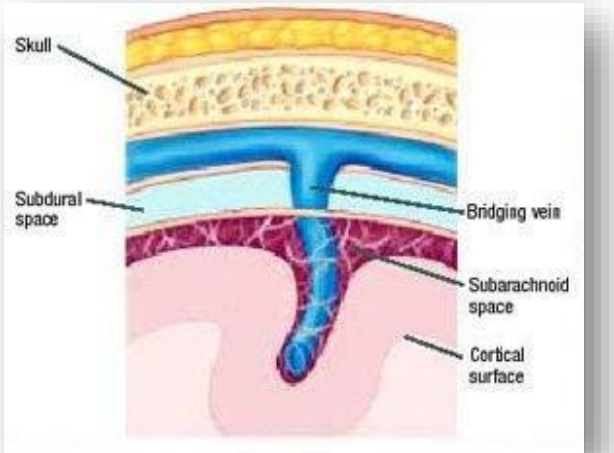
SUBDURAL HEMORRHAGE

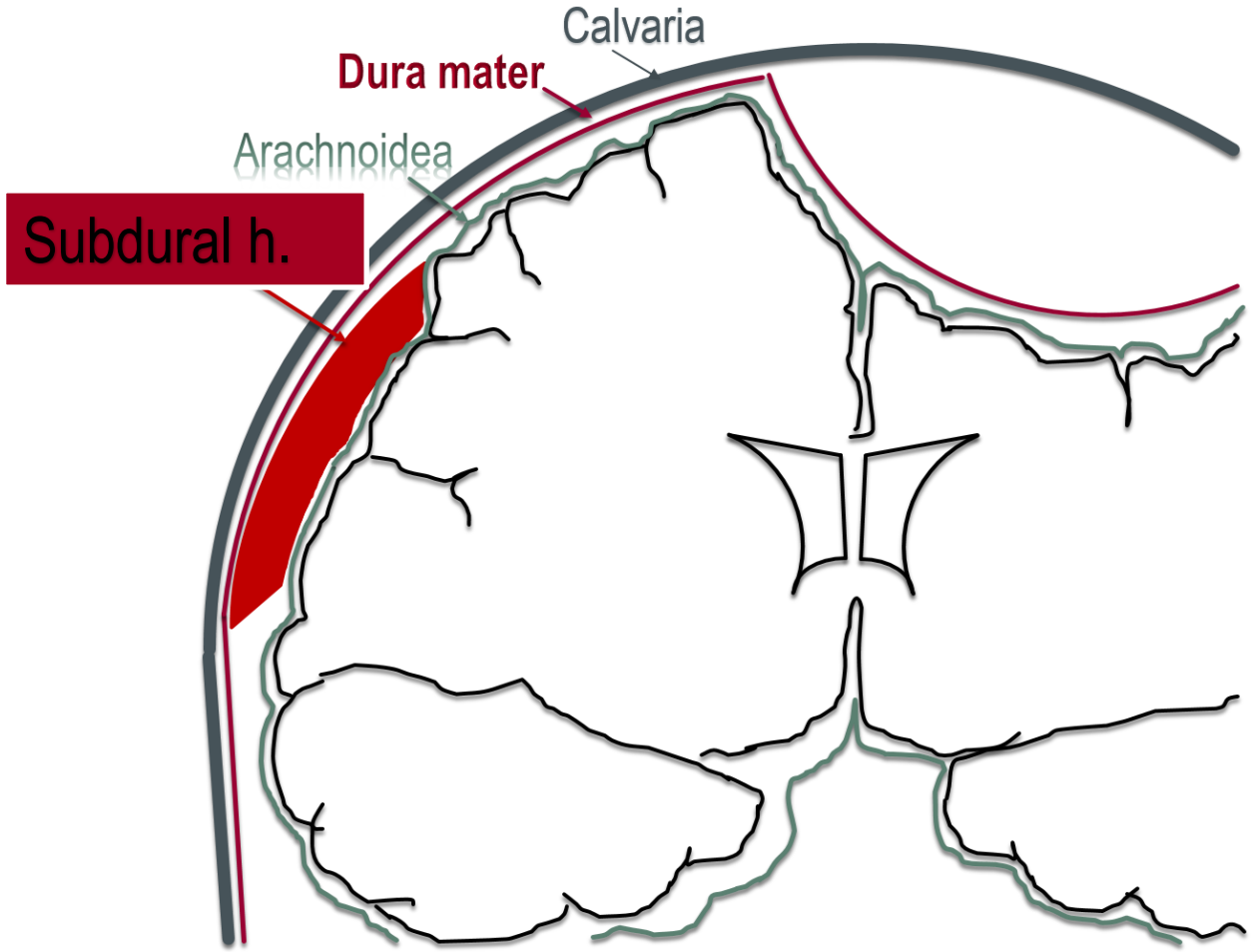




- Trauma!!!
  - Rapid acceleration or deceleration
- Increased risk
  - Elderly – Brain atrophy
  - Infants – thin walled bridging veins

- Rupture of bridging veins

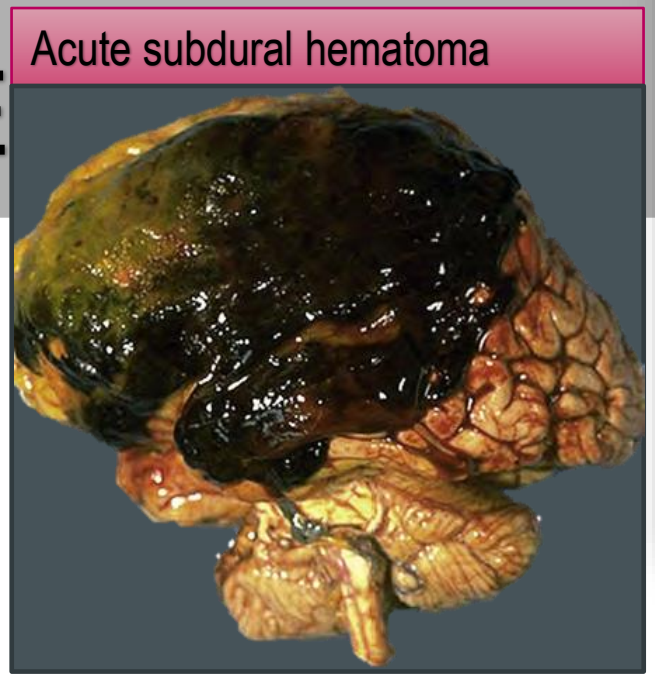


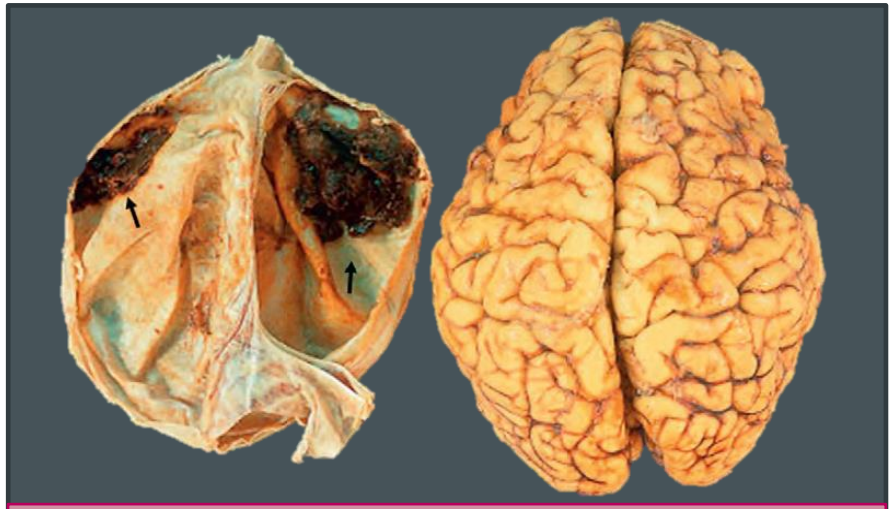


SUBDURAL HEMORRHAGE

- **Acut subduralis hematoma**
  - Slow progression of symptoms (manifest the first 48 hours)
  - Non-localizing symptoms
    - Headache
    - Slowly progressive neurologic deterioration

- **Chronic subdural hematoma**
  - Recidive trauma - Rebleeding
  - Older patients
  - Dementia –Alzheimer disease


Acute subdural hematoma

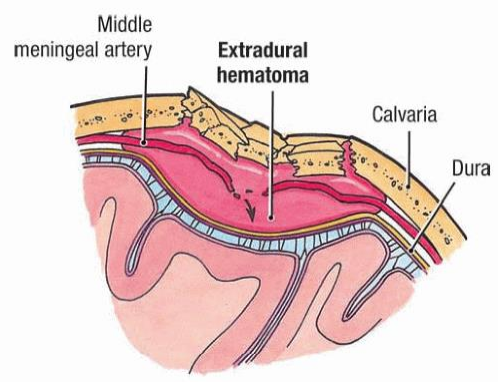

Chronic subdural hematoma

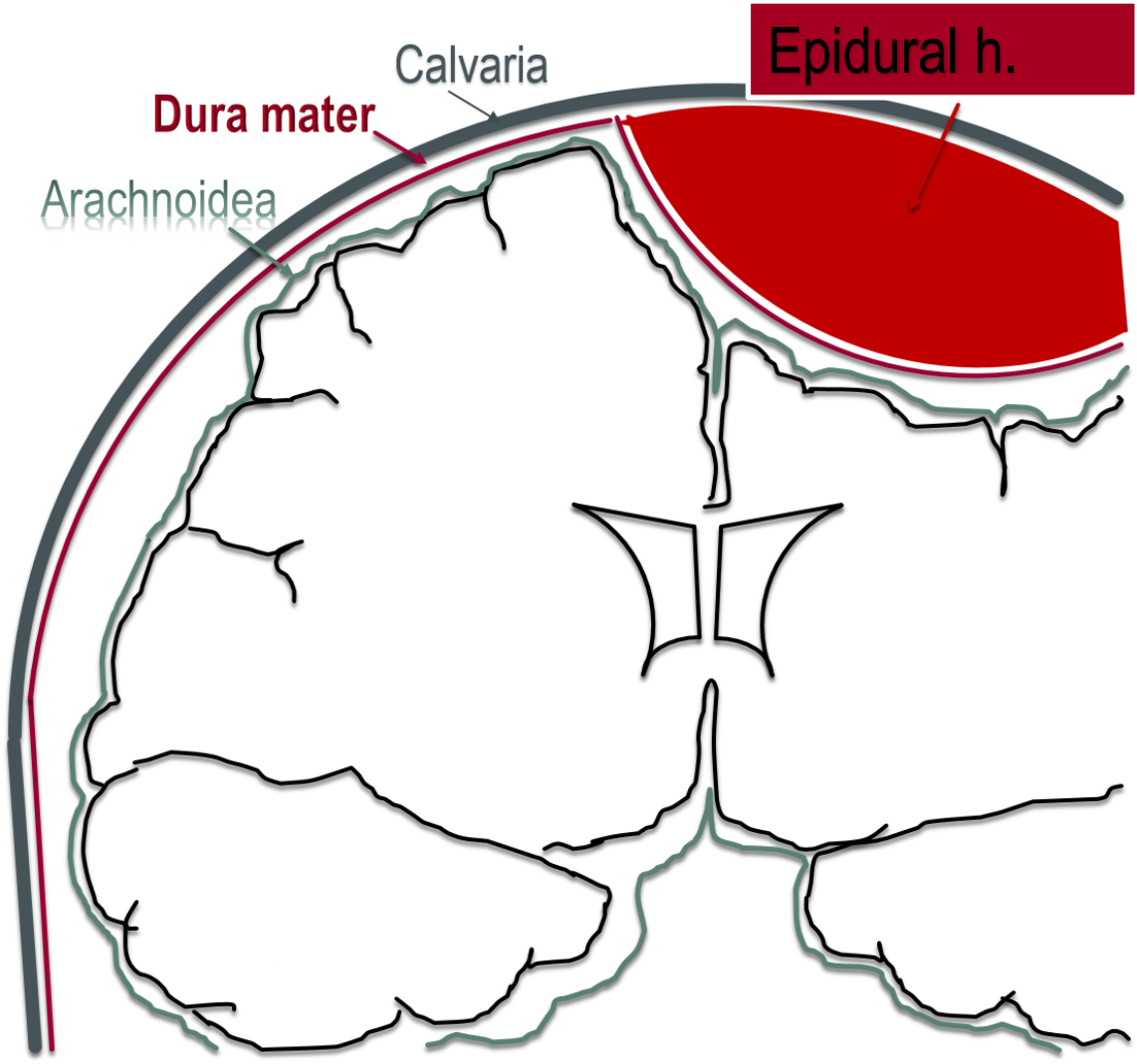
EPIDURAL HEMORRHAGE

- Trauma!!!

- Skull fracture
  - Middle meningeal artery
  - Sagittal, lateral sinus (rare)

- Adults – fossa temporalis
- Infants, children – fossa posterior

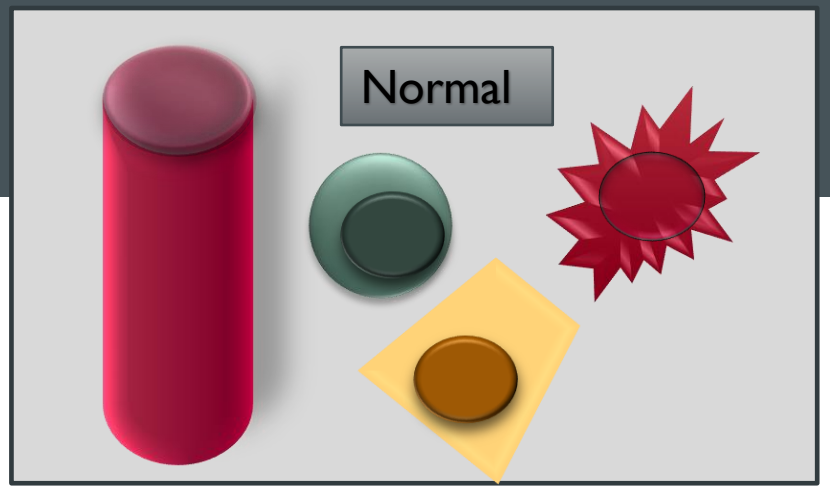




EPIDURAL HEMORRHAGE

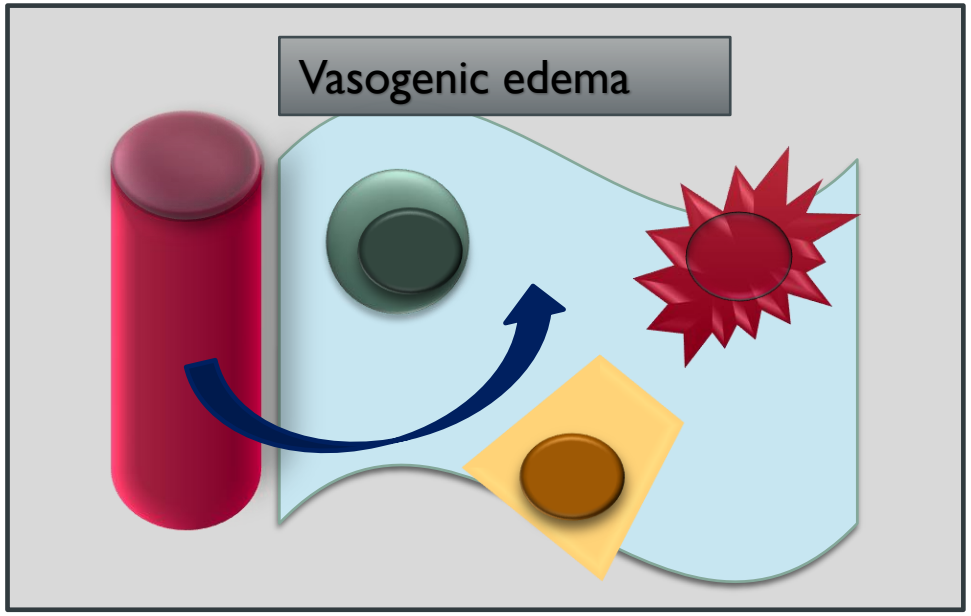
- Arterial bleeding ~ 75-100 ml fatal
- Lucid Interval !
- Rapid progression

CEREBRAL EDEMA



Vasogenic edema

- **Fluid shift to the extracellular compartment**
- Blood-brain barrier damage
  - Hemorrhage
  - Infarction
  - Tumor
  - Inflammation
- Affects predominantly the white matter
- Localised-generalised

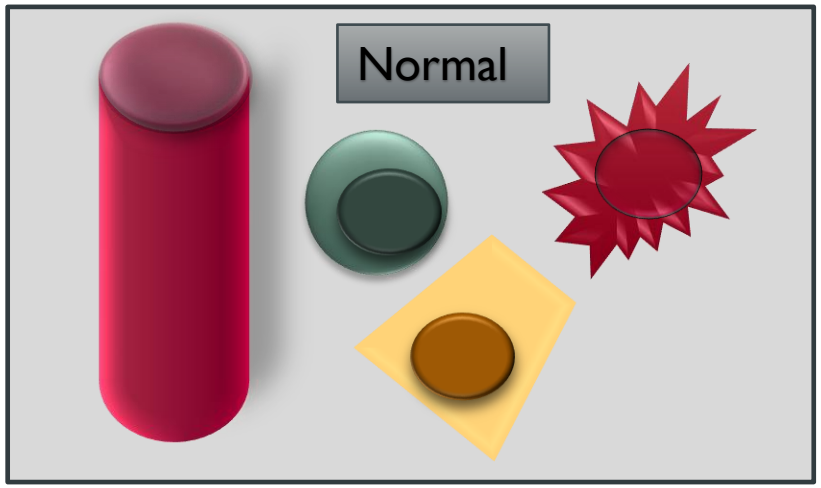


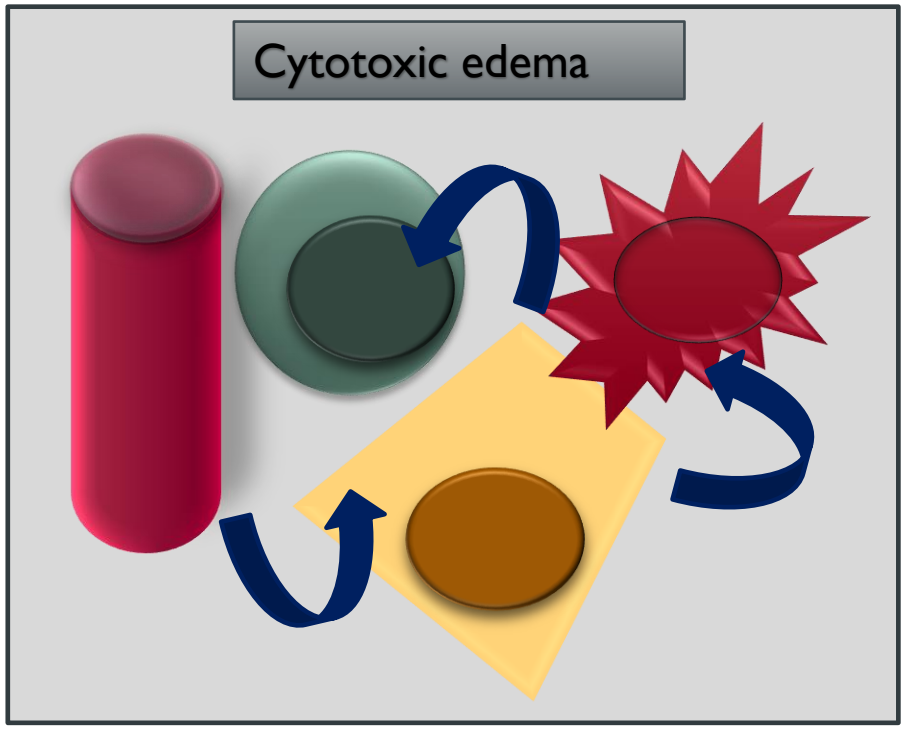
Cytotoxic edema

- **Intracellular fluid accumulation**
  - Neuron, glial cells, endothel
  - Cell membrane injury

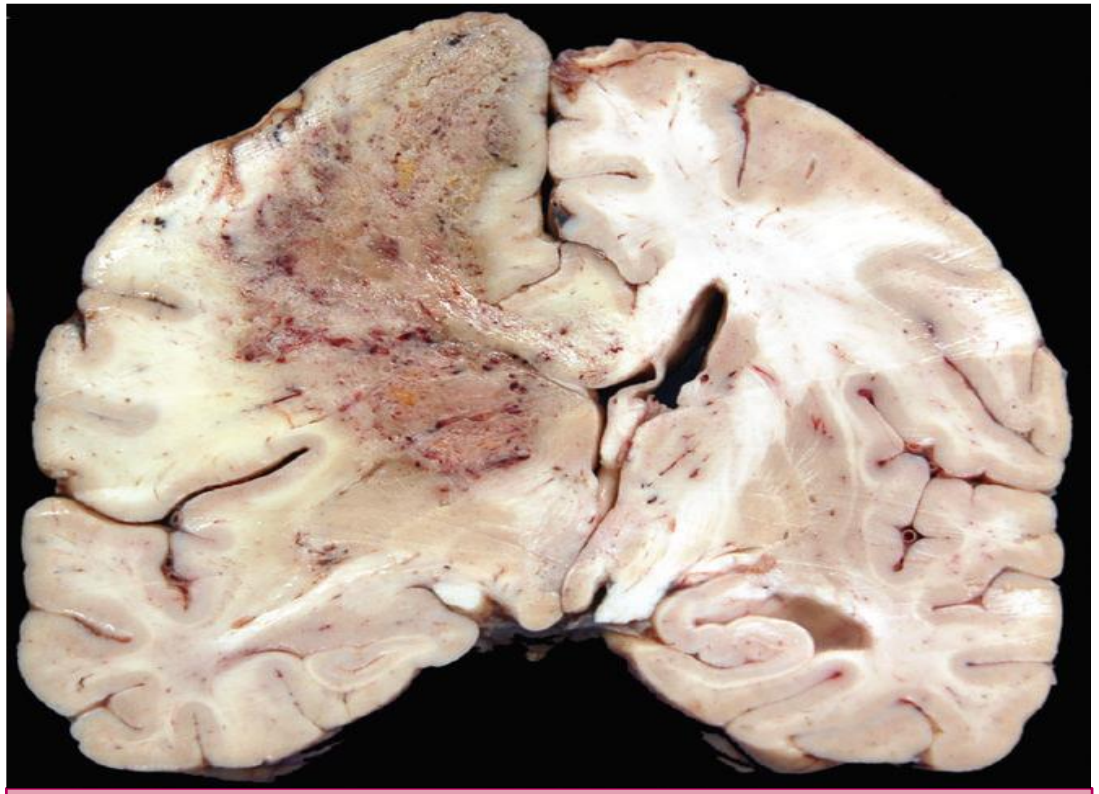
- Cause :
  - Hypoxic-ischaemic insult
  - Toxin
  - Hypoosmolar states
    - Kidney failure
  - Diabetic ketoacidosis

- Affects the white and grey matter



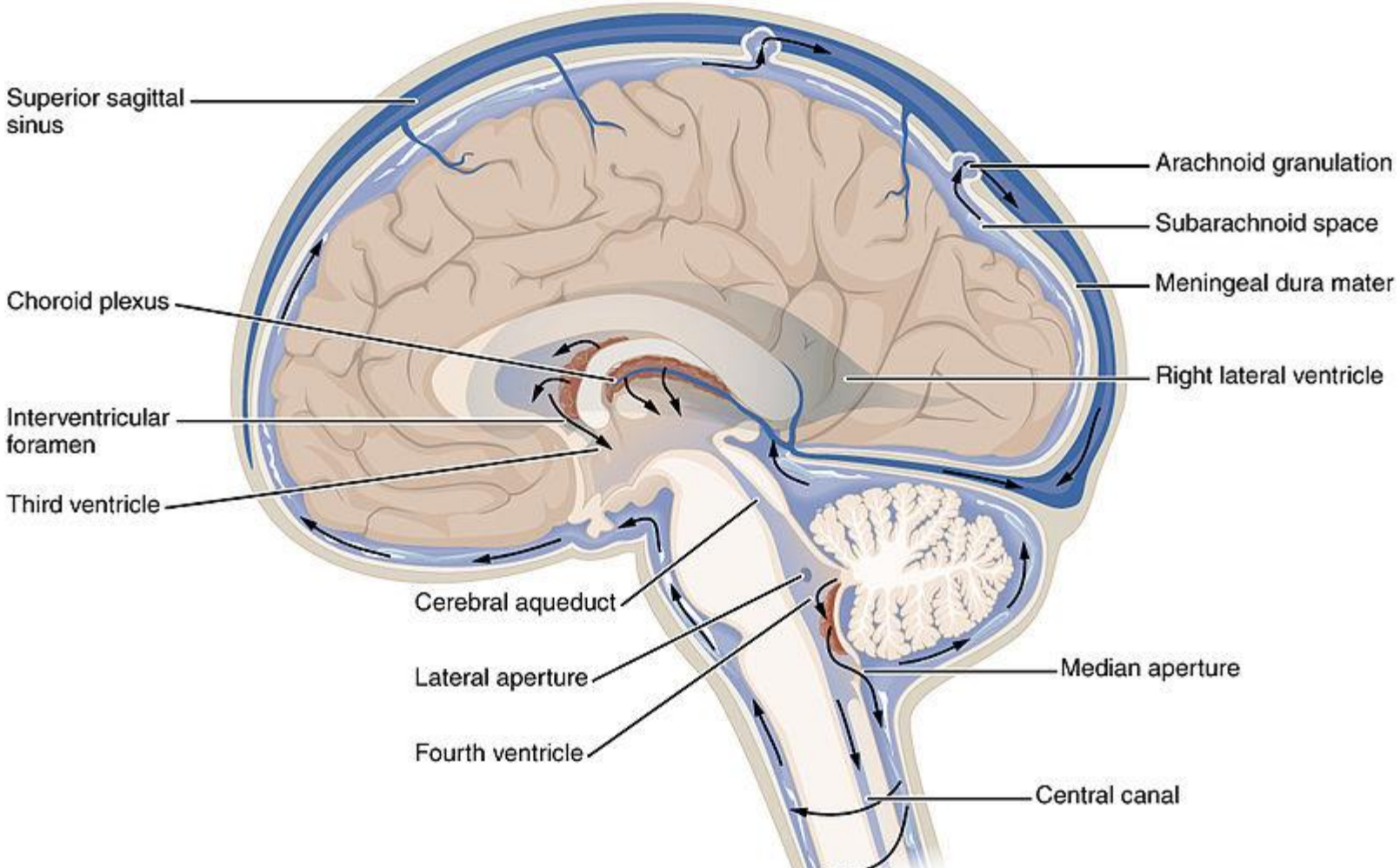


Generalised edema

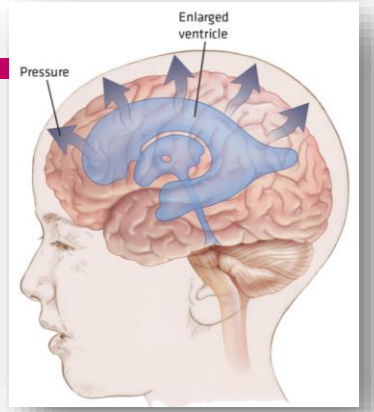


Lokalised edema

HYDROCEPHALUS



Hydrocephalus:



- **The accumulation of excessive CSF within the ventricular system**

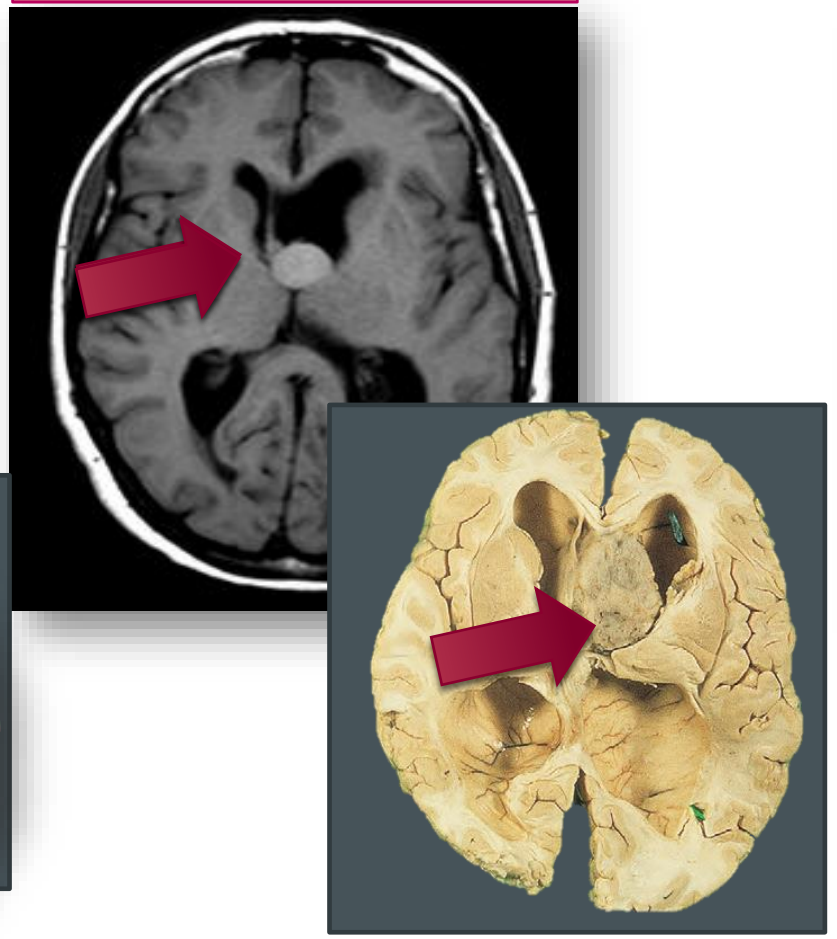
1. **Communicating hydrocephalus**
   - Impaired CSF production or absorption - Rare
     - Overproduction – Choroid plexus tumor
     - Impared resorption – Lack of arachnoideal granulations, Sinus thrombosis

2. **Non-communicating hydrocephalus**
   - Impaired CSF circulation
     - Cause: Malformation, Tumor, Hemorrhage, Infection

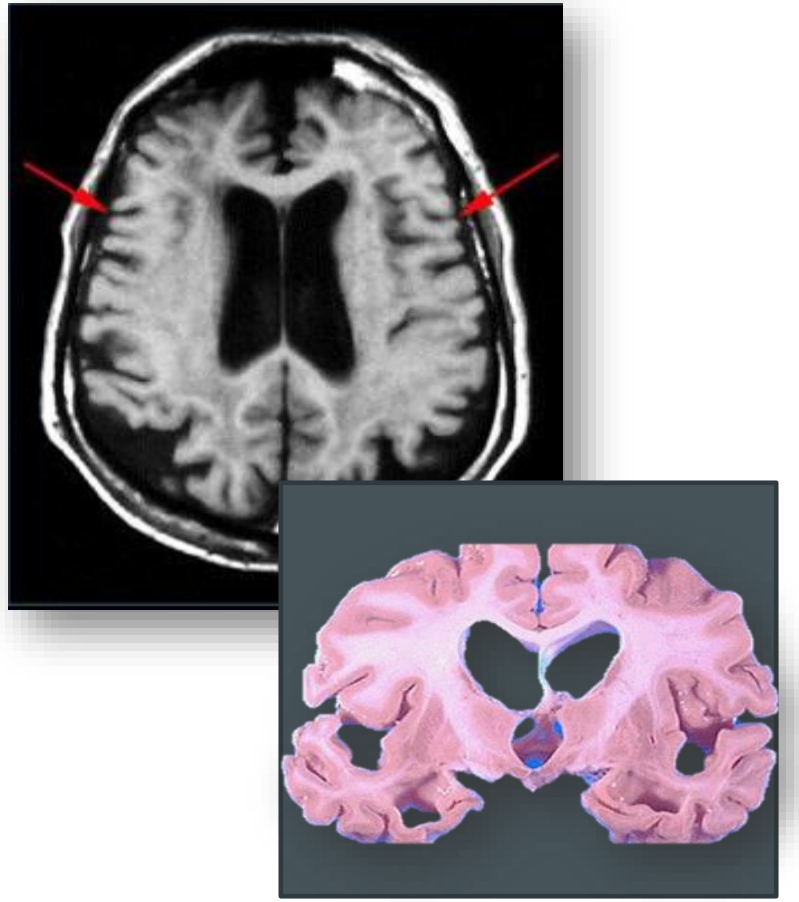
3. **Compensatory increase in CSF volume due to loss of brain parenchyma (Hydrocephalus ex vacuo)**
   - Infarcts
   - Neurodegenerative diseases

Communicating hydrocephalus

Noncommunicating hydrocephalus



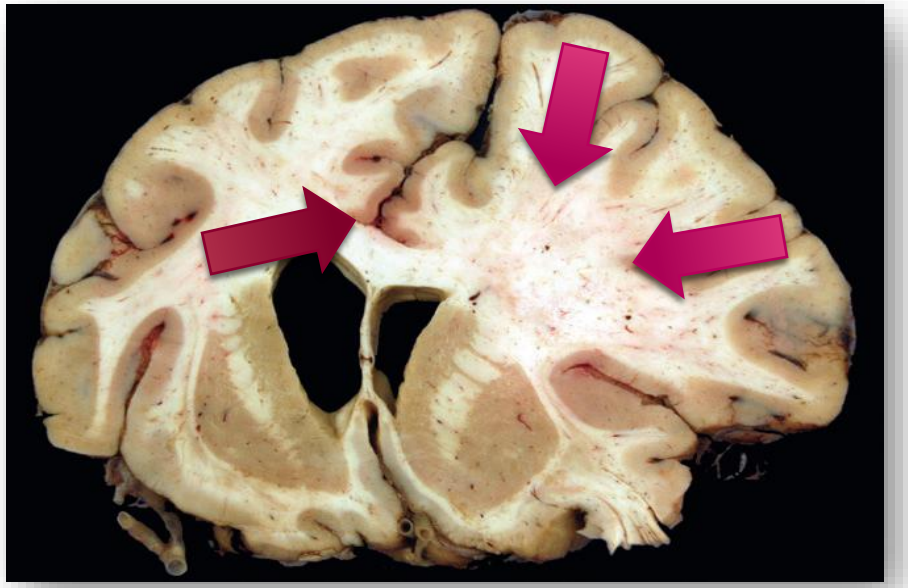
Hydrocephalus ex vacuo

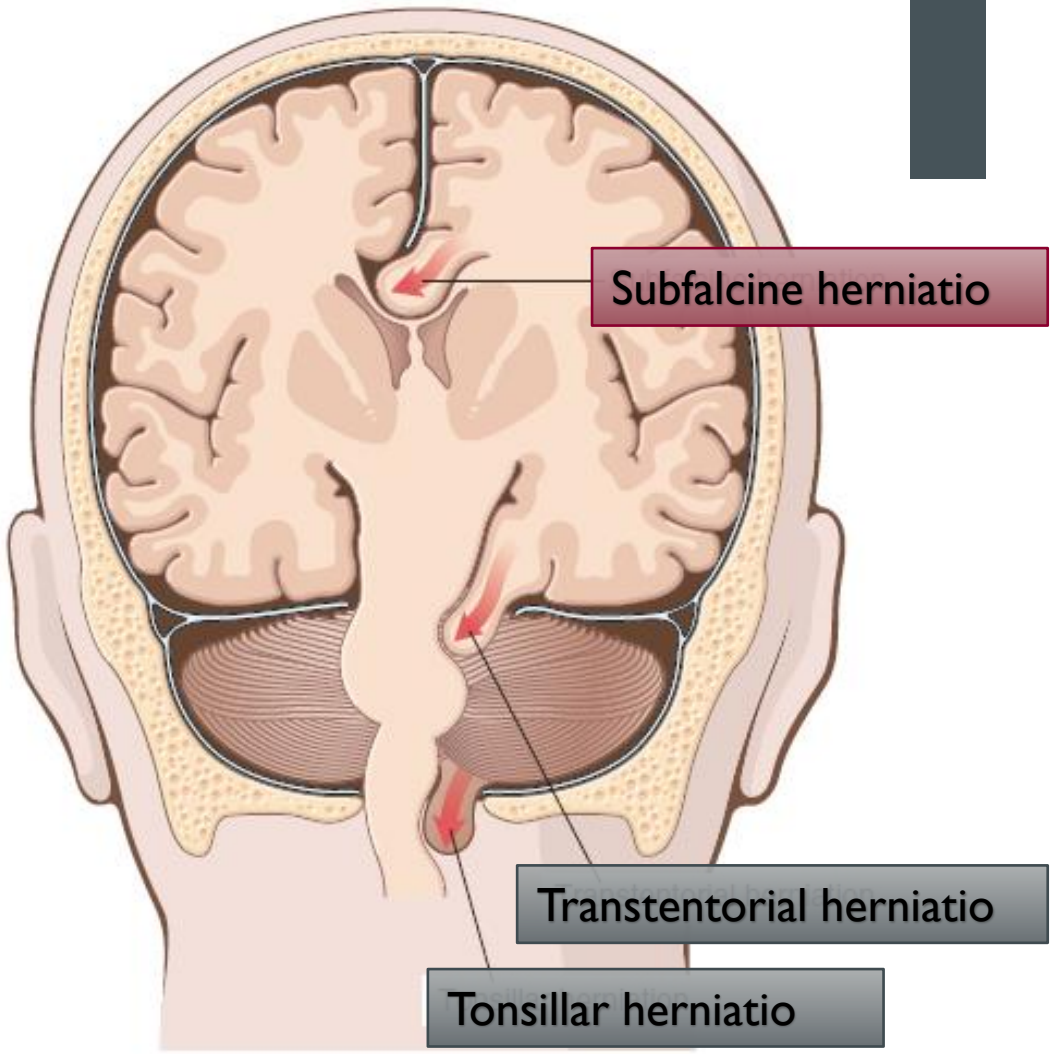


HERNIATION

Subfalcine herniatio

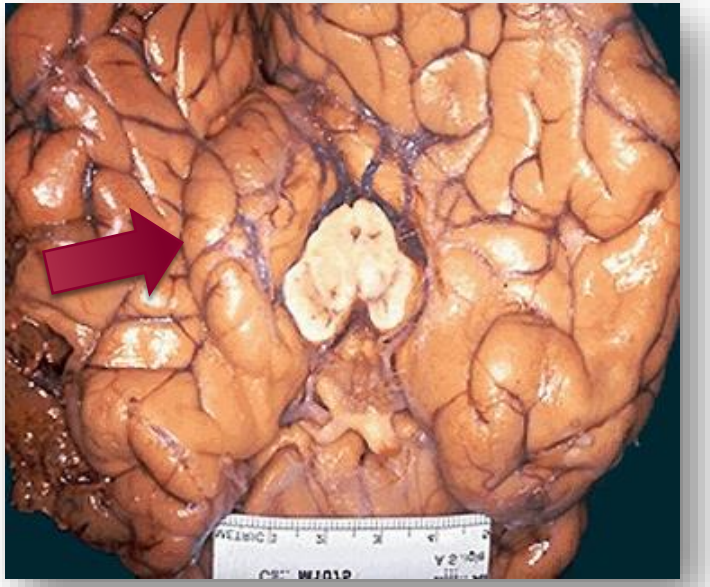
- Asymmetric expansion of a cerebral hemisphere
- Cingulate gyrus herniation
- Compression of anterior cerebral artery

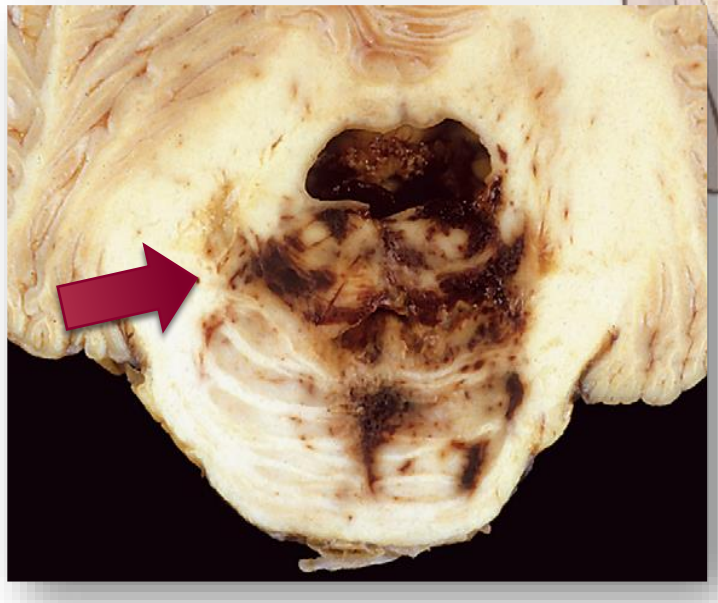


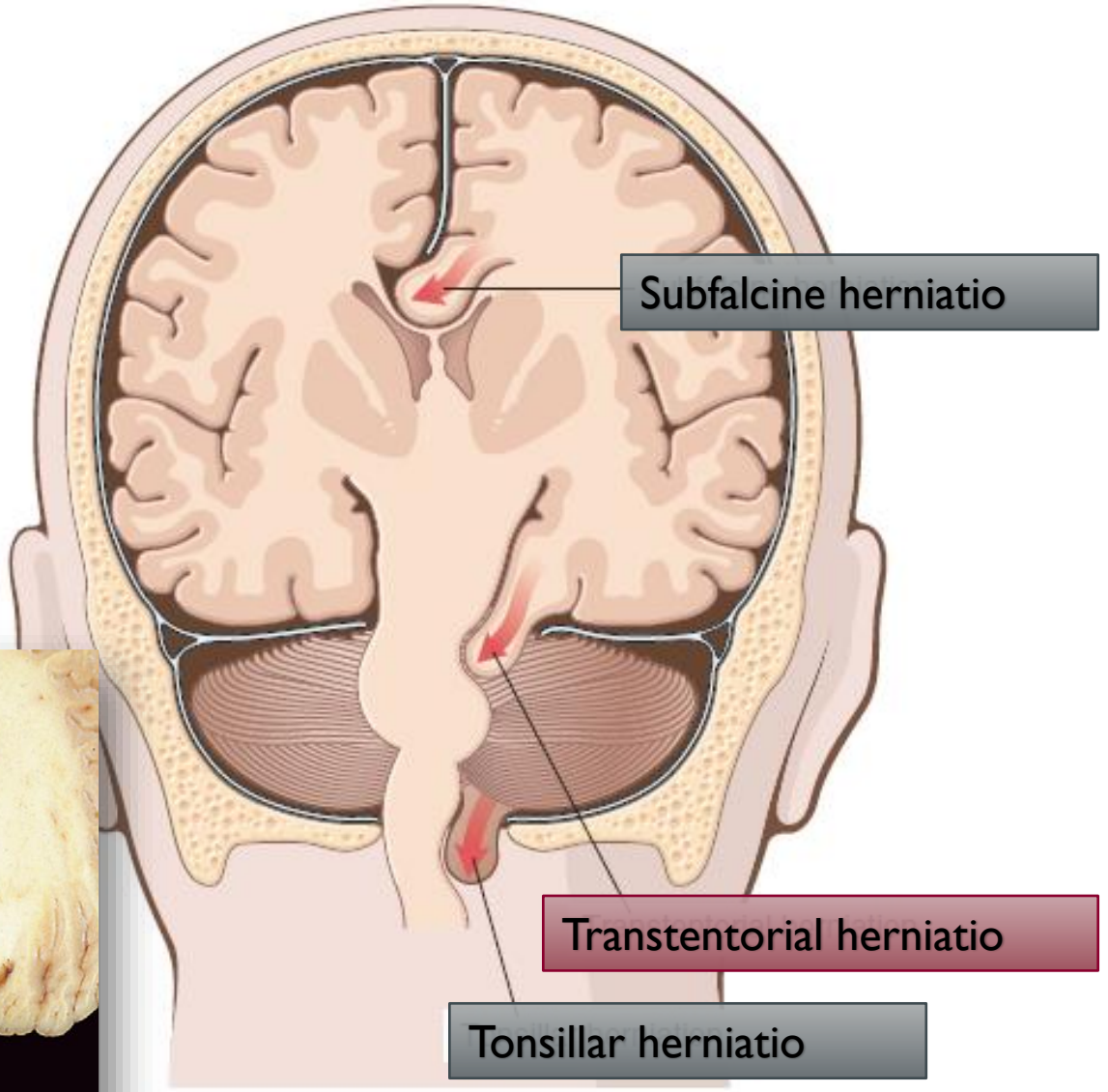


Transtentorial herniatio

- Temporal lobe herniation
- Third cranial nerve and poterior cerebral artery
- Contralateral pedunculus cerebri
- Kernohan's notch
- Midbrain and pons – Duret hemorrhages

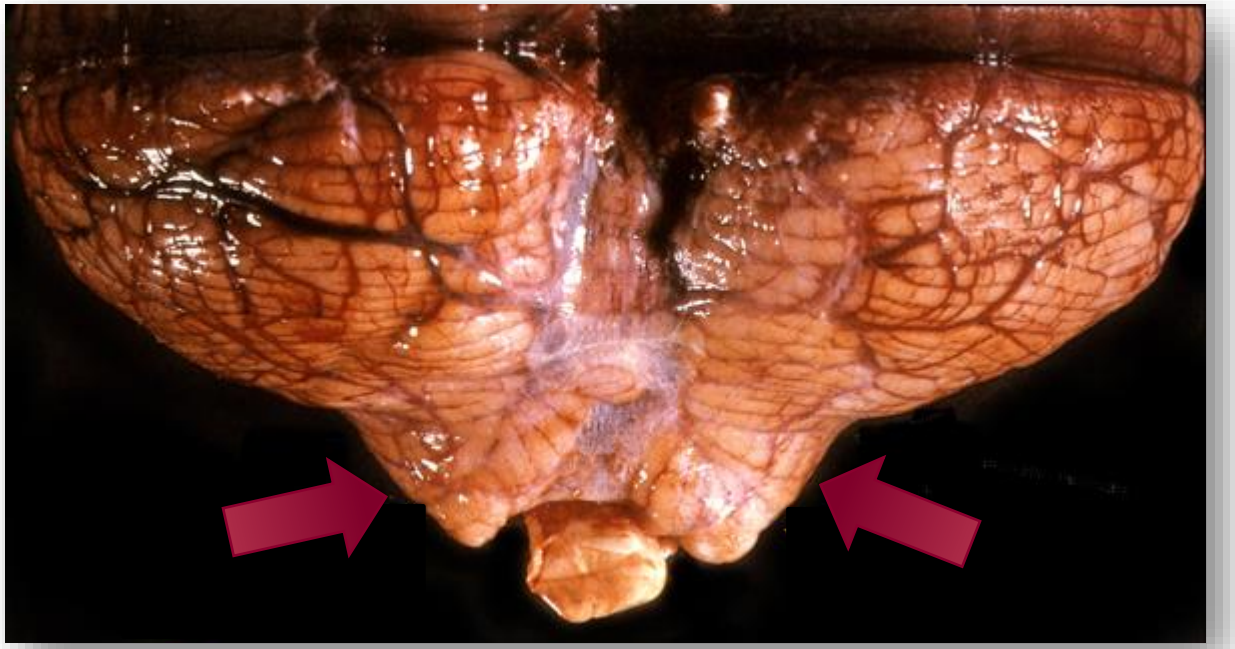


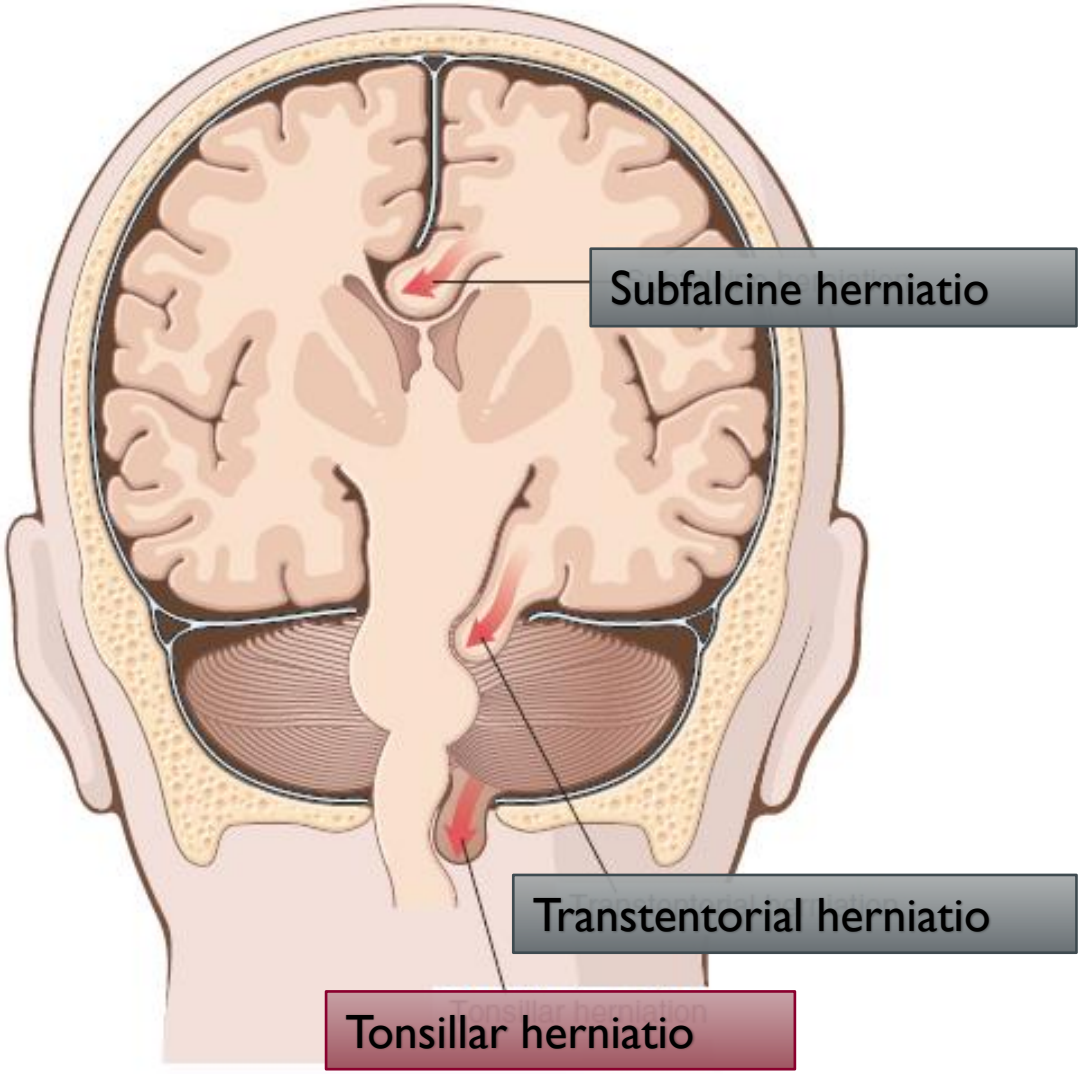




Tonsillar herniatio

- Displacement of the cerebellar tonsils
- Brain stem compression
- Medulla - respiratory and cardiac centers





- Robbins Basic Pathology, 10th Edition
- Neuropathology: A Reference Text of CNS Pathology, 3rd Edition