NEUROSURGICAL CEREBROVASCULAR PATHOLOGY: ANEURYSMS AND AVMS

34484 Pathology of the nervous system

Neurosurgery

Topic 21

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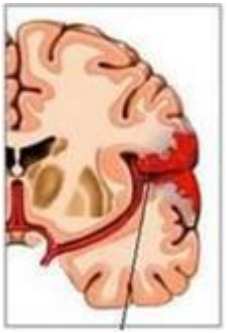
Key concepts

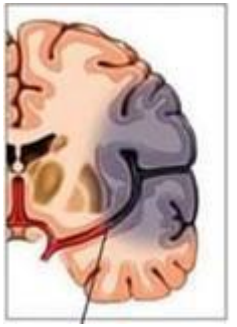
- **Subarachnoid haemorrhage**
  - *Brain aneurysm*
- **Brain vascular malformations**
  - *Cerebral and dural AVM and cavernous angioma (cavernoma)*
- **Spontaneous intracerebral hematomas**
  - *Brain hemispheres*
  - *Posterior fossa*
- **Cerebral ischemia**
  - *Cerebellar infarction*
  - *Malignant middle cerebral artery infarction*
  - *Venous sinus thrombosis*

➢ ***Spinal cord arteriovenous malformation***

Haemorrhagic stroke

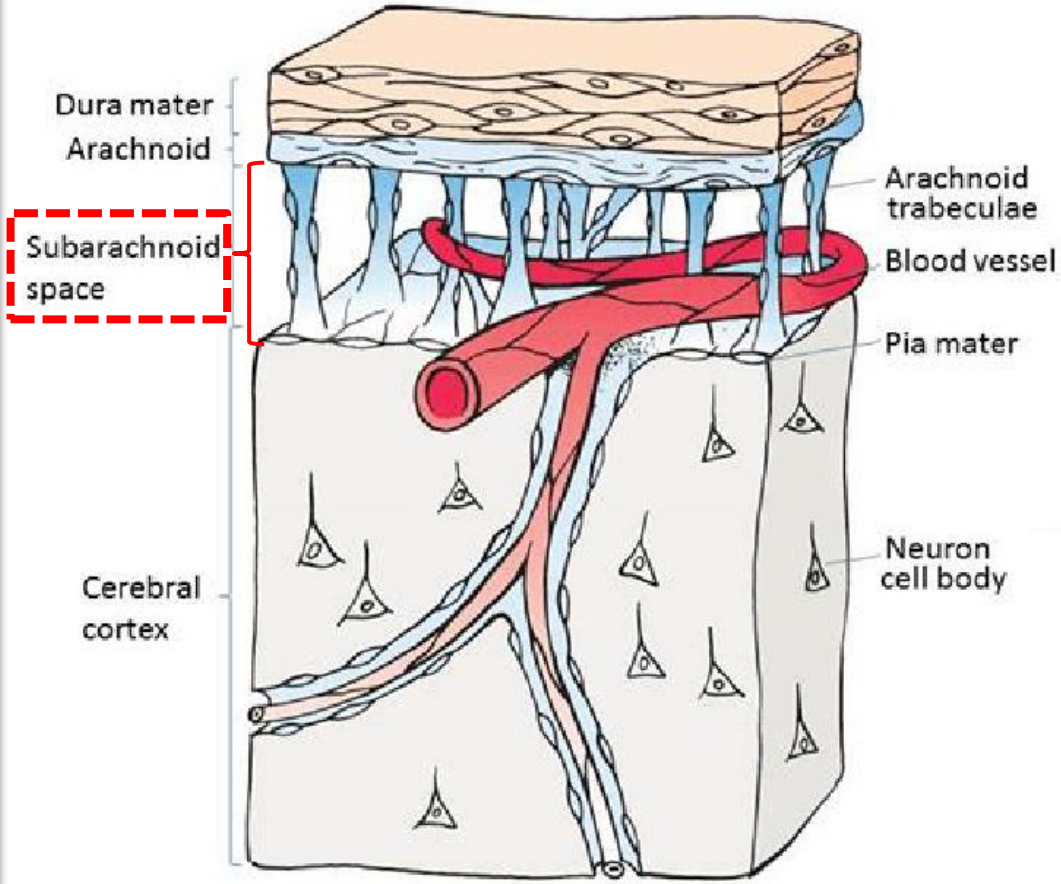


Ischemic stroke

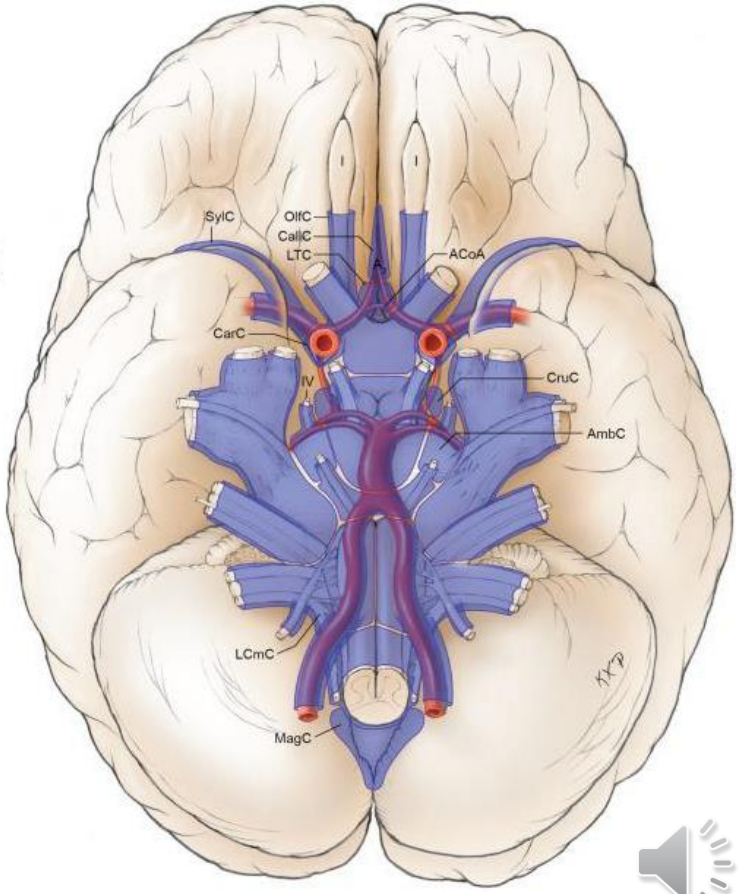


Subarachnoid space

- Between the parietal and visceral arachnoid membranes
- Bigger in the cranial base = CSF cisterns

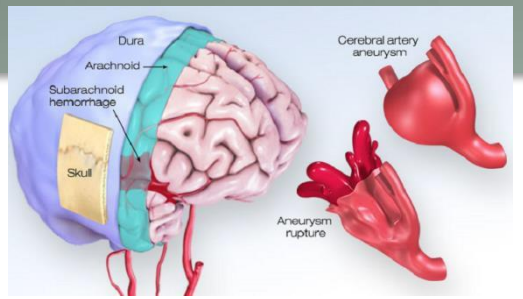


Subarachnoid space

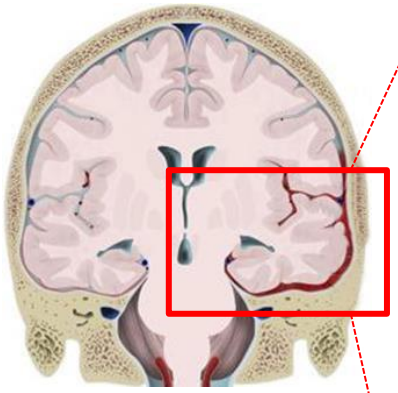


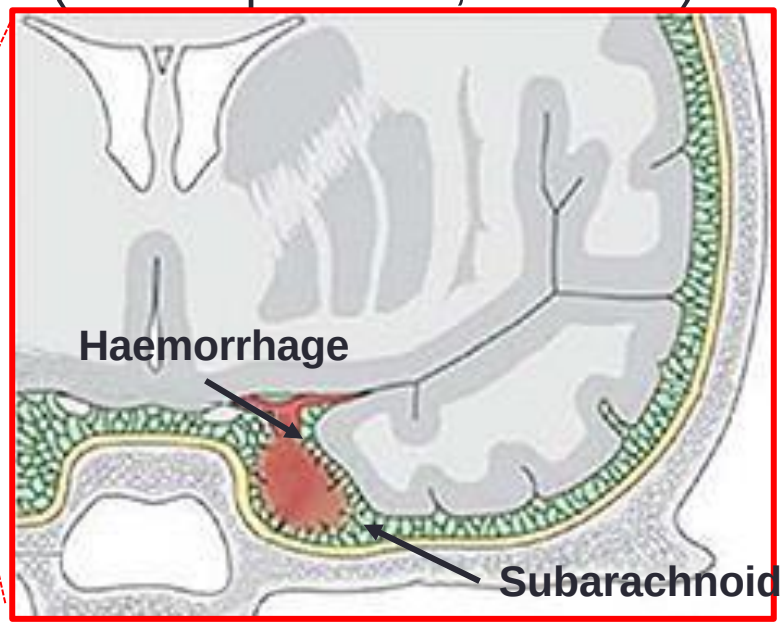
CSF cisterns

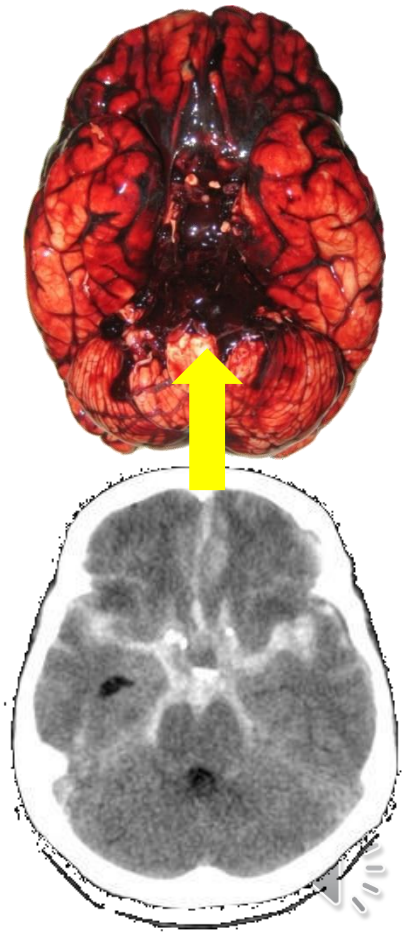
SUBARACHNOID HEMORRHAGE



- Blood in the subarachnoid space
  - Meningeal irritation
  - Sudden ↑ intracranial pressure ⇨ severe headache
    - *Sudden ↑intracranial pressure> 20 mmHg → loss of consciousness, possible death*
  - Minimal neurological deficit
  - Blood in CSF (lumbar puncture, CT-scan)







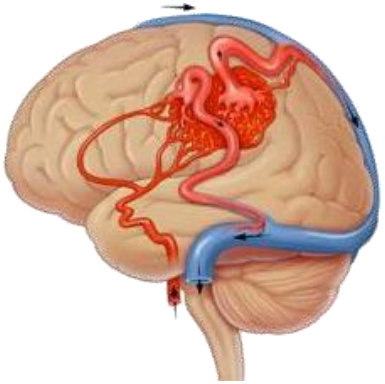
Subarachnoid hemorrhage (SAH)

- Incidence 10 / 100,000 population / year
- Age 45 – 65 years (80 %)
- “Most frequent cause = traumatic”
- Causes of spontaneous SAH
  - Brain aneurysm (> 70%)
  - Vascular malformation (5 %) –AVM
  - Hypertension (5 – 10 %) → *In*↓
  - Blood dyscrasia and anticoagulants / - antiaggregating agents (5 %) → *In* ↑
  - Extension of intraparenchymal haemorrhage
  - Mycotic aneurysms (septic emboli from endocarditis, usually middle cerebral artery)
  - Tumours (seldom)
  - Idiopathic (10 – 20 %)

Middle age

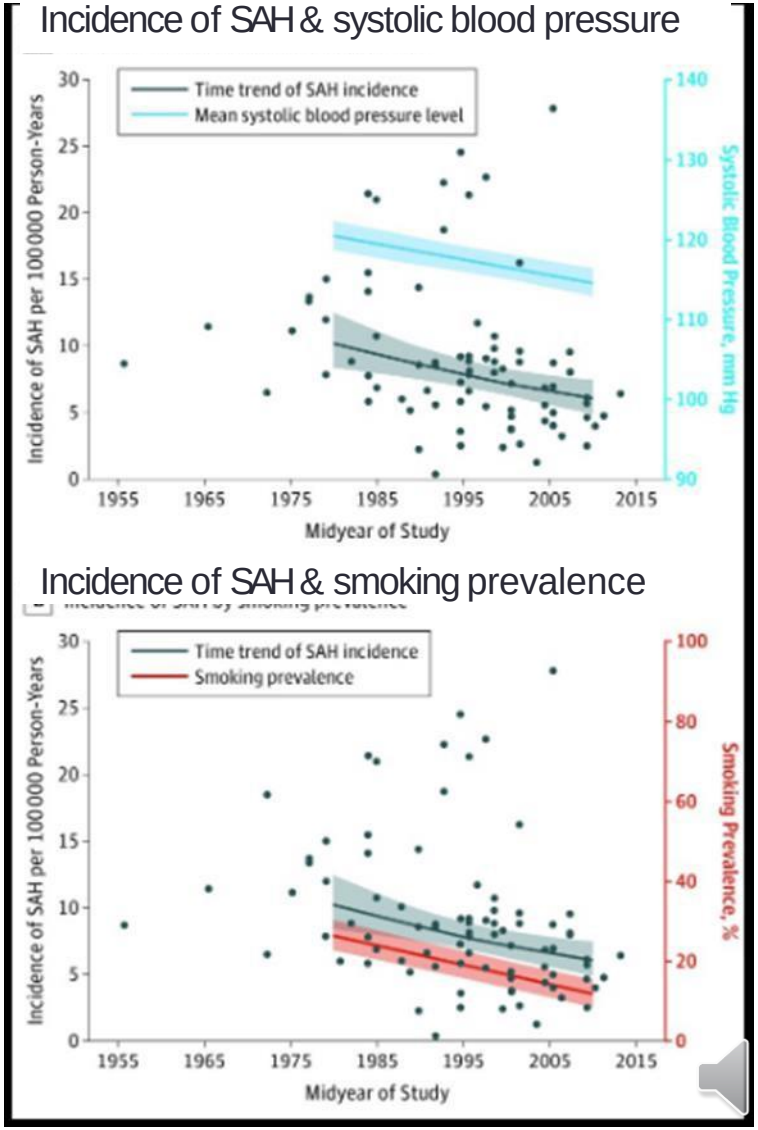
Young people

Advanced age



SAH

- Relationship with arterial hypertension and smoking
  - *Direct relationship worldwide*
  - *Positive SAH correlation ⇔ Hypertension, smoking, alcohol consumption*
- Relationship with increased consumption of antiplatelet and oral anticoagulants
  - *Ratio is lower than for intraparenchymal haemorrhage*



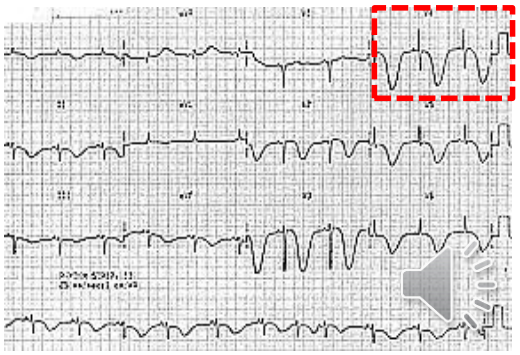
SAH

- Clinical features = raised intracranial pressure + sudden-onset MENINGISM
  - Intense thundering headache
  - Nausea and vomiting
  - Neck stiffness

SAH TRIAD

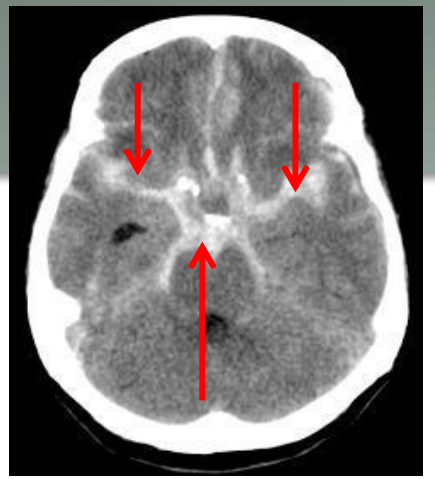
Brudzinsky sign

- Other manifestations
  - Photophobia
  - Consciousness level alteration, transitory (50 %)
  - Hydrocephalus (CSF obstruction at skull base)
  - Papilledema, sub hyaloid or vitreous haemorrhage
  - Neurological deficit (bleeding associated intraparenchymal)
  - EKG disturbances (ischemia, arrhythmias)



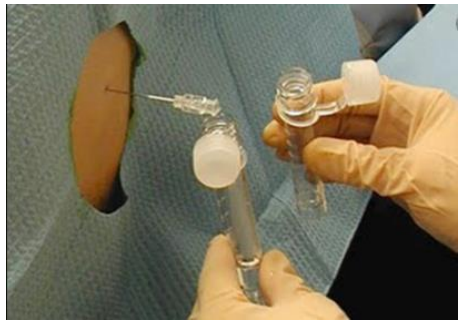
SAH

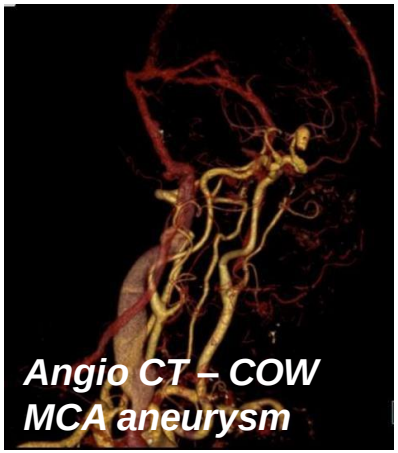
WE WILL CONSIDER THIS LATER



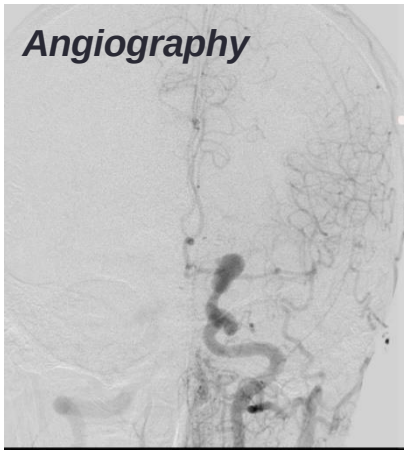
URGENT CT-scan

- Diagnosis
  1. Of the SAH
     - *CT-scan = prefer 1st test → Detects 95 % SAH*
     - *If you suspect clinical but CT Ø→ lumbar puncture*
  2. Of its cause = "angiography that determines the presence of aneurysm"
     - *Angio-CT can identify any brain aneurysm, but for the moment it is not the "gold standard"*

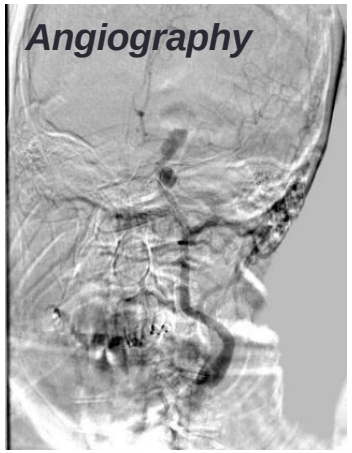




Angio CT – COW MCA aneurysm



Angiography



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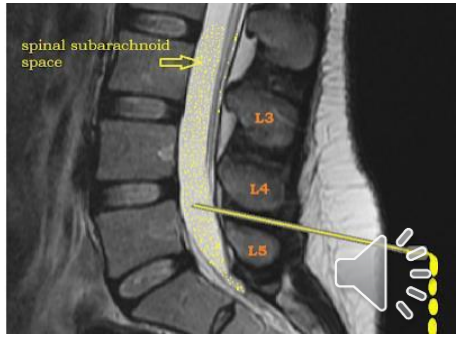
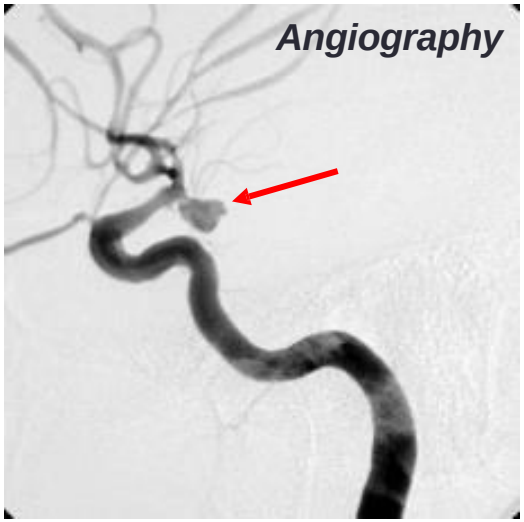
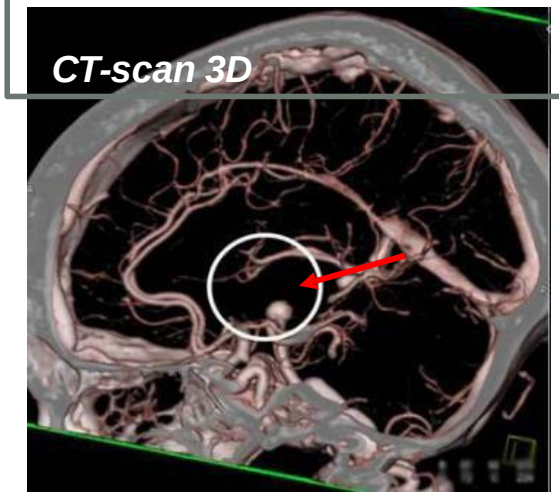
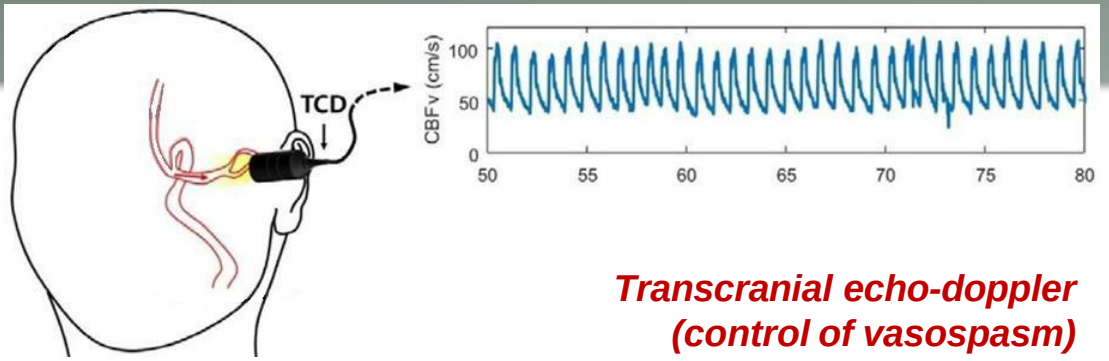


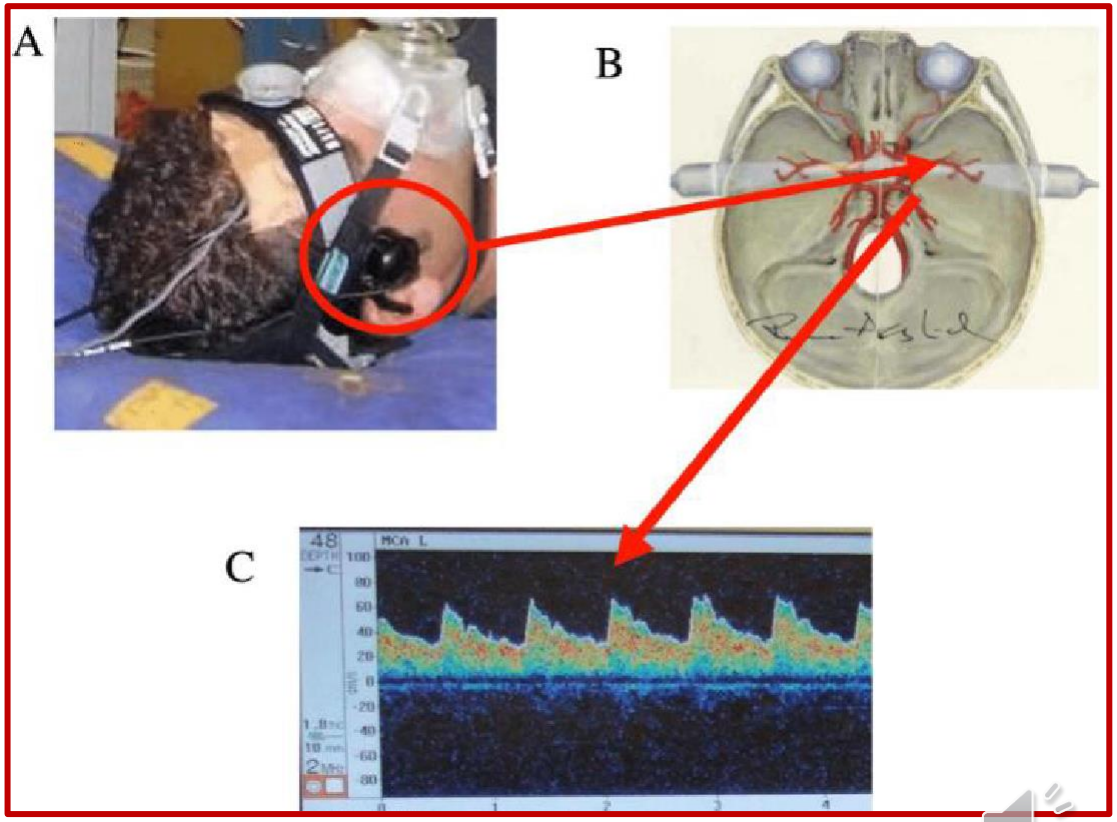
SAH







Transcranial echo-doppler (control of vasospasm)



SAH

- Predictive factors poor prognosis
  - Poor neurological status on admission (Hunt Hess scale)
  - Amount of blood on CT-scan or rebleeding
  - Location of aneurysm in the middle cerebral artery
  - *Age > 70 years, ventricular hematoma, anterior communicating aneurysm*

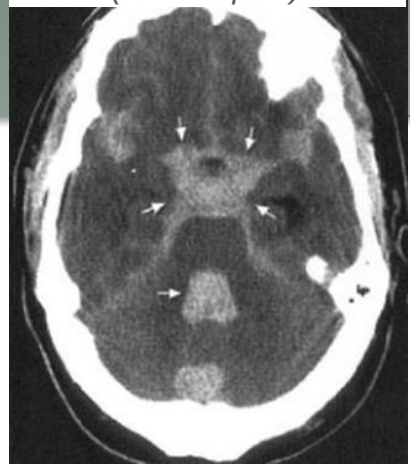
HUNT-HESS SCALE

- GRADE1:
- Mild headache, normal mental status, no cranial nerve or motor findings –(GCS* score 15, no motor deficits)
- GRADE 2:
  Severe headache, normal mental status, may have cranial nerve deficit –(GCS score 13–14, no motor deficits)
- GRADE 3:
  Somnolent, confused, may have cranial nerve or mild motor DEFICIT- (GCS SCORE 13–14, WITH MOTOR DEFICITS)
- GRADE 4 :
  Stupor, moderate to severe motor deficit, may have intermittent reflex posturing- (GCS score 7–12, with or without motor deficits)
- GRADE 5:
  Coma, reflex posturing or flaccid (GCS score 3–6, with or without motor deficits)

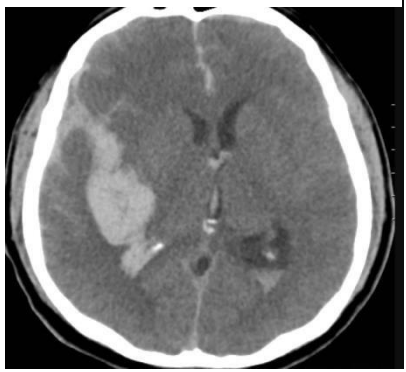
Preserved level of consciousness → **Stroke Unit**

Altered level of conciousness → **ICU**

Massive SAH (catastrophic)



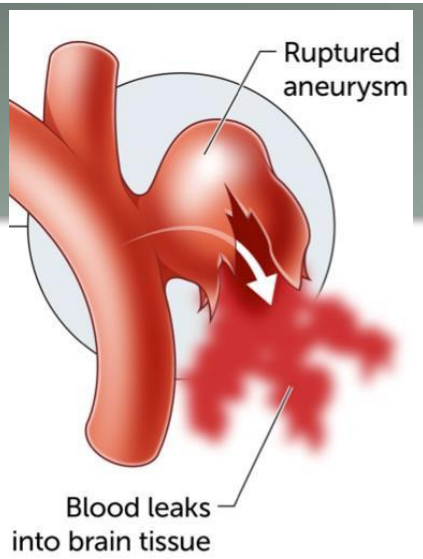
SAH due to middle cerebral artery aneurysm



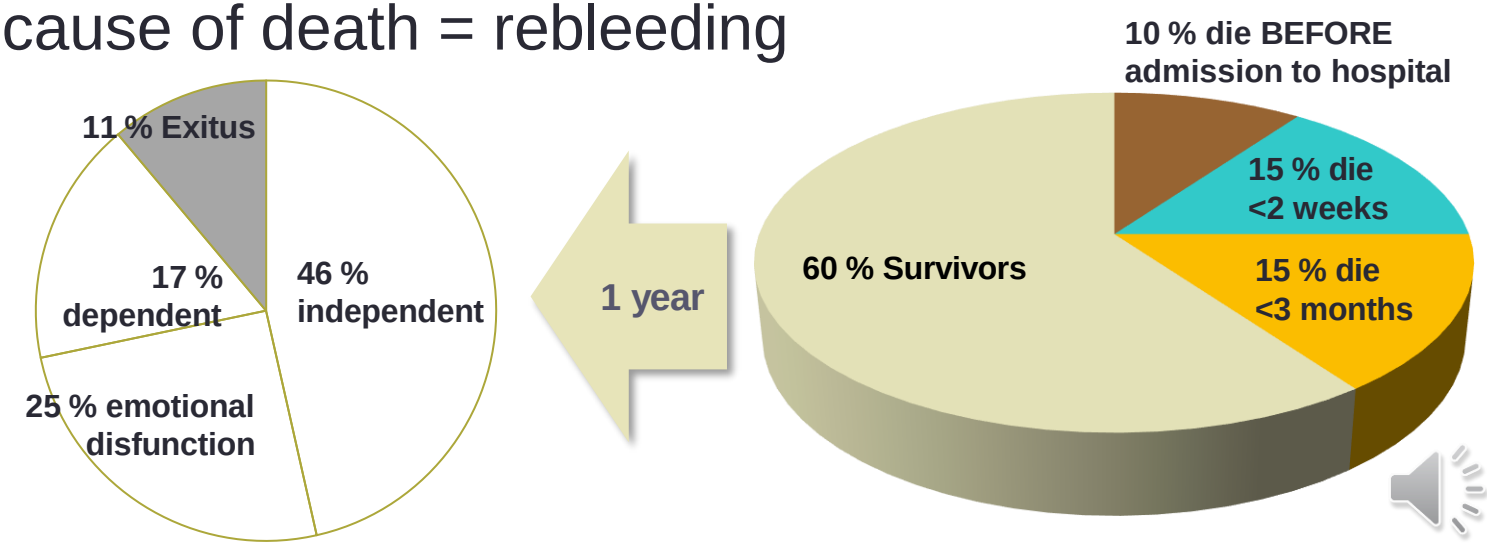
Hunt and Hess scale for non-traumatic SAH.

Add 1 grade if systemic disease (high blood pressure, COPD, diabetes mellitus) or severe vasospasm

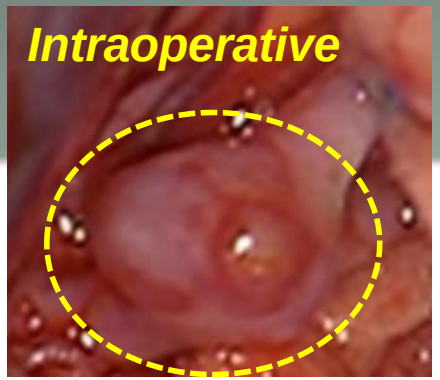
SAH

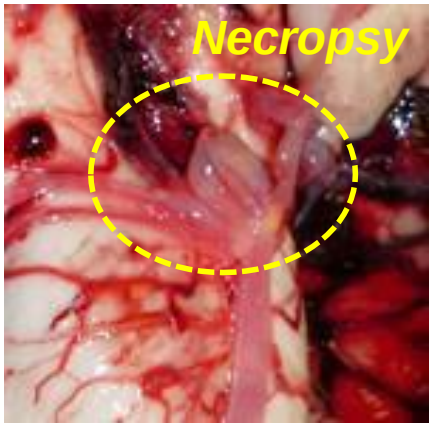


- Prognosis
  - Mortality → Hunt & Hess IV-V Grades = 5 times higher for grades I-III
    - *Global 40 – 50 %*
    - *1st day 10 % ⇒ up to 40 % 1st – 3rd month*
  - Survivors
    - *> 50 % with neurological deficits due to SAH or complications (30 % severe)*
- Leading cause of death = rebleeding

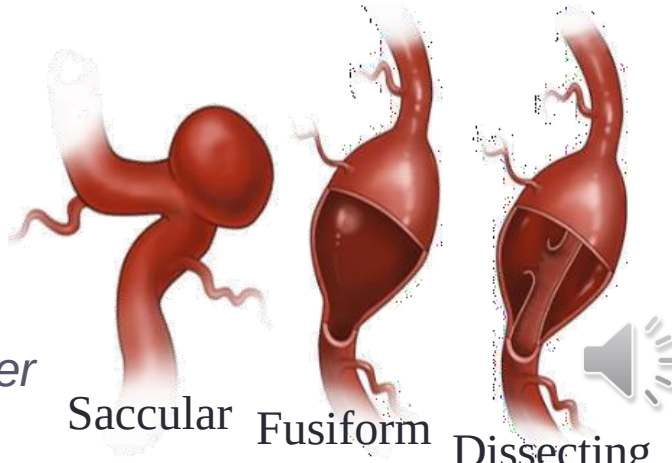


BRAIN ANEURYSM

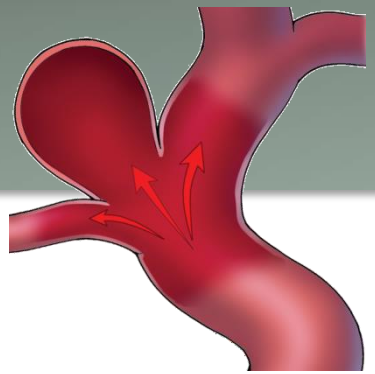




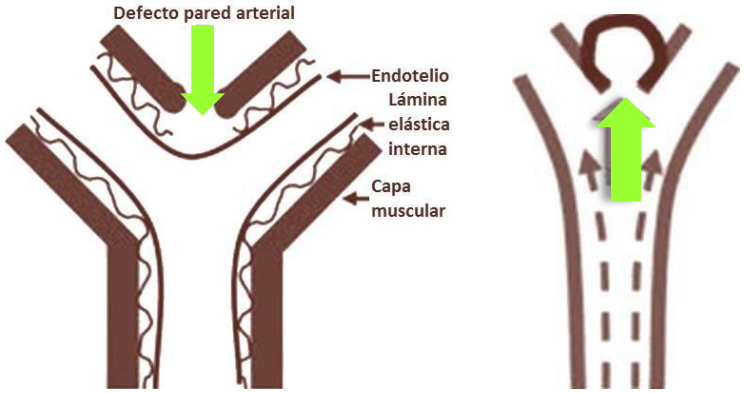
- Abnormal dilation of the arterial wall
  - Congenital
    - *Associated with polycystic kidney disease (autosomal dominant), fibromuscular dysplasia, aortic coarctation, Marfan syndrome*
  - Acquired → hypertension, arteriosclerosis
    - *Mycotic → infections (peripheral brain arteries, associated with endocarditis*
    - *Traumatic (rare)*
- Shape
  - Saccular → + frequent, bifurcation of vessels, ø variable (ø > 2'5 cm = giant), with neck and dome – rupture –
  - Fusiform → in other locations
    - *Possible dissecting = damaged only intima layer*

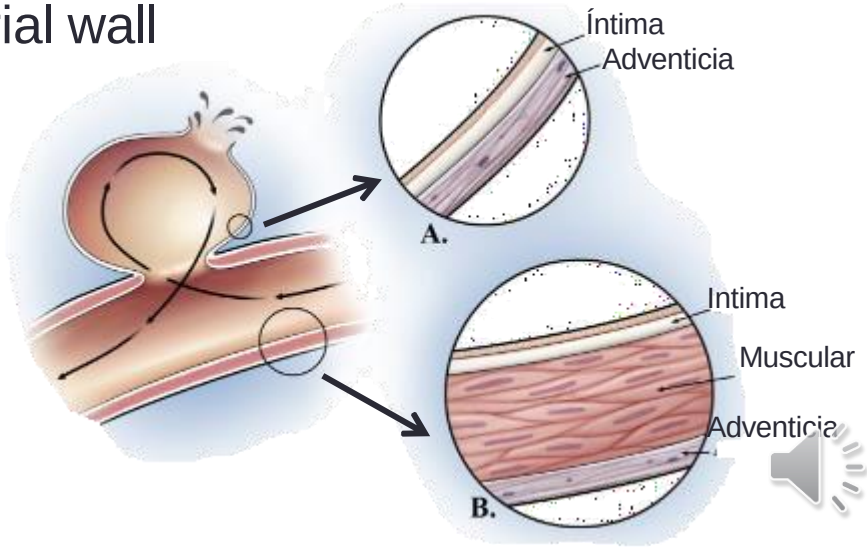


Etiopathogenesis



- Factors involved
  - Focal arterial wall weakness (collagen disease)
    - *Extracellular matrix defects or degeneration of one or more layers of the vessel*
      - Endothelial cells, internal elastic lamina, smooth muscle fibre, extracellular and adventitial matrix
  - Hemodynamic stress
    - *↑flow due to vascular anomaly or arteriovenous malformation*
    - *Arterial hypertension*
  - Inflammatory process of the arterial wall





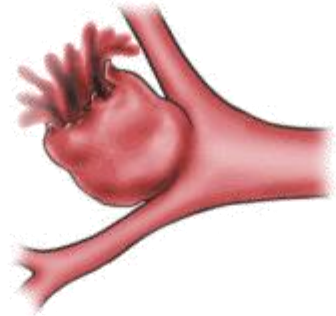
Factors involved

Patient-specific factor

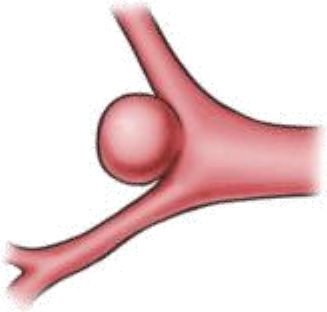
- Smoking
- High blood pressure
- Inflammatory disease
- Bone mineral loss
- Sex hormone exposure

Aneurysm-specific factor

- Large size
- Bifurcation site
- Multiplicity
- Daughter sac
- High dome-neck ratio
- Multilobularity
- Adjacent arterial geometry

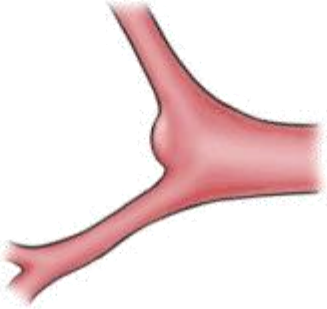


Growth & rupture



- Female
- Short stature
- Genetic disorder
- Bicuspid aortic valve
- Dilated aortic root
- Aortic aneurysm
- Arterial dissection
- Bone fragility
- Malnutrition, e.g. copper

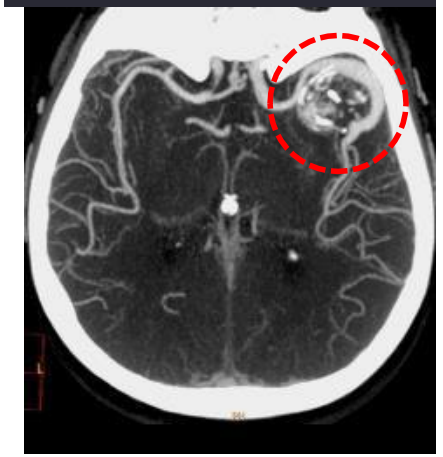
Development

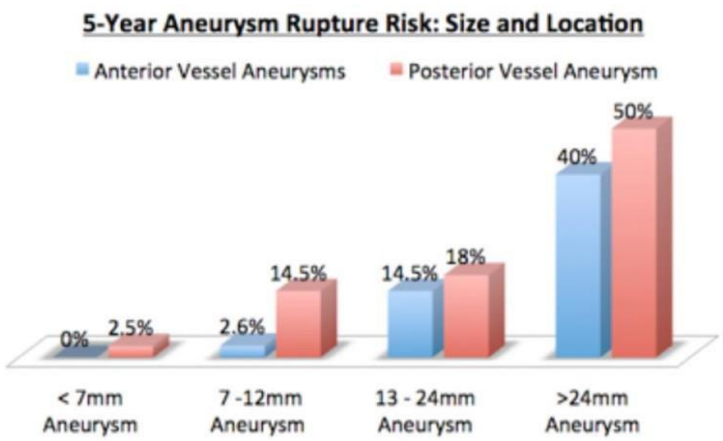


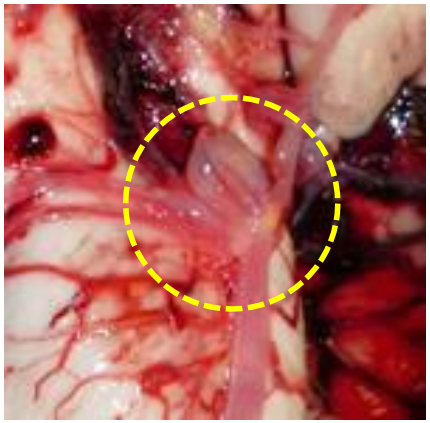
Epidemiology

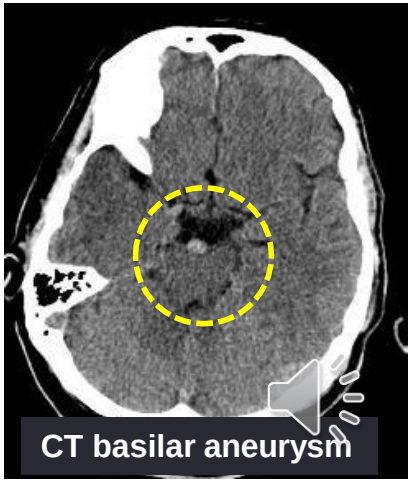
- Epidemiology
  - Prevalence ~ 0.2 – 1 % population (0.2 - 8.9 %)
    - *↑due to improvement in imaging techniques*
  - Possibility of aneurysm rupture
    - *1-2 out of 5 will rupture→peak 50 - 60 years*
    - *Rupture risk 0.7 – 4 % / year, according to size*
  - Incidence of rupture
    - *Ruptured aneurysms: 6 / 100,000 persons / year*
    - *Slightly > in ♀ (♂:♀ 2:3), but more in males < 40 years and females > 40 years*

CT-angio giant aneurysm middle cerebral artery



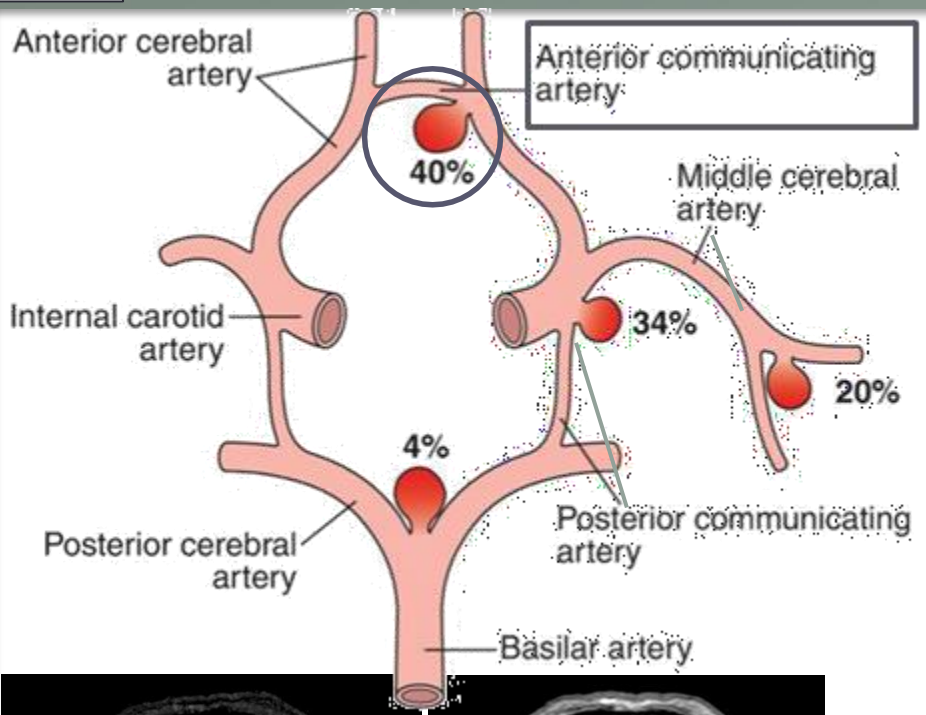


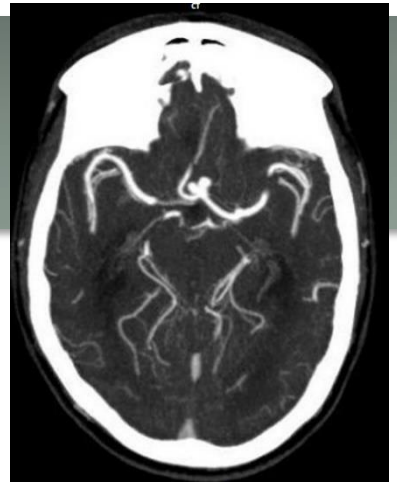


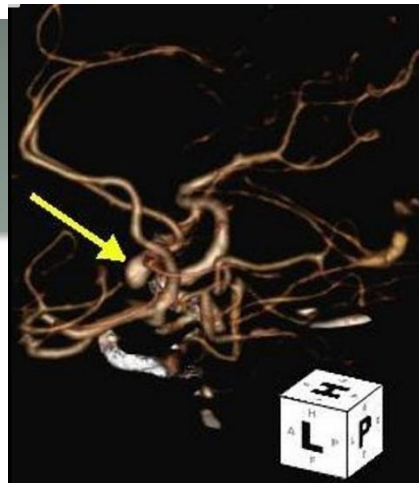


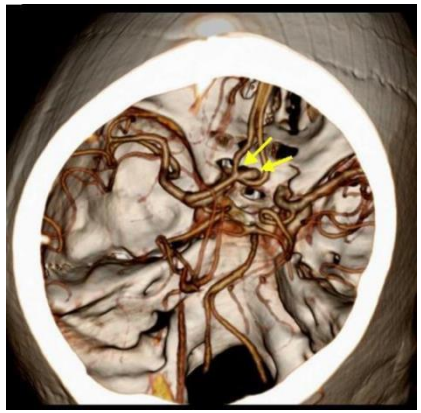
CT basilar aneurysm

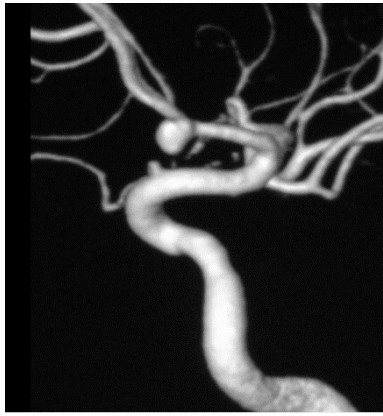
Location

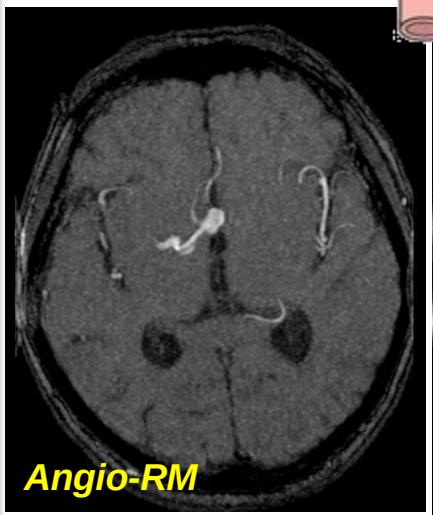


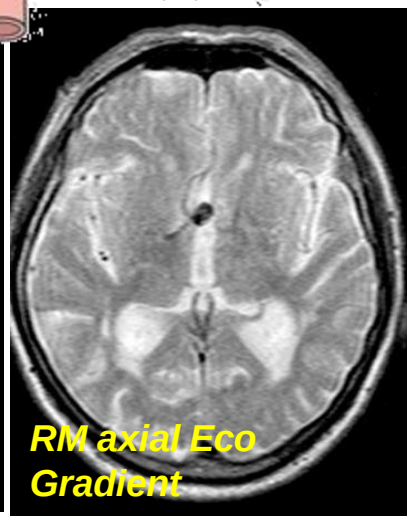




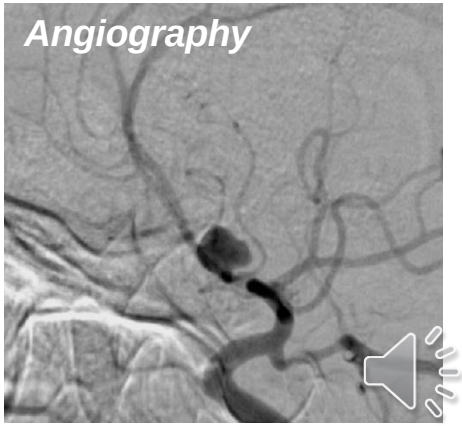






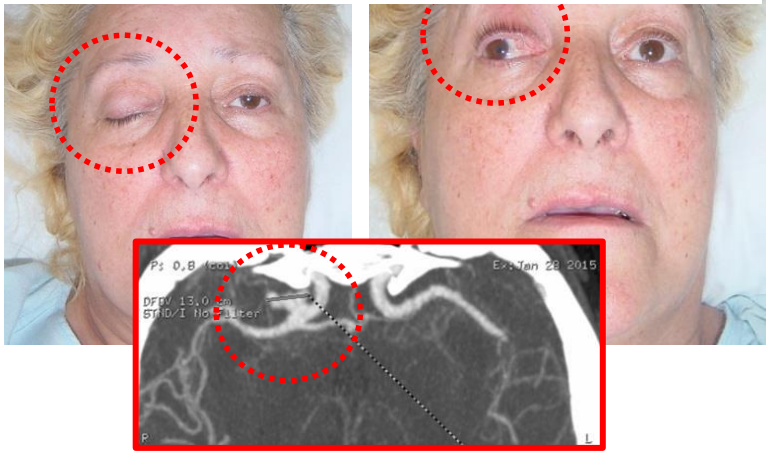


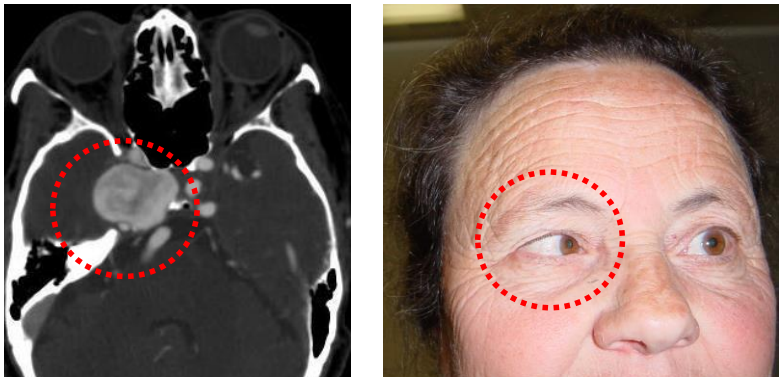
20 – 30 % multiple
10 – 20 % bilateral
1 % associated with arteriovenous malformation



Pre-bleeding symptoms

Posterior communicating artery aneurysm
Third nerve palsy - oculomotor





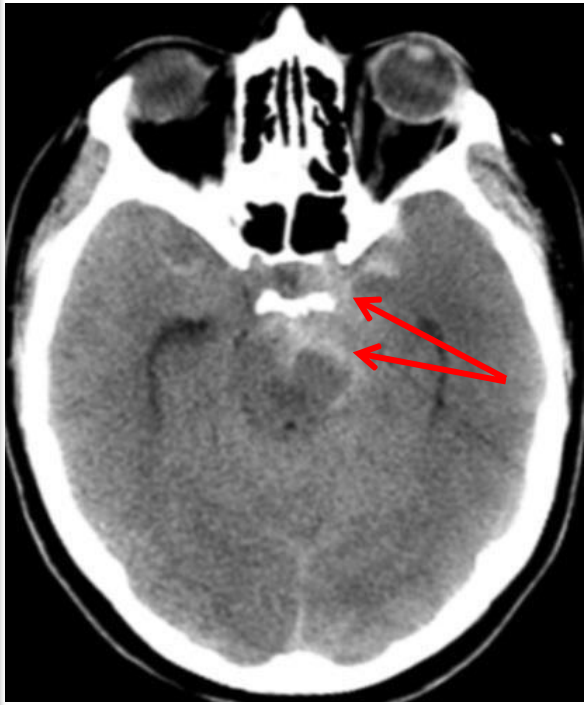
Sixth nerve palsy (abducens) with cavernous sinus internal carotid artery aneurysm

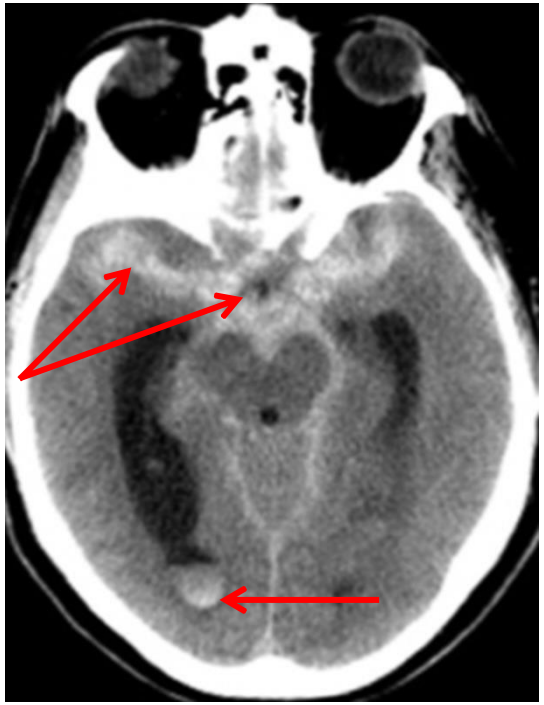
- By compression of nearby structures = premonitory symptoms
  - Sentinel headache (expansion of the aneurysm)
    - *Typically, retroocular (posterior communicating artery aneurysm)*
  - *Third nerve palsy* (oculomotor) ↔ *Posterior communicating artery aneurysm*
  - Sixth nerve palsy (abducens) ↔ internal carotid artery aneurysm
  - Visual field deficits ↔ anterior communicating artery aneurysm

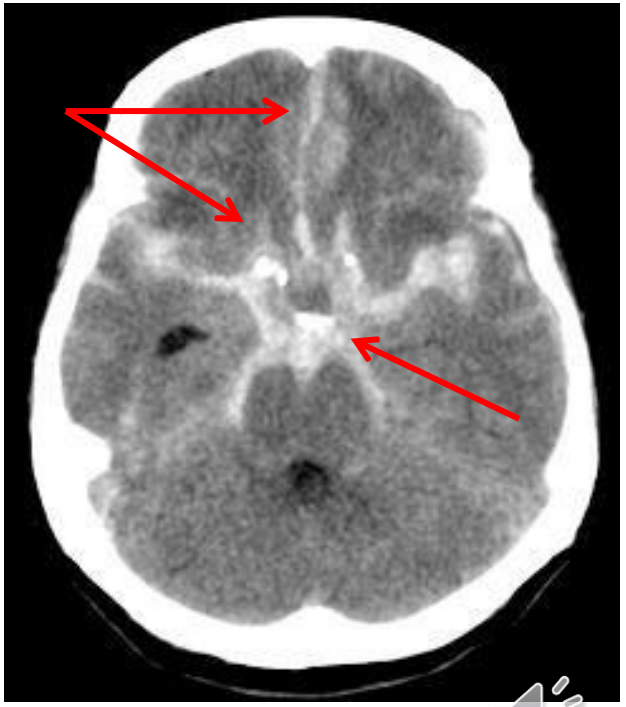
SAH diagnosis

We just saw it

- URGENT diagnosis = head CT-scan
  - Prefer first test → Detects 95 % SAH
  - If suspected clinically but CT-scan ø → lumbar puncture



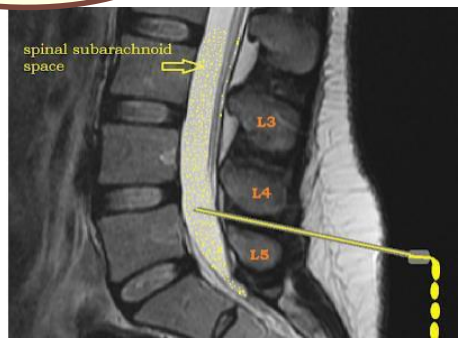


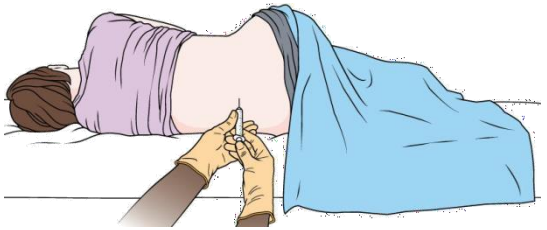


SAH diagnosis

We just saw it

- URGENT diagnosis
  - Prefer first test → Detects 95 % SAH
  - IF ØCT-scan but strong clinical suspicion → lumbar puncture in <u>lateral decubitus position</u>
    - *Risk of cerebellar tonsil herniation = cardiorespiratory arrest*
    - *Blood in subarachnoid space (CSF)*
    - *More sensitive, but second choice*
    - *Differential diagnosis with traumatic puncture*

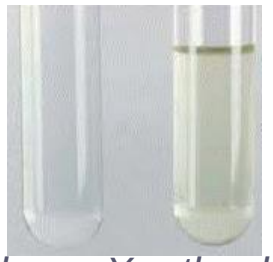




→

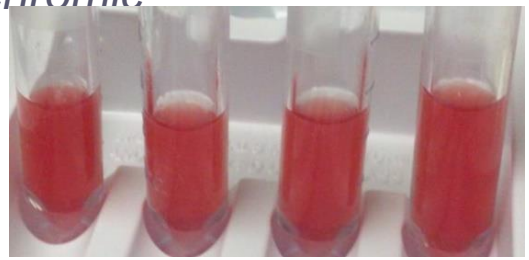
3-tube test

n ochromic

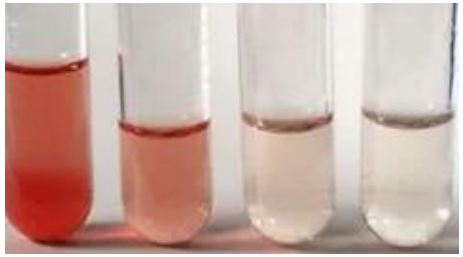


Normal CSF

Xanthochromic CSF (> 2nd day)



SAH – haemorrhagic CSF DOES NOT clear

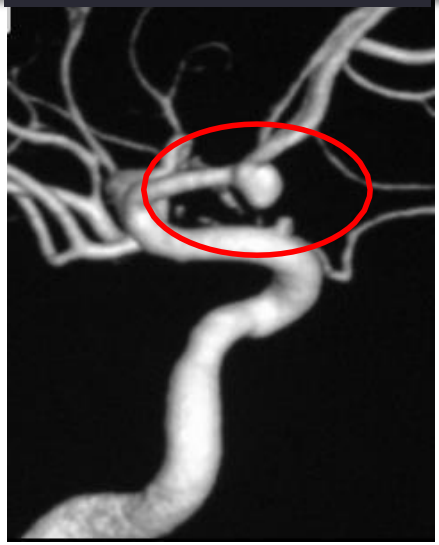


Traumatic lumbar puncture

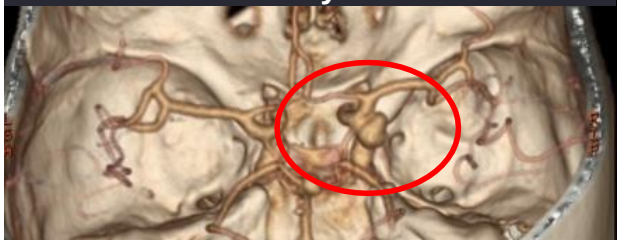
Diagnosis of aneurysm

- Urgent diagnosis of ruptured aneurysm = Angio-CT-scan
  - Can identify any brain aneurysm
  - It is the general practice, but nowadays it is not the *"gold standard"*
- *After the diagnosis of SAH, arteriography should be done to determine the presence of aneurysm*

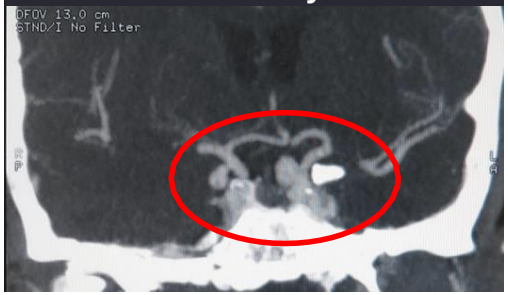
Angio-CT-scan 3D
Anterior communicating artery aneurysm



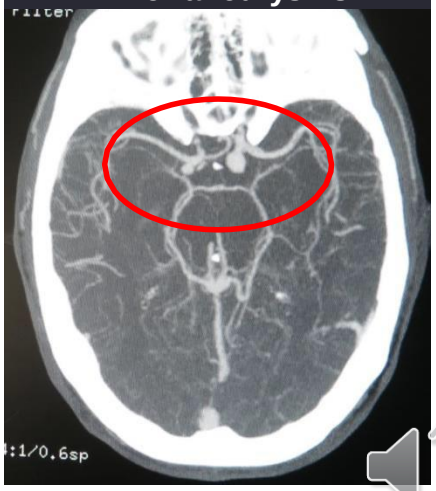
Angio-CT-scan 3D
Posterior communicating artery aneurysm



Angio-CT-scan
Mirror aneurysms

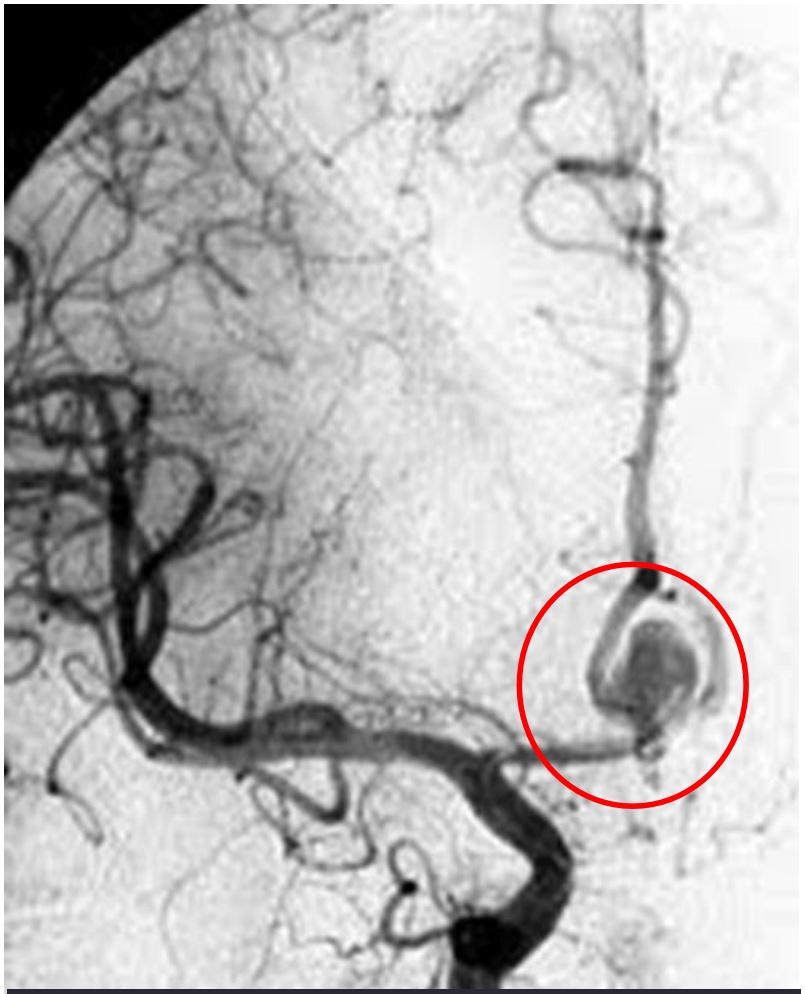


Angio-CT-scan
Mirror aneurysms



Diagnosis of aneurysm

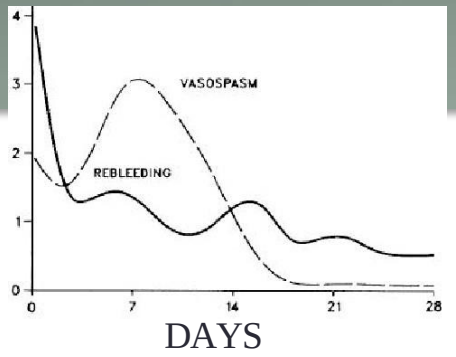
- Usefulness of cerebral angiography
  - Identify and define aneurysm and affected vessels
  - Identify other aneurysms (30 %)
  - Assess vasospasm
  - Plan the most indicated treatment (endovascular vs surgical)
  - *If Ø (15-20 %), repeat angiography after 2-3 weeks*
  - If stays at *Ø repeat 3 months later*

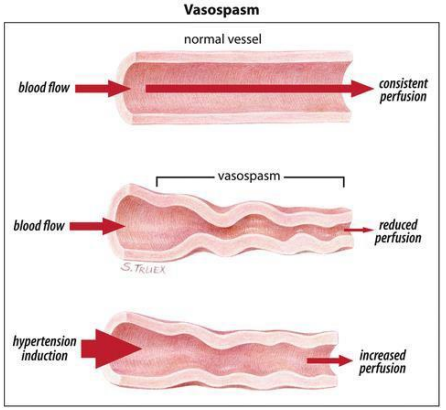


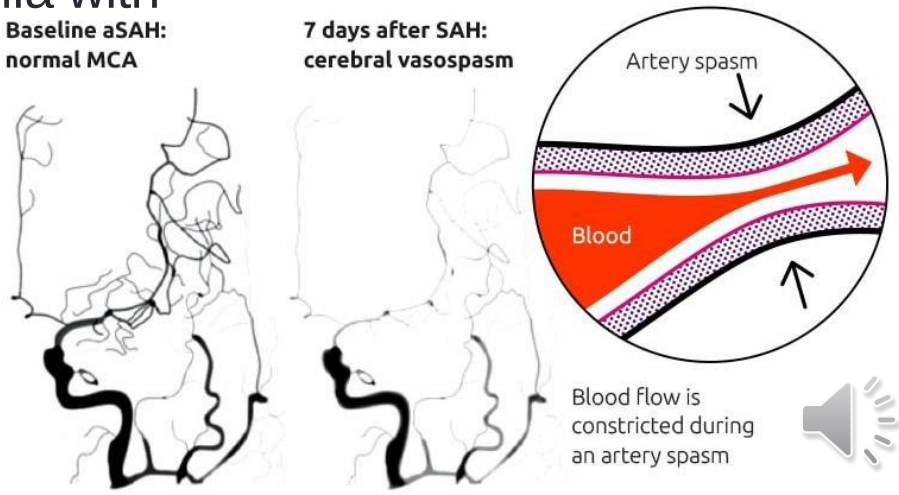
Angiography. Anterior communicating artery aneurysm

Complications (1/3)

- RE-BLEED = Main cause of death
  - Risk 4 % <24 h → 20 % in 15 days → 40 % in 30 days
  - Less for early treatment of the aneurysm (embolization or surgery)
- VASOESPASM = Main cause of morbidity
  - 50 % cases, days 4 – 10
  - Mortality 10 % *per se*
  - Morbidity: 25 % (will suffer ischemia with neurological deficits)
  - Check daily with Eco-Doppler
  - Treatment: Nimodipine
  - "Triple H therapy" is
  - OBSOLETE *(haemodilution - hypervolemia - hypertension)*

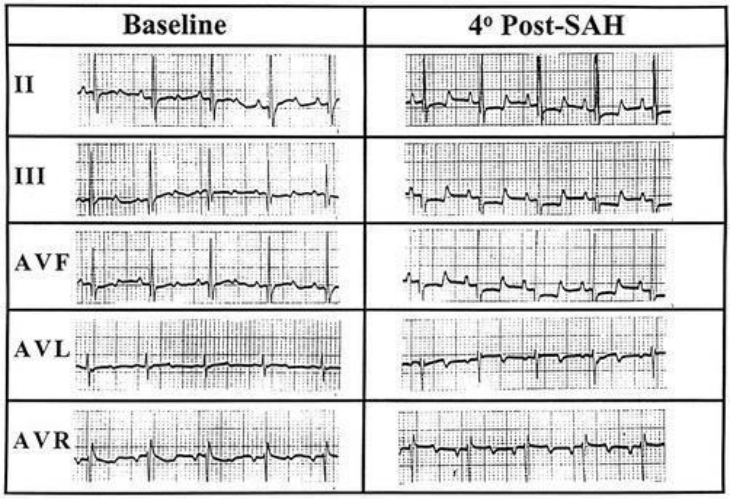


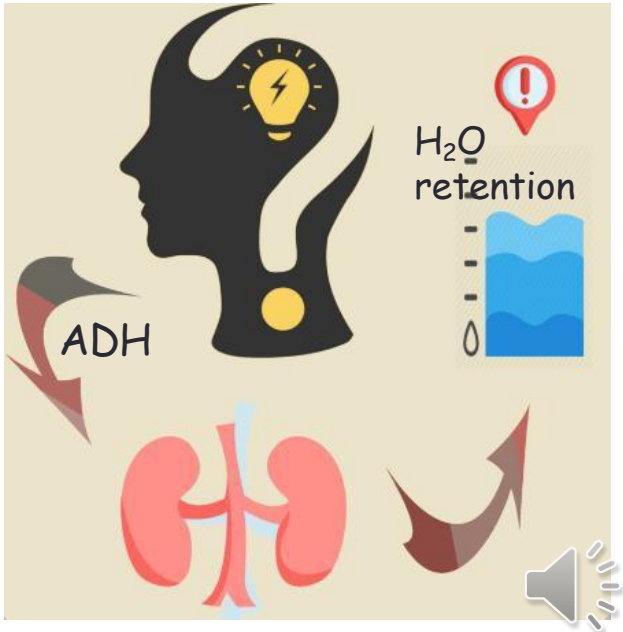




Complications (2/3)

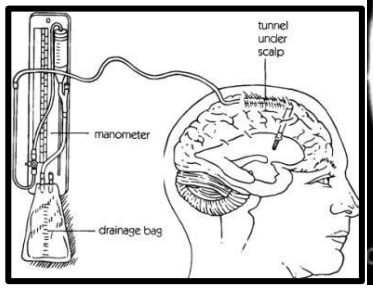
- EKG abnormalities (65 %)
  - Sinus tachycardia, T wave abnormalities, ST segment changes, prolonged QT …
  - Possible ischemia + focal necrosis → Impaired cardiac function → Pulmonary edema
  - Fluctuating heart dysfunction worsens prognosis
- SIADH
  - Hyponatremia: seen in 50 % SAH
    - *Multiple causes, sometimes iatrogenic*
  - Excess ADH ⇨ H_2O retention ⇨ ↑[Na^+] and urinary osmolarity, ↓[Na^+] and blood osmolarity
  - ➢ *Not to be confused with cerebral salt wasting syndrome (CSWS)*

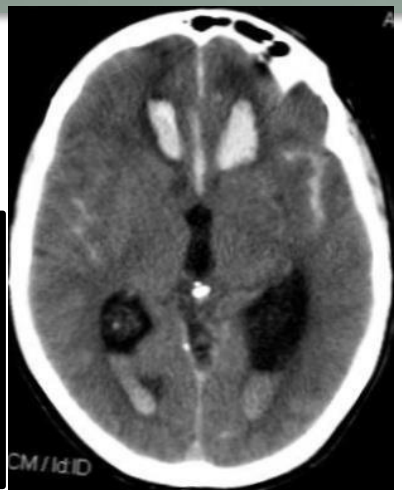


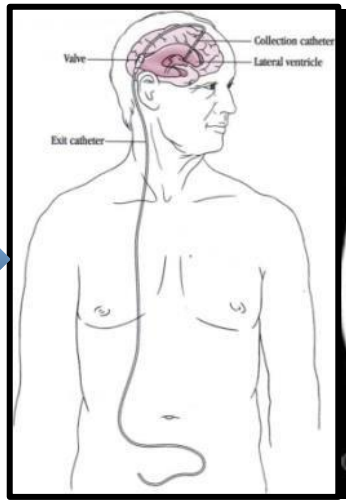


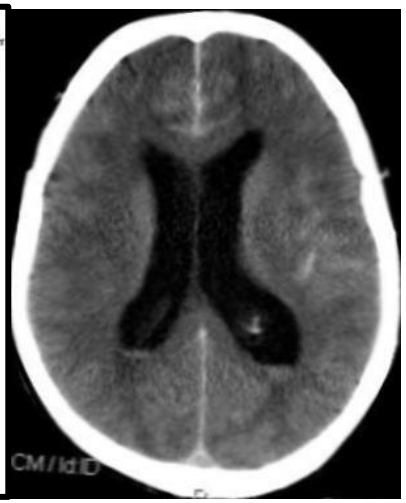
Complications (3/3)

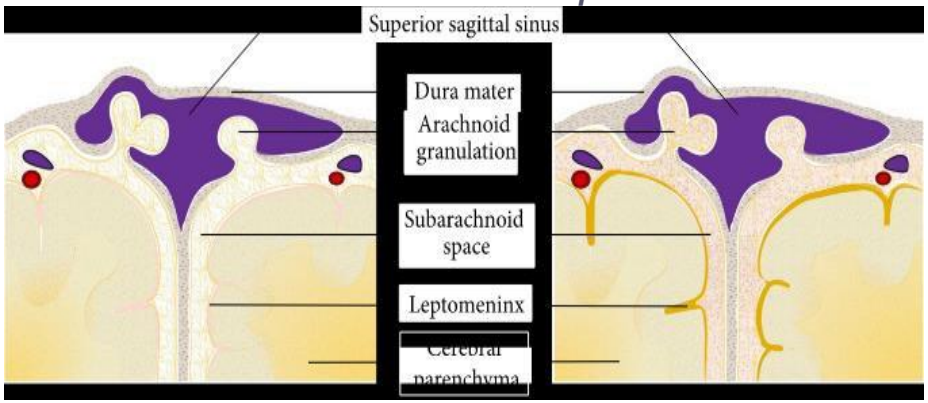
- Hydrocephalus
  - Generally non-obstructive, due to resorption blockage
  - **Early** → blood in subarachnoid space
    - *Treatment = external ventricular drainage*
    - *Possible secondary to ventricular haemorrhage = obstructive*
    - ***Late*** → *subarachnoid space fibrosis*
    - *Treatment = ventricleperitoneal shunt*









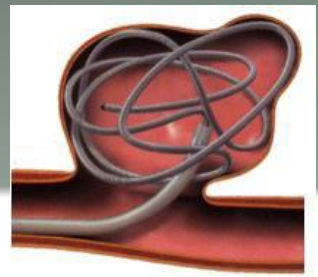


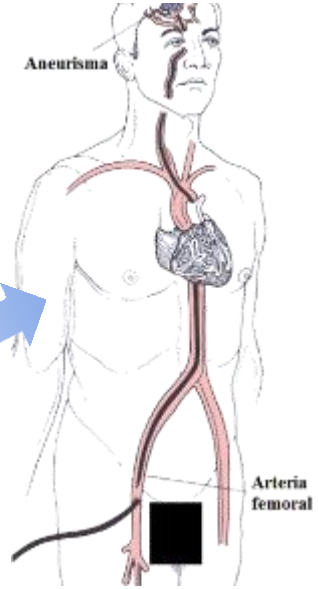
Treatment

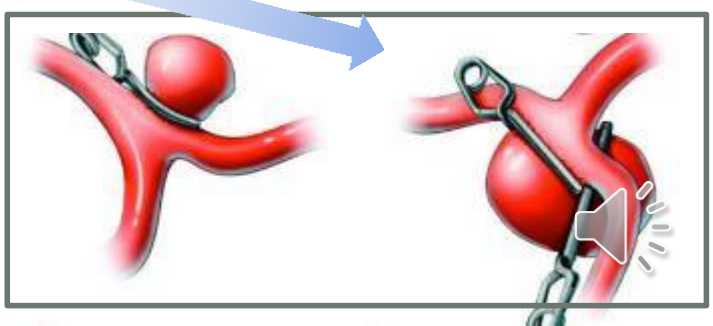
- Objective = treat the consequences of SAH and prevent rebleeding and vasospasm
- General measures (if there is no aneurysm)
  - Bed rest in a dark room, fluid therapy (or soft diet)
  - Antiemetics, pain relievers
  - Tight control of blood pressure
  - *Dexamethasone: only if uncontrolled headache or signs of ischemia*
- Specific measures
  - UCI
  - Aneurysm treatment
  - Powerful analgesia
  - Prevent / control vasospasm

Specific treatment (1/2)

- Consider UCI admission (grades Hunt-Hess > 2)
  - Airway protection, support measures
  - Possible ICP sensor or external ventricular drainage)
- Treating the cause of the bleeding (aneurysm)
  - Prevents rebleeding
  - Endovascular = prefer when doing angiography
    - *Coil embolization*
  - Surgical clipping
    - *"Emergency, in alert patients without neurological focus"*
    - *Delayed (1-2 weeks), in patients comatose, focal symptoms, or vasospasm*
- Analgesia
- Vasospasm control



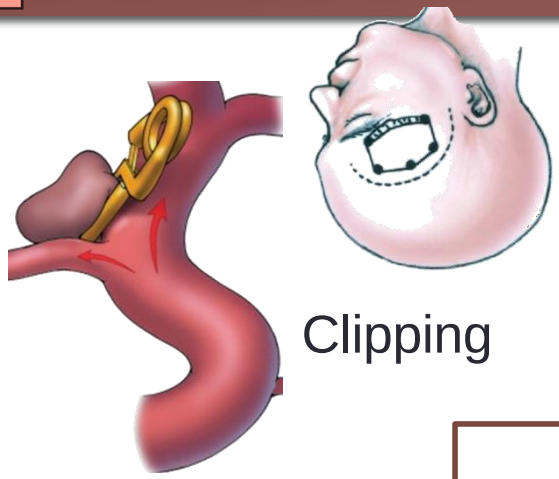




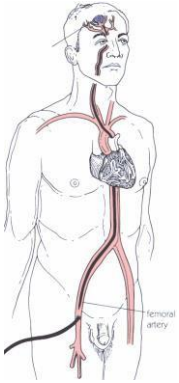
Surgical

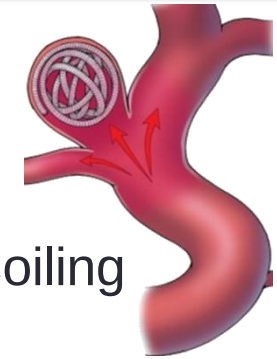
Endovascular

Saccular aneurysms

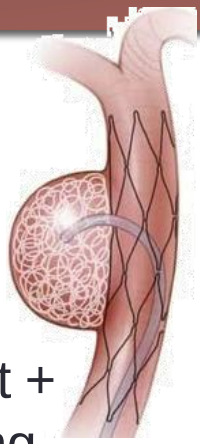


Clipping





Coiling

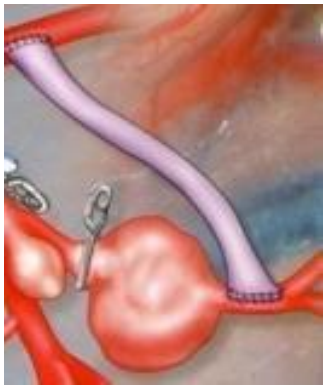


Stent + coiling

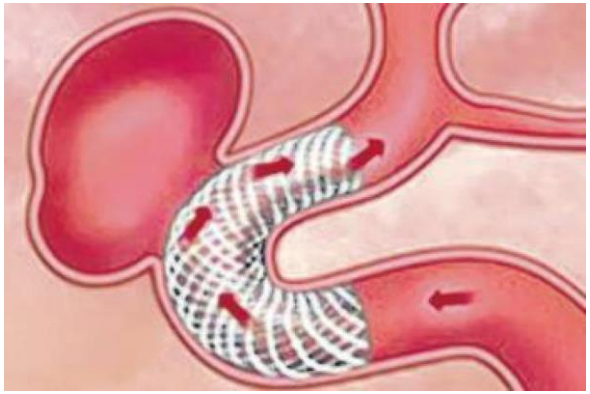
Treatment possibilities

Fusiform aneurysms

Wrapping



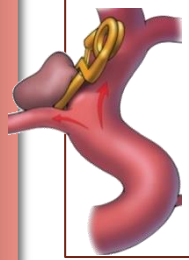
Bypass



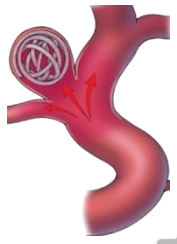
Flow diverter

Surgical versus endovascular treatment?

- **Surgical**
  - Location in **anterior** part of the circle of Willis
  - Requires craniotomy
    - *Higher initial morbidity*
    - *Cisterns washing → ↓blood → ↓vasospasm*
    - *Opening lamina terminalis → ↓risk of hydrocephalus*
  - Aneurysm obliteration ≈ 100%
  - 5-year recurrence rate 1.8 %
    - *Angio control at 5 years*

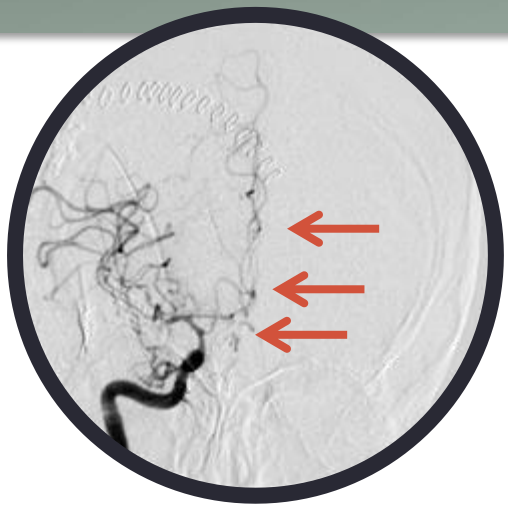


- **Endovascular**
  - Location in **posterior fossa**
  - Lower initial morbidity
    - *10 % still need craniotomy*
  - Recanalization rate 40 % at 5 years
    - *Yearly control angiography*
    - *Retreatment in 60 %*
  - Antiaggregation
    - *Coiling: 6 months*
    - *Stent flow diverters: double and lifelong*

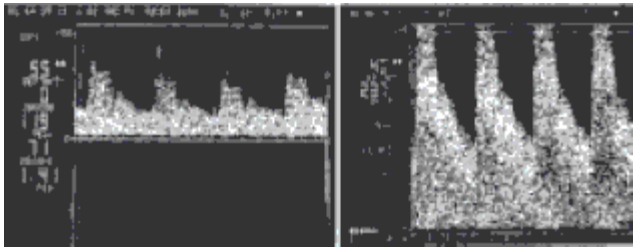


Specific treatment (2/2)

- Consider UCI admission (grades Hunt-Hess > 2)
- Treating the cause of the bleeding (aneurysm)

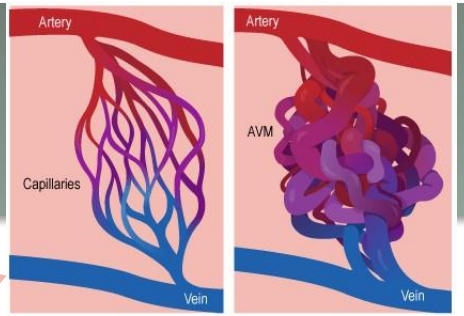


- Analgesia
  - Very painful condition, requires opiates + NSAIDs ± Dexamethasone

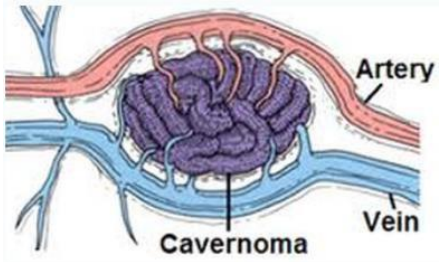


- Prevent vasospasm
  - Ca^{++} antagonists (nimodipine)
  - Optimise cerebral perfusion to achieve adequate perfusion pressure
    - *CPP = MAP – ICP*
    - *Remember the intracranial hypertension topic!*
  - If it has already occurred, consider angioplasty

VASCULAR MALFORMATIONS



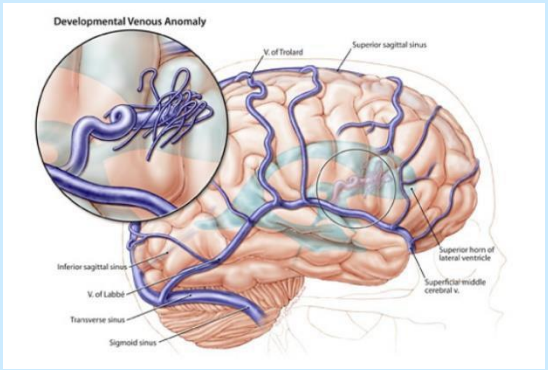
- Brain vascular malformations
  - AVM = arterio-venous malformations
    - *Dural arterio-venous fistula*
    - *Carotid-cavernous fistula*
  - Cavernous angioma (cavernoma)
  - **Venous angioma**
  - **Telangiectasias**



Capillary telangiectasias

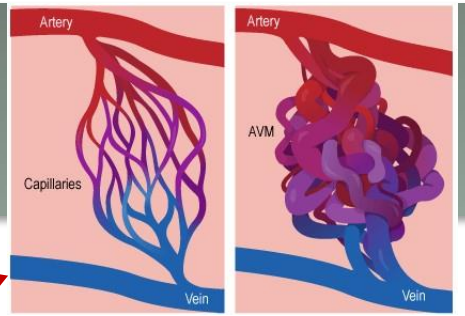
May bleed, but rarely produce a mass effect or significant symptoms

Venous angioma

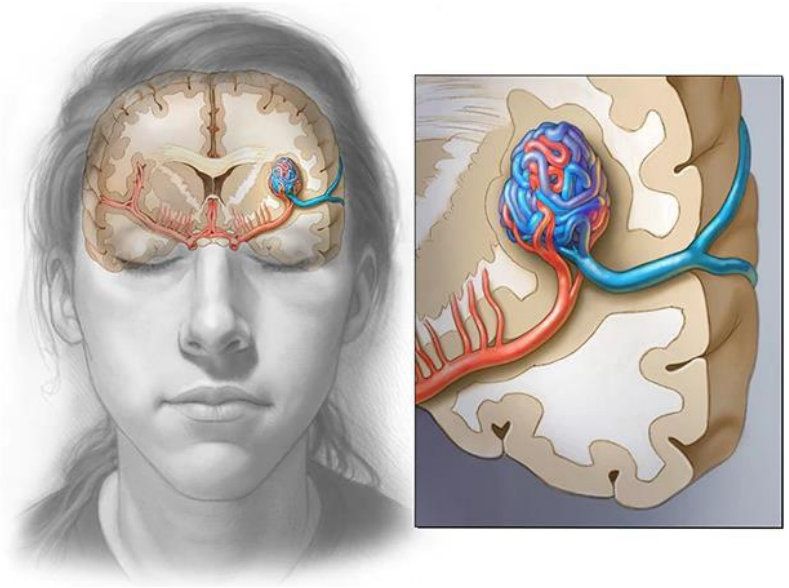


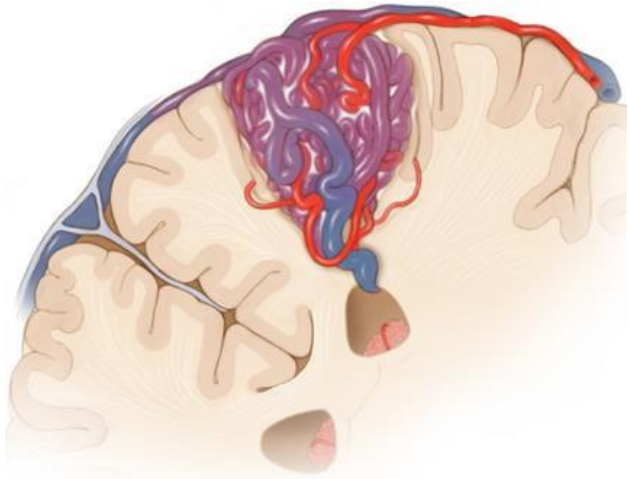
Associated with cavernomas
Asymptomatic
Low risk of bleeding

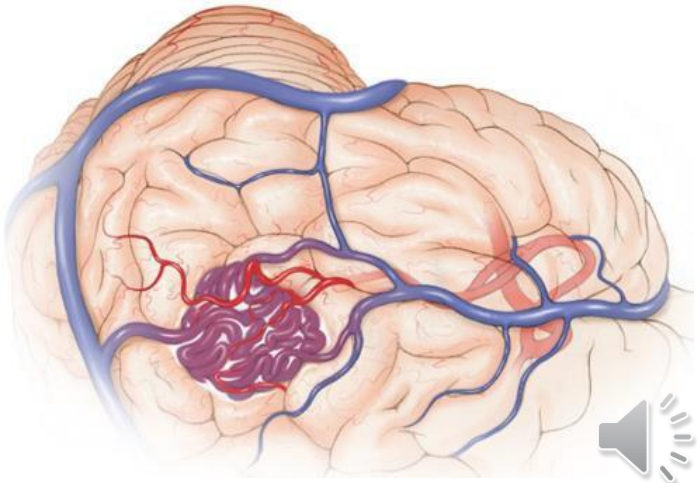
CEREBRAL AVMs



- Cerebral vascular malformations
  - **AVM = cerebral arterio-venous malformation**
    - *Dural arterio-venous fistula*
    - *Carotid-cavernous fistula*
  - Cavernous angioma (cavernoma)
  - Venous angioma
  - Telangiectasias

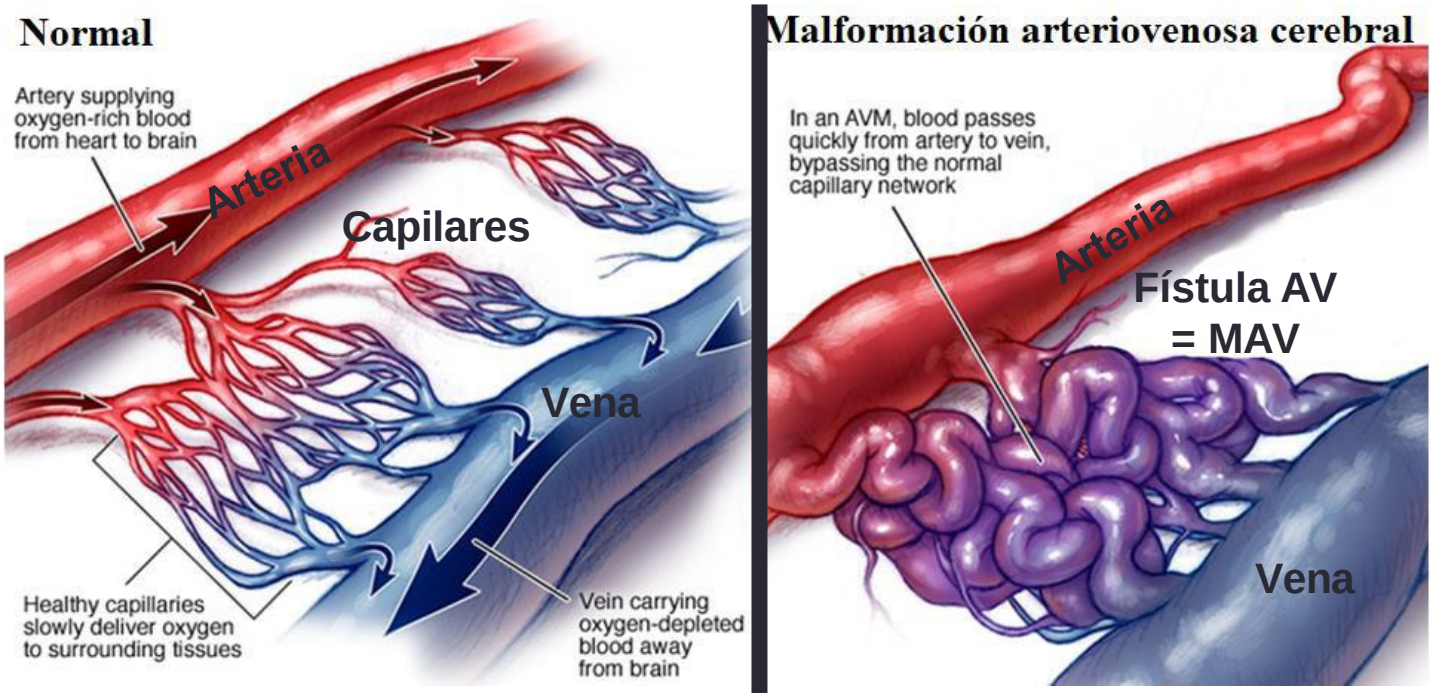






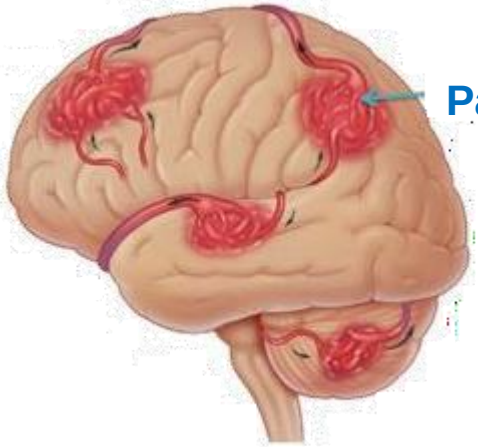
Cerebral AVMs

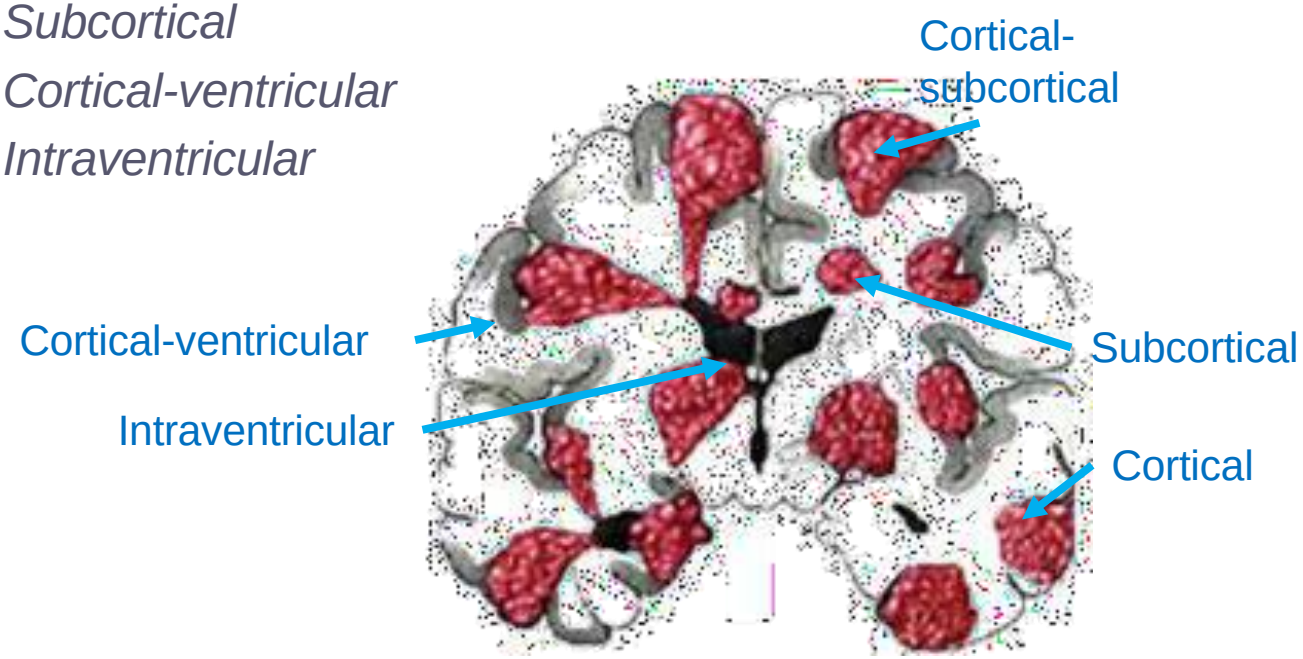
- AVM = vascular ball formed by dysplastic arteries and veins without capillary bed (with arteriovenous fistulas)
  - direct connection between artery and vein (AV fistulas)
  - no interposed capillary bed
  - no brain parenchyma within the nidus



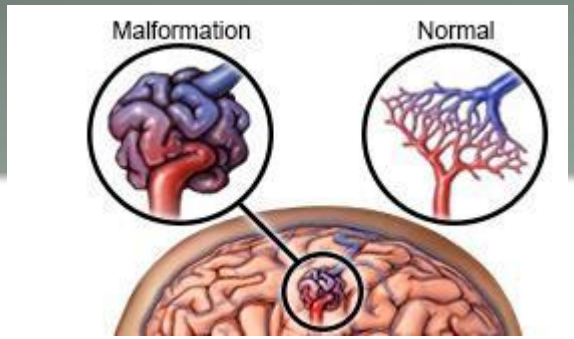
Cerebral AVMs

- Location
  - 90 % supratentorial
    - *Parietal > frontal > occipital*
  - Depth
    - *Cortical*
    - *Cortical-subcortical*
    - *Subcortical*
    - *Cortical-ventricular*
    - *Intraventricular*

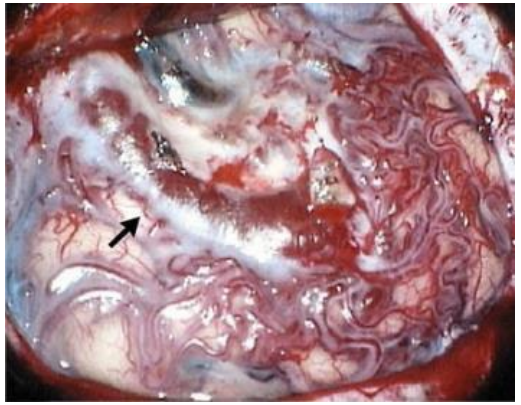




Cerebral AVMs

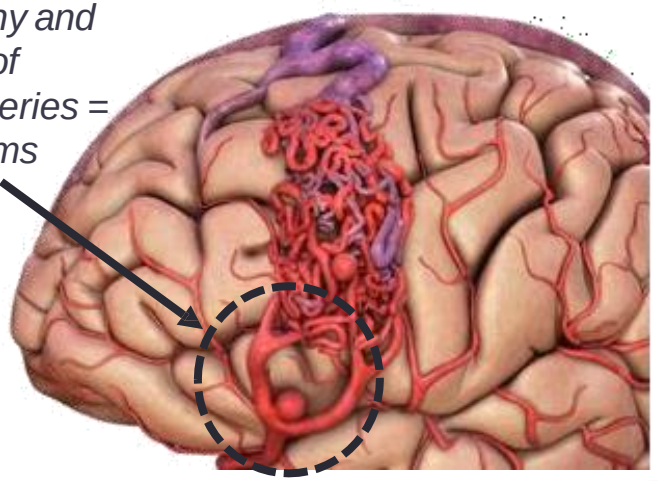


- Approximate prevalence
  - 1-2 cases / 100,000 persons (obsolete?)
  - 15 / 100,000 persons (in Scotland)
  - *It is usually said to be 5 times less frequent than aneurysms, but it is not known with certainty*



- Diagnosis in young people, 20 – 40 years
  - 2/3 diagnosed < 30 years
  - Somewhat more frequent in ♂

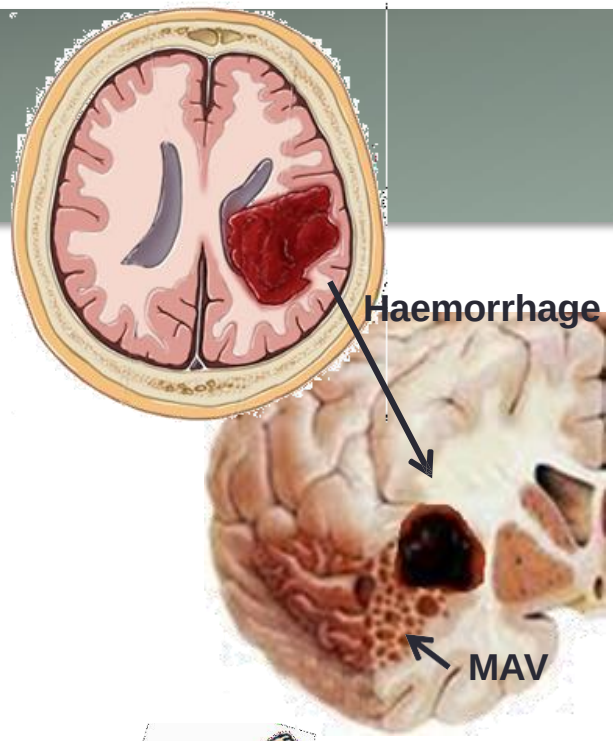
Hypertrophy and dysplasia of feeding arteries = ↑aneurysms

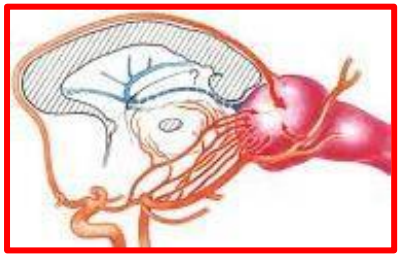


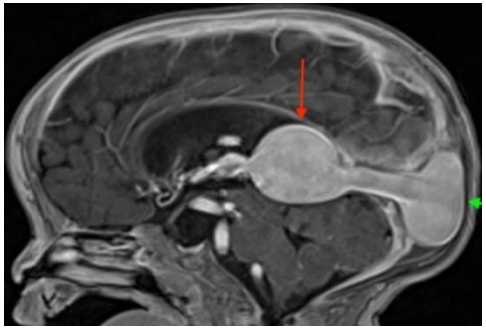
- Congenital
  - 10 – 15 % Rendu-Osler syndrome (hereditary haemorrhagic telangiectasia) harbour cerebral AVMs

Cerebral AVMs

- Clinical features ⇨ *Young men (20 years old) with a history of epilepsy presenting with brain hemorrhage*
  - Hemorrhage (50 %)
    - *Less explosive headache than SAH*
    - *Intraparenchymal (80 %)*
    - *Mortality 10 %, morbidity 40 % (focal deficit)*
    - *Risk of new rupture 20 % (3 % per year)*
  - Epileptic seizures (30 %)
    - *More frequent with age*
      - Frequent form of presentation in young patients
    - *With transient neurological deficit*
  - Headaches
  - Progressive focal deficits (ischemia focal)
  - Children: hydrocephalus with macrocephaly and congestive heart failure







Cerebral AVMs

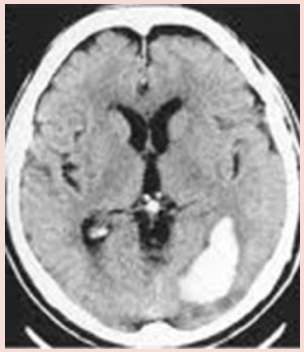
- Factors related to bleeding in AVMs

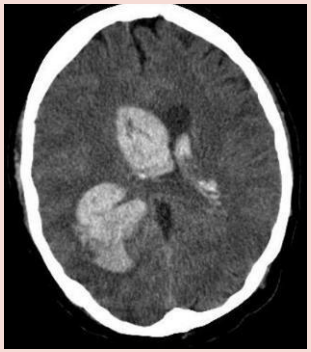
If no bleeding :

- Annual risk 2 – 5 %
  - *If epilepsy, lower risk of bleeding than with other symptoms*
- Risk factors for bleeding
  - *Younger age (< 30 years)*
  - *Women*
  - *Spetzler-Martin grades III-V*

If previous bleeding :

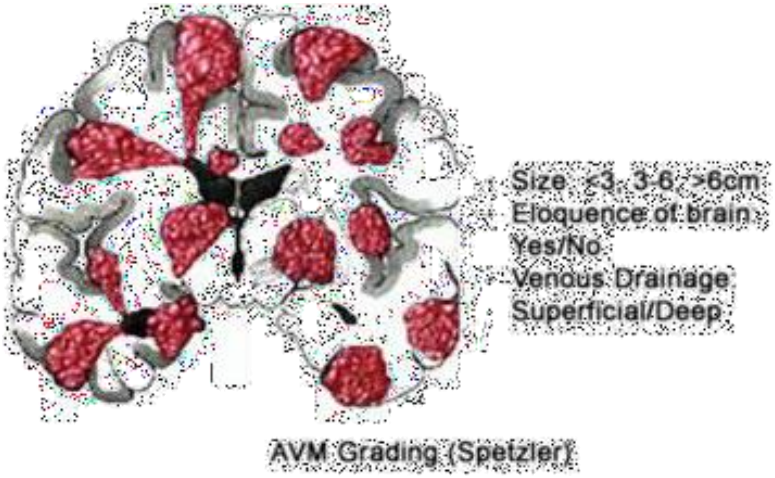
- Annual risk 4 – 12 %
- Risk factors for rebleeding
  - *Deep location*





Cerebral AVMs

- **Spetzler-Martin classification (1986)**



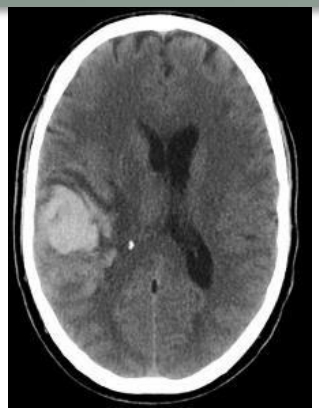
Criterion	Score
Nidus size	
• Small (< 3cm)	1
• Median (3-6 cm)	2
• Big (> 6 cm)	3
Elocuence	
• No	0
• Yes	1
Deep venous drainage	
• No (superficial)	0
• Yes (deep)	1
Total score =	**AVM grade**

I – II = Low grade
III – V = High grade → high risk of bleeding

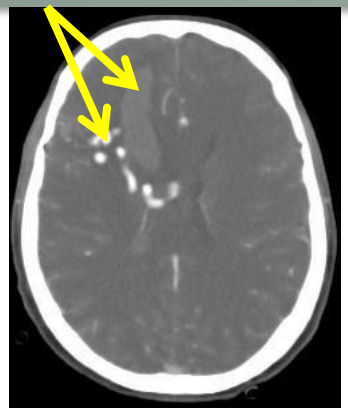
Eloquence = sensory-motor cortex, visual cortex, language cortex, hypothalamus, thalamus, brainstem, cerebellar nuclei, or regions adjacent to these structures

Cerebral AVMs

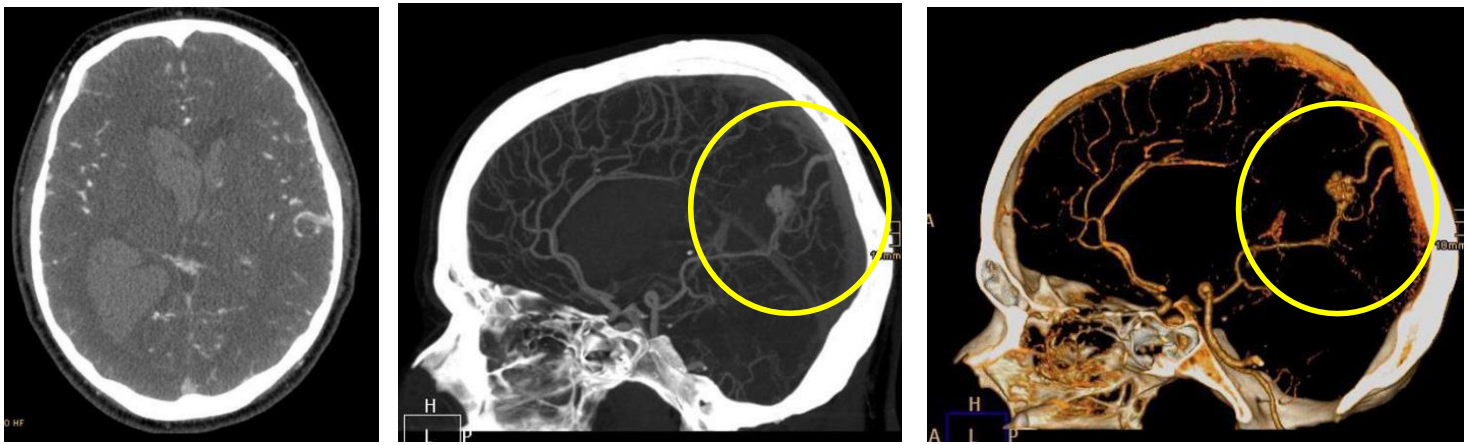
- Diagnosis according to clinic
  - If bleeding → head CT-scan
    - *Hemorrhage, calcifications*
    - *CT-scan C+ → The big ones enhance with C+*
    - *Angio-CT-scan → Good visualization*
  - MRI
  - First choice = Angiography



Plain CT. AVM bleeding. Differential diagnosis with other space-occupying lesions

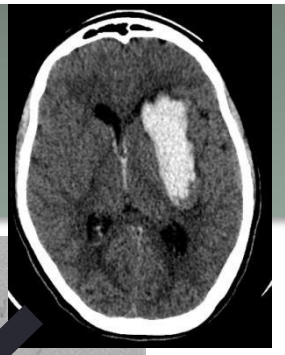


CT-scan C +. Hemorrhage and AVM, with rapid passage of C+

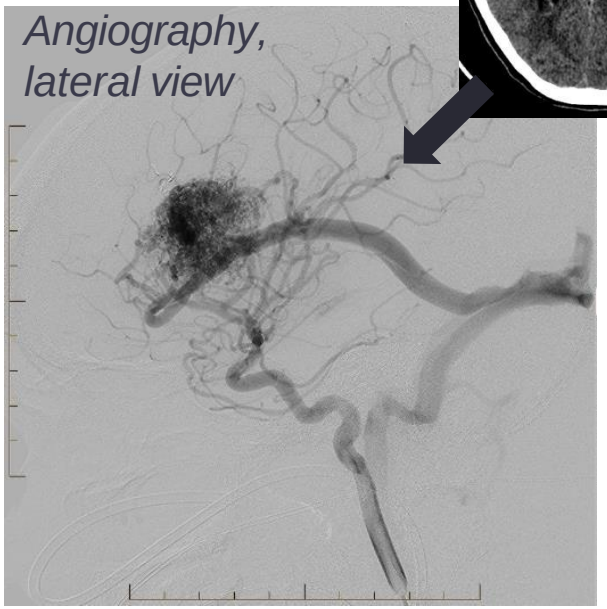


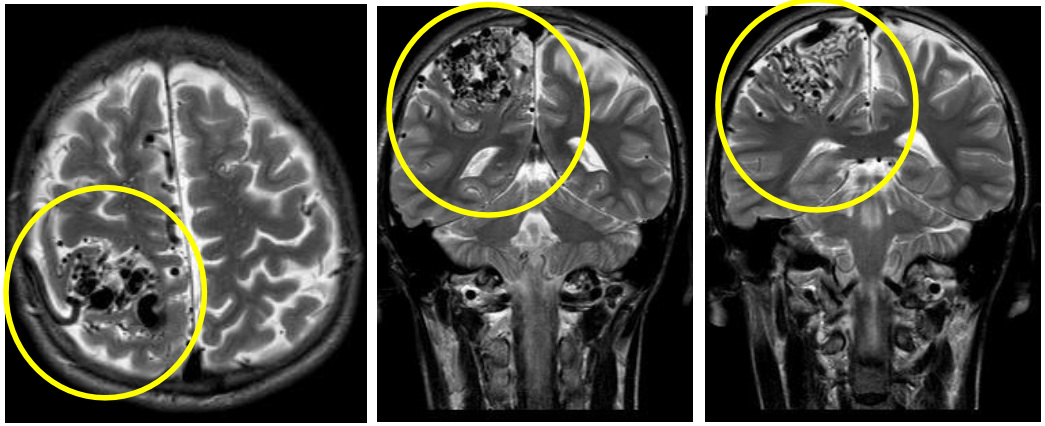
CT-scan C+ and plain Angio-CT-scan and with 3D reconstruction. AVM bleeding

Cerebral AVMs



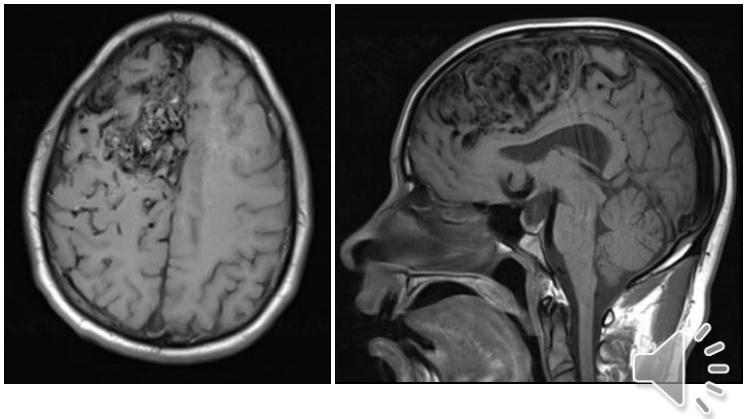
- Diagnosis
  - If haemorrhage→ head CT-scan
  - MRI
    - *Better anatomical visualization*
  - First choice = Angiography
    - *Identifies afferent vessels and drainage*
    - *Allows treatment (embolization)*





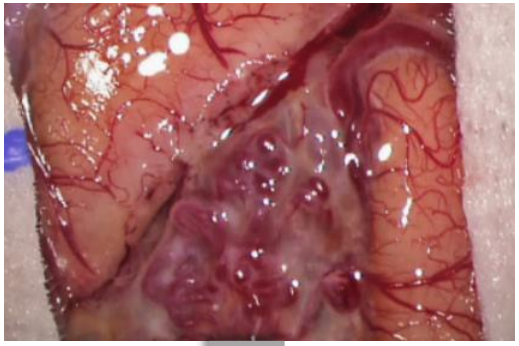
MRI T2, axial and coronal planes (afferent and efferent vessels can be seen)

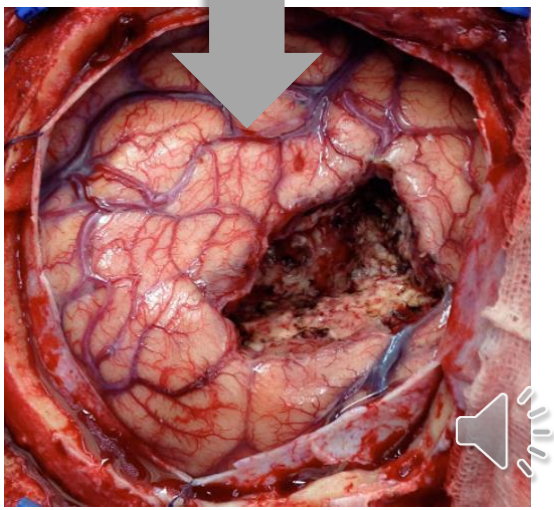
MRI T1 axial and sagittal



Cerebral AVMs

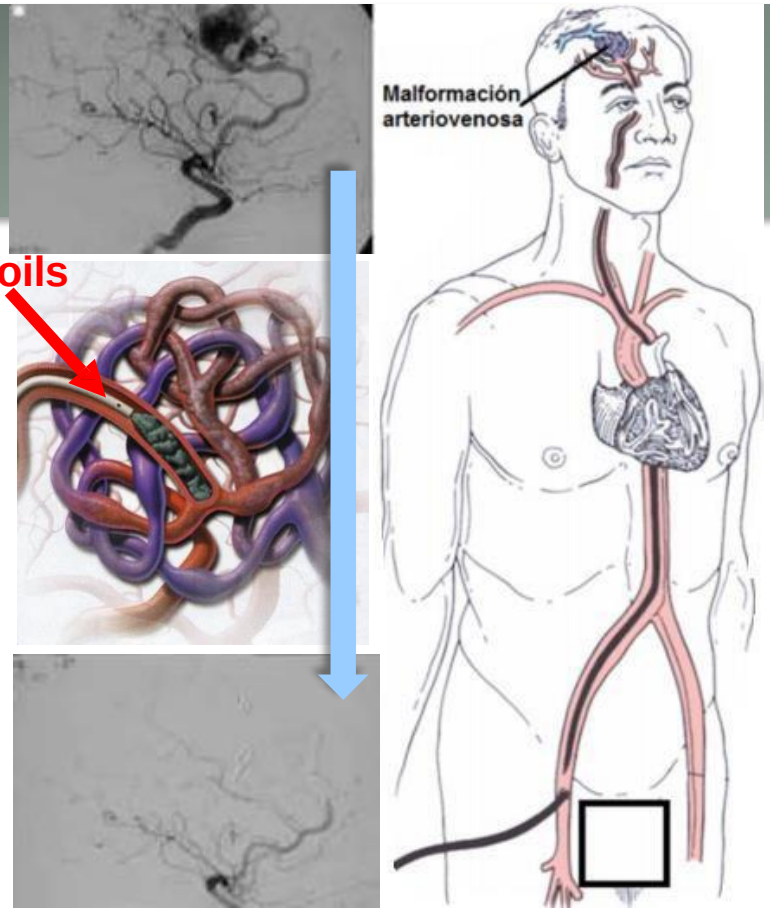
- Treatment
  - <u>All must be treated due to risk of bleeding</u>
    - *Exclude ALL vascular bed*
  - *Accessible symptomatic AVMs → surgery preceded by embolization*
  - Surgery
    - *Allows removal in almost 100% cases*
    - *0 – 15 % mortality*
    - *Indication: small or medium cortical AVMs (grades I-II)*
    - *Rest, combined treatment with interventional radiology and radiotherapy-radiosurgery*
  - Interventionism: embolization
  - Radiotherapy-radiosurgery

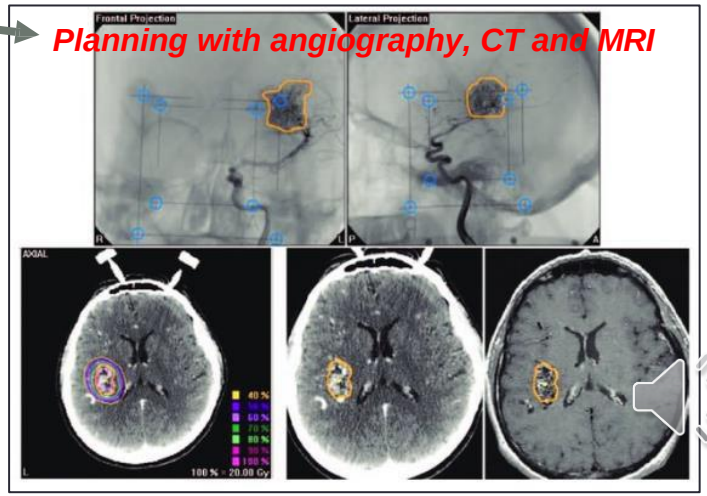




Cerebral AVMs

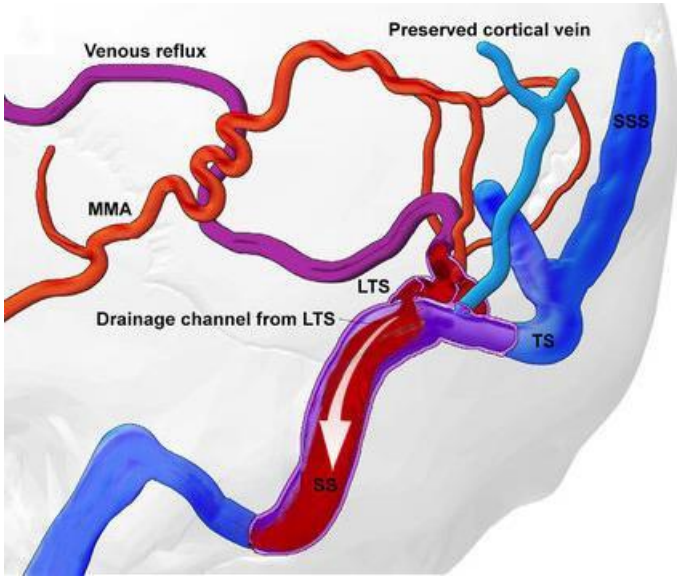
- Treatment
  - Surgery
  - Interventionism: embolisation
    - *Definitive treatment (complete obliteration) ONLY in15 % cases*
    - *Mortality 2 – 5 %*
    - *Reduces Ø AVM*
    - *Applied before surgery or radiosurgery*
  - Radiotherapy-radiosurgery
    - *AVM < 3 cm → 70 % total obliteration takes 2 – 3 years*
    - *While it is not closed, the risk of rebleeding continues to accumulate*
    - *Indicated in small AVMs of deep location or in eloquent areas*

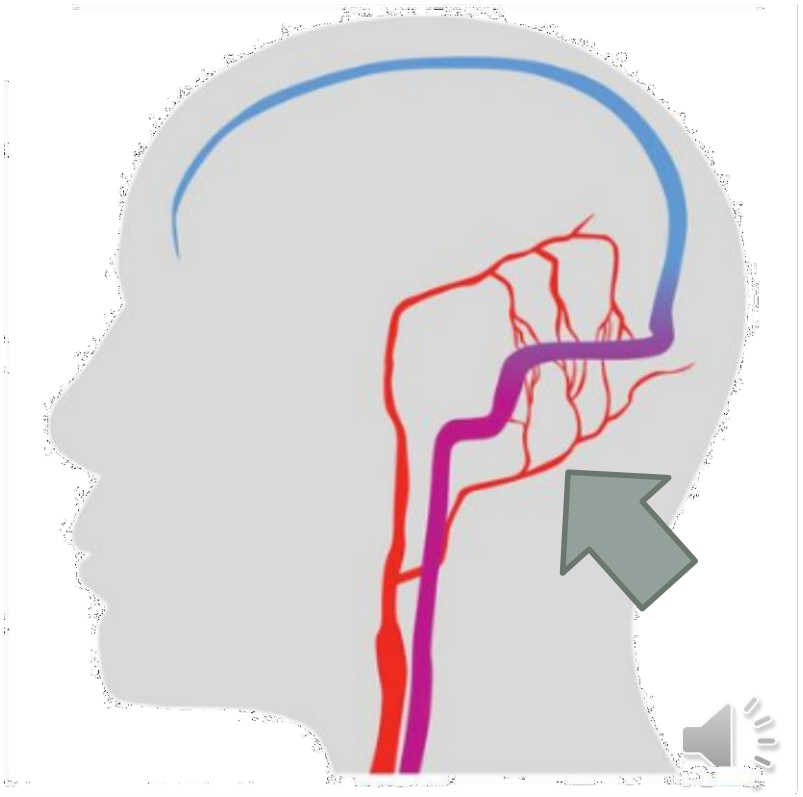




DURAL ARTERIO-VENOUS FISTULA

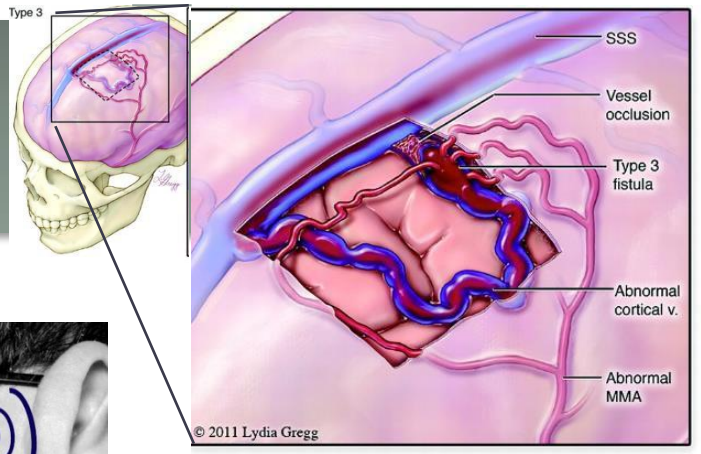
- Cerebral vascular malformations
  - AVM = arterio-venous malformations
    - ***Dural arterio-venous fistula***
    - *Carotid-cavernous fistula*
  - Cavernous angioma (cavernoma)
  - Venous angioma
  - Telangiectasias

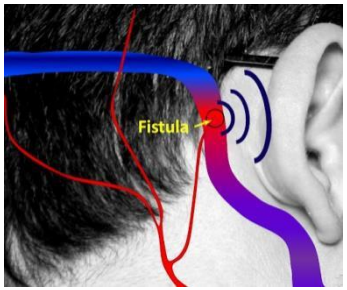




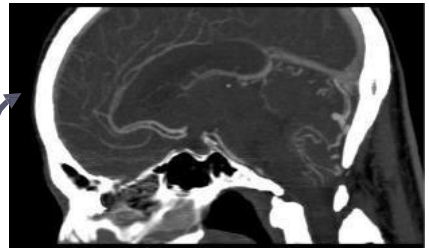
Dural AV fistula

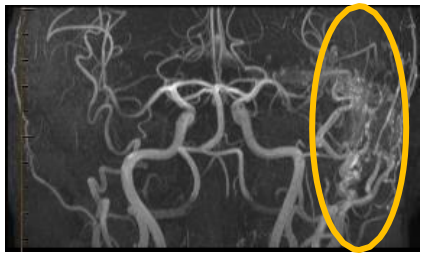
- Fistula between dural artery and cavernous sinus
  - Acquired injury: idiopathic, post-traumatic, sinus venous thrombosis …
- Clinical features
  - Headache
  - Pulsatile tinnitus, "noises in the head"
  - If venous pressure very ↓, possible cortical ischemia (steal) or cortical haemorrhage (sinus venous rupture)
- Treatment surgery / endovascular

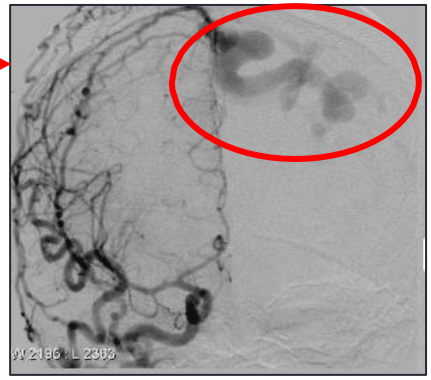


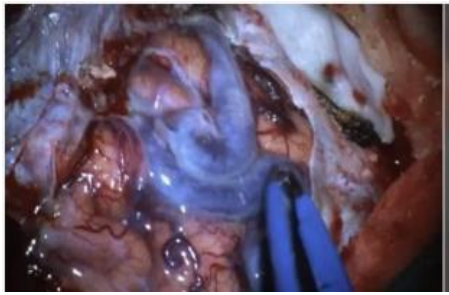


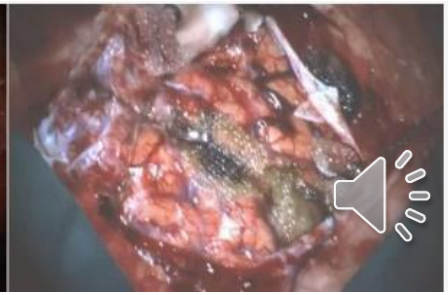
Diagnosis:
- Angio-CT-scan
- Angio-MRI
- Angiography





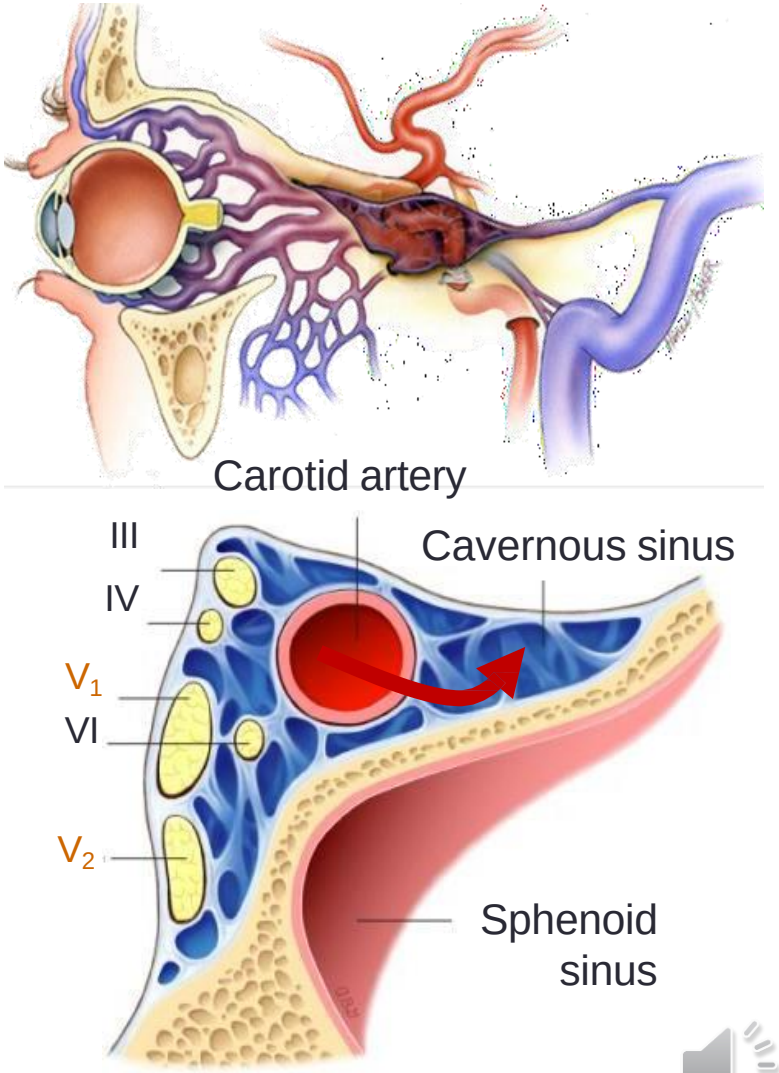






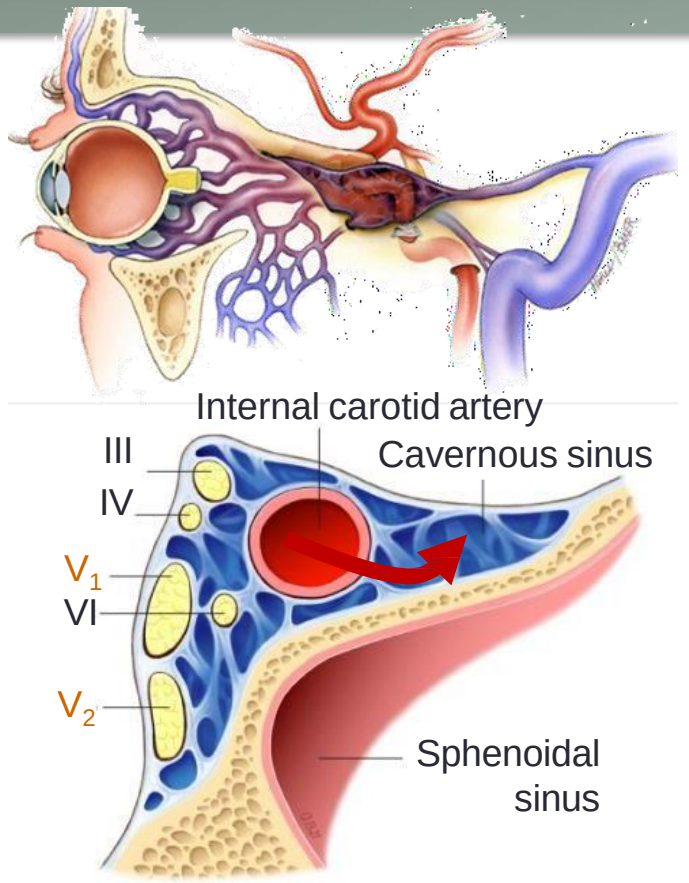
CAROTID-CAVERNOSA FISTULA

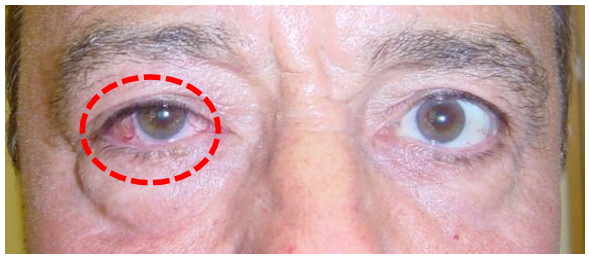
- Cerebral vascular malformations
  - AVM = arterio-venous malformations
    - ***Dural arterio-venous fistula***
    - ***Carotid-cavernous fistula***
  - Cavernous angioma (cavernoma)
  - Venous angioma
  - Telangiectasias

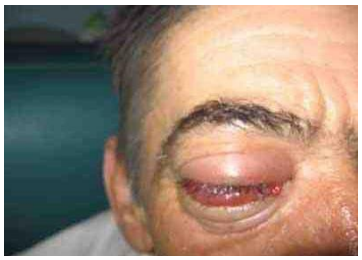


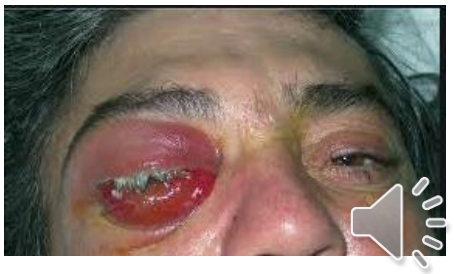
Carotid-cavernous fistula

- Communication between carotid and cavernous sinus
  - "Spontaneous" = dural fistula
  - Ruptured intracavernous aneurysm
  - Traumatic
- Clinic (acute)
  - Retroocular murmur (thrill) / mastoid
  - Exophthalmos
  - Conjunctival injection (chemosis)
  - Impairment cranial nerves III, IV, VI and $V_{1\text{-}2}$
- Possible haemorrhage
- Diagnosis
- Treatment



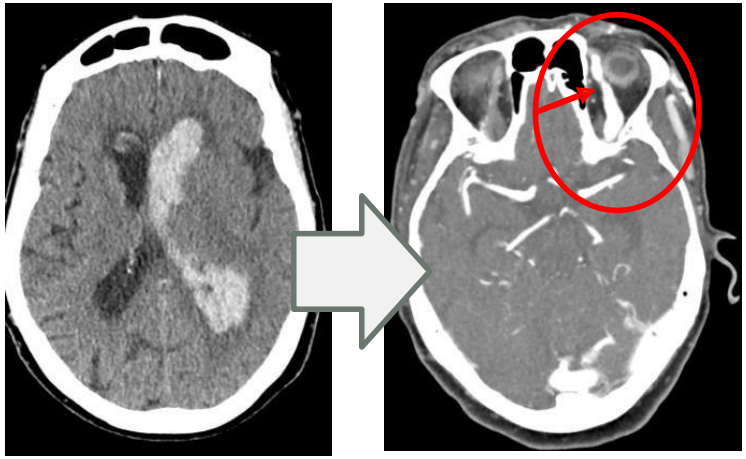




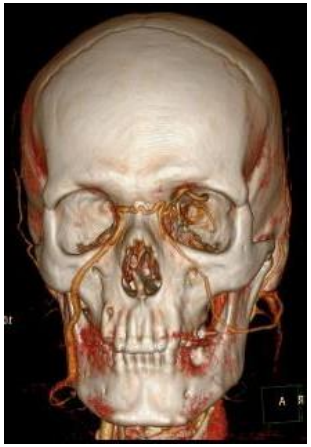


Carotid-cavernous fistula

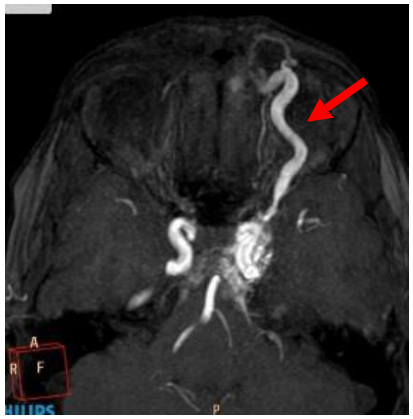
- Communication between carotid artery and cavernous sinus
- Clinic (acute)
- Possible haemorrhage!
- Diagnosis
  - Angio-CT-scan
  - Angio-MRI
  - Angiography (first choice)
- Treatment
  - Possible spontaneous remission
  - Embolization > radiosurgery
  - Surgery
    - *If other treatments fail*
    - *In case of hematoma*



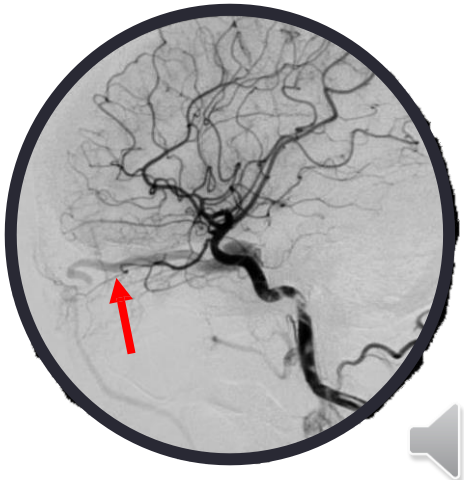
Plain CT-scan and Angio-CT-scan. Left ophthalmic vein engorged during arterial phase



Angio-CT-scan 3D reconstruction



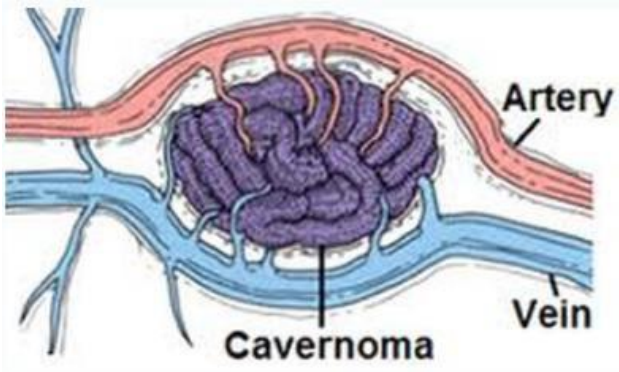
Angio-MRI. Left ophthalmic vein engorged

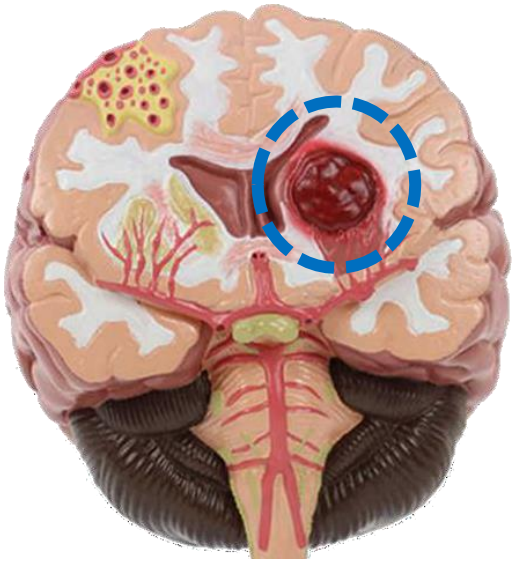


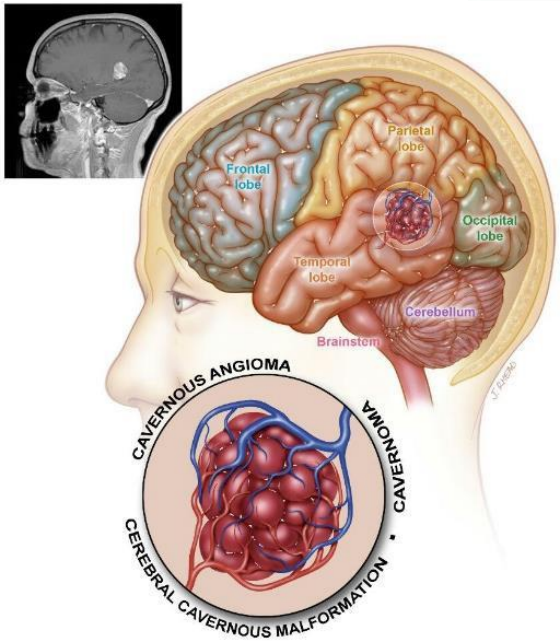
Angiography. Ophthalmic vein filling in arterial phase

CAVERNOUS ANGIOMA

- Cerebral vascular malformations
  - AVM = arterio-venous malformations
    - *Dural arterio-venous fistula*
    - *Carotid-cavernous fistula*
  - **Cavernous angioma (cavernoma)**
  - Venous angioma
  - Telangiectasias

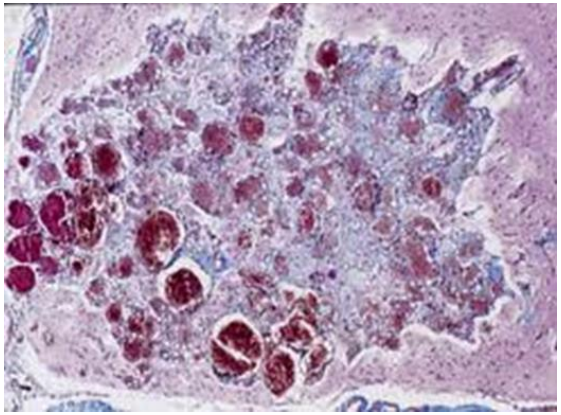






Cavernous angioma

- Sinusoidal vascular channels with no tissue between them
  - Family incidence, especially if they are multiple (multiple cavernomatosis)
  - Small size 1- 3 cm
  - LOCALIZATION ON THE ENTIRE CNS (including spinal cord)
  - Flow ⇓→ Angiographically negative
- Features
  - Endothelium-lined honeycomb vascular spaces separated by bands of collagen
  - No nerve tissue inside them
  - Well delimited
  - Calcifications, hemosiderin (bleeding)
    - *May ↑ Ødue to bleeding inside them (rare)*

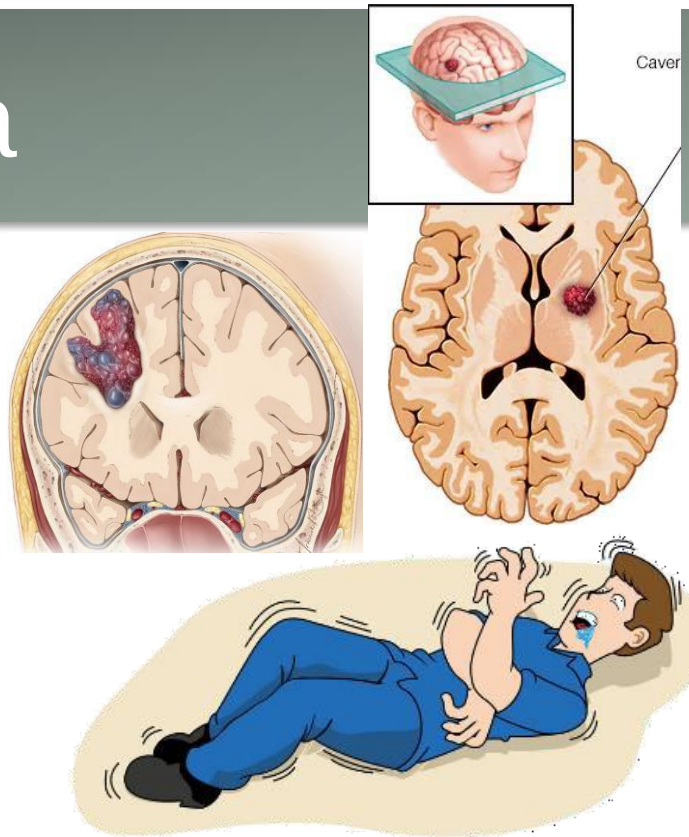


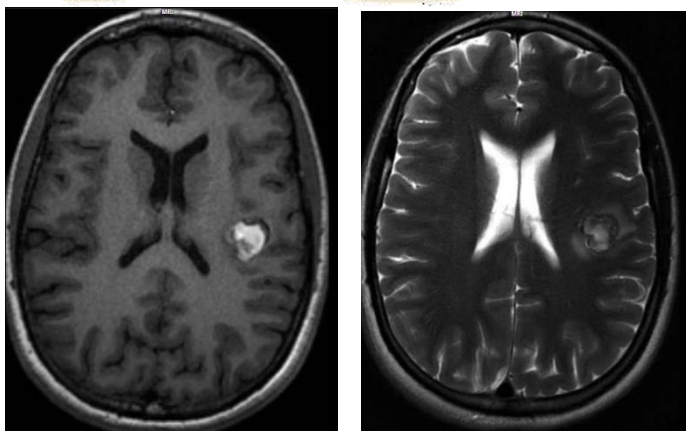
Microscopic image of cavernous angioma

Gross image ("berry"), raspberry

Cavernous angioma

- Clinical features
  - Epilepsy (70 %)
    - *Often drug-resistant*
  - Haemorrhages (10 %)
    - *Annual cumulative risk 0.5 – 1.5 %*
    - *Higher risk in ♀ and if located in the thalamus or brain stem*
    - *Spinal cord and brainstem → progressive neurological deficit*
    - *Cerebral → micro-bleedings → epilepsy*
  - Headache
  - Cranial nerve deficit, memory / attention loss
  - *In spinal cord, limb weakness*

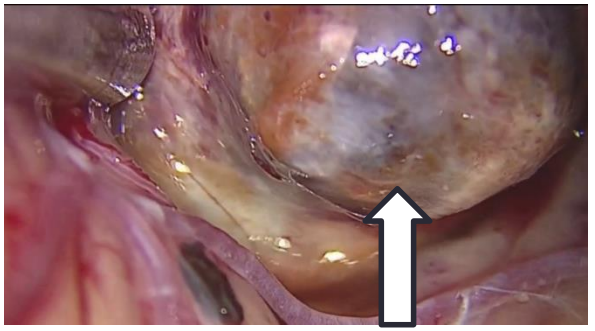




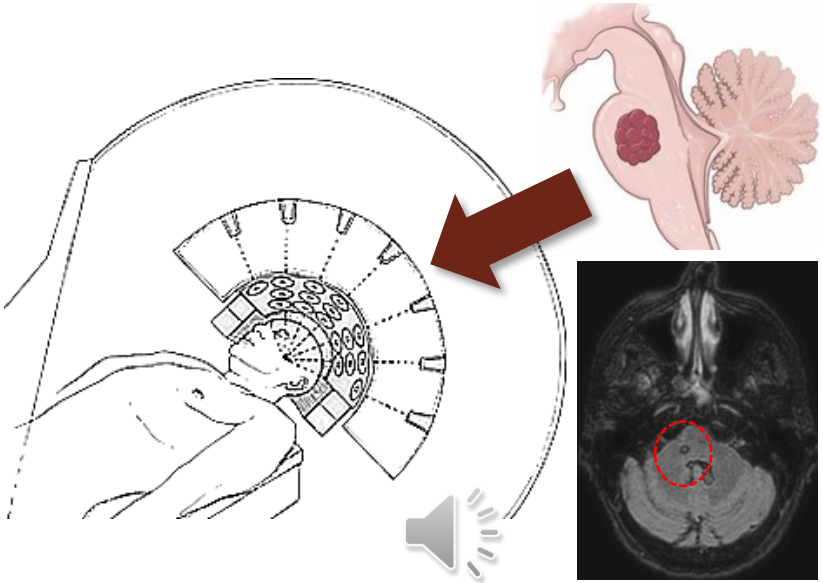
MRI axial T1 (left) y T2 (right). Cavernous angioma with recent bleeding

Cavernous angioma

- Diagnosis = MRI
  - Popcorn image with hemosiderin halo
- Treatment = surgical
  - Indication for the risk of severe bleeding and epilepsy
  - To take into consideration:
    - *More cavernomas = greater risk of bleeding*
    - *Localization → easier on the cerebral cortex*
    - *Other vascular abnormalities (40% with venous malformations)*
- Radiosurgery ↓risk of bleeding (radiotherapy ineffective)

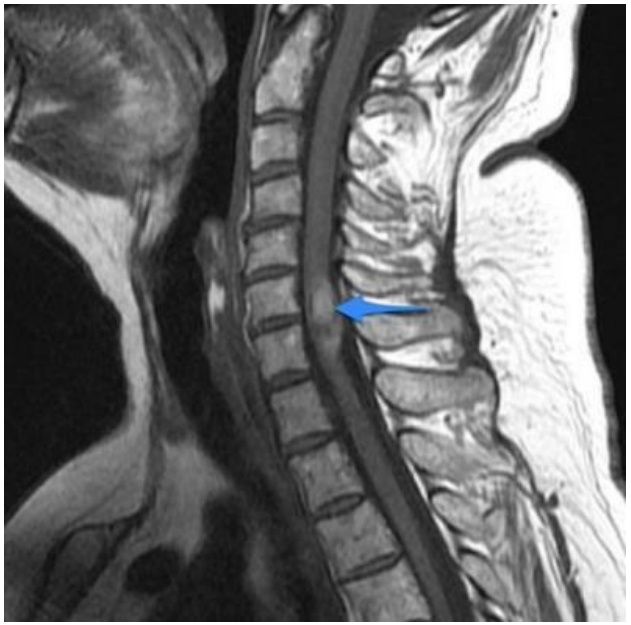


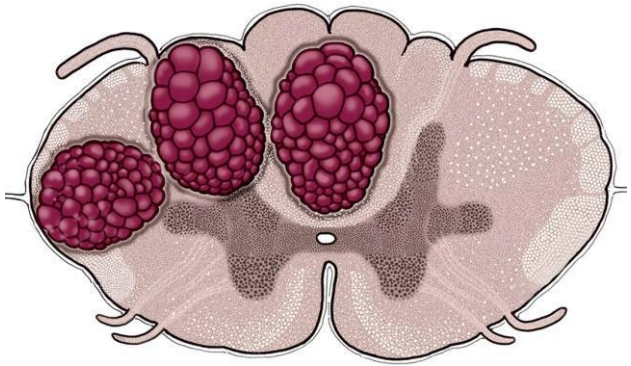
Surgical removal cerebellar cavernoma



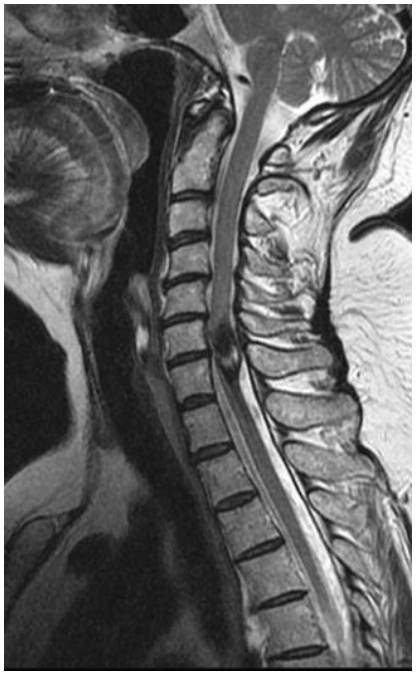
SPINAL CORD VASCULAR MALFORMATIONS

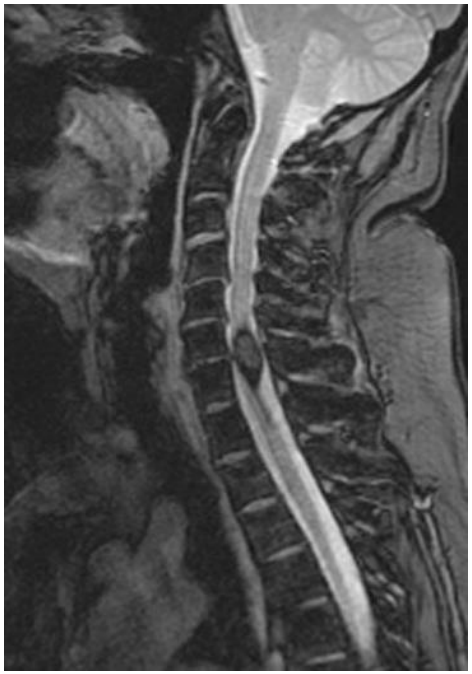
- Spinal cord vascular malformations
  - **Cavernous angioma**
  - AVM and dural fistula
  - Repeated bleeding
  - Acute or subacute loss of spinal function
  - Possible complete spinal cord injury
  - Young people
  - Diagnosis = MRI
  - Treatment = Surgical removal



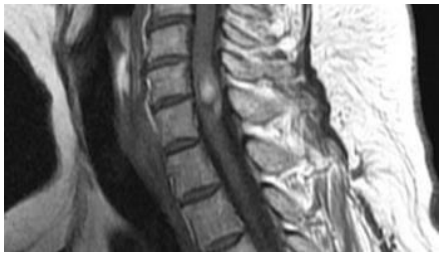


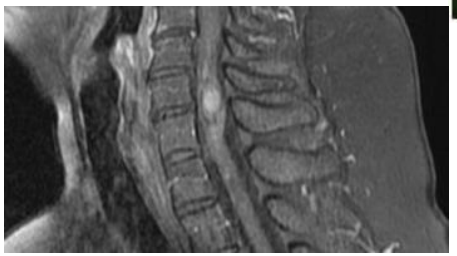
Cavernous angioma



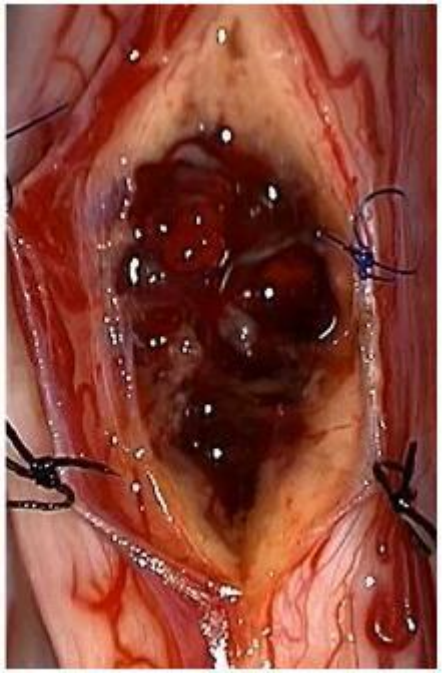


Spinal cord cavernous angioma. MRI sagittal T2 (left) and eco gradient (right).
As it has little flow, it will not capture contrast (below, T1 –left- y T1 C+ fatsat –right-)





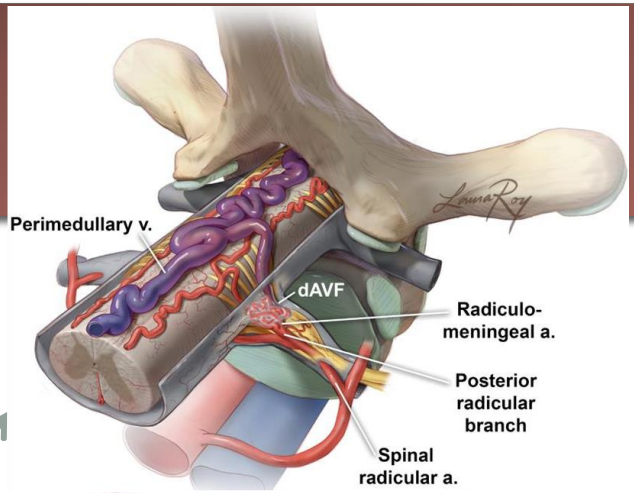
Intraoperative appearance, like a raspberry

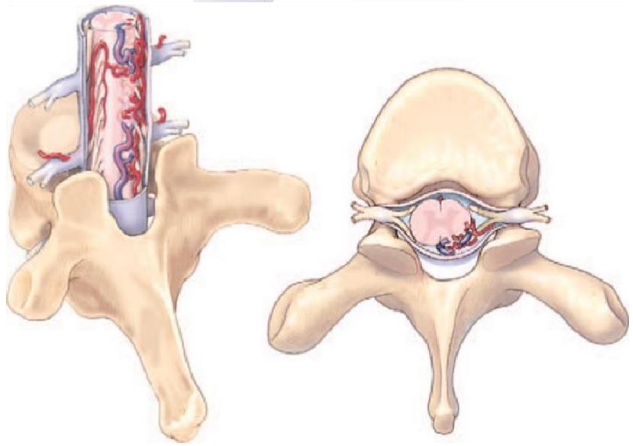


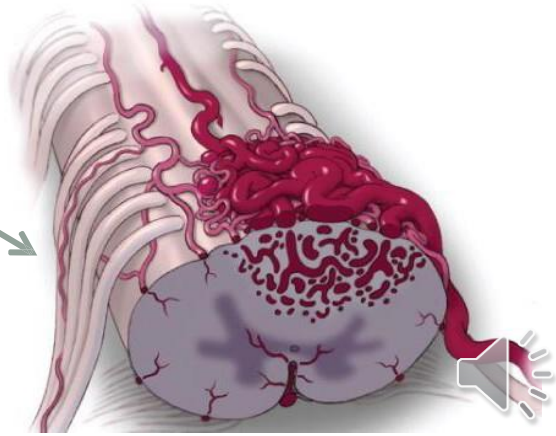
Spinal cord AVMs

- Spinal cord vascular malformations
  - Cavernous angioma
  - **AVM and dural fistula**

  - Males ~ 50 years (20-60 years)
  - Types
    - *Dural arterio-venous fistula*
    - *Perimedullary AVM*
    - *Intramedullary AVM*
  - Blood steal from normal spinal tissue + ↑ medullary vein tension



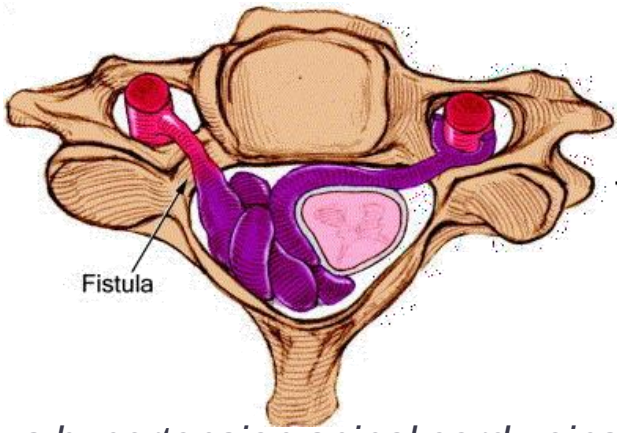




Spinal cord AVMs

- Spinal cord vascular malformations
  - Cavernous angioma
  - **AVM and dural fistula**

  - Clinical features
    - *Gait disturbance (spastic paraparesis, ataxia, limb weakness)*
    - *Nonspecific spinal pain*
    - *Possible subarachnoid or intraspinal hemorrhage (7 – 30 %)*
  - Diagnosis
  - Treatment

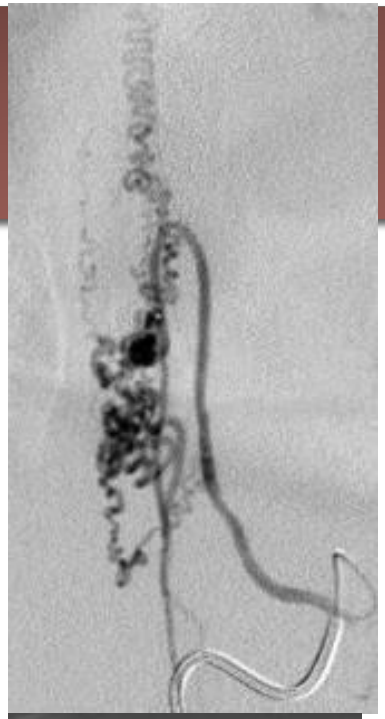


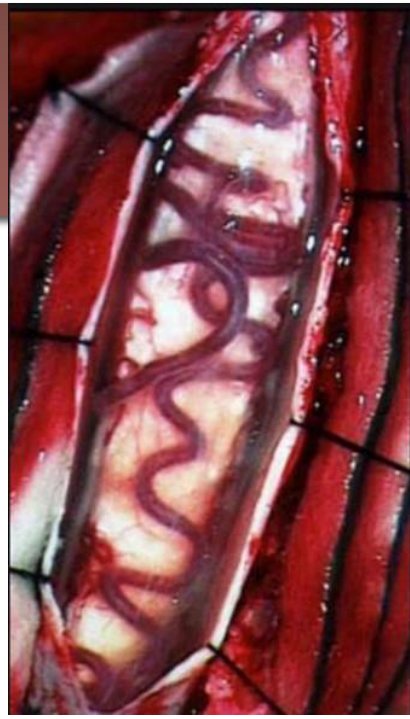
Venous hypertension spinal cord veins

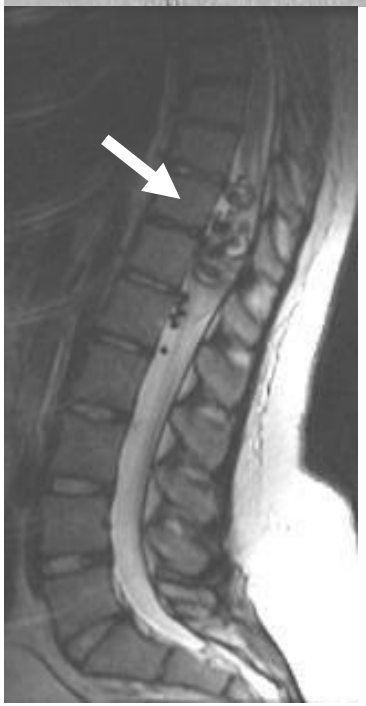
Spinal cord AVMs

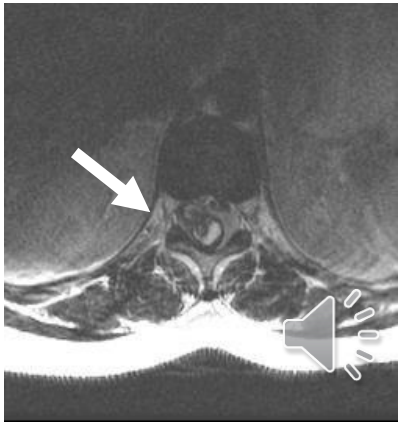
- Spinal cord vascular malformations
  - Cavernous angioma
  - **AVM and dural fistula**

    - Clinical features
    - Diagnosis
      - *Angiography = gold standard*
      - *MRI and angio-MRI*
    - Treatment
      - *Dural fistula = SURGERY*
      - *Other spinal cord AVMs = endovascular, surgical, or combined, all with risk of inducing spinal cord damage*



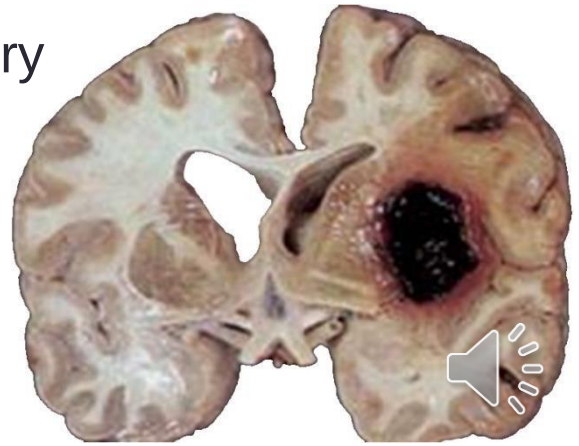






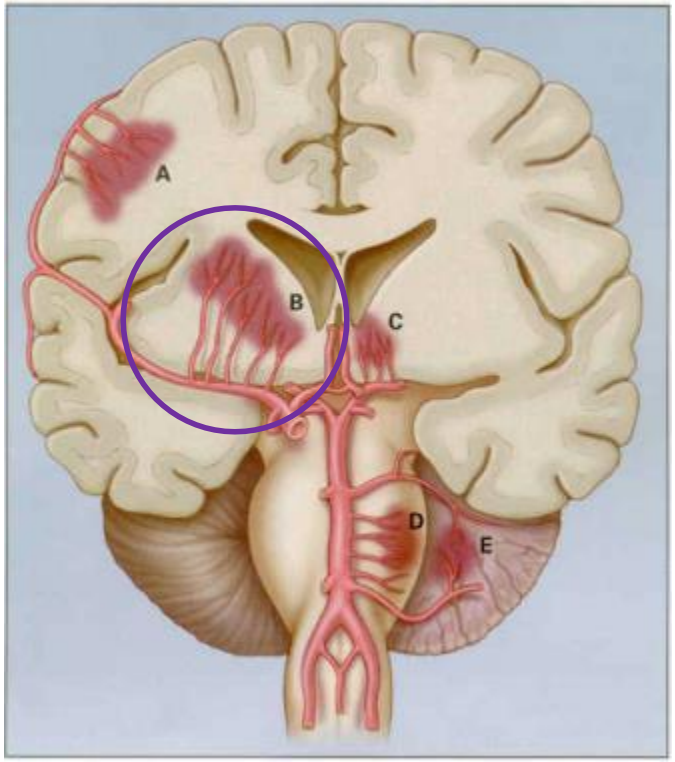
SPONTANEOUS INTRACEREBRAL HEMATOMAS

- Importance of intraparenchymal haemorrhage
  - Represents 10 % of strokes
  - Most common type of nontraumatic intracranial haemorrhage
    - *24 / 100,000 persons / year*
  - Most frequent etiology: arterial hypertension ← PREVENTION
  - Major morbidity: 80% survivors are left with neurological deficits
  - Significant mortality (40-45%) that has not improved in the last 20 years
- Features = like any other haemorrhagic stroke
  - Clinical features not limited to a vascular territory
  - Evolves in minutes (not sudden)
  - Raised intracranial pressure symptoms
    - *Headache, nausea, vomiting*
  - Diagnostic test of choice: head CT-scan



Intracerebral hematoma

- Etiology
  - Primary (spontaneous)
    - *Small vessel damage (high blood pressure, amyloid angiopathy)*
  - Secondary
    - *AVMs, aneurysms, coagulopathies, trauma, tumours, haemorrhagic transformation of ischemic stroke …*
- Localization
  - **High blood pressure** → tend to be deep (**penetrating arteries)**
  - Superficial → other etiologies



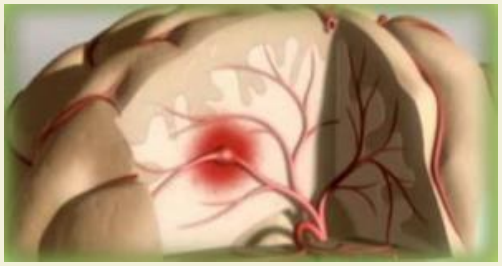
PENETRATING ARTERIES
The lenticlestriated vessels are the most frequently affected (thalamus and basal ganglia)
> lobar white matter
> pons, cerebellum

Intracerebral hematoma

- Intracerebral hematoma pathogenesis due to high blood pressure
  - Charcot-Bouchard microaneurysm rupture in penetrating arteries

HTA

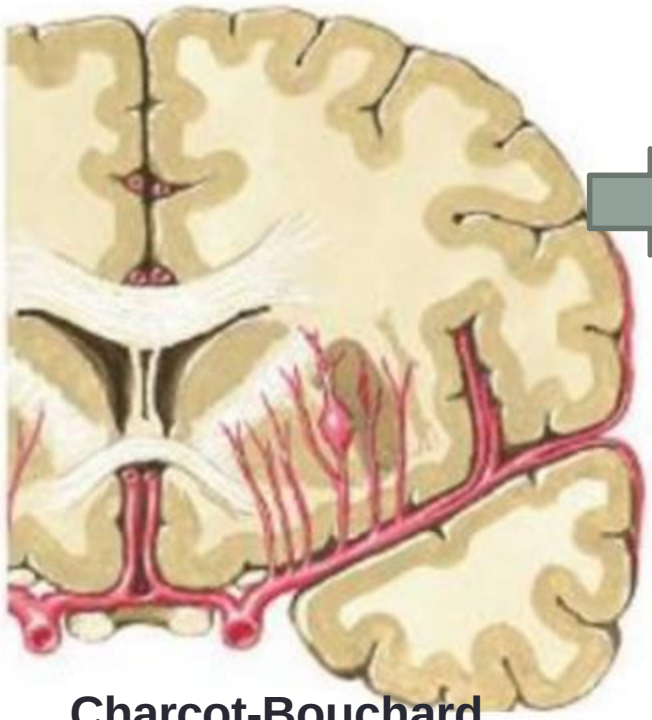
Segmental lipohyalinosis in small vessels



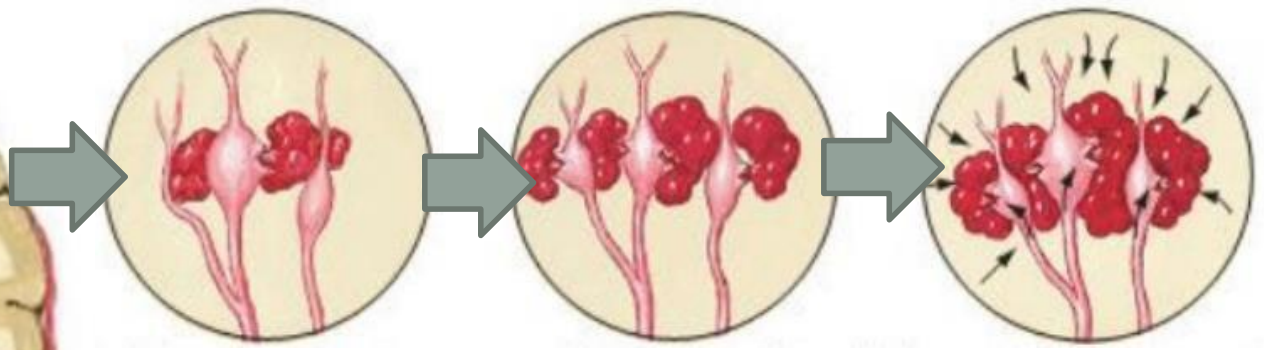
Charcot-Bouchard microaneurysms

Rupture → **INTRAPARENCHYMAL HEMORRHAGE**

- Clinical features
  - Headache, progressive deterioration in level of consciousness, focal deficit
  - In cerebellum, raised intracranial pressure due to hydrocephalus



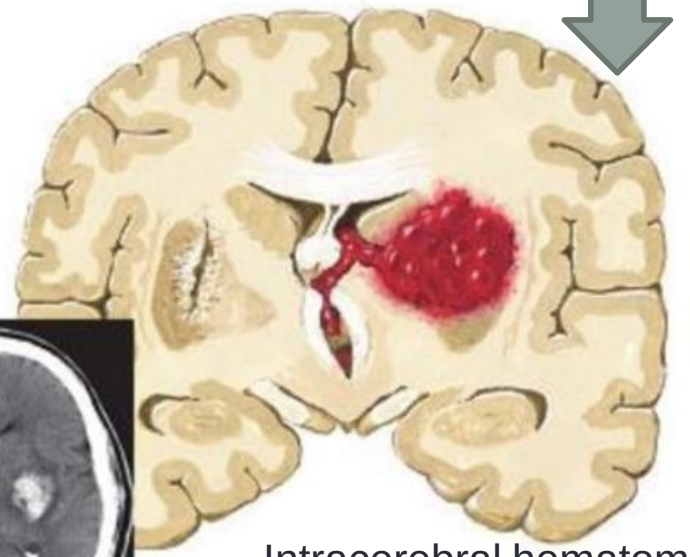
Charcot-Bouchard microaneurysms in lenticlestriated arteries (or other localization)



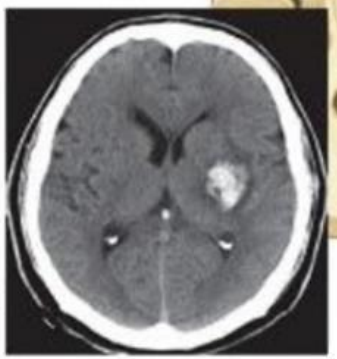
Rupture of microaneurysms pressure on adjacent vessels (satellite)

Satellite vessel rupture

Tissue turgor opposes extravasation



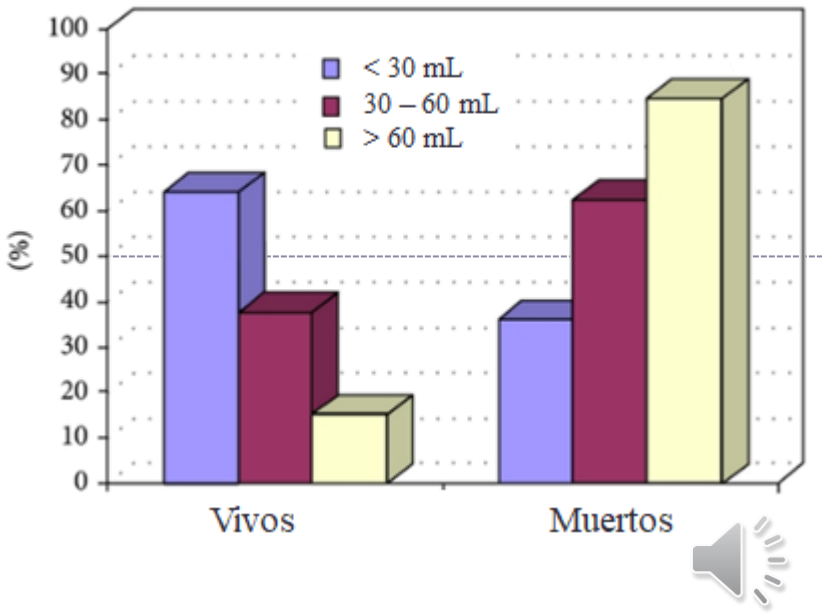
Intracerebral hematoma with rupture to the lateral ventricle and deviation of the midline. Scar from previous bleeding on the right side



Plain head CT-scan: large putaminal hematoma

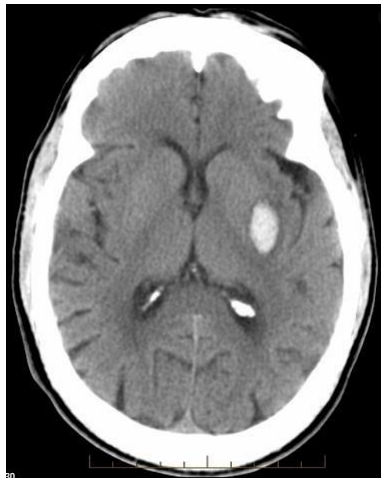
Intracerebral hematoma

- Diagnosis
  - CT-scan detects hematomas ø > 1 cm (blood + edema halo)
    - *MRI only to rule out underlying lesions*
- Prognosis
  - Morbidity
    - *Only 20% have full functional recovery at 6 months*
  - Mortality
    - *Major cause of mortality, which has not changed over time*
    - *Mortality at 30 days 44% (brainstem 75 %)*
    - *It depends on the GCS at admission, the size of the haemorrhage and the presence of intraventricular blood*

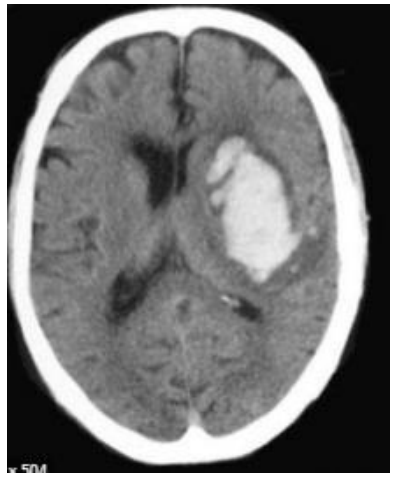


Intracerebral hematoma

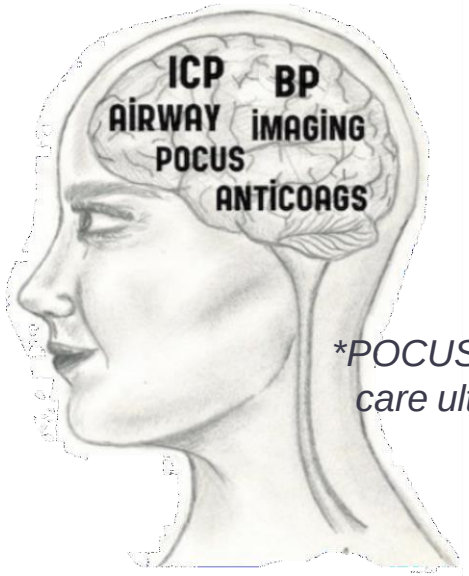
- Therapeutic decision
  - *"Control arterial pressure, glycemia & temperature"*
  - Anti-coagulation / -aggregation control
  - UCI treatment
    - *Airway protection*
    - *Cardio-respiratory support*
    - *Cerebral anti-edema measures (mannitol, hypertonic saline)*
  - Decision: surgical evacuation?
    - *Age*
    - *Previous neurological situation*
    - *Current neurological situation (GCS)*
    - *Hematoma size (30-60 mL)*
    - *Location of hematoma (cerebellum, superficial cortical)*



Basic medical treatment



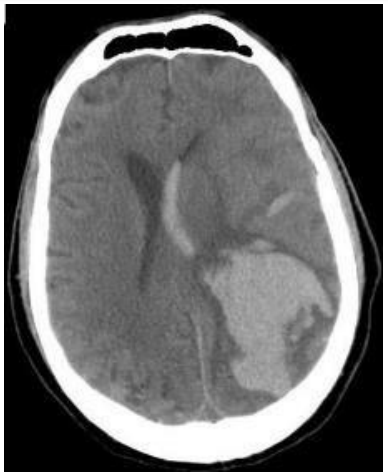
Aggressive ICU treatment



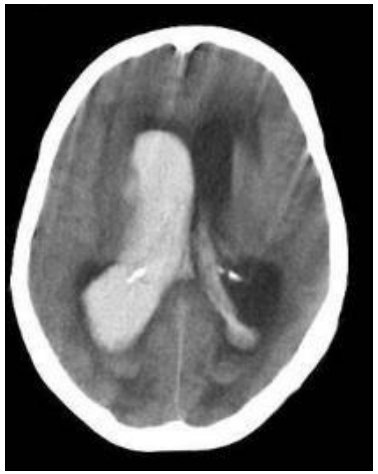
**POCUS = point of care ultrasound*

Intracerebral hematoma

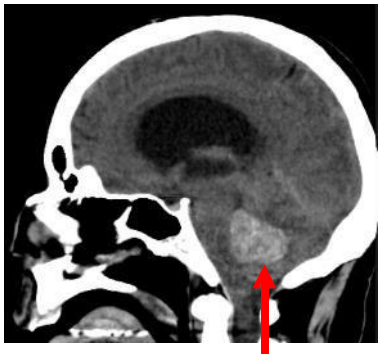
- Surgical treatment
  - Surgical evacuation = craniotomy + fibrinolysis + lavage
    - *Lobar hematoma > 30 mL, superficial (<1 cm depth), with progressive neurological deterioration*
    - *Cerebellar hematoma with brainstem compression or hydrocephalus*
    - ➢ *Patient with good previous condition*
  - "NOT if deep bleeding"
  - Hydrocephalus
    - *External ventricular drain + ICP monitoring*



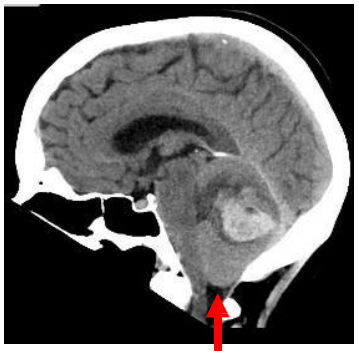
Surgical treatment



Conservative treatment (mortality ⇑)



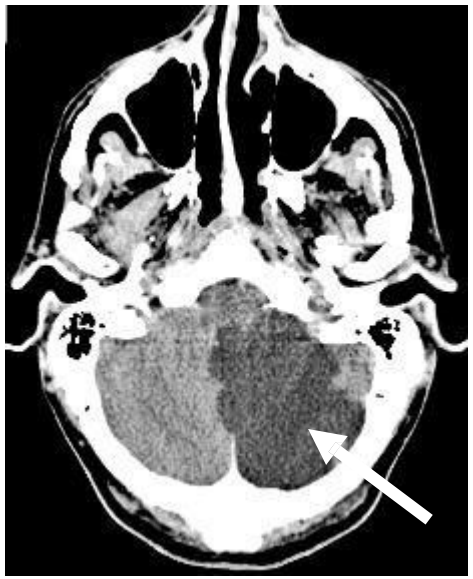
Cerebellar haemorrhage + hydrocephalus and brainstem compression



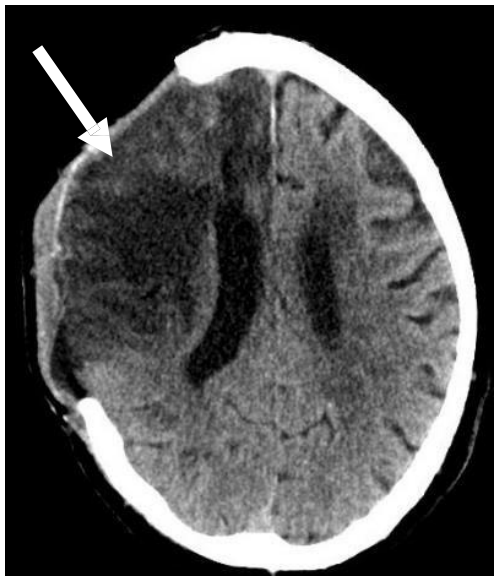
Cerebellar haemorrhage + tonsillar herniation

ISCHEMIC STROKE

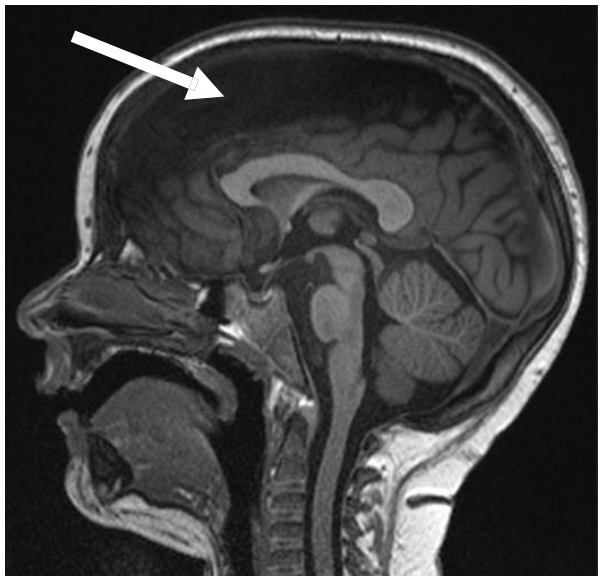
- **Brain ischemia with neurosurgery involvement**
  - *Cerebellar infarction*
  - *Malignant middle cerebral artery infarction*
  - *Venous sinus thrombosis*



Cerebellar infarction

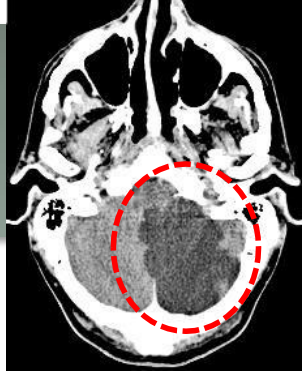


Hemicraniectomy in middle cerebral artery malignant infarction

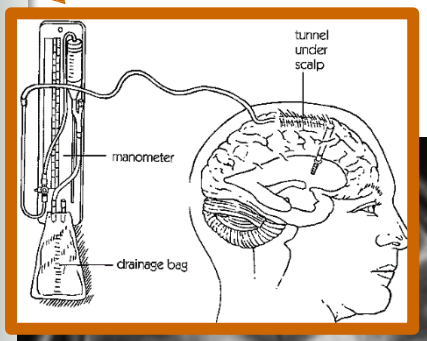


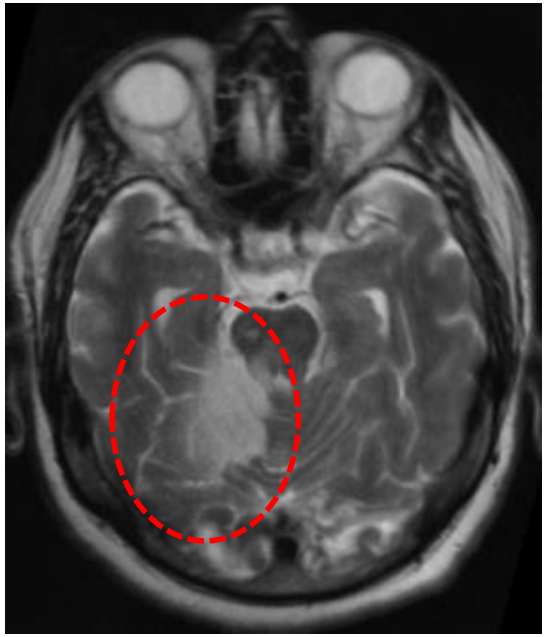
Superior sagittal sinus thrombosis

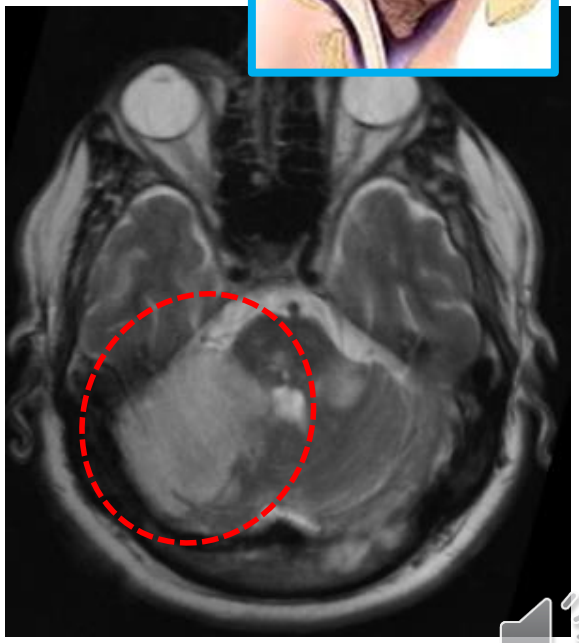
Ischemic stroke



1. Cerebellar infarction
   - If hydrocephalus → external ventricular drainage
   - If mass effect → posterior fossa craniectomy
   - Good survivor recovery

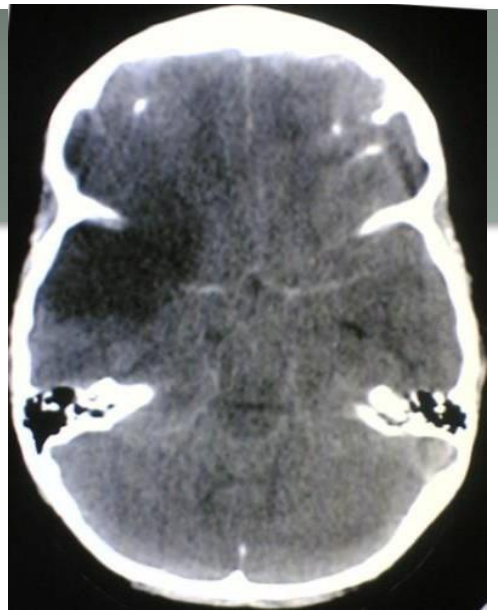




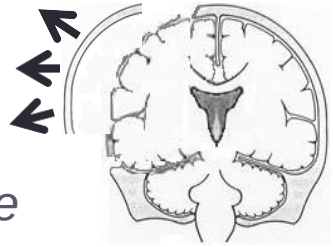


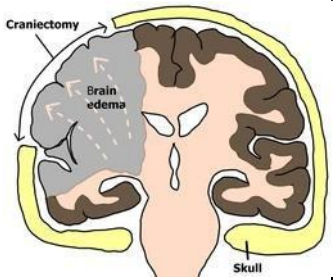
Ischemic stroke

2. *Malignant middle cerebral artery infarction*
   - Total or subtotal infarct (> 50%) of the middle cerebral artery territory
   - < 10 % supratentorial ischemic strokes
   - Clinic
     - *Complete hemiplegia*
     - *Hemineglect*
     - *Mixed aphasia if dominant hemisphere*
     - *↓level of consciousness*
   - Prognosis: death > 60 %
   - In selected cases, decompressive craniectomy (hemicraniectomy)
     - *Controversial*
     - *Survivors with major sequelae*

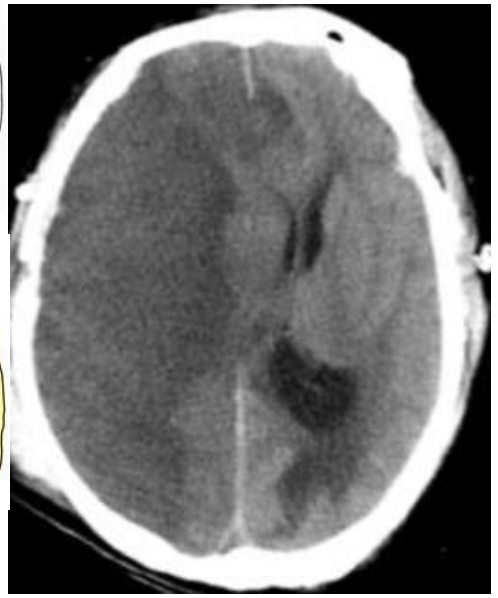


Middle cerebral artery infarction



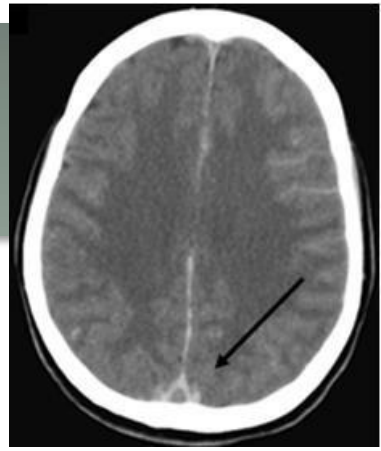


Decompressive craniectomy



Massive middle cerebral artery infarction

Ischemic stroke

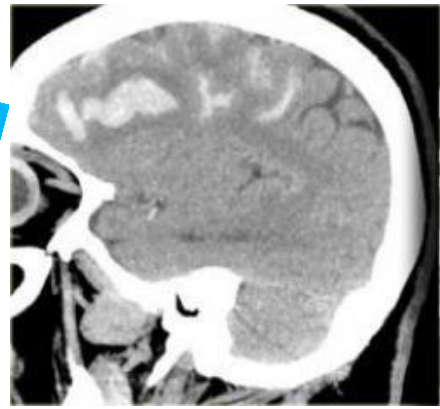


Empty delta sign

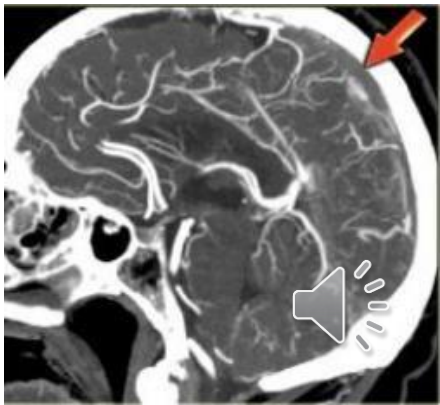
3. Venous sinus thrombosis

- Rare, ♀/♂ (3:1)
- Patients with prothrombotic risk
  - *Hereditary thrombophilia, neoplasms*
  - *Anti-coagulation, pregnancy, puerperium*
- Clinical features
  - ➢ *↓Venous return→↑ICP → Cerebral ischemia or secondary intraparenchymal haemorrhage*
  - *Raised intracranial pressure > focal symptoms of ischemia or bleeding*
- Diagnosis
  - *CT-scan C+ → Empty delta sign in superior longitudinal sinus*
  - *Angio-CT-scan C+ o angio-MRI in venous phase*
  - *Neonates: trans-fontanelle ultrasound → Angio-MRI*
- Treatment low molecular weight heparin (even if there is secondary bleeding). May require ICP monitoring

No surgical treatment



Superior sagittal sinus filling defect



SUMMARY KEY CONCEPTS TOPIC 7

- SAH (subarachnoid haemorrhage)
  - Sudden presentation - aneurysm
  - Thundering headache - nausea and vomiting - neck stiffness
  - High blood pressure, smoking, alcohol
- Cerebral aneurysms
  - ↓ Incidence but ↑mortality
  - SAH → head CT-scan, angiography
  - Rebleeding and vasospasm
- Cerebral AVMs
  - Young people, seizures, bleeding
  - All treated = surgery > surgery + embolization + radiosurgery
- Cavernomas
  - Epilepsy
  - Diagnosis = MRI
  - Treatment = surgical

SUMMARY KEY CONCEPTS TOPIC 7

- Spontaneous intracranial hematoma
  - Arterial hypertension
  - Conservative treatment – UCI
  - Surgical evacuation with specific indications
    - *Accessible lobar hematoma in a patient in good condition*
    - *Cerebellar hematoma compressing the brain stem or causing hydrocephalus*
- Cerebral infarct
  - Hydrocephalus treatment ± posterior fossa decompression
- Spinal cord AVMs
  - Mainly endovascular treatment
  - Poor results

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