Department of Surgery
Neurosurgery Teaching Unit

SEMINAR: CEREBRO-VASCULAR PATHOLOGY

34484 Pathology of the nervous system

Neurosurgery

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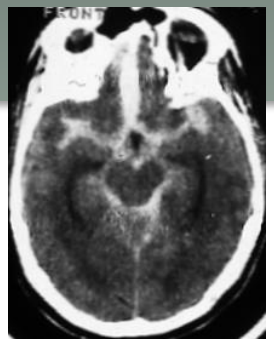
Key concepts

- **Subarachnoid hemorrhage**
  - Brian aneurysm
  - Cerebral arteriovenous malformation (cerebral AVM)
- **Spontaneous brain hemorrhage**
  - Brain hemispheres
  - Posterior fossa
- **Cerebral ischemia**
  - Stroke
  - Malignant middle cerebral artery infarction
- **Spinal cord arteriovenous malformation**



Subarachnoid hemorrhage

Aneurysm

Brain AVM

Cerebral haematoma

Cerebellar haematoma

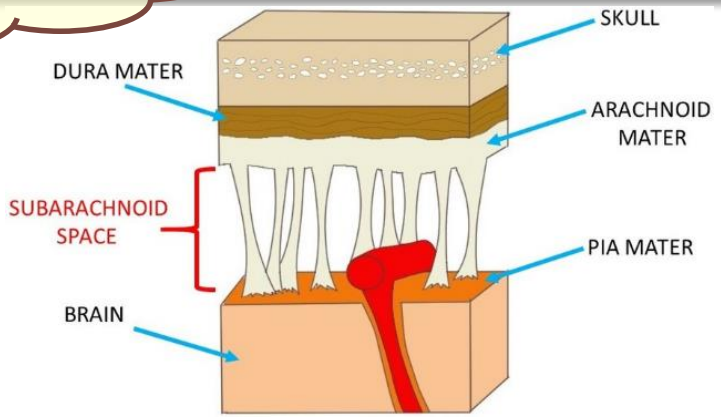
Middle cerebral artery infarct

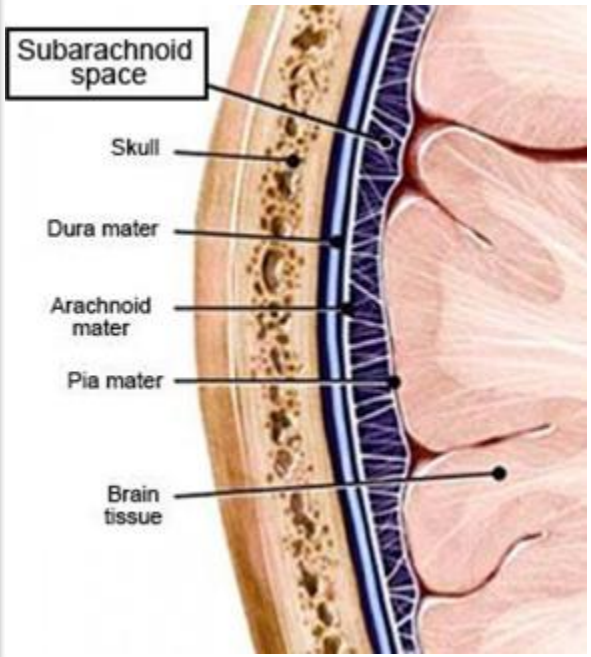
Spinal cord AVM

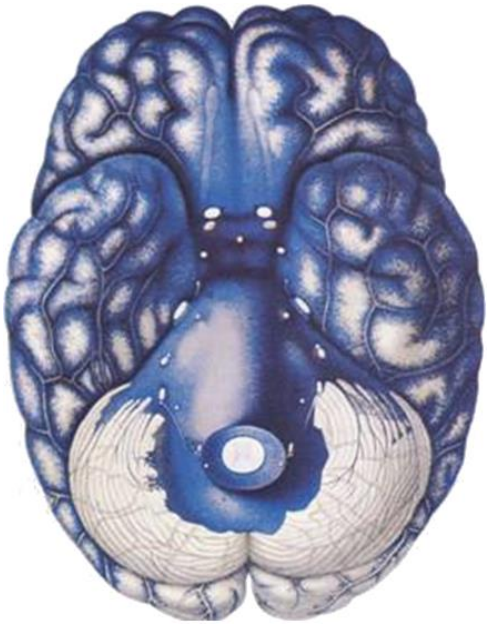
What is the subarachnoid space and where is it?

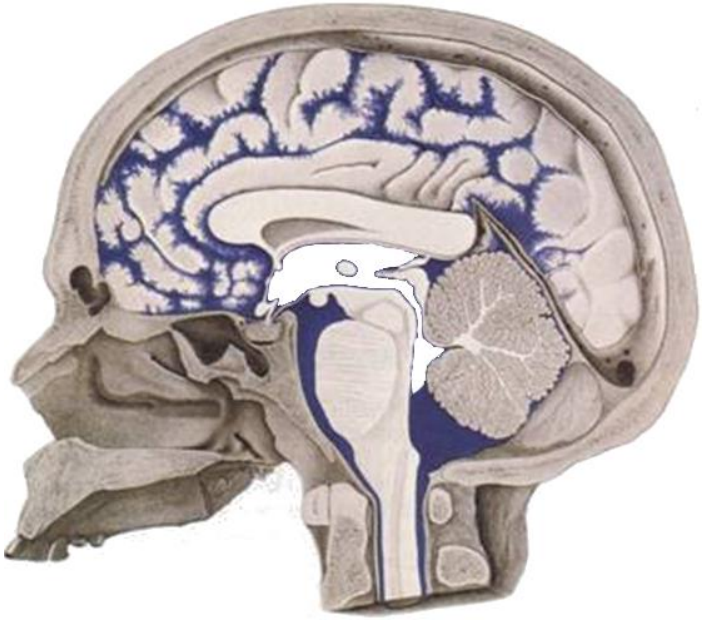
REMEMBER?

- It is the space between the visceral arachnoid and the pia mater





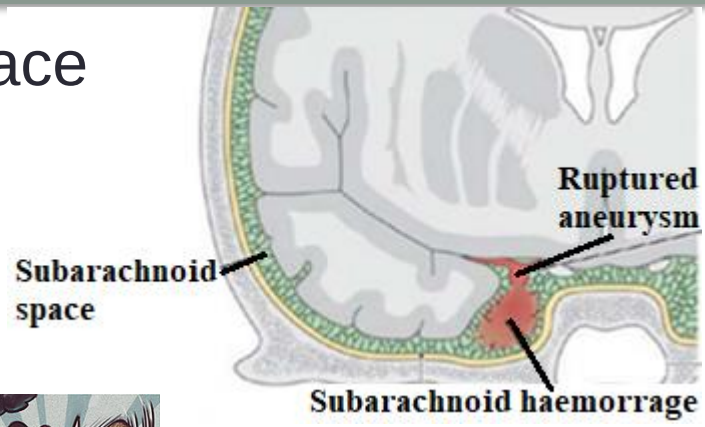




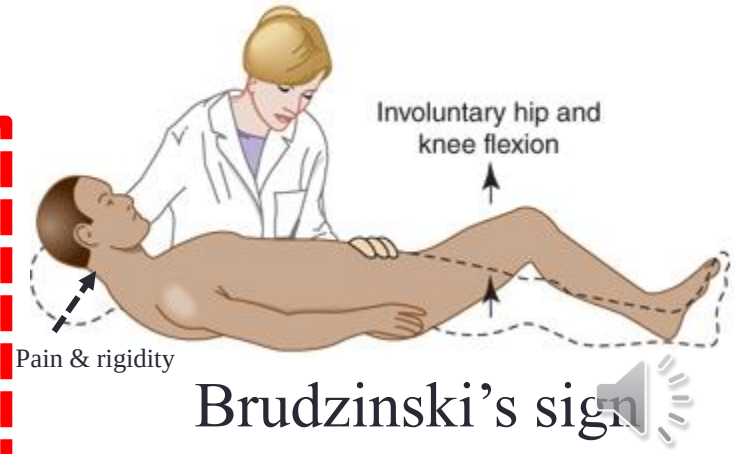
Blue shows distribution of the subarachnoid space

SUBARACHNOID HEMORRHAGE

- Arterial blood spill to subarachnoid space
- Sudden ⇑ intracranial pressure (ICP)
- Sudden ⇑ intense ⇑ severe headache
- Altered level of consciousness
- Neck stiffness: Brudzinski's sign
- Possible hydrocephalus
- Possible neurological deficit
  - Intracerebral haematoma

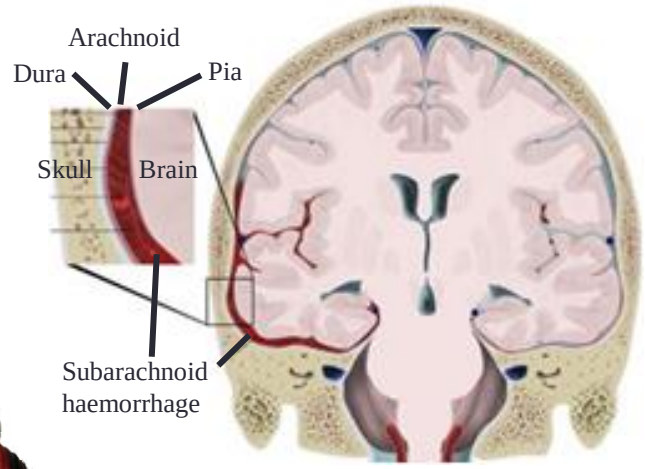


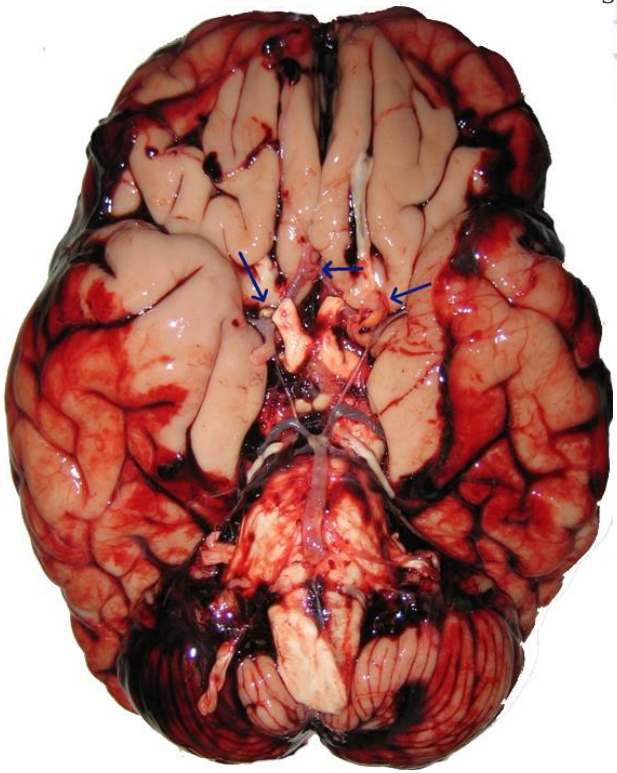
- 15% die on the spot, 46% in hospital
- 66% survivors with sequelae
  - 30% severe

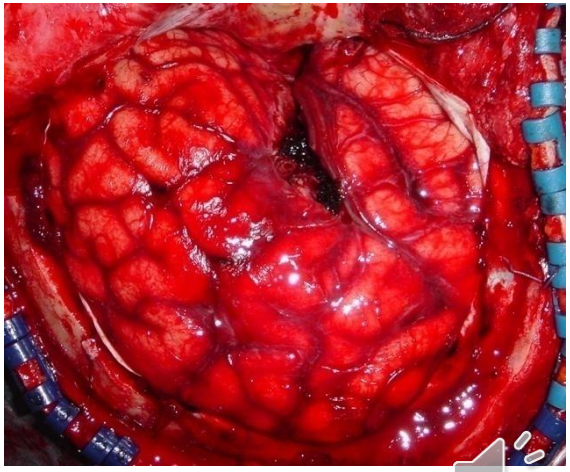


Subarachnoid haemorrhage

- Cause of death & severe disability in middle-aged people





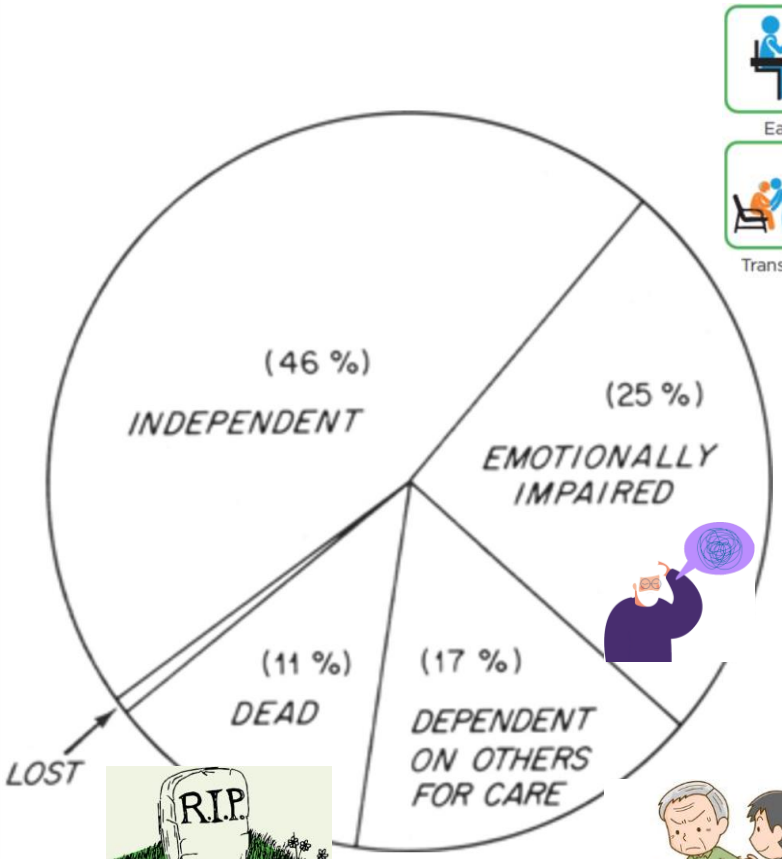


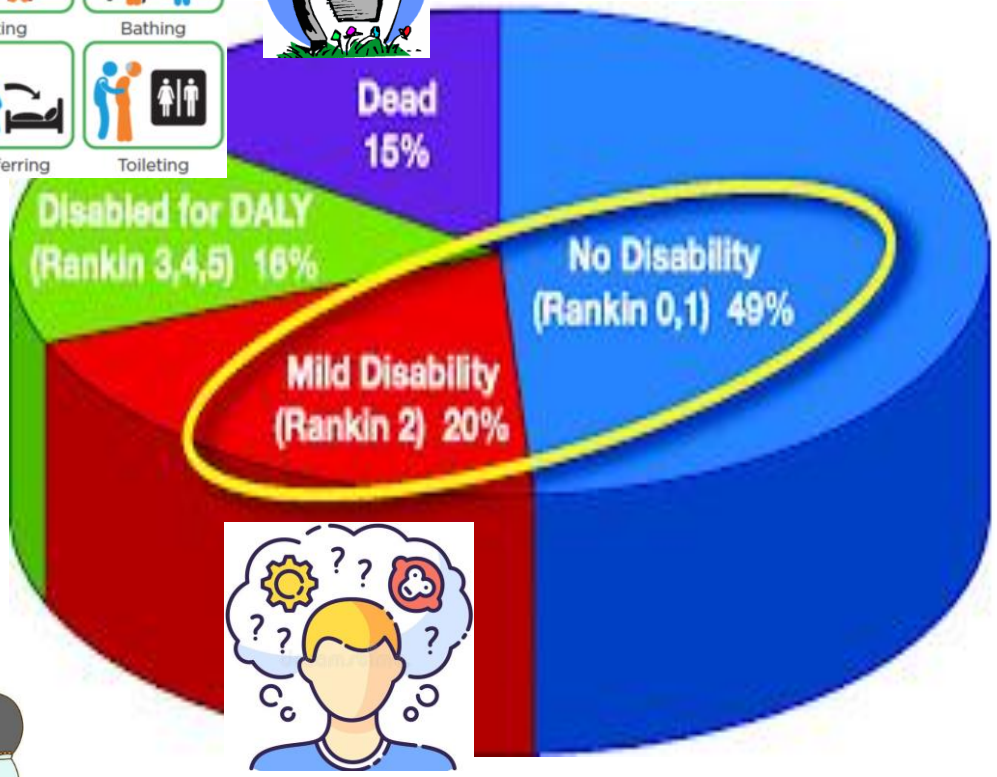
Necropsy

Intraoperative

Outcome at one year follow-up

- Many survivors have significant disabilities and sequelae





Clinical presentation: warning signs

- Sentinel headache
- Minor heart attacks or TIA
- Visual acuity loss
- Third nerve palsy
- Hemiparesis
- Craniofacial pain
- Epileptic seizures
- Incidental finding

Sentinel headache

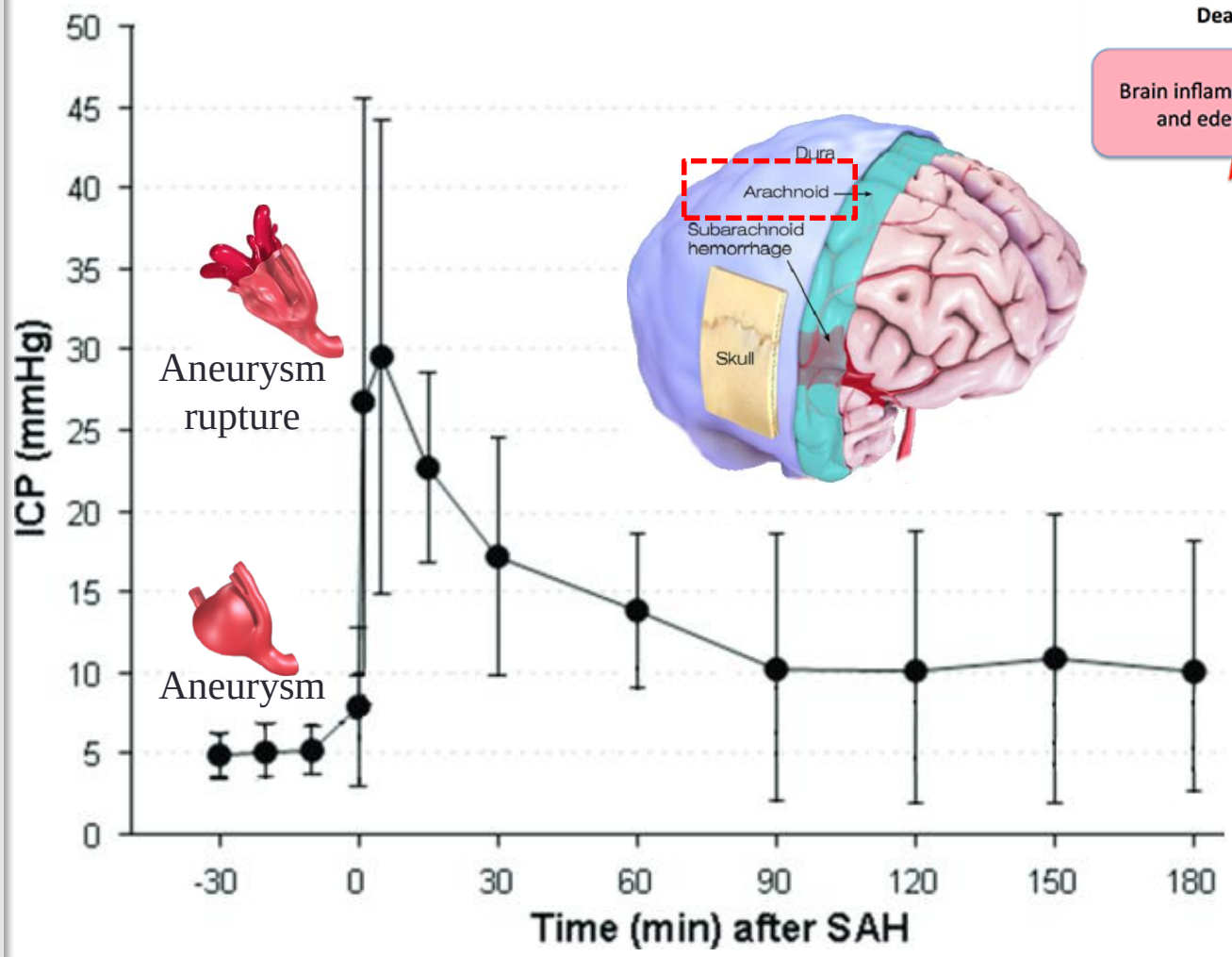
3rd nerve palsy

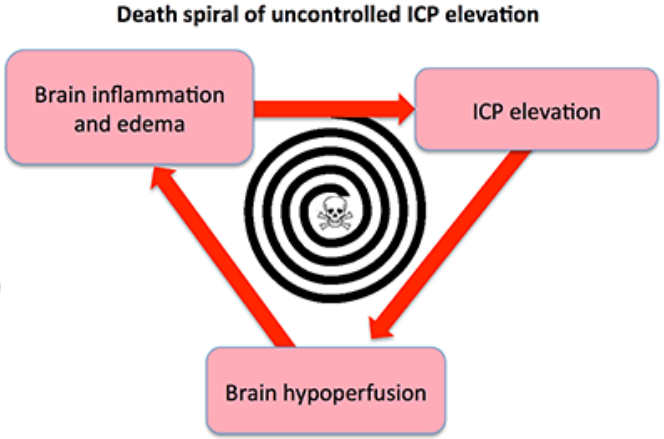
Peri-orbital pain

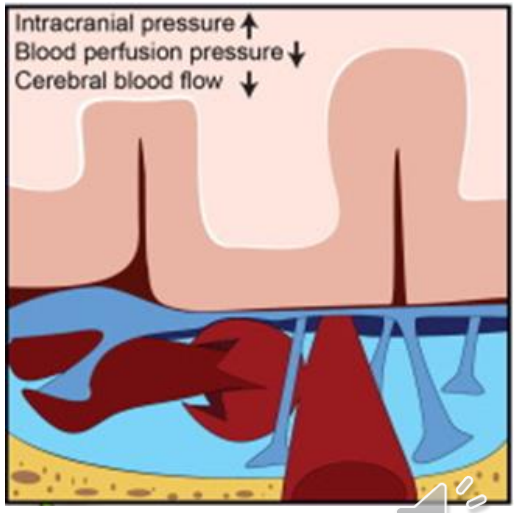
MRI incidental finding

Subarachnoid hemorrhage & ICP

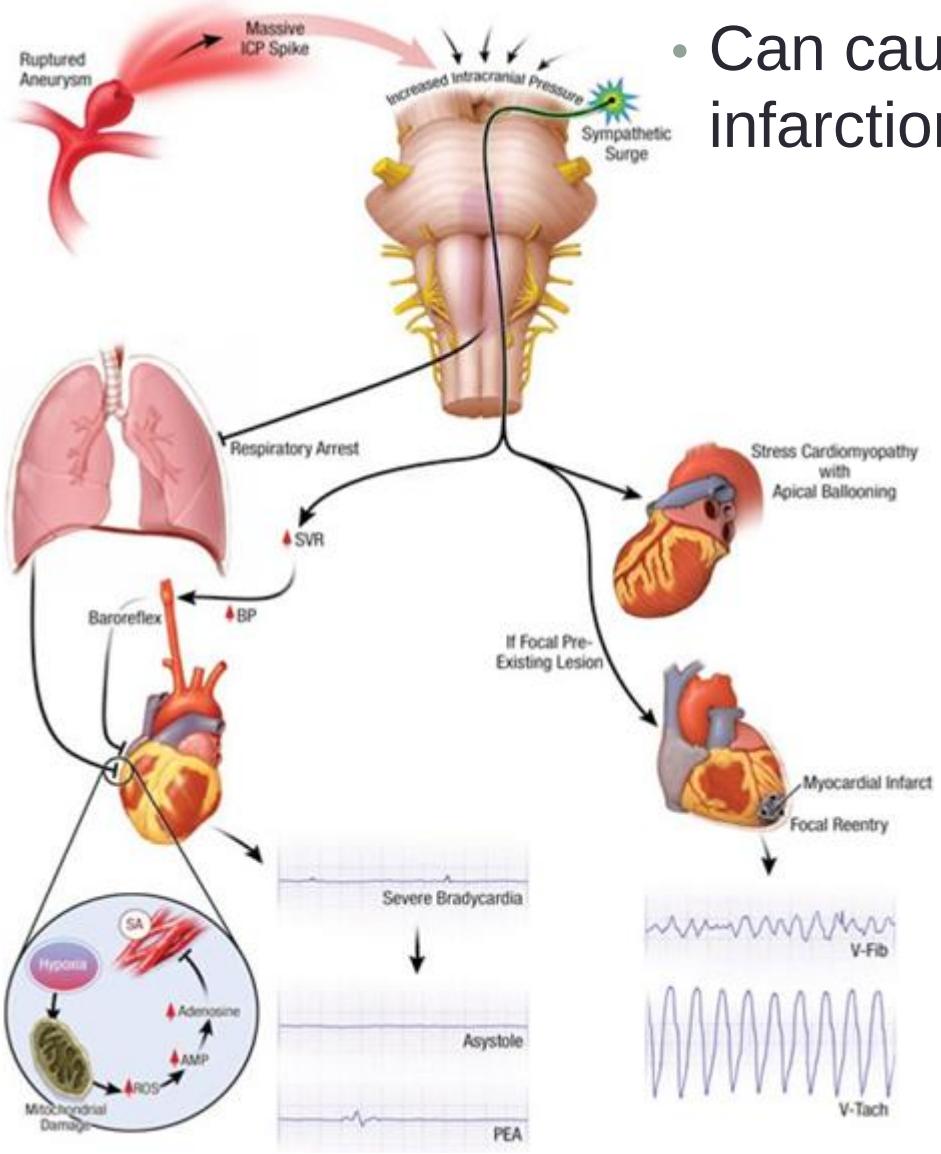
- Aneurysm rupture = sudden & ↑↑↑ ICP = possible death





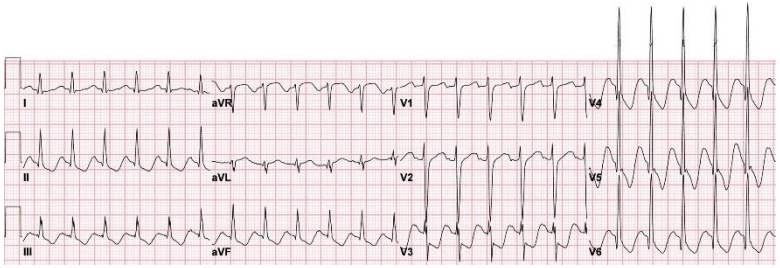


Subarachnoid hemorrhage & heart



- Can cause cardiac arrest / myocardial infarction / stunt myocardium

Stunt myocardium in subarachnoid haemorrhage



EKG with cerebral T waves in subarachnoid hemorrhage

Subarachnoid haemorrhage: clinical features

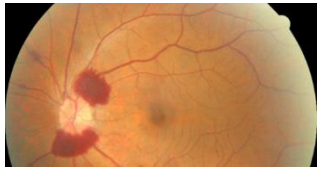
Headache: the ‘worst of my life’

Loss of consciousness

Photophobia

Neck stiffness

Vomiting

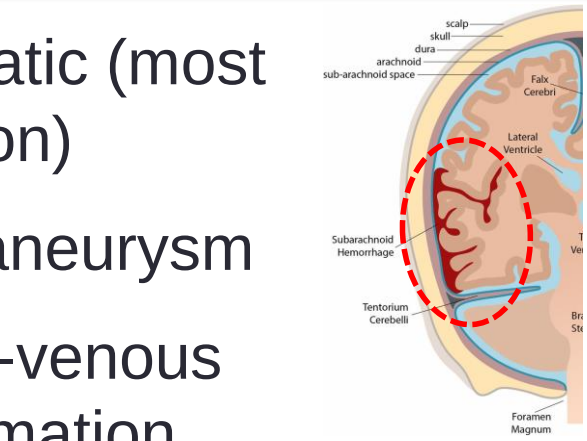


Vitreous haemorrhages

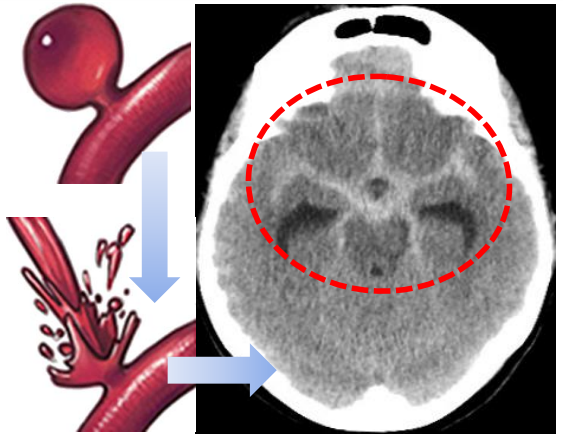
Arterial hypertension

Subarachnoid hemorrhage: causes

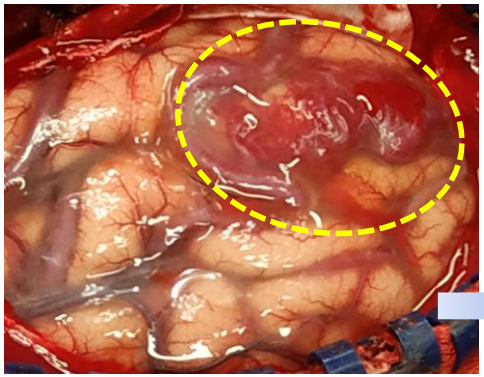
- Traumatic (most common)
- Brain aneurysm
- Arterio-venous malformation
  - Cerebral, rarely spinal cord
- Anti-coagulation
- Anti-platelet medication
- No known cause (10%)

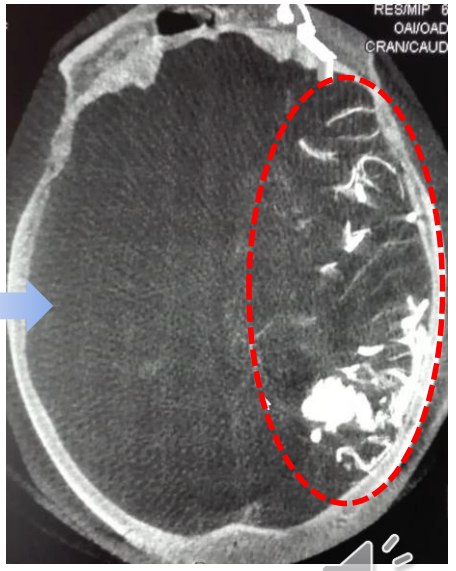


Traumatic



Aneurysm rupture

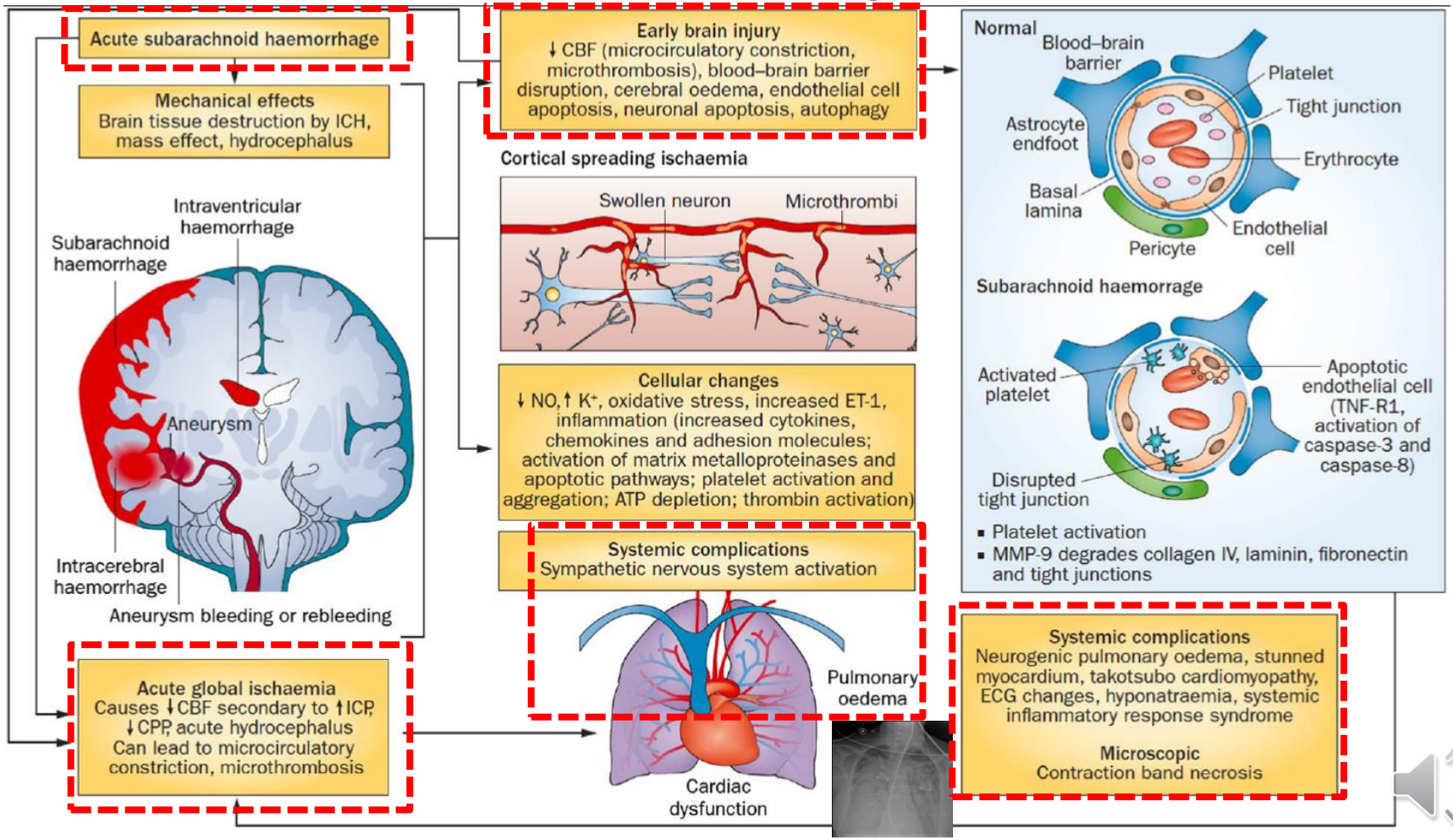




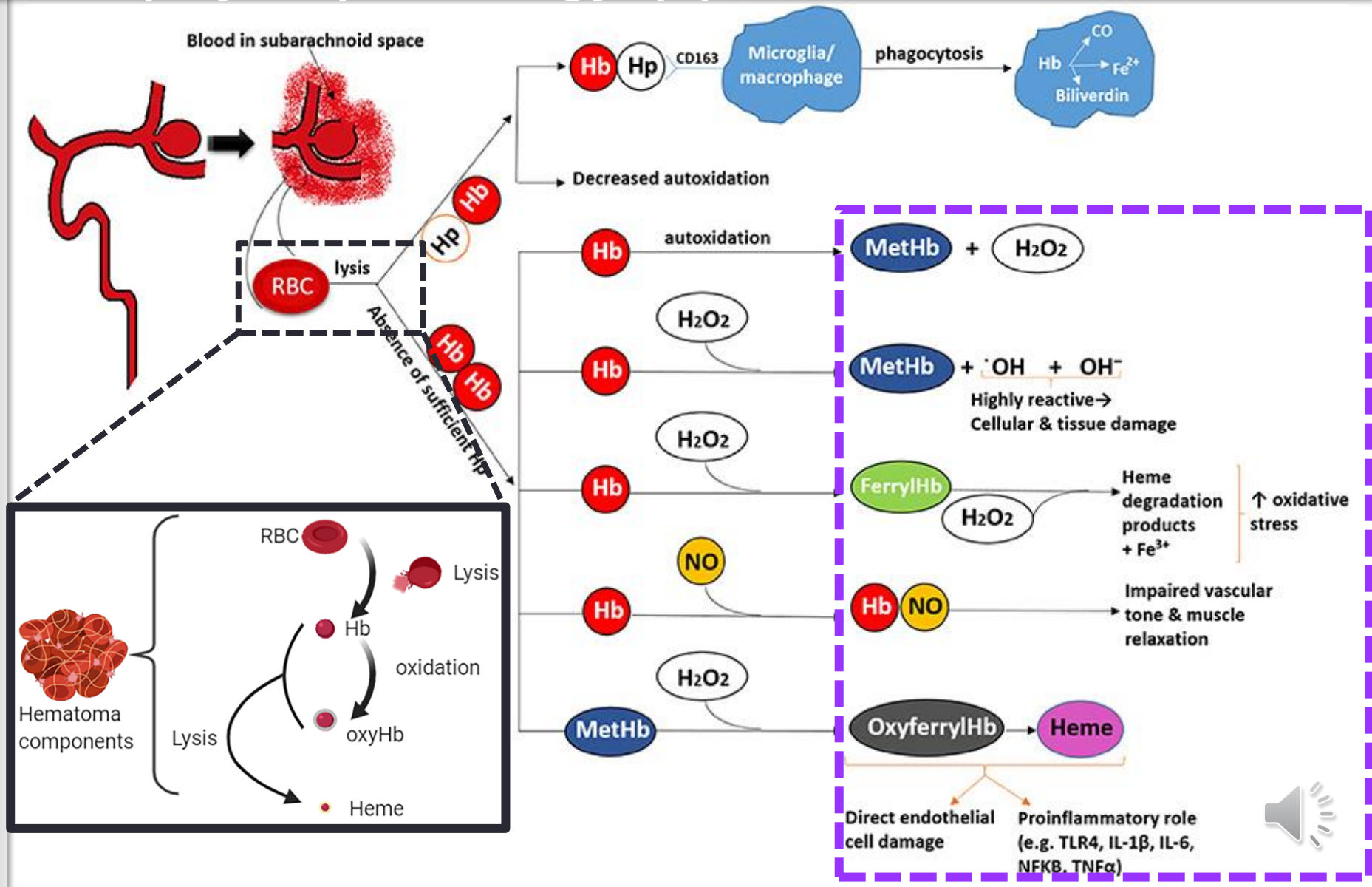
Arterio-venous malformation

Subarachnoid haemorrhage: physiopathology (1)

- Mechanical effect + complex biochemical interactions = free radical formation = cell damage → cell death

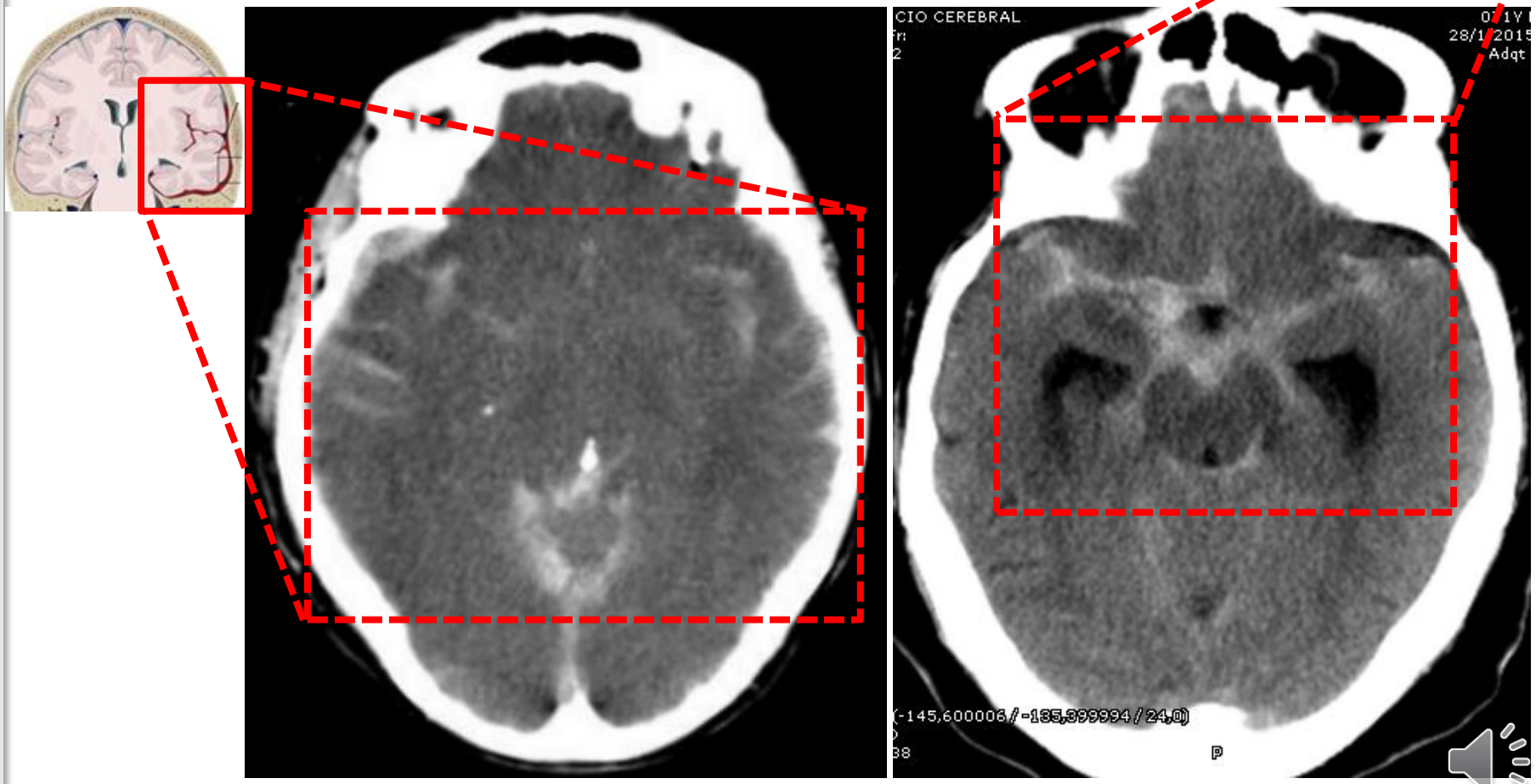


Subarachnoid haemorrhage: physiopathology (2)



Subarachnoid haemorrhage (SAH) diagnosis: CT scan

- CT scan FIRST diagnostic procedure
  - Lumbar puncture ONLY after negative CT scan

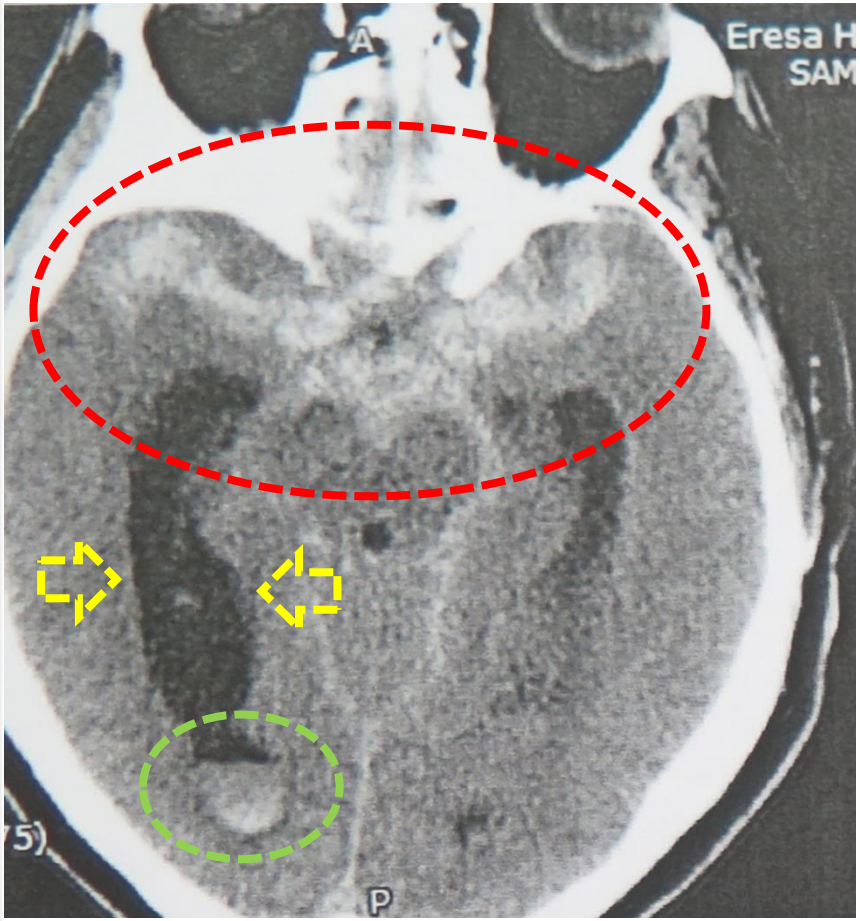


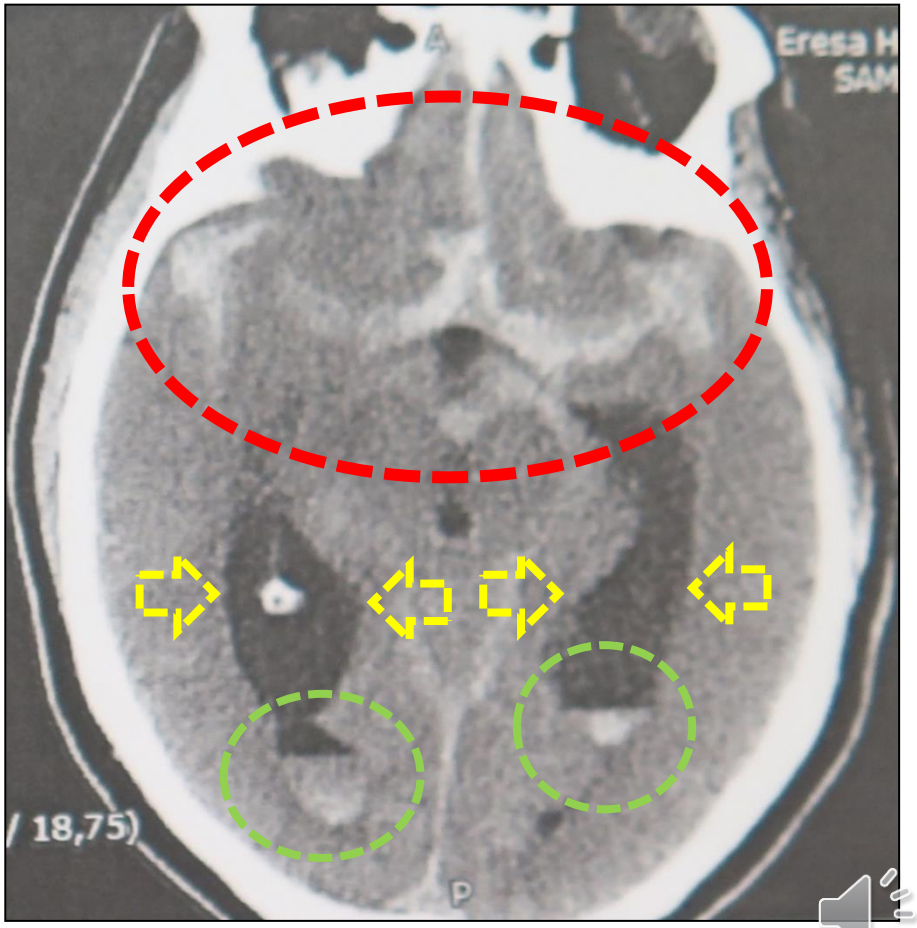
SAH at sylvian fissures

SAH at skull base CSF cisterns

CT scan: subarachnoid haemorrhage + intraventricular blood + hydrocephalus

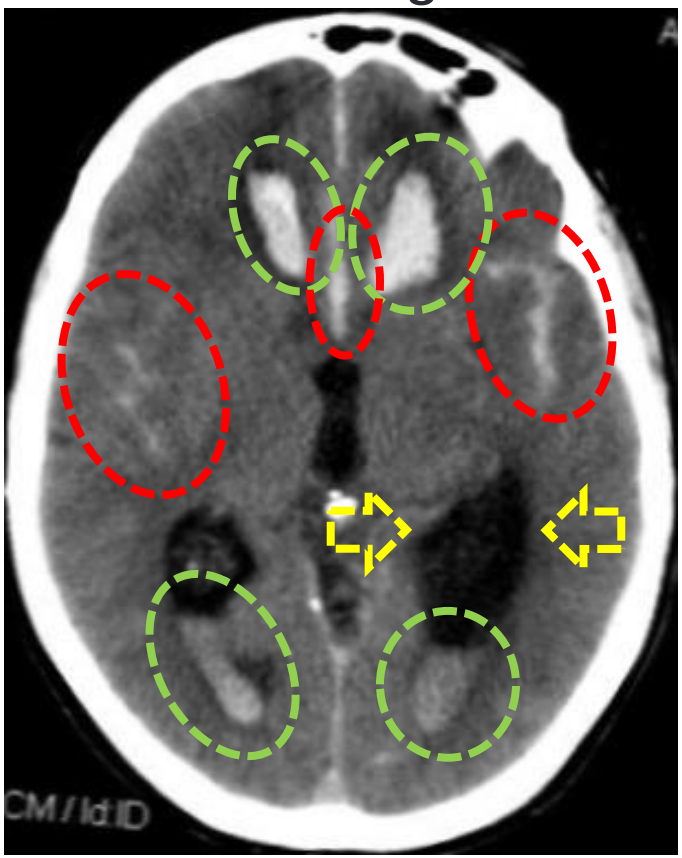
- Subarachnoid haemorrhage → intraventricular blood → hydrocephalus

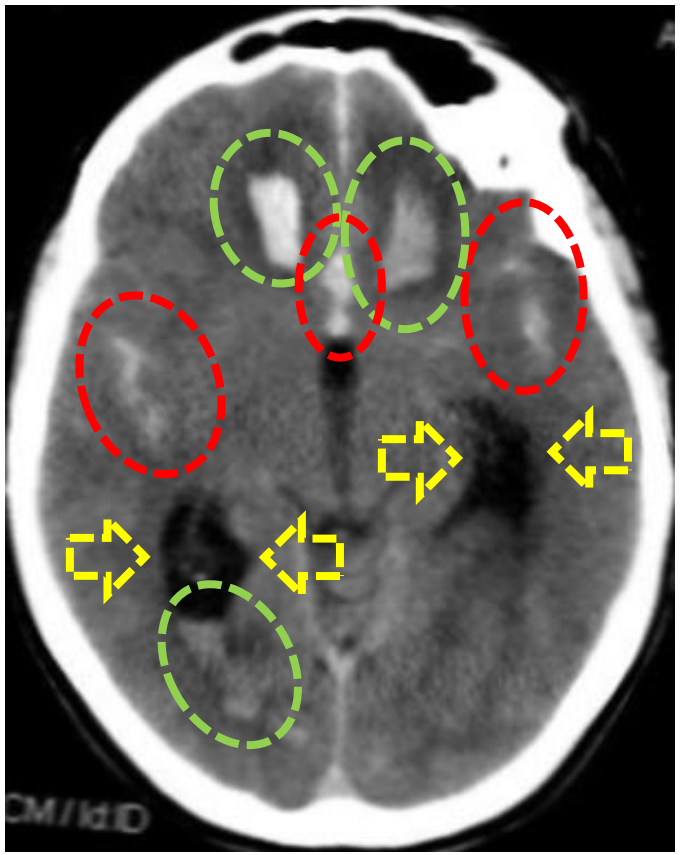




CT scan: subarachnoid haemorrhage + intraventricular blood + hydrocephalus

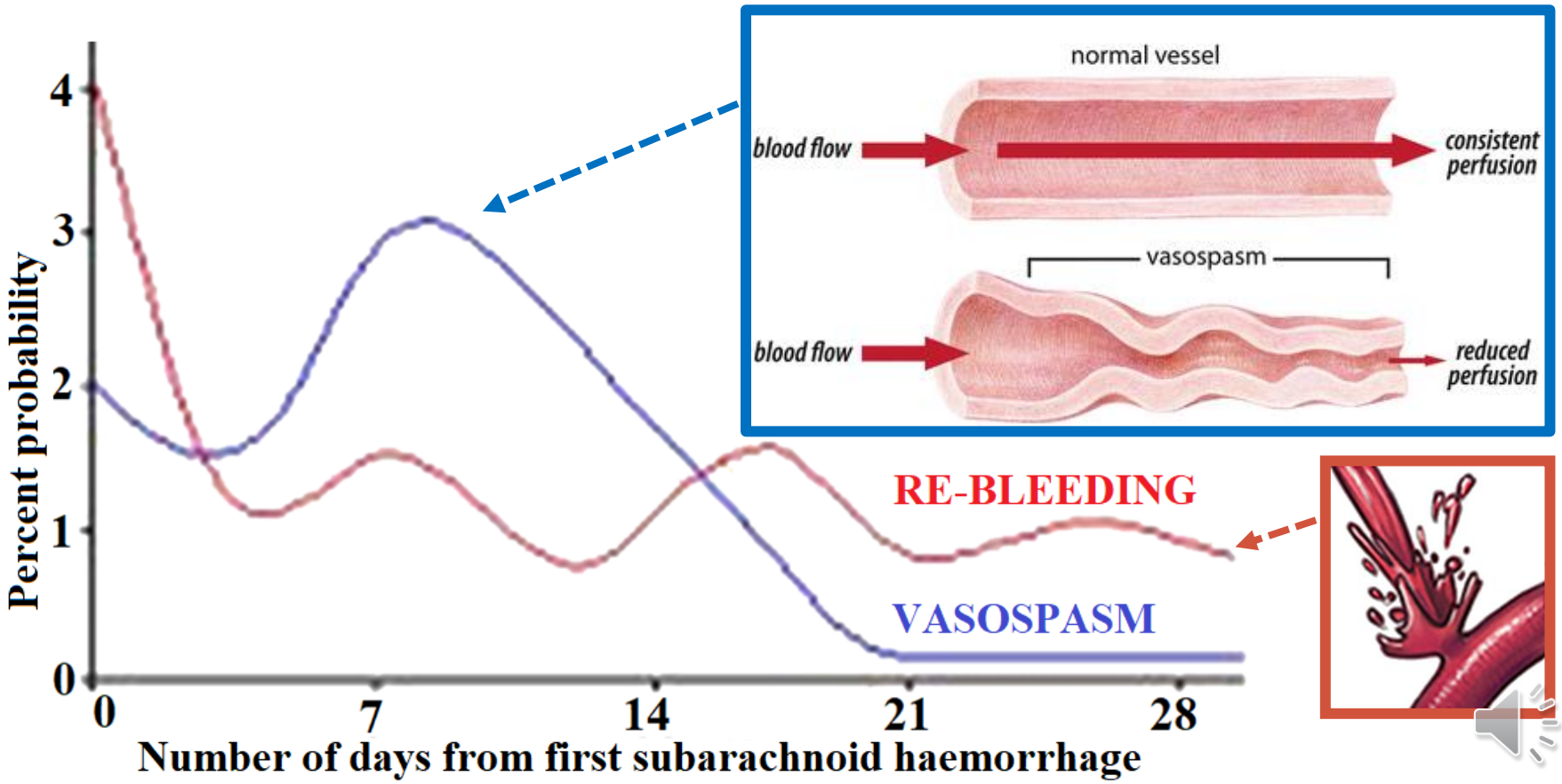
- Subarachnoid haemorrhage and intraventricular haematoma distribution can help identify the probable cause of bleeding





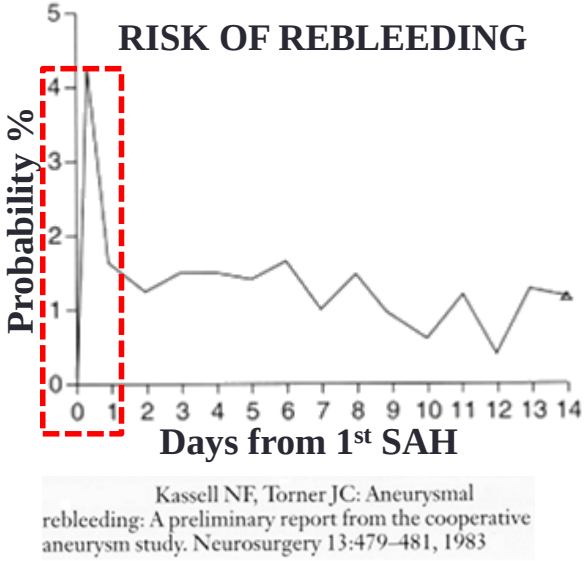
Subarachnoid haemorrhage risks: first-bleeding, second vasospasm

- Rebleed = 20% death
- Vasospasm = 7% death + 7% neurological deficits

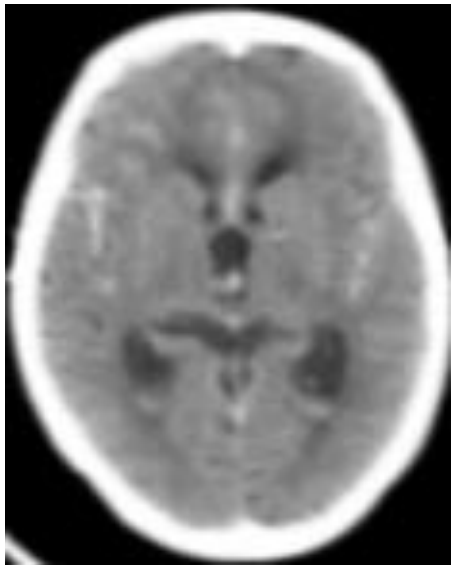


Subarachnoid hemorrhage: rebleeding

- Most frequent first 24h
- If aneurysm not excluded
  - 15% rebleed < 15 days
  - 50% < 6 months
- ↑↑ mortality (50% in six months)
- Prevention: early treatment (embolisation / surgery)

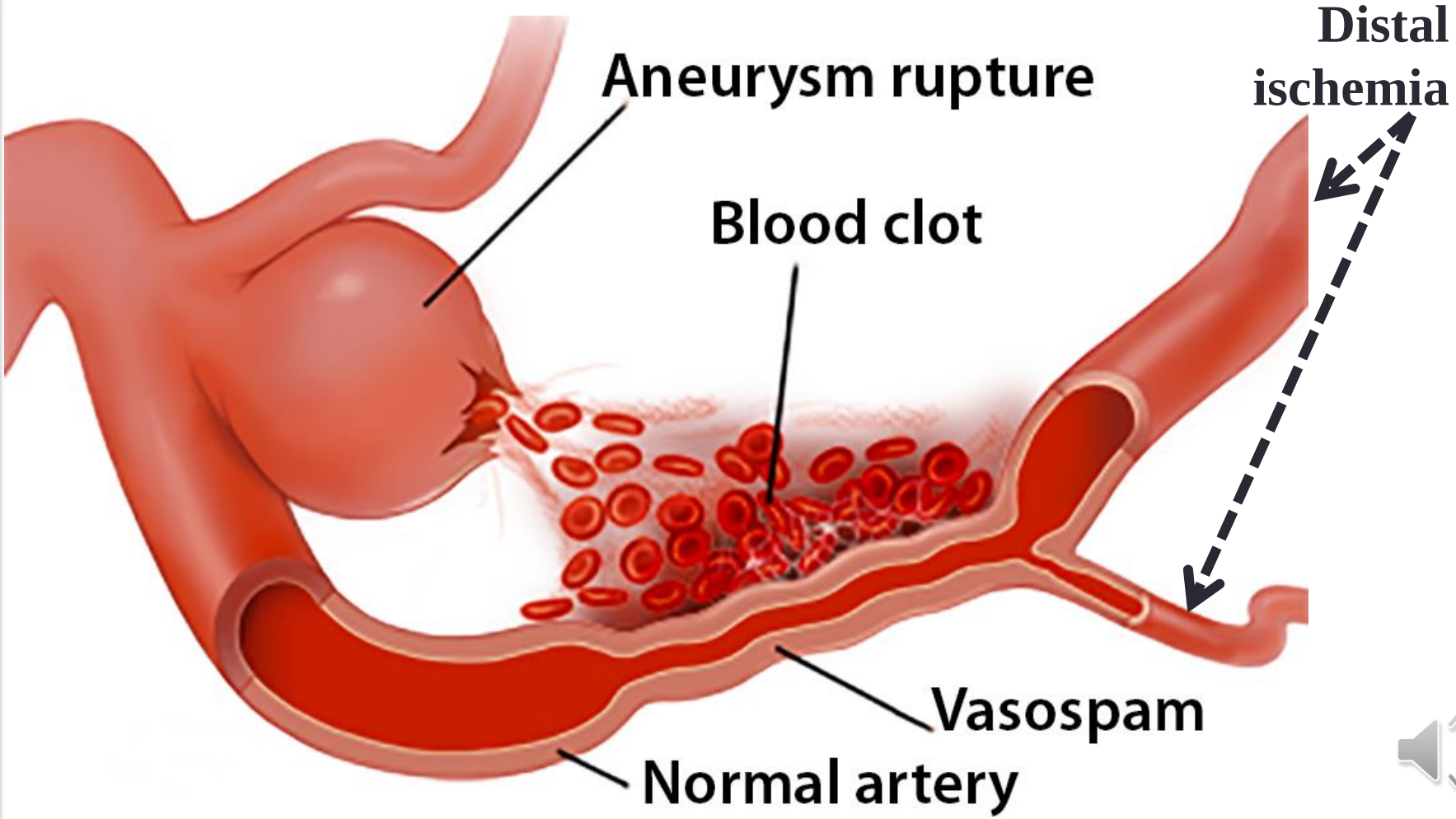


Kassell NF, Torner JC: Aneurysmal rebleeding: A preliminary report from the cooperative aneurysm study. Neurosurgery 13:479–481, 1983

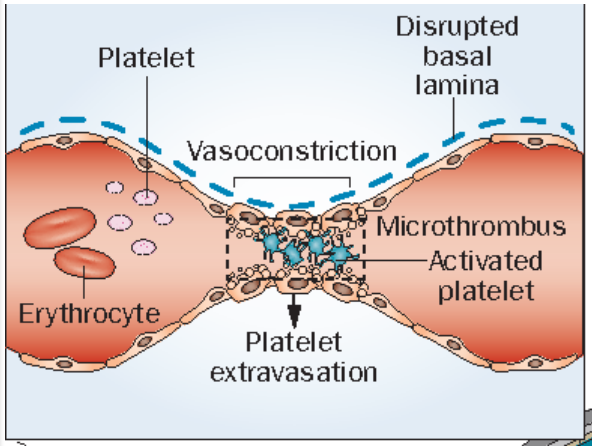


Subarachnoid hemorrhage = extravasated blood = vasospasm = ischemia

- Cause vasospasm = oxyhaemoglobin released from extravasated red blood cells



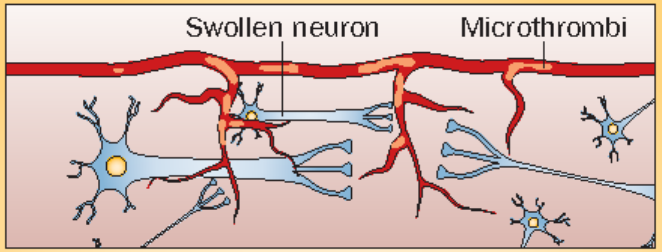
SAH vasospasm consequences



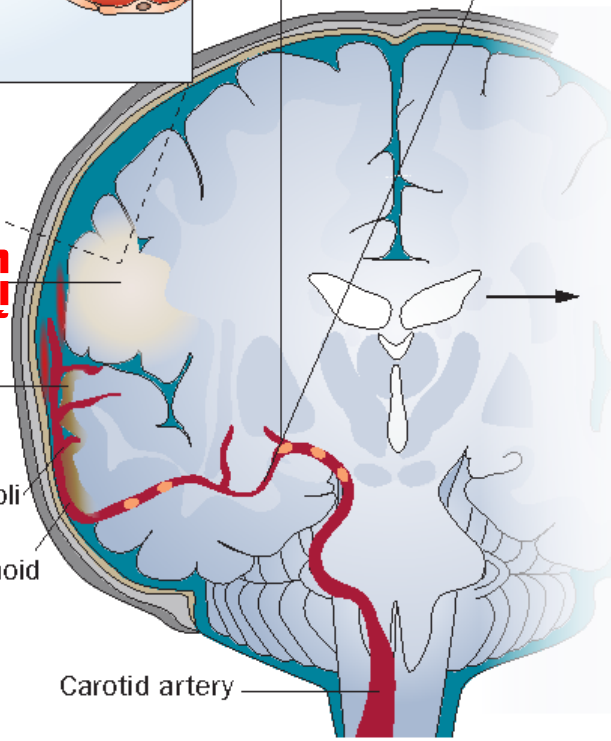
Microcirculatory constriction
Microthrombosis

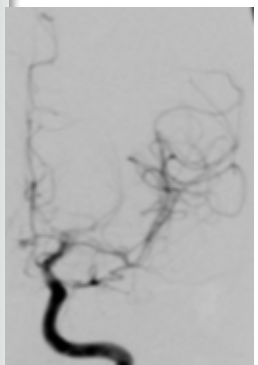
Angiographic vasospasm
↑Haemoglobin, oxidative stress and inflammation lead to endothelial cell and perivascular nerve injury, leading to ↑NO, endothelin, leading to membrane depolarization, open voltage-gated calcium channels and activation of TRP channels

Cortical spreading ischaemia



Spreading depressions, neuronal swelling, distorted dentritic spines, neuronal depolarization, glutamate release and depression, associated with pathological spreading wave of vasoconstriction, caused by ↑K, ↑haemoglobin and inhibition of NO synthase that are present after SAH and possibly mediated by increased astrocyte endfeet calcium that activates large-conductance calcium-activated potassium channels



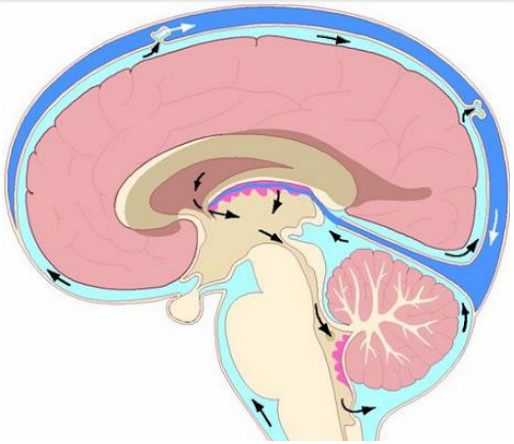


Delayed neurological complications
Seizures, infection, complications of aneurysm repair, aneurysm rebleeding, hydrocephalus, brain swelling, intracranial haematoma, increased ICP, reduced CPP

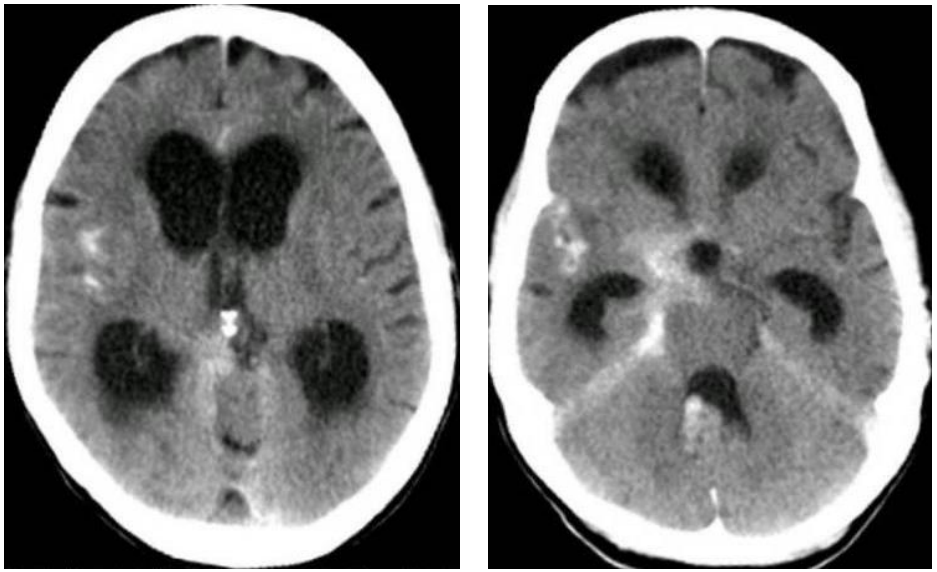
Delayed systemic complications
Infection, fever, pulmonary oedema, cardiac failure, organ failure, drug adverse effects, hyponatraemia, hypercarbia, hypoglycaemia, low haemoglobin, systemic inflammatory response syndrome

SAH consequences: hydrocephalus

- Cause: subarachnoid space blood tamponade
- Consequences: increased ICP, decreased cerebral perfusion pressure
- Types
  - Acute
    - Present on admission
  - Chronic
    - After the subarachnoid clot is completely gone



Normal CSF circulation through the subarachnoid space

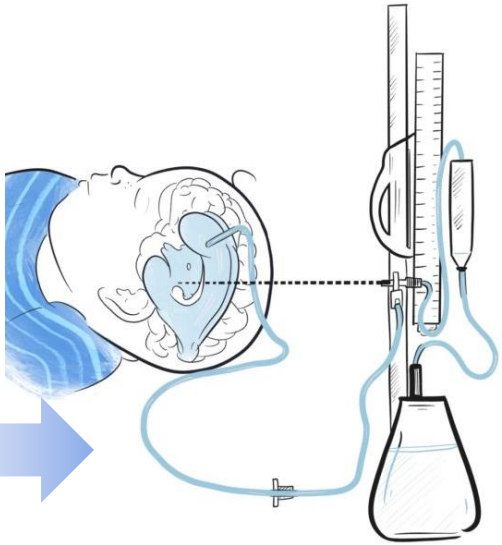


Acute SAH hydrocephalus

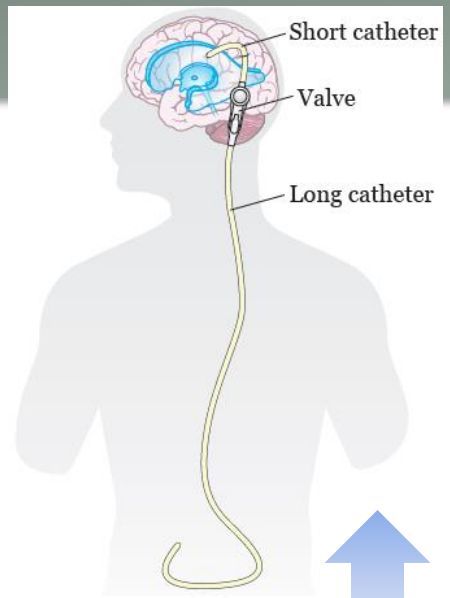
Chronic post SAH hydrocephalus

SAH consequences: treatment hydrocephalus

- **Acute**
  - In 15% admission CT scan
    - Symptomatic in 50%
  - ↑ frequent in poor clinical grade subarachnoid haemorrhages
  - Treatment: **external ventricular drainage**
    - To control hydrocephalus
    - To remove as much extravasated blood as possible
- **Chronic**
  - Related factors: age, amount of ventricular / subarachnoid bleeding, type of aneurysm treatment? Infection?
  - Treatment: **ventricleperitoneal shunt**



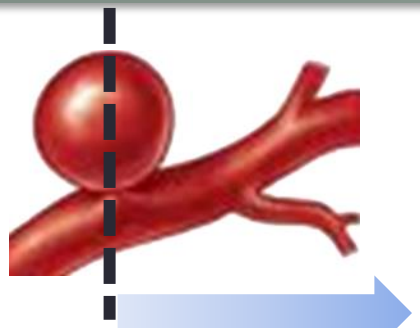
Acute hydrocephalus

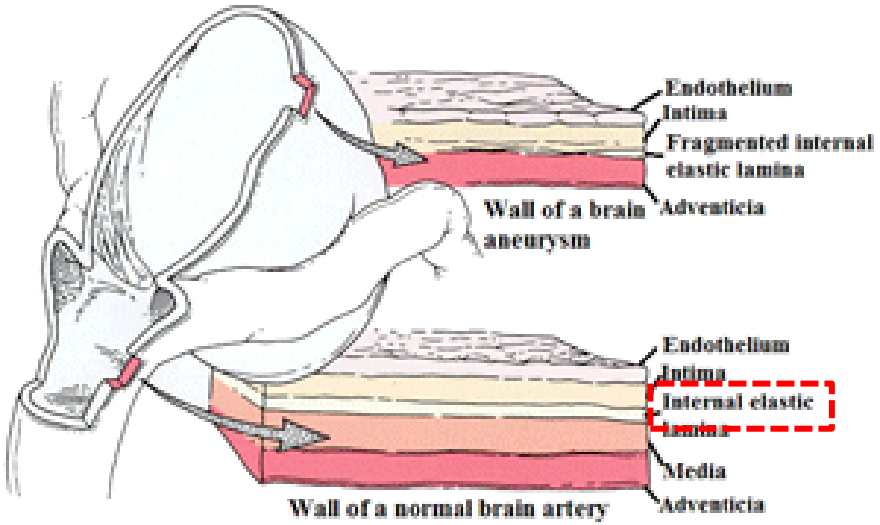


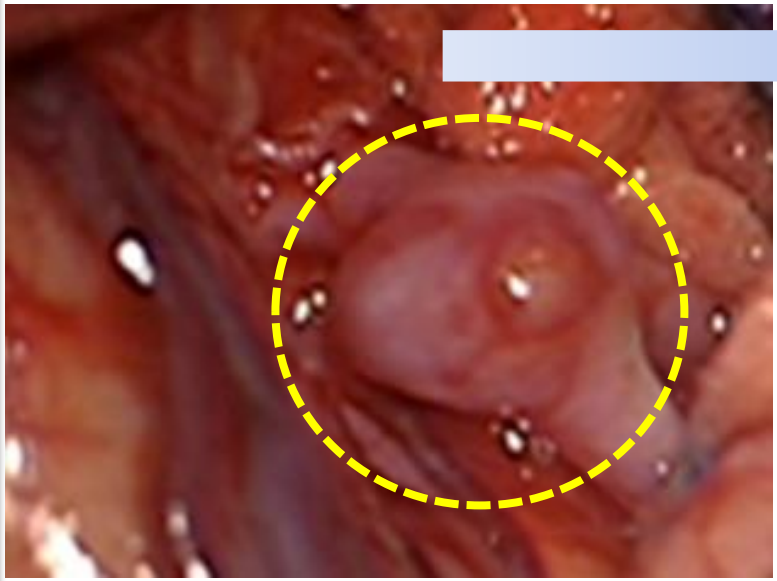
Post-subarachnoid hemorrhage hydrocephalus

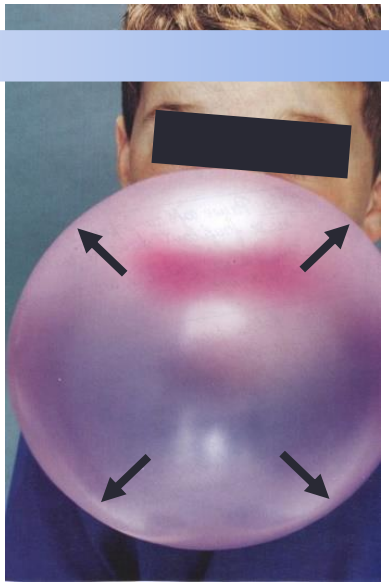
BRAIN ANEURYSM

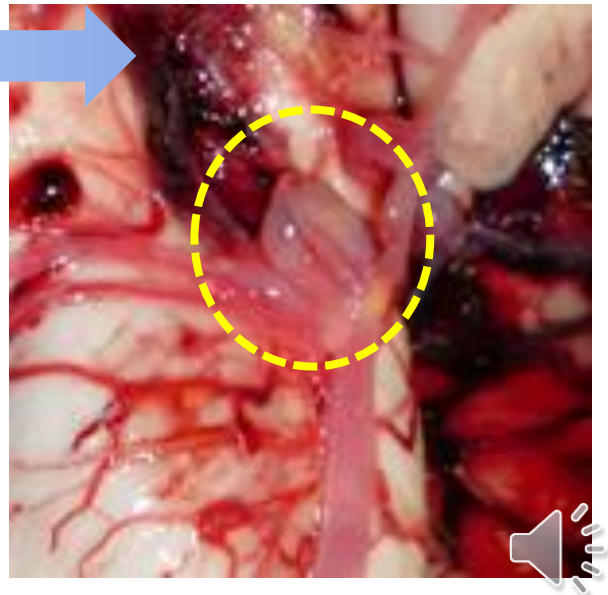
- Progressive dilatation of cerebral artery = wall thinning until rupture





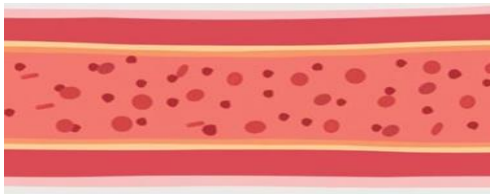




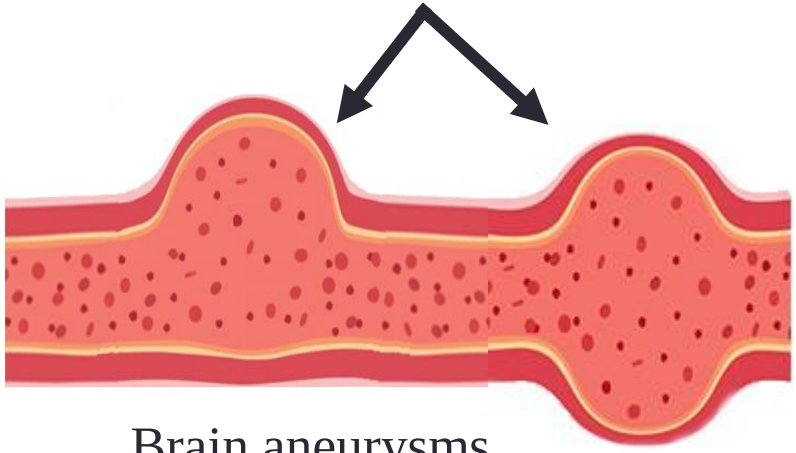


Brain aneurysm: formation

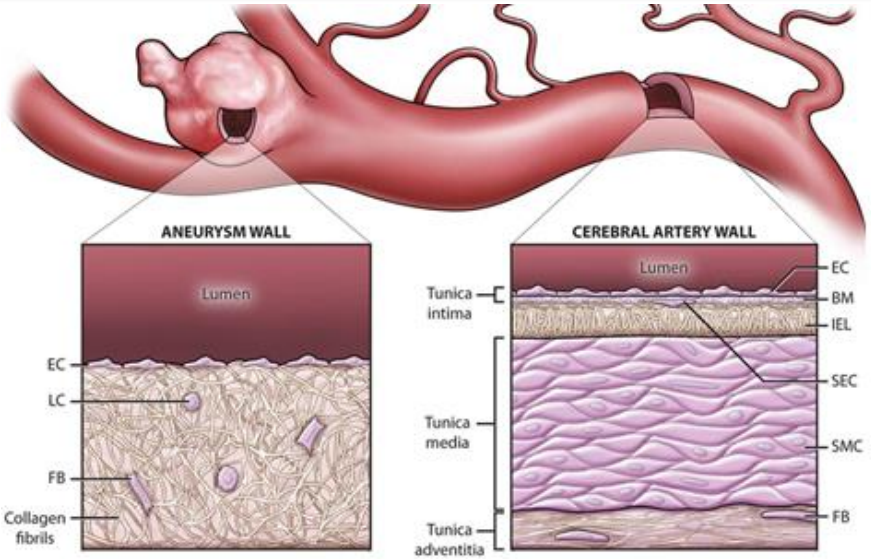
- Arterial wall degeneration = aneurysm formation
  - Intraluminal arterial pressure = aneurysm ballooning → progressive wall thinning = weakening → rupture



Normal brain artery

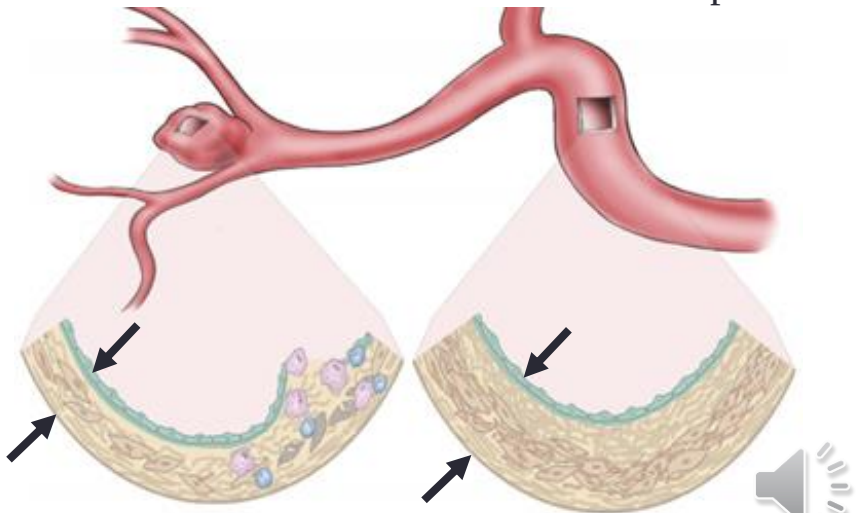


Brain aneurysms



Tunica media ABSENT

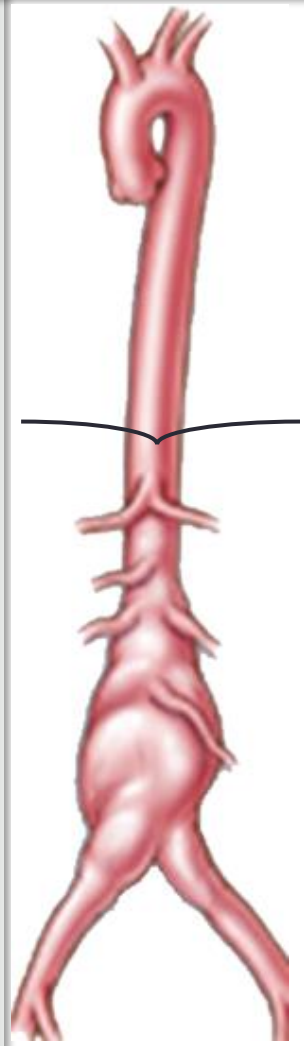
Tunica media present



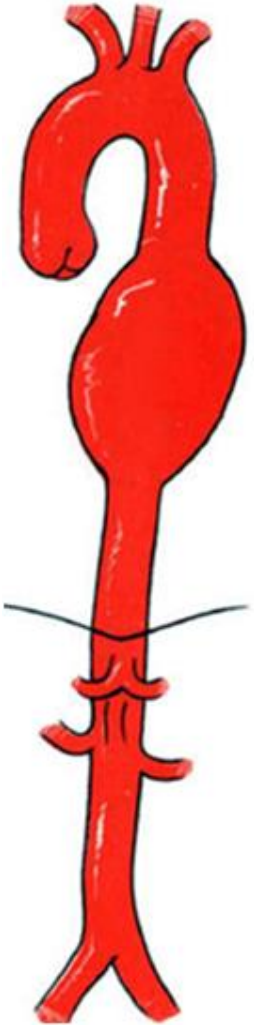
Brain aneurysm wall

Normal brain artery wall

Other arteries can also be affected

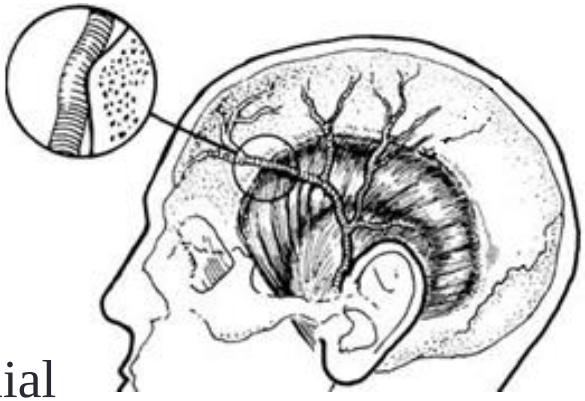


Abdominal aorta



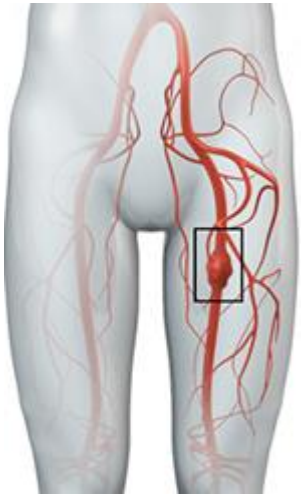
Thoracic aorta

Extracranial internal carotid artery

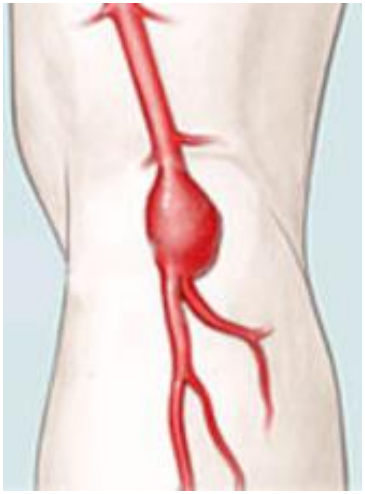


Superficial temporal artery

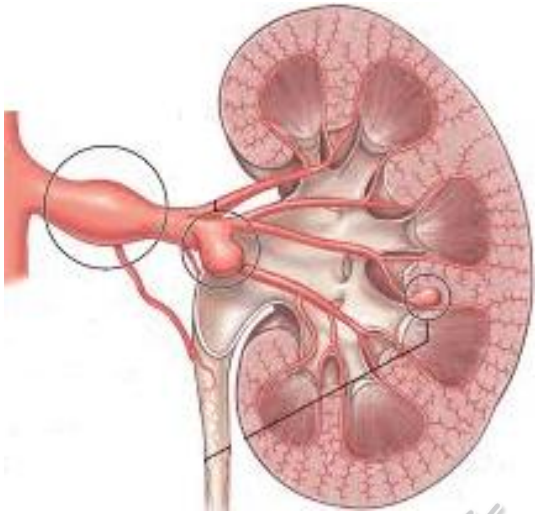
Subclavian artery



Femoral artery



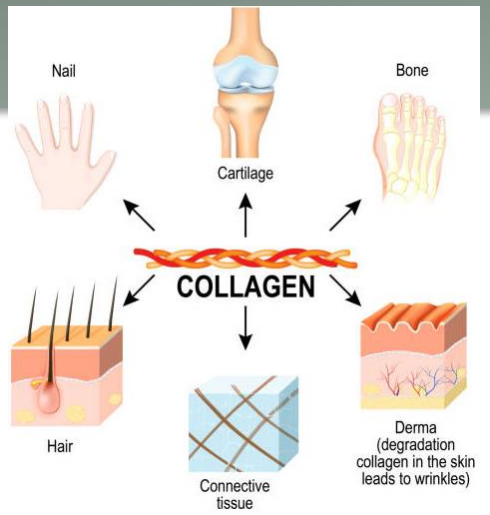
Popliteal artery



Kidney arteries

Brain aneurysms: association with connective tissue disorders

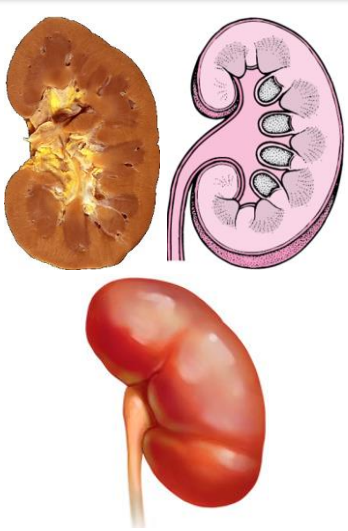
- Often overlooked
- No regular family screening
- Brain aneurysm just another of many problems suffered by a patient



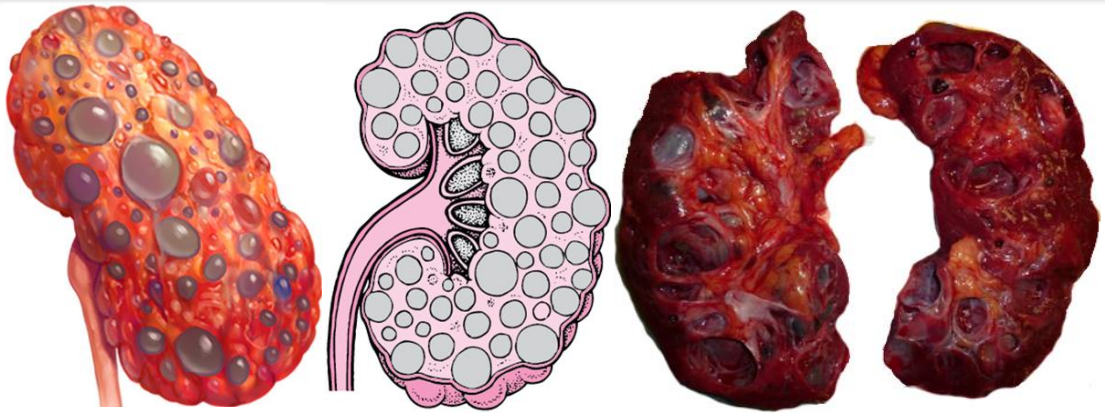
Connective tissue disorder	Common features	Intracranial Aneurysm
Marfan, EDS, NF-1 and LDS: -Fibrosis of SMCs -Cystic medial necrosis -Elastic fibres derangement -Dermal Collagen thin & rare -Fragmentation of Elastic fibres -Diffuse medial degeneration	*-Tunica Intima Fibrosis* *-Proliferation of intima* *-Muscle fibre disarray* *-Disorganized SMCs*	-Fragmentation of IEL -Metachromasia in the intima -Metachromasia in subendothelium -Muscle fibre disarray. -Depletion of cellular compon

Association with some connective tissue diseases: polycystic kidney disease

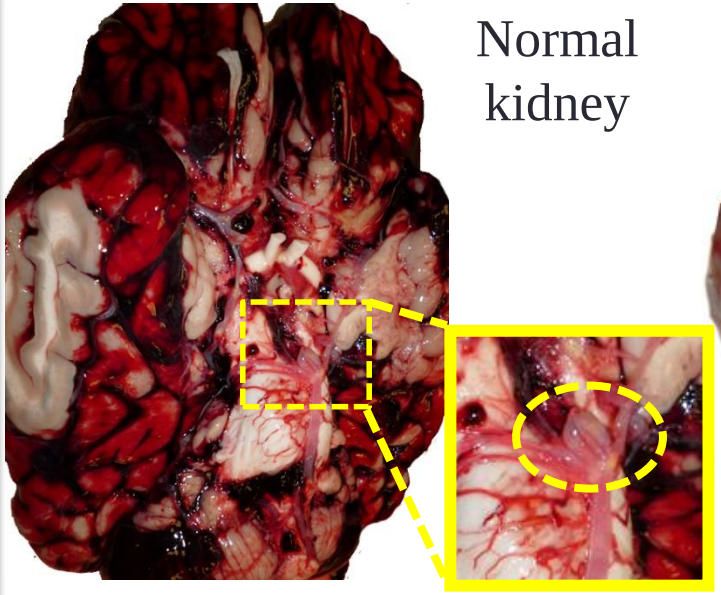
- Autosomal dominant
- 1:500 live births
- Kidney failure by 40-60 years
- Brain aneurysm incidence 9%



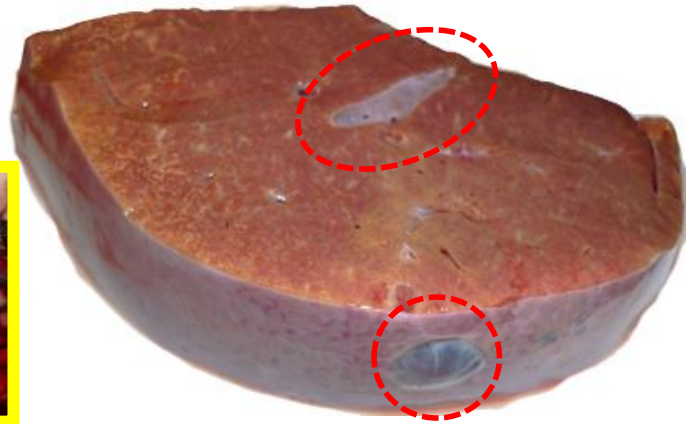
Normal kidney



Polycystic kidney



Brain aneurysm

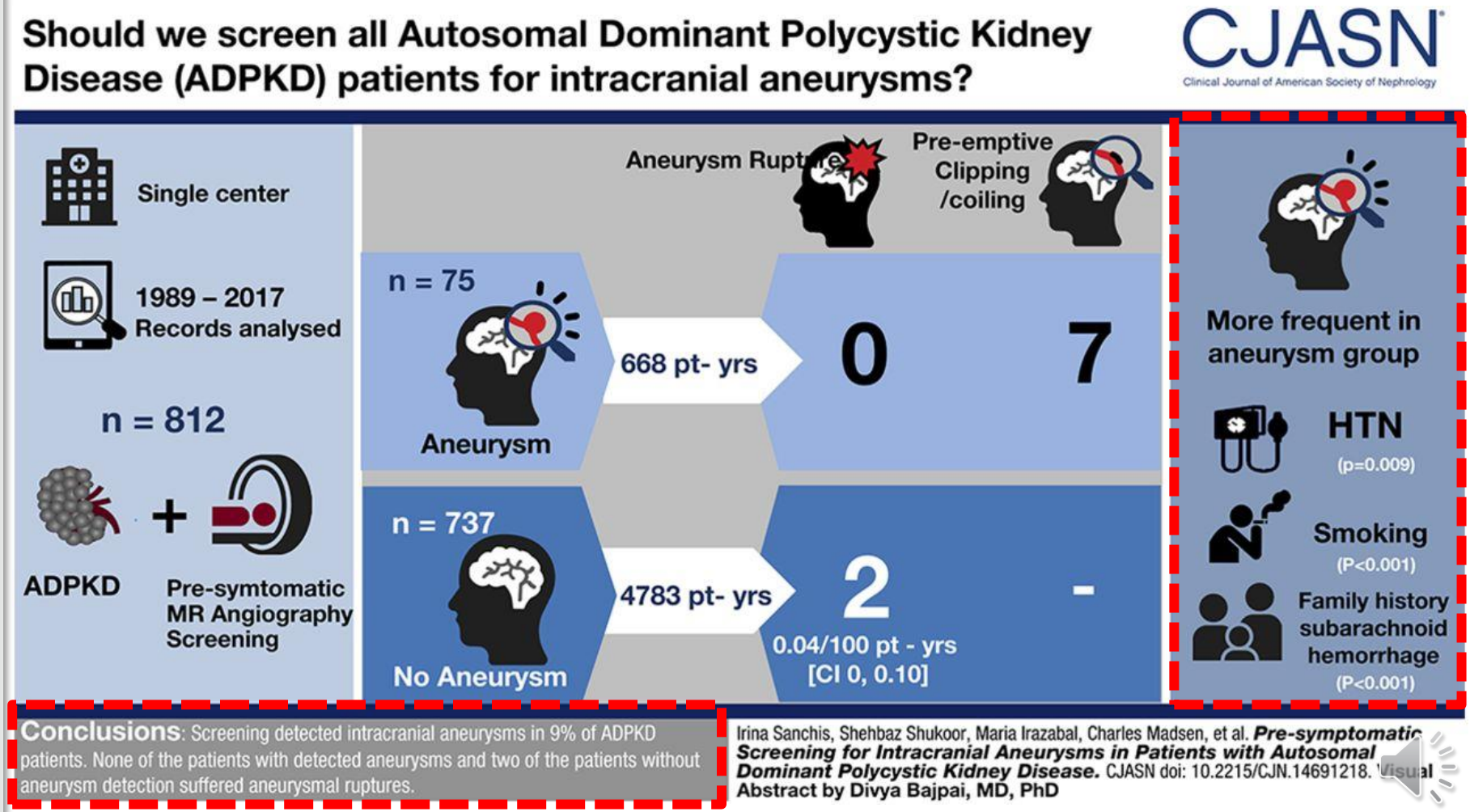


Polycystic liver

Polycystic kidneys

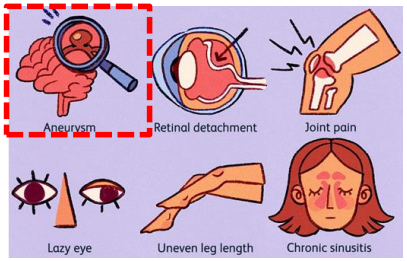
Brain aneurysms & polycystic kidney disease

- Low rupture risk but prophylactic treatment recommended



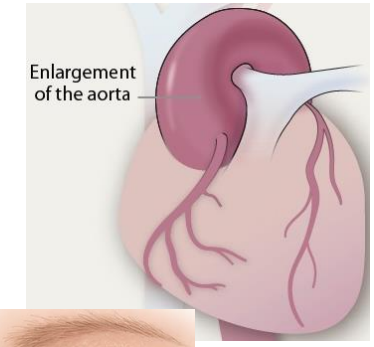
Association with connective tissue disorders: Marfan syndrome

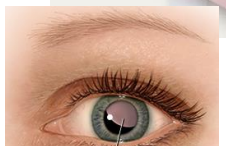
- Brain aneurysms one of many systemic disorders

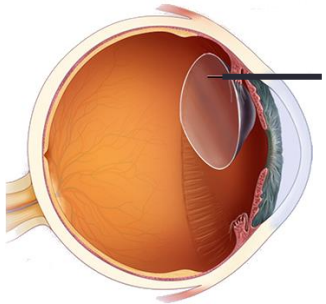


Pectus excavatum

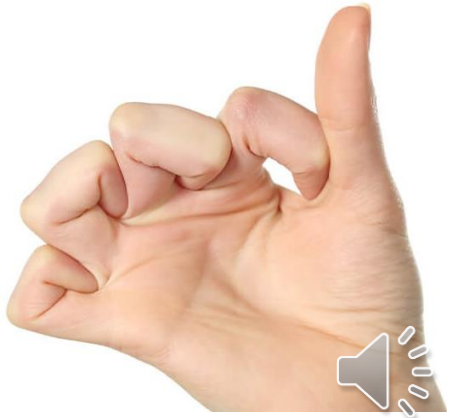
arachnodactyly



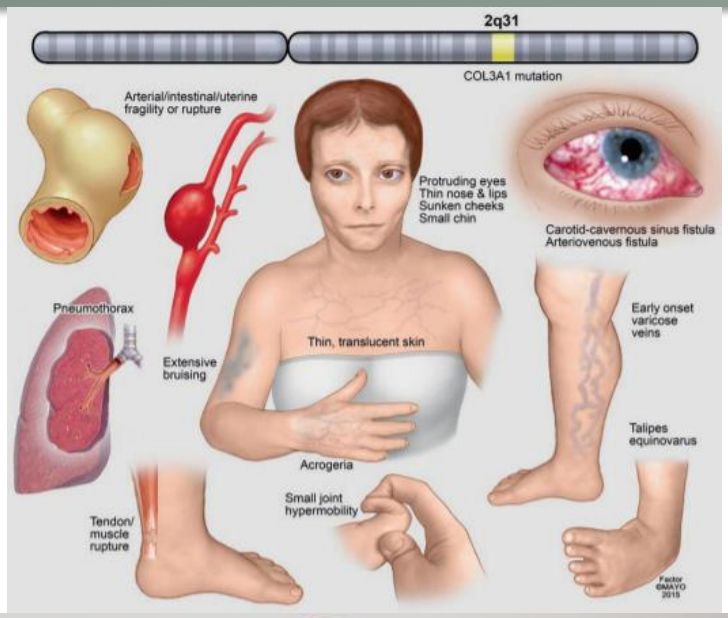




Lens dislocation

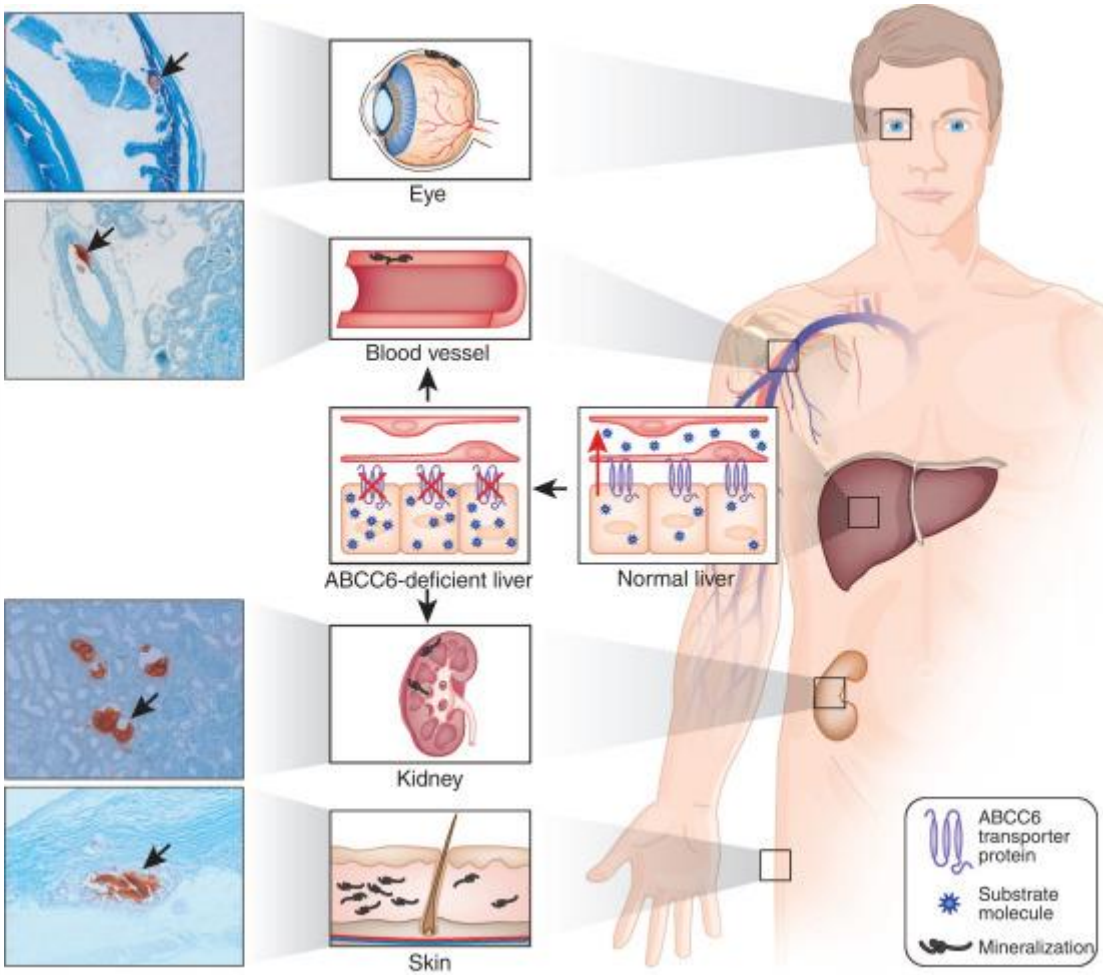


Association with connective tissue disorders: Ehlers-Danlos syndrome type 4



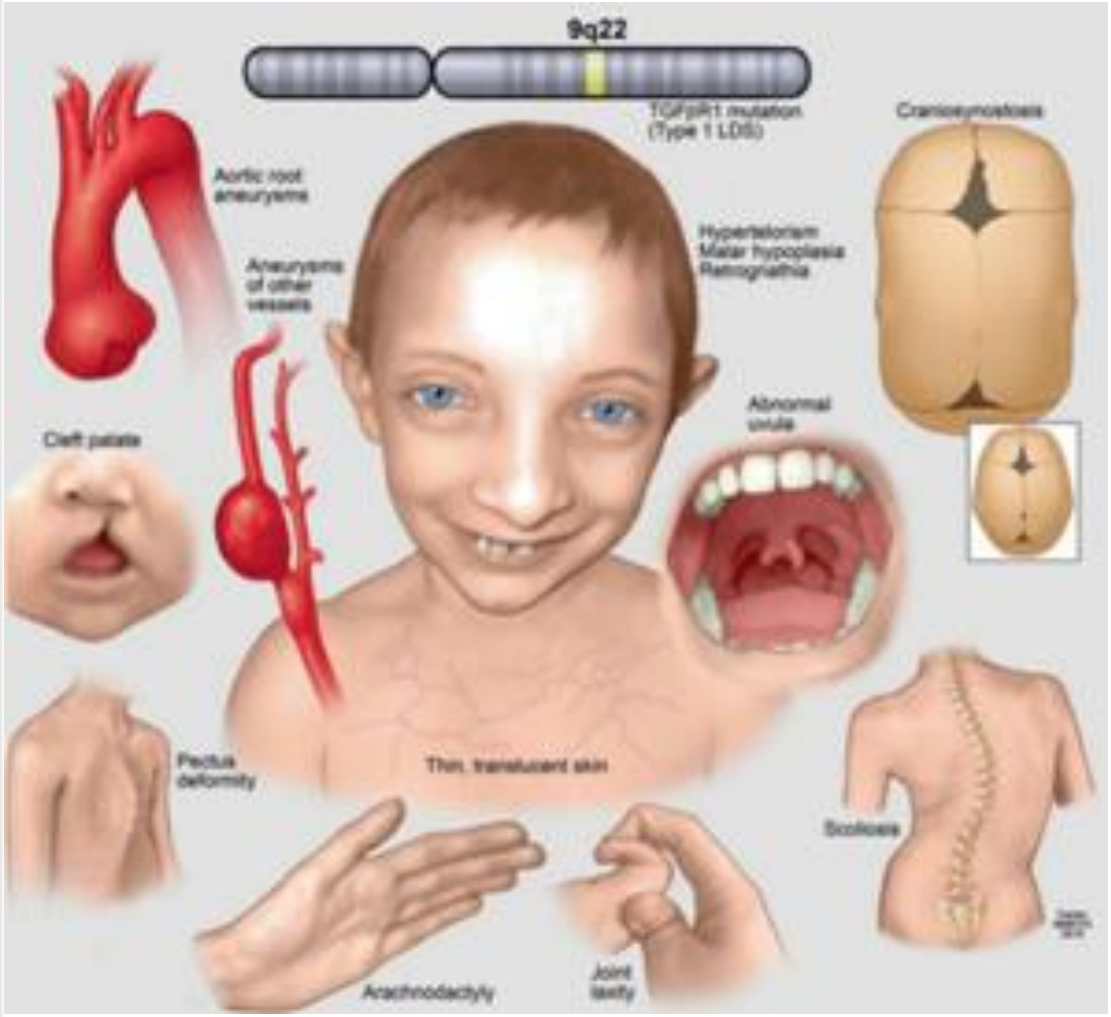
Association with connective tissue disorders: pseudoxanthoma elasticum

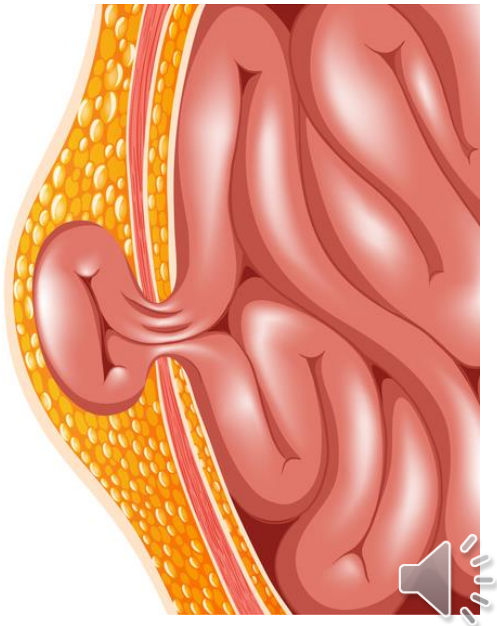
- Skin & articular laxity



Association with connective tissue disorders: Loeys-Dietz syndrome

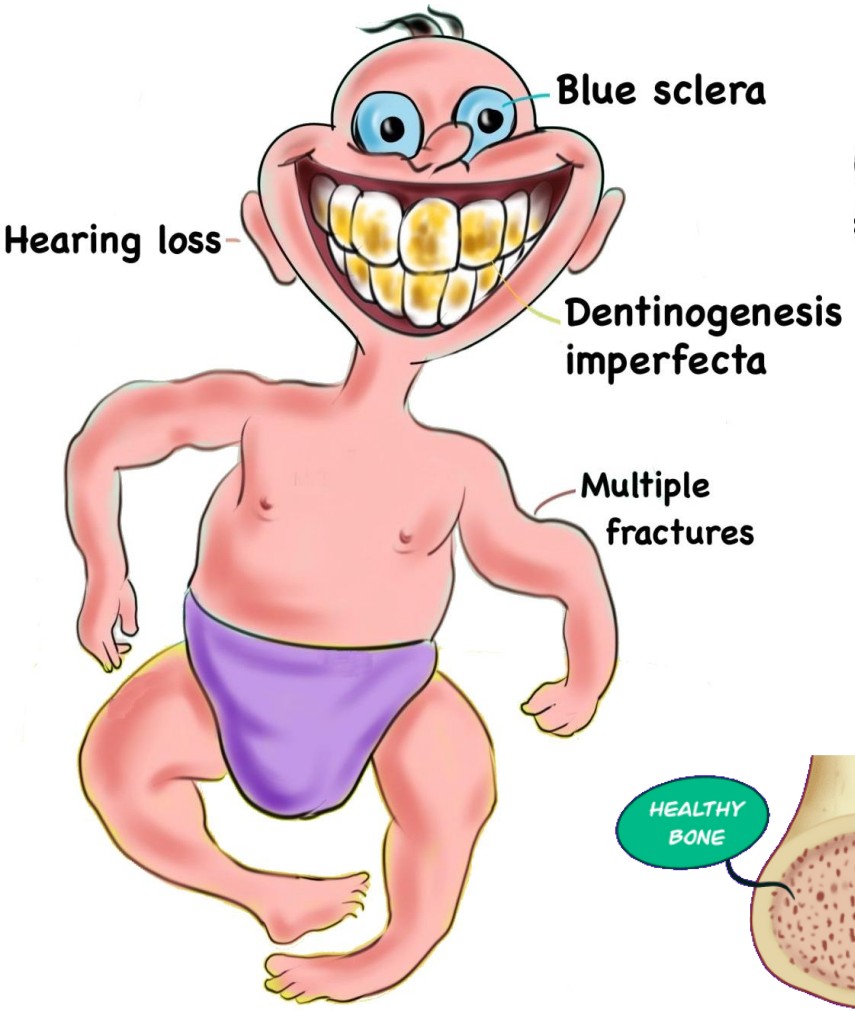
- Multiple systemic disorders

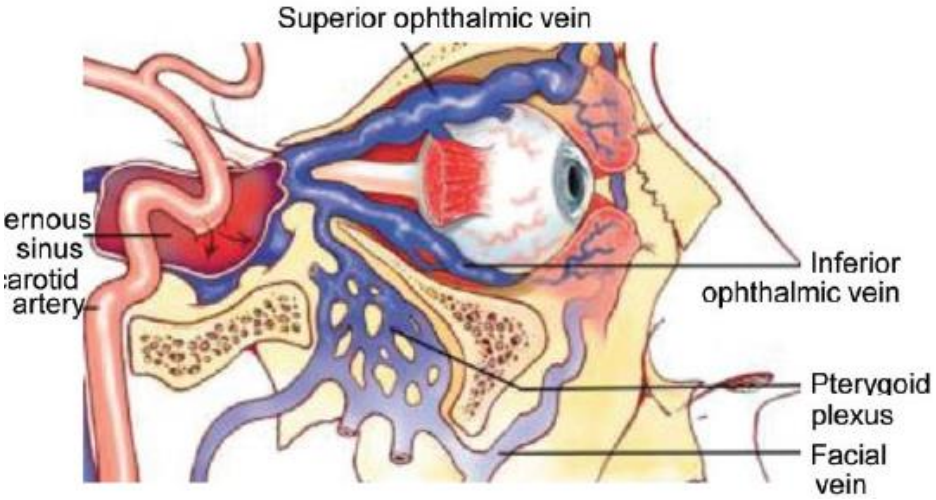


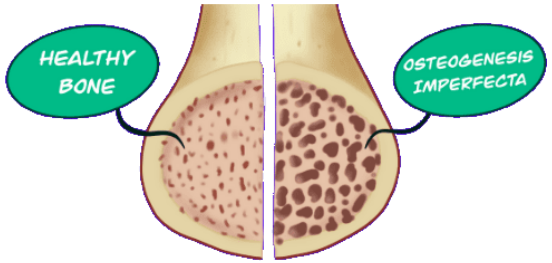


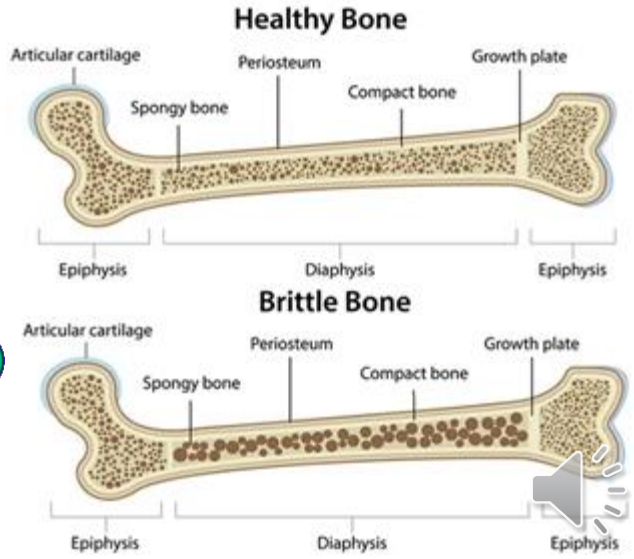
Association with connective tissue disorders: osteogenesis imperfecta

- Associated with carotid-cavernous fistulas



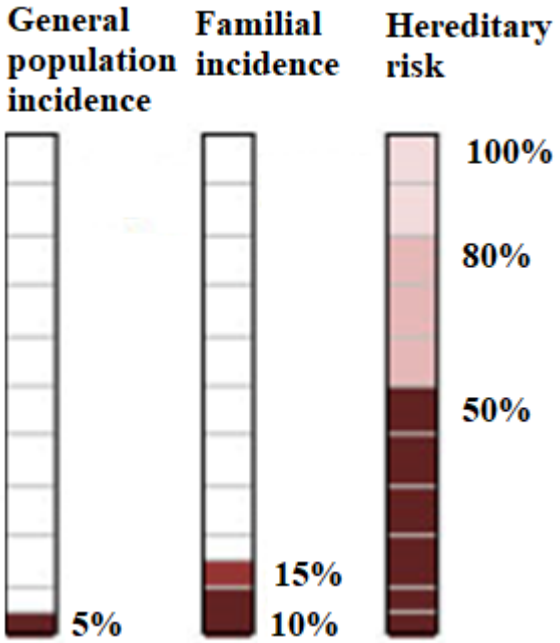


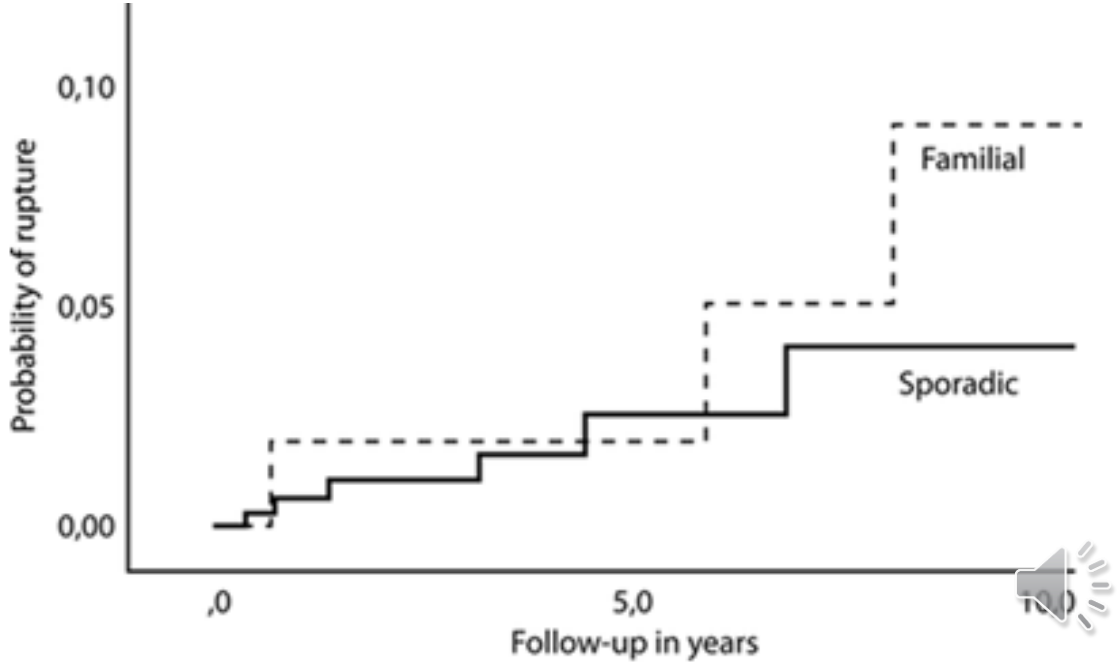




Brain aneurysms: familial incidence

- Low but exists
- Higher rupture risk
- Screening IF family with > two or more affected people (parents or siblings)
- Autosomal dominant or recessive pattern



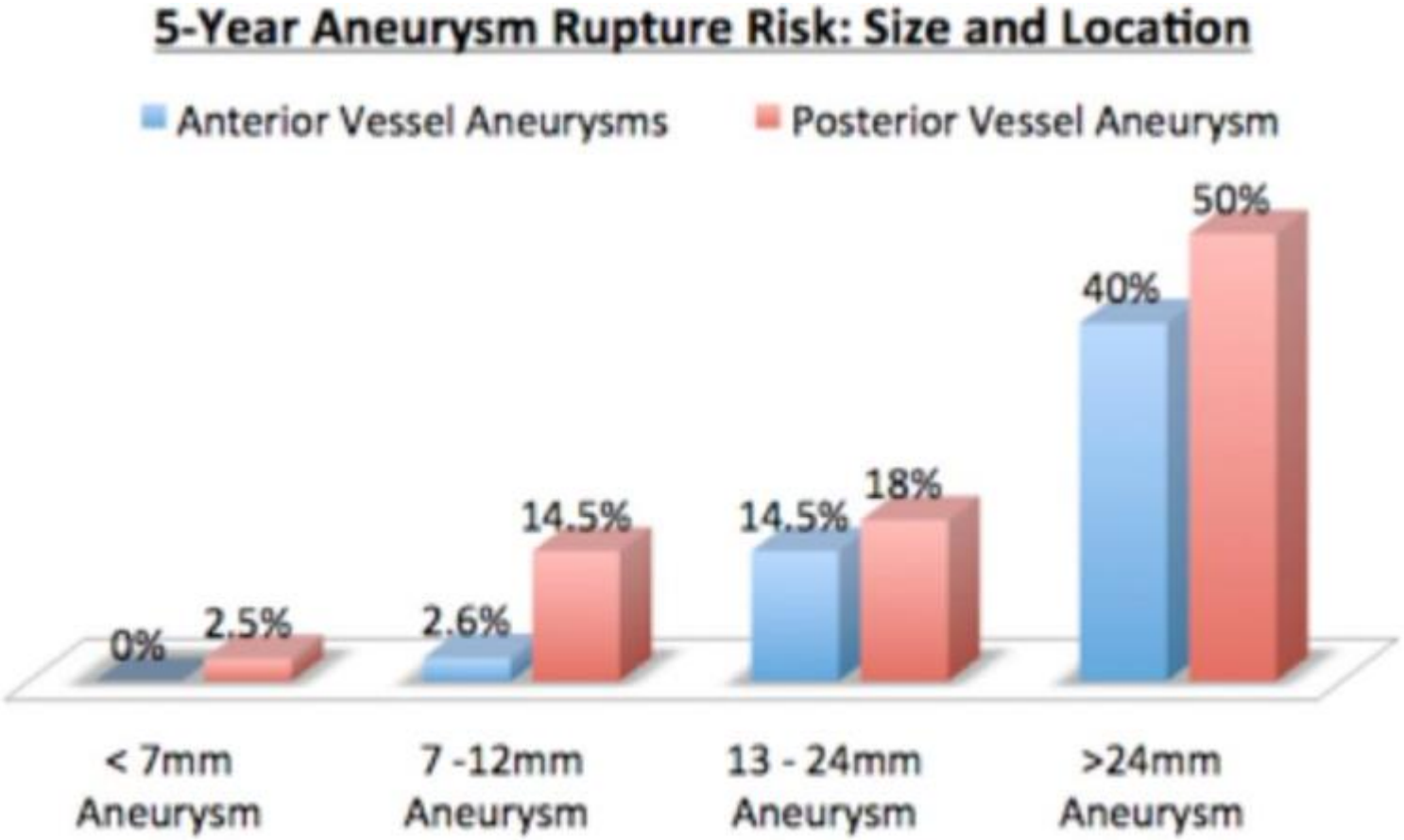


Brain aneurysm: epidemiology

- 0.2% population harbor brain aneurysms = 200 / 100,000 inhabitants
  - 1/50 persons
- At any age, **> 30-60 years**
- ♀ **/** ♂ **3/2**
- **93% aneurysms <10mm & 7% >10mm**
- Rupture risk 1.8% / year
  - Incidental aneurysm finding 0.8% / year
    - < 10mm 0.7% / year
    - > 10mm 4% / year
- Incidence of ruptured aneurysms: 6 / 100,000 inhabitants / year
  - 15% die BEFORE reaching the hospital
  - 46% die in the hospital
  - 60% of survivors with neurological / cognitive sequelae
    - 30% severe

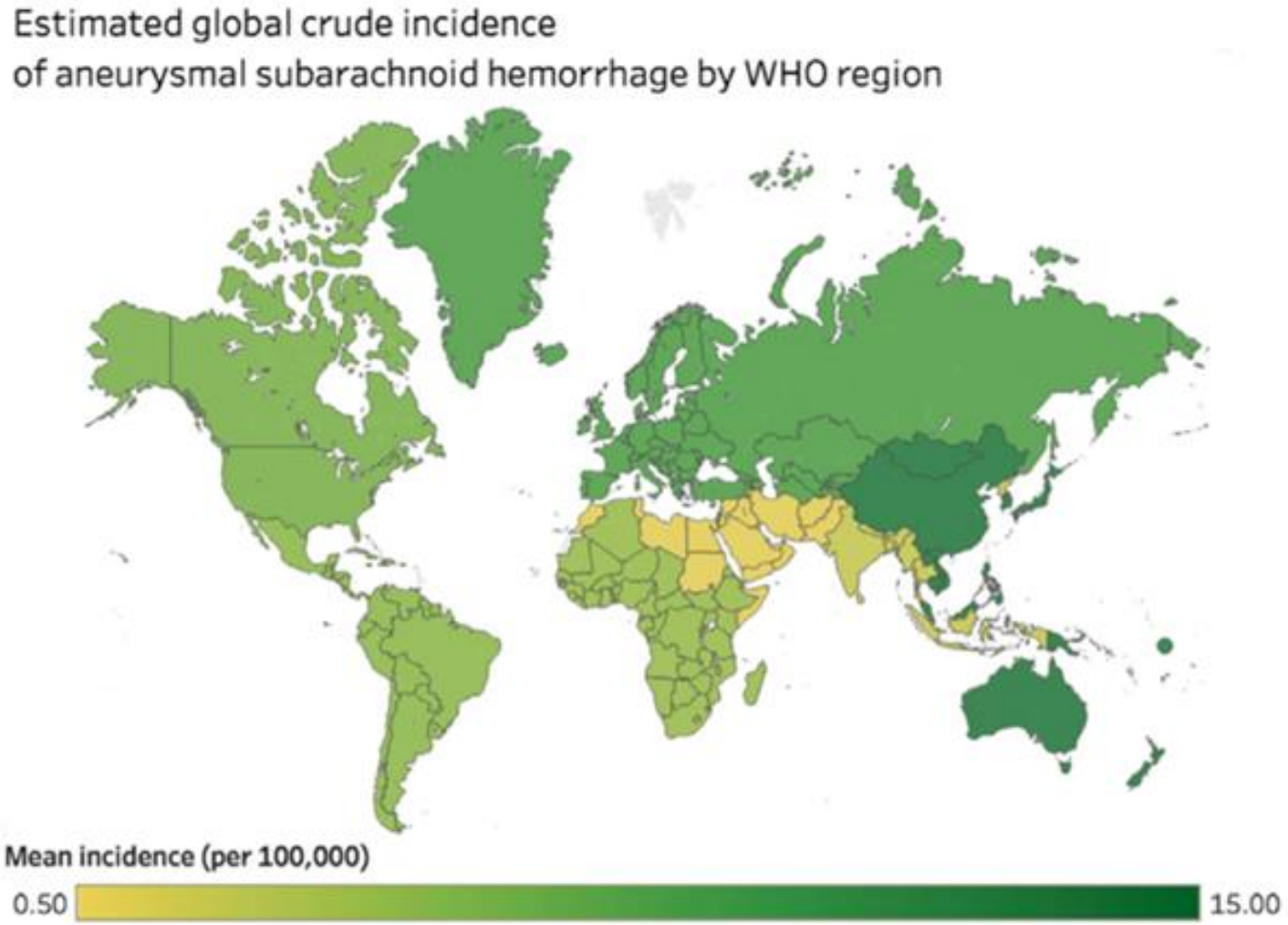
Brain aneurysms: rupture risk

- Risk increases with size and posterior circle of Willis location
  - Important for prognosis in case of incidental aneurysm finding

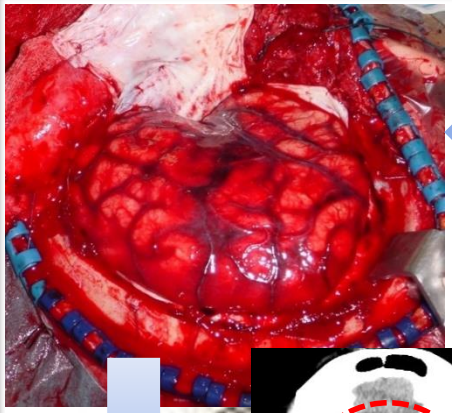


The incidence varies according to areas

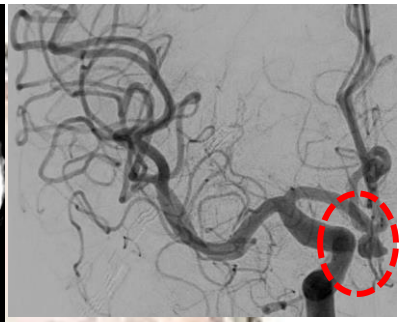
- Higher in Finland and Japan

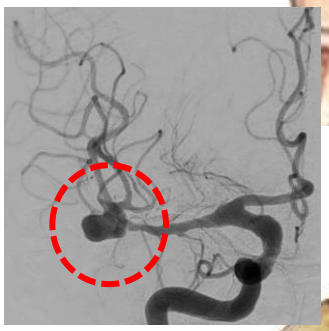


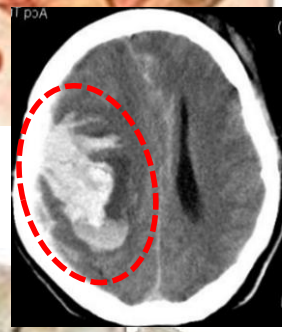
Aneurysm rupture: subarachnoid haemorrhage or intra-cerebral haematoma

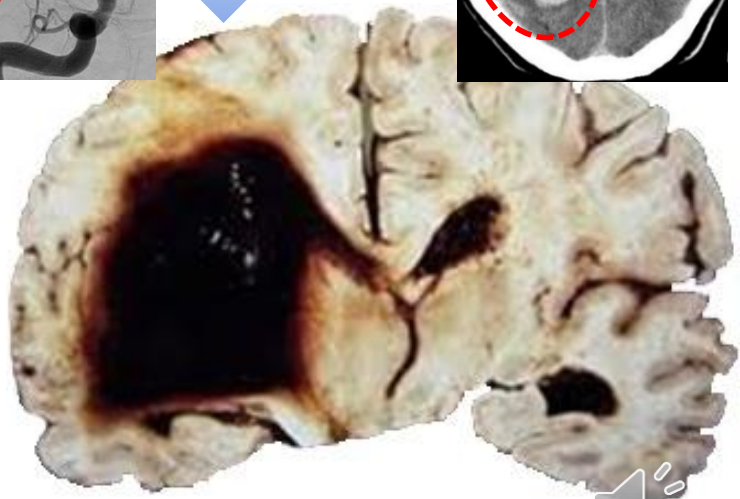


Aneurysm rupture





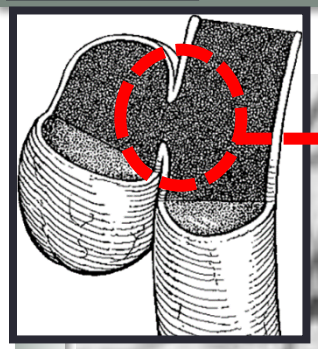




Subarachnoid haemorrhage

Intracerebral haematoma

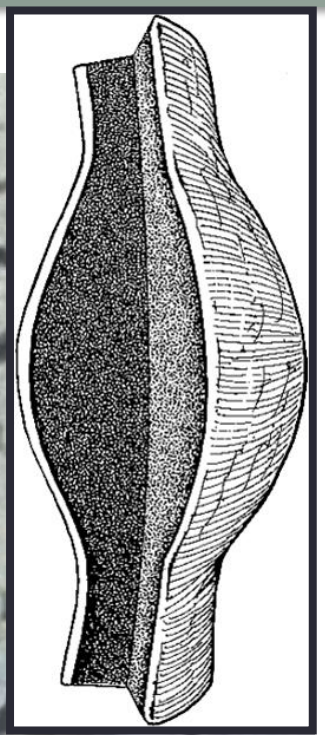
Brain aneurysms: types



Neck

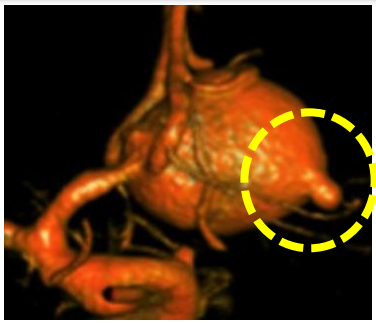
Neck

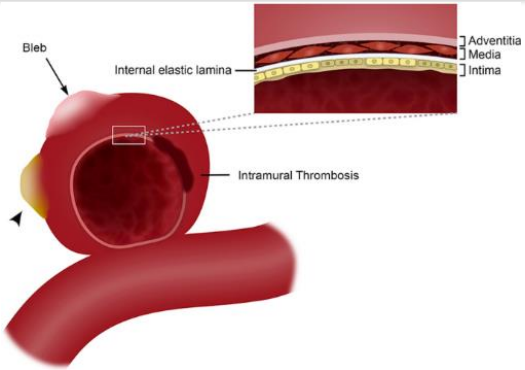
Saccular = has neck

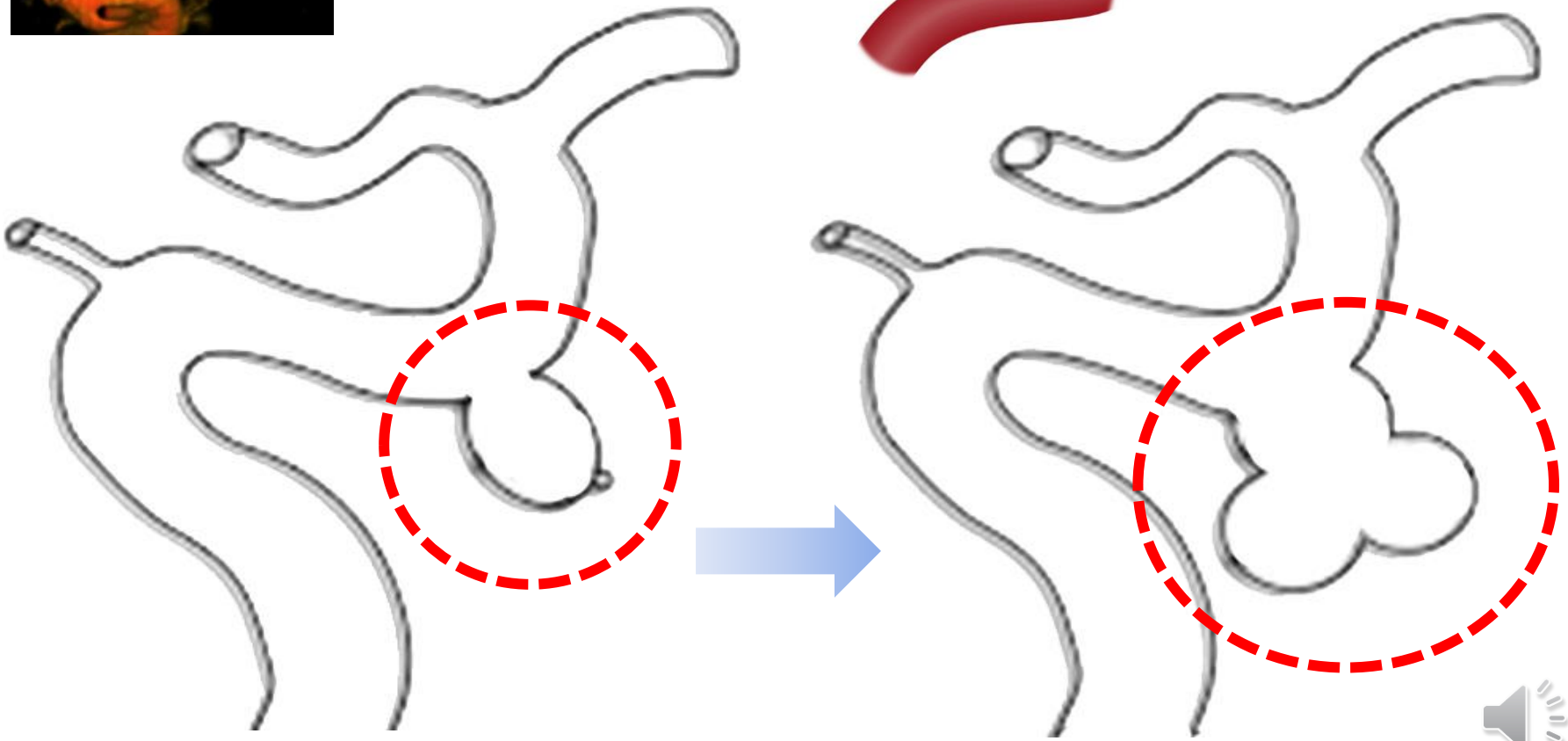


Fusiform = NO neck

Aneurysm growth = progressive wall thinning = ⇑ bleeding risk ≅ ☠

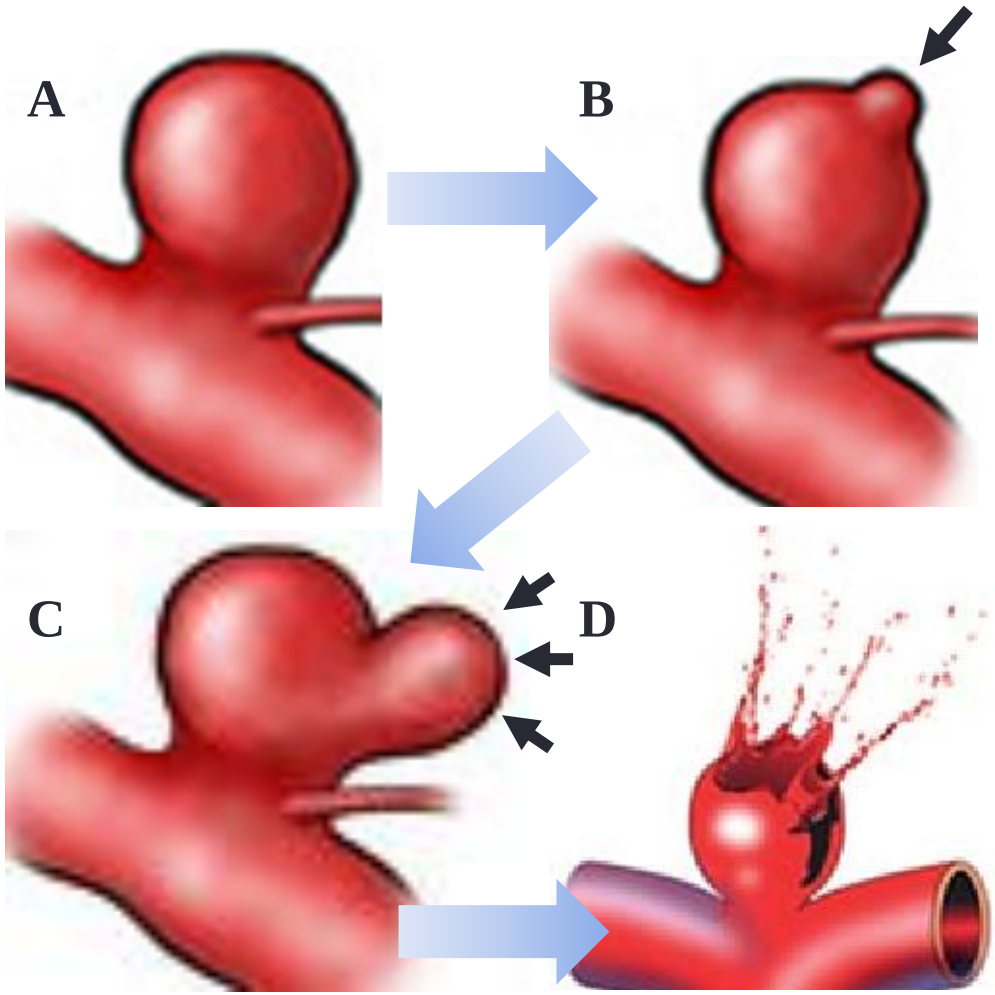


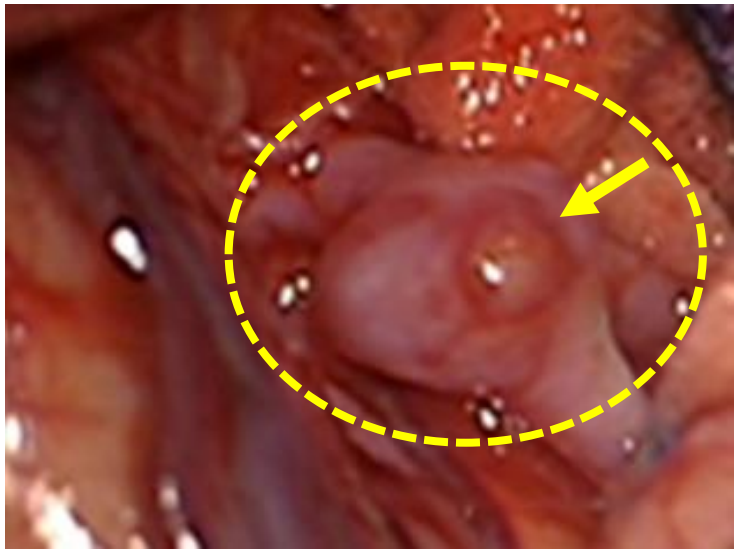


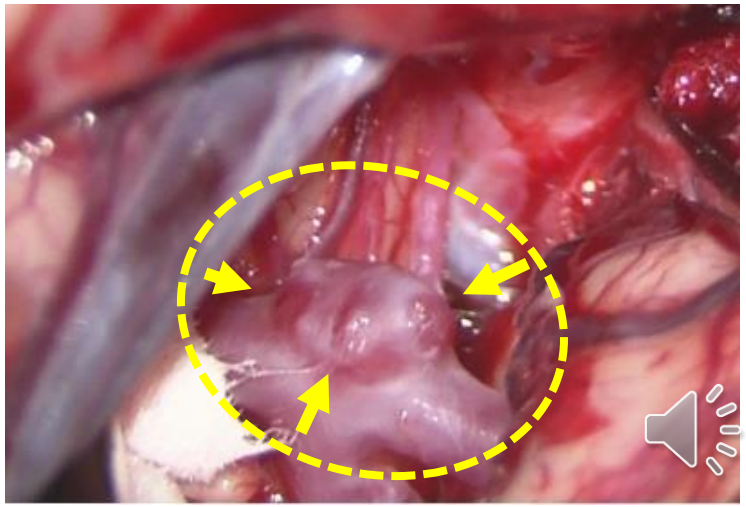


Baby aneurysm = thinner wall = higher rupture risk

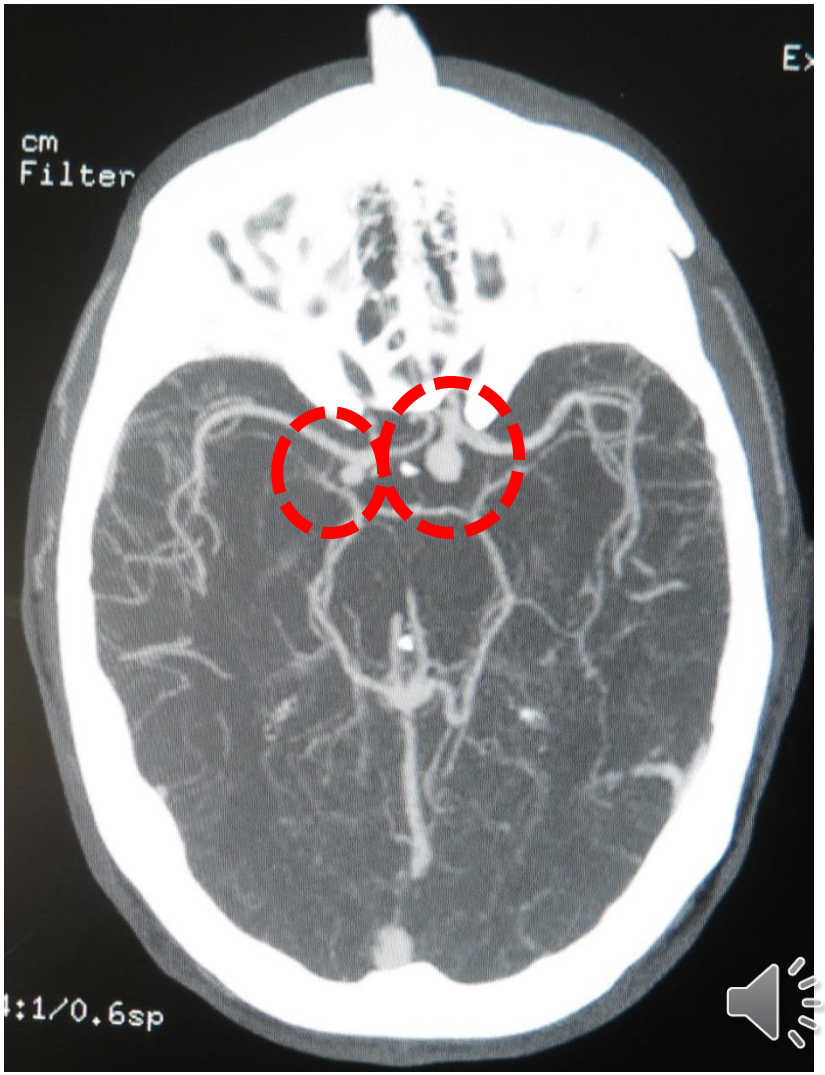
- A small aneurysm in the wall of an aneurysm = even small aneurysms may bleed



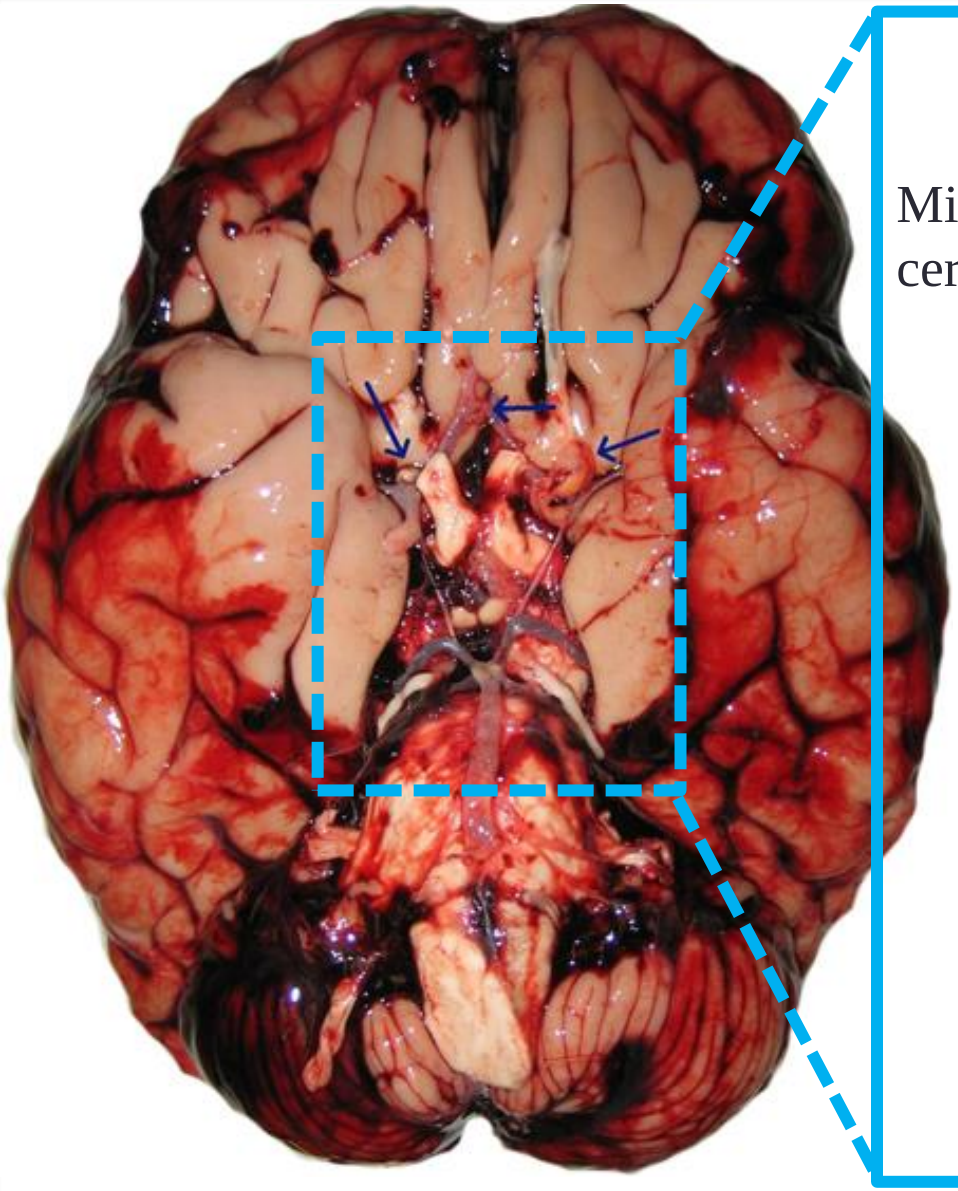


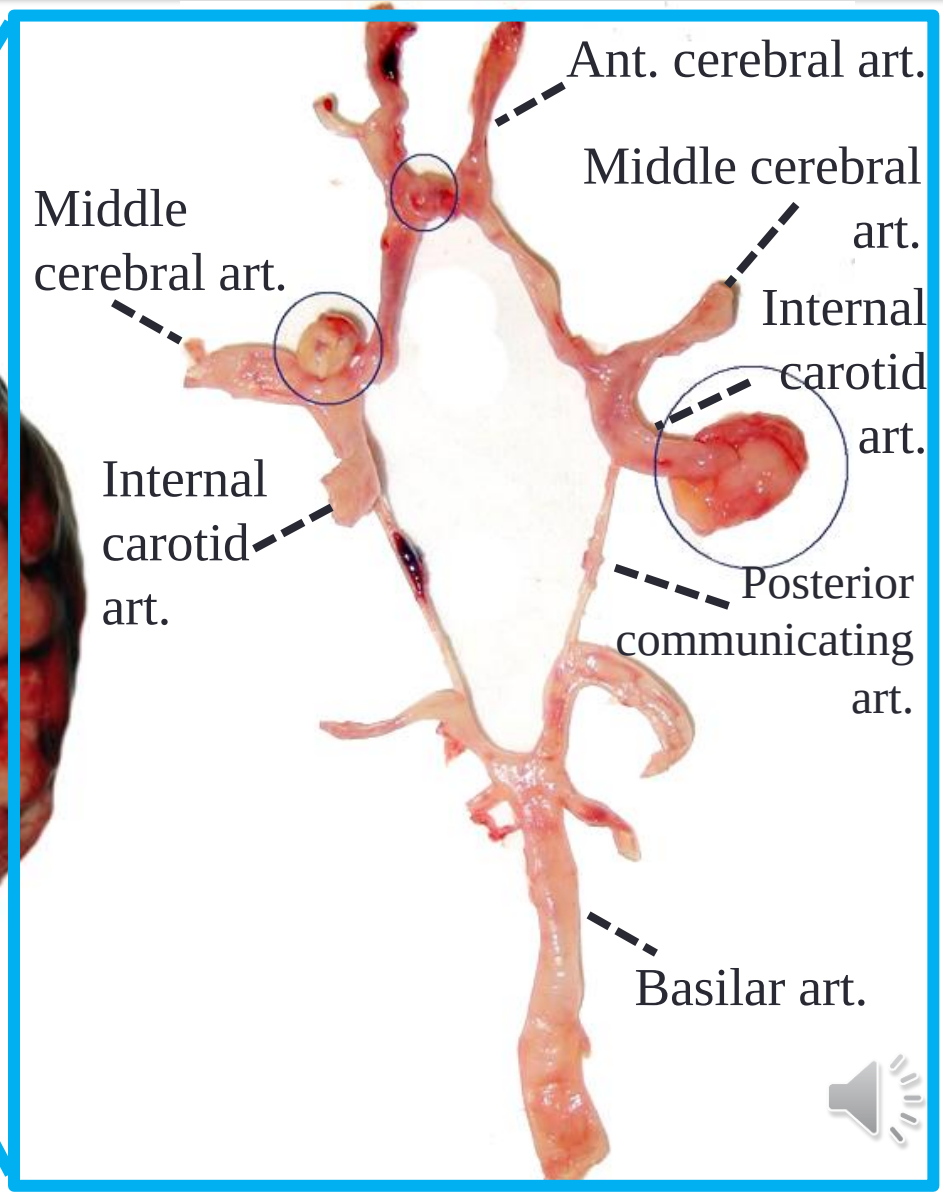


Brain aneurysms: often multiple



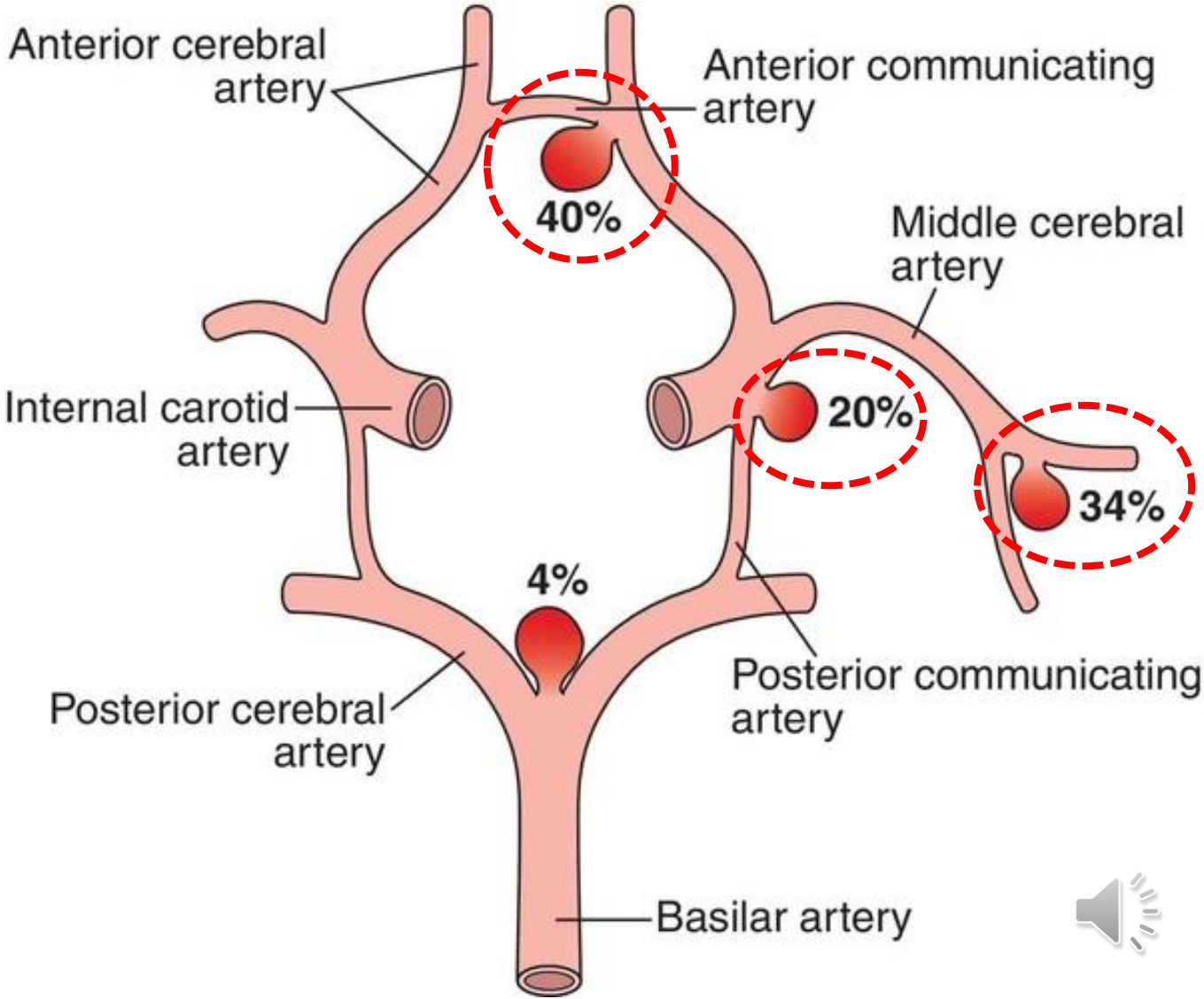
Multiple brain aneurysms: necropsy





Brain aneurysm location: percentage

- More common in anterior circle of Willis



Brain aneurysms: risk factors (1)

- ♀
- Smoking
- Arterial hypertension
- Family background
- Age over 40 years
- Bleeding risk factors
  - Contraceptives
  - Cocaine
  - Alcohol

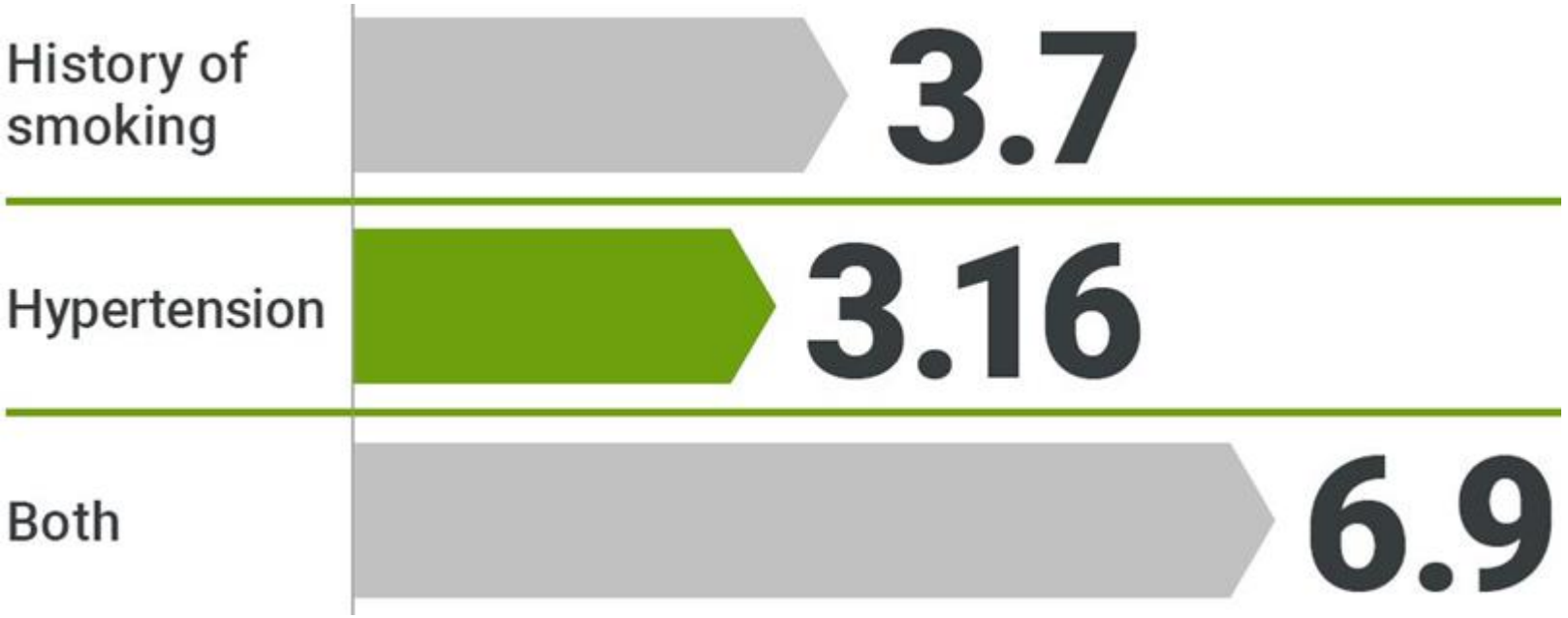
REMEMBER?

Usual risk factors: smoking + ♀

Possible new aneurysm in another location after some time

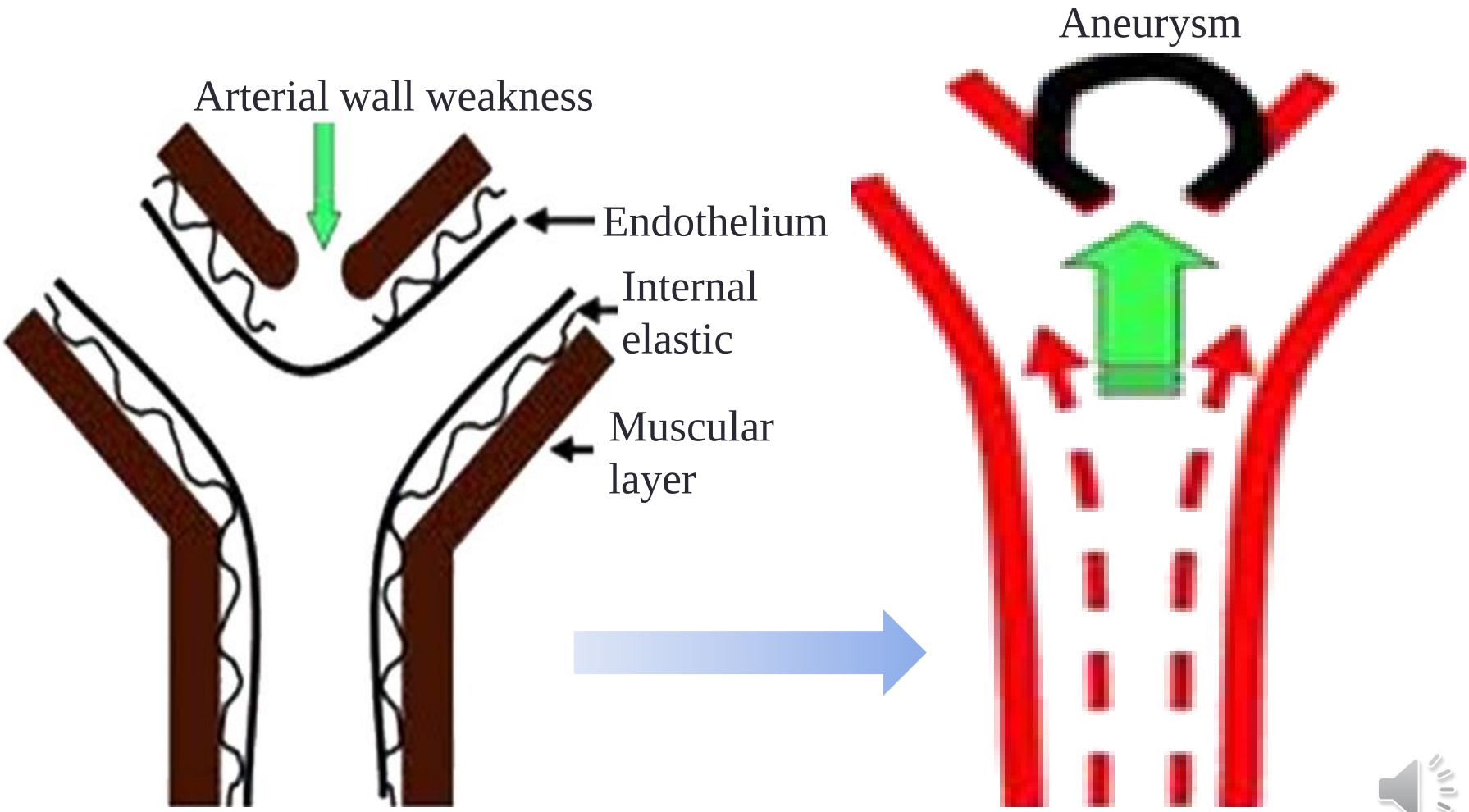
Brain aneurysms: risk factors (2)

- Association of smoking and arterial hypertension doubles aneurysm incidence for women 30-60 years old



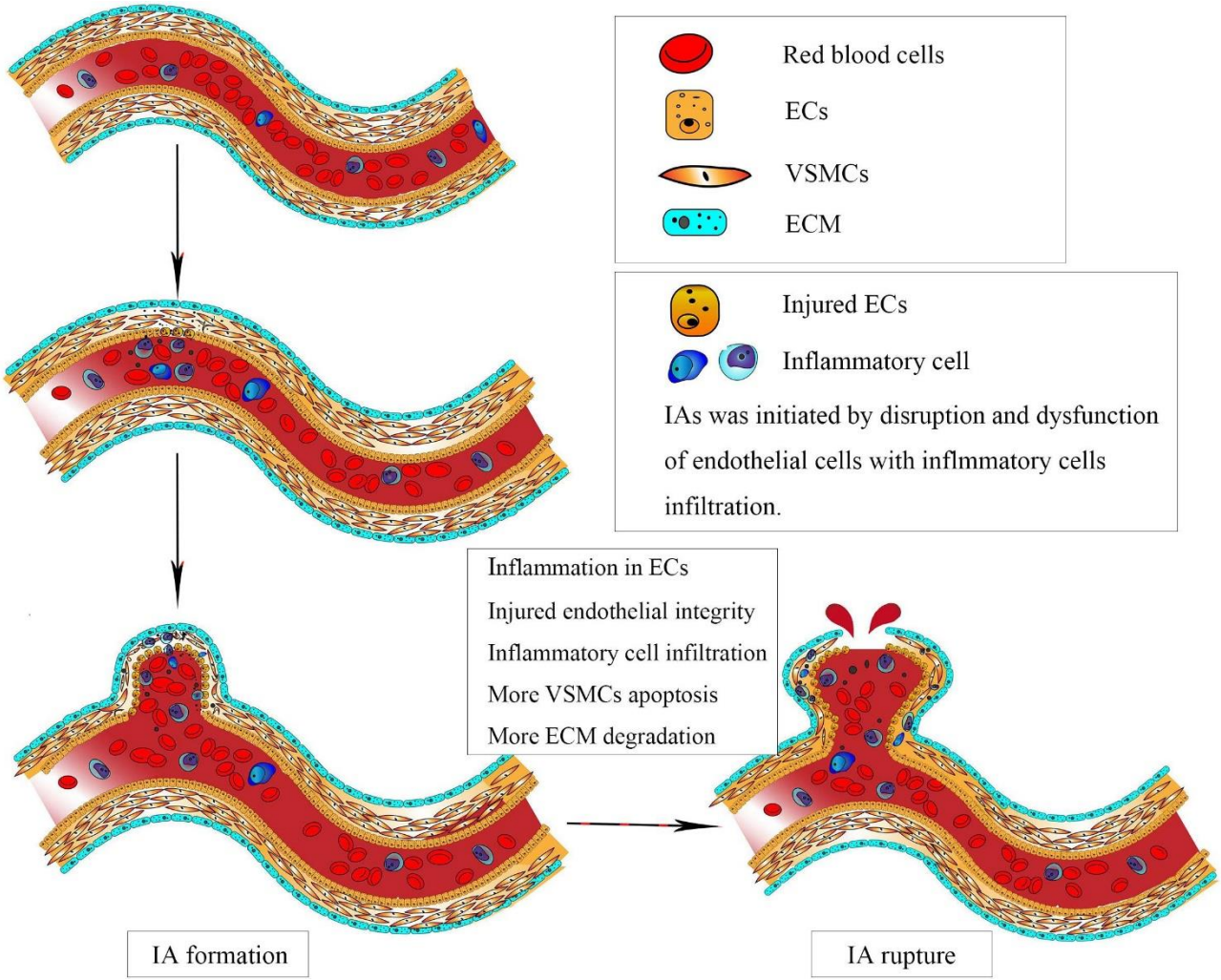
Arterial wall weakness = congenital?

- Difficult to prove



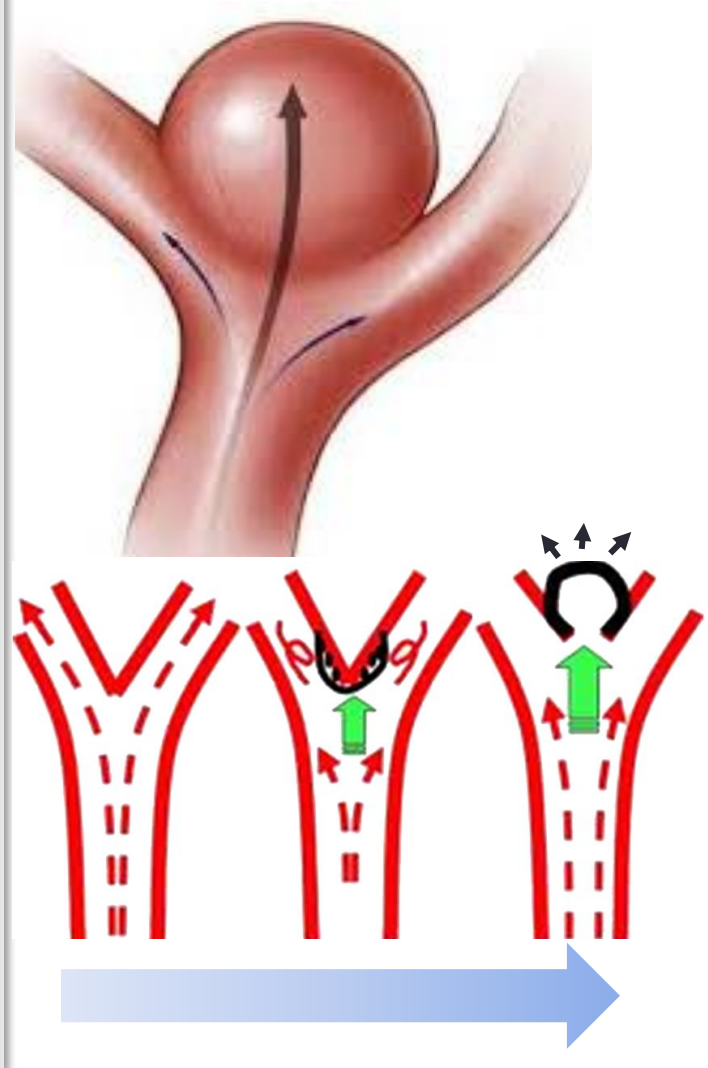
Arterial wall weakness = local / systemic inflammatory disease

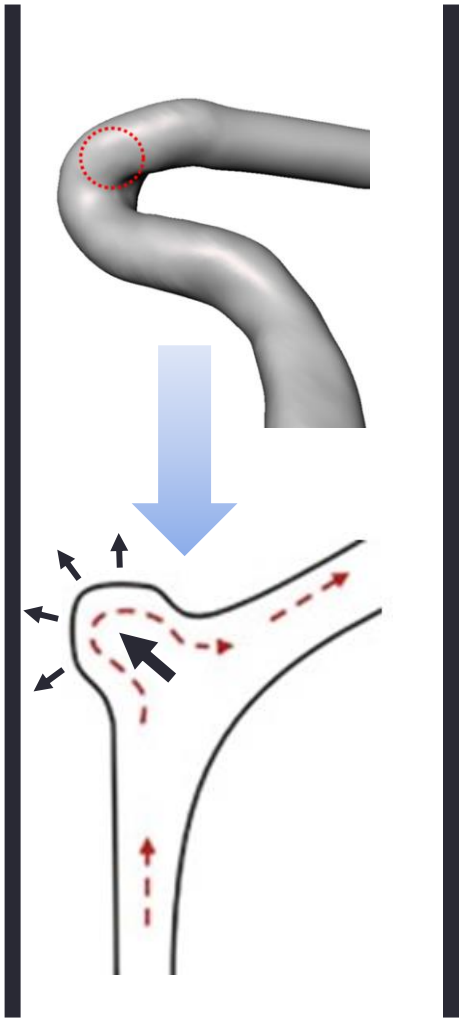
- If is a systemic process = more arterial locations possible

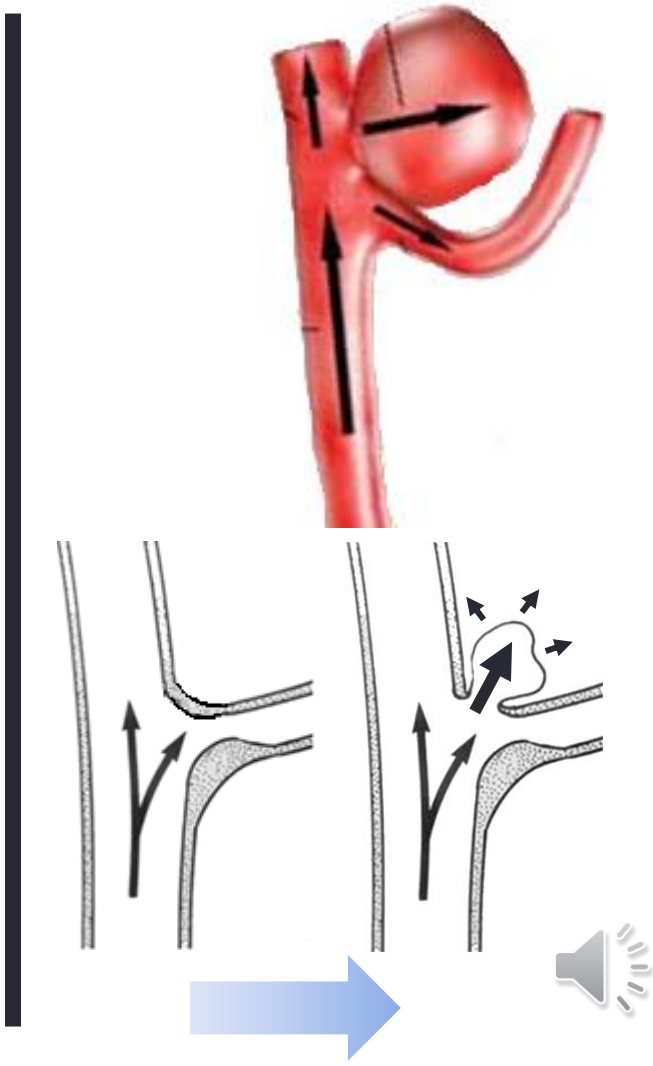


Brain aneurysm formation: haemodynamic factors (1)

- ↑ frequent at bifurcations, bends, and collateral vessel outlet

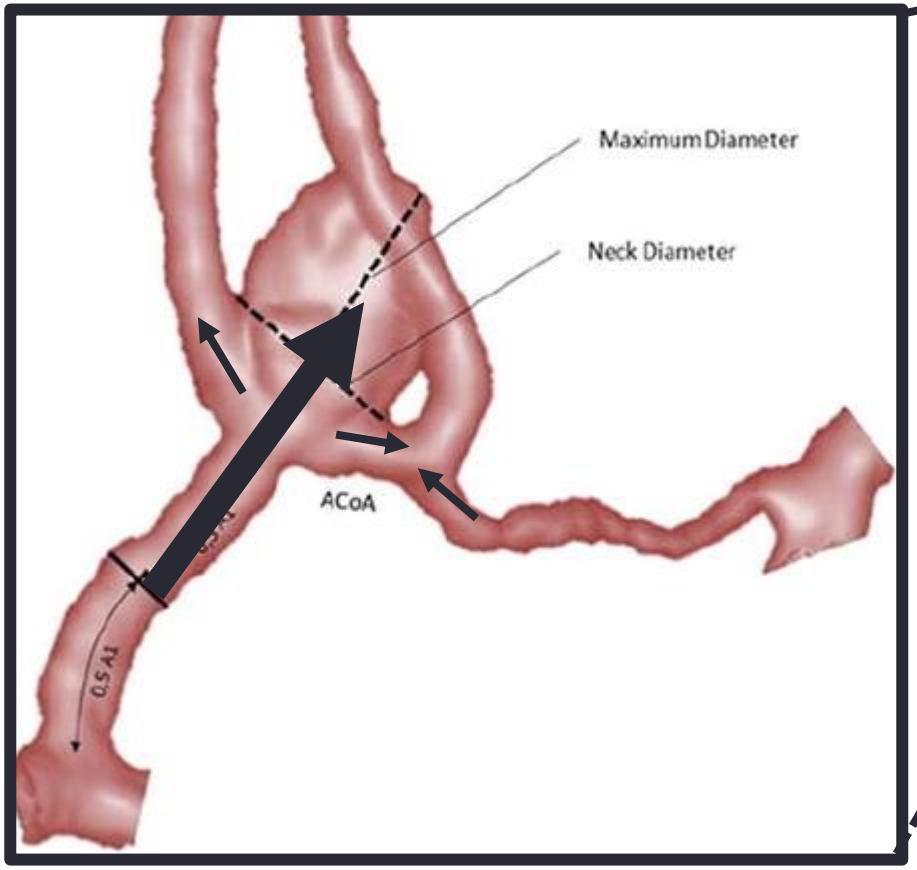


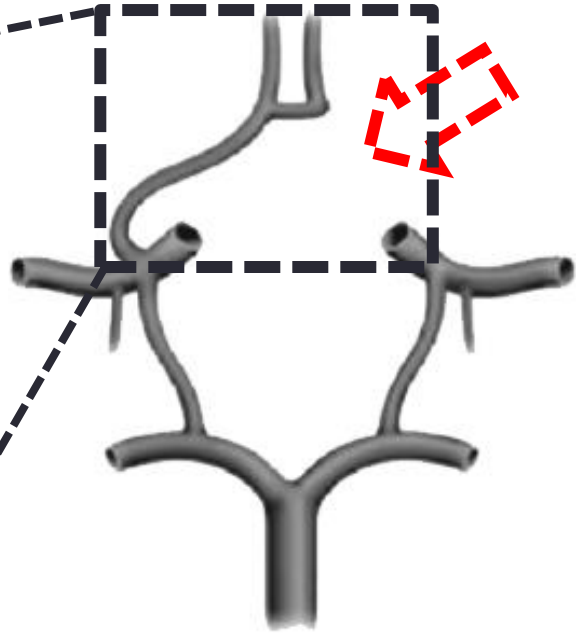




Brain aneurysm formation: haemodynamic factors (2)

- Agenesis of one artery = overflow on others = higher haemodynamic stress = ↑ chance aneurysm formation

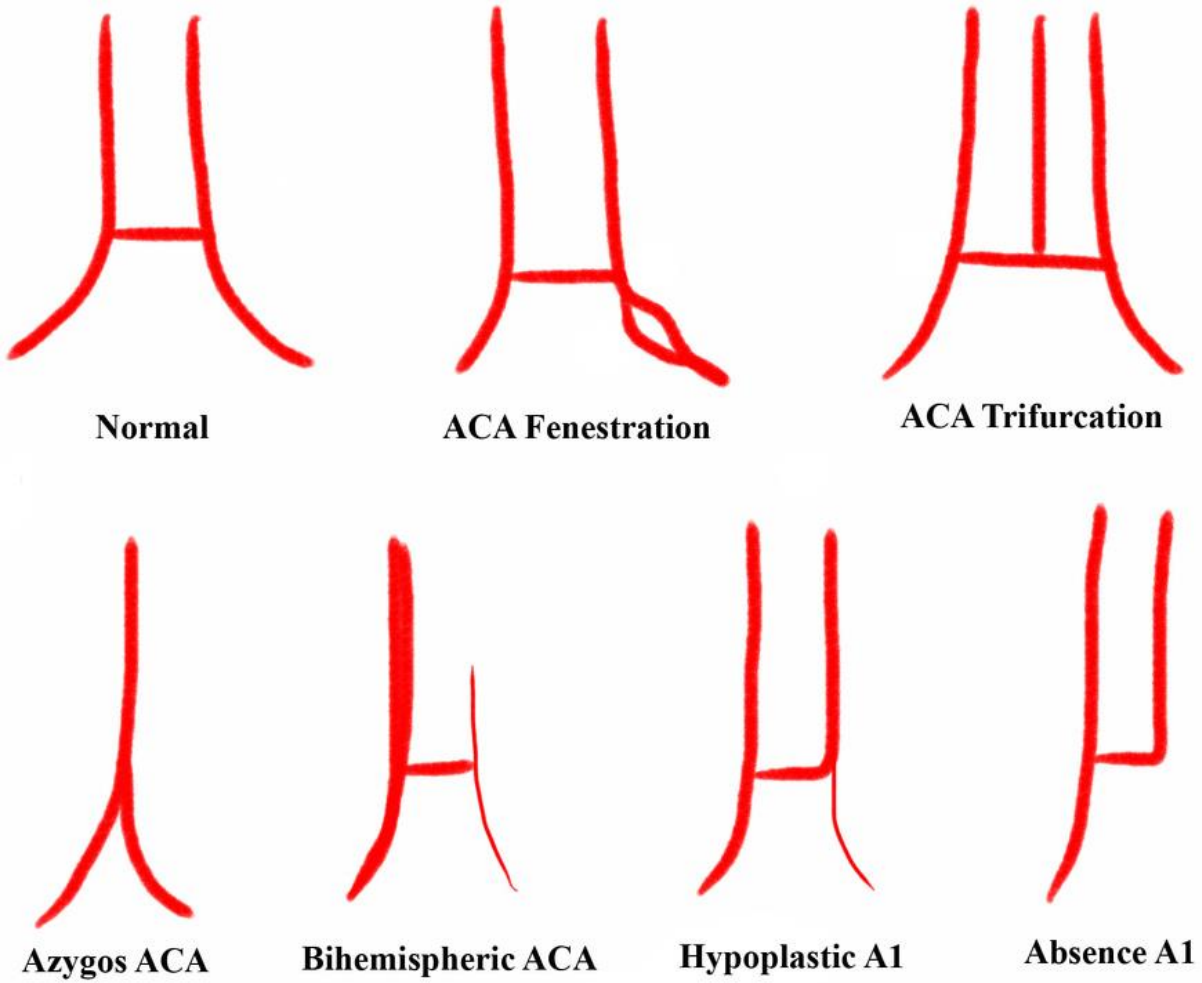




Anterior cerebral artery hypoplasia = hyperplasia contralateral artery = ⇑ risk aneurysms at that level

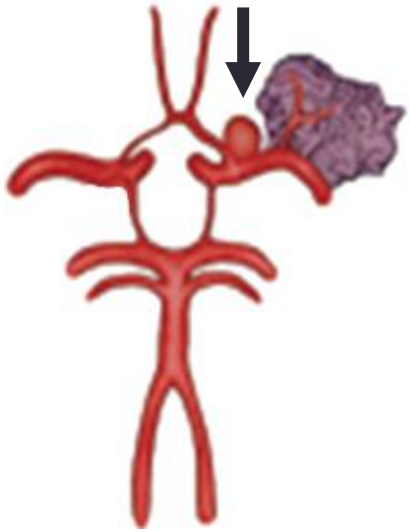
Brain aneurysm formation: haemodynamic factors (3)

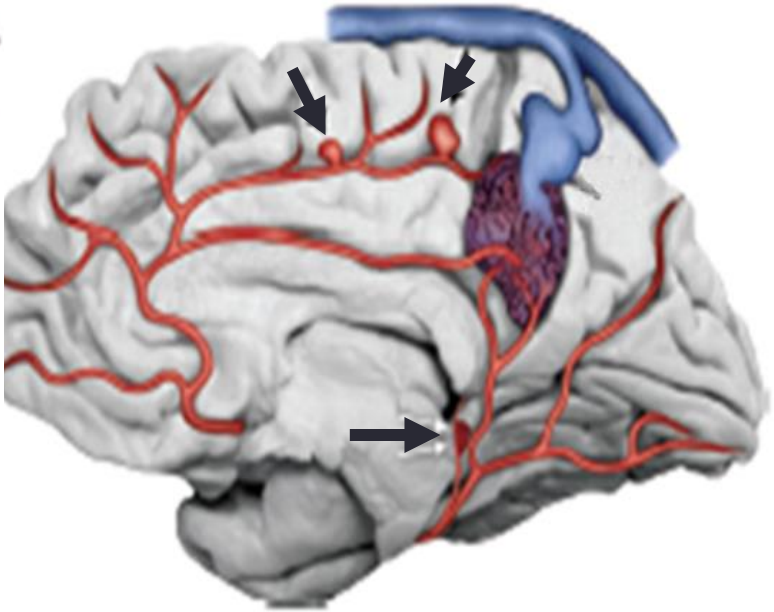
- The anatomical variations are multiple, but all increase the chance of aneurysm formation

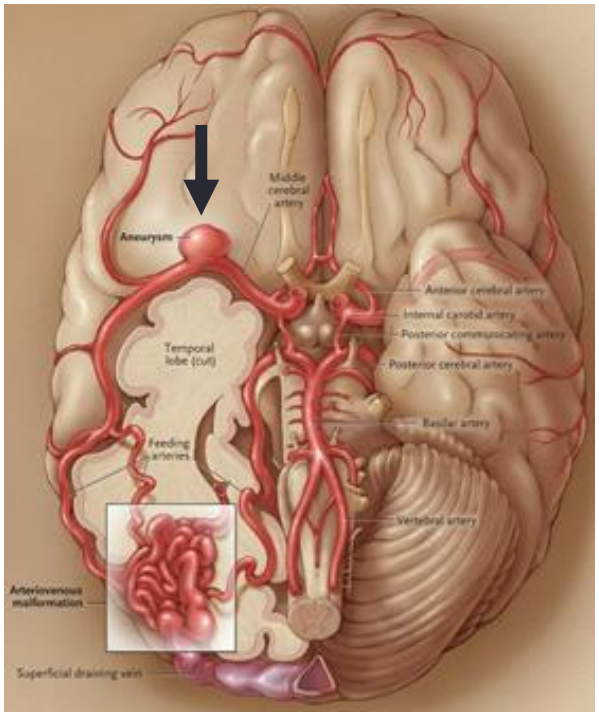


Brain aneurysm: association with cerebral arteriovenous malformations

- Due to increased blood flow caused by the arteriovenous malformation



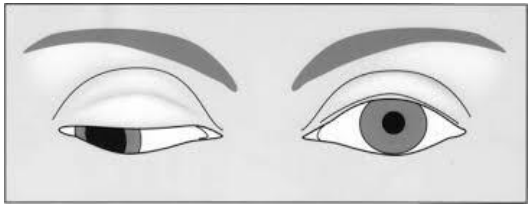




Brain aneurysms: symptoms BEFORE bleeding

REMEMBER?

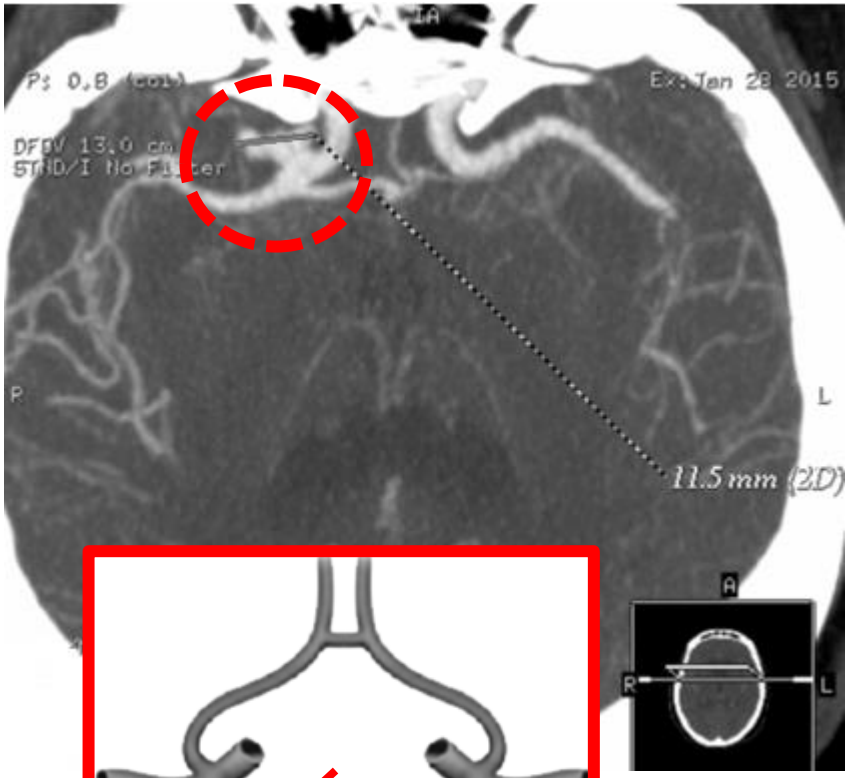
- Non-specific headache
- Incidental finding on CT, MRI, or cerebral angiography done for other reasons
- Cranial nerve deficits
  - third nerve palsy
  - sixth nerve palsy

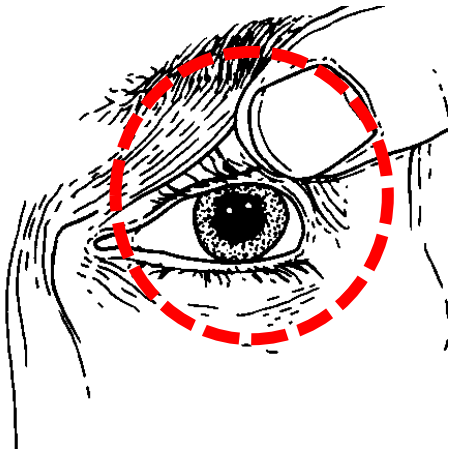


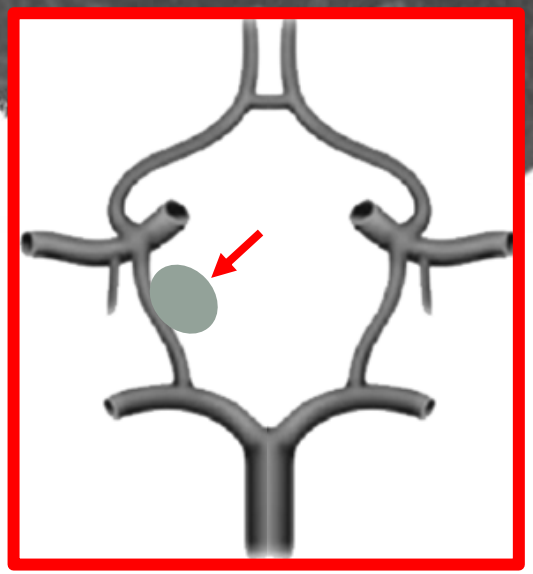
third nerve palsy

sixth nerve palsy

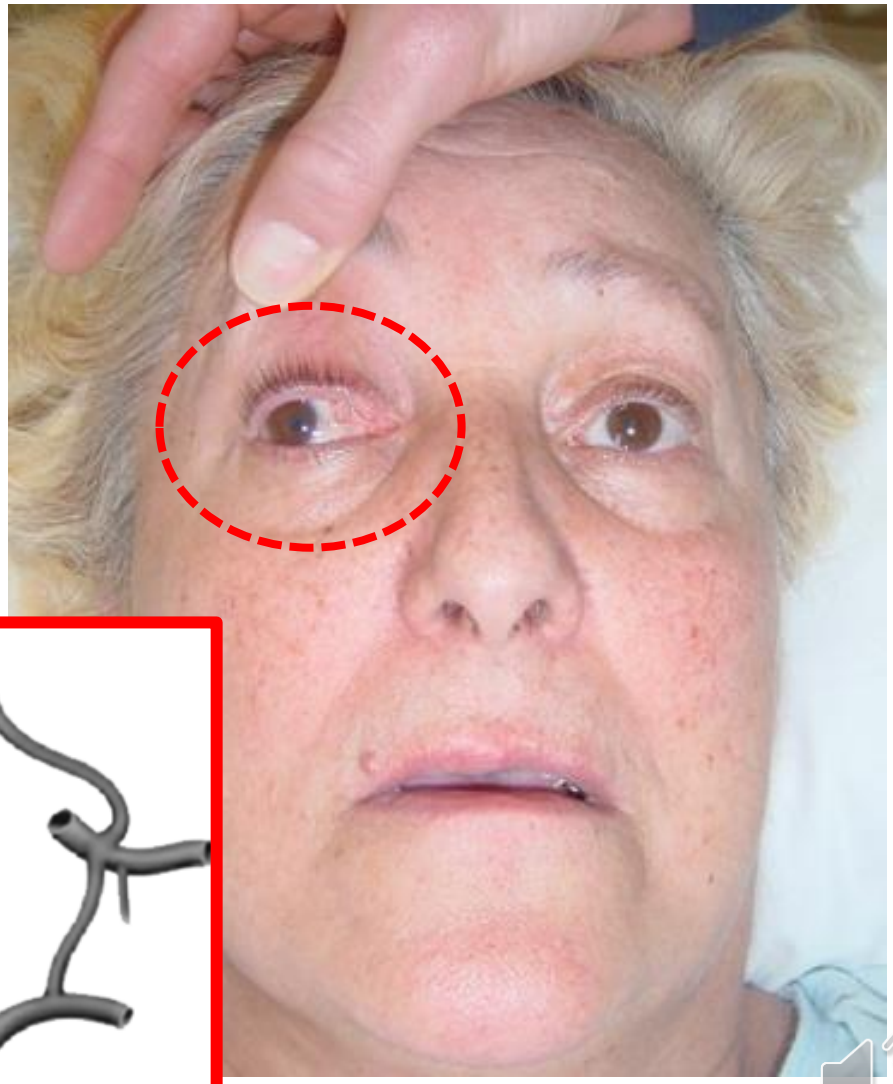
Third nerve palsy in posterior communicating artery aneurysm

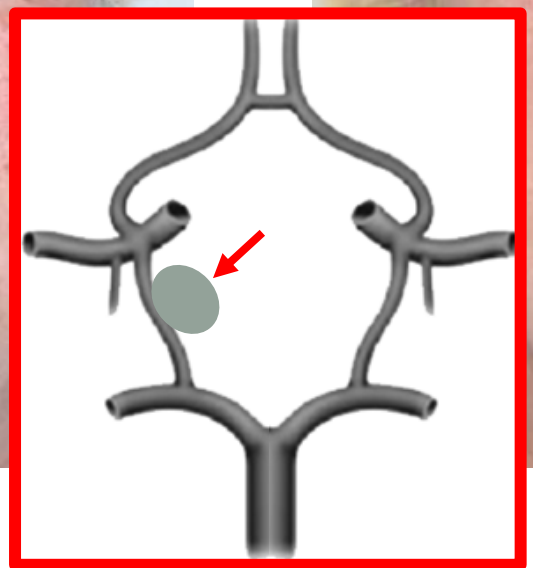




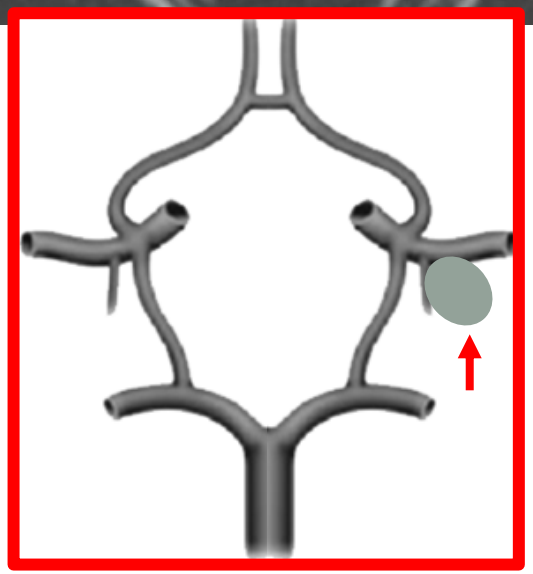


Third nerve palsy in posterior communicating artery aneurysm

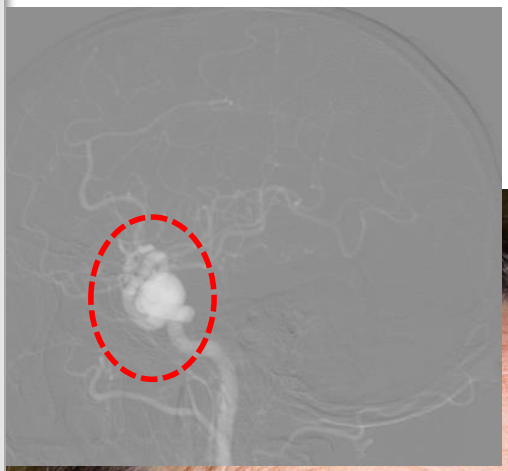


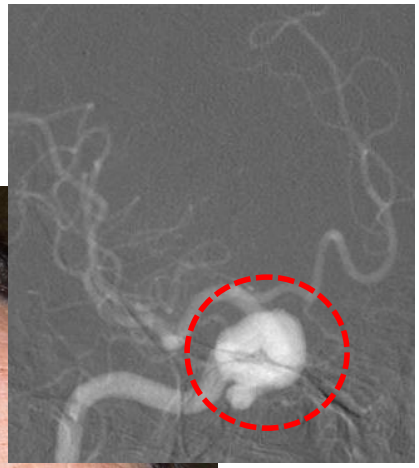


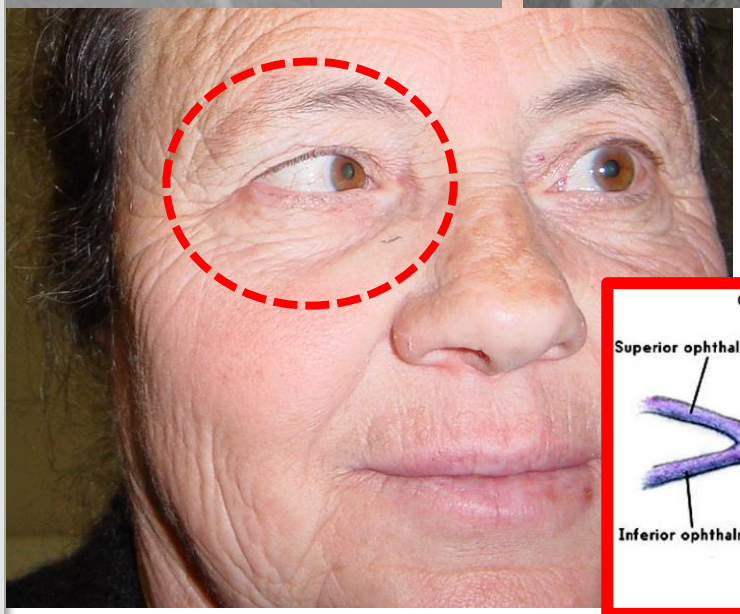
Third nerve palsy in carotid-ophthalmic artery aneurysm

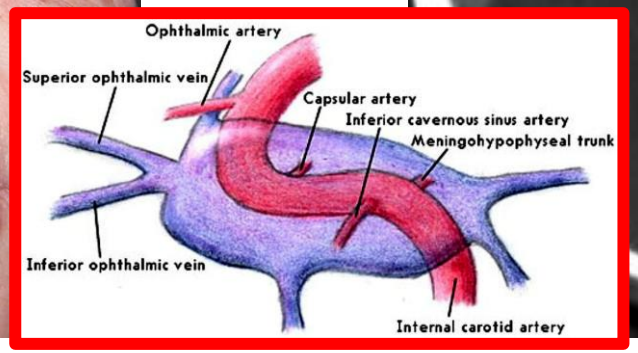


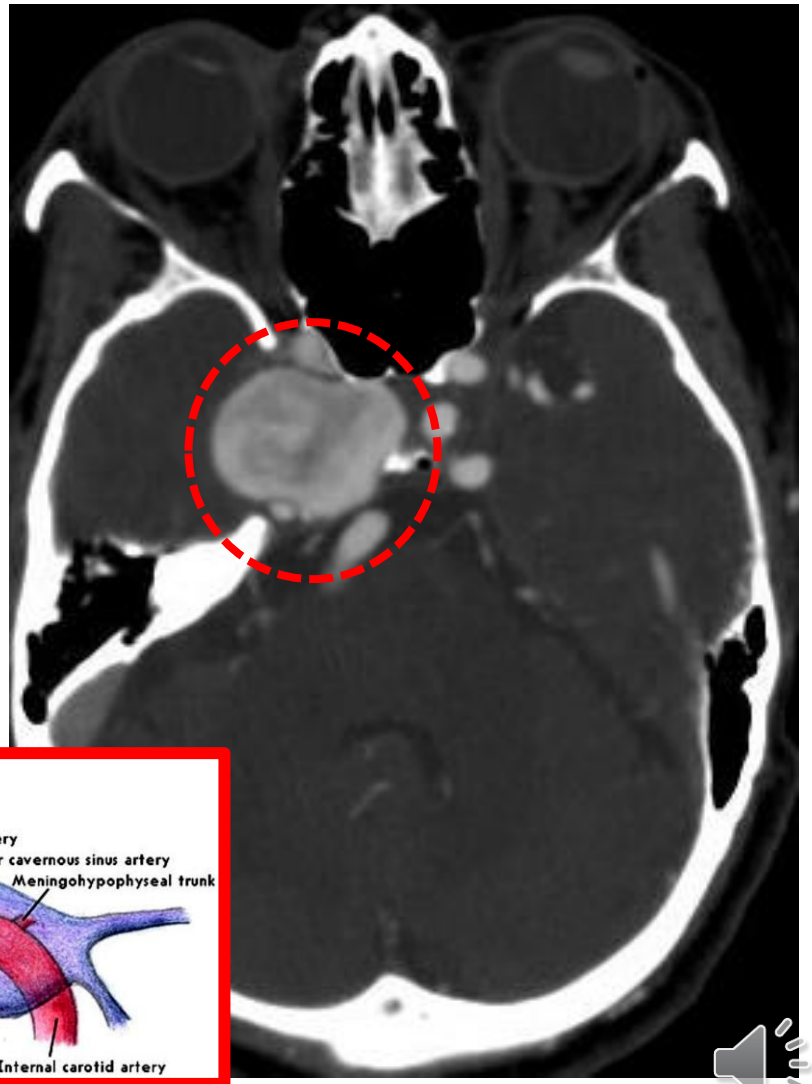
Sixth nerve palsy in cavernous sinus carotid artery aneurysm











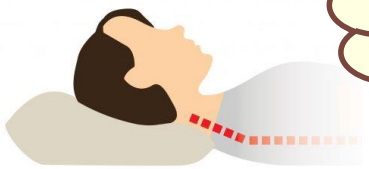
DIAGNOSIS SUBARACHNOID HAEMORRHAGE + CEREBRAL ANEURYSMS

Clinical symptoms

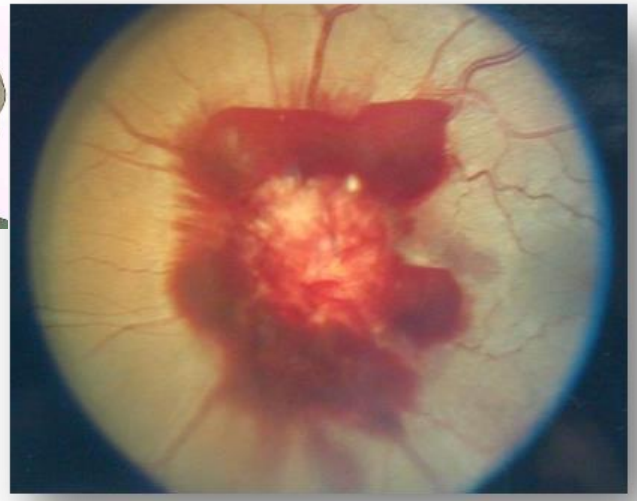
CT scan

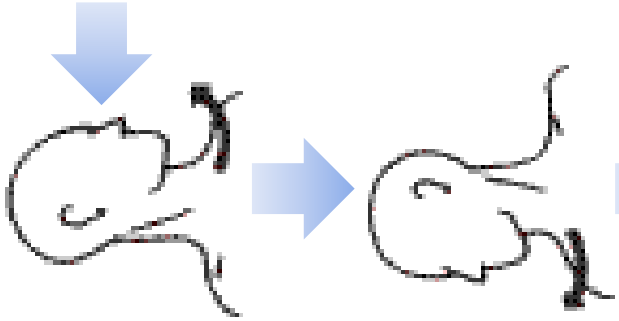
Angiogram

Clinical examination upon admission



REMEMBER?





Rule out cervical spine fracture

Neck stiffness

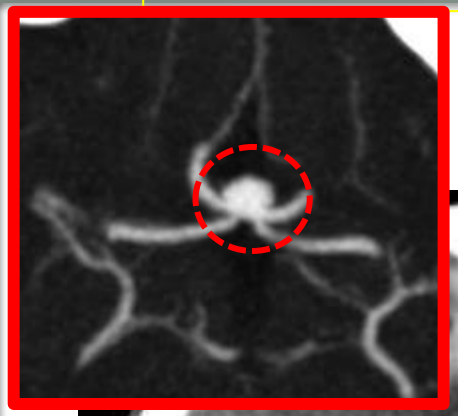
Vitreous haemorrhage
fundoscopic examination

Brudzinski's Sign

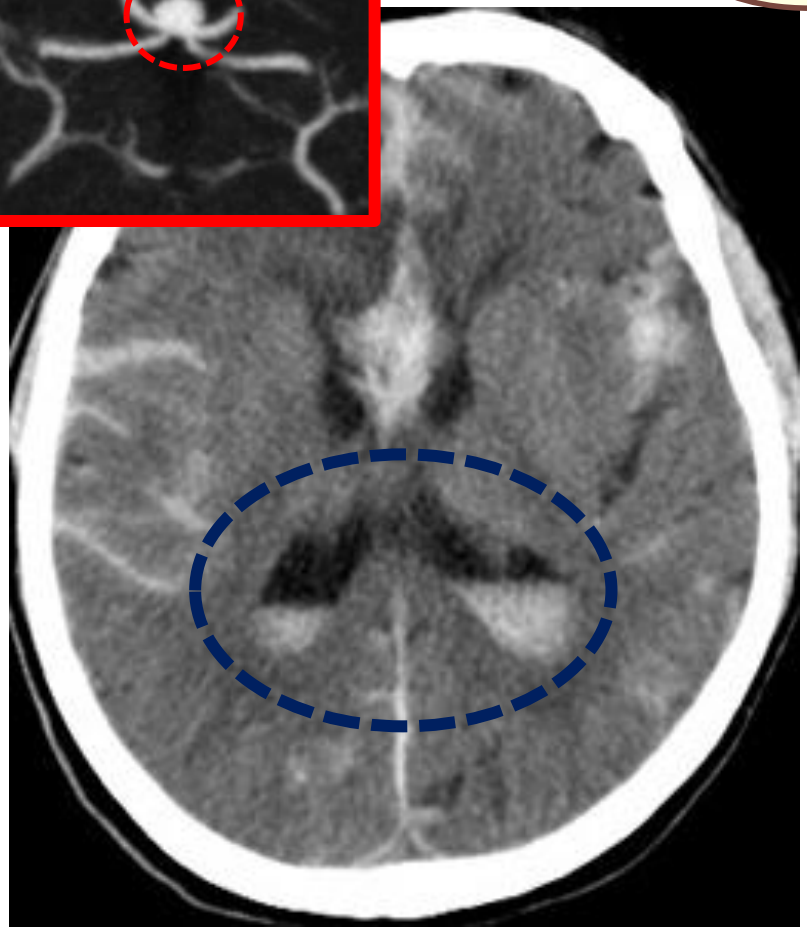
Kernig's Sign

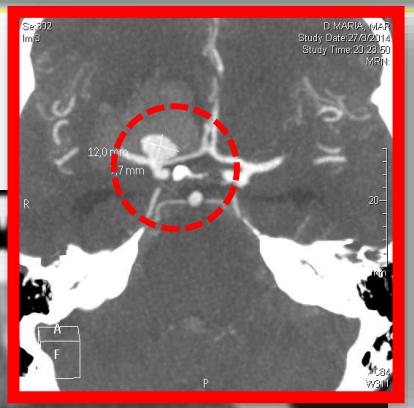
Subarachnoid haemorrhage diagnosis: brain CT scan

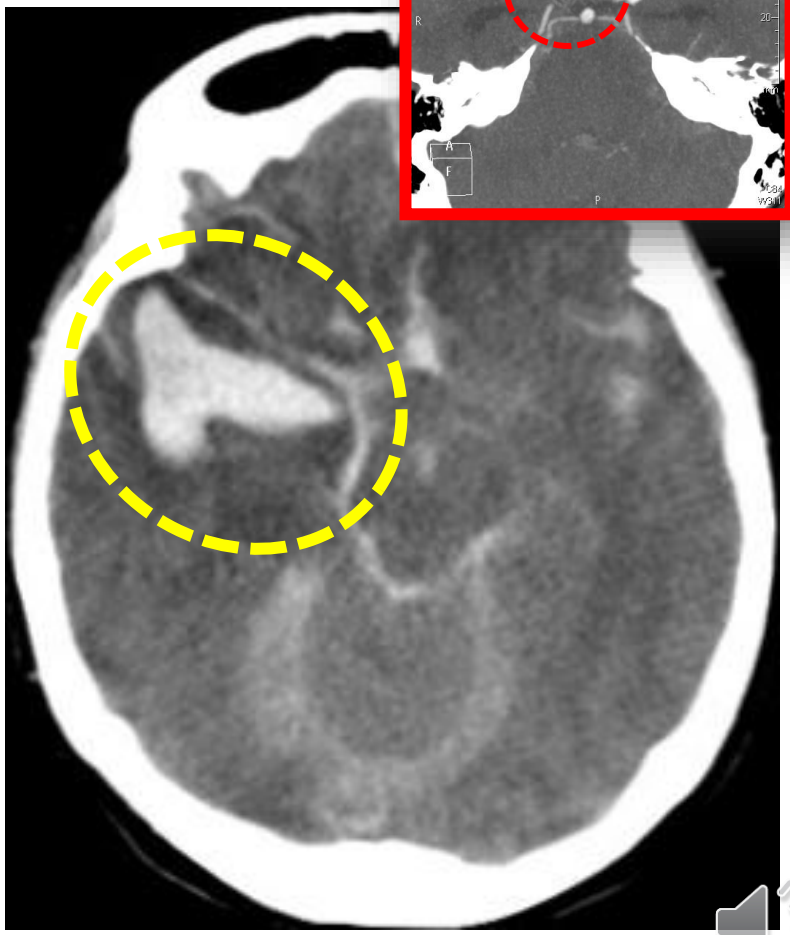
Brain CT scan: subarachnoid haemorrhage + intracerebral and / or intraventricular haematoma



REMEMBER?







Intraventricular

Intracerebral

Lumbar puncture?

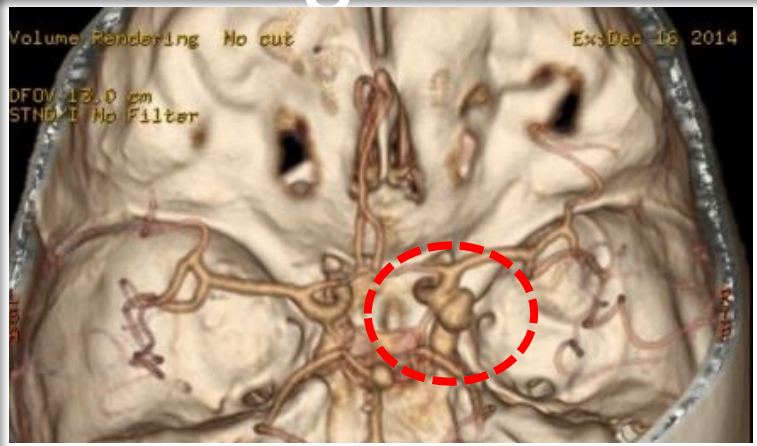
REMEMBER?

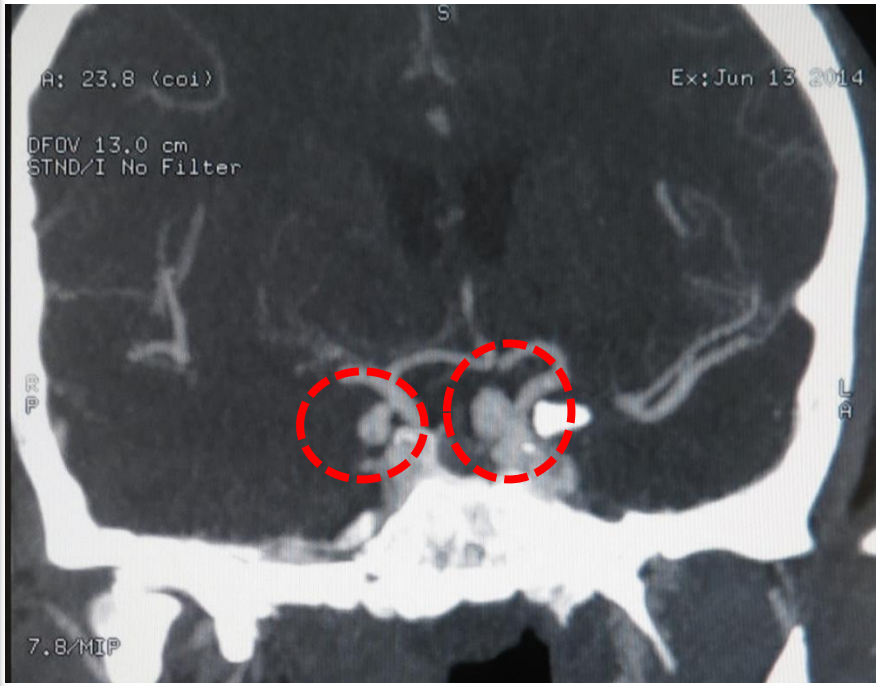
- **ONLY** if negative CT scan + clinical suspicion of subarachnoid hemorrhage
- Risk cerebellar herniation → cardiorespiratory arrest
- Search for xanthochromia

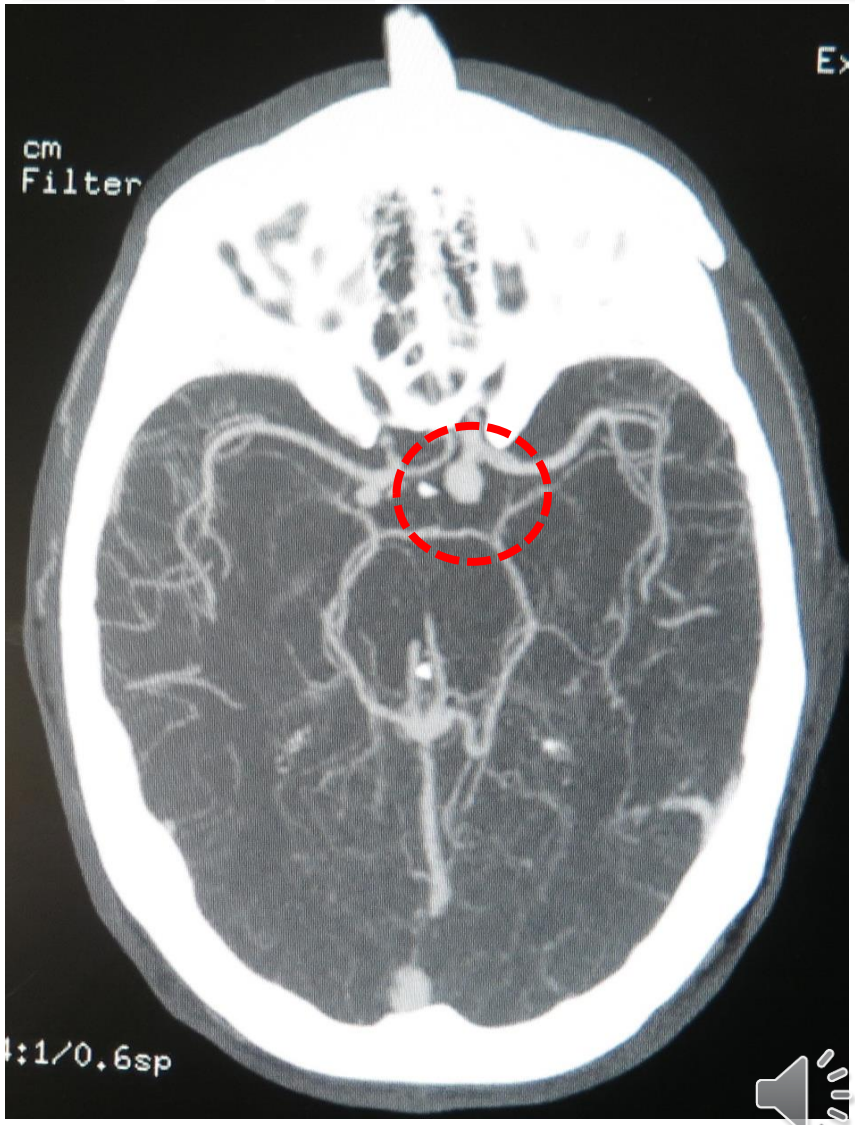
Normal CSF ⇒

⇐ Xanthochromic CSF

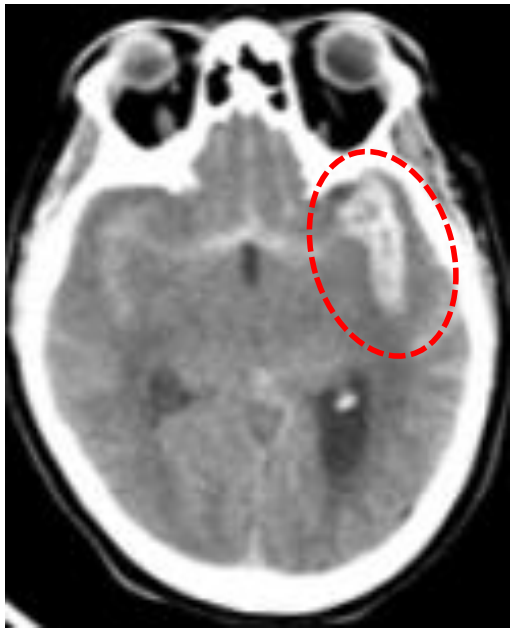
Ruptured aneurysm emergency diagnosis: CT angiography







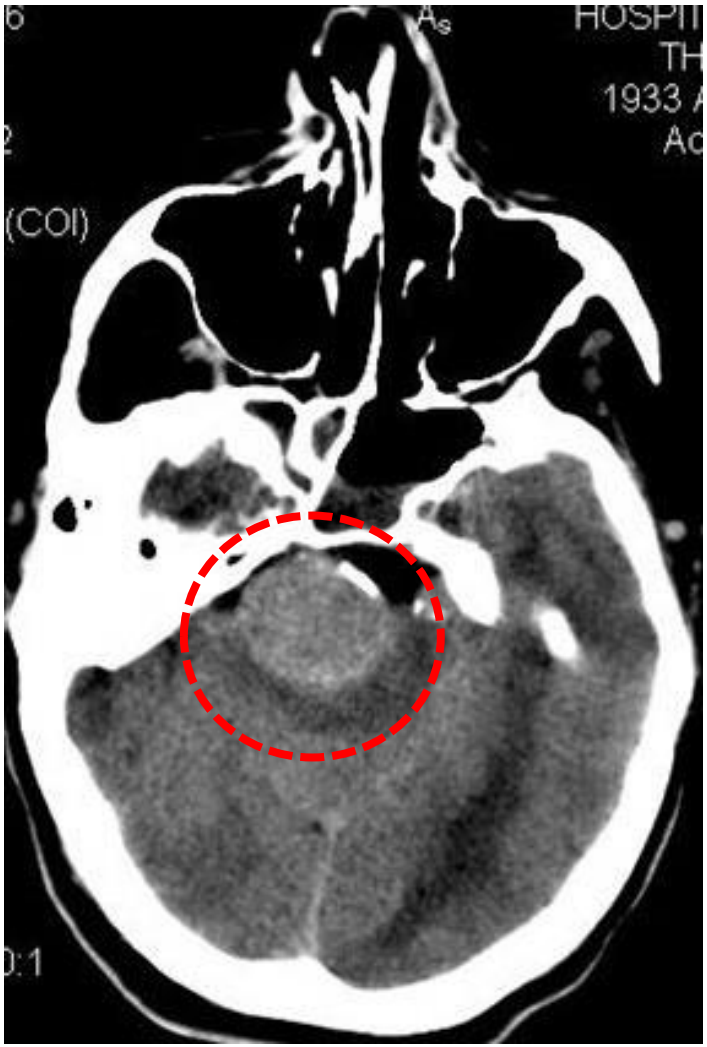
Multiple aneurysms: which one has bled?

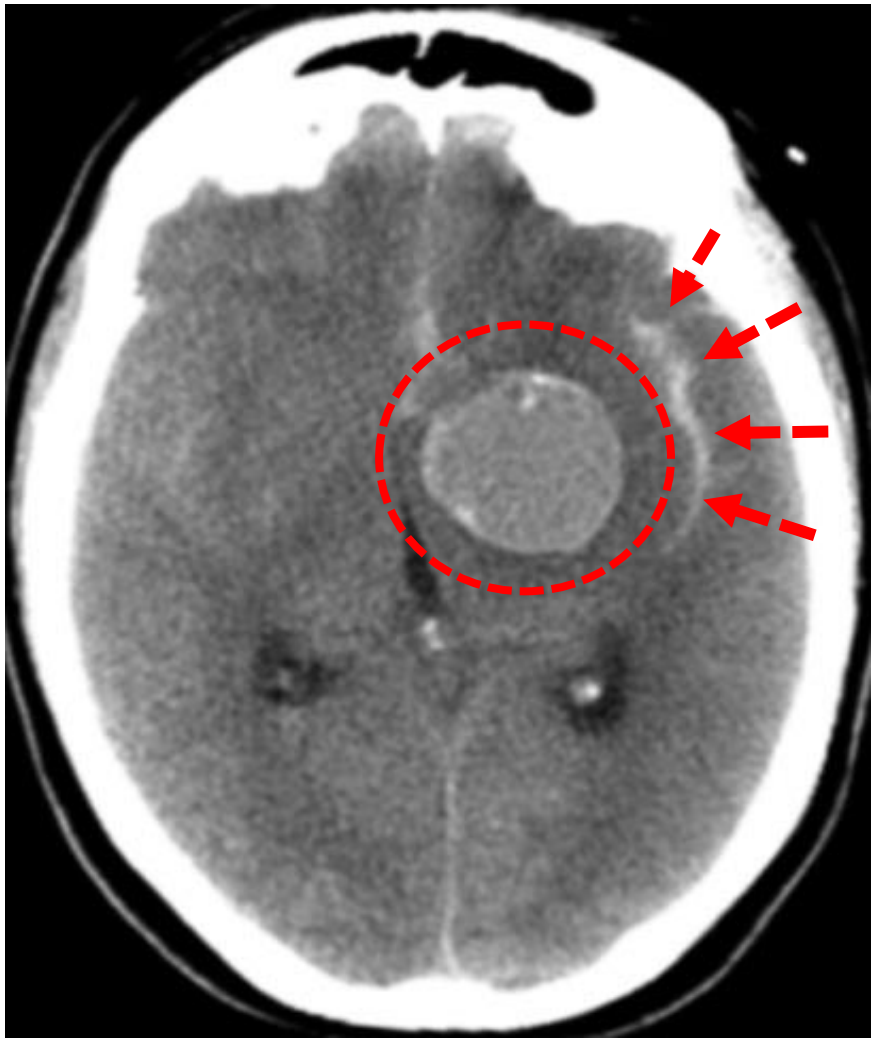


Suspicion according to:
Blood distribution in CT scan
Larger aneurysm
Irregular aneurysm, bleb
By location: aneurysm ant. com / pericallosal art.

CT scan: incidental aneurysm finding

- Often giant & calcified



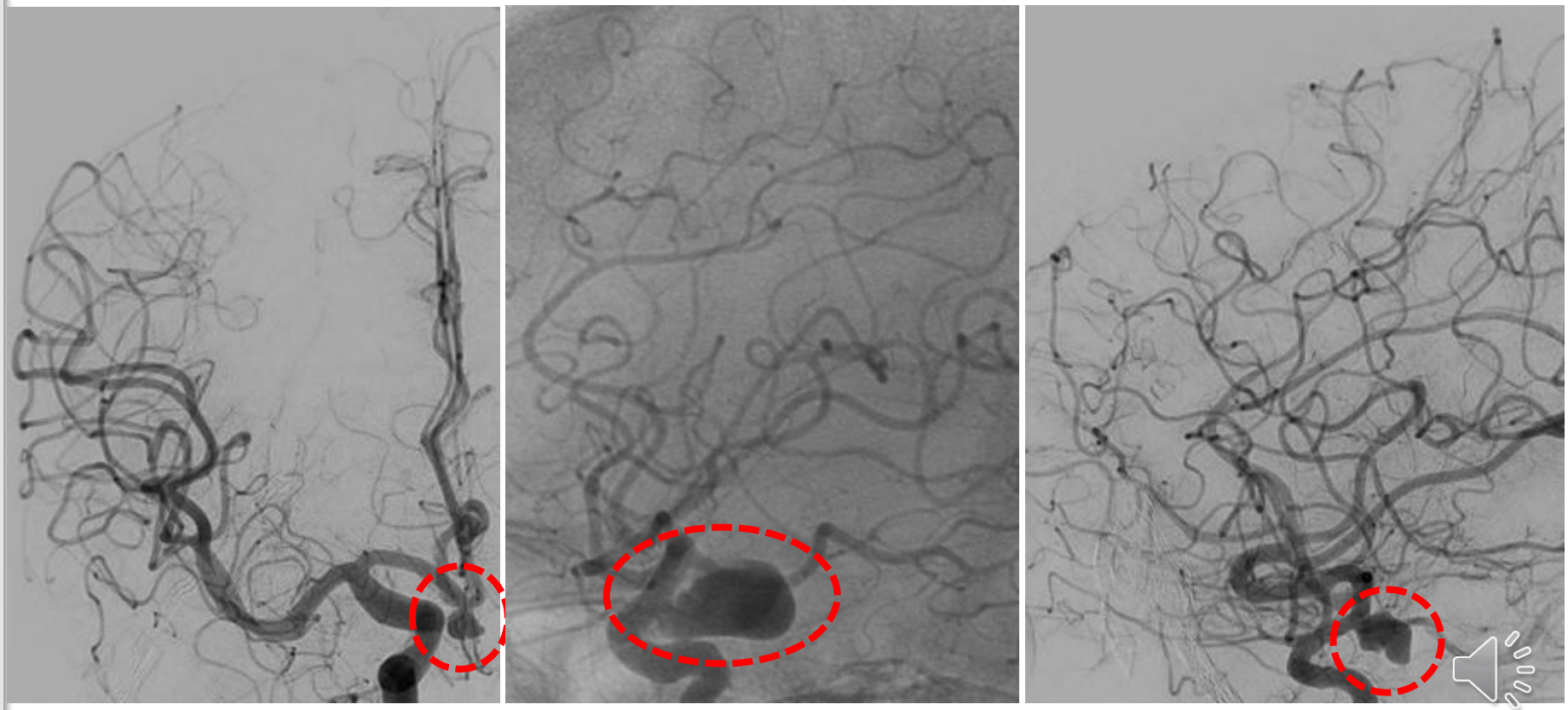


MRI: incidental aneurysm finding

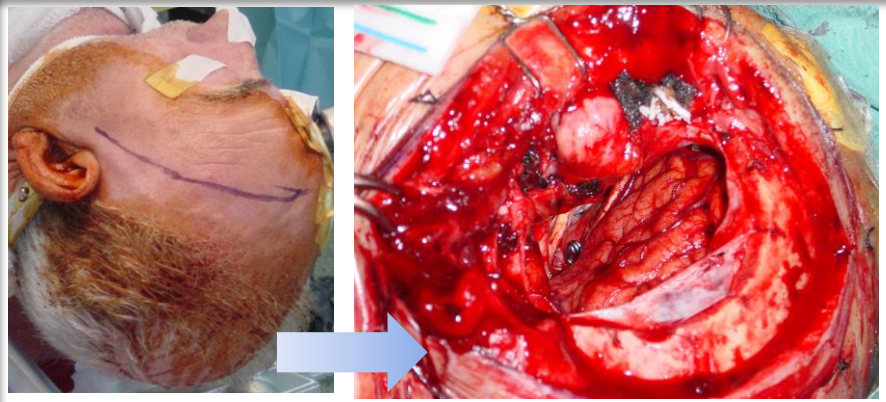
What to do?

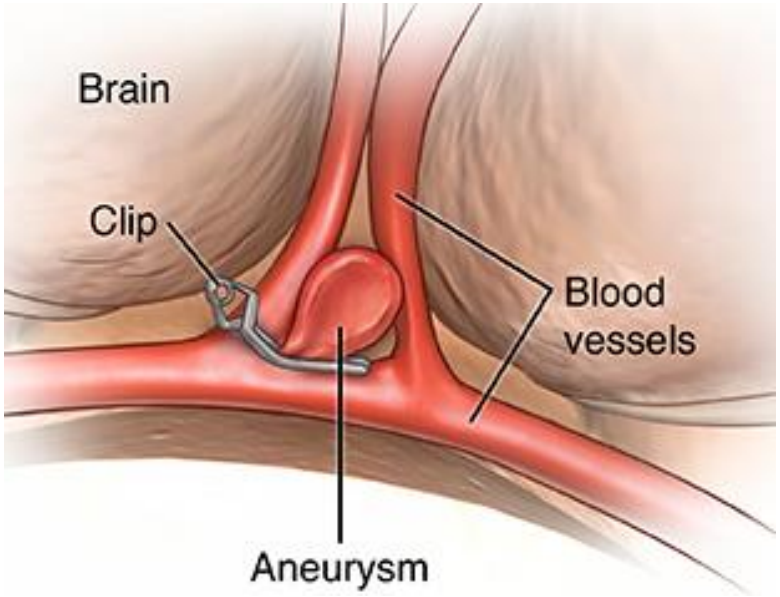
Aneurysm diagnosis + vascular tree imaging: cerebral angiography

- Best diagnostic technique
- Only available in a few hospitals on emergency basis
  - CT angiography best second choice

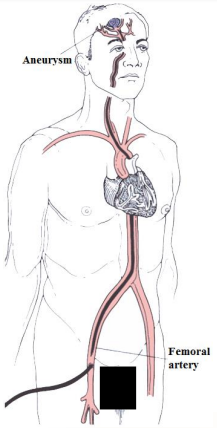


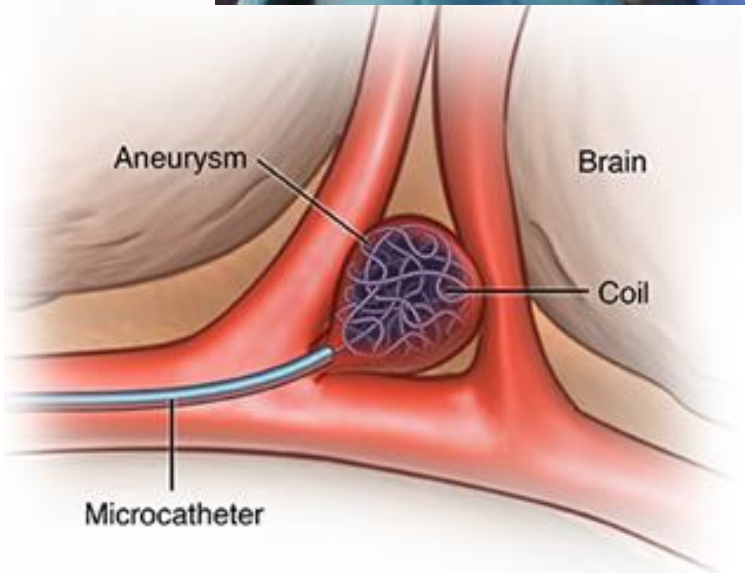
BRAIN ANEURYSM: TREATMENT





Surgical





Endovascular

Subarachnoid hemorrhage: scales

Hunt & Hess

Grade	Signs and symptoms
1	Asymptomatic or minimal headache and slight neck stiffness
2	Moderate to severe headache; neck stiffness; no neurologic deficit except cranial nerve palsy
3	Drowsy; minimal neurologic deficit
4	Stuporous; moderate to severe hemiparesis; possibly early decerebrate rigidity and vegetative disturbances
5	Deep coma; decerebrate rigidity; moribund

WFNS SAH

Grade	GCS	Focal neurological deficit
1	15	Absent
2	13–14	Absent
3	13–14	Present
4	7–12	Present or absent
5	<7	Present or absent

Fisher

Grade	Appearance of hemorrhage
1	None evident
2	Less than 1 mm thick
3	More than 1 mm thick
4	Diffuse or none with intraventricular hemorrhage or parenchymal extension

Subarachnoid hemorrhage: Hunt and Hess scale (associated with mortality)

- 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)

Subarachnoid hemorrhage: WFNS scale

WFNS SAH Grade		
WFNS Grade	**GCS Score**	**Major Focal Deficit**
0**		
1	15	-
2	13-14	-
3	13-14	+
4	7-12	+ or -
5	3-6	+ or -

*aphasia, hemiparesis or hemiplegia
** intact anseurysm

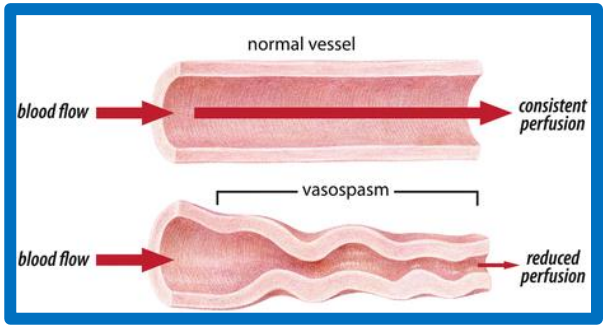
Prognosis: WFNS / Hunt & Hess scales

World Federation of Neurosurgical Societies Grading Scale	Grade	Hunt and Hess	Glasgow Coma Scale	Survival
No motor deficit	I	Asymptomatic or minimal headache and slight nuchal rigidity	15	70%
No motor deficit	II	Moderate to severe headache, nuchal rigidity, no neurological deficit other than cranial nerve palsy	13–14	60%
Motor deficit	III	Drowsiness, confusion, or mild focal deficit	13–14	50%
With or without motor deficit	IV	Stupor, moderate to severe hemiparesis, possibly early decerebrate posturing	7–12	20%
With or without motor deficit	V	Deep coma, decerebrate posturing, moribund appearance	3–6	10%

From Patel, V. N., & Samuels, O. B. (2012). The critical care management of aneurysmal subarachnoid hemorrhage. In Y. Murai (Ed.), *Aneurysm*. InTech. doi:10.5772/48474

Subarachnoid hemorrhage: Fisher scale

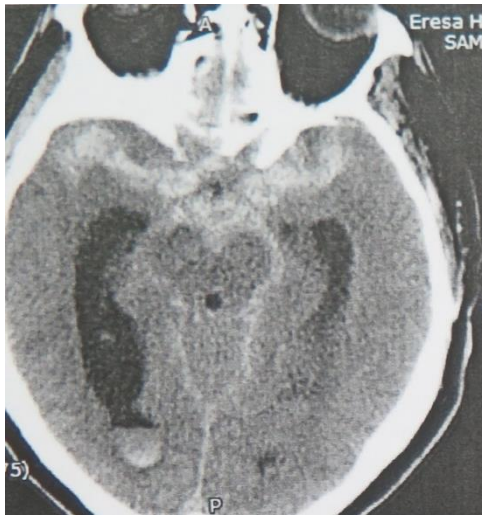
- Associated with vasospasm risk

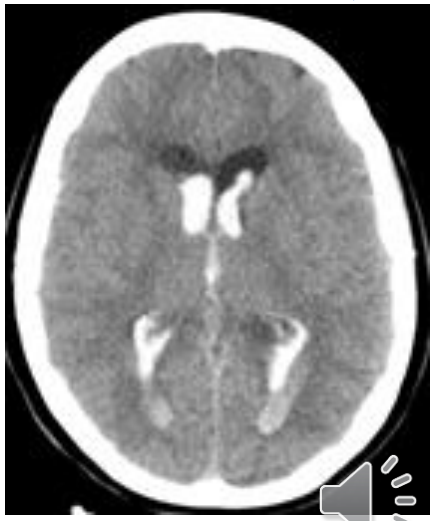


Grade	Descriptions
Fisher I	No blood detected
Fisher II	Diffuse deposition or thin layer with all vertical layers of blood (interhemispheric fissure, insular cistern, ambient cistern) < 1 mm thick
Fisher III	Localized clots and/or vertical layers of blood ≥ 1 mm in thickness
Fisher IV	Diffuse or no subarachnoid blood, but with intracerebral or intraventricular clots

II

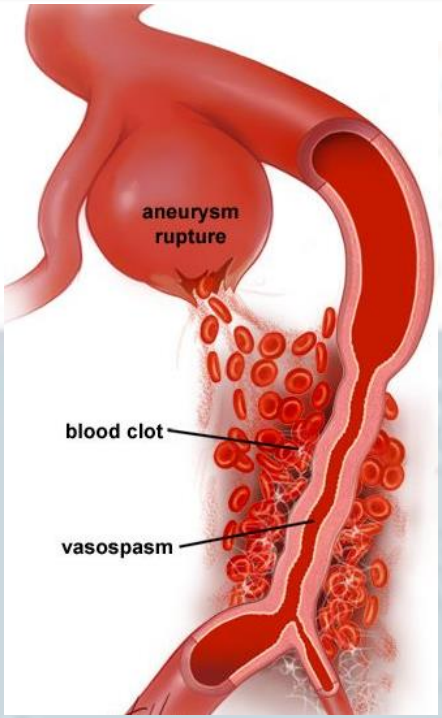
III





IV

Prognosis: Fisher scale



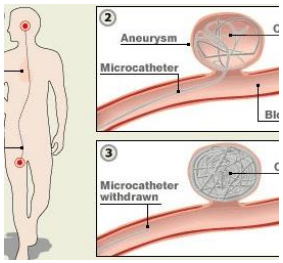
Grade	Fisher Scale	% with Symptomatic Vasospasm
1	Focal thin	21%
2	Diffuse thin SAH	25%
3	Thick SAH present	37%
4	Focal or diffuse thin SAH with significant ICH or IVH	31%

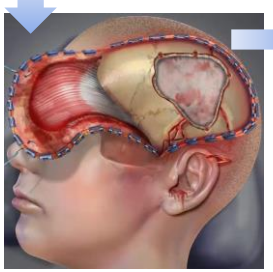
From Claassen, J., Bernardini, G. L., Kreiter, K., Bates, J., Du, Y. E., Copeland, D., . . . Mayer, S. A. (2001). Effect of cisternal and ventricular blood on risk of delayed cerebral ischemia after subarachnoid hemorrhage: The Fisher Scale revisited. *Stroke, 32*, 2012–2020.

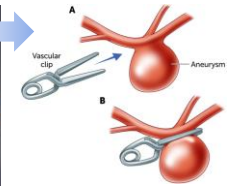
BRAIN ANEURISMS: TREATMENT

- 1st treatment **subarachnoid hemorrhage**: ICU

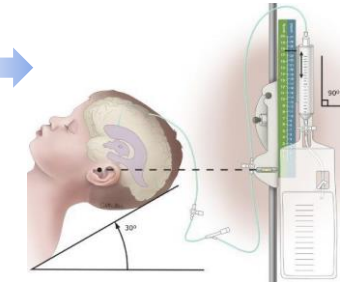
- 2nd treatment **aneurysm**
  - Endovascular treatment
  - Craniotomy + clip



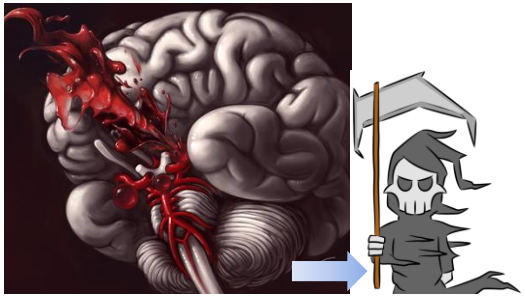


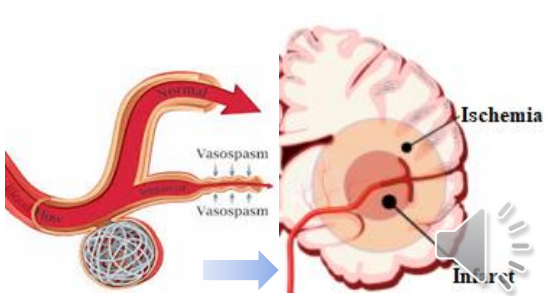


- 3rd treatment complications
  - **Hydrocephalus**: external ventricular drainage?
  - Drainage i**ntracerebral hematoma**?



- 4th **prevent** complications
  - **Rebleeding**
    - Kills 20% of patients
  - Control of **vasospasm**
    - Kills 7%
    - Neurological deficits 7%

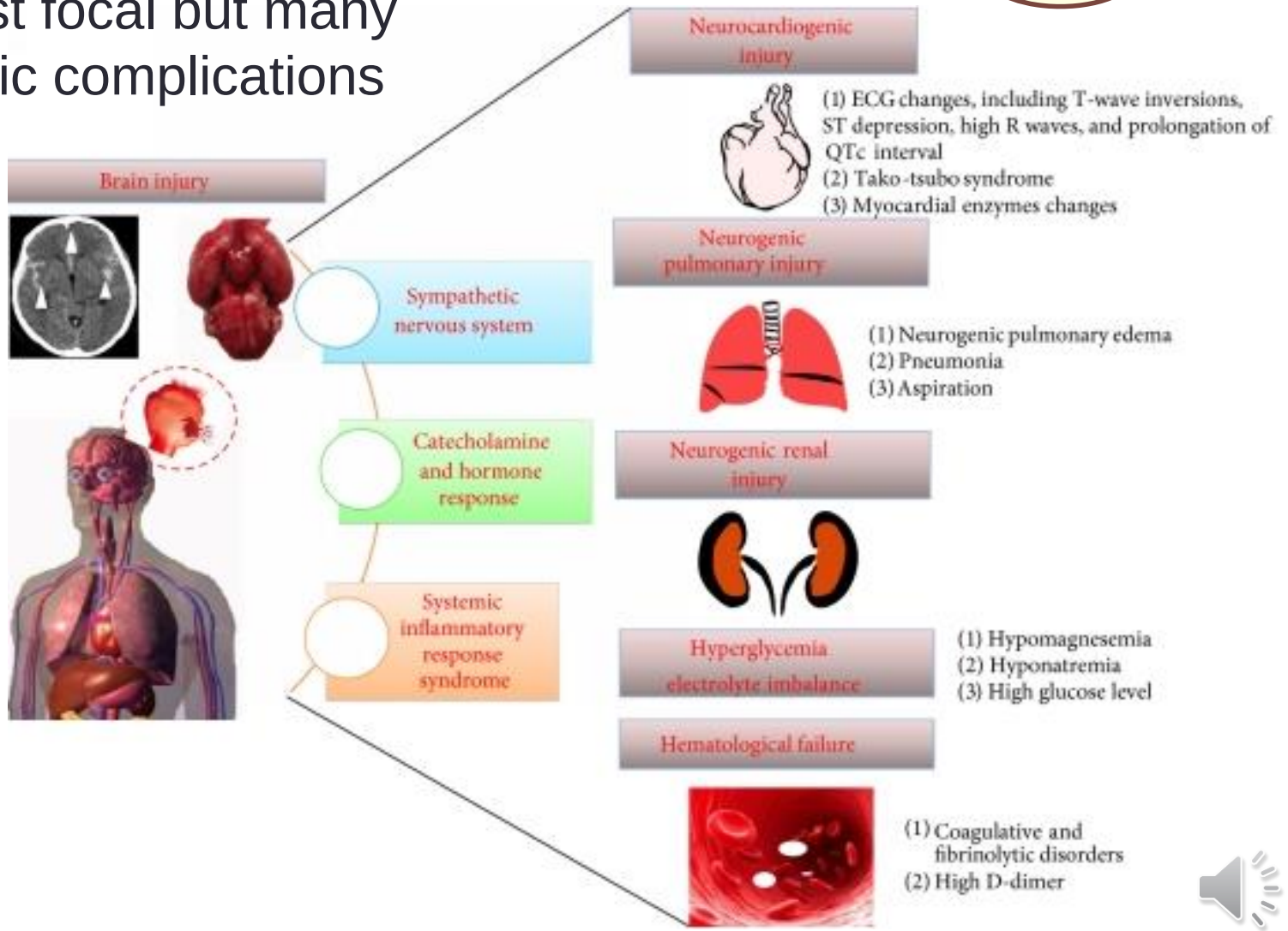




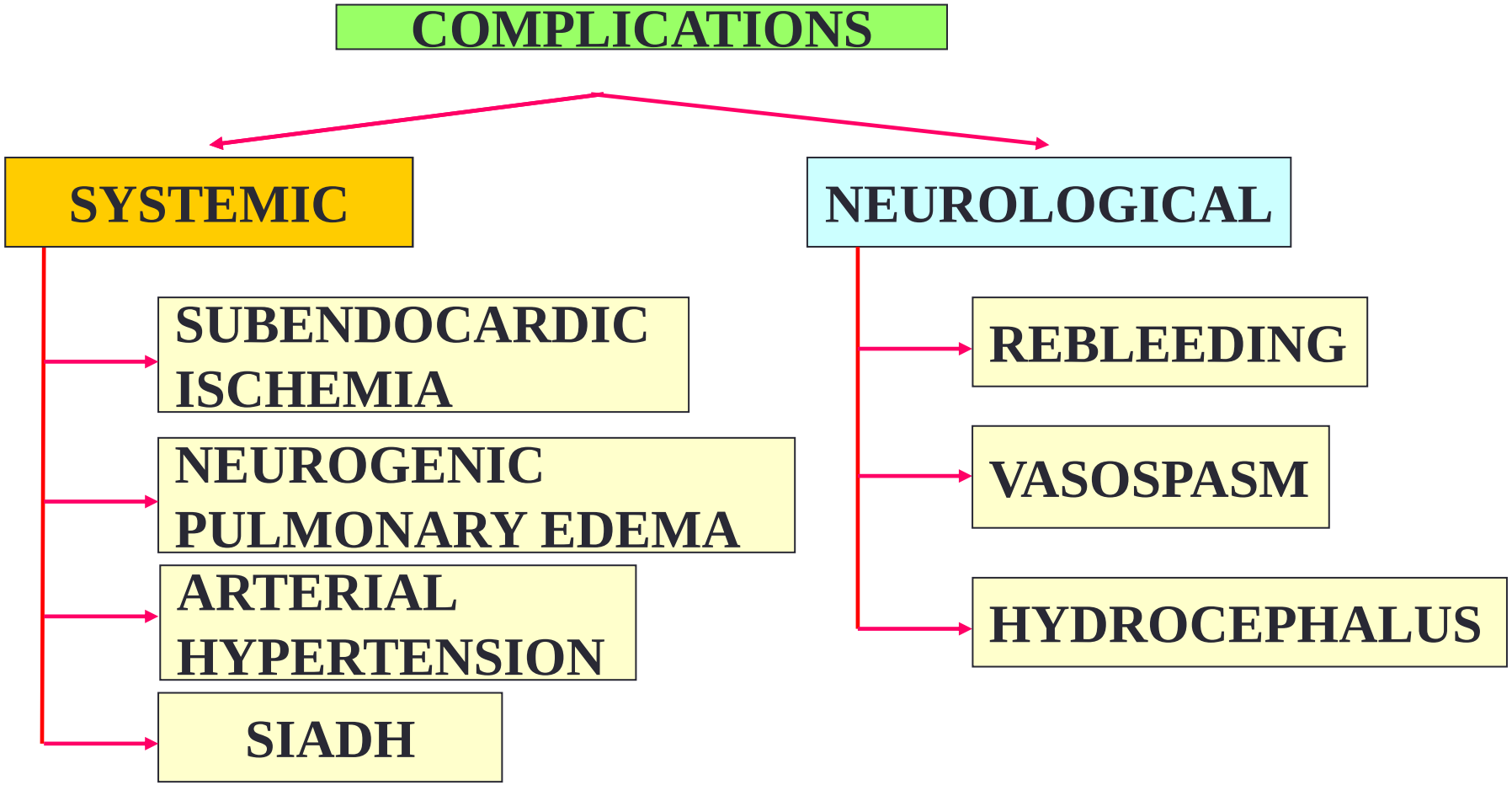
Subarachnoid haemorrhage: complications (1)

REMEMBER?

- Not just focal but many systemic complications

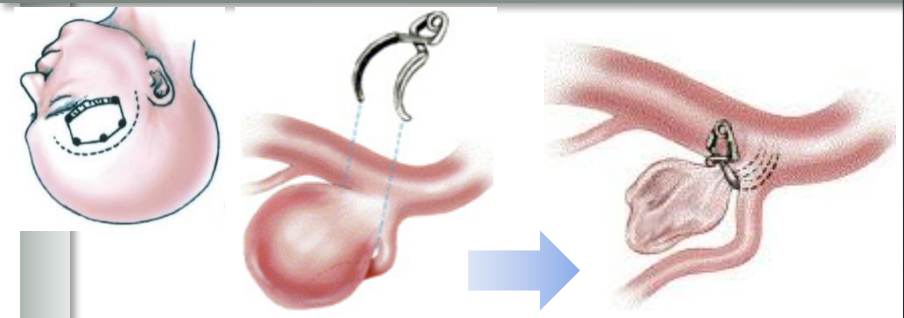


Subarachnoid haemorrhage: complications (2)



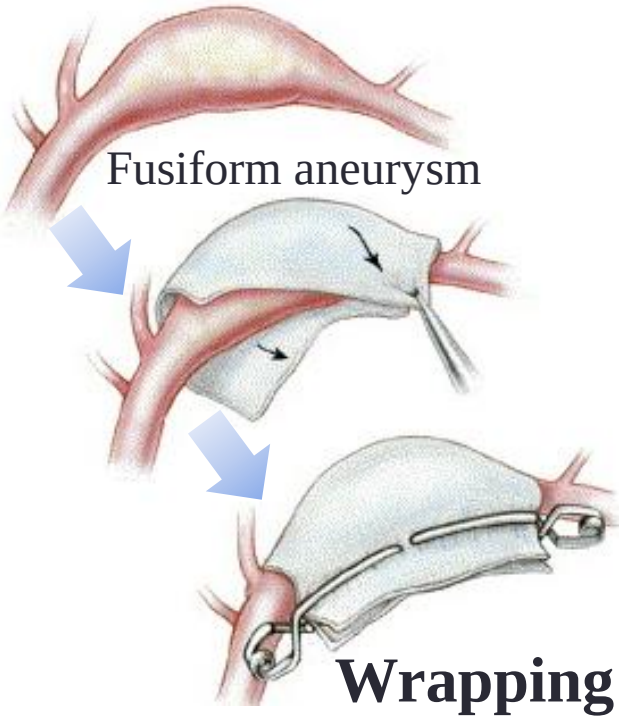
SIADH = Syndrome inadequate antidiuretic hormone secretion

Surgical treatment



Saccular aneurysm

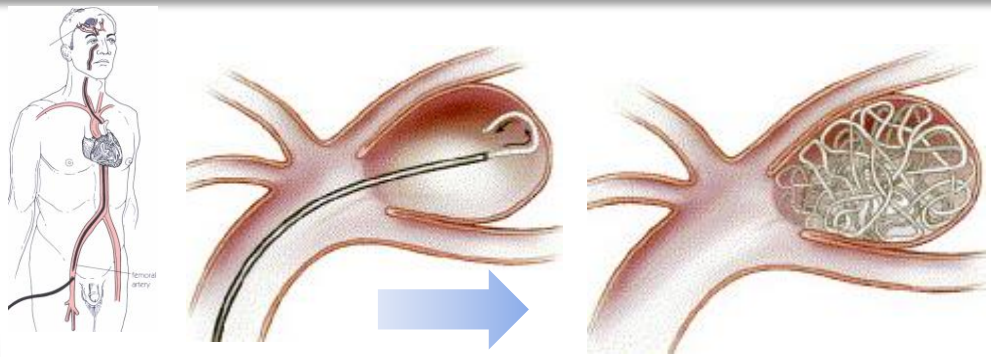
Clipping



Fusiform aneurysm

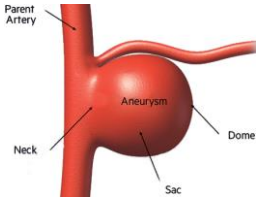
Wrapping

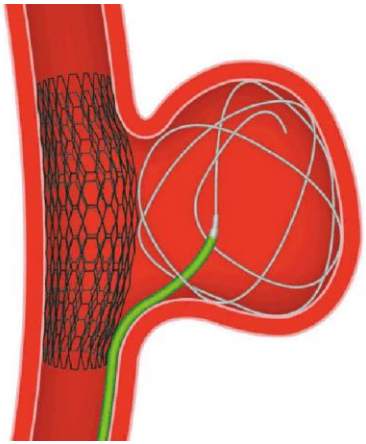
Endovascular treatment



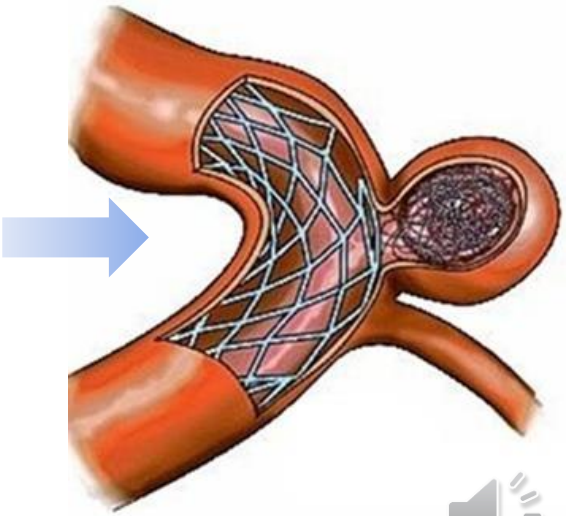
Narrow neck aneurysm

Coiling





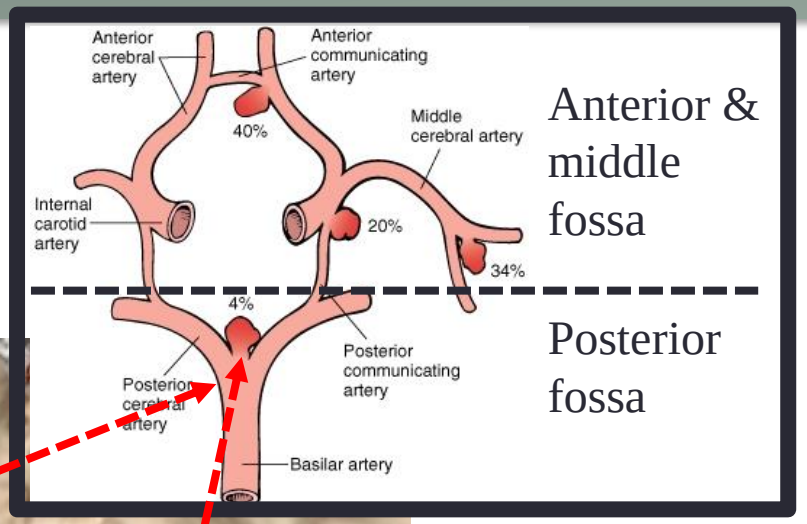
Wide neck aneurysm

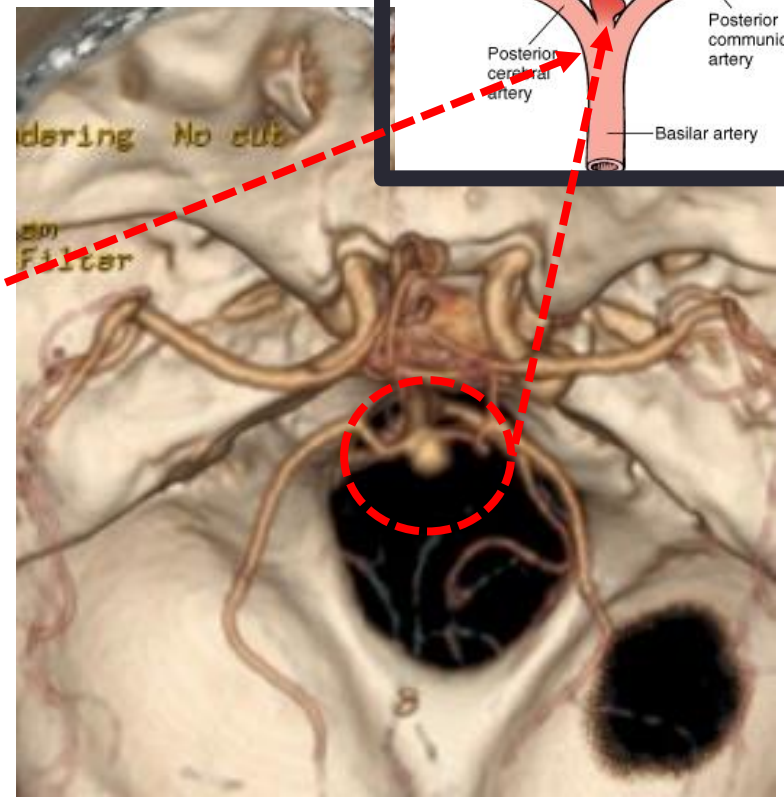


Stent + coiling

Treatment modality to recommend location

- Posterior fossa = endovascular
- Anterior and middle fossa depends on each case





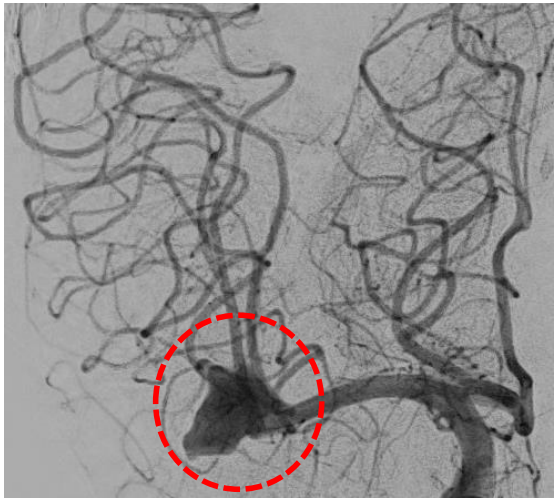
Endovascular ← treatment

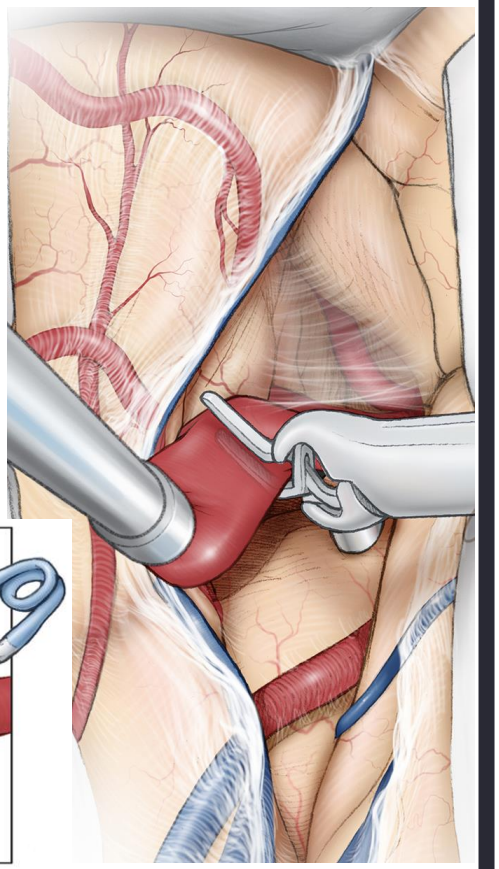
Superior cerebellar art. aneurysm

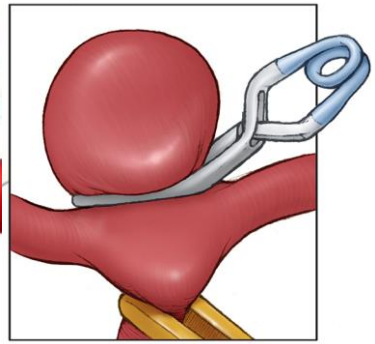
Basilar apex art. aneurysm

Treatment modality to recommend neck size

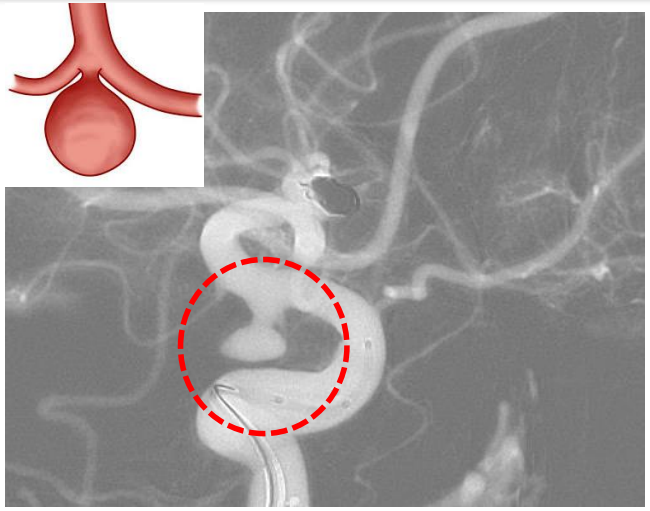
- Narrow neck = endovascular
- Wide neck ≅ surgical

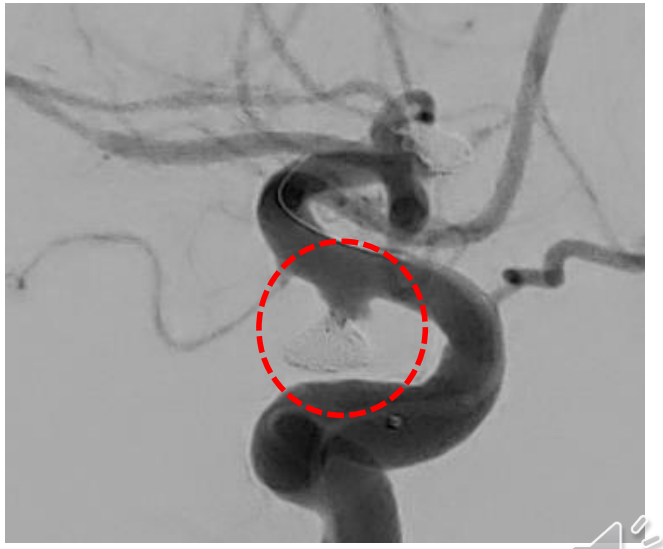






Surgery

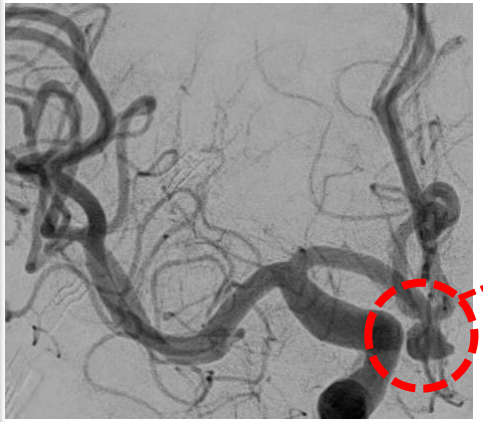


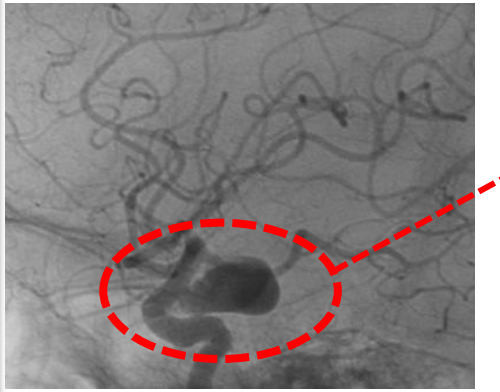


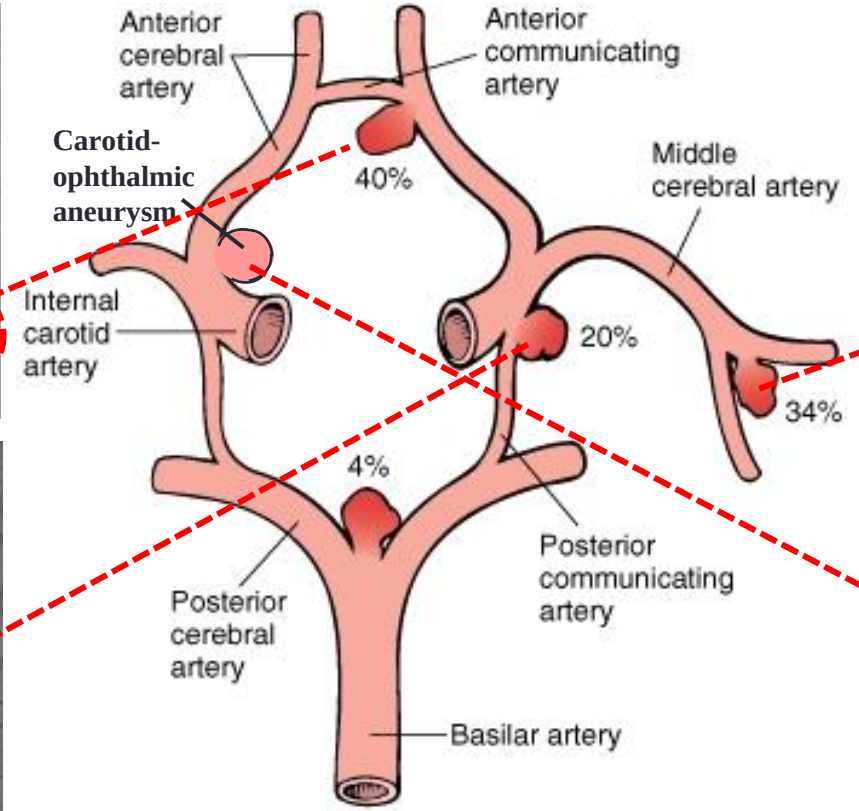
Endovascular

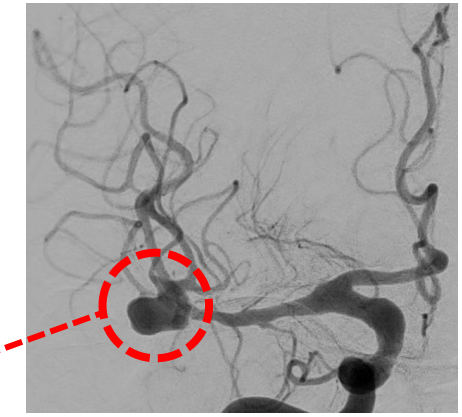
Treatment modality to recommend parent vessel

- Endovascular ≅ ant. com., post. com.
- Surgical ≅ carotid-ophthalmic, middle cerebral artery







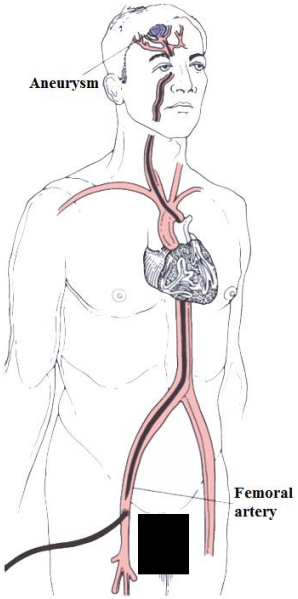


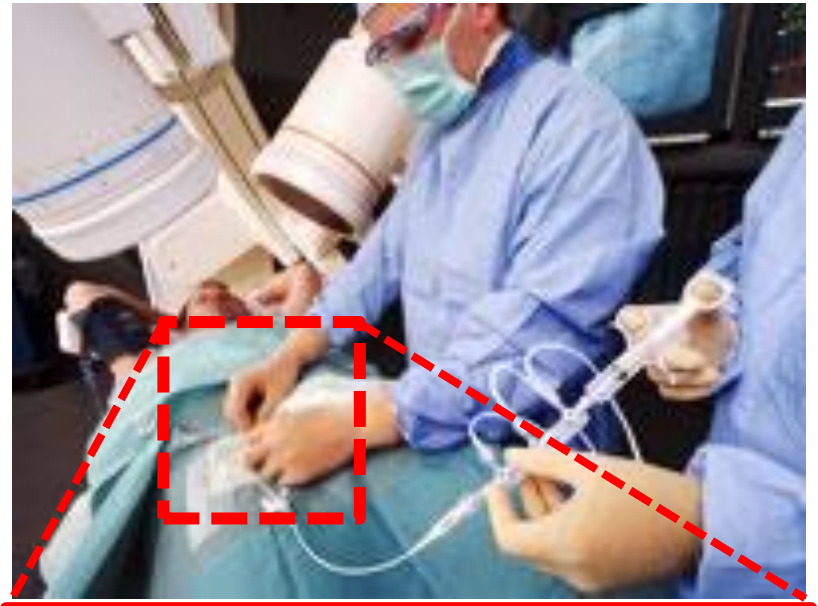
Endovascular

Surgery

Brain aneurysm: endovascular treatment

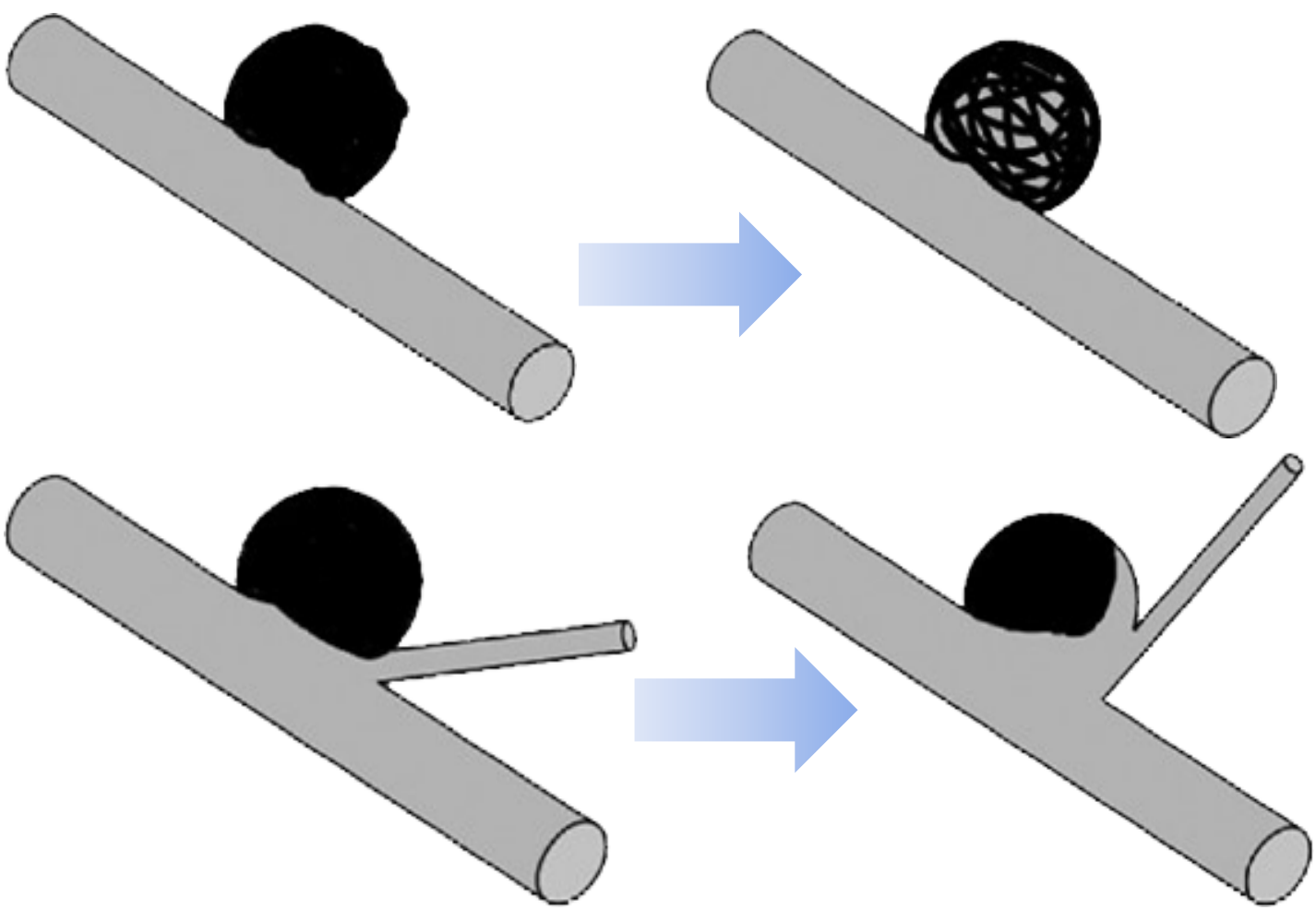
- ↓ aggressivity
- Faster recovery
- Ideal for Fisher grades II & III
  - Subarachnoid haemorrhage removal not easy
- Need yearly follow-up angiogram
- Recanalization 40-60%
  - Retreatment needed (endovascular / surgery)

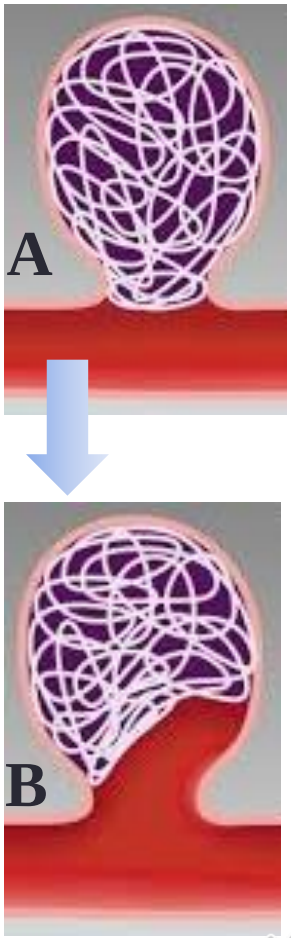




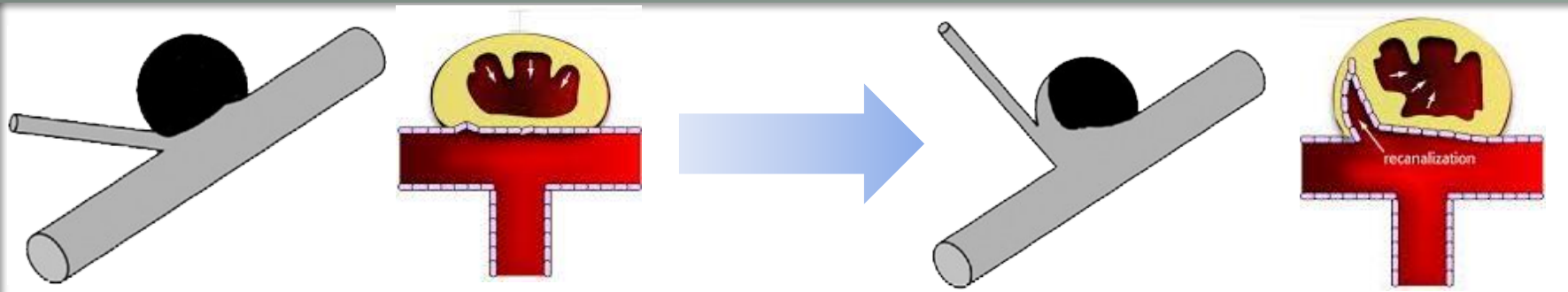
Recanalization embolized aneurysm

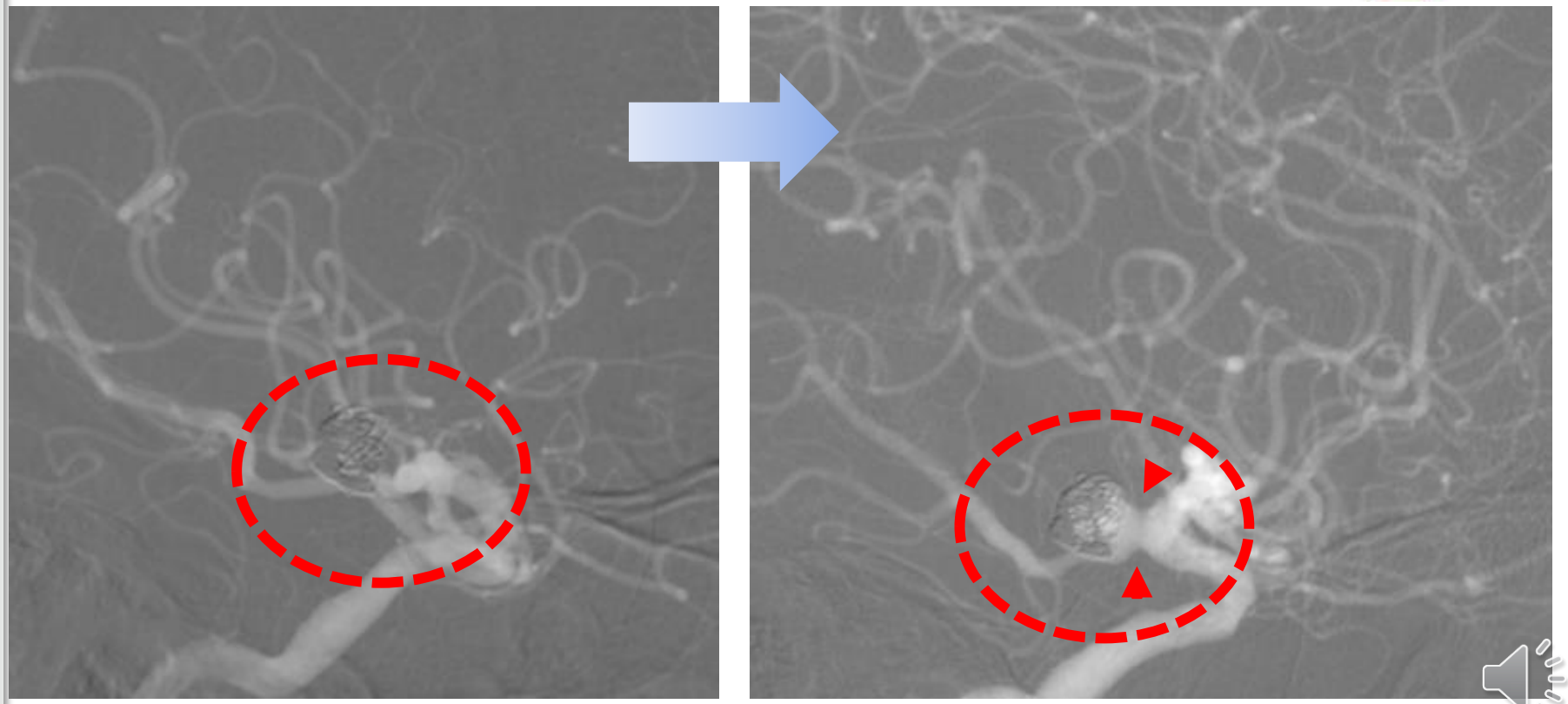
- Usually happens at the neck





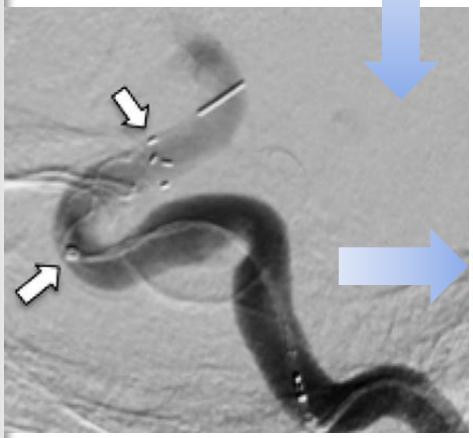
Recanalisation embolized aneurysm

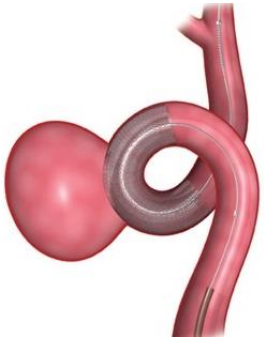


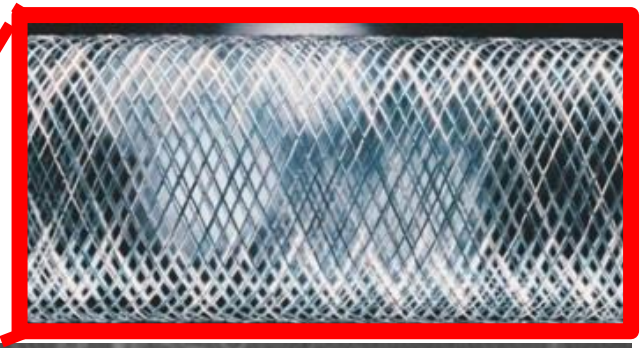


Fusiform aneurysm: flow diverter

- Double antiplatelet medication needed for life (acetylsalicylic acid + clopidogrel)
  - ↑ risk brain bleeding after minor trauma

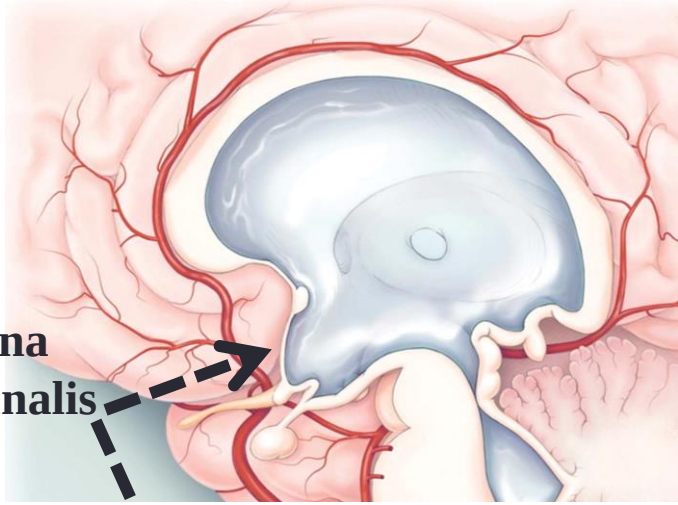


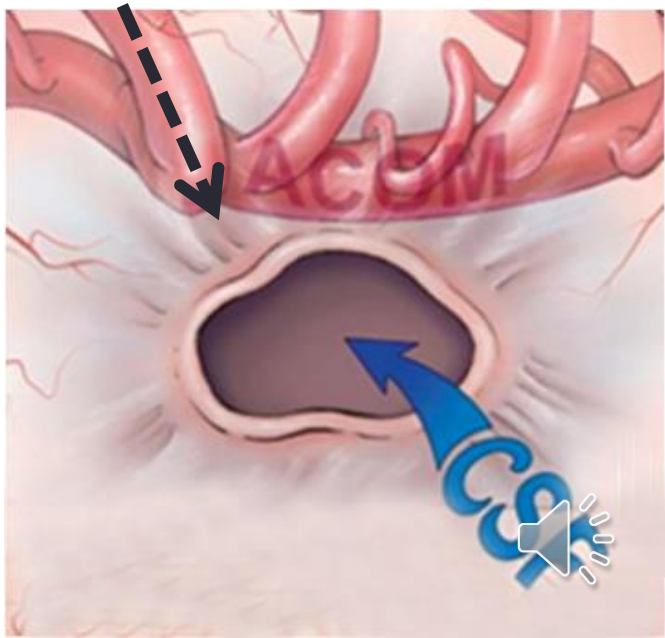




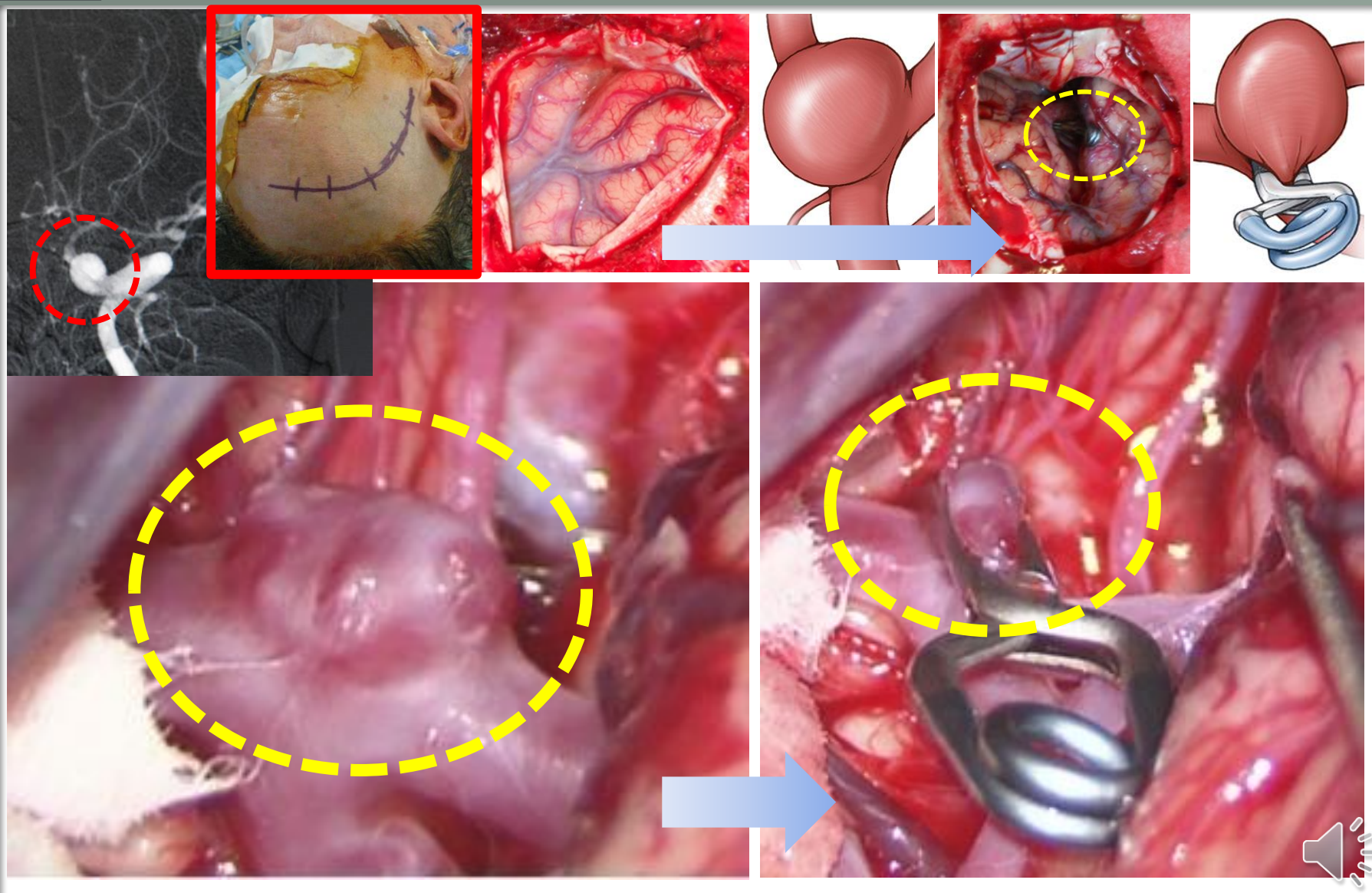
Brain aneurysm: surgical treatment

- ⇑ initial morbidity
- Washing blood from cisterns possible
  - Improves vasospasm control
- Allows opening lamina terminalis
  - ⇓ risk hydrocephalus
- 5-year aneurysm regrowth 1.8%
- Only one control (CT angiography or angiography) at 5, 10, and 20 years to rule out NEW aneurysms
- Emergency clipping minimizes rebleeding risk
  - Maximum risk in first 24 hours
    - Especially in the first 6 hours

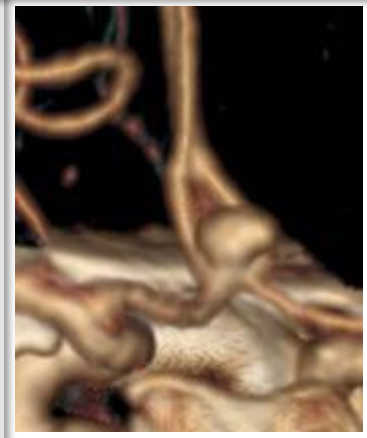


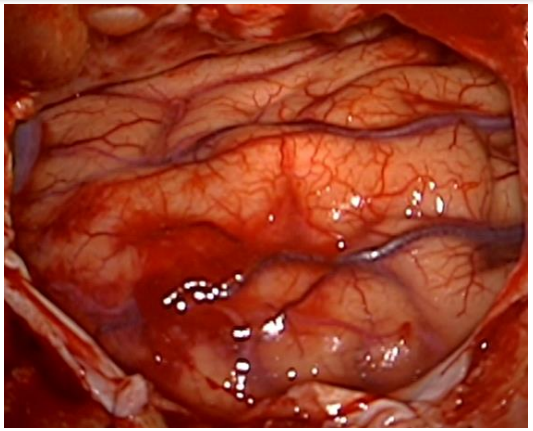
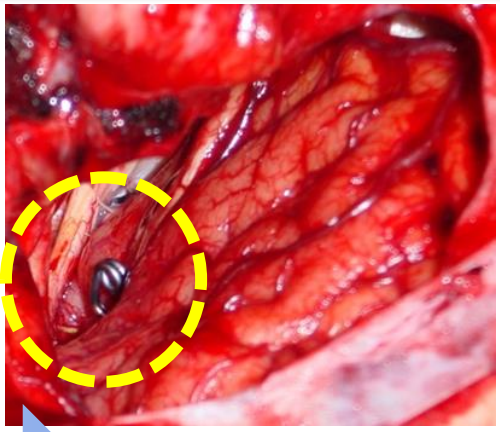
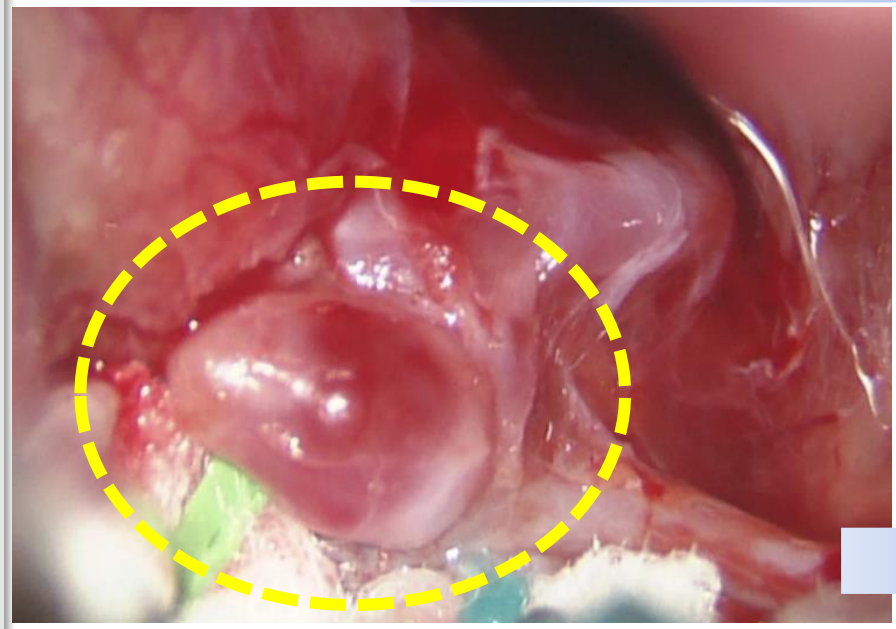
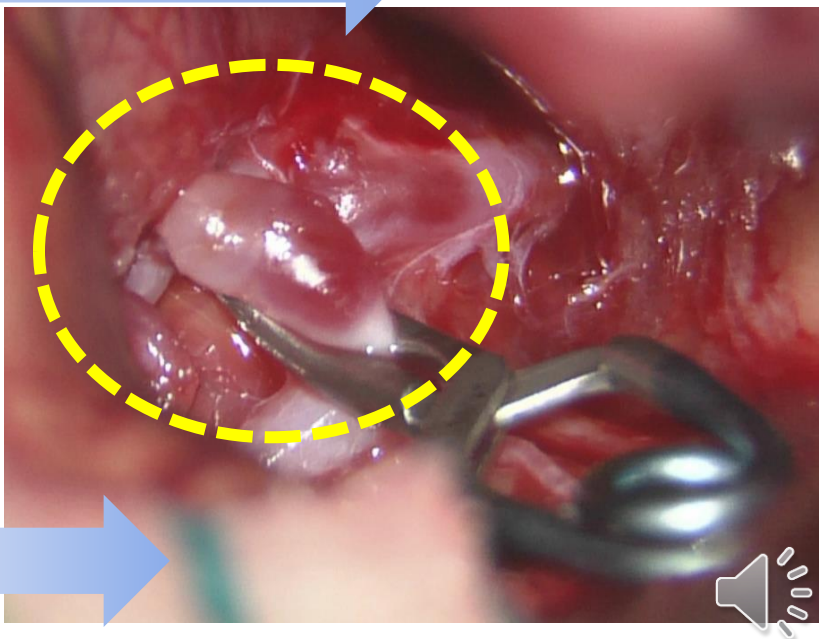


Middle cerebral artery aneurysm clipping

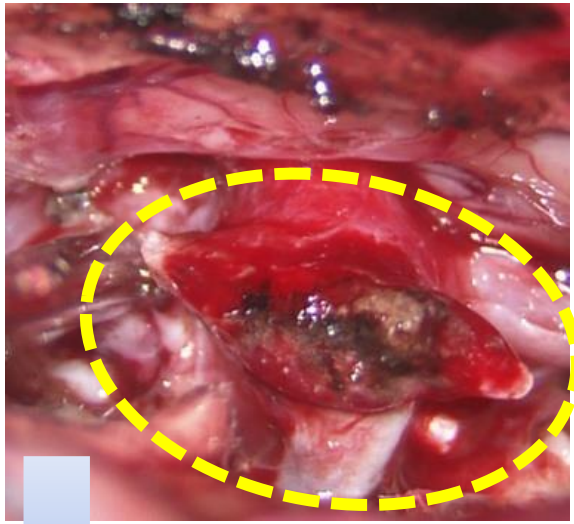


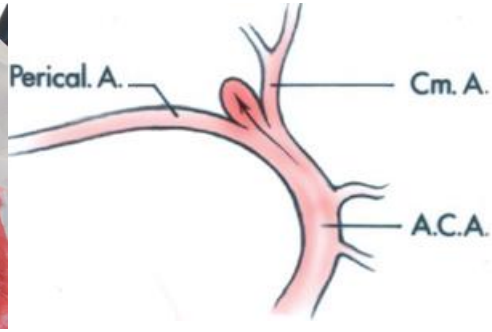
Anterior communicating artery aneurysm clipping

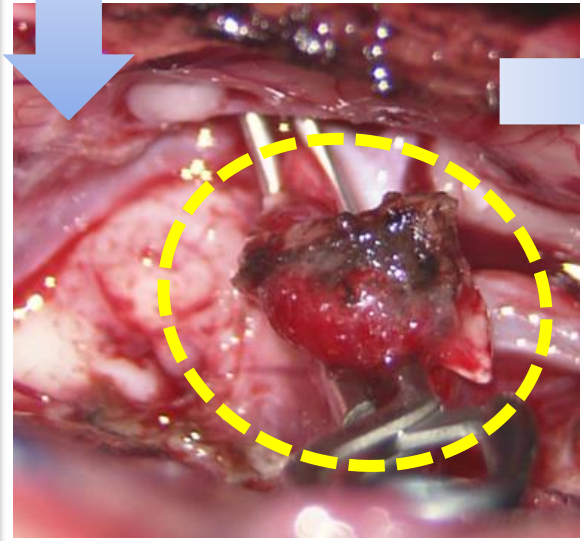


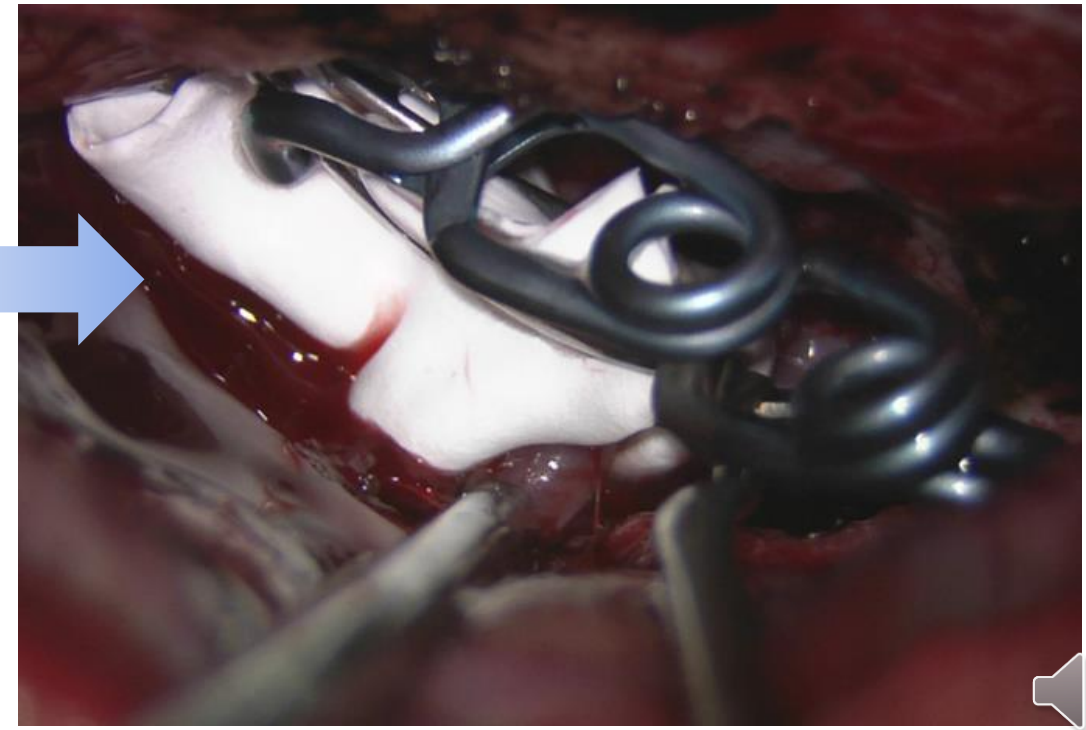





Wrapping a pericallosal artery fusiform aneurysm

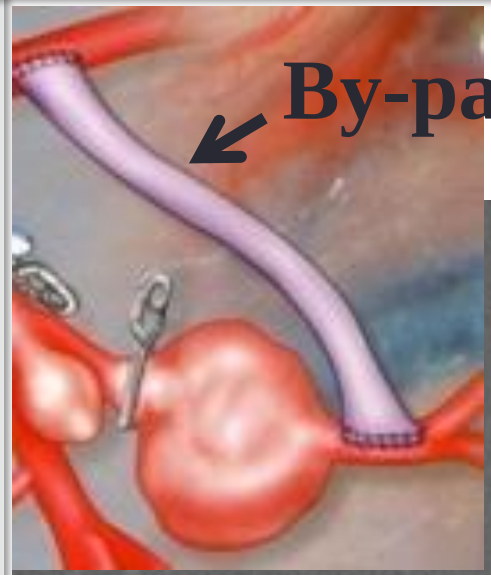


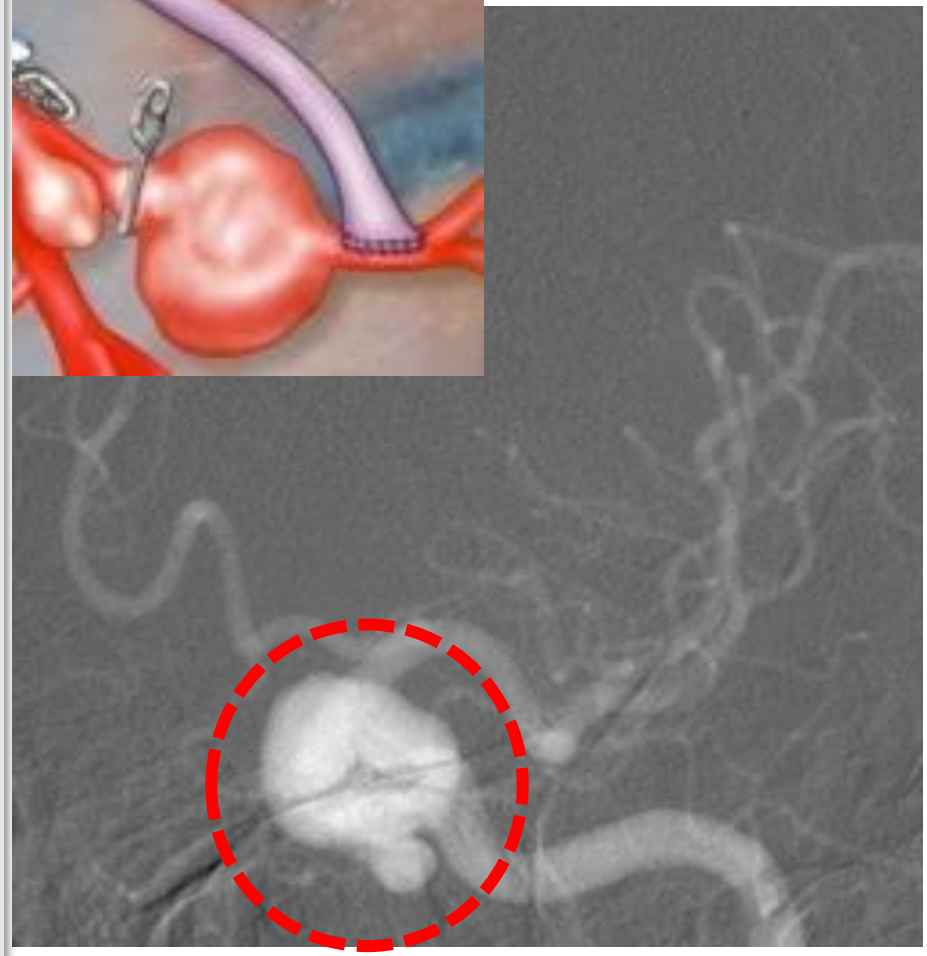


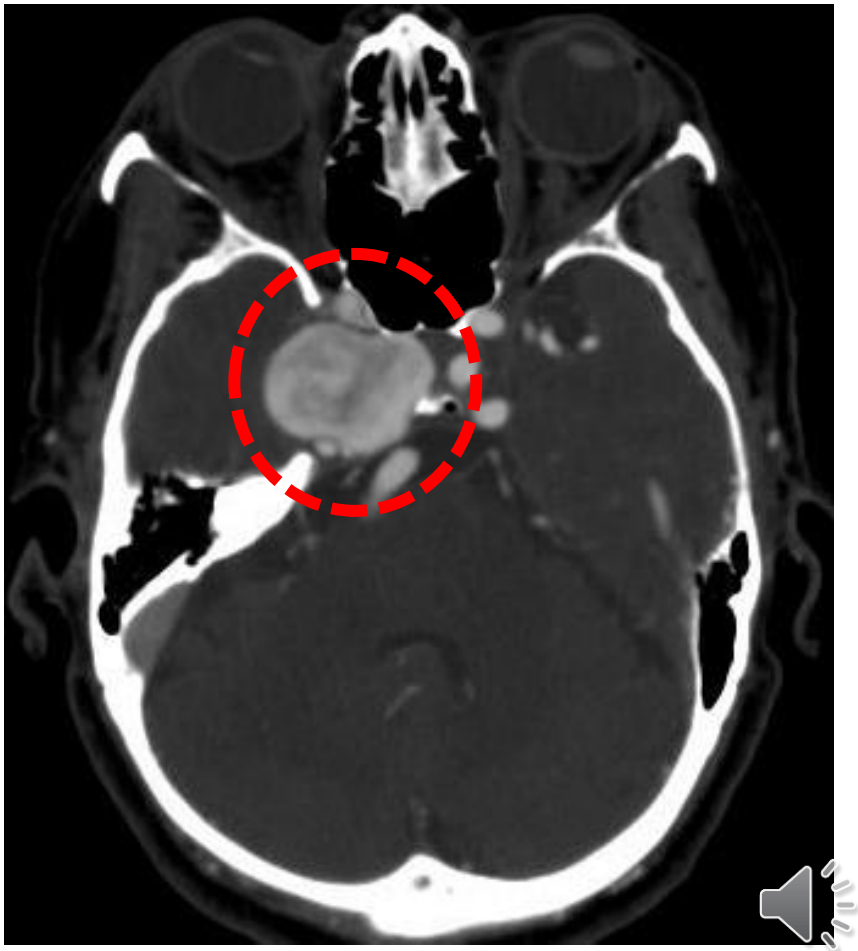




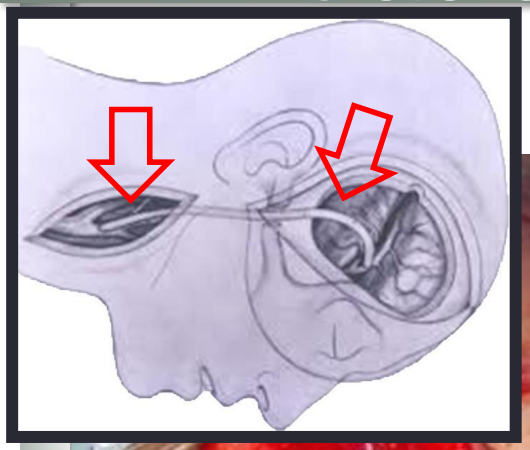
Giant aneurysm treatment: bypass

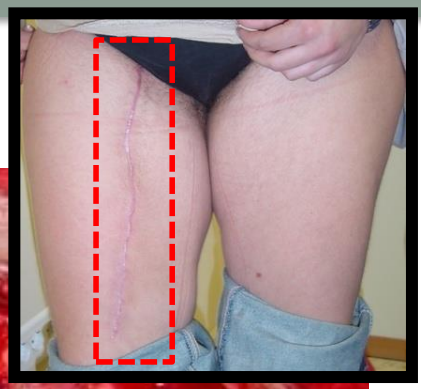


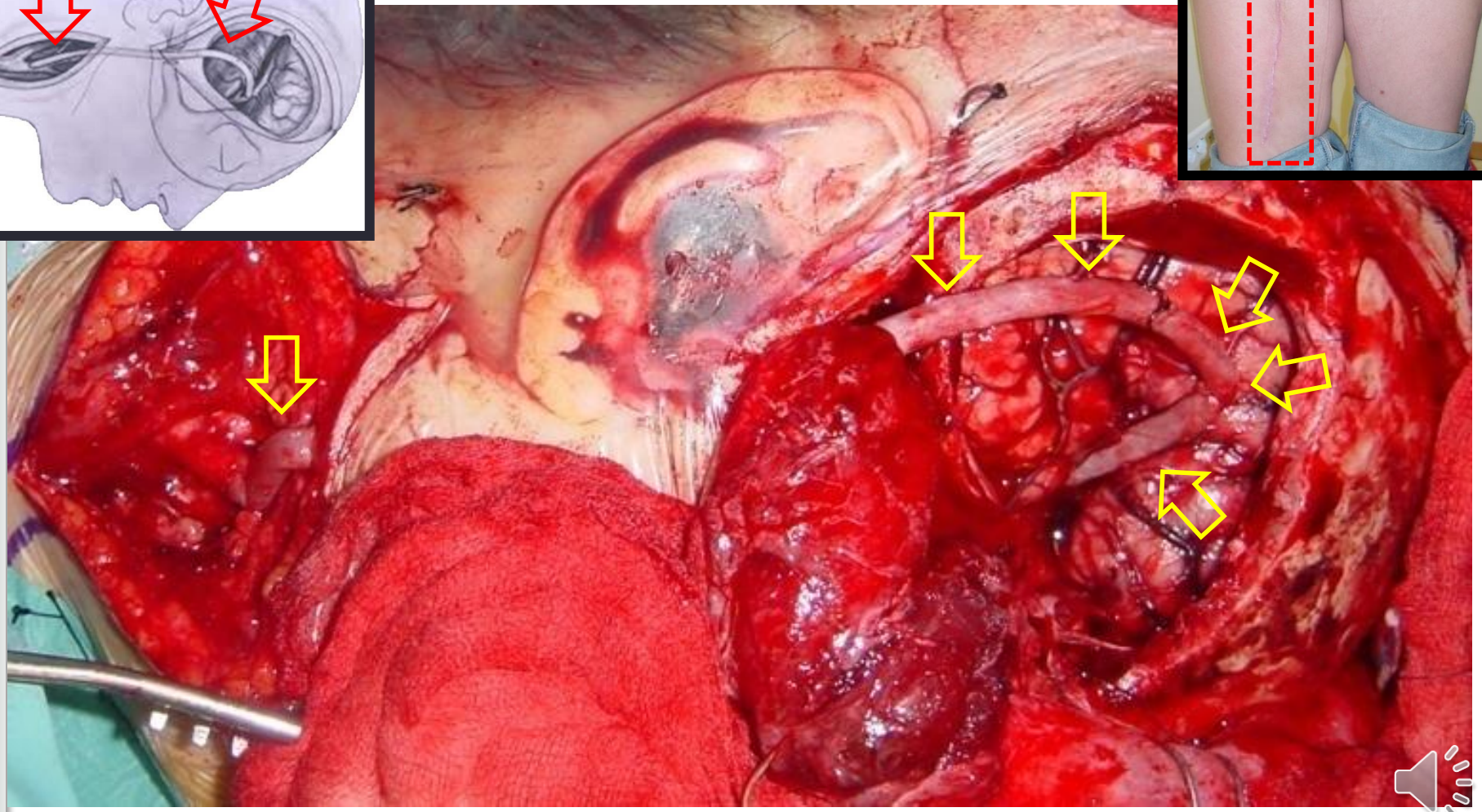




Giant aneurysm treatment: extra-intracranial bypass

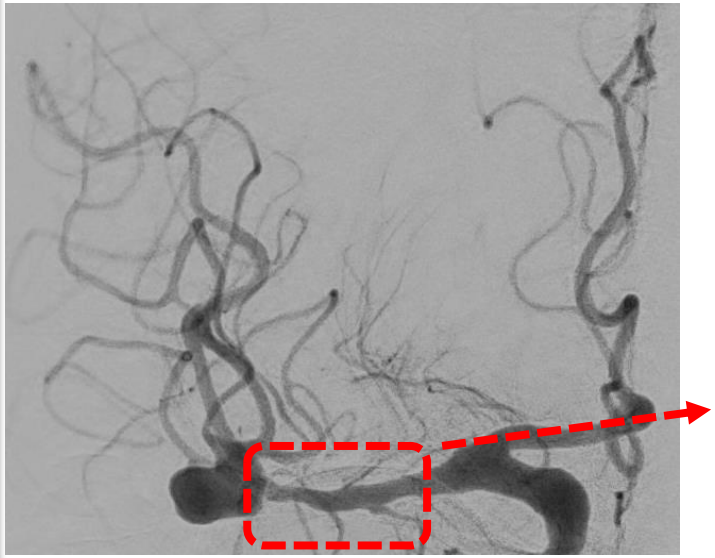


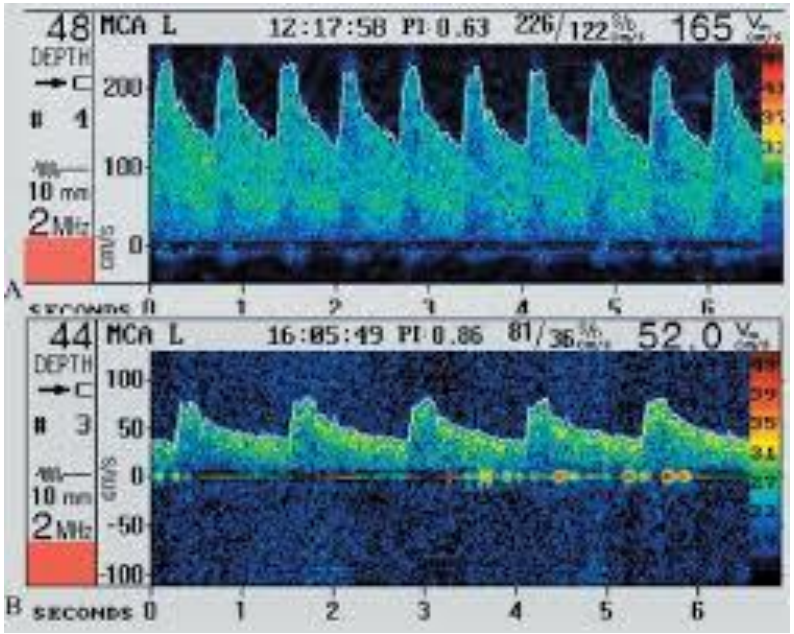




Complication prevention: vasospasm

- Nimodipine regularly
- Diagnosis = ecodoppler
  - Done daily
- Treatment
  - Increase mean arterial pressure
  - Intraluminal angioplasty

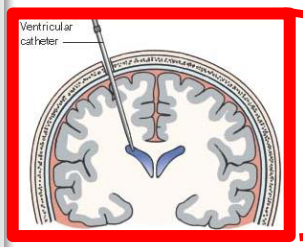


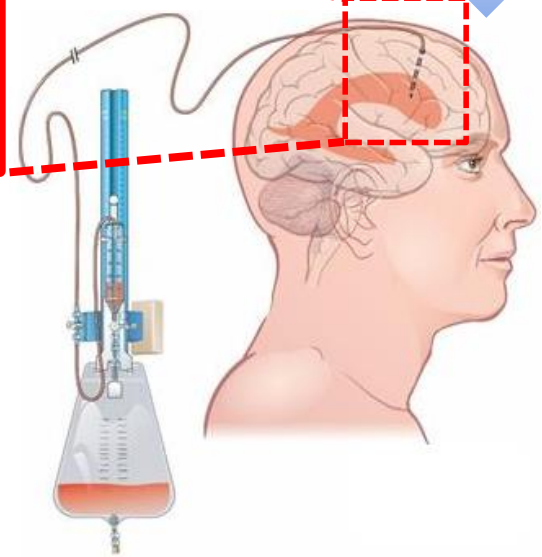


Normal

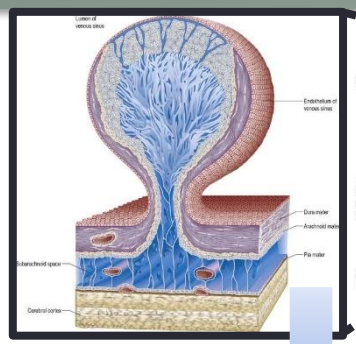
Vasospasm

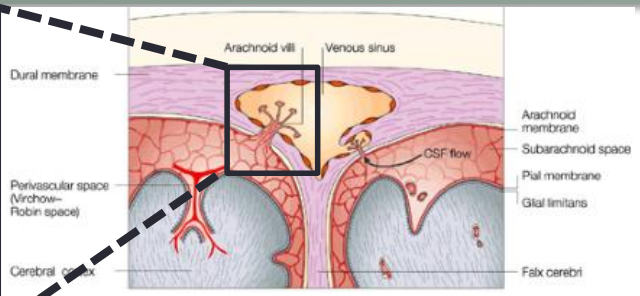
Complication treatment: hydrocephalus

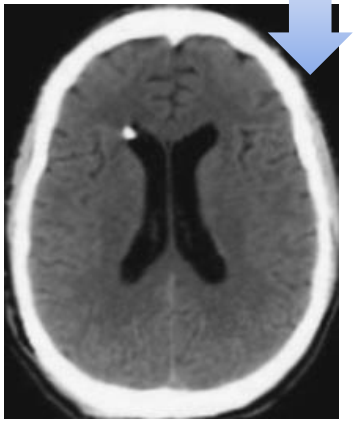


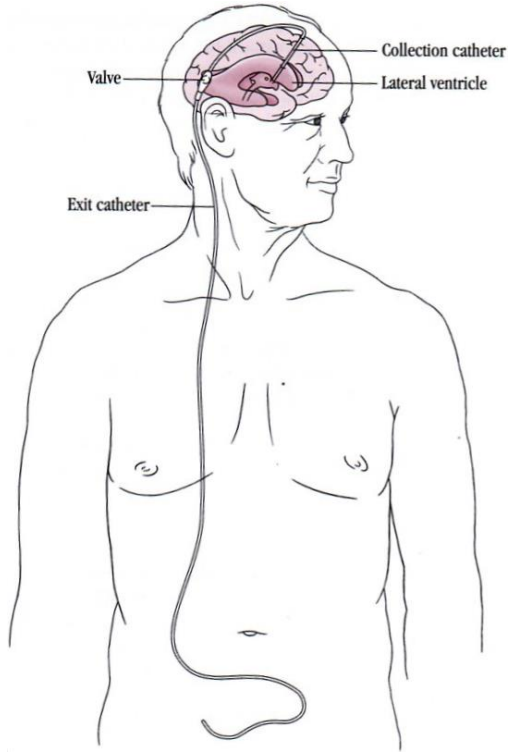


Urgent: external ventricular drainage



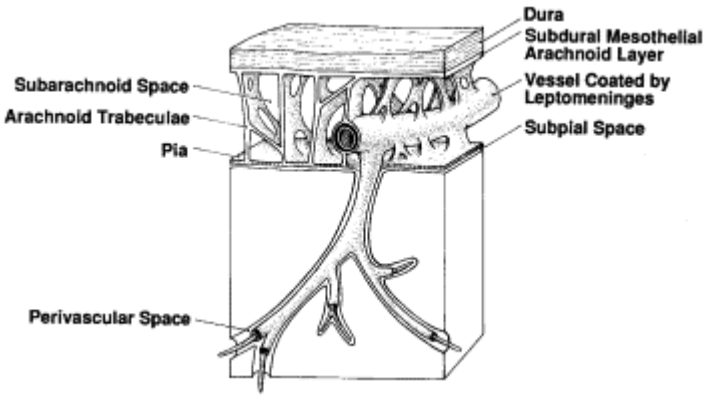


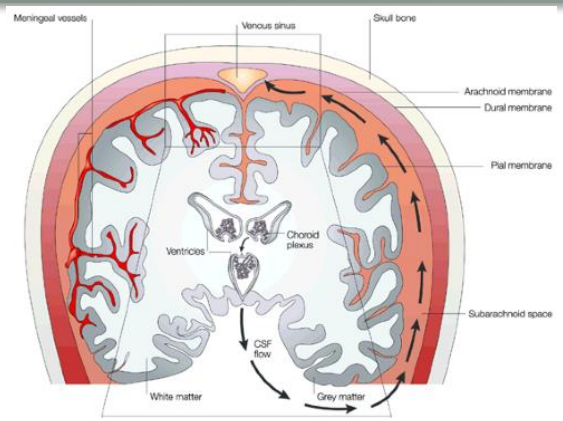


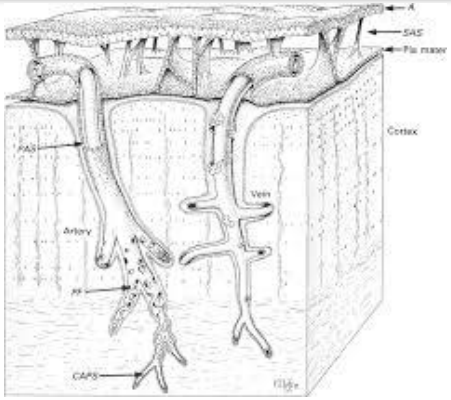


Definitive: ventricle-peritoneal shunt

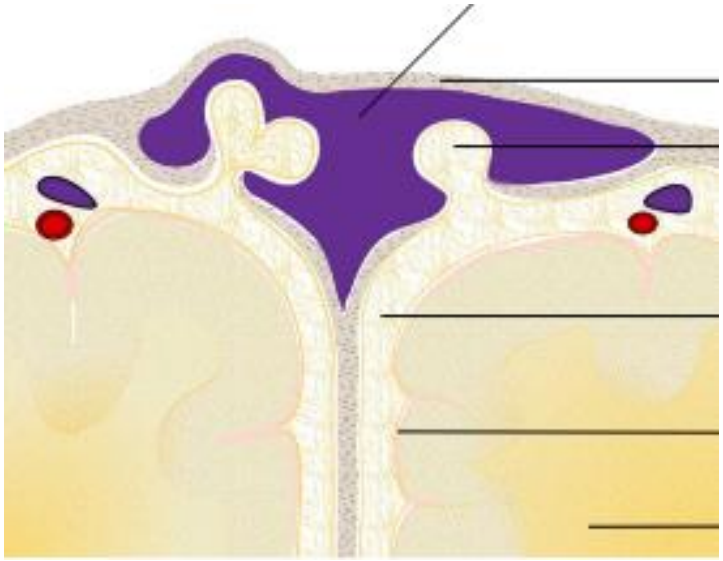
Hydrocephalus post subarachnoid haemorrhage = subarachnoid space + arachnoid villi fibrosis







Superior sagittal sinus



Dura

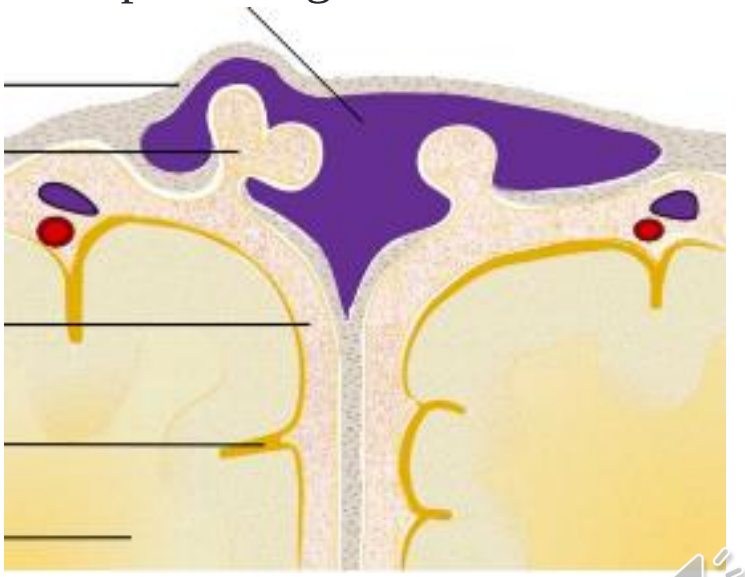
Arachnoid villi

Subarachnoid space

Pia

Cerebral parenchyma

Superior sagittal sinus

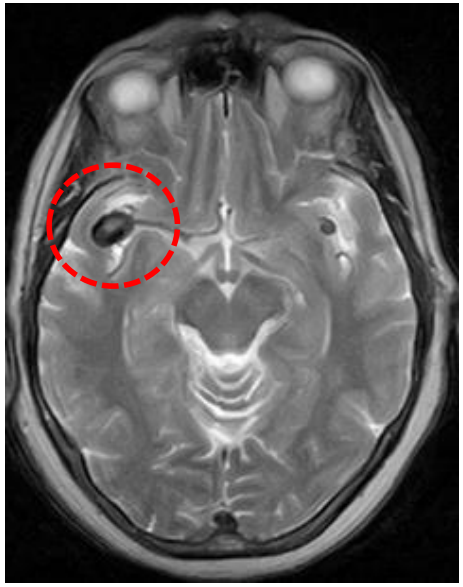


Normal

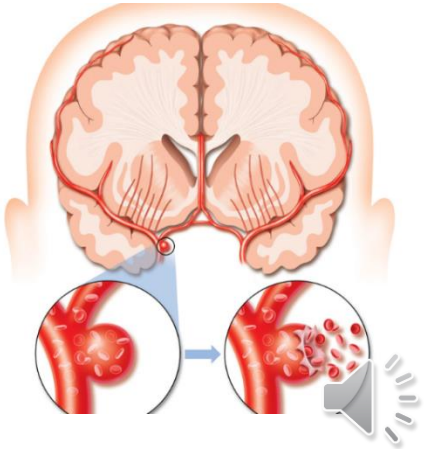
After subarachnoid haemorrhage

Incidental aneurysms: treatment

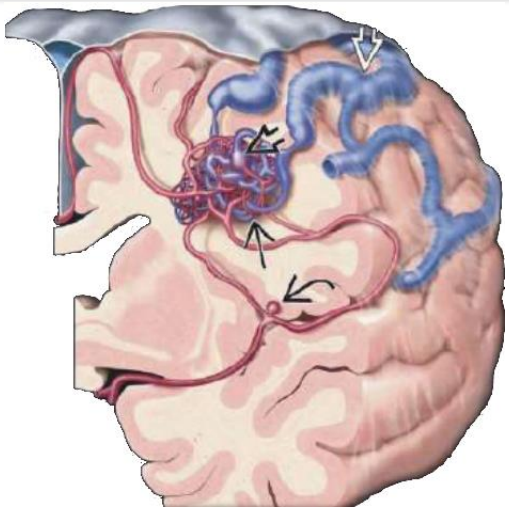
- Risk incidental aneurysm rupture 0.8% / year
- Ruptured aneurysms 71.8% <7mm & 87.9% < 10mm
- ⇑ patient age = ⇑ bleeding risk & ⇓ results
  - ∅ 2-4mm grow 2.4%
  - ∅ 5-9mm grow 9.1%
  - ∅ 10-20mm grow 50%
- Growth probability as ⇑ ∅ aneurysm



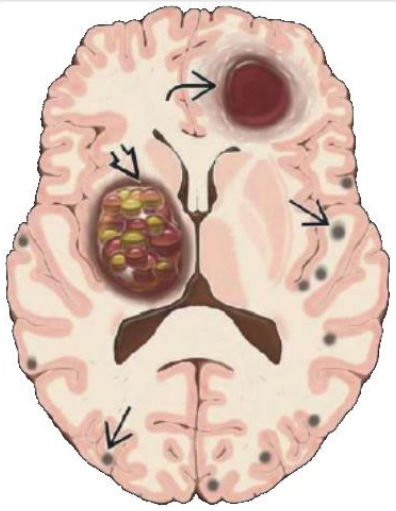
Incidental aneurysm on MRI



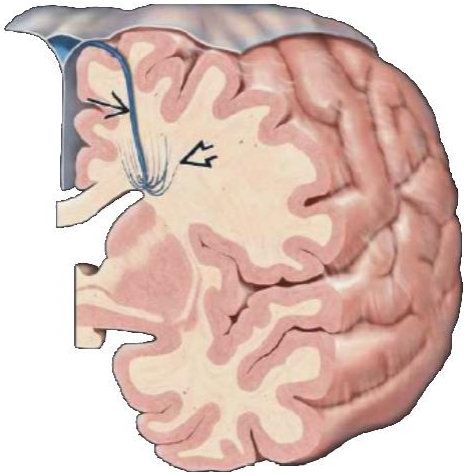
BRAIN VASCULAR MALFORMATIONS



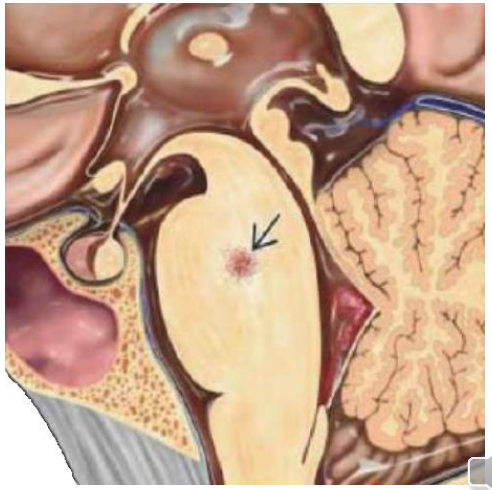
Brain arteriovenous malformation (AVM)



Cavernous angioma



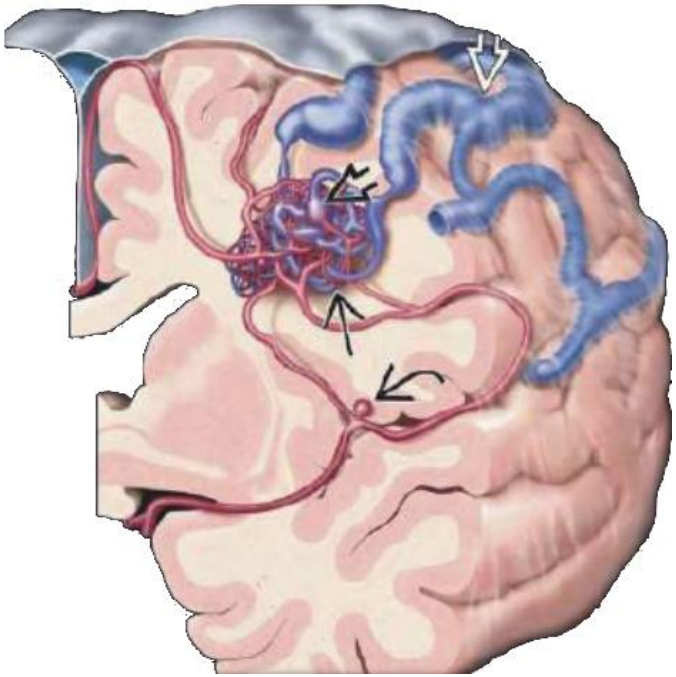
Venous angioma

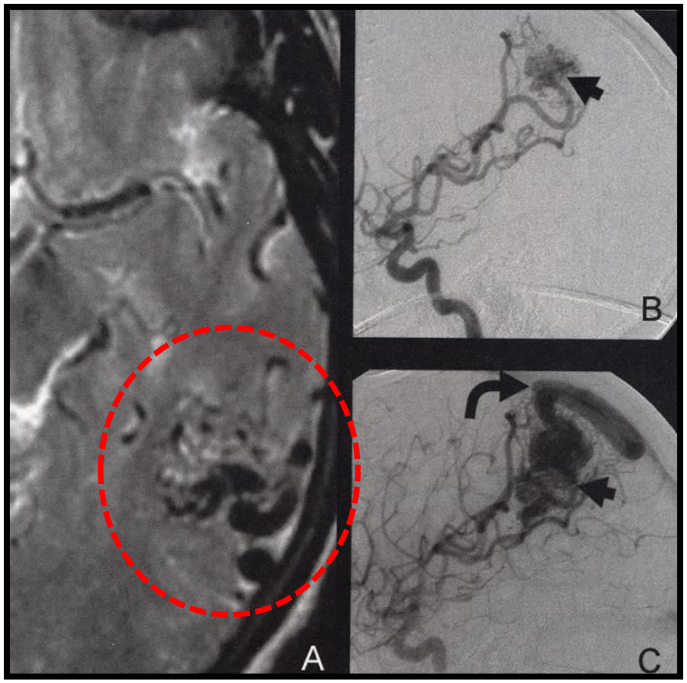


Capillary telangiectasia

Arteriovenous malformation (AVM)

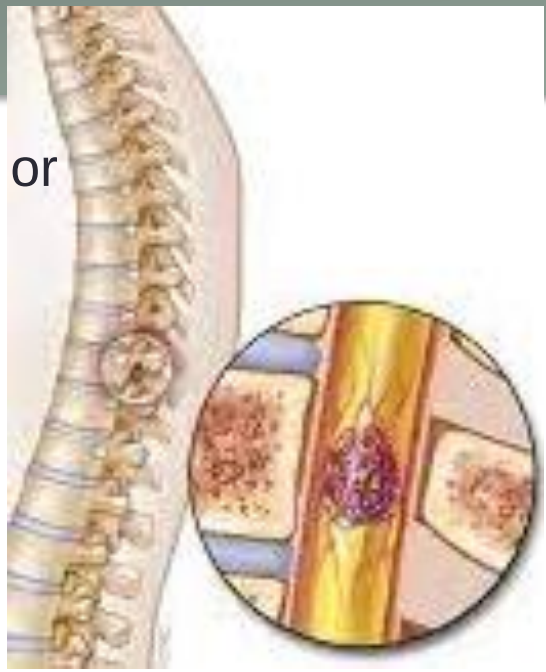
- The most frequently symptomatic
- Arteries connect directly with veins with no capillary bed
- Clinical symptoms: intracerebral haemorrhage / epileptic seizures
- Diagnosis: angiography, MRI, & CT scan
- Treatment: surgery, radiosurgery ± endovascular treatment

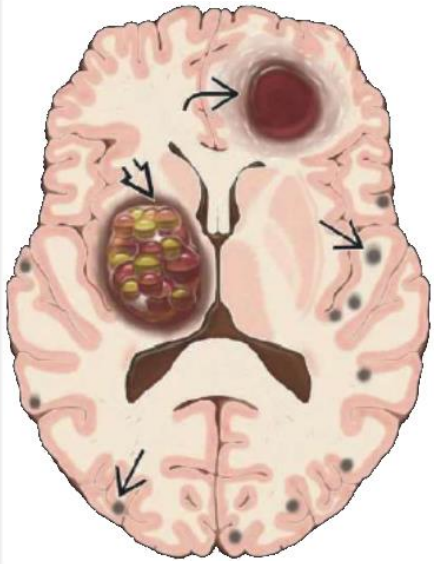


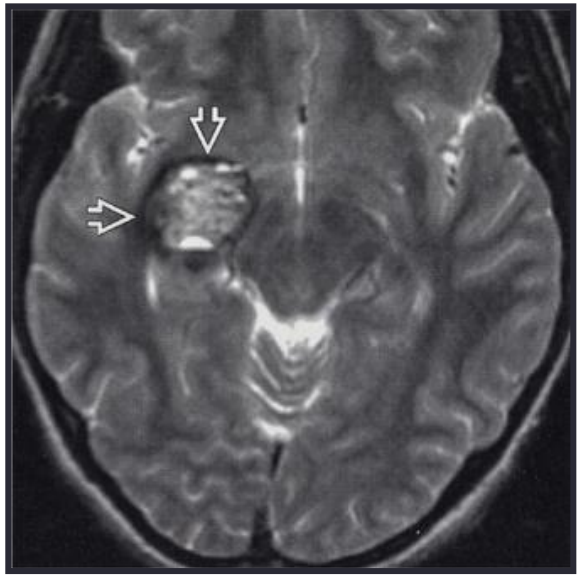


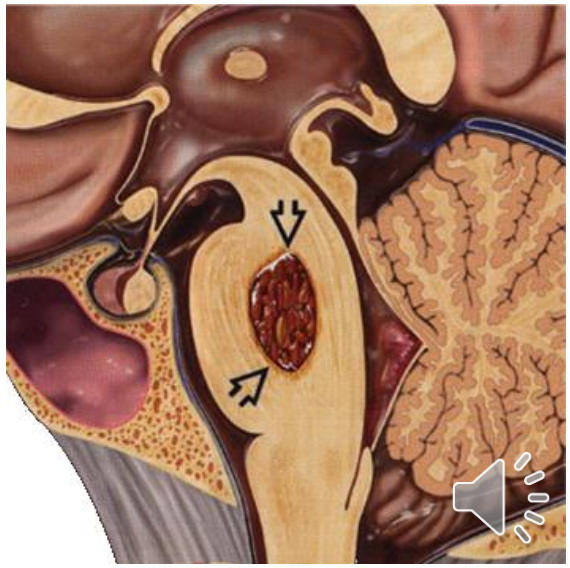
Cavernous angioma

- Enlarged vascular spaces without arteries, veins, or cerebral parenchyma
- Clinical symptoms: headache, epileptic seizures, haemorrhage
- Diagnosis: MRI, often multiple lesions
- Treatment: surgery if incontrollable seizures / brainstem or spinal cord location



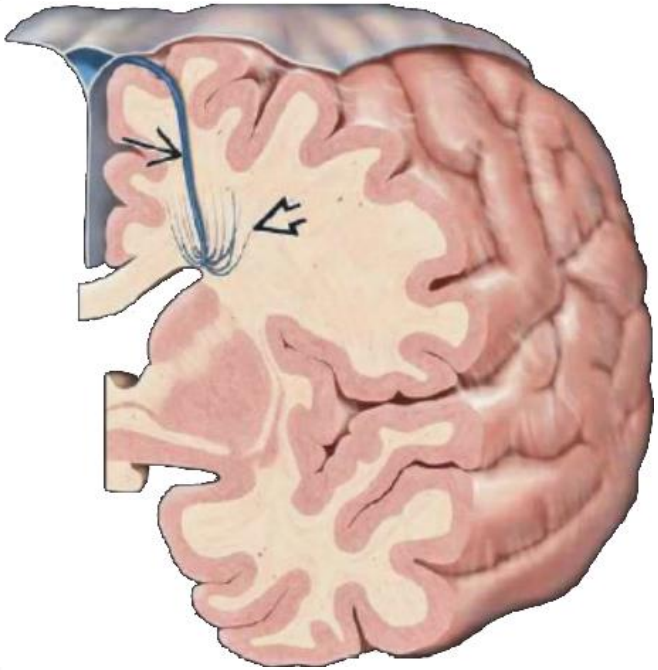


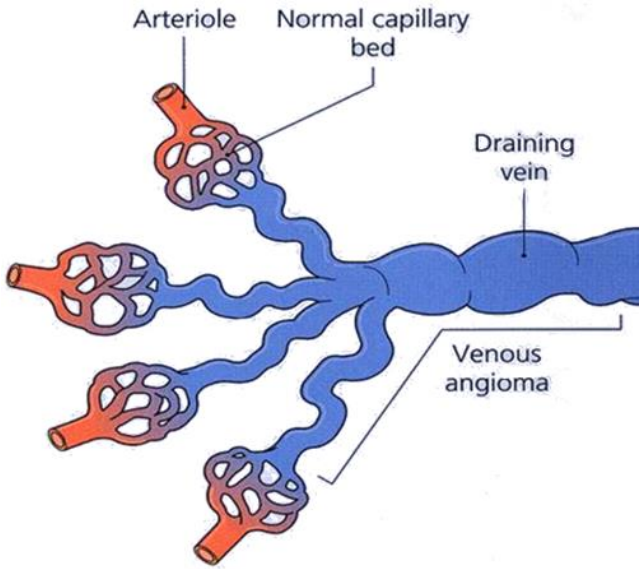




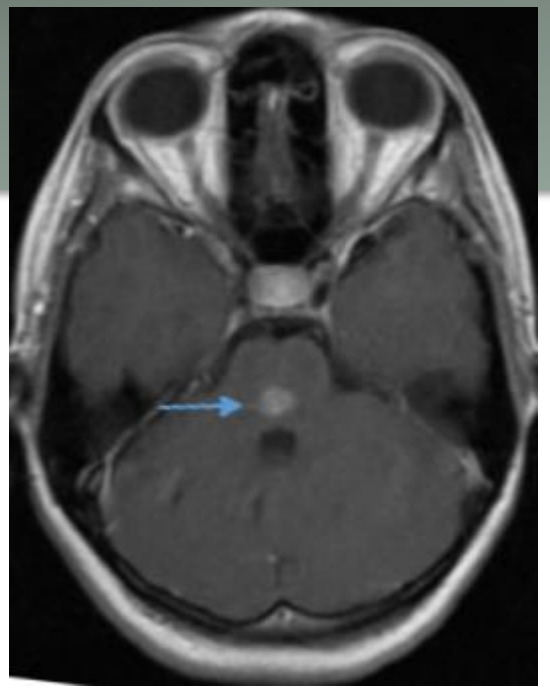
Venous angioma

- Normal veins that converge on a large venous trunk draining normal brain parenchyma
- Frequent but asymptomatic
  - Rarely bleed
- Diagnosis: angiography & MRI (caput medusae)
- No treatment needed

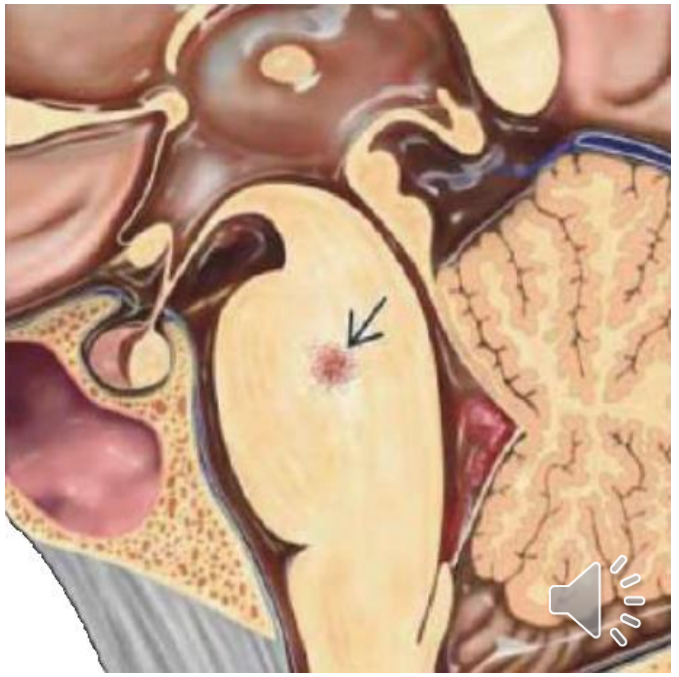




Capillary telangiectasia

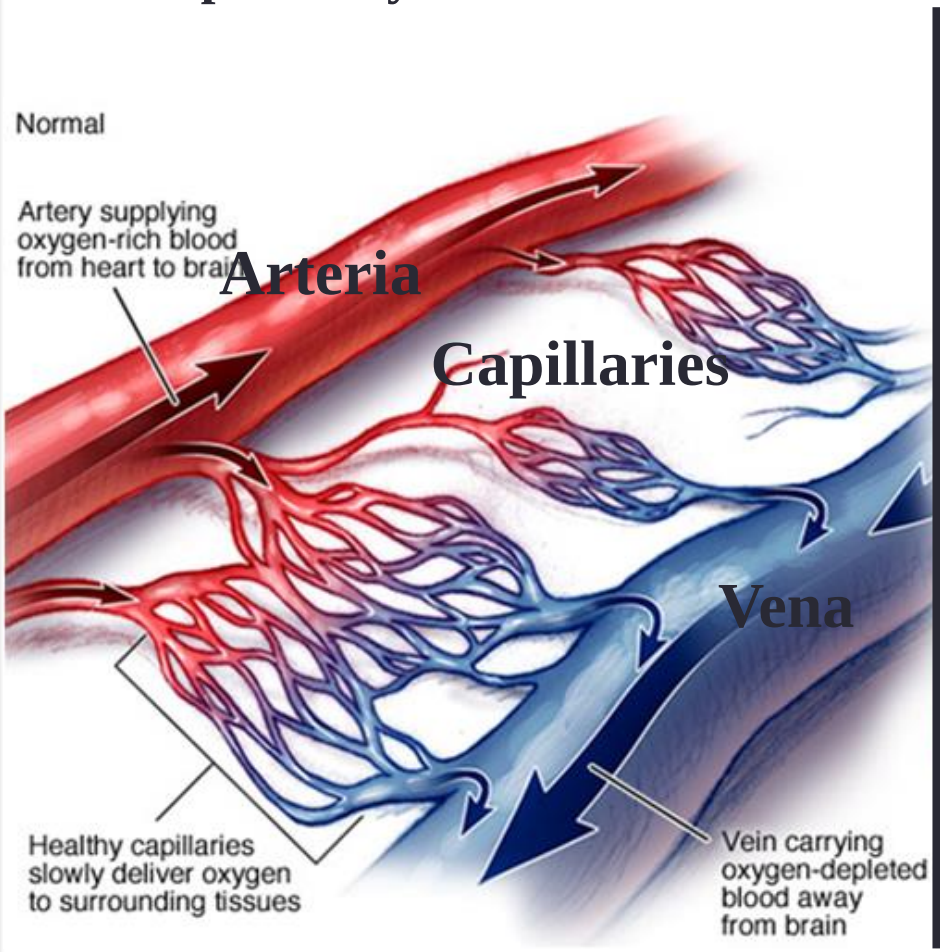


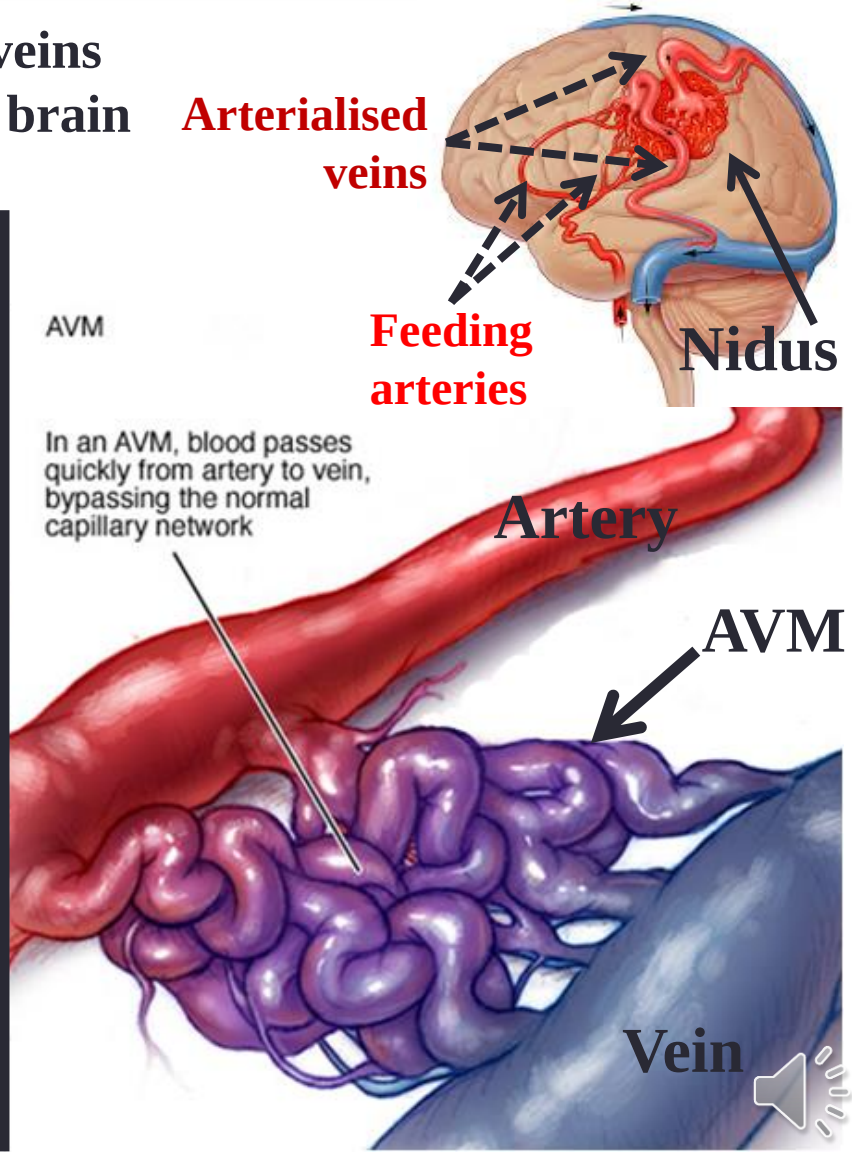
- Capillary dilations in normal brain parenchyma
- Location
  - Basal ganglia, brainstem, cerebellum
- Association
  - Rendu-Osler, ataxia-telangiectasia
- Usually no symptoms
  - If bleeding = dismal prognosis
- Diagnosis: only with MRI
- Treatment: not needed & extremely dangerous



Brain AVM (arteriovenous malformations)

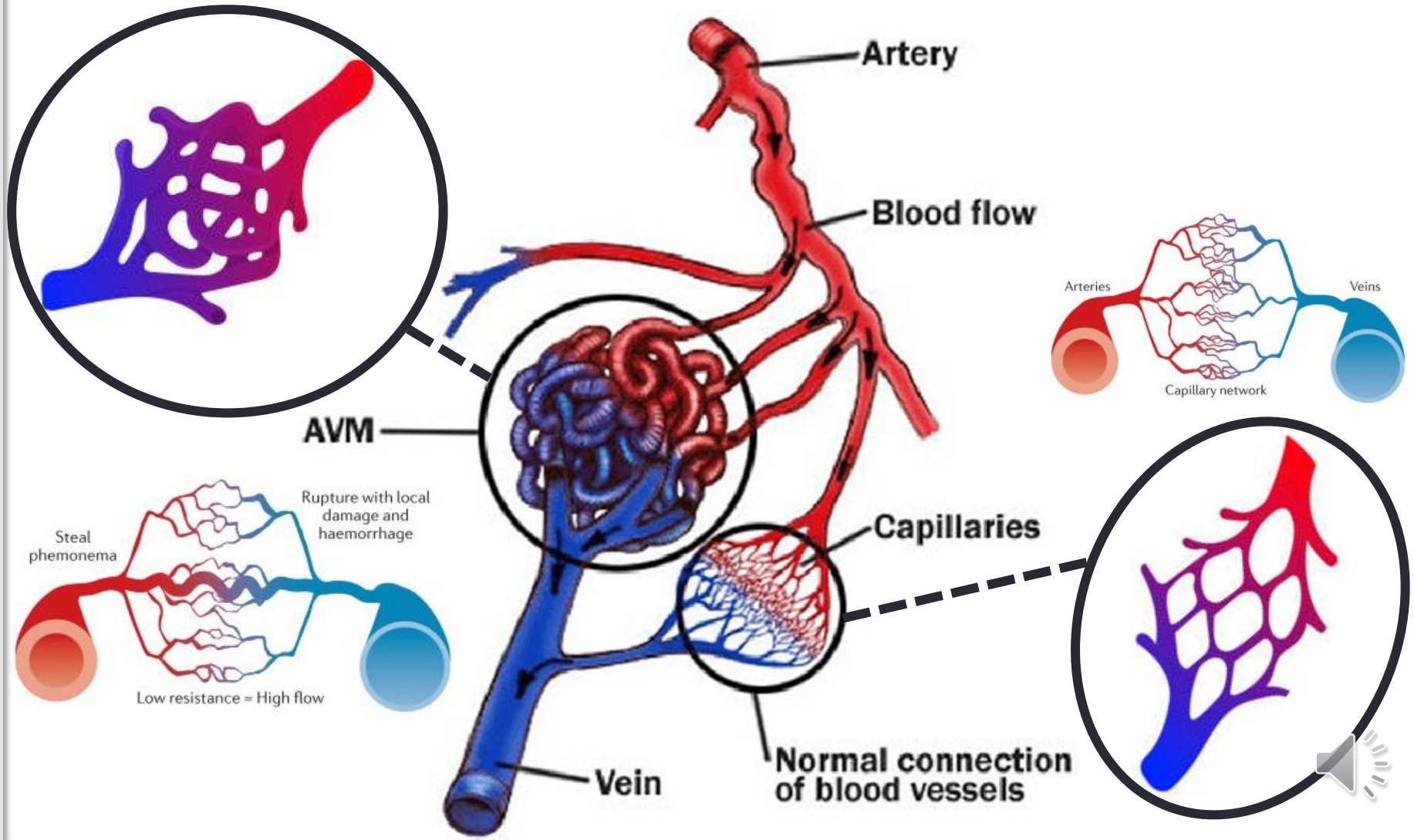
Direct connection between arteries and veins WITHOUT interposed capillary bed. NO brain parenchyma within the nidus





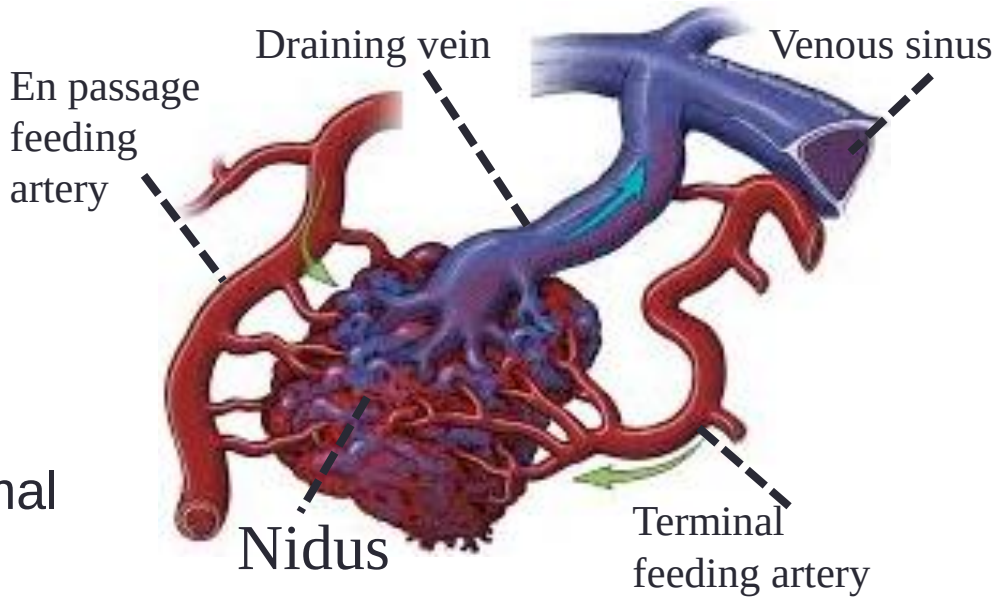
Differences normal brain vs AVM

- AVM = absence capillary bed

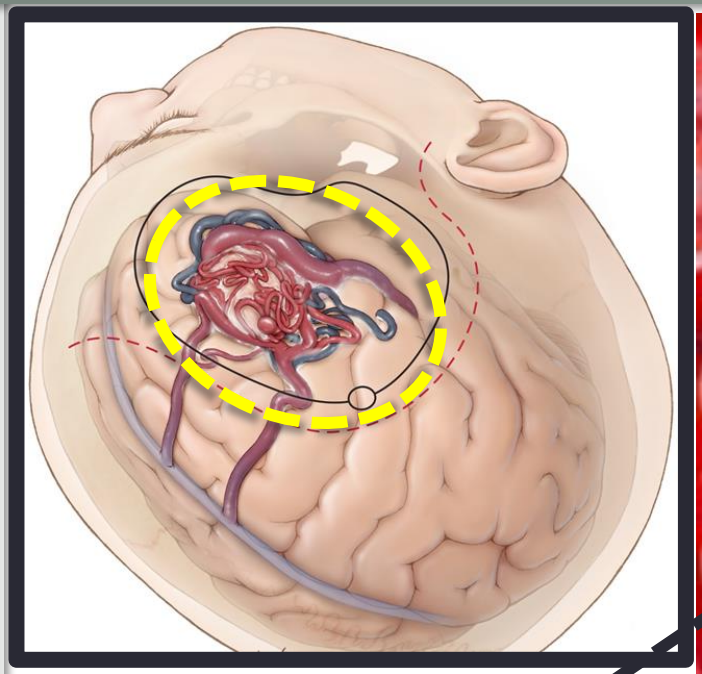


Brain AVM: basic components

- **Feeding arteries**
  - Terminal arteries = can be sacrificed
  - En passage feeding arteries = carry blood to the nearby brain = cannot be sacrificed
- **Nidus** = Vascular clew where arteries connect directly to veins
- **Draining vein/s**
  - They are arterialised = abnormal high pressure
    - Dilatation = wall thinning = risk of rupture = haemorrhage
    - ↑ venous sinus pressure = hydrocephalus



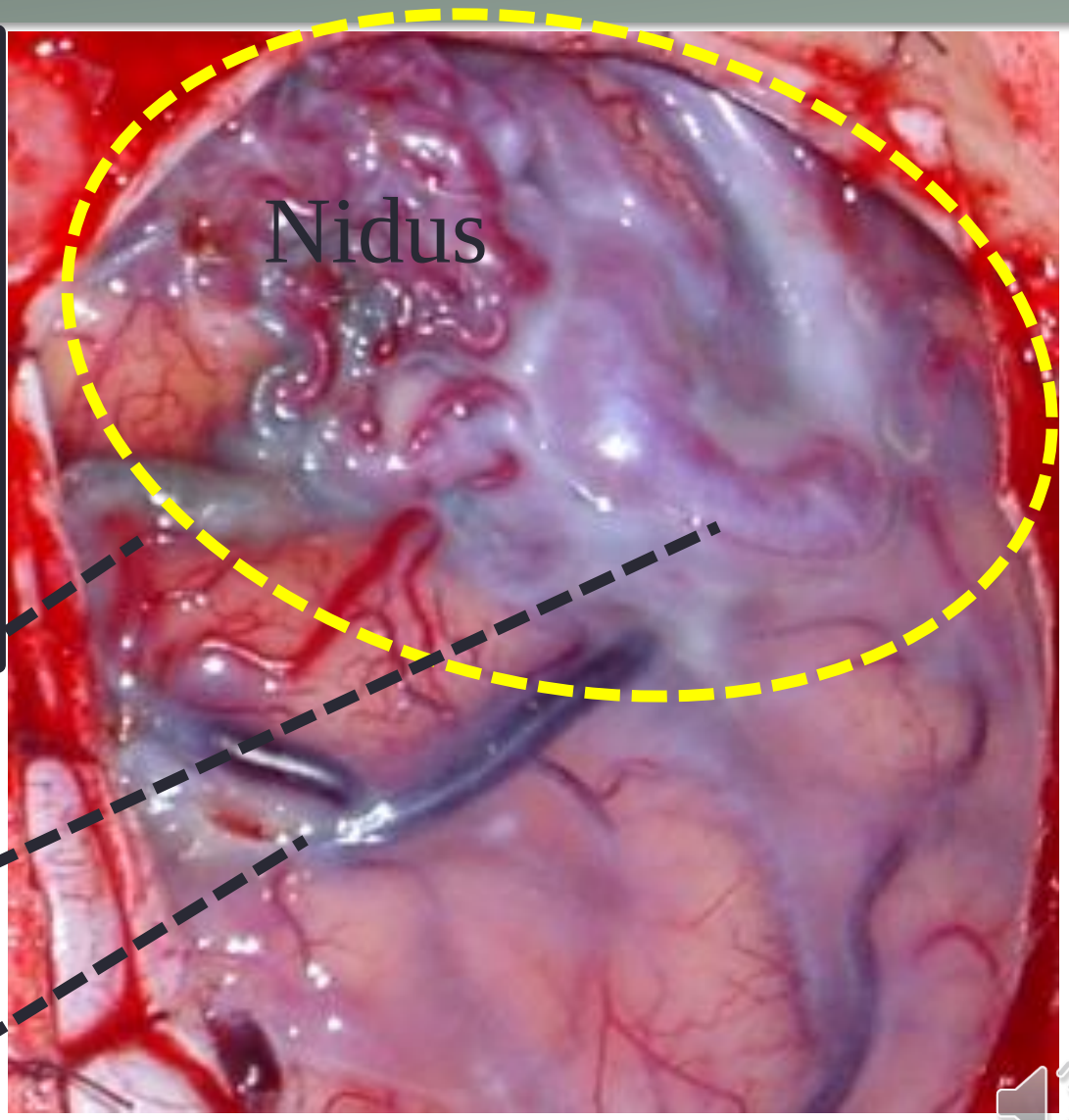
Frontal lobe AVM



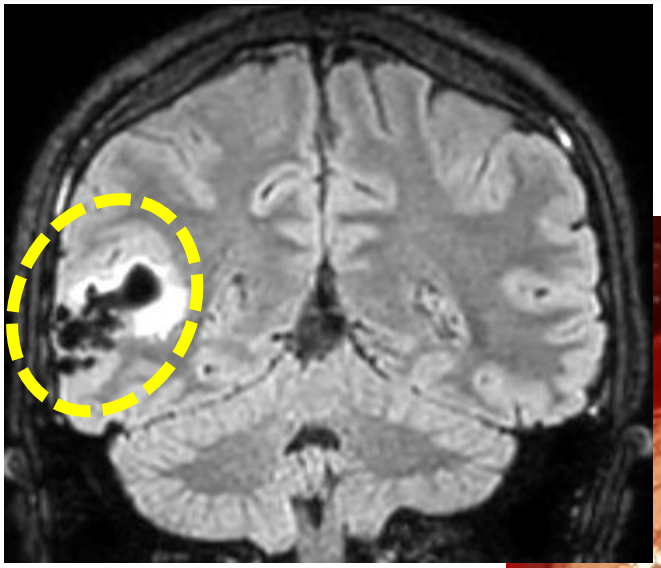
Embolised artery

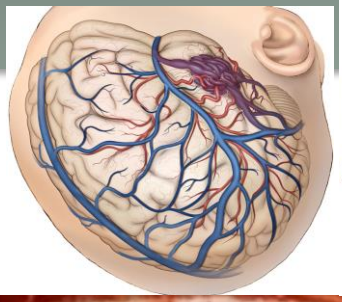
Arterialised draining vein

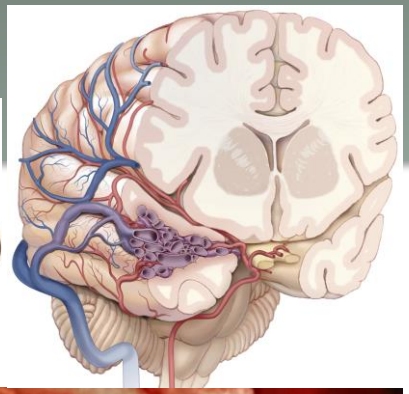
Normal vein



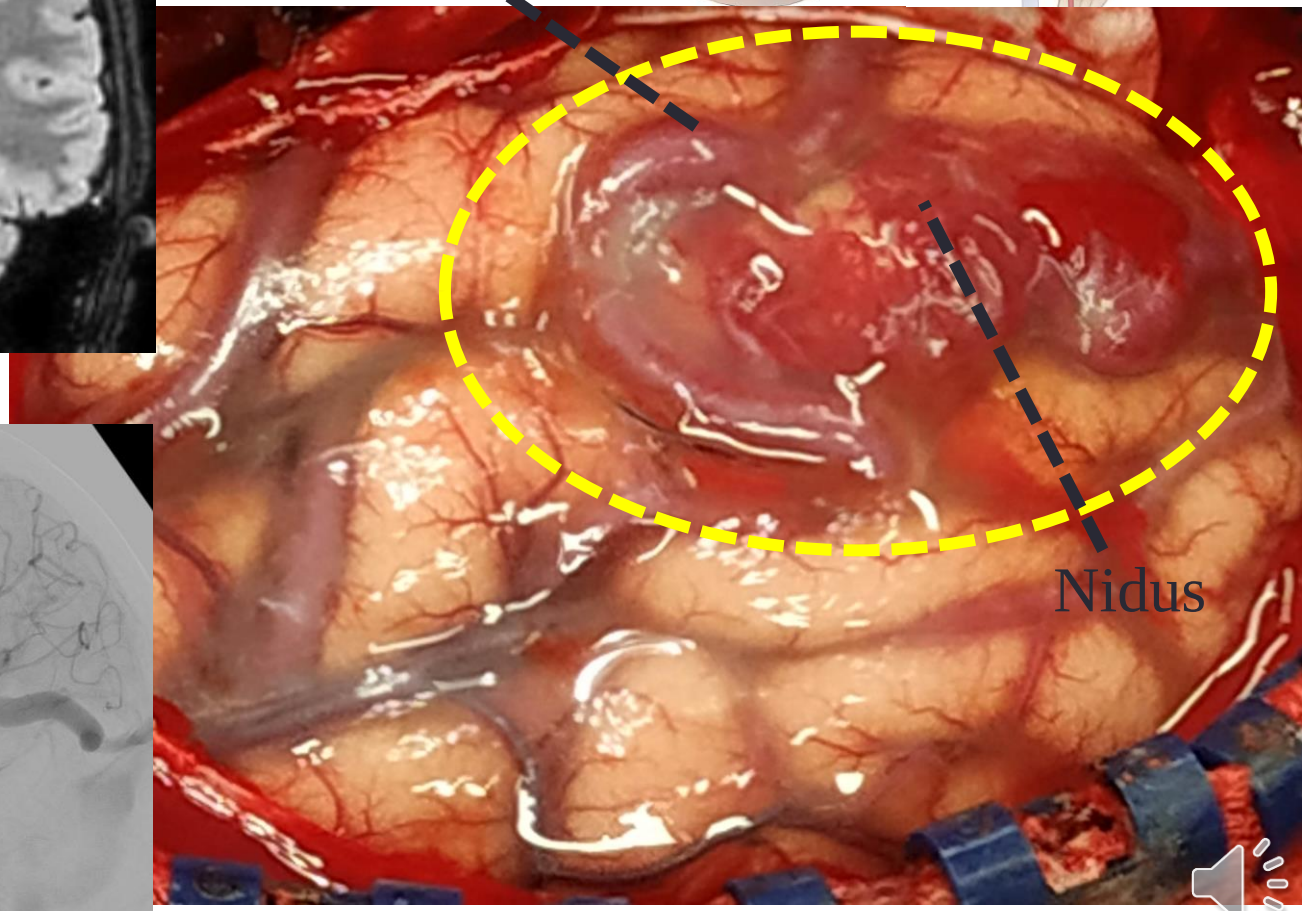
Temporal lobe AVM

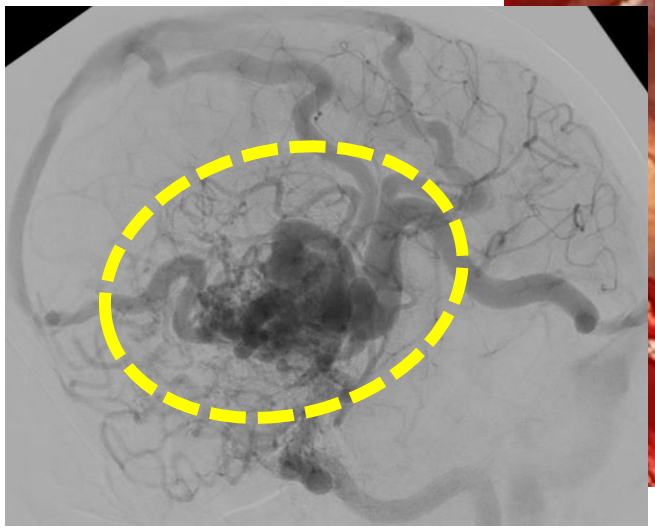




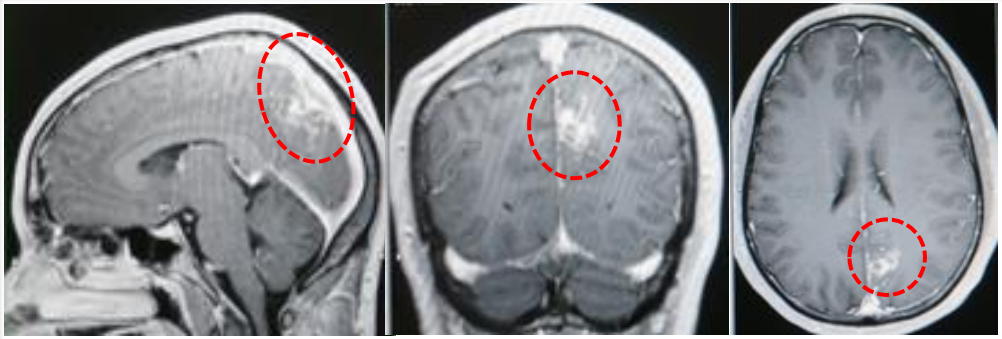


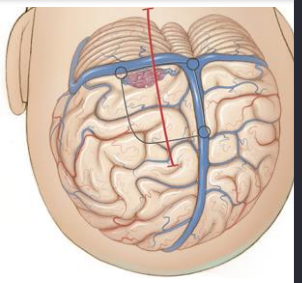
Arterialised draining vein

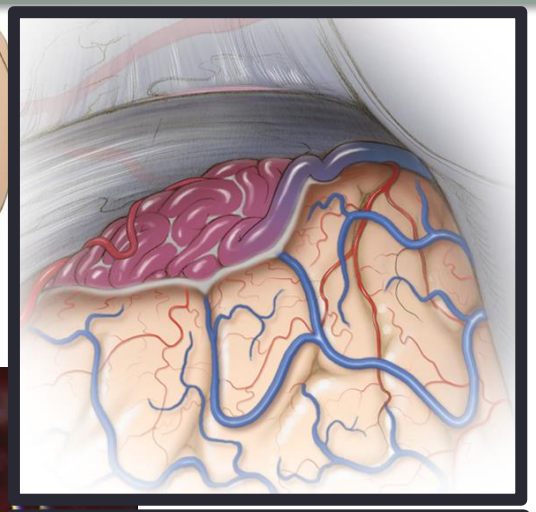


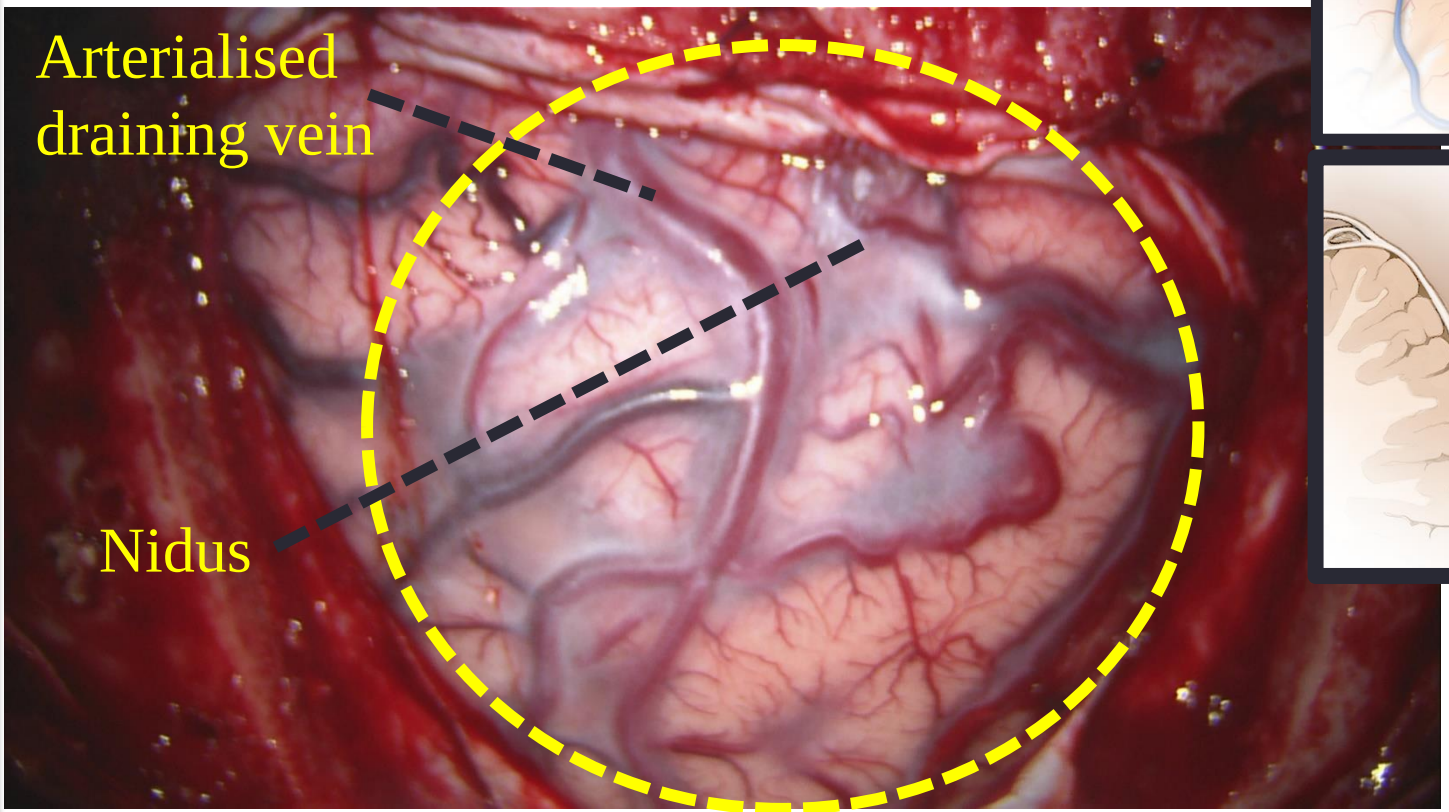


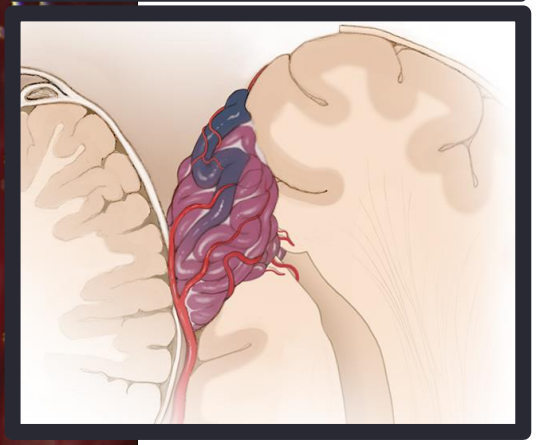
Left occipital AVM



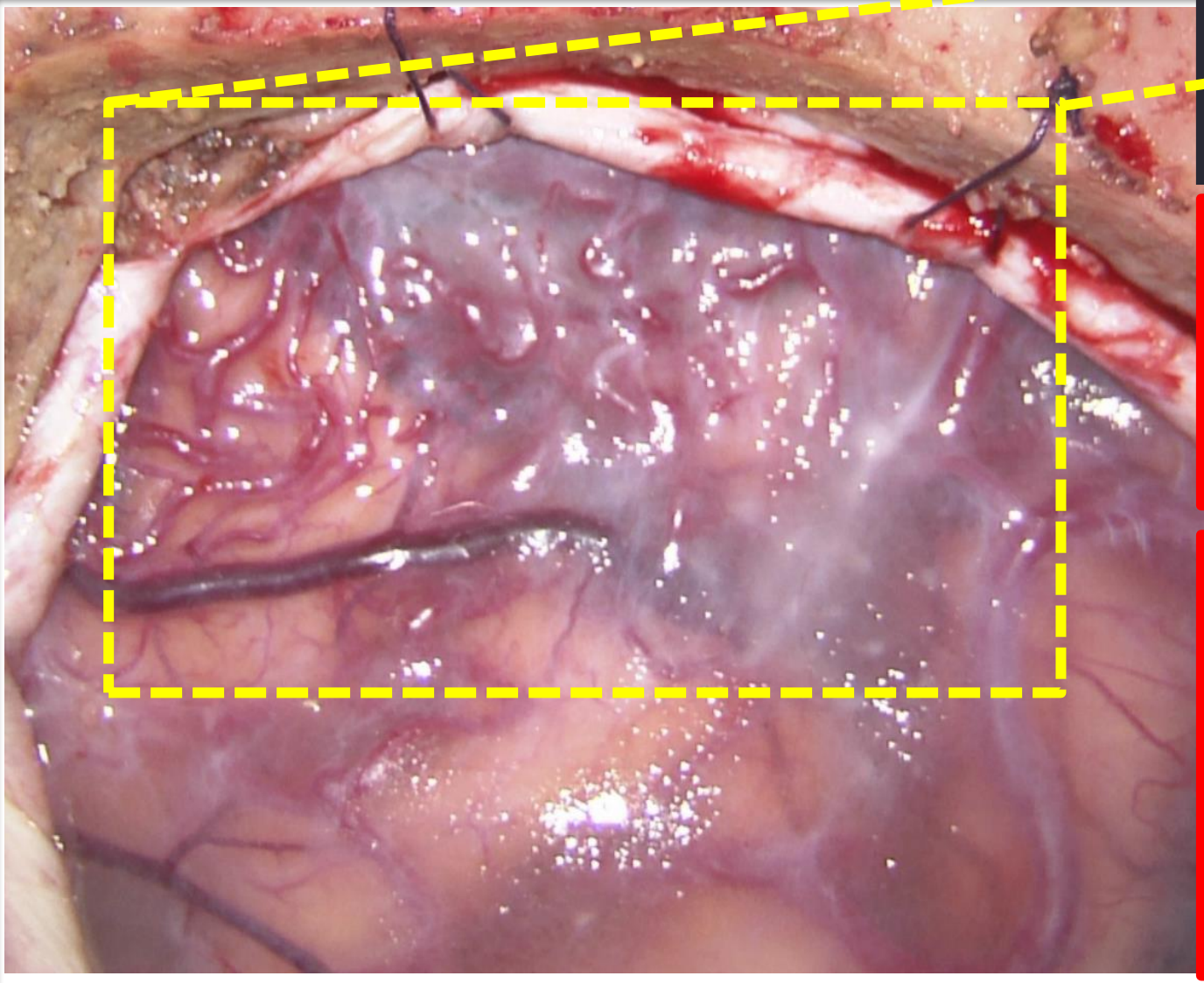


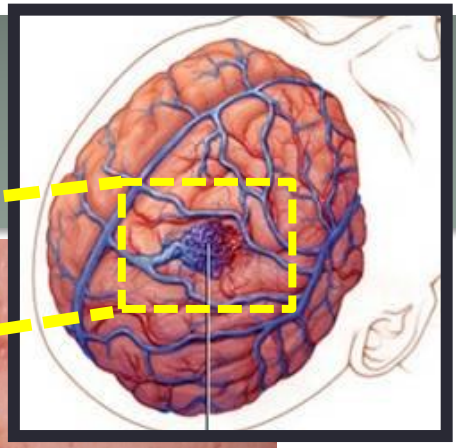


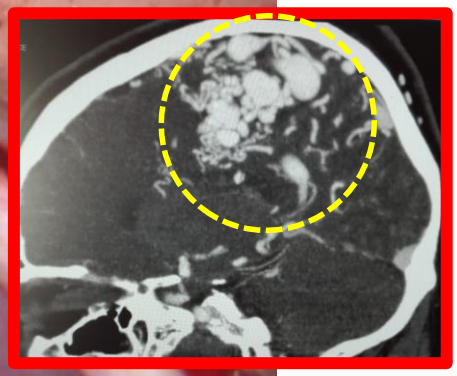




Right parietal AVM

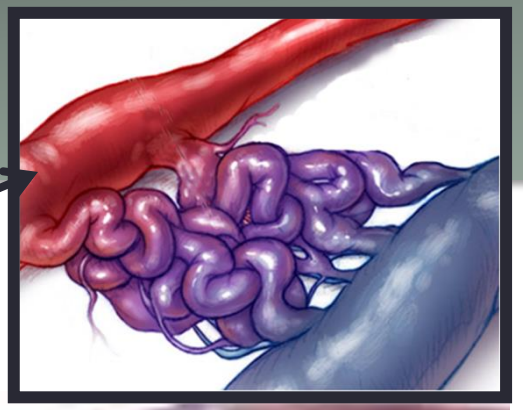




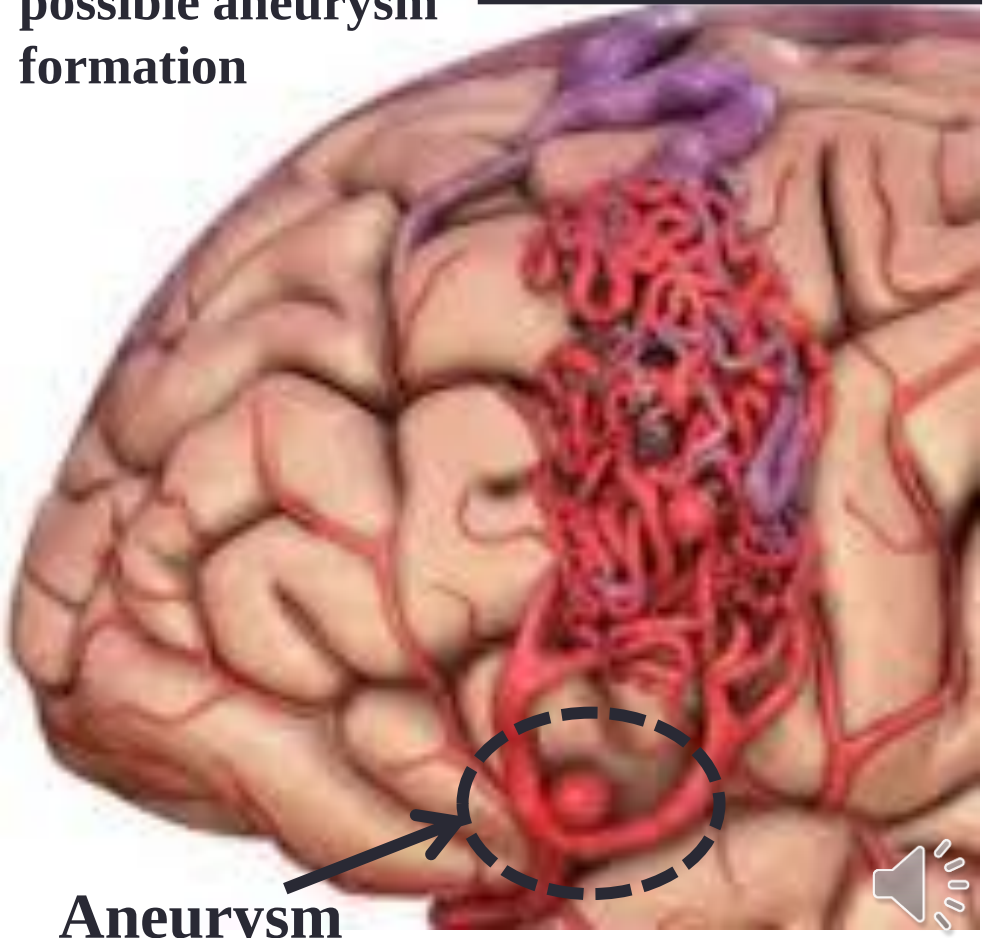


AVM: epidemiology

- Prevalence: 0.14%
- ⇑ frequent in ♂
- Congenital / early adolescence
- Age at diagnosis 30-40 years
  - 3-20% in childhood
- 15-20% Rendu-Osler have AVMs
- 1 AVM / 5.3 aneurysms
- 7% AVMs associated with aneurysms

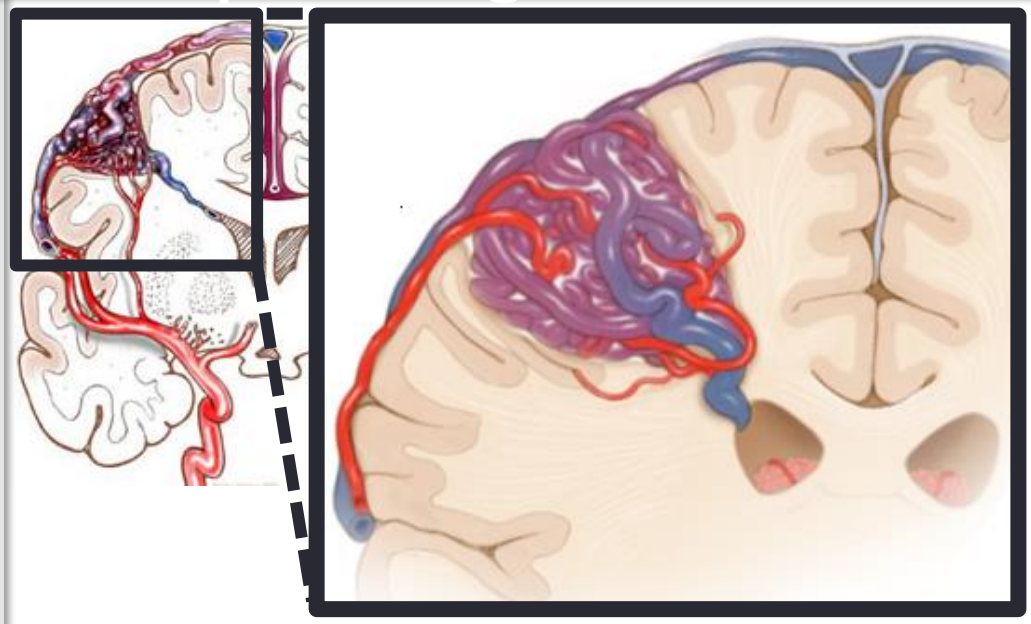


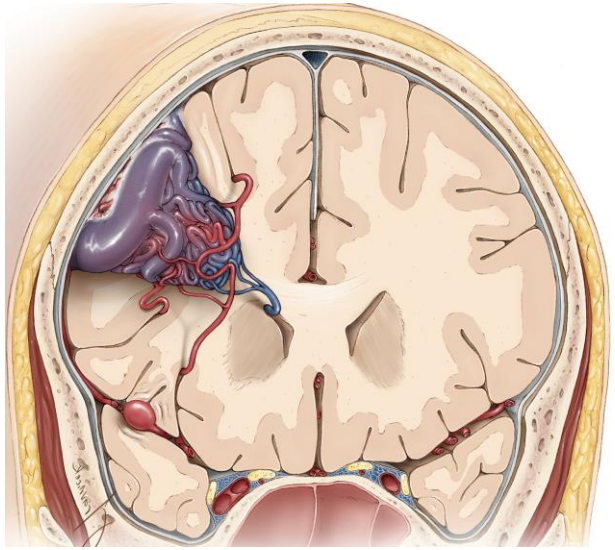
Feeding arteries hypertrophy = possible aneurysm formation

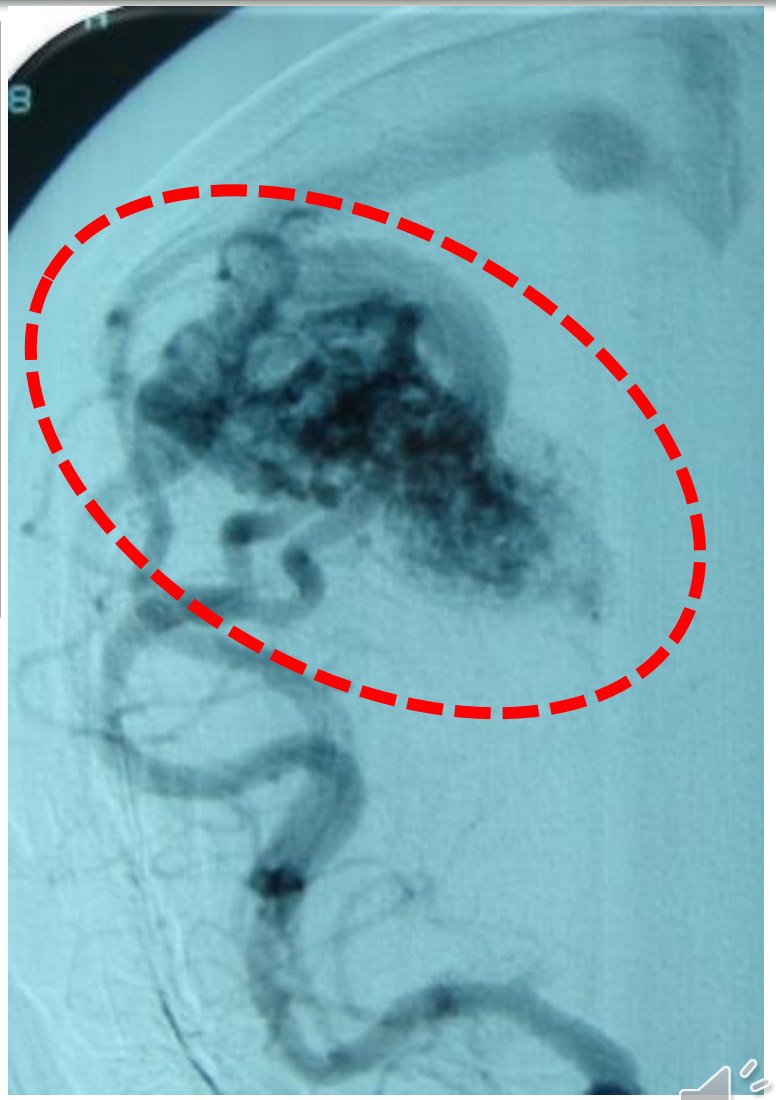


Aneurysm

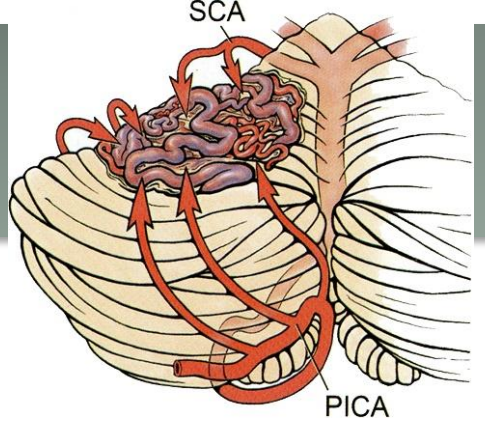
Usual form brain AVM: cone shape with tip facing ventricle



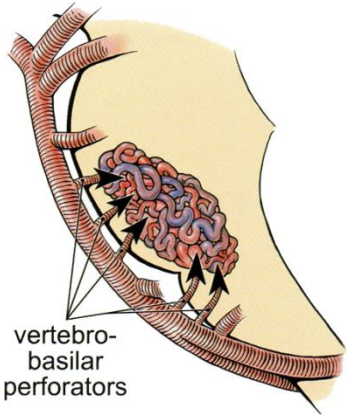




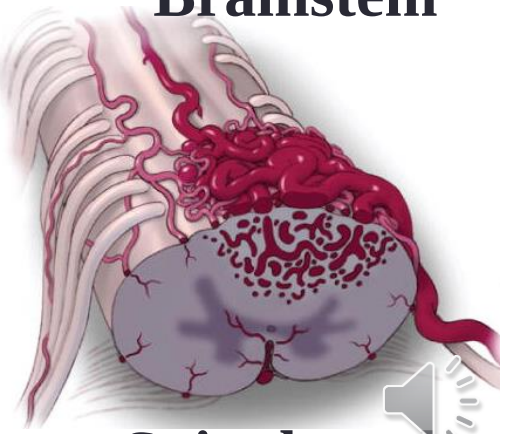
AVM location: anywhere in CNS



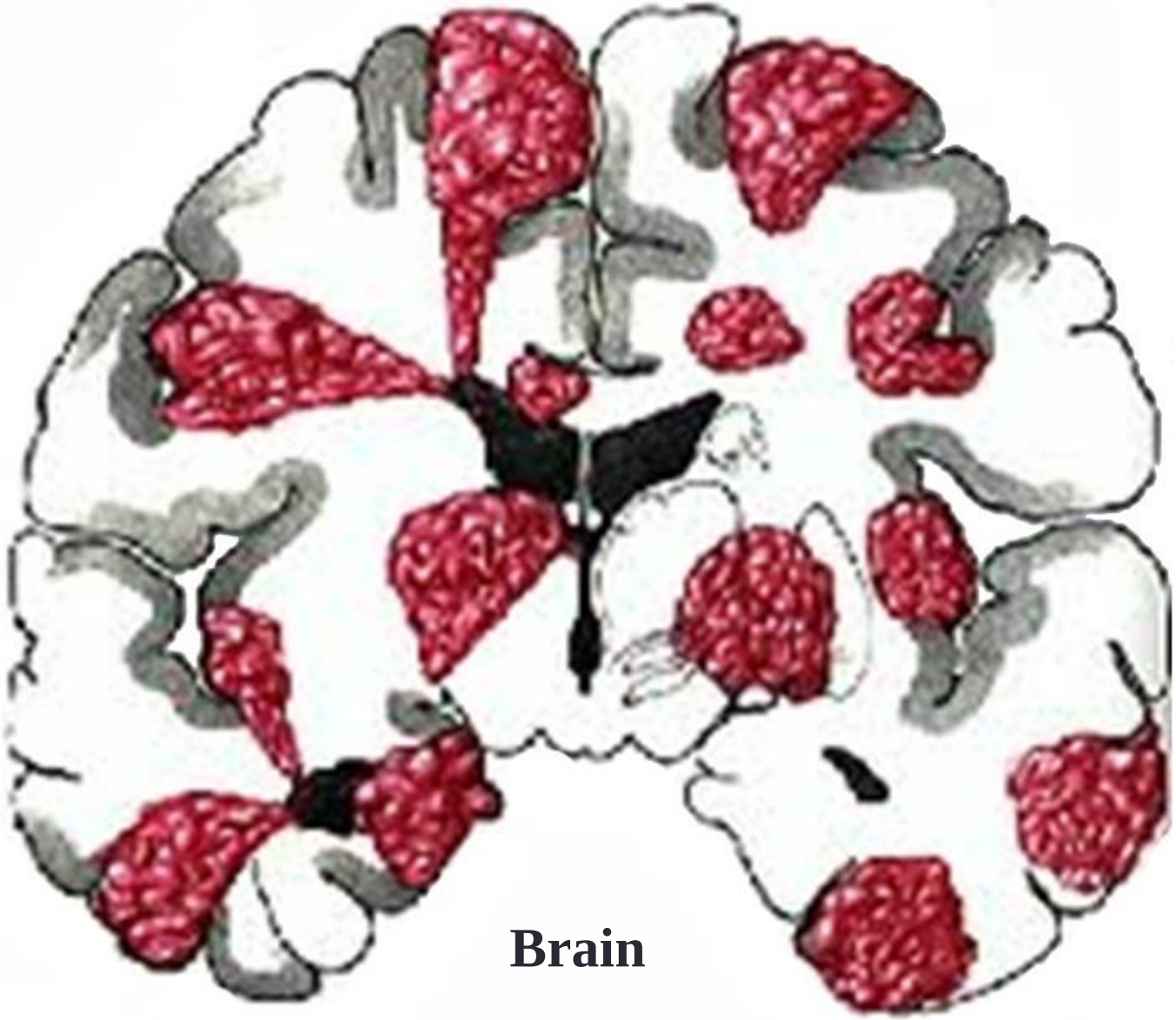
Cerebellum



Brainstem



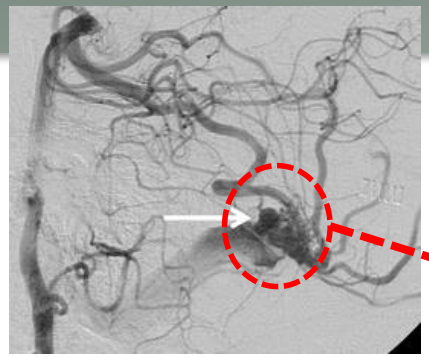
Spinal cord

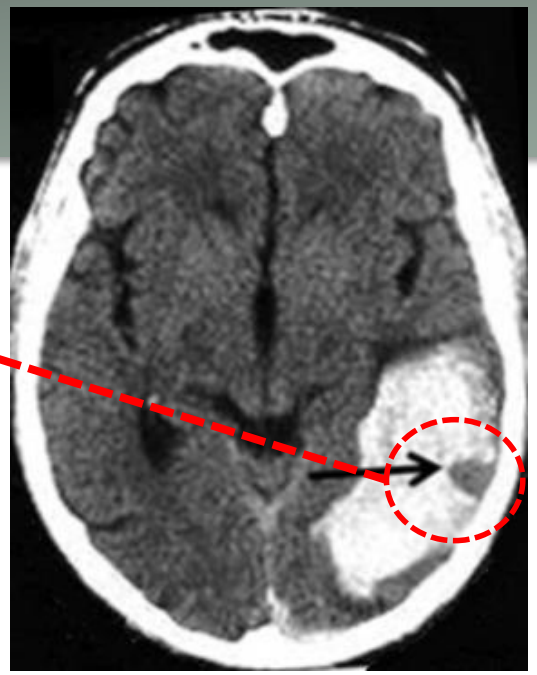


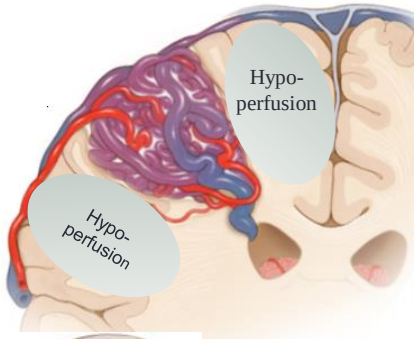
Brain

Clinical presentation

- Bleeding 50%
- Epilepsy 44%
  - ⇑ frequent at a younger age
- Mass effect
- Blood stealing from nearby brain
  - Ischemia = focal deficit
- Headache
- Raised intracranial pressure
  - ↑ venous sinus pressure = deficient CSF reabsorption
- In children: hydrocephalus, macrocephaly, congestive heart failure, large pericranial veins
- Asymptomatic 15%

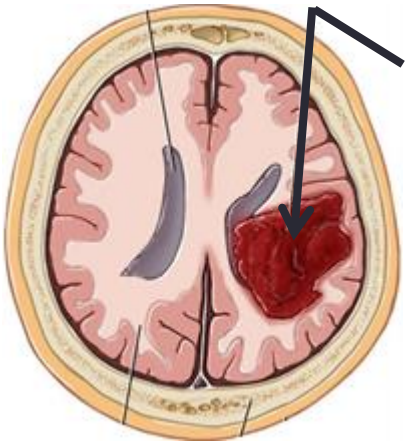


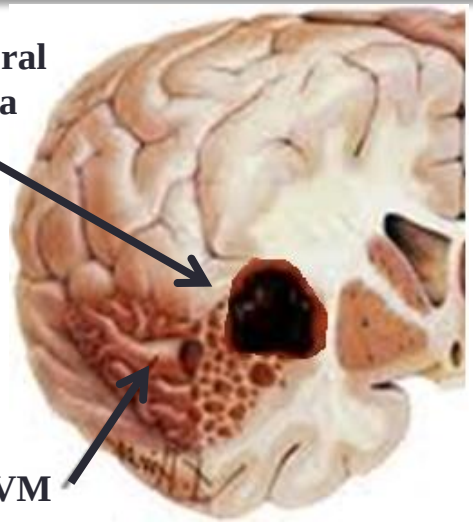


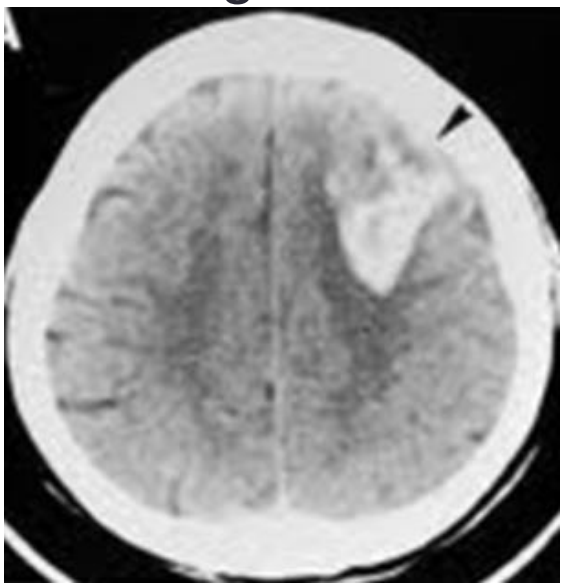


Brain AVM: haemorrhage

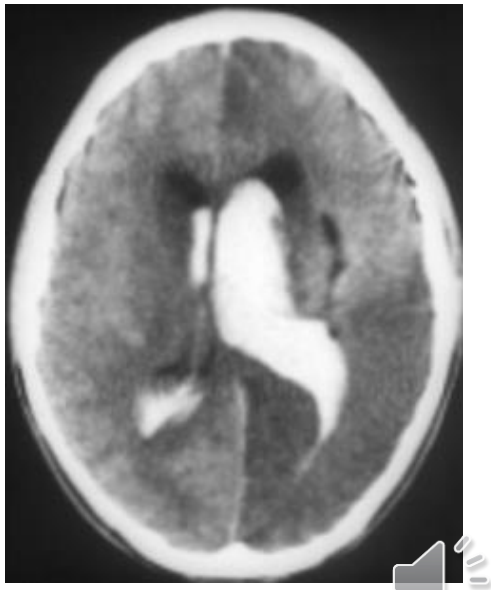
- Peak 15-20 years age
- 10% mortality
- 30-50% morbidity
- 82% intracerebral
- Bleeding risk ≅ 105-age patient







Intracerebral haemorrhage

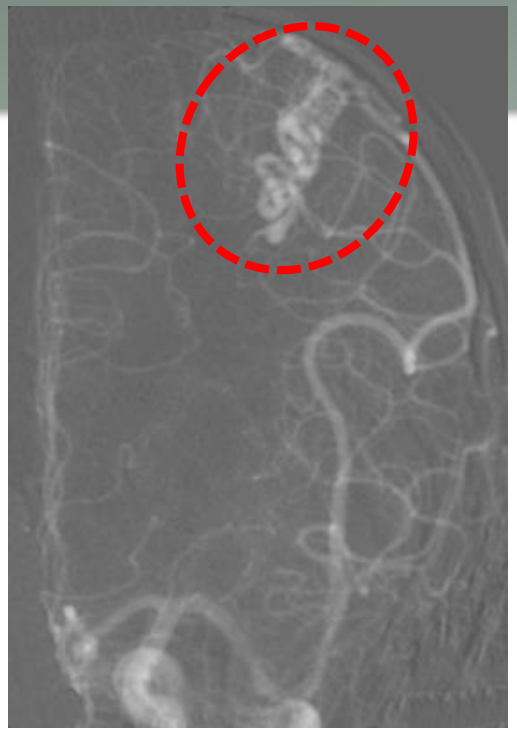


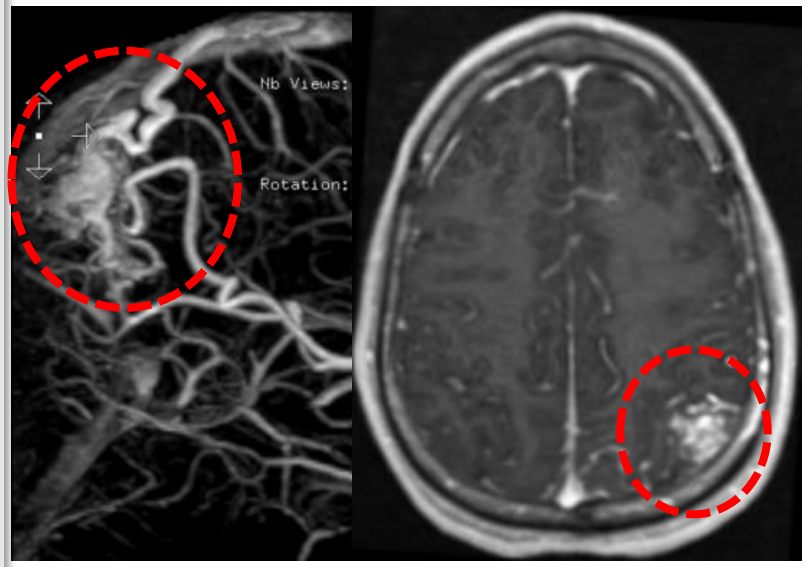
Intraventricular haemorrhage

AVM: neuroimaging

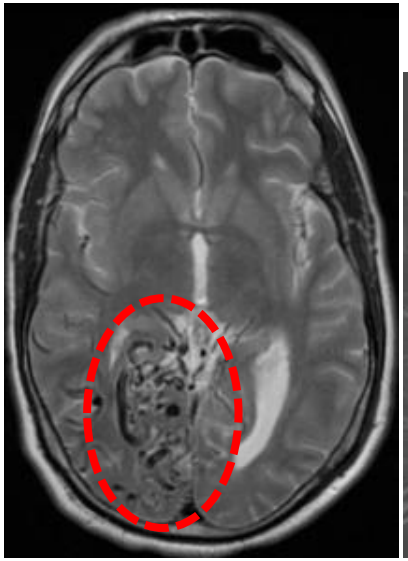
- CT scan
  - Bleeding
  - Calcifications
  - Angio CT
- MRI
- Angiography

CT scan

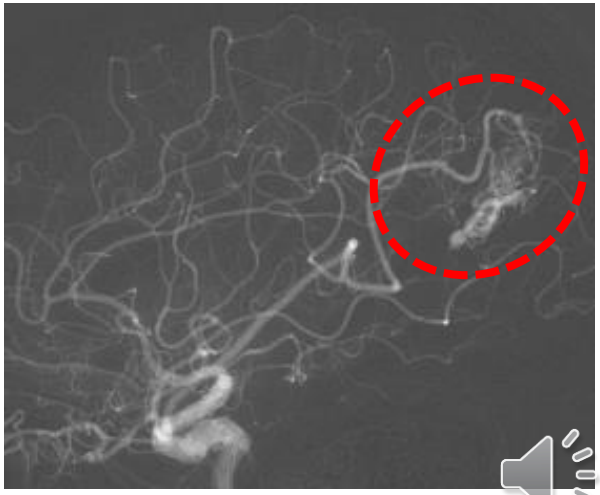




Angio MRI



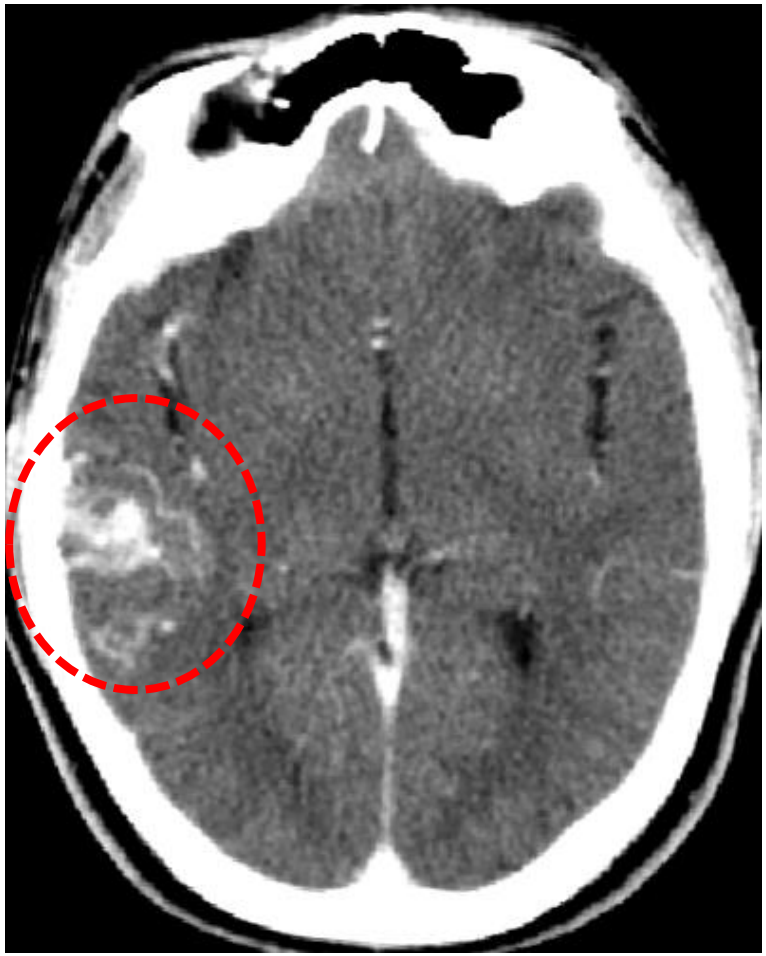
MRI

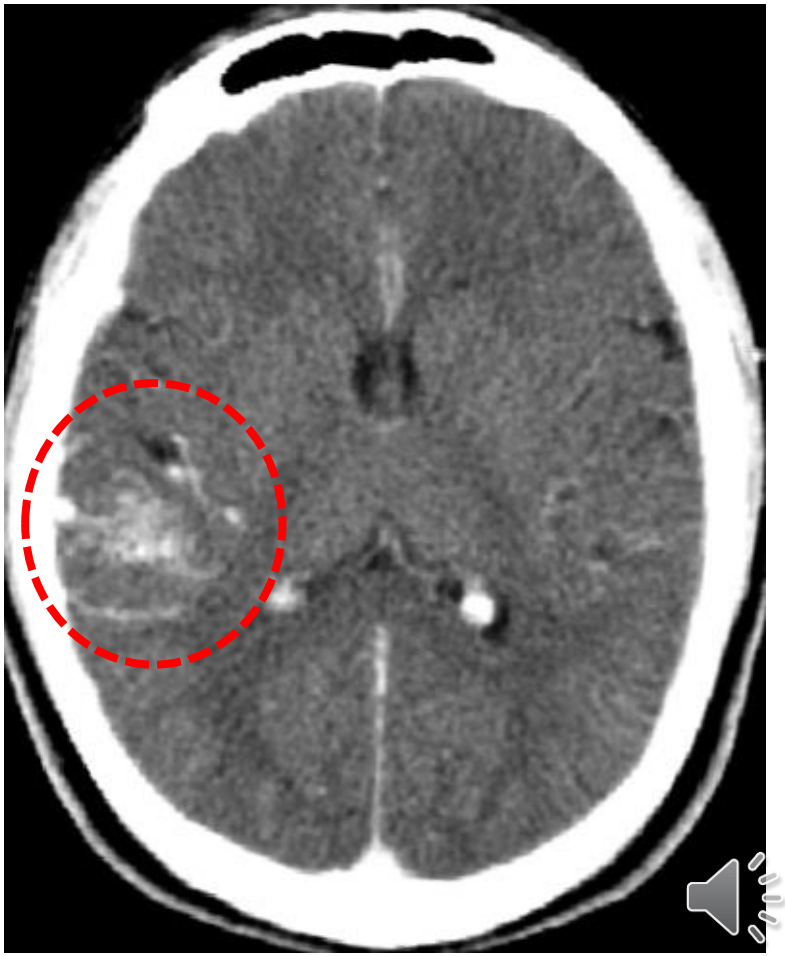


Angiography

Brain AVM: CT scan with contrast

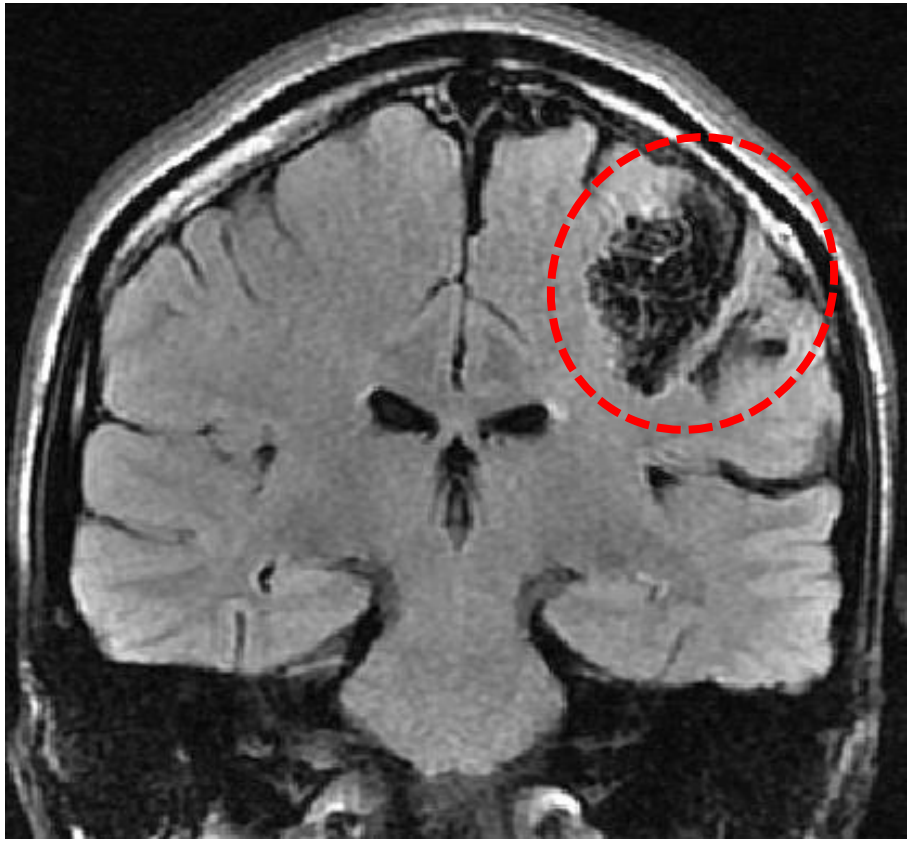
- Shows lesion but no anatomical details
  - Helps to make diagnosis, but not to decide treatment

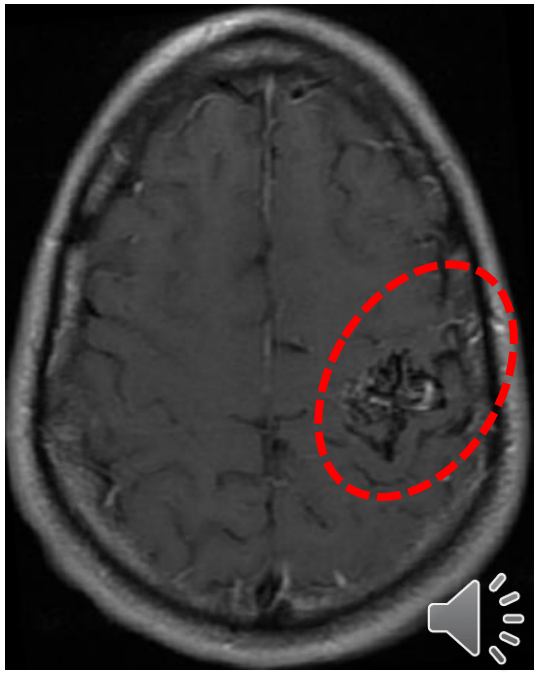




Brain AVM: MRI

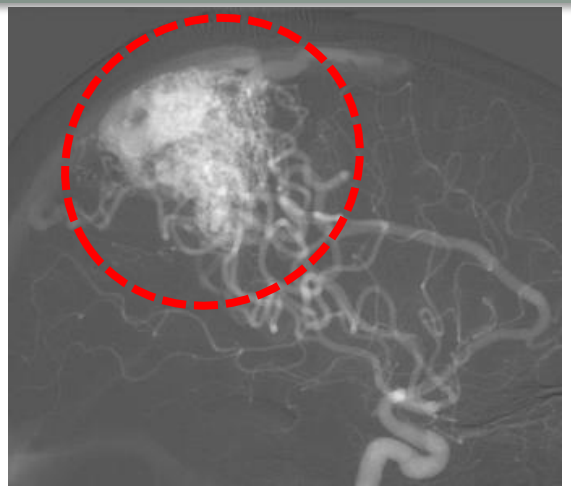
- Good 3D anatomical depiction
- Does not show feeding and draining vessels well

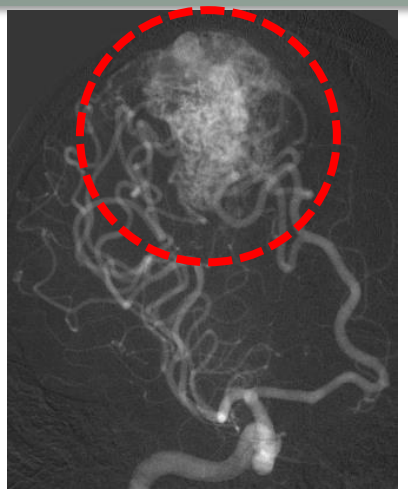


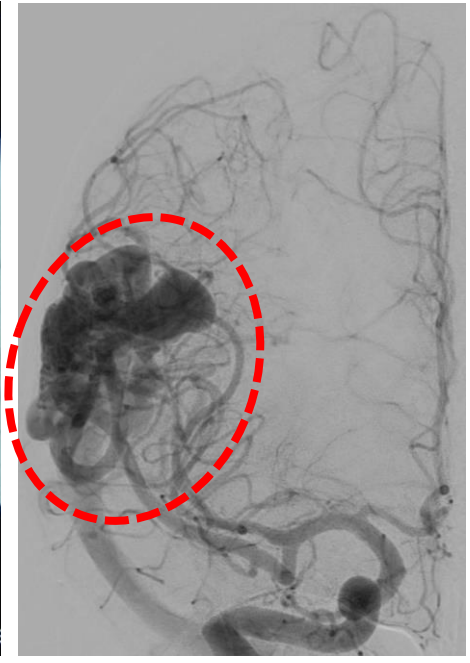


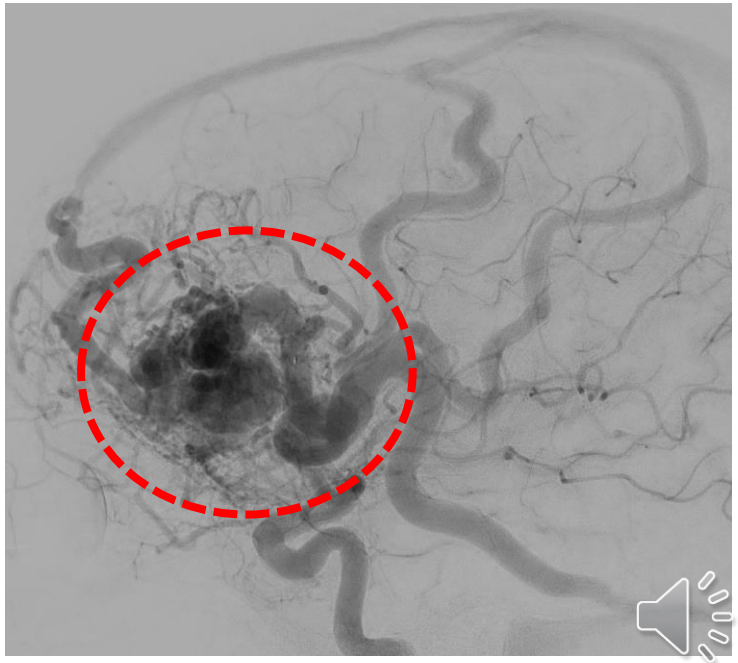
Brain AVM angiography: gold-standard

- Shows
  - Nidus
  - Feeding arteries
  - Draining veins
- Small AVM localization









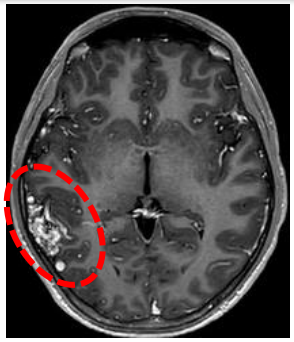
Brain AVM grading: Spetzler-Martin scale

- Internationally accepted

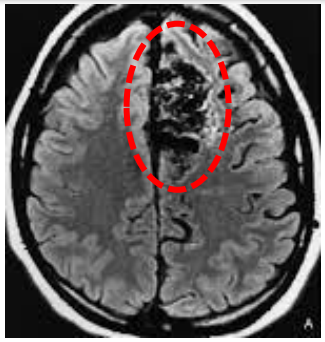
Spetzler-Martin Grading	Points	Supplementary Grading
Size, cm		Age, y
<3	1	<20
3-6	2	20-40
>6	3	>40
Venous drainage		Bleeding
Superficial	0	Yes
Deep	1	No
Eloquence		Compactness
No	0	Yes
Yes	1	No
Total	5	

Brain AVM grading: Spetzler-Martin scale

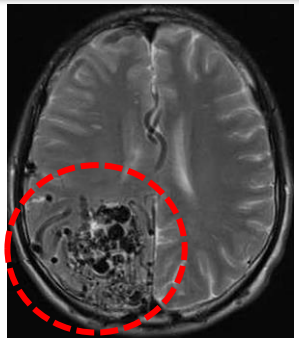
- Size
  - Small < 3 cm = 1
  - Medium 3-6 cm = 2
  - Large > 6 cm = 3
- Location: eloquent brain area
  - No = 0
  - Yes = 1
- Venous drainage
  - Only superficial = 0
  - Deep = 1



Small

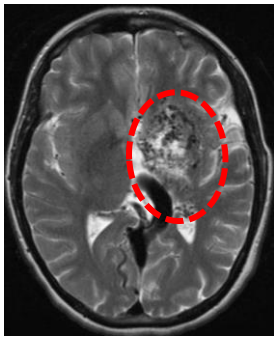


Medium



Large

Motor cortex

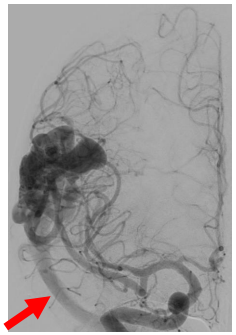


Thalamus

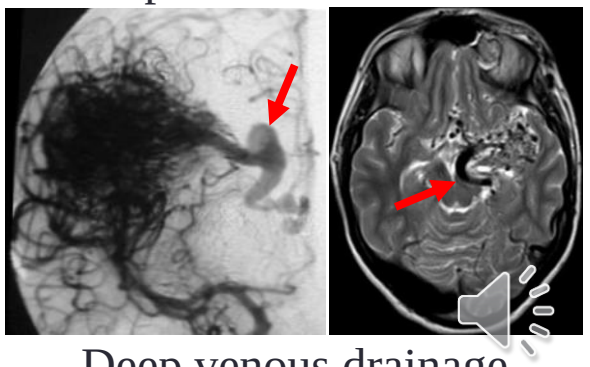
Int. capsule

Brainstem

Eloquent areas: sensorimotor cortex, visual cortex, internal capsule, thalamus, hypothalamus, brain stem, and cerebellum nuclei

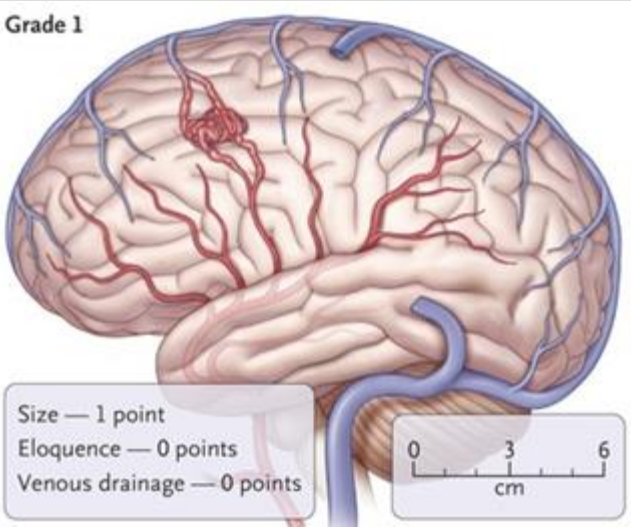


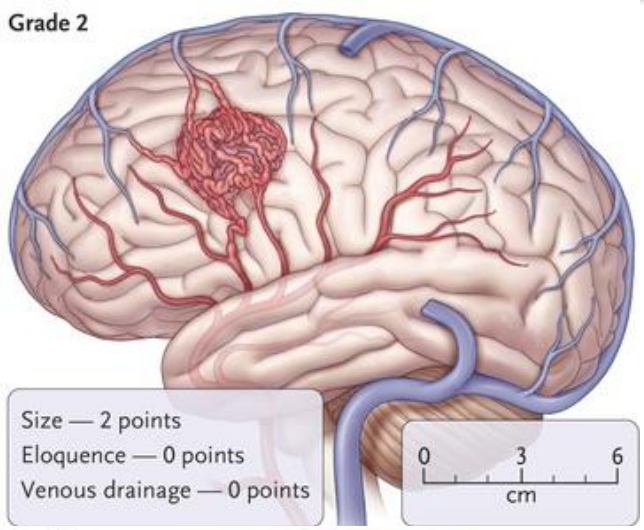
Superficial

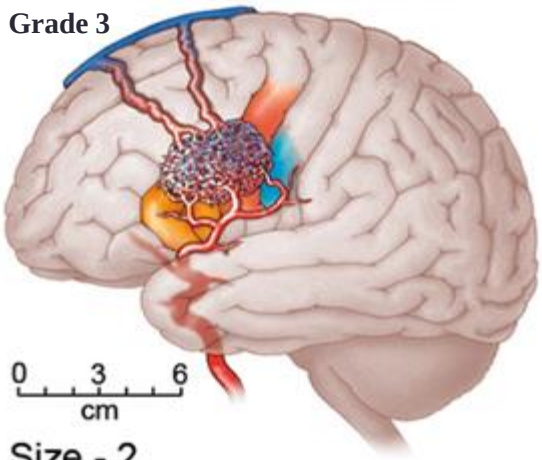


Deep venous drainage

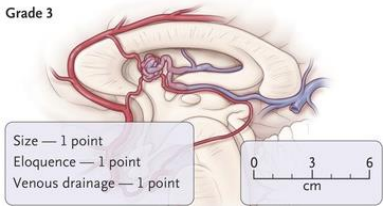
Brain AVM: Spetzler-Martin scale

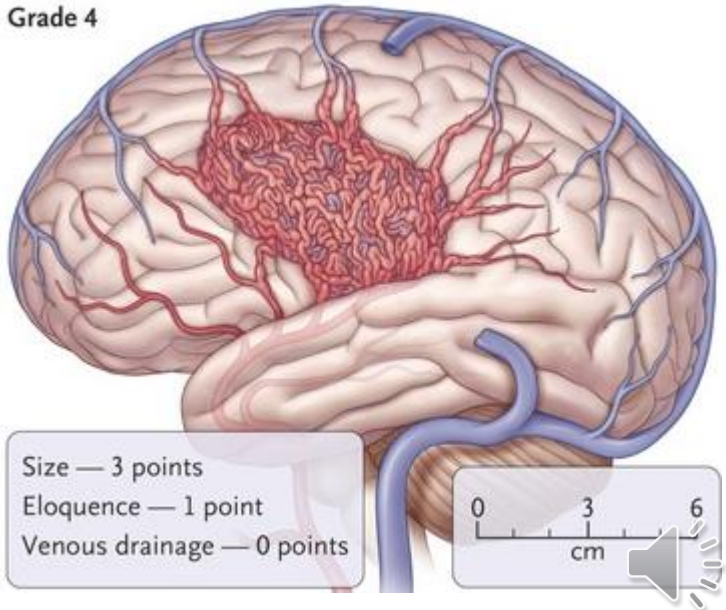






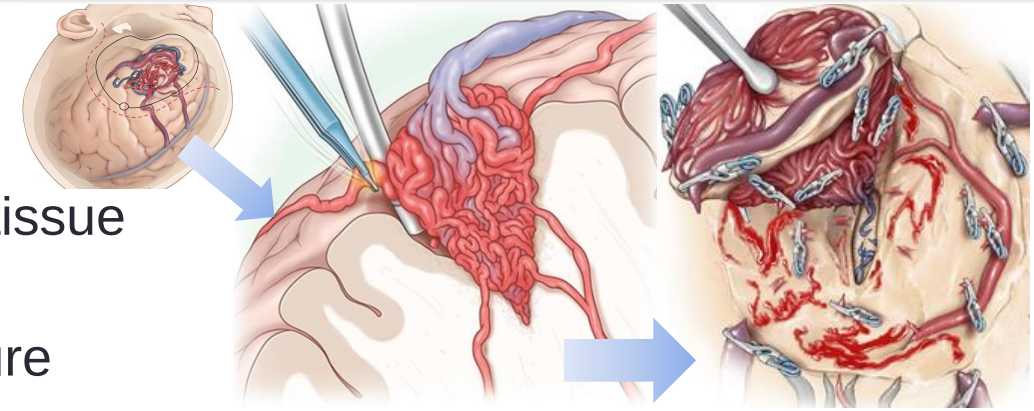
Size - 2
Eloquence - 1
Venous drainage - 0





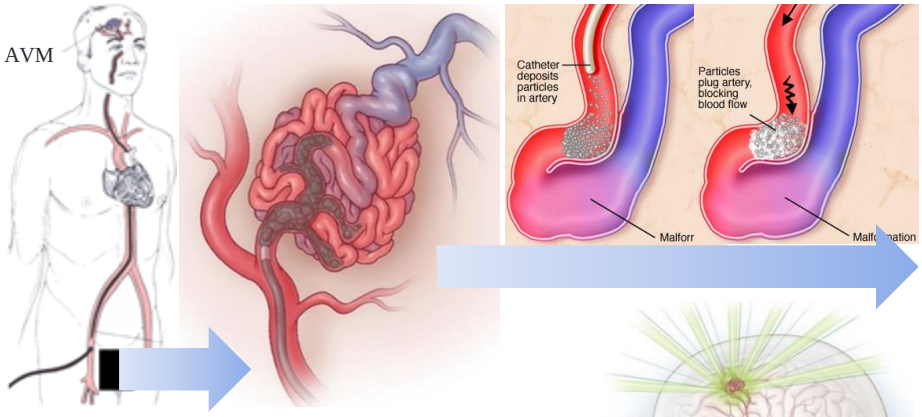
Brain AVM: treatment (1)

- **Surgical removal**
  - Requires craniotomy
  - Possibility of normal nerve tissue damage
  - Definitive and immediate cure



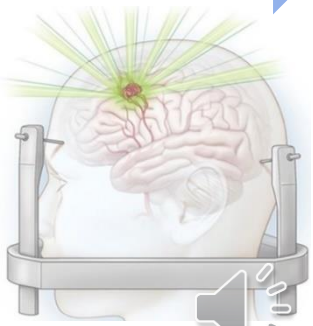
Surgery

- **Endovascular treatment**
  - Rarely curative
  - Reduces AVM size
    - Helps make AVM amenable to other treatment modalities



Endovascular

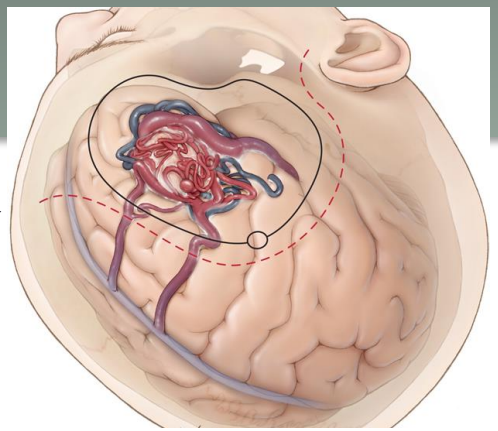
- **Radiosurgery**
  - AVM < 3 cm ∅
  - Minimum one year delay before AVM occlusion = risk of bleeding

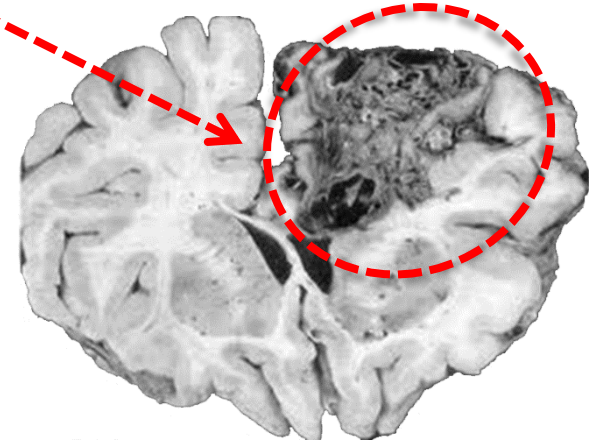


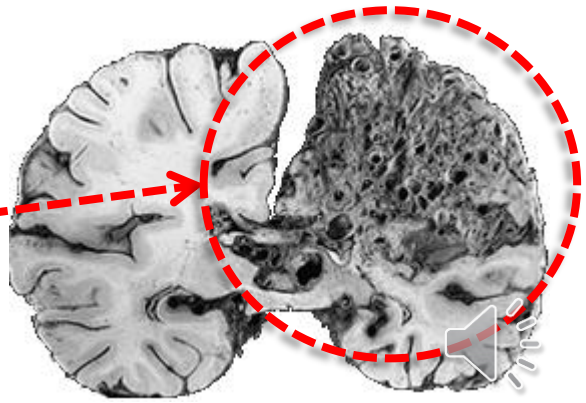
Radiosurgery

Brain AVM: treatment (2)

- Surgery: grades 1 & 2
- Endovascular: grade 3
  - Complete obliteration 15%
  - Usually more than one treatment required
  - Prepares for surgical treatment ± radiosurgery
- Radiosurgery: < 3cm ∅
- Combination of all: grades 4-5

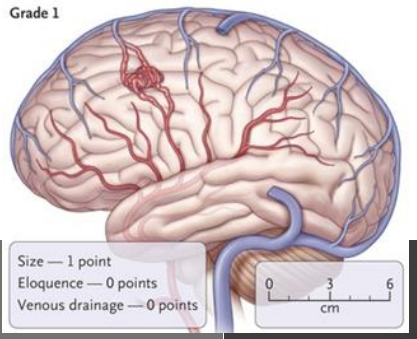






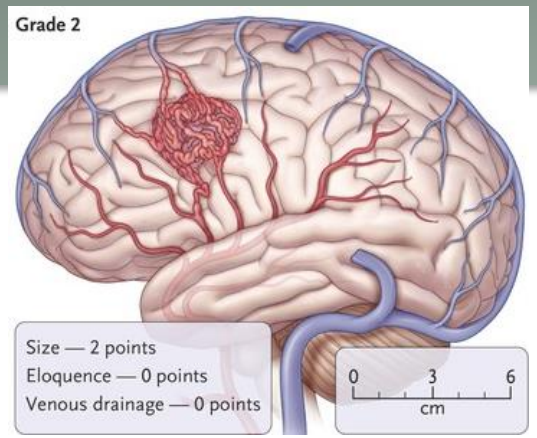
Spetzler-Martin grade 1

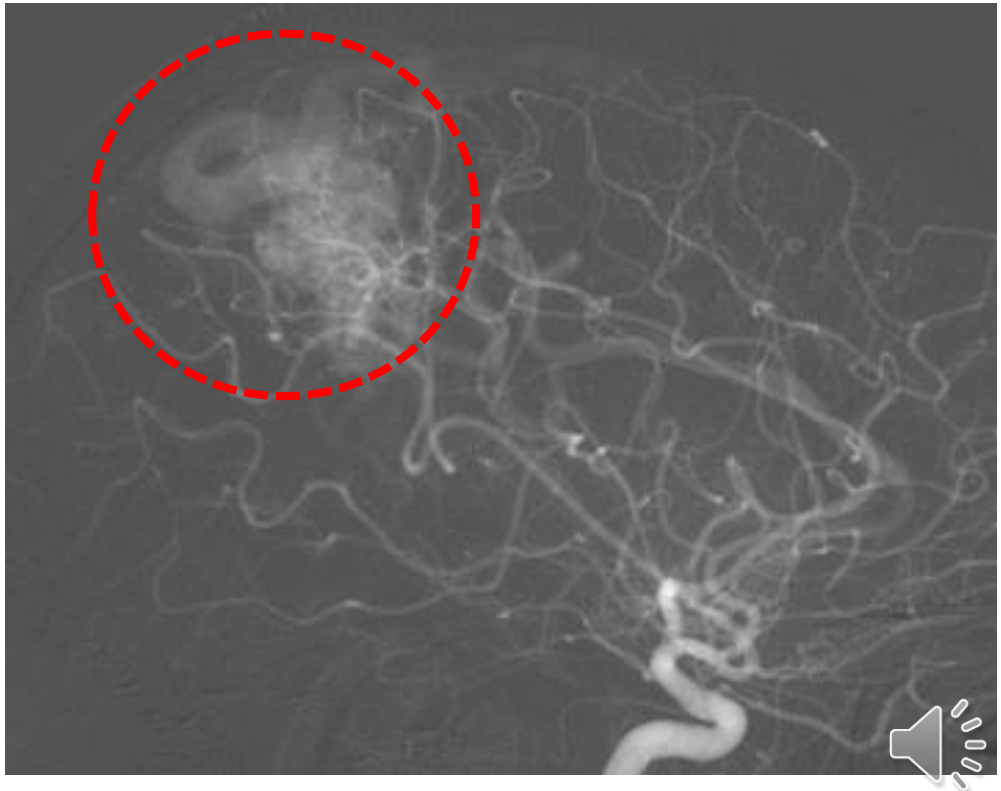
- Treatment: surgery



Spetzler-Martin grade 2

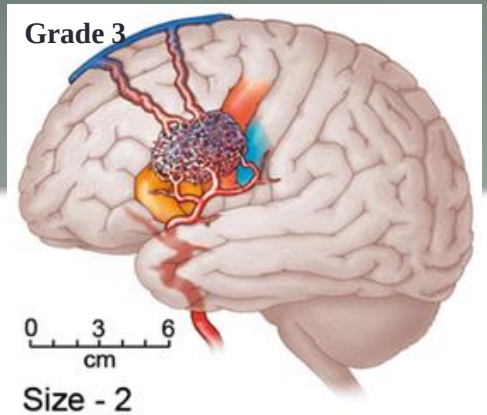
- Treatment: surgery

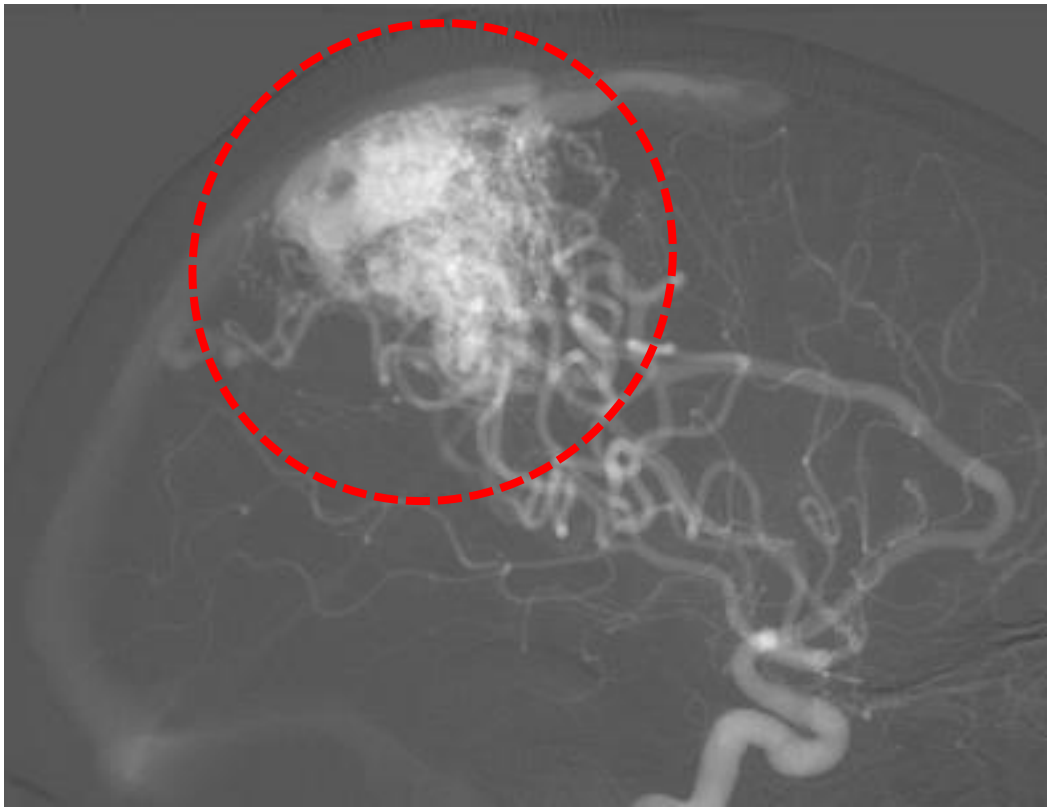




Spetzler-Martin grade 3

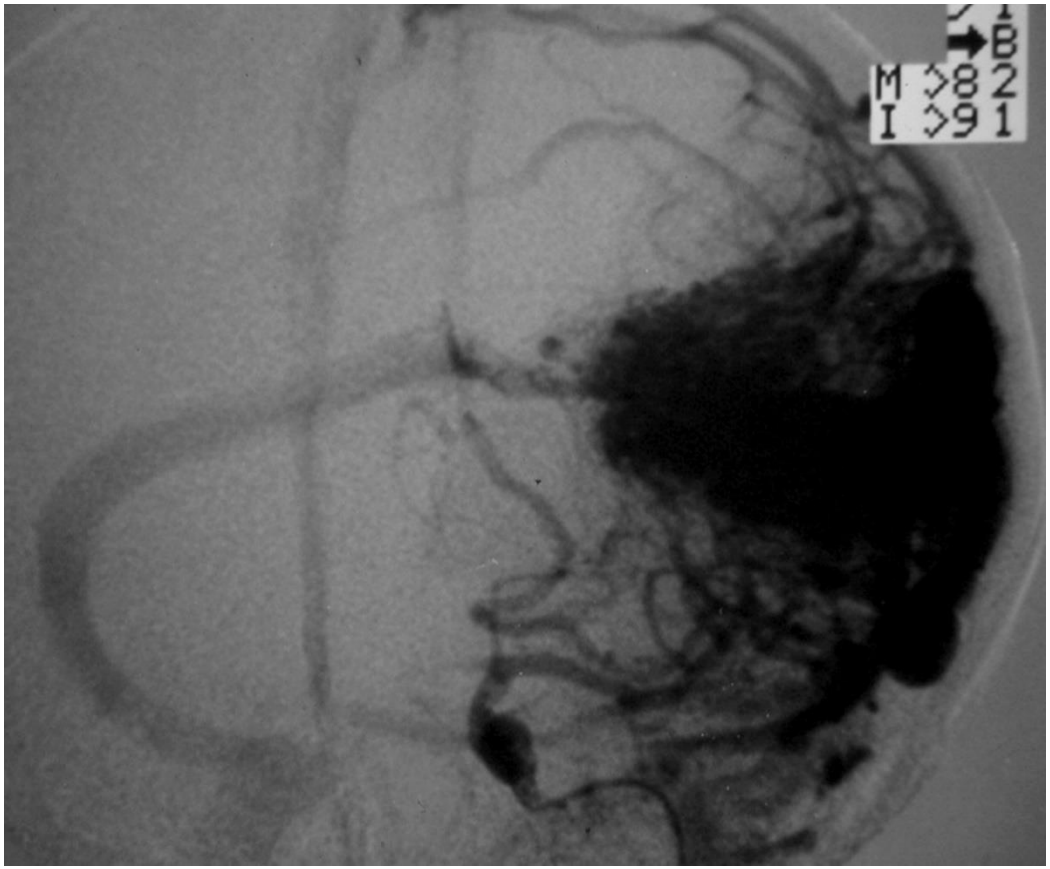
- Embolisation followed by surgery

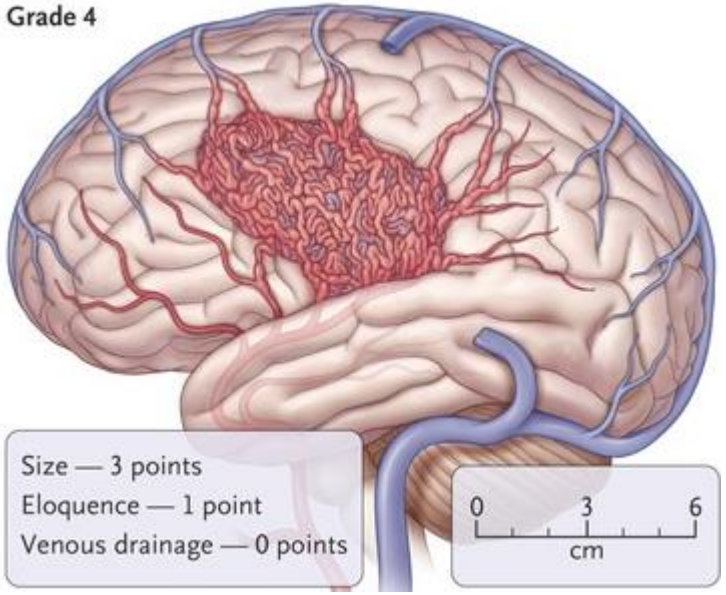




Spetzler-Martin grade 4

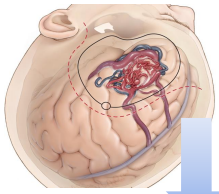
- Combined treatment embolisation / radiosurgery / surgery

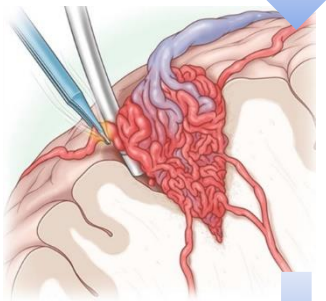


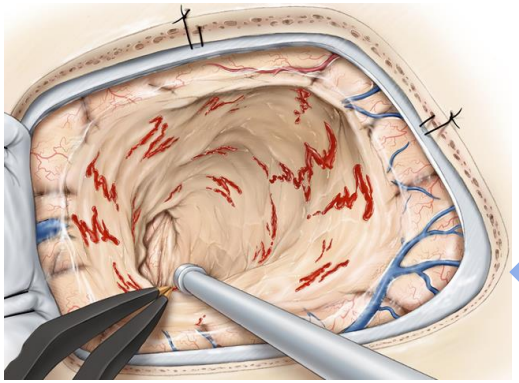


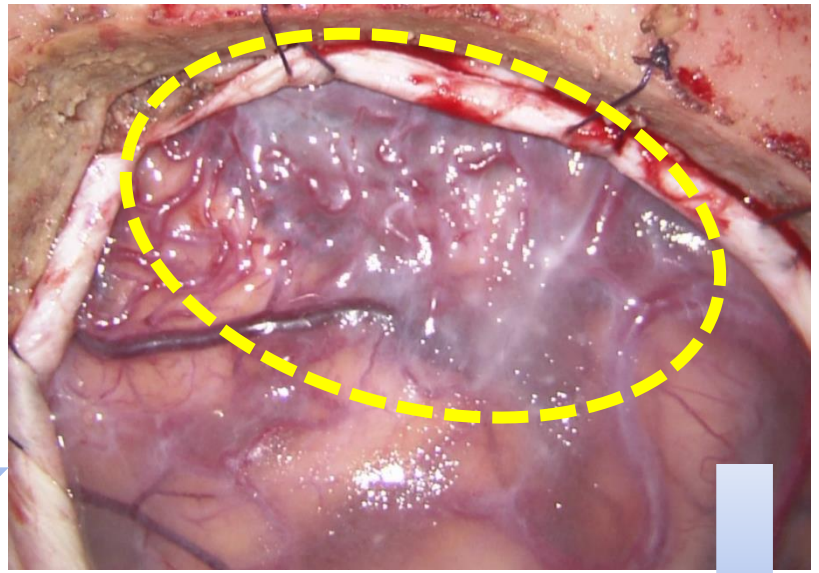
Brain AVM Surgical treatment

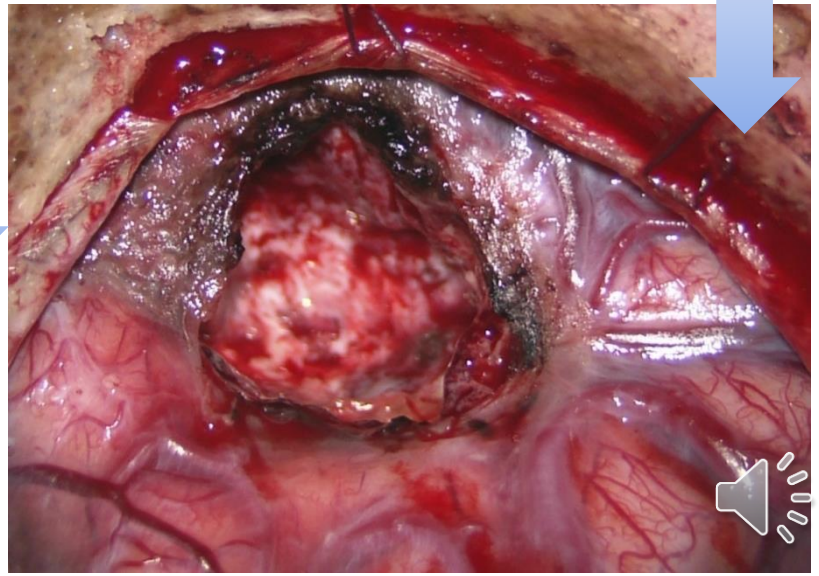
- First choice treatment when indicated
- Eliminates bleeding risk immediately
- Improves epilepsy
- Invasive
- Risks of surgery





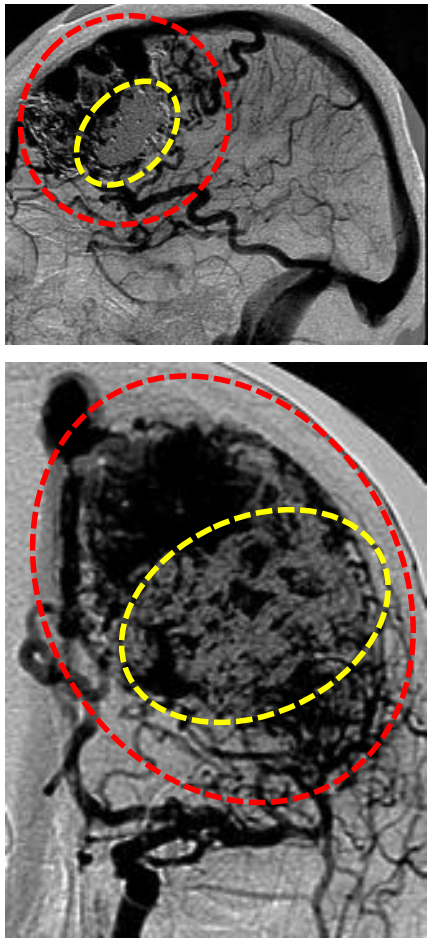




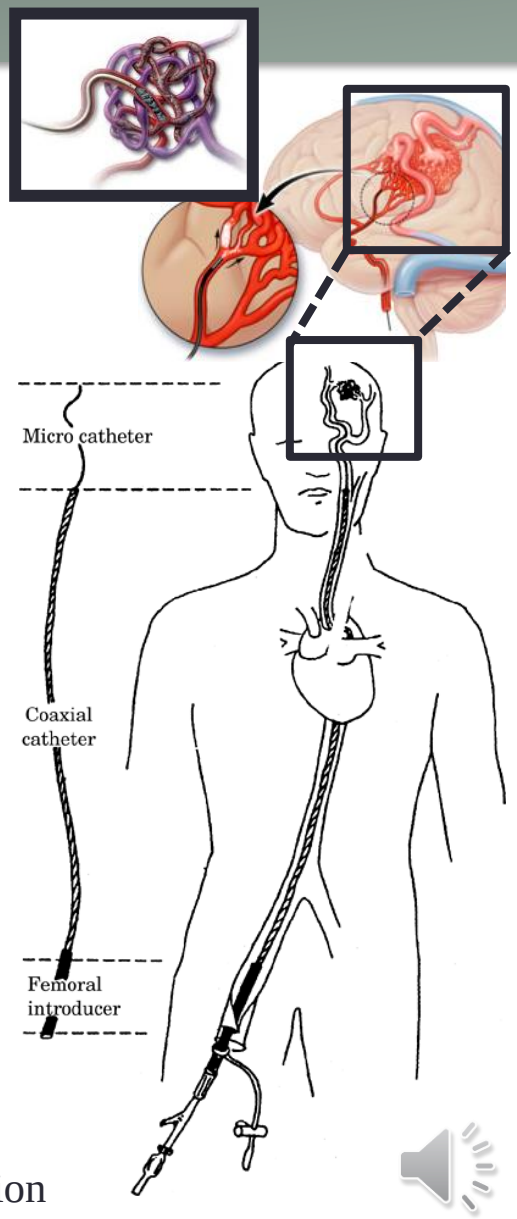


Brain AVM: endovascular treatment

- Preparation 3-30 days BEFORE surgery
- NOT recommended as solo and definitive treatment of AVMs
  - Only 15% complete occlusion
- Risks:
  - Death 2%
  - Neurological deficit
    - Severe 1.5%
    - Moderate 9%
    - Transitory 11%
  - Intra-op bleeding 3%
  - Spontaneous rebleeding 7%

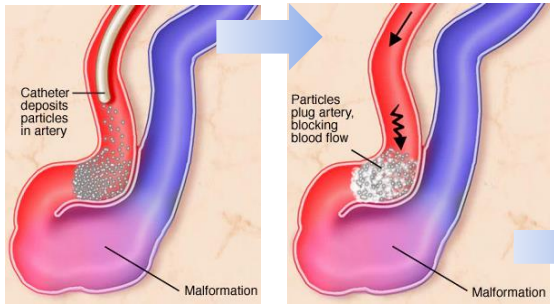


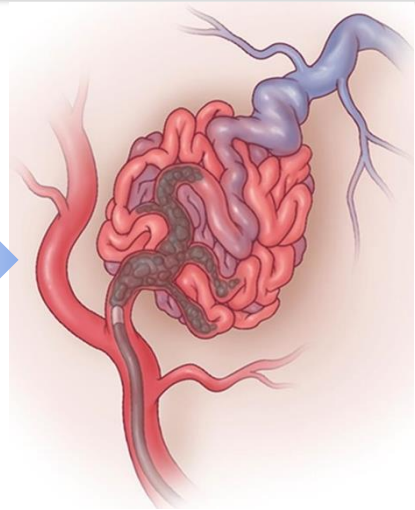
Red shows AVM, yellow shows embolised AVM portion

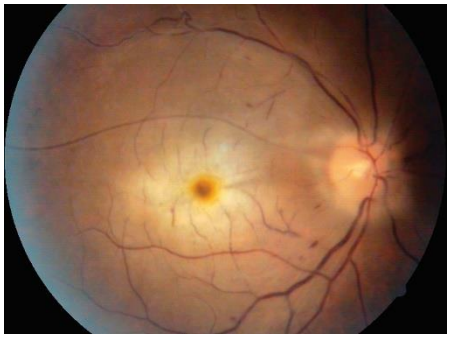


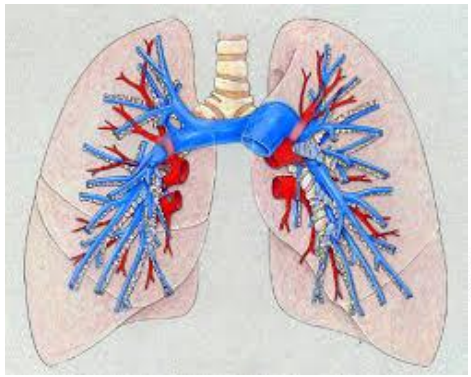
Brain AVM: further risks endovascular treatment

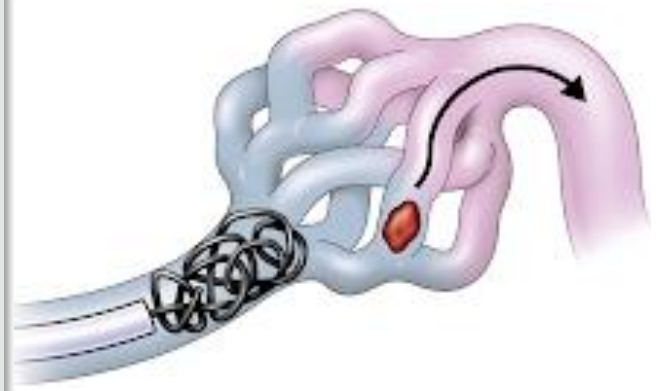
- Brain infarction in areas irrigated by vessels shared with AVM
- Distal embolisation
  - Retina
  - Lung

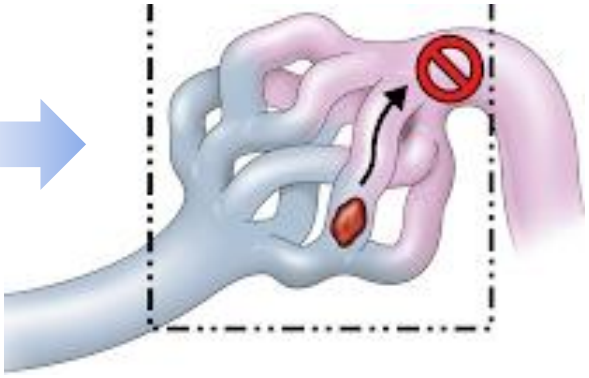


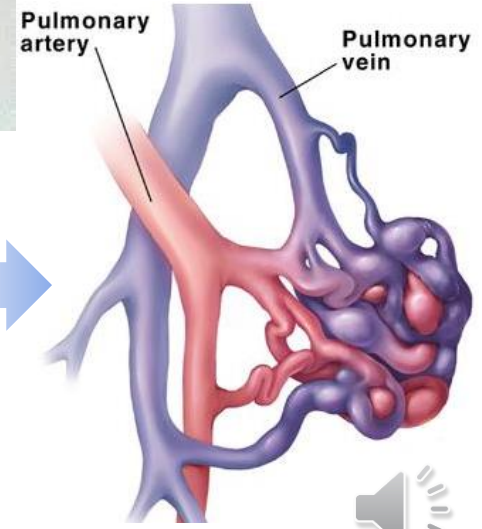








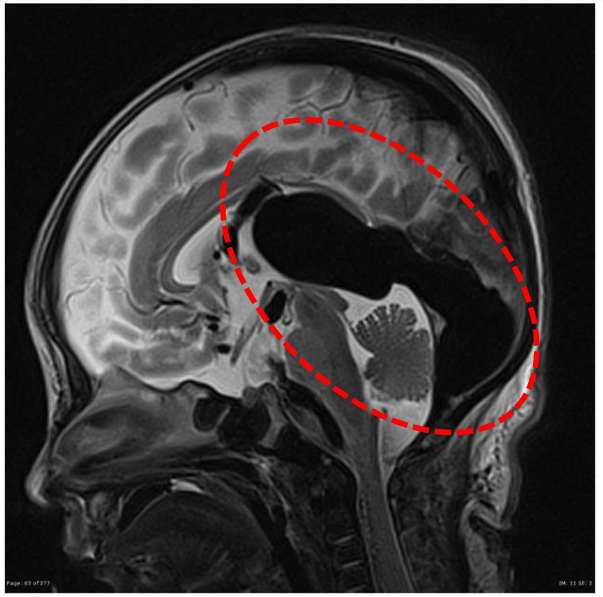


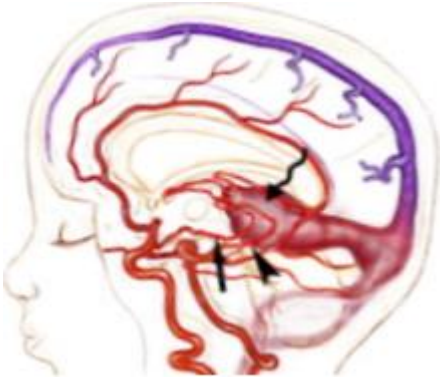


Vein of Galen AVM

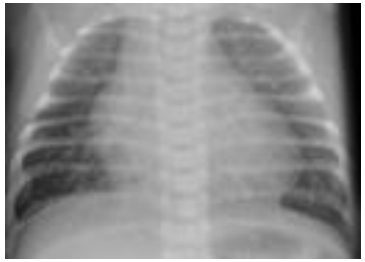
- Newborn
- Massive arterio-venous fistula → congestive heart failure
- Hydrocephalus
- Mental retardation
- Treatment: endovascular

Hydrocephalus

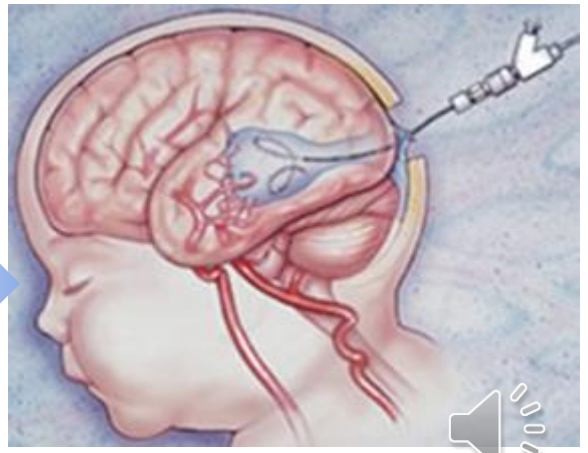




Vein of Galen AVM



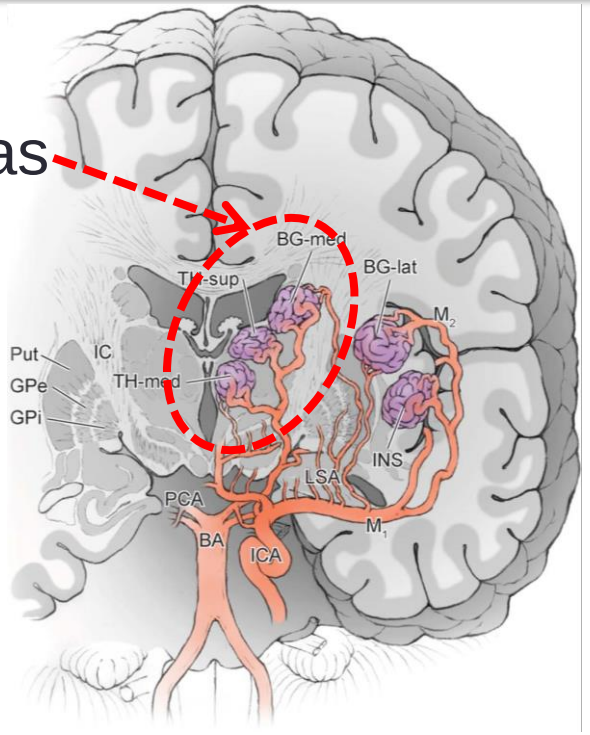
Congestive heart failure

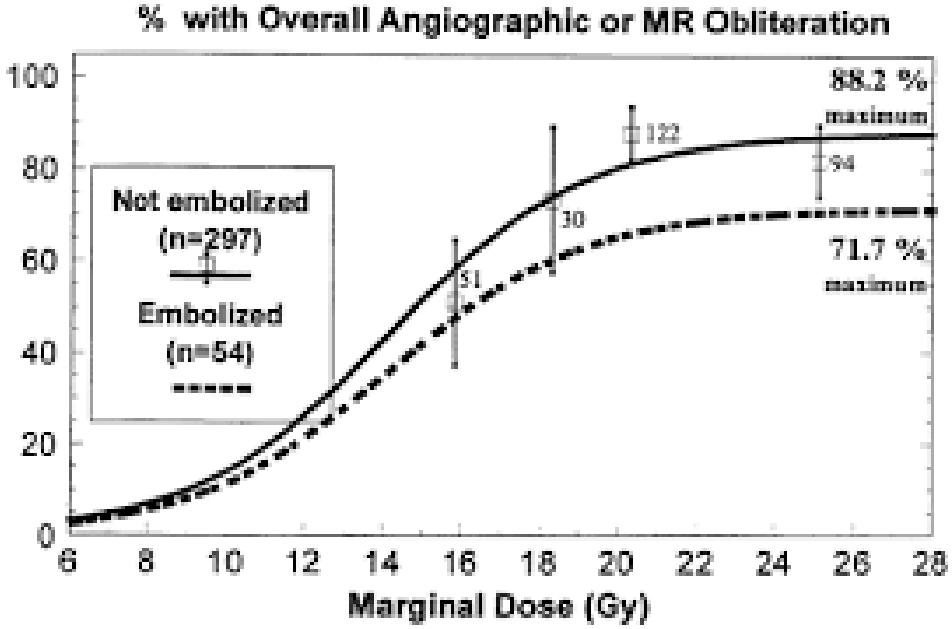


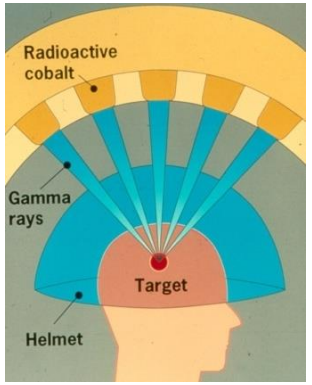
Endovascular treatment

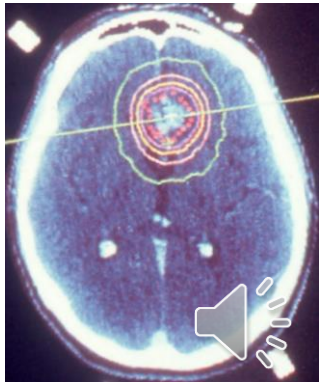
Brain AVM treatment: radiosurgery

- For AVM < 3 cm ∅
- Indicated in AVM in deep or eloquent areas
- Previous embolisation = worse results
- Definitive AVM closure takes 1-2 years
  - Meanwhile = risk of bleeding



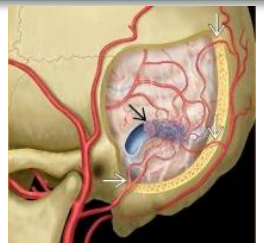


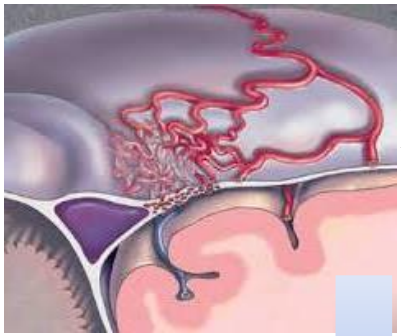


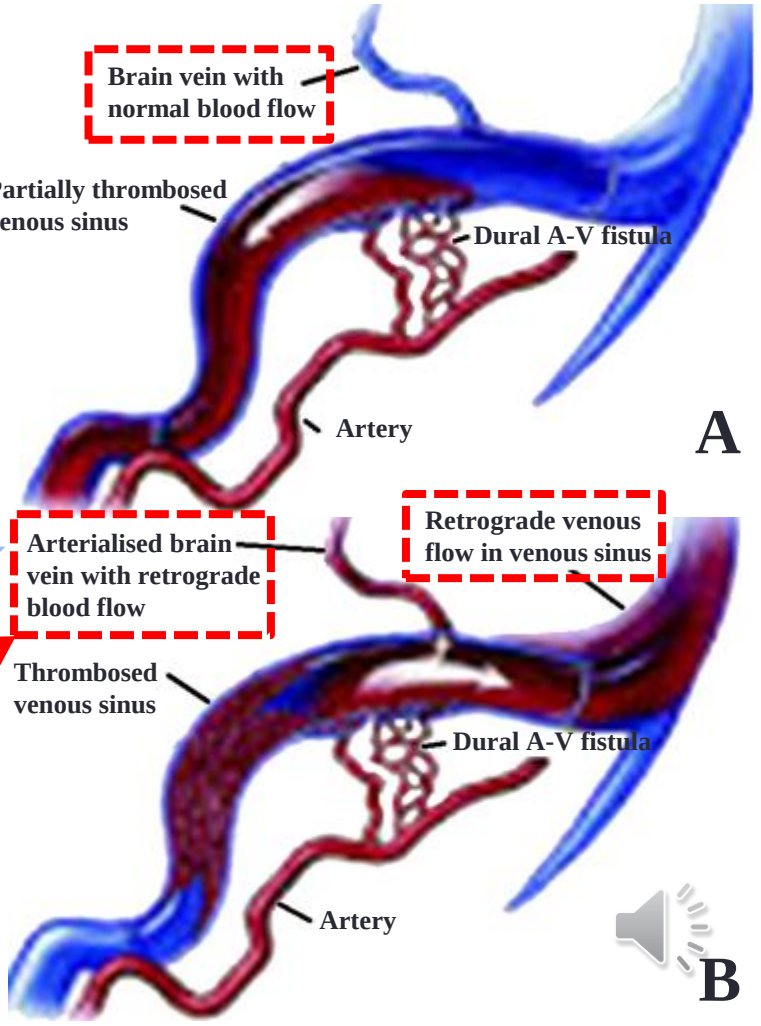


Brain dural arteriovenous (A-V) fistula

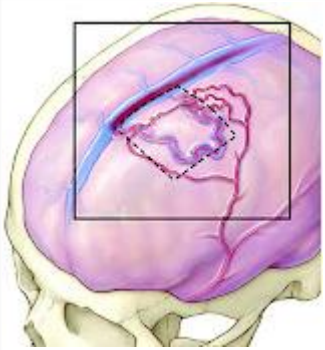
- Nonspecific clinical picture
  - Headache
  - Tinnitus
  - Head noises
  - Exophthalmos
  - Conjunctival injection
- Diagnosis: angiography
  - MRI does not always show lesion
  - CT scan only shows bleeds
- Classification by grades
  - If venous drainage to the brain ⇑ risk of bleeding

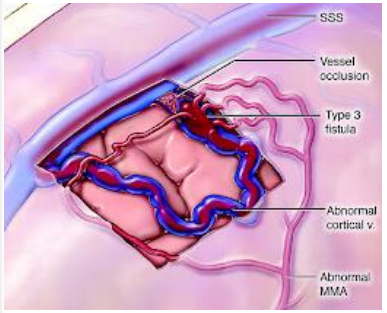


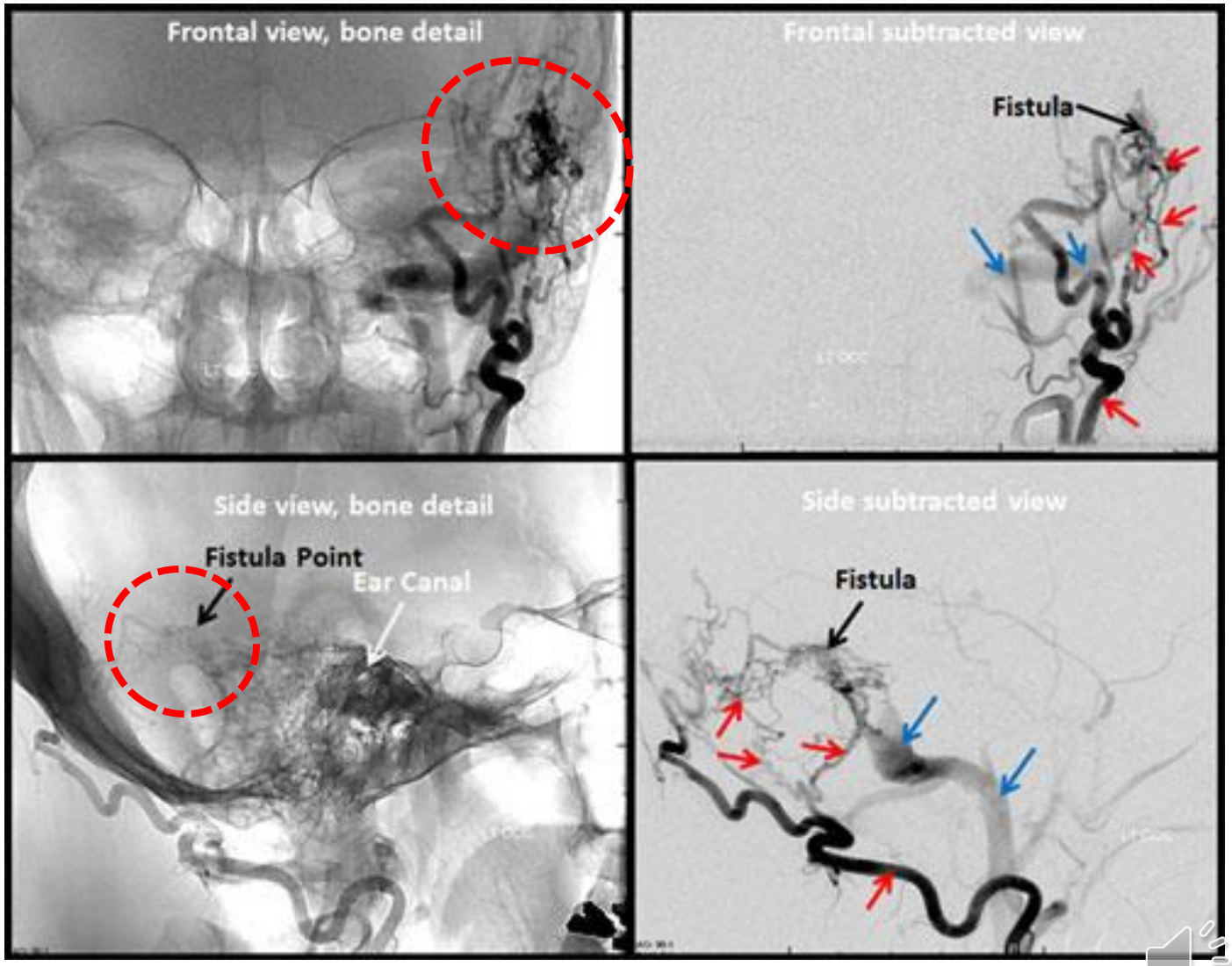




Brain dural A-V fistula diagnosis: angiography

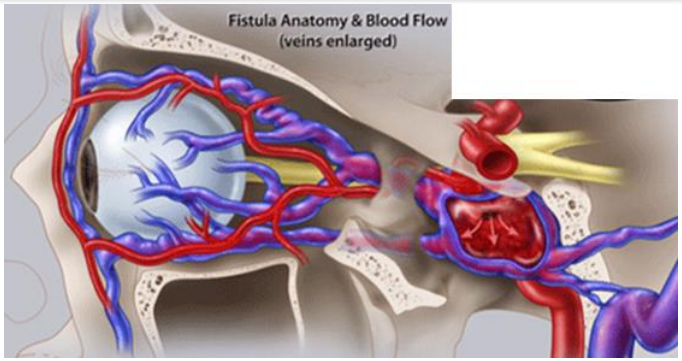






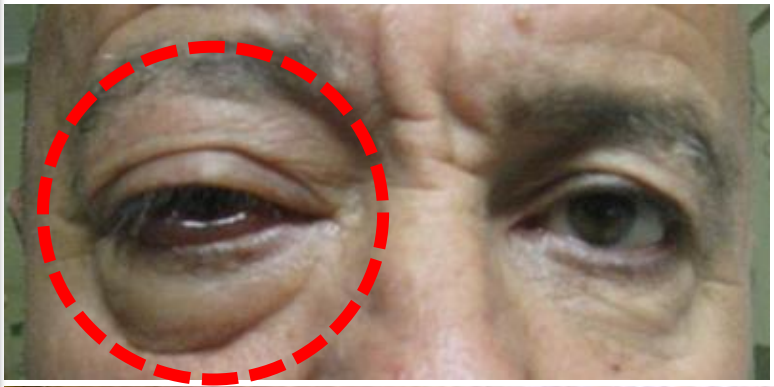
Spontaneous carotid-cavernous fistula

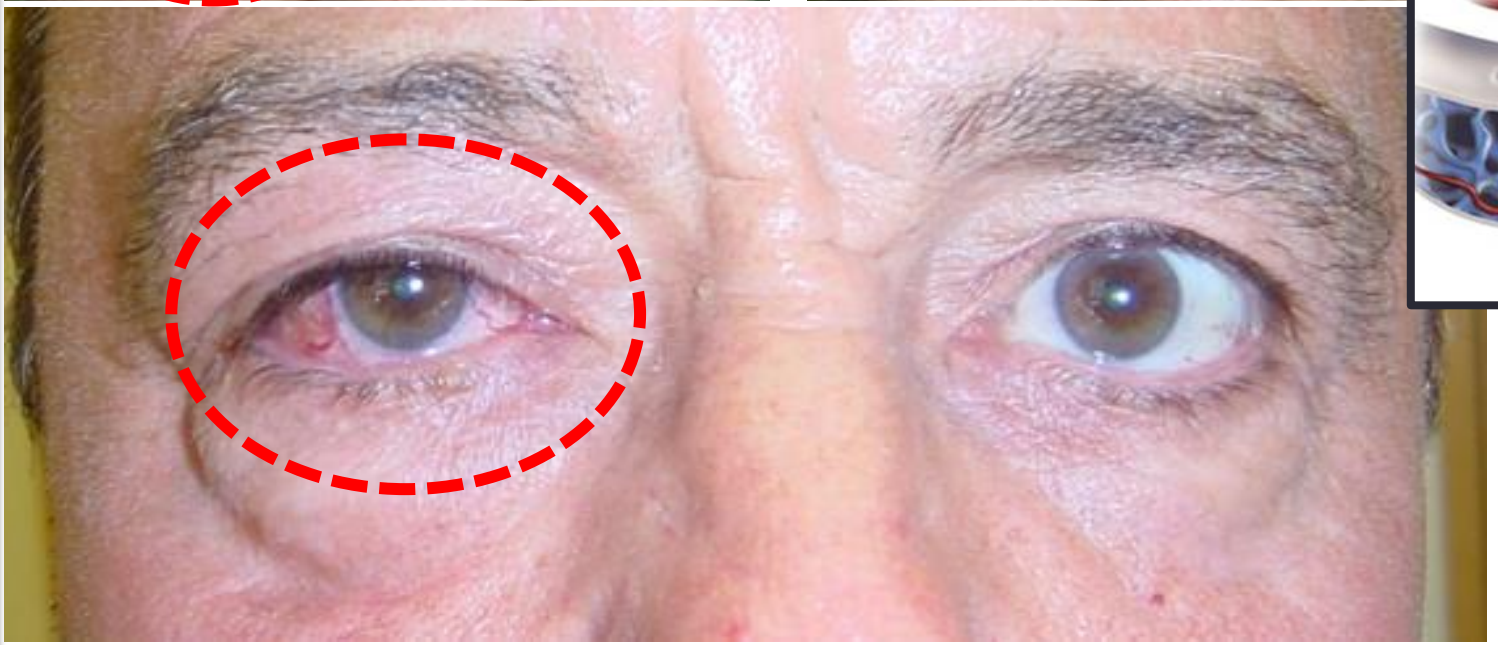
- Most minimal symptoms
- Many cure spontaneously
- Treatment: endovascular

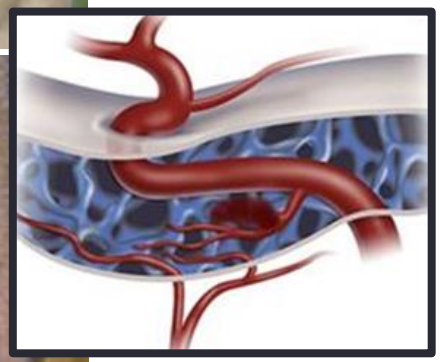


Spontaneous carotid-cavernous fistula

- In severe cases = glaucoma

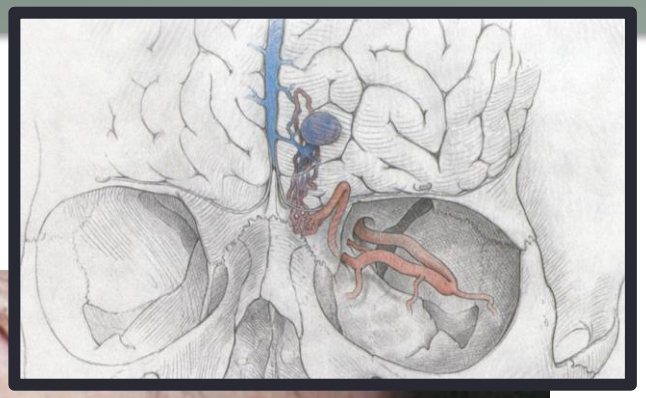






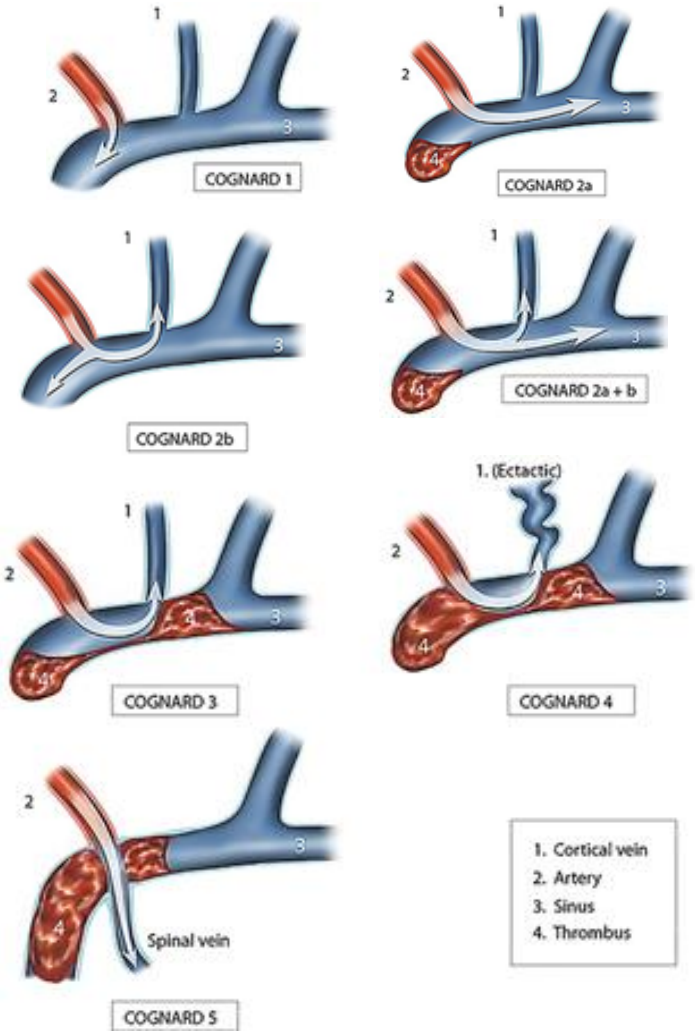
Brain dural AVM in anterior cranial fossa

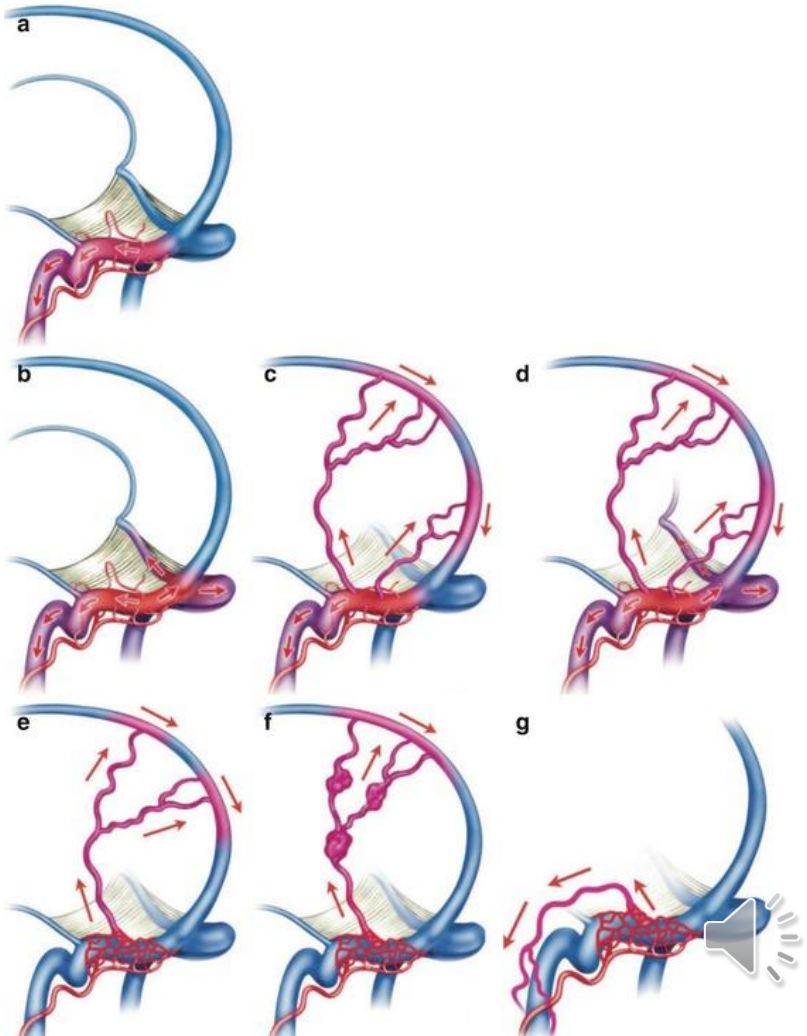
- Can induce hypertrophy of ophthalmic veins



Brain dural A-V fistula evolution: Cognard classification

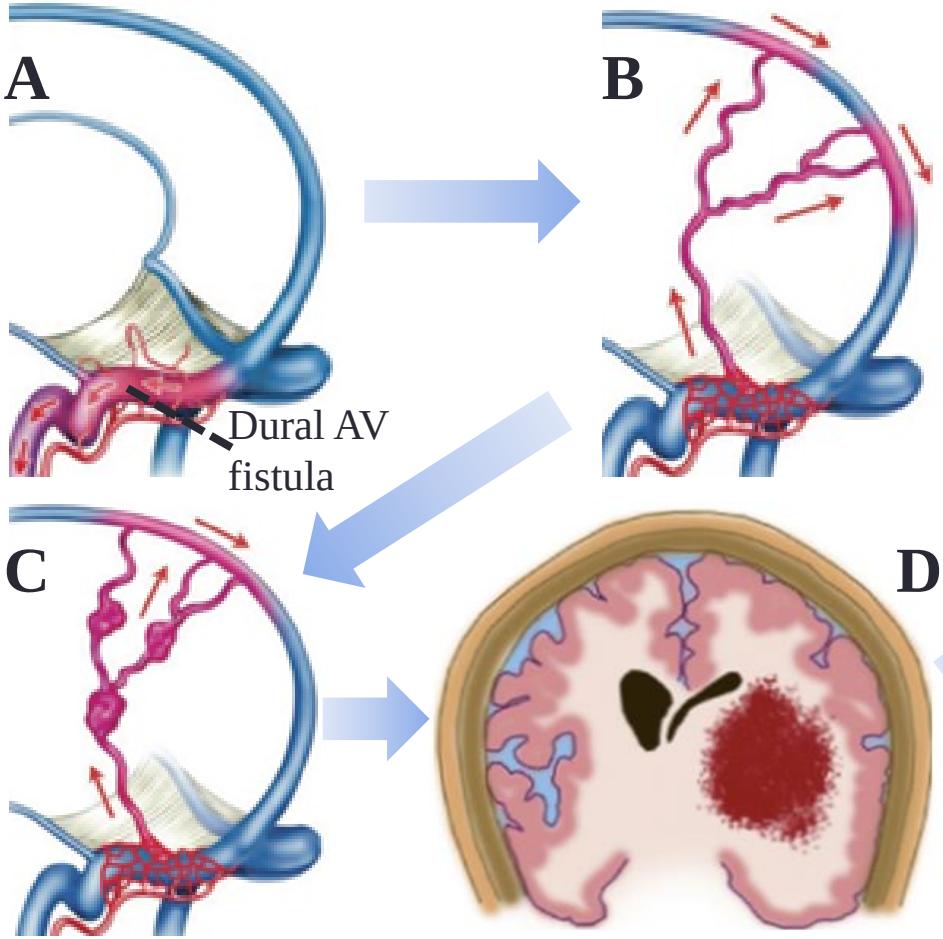
- Helpful to decide the treatment strategy

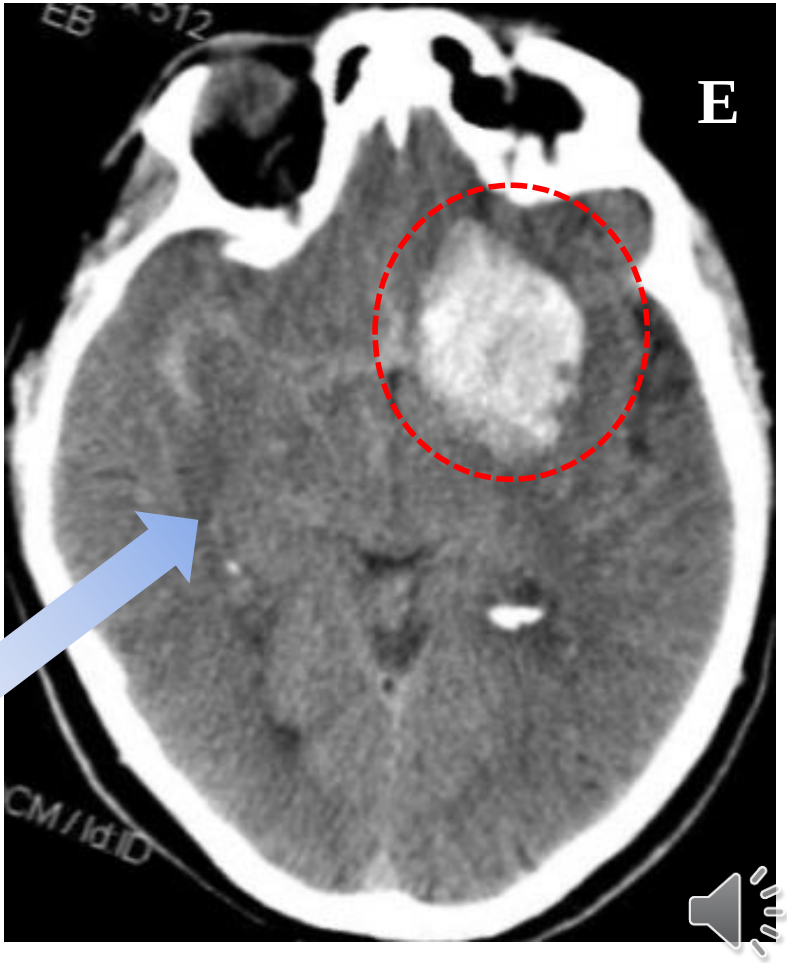




CT scan: intra-cerebral hemorrhage in dural A-V fistula with venous flow inversion

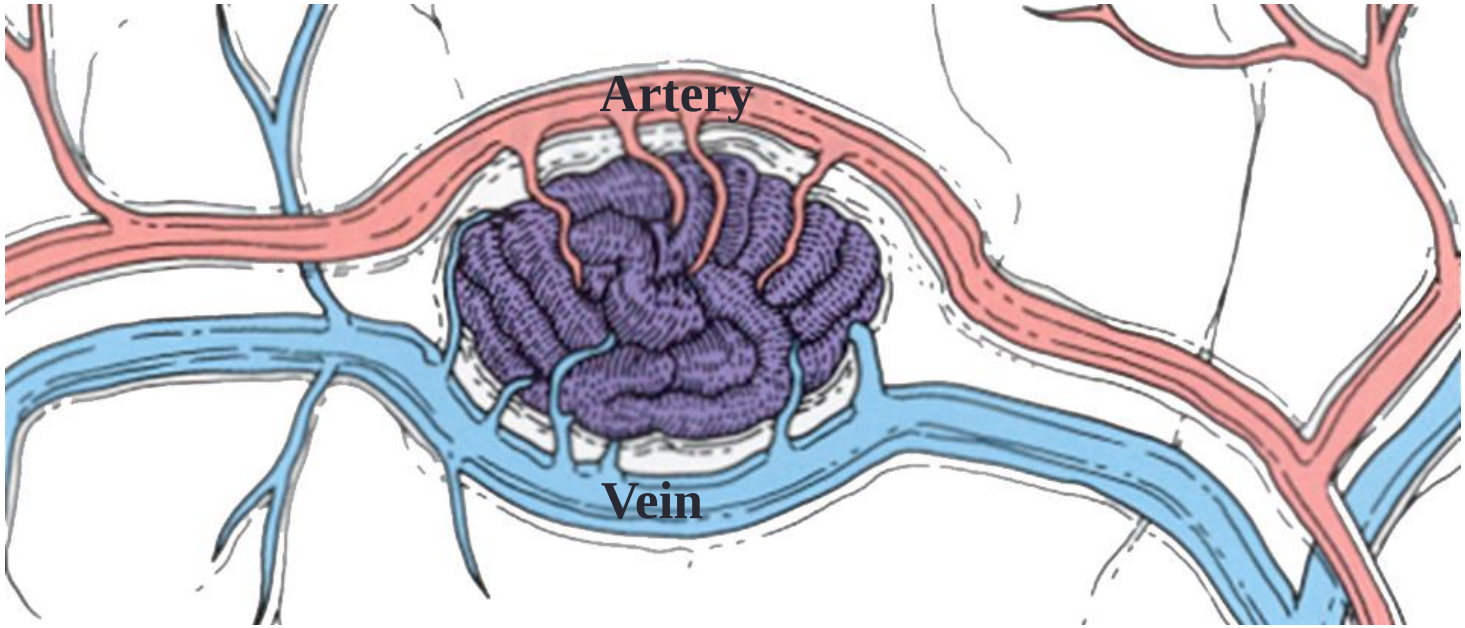
- Venous flow inversion = increase in cerebral vein pressure ≅ haemorrhage





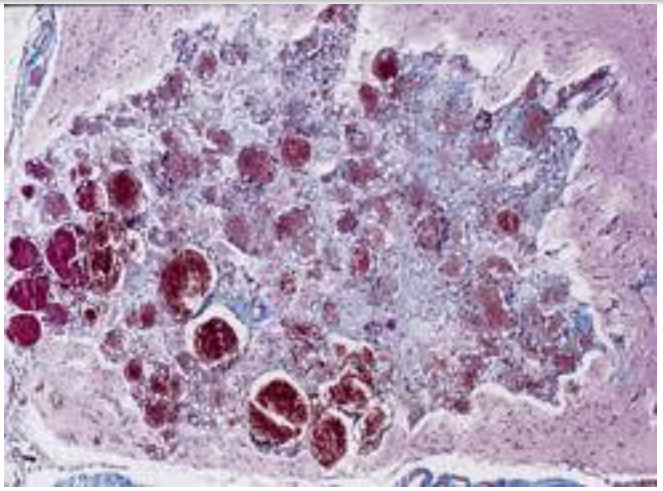
CAVERNOUS ANGIOMA

- Malformation of capillaries
- Can be located anywhere in the CNS (includes spinal cord)
- Small microbleeds
  - Brain = epilepsy
  - Spinal cord + brainstem = progressive neurological deficit
- Familial incidence

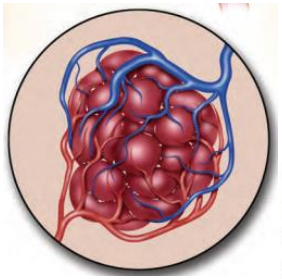


Cavernous angioma: features

- Honeycomb vascular spaces lined with endothelium separated by fibrous collagen bands
  - No nerve tissue inside
- Well delimited
- Hemosiderin & calcification frequently
- Slight contrast uptake
- Not seen on angiography
- Can ↑ size due to haemorrhages inside



Microscopic view

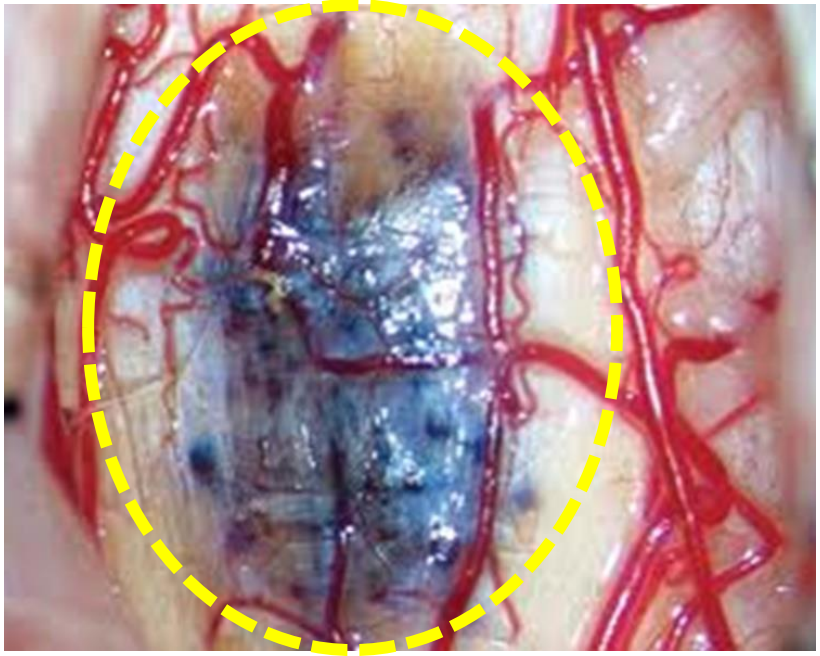


Macroscopic view

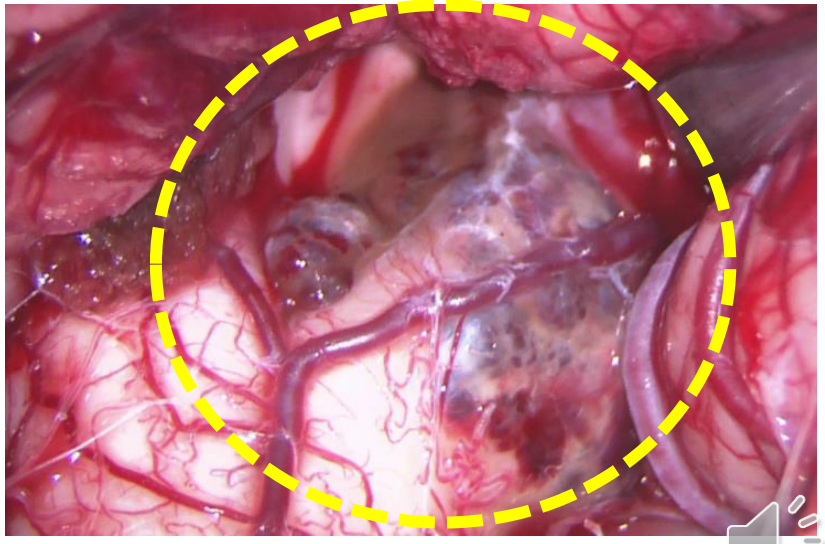
Cavernous angioma: macroscopic view

- Cause: endothelial dysmorphogenetic
- Not a tumour
  - Does not display endothelial hyperplasia
- Abnormal tissue = slowing of blood flow through the cavities or ‘caverns’

Post-mortem appearance brain



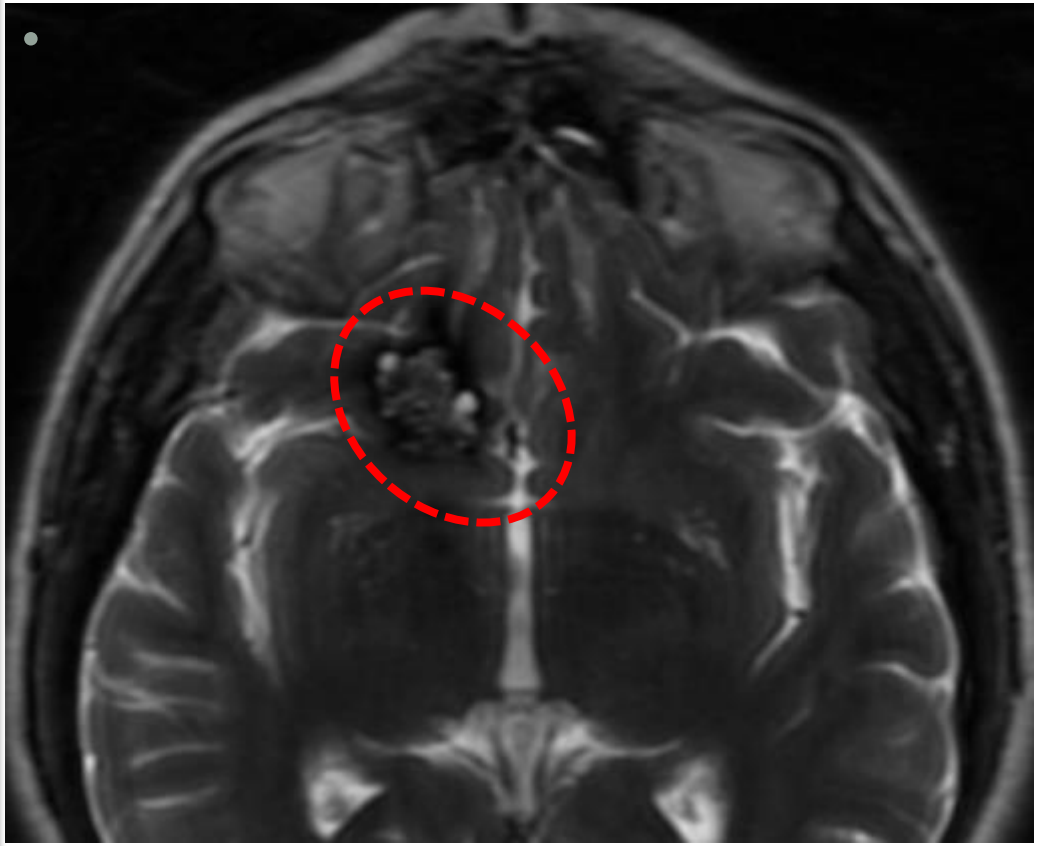
Intraoperative appearance spinal cord



Intraoperative appearance medulla

Cavernous angioma: diagnosis

- MRI: ‘Popcorn’ with hemosiderin halo
- CT scan: calcification in 50%

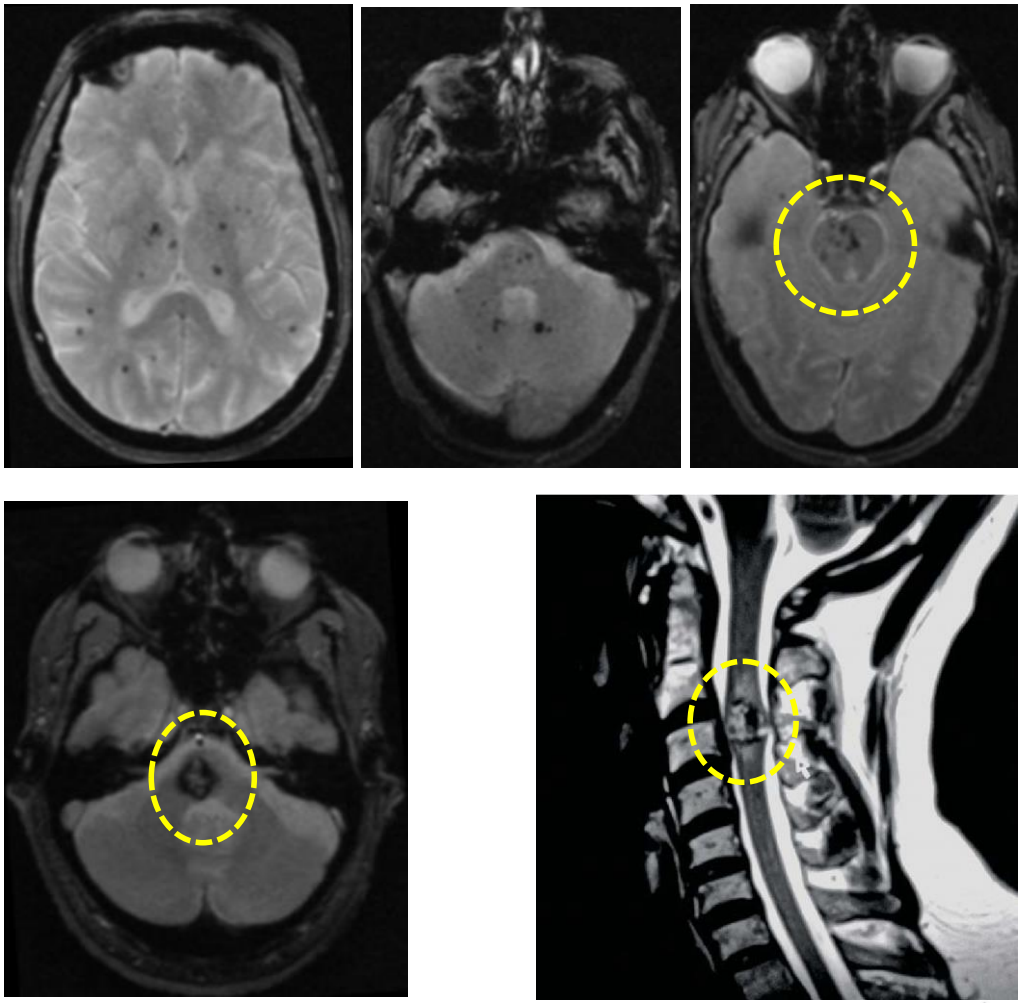


MRI: popcorn + hemosiderin halo

CT scan: calcification

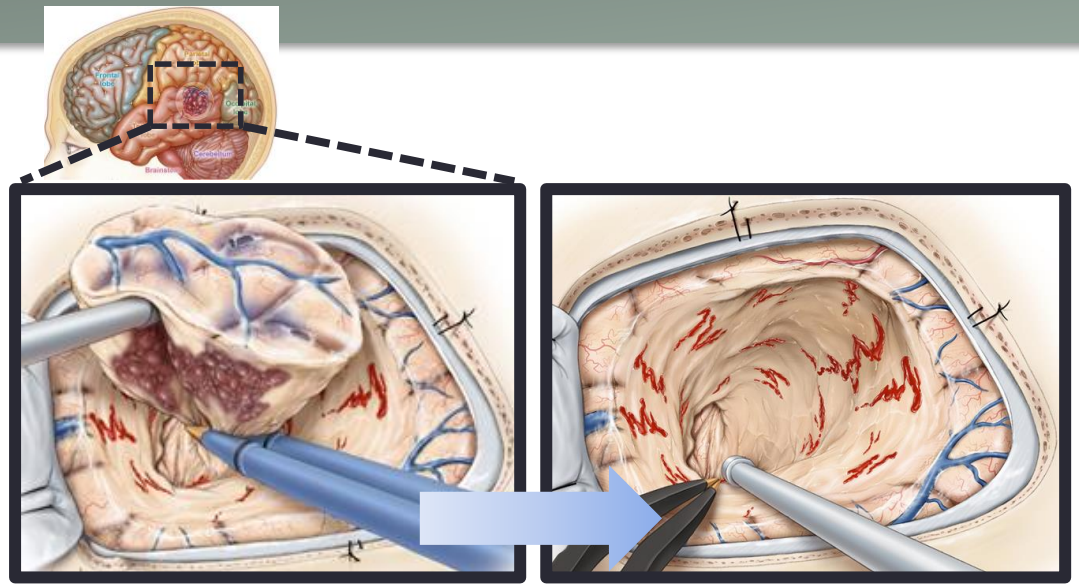
Cavernous angioma: clinical symptoms

- Epileptic seizures
  - The most common symptom
- Bleeding
  - Rarely symptomatic
  - Repeated microbleeds = Fe ++ in nervous tissue = free radical formation = epileptic seizures / progressive neurological deficits
- Cranial nerve deficits
- Spinal cord deficits
- Asymptomatic

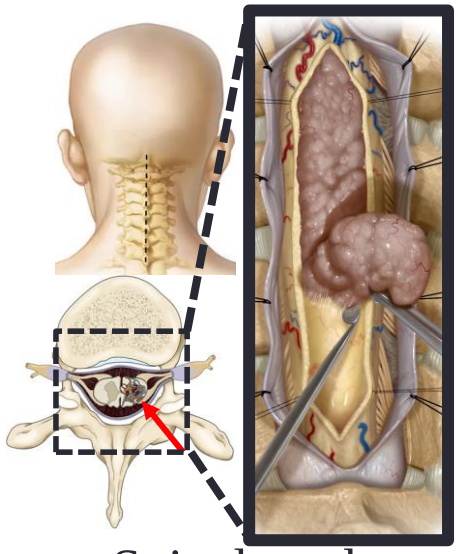


Cavernous angioma: treatment indications

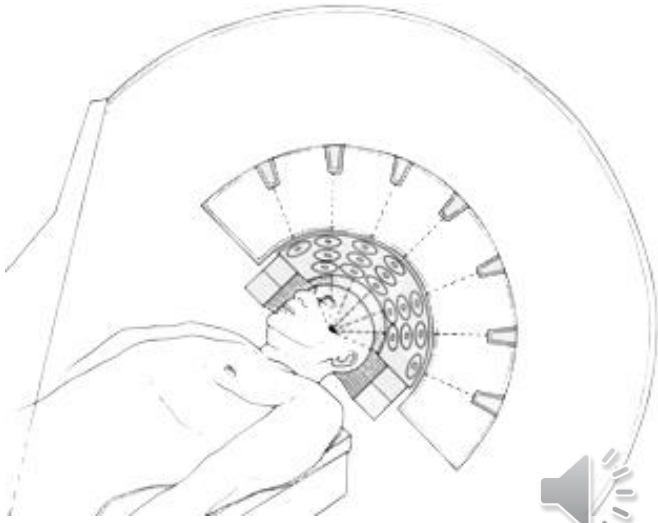
- Refractory epileptic seizures
  - Good response if cavernoma + hemosiderin marked area are removed
- Significant or repetitive bleeding
- Choices
  - Surgery
    - Most effective
  - Radiosurgery
    - Only reduces bleeding probability
    - Not very effective



Removal brain cavernous malformation



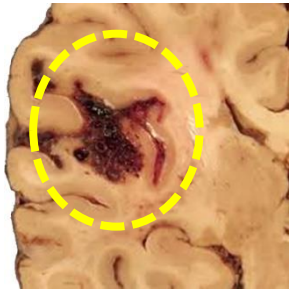
Spinal cord

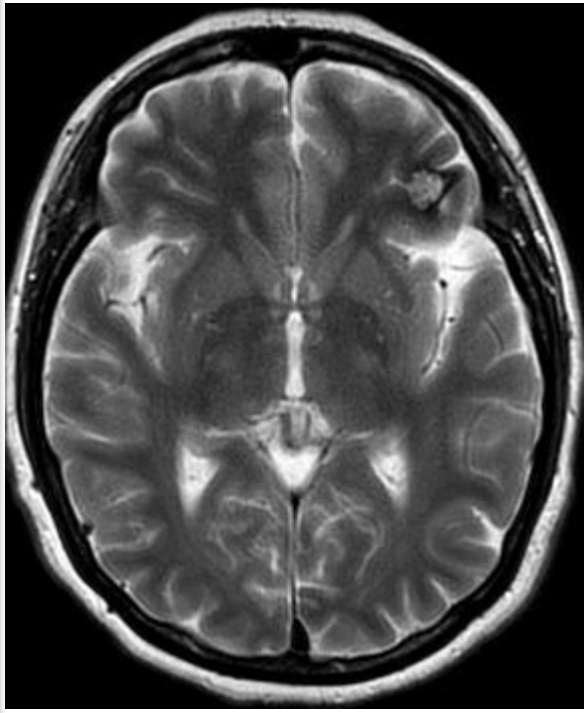


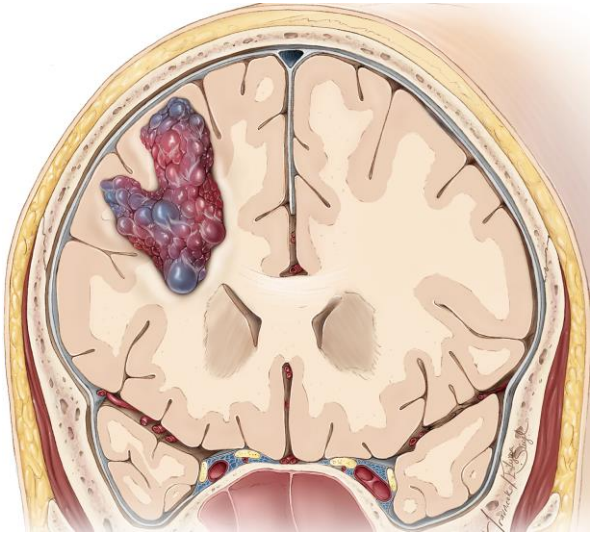
Radiosurgery

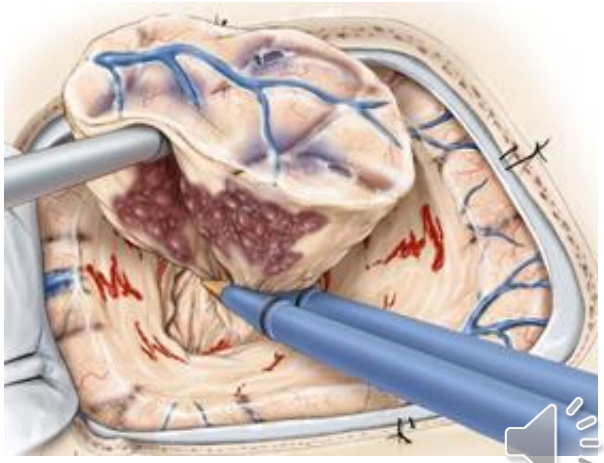
Cavernous angioma: brain

- Symptoms: epileptic seizures
- Often multiple
- Treatment: surgery



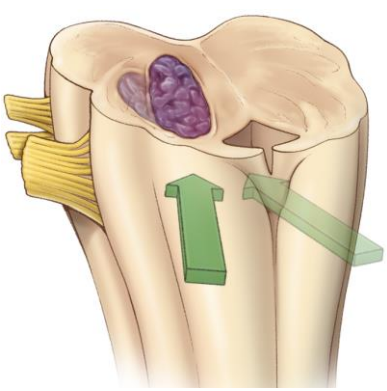


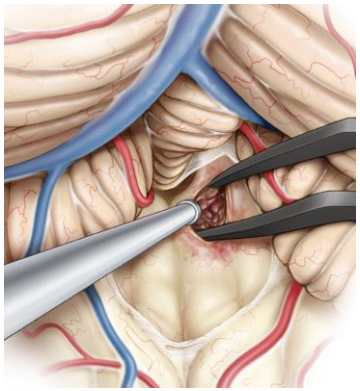


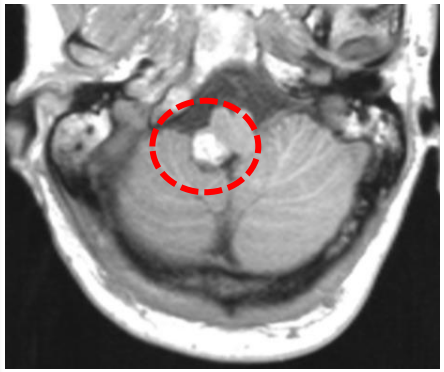


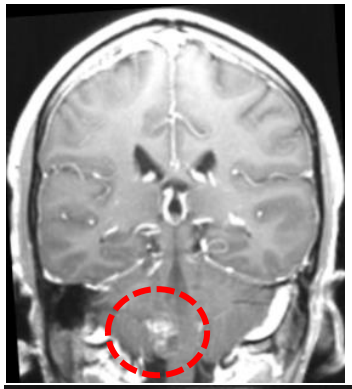
Cavernous angioma: brainstem

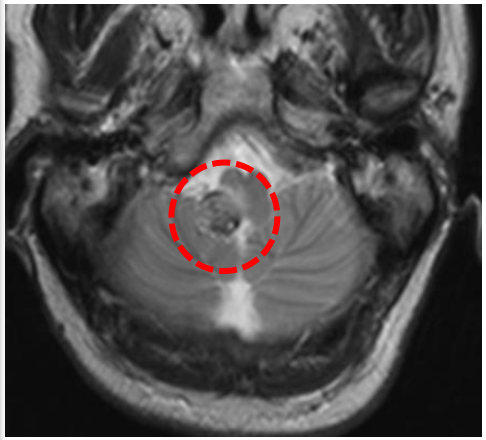
- Repeated microbleeds = progressive cranial nerve deficits
- Treatment: surgery
  - Radiosurgery: minor effect

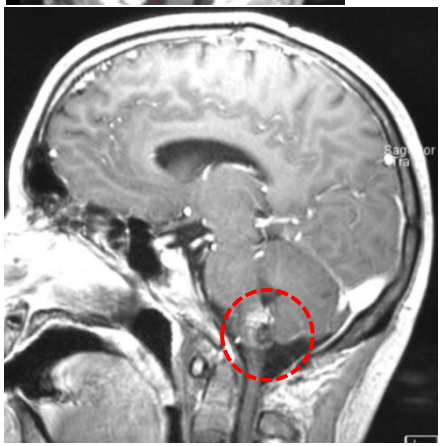


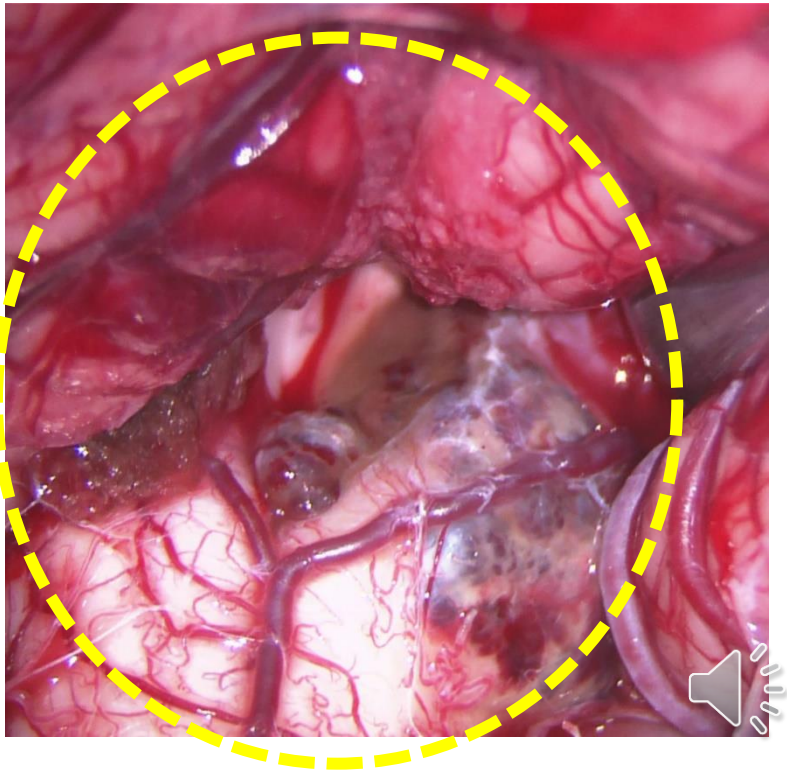






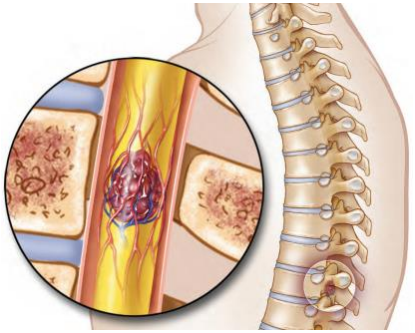


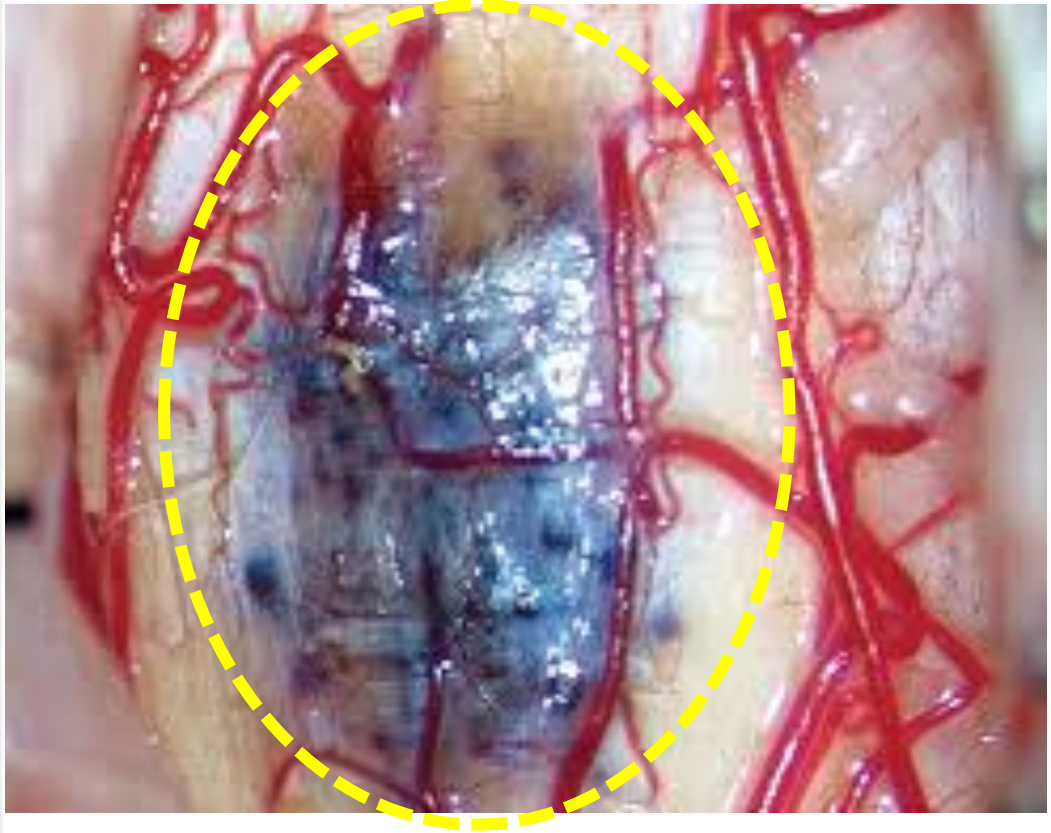


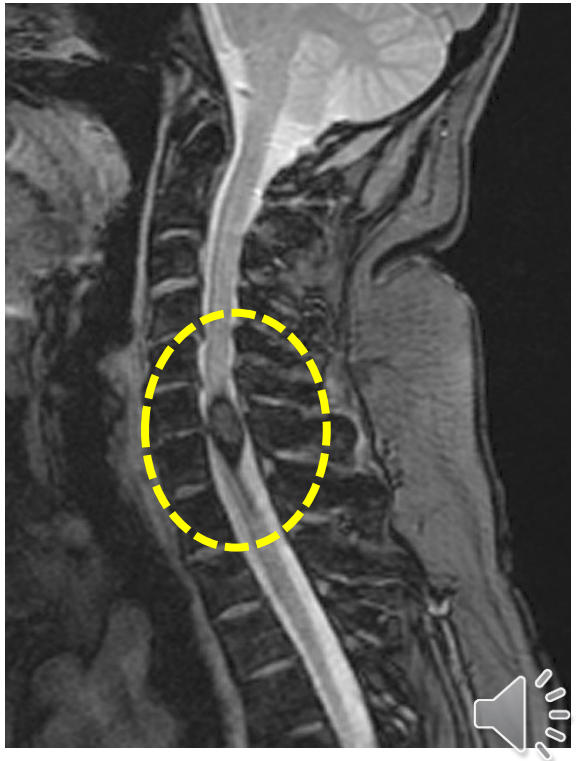


Cavernous angioma: spinal cord

- Repeated microbleeds = progressive tetra / paraplegia
- Treatment: surgery

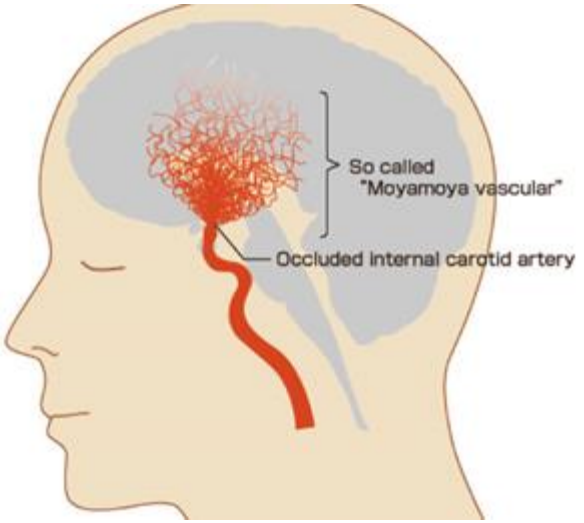


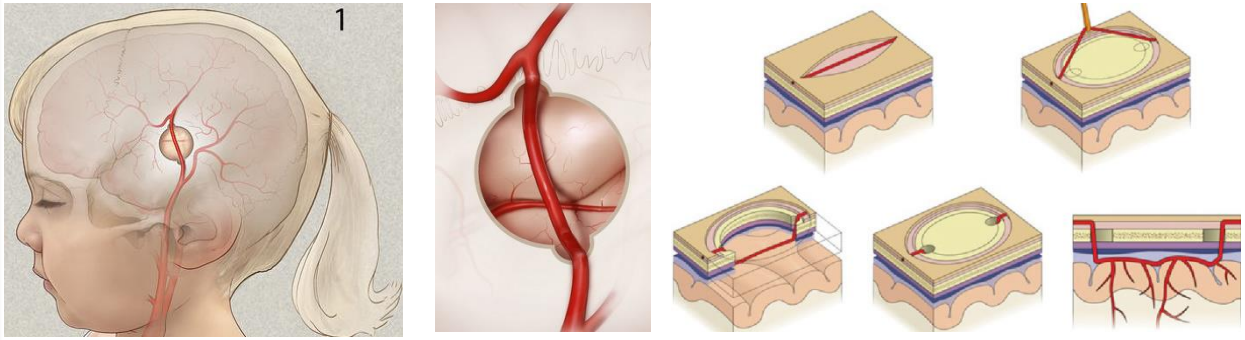




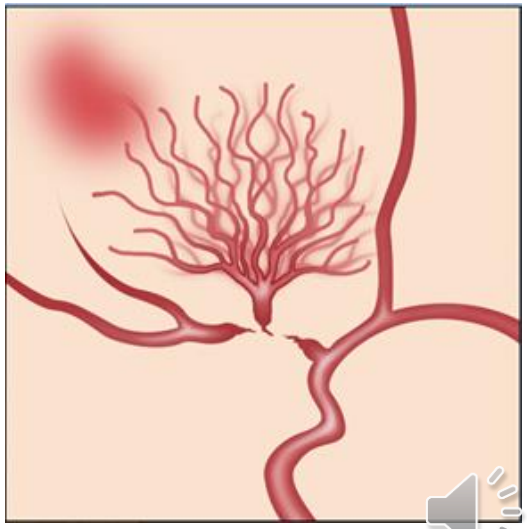
Moyamoya disease

- Moyamoya = ‘puff of smoke’ (Japanese)
- Arteritis intracranial vascularization with progressive occlusion = ischemia
- ↑ incidence in Asian people
- Progressive cerebral ischemia
- Treatment: surgical (brain revascularization)





Brain revascularization in moyamoya disease



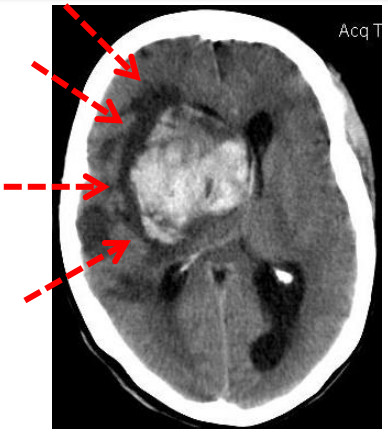
Moyamoya

INTRACEREBRAL HAEMATOMAS

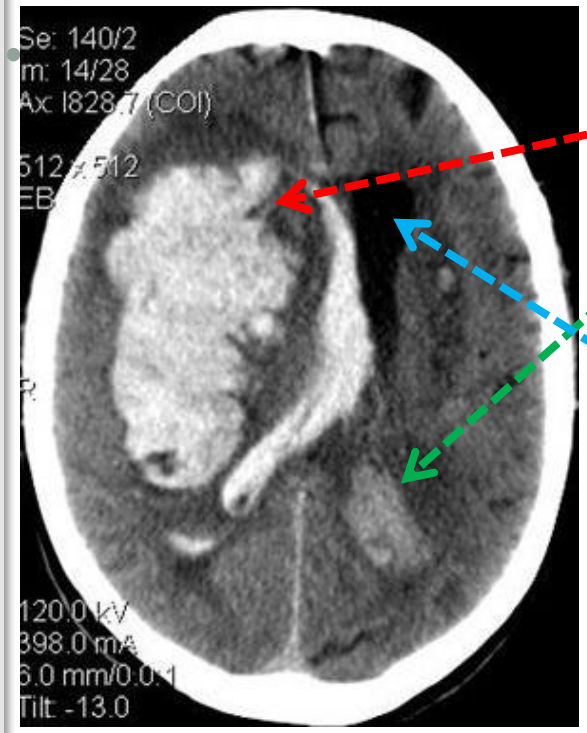
- Definition: haematoma within the cerebral parenchyma
- Consequence: destruction white matter fiber tracts = severe neurological deficits

Intracerebral haematoma: effects

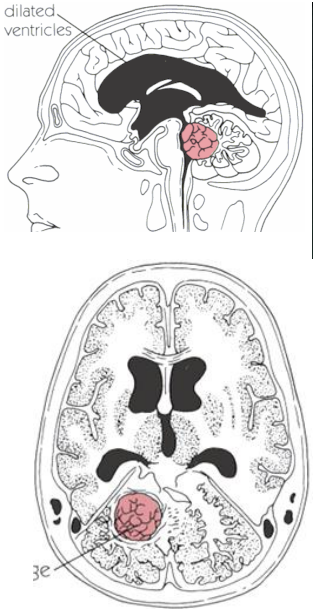
- Nearby nerve tissue compression → ischemia = perilesional edema
- CSF pathway obstruction → hydrocephalus
- Fibre tract damage

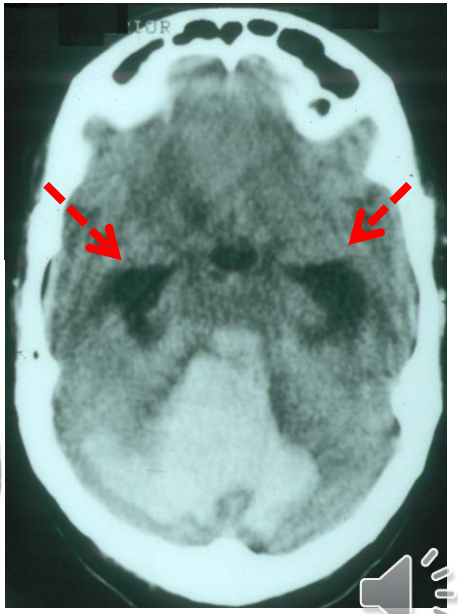


Edema in thalamic haematoma



Thalamic haematoma + intraventricular haemorrhage + hydrocephalus

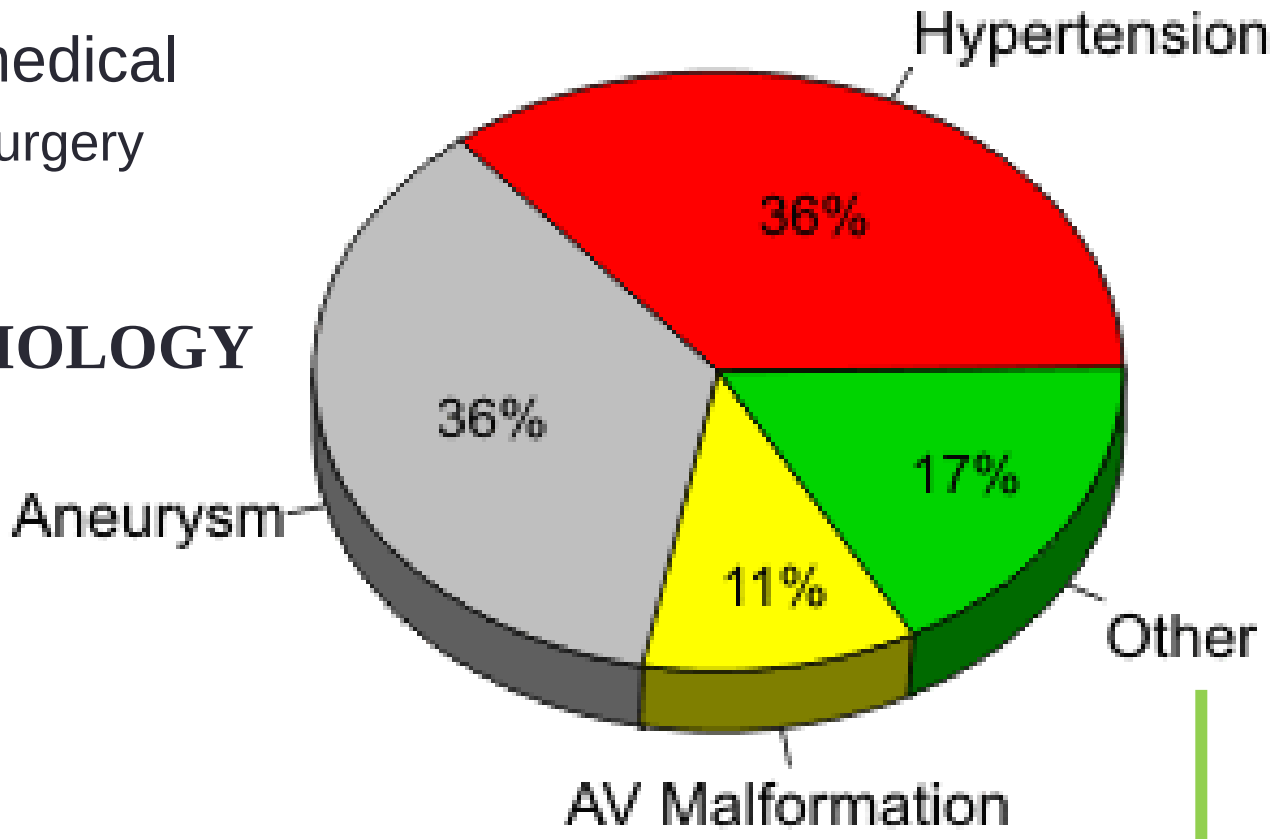




Posterior fossa haematoma + hydrocephalus

Spontaneous intracerebral haematomas

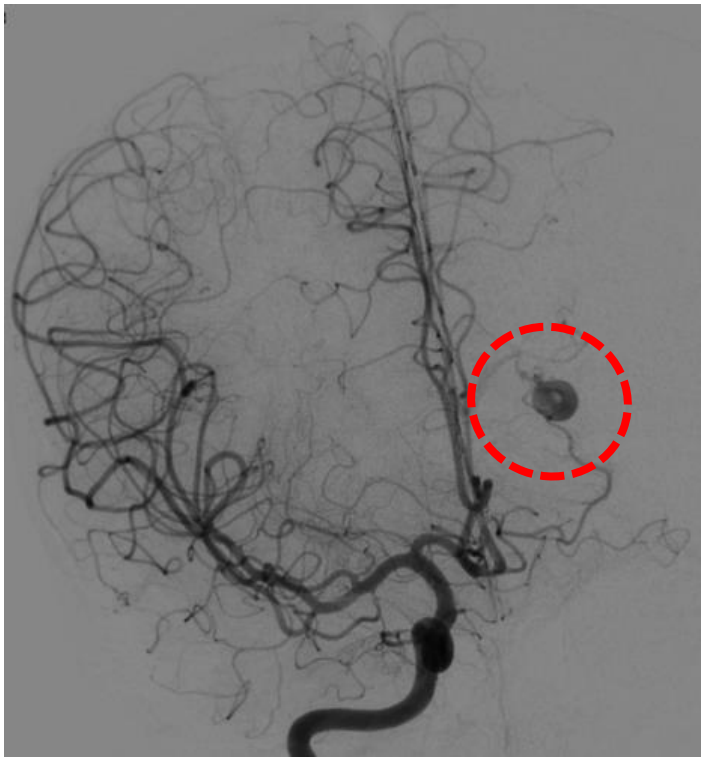
- Treatment = medical
  - Only a few = surgery

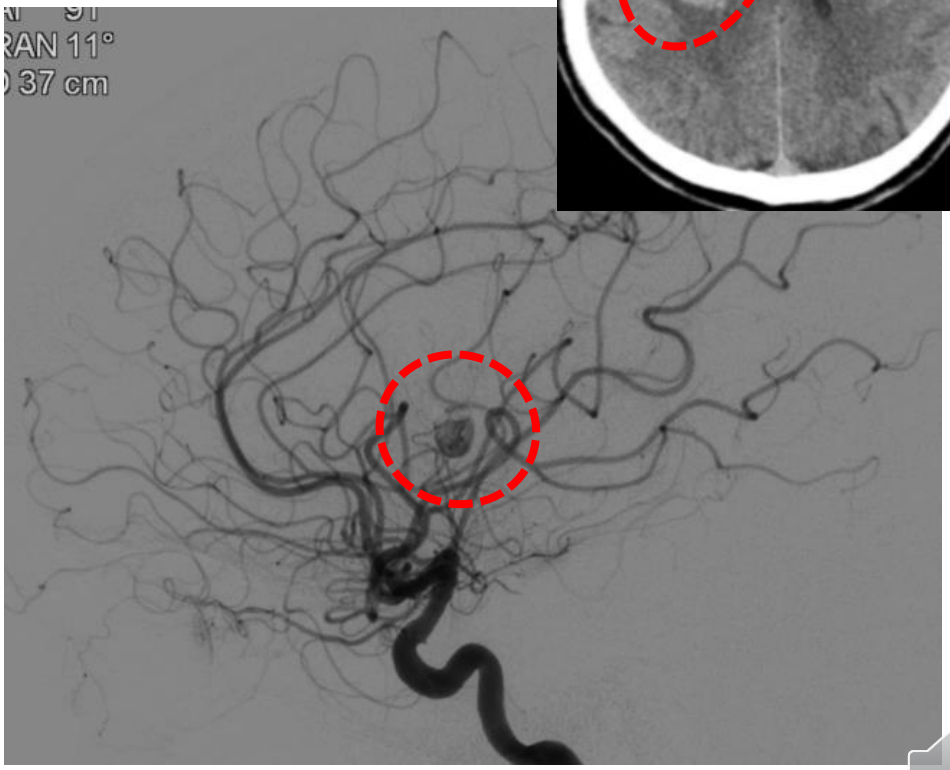


Other causes (17%): amyloid angiopathy, anti-coagulation, anti-platelet medication, intratumor haemorrhage, and haemorrhagic infarction

Haematoma secondary to high arterial pressure

- Bleeding medical treatment
- Prevention rebleeding = endovascular
- Dismal results = hemiplegia



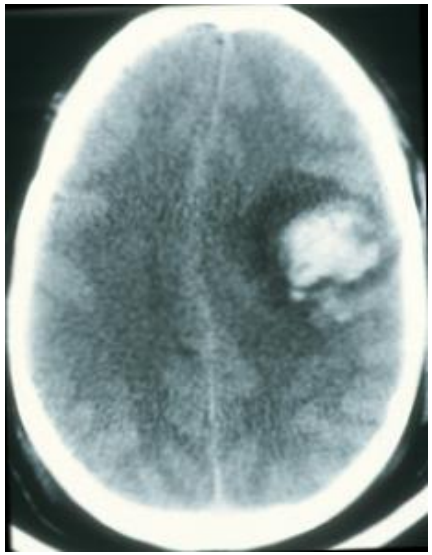


Thalamic hematoma secondary to Charcot-Bouchard aneurysm

Intracerebral haematoma: treatment (1)

- Reverse anti-coagulation / anti-platelet medication
- Control blood pressure
- UCI treatment
- Surgery
  - Drainage: hematoma > 35 cm^3, previous good condition (∅ dementia) + ∅ neurological deficit (hemiplegia)
  - Treatment hydrocephalus

Medical treatment



ICU treatment

Surgery

Conservative treatment

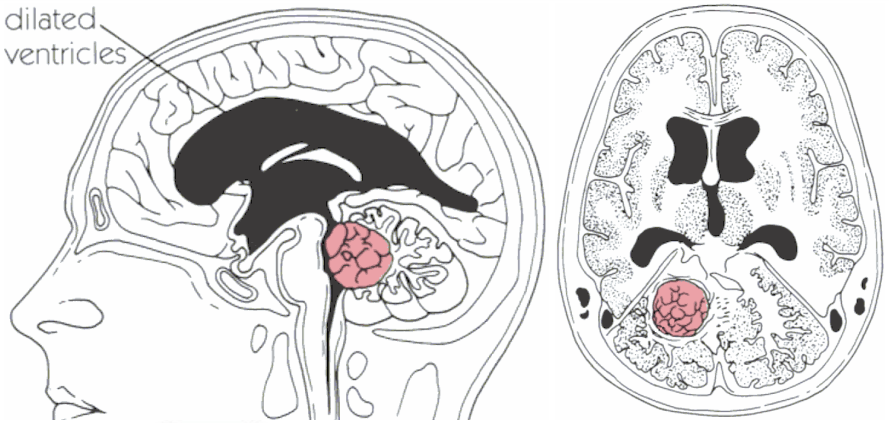
Intracerebral haematoma: treatment (2)

- Surgery only if good quality of life expected post-op

Surgical treatment possible if mass effect	Usually conservative treatment	Conservative treatment
		
Lobar haematoma	Putamen haematoma	Thalamic haematoma

Posterior fossa hematoma + hydrocephalus = external ventricular drainage ± clot removal

- Usually good quality of life post-op
  - Mild ataxia



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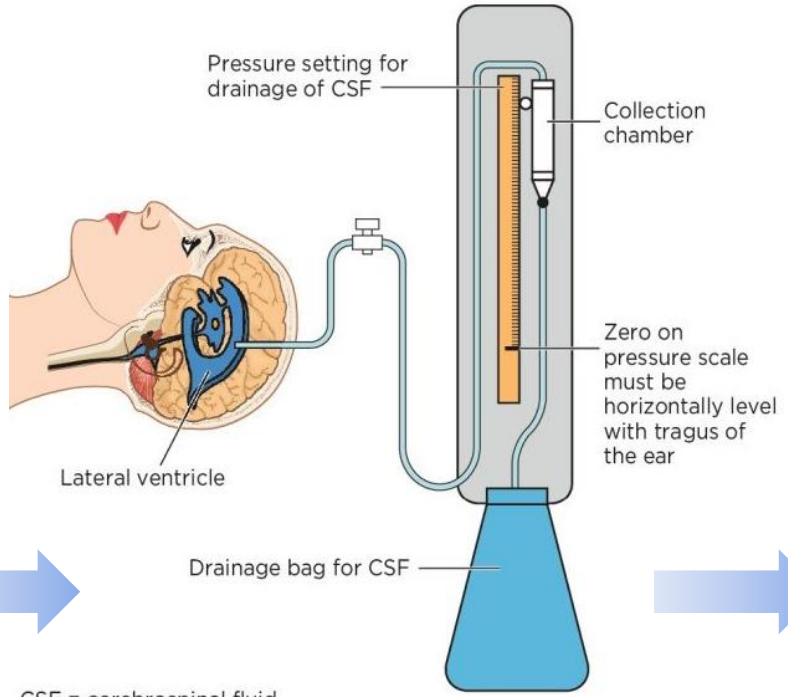
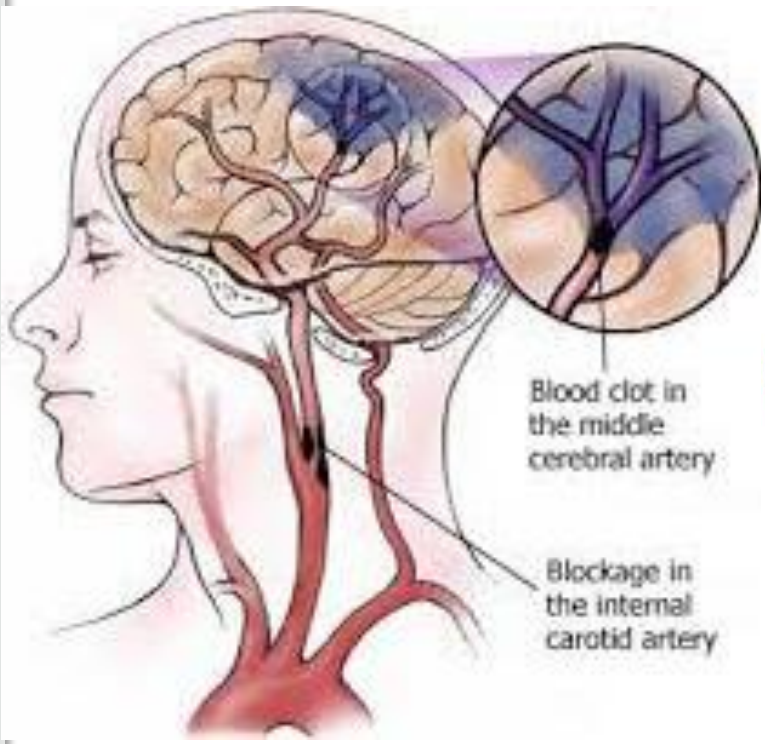
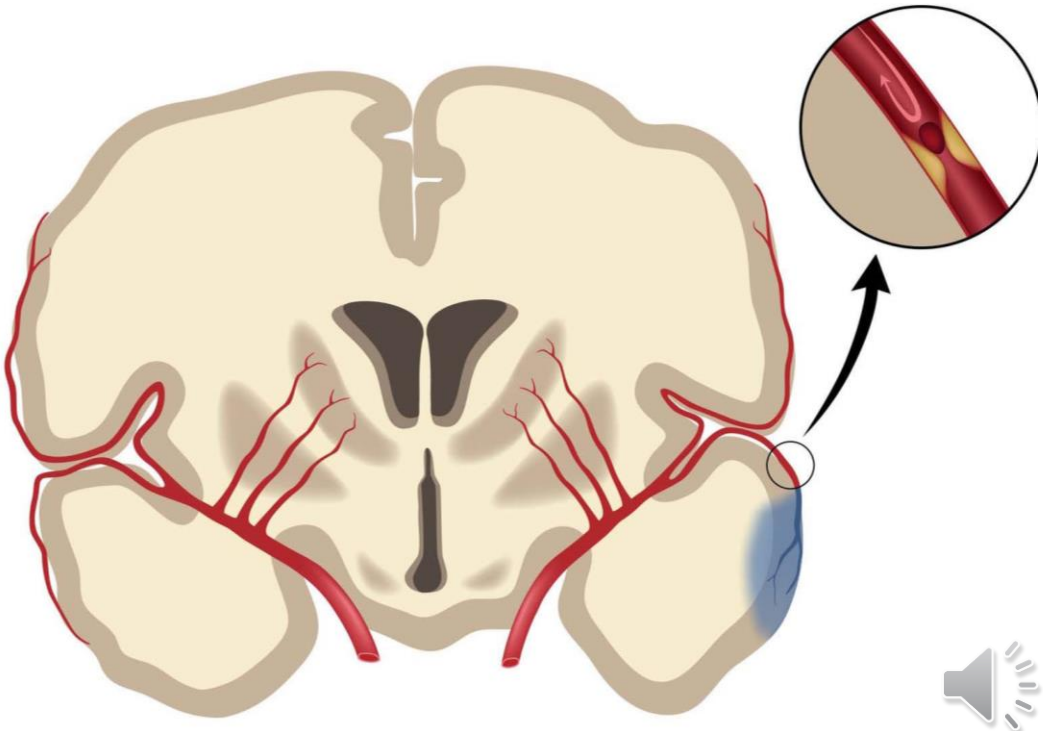


BRAIN ISCHEMIA: surgical treatment

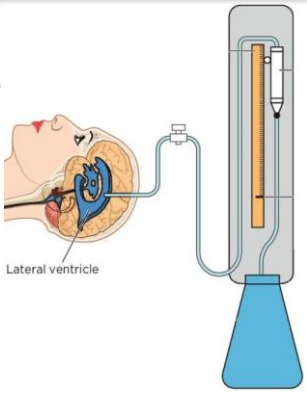
- All patients MUST start with medical treatment
- Only a few can benefit from surgery
  - Cerebellar infarct
  - Malignant middle cerebral artery infarct



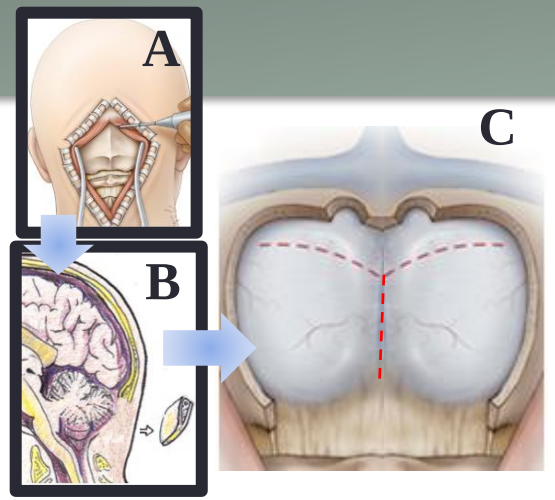


Cerebellar infarct = hydrocephalus + posterior fossa mass effect

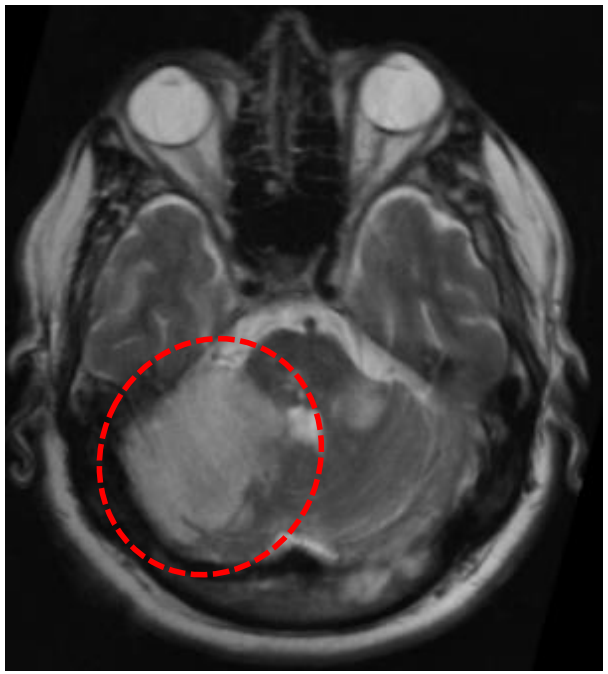
- Surgery = hydrocephalus drainage ± posterior fossa craniectomy
- Survivors have acceptable quality of life = ataxia



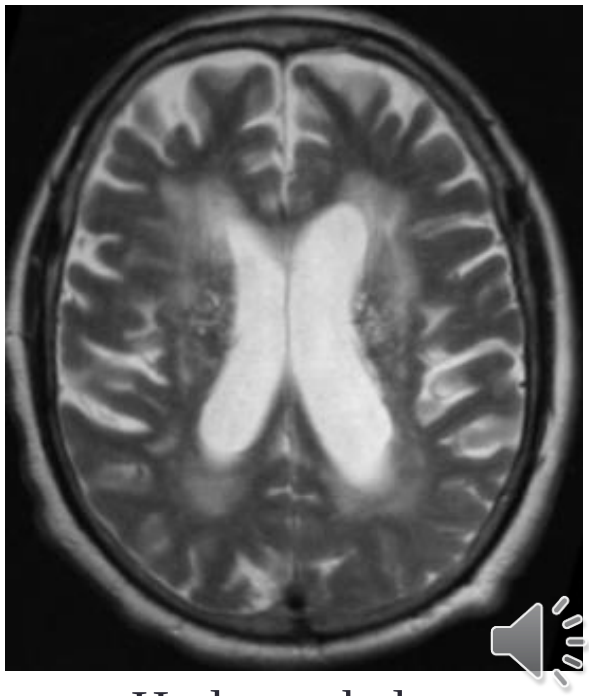
External ventricular drain



Posterior fossa decompression



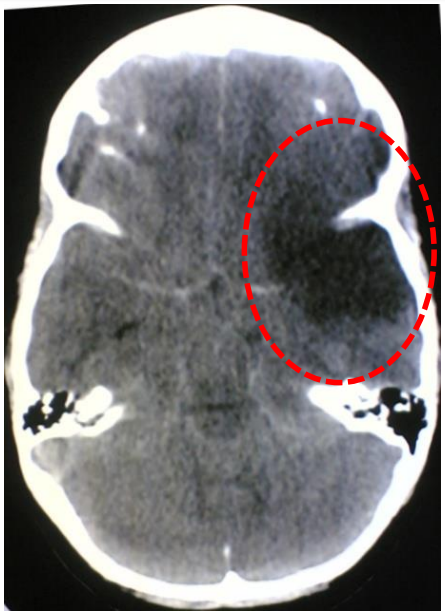
Cerebellar infarct



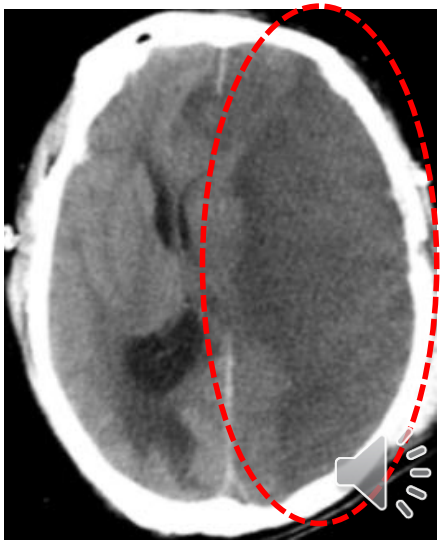
Hydrocephalus

Malignant middle cerebral artery infarct

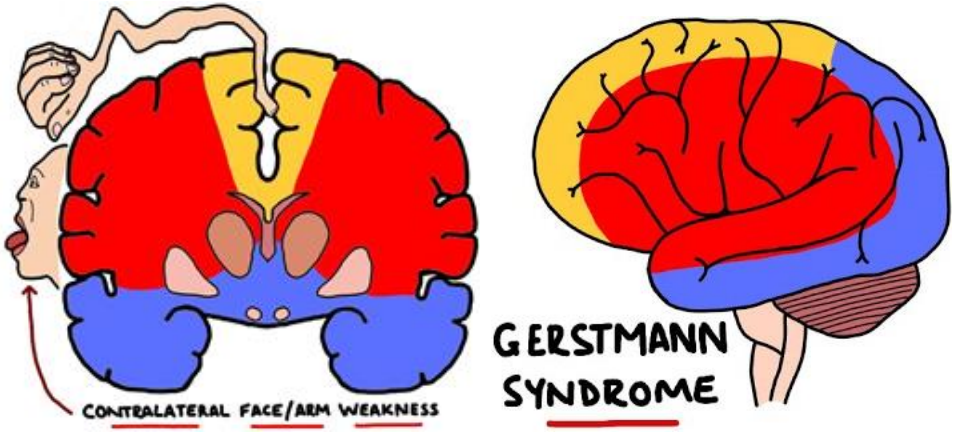
- Infarct > 50% middle cerebral artery distribution
- Death almost always
- ⇓ 10% supratentorial ischemic strokes
- Clinical features
  - Complete hemiplegia
  - Heminegligence
  - Dominant hemisphere = aphasia
  - ⇓ level of consciousness



Middle cerebral artery infarct



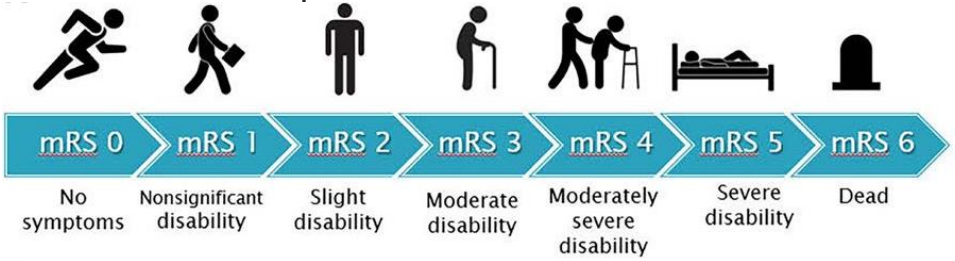
Malignant middle cerebral artery infarct

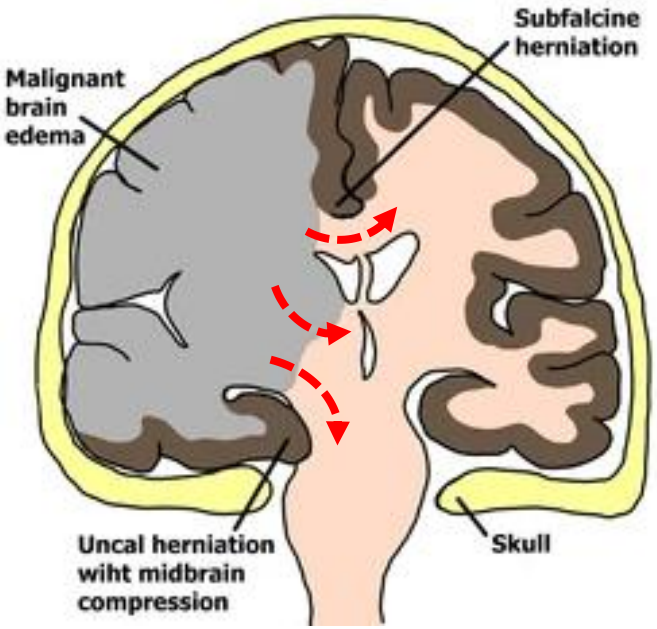


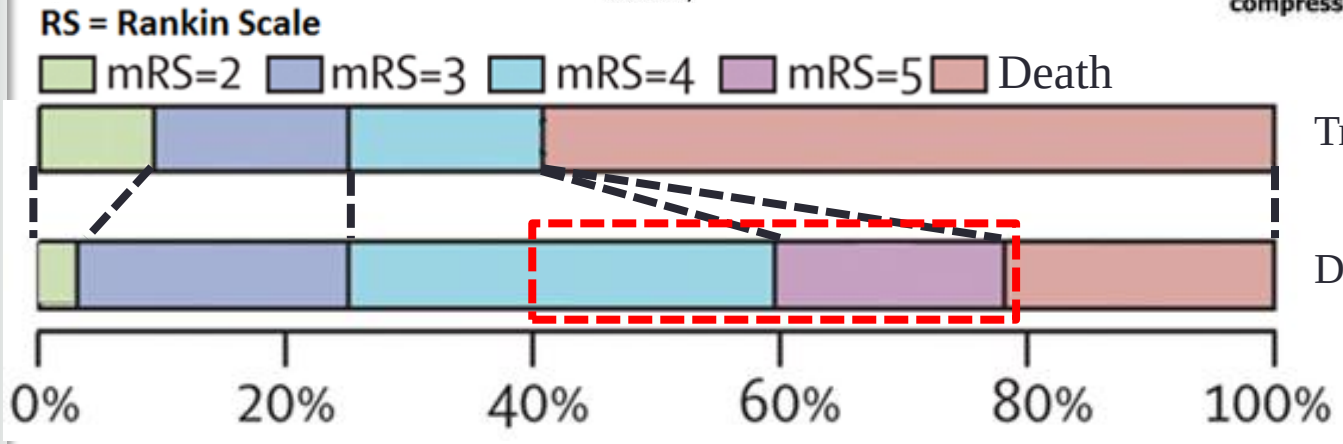
Middle cerebral artery distribution

Malignant middle cerebral artery infarct: physiopathology

- Malignant middle cerebral artery infarction = ⇑ extensive cerebral infarct
- Edema = ⇑ intracranial pressure = ⇓ level of consciousness, neurological deterioration, uncal & subfalcine herniation
- Treatment: ICU → death 60-80%
- Surgical treatment = decompressive craniectomy
  - Controls ⇑ intracranial pressure = ⇑ survival
  - Improves functional result (Rankin scale)
  - Survival with sequelae





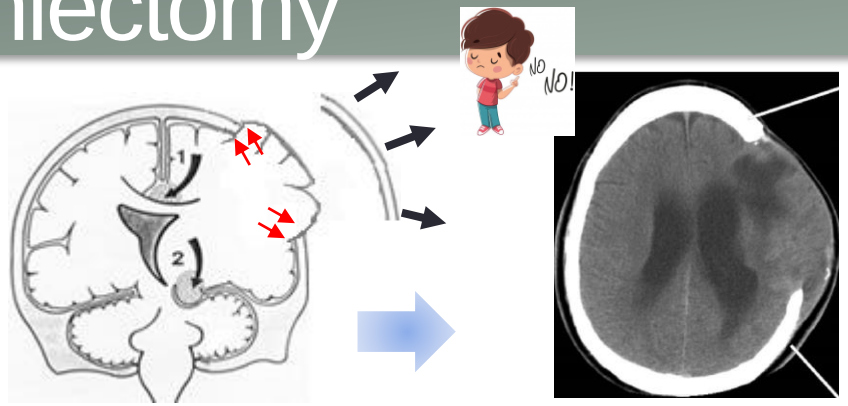


Treatment ICU

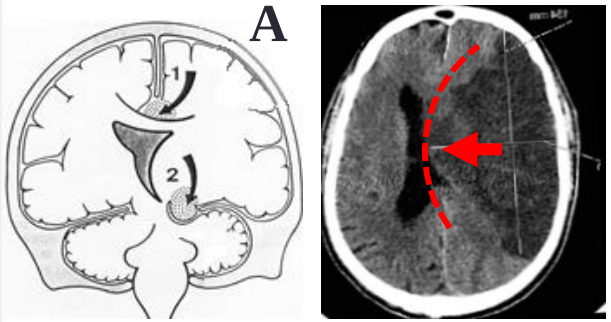
Decompressive craniectomy

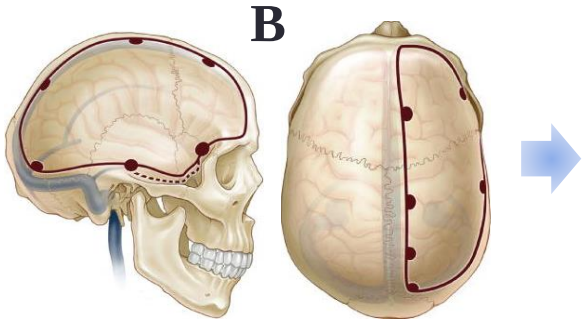
Malignant cerebral artery infarct: decompressive craniectomy

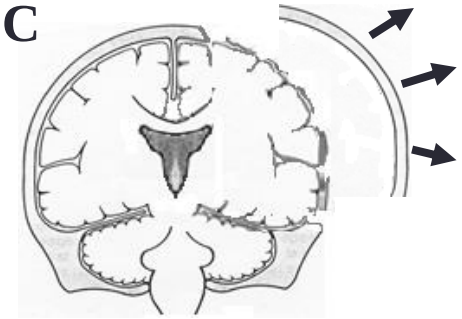
- ↑ survivors but with neurological sequelae
- MUST be done on time
  - Delays = more brain damage
- Extensive enough to decompress brain
  - Small craniectomy= brain necrosis against bone edges



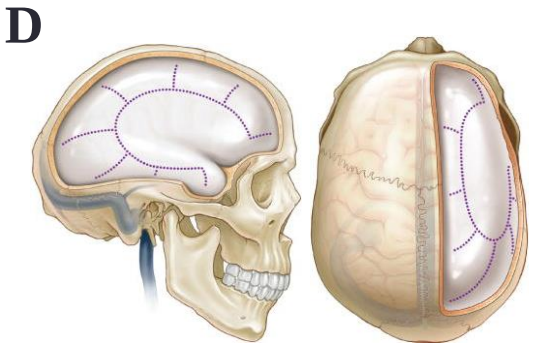
INCORRECT: small craniectomy



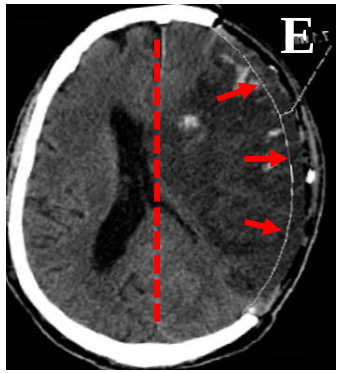




Hemicraniectomy



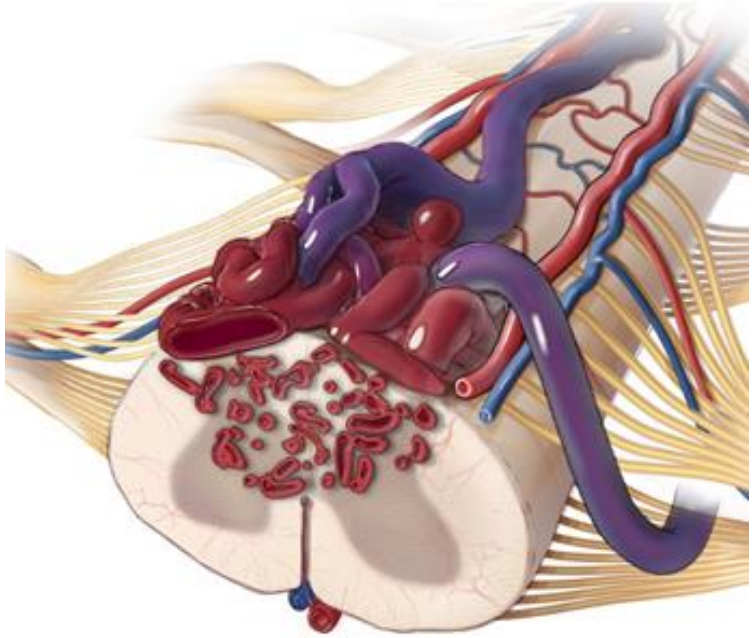
Dura opening

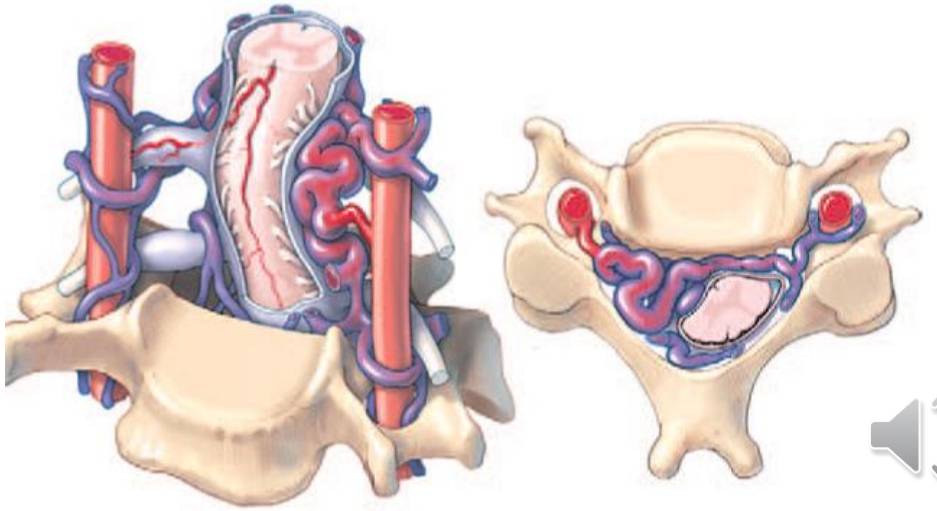


CORRECT: extensive craniectomy

SPINAL CORD AVMs (arteriovenous malformations)

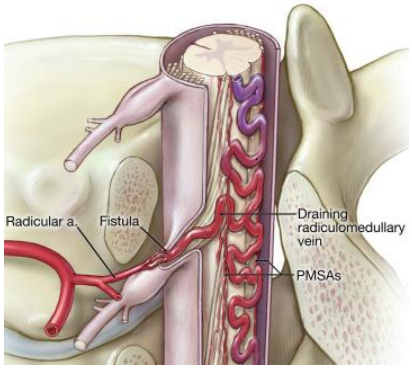
- Abnormal direct connection between spinal cord arteries and veins with NO capillary bed
- Consequences
  - Blood stolen from normal spinal tissue = spinal cord ischemia
  - Venous hypertension in the medullary veins = spinal compression

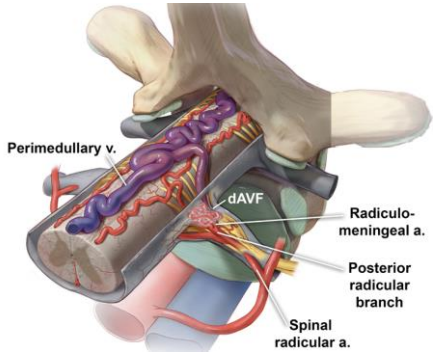




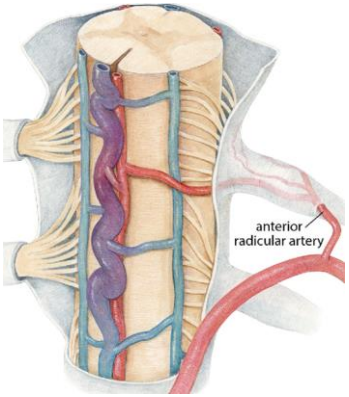
Spinal cord AVMs: types

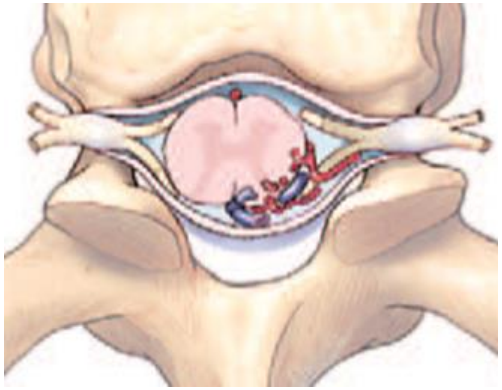
- Dural AV fistulas
  - The fistula is OUTSIDE the spinal canal
- Perimedullary AVM
  - AVM nidus AROUND the spinal cord
- Intramedullary AVM
  - AVM nidus INSIDE the spinal cord



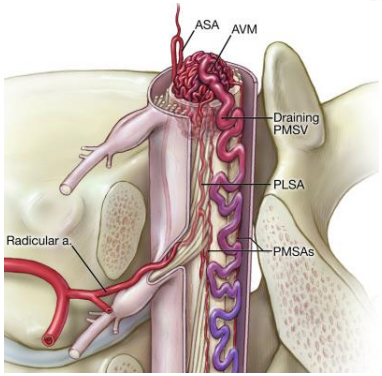


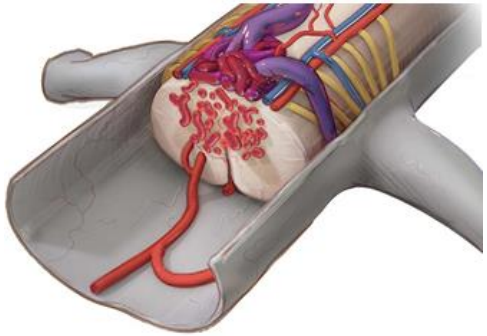
Dural AV fistula





Perimedullary AVM

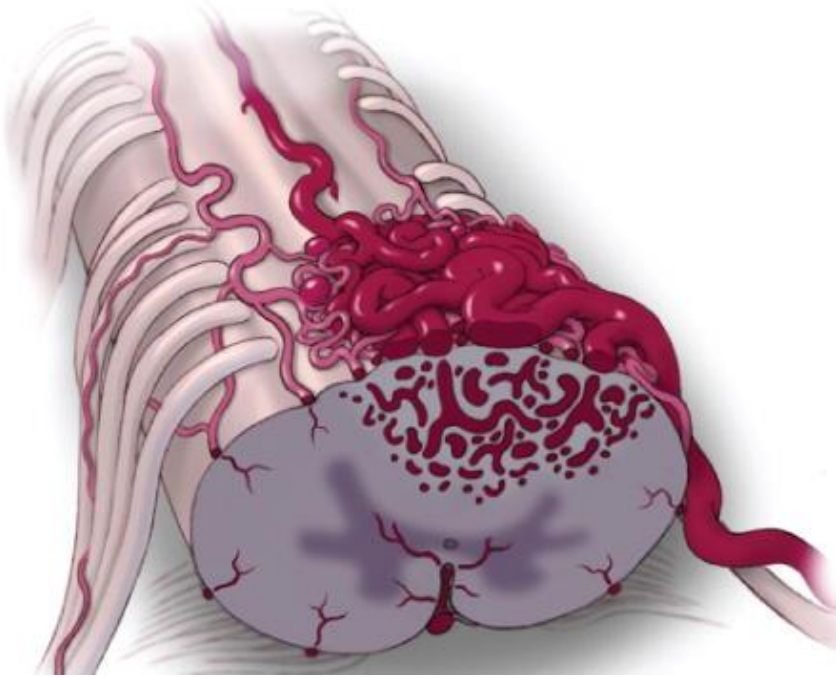




Intramedullary AVM

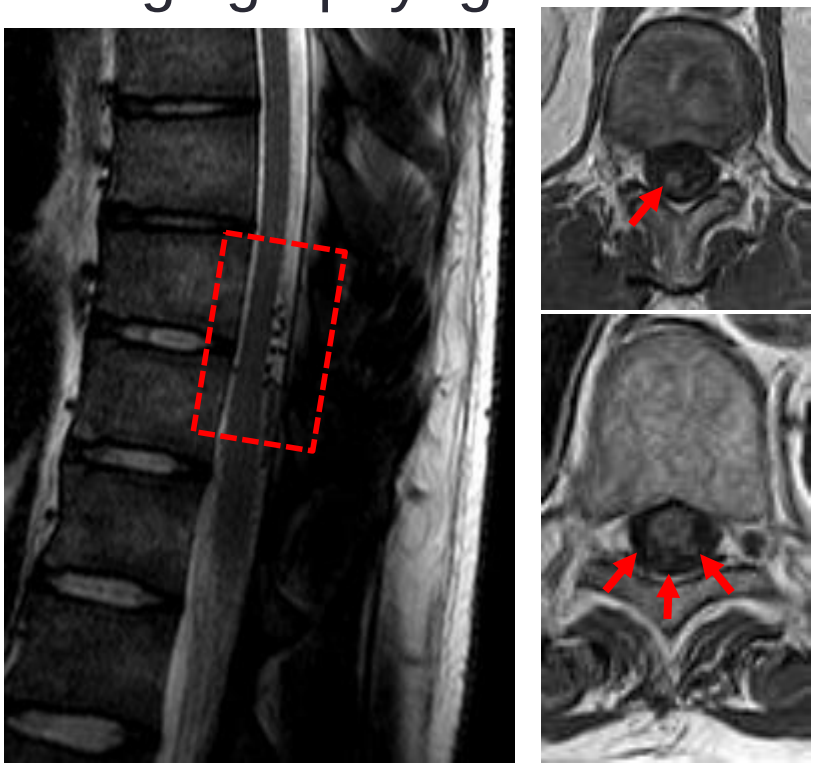
Spinal cord AVMs: clinical features

- ↑♂ 50-years old
- Progressive spastic paraparesis
- Hemorrhage
- Nonspecific spinal pain

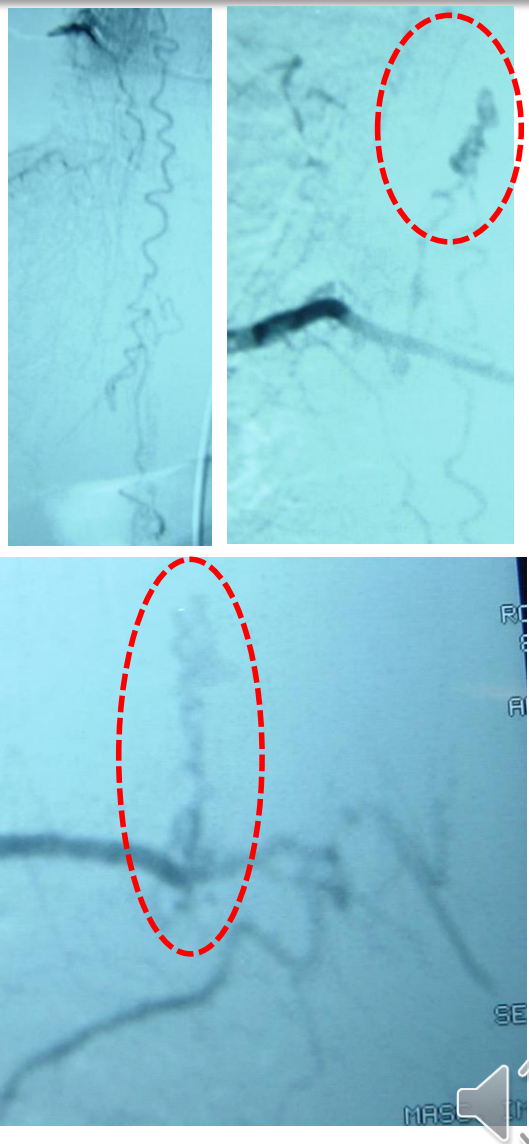


Spinal cord AVM's: diagnosis

- CT-scan useless
- MRI: visualisation of enlarged drainage veins = suspicion
- Angiography: gold-standard



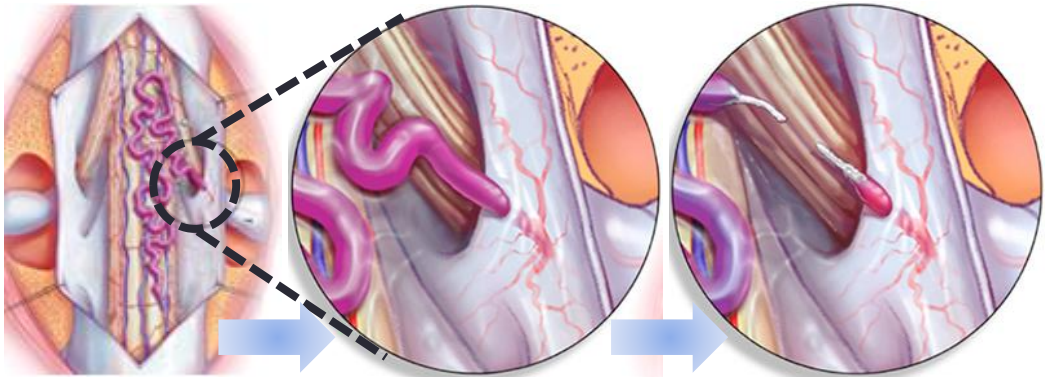
MRI: enlarged veins



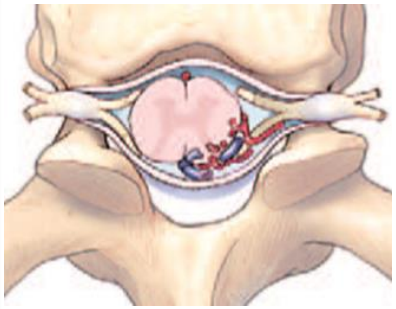
Angiography

Spinal cord AVMs: treatment

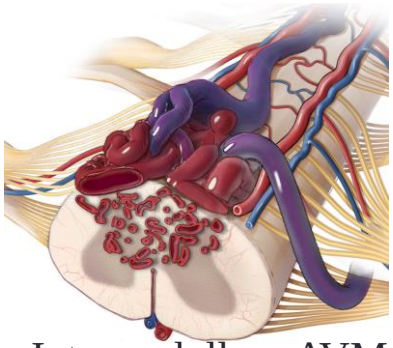
- AV dural fistulas = surgery
- AVMs = endovascular / surgery both according to the case
  - Endovascular preferred
  - Both risk spinal cord injury



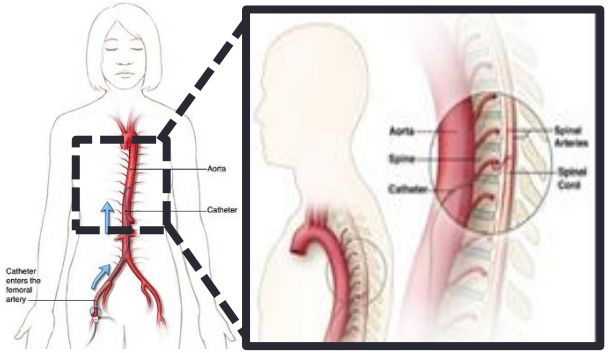
Dural AV fistula = surgery



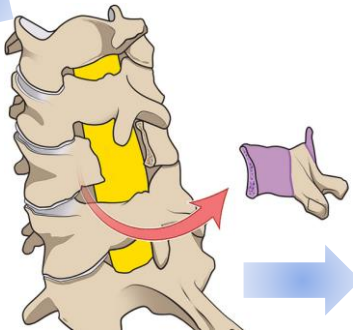
Perimedullary AVM

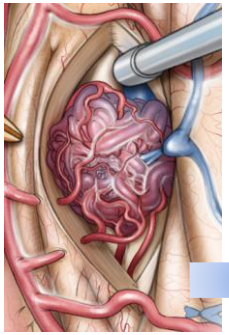


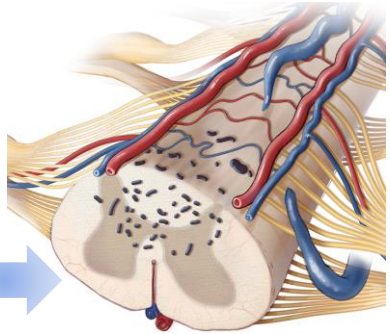
Intramedullary AVM



Endovascular treatment







Surgery

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