ANEURYSM

- pathologic focal blood vessel dilatation caused by **weakness of vessel wall** (Lat. *aneurysma* - dilatation).

1. **Saccular ("berry") aneurysms** (> 90%) – ***rounded outpouchings*** (i.e. neck with dome) at bifurcations of major arteries → rupture into basal cisterns
   - sac is composed of only **intima** and **adventitia**
   - 85% are in anterior circulation.
   - PREVALENCE ≈ 1-2% population.
   - RISK FACTORS: HTN, SMOKING

2. **Fusiform (s. dolichoectatic) aneurysms** (no identifiable neck):
   atherosclerotic - bleed rarely (mass effects are much more common)
   mycotic (infectious) - very friable - greater propensity to bleed; located on distal vessels

3. **Dissecting aneurysms** (e.g. in fibromuscular dysplasia, trauma).

AUTOSOMAL DOMINANT POLYCYSTIC KIDNEY DISEASE (ADPKD) - most common genetic abnormality associated with multiple intracranial aneurysms (prevalence of aneurysms is 7-fold vs. in general population) - all undergo screening MRA.

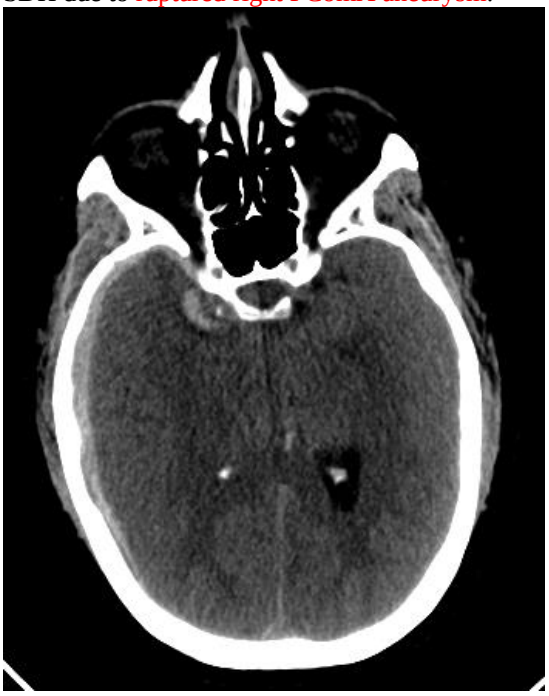
COCAINE, *HEROIN*, *EPHEDRINE*, *METHAMPHETAMINE* – can induce cerebral ***necrotizing angiitis*** → focal arterial ectasias.

Ophthalmic complex aneurysms:
1. **Dorsal ophthalmic** – on ICA above ophthalmic branch
2. **Ophthalmic artery**
3. **Superior hypophyseal artery**

CLINICAL FEATURES

Aneurysm rupture:
a) **minor aneurysmal hemorrhage** (SENTINEL BLEED) - may be clinically silent (or headache with meningeal irritation); may precede rupture with wide variation in latency.
b) **SAH** - most common presentation!
c) **ICH** (more common with distal aneurysms) ← direct rupture of aneurysm into brain, secondary rupture of SAH into brain parenchyma.
d) **IVH** (e.g. BA tip → 3rd → panventricular IVH)
e) **SDH** ← tearing of arachnoid by jet of blood.

SDH due to ruptured right PComA aneurysm:



Risk of rupture: up to 10% per year
- risk factors for aneurysm growth and rupture:
  1) younger age

2) female sex
3) hypertension
4) cigarette smoking
5) larger aneurysm size, esp. with daughter sac or documented growth
6) history of SAH from different aneurysm / family history of SAH
7) posterior circulation

Nonhemorrhagic symptoms (more common with giant aneurysms - diameter > 2.5 cm)
N.B. symptomatic aneurysms have significantly higher risk of rupture (6% per year).

1. **Mass effect**:
   1) **cranial neuropathies** (e.g. CN3 palsy due to PComA aneurysm - requires urgent treatment!!!)
   2) **visual loss** (ophthalmic aneurysm compresses CN2)
   3) **pituitary dysfunction** (intrasellar aneurysms)
   4) **seizures**
   5) **headaches** (aneurysmal expansion, thrombosis, intramural hemorrhage)
   6) **brain stem compression** (respiratory dysfunction, cardiovascular instability)

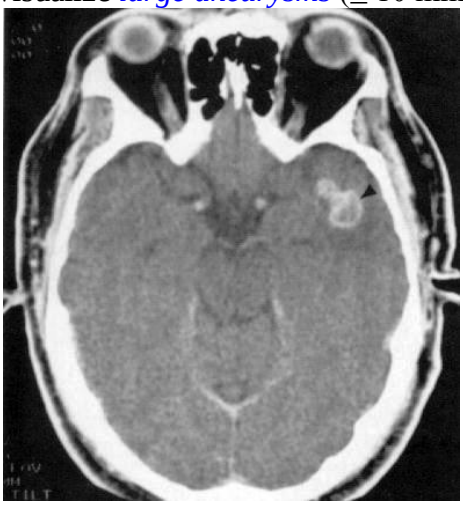
2. **Emboli → TIAs / infarction** H: anticoagulation.

DIAGNOSIS

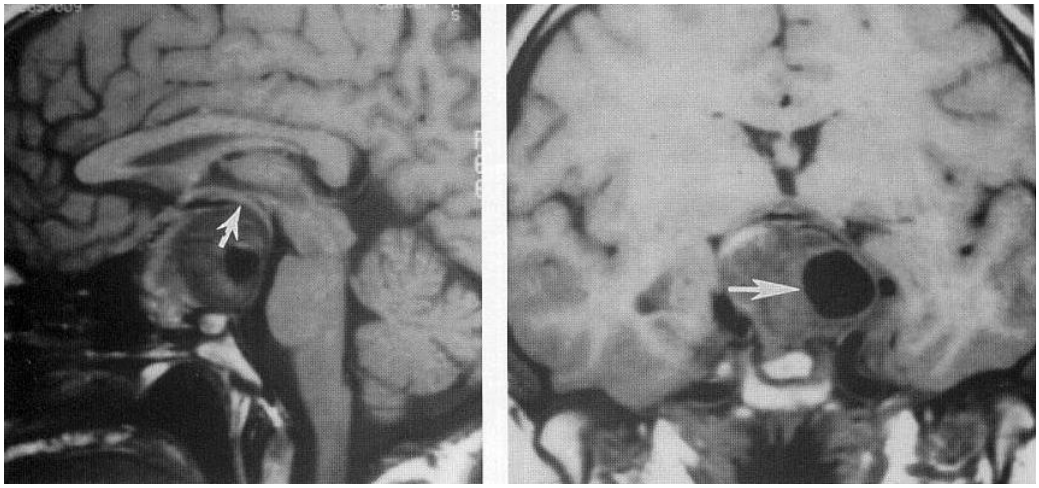
Angiography – criterion standard; preferred in SAH (6-vessel cerebral angiogram = both vertebral and internal carotid arteries + external carotid arteries ← for dural AV fistula as cause of hemorrhage.)
- thrombosed aneurysm – DSA occasionally appears normal.
- not indicated for benign perimesencephalic bleeds.

CTA – preferred for unruptured intracranial aneurysms.
MRA – alternative to CTA (esp. in screening for aneurysm or following after coiling*).
*CTA would have lots of metal artefacts and difficult visualization of any recurrences

Noncontrast CT can visualize *large aneurysms* (≥ 10 mm) or that *contain calcium*:



MRI - aneurysm appearance is highly variable and complex!
Partially thrombosed giant suprasellar aneurysm (arrow in B is giant flow void):



Infundibulum (incomplete regression of fetal vessel);

- most common location is at origin of PComA from ICA (less commonly - origin of anterior choroidal artery).
- smooth funnel-shaped triangular dilatations ≤ 3 mm in diameter, regular in shape, distal vessel exits from apex.
- rarely may bleed
- treat only if accessible during surgery for another reason – wrapping, or placing in encircling clip, or sacrificing artery if it can be done safely (infundibula lack true neck)

SCREENING

- with **MRA** wo (i.e. TOF) every 5-10 yrs

Indications:

1) ≥ 2 immediate **relatives** with intracranial aneurysms
2) AUTOSOMAL DOMINANT POLYCYSTIC KIDNEY DISEASE (ADPKD)

TREATMENT

Treatment indications:

1) young patients *regardless of aneurysm size (esp. > 4-5 mm)*
2) aneurysm symptoms
3) any growth of aneurysm (≥ 1 mm or development of bleb)
4) previous ruptured aneurysm
5) PComA / posterior circulation aneurysms
6) intraluminal thrombus with TIAs

- additional criteria – multiple aneurysms, familial hx of SAH, active smoking, untreated HTN.

For others – **observation** (e.g. annual CTA + stop smoking) – possible indications:

1) < 7 mm incidental aneurysms in anterior circulation have very low rupture risk
2) patient's life expectancy < 5 years – any treatment carries 5% risk of complications; rupture risk is 1%/year – to outweigh risks, patient must live > 5 yrs after treatment!
3) mycotic aneurysms – first treat with a/b (observe shrinkage with angiography q7-10 days; if aneurysm enlarges, it should be attacked surgically).
4) small AVM-related aneurysms (may disappear or shrink after treatment of AVM)

- treat if aneurysm [size, morphology] changes
- ACommA aneurysms are known to rupture at nearly any size – risk proportional to size!

International Study of Unruptured Intracranial Aneurysms (ISUIA) - natural history of unruptured aneurysms of different sizes: 5-year cumulative rupture rates.
the overall 1-yr morbidity and mortality:
12.6% - clipping
9.8% - coiling.

5-yr rupture risk:

Size	**Cavernous ICA**	**Anterior** (ICA, ACA, MCA)	**Posterior** (PComA, PCA, BA, VA)
< 7 mm	0	0 / 1.5	2 / 3
7-12 mm	0	3	15
12-25 mm	3	15	20
> 25 mm	6	40	50

N.B. prior SAH only matters in < 7 mm aneurysms

Annual rupture risk

Size	**No prior SAH**	**Prior unrelated SAH**
< 10 mm	< 0.05%	0.5%
> 10 mm	1%	1%

- most neurosurgeons would treat ant. circulation aneurysms if they are > 4-7 mm (esp. young patient with newly discovered aneurysm, positive family history, smokers, HTN, another ruptured aneurysm vs. leave alone stable 10 mm aneurysm that was discovered 10 years ago in 85 yo patient).

Features to take note of when selecting method:
1. **Dome size**:
   - *large aneurysms* (≥ 15 mm diameter) = lower rates of complete occlusion by coiling.
2. **Neck size**:
   - *broad necks* ≥ 5 mm are associated with incomplete occlusion and recanalization with coiling H: stent or balloon-assisted coiling
3. **Dome : neck ratio** ≥ 2 = higher rate of coil occlusion

SAH

a) 75-90% - **aneurysm** (tear is at dome and ≤ 0.5 mm long); peak age 50-60 yrs; most common site – AComA.
b) 4-10% - **AVM**; peak age 20-30 yrs.
c) 5-15% - **other**

Risk factors for aneurysmal SAH:
1) smoking (risk of SAH increased 3-6-fold); risk increased 6-fold if positive family history
2) hypertension (conflicting data)
3) blacks (2.1 times greater risk than in whites)
4) females (1.24 : 1)
5) 3rd trimester of pregnancy*:
   75% from aneurysms
   25% from AVMs

*recent studies - risk of SAH is not increased during pregnancy.

6) long-term (esp. > 3 years) low-dose **ASPIRIN** has protective effective against SAH.

CLINICAL FEATURES

1. Sudden excruciating **headache** ("thunderclap headache"* - "worst in patient's life")
   - reaches maximum intensity within 1 minute.
   - location of headache is variable
   - present in 97% cases; absence of headache represents amnesia for event.

*N.B. thunderclap headache is not limited to SAH and may be seen with cerebral venous thrombosis, Call Flemming syndrome, crash migraine, benign orgasmic cephalgia - lack of SAH evidence should prompt **MRV**, **CTA**; angiography no longer recommended

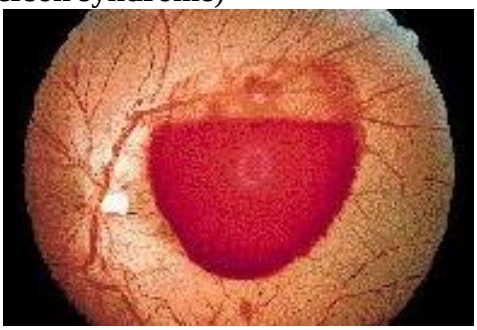
2. Sudden ICP elevation (± transient abrupt generalized vasospasm, seizures) → transient **alteration in consciousness**
   Massive SAH, in contrast to other kinds of stroke, may cause sudden death!

3. Blood induces **STERILE MENINGITIS** → **meningeal irritation** (possible LBP - irritation of lumbar nerve roots by dependent blood)

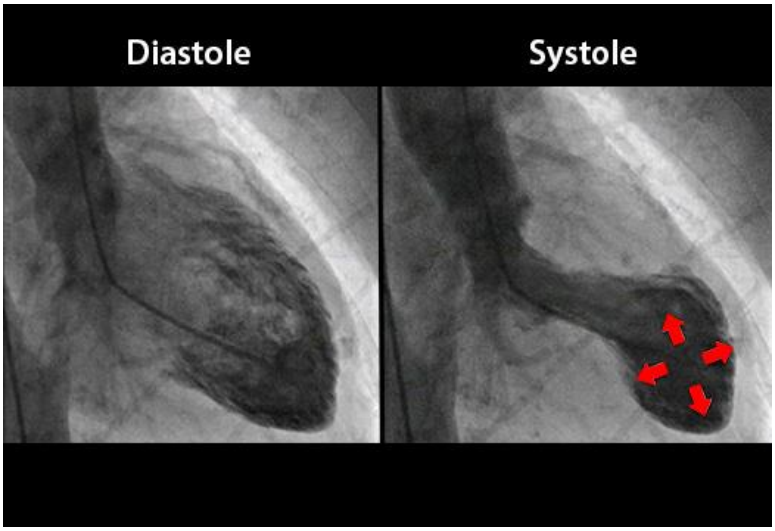
4. **Focal deficits**;
   - CN3 palsy is most frequent (PComA aneurysm).
   - CN6 palsy is due to ICP↑ (false localizing sign).

5. **Seizures** - majority are ***nonconvulsive*** (cannot be detected without EEG)

6. **Ocular hemorrhage** - elevated ICP causing venous hypertension and disruption of retinal veins.
   1) **retinal hemorrhage**.
   2) **subhyaloid hemorrhages** (**Terson syndrome**)



7. **Cardiac Manifestations** - SAH causes circulating catecholamines↑: arrhythmias, subendocardial ischemia, **TAKOTSUBO CARDIOMYOPATHY** (transient stress cardiac syndrome: left ventricular apical akinesis & ballooning - mimics acute coronary syndrome - chest pain, ST elevation, ↑ cardiac enzymes, ↓ ejection fraction + hypotension; H: DOBUTAMINE (for SBP < 90 and low SVR), MILRINONE (for SBP > 90 and normal or increased SVR)



8. Neurogenic pulmonary edema

- unrelated to HHH therapy.
- almost universal in severe SAH.
- H: gentle ***diuresis***, ***dobutamine***, ***PEEP***.

Patients undergoing triple-H therapy can develop **cardiogenic pulmonary edema** with volume expansion!

Hunt & Hess scale (on admission and pre-op):

Grade	Clinical Findings	Survival Rate	Vasospasm Rate
0	unruptured aneurysm		
1	asymptomatic *minimal HA, minimal meningismus*	70%	22%
1A	+ *fixed neurologic deficit*		
2	headache and nuchal rigidity, no neurologic deficit other than CN palsy	60%	33%
3	AMS: lethargy, confusion, mild focal deficit	50%	52%
4	stupor, moderate ÷ severe hemiparesis / early decerebrate	20-40%	53%
5	deep coma / moribund	10%	74%

Add one grade for **serious systemic disease** (HTN, DM, COPD, severe atherosclerosis) or **severe vasospasm** on angiography

0 – unruptured
1 – asymptomatic
2 – HA + nuchal rigidity
3 – AMA
4 – hemiparesis, posturing
5 – deep coma

New version - **World Federation of Neurological Surgeons (WFNS) Scale**:

Grade	GCS	Major focal deficit*	Mnemonics
I	15	-	normal
II	13-14	-	confused
III	13-14	+	aphasia or hemiparesis
IV	**7-12**	+/-	near coma
V	**3-6**		deep coma

*aphasia, hemiparesis / hemiplegia

DIAGNOSIS

Algorithm in suspected SAH:

CT without contrast

positive ↓ **CTA, DSA** ← positive **LP** ↓ negative **CTA, MRV**

Comment: LP allows to decide MRV vs DSA (CTA is done regardless)

Boards: always ask CTA + DSA (i.e. DSA even if CTA/MRA-negative)

N.B. you have only one chance with LP – if you repeat it, it will be false-positive from small amount of blood from first LP

Lumbar Puncture - most sensitive test for SAH - indicated if CT is negative - ***nonclotting hemorrhagic*** CSF with ***xanthochromic*** supernatant (may be absent within first few hours!; 100% present after ≥ 12 hours).

Parameter	"Traumatic Tap"	SAH
Xanthochromia	Absent	Onset: 4-6 hr Still present in 40% at 4 wks
RBC count (serial tubes)	Decreasing	Constant
Blood clot formation	Rapid	Nonclotting

FLAIR MRI is another sensitive way to detect SAH (dramatic hyperintensity in normally hypointense CSF).

blood in basal cisterns, sylvian fissure, or interhemispheric fissure - **saccular aneurysm** rupture;
blood over convexities or within superficial brain parenchyma - **AVM** or **mycotic aneurysm** rupture.

- *medial frontal ICH, lateral ventricle* blood – AComA
- *3rd ventricle* blood – BA tip.
- *4th ventricle* blood – almost pathognomonic for PICA!!!!

MODIFIED FISHER SCALE (MF) - ***risk of vasospasm progressively increases with each grade***:

mF grade	Blood pattern	Incidence of symptomatic vasospasm
0	No SAH, no IVH	0%
1	Focal or diffuse, thin SAH, no IVH	24%
2	Focal or diffuse, thin SAH, **IVH present**	33%
3	Thick SAH, no IVH	33%
4	Thick SAH, **IVH present**	40%

Note: no specified measurement or criteria to define thick vs thin hemorrhage.
Note: Any intraventricular hemorrhage, no matter how small, is counted.

4-vessel DSA - indicated in all patients after SAH diagnosis!

- signs of ruptured aneurysm (if ≥ 1 aneurysm is found - which aneurysm needs to be treated acutely):
    1) **contrast extravasation** (pathognomonic but extremely rare)
    2) **larger aneurysm** will be site of rupture more frequently than smaller one – most important (practically) criterion!
    3) **irregularly shaped** aneurysm (lobulation, smaller daughter dome)
        Murphey's "teat," "tit," or "excrescence" - Dr. Francis Murphey (the Semmes-Murphey Clinic in Memphis) - ***focal sacculation on the dome of an aneurysm*** may be angiographic evidence of a culpable aneurysm.

In 15-20% of spontaneous SAH patients, DSA is negative! (beware occult aneurysm - compressed by hematoma or did not opacify because of vasospasm or clot) → repeat angiography (1-2 wks later); indication for 3rd angiogram - blood more widely distributed (diffuse) – 3rd angiogram 1-6 months later.

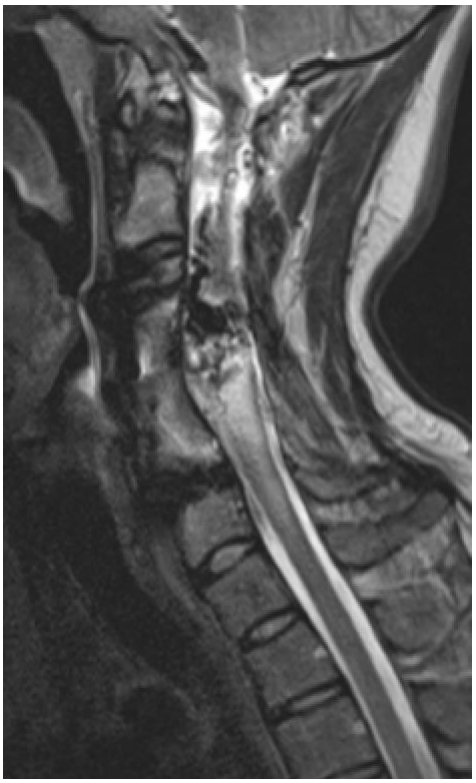
3 angiograms still give 1-4% false-negatives

Before calling angiogram negative, one must:

1) visualize **both PICA origins**.
2) visualize **AComA**.
3) see no **infundibulum** co-localized to SAH.

After first negative angiogram, order **cervical MRI** to rule out cervical AVM / AVF.

Cervical dural AVF:



Radiographic patterns of DSA-negative spontaneous SAH

1. **Perimesencephalic** (30-40%) – one* DSA
2. **Cortical** SAH → CTA, if negative → one* DSA (low chance of aneurysm but look for vasculitis, DAVF)
3. **CT-negative LP-positive** = same as cortical SAH
4. **Diffuse** SAH (prognosis ≈ aSAH) → CTA, if negative → DSA, if negative → second DSA 7 days later, consider MRI (look for cavernoma) → if all negative, some experts do third DSA

*i.e. no additional serial imaging

Summary:

perimesencephalic → one DSA;
cortical → CTA → one DSA
diffuse → CTA → DSA → DSA → MRI → DSA

TREATMENT

- **strict bed rest** in ICU (no out of bed for any reason for 2 weeks); low level of external stimulation.
- **SBP goal** < 130-140 (< 160 when aneurysm secured); up to 180-220 (if secured and in vasospasm) - consider **A-line**.
  - early aggressive fluid therapy to head off cerebral salt wasting: NS + 20 mEq KCl/L at 2 ml/kg/hr (typically 150 ml/hr)

  Always avoid HYPOVOLEMIA!

  – maintain **BP** in range that allows for sufficient ***cerebral perfusion**** yet limits risk of ***rebleeding***;
  – Ca-antagonists (NICARDIPINE**)/ β-blockers (LABETALOL) / HYDRALAZINE IV → start long-acting ACEI.
  – avoid nitrates (NITROPRUSSIDE, NITROGLYCERIN) which elevate ICP;

*state of consciousness may be used as guide to level of cerebral perfusion - administer hypotensive medications up to level that patient begins to experience drowsiness.

**drug of 1st choice!

- KEPPRA – continue for 7 days after aneurysm is secured (other experts do not continue after aneurysm is treated – AEDs may give poorer outcomes)
- vasospasm prophylaxis (for 21 d or until patient is discharged home in good neurological condition):
  1) **NIMODIPINE** 60q4 for 21 days
  2) PRAVASTATIN 40 mg/d – no clinical benefit
  3) MAGNESIUM SULFATE 64 mmol/d IVI for 14 days – safe but no benefit!
  4) CLAZOSENTAN – no clinical benefit
- if GCS < 14, place **EVD** (continuous drain at 10 or even 20; when aneurysm secured – drain at 0) + **LD** (at 5 mL/hr) esp if nonobstructive HCP (no IVH).
  – don't start challenging EVD until day 7.
  – don't start challenging EVD if on vasopressors.
  – don't start challenging EVD until CSF output becomes < 100 mL/8 hr.

EVD side:

anticipated clipping – opposite side of aneurysm
anticipated coiling – nondominant side

– if there is ***delay in aneurysm treatment*** plus significant risk for rebleeding (and no compelling medical contraindications) → short-term (< 72 hours) TRANEXAMIC ACID or AMINOCAPROIC ACID (Class IIa, Level B)

Other

No nicotine patch for smokers!

FAMOTIDINE + ONDANSETRON + DOCUSATE
FIORICET? (caffeine is vasoconstrictive!) / IBUPROFEN / KETOROLAC / OXY / FENTANYL ± DEXAMETHASONE 2Q8, CYCLOBENZAPRINE
DVT prophylaxis

ANEURYSM TREATMENT TIMING

- all ruptured aneurysm are treated within 72 h (ideally within 24 h) of SAH; even patients with H&H grade 5
  - one exception – *ruptured basilar tip aneurysm* (some experts recommend wait for 10-14 days)
  - rare exceptions - hemodynamic instability, extreme old age, clinical condition approaching brain death.
  - in absence of compressing hematoma, it is *not necessary to operate during nighttime*.
  - if cannot treat (e.g. presents **after 10 days** - worse outcome with any treatment) → TXA 1 g q6hr for up to 3 days.
  - avoid clipping during days 3-10 when maximal vasospasm is likely – better coiling!

Although operative mortality (of early surgery) is higher, overall mortality rate is lower (than of delayed surgery)

H&H grade 5:

a) just SAH → EVD only; if status improves → rediscuss aneurysm treatment
b) big ICH, thick SAH clot → be aggressive

ANEURYSM TREATMENT METHODS

N.B. whereas coiling is somewhat safer than clipping for both ruptured and unruptured aneurysms (at least in acute perioperative period), clipping is slightly more durable.

Long term outcomes ar similar - decision to ***clip*** or ***coil*** should be made on individual basis – don't force one or another method!

Practice guideline: Aneurysm treatment decisions:
Level C: Treatment decisions should be multidisciplinary (made by experienced cerebrovascular and endovascular specialists) based on characteristics of the patient and aneurysm.
Level C: **Clipping** may receive increased consideration in patients presenting with large (> 50 ml) intraparenchymal hematomas and MCA aneurysms.

N.B. MCA aneurysm always clipping unless CI (e.g. uncorrectable coagulopathy)

Level C: **Coiling** may receive increased consideration in the elderly (> 70 yo), in those presenting with poor-grade WFNS (IV/V), and in basilar apex aneurysms.
Level B: For patients with ruptured aneurysms judged to be technically amenable to both coiling and clipping, coiling should be considered.

Comparison (coiling vs. clipping)
Coiling carries lower risk of vasospasm (due to endothelial damage during angiographic manipulation → less endothelin release?)

Long-term rebleed rates might be slightly higher in **coiling**; thus, in patients < 40 yo **clipping** might be better.

Clipping better eliminates symptomatic mass effect created by aneurysm.

Coiling is easier for posterior circulation.

Seizures – no consensus which method is better (ISAT suggests **coiling** is better).

Hydrocephalus – suggestion (unconfirmed by many studies) – **clipping** is better.

Pregnancy - no studies have directly compared clipping versus coiling.

Ruptured Aneurysms - four randomized controlled trials: ISAT, BRAT, Finnish, Chinese.

International Subarachnoid Aneurysm trial (ISAT)

- class 1 evidence - greater impact than any other study → huge shift from surgery to endovascular.
- rates of rebleeding are higher after ***coiling*** (no statistical significance at 1 yr) but poor outcomes (mortality, dependence) are more common after ***clipping*** (coiling gives 23% relative risk reduction for poor outcome):

  1-yr disability or death (vs. unruptured aneurysms from ISUIA)

  30% (13%) - clipping

  24% (10%) - coiling

  - ISAT investigators - surgery may be better in **< 40-50 yrs** group (poor outcome at 1 year is much less in surgical group).
  - best modality is usually apparent (only 20% aneurysms in ISAT had true clinic equipoise).
  - what happens after 1 year (rebleeding)?

CLIPPING

see p. Vas25 >>

Indication:

1) wide aneurysm neck (as is often in MCA bifurcation aneurysms) - threshold for choosing coiling vs. clipping is 1 : 2 neck to corpus ratio or > 4 mm neck; alternative – stent-assisted-coiling

   N.B. if aneurysm is at vessel bifurcation (would need two stents in Y or X configuration) – use specialized ***bifurcation stents*** (pCONus, PulseRider) or ***endosaccular flow disruptors*** (e.g. WEB)

   N.B. flow diverters (Pipeline) are not an option at bifurcations!
2) vessel branching off from dome
3) MCA aneurysms – branch near the neck.
4) associated SDH/ICH (> 50 mL) that needs evacuation.
5) younger patient

Summary

Plan approach so can access aneurysm neck before dome!

CSF drainage

Femoral sheath

KEPPRA

type and cross 2 U pRBC

Radiolucent head-holder (lidocaine at pin sites!)

Microscope

EEG, MEP, SSEP

Wash clot off, open basal cisterns

Parent vessel preservation

Proximal vessel control!!! (prep neck)

Access aneurysm neck before dome! (i.e. check imaging - which direction aneurysm is pointing)

Temporary clip on parent vessel!!! - ***mobilize and inspect aneurysm in all directions*** - for perforating vessels

- if clip slips down onto base (occludes parent vessel and/or branch) H: add second tandem clip above it, then remove first clip.

TIM (THIOPENTAL*-ISOFLURANE-MANNITOL) ± hypotension-hyperventilation-hypothermia

*up to burst suppression

Doppler → IC green angio → completion DSA

if patient wakes up with new deficit → STAT DSA – gives much more reliable info than CTA (plus, CTA wastes more time)

ANTERIOR CIRCULATION aneurysms - pterional (fronto-temporal) - supine with head rotation:

PComA – 30 degrees
MCA – 45 degrees
AComA* – 60 degrees
*alternatives – subfrontal, anterior interhemispheric
A2 and more distal aneurysms - anterior interhemispheric

POSTERIOR CIRCULATION aneurysms:

- **BA bifurcation (basilar tip)** – depends on BA bifurcation height:
  - *above dorsum sellae* - modified pterional, right side preferred (transsylvian - Yasargil's approach), subfrontal through 3rd ventricle via lamina terminalis
  - *below dorsum sellae* – subtemporal (classic Drake's approach) with splitting tentorium behind CN4
- **BA trunk** - posterior subtemporal, OZ
- **lower BA, midline VA** - lateral suboccipital transcondylar
- **PICA** - far lateral inferior.
- **VA where it pierces dura** - midline suboccipital.

Measures to avoid INTRAOPERATIVE ANEURYSMAL RUPTURE:

- *prep and drape neck for rapid carotid proximal control* (absolutely for **ophthalmic** aneurysms, strongly recommended for **PComA** aneurysms)
- check **CTA** preop (e.g. if fetal circulation, cannot sacrifice PComA)
- type and cross 2 U **pRBC**.

1) pain control
2) minimal brain retraction
3) lumbar/EVD CSF drainage
4) completely mobilize aneurysm
5) at final stages of aneurysm approach:
   a. hyperventilation
      i. hypothermia 34°C
      ii. systemic hypotension rarely helpful and increases risk of ischemia
   b. focal hypotension with **temporary clips** on parent artery ± **aspiration of aneurysm sac**
      - if occlusion > 5 minutes, if long segment of ICA is trapped - administer 5000 U HEPARIN IV to prevent thrombosis.
      - if requires > 20 min occlusion, terminate surgery and consider either coiling or come back with deep hypothermia + circulatory arrest or bypass grafting around segment to be occluded.

Management:

1. Alert anesthesia and call for blood
2. Tamponade with cottonoid and apply suction
3. Place temporary clip for proximal control / temporary aneurysm clip
4. Uncontrollable torrential bleeding → IV ADENOSINE to induce brief (30–45 seconds) cardiac arrest → dissection and inspection of rupture point

SYLVIAN FISSURE DISSECTION

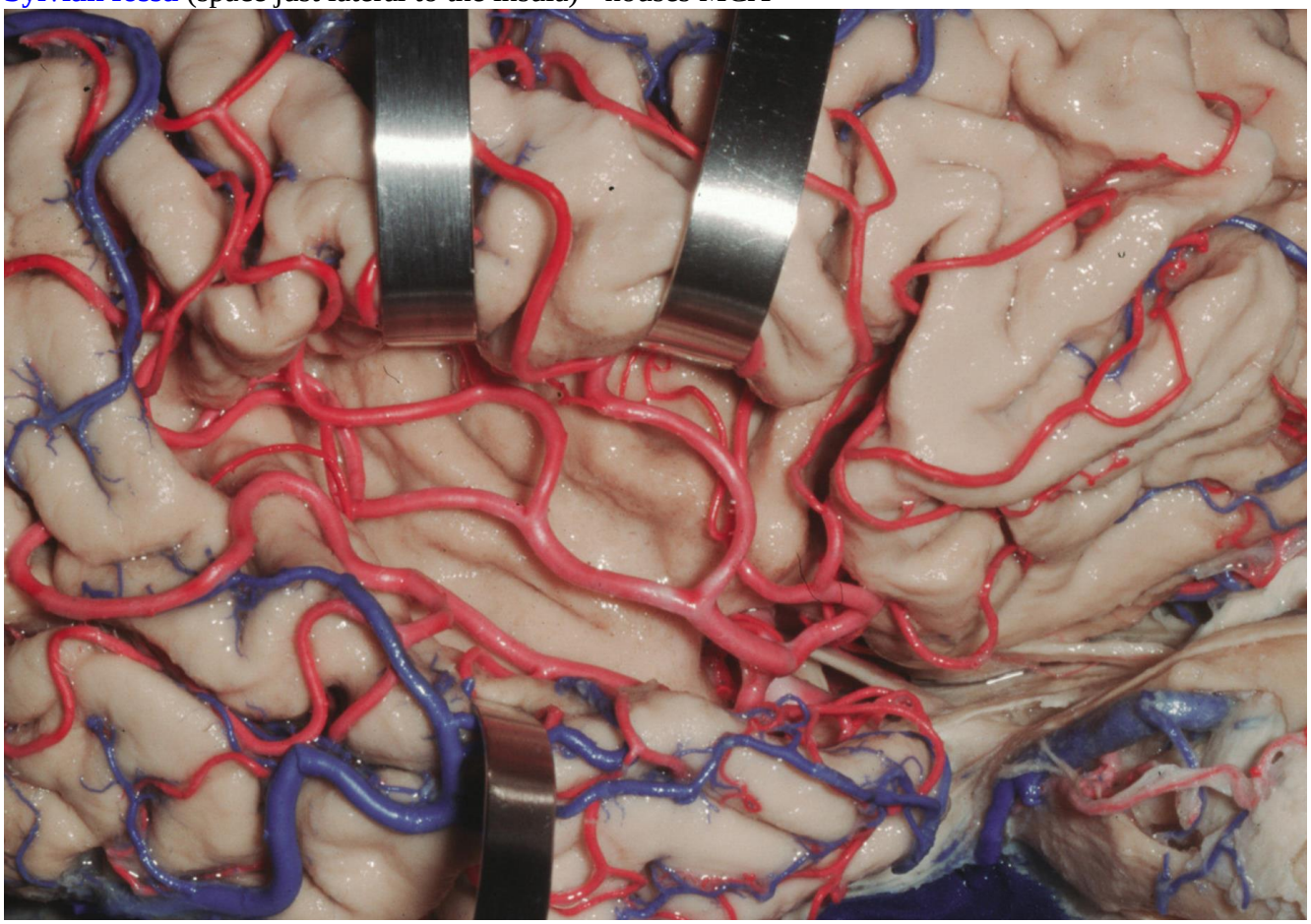
- for MCA aneurysms, for Yasargil approach to basilar tip aneurysms, for insular tumors - Sylvian fissure needs to be atraumatically split!
- "easy fissures" in older patients with brain atrophy vs "difficult fissures" in young patients with SAH.

ANATOMY

- dense network of pia-arachnoid fibers (around arteries, veins, pial surfaces of adjacent opercula and insular gyri) of the entire fissure.
- MCA is divided in four segments: M1 courses posterior and parallel to sphenoid ridge, M2 resides on limen insula, M3 segment spreads over opercula, and M4 segment is composed of convexity branches

N.B. dissection should be conducted along inferior - anterior aspect of M1 to prevent injury to lenticulostriate arteries!

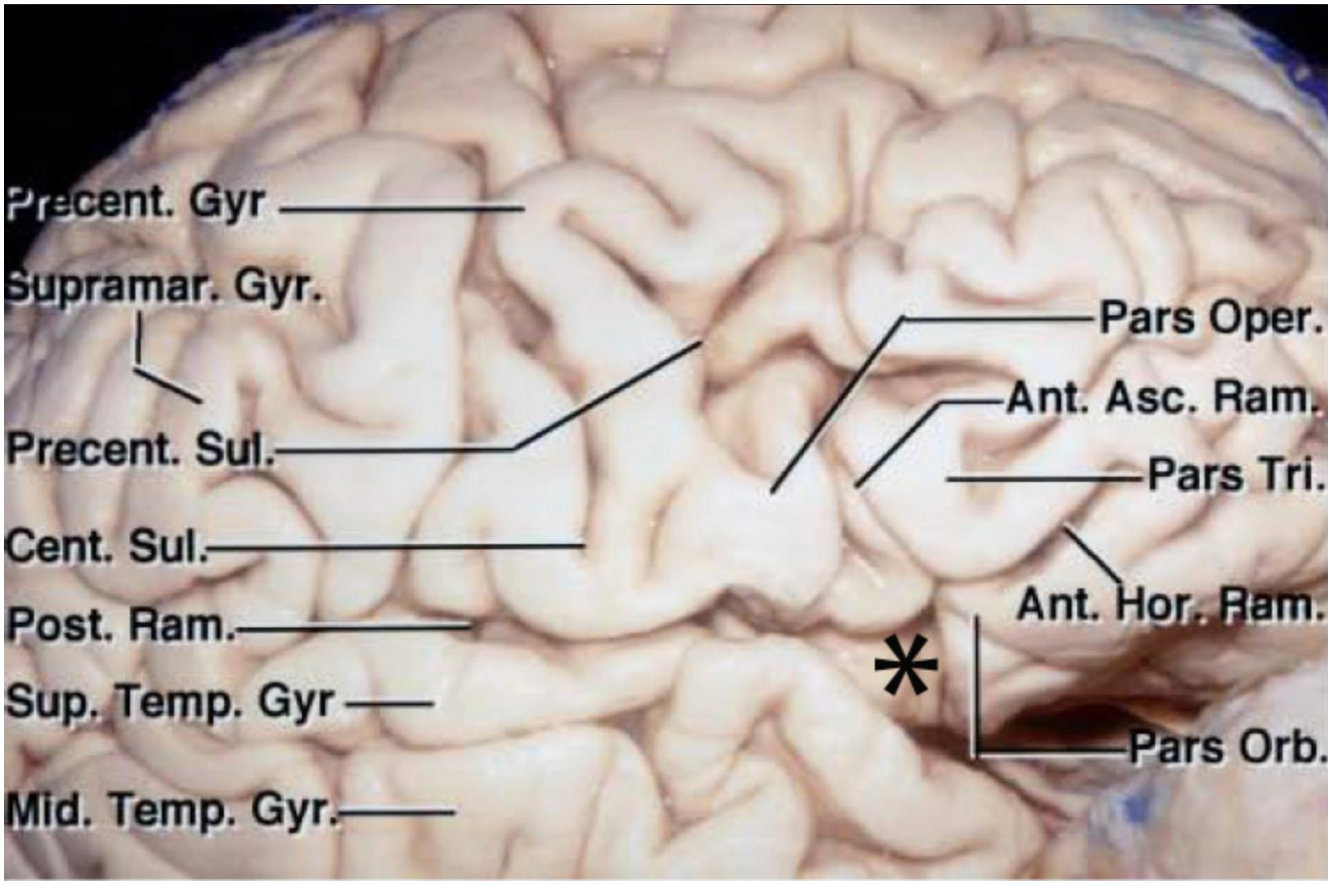
Sylvian cistern contains three parts:
Sylvian fissure – see below
Opercular sulci - between the opercular surfaces of lateral orbital, inferior frontal, inferior parietal, and superior temporal gyri – sulci are often oblique and curved - makes fissure dissection a demanding task requiring the surgeon's patience
Sylvian fossa (space just lateral to the insula) - houses MCA



Sylvian fissure is divided into compartments (separated by Sylvian point):
Anterior proximal (stem, vallecula) - originates inferiorly at the anterior perforated substance and extends laterally to the temporal pole; stem reaches laterally and divides into the ascending, horizontal, and posterior rami; the confluence of these rami has been referred to as the "Sylvian point."

- horizontal and ascending rami divide the inferior frontal gyrus into the pars orbitalis, pars triangularis, and pars opercularis
- vallecula houses ICA bifurcation and limen insula, where MCA bifurcates
- vallecula also contains lateral lenticulostriate perforators and deep Sylvian vein

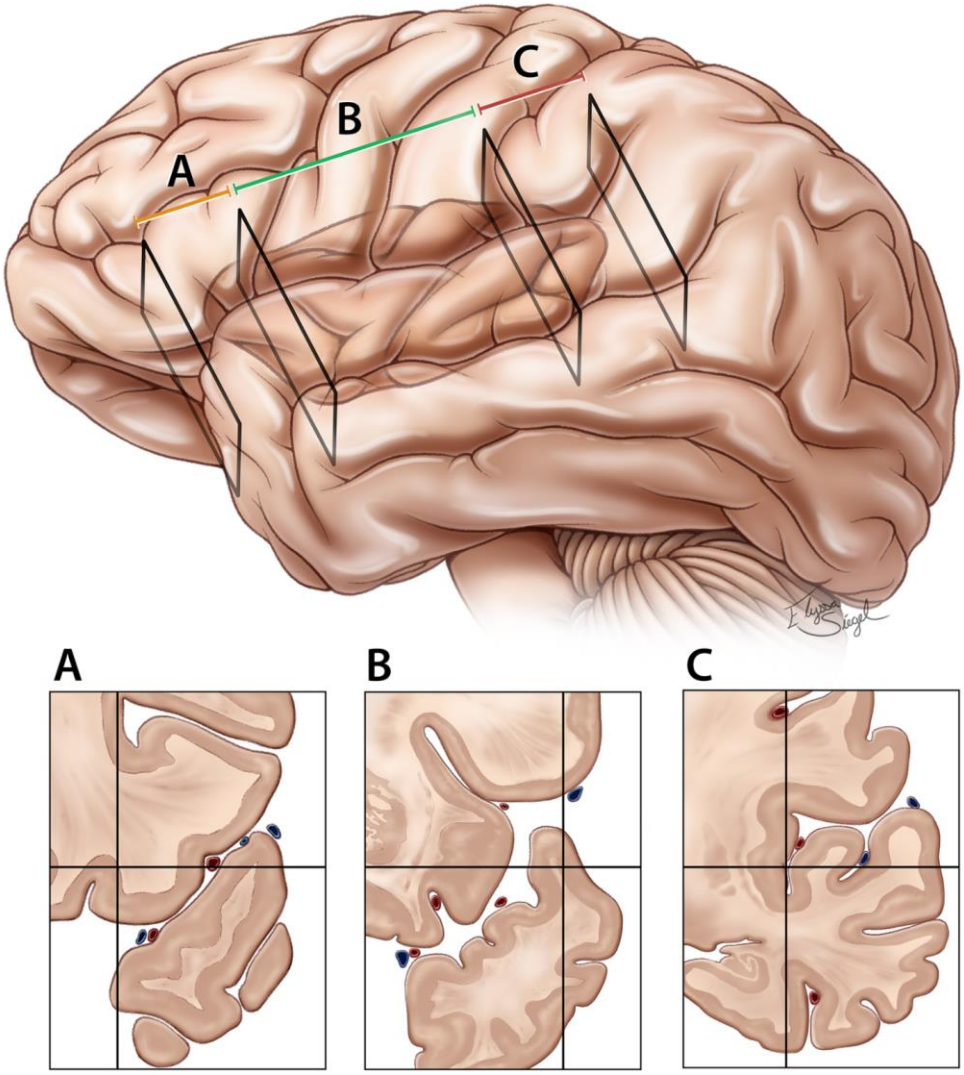
Posterior distal (insulo-opercular).



Proximal (sphenoidal) section (A) also includes planum polare - pial surfaces highly adherent, requiring gentle microdissection (paucity of vessels allows adherence of the frontotemporal opercula)

Middle (insular) section (B) is 6-7 cm in length - extends from limen insula to posterior insular point - sulci are less interdigitated simplifying fissure dissection.

Posterior (retroinsular) section (C) is short (4–5 cm) but deep - covered by complex interdigitations of supramarginal, transverse temporal, and transverse parietal gyri - dissection especially challenging (and may be risky in dominant hemisphere!) - dissection here is necessary only for large insular tumors, M2/M3 aneurysms, and giant MCA bifurcation aneurysms.



N.B. fissure is not perpendicular to the insula but is oblique!

SURGERY

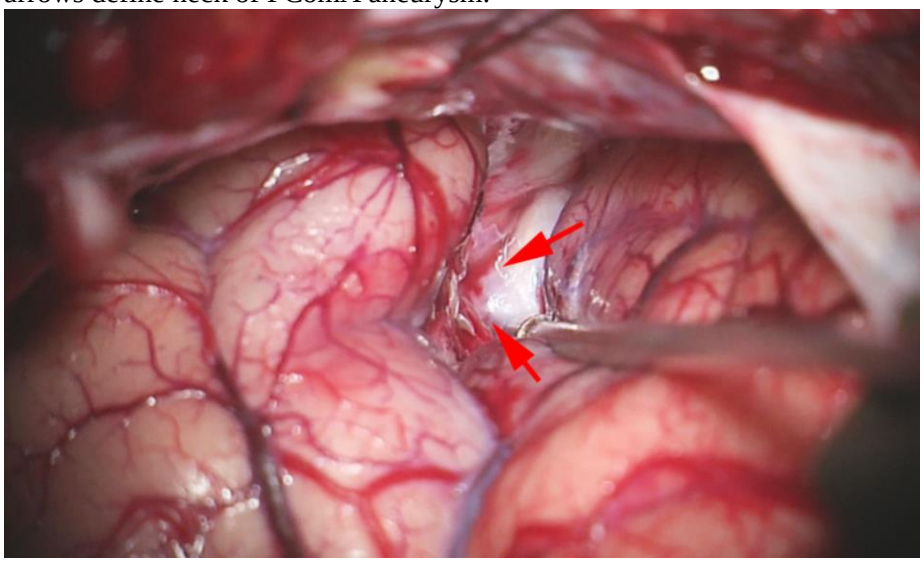
- amount of splitting:
    a) large insular tumors or giant MCA aneurysms → fissure must be dissected as widely as possible to the level of superior and inferior peri-insular sulci.
    b) anterior skull base tumors, anterior circulation aneurysms → dissect only the anterior limb of the Sylvian fissure, exposing the cistern just anterior to the M1
- direction of splitting
    a) **distal-to-proximal**: working from lateral aspect of the fissure medially (does not provide early proximal control!)
    b) **inside-to-outside**: starting at where ICA penetrates fissure and working laterally (easier when prolific veins overlie the junction of the frontal and temporal lobe).
- pterional craniotomy (or modification); fissure dissection is easiest with head rotated 30 degrees!
- open dura.
- brain relaxation may be immediately achieved by gentle elevation of anterior frontal lobe and opening arachnoid membranes over opticocarotid cisterns.
    - ***small cotton ball*** inserted underneath frontal lobe maintains CFS outflow during fissure dissection
- cover brain surface with ***pieces of moist Telfa*** - to avoid heat injury from intense microscope light.
- Sylvian fissure is covered along its entire length with a *thick band of arachnoid membrane*.

- identification of fissure (fissure is 10-14 cm in length, longer than often appreciated; subtle forms of cortical malformation can transform the fissure into a series of sulci, making the operator's job very difficult):
    - **superficial Sylvian veins** outline course of fissure.
    - recognition of **M4 branches** exiting fissure onto cortex.
- ***arachnoid bridging*** from frontal to temporal lobe (over anterior sylvian fissure) should be meticulously divided using microscissors, No. 6 Rhoton dissector, and bipolar cautery.

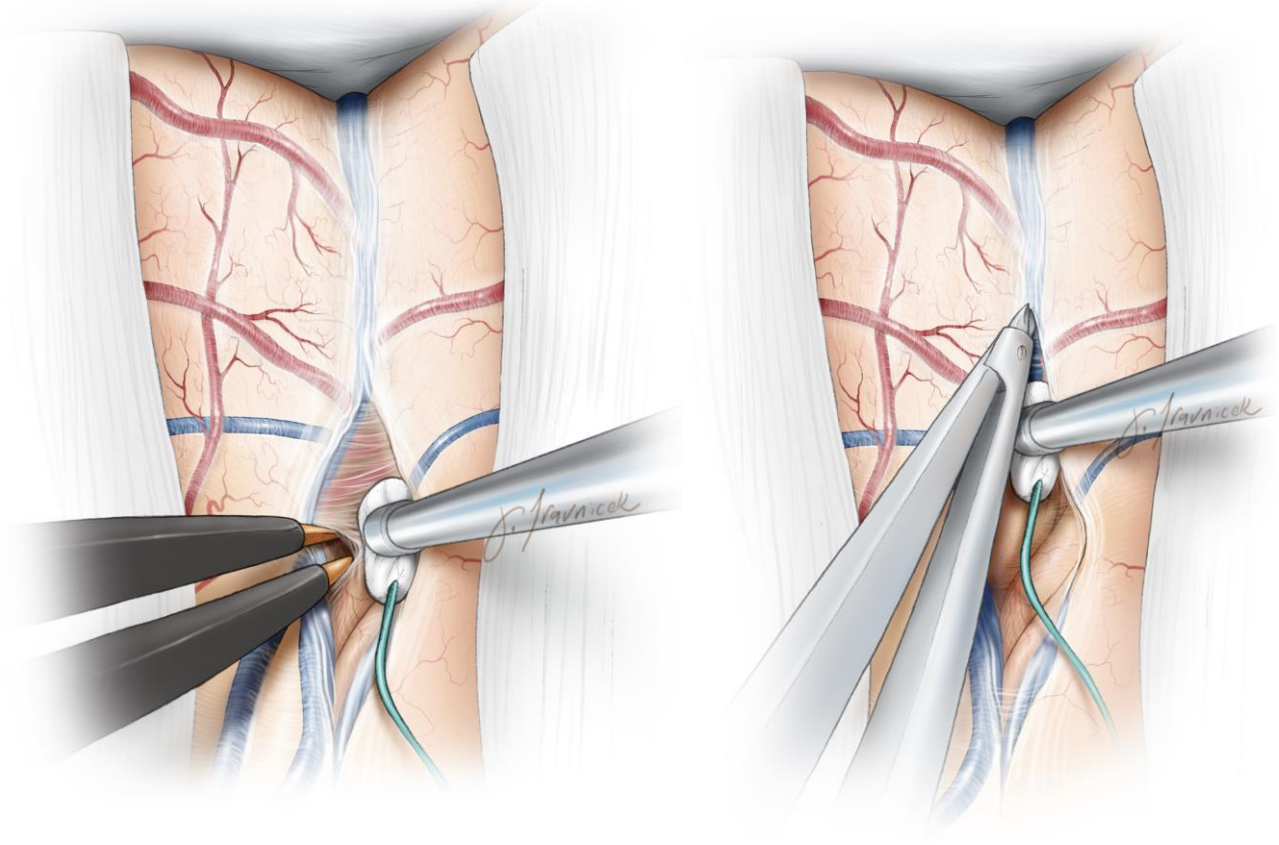
"Inside-to-outside" technique

- round blade (beaver knife), using two micro pick-ups (e.g. jeweler's forceps with fine tips) tear arachnoid apart in avascular spot → work with low power suction and Rhoton dissectors / microscissors → soft, moist, cotton pledgets or balls are gently glided between pial membranes of adjacent gyri → gradual and gentle compression over pledgets by the fine suction tube, in addition to spread action of bipolar forceps, will allow gradual extension of opening.
- if slight pial bleeding is encountered, hemostasis is achieved using a minute piece of Gelfoam covered with a small cotton pledget (bipolar coagulation is avoided!)
- start at **anterior one-third** of the fissure, at Sylvian point (Sylvian stem, proximal fissure: between ICA bifurcation and pars triangularis), exposing M1 and medial 2 cm of M2 and insula:

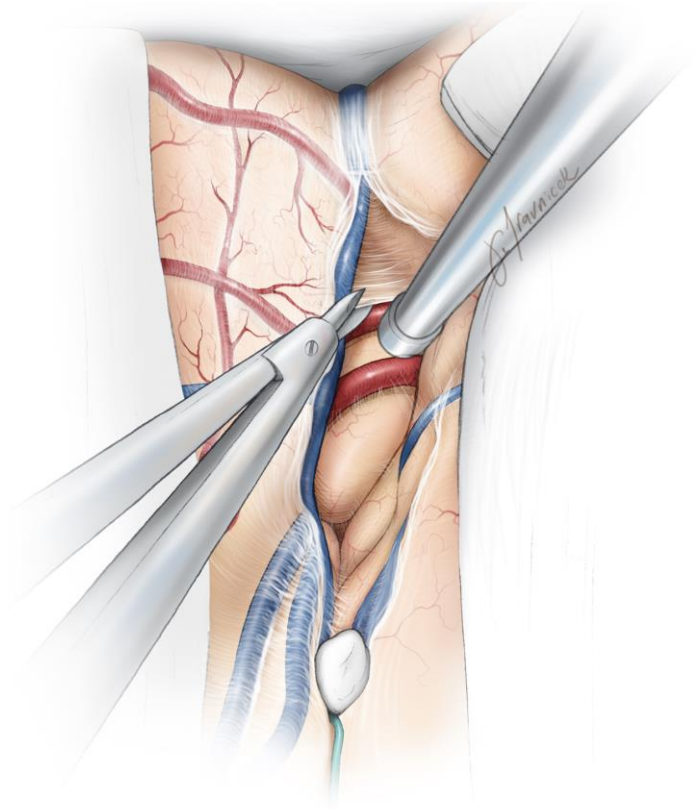
arrows define neck of PComA aneurysm:



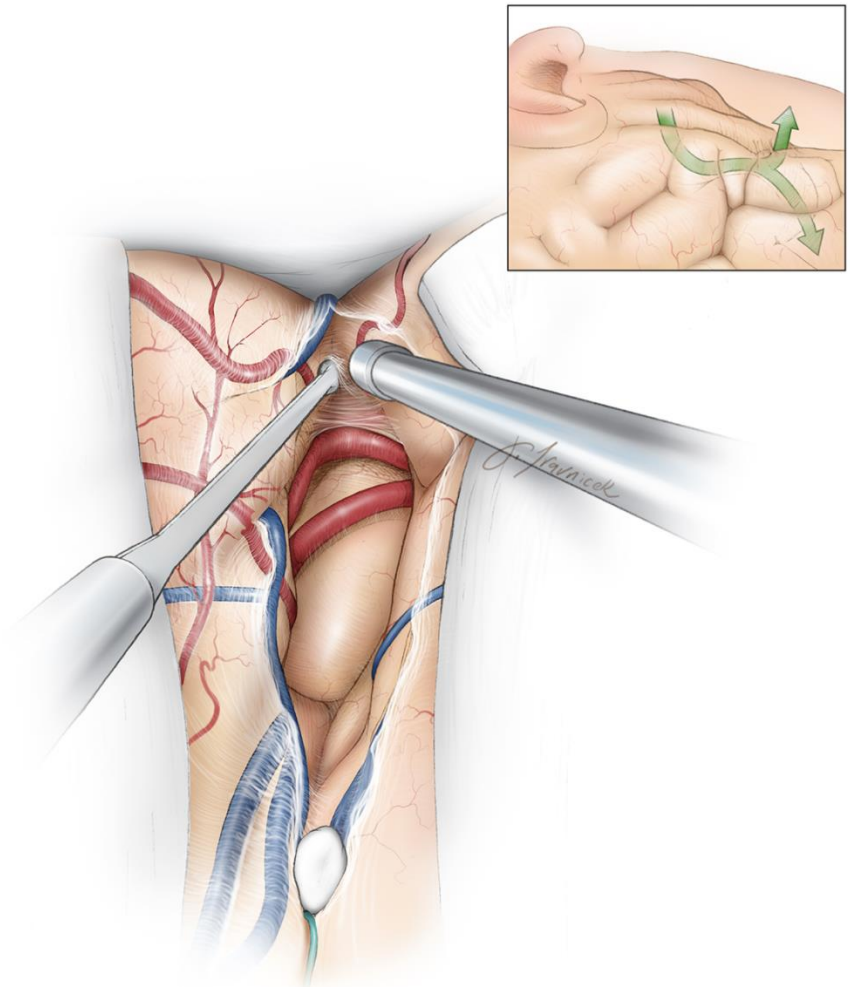
note use of small cotton pledget (avoids direct contact between suction tip and pial surfaces):



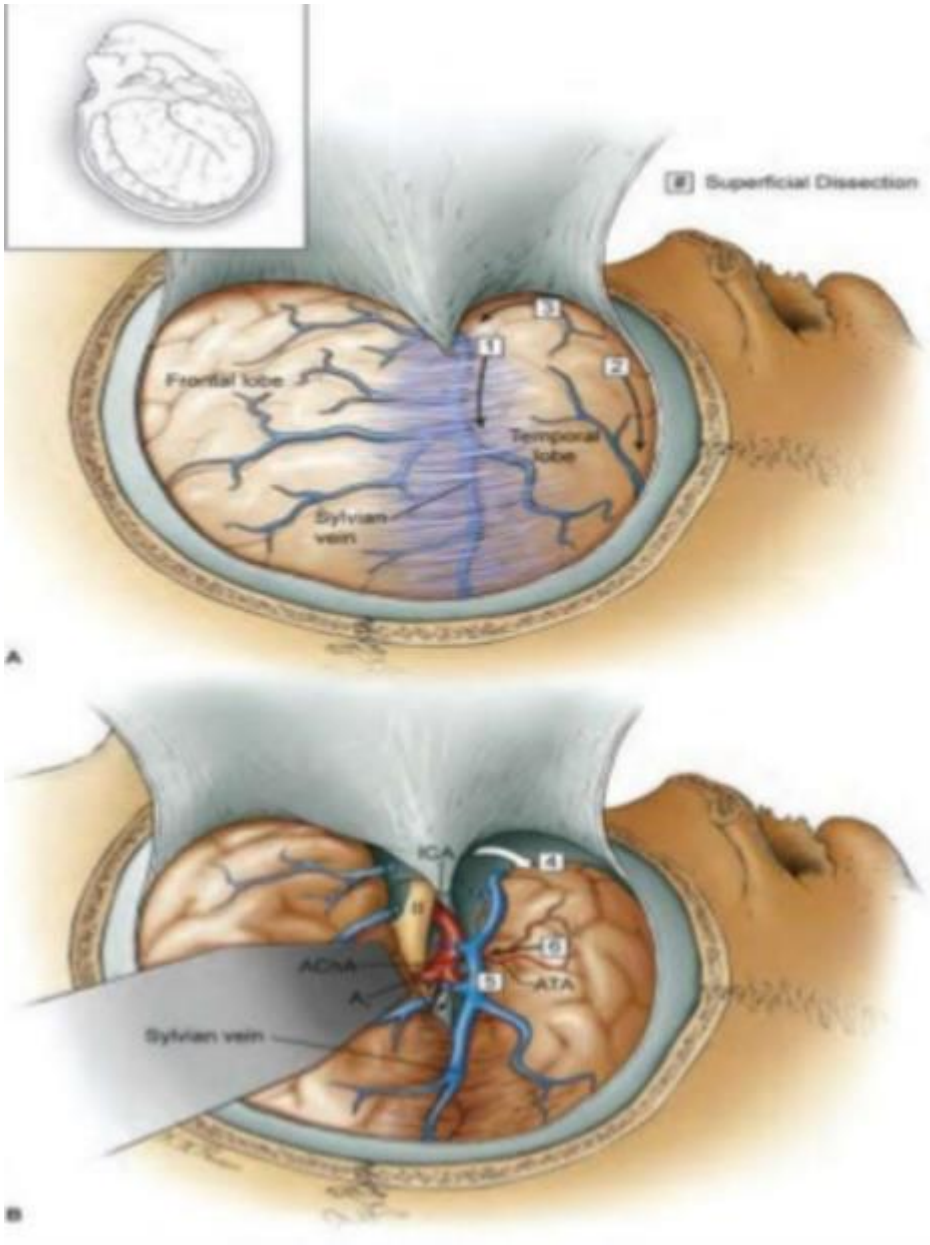
- Sylvian fissure is more readily split by conducting *dissection above* (rather than below) *superior Sylvian vein* (vein travels 4 mm below the fissure in > 80% patients).
    - fissure should preferably be *opened on frontal side of veins* - veins will not cross the fissure when frontal lobe in elevated.
    - if > 1 superficial Sylvian vein is present, *dissect between two veins*.
- resume Sylvian fissure opening at natural superior retraction of the *apex of pars triangularis* along Sylvian point - provides the widest trans-fissure corridor and will expose insular apex (important landmark for surgical orientation).
- continue inside-to-outside technique: dissection from Sylvian point is extended to the depth of the fissure (identify distal MCA branches on surface of insula):



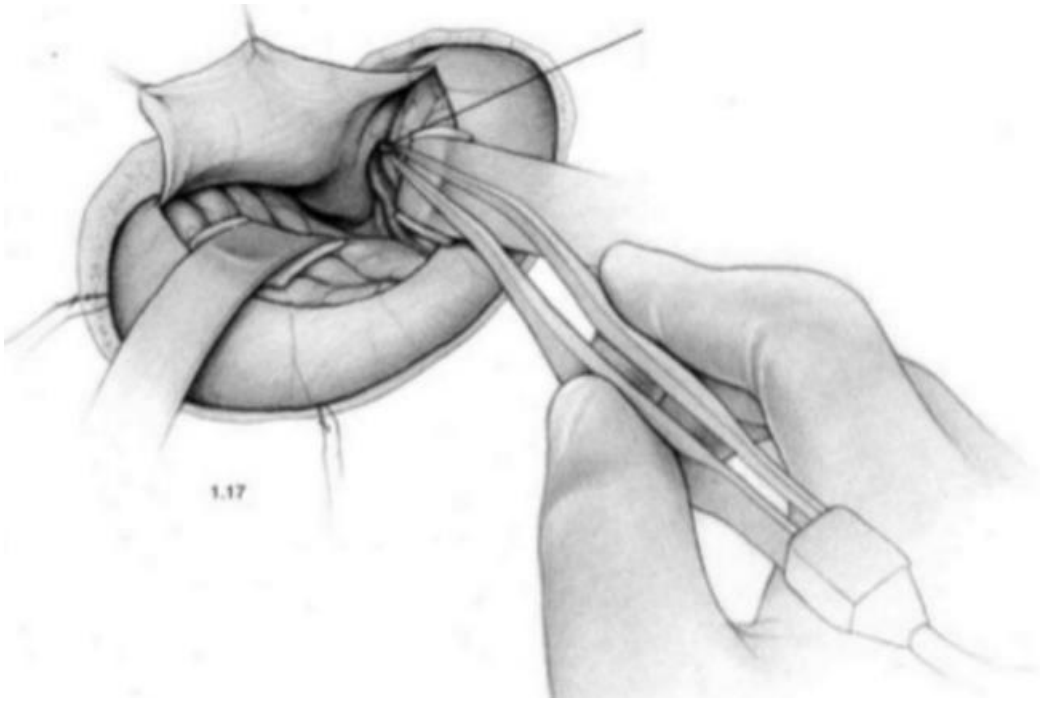
- pursue dissection from deep to superficial (Yasargil “radial splitting of peeled orange wedges” - it is difficult to separate edges from the outside, but it is easy to put finger into the middle of orange and radially separate the wedges without compressing the individual slices and releasing their juice):

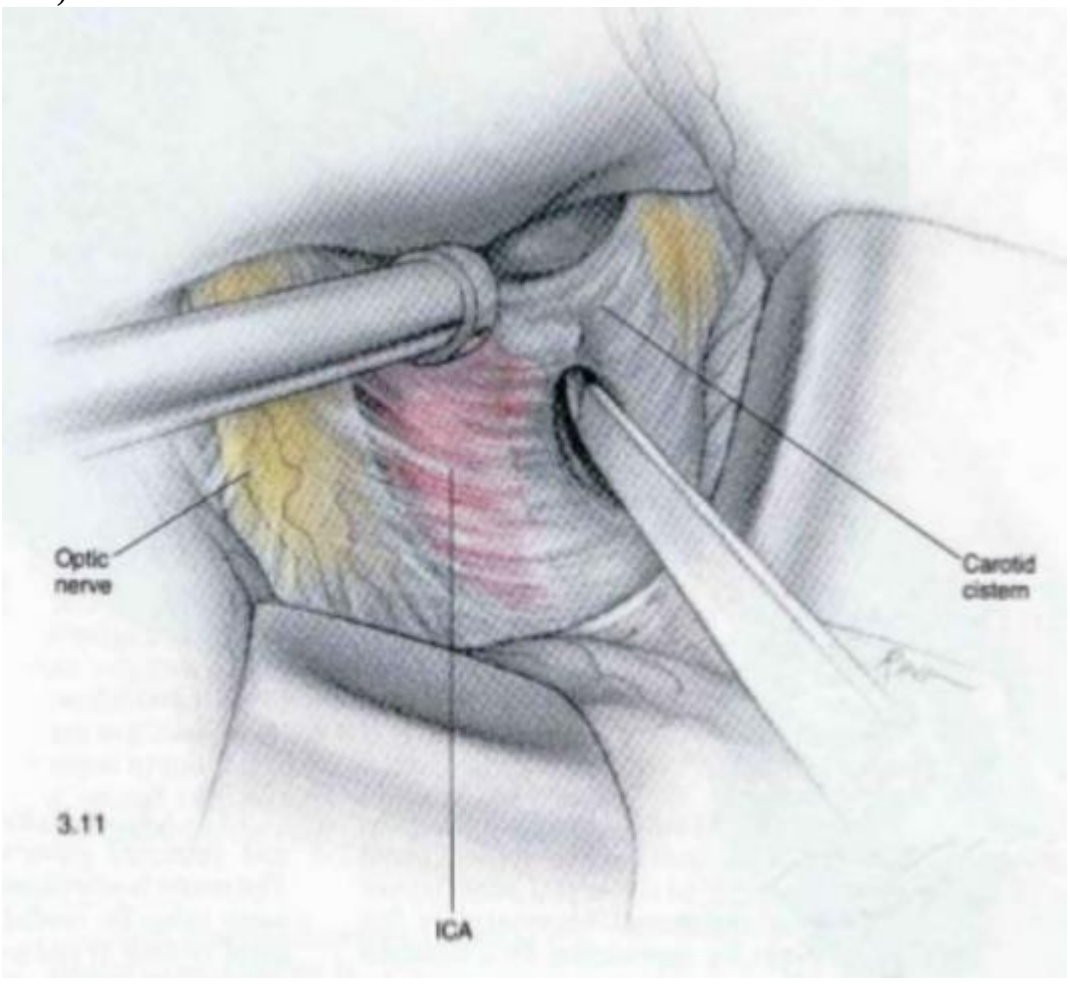
- planum polare may be very adherent
- cutting arachnoid adhesions along middle fossa floor frees inferior temporal lobe:



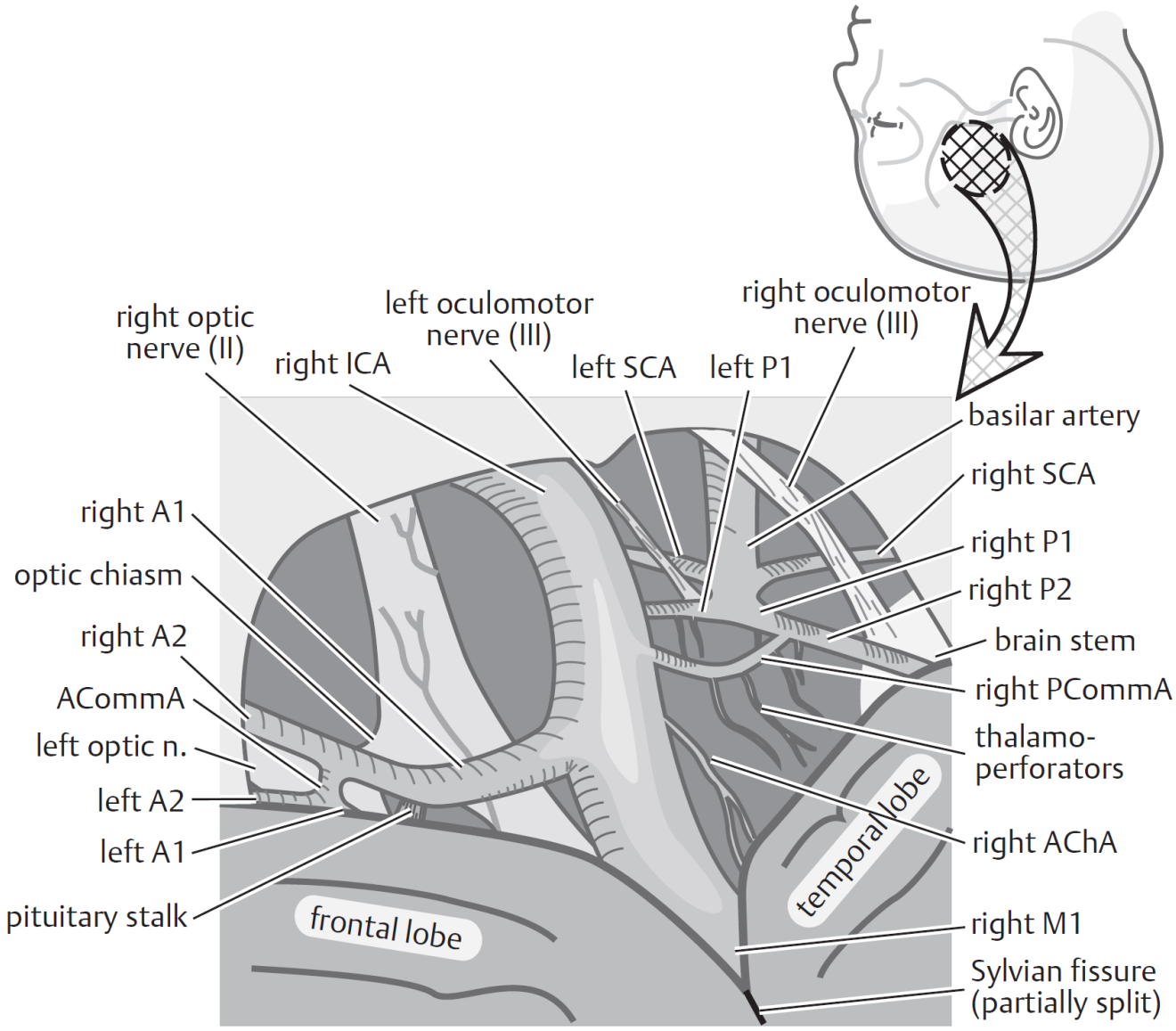
- <u>try to preserve all crossing vessels</u> (Dr. JRC) / it is fine to take if needed (Dr. Rivet).
  - N.B. it should never be necessary to take a vessel crossing fissure because these can always be separated to one lobe or another through careful dissection (i.e. one vessel trunk gives branches either to temporal or frontal lobe but not to both).
  - N.B. there are no arteries that cross Sylvian fissure - if correct plane is maintained, no arteries need to be sacrificed!
- temporal operculum is mobilized away from the insula.
- divide **temporopolar vein** – untethers anterior temporal lobe:

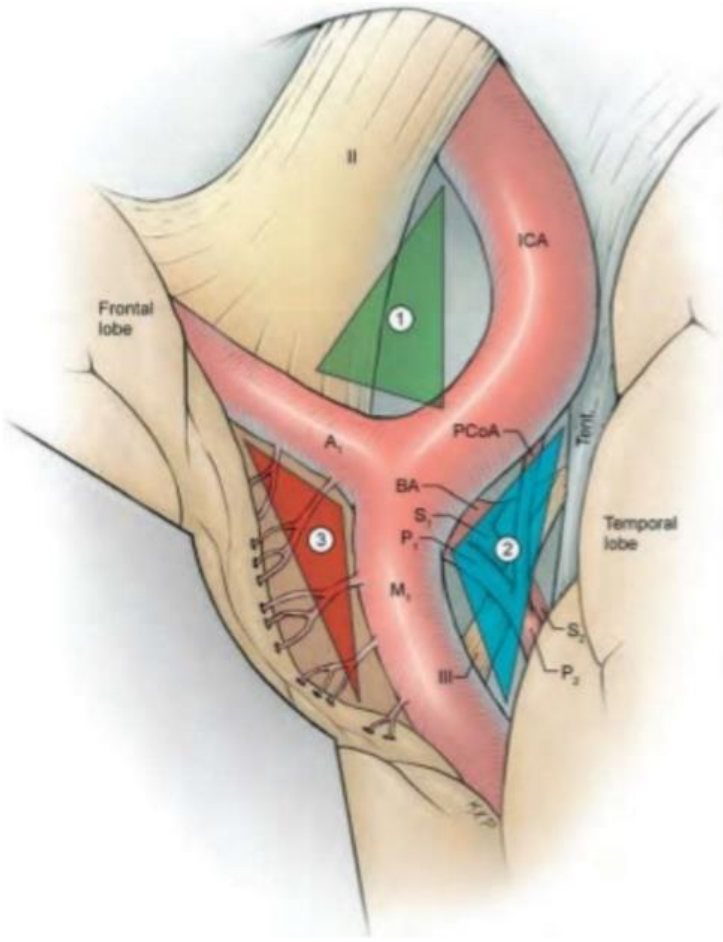


- gentle **dynamic retraction** of the frontotemporal opercula is used as long as possible.
- brain retractors are placed on the frontal and temporal lobes - visualization of basal cisterns.
- M1 may be used as a landmark to reach the opticocarotid cistern.
- open **opticocarotid cistern** (thick arachnoid band tethers frontal and temporal lobes to each other here):



Right sided Sylvian fissure dissection – theoretical exposure of the circle of Willis – diagram is semi-schematic as in reality dissection would be directed either anteriorly (e.g. to expose AComA) or posteriorly (e.g. for basilar tip aneurysms) but not both:





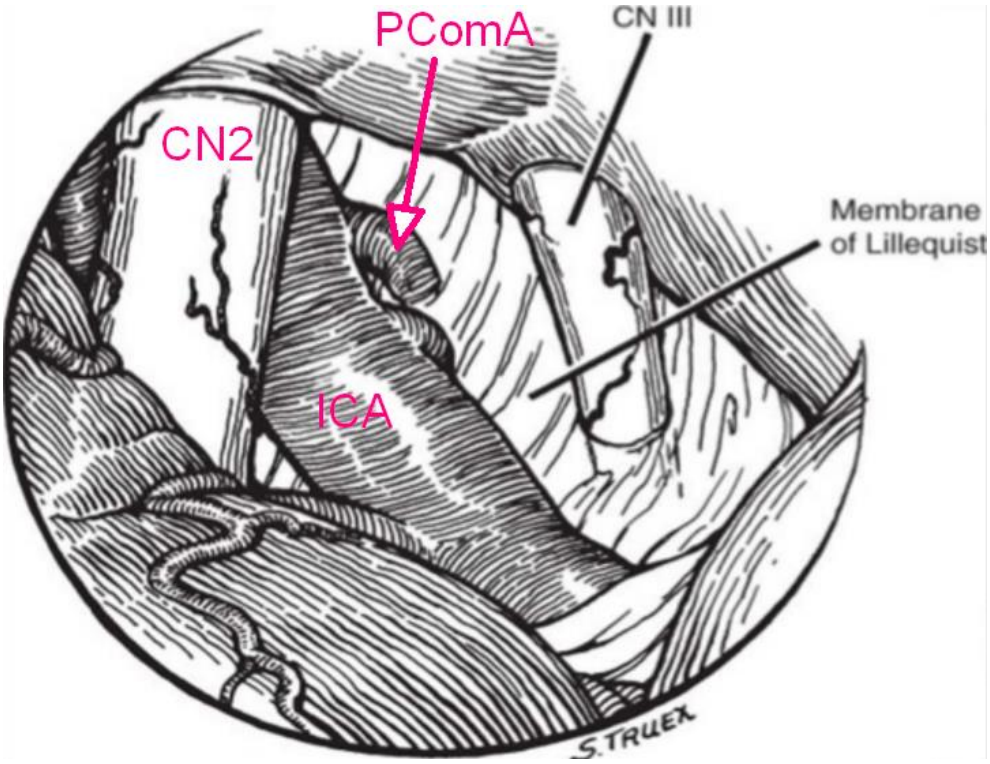
Anatomic triangles providing access to the basilar bifurcation:

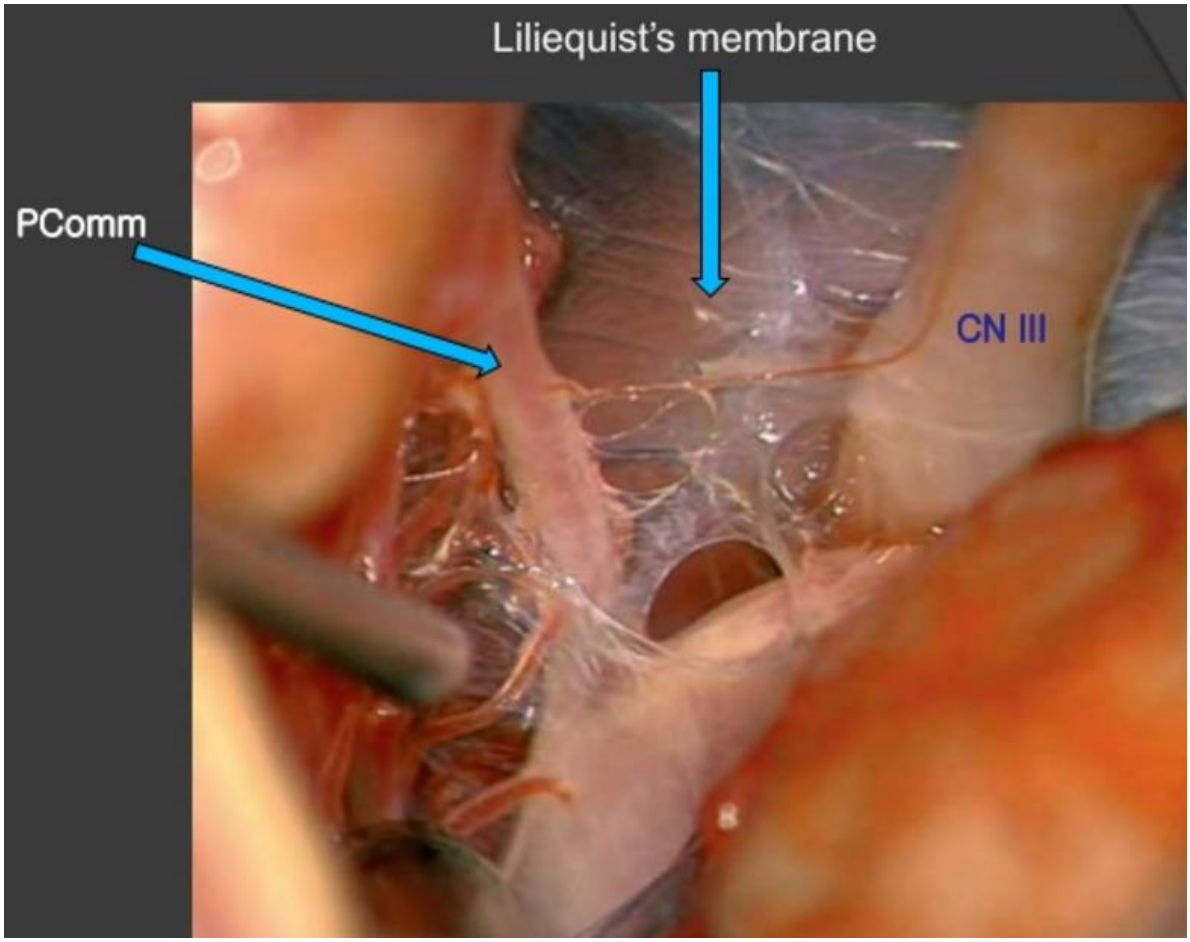
1 optic-carotid triangle

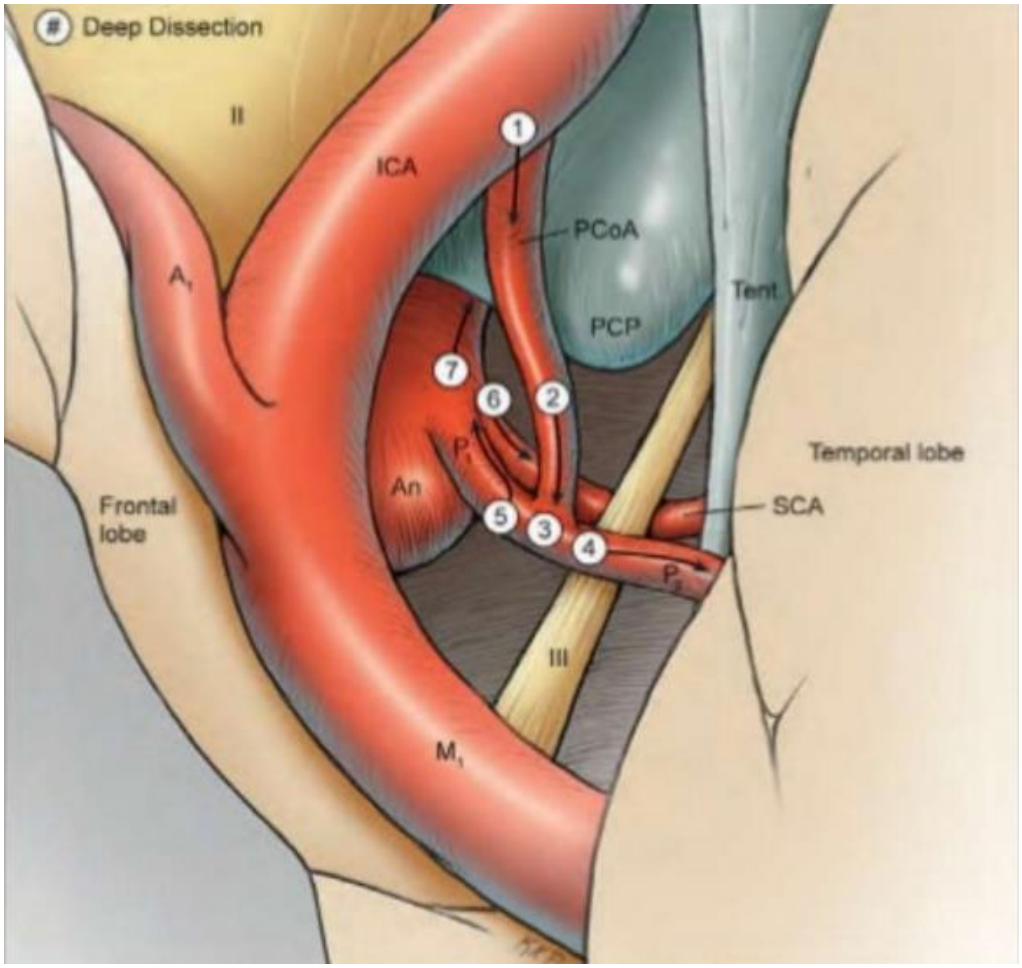
2 carotid-oculomotor triangle

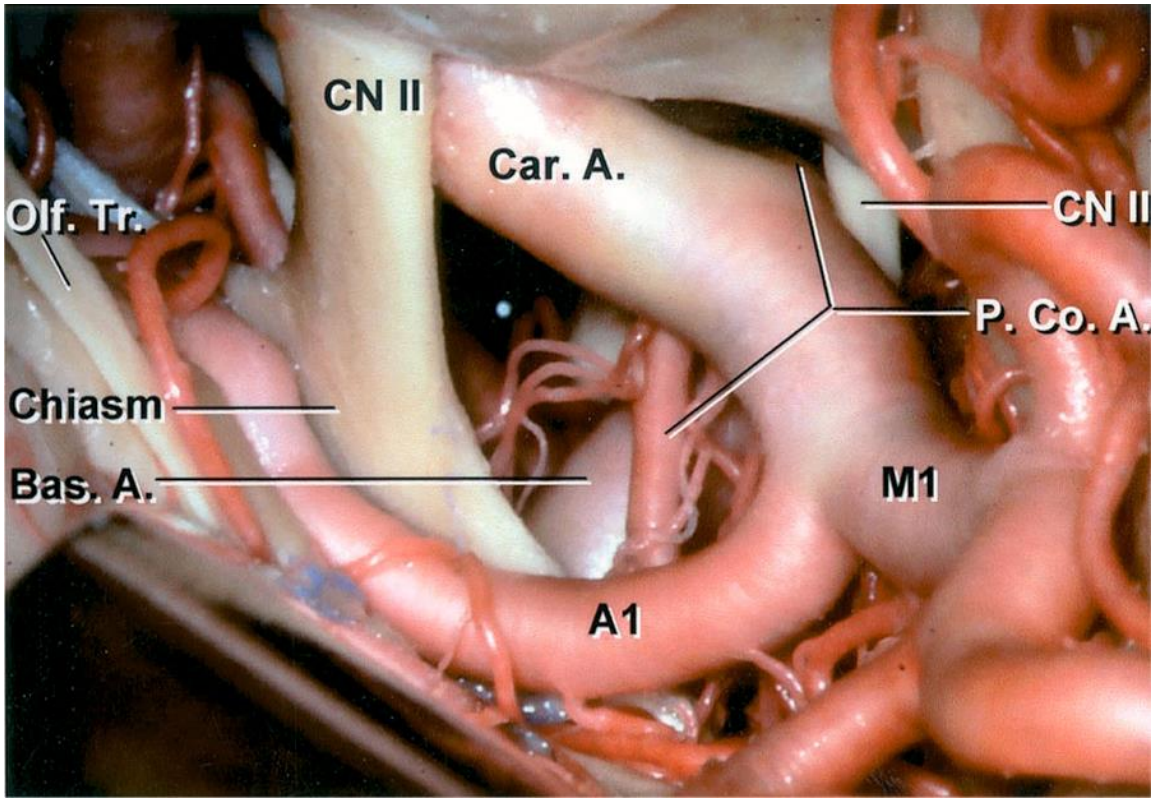
3 supracarotid triangle

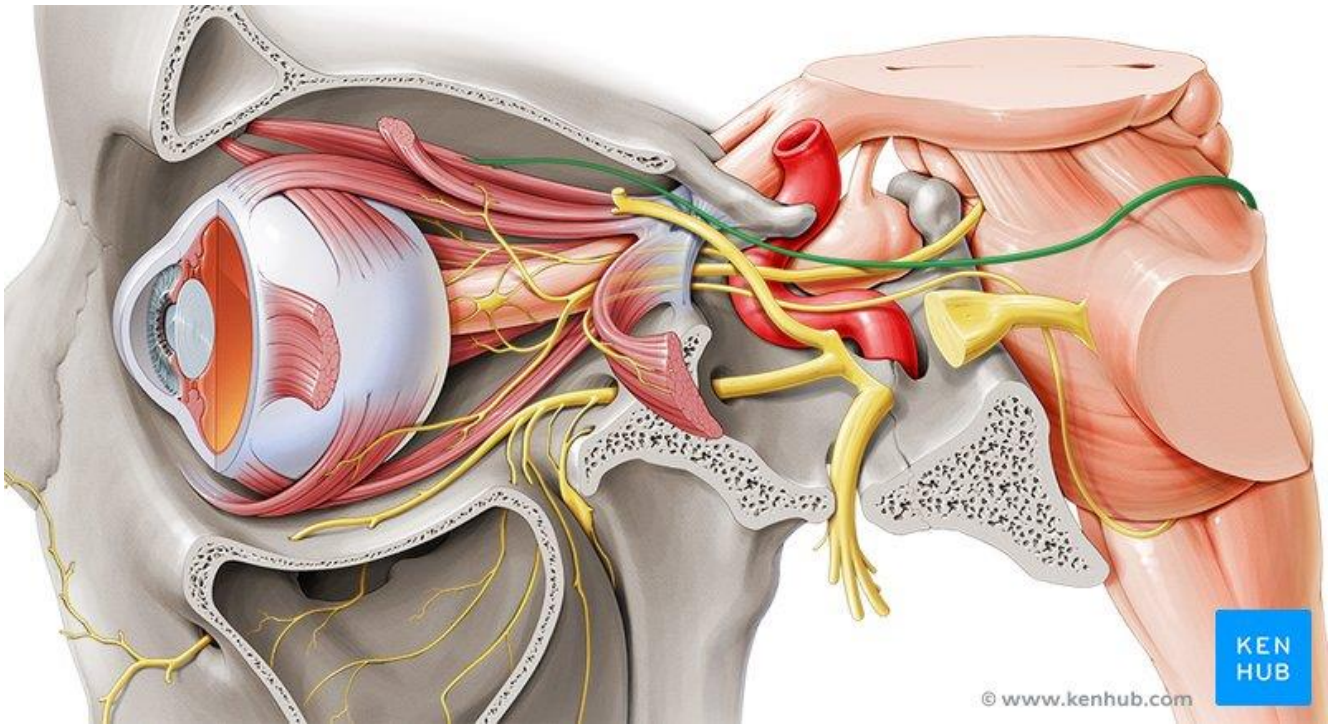
The carotid-oculomotor triangle is the one used most commonly for basilar bifurcation aneurysms.




Liliequist's membrane
PComm
CN III


Deep Dissection
II
ICA
PCoA
Tent
PCP
Temporal lobe
SCA
Frontal lobe
An
III
1
2
3
4
5
6
7





OPEN ALTERNATIVES TO SURGICAL CLIPPING

(if aneurysm cannot be clipped):

a) **wrapping** (for *dissecting aneurysms*, *fusiform aneurysms*) with plastic ***resins*** or ***muscle*** or ***fascia*** or ***gauze*** – only modest benefits.
b) **trapping** (for *fusiform aneurysms*) - distal and proximal arterial interruption with direct surgery; ***balloon occlusion test*** assesses which cases necessitate bypass.
c) **Hunterian (proximal) ligation of parent artery** - for *unclippable broad-based or giant aneurysms* (e.g. ICA in cavernous sinus, dissecting VA aneurysm).
   - check DSA for filling from contralateral side.
   - ***balloon occlusion test***; if fails → *bypass*

– ***adjustable clamps*** (gradual occlusion) allow time for collateral circulation to increase.

d) **aneurysm excision** (for *giant MCA aneurysms*) → end-to-end or branch reconstruction of parent artery.

COILING

GUGLIELMI detachable platinum coil (GDC) - can be used to treat most aneurysms

- induces ***electrothrombosis***
- coils can protrude into parent vessel (H: balloon-assisted and stent-assisted coil placements)
- *recurrence secondary to coil compaction*

Procedure

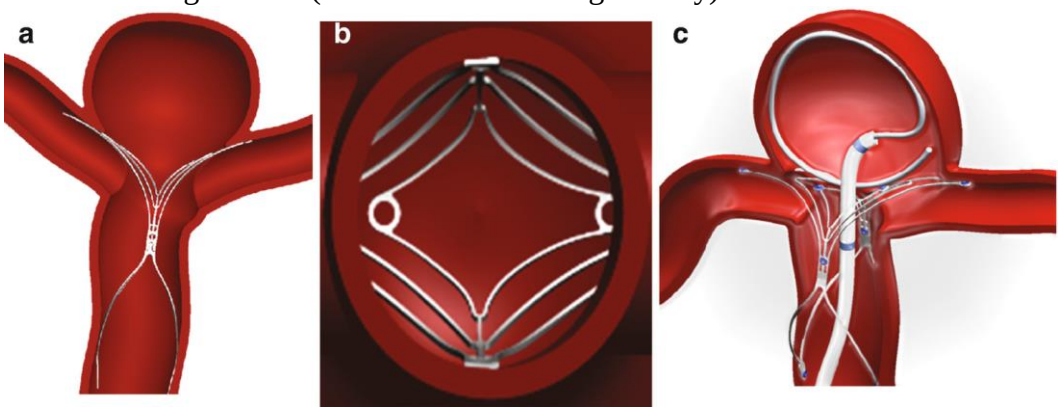
- complete patient immobilization (and thus general anesthesia) is mandatory.
- HEPARIN bolus IV (100 U/kg) to achieve activated coagulation time > 250 seconds (in ruptured aneurysms, patients may not receive heparin until first coil is deployed).
- some experts start ASPIRIN immediately (was important with older generation more thrombogenic coils; now – only if coils are too close to normal vessel lumen)

Stent-assisted coiling - generally, not used for *ruptured* aneurysms – because unable to use dual-antiplatelets after procedure! (not due to rebleeding risk* but if patient will need / has EVD** or shunting)

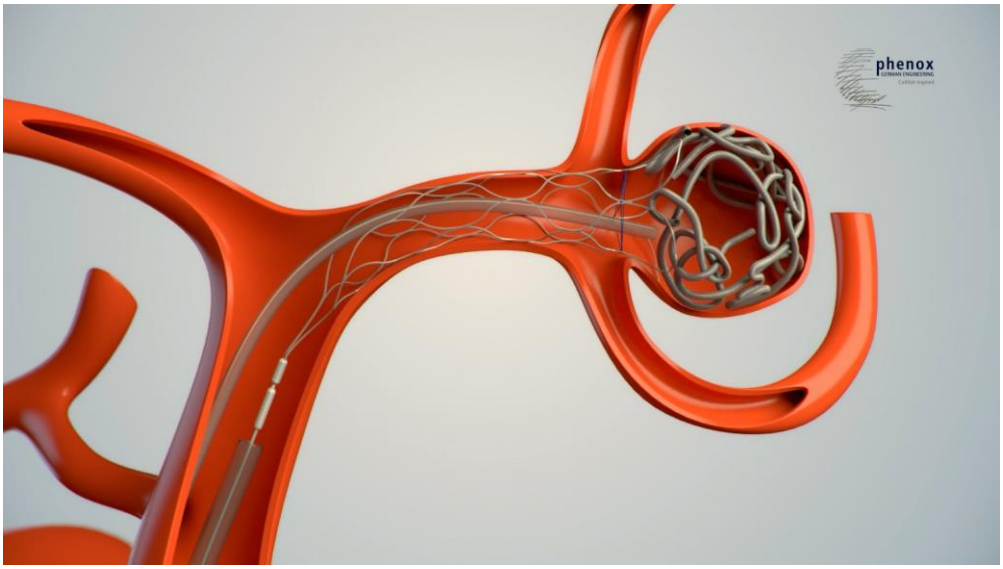
*aneurysmal bleeding is arterial and unaffected by platelet function
**alternative, if has already EVD, - trim EVD at skull level and come back 6 months later (no longer on dual-antiplatelet) to retrieve EVD

Bifurcation stents if aneurysm is at vessel bifurcation (otherwise, would need two stents in Y or X configuration)

PulseRider - self-expanding (retrievable) frame that opens to conform to vessel walls; comes in T and Y configurations (to match bifurcation geometry)



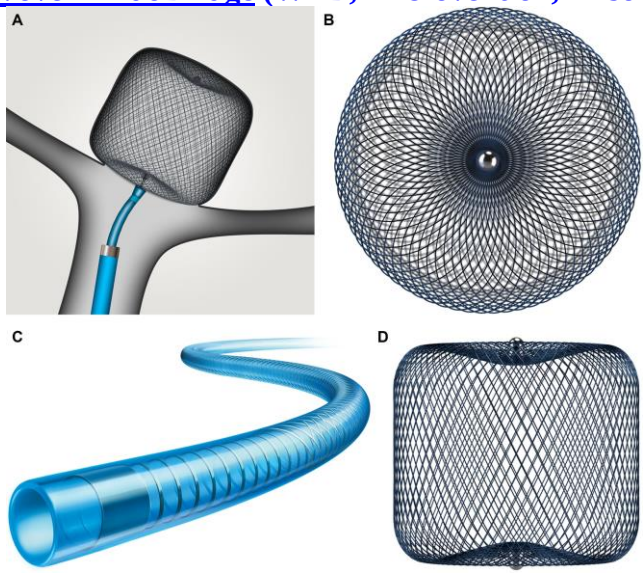
pCONus device (Phenox) - stent acts as a device for aneurysm neck protection for wide-neck aneurysm coiling, e.g. at MCA bifurcation (traditionally, treated surgically):



Endosaccular flow-disruptors - act from within the lesions.

- advantageous if aneurysm is at vessel bifurcation (otherwise, would need two stents in Y or X configuration), wide-neck.
- dual antiplatelet therapy is not required! – can use for ruptured aneurysms!

Woven EndoBridge (WEB; Microvention, Aliso Viejo, California)



Flow-diverting endoluminal stents (e.g. Pipeline) – operate from within parent artery (i.e. do not catheterize aneurysm sac – reduced risk of rupture): flow diverter + scaffold for endothelial cells

Indications:

a) wide-necked aneurysms (neck > 4 mm) with unfavorable dome/neck ratios (< 1.5)
b) fusiform / dissecting aneurysms

- FDA approved for carotid artery (from petrous to superior hypophyseal or even up to ICA terminus - **Pipeline™ Flex**)
- can also use in basilar artery (preserves perforators) but not at the basilar tip.
  Safe in posterior circulation!
- patients should start ***dual antiplatelet therapy*** before implantation → PLAVIX for 6 months + ASPIRIN indefinitely.

SPECIAL SITUATIONS

See p. Vas25 >>

Below only summaries:

CAROTID STENOSIS ASSOCIATED WITH ICA ANEURYSM

- stenosis may have "protective effect" on aneurysm; H: elective aneurysm clipping → endarterectomy.

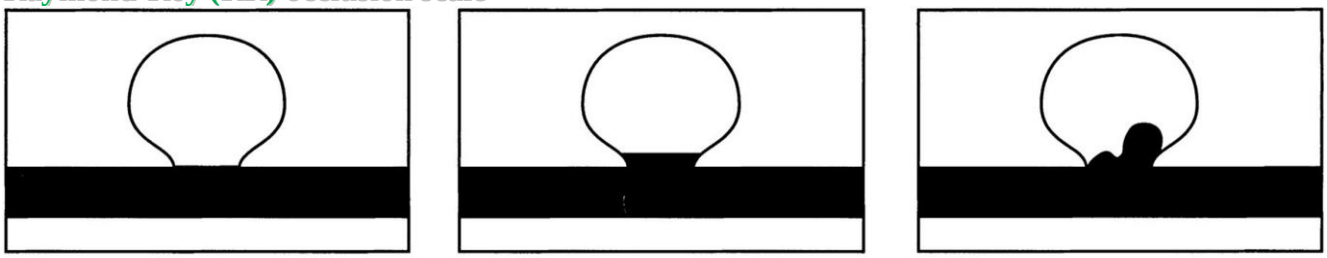
RECANALIZATION (OF PREVIOUSLY TREATED ANEURYSM)

Risk factors for recanalization:

1) large volume (> 600 mm^3) aneurysms
2) low volume (< 20%) packing.

- mechanisms: *coil compaction*, unorganized *unstable thrombus* formation, *absence of neointima* formation at the neck of coiled aneurysms.

Raymond-Roy (RR) occlusion scale



When to treat? If remnant ***keeps enlarging*** at each follow up (threshold to treat is lower for previously ruptured aneurysms).

- re-coiling is safe.

RECANALIZATION IN PREVIOUS STENT-ASSIST COILED ANEURYSM

– attempt coiling again.

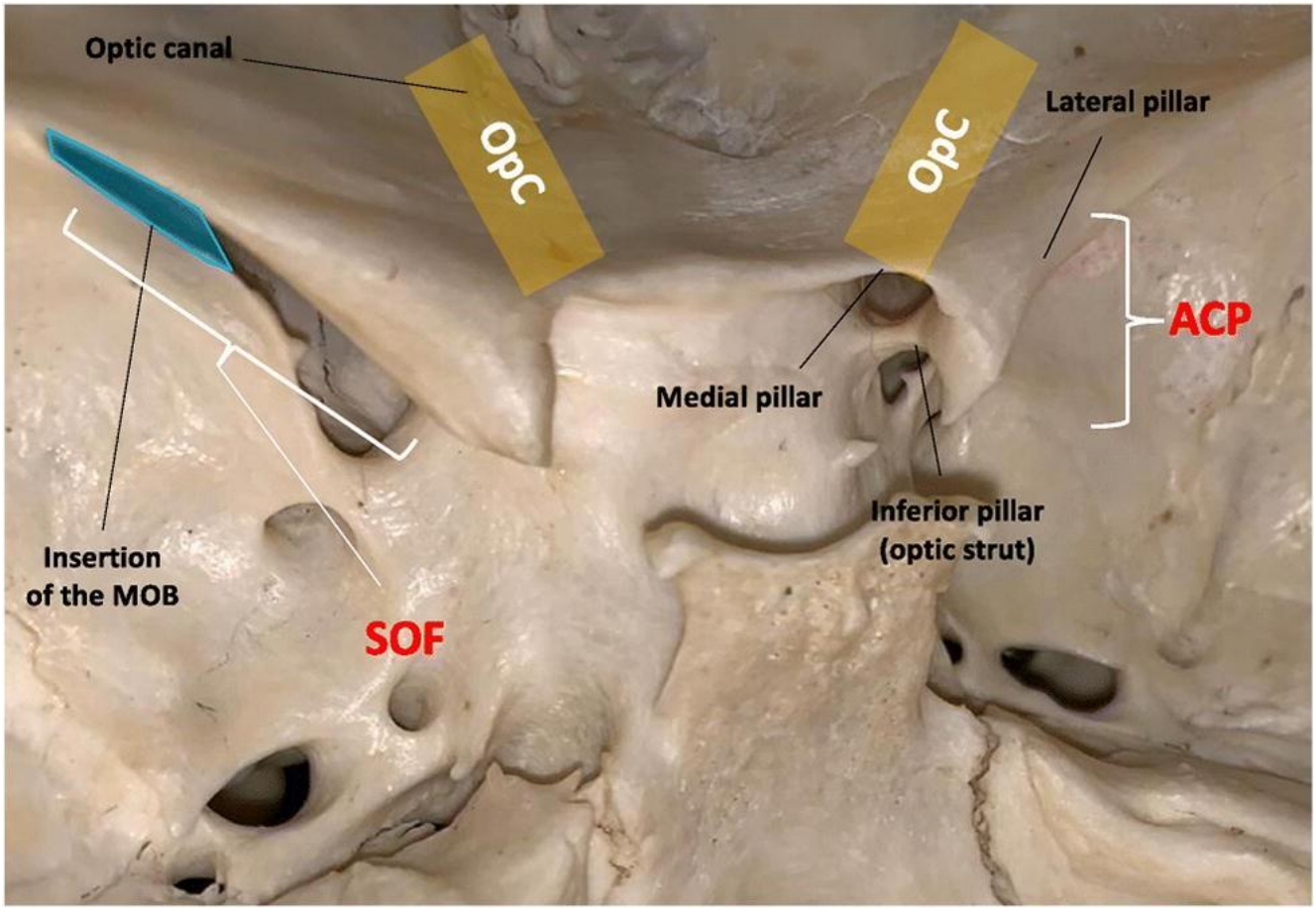
- if needs clipping, may need to open aneurysm sac to remove coils to allow neck clipping.

CAVERNOUS CAROTID ANEURYSM

– if ruptures, bleeding is contained but risk of CC fistula.

Differential from paraclinoid aneurysm (i.e. intradural) – CTA in coronal plane: look at **optic strut** (bony structure running transversely as floor of optic canal):

PARACLINOID aneurysm – above optic strut
CAVERNOUS aneurysm – below optic strut



Treatment indications (e.g. pipeline)

1) **diameter** > 1 cm (risk of erosion through dura → SAH in case of rupture)
2) **symptomatic**:
    a) CC fistula
    b) CN palsies
    c) thrombemboli (from turbulent flow within aneurysm)

See E2 case >>

CERVICAL ICA ANEURYSMS

– treatment not indicated.

MYCOTIC ANEURYSMS

- bacterial etiology (e.g. in subacute bacterial endocarditis)
- tend to form in distal (often unnamed) vessels
- fusiform and very friable – practically treatable by vessel sacrifice (some experts like the term "deconstructing the vessel"); if in eloquent area, may use Amytal+lidocaine injection to check if patient tolerates it
- treatment – **antibiotics** at least for 4-6 weeks → MRA: failure of aneurysm to reduce in size → clipping.

MULTIPLE ANEURYSMS

a) if incidental aneurysm is in field of surgical approach, it can be clipped along with ruptured aneurysm.
b) if incidental aneurysm is on contralateral side, then it can be clipped electively at another time (to prevent bilateral vascular injury at the same time).

ANEURYSM PERFORATION WITH COIL

First sign – sudden BP↑↑↑ (reaction to ICP↑)
Management:

1) notify **anesthesia** - immediately lower BP, hyperventilate, raise EVD
2) 50 mg of PROTAMINE (protamine should always be available during the procedure!)
    a. if antiplatelet agents were used – give platelet transfusion.
3) inflate balloon (if using balloon assistance)
4) if perforation at aneurysm dome - don't stop, do not remove coil, continue rapidly coiling with additional coils.
    a. if perforation at aneurysm neck / parent vessel – balloon, may need to embolize
5) place **EVD**.

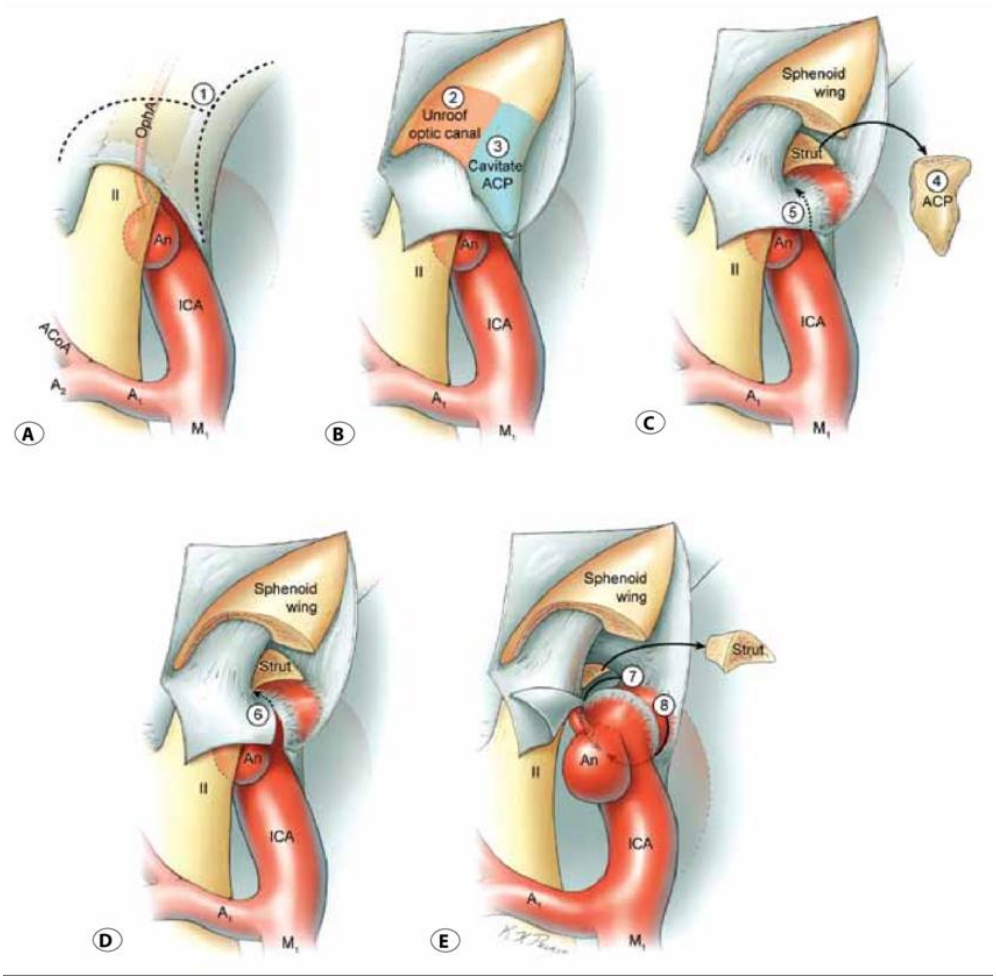
Postop – CT (hematoma needing evacuation?)

VESSEL OCCLUSION DURING DSA

1. HEPARIN (verify ACT)
2. Intra-arterial:
    a) TPA – make sure aneurysm well coiled before administering thrombolytics!
    b) REOPRO
    c) **thrombectomy** (if large vessel)

OPHTHALMIC ARTERY ANEURYSMS

- 45% present as SAH.
- 45% present as visual field defect (optic nerve compression).
- most can be coiled (even mass effect goes away after coiling) unless vessel comes off the dome; craniotomy needs **ICA exposure** in the neck for proximal control + **anterior clinoidectomy**. see p. Op300 >>



SUPERIOR HYPOPHYSEAL ARTERY

- may *mimic* pituitary adenoma.

ACOMA

- visual field deficits, prefrontal lobotomy syndrome (abulia / akinetic mutism), hypothalamic dysfunction, leg paraparesis (!)

[fragile hypothalamic perforators - coiling is very preferred!!! ***stent-assisted*** if wide neck]

Extended pterional craniotomy (head rotation 60 degrees, anterior edge of craniotomy extends lateral to midpupillary line) - arachnoid bands tethering the frontal lobe to the chiasm are placed on stretch and sharply divided; no need to expose proximal carotid: only anterior aspect of Sylvian fissure is split and A1 is identified - proximal control on ipsilateral A1, then contralateral A1 (first temp clip is on dominant A1); usually need **fenestrated** clip.
Side of Approach (in order of importance):

1) ***contralateral to the direction of aneurysm dome***!
2) ipsilateral to ***isolated dominant A1***
3) ipsilateral to ***ICH*** - to avoid any injury to the only intact contralateral gyrus rectus
4) right-sided pterional approaches are technically easier

See E1 case >>
See E3 case >>

PCOMA

- CN3 palsy (if acute – due to rapid aneurysm growth or sentinel bleed – both need urgent treatment!!!).
  30% of acute CN3 palsies are due to PComA aneurysms

Extended [lesser sphenoid wing, flatted orbit] pterional craniotomy with head rotation 30 degrees, sphenoid wing must be aggressively drilled, may need ***anterior clinoidectomy***

- **subfrontal corridor** (mobilization of the frontal lobe - dissection of arachnoidal attachments over chiasm and floor of frontal fossa; do not manipulate temporal lobe i.e. ***aneurysm must be exposed solely via frontal lobe retraction***) → proximal Sylvian fissure split → opening optico-carotid cistern → drain CSF + proximal control over supraclinoid ICA (proximal ICA at opticocarotid triangle) + prep neck.
  N.B. if dissection exposes proximal neck of aneurysm without adequate space for temporary clip deployment, additional exposure of proximal ICA is mandatory. Ensure proximal control at all costs!!!

MCA

Pterional craniotomy (head rotation 45 degrees): minimal subfrontal exposure; sylvian dissection proximally at the level of the carotid cisterns (major vector of retraction on temporal tip) → drain CSF, gain proximal control on M1 (just distal to lenticulostriate vessels) →"inside-to-outside" dissection from M1 (along anterior-inferior aspect) toward bifurcation.

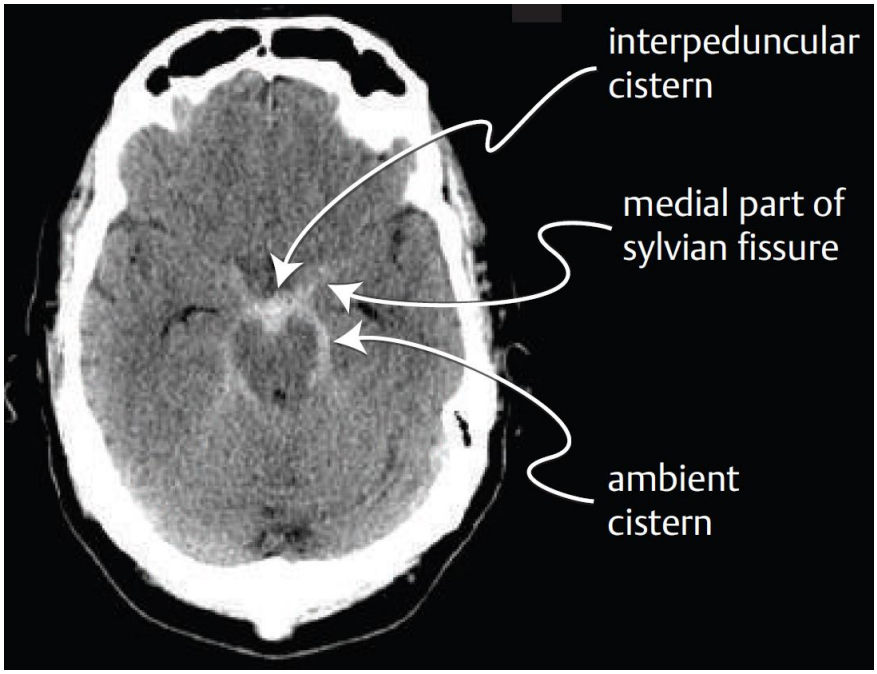
- preserve STA for potential bypass
- avoid any significant retraction on temporal lobe!
- ICH (80% in temporal lobe) – need much larger craniotomy, *early proximal control* is especially important - start subfrontal dissection proximally near carotid cisterns (early proximal control at supraclinoid ICA) → **transcortical approach**: corticotomy over anterior superior or middle temporal gyrus → evacuate safe part of clot, find M1 and apply temporary clip on M1.

See E4 case >>

BENIGN PERIMESENCEPHALIC (PRETRUNCAL) SAH

- bleeding immediately anterior to brainstem and adjacent areas (interpeduncular fossa, ambient cisterns) - rupture of *small pontine or perimesencephalic veins* (i.e. not arterial source - **prognosis is excellent – no rebleeds reported**! - no need for repeat angiography after negative first DSA)

  - imaging - blood should be contained inferior to Liliequist's membrane (LM) (i.e., perimesencephalic and/or prepontine cisterns); significant amounts of blood penetrating LM to chiasmatic, Sylvian, or interhemispheric cisterns should be viewed with suspicion!
  - one angio is needed to rule out basilar tip aneurysm!
    Board answer: one DSA → two CTAs (other experts skip CTAs)



COMPLICATIONS

REBLEEDING

(risk is highest within 1st day)

Rebleeding risk of untreated aneurysms:
1st day – 4%*
daily first 2 weeks – 1.5%** (2 wks cumulative – 20%, 6 mos cumulative – 50%)
after 6 months – bleeding risk returns to baseline (1-3%/yr)

*"blow out" hemorrhages – due to unstable thrombus
**lysis of clot sitting over rupture site

- clinically - new headache, sudden neurologic worsening, sudden ICP↑, bright blood in EVD.
- mortality ≈ 51-85%
- prevention/treatment: N.B. bed rest does not prevent rebleeding!
  1) control HTN → lower GB
  2) do not drain EVD below 10 → raise EVD and flush it
  3) TRANEXAMIC ACID 1 g q6h IV (e.g. if patient needs to be transferred before securing aneurysm; otherwise contraindicated - delays clot lysis → vasospasm, HCP)

VASOSPASM

(occurs at days 3 ÷ 21 or 4-14)
- delayed narrowing of large capacitance arteries at base of brain (radiographic vasospasm)

- angiographic incidence 30-70% (on bleed day 7); of these, < 50% become symptomatic.

Putative responsible agent – ENDOTHELIN

Clinically
Radiographic vasospasm can lead (20-50%) to *delayed ischemic neurologic deficit (DIND)* (clinical s. symptomatic vasospasm): **HA, AMS, new-onset focal neurologic deficits**.

Lethargy (with or without focal neurological deficit) is vasospasm, until proven otherwise! → emergency CT to rule out other pathology (vasospasm may be clinically indistinguishable from rebleeding or HCP!)

Monitoring
1) routine serial **neuro exams**
2) heralding *hyponatremia*
3) routine daily **TCD** (normal velocities are < 100) until risk of vasospasm decreases

Mean **MCA** velocity	MCA:ICA (Lindegaard) ratio**	Interpretation
< 120	< 3	Normal
120-200*	3-6	120-160 Mild spasm 160-200 Moderate spasm
> 200	> 6	Severe vasospasm

*velocities in this range are specific for vasospasm but are only 60% sensitive
**differentiates vasospasm from hyperemia

   – findings are often more helpful when baseline studies before vasospasm are available (e.g. increases of > 50 cm/sec/**d** suggest vasospasm)
4) alternative to TCD – **vEEG** (look for assymetry)
5) baseline and follow-up **perfusion CT, CTA**

Diagnosis

CTA with **pCT** – first test to do if SAH patient develops any neuro deficit*; some neurosurgeons do it routinely (e.g. at SAH bleed day 10 or if TCDs↑↑↑)
*at the same time put NPO order as patient potentially may go to OR
Angiography – can confirm diagnosis and afford treatment (e.g. intra-arterial VERAPAMIL)

Treatment
Symptomatic vasospasm (or TCD close to 200)
1. Drop **EVD** to 0
2. Start **HHH therapy, s. hemodynamic augmentation** (prophylactic HHH is not recommended)
   N.B. modern approach – mainly induced HTN (shift away from aggressive hypervolemia, active hemodilution)
   1) **hypertension** SBP up to 180-220 (160 if aneurysm is unsecured) - NOREPINEPHRINE; if tachycardia > 150 add PHENYLEPHRINE; if SBP still low (esp. if SVR > 800 and PCWP 12-14 mmHg), add DOBUTAMINE
   2) **hypervolemia** (target CVP 10-12 mmHg) – NS* > 200 mL/hr (plus, boluses of ALBUMIN); if UO > 200 mL/hr Greenberg recommends DDAVP; some add FLUDROCORTISONE. *change to ½NS if [Na] > 150
   3) **hemodilution** (optimal hematocrit is 30-33%); for Hct < 25% give packed RBCs; MANNITOL 20% at 0.25 gm/kg/hr may improve rheological properties.
   - risks of HHH therapy
     1) rebleeding (if aneurysm is still not treated!)
     2) pulmonary edema
     3) myocardial ischemia
   - reversal of deficits is seen within 1 h in 81%.

DANTROLENE - one of the few drugs shown to both prevent and reverse vasospasm.

Refractory vasospasm - if still symptomatic after 1-6 hour of HHH → **angiography**:
1) **angioplasty** – method of choice for *proximal vessels*! (contraindicated if stroke already happened)
2) **IA** VERAPAMIL **or** NICARDIPINE (takes 30 min for full effect; effect lasts 24 hrs**)** - for vasospasm in *distal vasculature*, where balloons may not access

ACUTE OBSTRUCTIVE HYDROCEPHALUS

(most present within first 24 hours)

- treatment: EVD with drainage at 10 (when aneurysm is secured – may drop to 0).

DELAYED COMMUNICATING HYDROCEPHALUS

(develops ≥ 10 days after SAH)

- prophylaxis: prolonged **EVD** + LD (clear blood from CSF) + **tPA** (esp. if ventricular casting is present)
- treatment: ***ventriculoperitoneal shunt*** (may temporize with repeat LPs* - it is temporary condition)

*esp. if patient was leaking CSF from EVD incision – want to make sure CSF is not infected

SAH-INDUCED HYPONATREMIA

- majority due to ***cerebral salt wasting***; the rest – due to ***SIADH***
- may be first sign of vasospasm!
- differential:
  1) urine [Na] (> 20 in CSW)
  2) CVP - SIADH and cerebral salt wasting (CSW) look similar in labs but SIADH causes hypervolemia and CSW causes hypovolemia (CVP < 5)
- treatment:
  SIADH – ***fluid restriction, conivaptan***
  CSW:
  1) fluid repletion with ***normal saline*** or ***slightly hypertonic (1.5-3%) NaCl***
  2) ***fludrocortisone***
  3) ***albumin***
  4) ***salt tabs***.

FOLLOW UP

Indefinitely for life! (aneurysm recurrence, de novo aneurysms)

Use the same modality for each follow-up to facilitate accurate comparison!

N.B. ***MRA with gadolinium*** better than CTA or MRA TOF visualizes previously coiled / clipped aneurysms (lots of metal artifacts on CT, esp. after coiling).
At some point perform ***simultaneous angiography and MRA*** so in the future, aneurysm recurrence can be monitored using MRI.

After coiling - **MRA**

1. 6 months (if it was stent-assisted coil, may discontinue PLAVIX, but will remain on ASPIRIN for life)
2. 18 months (1.5 yrs)
3. 42 months (3.5 yrs).
4. Every 5-10 years

After clipping - **CTA**

1 year
5 years
Every 10 years

PROGNOSIS

1/4 of patients with aSAH die, and roughly half of survivors are left with some persistent neurological deficit.

- overall MORTALITY is 45%
  50% deaths occur within 1 month
  rebleeding - mortality ≈ 51-85%
- MORBIDITY in survivors - 50%

See V1 case >>