# Subarachnoid hemorrhage

## Characteristics, pathophysiology & complications

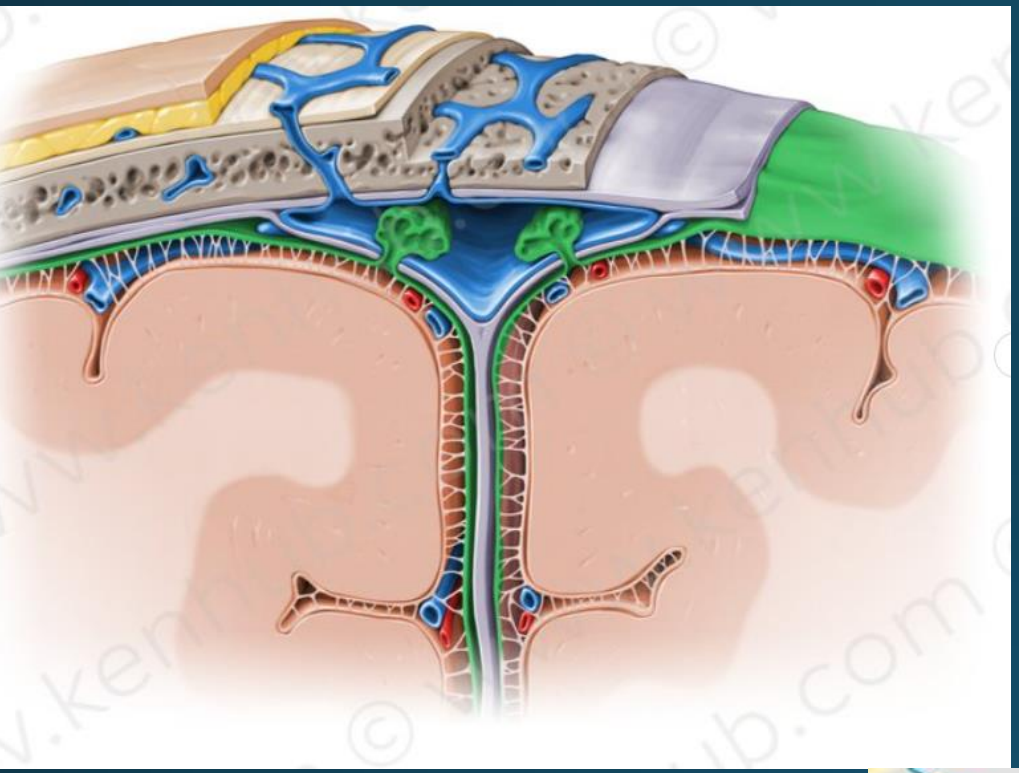
*Szymon Hoppe*

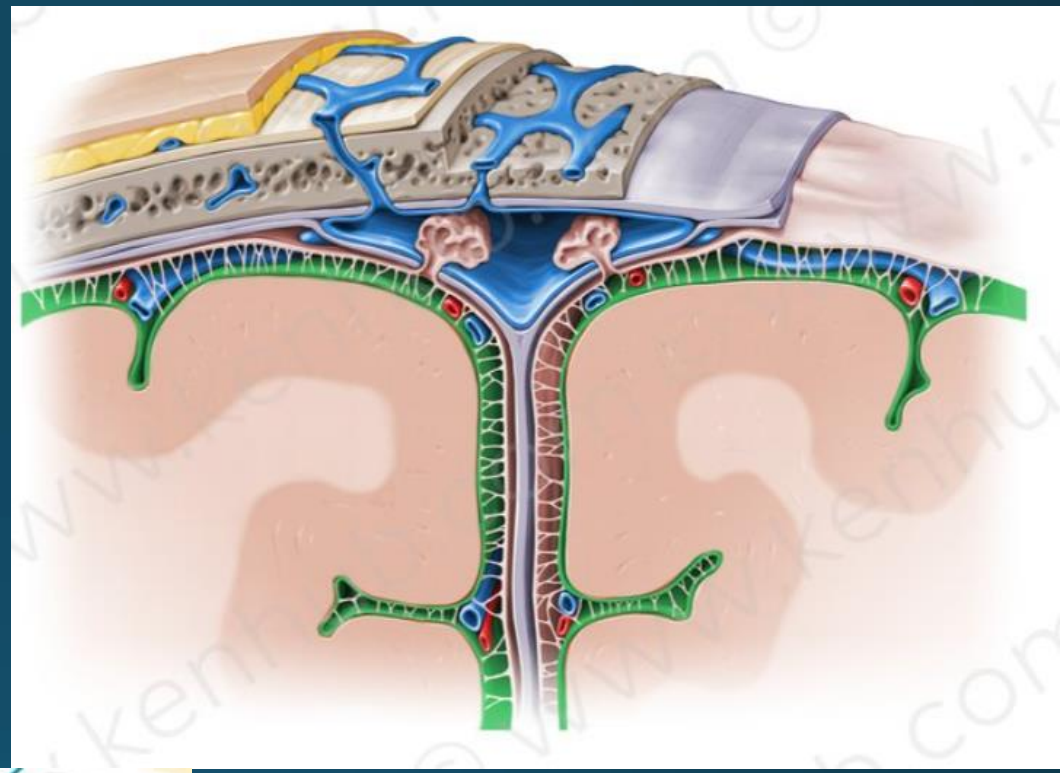
# Presentation plan

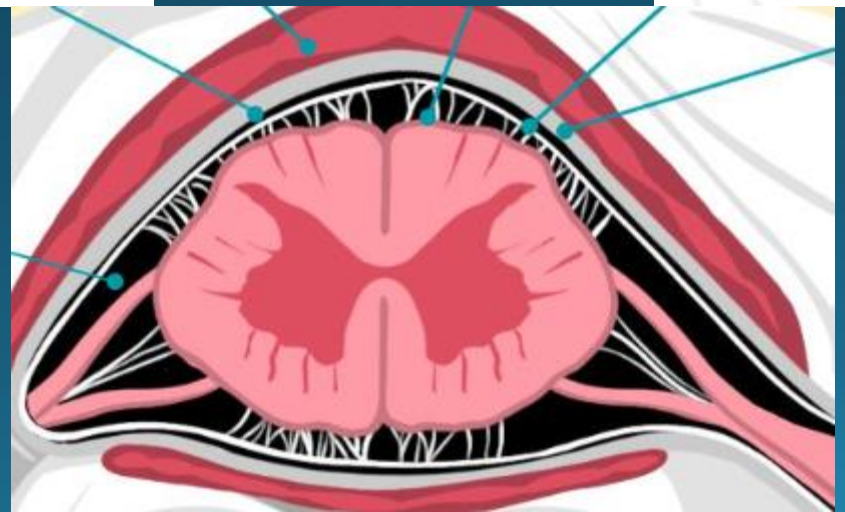
- Anatomy
- Epidemiology, etiology
- Presenting Sx
- Dx & scales

- **Pathopysio & novel research**
- **Complications**
  - **Surgical**
  - **Medical**
  - **Delayed Cerebral ischemia**

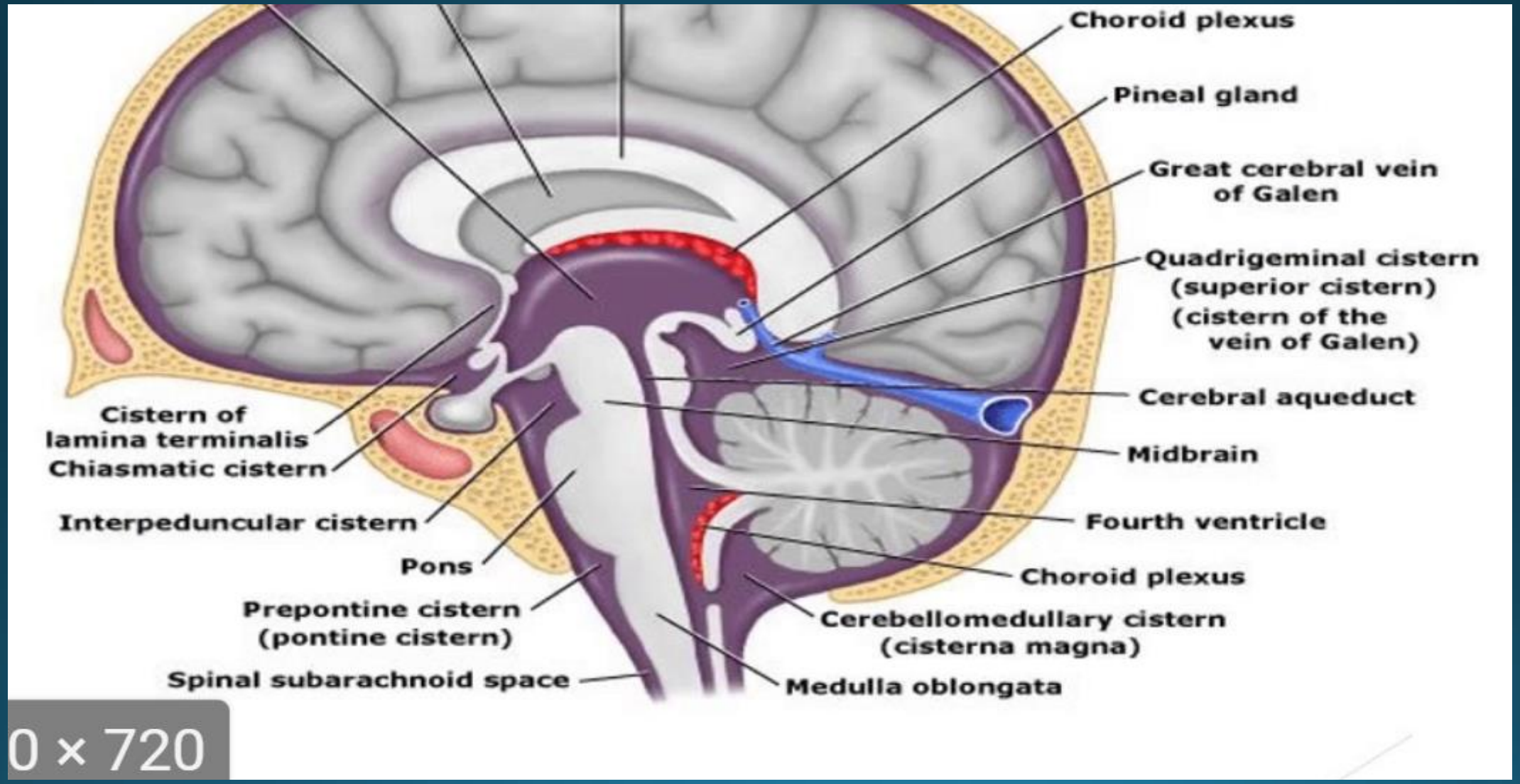
# Subarachnoid space (SAS)



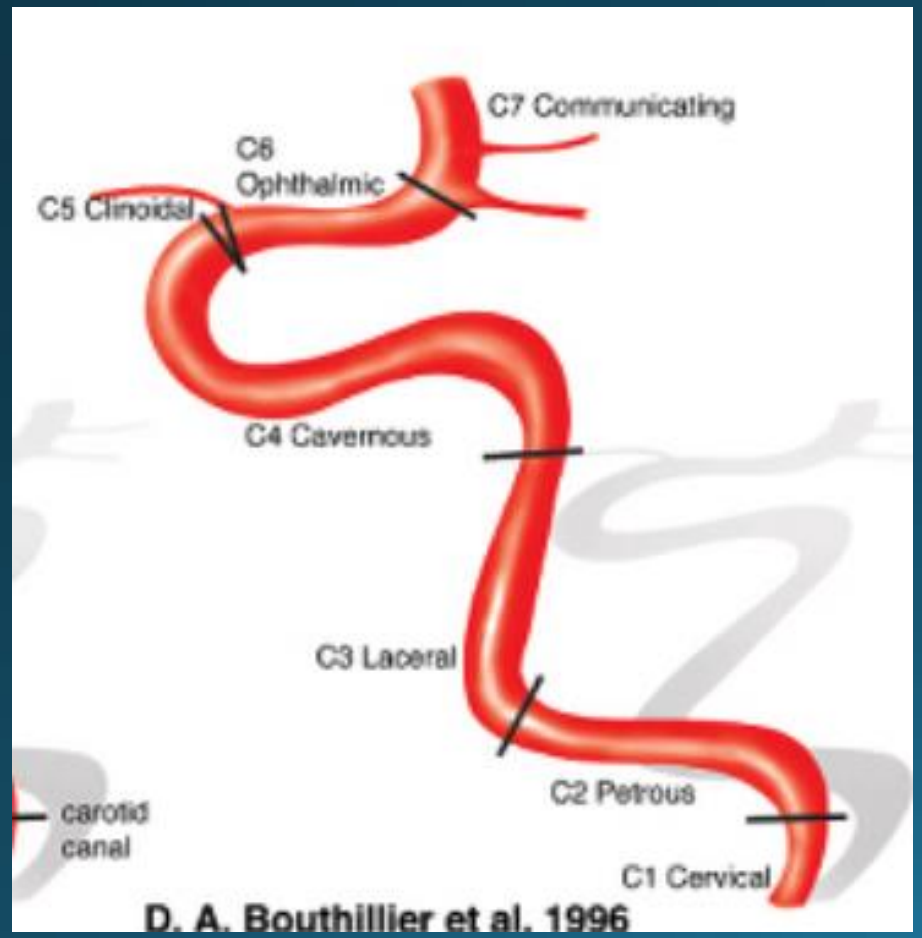


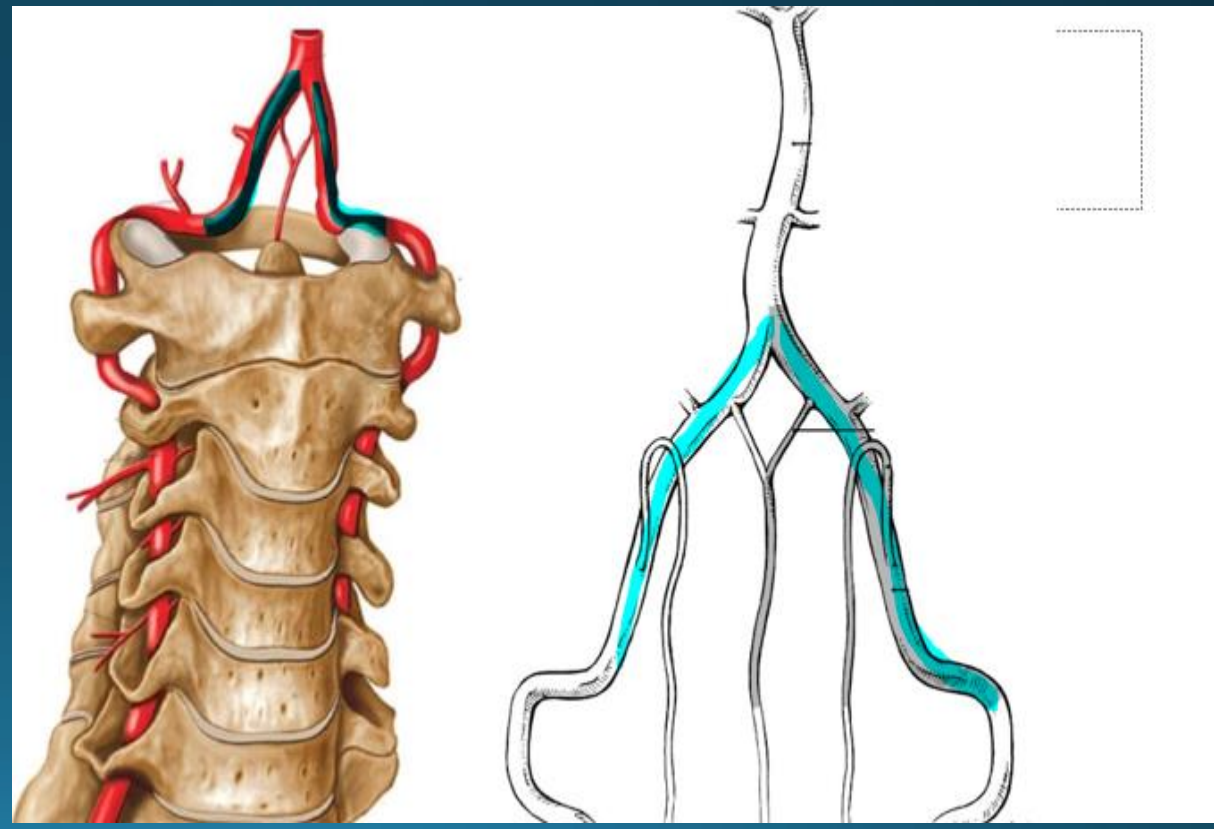


# SAS

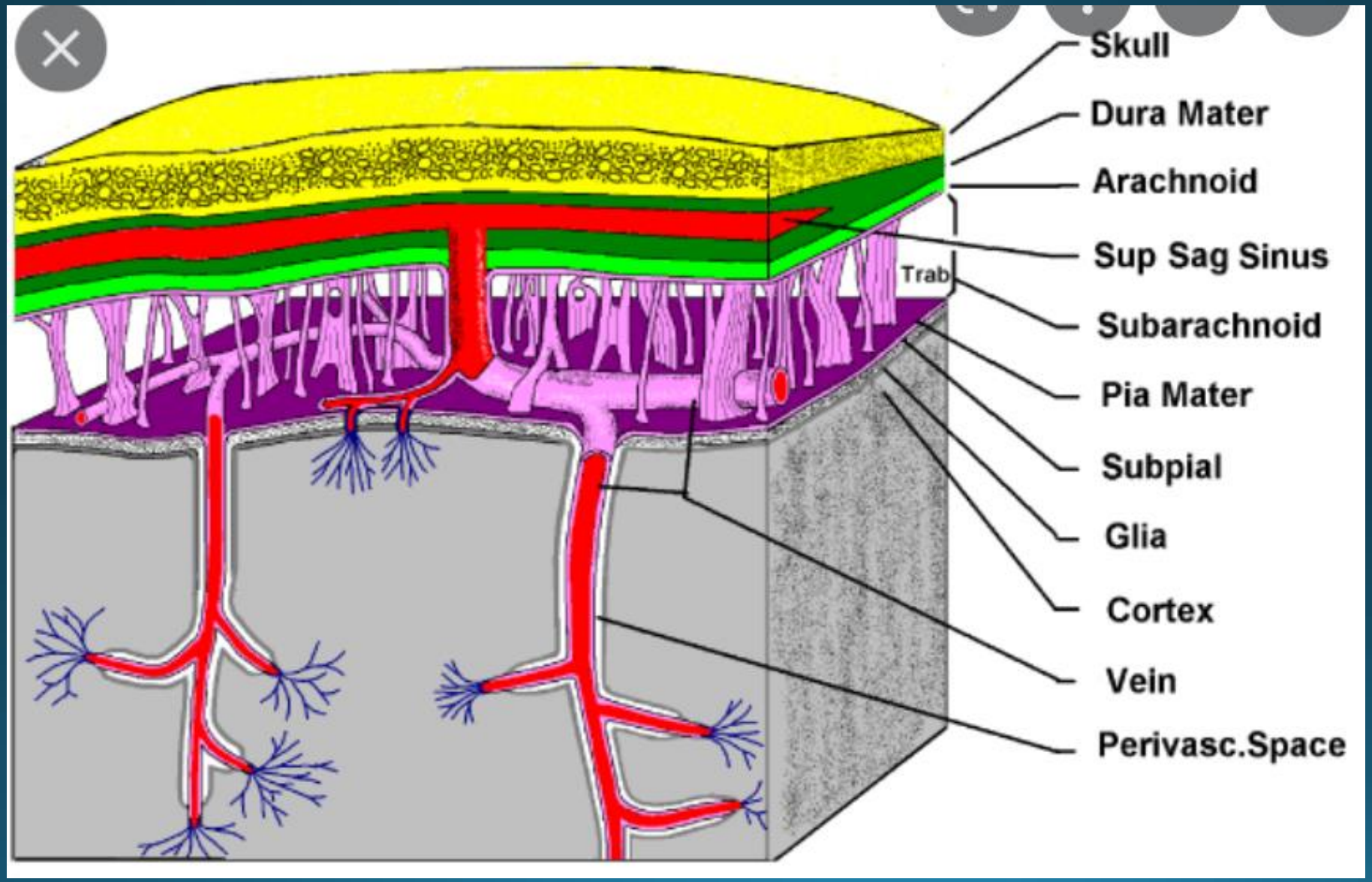

Choroid plexus
Pineal gland
Great cerebral vein of Galen
Quadrigeminal cistern (superior cistern) (cistern of the vein of Galen)
Cerebral aqueduct
Midbrain
Fourth ventricle
Choroid plexus
Cerebellomedullary cistern (cisterna magna)
Medulla oblongata
Cistern of lamina terminalis
Chiasmatic cistern
Interpeduncular cistern
Pons
Prepontine cistern (pontine cistern)
Spinal subarachnoid space
0 × 720

# When arteries enter SAS?



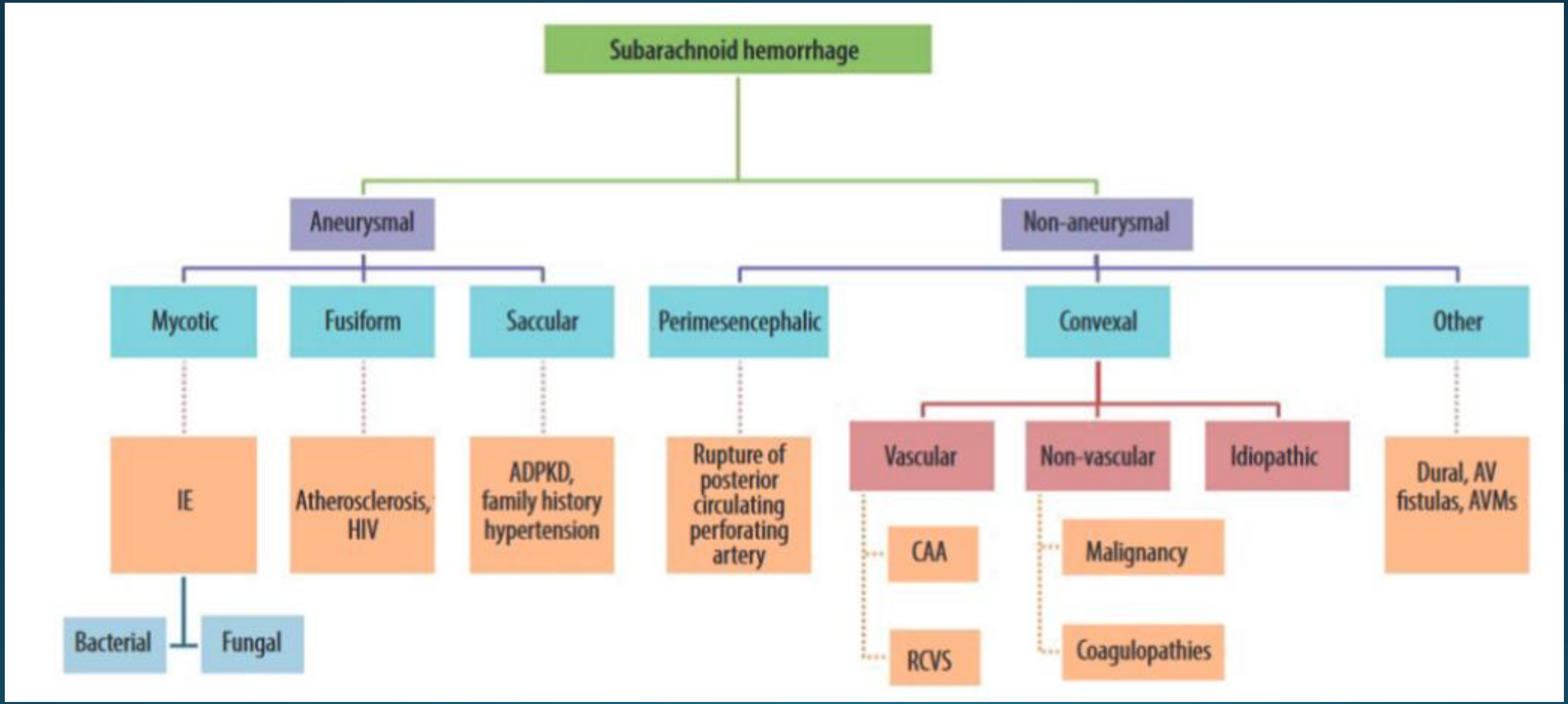



# When arteries *leave* SAS?



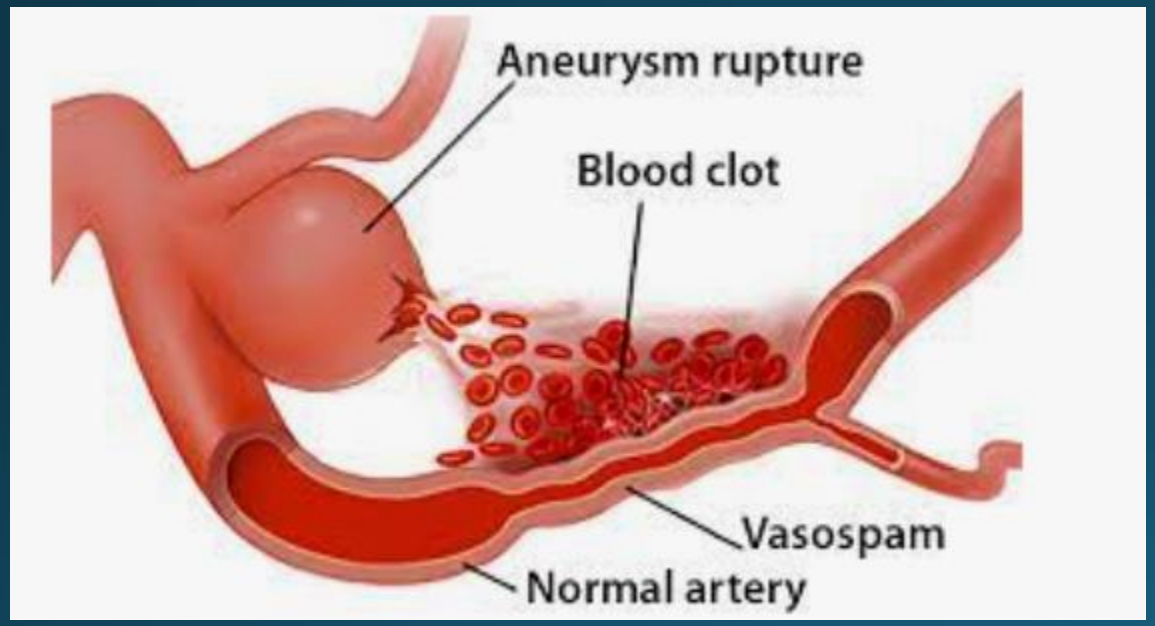
# SAH causes

- Most common cause is head trauma ~90% of SAH
  - 40-60% of head injuries -> SAH (+)
- nontraumatic is ***only*** 10%
  - 80% aneurysmal
  - 5% arteriovenous malformations
- RF: HTN, smoking, high alcohol intake, family history – 2,5x



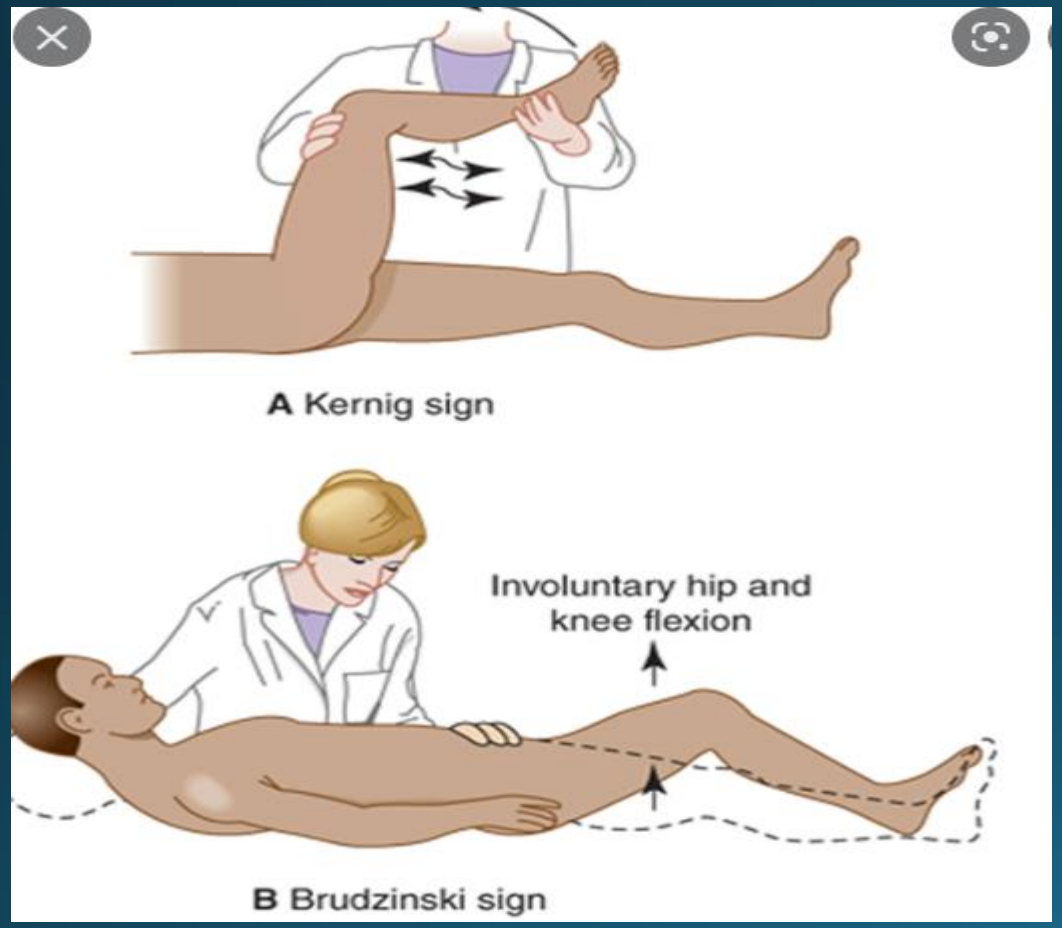

*Khan F., Sharma N., Ud Din M., Shirke S., Abbas S. Am J Case Rep 2021; 22:e931376 :: DOI:*

# Presentation

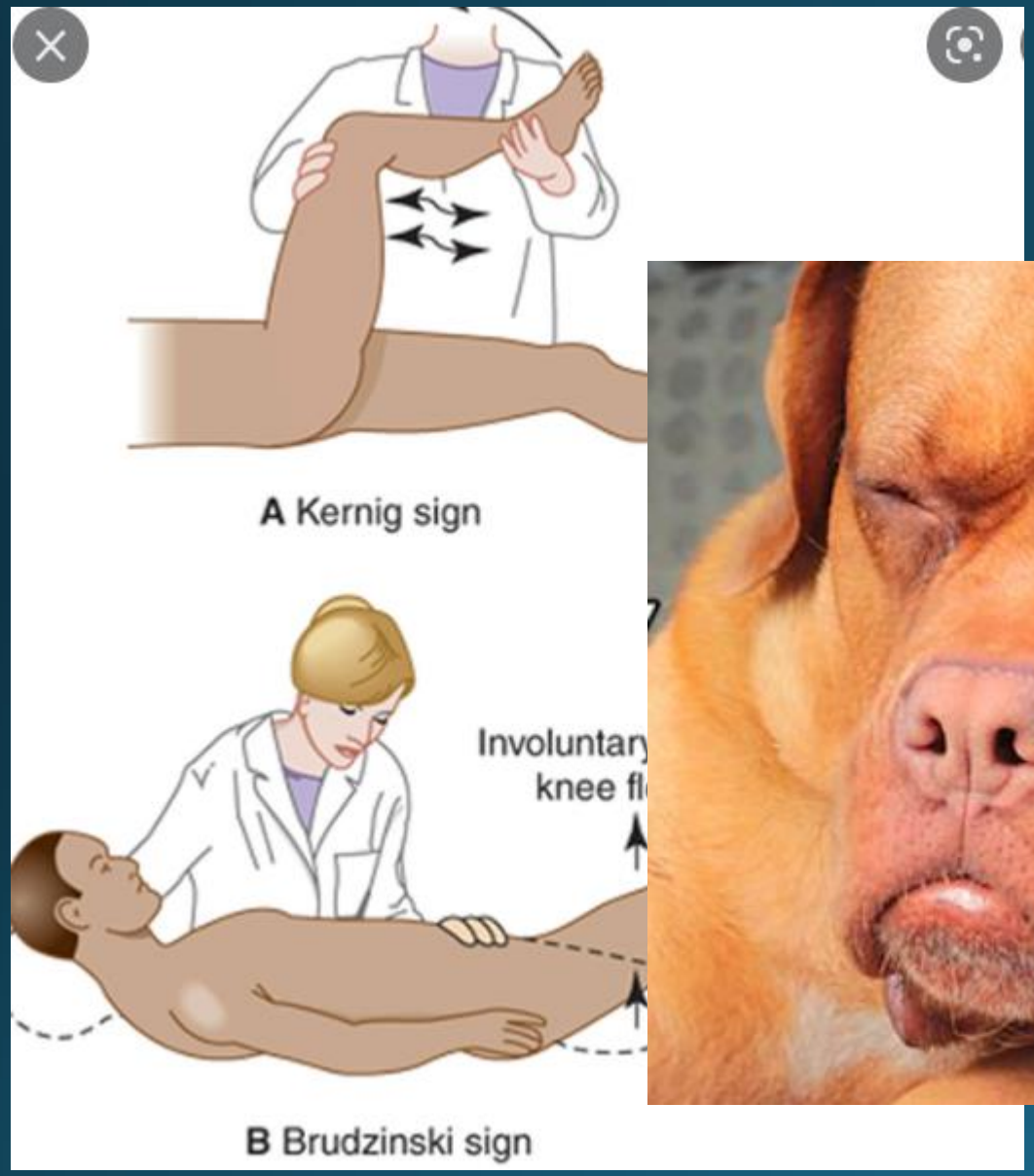


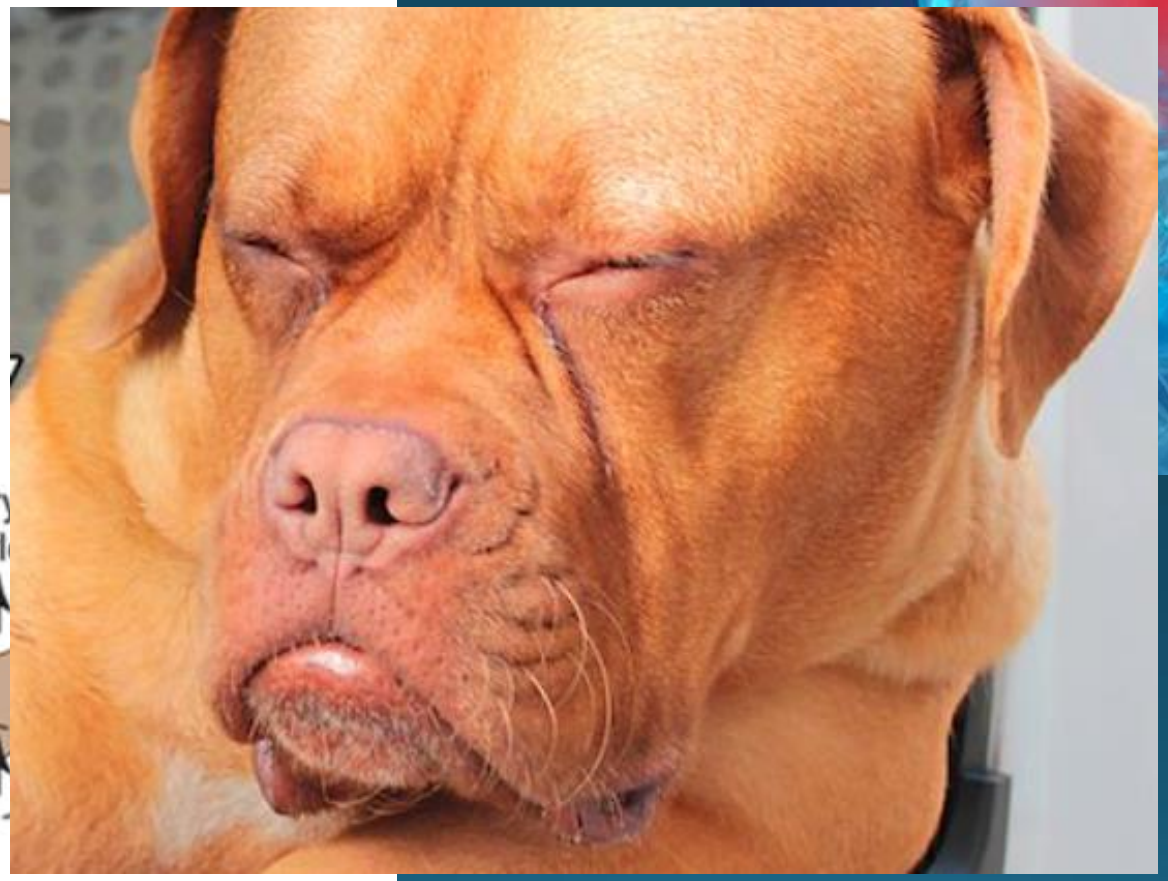

*Sekhon S, Sharma R, Cascella M. Thunderclap Headache. [Updated 2022 Apr 30]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK560629/*

# Meningeal signs




*Philippe Lepage, Bernard Dan,Chapter 115 - Infantile and childhood bacterial meningitis,Editor(s): Olivier Dulac, Maryse Lassonde, Harvey B. Sarnat,Handbook of Clinical Neurology,Elsevier, Volume 112,2013,Pages 1115-1125, ISSN 0072-9752,ISBN 9780444529107*



- *Aydin N, Kotan D, Keles S, Ondas O, Aydin MD, Baykal O, Gundogdu B. An experimental study of the neurophysical mechanisms of photophobia induced by subarachnoid hemorrhage. Neurosci Lett. 2016 Sep 6;630:93-100. doi: 10.1016/j.neulet.2016.07.013. Epub 2016 Jul 18.*

# Other symptoms

- Focal deficits - ischemia/compression
- Altered Mental Status
- Seizures
- *Prodromal sign?*

# Sentinel / Warning leak

- 30-50%

- Days-weeks
- HA + diplopia
- leak

→ thrombus

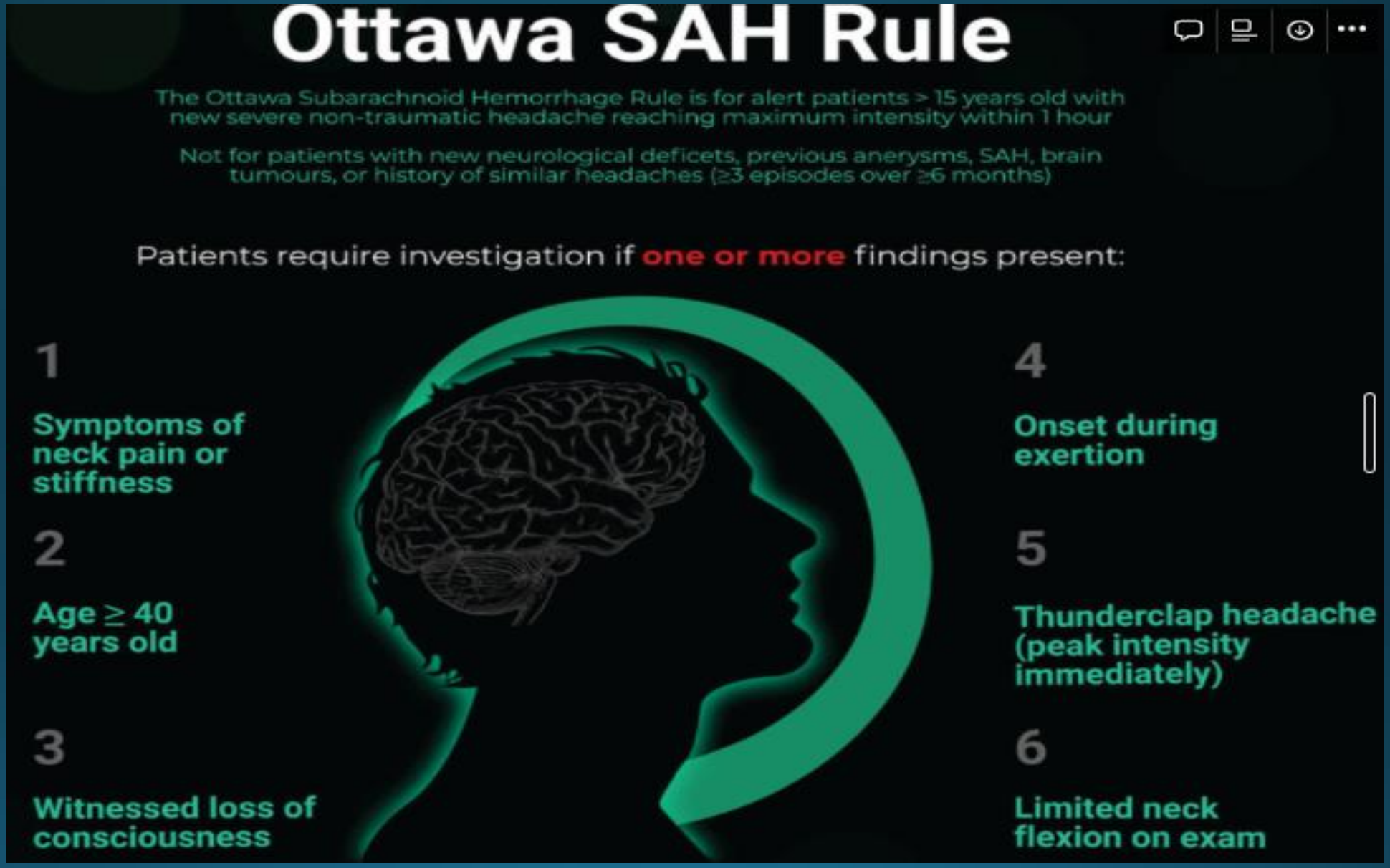
→ fibrynolysis

→ hemorrhage

# Diagosis

Population:
3700

Sensitivity:
100%




*Prospective Implementation of the Ottawa Subarachnoid Hemorrhage Rule and 6-Hour Computed Tomography Rule, Jeffrey J. Perry et al. https://www.ahajournals.org/doi/10.1161/STROKEAHA.119.026969*

# Diagosis

- Lubar puncture
- *if convexal (visible on GRE) - consider CAA/RCVS
- 3D TOF MRA, which, compared to conventional angiography, has an 80% sensitivity for detecting focal vasoconstriction
- Localize w/ CTA/DSA/MRA

*Khan F., Sharma N., Ud Din M., Shirke S., Abbas S. Am J Case Rep 2021; 22:e931376 :: DOI:*

# Scales

WFNS – patient status

| Grade | GCS score | Focal Deficit |
|---|---|---|
| 0 | | |
| 1 | 15 | – |
| 2 | 13–14 | – |
| 3 | 13–14 | + |
| 4 | 7–12 | +/– |
| 5 | 3–6 | +/– |

# Scales

## Hunt & Hess

- Mortality
  - Indicates hydrocephalus (HC)
  - -> External ventricular drainage > grade 3

| Grade* | Clinical presentation | Mortality |
|---|---|---|
| 1 | Alert and oriented, mild headache, minimal if any neck rigidity | 30% |
| 2 | Alert and oriented, moderate-to-severe headache, nuchal rigidity present, cranial palsy but no other focal neurological deficits | 40% |
| 3 | Confusion or lethargy, mild focal neurological deficit present | 50% |
| 4 | Stuporous, severe focal neurological deficit | 80% |
| 5 | Comatose with posturing on neurological examination | 90% |

* Note that one grade may be added for significant systemic disease such as atherosclerosis, chronic obstructive pulmonary disease, diabetes, hypertension, cardiomyopathy, or vasospasm.

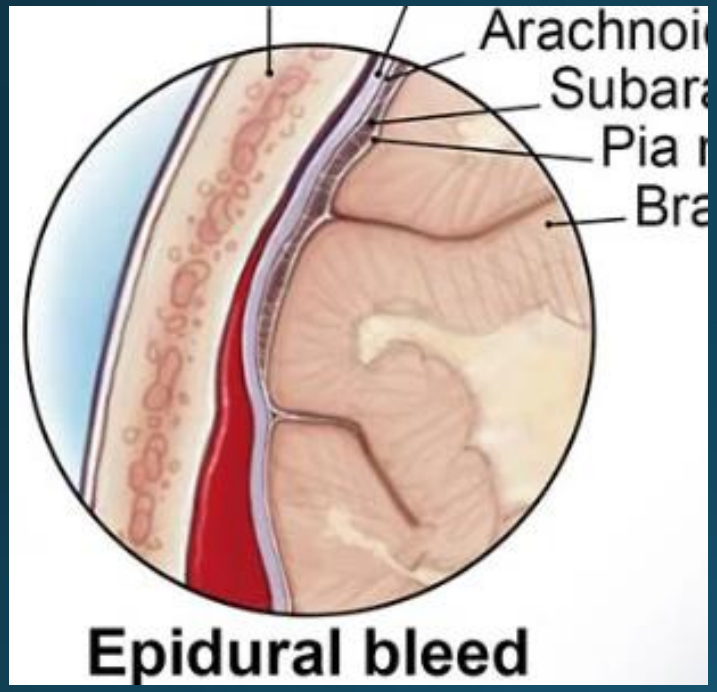
# Scales

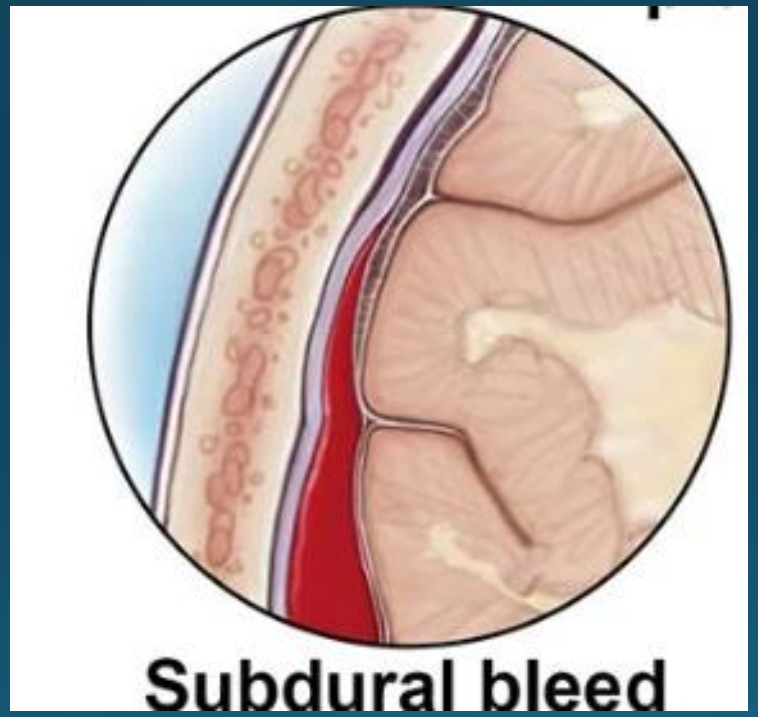
Modified Fisher

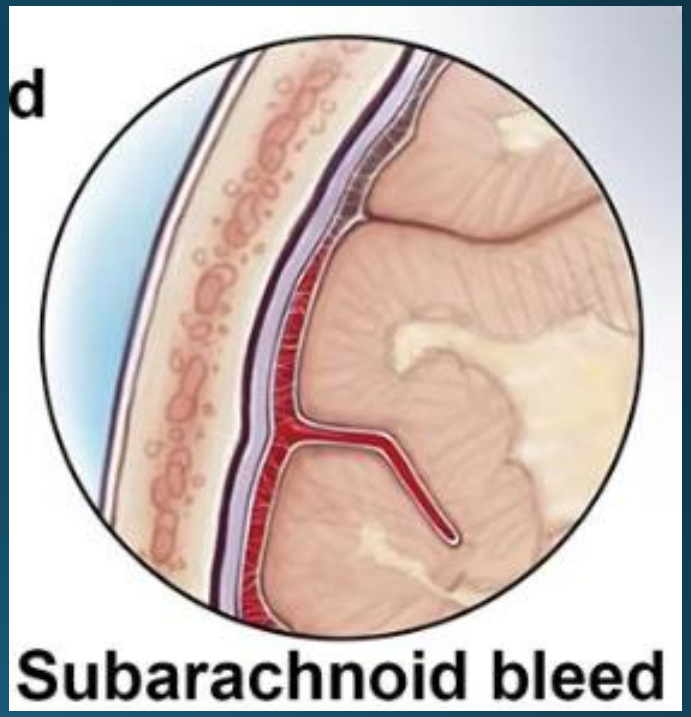
- Vasospasm risk

| Grade | Presentation | IVH | Risk |
|---|---|---|---|
| 0 | No radiographic evidence of SAH | o | 0% |
| I | Focal or diffuse thin SAH with thickness less than 1 mm | – | 6–24% |
| II | Focal or diffuse thin SAH with thickness less than 1 mm | + | 15–33% |
| III | Focal or diffuse thick SAH with thickness great-er than 1 mm | – | 33–35% |
| IV | Focal or diffuse thick SAH with thickness great-er than 1 mm | + | 34–40% |

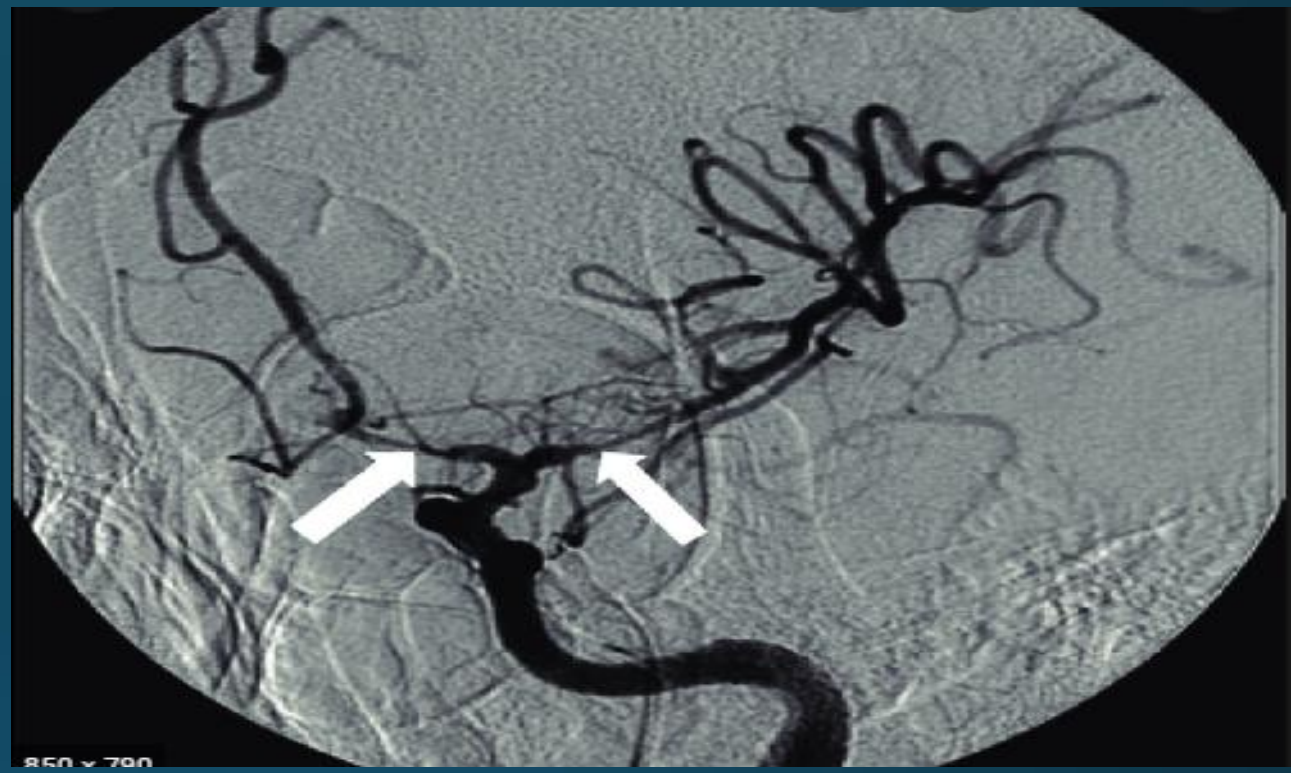
# SAH vs other ICH



# Delayed Cerebral Ischemia (DCI)



- 3-14 days
- Peak -> day 7
- Up to 21 days

- Affects 30%, of which 30% die & further 34% disabled
  - => 19% post-SAH patients

- Spectrum; radiographically - 30-70%

*Dorsch NW, King MT. A review of cerebral vasospasm in aneurysmal subarachnoid haemorrhage Part I: Incidence and effects. J Clin Neurosci. 1994 Jan;1(1):19-26. doi: 10.1016/0967-5868(94)90005-1. PMID: 18638721.*

# DCI

- Imaging:
  - DSA (nicardipine, **plasty**); CTA
  - Trancranial Doppler
    - Lindegaard ratio

The Lindegaard ratio is simply the ratio of flow in the ipsilateral MCA to ICA. Values are highly operator dependent.

- Prevention:
  - **Nimodipine within 96h p.o.**
    - ***Indirect action***
  - normothermia, euvolemia, euglycemia
  - anemia avoidance (***not*** *to transfuse*)

*Dorsch NW, King MT. A review of cerebral vasospasm in aneurysmal subarachnoid haemorrhage Part I: Incidence and effects. J Clin Neurosci. 1994 Jan;1(1):19-26. doi: 10.1016/0967-5868(94)90005-1. PMID: 18638721.*

# DCI

## Questionable

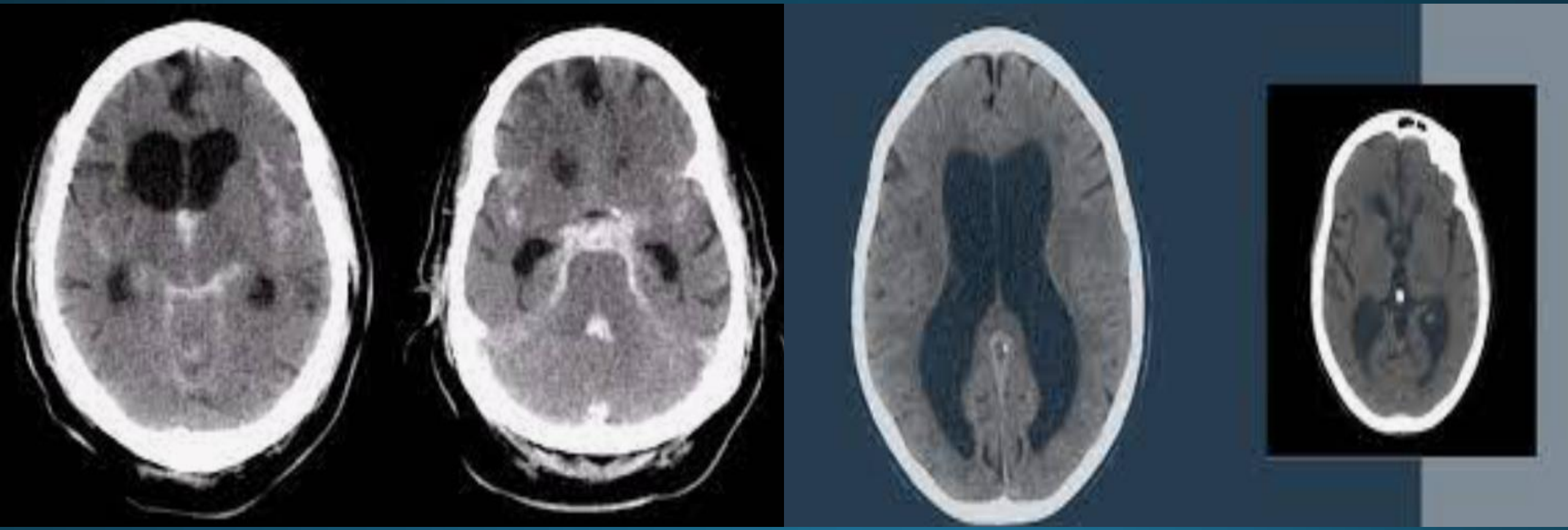
- Triple H therapy – no longer indicated
- Statins


*Lombard F, Britz GW, Warner DS. Simvastatin in subarachnoid haemorrhage: beyond the short-term. Lancet Neurol. 2014 Nov;13(11):1073.*

*Kirkpatrick PJ, Turner CL, Smith C, et al. Simvastatin in aneurysmal subarachnoid haemorrhage (STASH): a multicentre randomised phase 3 trial. Lancet Neurol. 2014; 13:666–675*

*Dorsch NW, King MT. A r eview of cerebral vasospasm in aneurysmal subarachnoid haemorrhage Part I: Incidence and effects. J Clin Neurosci. 1994 Jan;1(1):19-26. doi: 10.1016/0967-5868(94)90005-1. PMID: 18638721.*

# Hydrocephalus (HC)



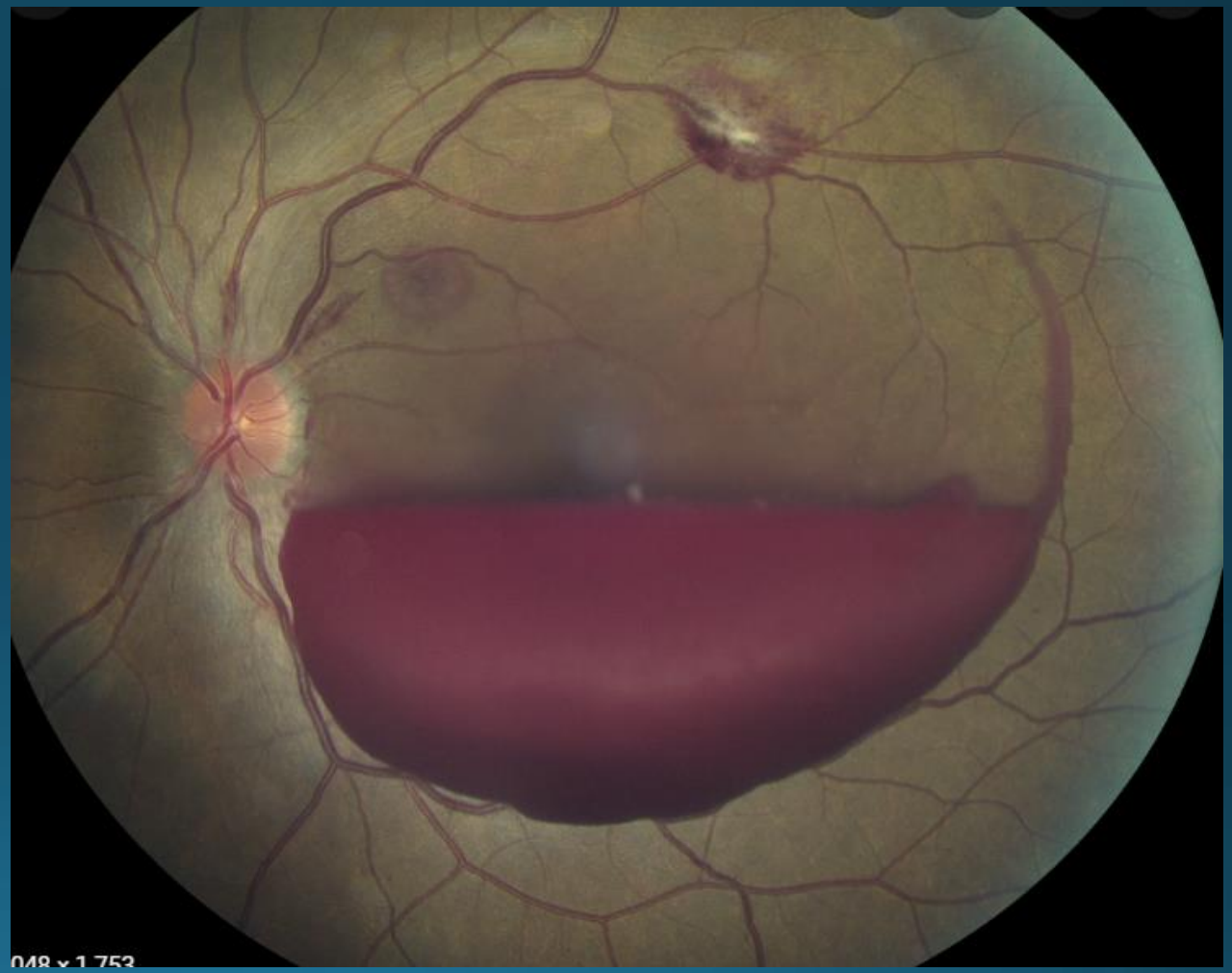
1.

*Wartenberg KE, Stephan A. Mayer, Mayer SA. Medical complications after subarachnoid hemorrhage. Neurosurgery Clinics of North America. 2010 Apr 1;21(2):325–38.*

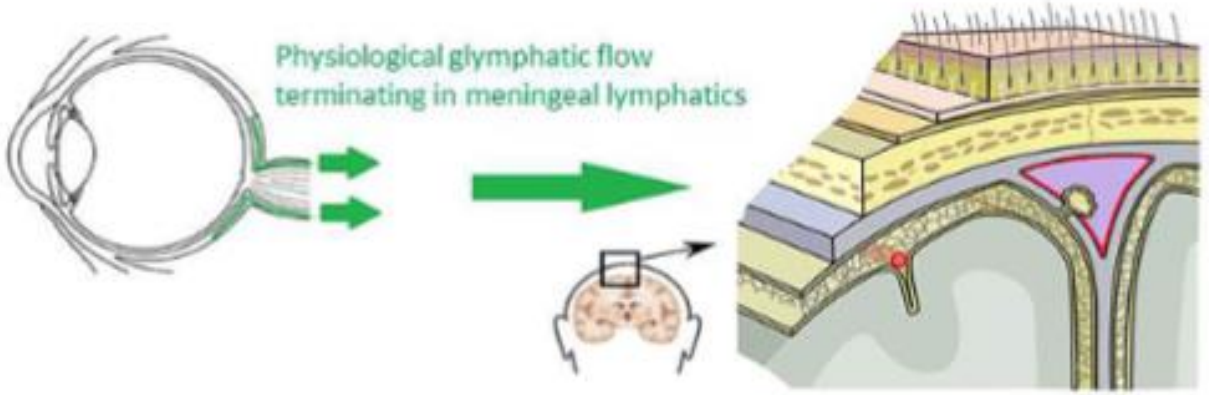
# Terson syndrome

- Prevalent – 25%
- Worse prognosis (ICP)

- neurosurgical ePearl candidate



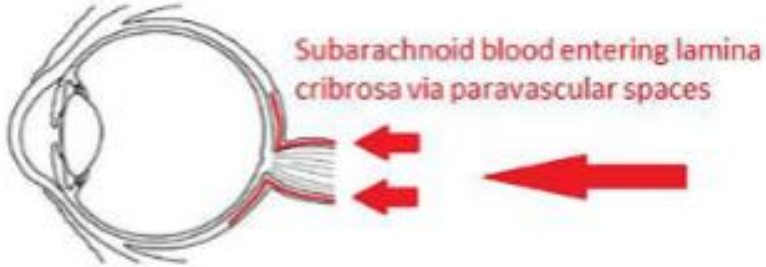
1.
*Kumaria A, Gruener AM, Dow GR, Smith SJ, Macarthur DC, Ingale HA. An explanation for Terson syndrome at last: the glymphatic reflux theory. J Neurol. 2022 Mar;269(3):1264–71.*

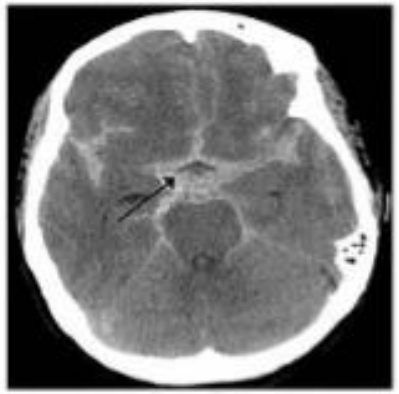



Physiological ocular glymphatic flow

Intraocular pressure > Intracranial pressure

Intracranial pressure > Intraocular pressure



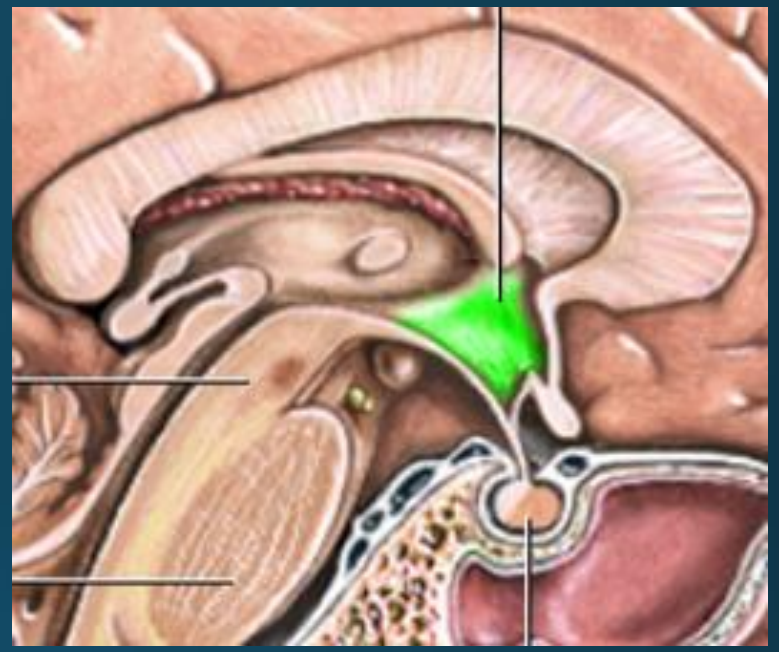
Glymphatic reflux when ICP is raised, causing subarachnoid blood to enter eye

Mechanisms of retinal bleeding:

- Raised pressure
- Bloodborne Inflammatory mediators

1. Kumaria A, Gruener AM, Dow GR, Smith SJ, Macarthur DC, Ingale HA. An explanation for Terson syndrome at last: the glymphatic reflux theory. J Neurol. 2022 Mar;269(3):1264–71.

# Medical complications



Symphatetic activation

- Fever

- hyperglycemia
  - >140 -> increases vasospasm risk

  - Neurogenic Stress Cardiomyopathy
  - Neurogenic pulmonary edema

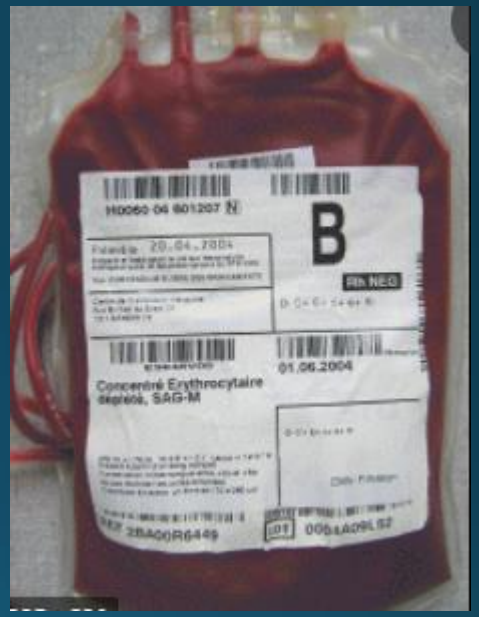
1.

*Wartenberg KE, Stephan A. Mayer, Mayer SA. Medical complications after subarachnoid hemorrhage. Neurosurgery Clinics of North America. 2010 Apr 1;21(2):325–38.*

# Medical complications

- Hyponatremia
  - SIADH (bleed -> VP surge -> dilution)
  - Cerebral salt wasting

- Anemia 36%
  - SAH & phlebotomies
  - **Transfusion**, not anemia is the risk
  - => only if <7 / <10 + Sx




*Tenny S, Thorell W. Cerebral Salt Wasting Syndrome. [Updated 2022 Feb 21]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK534855/*

1.

*Wartenberg KE, Stephan A. Mayer, Mayer SA. Medical complications after subarachnoid hemorrhage. Neurosurgery Clinics of North America. 2010 Apr 1;21(2):325–38.*

# Post-SAH syndrome

- Cognitive disabilities, emotional disturbance
- Hazard ratio 2,74 x population
  - Highest among any types of stroke (ischemic 1,72)
  - 30% v-spasm, **95% - dementia**
- 45% unable to get back to profession
- General atrophy esp. **temporomesial area**

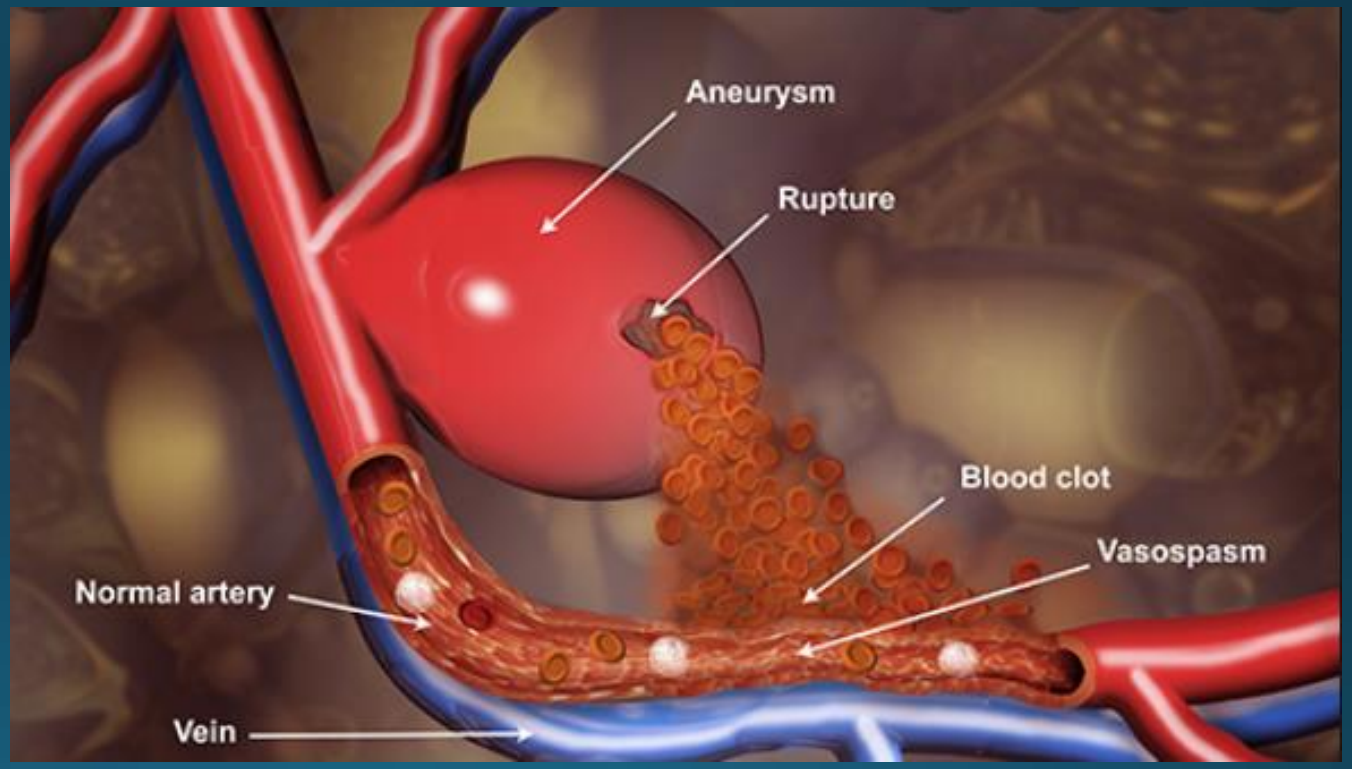
*Neurosurgery Grand Rounds – Gavin Britz, M.D.*
*https://www.youtube.com/watch?v=S257fdOvJDw&list=PL85QPzmgY94GQAEpHQkqokNqiRVbMDZtM&index=2*

# Impaired arterioral reactivity

- 2 days p-SAH:
  - Adenosine
  - NO
  - hyperCO2

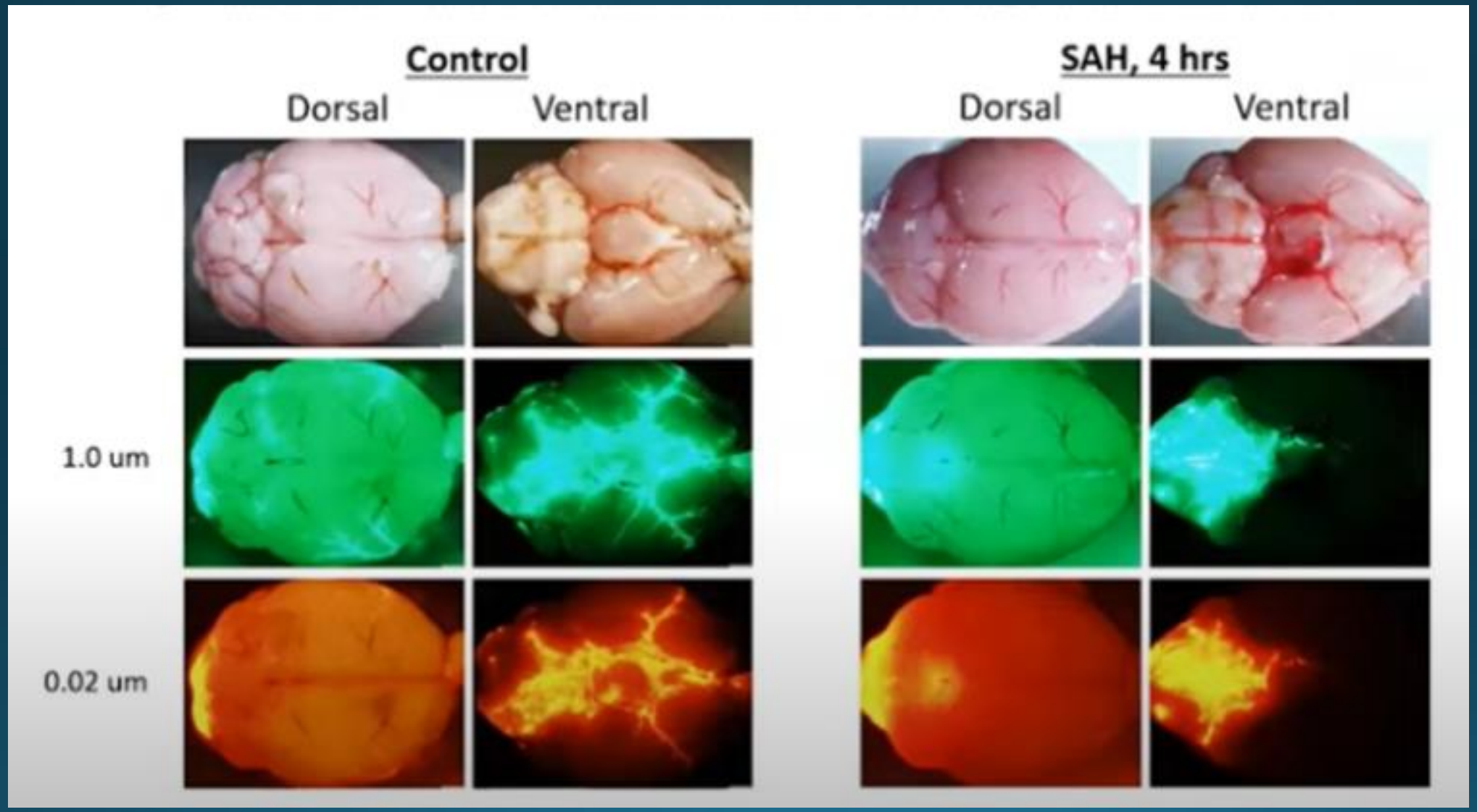
- -> Irreversible changes

- Part et al.
- Ishiguro et al.
- **Britz et al.**




1.

*Britz GW, Meno JR, Park IS, Abel TJ, Chowdhary A, Nguyen TSK, et al. Time-dependent alterations in functional and pharmacological arteriolar reactivity after subarachnoid hemorrhage. Stroke. 2007 Apr;38(4):1329–35.*

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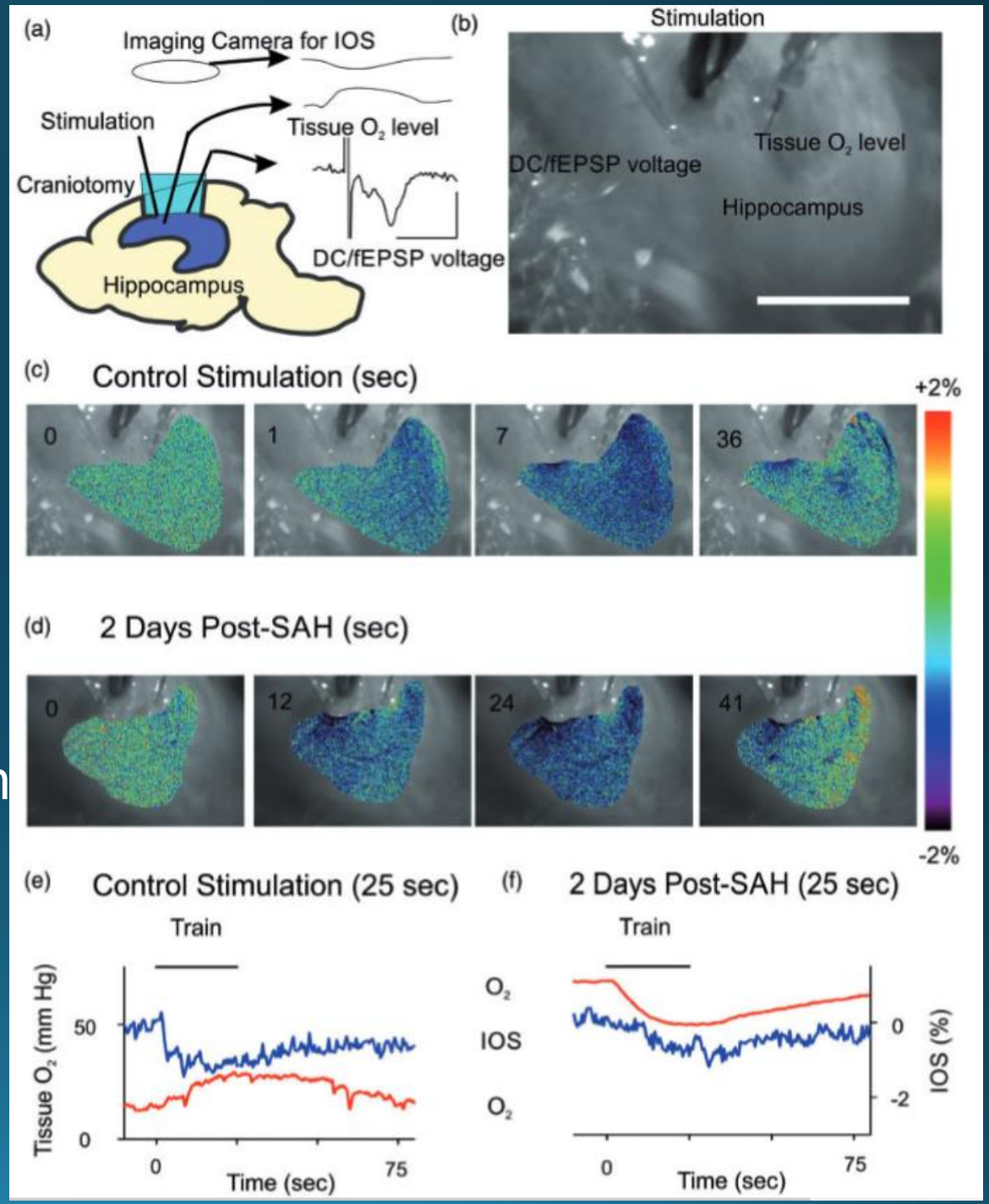
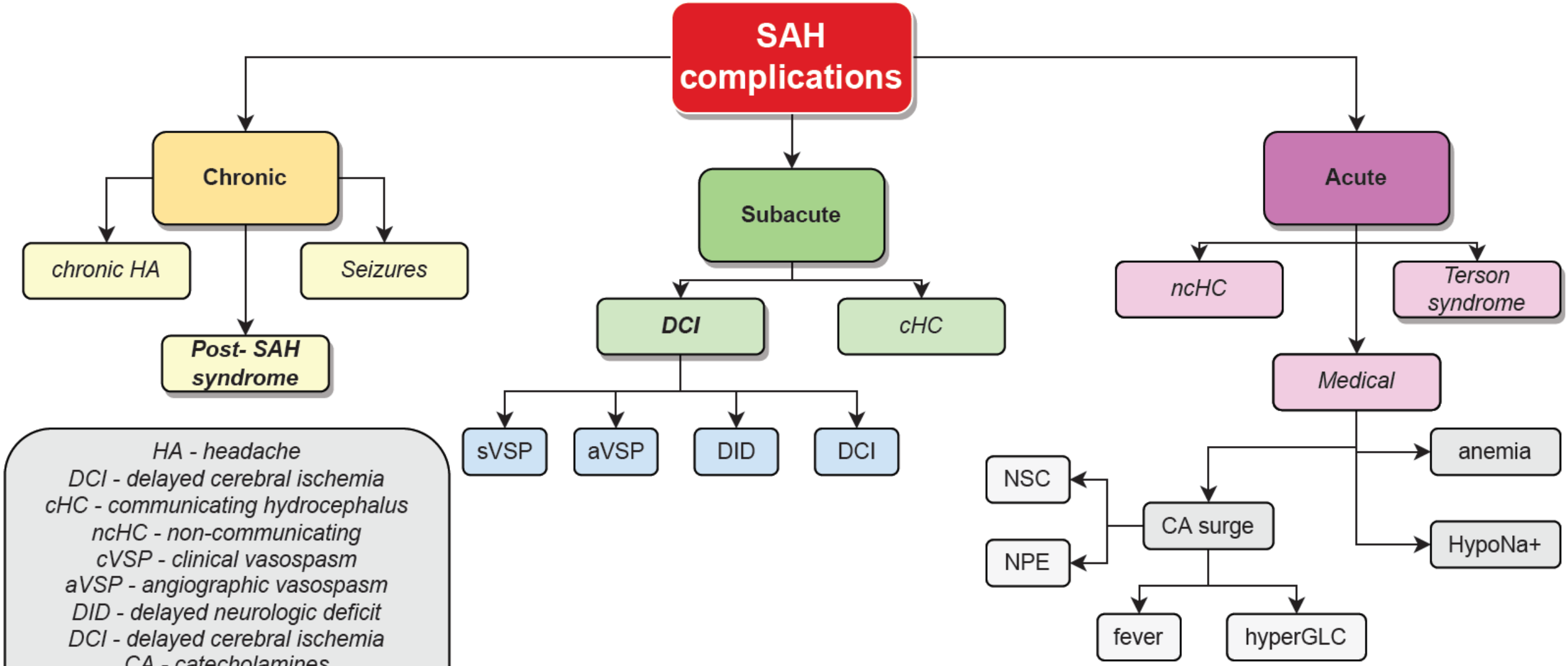
- Rats hippocampus oxygenation
- Sparse vasculature
- Paradoxical resting & active oxygenation




*Galeffi F, Degan S, Britz G, Turner DA. Dysregulation of oxygen hemodynamic responses to synaptic train stimulation in a rat hippocampal model of subarachnoid hemorrhage. J Cereb Blood Flow Metab. 2016 Apr;36(4):696–701.*

- Post-SAH chronic, **daily** headaches – **30%**
- Seizures development

# Summary




HA - headache
DCI - delayed cerebral ischemia
cHC - communicating hydrocephalus
ncHC - non-communicating
cVSP - clinical vasospasm
aVSP - angiographic vasospasm
DID - delayed neurologic deficit
DCI - delayed cerebral ischemia
CA - catecholamines
NSC - neurogenic stress cardiomyopathy
NPE - neurogenic pulmonary edema