

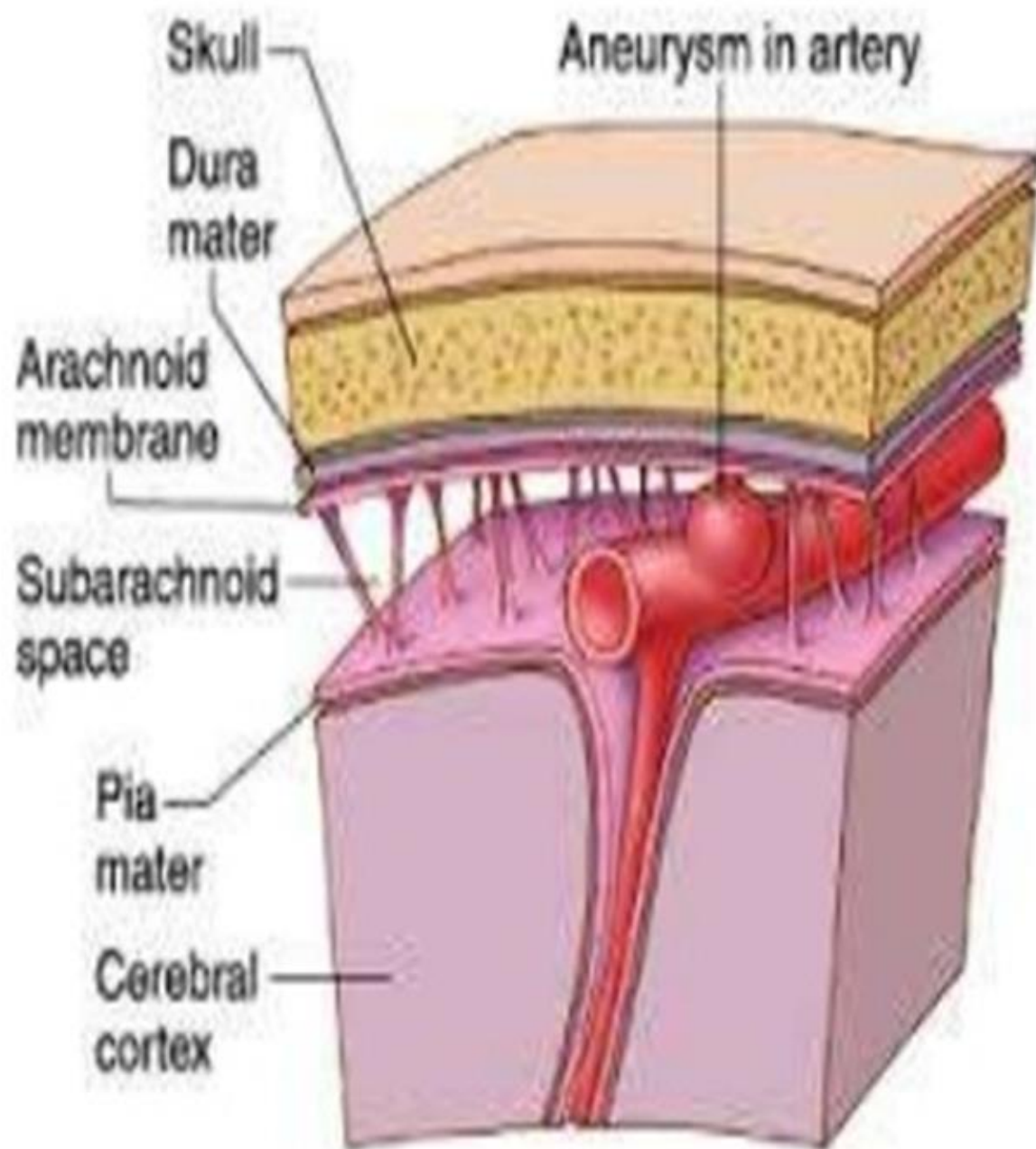
SUBARACHNOID HEMORRHAGE

Ali Ayyad

DEFINITION

- Extravasation of blood into the subarachnoid space between the pia and arachnoid membranes

- SAH is a neurological emergency
- Hemorrhage in the subarachnoid space



CAUSES

Traumatic

Spontaneous/ Nontraumatic

Unidentified cause 15%

Rarely: Spinal AV malformation

Brain Tumor

Blood Disorders

SPONTANEOUS SAH

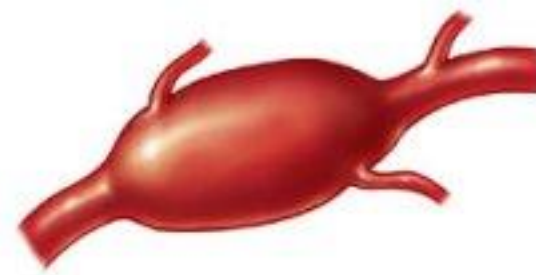
- Rupture of “berry,” or saccular, aneurysms of the basal vessels of the brain comprises 77% of SAH cases.
- AVMs are the second most identifiable cause of SAH, accounting for 10% of cases of SAH.
- AVMs are thought to occur in approximately 4-5% of the general population, of which 10-15% are symptomatic.

- Less common causes of SAH include the following:
- Fusiform and mycotic aneurysms
- Fibromuscular dysplasia
- Blood dyscrasias
- Moyamoya disease
- Infection
- Neoplasm
- Amyloid angiopathy (especially in elderly people)
- Vasculitis
- Idiopathic SAH

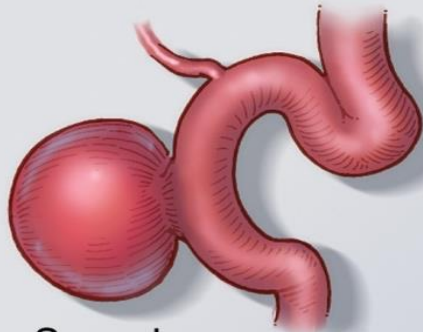
INTRACRANIAL ANEURYSMS



Saccular Aneurysm



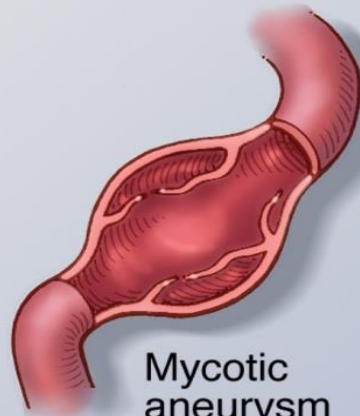
Fusiform Aneurysm



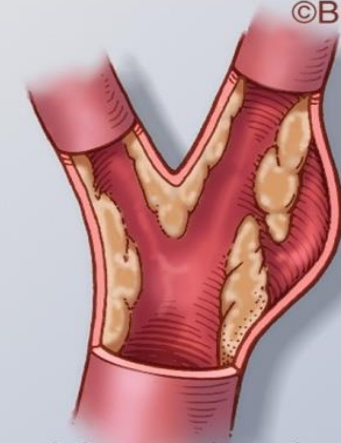
Saccular aneurysm



Ruptured Aneurysm



Mycotic aneurysm



Atherosclerotic aneurysm



Traumatic aneurysm

©BNI 1995

EPIDEMIOLOGY

- Age:
- Incidence increases with age and peaks at age 50 years
- 80% of cases of SAH occur in people aged 40-65 years
- Rare in children younger than 10 years (accounts for only 0.5% of all cases)
- Sex:
- Higher incidence in women (3:2)
- Risk of SAH is significantly higher in the third trimester of pregnancy
- Race: higher risk in blacks than in whites

EPIDEMIOLOGY

➤ In the US:

- 6-16 cases per 100,000 population
- 30,000 cases per year

➤ Worldwide:

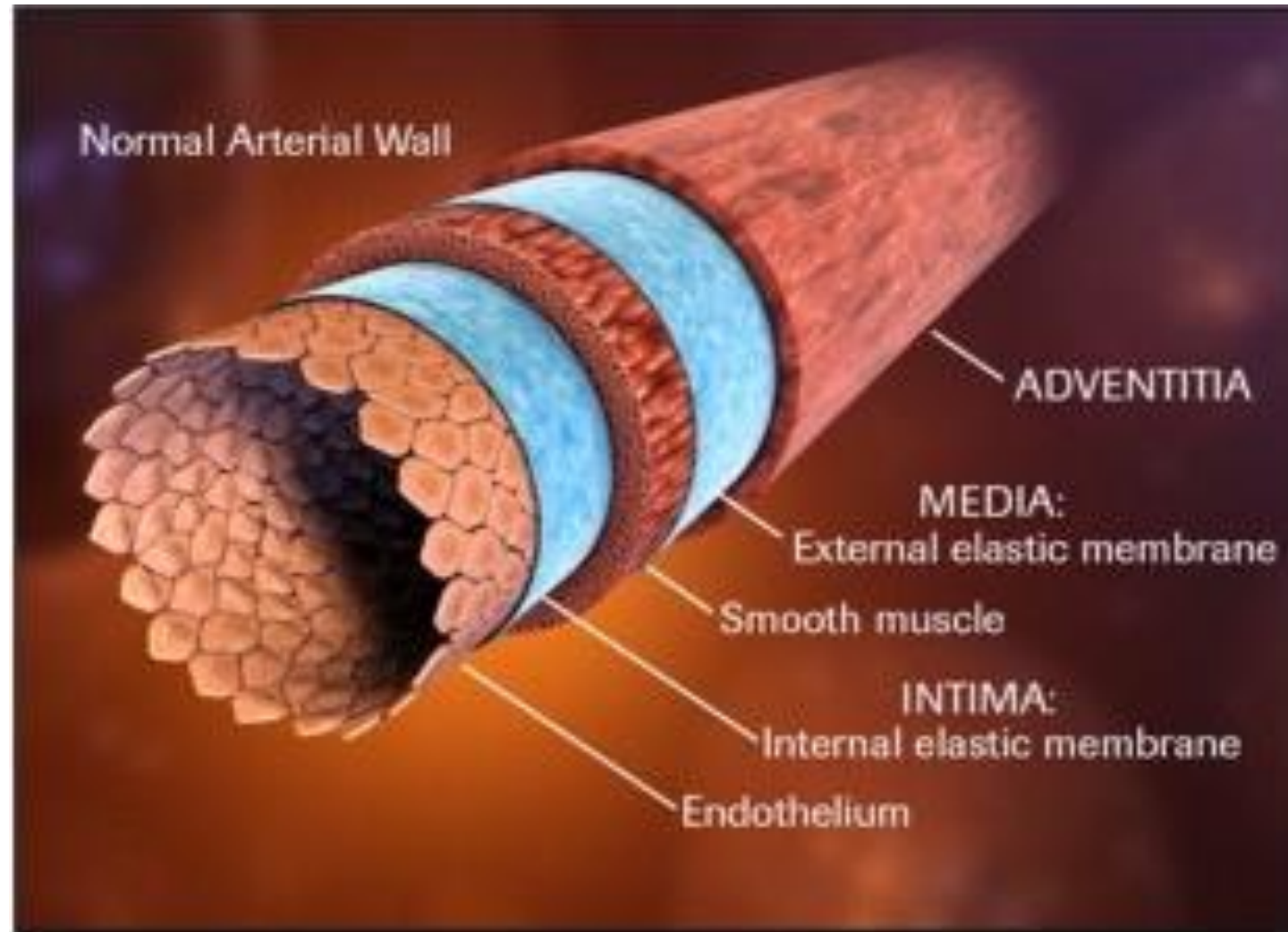
- 2-49 cases per 100,000 population
- Highest rates occur in Japan and Finland

NATURAL HISTORY

- An estimated 15% of patients die before reaching the hospital.
- Approximately 25% of patients die within 24 hours, with or without medical attention.
- The mortality rate at the end of 1 week approaches 40%.
- Half of all patients die in the first 6 months.
- Age-adjusted mortality rates are 62% greater in females than in males and 57% greater in blacks than in whites.
- 40% of all survivors have major neurologic deficits.
- Morbidity and mortality increase with age and are related to the overall health status of the patient.

ETIOLOGY

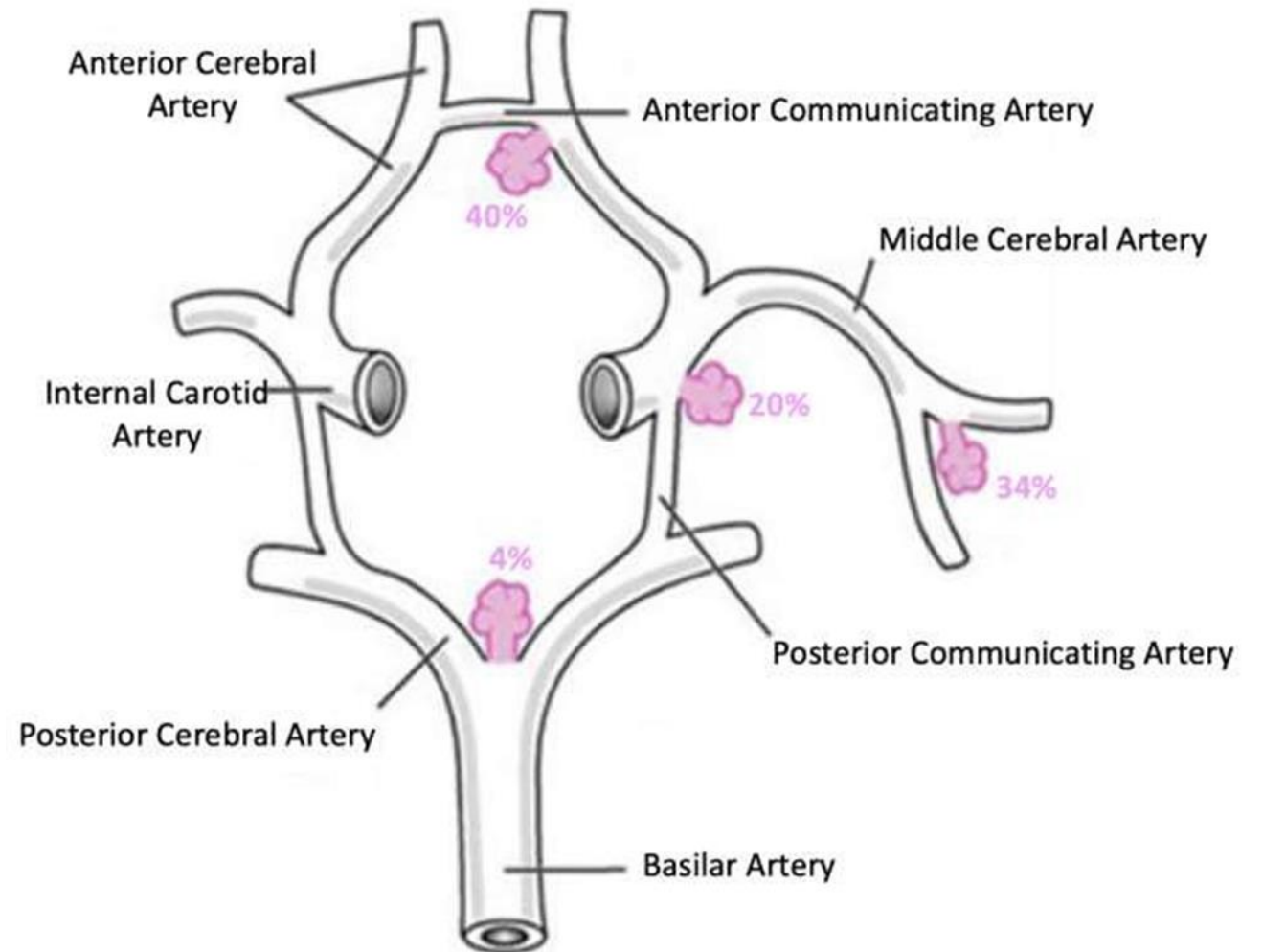
- Both congenital and acquired factors are thought to be involved in the etiology of cerebral aneurysms
- 80% of vessels at autopsy have congenital defects in the muscle and elastic tissue of the arterial media of the circle of Willis. These lead to microaneurysmal dilatation in 20% of the population (<2 mm) and larger dilation (>5 mm) and aneurysms in 5% of the population.
- Acquired factors thought to be associated with aneurysmal formation include the following:
 - Atherosclerosis
 - Hypertension
 - Hemodynamic stress



RUPTURE RISK FACTORS

- Hypertension
- Atherosclerosis
- Smoking
- Oral contraceptive pills
- Vigorous exercise and hemodynamic stress
- Pregnancy

LOCATION OF ANEURYSMS ON THE CIRCLE OF WILLIS



CLINICAL PRESENTATION

- Headache
- Decreased level of consciousness
- Meningism (neck rigidity, vomiting, photophobia, and fever)
- Seizure
- Focal neurological signs due to intracerebral hemorrhage, focal pressure by an aneurysm, or vasospasm



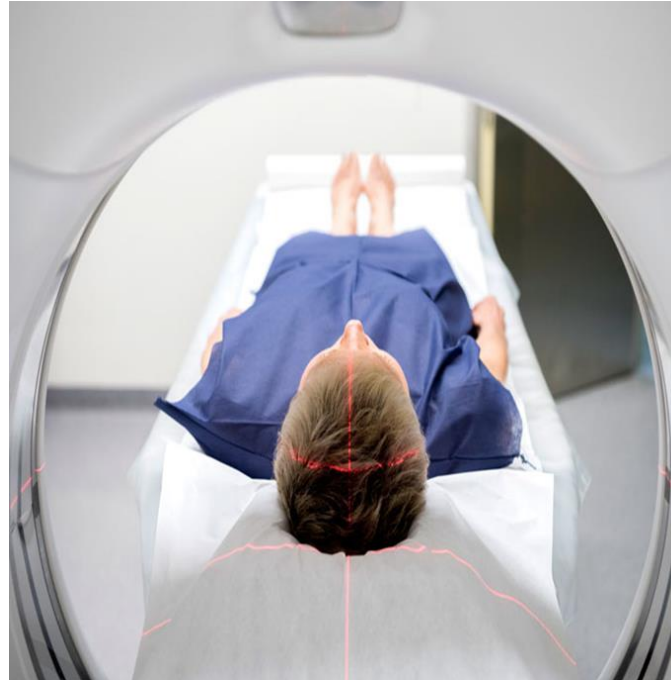
Figure 5—Third cranial nerve palsy. Complete ptosis of the left eyelid with obscuration of the visual axis, suggesting third cranial nerve palsy from an intracranial aneurysm.

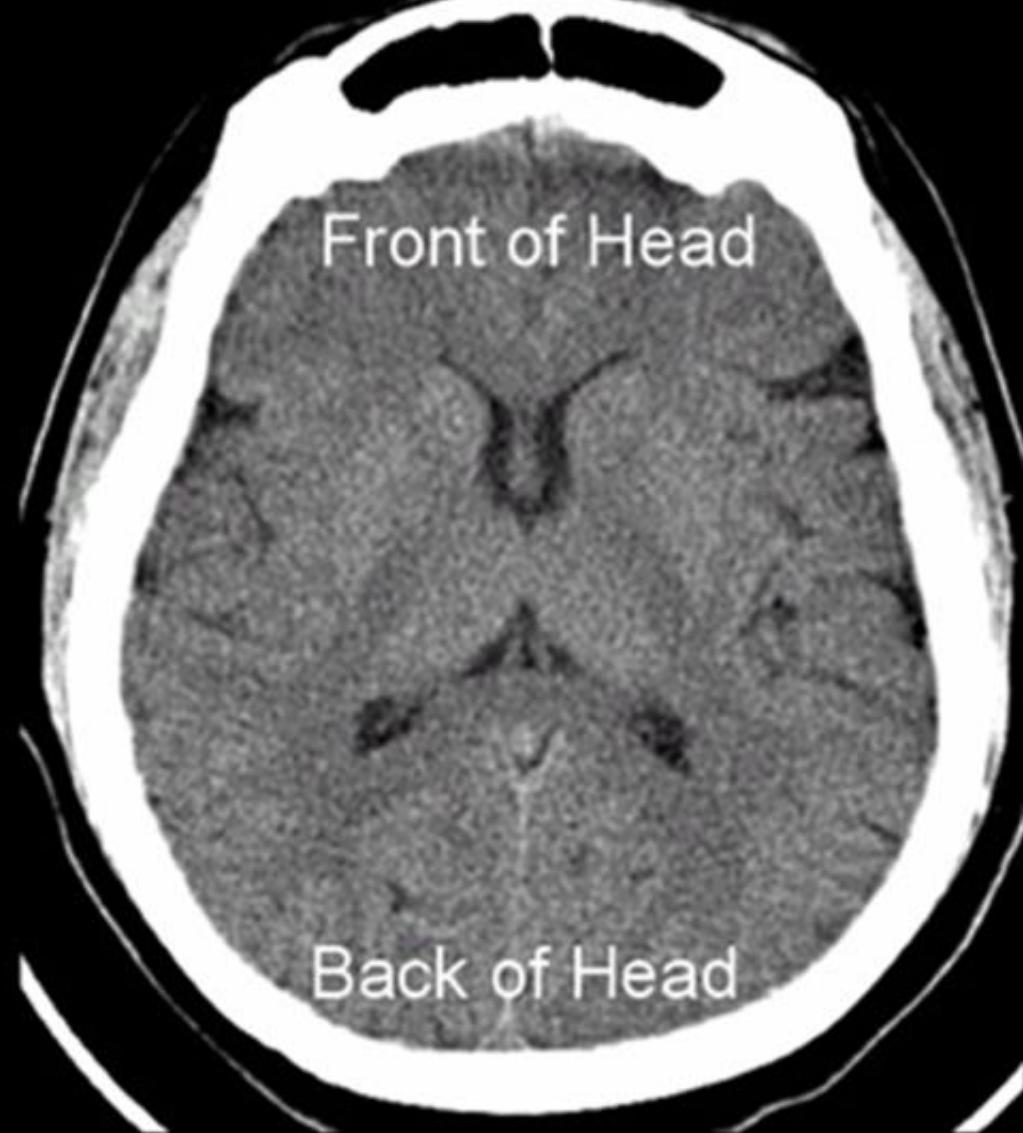


Figure 6—Dilated pupil. Left eye deviated outward with pupillary dilatation—signs of oculomotor nerve palsy.

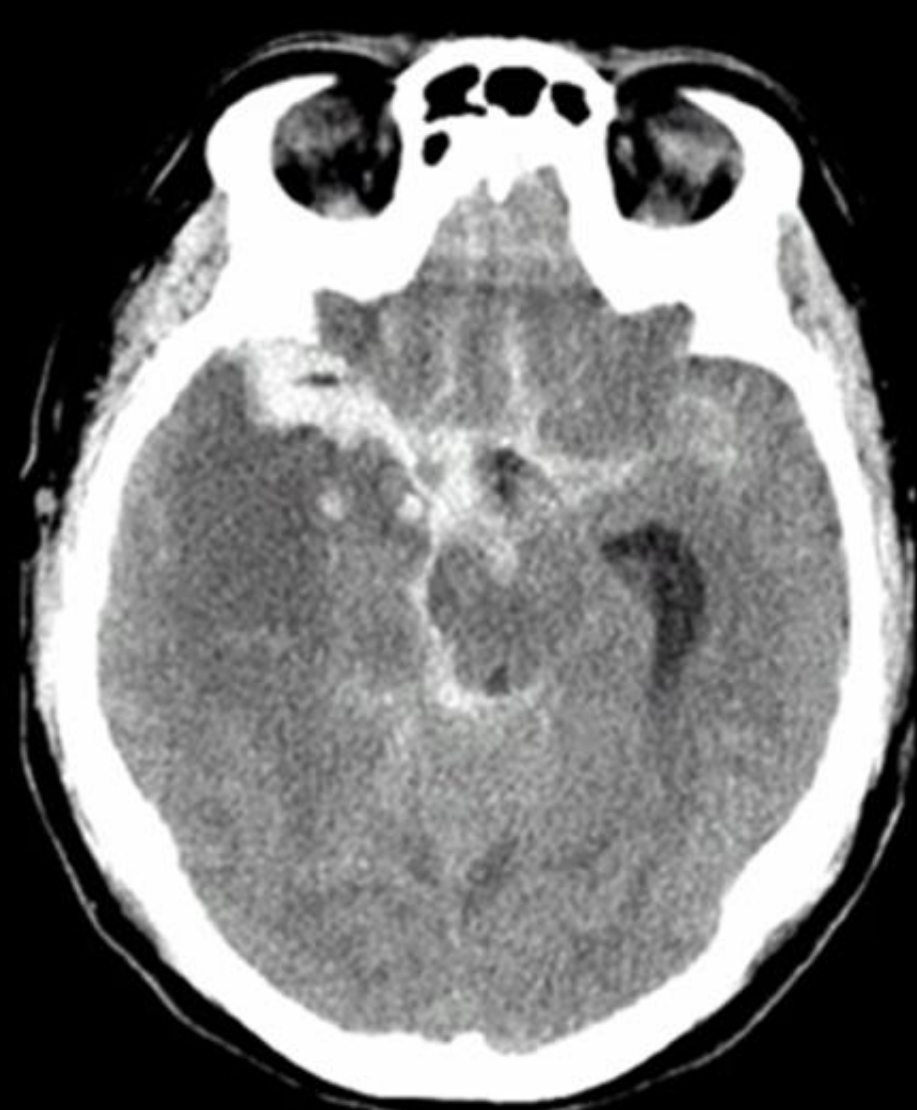
DIAGNOSIS

- Clinical suspicion
- Non contrasted CT
- Lumbar puncture





Normal CT Scan
Slice of Brain



Subarachnoid Hemorrhage
(bright white areas)
CT Scan Slice of Brain



Normal
CSF



Xanthochromic
CSF



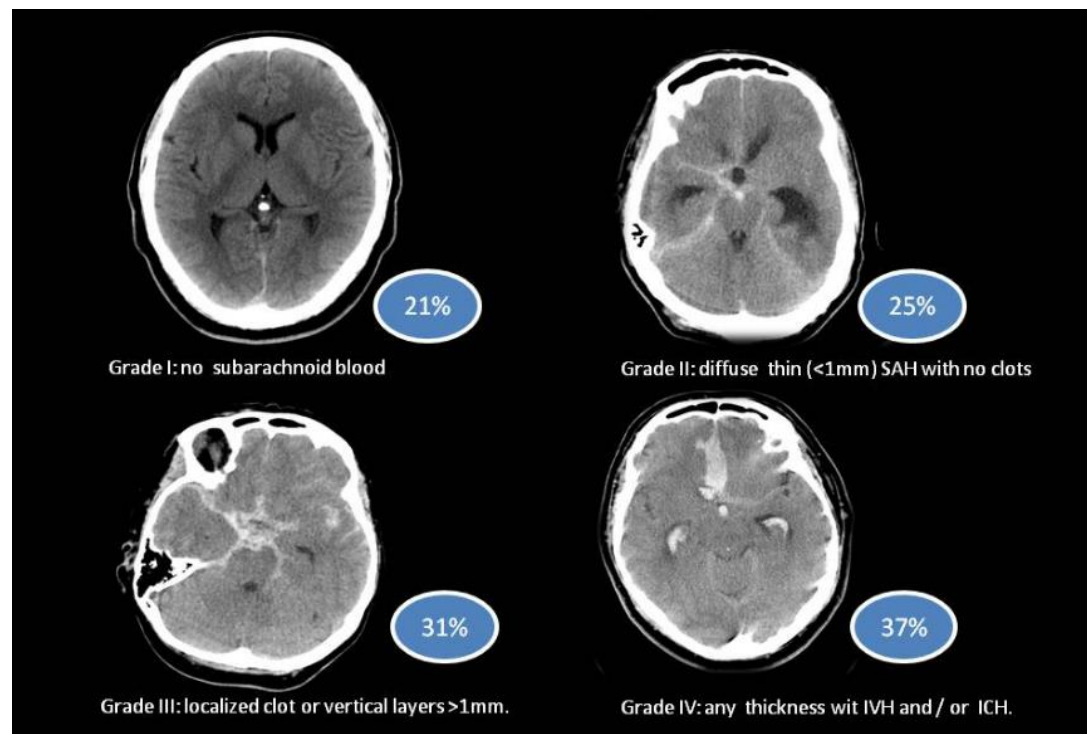
Table 9.3 Subarachnoid haemorrhage grading systems.

*Hunt and Hess grading system**

Grade	Description
1	Asymptomatic, or minimal headache and slight nuchal rigidity
2	Moderate to severe headache, nuchal rigidity, no neurological deficit (except cranial nerve palsy)
3	Drowsiness, confusion or mild focal deficit
4	Stupor, moderate to severe hemiparesis, possible early decerebrate rigidity and vegetative disturbances
5	Deep coma, decerebrate rigidity, moribund

WFNS grading system

Grade	Glasgow Coma Score (GCS)	Motor deficit
1	15	No deficit except a cranial nerve palsy
2	14–13	No deficit
3	14–13	Any deficit
4	12–7	With or without focal neurodeficit
5	6–3	Coma with or without abnormal posturing



Fisher CT Grading Scale

Fisher Group	Blood Pattern on Nonenhanced CT
1	No subarachnoid blood detected
2	Diffuse or vertical layers <1 mm thick*
3	Localized clot or vertical layers \geq 1 mm thick
4	Intracerebral or IV clot with diffuse or no SAH

*"Vertical" cisterns: interhemispheric, insular, and ambient.

MANAGEMENT

Once the diagnosis is confirmed and the patient placed in the correct clinical grade, the patient is admitted for management. The management entails:

- Stabilization of patient.
- Management of ICP.
- Prevention of complications.
- Finding the source of bleeding.
- Preparing the patient for surgery if needed.
- Treatment of complications.

MANAGEMENT

- ICU admission
- placed in a dark lit room to counteract the photophobia
- head elevated 30 degrees.
- medications: Codeine phosphate should be given for headache, laxatives administered to help prevent straining on defecation; a mild anxiolytic should be given.
- An IV line should be inserted and normal saline administered.
- A Foley's catheter should be inserted
- Blood Lab. investigations

MANAGEMENT

➤ Finding the cause

- Digital subtraction cerebral angiography (DSA)
- CT Angiography
- MRA

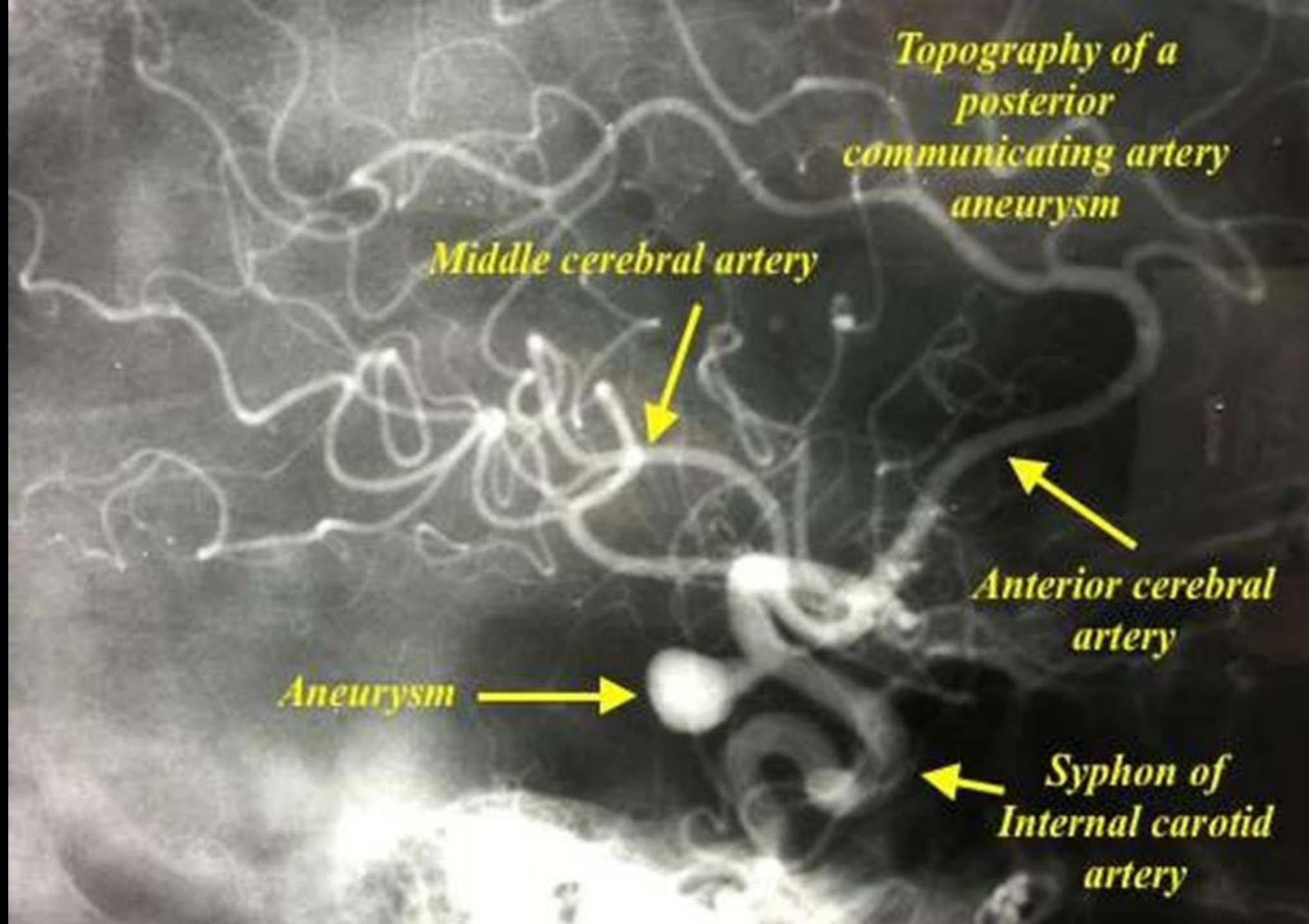
*Topography of a
posterior
communicating artery
aneurysm*

Middle cerebral artery

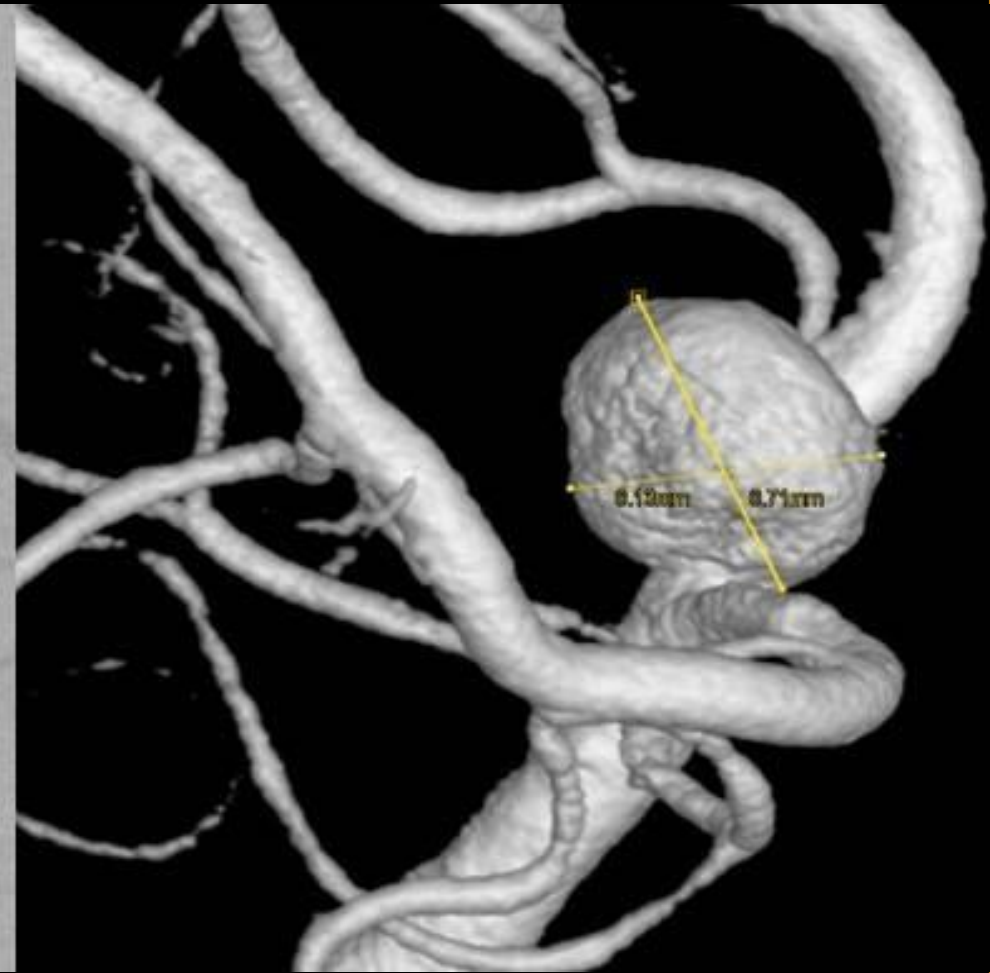
*Anterior cerebral
artery*

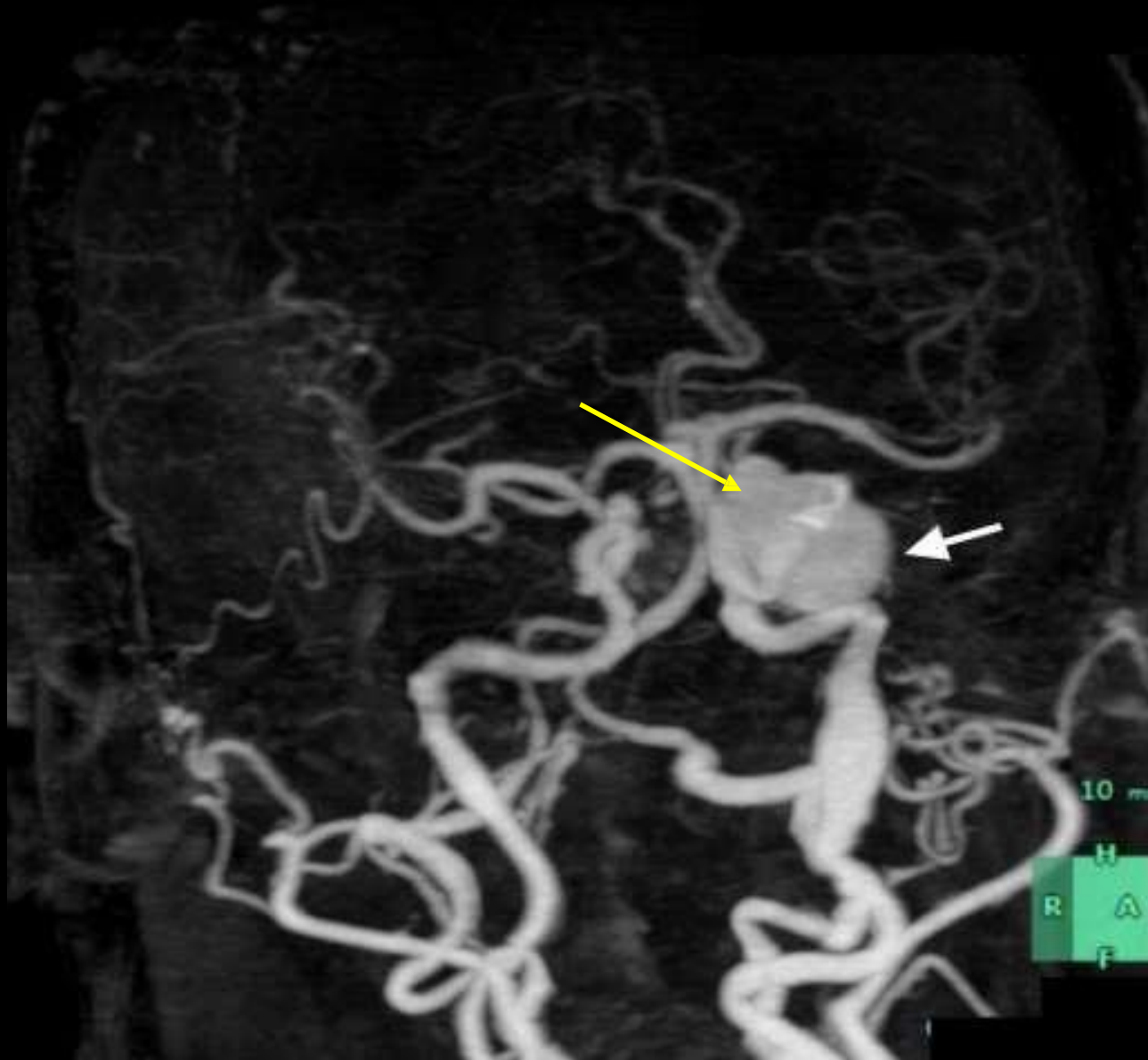
Aneurysm

*Syphon of
Internal carotid
artery*

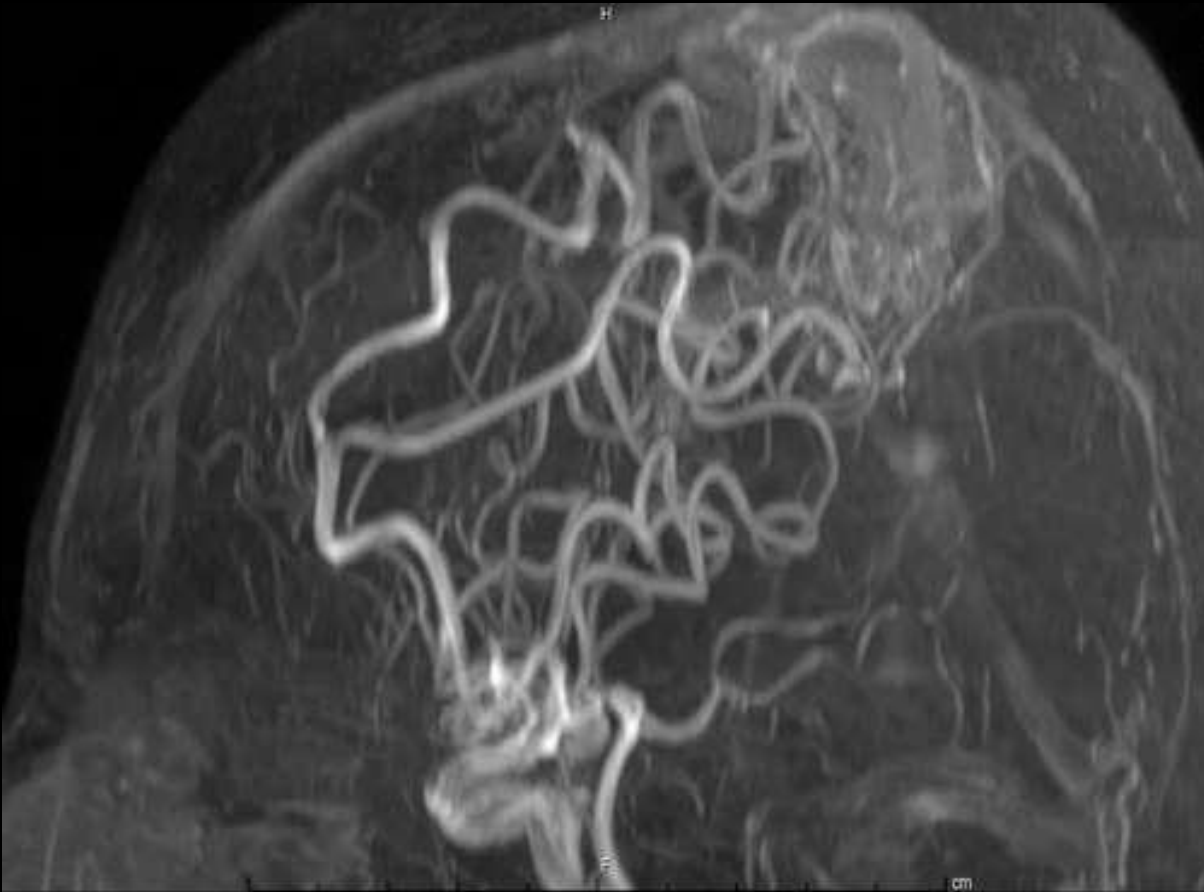


©UC Regents





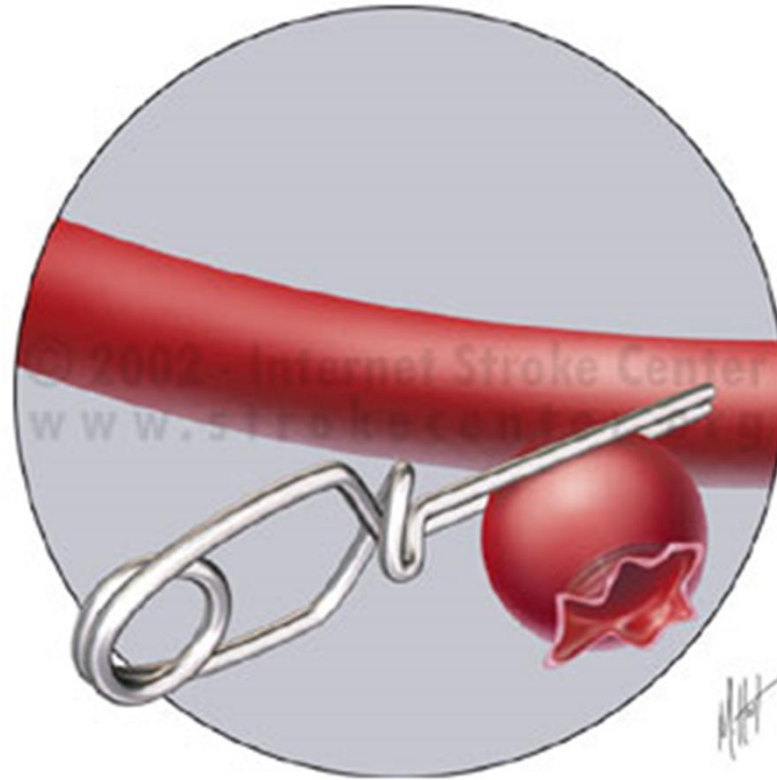
ANTERIOR COMMUNICATING
ARTERY ANEURYSM (ACoMA)



- MRI is a useful tool to diagnose AVMs that are not detected by cerebral angiography or spinal AVMs causing SAH.
- MRI can detect aneurysms 5 mm or larger with a high sensitivity.
- It can be useful for diagnosing and monitoring unruptured cerebral aneurysms.

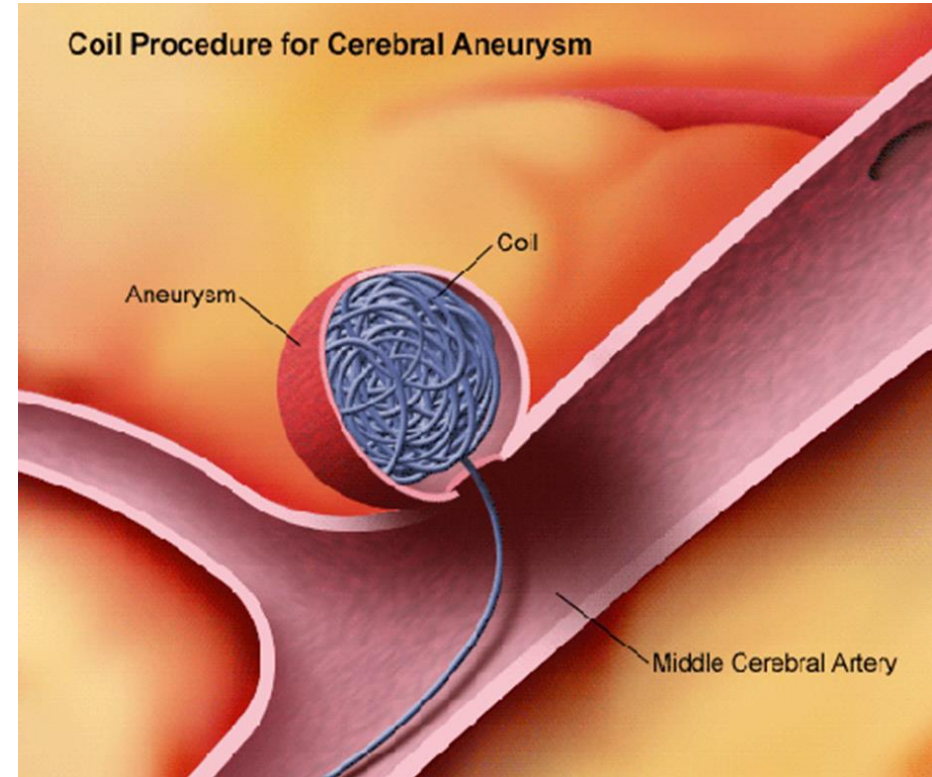
TREATMENT

Surgery



TREATMENT

Endovascular treatment





**Clipping
of an
Aneurysm**



COMPLICATIONS

➤ **Rebleeding**

The greatest risk of rebleeding occurs within the first 24 hours of rupture (4.1%).

The total risk of rebleeding is 19% at 2 weeks.

The mortality rate from rebleeding is reported to be as high as 78%.

➤ **Vasospasm**

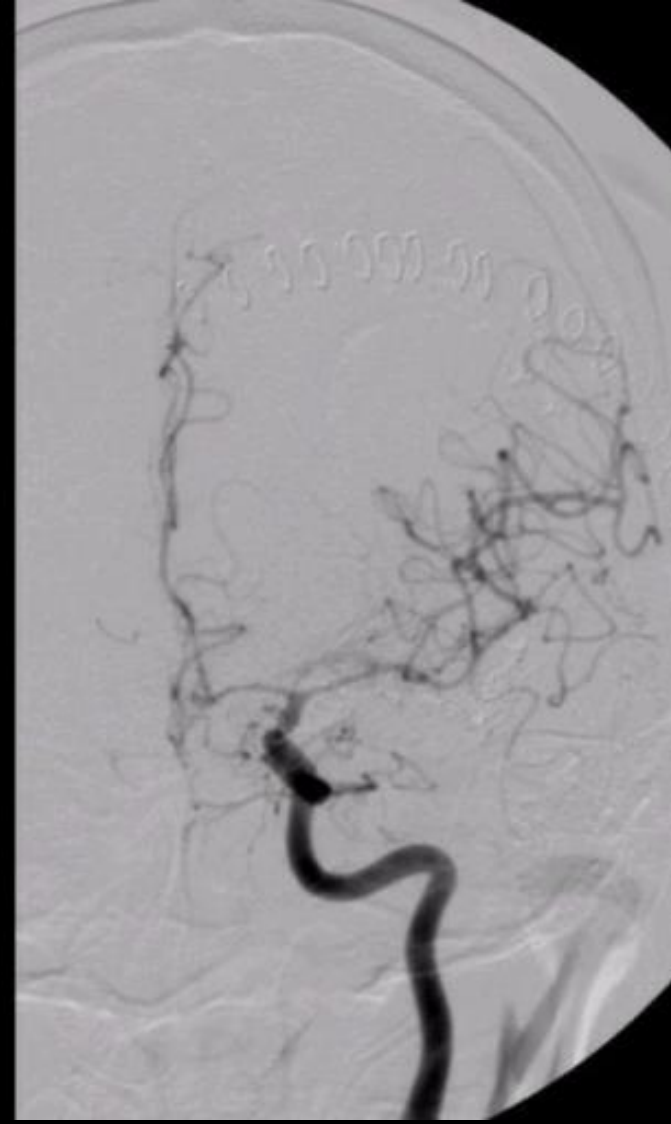
Most commonly occurs 4-14 days after the hemorrhage

➤ **Hydrocephalus**

- Acute 20% of SAH cases and usually occurs within the first 24 hours
- Chronic 10-15% of patients with SAH

COMPLICATIONS

- **Hyponatremia following SAH occurs in 10-34% of cases .There is a possible correlation with elevated levels of atrial natriuretic factor (ANF) and syndrome of inappropriate secretion of antidiuretic hormone (SIADH)**
- **Seizures occur in 25% of patients following SAH and are most common after rupture of middle cerebral artery aneurysms**
- **Acute pulmonary edema and hypoxia are almost universal in severe SAH**
- **Cardiac dysfunction manifested as arrhythmias in 90% of the cases**



MULTIPLE INTRACRANIAL ANEURYSMS

- **These occur in 15% of cases. Usually one of these aneurysms ruptures, leading to the characteristic signs and symptoms. The course of the disease is the same. However, multiplicity poses problems in diagnosis; Which aneurysm did bleed, and what to do to those, which did not?**

B

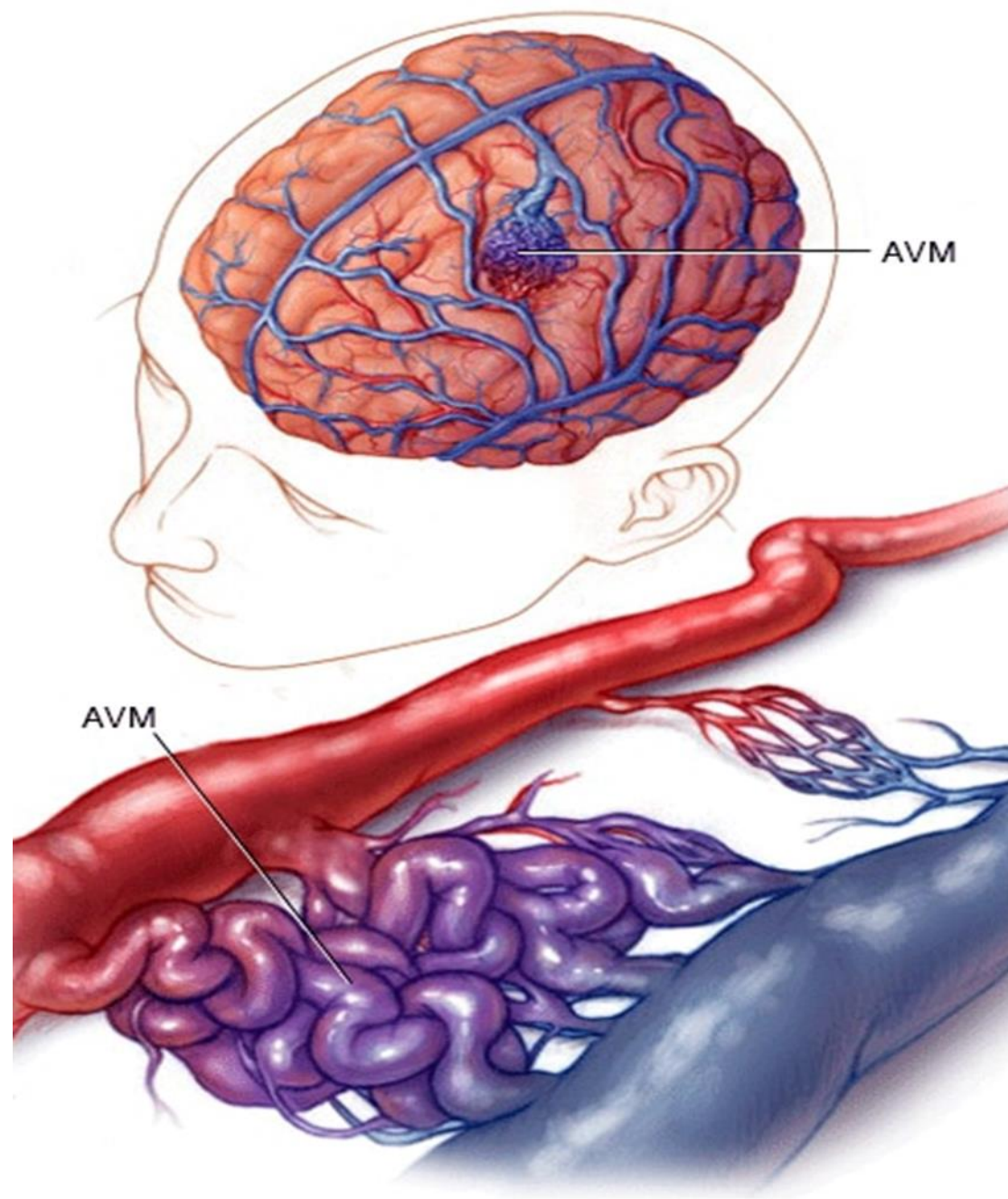


PROGNOSIS

- **Studies show that grade I Hunt and Hess has a 70% survival rate, 60% for grade 2, 50% for grade 3, 20% for grade 4, and 10% for grade 5**
- **Most survivors have either a transient or a permanent cognitive deficit**

INTRACRANIAL ARTERIO-VENOUS MALFORMATIONS (AVM)

- **An arteriovenous malformation (AVM) is an abnormal connection between an artery and a vein. The blood instead of passing from the artery to the capillaries and then to the vein; passes directly to the vein. The vein becomes arterialized and some brain tissue will be deprived of its normal blood supply.**
- **AVMs are considered a congenital anomaly in which the capillary bed is lacking with subsequent growth due to multiple biological factors. They tend to occur less than intracranial aneurysms by a ratio of 1 to 5, occurring in about one case in a thousand. They tend to occur in younger patients than aneurysms**



INTRACRANIAL ARTERIO-VEINOUS MALFORMATIONS (AVM)

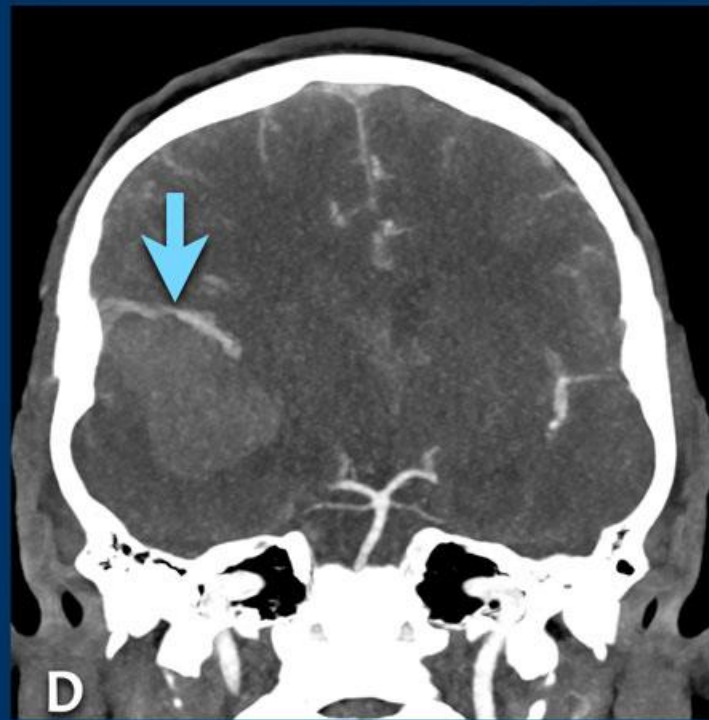
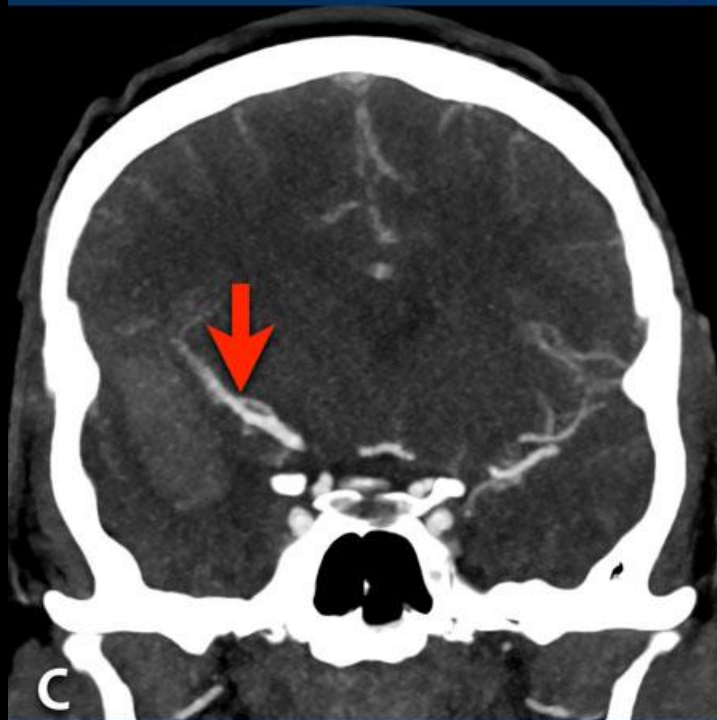
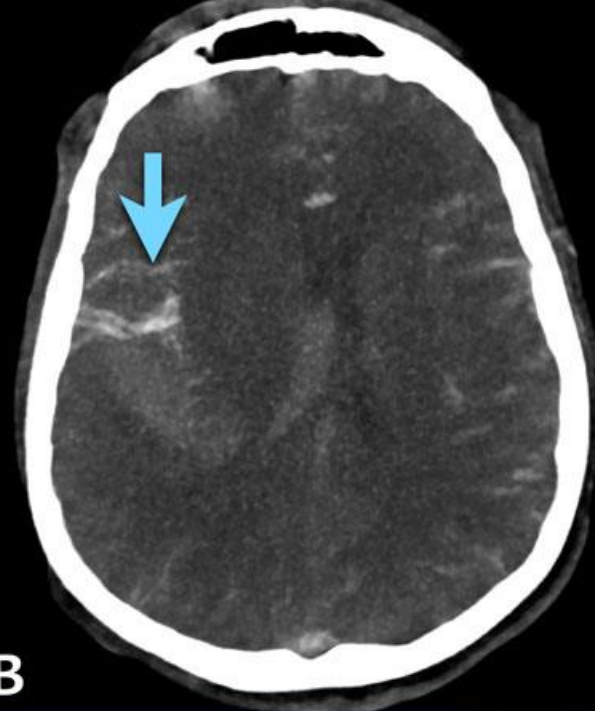
The presentation of AVMs can be one of four modes;

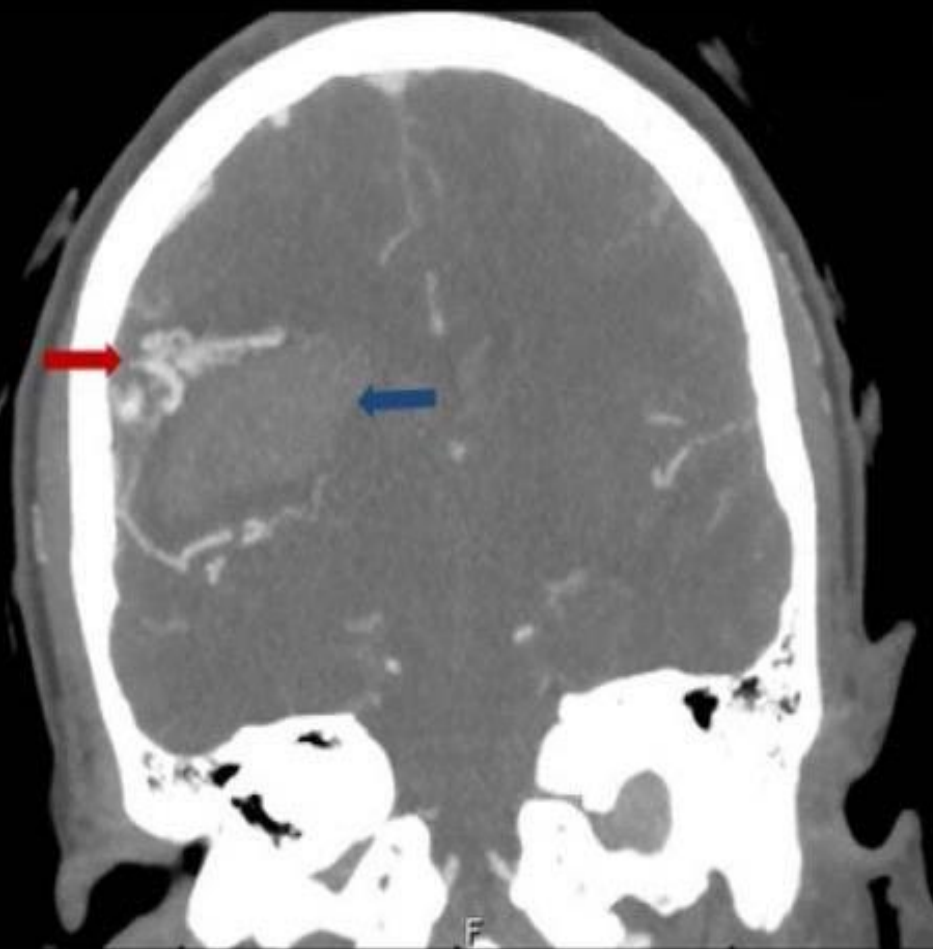
- ICH or SAH (50%) and usually caused by the smaller lesions.
- Seizures in as much as 45% of cases. Usually caused by the larger lesions.
- Recurrent headaches in about 30% of cases.
- Neurological deficits due to ischemia (shunting or steal syndrome), or pressure in about 20% of cases.

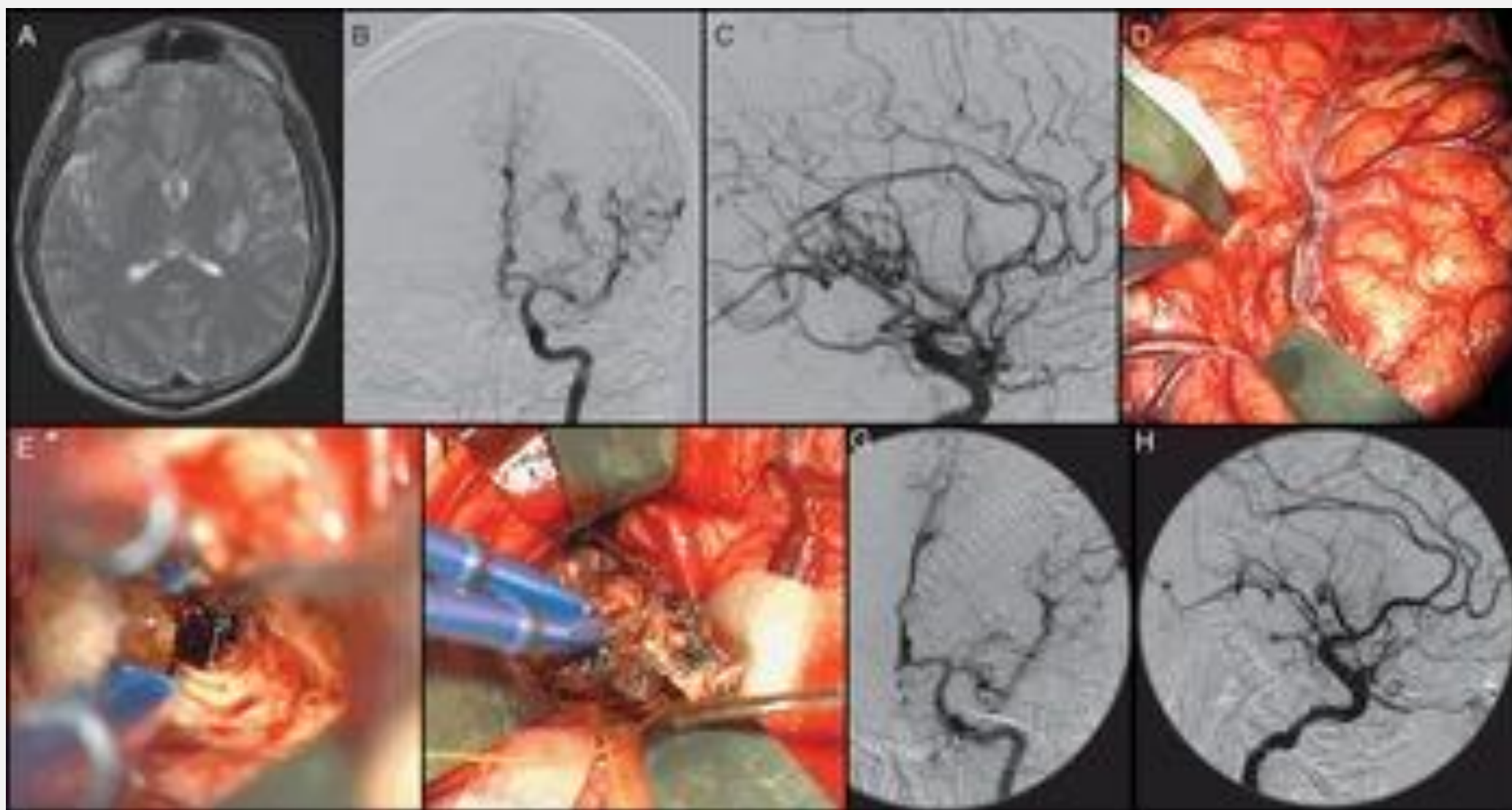
INTRACRANIAL ARTERIO-VEINOUS MALFORMATIONS (AVM)

- The annual risk of bleeding from an AVM is between 1 and 2%, less than that of aneurysms, and the mortality is much less at 10%. AVMs are found to develop aneurysms in about 5-10% .
- Diagnosis is made by CT and then angiography in the case of emergent presentation, and by MRI and angiography if the AVM present with headaches, seizures or neurological deficits.
- AVMs are usually classified by the Spetzler-Martin grading system, which grades AVMs into 5 grades (1-5), and a 6th inoperable grade. Criteria used for grading include the size of the lesion, the eloquence of adjacent brain and the venous drainage system.
- AVMs are treated by surgical excision, or embolization or radiotherapy by the Gamma Knife or a combination of any depending on grading system

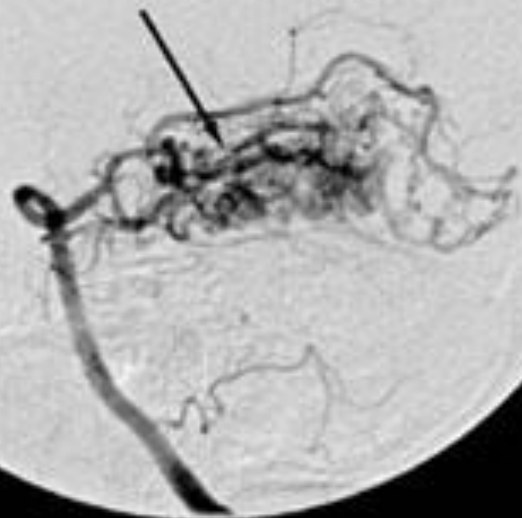
Spetzler-Martin classification				
		Characteristics	Points	
	Size of nidus	small <3 cm	1	
		medium 3-6 cm	2	
		large > 6 cm	3	
	Eloquence of adjacent brain	non-eloquent	0	
		eloquent	1	
	Venous drainage	superficial only	0	
		deep veins	1	







**Large vascular
malformation**



After embolization

