I_❤_NEUROSURGERY INITIATIVE

Subarachnoid Hemorrhage

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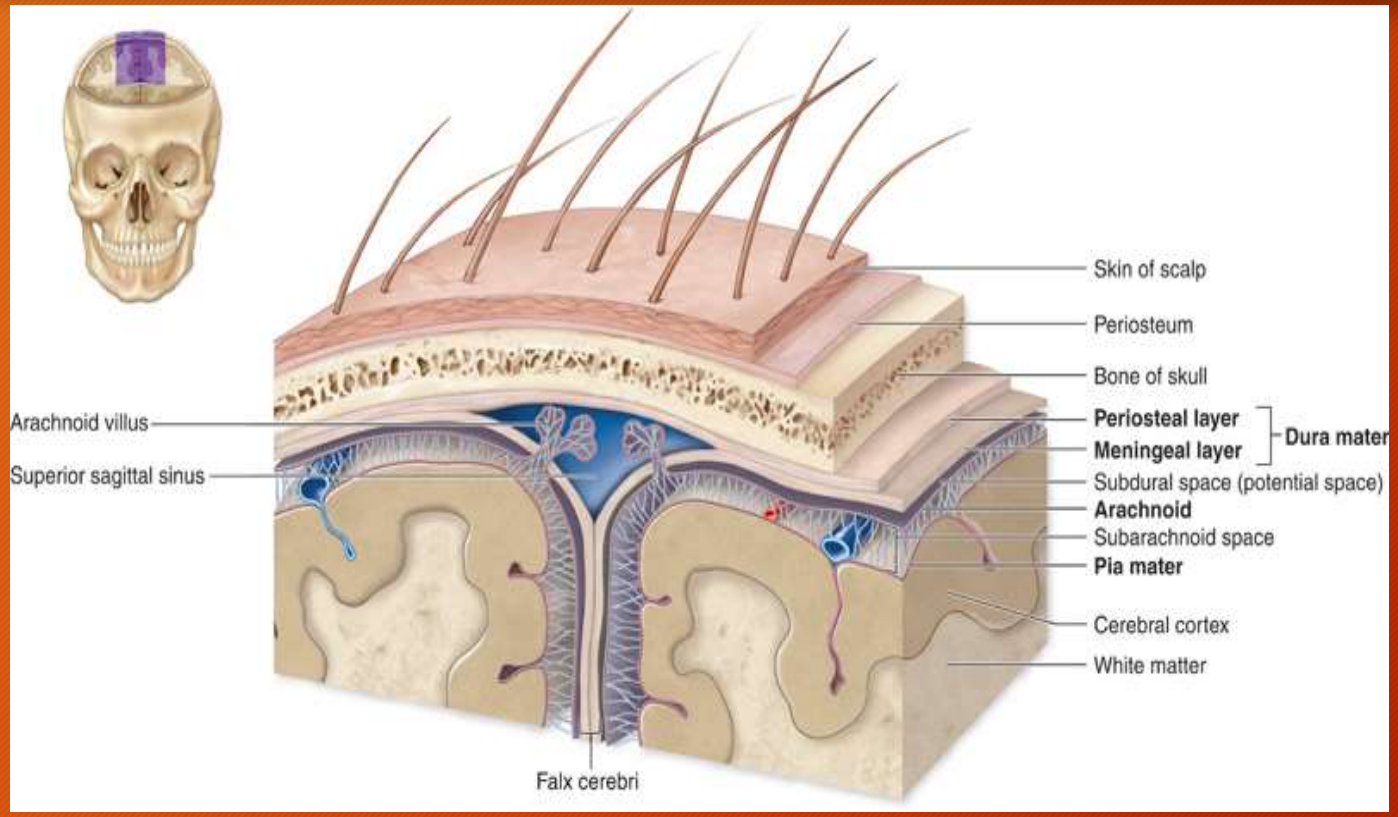
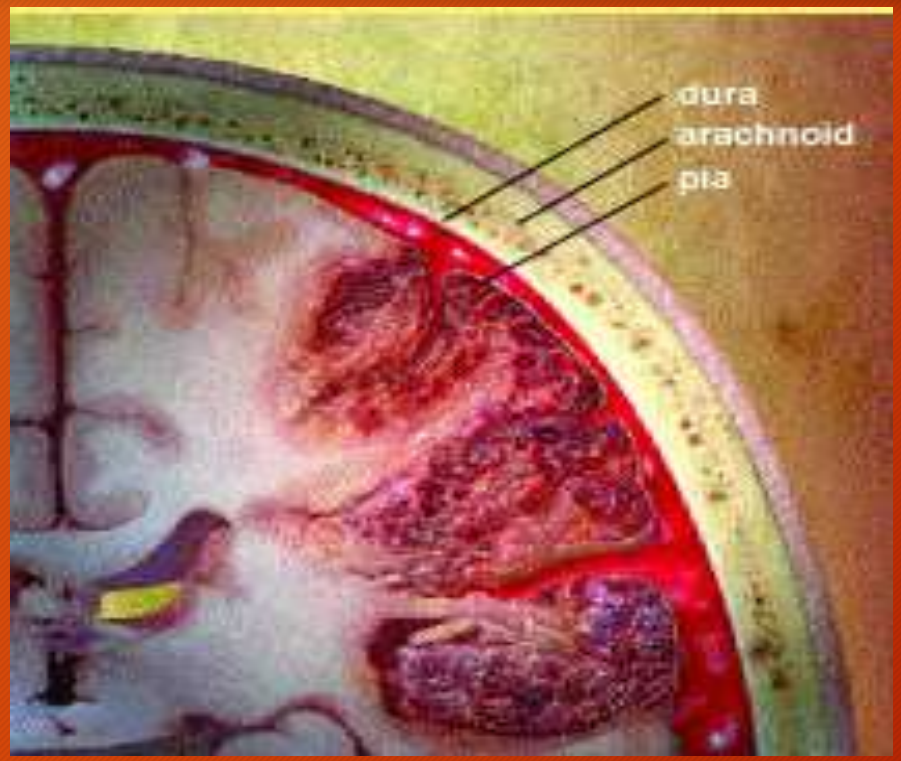
Definition

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

Layers of the Scalp and brain coverings



Subarachnoid Hemorrhage



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.

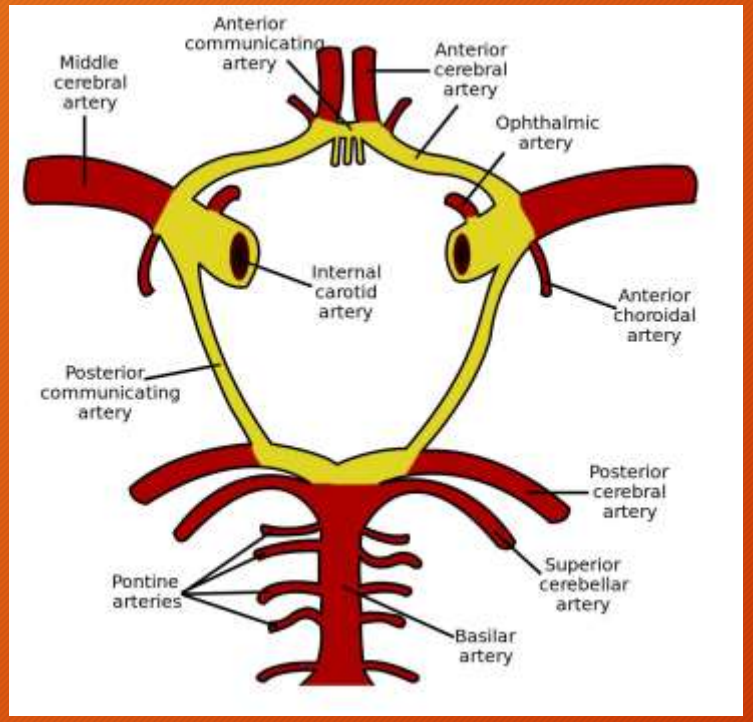
Etiology

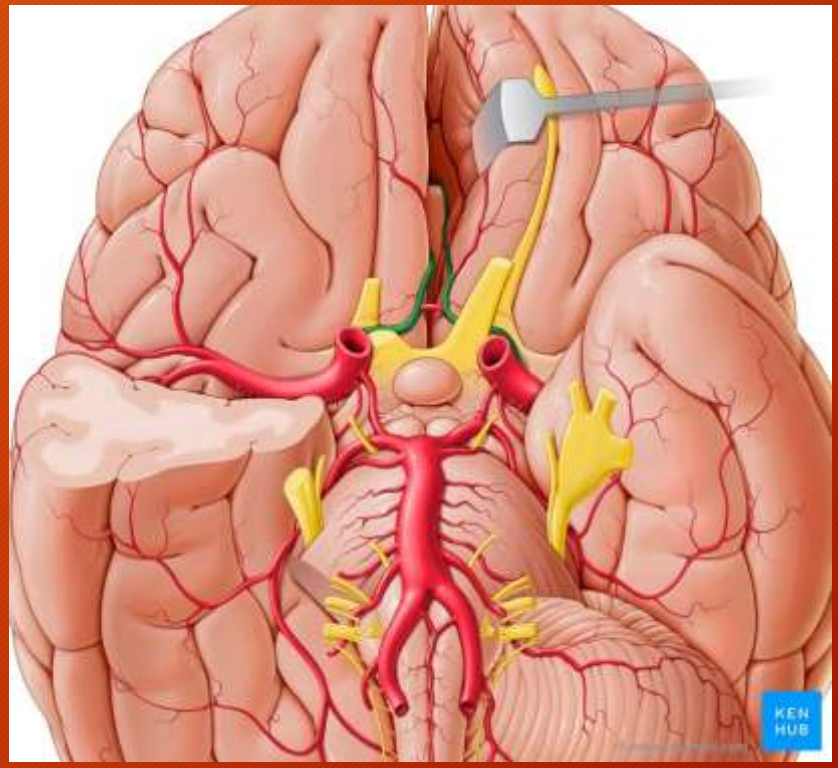
- The most common cause of SAH is trauma, but here we refer to non-traumatic SAH and we call it Spontaneous SAH
- Spontaneous SAH is usually the result of aneurysmal or arterio-venous malformation (AVM) leakage or rupture.

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

The circle of Willis





Causes

- 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.

causes

- SAH in children is much less common than in adults.
- In children the most common cause is due to AVM's.
- Cerebral aneurysm as a cause of SAH becomes more frequent than AVM's over the age of 20 years.

Causes

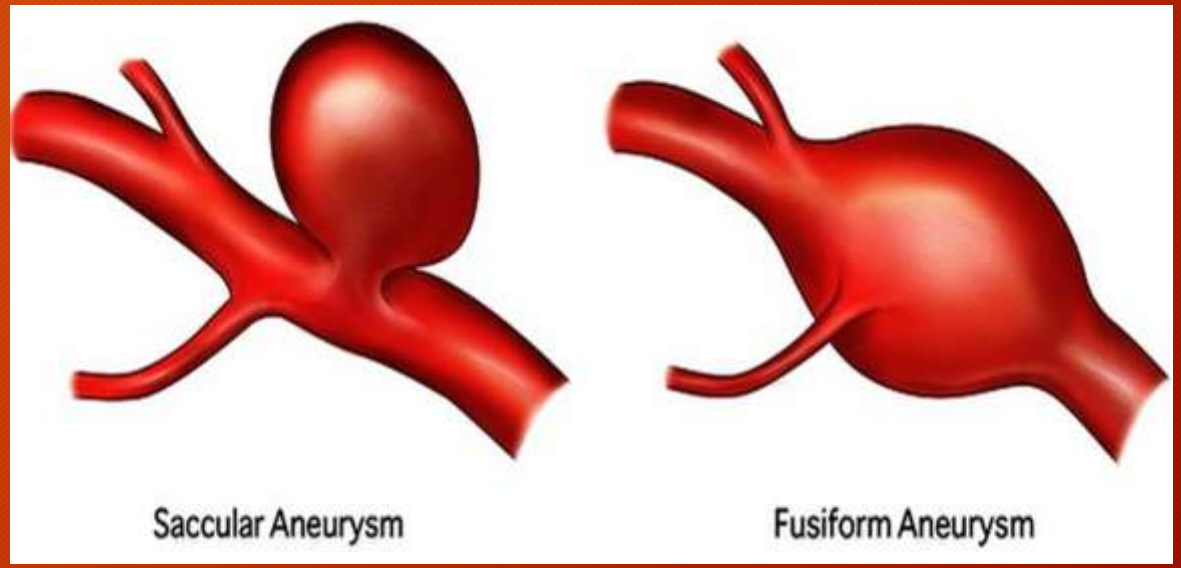
- Less common causes of SAH include the following:
  - Fusiform and mycotic aneurysms
  - Fibromuscular dysplasia
  - Blood dyscrasias
  - Moyamoya disease
  - Infection
  - Neoplasm
  - Amyloid angiopathy (especially in elderly people)
  - Vasculitis
  - Idiopathic SAH
- In 15% of the cases the cause remains unknown.

Risk Factors

- Smoking appears to be a significant risk factor, as does heavy alcohol consumption.
- The risk of AVM rupture is greater during pregnancy.
- The following do not appear to be significant risk factors for SAH:
  - Use of oral contraceptives
  - Hormone replacement therapy
  - Hypercholesterolemia

Types of aneurysms

Aneurysms are named according to their **shape** (configuration) into berry (saccular) or fusiform, or according to their **origin**, into congenital, traumatic or mycotic.



Types of aneurysms

Aneurysms are named according to their **shape** (configuration) into berry (saccular) or fusiform, or according to their **origin**, into congenital, traumatic or mycotic.

- ***BERRY*** aneurysms are called so because of their resemblance to berries. They are also called sacular and congenital. They range in size from the small to the large. When their size approaches 2.5 cm they are called giant aneurysms. These giant aneurysms produce their signs not through rupture, but like tumors, by pressure on the adjacent structures. Some arteries are known to have such aneurysms like the ophthalmic artery.
- ***FUSIFORM*** aneurysms are the result of atherosclerotic process affecting the vessel wall. They occur mostly in the posterior circulation of the brain, mainly the basilar artery. They do not usually rupture, but send emboli from detaching thrombi, and also through dissection.

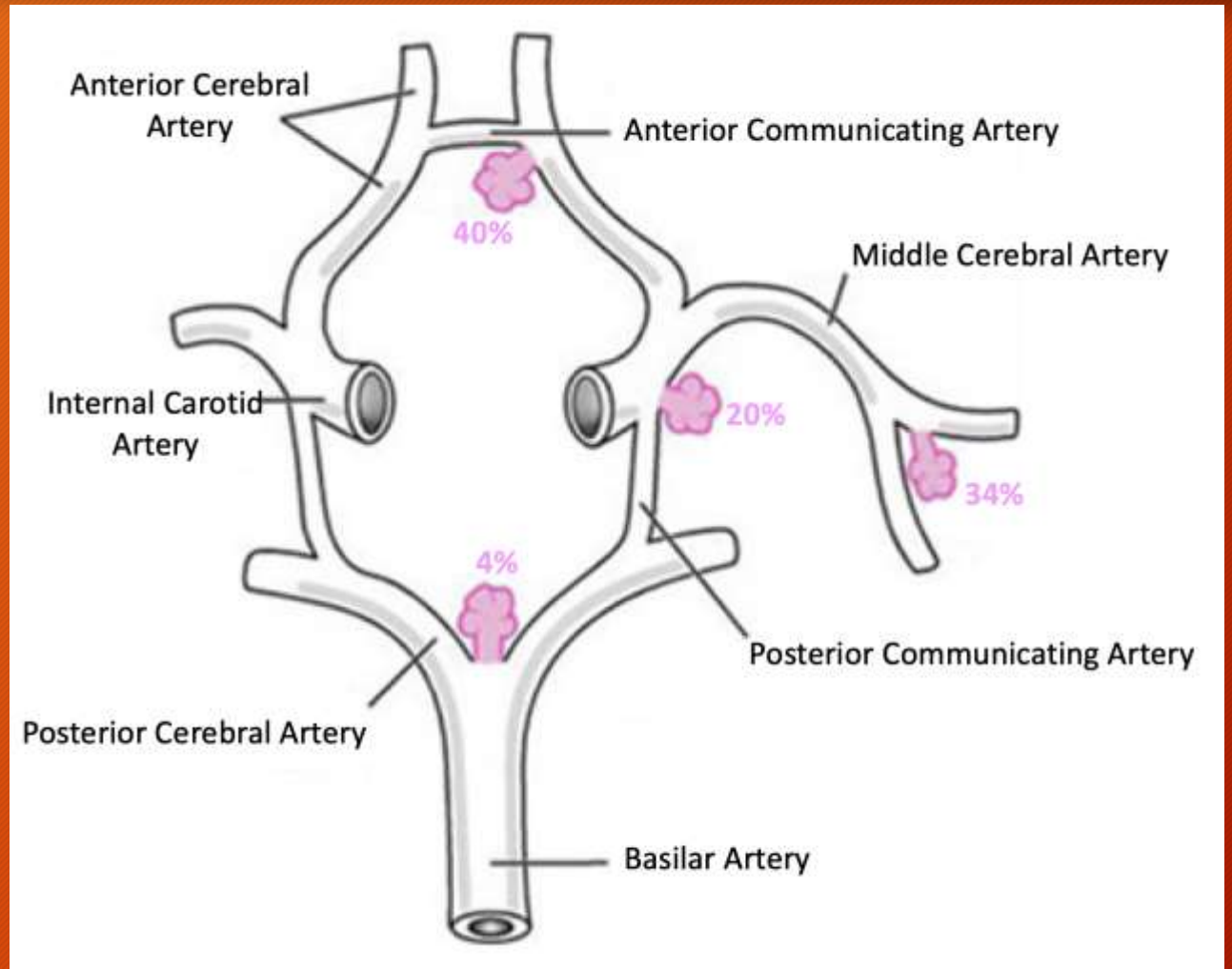
Types of aneurysms

- ***MYCOTIC*** aneurysms result from an infective process, which weakens the wall of a peripheral cerebral vessel when an infected embolus emanating from a condition like bacterial endocarditis lodges in the vessel. Streptococci and staphylococci are blamed. So, they are bacterial in origin and not fungal as the name implies.
- ***TRAUMATIC*** aneurysms are frequently seen in patients who suffered bullet injuries to the brain or in cases of head injuries when there are severe basilar fractures. The traumatized vessel's wall become weak and develops an aneurysm. The internal carotid artery is prone to developing an aneurysm within the cavernous sinus due to basilar trauma. When this aneurysm ruptures it does not produce subarachnoid hemorrhage, but it leads to a carotid cavernous fistula with all its pathognomonic signs and symptoms.

Pathophysiology: Aneurysms

- Aneurysms usually occur at the branching sites on the large cerebral arteries of the circle of Willis.
- The probability of rupture is related to the tension on the aneurysm wall.
- The law of La Place: tension is determined by the radius of the aneurysm and the pressure gradient across the wall of the aneurysm.
- Thus, the rate of rupture is directly related to the size of the aneurysm.
- Aneurysms with >= 5 mm diameter have a 2% risk of rupture, whereas 40% of those 6-10 mm have already ruptured upon diagnosis.

Location of aneurysms on the circle of Willis



Pathophysiology: Aneurysms

- Aneurysms may not rupture and present as a mass pressing on adjacent structures.
- Aneurysms may be multiple in 15% of cases.
- Aneurysms may be found incidentally.
- The incidence of cerebral aneurysms in the population is between 3 and 5%; half of these (50%) will rupture at some time in life

Pathophysiology: AVM's

- Rupture of AVMs can result in both intracerebral hemorrhage and SAH.
- Small AVMs (< 2.5 cm) rupture more frequently than large AVMs (> 5 cm). This till now has no explanation.
- AVM's are graded according to the Spitzler Martin grading system.

Clinical Findings

These include:

- Headache
- Diminished conscious state
- Meningism (neck rigidity, vomiting, photophobia, and fever)
- Seizures.
- Focal neurological signs due to intracerebral hemorrhage, focal pressure by an aneurysm, or vasospasm
- Fundal changes (papilledema, retinal or sub-hyloid hemorrhages)

The Headache

- Severe headache described as one that was never experienced before is a hallmark of SAH.
- Minor blood leakage occurs in 30-50% of aneurysms. These are called sentinel, or "warning," leaks. They precede aneurysm rupture by a few hours to a few months and produce sudden focal or generalized head pain that may be severe.
- Sentinel leaks usually do not occur in the setting of AVM.

Decreased Conscious State

- This occurs in most patients
- It varies widely according to the severity of the hemorrhage
- The conscious state of the patient is considered an important prognostic factor
- A sudden loss of consciousness (LOC) occurs in about 45% of patients as ICP exceeds cerebral perfusion pressure.
- LOC is often transient; however, approximately 10% of patients are comatose for several days, depending on the location of the aneurysm and the amount of bleeding.

Meningism

- Blood in the subarachnoid CSF will lead to features of meningism (neck rigidity, vomiting, photophobia, and fever)
- As blood extends to the lumbar theca, irritation of the cauda equina takes place, leading to sciatica-type pain and LBP
- This occurs in as many as 80% of patients with SAH (but may take several hours to manifest).

Seizures

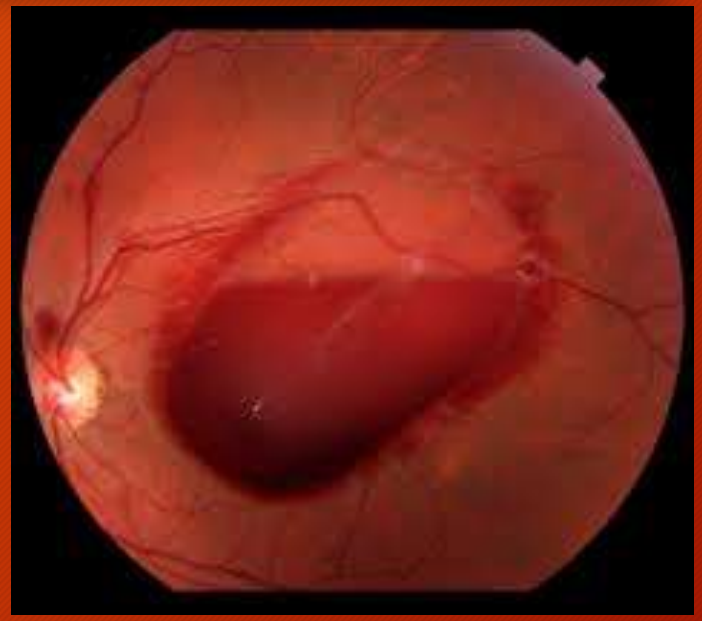
- Seizures during the acute phase of SAH occur in 10-25% of patients.
- No correlation exists between seizure focus and the anatomical site of aneurysm rupture.

Neurological Defects

- AVM's usually rupture within the brain parenchyma leading to associated intracerebral bleeding
- Cerebral vasospasms cause late focal neurological signs, usually manifesting 4-5 days after the hemorrhage

Optic Fundi

- Mild papilledema occurs within a few days due to increased ICP from cerebral edema or hydrocephalus
- Severe SAH → retinal hemorrhages
- Large retinal hemorrhages (sub hyaloid hemorrhages) may lead to permanent visual loss due to vitreous detachment



Clinical Assessment: History

- The diagnosis of a SAH can be sufficiently determined through the history alone from the patient, family, or a friend.
- The classical triad of SAH:
  - Sudden severe headache
  - Meningism
  - Decreased conscious state
- The condition could be confused with migraine or tension headache

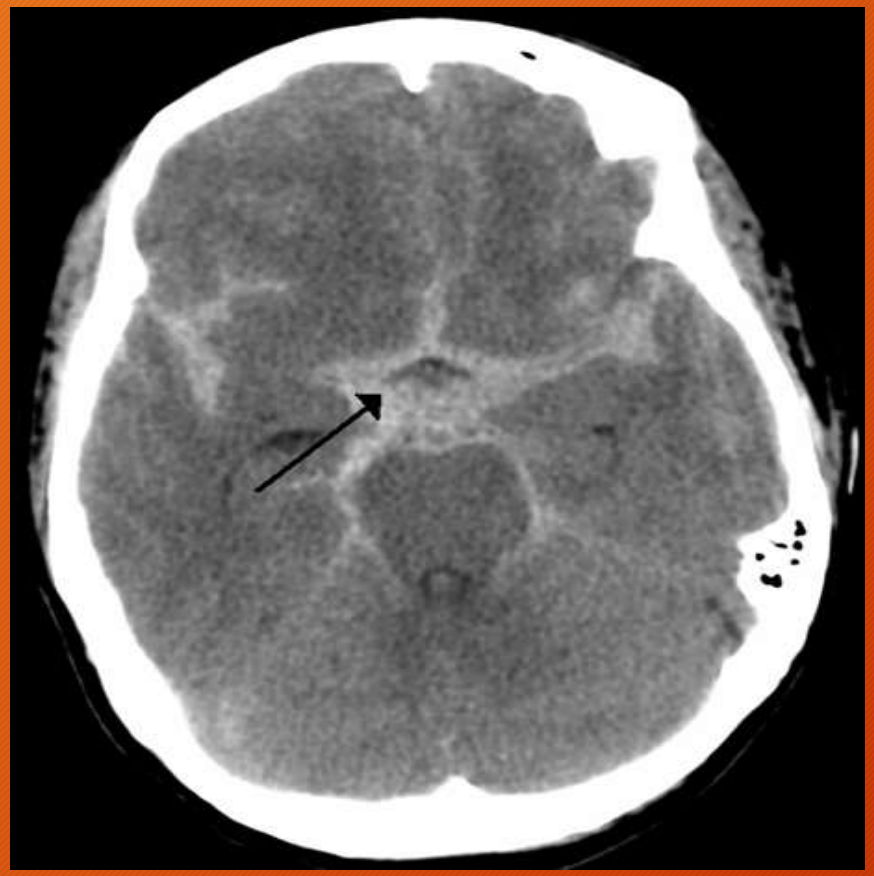
Clinical Assessment: Physical examination

- A full neurological examination should be performed
- Attention should be mostly given to:
  - Presence of neck rigidity
  - Altered conscious state by estimation of the GCS
  - Pupillary status
  - Fundal hemorrhage
  - The presence of neurological deficits

Diagnosis

- Clinical suspicion
- Non contrasted CT
- Lumbar puncture

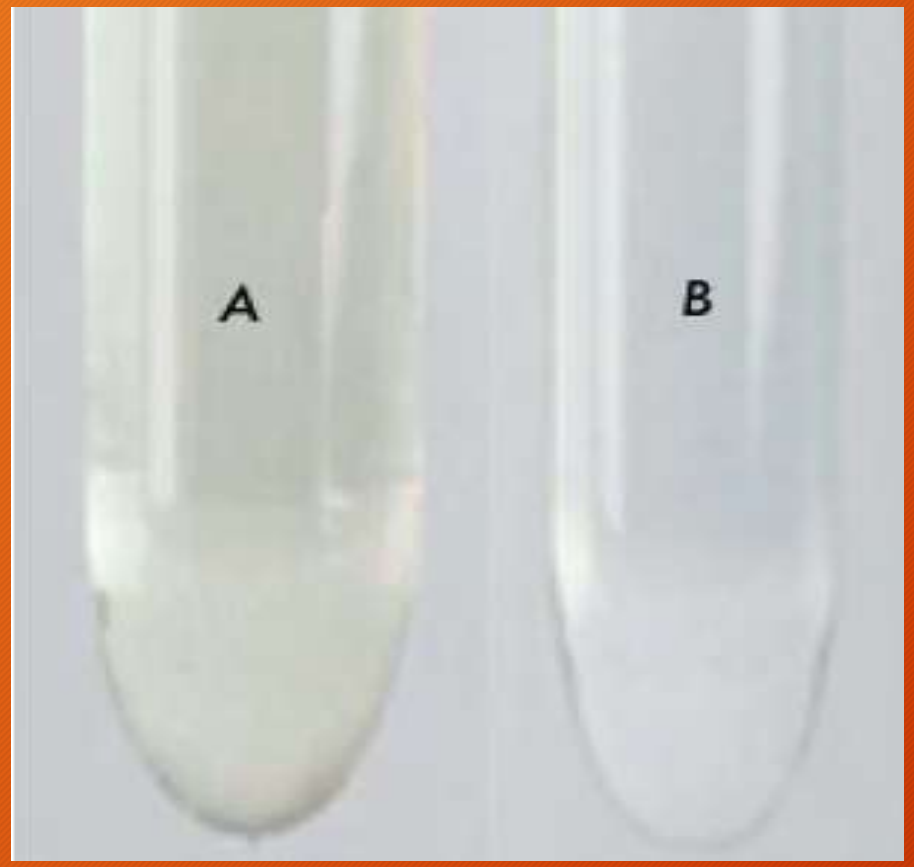
Diagnosis



NON CONTRASTED CT

The accuracy of CT in detecting recent SAH is high approaching 95%, however as time passes by this accuracy declines. So perhaps after 72 hours the CT will not be able to show any blood unless the original bleed was very big and or was associated with intracerebral hematomas

DIAGNOSIS



LUMBAR PUNCTURE

- If the story is correct, the clinical suspicion will remain. And if the CT is negative (as in late presentation or in certain case of anemia), then a lumbar puncture (LP) must be performed.
- LPs will start to be positive i.e. show blood in the CSF 4 hours after the SAH and evidence of the bleed will remain in the CSF for 14 days in the form of blood mixed with CSF, and later as xanthochromia

Grading Systems

- Clinical assessment of SAH severity commonly utilizes grading scales.
- The 2 clinical scales most often used:
  - **Hunt and Hess** grading system
  - **World Federation of Neurological Surgeons** (WFNS) grading systems.
- A third, the **Fischer scale**, classifies SAH based on CT scan appearance and the quantity of subarachnoid blood.

The Hunt and Hess grading system

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

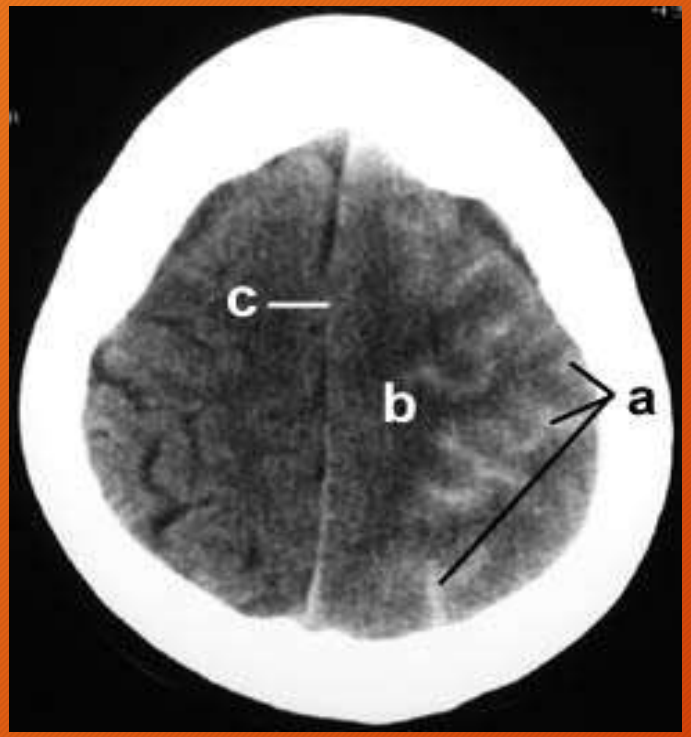
The WFNS grading system

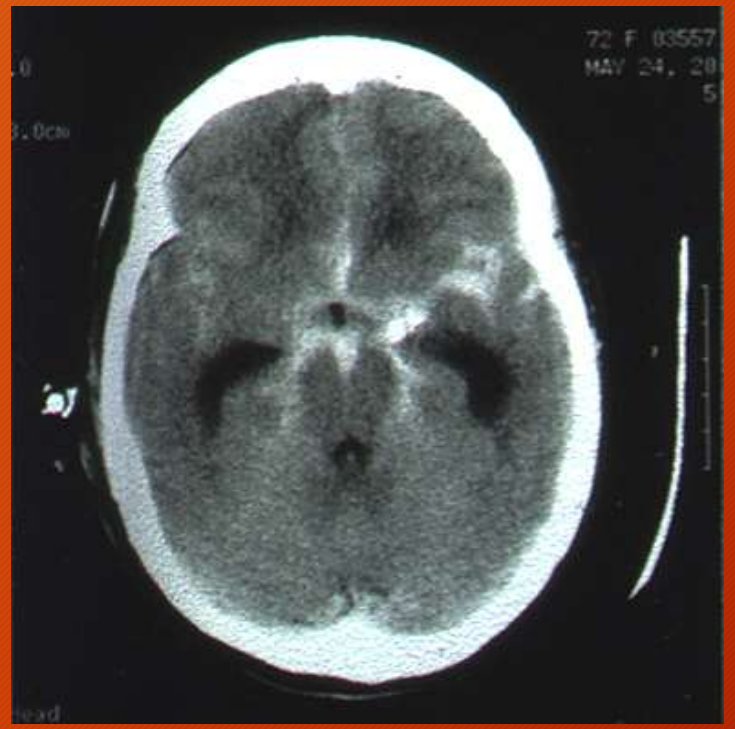
	GCS	MOTOR
Grade 1	15	No motor deficits
Grade 2	13-14	Without deficit
Grade 3	13-14	With focal neurological deficit
Grade 4	7-12	With or without deficit
Grade 5	3-6	With or without deficit

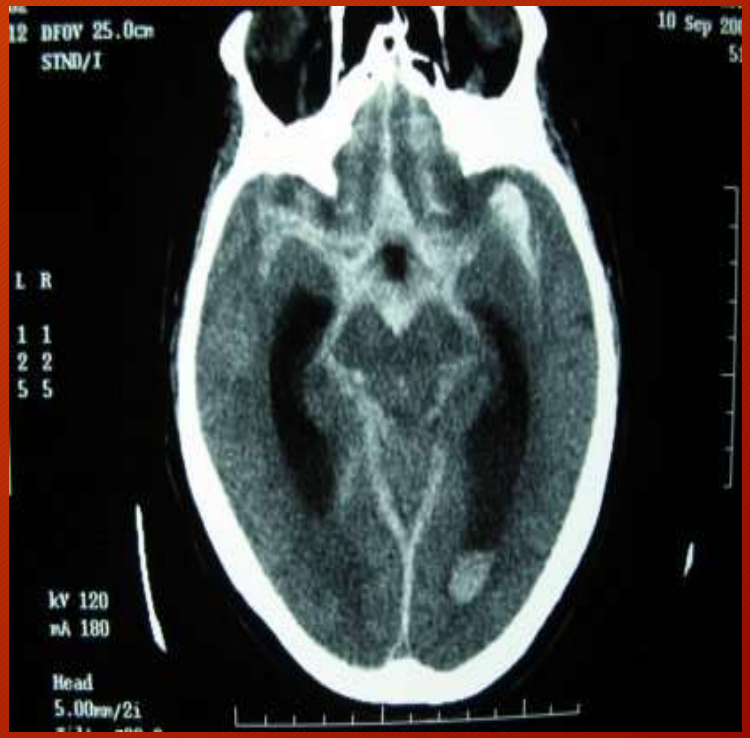
The Fischer scale

GRADE	SUBARACHNOID BLOOD
Grade 1	No subarachnoid (SAH) or intraventricular hemorrhage (IVH) detected
Grade 2	Diffuse thin (<1 mm) SAH. No clots
Grade 3	Localized clots and/or layers of blood >1 mm in thickness. No IVH.
Grade 4	Diffuse or no SAH. ICH or IVH present

EXAMPLES OF SEVERITY OF SAH







Grading Systems

- The Hunt and Hess and the WFNS grading systems have been shown to correlate well with the patient's prognosis.
- The Fischer classification has been used successfully to predict the likelihood of symptomatic cerebral vasospasm, the most feared complications of SAH.
- All 3 grading systems are useful in determining the indications for and timing of surgical management.

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

Patient of grades 1-3 should be placed in a dark lit room to counteract the photophobia; they should be nursed on a bed with the head elevated 30 degrees. 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.

MANAGEMENT

Blood should be withdrawn for CBC, electrolytes, grouping and cross matching of blood. PT and PTT should also be checked to rule out blood dyscrasias. Cardiac enzymes should be estimated as well as ABGs. A chest x-ray should be ordered. Cardiac arrhythmias and pulmonary edema tend to occur accompanying SAH due to sympathetic surge.

MANAGEMENT

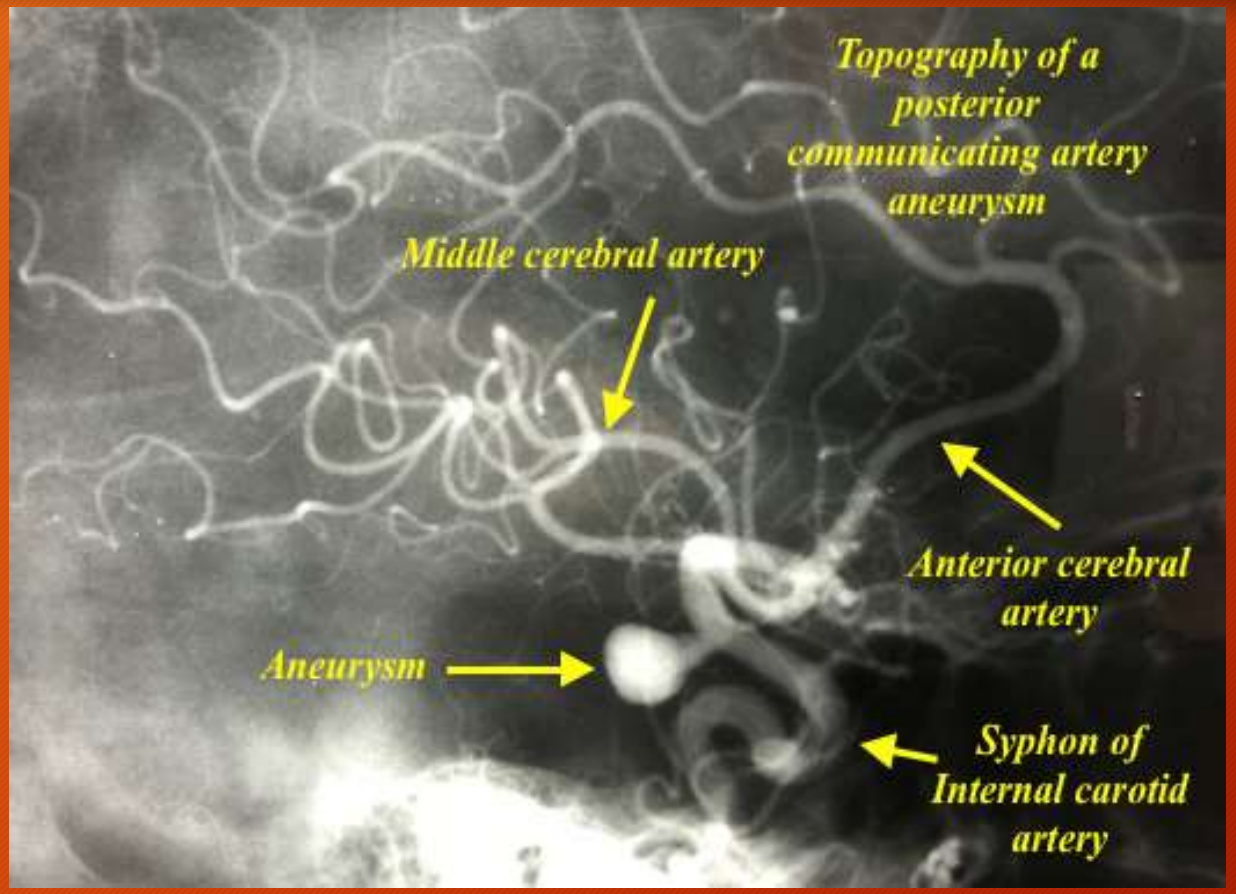
The following medications should be administered to the patient, in addition to the medication mentioned previously:

- Phenytoin 100 mg three times daily.
- Nimodipine, which is a calcium channel blocker in the dose of 60 mg orally every 4 hours. Nimodipine will help protect the brain against ischemia. It does not prevent vasospasm.
- Some centers still administer Cyclokapron (Tranexamic Acid); which is an antifibrinolytic agent, to help prevent the dissolution of the clot protecting the ruptured aneurysm by the normal potent fibrinolytic activity of the CSF . Some stopped using the medication for fear of some serious side effects like venous sinus thrombosis.

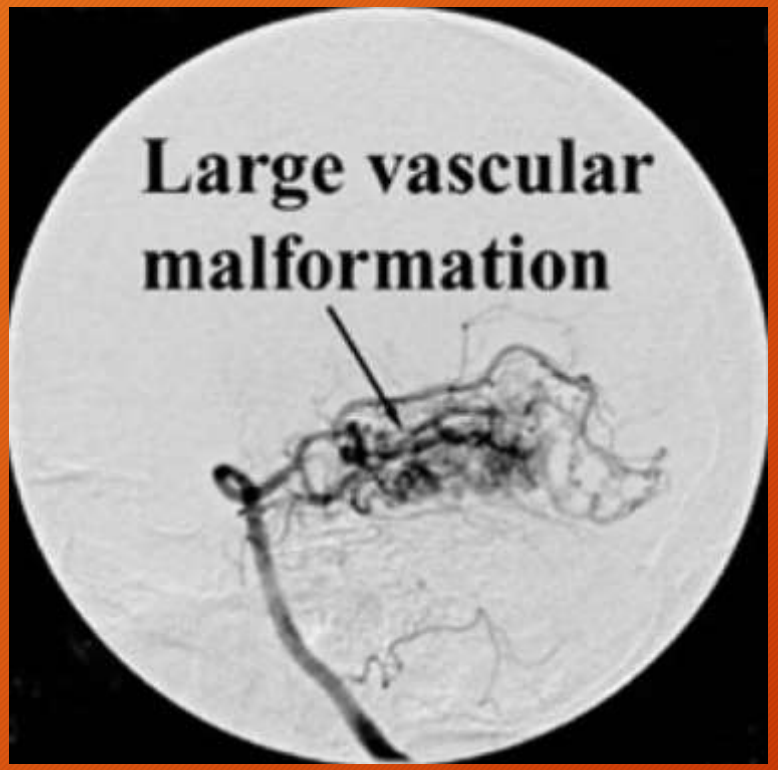
FINDING THE CAUSE: Cerebral Angiography

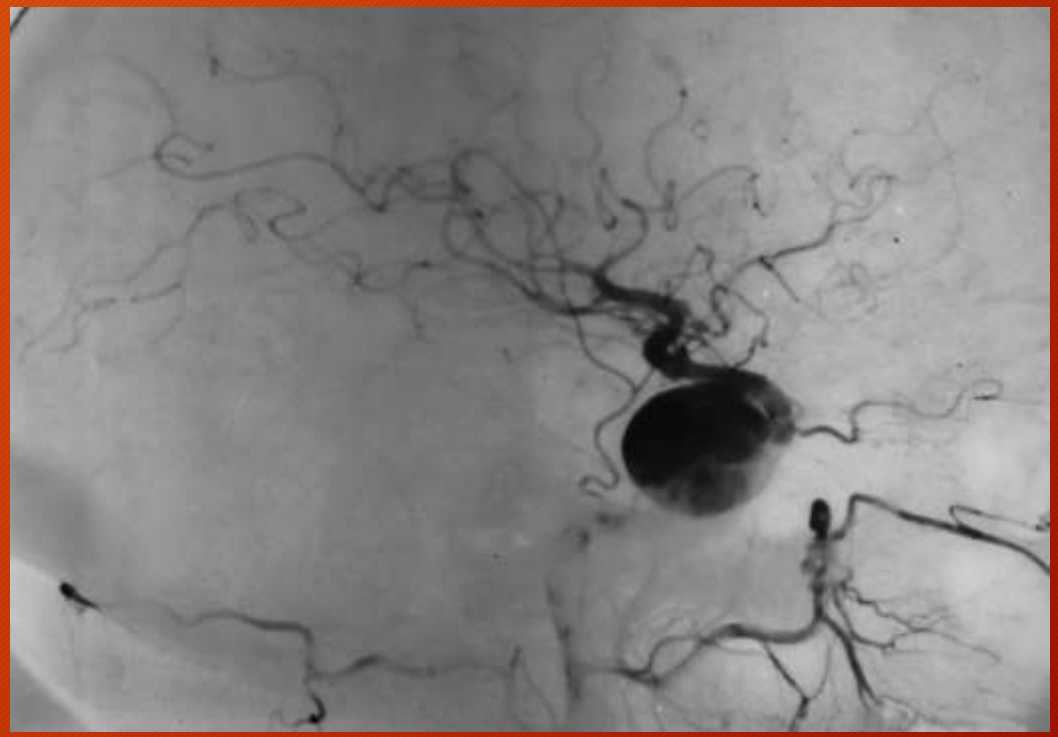
- Cerebral angiography (DSA) is the mainstay of management
- It can provide important information in the setting of SAH:
  - Cerebrovascular anatomy
  - Aneurysm location and source of bleeding
  - Aneurysm size and shape, as well as orientation of the aneurysm dome and neck
  - Presence of multiple or mirror aneurysms (identically placed aneurysms in both the left and right circulations)
- CT angiography or MRA can be performed as well

Standard angiography

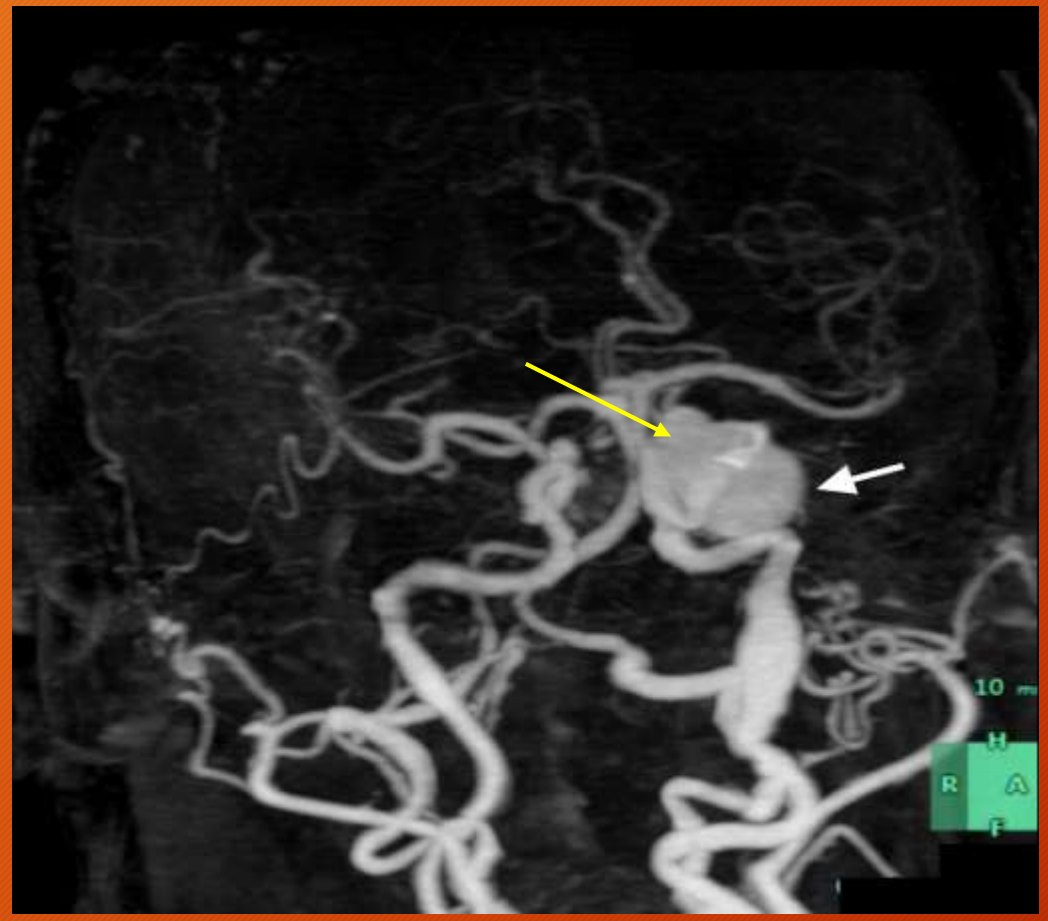


Digital subtraction angiography (DSA)



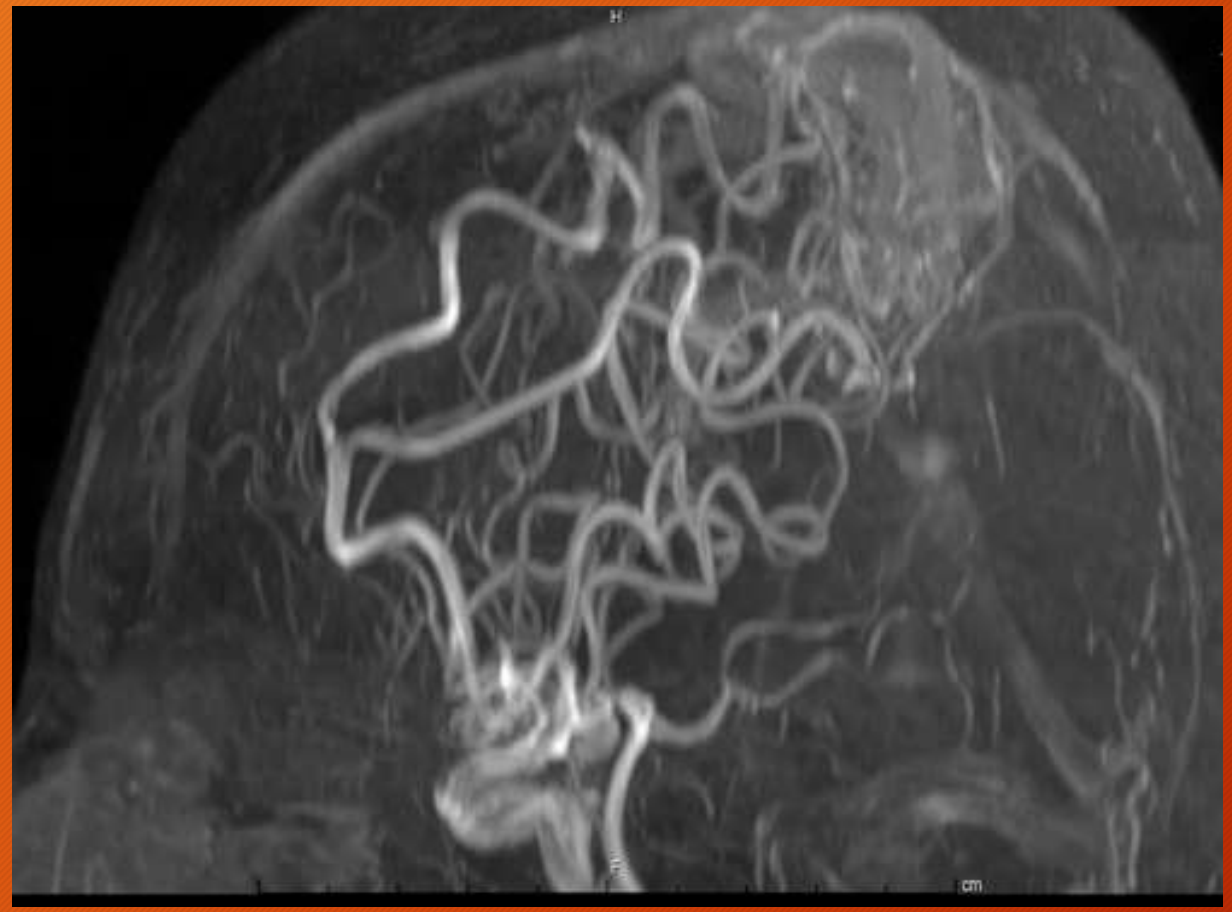


CT Angiography (CTA)



ANTERIOR COMMUNICATING ARTERY ANEURTYSM (AComA)

Magnetic resonance Arteriography (MRA)



- 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.

Surgery or Endovascular Treatment ?

Once the aneurysm was demonstrated by DSA then the mode of management decided.

1) Craniotomy and clipping.
2) Endovascular obliteration.

Controversy remains abound in this area especially after the introduction of endovascular management of intracranial aneurysms.

Early or Delayed Surgery?

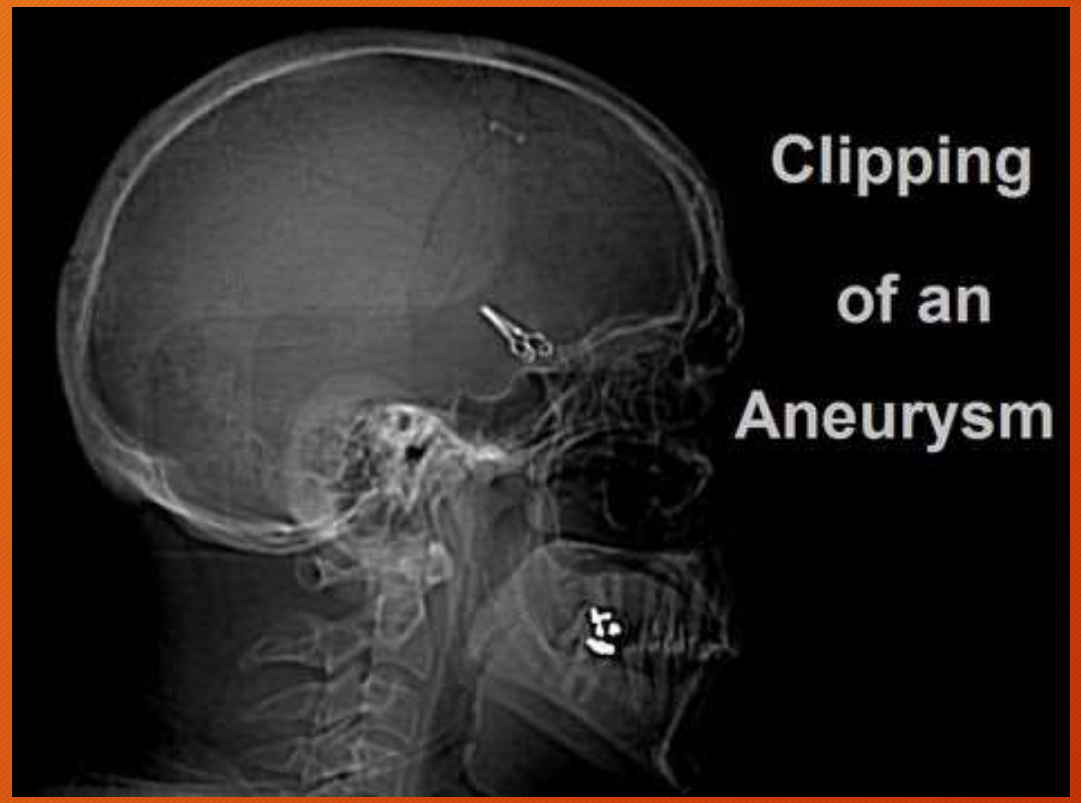
Neurosurgeons continue to debate, the timing of surgery: early or late? And the type of surgery: clipping or endovascular? Those in favor of early surgery cite the prevention of the lethal rebleeding which occurs in as many as 15% of patients during their stay in hospital, with a mortality rate of 80%, almost double that of the initial bleed (40-50%). While those who advocate late or delayed operation (10-14 days post ictus), cite the brain condition which is not ideal for surgery (high ICP, and edema) and the fragility of the aneurysm itself, which makes it easy to rupture during surgery

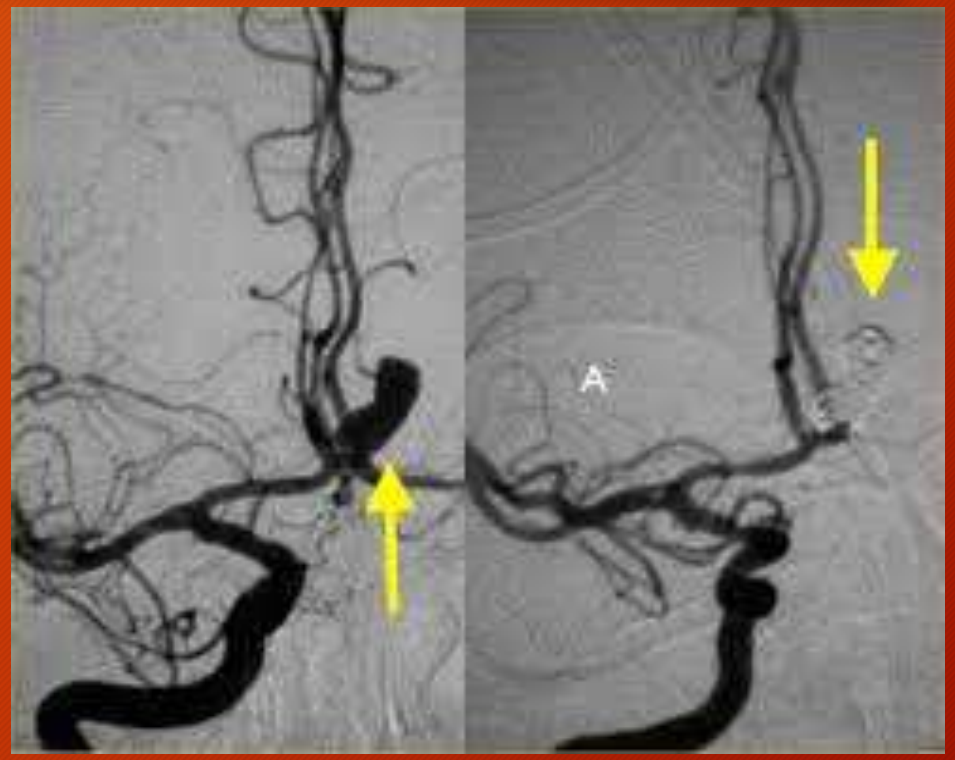
Which type of surgery

There is agreement that patients with SAH grade 1-3 are treated surgically with clips across the aneurysmal neck.

While, endovascular treatment is kept for those in grades 4 and 5. However certain centers prefer endovascular for all aneurysms if so indicated. Not all aneurysm could be treated with coils especially those with wide necks, although recent advances have combined coils with stents to prevent slippage of the coil outside the aneurysmal

Clipping and Endovascular Obliteration





COMPLICATIONS

- REBLEEDING
- VASOSPASM
- HYDROCEPHALUS

Rebleeding

- Rebleeding is the most dreaded early complication of SAH.
- 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%.

Cerebral Vasospasm

- It is the delayed narrowing of the large capacitance vessels at the base of the brain
- Vasospasm is reported to occur in as many as 70% of patients with SAH and is clinically symptomatic in as many as 30% of patients
- Symptoms include decrease in consciousness or focal neurological deficit.
- Most commonly occurs 4-14 days after the hemorrhage
- The terminal internal carotid artery or the proximal portions of the anterior and middle cerebral arteries are most involved
- Vasospasm may be confused with rebleeding; thus angiography should be performed to differentiate them

Hydrocephalus

- ACUTE NON-COMMUNICATING complicates 20% of SAH cases and usually occurs within the first 24 hours
  - It can precipitate life-threatening brainstem compression and occlusion of blood vessels.
  - Patients present with abrupt mental status change, such as lethargy, stupor, or coma.
  - CT scan differentiates hydrocephalus from rebleeding.
- CHRONIC COMMUNICATING caused by scarring of the arachnoid granulations and alterations in CSF absorption, occurs in 10-15% of patients with SAH.
  - Patients may present with incontinence, gait instability, and cognitive deterioration.

OTHER 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

Prognosis

- Studies show that grade 1 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

MULTIPLE INTRACRAINIAL 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 and management. Which aneurysm did bleed, and what to do to those, which did not?
- Neurosurgeons use the characteristics of the aneurysm, its size, its location, the presence or not of a clot within it, the presence of vasospasm around it or its proximity to the origin of the parent vessel, to determine which aneurysm bled. They use data from clinical examination and CT to help in the matter too.
- Once the offending aneurysm is determined, a management plan is formulated. If the plan was surgery then all aneurysms in the same operative field are dealt with. Any others will be dealt with as incidental findings and treated as such (see later). If the plan was coiling or endovascular procedures then perhaps an attempt will be made on all.

INCIDENTAL ANEURYSMS

More and more incidental aneurysms are being detected through the use of CT or MRI performed for different cranial diseases or complaints. Once one is found the question arises, what to do? Leave it alone or treat it? One has to understand the natural history of these aneurysms before deciding on what to do. The most important question is: did this person have a SAH before or not?

In those people who never had a SAH before, the risk of bleeding is 0.05% per year for aneurysms less than 10 mm in diameter, and 1% for those aneurysms' lager than 10mm. While in those people who had SAH before, the percentages for aneurysms less than 10mm was 0.5% (10 times higher). In this group size was not what mattered but the location of the aneurysm. Those in the posterior circulation were more likely to rupture, as indicated by the international study of unruptured intracranial aneurysms conducted in 2005 (ISUIA) .

Nowadays neurosurgeons suggest that aneurysms less than 10mm with no previous history of SAH should be observed while those above 10 mm or with daughter cysts should be treated[)].. The same debate about treatment modalities still goes on, surgical clipping or coiling. The international subarachnoid aneurysms trial (ISAT) [(]conducted also in 2005, was a little in favor of surgical clipping. Many, dispute these results and recommendations

INTRACRANIAL ARTERIO-VENOUS MALFORMATIONS

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.

INTRACRANIAL ARTERIO-VENOUS MALFORMATIONS

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-VENOUS MALFORMATIONS

The presentation of AVMs can be one of four modes;

- SAH as described earlier in the chapter (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-VENOUS MALFORMATIONS

- 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 6^{th} 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 excision, or embolization or radiotherapy by the Gamma Knife or a combination of any depending on grading system