CNS pathology

Dr Heyam Awad

Lecture 2: traumatic and non traumatic brain hemorrhage

- This is an online lecture.

Youtube link:

- https://www.youtube.com/watch?v=feVqml3Gmms

Intracranial hemorrhage

- When a patient shows up with parenchymal hemorrhage, and has no history of trauma, it is most important to ask about the status of hypertension.
- Since it usually shows on hypertensives, it typically presents at a later age (60s), and shows up with symptoms of increased ICP (headache, confusion, vomiting), as well as neurological deficit.

" He can't stand the sight of blood. "

Causes of intracranial hemorrhage

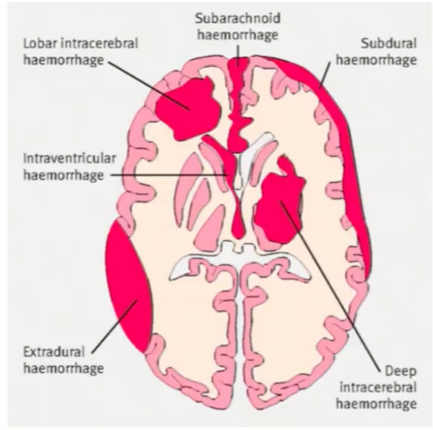
Intracranial hemorrhage can be traumatic or non traumatic.

Causes of non traumatic hemorrhage:

- 1. Primary brain parenchymal hemorrhage , which is caused mainly by hypertension.
- 2. Cerebral amyloid angiopathy =الداء النشواني
- 3. Ruptured aneurysmsتمدد كيسي دموي =
- 4. Vascular malformation
- 5. Vasculitis

Other (rarer) causes of intra-cerebral hemorrhage

- Bleeding disorders
- Drug related: anti-coagulants
- Cocaine use
- Tumors.. Can encroach on a vessel and cause bleeding



- Extradural and subdural hemorrhages are mostly caused by trauma.
- Subarachnoid and intracerebral can be traumatic or non-traumatic.

Most common cause of non-traumatic brain injury.

1. Primary brain parenchymal haemorrhage

- Primary = spontaneous = non-traumatic.
-Peak 60 years of age.
-Mostly due to rupture of a small intra-parenchymal vessel.
-Hypertension is the leading cause.
- Most affected sites: basal ganglia, thalamus, pons and cerebellum.
- Outcome depends of the site and extent of haemorrhage

Why hypertension causes parenchymal hemorrhage ?

- Hypertension causes hyaline arteriolosclerosis.
- This results in weak arterioles, so the arterioles can rupture especially if there is sudden or sustained increase in blood pressure.

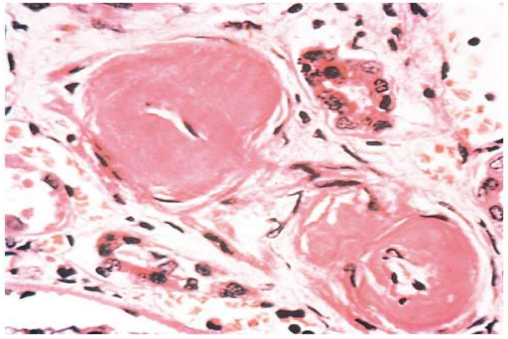
- Minute aneurysms can form (Charcot- Bouchard micro aneurysms) because of the weak vascular walls and these also can rupture.

Hypertension in the small blood vessels also leads to vessel weakness and aneurysms.

Hyaline arteriolosclerosis

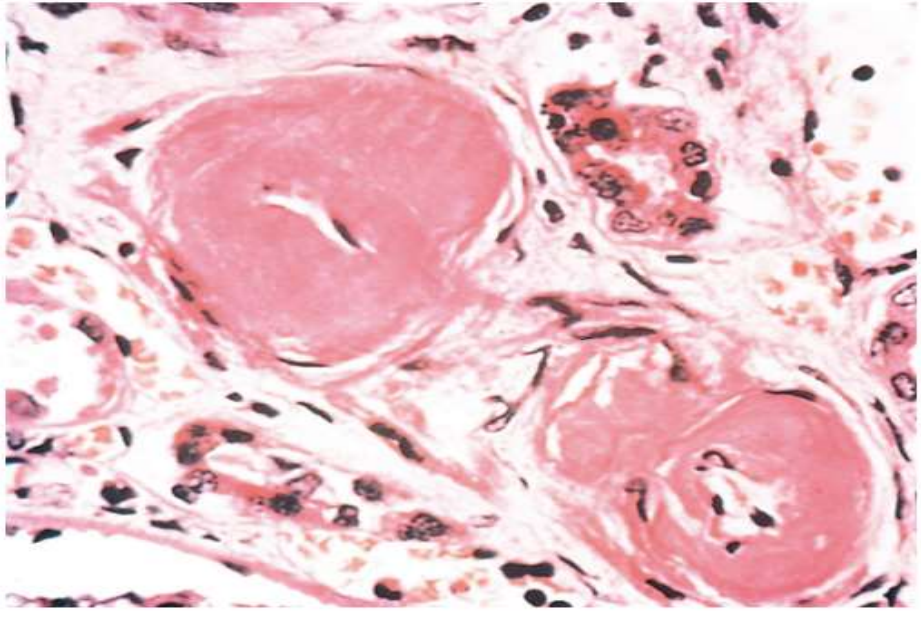
- Homogeneous pink hyaline thickening of the arteriolar walls with luminal narrowing and loss of underlying structural detail.
- Occurs due to leakage of plasma components across injured endothelial cells into vessel walls and increased extracellular matrix production by smooth muscle in response to chronic hemodynamic stress.

Hyaline arteriolosclerosis



Note the thick walls that contain hyaline, pink, material.

Hyaline arteriolosclerosis



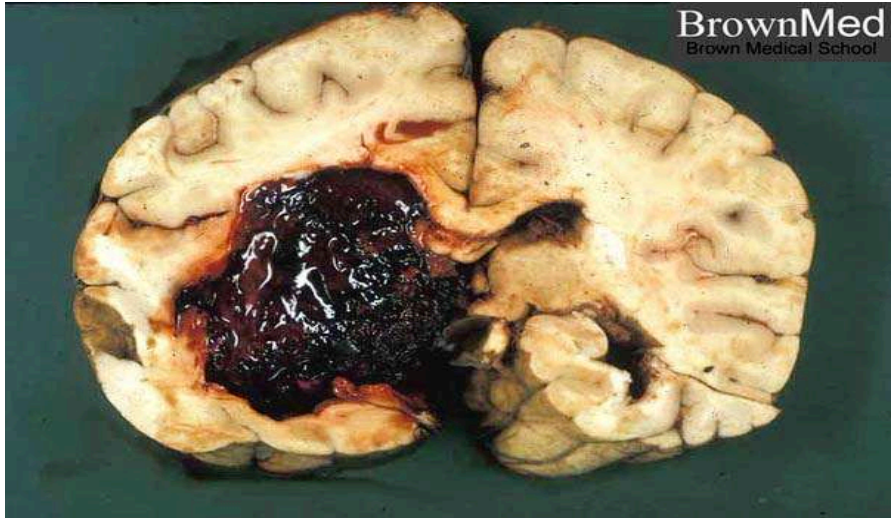
Symptoms of parenchymal brain haemorrhage

- 1. neurological symptoms related to the area affected
- 2. symptoms of increased intracranial pressure

morphology

- Extravagated blood.
- With time.. Resolution and cavity formation

Brain haemorrhage



If blood is absorbed, it will become a cavity.

Cavity.. Old infarct or old hemorrhage; both will end up with a cavity!

A cavity lined by gliosis.

Ischemic stroke, whether infarction or hemorrhage, will lead to the same result in the chronic stages, where:

- There'll be reabsorption of the infarction tissue.
- Nothing will fill up those spaces, ending up in a cavity.
- Gliosis will line this cavity.

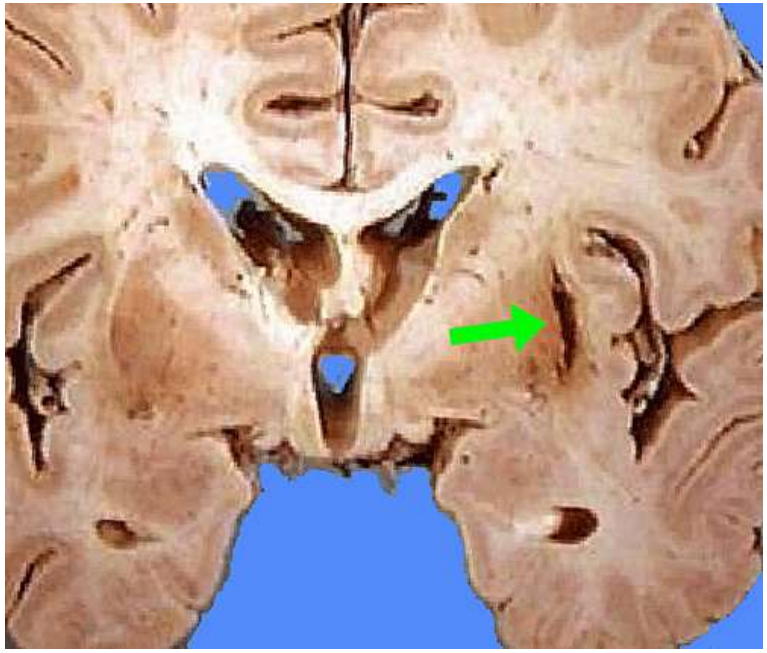
Hypertension/ effects of hypertension on the brain:

- Massive intracranial haemorrhage.
- Lacunar infarcts.
- Rupture of small penetrating vessels
- Acute hypertensive encephalopathy= edema When diastolic pressure exceeds 130 mmHg.

- *Lacunes* or *lacunar infarcts* are small cavitary infarcts, just a few millimeters in size, that are found most commonly in the deep gray matter (basal ganglia and thalamus), the internal capsule, the deep white matter, and the pons. They are caused by occlusion of a single penetrating branch of a large cerebral artery. Depending on their location, lacunes can be silent clinically or cause significant neurologic impairment.
- *Rupture of the small-caliber penetrating vessels* may occur, leading to the development of small hemorrhages. In time, these hemorrhages resorb, leaving behind a slitlike cavity *(slit hemorrhage)* surrounded by brownish discoloration.

Vessel rupture

- Small penetrating vessels may rupture.
- Cause small haemorrhages = slit haemorrhages.

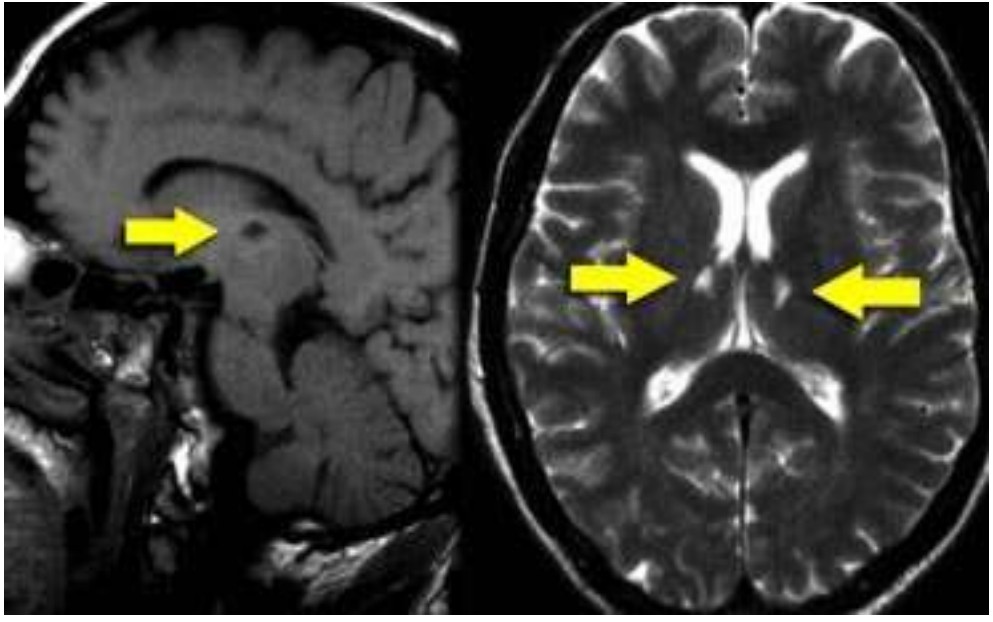


Lacunar infarcts

- Small infarcts, mostly in deep grey matter (basal ganglia and thalamus), internal capsule, deep white matter and pons.
- Caused by occlusion of penetrating branches of a large cerebral artery.
- Effect: depends on site

Lacunar infarct

Caused by narrowed small vessels, as a result of chronic hypertension leading to hyaline arteriolosclerosis in these small penetrating arteries, and thus get blocked and leads to infarct.



2. vasculitis

- = inflammation of the blood vessel wall
- Inflammation weakens the vessel wall so it can rupture and cause hemorrhage.

Causes of vasculitis

Infectious arteritis:

- previously seen with syphilis and TB.
- Now in association with: CMV, herpes, aspergillosis….. immunosuppression HIV, Transplantation, Chemotherapy.

systemic

Polyarteritis nodosa.

localized

Primary angiitis of CNS cause diffuse encephalopathy with cognitive dysfunction.

3. Cerebral amyloid angiopathy

- Amyloid deposition in the walls of arteries
- Causes weakness in vessel wall
- Bleeding , usually in the lobes of cerebral cortex (lobar hemorrhage)

That is, hemorrhage near the surface.
As opposed to deep hemorrhage in pons, basal ganglia, thalami, cerebellum that occur in hypertensive cerebrovascular accidents.

Amyloidosis

- Deposition of extracellular fibrillary proteins
- These abnormal fibrils are produced by the aggregation of misfolded proteins (which are soluble in their normal folded configuration).

Cerebral Amyloid Angiopathy

Cerebral amyloid angiopathy (CAA) is a disease in which the same amyloidogenic peptides as those found in Alzheimer disease (discussed later) deposit in the walls of medium- and small-caliber meningeal and cortical vessels. The amyloid confers a rigid, pipe-like appearance

And doc said "different chemical properties, same physical properties."

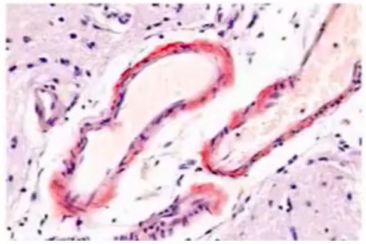
- Amyloid is deposited in the <u>extracellular space in various tissues and organs of the body</u>

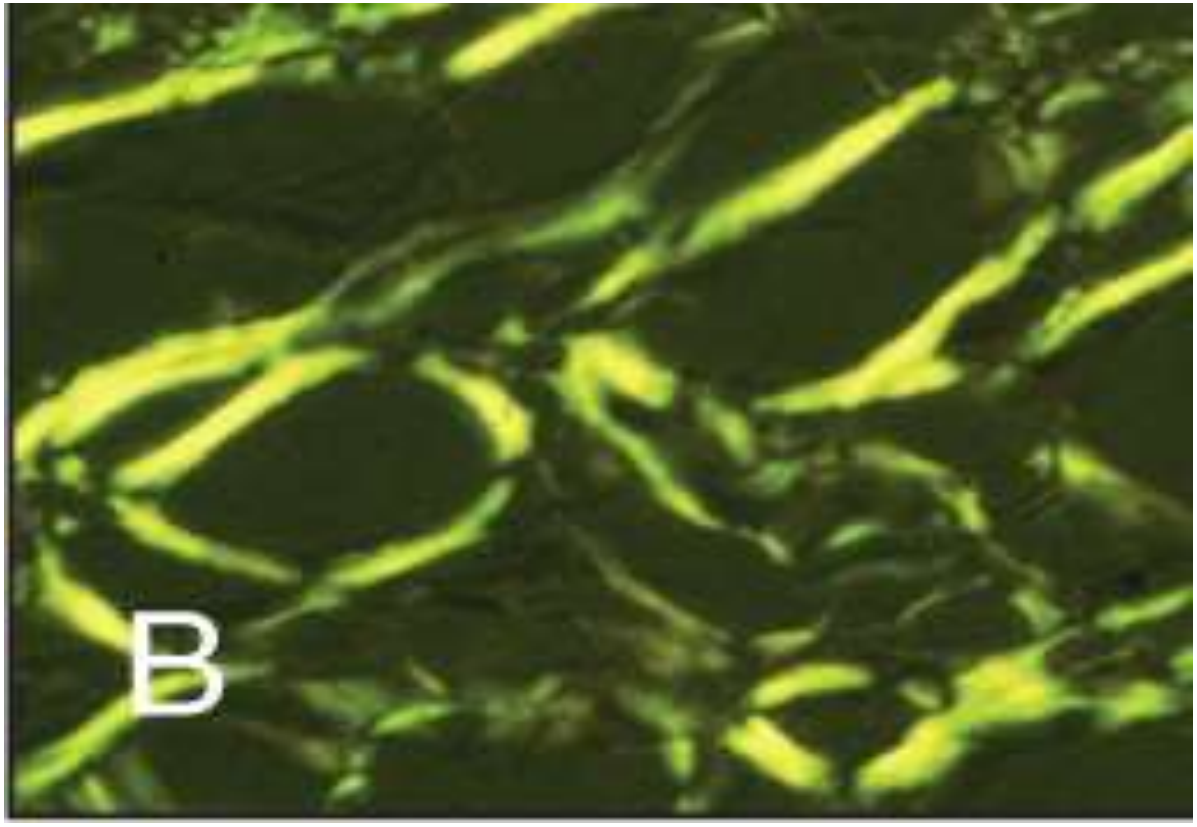
- These fibrillary proteins are responsible for <u>tissue damage and functional compromise</u>

People at risk of Amyloid Angiopathy are ones who have chronic inflammatory diseases, like RA, SLE, Osteoarthritis. Chronic inflammation increases acute phase proteins, thus increases the likelihood of getting a misfolded serum amyloid protein, which if continually synthesized will cause extracellular amyloid deposits, causing amyloid angiopathy.

Doctor here is talking about systemic amyloidosis, which is a similar disease but involves a different mechanism compared to CAA

Congo red stain





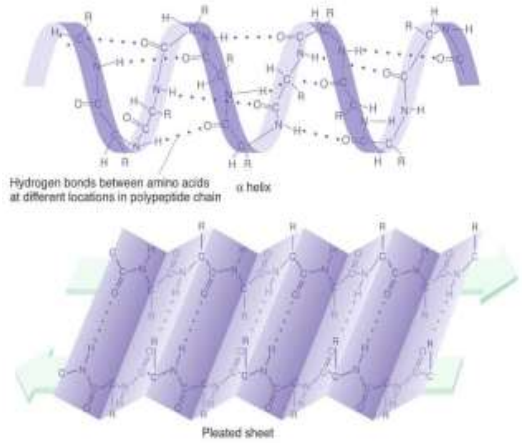
Serum Amyloid Protein

Congo red stain with fluorescent microscope, gives apple green color, indicates amyloidosis, used when doing autopsy.

By electron microscope

- All types of amyloid consist of continuous, non-branching fibrils with a diameter of approximately 7.5 to 10 nm. With a cross-β-pleated sheet conformation

Critical to its ability to aggregate and accumulate on the walls of vessels.



4. Ruptured berry aneurysm

- Rupture happens usually due to increased intracranial pressure.
- Sudden severe headache followed by loss of consciousness
- 25-50% die
- Survivors: risk of recurrent bleeding

Subarachnoid Hemorrhage and Saccular Aneurysms

The most frequent cause of clinically significant nontraumatic subarachnoid hemorrhage is rupture of a saccular (berry) aneurysm. Hemorrhage into the subarachnoid space may also result from vascular malformation, trauma, rupture of an intracerebral hemorrhage into the ventricular system, coagulopathies, and tumors. Epidural and subdural hemorrhages are typically secondary to trauma and are discussed later.

In about one-third of cases, rupture of a saccular aneurysm occurs because of an acute increase in intracranial pressure, such as occurs with straining at stool or sexual orgasm. Blood under arterial pressure

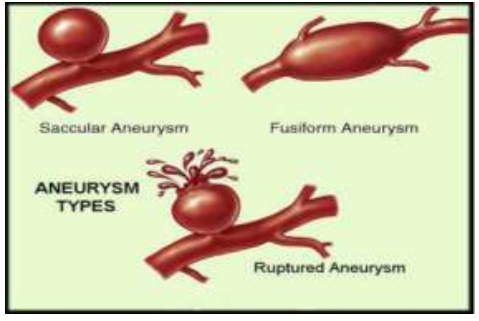
Ruptured aneurysm

- Mainly causes subarachnoid hemorrhage but also can cause hemorrhage within the brain paranchyma.

Subarachnoid haemorrhage

- Most common cause: ruptured berry aneurysm.
- Other causes: vascular malformations, trauma, tumours, haematological disturbances.

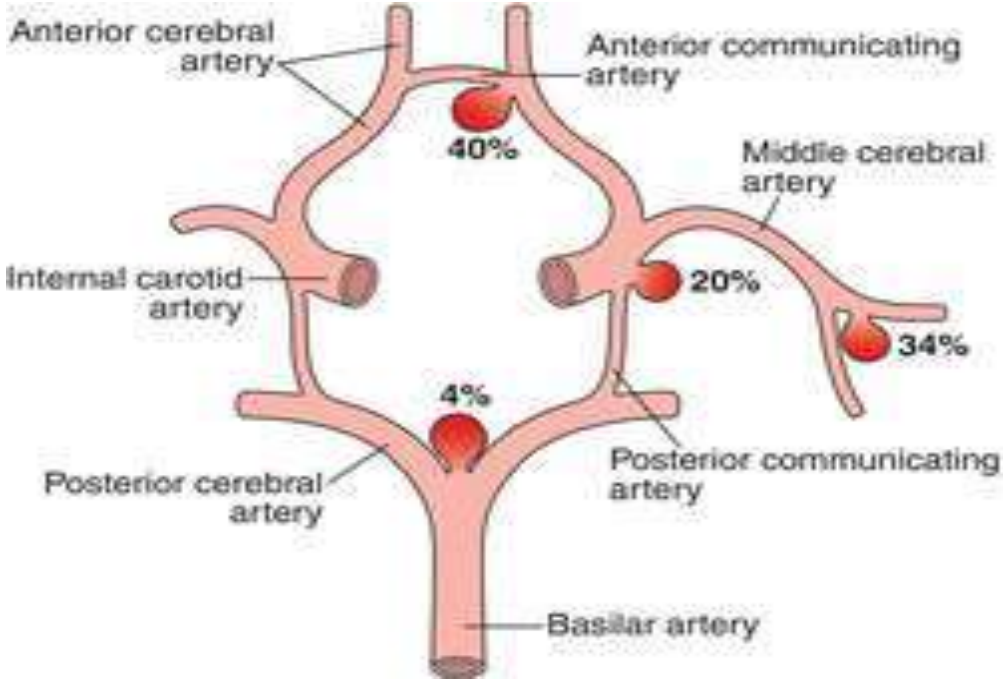
Subarachnoid haemorrhage
Ruptured berry (secular) aneurysm is the most common cause



Berry aneurysm

- 90% in the anterior circulation
- Near major arterial branching points
- Multiple in 20 – 30 % of cases

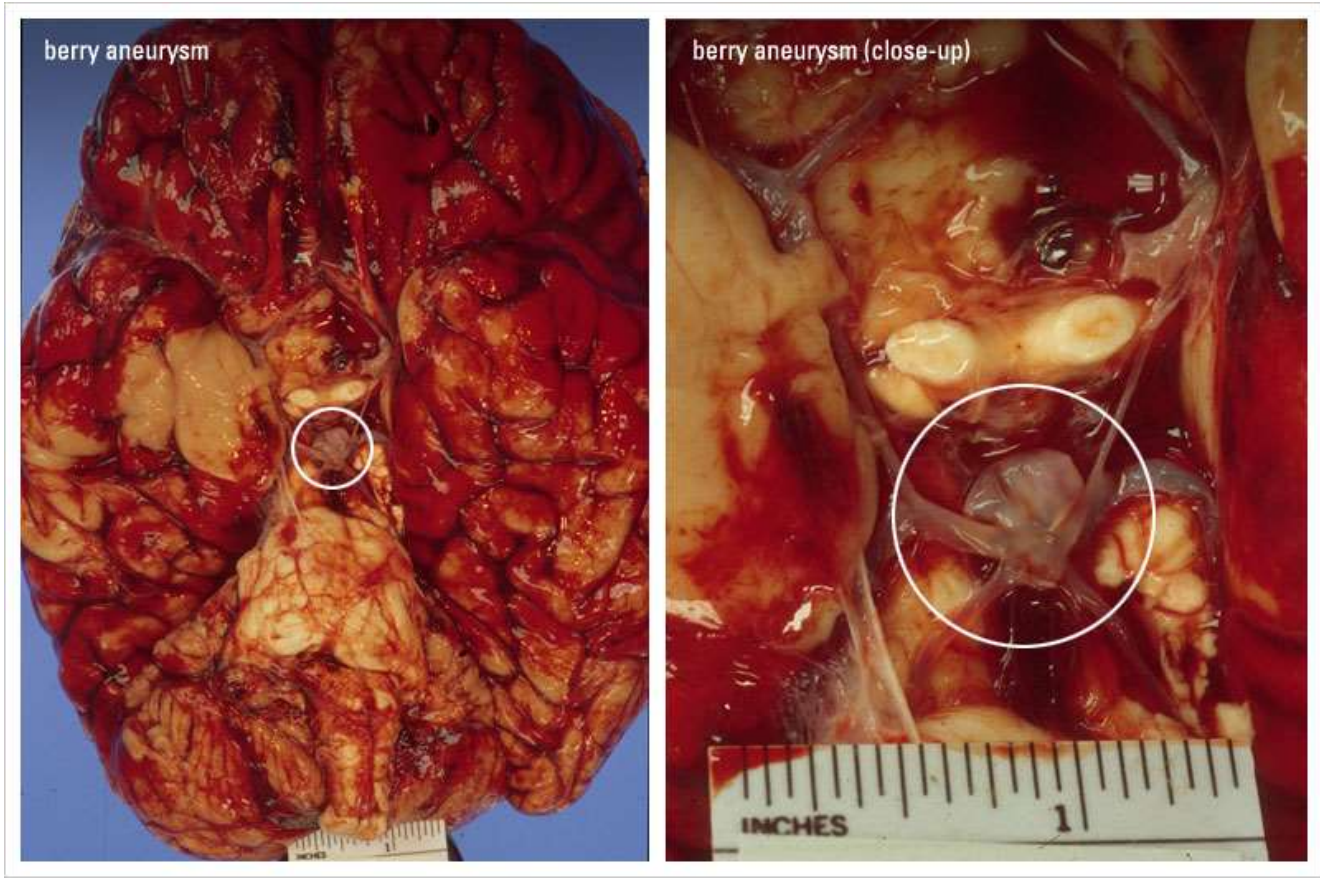
Thus, if the patient survives a first episode, there's a good chance there'll be a second episode.


Anterior cerebral artery
Anterior communicating artery
40%
Middle cerebral artery
Internal carotid artery
20%
34%
4%
Posterior communicating artery
Posterior cerebral artery
Basilar artery

Morphology

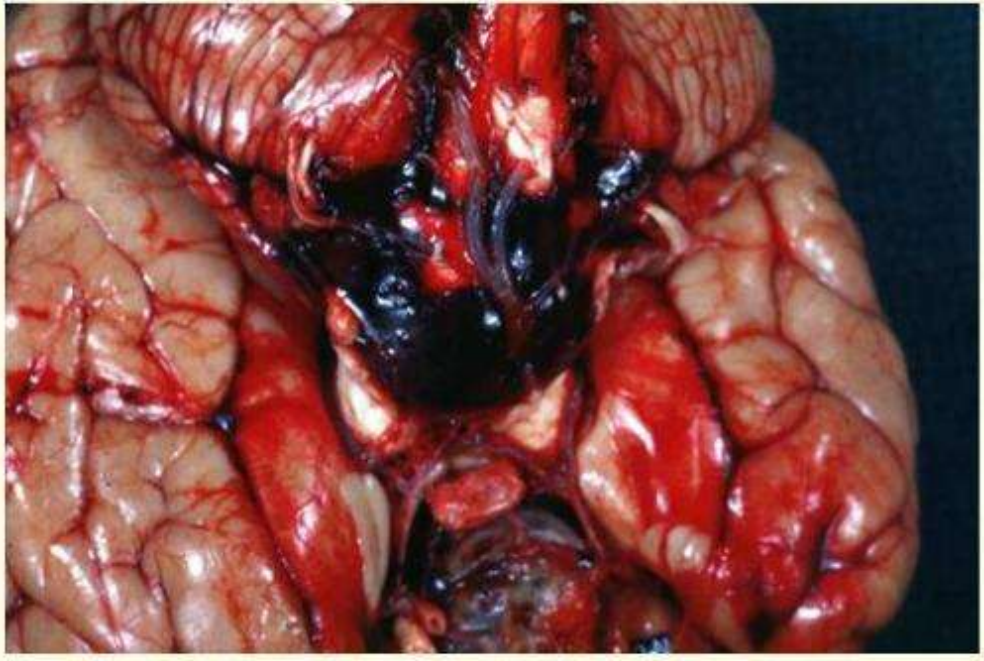
- Berry aneurysm: thin walled outpouching of an artery

- Very severe headache.
- Vomiting.
- 25% die in the first episode.



Sac

Subarachnoid Hemorrhage



*** Restricted use. PEIR; University of Alabama at Birmingham, Department of Pathology**

(c) 2007, Michael A. Kahn, DDS/Lynn W. Solomon, DDS

Sac blew up

5. Vascular malformations

- Arteriovenous malformations
- Cavernous malformations
- Capillary telengectasia
- Venous angioma

AV malformation

- Most common type of vascular malformation
- Males more than females
- Present at 10-30 years of age
- Symptoms: seizures and intracranial hemorrhage

Morphology of AV malformation

- Network of disorganised vascular channels

Disorganized thick and thin vessels.

MORPHOLOGY

Arteriovenous malformations (tangled networks of wormlike vascular channels with high blood flow due to prominent, pulsatile arteriovenous shunting) may involve vessels in the subarachnoid space, the brain, or both (Fig. 28.21). They are composed of greatly enlarged blood vessels separated by gliotic tissue, often with evidence of prior hemorrhage. Some vessels can be recognized as arteries with duplication and fragmentation of the internal elastic lamina, while others show marked thickening or partial replacement of the media by hyalinized connective tissue.

Traumatic lesions

- Trauma to CNS causes mortality or disability
- Outcome depends on extent of trauma and site affected.
- Spinal cord trauma.. can cause severe disability.
- Brain stem trauma… can be fatal

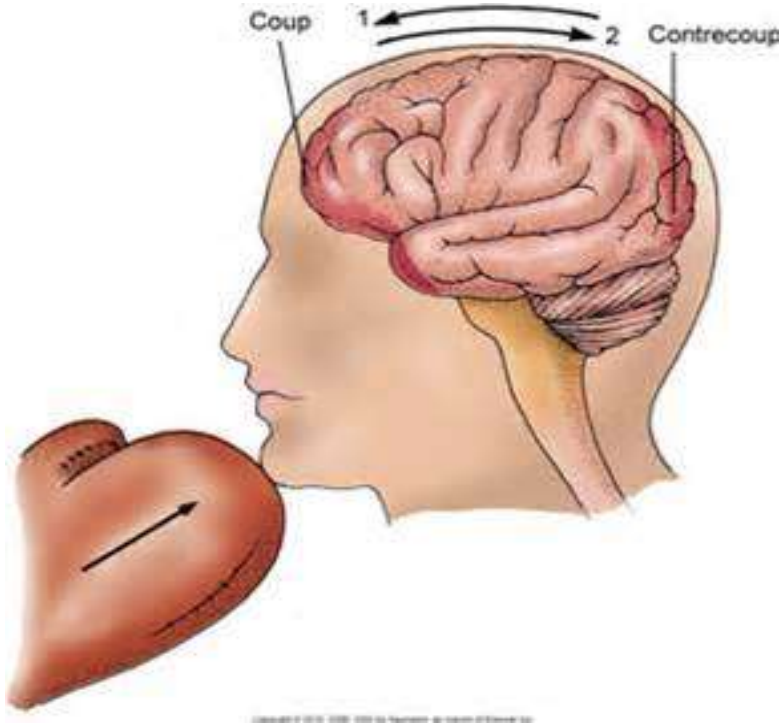
Head injury

- Blunt or penetrating.
- Open or closed.
- Severe brain damage can occur without external signs of head injury
- Lacerations and even skull fractures are not necessarily associated with brain damage

Traumatic parenchymal injury

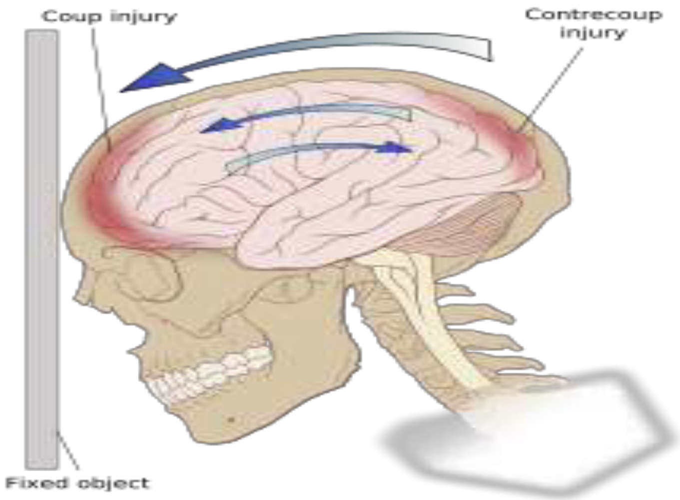
When an object impacts the head:

- Injury of brain at site of impact: coup injury
- Injury opposite to site of impact: countercoup
- Both are contusions


Coup
1
2
Contrecoup

Shot down in flames
Shot down in flames
Ain't it a shame?
To be shot down in flames?

Seems like a touch, a touch too much
Seems like a touch, a touch too much
Too much for my body, too much for my brain
This damn woman is gonna drive me insane



I'm gonna walk all over you
I'm gonna walk all over you
Do anything you want me to to you
I'm gonna walk all over you

Note:

- Repetitive episodes of trauma can later lead to neurodegenerative process e:g Alzheimer

Brain injury

- Concussions
- Contusions
- Lacerations
- Diffuse axonal damage

concussions

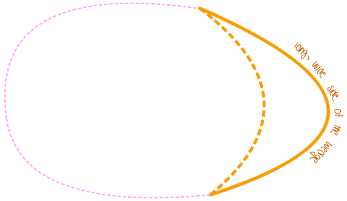
Mildest form of brain injury, caused by sudden shaking/movement.
It is NOT associated with tissue damage. It is transient and reversible.

- Reversible altered consciousness after head injury in the absence of contusions
- Transient dysfunction in the form of: loss of consciousness, temporary respiratory arrest, loss of reflexes.
- Pathogenesis: unknown
- Recovery is complete but amnesia of the episode.

contusion

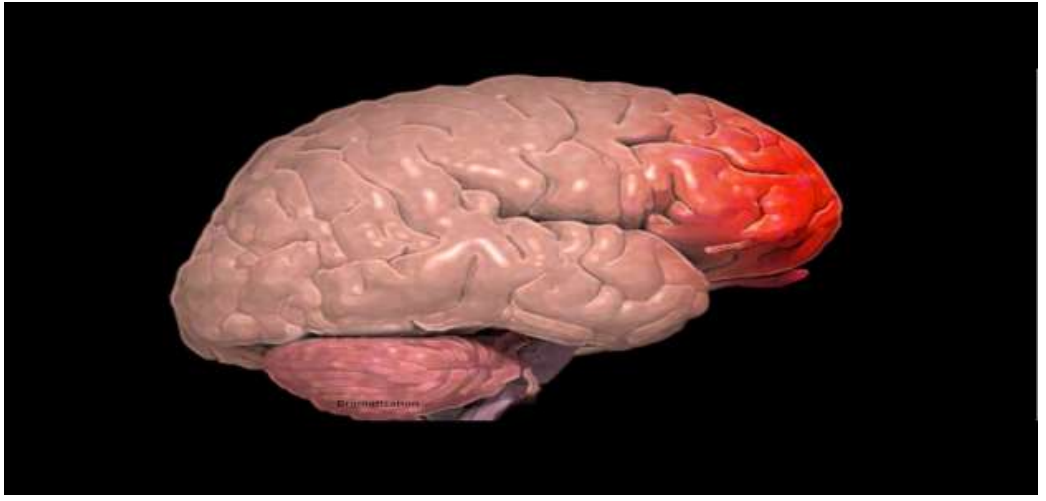
- Caused by rapid tissue displacement , disruption of vascular channels with subsequent haemorrhage, tissue injury and edema.
- Common in areas overlying rough and irregular bone surface: orbitofrontal region, temporal lobe tips.

Contusion/morphology



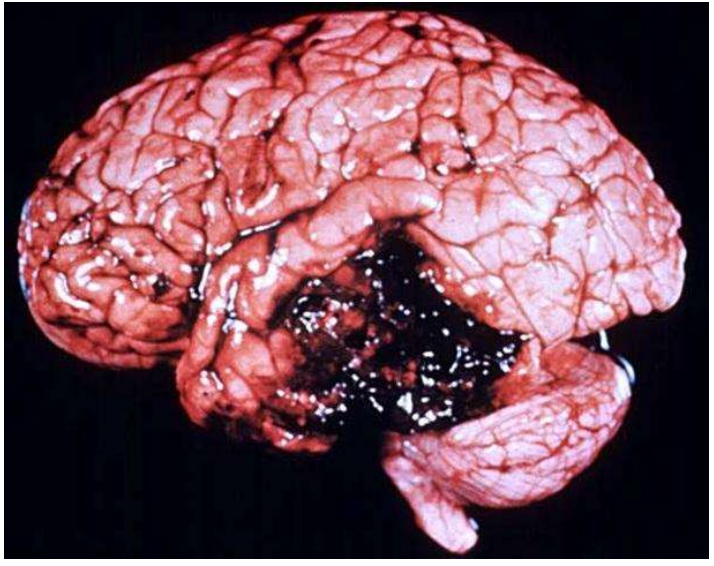
- Wedge shaped, widest aspect closest to point of impact.
- Edema and extravasated RBCs.
- Superficial aspects of cortex affected more (contrary to ischemic injury)

But still NO brain tissue damage.
Only vascular.



lacerations

- Penetrating injuries cause skull fractures and brain lacerations
- Laceration: tissue tearing and hemorrhage.



- Old traumatic injury: depressed, retracted, yellow brown patches involving the gyri.
- Larger lesions: cavity, resembling remote infarcts

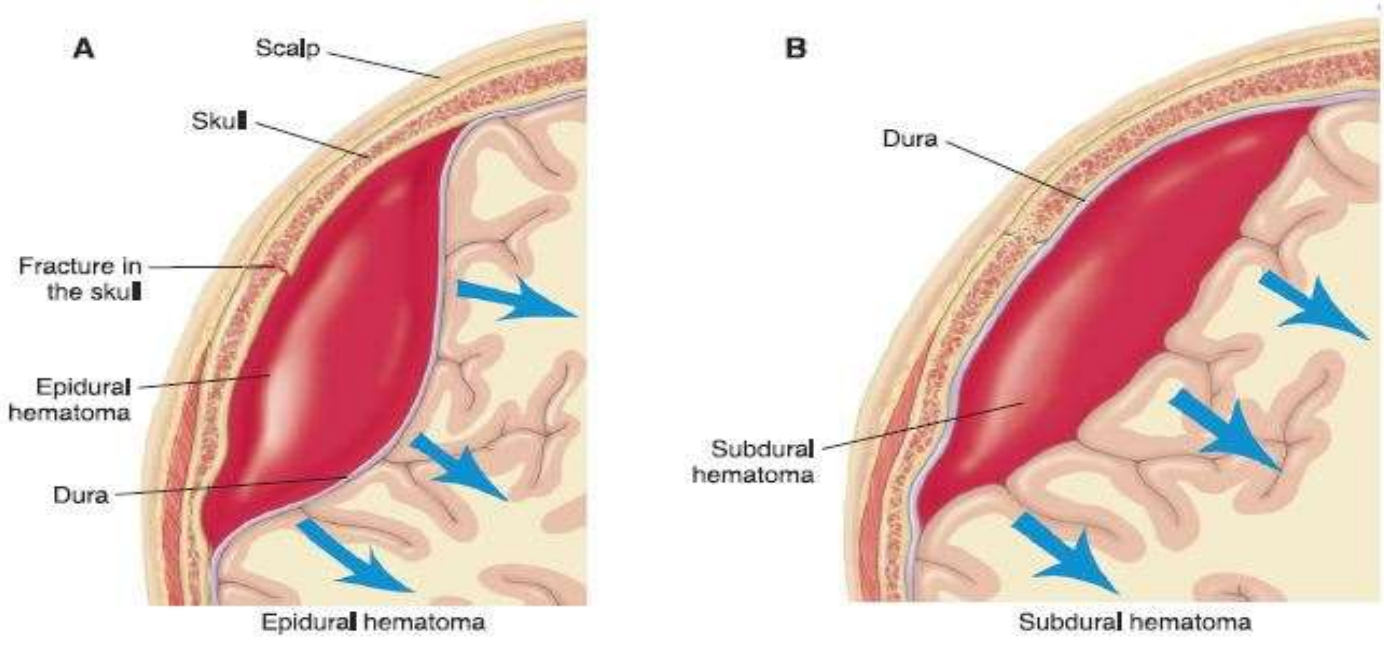
Diffuse axonal injury

- Brain trauma can cause subtle widespread injury to axons within the brain:= diffuse axonal injury
- Movement of one region of the brain relative to another.. disrupt axonal integrity. Axons move against each other and stretch, which causes damage that may be irreversible.
- Appear under LM as axonal swelling
- Can lead to severe irreversible neurologic deficit.

The mechanism is unknown, and upon checking the brain, we'll merely see some swelling.

Traumatic vascular injury

- Epidural
- Subdural
- Subarachnoid
- intraparenchymal



Dura peels off the bone, where hemorrhage is above the dura.

Epidural hematoma

- Dural vessel torn due to fracture.
- Usually: middle meningeal artery is torn
- Blood accumulates under arterial pressure and dissects the dura, compressing the brain parenchyma

Epidural hematoma

This s a CT scan showing blood between the dura and the skull

note the biconvex shape.. this is typical of epidural hematoma.

Subdural hematoma

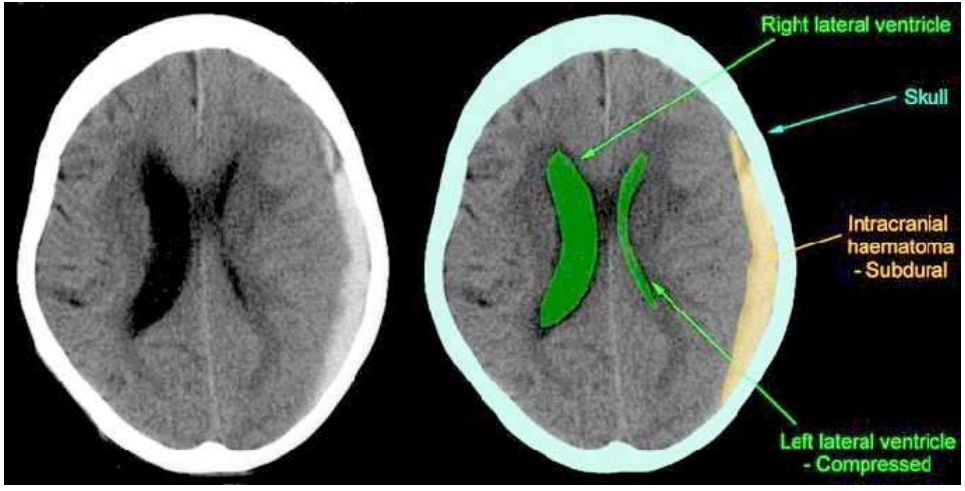
- Rapid movement of brain during trauma.. Can tear the bridging veins
- This leads to bleeding in the subdural space

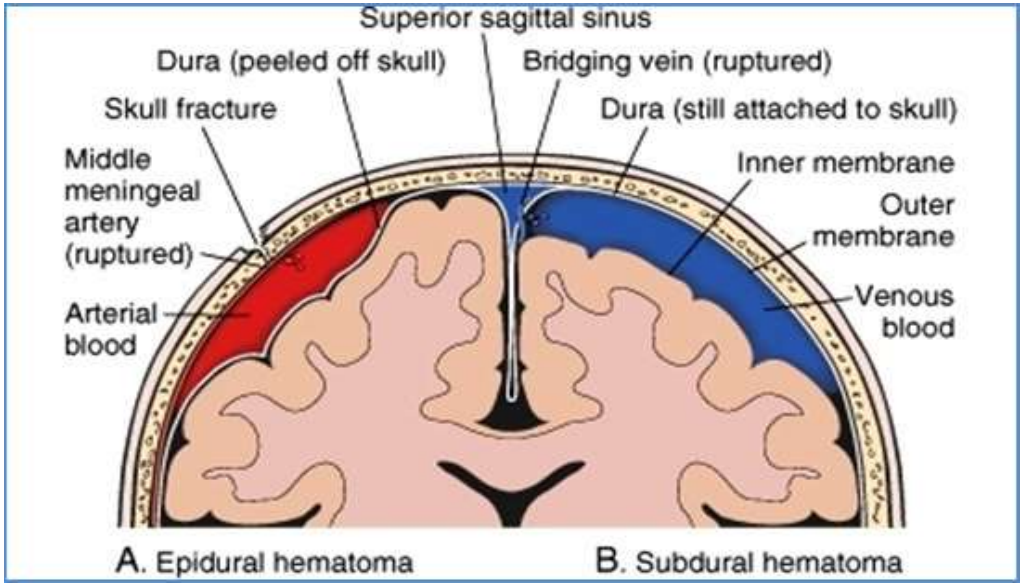
Subdural hematoma

here the blood collects between the dura and the brain tissue

It shows a crescentic shape.

Subdural hematoma





- Epidural hematoma usually caused by rupture of arteries.
- The most common affected artery is MMA.
- Usually caused by a stronger trauma that often results in fracture.

- Subdural hematoma usually caused by rupture of venous bridging veins.
- Usually no associated fracture, so there'd be a trauma that didn't cause fracture but affected the vulnerable bridging veins.

Summary 1/2

- Intracranial haemorrhage can traumatic or non traumatic.
- Intracranial haemorrhage can be intra-parenchymal, subarachnoid, epidural or subdural; the first two can be traumatic or non traumatic whereas the last two are usually traumatic.
- the most common cause of intraparenchymal haemorrhage is spontaneous haemorrhage which occurs in older individuals who are hypertensive.
- Hypertension causes haemorrhage via weakening blood vessel walls through hyaline arteriolosclerosis or micro-aneurysm formations.
- Hypertension also uses slit haemorrhages, lacunar infarcts and acute hypertensive encephalopathy.

SUMMARY 2/2

- Other causes of intraparenchymal haemorrhage include: amyloid antipathy, infections, autoimmune vasculitis, arteriovenous malformations and other causes.
- Subarachnoid haemorrhage can be traumatic but is mainly caused by a ruptured aneurysm.
- Traumatic brain haemorrhage can be subdural, epidural, intra-parenchymal or subarachnoid.
- epidural hematoma caused by arterial vessel (middle meningeal) torn due to fracture.Blood accumulates under arterial pressure and dissects the dura, compressing the brain parenchyma
- Subdural hematoma occurs due to rapid movement of brain during trauma.. Can tear the bridging veins.This leads to bleeding in the subdural space :


thank
you