

Central nervous system

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Cerebral Edema

- Cerebral edema is the accumulation of excess fluid within the brain parenchyma.
- This term should be distinguished from **hydrocephalus**, an increase in CSF volume within all or part of the ventricular system.

- There are **two underlying mechanisms** for the development of cerebral edema that often occur together particularly when there is generalized injury:
 - *1-Vasogenic edema*
 - *2-Cytotoxic edema*

- **1-Vasogenic edema**

- Occurs when the integrity of the normal blood-brain barrier is disrupted.

- With increased vascular permeability fluid shifts from the vascular compartment into the intercellular spaces of the brain.

-Vasogenic edema can be either:

A- localized

**because of abnormal permeability of vessels
adjacent to inflammation or tumors**

B- Generalized.

- **2-Cytotoxic edema**

- implies an increase in intracellular fluid secondary to neuronal, glial, or endothelial cell membrane injury.

- be encountered in an individual with a generalized hypoxic/ischemic insult or with exposure to some toxins.

CENTRAL NERVOUS SYSTEM TRAUMA

- Trauma to the brain and spinal cord is a significant cause of death and disability.
- **Severity** and **site** of injury affect the outcome.
- injury of several cubic centimeters of brain parenchyma may be :
 - clinically silent (if in the frontal lobe)
 - severely disabling (spinal cord)
 - fatal (involving the brain stem)

- **The magnitude and distribution of traumatic brain lesions depend on :**
- **1-the shape of the object causing the trauma**
- **2-the force of impact**
- **3- whether the head is in motion at the time of injury**

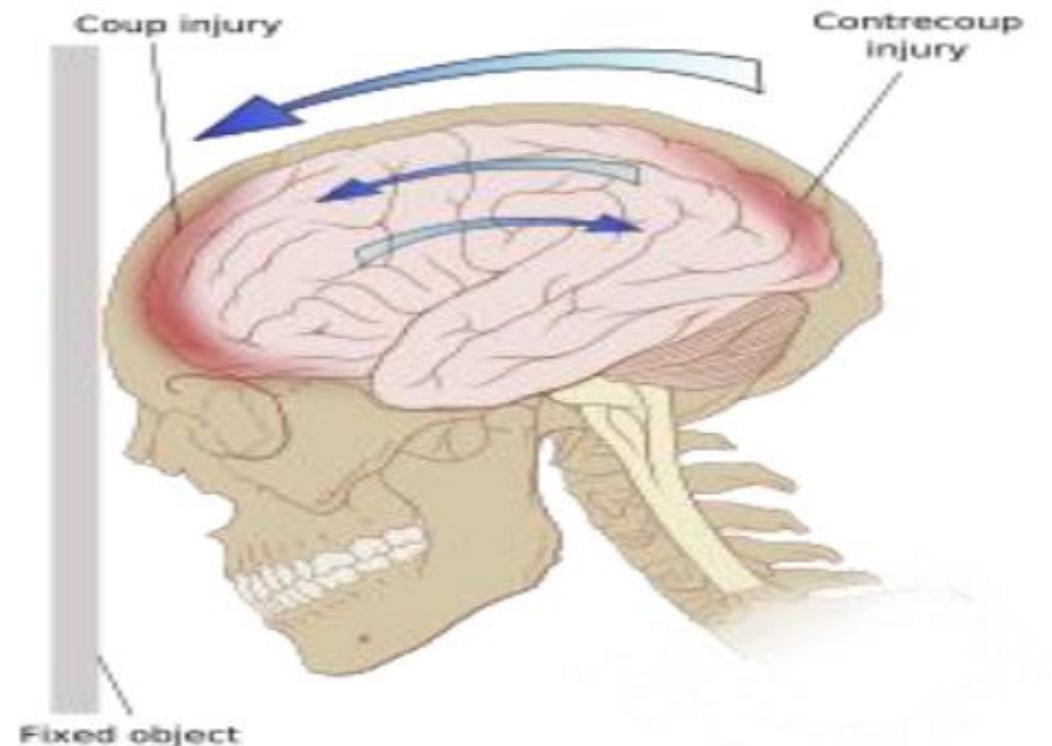
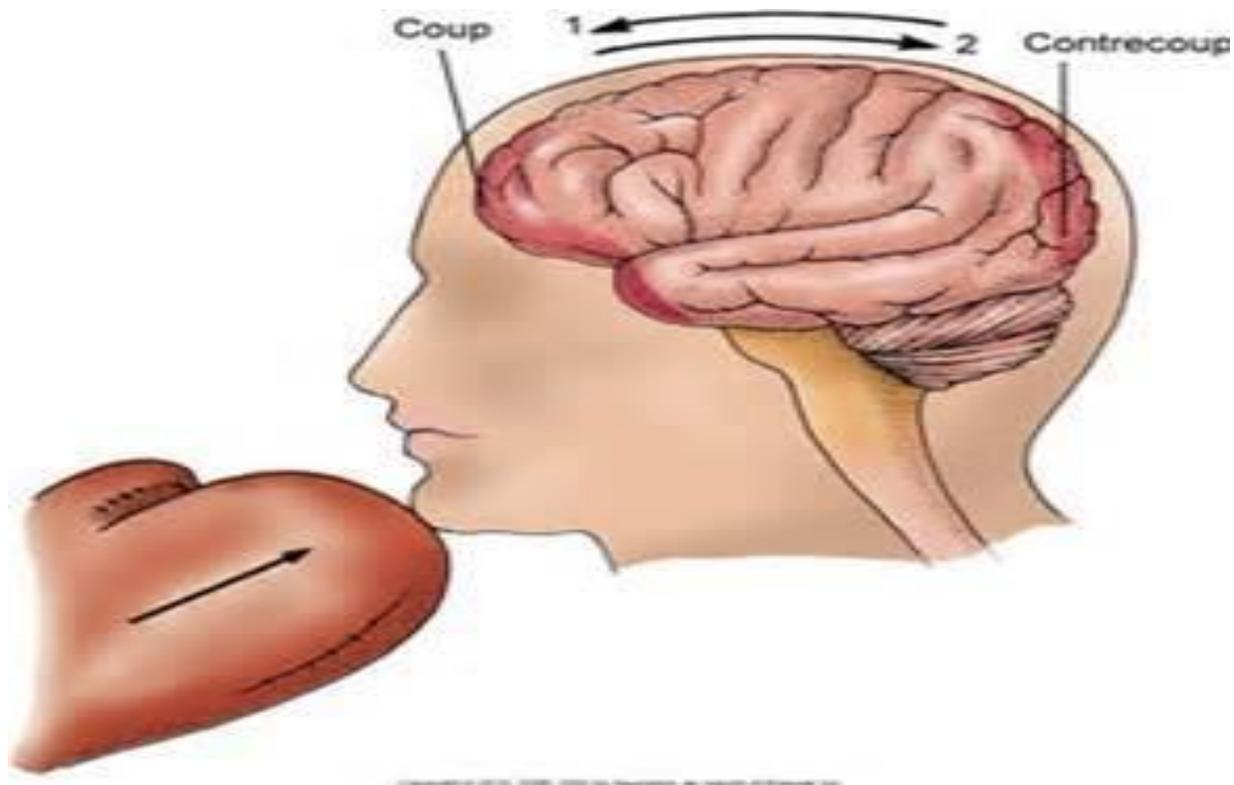
- **A blow to the head may be :**
 - *penetrating* or *blunt*
 - *open* or a *closed injury*.
- **Severe brain damage can occur in the absence of external signs of head injury.**
- **Severe lacerations and even skull fractures do not necessarily indicate damage to the underlying brain.**

- In addition to skull or spinal fractures, trauma can cause :
- 1-**parenchymal** injury
- 2-**vascular** injury
- 3-combinations of both

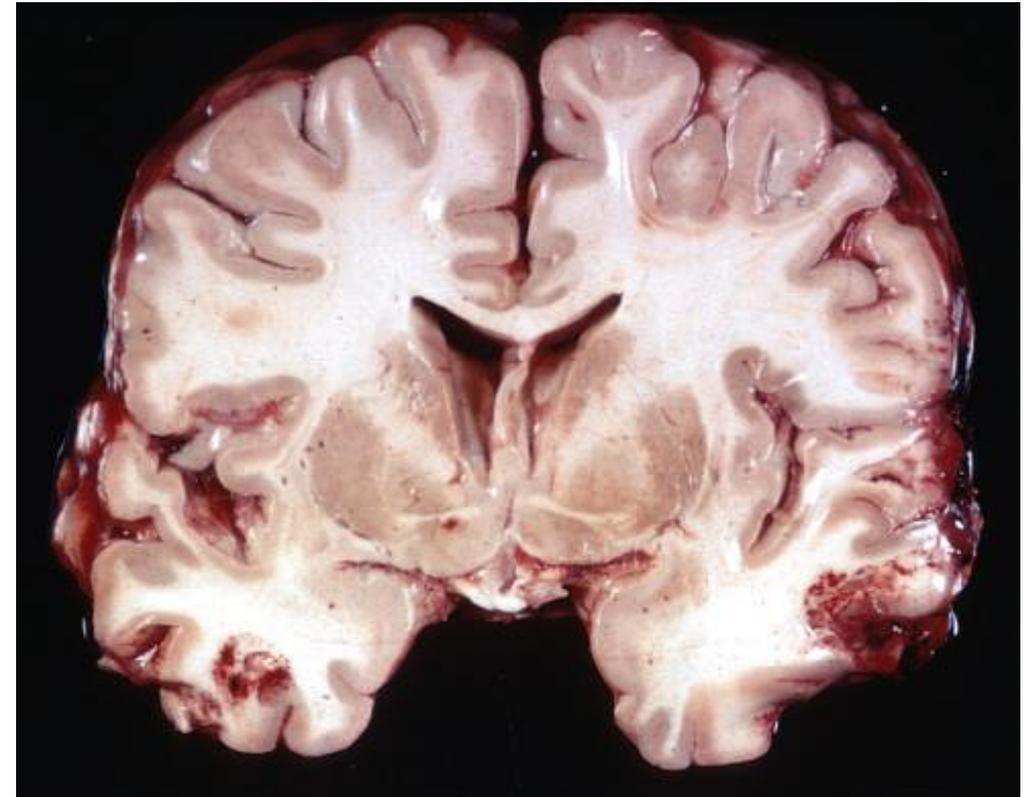
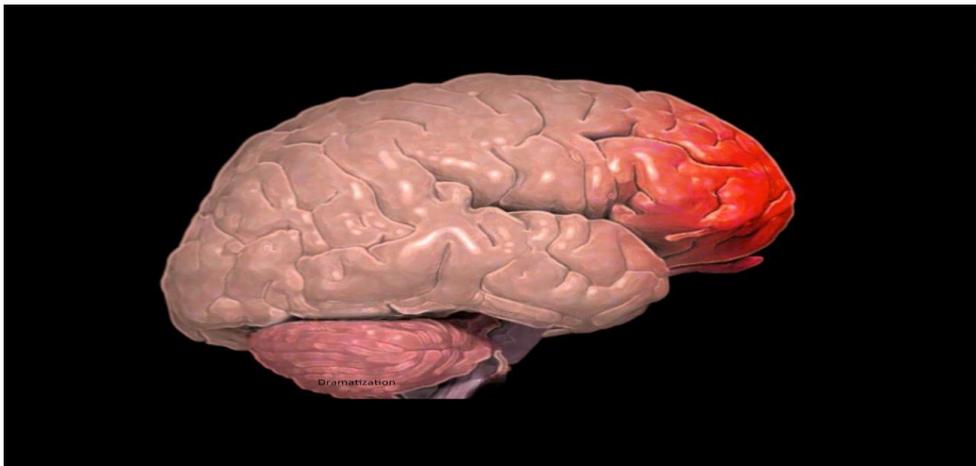
Traumatic Parenchymal Injuries

- Types of brain injury :
- 1- *contusions*
- 2- *laceration*
- 3- *diffuse axonal injury*
- 4- *Concussion*

- When there is impact of an object with the head
- ***contusions*** :brain injury that occur from collision of the brain with the skull at the site of impact (a ***coup*** injury)
- or on the opposite side (***contrecoup***)

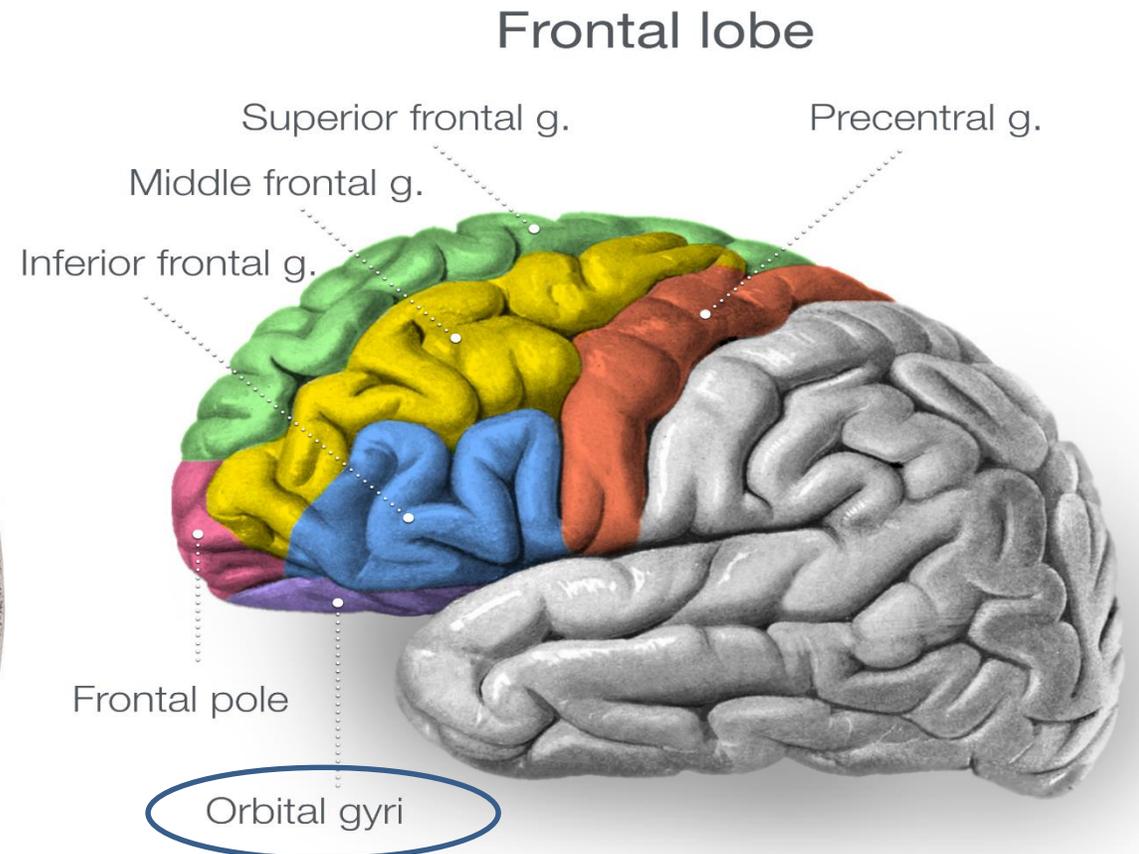
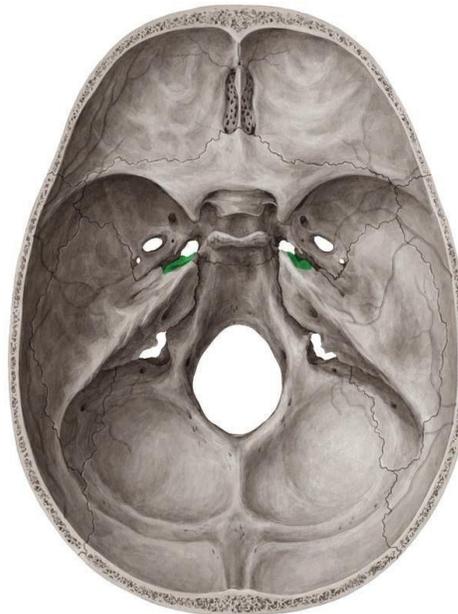
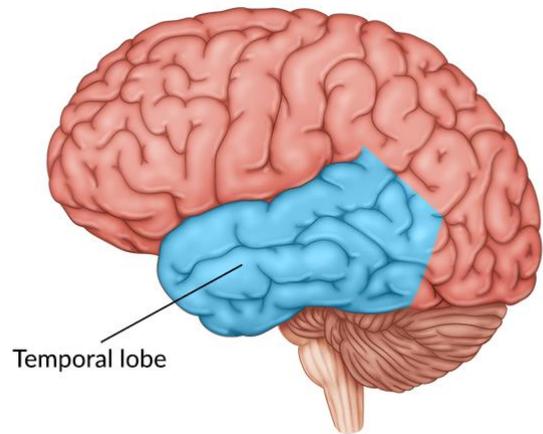


- A contusion is caused by rapid tissue displacement, disruption of vascular channels, and subsequent hemorrhage, tissue injury, and edema.
- Since they are the points of impact, **crests of gyri are most susceptible.**
- cerebral cortex along the sulci is less vulnerable .

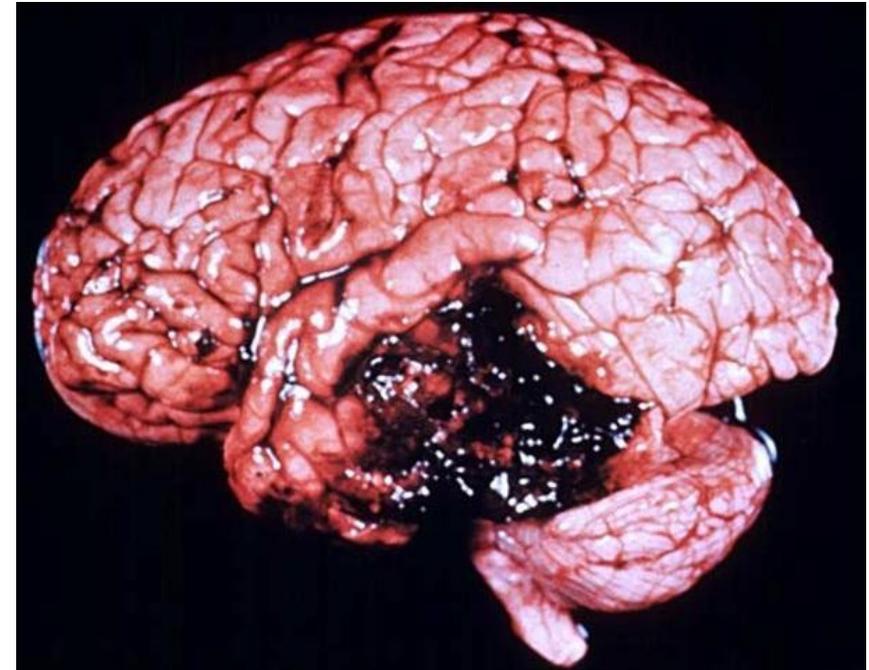


- The most common locations where contusions occur correspond to the most frequent sites of direct impact and to regions of the brain that overlie a rough and irregular inner skull surface, such as :

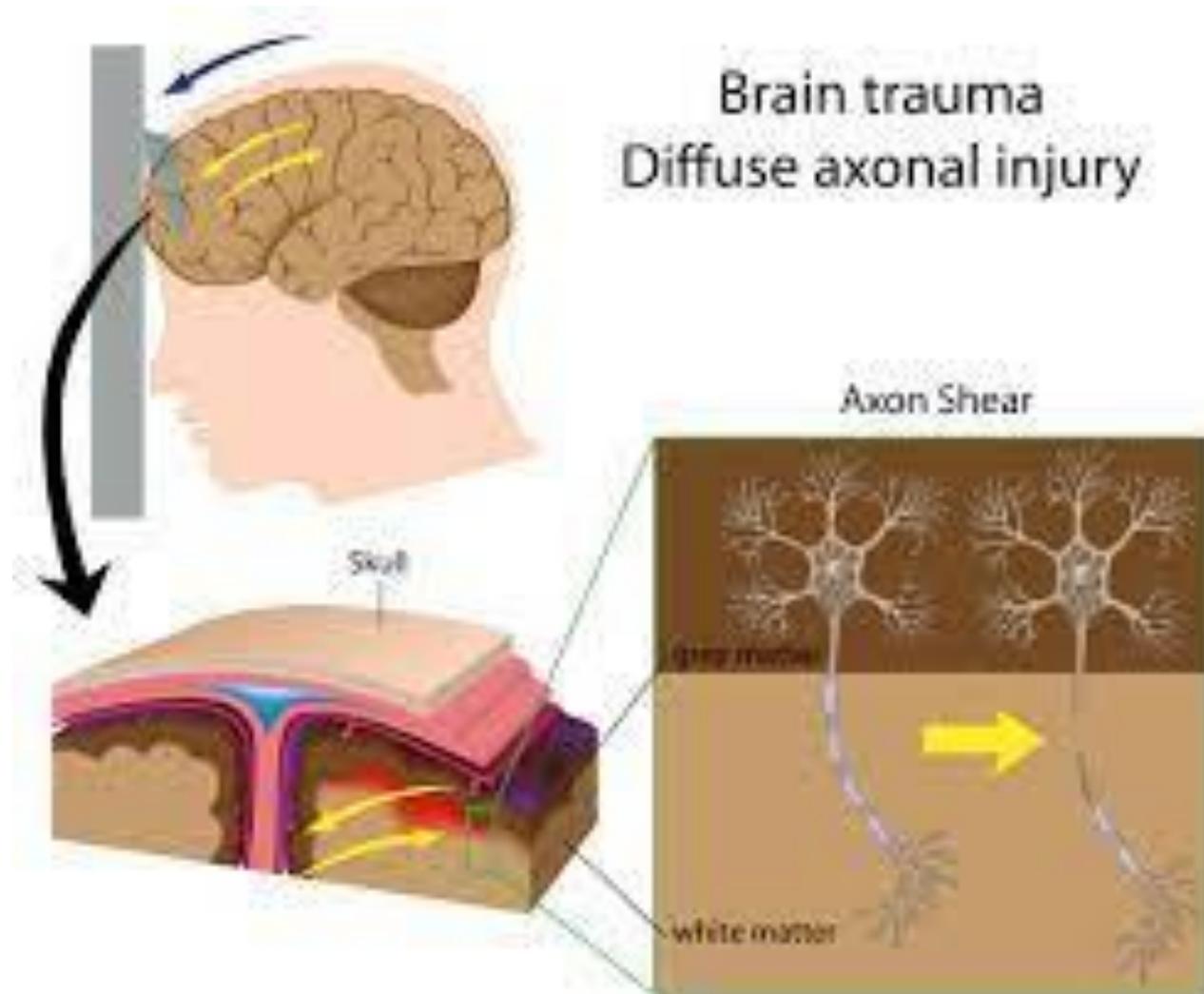
- 1-the frontal lobes
- 2-the orbital gyri
- 3-the temporal lobes.



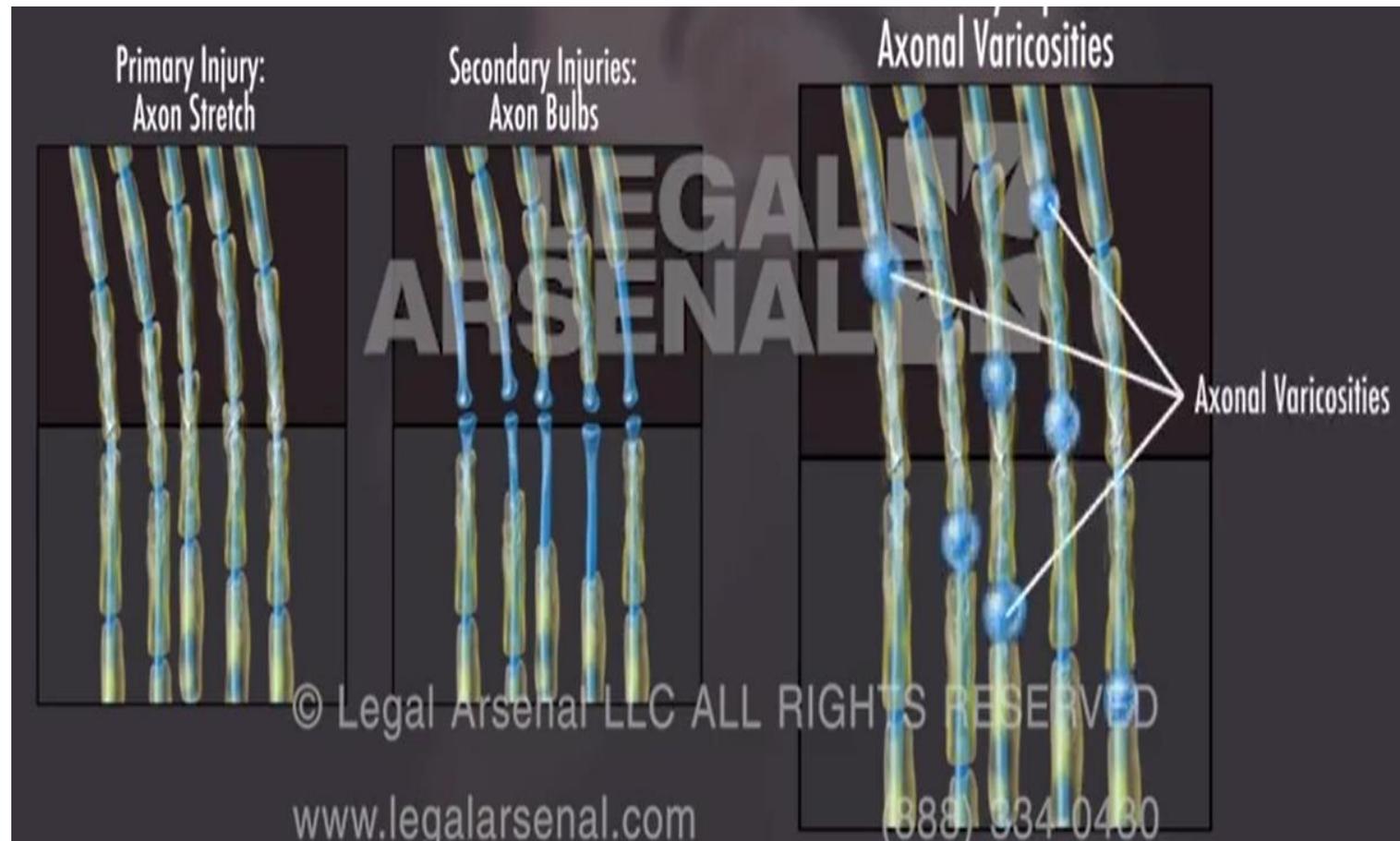
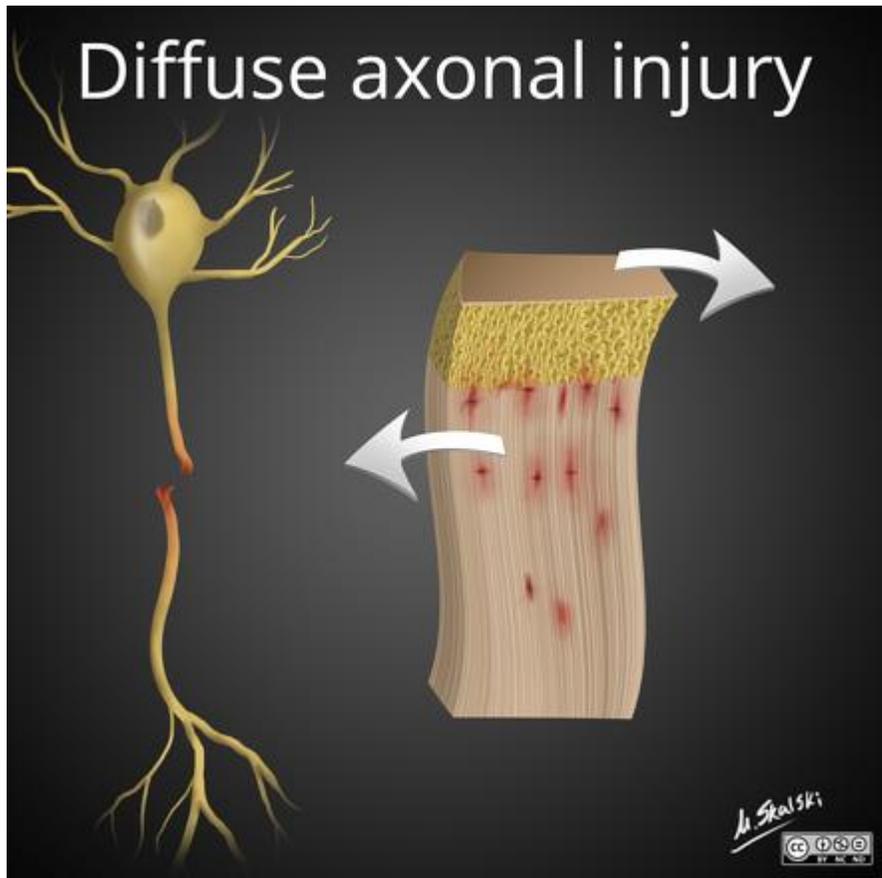
- ***laceration*** :
- penetration of the brain, either by a projectile such as a bullet or a skull fragment from a fracture رصاصة
- occurs, with tissue tearing, vascular disruption, hemorrhage, and injury along a linear path.



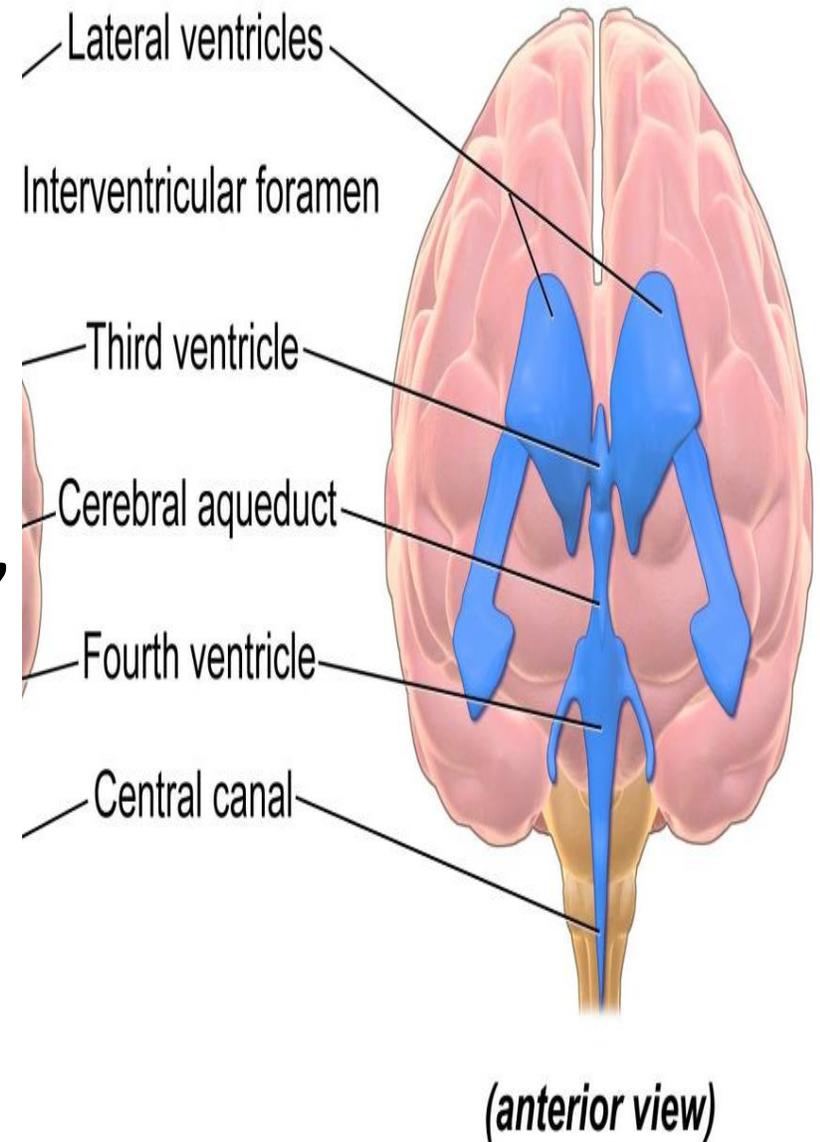
- ***diffuse axonal injury***
- Although injury to the surface of the brain is often the most dramatic, widespread injury to axons within the brain
- The movement of one region of brain relative to another is thought to lead to the disruption of axonal integrity and function.
- Angular acceleration alone in the absence of impact may cause axonal injury as well as hemorrhage.



- **Diffuse axonal injury is characterized by the wide but often asymmetric distribution of axonal swellings that appears within hours of the injury and may persist for much longer.**



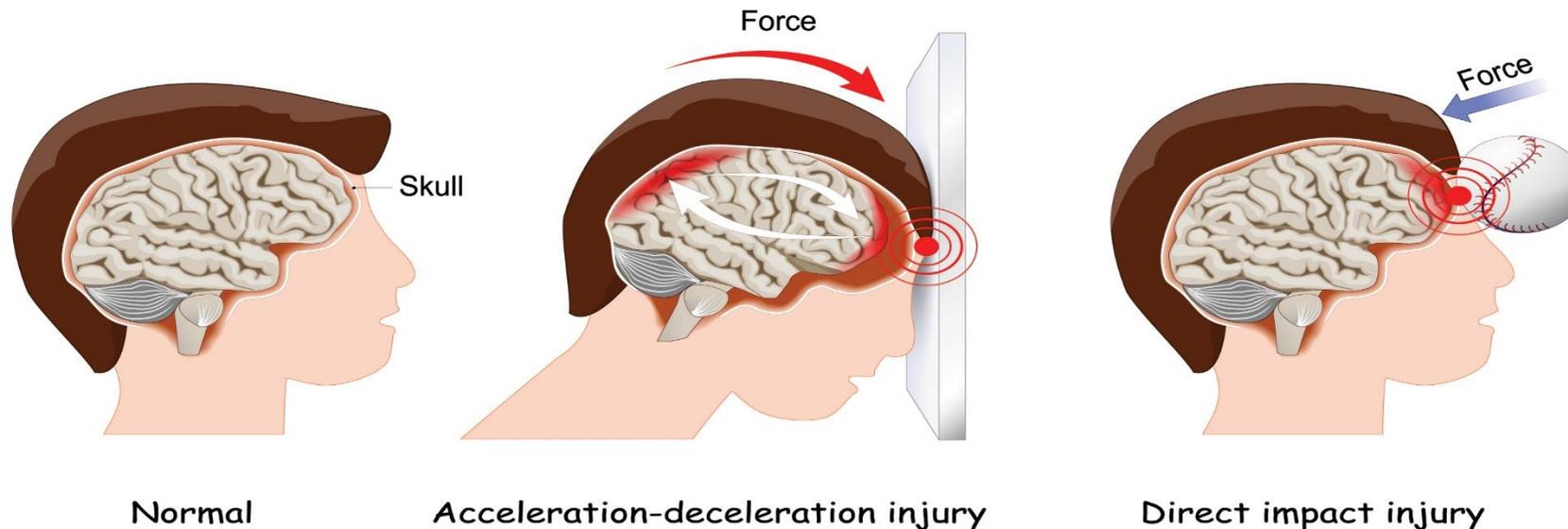
- As many as 50% of patients who develop coma shortly after trauma, even without cerebral contusions, are believed to have white matter damage and diffuse axonal injury.
- Although these changes may be widespread, lesions are most commonly found **near the angles of the lateral ventricles and in the brain stem.**



- **Concussion**
- describes reversible altered consciousness from head injury in the absence of contusion.

انه الشخص يتعرض لضربة
ما تسببه إصابة بس بتخلي
دماغه يتحرك جوا الجمجمة
ف يفقد الوعي

Concussion



- **The characteristic transient neurologic dysfunction includes :**
- **1-loss of consciousness.**
- **2-temporary respiratory arrest.**
- **3-loss of reflexes.**

- **Although neurologic recovery is complete, amnesia فقدان الذاكرة for the event persists.**
- **The pathogenesis of the sudden disruption of nervous activity is unknown**

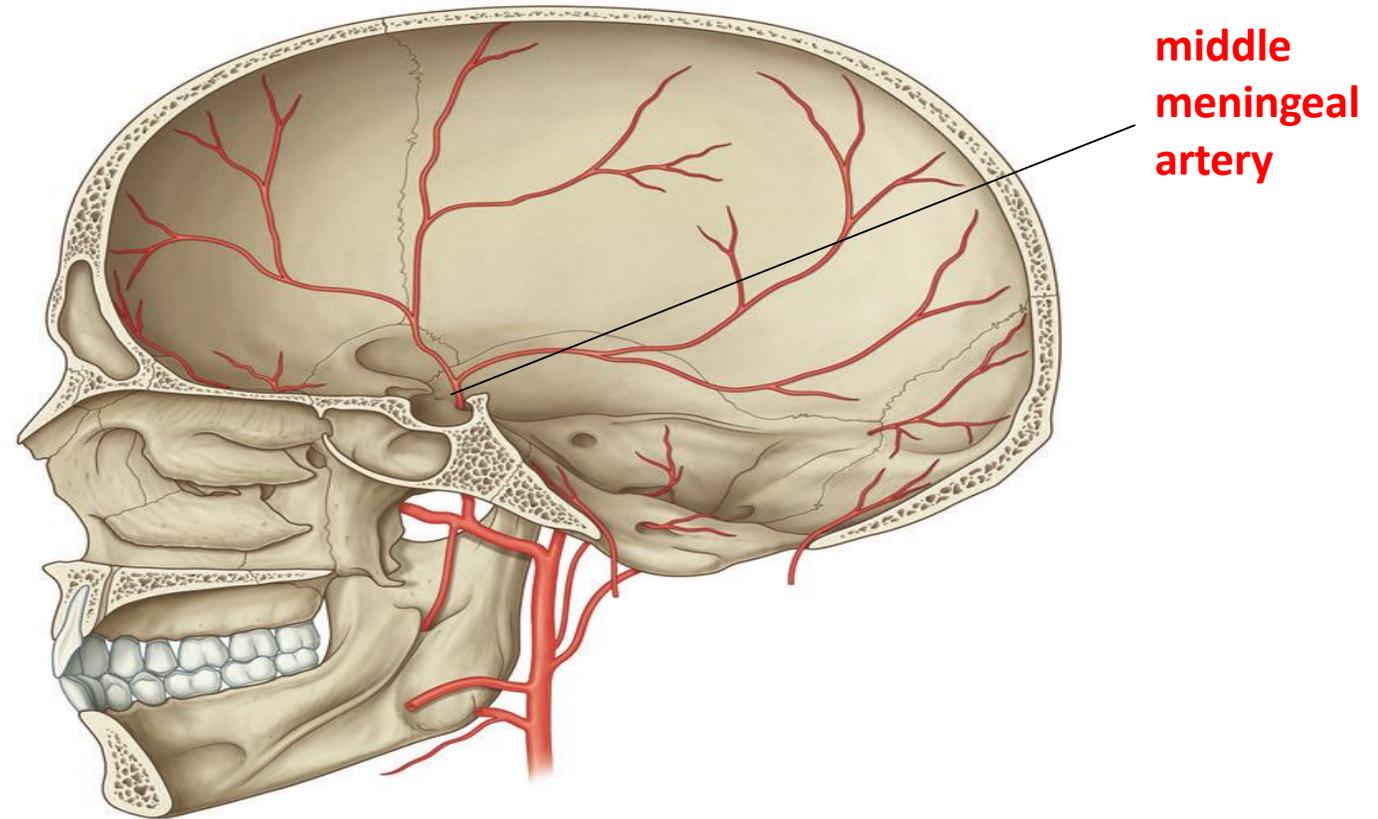
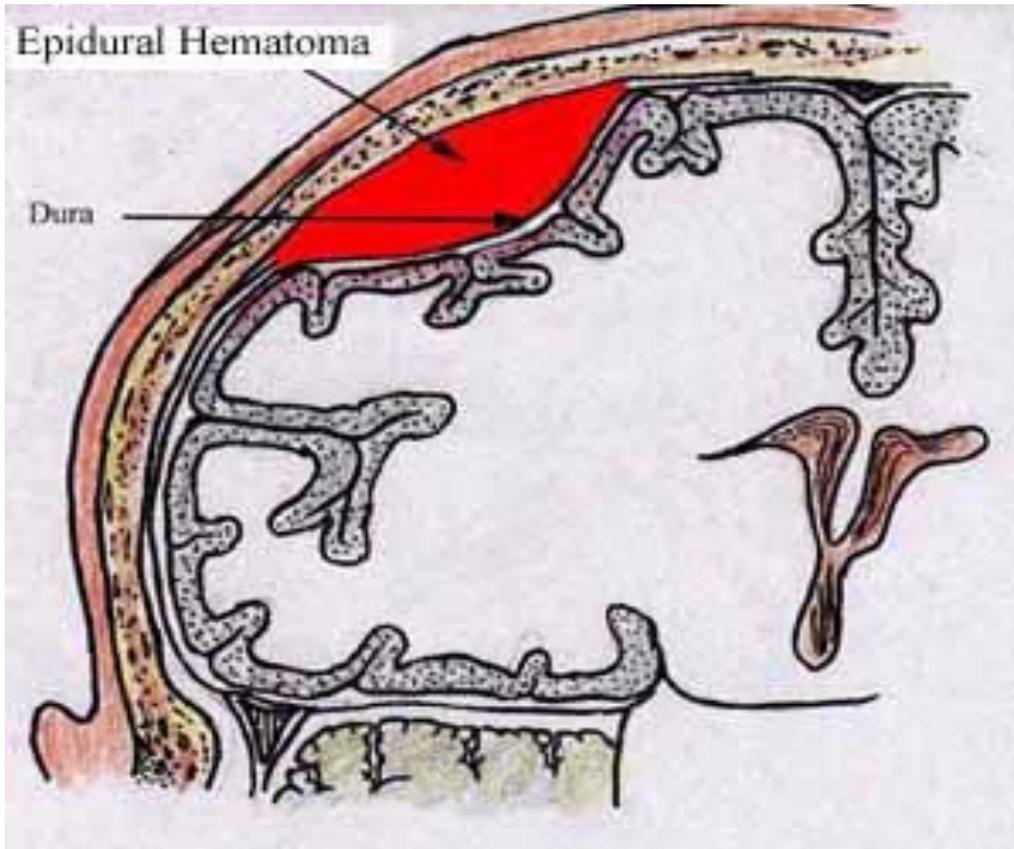
Traumatic Vascular Injury

- Vascular injury is a frequent component of CNS trauma and results from direct trauma and disruption of the vessel wall, leading to hemorrhage.
- Depending on which vessels rupture, hemorrhage may occur in any of several compartments :
 - *1- epidural*
 - *2-subdural*
 - *3-subarachnoid*
 - *4-intraparenchymal*

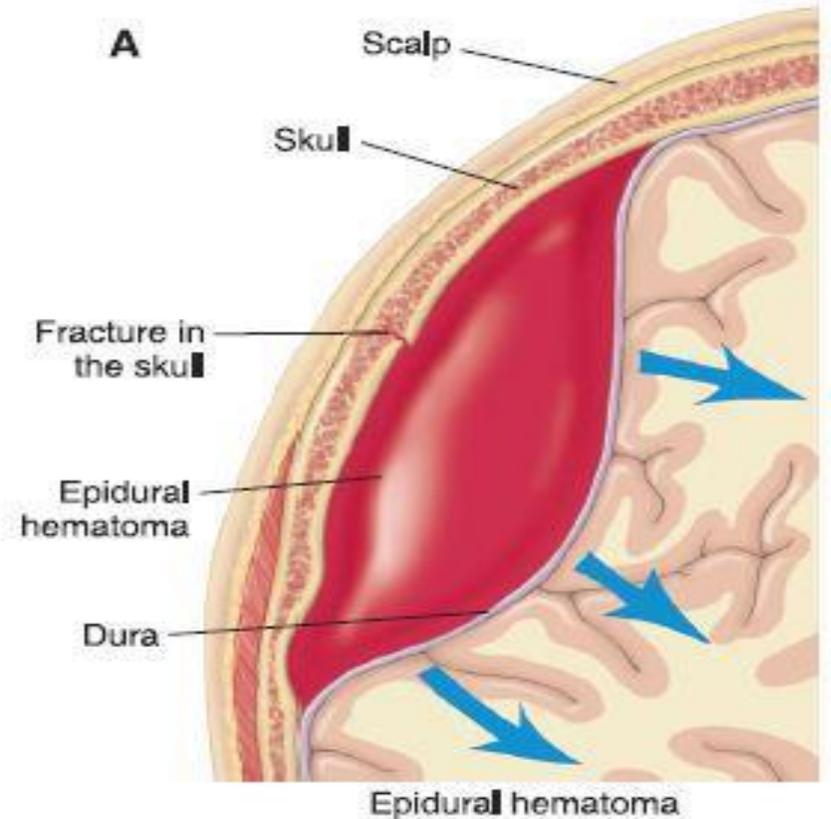
- **Subarachnoid and intraparenchymal hemorrhages most often occur at sites of contusions and lacerations.**

Epidural Hematoma

- The dura is normally tightly applied to the inside of the skull fused with the periosteum.
- Vessels that run in the dura most importantly the **middle meningeal artery** are vulnerable to injury particularly with skull fractures.
- In children in whom the skull is deformable, a temporary displacement of the skull bones may tear a vessel in the absence of a skull fracture.



- Once a vessel has been torn, the accumulation of blood under arterial pressure can cause separation of the dura from the inner surface of the skull
- The expanding hematoma has a smooth inner contour that compresses the brain surface.

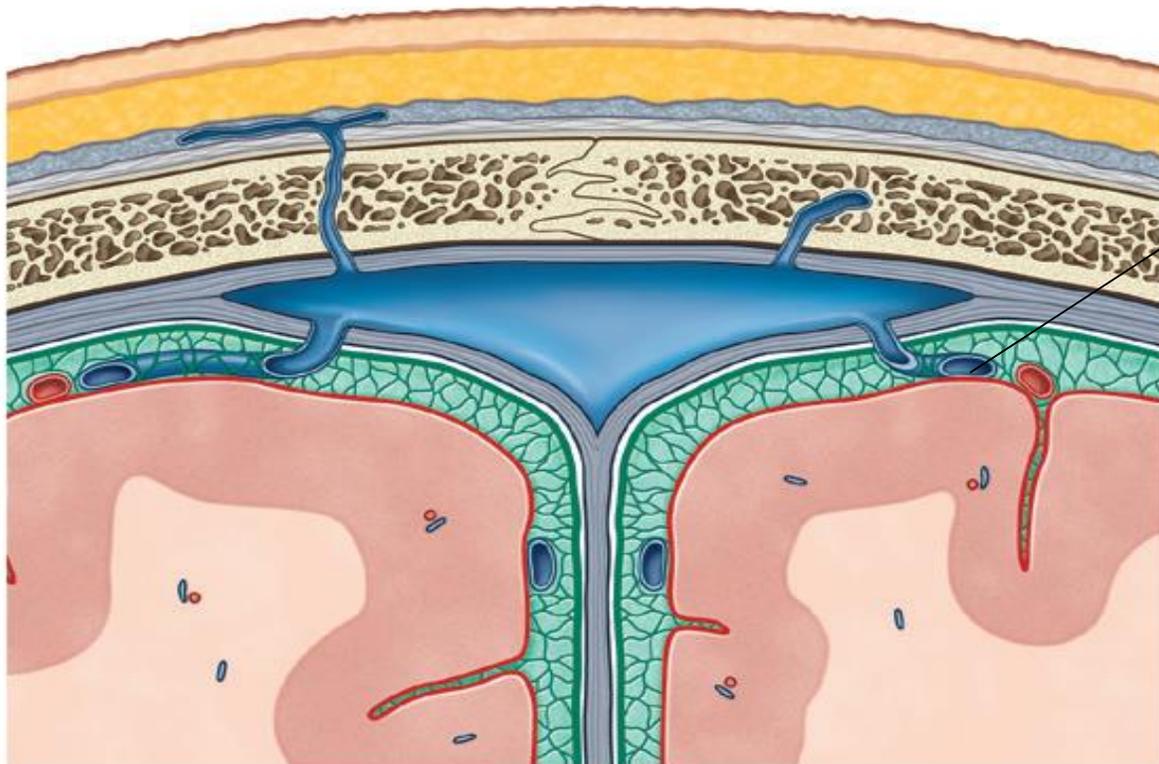


بدون أعراض

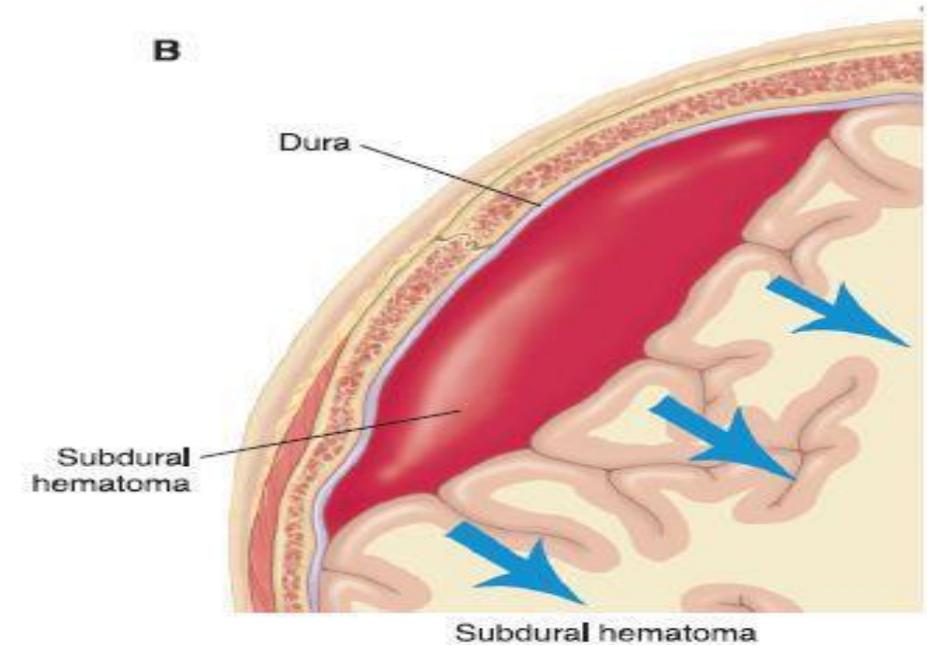
- ***Clinically, patients can be lucid for several hours between the moment of trauma and the development of neurologic signs.***
- **An epidural hematoma may expand rapidly and is a neurosurgical emergency requiring prompt drainage.**

Subdural Hematoma

- The rapid movement of the brain that occurs in trauma can **tear the bridging veins** that extend from the cerebral hemispheres through the subarachnoid and subdural space to empty into dural sinuses.
- These vessels are particularly prone to tearing, and their disruption leads to bleeding into the subdural space.



Bridging vein

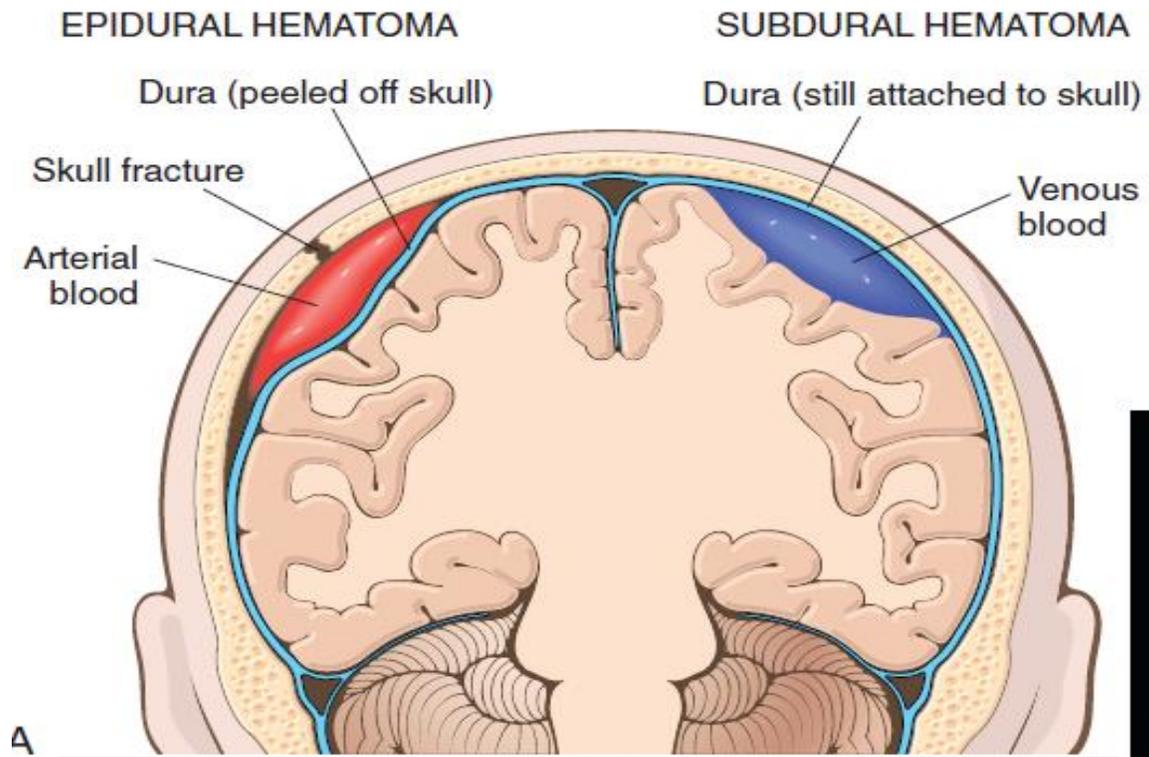


- **Risk factors :**
- **1-In elderly** patients with brain atrophy the bridging veins are stretched out and the brain has additional space for movement, accounting for the higher rate of subdural hematomas in these patients, even after relatively minor head trauma.
- **2-Infants** are also susceptible to subdural hematomas because their bridging veins are thin-walled.

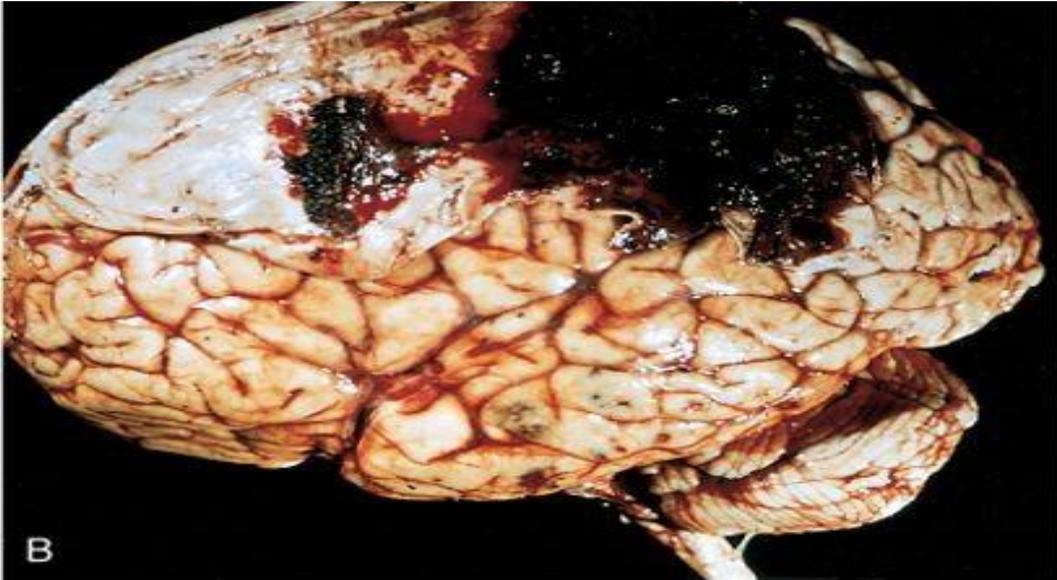
- **Subdural hematomas most often become manifest within the first 48 hours after injury.**
- **They are most common over the lateral aspects of the cerebral hemispheres**
- **bilateral in about 10% of cases.**



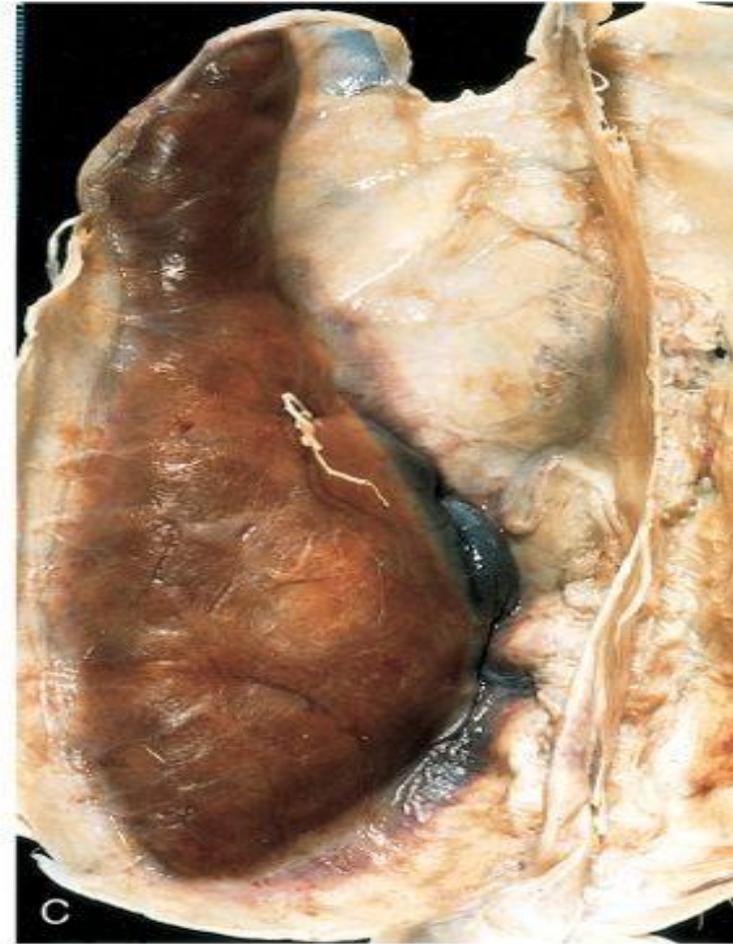
- **Neurologic signs are attributable to the pressure exerted on the adjacent brain.**
- **These may be focal, but often the clinical manifestations are nonlocalizing and include headache or confusion.**
- **In time there may be slowly progressive neurologic deterioration, rarely with acute decompensation.**



A



B



C

CEREBROVASCULAR DISEASES

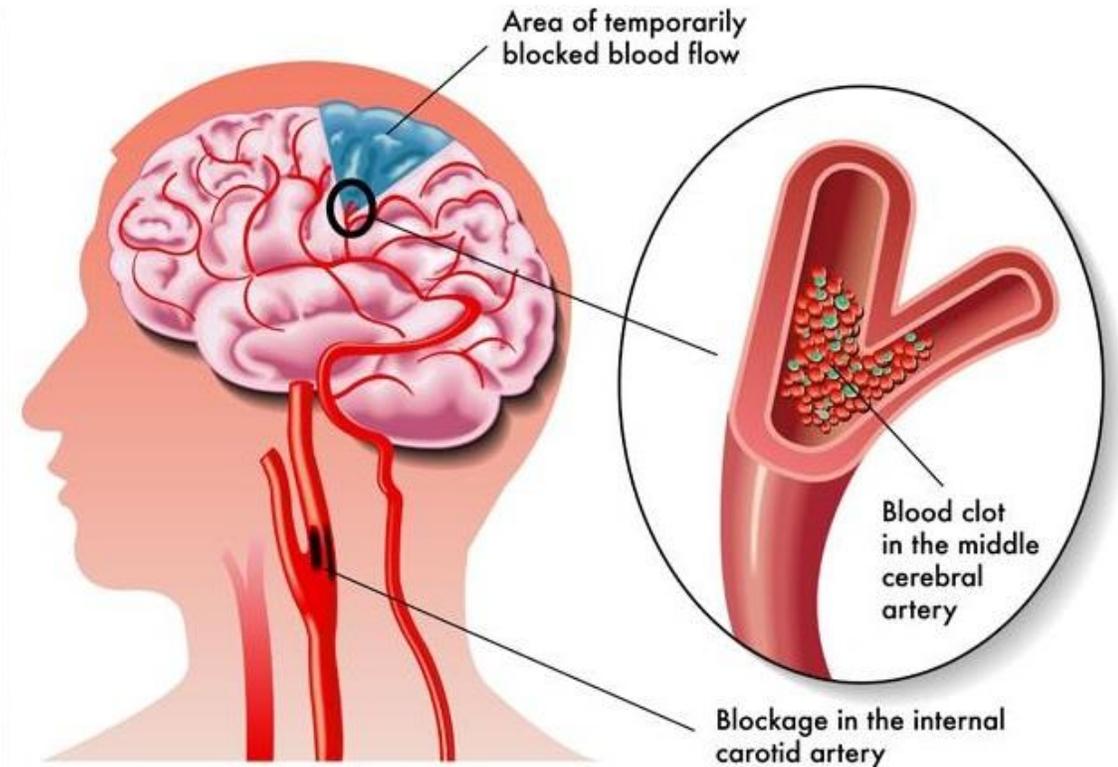
- any abnormality of the brain caused by a pathologic process involving blood vessels.
- The three basic processes are :
- (1) thrombotic occlusion of vessels,
- (2) embolic occlusion of vessels,
- (3) vascular rupture.
- The first two share many characteristics, because their effect on the brain is the same-loss of oxygen and metabolic substrates resulting in brain infarction.

- Thrombosis and embolism cause **ischemic injury or infarction** of specific regions of the brain, depending on the vessel involved.
- A similar pattern of injury occurs diffusely when there is **complete loss of perfusion** (or delivery of oxygen and metabolic substrate).

- Hemorrhage accompanies rupture of vessels, leading to direct tissue damage as well as secondary ischemic injury.
- "**Stroke**" is the clinical designation that applies to all these conditions, particularly when symptoms begin acutely.

Hemorrhagic stroke

SAMPLE USE ONLY



Hypoxia, Ischemia, and Infarction

- **The brain requires a constant delivery of glucose and oxygen from the blood.**
- **Although the brain accounts for only 1% to 2% of body weight, it receives 15% of the resting cardiac output and accounts for 20% of the total body oxygen consumption.**
- **Cerebral blood flow remains constant over a wide range of blood pressure and intracranial pressure because of autoregulation of vascular resistance.**
- **The brain is a highly aerobic tissue, with oxygen being the limiting substance.**

- **The brain may be deprived of oxygen by any of several mechanisms:**
- ***1-functional hypoxia* in a setting of :**
- **A-a low partial pressure of oxygen**
- **B-impaired oxygen-carrying capacity**
- **C-inhibition of oxygen use by tissue**

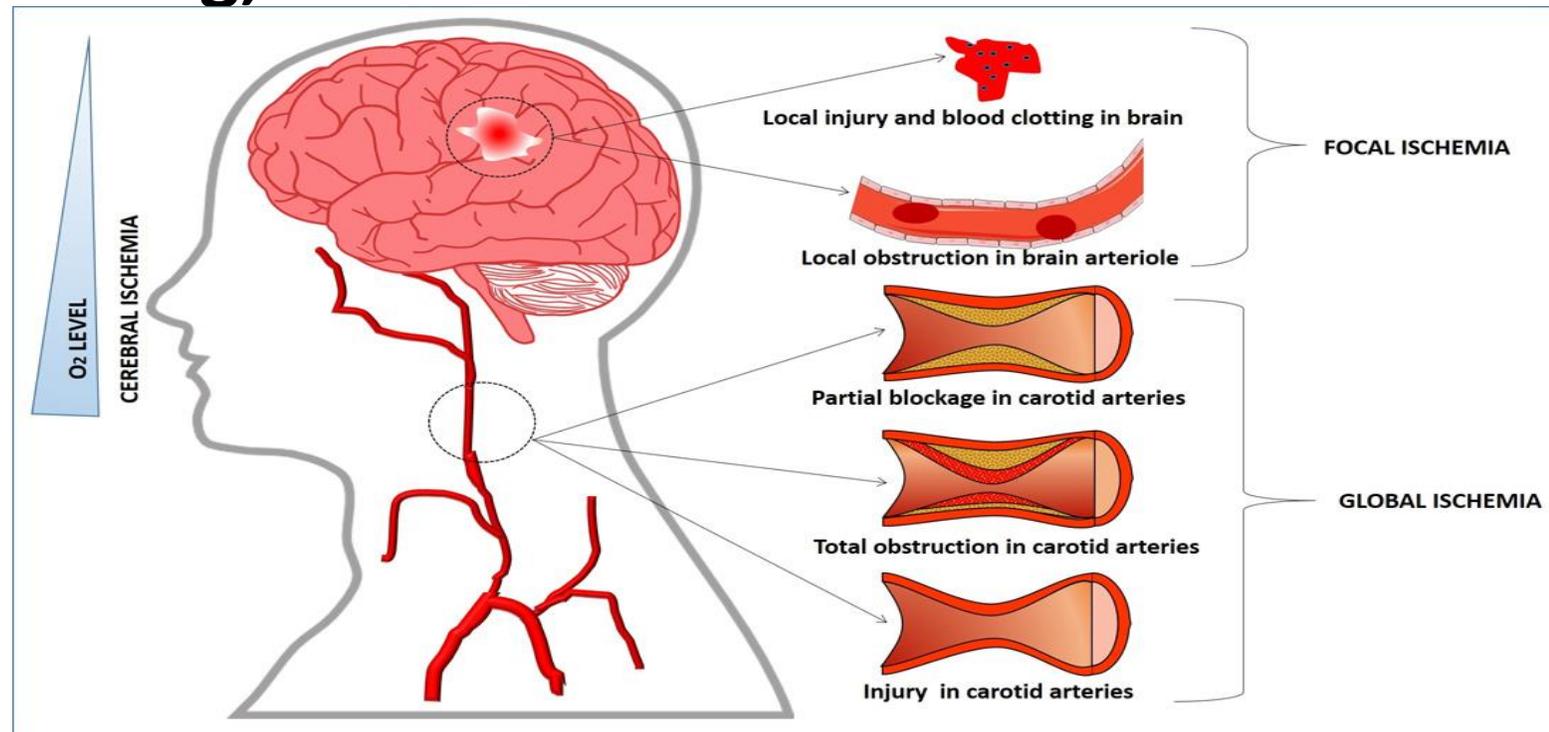
- **2-ischemia**
- ***A-transient***
- ***B-permanent***

- **Cessation of blood flow can result from:**
- **1-reduction in perfusion pressure, as in hypotension.**
- **2-secondary to vascular obstruction.**
- **3-both .**

Ischemia can be global or focal

Global Cerebral Ischemia

- **widespread ischemic/hypoxic injury occurs when there is a generalized reduction of cerebral perfusion, usually below systolic pressures of less than 50mmHg, such as in :**
- **1-cardiac arrest.**
- **2-shock.**
- **3-severe hypotension.**



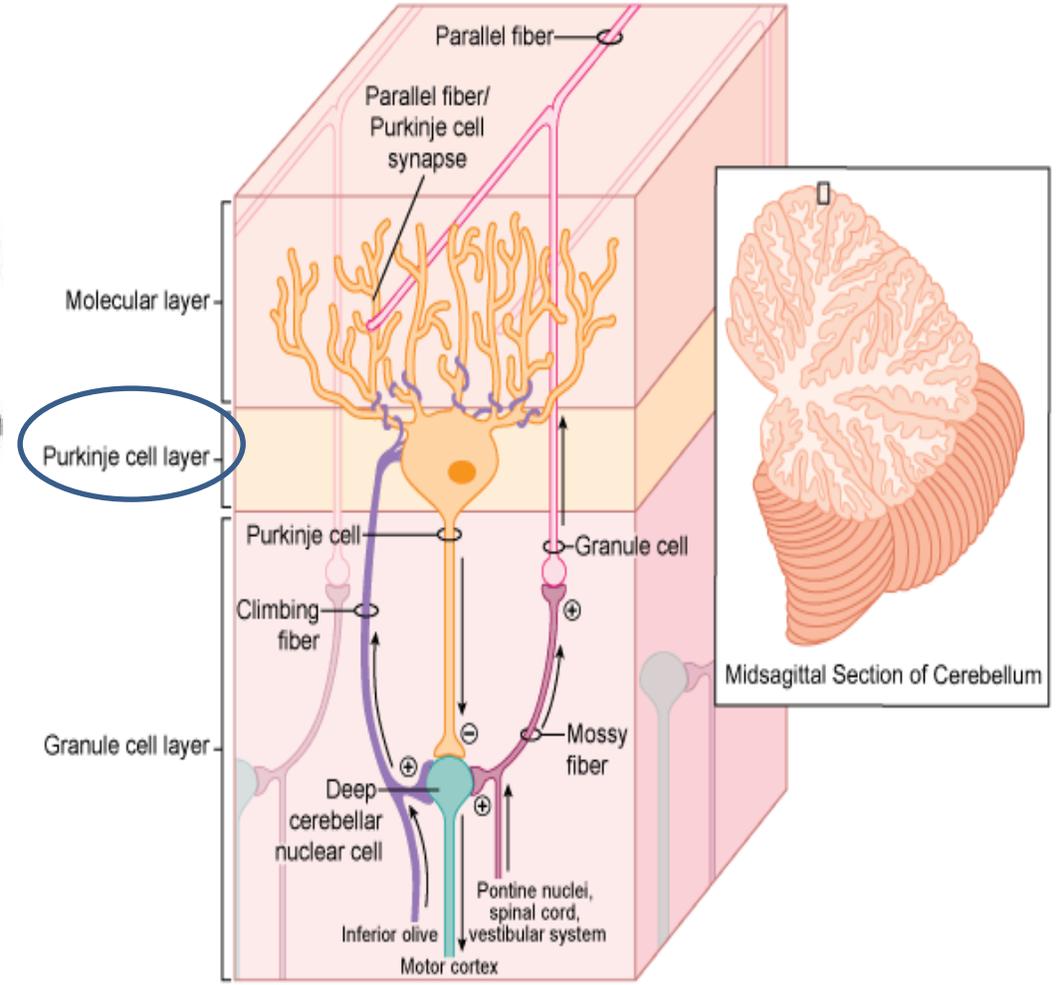
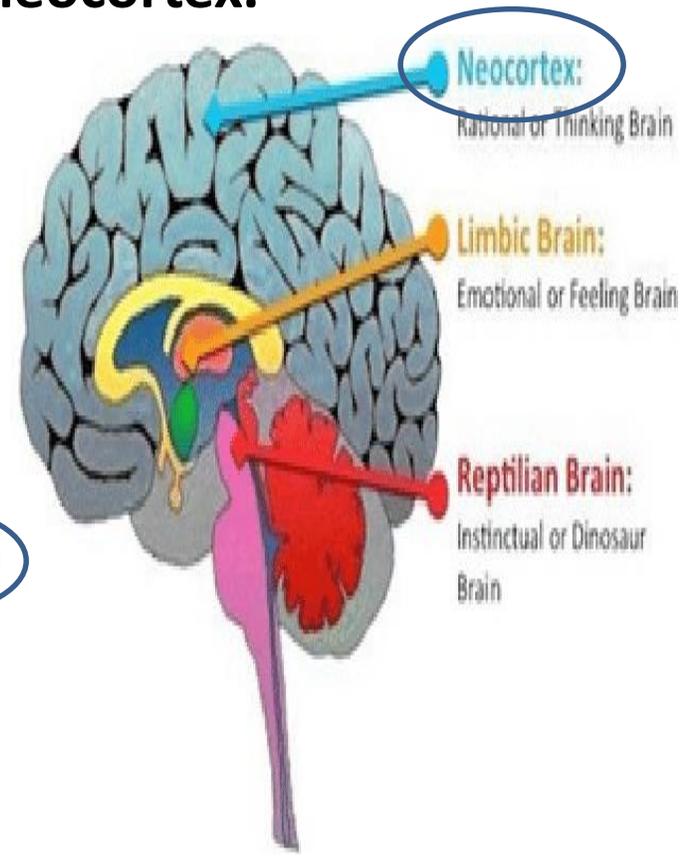
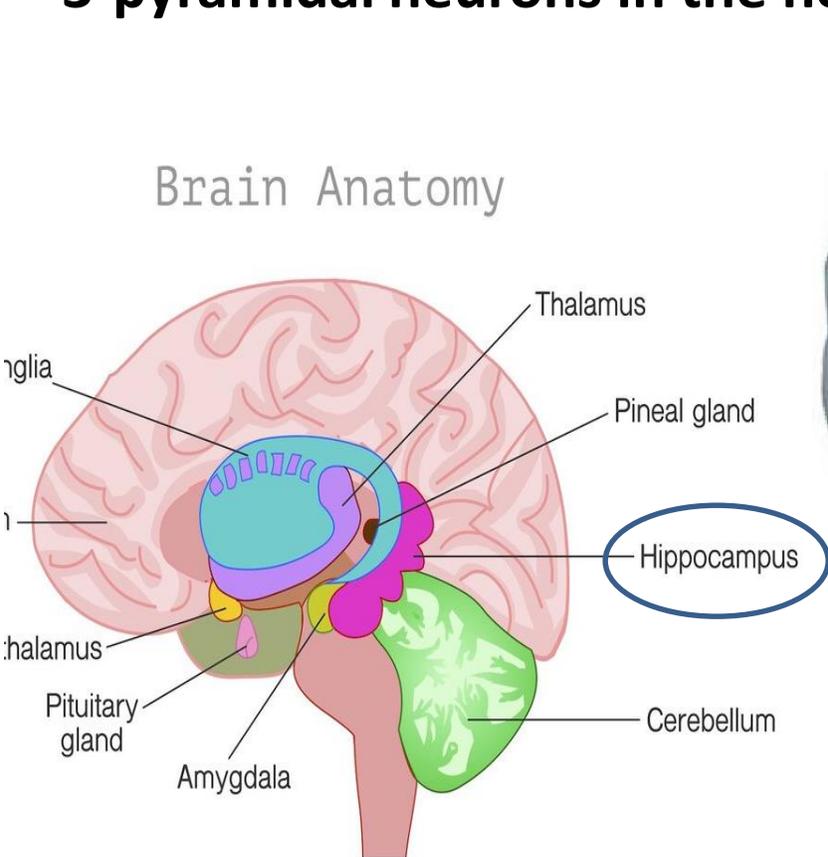
Clinical manifestation

- The clinical outcome varies with the severity of the insult.
- When **mild**, there may be only a **transient postischemic confusional state**, with eventual complete recovery.
- **Irreversible damage** of CNS tissue does occur in some individuals who suffer **mild or transient global ischemic insults**.
- Neurons are much more sensitive to hypoxia than are glial cells.

• There is also variability in the susceptibility of different populations of neurons in different regions of the CNS .

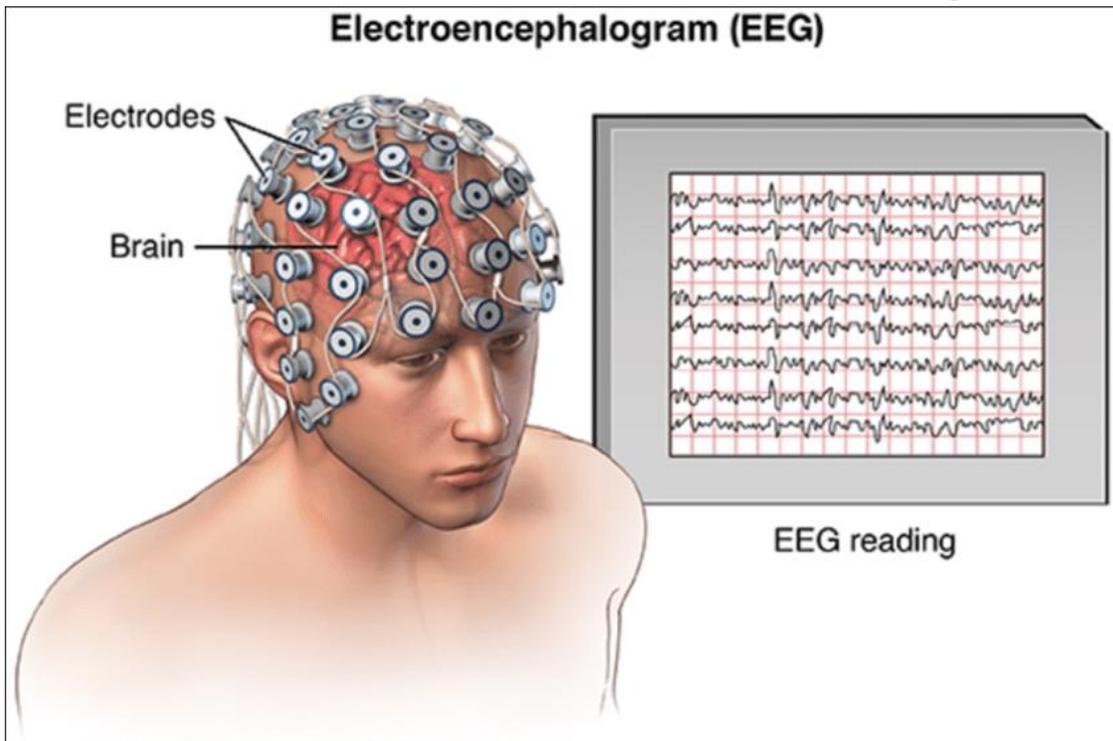
• The most susceptible cells to ischemia of short duration are :

- 1-pyramidal cells of the Sommer sector (CA1) of the hippocampus.
- 2-Purkinje cells of the cerebellum.
- 3-pyramidal neurons in the neocortex.



- In severe global cerebral ischemia, widespread neuronal death irrespective of regional vulnerability occurs.
- Individuals who survive in this state often remain severely impaired neurologically and deeply comatose (**persistent vegetative state**). حالة غيبوبة مستمرة.

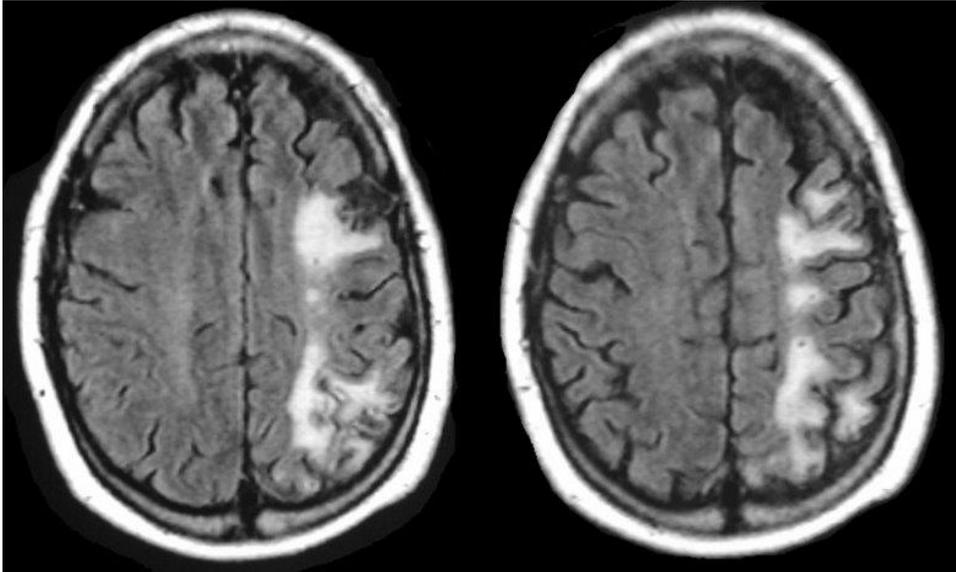
- The clinical criteria for "**brain death**" include :
- 1-evidence of diffuse cortical injury (isoelectric, or "flat," electroencephalogram)
- 2-brain stem damage including absent reflexes and respiratory drive.



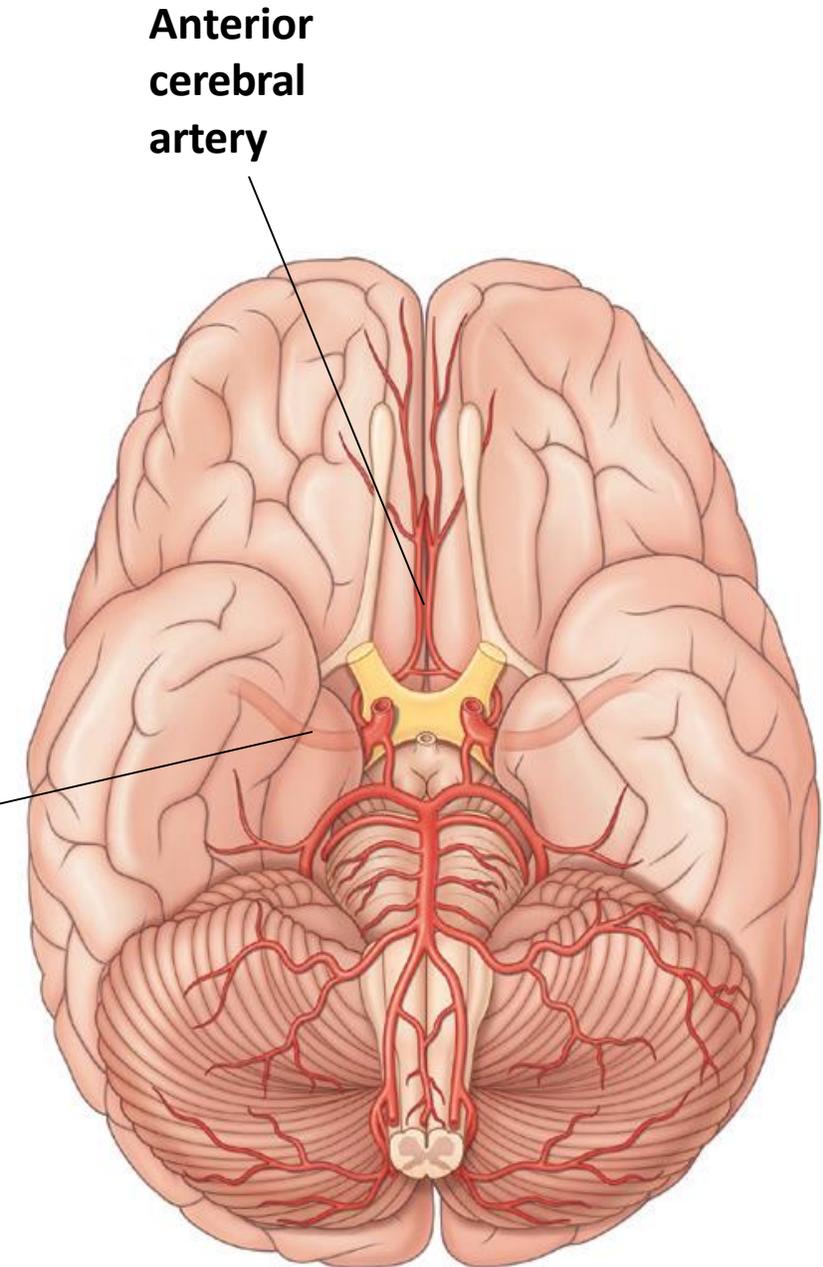
- When patients with this pervasive form of injury are maintained on mechanical ventilation, the brain gradually undergoes an **autolytic process**, resulting in the so-called "**respirator brain**."



- **Border zone ("watershed") infarcts**
- are wedge-shaped areas of infarction that occur in those regions of the brain and spinal cord that lie at the most distal fields of arterial perfusion.
- In the cerebral hemispheres, the border zone between the anterior and the middle cerebral artery distributions is at greatest risk.



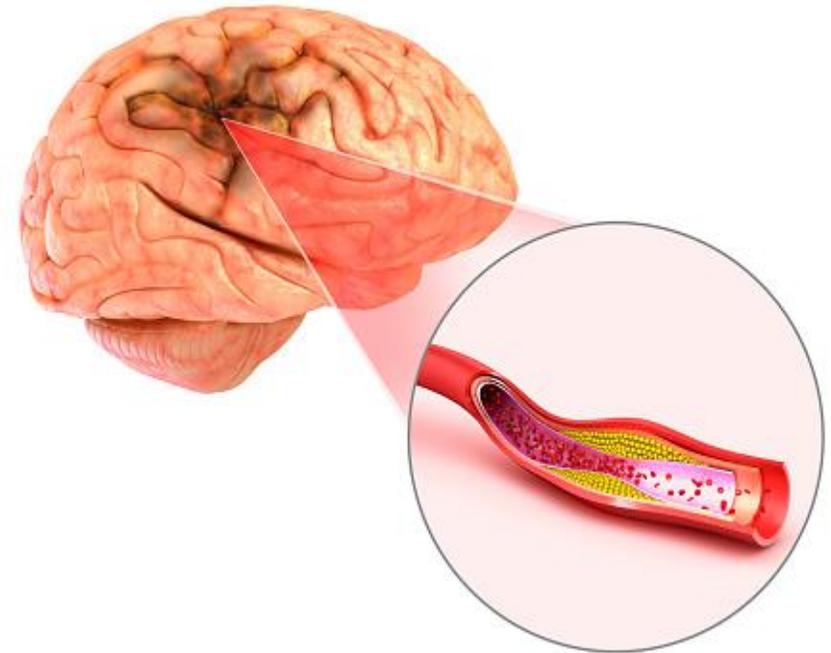
middle cerebral artery



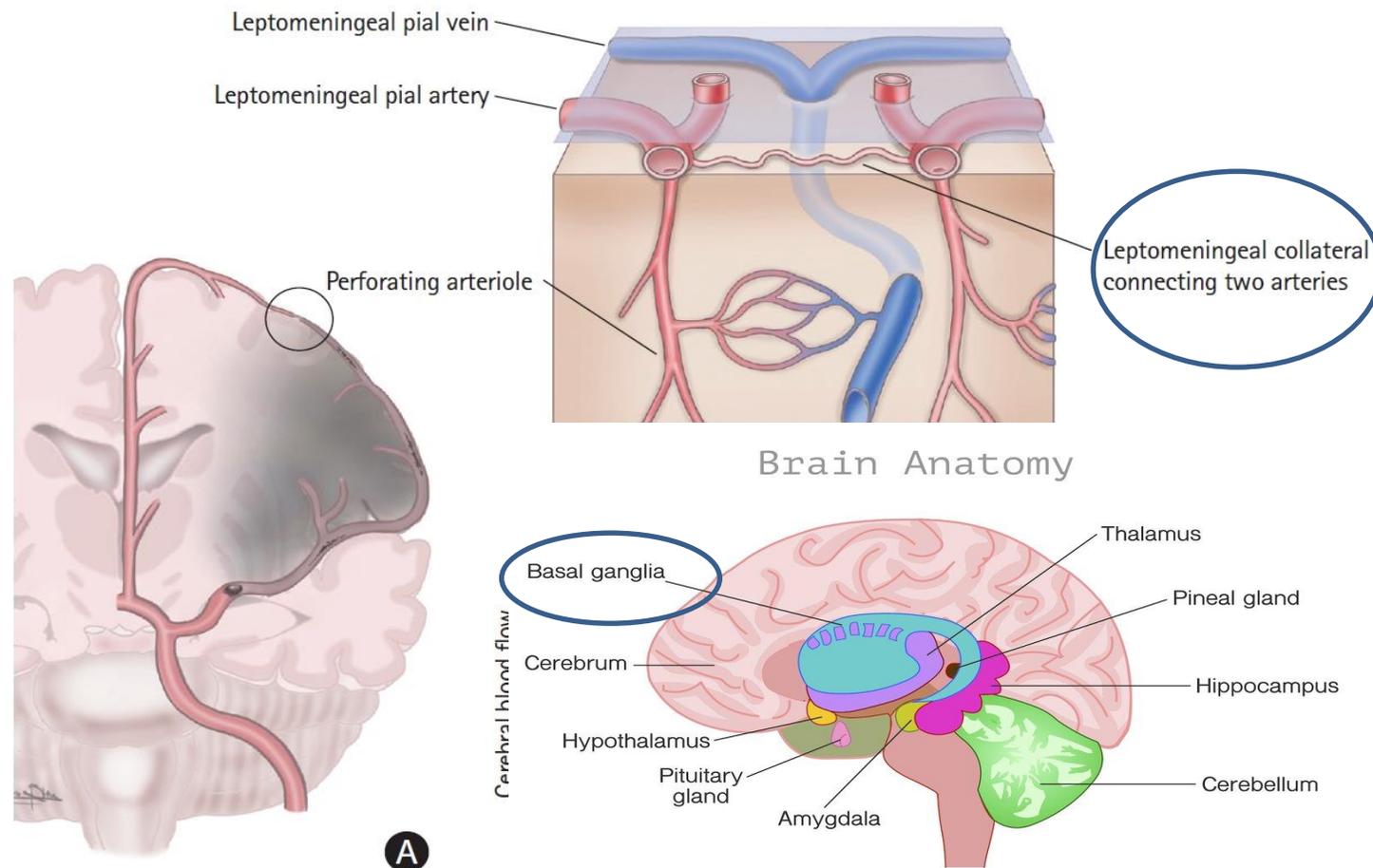
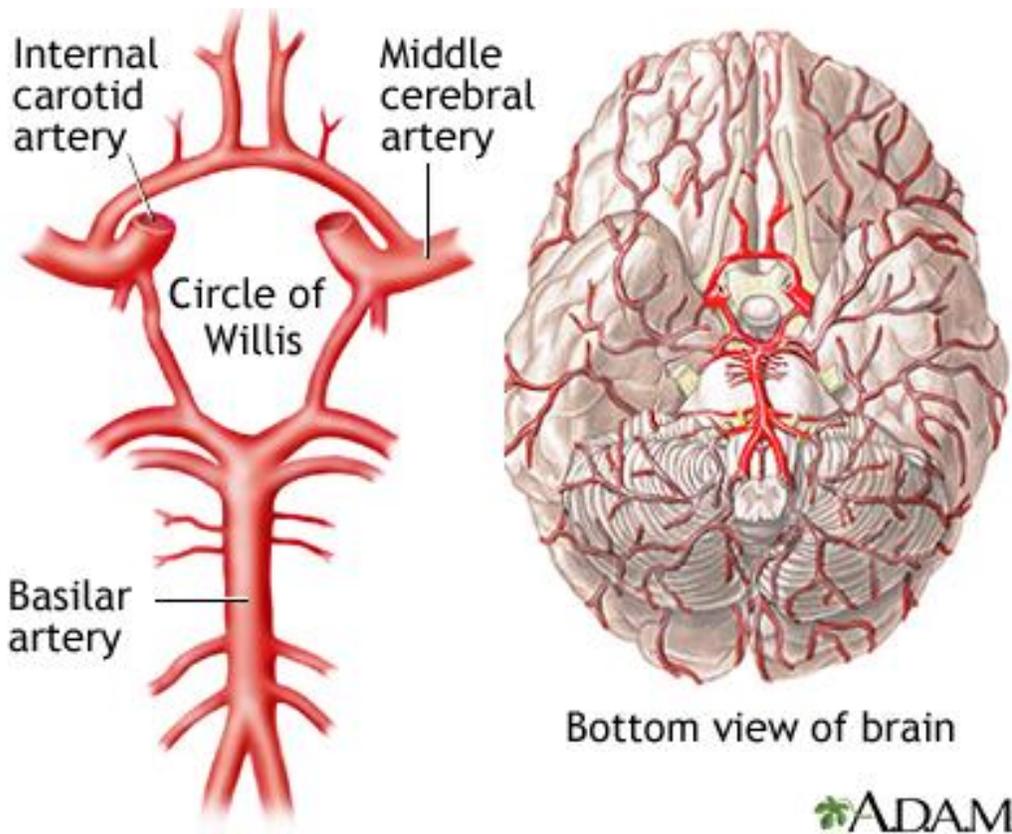
- **Damage to this region produces a band of necrosis over the cerebral convexity a few centimeters lateral to the interhemispheric fissure.**
- **Border zone infarcts are usually seen after hypotensive episodes.**

Focal Cerebral Ischemia

- Cerebral arterial occlusion leads to focal ischemia and-if sustained-to infarction of CNS tissue in the distribution of the compromised vessel.
- The **size, location, and shape** of the infarct and the **extent of tissue damage** that results are determined by modifying variables, most importantly the **adequacy of collateral flow**.



- The major source of collateral flow is the **circle of Willis**.
- Partial collateralization is also provided over the surface of the brain through cortical-leptomeningeal anastomoses.
- In contrast, there is **little if any collateral flow** for the deep penetrating vessels supplying structures such as the **thalamus, basal ganglia, and deep white matter**.

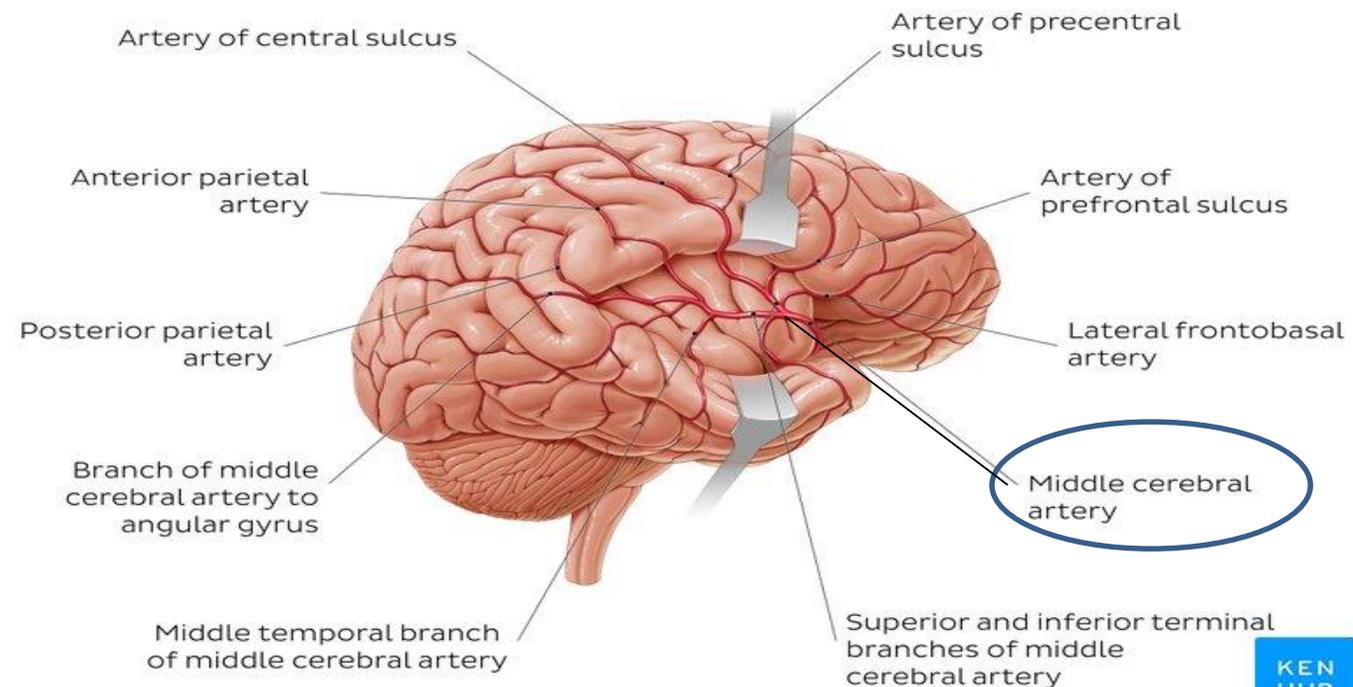


- **Occlusive vascular disease of severity sufficient to lead to cerebral infarction may be due to :**
- ***1-in situ thrombosis***
- ***2-embolization* from a distant source.**
- **Overall, embolic infarctions are more common.**
- **predisposing factors :**
- **1-Cardiac mural thrombi are a frequent source**
- **2-myocardial infarct**
- **3-valvular disease**
- **4-atrial fibrillation**

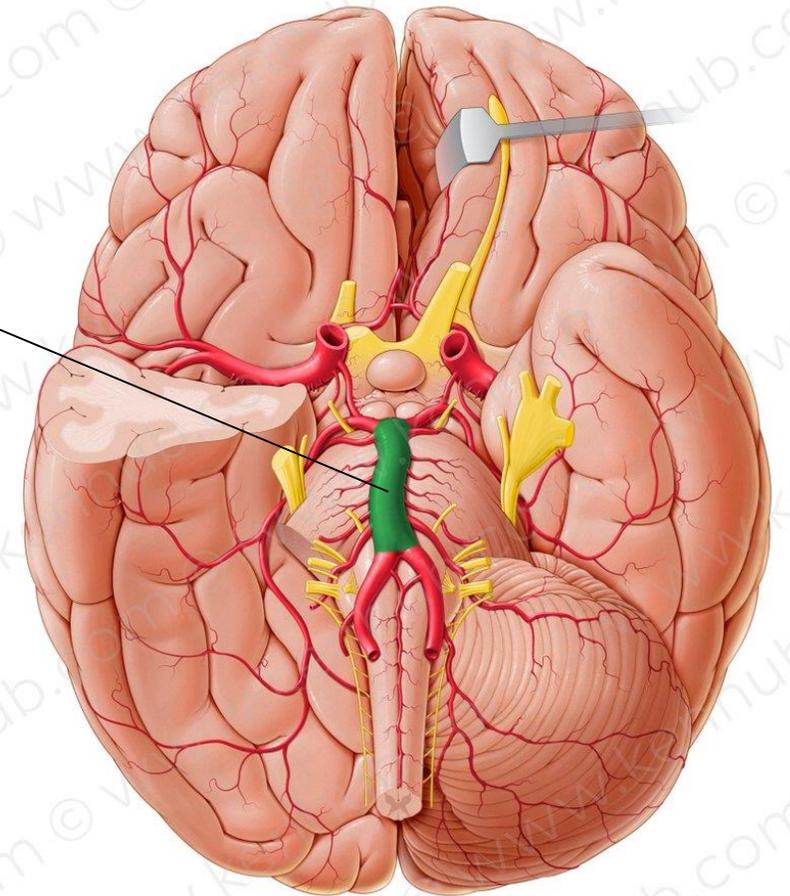
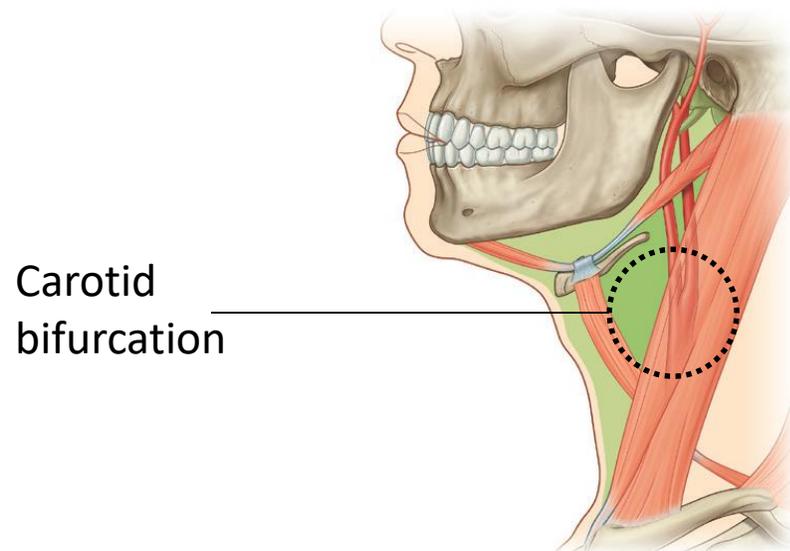
- Thromboemboli arise in:
- 1- atheromatous plaques within the **carotid arteries**.
- 2-**paradoxical emboli** particularly in children with cardiac anomalies
- 3-emboli associated with **cardiac surgery**
- 4-emboli of other material (tumor, fat, or air).

Extra
paradoxical Embolism (PDE) occurs
when a thrombus crosses an
intracardiac defect into the
systemic circulation.

- **Common sites of embolization :**
- **1-The territory of distribution of the middle cerebral artery-the direct extension of the internal carotid artery-is most frequently affected by embolic infarction**
- **2-Emboli tend to lodge where vessels branch or in areas of preexisting luminal stenosis**



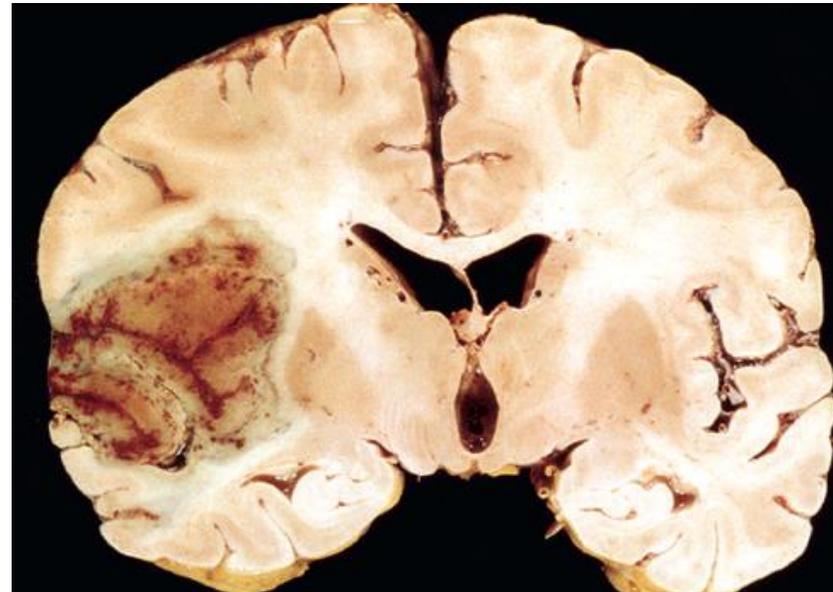
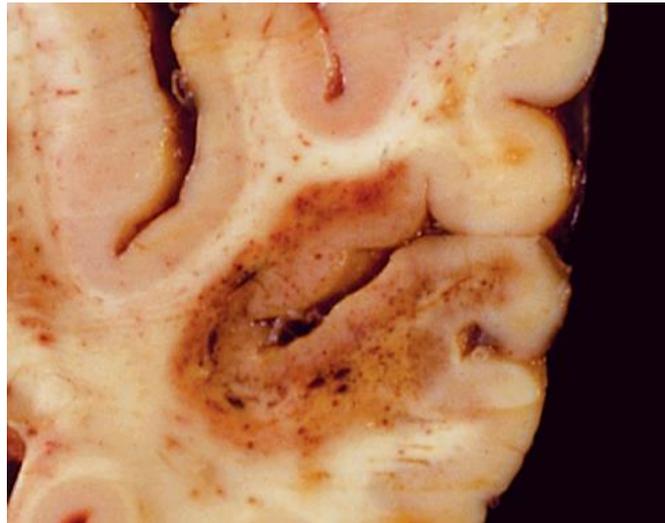
- The **majority of thrombotic** occlusions causing cerebral infarctions are due to ***atherosclerosis***.
- **the most common sites of primary thrombosis are :**
- **1-the carotid bifurcation.**
- **2-the origin of the middle cerebral artery.**
- **3-at either end of the basilar artery.**



- **Atherosclerotic stenoses can develop superimposed thrombosis, accompanied by anterograde extension, fragmentation, and distal embolization**

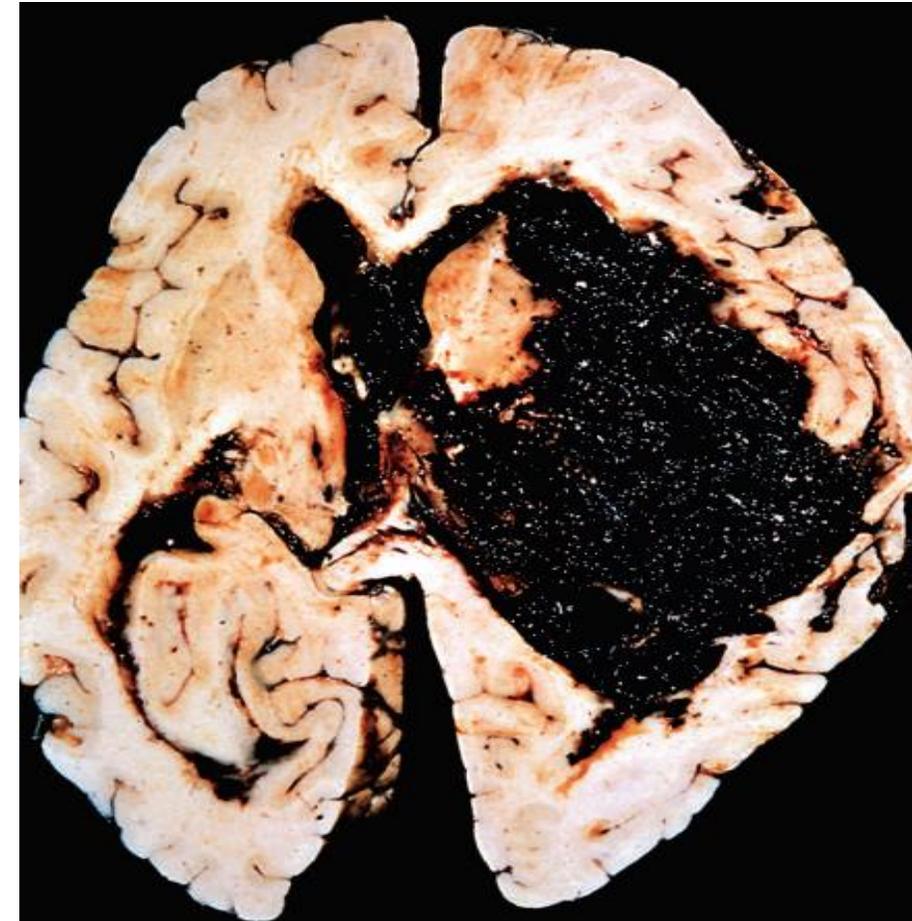
Brain infarction

- Infarcts can be divided into two broad groups based on their macroscopic and corresponding radiologic appearance :
- 1-Nonhemorrhagic infarcts
- can be treated with thrombolytic therapies, if identified shortly after presentation.
- 2-hemorrhagic infarcts
- multiple, sometimes confluent, petechial hemorrhages
- The hemorrhage occurs secondary to reperfusion of ischemic tissue, either through collaterals or after dissolution of intravascular occlusions.

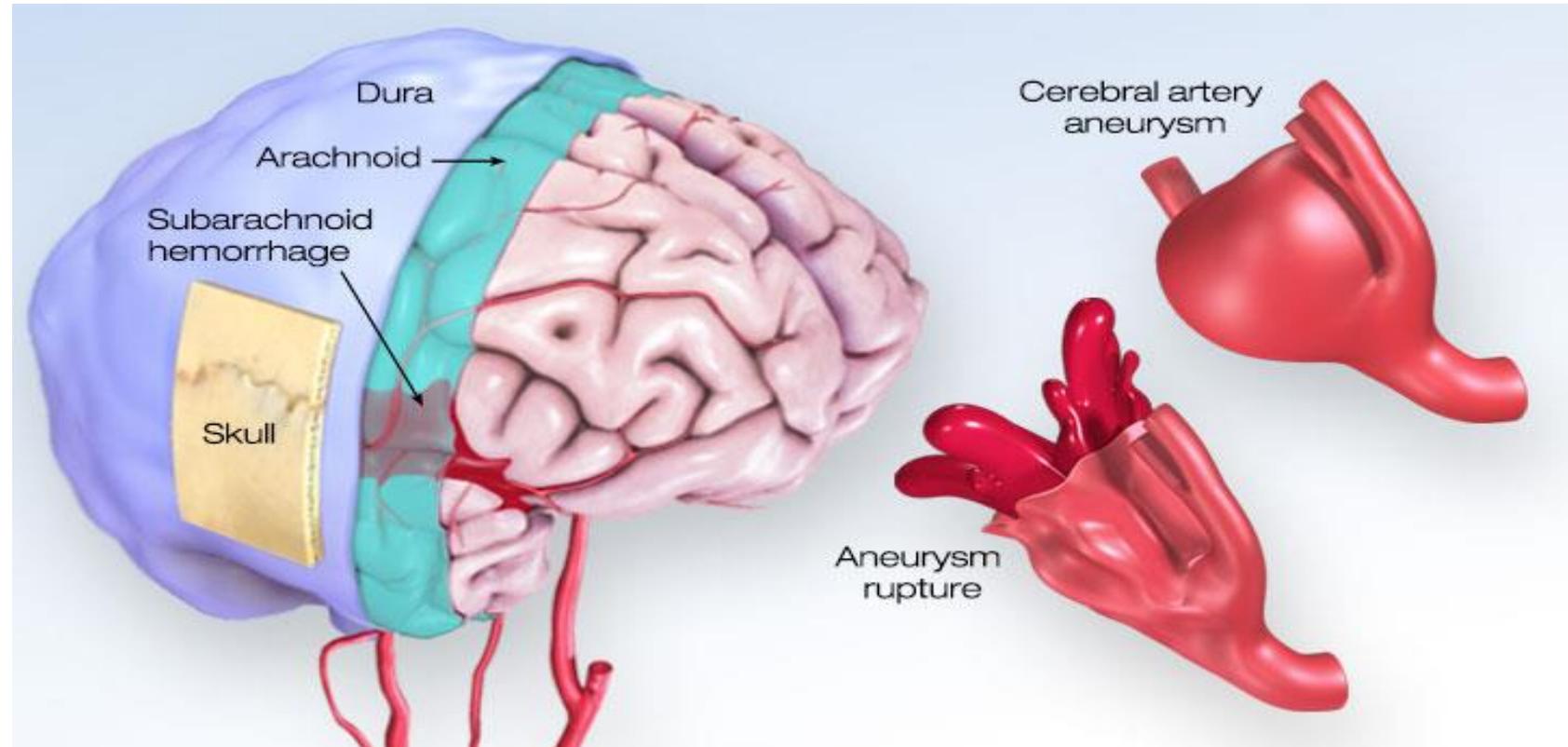


Intracranial Hemorrhage

- Hemorrhage within the skull can occur in a variety of locations, and each location is associated with a set of underlying causes.
- Hemorrhages within the brain itself can occur secondary to :
- 1-hypertension or other forms of vascular wall injury.
- 2-arteriovenous malformation.
- 3-cavernous malformation.
- 4-intraparenchymal tumor.



- **Subarachnoid hemorrhages** are most commonly seen with **aneurysms** but occur also with other vascular malformations.
- Hemorrhages associated with the dura (in subdural or epidural spaces) are associated with trauma.



Primary Brain Parenchymal Hemorrhage

- Spontaneous (nontraumatic) intraparenchymal hemorrhages occur most commonly in **mid to late adult life, with a peak incidence at about 60 years of age.**
- Most are caused by **rupture of a small intraparenchymal vessel.**
- **Hypertension** is the most common underlying cause.
- brain hemorrhage accounts for roughly **15%** of deaths among individuals with chronic hypertension.

- **Hypertensive intraparenchymal hemorrhages typically occur in the :**
- **1-basal ganglia.**
- **2-thalamus.**
- **3-pons.**
- **4-cerebellum .**

Clinical manifestations

- **The location and size of the bleed will determine the clinical manifestations.**
- **Intracerebral hemorrhage can be clinically devastating when it affects large portions of the brain and extends into the ventricular system.**
- **It can affect small regions and be clinically silent.**
- **Over weeks or months there is a gradual resolution of the hematoma, sometimes with considerable clinical improvement.**

Subarachnoid Hemorrhage and Saccular Aneurysms

- Causes :
- 1-rupture of a *saccular (berry) aneurysm*.
The most frequent cause of clinically significant subarachnoid hemorrhage .
- 2 vascular malformation.
- 3 trauma.
- 4 rupture of an intracerebral hemorrhage into the ventricular system.
- 5 hematologic disturbances and tumors.

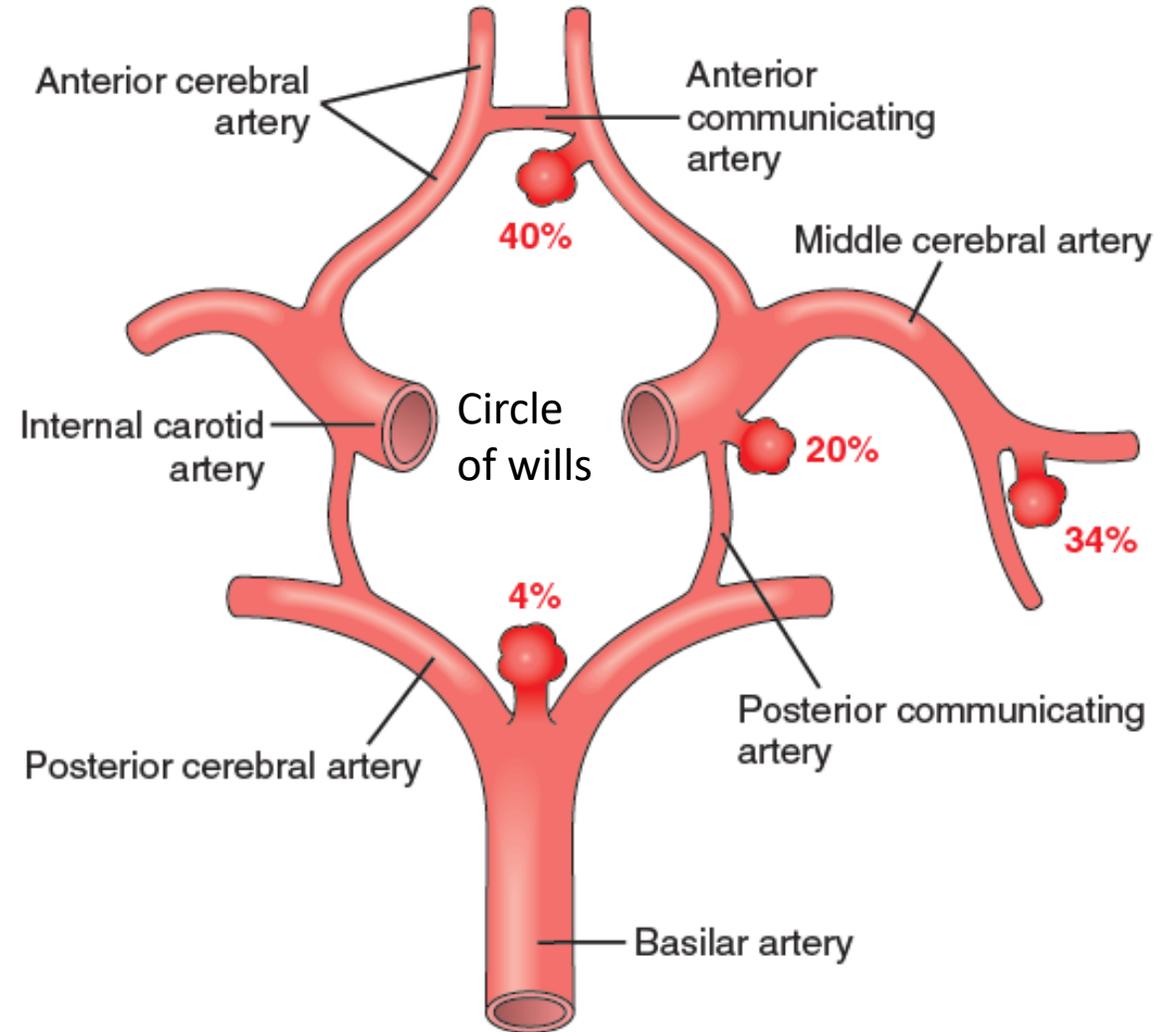
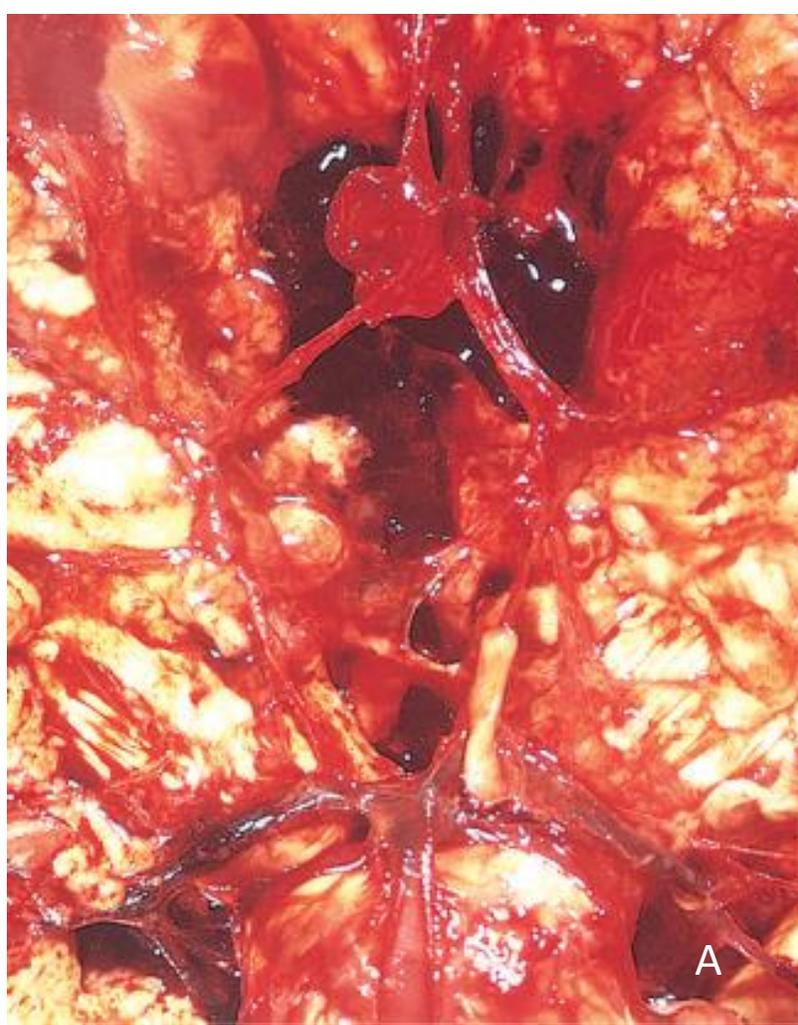


Fig. 23.9 Common sites of saccular aneurysms.



(A) View of the base of the brain, dissected to show the circle of Willis with an aneurysm of the anterior cerebral artery (*arrow*). (B) The circle of Willis is dissected to show a large aneurysm. (C) Section through a saccular aneurysm showing the hyalinized fibrous vessel wall. Hematoxylin-eosin stain.

Clinical manifestations

- Rupture can occur at any time, but in about 1/3 of cases it is associated with acute increases in intracranial pressure (as with straining at stool).
- Blood under arterial pressure is forced into the subarachnoid space, and individuals are stricken with **sudden, excruciating headache** (classically described as "the worst headache I've ever had") and **rapidly lose consciousness**.
- Between 25%-50% of individuals die with the first rupture, although those who survive typically improve and recover consciousness in minutes.

- Recurring bleeding is common in survivors.
- It is currently not possible to predict which individuals will have recurrences of bleeding.
- Aneurysms have a roughly 1.3% per year rate of bleeding.
- the **probability of rupture** increases with the **size** of the lesion, such that aneurysms greater than 10 mm have a roughly 50% risk of bleeding per year.
- The prognosis worsens with each episode of bleeding.

- About 90% of saccular aneurysms occur in the anterior circulation near major arterial branch points.
- **multiple aneurysms** exist in 20% to 30% of cases.
- Although they are sometimes referred to as *congenital*, **they are not present at birth** but develop over time because of underlying defects in the vessel media.

- **In the early period after a subarachnoid hemorrhage, there is a risk of additional ischemic injury from vasospasm involving other vessels.**
- **In the healing phase of subarachnoid hemorrhage, meningeal fibrosis and scarring occur, sometimes leading to obstruction of CSF flow as well as interruption of the normal pathways of CSF resorption.**

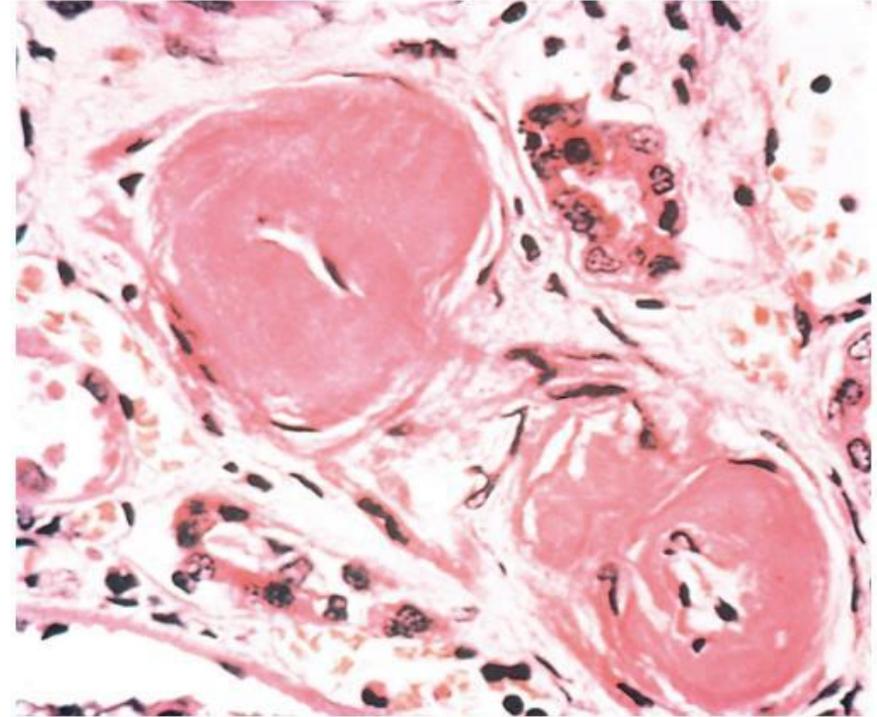
Hypertensive Cerebrovascular Disease

- **The most important effects of hypertension on the brain include:**
- **1- massive hypertensive intracerebral hemorrhage**
- **2- lacunar infarcts.**
- **3- slit hemorrhages.**
- **4- hypertensive encephalopathy.**

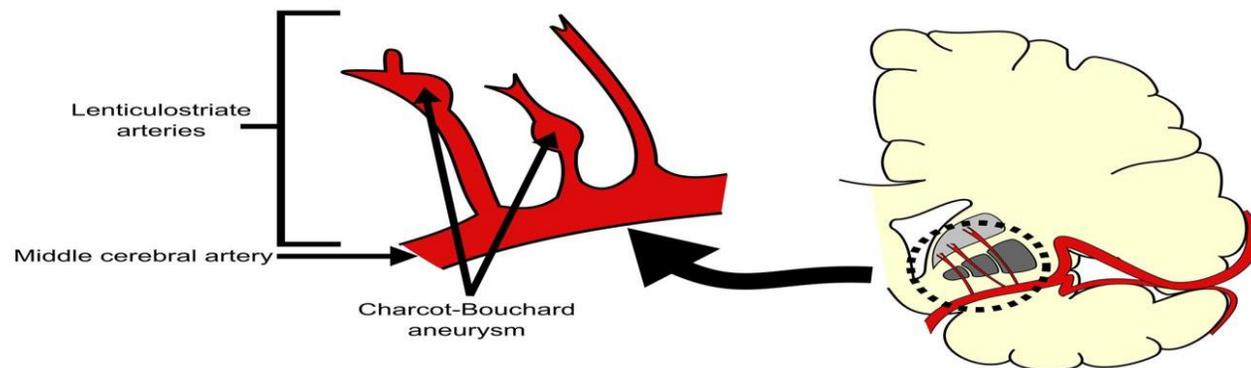
- Hypertension affects the deep penetrating arteries and arterioles that supply the basal ganglia and hemispheric **white matter** as well as the **brain stem**.

- Hypertension causes several changes including:
- 1- hyaline arteriolar sclerosis in arterioles. Arteriolar walls affected by hyaline change are weaker than are normal vessels and are more vulnerable to rupture.
- 2-development of minute aneurysms in vessels that are less than 300 μm in diameter(*Charcot-Bouchard microaneurysms*) which can rupture.

Hyaline arteriolosclerosis

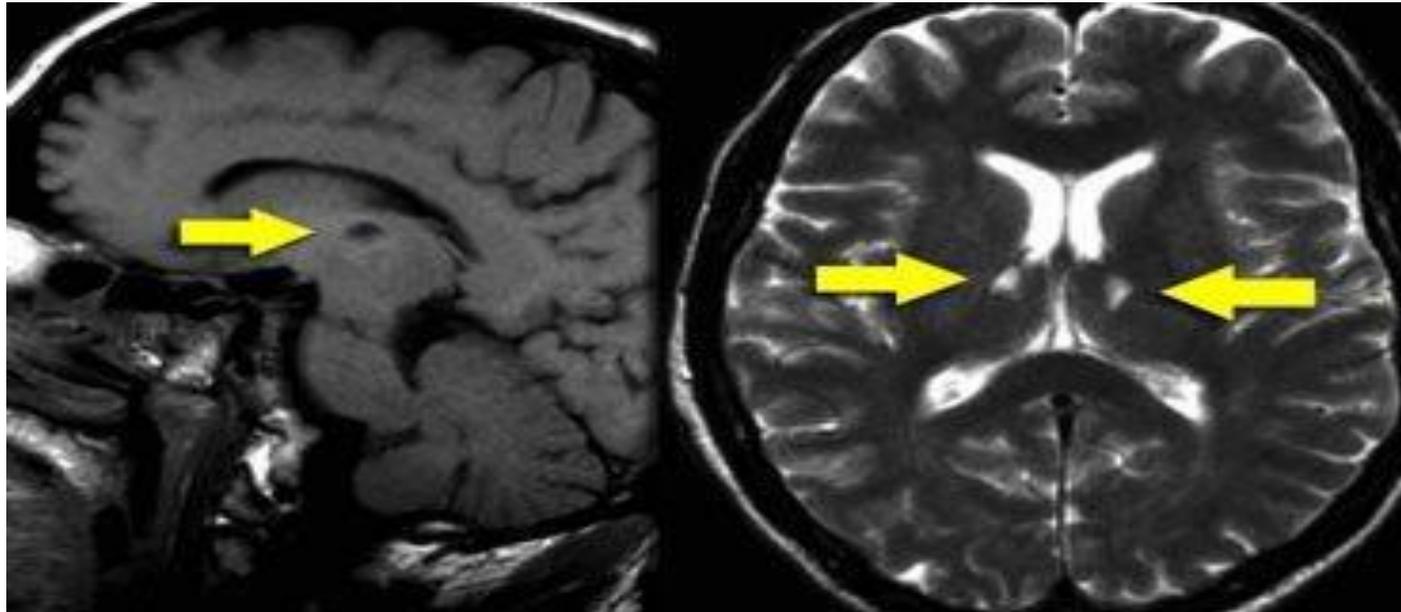


Charcot-Bouchard Aneurysm



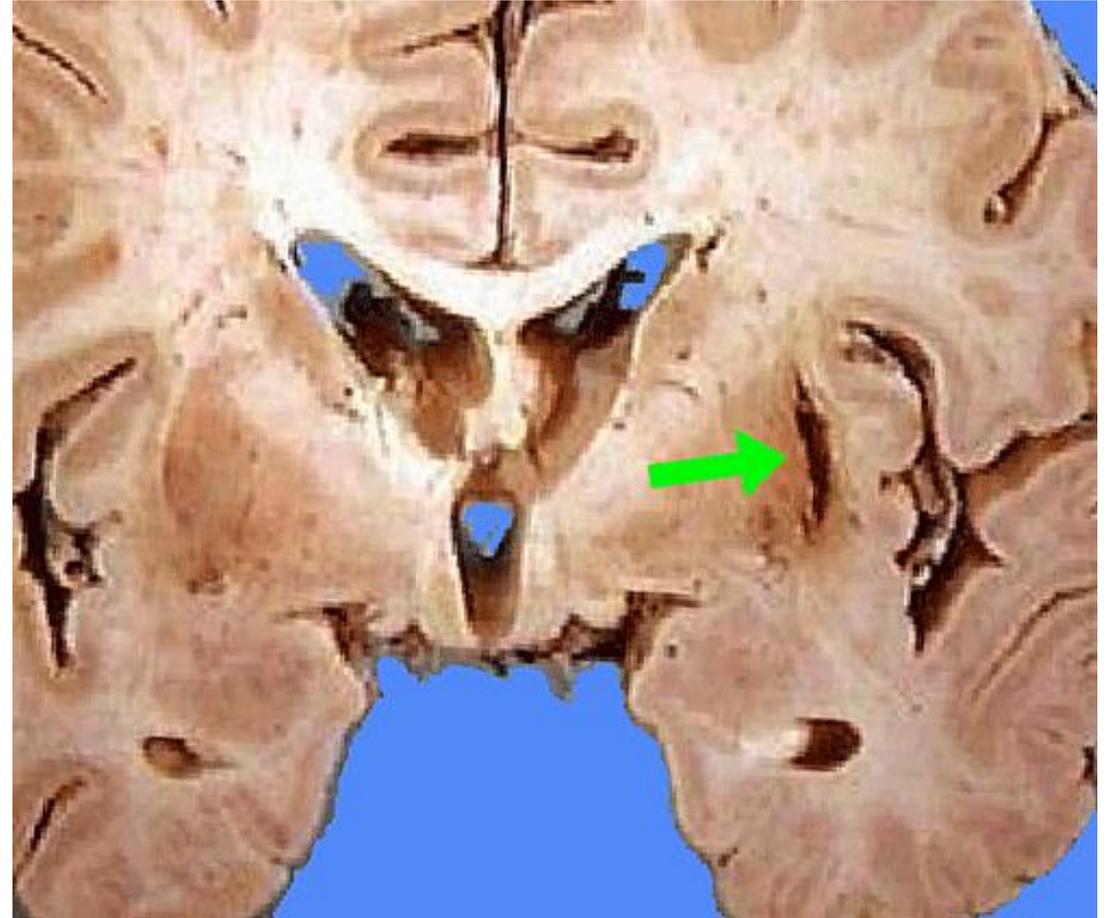
Lacunar infarcts

- An important clinical and pathologic outcome of arteriolar sclerosis is the development of *lacunes* or *lacunar infarcts*.
- These small cavitory infarcts are just a few millimeters wide (<15 mm).
- They are found most commonly in deep gray matter (basal ganglia and thalamus), internal capsule, deep white matter, and pons,
- Depending on their location in the CNS, lacunes can either be clinically silent or cause significant neurologic impairment.



Slit hemorrhages

- Hypertension also gives rise to rupture of the small-caliber penetrating vessels and the development of small hemorrhages.
- In time, these hemorrhages resorb, leaving behind a slitlike cavity (*slit hemorrhage*) surrounded by brownish discoloration .



Hypertensive encephalopathy

- **Acute hypertensive encephalopathy is a clinicopathologic syndrome characterized by diffuse cerebral dysfunction including:**
 - **1- headaches.**
 - **2- confusion.**
 - **3- vomiting.**
 - **4- convulsions, sometimes leading to coma.**

- **Rapid therapeutic intervention to reduce the accompanying increased intracranial pressure is required, since the syndrome does not usually remit spontaneously.**