

CENTRAL NERVOUS SYSTEM

SUBJECT : patho

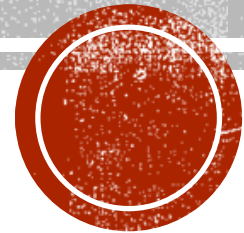
LEC NO. : 4

DONE BY : sara Nafi'

CNS LECTURE 4

CEREBROVASCULAR DISEASES

Dr. Dua Abuquteish



SUBARACHNOID HEMORRHAGE

Causes:

- ❑ Rupture saccular (berry) aneurysm
- ❑ **Vascular malformations**
- ❑ Trauma
- ❑ Coagulopathies
- ❑ Tumors



VASCULAR MALFORMATIONS

Classified into four principal types:

vascular malformations:

يمكن تعمل subarachnoid hemorrhage وكمات
يمكن تعمل intraparenchymal hemorrhage
بتعتمد وين موجود ال vascular malformation

↳ the most common

risk of hemorrhage
عالي

■ **Arteriovenous malformations (AVMs)**

■ **Cavernous malformations** → abnormal perforation of the vessels

risk of hemorrhage
قليل

■ **Capillary telangiectasias** within the capillaries أشياء صغيرة كثير

■ **Venous angiomas** زي مبدأ الدوالي

■ AVMs and Cavernous malformations are the types associated with risk of hemorrhage and development of neurological symptoms.

■ Capillary telangiectasias and venous angiomas are unlikely to bleed or to cause symptoms, and most are incidental findings



ARTERIOVENOUS MALFORMATIONS (AVM)

↳ +The most dangerous

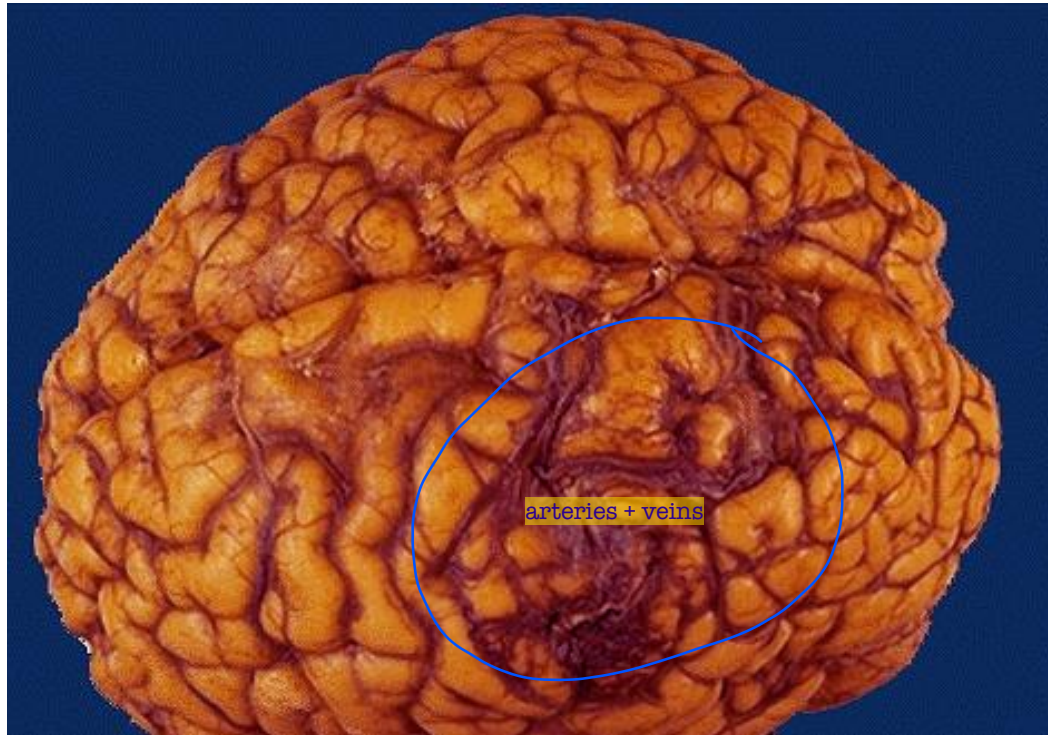
it's dangerous because the pressure of artery is putted direct inside the vein so the risk of rupture is high

- ❑ The most common type
- ❑ More common in Males
- ❑ Common age (10 and 30 years)
- ❑ May involve subarachnoid vessels extending into brain parenchyma or occur exclusively within the brain
- ❑ Can cause intracerebral hemorrhage, or a subarachnoid hemorrhage, and neurological symptoms (seizures)
- ❑ The risk for bleeding makes **AVM the most dangerous type of vascular malformation.**
- ❑ Multiple AVMs can be seen in the setting of hereditary hemorrhagic telangiectasia (AD disease)



ARTERIOVENOUS MALFORMATIONS (AVM)

كيف أشخص أي vascular prblem في الجسم؟
يستخدم angiogram
أو ct scan أو MRI



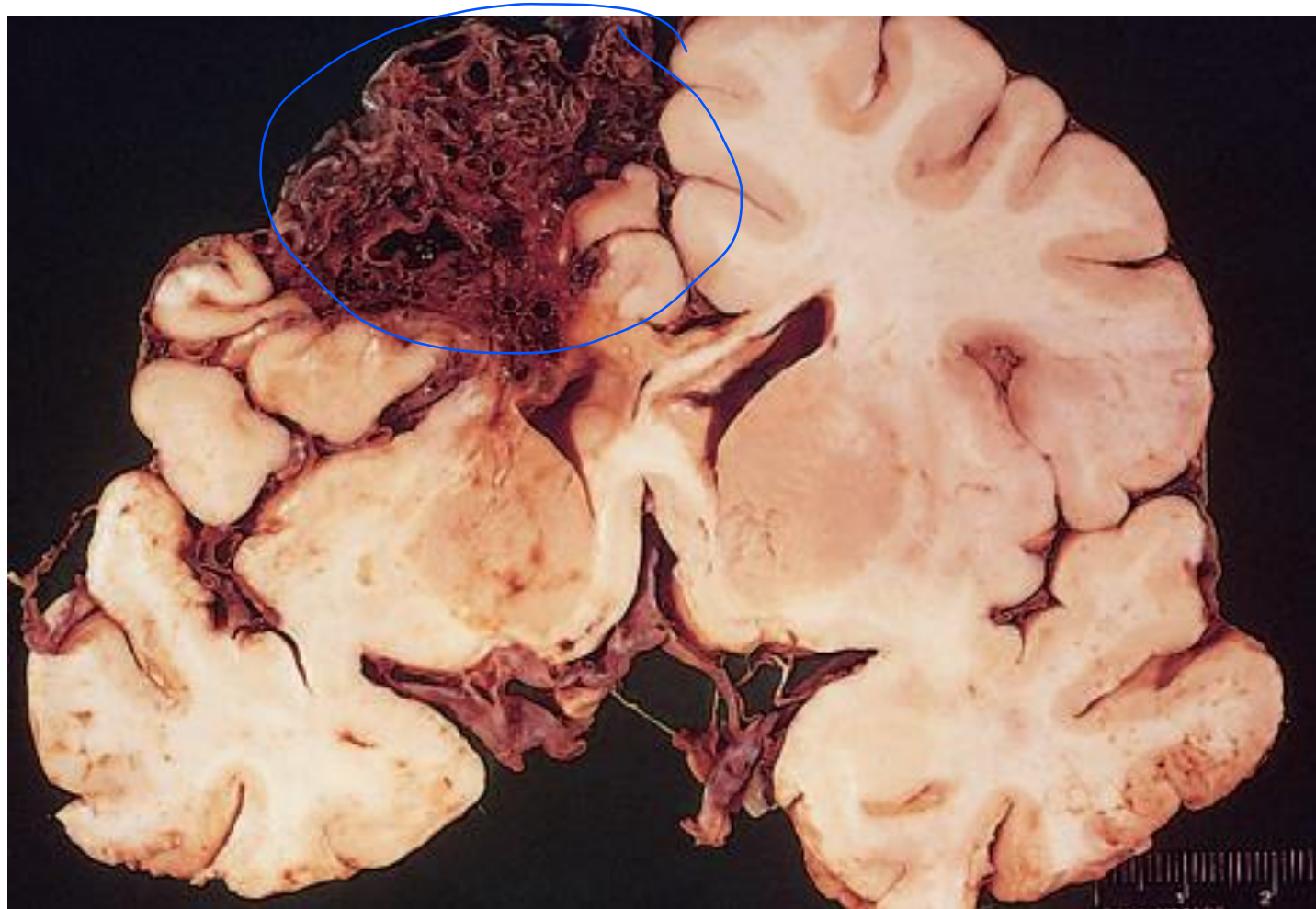
Morphology: a mass of irregular, tortuous vessels (wormlike-vascular channels)



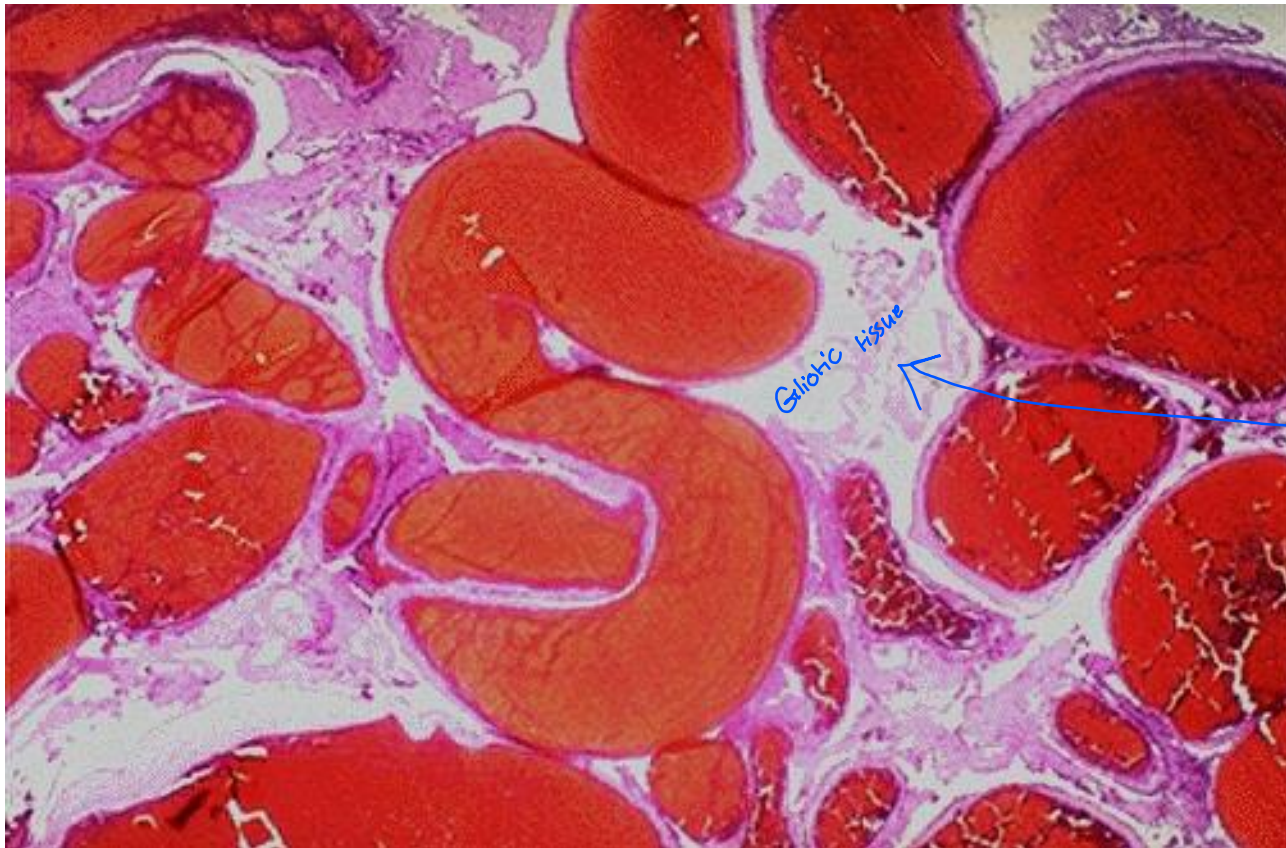
This angiogram demonstrates a tortuous collection of irregular small vessels



ARTERIOVENOUS MALFORMATIONS (AVM)



ARTERIOVENOUS MALFORMATIONS (AVM)



Histology: dilated, tortuous, worm-like vascular channels filled with blood (and appearing red). The vessels are separated by gliotic tissue.

حكت هي المعلومة مهمة لإنها راح
تميزها عن الcavernous
hemangioma

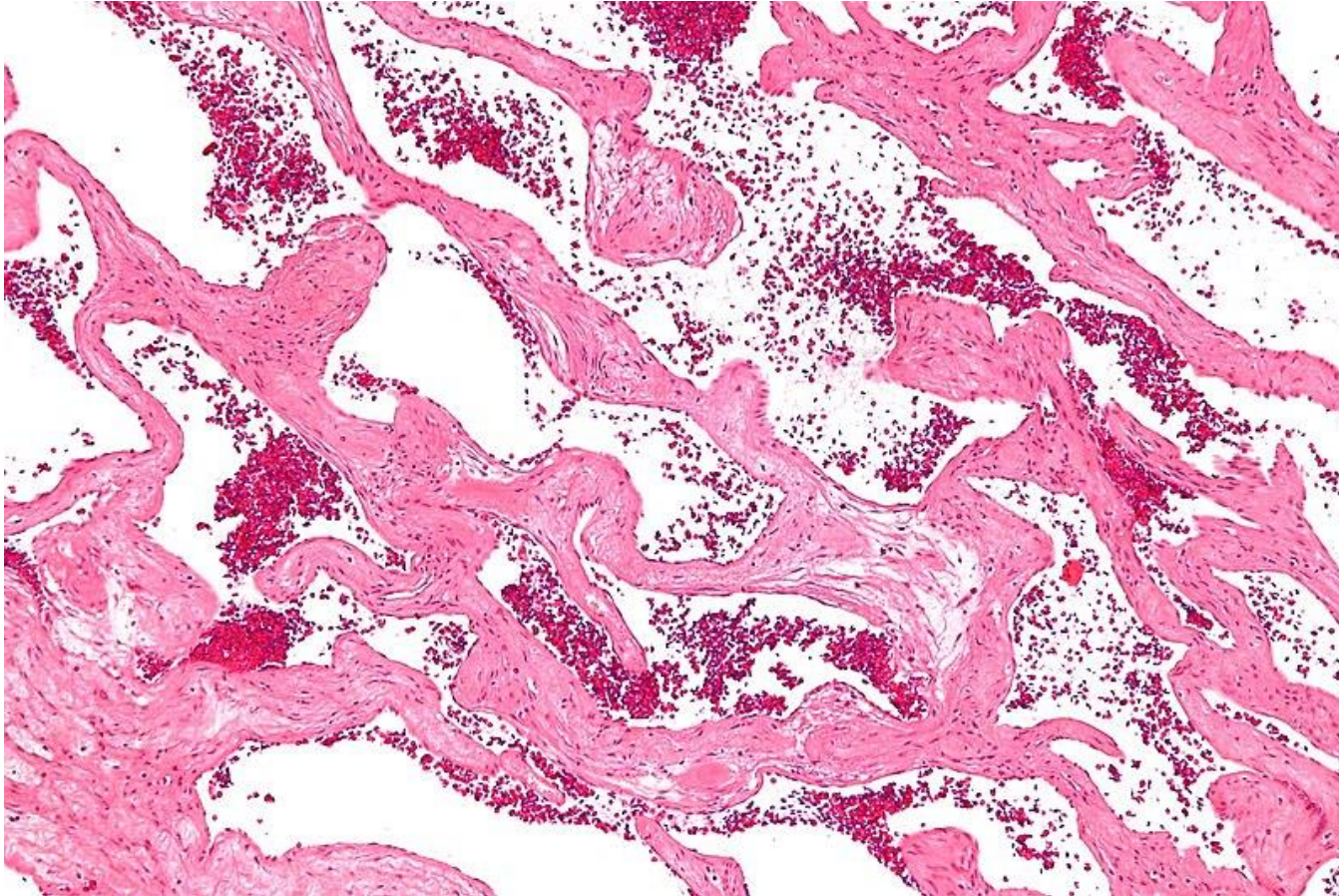
Such lesions may bleed a small amount and be the cause for a seizure disorder.



فيها bleeding بس أقل
من ال AVM

CAVERNOUS MALFORMATIONS

ال mass عاملة connections مع بعضها البعض ال veins وال arteries
وما في بين ال arteries أي pass for channels



- Dilated thin-walled vascular channels **devoid of intervening brain tissue.**

- They occur most often in the **cerebellum, pons, and subcortical regions,** and have a low blood flow without significant arteriovenous shunting

ما فيها كثير significant pressure زي ال AVM
لانه مافي blood of arteries بتروح عال veins



CAPILLARY TELANGIECTASIAS AND VENOUS ANGIOMAS (VARICES)

- **Capillary telangiectasias** are microscopic foci of dilated thin-walled vascular channels separated by relatively normal brain parenchyma that occur most frequently in the pons.
- **Venous angiomas (varices)** consist of aggregates of ectatic venous channels.



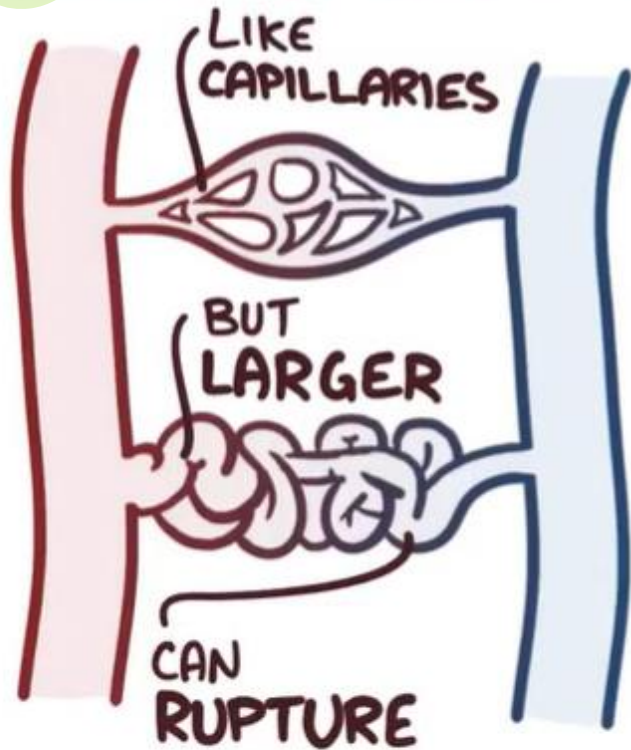
Table 23.1 Patterns of Vascular Injury in the Central Nervous System

Location	Etiology	Additional Features
Epidural space	Trauma	Usually associated with a skull fracture (in adults); rapidly evolving neurologic symptoms requiring intervention
Subdural space	Trauma	Level of trauma may be mild; slowly evolving neurologic symptoms, often with a delay from the time of injury
Subarachnoid space	Vascular abnormalities (arteriovenous malformation or aneurysm) Trauma	Sudden onset of severe headache, often with rapid neurologic deterioration; secondary injury may emerge due to vasospasm Typically associated with underlying contusions
* Intraparenchymal	Trauma (contusions)	Selective involvement of the crests of gyri where the brain contacts the skull (frontal and temporal tips, orbitofrontal surface)
	Hemorrhagic conversion of an ischemic infarction	Petechial hemorrhages in an area of previously ischemic brain, usually following the cortical ribbon
	Cerebral amyloid angiopathy	“Lobar” hemorrhage, involving cerebral cortex, often with extension into the subarachnoid space
	* Hypertension	<u>Centered in the deep white matter, thalamus, basal ganglia, or brain stem; may extend into the ventricular system</u>
	Tumors (primary or metastatic)	Associated with high-grade gliomas or certain metastases (melanoma, choriocarcinoma, renal cell carcinoma)

Most common cause of intraparenchymal bleeding



① ARTERIOVENOUS MALFORMATIONS



- other causes of intraparenchymal hemorrhage :

AND ALSO

* VASCULITIS ②

↳ INFLAMMATION OF BLOOD VESSEL WALLS

* VASCULAR TUMORS ③

↳ e.g. HEMANGIOMA

* CEREBRAL AMYLOID ANGIOPATHY ④

↳ PROTEIN DEPOSITS IN WALLS OF ARTERIOLES



CEREBRAL AMYLOID ANGIOPATHY AND VASCULITIS

أهم معلومة انها associated مع Alzheimer disease

↑ **Cerebral amyloid angiopathy (CAA):** AB amyloid plaques (Alzheimer disease) deposit in the walls of medium- and small-caliber **meningeal and cortical vessels**.

- Amyloid deposition weakens vessel walls and increases the risk for hemorrhages
- CAA-associated hemorrhages often occur in the lobes of the cerebral cortex (**lobar hemorrhages**)

ثاني أهم معلومة

Vasculitis: inflammation and destruction of vessel walls >> compromise blood flow

- (Causes: infections; syphilis, aspergillosis, herpes zoster, or CMV, or systemic vasculitis; polyarteritis nodosa PAN)



HYPERTENSIVE CEREBROVASCULAR DISEASE



HYPERTENSIVE CEREBROVASCULAR DISEASE

- Is the commonest cause of intraparenchymal hemorrhage (50%)
- Accounts for approx. 15% of deaths among patients with chronic hypertension
- Intracerebral hemorrhage can affect large portions of the brain (1) (devastating), or extends into the ventricular system; (2) It can affect small regions (3) and be (clinically silent).

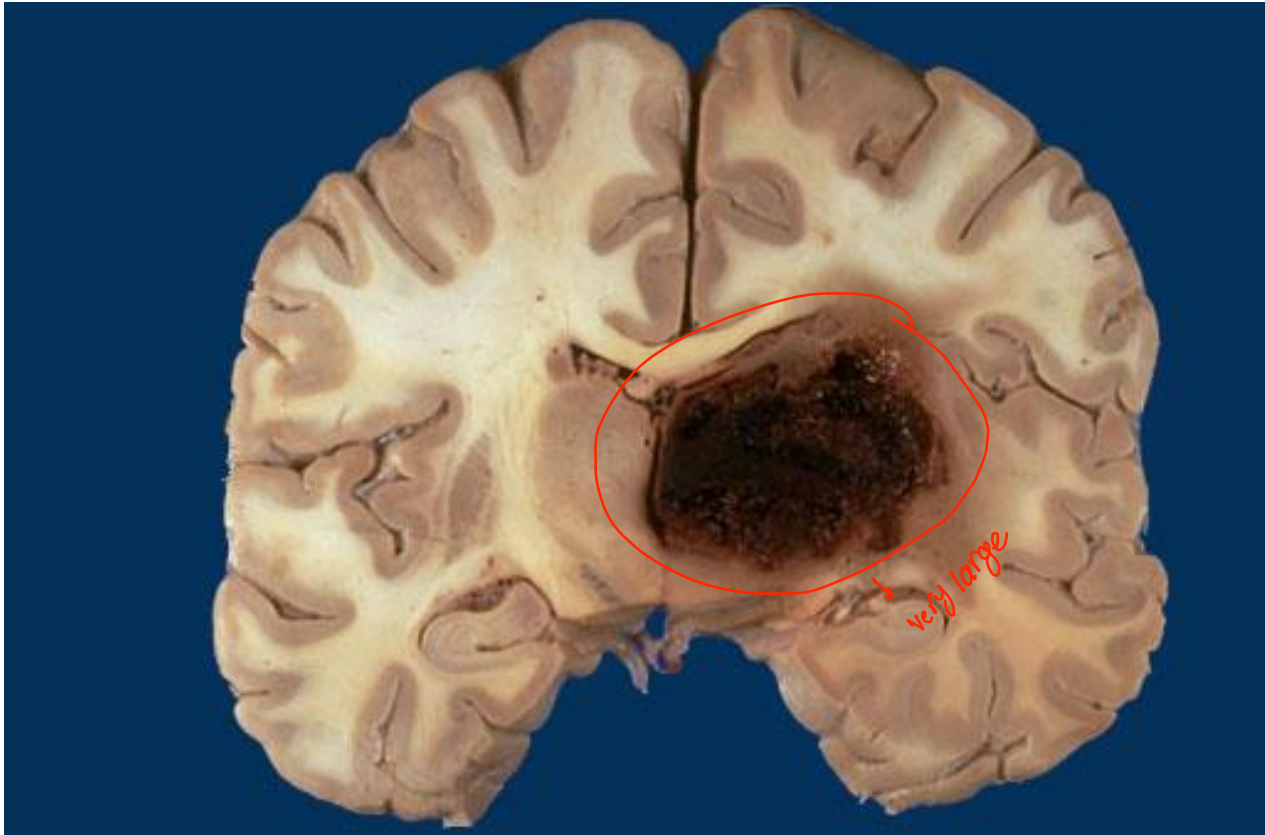
* كثير مهم

- Typically occur in the **basal ganglia (mainly putamen), thalamus, pons, and cerebellum.**
- If patients survives the acute event, gradual resolution of the hematoma ensues, with clinical improvement.

إذا كان ال hemorrhage قليل ممكن يصير resolution ويعيش المريض
ومرات بصير death نتيجة ال neurological symptoms



HYPERTENSIVE CEREBROVASCULAR DISEASE

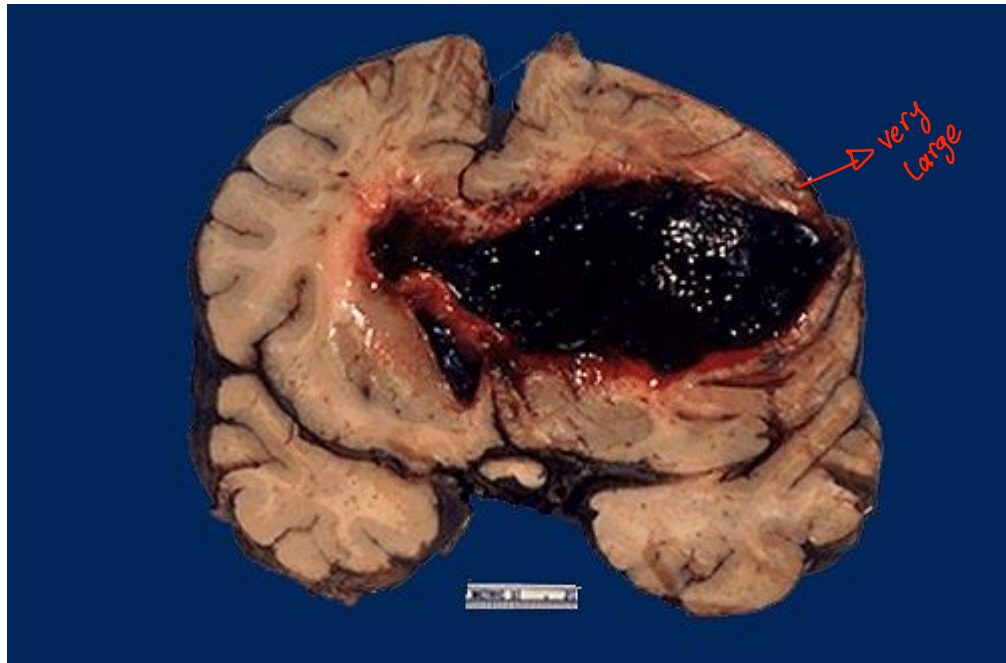


Hemorrhages involving the basal ganglia area (**the putamen in particular**) are usually caused by hypertension, which damages and weakens the small penetrating arteries.

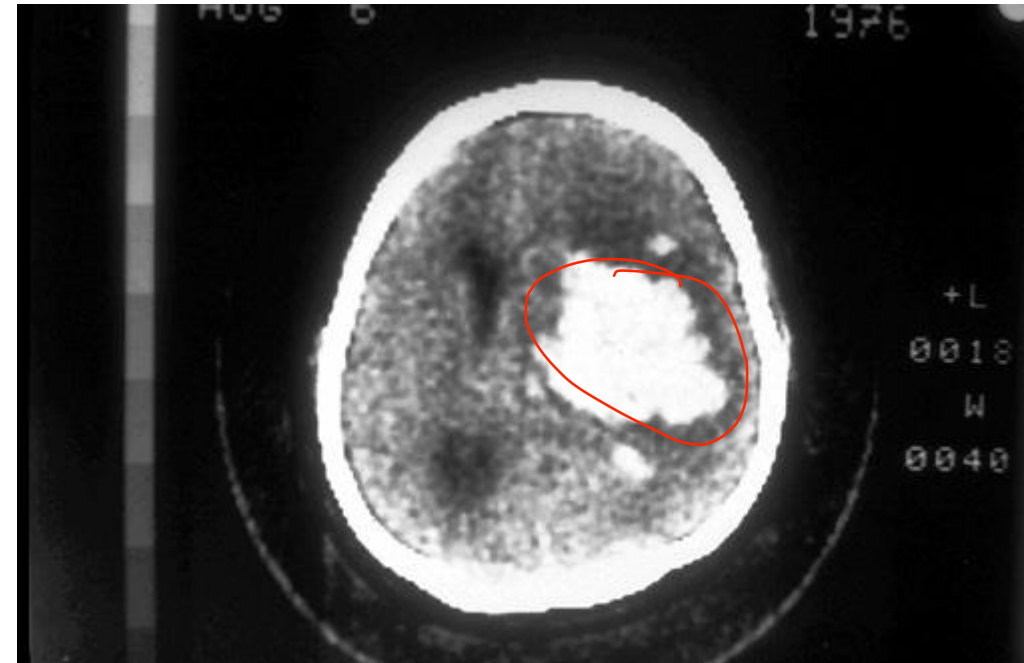
A mass effect with midline shift, often with secondary edema, may lead to herniation.



HYPERTENSIVE CEREBROVASCULAR DISEASE



The large hemorrhage in this adult brain arose in the basal ganglia region of a patient with hypertension. This is one cause for a "stroke".



This computed tomographic (CT) scan of the head demonstrates an area of hemorrhage in a patient with a history of hypertension.



HYPERTENSIVE CEREBROVASCULAR DISEASE

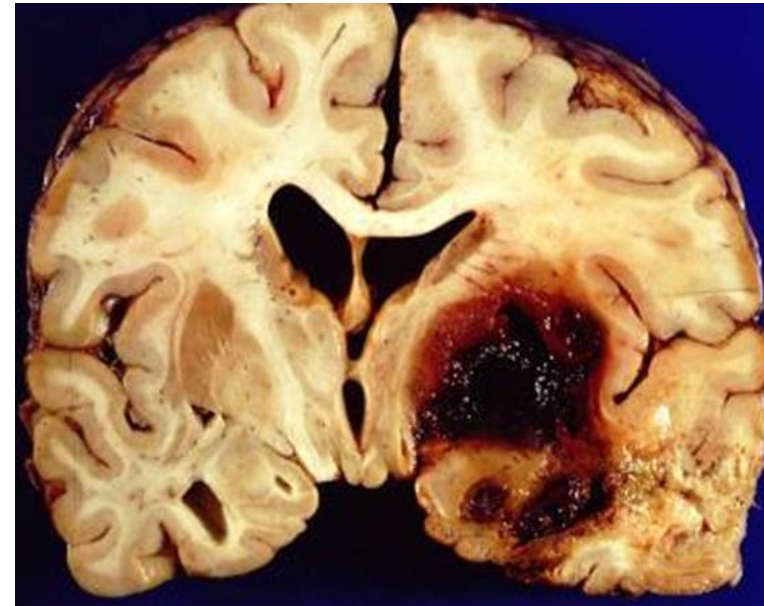
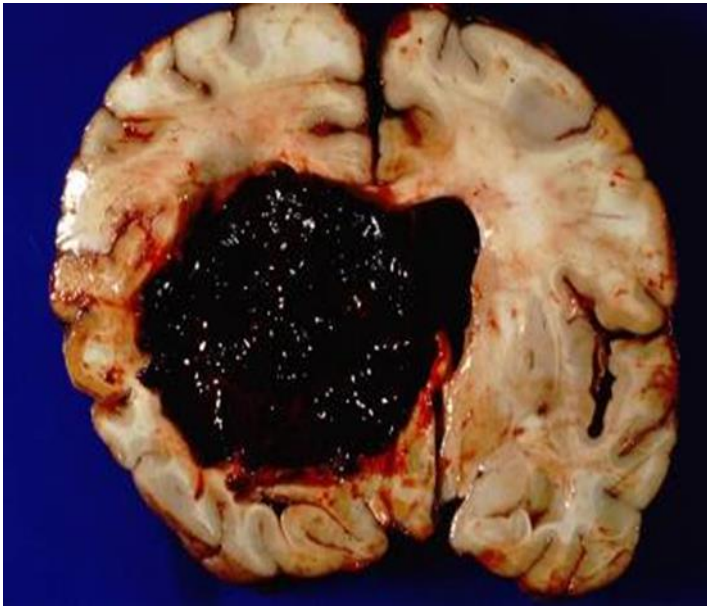
Intracerebral hemorrhage in hypertension:

Acute: Extravasation of blood with compression of adjacent parenchyma

Old: Cavitary destruction of brain with a rim of brown discoloration.



resolution of blood



HYPERTENSIVE CEREBROVASCULAR DISEASE

- Hypertension causes ^{→ due to hypertension or DM} hyaline arteriolar sclerosis of the deep penetrating arteries and arterioles.
- Affected arteriolar walls are weakened and are more vulnerable to rupture.

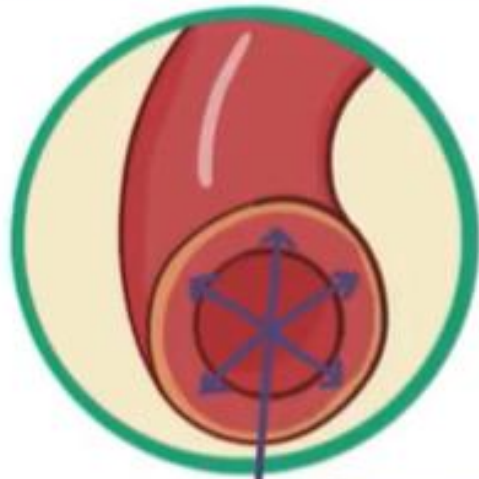
Other vascular lesions in HTN:

- Atherosclerosis in larger arteries
- Arteriolosclerosis in vessels $\leq 150 \mu\text{m}$ → **lacunar infarcts** very tiny small vessels
- Minute aneurysms (Charcot-Bouchard microaneurysms) form in vessels ($< 300 \mu\text{m}$ in diameter)
- Rupture of the small-caliber penetrating vessels → Slit-like hemorrhage (slit-like cavity after resorption)



HYPERTENSIVE CEREBROVASCULAR DISEASE

HYALINE ARTERIOLOSCLEROSIS

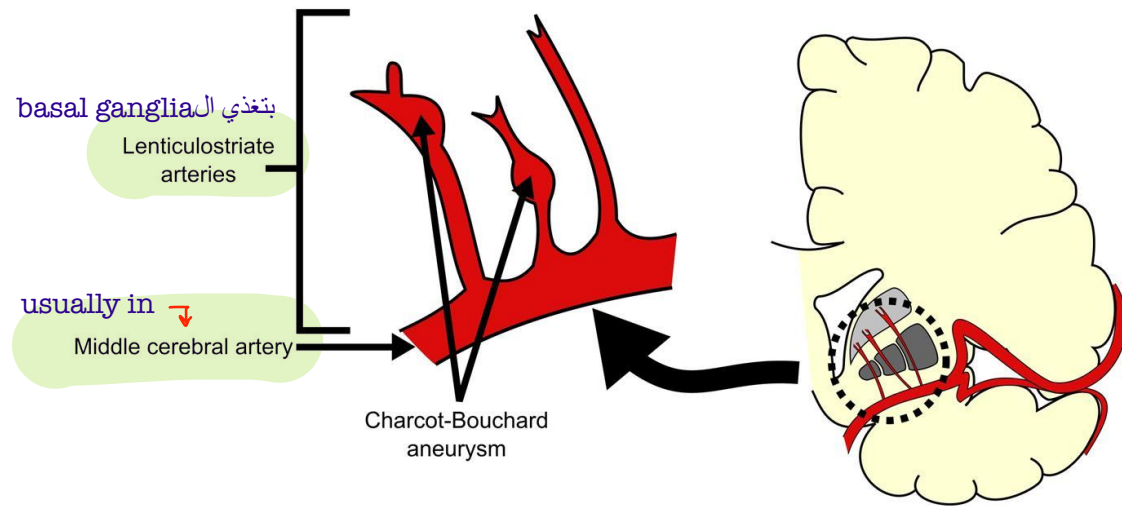


PROTEINS

بترسب جوا ال walls

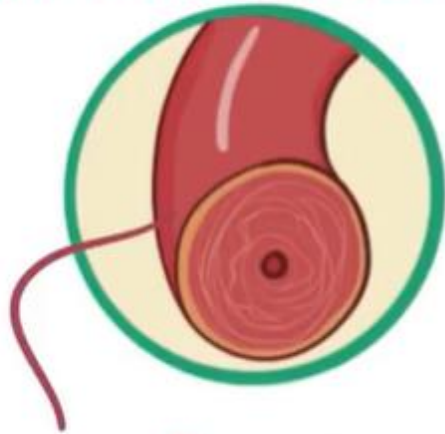
due to increased hydrostatic pressure in vessels

Charcot-Bouchard Aneurysm



HYPERTENSION (HIGH BLOOD PRESSURE)

HYALINE ARTERIOLOSCLEROSIS



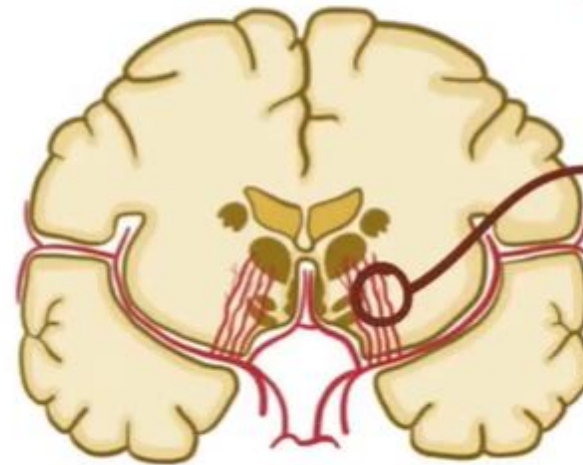
STIFF & BRITTLE
AND MORE
LIKELY to RUPTURE

MICROANEURYSMS

CHARCOT-BOUCHARD
ANEURYSMS



SMALL ARTERIES



LENTICULOSTRIATE
VESSELS

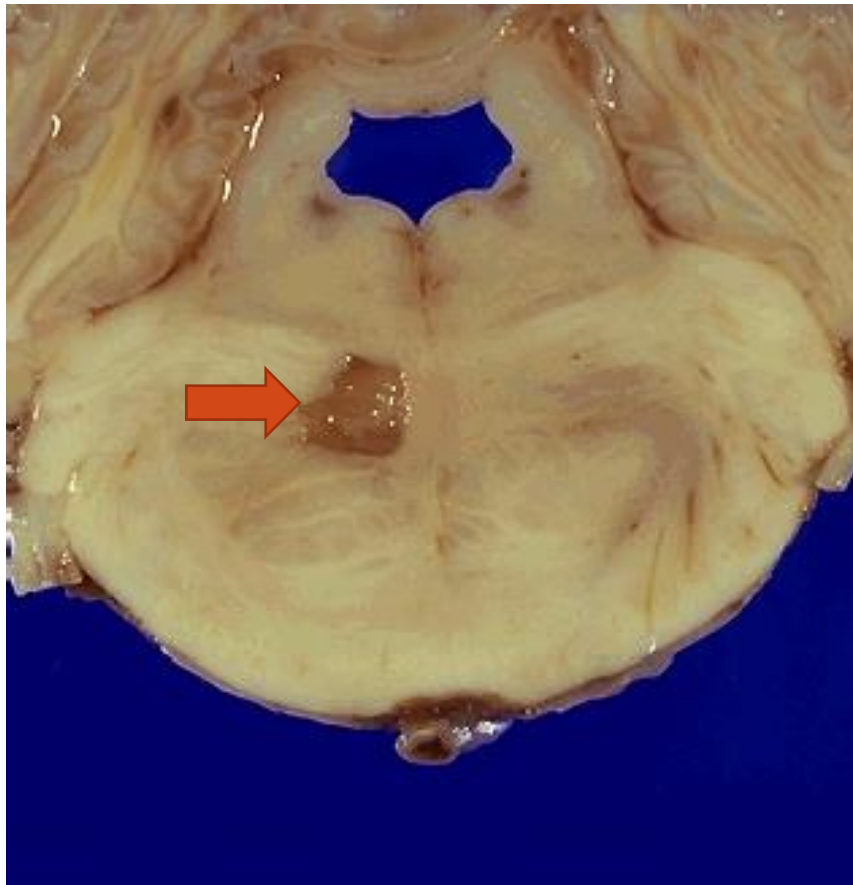
Supply the

BASAL GANGLIA

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HYPERTENSIVE CEREBROVASCULAR DISEASE



The arteriolar sclerosis that results from chronic hypertension leads to small **lacunar infarcts, or "lacunes"**. ^{Cyst Like space}

A remote, small lacunar infarct is seen here in the pons "arrow"

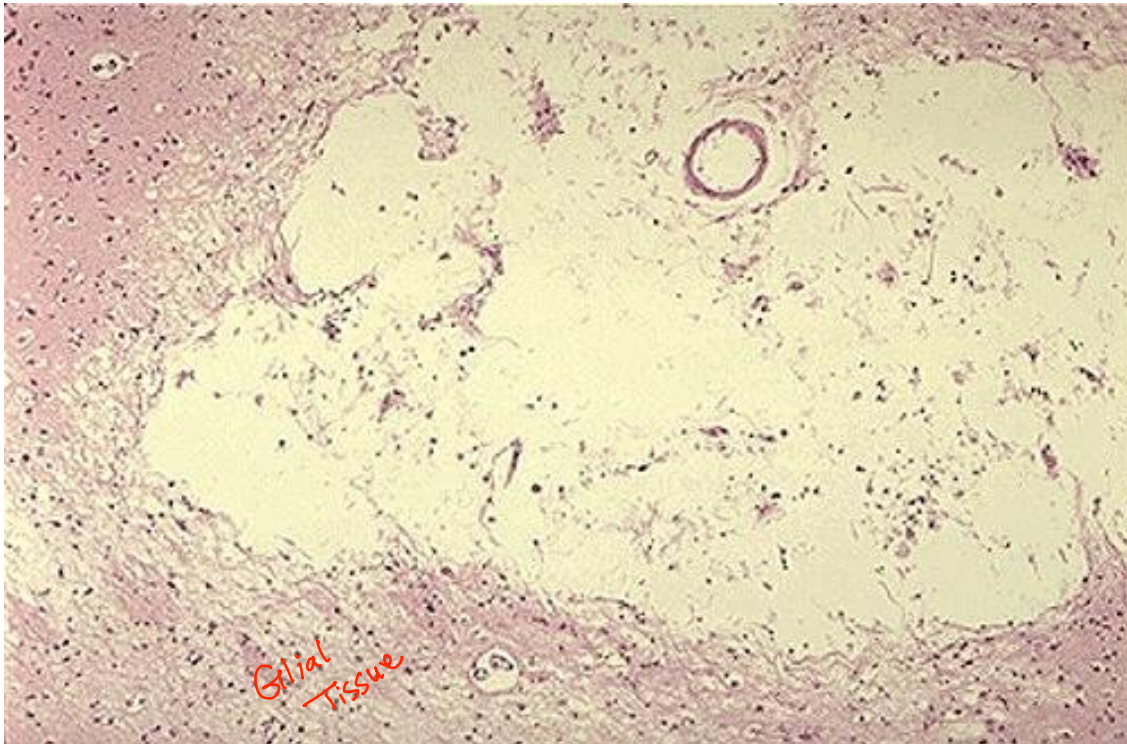
Such lesions are **most common in basal ganglia, deep white matter, and brain stem.**

Asymptomatic or severe neurologic impairment

in severe cases



HYPERTENSIVE CEREBROVASCULAR DISEASE



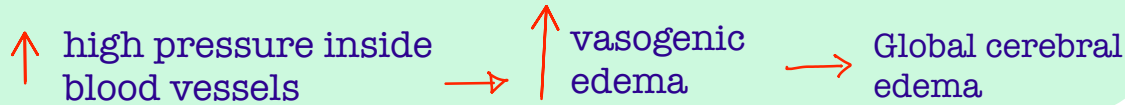
This is the microscopic appearance of a **lacunar infarct**.

Note that it is a cystic space
from the resolved
liquefactive necrosis.



ACUTE HYPERTENSIVE ENCEPHALOPATHY

من ارتفاع ضغط الدم



- *Acute hypertensive encephalopathy* most often is associated with sudden sustained increases in diastolic blood pressure >130 mm Hg.
- It is characterized by increased intracranial pressure and global cerebral dysfunction.
- Headaches, confusion, vomiting, convulsions, and sometimes coma.
- Treatment: rapid intervention to reduce the blood pressure
- Postmortem morphology: brain edema +/- herniation. Petechial and fibrinoid necrosis of arterioles in gray and white matter

treatment: بسرعة نازل
blood pressure



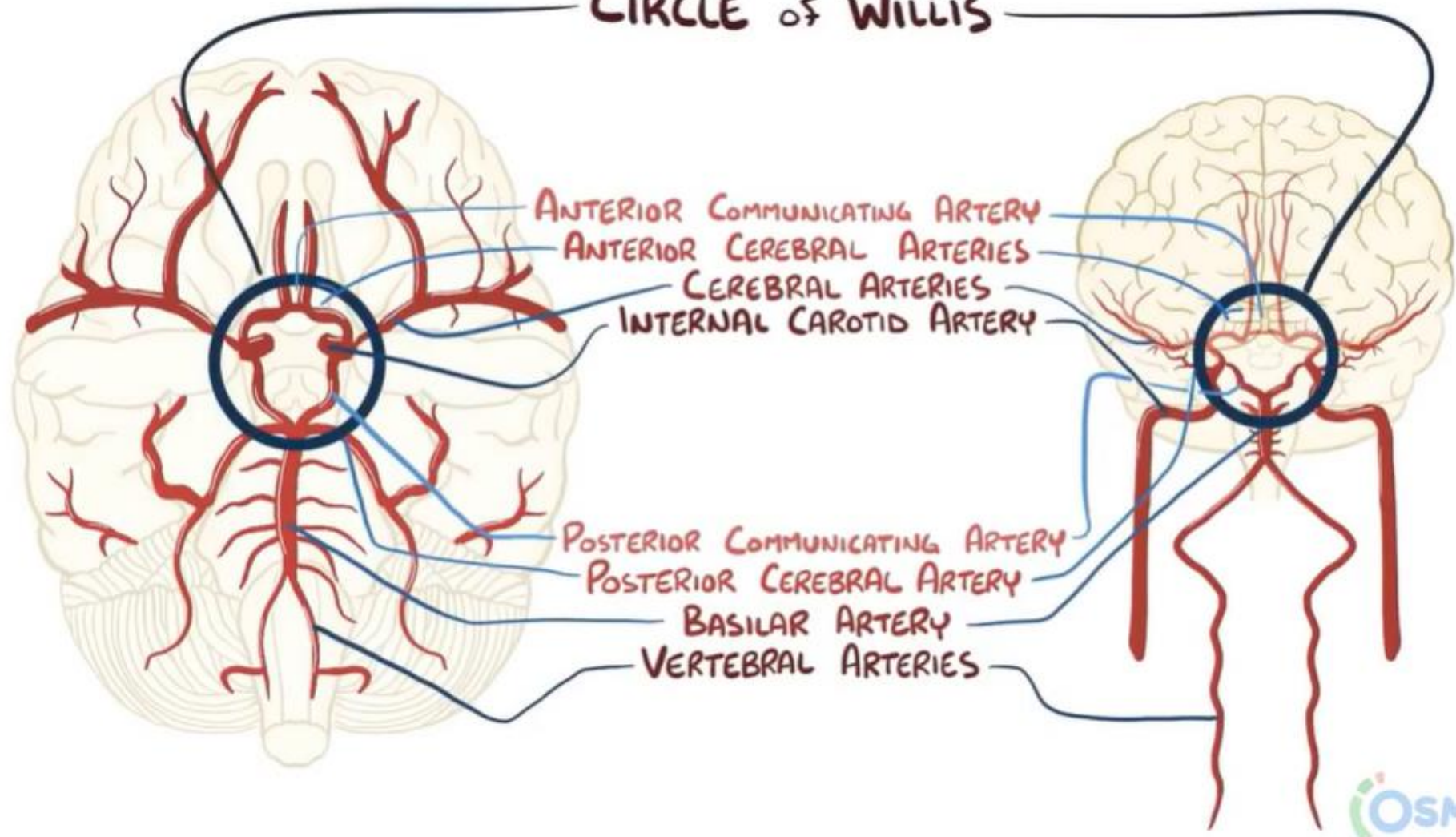
HYPOXIA, ISCHEMIA, AND INFARCTION



على شكل circle مشان اذا انقطع ال blood supply
من مكان ييجيه من مكان ثاني

CIRCLE OF WILLIS

The inferior of brain →



HYPOXIA, ISCHEMIA, AND INFARCTION

- The brain is a highly oxygen-dependent tissue
- Receives 15% of cardiac output and is responsible for 20% of total body oxygen consumption.
- Cerebral blood flow remains stable over a wide range of **blood pressure and intracranial pressure**

The brain may be deprived of oxygen by two general mechanisms:

- **Functional hypoxia**: (e.g., severe anemia, carbon monoxide poisoning)
- **Ischemia**: either transient or permanent, due to tissue hypoperfusion (e.g. hypotension, vascular obstruction)

نقص تروية الأنسجة
←



HYPOXIA, ISCHEMIA, AND INFARCTION

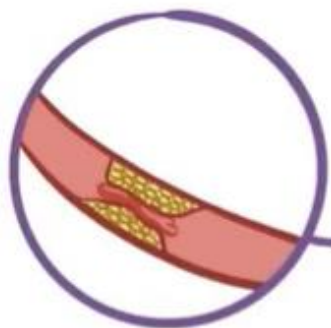
سystolic هاد

- ↓ BP ≤ 50 mm.Hg is critical → Hypoxia & ischemia
- ↑ BP ≥ 200 mmHg critical! → ↑ risk of Hemorrhage



الدكتورة: اذا طلعتوا من هي المحاضرة بس بتعرفوا
انه في two types of stroke
أنا بكون مبسوطه جداً

STROKE



ISCHEMIC

* **BLOCKED ARTERY**

* **MORE COMMON**

* **DAMAGE DEPENDS ON**

↳ LOCATION

↳ TIME

• if SELF-RESOLVES in 24 HOURS it is a **TRANSIENT** ISCHEMIC ATTACK

↳ MINIMAL LONG-TERM DAMAGE



direct CT scan

مشان أميز في hemorrhage او لا

HEMORRHAGIC

زي الي حكيناهم بصيروا
بالhypertension

* **ARTERY BREAKS**

Large area of bleeding → قطعت الدم عن المنطقة الي كان يغذيها يعني عملت
compression



STROKE

STROKE:

The clinical term of acute neurological dysfunction occurring as a result of

hemorrhagic or

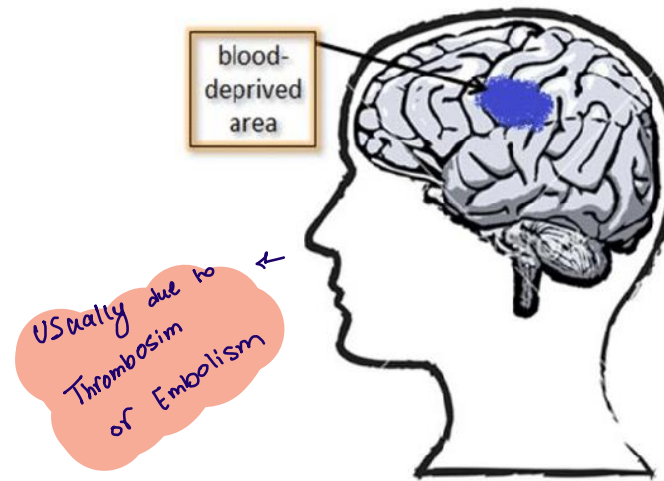
obstructive vascular

lesions, causing

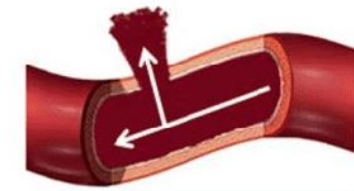
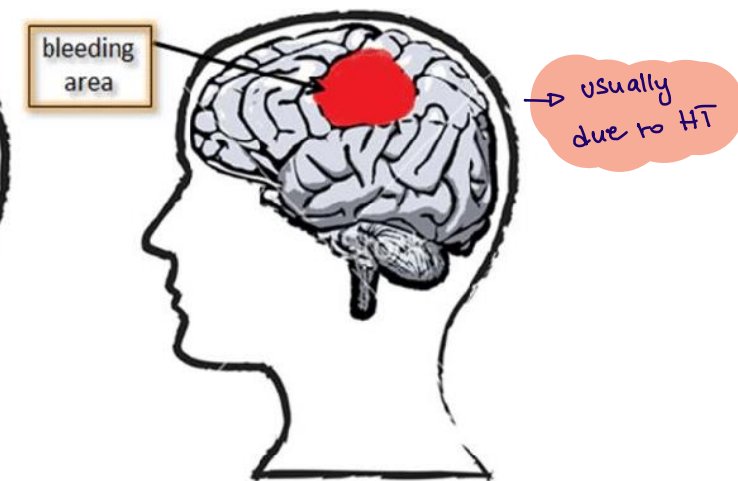
irreversible damage or

death .

Ischemic Stroke



Hemorrhagic Stroke



TYPES OF ISCHEMIC STROKE

Global Cerebral Ischemia

- ✓ Due to generalized decrease in cerebral blood flow
- ✓ **Causes:** severe hypotension, cardiac arrest, and shock
- ✓ Symptoms vary from transient mild confusion to irreversible damage

Focal Cerebral Ischemia

- ✓ Focal brain liquefactive necrosis due to complete and prolonged ischemia
- ✓ **Causes:** thrombosis or embolism (Infarction)



GLOBAL CEREBRAL ISCHEMIA

- neurons die before glial cells

- Neurons are much more sensitive to hypoxia than are glial cells.

* Selective Vulnerability of neurons in certain locations:

① Pyramidal cells of hippocampus

② Purkinje cells of the cerebellum

③ Cortical pyramidal neurons

very sensitive to hypoxia

generalized ischemia اذا صار

ischemia هم اول شي راح يصير فيهم

- In severe global cerebral ischemia, widespread neuronal death occurs irrespective of regional vulnerability.



GLOBAL CEREBRAL ISCHEMIA MORPHOLOGY

- The brain is swollen, with wide gyri and narrowed sulci.
- The cut surface shows poor demarcation between gray matter and white matter

Microscopic changes:

Acute: 12-24 hrs: RED NEURONS >> Infiltration by neutrophils

Subacute changes: 24 hrs – 2 weeks >> tissue necrosis, influx of macrophages, vascular proliferation & gliosis

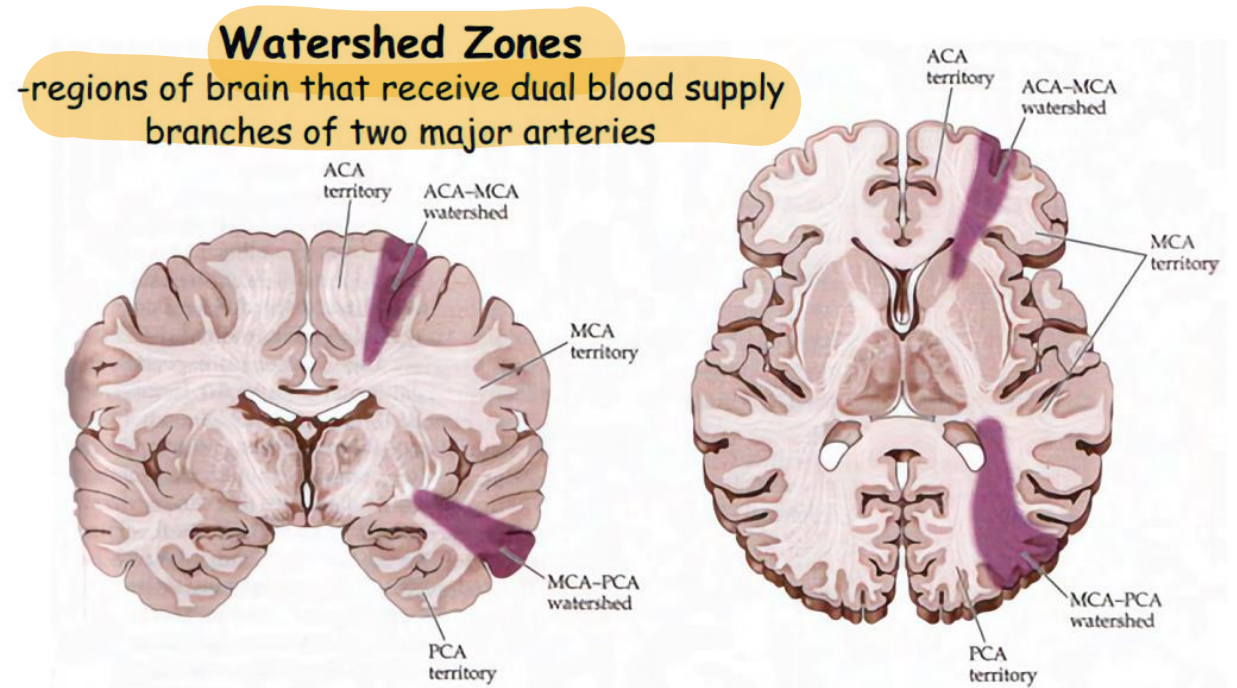
Repair: after 2 weeks >> removal of necrotic tissue and gliosis



GLOBAL CEREBRAL ISCHEMIA MORPHOLOGY

Border zone (“watershed”) infarcts:

- Regions of brain at the most distal reaches of arterial blood supply, border zones between arterial territories
- The border zone between the anterior and the middle cerebral artery distributions is at greatest risk.



FOCAL CEREBRAL ISCHEMIA

- Cerebral arterial occlusion leads first to focal ischemia and then to infarction in the distribution of the compromised vessel.
- The size, location, and shape of the infarct and the extent of tissue damage that results may be modified by collateral blood flow.

Infarcts can be divided into two broad groups:

- Non-hemorrhagic infarcts result from acute vascular occlusions and may evolve into hemorrhagic infarcts when there is reperfusion of ischemic tissue, either through collaterals or after dissolution of emboli.



FOCAL CEREBRAL ISCHEMIA

Embolitic occlusion: hemorrhagic/red

- **Source:** heart or atherosclerosis in carotid arteries
- Middle Cerebral artery most affected

Thrombotic occlusion, mainly due to atherosclerosis : Ischemic/pale

- Carotid bifurcation
- Origin of middle cerebral artery
- Basilar artery at either end



FOCAL CEREBRAL ISCHEMIA MORPHOLOGY

- No gross change before 48 hrs
- Soft swollen pale or hemorrhagic **wedge-shaped** infarct involving grey & white matter.
- Red infarcts: surrounding hemorrhage due to reperfusion of damaged vessels and tissue
- **After 10 days-3 weeks : liquefaction** → necrosis
- **Cavity formation within 1- 6 months**

Microscopic: Very similar to global ischemia but more regional.



STROKE CLINICAL FEATURES

Clinical picture: linked to site of infarction

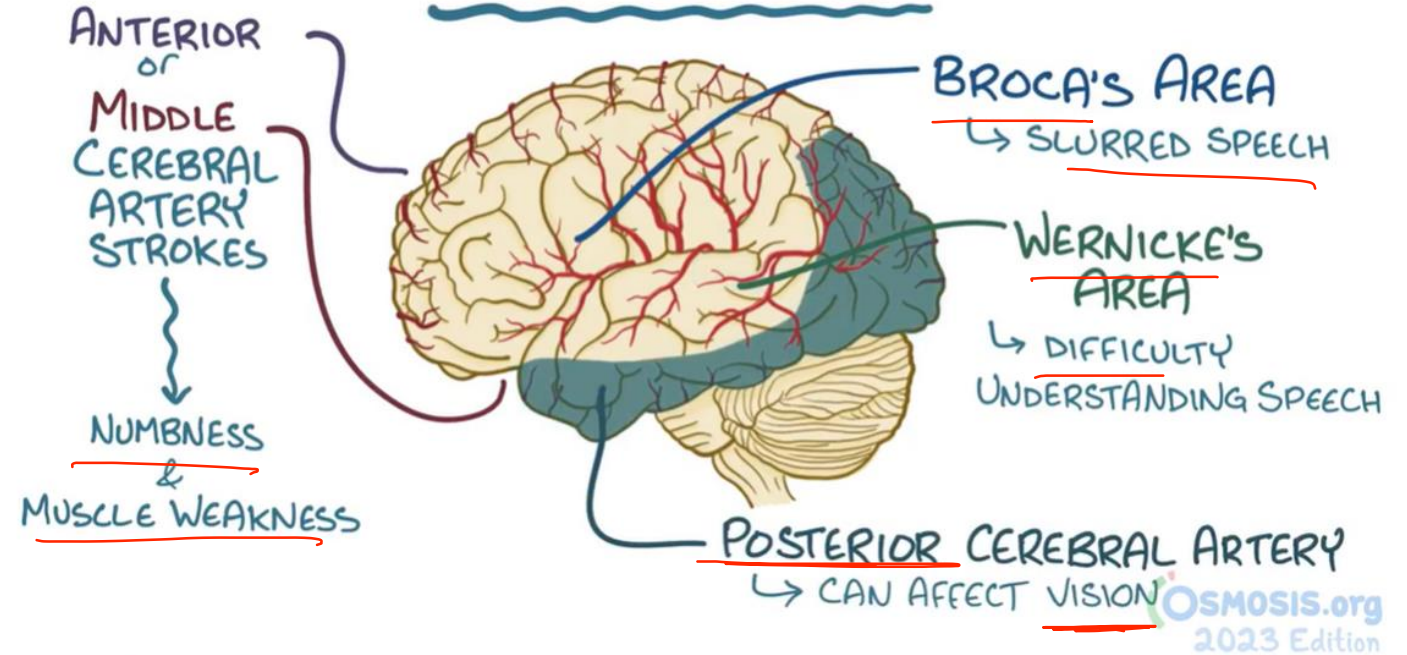
Contralateral hemiparesis

Loss of sensation

Visual field abnormalities

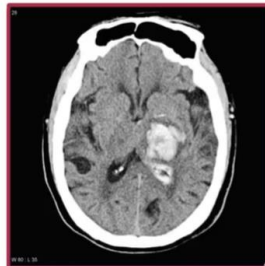
Aphasia etc

SYMPTOMS

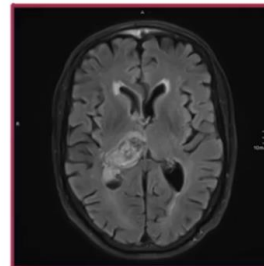


DIAGNOSIS

CT SCAN



MRI



ALSO ANGIOGRAPHY

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SYMPTOMS

FACIAL DROOPING

ARM WEAKNESS

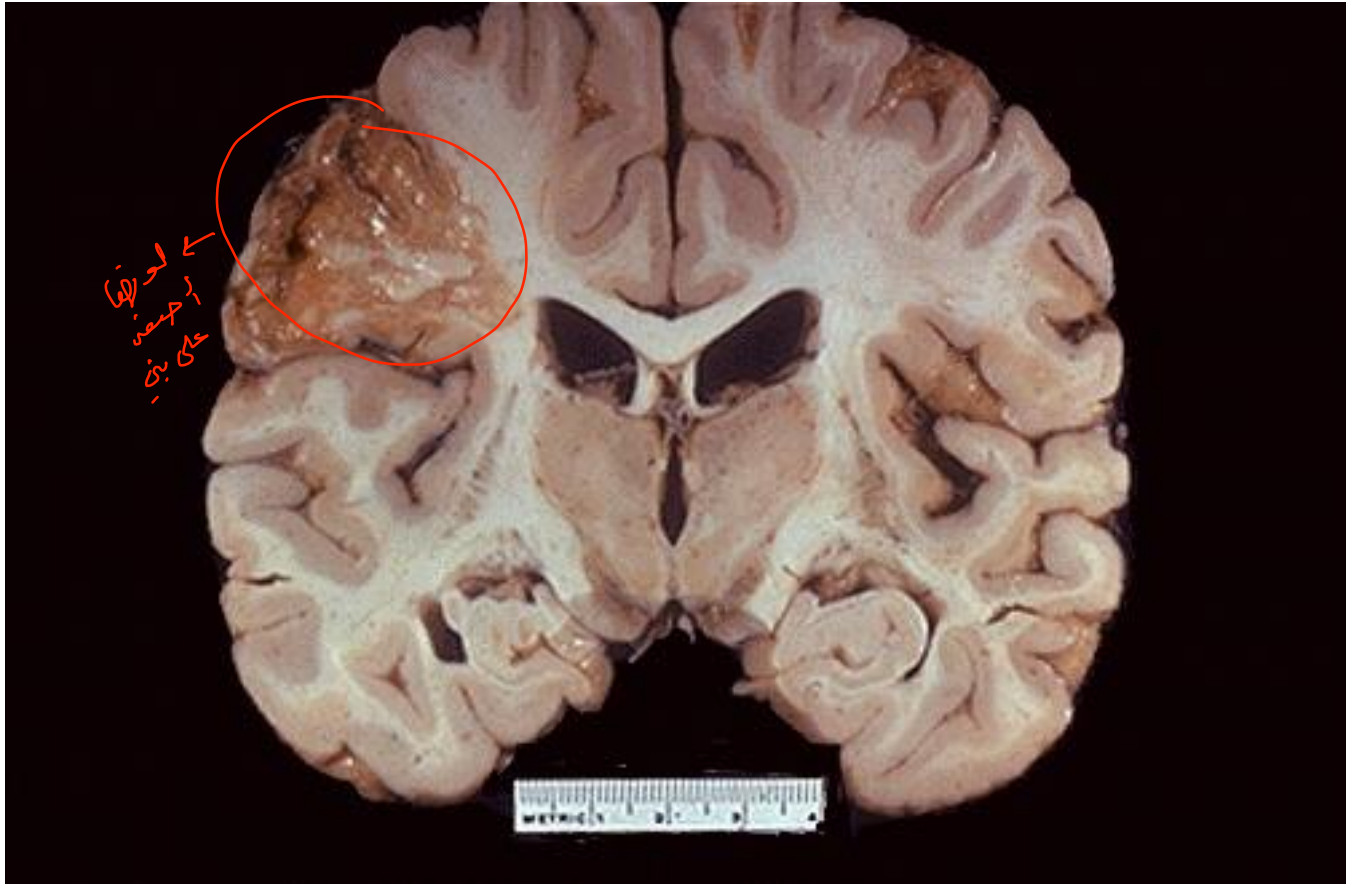
SPEECH DIFFICULTIES

TIME (GET HELP QUICKLY)

مش symptom بس لازم بسرعة نعالجه

first step : ST scan to make sure it's not hemorrhagic





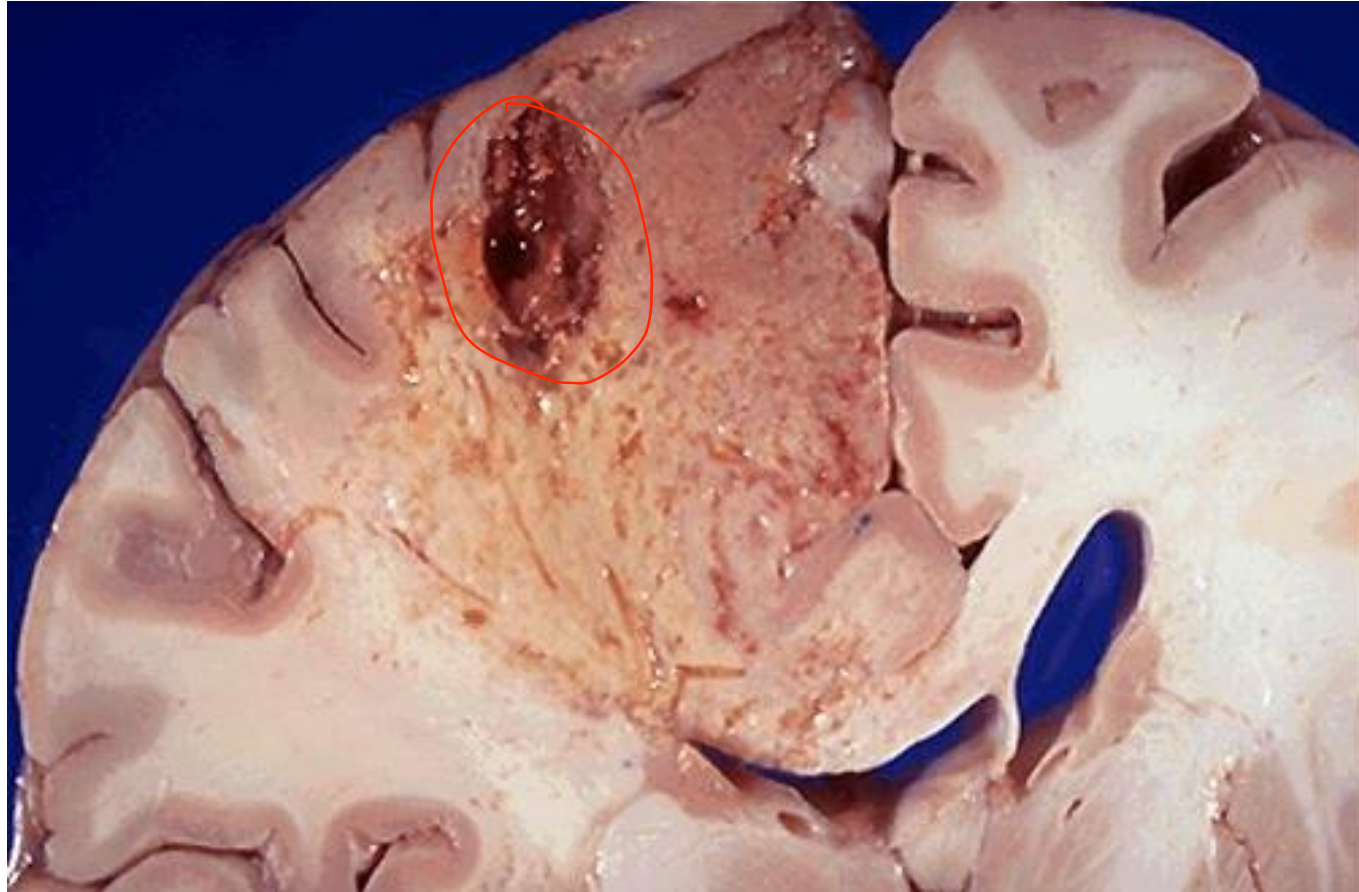
This is an intermediate to remote infarct in the distribution of the middle cerebral artery.





A thrombosis of the internal carotid artery is seen here above the arrowhead.

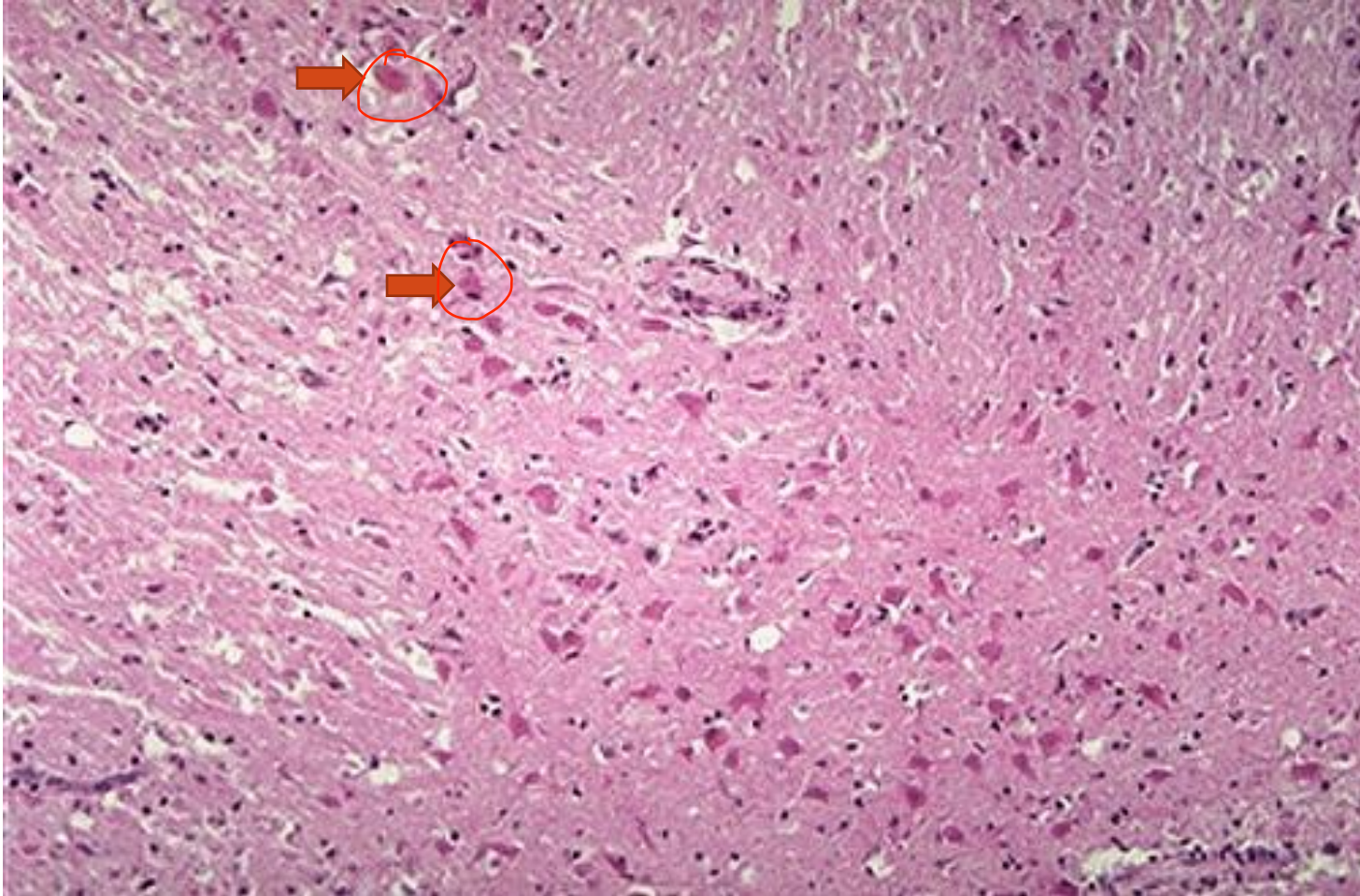




This subacute (intermediate, partially liquefied) infarct of the frontal lobe shows liquefactive necrosis with formation of cystic spaces as time passes and the process of resolution begins.



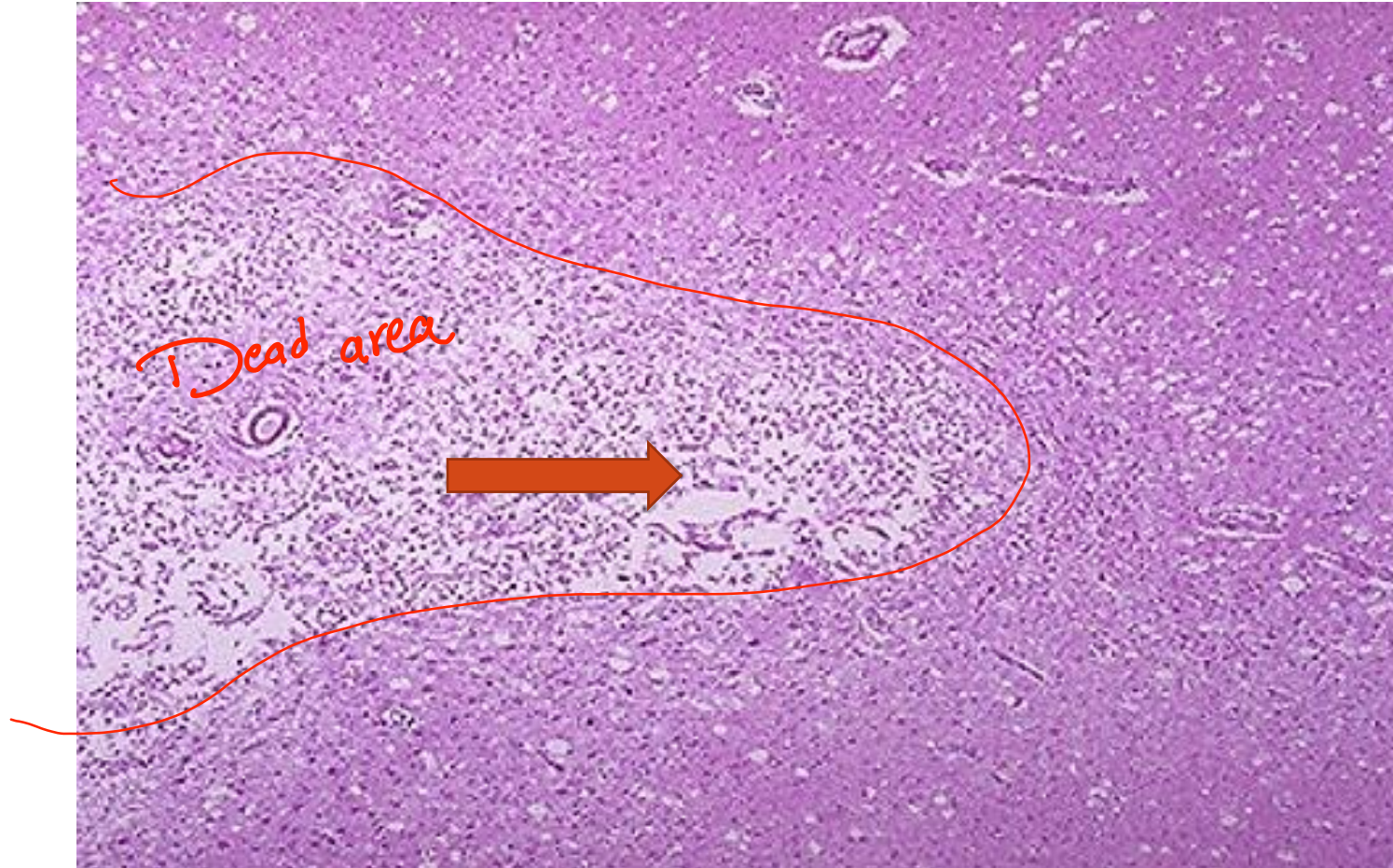
global ischemia



The neurons are the most sensitive cells to anoxic injury. Seen here are red neurons which are dying as a result of hypoxia.

One of the most sensitive areas in the brain to hypoxic injury is the hippocampus, as seen below.





Resolution of the liquefactive necrosis by an influx of blood monocytes to become tissue macrophages in a cerebral infarction over weeks leads to the formation of a cystic space.

