



PATHOLOGY

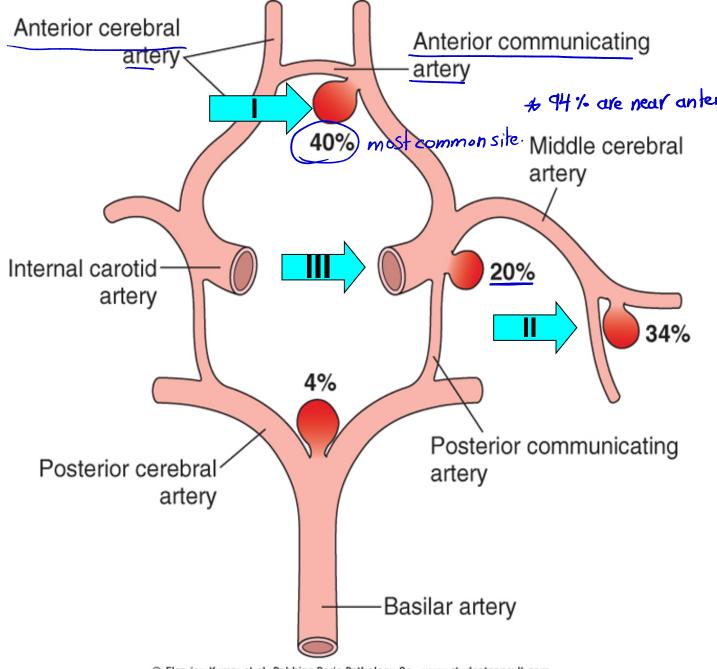


DONE BY: Hamzeh Alsalhi

- ▼ Without treatment, recurring bleeding is common in survivors & prognosis worsens with each episode of bleeding.
- p90% of saccular aneurysms occur in the **anterior** circulation near major arterial branch points (F23-9); **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 arterial media.
- ★There is an ↑ risk of aneurysms (1) in individuals with autosomal dominant polycystic kidney disease; & in (2) in an association with disorders of ECM

is more prone to rupture. حالاً المناس aneurysm احف والتالا على aneurysm كون الهالم

- ★ The probability of rupture ↑ with the aneurysm size, aneurysms >1cm have a roughly 50% risk of bleeding per year.
- In the healing phase of subarachnoid H, meningeal fibrosis & scarring occur, sometimes leading to obstruction of CSF flow as well as interruption of the normal pathways of CSF resorption. → Leading to Hydrocephalus



معنعا عمصه

94% are near anterior and middle cerebral arteries.

F23-9: Relative frequency of common sites of saccular (Berry) aneurysms in the circle of Willis.

► GROSSLY, ★ Berry saccular aneurysm is a thin-walled outpouching of an artery. At the neck of the aneurysm, the muscular wall & intimal elastic lamina stop short & are absent from the aneurysm sac itself and the sac is made up of thickened hyalinized intima. The adventitia covering the sac is continuous with that of the parent artery (F23-10).

Rupture usually occurs at the apex of the sac with extravasation of blood (mostly) into the subarachnoid space, or, less commonly in the substance of the brain, or in both.

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Berry saccular aneurysms are the most common type of intracranial aneurysm, in addition,

include atherosclerotic (fusiform, mostly of the basilar artery, F 9.39); & Mycotic, Traumatic, & Dissecting aneurysms, the latter 3, as with saccular aneurysms, are most often found in the **anterior** circulation.

 The 4 aneurysms usually present with cerebral infarction from vascular occlusion instead of subarachnoid H.

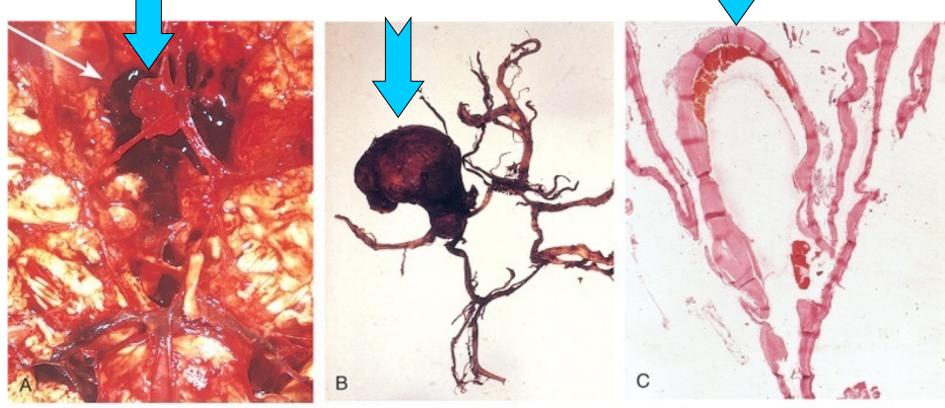


F23-10: Berry saccular aneurysms.

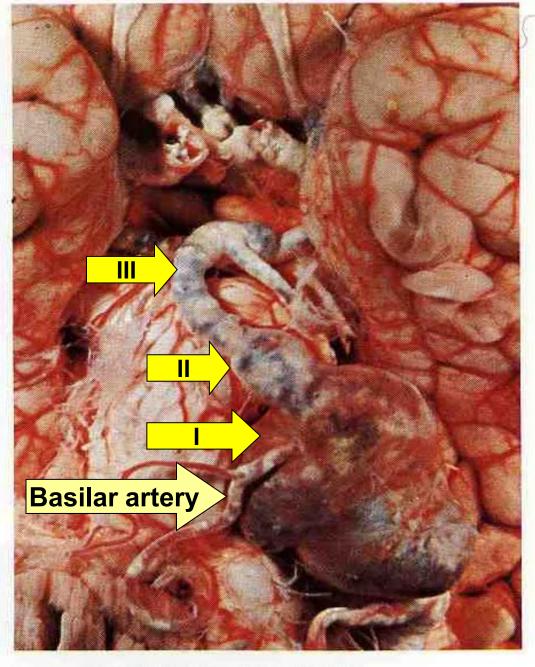
A, View of the base of the brain, dissected to show the circle of Willis with an aneurysm of the anterior cerebral artery (arrow).

B, Dissected circle of Willis to show the large aneurysm.

C, Section through a saccular aneurysm showing the hyalinized fibrous vessel wall (H&E).



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9.39 Aneurysm: basilar artery

F 9.39:
Atherosclerotic
aneurysm of
basilar artery.

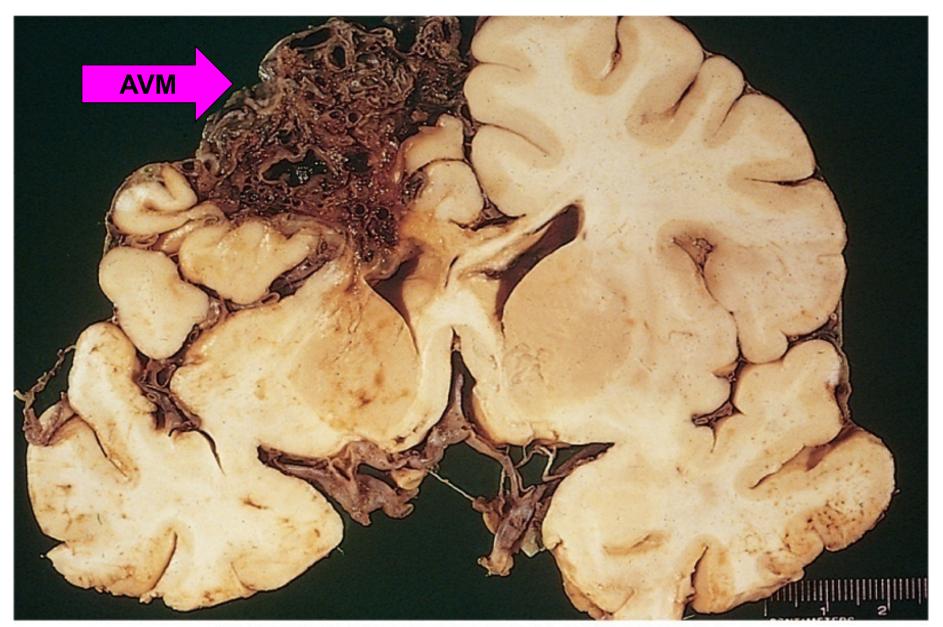
Large, bluish -black, part- (I) saccular & (II) fusiform part of the 1st part of the artery; (III) Above the site of aneurysm, the basilar artery is dilated with scattered atheromatous plaques seen.

Vascular Malformations

Vascular malformations of the brain are classified into 4 main types, based on the **nature** of the abnormal vessels:

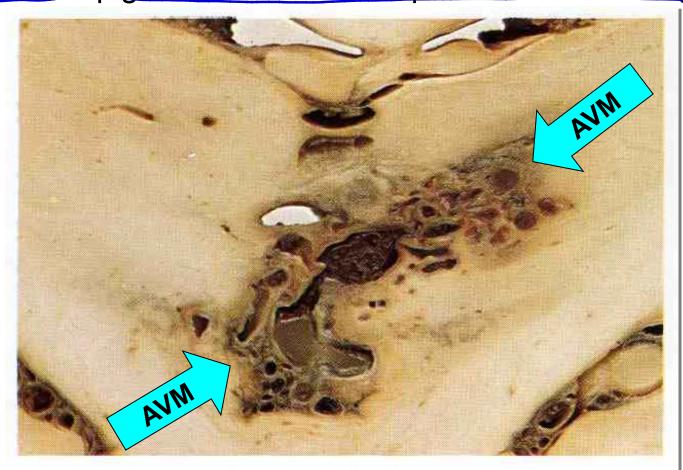
- (1) Arteriovenous malformations (AVM), The most common.
- (2) Cavernous angiomas, -> fumor مستع دلسته المستادة
- دواليے (3) <u>Capillary telangiectasias</u>,
- (4) Venous angiomas. This is Hemorrhage de Gous le troque
- (1) Arteriovenous malformations (AVM), the most common, affect males twice as frequently as females; the lesion is most often recognized clinically between the ages of 10 & 30 years, presenting as a (1) seizure (epilepsy), Or (2) H (intracerebral or subarachnoid, depending on their location).
- Large AVMs occurring in the newborn period can lead to high-output congestive heart failure because of blood shunting directly from arteries to veins. Locaused by: Anemia, arterial-venous malformation of thyrotoxicosis, of Paget disease
- The risk of bleeding makes AVM the **most dangerous** type of vascular malformation (F23-11 & 9-35).

F23-11: Arteriovenous malformation in subarachnoid space.



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F 9-35: Arteriovenous malformation (hamartoma): brain. A large complex intracerebral AVM is present within the thalamus & basal ganglia. The greyish-white vessels are thick-walled & many are thrombosed. The adjacent brain contains much brown hemosiderin pigment as a result of previous hemorrhages.



9.35 Arteriovenous hamartoma: brain

wormlike vascular channels involve vessels either in the (I) subarachnoid space (F23-11) extending into brain

parenchyma or they may occur (II) exclusively within the brain (F9-35).

■ H, they are enlarged BV **separated by gliotic tissue**, often with evidence of previous H. Some BVs can be recognized as arteries with duplicated & fragmented internal elastic lamina, while others show marked thickening or partial replacement of the media by hyalinized connective tissue.

Malformation Lean Lil

(2) Cavernous hemangiomas consist of aggregates of distended, loosely organized vascular channels with thin and ne collagenized walls; devoid of intervening nervous tissue capsale. (thus distinguishing them from capillary telangiectasias).

They occur most often in the cerebellum, pons, &

subcortical regions.

Old foci of H, infarction, & calcification frequently surround the abnormal lesion.

(3) Capillary telangiectasias (4.40) are microscopic foci of dilated, thin-walled vascular channels separated by relatively normal brain parenchyma & occurring mostly in the pons.

(4) Venous angiomas (varices) consist of aggregates of ectatic (dilated) venous channels (F 9-34).

Lesions 3 & 4 are unlikely to bleed or cause symptoms, & are most commonly discovered incidentally.

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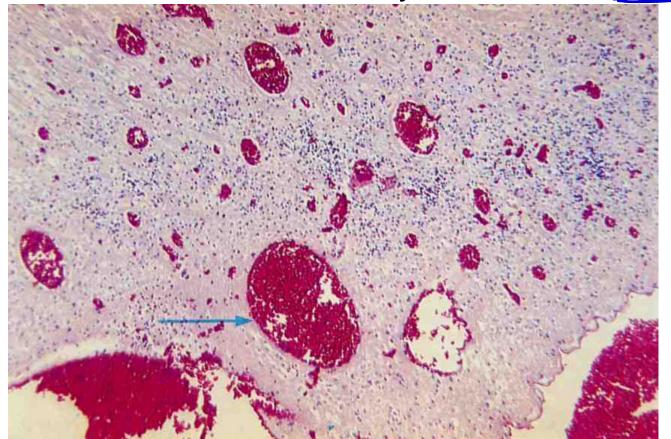
bain tissue Hypertensive cerebrovascular disease

Over the past few decades there has been a Ψ threshold for \odot treatment of hypertension & more extensive screening for early disease, both of which have contributed to an overall Ψ in the incidence of these complications.

Nevertheless, hypertension continues to be important, <u>due to</u> (<u>poor patient compliance</u>) or inadequate access to health care. * مريفة المعاروض يكمل العلاج تقريباً لباقي ميا ته معم العالم في العلاج العلاج تقريباً لباقي ميا ته معم العلاج العلاج تقريباً لباقي ميا ته العلاج العلى ميا ته العلاج العلى العلاج العلاء العلاج الع

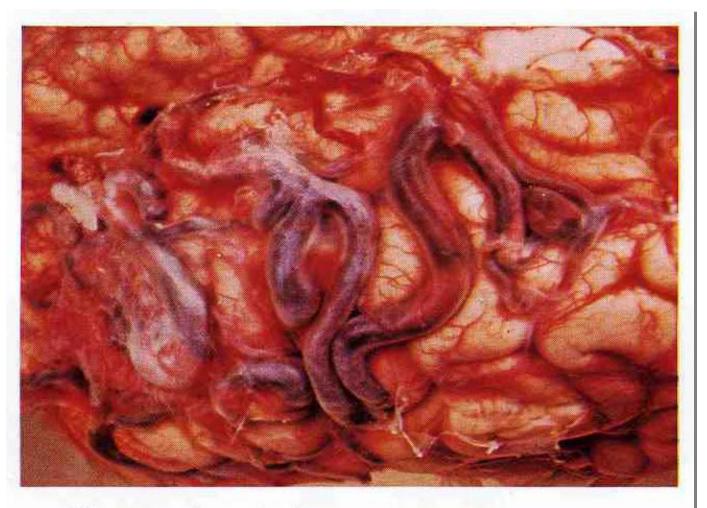
- Hypertension affects the deep penetrating arteries & arterioles that supply the basal ganglia & hemispheric white matter & the brain stem.
- ⊗4 Most important **effects of hypertension** on the brain are:

■ 4.40: Capillary telangiectasia: Brain X 80. A solitary lesion, consists of abnormally dilated capillaries, each with a very thin wall (arrow), surrounded by thin layer of eosinophilic hyaline amorphous material. The capillaries are separated by neural tissue & not by fibromuscular tissue (compare with those seen in an ordinary capillary/ cavernous hemangiomas). Complete resection of this lesion may be difficult or, impossible.



F 9-34: **Venous angioma: brain,** forming a complex tangle of dilated & thrombosed veins within the leptomeninges (arachnoid & pia mater) over the left parietal lobe. © **This rare lesion is** unlikely to bleed or cause symptoms

& is most commonly discovered incidentally.



9.34 Venous angioma: brain

- (1) Massive hypertensive intracerebral H (2) Lacunar infarcts (3) Slit H (4) Hypertensive encephalopathy
- (1) <u>Massive</u> hypertensive intracerebral H (see above), in which chronic hypertension is associated with the development of minute <u>Charcot-Bouchard microaneurysms</u> in vessels that are less than 300 μm in Ø, these aneurysms can rupture, resulting in **Spontaneous** intraparenchymal **H**.
- (2) <u>Hyaline arteriolar sclerosis</u>, in which arterioles become weaker than normal & are more vulnerable to rupture; the important clinical & pathologic outcome of which is the development of *lacunes* or *lacunar infarcts*. These small cavitary infarcts are <15 mm, are found most commonly in deep gray matter (basal ganglia & thalamus); internal capsule, deep white matter, & pons, & they consist of tissue loss *cavities*, with scattered lipid-laden macrophages & surrounding gliosis.

Depending on their location in the CNS, lacunes can either be clinically silent or cause significant neurologic impairment.

- (3) Hypertension also gives rise to rupture of the small-caliber penetrating vessels & the development of small H. In time, these H resorb, leaving behind a slitlike cavity (slit hemorrhage) surrounded by brownish discoloration.
- (4) Acute hypertensive encephalopathy is a clinicopathologic syndrome characterized by diffuse cerebral dysfunction, including headaches, confusion, vomiting, & convulsions, sometimes leading to coma@ باعرافيا المرتفيد
- Postmortem examination of fatal cases show an edematous brain, with/or without transtentorial or tonsillar herniation.
- H, Fibrinoid necrosis of arterioles & petechiae (of malignant hypertension) may be seen microscopically in the gray & white matter.

Vasculitis

- A variety of inflammatory processes that involve BV may lead to luminal narrowing & cerebral infarcts.

 Infectious arteritis of small & large BV was previously seen in association with syphilis & tuberculosis, but now more commonly occurs in the setting of immunosuppression & opportunistic infection (such as toxoplasmosis, aspergillosis, & CMV encephalitis).
- ★ Some of the systemic forms of vasculitis, such as polyarteritis nodosa (PAN), may involve cerebral BV & cause single or multiple infarcts throughout the brain.

*Primary angiitis of the CNS is an inflammatory disorder that involves multiple, small to medium-sized parenchymal & subarachnoid vessels, & is characterized by chronic inflammation, multinucleated giant cells (with or without granuloma formation), & destruction of the BV wall.

Affected individuals manifest a diffuse encephalopathic clinical picture, often with cognitive dysfunction; improvement occurs with steroid & immunosuppressive treatment.