



PATHOLOGY

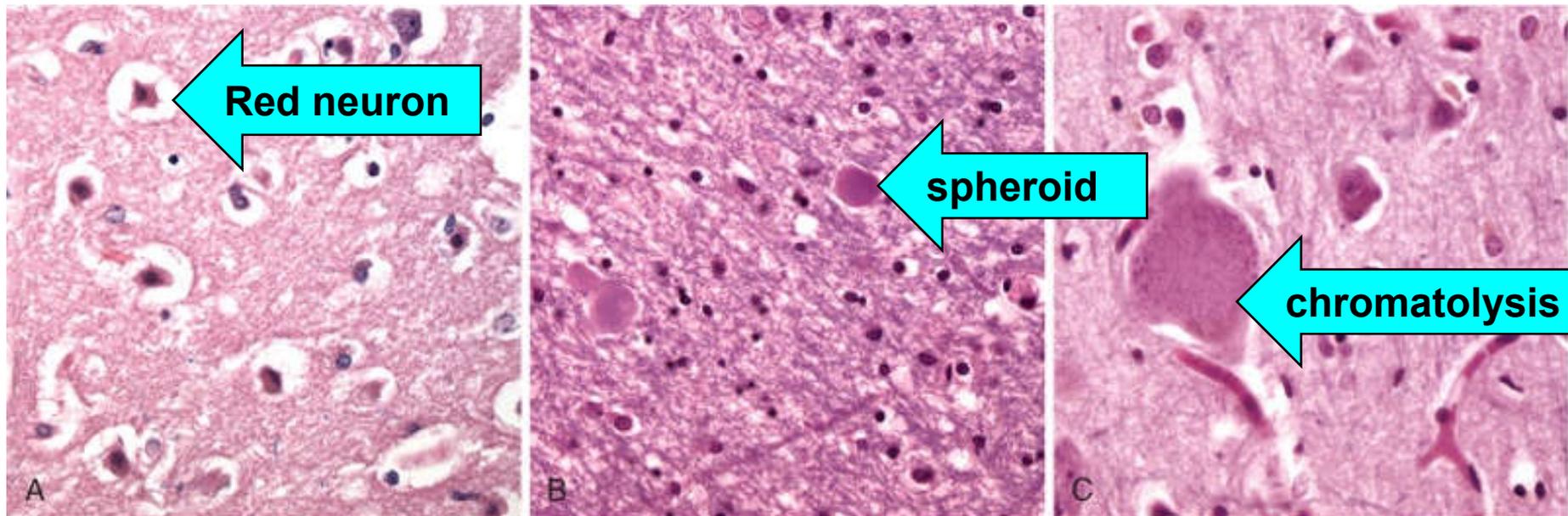
DONE BY : Volunteer

F 23-1: Patterns of neuronal injury.

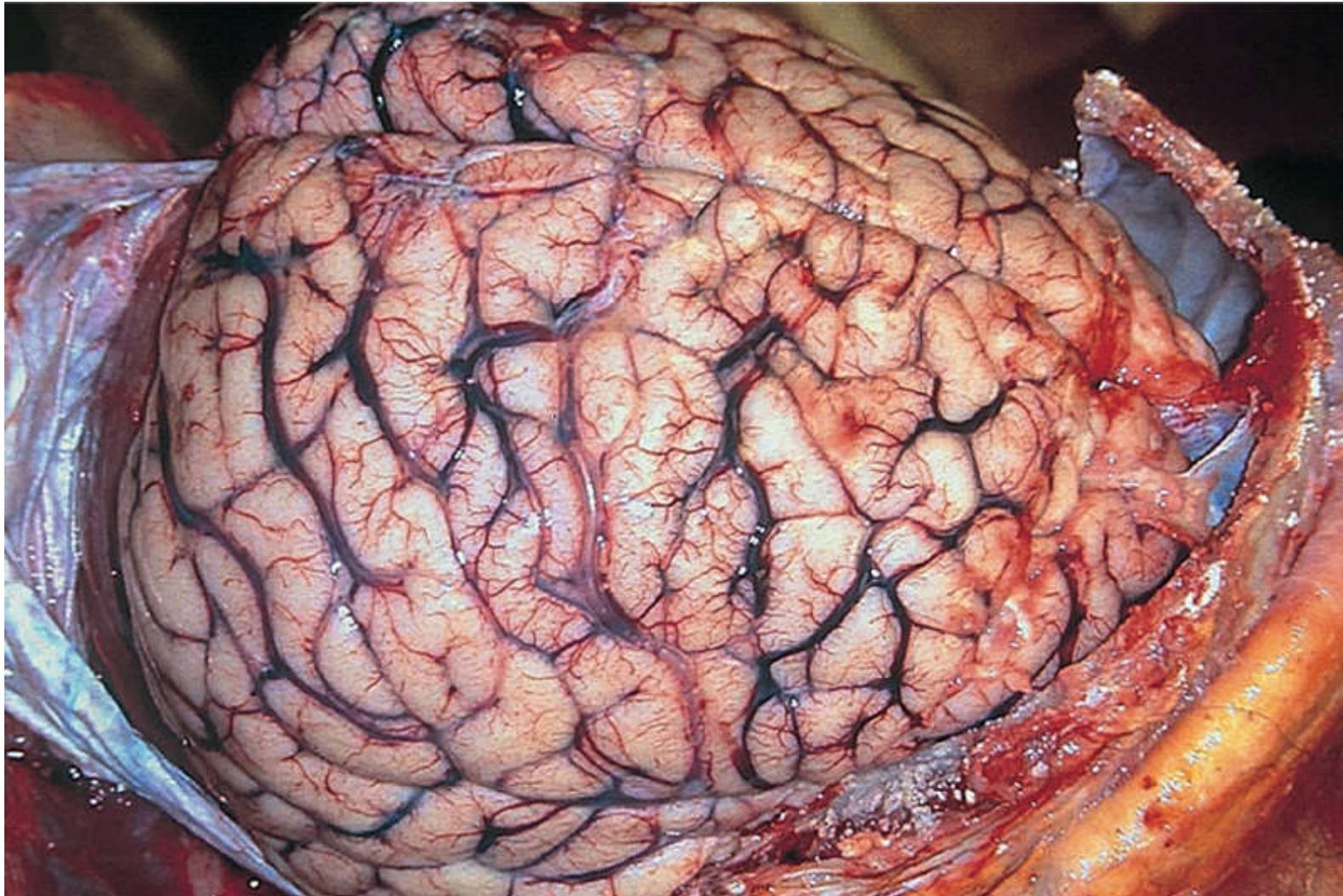
A, Acute hypoxic/ischemic injury in cerebral cortex. The necrotic neuronal cell bodies & their nuclei are shrunken & are prominently eosinophilic, so-called "red neurons".

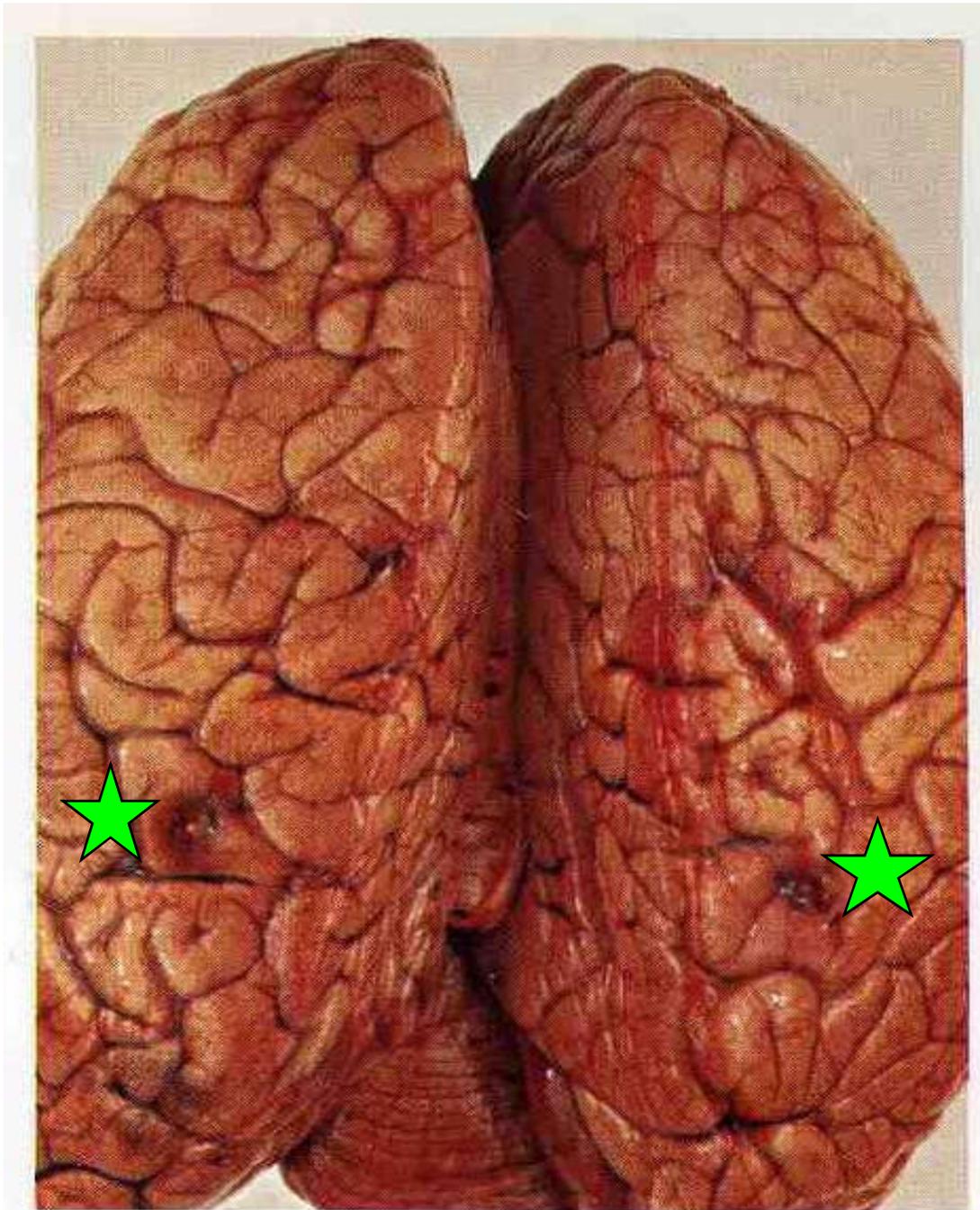
B, Axonal **spheroids** are visible as bulbous swelling at points of disruption or altered axonal transport.

C, With **axonal injury**, there is swelling of the cell body & peripheral dispersal of the Nissl substance i.e., **chromatolysis**.



F 23-2: **Cerebral edema.** The surfaces of the **gyri are flattened** as a result of compression of the expanding brain by the dura matter & inner surface of the skull, These changes cause \uparrow intracranial pressure (**ICP**) & death.





F 9-81: Brain edema:
Symmetrical cerebral swelling & edema,
with marked flattening of the convolutions (gyri) & compression of the groves (sulci).

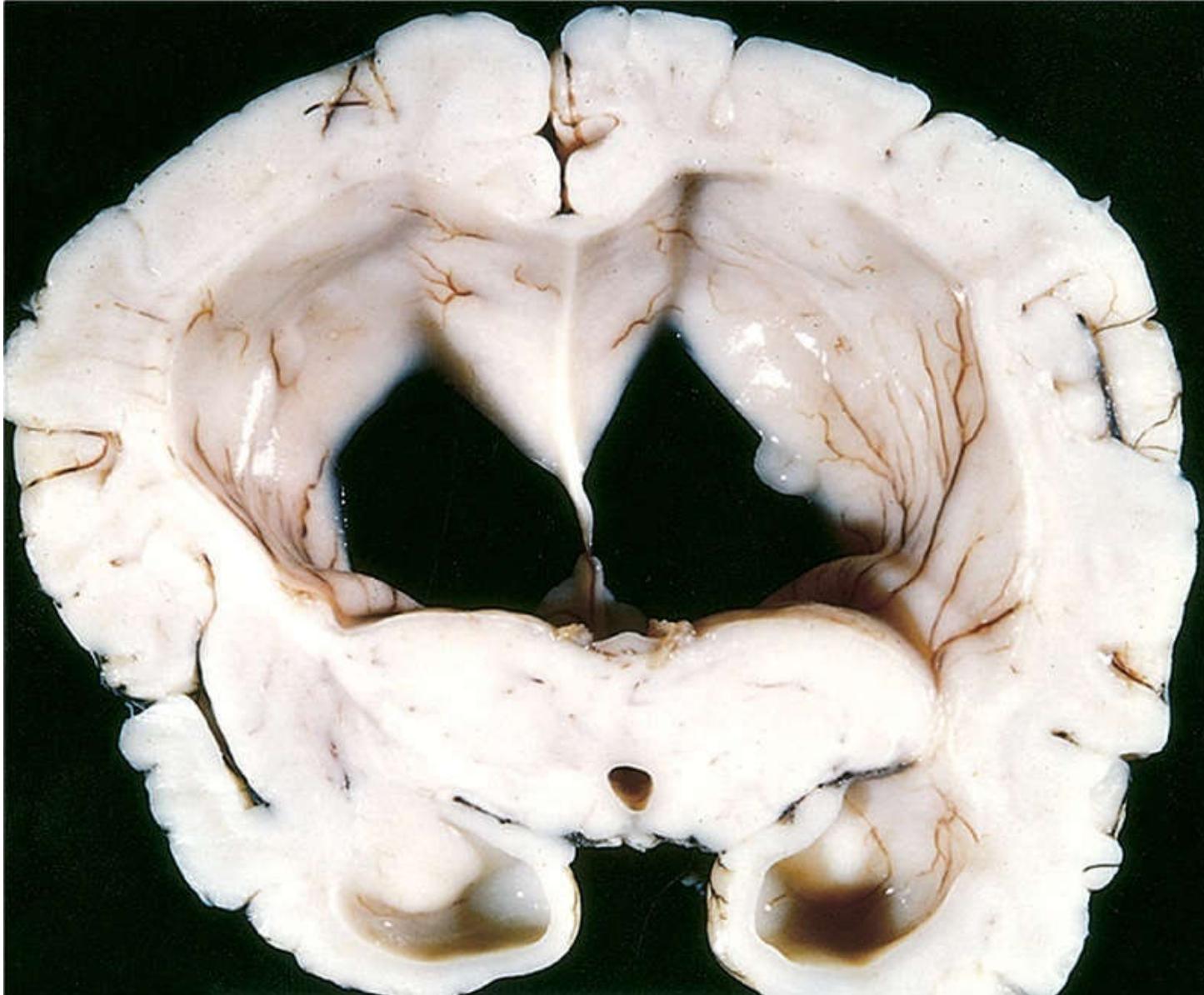
★ 2 parasagittal needle holes are present.

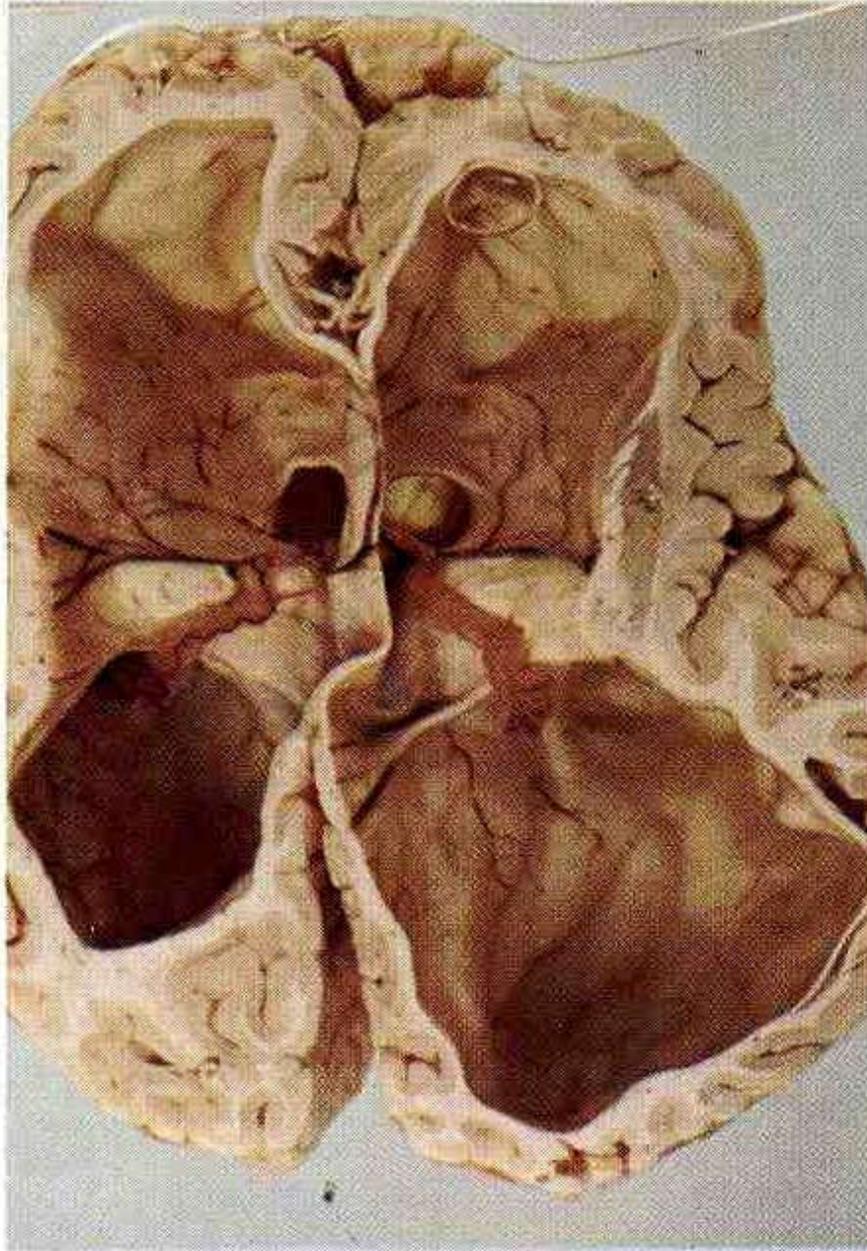
☠ **Cause:**

subependymal astrocytoma obstructing CSF flow, resulting in hydrocephalous & cerebral edema, leading
↑ ICP & ☠ death.

9.81 Swelling and oedema: brain

F 23-3: **Hydrocephalus.** Dilated lateral ventricles seen in a coronal section through the mid-thalamus.

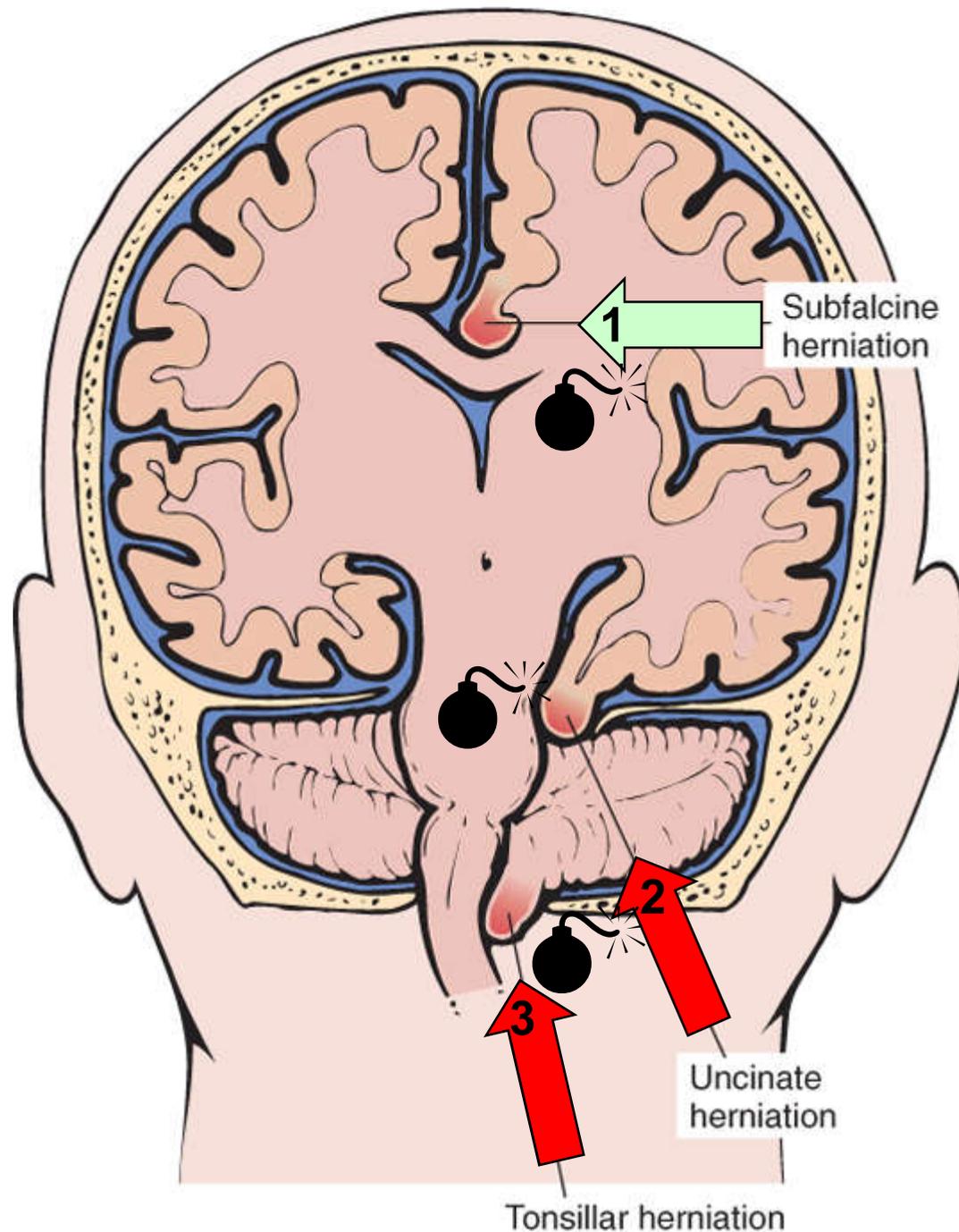




**F 9-17: Hydrocephalus:
brain.**

Noncommunicating, with marked symmetrical dilatation of the lateral ventricles & interventricular foramina, with subsequent thinning of the periventricular white matter.

9.17 Hydrocephalus: brain



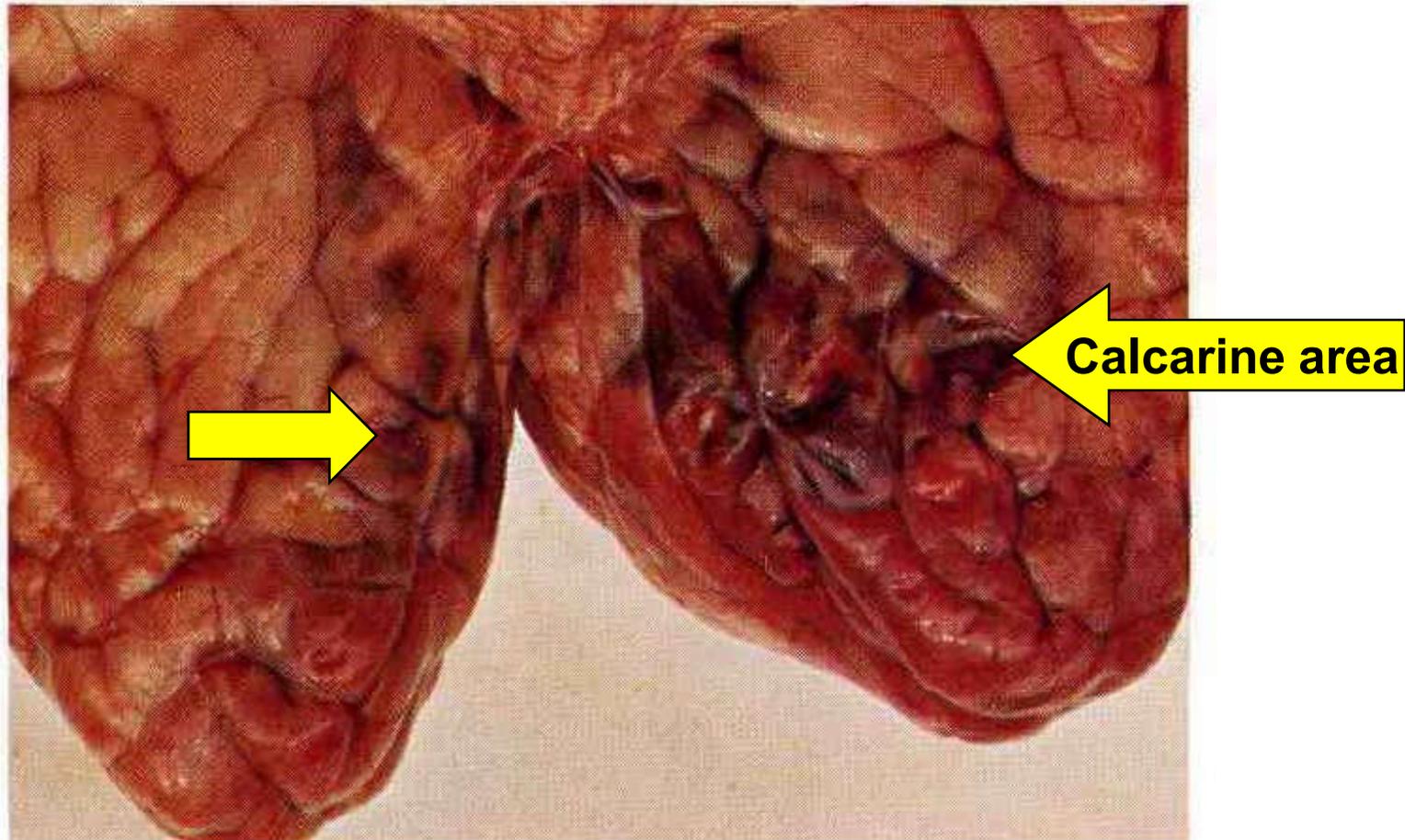
F23-4: Patterns of brain herniation:

(1) subfalcine (**cingulate**),

(2) **transtentorial**
(uncinate, mesial temporal), &

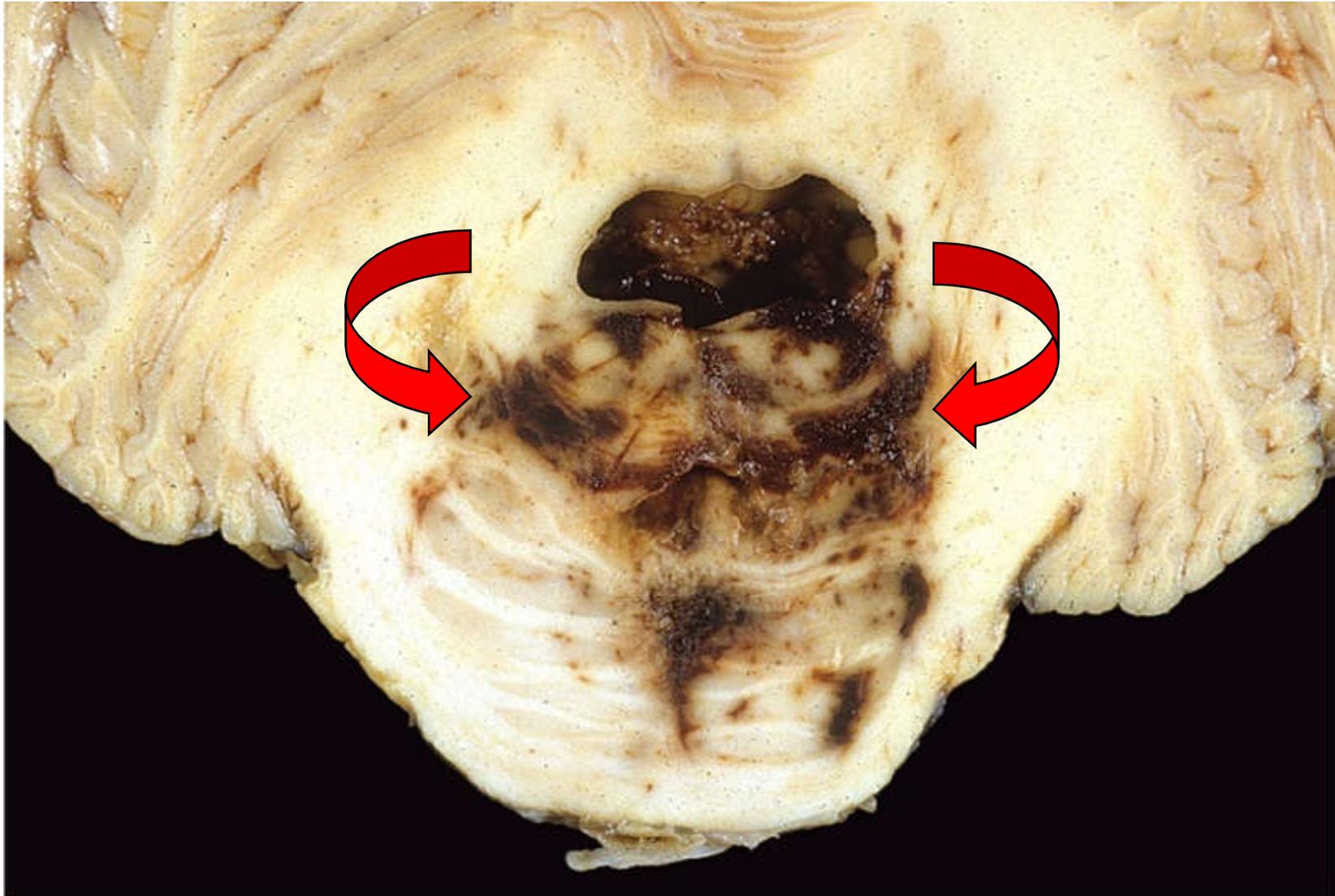
(3) **tonsillar.**

F 9.47: Infarction: Brain. ☠ The patient had transtentorial herniation obstructing the posterior cerebral arteries, resulting in **recent hemorrhagic infarction** of the infero-medial aspects of both occipital lobes, especially affecting the calcarine area.



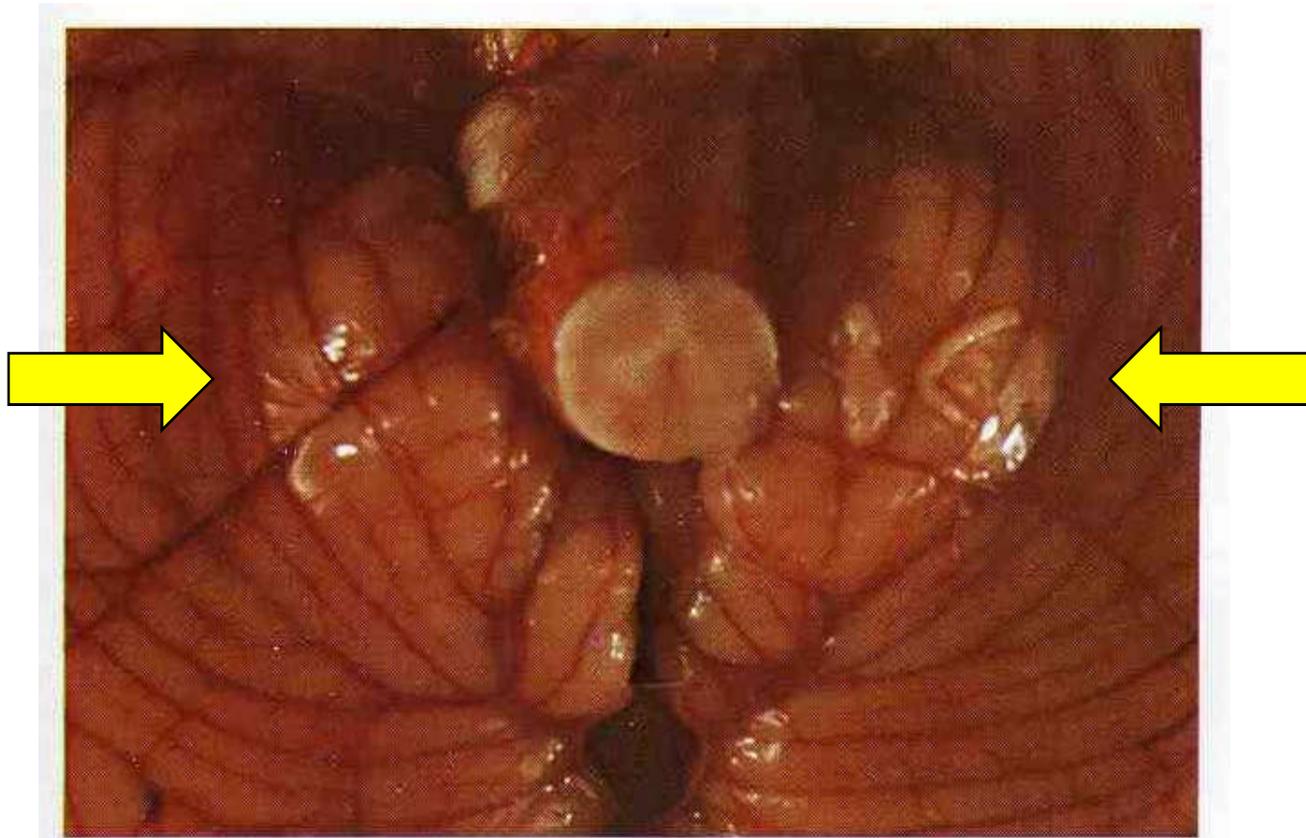
9.47 Infarction: brain

F23-5:Duret (Pontine) hemorrhage. As mass effect displaces the brain downwards, there is disruption of the penetrating BV that enter the pons along the midline leading to ☠ **fatal hemorrhage.**



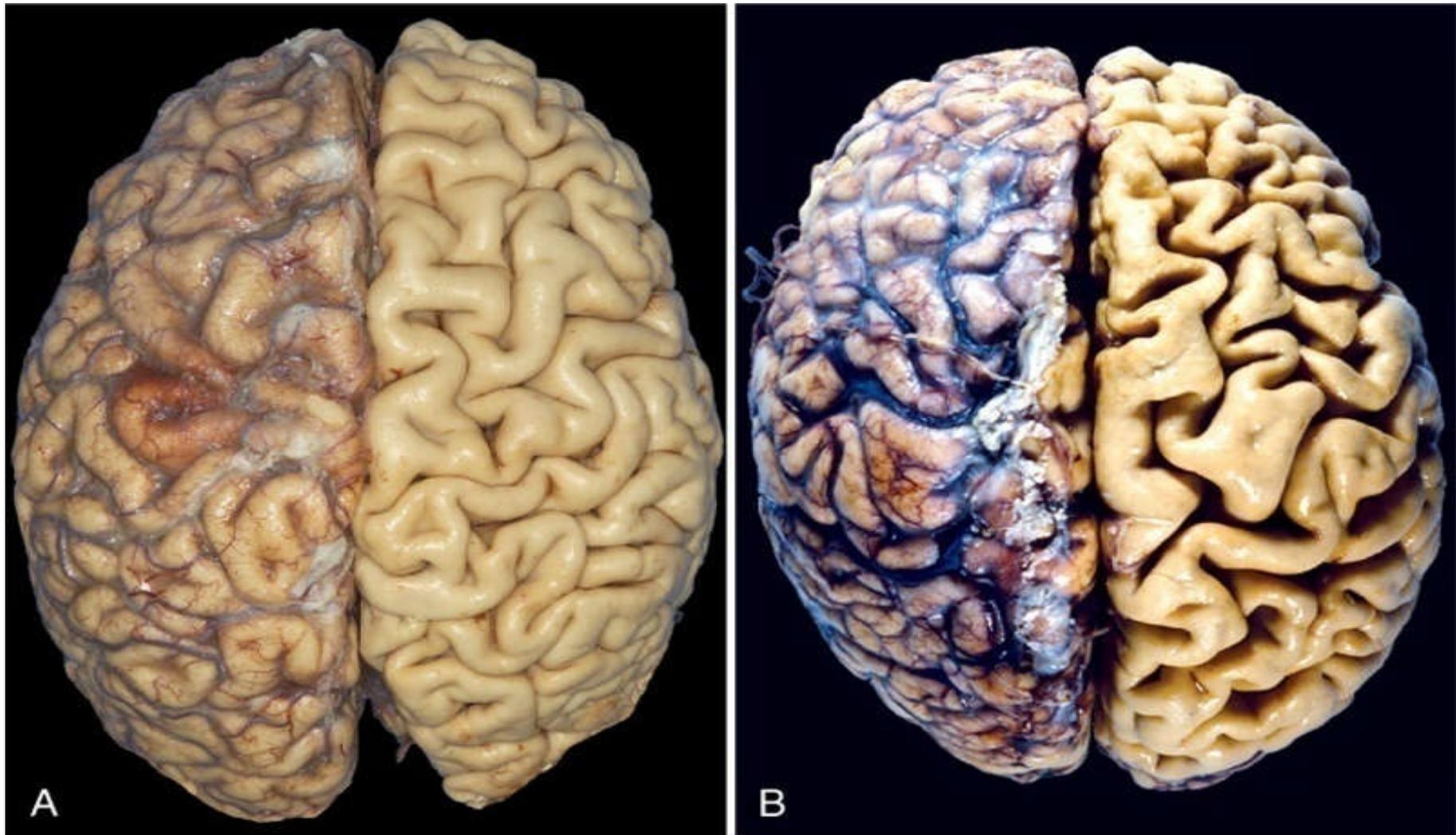
F9-82: Tonsillar herniation (“pressure cone”): cerebellum.

The cerebellum is deeply grooved, forming a well- marked ‘**pressure cone**’ (**arrows**), caused by the margins of the foramen magnum pressing on the cerebellar tonsils, which are displaced downwards through the foramen, compressing the medulla & causing ☠ death (**Why?**)

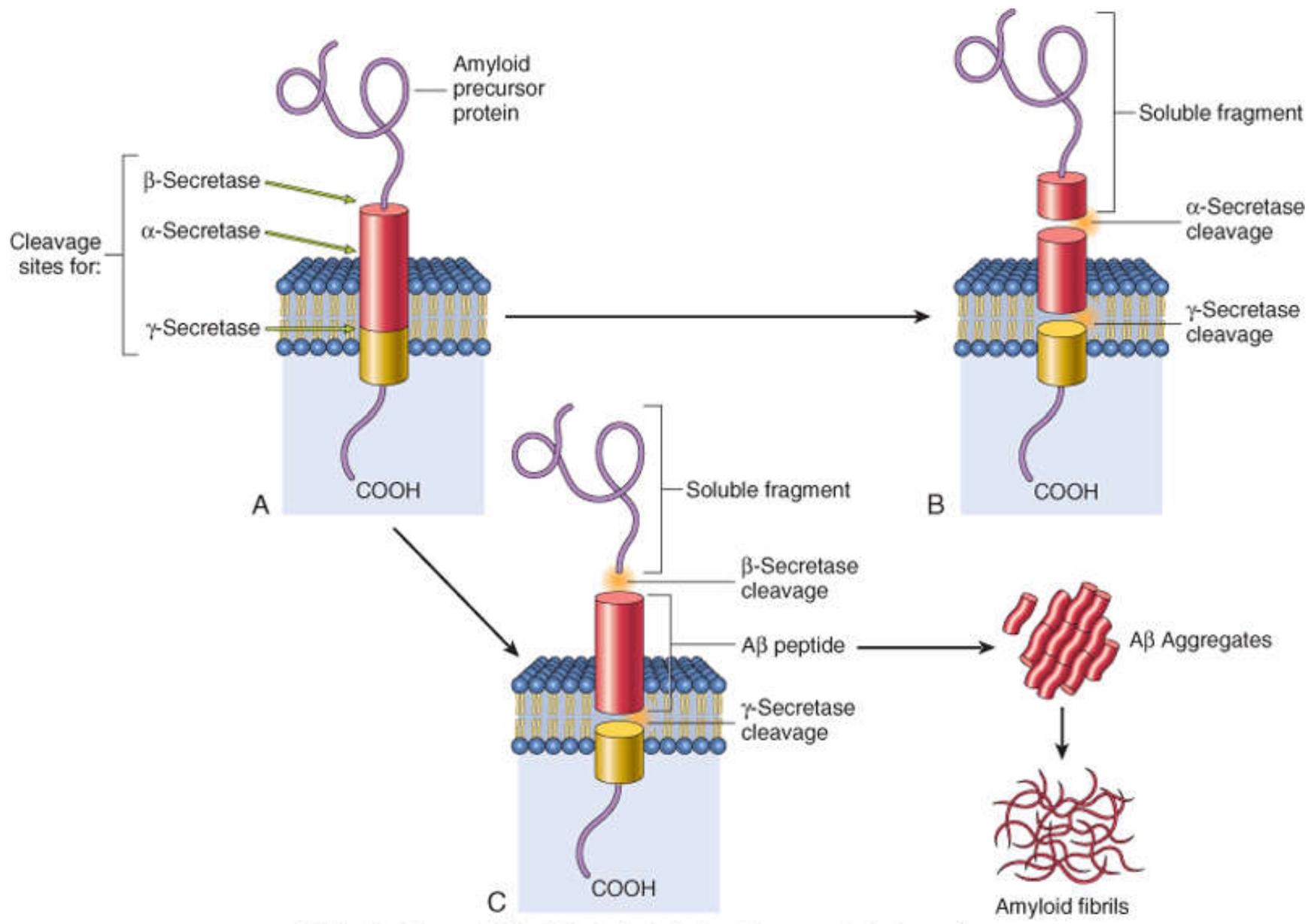


9.82 Tonsillar herniation (‘pressure cone’): cerebellum

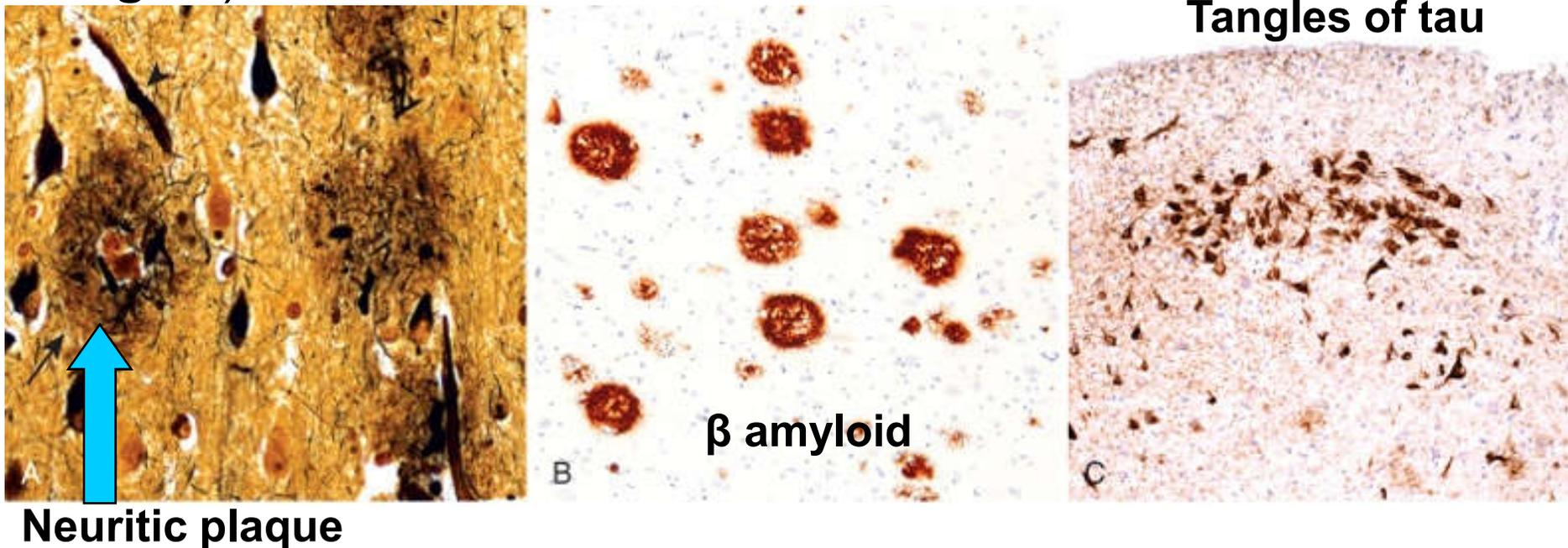
F1-4: **A, Normal** young adult brain., **B, Atrophy** of the brain in an 82 years-old male with atherosclerotic disease. Note that the loss of the brain substance (due to **aging & reduced blood supply**) narrows the gyri & widens the sulci.



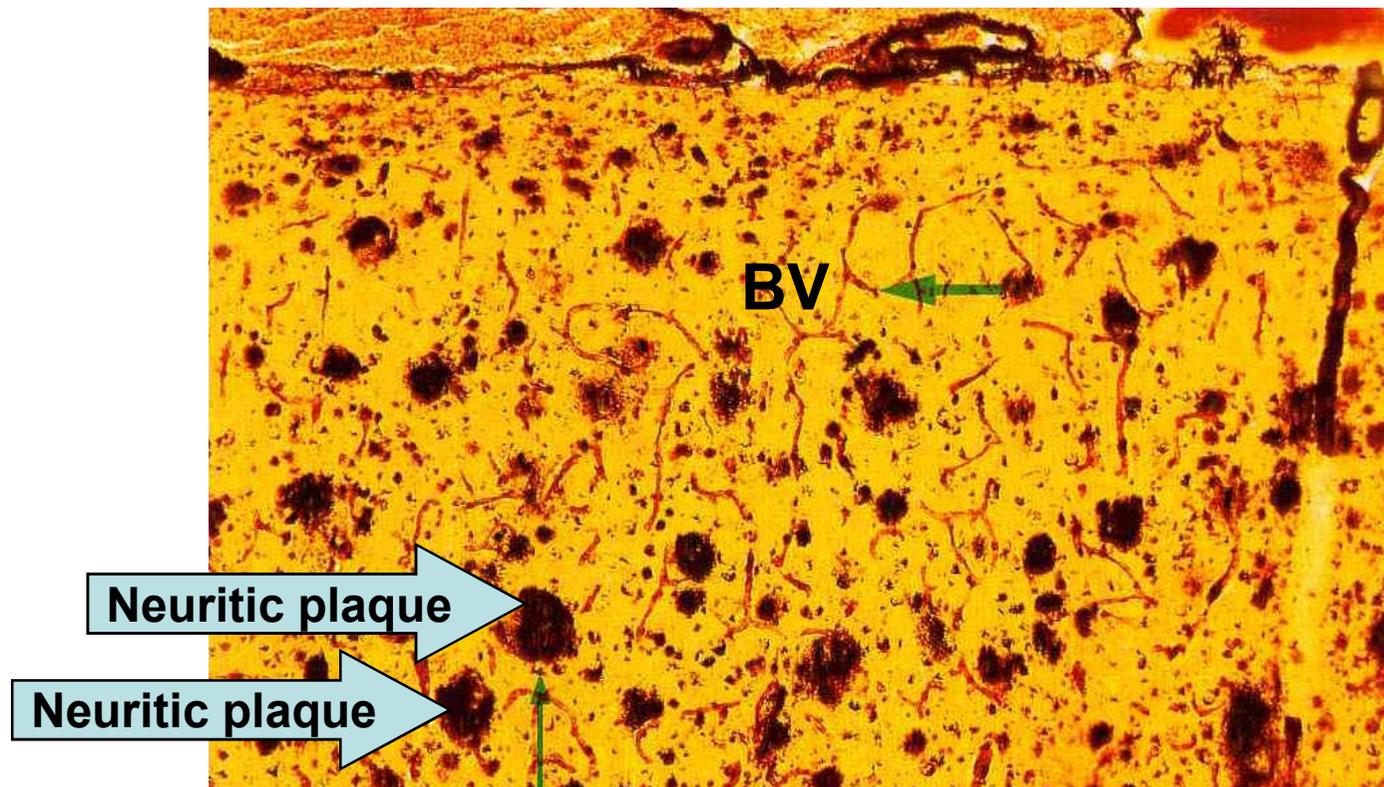
F23-28: Accumulation of the peptide (β amyloid, or $A\beta$) in the brain in Alzheimer disease.



F23-29: **Alzheimer disease. A, Neuritic plaque** {Bielschowsky stain, arrow} is 20 to 200 μm in \varnothing , focal spherical collections of dilated, tortuous, silver-staining dystrophic neurites & tangles which are filamentous extracellular inclusions, surrounding a central amyloid core,
B, Immunohistochemistry against **A β (β amyloid)** shows that the **A β** peptide is present in the core of the neuritic plaques & in the surrounding region.
C, Immuno stain for **tau** protein showing neurons containing **tangles**).



- 4.18: **Alzheimer disease: Brain X90.** Biopsy specimen from the cortex of a man of 63 stained by periodic acid silver method.
- The subarachnoid space & cortex surface are at top.
 - Many rounded & dark **neuritic plaques** (thin A) consisting of (I) *Central amyloid core* (contains **accumulated β amyloid**), (II) Surrounded by **Dystrophic neurites**, spherical collections of extracellular dilated, tortuous, silver-stained (argyrophilic) degenerated neuritic processes

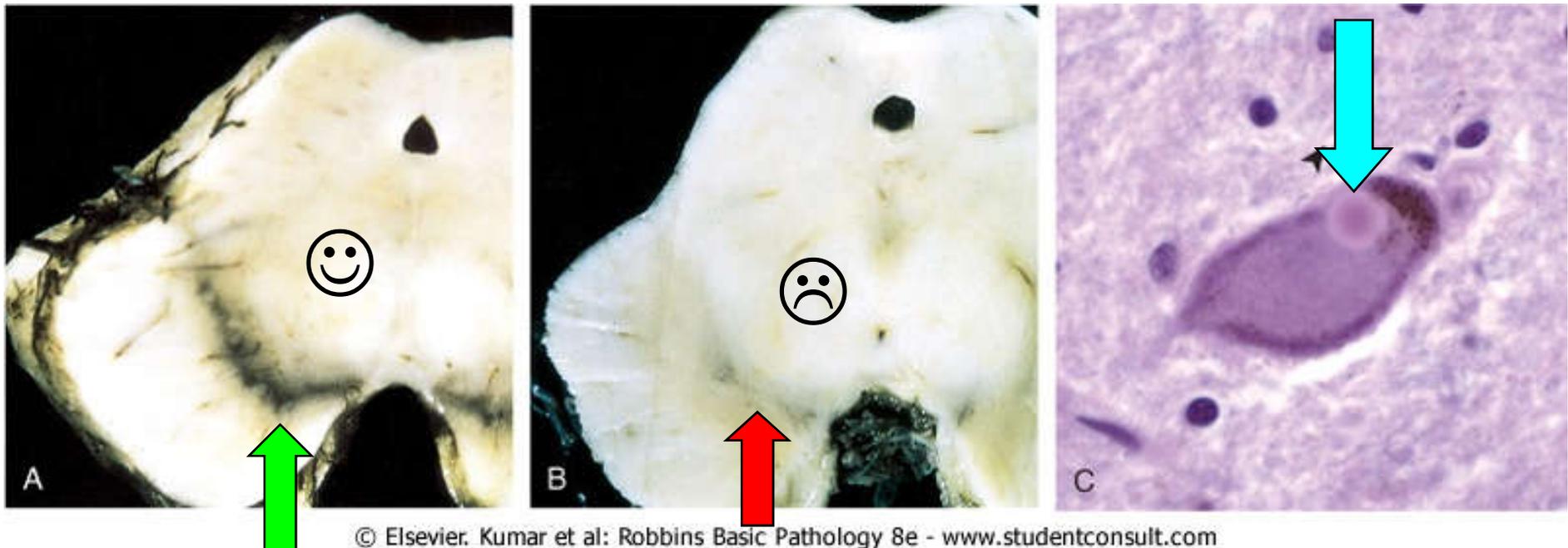


F23-30: Parkinson disease.

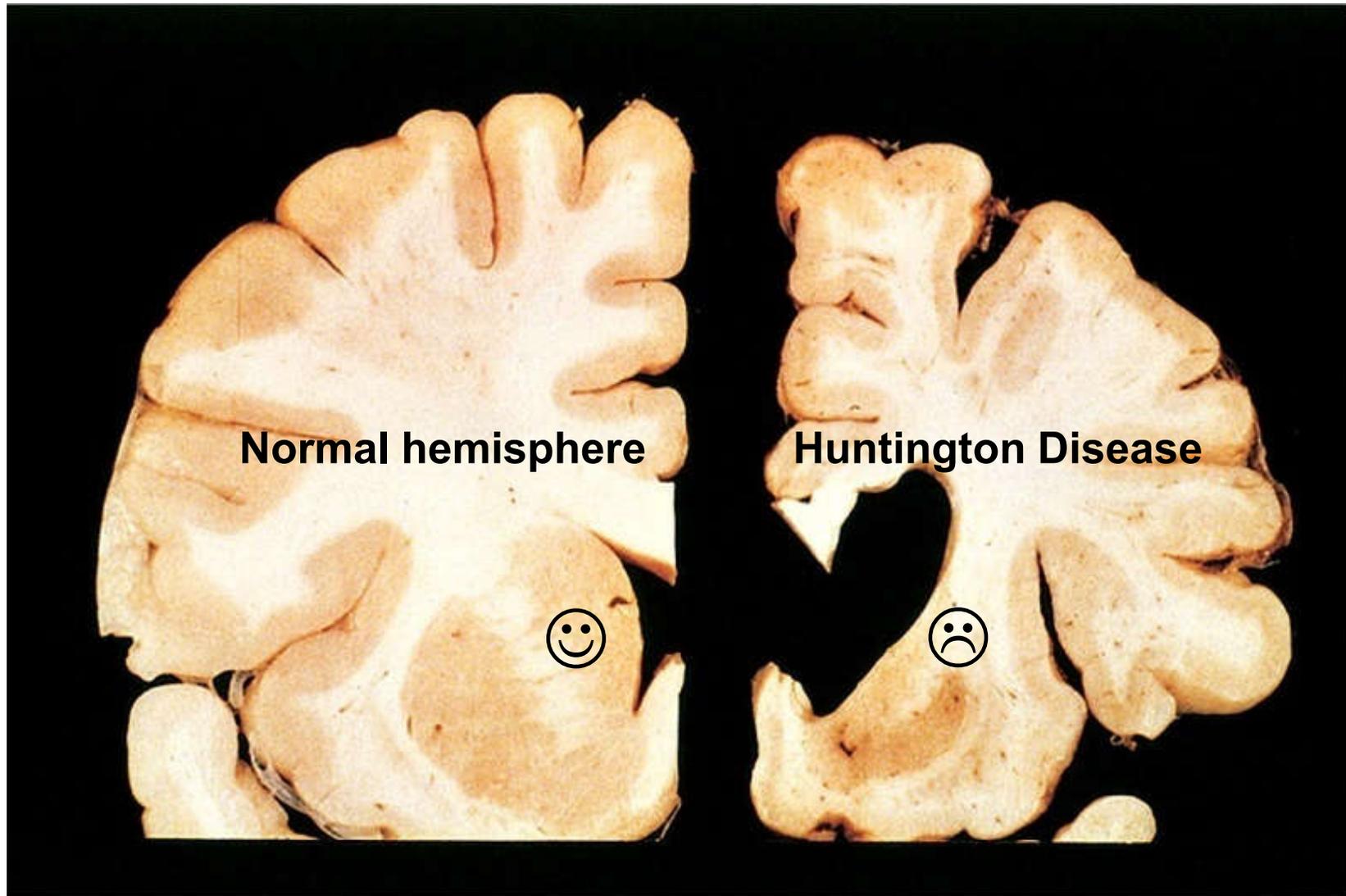
A, ☺ *Normal substantia nigra.*

B, ☹ *Depigmented* substantia nigra in idiopathic Parkinson disease.

C, *Lewy body* (arrow) in a neuron from the substantia nigra stains pink. An eosinophilic, round intracytoplasmic inclusion having dense core surrounded by a pale halo.



F23-31: Huntington disease. Normal hemisphere on the left compared with the hemisphere with Huntington disease on the right showing atrophy of the striatum & ventricular dilation



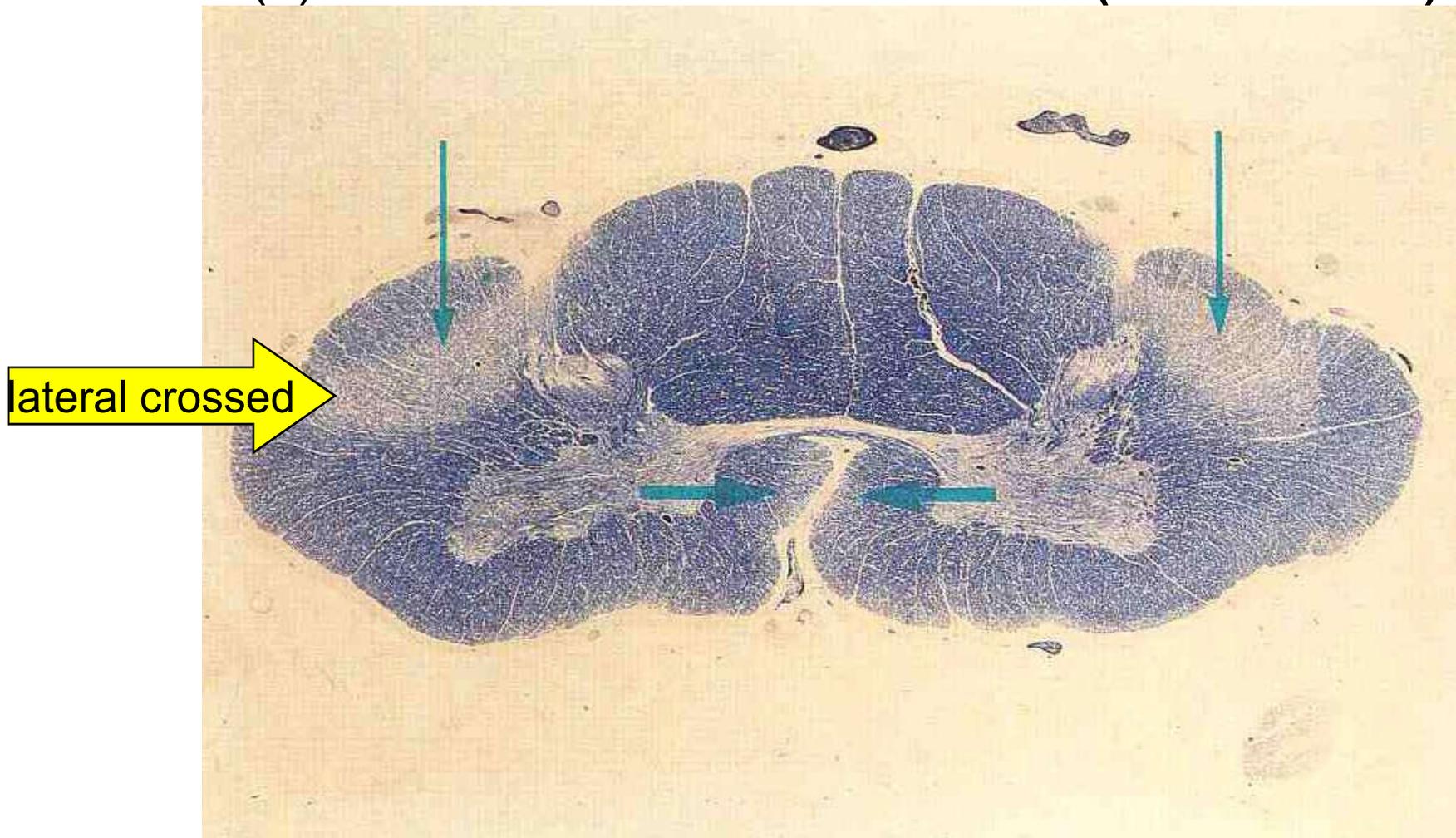
F9-25: Motor neuron disease: Ventral surface of spinal cord

☹ The anterior spinal nerve roots are atrophic & thin due to **reduction in the number of anterior horn cell neurons** throughout the length of the SC, with loss of anterior root myelinated fibers & reactive gliosis.

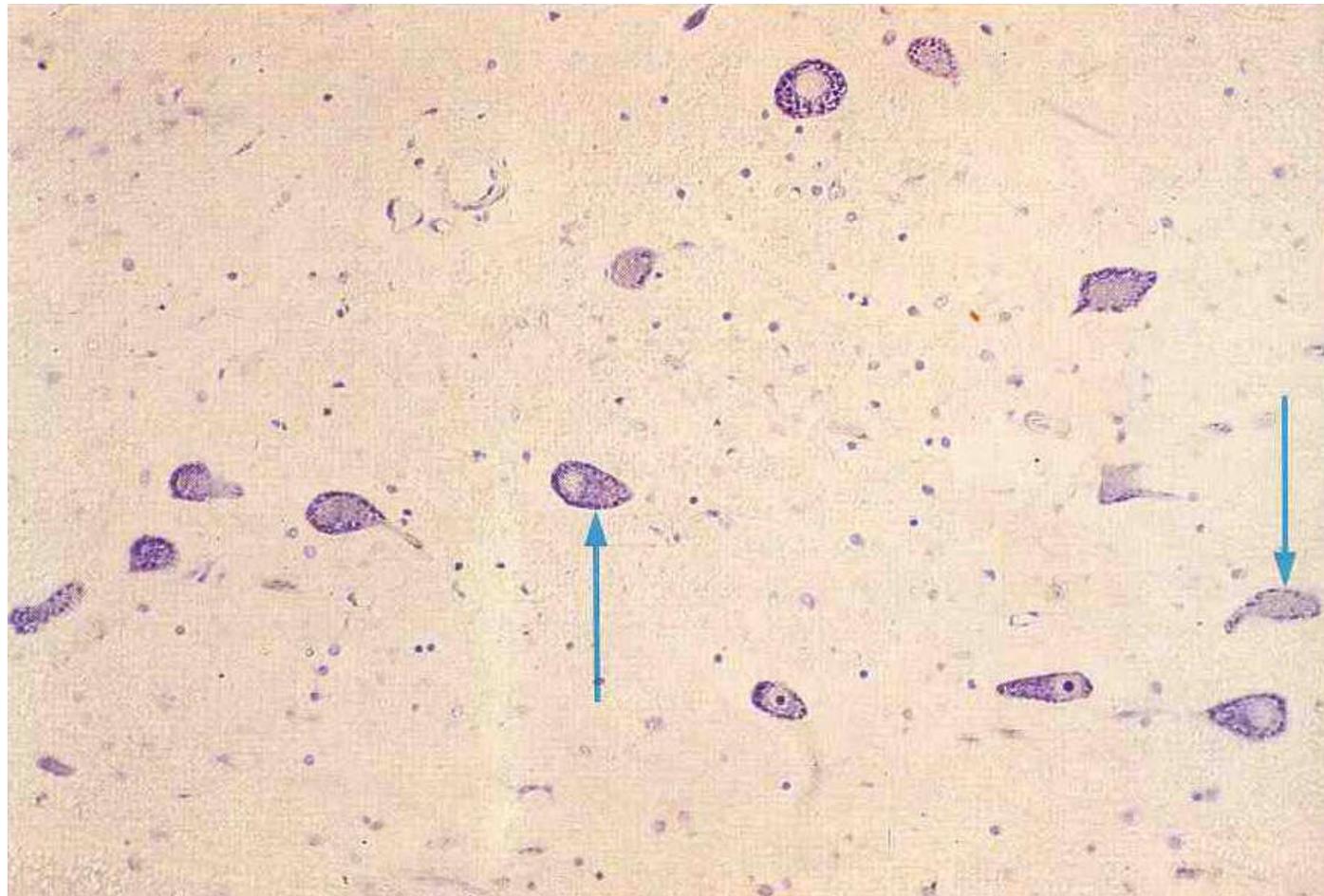


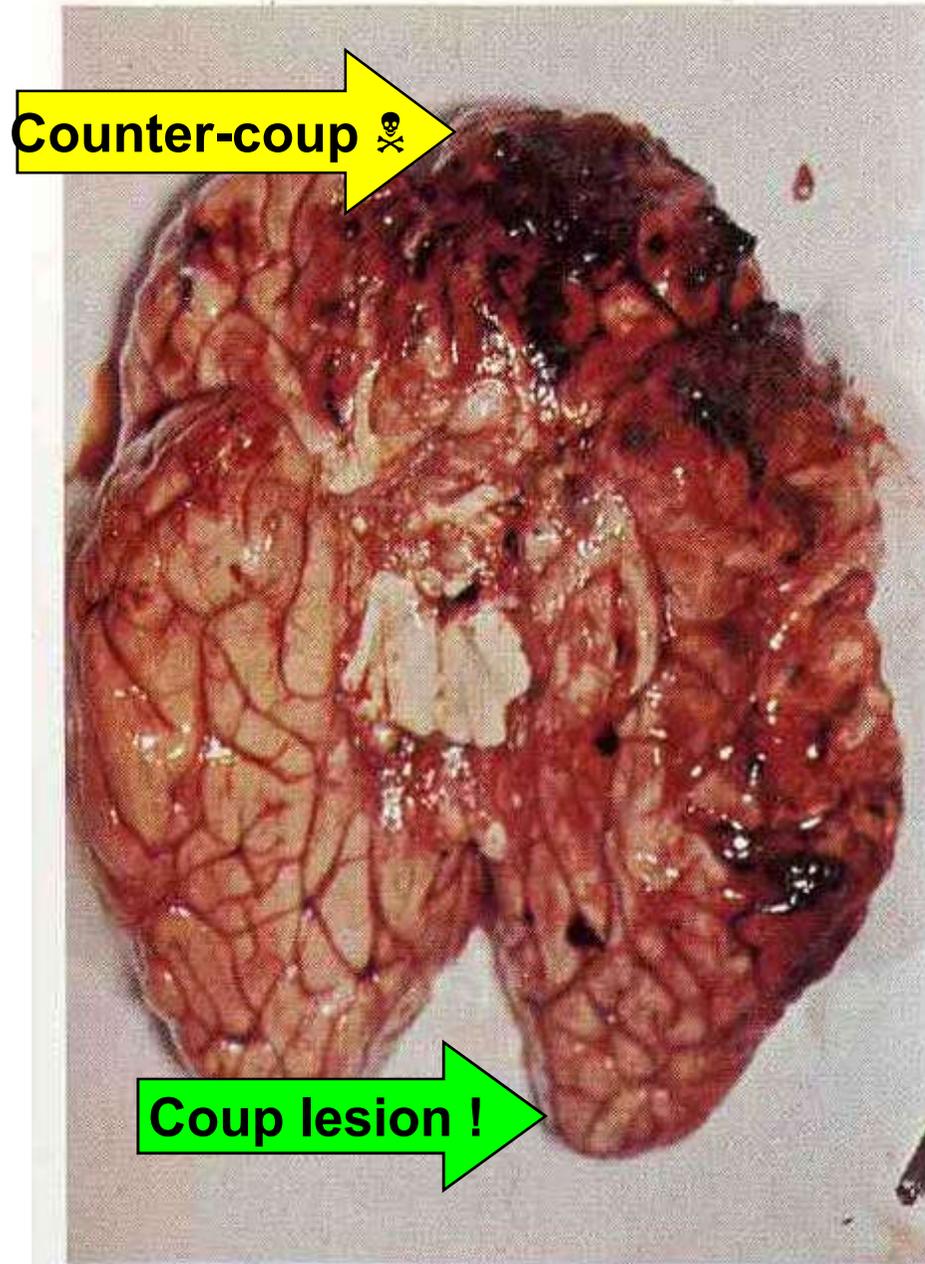
9.25 Motor neuron disease: spinal cord

■ 4.25: **Motor neuron disease (ALS): Spinal cord** section stained deep blue for myelin. There is loss of staining (demyelination with pallor) affecting both the **(I) lateral crossed cerebrospinal tracts (thin arrows)**, which is more pronounced than the **(II) anterior columns direct tracts (thick arrows)**



■ 4.26: **Motor neuron disease (ALS): Spinal cord** section, showing anterior horn from a patient, who had progressive muscular atrophy, stained with thionin to demonstrate the motor neurons selectively. The number of motor neurons is much less than normal & the few which remain are degenerated, shrunken (arrows) showing chromatolysis & karyolysis.

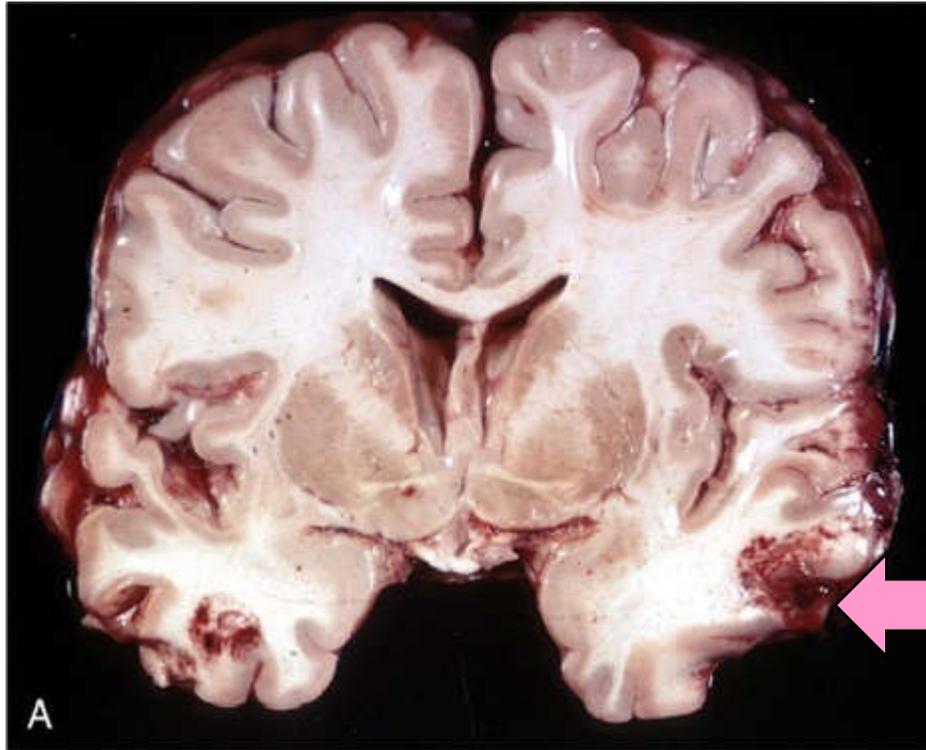




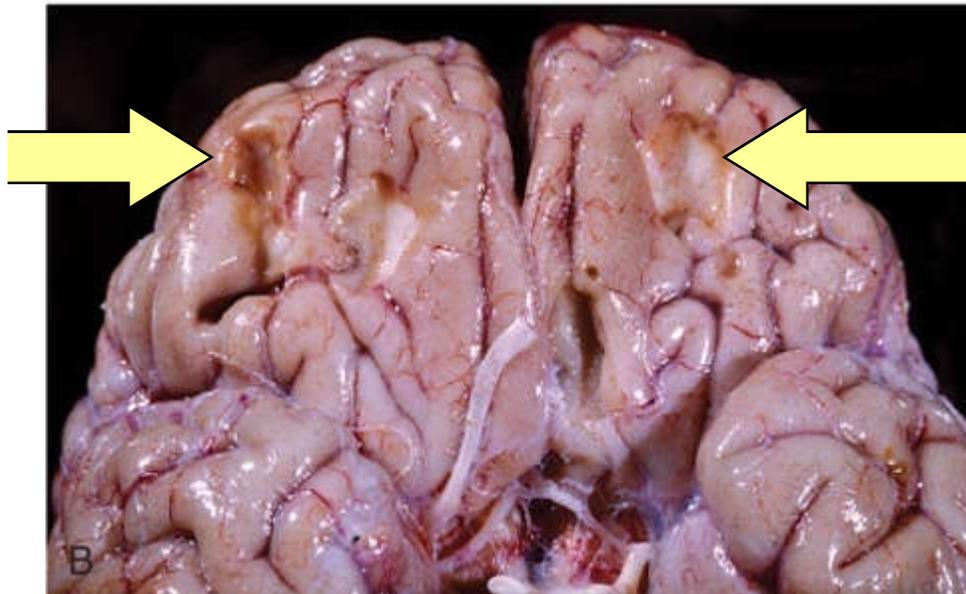
F 9-19: Contusions: brain.

★ This patient sustained severe **trauma to the left occipital region of the head** which has caused extensive fronto-temporal (Counter-coup) contusions & lacerations of the brain (at top center & bottom right).

☺ In this case, the counter-coup lesion is **much more extensive than the coup lesion** which are at the point of impact (occipital area)



F23-12: Cerebral trauma.
A, Acute contusions with areas of hemorrhage & tissue disruption, present in both temporal lobes,



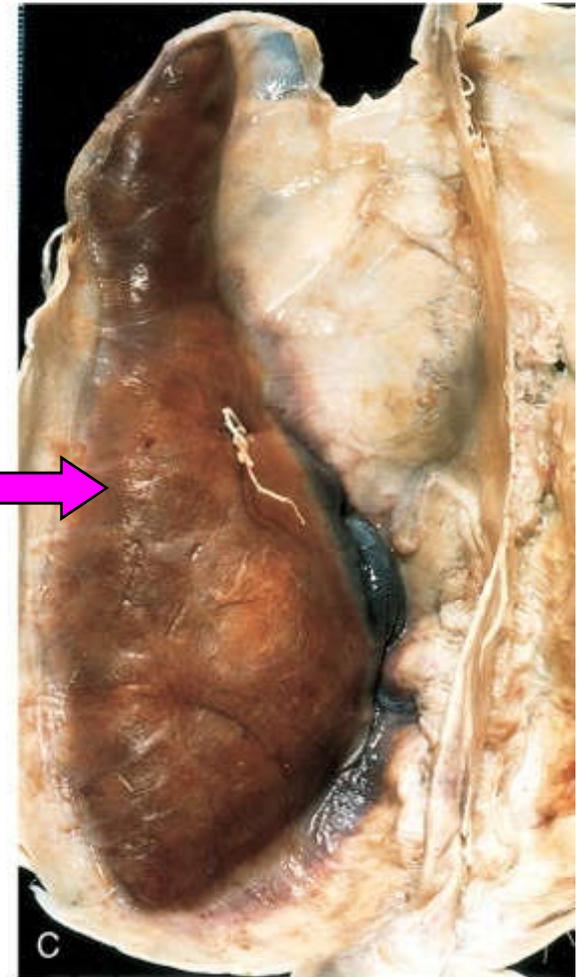
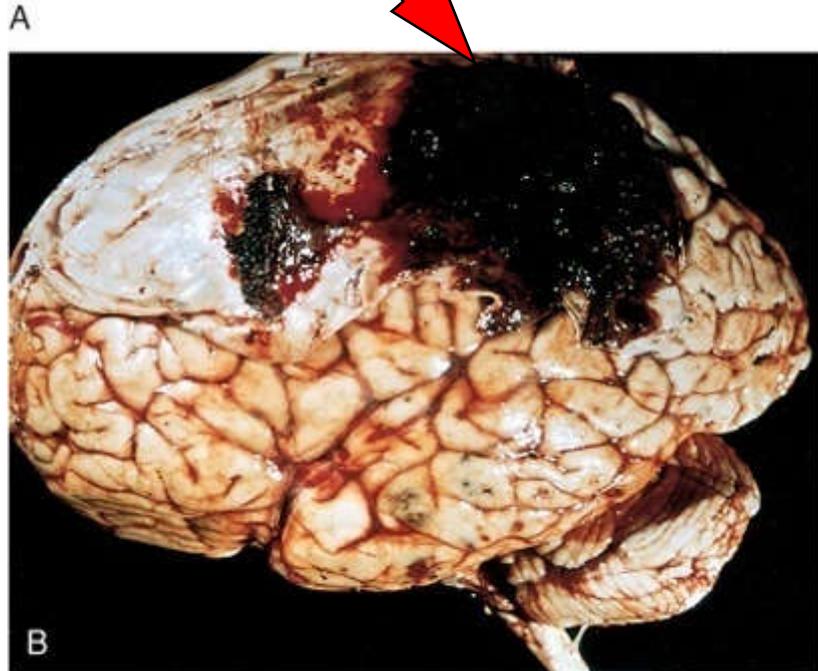
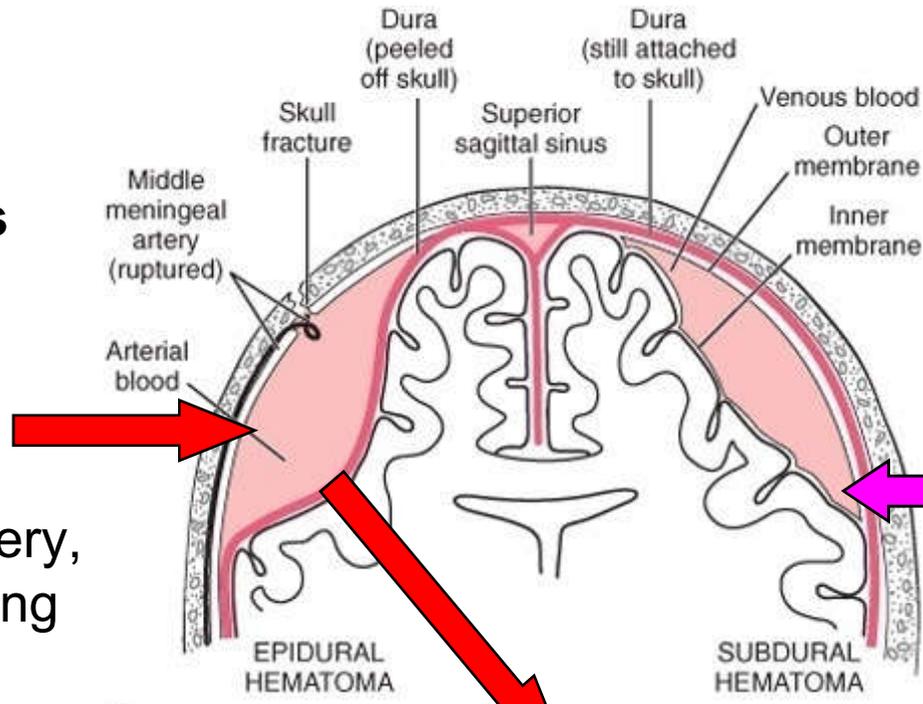
B, Old contusions are present on the inferior, (orbital gyri) of frontal lobes surfaces of this brain.

**F23-13:
Traumatic
intracranial
hemorrhages**

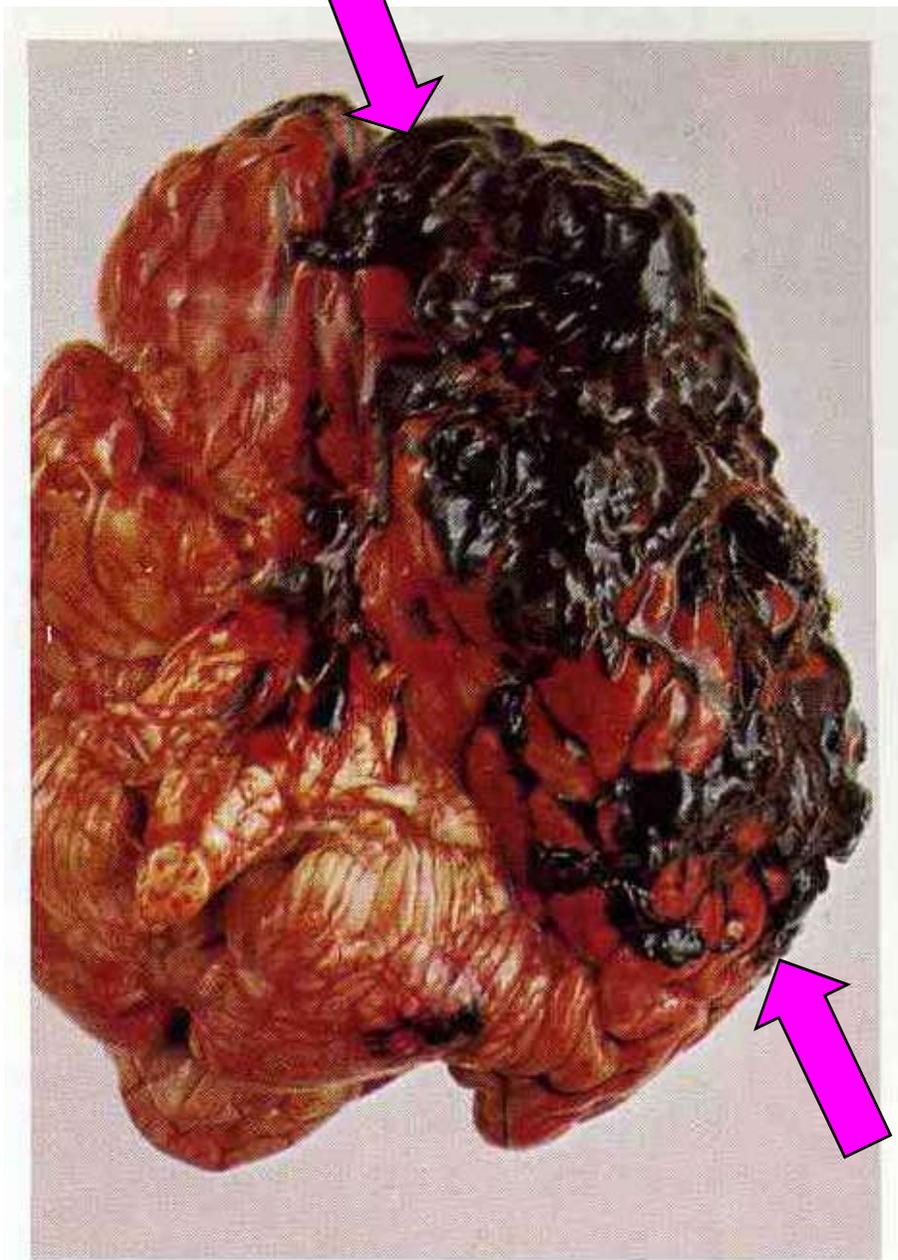
**A, Epidural
hematoma**

Rupture of a middle meningeal artery, usually following skull fracture, leads to accumulation of arterial blood between the dura & the skull.

**B, Epidural
hematoma**
covering a
portion of the
dura.



**C, Large organizing
subdural
hematoma**
attached to the dura



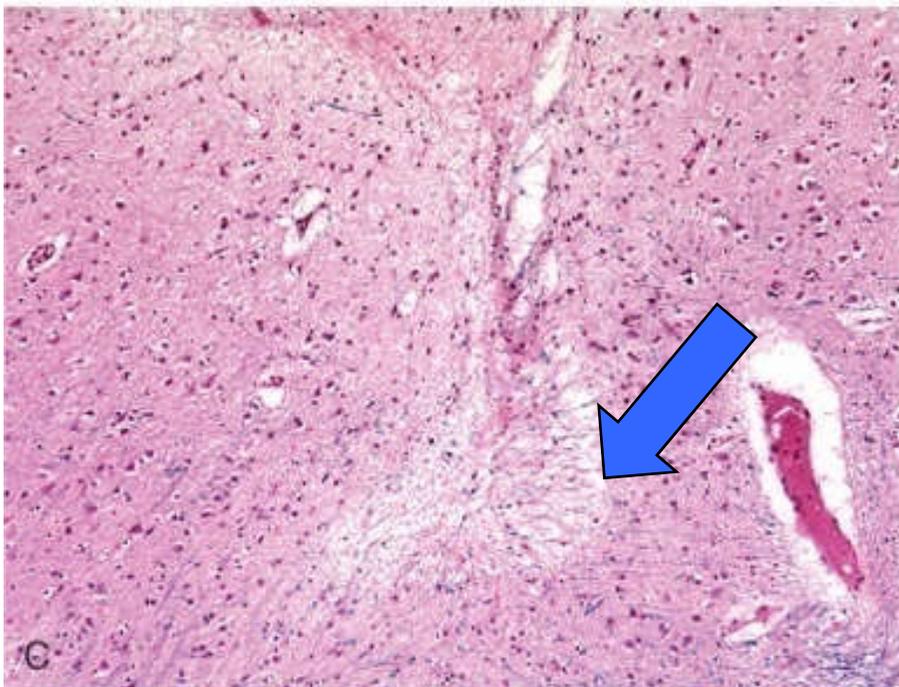
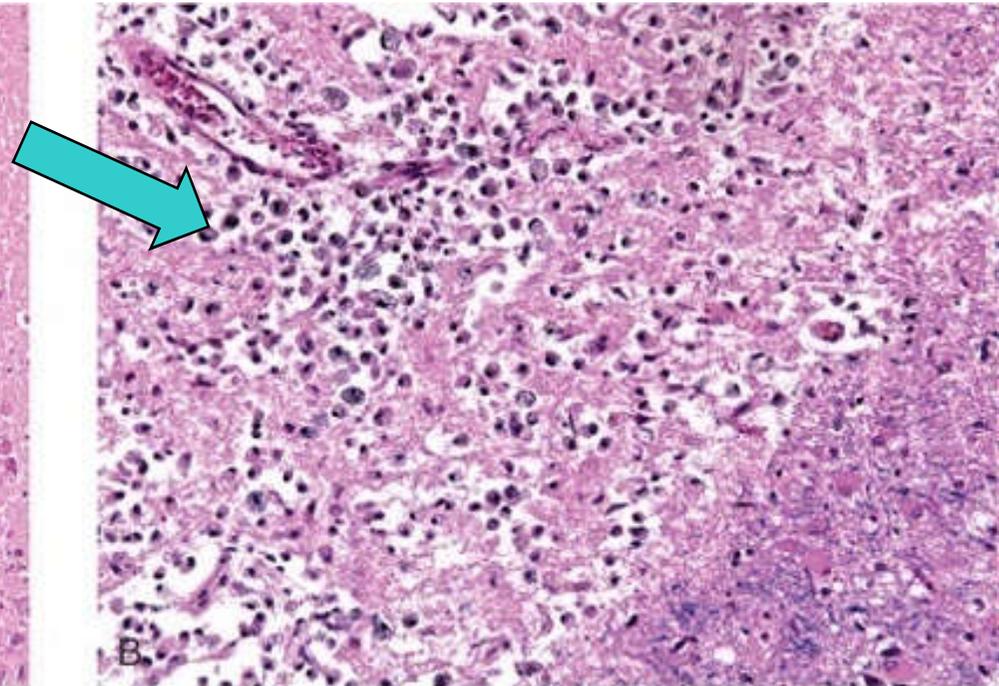
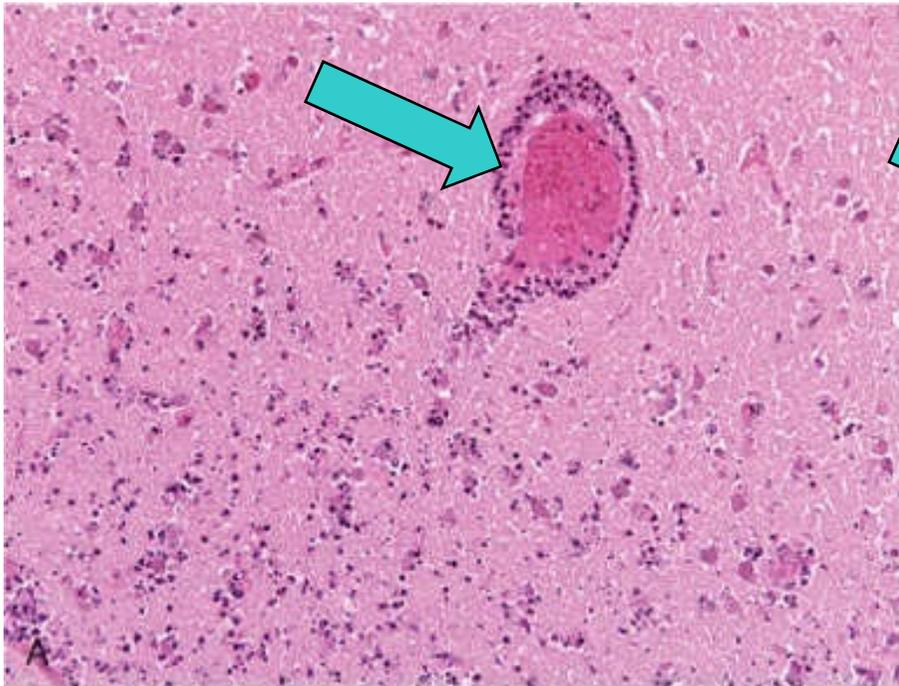
9.21 Subdural haemorrhage (subdural haematoma): brain

F 9-21: **Subdural hemorrhage (hematoma): brain.**

☠ A massive subdural hemorrhage over the left **fronto, temporo, parietal** regions extends over the inferior surface of the hemisphere.

★ **Most** subdural hemorrhages follow **blunt** injury to the skull ; & in the elderly they may occur without a history of direct injury to the head.

But in this case, the cause was an ☠ **extensive fracture of the skull.**



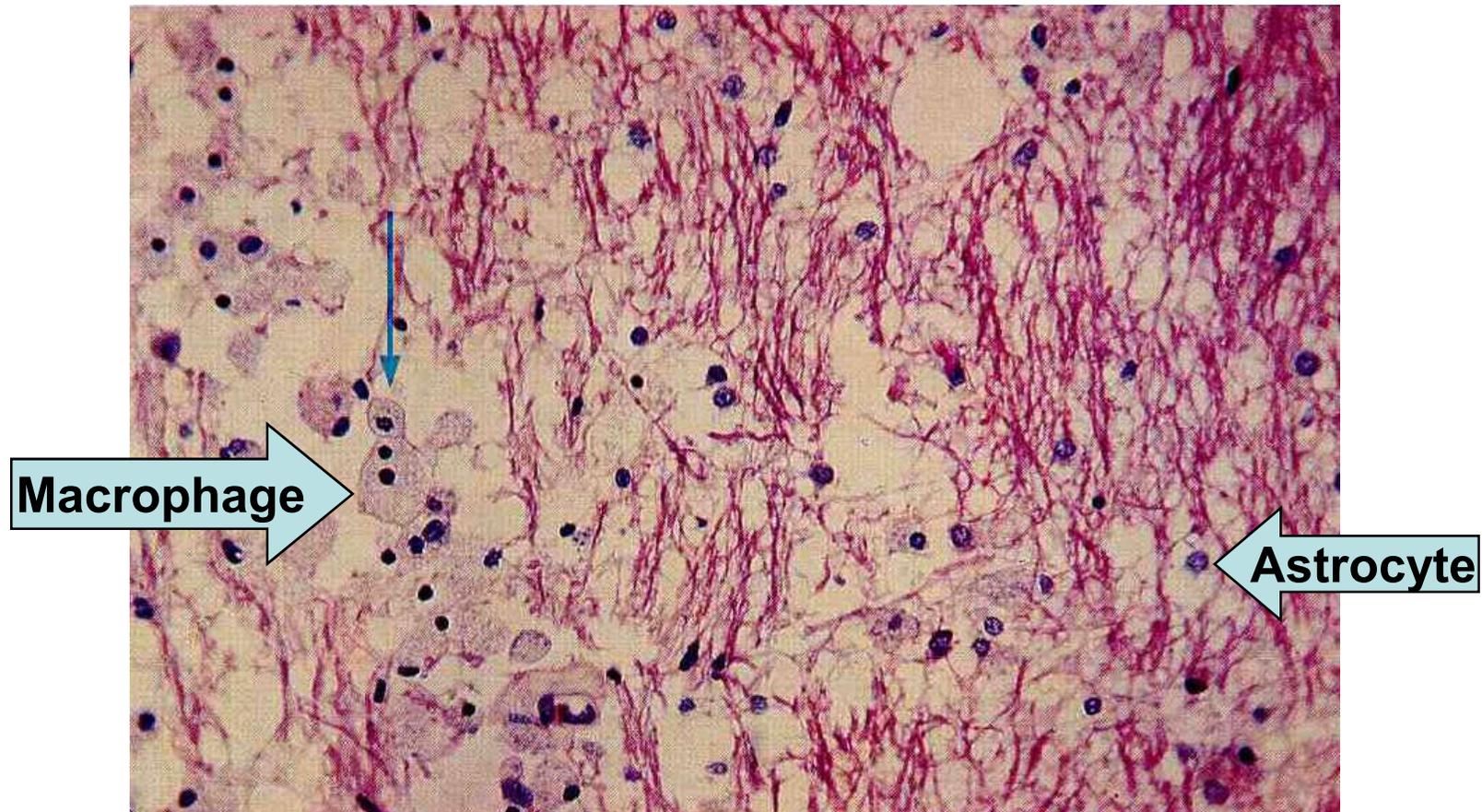
F 23-6: Cerebral infarction (CI) .

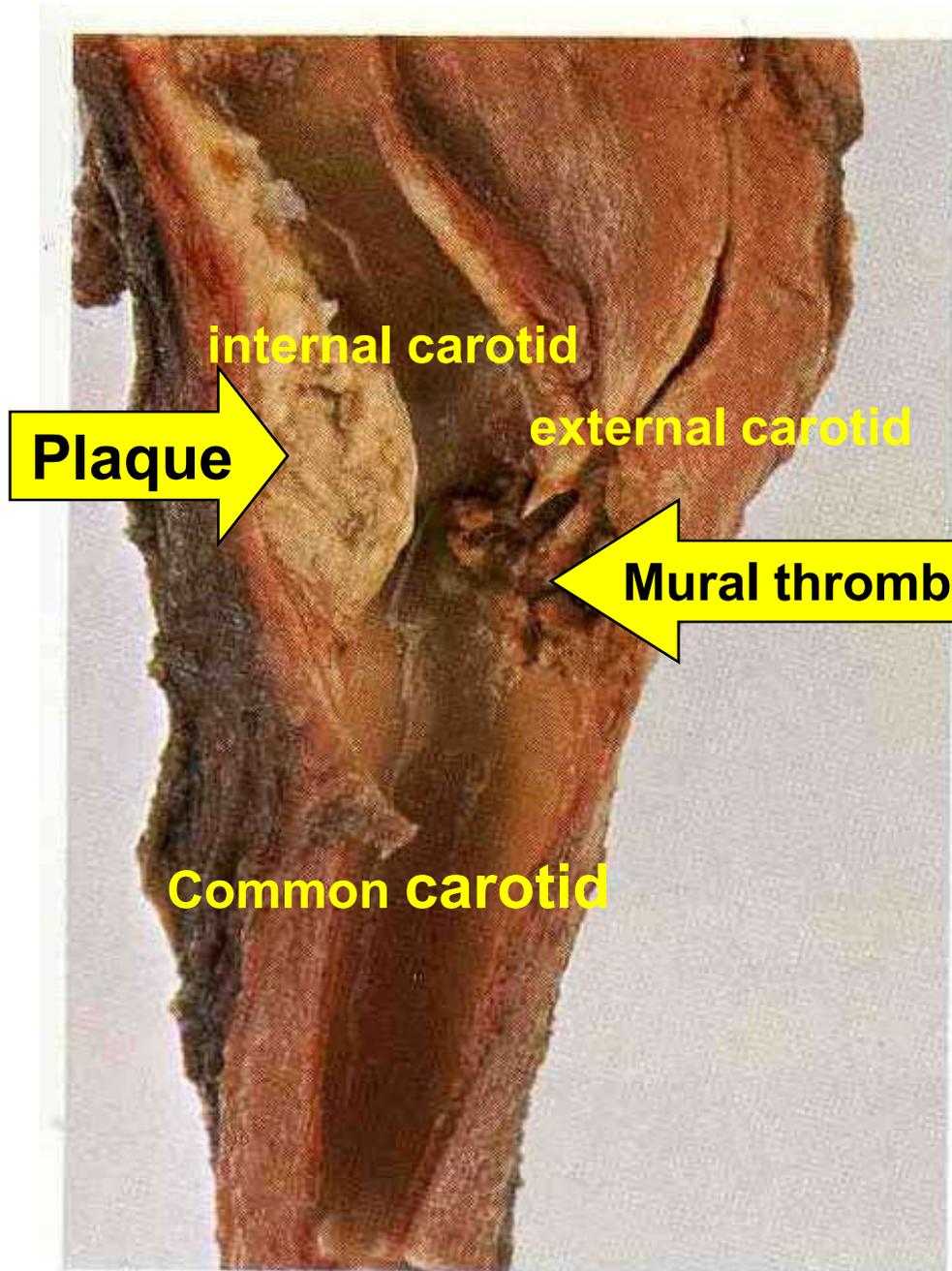
A, Recent CI infiltrated by neutrophils, begins at the edges of the lesion from intact BV.

B, After 10 days, the CI is infiltrated by macrophages & surrounded by reactive gliosis.

C, Old small intracortical CI seen as areas of tissue loss with a small amount of residual gliosis.

■4.4; **Brain infarction** of 6 weeks duration X335. White matter
(I) Most **myelinated fibers** undergone ischemic necrosis &
disappeared. (II) large round **Macrophages** with foamy
cytoplasm (from phagocytosed lipoproteins of the necrotic
tissue), lying in the spaces between the surviving fibers.
(III) **Astrocytes** with small round basophilic nuclei & ill-defined
cytoplasmic boundaries.





F6.61: Yellow-white

☹ calcified

atheromatous plaque

at the origin of the Rt
internal carotid artery

(top left), causing
marked luminal
stenosis. Opposite the

plaque, at the origin of
the Rt. external carotid

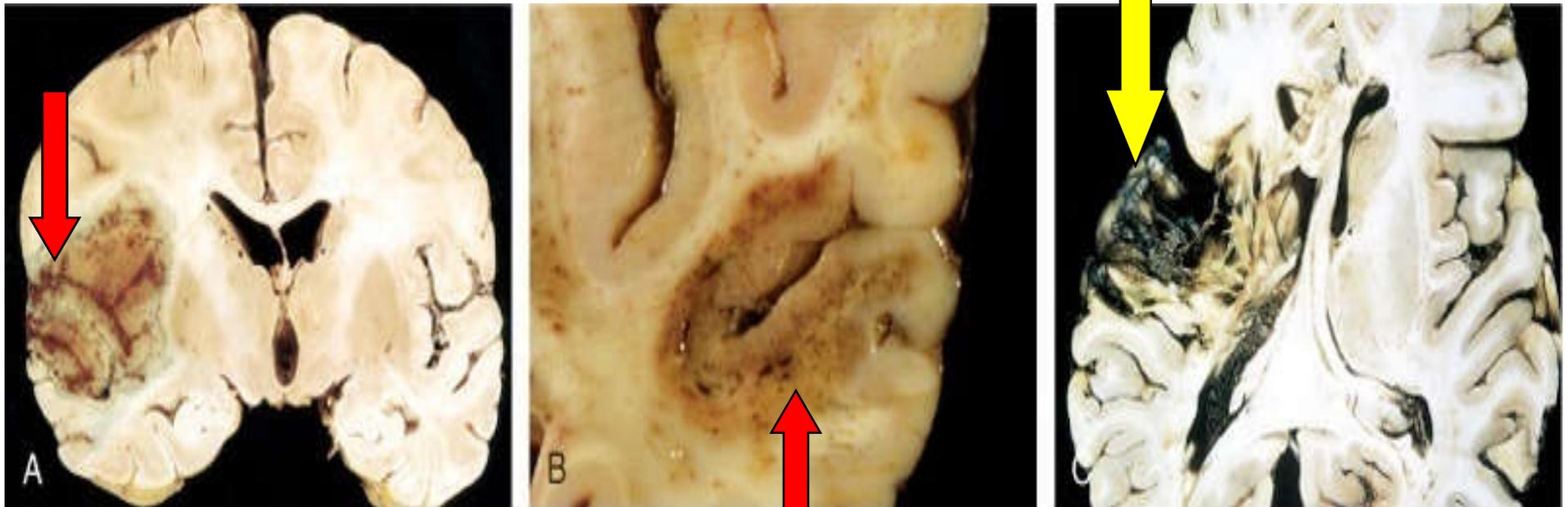
artery, there is an
irregular small brown

mass of ☹ mural

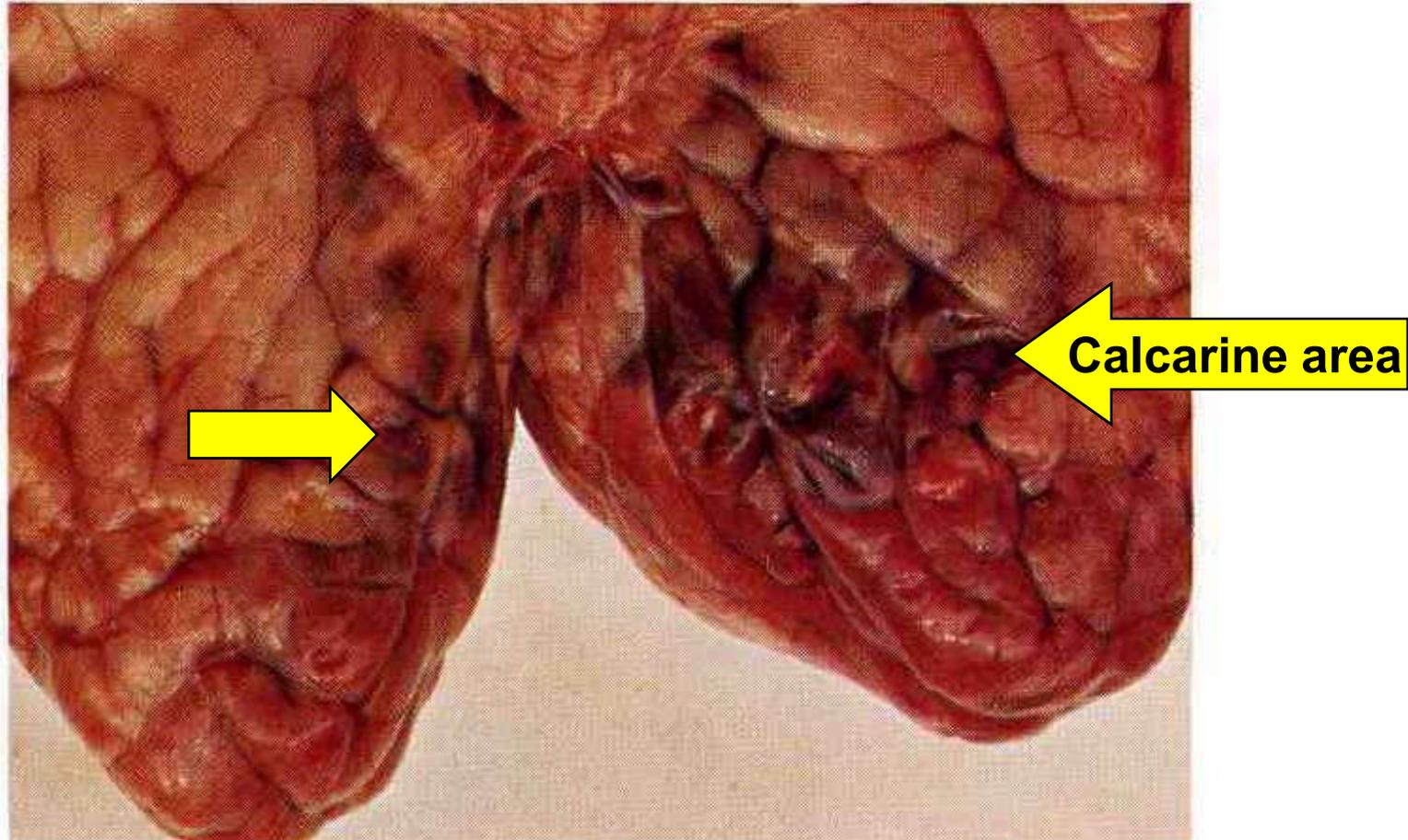
thrombosis.

6.61 Atherosclerosis: carotid arteries

F23-7: Cerebral infarction. Brain sections showing:
A, Large red hemorrhagic infarct in the distribution of the left middle cerebral artery.
B, Temporal lobe red infarct, with punctate hemorrhages, due to **ischemia-reperfusion injury**,
C, Old cystic infarct, shows destruction of cortex & surrounding gliosis.

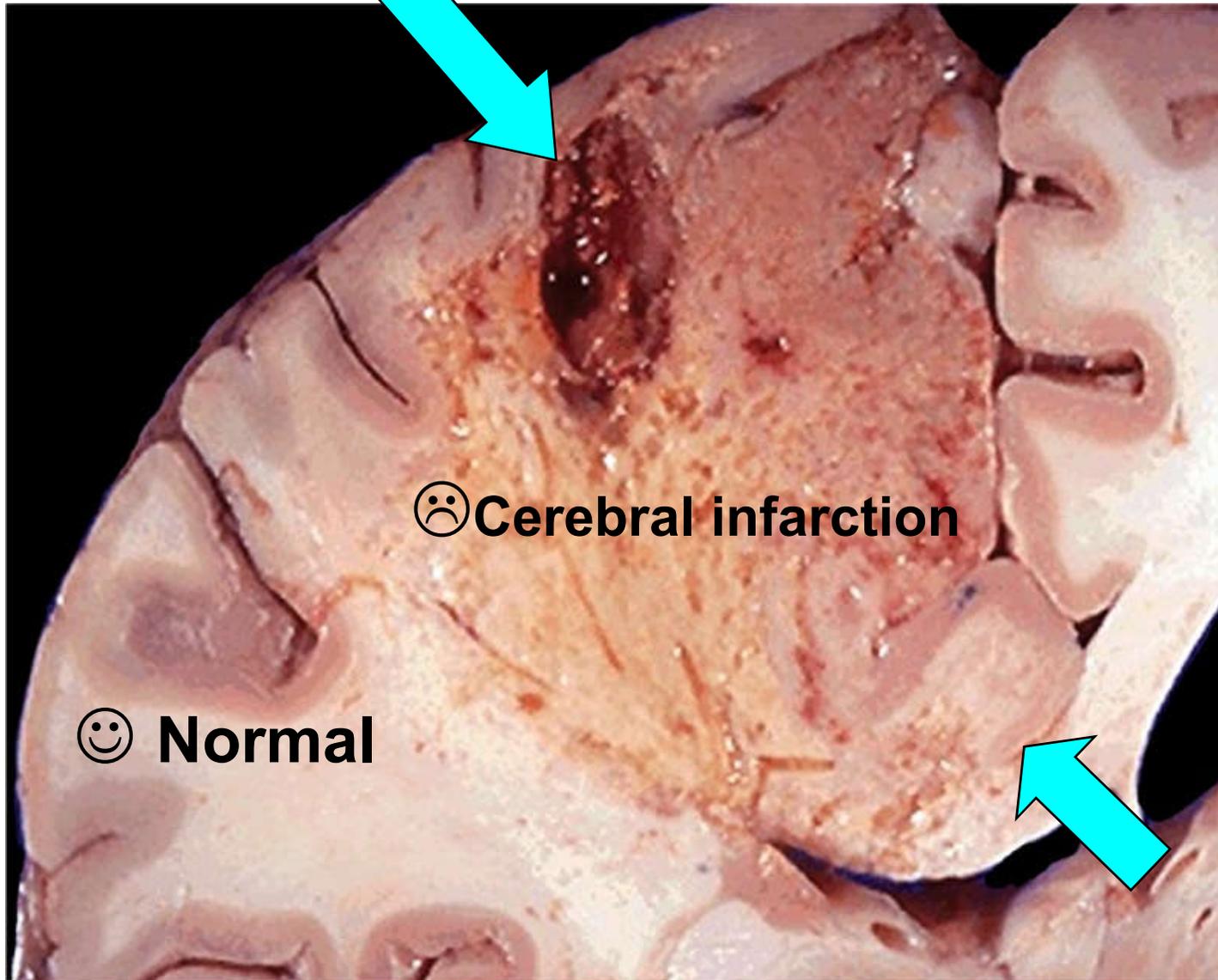


F 9.47: Infarction: Brain. ☠ The patient had transtentorial herniation obstructing the posterior cerebral arteries, resulting in **recent hemorrhagic infarction** of the infero-medial aspects of both occipital lobes, especially affecting the calcarine area.



9.47 Infarction: brain

F1-11: **Brain:** Cerebral infarct, 10 to 21 days after stroke, liquefactive necrosis of the brain tissue, eventually leaving a **fluid-filled cavity**.



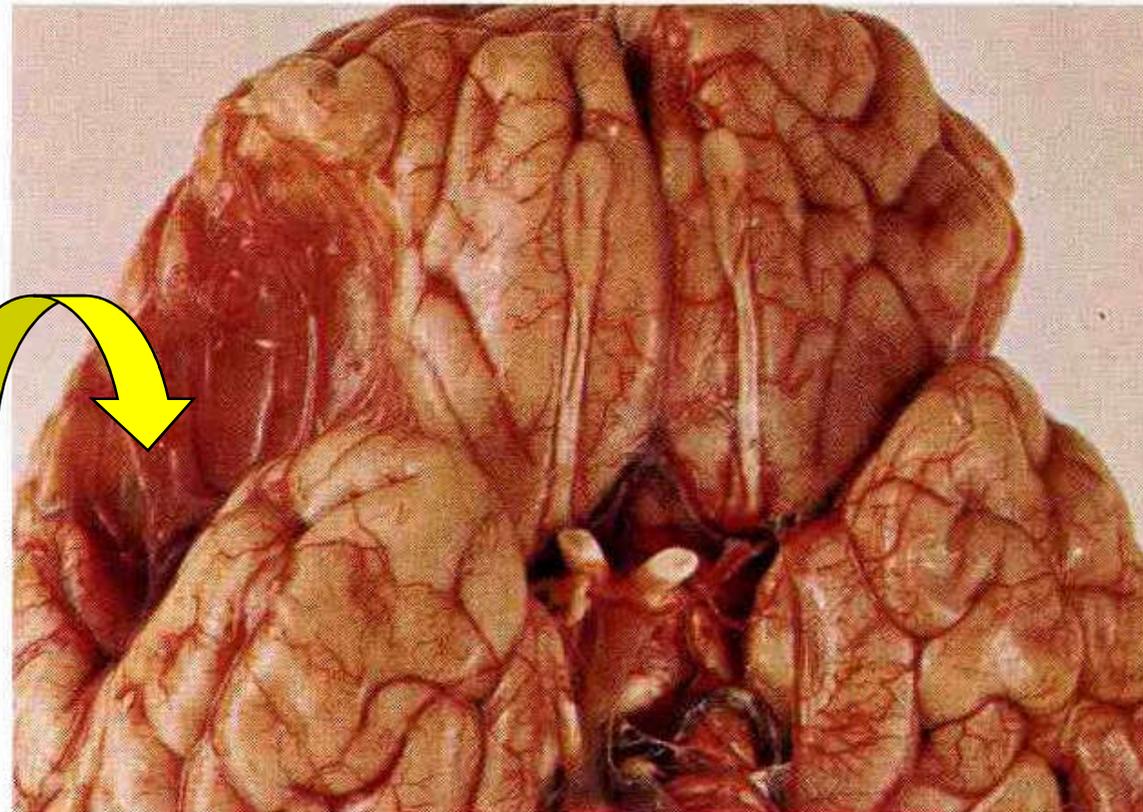
**Normal
Cerebral
grey &
white
matter.**

☹️ **Cerebral infarction**

😊 **Normal**

F 9.46: Infarction: Brain. The patient had chronic RHD with left **atrial thrombus** ⇒ **embolization** of which in the ⇒ Rt. middle cerebral artery causes large **infarction** of the ⇒ inferior aspect of the right fronto-temporal region. The **old infarct** appears as a large '**cavity**', covered by a thin, brown membrane, which is either filled with clear fluid or, appears collapsed (as here).

Old infarct

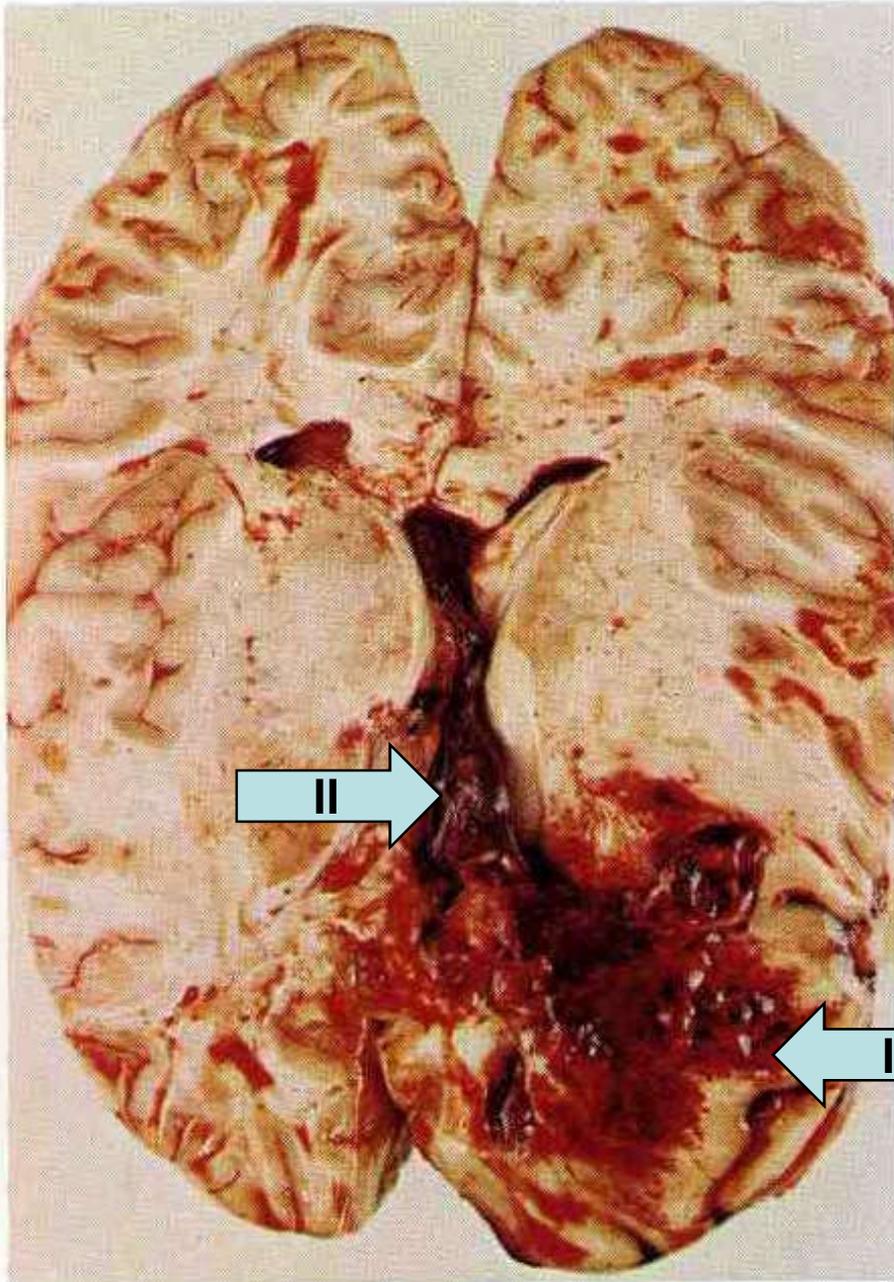


9.46 Infarction: brain



F23-8: Cerebral hemorrhage.

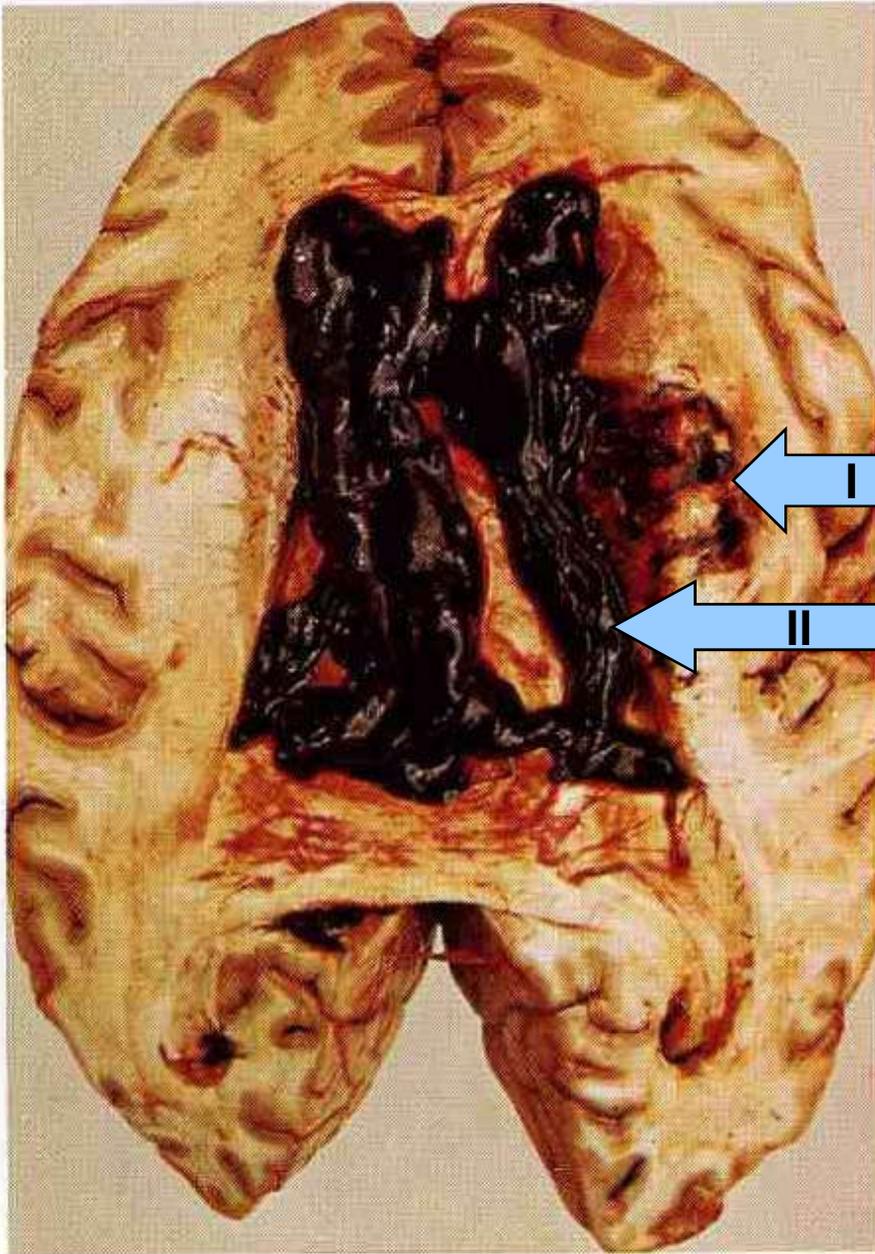
Massive hypertensive hemorrhage rupturing into a lateral ventricle.



F 9-41: Recent intracerebral hemorrhage: brain.

(I) Recent, large hemorrhage in the right occipital pole, extending to **(II)** the lateral ventricle.

9.41 Intracerebral haemorrhage: brain

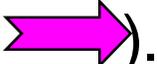


F 9-42: Intracerebral hemorrhage: brain.

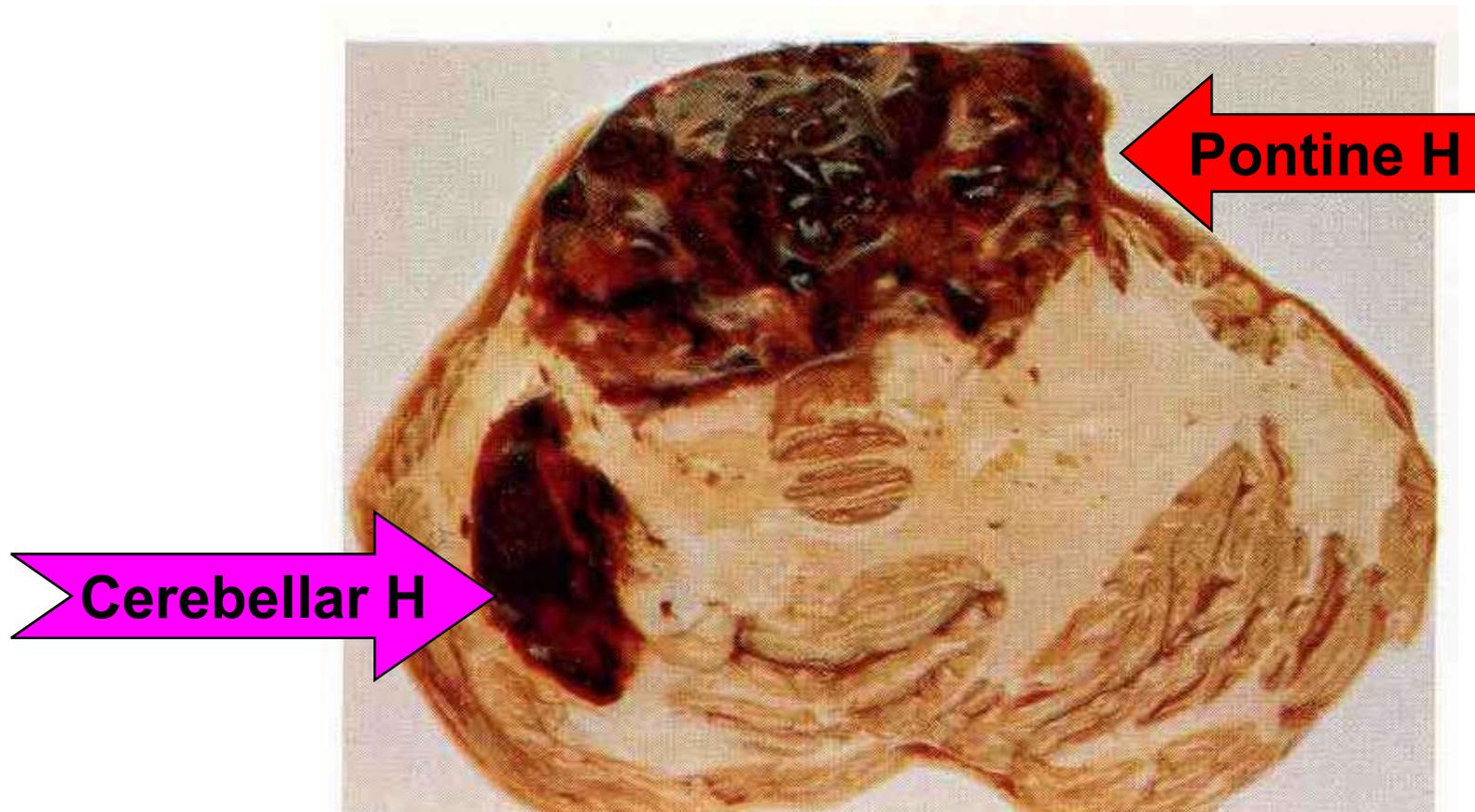
(I) There is ragged hypertensive intracerebral hemorrhage in the region of **right lentiform nucleus** (top right) which ruptured into & fills **(II)** both lateral ventricles.

NB. This **intraventricular hemorrhage** may pass through the foramina of the fourth ventricle **into...Where?**

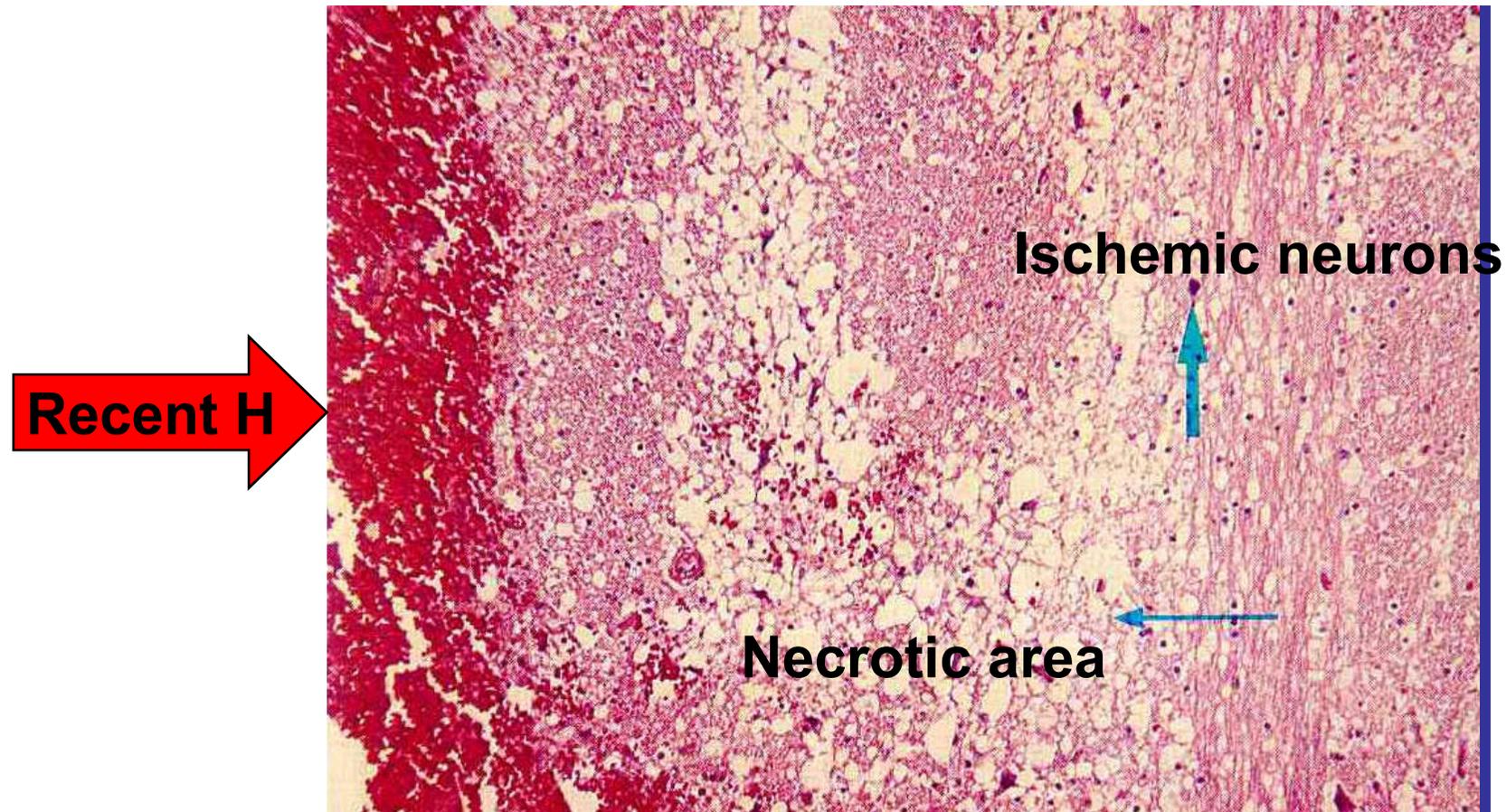
9.42 Intracerebral haemorrhage: brain

F 9- 43: **Massive** (more than 1.5 cm in \varnothing) **recent hemorrhage** destroying the **pons** (above ), & with 2nd hemorrhage in the central white matter of the **cerebellar hemisphere**().

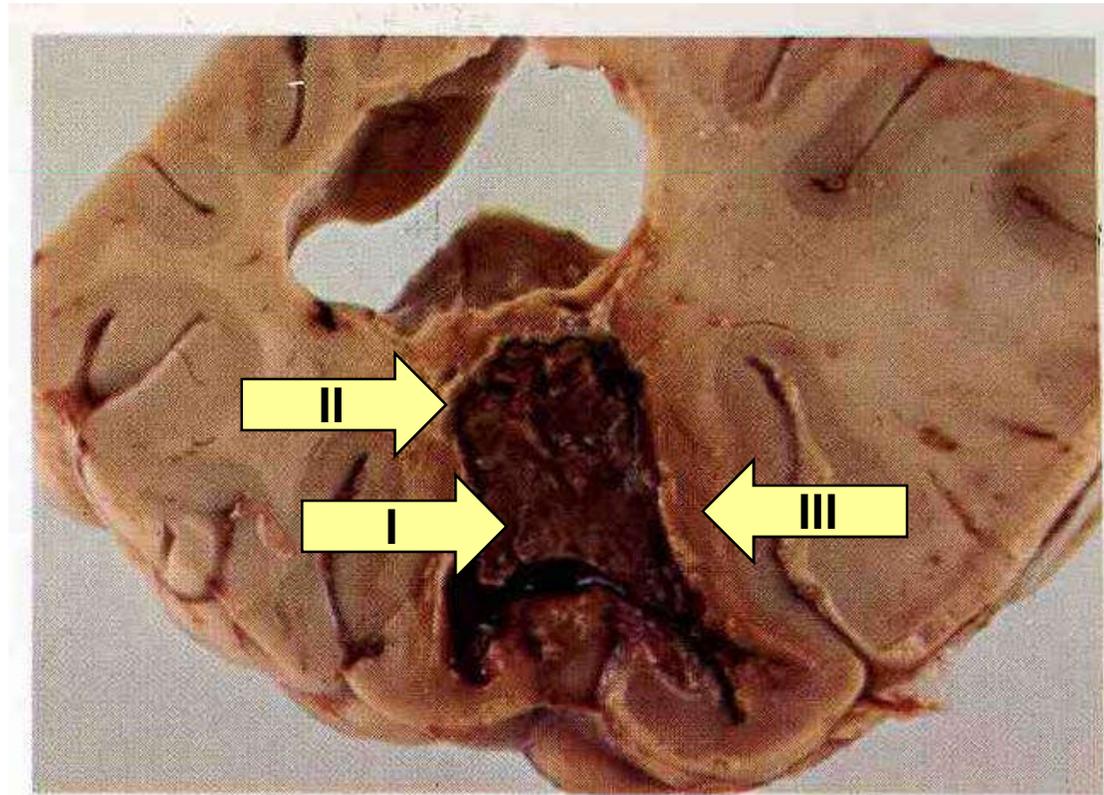
☠ The occurrence of **any one** of the above 2 hemorrhages alone, is almost always, rapidly ☠ fatal.



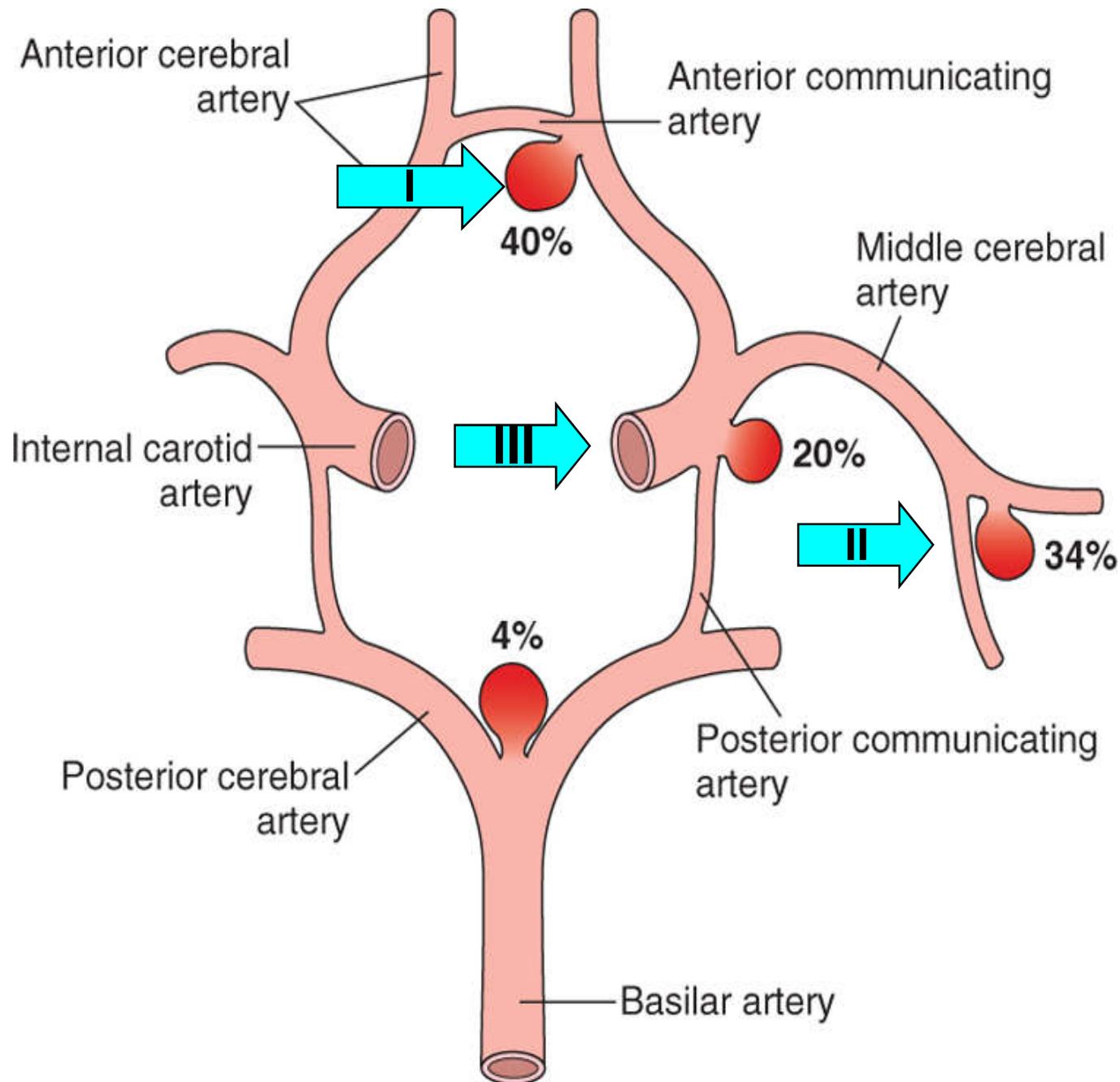
■ 4.5: **Recent Cerebral hemorrhage: Brain X145.** Edge of the hemorrhage. On the left, there is red zone of recent blood clot. Adjacent to it, there is an extensive necrotic area (thin arrow), pale, edematous & vacuolated (vacuoles contain water) & many neurons & glial cells have disappeared. Few ischemic neurons survive as basophilic round bodies (thick arrow)



F 9-44: **Old intracerebral hemorrhage: brain.** Coronal section of the occipital lobe, showing partly-organized old hemorrhage: **(I)** Central brown **hematoma** retracted from the surrounding brain, **(II)** thick **capsule** of reactionary astrocytic proliferation, **(III)** **both**, the capsule & the adjacent brain are stained **golden-brown** by breakdown products of hemoglobin.



9.44 Intracerebral haemorrhage: brain



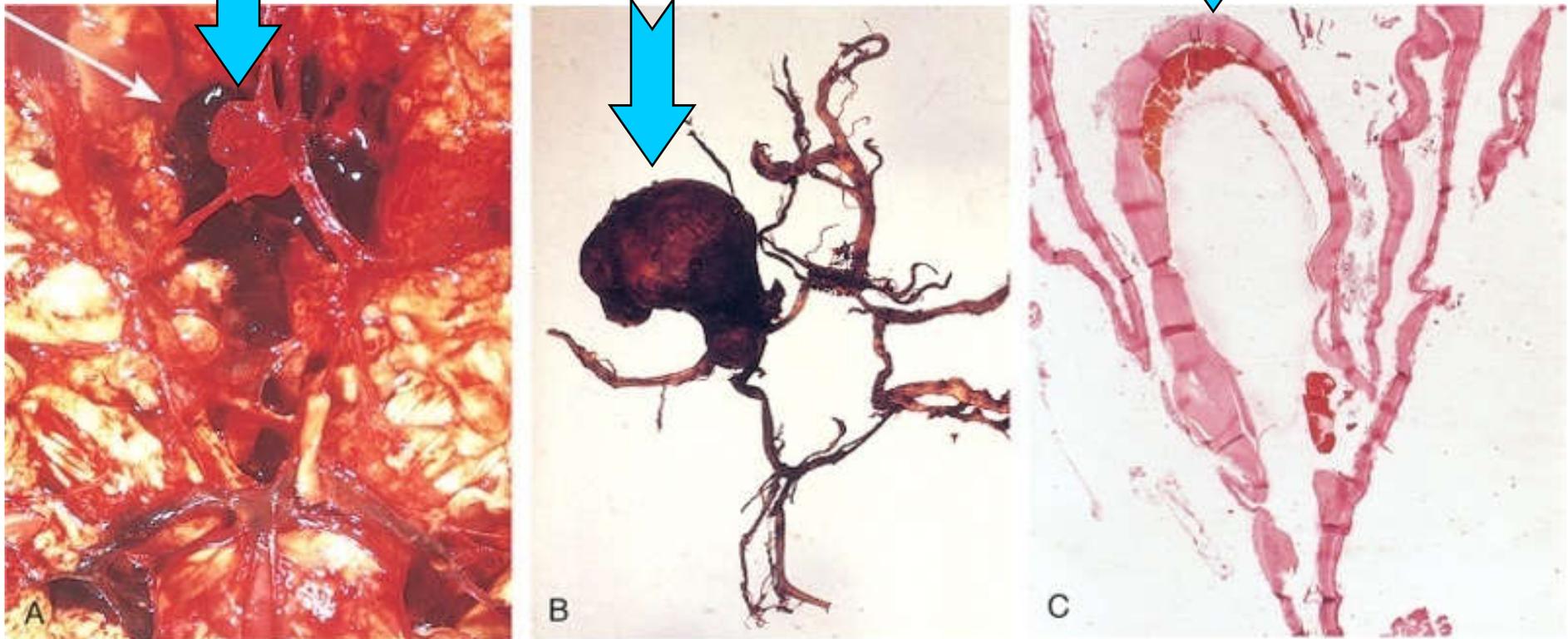
F23-9:
Relative
frequency
of common
**sites of
saccular
(Berry)
aneurysms**
in the circle
of Willis.

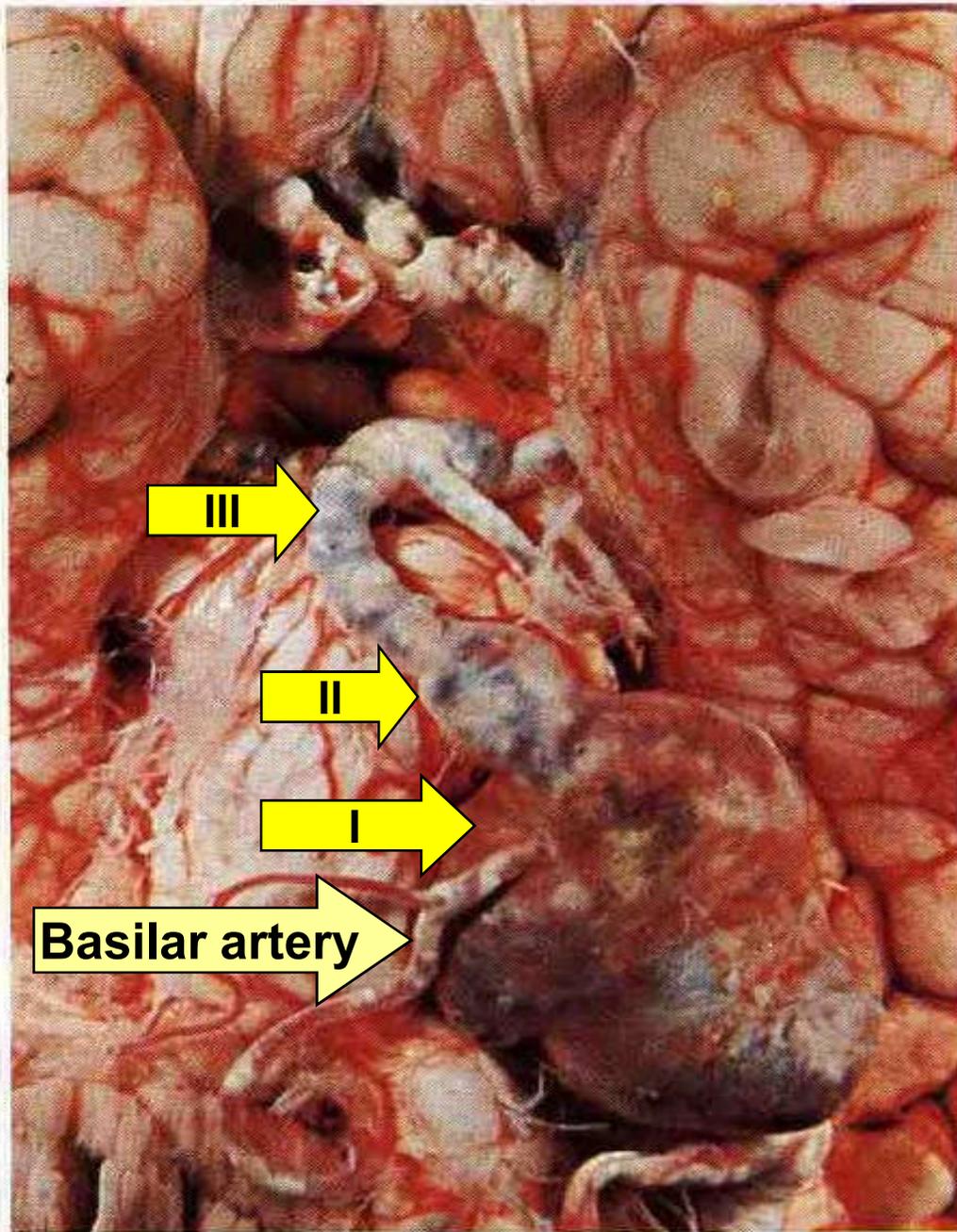
F23-10: **Berry saccular aneurysms.**

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).



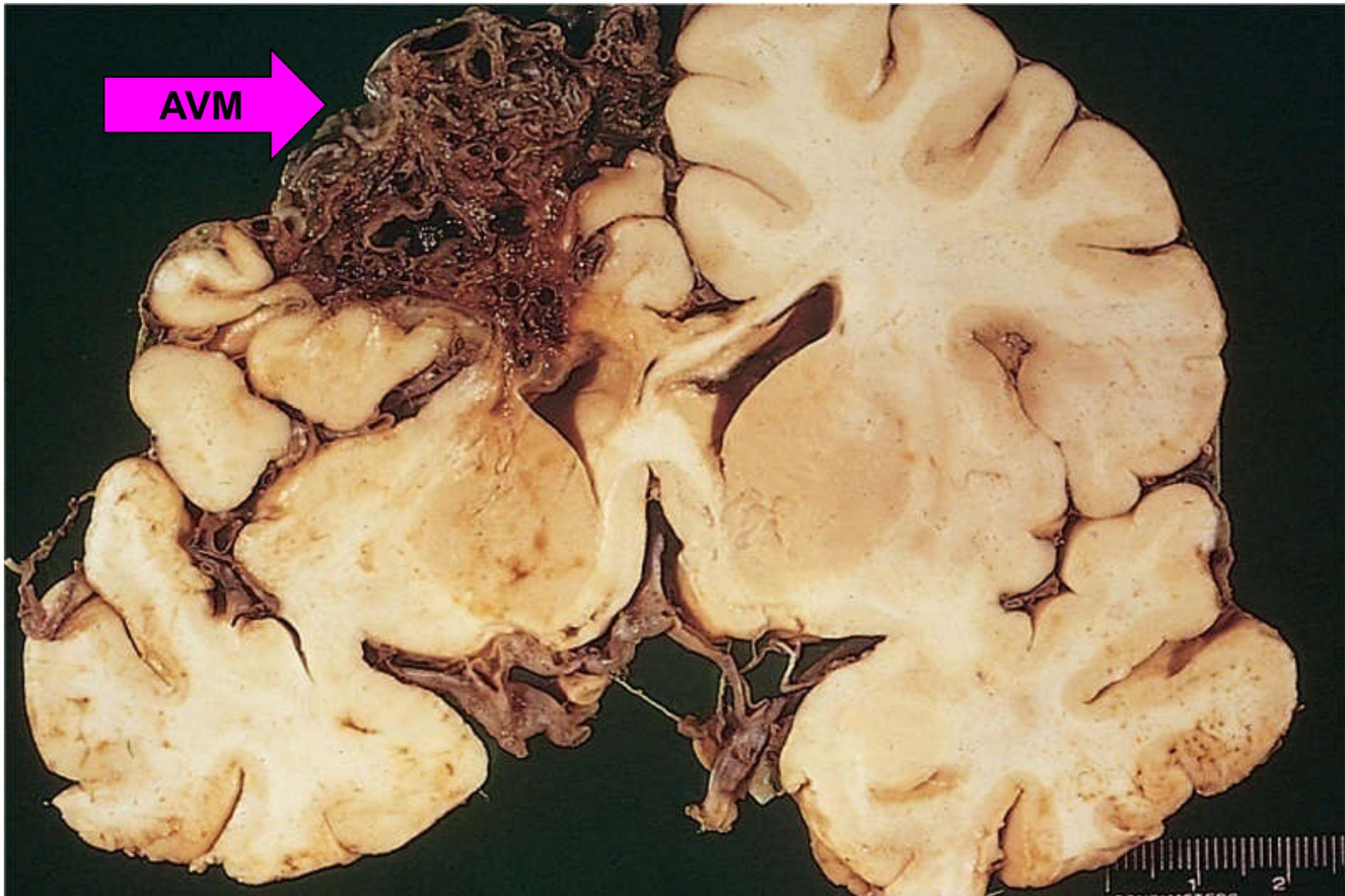


9.39 Aneurysm: basilar artery

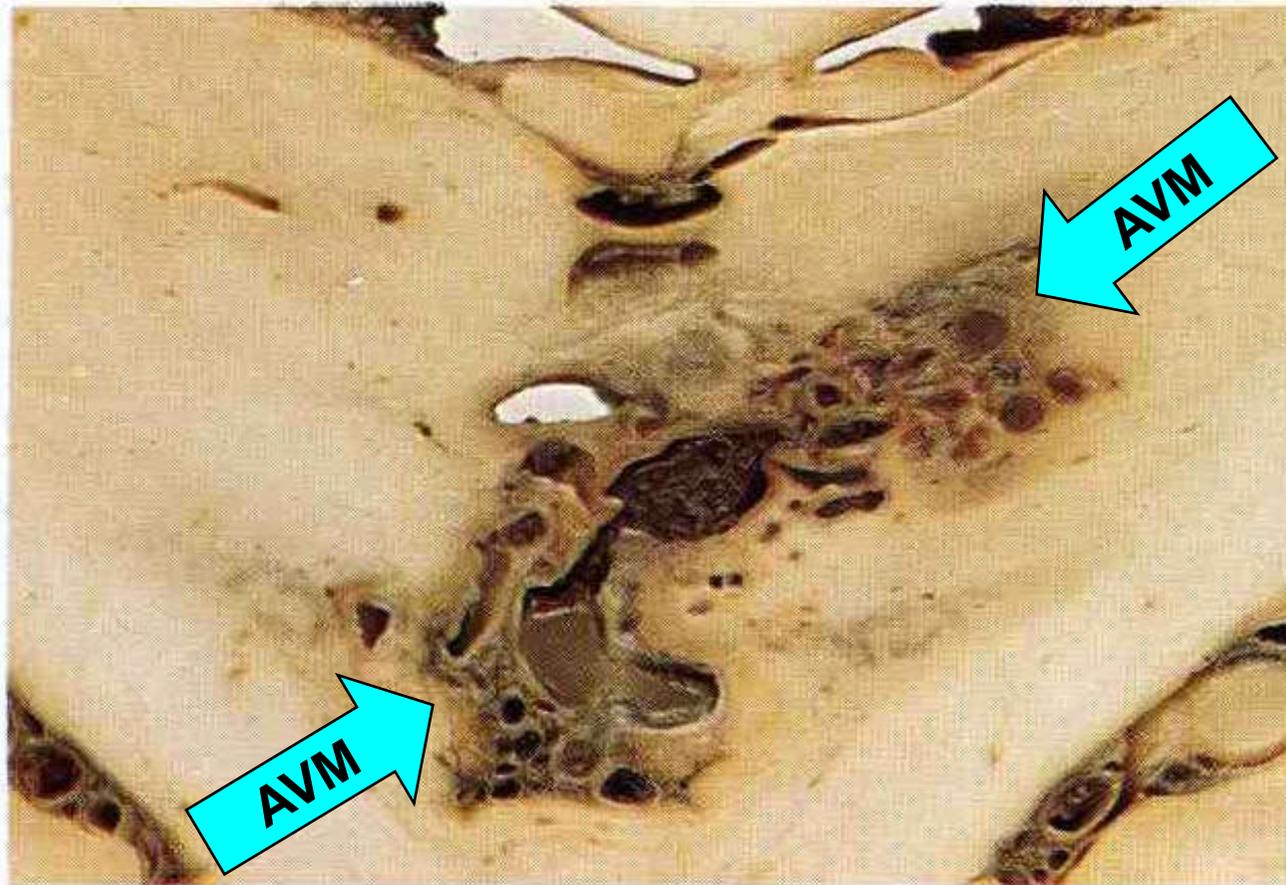
F 9.39:
Atherosclerotic
aneurysm of
basilar artery.

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.

F23-11: Arteriovenous malformation in subarachnoid space.

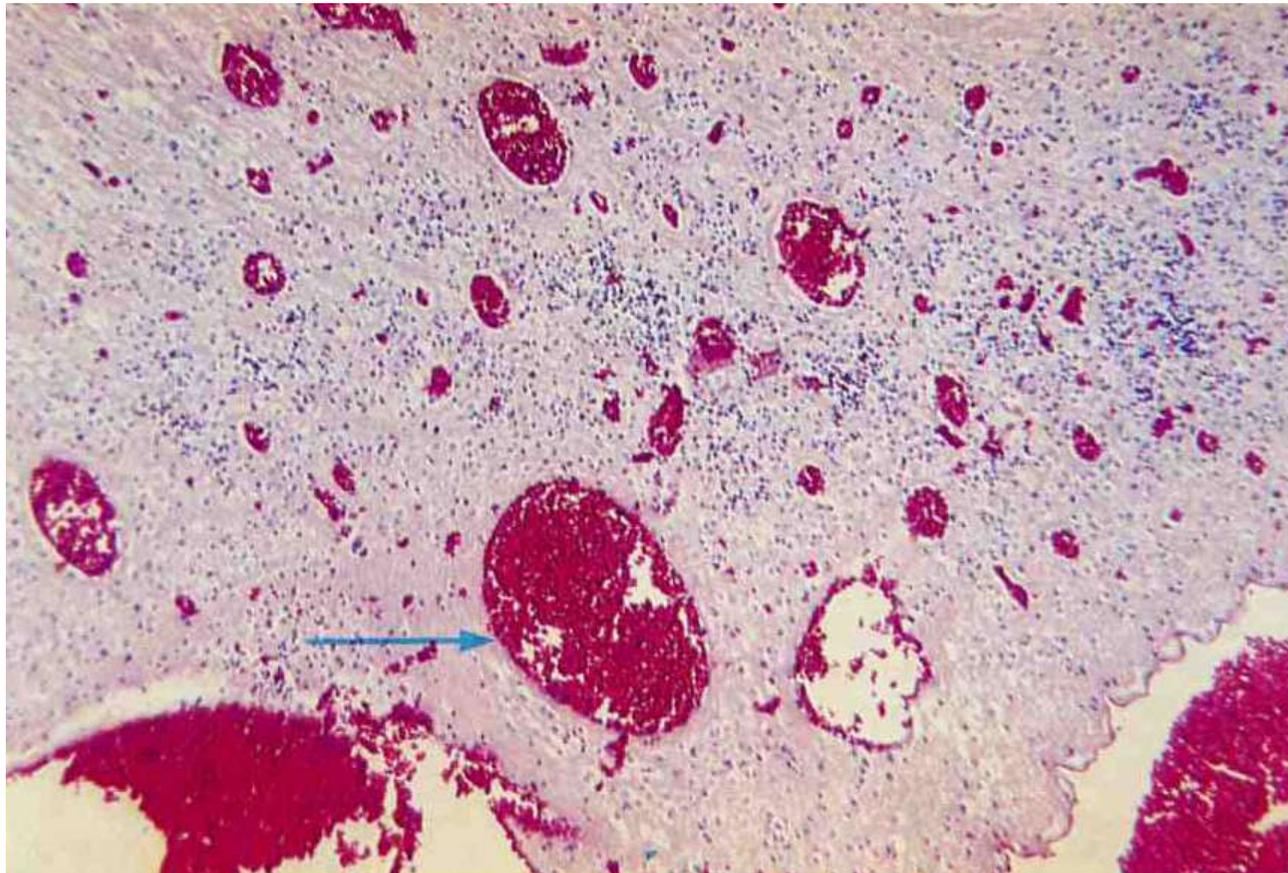


F 9-35: Arteriovenous malformation (hamartoma) : brain. A large complex intracerebral AVM is present within the thalamus & basal ganglia. The greyish-white vessels are thick-walled & many are thrombosed. The adjacent brain contains much brown hemosiderin pigment as a result of previous hemorrhages.



9.35 Arteriovenous hamartoma: brain

■ 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