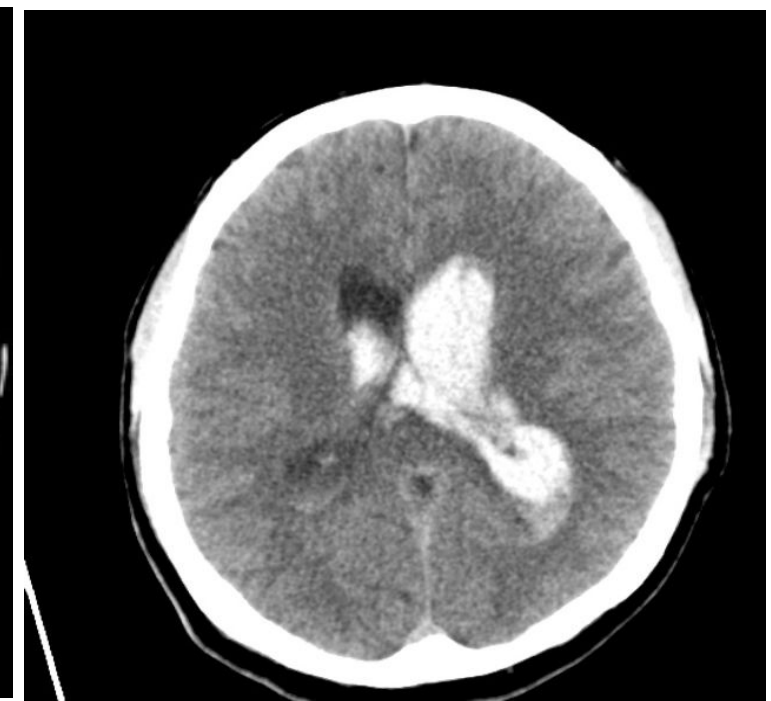
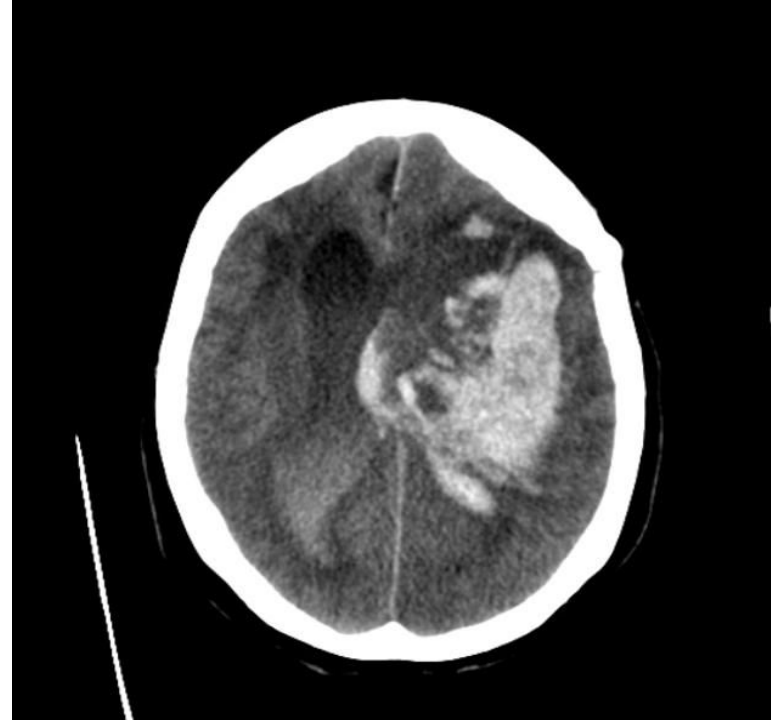


Intracranial hemorrhage

- This comprises:
 - 1- intracerebral and cerebellar hemorrhage
 - 2- subarachnoid hemorrhage
 - 3- subdural and extradural hemorrhage/hematoma.



intracerebral hemorrhage

- **Etiology**

- intracerebral hemorrhage causes approximately 10% of strokes.
- It is associated with higher mortality than ischemic stroke (up to 50%). A large hematoma may act as a space-occupying lesion, causing raised intracranial pressure with brain displacement and herniation.

Hypertensive

- Rupture of microaneurysms (Charcot-Bouchard aneurysms, 0.8-1.0 mm in diameter) and degeneration of small, deep, penetrating arteries are the principal pathologies.
- Such hemorrhage is usually massive, often fatal, and occurs in chronic hypertension and at well-defined sites: basal ganglia, pons, cerebellum, and subcortical white matter.
- Vasopressor drugs, such as cocaine, may cause hemorrhage.
- Alcohol is also a risk factor (probably as a result of impaired coagulation/platelet function).

.Cerebral amyloid angiopathy (CAA)

Deposition of amyloid-13 in the walls of small and medium-sized arteries in normotensive patients, particularly over 60 years, causes lobar intracerebral hemorrhage (especially posterior, i.e. occipital/parietal lobes), which is often recurrent.

CAA is associated with particular apolipoprotein E genotypes (E2) and is more common in patients with Alzheimer's disease. Cerebral microbleeds are usually seen on MRI sequences sensitive to hemosiderin deposition. CAA may occasionally cause TIA-like, transient neurological symptoms.

Secondary

- Arteriovenous malformations, cavernous, aneurysms, and dural venous thrombosis cause around 20% of intracerebral hemorrhages. Coagulopathies, anticoagulants, and thrombolysis may cause hemorrhage. Hemorrhagic transformation of a large ischemic infarct may sometimes present as a hemorrhage.

Subarachnoid hemorrhage

- Subarachnoid hemorrhage (SAH) means spontaneous arterial bleeding into the subarachnoid space and is usually clearly recognizable clinically from its dramatic onset.
- SAH accounts for some 5% of strokes and has an annual incidence of 6 per 100000.

Subarachnoid Hemorrhage

- “Worst headache of my life”
- Sudden onset symptoms
- Fever, nuchal rigidity common
- CT scan usually diagnostic
- Xanthochromia on spinal tap
- No focal deficits!

RED FLAGS

SNOOP :

Systemic signs / symptoms / disease (fever , myalgias , weight loss , history of malignancy ,or AIDS)

Neurologic signs or symptoms
(altered mentation , seizure , papilledema , focal neurologic findings)

Onset sudden (thunderclap headache)

Older age (new onset of headache after age 50)

Pattern change from previous headaches
(especially if rapidly progressive in severity or frequency)



Box 21.37 Underlying causes of subarachnoid haemorrhage

	(%)
Saccular (berry) aneurysms	70%
Arteriovenous malformation (AVM)	10%
No arterial lesion found	15%
Rare associations	<5%
Bleeding disorders	
Mycotic aneurysms – endocarditis	
Acute bacterial meningitis	
Tumours, e.g. metastatic melanoma, oligodendroglioma	
Arteritis (e.g. systemic lupus erythematosus)	
Spinal AVM → spinal SAH	
Coarctation of the aorta	
Marfan, Ehlers–Danlos syndrome	
Polycystic kidneys	

Saccular (berry) aneurysms

- Saccular aneurysms develop within the circle of Willis and adjacent arteries.
- Common sites are at arterial junctions:
 - 1- between posterior communicating and internal carotid artery - posterior communicating artery aneurysm
 - 2- between anterior communicating and anterior cerebral artery - anterior communicating and anterior cerebral artery aneurysm
 - 3- at the trifurcation or a bifurcation of the middle cerebral artery- middle cerebral artery aneurysm.

Digital subtraction angiogram: posterior communicating artery aneurysm.



- Aneurysms cause symptoms either by spontaneous rupture, when there is usually no preceding history, or by direct pressure on surrounding structures; for example, an enlarging unruptured posterior communicating artery aneurysm is the most common cause of a painful 3rd nerve palsy

AComm Aneurysm

- Headache •
- Visual field defects •



Bitemporal Hemianopsia
Optic Chiasm Compression
Pituitary Tumor/Aneurysm

Berry Aneurysms Associations

- ADPKD •
- Ehlers-Danlos •
- Aortic coarctation •
- Older age •
- Hypertension •
- Smoking •

PComm Aneurysm

- Unilateral headache, eye pain
- CN III palsy
 - Eye: "down and out"
 - Ptosis
- Pupil dilation – nonreactive to light

Pupil Sparing

- Is pupil normal (not dilated)?
- If yes, pupil is spared → lesion not aneurysm
- Pupillary constrictors easily compressed in subarachnoid space
- If pupil is “spared”
- Palsy often associated with DM
- Ischemic neuropathy of CN III (small vessel disease)
- Sometimes painful
- Spontaneously resolves
- “Rule of the pupil”

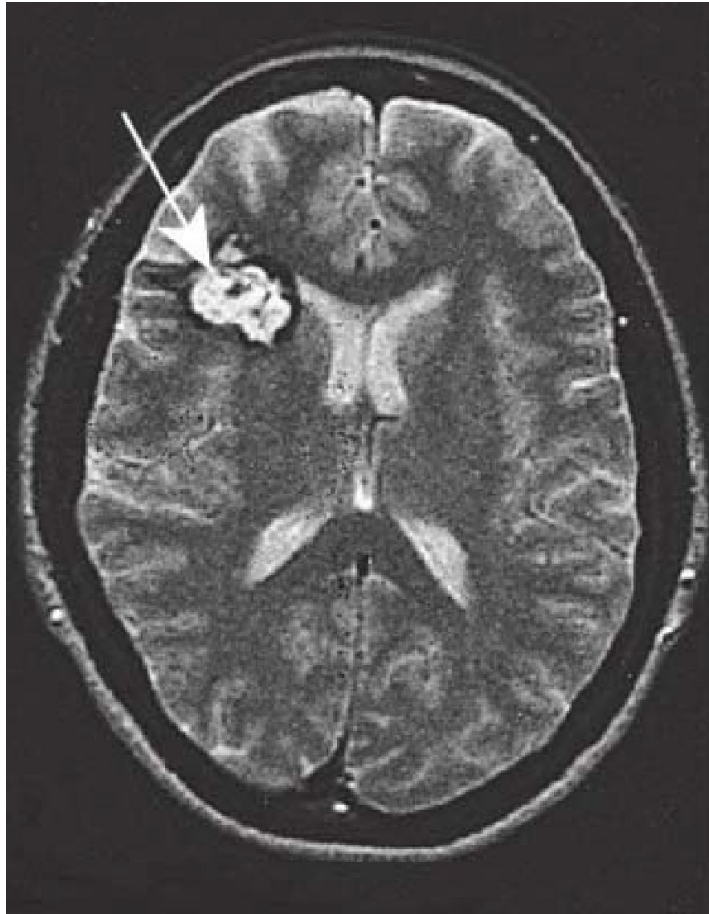
Charcot-Bouchard Aneurysms

- Micro-aneurysms
- Small branches lenticulo-striate arteries
- Basal ganglia, thalamus
- Possible origin of hypertensive ICH

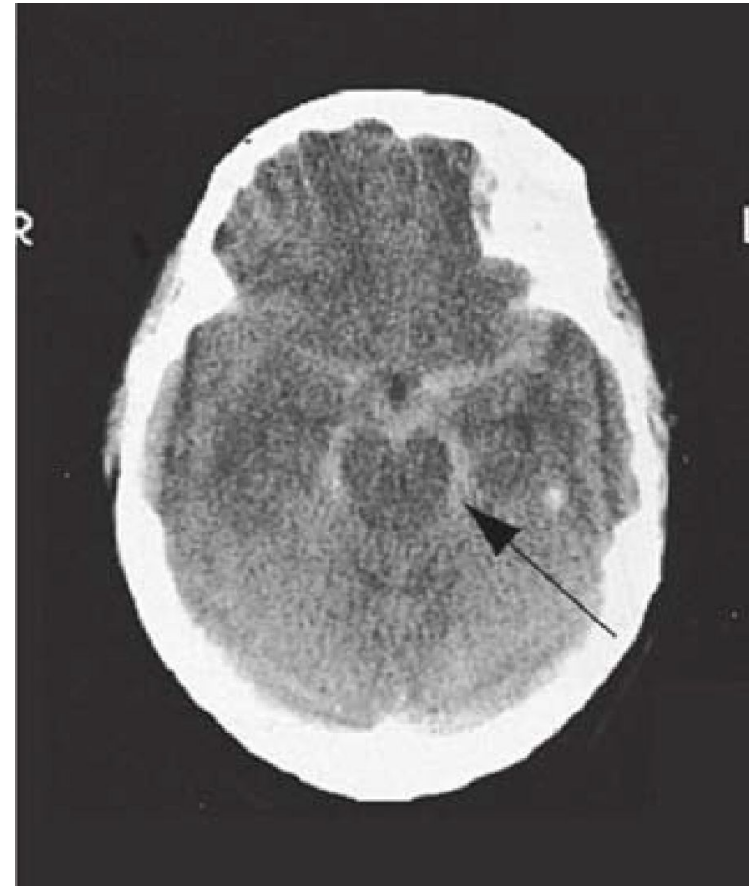
Arteriovenous malformation

- Arteriovenous malformation (AVM) is a vascular developmental malformation, often with a fistula between arterial and venous systems, causing high flow through the AVM and high-pressure arterialization of draining veins. An AVM usually presents following a spontaneous intracerebral hemorrhage or with a seizure, often focal in onset.
- The risk of the first hemorrhage in unruptured AVMs (20% fatal and 30% resulting in permanent disability) is approximately 2-3% per year. Once an AVM has caused a hemorrhage, the risk of rebleeds is increased to approximately 10% per year.

- Cavernous hemangiomas (cavernomas) are common (0.1- 0.5% prevalence) and consist of a tangle of low-pressure dilated vessels without a major feeding artery; they are frequently symptomless and seen incidentally on imaging.
- Multiple cavernomas often have a genetic basis. Cavernomas may cause seizures. Small hemorrhages may occur but are usually low-pressure bleeds and rarely cause severe deficits. Surgical resection is rarely needed, except where the cavernoma is gradually enlarging or causing significant neurological symptoms.



Magnetic resonance T2-weighted image:
symptomless frontal cavernoma
(arrowed).



Subarachnoid hemorrhage CT
showing blood around the brainstem
(arrowed).

Clinical features of subarachnoid hemorrhage

- There is a sudden, very severe headache, often occipital (mean time to peak headache 3 min). Headache is usually followed by vomiting and often by coma and death. Survivors of SAH may remain comatose or drowsy for hours, days or longer. SAH is a *possible* diagnosis in any sudden headache.
- Following major SAH, there is neck stiffness and a positive Kernig's sign.
- Papilledema is sometimes present, with retinal and/ or sub-hyaloid hemorrhage (tracking beneath the retinal hyaloid membrane). Minor bleeds cause few signs but almost invariably do cause headaches (approximately 17% of patients have small 'sentinel bleeds' in the weeks before presenting with SAH).

Subarachnoid Hemorrhage

Bleeding into space b/w •
arachnoid & pia mater



Subarachnoid Hemorrhage

- “Worst headache of my life”
- Sudden onset symptoms
- Fever, nuchal rigidity common
- CT scan usually diagnostic
- **Xanthochromia** on spinal tap
- No focal deficits!

Subarachnoid Hemorrhage

Usually from ruptured berry aneurysms •

Most common site: anterior circle of Willis •

Branch points of AComm artery •

AVMs •

Other associations: •

Marfan syndrome •

ADPKD •

Ehlers-Danlos •

Hemorrhagic Stroke

Intraparenchymal Bleed

- Often small arteries or arterioles
- HTN
- Anti-coagulation
- CNS malignancy
- Ischemic stroke followed by reperfusion



Image courtesy of OpenStax College

Sites of Bleed

Intraparenchymal Bleed

- Putamen (35%) •
- Subcortex (30%) •
- Cerebellum (16%) •
- Thalamus (15%) •
- Pons (5-12%) •

Hemorrhage Stroke

Intraparenchymal Bleed

- Putamen stroke •
- Contralateral hemiparesis (IC) •
- Hemisensory loss (thalamus) •
- Gaze deviation toward side of bleed (FEF) •
- Watch for: •
 - Left paralysis, sensory loss •
 - Eyes deviated to right •



Cerebral Amyloid Angiopathy

- Recurrent hemorrhagic strokes •
- Beta-amyloid deposits in artery walls •
 - Weak, prone to rupture •
- Typically lobar hemorrhages •
 - Frontal, parietal, occipital •
 - Usually smaller strokes •
- Contrast with HTN: Basal ganglia •
 - Watch for: •
 - Elderly person •
 - Recurrent hemorrhagic strokes •

Intraventricular Hemorrhage

- Complication of premature birth
- Hemorrhage into lateral ventricle
 - Usually first 5 days of life
 - Sometimes asymptomatic
- LOC, hypotonia, loss of spontaneous movements
- Massive bleeds can cause seizures, coma



Intraventricular Hemorrhage

- Clot can obstruct the Foramen of Monro

- Enlargement of lateral ventricles

- Normal 3rd/4th ventricle

- Treatment: Ventriculoperitoneal (VP)

- Germinal matrix problem

- Highly vascular area near ventricles

- Premature infants: poor autoregulation of blood flow here

- In full term infants, this area has decreased vascularity

Subdural and extradural bleeding

- **Subdural hematoma**

- Subdural hematoma (SDH) means the accumulation of blood in the subdural space following the rupture of a vein. This usually follows a head injury, sometimes a trivial one. The interval between injury and symptoms can be days or may extend to weeks or months. Chronic, apparently spontaneous, SDH is common in the elderly, and also occurs with anticoagulants.
- Headache, drowsiness, and confusion are common; symptoms are indolent and can fluctuate. Focal deficits, such as hemiparesis or sensory loss, develop. Epilepsy occasionally occurs. Stupor, coma, and coning may follow.

Subdural Hematoma

- Usually traumatic
- Rupture bridging veins
- Blood b/w dura and arachnoid space
- SLOW bleeding due to low pressure veins

Subdural Hematoma

- Crescent shaped bleed
- Crosses suture lines
- Limited by dural reflections
 - falx cerebri
 - tentorium
 - falx cerebelli



Subdural Hematoma

Risk factors •

- Old age •

- Alcoholics •

- Blood thinners •

- Brain atrophy increases space veins must cross •

- More vulnerable to rupture •

- Classic history is confusion weeks after head injury •

- Classic injury in shaken baby syndrome •

Extradural hemorrhage

- Extradural hemorrhage (EDH) typically follows a linear skull vault fracture tearing a branch of the middle meningeal artery. Extradural blood accumulates rapidly over minutes or hours. A characteristic picture is that of a head injury with a brief duration of unconsciousness, followed by improvement (the lucid interval).
- The patient then becomes stuporous; there is an ipsilateral dilated pupil and contralateral hemiparesis, with rapid trans tentorial coning.
- Bilateral fixed, dilated pupils, tetraplegia and respiratory arrest follow. An acute progressive SDH presents similarly.



Figure 21.43 Bilateral subdural haematomas. A. CT scan.
B. Magnetic resonance T1-weighted image.

Raised Intracranial Pressure

ICP

- Mass lesions (tumors)
- Cerebral edema (large stroke, severe trauma)
- Hydrocephalus
- Obstruction of venous outflow (thrombosis)
- Idiopathic intracranial hypertension
- Pseudotumor cerebri

Increased Intracranial Pressure

General symptoms

- Headache (pain fibers CN V in dura) •
- Depressed consciousness •
- Pressure on midbrain reticular formation •
- Vomiting •

Papilledema

- Optic disc swelling
- Due to $\uparrow$ ICP
- i.e. mass effect
- Also seen in severe HTN
- Usually bilateral
- Blurred margins optic disc on fundoscopy



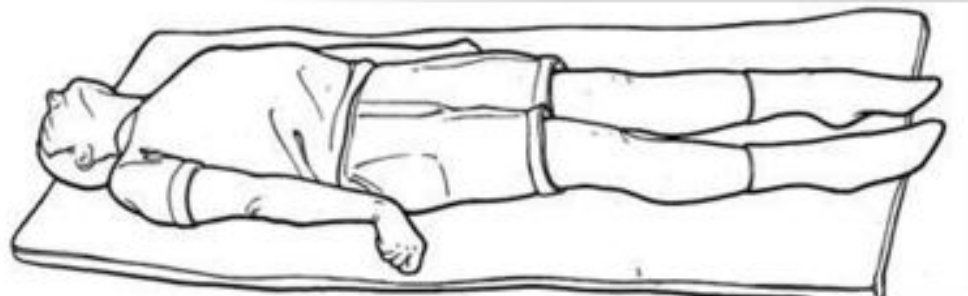
Cushing's Triad

- Hypertension •
- Bradycardia •
- Irregular respiration •

Posturing



Decorticate (arms flexed)
Cerebral Hemisphere Damage



Decerebrate (arms extended)
Brainstem Damage



Glasgow Coma Scale

Three tests: eye, verbal and motor •

GCS score: 3 to 15 •

Eye (1-4 points) •

Does not open, opens to painful stimuli, opens to voice, opens spontaneously •

Verbal (1-5 points) •

No sound, incomprehensible sounds, inappropriate words, confused, oriented •

Motor (1-6 points) •

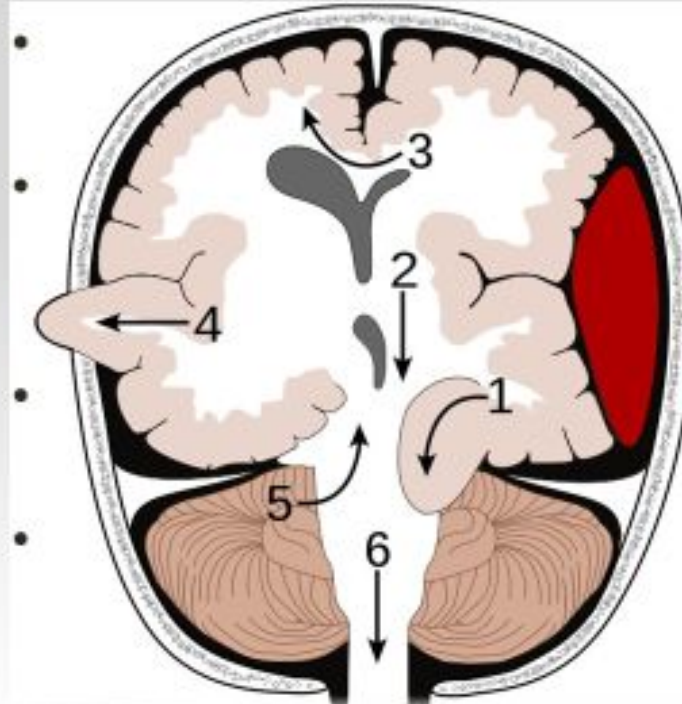
No movements, decerebrate posturing, decorticate posturing, withdrawal to pain, localizes to pain, obeys commands •

Herniation

- Expanding volume: blood, tumor •
- Forces brain through weakest points •

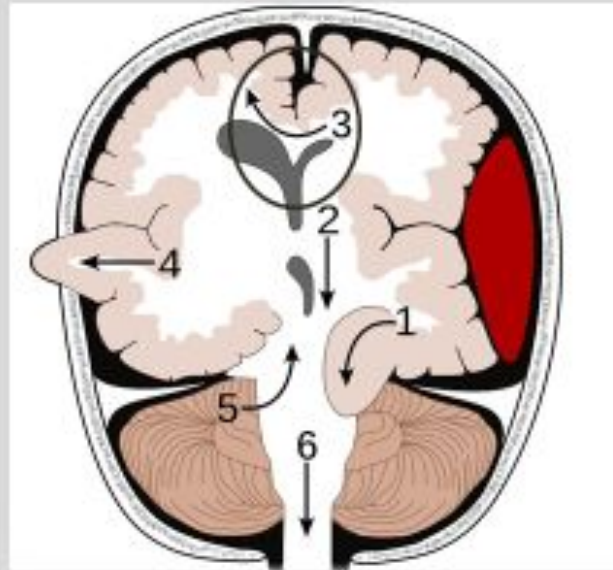
Where can displaced brain go?

- Subfalcine
- Side to side
- Uncal
- Side to bottom
- Transtentorial
- Central
- Diencephalon → midbrain
- Tonsillar
- Cerebellum thru the "hole"



Subfalcine Herniation

- Cingulate gyrus
- Extends under falx
- Drags ipsilateral ACA with it
- ACA compression
- Contralateral leg paresis



Uncal herniation

- Uncus = medial temporal lobe
- Across tentorium
- Midbrain compression



Uncal herniation



Ipsilateral CNIII compression
Loss of parasympathetic innervation

Dilated (“blown”) pupil

Lack of pupillary constriction to light

Collapses ipsilateral posterior cerebral artery

Visual loss – cortical blindness

Homonymous hemianopsia

Cerebral peduncle compression

Can be on side of lesion (contralateral paresis)

Can also be on opposite side (ipsilateral paresis)

Kernohan's notch

Duret hemorrhage of pons and midbrain

Perforating branches basilar artery draining veins

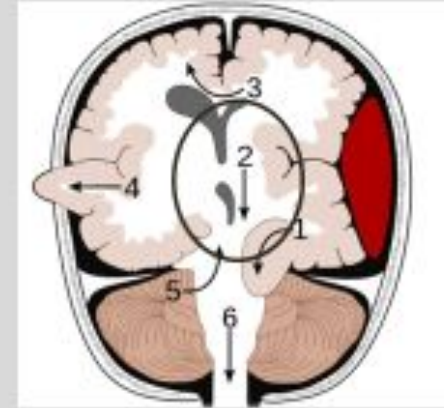
Uncal herniation

- Dilated pupil (side of lesion)
- Visual loss
- Hemiparesis or quadriparesis



Transtentorial Herniation

- Thalamus, hypothalamus, and medial parts of both temporal lobes forced through tentorium cerebelli
- Somnolence, LOC
- Initially: small, reactive pupils
- Later: nonreactive
- Posturing



Tonsillar Herniation

- Cerebellar tonsils herniate downward through the foramen magnum
- Most commonly caused by a posterior fossa mass lesion
- Compression of medulla results in depression centers for respiration and cardiac rhythm control
- Cardiorespiratory failure



