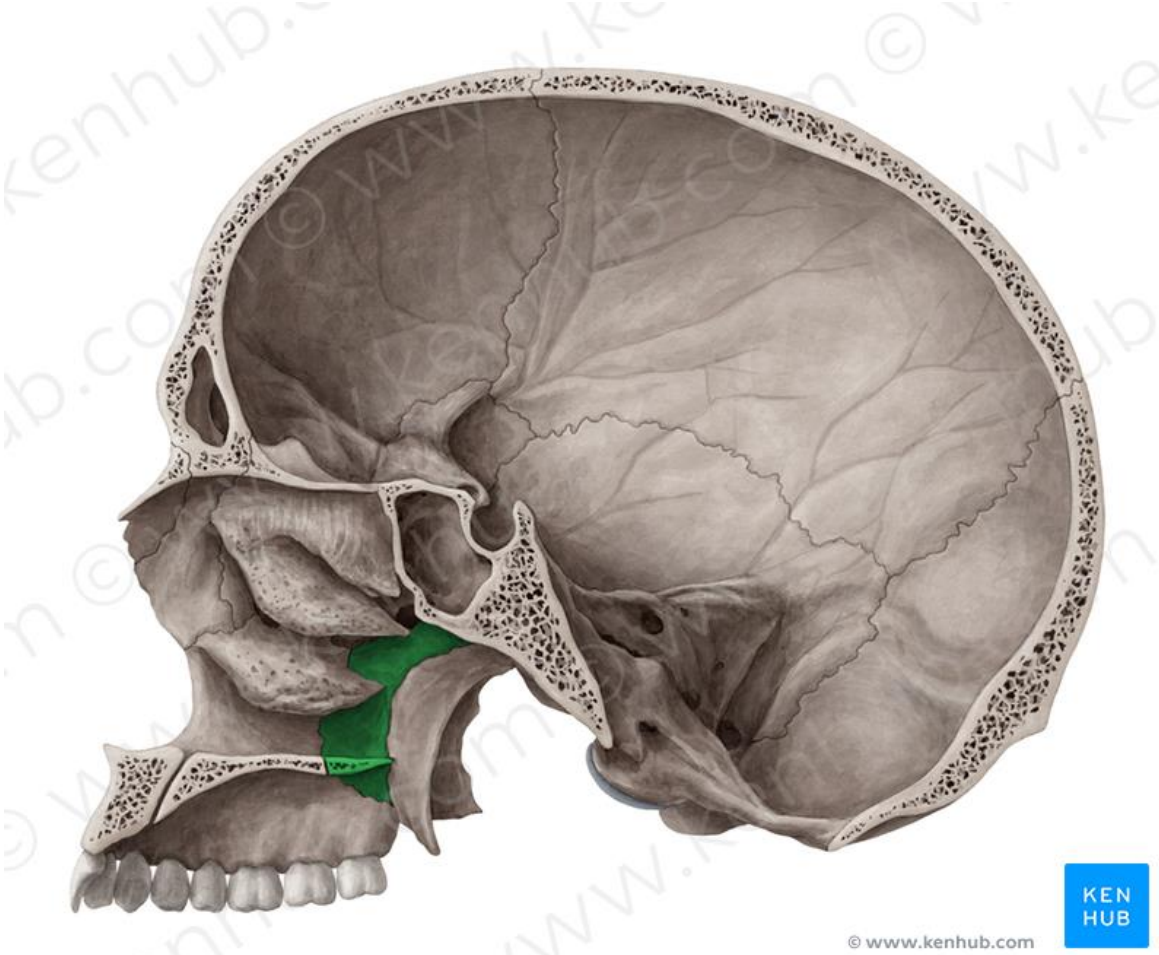
INTRACRANIAL PRUSSURE

MOHAMMAD ALDIBS

ICP

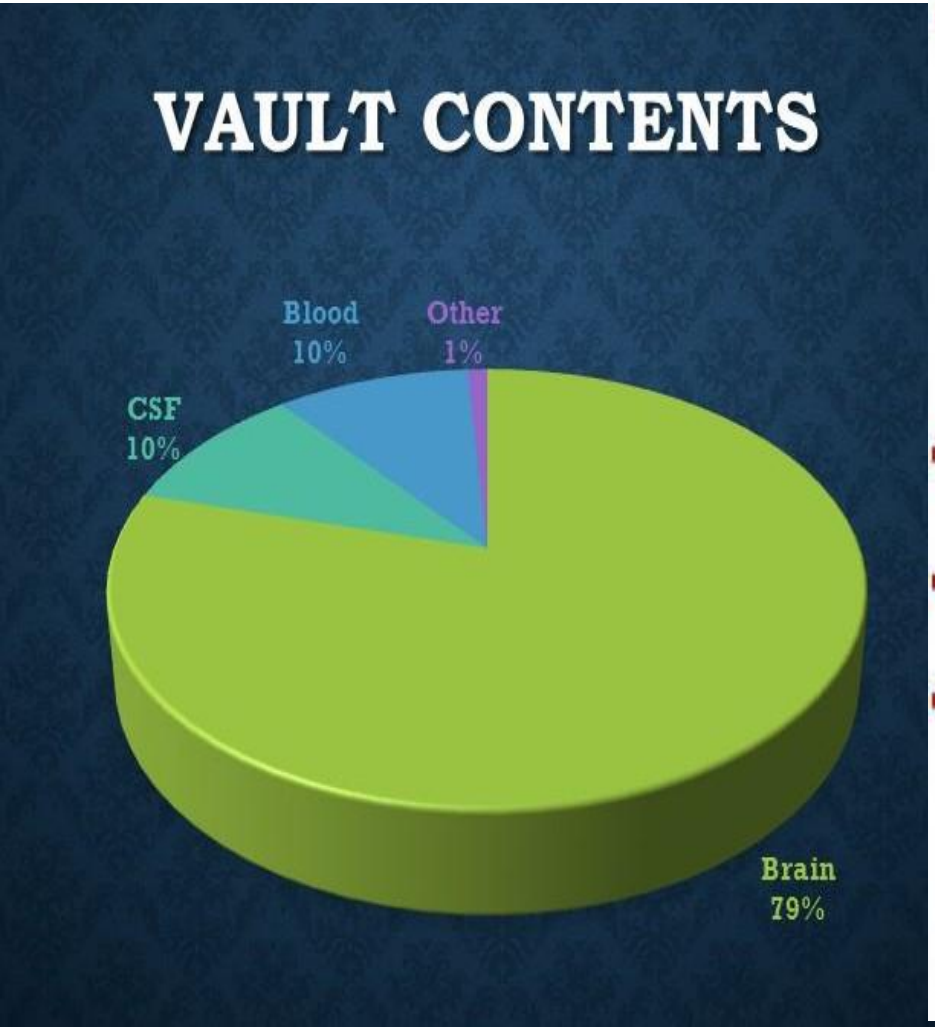
- The pressure within the cranial cavity

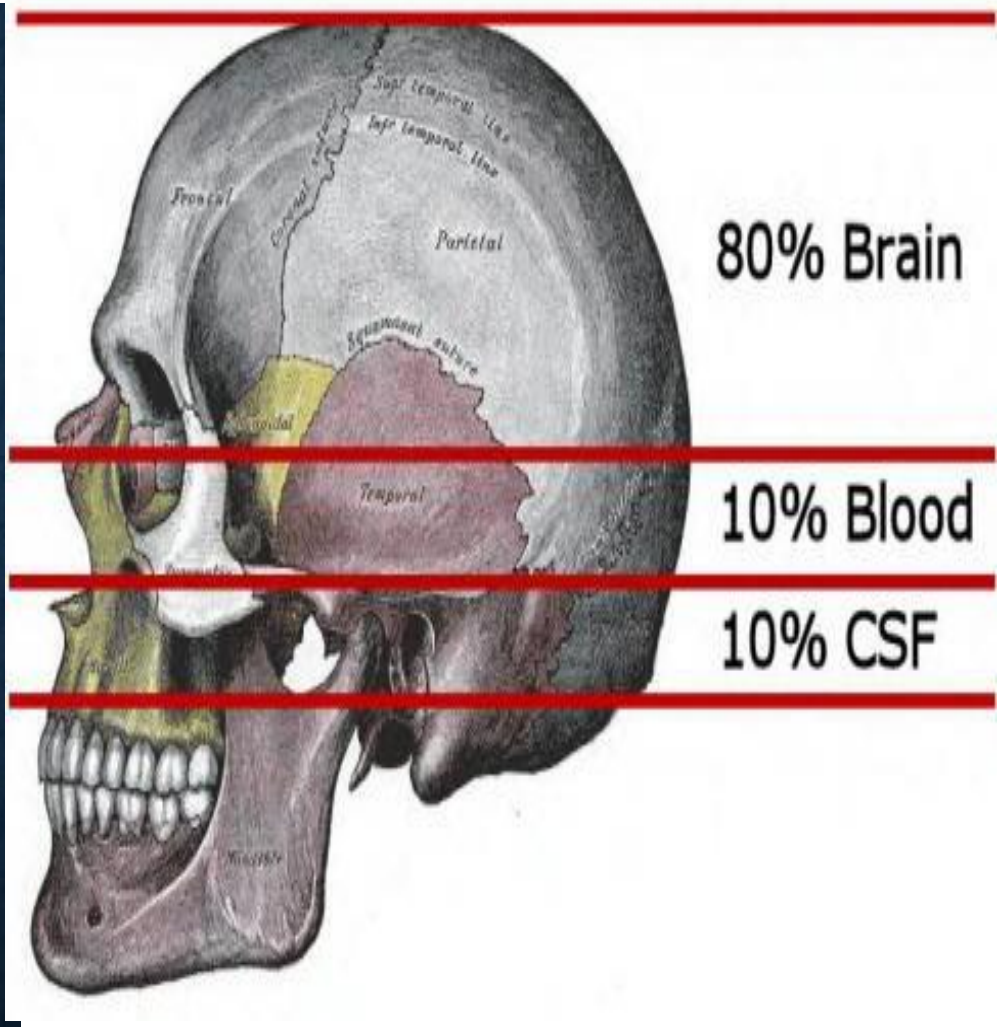


NORMAL ICP VALUES

Age group	mmHg	cmH2O (=mmHg*1.36)
Adults	10-15	8-18
Children	3-7	4-9.5
Infants	1.5-6	2-8

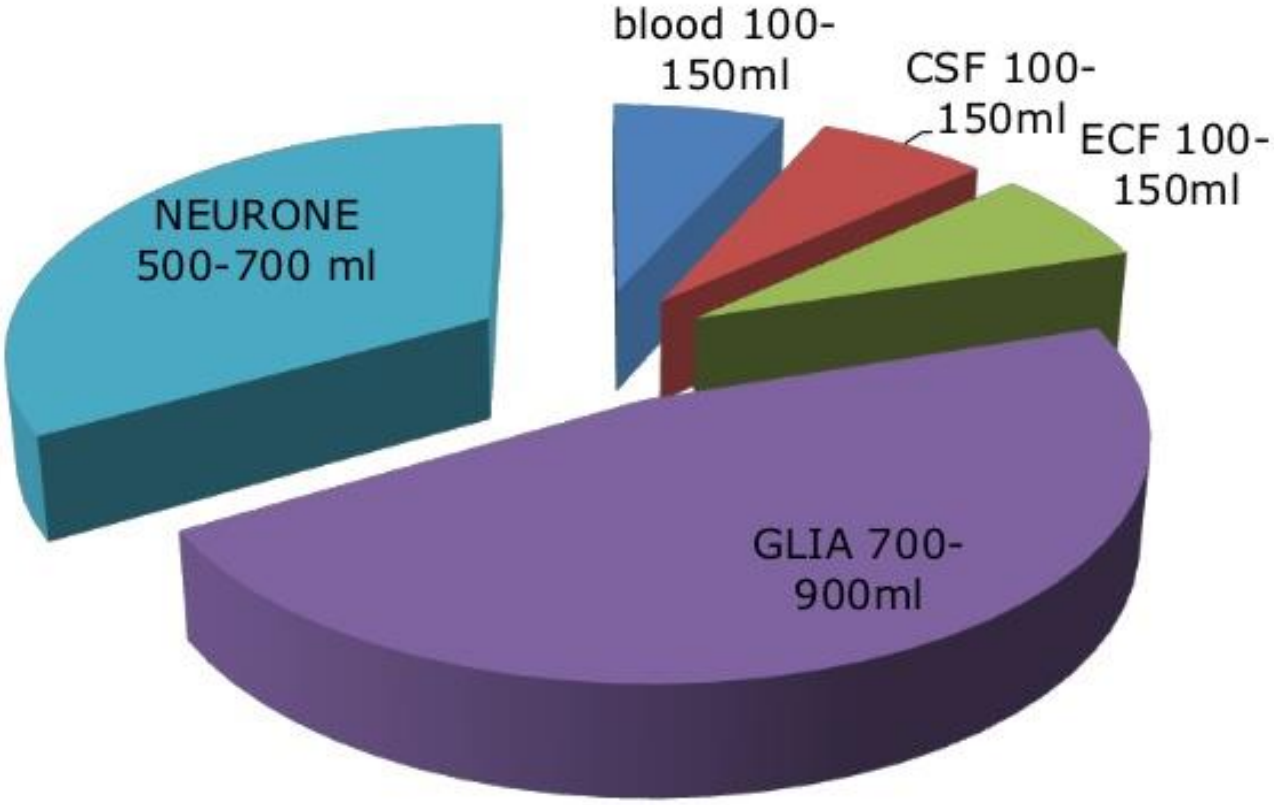
INTRACRANIAL CONTENTS





Volume of intracranial contents.

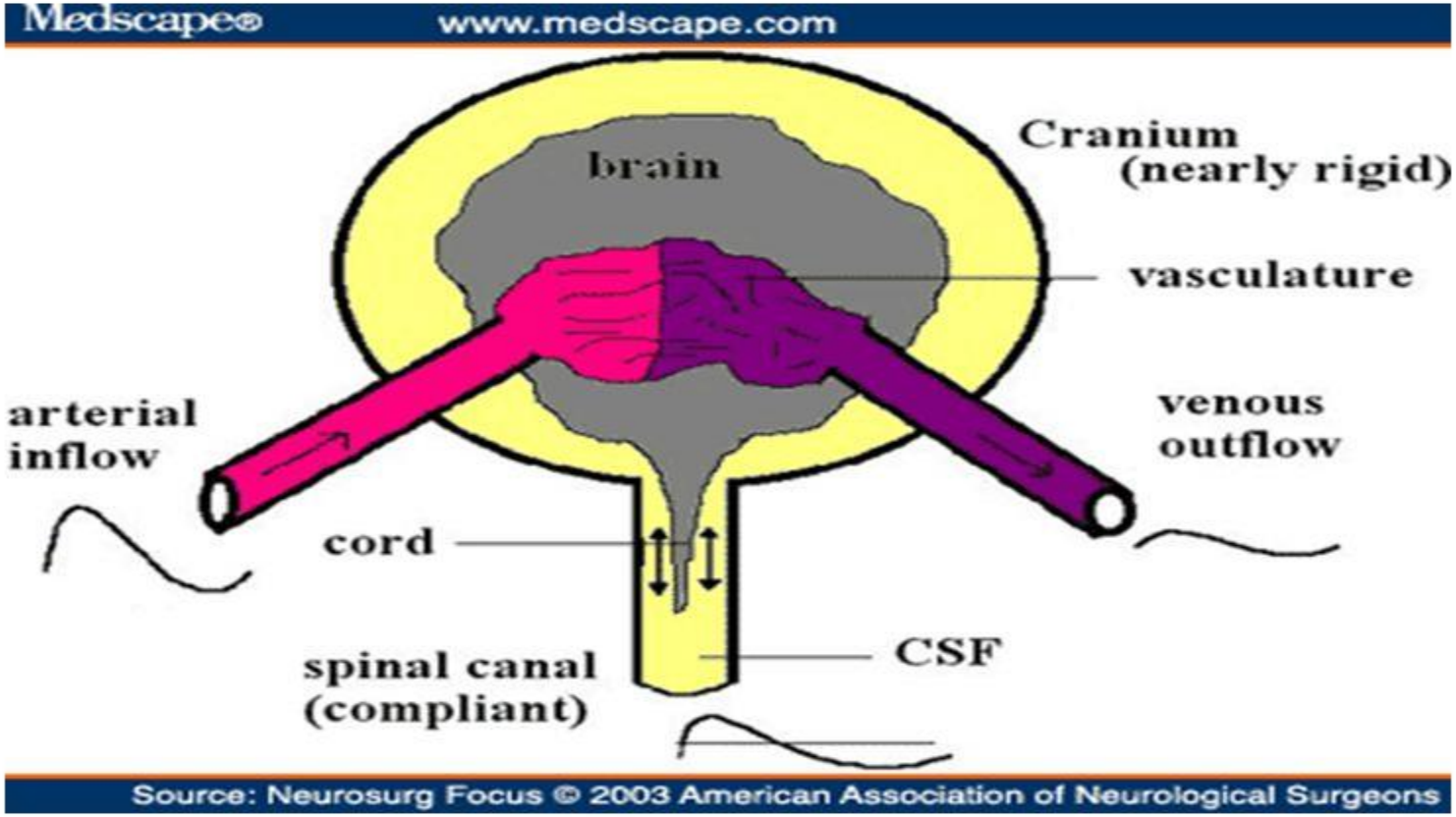
essential neurosurgery textbook



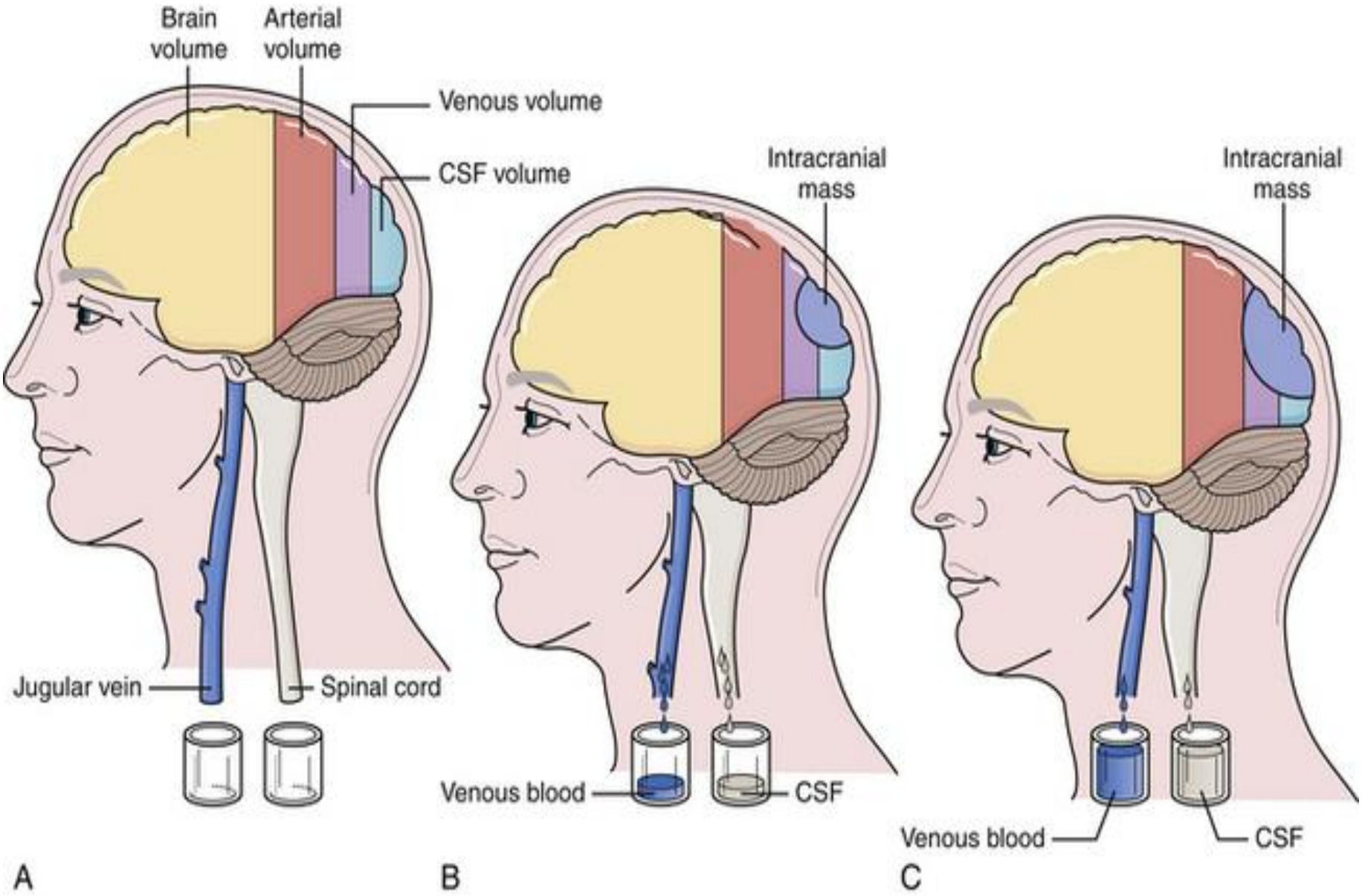
MONRO-KELLIE DOCTRINE

- The cranial cavity is a rigid sphere filled to capacity with non-compressible contents
- An increase in the volume of any one of the intracranial contents must be offset by a decrease in one or more of the others or be associated with a rise in ICP

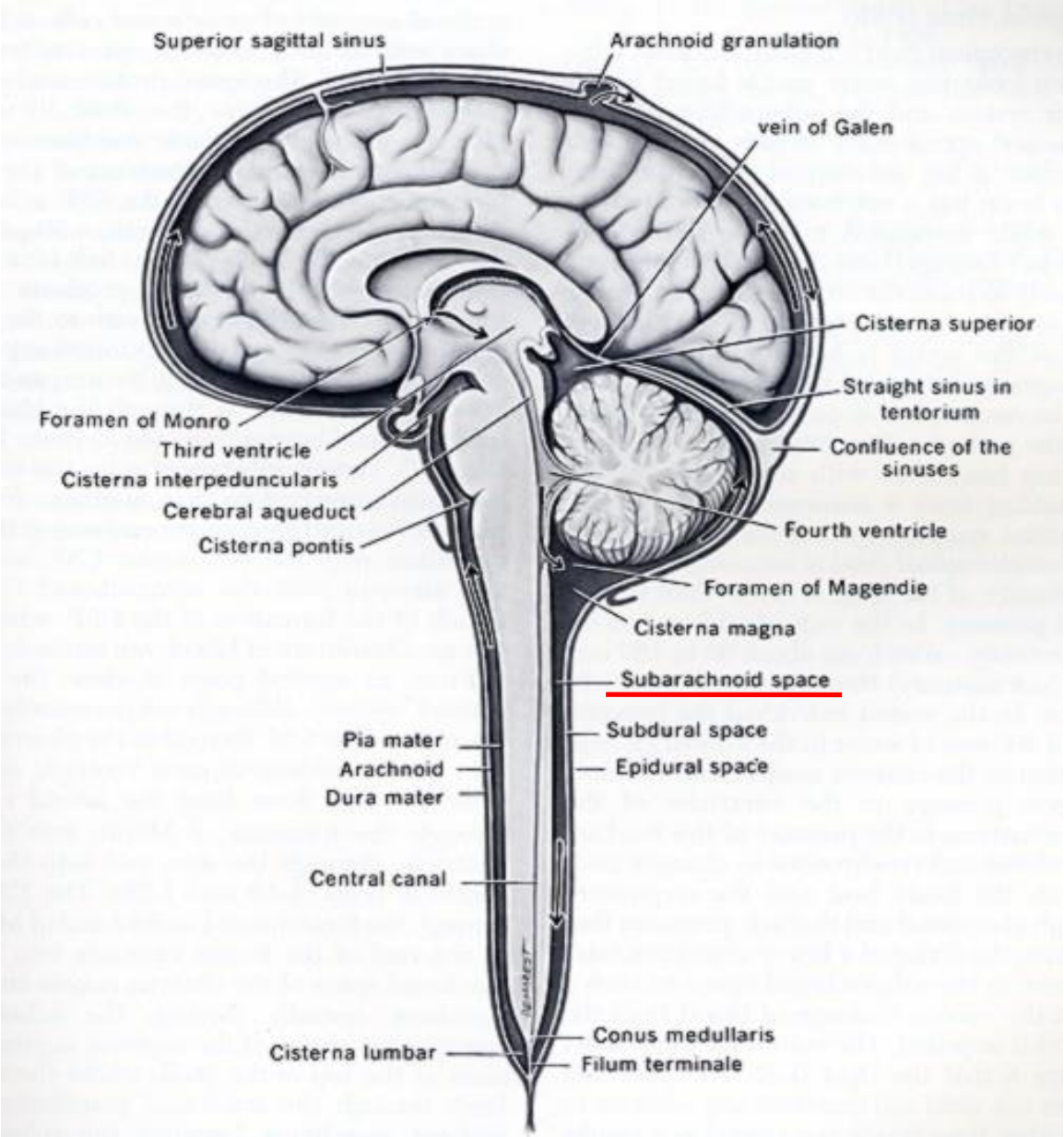
Monro-Kellie Doctrine



SPATIAL COMPENSATION



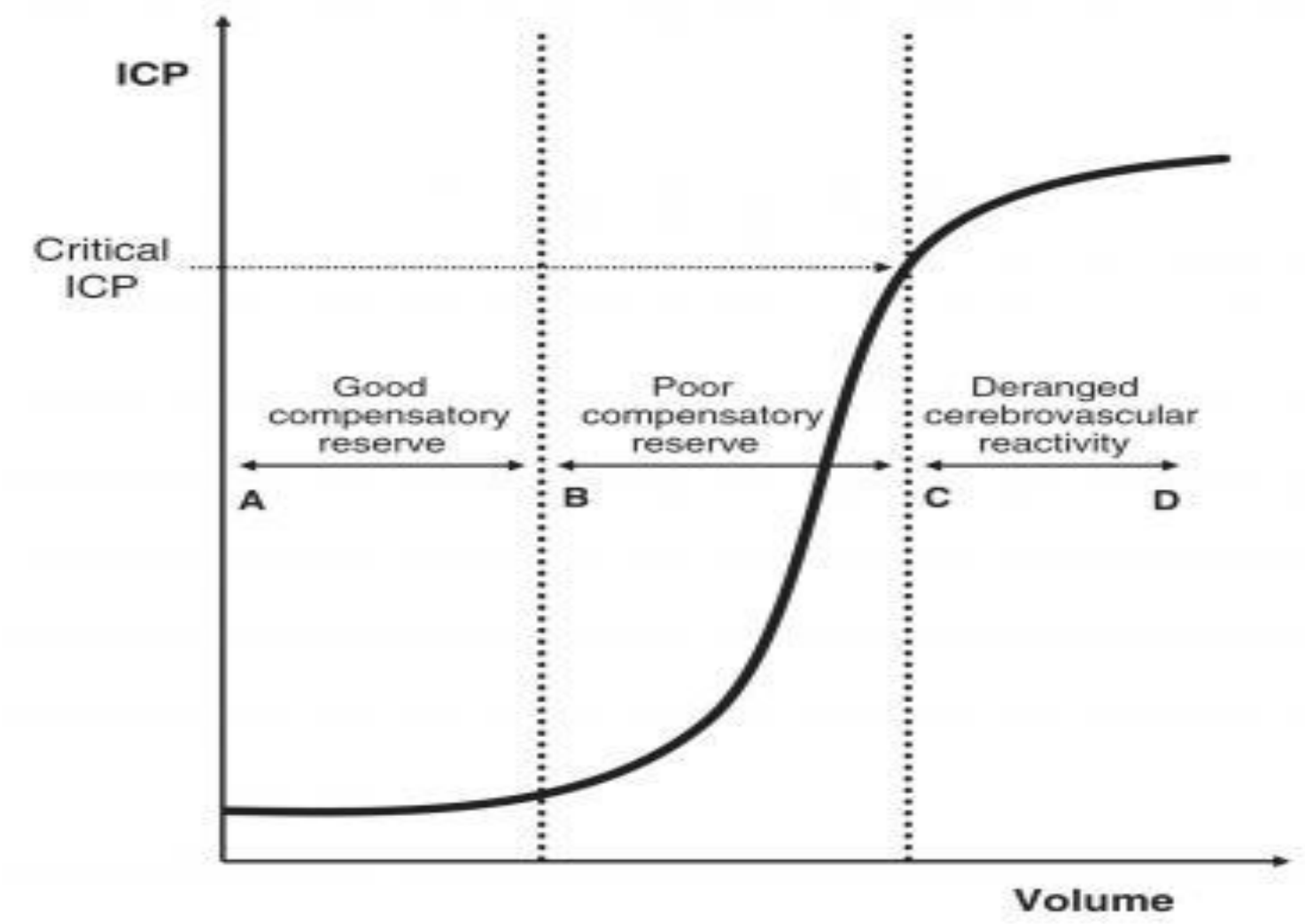
SPINAL SUBARACHNOID SPACE

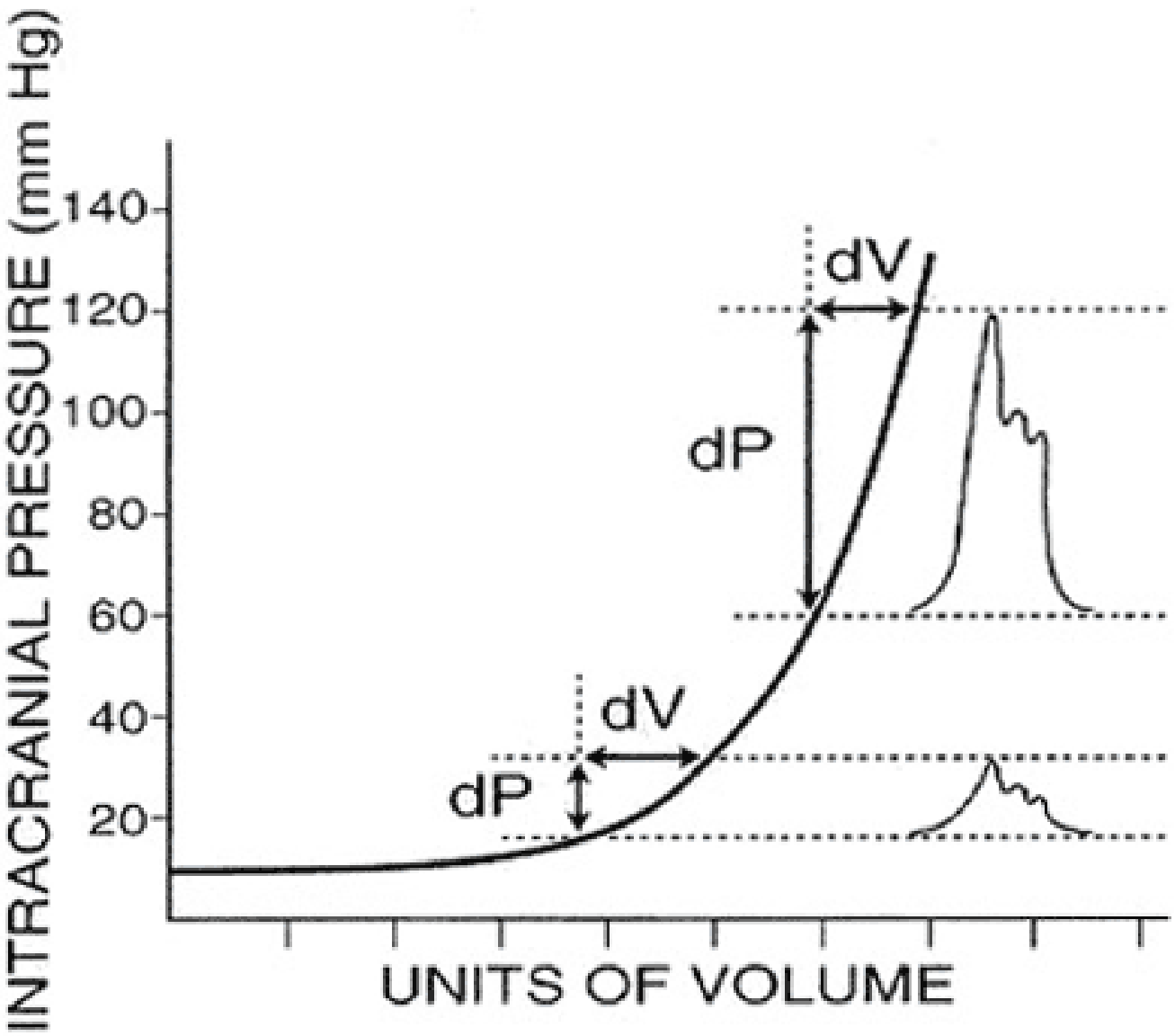


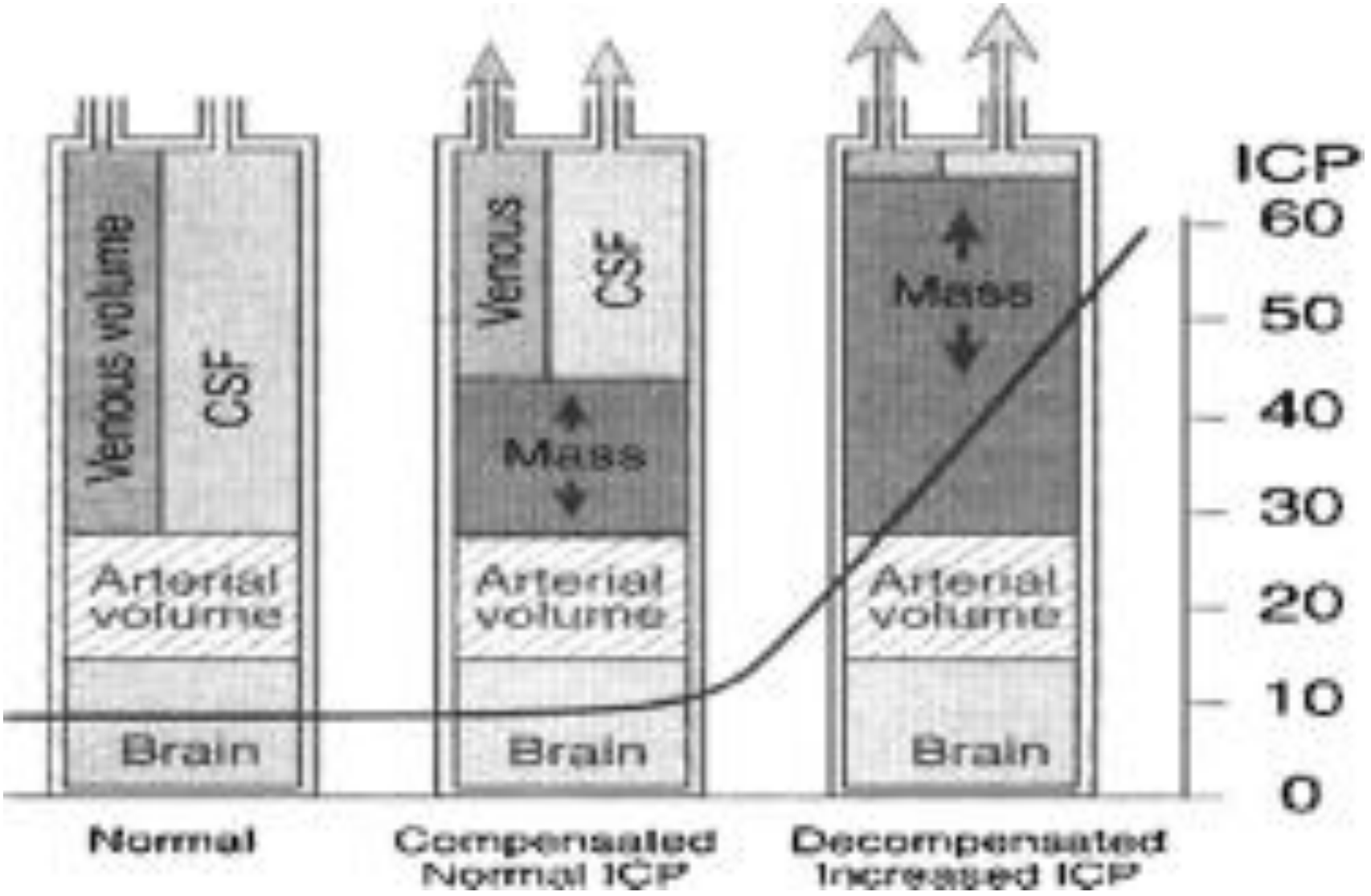
COMPLIANCE

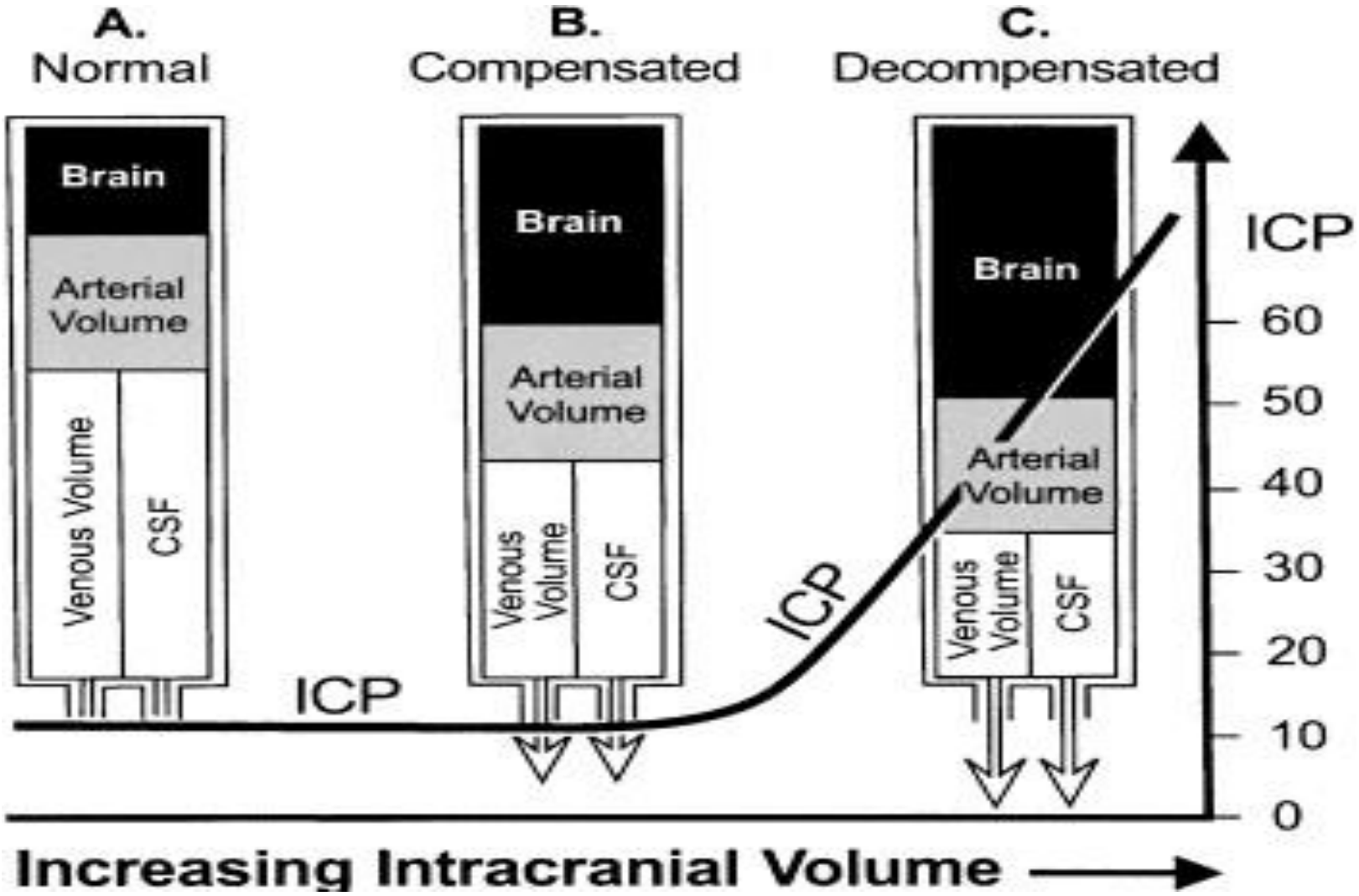
- The change in volume for a given change in pressure (dV/dP)
- Provides an index for compensatory reserve (spinal subarachnoid space, slightly distensible)

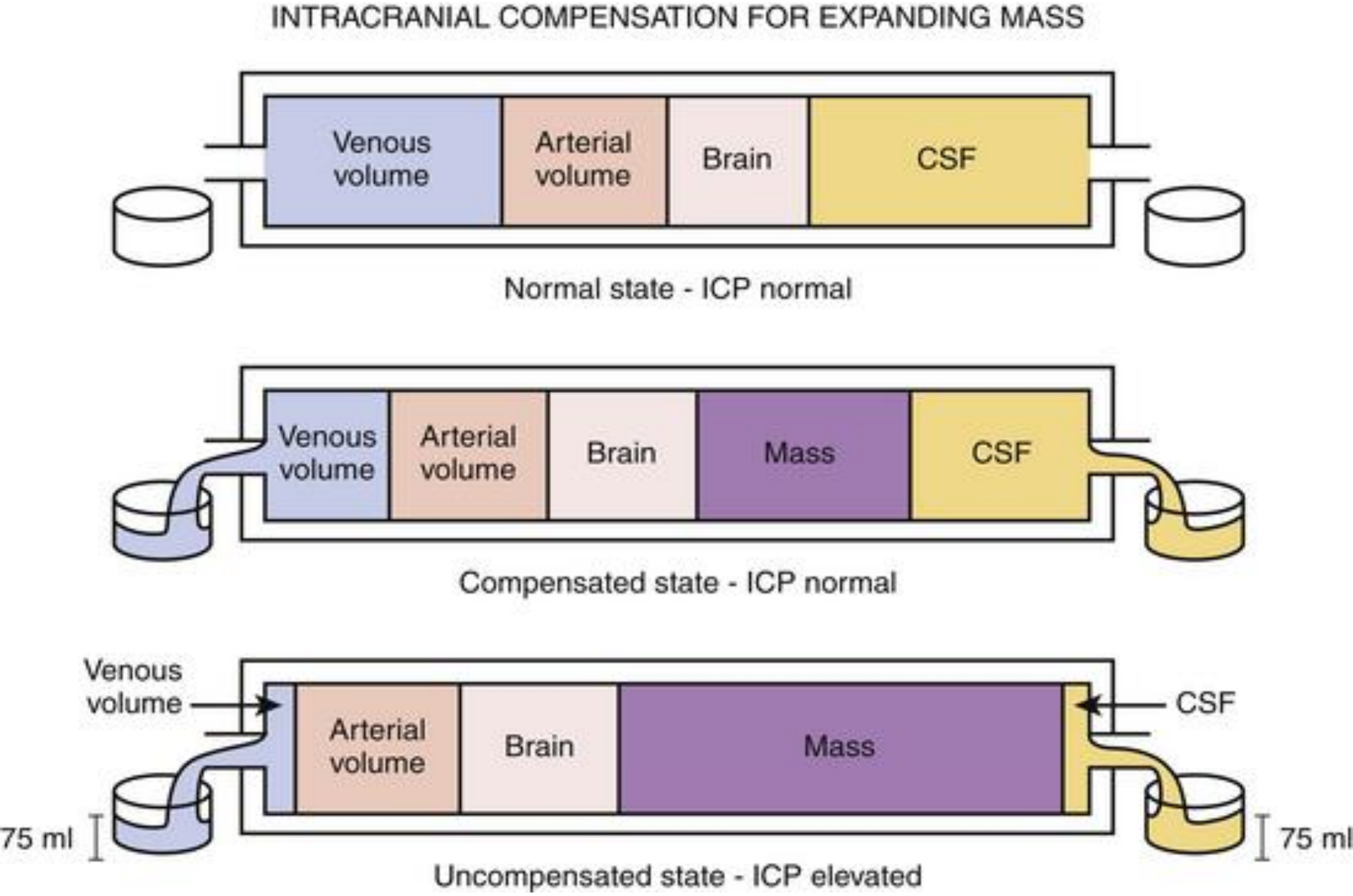
INTRACRANIAL COMPLIANCE CURVE






Venous volume
CSF
Arterial volume
Brain
Normal
Venous
CSF
Mass
Arterial volume
Brain
Compensated
Normal ICP
Mass
Arterial volume
Brain
Decompensated
Increased ICP
ICP
60
50
40
30
20
10
0


A.
Normal
B.
Compensated
C.
Decompensated
Brain
Arterial Volume
Venous Volume
CSF
Brain
Arterial Volume
Venous Volume
CSF
Brain
Arterial Volume
Venous Volume
CSF
ICP
ICP
ICP
60
50
40
30
20
10
0
Increasing Intracranial Volume


INTRACRANIAL COMPENSATION FOR EXPANDING MASS
Venous volume
Arterial volume
Brain
CSF
Normal state - ICP normal
Venous volume
Arterial volume
Brain
Mass
CSF
Compensated state - ICP normal
Venous volume
Arterial volume
Brain
Mass
CSF
75 ml
75 ml
Uncompensated state - ICP elevated

AUTOREGULATION

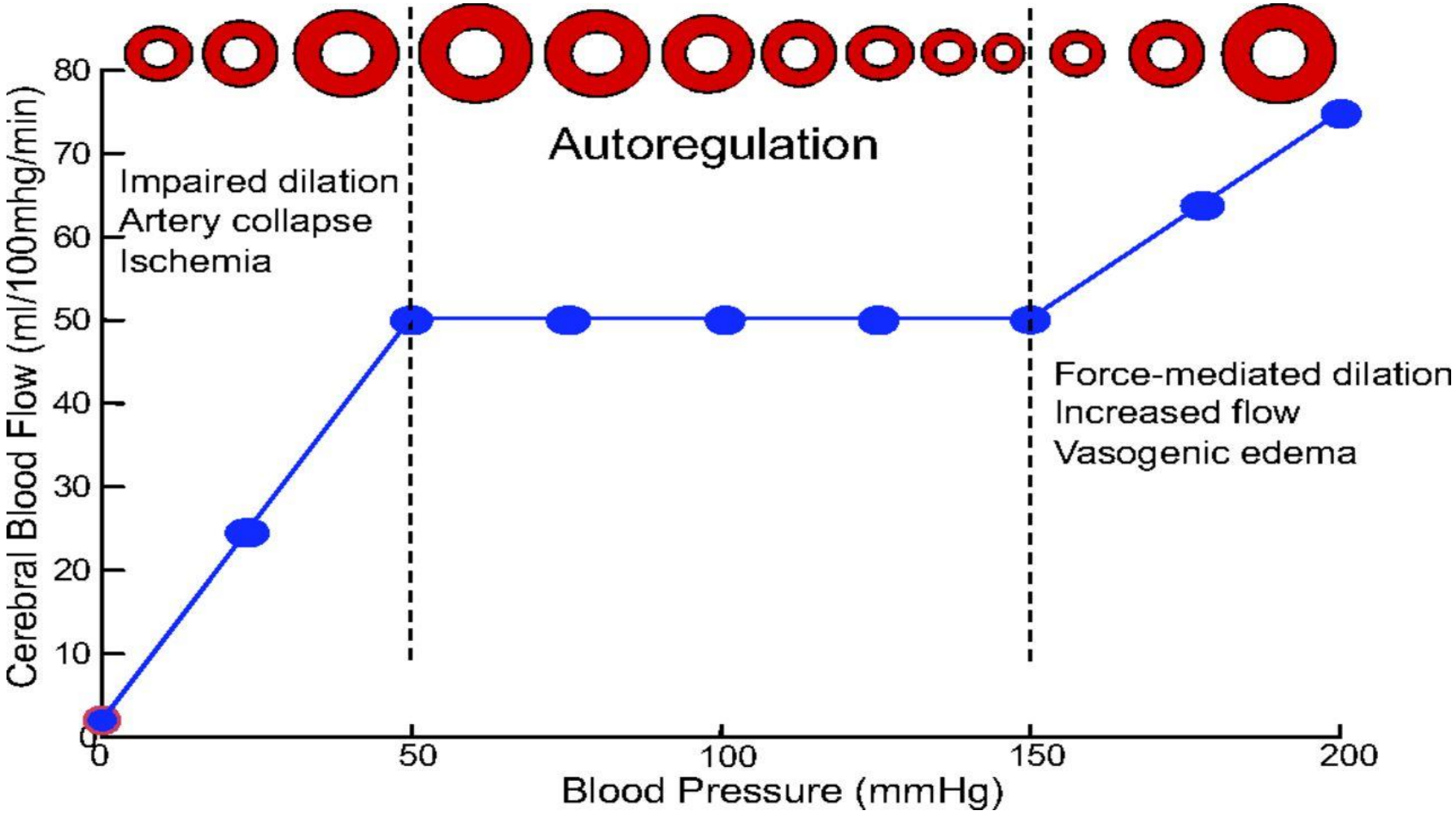
- CBF = CPP/CVR
- CPP = MAP-ICP

- Conditions associated with elevated ICP, including mass lesions and hydrocephalus, can be associated with a reduction in CPP.

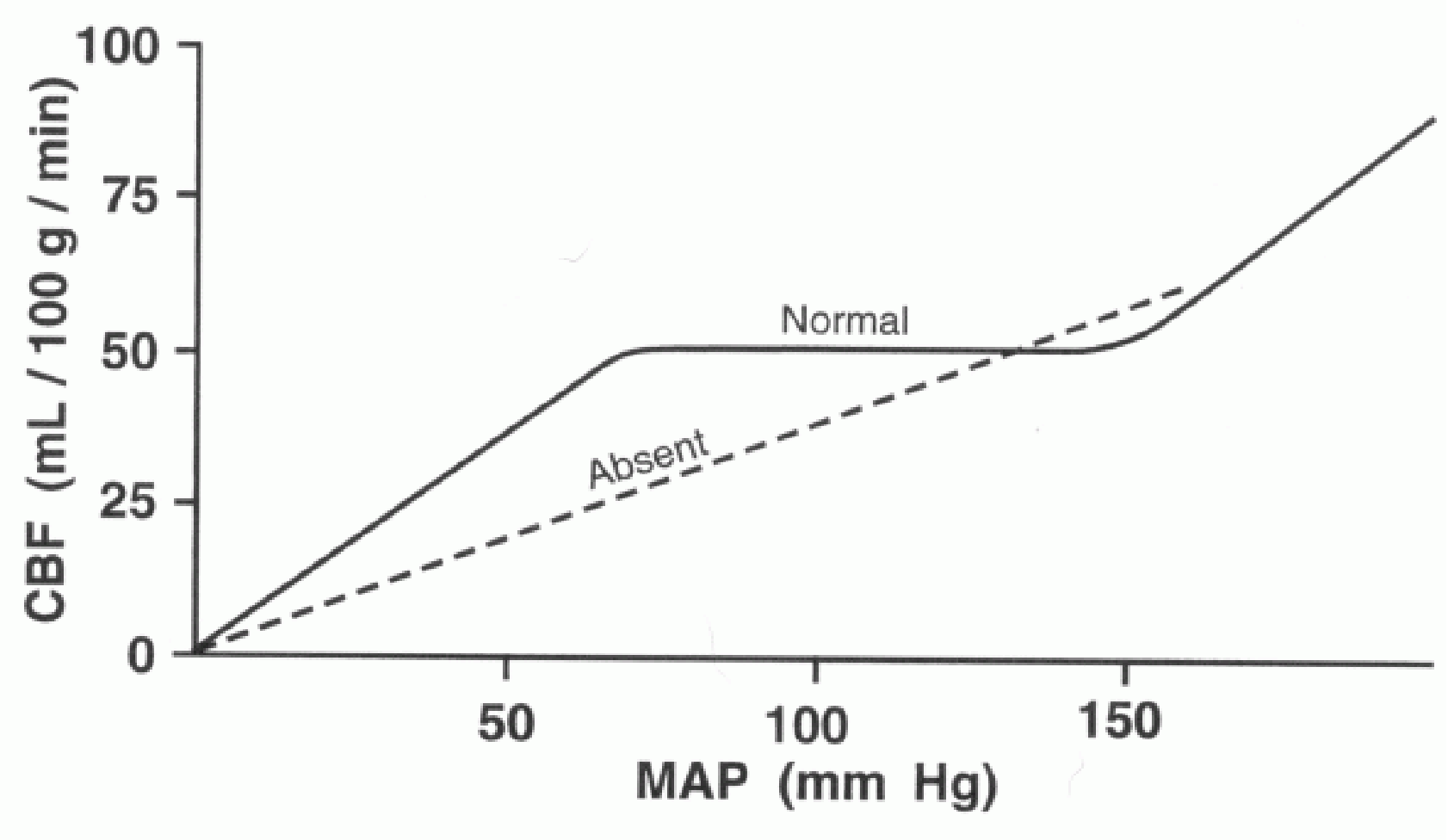
- This can result in devastating focal or global ischemia.

- On the other hand, excessive elevation of CPP can lead to hypertensive encephalopathy and cerebral edema due to the eventual breakdown of autoregulation, particularly if the CPP is >120 mmHg .

AUTOREGULATION



Impaired autoregulation



Increased ICP

- ICP greater than 20-25 mmHg for more than 5 minutes

Physiological variations of ICP

- Over the course of the day (higher in the morning) due to:

1- recumbency

2- rise in $PaCO_2$ during sleep caused by respiratory depression

3- decrease in CSF absorption

- Change in posture or position
- Pressure fluctuations in other compartments (cough, sneezing, straining and valsalva)

Why is ICP important?

1- Raised ICP is the final common pathway that leads to death or disability in most acute cerebral conditions

2- It is potentially treatable

The two major consequences of increased ICP are:

- brain shifts (herniation)
- brain ischaemia

CAUSES OF INCREASED ICP

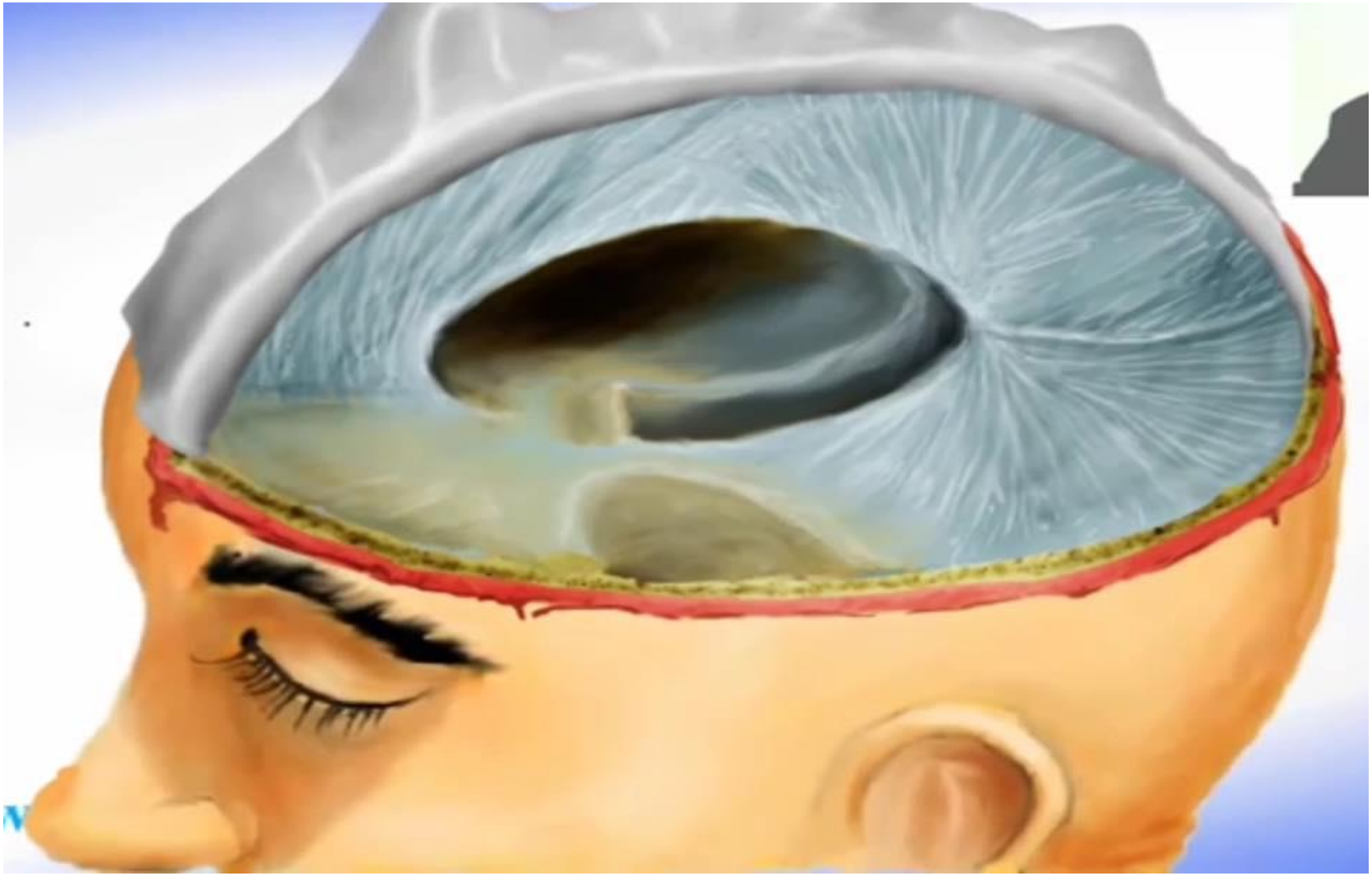
Increased volume of normal intracranial constituents

- brain : cerebral edema (ischemia, CNS infections), benign intracranial hypertension
- CSF: hydrocephalus
- blood volume: vasodilatation due to hypercapnia

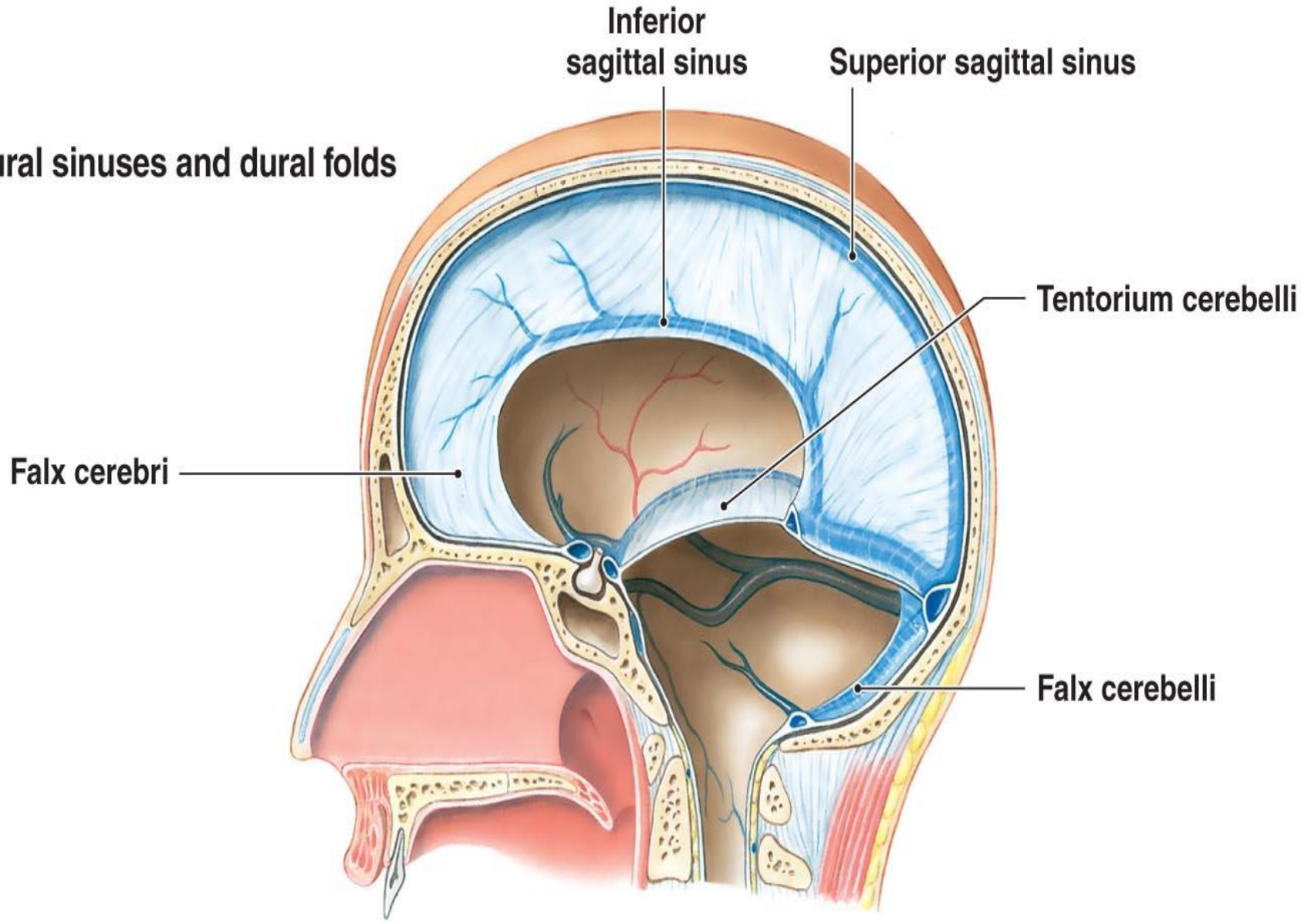
A space-occupying lesion

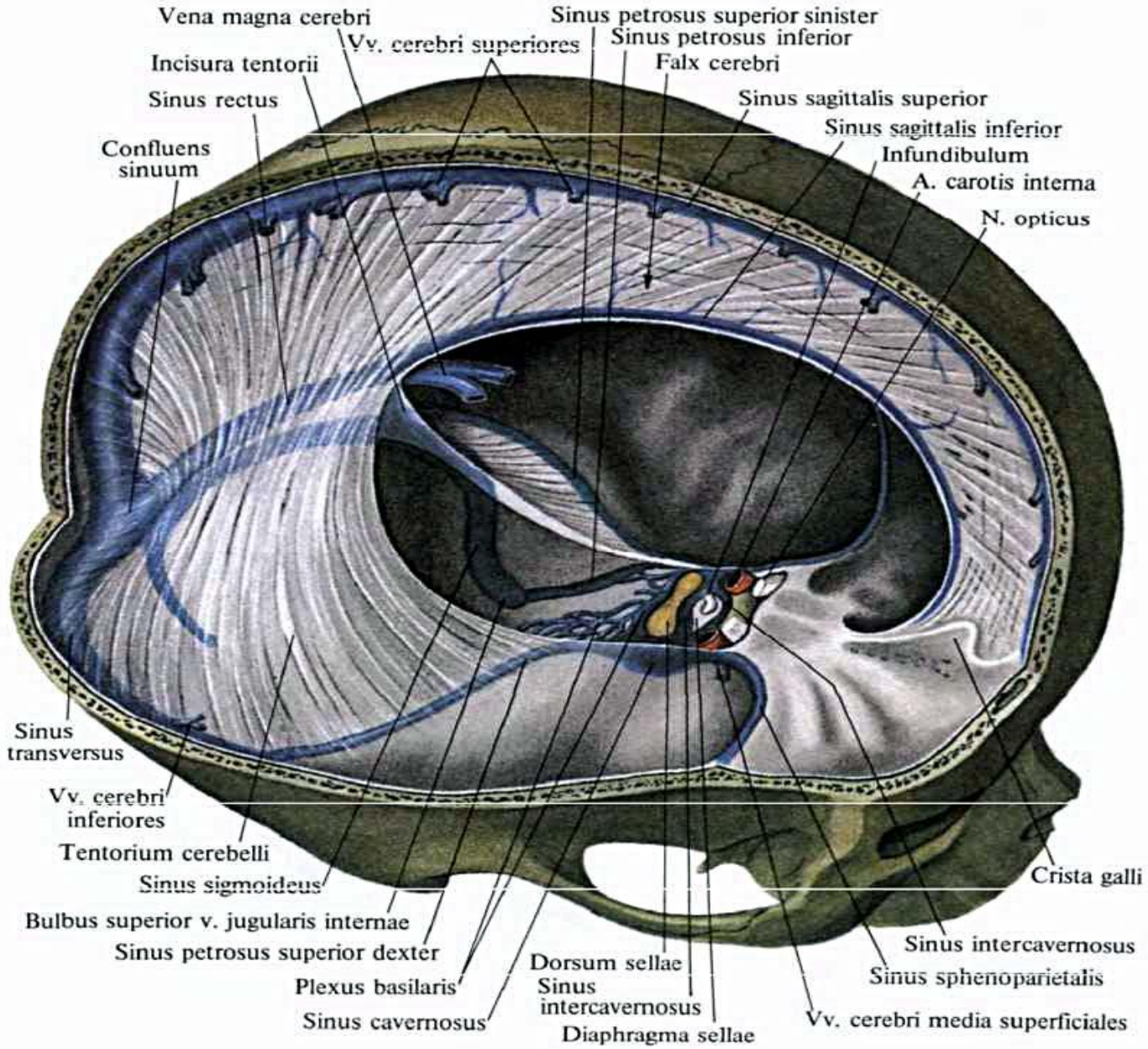
- cerebral tumour
- Abscess
- intracranial haematoma

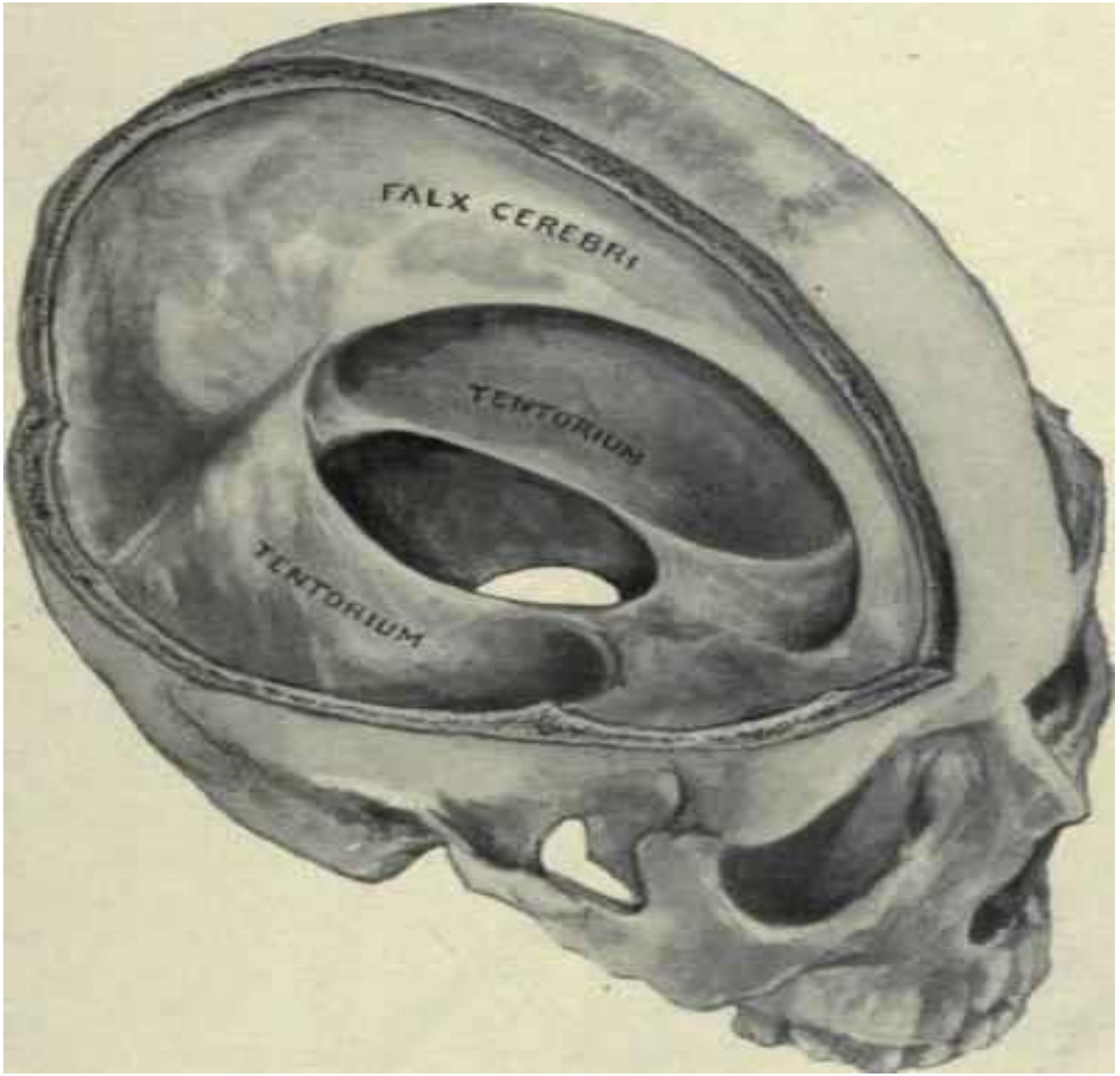
CEREBRAL HERNIATION

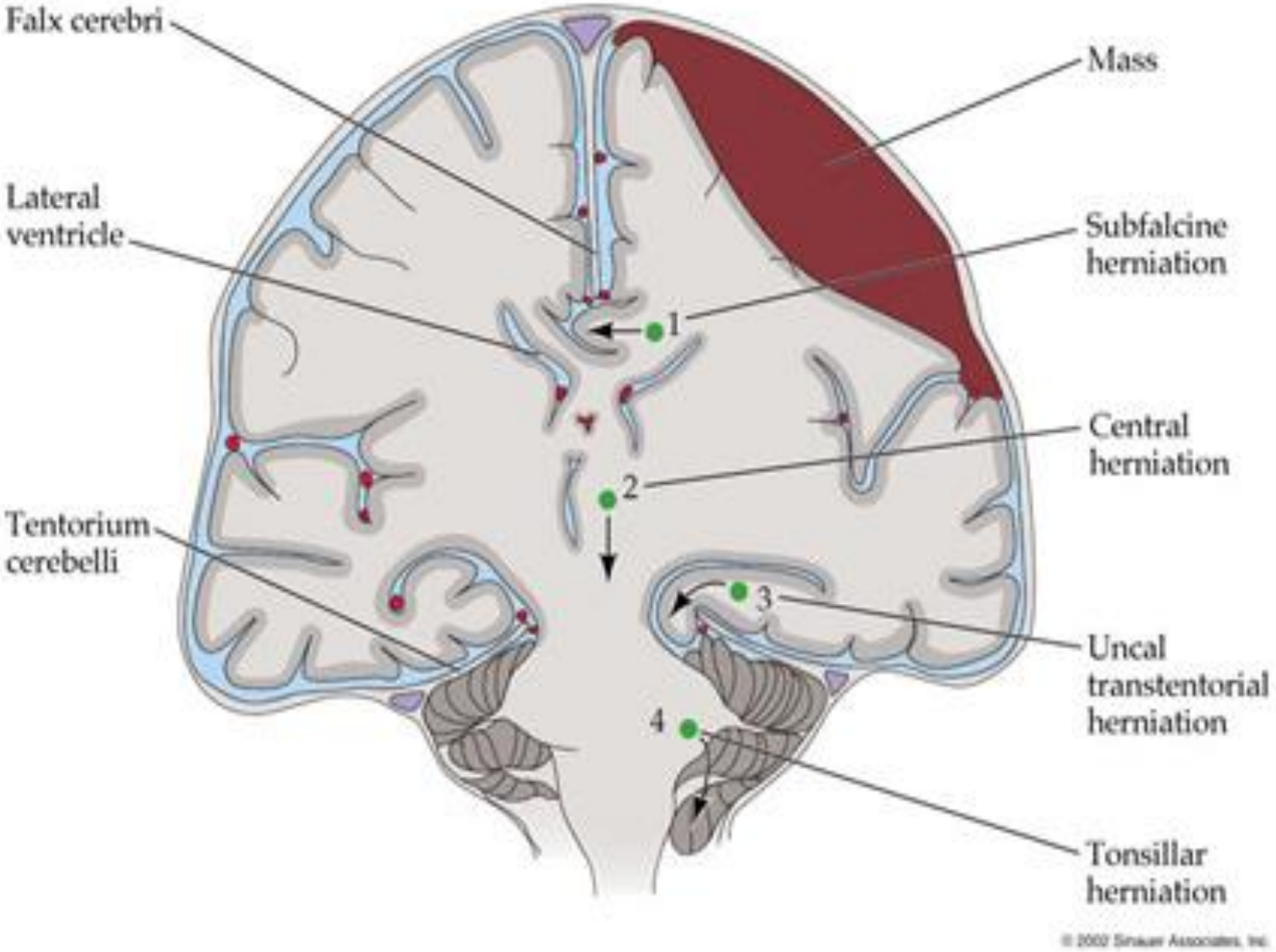


The dural sinuses and dural folds




Vena magna cerebri
Vv. cerebri superiores
Sinus petrosus superior sinister
Sinus petrosus inferior
Falx cerebri
Incisura tentorii
Sinus rectus
Sinus sagittalis superior
Sinus sagittalis inferior
Confluens sinuum
Infundibulum
A. carotis interna
N. opticus
Sinus transversus
Vv. cerebri inferiores
Tentorium cerebelli
Sinus sigmoideus
Crista galli
Bulbus superior v. jugularis internae
Sinus petrosus superior dexter
Sinus intercavernosus
Dorsum sellae
Sinus sphenoparietalis
Plexus basilaris
Sinus intercavernosus
Sinus cavernosus
Vv. cerebri media superficiales
Diaphragma sellae


FALX CEREBRI
TENTORIUM
TENTORIUM


Falx cerebri
Lateral ventricle
Tentorium cerebelli
Mass
Subfalcine herniation
Central herniation
Uncal transtentorial herniation
Tonsillar herniation
1
2
3
4
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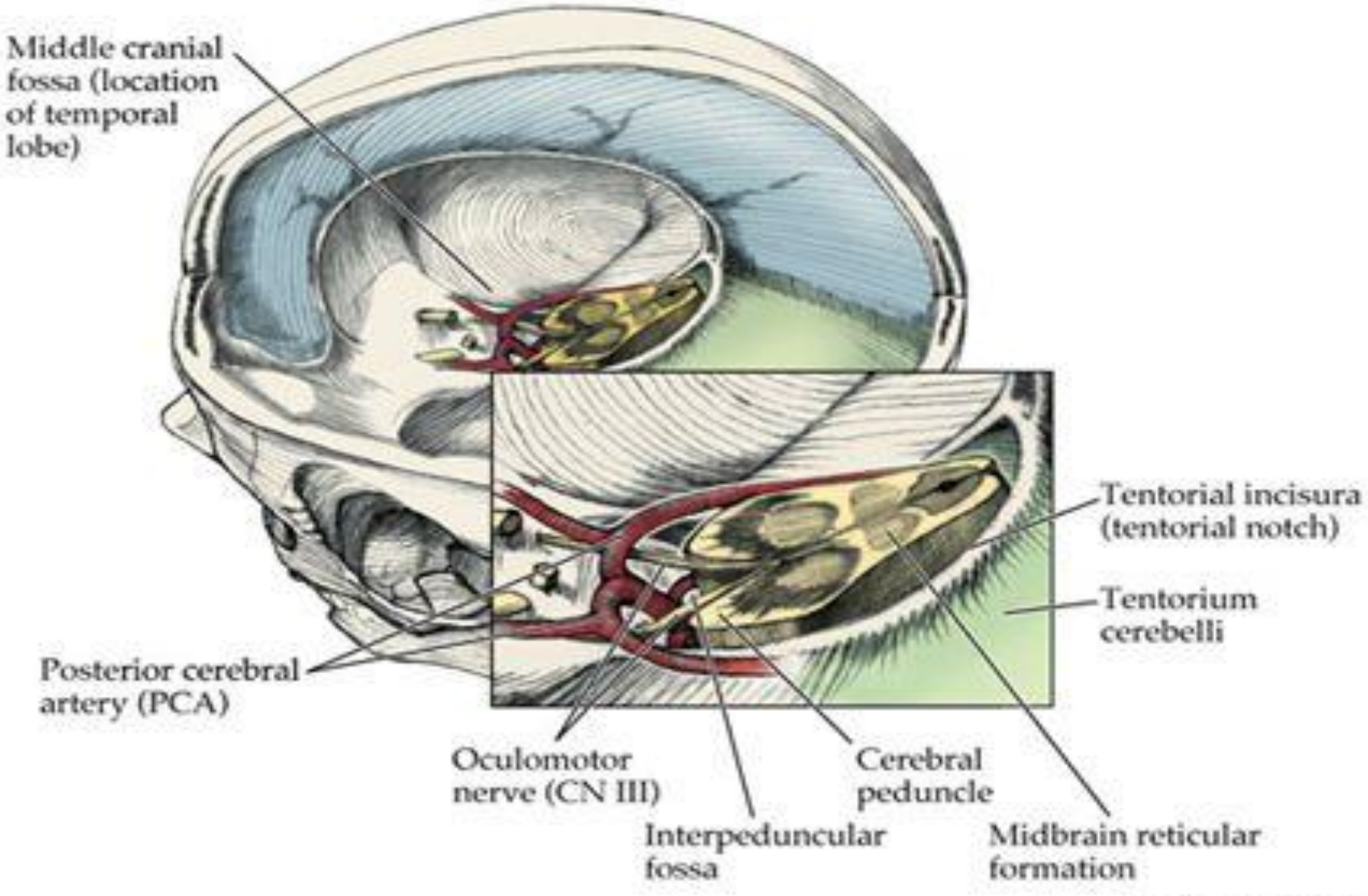
TRANSTENTORIAL HERNIATION



- **3rd cranial nerve**: initial dilatation of the ipsilateral pupil

- **The midbrain**

1- Hemiparesis, usually contralateral, occasional compression of opposite crus cerebri causes ipsilateral hemiparesis as **Kernohan's notch phenomenon** (crus cerebri; pyramidal tract)

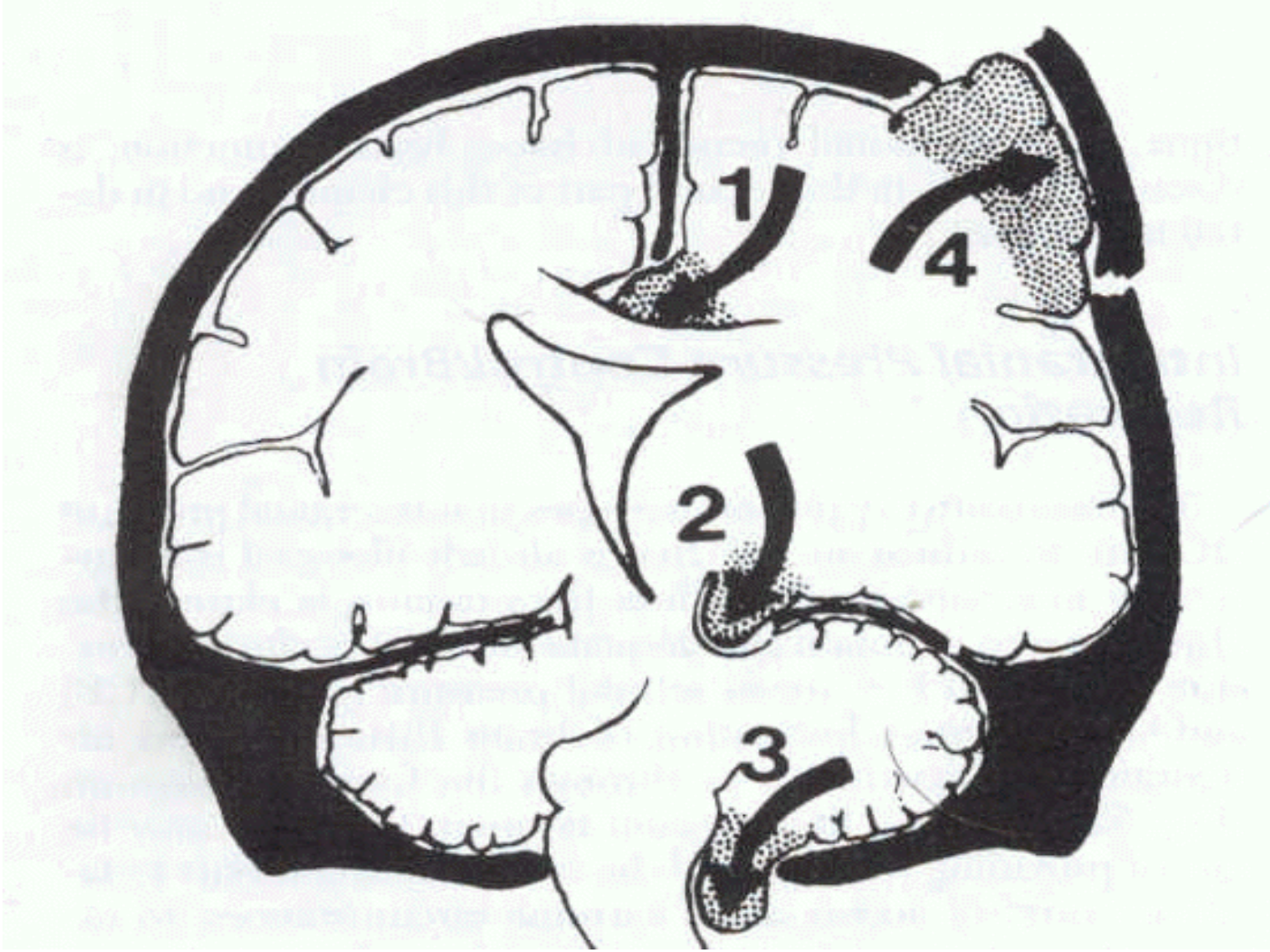
2- Hypertension, bradycardia and respiratory depression — Cushing response

3- Deterioration of conscious level (RAS)

4- Cheyne-Stokes periodic breathing

- **PCA**: hemianopia with macular sparing

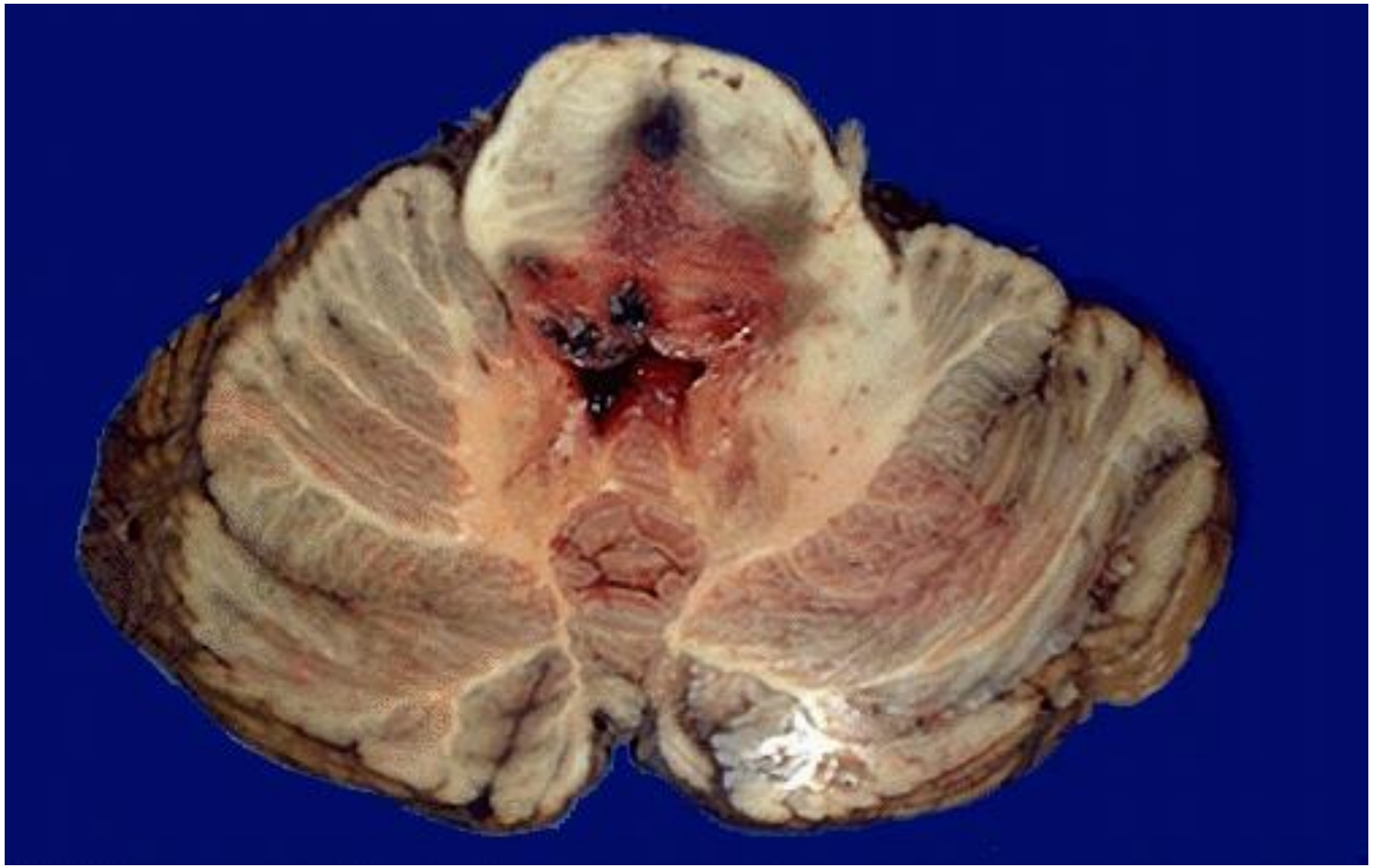
TONSILLAR HERNIATION



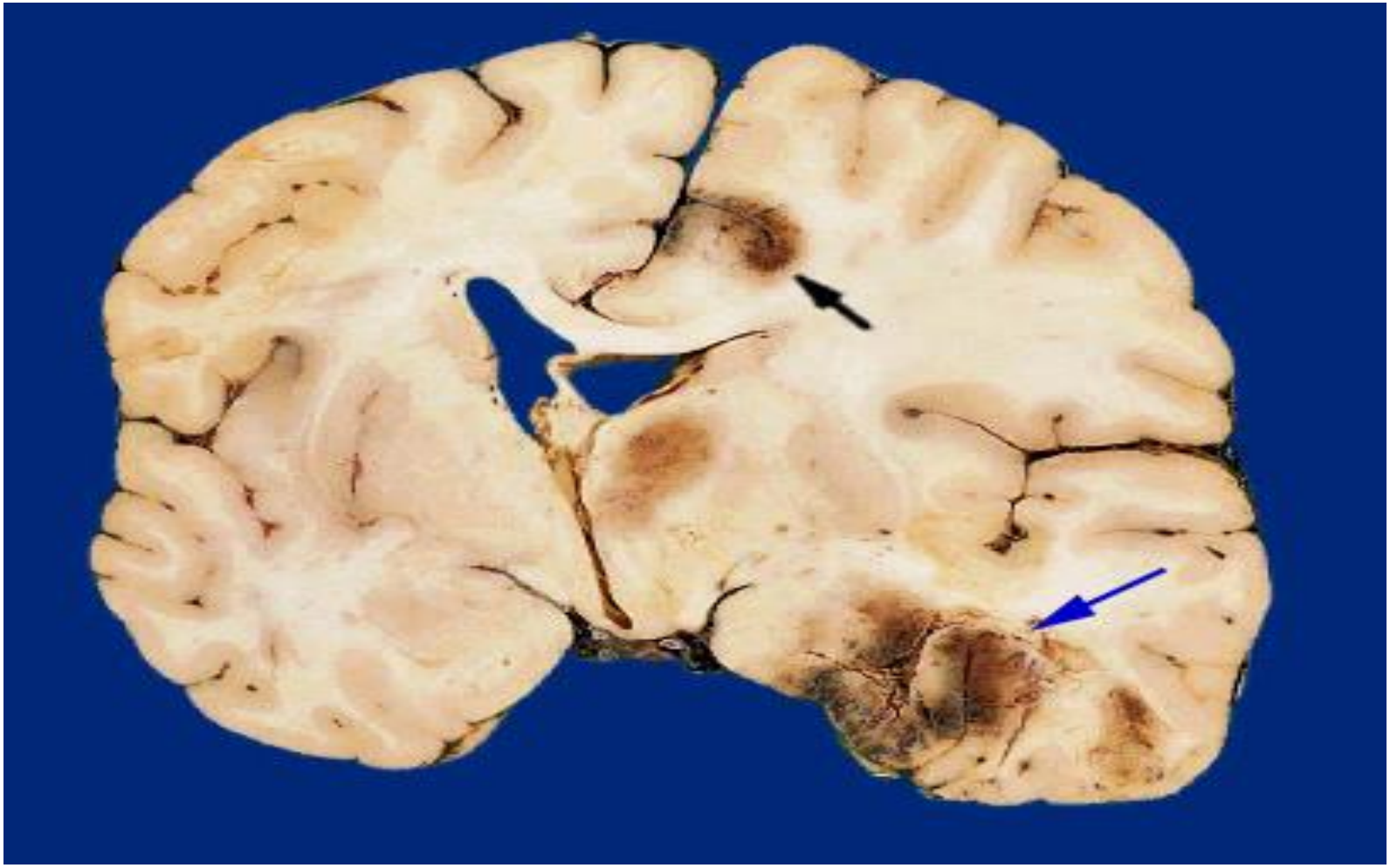
- Abnormal neck posture (head tilting in children) .. If slowly progressive
- Neck stiffness : irritation of dura around foramen magnum >> beware of performing LP

- Rapid respiratory failure (Cheyne-Stokes brathing)
- Limb paresis and sensory disturbance
- Coning: shearing of the perforators supplying the brainstem (Duret hemorrhages)
- Diabetes insipidus: traction damage to pituitary stalk
- Pupils change from dilated and fixed midsize and unreactive
- Brainstem death

Duret hemorrhages



Cingulate (Subflacian) Herniation



- ✓ Caused by frontal mass ex. Hematoma.
- ✓ May be silent unless the anterior cerebral artery is compressed and cause stroke.
- ✓ Results in :
- contralateral leg weakness.
- Abulia (lack of decision making).

CLINICAL PRESENTATION

- Classical triad:

1- headache

2- papilledema

3- projectile vomiting

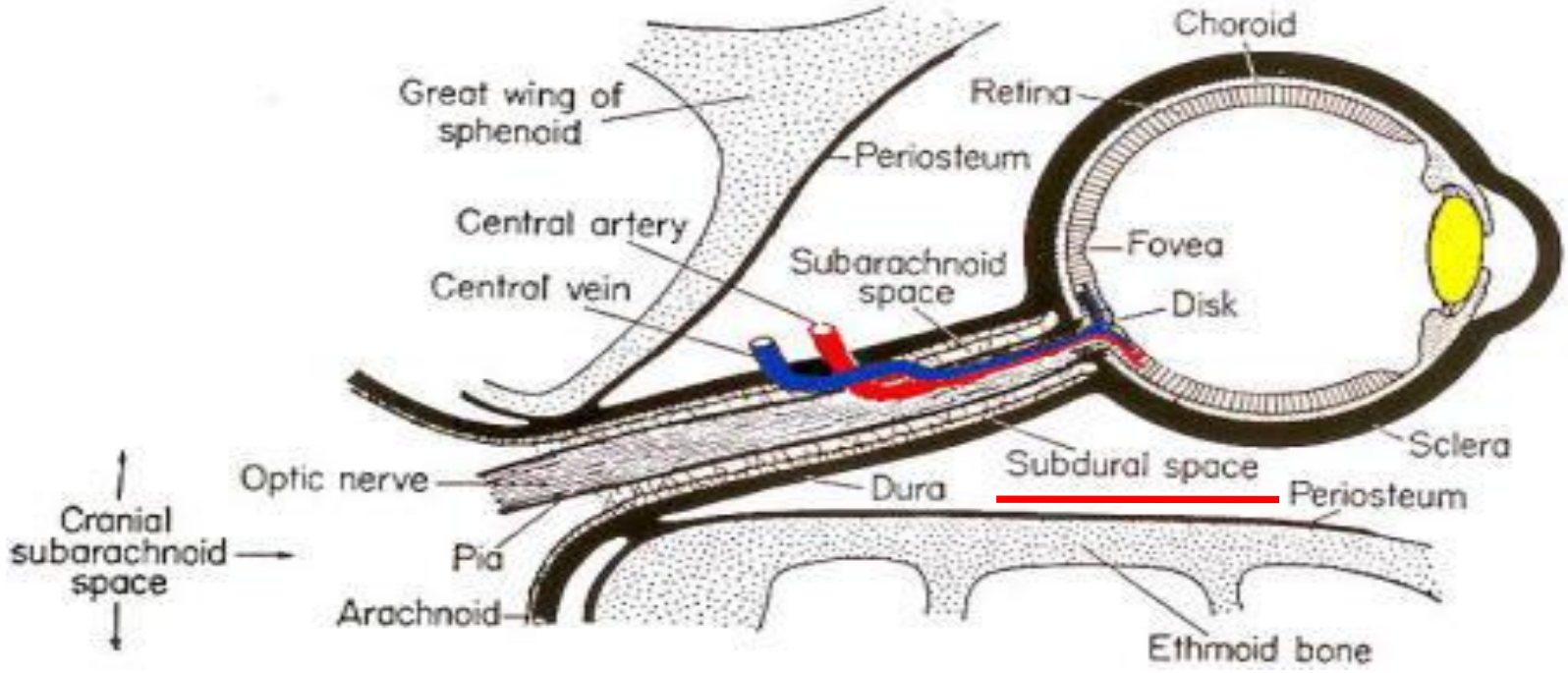
- There is no consistent relation between the severity of symptoms and the degree of hypertension

CLINICAL PRESENTATION

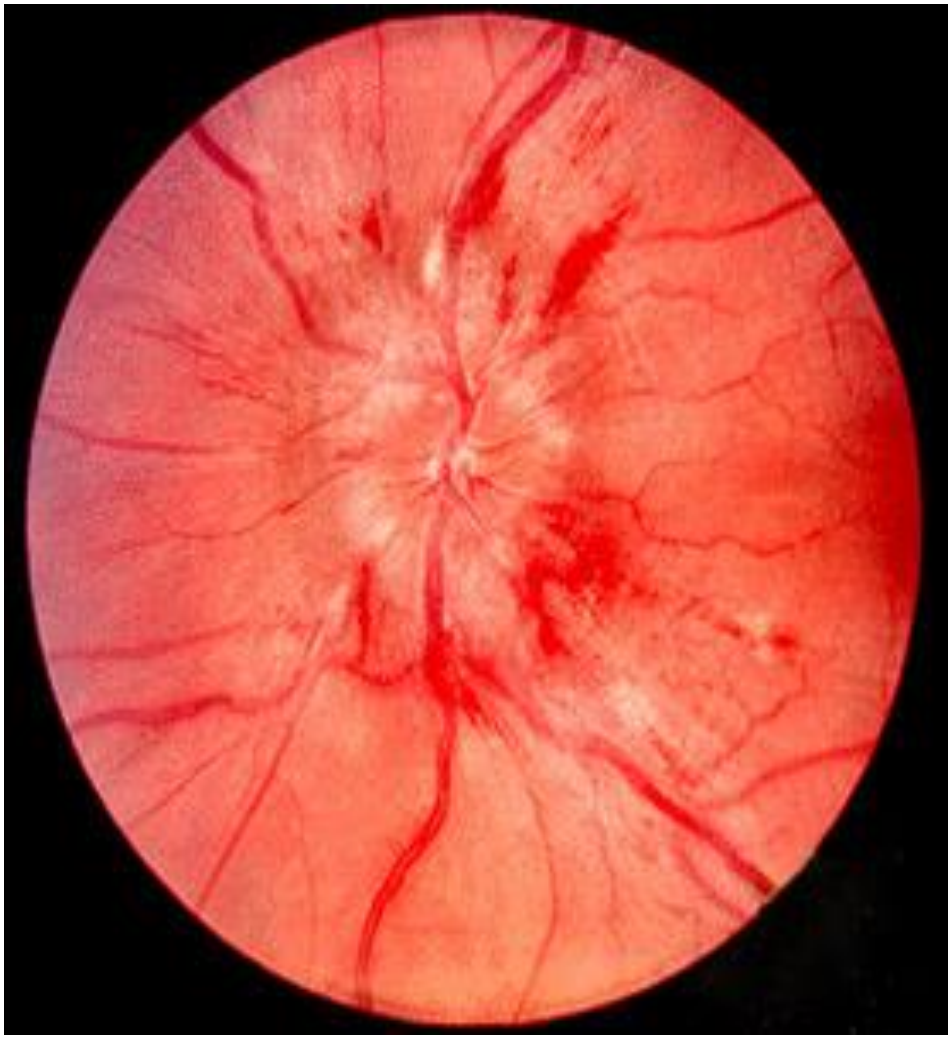
- **Headache**:
- throbbing or bursting
- 3.exacerbated by any factors that further increase ICP such as coughing, sneezing, or exertion
- Worse on waking
- Relieved by vomiting
- Due to traction on the pain-sensitive blood vessels and compression of the pain-sensitive dura at the base of the cranium
- **Nausea and vomiting**:projectile.. Late.. worse in the morning
- **Drowsiness:** assessed by GCS
- **Papilledema**

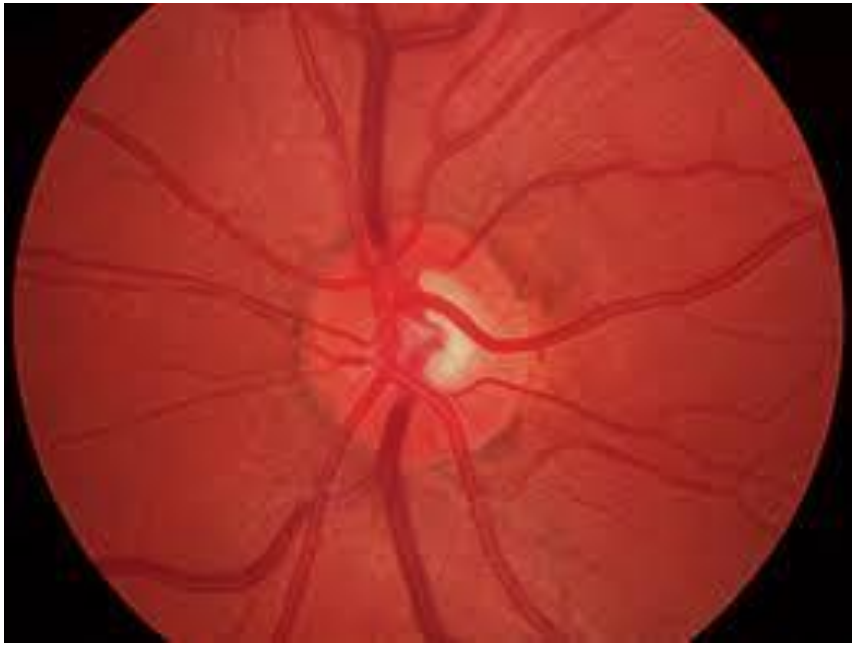
Papilledema

- Due to transmission of the raised pressure along the subarachnoid sheath of the optic nerve



- Filling of the optic disc
- Loss of physiologic cupping
- Retinal veins congestion
- Failure of normalpulsations of the the retinal veins

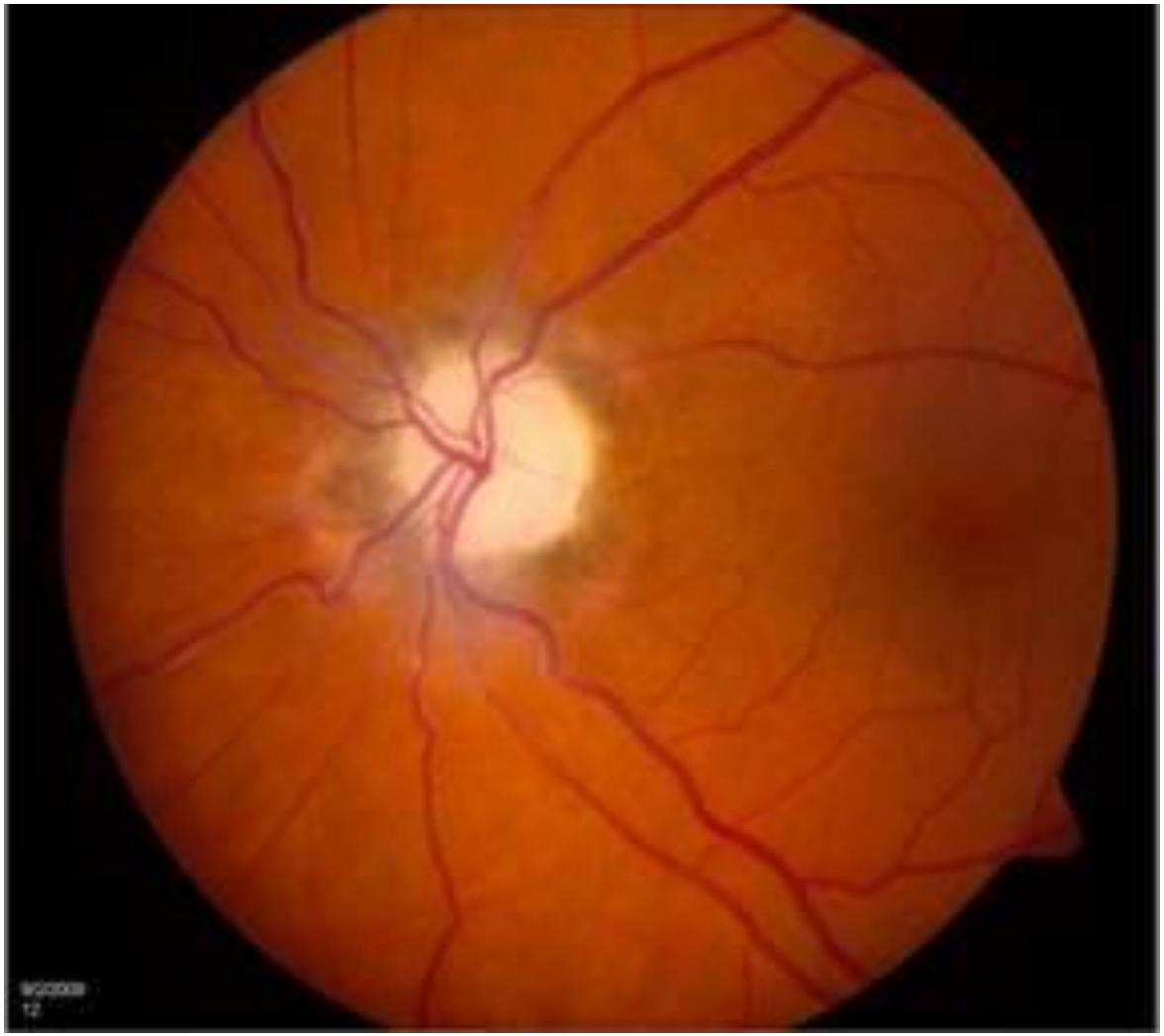




Flame-shaped hemorrhages

acute and severe rises in ICP

Secondary optic atrophy .. longstanding



Absence of papilledema doesn`t exclude increased ICP

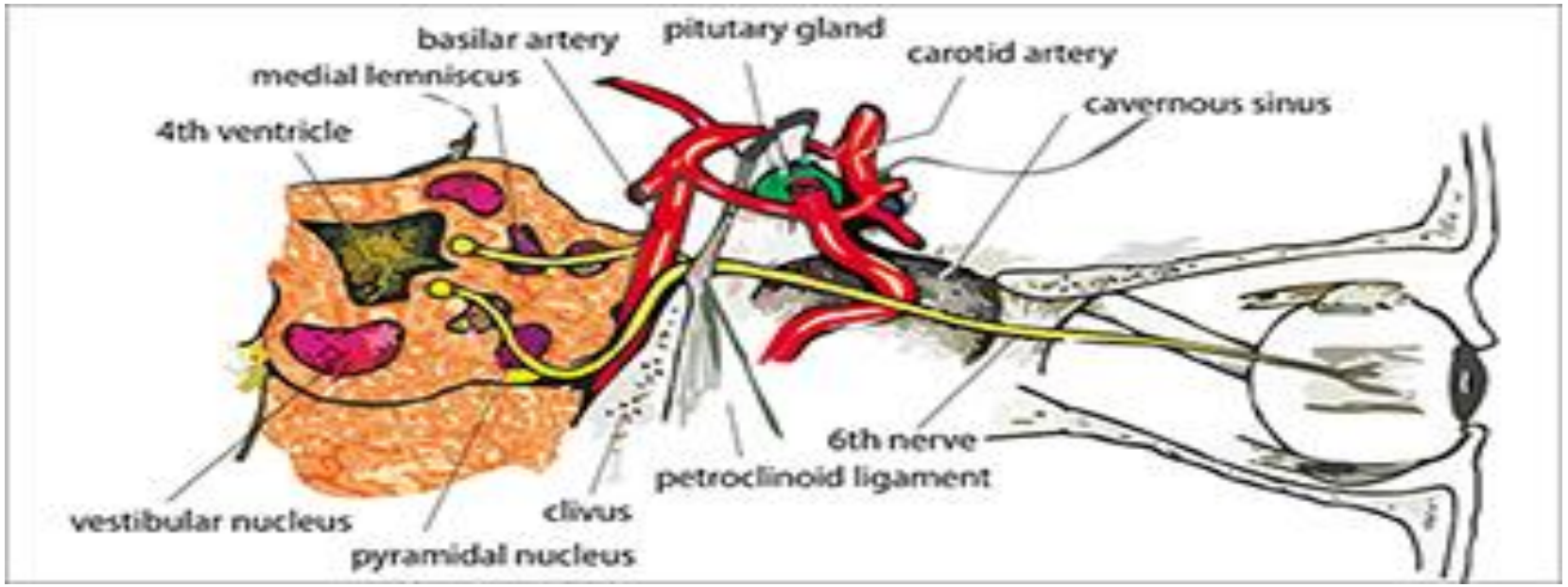
- Can require several days of raised pressure to develop
- Acutely increased ICP may present with rapidly deteriorating consciousness without focal neurological signs or papilledema
- Long standing raised ICP may fail to cause papilloedema if the subarachnoid sleeve around the optic nerve does not communicate with the subarachnoid space.

CUSHING TRIAD/RESPONSE

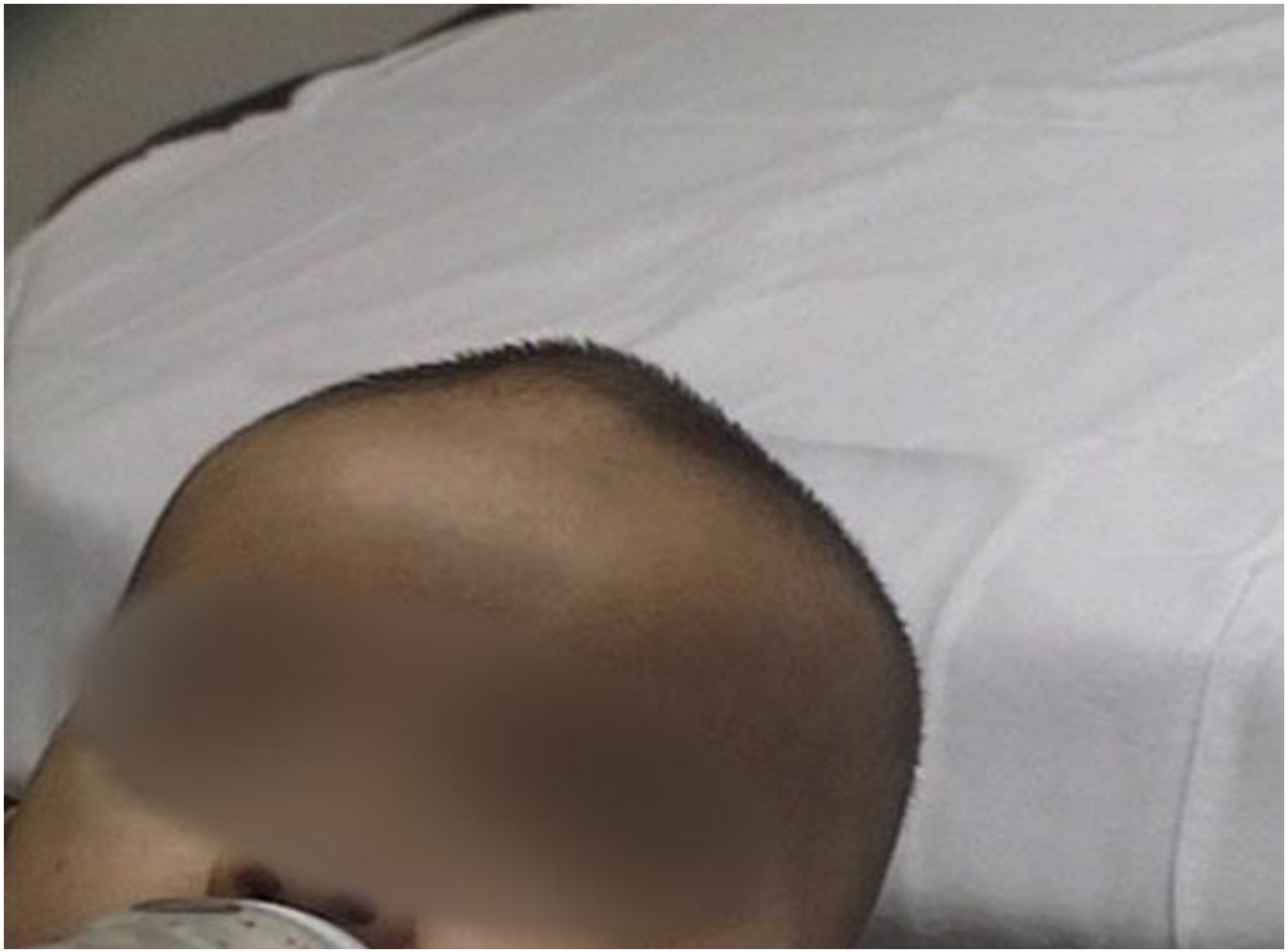
- bradycardia, respiratory depression, and hypertension
- As intracranial pressure rises, in order to maintain a constant CPP, there has to be a compensatory rise in the systemic blood pressure.
- A hypertensive response is therefore elicited which is classically associated with a bradycardia
- Usually associated with widened pulse pressure
- requires urgent intervention.

Sixth Nerve Palsy : false localizing sign

- Causes diplopia
- Due to stretching of the nerve in its long intracranial course, or compression against the petrous ligament or the ridge of the petrous temporal bone



Bulging fontanelle in infants



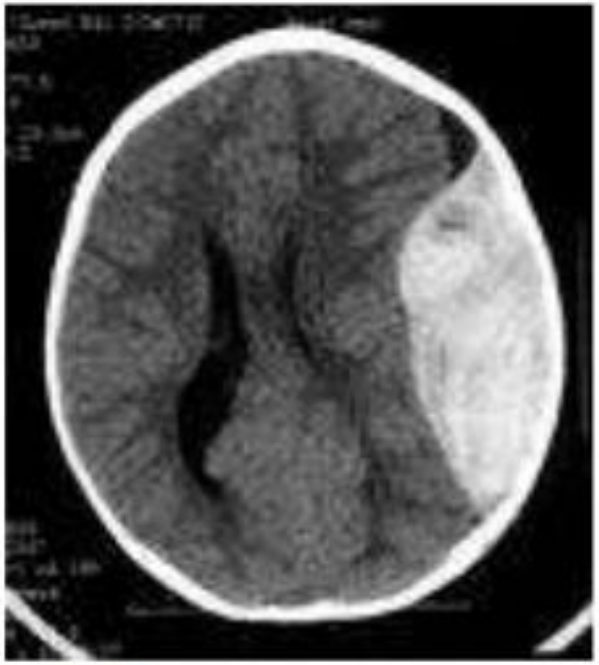
CT SCAN

- When raised ICP is suspected for any reason, it is important not to perform a lumbar puncture prior to a CT scan that would indicate if it is probably safe to do so.

- up to one-third of patients with initially normal scans developed CT scan abnormalities within the first few days after closed head injury .

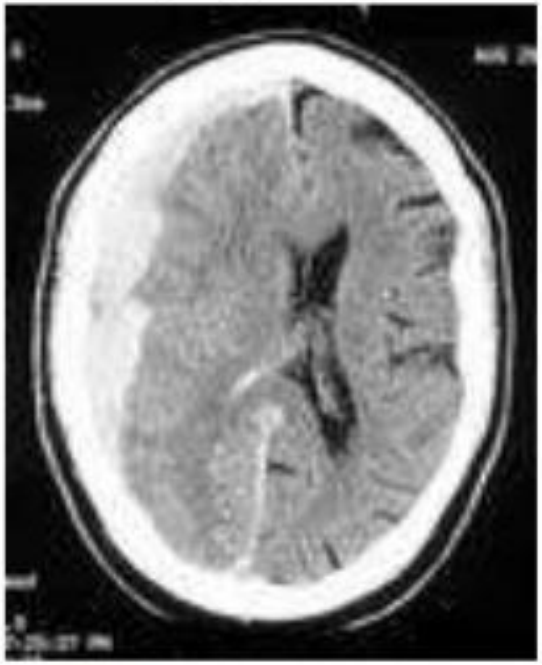
Classic Radiographic Findings

- Epidural Hematoma
  - Middle Meningeal Artery



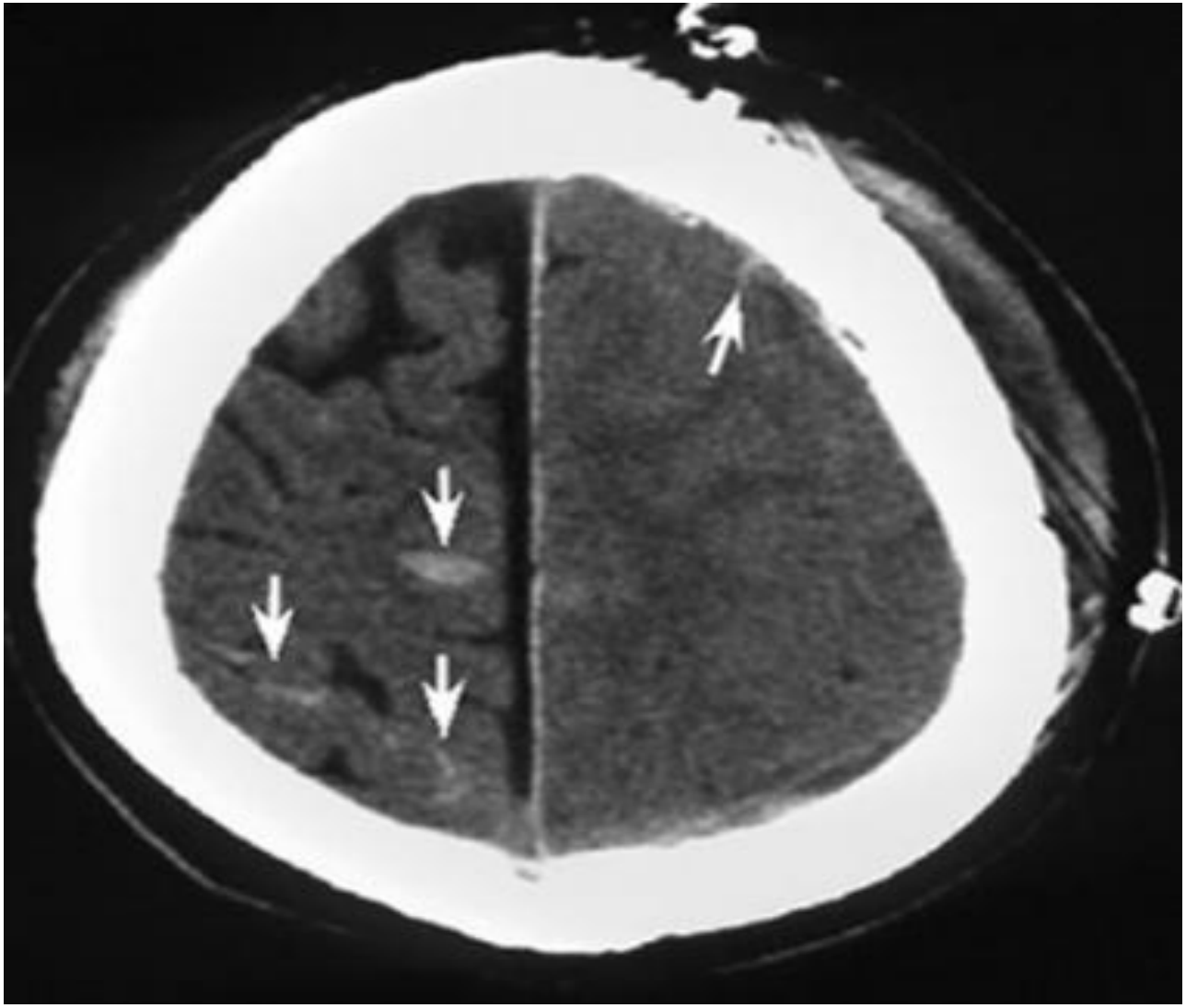
PD-1NEL Author unknown, http://rad.usuhs.mil/medpix/tachy_pics/thumb/synpic4098.jpg

- Subdural Hematoma
  - Bridging Veins

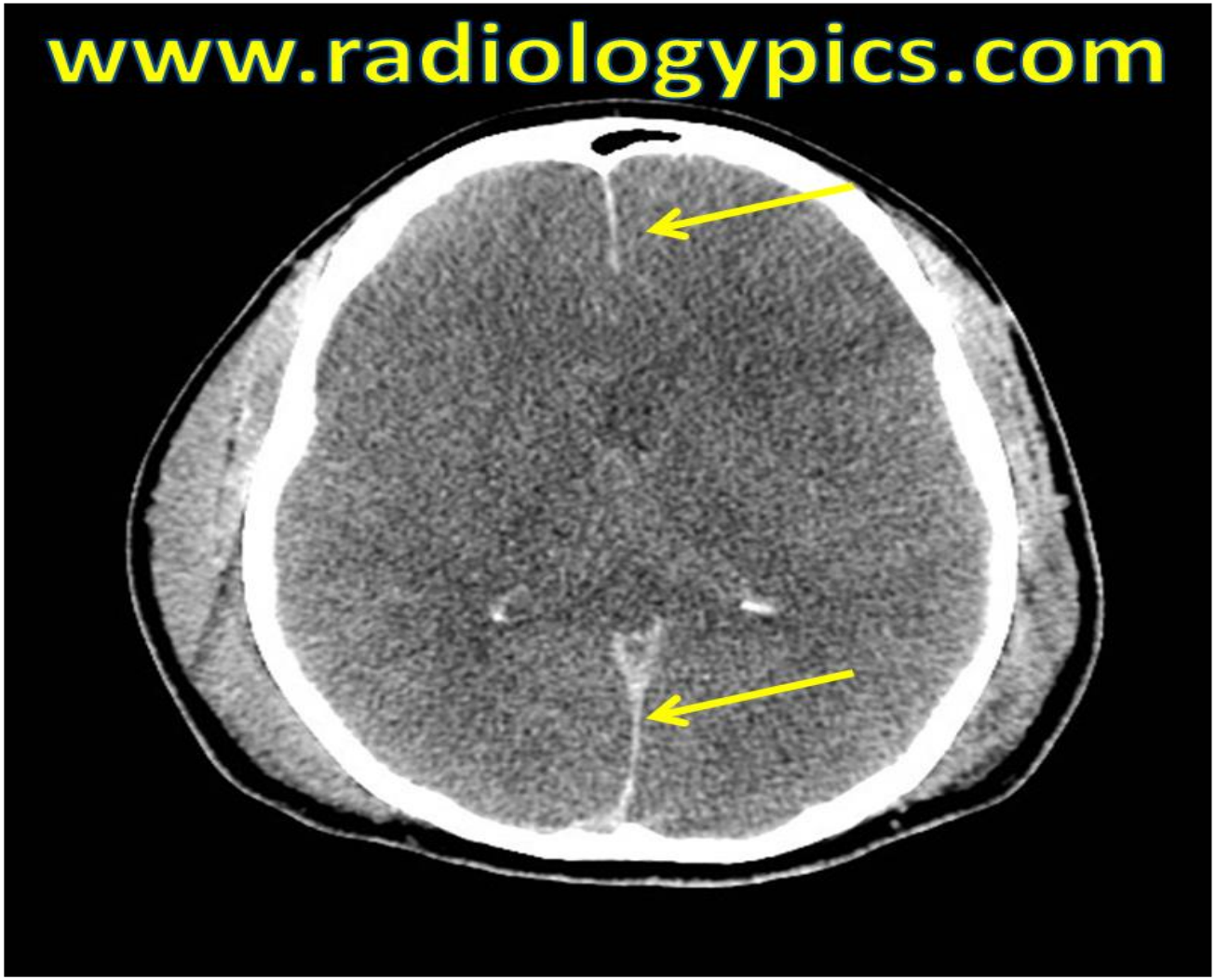


PD-1NEL Author unknown, http://rad.usuhs.edu/medpix/tachy_pics/thumb/synpic519.jpg

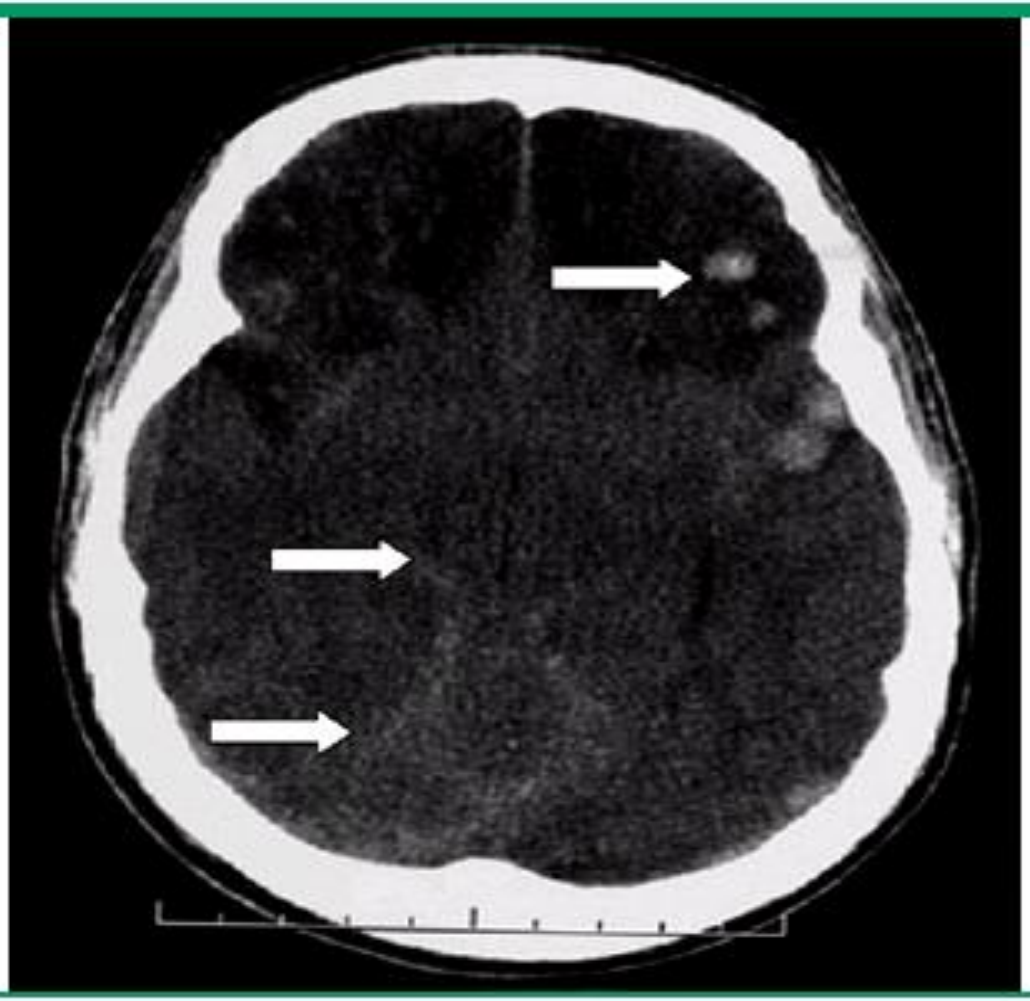
CONTUSIONS



CEREBRAL EDEMA



Radiographic findings suggestive of elevated ICP



Evidence of contusions with surrounding edema (top arrow), effacement of cisterns (middle arrow), and effacement of sulci (lowest arrow).

ICP MONITORING

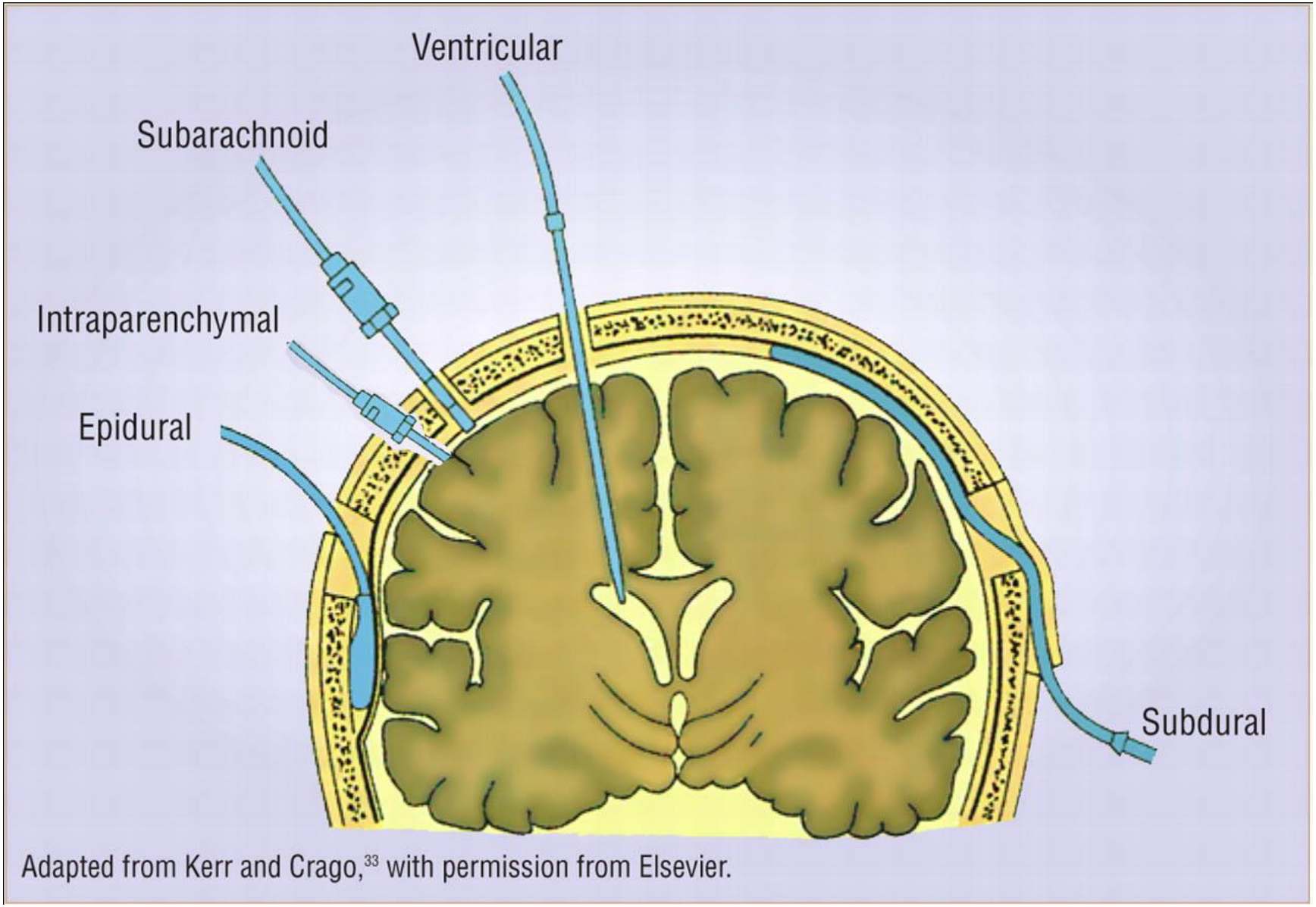
- INDICATIONS:

1- Severe closed head injury > most common

2- Following major intracranial surgery (e.g. posterior fossa surgery)

3- Benign intracranial hypertension : often by lumbar subarachnoid catheter

* The major complication is infection



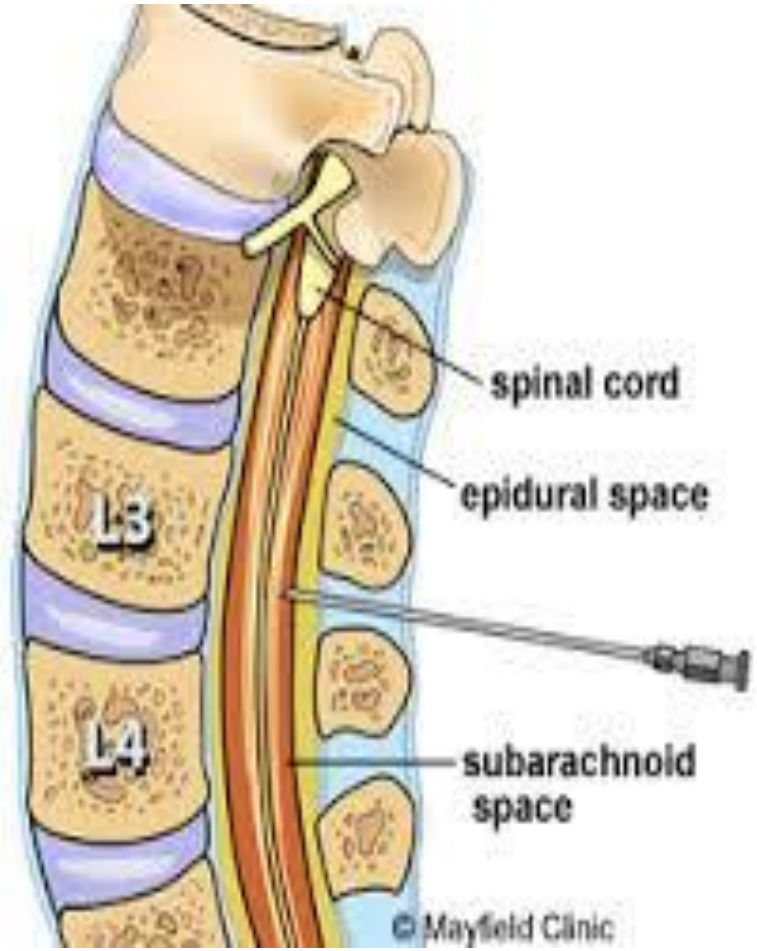
Adapted from Kerr and Crago,[33] with permission from Elsevier.

Interventricular catheter

- Gold standard technique
- It is not subject to the development of intercompartmental pressure gradient
- Provides a method for treatment by CSF drainage
- Zero drift
- Risk of infection 20%, hematoma 2%

Lumbar puncture

- Simple.
- Spinal fluid doesn't reflect the ICP if there is no communication.
- May cause acute brain stem compression



ICP WAVEFORMS

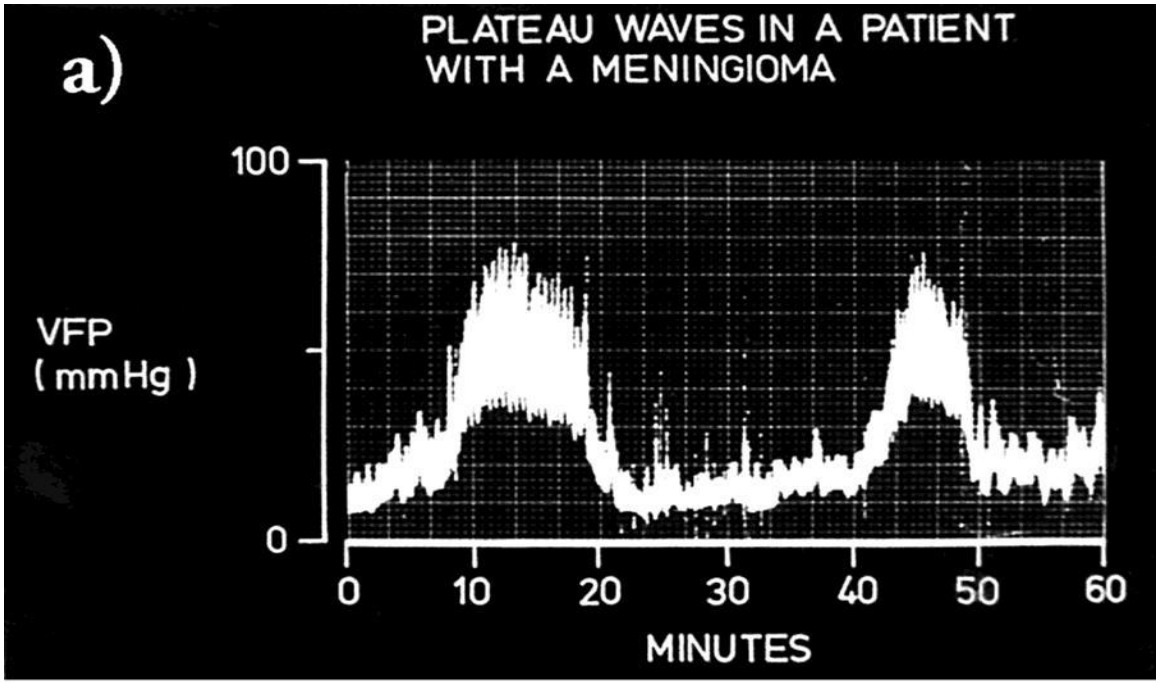
* The major abnormalities in the pressure are:
- elevation of the baseline intracranial pressure
- the development of pressure waves

* The waveforms result from :

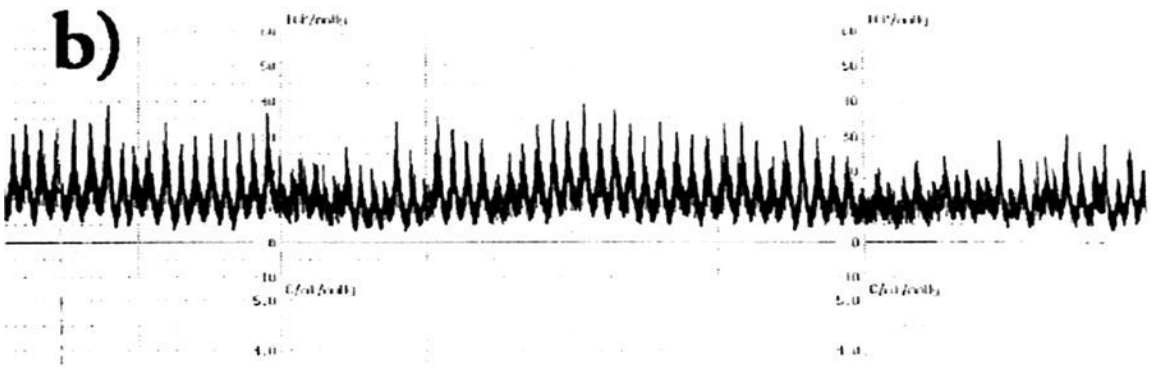
1- SBP pulsations transmitted in the intracranial cavity

2- effects of respiratory cycle on venous outflow

ICP WAVEFORMS



- CHEYNE-STOCKS RESPIRATION



MANAGEMENT

- Definitive treatment involves removing the underlying cause

- Head of bed elevation to 30 degrees improves venous return
- Hyperventilation > decreases PaCO2 > vasoconstriction > lowers ICP

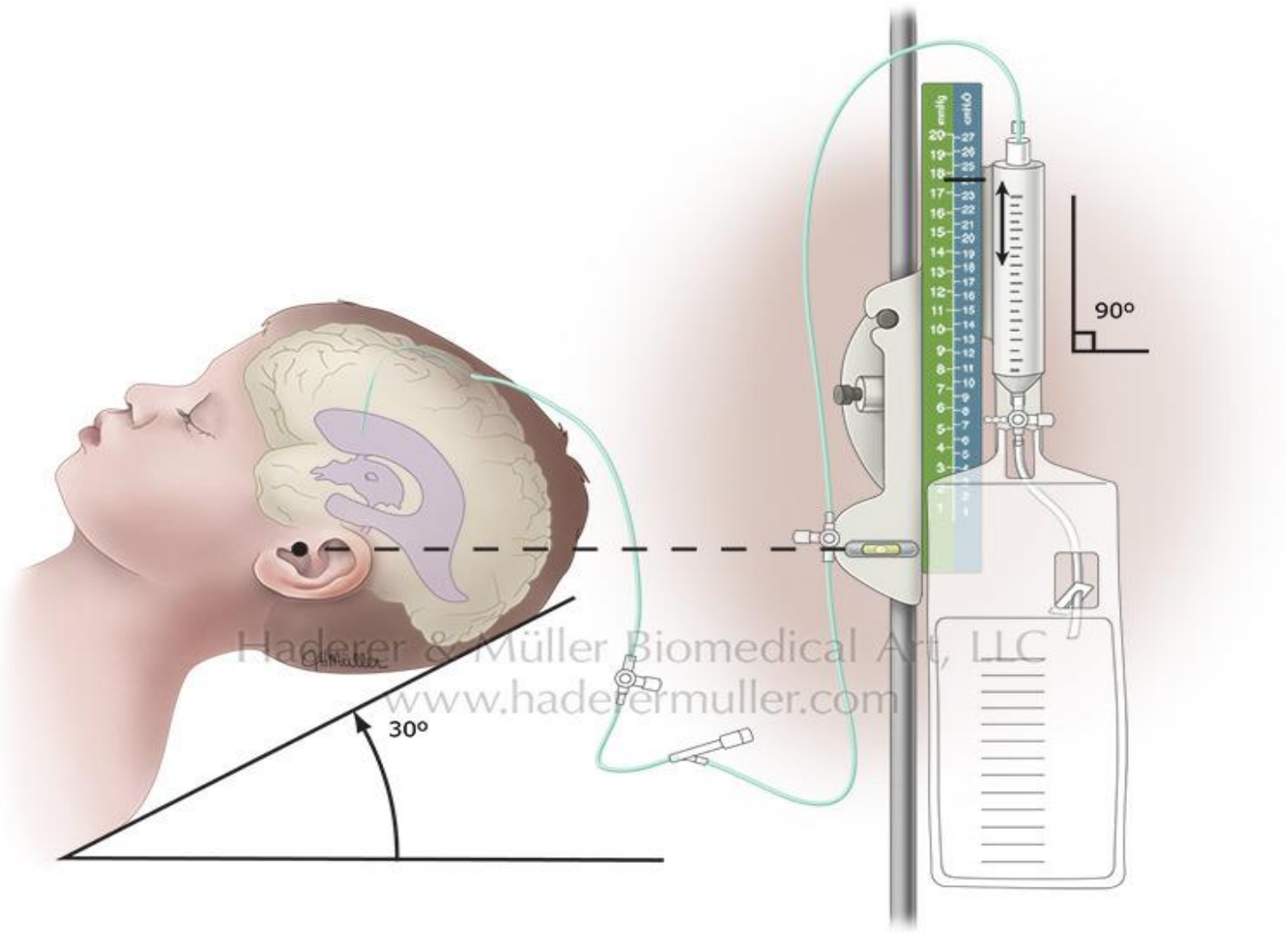
Medical management

- Sedation > propofol or benzodiazepines : decreases metabolic demand, and anticonvulsant activity
- Diuretics > mannitol
- Steroids : vasogenic edema (commonly with tumors
- Hypothermia : 34 C

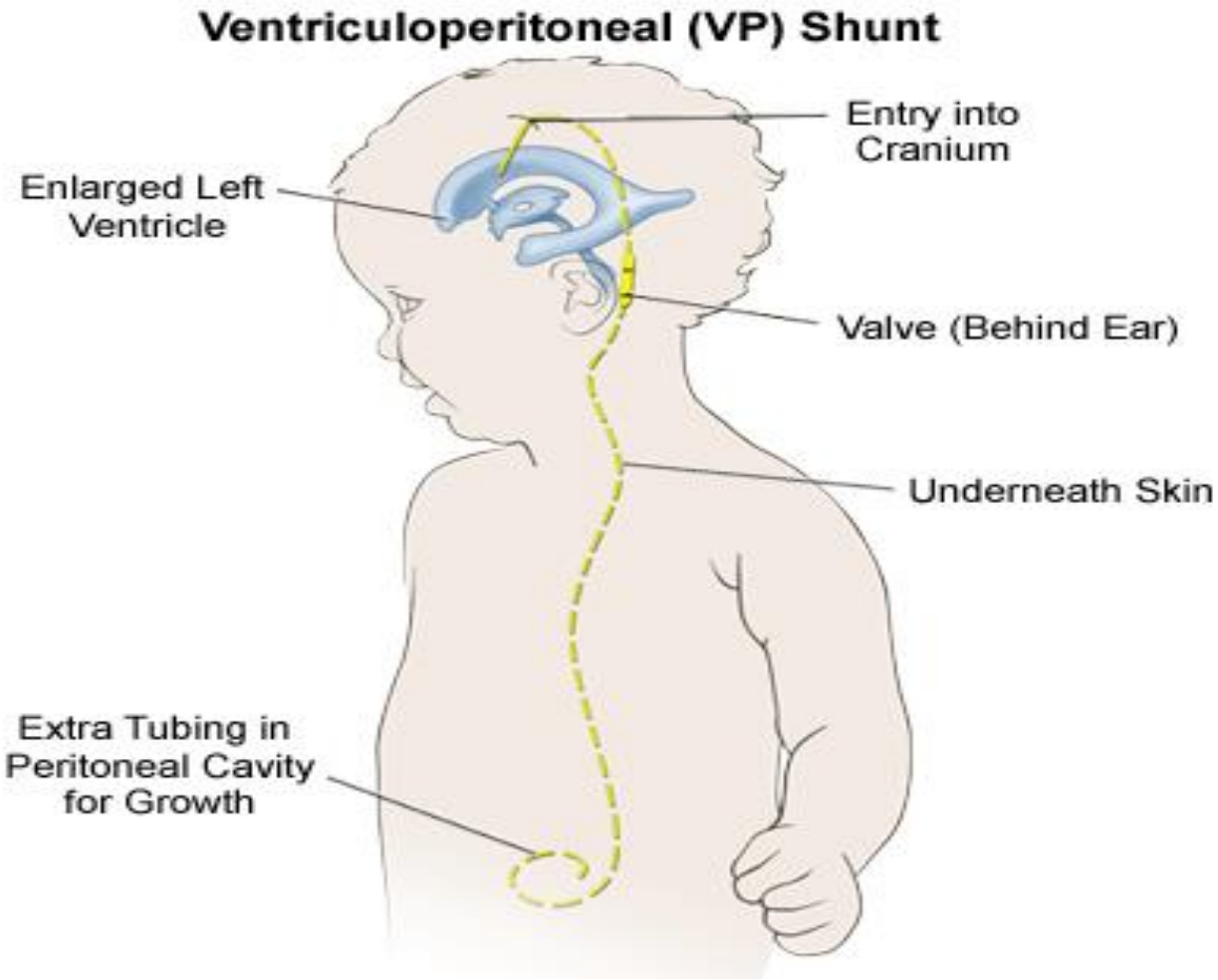
Surgical interventions

- Mass lesion removal (hematoma, abscess, secondary hydroceph.)
- CSF drainage:
- External ventricular drain
- Ventriculoperitoneal shunt
- Decompressive craniectomy

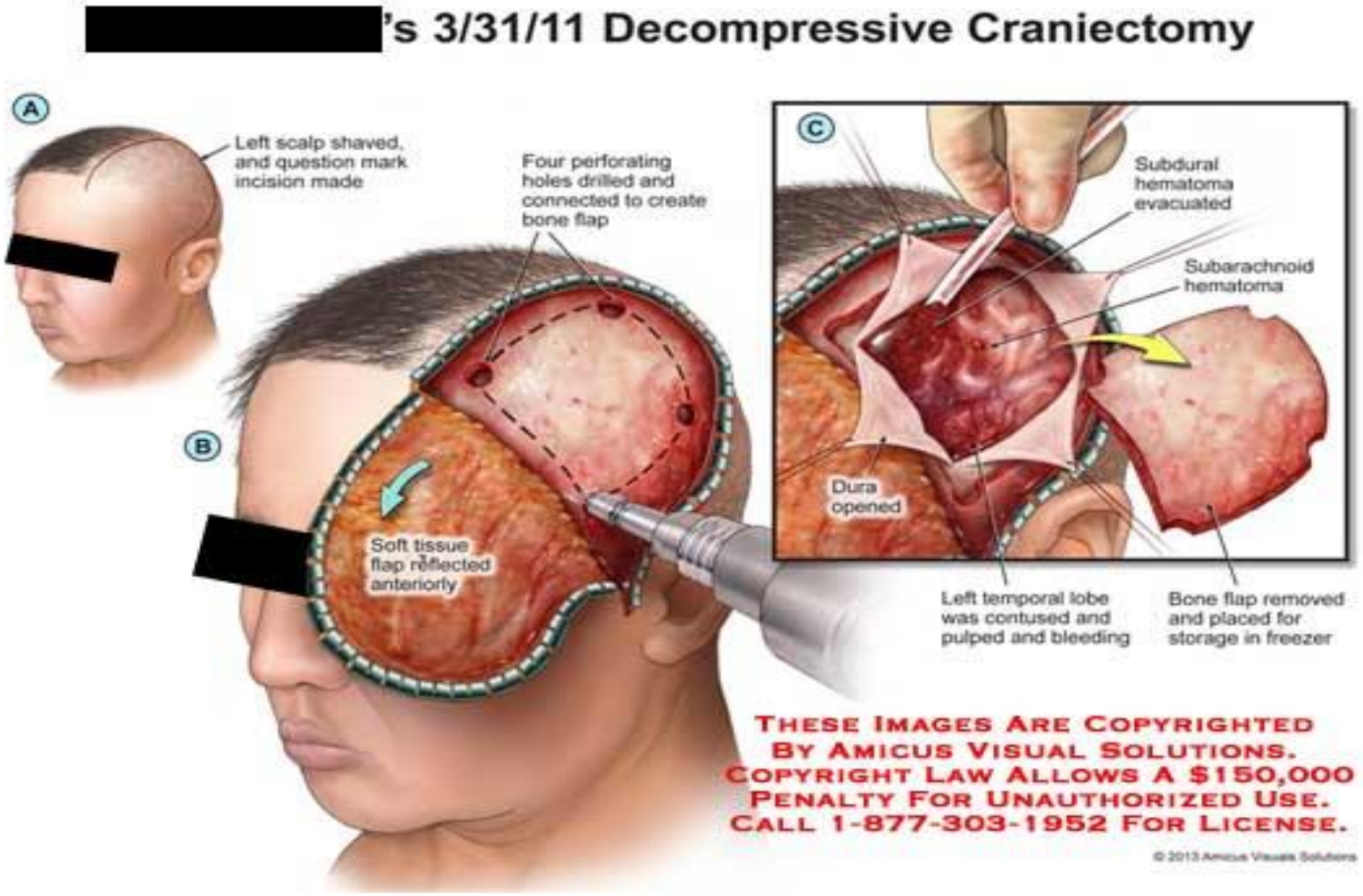
External ventricular drain



Ventriculoperitoneal shunt



Decompressive craniectomy



Idiopathic intracranial hypertension

- By definition, the term ‘IIH’ describes patients with isolated raised ICP—that is, not related to an intracranial disorder, a meningeal process or cerebral venous thrombosis.

Idiopathic intracranial hypertension

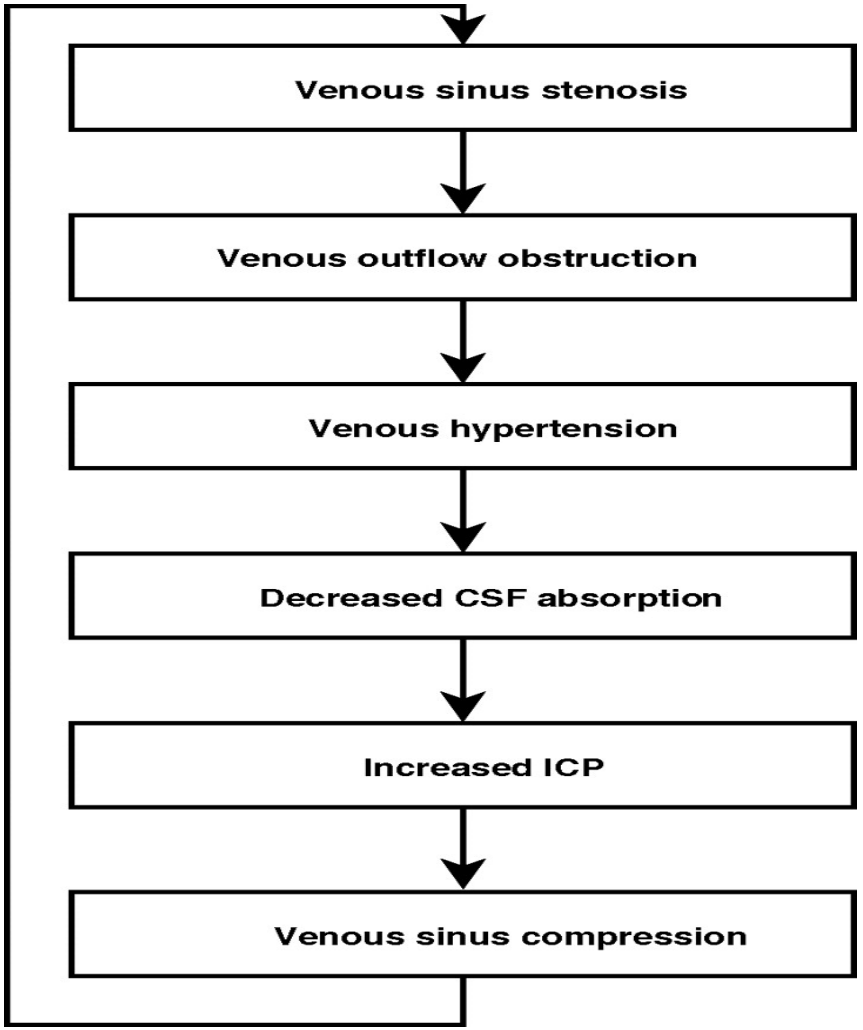
- Unknown aetiology.
- Typically affecting young obese women with menstrual irregularities or taking OCPs.
- produces a syndrome of increased intracranial pressure without identifiable cause.
- Various medications have been proposed to cause or, more likely, precipitate IIH (such as tetracycline , cyclosporine, lithium, nalidixic acid, nitrofurantoin, OCPs, levonorgestrel, danaxol and tamoxifen)

- **Modified Dandy criteria for the diagnosis of idiopathic intracranial hypertension:**

- ❑ Signs and symptoms of increased intracranial pressure (headaches, nausea, vomiting, transient visual obscurations, papilloedema).
- ❑ No localising focal neurological signs except unilateral or bilateral sixth nerve paresis.
- ❑ CSF opening pressure ≥25 cm H_2O* but without cytological or chemical abnormalities.
- ❑ Normal neuroimaging adequate to exclude cerebral venous thrombosis—that is, MRI of the brain, often with additional sequences (CT or MR venography).

- *The number of 25 cm of water is not an absolute cut-off, especially in children in whom CSF opening pressures as high as 28 cm of water have been documented to be normal.

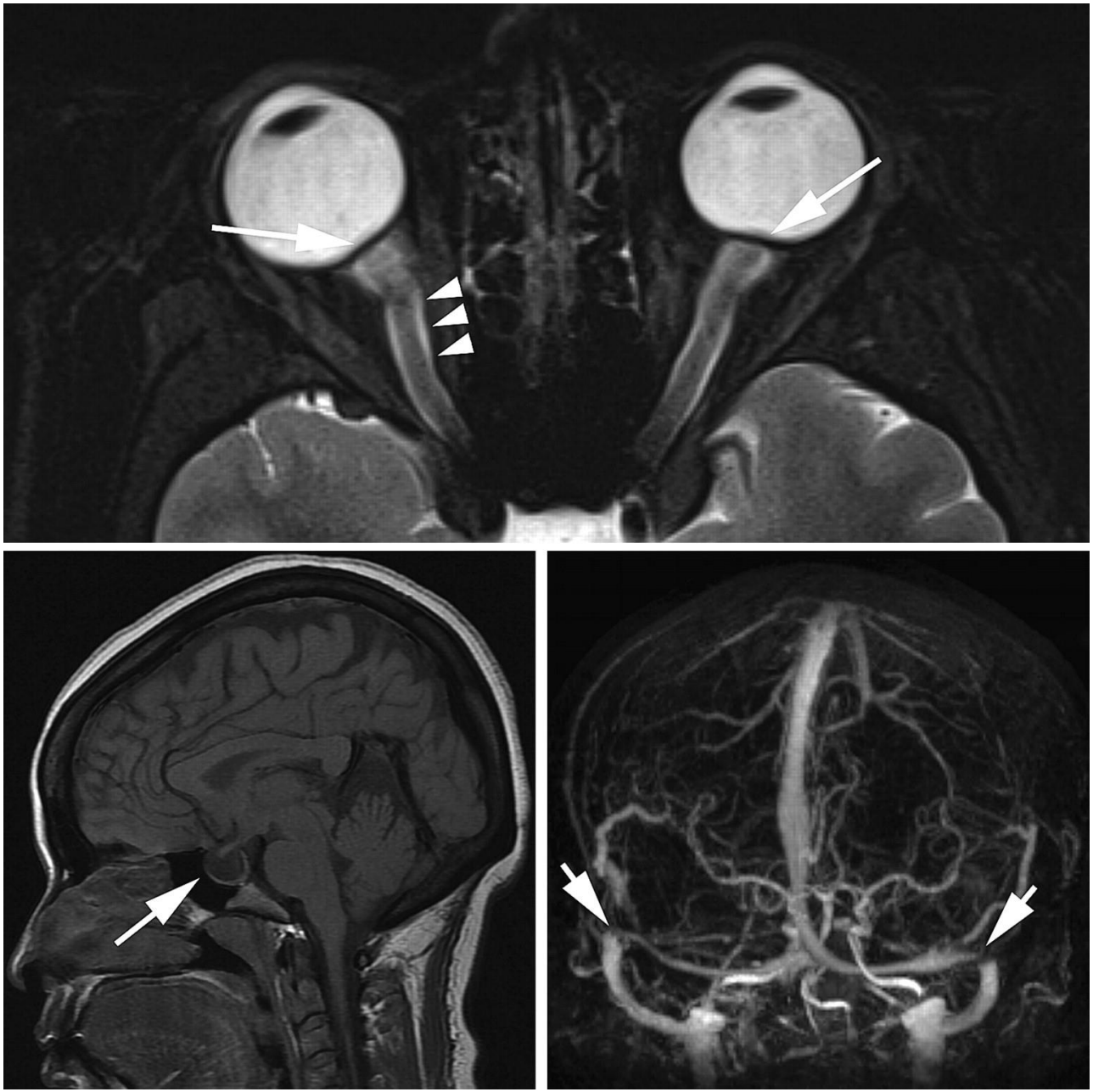
- patients who develop a syndrome of raised ICP triggered by certain medications or who are found to have cerebral transverse venous sinus stenosis (not thrombosis) are still conventionally classified as having ‘IIH’



Mechanism by which transverse sinus stenosis leads to increase intracranial pressure.

MRI findings in idiopathic intracranial hypertension.

Top: Posterior flattening of the globes resulting in shortening of the eyes and hyperopic shift (arrows). Dilation and tortuosity of the optic nerve sheath (arrowheads). Bottom left: Empty sella (arrow) Bottom right: Contrast-enhanced magnetic resonance venography showing bilateral distal transverse sinus stenosis (arrows).



pathophysiology

- The pathophysiological mechanisms remain unclear but those proposed classically include :

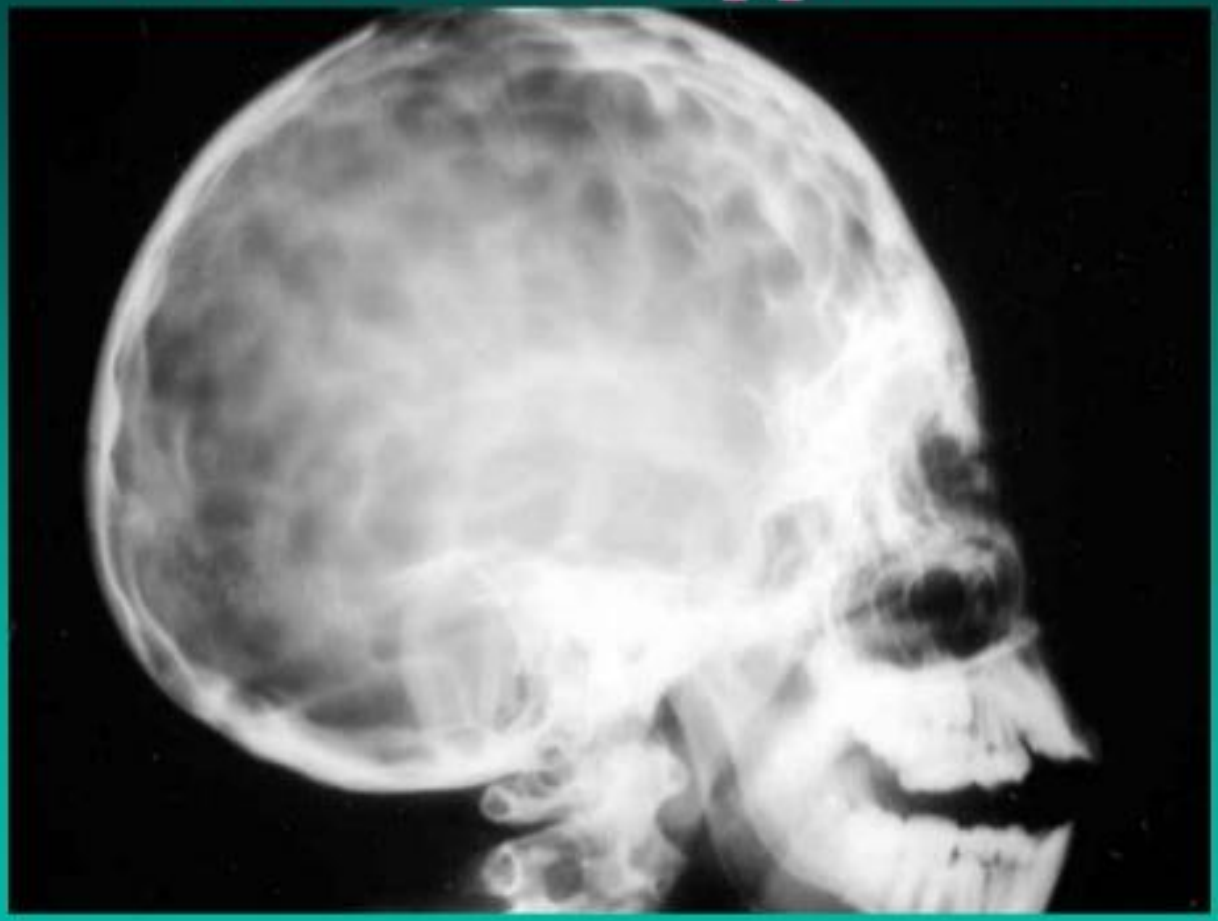
- increased brain water content.

- excess CSF production.

- reduced CSF absorption and increased cerebral venous pressure.

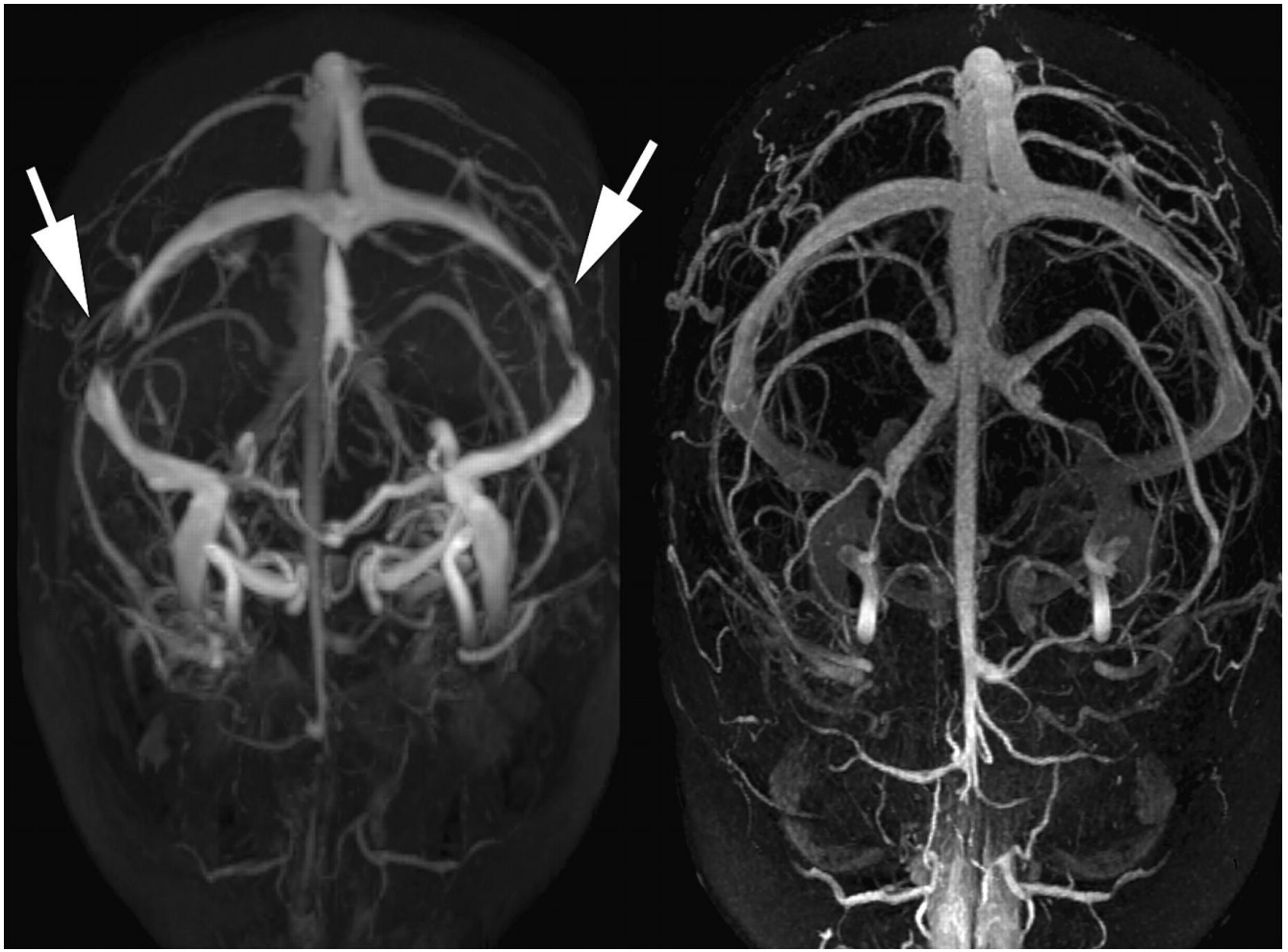
- Those with anomalous distal transverse sinuses resulting in bilateral transverse sinus stenosis (TSS)) are predisposed to developing raised ICP which might be triggered by specific events or situations, such as weight gain, endocrine changes, hypercoagulable states, specific medications and OSA

Silver Beaten Appearance



Resolution of bilateral transverse sinus stenosis after lumbo-peritoneal shunt in a young obese woman with idiopathic intracranial hypertension.

Resolution of bilateral transverse sinus stenosis after lumbo-peritoneal shunt in a young obese woman with idiopathic intracranial hypertension.



- **Factors independently associated with a worse visual outcome in idiopathic intracranial hypertension**

- Male gender

- Race (Black patients)

- Morbid obesity

- Anaemia

- Obstructive sleep apnoea

- Acute onset of symptoms and signs of raised intracranial pressure (fulminant idiopathic intracranial hypertension)

Treatment of IIH

- 1. lumber puncture:
- it is not uncommon to observe a lasting clinical remission following a single lumbar puncture in some IIH patients, obviating the need for further medical or surgical treatment.

- 2.weight loss:

➢ weight loss effectively reduced not only headaches and papilloedema, but also ICP.

➢ Weight loss is often not an effective short term treatment and thus usually must be initiated in association with other treatments

- # 3.Medical treatment:
- **Carbonic anhydrase inhibitors,** such as acetazolamide ,are the main medical treatment classically prescribed for IIH. Acetazolamide decreases the production of CSF in humans .

- Topiramate (which has weak carbonic anhydrase inhibition properties) has also been suggested for the treatment of IIH, particularly for the treatment of headaches.

- Oral steroids have been used as a treatment for IIH in the past but are associated with significant long term side effects, such as weight gain. High dose intravenous steroids are still occasionally used in patients with rapidly progressive visual loss from fulminant IIH while a more definitive treatment is organised.

- **Surgery:**

- is required in patients with a fulminant onset of disease or when other treatments have failed to prevent progressive visual loss.

- More rarely, surgery may be performed for refractory headaches related to chronically elevated ICP.

- The choice of procedure depends on local resources, as well as the patient's symptoms and signs.

- In patients with papilloedema who have severe visual loss, but minimal or no headache, optic nerve sheath fenestration (ONSF) is often advised, while in those with visual loss, papilloedema and headache, a CSF diversion procedure, such as ventriculo-peritoneal (VP) or lumbo-peritoneal (LP) shunting, is preferred.

- Aggressive management with CSF shunting is usually required to prevent catastrophic visual loss in those with acute and rapidly progressive visual loss.

- These patients might benefit from a transient lumbar drain while awaiting a more definite surgical procedure.

- It is now well accepted that stenting of transverse venous sinus stenosis reduces cerebral venous pressure, reduces ICP and improves symptoms and signs in selected IIH patients.

- Endovascular venous sinus stenting can result in serious complications, such as:
  - stent migration.
  - venous sinus perforation, in-stent thrombosis,
  - subdural haemorrhage and the development of recurrent stenosis immediately proximal to the stent.

- This procedure should be limited to selected patients with bilateral TSS or with a hypoplastic transverse sinus on one side and TSS on the other, and refractory symptoms and signs of increased ICP, who cannot undergo (or have failed) more conventional surgical treatments.

THANK YOU