Department of Surgery
Neurosurgery Teaching Unit

INTRACRANIAL PRESSURE AND BRAIN CIRCULATION. INTRACRANIAL HYPERTENSION. ADULT-ONSET HYDROCEPHALUS.

34484 Pathology of the nervous system

Neurosurgery

Topic 15

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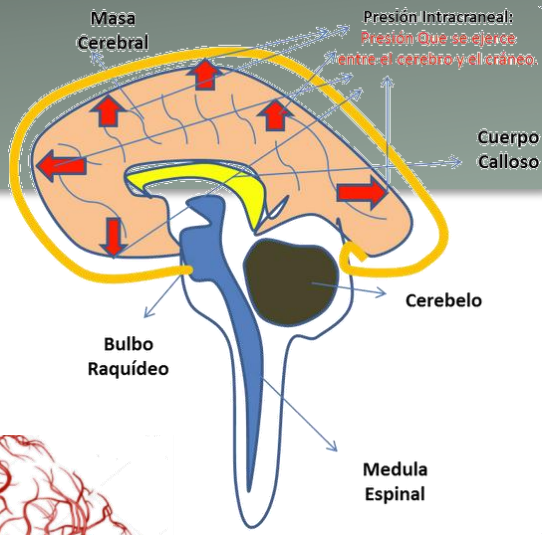
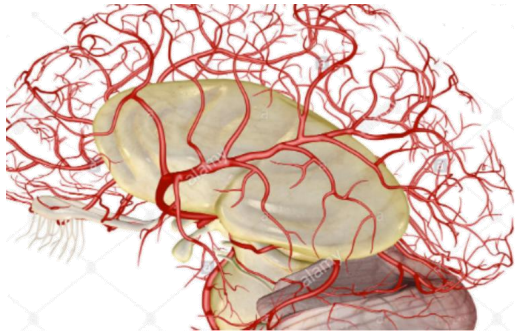
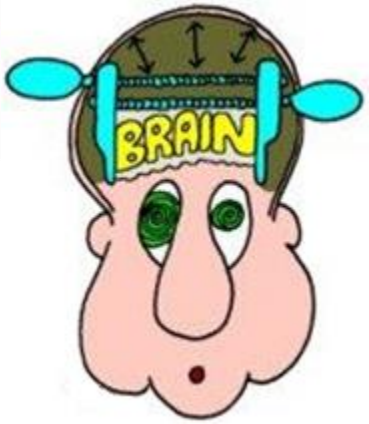
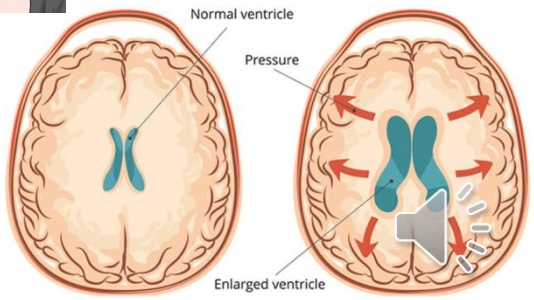
Key points



- **Intracranial pressure (ICP)**
  - Definition
  - Monro-Kellie doctrine
  - Factors
  - Measures
  - Cerebral hernias
- **Cerebral circulation**
- **Intracranial hypertension (ICHT)**
  - Diagnosis
  - Treatment
- **Benign intracranial hypertension**
- **Chronic adult-onset hydrocephalus**

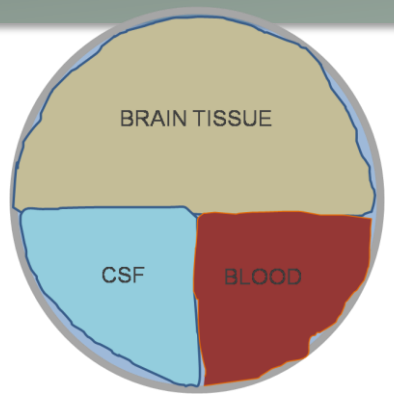


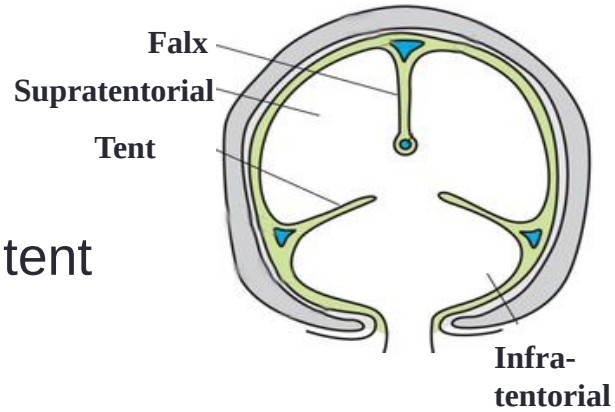


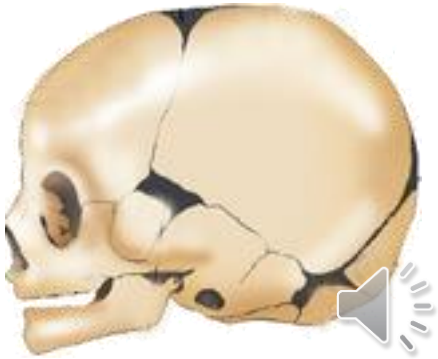


Cranial cavity: continent

- **Adult skull = bone shield**
  - Protects the brain → the intracranial space is CONSTANT
  - **$V_{BT} + V_{CSF} + V_{BL} = k$** (Monro-Kellie doctrine)
  - If one component ↑ another must ↓
  - If compensation is exceeded → **↑ICP (ICHT)**
- **Intracranial space ≠ single**
  - Partitions → possibility of displacement of content from one space to another → *Brain hernia*
- **Infant skull**
  - Sutures + fontanelles allow ↑ cranial cavity capacity ▸ Larger compensation of ↑ICR

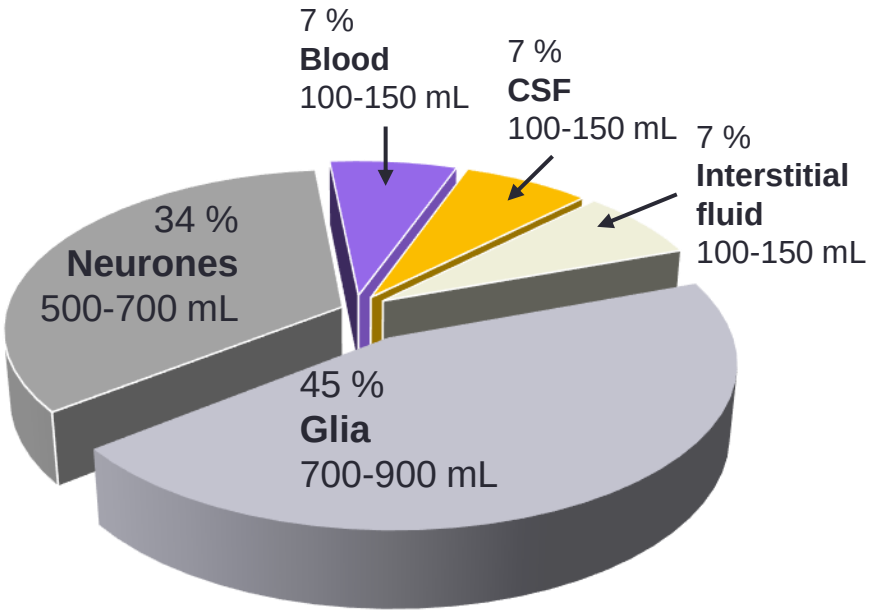


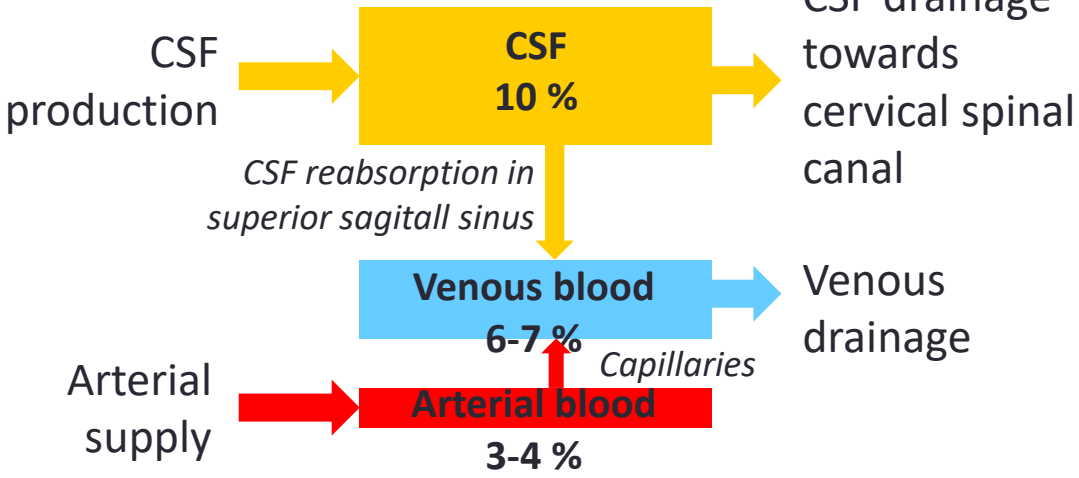




Content

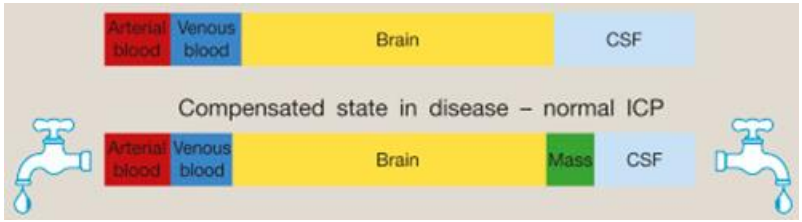
Brain tissue
80 %





Monro-Kellie doctrine

- Established conditions:
  - Skull is a non-expandable bone case
  - Brain tissue ≅ non-compressible
    - *May atrophy in weeks/months*
  - Intracranial blood volume ≅ constant
    - *Systole blood volume that enters = exits*
- **Volume of brain tissue + blood + CSF = constant**
  - If one component ↑ another must ↓
  - If compensation is exceeded → **↑ICP**

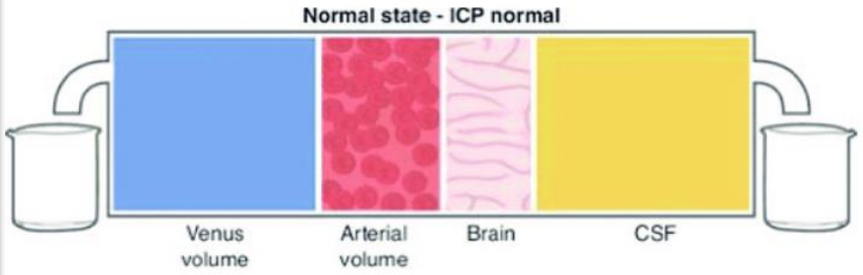


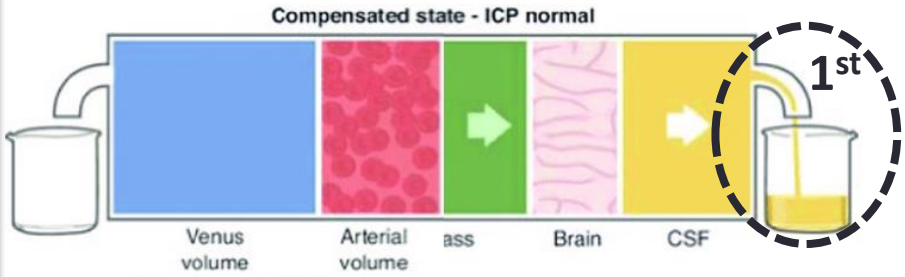
"The sum of volumes of brain, CSF, and intracranial blood is constant.
An increase in one should cause a decrease in one or both of the remaining two".

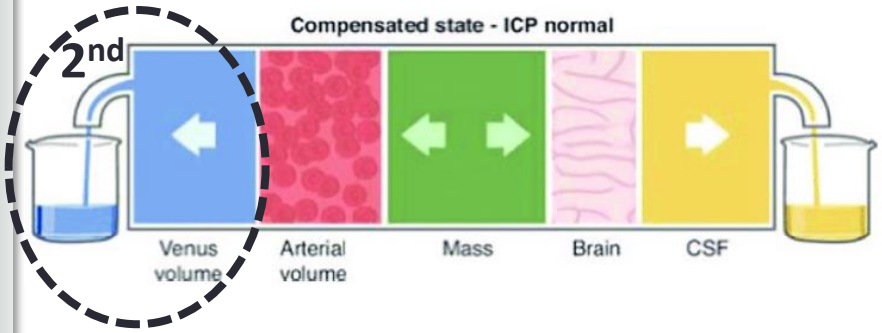
$$V_{BT} + V_{CSF} + V_{BL} = k$$

Change order in intracranial volume ↑

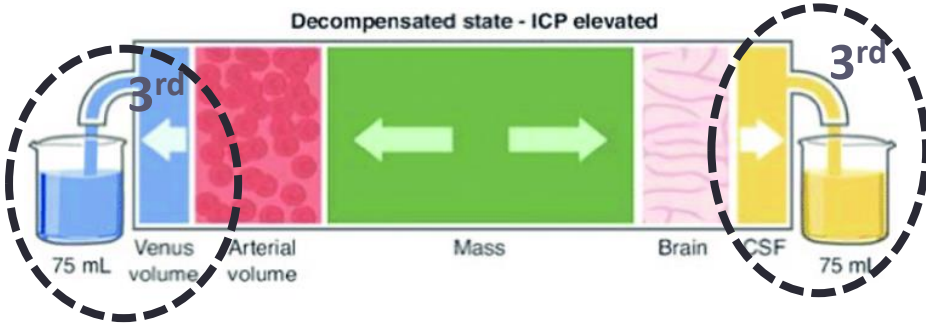
Compensated status

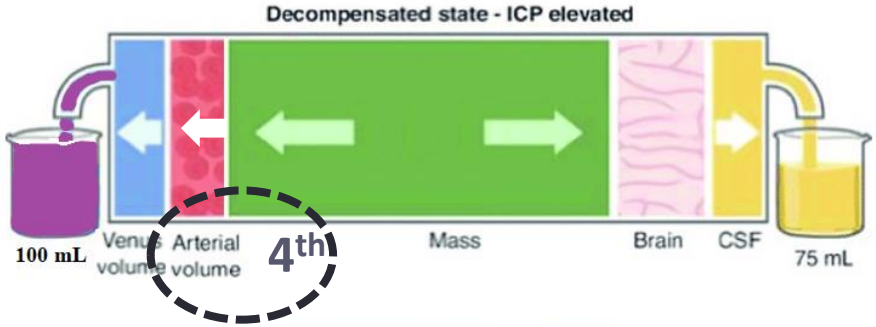


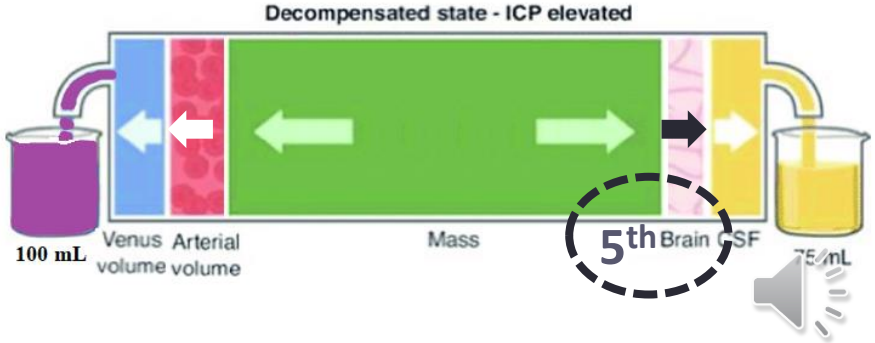




Decompensated status

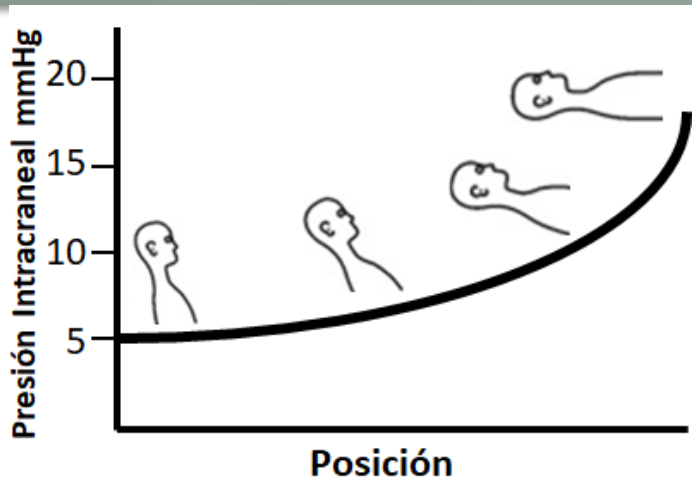






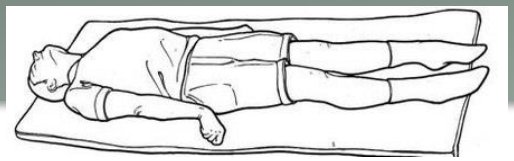
NORMAL ICP

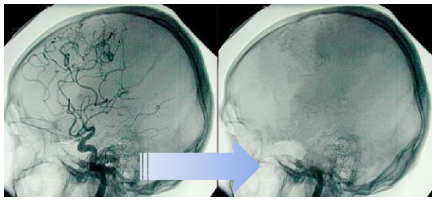
- **It depends on age and position:**
  - ICP ↓ standing and sitting
  - ICP ↑ in supine

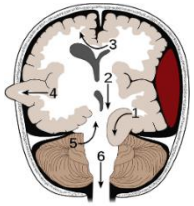


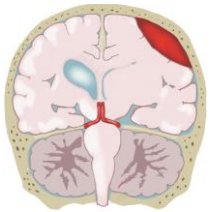
Age group	Normal range (mmHg)
Adults and older children	< 10-15
Young children	3-7
Neonates	1.5-6

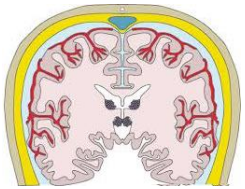
Curve volume / ICP











Intracranial pressure

50
40
30
20
10
0

Brain death

Vascular collapse

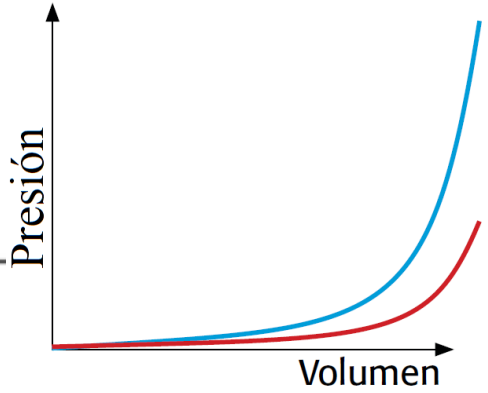
Brain herniation

Decompensation

Compensation

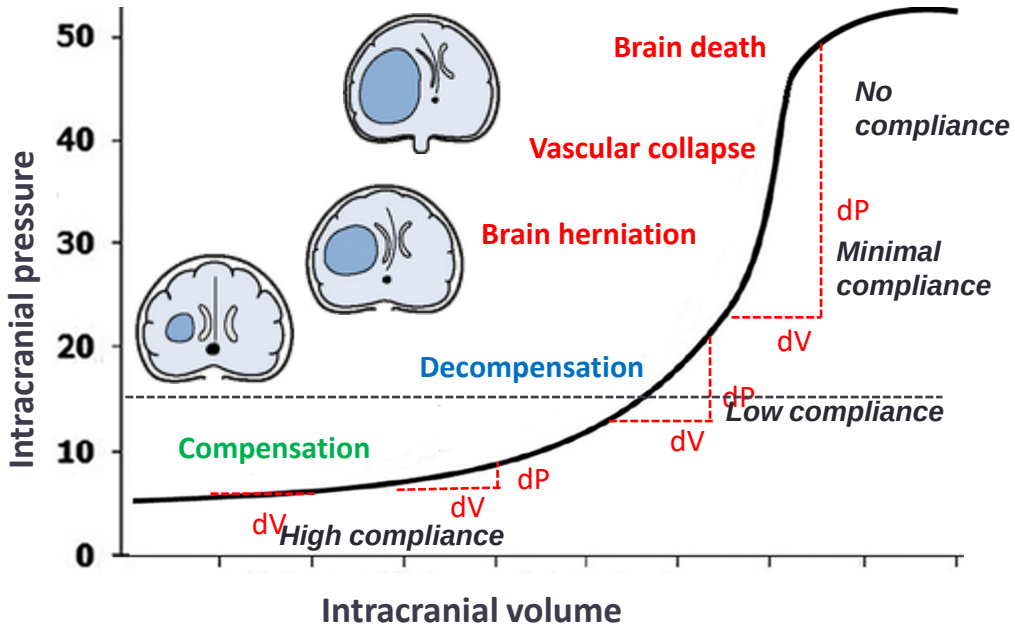
dP
dV
dP
dV
dP
dV

Intracranial volume



Intracranial space compliance

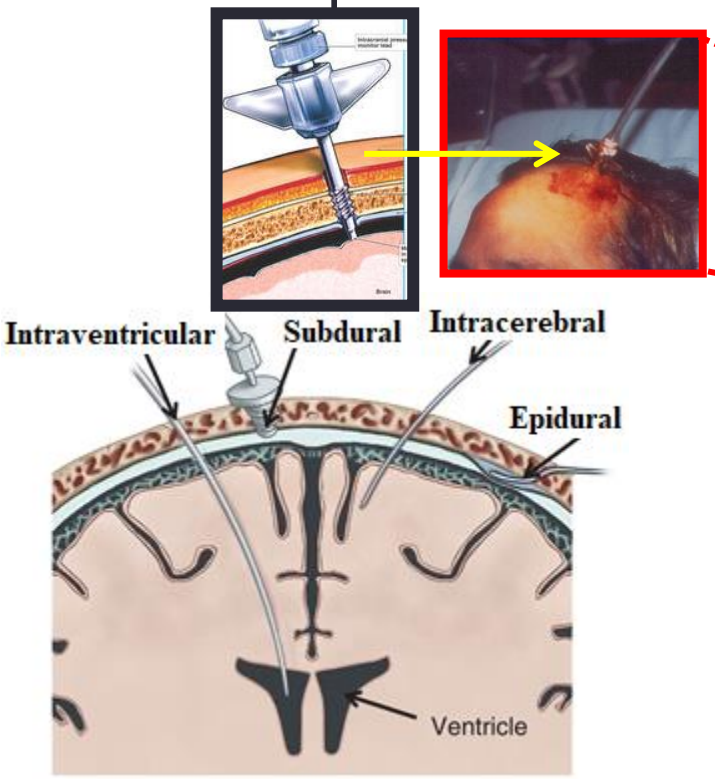
- **Compliance** = capacity of change in intracranial volume with no change in ICP

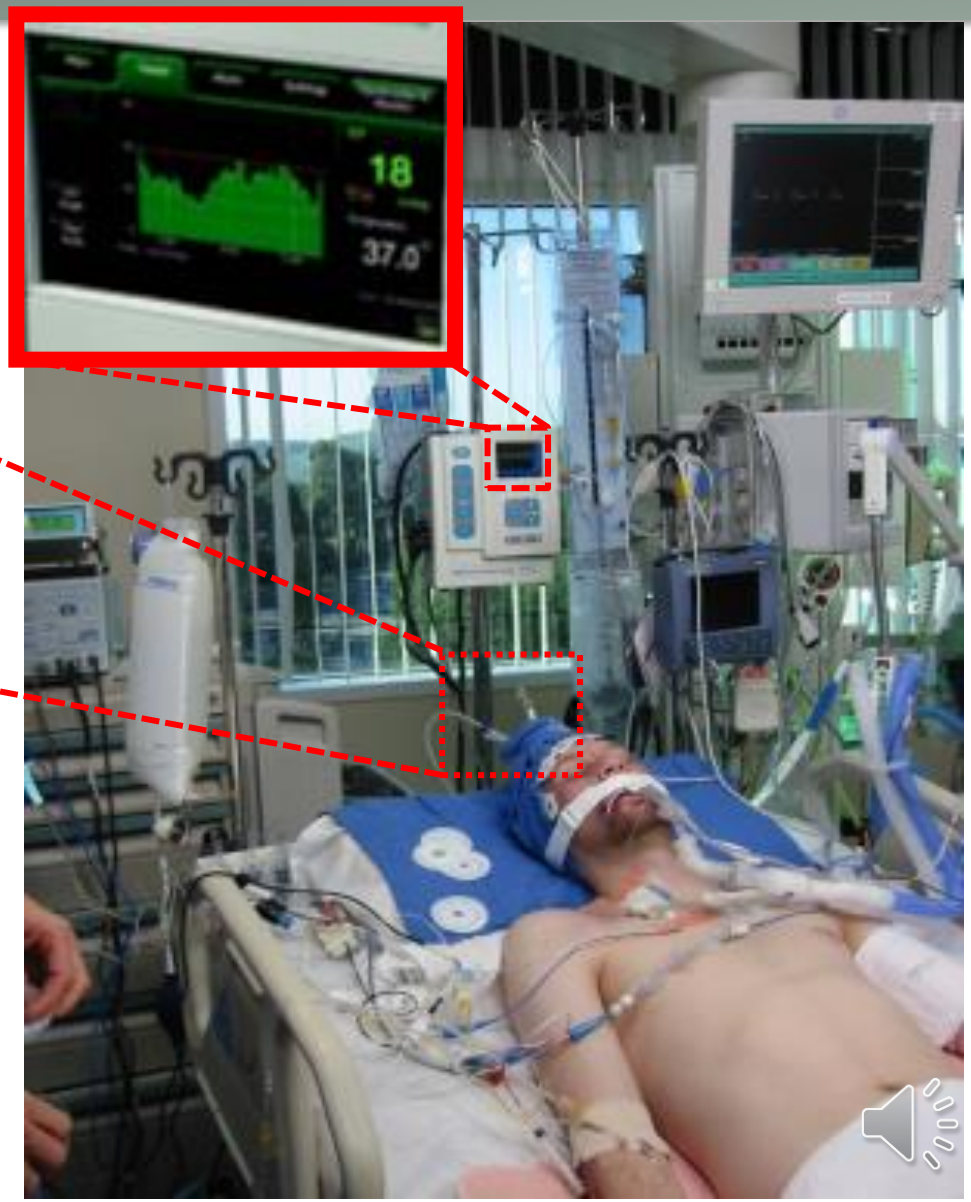


- ↑ **Compliance** = ↑ capacity of compensation of ↑intracranial volume
- ↓ **Compliance** = risk of decompensation with vascular collapse and brain death

ICP measurement

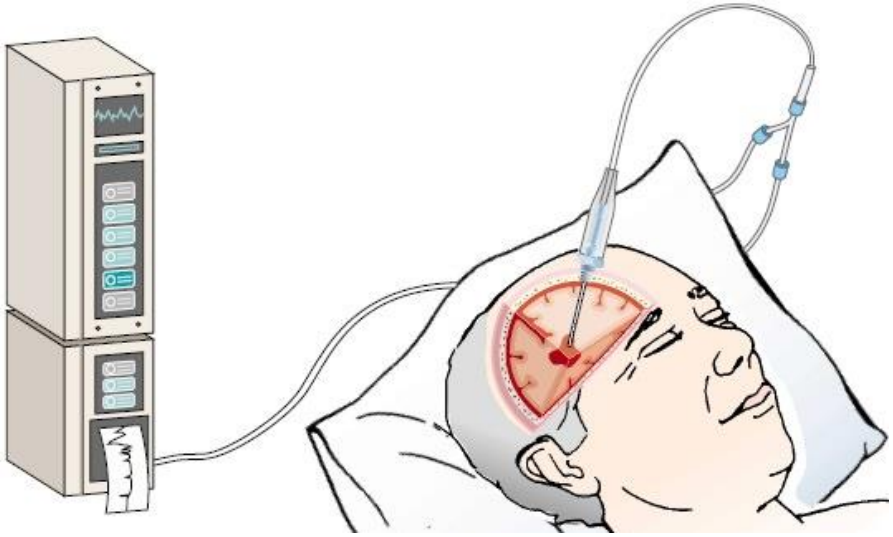
- Invasive
- Accurate and reliable
- Necessary to adapt the treatment to patient ICP



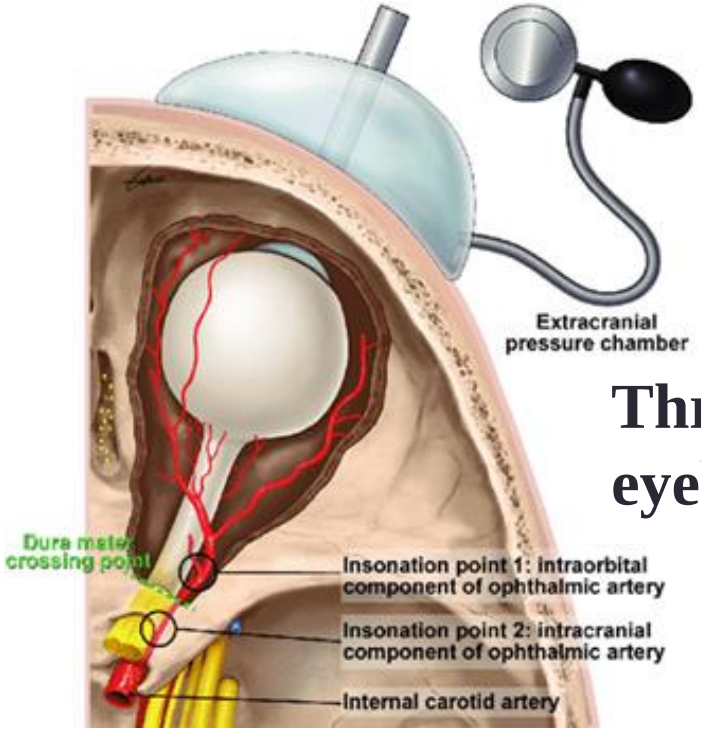


ICP measurement methods

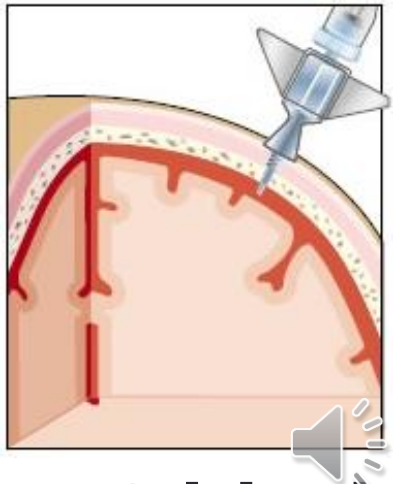
- Invasive
  - Subdural
  - Intraventricular
- Non-invasive



Intraventricular

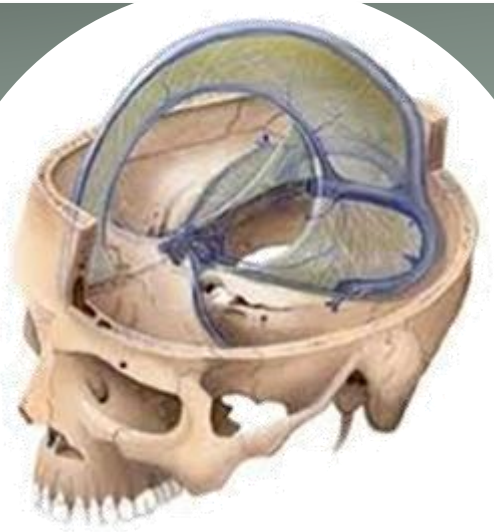


Through the eyeball

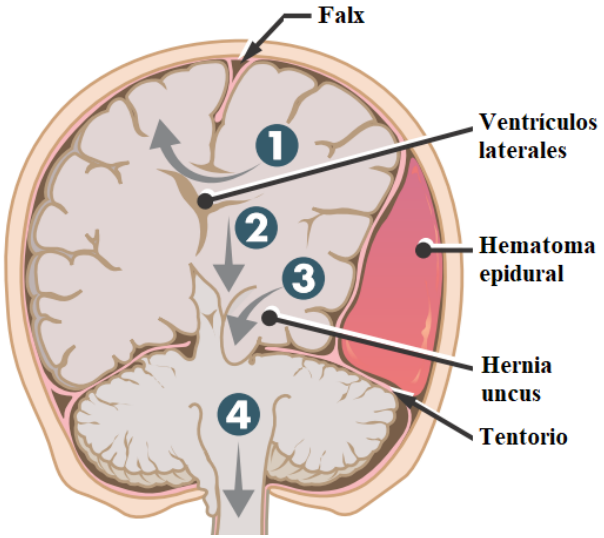


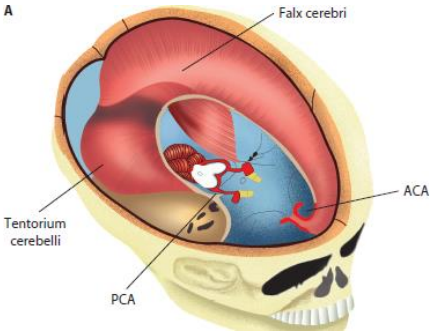
Subdural

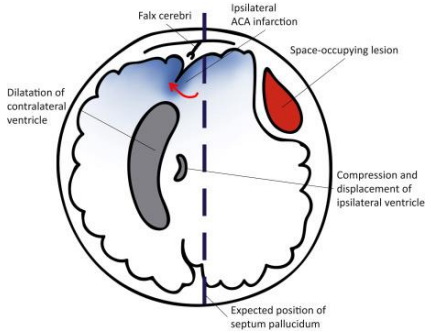
Brain herniation



- **Stiff partitions**: falx and tent
- ↑ ICP in one compartment ⇒ displacement of brain tissue
  - **<u>Brain hernias</u>**
  - Compression against the stiff borders of falx / tent → vascular occlusion
  - Brain tissue ischaemia→ neurological deficits + brain edema

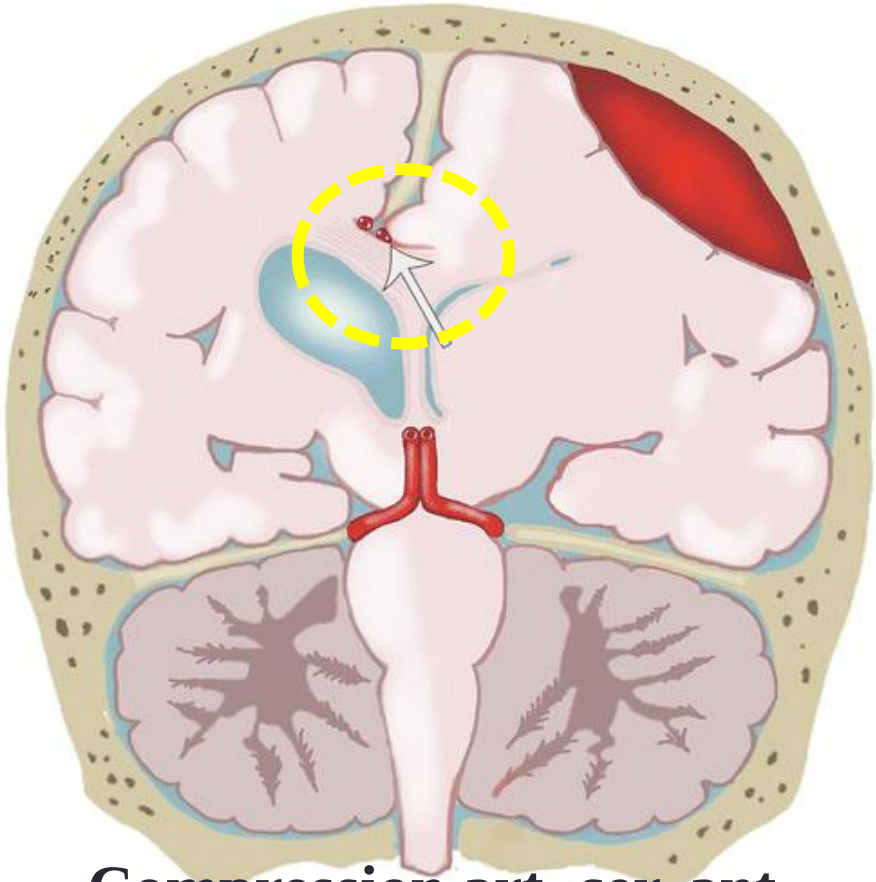




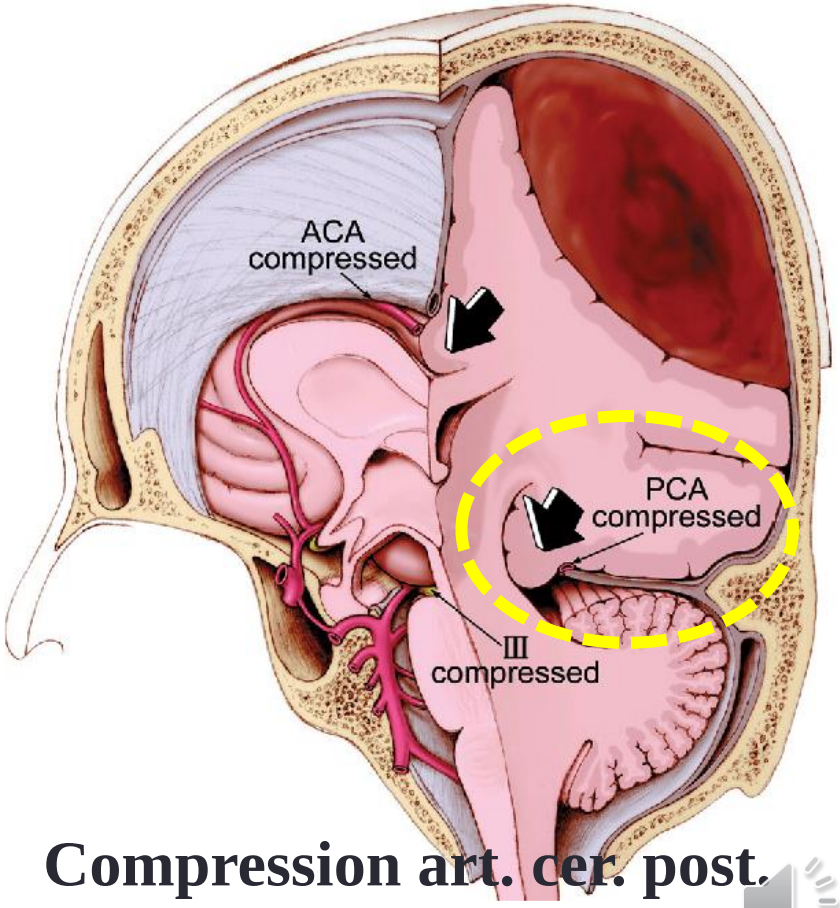


Brain herniations = compression of arteries

- Compression of arteries = ischemia nerve tissue = more cerebral edema

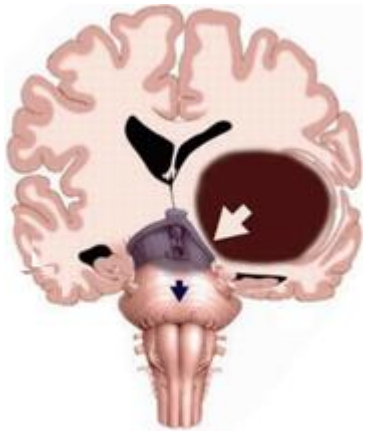


Compression art. cer. ant. by subfalcine herniation

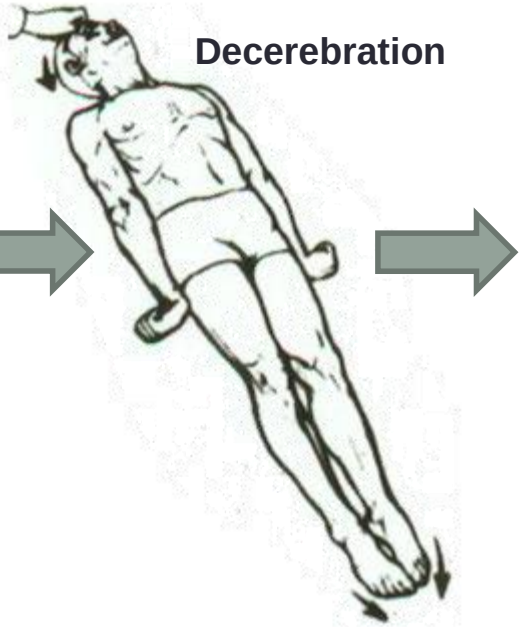


Compression art. cer. post. by hernia of the uncus

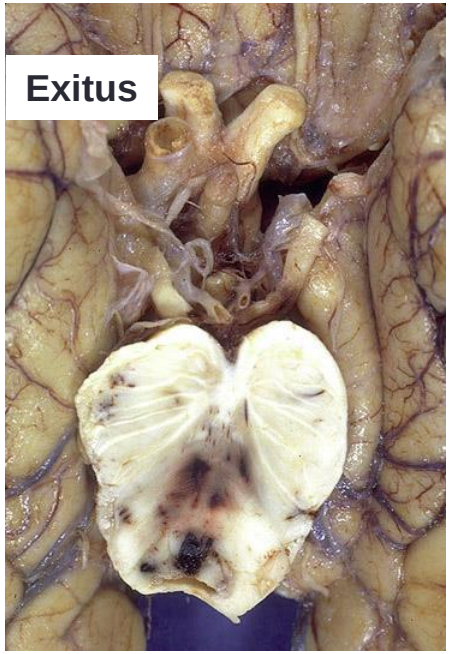
Uncal hernia



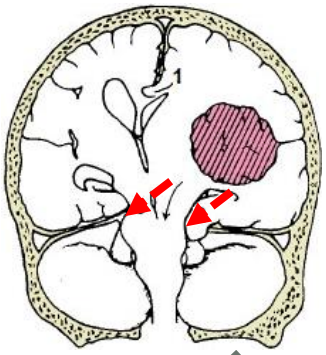
Brainstem compression

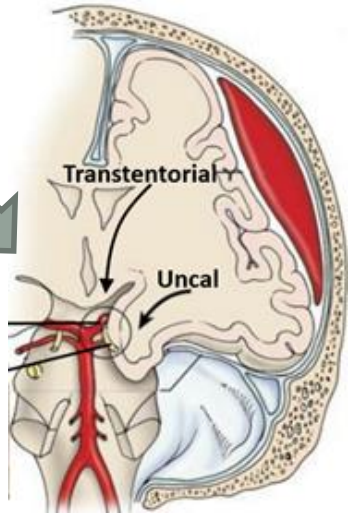


Decerebration



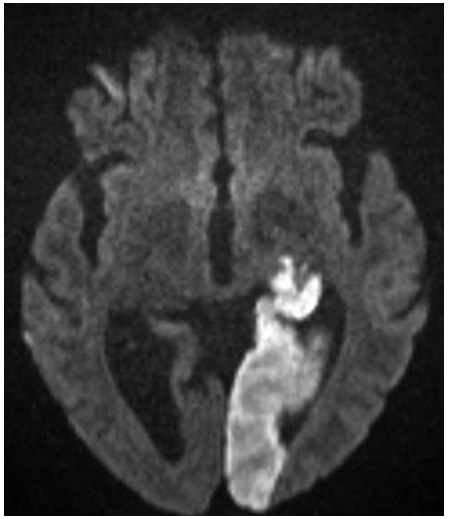
Exitus



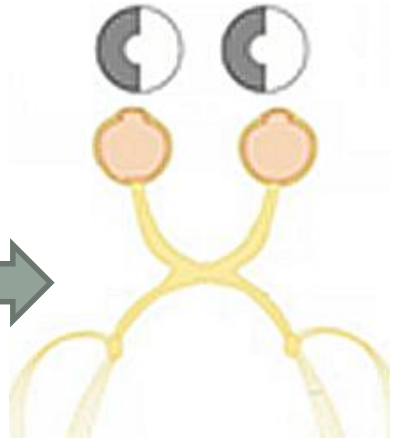


Posterior cerebral artery

Occlusion post. cerebral artery



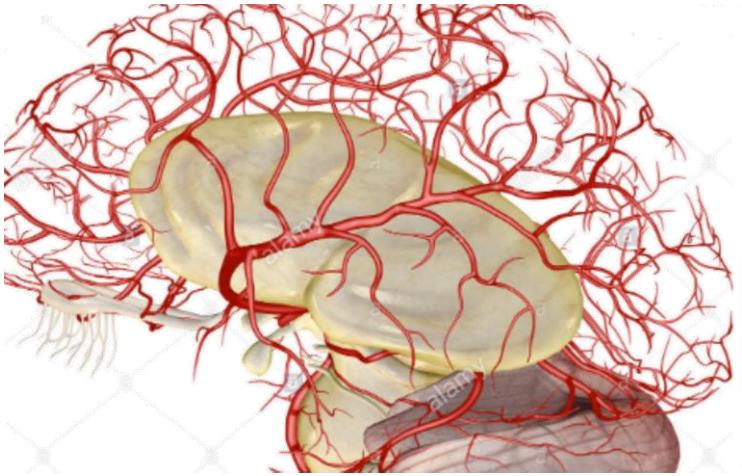
Calcarine fissure infarction



Contralateral homonymous hemianopsia

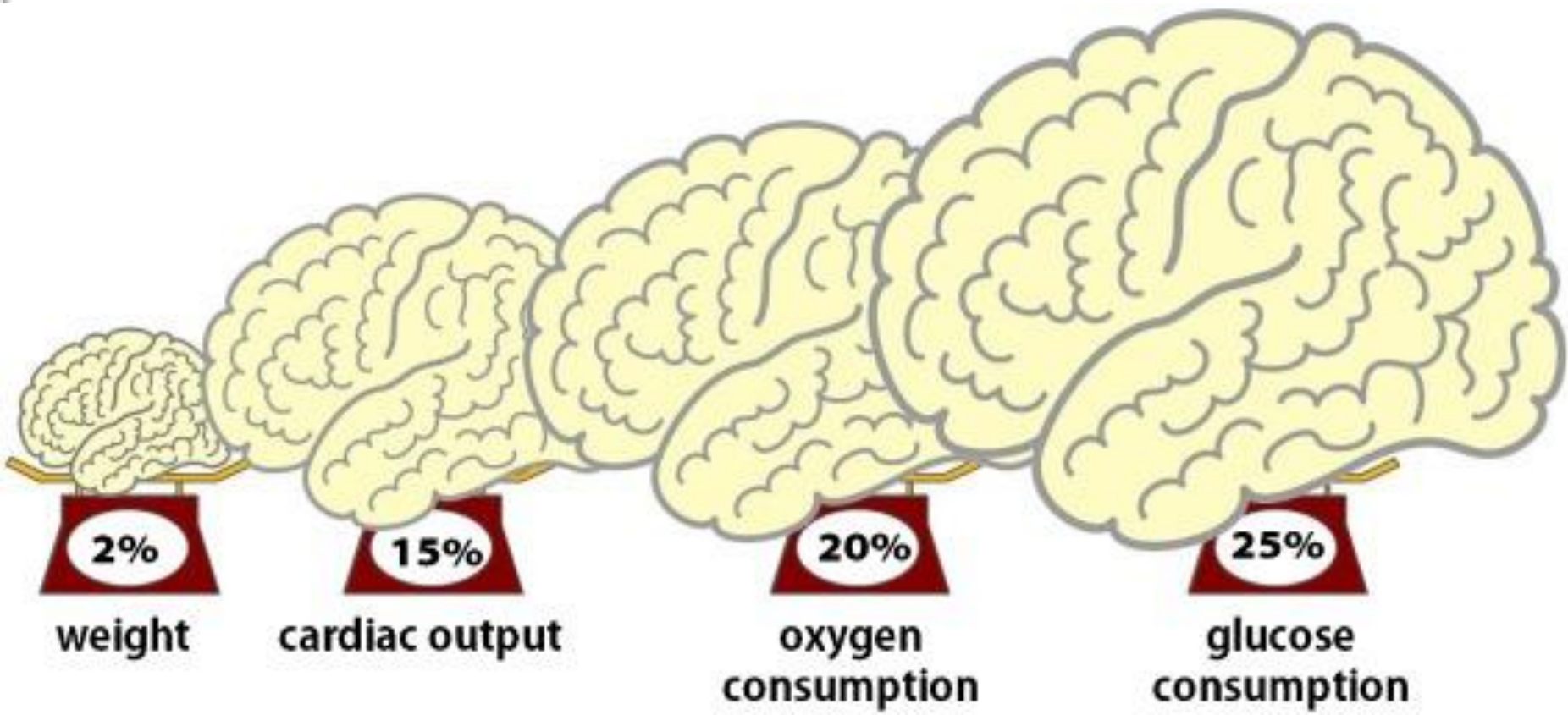
CEREBRAL CIRCULATION

- Brain tissue has NO energy storage = **needs constant blood flow**
- **Cerebral blood flow (CBF)** = blood supply to the brain in a given moment
- **Normal CBF =** 45 – 60 mL /100g /min
  - Grey matter 75 – 80 mL /100g /min
  - White matter 20 – 30 mL /100g /min
- **Hyperaemia:** CBF > 60 mL /100g /min
  - CBF > tissue demands = possible ↑ ICP
- **Ischaemia:** CBF < 20 mL /100g /min



16-18 ml/100g/min	Flat EEG
15 ml/100g/min	Reversible neuronal function abolition
12 ml/100g/min	Auditory evoked potentials abnormalities
≤ 10 ml/100 g/min	Cell death, stroke, brain death

Brain consumption



***(CMRO** = cerebral metabolic rate of oxygen)*

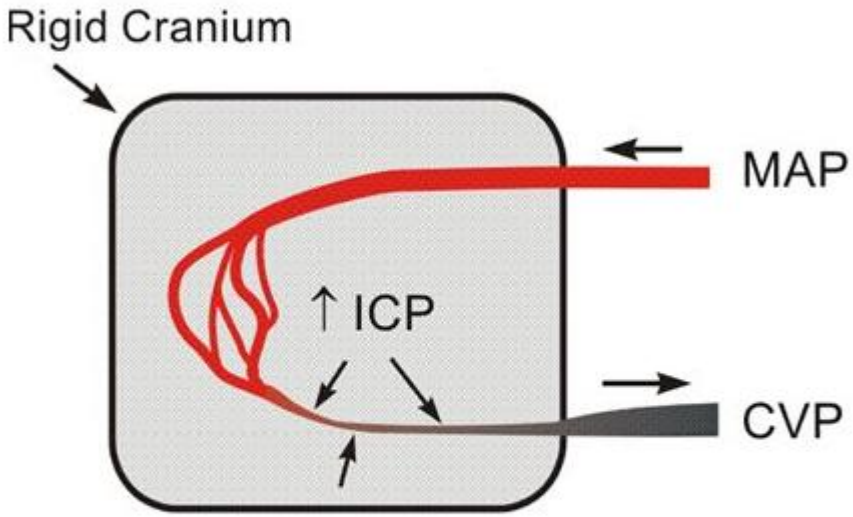
Cerebral blood flow (CBF) *vs* cerebral perfusion pressure (CPP)

- **Cerebral perfusion pressure (CPP)** = Pressure of the CBF that maintains brain metabolism
  - Difference between mean arterial pressure (MAP) and ICP

    CPP = MAP – ICP
  - Normal CPP = 60 – 70 mm Hg

➢ ***When ICP ⇑, MAP must increase to reach a CPP that guarantees blood flow to the brain***

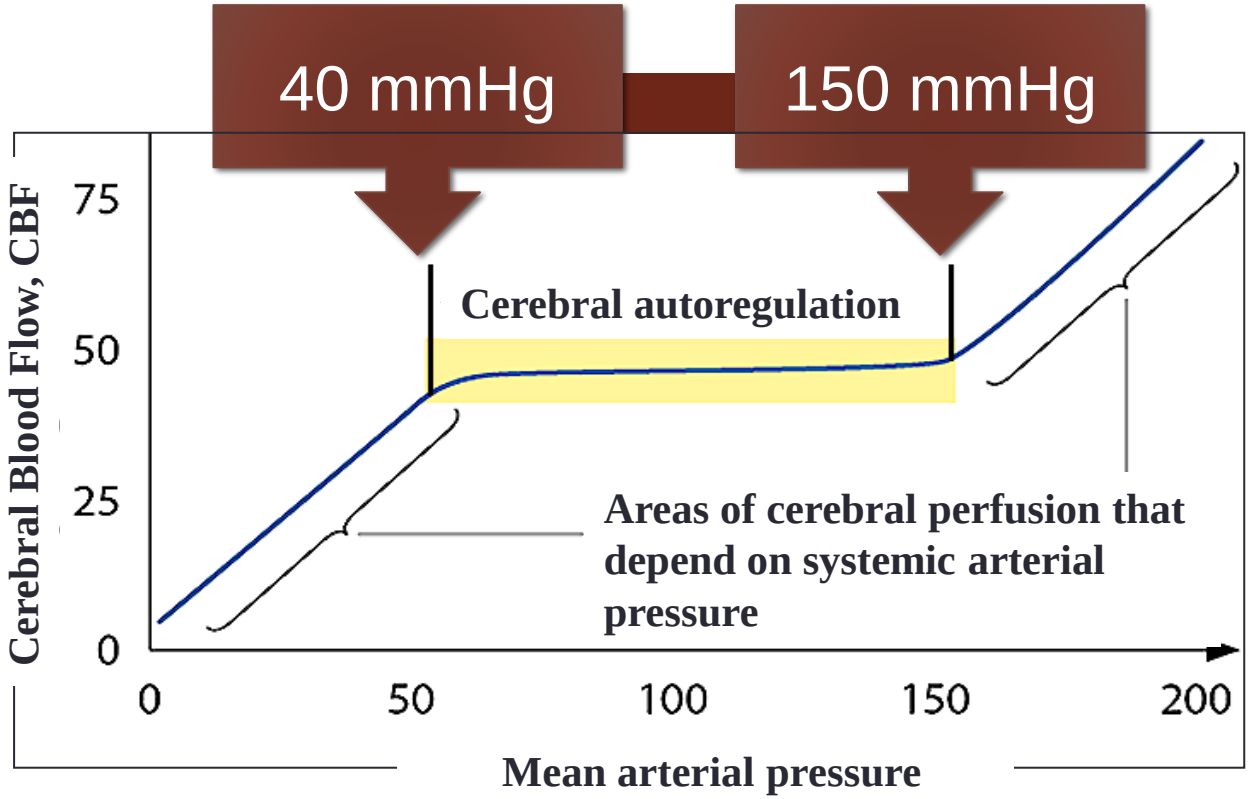
CPP = MAP – ICP



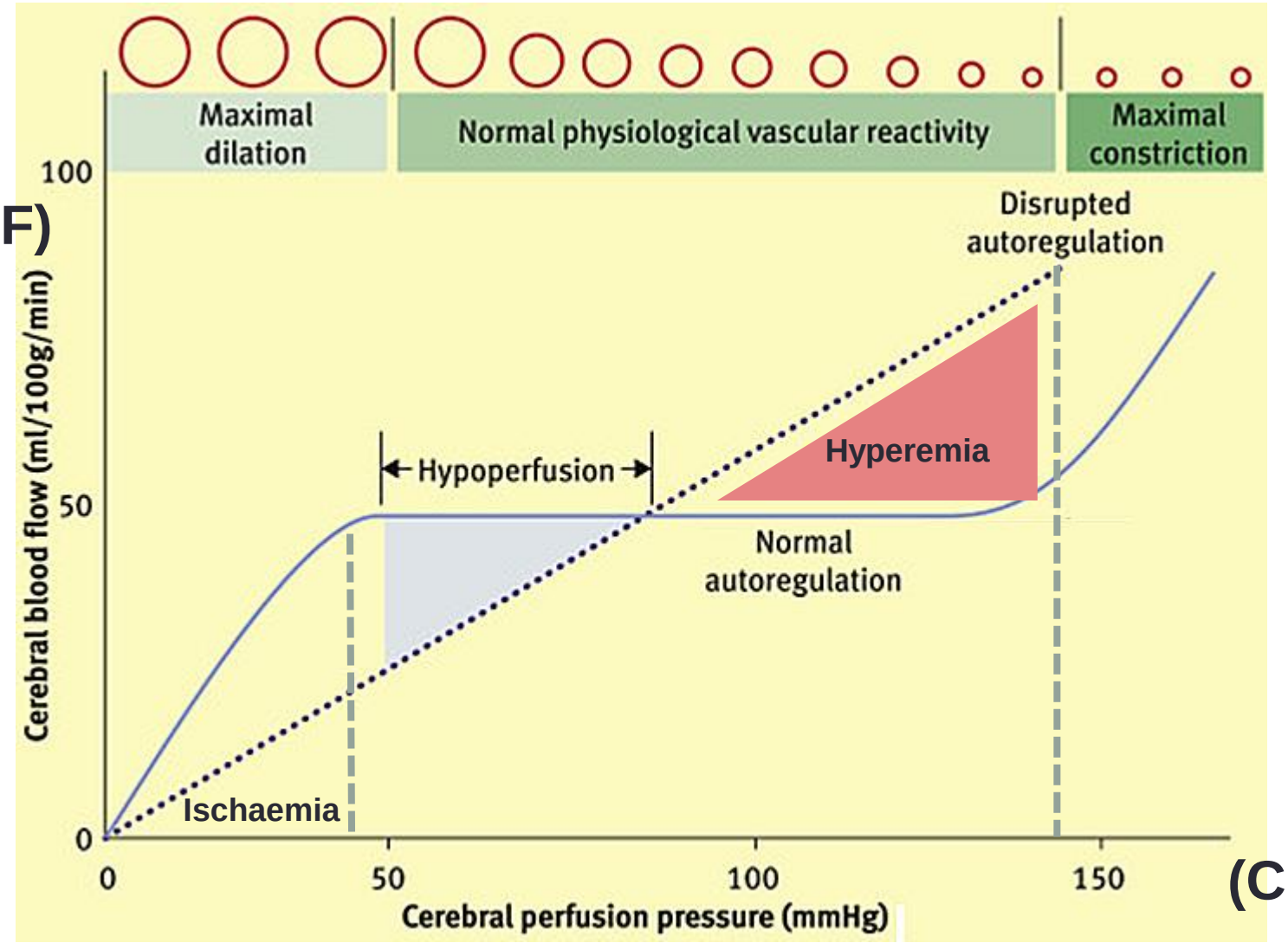
CPP = cerebral perfusion pressure
MAP = mean arterial pressure
ICP = intracranial pressure **(normally 0-10 mmHg)**
CVP = central venous pressure

Cerebral autoregulation

- CBF autoregulates within certain limits of systemic MAP

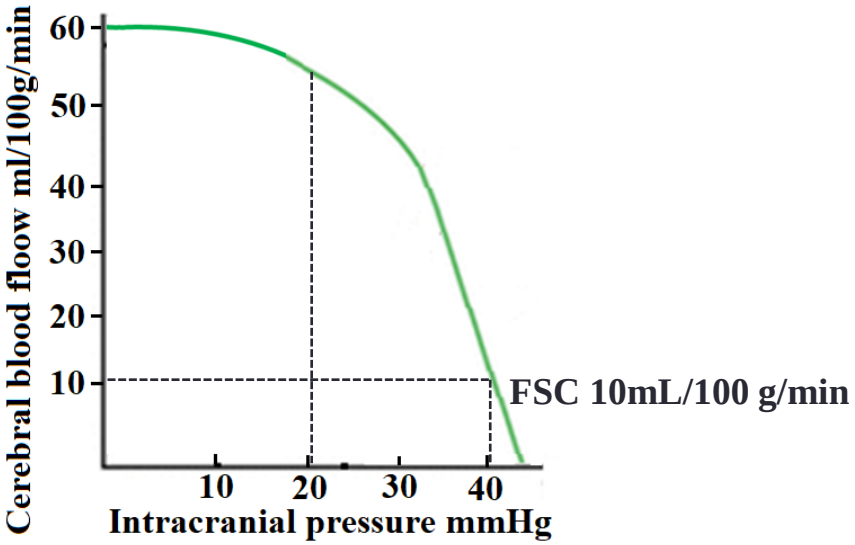


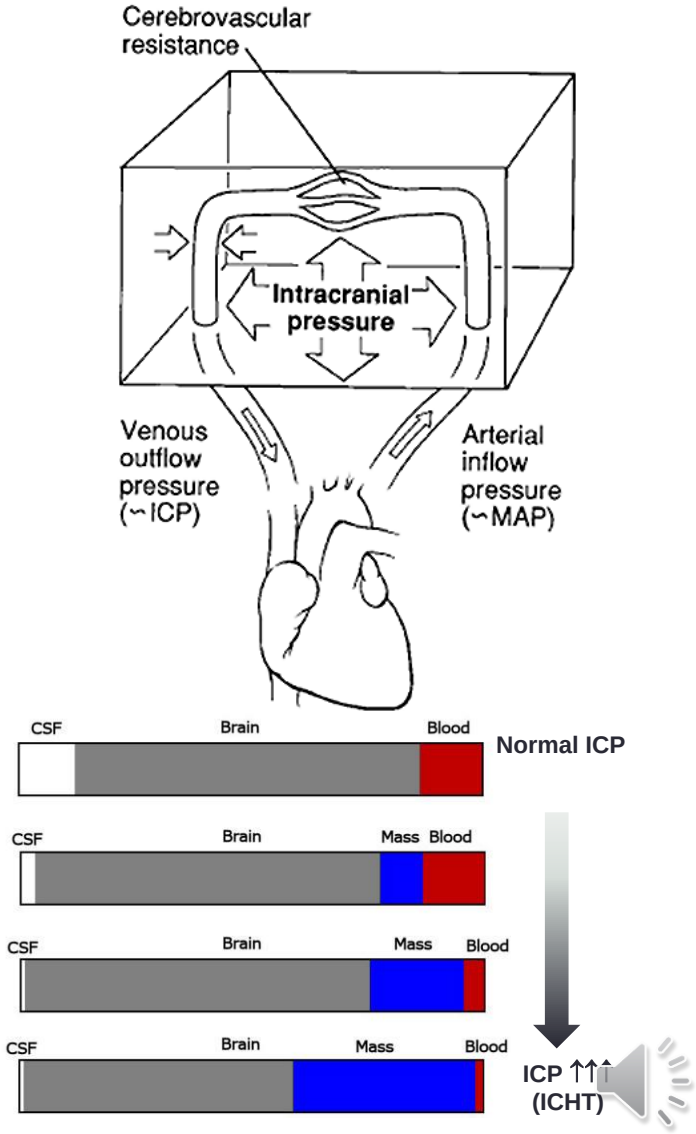
Cerebral blood flow (CBF) *vs* cerebral perfusion pressure (CPP)



Cerebral blood flow (CBF) and intracranial pressure (ICP)

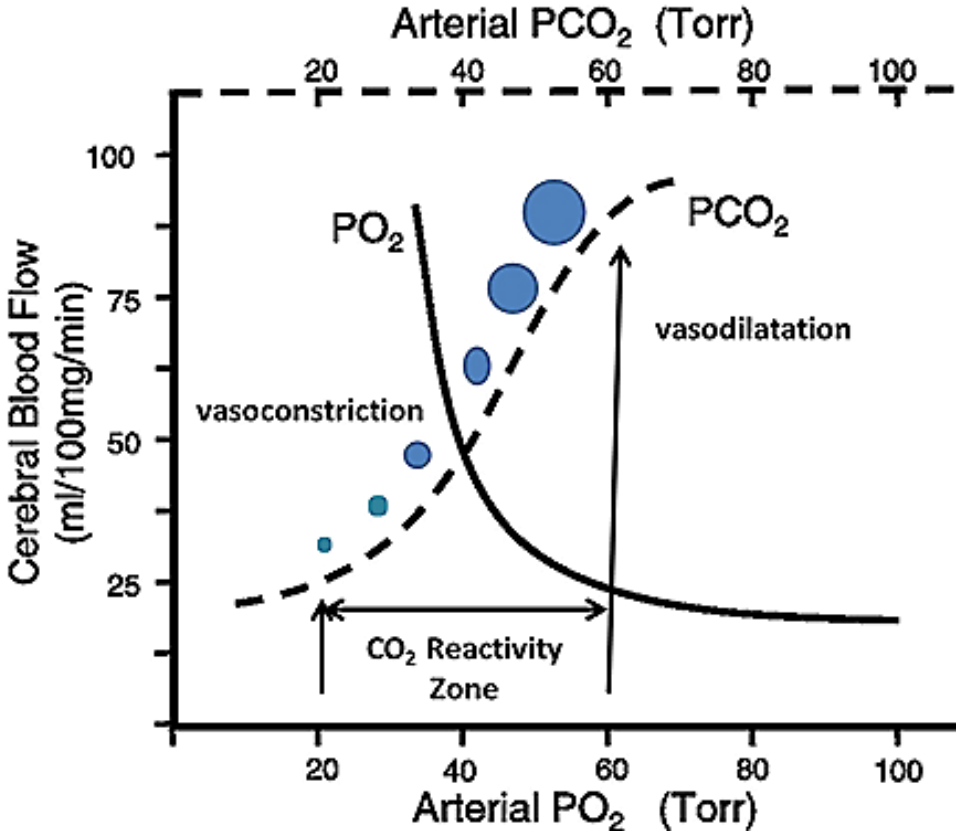
- Small ↑ICP = vasodilation + ↑ tissue O_2 extraction
- ICP > 20mmHg OK if CBF maintained around 60ml/100 g/min
- ICP ↑↑ ▸ CBF ↓↓ ▸ brain death





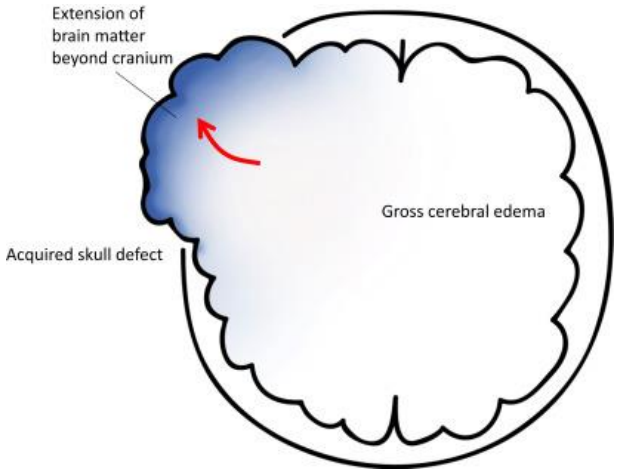
Cerebral blood flow and gasometry

- **$PaCO_2$**: most determinant factor for CBF
  - 20-80mmHg ⇨ vasodilation
  - <20mmHg ⇨ vasoconstriction
- **PaO_2**
  - < 60mmHg ⇨ vasodilation
  - > 100mmHg ⇨ vasoconstriction
- **pH**
  - ↓pH (acidosis) ⇨ vasodilation
  - ↑ pH (alkalosis) ⇨ vasoconstriction

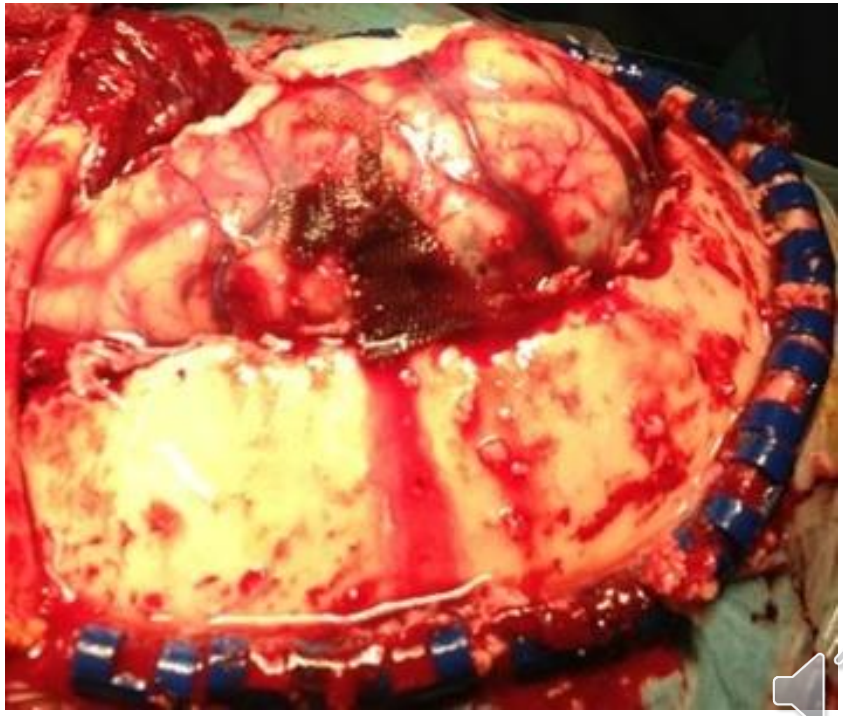


INTRACRANIAL HYPERTENSION (ICHT)

- **Normal ICP < 15 mmHg**
  - ICP > 20 mmHg → needs specific treatment
  - ICP > 25 – 30 mmHg → potentially lethal within hours / days
  - ICP > 40 mmHg → brain death within hours



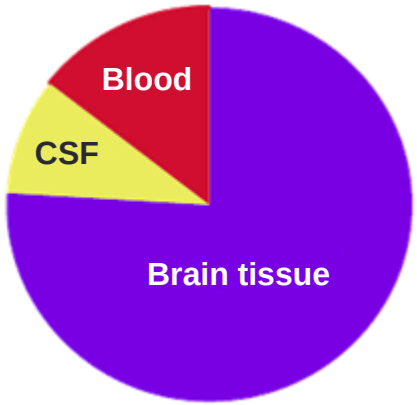
Massive brain herniation due to incontrollable ICHT (malignant ICHT)



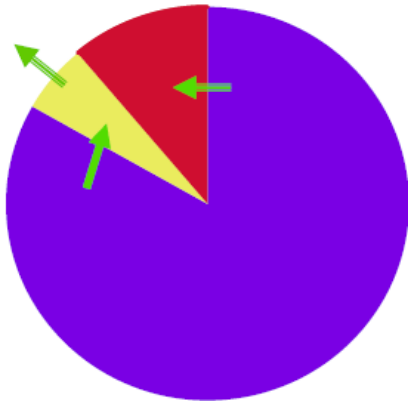
Etiology of ICHT

$$V_{BT} + V_{BL} + V_{CSF} = k$$

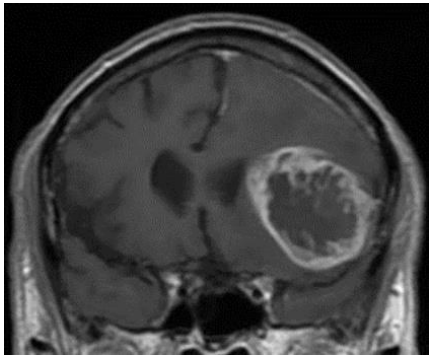
- ⇑ **Cerebral volume**
  - Space-occupying lesions
    - *Tumour, abscess, parasites*
  - Brain edema
    - *TBI, cardiorespiratory arrest, metabolic encephalopathy*
- ⇑ **Blood volume**
- ⇑ **CSF volume**



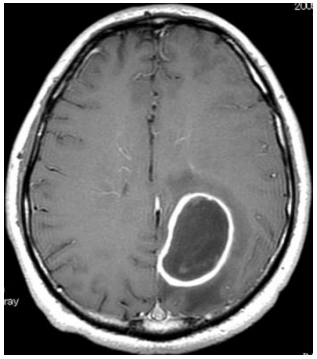
Normal



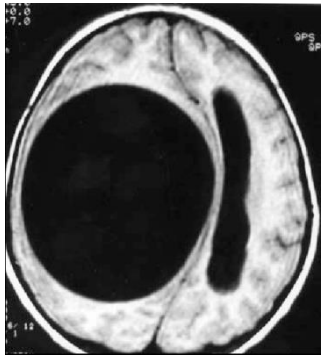
Brain edema



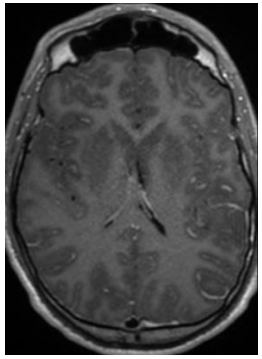
Tumour



Abscess



Hydatid cyst

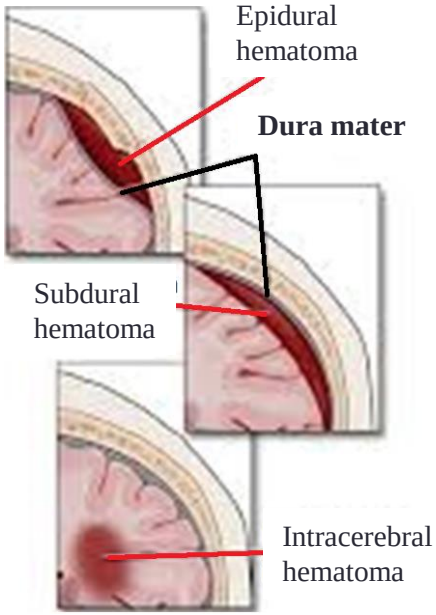


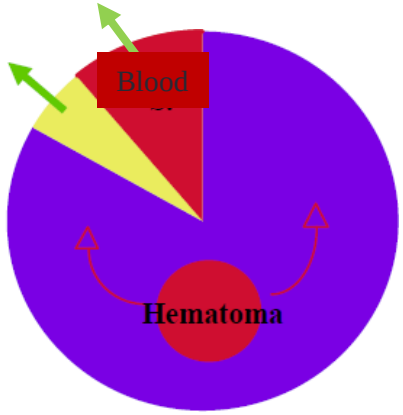
Oedema

ICHT etiology

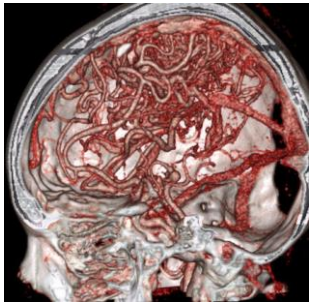
$$V_{BT} + V_{BL} + V_{CSF} = k$$

- ⇑ Brain volume
- ⇑ **Blood volume**
  - Extravascular: hematoma
    - *Epidural, subdural, intracerebral*
  - Intravascular
    - *AVM, giant aneurysms*
    - *Venous sinus thrombosis*
    - *Hyperaemia, brain congestion*
- ⇑ CSF volume

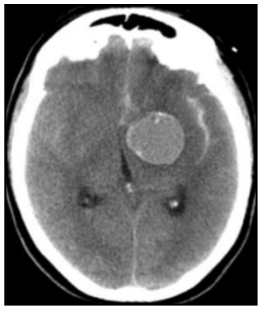




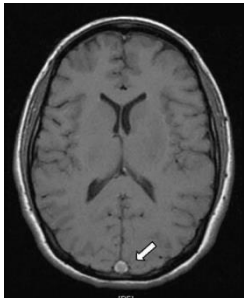
Intracranial hematoma



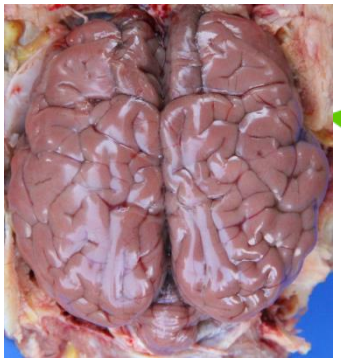
AV Malformation



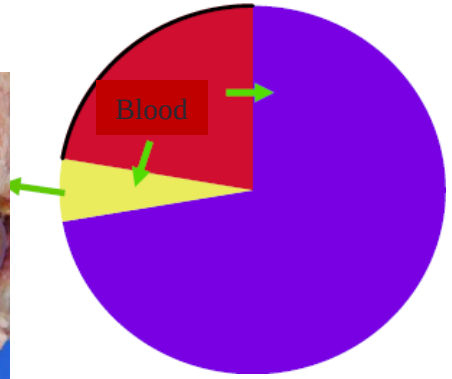
Aneurysm



Venous sinus thrombosis



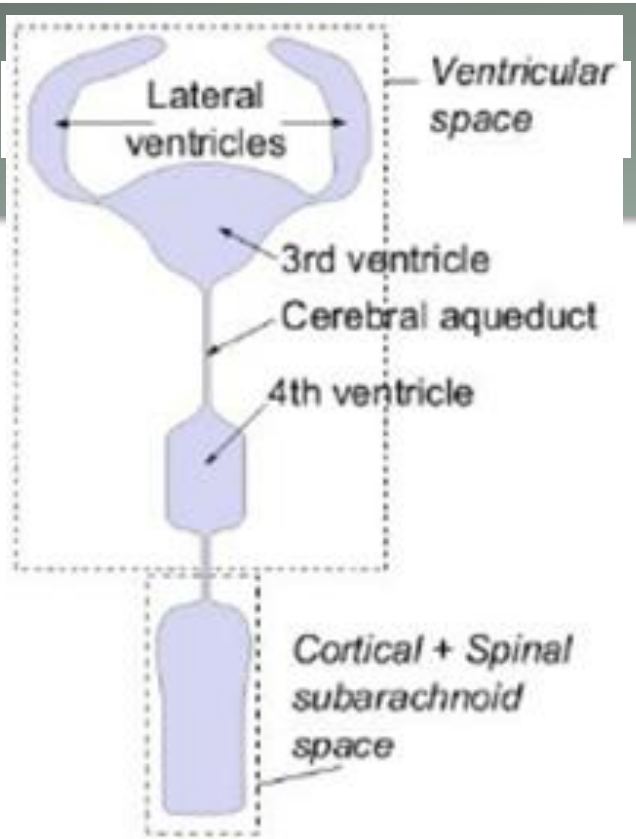
Brain congestion

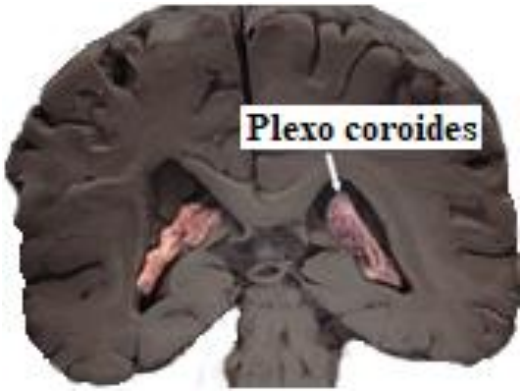


Brain congestion

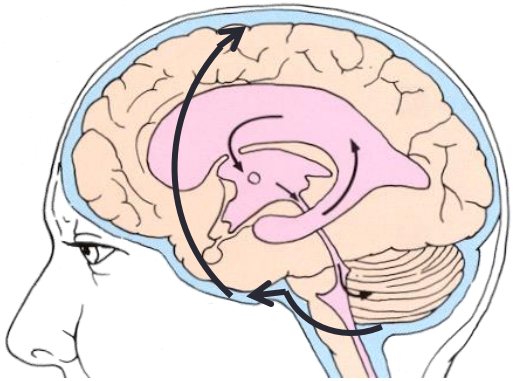
ICHT etiology

- ⇑ Brain volume
- ⇑ Blood volume
- ⇑ **CSF volume**
  - ⇑ Production
  - Flow obstruction
  - ⇓ Reabsorption

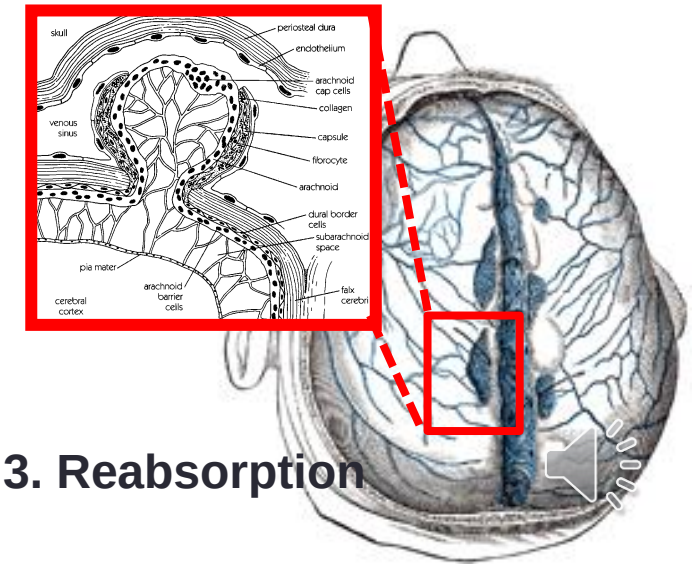




1. Production



2. Circulation

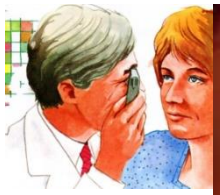


3. Reabsorption

ICHT clinical features

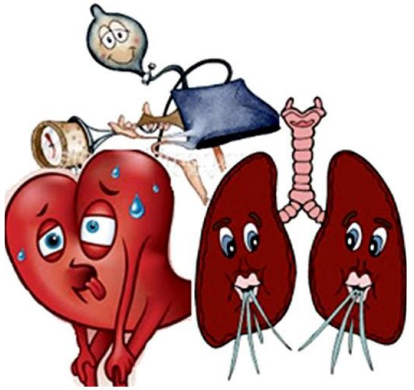
- **Headache**
  - ↑ at night, may awake the patient
  - ↑ with Valsalva manoeuvres

- **Vomiting**
  - ↑ in the morning (on waking-up from sleep)
  - Projectile vomiting



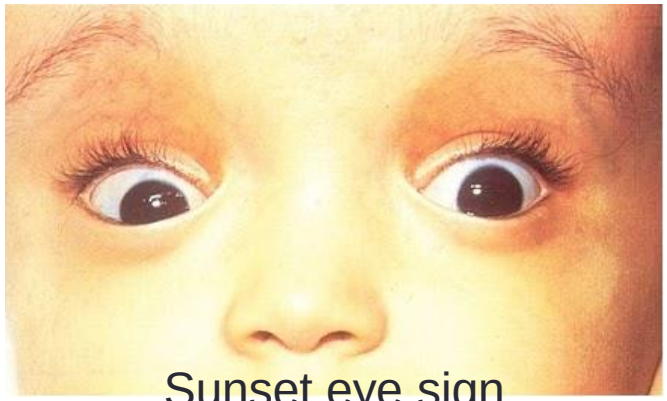
- **Papilledema** (fundoscopy)
- **Diplopia** (VI cranial nerve lesion), blurred vision

- **Decreased level of consciousness**
- **Cushing's triad**
  - Increased blood pressure, bradycardia, and irregular breathing (complete in only 30% cases)

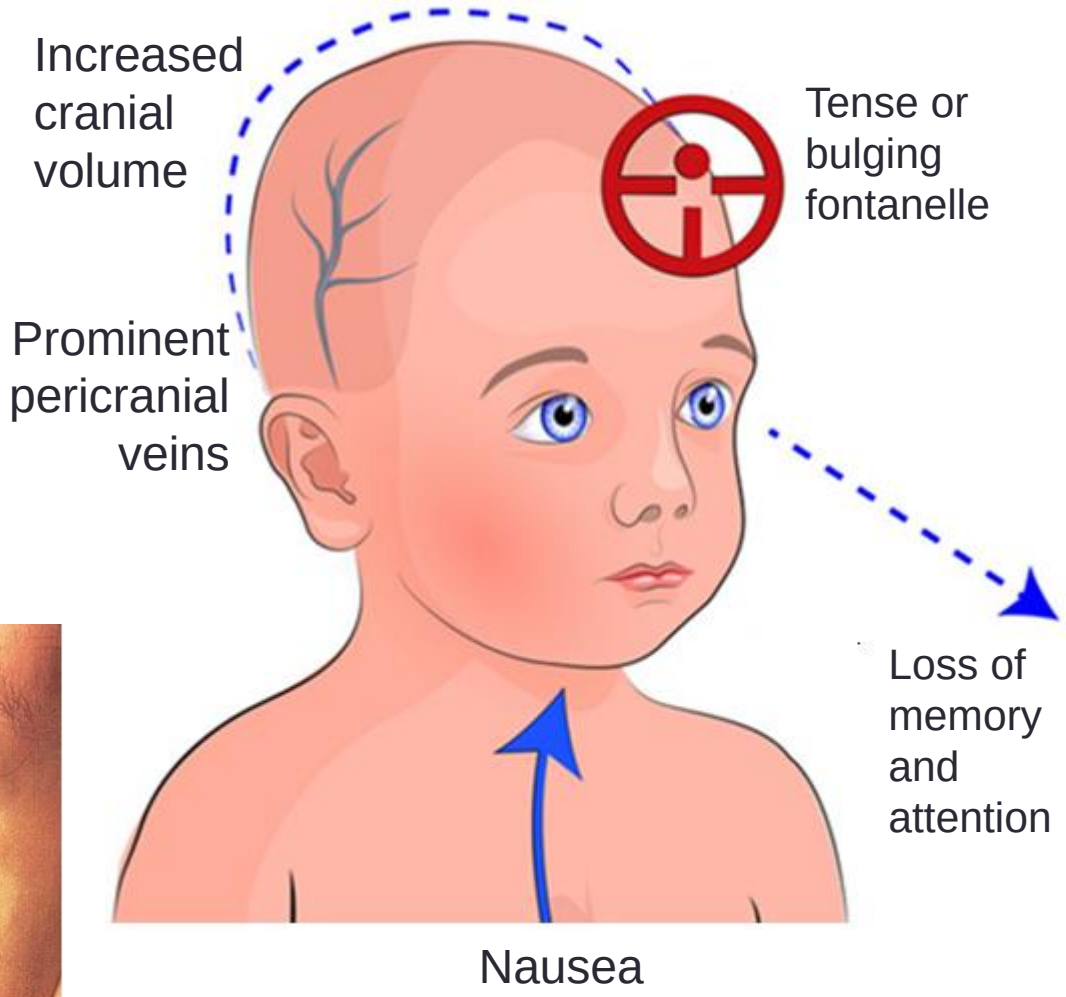


- **Focal neurological signs**

ICHT in young children

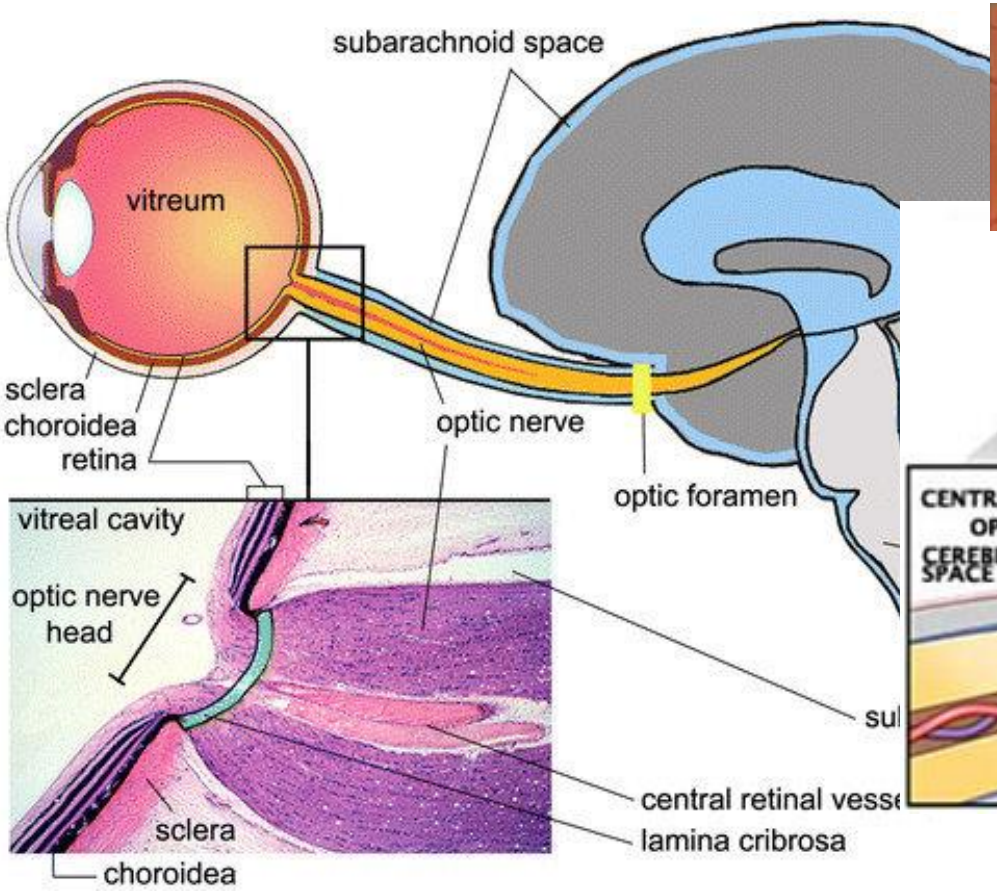


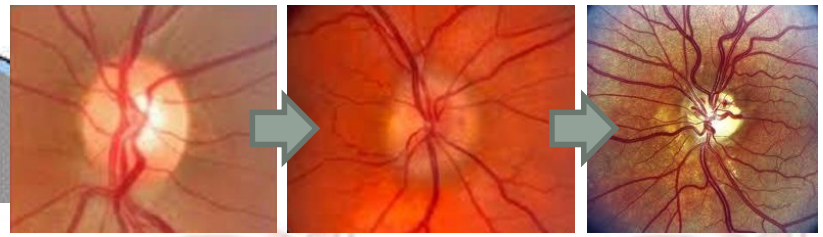
Sunset eye sign
(setting sun phenomenon)

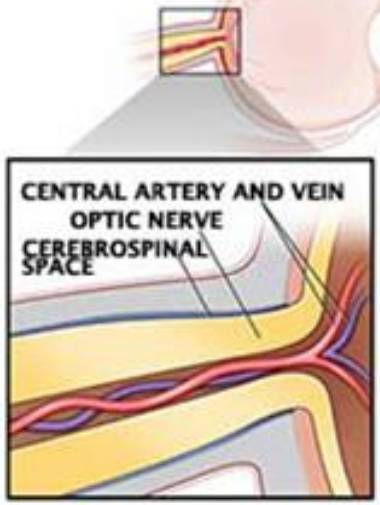


Effect of ICHT on optic nerve

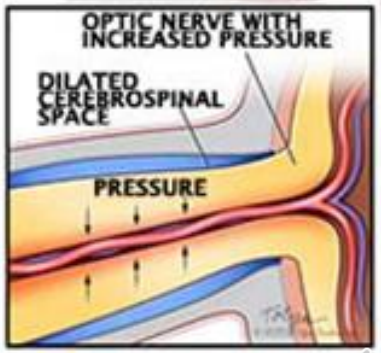
- Chronic ↑ ICP → atrophy of optic nerve papilla (pallor of optic nerve disc) → decreased visual acuity to blindness







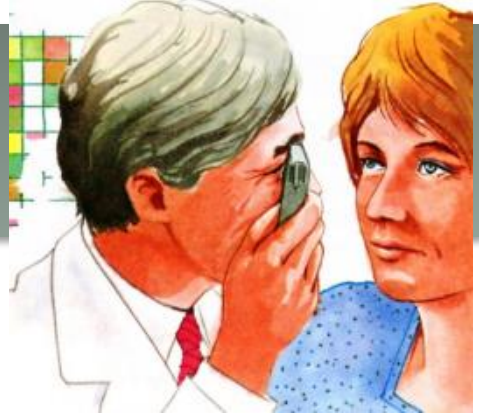
Normal



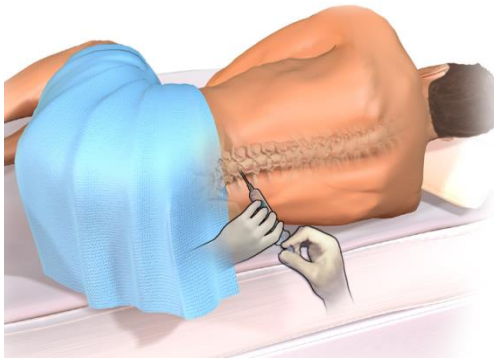
⇑ ICP

Diagnosis of ICHT

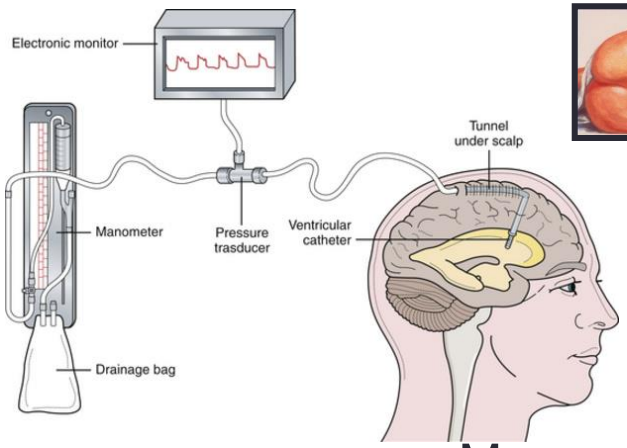
- Suspected from clinical features
- Ophthalmoscopy (fundoscopy)
- Lumbar puncture + CSF opening pressure
- **ICP measure**
- **Etiology diagnosis (image)**
  - MRI
  - CT
  - Cerebral angiography

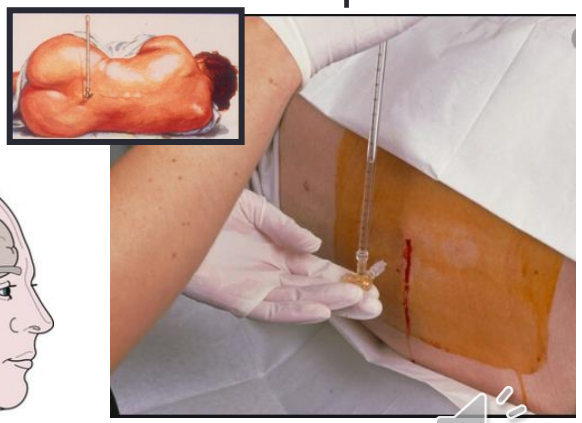


Fundoscopy



Lumbar puncture

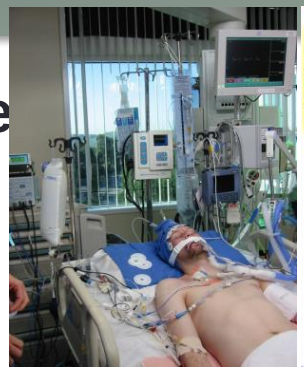


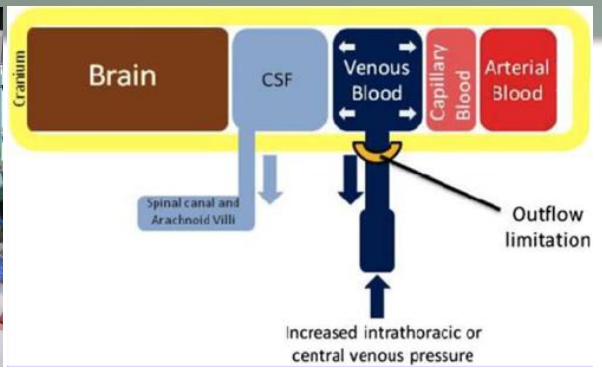


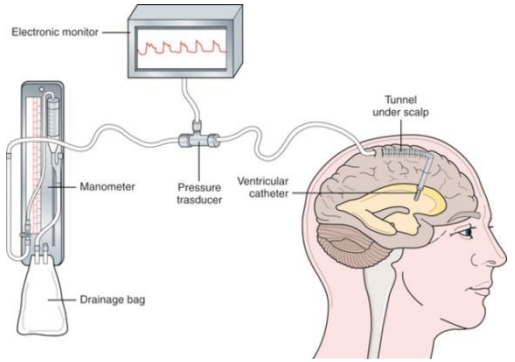
Measuring ICP

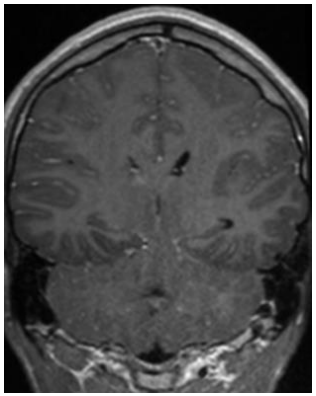
ICHT treatment

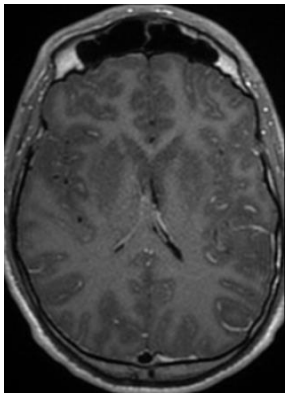
- **Etiological whenever possible**
- **1st level measures**
  - Raise head 30º
  - Sedation, intubation
  - Keep CPP (adequate MAP)
  - Hypertonic saline or Mannitol 20%
  - Measure ICP
  - External ventricular drainage
  - Hyperventilation (occasionally)
- **2nd level measures**
  - Barbituric coma
  - Decompressive craniectomy?
  - ~~Hypothermia~~





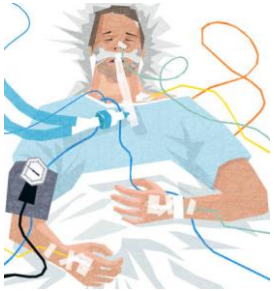


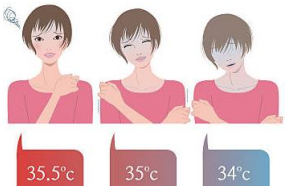


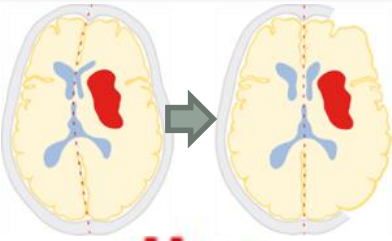


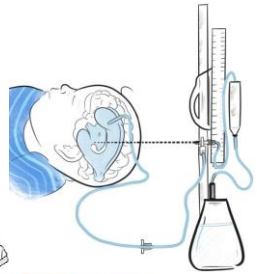
Treatment ladder ICTH

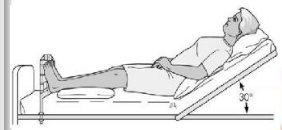
CPP > 60-70 mmHg
ICP < 22 mmHg











1 General prophylactic measures[a]

2 Resection of mass lesions CSF drainage

3 Hyperosmolar therapy

4 Hyperventilation

5 Therapeutic hypothermia

6 Barbiturates

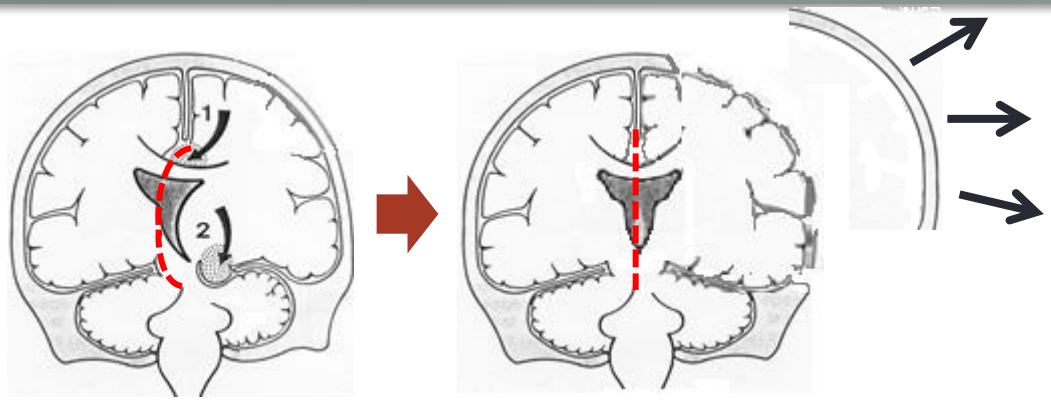
7 Decompressive craniectomy

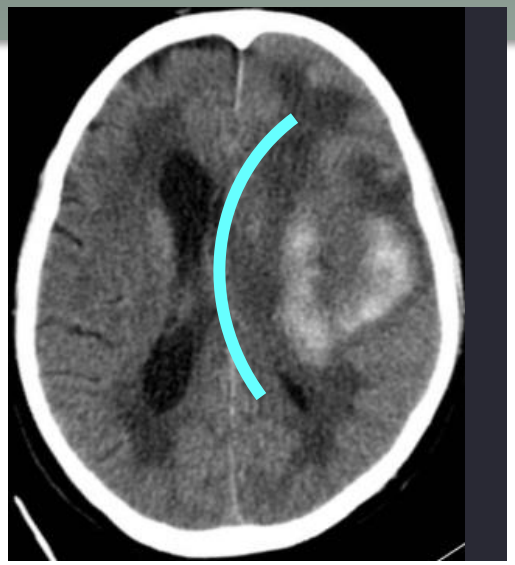
↓ : Mild or moderate effect on ICP decrease

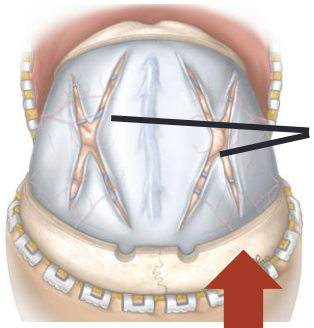
↓↓ : Significant effect on ICP decrease

[a]Intubation and mechanical ventilation
Avoidance of hypoxemia and hypercapnia
BP– CPP optimization
Head of the bed to 30°
Patient's head face midline
Fever control
Glycemic control
Seizure Prophylaxis

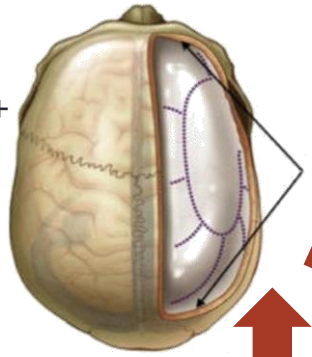
Decompressive craniectomy



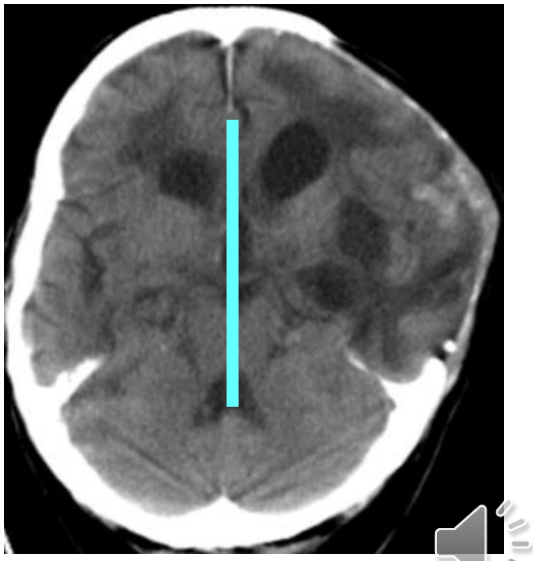


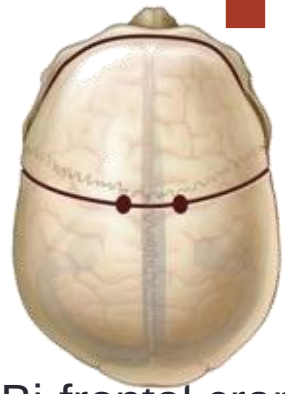


Cranieotomy + dural opening

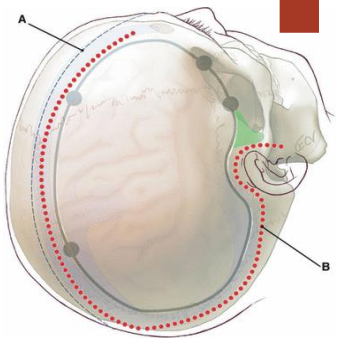


Cranieotomy + dural opening





Bi-frontal craniotomy

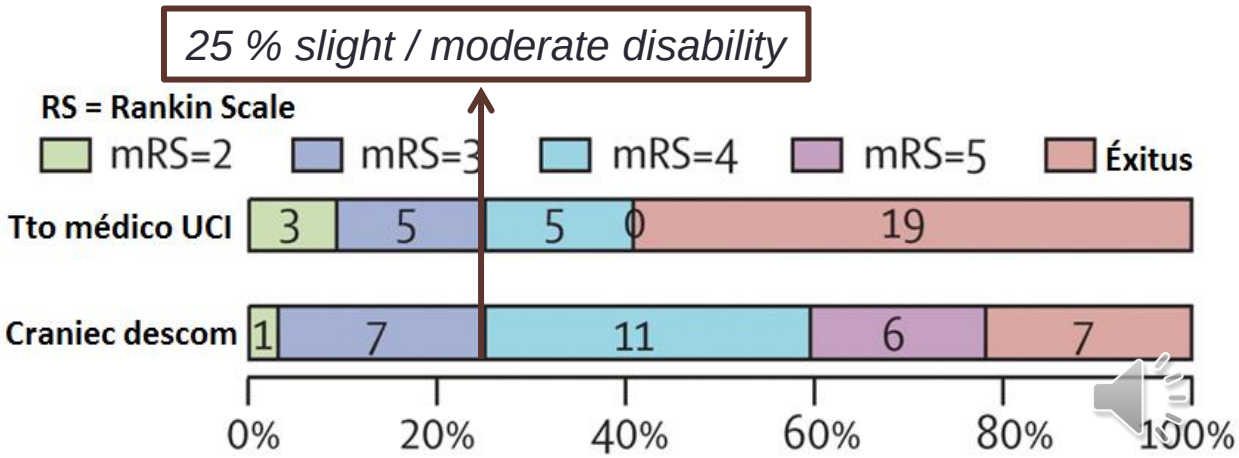


Hemicraniectomy

Decompressive craniectomy

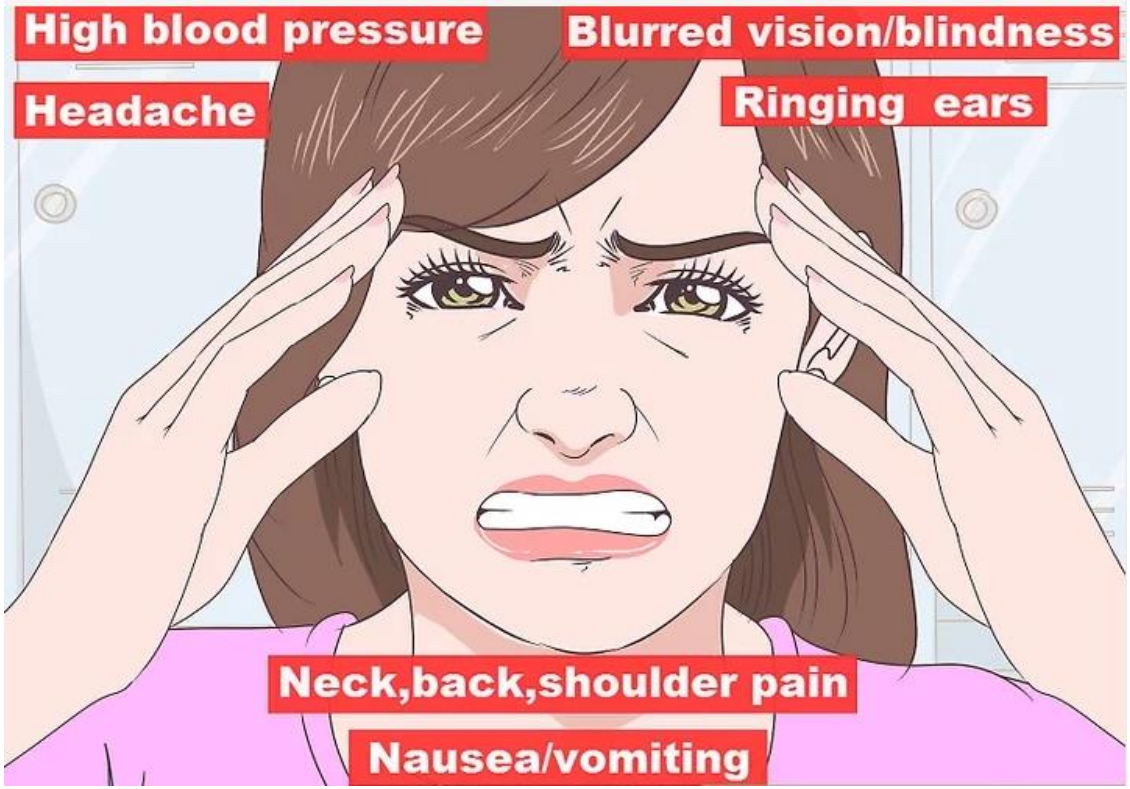
- Very restrictive indication
- Results:
  - ↓ ICP → ↑ survival
    - *25 % survivors with slight / moderate disability*
    - ***35 %** survivors with moderately severe disability (vs 15% with conservative tx)*
    - ***20 %** survivors with severe disability*

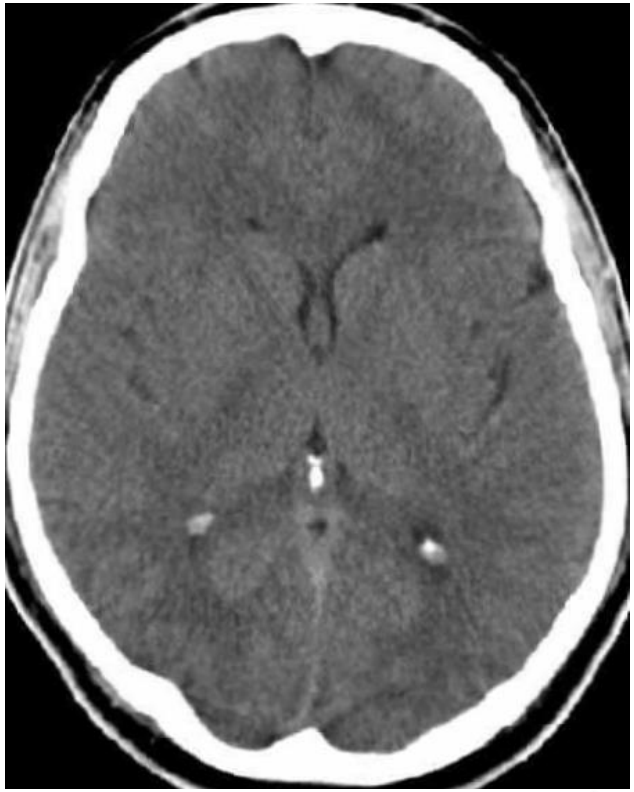
Modified Rankin Scale (MRS)	
0	No symptoms
1	No significant disability, despite symptoms; able to perform all usual duties and activities
2	Slight disability; unable to perform all previous activities but able to look after own affairs without assistance
3	Moderate disability; requires some help, but able to walk without assistance
4	Moderately severe disability; unable to walk without assistance and unable to attend to own bodily needs without assistance
5	Severe disability; bedridden, incontinent, and requires constant nursing care and attention
6	Death



BENIGN ICHT SYNDROME

- *Pseudotumor cerebri*: ICHT symptoms with **NO** clinical, lab, or imaging evidence of intracranial pathology



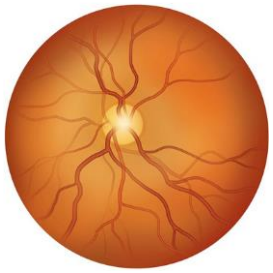


Benign ICHT symptoms

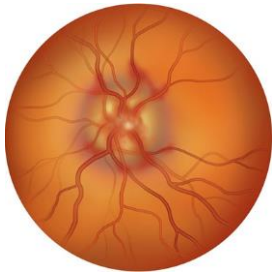
- **Young obese woman + symptoms and signs of ICHT**
  - Morning frontal headache, pulsatile tinnitus
  - Blurred vision > diplopia (lesion VI cranial nerve)
  - **Papilledema**
    - *↑ Blind spot*
    - *↓ Peripheral visual fields*
    - *Loss of vision (optic nerve atrophy)*
  - No altered level of consciousness
  - No neurological focal signs

Benign ICHT diagnosis

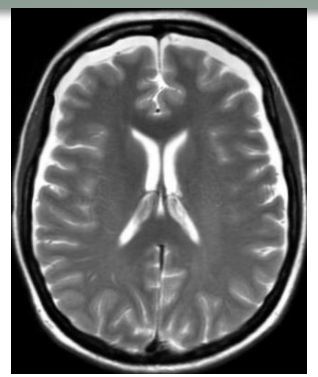
- **∅ findings in CT/MRI**
  - ∅ ventricle enlargement, masses
- Lumbar puncture
  - **↑ CSF pressure**
  - Normal CSF biochemistry
- Fundoscopy: **papilledema**
- Campimetry
  - Reduction of peripheral visual fields
  - **Loss of visual acuity to blindness**

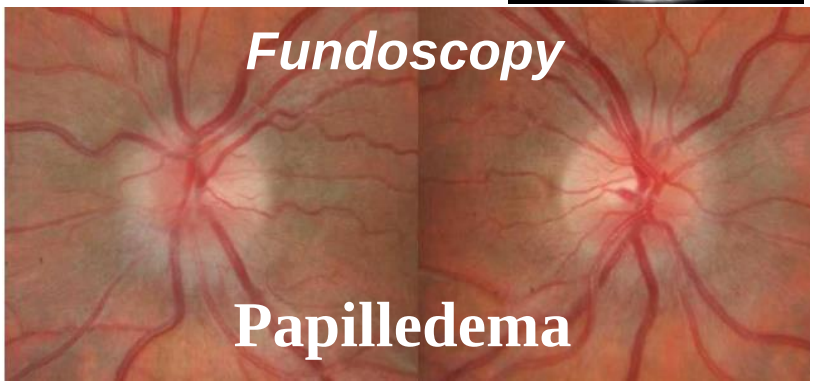


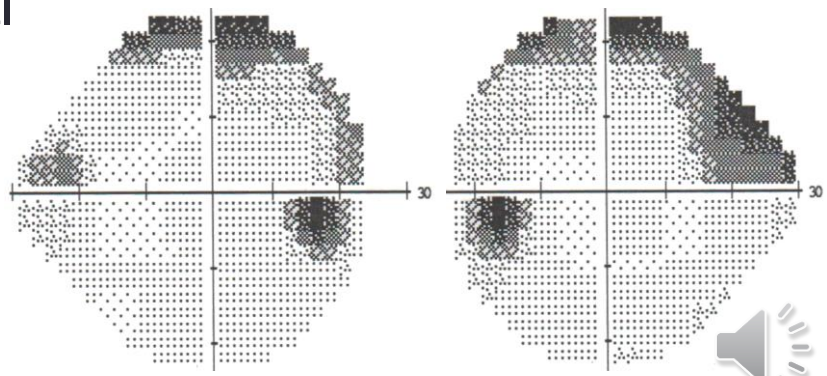
Normal



Papilledema



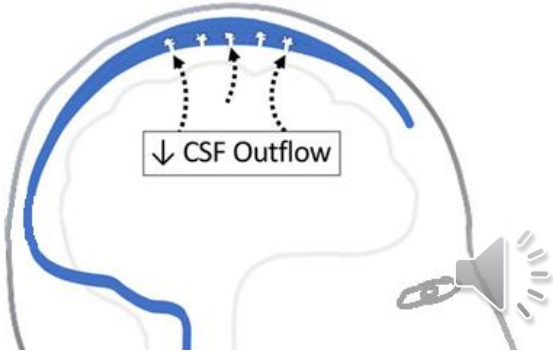




Campimetry

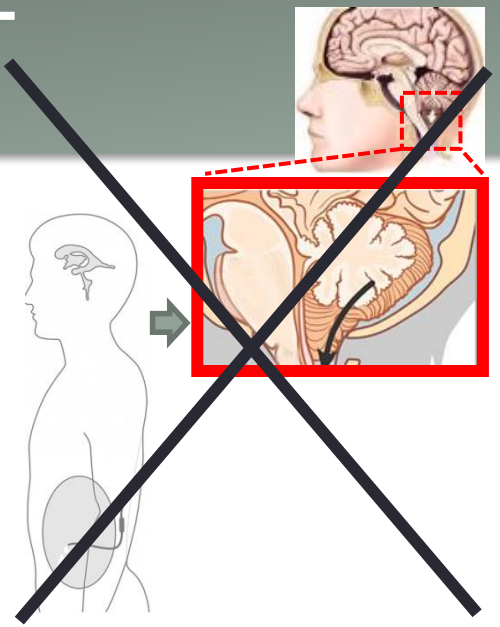
Etiology of benign ICHT syndrome

- **Idiopathic** (most frequent)
  - ***Woman, reproductive age, menstrual abnormalities, obesity, or recent weight gain***
- Abnormal venous drainage with cerebral venous sinus stenosis
- Abnormal hormonal status
  - *Pregnancy, hypo / hyperthyroidism, adrenal insufficiency, hypercortisolism…*
- Drugs
  - *Vit A, tetracyclines, nitrofurantoin,* sulphonamides*, indomethacin, nalidixic acid, phenytoin, lithium, anabolic steroids, corticoids,* ***contraceptives***
- Autoimmune
  - *Sarcoidosis*
  - *Lupus erythematosus*

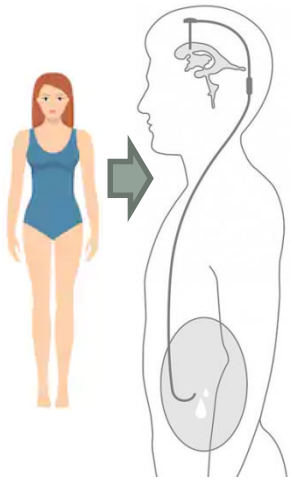


Treatment of benign ICHT

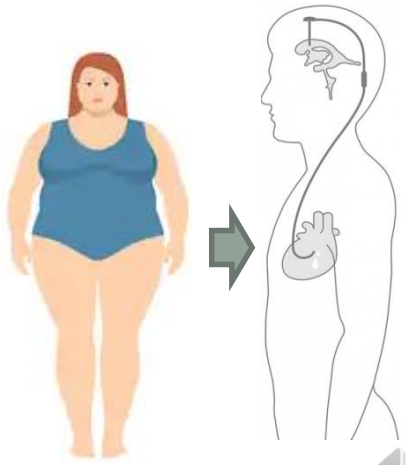
- Usually self-limited
  - If recurrent, possible progressive loss of vision
- Avoid associated factors
  - Weight loss
  - Diet: reduce salt and water
  - Review possible drugs (contraceptives)
- Treatment
  - Acetazolamide, furosemide
  - Repeated lumbar punctures
  - CSF drainage system = ventriculoperitoneal or ventriculoatrial shunt
  - Optic nerve sheath fenestration



Lumboperitoneal shunt

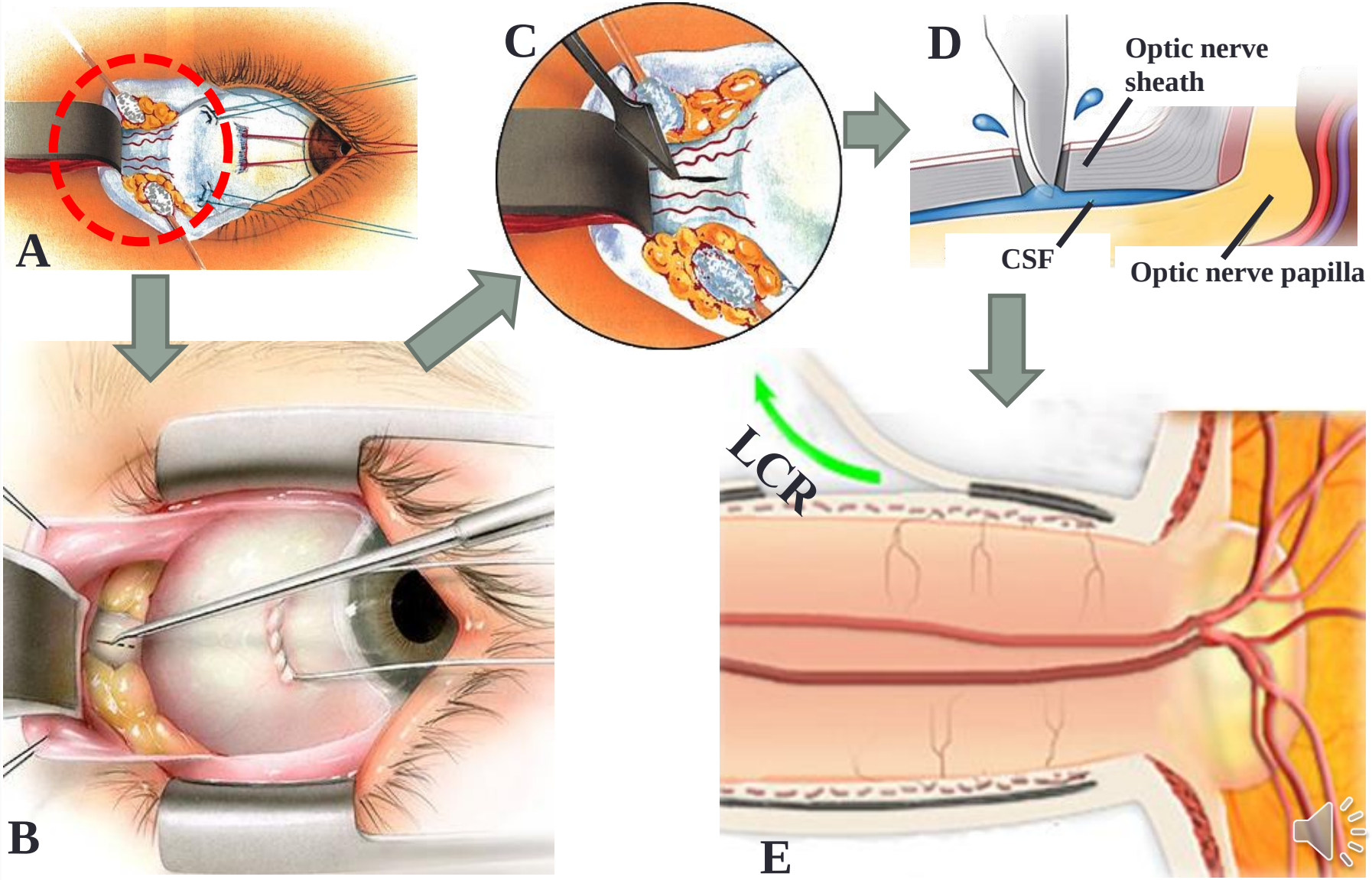


Ventriculoperitonal shunt



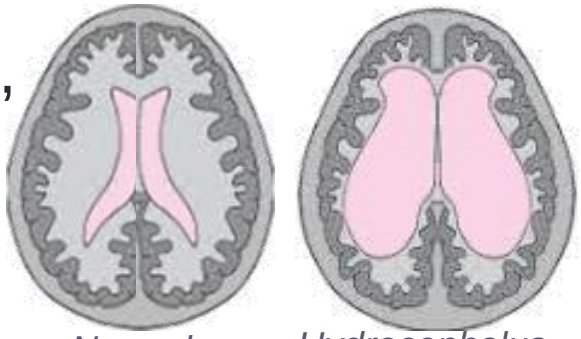
Ventriculoatrial shunt

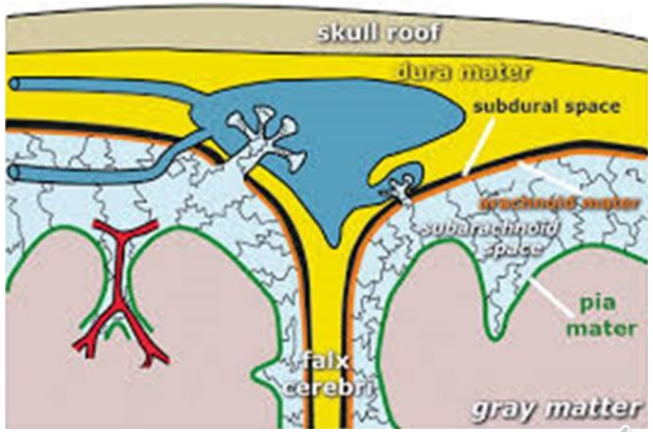
Optic nerve sheath fenestration



CHRONIC ADULT-ONSET HYDROCEPHALUS

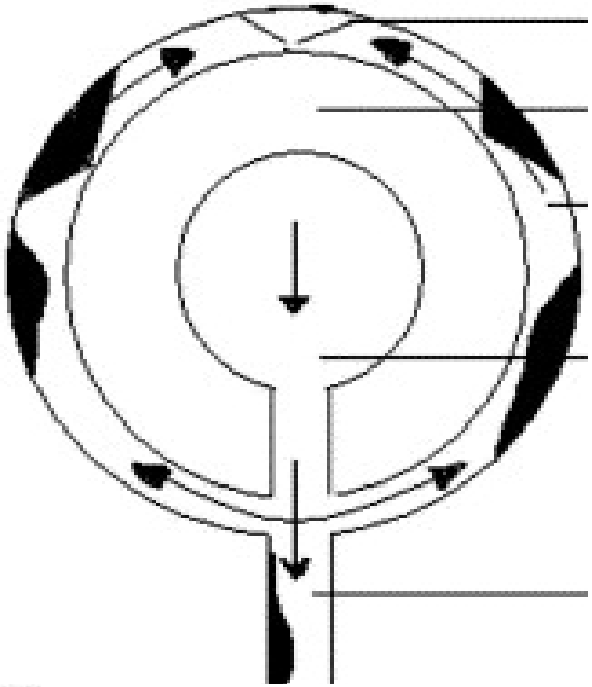
- Synonyms: normotensive hydrocephalus, normal pressure hydrocephalus
- Characteristics
  - *Communicans* hydrocephalus
  - Block at arachnoid villi or subarachnoid space
  - ICP is normal during the day, but increases at night during REM phase
  - **Only "curable" dementia**
- Epidemiology
  - Predominance ♂ **> 60-year-old**
  - Prevalence 0.5 % > 65-year-old
    - *9-14 % in senior centres*
  - Incidence 5.5 cases/100.000 in h/yr
  - Peak sixth-seventh decade

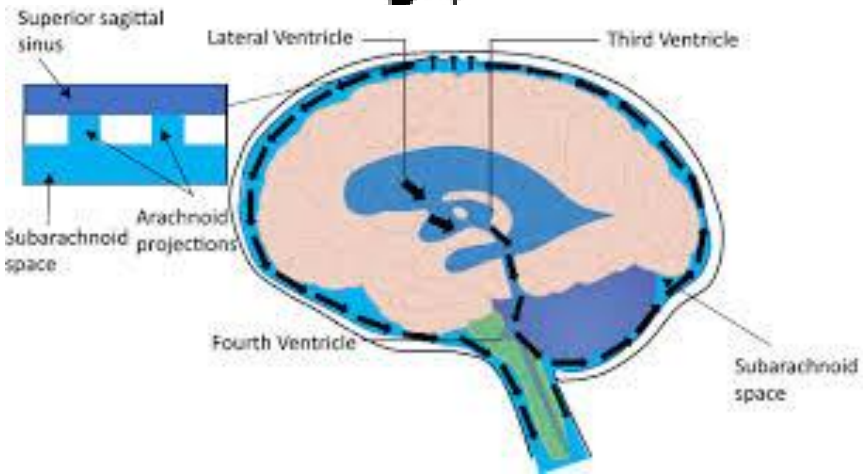




Etiology of chronic adult-onset hydrocephalus

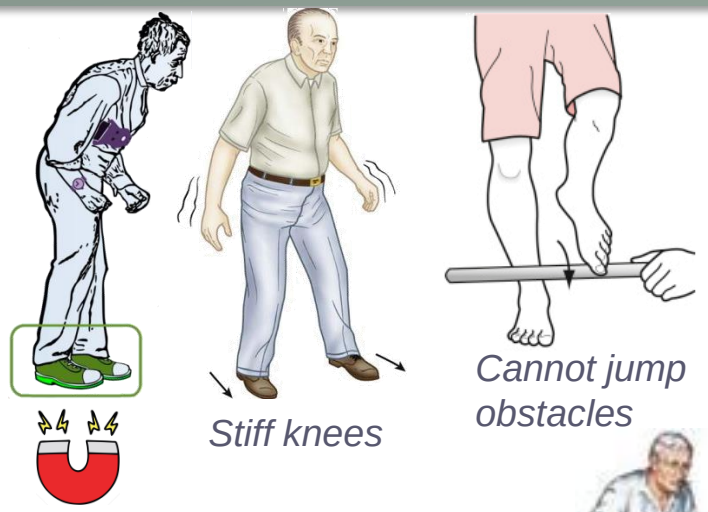
- **Idiopathic** 40-60%
- **Secondary**
  - Post-haemorrhagic
    - *Subarachnoid haemorrhage*
    - *Post-traumatic*
    - *Post-surgery*
      - *Especially posterior fossa*
  - Post-meningitis
  - Tumours
  - Alzheimer's (15%)

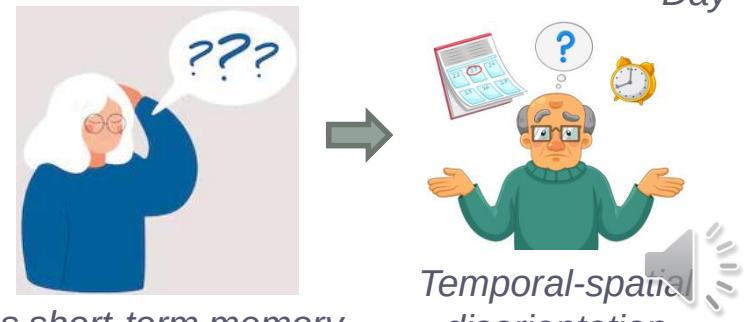




Symptoms: Hakim-Adams triad

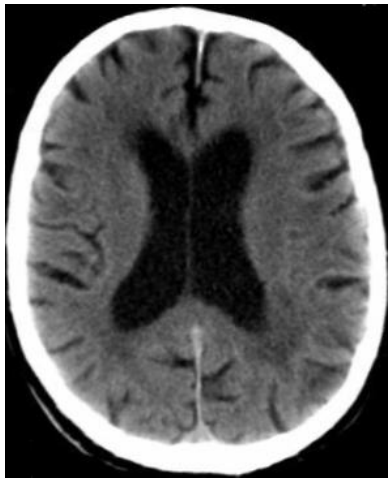
- Typical triad (not pathognomonic): "Wet, wacky and wobbly"
  - **Gait disturbance** ("magnetic gait")
    - *Early sign*
    - *Small steps, stiff knees, dragging feet*
    - *Sometimes also Parkinsonism*
  - **Urinary incontinence**
    - *Urinary urgency →→→ Nycturia*
  - **Dementia**
    - *Slow planning, organisation, attention, concentration*
    - *Loss of short-term memory*
    - ➢ *If patient gets lost or hallucinates, consider Alzheimer's!*



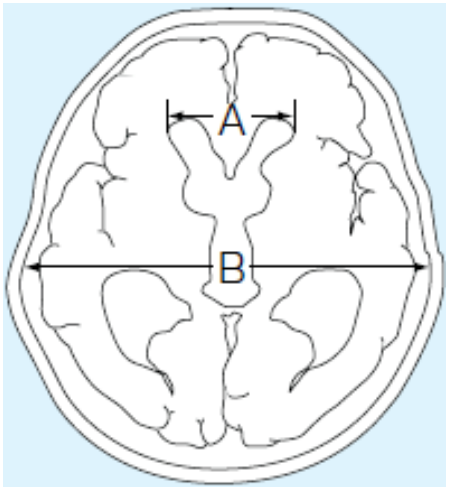


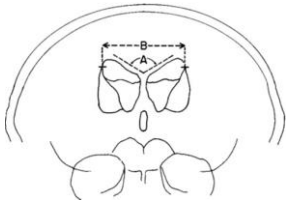
Diagnosis of chronic adult-onset hydrocephalus

- **Image: CT/ MRI**
  - Ventricular enlargement in CT/MRI
  - Size is variable (not related to hydrocephalus)
    - *Cerebral atrophy?*
  - **CT: Evans index** ≥ 0.30 = pathologic
    - *Ratio of maximum width of the frontal horns of the lateral ventricles and the maximal internal diameter of the skull at the same level (A/B)*
  - **MRI**
    - *Corpus callosum angle <90º*
    - *periventricular oedema (ependyma transudate, transependymal edema)*
    - *Non-specific, also present in cerebrovascular disease*
- Lumbar puncture
- Measurement of ICP

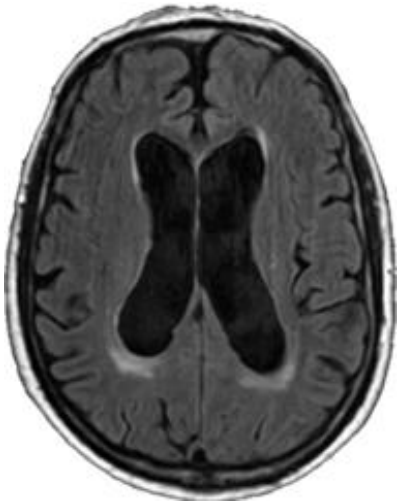


Brain atrophy

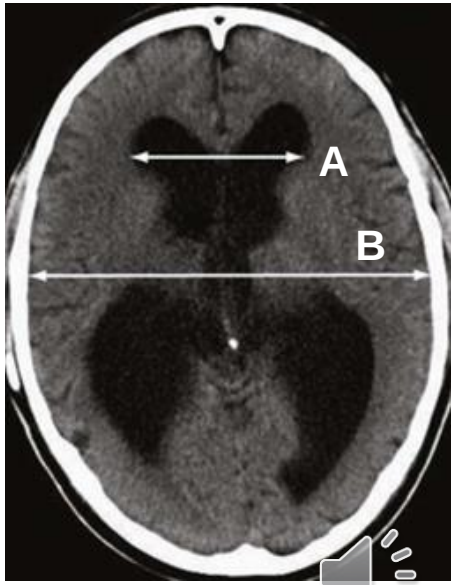




Corpus callosum angle



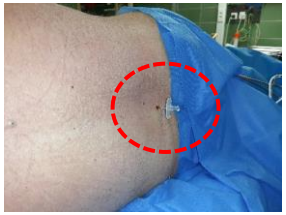
Periventricular edema

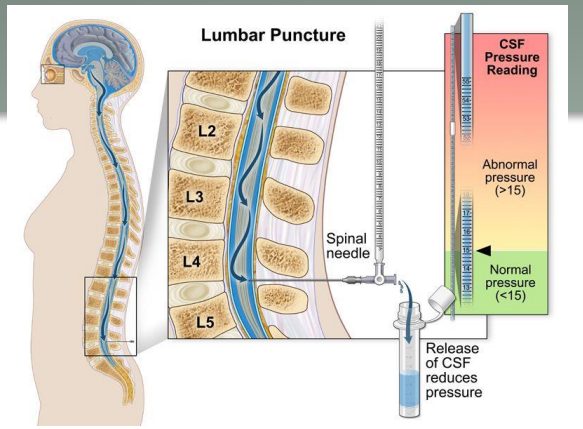


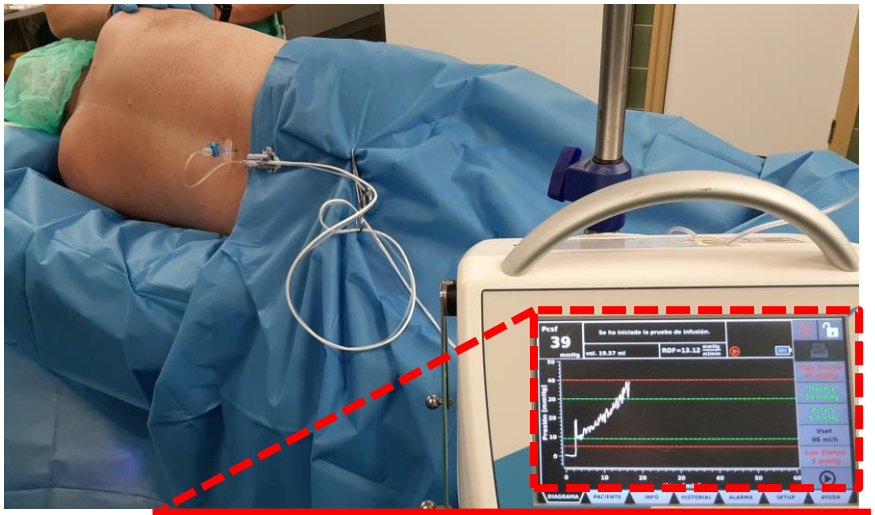
Evans index (A/B)

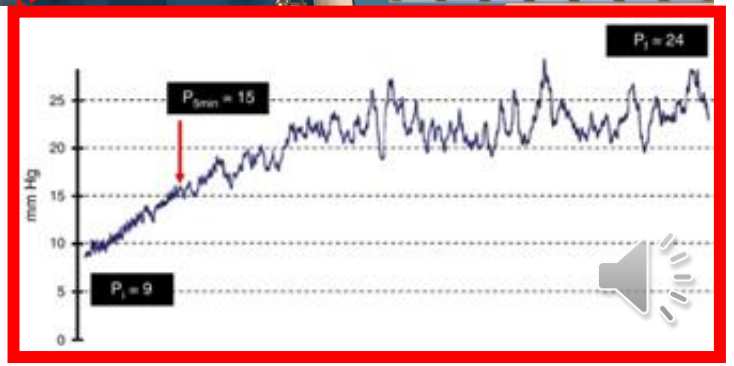
Diagnosis of chronic adult-onset hydrocephalus

- Image: CT/MRI
- **Lumbar puncture**
  - CSF opening pressure usually normal
  - **Drain 40-50ml CSF**
    - *If improvement for 3-5 days ⇨ positive (but NOT pathognomonic)*
  - **Katzman infusion test**
    - *Measures resistance to drainage of CSF*
- ICP measurement





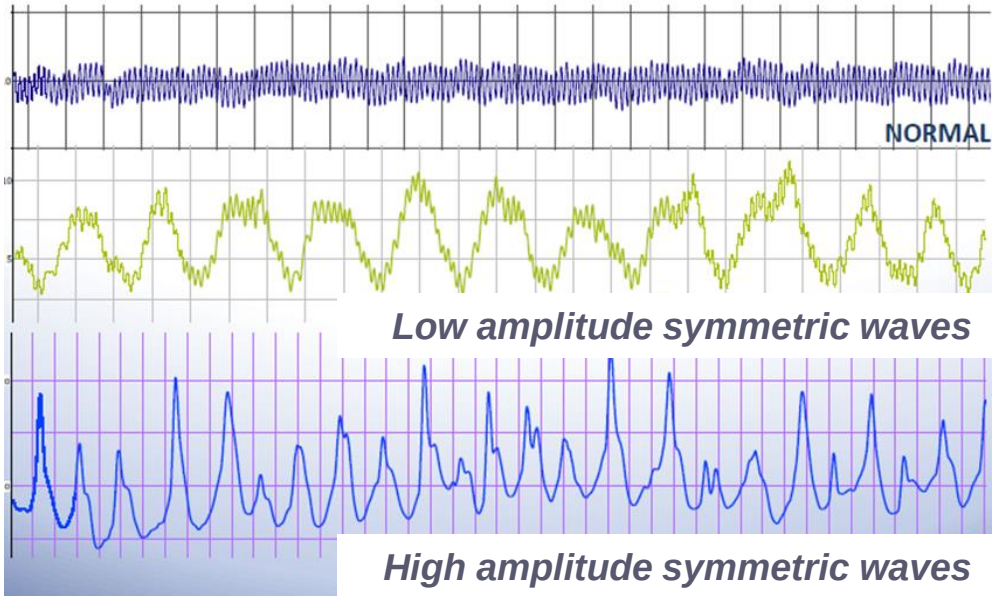




Diagnosis of chronic adult-onset hydrocephalus

- Image: CT/MRI
- Lumbar puncture
- **ICP measurement**
  - The most sensitive and reliable
  - Invasive
  - Needs 5-day hospital stay

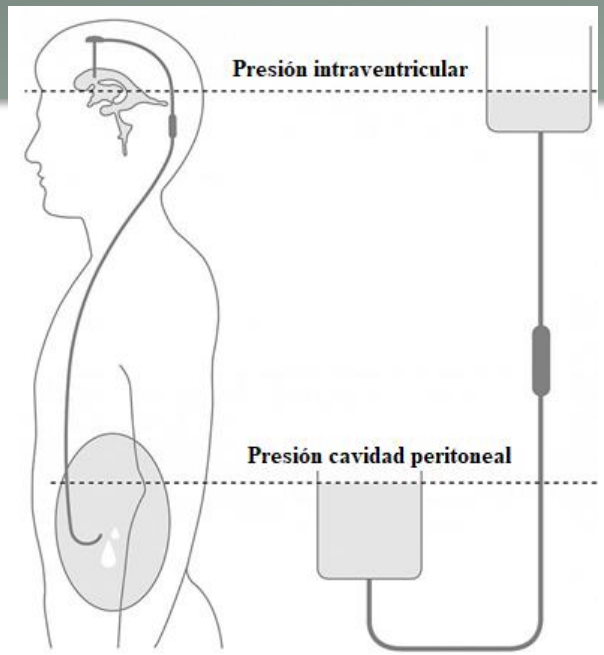
ICP measurement



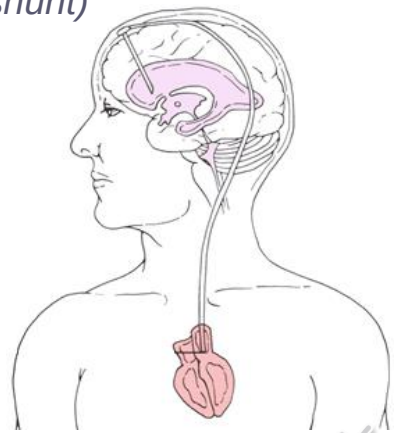
Night ICP graph during sleep at REM phase

Treatment of chronic adult-onset hydrocephalus

- CSF shunt
- Usually ventriculoperitoneal shunt (VP shunt)
  - Ventriculoatrial if obesity or peritoneal pathology
- Mixed dementia = chronic adult-onset hydrocephalus + other types of dementia
  - Early results initially good, but will deteriorate over time



Ventriculoperitoneal shunt (VP shunt)



Ventriculoatrial shunt

SUMMARY KEY CONCEPTS TOPIC 1

- Monro-Kellie doctrine
- Compensatory mechanisms against ICP increase
- Compliance
- Intracranial space partitions: brain hernias
- Cerebral blood flow: autoregulation, relationship with ICP, systemic MAP, $PaCO_2$, PaO_2 and pH
- ICHT: Cushing's triad, measurement of ICP, 1st and 2nd level manoeuvres (decompressive craniectomy)
- Benign ICHT syndrome: headache and loss of visual acuity, woman, obesity, contraceptives. Treatment: loss of weight, VP shunt or VA shunt
- Chronic adult-onset hydrocephalus: Hakim-Adams triad, atrophy *vs* hydrocephalus, measurement of ICP, ventriculoperitoneal and ventriculoatrial shunt, mixed dementia (good initial results that deteriorate over time)

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