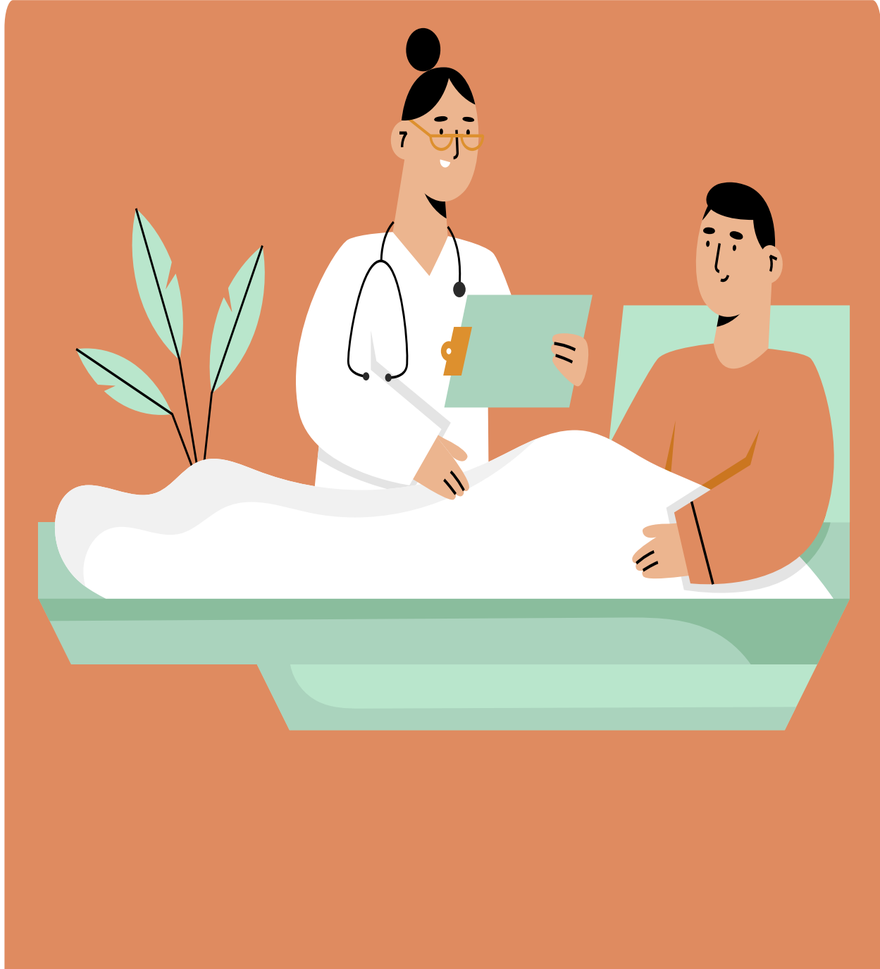
MED437
KING SAUD UNIVERSITY

SURGERY TEAM 437



Raised Intracranial Pressure

Objectives:

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Color Index:

- Important
- Doctor's Notes
- Extra
- Davidson's

Editing File / Feedback

Intracranial Pressure

Basics

★ The brain is enclosed within a rigid bony container. Intracranial pressure (ICP), therefore, depends on the relative volumes of **Components of cranium:**

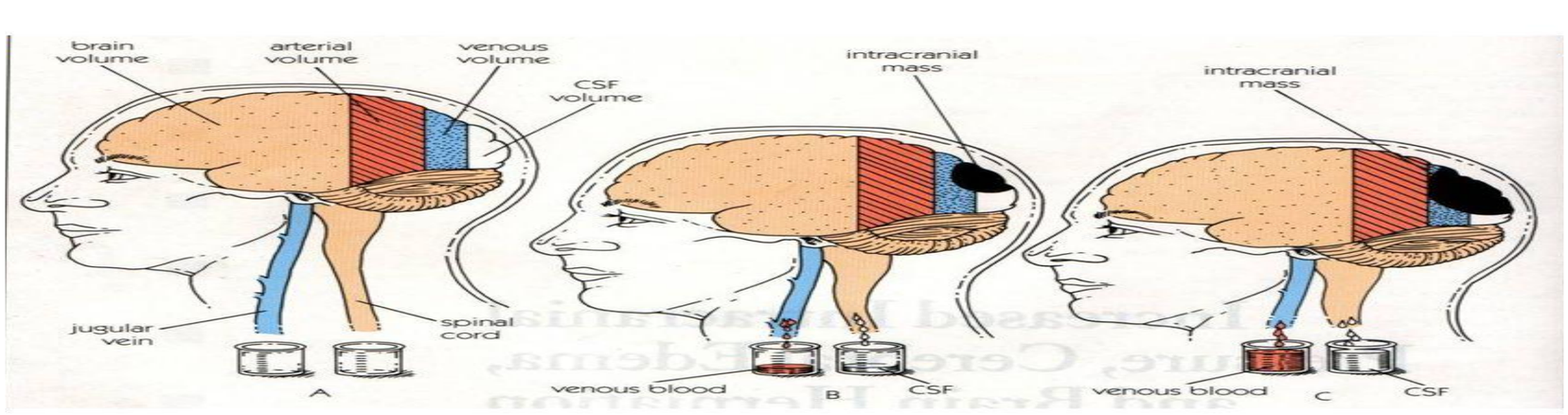
- Brain parenchyma = 1400 ml
- CSF = 75-100 ml
- Blood = 75 ml

In order for them to exist together they have to be in equilibrium, e.g. a brain tumor → takes more volume → CSF & blood go down to accommodate & maintain normal ICP and this is the **Monro-Kellie hypothesis**

★ ICP fluctuates in response to normal variation in intrathoracic pressure (e.g., increased by coughing, defaecation) and cardiac pulsation. These transient increases do no harm.

★ **Monro-Kellie Doctrine:**

- These contents are incompressible
  - bc they're fluid not solid; imagine having a cardboard box, if you crush it what will happen? The volume will go down, where if you have a bag full of fluid you're trying to crush the fluid will only move from one compartment to another
- Therefore, change in volume of the brain is associated with change in CSF or blood volume.



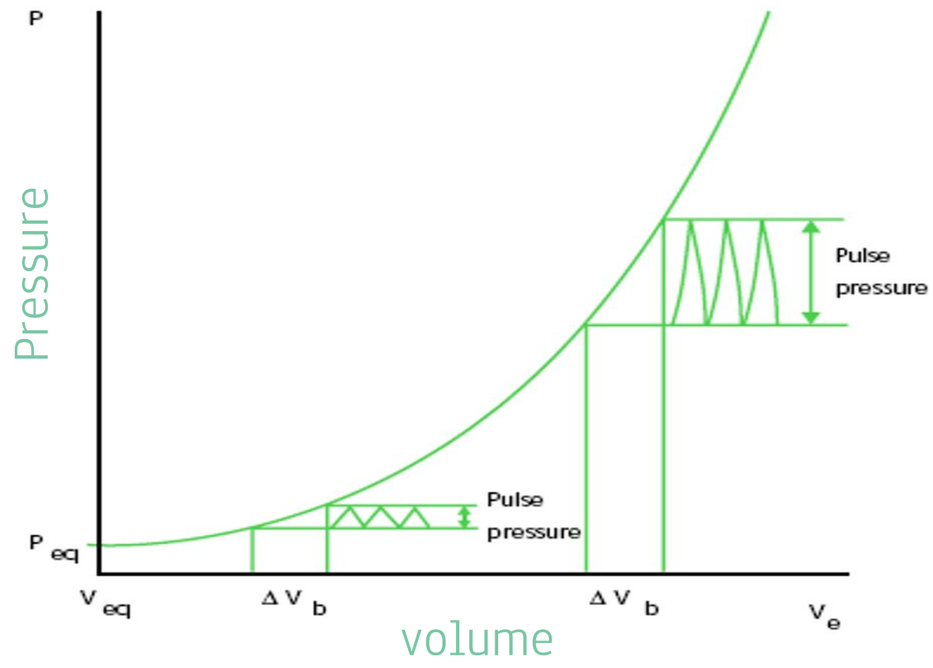
Pressure-Volume

- Increase in volume in one compartment leads to change in volume in the other ones.
  - E.g. brain tumor leads to decrease in CSF volume → decrease in blood volume (by increasing venous drainage to compensate)
- For how long could this go on? Until the system decompensate

Raised Intracranial Pressure

Pressure-Volume Curve

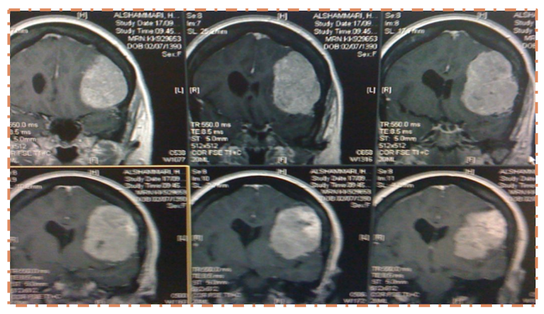
- The x-axis represents the volume while the y-axis is for pressure
- The relationship between pressure and volume is a non linear relationship, but a progressive one where things might get out of control very rapidly.
- At first when the volume increases for example in patients with intracranial mass lesions (Tumour, haemorrhage), oedema or CSF obstruction there is only a mild change in pressure because there is compensation by a reduction in cerebral blood volume and CSF volume. (so the patient may only have headache or nausea).



- However, a critical point is reached where no further compensation is possible, and any additional volume insult or any small change in volume will lead to exponential/rapid rise in intracranial pressure.
- An example is a patient suffering from uncontrollable hypertension causing small bleeding, early in the curve where the patient will start having headache, later on as the bleeding start accumulating the pressure will increase rapidly until the same amount of bleeding results in a huge pressure change, which can result in significant change in the level of consciousness.
- The *rate of increase* in the volume of intracranial mass is crucial because a rapid increase in ICP may precipitate acute reduction in conscious level.
- In patients with intracranial mass lesions (tumour, haemorrhage), oedema or CSF obstruction, the extra volume is at first compensated for by a reduction in cerebral blood volume and CSF volume. A critical point is reached where no further compensation is possible. Any additional volume (e.g., increasing haematoma) leads to an exponential rise in ICP. The rate of increase in the volume of intracranial mass is crucial

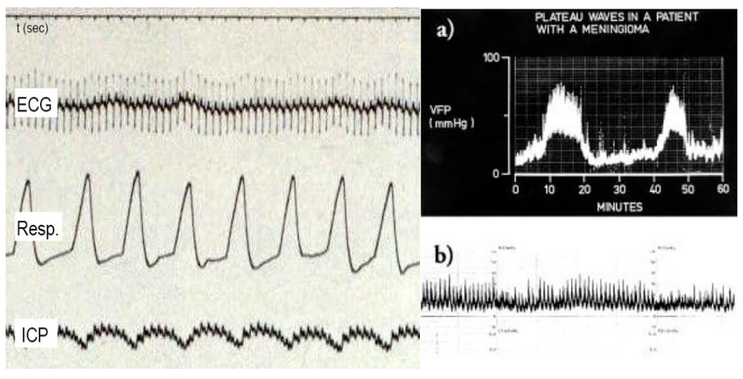
Can somebody walk around with a raised ICP? Yes

- This patient has left sided intracranial tumor (meningioma) which is very slowly growing causing symptoms such as right sided numbness and mild headache.



Intracranial pressure (ICP) waveform

- Can we measure the ICP? Yes, by placing a catheter Inserted through an opening in the skull.
- The waves recorded are related to the pressure coming from the heart into the brain's internal carotid artery.
- It has a waveform bc of the diastolic & systolic pressure



Normal Intracranial pressure (ICP)

★ Adults ⟶ <10-5 mm Hg
★ Children ⟶ 3-7 mm Hg
★ Term infants ⟶ 1.5-6 mm Hg (why is it lower? bc the skull sutures aren't fused yet)

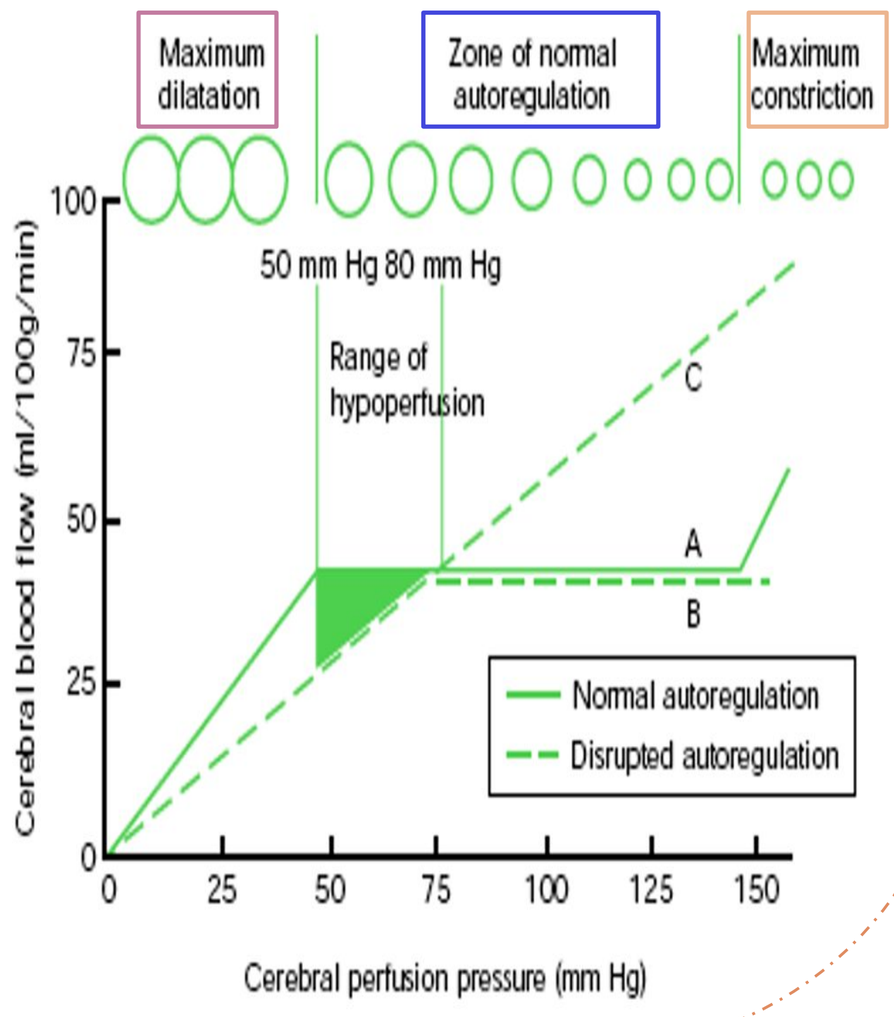
Cerebral Autoregulation

★ Ability of cerebral vessels to maintain cerebral perfusion within strictly determined limits:
- Rise in SBP leads to ⟶ Constriction of cerebral arteries.
  - To prevent rupture of the vessels due to the high pressure
- Low SBP leads to ⟶ Cerebral vessels dilate to accommodate.
  - while fasting, starving, or dehydration, the vessels dilate to deliver more blood to the brain
- However, if there is a severe and sustained elevation of ICP the compensatory mechanisms become ineffective & cerebral perfusion may be focally or generally compromised, leading to cerebral ischaemia and infarction.

★ Loss of autoregulation: Change in cerebral blood flow with the change in blood pressure (BP).

Blood pressure (BP) and Cerebral blood flow (CBF)

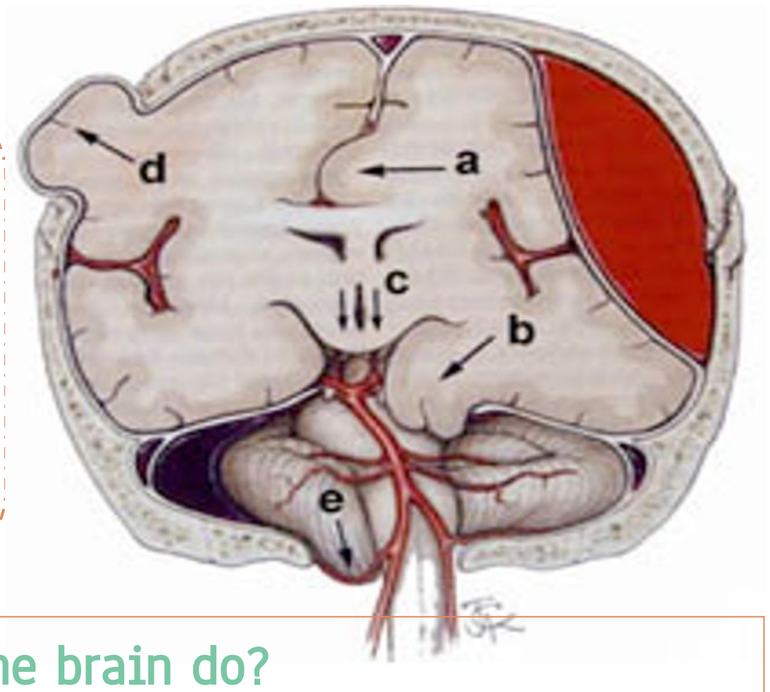
- If ICP goes up, how does the brain get perfusion?
- Process of autoregulation
- Cerebral Perfusion Pressure (CPP) is the difference between mean arterial pressure (MAP; pushing blood into the brain) and ICP (effectively resisting blood flow into the brain)
- CPP = MAP - ICP
- If: MAP=85 mmHg and ICP=15 mmHg then CCP will be = 70 (Normal range= 50-140 mmHg).
- If ICP increases, CPP will reduce unless the MAP increases. A reduction in CPP will reduce CBF unless the heart rate increases. There is, however, also a system of vascular autoregulation to maintain optimal CBF (60-140 mmHg).
- Increase in the perfusion causes increase in the blood flow until a certain point where the flow will stay the same; which is called the zone of normal autoregulation. In order to keep the blood flow from changing with the increase in pressure the cerebral arteries will keep constricting until they reach maximum construction where the pressure is the highest. And the blood flow will start increasing again.
- When the pressure is too low, maximum dilation will happen to increase the blood flow. So for example if someone is starving or dehydrated in a desert, their BP will decrease ⟶ cerebral vessels dilate to increase the blood flow to the brain ⟶ they'll be conscious and will try to look out for food and water.
- Blood vessel diameter is also controlled by arteriolar carbon dioxide concentration (PaCO2) increased PaCO2 causes vasodilation and increased CBF. Other compounds, such as nitric oxide and endothelin, also regulate local CBF.



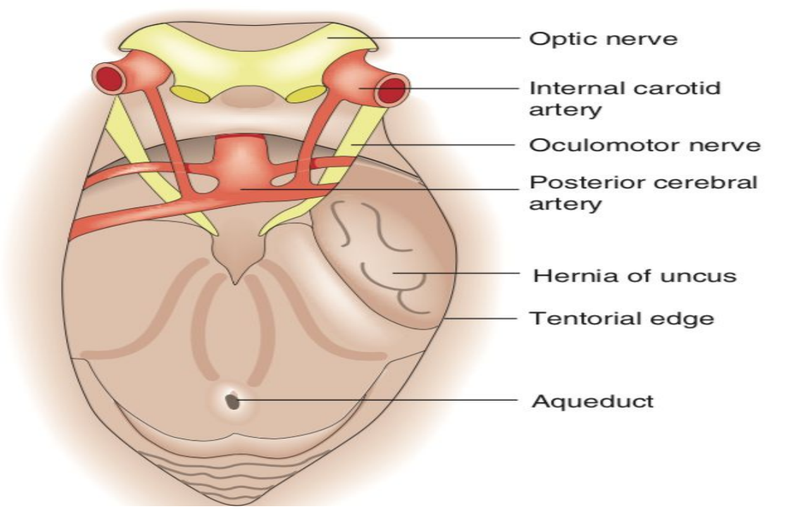
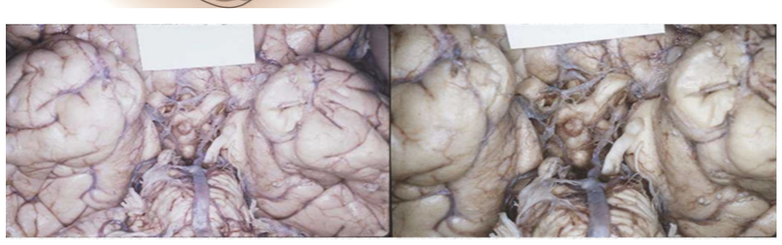
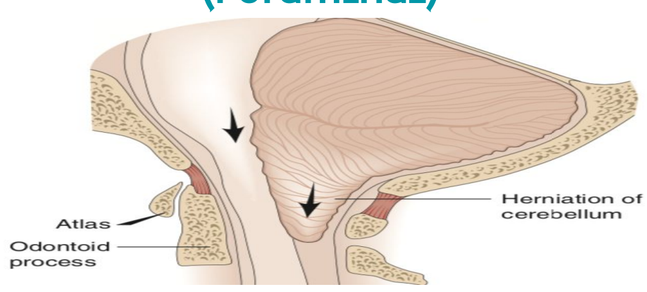
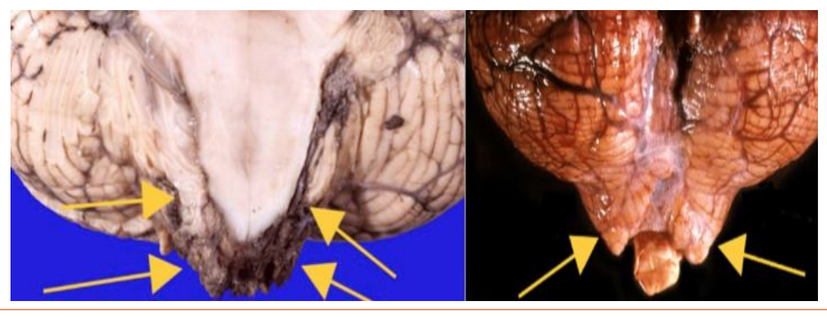
Brain Herniation Syndromes

Raised ICP and brain shift

- Generalised or localised increases in ICP can lead to marked dis- 24 placement (herniation) of intracranial structures causing blood vessel compression, further compromising brain perfusion.
- Because the head is composed of fluid we learned that they don't get compressed but shift from one compartment to another, just like someone changing rooms correct? This shift is what we call brain herniation.



When someone has epidural hematoma what would the brain do?

A	**Cingulate herniation (Subfalcine)**	With a parasagittal mass, the ipsilateral cingulate gyrus may herniate beneath the free edge of the falx to the other side. The principal clinical effect is deteriorating conscious level.
B	**Uncal herniation (Transtentorial)**  	With large ipsilateral brain lesions, common with epidural hematoma mesial (medial) part of the temporal lobe is called an uncus. Herniates over the edge of the tentorium to become wedged between the tentorial edge and the midbrain causing: 1. compression the brain stem → pressure on the ipsilateral 3rd cranial nerve (CN III) causing dilation of the pupil (if the lesion on the left it will cause a lift dilation and vise versa) 2. Compression on the midbrain causing contralateral weakness Clinical features: - Glasgow Coma Score (GCS) falls and the motor component may become asymmetrical - Ipsilateral pupil dilates and becomes nonreactive to light - Blood pressure rises, the pulse slows, the respiratory rate fall. (Cushing's response) - The patient becomes apnoeic.
C	**Central herniation**	Herninates toward the posterior fossa through the tentorial hiatus.
D	**Outside herniation (Transcalvarial)**	In case of a skull fracture
E	**Tonsillar herniation (Foraminal)**  	Can happen with posterior cranial fossa masses the cerebellar tonsils and medulla are displaced downwards through the foramen magnum Clinical features: Cerebellar impaction leads to compression of the medulla, leading to a dramatic decrease in the GCS, bilateral extensor responses (decerebration) and bilateral fixed dilated pupils. A Cushing's response and sudden respiratory arrest can follow; this is called 'coning' You can see that the tonsils are not round but thin pointed tip, the tonsils go down into the foramen magnum and compress the upper part of the cervical cord resulting in compromised breathing arrest because its where the respiratory centers are located.

False localization (Kernohan's notch phenomenon):

- It is a result of the compression of the cerebral peduncle, which is part of the mesencephalon, against the tentorium cerebelli due to transtentorial herniation.
- The patient will have right epidural hematoma, but instead of ipsilateral right dilated pupil and contralateral weakness, they will get a contralateral left dilated pupil and ipsilateral weakness.
- Because the pressure on the area of the midbrain & 3rd CN happen on the contralateral side of the mass not the ipsilateral side opposite to the tentorium and give the opposite clinical picture. This is why it's important to do a CT scan to identify the exact location.

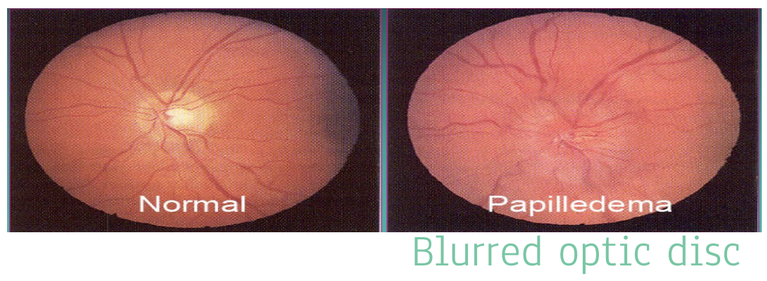
Raised Intracranial Pressure

Clinical Presentation:

★ Classic Triad: Headache, vomiting & papilloedema

➔ Headache (new onset of headache after 40 yo is suspicious & needs further imaging)
- Early morning (when lying down the venous return decrease resulting in increase ICP)
- Throbbing / Bursting
- ↑sneezing, coughing

➔ Papilloedema
- Reliable but may take several days
- Associated fundal hge indicates acute and severe rise in ICP



Blurred optic disc

★ A rapid increase in ICP may precipitate acute reduction in conscious level.

★ With more chronic, slow-growing lesions such as some brain tumours, abscesses or congenital abnormalities, extraordinary degrees of compensation can actually occur.

★ In some situations, even massive lesions can lead to minimal symptoms and signs, despite brain herniation.

★ Neurological signs of ↑ ICP:
- Ipsilateral pupillary dilation (due to compression of CN III)
- Contralateral hemiplegia (due to compression of crus cerebri on the midbrain)
- Cranial nerve deficit

★ Systemic signs of ↑ ICP:
- Raised BP (recall: CPP=MAP-ICP)
- Respiratory change:
  - Cheyne-Stokes breathing:
    - Oscillating periods of apnea-tachypnea
    - Respiratory centers compromise
- Cushing's triad: increase BP, bradycardia and low RR

Glascow Coma Scale (GCS)

Eye Opening (E)	Verbal Response (V)	Motor Response (M)
4= Spontaneous 3= To voice 2= To pain 1= None	5= Normal conversation (oriented) 4= Disoriented conversation (confused) 3= Inappropriate words 2= Incomprehensible sounds 1= None	6= Obeys commands (normal) 5= Localises pain 4= Withdrawals from pain (Flexion withdrawal) 3= Decorticate posture (abnormal flexion) 2= Decerebrate (abnormal extension) 1= None

★ We use this scale for measurements, following up with patients & for research.

★ Severe head injury = 3-8 (poor prognostic indicator)

★ Moderate head injury = 9-12

★ Mild head injury = 13 & 14

★ Normal = 15

★ The lowest GCS (close to death) = 3

Raised Intracranial Pressure

Cases

1) A 20 year old man. Had car accident (MVC) as unrestrained driver. He presented with BP 75/30 (low) & HR 125 bpm (high). He was unconscious, with right hemiplegia. **What is going on?** We want to increase his BP!
   Start first with **ABCDE**
   Airway: if it's not maintained like when the patient is unconscious or less than 8 GCS score → intubation
   Breathing: kills in minutes, so alway make sure that the chest movement, air reaching both lung, and normal O2 saturation
   Circulation: 2 large bores of IV line & IV fluid & give blood to raise the BP bc a comatose brain won't survive with a low pressure thus it needs blood.
   Disability: examine the pupils
   Exposure: expose everything and make sure there's no sign that was overlooked.
   And then send the patient for imaging to identify if there's a cranial hemorrhage or not.

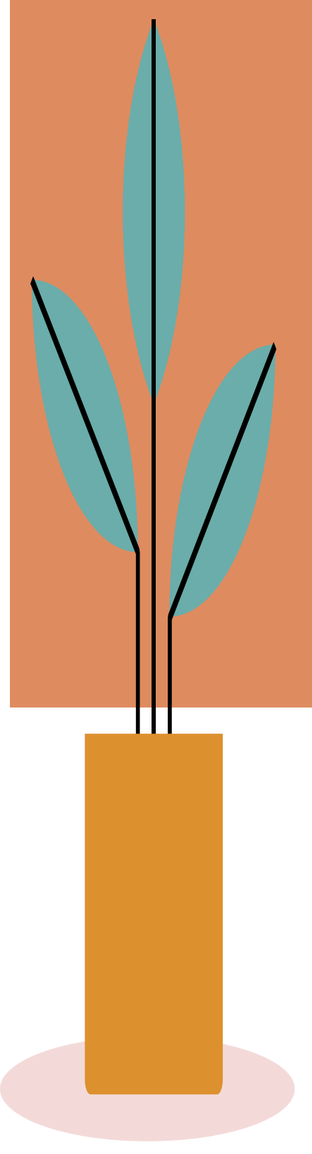
2) A patient comes to the ER with right hemiplegia & BP of 200/120, after CT, you notice that the patient is having a hemorrhagic stroke, **what's going on?** Start first with **ABCDE**
   We want to decrease his BP to 160 or 140 to not affect the cerebral perfusion. Because sometimes when the BP is high the brain will act as if there's a mass due to the high ICP → provide high MAP to overcome the ICP → causing higher BP (up to 220) which is very dangerous bc it can expand the hematoma or even cause more bleeding.

Possible causes:

- **VITAMEN D** (this has nothing to do with Vit. D it's just a mnemonic)
  - **V**ascular
  - **I**nfection
  - **T**rauma
  - **A**utoimmune
  - **M**etabolic
  - **E**ndocrine
  - **N**eoplastic
  - **D**rugs
- Others (rely on the anatomic structure of the brain)

Table 2 Examples of causes of raised intracranial pressure

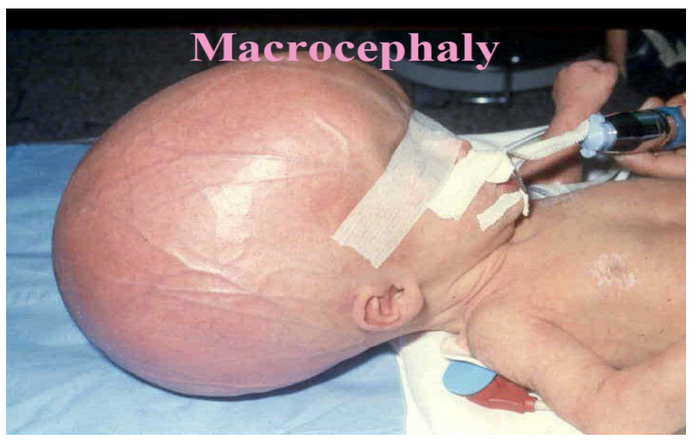
Pathological process	Examples
Localised mass lesions	Traumatic haematomas (extradural, subdural, intracerebral) Neoplasms (glioma, meningioma, metastasis) Abscess Focal oedema secondary to trauma, infarction, tumour
Disturbance of CSF circulation	Obstructive hydrocephalus Communicating hydrocephalus
Obstruction to major venous sinuses	Depressed fractures overlying major venous sinuses Cerebral venous thrombosis
Diffuse brain oedema or swelling	Encephalitis, meningitis, diffuse head injury, subarachnoid haemorrhage, Reye's syndrome, lead encephalopathy, water intoxication, near drowning
Idiopathic	Benign intracranial hypertension



Raised Intracranial Pressure

In Infants:

- ★ Widened sutures
- ★ Increased Head circumference (if you decrease the pressure the size will go back to normal)
- ★ Dilated head veins
- ★ Sunset" eyes (the eyes are looking down)
- ★ Bulging fontanelle



Investigations:

- ★ Ideal imaging for the brain is MRI but in trauma we need something quick, so **URGENT CT** for the head is the investigation of choice (it can also detect skull fractures).
- ★ **No lumbar puncture!** (Even if they present with signs & symptoms of meningitis) because when we take out the CSF for a sample the pressure in the spinal cord drops and due to high pressure with in the head, the brain will herniate to the area with less pressure through the foramen of magnum. غرفة مليانه وفتحنا الباب وش بيصير؟ كل شيء بيطيح ويحاول يطلع
  And the patient may die on the spot!

Treatment:

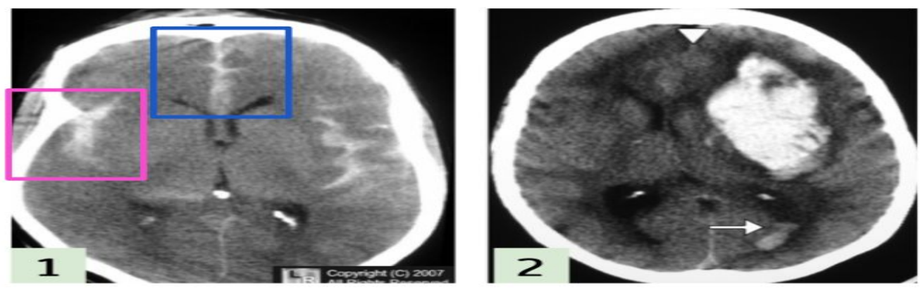
1. **General Measures** to reduce the ICP (we do all of these to patients with ICP if there is no contraindication)**:**

- ★ **Head elevation (30 degrees)** → to improve the venous return.
- ★ No neck compression (make sure collar is supporting but not tight or squeezing the neck).
- ★ **Mannitol** (osmotic diuretics) for patients who have decreased level of consciousness LOC (or Furosemide).
- ★ Steroids (Dexamethasone) for tumors only! If it's trauma steroids are **contraindicated**.
  - Steroids stabilize the membranes by stabilizing the junctions b/w the cells, and when those junctions are loose, it will result in edema which is exactly the case in tumors.
  - But with trauma edema is caused by cell death and lysis thus steroids aren't helpful!
- ★ Hyperventilation: controlled to PCO2 35-40 mmHg.
  - When you hyperventilate you wash out CO2 which is a potent vasodilator. So when it goes out we get vasoconstriction, but you want to wash it out to a certain degree because you don't want too much vasoconstriction which will cut off the blood supply. So you have to maintain the CO2 concentration within the normal range.
- ★ Sedation, muscle relaxants, hypothermia → lowers metabolic rate and demands.
- ★ Barbiturates: terminal option if everything fails (rarely done, if nothing works you can induce coma).

2. **Specific treatment:** depends on the cause (VITAMEN D)

Causes of Raised Intracranial Pressure

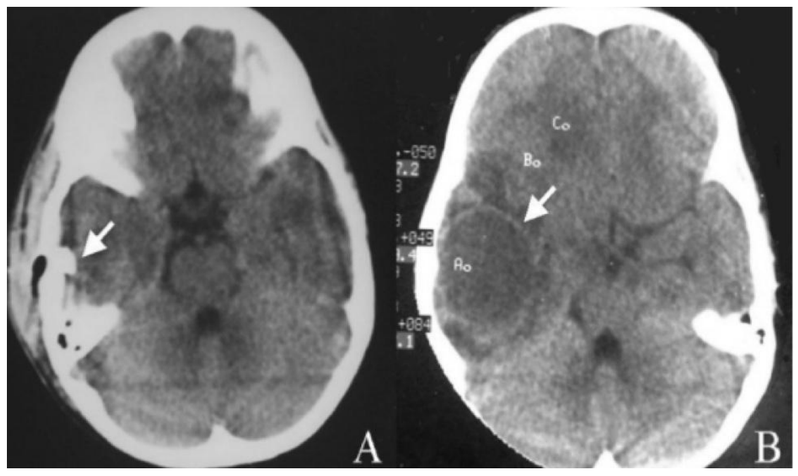
1- Vascular - SAH / ICH



1. Subarachnoid hemorrhage (the white is blood), showing sylvian fissure & interahemesphering bleeding
2. Large intracerebral hematoma with midline shift.

- Vascular causes need evacuation.

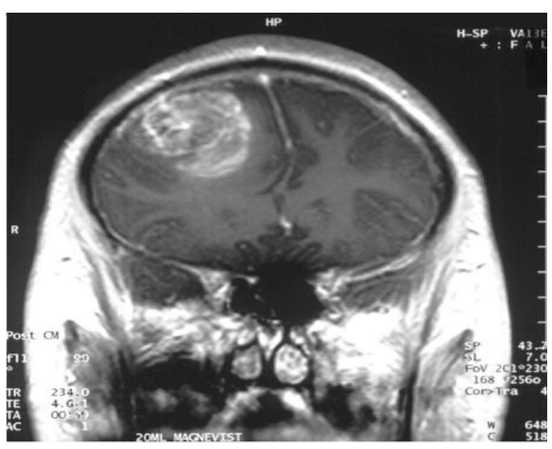
2- Infection / Abscess



- The arrow shows an abscess (dead necrotic tissue) and the wall enhances with contrast "ring enhancement".
- Abscess should be drained.

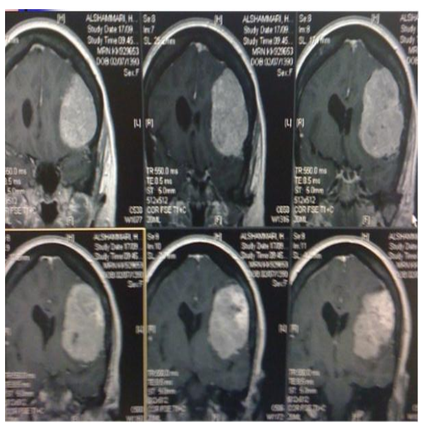
3- Tumors

Intrinsic brain tumors



Glioblastoma Multiforme & Astrocytoma

Extrinsic brain tumors



Meningioma

4- Hydrocephalus

Increase in the amount of fluid within the ventricles leading to pressure effect on the surrounding brain & edema around the cortices. Rounded tips ventricles & transependymal edema

The treatment is shunt to either drain the fluid to the abdomen or outside.

5- Trauma

A) Localized (focal)

Epidural (extradural) haemorrhage

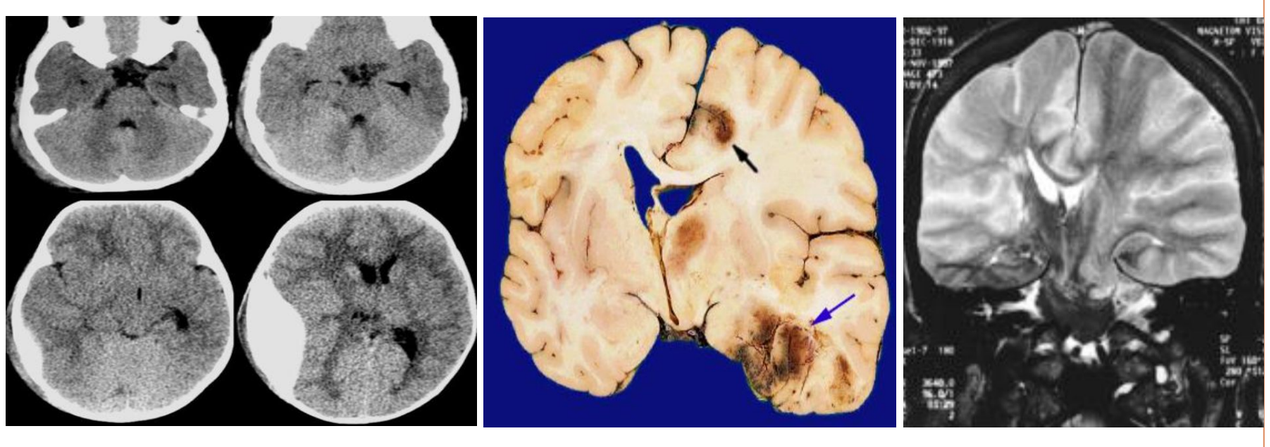
- Lenticular-shaped (biconvex) because the epidural space is a space that **doesn't exist**
- Skull fracture ⟶ injure the vessels ⟶ **bleeding creates the space** by pushing the dura until it hits the sutures where the dura is firmly attached to the periosteum of the bone, so it compresses the brain significantly.
- Most commonest vessel causing it is middle meningeal artery, bc it's grooved into the bone, easily injured & doesn't contract to stop the bleeding
- Has good outcome b/c the brain is normal.
- **Treatment:** the pressure must be reduced ⟶ craniotomy with clot evacuation

Subdural haemorrhage

- It's a potential space which means it's a real space that is already there, and if there is bleeding it will go to all around the brain.
- Common cause: brain got shaking results in brain base laceration or bridge vein laceration, so it's end up with bleeding around the brain.
- Treatment: the pressure must be reduced ⟶ craniotomy with clot evacuation

B) Diffuse (closed head injury)

- Here there is diffuse axonal injury.
- The nerves in the white matter are subjected to trauma when there is acceleration-deceleration (brain shaking). When the fibers are injured we see them as petechial hemorrhages "salt and pepper hemorrhage" with no midline shift, good ventricles ICP monitoring is useful.

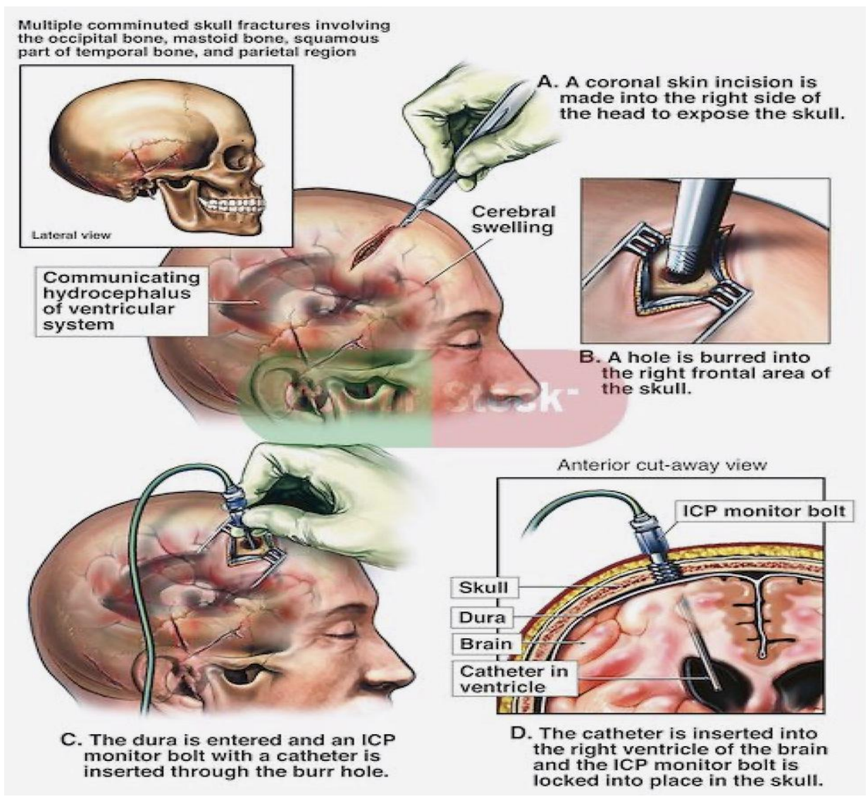


Epidural haemorrhage causing midline shift

Subdural haemorrhage

Monitoring Intracranial Pressure:

★ Can we monitor ICP? Yes.
★ Should we monitor all patients? No it depends on the case:
We monitor a patient with petechial hemorrhage (diffuse injury) to know the exact intracranial pressure. BUT a patient with epidural hematoma, for example, doesn't need monitoring; by the time you put a catheter in he will be dead. He needs head elevation, mannitol, hyperventilation and surgery (to evacuate the hematoma).



Surgical Recall:

What is the Glasgow Coma Scale (GCS)? An objective assessment of the level of consciousness after trauma.
What indicates coma by GCS score? <8 (Think: "less than eight—it may be too late").
What does unilateral, dilated, nonreactive pupil suggest 'blown pupil'?
- Focal mass lesion with ipsilateral herniation and compression of CN III.

What do bilateral fixed and dilated pupils suggest? Diffusely increased ICP.
What is the initial radiographic neuroimaging in trauma?
1. Head CT scan (if LOC or GCS < 15) 2. C-spine CT scan 3. T/L spine AP and lateral.

Should the trauma head CT scan be with or without IV contrast? Without!
What is normal ICP? 5 to 15 mm H2O
What is the worrisome ICP? > 20 mm H2O.
What determines ICP (Monroe-Kelly hypothesis)? Volume of brain, blood, & CSF.
What is the CPP? Cerebral Perfusion Pressure = mean arterial pressure−ICP (normal CPP is > 70).
What is Cushing's reflex? Physiologic response to increased ICP:
1. Hypertension 2. Bradycardia 3. Decreased RR (abnormal breathing).

What are the three general indications to monitor ICP after trauma?
1. GCS <9
2. Altered level of consciousness or unconsciousness with multiple system trauma.
3. Decreased consciousness with focal neurologic examination abnormality.

What non operative techniques are used to decrease ICP?
1. Elevate head of bed (HOB) 30 degrees (if spine cleared).
2. Diuresis-mannitol (osmotic diuretic), Lasix®, limit fluids.
3. Intubation (PCO2 control).
4. Sedation.
5. Pharmacologic paralysis.
6. Ventriculostomy (CSF drainage).

How does hyperventilation decrease ICP?
By decreasing Pco2 resulting in cerebral vasoconstriction (and thus less intracranial volume)
What is the acronym for the treatment of elevated ICP? "ICP HEAD":
- **I**NTUBATE, **C**ALM (sedate), **P**LACE DRAIN (ventriculostomy)/ **P**ARALYSIS, **E**LEVATE head, **D**IURETIC (e.g., mannitol).
- **H**YPERVENTILATE TO PCO2 approximately 35 ≈ , & **A**DEQUATE BLOOD PRESSURE (CPP> 70)

Can a tight c-collar increase the ICP? Yes (it blocks venous drainage from brain!).
Why is prolonged hyperventilation dangerous?
It may result in severe vasoconstriction and ischemic brain necrosis!. Use only for very brief periods.

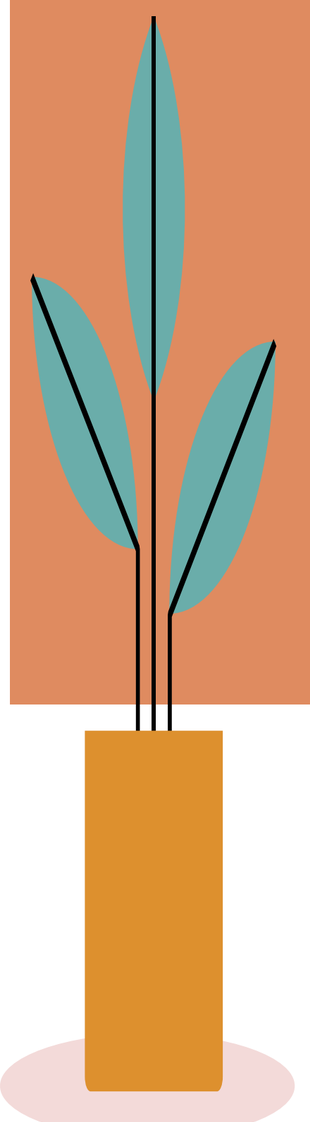
Special thanks to team 436 <3

Summary

- ★ The pressure in the skull is called the Intracranial pressure (ICP).
- ★ The rigid bony framework enclosing the central nervous system means that any increase in mass content increases intracranial pressure (ICP).
- ★ The ICP must stay balanced in order for the brain to survive.
- ★ Cerebral autoregulation is the ability of cerebral vessels to maintain cerebral perfusion within strictly determined limits.
- ★ Cerebral perfusion pressure (CPP) = Mean Arterial Pressure (MAP) - Intracranial pressure (ICP)
- ★ Acute increases in ICP lower perfusion pressure and, if unrelieved, lead progressively to decreased coma score, herniation syndromes, hypertension, and respiratory abnormalities Types of brain herniations :
  - ○ Cingulate herniation
  - ○ Uncal herniation (common due to epidural hematoma)
  - ○ Central herniation
  - ○ Outside herniation
  - ○ Tonsillar herniation (very common complication of LP in pt with high ICP)
- ★ The principal symptoms of raised ICP are headache (early morning and throbbing), vomiting and papilloedema.
- ★ They may also have neurological deficits such as: hemiplegia, pupillary dilation, and cranial nerve deficit.
- ★ Signs: hypertension, bradycardia, lower respiratory rate (cushing's triad).
- ★ Investigation: CT scan of the head! No LP
- ★ Treatment of high Intracranial Pressure
  - ★ General:
    - ○ Head elevation 30 degrees
    - ○ No neck compression
    - ○ Mannitol for patients with decreased LOC
    - ○ Steroids for tumors
    - ○ Hyperventilation : controlled to PCO2 35-40 mmHg.
    - ○ Sedation, muscle relaxants
    - ○ Hypothermia.
    - ○ Barbiturates: terminal option if everything fails.
  - ★ Specific treatment depending on the cause; VITAMEN D.

Glascow Coma Scale (GCS)

Eye Opening (E)	Verbal Response (V)	Motor Response (M)
4= Spontaneous	5= Normal conversation (oriented)	6= Obeys commands (normal)
3= To voice	4= Disoriented conversation (confused)	5= Localises pain
2= To pain	3= Inappropriate words	4= Withdrawals from pain
1= None	2= Incomprehensible sounds	3= Decorticate posture (abnormal flexion)
	1= None	2= Decerebrate (abnormal extension)
		1= None



Quiz

1. A 28-year-old male motorcyclist is brought into the emergency department where you, as the doctor on call, are asked to assess his consciousness as part of the primary survey. He is unresponsive when you speak to him but, on rubbing his sternum, opens his eyes and tries to push your hand away. During your assessment he also mutters something that you are unable to understand. His Glasgow Coma Scale score is:

    A. 7
    B. 8
    C. 9
    D. 10
    E. 11

2. A 20-year-old university cricketer is hit in the side of his head by a cricket ball while fielding close to the bat during a varsity match. He is reported to have briefly lost consciousness but at the time of examination in the emergency department his Glasgow Coma Scale score is 15/15. In addition, he complains of severe headache and blurred vision since regaining consciousness. A neurological examination reveals that his right pupil is dilated but no other focal neurology is elicited. A CT scan is performed on the basis of these findings, which shows a lenticular-shaped collection suggestive of an extradural haematoma. Damage to which one of the following vessels would classically be associated with this pathology?

    A. Maxillary artery
    B. Middle meningeal artery
    C. Cerebral veins
    D. Middle cerebral artery
    E. Anterior communicating artery

3. An 85-year-old pensioner is brought to the emergency department by her family who are concerned that she has become increasingly confused and drowsy in the past 3 weeks. She is pleasantly confused and unable to recall events clearly but oriented to time and person and complains only of occasional frontal headache. Her family inform you that she may have fallen while climbing from the bathtub some weeks previously. She has also started sleeping for long periods of time, which is not her normal habit. A head CT scan is performed, which shows mild generalized atrophy and a crescent-shaped collection. This presentation is consistent with:

    A. Intracerebral haemorrhage
    B. Subarachnoid haemorrhage
    C. Extradural haemorrhage
    D. Subdural haemorrhage
    E. None of the above

Answers:
1:C
2:B
3:D