Intracranial pressure

Presented by

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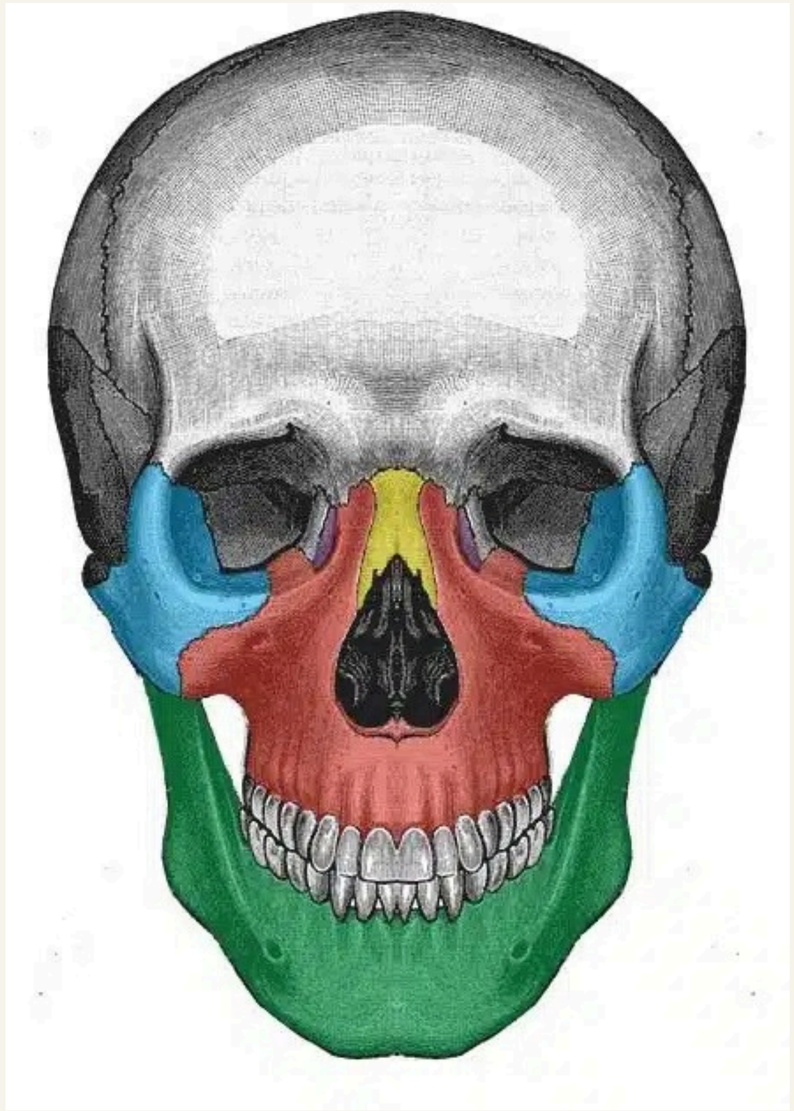


- **Skull:** 1) cranium(cranial bones)
  2)facial bones

- The **cranium** (or neurocranium) describes the **part of the skull** encasing the brain, made up of **8 bones** (frontal, ethmoid, sphenoid, occipital, paired parietals, and paired temporal)

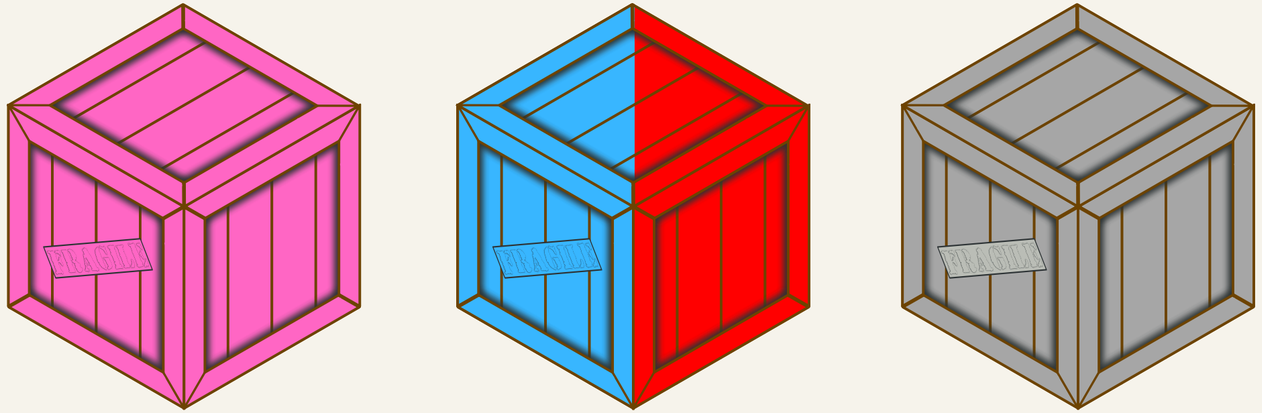
ICP principles (Model)

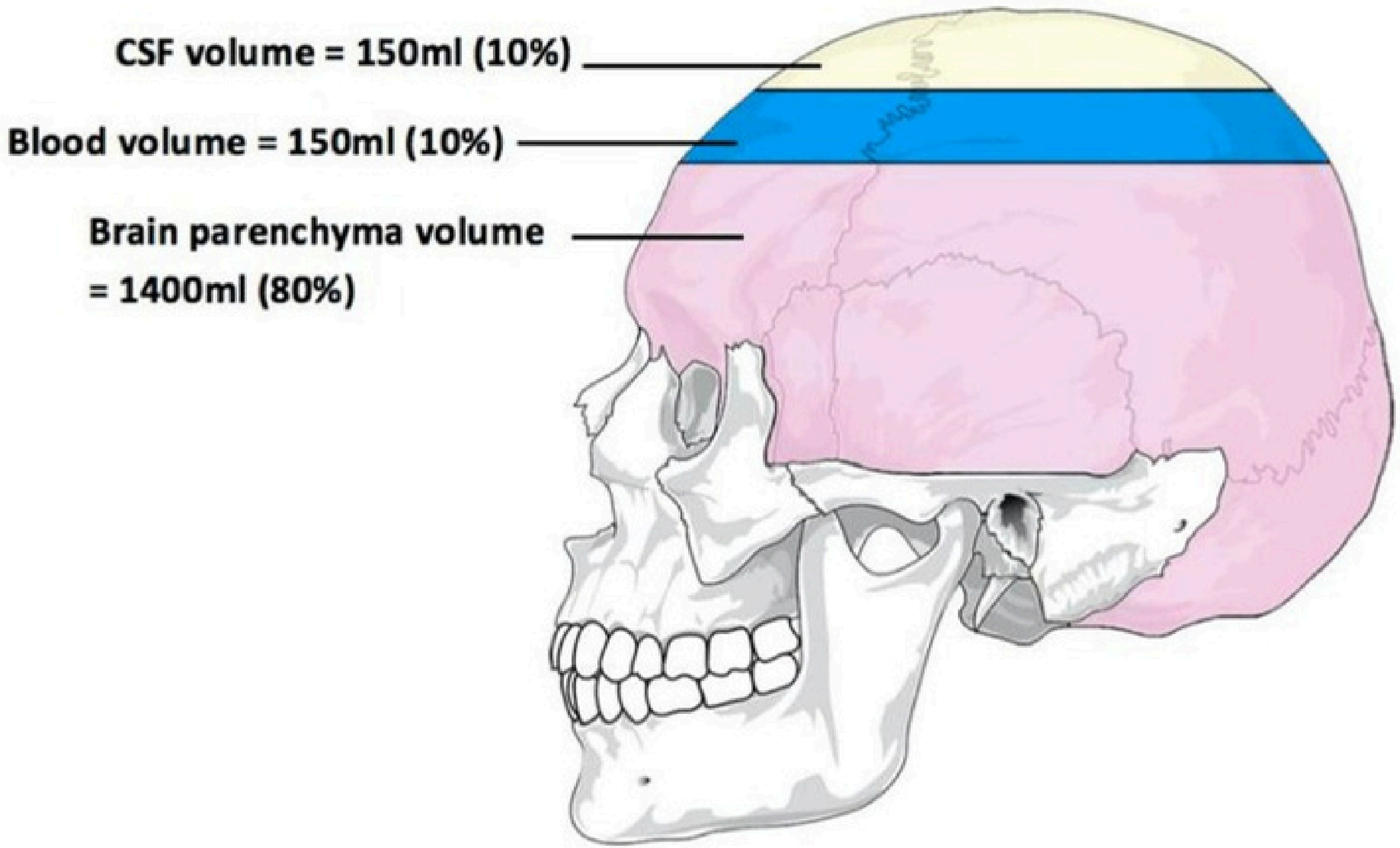
normal intracranial constituents(and approximate volumes):

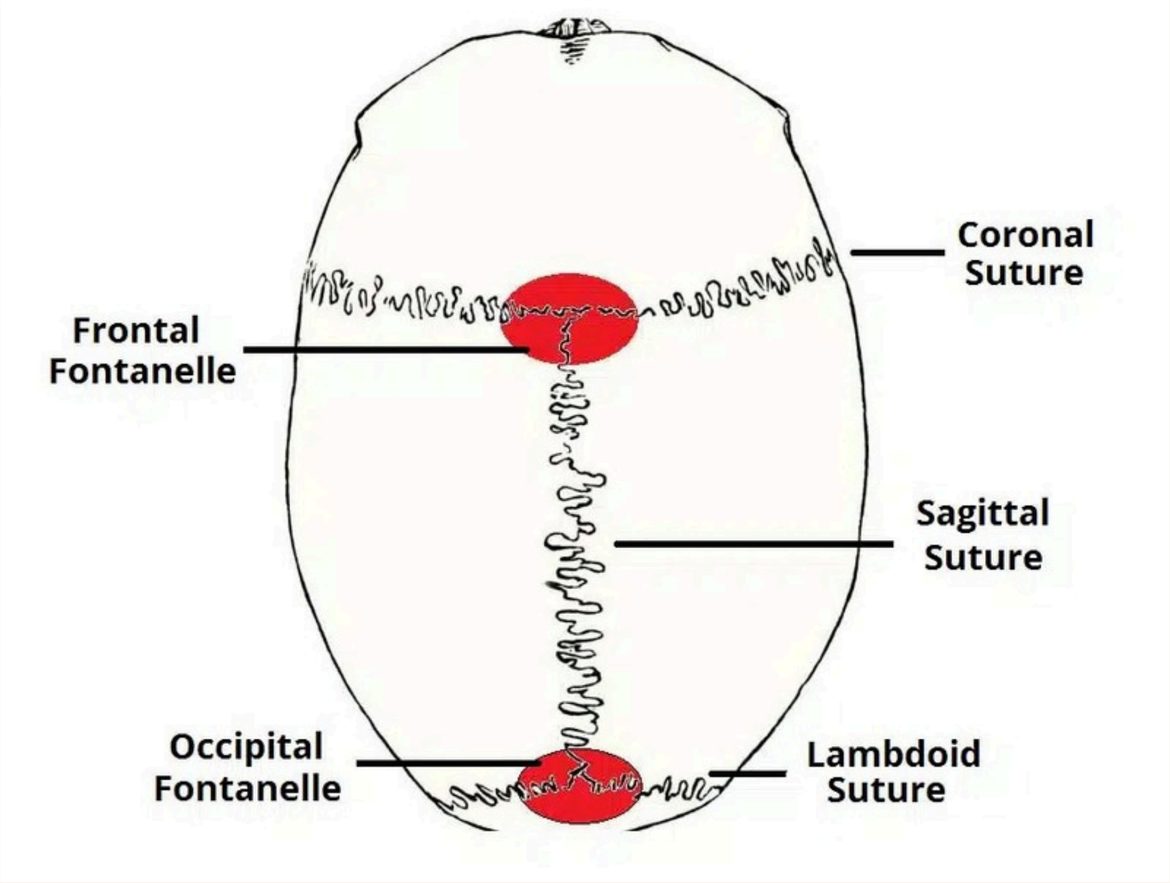
1) brain parenchyma (which also contains extracellular fluid): 1400 ml
2) cerebral blood volume (CBV): 150 ml
3) cerebrospinal fluid (CSF): 150 ml

- **Total intracranial volume =1700 ml**

- these volumes are contained in an **inelastic and fixed** container (the skull)




CSF volume = 150ml (10%)
Blood volume = 150ml (10%)
Brain parenchyma volume
= 1400ml (80%)



- Presence of fontanelles and open suture lines in **infants** that have not yet fused means there is potential for a **change in size and intracranial volume**

Normal ICP

- adults and older children =**7-15 mmHg**
- young children =**3-7 mmHg**

- **pressure is distributed evenly throughout the intracranial cavity**

- **Cerebral blood flow (CBF):** the volume of blood (in mL) supplied to 100 g of brain tissue per minute
- **CBF=CPP- CVR**

- **Cerebral perfusion pressure (CPP):** the effective pressure that delivers blood to the brain and is responsible for constant perfusion of brain tissue
- **CPP = MAP - ICP**

- **High ICP >> Low CPP >> Low CBF >> decreased tissue oxygenation**

ICP principle
(Modified Monro-Kellie doctrine)

- States that the sum of the **intracranial volumes** (CBV, brain, CSF) is **constant** , which means that an increase in one component volume will be compensated for by a decrease in other(s)

- If the pressure from one intracranial constituent increases (as when that **component increases in volume**),it causes the pressure inside the skull **(ICP) to increase**

- The craniospinal axis can **buffer** small increases in volume with no change or only a slight increase in ICP.If the expansion continues,then the new equilibrium will be at the higher ICP.
- **How?**

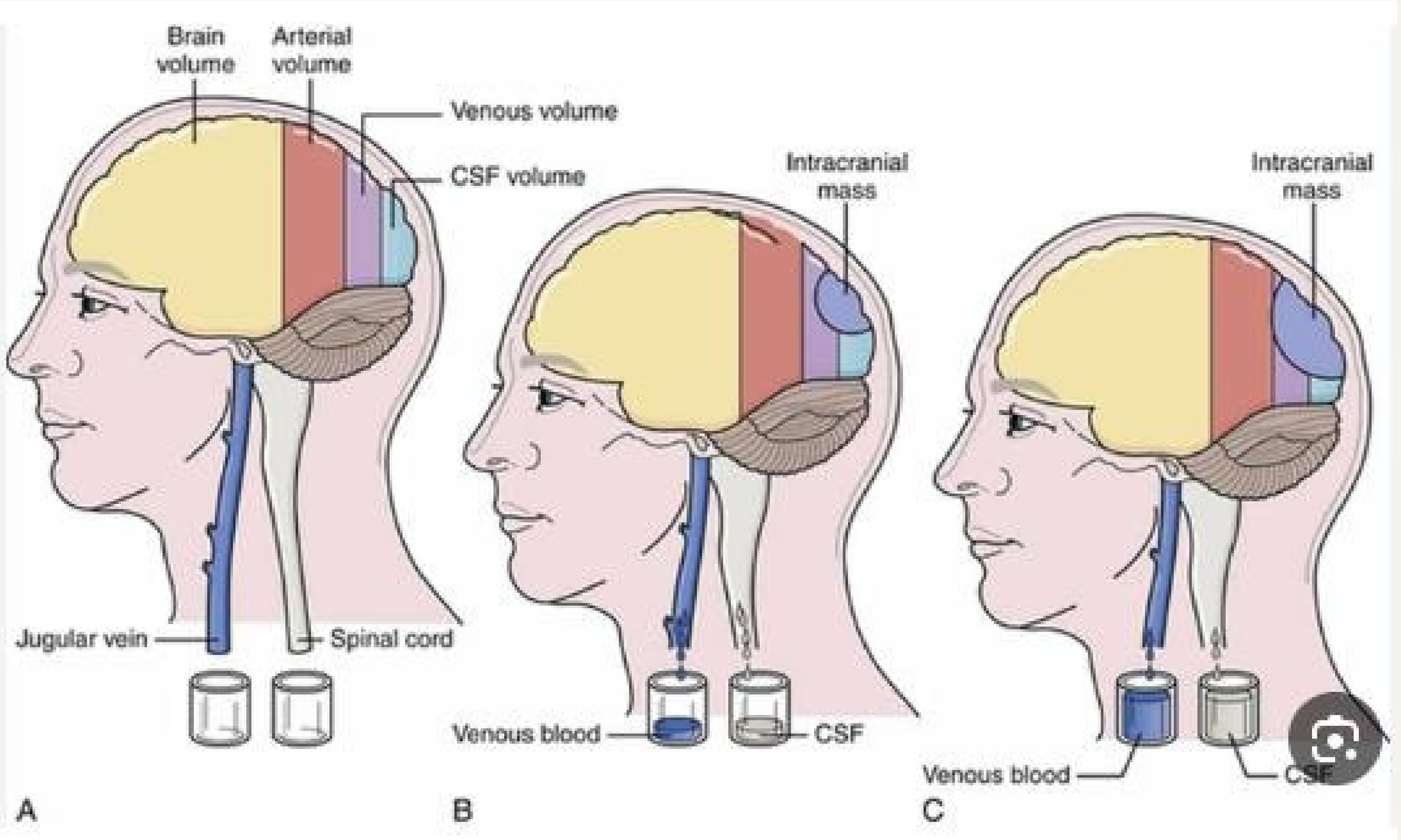
1) at pressures slightly above normal, if there is no obstruction to CSF flow (obstructive hydrocephalus), **CSF can be displaced from the ventricles and subarachnoid spaces and exit the intracranial compartment via the Foramen Magnum** (shifting into spinal canal)

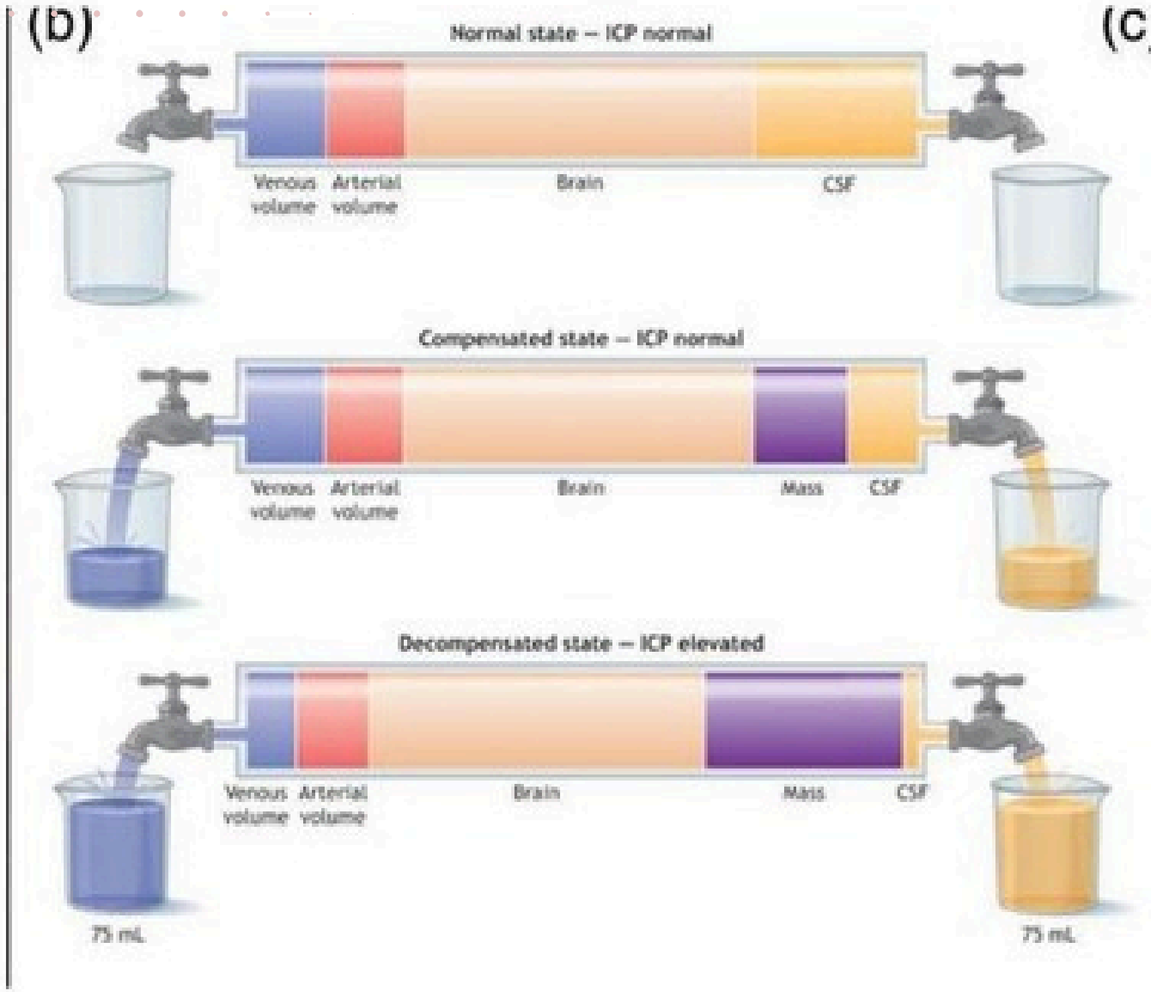
2)**venous blood can also be displaced through the jugular foramina via the IJVs (Internal Jugular Veins)**

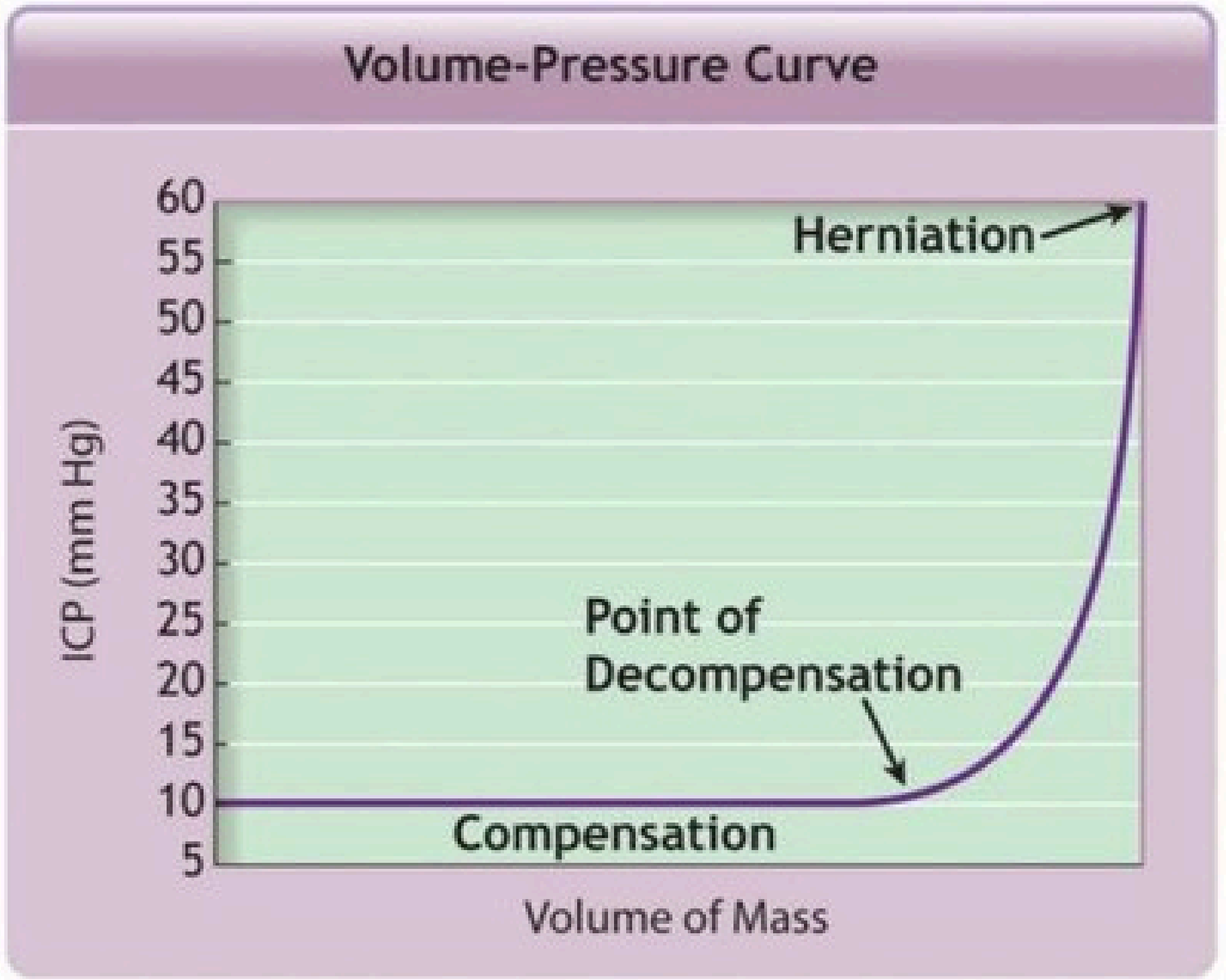
3)As pressure continues to rise ,**arterial blood** is displaced and CPP decrease eventually producing diffuse cerebral ischemia

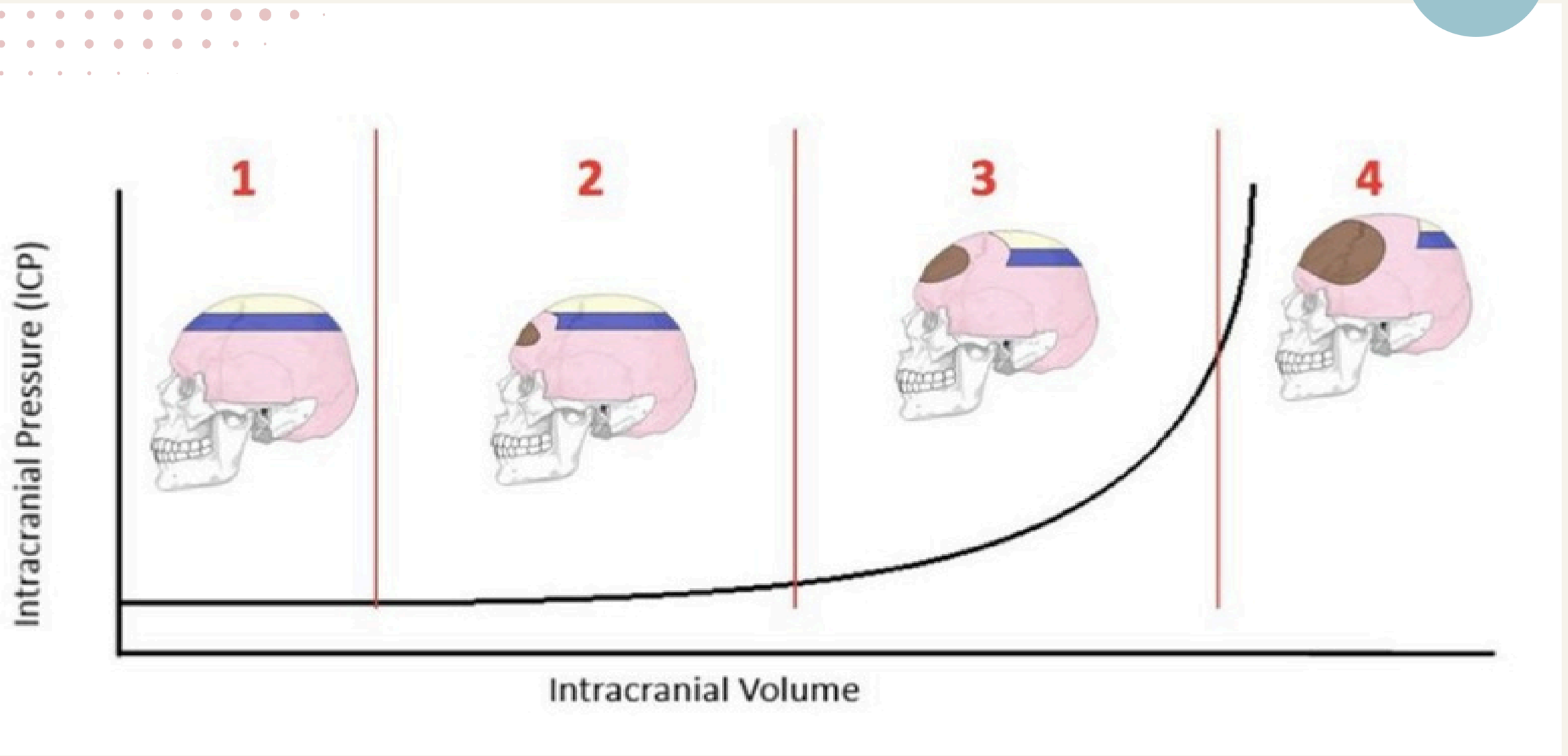
At pressures equal to mean arterial pressure, arterial blood will be unable to enter the skull through the FM, producing complete cessation of blood flow to the brain, with resultant massive infarction

4)Increased brain edema, or an expanding mass (e.g. hematoma) can push **brain parenchyma** downward into the foramen magnum (**cerebral herniation**) although brain tissue cannot actually exit the skull


Brain volume
Arterial volume
Venous volume
CSF volume
Jugular vein
Spinal cord
A
Intracranial mass
Venous blood
CSF
B
Intracranial mass
Venous blood
CSF
C


(D)
Normal state – ICP normal
Venous volume
Arterial volume
Brain
CSF
Compensated state – ICP normal
Venous volume
Arterial volume
Brain
Mass
CSF
Decompensated state – ICP elevated
Venous volume
Arterial volume
Brain
Mass
CSF
75 mL
75 mL

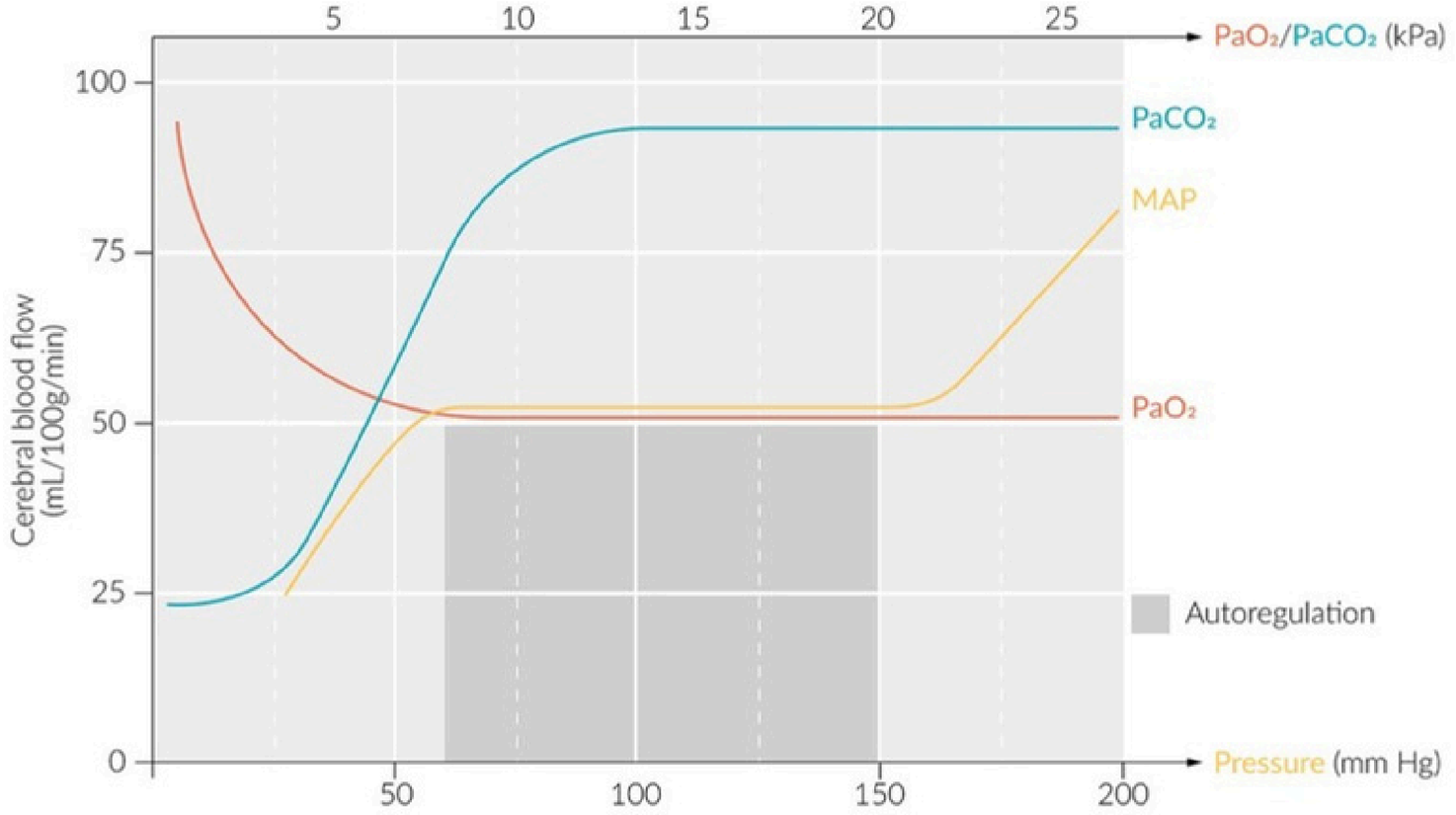

(C)
Volume-Pressure Curve
ICP (mm Hg)
60
55
50
45
40
35
30
25
20
15
10
5
Herniation
Point of Decompensation
Compensation
Volume of Mass

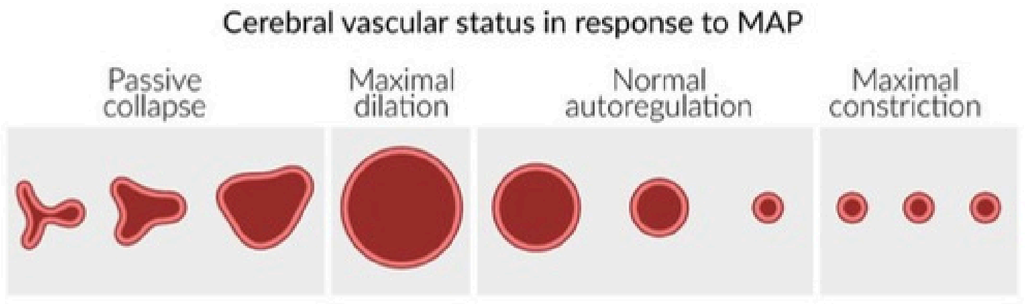

1
2
3
4
Intracranial Pressure (ICP)
Intracranial Volume

Autoregulation

- Normal adult CPP is > 50mm Hg
- Cerebral autoregulation is a mechanism whereby over a wide range, large changes in systemic BP produce only small changes in CBF
- Autoregulation is controlled by changing Cerebral vascular resistance (CVR) in response to changes in systemic blood pressure
- Due to autoregulation, CPP would have to drop below 40 in a normal brain before CBF would be impaired

Autoregulation





Intracranial Hypertension (Increased ICP) Causes

1. cerebral edema

2. hyperemia: the normal response to head injury Possibly due to vasomotor paralysis (loss of cerebral autoregulation). May be more significant than edema in raising ICP

3. traumatically induced masses

a)epidural hematoma
b)subdural hematoma
c)intraparenchymal hemorrhage (hemorrhagic contusion)
d)foreign body (e.g. bullet)
e)depressed skull fracture

4. hydrocephalus due to obstruction of CSF absorption or circulation

5. hypoventilation (causing hypercarbia → vasodilatation)

6. systemic hypertension (HTN)

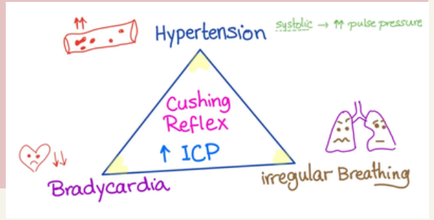
7. venous sinus thrombosis

8. increased muscle tone and Valsalva maneuver as a result of agitation orposturing→ increased intro thoracic pressure→ increased jugular venous pressure →reduced venous outflow from head

9. sustained posttraumatic seizures (status epilepticus)

Clinical presentation of intracranial hypertension

- Cushing’s triad: hypertension, bradycardia, respiratory irregularity



- Herniation syndromes (in compartmental increased pressure)

- Decreased level of consciousness: obtundation, coma

- Cranial nerve III palsy in subfalcine (uncal herniation)
- Fixed dilated pupil

Seen in unilateral cerebral mass

Indications for ICP monitoring in head trauma

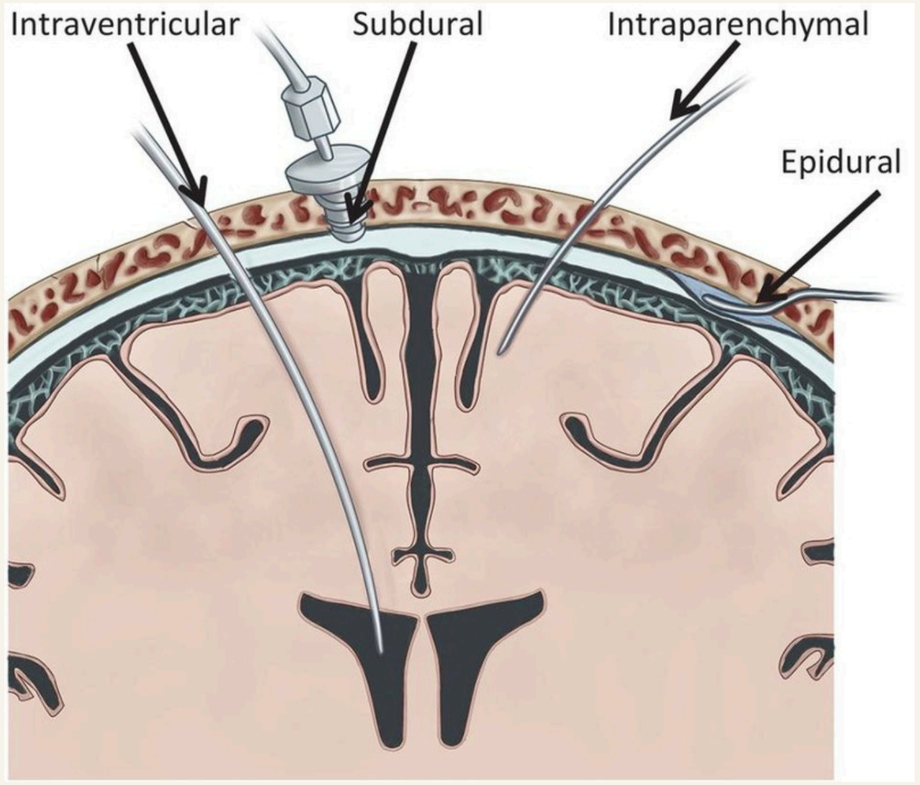
- For salvageable patients with severe traumatic brain injury (GCS ≤ 8 after cardiopulmonary resuscitation
- with an abnormal admitting brain CT (note: abnormal" CT: demonstrates hematomas (EDH,SDHorICH), contusions,compression of basal cisterns,herniation,or swelling
- with a normal admitting brain CT, but with ≥ 2 of the risk factors for IC-HTN
  1. age > 40yrs
  2. SBP < 90mm Hg
  3. decerebrate or decorticate posturing on motor exam (unilateral or bilateral)

Contraindication for ICP monitoring

(relative)

1. “awake” patient: monitor usually not necessary, can follow neuro exam
2. coagulopathy

Types of ICP monitor



- intraventricular catheter (IVC)
- intraparenchymal monitor
- subarachnoid screw (bolt)
- Subdural catheter
- Epidural monitor
- In infants: palpate fontanel

Peak of IC-HTN in trauma

day 2–3

day 9–11

Treatment

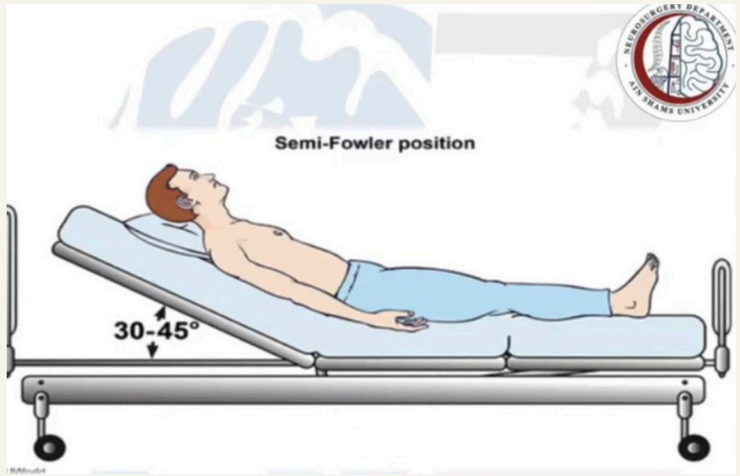
- the need for treatment should be based on ICP in combination with clinical examination & brain CT findings
- treatment for IC-HTN should be initiated for ICP > 22cm H2O
- Avoid CPP < 50mm Hg
- Avoid increasing CPP more than 70mmHg

Treatment: Goals of therapy

- keep ICP ≤ 22mm Hg
- keep CPP ≥ 50mm Hg

Treatment

(initial)



- elevate HOB to 30–45°: ↓ ICP by enhancing venous outflow
- keep neck straight, avoid neck constrictions (tight trach tape, tight cervicalcollar…):constriction of jugular venous outflow causes ↑ ICP
- avoid arterial hypotension (SBP < 90mm Hg)
- control hypertension if present

Treatment
(initial)

- avoid hypoxia (PaO_2 < 60mm Hg or O_2 sat < 90%)
- ventilate to normocarbia ($PaCO_2$ = 35–40mm Hg)
- light sedation: e.g. codeine
- controversial: prophylactic hypothermia: Hypothermia →↓$CMRO_2$
- unenhanced head CT scan for ICP problems: rule out surgical condition

Treatment:

(advanced)

- heavy sedation: fentanyl
- drain 3–5 ml CSF if IVC (intraventricular catheter) present
- hyperventilate to $PaCO_2$ = 30–35mm Hg
- mannitol 0.25–1 gm/kg
- 10–20 ml of 23.4%
- hypertonic saline (HS)
- Augmented hyperventilation to ↓$PaCO_2$ to 25–30mm Hg

Treatment
surgical

- traumatic intracranial masses should be treated as indicated
- decompressive craniectomy

$PaCO_2$ (mm Hg)	Description
35–40	normocarbia. Use routinely
30–35	hyperventilation. Do not use prophylactically. Use only as follows: briefly for clinical evidence of IC-HTN (neurologic deterioration) or chronically for documented IC-HTN unresponsive to other measures
25–30	augmented hyperventilation. A second tier treatment. Use only when other methods fail tc control IC-HTN. Additional monitoring recommended to R/O cerebral ischemia
<25	aggressive hyperventilation. No documented benefit. Significant potential for ischemia

THANK YOU