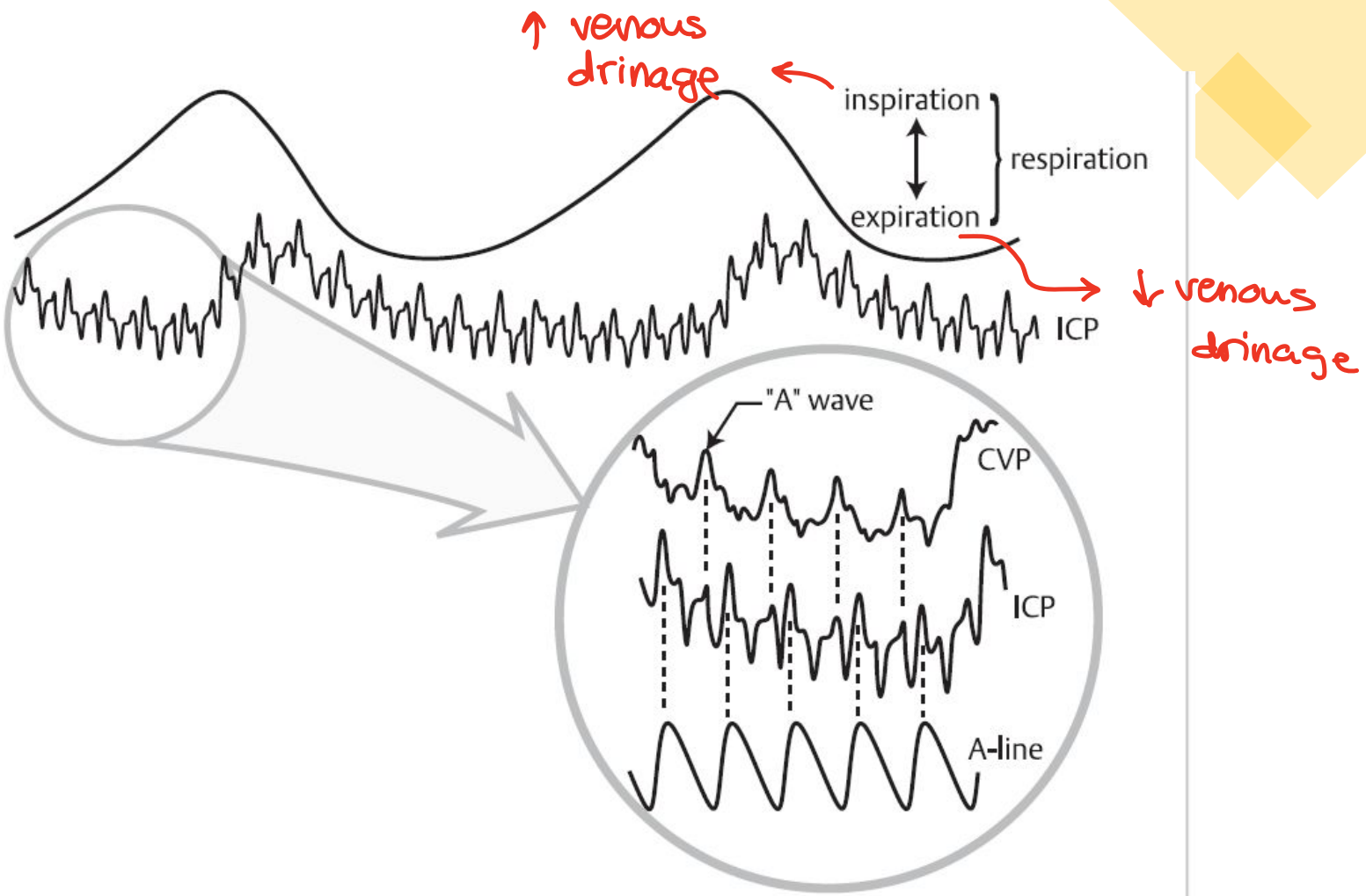
Intracranial pressure


↑ venous drinage
inspiration
respiration
expiration
↓ venous drinage
ICP
"A" wave
CVP
ICP
A-line

Why? Intracranial pressure

* اي Flow بتعتمد على

- Cerebral oxygenation depends on cerebral blood flow ✓ resistance + pressure ال
- Cerebral blood flow (CBF) = **Cerebral perfusion pressure (CPP)** ✓
  Cerebral vascular resistance (CVR) → اللي بتتحكم فيها هو: vasodilation / vasoconstriction
- Cerebral perfusion pressure ✓
  = Mean arterial pressure (MAP) – **Intracranial pressure (ICP)**

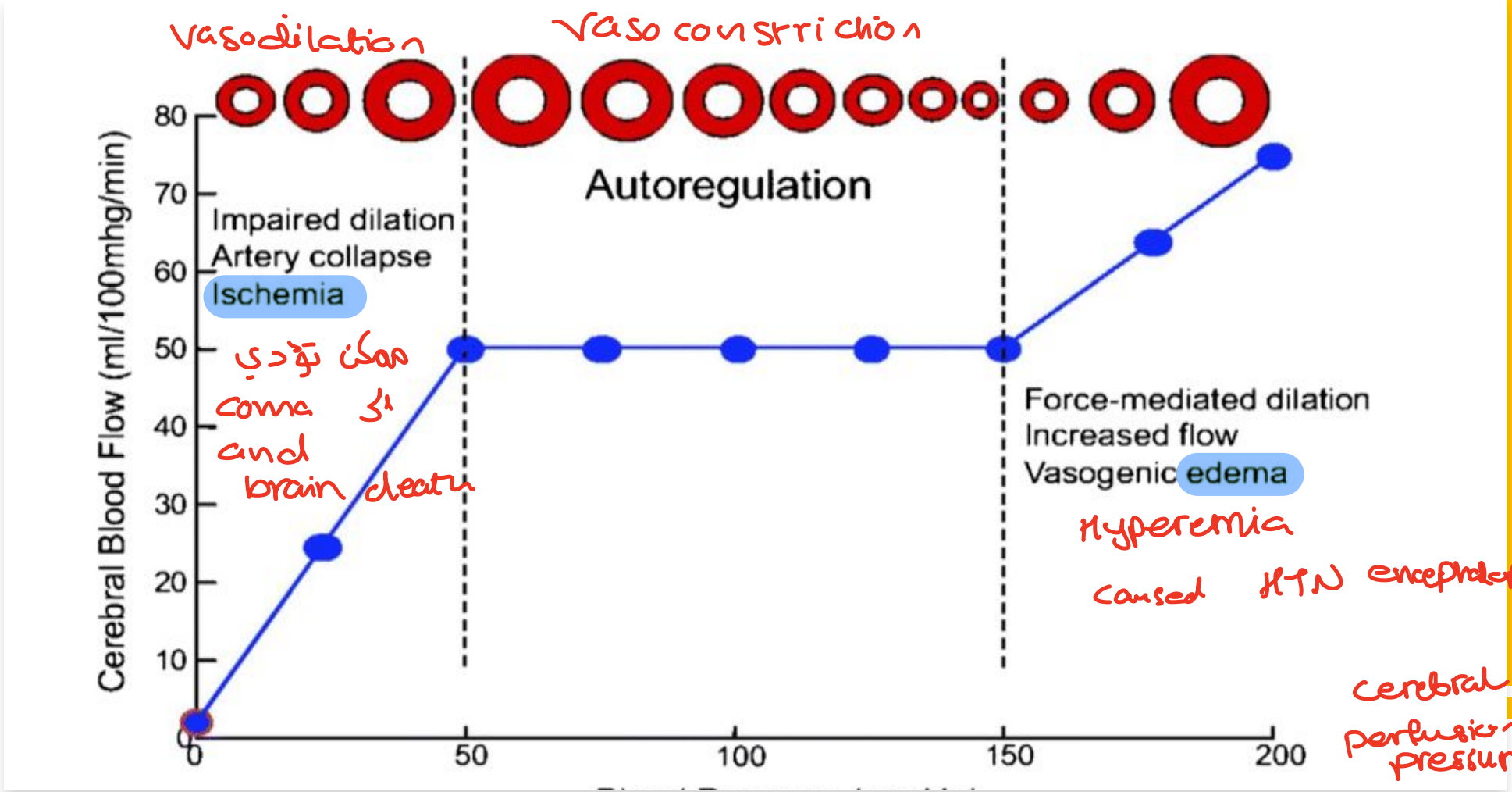
* لو ICP ارتفع ال CPP بالتالي CBF ↓ و بقل ال brain oxygenation و بجيب brain ischemia ← which can lead to brain death.

- In summary:
  High ICP >> Low CPP >> Low CBF >> decreased tissue oxygenation

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

in pysiological condition



ممكن تؤدي coma لـ and brain death

Hyperemia caused HTN encephalopathy

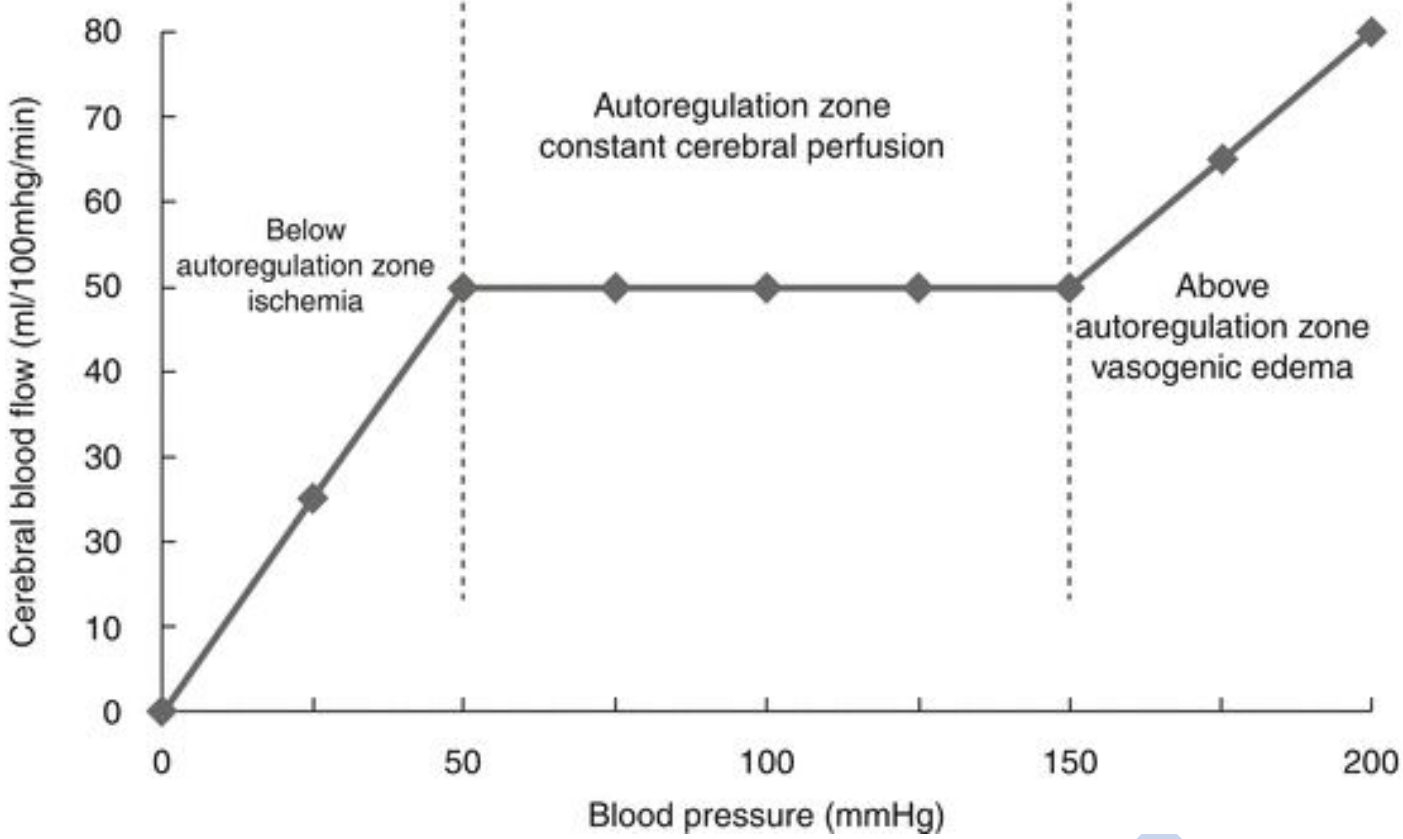
cerebral perfusion pressure

CPP between 50-150, CBF → constant by vasoconstriction

* في pathological condition ← ممكن تكون بين 50-70

ex: head trauma ممكن تأدي لـ brain edema

خلوا بالكم عن الـ 70 ممكن تأدي لـ brain edema.



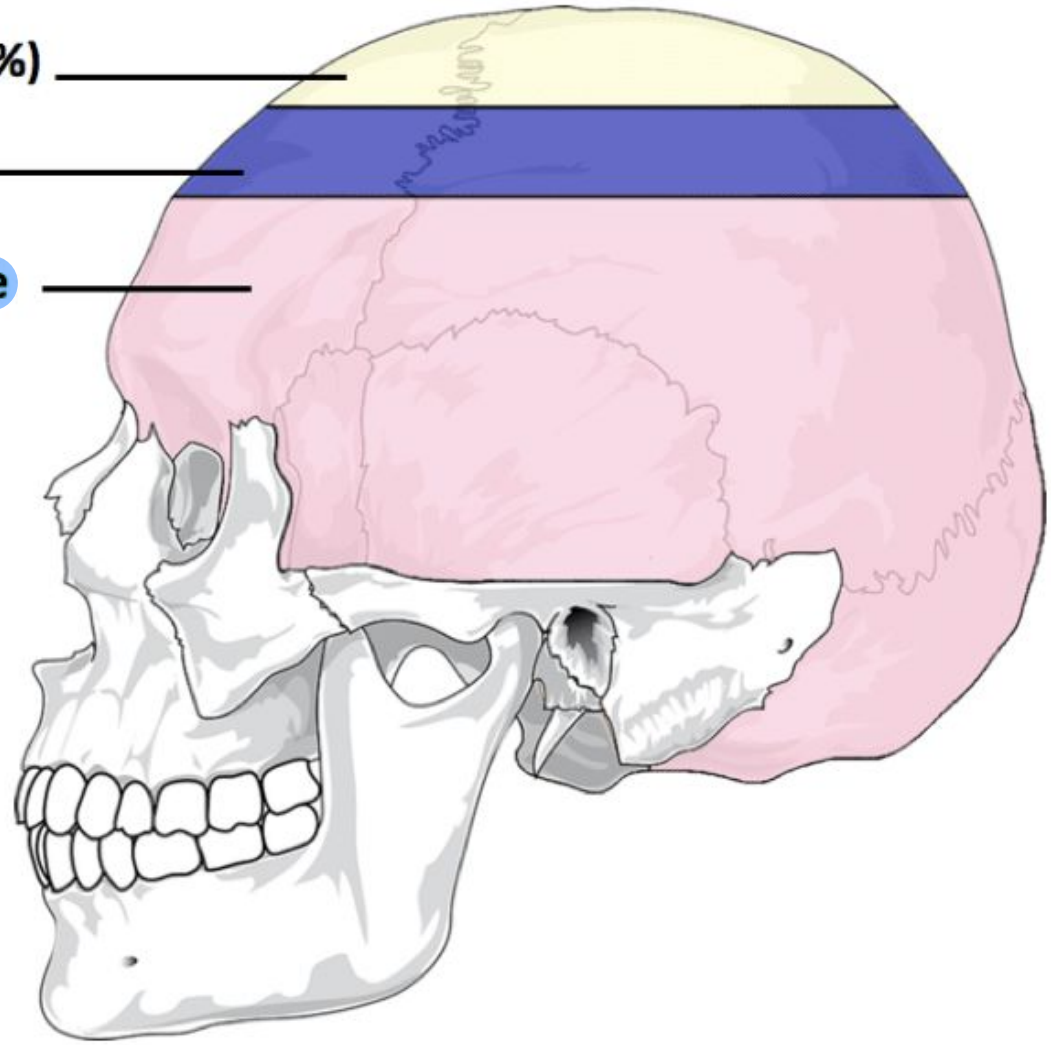
ICP principles (Model)

- normal intracranial constituents (and approximate volumes):
  - a) brain parenchyma (which also contains extracellular fluid): 1400 ml
  - b) cerebral blood volume (CBV): 150 ml
  - c) cerebrospinal fluid (CSF): 150 ml
- these volumes are contained in an inelastic, completely closed container
  (the skull)
- pressure is distributed evenly throughout the intracranial cavity → constant

CSF volume = 150ml (10%)

Blood volume = 150ml (10%)

Brain parenchyma volume = 1400ml (80%)



ICP principle (Modified Monro-Kellie doctrine)

- States that the sum of the intracranial volumes (CBV, brain, CSF, and other constituents (e.g. tumor, hematoma…)) is constant
- An increase in any one of these must be offset by an equal decrease in another
- The mechanism: there is a pressure equilibrium in the skull
- 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

Foramen magnum
brain بين ال
and the spinal
cord.

ICP principle (Modified Monro-Kellie doctrine)

- When this increased ICP exceeds the pressure required to force one of the other constituents out through the foramen magnum (FM) (the only true effective opening in the intact skull) that other component will decrease in size via that route until a new equilibrium is established
- The craniospinal axis can buffer small increases in volume with no change or only a slight increase in ICP
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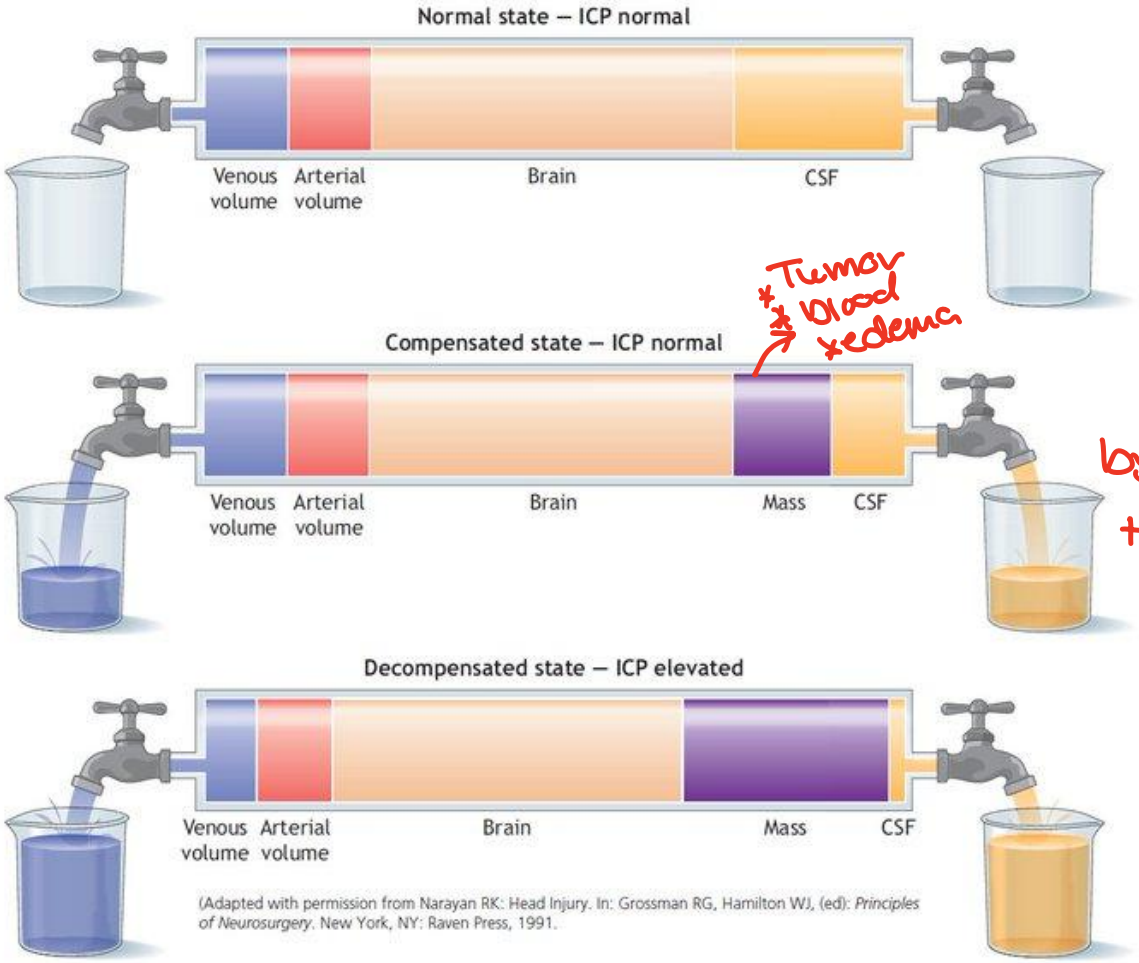
ICP principle (Modified Monro-Kellie doctrine)

- 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 FM (Foramen Magnum)
- Intravenous blood can also be displaced through the jugular foramina via the IJVs (Internal Jugular Veins)

ICP principle (Modified Monro-Kellie doctrine)

- as pressure continues to rise, arterial blood is displaced and CPP decreases, 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
- increased brain edema, or an expanding mass (e.g. hematoma) can push brain parenchyma downward into the foramen magnum (cerebral herniation)



The Monro-Kellie Doctrine Regarding Intracranial Compensation for Expanding Mass. The volume of the intracranial contents remains constant. If the addition of a mass such as a hematoma results in the squeezing out of an equal volume of CSF and venous blood, the ICP remains normal. However, when this compensatory mechanism is exhausted, there is an exponential increase in ICP for even a small additional increase in the volume of the hematoma.

* Tumor
* Blood
* edema

by Foramen magnum to spinal canal

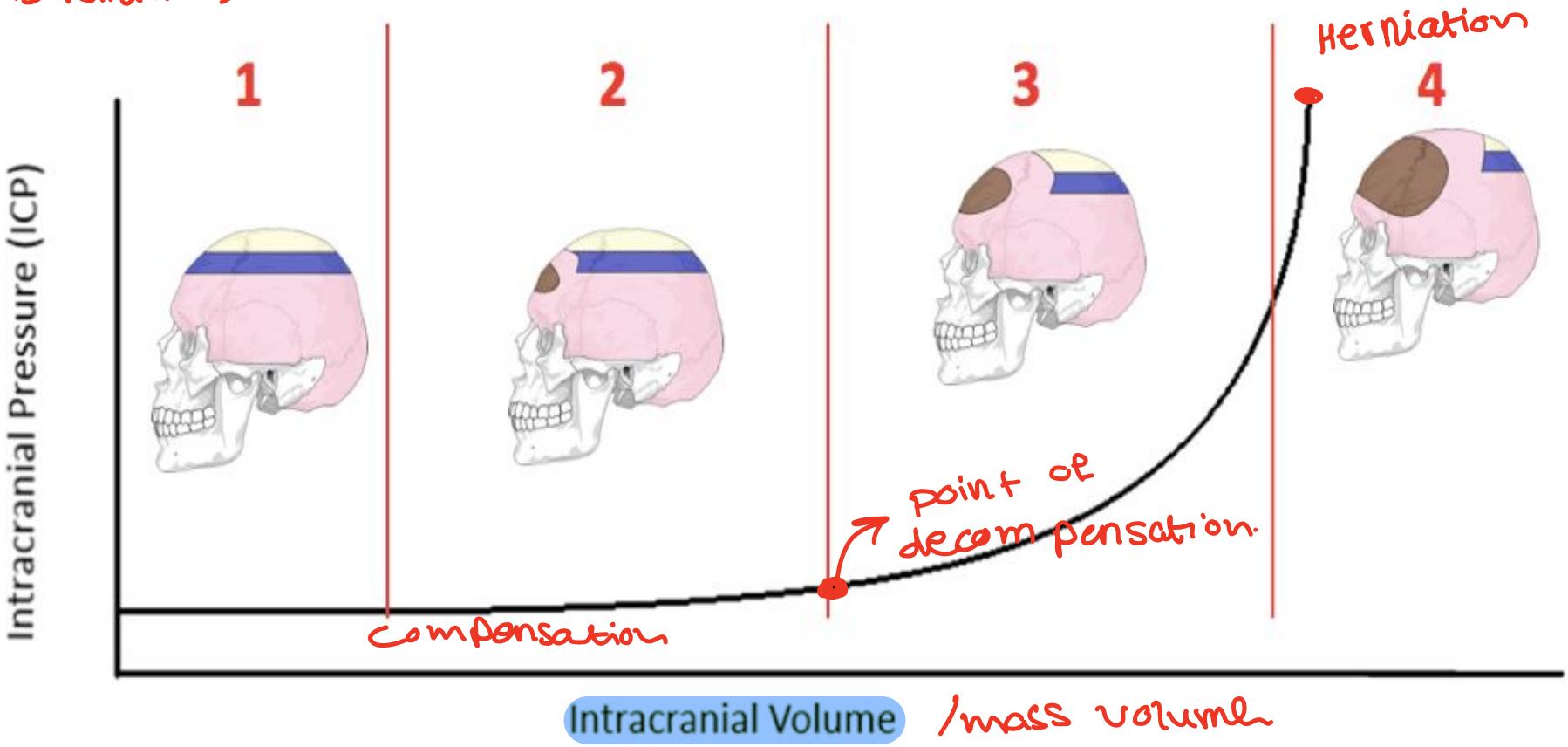
* بیشتر در CSF خارج

* بیشتر در blood و کمتر

* اذا ارتفع اکثر من هک حصر ischemia

*cycle:

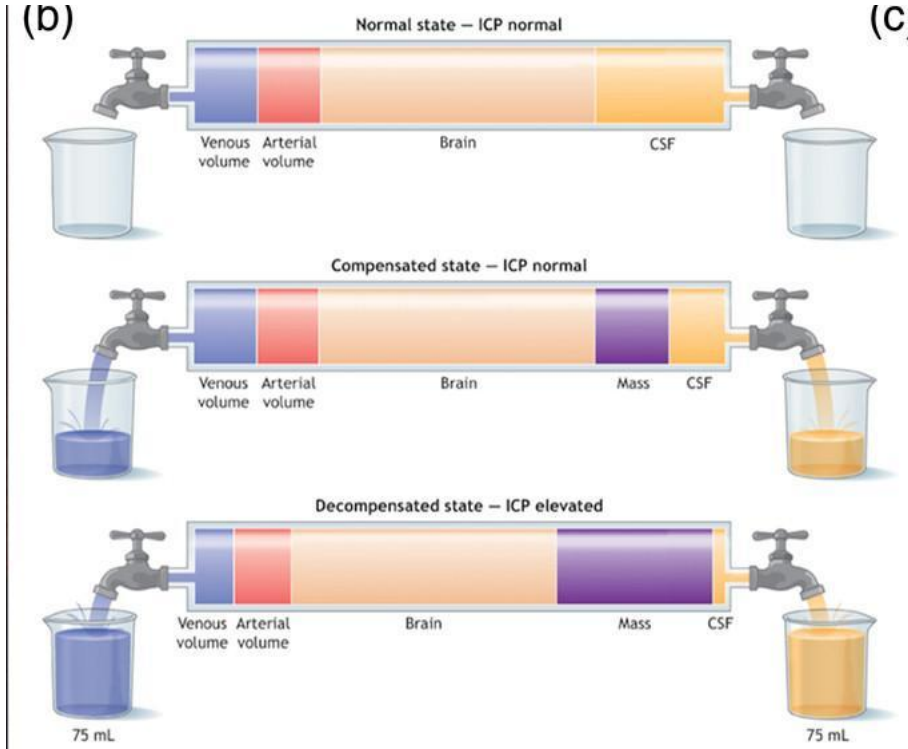
ischemia → edema → ischemia...

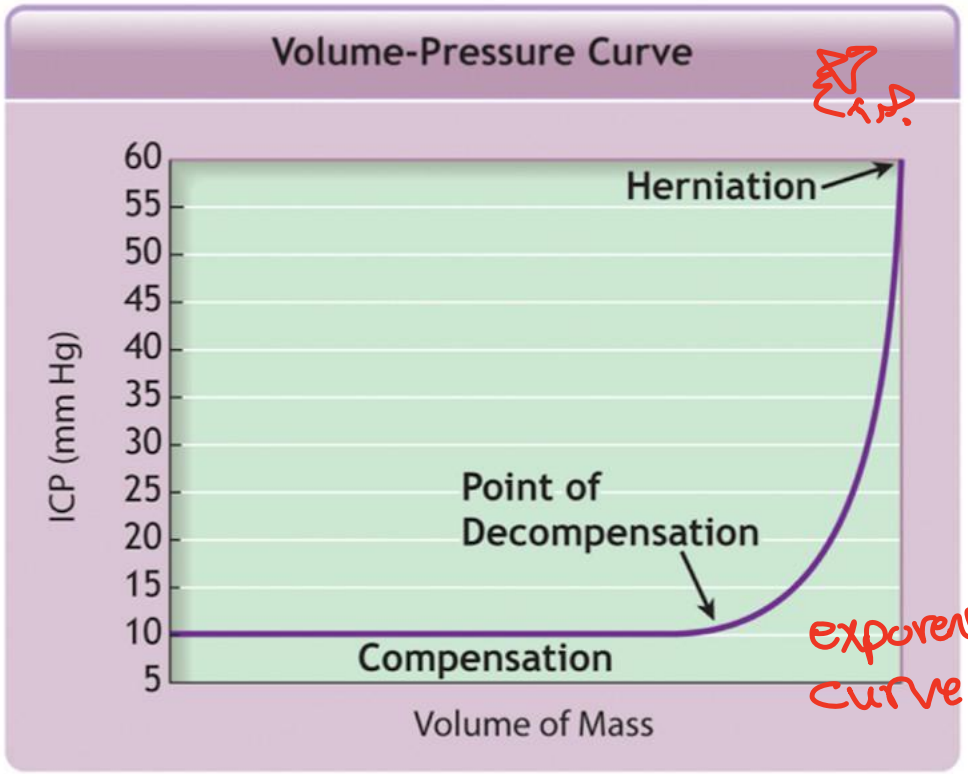


↑ ICP → ischemia → cytotoxic edema (volume ↑) → more ischemia

→ herniation of brain → brain death.

* نتابع المريض عن طريق : GCS / pupils / ICP monitor ← unconsious if





جدا

exponential curve.

* obstructed hydrocephalus ← خطيرة

لأنه واحد من ٣ compensatory mechanism هي موجودة

⊛ CSF production ↓↓.

⊛ Normal ICP

⊛ in supine position

- adults and older childrena < 10–15
- young children 3–7
- term infants 1.5–6

→ in supine erect possition maybe negative pressure

*in physiological condition we can say that
↳ CPP = MAP

Intracranial Hypertension (Increased ICP) Causes

* اشياء بتتحط في زيادة الـ ICP ← كلها الها علاقة في الـ volume.

1. cerebral edema (volume)

vasogenic 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

Intracranial Hypertension (Increased ICP) Causes

3. traumatically induced masses (volume)

a) epidural hematoma

b) subdural hematoma

c) intraparenchymal hemorrhage (hemorrhagic contusion)

d) foreign body (e.g. bullet)

e) depressed skull fracture

✓ * CSF production constant

✓ * no compensation (nor reach

Intracranial Hypertension (Increased ICP) Causes

4. hydrocephalus due to obstruction of CSF absorption or circulation

5. hypoventilation (causing hypercarbia ⟶ vasodilatation)

6. systemic hypertension (HTN)

7. venous sinus thrombosis

Intracranial Hypertension (Increased ICP) Causes

✓ *cough
✓ *straining

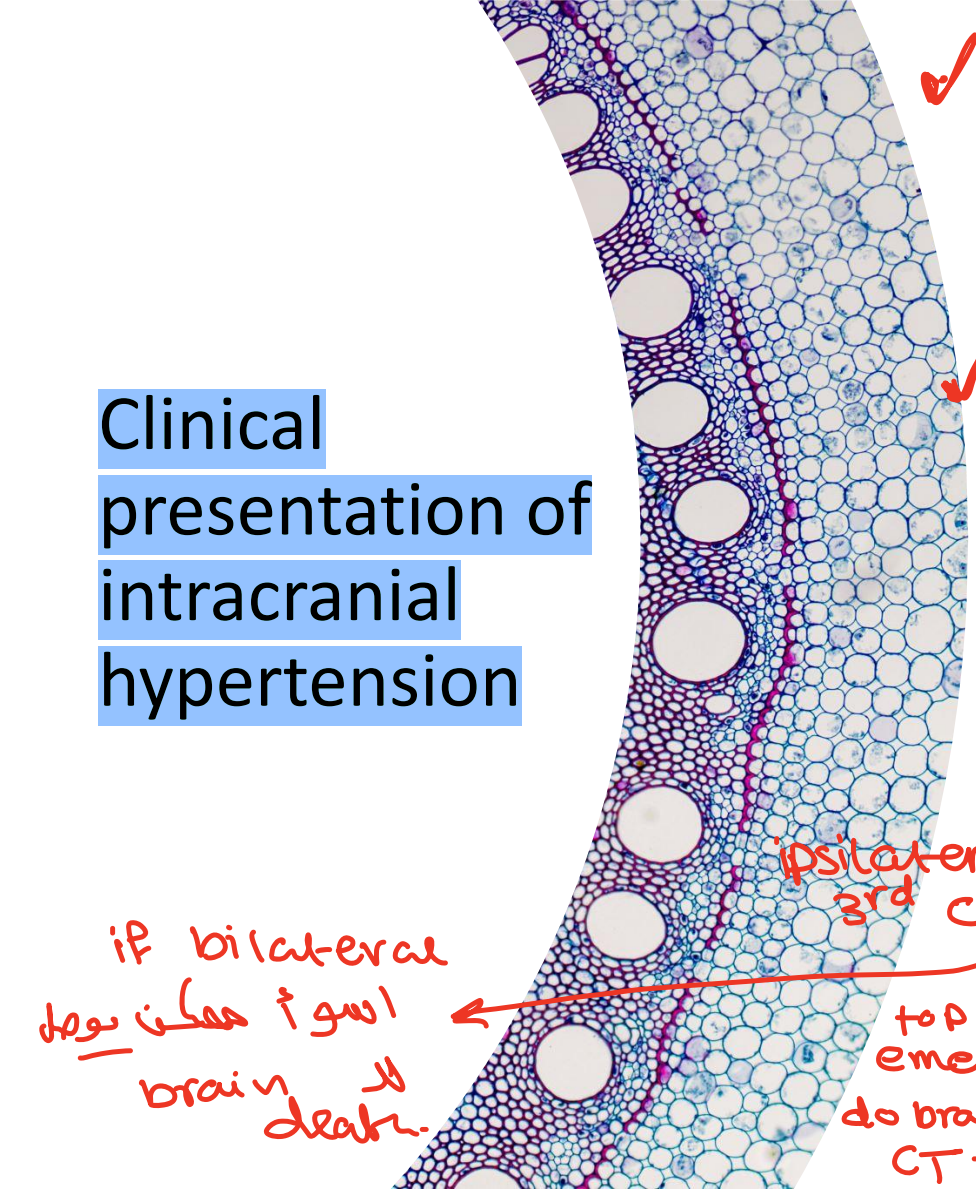
8. increased muscle tone and Valsalva maneuver as a result of agitation or posturing → increased intrathoracic pressure → increased jugular venous pressure → reduced venous outflow from head

9. sustained posttraumatic seizures (status epilepticus)

* ↑ muscle tone

* high consumption so cause vasodilation
+cause vasogenic edema

Clinical presentation of intracranial hypertension



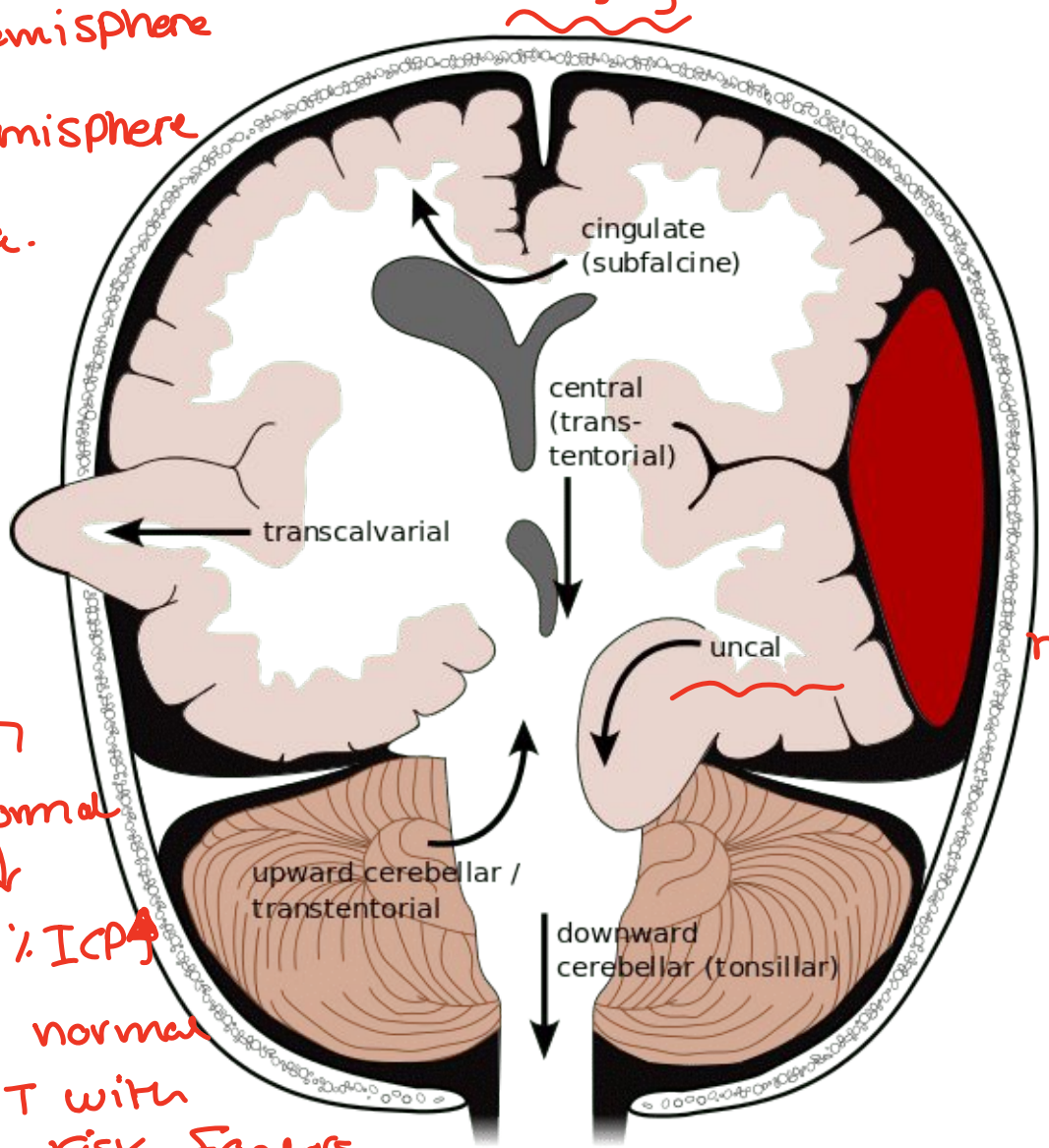
- Cushing's triad: hypertension, bradycardia, respiratory irregularity
  - Full triad seen in only 33%
- Decreased level of consciousness: obtundation, coma
- Herniation syndromes (in compartmental increased pressure)
  - Cranial nerve III palsy in subfalcine (uncal herniation)
  - Fixed dilated pupil
  - Seen in unilateral cerebral mass

*Rt cerebral hemisphere

*lt cerebral hemisphere

* posterior Fossa.

*dura → rigid



medial temporal lobe

*CT

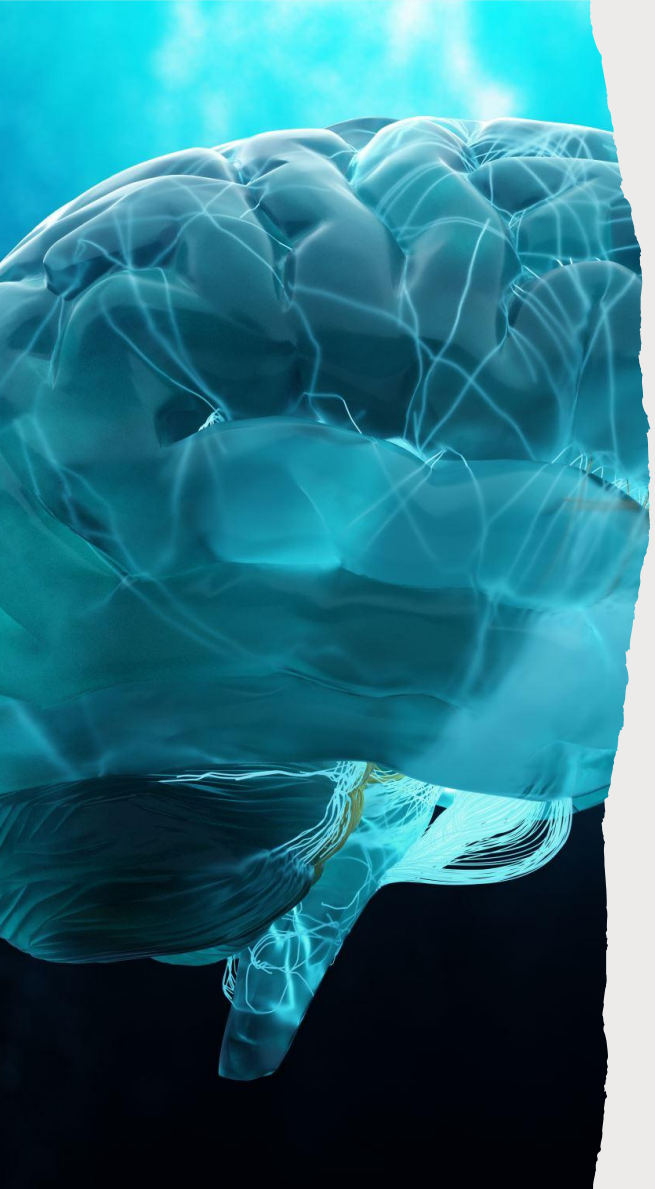
normal → 13% ↑ICP

abnormal → 60% ICP

*or normal CT with ≥2 risk factors

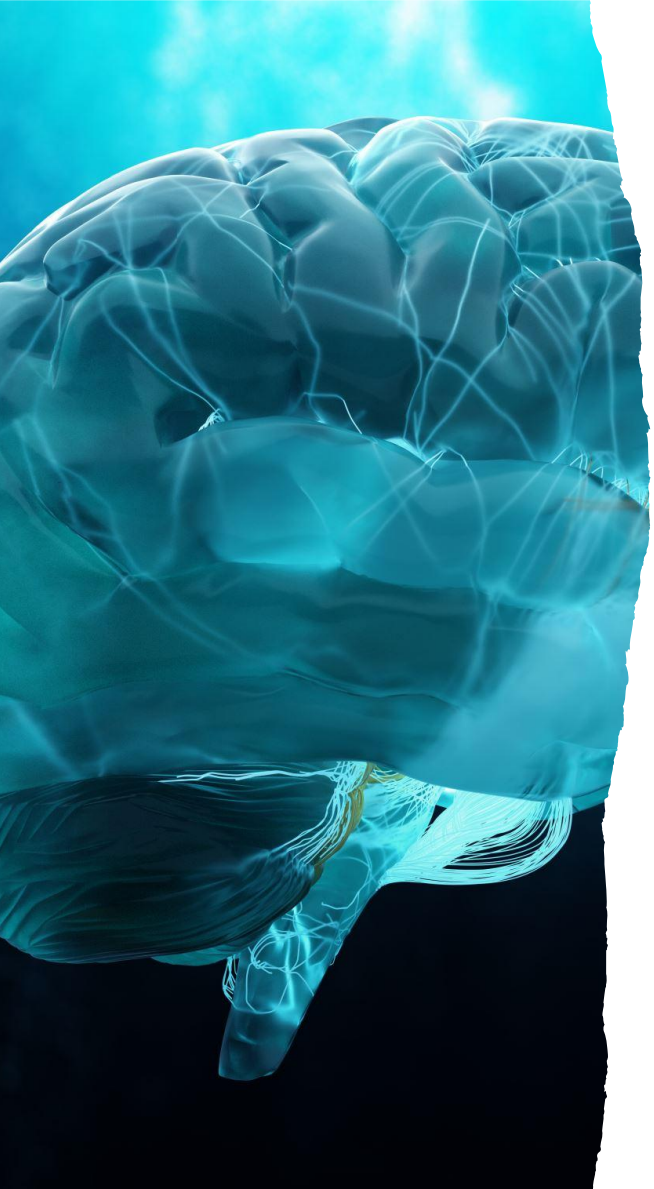
Intracranial pressure measurement and monitoring, CT scan

- In trauma: 60% of patients with closed head injury and an abnormal CT will have IC-HTN
- In trauma: Only 13% of patients with a normal CT scan will have IC-HTN
- In trauma: However, patients with a normal CT AND 2 or more risk factors identified have ≈ 60% risk of IC-HTN
  - age > 40 yrs
  - SBP < 90mm Hg
  - decerebrate or decorticate posturing on motor exam (unilateral or bilateral)



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, SDH or ICH), contusions,15 compression of basal cisterns (p. 959), herniation, or swelling on CT.
- with a normal admitting brain CT, but with ≥ 2 of the risk factors for IC-HTN
  - ✓ age > 40 yrs
  - ✓ SBP < 90mm Hg
  - ✓ decerebrate or decorticate posturing on motor exam (unilateral or bilateral)



Contraindication for ICP monitoring

Contraindications (relative)

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

monitoring عملية الـ

invasive هي

ممكن يعمل عنا :

Hemorrhage

→ Peak of IC-HTN in trauma

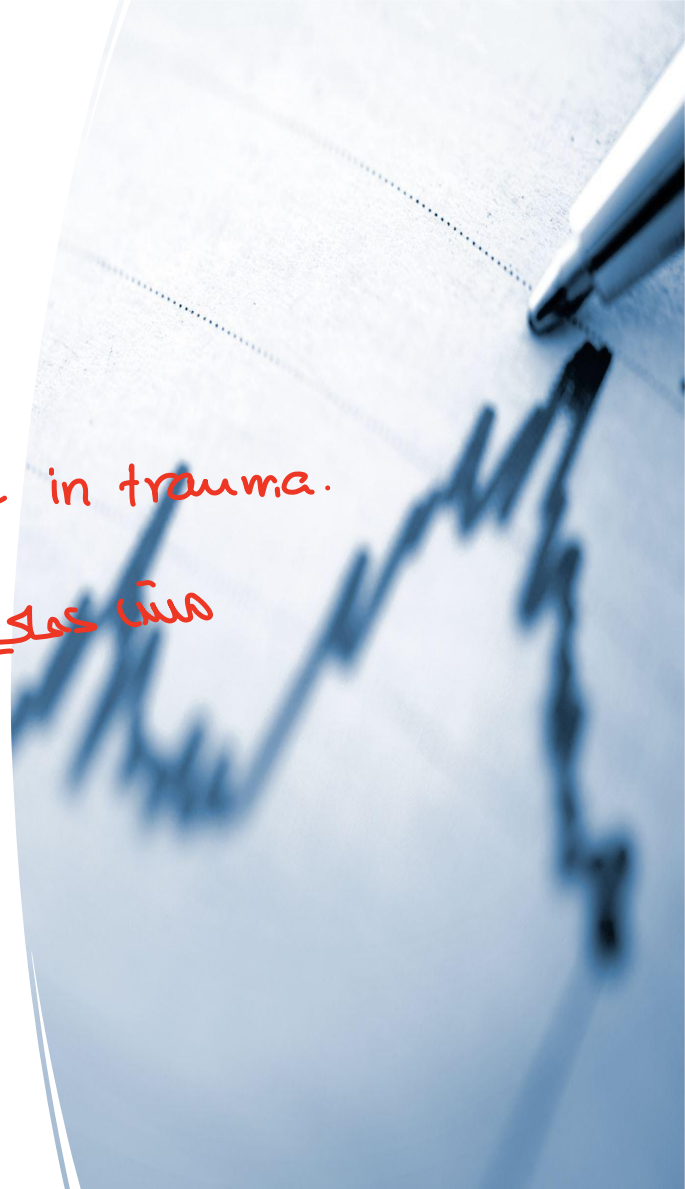
* Cerebral edema by the trauma.

- day 2–3
- day 9–11

Types of ICP monitor

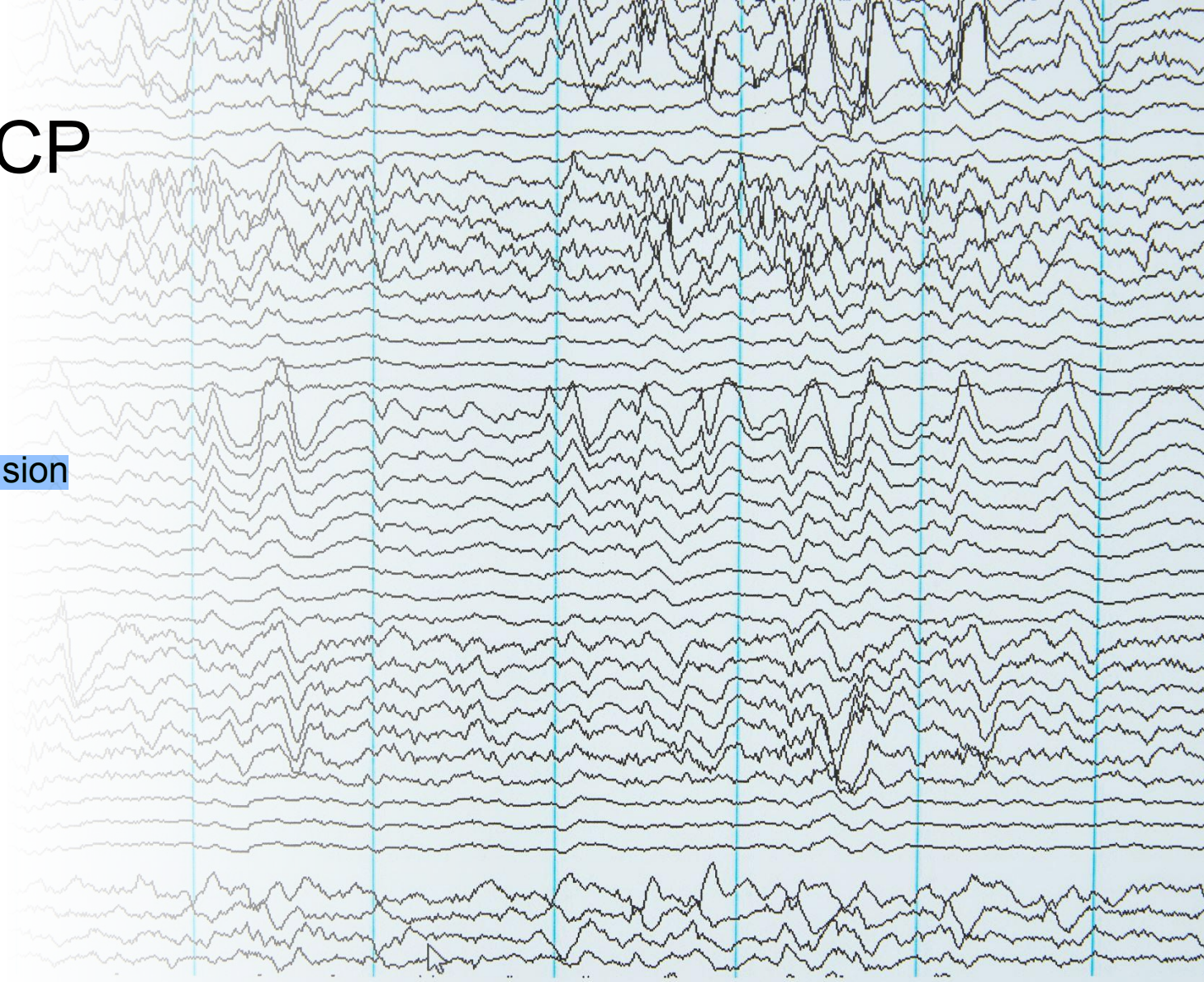
- intraventricular catheter (IVC) most accurate in trauma. (sec - sec)
- intraparenchymal monitor
- subarachnoid screw (bolt) edema في حالات
- Subdural catheter shrinkage لأنه يكون في
- Epidural monitor ventricle في

- *In infants: palpate fontanel*



Adjuncts to ICP monitoring

- ✓ Jugular venous oxygen monitoring
- ✓ Brain tissue oxygen tension monitoring (PbtO2)
- ✓ Bedside monitoring of regional CBF (rCBF)
- ✓ Cerebral microdialysis



Treatment

- treatment for IC-HTN should be initiated for ICP > 22mm Hg
- the need for treatment should be based on ICP in combination with clinical examination & brain CT findings

- Avoid CPP < 50mm Hg
- Avoid increasing CPP more than 70mmHg

ⓤ cardiac tamponade

Treatment: Goals of therapy

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

* ABC

Treatment: initial:

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

Treatment: initial:

- avoid hypoxia
  (PaO2 < 60mm Hg or O2 sat < 90%)
- ventilate to normocarbia
  (PaCO2 = 35–40mm Hg)
- ✓ light sedation: e.g. codeine — contraindicated if patient hypotensive
- ✓ controversial: prophylactic → at least not Febrile patient.
  hypothermia: Hypothermia → ↓CMRO2 — to prevent ← ischemia
- 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 PaCO2 = 30–35mm Hg
- mannitol 0.25–1 gm/kg
- 10–20 ml of 23.4%
- hypertonic saline (HS)
- Augmented hyperventilation to ↓PaCO2 to 25–30mm Hg

Treatment:

* surgical

- traumatic intracranial masses should be treated as indicated

- decompressive craniectomy

1. Focal lesion (بنشيلو)
2. Hydrocephalus → CSF drain. [EVD].

rigid بس dura بنفتح