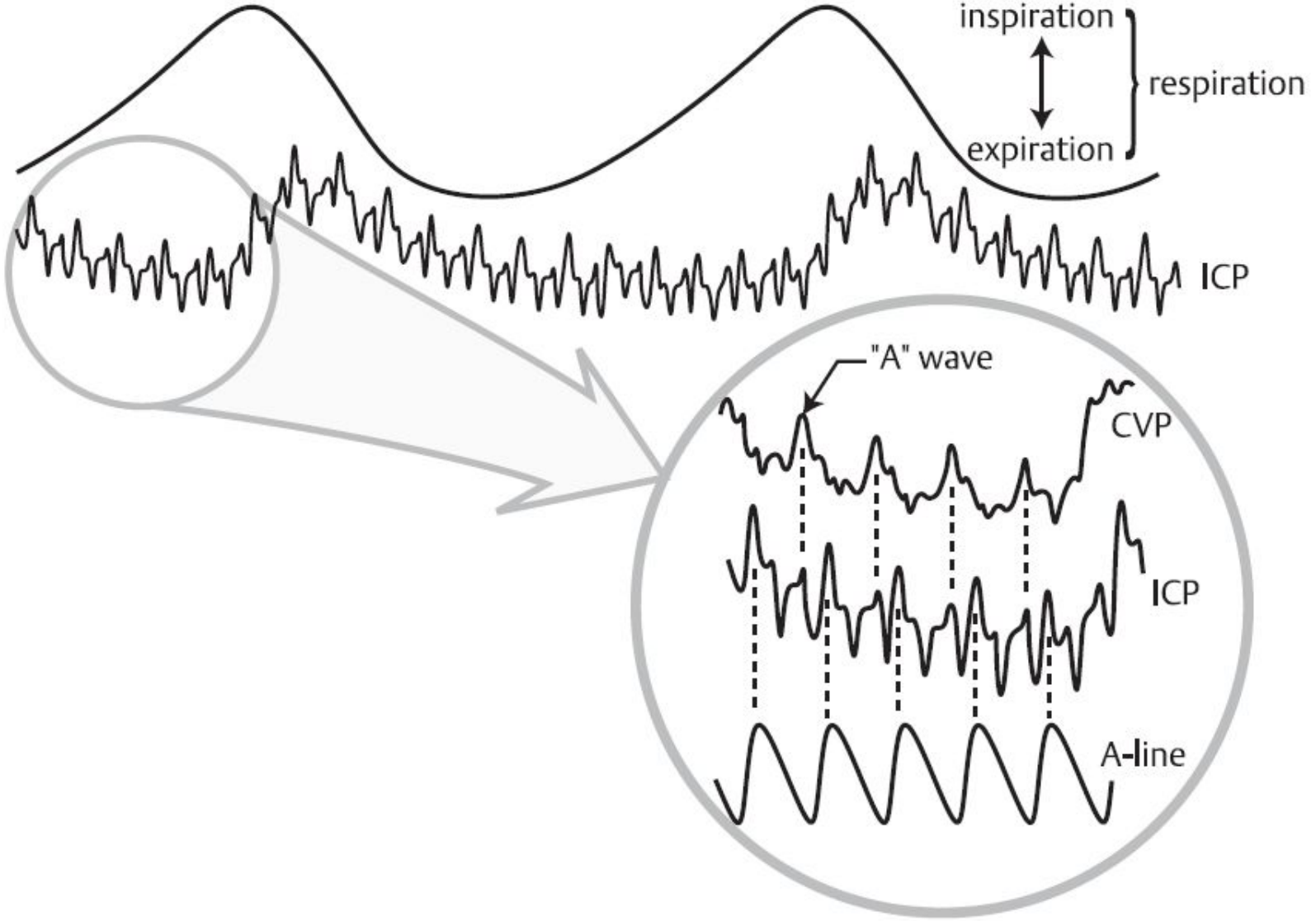
Intracranial pressure


inspiration
respiration
expiration
ICP
"A" wave
CVP
ICP
A-line

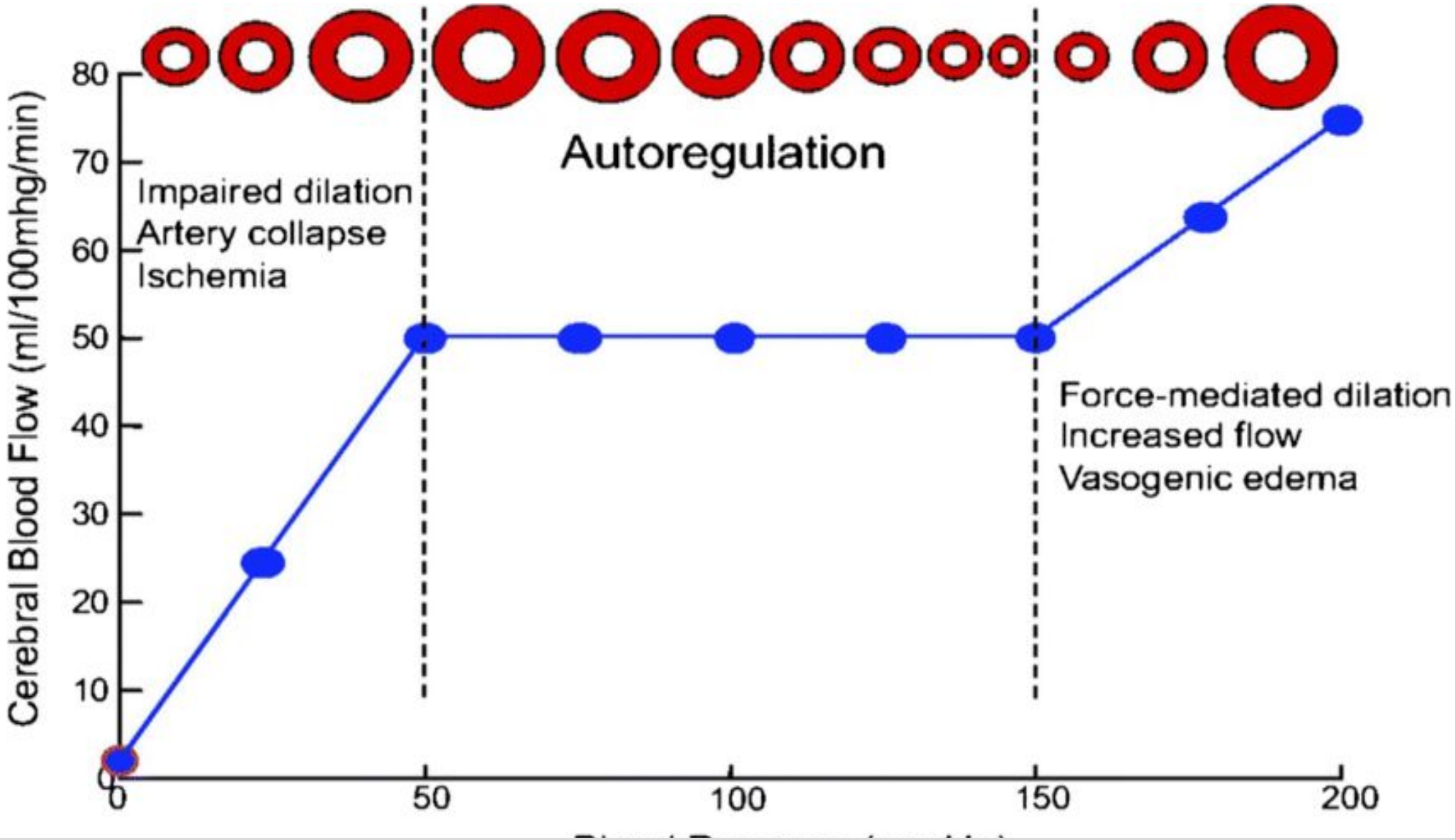
Why? Intracranial pressure

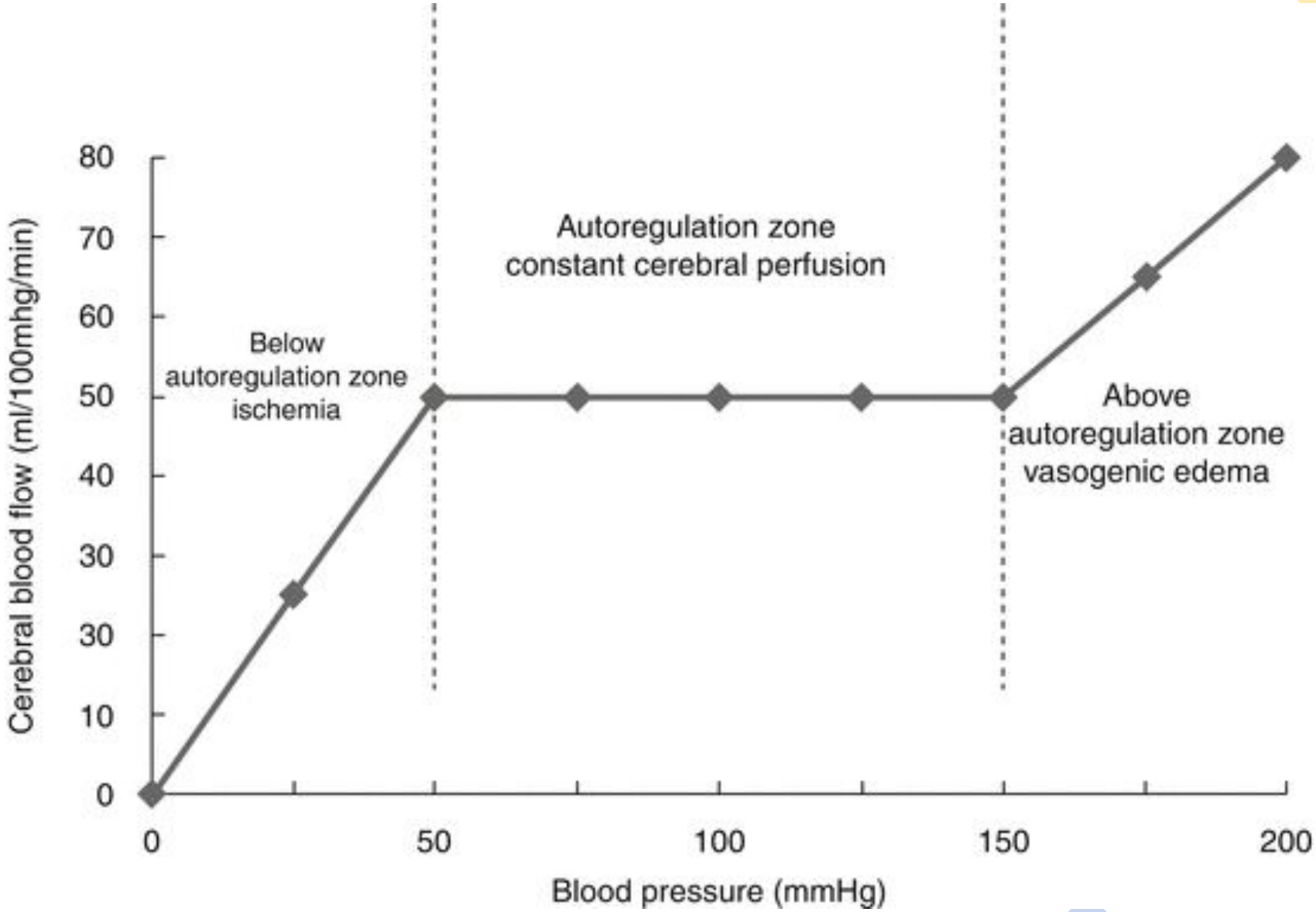
- Cerebral oxygenation depends on cerebral blood flow
- Cerebral blood flow (CBF) = **Cerebral perfusion pressure (CPP)**
  Cerebral vascular resistance (CVR)
- Cerebral perfusion pressure
  = Mean arterial pressure (MAP) – **Intracranial pressure (ICP)**

- 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


Autoregulation
Impaired dilation
Artery collapse
Ischemia
Force-mediated dilation
Increased flow
Vasogenic edema
Cerebral Blood Flow (ml/100mhg/min)
80
70
60
50
40
30
20
10
0
0
50
100
150
200


Autoregulation zone
constant cerebral perfusion
Below
autoregulation zone
ischemia
Above
autoregulation zone
vasogenic edema
Cerebral blood flow (ml/100mhg/min)
80
70
60
50
40
30
30
10
0
0
50
100
150
200
Blood pressure (mmHg)

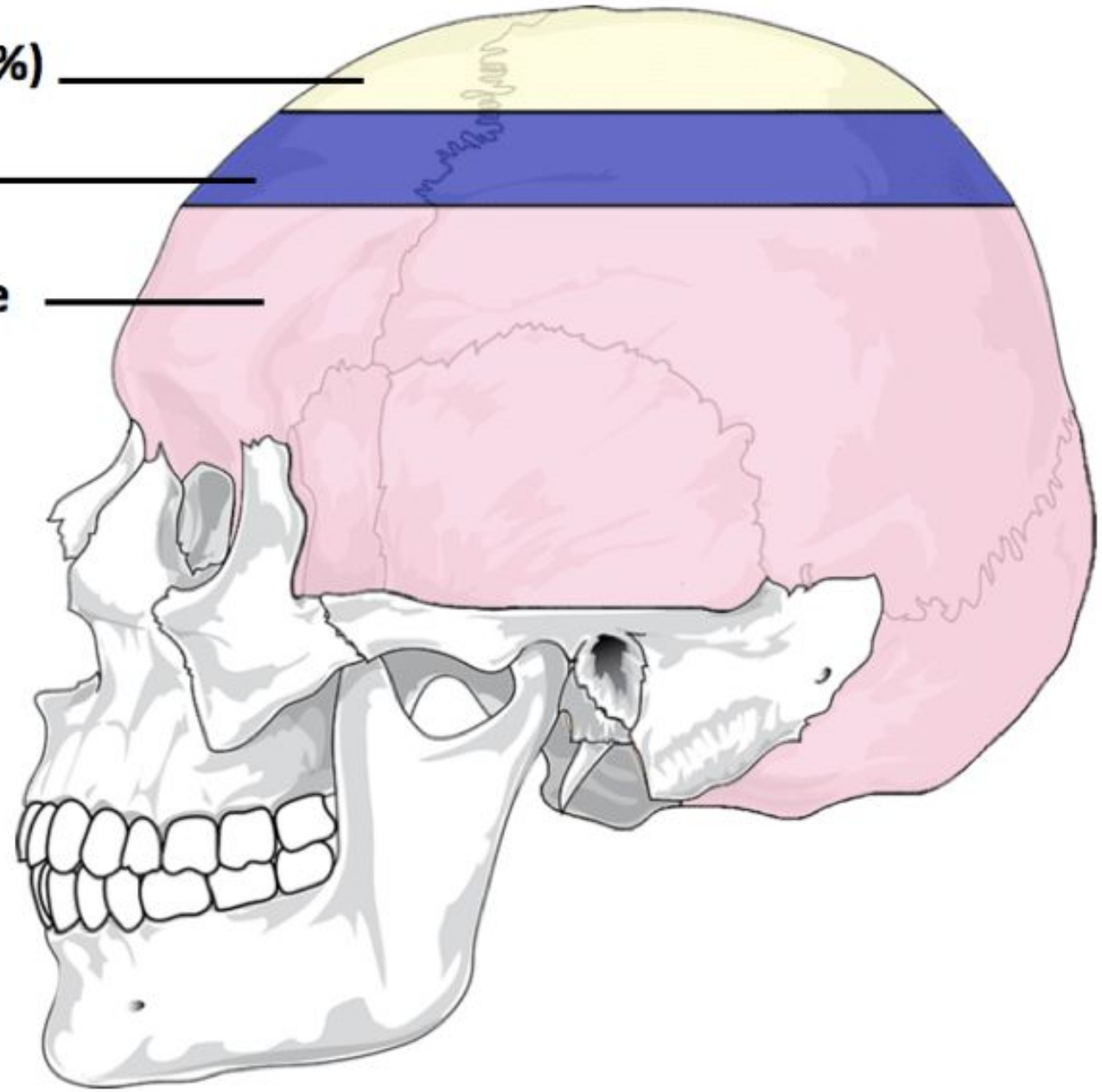
ICP principles (Model)

- 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

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

ICP principle (Modified Monro-Kellie doctrine)

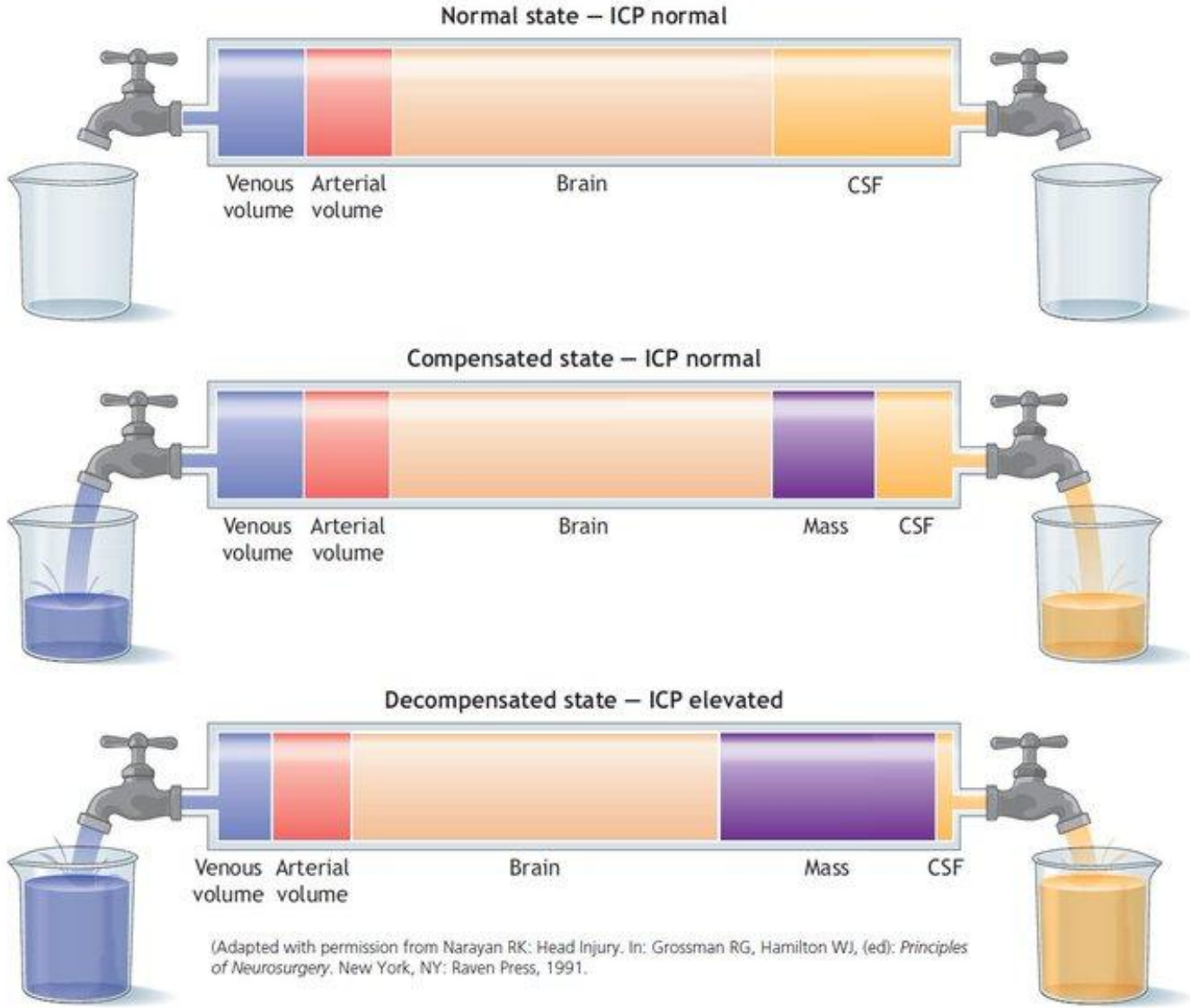
- When this increased ICP exceeds the pressure required to force one of the other constituents out through the foramen magnum (FM) (the only true effective opening in the intact skull) that other component will decrease in size via that route until a new equilibrium is established
- The craniospinal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

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)

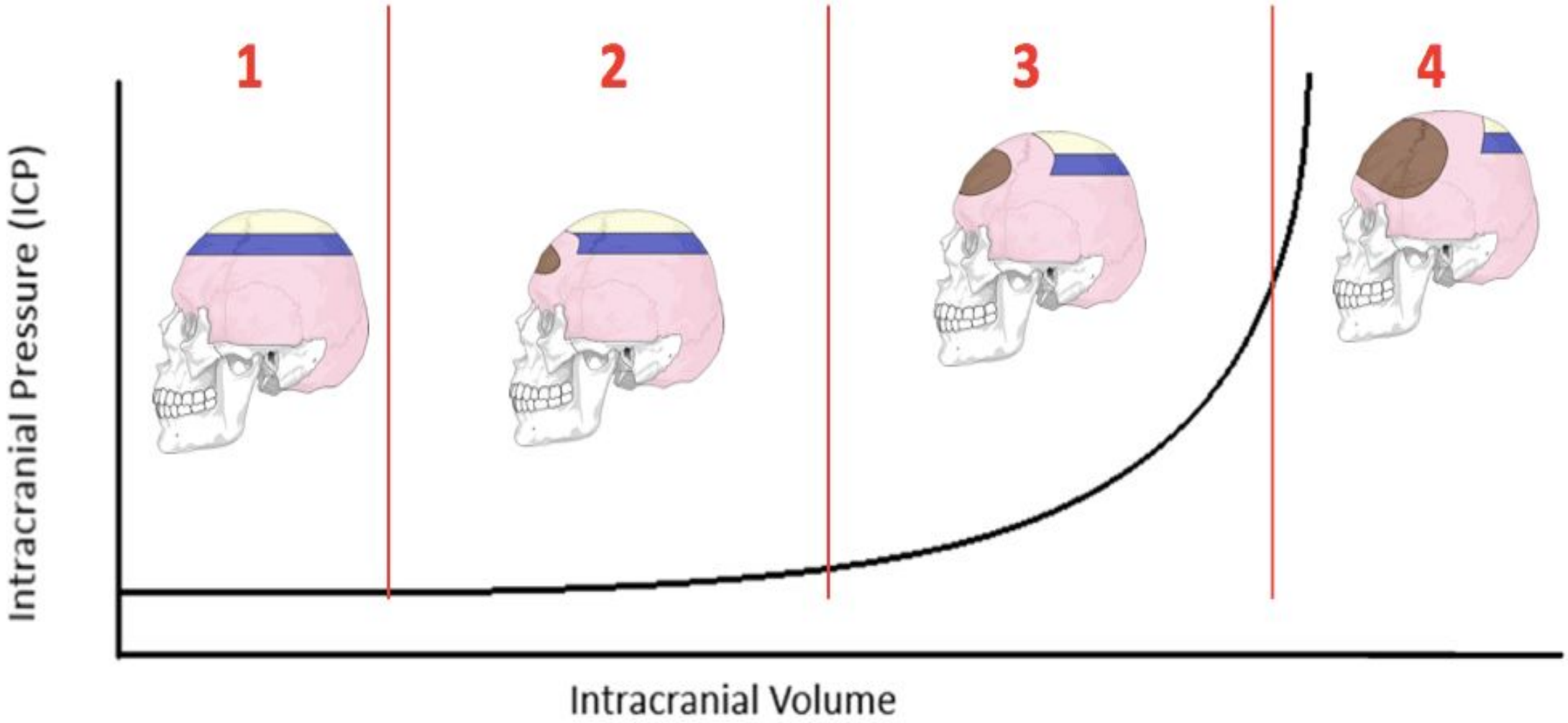
ICP principle (Modified Monro-Kellie doctrine)

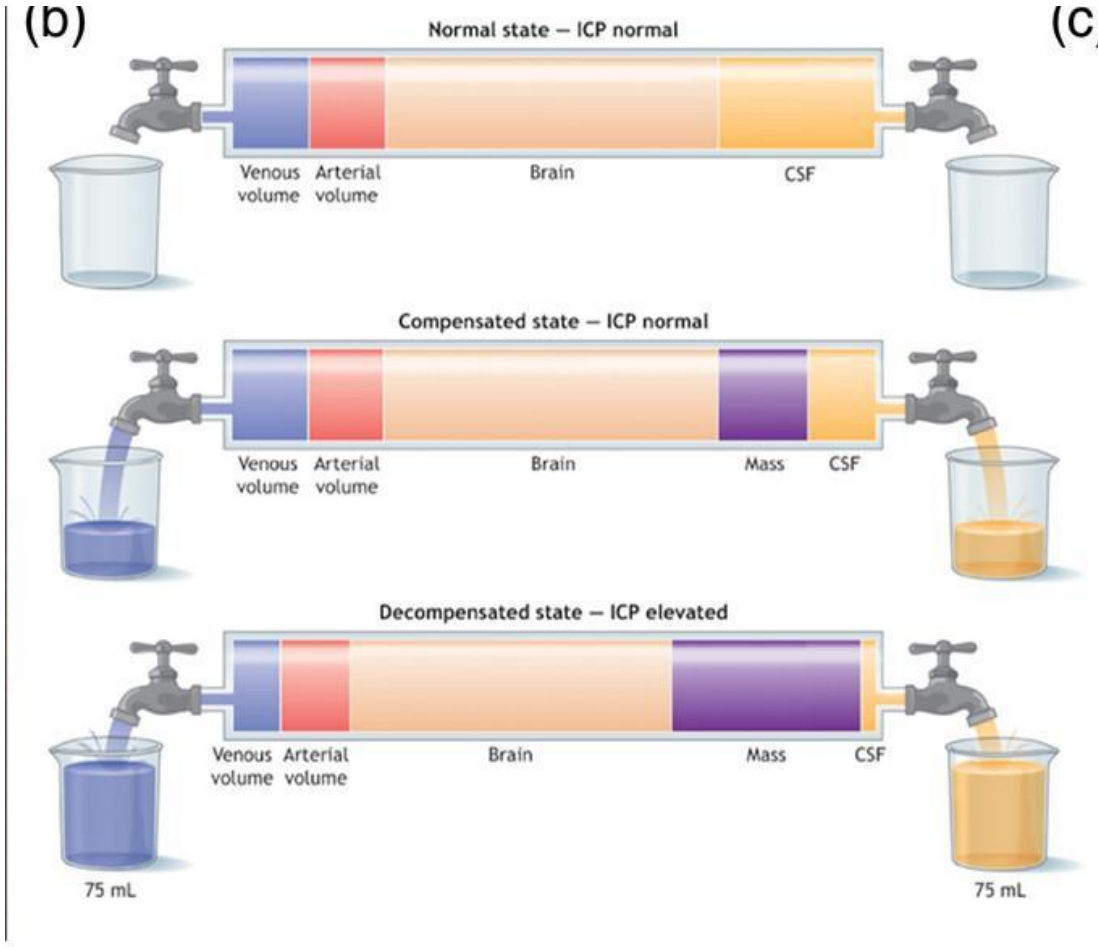
- as pressure continues to rise, arterial blood is displaced and CPP decreases, eventually producing diffuse cerebral ischemia. At pressures equal to mean arterial pressure, arterial blood will be unable to enter the skull through the FM, producing complete cessation of blood flow to the brain, with resultant massive infarction
- increased brain edema, or an expanding mass (e.g. hematoma) can push brain parenchyma downward into the foramen magnum (cerebral herniation)

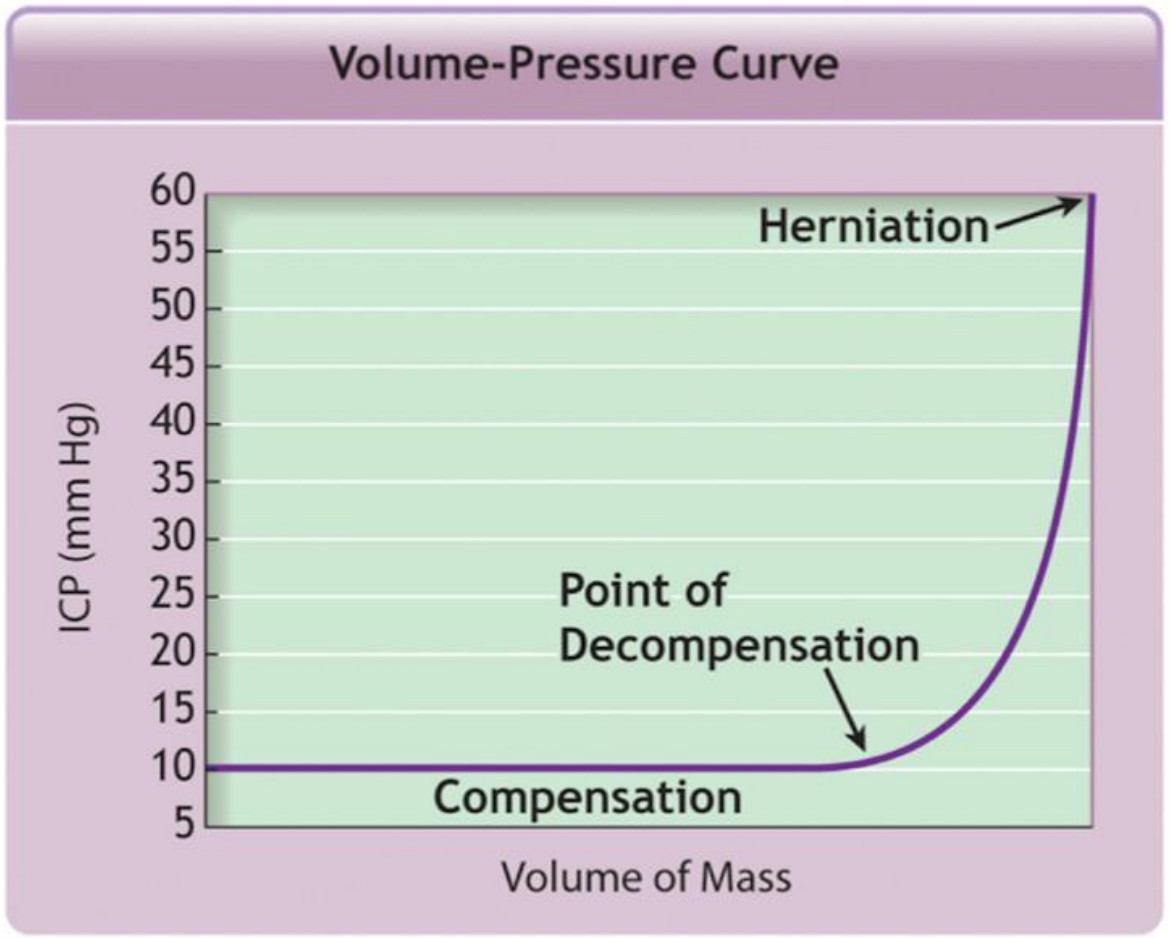


(Adapted with permission from Narayan RK: Head Injury. In: Grossman RG, Hamilton WJ, (ed): *Principles of Neurosurgery*. New York, NY: Raven Press, 1991.

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.


1
2
3
4
Intracranial Pressure (ICP)
Intracranial Volume


(b)
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

Normal ICP

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

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

Intracranial Hypertension (Increased ICP) Causes

- 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

Intracranial Hypertension (Increased ICP) Causes

4. hydrocephalus due to obstruction of CSF absorption or circulation

5. hypoventilation (causing hypercarbia → vasodilatation)

6. systemic hypertension (HTN)

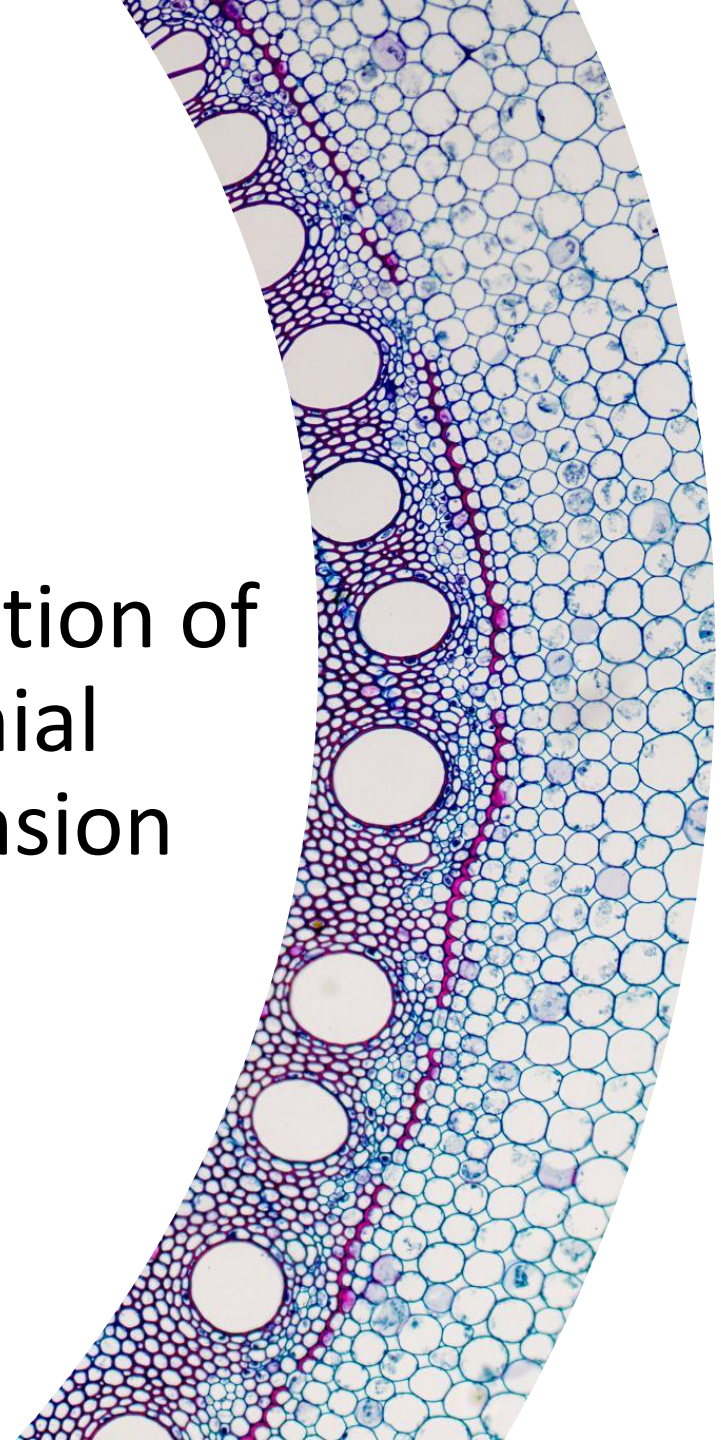
7. venous sinus thrombosis

Intracranial Hypertension (Increased ICP) Causes

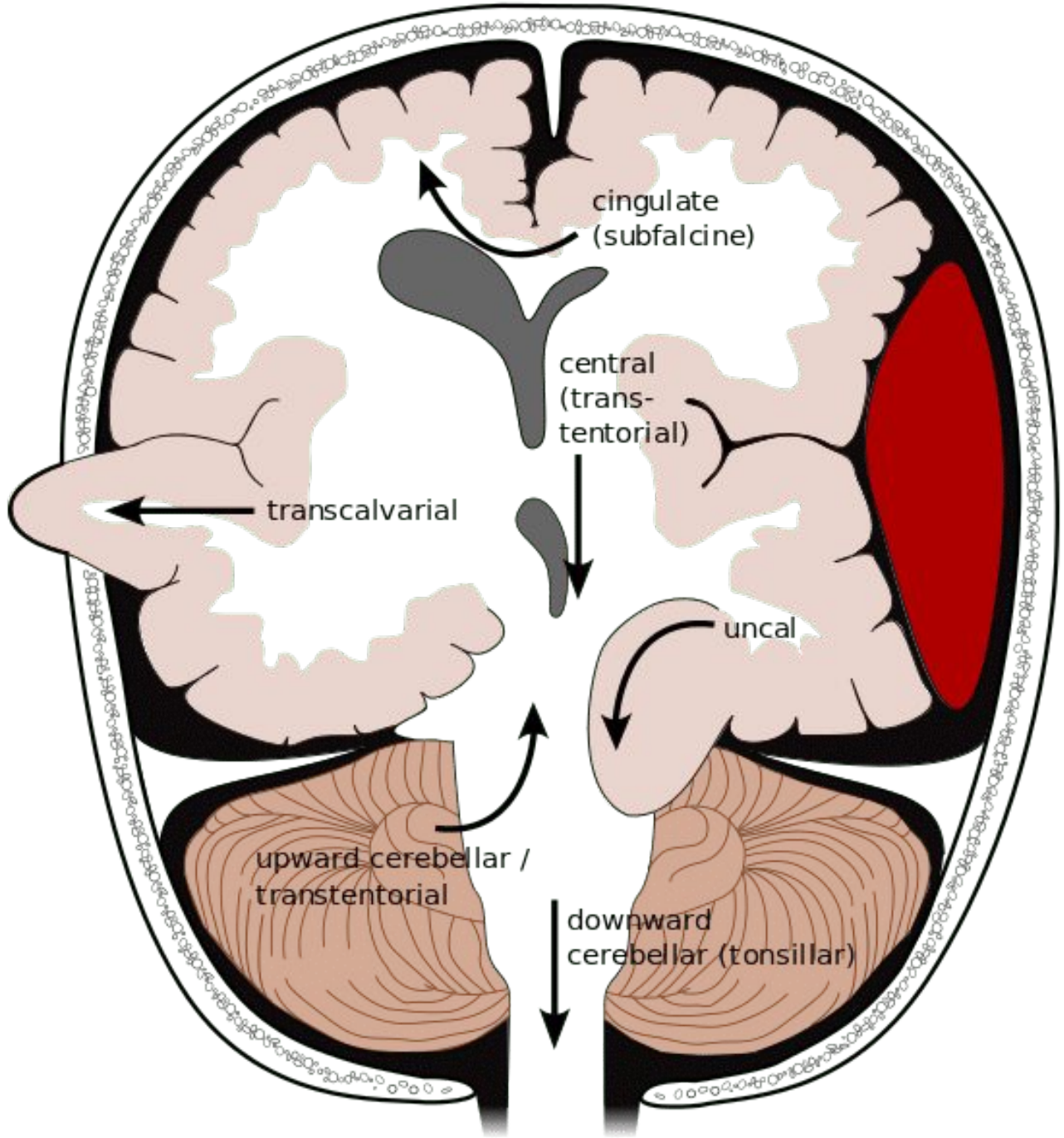
8. increased muscle tone and Valsalva maneuver as a result of agitation or posturing → increased intrathoracic pressure → increased jugular venous pressure → reduced venous outflow from head

9. sustained posttraumatic seizures (status epilepticus)

Clinical presentation of intracranial hypertension

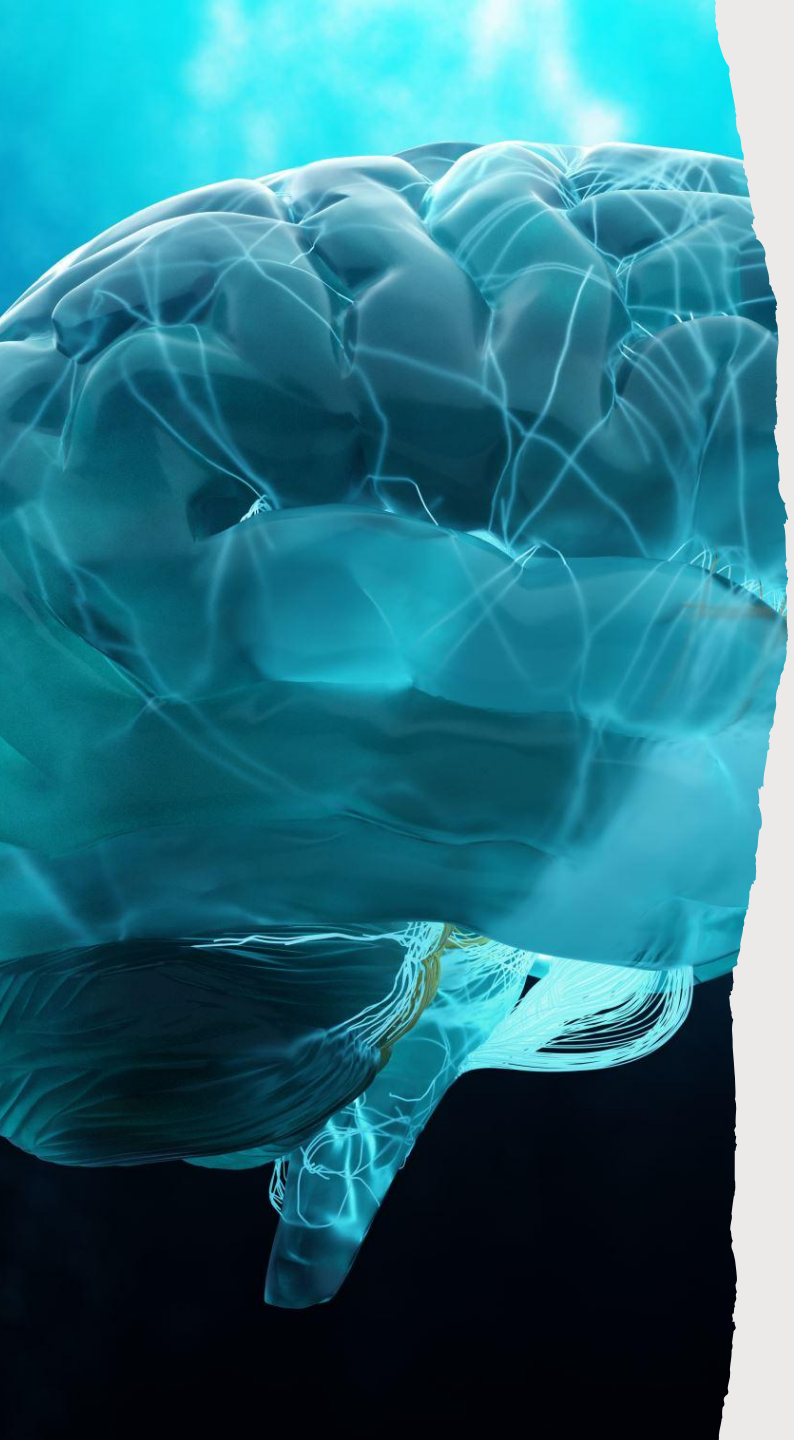


- Cushing's triad: hypertension, bradycardia, respiratory irregularity
  - Full triad seen in only 33%
- Decreased level of consciousness: obtundation, coma
- Herniation syndromes (in compartmental increased pressure)
  - Cranial nerve III palsy in subfalcine (uncal herniation)
  - Fixed dilated pupil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


cingulate
(subfalcine)
central
(trans-
tentorial)
transcalvarial
uncal
upward cerebellar /
transtentorial
downward
cerebellar (tonsillar)

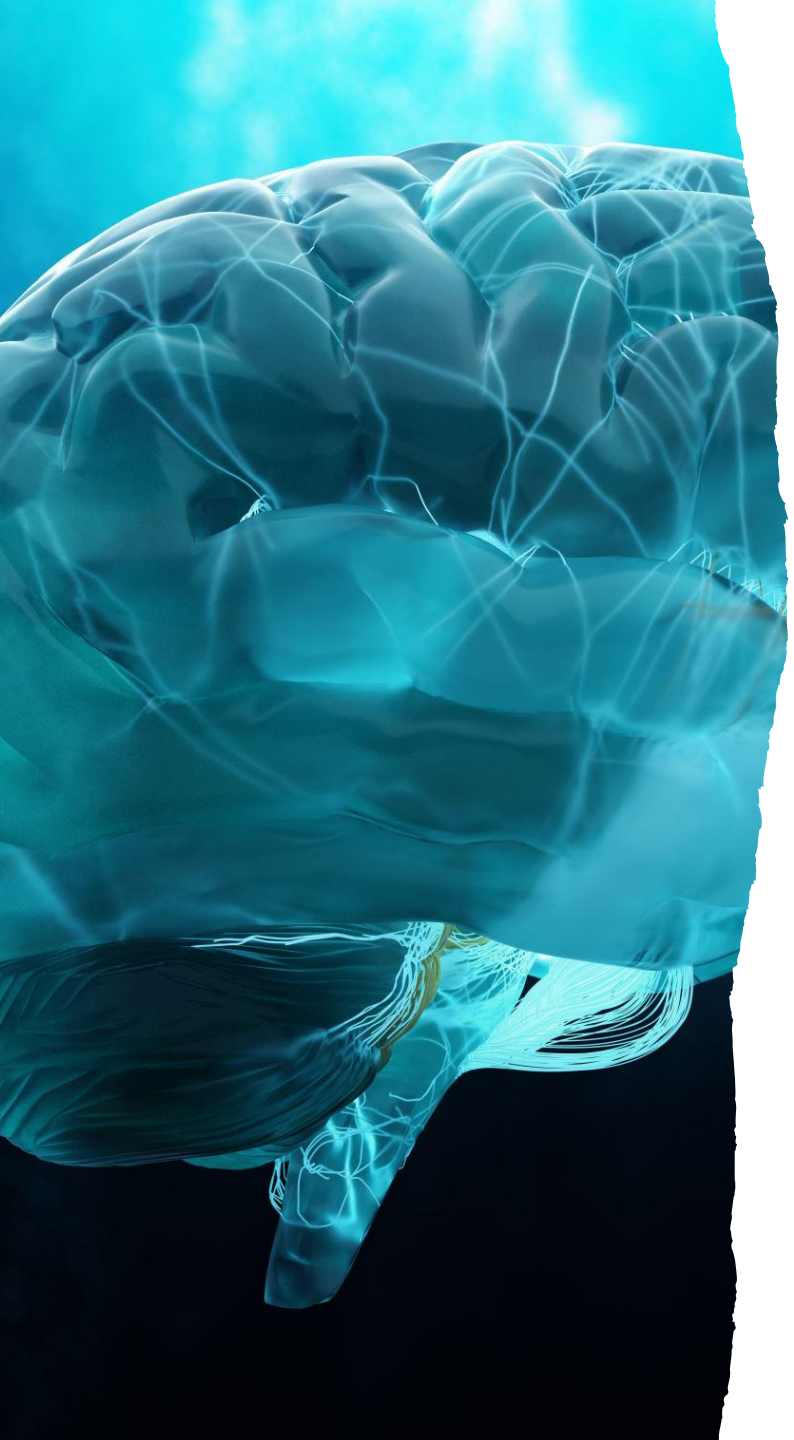
Intracranial pressure measurement and monitoring, CT scan

- 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
- 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

Peak of IC-HTN in trauma

- day 2–3
- day 9–11

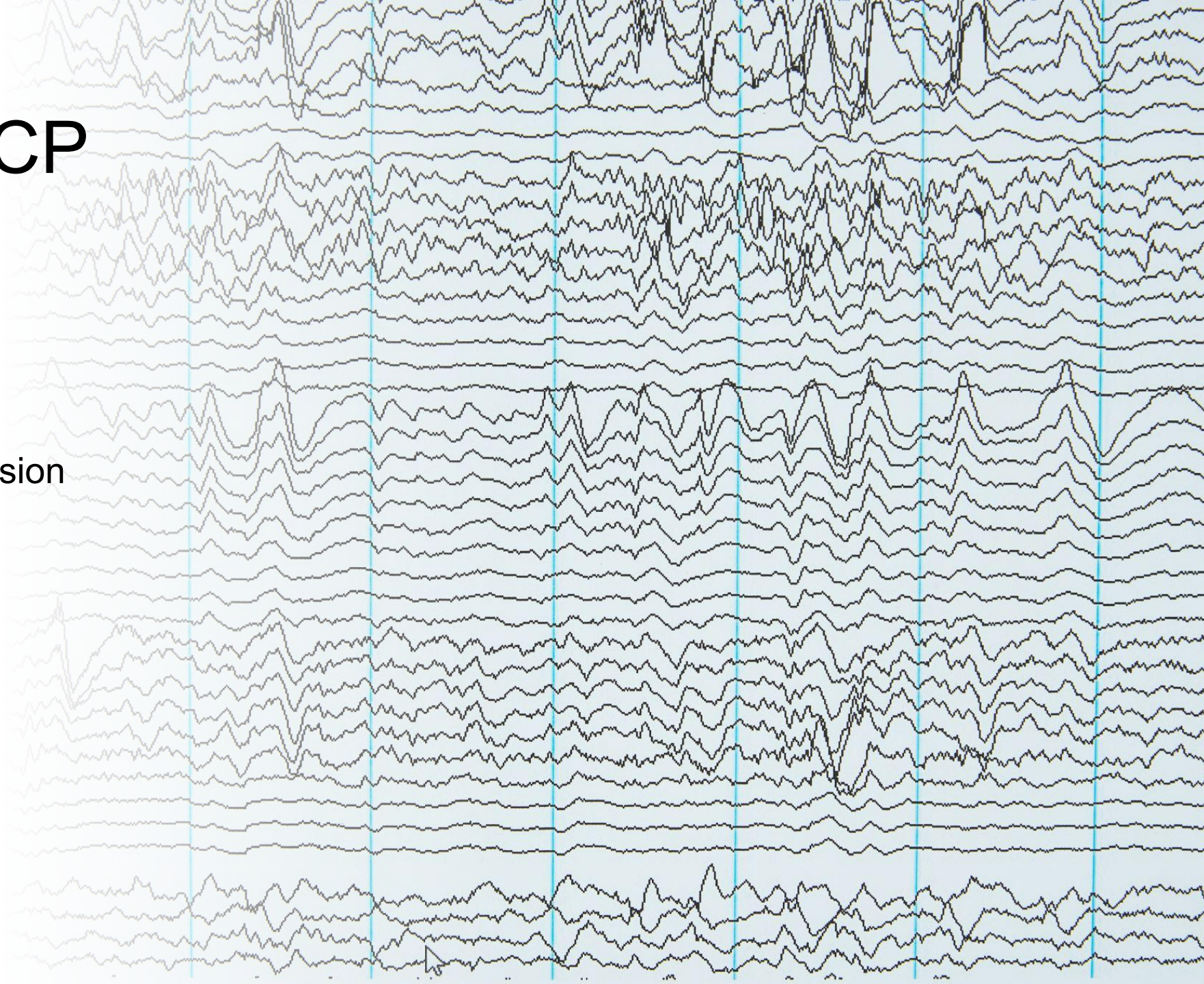
Types of ICP monitor

- intraventricular catheter (IVC)
- intraparenchymal monitor
- subarachnoid screw (bolt)
- Subdural catheter
- Epidural monitor

- *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

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 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
- controversial: prophylactic hypothermia: Hypothermia →↓CMRO2
- 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