TBI: Advanced Basics for General Surgery residents

Case Based Discussion Format

Ala'a Al-Mousa M.B.ch.B, FRCS(NS)

Associate Professor in Neurosurgery
Neurosurgery Consultant

Case I

Mr. Sameer, 45 yrs old male , works as Construction worker , Presents to the Emergency Department (ED) 1 hour after falling from a ladder, approximately 1.5 meters high.

Chief Complaint: Headache and dizziness.

- Mechanism of injury: Fell and hit the occiput on the ground.
- Loss of consciousness (LOC): Brief (less than 30 seconds), according to coworkers.
- Post-traumatic amnesia: Present (confused for ~5 minutes after the fall).
- Nausea/Vomiting: One episode of vomiting.
- Seizures: None.
- Bleeding/CSF leak: None observed.
- Medical history: Hypertension (on amlodipine), no anticoagulant or antiplatelet use.

- Now alert, GCS 15, complains of mild headache.
- GCS: 15/15
- Vitals: Stable
- Pupils: Equal and reactive
- Neurological exam: Normal tone, power, reflexes, co-ordination.
- Scalp: Mild swelling at the occiput, no open wounds.

Panel 1: **Canadian CT Head Rule**

CT Head Rule is only required for patients with minor head injuries with any one of the following:

High risk (for neurological intervention)

- GCS score <15 at 2 h after injury
- Suspected open or depressed skull fracture
- Any sign of basal skull fracture (haemotympanum, 'racoon' eyes, cerebrospinal fluid otorrhoea/rhinorrhoea, Battle's sign)
- Vomiting ⩾two episodes
- Age ⩾65 years

Medium risk (for brain injury on CT)

- Amnesia before impact >30 min
- Dangerous mechanism (pedestrian struck by motor vehicle, occupant ejected from motor vehicle, fall from height >3 feet or five stairs)

Minor head injury is defined as witnessed loss of consciousness, definite amnesia, or witnessed disorientation in a patients with a GCS score of 13–15.

Excluded

- Age under 16
- Minimal head injury with no LOC, amnesia, or disorientation
- Unclear history of trauma as the primary event (ie primary seizure or syncope)
- Obvious penetrating skull injury or depressed fracture
- Acute focal neurological deficit
- Unstable vital signs associated with major trauma
- Seizure prior to ED assessment
- Anticoagulation or bleeding disorder
- Pregnancy

Disrupted Brain Function:
There should be a disruption of normal brain function, as evidenced by at least one of the following: loss of consciousness, loss of memory for events immediately before or after the accident, alteration in mental state (e.g., feeling dazed, confused, or disoriented), or temporary neurological deficits.

Loss of Consciousness:
The period of loss of consciousness should be 30 minutes or less.

Glasgow Coma Scale (GCS):
After 30 minutes, if consciousness is regained, the GCS score should be 13-15.

Post-Traumatic Amnesia (PTA):
PTA, or loss of memory for events after the injury, should be no longer than 24 hours.

- Always consider **clinical decision rules** before CT to reduce unnecessary imaging.
- Patients with normal CT and no risk factors can be **safely discharged**.
- Mild head injury still carries risk of **post-concussion syndrome**—educate the patient.
- In elderly or anticoagulated patients, even minor trauma may require **more cautious** management.

Feature	**Concussion**	**Mild TBI (mTBI)**
Scope	Subtype of mTBI	Broader category
Use	Common in sports/pediatrics	Used in EDs, trauma, neuro, and research
Diagnosis	Clinical, often symptom-based	Based on criteria (GCS, LOC, PTA, imaging)
Imaging	Always normal	Typically normal, but can have mild findings
Pathophysiology	Functional disruption	May include both functional + subtle injury
Terminology Origin	Traditional/lay term	Medical/neurological classification

Post-Concussion Syndrome (PCS)

- **Definition:** Symptoms persisting >7–10 days (adults) or >4 weeks (children) post-concussion

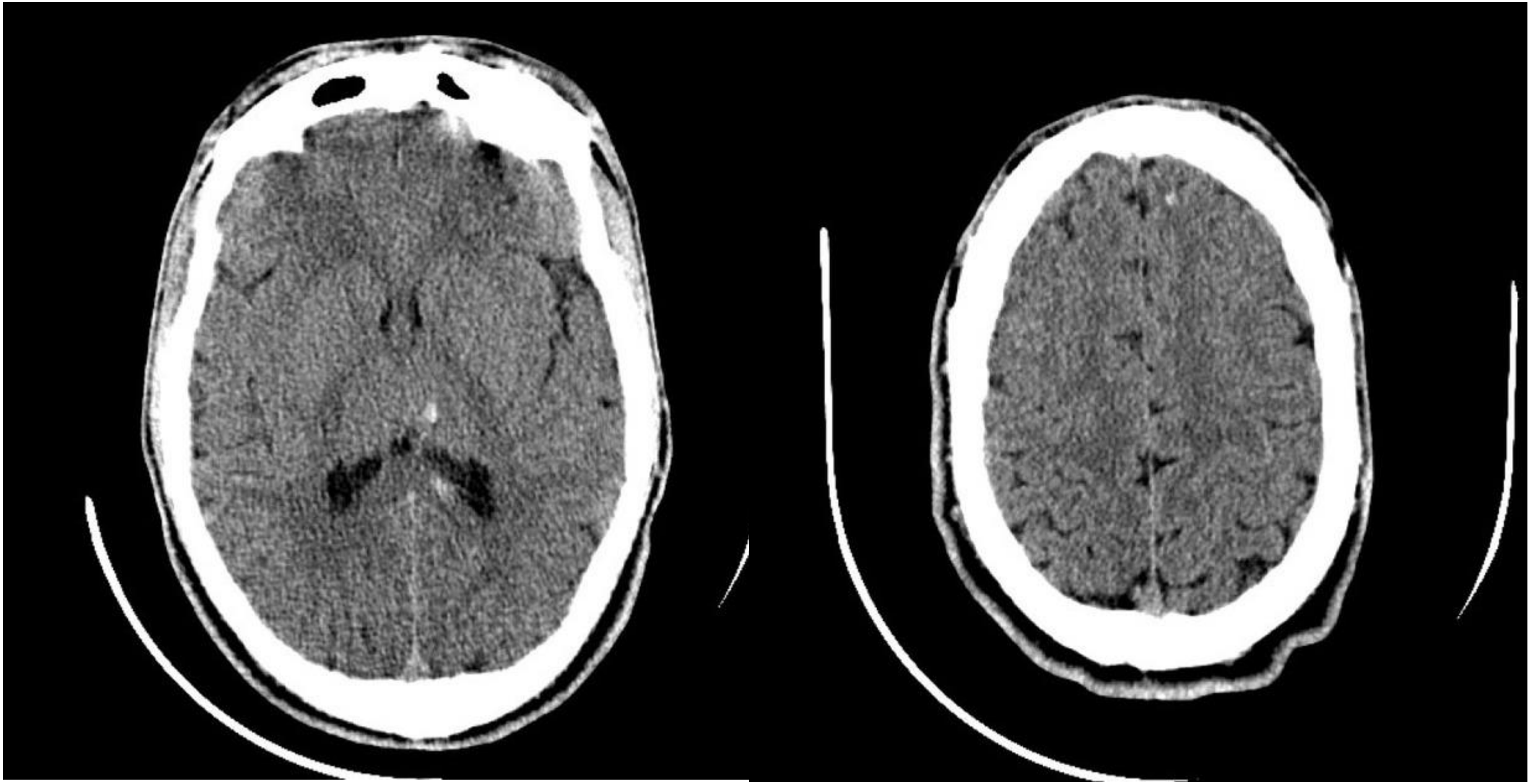
- **Symptoms:**
- Somatic: Headache, dizziness, nausea, fatigue, photophobia
- Cognitive: Memory issues, 'brain fog', poor concentration
- Emotional: Irritability, depression, anxiety, sleep problems

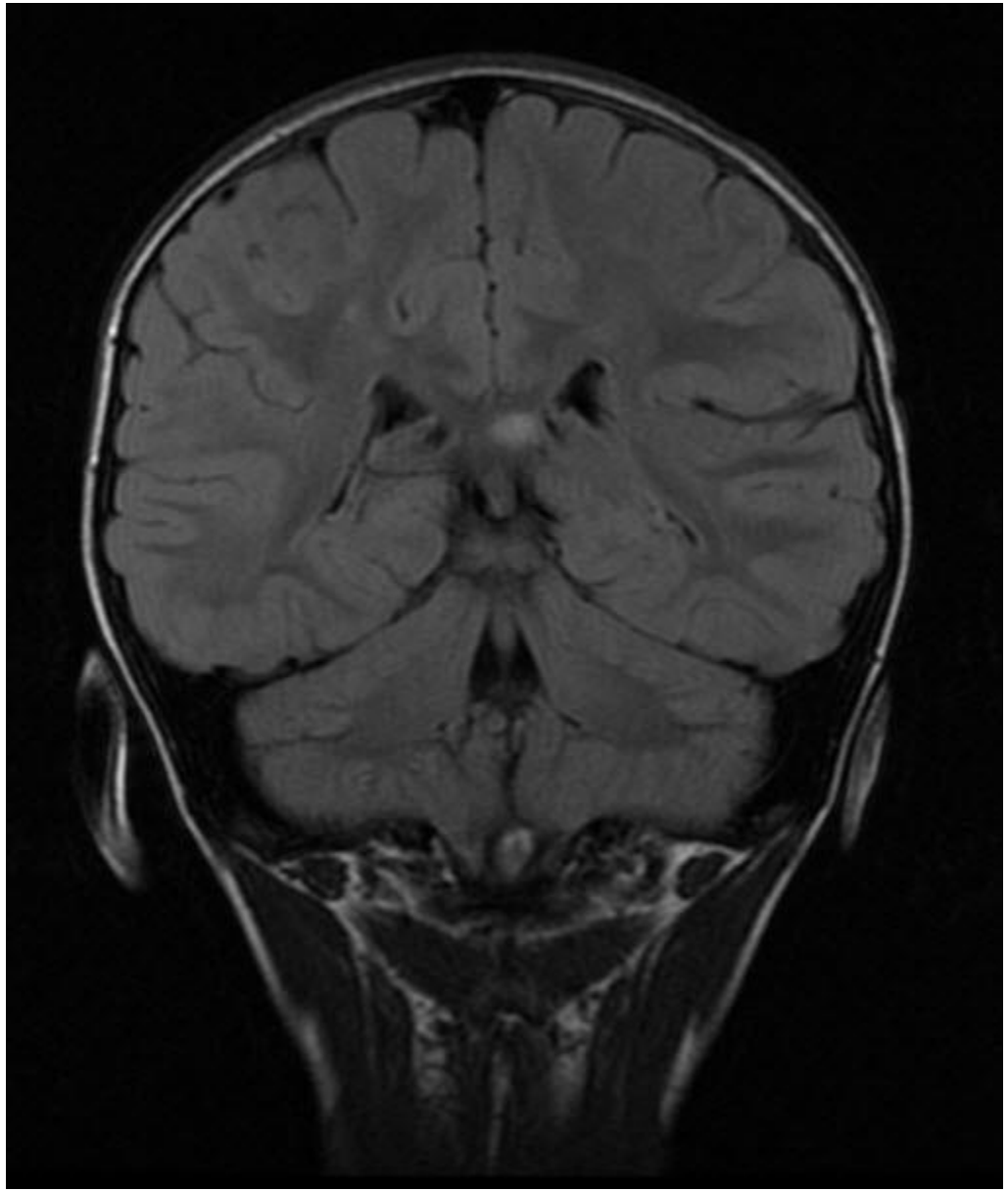
- **Diagnosis:**
- Clinical; no imaging abnormalities
- Rule out depression, PTSD, migraine, vestibular causes

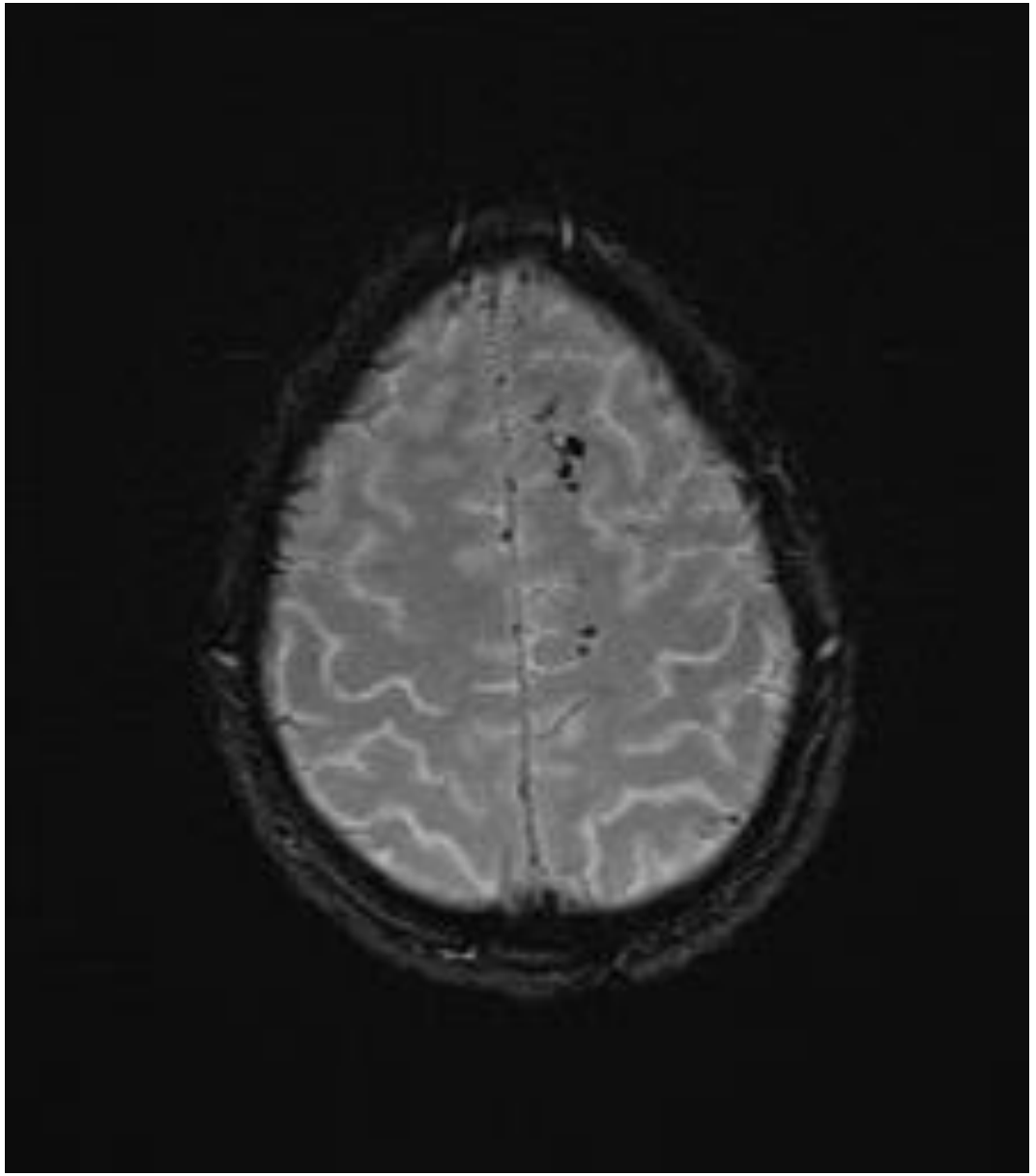
- **Management:**
- Reassurance and education
- Gradual return to activities
- Symptom-based medications (e.g., SSRIs, analgesics)
- Multidisciplinary care for persistent cases

Case II

28M post high-speed MVC, GCS 6, intubated, no external head trauma







Grades of Diffuse Axonal Injuries

Grade 1

- Mildest form of DAI
- Microscopic changes in the white matter of the cerebral cortex, corpus callosum, brain stem, and cerebellum

Grade 2

- Moderate form of DAI
- Grossly evident focal lesions isolated to the corpus callosum

Grade 3

- Severe form of DAI
- Additional and severe focal lesions on the brainstem itself

DAI Overview:

- Caused by shearing forces from acceleration/deceleration
- -Common sites: corpus callosum, brainstem, gray-white junction
- -Often CT-negative; MRI more sensitive

Management:

- Supportive ICU care
- Maintain ICP, CPP, oxygenation
- Early neuro-rehabilitation

- Prognosis:
- - Mild: possible recovery
- - Severe: often poor outcome, vegetative state

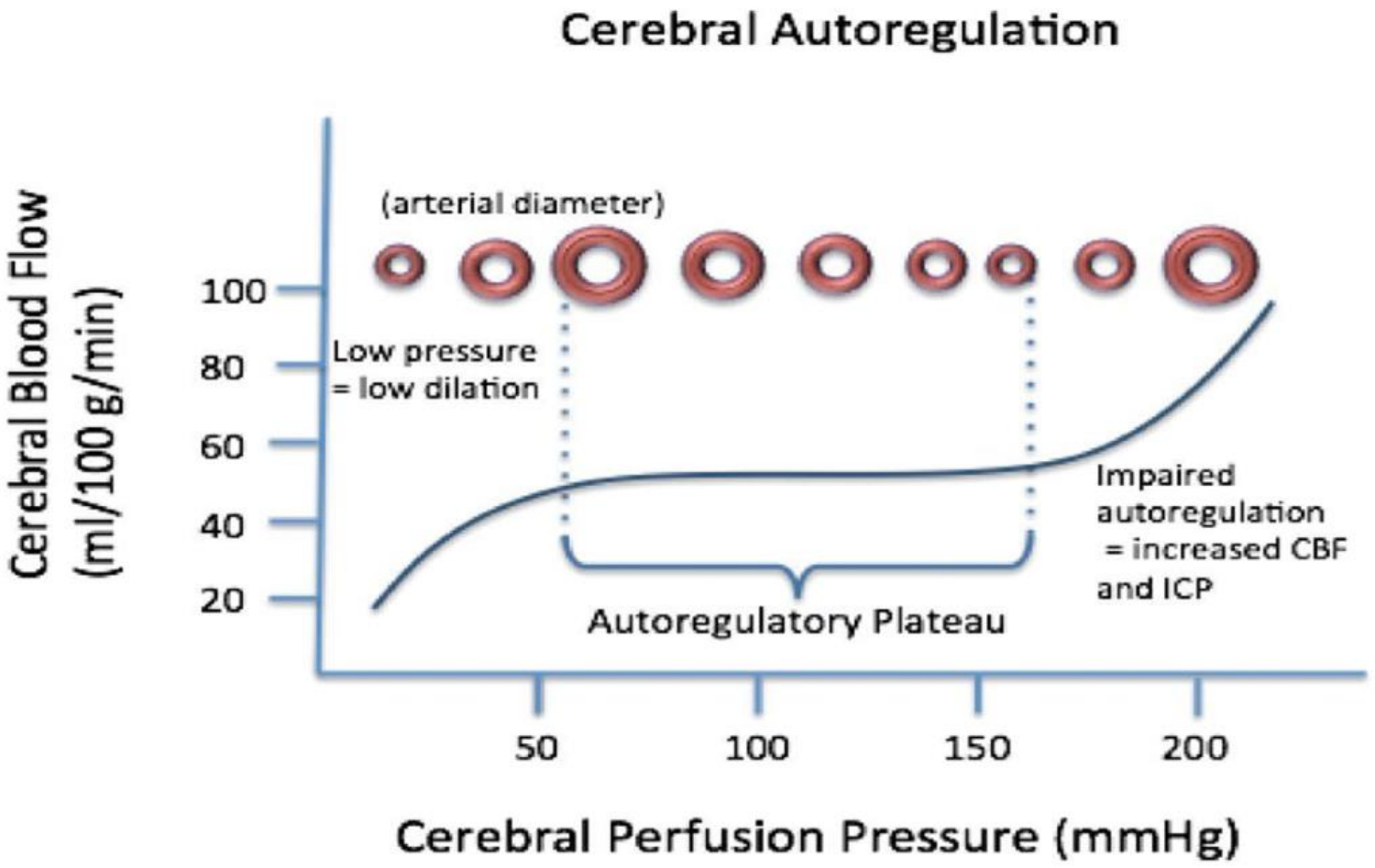
Early Manifestations

- *Change in level of consciousness*: restless, confusion, agitation;
- Decrease in Glasgow coma scale score;
- Headache;
- Nausea/vomiting;
- Slow/slurred speech;
- Diplopia/double vision; and
- *Pupillary changes*: unilateral change in size and shape, sluggish reaction to light.

Late Manifestations

- *Progressive decline in LOC*: stuperous to coma;
- Projectile vomiting without nausea;
- Significant speech impairment;
- *Abnormal motor posture*: decortications, decerebration;
- Unilateral/bilateral pupil: enlarged and fixed;
- Irregular respiration;
- *Abnormal reflexes*: Babinski;
- Cardiac arrhythmias; and
- *Cushing's triad*: (1) Systolic hypertension-widened pulse pressure, (2) Bradycardia, (3) Irregular respiration.

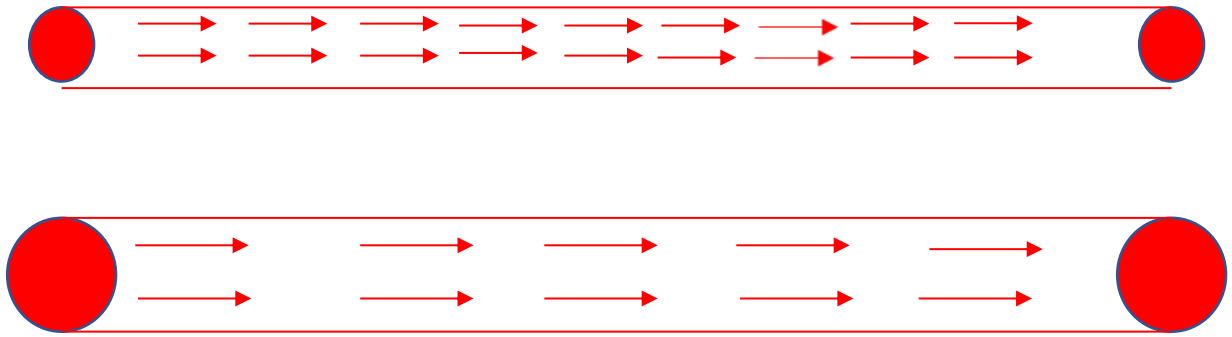
FIGURE 10. Cerebral autoregulation.

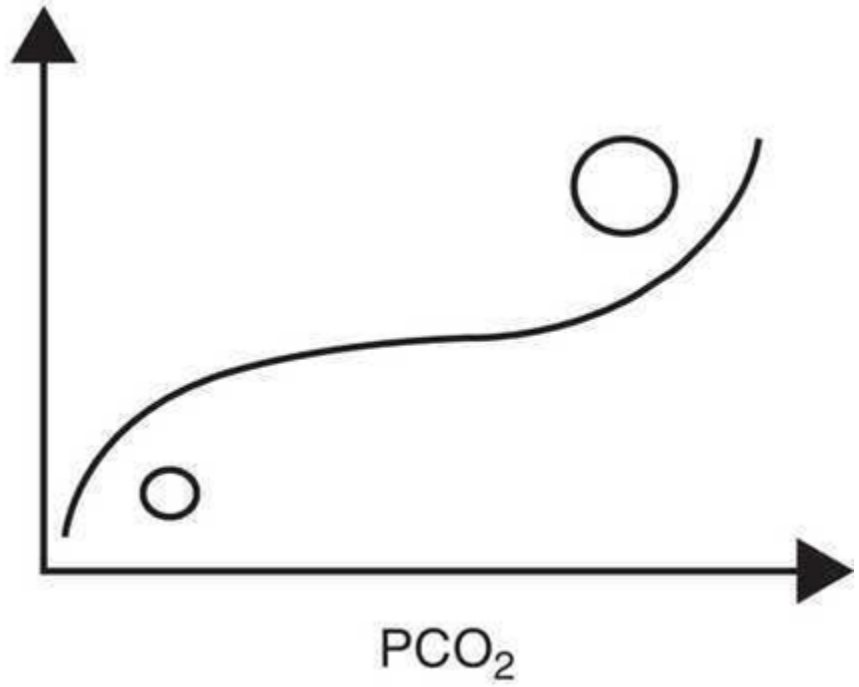


Operative Neurosurgery, Volume 17, Issue Supplement_1, August 2019, Pages S17–S44, https://doi.org/10.1093/ons/opz090

$$CBF = \frac{CPP}{CVR}$$

$$R = 8 L \eta / \pi r^4$$





Cerebral metabolic rate of oxygen (CMRO2) is the rate of oxygen consumption by the brain and is thought to be a direct index of energy homeostasis and brain health.

3.8ml/100g (in a range of 3.1 to 5.2 - thus, 46.5 - 78ml/minute for a 1.5kg brain).

- **Prevent Seizures**
- **Treat fever**
- **Control blood sugar**
- **Sedation**

FIGURE 9. Physical and chemical influences of CBF table.

	Diameter	Resistance	Flow
↑ CO_2	↑	↓	↑
↓ CO_2	↓	↑	↓
↓ CPP	↑	↓	↑
↑ CPP	↓	↑	↓
↑ Temperature	↑	↓	↑

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FIGURE 8. Cerebral blood floor thresholds table.

CBF (ml/100g/min)	Effects
>60	CBF>tissue demand
45-60	Normal
<20	Ischemia
12	Brainstem changes
10	Cell death

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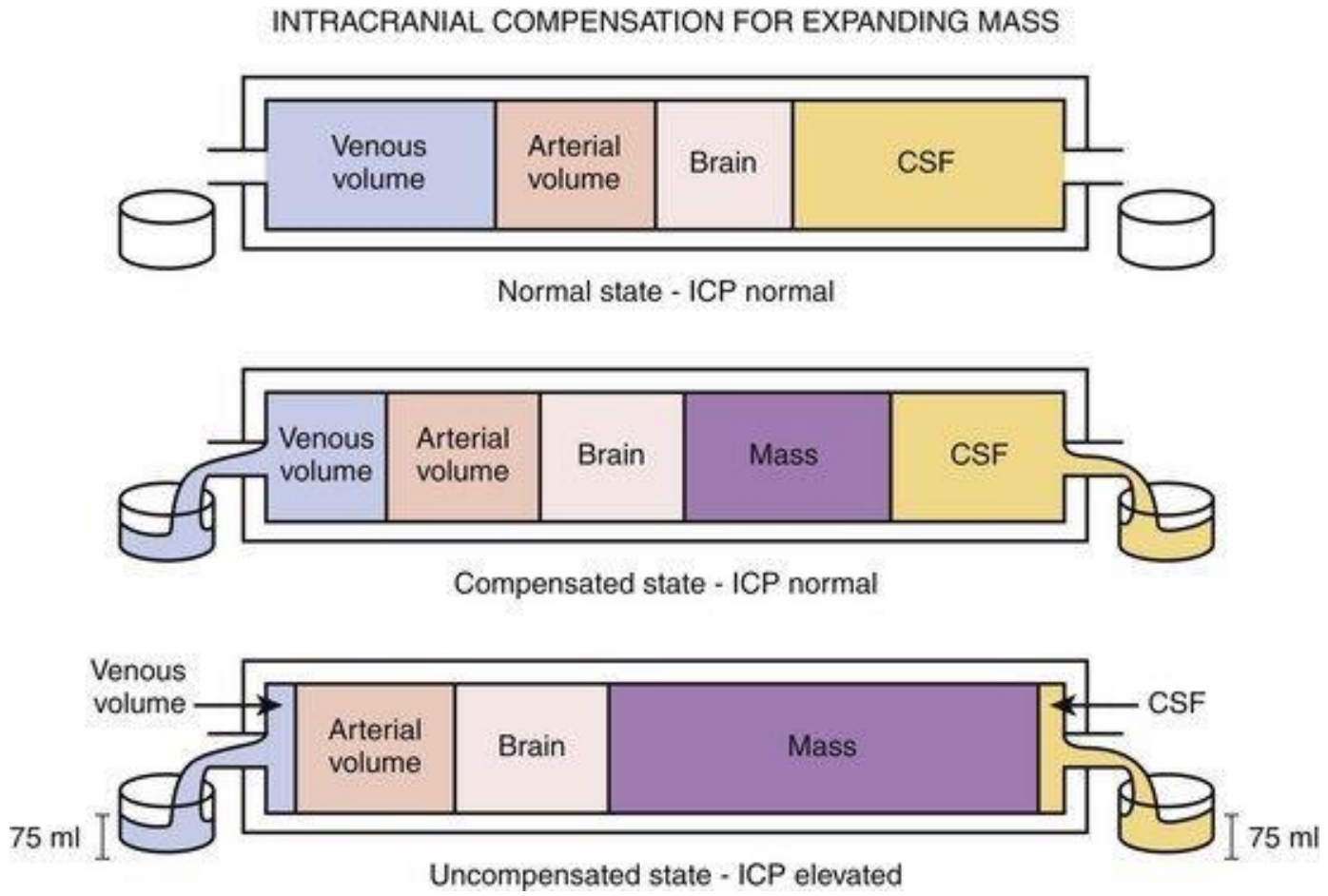
Monro-kellie doctrine



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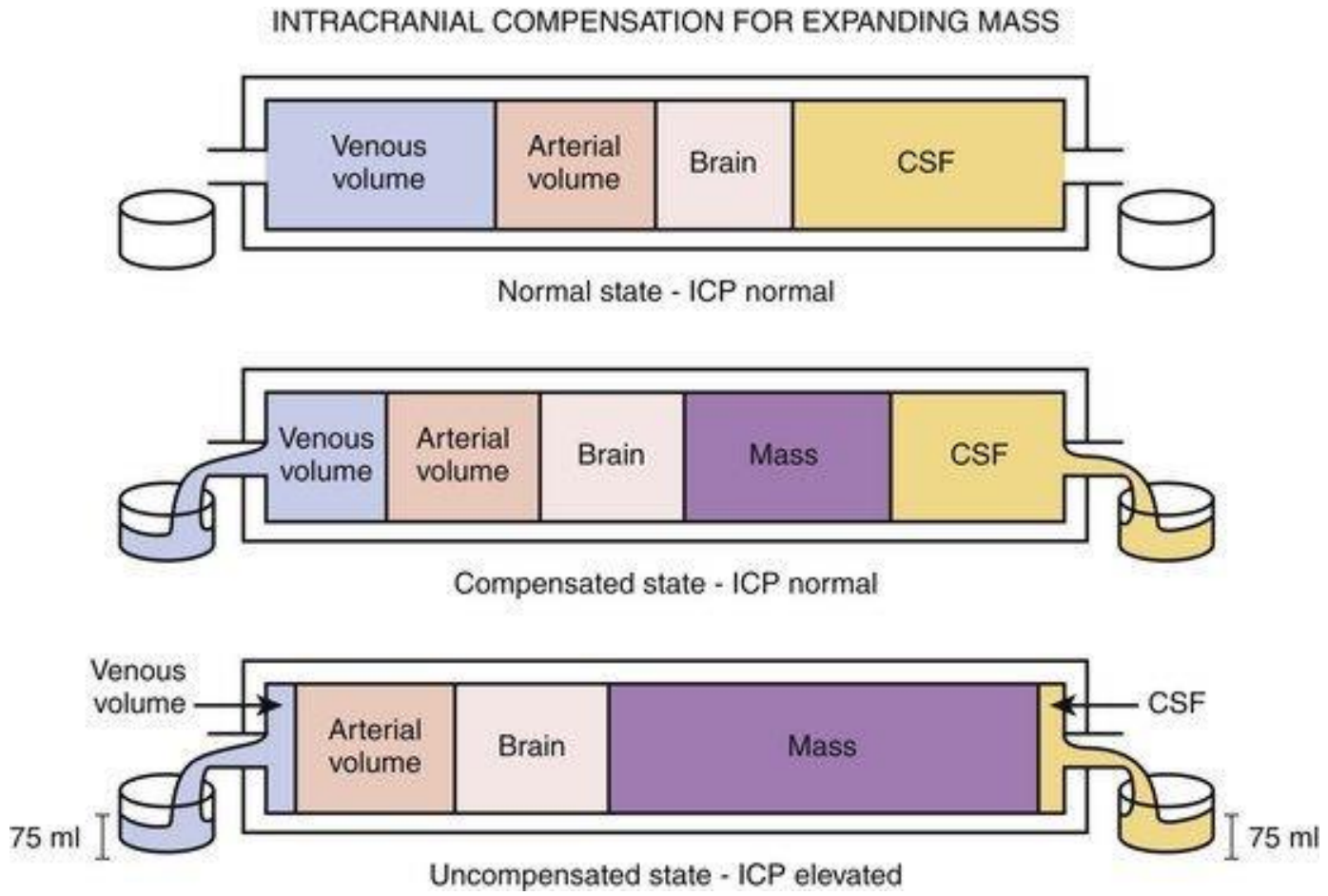


Intracranial Pressure

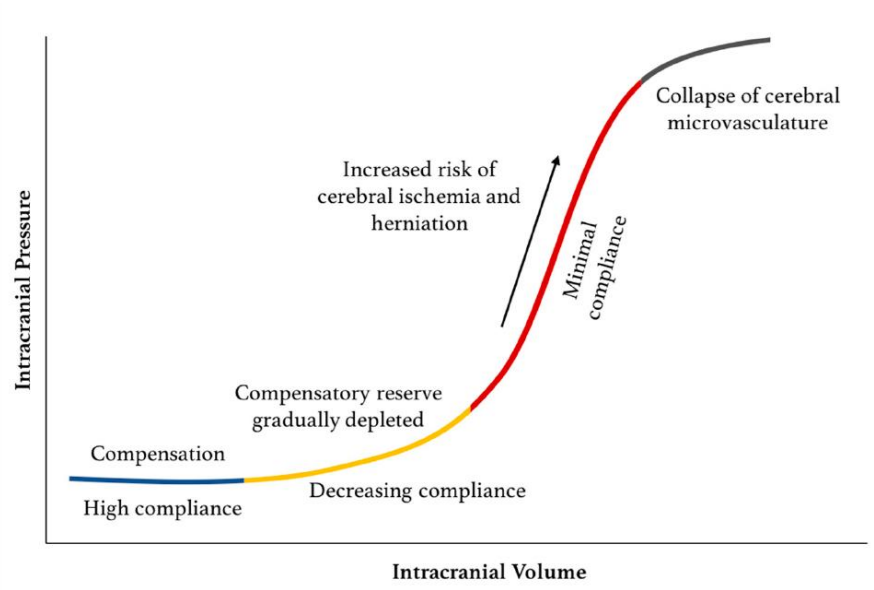
- **Definition:** Intracranial pressure (ICP) refers to the pressure within the cranial vault relative to the ambient atmospheric pressure.
- Normal ICP: adults: 10-15 mm Hg
- children: 3-7 mm Hg
- infants: 1.5-6 mm Hg
- neonates: <-2 mm Hg

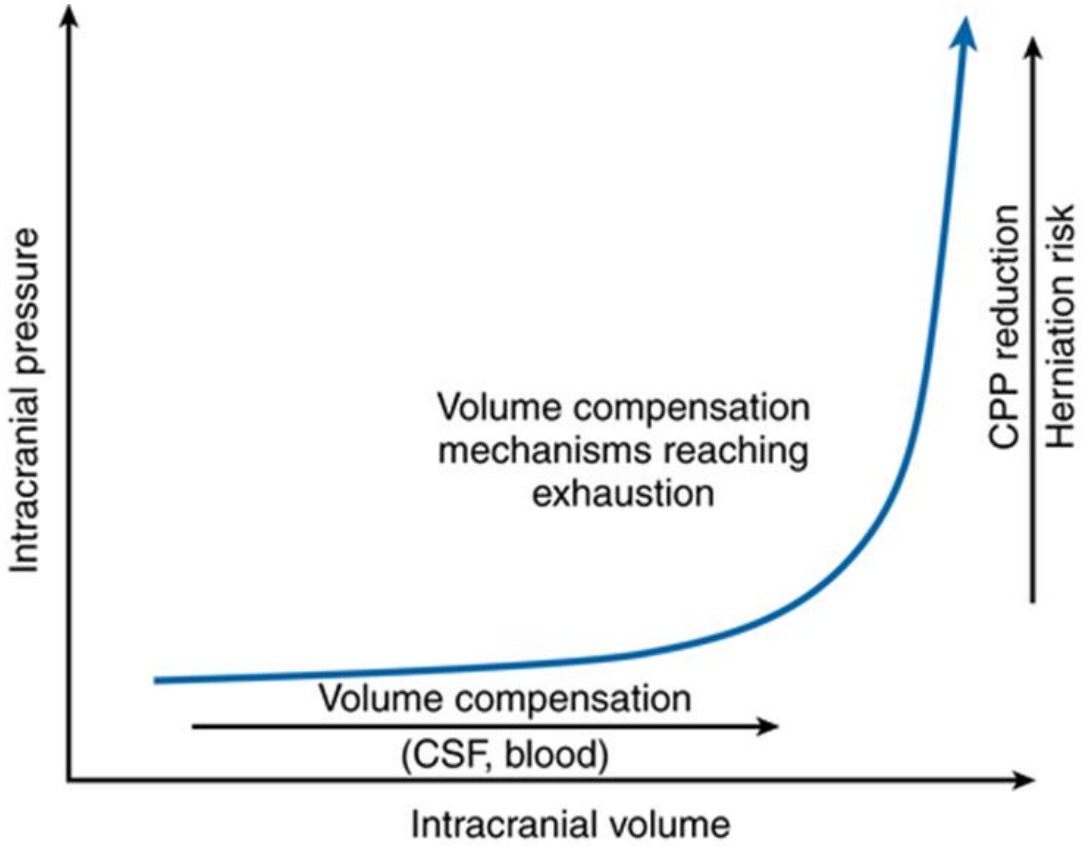
Intracranial compensation

- The brain is essentially non-compressible Any increase in intracranial volume decreases CSF or CBV
- CSF - primarily displaced into the spinal subarachnoid space
- Blood - venoconstriction of CNS capacitance vessels displaces blood in jugular venous system

Exhaustion of compensation

- Once these limited homeostatic mechanisms are exhausted additional small increases in intracranial volume produce marked elevations in ICP
- Raised ICP may decrease CPP & CBF eventually cerebral herniation & death


Intracranial Pressure
Intracranial Volume
Compensation
High compliance
Compensatory reserve gradually depleted
Decreasing compliance
Increased risk of cerebral ischemia and herniation
Minimal compliance
Collapse of cerebral microvasculature


Intracranial pressure
Intracranial volume
Volume compensation mechanisms reaching exhaustion
Volume compensation
(CSF, blood)
CPP reduction
Herniation risk

Meninges

Dura mater

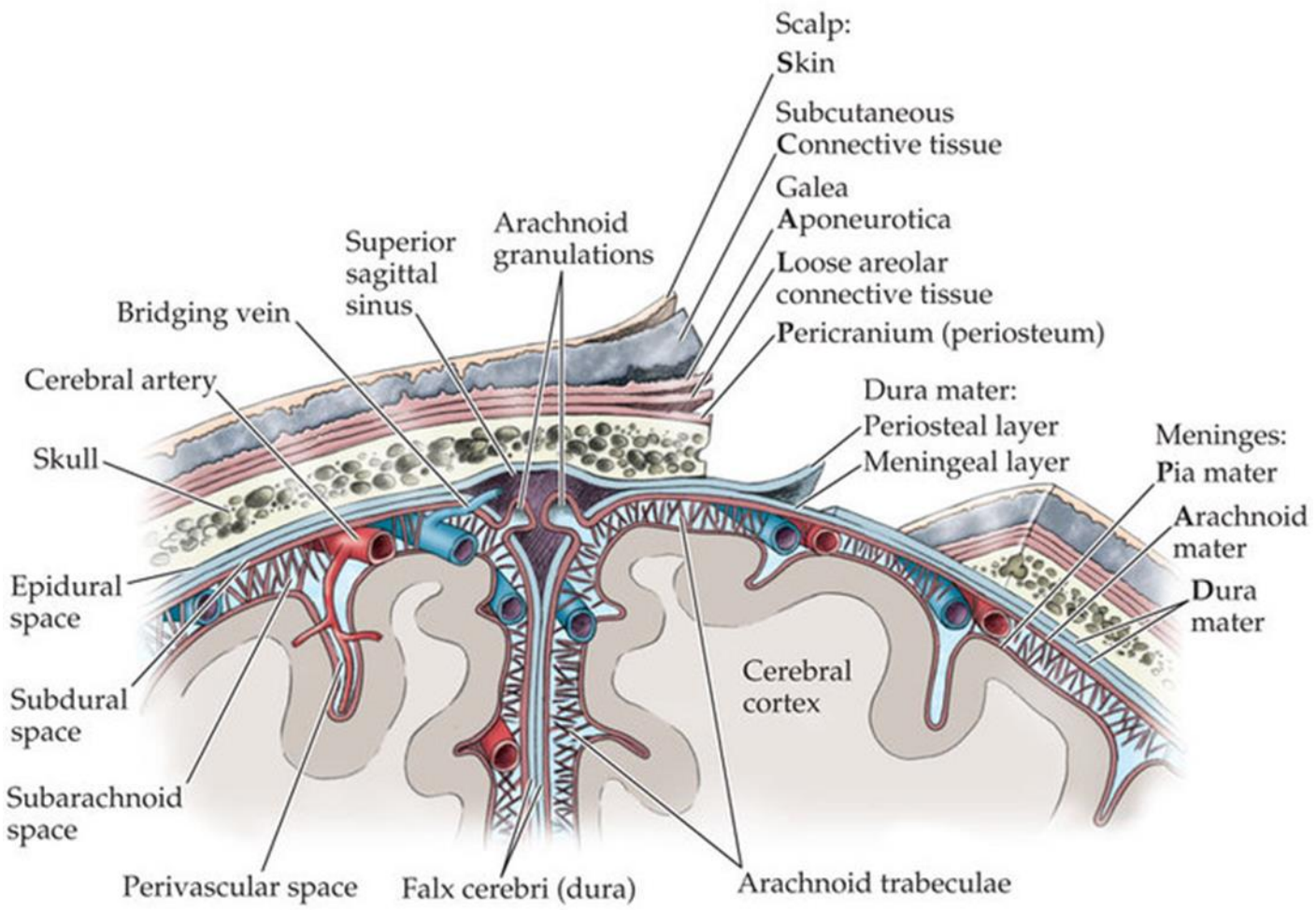
Arachnoid mater

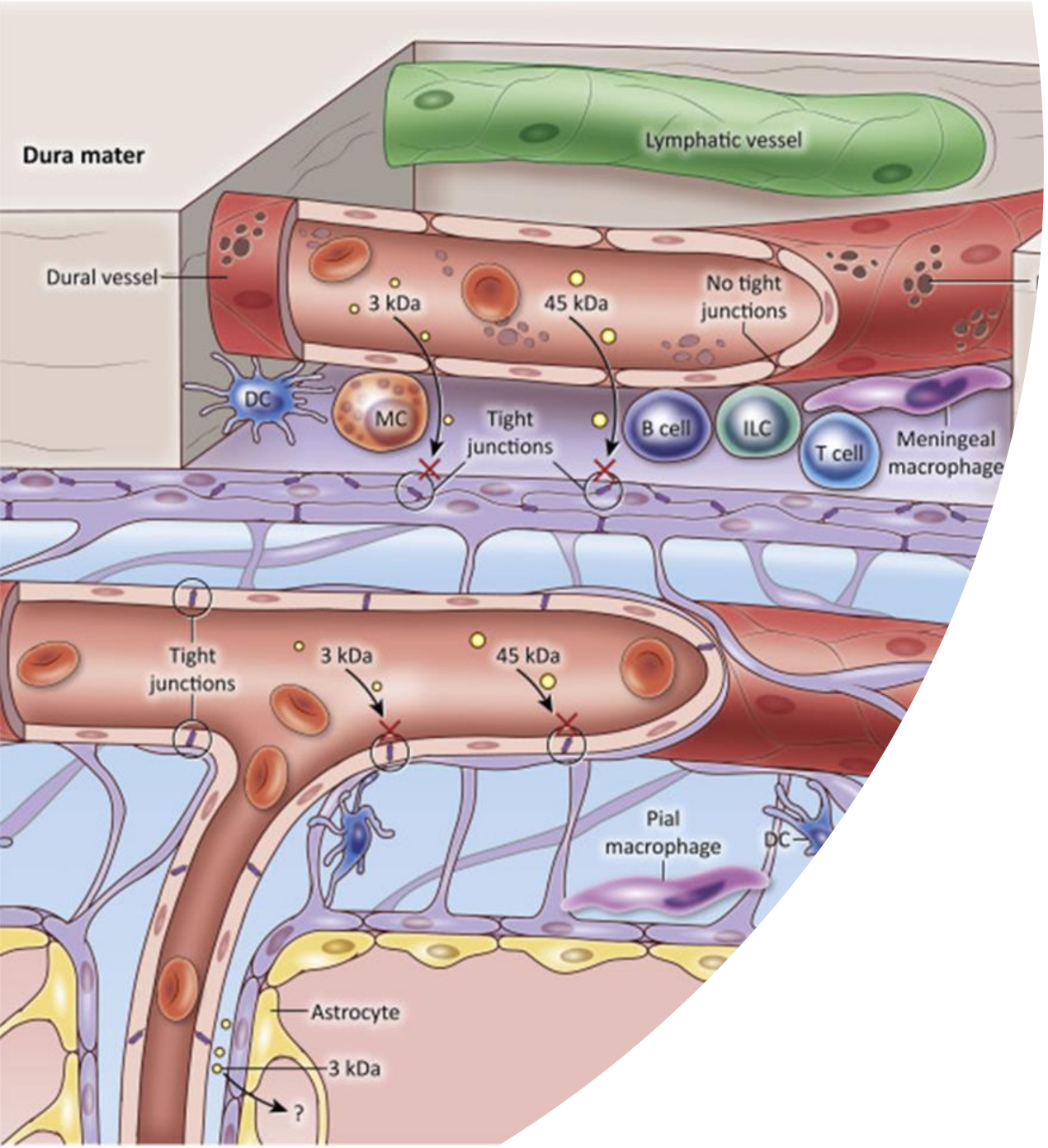
Pia mater

Cranium

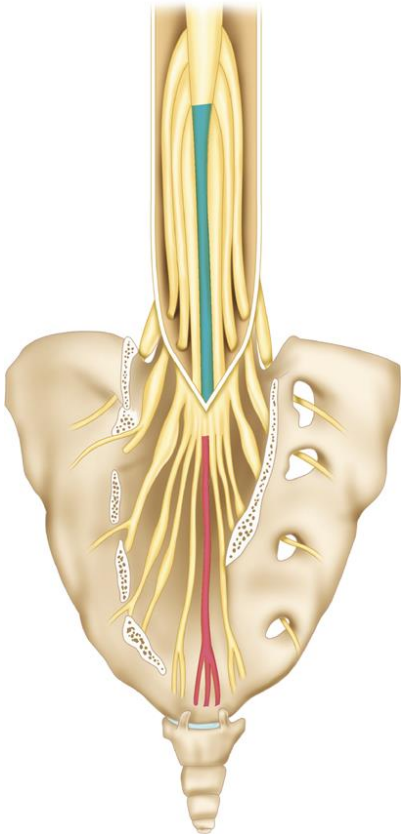
Meningeal dura

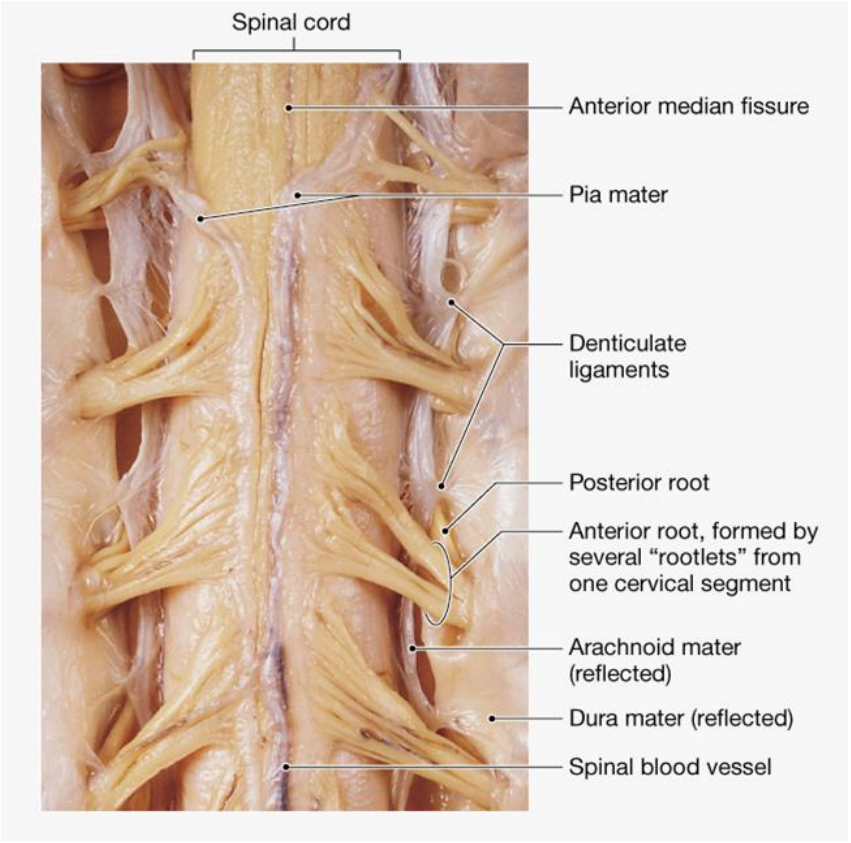
Periosteal dura

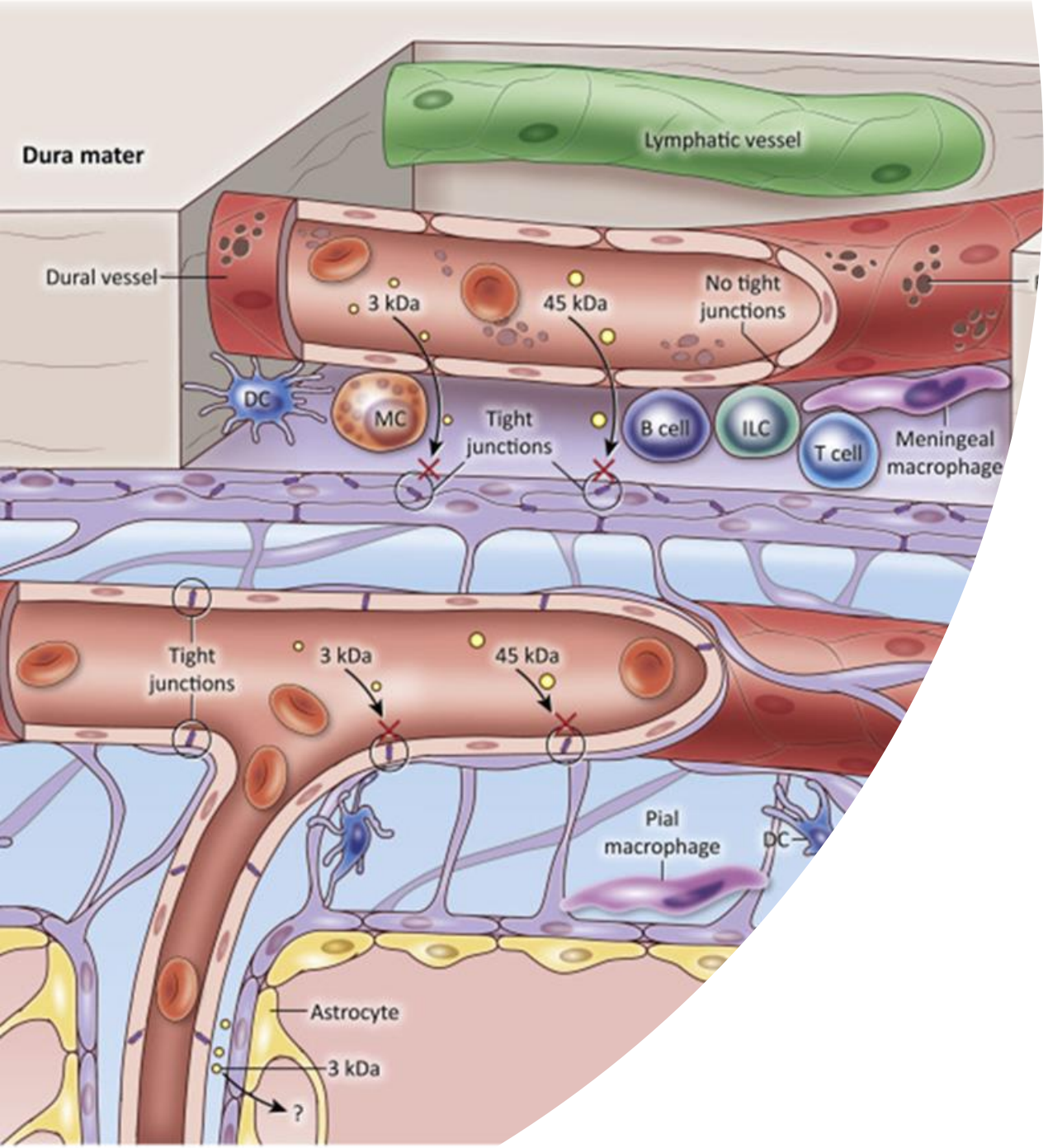


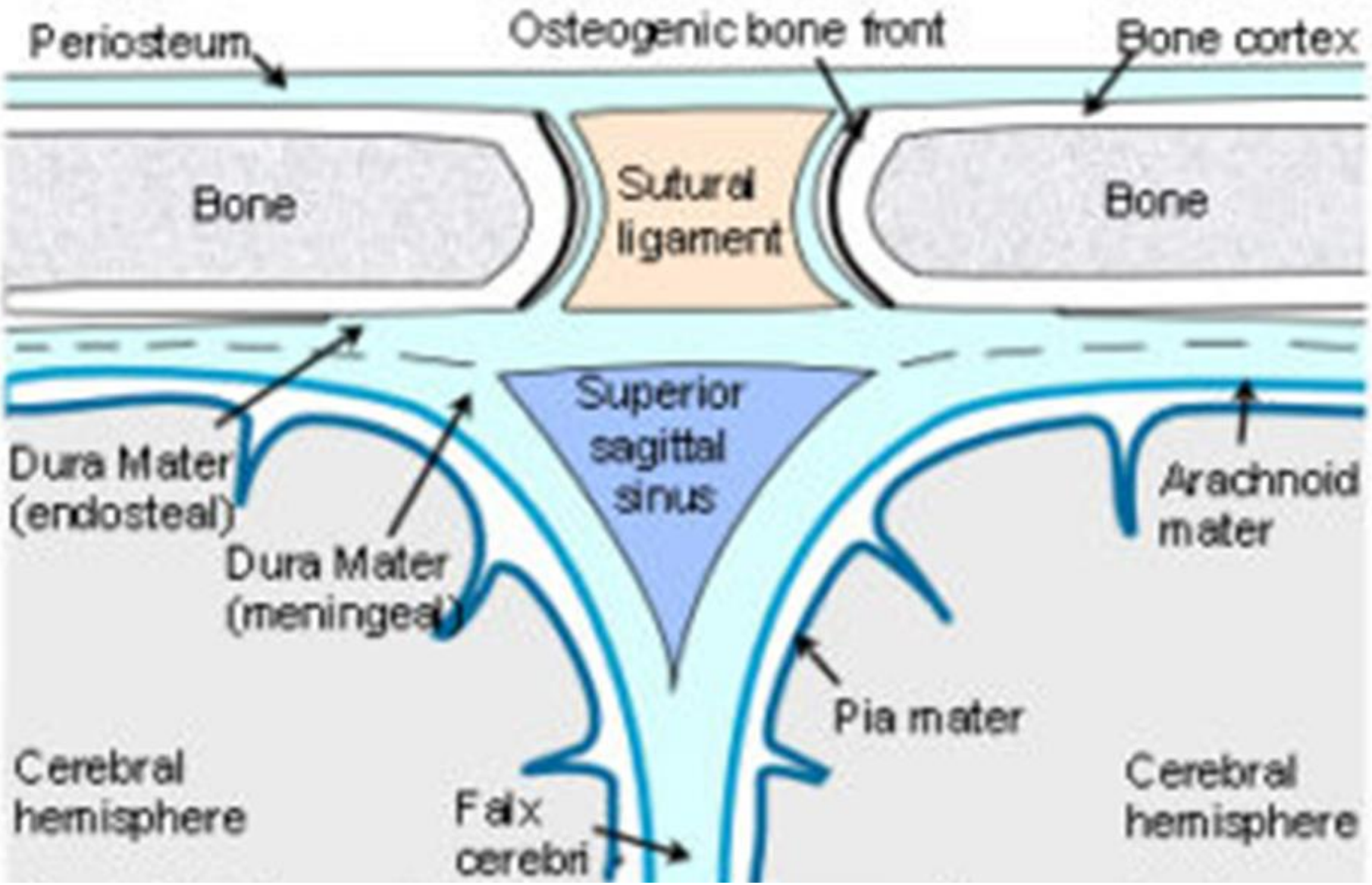

Dura mater
Lymphatic vessel
Dural vessel
3 kDa
45 kDa
No tight junctions
DC
MC
Tight junctions
B cell
ILC
T cell
Meningeal macrophage
Tight junctions
3 kDa
45 kDa
Pial macrophage
DC
Astrocyte
3 kDa
?

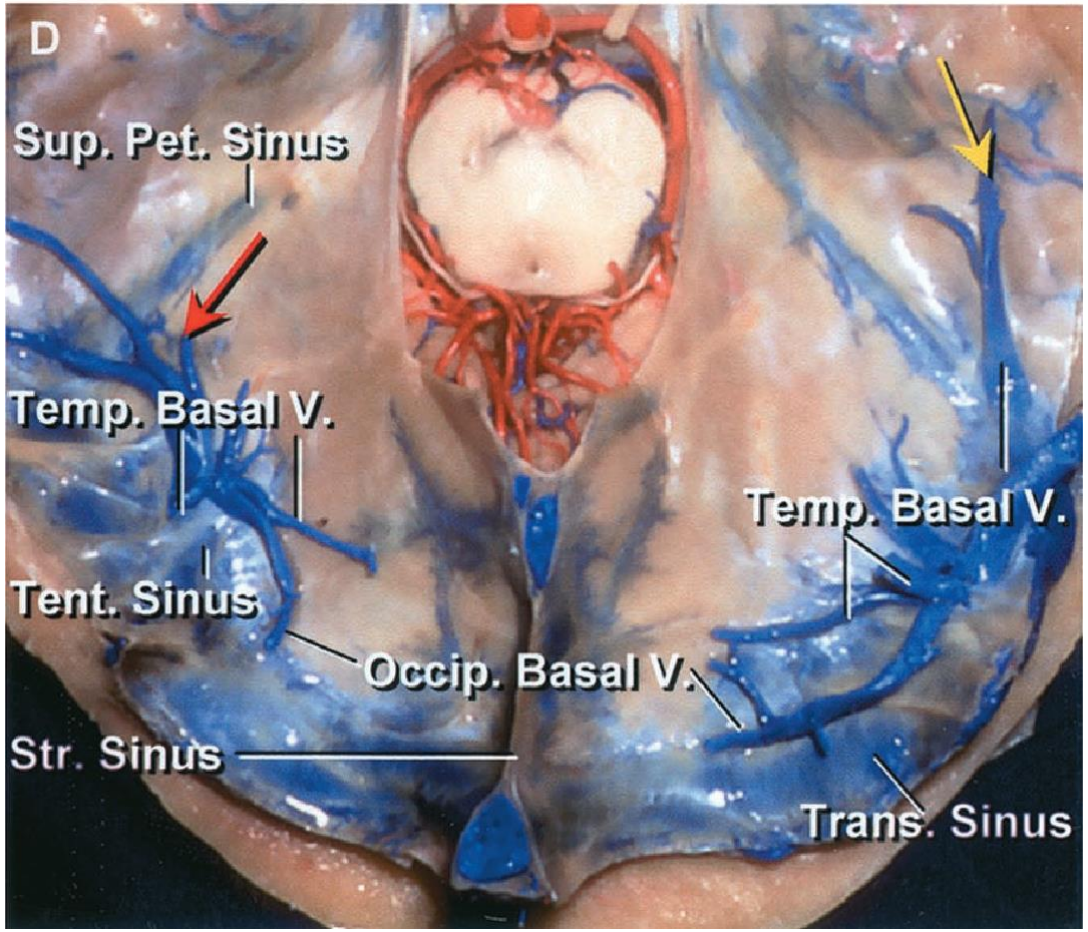
Pia Mater

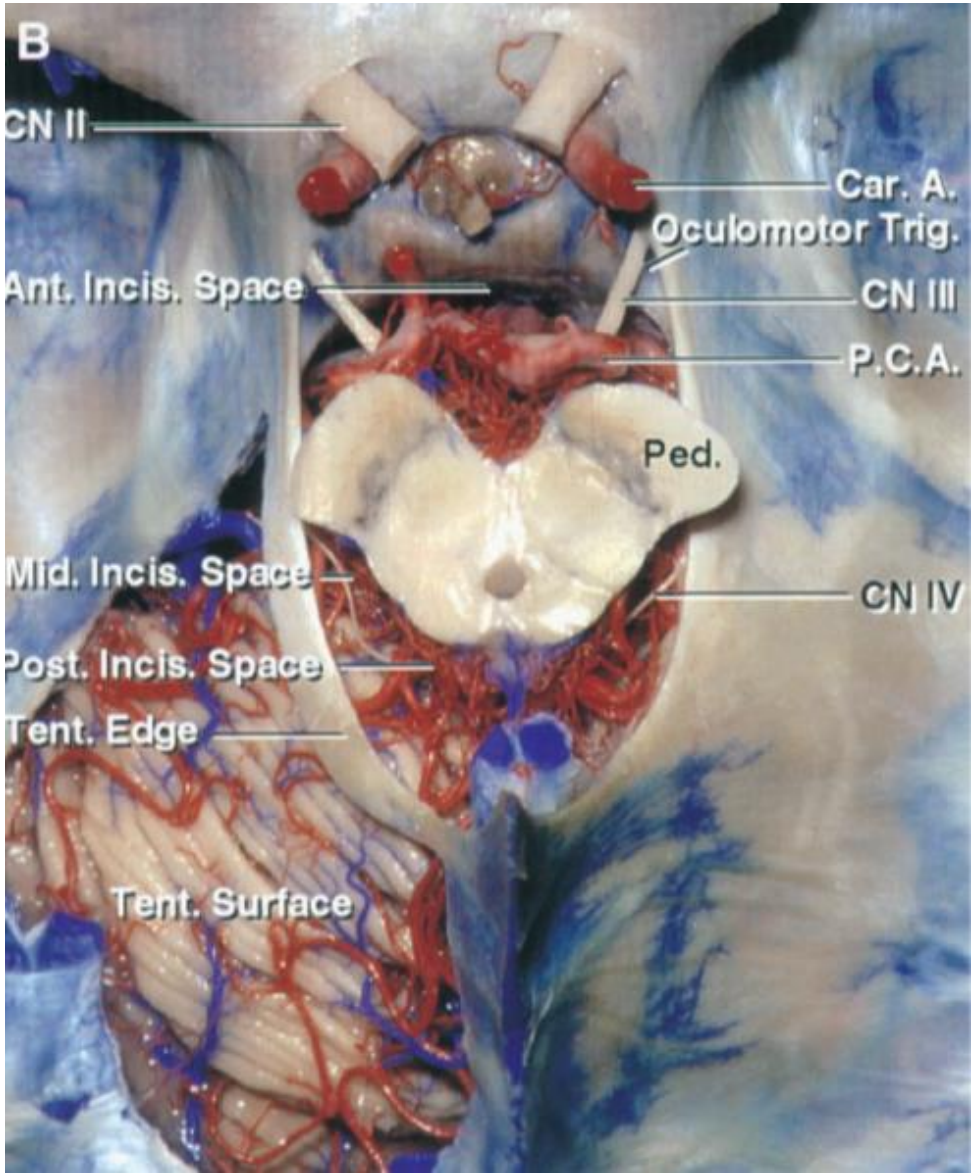


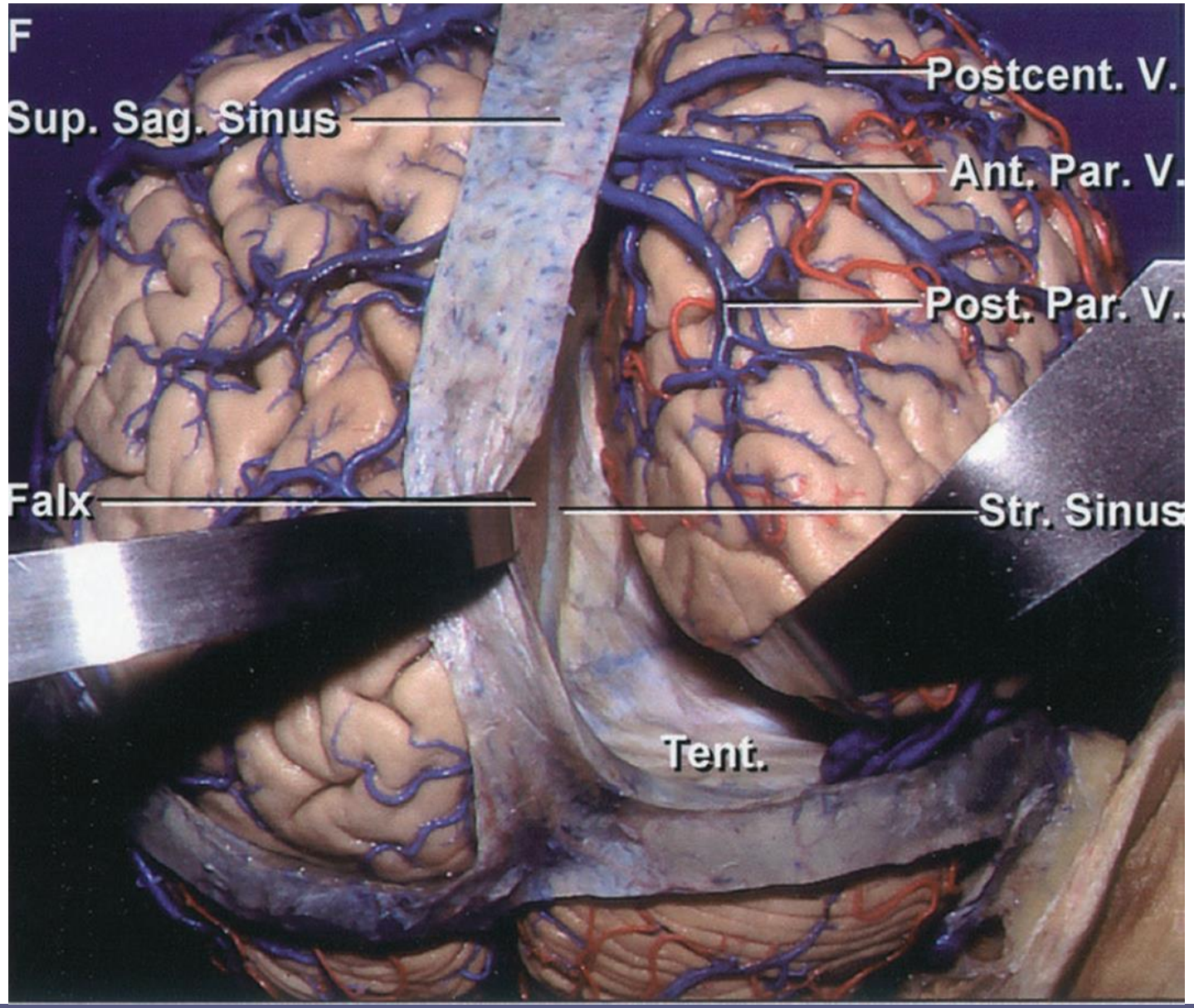

Spinal cord
Anterior median fissure
Pia mater
Denticulate ligaments
Posterior root
Anterior root, formed by several "rootlets" from one cervical segment
Arachnoid mater (reflected)
Dura mater (reflected)
Spinal blood vessel

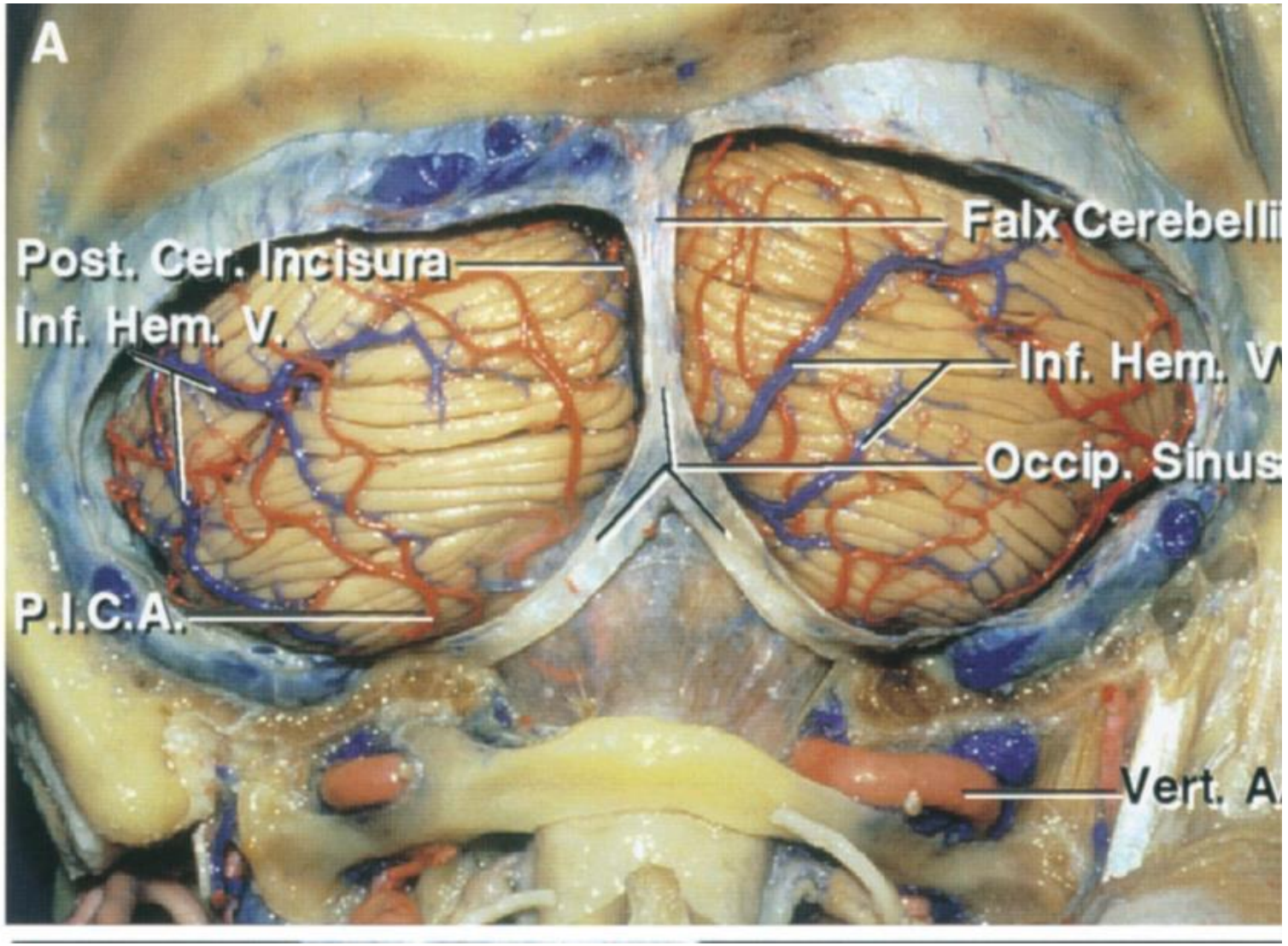

Dura mater
Lymphatic vessel
Dural vessel
3 kDa
45 kDa
No tight junctions
DC
MC
Tight junctions
B cell
ILC
T cell
Meningeal macrophage
Tight junctions
3 kDa
45 kDa
Pial macrophage
DC
Astrocyte
3 kDa
?

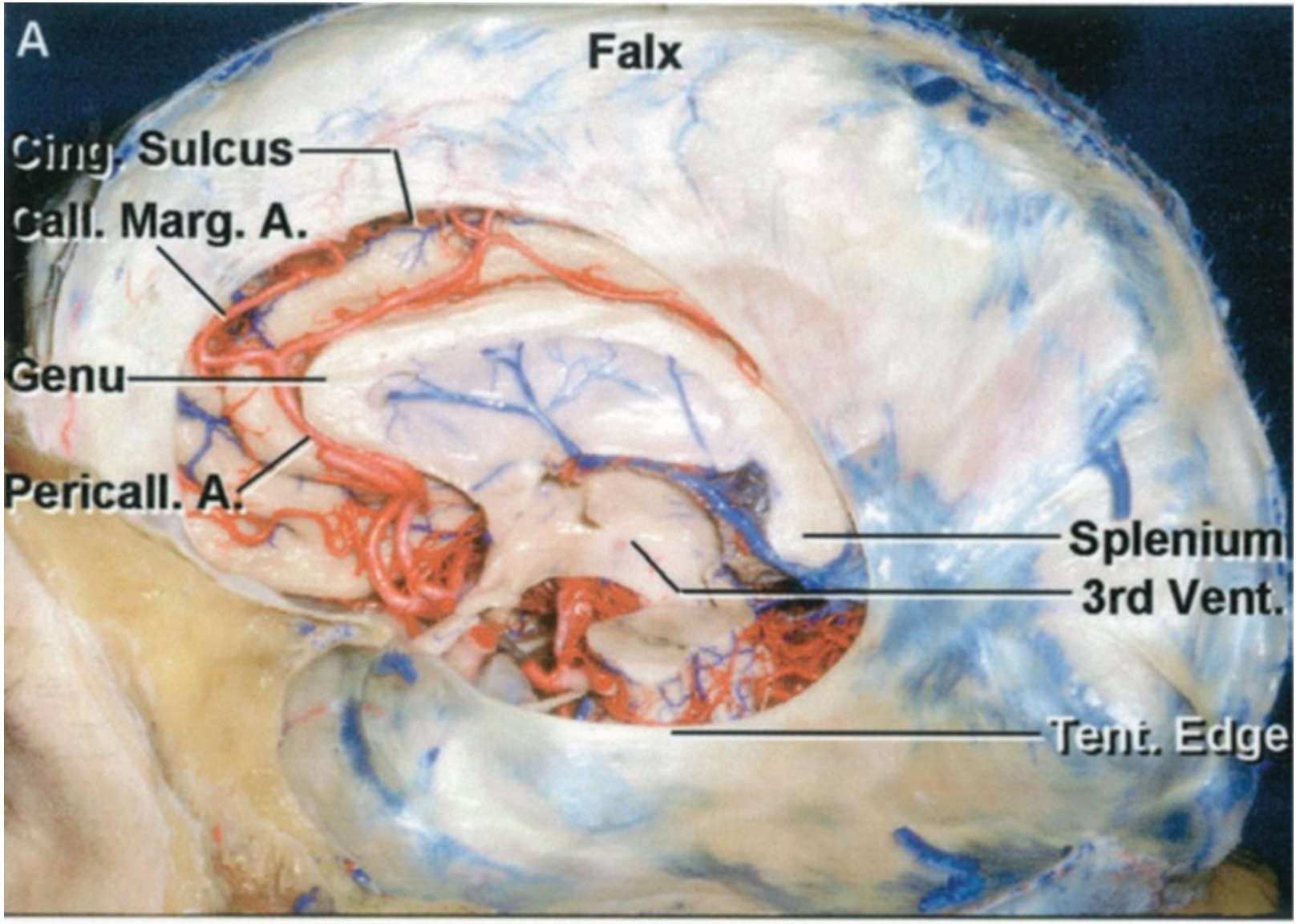

Periosteum
Osteogenic bone front
Bone cortex
Bone
Sutural ligament
Bone
Superior sagittal sinus
Dura Mater (endosteal)
Dura Mater (meningeal)
Arachnoid mater
Pia mater
Cerebral hemisphere
Falx cerebri
Cerebral hemisphere

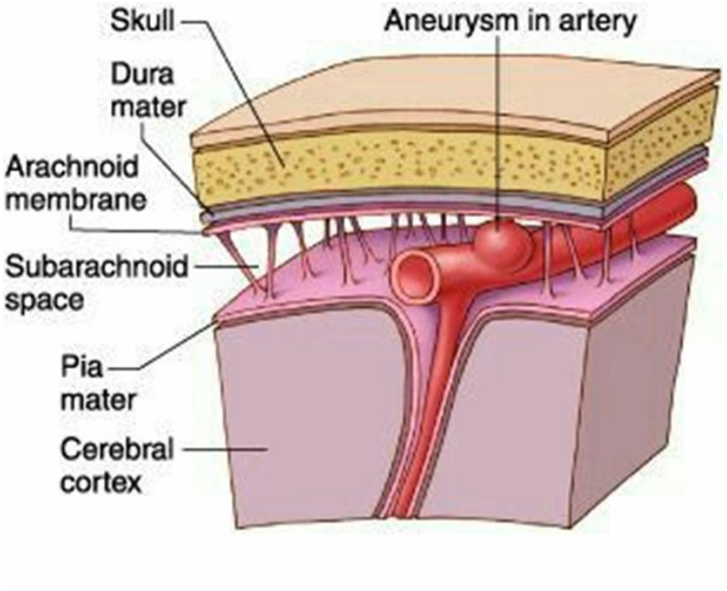

D
Sup. Pet. Sinus
Temp. Basal V.
Tent. Sinus
Occip. Basal V.
Str. Sinus
Temp. Basal V.
Trans. Sinus

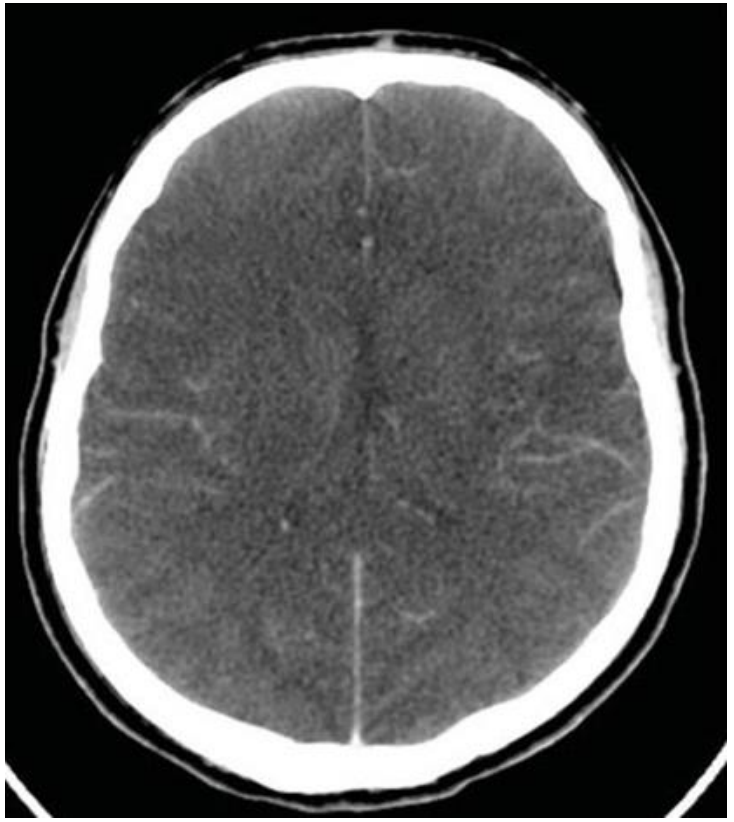

B
CN II
Car. A.
Oculomotor Trig.
Ant. Incis. Space
CN III
P.C.A.
Ped.
Mid. Incis. Space
CN IV
Post. Incis. Space
Tent. Edge
Tent. Surface


F
Sup. Sag. Sinus
Postcent. V.
Ant. Par. V.
Post. Par. V.
Falx
Str. Sinus
Tent.

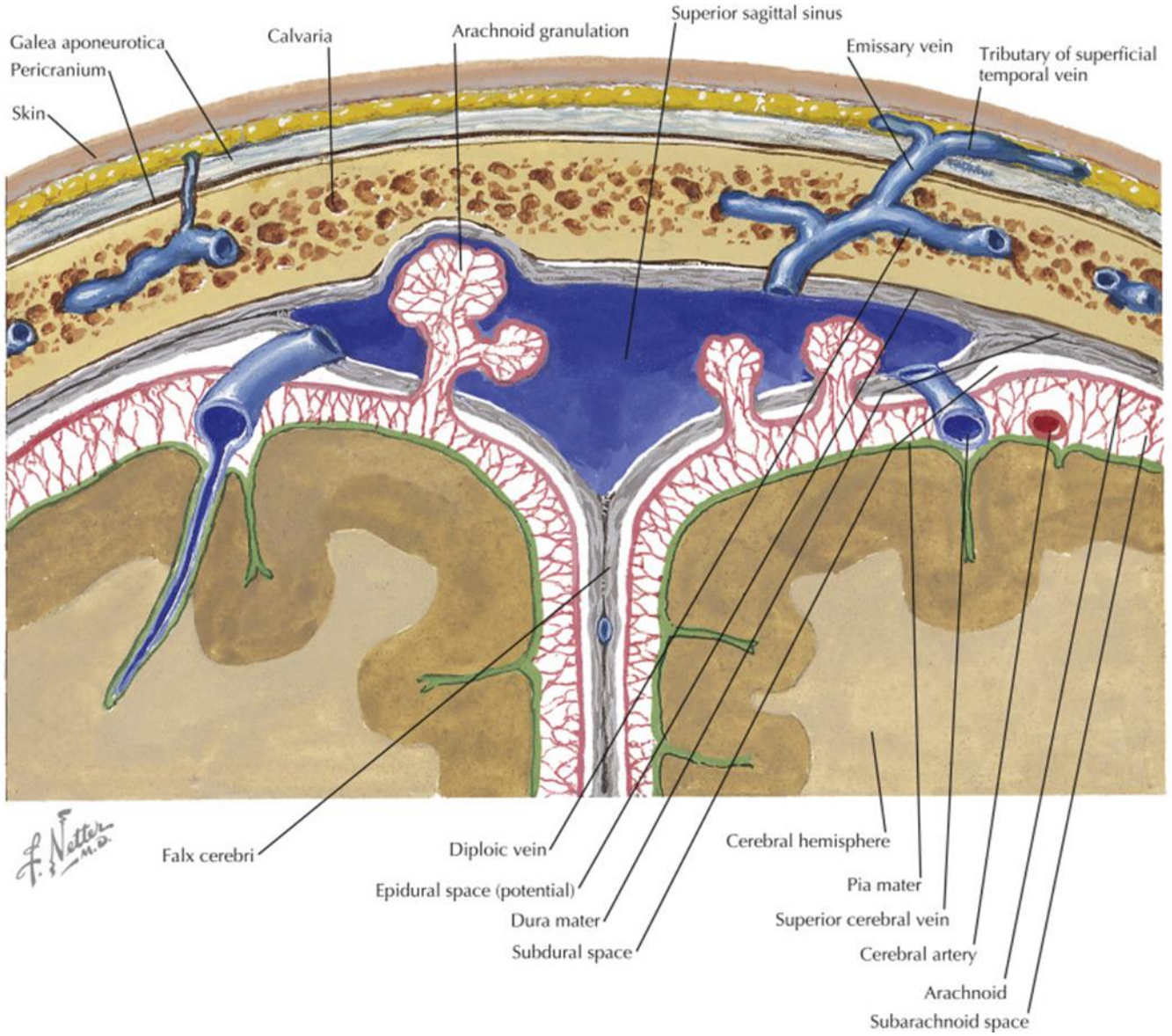

A
Falx Cerebelli
Post. Cer. Incisura
Inf. Hem. V.
Inf. Hem. V
Occip. Sinus
P.I.C.A.
Vert. A


A
Falx
Cing. Sulcus
Call. Marg. A.
Genu
Pericall. A.
Splenium
3rd Vent.
Tent. Edge

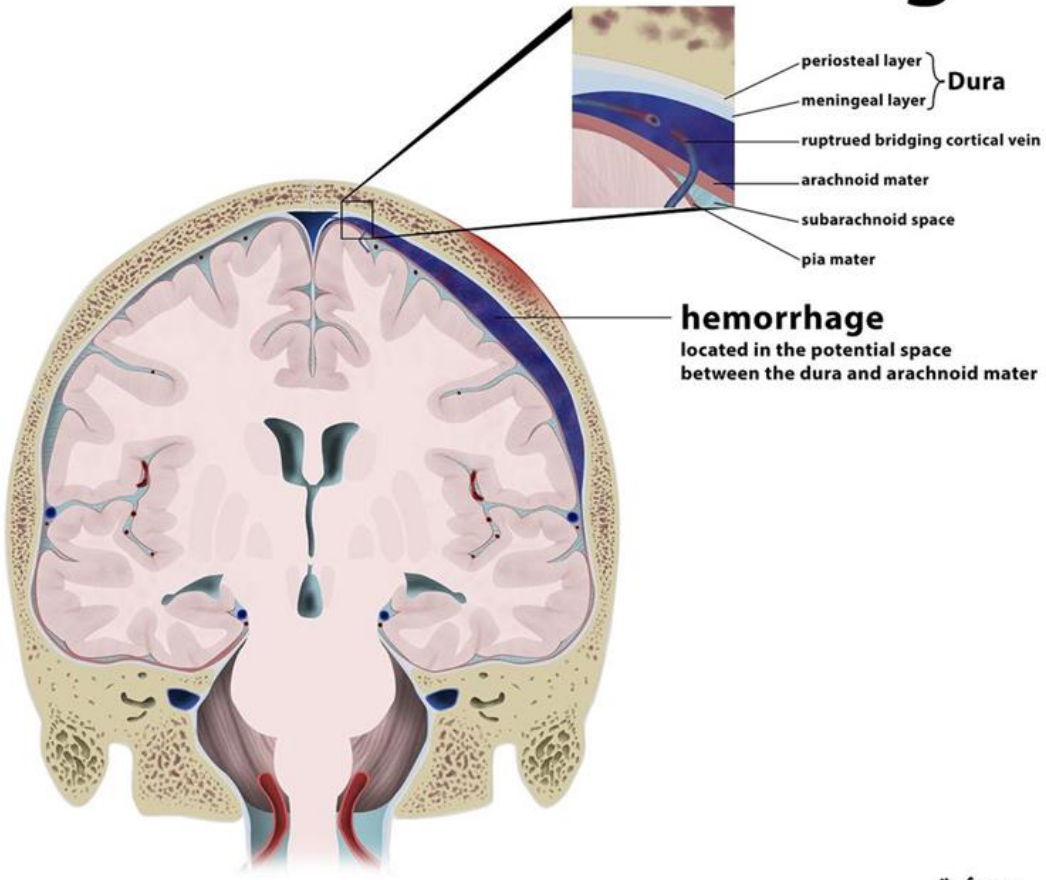




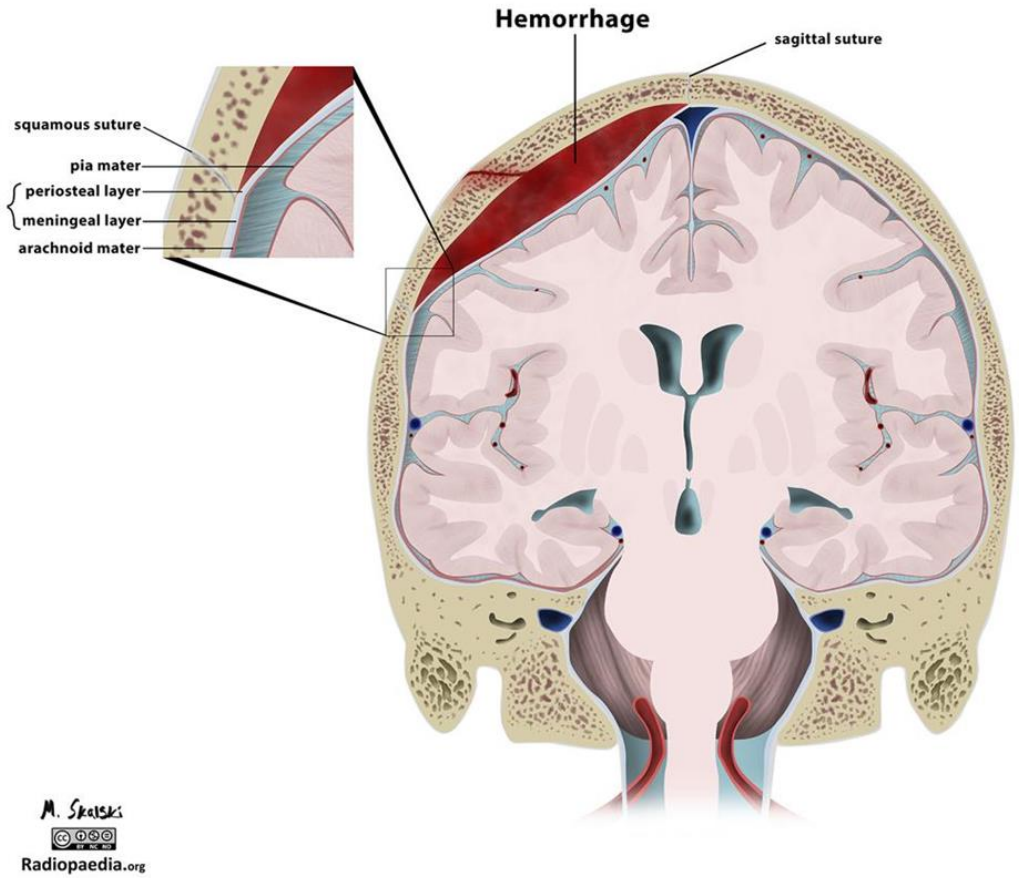
Arachnoid mater


Galea aponeurotica
Pericranium
Skin
Calvaria
Arachnoid granulation
Superior sagittal sinus
Emissary vein
Tributary of superficial temporal vein
Falx cerebri
Diploic vein
Epidural space (potential)
Dura mater
Subdural space
Cerebral hemisphere
Pia mater
Superior cerebral vein
Cerebral artery
Arachnoid
Subarachnoid space

Subdural hemorrhage



Extradural Hemorrhage



Increased ICP

Brain tissue

Tumours

Brain oedema

Cytotoxic oedema

Vasogenic oedema

Interstitial oedema

Blood

Intracranial bleeding

CSF

Hydrocephalus

Non communicating

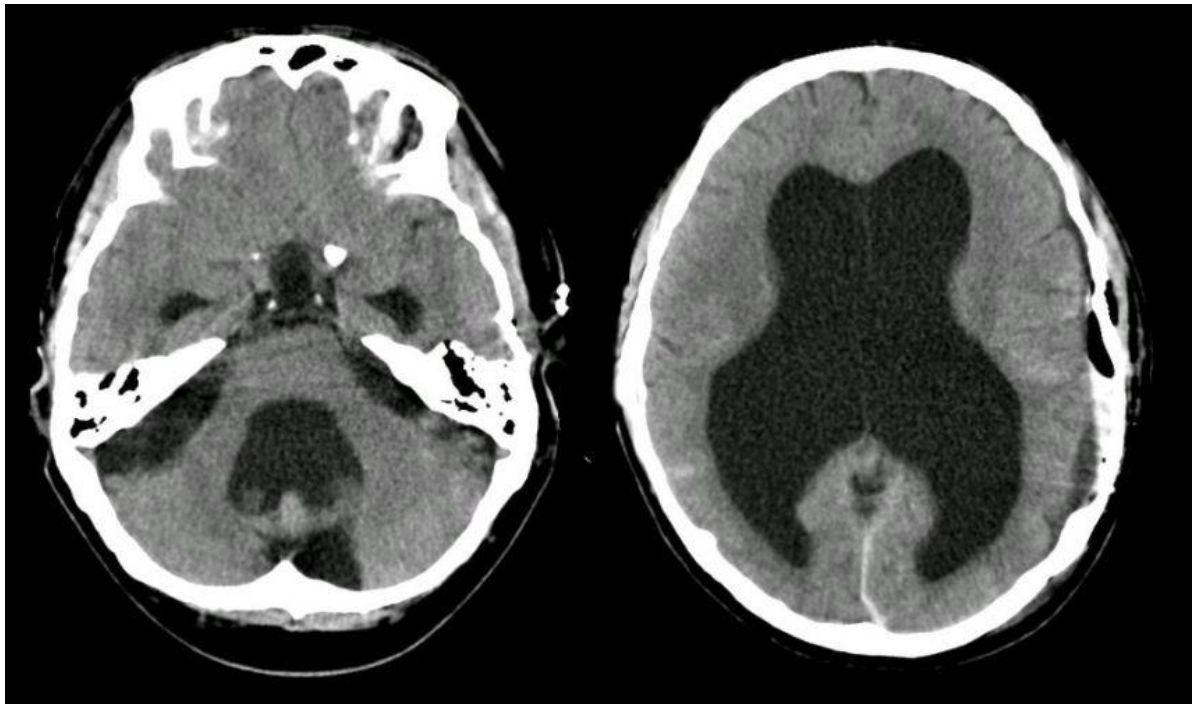
Communicating

Increased production

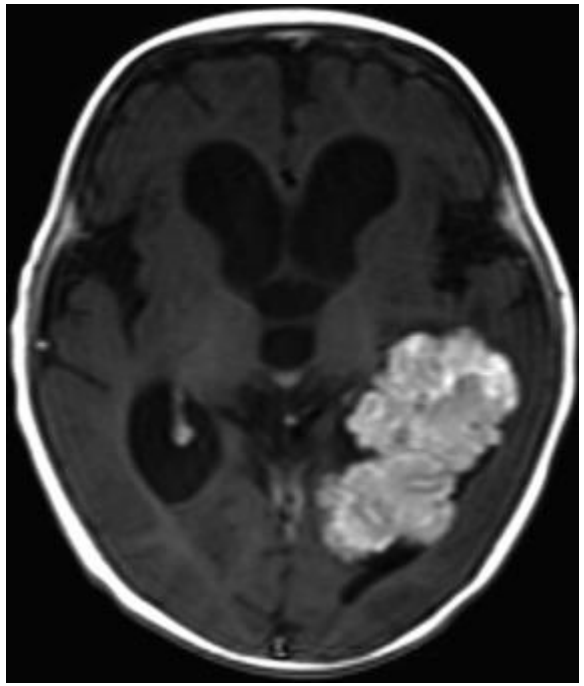
Hydrocephalus

Non communicating

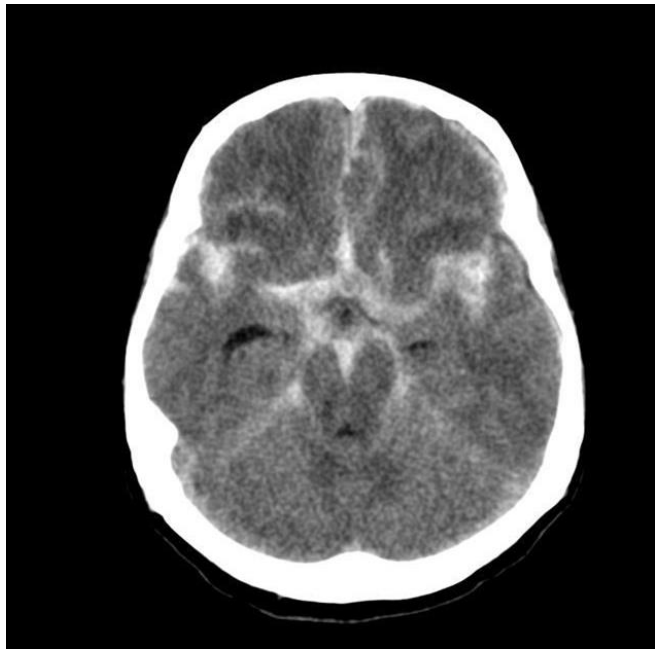
Communicating

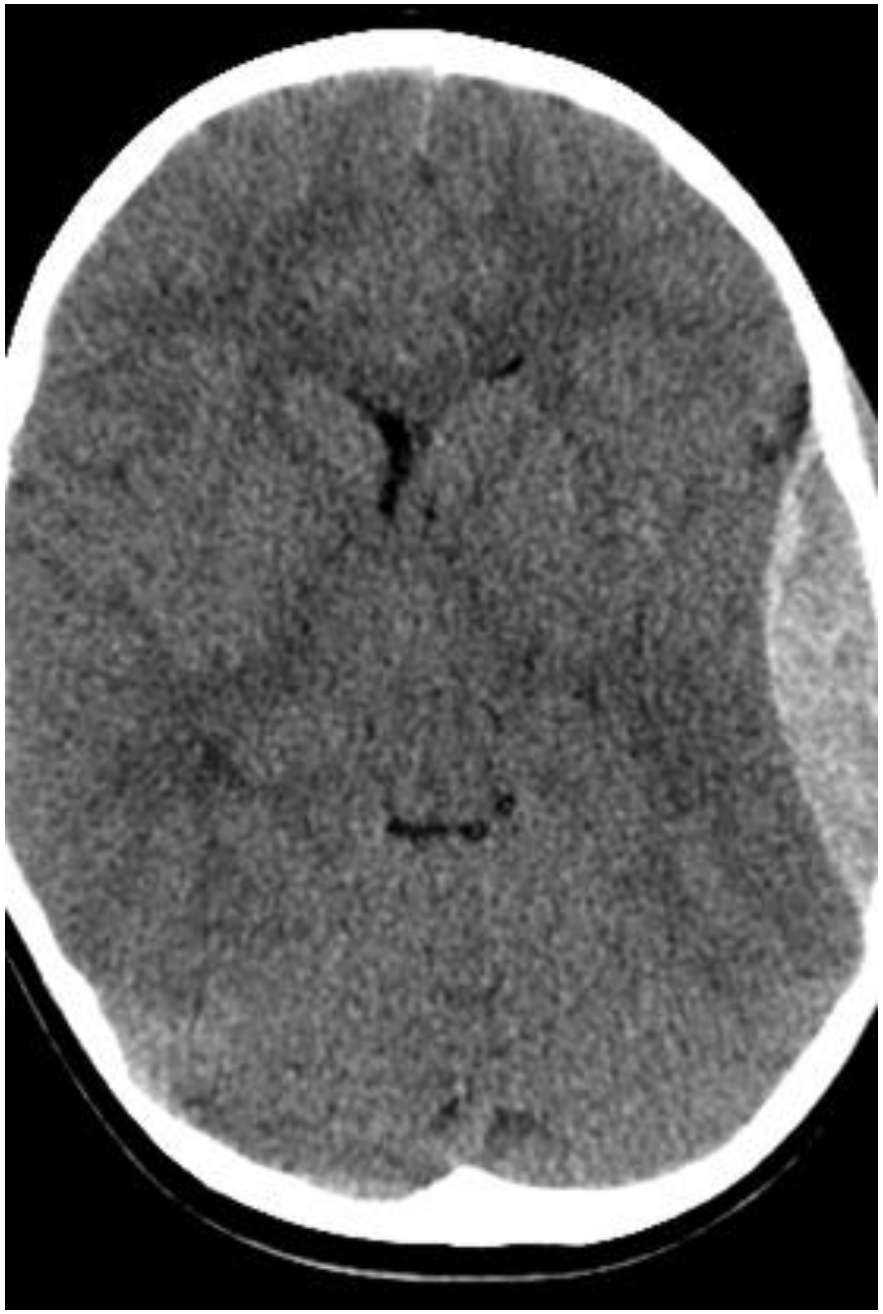


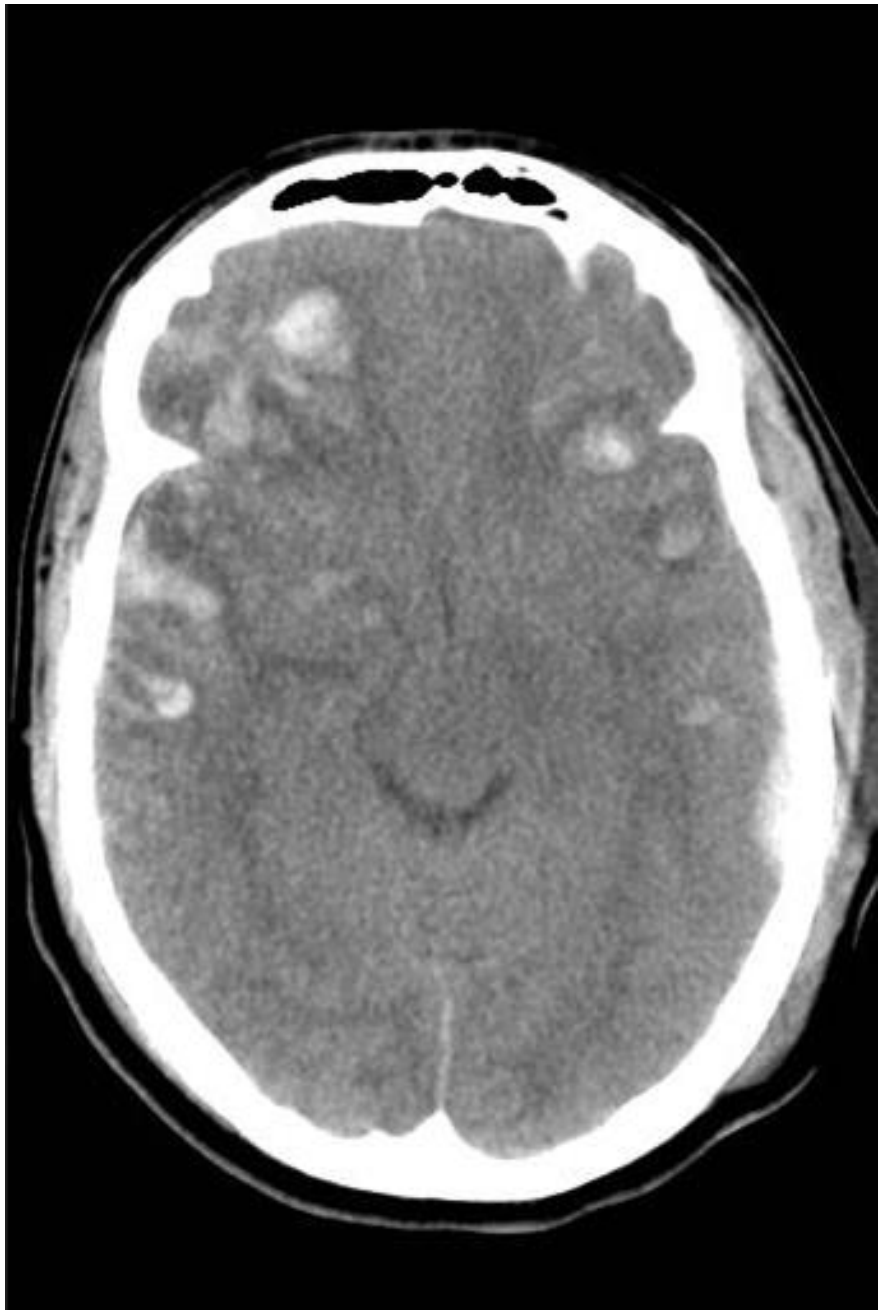
Increased production



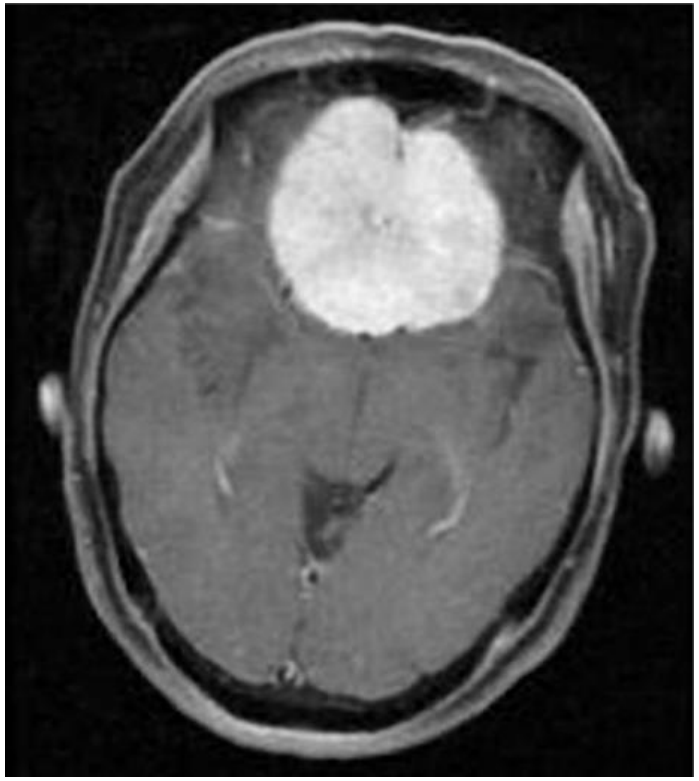
Blood

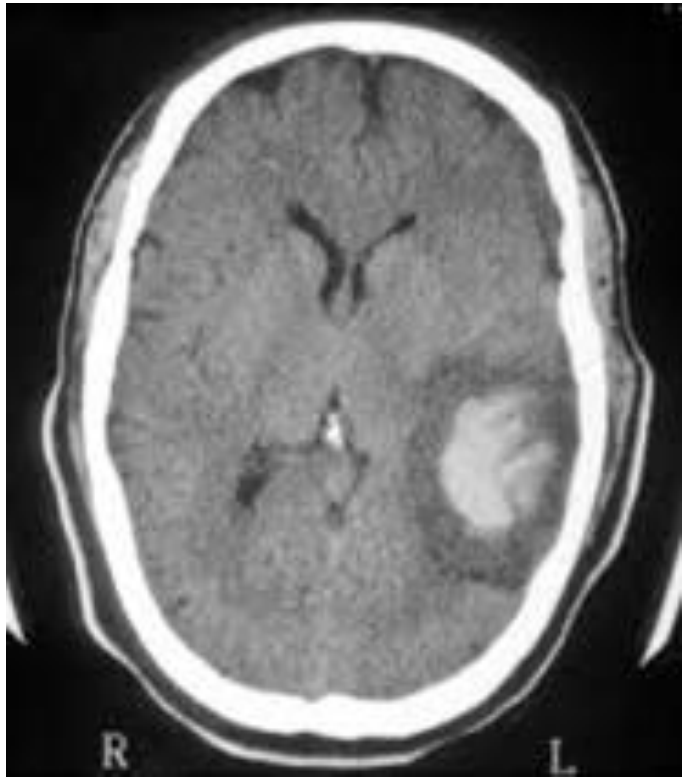






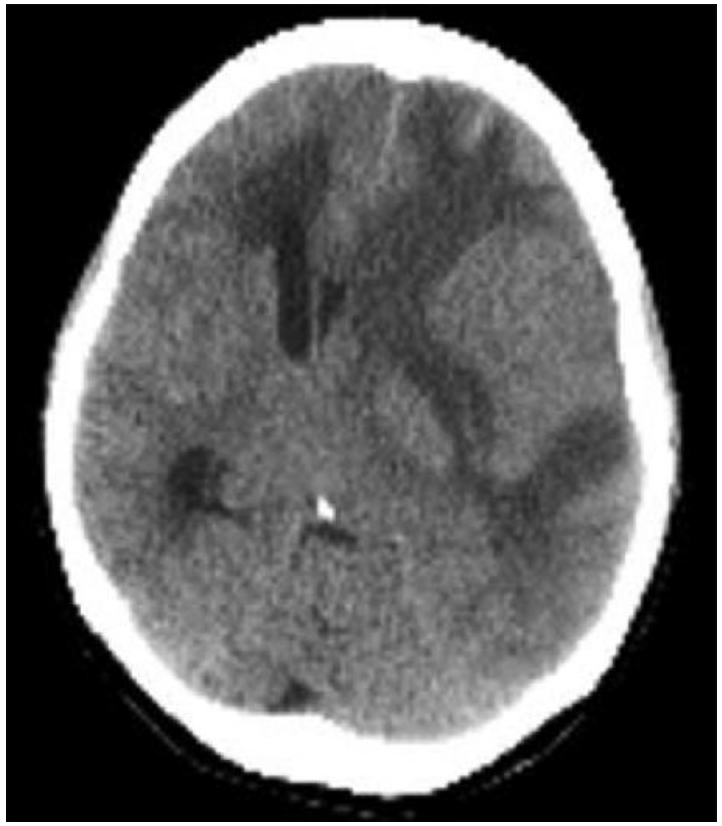
Brain Tumours





Brain oedema

Vasogenic oedema



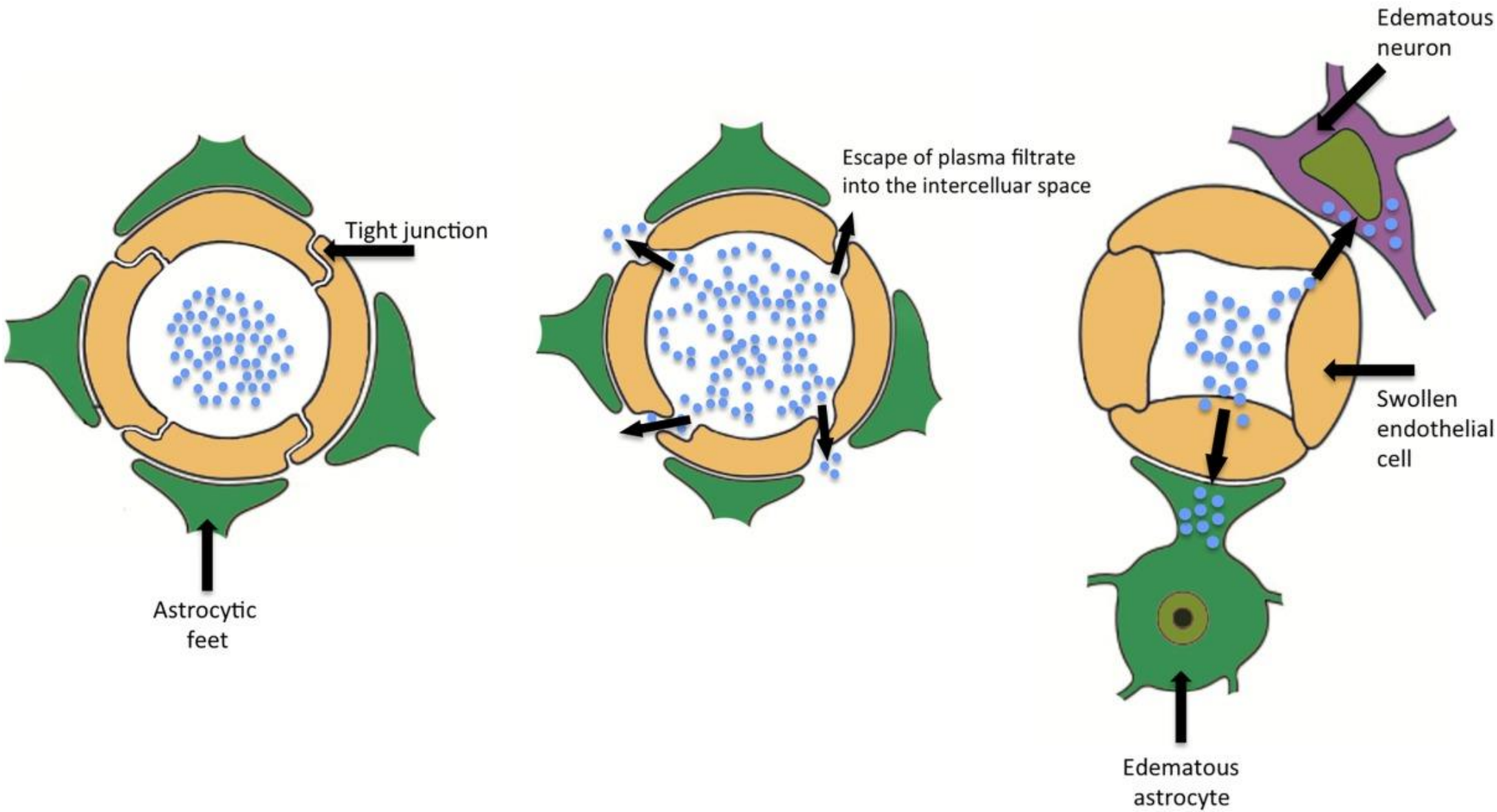
Interstitial oedema

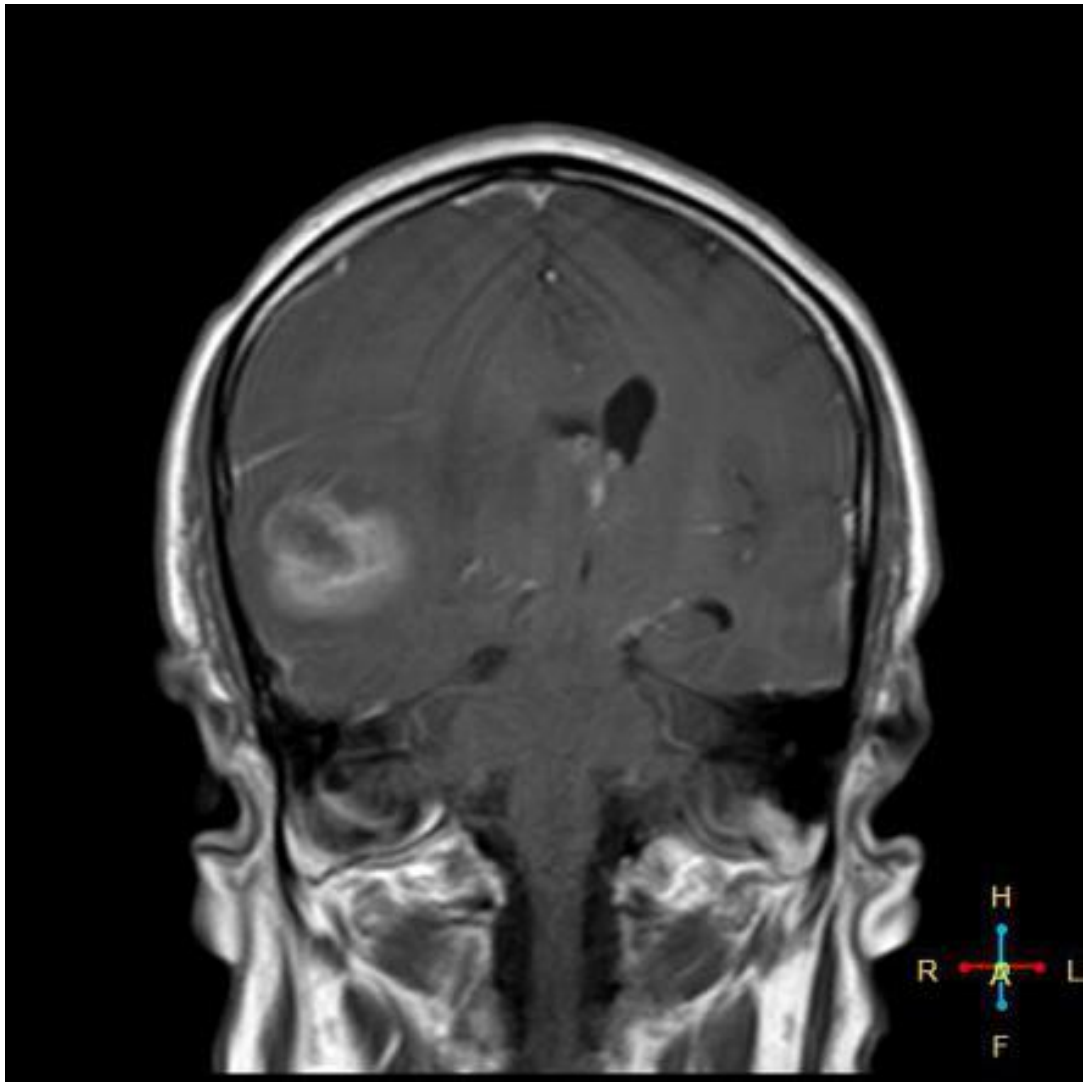
Cytotoxic oedema

Normal capillary

Vasogenic edema

Cytotoxic edema

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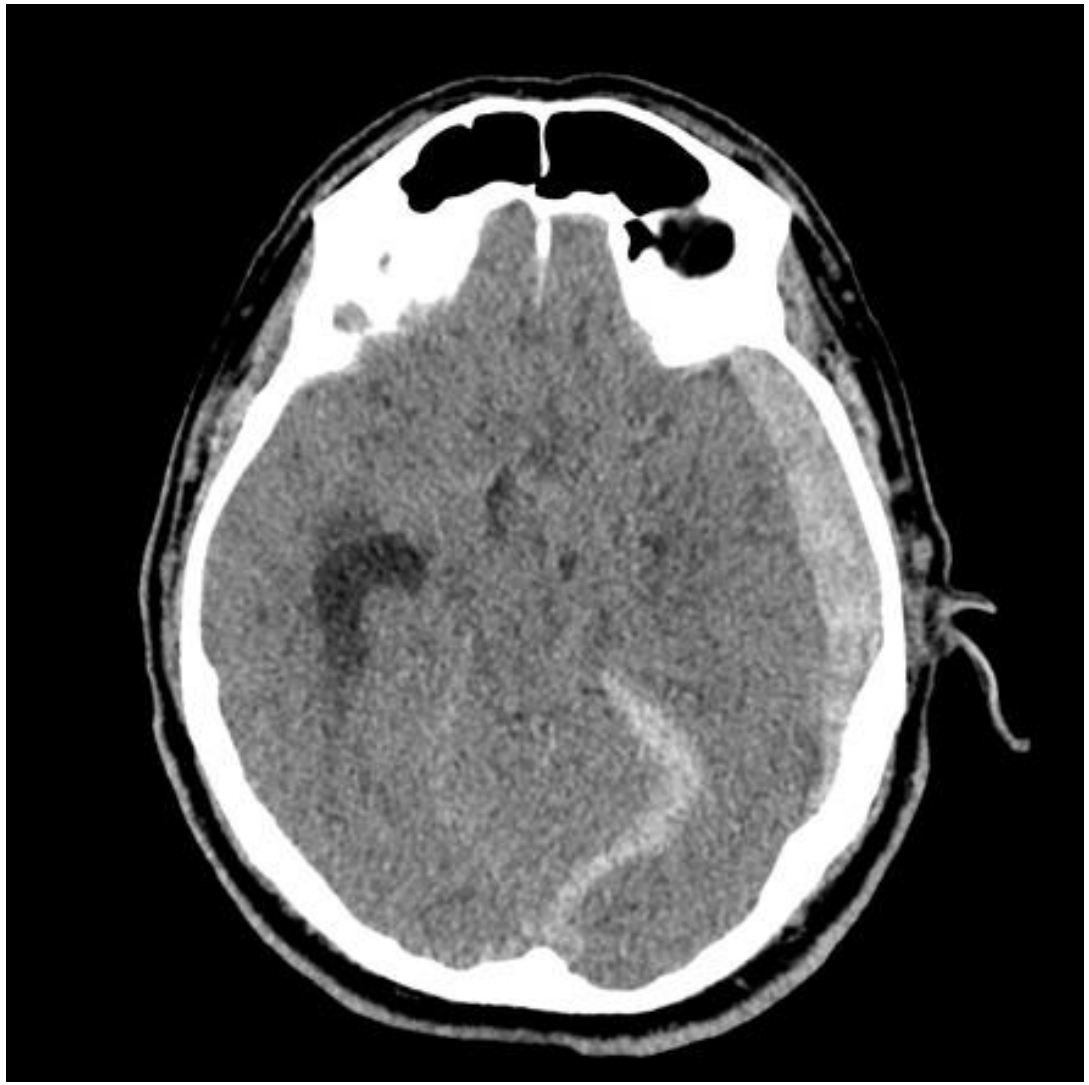
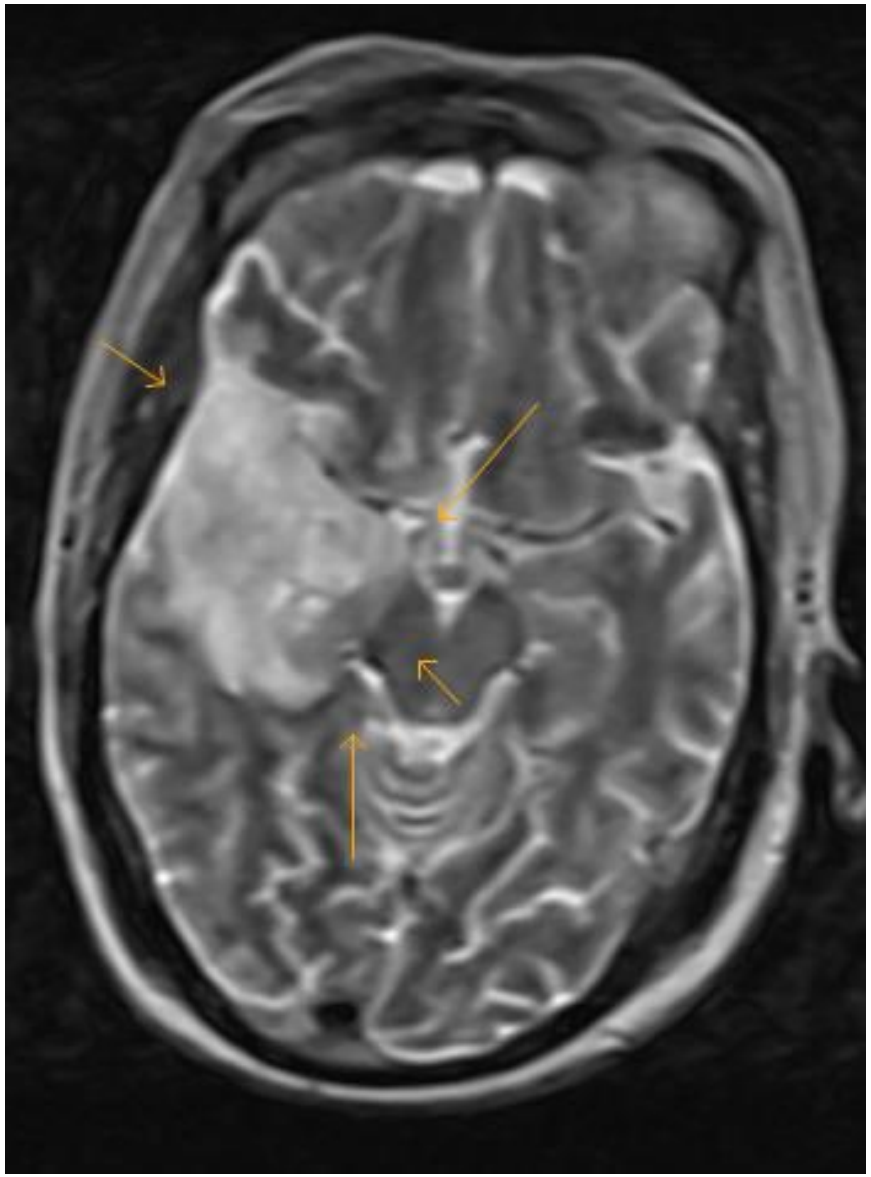
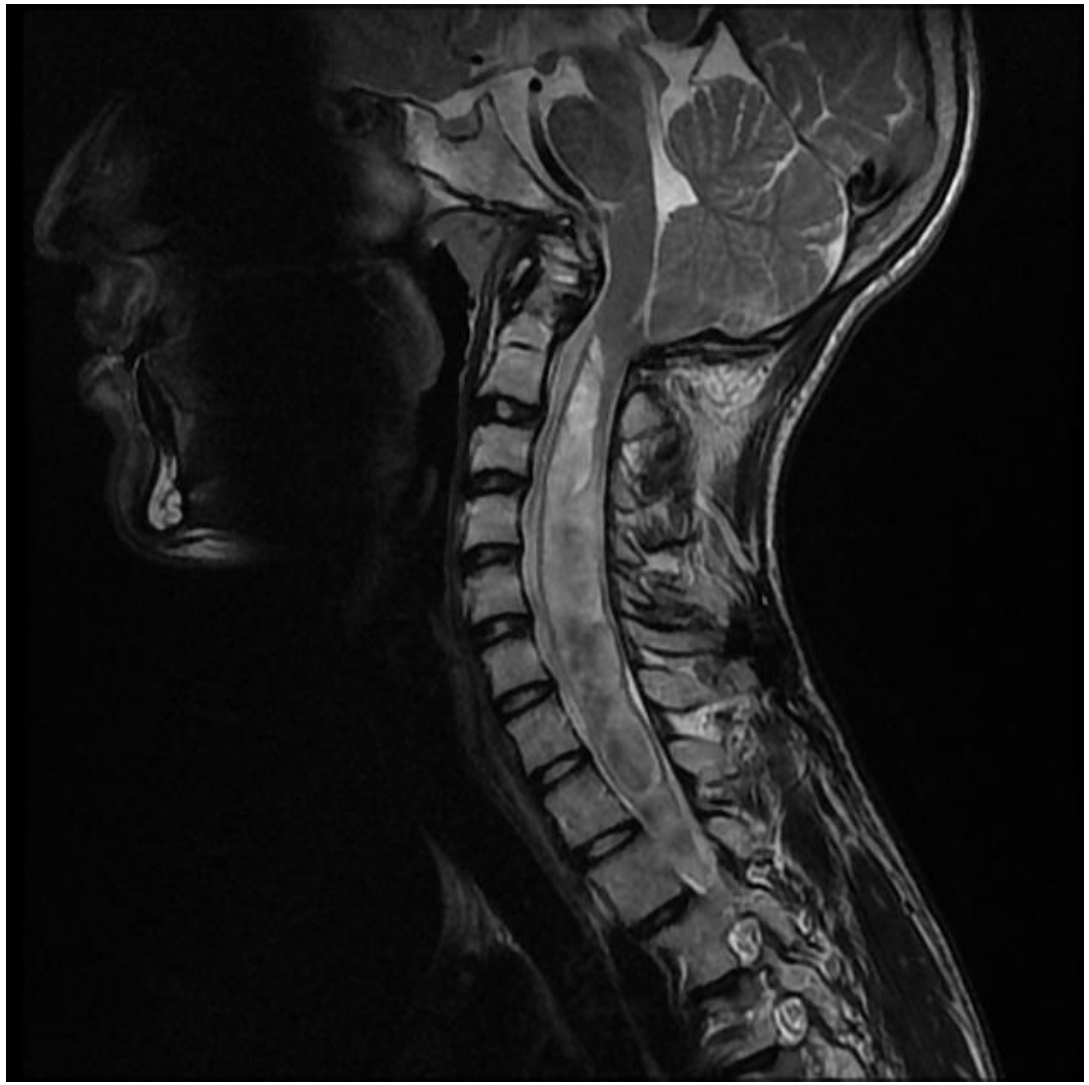
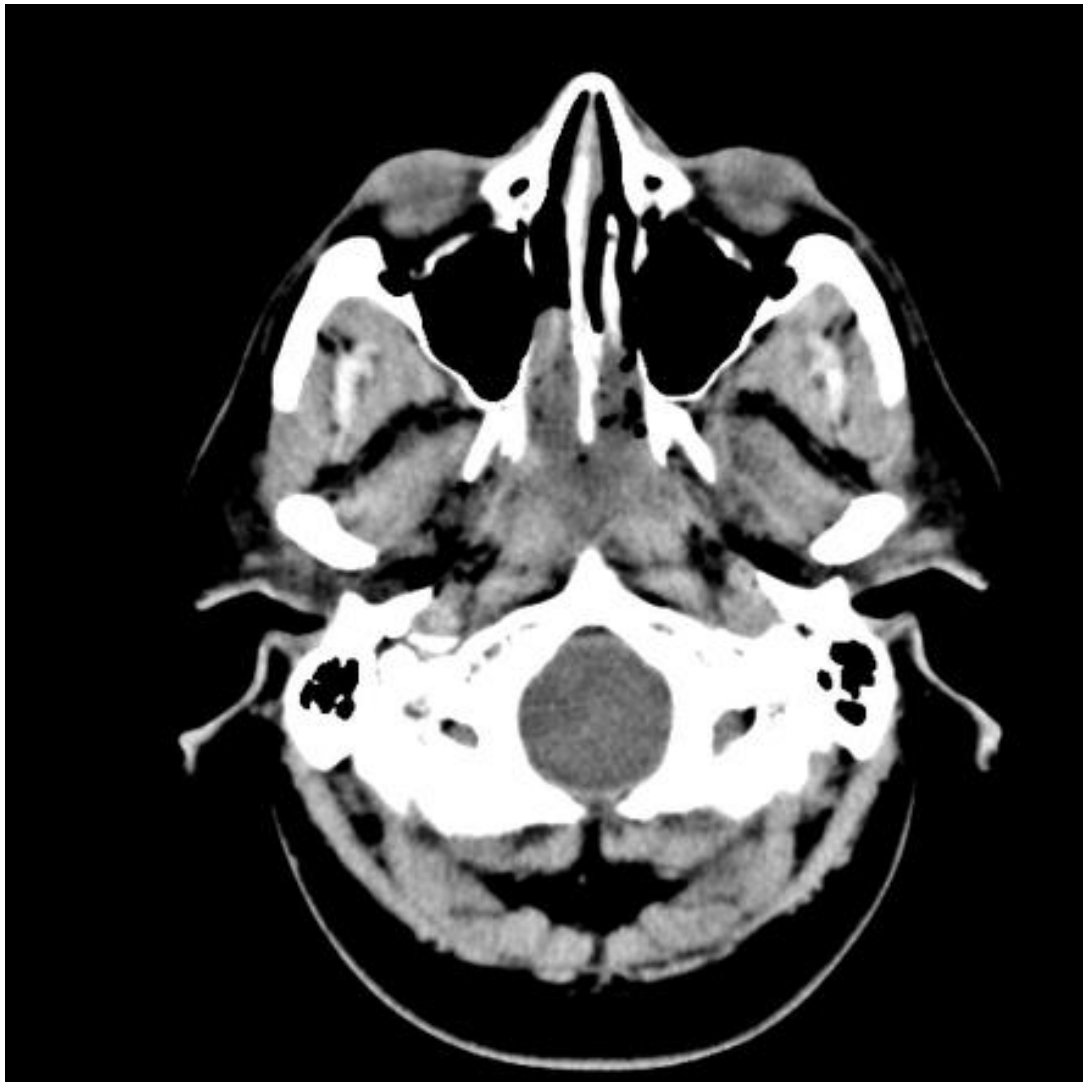








Indications for intracranial pressure monitoring

- **RECOMMENDATIONS**
- Level I - none
- Level II - all salvageable pts with GCS of 3–8 and abnormal CT
- Level III - in pts with severe TBI with normal CT if 2 or more of:
    i. age > 40 years
    ii. motor posturing
    iii. SBP < 90 mm Hg

J Neurotrauma. 2007;24 Suppl 1:S37-44. Guidelines for the management of severe traumatic brain injury. VI. Indications for intracranial pressure monitoring. Brain Trauma Foundation; American Association of Neurological Surgeons; Congress of Neurological Surgeons; Joint Section on Neurotrauma and Critical Care, AANS/CNS,

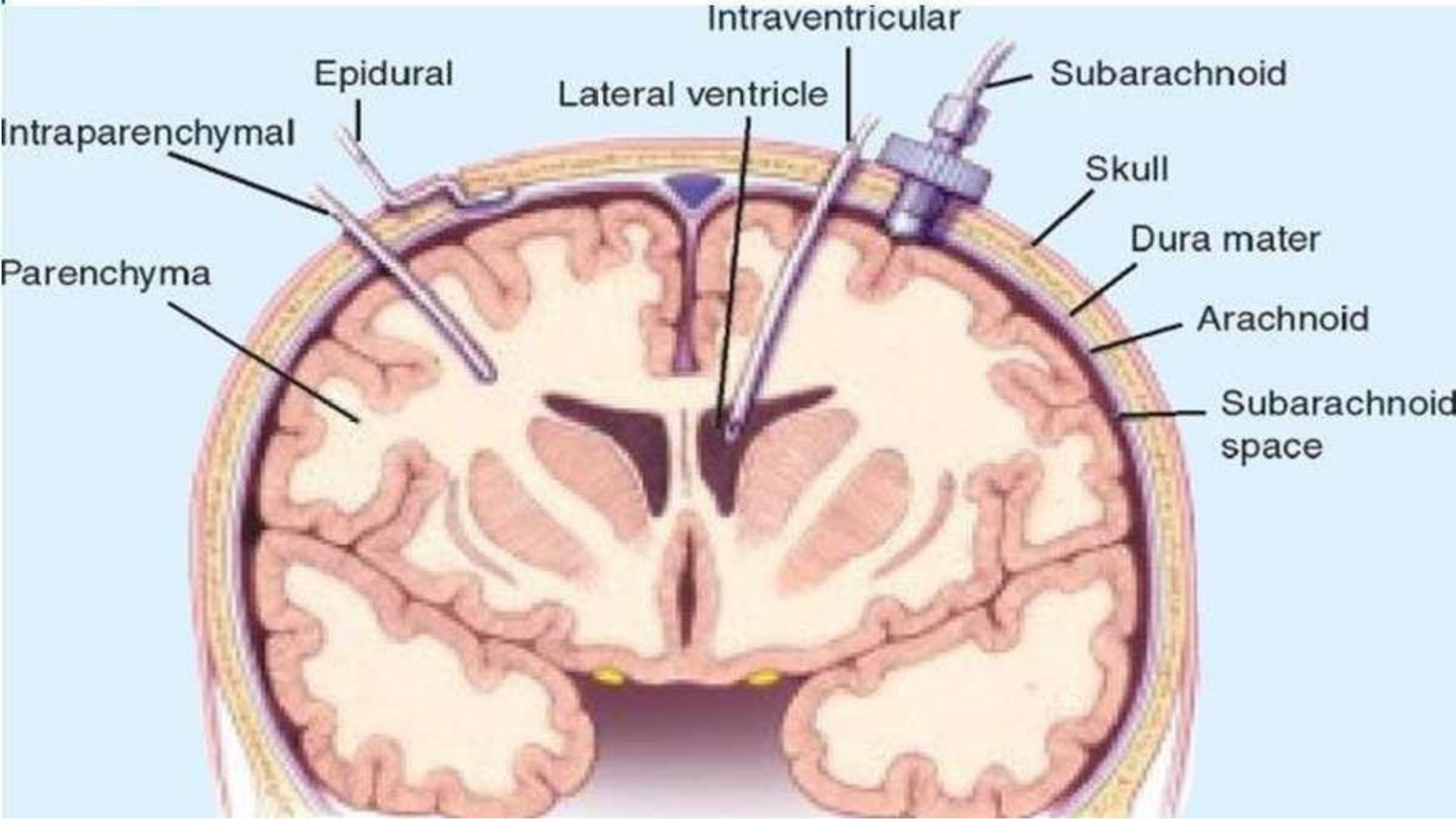
ICP Monitoring Techniques

Invasive:

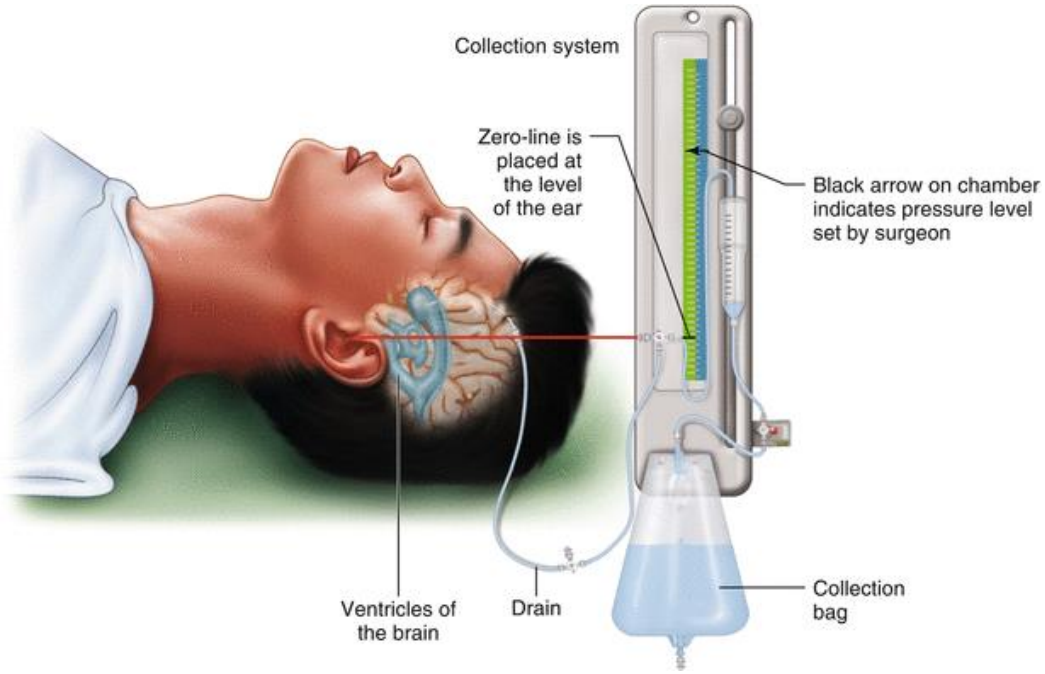
- External Ventricular Drainage (EVD) – Gold Standard
- Micro-transducer ICP Monitoring Devices
- Fiber-optic
- Strain Gauge
- Pneumatic

Non-invasive:

- Transcranial Doppler Ultrasonography (TCD) – Based on PI
- Tympanic Membrane Displacement (TMD)
- OpticNerve Sheath Diameter (ONSD) – via Transocular USG
- Magnetic Resonance Imaging (MRI) & Computer Tomography (CT)
- Pupillometry


Intraventricular
Epidural
Subarachnoid
Lateral ventricle
Intraparenchymal
Skull
Dura mater
Parenchyma
Arachnoid
Subarachnoid space

External Ventricular Drain



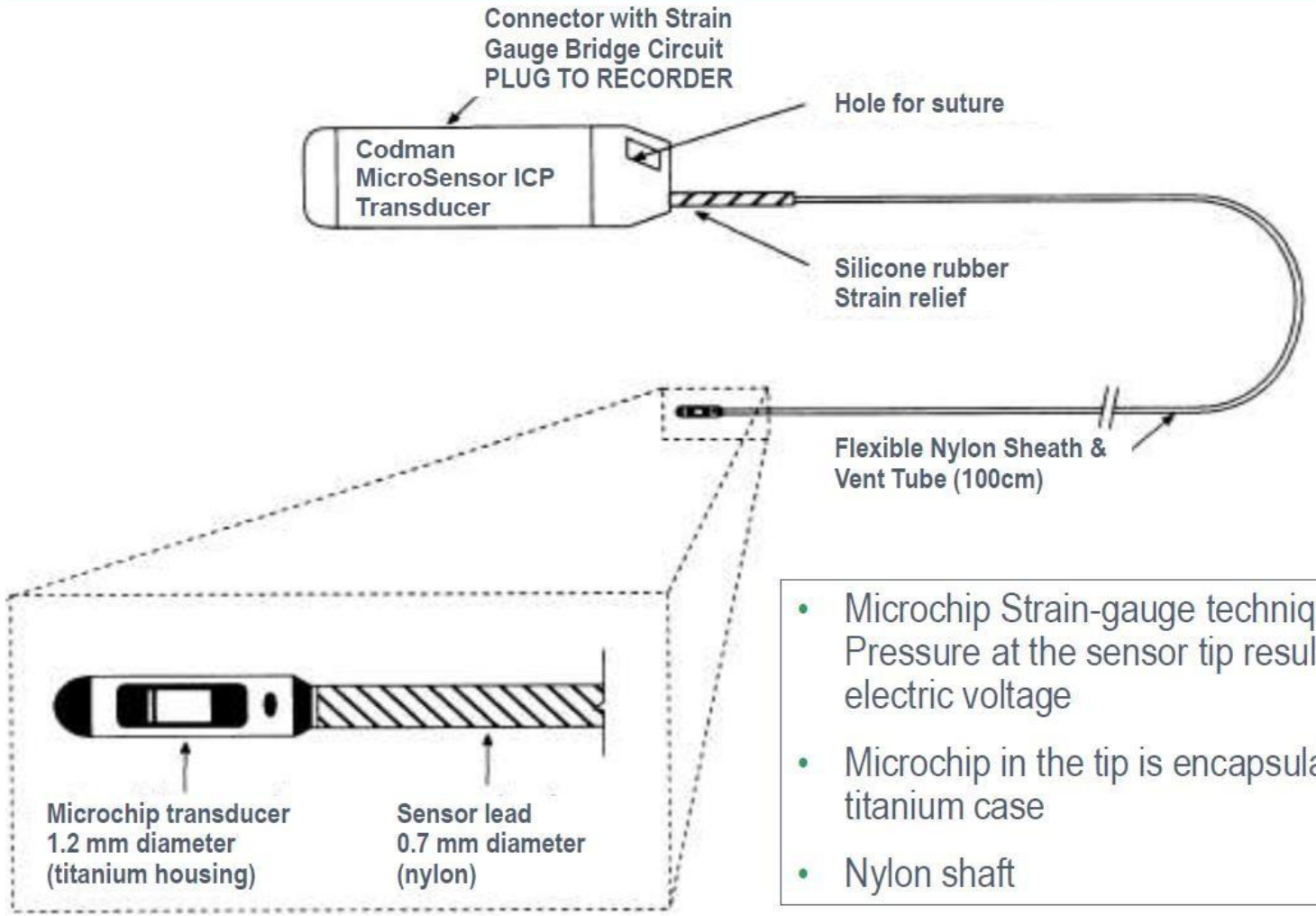
ADVANTAGES

- Serves also as a therapeutic device
- low cost
- most accurate

Disadvantages:

- malposition (4-20%)
- occlusion (8%)
- Hemorrhage (1.1%)
- And infection (8.8%)

ICP MICROSENSOR: Accuracy in its Design



- Microchip Strain-gauge technique: Pressure at the sensor tip results in an electric voltage
- Microchip in the tip is encapsulated in titanium case
- Nylon shaft

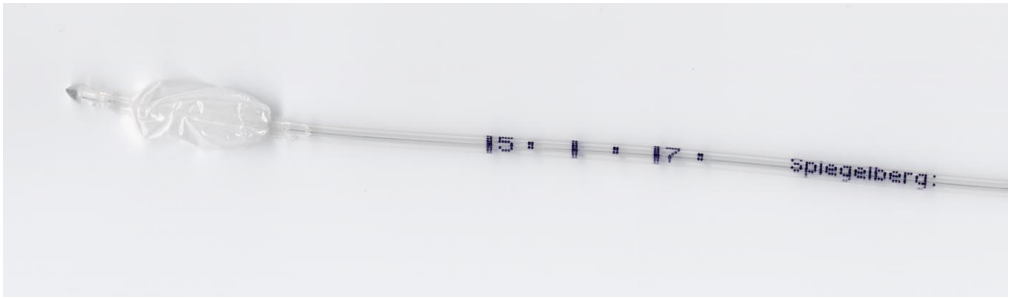
Spiegelberg ICP transducer

Principle:

Transmission of pressure from inside the body to the outside through air.

Partially filled air-pouch transmits pressure to the air-column.

First published by EJ Marey, 1881.



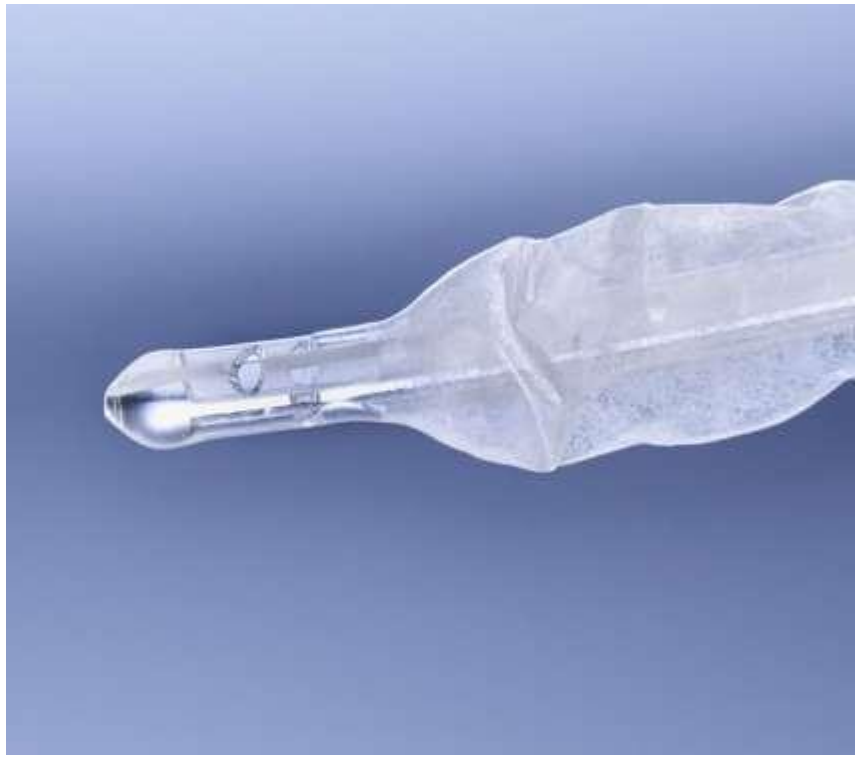


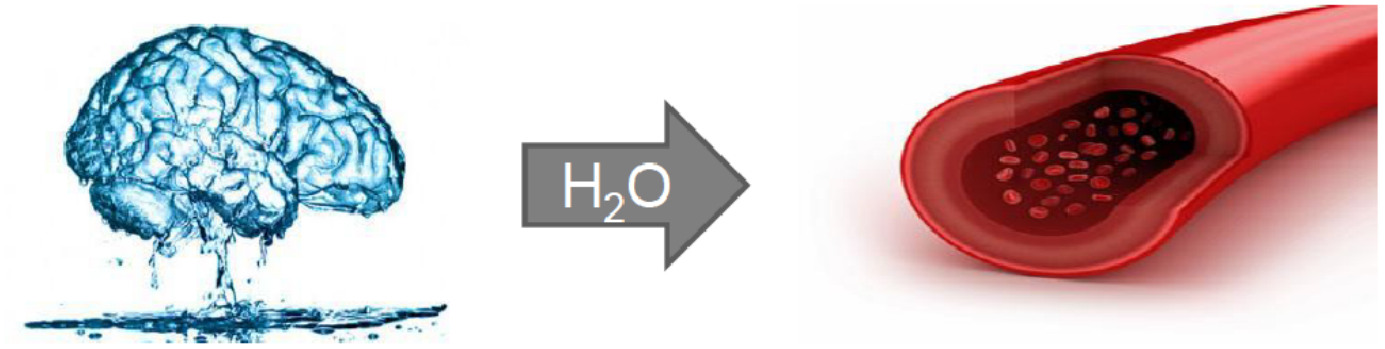
FIGURE 17. Approaches to IPC management.

WHAT TO DO	WHY
Elevate head of bed to 30 degrees	Facilitates venous drainage and lowers ICP
Discontinue cervical collar if able; if not able to remove, ensure that it is not so tight as to be causing impairment of venous return.	Facilitates venous drainage and lowers ICP
Straighten head/neck to neutral position	Facilitates venous drainage and lowers ICP
Remove ties circumferentially around neck (e.g., ET tube)	Facilitates venous drainage and lowers ICP
Treat fever	Decreases $CMRO_2$ and lowers ICP
USE THIS TIME TO TROUBLESHOOT EVD*	
Maintain normocapnia (check ABG)	Avoids vessel dilation with hypercapnia and avoids ischemic insult with hypocapnia (although mild hypocapnia is sometimes employed emergently to lower ICP it is not usually used prophylactically)
Initiate sedative and analgesic medications if not already in use (propofol/fentanyl/midazolam)	Decreases $CMRO_2$ and lowers ICP
Review CTH to look for potential new issues	Other potential issues/unnoticed structural abnormality
Increase sedative and analgesic medications as needed	Decreases $CMRO_2$ and lowers ICP
Drain CSF	Decreases intracranial CSF and lowers ICP
Check Na; consider hypertonic saline and/or mannitol	Decreases cerebral edema and lowers ICP
Repeat CTH	Investigates new/worsening structural abnormalities

Operative Neurosurgery, Volume 17, Issue Supplement_1, August 2019, Pages S17–S44, https://doi.org/10.1093/ons/opz090

20% Mannitol

- Mannitol decreases cerebral edema by removing water rapidly though diuresis

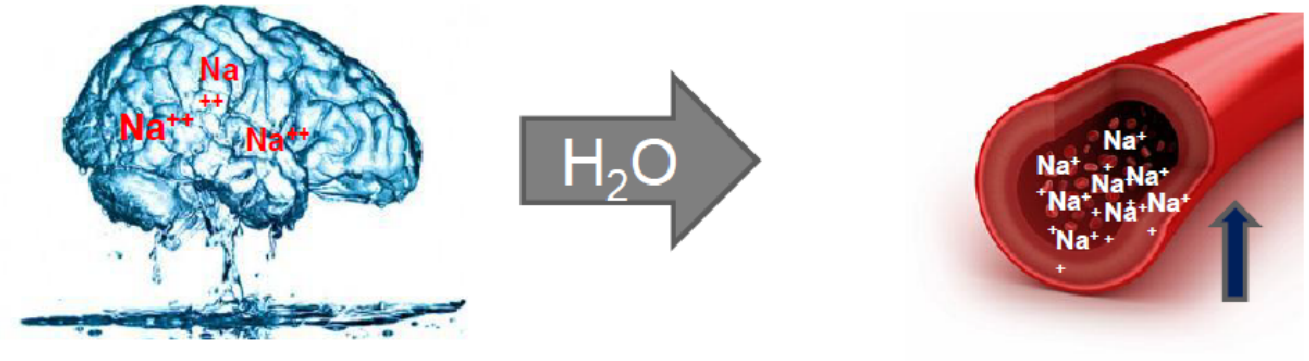


- The hypertonic concentration draws water from the brain and opens the kidneys. This draws water out of the brain, decreasing brain edema and lowering ICP

- Causes rapid fluctuations in serum electrolytes and hydration with large amounts of urine output

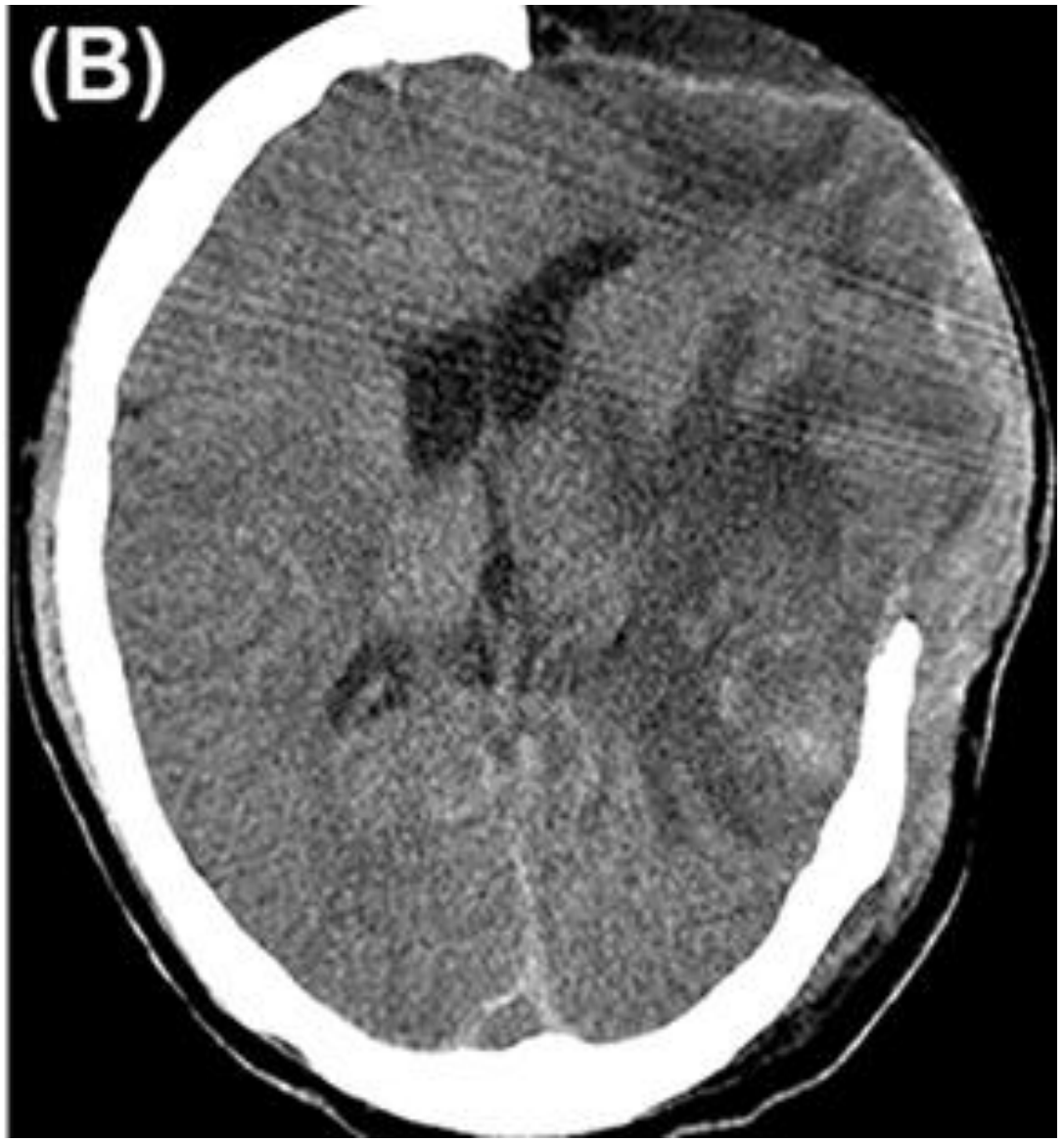
Hypertonic 3% NaCl

- Water moves by osmosis to the area of greatest Na concentration



- Hypertonic 3% NaCl administration increases sodium in the blood. This draws water out of the brain, decreasing brain edema and lowering ICP

- Slower process with > consistent decrease in brain edema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