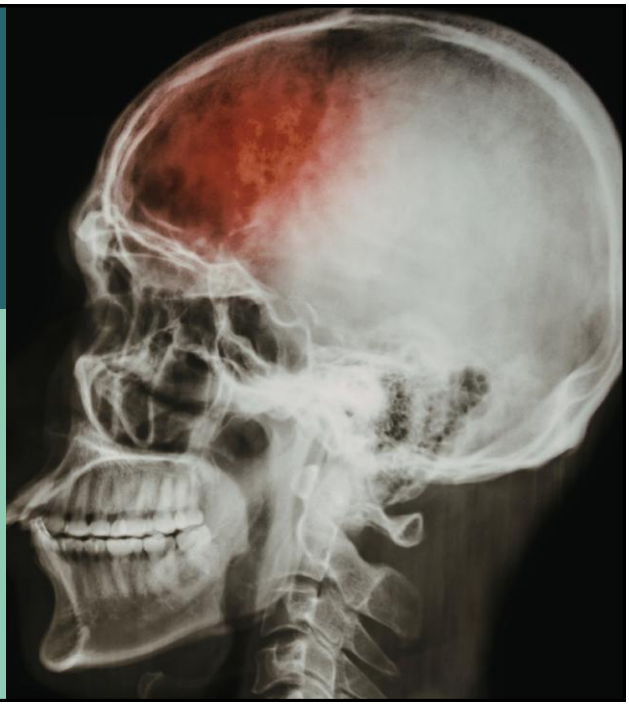
WHEN THE PRESSURE IS ON:

TRAUMATIC BRAIN INJURIES AND INTRACRANIAL PRESSURE MANAGEMENT



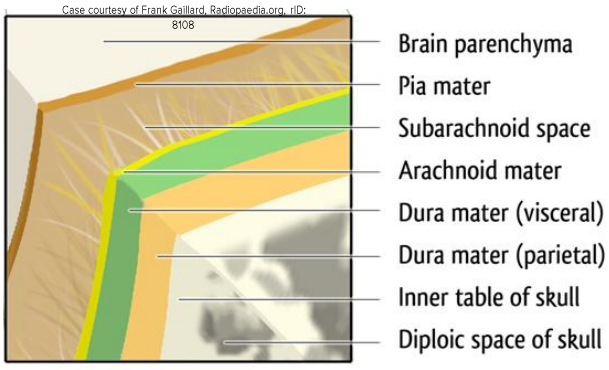
1

Objectives

- Explain how traumatic brain injuries alter cerebral perfusion and pressure regulation.
- Recognize early warning signs and monitoring data that indicate rising ICP.
- Discuss appropriate nursing interventions and critical care protocols for managing elevated ICP and improving patient outcomes.

2

Traumatic Brain Injuries



- **Pia mater** → innermost layer. Thin and fits like shrink wrap around the brain
- **Arachnoid mater** → CSF circulation
- **Dura mater** → outermost layer. Non-elastic tissue

Where is the epidural space?

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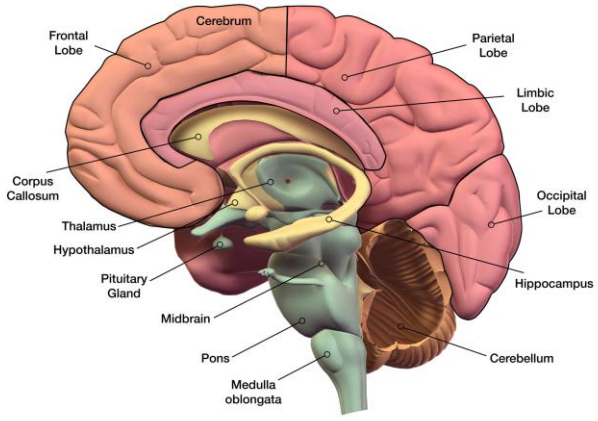
Anatomy & Physiology Review



- Consumes **15 - 20%** of total cardiac output
- The weight of the brain is **~ 2%** of the human body

Siwicka-Gieroba D, Robba C, Gołacki J, Badenes R, Dabrowski W. Cerebral Oxygen Delivery and Consumption in Brain-Injured Patients. J Pers Med. 2022 Oct 25;12(11):1763. doi: 10.3390/jpm12111763. PMID: 36573716; PMCID: PMC9698645

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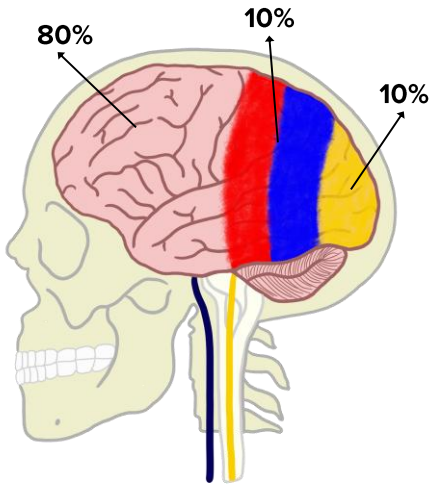
Anatomy & Physiology Review

- **Rigid cranial vault →**
  - Brain tissue (80%)
  - Venous & arterial blood (10%)
  - CSF (10%)
- Intracranial **pressure** is influenced by ***volume and compliance***
- **Normal ICP ≤ 15 mmHg**



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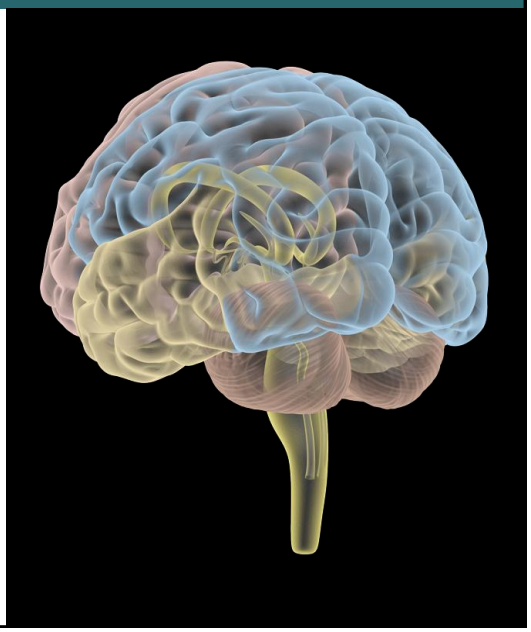
Anatomy & Physiology Review

- **Ventricles produce CSF**
  - 20mL/hour (500 mL/day) of CSF
  - ~ 150 ml in the brain
- CSF reabsorbed in the venous sinuses within the brain



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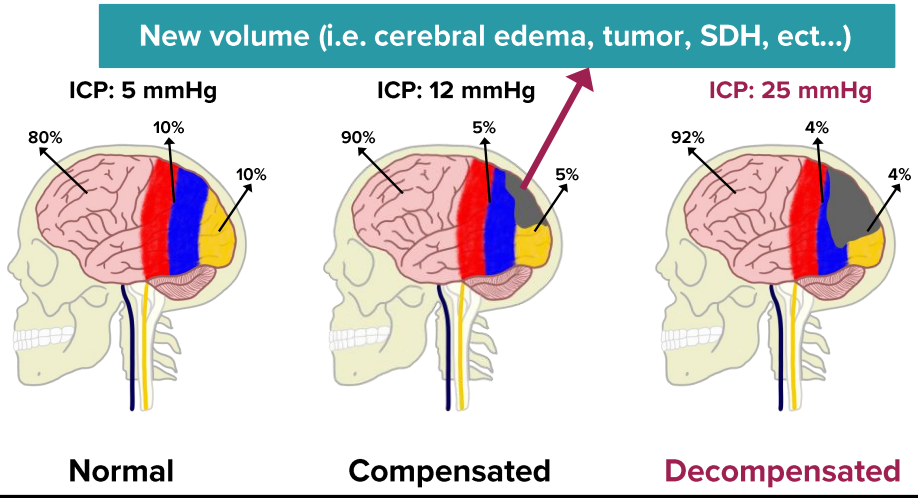
Monroe-Kellie Doctrine

- An ↑ in one will cause ↓ in the others…when ***compensation*** is intact
- An ↑ in **<u>total volume</u>** leads to ↑ ICP



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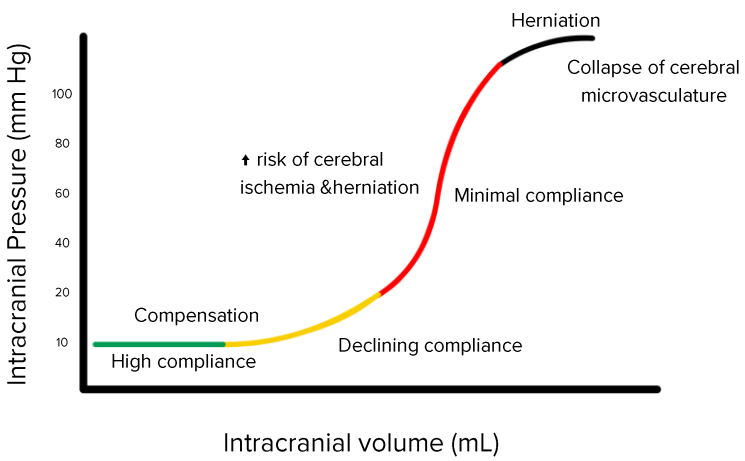
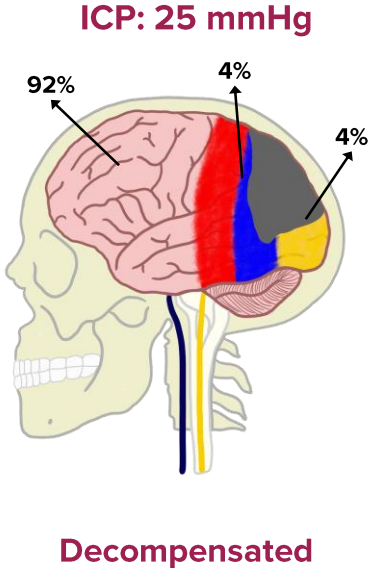
Monroe-Kellie Doctrine & CBF

- **↑ ICP = loss of compliance**
  - **Risk of herniation!**





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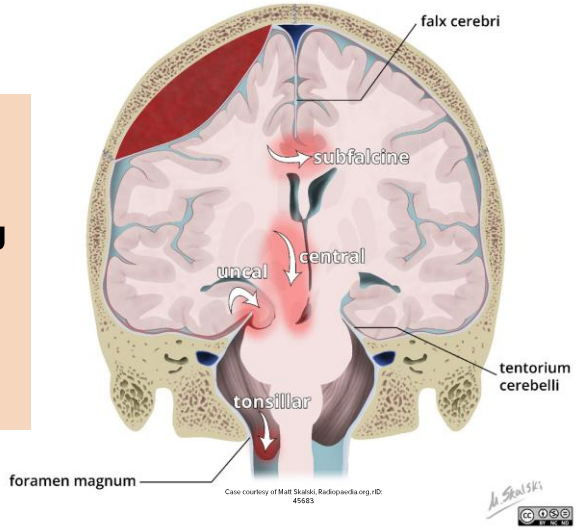
Intracranial Hypertension

- **Intracranial hypertension:**
  - **Sustained (> 5 min) elevation of ICP to > 22 mmHg or > 20 mmHg in pediatrics[1]**
  - Displaced brain parenchyma
  - Vascular compression



1. Bhalla, P., Dingman, J. S., & Aysenne, A. (2024). Intracranial hypertension and herniation protocol (Version 6.0). Emergency Neurological Life Support®. Neurocritical Care Society. https://www.neurocriticalcare.org/Portals/0/ENLS%205.0/ENLS%206.0/Protocol%20V6_0_Intracranial%20Hypertension%20and%20Herniation.pdf?ver=oGpOZQ2sI6-mgzJhEgTtTg%3d%3d

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Cerebral Blood Flow

CBF = Cerebral Perfusion Pressure (CPP) / Cerebral Vascular Resistance (CVR)
Normal ~ 55 mL/100g/min

Myogenic regulation

Chemical regulation

Metabolic regulation

Neurogenic & endothelial control

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Metabolic, Neurogenic, Endothelial Regulation

Metabolic Regulation → **Local brain activity**

Neurogenic & endothelial control → **Nervous System**; **NO & endothelin**

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Chemical Regulation

CO_2, pH, O_2 content

Carbon Dioxide	
↑	Vasodilation
↓	Vasoconstriction

Oxygen	
=	Minimal effects
↓	Vasodilation

pH	
↓	Vasodilation
↑	Vasoconstriction

Hypercapnia, PaO_2 < 50 mmHg, & acidosis increase CBF

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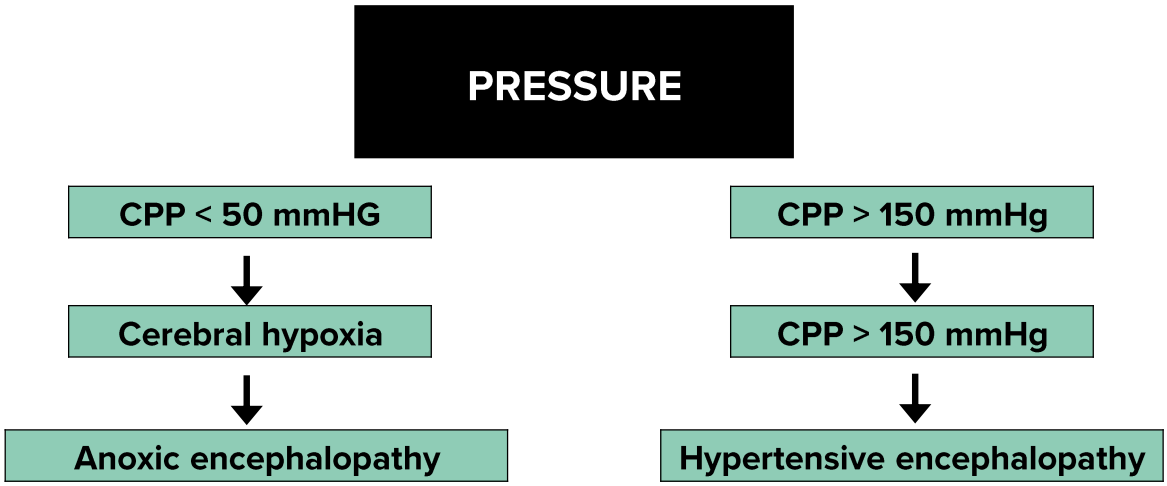
Myogenic Autoregulation



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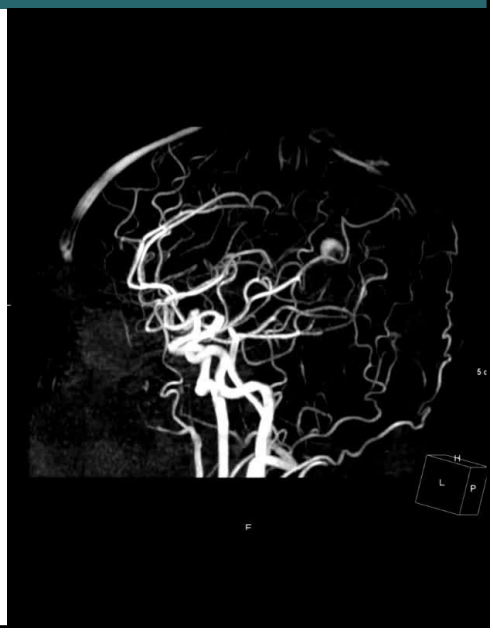
Cerebral Prefusion Pressure (CPP)

- **The MAP of the brain**
- **Pressure required to perfuse the brain**
- **CPP = MAP - ICP**
  - **Goal CPP: 60 - 70 mmHg**
  - < 30 mmHg irreversible ischemia



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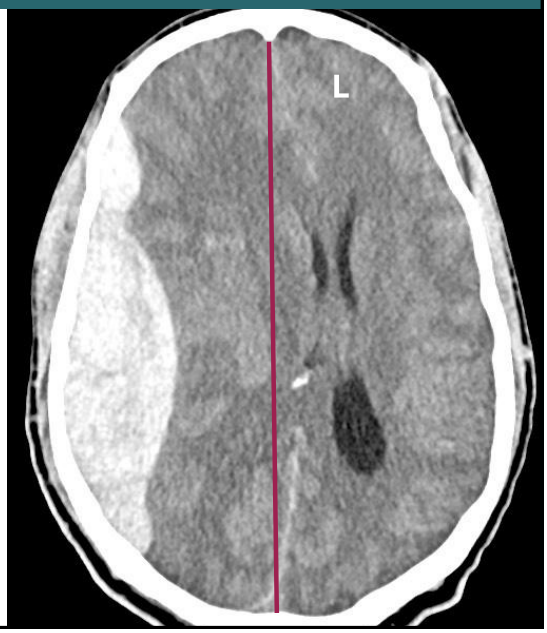
Cerebral Prefusion Pressure (CPP)

- MAP 65 - ICP 15 = CPP 50!
- MAP 90 - ICP 25 = CPP 65
- MAP 55 - ICP 25 = **CPP 30!!!!**
- MAP 60 - ICP 40 = **CPP 20!!!**

Hypotension + ↑ ICP =
LIFE THREATENING!



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What about this?

YIKES!

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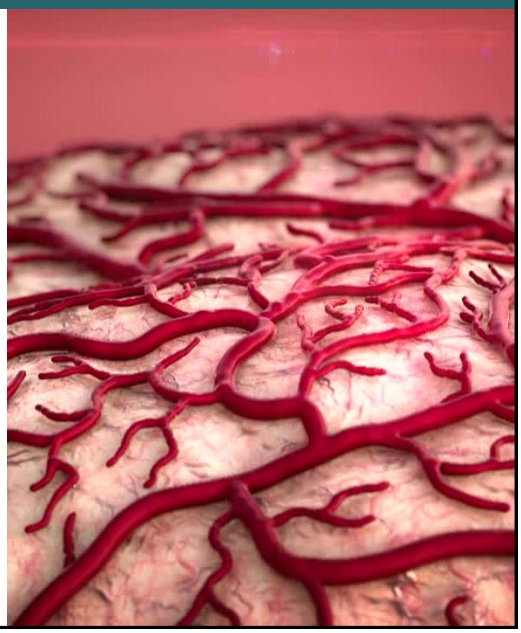
Cerebral Oxygen Metabolism

- Uses **25%** of glucose and **~ 20%** of available O_2 to function normally
- Functions almost entirely by ***aerobic metabolism;*** ineffective anaerobic metabolism
- Cerebral Metabolic Rate of Oxygen ($CMRO_2$) → ~3–3.5 mL O_2/100 g/min



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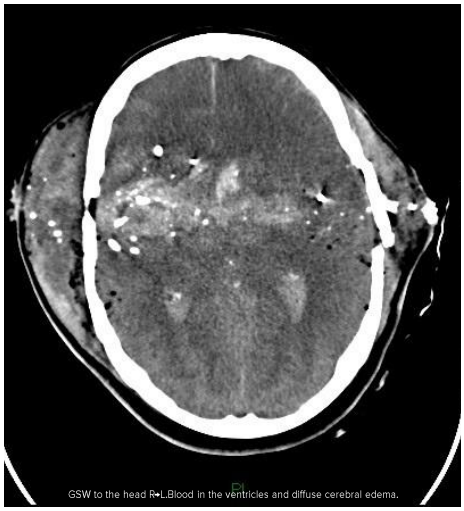
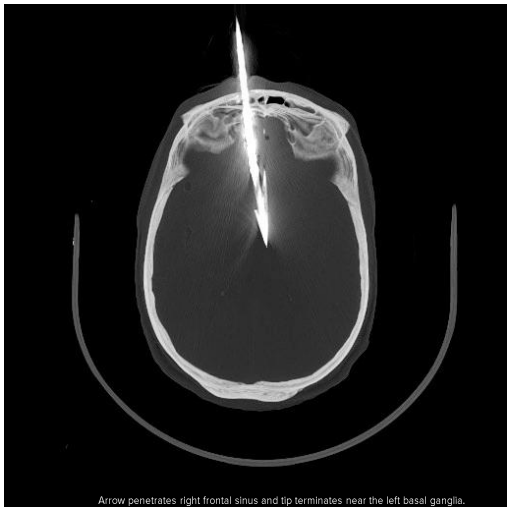
Penetrating Head Trauma



Case courtesy of Sajoscha A. Sorrentino, Radiopaedia.org, rID: 14487



Case courtesy of Michael P. Hartung, Radiopaedia.org, rID: 72101

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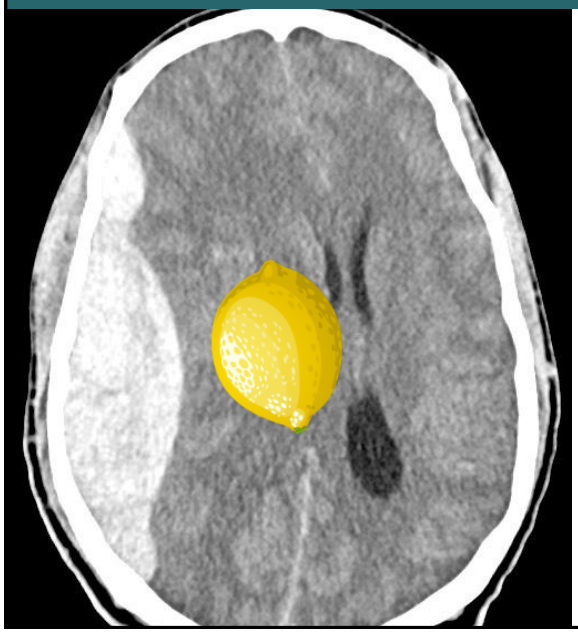
Epidural Hematoma



- No natural epidural space
- Can occur anywhere in the skull
- Usually in the temporal or parietal region
  - Location of meningeal artery/vein
- High association with skull fx
- **Arterial bleed!!**

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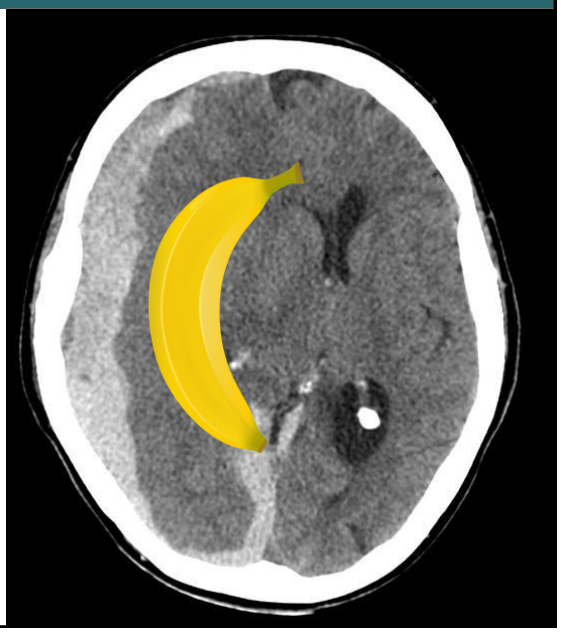
Subdural Hematoma

- Between dura and arachnoid membrane
- Tearing of the bridging **veins**
- Elderly
  - Anticoagulants!
  - Cerebral atrophy
- Acute, subacute, chronic



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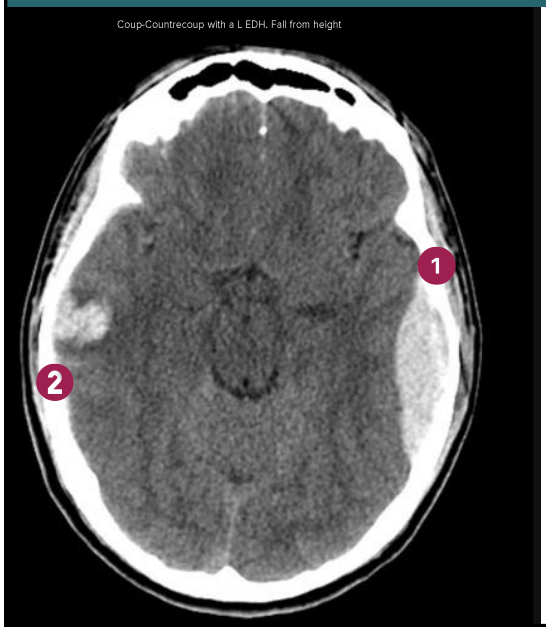
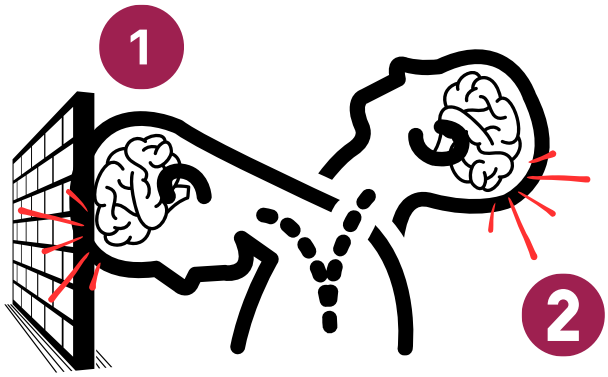
Coup-Countrecoup



1. **Coup -** direct impact
2. **Countrecoup -** injury distal to the impact site



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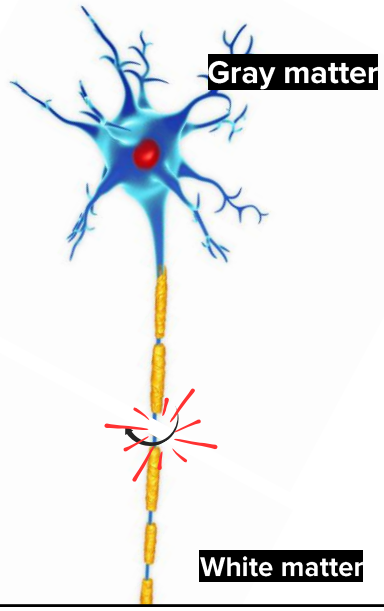
Traumatic axonal and/or microvascular injury (TAMVI)

- **Acceleration/deceleration injury**
- **Widespread microscopic damage to axons and/or microvasculature**
- **GCS ≤ 8 for > 6 hours without:**
  - lucid interval
  - mass lesion
  - midline shift on imaging
- **May not show up on CT scan!**
  - **Grade I - III**



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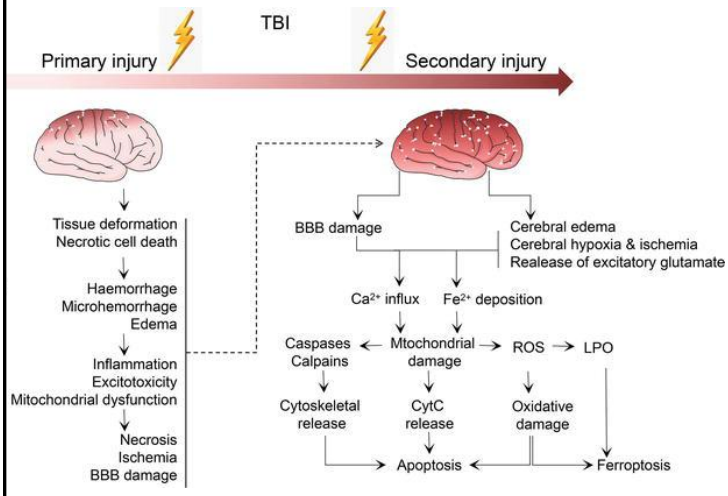
Primary vs Secondary Injury



- **Edema**
- **Inflammation**
- **Hypoxia**
- **Ischemia**
- **Oxidative damage**
- **Neurotransmitters release**
- **Neuronal damage/death**

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Cerebral Edema

Vasogenic Edema

Mechanism:
- Blood-brain barrier (BBB) disruption → plasma proteins & fluid leak into extracellular space

Location:
- White matter predominant — spreads along fiber tracts

Clinical Impact:
- Increased ICP, midline shift, herniation risk

Cytotoxic Edema

Mechanism:
- Cell energy failure → Na^+/K^+ pump dysfunction → intracellular water accumulation (cell swelling)

Location:
- Gray & white matter — affects neurons & glia

Clinical Impact:
- More refractory to treatment; BBB remains intact

Both types ↑ intracranial volume → ↑ ICP → ↓ CPP, TBI often involves BOTH

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Impaired Autoregulation in TBI

Normal Autoregulation

Cerebral vessels constrict/dilate to maintain constant CBF across a wide CPP range (50–150 mmHg)

After TBI — Autoregulation Fails

CBF becomes **pressure-passive** — it rises and falls directly with MAP, losing protective buffering

BBB Disruption

Damage to the blood-brain barrier causes loss of endothelial signaling, impairing the myogenic & chemical regulatory responses that normally adjust vessel tone.

Neuronal & Vascular Injury

Direct trauma injures cerebral vasculature and perivascular neurons, disrupting the local neurovascular coupling that links CBF to metabolic demand.

Inflammatory Cascade

Cytokines, free radicals & excitotoxic neurotransmitters released after TBI alter vascular reactivity, blunting CO_2 and pressure responses.

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Tools are great, but...

DO YOUR NEURO ASSESSMENTS

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Clinical S/S of ↑ ICP

Symptoms of Increased Intracranial Pressure	
Early	**Late**

• Altered LOC
  ○ **Confusion, agitated, restless, irritable**
• ↓ GCS
• ↓ Motor function
• Weak reflexes
• Pupillary changes
  ○ Sluggish
• Nausea, vomiting
• Visual disturbances
• Seizure
• Headache
	• ↓ LOC
	  ○ Obtunded, comatose
	• ↓ GCS
	• ↓ Motor function
	  ○ Decordicate, decerebrate
	  ○ Hemiparesis (ipsilateral, contralateral)
	• Pupillary changes
	  ○ **Ipsilateral** dilation → bilateral
	• Respiratory pattern changes
	• Rising systolic blood pressure
	• Hypertension with no obvious cause
	• Temperature changes

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Vital Sign Changes

EMERGENCY

CUSHING'S TRIAD

- Irregular respiration
- Widening pulse pressure
- Bradycardia

LATE SIGN!

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ICP Monitoring Devices

Does not replace serial neurological and radiographic examinations

When to consider ICP monitoring:

- Comatose patients (GCS ≤ 8) + structural brain damage on CT
- GCS > 8 + structural brain damage + high risk for progression
- GCS > 8 when knowing the ICP might facilitate management of other issues
- Progression on CT imaging or clinical deterioration
- Neurosurgical procedure

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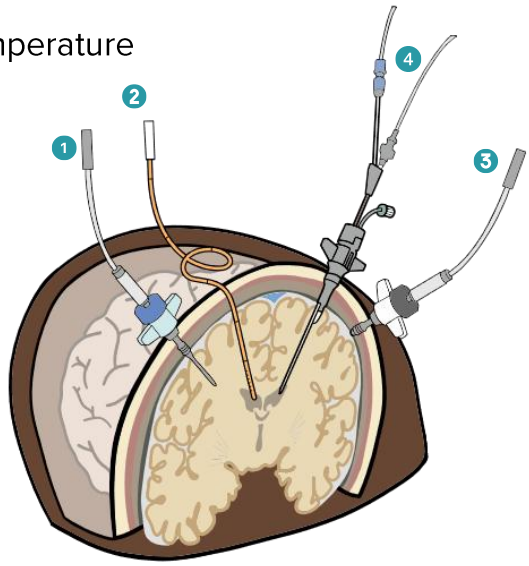
ICP Monitoring Devices

1. **Intraparenchymal/bolt:** ICP, $PbtO_2$, temperature
2. **Intraventricular/E.V.D:** Pressure, drain
3. **Combination / multimodality**
4. **Subarachnoid:** ICP, temperature
5. **Subdural, epidural**



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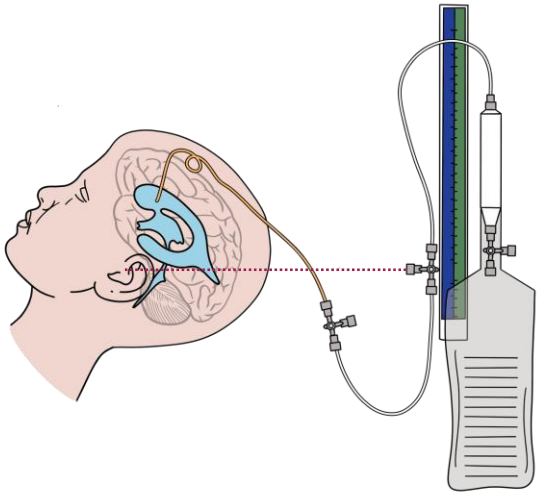
External Ventricular Device (EVD) / Ventriculostomy

- **Level to tragus**
  - EAM, foramen of Monro
- **Provider will set the height of burette**
- **Accurate leveling to avoid:**
  - Erroneous ICP readings
  - Under/over drainage of CSF
- **Understand stopcock positioning**
- **Know drainage orders!**
- **Dressing changes per unit policy**
- **Monitor/record output**



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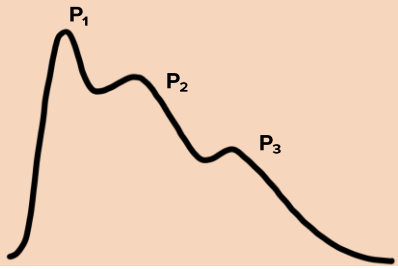
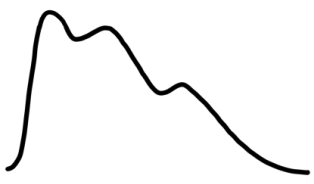
Intracranial Pressure Waveforms



Normal ICP waveform

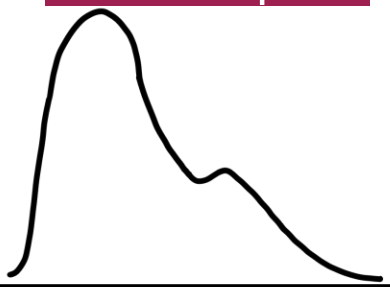
- **P_1 : Arterial pulsation**
- **P_2 : Intracranial compliance**
- **P_3 : Aortic valve closure**

Low SBP



↓compliance

Marked ↓compliance



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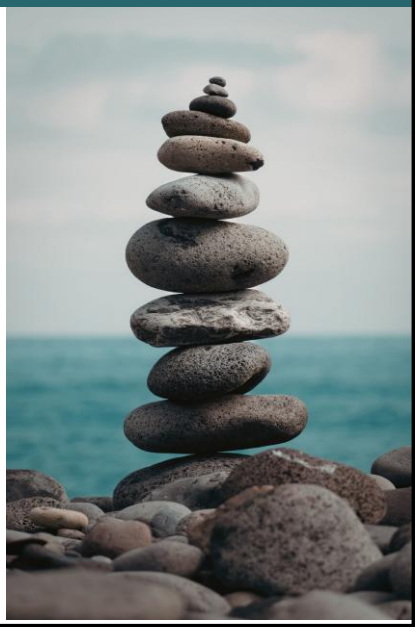
$PbtO_2$ monitoring

Is this a delivery issue?

Is this a consumption issue?

- **Balance between O_2 delivery & consumption**
- **Typical goal: > 20 mmHg**
- **Used with other multimodal monitoring devices**
- **Probe location can influence reading**

Lookout → BOOST-III Trial



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Standard TBI Care

Cluster, cool, calm, controlled, centered, CO_2

- **Positioning**
  - HOB > 30-45° → Improved venous outflow
  - Centered / straight
- **Pain & agitation management**
- **Normothermia**
  - Fever control 38.0°C (100.4°F)
- **Oxygenation & ventilation management**
  - $EtCO_2$ / $PaCO_2$ normal 35 - 45 mmHg
- **Decreased stimulation**
- **Avoid & correct hypothermia**
  - Serum Na 135–145 mEq/L

Parameter	Goal Range
Pulse oximetry	≥ 94%
PaO_2	80–100 mm Hg
$PaCO_2$	35–45 mm Hg
Systolic blood pressure	≥ 110 mm Hg
ICP	< 22 mm Hg
$PbtO_2$	≥ 15 mm Hg
CPP*	60–70 mm Hg
Serum sodium	135–145 mEq/L
Serum osmolality	≤ 320 mOsm
INR	≤ 1.4
Temperature	36.0–37.9°C
Platelets	≥ 75 × 10^3/mm^3
pH	7.35–7.45
Glucose	100–180 mg/dL
Hemoglobin	≥ 7 g/dL

*Depending on status of cerebral autoregulation
Key: PaO_2: partial pressure of oxygen; $PaCO_2$: partial pressure of carbon dioxide; ICP: intracranial pressure; $PbtO_2$: partial brain tissue oxygenation; CPP: cerebral perfusion pressure; INR: international normalized ratio.

American College of Surgeons. (2024). Best practices guidelines: The management of traumatic brain injury. American College of Surgeons. https://www.facs.org/media/vqfgipfk/best-practices-guidelines-traumatic-brain-injury.pdf

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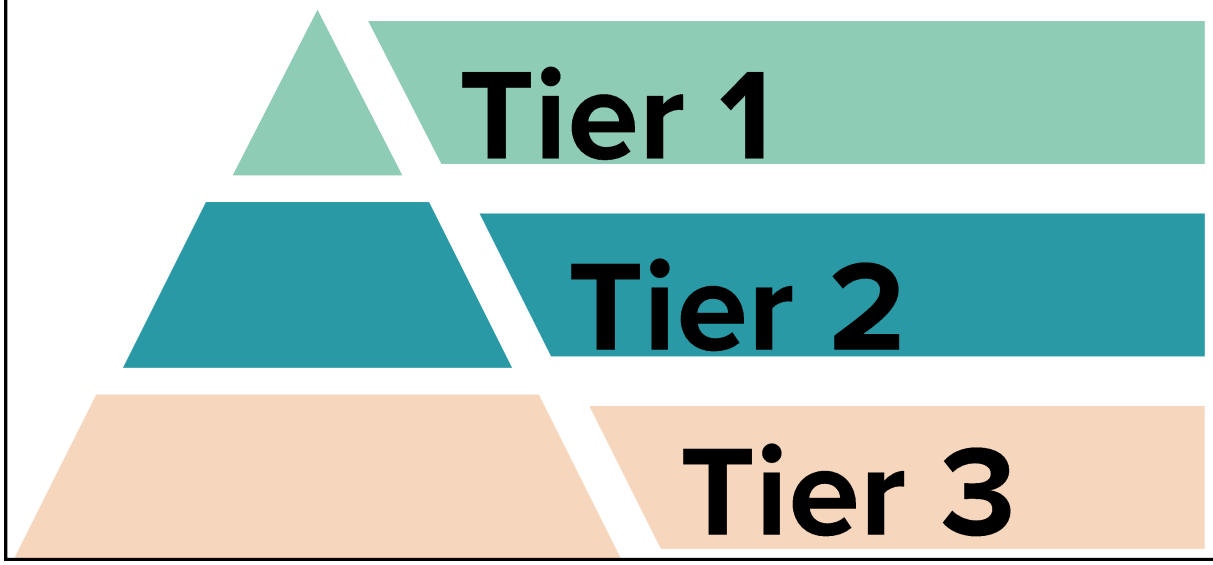
Tiered Approach to ICP Hypertension



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Tier One

Tier One
• ICP monitoring and CSF drainage • CPP 60 - 70 mmHg • $PaCO_2$ 35 - 48 mmHg • Hyperosmolar therapy • ICP guided analgesia & sedation • EEG monitoring • Anti-seizure medications

- **Opioid agonist:**
  - Fentanyl
  - Morphine
  - Hydromorphone
- **Sedatives/Anxiolytics:**
  - Propofol
  - Dexmedetomidine
  - Midazolam
  - Ketamine

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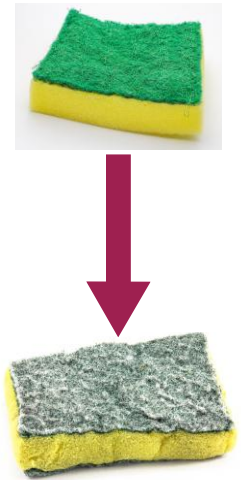
Tier One



- **Mannitol:**
  - 0.5-1 g/kg IV bolus q 4-6h
    - Onset 15 - 20 minutes, duration 90 - 6 hours
  - Crystallizes → Filter!
  - Monitor for **hypotension**
- **Hypertonic $NaCl^{-}$**
  - 2-23.4% continuous vs. bolus
  - Monitor serum Na^{+} q4-6h
  - Monitor for hypokalemia, hyperchloremic *acidosis*
- Limits for sodium (155 mEq/L) & osmolality (320 mEq/L)
- **Monitor for: rebound ICP, AKI, lytes, I/Os**

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Tier Two and Three

Tier Two	Tier Three
• MAP challenge & ICP guided CPP/MAP • $PaCO_2$ 32 - 45 mmHg • NMBA	• Pentobarbital or sodium thiopentone • Burst suppression • Decompressive craniectomy • Mild hypothermia (35 - 36°C)

- **MAP challenge:**
  - Record MAP, ICP, CPP, $PbtO_2$ baseline
  - ↑ MAP 10 - 15 mmHg for < 20 minutes
  - Record MAP, ICP, CPP, $PbtO_2$ during/post

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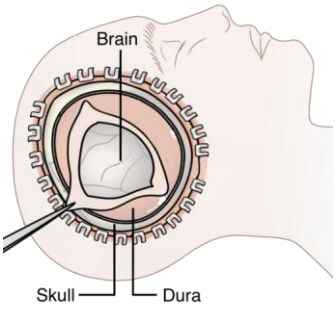
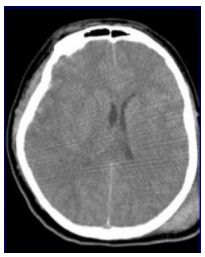
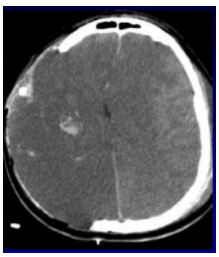
Surgical Interventions

- **Decompressive craniectomy**
  - Removal of a section of the skull to allow for swelling/expansion
- **Nursing Care:**
  - Caution with respositioning/mobility
    - Helment when OOB
  - Family education







Decompressive craniectomy diagram adapted from Cancer Research UK, via Wikimedia Commons (CC BY-SA 4.0).

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Recap

Monitor changes & trends	**Remember the 6Cs**	**Balance O_2/glucose supply & demand**
Limit secondary injury	**↓ ICP & improve CPP**	**Interdisciplinary collaboration**

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Resources

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