Severe TBI Management

Mechanisms of TBI

- **Focal** = contact injury
  - causing laceration, contusion, intracranial hemorrhage
- **Diffuse** = acceleration/deceleration injury, anoxic
  - leading to brain swelling, diffuse axonal injury

Primary v Secondary Brain Injury

- **Primary** = injury at the time of trauma/mechanical

- **Secondary** = delayed non-mechanical damage, result of a complication from the initial trauma
  - Cerebral edema,
  - Intracranial HTN
  - Neurotransmitter changes
  - Inflammation
  - **Hypoperfusion/hyperperfusion**
  - **Ischemia**

Primary v Secondary Brain Injury

- **Primary** = Injury prevention
  - Irreversible damage
  - + Necrotic death neurons, astrocytes, oligodendrocytes, neuronal interconnection disruptions (DAI)

- **Secondary** = Therapeutic/supportive measures
  - Penumbra – area of viable *but* threatened brain tissue around damaged tissue
  - Salvageable with support

Types of Primary TBI

- Skull Fracture
- ICH
  - EDH
  - SDH
  - SAH
  - IPH

Extra-axial

Intra-axial

- Coup–Contrecoup
- Diffuse Axonal Injury (DAI)

Skull Fractures

- Flat bones v skull base
- Linear v comminuted
- Degree of depression
- Degree of communication (dura, parenchyma)
  - Basilar w/ middle ear, nasopharynx, sinuses
- Greater risk of post-traumatic seizures
- Open – increased CNS infection risk

Epidural Hematoma (EDH)

- Laceration of dural veins/arteries between dura & skull
  - i.e temporal fracture & middle meningeal artery
- Arterial injury – higher pressure – faster neurologic deterioration
- "Lucid interval"

Subdural Hematoma (SDH)

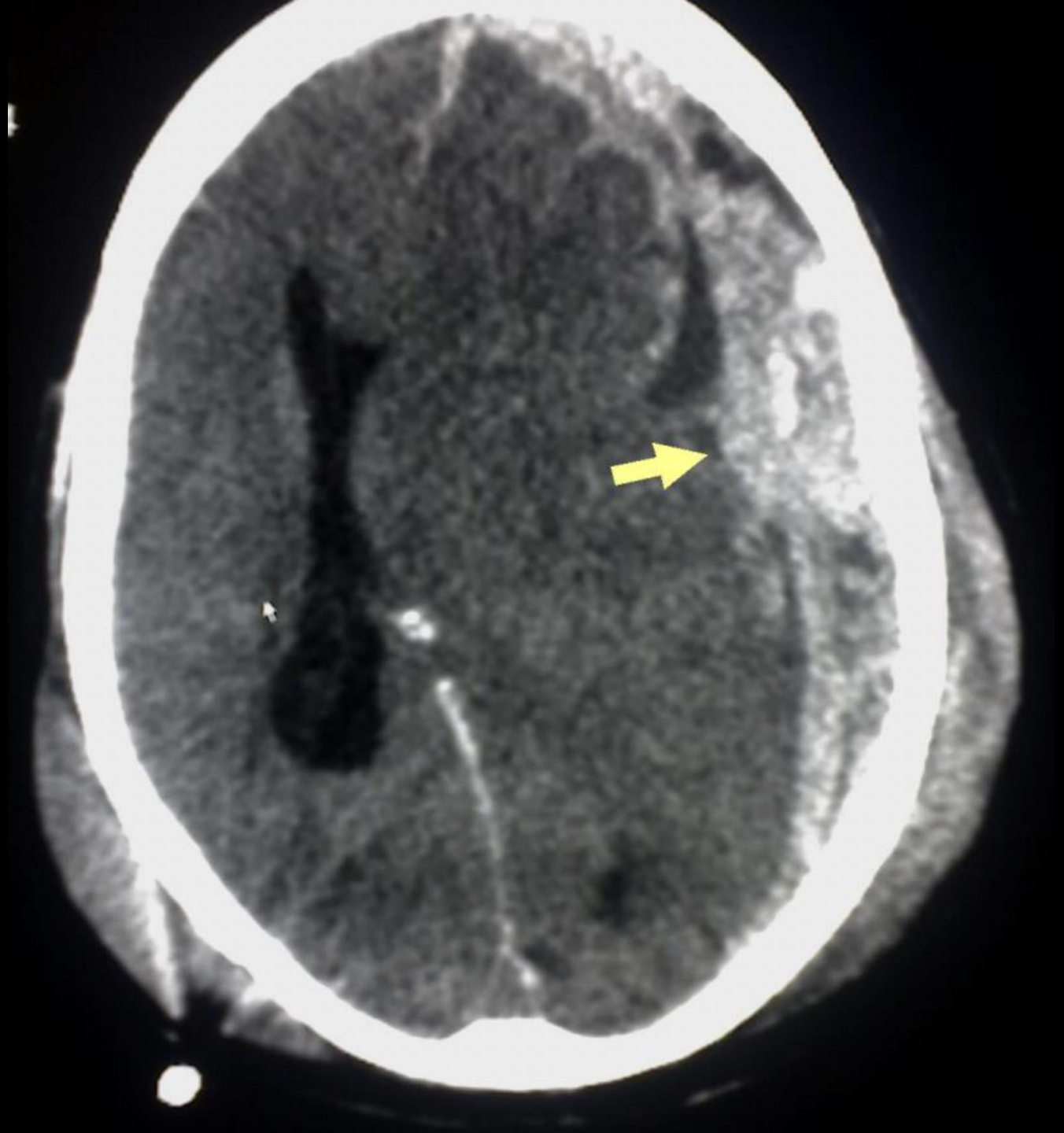
- Tearing of bridging veins
- Accumulation of blood w/in arachnoid membrane & dura
- Hematoma does not develop as rapidly, but leads to mass lesions
  - Mortality of 60–80% (higher than EDH)

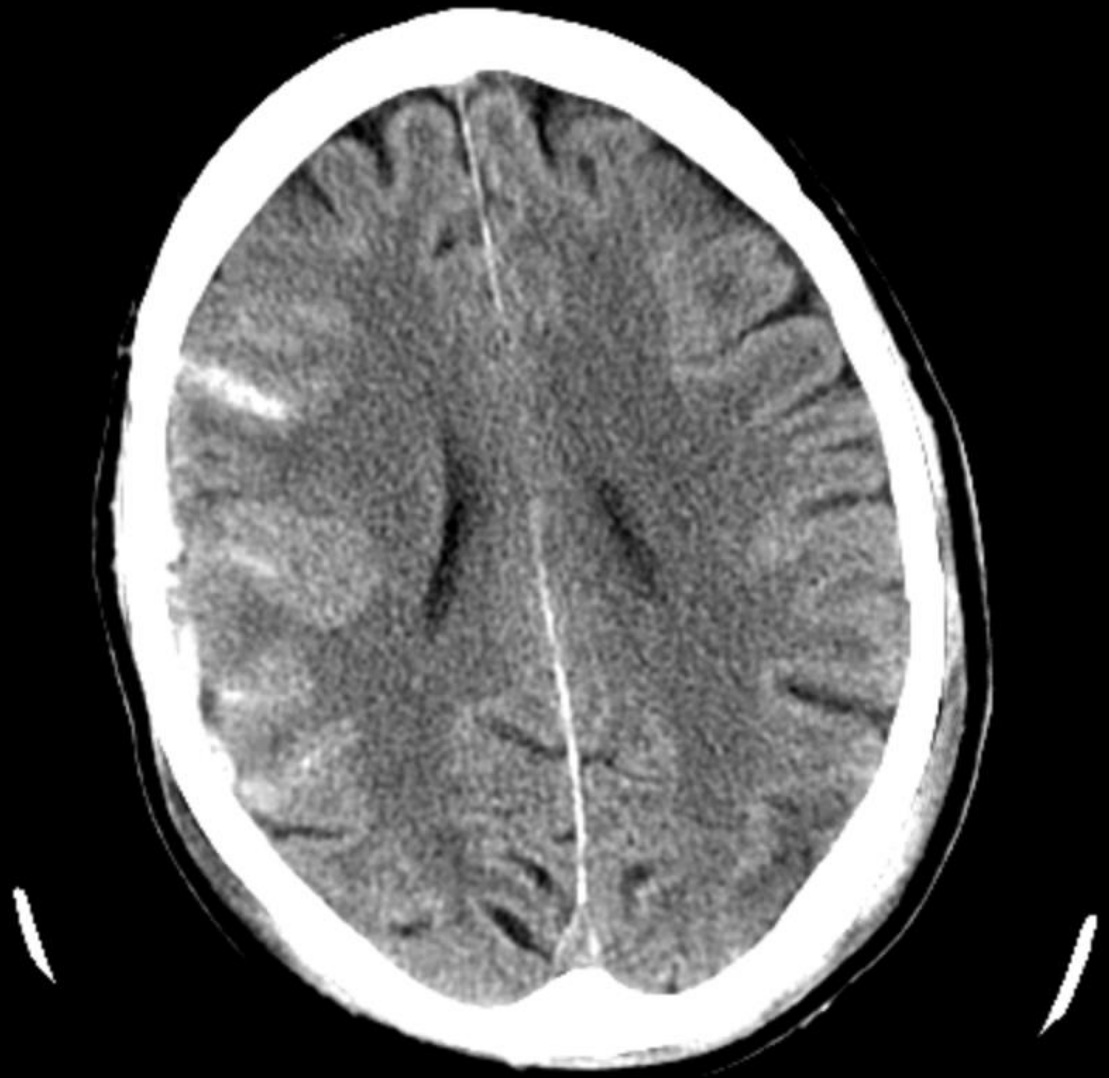
Subarachnoid Hemorrhage (SAH)

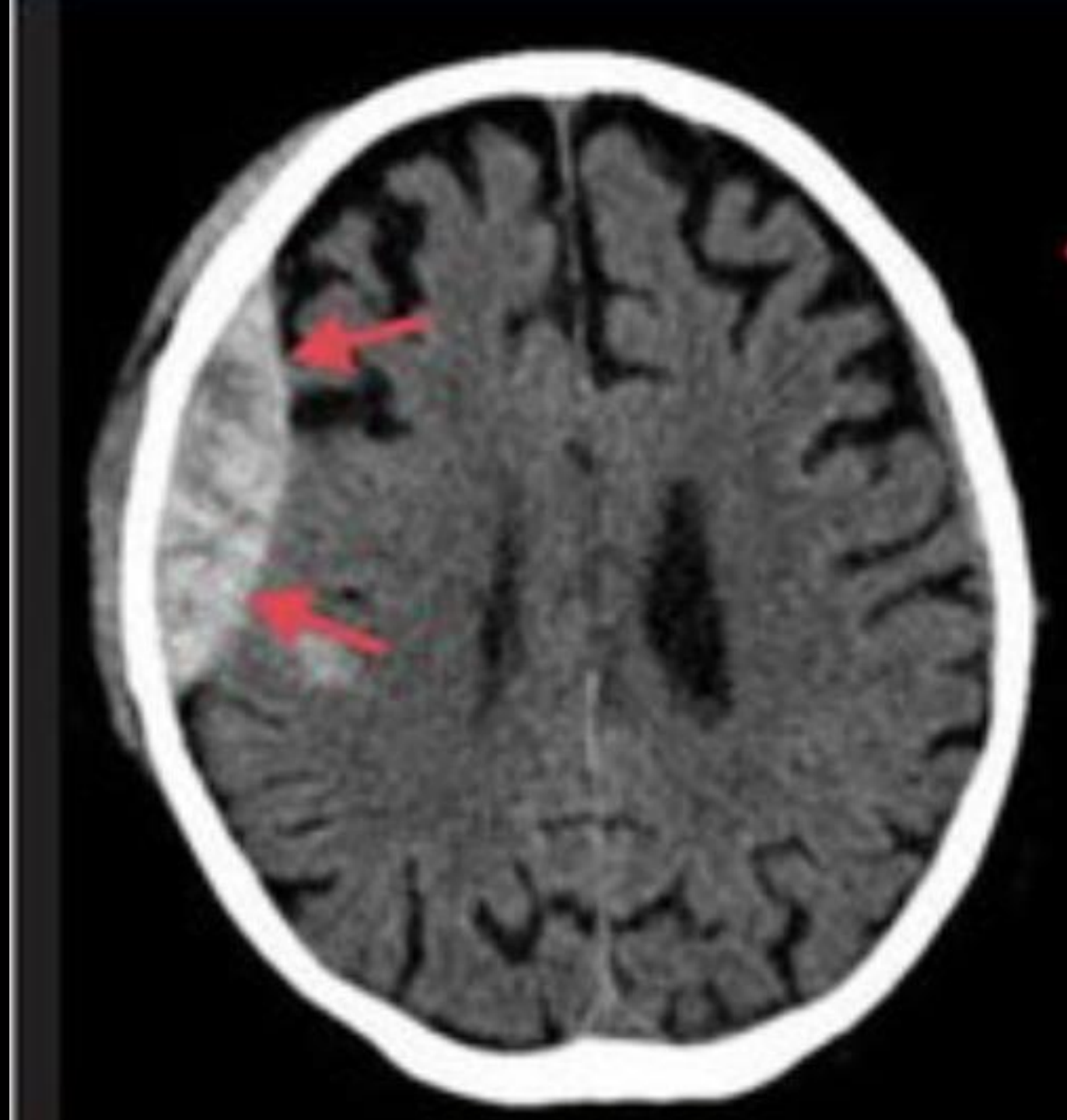
- Accumulation of blood between arachnoid & pia mater
- Adjacent to site injury/impact
- Portend worse outcome
- Outside of trauma associated w/ aneurysmal rupture
  - "Worst headache of life"
- Vasospasm

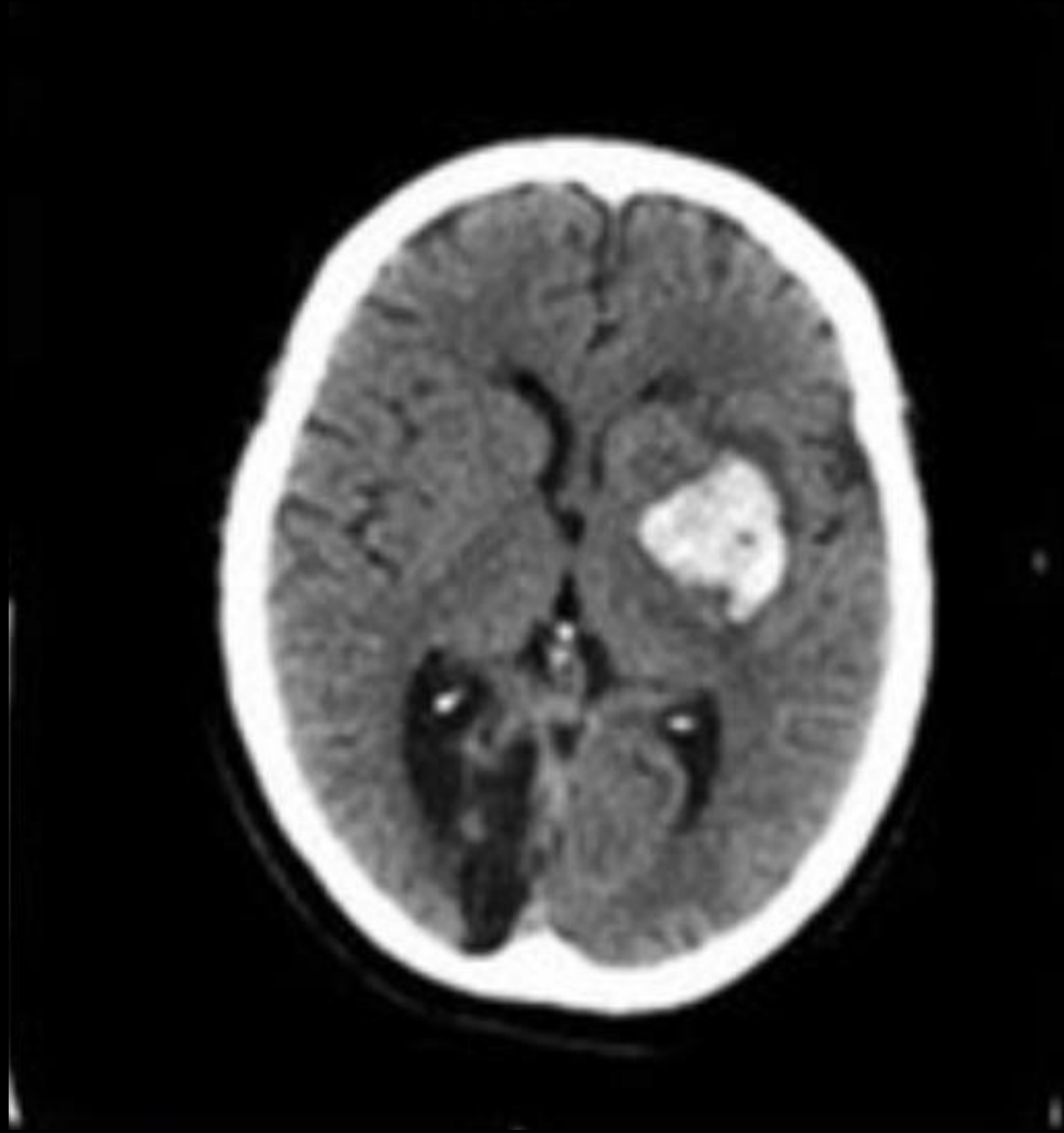
Intraparenchymal Hemorrhage (IPH)

- Frequently evolve
  - Increasing cerebral edema, mass effect
- Delayed IPH in 20% of TBI
  - Typically w/in 72h









Coup–Contrecoup Injury

- Contusion both initial site & opposite side of the insult,
  - Movement of the brain within skull
- Energy leads to rupture of micro vessels
  - Extravasation of blood & inability of these vessels to perfuse tissues

DAI

- Disruption of neuronal interconnections – shear/stretch injury
- CT normal in 50-80%
- Poor prognosis
- **Grades:**
- Grade 1: Mild diffuse external injury w/ microscopic white matter changes of the cerebral cortex, corpus callosum, brain stem
- Grade 2: Moderate DAI w/ focal corpus callosum lesions
- Grade 3: Grade 2 & additional brain stem lesions

Operative Indications

- **EDH**
  - Coma (GCS score < 9) with anisocoria
  - EDH > 30 cm^3 (regardless of GCS)

- **SDH**
  - Thickness > 10 mm **OR** midline shift > 5 mm (regardless of GCS)
  - Comatose (GCS < 9) & SDH < 10mm thick & midline shift < 5mm should undergo surgical evacuation if:
    - GCS decreased ≥ 2 between time of injury & admission
    - Presents with asymmetric or fixed & dilated pupils
    - ICP > 20 mm Hg
  -

Operative Indications

- **IPH**
  - Progressive neuro deterioration, refractory intracranial HTN, or mass effect (CT)
  - GCS 6-8 w/ frontal or temporal contusions > 20 cm^3 w/ midline shift > 5 mm and/or cisternal compression
  - Any lesion > 50 cm^3

- **Skull fractures**
  - Open, depressed > than thickness of cranium (to prevent infection)

Nonsurgical Treatment
aka
Critical Care Management

GOAL = Prevent Secondary Injury

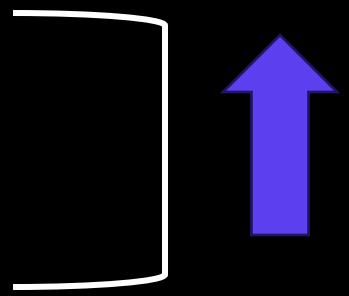
2° Injury in TBI

- **TBI = loss of cerebral autoregulation**
  - **Disrupted:**
    - blood flow/perfusion
    - O2 delivery
- Brain perfusion & oxygenation #1
- Hypotension & Hypoxia = INCREASE mortality with every episode

2° Injury in TBI

Other contributors to secondary injury:

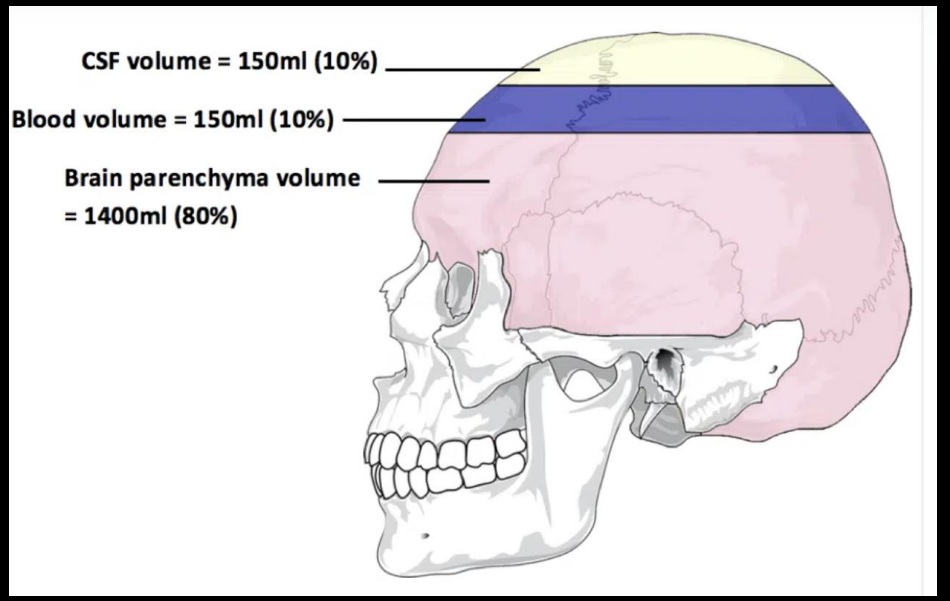
- Edema
- Electrolyte disturbances, hypoglycemia
- Infection
- Seizure
- Hyperthermia



Metabolic demand

ICP Management

- **Cerebral perfusion pressure (CPP) = MAP - ICP**
  - Represents cerebral blood flow & oxygen delivery
  - Goal 50-70mm Hg
  - TOO low - inability to meet metabolic demands – worse outcome
  - TOO high - cerebral edema
- ICP < 22mmHg
- **Monro-Kellie Doctrine =** total volume fixed (brain parenchyma, CSF, intracranial blood)
  - If 1 increases the volume of the others must decrease



ICP Monitoring

- Severe TBI (GCS 3-8 after resuscitation) & *abnormal* CT
- Or in severe TBI with *normal* CT if ≥ 2 of the following:
  - Age > 40 years,
  - Unilateral or bilateral motor posturing
  - SBP <90 mm Hg
- Internal monitors – invasive, introduced into specific anatomical locations (i.e intraparenchymal, intraventricular)
  - EVD is gold standard
- External monitors – accuracy? (TCD, TMD, ONSD)

ICP Management

- Treat pain & agitation
  - Propofol reduces cerebral oxygen consumption – possible neuroprotective effect (acute setting)
    - No evidence improved outcome
    - High dose therapy can worsen mortality
  - Barbiturates – only in high ICP refractory to max medical/ surgical treatment
- Elevation head of bed (30-45°)
  - Displaces CSF
  - Venous outflow

ICP Management

- **Hyperosmolar therapy**
  - Na goal 145-160 mEq/L - oncotic gradient
  - Hypertonic Saline
    - Oncotic gradient & volume expansion
    - Onset minutes (can last hours)
  - Mannitol
    - Osmotic diuresis
    - o.25-1g/kg, onset minutes (can last 6h)
    - AVOID in hypotensive

ICP Management

- **Hyperventilation**
  - Hypocarbia resulting in cerebral vasoconstriction
    - Acutely reduces cerebral volume – reduced ICP (temporizing therapy, bridge to emergent surgery ONLY)
    - Long term – vasoconstriction = reduced perfusion
    - Hypercarbia – vasodilation & increased ICP

Refractory ICP elevations

- Decompressive Craniectomy (DC) = consider for late-refractory ICP elevation *but not* early-refractory ICP elevation
- Diffuse Traumatic Brain Injury (DECRA) trial - secondary DC for early-refractory ICP elevation (w/in 72h)
  - No mortality benefit, poorer function outcomes (6m)
- Randomized Evaluation of Surgery with Craniectomy for Uncontrolled Elevation of Intracranial Pressure (RESCUEicp) trial- secondary DC for late-refractory ICP elevation
  - Mortality benefit
  - BUT HIGHER rates of vegetative state & severe disability

Hemodynamic goals

- **Blood pressure**
  - SBP ≥100 mm Hg (50 to 69 years)
  - SBP ≥110 mm Hg (15 to 49 or > 70 years)
- **Oxygenation/Ventilation**
  - Normal pH, normocarbia (35-40)
  - PaO2 80-200 mmHg (some suggest 120 mmHg as max)
  - Higher PEEP - increased intrathoracic pressure & impair venous return – can increased ICP & reduced CPP
    - Data mixed on whether clinically sig effect, must balance pt needs

Hemodynamic goals

- **Temperature** = Fever in 40-70% (pyrogens, disruption hypothalamic set point, infx)
  - Increase brain metabolic demand – cerebral ischemia/injury
  - Goal = normothermia
  - Avoid shivering – counter acts benefit via O2 reduction to brain tissue
    - Buspirone, meperidine
    - Dexmedetomidine
    - Magnesium?
- **Euglycemia**

Hemodynamic goals

- **Avoidance coagulopathy**
  - INR < 1.5, plt >100, Hgb>7
- **CRASH 3 (RCT TXA 2g within 3h of injury)**
  - Mild to moderate TBI (GCS>8) - reduction in head-injury-related
  - Severe TBI no difference
  - *Earlier* treatment more effective
  - No difference in VTE or seizures
- VTE ppx
  - ASAP
  - LMWH or SQH

Hemodynamic goals

- **Seizure ppx**
  - Phenytoin (or levetiracetam) recommended to decrease incidence of early PTS (w/in 7d of injury) (when benefit felt to outweigh the complication risk of meds)
- Early PTS not associated w/ worse outcomes
-