



Chapter 41 – Head injury

Episode Overview

- 1) List 7 causes of altered LOC in the trauma patient
- 2) List five herniation syndromes.
 - a. Describe the pathophysiology of uncal herniation and the typical presentation.
 - b. Describe the presentation of central herniation.
- 3) Describe how cerebral blood flow in relationship to the following parameters: PO₂ , PCO₂ , MAP and ICP. What are the indications for ICP monitoring?
- 4) What is the Canadian CT head rule? What are the inclusion criteria. What is the New Orleans CT head rule? What are the inclusion criteria? Which test is more sensitive? More specific?
- 5) What is a concussion? How is a concussion managed? What are potential complications? Define second impact syndrome & return to play
- 6) Outline the ED management goals of TBI.
 - a. differentiated between direct and indirect TBI
 - b. What are the indications for seizure prophylaxis following TBI?
 - c. What are the indications for antibiotics in TBI?
 - d. Complications of TBI?
- 7) 7 clinical features of basal skull fracture

Wisecracks

- 1) CT tips:
 - 3 signs of cerebral edema on CT
 - 5 differences on CT between SDH And EDH
 - List 3 CT findings in DAI
 - 2) What are: the Monroe-Kellie doctrine, the Cushing's reflex, What is kernihan's notch, and how does this syndrome present?
-

Rosen's in Perspective

- Most common causes: falls, MVC's,
- Leading cause of death for people < 25 yrs old
- There may be no external indicators on someone with a serious TBI

Principles of disease

ANATOMY AND PHYSIOLOGY

- Scalp and Cranium: "SCALP MAP"
 - **Scalp components:**
 - Dermis: thick layer of skin
 - Subcutaneous tissue: hair follicles, and BLOOD supply
 - Galea: tough fascia
 - Loose areolar tissue: loose tissue, where *subgaleal hematomas form*
 - Pericranium: firmly attached to the skull
 - Skull:
 - Frontal, ethmoid, sphenoid, occipital, and TWO parietal + temporal bones
 - Temporal bones are the thinnest



- Must consider **contrecoup injuries**
- Cranial vault can take up to 1900 ml of space
- Brain takes up 80% of the cranial vault
- And is covered by (outside to inside):
 - **Meningeal dura**
 - **Arachnoid layer**
 - **Pia**
 - **...then brain***
- Three divisions of the brain
 - Cerebrum, cerebellum, brainstem
 - CSF surrounds and protects the brain with buffering and cushioning.
 - Produced by the choroid plexus (in the lateral ventricles)
 - Pathway: ventricles → subarachnoid space → brain and spinal cord
 - CSF functions
 - Delivers brain substances, eliminates wastes, transports hormones,
 - **Normal pressure of CSF is 6-20 cm H₂O**
 - Blood in the ventricles can cause traumatic hydrocephalus
- **Blood brain barrier**
 - When intact it serves to balance ion and neurotransmitters.
 - ***Post-traumatic cerebral edema** affects the permeability of the BBB which can last for hours.
 - This can lead to vasogenic edema *

Brain cellular damage and death

Primary and secondary brain injuries:

- Primary:
 - Mechanical damage occurring at the time of head trauma -- brain lacerations, hemorrhages, contusions, tissue avulsions.
 - Permanent microvascular injury and cellular disruption lasting for hours and days
 - **Only interventions:** able to evacuate traumatic hematomas and reverse these injuries
 - Goal should be to promote primary prevention (avoid crashes, stop violence, prevent falls)
- Secondary
 - Intracellular and extracellular derangements due to massive depolarization of brain cells and ionic shifts
 - See Fig 41-3
 - ****all current therapies aim to prevent secondary neurologic injury***
 - All injuries lead to metabolic failure, cell membrane disruption, cellular edema, and cerebral edema...
 - No therapies to date show promise in consistently preventing secondary neurologic injury



What are the secondary systemic insults?

Secondary systemic insults:

- ***the final neurologic outcome is heavily affected by subsequent secondary brain injuries***

1) Hypotension

- a) Systolic BP < 90 mmHg
 - i) Potentiates ischemia and infarction
 - ii) **Doubles the mortality from head injury and worsens outcomes!**

2) Hypoxia

- a) PaO₂ < 60 mmHg
- b) Due to:
 - i) Transient or prolonged APNEA (brainstem compression or traumatic apnea)
 - ii) Partial airway obstruction due to blood/vomit/debris
 - iii) Chest wall injuries leading to interference in respiratory excursion.
 - iv) Pulmonary injury reducing effective oxygenation
 - v) Ineffective airway management (poor BVM, poor intubation,)
- c) Often occurs in the pre-hospital setting

3) Anemia

- a) Reductions in the oxygen carrying capacity of the blood → increased mortality due to limited oxygen delivery to the brain

4) Hyperpyrexia

- a) >38.5 degrees.
- b) Thought to worsen injury by stimulating injured brain tissue metabolism

5) Hypercarbia

6) Coagulopathy

7) Seizures

Pathophysiology

Increased ICP

- Three methods to decrease ICP:
 - 1) **hyperventilation**
 - 2) **osmotic and diuretic agents**
 - 3) **CSF drainage**
 - Has no prospective data to support its use, but is done to avoid brainstem compression and arrest

Brain swelling and cerebral edema

- Congestive brain swelling
 - Increased intracranial blood volume due to hyperemia caused by vasodilation needed to maintain increased metabolic needs of damaged brain tissue
- Cerebral edema
 - Absolute increase in cerebral water content
 - Diffuse cerebral edema may occur with a head injury, but does NOT correlate with the severity of head injury
 - 3 findings: loss of sulci, loss of grey-white interface and collapsed ventricles
 - Vasogenic edema
 - Failure of the BBB endothelial junctions → leading to transvascular leakage
 - Cytotoxic edema



- An **intracellular process due to membrane pump failure**
- Usually due to post-traumatic ischemia and tissue hypoxia caused by a decrease in cerebral blood flow (due to dying cells)
- This is often worsened by increasing ICP

1) List 7 causes of altered LOC in the trauma patient

Altered LOC

- Consciousness (awareness of SELF and the Environment) requires a functioning RAS and cerebral cortices.
- Etiologic factors:
 - Hypoxia
 - Hypoglycemia
 - Post-seizure
 - Post-intoxicating drugs
 - Brain or brainstem compression due to swelling or a mass
 - Bleed
 - Hypotension

2) List five herniation syndromes.

Herniation types:

First three are all transtentorial herniation types

- Think of the Cartesian plane, these things shift the cerebrum \leftrightarrow or \wedge or front-back
1. Uncal herniation
 - a. Most common type: due to traumatic extra-axial hematomas in the lateral middle fossa or temporal lobe.
 2. Central transtentorial herniation
 - a. Due to an expanding lesion at the vertex, frontal, or occipital brain
 3. Upward transtentorial herniation
 - a. Expanding posterior fossa tumour
 - b. Signs:
 - i. Rapid decline of LOC, downward conjugate gaze, pinpoint pupils
 4. Cerebellar-Tonsillar herniation
 - a. Due to cerebellar tonsils herniating through the foramen magnum
 - i. Cerebellar mass or large central vertex mass
 - b. Signs:
 - i. Sudden respiratory / cardio collapse, pinpoint pupils, *flaccid quadriplegia* (corticospinal tracts are compressed)



Describe the pathophysiology of uncal herniation and the typical presentation.

- See fig. 41-5
- Signs: anisocoria, ptosis, impaired EOMs, sluggish pupil on the Ipsilateral side
 - c. A dilated, non-reactive pupil = compressed oculomotor nerve
 - d. CONTRALATERAL: positive babinski's sign (upward going big toe and outward fanning of the other toes)
 - i. Contra-lateral hemiparesis
 - Then may progress to bilateral decerebrate posturing
 - ii. As the brainstem becomes compressed: agitation > restlessness > confusion > coma > cushing's reflex > cardiac collapse

Describe the presentation of central herniation.

Signs: may be subtle LOC changes → bilateral motor weakness, > pinpoint pupils > increased muscle tone and bilateral babinski's > Posturing, respiratory pattern changes (sighs, yawns, shallow-irregular breaths, then respiratory arrest)

3) Describe the effect on cerebral blood flow in relationship to the following parameters: PO₂ , PCO₂ , MAP and ICP.

- Brain consumes 20% of the body's oxygen content and optimal cerebral blood flow is maintained by the cerebral vessels adapting to physiologic conditions: between **MAP 60-150 mmHg**
- Cerebral vasoCONstriction:
 - Hypertension, alkalosis, hypocarbia
 - This is why we hyperventilate coning or severely head injured patients showing signs of brain herniation -- to vasoconstrict (pg. 341) -- 1 mmHg of CO₂ change can decrease cerebral diameter by 2-3%
 - If the PCO₂ becomes too low, the brain can become ischemic.
 - The Goal is **30-35mmHg** of CO₂
 - This loses its effectiveness after 12 hrs and the brain can become **DAMAGED** due to vasodilation and reperfusion injury
 - Therefore **hyperventilation only for TBI patients showing ACUTE signs of increased ICP**
- Cerebral vasoDILation:
 - Hypotension, acidosis, hypercarbia
 - In a TBI - **the brain is not able to respond normally to changes in oxygen levels**
 - Normal functioning BBB: declines in P_{O2} lead to cerebral vessel dilation, however injured brain is unable to properly DILATE to improve cerebral oxygenation (and also remove metabolic end products that are toxic to the brain)
- **Cerebral perfusion pressure: CPP = MAP - ICP** (resistance to CBF by mean systemic pressure and ICP)
 - **CPP is the pressure gradient across the brain**
 - **CBF = is constant from 50-160 mmHg**



- ICP effects are greater than mean systemic venous pressures
- Goals: to maintain MAP and reduce ICP so that CPP can sustain metabolic needs.

So in summary:

- *PCo2 = direct relationship with CBF.* So, high pco2 higher ICP.
- PO2 = inverse relationship with CBF
- MAP = direct
- ICP = inverse

What are the indications for ICP monitoring?

From Trauma.org:

- Severe head injury (GCS <9)
- Moderate head injury (GCS 9-12) who cannot be monitored with serial neurological exams

4) The CT head rules for MINOR head trauma

- (adapted from: <http://thesgem.com/2015/02/sgem106-o-canada-canadian-ct-head-rule-for-patients-with-minor-head-injury/>)

What is the Canadian CT head rule?

From CJEM:

Canadian CT Head Rule

CT Head is only required for minor head injury patients with any one of the following findings. Minor head injury patients present with a GCS score of 13-15 after witnessed loss of consciousness, amnesia, or confusion.

High-Risk (for Neurosurgical Intervention)

1. GCS score < 15 at 2 hours after injury
2. Suspected open or depressed skull fracture
3. Any sign of basal skull fracture *
4. Vomiting \geq 2 episodes
5. Age \geq 65 years

Medium-Risk (for Brain Injury on CT)

6. Amnesia before impact \geq 30 minutes
7. Dangerous mechanism **

** Signs of Basal Skull Fracture:*

- hemotympanum, 'raccoon' eyes, CSF otorrhea / rhinorrhea, Battle's sign

*** Dangerous Mechanism:*

- pedestrian struck by motor vehicle
- occupant ejected from motor vehicle
- fall from elevation \geq 3 feet or 5 stairs

Rule not applicable if:

- Non-trauma case
- GCS < 13
- Age < 16 years
- Warfarin or bleeding disorder
- Obvious open skull fracture



Primary Outcome:

Need for neurological intervention, defined as need for neurologic intervention as death within 7 days due to the head injury or need with 7 days for craniotomy, elevation of skull fracture, intracranial pressure monitoring, or intubation for head injury. ~ from thesgem.com

What are the inclusion criteria:

- “Adult patients presenting to the emergency departments at 10 large Canadian hospitals with **Glasgow Coma Scale 13** or greater within 24 hours after **blunt head trauma** resulting in witnessed loss of consciousness, amnesia, or witnessed disorientation.”
- Age < 16 excluded
 - Minimal trauma with no LOC/amnesia/disorientation
 - Anticoagulation
 - PTSSs
 - Unclear hx, focal deficits, penetrating injury, depressed skull #

What is the New Orleans CT head rule?

Head CT is required if one or more of the following are present:

- Headache
- Vomiting
- Age >60yr (!)
- Drug or Alcohol Intoxication
- Persistent anterograde amnesia (deficits in short-term memory)
- Visible trauma above the clavicles
- Seizure

What are the inclusion criteria?

- **Inclusion Criteria:** Age >18, GCS 15 and Blunt head trauma occurring within previous 24hr causing LOC, amnesia, or disorientation

Which test is more sensitive?

- Both 100% sensitive for pt's needing neurosx intervention

More specific?

- Canadian CT head rule!
 - “Both the New Orleans and the Canadian rules had 100% sensitivity, but the Canadian Rule had a higher specificity for all three outcome measures (36.3 versus 10.2 to identify traumatic intracranial lesions on CT, 35 versus 9.9 for clinically important brain injuries, and 80.7 versus 9.6 to identify need for neurosurgical intervention).” ~ from thesgem.com



5) What is a concussion? How is a concussion managed? What are potential complications? Define second impact syndrome & return to play.

Concussion:

- Is a complicated minor TBI
 - Commonly Occurring in collision sports
 - Leads to short-lived distortion of axons and neuroanatomy
 - Levels of neurotransmitters remain elevated and a hypermetabolic state persists up to weeks -- which can cause secondary injury
 - Functional PET scanning is the only imaging that shows signs of damage
- Post-concussive syndrome
 - Affects cognitive, psychomotor, behavioural performance
 - Headache, sensory sensitivity, memory/concentration problems, sleep changes, mood changes, etc.
 - People with dizziness, headache, and nausea early after head trauma may have a 50% chance of having symptoms at 6 months
- RE-injury risk is highest within the first 10 days
 - Due to deficits in balance, reflexes, speed, information processing
 - **Second impact syndrome:**
 - When an athlete sustains a second concussion before recovering completely from the previous concussion
 - Causes a rapid, at times fatal decline, due to cerebral edema
 - THIS IS a debated entity - some question its validity and existence
- A careful field side assessment is important for determining who can play and who can't.
- Return to play protocols are important - in a stepwise approach
 - Only move on to the next step if the athlete is asymptomatic
 - Generally athletes should return to play one week AFTER they are totally asymptomatic (during training and exercise)

6) Outline the ED management goals of TBI.

Management of TBI:

See box 41-2; 3; 4;

<p>SEVERE</p> <p>GCS<8 or intra-cranial contusion/hematoma by CT</p>	<p>Out of hospital care:</p> <ul style="list-style-type: none"> ● Necessary airway mgmt and IV access to treat hypotension and hypoxia <ul style="list-style-type: none"> ○ There is a risk-benefit debate in the literature about the safety of out of hospital intubation - requires good protocols and training to do safely ● Neuro exam ● Treatment of scalp bleeding ● Sedatives/paralytics for agitated patients 	<p>Notes:</p> <ul style="list-style-type: none"> ● 10% of TBI's in the ED are severe ● 25% of these need neuroSx ● ***initial GCS or CT does NOT reliably predict prognosis** <ul style="list-style-type: none"> ○ Depends on: ○ Age, combx, initial motor activity, pupil response, secondary insults ● 35% mortality rate
	<p>Emergency department care:</p> <p>Airway</p> <ul style="list-style-type: none"> ● Pre-intubation neuroexam: and then RSI +/- lidocaine 	<p>Ancillary evaluation:</p>



	<ul style="list-style-type: none"> • Caveats with etomidate, succinylcholine, <p>Hypotension</p> <ul style="list-style-type: none"> • Identify the cause** <ul style="list-style-type: none"> ◦ Bleeding, high spinal cord injury (hypotension despite fluids and inappropriate bradycardia) • Keep systolic BP > 90 mmHg • No consistent benefit from hypertonic saline <p>Hyperventilation</p> <ul style="list-style-type: none"> • An acute life saving intervention. Goal is to get PCO₂ to 30-35 mmHg. Causes vasoconstriction - onset 30 seconds and peaks at 8 mins. SHOULD NOT be used flippantly or prolongedly <p>Osmotic agents</p> <ul style="list-style-type: none"> • Mannitol (0.25 - 1 g/kg) - mixed evidence. Draws tissue water into the vascular space -- to reduce brain volume/edema. Peak effect in 60 mins <ul style="list-style-type: none"> ◦ Reserve for lifesaving situations - or rapidly deteriorating neuro exam. ◦ Helpful in hypotension and may help CBF by reduced blood viscosity and resistance • Hypertonic saline: reduces ICP, but can cause other complications (renal failure, rebound ICP increase) <ul style="list-style-type: none"> ◦ Variable evidence for and against it... <p>Barbiturates</p> <ul style="list-style-type: none"> • Used to "reduce cerebral metabolic demands of injured brain tissue" • Rarely used in the ED...Pentobarbital may be used in ICU? <p>Steroids</p> <ul style="list-style-type: none"> • NO benefit to giving steroids in head injury • Show INCREASED mortality <p>Hypothermia</p> <ul style="list-style-type: none"> • Inconclusive evidence: no clear benefit for improving morbidity or mortality in for mod/severe TBI • Some studies show promising results...but are underway still...as of 2016.. <p>Cranial decompression</p> <ul style="list-style-type: none"> • For those with signs of herniation, who don't respond to other means of decreasing ICP. • Emergency trephination/burr holes: for people who "talk then deteriorate" * • Emergency decompressive craniectomy: must be done EARLY <p>Seizure prophylaxis</p> <ul style="list-style-type: none"> • Post-traumatic seizures (12% blunt trauma, 50% in penetrating trauma) • Cause: <ul style="list-style-type: none"> ◦ Hypoxia, hypercarbia, increased ICP and worsen secondary neuro injury • See table 41-2: for prophylactic indications <ul style="list-style-type: none"> ◦ Depressed skull # ◦ paralyzed/intubated pt. ◦ Any seizure at any time (or hx of same) ◦ PENETRATING brain injury ◦ Any brain bleed (epi,SDH, ICH) • Benzo's for immediate seizures • Phenytoin, fosphenytoin, for long-term mgmt. • Consider continuous EEG monitoring <p>Antibiotic prophylaxis</p> <ul style="list-style-type: none"> • Risk in: <ul style="list-style-type: none"> ◦ Penetrating head injury ◦ Depressed skull # ◦ Complicated scalp lacerations • Not for mere otorrhea/rhinorrhea <p>Misc:</p> <ul style="list-style-type: none"> • Give recombinant complex concentrated factors for people with ICH and anticoagulant use, but not for all comers with ICH 	<p>Lab tests</p> <ul style="list-style-type: none"> • Urine tox, blood alcohol level • CBC, lytes, glucose • Coagulation studies <p>Neuroimaging</p> <ul style="list-style-type: none"> • Non-con CT head • MRI for more definitive evaluation once patient is stabilized. <p>Disposition</p> <p>Consultation</p> <ul style="list-style-type: none"> • Neurosurg. Consult ASAP post CT <p>Transfer to neuro surgical centre</p> <p>Priority management</p> <ul style="list-style-type: none"> • Some patients need general surgery STAT for abdominal or chest wounds -- so urgent ventriculostomies or bilateral trephinations are done concurrently if possible!
<p>Moderate</p> <p>GCS</p>	<p>Clinical features:</p> <ul style="list-style-type: none"> • Change in LOC, progressive headache, post-trauma seizures, vomiting • Confusion, somnolence, but can still obey 	<p>Damage:</p> <ul style="list-style-type: none"> • Often due to MVCs • Neuronal cell membrane dysfunction and a mild depletion of ATP



<p>9-13</p>	<ul style="list-style-type: none"> • **watch for the “talk and deteriorate” patient** within 48 hrs - due to epidural/subdural hematoma <p>Management:</p> <ul style="list-style-type: none"> • Close observation for changing mental status! • Urgent CT with neurosurgery with any new focal findings or change in GCS • Skull radiographs are rarely helpful - unless no CT available <p>Disposition:</p> <ul style="list-style-type: none"> • All should be admitted for observation, even if a normal CT scan <p>Complications:</p> <ul style="list-style-type: none"> • 20% mortality! • At 3 months 70% are unable to return to work • HUGE long term morbidity • 50% are left with long term disabilities 	<p>Most changes are reversible, if secondary injury is avoided</p> <p>“When no neurosurgeon is available and a pt. Develops an acute herniation syndrome which does not respond to hyperventilation and mannitol, ED trephination and hematoma evacuation should be considered”</p> <p>Some may need followup MRI to look for lesions not seen on CT</p>
<p>Mild* TBI</p> <p>Brief and temporary Interruption of neurologic function</p>	<ul style="list-style-type: none"> • Neurobiological damage, with no damage to microstructures <p>GCS 14-15 - **GCS is not sensitive enough to prognosticate minor TBI - it misses subtle alertness and Neuro changes >this is a CLINICAL diagnosis</p> <p>> requires high acceleration-deceleration or rotary forces to the head (whiplash should not cause this injury)</p> <p>Clinical and historical features:</p> <ul style="list-style-type: none"> • Headache, Nausea, disorientation, confusion, amnesia, • Signs: <ul style="list-style-type: none"> ○ Imbalance, impaired verbal memory, slowed speech, amnesia, inability to perform tasks of memory and concentration, retrograde amnesia ○ post-traumatic amnesia = impaired information encoding. This is the best predictor of the severity of injury • High risk Features: <ul style="list-style-type: none"> ○ Focal findings, signs of trauma above clavicles, worsening symptoms intoxication, age >60 or <2 <p>5-15% of people who come to the ED with mild TBI may have an abnormal CT scan; but < 1% need neurosurgery</p> <p>Low risk - factors: - see box 41-3</p> <ol style="list-style-type: none"> 1. Accurate Hx, trivial mechanism 2. Asymptomatic now; no or mild headache, no vomiting 3. No other injuries and no focal findings 4. Normal pupils 5. Intact orientation and memory 6. Initial GCS of 15 7. < 24 hrs since injury <p>Delayed complications likely to occur in:</p> <ul style="list-style-type: none"> • Anticoagulated pts. <ul style="list-style-type: none"> ○ All head injury rules exclude these populations - so the threshold to scan them is very low • VP shunts 	<p>Due to “heightened ionic flux, surges in levels of glutamate, enzyme pathway disruptions, accumulation of lactate, and nitric oxide”; axonal stretching and twisting</p> <ul style="list-style-type: none"> • May or may not have LOC <p>Imaging</p> <ul style="list-style-type: none"> • Large body of variable research based on study populations, methods and definitions, and outcomes • ACEP clinical policy : see box 41-4 <ul style="list-style-type: none"> ○ CT head only indicated in adults with LOC or posttraumatic amnesia if they meet one of the other risk factors/clinical features • “The final choice of which clinical decision rule to apply - rests on the clinician’s attitude toward uncertainty” <p>Best approach: selective CT scanning vs. Observation based on risk stratification</p> <ul style="list-style-type: none"> • No role for skull radiography • MRI is the test for: <ul style="list-style-type: none"> ○ DAI, ischemia, hemorrhagic lesions, posterior fossa ○ Will show things that a normal CT head misses <p>Ancillary studies:</p> <ul style="list-style-type: none"> • Consider toxic screens if a patient remains altered • No bio markers have shown promise in predicting long term outcome <p>Disposition:</p> <ul style="list-style-type: none"> • Based on risk stratification: d/c, d/c with close observation and followup; ED observation for 4-24 hrs; • Clinical sobriety should be achieved before discharge •



What are the indications for seizure prophylaxis following TBI?

Box 41-2 Indications for Acute Seizure Prophylaxis in Severe Head Trauma

Depressed skull fracture
Paralyzed and intubated patient
Seizure at the time of injury
Seizure at emergency department presentation
Penetrating brain injury
Severe head injury (Glasgow Coma Scale score 8 or lower)
Acute subdural hematoma
Acute epidural hematoma
Acute intracranial hemorrhage
Prior history of seizures

What are the indications for antibiotics in TBI?

1. Penetrating TBI
2. Depressed skull #
3. Complex, contaminated, deep scalp lacerations
4. Fever in someone with a basilar skull #
 - ***prophylactic abx in the acute setting of CSF leaks and basilar skull fractures are NOT recommended*** - but they do decrease the incidence of meningitis (no clear consensus)

Complications after head injuries?

Neurologic complications

- Seizures
 - Acute PTSs
 - Brief
 - Self-limited
 - Very frequent in children
 - Subacute PTSs - 24-48 hrs post
 - Due to worsening cerebral edema, small hemorrhages, penetrating injuries
 - Prophylaxis reasons: - see box above
 - Med Hx of seizures Or any seizure
 - intracranial blood
 - Penetrating wounds
 - Unable to assess seizure activity because of: low GCS, paralysis,
 - Meds:
 - Phenytoin or fosphenytoin
 - No recommendations to suggest long term prophylaxis - this depends on their clinical course
 - TBI is thought to account for 20% of adults with epilepsy - and late seizures may present up to 12 yrs post injury



- CNS infections
 - Meningitis after basilar skull fractures
 - Fever, AMS, focal findings,
 - Common in people with CSF leaks
 - Fever occurs within 3 days of injury
 - Caused by pneumococcus
 - Trxt: ceftriaxone and vancomycin
 - Fever > 3 days - usually due to gram -ve
 - Txt: ceftriaxone with vancomycin
 - ***prophylactic abx in the acute setting of CSF leaks and basilar skull fractures are NOT recommended*** - but they do decrease the incidence of meningitis (no clear consensus)
 - Brain abscess
 - Rare
 - May be part of a fragment of bone left in the cranium or post-op complication
 - Post-traumatic fistulae with tracts into sinuses are a risk factor
 - S+s:
 - Headaches, vomiting, fevers, declining mental status, increased ICP, focal findings, nuchal rigidity, seizures
 - NEED contrast enhanced CT head
 - LP is dangerous and should not be done in someone with signs of increased ICP
 - Trxt: is surgery, unless there is isolated cerebritis.
 - Common bugs: gram -ve anaerobes and staph aureus .
 - Cranial-osteomyelitis
 - Pain, tenderness, erythema at the site
 - MAY need bone scans to make the dx,
 - Abx, surgery are the treatment

Medical complications

- Disseminated intravascular coagulation
 - Injured brain releases tissue thromboplastin. -- which activates the extrinsic clotting system → can lead to DIC
 - Increased risk of intra-cranial hemorrhage
 - Dx:
 - INR, PTT, platelets, fibrinogen levels, fibrin degradation products.
 - Trxt:
 - To correct these abnormalities
 - ****patients should receive prophylactic LMWH to prevent VTE - they do not worsen ICH hematoma expansion
- Cardiac dysfunction
 - A clear relationship between intracranial bleeding and cardiac dysrhythmias:
 - In 50% of ICH, and 70% of cases of SAH
 - Etiology may be from concurrent blunt chest injury and/or underlying cardiac disease
 - Pathophysiology thought to be from autonomic nervous system dysfunction (changing ventricular depolarization); high levels of catecholamines,
 - Lead to:
 - SVT
 - Diffuse large upright or inverted T waves



- Prolonged QT interval
- ST segment elevation or depression
- U waves
- **Key priority:** to ensure adequate brain perfusion and oxygenation ; some may need cardiac monitoring!
- Neuro-cardiogenic pulmonary edema
 - May develop in minutes to days post injury
 - Pathophysiology hypothesis:
 - Catecholamine surges
 - blasts of elevated hydrostatic forces and increased capillary permeability due to the primary brain injury
 - A systemic inflammatory reaction
 - Key goal is to lower ICP
 - CLOSE ventilator management is essential

7) Name 7 clinical features of basal skull

- Basilar skull fracture findings: see box 41-1:
 - Blood in ear canal
 - Hemotympanum
 - Rhinorrhea
 - Otorrhea
 - Battle's sign (retro auricular hematoma)
 - Raccoon's sign (periorbital ecchymosis)
 - Cranial nerve deficits:
 - Facial paralysis
 - Decreased auditory acuity
 - Dizziness
 - Tinnitus
 - Nystagmus
- Carotid bruit (carotid artery injury) - hyperflexion and extension injury

Wisecracks

[1] CT tips: Three signs of cerebral edema on CT, Five differences on CT between SDH And ED, List 3 CT findings in DAI

CT tips:

Three signs of cerebral edema on CT

- Sulcal effacement
- Loss of grey white differentiation
- Compression of ventricles (ventricular effacement)



Five differences on CT between SDH and EDH

- SDH
 - Crescentic (crescent moon shaped)
 - Crossing suture lines - more extensive
 - Layers
 - Damage to the bridging veins
- EDH
 - Lens shaped
 - One cranial bone space
 - Middle meningeal artery damage

List 3 CT findings in DAI

- Trick question!
- “CT is particularly insensitive to non-haemorrhagic lesions (as defined by CT) only able to detect 19% of such lesions, compared to 92% using T2 weighted imaging 4. When lesions are haemorrhagic, and especially when they are large, then CT is quite sensitive. As such, it is usually a safe assumption that if a couple of small haemorrhagic lesions are visible on CT, the degree of damage is much greater.”~ from radiopaedia.org

[2] What are: the Monroe-Kellie doctrine, the Cushing’s reflex, What is kernihan’s notch, and how does this syndrome present?

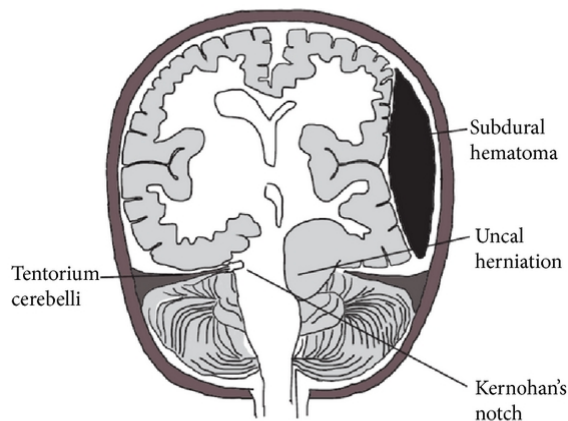
Monro-kellie doctrine

- Sum of brain volume, CSF, and blood must remain constant, so increases in any one leads to decreasing volumes of the others
- The CSF is the first to move out of the cranial vault. Once the 50-100 ml of space is filled ICP increases and CPP is compromised
- **Vasoparalysis occurs** and the autoregulation mechanics fail. → cerebral vasodilation → vasogenic edema → increases in ICP → cerebral blood flow cases and brain death occurs

Cushing’s reflex

- **Hypertension, bradycardia, fluctuating respiratory effort** - specific to increased ICP, but only occurs 30% of the time
- When a herniation syndrome presents, mortality is nearly 100% unless urgent reversal is undertaken

What is kernohan's notch, and how does this syndrome present?



- e. ***Kernohan's notch syndrome*** false localizing of motor findings due to contralateral cerebral peduncle compression (midbrain) by the tentorium cerebelli.
 - i. It is a **FALSE** localizing sign: ie. the patient has a left SDH causing an uncal herniation to the right, producing decreased LOC and **LEFT** (rather than right) sided hemiparesis.