HEAD INJURY

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POS Large Group Session
MUMC, Room 4E20
Mechanisms of Head Injury

- Three varieties of Head Trauma:
  - 1. Skull fractures (linear, depressed, basilar)
  - 2. Focal injuries:
    - Contusions (coup, contrecoup, intermediate)
    - Haematomas (EDH, SDH, ICH)
  - 3. Diffuse Injury:
    - Concussion
    - Diffuse axonal injury
Pathophysiology of Head Injury

1. Contact (blow to head):
   - Local – skull fracture, EDH, coup contusions
   - Remote – vault and basilar fracture
   - Shock wave injury – contrecoup, ICH

2. Acceleration (head motion):
   - Concussion
   - SDH, DAI, contrecoup and intermediate contusion
   - ICH
Skull Fracture - Linear:

- Linear skull fracture:
  - Dark black, straight, no branching, thin width

- Vessel groove:
  - Grey, curving, branching, thick width

- Suture line:
  - Grey, suture line, jagged, wide
Skull Fracture - Basilar:

- Usual locations:
  - Petrous temporal bone, orbital surface of frontal bone, basiocciput

- Clinical signs:
  - Battle sign
  - CSF otorrhoea, rhinorrhoea
  - Haemotympanum
  - Racoon eyes
  - CN deficits
Skull Fracture – Basilar (cont.):
Skull Fracture – Battle’s Sign:
<table>
<thead>
<tr>
<th>Endolymphatic (EAC) Pathway Disruption</th>
<th>Middle Ear Ossicles, Tympanic Membrane (TM) Disruption</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transverse</td>
<td>Longitudinal</td>
</tr>
<tr>
<td>20%</td>
<td>80%</td>
</tr>
<tr>
<td>Disrupts IAC/cochlea</td>
<td>EAC/ossicles, TM</td>
</tr>
<tr>
<td>Frontal-occipital, severe</td>
<td>Temporal-parietal, milder</td>
</tr>
<tr>
<td>Spread – foramen magnum</td>
<td>Temporal squamous</td>
</tr>
<tr>
<td>Haemotympanum</td>
<td>CSF/bloody otorrhoea (50/50)</td>
</tr>
<tr>
<td>No Battle’s sign</td>
<td>Battle’s sign</td>
</tr>
<tr>
<td>No ossicle injury</td>
<td>Ossicles injured – severe fracture</td>
</tr>
<tr>
<td>30-50% facial nerve injury</td>
<td>10-25% facial nerve injury</td>
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</tbody>
</table>
Skull Fracture - Growing:

- Fracture line that widens with time
- Progressive displacement of brain through dural defect
- Usually asymptomatic, most often presents as scalp mass
- Very rare, usually required a widely separated fracture and dural defect
- Most < 1yr, 90% < 3yrs of age
- Rarely develops > 6 months post-injury
- X-ray – widening of fracture and scalloping of edges
- Treatment – surgery with dural closure
Skull Fracture - Divisions:

- Perforating fracture – small mass moving at high velocity (i.e. bullet)
- Penetrating fracture – results from moderately high velocity impact
- Depressed fracture – results from slow moving object of high kinetic energy
CSF Leak / Fistula:

- Possible routes of egress:
  - Cribriform plate
  - Sphenoid air cells
  - Petrous bone
  - Mastoid air cells
  - Frontal sinus
  - Traumatic wound
CSF Leak / Fistula (cont.):
Traumatic vs. Spontaneous:

- Traumatic: Spontaneous
- 2%: Rare ++
- Anybody: 2x females, 40’s
- > 70% stop one week: Uncommon to stop on own
- Unusual to be profuse: Unusual to be profuse
- Laterality: No laterality
- Air – 20%: Unusual
- Anosmia – 80%: Unusual
- HA uncommon / mild: Headache common / severe
CSF Fistula - Characteristics:

- Clear, does not cause excoriation within or around nose
- Check glucose with dextrostix
- Collect and test for glucose; >30mg%, nasal secretions <5mg%
- Beta2-transferrin, present in CSF but absent in tears, saliva, nasal exudates and serum; can be detected by electrophoresis
- Ring sign
- Salty taste
- Imaging
Epidural Haematoma:

- **Biomechanics:**
  - Secondary to contact injury
  - Dural vessels are torn as a fracture propagates across the vessel or if there is sufficient skull bending
  - Not related to head acceleration

- **Source:**
  - Middle meningeal artery 50%
  - Middle meningeal vein 33%
  - Diploic vein / dural sinus 17%
Epidural Haematoma (cont.):
Epidural Haematoma (cont.):

↑ MRI

(less clearly diagnostic)

⇒ CT

(more clearly diagnostic)
Epidural Haematoma - Presentation:

- Classical (<25%):
  - Talk, then die
  - Brief post-traumatic LOC
  - Followed by lucid interval for several hours
  - Then obtundation, contralateral hemiparesis, ipsilateral pupil dilation

- Signs and symptoms:
  - 60% have dilated pupil
  - No LOC in 60%
  - In paeds, may also present with significant drop in hct
Epidural Haematoma (cont.):
Subarachnoid Haemorrhage:

- Most common cause of SAH is traumatic
- Usually not in basal cisterns
Subarachnoid Haemorrhage (cont.):
Subarachnoid Haemorrhage (cont.):
Acute Subdural Haematoma:

- Biomechanics:

- 1. Complicated SDH:
  - SDH associated with parenchymal or cortical laceration
  - More likely secondary to contact injury
  - Most commonly associated with acute SDH
2. Uncomplicated SDH:
   - Caused by disruption of surface vessels usually bridging veins
   - Results from acceleration-deceleration injury
   - Head acceleration that produces short-duration, high strain rate loading most commonly seen in setting of falls where the head strikes a broad surface
   - Little energy is dissipated by focal injury
   - The deceleration causes tensile and shear strains at the brain-skull interface
   - Commonly associated with DAI because the mechanism of the two injuries is similar and they often coexist
Acute Subdural Haematoma (cont.):
Acute Subdural Haematoma (cont.):
Subacute and Chronic SDH:

- Subacute: 3 days to 3 weeks
- Chronic: present 3 weeks post-injury

Risk factors:
- Alcohol abuse
- Seizures
- CSF shunts
- Coagulopathies
- Neoplasms
Subacute and Chronic SDH (cont.):
Subacute and Chronic SDH (cont.):
Contusions:

- What is a contusion?

  - Bruise of the neural parenchyma
  - Most commonly involves the crown of the gyrus
  - Superficial foci of punctate / linear haemorrhage
  - Caused by extravasation of RBCs around small lacerated vessels within the parenchyma
  - The pia-arachnoid membrane is intact; if breached it is a laceration
  - Wedge-shaped with apex extending into the neural parenchyma
Contusions – Types:

1. Coup:
   - Occurs beneath the immediate area of impact

2. Contrecoup:
   - Found remote from and most often in a straight line with the impact site on the other side
Contusions – Types (cont.):
Contusions – Types (cont.):

3. Fracture:
   - Occurs at the site of fracture

4. Intermediate:
   - Occurs within the neural parenchyma
   - Most commonly occurs in the sylvian fissure where the temporal and frontal cortices are slapped against each other
Contusions – Types (cont.):
Contusions – Types (cont.):

5. Gliding:
   - Occurs at the vertex
   - Haemorrhagic lesion affecting the parasagittal white matter in the superior part of the cerebral hemispheres
   - Produced by rostral to caudal movement of the brain during deceleration injuries

6. Herniation:
   - Temporal lobe and cerebellar tonsils make contact against edge of tentorium and foramen magnum
Contusions – Location:

- Most commonly occurs in areas where sudden deceleration causes brain to impact on bony prominences
- Frontal poles
- Orbital surfaces of the frontal lobes
- Temporal poles
- Interior surfaces of temporal lobes
- Cortex above and below sylvian fissure
Contusions – Location (cont.):
Mechanism of Coup Contusion:

- Caused by strains that arise from skull in-bending
- Induced by brain striking an osseous ridge or dural fold
- Rupture of pial blood vessels occurs because of high tensile strains (suction) that are produced when the focal depressed elastic skull rapidly returns to its normal configuration
Mechanism of Contrecoup Contusion:

- Superficial focal areas of vascular disruption and cortical damage remote from the site of impact; occurs principally because of head motion
- Can result from either translational or angular movements of the head
- Predominant mechanism for contrecoup contusions is acceleration
- Often not exactly opposite the point of impact
Mechanism of Contrecoup Contusion (cont.):
Contusion – Evolution:

- Perivascular haemorrhage (hence the linear deposition of haemorrhage)
- Blood extends into adjacent cortex where neurons in affected region undergo necrosis by the presence of ischaemic cells changes
- Later proliferation of capillaries, astrocytes and microglia
- Dead tissue is removed and shrunken, gliotic fenestrated scar often containing residual haemosiderin-filled macrophages results
- Old contusion tends to be triangular in shape, its broad base being at the crest of a gyrus and apex in white matter
Contusion – Evolution (cont.):

Old Contusion:

Ventral View

Coronal section
Intracerebral Haematoma:

- Caused by deceleration injury
- Confluent haemorrhages
- Most commonly found in frontal and temporal lobes (80 – 90%)
- Commonly associated with contusions as well as acute SDH and SAH
- May have delayed onset
- Multiple in 20%, associated with SDH in 50%
Intracerebral Haematoma (cont.):
Diffuse Axonal Injury

- Caused by deceleration injury
- Confluent haemorrhages
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Diffuse Axonal Injury (cont.):
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- Grades:

1. Widespread axonal damage in CC and cerebral hemisphere white matter

2. Grade 1 and focal abnormalities in CC often associated with small haemorrhages called tissue tear haemorrhages

3. Grade 2 and tissue tear haemorrhages in rostral brain stem
Diffuse Axonal Injury (cont.):

- **Corpus Callosum:**
  - Generally inferior part and to one side
  - 3 – 5 mm across but AP distance several cms
  - Sometimes restricted to splenium, where it may be bilateral
Diffuse Axonal Injury (cont.):
Diffuse Axonal Injury (cont.):

- Mechanisms:
  - Primary injury of rotational acceleration / deceleration
  - Axons are injured by shearing and impaired transport and organelle accumulation
  - Diffuse axonal injury; axonal retraction balls, microglial scar
  - Degeneration of white matter fibre tracts
Almost all cases of DAI, especially if severe, arise from vehicular injury. Impact to dashboard, resilient windshields, energy-absorbing steering columns in which acceleration is long. Conversely, most SDH occur because of falls or assaults in which the impact duration is short and angular, and acceleration is abrupt. Prolonged rotational acceleration.
Diffuse Axonal Injury (cont.):

- Axonal damage:
  - Parasagittal white matter of cerebral hemispheres
  - CC
  - Medial lemniscus
  - CTT
  - CST
  - Fornix, IC, EC
Diffuse Axonal Injury (cont.):

A. Twisted Axon
B. Torn Axon
C. Broken Axon

A. Pulled away at synapse
Concussion

- Alteration in consciousness, not necessarily LOC
- In general, no gross / microscopic abnormality
- Minimal or no change CT / MR
- Increased metabolism
- Susceptible to secondary impact syndrome
Concussion (cont.):

A concussion is a violent jarring or shaking that results in a disturbance of brain function.
Non-Accidental Head Injury in Children

- Trauma is the most common cause of death in childhood and inflicted head injury is the most common cause of traumatic death in infancy.

- Most common age group for child abuse:
  - Shaken impact syndrome is largely restricted to children less than 3 yrs, with the majority occurring from 0 – 1 yrs.

- Main risk factors for non-accidental injuries in kids:
  - Young parents, unstable family situations, low SES, disability or prematurity of child.
Non-Accidental Head Injury in Children (cont.):
Non-Accidental Head Injury in Children (cont.):

- Common symptoms in children:
  - Lethargy, irritability, seizures, tone changes, vomiting, poor feeding, breathing abnormalities

- Main findings on physical examination:
  - Decreased LOC, opisthoclonus, full fontanelle, Seizures 40 – 70%, retinal haemorrhages 65 – 95%
Non-Accidental Head Injury in Children (cont.):

- Mechanism of injury / death in shaken-infant syndrome:
  - Sudden deceleration associated with striking head against surface; responsible for most if not all severe inflicted injuries
  - Rotational forces about centre of gravity – DAI and SDH
  - Translational forces less harmful, i.e. household falls; rotational or angular deceleration very uncommon
Non-Accidental Head Injury in Children (cont.):  

6 mos old boy with Shaken Impact Syndrome
Non-Accidental Head Injury in Children (cont.):

- It is the sudden angular deceleration experienced by the brain and cerebral vessels, not the specific contact forces applied to the surface of the head that results in the damage.

- This angular force is distinct from the forces generated in most cases of accidental trauma in infants.

- More accurate to call it “Shaken Impact Syndrome”.

- Whether shaking alone can cause the constellation of findings associated with the syndrome is still debated.