Head Injury (PEDIATRIC)

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EPIDEMIOLOGY

20% of all TBIs occur in pediatric age group (birth ≥ 17 yrs).
Children have proportionately more head injuries than adults!

• males : females = 2 : 1

INCIDENCE

- peaks
  1) children < 2 yrs - falls and child abuse.
  2) children 5-7 yrs (often in early afternoon when school is closing) - falls and transportation-related crashes.
  3) children < 15 years (mainly in males) - sports and driving.

MORTALITY

Trauma is leading cause of death in children > 1 year.

• head trauma represents 75-97% of pediatric trauma deaths.
• mortality - 29%.

ETOLOGY

1. Motor vehicle accidents (27-37);
   - children aged 5-9 years – pedestrians
   - children aged 9-15 years – bicyclist
   young adults aged 15-19 years – passengers and drivers.
2. Falls (24%), esp. children < 4 yrs.
3. Child abuse (24-85%) patients are < 2 years, i.e. nonambulatory children.
4. Recreational activities (21%) have seasonal distribution (peak during spring-summer).
5. Assault (10%)
6. Firearm (2%).
7. Birth trauma (less common now than in past).

PATHOPHYSIOLOGY

More diffuse injuries, less focal injuries*

*emergency butt holes are generally ineffective; only 20% pediatric severe TBI cases are amenable to neurosurgery

• pediatric head is larger (in proportion to body surface area), stability is dependent on ligamentous (rather than bony) structures, poor control of neck muscles, brain has higher water content* (≈88% vs. 77% in adult) - pediatric brain is more prone to acceleration-deceleration injury (i.e. diffuse axonal injury) is more frequent, but intracranial hematomas are less frequent.
• water content is inversely related to myelination process (unmyelinated brain is more susceptible to shear injuries).
• skull is thin and sutures not fused - brain is susceptible to deformational forces.
• skull base is smoother - lower incidence of contrecoup injuries.
• infants tolerate ICP increases better - because of open sutures.
• infants often develop vasodilatation* in minutes - hours following mildly head injury → ICP↑ → rapid neurologic deterioration (minims enlarging intracranial mass); good prognosis with control of intracranial hypertension.
• CT cannot differentiate this type of swelling from that caused by diffuse axonal injury

CLINICAL FEATURES

• infants can lose significant blood amount into cranial cavity (from intracranial bleeding or skull fracture) blood can seep through fracture and produce large galeal or subperiosteal hematoma
• despite apparently trivial trauma, children may appear pale, lethargic, and have emesis (it is not a sign of ICP), headaches, dizziness.
• difficult to obtain accurate neurologic examination.
• early posttraumatic seizures are more common.

DIAGNOSIS

Special indications for skull X-ray in pediatric patients → see p. TIH 1


Level III recommendations: in the absence of neurologic deterioration or increasing ICP, obtaining a routine repeat CT ≥ 24 hrs after the admission and initial follow-up study may not be indicated for decisions about neurosurgical intervention.

• lifetime risk of fatal cancer resulting from one head CT in a 1-yr-old child is as high as 1 in 1500.
• children with severe TBI are medically unstable and (if portable CT is not available) may further deteriorate during transport to the CT scanner (hemodynamic instability, increased ICP, oxygen desaturation).
TREATMENT

- infants and children often seek increased amounts of ADHD after head injury - mild fluid restriction is appropriate (provided there is no hypotension).

**ICP MONITORING & TREATMENT**


Level III recommendations:
- ICP monitoring may be considered.
- treatment of ICP > 20 mmHg* may be considered.
- CSF drainage through EVD may be considered for increased ICP
- Initial brain in conjunction with a functioning EVD may be considered for refractory intracranial hypertension but only with open basal cisterns, and no evidence of a mass lesion or shift on imaging.

*exp. for > 3 mass; optimal ICP treatment threshold may be physiologically age-dependent (e.g. lower ICP therapeutic target for infants and young children than older children or adults)

- intracranial hypertension may be present in children with open fontanelles and sutures - ICP monitoring is of significant use in these patient populations.
- Use of evidence support the use of ICP monitoring in children with severe TBI:
  1. high incidence of intracranial hypertension.
  2. association of intracranial hypertension and poor neurologic outcome.
  3. concordance of protocol-based intracranial hypertension therapy and best-reported clinical outcomes.
  4. improved outcomes associated with successful ICP lowering therapies.

**CEREBRAL PERFUSION PRESSURE**


Level III recommendations:
- minimum CPP 40 mmHg may be considered in children. CPP threshold 40-50 mmHg may be considered; there may be age-specific thresholds with infants at the lower end and adolescents at the upper end of this range.
- if brain oxygenation monitoring is used, maintenance of partial pressure of brain tissue oxygen (PbtO2) > 30 mmHg may be considered.

**HYPOTHERMIA & THERAPY**


Level II recommendations:
- HYPERTONIC SALINE 3% 6.5-10 mL/kg should be considered for severe pediatric TBI with intracranial hypertension;
- HYPEROSMOLAR 3% are 0.1-1 mL/kg/hour on a sliding scale; the minimum dose needed to maintain ICP < 20 mmHg should be used; maintain serum osmolality < 360 mosmol/L.

Footnote: although MANNITOL is commonly used, no studies meeting inclusion criteria were identified for use as evidence.
- euvalolul rather than dehydration is the general therapeutic target.

**HYPERTENSION**


Level III recommendations:
- avoidance of prophylactic severe hyperventilation (PaCO2 < 30 mmHg) may be considered in the initial 48 hrs after injury.
- if hyperventilation is used for refractory intracranial hypertension, advanced neuromonitoring for evaluation of cerebral ischemia may be considered.

**ANAESTHESIA, SEDATIVES, AND NEUROMUSCULAR BLOCKADE**


Level II recommendations:
- STETOMARATE may be considered to control severe intracranial hypertension; however, the risks of adrenal suppression must be considered.
- THETOPHAR may be considered to control intracranial hypertension.
- high-dose barbiturates may be administered in hemodynamically stable patients with refractory intracranial hypertension despite maximal medical and surgical management (continuous ICP monitoring and cardiovascular support to maintain adequate CPP are required).

Footnotes below recommendations: In the absence of outcome data, the specific indications, choice and dosing of anesthetics, sedatives, and neuromuscular-blocking agents should be left to the treating physician. As stated by FDA, continued infusion of thiotrop, is not recommended.

**HYPOThERMIA**

- not recommended no benefit, plus, may increase the risk of mortality and arrhythmia.

Meta-analysis
Zhong B ("Meta-Analysis of the Efficacy and Safety of Therapeutic Hypothermia in Children with Acute Traumatic Brain Injury", World Neurosurg. 2015 Apr;83(4):577-583)
- therapeutic hypothermia could increase mortality compared with the normothermia (RR = 1.84, P = 0.01).
- GOS scores did not differ between the hypothermia and normothermia groups.
- hypothermia did not increase the rate of pneumonia (RR = 0.84, 95% CI = 0.63-1.2, P = 0.23) or bleeding (RR = 0.94, 95% CI = 0.39-2.26, P = 0.89), but incidence of arrythmias was higher (RR = 2.60, 95% CI = 1.06-6.41, P = 0.04).

Hypothermia Paediatric Head Injury Trial Investigators + the Canadian Critical Care Trials Group

- class 1 evidence, 17 centers in 3 countries.
- 225 pediatric patients (1-7 yr) with severe TBI.
- hypothermia group was cooled to 32.5 °C within 6 h of injury for 24 h.
- degree of hypothermia in the hypothermia group during the re-warming period was significantly greater.
- significantly less interventions were required to control ICP in the hypothermia group within the first 24 h.
• hypothermia is not associated with improved neurological outcome and may increase mortality:

<table>
<thead>
<tr>
<th>Hypothermia</th>
<th>Normothermia</th>
<th>Statistical significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unfavourable outcome at 6 months</td>
<td>31%</td>
<td>22%</td>
</tr>
<tr>
<td>Overall mortality</td>
<td>21%</td>
<td>12%</td>
</tr>
</tbody>
</table>

• study leaves questions about potential benefit of earlier or a more prolonged hypothermia in certain subgroups of patients.


**Level II recommendations:**
- moderate hypothermia (32–33°C) beginning early after severe TBI for only 24 hrs duration should be avoided vs. beginning within 8 hrs after severe TBI for up to 48 hrs duration should be considered to reduce intracranial hypertension.
- if hypothermia is induced for any indication, rewarming < 0.5°C per hour should be avoided*.
  *to avoid rebound intracranial hypertension

**Decompressive Craniectomy**

- may have a beneficial effect (in concert with RESCUET trial but vs. DECRA trial in adults)
- class II evidence.
- 27 pediatric patients (1–16 yrs) with severe TBI and functioning EVD.
- uncontrolled ICP (20–24 mmHg for > 30 min, 25–29 mmHg for > 10 min, > 30 mmHg for > 1 min) → randomization for bitemporal craniectomy* (removal of 3–4 cm discs of bone bi-temporally with no attempt to open the dura).
- craniectomy performed rather early (median 19.2 hrs after injury)
- results are positive but did not reach statistical significance:

<table>
<thead>
<tr>
<th>Decompressive craniectomy group</th>
<th>Conventional medical management group</th>
<th>Statistical significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Favouirable outcome at 6 months</td>
<td>54%</td>
<td>14%</td>
</tr>
<tr>
<td>Mean reduction in ICP post-randomization</td>
<td>8.98 mmHg</td>
<td>3.69 mmHg</td>
</tr>
</tbody>
</table>

Due to the changes in management protocol over the study period, statistical analysis was performed on two separate occasions. Owing to the repeated analysis the p value for statistical significance is reduced further to \( p < 0.0221 \).


**Level III recommendations:**
- decompressive craniectomy with large duraplasty may be considered for intracranial hypertension / herniation / neurologic deterioration refractory to medical management during the early stages of treatment.

**Glucose and Nutrition**


**Level II recommendations:**
- the evidence does not support the use of an immune-modulating diet to improve outcome.

**Antibiotic Prophylaxis**


**Level III recommendations:**
- prophylactic antibiotics may be considered to reduce the incidence of early post-traumatic seizures.
- incidence of early post-traumatic seizures (within 7 days post injury) in pediatric patients with TBI is 10%.

**Corticotrophin**


**Level III recommendations:**
- corticosteroids are not recommended to improve outcome or reduce ICP.

**Prognosis**

Much lower morbidity and mortality (than in adults) – children demand aggressive approach!
- pediatric brain is more "plastic" - if child recovers from coma within 14 days, likelihood of (near-) normal cognitive and neuromotor function is extremely favorable.
- recovery in children takes longer (vs. adults - reach maximum recovery by about 6 months).
- Prolonged rehabilitation, particularly in cognitive and emotional areas, is often required!

N.B. infants < 2 yrs with severe TBI have uniformly poor prognosis:
1) immature autoregulation
2) incompletely myelinated brain (early head injuries impair ability for new learning → mental retardation)
3) open cranial sutures permit greater distortion among meninges, cerebral vessels, and underlying brain.

**Special Situations**

--- "SHAKEN BABY" SYNDROME
- infant is held by shoulders (or grabbed around chest by 2 hands) and violently shaken → typical acceleration-deceleration injury.
Neurologic damage + minimal external signs of trauma + no explanatory severe trauma?

1. SAH
2. Subdural hematomas, esp. posterior interhemispheric & tentorial (repeated abuse → hematomas of different ages – seen as heterogeneous subdural accumulations).
3. Cerebral contusions and shearing injuries (diffuse cerebral swelling with absent differentiation between white and grey matter - may be refractory to medical management; after 2-3 weeks significant atrophy, multicystic encephalomalacia develops → severe mental / motor disability → severe microcephaly).
4. Retinal hemorrhages (bilateral) in absence of coagulopathy - most specific sign of shaken baby syndrome!!; intraretinal, preretinal, or vitreal; fundoscopy should be performed quickly in any child with suspected child abuse before retinal hemorrhages disappear (flame-shaped hemorrhage disappears within few days; round intraretinal hemorrhage may last 2 weeks); retinal hemorrhages also may occur from childbirth and persist for up to 4 wk.!!

- falls from < 3 feet are insufficient to explain such injuries?
- skull fractures cannot occur with shaking!
- recent studies have shown that simple act of shaking is insufficient to cause subdural hematoma (these children probably sustain deceleration injury when they are slammed onto surface, even padded surface - SHAKING-IMPACT SYNDROME).
- skeletal fractures (metaphyseal, posterior rib) occur while child is shaken violently in to-and-fro fashion.

• presents as infant (usually < 1 yr old) with seizures, lethargy, apneic spell or becoming suddenly unresponsive;
• history is often confusing and inconsistent; often frequent visits to doctors because of irritability, feeding problems, vomiting or symptoms of encephalopathy;
• careful systemic survey for evidence of other injuries (e.g. skin bruising); injuries caused by choking, squeezing, and throwing are often associated.
• perform X-ray: see p. Ped3 > skull - look for unexplained skull fractures: multiple (involving > 1 bone), bilateral, non-parietal, old (e.g. leptomeningeal cysts).
• N.B. accidental falls cause single, narrow, linear fractures, most commonly in parietal bone!!

for children < 2 years, skeletal survey is recommended (look for old fractures); isotopic bone scans may be useful.
• victim child is often brought to medical attention in delayed fashion = 25% mortality!
• refer to proper child welfare agency.

<table>
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<tr>
<th>Subdural Hematoma in Shake-impact Syndrome</th>
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<tr>
<td>Violent shaking or sudden impact may cause excessive brain movement and damage bridging cortical veins</td>
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<tr>
<td>Cerebral veins are at high risk of rupture when they traverse subdural space</td>
</tr>
<tr>
<td>Shearing forces tear bridging cortical veins</td>
</tr>
<tr>
<td>Subdural Hematoma: expanding, chronic, and sometimes hemorrhagic</td>
</tr>
<tr>
<td>Hemorrhage into subdural space</td>
</tr>
</tbody>
</table>

Forces generated by violent shaking can produce stretching of axons; strong force may shear off axons, ends of which retract into globoid shapes (“retraction balls”):
Retinal hemorrhages.

Axial noncontrast CT - poor differentiation between white and grey matter in large areas of brain with intervening normal areas; acute interhemispheric subdural hematoma:

Axial noncontrast CT - large amount of acute subdural hemorrhage, both between hemispheres and over convexities; diminished density and loss of grey-white differentiation in left hemisphere is because of associated hypoxic-ischemic injury.

BIBLIOGRAPHY for ch. “Head Trauma” ➔ follow this LINK ➔