Pediatric Head Trauma: Jack Fell Down and Broke His Crown . . .

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Anatomy

- Child's brain more susceptible than adult brain to certain types of injuries following TBI
- Pediatric head is larger in proportion to body surface area
- Stability depends on ligamentous rather than bony structure
- Higher water content (88% peds/77% adults) makes brain softer and more prone to acceleration-deceleration injury
- Water content inversely related to myelination process
- Unmyelineated brain more susceptible to shear injuries
- Infants and young children tolerate ICP increases better because of open sutures

Level of Consciousness

- Worsening or improving? **Looks Bad – Looks Good**
- Most important indicator of neurological status
- Neuro intact = awake, alert, responsive to surroundings
- LOC varies with developmental age
- Infants – should respond to feeding and measures to console them
- Toddlers and older children – recognize and respond to parents
- Older children and adolescents – follow commands
- All – withdraw from painful stimulus
- Use central stimulus
- Peripheral stimulus elicits spinal reflex

Glasgow Coma Score

- Modified GCS for infants and children
- Measures severity of head injury and child's level of responsiveness
- Scale 3-15 (4 eyes, 5 voice, 6 motor)
- Change of 2 or more points is significant and should be reported immediately
- GCS 14-15 = mild head trauma
- GCS 9-13 = Moderate head trauma
- GCS $\leq$ 8 = severe head trauma
- GCS of 8 or less is considered an operational definition of coma

Overview

- Trauma is leading cause of death in children > 1 year of age in U.S.
- Head Trauma = 80% or more of injuries
- 1 - 2% of all ED presentations in children (473,947 visits to ED annually by children 0-14 years of age)
- Mild - GCS >12 = 82%

- Moderate to severe - GCS < 12 = 13%
- 5% die (7,000 deaths per year)
- 4x mortality from leukemia and 18x mortality from brain tumors
- High emotional, psychosocial and economic impact
- Usually due to MVAs, falls, assaults, recreational activities, child abuse (varies according to age, group, sex)
- 0-4 yrs and 15-19 years most likely to sustain TBI
- 5-10% d/c to long-term care facility
- Costs in U.S. = $76.5 billion in 2000

Pathophysiology

Primary Injuries

- Occur at time of impact either by direct injury to brain or to long white-matter tracts through acceleration-deceleration forces which leads to axonal disruption and 2ndary cell death
- Direct injury to brain parenchyma

Secondary Injuries

- Occur in response to primary injury and contribute to neuronal damage and cell death
- Hypotension, hypoxia, hypercapnia, hyponatremia
- Inflammatory changes
- Leads to microcirculation disruption and neuronal disintegration
- Cerebral edema, increased ICP, hyperemia and ischemia

The brain is dependent on a constant CBF to provide oxygen and glucose.

Skull Fractures

- Pediatric skull is protective box
- Fractures occur when the skull cannot stand the force of impact
- Pediatric skull is thinner and more flexible than adult skull
- 20% of children presenting with head trauma will have skull fracture
- Key issue is identifying underlying pathology

Linear nondepressed skull fracture

- Most common in children
- Usually in parietal bone (thinnest cranial bone and most frequent site of impact in falls)
- Frontal and occipital bones are thickest and require a more severe impact to cause fracture and therefore associated with higher degree of brain injury
- Ping-pong skull fractures occur in newborns and young infants due to the thin, pliable skull
- Open, depressed, or comminuted skull fractures may require surgical intervention and more frequently associated with underlying hemorrhage or brain injury

Basilar skull fracture

- Occur in the anterior, middle or posterior fossa at the base of the skull
- Temporal bone fractures can cause sensorineural hearing loss (CNVIII) and facial nerve dysfunction (CNVII), hemotympanum, torn TM, CSF leak and conductive hearing loss

Nursing assessment

- Gentle palpation to assess for stepoffs, depressions, hematoma, lacerations (wear gloves)
- Basilar skull fractures: battle sign (postauricular ecchymosis) and can be associated with CSF otorrhea
- Frontal basilar skull fracture: Racoon eyes (periorbital ecchymosis) and sometimes CSF rhinorrhea
- For CSF leaks: elevate HOB, restrict nose blowing, report fever or signs of meningitis
- NO NG TUBE (frontal fx through cribiform plate may allow tube to go into brain)
- May need lumbar drain

Monroe-Kellie Doctrine - Small increases in the volume of one can be offset by an equal, compensatory decrease in another

80% brain
10% blood
10% CSF

Signs and Symptoms of ↑ICP

- H/A, vomiting
- ↓LOC - restless, agitation, more stimulation required, confusion, less able to follow commands
- Posturing – decerebrate, decorticate
- Cushings - ↑BP, widening pulse pressure, bradycardia
- Parinaud's (loss of upward gaze)
- Infants have full fontanel, prominent scalp veins, increasing head circumference, diastatic sutures

Motor Changes

- Asymmetrical weakness
- Decorticate
- Decerebrate

Vital Sign Changes

- Tachycardia
- Increased systolic BP and widening pulse pressure
- Irregular respirations

Cushings Response (late sign)

- Ischemic response
- Pressure on medullary center

- ↑BP, ↓HR, irreg. resp.

ICP (increased intracranial pressure)

- Increase in CSF volume
- Increase in brain tissue volume
- Pathological space-occupying lesion
- Increase in blood volume

Increase in CSF Volume

- Tumor
- Congenital abnormalities (aqueductal stenosis)
- Herniation

Increase in Brain Tissue Volume (edema)

Pathological Space-Occupying Lesion

- Tumor
- Abscess
- Hemorrhage
- Hematoma
- Pneumocephalus
- Foreign body

Extraparenchymal Hemorrhage – bleeding outside of the brain

- Lower incidence of mass hemorrhage in infants and children than adults
- This is due to anatomy of pediatric skull
- Thin, deformable skull, wider CSF spaces, softer brain
- True up to 4 years of age – then skull is closed, rigid box
- However, children predisposed to shearing injuries and subarachnoid hemorrhage

Epidural Hemorrhage

- Less common in infancy but increases with age
- Outcomes better in children
- Can be venous or arterial in origin
- Venous in neonates and infants due to tearing of the dural veins
- More commonly, especially in older children, EDH results from tear of middle meningeal artery
- Bleeds into space between dura and periosteum
- Usually in frontal, temporal and parietal regions

Presentation: Symptoms can be delayed due to plasticity of child's skull

- Lucid interal in child but older child or adult may have loss of consciousness
- Rapid expansion not well-tolerated

Nursing assessment: Focal neurologic exam, Increasing drowsiness

Radiodiagnostics: CT shows extraparenchymal fluid, maybe a midline shift

Subdural Hemorrhage

- More common in infants and less common in older children (opposite of EDH)
- More often underlying brain injury than with EDH
- Causes: birth trauma, accidental falls, child abuse
- Due to stretching and tearing of bridging veins in subdural space
- Not limited to suture lines and can result in large bilateral blood collection over entire brain
- **Presentation**: Symptoms include seizures, irritability, lethargy, vomiting, increase head circumference
    - Older children have decreased LOC, pupil asymmetry and hemiparesis
    - Require emergent crani to prevent herniation and death
    - Small subdurals in child with minimal neuro deficits can be observed closely with follow-up imaging
    - Needle aspirations in infants with open fontanel
    - Acute vs chronic

Subarachnoid Hemorrhage

- Subarachnoid space located between arachnoid and pia mater
- CSF circulates in subarachnoid space

Presentation: irritability, H/A, stiff neck
Can develop post-traumatic hydrocephalus
May need ventric or shunt

Parenchymal Hemorrhage

- Children have generalized brain swelling after severe head trauma
- Cerebrovascular resistance decreases significantly which results in vasodilation and cerebral edema
- CT can look normal soon after injury but bad 24 hours later
- Mass effect can be compartmentalized or globalized
- Mass effect confined to temporal (middle) cranial fossa can lead to transtentorial or uncal herniation
- Nurse needs to prevent 2ndary injury due to hyponatremia, hypercapnea, hypotension and present life-threatening edema or herniation
- Monitor LOC and report any changes

Contusion

- Focal bruise to the surface of the brain
- Occurs when skull impacts stationary object with sudden deceleration, causing brain to collide with bony prominence of frontal, temporal or occipital skull
- Coup – point of initial impact of brain on internal skull
- Contrecoup (counter blow) – brain, suspended in CSF, strikes the opposite side of the skull
- Contrecoup injuries less likely in children
- Follow up imaging can show “dropout” areas in brain (encephalomalacia)
- If contusion occurs in eloquent area, child may have speech or motor deficits

Posttraumatic Seizures

- More common in young children than in adults
- Occur in 10% of young children following head trauma, usually in 1st 24 hrs
- Seizure meds? – controversial – not a standard of care but is an option
- Usually given for 7 days post head trauma
- Seizures can result in increased brain metabolism and increased ICP, causing 2ndary brain damage – avoid

Diffuse Axonal Injury

- Decreased LOC and generalized increased ICP more likely with DAI
- Occurs when pediatric skull is subjected to rotational forces during an acceleration or deceleration injury
- Rare in infants
- Neuronal injury occurs when axons are stretched until they fracture

- DAI typically occurs at the gray-white matter junctions.

  Presentation: Immediate loss of consciousness that often lasts more than 6 hrs
  - Abnormal flexion (decorticate) posturing or extension (decerebrate) posturing
  - Variations in GCS on serial assessments
  - Pupillary and other cranial nerve dysfunction and brainstem abnormalities

  Radiodiagnostics: MRI most sensitive to shearing injury
  - No treatment for DAI
  - Treat ICP when GCS is 3-8
  - Recovery is slow and gradual process and can continue for weeks or months

  1. Teeth grinding, chewing motions, eyes open but not responsive with persistent posturing
  2. Periods of agitation with arms flailing
  3. Return of ability to localize painful stimulus
  4. Ability to follow commands, which represents the end of coma

Penetrating Craniocerebral Injury

- Less common in children but often fatal
- 12% of all pediatric TBI deaths
- Causes: accidental falls, impalement with sharp objects, accidental and nonaccidental gunshot wounds and suicide
- High velocity penetrating skull injury associated with skull fractures
- Low-velocity penetrating skull injury associated with increased (43%) of infection or brain abscess within path of projectile
- Treat with broad-spectrum prophylactic antibiotics
- Protruding object left in place until surgical removal
- Hemorrhage, cerebral edema, severe increased ICP and herniation can occur
- Goals of surgery: remove object without causing more damage, debridement of necrotic brain tissue, evacuation of blood and placement of ICP monitor
- Seizure prophylaxis
- MRI contraindicated if metal fragment

Increase in Blood Volume

Increase in arterial blood flow

- Hypercapnea
- Hypoxia
- Hypotension
- Hyperthermia
- Seizures

Decrease in venous return

- Constriction of jugular veins during valsalva
- Extreme turning of head blocking jugular
- Increased intrathoracic pressure
- Trendelenburg
- Choking
- Trach ties too tight

Why is this a problem?

Key Concepts of Cerebral Perfusion

- Intracranial volume
- Monroe-Kellie Doctrine
- ICP
- CPP
- CO_2, hypoxia, hypotension

Monroe-Kellie Doctrine - Small increases in the volume of one can be offset by an equal, compensatory decrease in another

80% brain
10% blood
10% CSF

When the compensatory capacity is exhausted, any further increase in intracranial volume can lead to increased ICP (pressure-volume curve)

Volume-Pressure Relationship: regulatory mechanisms

- Brain has some capacity to compensate for additional mass from tumor, blood, hydrocephalus, etc.
- Shifting of intracranial CSF to the spinal subarachnoid space
- ↓CSF production
- Venous compression
- Vasoconstriction to limit blood volume
- Shunting of blood

Cerebral Perfusion Pressure

- CPP = MAP-ICP
- MAP = force trying to drive blood (and the O_2 it transports) to the brain
- ICP = force that resists (or regulates) how much blood gets through
- CPP = Net pressure of flow into brain or BP gradient across brain (normal adult CPP = 70-100mmHg)
- If CBF is ↓d below the lower threshold of autoregulation (40-50 mmHg), CBF proportionately ↓s as the autoregulatory capacity is exhausted
- CBF below 18-20 ml/100g/min causes cerebral ischemia

Several cerebral regulatory mechanisms influence ICP

- Autoregulation allows constant blood flow in brain at MAP of 60-150 mmHg
- Normally, with ↑ BP and autoregulation is intact, vessel diameter ↓s and reduces CBV and ICP.
- MAP = 60 then cerebrovasculature maximally dilates
- MAP = 150 then cerebrovasculature maximally constricts
- With impaired autoregulation, (as with TBI), ↑ BP passively distends these cerebral arterioles causing ↑ CBV and ↑ICP. This is transmitted to the capillary bed and vasogenic edema occurs.

Mechanisms that affect autoregulation of CBF

- Metabolic products - fever, seizure ↑ metabolic activity and leads to ↑ in CBF
- Arterial blood gas content - partial pressure of O_2 and CO_2 alters CBF
  - Hypoxia causes vasodilation with ↑ in CBF
- Hypercarbia (↑CO_2) ↑s CBF up to 350% of normal
- Arterial C_{O2} reactivity (alters vascular tone - hyperventilation decreases CBV and ICP but may increase ischemia)
- Blood pressure

Compliance and Compensatory Mechanism Exhaustion

- If brain compliance is low, then the patient is fast approaching the point where maximum displacement of CSF or blood has occurred and ICP is about to increase precipitously.
- Commonly known as “tight brain” and patient at risk for herniation

Raised ICP generates pressure gradients in brain that result in herniation

- Subfalcine
- Transtentorial (central and uncal)
- Tonsillar (foramen magnum)

Management of Raised ICP

Avoid **H**ydrocortisone (steroids)
Avoid **H**yperthermia (fever)
Avoid **H**ypotension (shock)
Avoid **H**ypoxia
Avoid **H**yperventilation

Guidelines for the Acute Medical Management of Severe Traumatic Brain Injury in Infants, Children, and Adolescents - Pediatric Critical Care Medicine, January 2012

ICP

- Sustained elevations of ICP >20 mmHg are associated with poor outcome
- Possibly decreased for age
- ICP monitoring is beneficial in severe TBI
- Also in infants with open fontanels

CPP

- Age-related differences
- ICP is zeroed to tragus (foramen of Monroe)
- MAP zeroed to R atrium with HOB ↑30°

Advanced Neuro Monitoring

- Limited studies in children when used to guide therapies
- Adult guidelines support jugular venous saturation and $PbtO_2$ and ICP monitoring
- Jugular venous sats <50% are associated with poor outcomes
- PbtO2 - Measures intracranial 0_2 indicating perfusion status of cerebral tissue local to probe - Probe placed in parenchyma close to injury - Measures oxygenation, brain temp, ICP
- $PbtO_2$ < 15 mmHg for >30 min are associated with high rates of mortality
- Further investigation: cerebral microdialysis, thermal diffusion probes, transcranial dopplers, near-infrared spectroscopy

Hyperosmolar therapy

- 3% hypertonic saline
- Mannitol - not enough evidence to support or refute as there has been long-term clinical acceptance
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Temperature

- Cooling? No fevers - Keep at 35-36 degrees

Hi Dose Barbiturates

- Can ↓ICP but causes hypotension and cardiac side effects
- Alters vascular tone
- Suppresses metabolism
- EEG to measure burst -suppression

Decompressive Craniectomy
No evidence either way

Hyperventilation

- Avoid $PaCO_2$ <30 mmHg in initial 48 hrs after injury

- If hyperventilation is used, consider advanced neuromonitoring to monitor ischemia
- Keep CO_2 between 35-40 mmHg

Steroids
- No

Sedation
- Leave to treating physician
- No propofol (lethal metabolic acidosis)

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