



Pediatric Head Trauma August 2016

EDUCATION COMMITTEE

PEER EDUCATION

Quick Review of Pathophysiology of TBI

Nuggets of knowledge to keep in mind with TBI

Intracranial compartment is comprised of 3 contents:

1. Brain parenchyma- 80%
2. Cerebral spinal fluid- 10%
3. Blood- 10%

Intracranial pressure (ICP) is a function of the volume and compliance of each component. Because the vault of the cranium cannot change (the skull), any increase in one of the 3 components will result in an increase in ICP. (2) Self-explanatory, right? ☺

So what is a normal value for ICP? Literature has defined ICP <15 mm Hg as normal [4]

In the end, our primary goal with TBI is to maintain perfusion and oxygenation at the cellular level. How do we know if we are doing that?

Cerebral perfusion pressure (CPP) is defined as a value to decipher if cerebral perfusion is adequate, where $CPP = \text{Mean Arterial Pressure (MAP)} - \text{ICP}$. In pediatrics, ideal CPP values vary due to varying adequate levels of MAPs depending on age. As a general rule of thumb, ideal $MAP = 1.5 \times \text{age} + 55$ mmHg in pediatrics. Neonates are often defined as gestational age = ideal MAP levels. (2) When the CPP falls below the normal level, the brain tissue

perfusion is compromised and secondary injury ensues.

Traumatic Brain Injury (TBI) is classified into three types of severity and are based on GCS scores.

Mild- GCS of 13-15, Moderate- GCS of 9-12, and Severe- GCS of < 9. (1)

In addition to severity classification, TBI is divided into two type of insults: Primary injury and secondary injury.

Primary injury- The direct injury to brain parenchyma. **Factors of which we have no control. The damage has already been done.**

Secondary injury- Cascade of biochemical, cellular and metabolic response, external insults such as hypoxia and hypotension. **As a transport team, we are responsible for the minimization of this type of injury.** [1]

-Biochemical release refers to the release of neurotransmitters such as acetylcholine, glutamate, and aspartame causes additional neuronal damage. [1]

-Diffuse cerebral swelling usually peaks 24-72 hrs post injury. Be aware that swelling may only be the tip of the iceberg if this incident has recently occurred. [1]



Remember:
CPP=MAP- ICP



Remember:

Ideal $MAP = 1.5 \times \text{age} + 55$ in pediatrics

Neonate ideal $MAP = \text{adjusted gestational age}$

Types of TBI

Diffuse Brain Injury- Most common type of severe traumatic brain injury in children. Usually produced by acceleration and deceleration forces. The mildest form of DBI is a concussion. [1]

Diffuse Axonal Injury (DAI) is a more severe form of diffuse brain injury. DAI results as a result of the shearing at the interface of grey and white matter

Epidural hematoma- usually arterial in nature, but can also be venous. Rapid expanding if arterial. Classic symptoms include brief loss of consciousness followed by period of lucidity. If not treated, may rapidly progress to secondary loss of consciousness and death. Classical finding on CT is "contact-lens" hematoma (perfect contact shape). Commonly seen with skull fractures.

Subdural hematoma- caused by bleeding of bridging veins lining the dura and brain matter. Typically more slow growing and may tamponade off due to being venous in nature. Watch for early signs of neuro deterioration for likely worsening of the size of the hematoma. [1]

Subarachnoid hemorrhage- rupture of a blood vessel outside of the brain filling the subarachnoid space with blood (space filled with circulating CSF). Presence of blood in this space is irritating

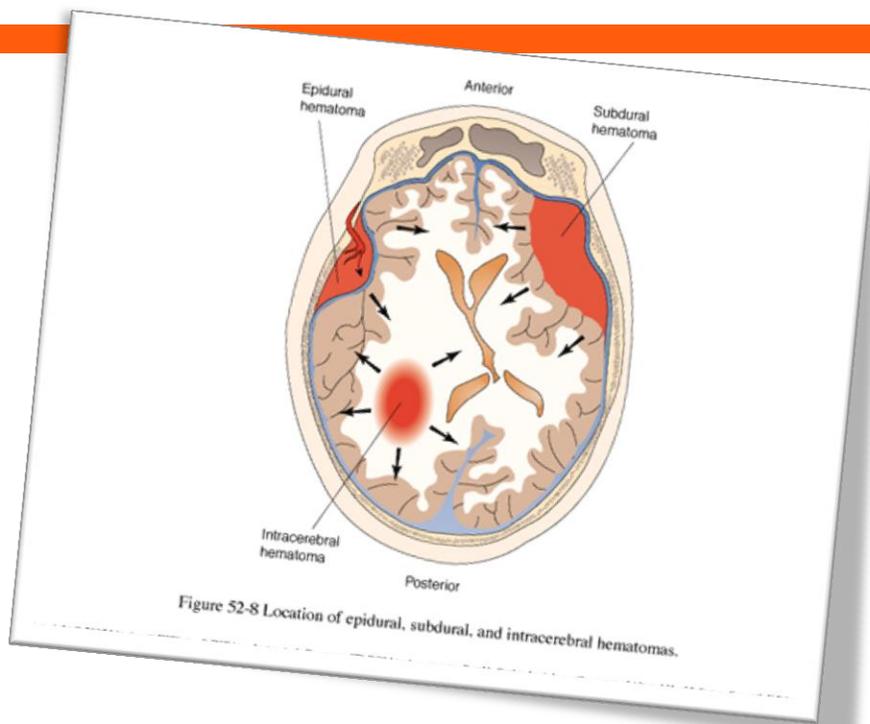


Figure 52-8 Location of epidural, subdural, and intracerebral hematomas.

and causes damage to brain tissue. Most commonly referred to as "the worst headache of the patient's life". Vasospasm is a common complication seen at 5-10 days post SAH and is treated by adequate BP and fluid status.

Intracranial hemorrhage- bleeding within the brain tissue itself. May be caused by fragile blood vessels (stroke) or trauma. [1]

PEDIATRIC TAKE-AWAY

Epidural hematoma, subdural hematoma and subarachnoid hemorrhage usually occur as a result of blunt trauma. SDH and SAH usually occur as a result of severe trauma and are frequently associated with other intracranial injuries. [4]



Different Types of Cerebral Edema

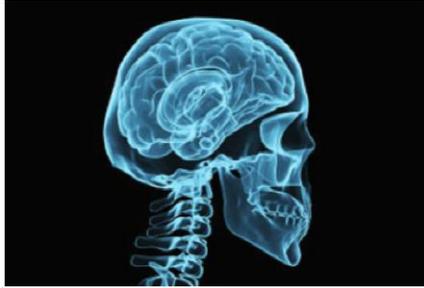
Cerebral edema can be a result of 3 types of swelling:

Cytotoxic swelling- Defined as intracellular swelling due to direct cell injury. This type of swelling occurs due to TBI, DAI and hypoxic ischemic injury. There is direct injury to the neurons and they are irreversibly injured.

Vasogenic edema- Defined as an increased permeability of the capillary cells, which allows fluid to escape intravascularly and into the extracellular space. In this type of swelling, the neurons are NOT directly injured. Examples include swelling resulting from tumors, intracranial hematomas and infarcts. [4]

Interstitial edema- Defined as increased fluid in the white matter. Commonly associated with increased cerebral spinal fluid (CSF) as related to hydrocephalus. This form of edema is easily manipulated with the removal of CSF (i.e. ventric drains) [4]

HEADING 4



To CT or MRI... That is the question

MRI is more sensitive in showing small areas of contusion, axonal injury and petechial hemorrhage. MRI was successful in finding abnormalities in 30% of cases with normal CT results. [2]



When in doubt, sit them out!

In young athletes and those with concussions, initial management is aimed at immediate removal from competition to avoid additional head injury. [3]

FAST FACTS

20%

Percent of high school football players who sustain brain injuries or concussions each season

1.74 million

Number of people who sustain TBI in United States each year.

FULL PHYSICAL REST

In addition to removal from organized activity after concussion, discussion needs to take place to educate on avoidance of recreational activities that may result in second head injury (cycling, skateboarding, climbing). [3]



Concussions and Secondary Injury

Concussion is defined as a mild TBI which is a trauma-induced alteration in mental status that may or may not involve loss of consciousness. The symptoms reflect a functional disturbance (brief confusion) rather than a structural injury (acute hemorrhage). Symptoms typically resolve spontaneously but may persist for minutes to hours. Hallmark symptoms of concussion include confusion and amnesia. [2]

Symptoms of concussion:

- Vacant stare
- Slow to answer questions
- Inability to focus
- Slurred speech
- Incoordination
- Emotionally labile
- Memory deficit
- LOC
- Nausea

Second Impact Syndrome

Diffuse cerebral swelling is a rare but potentially fatal complication of a secondary head injury (concussion) prior to full recovery from a previous concussion. This phenomenon is thought to also be present in shaken baby syndrome. Each successive concussion is at increased risk for more pronounced cerebral swelling and long term effects such as dementia.

Chronic traumatic encephalopathy refers to the permanent changes in mood, behavior, cognition, somatic symptoms and in severe cases, dementia or Parkinson's symptoms that occur with patient with multiple

concussions over the course of an athletic career.

90% of high school athletes who experience sport-related concussions will be symptom free within one month of injury if properly removed from physical activity until resolution of symptoms without medication.

Education is paramount in proper treatment and minimization of chronic complications for the pediatric and adolescent populations.

[3]



Treatment considerations for transport

Airway and breathing

- Consider intubation when GCS < 8, marked respiratory distress, and hemodynamic instability
- Always remember to maintain C spine precautions when mechanism suggests possible cervical injury.
- RSI considerations include pretreating with Lidocaine to minimize increase in ICP
- **Since the presence of CO₂ causes vasodilation**, keeping ETCO₂ between 35-38 (low normal levels) is ideal. By decreasing CO₂ we are helping control ICP and minimizing intracerebral blood volume. Lower CO₂ levels by increasing TV and increasing RR on the ventilator. **DO NOT hyperventilate below 35 unless signs of herniation are present.** [1]

Fluid management

- Prevent hypotension by administering isotonic fluids. The presence of NaCl increased intravascular Na levels, and where Na goes, water goes. You are essentially "pickling" the brain and helping control intracranial pressure. [1]

Other considerations:

- Maintain neutral head positioning
- Make sure C spine immobilization is not compressing at the carotids and causing decreased intracranial venous drainage.
- Increase HOB to at least 30 degrees
- Control shivering- increases ICP
- Prevent seizure activity. The key is prophylactic treatment before seizure activity occurs. Seizure activity grossly increases ICP and can cause

significant secondary injury. [1]

- Hyperthermia should be aggressively treated- increases metabolic demand and results in decreased O₂ at the tissues.
- Mannitol- Osmotic diuretic. Be mindful that administration may cause or worsen hypotension.
- Consider hyperosmolar therapy- "pickle the brain"
 - 3% NaCl
 - Does not cause hypotension and decreases ICP while increasing CPP.
- Control glucose by keeping levels < 200.
- Request neurosurgery evaluation for possible surgical evacuation of hematoma or removal of skull flap to provide acute swelling allowance.
- [1]

AROUND TOWN



We know children.

References

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