

Head Trauma

Epidemiology

Incidence:

200/100,000 children per year
Higher in spring and summer, weekends and afternoons

Etiologies

falls from heights
motor vehicle accidents
sports and recreational injuries
acts of violence
25% of head injuries in children <2 years of age are inflicted
interchild violence is becoming more common

Severity

Mild 82% (GCS > 12)
Moderate to severe 14% (Moderate: 12 GCS >8; Severe GCS 8)
Fatal 5%

Injuries:

Primary (frequently there are components of both of the following)

Impact (the head striking a surface)

scalp bleeding
skull fractures
closed
open
depressed
epidural hematomas
where the impact disrupts a dural vessel
leading to blood accumulation in the epidural space
Brain contusions and Lacerations
Subgaleal Hematoma
potential space outside periosteum, can lead to large hemorrhage and
hemodynamic compromise can occur even with minor trauma

Inertial (violent motion inside the skull)

subdurals
disruption of blood vessels feeding the arachnoid
concussion
diffuse axonal injuries
most common cause of prolonged coma

Neck Injuries

sciwra - spinal cord injury without radiological abnormality
even with normal c-spine films. The flexibility of the neck in childhood, and the large head on a relatively weak neck can result in neck and cervical spine injuries despite normal films. MRI or other cord imaging techniques should be considered when the history is suggestive of severe head and neck trauma.

Secondary Injuries (are due to the following and therefore are potentially limitable)

hypoxia
hypotension
brain swelling/edema
infarction
delayed hemorrhage
pressure necrosis
herniation

Assessment

In addition to evaluation of injuries, an important component of management of Head Trauma involves assessment of neurologic status. The first clinical sign of increased intracranial pressure is altered level of consciousness. Cushing's Triad, pupillary changes and papilledema occur later. The Glasgow Coma Scale (GCS) and Modified GCS for Children are the most commonly applied tools for assessment of level of consciousness. The score is the sum of scores for the best response in each category, giving a scale from 3-15.

Glasgow Coma Scale

<u>Activity/Best Response</u>	<u>Score</u>
Eye Opening	
Spontaneous	4
To Verbal Stimuli	3
To pain	2
None	1
Verbal	
Oriented	5
Confused	4
Inappropriate Words	3
Nonspecific Sounds	2
None	1
Motor	
Follows Commands	6
Localizes Pain	5
Withdraws to pain	4
Flexion to pain	3
Extension to pain	2
None	1

Children's Memorial GCS Modification

<u>Activity/Best Response</u>	<u>Score</u>
Eye Opening	
Spontaneous	4
To Speech	3
To pain	2
No response	1
Best Motor	
Spontaneous (obeys commands)	6
Localizes Pain	5
Withdraws to pain	4
decorticate posture to pain (flexion)	3
decerebrate posture to pain (extension)	2
No response	1
Best Verbal Response	
Oriented (Social Smile, orients, follows)	5
Confused/disoriented (consolable)	4
Inappropriate words/cries	3
Incomprehensible sounds or agitation	2
None	1

Management

Goal: to minimize secondary injuries by prevention of the items listed above.

Before we move on to discussion of the individual items above we will review the basics of Head Trauma management

Priorities:

- 1) **A**irway
- 2) **B**reathing
- 3) **C**irculation

Always remember: if your patient is not breathing, and/or does not have a heart beat, it doesn't matter what the extent of other injuries are, they are not going to survive without rapid assessment and intervention.

Issues of ABC's particular to Head Trauma patients:

Airway:

- high association of spinal cord injury with severe head trauma
- maintain head in "sniffing" position for intubation
- SCIWRA - spinal cord injury without radiographic abnormalities is an emerging entity, don't be lulled into complacency by spine films
- frequently have associated facial injuries and or basilar skull fracture
- avoid nasal intubation (including NG placement)
- airway manipulation will commonly cause spikes in ICP
- premedication with Atropine (0.02mg/kg), Pentothal or Versed, Lidocaine (1mg/kg) and muscle relaxants can minimize the ICP spikes

Breathing:

- one of our most powerful tools in emergent ICP management is PCO₂
- hyperventilation will be discussed in the next section

Circulation:

Frequently patients with head trauma will have associated other injuries, often including significant blood loss, leading towards shock. As with any patient in shock aggressive fluid management is necessary.

Your goal is to achieve euvolemia. As will be discussed in the next section, maintenance of the patients blood pressure is especially important in managing patients with severe head trauma

Episodes of Hypotension and Hypoxia are associated with increased morbidity and mortality in patients with severe head trauma. By aggressively addressing the ABC's one can attempt to minimize the occurrence of these events.

Cerebral Perfusion Pressure

$$\text{CPP} = \text{MAP} - \text{ICP}$$

Where MAP is the mean arterial pressure

ICP is the intracranial pressure

Cerebral Blood flow is highly dependent on cerebral perfusion pressure. The brain autoregulates to maintain stable CBF at pressures between 50 and 150mmHg. As the perfusion pressure falls below the numbers listed below the blood flow to the brain drops off rapidly and therefore the metabolic demands of the brain go unmet.

Goal CPP's

Adults	>60-70mmHg
Children	>50-60mmHg
Infants	>40mmHg

Mean Arterial Pressure

$$\text{MAP} = \text{CO} \times \text{SVR}$$

Where

CO is cardiac output

SVR is systemic vascular resistance

$$\text{CO} = \text{HR} \times \text{Stroke Volume}$$

SV is a function of preload, contractility and afterload (SVR)

To Increase MAP:

s CO

increase heart rate (chronotropic medications)

increase preload (volume)

increase contractility (inotropic medications)

s SVR

vasopressor medications (epinephrine, dopamine, phenylephrine)

Intracranial Pressure

To understand ICP and it's management there are several concepts that one must first understand.

The head as a closed box

Total volume inside calvarium =

$$V_{\text{Brain}} + V_{\text{blood}} + V_{\text{Cerebrospinal fluid}} + V_{\text{other}} = \text{constant (in patient with closed fontanel)}$$

Monro-Kellie Doctrine (Manifest Destiny ... not)

a change in one compartment must be balanced by a change in another

i.e. if the volume of blood in the brain increases the volume of brain or spinal fluid must decrease to maintain the volume,

V_{Brain} - brain volume increases with swelling and edema

Edema

- vasogenic (common around tumors, uncommon in trauma)
- cytotoxic (secondary to cell injury - common in trauma)
- interstitial (due to increased CSF hydrostatic pressure)

Management

- fluid restriction, but maintain euvolemia (see MAP discussion)
- hyperosmolar therapy
 - mannitol 0.25-1 gm/kg/dose
 - make sure patients have been adequately volume resuscitated first
 - recent evidence suggests using prn for increased ICP, not as scheduled med
 - hypertonic saline (also increases osms)
- Steroids - ONLY BENEFICIAL IN VASOGENIC EDEMA, they have not been shown to be of any benefit in trauma

V_{blood}

cerebral blood flow is fairly constant with perfusion pressure in the 50-150 range

factors that influence CBF

- perfusion pressure (see discussion on CPP)
- pCO₂ - cerebral blood vessels are very sensitive to pCO₂
 - hyperventilation
 - decreases CBF to both normal and injured brain
 - can be source for further injury by decreasing blood flow

current views:

- hyperventilation is not recommended in the early stages of increased ICP
- except aggressive hyperventilation (pCO₂ <30) for episodes of acute IICP

cerebral metabolic rate

- hypothermia/fever control
 - mild hypothermia and preventing fever is helpful, no evidence of deep hypothermia
 - being of benefit (at or below 32°C is too cold)

sedation/analgesia

- pain, noxious stimuli increase ICP
- benzodiazepines and narcotics are recommended to prevent spikes

seizure control (will be discussed later)

- seizures increase ICP and metabolic rate, important to gain control

pentobarbital coma

- last resort, decreased metabolic rate but no study to show improvement of outcome
- causes significant hemodynamic instability
- versed coma has also been used with fewer side effects

cerebral oxygen delivery

- hypoxia causes vasodilation increasing CBF and increasing ICP
- profound hypoxia causes injury
- maintain adequate oxygenation

V_{Cerebrospinal fluid}

- ventriculostomy - gold standard for ICP monitoring and CSF removal
- other methods include: intraparenchymal monitor, subarachnoid, subdural (bolt) and epidural
- none of which are as reliable as the ventriculostomy

V_{other}

- in the case of trauma this is usually hematoma
- optimal management would therefore be evacuation

When to Monitor ICP?

When GCS <8, and
clinical evidence of elevated ICP
from physical exam
CT evidence
OR observation

There is increasing debate as to whether monitoring itself changes outcome, as opposed to aggressive management of presumptive increased ICP

Summary:

To decrease ICP

fluid restrict (but not below euvoemia)
hyperosmolar therapy
avoid hyperventilation
 save both hyperosmolar and hyperventilation therapy for acute spikes in ICP
minimize metabolic demands by
- preventing fever, keep slightly cool
- pain control/sedation
- control seizures
monitor ICP with ventriculostomy if possible, also drain CSF as possible
evacuate hematomas if compromising pressure

Seizure Management

As many as 9% of patients have a generalized tonic-clonic seizure after severe head trauma. Seizures increase metabolic demand and can result in hypoxemia and hypercarbia in patients worsening ICP. For this reason aggressive early management is important. Seizures are more common in patients with penetrating injuries or parenchymal bleeds.

For patients with seizures:

immediate control with rapid acting benzodiazepine
diazepam (Valium) 0.1mg/kg IV, or
lorazepam (Ativan) 0.1mg/kg IV
Then load with Dilantin (20mg/kg total dose)

For patients without seizures:

because of risk associated with seizures, prophylaxis is recommended for all patients with severe head trauma who are comatose, after hemodynamic stability is achieved.
Dilantin 20mg/kg load recommended
this is preferred over phenobarb because it has less respiratory depression and less effect on the patients level of consciousness

Review of Secondary Injury Prevention

We stated earlier that the following list is responsible for a majority of the preventable injury following severe head trauma. Now we can look at them with regard to cerebral perfusion pressure management principles

Secondary Injuries

hypoxia
hypotension
brain swelling/edema
infarction
delayed hemorrhage
pressure necrosis
herniation

Hypoxia:

- effect:* causes local vasodilation which increases V_{blood} and therefore increases ICP, decreasing CPP
- management goals:*
 - maintain O_2 delivery
 - adequate Hgb, goal usually 9-10gm/dl if needing to transfuse. getting above 12 may cause viscosity problems
 - supplemental O_2

Hypotension

- effect:* decreases CPP, therefore decreases cerebral blood flow
- management goals:*
 - euvolemia
 - fluid resuscitate as needed, goal to not overly hydrate
 - inotropes other mechanisms to increase BP, Dopamine, epinephrine, dobutamine

Brain Swelling/Edema (V_{brain})

- effect:* increases V_{brain} , increasing ICP and decreasing CPP
- management goals:*
 - minimize edema as outlined earlier

Infarction

- effect:* tissue damages causes inflammation, cytotoxin release, can increase metabolic demand, alter blood flow, changing CPP
- management goals:*
 - maintain CPP

Delayed Hemorrhage

- effect:* increases V_{other} by formation of epidural, subdural hematomas. This increases ICP, causing decreased CPP
- management goals:*
 - monitor neuro status for acute changes
 - monitor coagulation status, try to normalize coags to avoid bleed
 - early recognition and intervention are key

Pressure Necrosis

- effect:* causes loss of brain parenchyma due to increased ICP and decreased CPP, especially in local areas
- management goals:*
 - decrease ICP as outlined earlier

Herniation

- effect:* death due to compression of brain stem and loss of autonomic function
- management goals:*
 - close neurologic monitoring and intervention
 - monitoring pupils
 - acute changes in neurologic status (pupil change, posturing, etc.) must be met by hyperventilation (now keep $pCO < 30$)
 - hyperosmolar therapy - stat dose of Mannitol 1gm/kg
 - other intervention to decrease ICP

Outcome

neurological sequelae

motor deficits

spasticity, incoordination and ataxia are most common

feeding difficulties

dysphagia, GER

sensory deficits

vary depending on injury, any cranial nerve can be involved

hearing and vision should be formally tested

communication

both speech (motor) and language (cognitive) deficits are common

cognitive

directly related to severity of trauma

don't sustain as complete recovery as from motor deficits

behavioral

personality changes related to lack of control are common

Predictors of neurologic deficits:

20% of mild brain injury have some deficit

90-100% of moderate to severe will have deficit

related to:

duration of coma (longer is worse)

focal injuries are worse

secondary brain injuries (diffuse swelling has high incidence of deficits)

epidurals evacuated early have good recovery

insidious subdurals that are not recognized early have high incidence of deficits

Seizures

2-9% of children will have seizures following trauma

risk factors:

brain contusion/hematoma (particularly subdural)

diffuse cerebral edema

GCS < or = 12

may not have seizures in initial period after trauma

most (95%) will develop them within 3 years of injury

Prevention

All head trauma is potentially preventable

Falls from heights

protective environment

child safe playgrounds

use of gates by stairs

minimize use of walkers

Motor vehicle accidents

use of helmets by bicyclists

(88% reduction in head injury in Seattle)

use of car seats

pedestrian education

driver education

better car design

Assault

limitation of gun availability

education about shaken babies

Resources:

Noah ZL, et al "Management of the Child with Severe Brain Injury." **Critical Care Clinics 8(1):** 59-77. January 1992

Reviews the basics of initial and ICU management of patients with severe brain injury.

Michaud LJ, et al. "Traumatic Brain Injury in Children." **Pediatric Clinics of North America 40 (3):** 553-565. June 1993.

Written from a rehabilitation standpoint, has nice summary of the types of injuries and prognosis. a large amount of the epidemiology in this handout was taken from this paper.

Temkin NR, Dikmen SS and Winn HR. "Post-Traumatic Seizures." **Neurosurgery Clinics of North America 2(2):** 425-435. April 1991.

An excellent review of the literature of post-traumatic seizures. Looks more at efforts to prevent long term seizures, which unfortunately seems unfruitful.

Levin HS. "Head Trauma." **Current Opinion in Neurology 1993 6:** 841-6.

A concise review of the gamut of head injury both in pediatrics and adults. Has an excellent summary of the literature in the resource section.

Chestnut RM, Prough DS eds. *Critical Care of Severe Head Injury.* **New Horizons 3 (3):** 365-593.

A very comprehensive review of the most recent research on almost every aspect of head trauma from bench work to clinical practice.