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Pediatric head trauma is common and can range from minor to severe. Most head injury in children is mild and not associated with brain injury or long-term problems. However 40% of deaths from trauma are the results of traumatic brain injury.

Severity of traumatic brain injury (TBI) is usually defined by the initial Glasgow Coma Score (GCS):

Mild – GCS 13 – 15

Moderate – GCS 9 – 12

Severe – (GCS < 9)

### **Mechanisms of injury**

Falls are the most common mechanism of injury for children sustaining minor head trauma, followed by MVA, pedestrian and bike accidents, assaults, sports related trauma and abuse.

Infants sustain more falls and are at an increased risk for inflicted injuries.

Brain injury can occur following a minor head trauma because of rotational acceleration-deceleration of the head generates shearing forces that cause mechanical disruption of nerve fibers, resulting in diffuse axonal injury. This process has been described in association with severe brain injury and occurs in mild traumatic brain injury as well. The type of mechanical forces applied to the brain may determine to some extent the nature of the resultant injury.

- Acceleration occurs when a moving object strikes a stationary head. Linear acceleration is considered the least injurious force and typically is associated with superficial contusions or, in some cases, subdural hematomas.
- Deceleration results when a moving head strikes a stationary surface. Sudden deceleration is thought to be responsible for most severe brain stem injuries.
- Rotation of the brain occurs when the head is struck in an asymmetric manner or an infant is vigorously shaken. Rotational acceleration-deceleration can induce widespread injury.



## **Pathophysiology of traumatic brain injury**

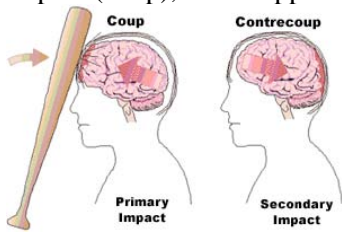
Mild TBI usually occurs with head injury due to contact and/or acceleration /deceleration forces. The pathophysiology of severe TBI involves two insults. The primary event is the direct injury to the brain parenchyma. Initially following brain injury, cerebral blood flow appears to be decreased in children. Hypoperfusion along with increased metabolic demand makes the brain more susceptible to secondary insults such as hypoxemia and hypotension. Cerebral perfusion may be particularly dependent on maintaining adequate blood pressure because cerebral autoregulation is often impaired following severe pediatric TBI. Following this initial phase, cerebral swelling develops that generally peaks 24 to 72 hours after the injury. The resulting intracranial hypertension can further compromise cerebral perfusion leading to more ischemia, swelling, herniation and death.

## **Types of brain injury**

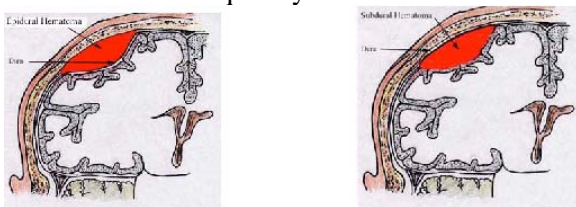
Diffuse brain injury (DBI) is the most common type of severe traumatic brain injury in children and is usually produced by acceleration or deceleration forces. The mildest form of DBI is a concussion.

Diffuse axonal injury (DAI) is a more severe form of diffuse brain injury. DAI develops as the result of tissue shearing at the interface of grey and white matter. Axons are the microscopic nerve fibers of neurons, the brain cells that communicate with each other. Axons form the long connecting nerve fibers of the neural networks throughout the brain. After a closed head injury, the shifting and rotation of the brain inside the skull causes a shearing injury to the brain's complex circuitry. This axonal shearing can occur in localized areas or throughout the brain. The latter is called "diffuse axonal shear." Furthermore, the brain cells particularly important to learning and memory (cholinergic neurons), are apparently more vulnerable to trauma than other neurotransmitter systems.

Focal injuries include brain contusions and intracranial hemorrhage. An acceleration or deceleration injury can result in a cerebral contusion. Contusions may be in the location of the impact (coup), or the opposite side of the brain (contrecoup), or both. Intracranial



Epidural hematoma, subdural hematoma, or subarachnoid hemorrhage usually occurs as the result of blunt trauma. In children, an epidural hematoma may arise from the middle meningeal artery, the middle meningeal vein, diploic veins, or venous sinuses. A subdural hematoma results from rupture of bridging veins. Subarachnoid hemorrhage develops from tearing of small vessels in the pia mater. Subdural hematoma and subarachnoid hemorrhage usually occur as the result of severe trauma and are frequently associated with other intracranial injuries.

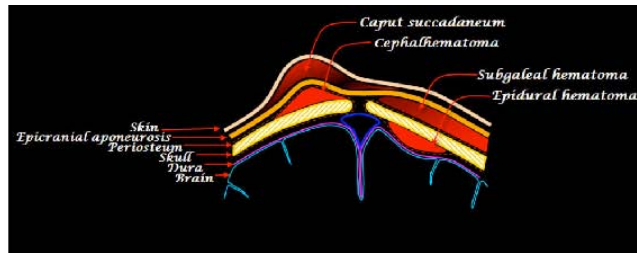
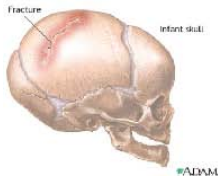


## **Skull Fractures**

Skull fractures are relatively common injuries in children. Although most are the result of accidental mechanisms such as falls and motor vehicle crashes, inflicted injury causes significant

morbidity and mortality in young children.

Linear fractures account for approximately 75% of skull fractures and 15 to 30% of children with linear skull fractures have associated intracranial injury. Physical findings include overlying hematoma or soft tissue swelling. Most linear skull fractures heal without complication. Rare complications include formation of subgaleal (space between the skull periosteum and the scalp galea aponeurosis) or epidural hematoma.



Depressed skull fractures are the result of significant force and frequently are associated with underlying cerebral injury by lacerating the underlying dura or penetrating the brain tissue. Depressed skull fractures increase the likelihood of posttraumatic seizures and infection.



Open skull fractures permit communication between the skull and the scalp or mucosal surfaces of the upper respiratory tract, establishing a risk for central nervous system infection.



Basilar skull fractures involve the base of the skull. Distinct clinical findings are present in approximately 80% of cases. These include bleeding over the mastoid process (battle sign) or around the orbit (raccoon eyes), hemotympanum (blood behind the tympanic membrane, cerebrospinal fluid rhinorrhea (CSF leakage through the nose) or otorrhea (CSF leakage through the ear) and cranial nerve deficits



Cervical spine injury must always be suspected for children with traumatic brain injury. Multiple trauma is common among children with severe traumatic brain injury.

### **Evaluation**

Children with severe traumatic brain injury must be promptly recognized and their conditions emergently stabilized in order to limit secondary brain injury and improve outcomes. Injuries that required immediate neurosurgical intervention and associated injuries that may impact management must also be identified. A systematic primary survey to rapidly identify and treat potentially fatal conditions such as airway compromise, impaired respiratory mechanics and shock should be initially performed. This should be followed by a secondary survey that includes a fully exposed head-to-toe examination of the child with a neurological assessment...

Historical features that may suggest an increased risk of intracranial injury include the following:  
 High-risk mechanism such as fall from a significant height, any mechanism involving a motor vehicle, penetrating injury, inflicted injury, or unknown mechanism.

Seizure, confusion, headache, vomiting, severe headache, progression of symptoms, or loss of consciousness and/or altered mental status

Preexisting conditions that place the child at risk for intracranial injury, which include ventricular shunt, arteriovenous malformation or bleeding disorder.

The initial assessment of the child with severe TBI should include a focused neurological examination and assignment of a Glasgow Coma Scale score. A focused neurologic examination should include the following:

Mental status (arousal, orientation, memory, GCS)

Pupillary examination for size, reactivity, equal, gaze palsy)

Respiratory rate/pattern/depth

Pulse pressure (the difference between systolic and diastolic pressure)

Motor/sensory integrity/deficits in all extremities

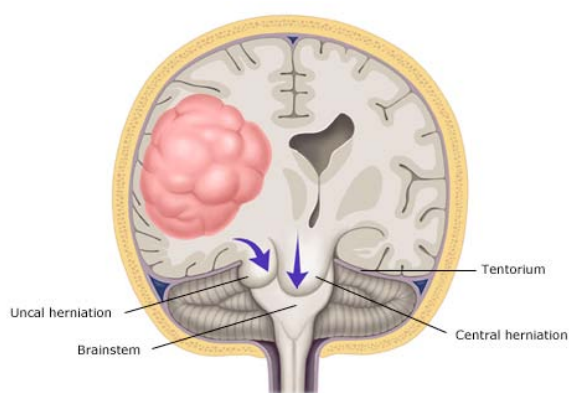
Any abnormalities in this examination may suggest increased ICP with impending herniation.

Signs of herniation that must be recognized include the following:

Third cranial nerve palsy, followed immediately by hemiplegia.

Progressive changes in respiratory pattern, pupil size, vestibuloocular reflexes (nystagmus or deviation of the eyes in response to stimulation of the vestibular system by angular acceleration or deceleration or when the caloric test is performed), and posture that correlate with the anatomic level of brain involvement.

Cushing's triad (hypertension, bradycardia and slow irregular respirations).



Clinical Signs of Central Transtentorial Herniation with Rostral-Caudal Deterioration

Anatomic stage pattern	Respiratory pattern	Pupils	Vestibulo-Ocular reflexes	Motor response
Diencephalon	Regular or Cheyne-Stokes	Small, reactive	Present, normal	Local noxious stimuli with nonparetic limb; later decorticate posturing
Midbrain-upper pons	Hyperventilation or Cheyne-Stokes	Midposition, fixed	Absent or abduction only	Decerebrate or no movement
Lower pons-upper medulla	Ataxic	Midposition, fixed	Absent	No movement or triple flexor withdrawal in legs only
Medulla	Irregular	Midposition, fixed	Absent	Absent

## Management of the Pediatric Head Injured Patient

Scene size up Initial assessment

General Impression

Determine if life threat exists

Level of Consciousness (AVPU)  
 Airway/Spine motion restrictions  
 Breathing/Oxygenation – O2 adjuncts  
 Circulation/Perfusion – assess perfusion, IV/IO access  
 Rapid neuro assessment – Peds GCS  
 Blood glucose

<b>Eye Opening</b>	
Spontaneously	4
To speech	3
To pain	2
None	1

<b>Best Verbal &gt; 5 years</b>		<b>Best Verbal 2 – 5 years</b>		<b>Best Verbal &lt;2 years</b>	
Oriented/converses	5	Appropriate words/phrases	5	Smiles, coos, cries appropriately	5
Disoriented/Converses	4	Inappropriate words	4	Cries & is consolable	4
Inappropriate words	3	Cries/Screams	3	Persistent inappropriate crying/screaming	3
Incomprehensible sounds	2	Moans/grunts to pain	2	Moans/grunts to pain	2
None	1	None	1	None	1

<b>Best Motor</b>	
Moves spontaneously and purposefully	6
Localizes pain/ withdraws to touch	5
Withdraws to pain	4
Abnormal flexion – decorticate	3
Abnormal extension – decerebrate	2
None	1

- Expose and keep warm Transport decision
- Level I or II trauma center patients are time sensitive, scene time 10 minutes or less Focused history and physical Watch of seizure (treat per SOP), vomiting Frequent reassessments

**Pediatric Glasgow Coma Score for ages 3 years and under**