

***MONITORING OF ACUTE TRAUMATIC CEREBRAL  
INJURY IN PEDIATRIC***

***Essay***

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In Anesthesiology***

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# ABBREVIATIONS

<i>ABG</i>	<i>Arterial Blood Gases</i>
<i>AjvDO<sub>2</sub></i>	<i>Difference in Oxygen content between Arterial and Jugular Venous Blood</i>
<i>AMP</i>	<i>Amplitude</i>
<i>ATP</i>	<i>Adenosine Tri Phosphate</i>
<i>AVMs</i>	<i>Arterio Venous Malformations</i>
<i>BSI</i>	<i>Bi Spectral Index</i>
<i>CaO<sub>2</sub></i>	<i>Arterial Oxygen Content</i>
<i>CBC</i>	<i>Complete Blood Picture</i>
<i>CBF</i>	<i>Cerebral Blood Flow</i>
<i>CBFV</i>	<i>Cerebral Blood Flow Velocity</i>
<i>CBV</i>	<i>Cerebral Blood Volume</i>
<i>cEEG</i>	<i>Continuous Electroencephalography</i>
<i>CFM</i>	<i>Cerebral Function Monitor</i>
<i>CFAM</i>	<i>Cerebral Function Analysing Monitor</i>
<i>CjvO<sub>2</sub></i>	<i>Oxygen Content of Jugular Venous Blood</i>
<i>CMRO<sub>2</sub></i>	<i>Cerebral Metabolic Rate of Oxygen</i>
<i>CNS</i>	<i>Central Nerves System</i>
<i>CPB</i>	<i>Cardio Pulmonary Bypass</i>
<i>CPP</i>	<i>Cerebral Perfusion Pressure</i>
<i>CSF</i>	<i>Cerebro Spinal Fluid</i>
<i>CT</i>	<i>Computed Tomography</i>
<i>CVR</i>	<i>Cerebrovascular Resistance</i>
<i>DAI</i>	<i>Diffuse Axonal Injury</i>
<i>DO<sub>2</sub></i>	<i>Cerebral Oxygen Delivery</i>
<i>DRS</i>	<i>Disability Rating Scale</i>
<i>DVT</i>	<i>Deep Vein Thrombosis</i>
<i>EBIC</i>	<i>European Brain Injury Consortiums</i>
<i>ECF</i>	<i>Extra Cellular Fluid</i>
<i>ECG</i>	<i>Electrocardiogram</i>

<i>EEG</i>	<i>Electroencephalogram.</i>
<i>ETCO<sub>2</sub></i>	<i>End Expiratory CO<sub>2</sub></i>
<i>EVD</i>	<i>External Ventricular Device</i>
<i>FIM</i>	<i>Functional Independence Measure</i>
<i>FV<sub>mean</sub></i>	<i>Mean Flow Velocity</i>
<i>GABA</i>	<i>Gamma Amino Butyric Acid</i>
<i>GCS</i>	<i>Glasgow Coma Score.</i>
<i>GI</i>	<i>Gastro Intestinal</i>
<i>GOS</i>	<i>Glasgow Outcome Scale</i>
<i>GU</i>	<i>Genito Urinary</i>
<i>HB</i>	<i>Hemoglobin</i>
<i>HbO<sub>2</sub></i>	<i>Oxygenated Haemoglobin</i>
<i>Hct</i>	<i>Hematocrit Value</i>
<i>HPLC</i>	<i>High Performance Liquid Chromatography</i>
<i>HR</i>	<i>Heart Rate</i>
<i>ICP</i>	<i>Intracranial Pressure.</i>
<i>NICU</i>	<i>Neuro Intensive Care Unite.</i>
<i>JVP</i>	<i>Jugular Venous Pressure</i>
<i>LED</i>	<i>light Emitting Diode</i>
<i>LOC</i>	<i>loss of Consciousness</i>
<i>MABP</i>	<i>Mean Arterial Blood Pressure.</i>
<i>MAP</i>	<i>Mean Arterial Pressure.</i>
<i>MCA</i>	<i>Middle Cerebral Artery</i>
<i>MD</i>	<i>Microdialysis</i>
<i>MEPs</i>	<i>Motor Evoked Potential</i>
<i>MRI</i>	<i>Magnetic Resonance Image.</i>
<i>MV</i>	<i>Motor Vehicle</i>
<i>NIRS</i>	<i>Near-Infrared Spectroscopy</i>
<i>PaO<sub>2</sub></i>	<i>Arterial Partial pressure of Oxygen.</i>
<i>PaCO<sub>2</sub></i>	<i>Arterial Partial Pressure of Carbon dioxide.</i>
<i>PbtO<sub>2</sub></i>	<i>Brain Tissue Partial Pressure Oxygen</i>
<i>PE</i>	<i>Pulmonary Embolism</i>
<i>PET</i>	<i>Positron-Emission Tomography</i>
<i>PRx</i>	<i>Pressure-Reactivity Index</i>

<i>PTA</i>	<i>Post-Traumatic Amnesia</i>
<i>PTS</i>	<i>Posttraumatic Seizures</i>
<i>PT</i>	<i>Prothrombin Time</i>
<i>PTT</i>	<i>Partial Thromboplastin Time</i>
<i>P1,2,3</i>	<i>Peak 1,2,3</i>
<i>RAP</i>	<i>Index of Compensatory Reserve</i>
<i>ROS</i>	<i>Reactive Oxygen Species</i>
<i>rSO<sub>2</sub></i>	<i>Regional Oxygen Saturation</i>
<i>SAH</i>	<i>Sub-Arachnoid Haemorrhage</i>
<i>SBP</i>	<i>Systolic Blood Pressure</i>
<i>SEPs</i>	<i>Sensory Evoked Potential</i>
<i>SHI</i>	<i>Severe Head Injury</i>
<i>SjvO<sub>2</sub></i>	<i>Jugular Venous Oxygenation Saturation</i>
<i>SPECT</i>	<i>Single-Photon Emission CT</i>
<i>SSEPs</i>	<i>Somatosensory Evoked Potential</i>
<i>TBI</i>	<i>Traumatic Brain Injury</i>
<i>TCD</i>	<i>Transcranial Doppler</i>
<i>TCU</i>	<i>Transcranial Doppler Ultrasonography</i>
<i>VMCA</i>	<i>Velocity of Middle Cerebral Artery</i>



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# *INTRODUCTION*

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# INTRODUCTION

Traumatic injuries remain the leading cause of death in children and in adults aged 45 years or younger. Head trauma is more likely in young children than in adults given the same deceleration of the body due to their large and heavy heads and weak cervical ligaments and muscles. Resulting brain injury is more severe due to their thin, pliable skulls and the yet unfused sutures. Accordingly, children below the age of 4 years have lower chances of a full recovery after severe TBI, although in general, neurologic recovery after severe brain injury in children is better than in adults. [1]

The pediatric brain has higher water content, 88% versus 77% in adult, which makes the brain softer and more prone to acceleration-deceleration injury. The water content is inversely related to the myelination process. The unmyelinated brain is more susceptible to shear injuries. Infants and young children tolerate intracranial pressure increases better because of open sutures. [2]

The time course of brain injury can be divided into two steps: primary and secondary injury. Primary brain injury exclusively results from the initial impact. [1]

Adverse physiologic conditions during recovery after head trauma may account for additional brain damage, which is then referred to as secondary brain injury which may occur hours or even days after the inciting traumatic event. Injury may result from impairment or local declines in cerebral blood flow after a traumatic brain injury. Decreases in

cerebral blood flow are the result of local edema, hemorrhage, or increased intracranial pressure. As a result of inadequate perfusion, cellular ion pumps may fail, causing a cascade. As the cascade continues, cells die, causing free radical formation, proteolysis, and lipid peroxidation. These factors can ultimately cause neuronal death. [3]

Three evidence based measures are of critical importance to prevent or minimize secondary brain injury: (a) avoid hypoxemia, (b) avoid post-traumatic arterial hypotension, and (c) refer the traumatized child to an experienced trauma team. [1]

The goal of monitoring in the injured brain is to enable the detection of harmful physiological events before they cause irreversible damage to the brain, thereby allowing diagnosis and effective treatment and providing ‘on-line’ feedback to guide therapy. Aims to minimize secondary injury in an attempt to optimize patient management and outcome. [4]

Continuous monitoring of intracranial pressure (ICP) and cerebral perfusion pressure (CPP) has become a standard in neurointensive care of severe head injured patients. In addition, head injured patients should have systemic parameters closely monitored, including ECG, heart rate, blood pressure, temperature, fluid intake and output. Routine monitoring of oxygen saturation and capnography is paramount in severely head injured patients so as to avoid unrecognized hypoxemia or changes in ventilation. New devices including: jugular venous oxygen saturation monitoring, Near-Infrared Spectroscopy, Cerebral Microdialysis, Cerebral Imaging Monitoring, Transcranial Doppler Ultrasonography, Continuous electroencephalography. [4]

# ANATOMY OF THE PEDIATRIC

## NEUROLOGY

Anatomy of pediatric head differs from that of adult so knowledge of the basic anatomy of the pediatric head, brain and its coverings is essential to understand the mechanism and types of traumatic brain injury in pediatric [5].

**The scalp** is the outer most covering and is highly vascular, tending to bleed profusely when lacerated. Under the scalp is a tendentious sheath extending from frontal to occipital regions called **the galea**. The potential space beneath the galea is the **subgaleal compartment** which an occasional site of bleeding after head injury. [6]

### Anatomy Of The Newborn Skull:

The skull is the next part after the scalp which is composed of three layers; the bony outer layer and inner tables layer separated by diploic space which is more vascular. Although the skull appears to be one large bone, there are actually several major bones that are connected together. These include: one *occipital* bone, two *parietal* bones and two *frontal* bones. These bony plates cover the brain and are held together by fibrous material called sutures which do not fuse in the child's skull until as late as the tenth year (fig.1-1). [7]