# Anesthetic Management of Pediatric Head Trauma

Essay

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# المعالجة التخديرية لحالات إصابات الرأس عند الأطفال

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# Summary

Head injuries account for 75% of all pediatric hospitalizations for trauma and contribute to nearly 80% of trauma deaths in children. Childhood head injuries are often the result of domestic falls, motor vehicle accidents, and recreational injuries such as sports mishaps. Child abuse remains a significant cause of head injuries in children under 2 years of age.

Traumatic brain injuries can be divided into two major stages: Primary brain injuries that occur at the time of impact include extradural and subdural hematomas, intra-cerebral contusions, and diffuse axonal injuries; Secondary injury occurs any time after the primary event, and potentially preventable causes include systemic hypotension, hypoxemia, hypercapnia, and hyperthermia.

Peri-operative management of head-injured patients focuses on aggressive stabilization of the patient and avoidance of systemic and intracranial insults that cause secondary injury.

The goal of the primary survey of trauma (the initial phase of resuscitation) is to rapidly evaluate and treat any immediate life-threatening injuries. It is performed in a prescribed sequence including evaluation of airway, breathing, circulation, disability, and exposure (ABCDEs).

Care should be taken in patients with suspected cervical spine fracture and a baseline neurologic examination should be performed after initial resuscitation,

and should be repeated at frequent intervals because the patient's condition may change rapidly.

Only by frequent evaluation and assessment can the physician detect and treat the child appropriately prior to decompensation.

The major goals of anesthetic management are to optimize cerebral perfusion and oxygenation, avoid secondary damage, and provide adequate surgical conditions for the neurosurgeons.

Most patients should be transferred to the ICU, the main objectives are to optimize recovery from primary brain injury and prevent secondary injury. This requires provision of optimal systemic support for cerebral energy metabolism and adequate CPP, and normalizing of ICP for the injured brain. To achieve this, multi-modality systemic and cerebral monitoring should be instituted.

# Table of contents

# Introduction

Head trauma is a common cause of injury in children, and it is responsible for the high number of hospital admissions, with a significant rate of morbidity and mortality ***(Löhr, 2002).*** Although there is increasing information concerning traumatic brain injury in adults, relatively little is known regarding cerebrovascular pathophysiology specific to children **(*Philip et al, 2009).***

Children are more susceptible to traumatic brain injury because they have a larger head to body size ratio, thinner cranial bones providing less protection to the intracranial contents, less myelinated neural tissue that makes them more vulnerable to damage, diffuse injury and cerebral edema compared with adults ***(Sookplung & Vavilala, 2009).*** There are many numerous differences between adults and children that affect anesthetic management. In addition to the differences in size, communication skills, and issues involving parents, there are important anatomic, physiologic and pharmacologic differences between pediatric and adult patients ***(Wetzel, 2008).***

The Glasgow Coma Scale is used to clinically classify traumatic brain injury as mild, moderate, or severe. Although useful in the acute phase, additional modalities such as high resolution computed tomography (CT), and magnetic resonance imaging (MRI) can be used to further classify traumatic brain injury in the acute setting ***(Saatman et al, 2008).***

The development of neuroprotective treatments for traumatic brain injury requires a clear classification of injury severity. A common method of classification separates immediate (primary) injury from delayed (secondary) injury. Primary injury results from direct mechanical force that leads to compression and shearing of neural, glial, and vascular cells. Secondary injury results from the release of biochemical or inflammatory factors that alter the loco-regional milieu in the acute, subacute, and delayed intervals after a mechanical insult ***(Walker et al, 2009).***

Current management strategies are directed towards providing an optimal physiological environment in order to minimize secondary insults and maximize the body's own regenerative processes. Modern management utilizes a host of monitoring techniques to identify or predict the occurrence of secondary insults and guide subsequent therapeutic interventions in an attempt to minimize the resulting secondary injury. Recent data suggest that the use of protocolized management strategies, informed by multimodality monitoring, can improve patient outcome after TBI ***(Tisdall & Smith, 2007).***

# Anatomical, Physiological & pathophysiological considerations

The provision of safe anesthesia for the pediatric patient requires a clear understanding of the anatomical, psychological, physiological, and pharmacological differences between patients in different age groups from newborn to adolescent. Consideration of these differences when providing anesthesia care to children is critical for achieving high-quality and efficient care ***(Cravero & Kain, 2006).***

The ranges of normal physiological parameters such as cardiovascular observations change with age (table 1) ***(Hall, 2009).*** At birth cardiac output is 200 ml/kg per minute and progressively decreases to 100 ml/kg per minute by adolescence. Resting stroke volume remains fairly constant at about 1 ml/kg per minute, the increased cardiac output in younger patients being maintained by an increase in the heart rate Heart rates up to 200 bpm can be tolerated ***(Astuto et al, 2009)***. Neonatal and infant heart rates less than 60 bpm require external cardiac compression ***(McQuillan et al, 2008).***

***Table (1)*****:** Normal vital signs by age ***(Holzman, 2008).***

| Age-group | Heart rate (beats/min) | Systolic blood pressure (mm Hg) | Respiration (breaths/min) |
|---|---|---|---|
| Newborn | 95-145 | 60-90 | 30-60 |
| Infant | 125-170 | 75-100 | 30-60 |
| Toddler | 100-160 | 80-110 | 24-40 |
| Preschool | 70-110 | 80-110 | 22-34 |
| School age | 70-110 | 85-120 | 18-30 |
| Adolescent | 55-100 | 95-120 | 12-16 |

The weight of the child increases with age and it is often necessary to estimate it. Approximate weights can be determined from the following formula ***(Hall, 2009)***:

- At birth: 3-3.5 kg
- 3-12 month: weight (kg) = [age (month) + 9]/2
- 1-6 yr: weight (kg) = [age (yr) + 4] × 2

The infant brain grows rapidly. It doubles in size in the first year and reaches 80% of adult weight by the age of 2. Brain weight at birth represents a larger percent of total body weight than in the adult (10% *versus* 2%), and a proportionally larger part of the cardiac output is directed to the brain. The infant skull sutures are not fused. The fontanelles are open until age 2 to 3 months (anterior) and 7 to 19 months (posterior). Open fontanelles allow for non-invasive assessment of intracranial pressure (ICP) and ultrasound imaging of intracranial structures. A bulging fontanelle suggests elevated ICP. The skull bones grow in response to increases in intracranial volume. Under normal conditions, fusion of the skull sutures is not complete until adolescence ***(Van de Wiele, 2006).*** Children have smaller subarachnoid space where CSF flows cushioning the brain, offers less protection to the underlying brain tissue ***(Hall, 2009).***

Although the nervous system is anatomically complete at birth, myelination continues and functionally it remains immature. Myelination of the nervous system is rapid during the first 2 years of life and is complete by 7 years of age ***(Astuto et al, 2009)***.

CBF is usually higher in children and adolescents and drops further with age. Irreversible neuronal damage occurs when CBF drops below 10-15ml/100g/min.

Reversible neuronal injury occurs with CBF between 15 and 20ml/100g/min (fig.1). The central nervous system (CNS) has a high metabolic rate for oxygen ($CMRO_2$) and uses glucose predominantly as the substrate for its energy needs. At rest, the brain consumes oxygen at an average rate of approximately 3.5 ml of oxygen per 100 g of brain tissue per minute. Whole-brain $O_2$ consumption represents about 20% of total-body oxygen utilization (fig.2) ***(Torbey & Bhardwaj, 2004)***.

ICP is the pressure within the intracranial space relative to atmospheric pressure. It is difficult to establish a universal "normal value" for ICP as it depends on age (table 2), body posture, time and clinical conditions. The definition of raised ICP depends on the specific pathology. ICP values of 20 to 30 mmHg represent intracranial hypertension ***(Castillo et al., 2008)***.

Perfusion of the brain is dependent on the pressure gradient between the arteries and the veins and this is termed the cerebral perfusion pressure (CPP). This is the difference between the mean arterial blood pressure (MAP) and the mean cerebral venous pressure. The latter is difficult to measure and approximates to the more easily measured intracranial pressure (ICP) ***(Hill & Gwinnutt, 2008)***.

Autoregulation is the ability of the cerebral circulation to maintain CBF at a relatively constant level by altering cerebrovascular resistance (CVR) despite wide fluctuations in cerebral perfusion pressure. In normal human subjects, the limits of autoregulation occur at MAP values of approximately 50 and 150 mmHg ***(Nortje, 2008)***.

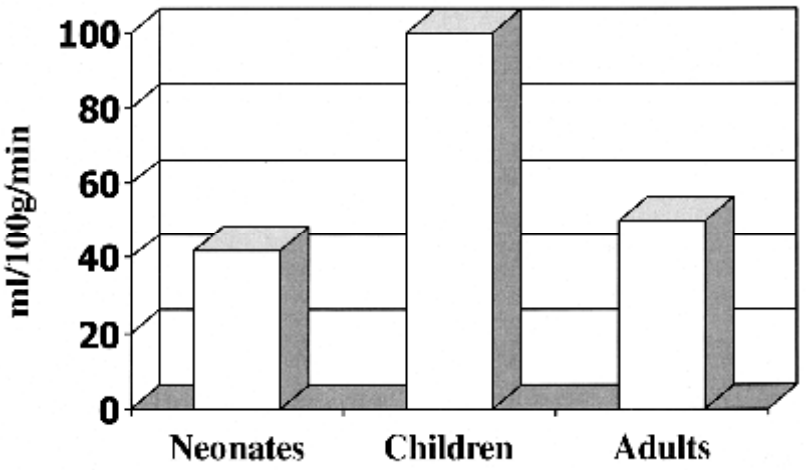


***Figure (1):*** Age-related differences in global cerebral blood flow (ml/100 g/min) ***(Van de Wiele, 2006).***

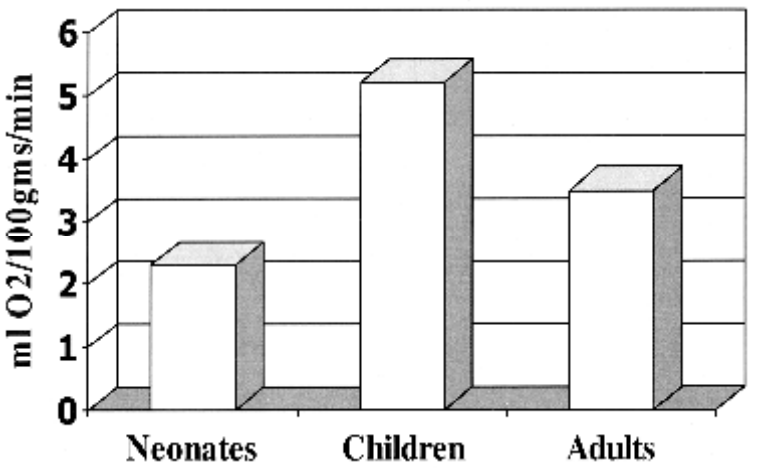


***Figure (2):*** Age-related differences in cerebral oxygen consumption (ml $O_2$/100 g/min) ***(Van de Wiele, 2006).***

***Table (2):*** Normal values of ICP in different age groups ***(Castillo et al, 2008)***.

| Age | ICP |
|---|---|
| Newborn | Sub-atmospheric |
| Infants | 1.5-6 mmHg |
| Younger children | 3-7 mmHg |
| Older children | 10-15 mmHg |

*Factors that influence cerebral blood flow and intracranial pressure:*

***1- Arterial blood gases:*** Arterial $CO_2$ tension ($PaCO_2$) is a major regulator of cerebral blood flow within the physiologic range of arterial $CO_2$ tensions (30-45 mmHg). CBF linearly increases 1 to 2ml/100g/min for each 1 mmHg increase in $PaCO_2$. Below an arterial $CO_2$ tension of 25 mmHg (fig. 3), the cerebral blood flow response to $PaCO_2$ is attenuated. The CBF changes in response to alterations in $PaCO_2$ are not sustained because bicarbonate is eventually transported out of the brain extracellular fluid, thereby returning pH to a normal value over a period of 6 to 8 hours. In contrast to the effects of respiratory acidosis on cerebral blood flow, the actions of metabolic acidosis are more gradual because the blood-brain barrier is relatively impermeable to $H^+$ ***(Kampine et al, 2009)***.

Changes in Arterial $O_2$ tension ($PaO_2$) from 60 to over 300 mmHg have little influence on CBF. At high $PaO_2$ values, CBF decreases modestly. Below a $PaO_2$ of 60 mmHg, CBF increases rapidly (fig. 3) ***(Kampine et al, 2009)***.

***2-Blood pressure***: Normally, cerebral auto-regulation keeps flow steady despite variations in blood pressure. Sudden increase in blood pressure can raise CBF (fig. 3). The most common stimuli are laryngoscopy and intubation, suctioning and skeletal fixation of the head, which must be considered during pre-operative visits. Wide fluctuations in blood pressure are poorly tolerated ***(Rammohan, 2007).***

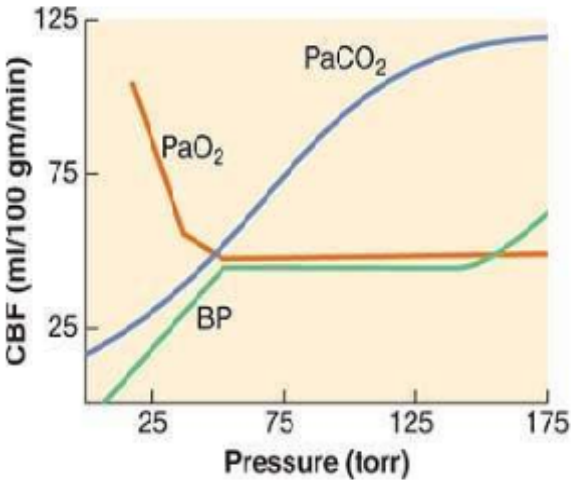


***Figure (3):*** Relation between CBF and changes in $PaO_2$, $PaCO_2$ and blood pressure ***(Kampine et al, 2009)***.

***3-Anesthetic agents:*** Both anesthetic techniques and agents greatly influence the ICP curve by their effect on intracranial blood volume, which is affected through change in blood flow ***(Reza, 2007).***

Thiopental, propofol, etomidate, benzodiazebines and all inhalational anesthetics except $N_2O$ reduce neuronal activity and so reduce the brain cerebral metabolic requirement for oxygen ($CMRO_2$). They provide a protective mechanism when oxygen demand may outweigh supply ***(Stanley & Norfolk, 2008).***