Perioperative Anesthetic Management of Traumatized Pediatric Patient

An essay
Submitted for Partial Fulfillment of Master Degree in Anesthesiology

By:

Usama El Sayad Abd El kawi
M.B., B.CH

Supervisors

Prof. Dr. Seham Hussein Mohammed Ewiss
Professor of Anesthesia and Intensive Care
Faculty of Medicine
Ain Shams University

Prof. Dr. Rasha Samir Abd Elwahab Bondok
Assistant Professor of Anesthesia and Intensive Care
Faculty of Medicine
Ain Shams University

DR. Mahmoud Ahmed Abd El Hakim
Lecturer of Anesthesia and Intensive Care
Faculty of Medicine
Ain Shams University

Faculty of Medicine
Ain Shams University
2009

Acknowledgment

First and foremost, thanks to Allah, the most gracious and merciful.

In grateful words, I would like to express my greatest thanks to all my professors who helped me throughout this work.

I can hardly find the words to express my gratitude to **Prof. Dr. Seham Hussein Mohammed Ewiss,** professor of Anesthesia and Intensive Care, Faculty of Medicine, Ain Shams University, for suggesting, supervising and planning this work. I am very much grateful for her noble characters and generous help. It is great honor to work under her guidance and supervision.

Also, I want to thank **Prof. Dr. Rasha Samir Abd Elwahab Bondok,** assistant professor of Anesthesia and Intensive Care, Faculty of Medicine, Ain Shams University, for her great help and advice. She granted me a great deal of her precious time and effort guiding me throughout the preparation of this essay.

Finally, I express my best feeling and thankfulness to **Dr. Mahmoud Ahmed Abd El Hakim,** lecturer of Anesthesia and Intensive Care, Faculty of Medicine, Ain Shams University, for his sincere care, extreme effort and his great assistance during every step in this essay.

Usama Elsayad Abd El Kawi

List of Abbreviations

ABG…….. Arterial Blood Gas
ADH……..… Anti Diuretic Hormone
AED…….. Automated External Defibrillator
ALS…….. Advanced Life Support
APSA…… American Pediatric Surgical Association
ARDS…… Acute Respiratory Distress Syndrome
BLS…….. Basic Life Support
CPAP……. Continuous Positive Airway Pressure
CPP…….. Cerebral Perfusion Pressure
CPR…….. Cardio Pulmonary Resuscitation
CSF……..… Cerebrospinal Fluid
CVP……..… Central Venous Pressure
DI…….…… Difficult Intubation
EBV…….…. Estimated Blood Volume
ECG..... Electrocardiography
ECMO…….. Extracorporeal Membrane Oxygenation
ED……. Emergency Department
ERP……... Endoscopic Retrograde Pancreatography
ETC O_2…….. End-tidal carbon dioxide pressure
GCS…….. Glasgow Coma Score
GFR…….. Glomerular Filtration Rate
HbA…….. Adult Hemoglobin
HbF……... Fetal Hemoglobin
I: E……. Inspiratory/Expiratory Ratio
ICP…… Intracranial Pressure
IO…….. Intraosseous
LMA…….. Laryngeal Mask Airway
MAC…… Minimal Alveolar Concentration
MAP…… Mean Arterial Pressure
MH…….. Malignant Hyperthermia

List of Abbreviations (cont.)

MILS……..	Manual Inline Stabilization
MVAs…..…	Motor Vehicle Accidents
NPSA……..	National Patient Safety Agency
NPTR……	National Pediatric Trauma Registry
$PaCO_2$……..	Arterial Carbone dioxide Tension
PCA…….	Post Conceptual Age
PEEP……..	Positive End-Expiratory Pressure
PO_2……..	Oxygen Tension
Sa O_2……..	Arterial Oxygen Saturation
SBS……..	Shaken Baby Syndrome
SCI……...	Spinal Cord Injury
SCIWORA..	Spinal Cord Injury without Radiologic Abnormality
*V*d/*V*t……	The ratio of physiological dead space to tidal volume
VF……	Ventricular Fibrillation
VT……...	Ventricular Tachycardia

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INTRODUCTION

The pediatric trauma continues to be one of the major threats to the health and well-being of children. The provision of safe anesthesia for the pediatric patient depends on a clear understanding of the anatomic, physiologic, pharmacologic, and psychologic differences between children and adults, Physicians who are responsible for the care of a pediatric trauma patient, must be familiar with every tenet of modern trauma care ***(American Academy of Pediatrics, 2008).***

Organizational changes for the delivery of emergency care have had a tremendous impact on trauma morbidity and mortality. The challenge is to reduce the mortality from trauma for patients who arrive at the hospital alive. The term "golden hour" This term emphasizes the need for rapid response to treat traumatic shock and to transport injured patients to definitive treatment centers. The future challenge for anesthesiologists and trauma surgeons is to prevent death from irreversible shock and to extend the treatment window of opportunity to golden 2 hours by perfecting in-hospital treatment of seriously injured patients. Such optimum care will give the trauma patient a chance to return to functional life. Efforts are currently underway worldwide to increase the level of involvement of anesthesiologists in clinical traumatology and research ***(Dodson and Kaban, 2000).***

This article focuses on the anesthetic considerations that apply to pediatric trauma patients, provides the physiological and anatomical consideration, types of trauma and pathological effect, preoperative evaluation and preparation, intraoperative management and postoperative intensive care management.

Physiological and Anatomical consideration

Anatomical consideration

Head& Neck

Neonates and infants have a proportionately larger head and tongue, narrow nasal passages, cephalad larynx (at a vertebral level of C4 versus C6 in adults), and a short trachea and neck (Figure 1) These anatomic features make neonates and most young infants obligate nasal breathers until about 5 months of age ***(Morgan et al., 2006).***

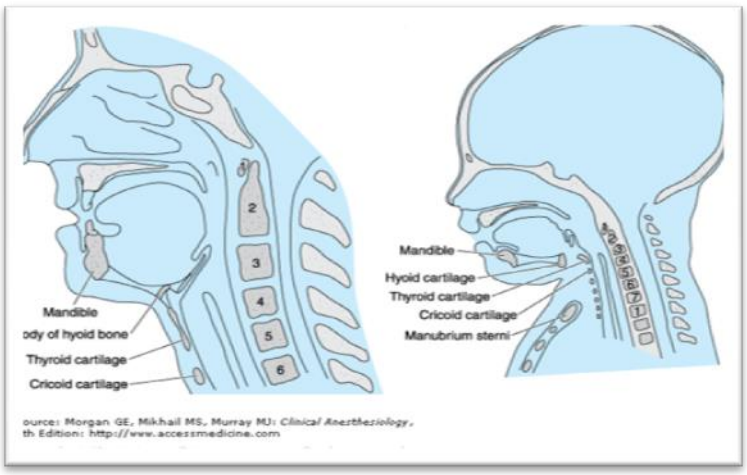

Figure: (1) Sagittal section of the adult and infant airway *(Morgan, 2006)*

Airway

Differences in airway anatomy make the potential for technical airway difficulties greater in infants than in teenagers or adults. The airway of the infant differs in ***five ways*** ***(Negus, 1995*)**.

1- The relatively large size of the infant‘s tongue in relation to the oropharynx increases the likelihood of airway obstruction and technical difficulties during laryngoscopy.

2- The larynx is located higher in the neck, thus making straight blades more useful than curved blades.

3- The epiglottis is shaped differently, being short and stubby and is angled over the laryngeal inlet.

4- The vocal cords are angled, so that a "blindly" passed endotracheal tube may easily lodge in the anterior commissure rather than slide into the trachea.

5- In the adult, an endotracheal tube that passes the vocal cords will readily pass into the trachea, because the glottic opening is the narrowest portion of the larynx (Figure 2).The infant larynx is funnel-shaped; the narrowest portion occurring at the cricoid cartilage so, an endotracheal tube that easily passes the vocal cords may be tight in the subglottic region because of the narrowing at the cricoid cartilage. For this reason, uncuffed endotracheal tubes are the preferred choice for patients younger than 10 years of age ***(Eckenhoff, 1981).***

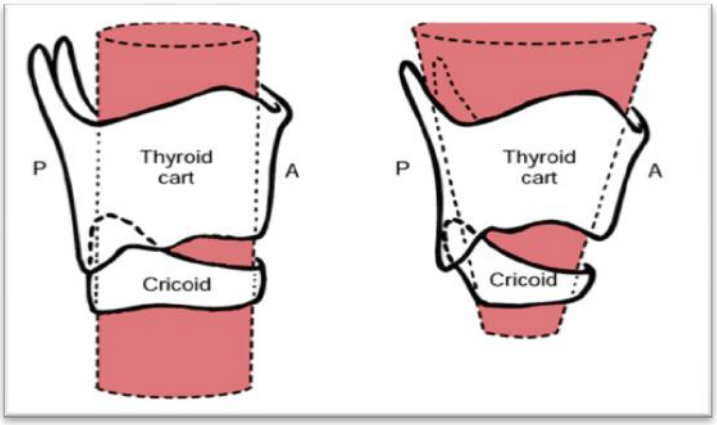

Figure: (2) Adult larynx (cylindrical shape) (left), infant larynx (funnel shape) (right) A, anterior; Cricoid, cricoid cartilage; P, posterior; thyroid cart, thyroid cartilage *(Coté and Todres, 1999)*

Airway obstruction is often attributed to occlusion of the oropharynx by the tongue. However, other studies indicate that obstruction may reside at other levels, such as the epiglottis or the soft palate ***(Reber et al.2001).***

Respiratory system

Several anatomic differences make respiration less efficient for the infant. The small diameter of the airways increases resistance to air flow; resistance is inversely proportional to the radius raised to the fourth power. The airway of the infant is highly compliant and poorly supported by surrounding structures. The chest wall is also highly compliant, so that the ribs provide little support for the lungs; that is, negative intrathoracic pressure is poorly maintained. Thus, each breath is accompanied by functional airway closure ***(Gann, 1996).***

One millimeter of edema will have a proportionately greater effect in children because of their smaller tracheal diameters ***(Morgan et al., 2006).***

Physiological consideration

Pediatric patients present unique physiologic consideration, so they deserve special care with respect to these differences from adults. Neonates are considered from (0–1 months), infants (1–12 months), toddlers (1–3 years), and small children (4–12 years of age) ***(Morray et al., 2000).***

Physiological differences between children and adults are important determinants when planning management of anesthesia in pediatric patients. Monitoring vital signs and organ function during the preoperative period is especially important, as neonates and infants have decreased physiologic reserves ***(Stoelting et al., 2002).***

Respiratory system

Control of respiration in newborn infants, especially premature neonates is poorly developed. The incidence of central apnea (defined as a cessation of respiration for 15 s or longer) is not uncommon in this group especially with a drug with a sedative effect. Unlike the adult, hypoxia in the neonate and small child appears to inhibit rather than stimulate respiration. The newborn has between 20 and 50 million

terminal air spaces. At 18 months of age, the adult level of 300 million is reached by a process of alveolar multiplication. Subsequent lung growth occurs by an increase in alveolar size. The lung volume in infants is disproportionately small in relation to body size. Their metabolic rate is nearly twice that of the adult, and therefore ventilatory requirement per unit lung volume is increased. Thus they have far less reserve for gas exchange ***(Aitkenhead et al., 2001).***

The high alveolar minute ventilation explains why induction and emergence from inhalation anesthesia are relatively rapid in small children. The high metabolic rate explains why desaturation occurs very rapidly in children. The ratio of physiological dead space to tidal volume *V*d/*V*t is similar to that of the adult at about 0.3. However; because the volumes are smaller, modest increase in *V*d produced by equipment such as humidification filters may have a disproportionately greater effect (Table1).Ventilation in small children is almost entirely diaphragmatic. Because the ribs are horizontal, there is no bucket handle movement of the ribs as occurs in the adult. It is therefore important to appreciate the normal minute ventilation is respiratory rate dependant. It is important to appreciate that the infant's response to hypoxemia can be bradypnoea and not tachypnoea as one would see in the adult ***(Aitkenhead et al., 2001).***

Table: (1) Respiratory variables in the neonate *(Aitkenhead, 2001)*

Tidal volume (*Vt*)	7 ml /kg
Dead space(*V*d)	(*V*t) x 0.3 ml
Respiratory rate	32 breath / min

In the premature infant, the work of breathing is approximately three times that of the adult, and this work can be increased significantly by cold stress or partial airway obstruction. These differences partially explain the high

respiratory rate of the infant and the rapidity with which hemoglobin desaturation can occur ***(Feliciano et al., 1991).***

Another important factor is the composition of diaphragmatic and intercostal muscles. These muscles do not achieve the adult configuration of type 1 muscle fibers until the child is approximately 2 years old (Figure 3) because type I muscle fibers provide the ability to perform repeated exercise and because the newborn and the infant are somewhat deficient in type I muscle fibers, any factor that increases the work of breathing contributes to the early fatigue of the respiratory muscles. Fatigue, in turn, leads to apnea or carbon dioxide retention and respiratory failure ***(Trunke, 1983).***

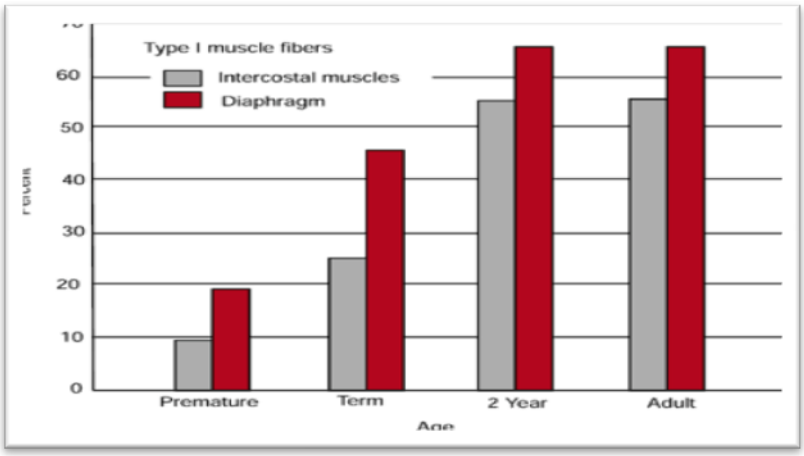

Figure: (3) The composition of the diaphragm and intercostal muscles changes markedly during the first 2 years of life *(Keens et al., 1978).*

Cardiovascular System

The myocardial structure of the heart, particularly the volume of cellular mass devoted to contractility, is significantly less developed in the neonate than in the adult. These differences and others produce a leftward displacement of the cardiac function curve and less compliant ventricles. This developmental myocardial immaturity accounts for the tendency toward biventricular failure, sensitivity to volume loading, poor tolerance to increased after load, and heart rate–dependent cardiac output ***(Kirkpatrick et al., 1996*).**

Stroke volume is relatively fixed by a noncompliant and poorly developed left ventricle in neonates and infants. The cardiac output is therefore very dependent on heart rate. Although basal heart rate is higher than in adults (Table2) activation of the parasympathetic nervous system, anesthetic overdose, or hypoxia can cause bradycardia and profound reductions in cardiac output ***(Morgan et al., 2006).***

Table: (2) Age-Related changes in vital signs *(Morgan, 2006).*

			Arterial Blood Pressure	
Age	Resp. Rate	Heart Rate	Systolic	Diastolic
Neonate	40	140	65	40
12 months	30	120	95	65
3 years	25	100	100	70
12 years	20	80	110	60

The process of growth demands a high metabolic rate. It should therefore come as no surprise that infants and children have a higher cardiac index (compared with adult) so that oxygen and nutrient can be delivered to actively growing tissues. The ventricles of neonates and infants are poorly compliant, so even though the ventricles of infants demonstrate the Frank-Starling mechanism, the main determinate of cardiac output is heart rate. Infants tolerate heart rates of 200 beats/ min with ease (Table3). Bradycardia may occur readily in response to hypoxemia and vagal stimulation and it represents a decrease in cardiac output. Immediate cessation of the stimulus and treatment with oxygen and atropine are absolutely crucial, a heart rate of 60 beats/ min in an infant is considered a cardiac arrest and requires cardiac massage. Arrhythmias are rare in the absence of cardiac disease. The usual cardiac arrest scenarios are electromechanical dissociation and asystole, not ventricular fibrillation ***(Aitkenhead et al., 2001).***

Table: (3) Variation in heart rates (beat/ min) with age *(Aitkenhead, 2001).*

Age	Mean value	Normal range
Neonate	140	100-180
1 year	120	80-150
2 years	110	80-130
6 years	100	70-120
12 years	80	60-100

Even though infants and children have a higher cardiac index, arterial pressure tends to be lower than in adults because of a reduced systemic vascular resistance associated with an abundance of vessel-rich tissues in the infant. The pressure increases from approximately 80/50 mmHg at birth to the normal adult value of 120/70 mmHg by the age of 16 years. Children under the age of 8 years who are normovolaemic at the start of anesthesia tend not to exhibit a decrease in arterial pressure when central neural blockade such as spinal anesthesia is administered. They do not require fluids pre-loading as an adult would to avoid hypotension, because venous pooling tends not to occur as venous capacitance cannot increase much. The reasons for this are that the sympathetic nervous system is less well developed and so infants tend to be venodilated at rest, and so vasoconstrictive responses to hemorrhage are less in neonates than adults ***(Aitkenhead et al., 2001).***

Caudal anesthesia neither altered heart rate (HR) nor mean arterial pressure (MAP). However, significant increases in descending aortic blood flow and in stroke volume were associated with a decrease in lower body vascular resistance. These results suggest that caudal anesthesia results in arterial vasodilatation in the anesthetized location. The local anesthetic-induced sympathetic block is the probable cause of this vasodilatation ***(Larouss et al., 2002).***

Renal function

Renal function is markedly diminished in the neonate because of low perfusion pressure and immature glomerular and tubular function. (Figure 4) Nearly complete maturation of glomerular filtration and tubular function occurs by approximately 20 weeks after birth, although maturation is somewhat delayed in premature infants. Complete maturation of renal function occurs by about 2 years of age ***(Jose et al., 1994).***

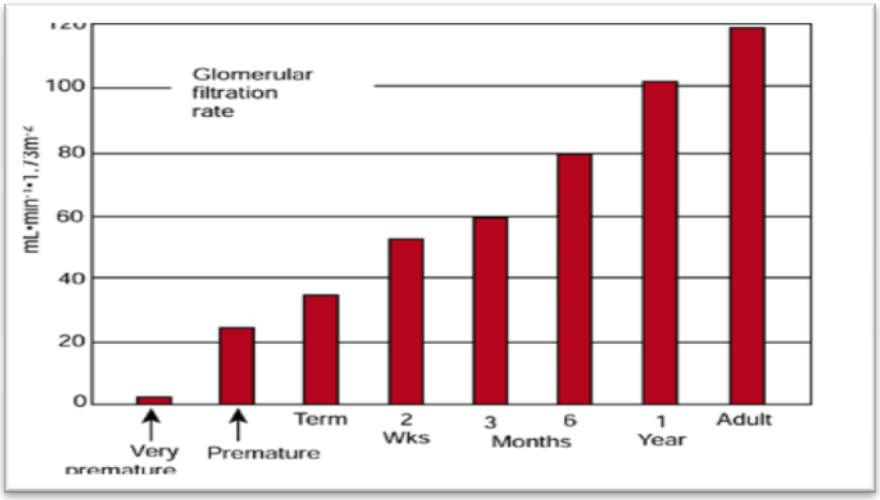

Figure: (4) Glomerular filtration rate *(Jose et al., 1994)*

Renal blood flow, glomerular filtration, and tubular secretion increase in the first weeks of life, approaching adult values by 8 to 12 months. Renal drug clearance may be particularly decreased in preterm neonates ***(Van den Anker, 1999).***

Kidneys are 80% to 90% mature by 1 month of age, before that time the infant cannot tolerate the extremes of renal stress. Neonates are obligate sodium losers therefore, exogenous sodium should be supplied. Also there is inability to handle excessive water and sodium loads. Over transfusion may lead to pulmonary edema and cardiac failure, and drugs eliminated by the kidney should be used cautiously ***(Yao, 2003).***