

Neuroanesthesia In Pediatrics

Essay

*Submitted for partial fulfillment of master degree
In Anesthesiology*

By

Ismail Mohammed Ibrahim Mahmoud

M.B.B.Ch, Faculty of Medicine, Ain Shams University.

Supervised by

Prof. Dr. Galal Adel Mohammed Elkady

*Professor of Anesthesia and Intensive care
Faculty of Medicine Ain Shams University*

Dr. Rasha Samir Abd El -Wahab Bondok

*Assistant professor of anesthesia & intensive care
Faculty of Medicine Ain Shams University*

Dr. Ashraf El-Sayed Elagamy

*Lecturer of anesthesia & intensive care
Faculty of Medicine Ain Shams University*

**Faculty of Medicine
Ain Shams University
2010**

List of contents

Acknowledgement	--
List of figures	--
List of tables	--
List of Abbreviation	--
Introduction	1
Differences between adult and children in neuroanesthetic management.....	3
Neuroanesthetic management in pediatrics	17
Anesthetic management of different neurosurgical operations	35
Postoperative care after neuroanesthesia for pediatric patients	61
Summary	84
References	86
Arabic summary	--

List of Figures

<i>Figure</i>	<i>Title</i>	<i>Page</i>
(1)	Age related differences in cerebral blood flow.	6
(2)	The effect of changes in PaCo ₂ &PO ₂ on cerebral blood flow.	7
(3)	Change in cerebral blood flow and cerebral metabolic rate of oxygen caused by intravenous anesthetics	8
(4)	The pressure volume curve & role of compensatory mechanisms	9
(5)	Developmental difference between adults and children in cricothyroid and tracheobroncheal tree	23
(6)	The sitting position affords optimal chest wall compliance in children with respiratory disease and obesity.	27
(7)	Prone infant. Lateral rolls are used to elevate the infant and minimize thoracic and abdominal pressure	27
(8)	Picture of a neonate with a myelomeningocele	58
(9)	A -shoulder support , B - proper positioning of arm, C -improper positioning of arm with >90° abduction	79

List of Tables

<i>Table</i>	<i>Title</i>	<i>Page</i>
(1)	Perioperative concerns for infants and children with neurological disease	18
(2)	Anesthetic effects on cerebral metabolism	22
(3)	various surgical positions &their physiological sequel	26
(4)	Predicting Shunt Failure on the Basis of Clinical Symptoms and Signs in Children	38
(5)	Glasgow coma score for adults and children	50
(6)	Average heart rate, systolic pressure, respiratory rate and blood volume values According to age	66
(7)	Peripheral nerve injury due to malpositioning of patients in Operating room.	77

List of Abbreviation

MAc	Minimal alveolar concentration
BBB	Blood Brian Barrier
BPM	Beat Per Minute
BPS	Systolic Blood Pressure
CBF	Cerebral blood flow
CMRO ₂	cerebral metabolic requirement for oxygen
CNS	Central Nervous System
CPP	Cerebral perfusion pressure
CRAO	Central retinal artery occlusion
CSF	cerebrospinal fluid
CVP	Central venous pressure
ECG	Electrocardiography
EEG	Electroencephalography
GCS	Glasgow Coma Score
HR	Heart Rate
ICp	Intracranial pressure
IM	Intramuscular
IV	Intravenous
MAP	Mean arterial pressure
MEP	motor evoked potential
NMDA	N-methyl D-aspartate receptors
PaCO ₂	Arterial carbon dioxide tension
PaCU	Postanesthetic Care Unit
PaO ₂	Arterial oxygen tension
peep	Positive end expiratory pressure
PICU	Pediatric Intensive Care Unit
PONV	Postoperative Nausea and Vomiting
POV	Postoperative Vomiting
RR	Respiratory Rate.
SBI	Secondary Brain Insult.
SjVO ₂	Jugular venous oxygen saturation.

List of Abbreviation (Cont.)

SSEP	Somatosensory Evoked potential.
TBI	Traumatic brain injury.
TCD	transcranial Doppler sonography
V/Q ratio	Ventilation /perfusion ratio
VAE	Venous air embolism
VPS	Ventriculo-peritoneal Shunt

Acknowledgements

First of all, all gratitude is due to **God** almighty for blessing this work, until it has reached its end, as a part of his generous help, throughout my life.

Really I can hardly find the words to express my gratitude to **Prof. Dr. Galal Adel Mohammed Elkady** Professor of anesthesia and intensive care, faculty of medicine, Ain Shams University, for his supervision, continuous help, encouragement throughout this work and tremendous effort he has done in the meticulous revision of the whole work. It is a great honor to work under his guidance and supervision.

I am also indebted to **Prof. Dr Rasha Samir Abd El-Wahab Bondok** Assistant professor of anesthesia and intensive care, faculty of medicine, Ain Shams University for her guidance, continuous assistance and sincere supervision of this work.

I would like also to express my sincere appreciation and gratitude to **Dr., Ashraf El-Sayed Elagamy** lecturer of anesthesia and intensive care, faculty of medicine, Ain Shams University, for his continuous directions and support throughout the whole work.

Last but not least, I dedicate this work to my family, whom without their sincere emotional support, pushing me forward this work would not have ever been completed.

Ismail Mohammed Ibrahim Mahmoud

Introduction

Pediatric neuroanesthesia can be seen as a specific branch of anesthesia half way in between pediatric anesthesia and neuroanesthesia. As a matter of fact, we must keep well in mind the peculiarities of the pediatric patient and the different pharmacodynamic and pharmacokinetic properties of the anesthetic drugs, particularly in neonates and infants (**Pietrini D, et al., 2003**).

The perioperative management of pediatric neurosurgical patients presents many challenges to neurosurgeons and anesthesiologists. Many conditions are unique to pediatrics. A basic understanding of age-dependent variables and the interaction of anesthetic and surgical procedures are essential in minimizing perioperative morbidity and mortality (**Soriano SG, et al., 2002**).

Recent advances in pediatric neurosurgery have drastically improved the outcome in infants and children afflicted with surgical lesions of the central nervous system (CNS). Because most of these techniques were first applied to adults, the physiologic and developmental differences that are inherent in pediatric patients present challenges to neurosurgeons and anesthesiologist (**Soriano SG, et al., 2007**).

Aim of the work

The purpose of this essay is to attain knowledge about the anesthetic management of pediatric neurosurgery patient and to choose and perform an appropriate anesthetic techniques, throughout surgical procedures and postoperative care.

Introduction about developmental considerations:

Age-dependent differences in cerebrovascular physiology and cranial bone development influence the approach to the pediatric neurosurgical patient. Cerebral blood flow is coupled tightly to metabolic demand, and both increase proportionally immediately after birth. Estimates from animal studies place the autoregulatory range of blood pressure in a normal newborn between 20 and 60 mmHg. This range is consistent with relatively low cerebral metabolic requirements and low blood pressure during the perinatal period. More importantly, the slope of the autoregulatory curve drops and rises significantly at the lower and upper limits of the curve, respectively. This narrow range, with sudden hypotension and hypertension at either end of the autoregulatory curve, places the neonate at risk for cerebral ischemia and intraventricular hemorrhage, respectively **(Pryds O, 1991)**.

Another developmental difference between adults and pediatric patients is the larger percentage of cardiac output that is directed to the brain, because the head of the infant and child accounts for a large percentage of the body surface area and blood volume. These factors place the infant at risk for significant hemodynamic instability during neurosurgical procedures. The infant cranial vault is also in a state of flux. Open fontanelles and cranial sutures lead to a compliant intracranial space. The mass effect of a tumor or hemorrhage are often masked by a compensatory increase in the intracranial volume through the fontanelles and sutures. As a result, infants presenting with signs and symptoms of intracranial hypertension have fairly advanced pathology **(Pryds O, 1991)**.

Neuroanatomy:-

▪ **Skull and Brain:**

The infant's 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 the age 2 to 3 months (anterior) and 7 to 19 months (posterior), open fontanelles allow for non invasive assessment of intra cranial 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, children with untreated progressive hydrocephalus can have very large heads (**Bisonette B, et al., 2002**).

▪ **Spinal cord:**

The anatomic position of the spinal cord is age related. In infants, the end of the spinal cord is at the level of L3 caudal to the adult position opposite the L1-2 disk. Spinal cord migration is hindered in children with a tethered cord who develop progressive neurologic deficits (bladder and bowel dysfunction and sensory loss) without surgical correction. A midline dimple over the spine above the gluteal fold may signal a tethered cord in the asymptomatic child and an increased risk of neurologic injury with regional anesthesia or diagnostic lumbar puncture (**Soriano SG et al., 2002**).

▪ **Cerebral blood flow and metabolism:**

Cerebral metabolism and cerebral blood flow (CBF) are age-related (40 and 100ml/100 gm brain tissue/min in neonates and children respectively), Global cerebral blood flow(indexed to weight of brain tissue) is lower in neonates than adults and higher in children than adults (Fig. 1). Similarly compared with adults, brain oxygen and glucose utilization are lower in neonates and higher in children. In neonates, autoregulation of CBF occurs at lower absolute pressures and over a narrow range of blood pressures. A linear relationship between the upper limit of autoregulation and postconceptual age was shown in a study of healthy neonates, with the upper limit between 45 and 60mmHg at 33 to 35 weeks postconceptual age and at 100mmHg at 47 weeks. Experimental evidence suggests that the mechanisms that regulate CBF and autoregulation also vary as a function of age (**Valvilala MS, et al., 2003**).

Factors that influence cerebral blood flow and intracranial pressure (ICP):-

A) Blood gases :

A high PCO₂ is the most potent stimulator of cerebral blood flow (C.B.F) (**RammohanN, 2007**).

While C.B.F is not affected until PO₂ decreases to about 50 mmHg, beyond this, cerebral vasodilation occurs. Hypoxia and hypercarbia produce a synergistic effect to produce a marked increase in C.B.F. and therefore cerebral blood volume as shown in (Fig. 2) (**Clayton T &Manara A, 2008**).

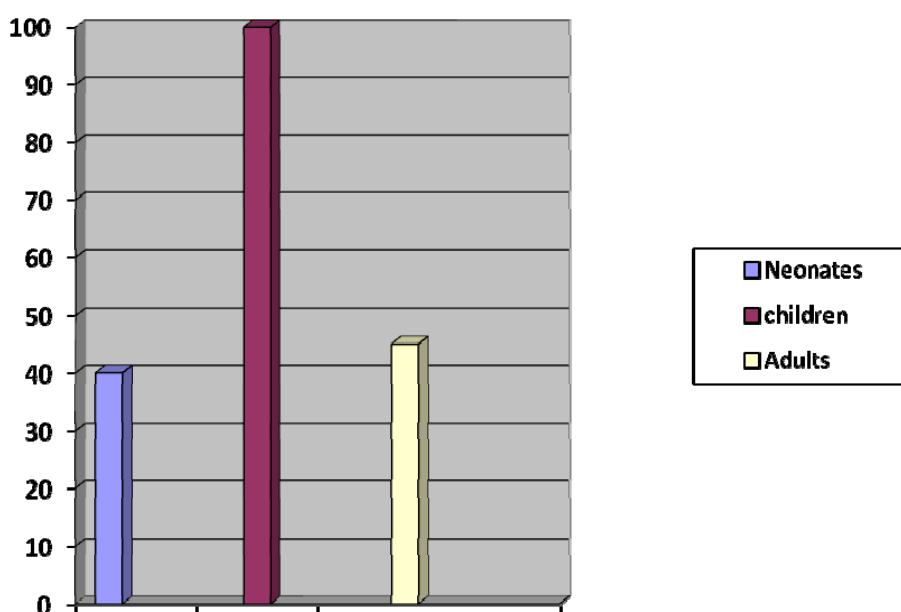


Fig.(1): Age related difference in global cerebral blood flow (ml/100g/min) (Faillace WJ, 2002).

B) Blood pressure :

Normally, cerebral autoregulation keeps flow steady despite variations in blood pressure. Sudden increase in blood pressure can raise C.B.F. The most common stimuli are laryngoscopy and intubation, suctioning and skeletal fixation of the head, which must be considered during perioperative visits. Wide fluctuations in blood pressure are poorly tolerated (Rammohan N, 2007).

C) Venous pressure:

Increase in central venous pressure is directly transmitted to the intracranial cavity and so increase the ICP. This can be harmful if there is:

- 1) Coughing and straining on the endotracheal tube.
- 2) Flexion of the neck, producing kinking of the neck veins.
- 3) A head position in which the head hangs too low

(Rammohan N, 2007).

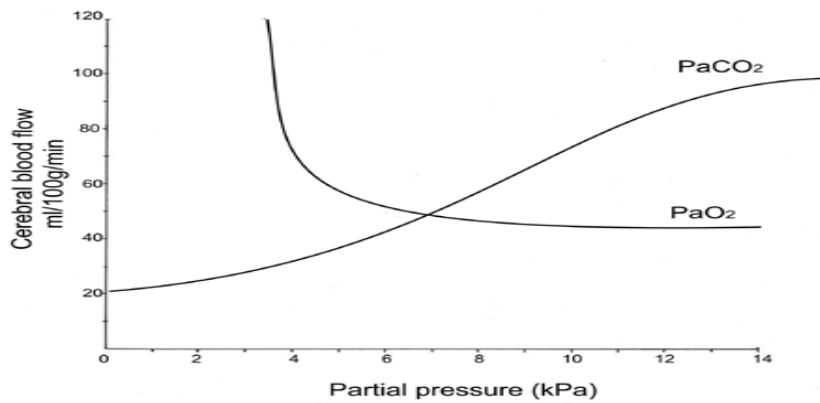


Fig. (2):The effect of changes in PaCO₂ and PaO₂ on cerebral blood flow (Clayton T & Manara A, 2008).

D) Anesthetic agents:

Both anesthetic techniques and agents greatly influence the I.C.P curve by their effect on intracranial blood volume, which is affected through change in blood flow (Reza G, 2007).

Most intravenous (IV) anesthetic agents reduce neuronal activity and so reduce the brain cerebral metabolic requirement for oxygen (CMRO₂) as shown in Fig. (3). They provide a protective mechanism when oxygen demand may outweigh supply (Stanley B & Norfolk M, 2008).

All inhalation agents are cerebral vasodilators causing increase in intracranial blood volume and ultimately I.C.P (Reza G, 2007).

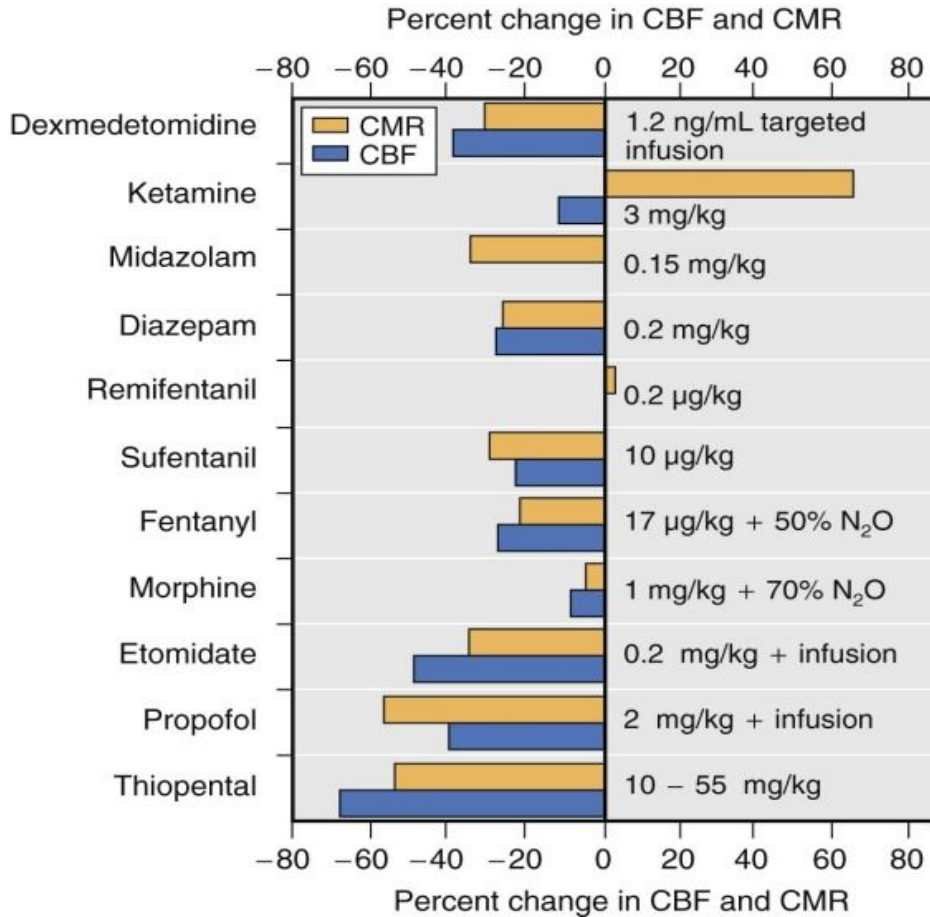


Fig.(3): Change in cerebral blood flow and cerebral metabolic rate of oxygen caused by intravenous anesthetics (**Drummond JC, et al, 2008**).

Pressure Volume Relationships :-

The cranial cavity is a semi-closed, non-distensible cavity containing brain and water (80%), blood (12%) and CSF (8%); as change in the volume of any one will require acute changes in the other two to avoid sudden shifts in pressure (**Reza G, 2007**).

Pathophysiological consequences of injury depend upon the speed of onset and efficiency of the compensatory