Anesthetic considerations and management in pediatric surgical emergencies

An Essay

Submitted for fulfillment of master degree in

Anesthesia

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2010

ABSTRACT

The commonly used anesthetic, muscle relaxants, narcotics and other drugs differ in their pharmacokinetics and pharmacodynamics in the neonates as compared to the older children and adult. These differences can be attributed to age related variations in lung volumes, alveolar ventilation, and cardiac function distribution of bloodflow.

KEY WORDES

Anesthetic

Considerations

pediatric

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ABBREVIATIONS

ABG	Arterial Blood Gases
APLS	Advanced pediatric life support
ARF	Acute renal failure
ASA	American society of anesthesiologists
ASD	Atrial septal defect
AVSD	Arterioventricular septal defect
BSA	Body surface area
CAM	Congenital adenomatoid malformations
CBC	Complete Blood Count
CBF	Cerebral Blood Flow
$CMRO_2$	Cerebral Metabolic Rate of Oxygen
CNS	Central Nerves System
CO	Cardiac output
CO2	Carbon dioxide
CPB	Cardio Pulmonary Bypass
CPP	Cerebral Perfusion Pressure
CSF	Cerebro Spinal Fluid
CT	Computed Tomography
CV	Cardiovascular
DA	Ductus arteriosus
DLT	Double lumen tube
ECF	Extra Cellular Fluid
ECG	Electrocardiogram
ECMO	Extracorporeal membrane oxygenation
EEG	Electroencephalogram.
EMLA	Eutectic mixture of local anaesthetic
ETCO2	End Expiratory CO2
ETT	Endotracheal tube
GCS	Glasgow Coma Score

GI	Gastro Intestinal
HB	Hemoglobin
Hct	Hematocrit Value
HPV	Hypoxic pulmonary vasoconstriction
HR	Heart Rate
ICP	Intracranial Pressure
ICU	Intensive care unit
IO	Intraosseous
IV	Intravenous
IVC	Inferior vena cava
L	Litre
MAC	Minimum alveolar concentration
MAP	Mean Arterial Pressure
mcg	Microgram
min	Minute/s
ml	Milliliter
mmHg	Millimeters of mercury
mmol	Millimols
NSAID	Non-steroidal anti-inflammatory
PA	Pulmonary artery
PDA	Patent ductus arteriosus
PaO_2	Arterial Partial pressure of Oxygen
$PaCO_2$	Arterial Partial Pressure of Carbon dioxide
RA	Right atrium
RSI	Rapid sequence induction
RV	Right ventricle
sec	Second/s
SV	Stroke volume
SLV	Single lung ventilation
SVC	Superior vena cava
US	Ultrasound
VSD	Ventricular septal defect

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Introduction

Emergency is a critical condition that requires rapid and correct management. It represents a difficult challenge for anesthesiologist as it provides only a short time for optimization and preparation, also emergency conditions frequently present outside daytime hours when medical staff levels are reduced. [1]

Emergency conditions generate a higher anesthetic risk, in adults as well as in children and strict management is necessary to ensure optimal conditions. The challenge in administrating anesthesia to a child and neonate as compared to adult is the immaturity of organ system and the resultant altered pharmacology and physiology. [2]

The successful anesthetic management depends on appreciation of anatomic, physiologic, and pharmacologic characteristics of each group. These characteristics which differentiate them from each other and from adult necessitates modification of anesthetic equipments and techniques. Indeed infants are at increased risk for anesthetic morbidity and mortality than older children; risk is generally inversely proportional to age, neonates being at highest risk .In addition, pediatric patient are prone to illness that require unique surgical and anesthetic strategies. [3]

The physiology of the preterm and term neonate is characterized by a high metabolic rate, limited pulmonary, cardiac and thermoregulatory reserves, and decreased renal function. Multisystem immaturity creates important developmental differences in drug handling and response when compared to the older child or adult. Neonatal anesthetic management requires an understanding of the pharmacophysiological limitations of the neonate as well as the pathophysiology of coexisting surgical disease. [4]

NEONATAL ANATOMY

Respiratory system:

There are many differences between adult and pediatric anatomy that affect mask ventilation and intubation. In the neonate and infant there is a proportionately larger head and tongue, narrow nasal passage ,an anterior and cephaled larynx (opposite a vertebral level of C4 versus C6 in adults),a long epiglottis and a short trachea and neck. These factors contribute to obligate infants to breathe nasally. The cricoid cartilage is the narrowest point of the airway in children younger than five years of age, as opposed to the glottis in adult. Therefore one millimeter of edema will have a proportionately greater effect in children because of their smaller tracheal diameters. A prominent occipit tends to place the head in a flexed position prior to intubation. This is easily corrected by slightly elevating the shoulders with towels and placing the head on a doughnut-shaped pillow. Compression of submandibular soft tissue should be avoided during mask ventilation to prevent upper airway obstruction. Straight laryngoscope blades aid intubation of the anterior larynx in the infants and young children. Endotracheal tubes that pass through the glottis may still impend on the cricoid cartilage, causing postoperative oedema, stridor, croup and airway obstruction. [5]

Uncuffed endotracheal tubes are usually selected for children under the age of ten years in order to reduce the risk of post- intubation croup and to provide a leak to minimize the risk of accidental barotraumas. The endotracheal tube inside diameter is initially estimated by a formula based on age: **(age/4)+4 =**tube diameter in mm, exceptions include premature (2.5-3mm) and full term neonates (3-3.5 mm), endotracheal tubes 0.5mm larger or smaller than predicted should be readily available. Correct tube size is confirmed by easy passage into the larynx and the development of gas leak at

10-25 cm H_2o pressure. No leak indicates an over sized tube that should be replaced to prevent postoperative laryngeal edema, while an excessive leak may preclude adequate ventilation and contaminate the operating room with anesthetic gases. Endotracheal length is estimated by the following formula **(age/2)+12 =** length of cut tube in cm. This formula provides only guide line and the result must be confirmed by auscultation and clinical judgment. To avoid endobronchial intubation, the tip of the endotracheal tube should pass only 1-2 cm beyond an infant's glottis. Several anatomic characteristics decrease the efficiency of respiration in neonates and infants including weak intercostal and diaphragmatic musculature due to a paucity of type I muscle fibers, horizontal and pliable ribs and protuberant abdomen. The paucity of small airway and alveoli is responsible for a high total resistance to airflow. [6]

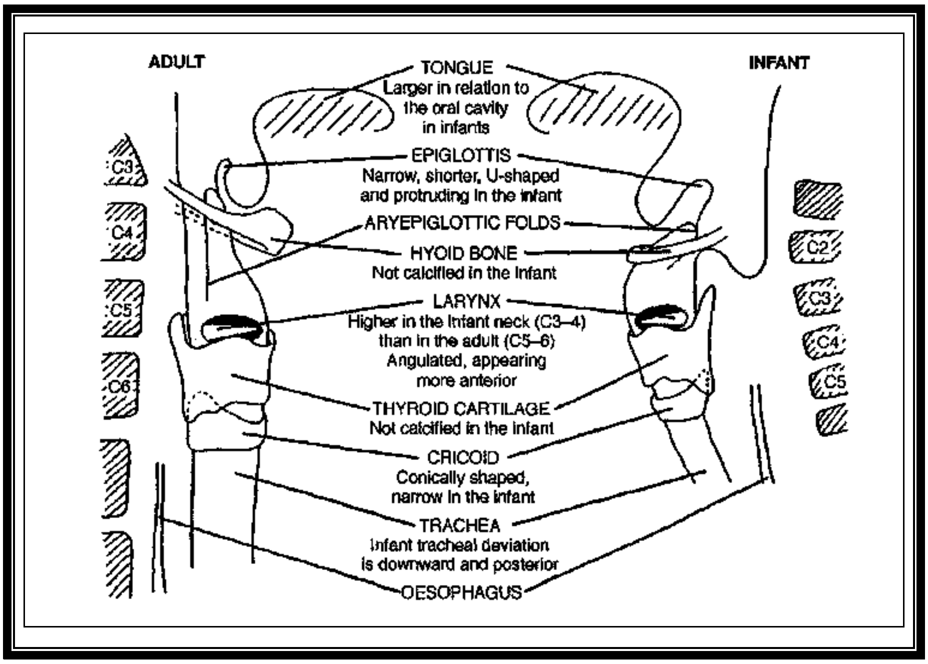

Figure (1) infant and adult upper respiratory tract [6]

Cardiovascular system:

The newborn infant is in a state of transition from the fetal to an extra uterine circulatory pattern. The fetal circulation is characterized by high pulmonary vascular resistance, low systemic vascular resistance (placenta), and right-to-left shunting via the foramen ovale and ductus arteriosus.

figure (2) shows right to left shunts. Aeration of the lungs at birth causes a rapid, progressive decrease in pulmonary vascular resistance and an increase in pulmonary blood flow. The decrease in pulmonary vascular resistance at birth is mediated by the endogenous production of nitric oxide. [7]

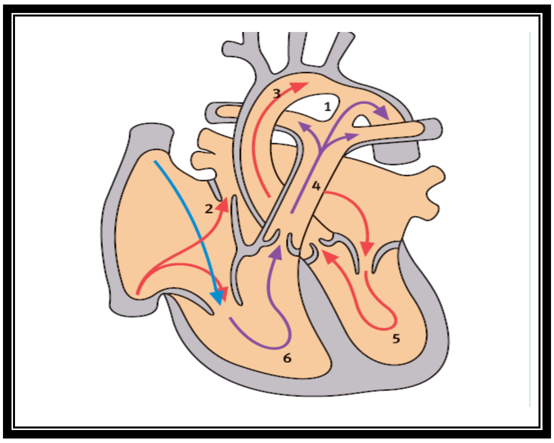

Figure (2) **Right-to-left shunts** [3]

1 Ductus arteriosus	**4 pulmonary artery**
2 Foramen ovale	**5 left ventricle**
3 Aorta	**6 right ventricle**

Increasing blood return to the heart via the pulmonary veins raises the pressure of the left atrium above that of the right, causing a functional closure of the foramen ovale. Anatomic closure of the foramen ovale usually occurs between 3 months and 1 year of age. The functional closure of the ductus arteriosus is, in part, mediated by an increase in arterial oxygen tension and is complete within the first 10 to 15 hours. However, anatomic closure does not occur until 2 months of age. Because the foramen ovale and ductus arteriosus are only functionally closed in the neonatal period, the circulatory pattern can readily revert to the fetal pattern. The neonatal pulmonary circulation is very reactive. Hypoxemia, hypercarbia or acidosis can cause pulmonary vasoconstriction and dilatation of the ductus arteriosus. Increases in pulmonary vascular resistance result in a right-to-left shunt across the foramen ovale and ductus arteriosus. Right-to-left shunting, by causing hypoxemia, causes a further increase in pulmonary vascular resistance, creating a vicious circle.[(8)]

The neonatal myocardium contains immature contractile elements and is less compliant than the adult myocardium. The Frank–Starling relationship is functional only within a very narrow range of left ventricular end diastolic pressure. Thus, there is a limited increase in cardiac output to be gained from aggressive volume loading in the normovolemic newborn. However, if preload is reduced, normalization of volume status will increase cardiac output. Because stroke volume cannot be increased by volume loading and contractile reserve is limited, neonatal cardiac output is exquisitely heart-rate dependent.[(9)]

To meet the elevated metabolic demand, neonatal cardiac output, relative to body weight, is twice that of the adult. This is achieved by a relatively rapid heart rate (140 beats per minute) because stroke volume cannot be significantly increased. Circulation in the neonate is characterized by centralization (increased peripheral vascular resistance and distribution of cardiac output primarily to vital organs), a situation comparable to an adult in compensated shock. Because neonatal baroreflex activity is impaired, the response to hemorrhage produces little increase in heart rate or change in total peripheral resistance. Thus, even a modest (10%) reduction in blood volume will cause a 15% to 30% decrease in mean blood pressure in the newborn infant. The structural and functional immaturity of the neonatal cardiovascular system severely limits the reserve that is available in the face of common perioperative insults such as hypovolemia, anesthetic-induced depression of contractility, and positive pressure ventilation induced decreases in venous return, 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 adults, activation of parasympathetic nervous system, anesthetic overdose, or hypoxia can cause bradycardia and profound reduction in cardiac output. Sick infant undergoing emergency or prolonged surgical procedures appear especially prone to episodes of bradycardia that can lead to hypotension, asystole and intraoperative death. The sympathetic nervous system and baroreceptor reflexes are not fully mature. The infant cardiovascular system maintains lower catecholamine stores and displays a blunted response to exogenous catecholamine. The vascular tree is less able to respond hypovolemia with vasoconstriction. The hallmark of intra vascular depletion in neonates and infants is therefore hypotension without tachycardia. [3]