PEDIATRIC ANAESTHESIA

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Dr Azam’s Notes in Anesthesiology

2nd Edition

Pediatric Anesthesia

By

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PREFACE

This book grew from notes first written in 2003 - 2004 for the students at the J J M Medical College in Davangere.

There are many textbooks to choose from when preparing for the “Anesthesiology examination”. The candidate suffers not from the lack of information but rather from being inundated with it. The candidate then has the task of information sorting and data compression to memorize and utilize all this information.

Graphic representation of data is an excellent form of data compression; figures or drawings are frequently asked about at the viva examination, particularly since the candidate’s understanding of a problem comes across most clearly when drawing a figure or a using a picture. Figures are also a good way of approaching a topic.

I constructed parts of Dr Azam's Notes in Anesthesiology for Postgraduate students when preparing for the Anesthesiology examination and later when preparing for tutorials.

Dr Azam's Notes is aimed primarily at trainees in Anesthesia though more experienced practitioners may find it useful as a refresher in recent concepts and advances

Dr Azam's Notes is not a substitute for the major anesthesiology text books but concentrates on principles of management of the most challenging anesthetic cases.

The format is designed to provide easy access to information presented in a concise manner. I have tried to eliminate all superfluous material. Selected important or controversial references are presented as well as suggestions for further reading. Some relate more to basic principles, physiology, pharmacology, etc. – bookwork. Others are more practical in nature, discussing the principles of anesthetic techniques for certain high-risk situations.

Dr Azam's Notes have been created keeping the Postgraduate needs while preparing for the exams, and also help in his day to day practice. I am sure that Dr Azam’s Notes will not only help him to secure highest marks but also help him to gain knowledge to its full.
A NOTE TO THE READER

Anesthesiology is an ever-changing field. Standard safety precautions must be followed, but as new research and clinical experience broaden our knowledge, changes in treatment and drug therapy may become necessary or appropriate. Readers are advised to check the most current product information provided by the manufacturer of each drug to be administered to verify the recommended dose, the method and duration of administration, and contraindications.

However, in view of the possibility of human error or changes in medical sciences, neither the author nor the publisher nor any other party who has been involved in the preparation or publication of this work warrants that the information contained herein is in every respect accurate or complete, and they disclaim all responsibility for any errors or omissions or for the results obtained from use of the information contained in this work. Readers are encouraged to confirm the information contained herein with other sources. It is the responsibility of the licensed prescriber, relying on experience and knowledge of the patient, to determine dosages and the best treatment for each individual patient. Neither the publisher nor the editor assumes any liability for any injury and/or damage to persons or property arising from this publication.
DEDICATION

To Mohammed Shafiulla, my father, my oxygen, companion, and best friend; for being my major pillar of support and making this vision a reality. Thank you for your continual sacrifices with boundless love and limitless gratitude, for the sake of your children. I owe you a debt I can never repay.

I also would like to thank my mom (Naaz Shafi), my wife (Roohi Azam), my two lovely kids (Falaq Zohaa & Mohammed Izaan), for their support, ideas, patience, and encouragement during the many hours of writing this book. My colleagues Dr Rajshekar Reddy & Dr Sachin for their support.

Finally, I would like to thank my teachers. The dream begins with a teacher who believes in you, who tugs and pushes and leads you to the next plateau, sometimes poking you with a sharp stick called "truth."
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Chapter 1 - ANATOMY AND PHYSIOLOGY

Classification by Age:

**Neonate:** 1-28 days of age  **Toddlers** – 2-3 yrs

**Infant:** 1-12 months of age  **Children** – 1-12 yrs

- Pediatric patients deserve special considerations with respect to anatomic, physiologic and pharmacologic differences from adults.
- Premature infants (gestational age less than 37 wks) and low birth weight infants (birth weight less than 2,500g) can have abnormal organogenesis / abnormal organ function / smaller organs / reduced muscle and fat mass.

1) **Body size:**
- Body surface area (BSA) is considered a better criteria in judging fluid and nutritional requirements.
- BSA at full term birth – average 0.2m²
- BSA in relation to weight → predisposes the infant to ↑ heat loss and insensible H₂O loss → hypothermia and dehydration.

2) **Airway anatomy:**
- Large head, short neck, narrow shoulders → neutral / slightly flexed position of head for intubation.
- Nasal passages narrow → smaller size of ETT.
- Large tongue in relation to oropharynx → difficulty in laryngoscopy / visualization of larynx / ↑ chance of airway obstruction.
- Epiglottis – short and stubby, hard and narrow folded into an ‘Ω’ omega or ‘V’ shaped → angled at 45° over laryngeal inlet → difficult to lift with the tip of a laryngoscope blade
- Vocal cords – angled forwards and downwards blindly passed ETT may lodge in the ant. Commissure. → flexion of head
- Larynx high – C₄ vertebrae – glottis higher and anterior than adults → straight blades more useful than curved blades.
- Narrowest portion – cricoid cartilage (adults-glottis) – completing
- Subglottic region easily damaged by large ETT
- Any oedema at this region ↓ airway diameter by as much as 60-70% of neonates.
- Uncuffed ETT used in children younger than 10 yrs.
- Trachea 4cm in length and 6-8 mm diameter → term infants
Bronchial intubation more likely
1. Chest relatively small in relation to abdomen.
2. Poorly developed body support (bone and muscle) with disproportion.
   o Difficulty in positioning
   o Sitting position – craniotomies – head secured safely as neck is weak for heavy head.
   o Prone position – shoulders need adequate support – rolls underneath both shoulder.

3) Respiratory physiology:
1. Ribs are more horizontal \(\rightarrow\) less A-P and lateral chest expansion.
2. Sternum and thoracic cage-soft and complaint \(\rightarrow\) -ve intrathoracic pressure poorly maintained.
3. Intercostal muscles weak.
   - Diaphragm is high and movement like piston \(\rightarrow\) abdominal distension – diaphragm splinting.
   - Diaphragm and intercostal muscle are deficient in type I – type II fibers are predominant.
   - Type II fibers – rapidly stimulated, easily fatigable, glycolysis metabolism \(\rightarrow\) any \(\uparrow\) work of breathing (WOB) – fatigue – apnea and CO\(_2\) retention – resp. failure.
   - Alveolar malnutrition incomplete until late childhood
   - And small alveoli are associated with low lung compliance
     \[
     C_L = \frac{\text{Change in lung volume}}{\text{Change in bbronchopulmonary pressure}}
     \]
   - Small diameter of airways - \(\uparrow\) resistance to airflow \(R \propto 1/r^4\) spontaneous ventilation not advised as \(\uparrow\) work of breathing – fatigue.
   - Tidal volume 6-8ml/kg and dead space to T.V – 0.3.
   - Alveolar ventilation – 150ml/kg/min (twice of adults) RR - \(\uparrow\) 35/min.
   - O\(_2\) consumption 5-6ml/kg/min – higher than adults
   - FRC 27-30 ml/kg – smaller than adults

4) Cardiovascular system:
- Birth and the initiation of spontaneous ventilation initiate circulatory changes, permitting neonates to survive in an extra uterine environment.
- Fetal circulation – high PVR, low SVR (placenta) and R \(\rightarrow\) L shunting through foramen ovale and ductus arteriosus.
At birth – placental removal from circulation – SVR ↑ rapidly exposure of ductus arteriosus to ↑O₂ conc. → ductus arteriosus closure.
- SVR - ↓PVR → ↑left side pressure → foramen ovale closure.
- Closure of ductus arteriosus: Functional closure 10-15 hrs after birth
- Anatomic closure 2-3 wks after (ligamentous Arteriosus)
- Closure of foramen ovale: Functional closure – at birth
- Anatomic closure – 6 wks
- Closure of ductus venosus: Functional closure – 3 to 7 days after birth
- Anatomic closure – 2-3 months of age (ligamentous Venousum)
- Transitional circulation – until true mechanical closure of ductus arteriosus at 2-3 wks of age adult circulation is not establishment.
  - Persistent fetal circulation / persistent Pulmonary HTN of newborn
  a. Diaphragmatic hernia
  b. Meconium aspiration
  c. Polycythemia.

Severe hypoxemia → intense pulmonary vasoconstriction → ↑PVR

Poorly oxygenated blood enter descending aorta

R → L shunt through DA

Pul. HTN

RA and RV HTN

R → L shunt through FO

a. Diagnosis – PaO₂ in blood samples obtained simultaneously from preductal (right radial) and post ductal (umbilical, post tibial, dorsalis pedis) arteries – PaO₂ diff more than 20mmHg.
- Cardiac output - HR dominant determinant, does not depend on contractility.
- Parasympathetic system fully developed and dominant → hypoxia, hypercarbia, hypovolemia, drug induced, laryngoscopy, intubation crying → bradycardia → ↓ C.O.
- Sympathetic N.S and baroreceptors – reflex not fully mature vasoconstrictor responses to hemorrhage and hypovolemia less.

5) Hematology:
- Blood volume → Preterm 90-105 ml/kg
- Full term neonate 80-90ml/kg
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- Hb - 19g% at 8-12 wks → 10-11g% at 2-3 months – physiologic anemia.
- Fetal Hb (HbF) - at birth HbF 80% HbA 20%
  - [HbF → P_{so} 18-20mmHg, ↓2,3-DPG, ↓affinity to 2,3-DPG - ↑O_{2} affinity
  - ODC – left → better O_{2} transport but poor O\_{2} delivery.
- By 5 months HbF ↓ to 5%, 2,3-DPG ↑ by about – 37%
- HbA ↑ → ODC – right
- Leukocyte function impaired – prone for infection.
- Platelet count (NL) – defective function
- Prothrombin time prolonged (liver immature and minimal placental transfer of
  vitamin K dep. factors)
- Corrected with vitamin K treatment IM preoperatively and at birth.

6) Renal:
- Nearly complete maturation of glomerular filtration and tubular function – 20
  wks (5 months) of age.
- Complete maturation of renal function – 2 yrs of age.
- Diminished renal function in neonate – low perfusion pressure and immature
  glomerular and tubular function.
- Newborn – GFR 21 ± 4 ml/min/BSA
  - 6mon – 1 yr – 77 ± 14 ml/min/BSA
  - Adults – 125 ± 15ml/min/BSA
- Half life of drugs excreted by glomerular filtration prolonged (pancuronium,
  digoxin, antibiotics).
- Lower renal threshold for glucose (glycosuria – osmotic diuresis)
  o avoid hyperglycemia
- Neonates obligate Na\^{+} loser and cannot conc. urine → exogenous Na\^{+} and H_{2}O
  during perioperative period.

7) Hepatic:
- At term → functional maturity incomplete
- Neonates – conjugation reactions impaired - ↓degradation reactions
  o long t \(\frac{1}{2}\) of drugs
- Neonates – plasma albumin and other proteins lower →
- Greater levels of free drug (thio ↓ dose), neonatal coagulopathy
- Minimal glycogen stores – prone for hypoglycemia (avoid prolonged fasting)
- Unable to handle large protein loads – prone for academia.
8) Body composition:
- Total body water content and ECF volume - ↑ proportionately in neonates ↓ with age (premature infant > term infant > 2yr old)
- ECF vol. – 40% of body weight in neonates (20% of adults)
- 18-24 months become same as adults.
- Fat and muscle content ↑ with age
  - Water sol. drugs – large Vd → higher initial doses
  - Drugs that redistribute to muscle – longer clinical effect e.g. Fentanyl
  - Drugs that redistribute to Fat for termination – longer clinical effect
  - Thiopentone

9) Nervous system:
- Brain – development of cells in cortex and brain stem incomplete till 1 yr
  Vulnerable to cerebral damage due to hypoxia, hypercarbia, ischemia, seizures and hemorrhage.
- BBB immature – hyperbilirubinemia – kernicterus
  - ↑ sensitivity to thiopentone – lower dose
  - ↑ Sensitivity to morphine – resp. depression
- Cerebral blood flow autoregulation impaired in prematures – hypoxia / hypercarbia
- CBF becomes pressure dependent.
  - BP – intra ventricular / intracranial hemorrhage
  - BP – ischemia
- Spinal cord extends upto L₃ – at birth L₁ at 1yr of age.
- SA / spinal tap – below L₃

ANS
a. Parasympathetic – fully developed at birth
b. Sympathetic – not fully developed until 4-6 months age

- Infants and newborns more prone for bradycardia (with hypoxia, hypovolemia, laryngoscopy, pharyngeal suctioning, drugs – halothane, scoline) – atropine

10) Thermoregulation:
- Infants and small children with their small size, ↑ BSA to body weight ratio, ↑↑ thermal conductance – body heat is lost more rapidly
- ↓ ability to produce heat
- Shivering is of little significance
Non-shivering / cellular thermogenesis – primary mechanism – mediated by brown fat.

Steps to ↓ loss of body heat taken to present hypothermia.

1. Body size – BSA 0.2m² average
2. Airway anatomy
3. CVS – fetal circulation, persistent fetal, cir, C.O
4. Respiratory – ribs, alveoli, diaphragm, I.C muscles, airway diameter, O₂ cons, FRC, Alveolar Ventilation
5. Renal – GFR, drugs
6. Hepatic – metabolism, glycogen, proteins
7. CNS – brain, BBB, CBF, spinal cord, ANS
8. Hematology – HbF, platelets, leukocytes
9. Body composition TBW, fat, muscle
10. Themoregulation
Chapter 2 - APPLIED ASPECTS OF ANATOMY AND PHYSIOLOGY OF RELEVANCE TO PEDIATRIC ANESTHESIA

Introduction:
Pediatric patients include neonates (less than 30 days of age), infants (1-12 months of age), and children (1-12 years of age) who are not merely small adults. Their successful and safe anesthetic management depends on an appreciation and clear understanding of the physiologic, anatomic, pharmacologic and psychological differences between each group and adults. These characteristics necessitate modification of anesthetic equipment and techniques. This discussion describes how the unique characteristic of Pediatric patients influences the safe conduct of anesthesia.

Classification according to gestational age:
- Pre-term infant – born <37 weeks gestation (<259 days)
- Moderately premature – 31 – 36 weeks gestation
- Severely premature – 24-30 weeks gestation
- Post term infant – born after 42 weeks gestation

Classification according to birth weight
- Low birth weight (LBW)-Birth weight <2500gms (regardless of duration of pregnancy).
- Very low birth weight – weight <1500gms.

Airway:
Airway in Pediatrics, unlike the adult airway is not a uniform entity, but encompasses a huge spectrum of assorted heterogenous entities. The difficulties of management of a normal airway in a neonate are very different and complex compared to the airway of a two year old and that of an adult. The neonatal airway is at the most difficult end of spectrum and as the infant grows into childhood the normal airway becomes easier to handle; but in many varieties of the abnormal airway, the difficulties may grow with the child. A thorough understanding of the normal airway, the mechanism of laryngoscopy and facilitation of tracheal intubation is mandatory.

Pediatric patients have a proportionately larger head and tongue, narrow nasal passages, and anterior and cephalad larynx (at a vertebral level of C3-C4), a long epiglottis and a short trachea and neck. These anatomic features make neonates and most young infants’ obligate nasal breathers until about 5 years of age. The cricoid cartilage (subglottis) is the narrowest point or the airway in children younger than 5 years of age. One millimeter of edema will have a proportionately greater effect in children because
of their smaller tracheal diameters. Also, due to the shorter length of trachea endobronchial intubation and accidental extubation are more common with head movement.

During laryngoscopy optimal head positioning displacement of tongue and soft tissues into mandibular space and pressure on the larynx are needed to achieve a straight line of vision between the eye and larynx. These are difficult to achieve in Pediatric airway. Certain modifications in the technique may aid in better visualization.

Due to the large occiput, a small pillow placed under the occiput (similar to adults), will flex the head on neck instead of extending if for “sniffing position”. Thus it is preferable to place a pad under the neck and shoulders, with a large ring under the occiput to stabilize the head to aid in optimum head positioning for laryngoscopy. The relatively large tongue can pose difficulty in being pushed into the “mandibular space”. It can also cause obstruction to ventilation. The visualization of larynx becomes further difficult in presence of abnormalities like Pierre Robin syndrome, Goldenhar syndrome, Cystic hygroma etc.
Respiratory system:
Independent life is not possible until gestational age is 2-26 weeks. Alveoli increase in number and size until the child is approximately 8 years old. Further growth is seen as an increase in the size of the alveoli and airways. At term, a full complement of surface active proteins help to maintain patency of the airway. In premature children respiratory failure (respiratory distress syndrome) is common due to deficiency of these surface active proteins.

Oxygen consumption in the neonate 7 mlkg⁻¹ min⁻¹ is almost twice that of adult value. This is seen as increased minute ventilation (200 mlkg⁻¹ min⁻¹) at puberty. As tidal volume remains constant at 7 mlkg⁻¹ throughout life, increased ventilation is brought about by an increase in respiratory rate; approximately 30/min at birth which progressively falls to adult values by adolescence.

FRC in young infants at complete relaxation (central apnea, under general anesthesia, use of muscle relaxants) decrease to mere 10-15% of TLC. This low FRC is substantially below the closing capacity and results in small airway closure, atelectasis, ventilation/perfusion imbalance and hemoglobin desaturation.

The small diameter of airways increase resistance to airflow. The airway of the infant is highly compliant and poorly supported by the surrounding structures. The chest wall is also highly compliant, so that the ribs provide little support for the lungs, thus the negative intra thoracic pressure is poorly maintained. Thus the work of breathing increases to approximately three times of the adult.

Another important factor is the composition of the diaphragmatic and intercostals muscles. Type I muscle fibers which are fatigue resistant and able to perform repeated exercise are deficient in newborn and infants. The adult configuration is reached only by approximately 2 years of age. Any factor increasing the work of breathing contributes to early fatigue of the respiratory muscles. This fatigue can lead to apnea or carbon-dioxide retention and respiratory failure.

Control of breathing:
Maturation of neuronal respiratory control is related to postconceptional age rather than postnatal age. Both hypoxic and hypercapnic ventilatory drives are not well developed in neonates and infants. Hypoxia and hypercapnic depress respiration in these patients. An immature respiratory control combined with increased susceptibility to fatigue of the respiratory muscles. May be responsible for the increased risk of postoperative apnea especially in preterm infants with gestational age less than 46 weeks.

The biphasic depression of ventilation during hypoxemia is due to central depression, rather than depression of peripheral chemo receptors.
Both fullterm and premature neonates breath irregularly. Periodic breathing i.e. rhythmic breathing interspread with a series of short apneic spells lasting less than 10 seconds without cyanosis or bradycardia, occurs both during REM and non REM sleep and even during wakefulness. Periodic breathing is about 80% in full term neonates, whereas it is nearly 1000% in premature neonates. The frequency of periodic breathing diminishes after 44 weeks postconception and with maturation during the first year of life.

Central apnea is the cessation of breathing activity lasting longer than 15 to 20 seconds or a shorter apnea associated with bradycardia (HR<100) cyanosis or pallor. The mechanism is not understood but may be related to an immature respiratory control mechanism. Central apnea is rare in full term neonates but occurs in the majority of premature infants.

**Postoperative apnea:**

Postoperative apnea is an important clinical issue in Pediatric anesthesia. Prematurely born infants, less than 44 weeks postconception, especially those with a history of apnea, are at high risk (20 to 40%) of developing postoperative apnea. Apnea on occur mostly within 12 hours postoperatively. There are a number of compounding factors associated with the development of postoperative apnea, such as the extent of surgery, anesthetic techniques, anemia and postoperative hypoxemia. High risk of postoperative apnea is associated in anemic infant (HCT <30%) regardless of postconceptional age. Both caffeine and theophylline are known to be effective in reducing the incidence of apnea in premature infants, strengthen muscle contractility and prevent fatigue and stimulate respiration. Apnea or hypoventilation unrelated to neuronal or central apnea can occur in infants and children of all ages who are predisposed to upper airway obstruction and may be exaggerated due to the residual depressant effect or anesthetics, opioids or sedatives.

**Cardiovascular system:**

The fetal circulation, well adapted to the hypoxic intrauterine mileu, differs from the postnatal counterpart in a number of significant ways.

At birth, the fetal circulation begins the transition to the postnatal type. With the first breath, the lungs become aerated and pulmonary vascular resistance falls, resulting in an increase in pulmonary blood flows. Left atrial pressure increases above right atrial pressure. Leading to closure of foramen ovale. Increased arterial oxygen tension causes constriction of ductus arteriosus. The ductus venosus and the umbilical arteries also constrict over several days.

Fetal hemoglobin (HbF) in the intrauterine life is beneficial is it allows oxygen extraction from the maternal hemoglobin even at relatively low venous oxygen tension.
This HbF is a disadvantage postnatal, as it impairs oxygen delivery to the tissues. Resting cardiac output is high in the neonate as compared to that of older child and adult. This allows the infant to meet oxygen demand, but ability of the newborn to further increase the cardiac output during stress is limited. Stroke volume is relatively fixed due to a noncompliant and poorly developed left ventricle in the neonate and infant. The contractile apparatus comprise only about 30% of the neonatal heart compared to 60% in the adult heart. Thus the immature ventricle is characterized physiologically by both poor compliance and reduced contractility.

Cardiac output at birth is 200 mlkg\(^{-1}\)min\(^{-1}\) which progressively decreases to 100 mlkg\(^{-1}\) min\(^{-1}\) by adolescence. Resting stroke volume remains fairly constant at about 1 mlkg\(^{-1}\). The increased cardiac output in younger patients being maintained by increase in the heart rate. Normal heart rate which is approximately 150 beats per minute in neonates, progressively decreases throughout childhood. Activation of parasympathetic nervous system, anesthetic overdose or hypoxia can cause bradycardia and profound reductions in cardiac output. The sympathetic nervous system and baroreceptor reflexes are not fully mature. The infant has reduced catecholamine stores and displays, blunted responses to exogenous catecholamines. Thus vasoconstriction in response the hypotension is less manifested and hypotension without tachycardia is the hallmark of intravascular fluid depletion in neonates and infants.

Systolic blood pressure which is around 80 mmHg at birth, progressively, increases to 120 mmHg at puberty, keeping pace with the perfusion demands as the child assumes sitting and standing positions. Diastolic blood pressure also increases associated with increase in myocardial mass and to ensure adequate coronary blood flow during diastole.

ECG findings in neonates and children are different from that of adult. The ECG changes with age reflect the development of the myocardium. Due to the right sided predominance of the fetal heart, the neonatal ECG Shows a marked right axis deviation (+30\(^{0}\) to + 180\(^{0}\)) compare to adults (-30\(^{0}\) to + 105\(^{0}\)). Also seen are tall ‘R’ waves in the right leads and deep ‘S’ waves in the left leads shorter QRS duration, shorter PR interval, T waves inverted toward the left.

Innocent murmurs are common in children and may be present in upto 80% of children. Innocent systolic murmurs include vibratory Stills murmur basal systolic ejection murmur, cardio respiratory murmur and murmur of physiologic peripheral pulmonary stenosis. Venous hum is continuous murmur heard throughout the cardiac cycle Murmurs heard only during diastole are pathologic. Other innocent heart sounds, present in childhood include carotid bruit and third heart sound.

Certain risk factors increase the likelihood of reversion from adult circulation to a fetal type of circulation, known as transitional circulation. Factors include hypoxia,
hypercarbia, anesthesia induced changes in peripheral vascular tone. When this reversal occurs pulmonary artery pressure increases to systemic levels. Blood gets diverted past the lungs via the patent foramen ovale, and opening of the ductus arteriosus, allowing blood to shunt at the ductus levels. A rapid downhill course may occur, causing severe hypoxia.

Certain risk factors increasing the likelihood of prolonged transitional circulation include prematurity, infection, acidosis, pulmonary diseases resulting in hypercarbia or hypoxemia, hypothermia and congenital heart disease. Thus care must be directed to keeping the infant warm, maintaining normal arterial oxygen and CO$_2$ tension, and minimizing anesthetic induced myocardial depression.

Developmental myocardial immaturity accounts for the tendency toward biventricular failure, sensitivity to volume loading poor tolerance top increased after load and heart rate dependant cardiac output.
Physiological differences between neonatal and adult myocardium

<table>
<thead>
<tr>
<th></th>
<th>Neonate</th>
<th>Adult</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cardiac output</strong></td>
<td>Heart rate dependant</td>
<td>Stroke volume and heart rate dependent</td>
</tr>
<tr>
<td><strong>Contractility</strong></td>
<td>Reduced</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>Starling response</strong></td>
<td>Limited</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>Compliance</strong></td>
<td>Reduced</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>After load compensation</strong></td>
<td>Limited</td>
<td>Effective</td>
</tr>
<tr>
<td><strong>Ventricular interdependence</strong></td>
<td>High</td>
<td>Relatively low</td>
</tr>
</tbody>
</table>

Age-related changes in vital signs.

<table>
<thead>
<tr>
<th>Age</th>
<th>Respiratory rate</th>
<th>Heart rate</th>
<th>Arterial blood pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Systolic</td>
</tr>
<tr>
<td>Neonate</td>
<td>40</td>
<td>140</td>
<td>65</td>
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<tr>
<td>12 months</td>
<td>30</td>
<td>120</td>
<td>95</td>
</tr>
<tr>
<td>3 years</td>
<td>25</td>
<td>100</td>
<td>100</td>
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<tr>
<td>12 years</td>
<td>20</td>
<td>80</td>
<td>110</td>
</tr>
</tbody>
</table>

**Blood:**

Blood volume is highest in new born averaging 86 mlkg\(^{-1}\) in term neonates and 106 mlkg\(^{-1}\) in premature and small for gestational age infants. Normal adult values are 70-80 mlkg\(^{-1}\).

In the neonates hemoglobin is primarily of the fetal type (HbF) which has a higher affinity for oxygen than adult hemoglobin. This combined with low 2,3-DPG levels, results in a left shift of the oxy-hemoglobin dissociation curve and poor oxygen delivery to the tissues. Hemoglobin levels which are at 16 – 19 gm/dl and high hematocrit of approximately 55 at birth progressively decrease, reaching the lowest values at about 2 months. This is due to a relatively hypoxic environment in utero, which stimulates the production of erythropoietin and red cell production. After birth, there is a sharp fall in erythropoietin activity due to the greater availability of oxygen. By 6 months of age, mean hemoglobin is 12.5 gm/dl\(^{-1}\) and maintained till 2 years of age. Thereafter there is gradual increase upto puberty.

**Renal function:**

The kidneys are immature at birth and both glomerular filtration and tubular function are reduced. The GFR is low in the newborn, rises sharply in the first 2 weeks of life, and reaches adult values by 2 years, when complete maturation of renal function
occurs. The ability to handle free water and solute loads is impaired and half life of drugs excreted through glomerular filtration will be prolonged.

Premature neonates often possess multiple renal defects, including decreased creatinine clearance, impaired sodium retention, glucose excretion, bicarbonate reabsorption and poor diluting and concentrating abilities. Thus, meticulous attention is in fluid administration.

Fluid balance:

At birth, total body water constitutes 80-85% of body weight, which decreases with increasing age to reach adult values of 65% by about 3 years of age. This reduction is due to a decrease in the extracellular fluid compartment which is about 45% at term and reaching adult values of 35% by 3 years of age.

| Table 1. Age related changes in TBW and its distribution |
|-------------|-------------|-------------|-------------|
|            | Preterm     | Term        | 1-3 years   | Adults      |
| TBW        | 85%         | 80%         | 65%         | 65%         |
| ECF        | 53%         | 45%         | 25%         | 25%         |
| ICF        | 30%         | 35%         | 40%         | 40%         |

Maintenance fluid requirements are related to metabolic rate. In general 1 ml fluid is required for every 1 Kcal expended. Requirements for sodium, potassium and chloride are usually quoted as 30, 20 and 20 mmol 1000 kcal\(^{-1}\). This requirement of both fluids and electrolytes can be met with infusion of a solution of 0.18 % NaCl, 4% dextrose and 20 mmol kCl/lit at a rate equal to caloric expenditure based on Holiday and Segar rule of 4:2:1. The rationale behind adding dextrose in Pediatric maintenance fluid is that though it provides only 20% of total calories required for a child <10 kgs, it is sufficient to prevent ketosis.

Perioperative fluid management is divided into three phases maintenance, deficit and replacement of losses. Maintenance fluid requirement can be managed as outlined above by Holiday and Segar rule. Fluid deficits are calculated and replaced based on duration of fasting, presence of associated conditions like fever, vomiting diaphore, sweating and particular disease state or surgical problem likely to affect fluid status (bowel obstruction, peritonitis etc).

Intraoperative losses are subdivided into third space loss and blood loss.

Third space losses surgical trauma, blunt trauma, infection and may surgical conditions are associated with the isotonic transfer of fluid from the ECF to a non functional interstitial compartment. This is called third space loss and is impossible to measure, and may be estimated by the extent of surgery and the clinical response to appropriate fluid replacement. The magnitude of third space loss is usually highest in
infants undergoing intra-abdominal procedures, and least in superficial surgery or neurosurgery and approximate ranges are:

- Intra-abdominal surgery: 6-10 mlkg\(^{-1}\)h\(^{-1}\).
- Intra-thoracic surgery: 4-7 mlkg\(^{-1}\)h\(^{-1}\).
- Eye surgery
- Neurosurgery
- Superficial surgery\(\left\{1.2\text{ mlkg}^{-1}\text{hr}^{-1}\right\}\)

The aim is to replace sequestered plasma volume and Ringer's lactate is an appropriate replacement fluid. The clinical response to appropriate replacement is a sustained and adequate blood pressure and heart rate adequate tissue perfusion and uterine output of 1-2 mlkg\(^{-1}\)h\(^{-1}\).

All blood loss in Pediatric patients requires replacement. The anesthesiologist should have a preoperative plan regarding blood loss replacement, based on the patient's preoperative condition, preoperative hematocrit and nature of surgery. The concept of an allowable blood loss (ABL) is a useful approach. Generally, a hematocrit of 28-30% is acceptable, although in neonates a value of 40% is more appropriate. In determining ABL, an estimate of blood volume (EBV) must be first made:

- Premature neonate – 90-100 mlkg\(^{-1}\).
- Term neonate – 80-90 mlkg\(^{-1}\).
- 3 months to 1 years - 75-80 mlkg\(^{-1}\).
- 3-6 years – 65-70 mlkg\(^{-1}\).

ABL is calculated using the formula:

\[
ABL = \text{Weight} \times \text{EBV} \times \frac{(\text{Ho} - \text{H1})}{\text{Ha}}
\]

\(\text{Ho}\) is the starting hematocrit
\(\text{H1}\) is the lowest acceptable hematocrit
\(\text{Ha}\) is the average hematocrit \(\frac{\text{Ho} + \text{H1}}{2}\)

Intraoperative blood loss replacement is done with Ringer's lactate 3 ml per 1ml of blood loss 1 ml of colloid solution for each ml of blood loss and 0.5 ml of red cell concentrates for each ml of blood loss.

**Central and autonomic nervous system:**

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 two years of life and is accompanied by rapid advances in motor function. These advances occur in a rostrocaudal fashion. Myelination is complete by 7 years of age.
Brain is solely dependent on glucose for its energy source as it is the only molecule capable of crossing the blood brain barrier (BBB). Despite the enormous glucose requirement (6.8 mg glucose 100 mg⁻¹ min⁻¹ in child versus 5.5 mg glucose /100mg/min in adult), brain does not store glucose and does not elaborate any glycogen. The brain glucose reserve only secures 3 minutes of energy supply, enough to maintain normal cerebral function.

Basic glucose consumption - 0.3 to 0.8 mmol 100g⁻¹min⁻¹.
Oxygen consumption (CMRO₂) - 3.5 ml O₂ 100 g⁻¹min⁻¹ (adults)
5.5 ml O₂ 100 g⁻¹min⁻¹ (children )

Increased O₂ consumption is related to the energy requirements of growth.

**Cerebral blood flow:**
- 50 ml 100 g⁻¹min⁻¹ [30-90 ml 100 g⁻¹min⁻¹] in adults
- 42-48 ml 100 g⁻¹min⁻¹- term neonates
- 90 ml 100 g⁻¹min⁻¹-4-6 months
- 110 ml 100 g⁻¹min⁻¹-3-4 years
- 78 ml 100 g⁻¹min⁻¹ - 9 years

Age related reduction in cerebral metabolism and CBF is mainly due to loss of synapses or a synapse or a drop in their activity rather than to a neuronal anatomic loss. Cerebral blood flow is matched to CMRO₂. Cerebral autoregulation is present and capable of rapid response in infants and young children.

The blood brain barrier (BBB) consists of tight junctions and is impermeable to electrolytes. This BBB is immature at birth, but develops rapidly in postnatal life. The less mature BBB permits the passage of larger, lipid soluble molecules and various drugs.

Sympathetic and parasympathetic functions do exist in neonates but do not mature until later in infancy. There is a predominance of parasympathetic response system.

New born also respond to noxious stimuli with facial grimaces as well as cardiovascular and metabolic stress responses, suggesting perception of pain. Thus, the anesthesiologist should aim at attenuating the stress response as well as to prevent the perception of pain by administering sufficient analgesia.

Spinal cord following retrogressive differentiation at birth, the spinal cord ends at the intervertebral level L₃. Reaching the adult level of L₁ to L₃ at the age of 8 years. During embryonic life, the spinal cord fills the spinal canal, but from the fetal period onwards, the growth of osseous structures exceeds that of neural structures, thus, the cord and dural sac terminates at progressively higher levels.
Due to lower termination of these essential structures, lower intervertebral approaches to the equidural and subarachnoid spaces are recommended in infants to avoid any inadvertent neurologic damage. Myelination begins in cervical neuromeres and progressively extends downwards and upward, but is not achieved until 12 years shorter distance between successive nodes of Ranvier favor penetration of local anesthetics and rapid onset of nerve blockade even with the use of diluted solutions.

**Thermoregulation:**

Temperature derangements are frequently associated with anesthesia, and transient dysfunction in the thermoregulatory system may lead to potentially serious complications. When the thermoregulatory system is affected by environment, drugs or illness, metabolic changes appear and may lead to significant organ dysfunction. During the perioperative period patients are at risk for developing thermoregulatory disturbances due to both anesthesia and surgery.

The infant is particularly vulnerable to hypothermia because of both large ratio of body surface area to weight and a limited ability to cope with stress. The premature infant is even more susceptible because of very thin skin and limited fat stores. Compensatory mechanisms to this may be shivering and non shivering (cellular metabolic) thermogenesis. Thermogenesis by shivering is minimally developed during the first three months of life, making non shivering thermogenesis (metabolism of brown fat) the principle method of heat production. This non shivering thermogenesis depends on:

a. Intact surface and central responses to the difference between the skin temperature and that of the environment.

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b. Intact sympathetic nerve endings to release the catecholamines that stimulate the catabolism of fat
c. The cardiopulmonary capacity to increase oxygen uptake and delivery and
d. The availability of fat stores to provide free fatty acids which undergo complete metabolism and generate calories.

Oxygen consumption also increases in direct relation to the increasing differences between skin temperature and environmental temperature as the difference exceed 4°C. Thus all steps should be undertaken to minimize heat loss. This can be achieved by various devices like; use of warming. This can be achieved by various devices like; use of warming mattresses, blankets, warm IV fluids and blood, warming mattresses, blankets, warm IV fluids and blood, warming and humidifying anesthetic gases, over head radiant heaters of incubators for transport, use of plastic wrap of decrease evaporative loss, warming of preparation solution, and increasing the operating room temperature.

**Gastrointestinal system:**

At birth, the functional maturity of the liver is incomplete. Most enzyme systems for drugs metabolism although developed, are not yet induced (stimulated) by the agents they metabolize. As the infant grows, the ability to metabolize drugs increases rapidly in two ways.

a. Hepatic blood flow increases and more drug is delivered to the liver and
b. The enzyme systems develop and are induced.

Conjugation reactions are often impaired in the neonates, resulting in jaundice, decreased degradation reaction leading to long drug half lives. Thus longer drug elimination half life is seen in the neonate, whereas infants and older children have shorter drug half life.

Also seen are minimal glycogen stores, inability to handle large protein loads, lower levels of plasma albumin and other drug binding proteins. These factors account for tendency to hypoglycemia and acidosis. The lower albumin levels contribute to neonatal coagulopathy, decreased drug binding and higher levels of free drug.

At birth, the gastric pH is alkalotic, and is gastric acid production increases it reaches normal adult values by the second day. In neonates, infants lower esophageal sphincter tone is decreased. Also the ability to co-ordinate swallowing with respiration is not fully matured till 4-5 months of age. These two factors increase the incidence of gastroesophageal reflux.

**Conclusion:**

Pediatric anesthesia involves perioperative and critical care of patients of all ages ranging from preterm infants to teenagers. The differences in physiological
characteristics makes anesthetic management different and extremely challenging for the anesthesiologist.

It is imperative to have a good knowledge of the anatomic and physiologic patient for conduct of safe anesthesia.
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Chapter 3 - PHARMACOKINETICS AND PHARMACODYNAMICS OF ANESTHETIC DRUGS IN PEDIATRICS

Introduction:
Pharmacokinetics and pharmacodynamics of drugs and inhalational agents used in anesthesia have been studied extensively. However, it is only recently that some of the flaws in the studies and the use of drugs have been noted. For example, we know that all inhalational agents potentiate the effects of nondepolarizing muscle relaxants, but it must be remembered that this effect is both age and time dependent. Firstly, infants and young children more rapidly establish maximum potentiation than older children do and secondly this potentiation reaches maximum levels only within 1-2 hours. Therefore, pediatric studies related to potency, onset time or maintenance requirement of muscle relaxant are often difficult to compare as the duration of inhalational anesthesia may have varied considerably and is often not even described. Another problem in analysis which may arise is that monitoring of neuromuscular response by accelerography producers results that may vary from those of electro and mechanomyography in comparative studies. The extrapolation of data from adult studies without considering the drug effects on children is another consideration. For example, the initial use of prolonged infusions of aminoacid local anesthetics in the epidural space led to a number of cases of seizures and cardiac arrest. Subsequent pharmacokinetic studies have now helped formulate maximum safe infusion rates for infants and children.
Pharmacokinetics of drugs in children:

The dual processes of pharmacokinetics and pharmacodynamics of drugs administered to patients in general is illustrated in the figure 1.

Absorption of drugs:

Different modes are used to administer drugs to children. The most common of these involve extravascular routes preoperatively and postoperatively, and intravenously in the operation theatre or ICU.

Oral: The efficacy of orally administered drugs depends on the rate and extent of absorption from the gastrointestinal tract (mainly the small intestine), physicochemical nature of the drug, nature of gastrointestinal juices, rate of gastrointestinal emptying and gut blood flow. Several of these factors are affected in the neonate. The gastric pH, which is 6 to 8 at birth, decreases to 1 to 2 within 24 hours and finally reaches adult levels between 6 months and 3 years of age. Decreased basal acid output and total volume of gastric secretions is seen in the neonate. Bile acid secretion being less in the neonate may reduce the absorption of lipid soluble drugs. The rate of gastric emptying varies during the neonatal may reduce the absorption of lipid soluble
drugs. The rate of gastric emptying varies during the neonatal period, but can be markedly increased in the first week of life. Long chain fatty acids (as found in certain neonatal formulae) can delay gastric emptying, and this must be remembered while determining the fasting status of neonates appearing for surgery. Processes of both passive and active transport are fully mature in infants by approximately 4 months of age. Intestinal enzymatic changes in the neonate such as low activity levels of cytochrome P-450 1A1 (CYPIA1) can alter the bioavailability of drugs. Disadvantages of the oral route include, emesis, destruction of the drug by digestive enzymes or their metabolism prior to absorption, presence of food or other drugs, which cause irregularities in absorption and first pass hepatic effect.

Oral transmucosal drug or nasal administration: This route of administration of drugs bypasses the first pass hepatic effect and causes a rapid onset of drug action e.g. Sublingual nitroglycerine and nasal midazolam and ketamine.

**Parenteral:**
The rate of systemic absorption of drugs after IM administration is more rapid predictable than after or rectal administration due to high density of skeletal muscle capillaries in infants than older children. Reduced skeletal muscle blood flow and inefficient muscular contractions (responsible for drug dispersion) can theoretically reduce the rate of IM absorption of drugs in neonates. Drugs injected intravenously act almost immediately. However some drug may be lost because it is adsorbed to the glass or plastic infusion system. Its effects may be delayed if the infusion rate is slow. This can lead to an incorrect conclusion about the patient’s need for more or less drug.

Premedicant drugs such as morphine, pentobarbital or atropine do not alter the volume of gastric juice but glycopyrrolate does reduce the volume of gastric juice by a third and increased the pH of 68% gastric samples to above 2.5.

**Transdermal:**
This method provides sustained therapeutic plasma drug concentrations and presently used drugs in this method include fentanyl clonidine, nitroglycerine and EMLA. Enhanced percutaneous absorption of drugs in infancy is due to the presence of a thinner stratum corneum in the preterm neonate and greater extent of cutaneous perfusion and hydration of the epidermis throughout childhood. Neonates have a large ratio of body surface area to body mass. There is a potential for drug overdose in neonates by this route.

**Rectal:**
Drugs are given rectally to avoid some of the problems of orally administered drugs. This route should be avoided in immunosuppressed patients or those undergoing
Chemotherapy. Drugs administered in the anal canal below the ano-rectal or dentate line bypass the liver after absorption, while inserted above this line by absorption via the superior rectal vein undergo first pass hepatic metabolism. Absorption of rectally administered drugs is thus slow and erratic and also depends on whether the drugs are given in the form of suppositories, rectal capsules or enemas. Premedicant drugs used per rectum include thiopentone, methohexital, diazepam, atropine, and acetaminophen.

**Intrapulmonary:**
This mode of administration if increasingly being used in infants and children e.g., surfactant and adrenaline. Though the goal is to achieve a predominantly local effect systemic exposure does occur. Developmental changes in the architecture of the lung and its ventilatory capacity (e.g., Minute ventilation, vital capacity and respiratory rate) can alter pattern of drug deposition and hence systemic absorption after intrapulmonary administration of drug.

Irrespective of the route of administration of drug (intravenous or inhalation), the expected anesthetic effect occurs only when the concentration of the drug at the receptor site reaches the target concentration to produce anesthesia.

**Uptake and Distribution (of drugs other than inhalational agents):**
Removal of a drug from the site of administration and distribution to the effectors site depends on cardiac output, tissue perfusion and blood tissue partition coefficient of the drug. Age dependent changes influence the apparent volume of distribution (Vd) of drugs. The relatively larger extracellular and total body water spaces in neonates and infants compared to adults, coupled with adipose tissue that has a higher ratio of water to lipid, result in lower plasma concentrations of these drugs.

Fetal albumin has a lower binding affinity and capacity for drugs like weak acids (salicylates). Substances like free fatty acids, bilirubin, sulfa and maternal steroids can displace a drug from albumin binding site and increase free fraction of drug in the neonate. Serum albumin concentrations reach adult levels y 5 months of age. However, albumin only accounts for a small fraction of drug binding. Another protein of significance which binds drugs is α1-acid glycoprotein, reduced amounts of which is probably responsible for a significant proportion of unbound drug in infants. Drugs like diazepam, propranolol and lignocaine are less highly bound to α1-acid-glycoprotein in children than in adults.

A reduction in the quantity of total plasma proteins (including albumin) in the neonate increase the free fraction of drug. This decreased binding to proteins, coupled with an incompletely developed blood brain barrier can lead to accumulation of drugs like barbiturates and morphine in the CNS of neonates.
Metabolism:

Development of phase I and phase II enzymes: phase I reactions (oxidation, reduction and hydrolysis) are cytochrome p-450 dependent. The activity of many cytochrome p-450 isoforms including CYP3A4, CYP2C, and CYP1A2 is markedly decreased in the first 2 months of life. The clearance of intravenously administered midazolam from plasma is primarily a function of hepatic CYP3A4 and CYP3A5 activity and the level of activity increases during the first 3 months of life. Most phase I enzymes function at adult levels by 6 months of life. It is seen that some phase II pathways (e.g. Sulfonation) or mature at birth while others (e.g. Glucuronidation) are not. All phase II enzymes mature by 1 year of age. Phase II reactions involve conjugation with acetate, glycine, sulfate and glucuronic acid. Individual isoforms of glucuronosyl transferase (UGT0 have unique maturational profiles. Levels of UGT2B7 (responsible for glucuronidation of morphine) are markedly diminished in the first 2 months of life. Glucuronidation of acetaminophen (a substrate for UGT1A6) and salicylates is decreased in newborns. A compensatory pathway (glycine pathway) for metabolism of salicylates makes their elimination half life only slightly longer in neonates. Both phase I and phase II reactions can be induced by barbiturates. The rate of drug metabolism is also determined by other factors such as intrinsic rate of the process, hepatic blood flow etc.

Excretion:

The neonatal kidney receives only 5-6% of cardiac output compared to adults who receive 20-25 of cardiac output. Term neonates have a full complement of glomeruli while preterm neonates do not have a full number of glomeruli. The glomerular filtration rate (GFR) is approximately 2-4 ml per 1.73 m² in term neonates. This increases rapidly over the first 2 weeks of life and reaches adult values by 8-12 months of age. Tubular secretion is immature at birth and reaches adult values during the first year of life. Renal drug clearance is also affected by the renal extraction ratio and by glomerular pore size. A slightly acidic urine at birth (pH 6-6.5) decreases the elimination of weak acids. If the kidney is the primary route of drug elimination, the neonate’s reduced renal function can delay the drug elimination and clinicians must individualize therapy in an age appropriate fashion.
Pharmacokinetics of drugs during their absorption, distribution, metabolism and excretion

Absorption
- Age dependent changes in structure and function of GIT. Affect oral absorption.
- First pass hepatic metabolism is a problem that is avoided in oral transmucosal drug administration.
- Skin thickness is similar in infants and adults, but extent of perfusion and hydration diminishes from infancy to adulthood.
- Rectal absorption of drugs is erratic and first pass hepatic metabolism occurs in drugs administered above ano-rectal line.

DISTRIBUTION
- Age dependent changes in body composition, influence the apparent volume of distribution of drugs.
- In the first 6 months of life infants have an expanded total body water and extracellular water, expressed as a percentage of total body weight, as compared with older infants and adults.
- Reduced levels of fetal albumin and acid-glycoprotein contribute to the increased free fraction of drug.

METABOLISM
1. The activity of many cytochrome P-450 (CYP) isoforms and a single glucuronosyl transferase isoform is markedly decreased during the first 2 months of life.
2. The acquisition of adult activity over time is enzyme and isoform specific.
3. Compensatory pathways can help in drug metabolism.
4. Both phase I and phase II metabolic reactions mature by 1 year of age.

Excretion
The processes of glomerular filtration and active tubular secretion approximate adult activity by 6 to 12 months of age.
Individual drugs

Intravenous induction agents:

Thiopentone-
Thiopentone requirements for induction of anesthesia reveal an inverse relation with age. A significantly larger volume of distribution in the infant, makes the ED-50 of thiopentone in infants significantly greater (7 mgkg⁻¹) than that in adults (4 mgkg⁻¹). This larger dose increases the store of drug in the body and prolongs the drug’s half-life. In neonates due to their low body fat and muscle content, less thiopentone is apportioned to these tissues; so concentration in CNS may remain high and delay awakening.

Benzodiazepines
The premature and the mature infant at term eliminate diazepam at a slower rate than adults do. Differences in metabolism as described earlier alter may way in which neonates and infants reduce the plasma concentration of drugs. Neonates hydroxylate and N-demethylate diazepam less well than adults or children do which prolongs the elimination half-life of diazepam (75 ± 38 hours in preterm infants as compared to 18 ± 3 hours in children) and thus prolongs its effect.

Ketamine
In infants less than 3 months of age. The Vd is similar to that in older infants but the elimination half-life is prolonged. Hence, clearance is reduced in the younger infants. Reduced metabolism and renal excretion in the young infant are the likely causes. The pharmacokinetic details of ketamine are outlined in the table 1.

<table>
<thead>
<tr>
<th>Table – 1: Pharmacokinetic of ketamine: effect of age</th>
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<tbody>
<tr>
<td><strong>Age</strong></td>
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<tr>
<td>&lt;3 month</td>
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<tr>
<td>4 - 12 month</td>
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<tr>
<td>4 year</td>
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<tr>
<td>Adult</td>
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T₁/₂: Elimination half-life; VdSS: volume distribution at steady state; Cl: Clearance.

Propofol – Propofol has been used in the induction and maintenance of general anesthesia in children. In a study done by Murat et al in 12 children aged between 1 and 3 years given a single dose of propofol 4 mgkg⁻¹ an average vd of 9.5 ± 3.7 Lkg⁻¹ with an average total body clearance of 53 ± 13 mlmin⁻¹kg⁻¹ was reported. Younger children demonstrated a larger Vd with a similar rate of clearance.
**Narcotics:**

Morphine - Studies have demonstrated that morphine depresses the respiratory centre of newborns more than does pethidine. When the brain uptake index (BUI) for morphine was determined in developing rates, it was higher in the younger than the older rates. Pharmacokinetic studies of morphine also show that infants less than 1 week of age demonstrate longer elimination half-life compared to older infants.

Pethidine - Though pethidine is more lipid soluble than morphine there is reduced CNS uptake and sensitivity to pethidine. It produces only 1/10 the respiratory depression and less sedation than morphine. The activity of pethidine may be less because the opioid receptors of the brain are more primitive and do not recognize structural analogues.

Fentanyl - In the neonate, fentanyl clearance seems comparable to that of the older child or the adult, while in the premature infant fentanyl clearance is markedly reduced.

**Neuromuscular blocking drugs (NMBD):**

Clinically most neuromuscular blocking drugs are studied under anesthesia as the children are first anaesthetized and then the drugs given. Infants have a much greater potentiation and reduction in dose requirements than older children (sevoflurane decreases the dose requirement of non depolarizing muscle relaxants by 70% if administered for 90 min in school age children and 40 min in infants by prolonging their duration of action.

Depolarizing muscle relaxants - On a weight basis more succinylcholine is needed in infant than in older children or adults. Succinylcholine is rapidly distributed throughout the ECF because of its relatively small molecular size. The blood volume and ECF volume in infants are significantly greater than that of a child or adult on a weight basis. Therefore, the recommended dose is twice that of adults (2 mgkg⁻¹). The rate of succinylcholine hydrolysis may be slower in the preterm infant than in the older child due to their immature liver.

Non depolarizing muscle relaxants (NDMR) – There is substantial evidence to suggest that the neuromuscular junction in neonates is three times more sensitive to NDMR's than that of adults. However this sensitively is balanced by an almost identical increase in the volume of distribution (because of large ECF) so the required dose is unaffected. However, because of a prolonged elimination time, doses of additional relaxants should be reduced and given less frequently. Children of all ages are more resistant than adults to pancuronium.

Anticholinesterases – Neuromuscular blockade in children is antagonized much faster and by much smaller doses of anticholinesterases as compared to adults. Both cholinesterase and pseudocholinesterase levels are reduced in premature and term
newborns. Adult levels are not reached until 1 year of age. Inspite of reduced pseudocholinesterase levels, Newborns are more resistant to succinylcholine than adults are.

**Local anesthetic agents:**

Local anesthetics are used in children as topical application or subcutaneous injection for needle procedures, neuraxial blockade and peripheral nerve blockade. Uptake into the nerves from perineural injection sites competes with uptake into the central circulation. Direct measurement of intra-neural concentration of radiolabelled anesthetics in animal models indicate that less than 2-3% of an injected dose ever enters the nerve and within 30 minutes of injection more than 90% of an injected dose is taken up into the systemic circulation. This is significant as all the amino amides including bupivacaine, levobupivacaine, lignocaine and ropivacaine show diminished clearance in neonates with maturation over the first 3-8 months of age. Limited information suggests that the amino esters, which are metabolized by plasma esterases, have a very rapid clearance even in neonates.

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**Fig. 3a**

**Fig. 3b**: Fixed dose (mg) independent of age or body size

**Fig. 3c**: Weight scaled dosing (constant mgkg⁻¹)
Most drugs in pediatric dosing are usually on a weight scaled basis. However, it has been found that neonates have a shorter block duration and require a much larger (four fold) weight scaled dose to achieve a similar dermatomal levels when given a subarachnoid block or achieve a similar peripheral nerve block as an adult. This is not only due to higher weight scaled volume of CSF but there may also be age related differences in pharmacodynamic response, myelination, spacing of nodes of Ranvier, tissue barrier and other factors. Another important factor is the dependence of minimal blocking concentration on the length of nerve exposed to the local anesthetic. The minimal blocking concentrations on the length of nerve exposed to the local anesthetic is increased as shown in figure 3a. Hence the absolute dose of local anesthetic required to block a nerve should depend on the length of nerve exposed to drug and should be only weakly dependent on body size (figure 3b). Conversely if adult an infant nerves receive the same weight scaled dose (figure 3c), other factors being equal, the adult nerve will have a longer duration nerve blockade than the infant nerve.

The implication of the above include the fact that the therapeutic index of local anesthetics in infants may be so narrow that maximum safe infusion rates of the amino amides are too low to provide sole analgesia for most major surgery of the thorax, abdomen or pelvis. Thus, infants require higher weight scaled infusion rates than adults to achieve blockade but they can safely receive only a lower weight scaled infusion rate than adults from the viewpoint of toxicity. To provide adequate safe analgesia other agents such as opioids, clonidine or ketamine may be used in the epidural space to provide synergistic analgesia. Another approach is to use single stereoisomer like ropivacaine and levobupivacaine to decrease the likelihood of cardiac toxicity.

Inhalational agents:

The use of inhalational anesthetics in children has been the mainstay of anesthetic practice for the last 150 years. The potency of an inhaled anesthetic is determined by its minimum alveolar concentration (MAC). The requirement of inhalational agents varies inversely with age. MAC is lower with preterm infants than in term infants and increases with post conceptual age. Age related changes in MAC imply that the same alveolar concentration will produce different levels of anesthesia in children of different ages.

Factors affecting $F_E/F_I$ that is, the ratio between the end tidal anesthetic concentration and the inspired anesthetic concentration, which is a measure of how rapidly gas equilibrates between lung and tissue, are inspired anesthetic concentration, blood gas partition coefficient and cardiac output. Pediatric considerations of these will be discussed in detail.

$F_I$ or inspired anesthetic concentration – The higher the anesthetic concentration the more quickly $F_E$ moves toward $F_I$.  

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Changes in ventilation and FRC – The greater the minute ventilation, as in infants and children, the more rapid the rise occurs (assuming a constant cardiac output). The smaller the FRC the faster the $F_E/F_I$ increases. The FRC in infants is smaller than that of adults but tidal volume per kg body weight is the same as that of adults. The more rapid respiratory rate, though, increases the minute ventilation in the young.

Right to left shunting of blood – Which is often present in neonates and infants, slows induction of anesthesia because in them the blood concentration of anesthetics rises more slowly.

Blood/gas partition coefficients and anesthetic solubility – The $F_E/F_I$ of an insoluble gas like sevoflurane or nitrous oxide rises rapidly while with a more soluble gas like halothane it rises more slowly. In general, however, inhaled anesthetic agents are less soluble in the blood of Pediatric patients. For example, the blood/gas partition coefficients of halothane and isoflurane are 18% lower in neonates than in young adults (20-40%) and in children (1-7 years) are 12% less than in young adults. This difference, which accounts for the more rapid rise of $F_E/F_I$ in neonates, is due in part to lower albumin concentration.

During anesthesia, the blood gas partition coefficient can drop by approximately 10% due to haemodilution with crystalloid and reduction in hematocrit.

Blood tissue partition – Anesthetic solubility in brain, heart and liver increase by approximately 50% between the newborn period and middle age and is probably due to a decrease in water and an increase in lipid content with age.

Partial pressure of anesthetics – Because the blood flow per unit tissue mass is greater in neonates the level of anesthetic in the tissue increases more rapidly and the induction of anesthesia in more rapid.

Increased cardiac output reduces the rate of rise of alveolar concentration of anesthetic because more anesthetic is removed per unit of time. The cardiac output of neonates per kilogram is normally twice that of adults. However $F_E/F_I$ rises more rapidly because much of the cardiac output of neonates and infants directed to the vessel rich tissues (VRG), which then get saturated sooner.

**Specifics:**

The CNS – Drugs can penetrate the CNS of neonates more easily than that of adults either because the neonatal blood brain barrier is more permeable or because cerebral blood flow is slower and the drugs have a longer time to dissociate from plasma proteins. The blood brain barrier in neonates is also more easily disrupted by hypoxia and acidosis than it is in adults.

The cardiovascular system – The incidence of bradycardia, hypotension and cardiac arrest during induction is higher in infants and small children than in adults. This has been attributed to an increased sensitivity of the CVS to potent agents.
Induction characteristics – The most commonly used induction agent in pediatrics is halothane and more recently, sevoflurane. The primary criterion to assure a rapid induction is the ‘wash in’ curve in first minute or two of anesthesia. During the first couple of minutes of an inhalational induction, the alveolar-to-inspired concentration ratio of these potent inhaled anesthetics reaches to about 0.33. When the wash in of halothane and sevoflurane were compared in the first few minutes 5% inspired halothane achieved an alveolar concentration of 1.65% or 1.65 MAC, whereas 8% sevoflurane achieved a concentration of 2.64, just in excess of 1 MAC. Although both anesthetics provide for a rapid loss of eyelash reflex (1/3 faster with 8% sevoflurane than 5% halothane with single breath induction the depth of anesthesia achieved with sevoflurane is less than that of halothane because of the reduced MAC multiple in sevoflurane. It is also suggested that if sevoflurane is introduced slowly as is the practice with halothane a protracted excitement phase is seen before and adequate depth of anesthesia is achieved. Lerman therefore suggests based on the above observations, an increase to an inspired concentration of 8% sevoflurane as quickly as possible.

Metabolism: Inhalational agents are apparently metabolized by Pediatric patients to a lesser degree than adults. Infants can biotransform halothane but do so to a lesser extent than adults. Cited as further evidence for this is the low incidence of halothane induced hepatitis in the Pediatric age group despite repeated doses of halothane.

5% of inhaled sevoflurane is metabolized in vivo producing increased levels of fluoride. However, the limited metabolism of sevoflurane within the kidney does not provide sufficient fluoride to inhibit tubular reabsorption to any extent. This together with its rapid washout explains the lack of nephrotoxicity with sevoflurane compared with methoxyflurane. Other degradation compounds have been seen in vitro, but are not of significance. What is important, though, are the isolated reports of extreme heat and flammability occurring in breathing circuits using desiccated carbon-dioxide absorbent.

Emergence delirium is common with inhalational agents, more so with sevoflurane than with halothane. Pain is a confounding factor in the study of emergence delirium and the lack of understanding ED is the lack of a specific tool to assess it. A recently developed scale may help clarify this.
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Chapter 4 - PREANESTHETIC EVALUATION AND PREMEDICATION IN PEDIATRICS

Introduction:
The outcome of any anesthetic is determined by how well prepared the anesthesiologist is, to handle that particular patient. The pre operative visit paves the way for medical and the equally important psychological aspect of preparation.

Factors governing the Psychological response of a child:
Psychological preparation and premedication are much needed in neonates and infants. They are comfortable with anybody who handles them gently since communication is more by handling than verbal. Sedatives may increase the risk of apnoea and interfere with early resumption of feeds. However it must be understood that analgesia for any painful procedure is mandatory as it is indisputable that neonates do feel pain and it adverse effects.

Infants more than 6 months do resent separation from parents and it is advisable to either have the parent hold the child for “stealing with inhalational induction”. The angle piece without the mask is held away from the face to create a cloud of anesthetic (simple nitrous oxide and/or halothane in oxygen) or a nipple from which the infant sucks. The parental touch and voice soothes the body while he goes to sleep. Without this individualized attention, infants and toddlers 6 m-5 years, may develop post hospitalization regressive behaviour, fear of separation, clinging to mother in presence of strangers, screaming on entering a closed room, poor sleeping, feeding nightmares, bedwetting, loss of toilet training etc.

They dread injections and should be reassured that they will receive most medications orally till they go to sleep. They should be told about use of EMLA cream if at all injections become necessary.

To summarize, children of all ages need careful, affectionate consideration at this difficult time in their life. With a little effort, anesthesiologists can start this process in the preoperative visit.

Medical preparation:
A careful history and examination will reveal the presence of:
1. Airway problems (anatomical, physiological, allergic, asthma etc.)
2. Convulsions, sleeping disturbances (obstructive sleep apnoea, particularly in children coming for tonsillectomy).
3. Cardiorespiratory problems.
4. Urinary and bowel problems

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5. Hematological problems like thalassaemia major and minor, clotting factor deficiencies. Sickle cell disease etc., seen in some of the tribals and communities. They require a preoperative transfusion to raise their Haemoglobin above 10 gdl, or platelets etc., and specific avoidance of hypovolemia, hypotension and hypoxia.

6. Inborn errors of metabolism or deficiencies may run in families or communities, like pseudocholinesterase deficiency in vysya community.

7. Medications that cause alterations in physiology and interact with anesthetic drugs particularly relevant are corticosteroids, cardiorespiratory medications (β stimulants, ophthalmic drugs), anticonvulsants, and chemotherapeutic agents which cause serious compromise like bone marrow depression, pulmonary fibrosis (bleomycin), cardiac (adriamycin) or renal (cisplatinum) side effects.

Specific problems that require special preparation:

Child with runny nose: It is one clinical situation where the previous experiences of the anesthesiologist and lack of clarity in the literature cloud the issue. A sudden severe laryngospasm in a chubby child who becomes blue and bradycardia with alarming rapidity can demoralize a young anesthesiologist intern lifetime of nervous indecision, postponing every child with runny nose. Conversely, previous uneventful anesthetics in children with apparent cold may embolden one to fool hardly accept a child with a significant URI.

There is clearly a higher incidence of respiratory complications in children with URI. Once a child has contacted URI there is an increased incidence of adverse airway related events during the recovery period, of 6-8 weeks it takes for the irritability of the respiratory mucosa to diminish. Canceling and rescheduling the surgery after 2 weeks does not eliminate the increased frequency of problems. Thus a child who is postponed will be safe to anaesthetize only six weeks later. This kind of rigid protocol is essential in surgeries where the airway is shared between surgeon and anesthesiologist (cleft lip and palate corrections, thyroglossal cyst excision or tonsillectomies). Head neck surgeries where the access to airway for emergency intubation is restricted by surgical drapes or personnel, necessitates a planned endotracheal intubation e.g. Biopsy of lymph node from neck, torticollis, brachial cyst etc. are relatively contraindicated in the absence of a senior Pediatric anesthesiologist even if there is a slightest suspicion of URI. A sudden onset of runny nose or cough or change in voice at home (before crying at a hospital environment starts) crankiness, irritability, a bout of fever (>39°C) and reduction in activity or playfulness are definite indication of either the prodrome or actual infection. An observant intelligent mother would be the ideal person to give this information.
However it becomes impractical to postpone every suspected URI and reschedule them 6 weeks later. There is a gray area where ambiguity exists between a suspected and significant URI. One of the confusing issues is to distinguish between a runny nose and hoarse voice of prolonged crying or allergic rhinitis and a frank URI. With the rising vehicular and industrial pollution, chronic dry cough or a runny nose at a specific time of the day or season is not uncommon. Such a history in bouncy playful child, who has been investigated to be normal, is unlikely to be URI.

Benefit of doubt and judicious anesthesia may be given to a clear rhinitis with a dry cough or a child is recovering from it. Obviously this concession is only for short procedures which are unlikely to lead to significant homeostatic disturbances (e.g. Surface surgery on distal extremities amenable to regional anesthesia). The choice of anesthesia is such that the GA is given only to place the regional anesthetic block. Once the block starts acting GA is tapered off to a minimum. Elective herniotomy done to avoid an emergency surgery for obstruction is a special consideration in this context.

The anesthesia has to be light enough for a daycare admission but deep enough to allow handling of the peritoneal sac requiring analgesia from T4 to L3 level. This aim is possible with a brief anesthesia for a few minutes to place a 2 level hernia block, superficial and deep to the internal oblique muscle. Ilioinguinal nerve block (between the external oblique aponeurosis and internal oblique muscle at the neck of the sac. Awake spinal anesthesia with EMLA also achieves this goal. Caudal epidural may or may not, depending on the level of spread of LA in the neuraxis. A coughing fit at the time of tying the peritoneal sac can make surgery impossible and lead to significant desaturation. In such a patient all care should be taken to avoid irritation of the airway. In case problems develop they can be dealt with in a planned manner. Continuous oxygen to maintain optimal baseline oxygen in plasma and haemoglobin, β-agonist and/or inhalation agent kept ready to relieve bronchospasm. Coughing fit is best left alone unless desaturation is significant. In that case as with laryngospasm, it is found that a small bolus of propofol to be useful as it suppresses laryngeal reflexes. Refractory laryngospasm not resolving with CPAP ventilation alone may need succinylcholine and IPPV. Endotracheal intubation in such a case is best avoided as it only helps the immediate ventilation but postpones the problem to the post extubation period. Mask ventilation is preferable and lignocaine throat spray can be used to avoid further irritation. The child should be allowed to breathe without undue assistance as he is coming out of the effects of scoline. All these complications are disturbing but not life threatening provided one is prepared with drugs and equipment ready and has the requisite experience to handle the situation confidently.

It is essential to balance the risk of complications against pressure from surgeons, about the need to proceed with elective surgeries. Discretion in planning the
type of airway manipulation like ETT or LMA, and the ability to manage any ensuring complications calmly and competently can only come from objective learning and experience.

Reactive airway disease (Asthma). It is quite simple to manage with non irritant inhalational agents by a careful anesthesiologist. An asthmatic child can be safely anesthetized after ensuring that there is no active or brewing URI and that he receives his regular bronchodilators on the day of surgery. It is advisable to give him a puff prior to induction. Use of LMA reduces lower airway manipulation. When necessary, intubation as well as extubation has to be done in a deeper plane of anesthesia.

Difficult airway: Patients with major airway problems like Treacher Collins, Pierre Robins, Midface hypoplasia and TM joint ankylosis may present for corrective surgery. Other complex syndromes like Freeman Sheldon, Aperts, Beckwith Wideman syndrome etc., may present for other surgeries but their airway problems need to be addressed. Muscular dystrophies may present for orthopedic procedures or muscle biopsy. Since it is impossible to know all the congenital problems, references and patient’s papers should be scrutinized for the anatomical and physiological ramifications of the syndrome and it implications. A child with previous uneventful anesthetic may prove to be presently impossible to intubate, because the skeletal and soft tissue abnormalities grow with the patient.

The assessment of the airway will include assessment of mandibular space; i.e assessing how many fingers can be inserted between the mandible and hyoid. Normally 2-3 adult fingers can be accommodate. Any reduction should alert the examiner to a compromised laryngoscope view.

Any difficulty with laryngoscope and intubations is because of a reduction of the space available inside the mouth for introduction of the laryngoscope, or increase in the soft tissues of the floor or the mouth that have to be compressed to form the line of vision to the larynx. The space into which the soft tissues of the tongue are compressed by the laryngoscope is called the mandibular space, the incomplete bony ring formed by the rami of mandible in front and hyoid bone behind. Any reduction of this space by micrognathia and retrognathia will make laryngoscopy difficult. Conversely any increase in the bulk of soft tissues in the mouth as in lymphangioma, hemangioma or mucopolysaccharides diseases can also make intubations extremely difficult. Mucopolysaccharides disease deserve a special mention because they have a high failure rate of intubations even in the hands of experienced consultants. They usually present for minor problems like hernia but have to be approached with extreme caution and preparation. Regional techniques may fail because of abnormal chemical depositions in the nervous system.
Another very simple investigation is lateral X-ray of head and neck to outline the air shadow of the upper airway from the mouth, oropharynx down to the trachea. A well taken X-ray will delineate the air shadow, the epiglottis and any obstructions well.

Different types of laryngoscopes, LMA's, fiberoptic laryngoscope, airway catheters (Cooka), cricothyrotomy and tracheostomy should be planned and to be kept ready in such cases.

Patients with major cardiovascular problems. Unlike adults cardiovascular problems are uncommon other than congenital heart disease. The rare exceptions are Pheochromocytoma and adrenal hyper or hypoplasia, deposition of mucopolysaccharides in coronary arteries causing ischemic heart disease.

These require a meticulous planning of anesthesia, of cardiovascular drugs and electrolytes for medical optimization of the patient before the surgical handling of these physiologically labile patients.

In general soft systolic variable murmurs are usually innocent unless associated with symptoms. Loud, constant transmitted murmurs and diastolic murmurs are likely to have structure defects. History of breathlessness on exertion, inability to run and play in older children, and reduction of general activity point to a compromise. After corrective surgeries in infancy residual problems like pulmonary hypertension may persist. Cardiology consultation and a 2D echocardiograph will solve the dilemma about the significance of any murmur, need for prophylactic antibiotics facilitates the best plan of management for children with complex hemodynamics.

An impeccable perioperative analgesic management is mandatory as it avoidance of stress and emotional disturbances which can provoke or worsen the dreaded complication of pulmonary hypertension. It is important to reiterate that good rapport and gentle handling is vital in children with a psychological baggage from previous surgery.

Ex-premature baby – With improved perinatal care, the number of NICU graduates coming for herniotomy or cataract is rising. They require special care for certain vulnerabilities like apnea, retinopathy of prematurity, bronchopulmonary dysphasia etc. Apnoea was defined as that lasting > 15 secs or that < 15 secs but associated with a bradycardia of <80 beats/min\(^{-1}\). The risk of apnoea was found to be inversely related to both gestational age as well as postconceptual age. E.g. Body born at 30 weeks had a higher risk of apnoea than the body born at 35 weeks though both were of same postconceptual age of 50 weeks. Conversely if both were born at 26 weeks gestational age, one who was 50 weeks of postconceptual age at surgery had a higher risk than another of 55 wks. Another major risk factor was anemia. They concluded that all preterm infants less than 56 weeks postconceptual age were at risk, especially those
with obvious apnoea in the recovery room, and all those ex-preterm infants with anemia should be admitted and monitored with oximeter as well as apnoea monitors. The incidence of apnoea may be reduced by use of theophylline or caffeine or use of regional anesthesia unsupplemented by sedatives or other agents. Ketamine in particular, seems to markedly increase the propensity towards apnoea. However regional anesthesia can itself be associated with life threatening complications. So a balanced view should be presented to the parents at the pre-anesthetic visit and anesthesia preparation and management has to exemplary in this difficult subgroup of infants.

Post operative nausea and vomiting. Parents should be told that strabismus repair, tonsillectomy and middle ear reconstruction are associated with an increased risk of post operative nausea and vomiting. Use of opioids, pentazocine and tramadol increases this tendency. Prophylactic preoperative use of serotonin inhibitors like ondansetron or granisetron reduces this incidence. A single preoperative dose of dexamethasone has been used in tonsillectomy. Treatment of established nausea and vomiting is more difficult but metoclopramide either preoperatively or later does help in emptying the stomach and reducing the vomiting.

Pre anesthetic laboratory testing: Even though institutions abroad have eschewed routine haemoglobin estimation and urinalysis, it is still necessary in our country with parasitic infestations, malnutrition etc, if a clinical examination warrants it. It is particularly indicated in infants below 6 months with physiological anemia in ex prematures are prone to iatrogenic anemia from repeated investigations and resultant apnoea risk. Special investigations may be warranted by the preoperative medical problems eg., electrolytes in chronic diarrhea vomiting, full coagulation profile.

Special pre anesthetic preparation: Advances in Pediatric anesthesia have made surgeries like scoliosis correction, transplants, excision of hepatomas etc., possible. These may require special techniques like induced hypotension, hypothermia, acute normovolemic haemodilution and preoperative autologous blood collection with intermittent erythropoietin injections. These procedures require a significant coordination between the family, anesthesiologist, hematologist and other specialties for immaculate preoperative planning and execution.

Pre-anesthetic fasting:
Instruction to the family regarding the solid and fluid intake and the type of fluid is essential. Clear liquids (normal saline, apple juice, sugar water) are rapidly emptied from the stomach (halt life about 15 mins). So a limit of about 2 hours for these is reasonable. Milk, either from a formula or breast milk or cow’s milk is considered to be a solid as it curdles in the stomach, and should be restricted for 4 hours before anesthesia in infants below 6 months. The fat content which determines its gastric
emptying time of the breast milk depends on the maternal diet and this is the reason why it is now considered in the same category as other types of milk. In children more than 6 months, all solids, and milk is restricted for 6 hours before surgery. But they can have unlimited clear fluids for 3 hours before. This liberal approach avoids irritability excessive hunger and acid accumulation in the stomach with resultant problems of aspiration. Special care should be taken to adhere to strict like burns, septicemia, those on enteral or parenteral nutrition for debilitating disorders, insulinomas etc. These children are highly dependent on continuous adequate nutrition. Their IV intake has to be increased to avoid significant hypoglycemia or dehydration even with routine starvation, which would be uneventful in a normal child.

**Child with full stomach:**
There are very few conditions which warrant immediate surgery irrespective of starvation status. Usually even after prokinetic agents like metoclopramide. It is preferable to wait for 6 hours. Exceptions may be unstable foreign body in the airway, torsion testis, volvulus, trauma with haemorrhage, or intestinal obstruction where integrity of gut is threatened from delayed diagnosis, etc. metoclopramide; H2 blockers like ranitidine are given to act while stabilizing the patient. A nasogastric tube is useful to empty the stomach in various angles while turning the table head up, down lateral etc. it is removed just prior to the rapid sequence induction to maintain the integrity of both cricopharyngeal and oesophagogastric sphincters.

**Pre medication:**
Best premedication is the presence of the parent till the child goes to sleep. However the parents may be equally anxious, can faint in theatre and they need to be escorted out by a responsible person (enough staff!) etc.
Discretion should be used in the select group of children at risk from premedication like those with upper airway obstruction, those with poor reflex control, prone to aspiration like in coordinate swallowing and coughing. Sleep apnea central or obstructive (adenoid hypertrophy, functional macroglossia as in Beckwith Wideman, Down's or Pierre Robin syndrome) is an absolute contraindication as are children with muscular dystrophy with border line respiratory reserve with increased susceptibility to drugs.

**The premedicant drugs:**
The commonly used drugs to avoid separation anxiety are midazolam, and ketamine. An attractive alternative but presently unavailable in India is transmucosal fentanyl and oral clonidine. As we no longer use irritant anesthetics or succinylcholine, atropine is not specifically indicated in routine anesthesia. Its interference with thermoregulation in hot climate and in children with fever is a major deterrent to its routine use in our country. It also causes relaxation of gastro-oesophageal sphincter and can predispose to
gastro-oesophageal reflux particularly in children at risk. A few situations where there is a relative indication for atropine are neonates who have a rate dependent cardiac output where bradycardia can mean a significant fall in cardiac output, especially when cardiac depressant agents like halothane are used. Other conditions where antisialagogue effect is necessary are oral surgery (tonsillectomy, cleft palate), difficult intubation or when secretogogues like ketamine are used. It may also be preferred in strabismus surgery to forestall the oculocardiac reflex. Glycopyrrolate has a longer duration of action and does not cross the blood brain barrier. It can be used orally in a dose of 50 μgkg⁻¹ in lieu of atropine.

The route of administration

Oral route:
The physiological route is the one most preferred by the child. Previously valium, triclofos, and presently midazolam, ketamine singly or in combination have become very popular with children and the anesthesiologists alike. The intravenous formulation of both midazolam and ketamine have a bitter and astringent taste. So to make it palatable they have been mixed with cola, honey, apple juice etc. Concentrated orange crushes which are much sweeter than cola and can effectively mask the taste of these drugs is preferred. Alternatively these may be mixed with paracetamol syrup. The speed of onset can be augmented by 4 minutes by the addition of sodium citrate. Midazolam 0.5-0.75 mgkg⁻¹ with the maximum dose of 15 mg results in a sedated child in about 10-30 minutes. By 45 minutes the scores for satisfactory separation start to decline but light sedation may persist for up to 2 hours. As such, the time to hospital discharge is unchanged. Increasing the dose increases the side effects (loss of balance, blurring of vision, dysphoria) more than enhancing the sedation. In case of an overdose it can be immediately reversed by IV flumazenil in increments of 10μgkg⁻¹ upto 1 mg.
Ketamine in a dose range of 3-10 mgkg⁻¹ provides dissociation in that the child is distant or totally unaware, unlike with midazolam where the child is aware but calm. It also has the added advantage of providing analgesia (in the higher range) for the prick of the IV or regional block. Whether the children get dreams with this is not known but obvious emergence phenomena have not been reported. For avoiding dreams it is preferable to mix -ketamine (5-8 mgkg⁻¹) with midazolam (0.3-0.5 mgkg⁻¹). Oral atropine (0.02-0.04 mgkg⁻¹) can oppose the sialogogue action of ketamine and possibility of laryngospasm.

Nasal route:
The nasal route accesses the blood stream directly from the capillaries in the nose so there is no first pass effect and the sedation sets in rapidly within 5-10 minutes so resuscitation facility should always be available. However it is not used commonly

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because the nasal route is irritating and most children don’t like it. A theoretical cause of concern is that the drugs can directly access the CNS along the olfactory nerves through the cribriform plate drugs with preservatives should be avoided. Midazolam and sufentanyl have been used by this route for the rapid onset and effectiveness. Preservative free ketamine can also be used, but reserve it for those children who are very rowdy, refuse oral premedication and need to be quietened quickly to reduce their screaming and distress.

**Rectal route:**
It is usually used in the younger age group still in diapers. It used to be popular for administering methohexitone, thiopentone etc., but now midazolam, ketamine and atropine can be used to induce sedation in 9-11 mins. The parent can assist in the administration and thus the child accepts it more readily. The venous drainage of the lower rectum is into the inferior hemorrhoidal veins which do not go into the portal circulation so a first pass effect is avoided. However if the drug is placed above the pectinate line in the rectum, then the superior hemorrhoidal vein drains it into the portal system so that the first pass metabolism in the liver and loss of effective drug does occur. Another problem is that the child may expel some of the drug.

**Intravenous route:**
Has become popular again with the liberal use of EMLA and parental presence in OT. To be effective EMLA (eutectic mixture of local anesthetics) should be applied at least 1 hour prior to the induction with a plastic occlusive dressing applied over it to confine it over the vein. This requires adequate nursing staff, clear instructions and affordability. Another consideration is that the veins become smaller after EMLA so that venepuncture may become more difficult. If the IV route is used to give the first sedative it is necessary to remember that midazolam takes some time to induce EEG changes of sedation (3 times more time than diazepam. So IV route does not mean instantaneous sedation unless induction agents like Pentothal or propofol are used.

**Intramuscular route:**
Was mainly used to administer ketamine (5-10 mgkg⁻¹) and atropine (0.02 mgkg⁻¹) and midazolam (50 μgkg⁻¹) for radiotherapy, and prior to interventional cardiac procedures in the catheter laboratory. Never popular with the children, it has gone out of favour with Pediatric anesthesiologists in the last 2 decades. It has no present relevance because it takes as much time as the oral route or longer than the nasal or rectal route to act, and is extremely unpleasant for the child.
**Conclusion:**

To provide an empathetic care to a child, anesthesiologist has to be concerned enough to study and understand the emotional needs and responses of the child, develop an easy playful upbeat manner with them, and cultivate the gentleness and consummate skill for the interventions needed for anesthesia. It is as important to acquire the art of Pediatric anesthesia as the diligence to science. This is only possible to a dedicated well informed physician who acknowledges the importance of being concerned and practices it uniformly with every child.
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Chapter 5 - PERIOPERATIVE FLUID AND ELECTROLYTE MANAGEMENT IN PEDIATRIC PATIENTS

Introduction
Fluid management of the Pediatric surgical patient is a critical element in the care of infants and children who are sensitive to small degrees of dehydration. Complex surgical procedures are often associated with rapid changes in fluid requirements necessitating frequent assessment and modifications of fluid therapy. In the operating room, the fluid requirements may rapidly change during the conduct of anesthesia and surgery, coincident with changes in temperature, metabolism and fluid volume shifts. The trauma, haemorrhage and tissue exposure associated with surgery shifts body fluids between compartments, necessitating fluid replacement with solutions that compensate for energy, water, protein and electrolyte losses. The anesthesiologist must determine the nature and magnitude of these losses and be alert both to the obvious fluid losses of serum and urine and to hidden fluid losses, which can occur, with insensible loss and third space loss of fluid.

This short review will deal with the fluid and electrolyte management in the perioperative period of infants and children without going into any specific situations.

Physiological considerations in infants and children
Before one can scientifically approach the subject of fluid management in infancy and childhood, one must understand neonatal physiology and the changes that take place with time.

Total body fluid
The total body fluid (TBF) is divided into extra cellular fluid (ECF) and intra cellular fluid (ICF). Although body cells and the surrounding fluid remain in electrical equilibrium, the proportion of ECF and ICF changes with age. A 28 week foetus weighing 1 kg will be 80% water and only 1 % total body fat. At term, the total body fluid (TBF) decreases to 70-75% and a gradual shift of the extra cellular fluid into the intra cellular compartment has occurred. The fat component has now increased to 17%. At 3 months, when most infants have doubled their weight to 6 kg the fat component is 30% of their weight. In addition, the TBF has decreased to 65 %. This is associated with a further increase in their intracellular fluid.

Extra cellular fluid
This fluid includes the intravascular plasma volume and the interstitial fluid volume. The plasma volume and the interstitial fluid volume together constitute the functional...
extra cellular fluid volume (FEFV) Extra cellular fluid also includes the physiologically non-functional third space or trans cellular fluid.

The interstitial space acts as a reservoir, which can accept fluid filtered from the vascular compartment when the circulating volume is high. In situations where the circulating volume is low (haemorrhage), fluid from the interstitial space move into the vascular compartment to build up the circulating volume, the interstitial space also acts as a reservoir where proteins generated from the cells are stored before they are actually transferred to the vascular compartment through lymphatic channels.

During adolescence the volume of the interstitial space is about 20%. Adding the plasma volume of 7-10% to the interstitial volume, gives a FEFV of 27-30% in this age group. In term infants the FEFV may be as high as 45%.

Trans cellular (third space) fluid is non-functional extra cellular fluid. It is an unavailable pool of water formed by transudation of fluid from the cells and the extra cellular space. This includes fluid within the gastrointestinal tract formed during intestinal obstruction, ascites, urine, pleural effusions etc. Fluid that enters the trans cellular space is essentially lost from the FEFV.

**Intracellular fluid:**

TBF minus the ECF gives the intra cellular fluid volume. Cell volume remains constant during administration of isotonic solution due to free movement of water from within the cells. However, cell volume may rapidly increase during administration of hypotonic solutions due to inward movement of water. Much of the intracellular water is bound to proteins. Energy is required to transport potassium into the cell and for the sodium to be transported outside the cell.

**Renal physiology in neonates**

Most of the postnatal shifts in body fluids are mediated by sodium and water excretion by the immature kidneys. At birth the glomerular filtration rate (GFR) is just 25% (20 ml/min/1.73 m^2) of the adults. The GFR rapidly increases during the first two weeks of life and then at a slower rate till it reaches adult function by the age of 2 years. But despite the low GFR, all infants can handle up to twice the normal maintenance fluid load because the negative effects exerted by the low GFR is countered by the positive effects of low concentrating and high diluting capacity of the newborn kidney.

**Concentrating capacity:**

The concentrating capacity of an infant’s kidney is well below that of the adult. In response to water deprivation, the infant kidney can increase osmolality to a maximum 500-600 mOsmkg\(^{-1}\). In contrast, an adult kidney can generate urine with an osmolality of 1200 mOsmkg\(^{-1}\). This is because of decreased tonicity in the medullary interstitium.
Diluting capacity:
Although dehydrated newborns cannot concentrate their urine as efficiently as adults, water loaded term infants have free water clearance well above adults. After a water load, infants can excrete markedly dilute urine of 30-50 mOsmkg\(^{-1}\) in contrast to adults who can concentrate only up to 70-100 mOsmkg\(^{-1}\).

Electrolyte physiology in infants and neonates

Sodium physiology:
Serum sodium is variable in the neonate and hence cannot be used as an indicator of the hydration status of the infant. The daily sodium requirement of a term infant is 2-5 mEqkg\(^{-1}\)day\(^{-1}\) (table 1). Term infants, like adults, can retain sodium in the face of a negative sodium balance but have a diminished capacity to excrete excess sodium when in positive balance. Sodium administration stimulates growth. Acute changes in sodium balance can lead to gross variations in blood pressures and intracerebral haemorrhage. Positive pressure ventilation and use of positive end expiratory pressure (PEEP) irrespective of the presence or absence of respiratory distress, is associated with natriuresis and with increased water retention and vasopressin release.

Potassium physiology:
The usual recommended dose is 2-4 meqkg\(^{-1}\)day\(^{-1}\) given after the first few days of life (table 1). There has been a reluctance to administer potassium in the first two postnatal days or immediately after surgery. This is due to the fear of immature kidneys and defective renal function leading to hyperkalemia. In critically ill infants, many factors, including increased steroid and prostaglandin secretions, high urine output and the use of diuretics, lead to a negative potassium balance. To prevent this hypokalemia, 1-2 meqkg\(^{-1}\)day\(^{-1}\) of potassium is recommended parenterally to postoperative infants when there is adequate urine output. Because more than 98% of the total body potassium stores are in the intracellular compartment, serum potassium levels are a poor indicator of the total potassium stores.
Table 1: Daily electrolyte requirements for Pediatric patients

<table>
<thead>
<tr>
<th></th>
<th>Patients weighing &gt; 10kgs</th>
<th>Patients weighing &lt; 10kgs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium</td>
<td>20-150 mEq</td>
<td>2 – 5 mEq/kg</td>
</tr>
<tr>
<td>Potassium</td>
<td>20-240 mEq</td>
<td>2 – 4 mEq/kg</td>
</tr>
<tr>
<td>Acetate</td>
<td>20-120 meq</td>
<td></td>
</tr>
<tr>
<td>Chloride</td>
<td>20-150 meq</td>
<td></td>
</tr>
<tr>
<td>Calcium</td>
<td>5-20 meq</td>
<td>0.5 – 3 mEq/kg</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>4-24 mmol</td>
<td>0.5 – 1.5mmol/kg</td>
</tr>
<tr>
<td>Magnesium</td>
<td>4-24 meq</td>
<td>0.25 – 1 mEq/kg</td>
</tr>
</tbody>
</table>

Cardiovascular considerations in infants and neonates

The incomplete development of the myocardium and the immature sympathetic nervous system makes infants and neonates more sensitive to hypovolaemia than older children and adults. Myocardial contractility, ventricular compliance and vascular tone are lower and less variable, making tachycardia the primary compensatory mechanism during volume depletion. Cardiac output decreases when the limits of tachycardia are reached. Anesthetic depression of cardiovascular function further accentuates the effects of hypovolaemia. Thus maintenance of effective vascular volume in Pediatric patients is essential to sustain circulatory function and vital organ perfusion in the perioperative period.

Determining fluid requirements

Methods

Many systems have been devised to calculate the amount of fluids, calories and minerals required for continuing growth, maintenance during anesthesia, replacement of fluid losses and fluid shifts and recovery from surgical stress.

Body Surface Area (BSA) method

BSA method of calculating the fluid and energy requirements is based on the concept that caloric expenditure is proportional to body surface area. Based on this principle, the water and electrolyte requirements are 1500mlm⁻² day¹, the sodium requirements are 30-50meq m⁻²day⁻¹ and potassium requirements are 20-40meq m⁻² day⁻¹. However, fluid requirements based on BSA are not recommended at present, as they are prone to errors.
Calorie consumption and body weight

Calorie expenditure has become the standard for determining the fluid and energy requirement in children. Caloric requirements equal the fluid requirements in infant. Metabolism of 1 calorie produces 0.2 ml of water and also consumes 1.2 ml of water. Thus in an awake child the fluid and caloric consumption are considered equal. In 1957 Holliday and Segar assessed the metabolic and active energy requirements in awake hospitalized children. The calculated energy requirements of hospitalized infants up to 10 kg were 100 cal kg\(^{-1}\)day\(^{-1}\). Of this 50% was utilized for basal metabolism while the remaining 50% was utilized for growth. In children weighing more than 10 kg, growth slowed and the caloric requirements decreased to 50 cal kg\(^{-1}\)day\(^{-1}\) for the weights above 10 kg (i.e. 1000 cal + 50 cal kg\(^{-1}\)day\(^{-1}\)). Metabolic requirements were further reduced in children weighing more than 20 kg. For the weights above 20 kg, the caloric requirements were reduced to 20 cal kg\(^{-1}\)day\(^{-1}\) (i.e. 1500 cal + 20 cal kg\(^{-1}\)day\(^{-1}\)).

Fever increases the caloric requirements by 10-12% for every centigrade rise in temperature above normal. Reduction in metabolic requirements reduces energy requirements in a similar manner.

Fluid management

This is divided into 3 phases

a. Deficit therapy
b. Maintenance therapy
c. Replacement therapy

Deficit therapy

This refers to the management of fluid and electrolyte losses that occur prior to presentation for surgery and itself has 3 components: a) estimation of dehydration severity, b) determination of fluid deficit type and c) deficit repair.

Dehydration severity is usually estimated from the history and clinical evaluation (table 2). Four pertinent investigations that will confirm the type of dehydration include:

a. Serum osmolarity and serum sodium
b. Acid-base status, serum pH and base deficits
c. Serum potassium compared with the pH
d. Urine output (rule out acute tubular necrosis)
Table 2: Assessment of dehydration severity in neonates and infants

<table>
<thead>
<tr>
<th>Signs and symptoms</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight loss</td>
<td>3-5%</td>
<td>6.9%</td>
<td>&gt; 10%</td>
</tr>
<tr>
<td>General condition</td>
<td>Alert, restless</td>
<td>Thirsty, lethargic</td>
<td>Cold, sweaty, limp</td>
</tr>
<tr>
<td>Pulse</td>
<td>Normal rate &amp; volume</td>
<td>Rapid, weak</td>
<td>Rapid, feeble</td>
</tr>
<tr>
<td>Respiration</td>
<td>Normal</td>
<td>Deep, rapid</td>
<td>Deep, rapid</td>
</tr>
<tr>
<td>Ant. Fontanelle</td>
<td>Normal</td>
<td>Sunken</td>
<td>Very sunken</td>
</tr>
<tr>
<td>Systolic pressure</td>
<td>Normal</td>
<td>Normal or low</td>
<td>Low, unrecordable</td>
</tr>
<tr>
<td>Skin turgor</td>
<td>Normal</td>
<td>Decreased</td>
<td>Markedly decreased</td>
</tr>
<tr>
<td>Eyes</td>
<td>Normal</td>
<td>Sunken, dry</td>
<td>Grossly sunken</td>
</tr>
<tr>
<td>Mucus membrane</td>
<td>Moist</td>
<td>Dry</td>
<td>Very dry</td>
</tr>
<tr>
<td>Urine output</td>
<td>Adequate</td>
<td>Less, dark</td>
<td>Oliguria, anuria</td>
</tr>
<tr>
<td>Estimated deficit</td>
<td>30-50 mlkg⁻¹</td>
<td>60-90 mlkg⁻¹</td>
<td>100mlkg⁻¹</td>
</tr>
</tbody>
</table>

The above clinical and biochemical investigations will reveal whether the type of dehydration is hyponatraemia (serum osmolarity <270mOsmL⁻¹, serum Na < 130meqL⁻¹), Isonatremic (serum osmolarity 270-300 mOsmol, serum Na 130-150 meqL⁻¹) or Hypernatremic (serum osmolarity >310 mOsmL⁻¹, serum Na >150 meqL⁻¹). However, treatment of the fluid deficit should be initiated before all the investigations are available. Initial fluid resuscitation can be initiated with a bolus of normal saline given over 10-20 minutes to improve circulation and restore renal perfusion. For patients with known contraction alkalosis, 5% Dextrose with 0.9% saline would be a reasonable fluid of choice. In patients with known metabolic acidosis, removing 250 ml of 0.9% saline from a 1 L container and replacing it with 28 ml of 7.5% sodium bicarbonate solution and 232 ml of 5% dextrose in 0.9% saline can formulate a more appropriate solution. The resulting solution contains approximately 1.2% Dextrose, 140 meq of sodium, 115 mEq chloride and 25 mEq of sodium bicarbonate. Giving lactate or acetate containing solution to children with severe metabolic acidosis can aggravate their acidosis especially if these precursors of bicarbonate cannot be metabolized to bicarbonate by the liver because of the poor circulatory status.

Even a 1% reduction of blood volume is associated with a rectal temperature rise of 0.3°C. The mechanism for a febrile response to volume contraction may be related to a decrease, in skin blood flow, which prevents dissipation of heat. In addition, hyperosmolarity elevates the threshold for Sweating. This in turn will increase their caloric and fluid requirements.
**Fluid deficit due to overnight fasting**

To avoid the complication associated with pulmonary aspiration during induction of anesthesia, prolonged fasting has generally been advocated. Recent studies have shown that the residual gastric volume was lower and the pH higher, in children allowed clear fluids up to 2 hours before surgery. Sips of clear fluids stimulate peristalsis but don’t stimulate gastric secretion if no protein is present. H$_2$ blockers effectively elevate the gastric pH and further reduces gastric volume. Present recommendations include administration of clear fluids up to two hours before surgery or milk feeds up to 4 hours before surgery.

In general, deficits caused by preoperative restriction of fluids are calculated by multiplying the hourly maintenance requirements times the number of hours of fluid restriction. Of the total amount, 50% is replaced in the first hour and 25% each in the next 2 hours. The fluid requirements for covering the deficits caused by preoperative fasting in neonates and older children are given in table 3. This is similar to maintenance fluid requirements during the course of the surgery.

**Table 3: Maintenance fluid requirements in neonates**

<table>
<thead>
<tr>
<th>Age (days) / weight (kgs)</th>
<th>Requirements: mlkg$^{-1}$ day$^{-1}$</th>
<th>Hourly: mlkg$^{-1}$ hr$^{-1}$</th>
<th>Type of fluid</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>20-40</td>
<td>2-3</td>
<td>10% dextrose</td>
</tr>
<tr>
<td>2</td>
<td>40-60</td>
<td>3-4</td>
<td>10% dextrose in 0.22% saline</td>
</tr>
<tr>
<td>3</td>
<td>60-80</td>
<td>4-6</td>
<td>10% dextrose in 0.22% saline</td>
</tr>
<tr>
<td>4</td>
<td>80-100</td>
<td>6-8</td>
<td>5-10% dextrose in 0.22% saline</td>
</tr>
<tr>
<td>0-10 kgs</td>
<td>100</td>
<td>4 mlkg$^{-1}$ hr$^{-1}$</td>
<td>5% dextrose in 0.45% saline</td>
</tr>
<tr>
<td>10-20 kgs</td>
<td>1000-50 mlkg$^{-1}$</td>
<td>40ml + 2mlkg$^{-1}$ hr$^{-1}$</td>
<td>5% dextrose in 0.45% saline</td>
</tr>
<tr>
<td>&gt; 20 kgs</td>
<td>1550-20 mlkg$^{-1}$</td>
<td>60ml + 1 mlkg$^{-1}$ hr$^{-1}$</td>
<td>5% dextrose in 0.45% saline</td>
</tr>
</tbody>
</table>

Deficit replacement revolves around the restoration of cardiovascular function, CNS function and renal perfusion. As mentioned earlier, deficit should be replaced with a balanced salt solution based on the type and severity of the dehydration. This should take into account the type of fluid loss from the body (table 4) and a suitable type of balanced salt solution (table 5). Total fluid replacement may take considerable time and potassium losses in particular cannot be replaced immediately. Potassium should be replaced only after adequate renal perfusion is established, acidosis corrected and child starts putting out urine.
Table 4: Electrolyte composition of body fluids

<table>
<thead>
<tr>
<th>Electrolytes (meqL⁻¹)</th>
<th>Gastric</th>
<th>Pancreatic</th>
<th>Bile</th>
<th>Ileostomy</th>
<th>Diarrhea</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na⁺</td>
<td>70</td>
<td>140</td>
<td>120</td>
<td>130</td>
<td>50</td>
</tr>
<tr>
<td>K⁺</td>
<td>5-15</td>
<td>15</td>
<td>5</td>
<td>15-20</td>
<td>35</td>
</tr>
<tr>
<td>Cl⁻</td>
<td>120</td>
<td>50-100</td>
<td>100</td>
<td>120</td>
<td>40</td>
</tr>
<tr>
<td>HCO₃⁻ (meqL⁻¹)</td>
<td>0</td>
<td>35</td>
<td>40</td>
<td>25-30</td>
<td>50</td>
</tr>
</tbody>
</table>

Table 5: Composition of commonly used intravenous fluids in children

<table>
<thead>
<tr>
<th>Electrolytes (meqL⁻¹)</th>
<th>Normal saline</th>
<th>Ringers lactate</th>
<th>IVEOLYTE P</th>
<th>PLSMATELYTE A</th>
<th>Dextrose 5%</th>
<th>Albumin 5%</th>
<th>Hetastarch 6%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na⁺</td>
<td>154</td>
<td>130</td>
<td>26</td>
<td>140</td>
<td>-</td>
<td>145 ±15</td>
<td>154</td>
</tr>
<tr>
<td>K⁺</td>
<td>-</td>
<td>4</td>
<td>21</td>
<td>5</td>
<td>-</td>
<td>&lt;2.5</td>
<td>-</td>
</tr>
<tr>
<td>Cl⁻</td>
<td>154</td>
<td>109</td>
<td>21</td>
<td>98</td>
<td>-</td>
<td>100</td>
<td>154</td>
</tr>
<tr>
<td>Ca²⁺</td>
<td>3</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Mg²⁺</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Acetate</td>
<td>-</td>
<td>-</td>
<td>3</td>
<td>3</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Lactate</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Glucose (gm%)</td>
<td>-</td>
<td>-</td>
<td>5</td>
<td>-</td>
<td>5</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Phosphate (mg%)</td>
<td>-</td>
<td>-</td>
<td>3</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Osmolarity (mOsmL⁻¹)</td>
<td>308</td>
<td>274</td>
<td>-</td>
<td>295</td>
<td>252</td>
<td>330</td>
<td>310</td>
</tr>
</tbody>
</table>

Maintenance fluid therapy
The maintenance fluid meets the ongoing fluid and electrolyte requirements during the course of surgery. It does not take into account blood loss or third space loss of fluid into interstitial space or gut. Based on the Holliday-Segar calculations, the hourly maintenance fluid requirements should be replaced (table 3).

The fluids for maintenance therapy replace losses from two source:
1. Insensible losses (evaporative loss)
2. Urinary loss.
Evaporative loss is composed of solute free loss of water through the skin and lungs. Under ordinary conditions this accounts for 30-35% of the total maintenance requirements. Insensible losses are affected by ambient humidity and temperature, gestational age of the infant, type of respiration and surface area exposed. Ventilation with humidified gases results in significantly lower insensible loss.
In a euvoletic state, urinary loss concentrations range from 280-300 mOsmkg⁻¹ of water, with a specific gravity of 1.008-1.015. In some circumstances (e.g. diabetes
insipidus, premature infants), an obligatory production of dilute urine exists, and appropriate increases in maintenance fluid requirements should be made. On other occasions (excessive ADH secretion), a patient may be unable to decrease urine osmolality to 300 mOsmkg⁻¹ of water, and the volume of maintenance fluids should be decreased. If the estimate of the maintenance fluid requirements is correct, the patient's electrolyte levels should remain stable and the patient should remain clinically euvoletic.

**Glucose requirements in maintenance fluids:**
In addition to the lower body stores or glycogen, infants have a higher-metabolic rate and oxygen consumption than older children. Neonatal surgery, particularly pre-bypass surgery can induce significant life threatening hypoglycemia. However, intraoperative hypoglycemia is exceedingly rare in children. On the other hand, hyperglycemia is more commonly encountered during anesthesia and surgery. Intraoperative glucose uptake by the muscle is reduced. The response to anesthesia surgery, anxiety and pain further increase the blood sugar levels. Hyperglycemia may be further aggravated by the impaired effectiveness of insulin during anesthesia. A glucose administration rate of more than 10 mgkg⁻¹min⁻¹ may overwhelm the renal threshold and result in glycosuria and osmotic diuresis.

With decreased tolerance to exogenous glucose and increased endogenous glucose production, a solution containing low concentrations of glucose in balanced salt solution may be required as maintenance fluid. The replacement fluid should either be free of dextrose or should not have more than 1 % dextrose. Other studies have similarly confirmed the usefulness of low concentrations of glucose in the maintenance fluid. The present recommendations include the use of low dextrose containing solutions for maintenance fluid therapy, this would ensure adequate blood sugar levels without inducing hyperglycemia.

**Replacement therapy**
Replacement therapy is designed to replace ongoing abnormal fluid and electrolyte losses. Because the constituents of replacement fluid are usually different from that of the maintenance fluid, simply increasing the volume of maintenance fluid for the losses may be harmful.

Replacing fluid losses with balanced salt solution leads to less fluid retention and a natriuretic response is induced. In most patients Ringer's lactate solution is a reasonable choice as the replacement fluid and is less expensive than other balanced electrolyte solutions. Normal saline with its higher sodium content may be preferable in children at risk of cerebral edema. Table 5 shows the commercially available intravenous fluids that can be used for maintenance or replacement therapy and their
electrolyte composition. Three factors are active in the restoration of fluid homeostasis when using a balanced salt solution:

a. Fluid balance is sensed by volume receptors and osmoreceptors. Infusing a balanced salt solution that contains a relative excess of sodium replaces water while increasing total body sodium.

b. This relative sodium excess helps to maintain circulation and to replete the FEFV. Increases in vascular capacitance stretch the cardiac atrium and increases renal blood flow. Natriuretic hormone further increases blood volume.

c. By increasing renal blood flow and stimulating sodium excretion, body fluid homoeostasis (via a diuretic and natriuretic response) re-equilibrates, which reduces peri-operative fluid retention.

Excess water on the other hand produces tissue edema. This in turn hampers the transcellular flow of nutrients and outflow of waste from the cells, which ultimately leads to a vicious cycle leading to cell death.

**Third space loss**

Surgical trauma leads to fluid translocation from the FEFV to a non-functional compartment and these deficits must be replaced in order to maintain an adequate FEFV. Functionally this fluid is neither available to the vascular compartment nor the FEFV. The composition of this fluid is similar to that of ECF in addition to a small amount of proteins. A balanced salt solution like Ringer’s lactate is again the preferred fluid for replacement of the third space loss. The replacement for third space losses depends on the severity of the surgical trauma. Table 6 shows the guidelines for replacing third space losses.

**Table 6: Replacement for third space and evaporative losses**

<table>
<thead>
<tr>
<th>Surgical trauma</th>
<th>Type of surgery</th>
<th>Fluid replacement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Minimal</td>
<td>Inguinal hernia repair</td>
<td>1-2 mlkg⁻¹hr⁻¹</td>
</tr>
<tr>
<td>Moderate</td>
<td>Urethral implantation</td>
<td>4 mlkg⁻¹hr⁻¹</td>
</tr>
<tr>
<td>Severe</td>
<td>Scoliosis, bowel obstruction</td>
<td>&gt; 6mlkg⁻¹hr⁻¹</td>
</tr>
</tbody>
</table>
Replacement of blood loss

In children, all blood loss should be replaced. Replacement of blood loss can be either in the form of packed red cells, whole blood, colloids or crystalloids. The clinical evaluation of the “amount of blood loss” can be on simple clinical judgment, swab weighing or laboratory data. “Davenport’s law” is simple to apply, mainly for those who do not deal with children frequently; under 10% of blood loss no blood is required, over 20% losses must be replaced with either packed red cells or whole blood and, between 10-20% we must consider case by case. “Davenport’s law” is questionable and not generally followed.

When a crystalloid is used for replacing blood loss, for each ml of blood lost, 3 ml of crystalloid should be replaced. Being a balanced salt solution it rapidly distributes to the extracellular space and only 20-30% remains in the intravascular compartment. An adequate oxygen transport should be ensured. An acceptable hematocrit that ensures an adequate oxygen transport depends on various factors including age, duration of surgery, and expected further blood loss. However, a minimum hematocrit of 30% in older children and 40% for neonates is generally agreed upon.

Crystalloid or Colloid: The Controversy.

In spite of the controversy surrounding the use of a crystalloid or colloid, most agree that parenteral fluid administration should start with a balanced salt solution. The major disadvantage with the use of balanced salt solution is that within a period of two hours most of the administered solution distributes to all the active fluid compartments of the body. Therefore, hemorrhagic fluid losses require three to four times the volume as opposed to whole blood replacement.

Human albumin or synthetic colloids are recommended by some to maintain the intravascular compartment. In theory, administration of albumin—should raise the oncotic and osmotic pressure which would mobilize the intracellular and interstitial fluid into the vascular compartment and by paralyzing the distal tubule would resolve the tissue edema and maintain the intravascular compartment. However, this effect is rarely seen, particularly in patients with capillary leak syndrome, which prevents the albumin from remaining in the intravascular compartment. Albumin is most appropriately used during major surgery to maintain vascular volume.

Hetastarch is a group of compounds that has an intravascular life upwards of 3 hours and some particles remain in the vascular compartment for many days. The daily recommended dose should not exceed 20mlkg⁻¹. A hybrid solution containing 6% Hetastarch in a balanced salt solution is also available. This combines the advantages of a colloid kick with the buffer in lactated Ringer’s solution.

Table 4 gives an idea of the various crystalloids and colloids available for use in various situations.
Electrolyte imbalance in the perioperative period

Disturbances in sodium physiology
Sodium is the most abundant cation in extra cellular fluid and is critical in determining the extra cellular and intra cellular osmolality. Sodium requirements may vary with age. Sodium requirement for term infants is 2-3 meqkg⁻¹day⁻¹. Neonatal stool losses are 1 meqkg⁻¹day⁻¹ and growth requirements are about 0.5 meqkg⁻¹day⁻¹.

Hyponatraemia
Serum sodium concentration is less than 130 meqL⁻¹. This is a common perioperative electrolyte disturbance. It can be of different types based on the volume status of the patient.

1. Hypovolemic hyponatraemia: There is a reduction in extra cellular fluid volume e.g. gastroenteritis, diuretic therapy or renal losses. Hypovolemic hyponatraemia causes cerebral edema.
2. Hypervolemic hyponatraemia. There is excessive extra cellular fluid volume e.g. nephrotic syndrome.
3. Isovolumic hyponatraemia. Associated with normal extra cellular volume e.g. SIDH, glucocorticoid therapy.

Hyponatraemia is usually asymptomatic but obtundation and seizures can occur if the serum sodium level becomes very low (< 120 meqL⁻¹). Cardiac symptoms occur at levels below 100 meqL⁻¹.

Management
Appropriate therapy requires identification of the underlying cause. In hyponatraemia with decreased circulating volume, treatment is with administration of supplemental volume. Patients with symptomatic hyponatraemia and clinical euvoletic or hypervolemic require infusion of hypertonic saline (3% or 5% saline). It has been shown that even small increases in serum sodium of the order of 5% can reduce cerebral edema or stop seizures. Correction should be of sufficient pace so as to reverse the manifestation of hypotonicity but at the same time it should not pose a threat of demyelination. Rapid treatment of hyponatraemia can lead to central pontine myelinolysis. It is characterized by insidious onset of flaccid quadriplegia and cranial nerve abnormalities. Hypervolemic hyponatraemia indicates excess body water and is often treated with a combination of Frusemide and hypertonic saline. The formula detailed below tells the change in serum sodium that can be expected after the infusion of 1 Litre of the solution.

Change in serum Na⁺ = Infuscate Na⁺ - \[ \frac{\text{Serum Na}}{\text{TBW}+1} \]
Estimates the effect of 1 litre of any infuscate on serum Na\. Total body water ((TBW) in liters) = Body weight x 0.6.

The infuscate sodium for 5% saline is 855 meqL⁻¹, 3% saline is 513 meqL⁻¹ and normal saline is 154 meqL⁻¹. Half the deficit can be corrected over 12-14 hours and the remaining over the next 1-3 days. Recommended indications for stopping the rapid correction of symptomatic hyponatraemia includes cessation of life threatening manifestations, moderation of symptoms or achievement of a serum sodium concentration of 125 to 130 meqL⁻¹. It is generally agreed that the targeted rate of correction should not exceed not more than 8 meqL⁻¹ on any day.

**Hypernatremia**

Hypernatremia represents a deficit of water in relation to the body stores of sodium, which can result from a net water loss or hypertonic sodium gain. It can occur in the absence of a sodium deficit or in its presence. Hypernatremia is defined as serum sodium levels in excess of 145 meqL⁻¹. In children this may be associated with muscle weakness, hyperpnoea, restlessness, insomnia, lethargy and coma. Brain shrinkage can lead to cerebral haemorrhage.

**Management:**

Of hypernatremia is two pronged. The initial step is to identify and control the underlying cause (gastrointestinal losses or hyperglycemia induced diuresis). The amount of fluid that should be given to correct the hypernatremia is given by the formula:

Only hypotonic fluids are preferred. This includes 5\% dextrose (infuscate Na=0), 0.45\% saline (infuscate sodium 77) or 0.2\% saline in 5\% dextrose (infuscate sodium 34). The more hypotonic the infuscate the lesser the rate of infusion required for correction of the deficit. In patients who have developed hypernatremia over a period of few hours, rapid correction of the deficit over a few hours improves prognosis without increasing the risk of cerebral edema as the accumulated electrolytes gets washed out rapidly from the brain cells. In such patients reducing the serum sodium by 1 meqL⁻¹hr⁻¹ is appropriate. In patients who have developed hypernatremia over a period of days it would be more prudent to correct the deficit by no more than 0.5 meqL⁻¹day⁻¹. This would avoid the complications related to cerebral edema and convulsions. It is generally recommended that the reduction in serum sodium should not be more than 10 meqday⁻¹.

**Disturbances in potassium physiology**

Potassium is the most abundant cation in the intracellular fluid. Potassium levels are influenced by insulin, pH of the blood and tissues, β-adrenergic agonists and aldosterone.
Hypokalemia
This is a common postoperative finding and usually caused by gastrointestinal (diarrhea, vomiting, villous adenoma) or renal losses diuretics, chronic metabolic alkalosis, renal tubular acidosis) or abnormal electrolyte shifts induced by drugs (β-adrenergic agonists, alkalosis, insulin). Clinical symptoms include lethargy, muscle weakness, ECG changes and ventricular arrhythmias.

Management:
Emergency management is indicated when the hypokalemia is associated with cardiac arrhythmias. Rate of intravenous correction should not exceed 0.2 to 0.5 meqkg⁻¹ hour⁻¹. Deficits can be calculated from the formula:
Potassium deficit (meqL⁻¹) = Body weight x (Expected serum K⁺ - observed serum K⁺) x 0.3
Intravenous potassium should always be administered through a central line. The serum potassium levels should be monitored at very close intervals and the electrocardiogram should always be monitored.

Hyperkalemia
Serum potassium levels in excess of 5.5 meqL⁻¹ leads to hyperkalemia. The condition requires immediate attention and may be fatal if not managed on an emergency basis. In the surgical setting the most common causes for hyperkalemia include (acute renal failure, metabolic acidosis, exogenous administration of potassium effects of cardioplegia during cardiac surgery, stored blood transfusions and extensive tissue necrosis. In a perioperative setting, succinylcholine has been implicated to cause potassium release from depolarized muscle tissue. This can lead to an increase in the serum potassium by 0.5 to 1 meqL⁻¹ in a normal patient; this may be disastrous in a situation where there is a pre-existing hyperkalemia. Succinylcholine induced hyperkalemic cardiac arrest was reported in apparently healthy children, almost half of whom had received a diagnosis of neuromuscular disorder. Acidosis is another common cause for hyperkalemia. For every 0.1 unit decrease in serum pH, the serum potassium increases by 0.2 to 0.4 meqL⁻¹.
ECG changes associated with hyperkalemia include:
- 5.5-6.5 meqL⁻¹: tall "T" waves
- 6.5-8 meqL⁻¹: small "p" and widening of QRS complex
- 8-9 meqL⁻¹: disappearance of P waves, QRS and T merge to form sine waves
- >9 meqL⁻¹: ventricular tachycardia, ventricular fibrillation, atrio-ventricular dissociation and cardiac standstill.
**Management:**
Immediate therapy for hyperkalemia is to prevent life threatening cardiac arrhythmias. If the patient is hemodynamically stable without ECG changes, then a recheck of the values is reasonable. If ECG changes are present, any exogenous source of potassium should be replaced with saline. This includes any potassium containing maintenance or replacement fluids. Intravenous calcium chloride $10-20\, \text{mgkg}^{-1}$ or calcium gluconate $60\, \text{mgkg}^{-1}$ will stabilize the myocardium and serve as a physiological antagonist to potassium. Correction of any metabolic acidosis with sodium bicarbonate will shift the potassium into the intracellular compartment. Intravenous glucose-insulin ($0.1\, \text{Ukg}^{-1}$ of insulin with $0.5\, \text{gmkg}^{-1}$ of glucose) will have a similar effect.

The potassium binding resin, sodium polystyrene sulfonate may be given orally or rectally in a dose of $0.5$ to $1\, \text{gmkg}^{-1}\text{dose}^{-1}$. This will bind the potassium intraluminally and reduce total body potassium. The onset of its action will take several hours. If hyperkalemia cannot be controlled with these measures, emergency dialysis is indicated.

**Disturbances in calcium physiology**
Calcium plays a key role in bone formation, in cell division, growth, coagulation and excitation-contraction coupling of muscle tissue. Body contents of calcium in infants are $400\, \text{meqkg}^{-1}$ whereas it is $950\, \text{meqkg}^{-1}$ in adults. Body reserves of calcium are very low in preterm infants compared with those of term infants and hypocalcaemia occurs in about $90\%$ of preterm infants.

**Hypocalcaemia**
Hypocalcaemia is defined as a serum calcium less than $4.5\, \text{meqL}^{-1}$. Although there are a number of non surgical causes of hypocalcaemia (Hypoparathyroidism, Vit. D deficiency, pancreatitis etc), the common causes for hypocalcaemia in the operating room is administration of blood in excess of $1.5\, \text{mlkg}^{-1}\text{min}^{-1}$ or due to acute hyperventilation. Low albumin levels can result in manifestation of symptoms of hypocalcaemia. Symptoms include neuromuscular irritability, weakness, paresthesia, cardiac dysrrhythmias and prolonged QT interval in the ECG and Carpopedal spasm.

**Management:**
Parenteral calcium therapy for hypocalcaemia in preterm neonates can significantly improve their myocardial performance, particularly in patients with impaired myocardial function. Treatment includes correction of the underlying cause and intravenous infusion of calcium chloride ($20\, \text{mgkg}^{-1}$) or an equivalent dose of $10\%$ calcium gluconate ($60\, \text{mgkg}^{-1}$). In these concentrations, the chloride and gluconate forms of the calcium salt are equally effective for increasing the ionized calcium concentrations in children.
Hypercalcemia:

Hypercalcemia is commonly seen in patients with hyperparathyroidism, Vit. D intoxication, prolonged immobilization, use of thiazides diuretics, milk alkali syndrome, errors in total parenteral nutrition malignancies etc.

Management:

The clinical picture dictates the management. Gastrointestinal symptoms of hypercalcemia include anorexia, nausea, vomiting and constipation. Hypertension, augmentation in digoxin toxicity, renal dysfunction and central nervous system disturbances including coma are also seen during hypercalcemia. Management requires immediate hydration with normal saline. Loop diuretics, Biphosphonates, Plicamycin, calcitonin, steroids, phosphates and prostaglandin all have their place in chronic and acute therapies to reduce serum calcium.

Disturbances of Magnesium physiology

Magnesium is the fourth most abundant cation in the body. Because of its relative intra cellular abundance, it plays a major role in cellular enzyme regulation. Body magnesium content is 22 meqL$^{-1}$ in infants and 28 meqL$^{-1}$ in adults. Bone and muscle cells are major intra cellular pools of magnesium. Normal levels range from 1.5 to 1.8 meqL$^{-1}$.

Hypomagnesaemia

Magnesium deficiency is rare in newborns. In older infants it is associated with prolonged use of magnesium free hyper alimentation solutions. It is a common finding in critically ill children. Sixty five percent of patients in a critical care setting have magnesium deficiency, which may be aggravated by epinephrine infusions. Other causes include hyperaldosteronism, intestinal fistulas, starvation, pancreatitis and use of β-adrenergic agonists. Calcium deficiencies often accompany magnesium deficiencies. Manifestations include increased neuromuscular irritability, tetany, seizures, tremors and hyperreflexia. In older children, serum total magnesium levels less than 0.7-0.8 mmolL$^{-1}$, and for neonates, serum total magnesium levels less than 0.6 mmolL$^{-1}$ are recommended to diagnose hypomagnesaemia.

Magnesium has important cardiovascular and non-cardiovascular functions. It’s important cardiovascular functions include energy metabolism in adenosine triphosphate dependant processes (e.g. myocardial contraction), maintenance of cell membrane electrical potential and regulation of cardiovascular function. The primary risk to patients with hypomagnesaemia is ventricular dysrhythmias. Ventricular tachycardia, fibrillation and torsades de pointes can all occur with hypomagnesaemia.
Management:
Treatment is with administration of intravenous magnesium sulphate 15-30 mgkg⁻¹ intravenously over 15-30 minutes.

Hypermagnesemia
High magnesium concentrations are present in newborns of mothers getting magnesium salts for treatment of toxemia of pregnancy. These salts can cause sedation and may potentiate muscle relaxants in parturient and infants. High magnesium concentrations can also result from renal failure, administration of magnesium containing laxatives and antacids.

Management:
Any supplementary magnesium should be stopped. Elimination of magnesium involves fluid loading followed by or with concomitant diuresis. Definitive therapy involves dialysis. Temporary reversal of effects of magnesium can be managed with calcium therapy. As hypomagnesaemia potentiates the effects of depolarizing and non depolarizing muscle relaxants, these agents must be carefully titrated in conjunction with appropriate assessment of neuromuscular blockade.

Conclusion
Appropriate perioperative fluid management is essential for the Pediatric patient. Management must be individualized, taking into account developmental and physiological considerations. The caloric method is appropriate for estimating maintenance fluid requirements, but the clinical response is the best indicator of adequate therapy. Preoperative fasting should be confined to the minimum time compatible with patient safety. Dextrose containing solutions are best confined to children who are at risk of hypoglycemia, particularly preterm infants and neonates. Restoration of the circulating volume and vital organ perfusion is the first priority in perioperative fluid management and is best accomplished with isotonic crystalloid. Therapy for perioperative fluid and electrolyte disorders maybe guided by serum sodium and potassium concentrations, volume status, urine output and urinary loss of electrolytes. Symptomatic hyponatraemia and hyperkalemia are the electrolyte disturbances that warrant emergency management.
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Chapter 6 - MONITORING IN PEDIATRIC ANESTHESIA

Introduction
The word 'monitor' is derived from the Latin verb 'monere' - to warn. The purpose of a monitoring device is to measure a physiological variable and to indicate trends of change, thus enabling appropriate therapeutic measures to be taken. Monitoring in general is continuous; however, it can be intermittent also. The extent of monitoring depends upon various factors such as, the physical status of the patient and the invasiveness of the surgical procedure. However, monitoring of certain variables is mandatory and considered as standard of care. Whatever is the accuracy and sensitivity of the monitoring equipment, there is no substitute for the continuous presence of a vigilant anesthesiologist. Direct observation of the patient by the anesthesiologist is the most important single method of monitoring. The anesthesiologist may observe changes in clinical parameters such as, colour of the patient (cyanosis or pallor) or of the surgical field, the respiratory pattern, the peripheral temperature, the peripheral pulses and the capillary filling of the nail bed. Moreover, there are certain physical signs that remain outside the scope of the monitoring equipments such as signs of insidious onset of airway obstruction, status of the eyes, pulsatility of the mesenteric vessels, and palpation of the fontanel etc. Knowing the different stages of the surgical procedure and observing the surgical field may explain major hemodynamic changes that can either be anticipated or rapidly reversed (e.g. bradycardia caused by traction on the extraocular muscles during strabismus surgery, systemic hypotension caused by compression of the inferior vena cava during major abdominal surgery). Monitoring in these circumstances can detect the change in the parameter but not the cause.

The well being of various organ systems depend upon adequate perfusion of tissues with oxygenated blood and nutrients. Consequently the physiological variables that indicate adequacy of the oxygenation and perfusion are routinely monitored during the conduct of anesthesia. The tissue perfusion is determined by cardiac output; therefore, the variables that determine the cardiac output (eg. heart rate and rhythm, preload, etc) are routinely monitored. Hemodynamic monitoring reflects perfusion data, while respiratory monitoring concentrates on determination of the adequacy of oxygenation and ventilation. The other objectives of monitoring during anesthesia are to ensure adequate depth of anesthesia and muscle relaxation and recovery from relaxants at the end of the surgical procedure. The depth of anesthesia is monitored by lower oesophageal sphincter contractility, frontalis EMG, BIS and auditory evoked potential while the relaxant effect is monitored by peripheral nerve stimulator. The monitoring aids may remain non invasive or invasive (transgress tissue barriers); various routinely employed aids include the use of:
- Stethoscope
- Electrocardiography
- Pulse oximetry and plethysmography
- Non invasive blood pressure
- Finger blood pressure and arterial tonometry
- Invasive arterial pressure
- Temperature monitoring
- Urine output
- Central venous pressure and pulmonary artery pressure*
- Cardiac output*
- Transesophageal echocardiography*
- Transcranial doppler*
- Gastric tonometry*
- Respiratory gas monitoring
- Ventilatory parameters and lung characteristics: Inspiratory oximetry, Airway pressure and disconnection alarm, Inspiratory and expiratory tidal volume, lung compliance
- Arterial blood gas monitoring*
- Transcutaneous blood gas monitoring*
- Lower oesophageal sphincter contractility, Frontalis EMG, BIS and Auditory evoked potential*
- Neuromuscular junction monitoring

* Monitoring aids not discussed in this review.

**Monitoring of cardiovascular system and circulation: Stethoscope**

The use of a stethoscope, whether oesophageal or precordial, allows easy, cheap, safe, and continuous monitoring of circulation and ventilation. In addition to the auscultatory findings, in infants, stethoscope also allows monitoring of the beat to beat variation in the intensity of the heart sounds that varies with the stroke volume and is quite useful during inhalation induction of anesthesia. However, there are certain limitations such as the changes in the heart rate are quantitative and an ECG is necessary to know the existing rhythm, the changes in the heart rate occur rapidly during induction with halothane and are difficult to hear or interpret in the noisy environment of the operating room, moreover, the appreciation of changes in the heart sounds varies with the level of attention of the anesthesiologist that may vary during long procedures.
Electrocardiograph

Electrocardiography is mandatory for all Pediatric anesthesia and it is the most accurate method to measure heart rate and diagnose dysrrhythmias. However, it may give a false sense of security. In neonates and infants, a normal ECG may still be present in spite of a significant fall in cardiac output, particularly when halothane is used. Therefore an ECG is not a substitute for other monitors of tissue perfusion.

The normal ECG tracing changes with age and has several differences from adult tracing, as a rule heart rate decreases and the intervals (PR, QT) and duration (QRS) increase with increasing age (table I). The right ventricle predominates in early infancy due to larger mass and the T wave is larger because the electrodes are closer to the heart. This large T wave can lead to erroneous double counting of die heart rate, if both QRS and T are interpreted as a cardiac complex. Lead II gives the best P wave configuration and is therefore most useful to diagnose rhythm disturbances. Cardiac rhythm abnormalities are rare in children in the absence of a cardiac anomaly. Therefore their occurrence should prompt ruling out hypoxia, hypercarbia, light anesthesia, an overdose of halothane, malignant hyperthermia, rhabdomyolysis etc. However, dangerous levels of hypoxia and/or hypercarbia can exist without dysrrhythmias. Morphological changes of ECG complexes can help diagnose hyperkalemia due to various causes (prominent T wave), cardiac ischemia (ST segment changes), and hypocalcaemia (prolonged QT interval).

Table 1: Normal changes in ECG with age

<table>
<thead>
<tr>
<th>Age</th>
<th>Heart rate (beats per minute)</th>
<th>Axis</th>
<th>PR interval (range, sec)</th>
<th>QRS duration (sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>New born</td>
<td>110-160</td>
<td>+30 + 180</td>
<td>0.07-0.14</td>
<td>0.05</td>
</tr>
<tr>
<td>1 week – 1 month</td>
<td>105-180</td>
<td>+65 + 165</td>
<td>0.07-0.14</td>
<td>0.05</td>
</tr>
<tr>
<td>1 to 6 months</td>
<td>105-185</td>
<td>+10 + 110</td>
<td>0.07-0.15</td>
<td>0.05</td>
</tr>
<tr>
<td>6 months to 3 years</td>
<td>90-165</td>
<td>+5 + 105</td>
<td>0.07-0.16</td>
<td>0.055</td>
</tr>
<tr>
<td>3 to 8 year</td>
<td>65-140</td>
<td>+5 + 130</td>
<td>0.09-0.18</td>
<td>0.07</td>
</tr>
<tr>
<td>8 to 16 years</td>
<td>60-120</td>
<td>0 + 90</td>
<td>0.09+0.18</td>
<td>0.07</td>
</tr>
<tr>
<td>Adult</td>
<td>60-100</td>
<td>0 + 120</td>
<td>0.12+0.2</td>
<td>0.08</td>
</tr>
</tbody>
</table>

Pulse oximetry (SpO₂)

Pulse oximetry is a non invasive method and it gives beat to beat measurement of the oxygen saturation of the patient's haemoglobin, which is difficult to evaluate clinically because cyanosis is a late sign of hypoxemia. It has a considerable impact on patient safety and should be used for any anesthetic or sedation. The colour of blood is a function of oxygen saturation of haemoglobin and the colour change with oxygen

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saturation is due to the optical properties of the haemoglobin molecule, more precisely, the haeme. As the blood deoxygenates, it becomes less permeable to red light. The tissues then lose their pinkish appearance, taking on a blue tint. With oxygenation, haeme becomes transparent to red light hence imparting pinkish colour to the tissues.

Pulse oximetry consists of measurement of the absorption of two wavelengths of light (red 660, and infrared 940 nm) between a light source and a photo detector. The ratio of red to infrared waves absorption varies with the oxygen saturation of the haemoglobin; moreover, the pulsatile expansion of the arteriolar bed interposed between the source and the detector produces a pulse added increase in the path length of the light beam and a variation of its absorbance (fig. 1). Because only two wavelengths are used, pulse oximeters can measure only two forms of haemoglobin, oxyhemoglobin and deoxyhemoglobin. At each wavelength both the non pulsatile and the pulsatile absorbance measured and this allows the calculation of a ratio which has been empirically correlated to $O_2$ saturation measured in healthy non smoking adult volunteers; a ratio of 1 correspond to an $O_2$ saturation of 85%. Proper functioning of all the pulse oximeters is based on the assumption that the only pulsatile absorbance present is that of arterial blood, and the patient has normal haemoglobin A and no other form of haemoglobin.

![Image of pulse oximetry diagram]

**Limitations and Disadvantages:** There is a small but definite incidence of failure with pulse oximetry (table 2). Factors reported to contribute to higher failure includes ASA grade III, IV, V patients, young patients, patients undergoing orthopedic, vascular, or cardiac surgery, use of electro surgical unit (ESU), coexisting hypothermia, hypotension, chronic renal failure, low hematocrit, pigmented skin, and prolonged duration of
intraoperative procedure. Monitors that can analyze the signal and reject artifacts have fewer episodes of failure.

**Table 2: Physiological limitations of pulse oximetry**

**Oxyhemoglobin dissociation curve**
- Poor monitoring of hyperoxia
- Poor monitoring of hypoventilation if supplemental O₂ is given
- Relatively low PaO₂ for same SpO₂ in presence of HbF and β-Thalassaemia

**Presence of dyshemoglobin**
- CO Hb: Overestimation of SpO₂ by an amount close to CO Hb%
- Met Hb: If<20% SpO₂ decreases by about half of Met Hb%
- If >20% SpO₂ = 85%

**Reduced perfusion**
- Hypothermia: If < 30°C - underestimation of SpO₂
- If > 30°C - usually SpO₂ reading is correct
- Hypovolemia: Loss of signal

**Dyes**
- Bilirubin - No influence except if acute hemolysis (Co Hb)
- Nail polish - Effect variable, position probe sideways
- Meconium staining of the skin - Falsely low SpO₂
- IV methylene blue, indigo carmine, indocyanine green: short lived low SpO₂

**Venous pulsations**
- Tricuspid regurgitation - Falsely low SpO₂

Poor function with poor perfusion: Pulse oximeters require adequate pulsations to distinguish arterial blood absorption from venous blood and tissue absorption. Readings may be unreliable or unavailable if there is loss or diminution of the peripheral pulse (the various reasons are proximal blood pressure cuff inflation, leaning on an extremity, improper positioning, hypotension, hypothermia, cardiopulmonary bypass, low cardiac output, hypovolemia, peripheral vascular disease, or infusion of vasoactive drugs).

Under these conditions, some pulse oximeters blank the display or give a message such as low quality signal or inadequate signal while some others freeze the display. Methods to improve the signal quality include application of vasodilating cream, digital nerve block, administration of intra-arterial vasodilators or placing a glove filled with warm...
water in the patient’s hand. Warming cool extremities may increase pulse amplitude, provided cardiac output is not depressed.

difficulty in detecting high oxygen partial pressures: At high saturations, small changes are associated with relatively large changes in PaO₂. Thus the pulse oximeters have limited ability to distinguish high but safe levels of arterial oxygen from excessively elevated levels.

Erratic performance with irregular rhythm: Irregular heart rhythms can cause erratic performance. Erratic arterial pressure waveform may confuse the pulse oximeter so that it may not provide a reading.

Inaccuracies

Extrinsic interferences:
Results from factors external to the patient that artifactually alter light transmission between the LED's and photo detector (patient movement) or interfere with the very weak electrical signal developed by pulse oximeter probes (electro surgical unit). Because extrinsic interferences can be reduced or eliminated by engineering modifications, oximeters vary in their ability to reject it.

Optical interferences:
Stray light or light flickering at frequencies similar to the frequencies of the LEDs, including sun light, operating room lights, infrared heating lamps, and light sources of various scopes, xenon lights, etc can enter the photo detector resulting in inaccurate or erratic readings. Several ways exist of minimizing the effect of external optical interferences. These include selection and use of the correct sensor for the patient, and shielding the sensor from brighter light and other nearby sources. Some manufacturers try to minimize the effect of stray light by taking intermittent background readings when both the LEDs in the sensor are turned off and subtracting these readings from measurements taken by the photo detector when either LED is turned on.

Nail polish and Coverings:
Light absorption by nail polish is constant; therefore it is not expected to affect pulse oximeter accuracy. However, opaque coatings may significantly decrease light transmission, rendering oximeters inoperative. Some shades of black, blue and green nail polish may cause significantly lower saturation readings. One way to overcome this problem is to orient the probe so that it transmits light from one side of finger to the other side.
**Intrinsic interferences:**

Because current pulse oximeters use only two wavelengths, they are able to distinguish between only Hb and HbO₂. Clinically, this limitation does not present a problem if concentrations of methemoglobin, (met Hb) and carboxyhaemoglobin (CO Hb) are less than a few percent; however, when they are present in a higher concentration, oximeters may display erroneous and potentially dangerous data. The effect of dyshemoglobin on pulse oximeter readings depend on how their light absorption compares to that of Hb and HbO₂ at 660 and 940 nm. COHb₂ for instance absorbs almost identically to HbO₂ at 660 nm. Therefore, oximeters respond to COHb as if it were HbO₂ and the pulse oximeter reading is the sum of COHb and HbO₂ i.e., (if a patient had 90% HbO₂, 7% COHb₂ and 3% Hb a pulse oximeter would read 97% SpO₂). In patients with significantly elevated COHb (cigarette smokers, fire victims), this is dangerous for two reasons: 1) pulse oximeter overestimates the O₂ content of arterial blood, 2) the presence of COHb shifts the O₂ dissociation curve to the left, thus decreasing O₂ delivery to the tissues. Methemoglobin has approximately the same absorption coefficient at 660 and 940 nm. Oximeters interpret this 1:1 adsorption ratio as corresponding to a SaO₂ of 85%. Therefore, in the presence of metHb₂ pulse oximeter readings correspond to a weighted average of HbO₂ and metHb at 85%. Elevated levels of metHb may occur in patients exposed to drugs such as amyl nitrite, nitric oxide and nitroglycerine. Methylene blue and indocyanine green have significant absorption at 660 nm. Oximeters interpret this increased absorption as resulting from increased Hb implying lower oxygen saturation. However, this effect is short lived.

**Blirubinemia:**

Normally does not affect pulse oximetry.

**Low saturation:**

Pulse oximeters become less accurate at low saturations (<70%).

**Malpositioned sensor:**

Oximeters with sensors that are not well positioned vary greatly in their behaviour, and depend on the actual SpO₂, the manufacturer and the model. Use of too large a probe may result in inaccurate readings. To avoid these problems, sensor position should be checked frequently and inaccessible locations avoided wherever possible.

**Venous pulsation:**

Pulse oximeter design assumes that the pulsatile component of light absorbance is due to arterial blood. Prominent pulsations of venous blood may lead to underestimation of SpO₂. High airway pressures during artificial ventilation may cause phasic venous
congestion, which may be interpreted by the oximeter as a pulse wave. In some cases, it may be necessary to turn off ventilator momentarily to obtain a correct reading.

**Anemia:** The pulse oximeters tend to overestimate SpO\(_2\) at low saturations in patients with anemia.

**Skin pigmentation:** Studies show that pulse oximeters readings are erroneously high in patients with dark skin colour. The effect is more pronounced at low saturation.

**Pressure on the sensor:** Pressure on the sensor may result in inaccurate readings without affecting the pulse rate determination.

**Hyperaemia:** If a limb is hyperaemic, the flow of capillary and venous blood becomes pulsatile. In this situation, the absorption of light from these sources will be included in the saturation computations with resulting decrease in accuracy of the oxygen saturation measured by pulse oximetry.

**Alarms:** Ideally, pulse oximeters should have an alarm that is distinct and not easily confused with other alarms or sounds in the operating room. When alarms are disabled, a message should be displayed. A high percentage of pulse oximetry alarms are spurious or trivial. False alarms are most commonly caused by motion artifact but also are associated with poor signal quality, sensor displacement and electro-surgical interference. Some pulse oximeters have sophisticated methods to reject noise and motion artifacts. Analyzing the arterial waveform can reduce false alarms. The arterial waveform is characterized by a rapid rise and slow decay, whereas artificiually pulsations caused by motion are characterized by equally fast rise and decay. By rejecting these, motion artifacts are filtered out. Pulse oximeters that display the plethysmography waveform have an advantage over those that display only amplitude of the signal, because this allows the operator to assess the quality of the signal and to observe noise such as motion artifact that may alter its accuracy.

**Patient complications:** In general very few complications have been reported; these include corneal abrasions, pressure and ischemic injuries, burns and electric shock etc. Pressure necrosis can occur if the probe is left in place on a single site for a long time. Therefore the probe should be such positioned that no pressure is applied to the probe or the site of measurement. Further, the probe site should be checked frequently and the probe position changed if necessary.

**Blood pressure**

The methods of blood pressure measurement are non-invasive or continuous invasive measurement. The normal value of blood pressure varies according to age (table 3). The most accurate method is invasive measurement after catheterization of a peripheral artery; however, the method is restricted to critically ill children or major surgical procedures, and the non-invasive method is used during routine surgical procedures.
Table 3: Normal values of blood pressure according to age

<table>
<thead>
<tr>
<th>Age</th>
<th>Systolic and diastolic pressure (mmHg)</th>
<th>Mean arterial pressure (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neonate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 1kg</td>
<td>44</td>
<td>33</td>
</tr>
<tr>
<td>1 kg</td>
<td>49</td>
<td>34</td>
</tr>
<tr>
<td>2 kg</td>
<td>54</td>
<td>41</td>
</tr>
<tr>
<td>3 kg</td>
<td>62</td>
<td>46</td>
</tr>
<tr>
<td>&gt; 3kg</td>
<td>66</td>
<td>50</td>
</tr>
<tr>
<td>1-6 months</td>
<td>80/46</td>
<td>63</td>
</tr>
<tr>
<td>6 months–1 year</td>
<td>89/60</td>
<td>70</td>
</tr>
<tr>
<td>1-2 years</td>
<td>99/64</td>
<td>76</td>
</tr>
<tr>
<td>2-4 years</td>
<td>100/65</td>
<td>77</td>
</tr>
<tr>
<td>4-12 years</td>
<td>105/65</td>
<td>78</td>
</tr>
<tr>
<td>12-15 years</td>
<td>118/68</td>
<td>85</td>
</tr>
</tbody>
</table>

Non invasive blood pressure measurement (NIBP)
Automated Oscillometry is the commonest method used. It allows measurement of systolic, diastolic and mean blood pressures. Monitors based on this principle are easy to use and accurate. The components are an inflatable cuff with a tube, a microprocessor and a transducer, and a display unit. The cuff is automatically inflated above systolic pressure and slowly deflated in steps controlled by the microprocessor. At each stage both mean cuff pressure and oscillations resulting from the arterial pulse wave are sensed and stored. Systolic BP corresponds to the cuff pressure at which the oscillations rapidly increase, the mean arterial BP corresponds to the lowest cuff pressure at which maximal oscillations are recorded, and diastolic BP to the cuff pressure at which the oscillations fade. The size of the cuff is very important, if the cuff is too small, the blood pressure is over read, and it is under read if the cuff is too large. As a general rule, the cuff should cover (2/3 rd of the upper arm; the width of the cuff bladder should be 40% of the mid circumference of the limb. The guidelines to obtain accurate data with a NIBP device include:

1. Use of a proper sized BP cuff.
2. Squeeze out all residual air from the cuff before connecting it to the tubing.
3. Wrap the cuff snugly around the limb.
4. Keep the cuff and the heart at the same level.
5. In case of malfunction of the apparatus, check the child's condition first.
6. Common causes of malfunction are: Inappropriate size of the BP cuff, a leak in the cuff-tubing or connections, and extrinsic cuff compression.
Non invasive blood pressure can also be measured by a sphygmomanometer using either Palpatory or auscultatory method.

**Finger blood pressure (Finapress)**

Finapress consists of a small pneumatically inflated cuff that is applied to the phalanx of a finger. The cuff also contains an infrared photo plethysmography, the wavelength of which is absorbed by haemoglobin that allow arterial pulsation to be sensed. The finger cuff pressure is continuously adjusted by a servo-controlled loop to keep the infrared signal, and hence the finger blood volume, constant over the arterial pressure cycle. The fluctuations of pressure in the cuff are the same as in the patient's artery, which allow continuous monitoring of blood pressure. The use of this device is limited to children above 5 years of age. Arterial tonometry is the other method under evaluation.

**Intra-arterial measurement**

Intra-arterial blood pressure measurement gives precise and instantaneous information about blood pressure changes. It also allows sampling of blood for measurement of arterial blood gases, electrolytes, blood glucose, and other parameters. The indications are based on hemodynamic and pulmonary considerations, the preoperative physical status of the child, the invasiveness of the procedure and the likelihood of blood volume changes and fluid shift. In general, all peripheral arteries are suitable for cannulation; radial and femoral arteries are most commonly used. The temporal artery is no longer used due to a risk of cerebral embolization during flushing. The femoral artery is usually easy to locate and cannulated, especially in cases of low cardiac output. Although, there is a theoretical increased risk of contamination due to the proximity of perineum, the incidence of infection is no greater than with radial catheters. However, the incidences of perfusion related complication is higher. The umbilical artery is accessible during the first 3 days of life; however, it is not suitable in cases of intra-abdominal surgery because of its mobilization during surgery.

Usually 20 or 22 G non tapered cannula is used for arterial cannulation depending on the weight of the child; 24 G is reserved for babies weighing < 3 Kg. Constant flushing with heparinized saline (heparin 0.5-1 IUml⁻¹) with an automatic pressurized system at a rate of 1-3 mlhr⁻¹ is recommended. The tubing connecting the cannula to the transducer should be rigid and as short as possible. The transducer and the connecting tubing should be carefully desired and zeroed at the mid axillary line; in case of Lead up or sitting position (neuro surgery) it should be zeroed at the level of brain (outer canthus of the eye) to measure brain perfusion pressure. During arterial pressure monitoring, the position of the zero base line should be checked regularly and corrected if necessary. The dressing should allow visualization of the skin proximal and distal to the catheter to detect any blanching or cyanotic discolorations which are early signs of
thrombotic complication. The stopcocks of the arterial line should be carefully labelled to avoid accidental intra-arterial injection of anesthetic agents, antibiotics, etc. Blood sampling should be performed slowly to avoid collapse of the artery and damage to its endothelium; similarly tubing should be flushed gently. Too rapid or forceful flushing results in blanching of the skin proximal to the cannula and in transmission of the iatrogenic pressure wave and embolization to the distal sites such as cerebral or splanchnic circulation therefore flushing with 0.5 ml flush solution over 5 seconds is recommended in neonates.

Local hematoma is the most common complication; however, it resolves quickly. The arterial thrombosis rate varies with the age of the child, and recannulation usually occurs during the following days. The most important factors implicated are cannula to artery diameter ratio in excess of 0.7, transfixion of artery during cannulation and presence of hematoma at the site of cannulation. Removal of the cannula is recommended in cases of increasing difficulties with blood sampling or the appearance of cyanotic discoloration of the skin distal to the entry site. Ischemic damage is more likely in cases of low cardiac output or use of vasopressors and can lead to serious complications (amputation). Local infection is rare if the rules of the asepsis are observed and the cannula is left in place for less than 4 days. Other complications described are nerve damage by the needle or by a compressive hematoma, tendon sheath injury, accidental intra-arterial injection, formation of arterio-venous fistula, and occurrence of compartment syndrome.

Central venous pressure
In children with normal right ventricular function, its value is well correlated with intravascular volume and left ventricle preload. But, more than the absolute value, the trend of readings is more important in therapeutic management. Normal CVP is 2-6 mmHg or 3-12 cm H$_2$O. However, it should be interpreted in association of other hemodynamic parameters and surgical events. The indications of CVP measurement are: major surgery involving significant fluid shift or blood loss, cardiac surgery, procedures carrying a high risk of air embolism or in which infusion of inotropic drugs is necessary. The central venous circulation is usually accessed by placing a catheter in superior vena cava through internal jugular vein. The pressure transducer should be placed at the level of the mid-axillary line and the zero base line should be checked regularly. The measurement should be made at the end of expiration to avoid respiratory interference and only when a good CVP waveform is present on the monitoring screen. In case of difficulty or contraindication to the catheterization of IJV, the common iliac vein or inferior vena cava can also be used provided the measurement is made at the end of expiration and there is no obstruction of flow from IVC to right atrium (ascites, abdominal tumour, or abdominal surgery, etc).
**Temperature monitoring**

Body temperature should be monitored during any anesthetic lasting more than 30 minutes. Intraoperative temperature monitoring is necessary to detect hypothermia and hyperthermia, manipulate body temperature (brain protection during cardiac surgery) and to prevent complications associated with variations from normal temperature. Hypothermia directly depresses the level of consciousness and interferes with recovery from anesthetic agents due to delayed metabolism. In children, thermoregulation is less efficacious during anesthesia than in adults.

Temperature probes are thermistor, thermocouples or liquid crystals. The most recent development is the infrared tympanic thermometer. The response time of these devices is short and accuracy is close to 0.2°C. The interpretation of the body temperature depends upon the site of measurement. The site for the intraoperative temperature measurement should be chosen according to the child's age and the procedure undertaken. In anesthetized infants and children undergoing superficial surgery, there is indifference between tympanic, oesophageal, rectal or axillary temperature measurements. The core temperature can be monitored with adequate accuracy in the distal oesophagus, nasopharynx, or tympanic membrane for superficial procedures. Monitoring temperature at two sites (usually oesophagus and rectal or vesicle) is critical during procedures in which large and rapid changes in temperature occur (cardiac bypass). The large gradients are indicative of non-uniform heat distribution or poor cardiac output. The skin temperature is influenced by many factors such as cardiac output, anesthetic technique, and environmental temperature; it shows great variability in relation to core temperature. Its intraoperative monitoring is of little use during anesthesia.

**Urine output**

Adequate urine output is a sign of good renal perfusion and is an indirect indicator of adequate volume loading and peripheral perfusion of the tissues. Urine output should be monitored when, large shifts of body fluids are anticipated, in cases of anticipated, blood loss in excess of 25% estimated blood 25% estimated blood volume, during cardiopulmonary bypass, transplant, surgery, and neurosurgery or during long surgical procedures to prevent bladder distension. Urine output of 0.5-1 mlkg⁻¹h⁻¹ indicates adequate renal perfusion; however, it should be interpreted cautiously in cases of diuretic use, intravenous contrast media and glycosuria. Large amounts of urine can occur in presence of diabetes insipidus, head injury, post obstructive diuresis, salt loosing nephropathy or other metabolic disorders. Patency of the bladder catheter or absence of distended bladder should be checked before establishing the diagnosis of anuria or oliguria. During the first week of life, urine output alone is not a good indicator.
of changes in intravascular volume or cardiac output because the neonatal kidney has a limited capacity to concentrate or dilute urine.

**Oxygenation and ventilation**

**Respiratory gas monitoring:** Measurement of inspired and expired O\(_2\), CO\(_2\), and volatile agents, and computed MAC (minimum alveolar concentration) value allows the anesthesiologist to continuously check for proper functioning of anesthesia delivery unit, adequacy of the patient's ventilation, and quality and adequacy of anesthesia.

**Inspired oximetry:** The measurement of oxygen concentration in the inspired and expired gases is achieved by either placing sensors in the inspiratory and expiratory limbs or by mass spectrometry or by infrared analysis. The measurement and inbuilt alarm system allows detection and prevention of accidental delivery of mixture of hypoxic gases. In addition, the measurement of expired O\(_2\) allows the evaluation of pre-oxygenation before induction of anesthesia in patients at high risk for hypoxia such as anticipated difficult intubation, pulmonary disease, and obesity.

**Measurement of volatile anesthetics:** This measurement allows assessment of the function of the vaporizer in use, detection of volatile anesthetic in use, and its concentration during low flow anesthesia, and indirectly the depth of anesthesia. The assessment is made by either mass spectrometry or infrared analysis.

**Capnography:** Capnography is the graphic display of instantaneous CO\(_2\) concentration versus time (time capnogram). ASA consider it a standard of care and a mandatory monitoring in patients undergoing endotracheal anesthesia. Infrared method is the commonest method used for capnography, infrared rays are absorbed by non-elementary gases (gases composed of dissimilar atoms). The intensity of the infrared rays projected through a gas mixture containing CO\(_2\) is diminished by its absorption; this loss in intensity is proportional to the amount of CO\(_2\) present in the mixture. Capnography provides information about CO\(_2\) production, pulmonary perfusion, alveolar ventilation, respiratory pattern and elimination of CO\(_2\) from the anesthesia circuit. Capnography allows rapid detection of malposition of tracheal tube, ventilatory failure, circulatory failure, and defective breathing circuit. There are two types of capnography namely side stream and mainstream capnography (fig. 2a and 2b). The comparison of the two methods is shown in table 4.
Fig. 2a: Side stream canograph

Fig. 2b: Main stream canograph
Table 4: Comparison of main stream and side stream capnography

<table>
<thead>
<tr>
<th>Type</th>
<th>Advantage</th>
<th>Disadvantage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Main stream</td>
<td>No response delay</td>
<td>Delicate sensor close to the patient; risk of kinking, disconnection, burn</td>
</tr>
<tr>
<td></td>
<td>Accurate oven at rapid rate</td>
<td>Increased dead space in infants</td>
</tr>
<tr>
<td></td>
<td>No scavenging needed</td>
<td>Presence of blood, water or secretions in the cuvette interferes with accuracy</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Difficult to use in non-intubated patient</td>
</tr>
<tr>
<td>Side stream</td>
<td>Light adapter distal sampling easy</td>
<td>Transport causes response delay</td>
</tr>
<tr>
<td></td>
<td>It can be used in non intubated patient</td>
<td>Choice of sampling site critical for accuracy</td>
</tr>
<tr>
<td></td>
<td>Measurement of other gases possible</td>
<td>Risk of obstruction of sampling</td>
</tr>
<tr>
<td></td>
<td>Accurate zeroing</td>
<td>Accuracy affected by rapid respiratory rate (&gt;40 min(^{-1}))</td>
</tr>
</tbody>
</table>

A typical capnograph has four phases; different phases indicate concentration of CO\(_2\) in the expired or inspired gases against time (fig. 3). At the end of inspiration, if there is no rebreathing, the airway and the lungs are filled with CO\(_2\), free gases. Exchange of the gases in the alveoli causes build up of CO\(_2\) in the alveolar gases. At the beginning of expiration, the capnograph remains at the base line since the gases exhaled from the airways do not take part in the gaseous exchange; as the expiration continues the CO\(_2\) concentration begins to increase because of the contribution of gases from alveoli and is reflected by a sharp rise in the capnograph and it is followed by a slightly ascending plateau phase that is interrupted by next inspiration. The shape of the capnograph correlates with the characteristics of the airways, the ventilation-perfusion relationship, the mechanical properties of the lung parenchyma, and the ventilator settings. Some capnographs encountered during various clinical conditions are shown in figure 4. The table 5 shows interpretation of some capnographs.

Table 5: Interpretation of some capnographs

1. Sudden disappearance of capnograph indicates: Disconnection, obstruction, cardiac arrest, embolism or automatic zeroing of the device
2. Gradual decrease of ETCO\(_2\), indices hypo ventilation or decreased cardiac output.

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3. Slow increase of ETCO₂, hypoventilation, fever, or exhausted soda lime.
4. Increased inspiratory CO₂ (PiCO₂): rebreathing. Artifact due to rapid respiratory rate.
5. Rapid increase in ETCO₂: Malignant hyperthermia.
6. Transient and short lived increase in ETCO₂: Blood or sodabicarb administration.
7. Gradual increase ETCO₂ and/or PiCO₂: Faulty unidirectional valve.
9. Sharp upstroke of the plateau phase: Bronchospasm or kinking of trachea tube.

Airway pressure, disconnection alarm, and Inspiratory and expiratory tidal volume: A simple manometer that measures the pressure of the gases delivered to the airways is incorporated into most mechanical ventilators.
A change in the airway pressure may reflect change in the lung or the chest wall compliance provided the ventilator is of time cycled volume preset variety. Chest wall compliance may be influenced by the degree of muscle relaxation, surgical manipulation and the position of the patient; and the lung compliance by accumulation of secretions, or the development of pneumothorax, etc. Increased resistance to the airflow caused by bronchospasm or obstruction of the tracheal tube is reflected by an increased peak airway pressure. Disconnection alarm is activated if the airway pressure decreases below a preset minimum for a preset time interval. Modern ventilators also incorporate a respirometer or a flow transducer in the inspiratory and/or expiratory limb to monitor inspiratory and expiratory volume delivered to the patient. Expiratory volume measurement allows detection of leaks in the breathing circuit. Absence of leak around endotracheal tube on application of appropriate PEEP to achieve a peak airway pressure of 25 cm H₂O allows objective detection of oversized endotracheal tube.

**Neuromuscular monitoring**

Muscle relaxants act by occupying post-junctional acetylcholine receptors. More than 70-75% receptors are needed to be occupied before the appearance of a detectable block, and the paralysis is complete at 90-95% receptor occupancy. Therefore, adequate muscle relaxation corresponds to a narrow range of 85-90% receptor occupancy (therapeutic window). Neuromuscular blockade (NMB) following administration of a muscle relaxant may vary considerably in neonates and infants; therefore, the NMB must be monitored to titrate its need according to the patient's response and the depth of blockade needed by the procedure, to determine the optimal time to intubate in situations where coughing and bucking must be avoided, to determine the appropriate time to reverse NMB, and to diagnose unusually prolonged blockade or incomplete reversal. Neuromuscular monitoring consists of stimulating a peripheral nerve and evaluating the resultant motor response of the supplied nerve. The technique is dependent of many factors, such as the pattern of stimulation, the sensitivity of the muscles tested, the age and body temperature of the patient, the method used to assess the evoked muscular response, and the pharmacodynamics of the muscle relaxant used. Nerve muscle stimulation response can be monitored at several places such as, at thumb, great toe and eye by stimulating ulnar nerve, posterior tibial nerve and facial nerve respectively. However, there are differences in sensitivity to NMB between the muscles evaluated and the ones controlling vital functions. The most resistant muscles to non depolarizing muscle relaxants are the diaphragm, the vocal cords, the abdominal wall muscles, and the orbicularis occuli. The most sensitive muscles are the upper airway muscles (tongue, pharynx), the masseter, the adductor pollicis (great toe), and the flexor hallucis brevis (thumb). Thus stimulation of the facial nerve is the best indicator of relaxation of laryngeal muscles and the diaphragm (before intubation) and
stimulation of the ulnar or posterior tibial nerve is best for evaluation of relaxation of upper airway muscles during recovery. The depth of NMB is assessed by evaluating muscle response to various modes of stimulation. The various modes of stimulation are - Single twitch (ST) Train of four (TOF), Tetanus, Post-tetanic count (PTC) and Double burst stimulation (DBS). TOF, PTC and DBS are the modes routinely used during anesthesia. TOF and PTC are used for monitoring depth of relaxation while TOF and DBS are used to ensure adequate recovery from relaxant effect.

Characteristics of various modes of stimulation: The summary of various modes of stimulation and its usefulness in detecting NMB is described in table 6. The graphic pattern of various modes of stimulation is shown in figure 5.

Table 6: Characteristics of nerve stimulation
<table>
<thead>
<tr>
<th>Feature</th>
<th>Single-twitch</th>
<th>TOF</th>
<th>Tetanus</th>
<th>DBS</th>
<th>PTC</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Current strength</strong></td>
<td>Supramaximal</td>
<td>Supra or submaximal</td>
<td>Supra or submaximal</td>
<td>Supra or submaximal</td>
<td>Supra or submaximal</td>
</tr>
<tr>
<td><strong>Frequency</strong></td>
<td>0.1-1 Hz</td>
<td>2 Hz (four stimuli)</td>
<td>30-50 Hz for 5 sec</td>
<td>3 impulses at 50 Hz repeated after 750 msec</td>
<td>30 Hz for 5 sec followed by ST at 1 Hz</td>
</tr>
<tr>
<td><strong>Interval between successive stimulation</strong></td>
<td>5 sec</td>
<td>12 sec</td>
<td>6 min</td>
<td>12-15 sec</td>
<td>6 min</td>
</tr>
<tr>
<td><strong>Sensitivity of manual detection</strong></td>
<td>Not sensitive</td>
<td>Not sensitive at TOF ratio of 0.5 – 0.7</td>
<td>Sensitive</td>
<td>Highly sensitive</td>
<td>Sensitive</td>
</tr>
<tr>
<td><strong>Receptor occupancy detection</strong></td>
<td>75-90%</td>
<td>70-90%</td>
<td>70-90%</td>
<td>70-90%</td>
<td>&gt;90%</td>
</tr>
<tr>
<td><strong>Sensitive for detection of subtle block</strong></td>
<td>Not sensitive</td>
<td>Sensitive</td>
<td>Sensitive</td>
<td>Sensitive</td>
<td>Not applicable</td>
</tr>
<tr>
<td><strong>Monitoring of profound block</strong></td>
<td>Not useful</td>
<td>Not useful</td>
<td>Not useful</td>
<td>Not useful</td>
<td>Useful</td>
</tr>
</tbody>
</table>

**Train of four**

In this mode of stimulation (fig. 5) for monitoring of NMB, four successive stimuli are delivered at 2 Hz (every 0.5 sec). The stimulation of nerve causes release of acetylcholine, the neurotransmitter, which elicits contraction of normal muscle. Because of the wide margin of safety, TOF response is unaffected if the receptor occupancy is < 70% while there would be no response if the receptor occupancy by the NMB is > 90-95%. Within this narrow range of 70-95% receptor blockade, the response varies between no responses to normal response (fig. 6) and is interpreted as - TOF response 1, 2, 3, and all 4, and as TOF ratio. For routine abdominal surgery TOF 1 or 2 is sufficient that roughly correspond to 80% receptor blockade.
**Post tetanic count**

During profound neuromuscular blockade, when there is no response to single twitch, tetanic or train of four stimulation, the blockade can be quantified by using PTC. The evaluation is based on the principle of post tetanic facilitation. Following tetanic stimulation, there is an increased availability of acetylcholine. Thus an increased amount of acetylcholine is released in response to nerve stimulation and a single twitch elicited after cessation of tetanus may be stronger than the pretetanic control. Tetanus at 50 Hz is applied for 5 sec then after 3 sec single twitches are applied at 1 Hz (fig. 5). The number of post-tetanic twitches elucidated is called the post-tetanic count. The number of post tetanic twitches correlates inversely with the time for spontaneous recover. A PTC of S-11 indicates imminent return of TOF stimulation following various muscle relaxants. The main application of PTC is in evaluating the intensity of NMB when there is no response to single twitch or TOF as may occur following a large dose of muscle relaxant.

**Double burst stimulation (DBS)**

It consists of 2 short bursts of 60 msec at 50 Hz separated by 0.75 sec (fig. 5). The interval between the 2 tetanic stimuli allows the muscle to relax completely, so that the responses are perceived as 2 separate twitches. These bursts fatigue the neuromuscular junction more than single twitches thus exaggerating fade. This type of stimulation is
mainly used to detect residual NMB. DBS is more sensitive than TOF for the assessment of recovery from NMB. Using TOF, clinical fading is no longer detected when the TOF ratio is $0.44 \pm 0.033$, but remains detectable by DBS until the TOF ratio is $0.67 \pm 0.04$. This is because the 2 DBS elicited contraction are stronger and easier to compare than first and fourth response to TOF.

**Blood chemistry**

The presence of arterial or central venous catheter allows sampling of blood to measure blood gases, electrolytes, glucose and other parameters. One should note that the normal values of arterial $O_2$ tension and blood glucose in neonates is significantly lower than normal adults. The neonate has a poorly developed system for maintaining adequate plasma glucose concentration and, therefore, is susceptible to the development of hypoglycaemia. By definition, hypoglycaemia is a plasma glucose concentration of $< 25$ mg% for a preterm neonate and $< 35$ mg% for a neonate younger than 3 days of age. The plasma glucose concentration should be greater than 45 mg% for a term neonate. Hyperglycemas should also be avoided as plasma concentration in excess of 125 mg% can produce osmotic diuresis and dehydration. The Pa$O_2$ at birth is 70-75 mmHg; higher values, > 80 mm Hg for prolonged periods, may be associated with an increased incidence and severity of retinopathy. To reduce the risk of retinopathy in a susceptible infant, it is recommended that the Pa$O_2$ be maintained between 60-80 mmHg. Continuous intra-arterial blood gas monitoring is available. Similarly transcutaneous blood gas measurement is also available; however, the technique is not widely used because of various limitations.
Chapter 7 - ANESTHETIC CONSIDERATIONS FOR SPECIALIZED SURGERIES PECULIAR TO PEDIATRIC AGE GROUP

The most common conditions encountered by the Pediatric anesthesiologist, those pose befitting challenges are:

I. Congenital diaphragmatic hernia
II. Tracheo-oesophageal fistula
III. Foreign body removal from airways
IV. Laparoscopic surgeries in children
V. Pyloric stenosis

I. CONGENITAL DIAPHRAGMATIC HERNIA

Introduction

Congenital diaphragmatic hernia (CDH) may present as a life threatening emergency which warrants rapid resuscitation; correction of acidosis fluid deficit, hypothermia and baby prepared for emergent surgery. CDH occurs in approximately 1:2500 live births, with a male to female ratio of 2:1. Left diaphragm is more often (95%) involved than right (5:1).

Embryology

The failure of closure of postero-lateral aspect of the diaphragm (foramen of Bochdalek) or non fusion of anterior, central and lateral portions of the diaphragm retrosternally (foramen of Morgagni) by the 7-8th fetal week, produce defects in diaphragm. Slowly the growing gut and abdominal contents migrate into the pleural cavity causing ventilatory and circulatory crisis with pulmonary hypoplasia. The severity of the symptoms depend on the age of the intra-uterine life when the thorax gets invaded.

Pathophysiology:

The spectrum of clinical presentation varies, depending upon the period of lung invasion and pulmonary agenesis. Developmental defect in the diaphragm leads to herniation of the abdominal viscera into the pleural cavity leading to pulmonary hypoplasia and mediastinal shift with consequent effects. The commonest site being right or left foramen of Bochdalek.
As the contralateral lung is always spared, the infant is capable of sustaining respiration and gradually ipsilateral lung expands and becomes functional. Subsequent picture depends on the development of pulmonary hypoplasia and pulmonary vasculature.

**Causes of hypoxemia:**

- Atelectasis; compression of developed lung.
- Persistent pulmonary hypoplasia with decreased bronchopulmonary generations and pulmonary vasculature.
- Persistent pulmonary hypertension, increasing right to left (R→L) shunt through the ductus arteriosus or foramen ovale.
- Systemic hypotension following kinking of major blood vessels.
Classification based on anatomical defect in the diaphragm:

1. Absent diaphragm - very rare
2. Diaphragmatic hernia
   - Posterolateral Bochdalek 80% (Left > Right)
   - Anterior (Morgagni) 2%
   - Para-esophageal 15-20%
   - Eventration rare

Assessment of severity of pulmonary hypoplasia

- PAO$_2$ – PaO$_2$ > 500 mmHg breathing 100% O$_2$ - predicts non survival; 300-500 mmHg - survival uncertain and values <400 mmHg; has better prognosis.
- Cardiac catheterization, echocardiography, colour doppler pulmonary angiography.
- Bohn's index prognosticates information as 'ventilatory index' (V.I.): product of mean airway pressure X respiratory rate..
- Neonate with PaCO$_2$ < 40 mmHg and VI < 1000 always survive, while PaCO$_2$ > 50 mmHg, VI < 1000 or PaCO$_2$ < 40 mmHg, VI > 1000 usually die.

Signs and symptoms

- Cyanosis and tachypnea
- Scaphoid abdomen, barrel chest, bowel sounds heard in the chest
- Heart sounds heard on right hemithorax (apparent dextrocardia)
- Absent breath sounds on left side chest
- CXR confirms gas filled bowel in the left hemithorax with mediastinal shift
- Radio opaque dye through the nasogastric tube may delineate the bowel in the chest

Monitoring

Precordial / oesophageal stethoscope, pulse oximeter both above / below the nipple for preductal and postductal SpO$_2$, capnography, inspiratory pressure, FiO$_2$, acid base status, ECG ,invasive blood pressure in right radial artery for preductal PaO$_2$, CVP volume status and right ventricular performance, thermoregulation - oesophageal, rectal temperature.

Surgical Correction

Repair of hernia is not a surgical emergency unless the contents of hernia are incarcerated. Improved ventilation and maintenance on ECMO or other treatment strategies to control pulmonary hypertension, ductal shunting and ineffective oxygenation are continued till pH is reversed and lung functions improve (7-10 days).
Patients not amenable to improve on ECMO or with 3 weeks treatment have bad prognosis. Subcostal or thoraco-abdominal incision is taken, herniated abdominal contents are pulled back and the muscular defect in the diaphragm repaired. If closure of abdomen exacerbates respiratory distress only skin may be sutured or a separate pouch be created temporarily. Sudden hypotension, bradycardia, decreased SaO\textsubscript{2} and pulmonary compliance suggest tension pneumothorax. Contralateral pneumothorax can be disastrous. Decreased venous return due to tight abdomen should be suspected if no improvement is seen after treatment of pneumothorax.

**Preoperative management**

- Assess associated anomalies.
- Hypothermia increases O\textsubscript{2} consumption, optimal environmental temperature suggested is 30\textdegree C-40\textdegree C.
- Arterial blood gases, complete blood count, serum electrolytes, blood sugar, cross matching.
- Correct metabolic acidosis (glucose, fluids) and respiratory acidosis (proper ventilation/sodium bicarbonate) prior to surgery. Sodium bicarbonate may be given empirically as diluted 0.5 mEqml\textsuperscript{-1}, (2-4 mEqkg\textsuperscript{-1}) I.V. infusion at < 1 mEqKg\textsuperscript{-1}min\textsuperscript{-1}.
- Venous access must be secured in the upper arm; neck veins reserved for ECMO. Central vein access is via umbilical or femoral veins.
- Use of vasodilators - tolazoline, prostacycline, dipyridamole, nitric oxide.
- Minimize sympathetic discharge by high dose opioids.
- Nasogastric suction for gastric decompression.
- Transport to OT with manual ventilation.

**Anesthetic considerations**

- Infant in semi-sitting position is warmed with warming devices, humidified inspired gases and warm transfused fluids at 37\textdegree C. After pre-oxygenation, atropine 0.02 mgkg\textsuperscript{-1} is administered I.V. Awake intubation is preferred. Alternatively infant is induced with halothane / Sevoflurane in O\textsubscript{2} and intubated while breathing spontaneously. Avoid mask ventilation, to obviate gastric distension and respiratory embarrassment.
- Gentle hand ventilation is preferred to avoid ipsilateral or contralateral pneumothorax. Anesthesia is maintained with volatile agent in 100\% O\textsubscript{2} or with 0.5 mgkg\textsuperscript{-1} ketamine with titrated dose 1-3 \textmu kg\textsuperscript{-1} fentanyl. PPV is avoided till intubation.
Patient in shock and severe hypoxemia is maintained on supplemental O\textsubscript{2}, non depolarizing muscle relaxants (pancuronium / vecuronium) and analgesics. Inhalational agents, opioids (fentanyl) and muscles relaxants (pancuronium) are added in titrated doses.

N\textsubscript{2}O is avoided. It may produce intrathoracic gut distension, inability to close abdomen and increased intra abdominal pressure, leading to precipitous hypotension.

Selection of appropriate FiO\textsubscript{2} depends upon severity of pulmonary dysfunction. To avoid hypoxia increasing R \rightarrow L shunt of Desaturated blood, inhaled gases with higher O\textsubscript{2} content is suggested. PaO\textsubscript{2} should be optimally kept between 80-100 mmHg or arterial O\textsubscript{2} saturation at 95-98%.

**Ventilation:**
Infant is ventilated with small tidal volume and low inflation pressure (< 20 mmHg) to prevent contralateral pneumothorax. Respiratory rate (60-120 min\textsuperscript{-1}) is adjusted to achieve hypocarbia (PaCO\textsubscript{2} 25-30 mmHg), lower pulmonary vasoconstriction and minimize R \rightarrow L shunt through the ductus arteriosus.

**Pulmonary hypertension and management**
Factors responsible for pulmonary hypertension may be variable but reversible PVR, due to medial hyperplasia of pulmonary arterioles stress surfactant deficiency acidosis fluctuating pulmonary blood volume / ventilator induced lung injury or fixed elevation of PVR, due to underlying pulmonary hypoplasia which requires therapies aimed at improving pulmonary development.

**Treatment**
- Continue ventilation in ICU using fentanyl 3\textmu kg\textsuperscript{-1}hr\textsuperscript{-1}, pancuronium 0.1 mgkg\textsuperscript{-1}hr\textsuperscript{-1} to blunt autonomic cardiovascular response (pulmonary vasoconstriction) to stimulation.
- Minimize endotracheal suction, to avoid transient hypoxemia.
- Hyperventilate the neonate with low V\textsubscript{T} and high RR (60-120 min\textsuperscript{-1}) to maintain pH of 7.55-7.60 and induce pulmonary vasodilation.
- Restrict fluids to 2-4 mlkg\textsuperscript{-1}hr\textsuperscript{-1}.
- Administer pharmacologic pulmonary vasodilators morphine, chlorpromazine, prostaglandin E\textsubscript{1} and inhaled nitric oxide.
- Severe lung hypoplasia and refractory pulmonary hypertension with arterial O\textsubscript{2} saturation <50 mmHg at FiO\textsubscript{2} of 1.0 needs ECMO therapy immediately to avoid progressive pulmonary injury. ECMO is associated with 50-60% survival rate.
Nitric oxide (NO) at 20-80 ppm a endothelial derived relaxing factor is selective pulmonary vasodilator with no effect on systemic circulation and is immediately inactivated on exposure to Hb.

High frequency oscillatory ventilation (HFOV) has been used as pulmonary ventilatory therapy to improve oxygenation.

**Fluid replacement**
- Correct preoperative deficit, provide maintenance fluid and replace intraoperative blood loss. Glucose should be given as neonates have decreased glycogen reserves.
- Maintenance fluid 5% dextrose in 1/4th – ½ strength saline at 4 mlkg⁻¹hr⁻¹.
- Intraoperative and third space losses are replaced by (Ringer’s lactate or saline 6-8 mlkg⁻¹hr⁻¹. Each ml blood loss is replaced by 3 ml Ringer lactate or 1 ml of 5% albumin.

**Postoperative management**
Postoperative intubation and ventilation should be planned. The FiO₂ is adjusted to maintain PaO₂ over 150 mmHg till the infant is slowly weaned in 48-72 hrs to avoid honeymoon phenomenon, characterized by early smooth course followed by development of pulmonary vasoconstriction and lethal persistent pulmonary hypertension, hypercarbia and acidosis. Do not extubate till the child is fully awake, breathing spontaneously and rhythmically, open eyes and maintains SpO₂ of 100%.
Mortality depends on the reappearance of Fetal circulation due to hypoplasia associated congenital anomalies (especially cardiac), inadequate pre operative preparation (hypothermia, hypoxia.. acidosis) shock and pneumothorax.

**TRACHEOOESOPHAGEAL FISTULA**

**Introduction**
Tracheo-oesophageal fistula (TOF) is one of the most frequent congenital defect (1 in 4000 live births).
Embryologically, it is attributed to incomplete closure of the laryngo-tracheal groove. The defect results from imperfect division of the foregut into the anteriorly positioned trachea and posteriorly positioned oesophagus, in 4th – 5th week of intra-uterine life. This may be part of the larger constellation of anomalies as VATER or VACTERL association (vertebral / ventriculo septal defect, anal atresia, cardiac, TOF, oesophageal atresia, renal, limb, anomalies).
Risk categorizations

Spitz classification
- Grade I - Body weight > 1500 g without cardiac disease (survival 97%)
- Grade II - Body weight > 1500 g and major cardiac disease (survival 59%)
- Grade III - Body weight <1500 g and major cardiac disease (survival 22%)

Waterston and colleagues
- Group A - BW>2500 gm and well: Can undergo surgery
- Group B1 - BW 1800-2500 gm and well: Staged surgery
- Group B2 - BW>2500gm, moderate pneumonia and congenital anomalies
- Group C1 - BW <1800gm
- Group C2 - Higher BW, severe pneumonia and anomalies
- Delayed surgery

TOF types: Cross and Vogt classification
Type I - Oesophageal atresia with no fistula
Type II - No atresia, communication between trachea oesophagus (H-type fistula)
Type III- Oesophageal atresia, upper segment communicating with trachea
Type III B - Oesophageal atresia with blind upper pouch and lower segment communicating with the trachea (commonest)
Type III C - Oesophageal atresia with both upper and lower segments communicating with the trachea
Pathophysiology
Physiology of swallowing is disturbed and secretions/saliva accumulates in the blind upper pouch. Fistulous communication entrains air into the stomach via the trachea causing gastric distension. Overflow of saliva feed from the blind upper pouch and regurgitation of gastric contents via the fistula leads to clinical pneumonitis. Two main pathological entities in TOF are dehydration and aspiration pneumonitis.

Clinical presentation and diagnosis:
- 3Cs – choking, coughing, cyanosis. Cyanosis of oesophageal atresia are secondary to oesophageal discontinuity and respiratory complications.
- Every feed is regurgitated
- Abdomen usually distended but in the absence of fistula, respiratory symptoms are less and abdomen is Scaphoid.

Diagnosis is established when there is inability to pass suction catheter beyond 10cm

Radiography shows a coiled catheter in the blind proximal pouch and air accumulation in the stomach. Instillation of radio-opaque dye into the proximal blind loop is avoided for fear of pulmonary aspiration.

Evaluation
Preoperative assessment aids in diagnosis and quantifies the amount of pulmonary aspiration, prematurity of infant and associated cardiac (30-35%), craniofacial (4%), gastrointestinal, renal (10%) anomalies. It includes; CXR, arterial blood gas analysis, echocardiography, cardiac catheterization, ultrasound for KUB and radiography of limbs (and routine laboratory investigations).

Monitoring
- Precordial stethoscope is secured in left axilla;
- Heart rate, ECG, NIBP (in right upper arm), pulse oximetry end tidal CO₂ temperature.
- Arterial line for invasive BP and multigas analysis is optional.

Preoperative preparation
The priorities are to save life, achieve alimentary continuity and preserve oesophagus. 

Prevention of aspiration:
Avoid feeding, nurse the baby in propped up position, keep proximal pouch empty by aspirating the pooled saliva every 15 minutes. Catheter should be left in upper blind pouch.
- Before shifting, the operating room temperature should be above 25°C.
Optimize pulmonary status: chest physiotherapy, tracheal suction, supplemental oxygen and antibiotics.
- Secure appropriate intravenous and arterial access.
- Maintain volume and metabolic status.
- Arrange blood.

**Surgery**
Immediate operation is seldom essential. 24-48 hours stabilization allows full assessment, better transition from Fetal to neonatal state and treatment of pulmonary insufficiency. Surgery if often delayed till pneumonitis improves. Gastrostomy may be performed under local anesthesia, for nutrition. Continuity of the oesophagus is restored through the right extra-pleural thoracotomy in the 4th intercostal space. The proximal blind pouch is identified, dissected and mobilized for primary anastomosis.

Gastrostomy tube is placed under water seal. Occlusion of the fistula is confirmed by cessation of bubbling through the underwater tube.

**Anesthetic considerations:**
Aspiration pneumonia, securing airway, oxygenation, gastric over distension, and problems associated with coexisting anomalies. Management may be staged, if TOF coexists with prematurity, pneumonitis or other congenital anomalies.

**Problems**
- Inadequate oxygenation and ventilation; leakage of gases through the fistula, ETT misplacement or of endobronchial intubation.
- ETT blockade; periodic ETT suctioning; in case of blocked ETT, replace with a new ETT over a tube exchanger.
- Ventilation/perfusion (V/Q) mismatch in anesthetized lateral decubitus position may be compounded by atelectasis secondary to retraction of the non-dependent lung, bronchial traction and ETT kinking. A high FiO₂ is desirable to keep the SpO₂ between 95-98%. The right lung is intermittently inflated to prevent hypoxemia.
- Vagal response to tracheal manipulation is avoided by prior atropine administration.
- Hypothermia produces peripheral vasoconstriction, non shivering thermogenesis and increased O₂ consumption. It also affects pharmacokinetic and pharmacodynamic profile of anesthetics leading to overdosage, postoperative hypoventilation, apnoea, coagulopathy and metabolic acidosis. Therefore, body and ambient temperature is continuously monitored. Infant must be protected by heating devices and use of warm infusion fluids.
Proper ETT positioning is achieved by placing the tip beyond the fistulous opening and just short of carina to avoid gastric distension during positive pressure ventilation (PPV), and endobronchial intubation [confirmed by flexible fiberoptic bronchoscope (FOB)]. The tube is inserted with bevel facing posteriorly to avoid entering the fistula and then secured with the bevel facing anteriorly.

Prevention of gastric distension to prevent respiratory embarrassment is achieved by:

- Preoperative gastrostomy or an emergency procedure if massive gastric distension produces respiratory embarrassment and/or difficult PPV.
- Retrograde placement of balloon-tipped catheter in the fistula, through the gastrostomy under guidance with FOB or antegrade occlusion of TOF with a balloon tipped Fogarty catheter advanced through the trachea.
- Application of snug abdominal binder.

Sterile nasogastric tube placed in the blind pouch is used as a bypass conduit after establishing the anastomosis. Secretions are aspirated periodically. Moist pharyngeal pack will prevent aspiration of accumulated secretions and stabilize ETT position. Surgeon should wait whenever there is difficulty in ventilation or decreased oxygenation. Manual palpation and manipulation by surgeon can help ascertain proper positioning of ETT.

**Anesthetic technique**

Precordial stethoscope is fixed in left axilla, infant is placed in left lateral position with right upper limb positioned above the head (pulse oximetry is important to diagnose dislodgment of ETT). Awake incubation is preferred. No anesthetic agent is contraindicated. Infant is maintained on spontaneous or assisted ventilation. (Paw <10-15cmH₂O) till the fistula is ligated. Inhalational induction with sevoflurane or halothane is preferred alternative. 90-95% SpO₂ saturation must be maintained. Adequate oxygenation should never be compromised to avoid retrolental hyperplasia.

Use of N₂O in anesthetic mixture is debatable owing to possibility gastric distension. Narcotics are given for analgesia. Anesthesia is maintained with O₂ in volatile anesthetic agents (sevoflurane or halothane) along with small titrated doses of non depolarizing muscle relaxants.

Atraumatic suction of the dependent lung and tracheo-bronchial toilet is essential at the end of surgery, to avoid atelectasis or accumulation of secretions/blood.

**Postoperative Care**

The effects of newer anesthetic drugs, muscle relaxants and opioids may be prolonged because of their altered pharmacokinetics and pharmacodynamics. Compression atelectasis and aspiration pneumonitis may need short period of postoperative
ventilation/PEEP with ETT in situ. Assessment and planning of tracheal extubation should be meticulous.

**Fluid management**
- Maintenance of fluid should be with 5% dextrose in 0.2 - 0.25% saline solution. Insensible loss should be replaced with a balanced salt solution at the rate 6-8 mlkg⁻¹hr⁻¹.
- Blood loss should be carefully monitored and replaced.

**Pain management**
Neonates require adequate pain control by neuraxial or parenteral route.
- Morphine: 0.1 mgkg⁻¹hr⁻¹ in full term and, 0.05 mgkg⁻¹hr⁻¹ in premature babies.
- Epidural Bupivacaine 0.1%, Fentanyl 0.5 ml⁻¹ at the rate 0.1-0.2 mlkg⁻¹hr⁻¹.
- Acetaminophen: Suppositories 35-40 mgkg⁻¹ followed by 20 mgkg⁻¹.
- Infiltration of surgical incision with 0.25% bupivacaine in a dose of 0.5 mlkg⁻¹.

**Postoperative complications**
**Immediate:** Anastomatic leak (16%), symptomatic stricture (35%), gastro-oesophageal reflux (58%), tracheomalacia (15%).
**Late:** due to food trapping in oesophagus, recurrent bronchitis, lung infection and recurrence.

**FOREIGN BODY REMOVAL FROM AIRWAYS**

**Introduction**
Foreign body (FB) aspiration into the trachea and larynx is most common in toddlers (1-3 yrs) and may cause life threatening airway obstruction. 95% FBs get lodged in the right main bronchus.

**Pathophysiology:**
Sequelaes and effects of FB aspiration depend on the site degree and duration of airway obstruction.

**Acute phase:**
Starts soon after FB enters, producing spasmodic cough and/choking. The FB may be expelled or may get lodged in some part of tracheobronchial tree.

**Latent phase: (few hrs to months):**
Careful examination, expiratory wheeze or obstructive symptoms may localize FB.
Chronic phase:
Is demonstrated by productive cough or superadded infection.

Four types of obstruction and their effects
- Check valve – Air can be inhaled but not exhaled (emphysema)
- Ball valve - Air can be exhaled but not inhaled (bronchopulmonary segment collapse).
- By pass valve - FB partially obstructs both inspiration and expiration
- Stop valve - Total obstruction, airway collapse and consolidation

Pharmacology
- Coughing, gagging, laryngo-bronchospasm, hypertension, arrhythmias and secretions must be controlled.
- Anticholinergics reduce secretions and attenuate vagal mediated bradycardia and reflex bronchoconstriction.
- Local anesthetics (lidocaine IV/spray) diminish airway reflexes secondary to endoscopic manipulations.
- Opioid analgesics (fentanyl 1μgkg⁻¹) suppress airway reflexes and administered only after the airway is secured.
- Sevoflurane with 100% oxygen provides smooth induction and can be replaced by isoflurane once deeper plane of anesthesia is achieved.
- Propofol with good reflex suppression has rapid action, quick recovery.

Symptoms and signs
- Large FB with laryngeal obstruction present as bidirectional stridor and/or aphonia.
- FB in trachea present with brassy cough.
- FB in bronchus present as coughing, wheezing, dyspnoea and ipsilateral decreased air entry. Distal hyperinflation from air trapping and/or, inflammatory oedema due to local reaction may be evident.
- CXR provides direct evidence if FB is radio-opaque. Radiolucent FBs show indirect evidence by demonstrating hyper-inflation of the affected lung after 24-48 hrs, with distal atelectasis. Hyper inflation gets prominent during exhalation.

Problems
- N₂O should be withheld to limit further pulmonary inflation and potential rupture.
- Sharing the airway with endoscopist while maintaining alveolar ventilation and providing an unobstructed surgical access.
• Rigid ventilating bronchoscope equipped with an optical telescope and fibreoptic light source is essential.

Goals
• Adequate oxygenation and ventilation.
• Controlled cardio respiratory reflexes during bronchoscopy.
• Rapid return of upper airway reflexes
• Prevention of pulmonary aspiration
• Meticulous monitoring

Preoperative considerations
• Severity of airway obstruction, gas exchange and level of consciousness.
• Mature and location of FB, degree and duration of obstruction.
• Adequate pre-induction fasting: Delaying intervention must be balanced against potential functional impairment, adequate oxygenation.
• Latest CXR (inspiratory, expiratory) for Atelectasis, air trapping, mediastinal shift or pneumonitis.
• Atropine 6-10 μgkg⁻¹ IV is administered to decrease sections and obtund autonomy reflexes during airway instrumentation.
• IV metoclopramide 0.15 mgkg⁻¹ hastens gastric emptying.
• Secure good IV access and ensure adequate hydration.
• Heimlich maneuver may be life saving emergency procedure for FB trachea.
• Check adequate bronchoscopic, resuscitative equipment and oxygen source with anesthesia machine and emergency drugs.

Monitoring
• Precordial stethoscope -inspection of chest movements. Spo₂, ETCO₂
• Neuromuscular transmission with TOF nerve stimulator.
• One overhead spotlight towards anesthetic machine, second over the child’s feet to assess saturation and perfusion.

Anesthetic management

Acute emergency
• GA is always required because of fighting irritable, child removal of distally lodged invisible FB and if prolonged bronchoscopic procedure is contemplated. Based on feeding history, anesthesia can be induced with inhalational or intravenous technique.
Spontaneous technique is safer than apnoeic technique with complete neuromuscular blockade for fear of distal dislodgement. Sevoflurane is preferred over halothane because of smooth induction, its antitussive and cardio-respiratory stability properties.

- Anesthesia is induced with sevoflurane 3-5% in 100% oxygen and reinforced with topical lidocaine 1-4 mg kg⁻¹ prevent laryngospasm.
- Anesthesia is then maintained with 100% oxygen in halothane or isoflurane as these are more soluble and eliminated slowly, giving more time for airway manipulation, avoiding lighter plane and associated hemodynamic reflexes. Deeper plane of anesthesia with spontaneous respiration is required during attempts to extract FB.
- During instrumentation or sanction of FB if the child coughs, small increments of propofol or fentanyl are administered.
- Propofol based IV anesthesia supplemented with topical spray of lidocaine can alternatively maintain a steady level of anesthesia, independent of ventilation or O.T. pollution.

**Stable child**

After premedication with atropine, anesthesia is induced with thiopentone (4-6 mg kg⁻¹), fentanyl (0.5-1 µg kg⁻¹), followed by atracurium (0.3-0.5 mg kg⁻¹) vecuronium (0.07-0.1 mg kg⁻¹) / mivacurium (0.2-0.3 mg kg⁻¹) depending on the estimated duration of endoscopy.

Muscle relaxant and PPV enables a lighter plane of anesthesia and ensures quieter field for the surgeon. Anesthetic gases are attached to the side port of the ventilating bronchoscope and flow adjusted to control respiration, keeping in mind the peri-bronchoscope gas leakage.

Continual monitoring of chest excursions, breath sounds and oxygen saturation is essential because of widespread-atelectasis, low FRC and low O₂ reserve, increased shunting with a possibility of sudden drop in SpO₂ and rise in PaCO₂ during apnoeic spells.

During active ventilation, the telescope/forceps is withdrawn to mid tracheal level, proximal end of endoscope is blocked with glass obturator or thumb for optimal ventilation. Apnoea time should not exceed one minute.

Irrespective of the anesthetic technique (spontaneous/controlled), once the FB is grasped, bronchoscope holding forceps and FB are withdrawn as a single unit. Depth of anesthesia may need to be increased or child temporarily paralysed with succinylcholine 0.25-0.5 mg kg⁻¹ to provide quiet, relaxed upper airway.
**Child with respiratory distress and emphysema**

100% O\(_2\) is supplemented, anesthesia induced in sitting position with sevoflurane and O\(_2\) with facemask. IV access established and atropine given. Anesthesia deepened while ventilation assisted with mask and bag, bronchoscopy, carried out. Muscle relaxants are avoided till the airway is secured.

When the large FB removed from the bronchus gets dislodged in the trachea or larynx, blocking the entire airway, it must be removed immediately or pushed back into the bronchus and the child ventilated.

**Child with suspected full stomach / severe respiratory distress**

After atropine and metoclopramide administration child is preoxygenated in head up position. Rapid sequence induction (RSI) with thiopentone and succinylcholine carried out and airway Secured with ETT. When glottis is withdrawn bronchoscopy done with adjusted gas flow rates.

**3 Ventilation technique**

The ventilation can be manual, IPPV, HFJV or HFPPV. HFJV is preferred through 14G needle attached to bronchoscope with respiratory rate of 120-140 min\(^{-1}\), flow rate of 5-15 L/min\(^{-1}\) with 30-40% inspiratory time. Uninterrupted oxygenation and free, quiet surgical field is provided to visualize the FB.

**Intraoperative complications**

Laryngospasm, bronchospasm, pneumothorax and cardiac arrhythmias may occur. Atropine to minimize secretions; muscle relaxation for adequate ventilation and oxygenation; pulse oximetry to monitor O\(_2\) saturation are preferred. Arrhythmias associated with hypercarbia are treated with hyperventilation and IV lignocaine (1 mg kg\(^{-1}\)). Chest tube is inserted if pneumothorax is confirmed.

**Postoperative care**

Bronchoscopy is repeated to evaluate residual FB fragments and the impact site for trauma, bleeding or granulations. Child may be intubated for tracheobronchial toilet and ventilation until adequate spontaneous breathing is established.

Minimize postoperative stridor and distress by Dexamethasone (0.1-0.25 mg kg\(^{-1}\)) for laryngeal, oedema, humidified oxygen, nebulized racemic epinephrine (2.25% solution given 1:6 to 1:10 dilution) under ECG monitoring, \(\beta\)-agonist bronchodilators such as albuterol for postoperative wheezing.

24-48 hrs intubation may be necessary till oedema subsides.
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Chapter 8 - LAPAROSCOPIC SURGERIES IN CHILDREN

Introduction
Kelling introduced laparoscopy in 1923, but it gained momentum in pediatric patients in last decade. In 1998 series of workshop relating to Pediatric laparoscopy invoked interest of pediatric surgeons towards minimal access surgery (MAS). This interest got sustenance owing to advances in optical and video technology and better understanding of pharmacokinetics and dynamics of newer drugs in Pediatric population. Owing to the advantages of MAS in children, like less pain, small incision, improved recovery, decreased incidence of postoperative pulmonary adversity along with shorter hospital stay and reduced costs, major surgeries (splenectomy, nephrectomy, colectomy, appendectomy, fundoplication, pyeloplasty, Pheochromocytoma excision) are being increasingly performed laparoscopically now.

Physiological changes
Anesthesia for laparoscopy is challenging due to; pneumoperitoneum (PNP) induced increase in intra-abdominal pressure (IAP) and various patient positions to facilitate operation.

Pneumoperitoneum (PNP)
Carboperitoneum permits improved visualization of the operative field. The volume of insufflating gas required in children is typically 0.9 L/10 kg as against 2.5-5.0 L in adults. Low insufflating pressures help control hypercarbia, allow better ventilation and improved oxygenation, limit increased peripheral venous pressure following insufflation and reduce the risk of CO₂ embolism.
Complications of Veress needle puncture of hollow viscera and blood vessels with consequent visceral complications must be kept in mind.
Ideal gas for PNP should not support combustion, be rapidly excreted, have high blood solubility and allow minimal absorption, physiologic perturbation and possibility of embolism.
CO₂ is safe for use during electrocautery, and gets eliminated through the lungs quickly. But smaller distance between capillaries and peritoneum and greater absorptive area of peritoneum in relation to body weight leads to significant vascular absorption in children.
Hypercapnia leads to increased sympathetic activity sensitizes the myocardium to volatile anesthetic agents induced catecholamines (arrhythmia). In the postoperative period ventilation is impaired by residual anesthetic drugs and diaphragmatic
dysfunction. Slow CO\textsubscript{2} insufflation might need increased minute ventilation (upto 60%) to restore EtCO\textsubscript{2} to base line specially during prolonged surgery.

**Other means for pneumoperitoneum**

Helium (costly, CVS complications) and N\textsubscript{2}O (supports combustion) induced PNP did not gain popularity. Alternatively, gasless technique lifts the abdominal wall without implications on IAP.

**Intra-abdominal Pressure (IAP)**

- Pneumoperitoneum raises the IAP with significant effects on all systems.
- Cardiovascular effects during laparoscopy are due to IAP and patient positioning.
- IAP < 15 mmHg - Venous return (VR) is augmented as blood is squeezed out of the splanchnic bed producing increased cardiac output (CO). It benefits infants with hypovolemia who cannot tolerate volume loss from the sequestration of blood.
- IAP > 15 mmHg - Decreases VR as IVC is compressed, decreasing CO and arterial BP. The compensatory flow via the capillaries is ineffective as the collateral vessels are simultaneously compressed.
- There is a progressive decrease in cardiac index (CI) with an increase in IAP (fall in CI is 55% and 38% of baseline at 20 and 30 mmHg IAP respectively). There is simultaneous (35%) fall in renal, hepatic and mesenteric blood flow at an IAP of 25 mmHg.
- Low IAP in cardiac compromised patients is recommended as IAP upto 12 mmHg had minimal effect on CI (13% reduction) while at 6 mmHg IAP neither cardiac parameter nor any surgical condition is effected.

There is no hemodynamic compromise in Trendelenburg position during laparoscopic hernia repair with pneumoperitoneum less than 10 mmHg IAP.

**Positioning**

**Cardiovascular effects**

- Head up position for upper abdominal surgery reduces VR and CO depending upon the degree of tilt. Fundoplication (25°- 30°) shows greater changes than during cholecystectomy (15°-20°). Dissection at the gastro-oesophageal junction around the oesophageal hiatus, produces greater and sustained rise in mediastinal and pleural pressures.
- Head down position during pelvic surgery augments VR with normal or supra normal BP.
Cardiovascular changes are same irrespective of gases used implying that the changes are due to IAP and position rather than the effect of the gases. So IAP should be kept low (< 13 mmHg) to minimize adverse cardiovascular effects. Children have high vagal tone. Sudden gas insufflation or peritoneal stimulation by trocar/laparoscope can provoke bradycardia. Patients with CVS dysfunction, anemia, hypovolaemia, show drastic changes in preload and after load, so care should be taken during volume loading, positioning and gas insufflation.

**Respiratory effects**

Raised IAP shifts the diaphragm cephalad. Splinting results in closure of smaller airways, increased airway pressure, reduced thoracic /compliance and FRC. V/Q mismatch increases because of preferential ventilation of non dependent lungs. Effects are accentuated during trendelenburg position and positive pressure ventilation (PPV). In children FRC is low (10% of TLC) and quickly falls below the closing capacity producing small airway closure, atelectasis, intrapulmonary shunting and hypoxemia. These effects are aggravated in steep head down tilt because of the weight of the abdomen viscera on the diaphragm. Use of PEEP reverses this action and improves O₂ saturation.

At low IAP (10 - 15 mmHg) minimal changes are seen in increased PAP (<20%), EtCO₂ (20-27%) and lung compliance. All changes revert back to normal within 10 minutes of insufflation.

Post operative hypoxemia is due to diaphragmatic dysfunction rather than creation of pneumoperitoneum because changes seen during fundoplication exceed that during inguinal Herniorraphy. High IAP may result in leakage of gases to tissue spaces leading to pneumothorax, Pneumomediastinum, especially during Nissans fundoplication. A post operative chest radiograph is, therefore, advisable.

Diagnostic laparoscopy in young children with facemask and spontaneous ventilation shows no significant CVS alteration or V/Q mismatch. Elevation of V₇, EtCO₂ and RR return to normal within 10 minutes of insufflation. It is advantageous in patients with irritable tracheobronchial tree but should be restricted to healthy children for brief procedures in supine position.

**Neurologic effects**

Elevated IAP raises ICP and reduces the cerebral perfusion pressure (CPP). IAP of 25 mmHg increases the ICP from 7.6 to 21.4 mmHg (mean) and reduces the CPP from 82 to 62 mmHg. Laparoscopic procedures are avoided in patients with reduced intracranial compliance. Hypercapnia, increased SVR and head down lilt further aggravate the condition.
Endocrinal effects
Like any conventional surgery, laparoscopic procedures increase blood levels of insulin, cortisol, prolactin, epinephrine. Blood levels of lactate, glucose and interleukin-6 too are raised.

Anesthetic management Preoperative evaluation
Thorough preoperative history and detailed physical examination should be tailored to the severity of systemic disease and urgency of the operation. Minimum baseline investigations are desirable according to the co-existing medical disease.

Premedication
Children younger than 9 months do not suffer parental separation, anxiety and need no premedication or anticholinergics. In grown up children atropine is given, 20μgkg⁻¹ IM or 30-40μgkg⁻¹ oral 30-45 minutes preoperatively to prevent vasovagal reflexes cardiovascular and airway events, perioperatively. Pre medication (via nasal, rectal, IM transmucosal) in healthy outpatients can be; oral midazolam 0.5-0.75mgkg⁻¹ dissolved in acetaminophen or Ibuprofen elixir 10 mgkg⁻¹ 15-30 min preoperatively. Antacids, H₂-antagonists, gastrokinetics may be necessary. Opioids, antisialogogue or ketamine are included depending on the type, duration, severity of procedure.

Monitoring
Continuous ECG, automated NIBP, pulse oximetry, capnography, peripheral nerve stimulator and core temperature should be monitored.

- Exhaled tidal volume signifies leakage around the tracheal tube.
- Precordial / oesophageal stethoscope to exclude endobronchial intubation during pneumoperitoneum.
- Precordial Doppler, TEE are useful for early diagnosis of venous air embolism.

Induction of Anesthesia
Local and regional techniques are unsuitable for children. Peripheral IV access is secured (after application of EMLA cream), preferably above the diaphragm to ensure the drugs and fluid reach central circulation. 20 mlkg⁻¹ fluids is administered to offset hemodynamic effects of pneumoperitoneum during induction. Orogastric tube is inserted after induction to deflate stomach. Rapid sequence induction is recommended till the airway is secured to reduce the risk of pulmonary aspiration. Alternatively, inhalational induction using sevoflurane or halothane in N₂O and O₂ is helpful for smooth induction.
Perioperative care

- Balanced anesthetic technique involves controlled ventilation, inhalational agents (Halothane, Isoflurane, Sevoflurane, Desflurane), short acting IV opioids, neuromuscular blockers (vecuronium, Rocuronium, cisatracurium, mivacurium, or Rocuronium). TIVA may be opted if myocardial depression is the concern. Use of N₂O is debatable because of possibility of venous gas embolism, distension of bowel, increased incidence of nausea/vomiting.
- Halothane should be discontinued once the trachea is intubated as it sensitizes myocardium to hypercapnia. Reduced hepatic blood flow following pneumoperitoneum can make the children prone to halothane hepatotoxicity.
- Controlled ventilation facilitates removal of exogenous CO₂ and minimizes the reduction in FRC during increased IAP, head down tilt and use of volatile agents.
- Minute ventilation may be increased by 20% by increasing RR. In children ventilated with uncuffed ETT, peri-ETT gas leakage reduces VT and should be compensated by increasing minute ventilation by about 30%.
- Insufflation should be complete to minimize hyperventilation (due to delayed excretion of CO₂) referred shoulder pain (diaphragmatic irritation).
- Euthermia should be maintained with warmers, heated humidified gases to offset heat loss during surgery (high body surface: mass ratio, continuous insufflation of cold, dry gases).

Postoperative care

Monitor signs of hypoventilation. Maintain euthermia. Chest X-ray should be done following laparoscopic fundoplication, O₂ therapy is continued. The incidence of PONV can be decreased by H₂, blocked 5HT₃ antagonist; ondansetron (100 µg/kg, maximum 4 mg), dexamethasone 150 µg/kg, droperidol 25 µg/kg (maximum 0.625 mg).

Pain management

Pain though mild may be due to rapid distension of the peritoneum, excitation of phrenic nerve, instrumentation or undesirable stretching of nerves during positioning. Less shoulder pain is seen in children. Polymodal method of spraying LA over the operation site, infiltration of port sites, caudal block and administration of NSAID’s/opioids is preferred.

Use of laryngeal mask airway (LMA)

LMA can be utilized for pelvic laparoscopy while patient breathing spontaneously. No cardiac respiratory events are encountered in procedure of <30 minutes duration with an IAP < 12 mmHg. It should be avoided in prolonged surgery with extreme head down tilt and compromised cardiovascular status. Repeated auscultation over the stomach to
detect gas insufflation is recommended. Incidence of aspiration/regurgitation is increased in trendelenburg position, increased IAP pressure, pressure on anterior abdominal wall, peritoneal stimulation and LMA cuff inflation over hypopharynx (lowers LES tone).
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Chapter 9 - PYLORIC STENOSIS

- 1 in 500 live births – male preponderance – infants 2 to 5 wks of age.
- Common in term and preterm neonates
- Hypertrophy of pyloric smooth muscle with edema of the pyloric mucosa and submucosa → Over days to weeks → leads to progressive obstruction of the pyloric valve – causing persistent vomiting.

**Signs and symptoms:**
- Vomiting → loss of fluids and electrolytes (Na+, K+, Cl-, H+ ions) decreased
- Dehydration; hyponatremic, hypokalemic, hypochloremic metabolic alkalosis with a compensatory respiratory acidosis.
- Medical emergency – not surgical emergency
- Surgical repair done after adequate fluid and electrolyte hemostasis → normal skin turgor Na+ > 130 mEq/L, K+ > 3 mEq/L, Cl- > 85 mEq/L and increasing, urine output 1-2ml/kg/hr.
- Resuscitation with full strength balanced salt solution and after infant begins to urinate – add KCl. (5% D with 0.45% NS – ISOLYTE-P).

**Anesthetic management:**
- Aspiration of gastric contents – definitive risk, further ↑ after radiographic examination of upper GIT with barium.
- Large orogastric tube is passed and stomach contents aspirated as much as possible.
- Awake tracheal intubation / after induction of anesthesia.
- IV line already obtained – rapid seq. tracheal intubation (explain with cricoid pressure).
- IV line not in place – stomach emptying → inhalational induction with N₂O + Halothane / Sevoflurane. N₂O discontinued after loss of lid reflex → if vein becomes evident IV line placed – rapid seq. induction with cricoid pressure. If IV line still not placed – 2 options exist
  1. Deepen inhalational anesthesia with halothane / Sevoflurane, insert laryngoscope, topically anesthetize the vocal cords with 4mg/kg lidocaine, and intubate the trachea.
  2. Administer 4mg/kg scoline IM before intubation
  3. Secure IV line before beginning surgery – if needed cut down
- Maintenance: volatile anesthetic + opioids ± N₂O + skeletal muscle relaxants + mechanical ventilation.

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Surgeons need muscle relaxation twice
a. When they deliver the pylorus at the beginning of surgery
b. When they replace the pylorus into the abdomen at the end of surgery, shortly before closing the peritoneum → short acting drugs like succinylcholine / mivacurium are considerations.

Caudal anesthetic (1.25ml/kg 0.25% bupivacaine with epinephrine) after induction of GA with tracheal intubation.
- Provides intraoperative relaxation
- Reduces anesthetic requirement
- Provides postoperative analgesia

Postoperative management:
- Infants should be fully awake and displaying acceptable patterns of ventilation before tracheal extubation considered.
- Postoperative depression of ventilation – causes CSF alkalosis and intraoperative hyperventilation of lungs.
- Hypoglycemia – may occur 2 to 3 hrs after surgical correction.
- Hypothermia
Chapter 10 - PEDIATRIC AMBULATORARY SURGERY
PERIOPERATIVE CONCERNS

Introduction
Pediatric surgery in an ambulatory setting shortens hospital stay, reduces exposure to nosocomial infections, and allows for the active parental participation (table 1). The popularity of this subspecialty has created new challenges and rewarding opportunities for pediatric anesthesiologists. Thorough preoperative screening, proper patient selection, optimum perioperative care and follow up are considered the most important issues in the day care system.

<table>
<thead>
<tr>
<th>Table 1: Advantage of pediatric outpatient anesthesia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Minimizes parental separation</td>
</tr>
<tr>
<td>Uninterrupted feeding schedule/sleep patterns</td>
</tr>
<tr>
<td>Less risk of nosocomial infections</td>
</tr>
<tr>
<td>Convenience/Improved patient satisfaction</td>
</tr>
<tr>
<td>Availability of beds for complex needy patients</td>
</tr>
<tr>
<td>Reduced cost of hospitalization</td>
</tr>
<tr>
<td>Historical perspective</td>
</tr>
</tbody>
</table>

Preoperative fasting

Table 3: Preoperative fasting guidelines (ASA task force 1999)

<table>
<thead>
<tr>
<th>Patient</th>
<th>Clear liquids</th>
<th>Milk (breast / formula)</th>
<th>Solids</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neonates</td>
<td>2 hours</td>
<td>&gt; 4 hours</td>
<td>&gt; 6 hours</td>
</tr>
<tr>
<td>Infants</td>
<td>2 hours</td>
<td>4-6 hours</td>
<td>&gt; 6 hours</td>
</tr>
<tr>
<td>Children</td>
<td>2 hours</td>
<td>4-6 hours</td>
<td>&gt; 6 hours</td>
</tr>
</tbody>
</table>

Table 5: Laryngeal mask airways

<table>
<thead>
<tr>
<th>Patient weight (Kg)</th>
<th>LMA size</th>
<th>Maximum inflation volume (ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neonate upto 6.5</td>
<td>1</td>
<td>2-4</td>
</tr>
<tr>
<td>6.5-10</td>
<td>1.5</td>
<td>5</td>
</tr>
<tr>
<td>10-30</td>
<td>2</td>
<td>10</td>
</tr>
<tr>
<td>20-30</td>
<td>2.5</td>
<td>15</td>
</tr>
<tr>
<td>30-50</td>
<td>3</td>
<td>20</td>
</tr>
</tbody>
</table>
### Table 5: Modified aldrete post anesthesia recovery score (PARS)

<table>
<thead>
<tr>
<th>Activity</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Able to move 4 extremities voluntarily or on command</td>
<td>2</td>
</tr>
<tr>
<td>Able to move 2 extremities voluntarily or on command</td>
<td>1</td>
</tr>
<tr>
<td>Unable to move extremities voluntarily or on command</td>
<td>0</td>
</tr>
<tr>
<td>Respiration</td>
<td></td>
</tr>
<tr>
<td>Able to breath deeply and cough freely</td>
<td>2</td>
</tr>
<tr>
<td>Dyspnea or limited breathing</td>
<td>1</td>
</tr>
<tr>
<td>Apneic</td>
<td>0</td>
</tr>
<tr>
<td><strong>Circulation</strong></td>
<td></td>
</tr>
<tr>
<td>BP + 60% of pre anesthetic level</td>
<td>2</td>
</tr>
<tr>
<td>BP + 40% of pre anesthetic level</td>
<td>1</td>
</tr>
<tr>
<td>BP + 20% of pre anesthetic level</td>
<td>0</td>
</tr>
<tr>
<td><strong>Consciousness</strong></td>
<td></td>
</tr>
<tr>
<td>Fully awake</td>
<td>2</td>
</tr>
<tr>
<td>Arousable on calling</td>
<td>1</td>
</tr>
<tr>
<td>Not responding</td>
<td>0</td>
</tr>
<tr>
<td>Oxygen saturation</td>
<td></td>
</tr>
<tr>
<td>Able to maintain O₂ saturation &gt; 92% on room air</td>
<td>2</td>
</tr>
<tr>
<td>Needs O₂ inhalation to maintain O₂ saturation &gt; 90%</td>
<td>1</td>
</tr>
<tr>
<td>O₂ saturation &lt; 90% even with O₂ supplementation</td>
<td>0</td>
</tr>
</tbody>
</table>

A score of > 9 required for discharge from acute post anesthesia care unit

### Table 6: Modified post anesthesia discharge scoring system (PADSS) – Marshall and Chung

<table>
<thead>
<tr>
<th>Vital signs (stable / consistent with age and pre operative baseline)</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>BP and Pulse within 20% of pre operative baseline</td>
<td>2</td>
</tr>
<tr>
<td>BP and Pulse 20%-40% of pre operative baseline</td>
<td>1</td>
</tr>
<tr>
<td>BP and Pulse &gt;40% of pre operative baseline</td>
<td>0</td>
</tr>
<tr>
<td><strong>Activity Level (Ambulation at pre operative level)</strong></td>
<td></td>
</tr>
<tr>
<td>Steady gait, no dizziness</td>
<td>2</td>
</tr>
<tr>
<td>Requires assistance</td>
<td>1</td>
</tr>
<tr>
<td>Unable to ambulate</td>
<td>0</td>
</tr>
<tr>
<td><strong>Nausea and vomiting</strong></td>
<td></td>
</tr>
<tr>
<td>Minimal: Treated with PO medication</td>
<td>2</td>
</tr>
<tr>
<td>Moderate: Treated with IM medication</td>
<td>1</td>
</tr>
<tr>
<td>Continues after repeated treatment</td>
<td>0</td>
</tr>
<tr>
<td>Pain</td>
<td></td>
</tr>
<tr>
<td>Acceptable to patient; controlled with post operative medications</td>
<td></td>
</tr>
<tr>
<td>---------------------------------------------------------------</td>
<td>---</td>
</tr>
<tr>
<td>Yes</td>
<td>2</td>
</tr>
<tr>
<td>No</td>
<td>1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Surgical bleeding</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Minimal: No dressing change required</td>
<td>2</td>
</tr>
<tr>
<td>Moderate: Upto two dressing changes required</td>
<td>1</td>
</tr>
<tr>
<td>Severe: More than three dressing change required</td>
<td>0</td>
</tr>
</tbody>
</table>

**Maximum score = 10  Score > 9 required for discharge**
Chapter 11 - REGIONAL ANAESTHESIA IN PEDIATRIC PATIENTS

The advantages of RA are:

a. It provides complete block of sensory transmission, hence offers complete pain relief.

b. The anesthetic requirement (inhalational agents) comes down drastically. It has opiate sparing effect. Hence the recovery from GA is faster and smoother.

c. It can be extended to the post-operative period especially after major operation to provide pain relief.

Indications

I. Whenever possible all children should receive RA in some form or other, appropriate to the proposed surgery.

II. RA in children is usually administered and practiced after induction of general anesthesia except in certain situations like, premature baby or ex-premature baby up to a conceptual age of 60 weeks when there is fear of post operative apnoea. It is a well-recognized fact that the incidence of post operative apnoea is least under spinal block as compared to spinal with sedation or general anesthesia. Whatever technique is practiced in this group of infants proper monitoring is a must.

III. Children undergoing thoracic and upper abdominal surgeries those need aggressive pain management in the post-operative period.

Contra indications

a. Lack of parental consent

b. Infection at the site of administration of the block

c. Any coagulation disorder.

Common regional analgesia techniques

The most commonly practiced RA technique is the caudal (epidural) block. Other common blocks are ilioinguinal ilio hypogastric block (hernia) and block of the dorsal nerve of penis (penile).

Caudal block

The sacral anatomy and caudal block technique described in the textbooks are extrapolation of adult anatomy and techniques and are not children specific. That's the reason why a high rate of inability to feel the sacro-coccygeal ligament and high failure rate of the block is described.
**Anatomy:**
The sacrum in infants and children is flat as compared to that of an adult (fig. 1). The sacral hiatus is situated near the natal cleft in adults whereas in infants it is located much higher. In neonates it could be at the middle of sacrum. The filum terminale terminates at a lower level than S₂, might be S₃ or S₄. The size of the vertebrae is so small that little advancement of needle might reach two or three segments cephalopod.

![Image of anatomical differences in adults and child sacrum.](image1)

The sacral hiatus is bordered by spine above, two laminae and two cornue on the sides. The Sacrococcygeal ligament extends from the posterior aspect of these bony structures to dip down anteriorly. The depth of the epidural space is maximum at the apical region of the hiatus (fig. 2). It gradually reduces to negligible space at the level of the cornue.

**The technique**
The approach to the hiatus should be made from above alone the sacral spines and not from the tip of the coccyx. That makes it easy to identify the commonly seen sacral anomalies in children.

![Image of sacrococcygeal ligament and depth of epidural space.](image2)
An imaginary line is drawn between the two posterior superior iliac spines. They can be identified as projections or dimples in chubby kids. This line passes through the \( S_1-S_2 \) space or \( S_2 \) spine. Feel the \( S_2 \), spine and proceed downwards feeling the spine of \( S_3, S_4 \) till one feels the hiatus. The hiatus might be at the level of any of these spinous processes. At higher level chance of puncturing the dural sac is a possibility. Once the hiatus is identified by the thumb, it is moved across to feel the laminae. Then the skin is pulled up to the spinous process (the point of injection is not touched). This way there is a overlap between the site of puncture of skin and the ligament, preventing any leakage of the drug administered. A 22/23 G hypodermic needle is advanced at an angle of 70°-90° at the apical region of the hiatus (fig.4). The ligament can be identified by the loss of resistance or the 'pop' of overcoming the resistance. The sacro coccygeal ligament is always felt. Once the needle crosses the ligament, it should be stopped and drug deposited. Further advancement or change of direction is not necessary. In case thinner needles are used it should be disconnected from the syringe to look for any back flow. The negative pressure generated by aspiration often collapses the vein over the needle tip. Some people advice routine use of styletted needle for fear of introducing dermal plug. However it has been seen that microscopic plugs or cells is not possible to avoid even in a 26,27G styletted needle. Some people advocate routine use of test dose. However we do not practice it and have not had any complication in our practice of last over twenty years.

**Catheter insertion:**

In patients where post operative pain relief is necessary for longer duration a catheter might be inserted through the caudal space and advanced to the dermatomes involved in incision. An infusion with more lipophilic drug like fentanyl is used. When the catheter is left at the caudal epidural space only hydrophilic opioid like morphine can be used intermittently. It is effective even for thoracic and upper abdominal surgery. Catheter can be inserted through a Touhy needle (fig.5). The length of catheter to be advanced has to be measured prior to insertion. It can also be inserted through an
indwelling cannula. An 18 G catheter easily passes through 18 G cannula. The possibility of catheter not reaching or kinking or knotting is very high. Hence one should try to confirm the tip radiologically using a water-soluble dye. As the sacral hiatus is close to the anus there is possibility of contamination of the catheter and the puncture site. It can be prevented by occlusive dressing with a water resistant flap cover or by bringing out the catheter on the lateral flank by subcutaneous tunneling. The 18G catheter has less chance of kinking than 20G/22G ones. The catheter advancement from caudal route has less chance of kinking than through the lumbar route.

**The common drugs used are**

**Single shot caudal block:**
Bupivacaine 0.25%. Higher concentration causes undesirable motor block and should be avoided. For surgery of various sites different doses are recommended:

- Genital and anal canal - 0.5 mlkg⁻¹
- Upto T₁₂ (hernia) - 0.75 mlkg⁻¹
- Upto T₁₀ - 1.00 mlkg⁻¹

This recommendation is a modification of Armitage formula used in our patient population.

**Intermittent:**
Morphine 30-50 µgkg⁻¹ in 10 ml for infants and children weighing more than 5 kg and 5 ml for less than 5 kg and 5 ml volume though empirical has been seen to be effective and safe.

**Continuous:**
- Bupivacaine 0.125% with fentanyl 1-2 µgml⁻¹ or bupivacaine 0.0625% with fentanyl 2µgml⁻¹.
- The infusion can be started with 1-1.5 mlhr⁻¹. Up to 2 mlkg⁻¹ is good enough for most of the cases.

**Other neuraxial blocks**

**Lumbar Epidural**
The lumbar epidural block is technically similar to that of an adult. The only differences are:

I. Depth of the epidural space is small
II. The ligaments are thinner, hence difficult to feel the loss of resistance.
III. The midline approach is preferred as the laminae are not well developed.
Subarachnoid block

The differences are:
I. The spinal cord is at a lower level might be L₃ or L₄ in infants. Hence a lower space L₅-S₁ or L₄-₅ is always preferred.
II. The laminae are not well developed hence the midline approach is preferred.
III. The CSF volume is higher and the CSF turn over is faster hence the drug "gets diluted and removed faster. The duration of action gets reduced.

The technique:
Technically it is the same as that in adult except that a thinner (25-30G) and smaller needle are used at a lower space. In these babies spinal in sitting position has been described but my personal preferences is lateral position.

Combined Spinal Epidural Anesthesia (CSEA)
Because of the limitations of single shot subarachnoid block and epidural block, viz shorter duration of action and in ability to produce post operative pain relief this combined technique was described by Williams. A variety of extensive surgical procedures including small bowel resection and genitourinary procedures were successfully performed. The post operative analgesia was satisfactorily maintained in all the patients. It can be said that CSEA is a potential option to GA for major abdominal surgery in infants.

Ilio Inguinal and Ilio Hypogastric nerve block (hernia):

Anatomy:
This pair of nerves transverse along the lateral wall of pelvis. They emerge through the internal oblique muscle to lie under external oblique aponeurosis medial to the anterior superior iliac spine (fig.7).
Technique:
A point, one centimeter medial and lower to the anterior superior iliac spine is identified. A needle with short bevel is chosen. As the needle is advanced at this point through skin and subcutaneous tissue the external oblique aponeurosis offers resistance. A ‘pop’ or loss of resistance is felt as the needle penetrates the external oblique aponeurosis. The local anesthetic injected here will bathe the nerves and block them.

This block is used for hernia repair. Often manipulation of cords lead to noxious stimulation. This can be effectively blocked by modification of this classical block. The needle after initial drug deposit is further advance in to the internal oblique muscle. If a loaded syringe is used it "Will be difficult to inject in to the muscle. The needle is advanced gradually keeping gentle pressure on the plunger. Once it crosses the muscle it becomes easy to inject. The drug is deposited here. It lies between internal oblique and fascia transversalis and gradually trickle down to bathe Tile cord at the inguinal canal . In infants it might be difficult to identify the space and may lead to intra-peritoneal injection.

Dorsal Nerve of Penis block (Penile)

Anatomy:
The dorsal nerve of penis emerges from under the symphysis pubis on the dorsal surface of the corpora cavernosa. They lie in a triangular compartment bounded by the symphysis pubis above, the corpora cavernosa below and the membranous layer of fascia in front (fig. 10). When viewed in the anterior posterior view this space is divided by the suspensory ligament of the penis derived from the deep surface fascia. The suspensory ligament then divides into two sheets, which passes around the shaft of the penis. The nerves lie deep in the triangular space formed by the division of the suspensory ligament, and are accompanied by their arteries and vein.
There are potential pear shaped spaces on each side of the suspensory ligament and it is into these spaces that the local anesthetic should be deposited. Any attempt to get close to the nerves might lead to injury to the vessels.

**Techniques:**
The symphysis pubis is palpated. A needle (short beveled) inserted at right angles until it contacts it. The needle is then withdrawn and redirected below the symphysis, through the fascia into the space above the corpora cavernosa. It has been observed that single injection most of the times enter the compartment on one side of the suspensory ligament. It is therefore best to inject on both sides by angling the needle by fifteen degree.
The volume needed for an effective block is 1.5-2 ml on each side in infants and adding 1 ml 10 kg\(^1\) on each side for bigger boys. Often the ventral branch escapes the block and may need additional subcutaneous infiltration of the ventral surface of penis.

**Complications**
The complications of regional analgesia may be drug related or technique related.

**Drug related**
The major toxic affect of LA is on the cardiovascular and central nervous system. The effects depend on total dose, site and route of administration, rate of degradation, metabolism and elimination. It can be prevented by use of recommended doses only. Minimum recommended dose for lignocaine is 1.5 mg kg\(^{-1}\) and with adrenaline 3 mg kg\(^{-1}\). Bupivacaine up to 3 mg kg\(^{-1}\) is recommended. For infants below 6 months the dosage should be reduced by 30%.

In case of intravenous injection of bupivacaine, \(\text{\textbackslash may manifest with cardio toxicity. The risk of cardiac toxicity might increase with concomitant use of inhalational agents. Though in children CNS toxicity also occurs almost at the same time, it’s clinical manifestation might be masked by general anesthesia. The treatment is basically symptomatic. The cardiac toxicity of bupivacaine is difficult to treat.}\)

**Technique related**
The technique related complications have been studied both retrospectively as well as prospectively. The incidence is low and is of minor in nature and transient. The incidence is practically negligible, with peripheral nerve blocks. The incidence of serious complications are seen only with central neuraxial blocks and is 1 in 40000-50000 cases.

**Complications after  Spinal**
- Total spinal block
- High spinal and respiratory paralysis
- Post dural puncture headache (PDPH)
- Meningitis

The incidence of PDPH has been reported to be as high as in adult. It might be difficult to make a diagnosis. A crying child in upright position if becomes quiet on lying down he/she probably is having a headache.

**After epidural**
- Dural tap
- Total spinal
- Catheter knotting, kinking.
Chapter 12 - COMPLICATIONS FOLLOWING GENERAL ANESTHESIA IN PEDIATRIC PATIENTS

Introduction

"In anesthesia, as in other areas of life, everything does not always go as planned. Undesirable outcomes occur regardless of the quality of care provided."

*Posner KL, Cheney FW, Kroll DA*

Incidence of undesirable outcomes is more in Pediatric patients. In a retrospective study by Keenan RL and Boyan CP, higher incidence (three times) of cardiac arrest was reported in children compared to adults. Complications leading to cardiac arrest in this study were mostly due to perioperative laryngospasm, difficult intubation, pulmonary aspiration or halothane overdosage. Infants younger than 1 month old have the greatest risk for perioperative complications because they are more likely to have major surgery and sicker than older children. Prematurity further complicates the situation. Therefore post general anesthesia complications in Pediatric patients may be discussed under following headings.

I. Complications due to prematurity
II. Complications due to congenital anomalies
III. Complications due to genetic disorders
IV. Complications related to anesthetic techniques
V. Complications due to succinylcholine

A. Complications due to prematurity

Infants are considered premature if they are born before 37 weeks of gestation. Prematurity is one of the leading causes of perioperative mortality and morbidity. Incidence of anesthetic morbidity increases directly with the degree of prematurity. They are more prone to perioperative hypothermia, apnoea, respiratory distress, congestive heart failure, retinopathy and intracranial haemorrhage.

a. Impaired thermoregulation

Premature infants are very much prone to hypothermia, due to impaired thermoregulation. Consequences of hypothermia are apnoea, bradycardia, metabolic acidosis, and hypoglycaemia.

Causes of hypothermia:

- Lack of fat insulation
- Excessive heat loss due to increased surface to volume ratio
• Fewer brown fat cells
• Increased heat loss due to thin skin

To prevent hypothermia, premature infants should be kept in the incubator and operation theatre temperature should be raised during operation of such a baby.

b. Apnoea

• Spells of apnoea is very common in premature infants. The incidence of apneic episodes is inversely related to conceptual age of the infants. It is rarely seen after 44 - 48 weeks of conceptual age.
• Apnoea may brief (respiratory pause < 15 seconds and not associated with bradycardia) or it may be prolonged and life threatening. Life threatening apnoea is more than 15 seconds of duration and usually associated with bradycardia (heart rate < 100 beats min-1 for at least 5 seconds).
• Several studies have demonstrated an increased risk for postoperative apnoea in former preterm infants. Administration of different inhalational anesthetics, sedatives, narcotics and muscle relaxants may increase the incidence of apnoea in postoperative period.

This risk can be minimized by:

• Perioperative administration of caffeine or theophylline,
• Use of spinal anesthesia instead of general anesthesia and
• Relaying the surgery, until the child is older than 48-60 weeks post conceptual age.

Use of caffeine 10 mgkg⁻¹, as a premedicant virtually eliminate the postoperative apnoea. However, as a precautionary measure, all premature infants should be admitted for all surgery and should be monitored for 12-24 hour following surgery to prevent apnoea and bradycardia. Infants more than 50 weeks of post conceptual age can be managed as ambulatory patients.

c. Respiratory distress syndrome

Respiratory distress syndrome is caused by deficiency of surfactant resulting in alveolar collapse, right to left shunt, hypoxemia and metabolic acidosis. It is more common in neonates born by caesarean section before 34 weeks of gestation. Administration of artificial surfactant immediately after birth specially in high risk cases significantly reduces the severity of illness.

d. Retinopathy of prematurity

Premature infants are susceptible to retinopathy. It is inversely related to gestational age and birth weight of the infant. Incidence is highest in infants weighing less than 1000gms.
Various attempts were made to find out the role of oxygen therapy in neonatal retinopathy, but failed to demonstrate clear cut relationship. Brief exposure to 100% oxygen does not increase the incidence of ROP. However, every attempts should be made to control oxygenation by monitoring, oxygen saturation and keeping it between 94 and 97 per cent.

e. Periventricular - intraventricular haemorrhage
Newborn immaturity is the single most important risk factor for intracranial haemorrhage. In majority of cases, it occurs in first 72 hours of life and is rare after 10 days. Neonatal hypoxia is another important cause of intracranial haemorrhage. Hypoxia impairs cerebral autoregulation. As a result, any increase in systemic arterial pressure may increase the cerebral blood flow and may cause periventricular or intraventricular haemorrhage. Various anesthetic procedures such as starting of intravenous channel or awake intubation often induce systemic hypertension and increase cerebral blood flow leading to intracranial haemorrhage. Therefore, every precaution should be taken to avoid hypoxemia, hypercarbia, and cerebral hyperperfusion by maintaining blood pressure in the normal range. All stressful procedures should be done under sedation or anesthesia, unless the infant is so critically ill which prevents the anesthesiologist to do so.

Hyperosmolarity is another contributory factor for intracranial hemorrhage in premature baby. Hyperosmolar fluids such as sodium bicarbonate should be avoided as far as possible or they should be diluted and administered slowly to prevent such complications.

B. Complications due to congenital anomalies
After prematurity, congenital anomalies are the second leading cause of mortality and morbidity in the first 30 days of life. Common congenital anomalies associated with perioperative complications are congenital heart defects, congenital diaphragmatic hernia, tracheoesophageal fistula and anterior abdominal wall defects.

a. Congenital heart disease
Cardiac murmurs are very common in children. It may be either functional or pathological. Presence of murmur is not a contraindication for general anesthesia, if the patient is clinically otherwise normal. However, presence of cyanosis, decreased exercise tolerance, poor weight gain, sweating, decreased femoral pulses and precordial heave along with a cardiac murmur usually indicates some organic lesion in the heart. These patients need thorough preoperative evaluation and expertise opinion from Pediatric cardiologist.
Hypoplastic left heart syndrome is a relatively rare congenital defect, but it accounts for 15% of neonatal deaths associated with congenital heart disease. It is often associated with other congenital defects and carries high perioperative morbidity and mortality.

**Intracardiac shunts:**
After birth, clamping of the umbilical cord and initiation of respiration produce tremendous change in circulatory system of new born baby. Reduction in pulmonary vascular resistance is accompanied by constriction of the ductus arteriosus due to increased partial pressure of oxygen in the blood. This increases pulmonary blood flow as well as left atrial pressure resulting into functional closure of foramen ovale. These two neonatal shunts (ductus arteriosus and foramen ovale) may open during anesthesia if there is any alteration in cardiopulmonary mechanics.

Rise in systemic vascular resistance caused by lighter plane of anesthesia may increase left to right intracardiac shunt, and may produce pulmonary over circulation and failure. Similarly hypoxia, hypercarbia, acidosis, hypotension and hypothermia may increase the pulmonary vascular resistance and may reverse the direction of shunt (right to left) leading to hypoxemia or acute cor pulmonale.

**b. Congenital diaphragmatic hernia**
Congenital diaphragmatic hernia is a surgical emergency, often associated with other congenital anomalies such as hydrocephalus, encephalopathy, intestinal atresia, atrial septal defect, ventricular septal defect, tetralogy of fallot and coarctation. It carries high mortality rate, in spite of intensive perioperative care. Postoperative recovery depends on the degree of pulmonary hypertension and pulmonary hypoplasia. Most infants suffer from ventilatory insufficiency in the postoperative period and need ventilatory support. Long term sequelae includes bronchopulmonary dysplasia, pulmonary hypoperfusion and decreased FEV, and ventilatory capacity.

**c. Tracheoesophageal fistula**
This is a surgical emergency of newborn baby. Postoperative complications are mainly due to associated prematurity and congenital heart defect, which is present approximately in 20-25% of cases.

There are two major complications of tracheoesophageal fistula; aspiration pneumonia and dehydration. Sometimes gastric juice reflux, aspiration and pneumonia is so severe that patient may need prolonged postoperative ventilatory support. Presence of congenital heart disease may further complicate the situation. Tracheal compression secondary to tracheomalacia and persistent gastroesophageal reflux due to abnormal swallowing reflex may complicate the postoperative period.
**d. Anterior abdominal wall defects**

Omphalocele and gastroschisis are the two congenital anomalies associated with anterior abdominal wall defects.

Primary closure of defect may increase the intra abdominal pressure significantly and compromise ventilation. Hence ventilatory support may be required for a period of 3-7 days following operation. Additional complications include postoperative hypertension, oedema of the extremities, prolonged ileus and compromised hepatic clearance of the drugs. Increased intra abdominal pressure causes compression of IVC and impaired visceral blood flow. Increased intra abdominal pressure can reduce the circulation to the kidneys resulting into release of rennin and activation of rennin - angiotensin - aldosterone system.

**C. Complications due to Genetic Disorders**

Various genetic disorders offer significant challenge to the Pediatric anesthesiologist. One of the major problem is that they may remain unrecognized initially, till some complications manifest. Some of the common genetic disorders associated with frequent postoperative complications are Trisomy-21, Duchenne's muscular dystrophy and sickle cell anemia.

**a. Trisomy-21**

Trisomy-21, commonly known as Down's syndrome, is the most common chromosomal anomaly. This is characterized by oblique palpabral fissures, flat facies, single palmer crease and dysplastic middle phalanx of the fifth digit. Major anesthetic problems are mental retardation, obesity, difficult airway and cardiac anomalies. Difficulty in intubation is because of narrow nasopharynx, large tonsils and adenoids, cervical spinal stenosis with atlanto axial Subluxation and subglottic stenosis. Anesthesiologist should take proper care during endotracheal intubation to prevent hyperextension of cervical spine. Care should also be taken during extubation as upper airway obstruction and post extubation stridor are very common. Surgical correction of various cardiac defects in Down's syndrome is often associated with postoperative respiratory complication. Abnormal development of alveoli and the pulmonary vasculature predispose to development of pulmonary hypertension. Postoperative recovery may be prolonged due to unusual susceptibility of these patients to various anesthetic agents.

**b. Genetic neuromuscular disorders**

Duchenne’s muscular dystrophy is a classical example of a neuromuscular disorder which carries significant anesthesia related mortality and morbidity. Altered muscle cells of these patients produce a flux of K⁺ in response to succinylicholine, resulting into hyperkalemia, severe circulatory instability or even cardiac arrest.
Treatment is directed towards the lowering of potassium level, which includes the administration of epinephrine and sodium bicarbonate.

Another problem in children with Duchenne’s muscular dystrophy is higher incidence of malignant hyperthermia.

c. Sickle cell anemia

Children with sickle cell anemia are at increased risk for anesthesia and surgery related complications. Sickling may precipitate with hypoxia, hypercarbia, acidosis, hypothermia, hypovolaemia and hypoperfusion states, all of which is very common during perioperative period.

Patients are usually anemic, hence preoperative transfusion may be necessary.

Sickle cell anemia is very often associated with cardiomyopathy, nephropathy and respiratory dysfunction, which increase complications following general anesthesia.

D. Complications associated with anesthetic technique

Children experience greater anesthetic risk than adult. Most of the complications are either due to inadequate ventilation or anesthetic overdose. Mostly, complications occur during early postoperative period. Hence intensive monitoring is recommended during shifting of the baby from operation theatre to recovery room.

a. Emergence delirium

Children are more prone to disorientation, hallucinations and uncontrolled physical activity during emergence from general anesthesia. If is more commonly seen in patients who have received potent inhalational anesthetic agents. Postoperative pain sensory deprivation (e.g., eye bandages), residual effect of anesthetic agents, unfriendly environment are, other contributory factors. Occasionally this hyperexcitable state may persist for several hours, specially in anxious patients, who have not received any premedication.

b. Respiratory depression

Respiratory depression in children following general anesthesia may be because of residual effect of potent anesthetic agents. Mechanical factors such as abdominal distension or tight abdominal bandage may also be responsible for such complication. Elevated PaCO$_2$ not always indicates inadequate ventilation Respiratory depression should be suspected when; (1) tachycardia, dyspnoea, anxiety and labored ventilation is associated with respiratory acidosis, (2) hypercarbia reduces the arterial pH<7.25 or (3) PaCO$_2$ increases progressively along with decrease in arterial pH.

Postoperative respiratory depression is usually due to residual effects of muscle relaxants, intravenous or inhalational anesthetic agents. Immediately after extubation, ventilation may be normal but after sometime respiratory depression may be evident.
Due to absence of noxious stimuli, residual effect of different anesthetic agent may be unmasked. Careful monitoring is necessary during early recovery phase to exclude such complication.

c. Postoperative hypoxemia
Hypoxemia most frequently occurs after termination of anesthesia during immediate postoperative period and then later in the recovery room. The administration of 100% oxygen at the end of anesthesia have no effect on the incidence of early hypoxemia. Late hypoxemia is usually associated with crying or breath holding, which reduces significantly by supplemental oxygen. Intubation, use of muscle relaxants, intravenous induction and duration of anesthesia more than 1 hour is associated with higher incidence of hypoxemia.

In recovery room, the acceptable lower limit of PaO₂ is 80-100 mmHg which correspond to 93-97% of SpO₂. However, adequate arterial oxygenation does not mean adequate tissue oxygenation. Sepsis, hypotension, anemia and CO-poisoning may hamper tissue oxygenation in spite of good oxygenation.

Oxygen supplementation should be done in all high risk patients or the patients with low SpO₂ readings. Use of 100% oxygen for transient period does not produce any harmful effect on newborn baby. Early signs of oxygen toxicity can only be seen after 72 hours.

d. Complications associated with intubation

a. Sore throat
Many children complain of sore throat following laryngoscopy and endotracheal intubation. Use of dry anesthetic gases is another contributory factor. Incidence of sore throat is less with laryngeal mask airway. Steam inhalation, cough lozenges and analgesics provide good relief.

b. Post extubation croup
- Post extubation croup is a well recognized complication in children following endotracheal intubation.
- Children are more prone to airway obstruction or croup because they have narrow laryngeal and tracheal lumen that may be blocked by mucosal oedema following trauma. Various precipitating factors are traumatic or repeated intubations, coughing or bucking on the tube, changing the patient’s position after intubation and, presence of upper respiratory tract infection.
- The incidence of post extubation croup has been reduced because of use of sterile, implanted tested endotracheal tube of proper size and use of heated humidified anesthetic gases.
Treatment consists of - humidified oxygen therapy and nebulized epinephrine. Role of corticosteroids is controversial.

e. Postoperative pulmonary oedema

Pulmonary oedema in postoperative period occurs mostly because of over hydration or airway obstruction. Post obstructive pulmonary oedema resolves quickly automatically. Treatment consists of positive pressure ventilation with application of PEEP and diuretics.

f. Postoperative nausea vomiting

- Postoperative nausea vomiting (PONV) is the commonest complication of general anesthesia. It is not only responsible for delayed discharge from PACU but also for unanticipated hospitalization.
- Apart from unpleasantness for the patients> PONV increases medical risks. Raised central venous pressure increases morbidity after ocular, tympanic or intracranial procedures. Increased intra abdominal pressure may jeopardize suture lines.

Incidence of PONV is very high in children specially following strabismus surgery, middle ear surgery, orchiopexy and umbilical hernia repair. Antiemetics can be used prophylactically and also for treatment of PONV. Commonly used antiemetics are phenothiazines, butyrophenones, anticholinergics, and benzamides and serotonin antagonists. All antiemetic except serotonin antagonists (e.g., ondansetron) produce sedation, which may delay recovery of the patient. These antiemetic have different site of action, so combination therapy may provide better results by simultaneously treating two or more precipitating factors.

Hepatic dysfunction

Postoperative hepatic dysfunction may be caused by the surgical procedure, the stress of surgery, ischemia, infection, preexisting undiagnosed liver disease or drugs.

Initial evaluation of the patient with hepatic dysfunction includes a thorough review of the past medical history for any evidence of genetic disorders (e.g., glucuronyl transferase abnormality), blood transfusion (reaction, hepatitis) or exposure to drugs known to produce hepatitis. Medical record should be reviewed for any evidence of sepsis, hypotension, hypoxemia, shock or congestive heart failure. Although halothane hepatitis is rare in children, Kenna et al have reported few cases in their series. Anesthetic related hepatitis is less likely to occur with newer potent inhalational anesthetic agents, such as sevoflurane, isoflurane and desflurane because their metabolism is less than halothane.
5. Complications due to succinylcholine

a. Myalgia
Administration of succinylcholine in infants and small children causes damage of the muscle cells leading to myalgia and increased plasma levels of creatinine phosphokinase and myoglobin. This myalgia is intense and may take several days to resolve. It can be minimized or prevented by pretreatment with non depolarizing muscle relaxants. Treatment is supportive and patients usually recover spontaneously.

b. Masseter spasm
Tone of masseter muscle is increased following administration of succinylcholine. This tone is maximum immediately after the caesation of fasciculation. In some patients it may be difficult to open the mouth because of the increased muscle tone, called masseter spasm. To avoid such complication two techniques have been adopted:

- Administration of larger dose of succinylcholine (2 mgkg⁻¹)
- Waiting for twenty seconds after caesation of fasciculation.

Conclusion
Pediatric patients in their first year of life are at increased risk of anesthesia related complications. Higher incidence of respiratory complications specially inadequate ventilation and hypoxemia have been observed in this group of population. Prompt diagnosis and management can prevent serious mishaps associated with these complications.

Hypoxemia shortly after discontinuation of anesthesia is a constant problem in children. Therefore, oxygen supplementation and careful attention for clear airway are essential during transport of the patient from operation theatre to recovery room.

Although presence of URI is not a contraindication for general anesthesia, it increases perioperative complications. Hence these children need intense perioperative monitoring by experienced Pediatric anesthesiologist.

Thorough preoperative assessment is also mandatory to exclude presence of any congenital abnormalities.

Strict application of these safety rules can reduce the rate of anesthesia related complications and mishaps in Pediatric population.
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Chapter 13 - POSTOPERATIVE PAIN MANAGEMENT IN PEDIATRIC PATIENTS

Introduction

Pain is perhaps the most feared symptom of disease, which a man is always trying to alleviate and conquer since ages. It is defined by the international association for study of pain as an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in term in terms of such damage. Children are special in this regard because, in them it is a very complex phenomenon. It is also very difficult to differentiate restlessness or crying due to pain from that of hunger or fear in the children. An effective pain therapy to block or modify the myriad physiologic responses to stress has become an essential component of modern pediatric anesthesia and surgical practice.

Pain assessment in children

Pain assessment is the most important and critical component of pain management. Assessing pain in children is an ever challenging as well as a difficult task, mainly because so far no reliable method of assessing and measuring child's pain is available. However, the child's self report is the single most reliable indicator of the existence and intensity of pain. Cognitive and emotional developments together with psychological defense mechanisms are important variables to be considered with Pediatric pain. Unfortunately, this is possible only in youngsters with sufficient cognitive and communicative abilities. In the infants, or children with cognitive or physical impairments, self-report is not always possible and observational assessment in the form of behavioral or biological methods are the only options available. One such standard approach of assessment of pain is QUESTT which is as follows-

- Q - Question the child
- U - Use pain rating scales
- E - Evaluate child’s behavior
- S - Secure parent’s involvement
- T - Take cause of pain into account
- T - Take earliest action
a. Question the child

Self Report:
The child's verbal statement and description of pain are important factors in assessment of pain. Children up to 2 years can report and locate the pain, although, at this age they will not be able to quantify the intensity. Questioning should be patient and in the words familiar to the child should be used. It is the best to talk to the parents before asking the child and the words that are used to describe the pain in the family "should be used. Children, at any age can deny pain if the questioner is a stranger," or are afraid of receiving injections for pain.

b. Use a pain rating scale

Faces scale:
Children up to 4-5 years old can use standardized measuring scales. One must introduce and discuss the detailed aspects of the scale to the child and his parents, before using them. Some of the methods available for self report are Hester's poker chip tool, Faces scale of Bieri et al, faces scale of Kutner and Le Page, Eland's colour scale, Visual Analog Scale (VAS), Smiley Analog Scale, Oucher Scale of Beyer and Wells, and Work Graphic Scale of Tesler et al. Ideally speaking, no one scale is better than the others.
Children older than 7-8 years can use a zero to ten numeric scale or even VAS scale. Using the above scales, pain is measured for the treatment plan as well as to gauge the success of the therapy instituted in the child.

c. Evaluate behavior and physiologic changes

Behavioral and physiologic changes:
Specific distress behaviors eg. cry, ouch, facial expression (grimace), posture (guarding) and body movements are typically associated with pain and are useful in evaluating pain in children with limited communication skills. However, it is difficult to discriminate between behavior due to pain and other types of distress eg. hunger, fear or anxiety.
<table>
<thead>
<tr>
<th></th>
<th>0</th>
<th>1</th>
<th>2</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Crying</strong></td>
<td>No</td>
<td>High pitched</td>
<td>Inconsolable</td>
</tr>
<tr>
<td><strong>Requires O2 for saturation &gt; 95%</strong></td>
<td>No</td>
<td>&lt; 30% $O_2$</td>
<td>&gt; 30% $O_2$</td>
</tr>
<tr>
<td><strong>Increased vital signs</strong></td>
<td>$HR$ and $BP = or &lt; $preop</td>
<td>Increase in $HR$ or $BP &lt; 20%$ preop</td>
<td>Increase in $HR$ or $BP &gt; 20%$ preop</td>
</tr>
<tr>
<td><strong>Expression</strong></td>
<td>None</td>
<td>Grimace</td>
<td>Grimace/Grunt</td>
</tr>
<tr>
<td><strong>Sleepless</strong></td>
<td>No</td>
<td>Wakes at frequent intervals</td>
<td>Constantly awake</td>
</tr>
</tbody>
</table>

$preop = preoperative$

Many scales for behavioral assessment have been described, namely. Directly Observed Behaviors, Children’s Hospital of Eastern Ontario Pain Scale (CHEOPS), Toddler Preschool Post operative Pain scale, Ten Item Post operative Pain Score, CRIES scale, facial expression scale of Wong et al and Nurse or Parent rating of pain.

**Physiologic changes:**

As like the behavioral measures, the physiologic changes do not discriminate between physical responses to pain and other forms of stress. Most studies of physiologic measures have assessed to the acute pain but are unreliable indicators of persistent pain. Examples of physiologic changes to pain are increase in heart rate, respiration and blood pressure, crying, sweating, decrease in oxygen saturation, dilation of pupils, flushing or pallor, nausea and muscle tension. Heart rate is probably the simplest and therefore the most appropriate. Vagal tone and heart rate variability such as during breathing have been used as indices of pain and distress. Heart rate initially decreases and then increases in response to short sharp pain.

Surgery also triggers the release of stress hormones (corticosteroids, catecholamines, glucagon and growth hormone). Except in laboratories and researches, these measurements are not been found to be useful clinically to assess and treat the pain.

**d. Secure parent’s involvement**

Parent’s should be questioned about the early recognition and child’s behavior during pain. They should be also encouraged to get actively involved in assessment, progress as well as treatment strategies of pain in their child.

**e. Take cause of pain in to account**

Etiology and or procedure may give clues to the expected intensity and type of pain.
f. Take a quick action to relieve the pain
Establish the acceptable pain level in the child and use appropriate methods to relieve it.

Pain Management in Pediatrics
There are many different modalities to treat the Pediatric pain. But before opting for an appropriate modality of pain relief, one must evaluate the relative risks or benefits, its analgesic efficacy, safety, side effects, costs and the course of recovery. The child should be prepared properly for that particular method of pain relief. A good psychological preparation of the child as well as parents, proper premedication and smooth anesthesia course always helps in reducing the anxiety and needs of pain medications in the post-operative period.
The treatment modalities include general measures, systemic drug therapy, regional techniques and non-pharmacologic approaches.

A. General measures
Child should be made comfortable and less distressed, before surgery as well us during hospital stay. These measures include presence of parent with the child, nursing in a comfortable environment allowing the child to adopt most comfortable position and feeding if permissible.

B. Systemic drug therapy

I. Non-narcotic analgesics
This group of drugs has become extremely popular for treating postoperative pain in children as they are effective with few side effects and produce an opioid sparing action through decreasing the inflammatory mediators generated at the site of tissue injury. These drugs act peripherally by inhibiting prostaglandin (PGs) and thereby, blocking the afferent pain mediators and impulses. These drugs have a ceiling effect in the higher doses, though this may increase the side effects. These are useful for mild to moderate pain or as adjuncts with narcotics to decrease the side effects of narcotics.

a) Paracetamol (Acetaminophen):
This is the most common analgesic used in the children. It is very useful as a postoperative analgesic specially if used with Ibuprofen. Owing to its safe therapeutic profile, it should be the primary postoperative pain management tool in a majority of surgical procedures. Though dose response in children is not known, <15-20 mgkg⁻¹ can be used safely orally every 4 hours. An injectable formulation of paracetamol also exists as pro-drug paracetamol. Nephrotoxicity and hepatotoxicity are the commonly encountered complications but, are not seen with short term use.
Ibuprofen:  
This is a better analgesic than acetaminophen. Safety of Ibuprofen for use in children less than 6 months of age is yet to be established. However, the pharmacokinetics in infants over 3 months is similar to adults. Oral formulations are available and 4-10 mg kg\(^{-1}\) dose\(^{-1}\) every 6-8 hours is quite effective.

c) Diclofenac: This is more powerful anti-inflammatory drug than acetaminophen and ibuprofen. However, the incidence of nephrotoxicity and GI complications are also higher with this drug. It is available in tablet, syrup as well as suppository form. The oral dose is 1-1.5 mg kg\(^{-1}\) 12 hourly.

d) Ketorolac: Ketorolac is a very useful analgesic in children and it's opioid sparing effect has been confirmed. Being a non-narcotic and with a duration of action for 4-6 hours, it is routinely prescribed even for children in empirical doses. Recently, IV route has also been declared safe in children. The IV or IM dose of ketorolac is 0.2-0.5 mg kg\(^{-1}\) every 6 hours for 48 hours. Maximum permitted total dose per day is 120 mg. The commonly seen side effects with NSAID’s are increased chances of bleeding, thrombocytopenia, precipitation of asthma attacks, increases in heart rate, retention of sodium and water, GI ulcerations, bleeding, hepatotoxicity, nephrotoxicity, nausea, vomiting, and dyspepsia etc.

e) Ketamine: Ketamine is in use routinely for almost 3 decades. The role of the N-methyl-D-aspartate receptor (NMDA) in the processing of nociceptive input has led naturally to a renewed clinical interest in the NMDA receptor antagonists such as ketamine. It can be administered alone or in conjunction with other agents via the oral, rectal, intramuscular, subcutaneous, intravenous and intraspinal routes. There are evidences about the efficacy of low dose ketamine (of less than 2 mg kg\(^{-1}\) intramuscularly or less than 1 mg kg\(^{-1}\) intravenously or epidurally) in the management of acute postoperative pain. It is been commented that a low dose ketamine may play an important role in postoperative pain management in the future but, some more study may be needed as regards the associated side effects.

II. Narcotic analgesics  
Opioids are the mainstay in the management of post-operative pain and they provide increased tolerance to pain. In newborns, clearance is diminished and elimination half lives are prolonged as compared to the older children. Maturation gets completed by 3-6 months and infants become no more susceptible to respiratory depression. But close observation of the infants is still needed, as the titration to the clinical effects is hampered due to difficulty in the pain assessment and also sometimes the presence of high risk factors like cardio-respiratory and neurological abnormalities.
The use of opioids in infants less than two months must be with proper monitoring in the intensive care setting. The elimination half life and clearance of morphine in infants older than two months of age is similar to adults. In infants from six months up to one year, injection morphine 0.1 mg kg\(^{-1}\) in or 0.05 mg kg\(^{-1}\) I.V. may be used. Careful respiratory monitoring and facilities for resuscitation must be available because of the problem of respiratory depression. In children 1-6 years, narcotics can be safely used. The intravenous route is the best for the postoperative analgesia as it provides immediate pain relief. Injection morphine 0.1 mg kg\(^{-1}\) or pethidine 1 mg kg\(^{-1}\) I.V. are the usual drugs. Children more than six years can usually communicate well about the pain perception and can cooperate with the staff in pain management. So, in them, besides all the above techniques, a number of newer techniques can be used.

a) Morphine: Morphine still remains the standard opioid for pain relief in infants and children of all age groups. It is considered safest in a dose of 0.1 mg kg\(^{-1}\) intramuscularly, in a spontaneously breathing child. However, intramuscular injections are discouraged because they result in fluctuating plasma levels and cycles of pain, comfort and sedation.

b) Codeine: This drug is used mainly as a powerful antitussive, than analgesic. A single oral dose of 1 mg kg\(^{-1}\) is good enough as both antitussive and analgesic. Respiratory depression is never seen after a single dose.

c) Pethidine: Pethidine is not very popular for post-operative pain management in children because practically it offers no advantages over morphine. Injection pethidine in a dose of 1.5-2 mg kg\(^{-1}\) IM is a useful premedicant and in a dose of 1 mg kg\(^{-1}\) I.V. is used for intraoperative and postoperative analgesia.

d) Fentanyl: Though fentanyl has been tried in doses of 1-2 μg kg\(^{-1}\), it is not a popular systemic analgesic for conventional post-operative analgesia in children.

e) Buprenorphine: In a dose of 3-5 μg kg\(^{-1}\) is a useful analgesic for intra-operative and post-operative analgesia. A tablet form for sublingual administration is suitable for use in older children who do not like injections.

f) Pentazocine: A partial agonist, can also be used in a dose of 1 mg kg\(^{-1}\) IM or 0.5-0.75 mg kg\(^{-1}\) I.V. When it is given I.V. in very small infants, careful respiratory monitoring is essential.

Common side effects encountered with opioids are nausea, vomiting, dyspepsia, constipation, urinary retention, respiratory depression, drowsiness, euphoria etc.

**Intravenous analgesia using Opioids:**

Intravenous analgesia provides immediate relief of pain. After an intravenous bolus dose of 0.1 mg kg\(^{-1}\) injection morphine, the child gets relief from pain for 1-3 hours. Intravenous analgesia can be given by two different ways as i) continuous intravenous infusion or ii) patient controlled analgesia (PCA).
i. Continuous I. V. infusion
This technique maintains the drug concentration above the therapeutic level so that it avoids the painful periods in between the empirical doses, pt requires a careful monitoring of the patient for the therapeutic effects as well as possible complications, so as to titrate the appropriate dosage.
Usually, this can be achieved by an Initial dose of 0.05 mgkg$^{-1}$ IV morphine, followed by an infusion of 0.015-0.025 mgkg$^{-1}$hr$^{-1}$ in children $<$ 6 months and 0.025-0.030 mgkg$^{-1}$hr$^{-1}$ in older children. This provides a satisfactory analgesia without respiratory depression. If a child is already intubated and being ventilated, higher doses like 0.025 mgkg$^{-1}$hr$^{-1}$ can be given even in small babies. In the newborn the dose must not exceed 10 mgkg$^{-1}$hr$^{-1}$ as the neonates have a reduced clearance of morphine and increased sensitivity to toxic effects.
'Apnea monitors' and pulse oximeters should be used specially, if opioids are being used in infants $<$ 6 months of age, or in children with acute or chronic respiratory dysfunction etc.

ii. Patient controlled analgesia (PCA)
This is another method to ensure a continuous pain relief. Patient-controlled analgesia (PCA) has been studied in the adult clinical setting since 1971. However, it was not until the late 1980s that PCA was investigated for use in the Pediatric population.
PCA has been documented as decreasing children's anxiety about painful intramuscular injections and improving their sense of control postoperatively. This is important because children may tolerate pain rather than request another analgesic injection.
An adequate preoperative preparation of the patient is needed for the use of PCA. Though expensive, still, there is a high degree of patient satisfaction as the patient himself participates in pain managements. Following appropriate pre-operative teaching children, $>$ 6 years of age can learn to use a PCA pump. An anesthesiologist states, 'If a child can play video games, he or she can master the use of PCA. To use PCA, the child must understand the relationships between a stimulus (pain), a response (pushing the button), and a delayed result (pain relief). It is important that the child understand the expectation of PCA is pain control, not elimination of pain. The children must be carefully screened for their cognitive and physical ability to manage their pain using PCA.
This can be used as either PCA infusion alone or PCA with basal infusion. PCA has been reported to result in lower pain scores, and better satisfaction than intramuscular morphine use, even though, the total morphine used, time to oral intake, incidence of nausea and vomiting or urinary retention is same. Total hourly dose of 0.05- 0.1 mgkg$^{-1}$hr$^{-1}$ of morphine can be used. Giving a basal infusion of 1/3-1/4 of total hourly dose and PCA bolus as remaining hourly dose divided in equal doses at 6-15 minutes of
lockout periods works well. A basal morphine infusion of 12-15 mgkg⁻¹hr⁻¹ has been successfully used without any side effects.

Problems and contraindications to Pediatric PCA
Contraindications to children using PCA are specific physical or cognitive disabilities or conditions that may prevent safe and effective self administration. This may include the inability to activate the device to deliver the opioid dose or an inability to understand the process. The child must have the ability to comprehend PCA instructing and understand the concepts of "greater than" and "less than" to report pain scores.
Allowing someone other than the patient to activate the PCA button removes a PCA safety mechanism. If patient is the only person pushing the PCA button, and if he falls asleep the dosing will get interrupted. Even, Family Controlled Analgesia (FCA) and Nurse Controlled Analgesia (NCA) have caused over sedation and respiratory depression in some cases. FCA remains a controversial pain management technique.
A 1.7% incidence of respiration depression was reported in children receiving parent controlled analgesia in combination with nurse controlled analgesia (NCA).

Adverse effects of PCA in the pediatric population
It is difficult to estimate the number of adverse events associated with PCA therapy in pediatrics. However, medication adverse events are grossly underreported, especially in pediatrics. Overall, pediatric patients are at high risk for adverse drug events because of several factors. These include the need for calculation of individualized doses based on age, body weight, BSA, and clinical condition, as well as the unique and rapidly changing pharmacokinetic parameters exhibited by infants and children at various ages and stages of maturational development.
Respiratory depression in pediatric patients receiving PCA therapy alone has been reported by one reviewer to range from 0 to 1.1%.
To decrease such complications due to an inadvertent overdose secondary to supplemental opioid doses, an assessment of the patient's use of the PCA before the additional dosing or dosing adjustments is necessary. Addition of an adjuvant agent, such as intravenous ketorolac, may improve analgesia without contributing to opioid adverse effects.

C. Regional techniques
Regional blocks are becoming increasingly popular in Pediatric surgery. It is also known that supplementing general anesthetic with regional or nerve blocks, allows a smooth intra-operative course, decreased requirements for general anesthesia drugs, decreased stress response, pain free awakening, and avoidance of potentially deleterious side effects that may occur with parenteral administration of narcotics during surgery and
above all, an excellent post operative pain relief. If used for thoracic and upper abdominal surgery, regional anesthesia improves pulmonary function also. Sometimes, regional anesthesia is given to a child even without a general anesthesia eg. older and co-operative child who requires emergency peripheral surgery after recent food ingestion, child with chronic airway disease like asthma, child with neuromuscular disease having compromised respiratory reserve and poor pharyngeal and laryngeal reflexes, and a child with family history of malignant hyperpyrexia.

Local anesthetic drugs are used for regional blocks. One must be aware that in infants less than 2 months less bupivacaine is bound to the plasma proteins because of low levels of albumin resulting in higher concentration of the free drug. Elimination half life is also prolonged. The myelination of nerves is incomplete in infants.

Various regional techniques which have been used in children are lumbar epidural, caudal epidural, intercostal, ilio inguinal and ilio hypogastric, 3 in 1 block, sciatic nerve block, fascia iliaca block, brachial plexus block, wrist block, penile block, infiltration block and topical analgesia.

Before a regional or nerve block is done, considerations must be given regarding NPO status, emergency airway accesses, intravenous access, standard monitoring of cardio-respiratory function and resuscitative measures like oxygen, suction, equipment for ventilation and intubation, and emergency drugs etc.

**Epidural injection**

Epidural injection can be done at thoracic, lumbar and caudal levels in children. Single shot caudal blocks are quite popular in the routine clinical practice in the children. The child almost always requires another method of pain relief after 3-4 hours in case of bupivacaine and 8-16 hours in case of morphine injection given caudally.

**Sacral epidural (caudal) analgesia**

This is the most popular and useful regional block in Pediatrics. It is simple to perform and easily adaptable to day-care surgery. Common indications of caudal block are circumcision, hypospadiasis repair, Cystoscopy, anal surgery and club foot repair. Inguinal surgery like hernia, hemicolecotomy and orchidopexy can also be performed under sacral epidural analgesia.

Undesired effects like numbness and motor weakness after an epidural injection can be often distressing for the child in the post-operative period. Epidural narcotics in this situation have a more promising role.

**Epidural clonidine**

Recently, clonidine has been discovered to enhance and prolong the analgesia produced by epidural blocks. A 3 mgkg⁻¹ of clonidine by lumbar epidural route and 5 mgkg⁻¹ by caudal route does not have significant hemodynamic or sedative effect. However, 5 mgkg⁻¹ by lumbar route have been found to cause significant hypotension and bradycardia.
Neuraxial opioids
Morphine, fentanyl and sufentanyl have been used in single bolus doses or by continuous infusions by the epidural route. Pain relief is obtained without any motor or sensory blockade. Neuraxial opioids should be avoided in all infants who are born premature. Moreover, the child should be subjected for monitoring by an apnoea monitor and oximetry under the supervision of a nurse or a doctor.
Caudal epidural morphine can be given in a dose of 0.03-0.05 mgkg⁻¹. Higher dosages like 0.1 mgkg⁻¹ has been reported to cause respiratory depression." Single dose lumbar epidural morphine has been used in doses of 0.05 mgkg⁻¹ for abdominal and lower extremity surgeries and in doses of 0.12-0.15 mgkg⁻¹ for thoractomies in a volume of 0.05-0.5 mlkg⁻¹ segment. The analgesia lasts for 8-12 hours. For epidural infusion, a bolus of 0.03-0.05 mgkg⁻¹ is injected followed by an infusion of 0.004-0.006 mgkg⁻¹ hr⁻¹. The dose of epidural fentanyl is 0.5-1 µgkg⁻¹ single dose in a volume of 0.05 mlkg⁻¹ but the analgesia lasts for only 3-4 hours. Epidural sufentanil can be given in a single dose of 0.75 µgkg⁻¹ in the same volume as fentanyl but the duration of analgesia is only 2 hours. The adverse effects due to epidural opioids are almost same like an opioid given by any other route, though they may not be that severe and commoner. Though rare, but the respiratory depression, following epidural opioids is the most serious complication. Nausea and pruritis are also commonly seen and can be taken care as suggested in (table 1).
Urinary retention can occur with both epidural local anesthetics as well as epidural opioids. Table 1

<table>
<thead>
<tr>
<th><strong>Adverse effect</strong></th>
<th><strong>Treatment options</strong></th>
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<tbody>
<tr>
<td><strong>Respiratory Depression</strong></td>
<td>Stop opioid + Airway management</td>
</tr>
<tr>
<td></td>
<td>Naloxone – 0.5-1 µgkg⁻¹ dose⁻¹ – as I.V. bolus</td>
</tr>
<tr>
<td></td>
<td>0.5-1 µgkg⁻¹hr⁻¹ as infusion</td>
</tr>
<tr>
<td><strong>Sedation</strong></td>
<td>Stop opioid</td>
</tr>
<tr>
<td></td>
<td>Stimulant medication like Methylphenidate</td>
</tr>
<tr>
<td><strong>Dysphoria</strong></td>
<td>Change opioid drug</td>
</tr>
<tr>
<td><strong>Constipation</strong></td>
<td>Stool softener like cremaffin / enema</td>
</tr>
<tr>
<td><strong>Nausea and vomiting</strong></td>
<td>Promethazine 0.25 mgkg⁻¹ up to 25 mg I.V./I.M.</td>
</tr>
<tr>
<td></td>
<td>Droperidol 0.01 mgkg⁻¹ up to 0.625 mg</td>
</tr>
<tr>
<td></td>
<td>Ondansetron 0.1 mgkg⁻¹ I.V. up to 4 mg</td>
</tr>
<tr>
<td><strong>Pruritis / itching</strong></td>
<td>Diphenhydramine 0.05 µgkg⁻¹ dose⁻¹</td>
</tr>
<tr>
<td></td>
<td>Local smoothening agents like caladryl etc.</td>
</tr>
<tr>
<td><strong>Urinary retention</strong></td>
<td>Catheterization, if needed</td>
</tr>
<tr>
<td></td>
<td>Bethanechol 0.05 mgkg⁻¹ dose⁻¹ s.c.</td>
</tr>
</tbody>
</table>
Continuous intercostal block

This technique provides analgesia in fractured ribs and upper abdominal or thoracic surgery. This block has been seen to accelerate extubation and to improve the vital capacity postoperatively, and thereby to decrease pulmonary complications following thoractomies. The catheter is placed by the surgeon with the chest open, medial and superior to the posterior edge of the incision and dorsal to parietal pleura. The tip should be posterio-medial, a few centimeters lateral to spine. Lignocaine dose should be limited to 4-6 mgkg$^{-1}$, while, Bupivacaine should be limited to 0.3-0.4 mgkg$^{-1}$hr$^{-1}$.

Nerve block of the penis

I. The dorsal nerve of penis block: This is performed by injections at 10.30 and 1.30 clock positions deep to the Buck’s fascia. 1-3 ml of 0.25% bupivacaine or 1% lignocaine is used both sides of the midline for the block. It must be ensured that there is no intravascular injection and that the local anesthetics do not contain epinephrine.

II. Subcutaneous ring block: This involves subcutaneous infiltration of 0.25% bupivacaine outside Buck’s fascia.

III. Topical lignocaine: Lignocaine jelly has been used to provide analgesia after circumcision and has been used in the post discharge treatment of pain.

D. Non-pharmacological approaches

Various non-pharmacological approaches eg. psychological interventions like hypnosis, behavioral therapy, acupuncture, transcutaneous electrical nerve stimulation have been described for post-operative analgesia. As all these techniques need a co-operation from the child, it’s usefulness is limited only in a select group of children. TENS have been seen to reduce postoperative narcotic requirement after thoractomies.
Chapter 14 - NEONATAL ASPHYXIA

Definition:
Defined by WHO as a condition wherein there is hypoxia, hypercarbia and metabolic acidosis.

Incidence: 3.7 to 9 / 1000 infants

**Causes of birth asphyxia**

Maternal  Placental  Fetal

**Placental factors:**
- Utero placental blood flow – greater it is more drug transfer ↓ drug transfer during uterine contractions, hypotension and aortocaval compression.
- Placental membrane – as pregnancy, advances, thickness of membrane ↓ and permeability ↑.

**Fetal factors:**
Fetal circulation: major portion of the drug entering Fetal circulation is extracted by liver before it reaches the brain and myocardium.
Fetal pH: during asphyxia with ↓ pH, blood flow through the ductus venosus ↑, allowing more drug into central circulation affecting target organs.
With local anesthetic drugs – ion trapping
Fetal metabolism: ↓ hepatic enzyme activity, there is ↓ drug metabolism, drugs remain longer in fetus – toxic effect.
### EFFECTS OF ASPHYXIA IN VARIOUS SYSTEMS:

<table>
<thead>
<tr>
<th>Systems</th>
<th>Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Central nervous system</td>
<td>Cerebral hemorrhages (intraventricular and subdural)</td>
</tr>
<tr>
<td></td>
<td>Cerebral edema</td>
</tr>
<tr>
<td></td>
<td>Hypoxic ischemic encephalopathy</td>
</tr>
<tr>
<td></td>
<td>Seizures</td>
</tr>
<tr>
<td>Cardiovascular system</td>
<td>Myocardial dysfunction</td>
</tr>
<tr>
<td></td>
<td>Cardiogenic shock, hypotension</td>
</tr>
<tr>
<td></td>
<td>Functional T.R sec to acute cardiac dilation</td>
</tr>
<tr>
<td></td>
<td>Arrhythmias, papillary muscle necrosis</td>
</tr>
<tr>
<td></td>
<td>Persistence of fetal circulation (persistent D.A and F.O)</td>
</tr>
<tr>
<td>Respiratory system</td>
<td>Delayed onset of respiration (&gt; 90sec)</td>
</tr>
<tr>
<td></td>
<td>Respiratory distress syndrome (due to surfactant deficiency</td>
</tr>
<tr>
<td></td>
<td>and hypoperfusion of pulmonary vasculature)</td>
</tr>
<tr>
<td></td>
<td>Meconium aspiration syndrome</td>
</tr>
<tr>
<td>Renal system</td>
<td>Acute tubular necrosis oliguria, Ac retention of urine</td>
</tr>
<tr>
<td>GIT</td>
<td>Necrotizing enterocolitis</td>
</tr>
<tr>
<td>Hematological</td>
<td>DIC</td>
</tr>
<tr>
<td>Metabolic</td>
<td>Acidosis, inappropriate ADH secretion, resulting in</td>
</tr>
<tr>
<td></td>
<td>concentrated urine, dilute plasma and hyponatremia</td>
</tr>
<tr>
<td>Hormonal</td>
<td>↑levels of catecholamines, vasopressin, rennin</td>
</tr>
</tbody>
</table>

**Asphyxia detected by**

- Fetal scalp pH < 7.2 and cord blood pH 7.26 (umb. A)
- Abnormal cardiotocography traces with late deceleration
- Meconium stained amniotic fluid
- Apgar scoring significant indicator of severity of asphyxia
Chapter 15 - PLACENTAL TRANSFER OF DRUGS OF ANESTHETIC IMPORTANCE

Narcotics:

1) Pethidine:

*Crosses placenta*
Safe dose – 50-100mg
Administration during 1st trimester – polydactyly, hypospadiasis.
Higher doses – changes in FHR variability, Apgar scores – low, changes in oxygen saturation, convulsions.
Dose delivery interval: < 1 hr: less neonatal depression
> 5 hrs: lower APGAR scores

Elimination ½ life: in adults 3 hrs
In neonates 24-30 hrs

Pentazocine:
Transfer less than pethidine
Causes maternal and neonatal CNS depression chronic maternal administration causes withdrawal symptoms in neonate – trembling, hypertonia, irritability, high pitched cry.

Morphine:
- Causes greater neonatal depression and FHR variability than other narcotics.
- Longer duration of action.
- Dose delivery of 3 ½ hrs causes neonatal depression.
- Teratogenic effects - ↓ in fetal brain size.

Fentanyl:
- Rapidly crosses placenta
- Safe dose 1μg/kg – no effect on Apgar and neuro behaviour scores.

Naloxone:
- Crosses placenta and detected in fetus in min.
- Reverses respiratory depression in neonates exposed
- To narcotics in utero in dose of 0.01mg/kg to 0.1mg/kg (IM/IV/ETT/SC)

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Should not be given prophylactically to mother to antagonize the narcotic effects because it reverse maternal analgesia.

**Propofol:**
use is limited
In dose of 2.5mg/kg (induction) – neonatal depression and lower APGAR scores, muscular hypotonia.

**Benzodiazepines:**

**Diazepam:** crosses placenta rapidly
- **1st trimester:** oral clefts, craniofacial, asymmetry, cardiac defects, pyloric stenosis.
- **2nd trimester:** hemangioma, cardiac defects (very rare)
- **Larger doses:** 30mg over several hrs causes – hypotonia, hypothermia, reduced variability in FHR, lethargy, poor feeding, low APGAR scores floppy baby syndrome.
- **Safe dose:** < 5mg or single injections of 0.3mg/kg. The drug and its metabolite persist in neonate ~ 1 wk.

**Midazolam:** transfer is less than diazepam
Produces neonatal depression < diazepam but more than thiopentone

**INHALATION AGENTS:**

**Nitrous oxide:** rapid placental transfer
Reaches 87% of maternal concentration in 20mm. Its use can adversely affect a severely asphyxiated fetus.

**Halogenated volatile agents:**
Safe concentration: Halothane 0.5%, enflurane 1%, and isoflurane 0.75%
No change in uterine tone, responsive to oxytocin
No effects on neonatal acidosis and oxygenation.

**Halothane:**
2 Mac of halothane causes ↓ maternal B.P and cardiac output and ↓ uterine blood flow and fetus – hypoxic and acidotic. Being myometrial relaxants in higher concentrations - ↓ uterine contractility and cause PPH.
Enflurane:

At concentration > 1% causes maternal and fetal bradycardia, \( \downarrow \) uterine blood flow and fetal acidosis.

Metabolized to inorganic fluoride – negligible chance for nephrotoxicity.

Isoflurane: 2 Mac isoflurane causes \( \downarrow \) cardiac output and uterine blood flow resulting in fetal acidosis.

Studies following use of halogenated agents reveal – carcinogenicity, teratogenicity and mutagenicity in fetus.

Barbiturates:

Thiopentone:

- Rapid transfer because largely unionized at physiological pH. But fetal concentration is less due to extensive protein binding in mother.
- During concentration in fetal brain is less because –
  - Major portion of fetal blood from placenta enters portal circulation and drug is extracted before it enters systemic circulation.
  - Dilution of blood (from viscera and lower extremities) before the drug reaches the brain.

Thus, with a dose of 4mg/kg no fetal CNS depression occurred.

Dissociative anesthetic ketamine
Safe dose 0.2-0.4mg/kg (analgesia)

1mg/kg (induction)

Doses > 1.5 mg/kg \( \rightarrow \) low APGAR scores and hypertonia noted in 1st trimester – cranial anomaly (rare).

Local anesthetics:

Local anesthetics are weak bases and fetal acidosis leads to ion trapping in the fetal circulation i.e. they cross the placenta in nonionized form and become ionized in fetal circulation which cannot reenter the maternal circulation.

Lignocaine:

- Rapidly crosses placenta
- Produces indirect effects on fetus following epidural and spinal anesthesia resulting in fetal bradycardia and acidosis.
- When used in cont. epidural analgesia – tachyphylaxis drug accumulation \( \rightarrow \) \( \uparrow \) placental transfer.
Bupivacaine:
- Limited transfer due to high protein binding and ionization.
- Drug accumulation is less.
- Following, Spinal, very small amounts, cross placenta and do not have any detrimental effects on fetus.
- Following Epidural maternal peak concentration – 15-30min and the umbilical vein concentration is < 30% maternal concentration even after repeated injections.

Magnesium sulphate:
Given during severe eclampsia may cause hypotonia and resp. failure in neonate.

Adrenaline:
- Have limited transfer because they are metabolized by the monoamine oxidase present in the placental membrane.
- Decrease utero placental perfusion, cause neonatal depression. Alteration in FHR and decreased movement.
- Ephedrine – vasopressor used in maternal hypotension crosses placenta and causes significant increase in H.R and beat to beat variability.
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Chapter 16 - NEONATAL RESUSCITATION

- The word “Resuscitation” is derived from the Latin word “resuscitate” meaning to arouse again.
- The neonatal resuscitation should be performed with high competence because the way in which the asphyxiated neonate is managed in the first few minutes of life, have consequences over in entire lifetime, affecting one’s quality of life.
- The goal of neonatal resuscitation should be – to initiate a timely and effective resuscitation so that the insults of hypoxia are reversed before permanent injury occurs.
- The Pediatricians and anesthesiologists are usually trained to provide the resuscitation and the delivery room personnel also share this responsibility.

NEONATAL RESUSCITATION EQUIPMENT:

Resuscitation trolley:
- Resuscitation bed with a tilt.
- Servo controlled radiant heater.
- Oxygen supply (5L/min) flow through variable PR value SBT at 30cm H₂O.

Suction equipment
- Bulb syringe
- Mechanical suction apparatus
- Soft suction catheters
  - FG 8 and 10 (airway)
  - FG 2 and 3 (ETT)
- Nasogastric tubes – FG 6 and 8

Bag and mask and intubation equipment:
- Self inflating resuscitation bag, AMBU bag
- Face mask
- Infant laryngoscopes. 0, 1 size Straight blade / magill / oxford
- Oropharyngeal airways – sizes 00 and 000
- Endotracheal tubes sizes – 2, 2.5, 3 and 3.5
- Endotracheal tube introducers

Ventilation system:
- Modified Jackson Rees system – to provide peep and rapid ventilatory rates.
- Should not have one-way valve

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**Umbilical vessel catheterization tray:**
Umbilical catheters 3 ½ and 5 Fr
3 way stop cock, sterile disposable syringes

**Monitoring:**
- ECG, pulse oximeter, umbilical venous catheter for CVP measurement.
- Umbilical artery catheters with $\text{PaO}_2$ electrodes
- Umbilical artery catheterization provides – measurement of blood gases, pH, MAP, drug administration.
- To expand blood volume

**THE INITIAL EVALUATION AND STABILIZATION OF THE NEONATE:**
Following the delivery, 1st the mouth and then the nose are suctioned.
Baby is dried and wrapped in warm towel. Placed in the radiantly heated resuscitation bed with the head at a lower level.
Tactile stimulation to initiate respiration
- Flicking the foot
- Rubbing the back
Simultaneously H.R auscultated, respiratory pattern and color notes apgar scoring done.

**Apgar score:**
- Devised by virgina APGAR (1953)
- Is a simple useful guide to neonatal wellbeing and resuscitation.
- Has five physiological variables and each variable is evaluated and scored – 0 to 2 in an infant at 1min and 5 min of age.
- The 1min score correlates with acidosis and survival.
- The 5min score predictive of the neurological outcome.

<table>
<thead>
<tr>
<th>Clinical feature</th>
<th>Score</th>
<th>0</th>
<th>1</th>
<th>2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate</td>
<td>Zero</td>
<td>&lt; 100/min</td>
<td>&gt; 100/min</td>
<td></td>
</tr>
<tr>
<td>Respiration</td>
<td>Absent</td>
<td>Gasping or irregular</td>
<td>Regular and crying</td>
<td></td>
</tr>
<tr>
<td>Muscle tone</td>
<td>Flaccid or limp</td>
<td>Some flexion of the extremities</td>
<td>Active body movements</td>
<td></td>
</tr>
<tr>
<td>Reflex irritability (response to the insertion of nasal catheter)</td>
<td>No response</td>
<td>Grimace</td>
<td>Cough, sneeze</td>
<td></td>
</tr>
<tr>
<td>Color</td>
<td>Blue, pale</td>
<td>Body pink extremities blue</td>
<td>Completely pink</td>
<td></td>
</tr>
</tbody>
</table>
Resuscitation after initial assessment:

On the basis of initial assessment, by 60-90 sec of age most infants fall into following groups and acc. resuscitated

Fit and healthy neonate:

- Apgar score 8 to 10
  - No active resuscitation req.
  - Gentle oral and nasal suctioning
  - Drying and wrapping with warm blanket, maintain body temperature.

Blue and apnoeic, breathing inadequately,

Heart rate 80-100, in primary apnoea APGAR score 4 to 7

- Respond to peripheral stimuli.
- Attempt to breathe
- Treat by administration of oxygen by face mask.
- (if no response) then bag and mask ventilation.
- If above measures fail, then intubate and PPV response is rapid – baby starts breathing and --. Pink

Neonates in terminal apnoea:

Is white / pale, heart rate < 80 / min. Apgar score < 3

No response to ext. stimuli

- No attempt to breathe
- No muscle tone
- Asphyxiated and require skillful resuscitation.

Resuscitation involves

Pulmonary resuscitation

- Establish - airway
- Initiate - breathing

Cardiovascular resuscitation

- Chest compression
- Maintenance of circulation
  - Volume expanders
  - Medications

Correct metabolic status
PULMONARY RESUSCITATION:
Asphyxiated neonates in terminal apnoea should be intubated immediately and PPV administered.

Tracheal intubation:
- After initial oral and nasal suction
- Done with a straight blade laryngoscope (sizes 0, 1)
- ETT size of 3.0 I.D for full term neonate and 2.5mm I.D in a preterm
- Position: head in same plane of the body

Tracheal suctioning:
Is done in meconium stained amniotic fluid / vaginal bleeding before ventilation is started.

Ventilation:
- PPV is provided with airway pressure of 15-25 cm H₂O.
- 2 respiratory rate of 40-60 breaths / min. Every 5th breath held for 2-3 sec to expand the atelectasis lung and remove the lung fluid.
- High inflatory pressure required: meconium aspiration, diaphragmatic hernia, congenital lung anomalies.
- In preterm neonates it is important to maintain oxygen saturation 87-95% as they are prone for retrolental fibroplasia.
CARDIOVASCULAR RESUSCITATION:

Chest compression / cardiac massage
- Started when H.R < 60/min or 60-130 / min not responding to PPV with 100% oxygen at rate of 100-150 times / min ratio to ventilation 3:1.

Umbilical vessel catheterization:
- Done in asphyxiated and preterm neonates
- Umbilical artery catheterization: Measure blood gases, pH
  - Arterial PR
  - Expand blood volume
  - Drug administration

Umbilical vein catheterization: CVP, IVF and drug administration.

CORRECTION OF ACIDOSIS:
- Respiratory acidosis is corrected by controlled ventilation.
- Metabolic acidosis treated by infusing sodium bicarbonate
- When APGAR score is 2 or less at 2’ and 5 or less at 5’
- When pH < 7.0 and PaCO₂ < 35mm Hg then

Dose of bicarbonate = \( \frac{0.3 \times \text{wt in kg} \times \text{Base deficit}}{4} \)

During sodium bicarbonate administration to consider the following.
- There should be effective ventilation
- (because \( \uparrow \text{PaCO}_2 \) – dilates cerebral blood vessels - \( \uparrow \) cerebral blood flow and may cause intracranial haemorrhage).
- May induce hypotension
- (correction of acidosis \( \downarrow \) peripheral vascular resistance and the neonates blood volume is inadequate to fill the expanded intravascular space thus causing hypotension)
- Sodium bicarbonate is very hypertonic (1800 mosm/L) therefore when a large volume of sodium bicarbonate given rapidly (1mEq/L/min). the intravascular volume expands rapidly causing intracranial haemorrhage.
- 1/4\(^{th}\) dose, diluted with sterile water 1:1 and given over 2 min blood gases and pH to be repeated before giving further doses.

HYPOVOLEMIA:
- Occurs in 60% asphyxiated preterm neonates weighing < 1.5Ka.
- With intrauterine asphyxia, with placental abruption.
- When cord is clamped early or when the cord is tightly wound around the neck must be cut to deliver the baby.
- Clinical features: pale, cold extremities, absent pulses, poor perfusion, poor capillary refill.

**Detected by:** CVP through umbilical vein catheterization
- Normal CVP 4 to 12cm H₂O
- < 4cm H₂O umbilical artery catheterization
- MAP through umbilical artery catheterization
- Normal 40-50mmHg.

**Treated by:** Best replacement is with 10ml/kg of packed RBCs
- Or 10ml/kg plasma or 10ml/kg of R.L.
- Or 10ml/kg 5% albumin – saline
- The volume replacement should be done slowly over 5-10min.
- In severely asphyxiated neonates with myocardial failure, there is persistent hypotension even after resuscitation, they are started on dopamine 2.5 to 5μg/kg/min starting dose and increased to 15 to 20 μg/kg/min.

**Neonates failing to respond to resuscitation:**

**Causes:**
- Technical errors
- RDS, meconium aspiration
- Anemia
- Pneumothorax
- (due to high inflation PR during ventilation and small ETT pushed into lobar bronchus)
- Pierre robin syndrome
- Laryngeal webs
- Tracheal atresia, pulmonary hypoplasia
- Diaphragmatic hernia
- Structural CNS malformations
- Dystrophia myotonia
- Congenital myasthenia gravis
### Medications Used for Neonatal Resuscitation:

<table>
<thead>
<tr>
<th>Medication</th>
<th>Concentration</th>
<th>Indication</th>
<th>Dosage / Route</th>
<th>Rate / Precautions / Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Epinephrine</strong></td>
<td>1:10,000</td>
<td>Heart rate &lt; 80/min after 30 sec of IPPV and cardiac massage</td>
<td>0.1 to 0.3ml/kg IV or ET</td>
<td>Give rapidly dilute with N.S to 1-2ml if giving ET</td>
</tr>
<tr>
<td><strong>Volume expanders</strong></td>
<td></td>
<td>Hypovolemia</td>
<td>10ml/kg IV</td>
<td>Give over 5-10 min not to over expand may cause hypertension and intracranial haemorrhage in asphyxiated</td>
</tr>
<tr>
<td>5% albumin saline</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal saline</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ringer lactate</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Sodium bicarbonate</strong></td>
<td>0.5mEq/ml (4.2% soln)</td>
<td>Metabolic acidosis</td>
<td>2mEq/kg IV</td>
<td>Give slowly over 2 min should be effectively ventilated</td>
</tr>
<tr>
<td><strong>Naloxone hydrochloride</strong></td>
<td>0.4mg/kg or 1mg/ml</td>
<td>Neonatal respiratory depression following narcotic administration in mother within 4 hrs prior to delivery</td>
<td>0.1mg/kg IV, ET, IM, SC</td>
<td>Give rapidly ventilated effectively</td>
</tr>
<tr>
<td><strong>Dopamine</strong></td>
<td>6 x wt x desired dose in kg (µg/kg/min)</td>
<td>Begin with 2.5 to 5 µg/kg/min and increased to 15 to 20 µg/kg/min</td>
<td>Given as continuous infusion, monitor H.R and B.P.</td>
<td></td>
</tr>
</tbody>
</table>

**Resuscitation of newborn:**

- Place under radiant heater
- Position – head down tilt
- Suction – mouth then nose

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- If meconium stained – laryngoscope suction pharynx, larynx and trachea
- Provide tactile stimulation
If No Drug depression

**EVAL HR**

<table>
<thead>
<tr>
<th>BELOW 60</th>
<th>60 - 100</th>
<th>ABOVE 100</th>
</tr>
</thead>
<tbody>
<tr>
<td>Continue ventilation</td>
<td>HR NOT INCREASING</td>
<td>Watch for spontaneous respiration</td>
</tr>
<tr>
<td>Chest compression</td>
<td>Continue ventilation</td>
<td>Then discontinue ventilation</td>
</tr>
<tr>
<td>Continue ventilation</td>
<td>If HR &lt; 80</td>
<td></td>
</tr>
</tbody>
</table>

Initiate medications if:
HR < 80 after 30 sec.
PPV ≤ 100% oxygen & Cardiac massage

**EPINEPHRINE**

May be repeated every 3-5 min.

If HR > 100
- Yes: Discontinue medication
- NO:

Metabolic acidosis (give Sodium bicarbonate)

Evidence of Acute blood loss & hypovolemia
- Give plasma expander
- If evidence of shock: Dopamine & consult
Chapter 17 - MENINGOMYEOLOCELE

- Meningomyelocele is the commonest congenital primary neural tube defect.
- 1 in 1000 live births.
- Failure of neural tube closure during 4th week of gestation.
- Failure of caudal end of neural tube to close can result in spina bifida – characterized by defects of vertebral arches.
- Meningocele – characterized by sac that contains meninges.
- Meningomyelocele – involves meninges and neural components.
- Lumbosacral: cystic mass on the back comprising a neural blockade, arachnoid, dura, nerve tissue and roots and CSF.
- Diagnosis: prenatal ultrasound, ↑ maternal serum AFP, amniotic fluid AFP folic acid def.

Signs and symptoms:
- Meningocele: born without neurologic deficits.
- Meningomyelocele: varying degree of motor and sensory deficits.
- Lumbar Meningomyelocele → flaccid paraplegia, loss of sensation to pin prick, loss of anal, urethral and vesicle sphincter tone.

Associated congenital anomalies:

Neurologic: Arnold – Chiari II malformation
(i) Caudal displacement medulla oblongata and cervical spine
(ii) Kinking of medulla
(iii) Obliteration of cisterna magna
Brain – stem dysfunction.
S/s – stridor, apnea and bradycardia, aspiration pneumonia, vocal cord paralysis, incoordination, spasticity, Hydrocephalus.

Orthopedic – club foot – dislocation of hips – scoliosis

Urogenital: extrophy of bladder, prolapsed uterus, severe dilatation of upper urinary tract (urinary diversion procedure)
Recurrent UTI → septicemia gram negative

Congenital cardiac defects

Latex allergy:
- High prevalence of clinical latex allergy and latex sensitization
- Prevalence ↑ with age.
Early intense exposure to the allergen as occurs with freq. surgeries and bladder catheterization – contributes
Preoperative H/O itching, rashes or wheezing after wearing latex gloves or inflating latex balloons.
Manifests as intra op cardiovascular collapse and bronchospasm.

**Preoperative care:**
Focus on

1. Prevention of infection
2. Maintenance of ECF for volume
3. Assessment for other congenital anomalies.
   - The sac is prone to trauma, leakage and infection → placed in prone position sac covered with saline – soaked gauze, antibiotic therapy initiated.
   - Rupture of cyst – CSF leakage → replaced with full strength balanced salt solution.
   - Have gastroesophageal reflux + vocal cord abnormality – aspiration prophylaxis

**Anesthetic management:**
Meningomyelocele operated with 72 hrs of life – to prevent development of ventriculitis and prevent neurologic deficits.

- Closure performed under local / general anesthesia.
- Positioning for induction – lateral decubitus position – intubation is challenging. Supine position – with the defect – resting in a “dough nut” support to minimize trauma.
- Awake tracheal intubation.
- IV line present – I.V induction + succinylcholine to facilitate intubation without risking hyperkalemia.
- Difficult IV access – inhalational induction
- Surgery in prone position
- Maintenance with inhaled / opioids anesthetics. Avoid muscle relaxants as identification of neural tissue may require nerve stimulation.
- Surgical closure tight enough to prevent CSF leakage – confirmed by increasing the pressure in sac with positive airway pressure.
- High index of suspicion for raised ICT → shunt insertion.
Meningocele: protrusion of meninges, contains only CSF
Meningomyelocele: protrusion of meninges with spinal cord / cauda equina and they may be adherent to the post aspect of sac.
Encephalocele: protrusion of the brain
Meningoencephalocele: protrusion of meninges as well as brain. Common sites – root of nose, occipital region, ant .fontanells
Syringomyelocele: central cord of spinal cord becomes dilated and the cord lies within the sac and becomes adherent to the post part of sac.
Myelocele: central cord of the spinal cord opens out on the surface and discharges CSF continuously. Commonest type of spina bifida but incompatible with life.

Associated with paralysis of foot and incontinence of feces and urine.

Precautions:
- The lesion should be covered with sterile dressing.
- OT should be warm.
- Awake intubation in lateral position / supine position with doughnut shaped support for the head which will support the meningomyelocele.
- Sch . for the procedure – as the surgeon used to assess the nervous system intermittently, no adverse effects like hyperkalemia are not seen.
- Less responsive to hypoxia / hypercarbia.
- Blood to be kept ready
- Trachea small, vocal cord mobility abnormal
- Surgery done in prone position and post op nursing also done in prone position.
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Chapter 18 - HYDROCEPHALUS

- In Pediatric patients is due to ↑ CSF volume, resulting in enlarged cerebral ventricles and ↑ ICP.

Classification:

Non-obstructive / communicating hydrocephalus
i. Due to overproduction or abnormal absorption of CSF
ii. No obstruction to flow of CSF

Obstructive hydrocephalus
i. Obstruction to the flow of CSF and its absorption in SAS
ii. Due to congenital, neoplastic, post traumatic and post inflammatory lesions.

Congenital causes of obstructive hydrocephalus
A. Arnold – chiari malformations – basilar subarachnoid pathways under development.
B. Aqueductal stenosis – between 3rd and 4th ventricle.
C. Dandy-Walker syndrome – occlusion at the outlet of 4th ventricle by a congenital membrane.

Intraventricular – periventricular hemorrhage which often occurs in premature infants is followed by ventricular dilatation.

Signs and symptoms:
Depends on age of child and rapidity with which ICP↑.
- Congenital hydrocephalus – abnormal enlargement of the head usually prominent infrontal areas.
  a. Cranial vault transilluminates in affected areas
  b. Cranial sutures are separated
  c. Percussion of skull – resonant note
  d. Eyes deviated inferiorly (sun-set sign)
  e. Scalp veins dilated – skin thin and shiny
  f. Optic atrophy in chronic, untreated cases
- Later onset hydrocephalus – no enlarged head, significantly ↑ ICP
- Arnold-Chiari malformation and aqueduct stenosis – medullary and lower cranial nerve dysfunction.
  a. Swallowing abnormalities, stridor and atrophy of tongue.
  b. Varying degrees of intellectual dysfunction – does not correlate with size of ventricle / thinness of cortical mantle.
Diagnostic tests:
- Serial head circumference measurements
- Skull radiographs – CT scan of head

Treatment:
Depends on mechanisms responsible for hydrocephalus
- Operative excision of lesions responsible for obstructing flow of CSF performed if feasible.
- Shunting procedures – necessary if obstruction cannot be relieved surgically – shunt system employs a one-way valve, that directs flow of CSF away from ventricles.
  a. Ventriculosternostostomy (Torkilden’s procedure)
  b. Ventriculoatrial shunts
  c. Ventriculoperitoneal shunts (V-P shunts)

Less common → ventriculocholecystostomy and ventriculospinal shunt
- Ventriculoatrial shunts for obstructive / nonobstructive hydrocephalus
  a. Distal end of catheter placed in right atrium while monitoring the changes in venous pressure wave patterns while advancing the catheter in right atrium through SVC.
  c. Growth of child displaces the cardiac end of catheter into SVC revision of shunt / V-P shunt

Anesthetic management:
- Operative procedures – placement, revision or removal CSF shunt system.
- Hydrocephalic infants and children with (NL) intracranial pressures.
  a. Induction with short acting induction drugs + muscle relaxants → tracheal intubation
  b. Maintenance with volatile anesthetics / opioids + N₂O + MR
- Hydrocephalus with co-existing intracranial hypertension – precautions during anesthesia.

  Shunt is to be inserted before craniotomy to excise an intracranial tumor.
- Potential for further increases in ICP in association with use of scoline.
- Nevertheless, scoline induced ↑ in ICP does not always occur and its use can be justified if there is need for rapid onset of muscle paralysis.
- Sudden hypotension sometimes occurs if tensely distended cerebral ventricles are decompressed.
Venous air embolism or and ↑ blood loss can occur when large neck veins are opened to place an atrial catheter.

- Postoperatively – slightly head up position – to permit free drainage of CSF.
- During surgery in children with V-P shunts – excessive pressure on the skin of scalp overlying the shunt avoided by rotating the head to the opposite side of shunt.
- Awake tracheal intubation, crying, struggling and straining - ↑ ICP.
- Major concerns – protection of airway and control of ICP. Rapid sequence induction pretreatment with atracurium 0.05mg/kg I.V. thio – 3-4mg/kg / propofol 2mg/kg IV + scoline 2mg/kg + cricoid pressure.
- Hyperventilation + Barbiturates – rapidly controls ICP.
- Maintenance – volatile anesthetics, N₂O and opioids
- Post op - trachea should remain intubated and receive PEEP if they are experiencing period of apnea / bradycardia before surgery because of intracranial abnormalities.
Chapter 19 - DOWN SYNDROME / TRISOMY 21

- An additional chromosome 21 – whole or part – results in the most common pattern of human malformation.
- Incidence 1 in 700 live births (0.15% of live births – stoelting)
- Increased maternal age during conception, multiparity – risk factors.

Characteristics:
Flat facies with oblique palpebral fissures – “mongolism” single palmar crease (“simian crease”)

Upper airway:
- Narrow nasopharynx, large tonsils and adenoids, short neck, irregular dentition, large tongue (children with mouth open and tongue protruding) chronic airway obstruction – arterial hypoxemia.
- CHD – 40% of patients (endocardial cushion defects (50%), VSD (25%) TOF, PDA, ASD secundum type.
- Surgical correction of CHD - ↑ morbidity and mortality.
- Pulmonary – subglottic stenosis, TEF, chronic pulmonary infections, impaired development of alveoli and pulmonary vasculature.
  → preop pulmonary hypertension and postoperative pulmonary complications.

GIT: chronic duodenal atresia – 300 times more frequency
CNS: microcephaly, small brain mass, mental retardation
Eye: oblique palpebral fissures, brush field spots, cataracts and strabismus → surgical correction.
Ear: otitis media and hearing loss common → freq. ear examination and myringotomies.
Teeth: dental caries → surgical repair.

Hematologic: polycythemia - ↑ risk for transitional circulation, hypotonia
Musculoskeletal: (20% - care during manipulation during laryngoscopy) asymptomatic dislocation of atlas on axis → screening for atlanto axial instability lateral radiographs in flexed, extended, and neutral position. Distance ant. arch of atlas and adjacent odontoid process > 5mm – diagnostic.

Anesthetic management:
Care should be taken during manipulation during laryngoscopy.
  a. Preop neurologic evaluation
  b. Premedication
  c. Anticholinergic drugs (atropine / glycopyrrolate) to ↓ airway secretions.
  d. Sedatives – response unpredictable oral midazolam m commonly used.

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e. IM ketamine to prepare stubborn children
   - Obesity and folds of skin at wrists and ankles → venous cannulation technically difficult.
   - Patency of upper airway difficult to maintain (short neck, small mouth, narrow nasopharynx, large tongue)/
   - Tracheal intubation usually not difficult – atlanto axial instability → extreme movement of pH head and neck during laryngoscopy to be avoided → spinal cord compression.
   - Size of ETT required is smaller – subglottic stenosis
   - Possibility of associated CHD must always be considered
   - Avoid air bubbles in IV line because of possible R → L shunts (paradoxic air embolus).
   - Respiratory complications such as post op stridor and apnea and post op pulmonary complications.
Chapter 20 - TEMPERATURE REGULATION IN INFANTS

Temperature control is a balance between heat loss and heat production.

Heat loss is due to
- ↓ thermal mass
- ↓ insulating tissue
- Increase BSA / weight ratio
- Radiation
- Conduction
- Convection.
- Evaporation through the lungs and skin

**Effects of hypothermia:**
- Metabolic acidosis + $\uparrow$ $O_2$ uptake and right → left shunting
- Resp. depression and hypoventilation
- Depressed conscious state, delayed recovery from anesthesia
- Prolonged action of drugs
- Difficult in assessing hypovolemia
- Decreased surfactant
- Dysrhythmias and cardiac depression
Chapter 21 - NONSHIVERING THERMOGENESIS (NST)

In adults shivering generates heat, and when there is hypothermia. But in infants shivering mechanism is not well developed, hence rely on “Non Shivering Thermogenesis”.

NST is brought about by brown adipose tissue (BROWN FAT) in infants. Very-LBW babies have very little brown fat.

- Brown fat constitutes 25% of body weight
- Location of brown fat: scapular, mediastinum, around kidneys and adrenal glands.
- Speciality / contents of brown fat
- These contain ↑ no of mitochondria and fat vacuoles and rich blood supply and autonomic nerve supply.

Mechanism:

```
Hypothermia
Noradrenaline released from sym nerve ending
↑ metabolic activity in brown fat

Hydrolysis of triglycerides
saturated fatty acids + glycerol

Increases O₂ consumption +
heat production
```

- NST → increases O₂ and glucose utilization → acidosis
- NST is lost ↓ anesthesia
- Brown fat deposits decline during the first few wks of extra uterine life.
Chapter 22 - FETAL HEMOGLOBIN

- In the fetus there is an altogether different hemoglobin called fetal hemoglobin (HbF).
- It is made up of 2 α and 2γ chains
- This is gradually replaced by HbA
- At birth HbF is 85% and HbA is 15%.
- HbF is resistant to denaturation by alkali – this property is used in the identification of HbF.
- By 5 months of age HbF is decreased to 5% and HbA is ↑. 2,3 -DPG conc. ↑ by about 37%.

HbF: $P_{50} = 18$ to $20 \text{ mmHg}$ Adults
$\uparrow O_2$ affinity HbA $\uparrow$
$\downarrow$ Concentration of 2,3-DPG $P_{50} 27\text{ mmHg}$
$\downarrow$ Affinity to 2,3-DPG $\uparrow$ affinity to 2,3-DPG

The ODC is shifted to left
This results in $\uparrow O_2$ affinity and $\uparrow O_2$ transport necessary to extract $O_2$ from the adult HbA from the placental circulation but the disadvantage is poor $O_2$ delivery to fetal tissue.

**Diagram:**
- O$_2$ content
- PO$_2$
- HbF
- HbA

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Chapter 23 - LOBAR EMPYSEMA

Pneumonectomy

- Rare cause of respiratory distress in newborn.
- Pathologic causes of congenital emphysema
  a. Hypoplasia of supporting cartilage – collapse of bronchi
  b. Bronchial stenosis
  c. Obstructing cysts
  d. Vascular compression of bronchi
- Acquired labor emphysema – bronchopulmonary emphysema
- Left upper and right middle lobes most commonly affected.

Signs and symptoms:


- Tachypnea
- Tachycardia
- Cyanosis
- Wheezing
- Asymmetrical breath sounds
- CXR – hyper inflated lobes mediastinal shifts.
- Bronchovascular markings in the hyper inflated lungs differentiate lobar emphysema from pneumothorax.

Anesthetic management:

Surgical lobectomy / Pneumonectomy:

Cardiovascular and pulmonary changes during mechanical ventilation.

PPV during induction – rapid expansion of emphysematous lobes (gas enters but cannot leave)
- Sudden mediastinal shifts
- Cardiac arrest

- Spontaneous breathing with minimal positive airway pres recommended.
- GA supplemented with local anesthesia until chest is opened and emphysematous lobes are delivered.
- Thereafter – infants paralyzed and lungs mechanically ventilated.
- Nitrous oxide not used as its diffusion into diseased lobes ↑ distension.
Severely decompensated infants may require emergency needle aspiration or thoracotomy for decompression of the affected lobe or lobes.
Chapter 24 - CEREBRAL PALSY

- Symptom complex rather than a specific disease.
- Comprises a group of non progressive, but often changing, motor impairment syndromes secondary to lesions or anomalies of brain that arise during early stages of development.
- Cerebral palsy is classified according to extremity involved
  o Monoplegia, hemiplegia, diplegia, quadriplegia
  o Depending on characteristics of neurologic dysfunction
  o Spastic, hypotonic, dystonic, athetotic

Incidence and risk factors:
- 1.5-2.5 per 1000 live births
- Risk factors classified according to whether they occur before pregnancy, during pregnancy or during the peritoneal period.
- Before pregnancy: H/o of fetal wastage
  o Long menstrual cycle
- During pregnancy:
  a. Low social class
  b. Congenital malformations
  c. Fetal growth retardation
  d. Twin gestation
  e. Abnormal fetal presentation

During labor and delivery:
- Premature separation of the placenta.

During the early postnatal period:
- Newborn encephalopathy
- Rate of cerebral palsy is 25 to 31 times higher among infants who weigh less than 1500g at birth than among full size newborn.

Signs and symptoms:
- Most common manifestation of cerebral palsy is skeletal muscle spasticity.
- Extrapyramidal cerebral palsy is associated with Choreoathetosis and dystonia and cerebellar ataxia is characteristics of atonic cerebral palsy.
- Varying degrees of mental retardation and speech defects can accompany cerebral palsy.

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Seizures disorders co-exist in approximately 1/3rd of individuals affected with cerebral palsy. Children with cerebral palsy may have varying degree of spasticity of different skeletal muscle groups, resulting in contractures and fixed deformities of several joints of both upper and lower extremities.

Stereotactic surgery may be performed in attempts to decrease skeletal muscle rigidity, spasticity and dyskinesia.

Gastroesophageal reflux is common in children with CNS disorders and antireflux operations may be recommended.

Children with cerebral palsy frequently receive antiseizure medications and dantrolene for relief of skeletal muscle spasticity.

Phenytoin → gingival hyperplasia and megaloblastic anemia.

Phenobarbitone → hepatic microsomal enzymes – alters liver metabolism.

Management of anesthesia:

- Tracheal intubation because of gastroesophageal reflux and poor function of laryngeal and pharyngeal reflexes.
- Body temperature – maintained, as these patients susceptible for hypothermia during intraoperative period.
- Emergence from anesthesia is quite slow because of cerebral damage due to cerebral palsy and the presence of hypothermia.
- Tracheal extubation – delayed until patient is fully awake and body temperature has returned towards normal.
- Biliary excretion of drug metabolites is unaffected by age, but reveal excretion of water soluble drugs and drug metabolites may be reduced by age related reduction in GFR and tubular secretion.
- Reduction in excitatory neurotransmitting in the brain with grey matter atrophy is thought to be the brain for the enhanced sensitivity to intravenous induction agents and reduces MAC to volatile anesthetics.

S/s: cerebral palsy

1. Skeletal muscle spasticity
2. Choreoathetosis
3. Ataxia dystonia
4. Mental retardation
5. Speech defects
6. Seizure disorders
7. Spasticity → contractures and fixed deformity
8. Gastroesophageal reflex
Chapter 25 - APGAR SCORE

- Devised by Virginia Apgar in 1953.
- It is a simple and useful guide to neonatal wellbeing and resuscitation.
- Scoring is for assessing the effectiveness of resuscitation and not the need for resuscitation.
- If resuscitation efforts are required, they should be initiated promptly and should not be delayed while Apgar score is obtained.
- It consists of 5 variables and each allotted a score 0, 1, 2 accordingly.
- It is recorded at 1st and then at 5th mins.
- If 5th min scores less than 7 then it should be recorded every 5th min till 20 mins.
- 1st min score → acidosis and survival
- 5th min score → neurological outcome

<table>
<thead>
<tr>
<th>Sign</th>
<th>0</th>
<th>1</th>
<th>2</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Heart rate (beats / min)</td>
<td>Absent</td>
<td>&lt; 100</td>
<td>&gt; 100</td>
</tr>
<tr>
<td>2. Resp. effort</td>
<td>Absent</td>
<td>Slow irregular</td>
<td>Good, crying</td>
</tr>
<tr>
<td>3. Reflex irritability</td>
<td>No response</td>
<td>Grimare</td>
<td>Crying</td>
</tr>
<tr>
<td>4. Muscle tone</td>
<td>Limp (flaccid)</td>
<td>Flexion of extremities</td>
<td>Active</td>
</tr>
<tr>
<td>5. Colour</td>
<td>Pale cyanotic</td>
<td>Body pink extremities cyanotic</td>
<td>Pink</td>
</tr>
</tbody>
</table>

1) **Score 8 to 10 (Normal):**
- Achieved by > 90% of all neonates.
- Nothing is required except nasal / oral suctioning drying of skin, maintenance of body temperature.
- Re-evaluate at 5 mins of age.

2) **Score 5 to 7:**
- Mild asphyxia
- Usually respond to vigorous stimulation and to O₂ blown over the face.
- If they are slow to respond and to become pink, they should be ventilated with 80 to 100% O₂ via bag and mask.
- At 5 minutes of age → usually well

3) **Score 3 to 4:**
- Moderate asphyxia
- Usually cyanotic and have poor respiratory efforts but usually respond to bag and mask.
● If they have not breathed spontaneously, consider ETT and ventilation. do not try to ventilate with bag and mask to avoid stomach distension and aspiration.
● Measure ABG and pH of blood → umbilical blood sample. If necessary sodium bicarbonate be administered.

4. Score 0 to 2:
● Severely asphyxiated
● Require immediate resuscitation
● Aspiration of UR tract
● ETT or IPPV with 100% O₂
● External cardiac massage
● Umbilical venous catheter
● 2-3ml /kg 8.4% sodium bicarbonate diluted 1:1 with sterile water or 10% glucose.
● Volume expansion with normal saline or blood 10-20ml/kg of indicated. Adrenaline 1 in 10,000 0.3ml/kg Umbilical Vein or endotracheal.