Principles and Practice of Pediatric Anesthesia
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To our families
For their support and patience
&
To all our little patients who inspire us to do better
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Pediatric surgery has made tremendous progress in the last three decades, because of the great advances in pediatric anesthesia. Pediatric anesthesia is now considered a superspecialty needing special skills and knowledge. Pediatric anesthesia has made the impossible possible. I am happy to state that this book is the result of great efforts taken by senior and experienced pediatric anesthesiologists from across the Indian subcontinent. They have shared their knowledge and personal experiences in their respective chapters. This book provides a systematic, comprehensive and accurate compilation of wide ranging topics pertaining to pediatric anesthesia.

It is said that children are not miniature adults, but differ anatomically and physiologically with different pharmacokinetics and pharmacodynamics. This has been well dealt with in the Basic Principles Section. All the pediatric specialties, radiological imaging procedures, cardiopulmonary resuscitation, etc. have been well written by specialists; a special mention is made on monitoring, interpretation of chest radiographs, electrocardiographs, vascular access and ultrasound-guided regional blocks.

The chapters on airway problems, special situations and medical problems, and syndromes will be very useful in day-to-day practice. I recommend this book as a valuable update on pediatric anesthesia. I am certain it will be useful to postgraduate students and pediatric anesthesiologists as a reference book, on the shelf of every hospital operation theater and library.

I appreciate the sincere efforts and congratulate the editors for this informative and well-organized book on the subject.

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Sir Robert Reynolds Macintosh has quoted almost 5 decades back; “Theme of clinical academic practice of anesthesia should be based on triad of Science, Safety and Simplicity”. The first two words, Science and Safety, will hold true at all times. However, Simplicity has to be considered in different context. The success with complexity of pediatric surgical procedures and demand for excellence in anesthesia can be achieved only by incorporating technically advanced complex anesthesia machines, monitoring systems, special skills and various complex invasive procedures.

It is time to pen down what has changed. Humongous developments have occurred in the scientific arena of pediatric anesthesia. Knowledge and understanding have expanded in all branches of pediatric anesthesia. The unique developmental aspects regarding anatomical, physiological, pharmacological, psychological and surgical conditions that require special attention and thought make pediatric anesthesia distinct. The landscape of modern pediatric anesthesia is vast in the true sense.

The purpose of this book is to provide a clear roadmap for understanding principles and practical approach to pediatric anesthesia. Our mission is translated into offering comprehensive text covering wide range of pediatric anesthesia and allied topics. We have divided the text into six sections: Basic Principles, Anesthetic Management, Subspecialty Anesthesia, Special Problems and Situations, Anesthetic Techniques, and Notes on Allied Topics. Appendices provide quick reference to pediatric drug dosages, syndromes, and handy formulae.

All the contributing authors are experienced pediatric anesthesiologists and teachers in the field, and they have offered current perspectives on the subject of their chapters. Along with compiling scientific information, each one has added their individual experience and clinical expertise for more practical and realistic application.

The book begins with a page on historical milestones in pediatric anesthesia.

In the first section of “Basic Principles”, along with anatomical growth and physiological characteristics at various stages of development and essentials of pharmacology, we have intentionally included chapters on pediatric chest X-ray and electrocardiogram. Senior pediatric cardiologists and radiologists have comprehensively described normal electrocardiogram and chest radiographs respectively, along with illustrations in different clinical scenarios.

In the second section of “Anesthetic Management” the entire process of anesthetizing a child, from the evaluation of physical status, along with anesthesia techniques and monitoring, fluid and transfusion therapy, various methods of pain management, including regional techniques, ventilation strategies are compiled in detail. Anesthesiologist’s role in the assessment and management of difficult airway is described with excellent illustrations.

In the third section of “Subspecialty Anesthesia”, the authors have detailed current perspectives of anesthetic management in different surgical branches along with chapters devoted to anesthesia in remote locations and also in the neonate for various surgical procedures. All the chapters bring us up-to-date on safe, effective and efficient perioperative practices.

The fourth section on “Special Problems and Situations” comprises of a chapter dealing with management of common medical conditions anesthesiologists face in day-to-day practice written by pediatricians, and a chapter on anesthetic management of some rare and some not so rare conditions needing special considerations. This section also includes an important chapter on cardiopulmonary resuscitation in keeping with the AHA 2015 guidelines. Pediatric anesthesiologists should also be aware of all types of complications during anesthesia, and so a separate chapter is devoted to complications during anesthesia.

The fifth section on “Anesthetic Techniques” includes a chapter on vascular access describing indications, safe techniques and complications and a chapter on ultrasound-guided regional blocks with good compilation of appropriate pictures.
The sixth section on “Notes on Allied Topics” offers pertinent information on safety and quality, ethical issues and utility of simulation in pediatric anesthesia.

The “Appendices” are intended to provide an information capsule on syndromes, drug dosing guide, and handy formulae and tables.

We are delighted to include a “Photo Gallery” which showcases various rare conditions encountered in clinical practice.

We offer our sincere thanks to all the authors for sharing their knowledge and expertise. We thank Shri Jitendar P Vij (Group Chairman), Mr Ankit Vij (Group President) and Mr Tarun Duneja (Director–Publishing) of M/s Jaypee Brothers Medical Publishers (P) Ltd, New Delhi, India, for their support and encouragement. We hope that this book will be well received and will offer comprehensive information to practising anesthesiologists, and to postgraduate students aspiring to become pediatric anesthesiologists.

Snehalata H Dhayagude
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Old anesthetic practice centuries ago comprised of "Hypnosis and trance", "Application of cold", "Pressure over peripheral nerves and blood vessels", "Alcohol intoxication", and "Ingestion of herbal concoctions". "Whisky nipple" had been used as sedative supplement to local anesthesia in infants for major surgical procedures and "wine" was used for pain relief for circumcision surgery for millennia.

- 1540—Paracelsus, Swiss Physician discovered Ether
- 1774—Joseph Priestley liberated Oxygen and obtained Nitrous oxide
- 1842—Dr Crawford Long used Ether inhalation for amputation of toe for 8-year-old child
- 1846 October 16th—WTG Morton demonstrated use of ether for tooth extraction. Every year 16th October is celebrated as "World anesthesia day".
- 1847—First recorded anesthetic deaths in children aged 11 years and 15 years
- 1857—Dr John Snow reported 100 cases of inhalational anesthesia with chloroform in children less than 1 year old
- 1858—Dr John Snow published text on chloroform and other inhalational anesthetics
- 1884—Freud and Karl Koller invented local anesthetic drugs
- 1898—August Bier of Germany introduced spinal anesthesia and used it in children also
- 1902—Cushing coined the word "Regional Anesthesia"
- 1907—James Gwathmey voiced his concern for children’s preoperative anxiety and later tribromoethanol as rectal sedative agent became popular around 1928
- 1910—Dr Tyrell Gray published detailed paper of spinal anesthesia in more than 100 children
- 1919 onwards—Ralph waters investigated toxicity of chloroform and pharmacology of cyclopropane. He invented cuffed endotracheal tubes, laryngoscopic blades, oropharyngeal airways, Carbon dioxide absorption canisters and precision controlled anesthetic vaporizers
- 1923—Sir Ivan Magill demonstrated the use of double lumen insufflations catheter for a cleft palate case
- 1930—Dr Charles Robson practiced both open drop ether and cyclopropane with tracheal intubation in kids. He advocated preinduction fasting for 4 hours in kids. He established pediatric anesthesiology in USA and Robert Cope established it in UK
- 1930—Dr Philip Ayre developed a pediatric anesthesia breathing system to be used with tracheal tube—Tpiece, valveless, non-rebreathing unit with low dead space and low resistance
- 1933—Cambell wrote an article on caudal anesthesia in children
- 1935—Leech and Leigh (1946) experimented with morphine, scopolamine, and pentobarbital for sedation and analgesia to improve perioperative experience in children
- 1937—Guedel described clinical signs of anesthetic depth and introduced airways
- 1939—Leven and Ladd performed multiple procedures for repair of tracheoesophageal fistula
- 1940—Ladd mentioned importance of supportive warming, significance of correction of electrolyte balance and intraoperative charting of clinical signs of anesthetic depth
- 1942—Griffith and Johnson from Montreal used “curare”, a relaxant in anesthesia
- 1948—M Digby Leigh from Canada authored book on "Pediatric Anesthesia"
- 1950—Dr Jackson Rees modified Ayre’s T-piece open circuit by attaching a valve-less open-ended
bag at the other end of tubing, which helped monitor spontaneous respiration or assist breaths intermittently. He advocated controlled respiration in infants with reduced tidal volumes and breathing rate of 60–80/min

- 1950—Halothane was invented in UK, introduced in practice in 1956. WT Salter stated “Without vision and research the professions die”
- 1951—Pediatricians’ Holliday and Segar derived a formula for administration of intravenous fluids in children based on daily caloric requirement. The 4-2-1 rule used by anesthesiologists to calculate hourly fluid administration is based on this
- 1950’s—Virginia Apgar standardized method of neonatal assessment at birth, coined as APGAR score
- 1963–65—Dr George Gregory and his mentor WK Hamilton (San Francisco) applied continuous positive airway pressure to infants with respiratory distress syndrome and demonstrated dramatic improvement
- 1970—Dr Alvin Hackel developed highly coordinated regional emergency transport system for sick infants and children
- 1981—Dr George Gregory reported, a series of PDA ligations in premature infants using high dose fentanyl technique
- 1980’s and 1990’s—Pediatric anesthesia grew beyond operation theaters in to outpatient clinics, procedural rooms, pain clinics. Technologically advanced monitoring equipment became available—pulse oximetry, capnography, automated blood pressure and electrocardiography—all into one multi-parameters’ monitor. Safer inhalational anesthetics—Isoflurane and Enflurane were introduced
- 1987—‘Society of Pediatric Anesthesia’ was formed
- 1991—Dr Elliot Crane and Dr Don Tyler hosted first ‘World Conference of Pediatric Pain’
- 1995 onwards—Sevoflurane, Desflurane were introduced with better safety profile
- 1980-2000 – Developments in pediatric anesthesia
  - Addressing pain response in neonates
  - Understanding narcotics in infants
  - Pediatric pain management
  - Awareness and management of apnea in premature infants
  - Evidence to help formulate preoperative fasting guidelines
  - Growth of day-care surgery
  - Safe procedural sedation
  - Evolution of pediatric cardiac anesthesia as subspecialty
  - Anesthesia education and formation of societies
- 2006—Formation of “Indian Association of Pediatric Anesthesiologists” (IAPA).
Basic Principles

Chapter 1: Anatomy, Growth and Development
Chapter 2: Physiological Characteristics and Anesthetic Implications
Chapter 3: Essentials of Pharmacology in Neonates, Infants and Children
Chapter 4: Understanding the Pediatric Chest Radiograph
Chapter 5: Interpretation of Pediatric Electrocardiogram
INTRODUCTION

Human life begins as the fertilized egg which transforms into the embryo and fetus. After completion of intrauterine gestation, begins the extrauterine life as neonate, infant, toddler, child, adolescent and eventually the adult. Organogenesis is usually complete within 8 weeks of conception, functional development of organs occurs during the second trimester and weight is gained during the third trimester. Growth and development occur simultaneously following a predictable trend. Growth denotes a net increase in size or mass of the tissues while development signifies maturation of functions and acquisition of skills needed for optimal functioning of the individual. The anatomical, physiological and pharmacological variations at each stage of growth have numerous implications as far as anesthesia is concerned. The neonate is as different from an infant as the child is from the adolescent. The job of the pediatric anesthesiologist is even more challenging with the advent of fetal surgeries and increasing survival of micropreemies.

There exist certain medical and surgical conditions unique to neonates of a particular postconceptual age; hence one needs to be familiar with the following terms:

- **Neonatal period**: This period is from birth to under 4 weeks (<28 days) of age. Early neonatal period is the first week of life (<7 days). Late neonatal period extends from the 7th to <28th day.
- **Postneonatal period**: It is the period of infancy from 28 days to < 365 days of life.
- **Perinatal period**: If extends from the 22nd week of gestation (≥154 days or weighing ≥500 gram birth) to less than 7 days of life.
- **Term neonate**: A neonate born between 37 and <42 weeks of gestation.
- **Preterm neonate**: A neonate born before 37 weeks (<259 days) of gestation from the first day of the last menstrual period irrespective of the birth weight. Neonatal problems associated with prematurity include hyaline membrane disease, bronchopulmonary dysplasia, apnea, patent ductus arteriosus, hyperbilirubinemia, hypoglycemia, hypocalcemia, hypothermia, poor gastrointestinal motility, intraventricular hemorrhage, hypotonia and electrolyte disturbances.
- **Post-term neonate**: A neonate born after 42 completed weeks (294 days or more) of gestation as calculated from the mothers last menstrual period regardless of birth weight.
- **Small for gestational age**: These are those infants whose weight is below the 10th percentile at any gestational age. This could be the result of various factors that affect intrauterine growth, e.g. toxemia, infections, congenital malformations, chromosomal anomalies, etc. Problems faced by these neonates are hypoglycemia, hypocalcemia, hypomagnesemia, thrombocytopenia, polycythemia, respiratory distress syndrome etc.
- **Large for gestational age**: Infants whose weight is above the 90th percentile at any gestational age.
They are prone to birth injuries, e.g. fractures or intracranial bleeds. Those born to diabetic mothers may have difficulty maintaining normal blood glucose concentration.

- **Low-birth weight neonate (LBW):** A neonate weighing less than 2,500 gram at birth irrespective of the gestational age.
- **Very low birth-weight neonate (VLBW):** A neonate weighing less than 1,500 gram at birth irrespective of the gestational age.
- **Extremely low-birth weight neonate:** A neonate weighing less than 1,000 gram at birth irrespective of the gestational age.
- **Intrauterine growth retardation (IUGR):** It is classified as symmetric IUGR (head circumference, length and weight are equally affected) and asymmetric IUGR (relative sparing of head growth). Symmetric IUGR often has an earlier onset and is associated with diseases that affect fetal cell number, e.g. chromosomal, genetic, teratogenic, infectious or severe maternal hypertension. Asymmetric IUGR is often of late onset and associated with poor maternal nutrition or late onset of maternal vascular disease. IUGR babies are more prone to perinatal asphyxia, polycythemia, hypoglycemia and hypothermia.

**ANTHROPOMETRY**

Anthropometric measurements are an indicator of general health of the child (Table 1). A single reading does not have much importance; it is the percentile for that particular age that is significant.

**Weight**

This being the most sensitive measure of well-being, is usually the first indication of an underlying problem. Failure to thrive could be due to various reasons, e.g. metabolic and endocrine disorders, infections, malignancies, congestive heart failure, etc.

Weight usually decreases 10% below birth weight in the first week as a result of excretion of excess extracellular fluid and limited nutritional intake. Preterm infants may lose up to 15% of their body weight during the first 7–10 days of life. While healthy LBW infants can regain birth weight in 10–14 days, VLBW babies may take as long as 3–4 weeks. In case of premature infants, it is the corrected gestational age and not the chronological age that is plotted on the growth chart while deriving the percentile for weight. Neonates regain or exceed their birth weight by 2 weeks of age and should grow at approximately 25–30 grams/day during the first month. A healthy child is expected to gain about 10 pounds per year until 12–13 years for females and 16–17 years for males. Weight in pounds can be converted to kilograms by dividing by 2.2.

**Length or Height**

Failure to increase in height follows significant weight loss. Length is measured up to 2 years and subsequently height is measured using an infantometer and a stadiometer respectively. Infants measure approximately 50 cm at birth, 60 cm at 3 months, 65 cm at 6 months, 70 cm at 9 months, 75 cm at 1 year and 90 cm at 2 years. After 4 years of age, the child gains about 6 cm in height every year until the age of 12 years.

Length in centimeters is estimated by: (age in years $\times$ 6) + 77

**Head Circumference**

It is the last to be affected and signifies severe malnutrition. It is usually measured in children up to age of 5 years using a non-stretchable tape across the occipital prominence and the glabella. Certain syndromes and craniosynostosis are associated with a small head size. At birth the head is one-fourth the total body length whereas in the adult it is one-seventh. One should suspect underlying neurologic disorders when there are significant changes in head circumference measurements.

Beginning at 34 cm at birth, the head circumference increases approximately 2 cm per month for the first 3 months, 1 cm per month between 3–6 months and 0.5 cm per month for the rest of the first year of life. It is 52 cm by 12 year of age. Thinner cranial bones of children do not afford as much protection to the brain tissue as the thicker bones of the adult skull. Larger proportion of head to body results in greater heat loss from the exposed surface. The skin over the scalp is thin and distended scalp veins are markedly visible in case of increased intracranial pressure.
Chest Circumference
It is about 3 cm less than head circumference at birth. Circumference of the head and chest are almost equal by the age of 1 year. Thereafter, the chest circumference exceeds the head circumference. At birth the chest is circular but as the infant grows the transverse diameter becomes longer than the anterior-posterior dimension giving the chest an elliptical appearance.

Midarm Circumference
It is an indicator of the nutritional status of the child. The left arm is used, the midpoint between the acromion and olecranon process is identified, and the circumference measured at this point.

Body Mass Index
During childhood, levels of body fat change beginning with high adiposity during infancy. Children >2 years with a BMI ≥ 95th percentile or >30 kg/m² fulfill the criterion for obesity. Those with a body mass index (BMI) between the 85th and 95th percentiles fall in the overweight range.

Body Surface Area
At full-term body surface area (BSA) averages 0.2 m² whereas in adults it averages 1.75 m². A normal newborn infant who weighs 3 kg is one-third the size of an adult in length but one-ninth the adult size in body surface area and 1/21 of the adult size in weight.³

BSA is recommended as the principal basis for drug dosage as the rate of metabolism or redistribution of a drug is proportional to the metabolic rate measured in kcal/m²/h.⁴ Many measurements of organ size, fluid compartment volumes and assays of blood concentration of drugs correlate well with BSA.⁵

The caloric need in relation to BSA of a full-term infant is about 30 kcal/m²/h, increases to about 50 kcal/m²/h by 2 years of age and then decreases gradually to adult level of 35 to 40 kcal/m²/h. Infants and young children have a higher metabolic rate and a larger body surface area to weight ratio than adults. Since they become dehydrated more easily, liberal fasting guidelines should be encouraged to reduce incidence of hypovolemia during the induction period.

FACE
At birth the mandible is small but as a child develops forward growth occurs and a change in facial configuration is seen. The upper jaw grows rapidly to accommodate the developing teeth. The frontal sinuses develop by 2–6 years of age. The maxillary, ethmoidal and sphenoidal sinuses appear after 6 years.

TEETH
Children between the ages of 6–10 years are prone to dental trauma during laryngoscopy or oral airway insertion since the deciduous teeth fall off during this period (Tables 2 and 3).

FONTANELLES
Anterior fontanelle (AF) closes by about 18 months. Delayed closure can occur in hydrocephalus and rickets. Early closure is found in craniosynostosis with premature closure of the sutures. AF (normal 20 ± 10 mm) should be checked in all children below 2 years with the baby in an upright position. A depressed AF suggests dehydration while a full non-pulsatile AF may point to raised intracranial pressure (ICP). In such cases, the sutures should be palpated for abnormal separation due to increased ICP. The posterior fontanelle closes by 4–6 months. The mastoid fontanelle between the occipital and parietal bones closes about 6–8 weeks after birth.

NEONATAL REFLEXES
These are unique to infants and are not seen beyond the first few months of development. A baby is born with certain reflexes which help it to feed.

Table 2: Timing of primary dentition⁶

<table>
<thead>
<tr>
<th>Primary dentition</th>
<th>Time of eruption (months)</th>
<th>Time of fall (years)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Upper</td>
<td>Lower</td>
</tr>
<tr>
<td>Central incisors</td>
<td>8–12</td>
<td>6–10</td>
</tr>
<tr>
<td>Lateral incisors</td>
<td>9–13</td>
<td>10–16</td>
</tr>
<tr>
<td>First molar</td>
<td>13–19</td>
<td>14–18</td>
</tr>
<tr>
<td>Canine</td>
<td>16–22</td>
<td>17–23</td>
</tr>
<tr>
<td>Second molar</td>
<td>25–33</td>
<td>23–31</td>
</tr>
</tbody>
</table>

Table 3: Timing of permanent dentition⁷

<table>
<thead>
<tr>
<th>Permanent dentition</th>
<th>Time of eruption (years)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Upper</td>
</tr>
<tr>
<td>First molar</td>
<td>6–7</td>
</tr>
<tr>
<td>Central incisors</td>
<td>7–8</td>
</tr>
<tr>
<td>Lateral incisors</td>
<td>8–9</td>
</tr>
<tr>
<td>Canine</td>
<td>11–12</td>
</tr>
</tbody>
</table>
Principles and Practice of Pediatric Anesthesia

- **Rooting reflex:** It helps the baby to find the nipple. When the cheek or side of the mouth is touched, the baby opens its mouth and searches for the nipple.
- **Suckling reflex:** It is very strong immediately after birth. When the baby’s palate is touched with the nipple, the baby starts sucking movements.
- **Swallowing reflex:** When the mouth is filled with milk, the baby reflexly swallows the milk; it requires coordination with breathing.

**Primitive Reflexes**

These indices of central nervous system maturity are present at birth and disappear between 3–6 months.
- **Moro reflex:** In response to a loud sound or sudden lowering of the head in relation to the trunk, the legs and head extend and the arms raise up and out. This is followed by adduction of the arms and tight fist formation with an audible cry. The hand opening is present by 28 weeks, extension and abduction by 32 weeks and anterior flexion by 37 weeks. In traumatic deliveries associated with fracture clavicle or brachial plexus injury, this reflex could be absent in that half of the body. Damage to the CNS may be associated with depressed or absent reflex bilaterally. It disappears by 3–6 months in normal infants.
- **Stepping reflex:** When the foot touches a flat surface, the infant makes a stepping motion by bringing one foot in front of the other.
- **Palmar grasp reflex:** The infant’s palm closes around the object that is placed into its hand. As the early grasp reflex disappears, they begin to hold objects in both hands. This reflex is present at 28 weeks of gestation, is strong by 32 weeks and persists until 4–6 months of age.
- **Fencing posture or asymmetric tonic neck reflex:** It is not present at birth. When the infants head is rotated to one side, the arm on that side straightens and the opposite arm flexes. It prepares the child for hand-eye co-ordination and reaching out to objects. It disappears by 4–6 months as the infant begins to roll over.
  
  As the higher cortical center develops, the primitive reflexes are replaced by the postural reflexes that enable the child to maintain a stable posture. Children who have suffered neuronal damage exhibit delayed postural reactions and problems with coordination and motor development.
- **Parachute reflex:** It develops at around 8–9 months. When the prone infant is lowered suddenly, the arms fling out in a protective manner.
- **Protective equilibrium response:** It is seen at 4–6 months. A sitting infant who is suddenly jerked laterally, extends the arm on the opposite side and flexes the trunk towards the side of the force to regain the center of gravity.

**UPPER AIRWAY**

**Head**

Infants have a large occiput. Placing a pillow under the occiput as in adults will flex the head on the neck instead of extending it for the sniffing position. Placing a small roll beneath the shoulders and a small ring beneath the occiput ensures that the airway is free. The large head is difficult to stabilize during induction of anesthesia as it tends to fall on the chest causing airway obstruction. Any condition which increases the head circumference like hydrocephalus aggravates this problem. Laryngoscopy in the extended head position swings the anteriorly placed larynx even more anterior as one lifts the laryngoscope blade, making visualization more difficult. It is better to keep the head in neutral position for intubation.

**Nares**

Although obligate nasal breathers, most neonates convert to combined nasal and oral breathing by 5 months of age. Most neonates and infants can resort to oral breathing later.
if obstruction lasts longer than 15 sec. Nasal resistance may contribute up to 50% of total airway resistance and respiration may be hampered in the presence of nasal secretions or a nasogastric tube. Choanal atresia would give similar problem and an oral airway needs to be inserted to ease breathing. Owing to the more cephalad placed larynx, the epiglottis approximates the soft palate and hampers oral breathing. The nares have to be patent so that the infant can breathe while sucking and swallowing.

**Tongue**

The tongue is large in comparison to the small oral cavity and is more difficult to manipulate and stabilize with a laryngoscope blade. A straight laryngoscope blade more effectively lifts the tongue from the field of view and facilitates visualization of the larynx. Since, the larynx is more cephalad, distances between the tongue, hyoid, epiglottis and roof of the mouth are smaller than in the older child or adult. With growth, the oral cavity, pharynx and the mandible enlarge, the larynx descends from C2 to C4 and the tongue begins to occupy a more anterior position. In patients with mandibular and midfacial hypoplasia, the base of the tongue is positioned in closer proximity to the laryngeal inlet than normal. In these patients owing to greater acute angulation between the plane of the tongue and the plane of the larynx, the esophageal rather than the laryngeal inlet is visualized during laryngoscopy.

**Pharynx**

The pharynx is almost completely made of soft tissues and easily collapsed by posterior displacement of mandible or external compression of hyoid. Tonsils are small in the newborn but grow to maximal size at 4–7 years. They can make mask ventilation difficult and cause obstruction during spontaneous ventilation.

**Epiglottis**

Epiglottis is narrow, tubular, omega shaped, more vertical and angles over the laryngeal inlet making glottis visualization difficult. Epiglottis locks itself with soft palate thus making a free passage of air from nose to nasopharynx to larynx making newborns obligatory nose breathers for first few months. This high position of larynx and interlocking of soft palate with epiglottis allows infants drink and breathe at the same time. Respiratory obstruction during induction of anesthesia is more because of floppy and long epiglottis folding over the glottis or flexion of large head over chest rather than tongue fall.

**Larynx**

The major differences between the pediatric and the adult larynx are size, shape and position in the neck. Development of the larynx begins at approximately 21 days and the epiglottis at 30 to 32 days. By the end of the 2nd trimester laryngeal epithelium changes from primitive...
mesenchyme into cuboidal and stratified squamous epithelium. The fetal larynx is positioned high in the neck, usually at the level of 2nd or 3rd cervical vertebra. In the preterm infant, the larynx is located at the middle of the third cervical vertebra, in a full-term infant at the C3-4 interspace and in the adult at the C4-5 interspace. At birth it is about 2 cm long with dimensions of 7 mm anteroposterior and 4 mm laterally. The subglottis which is the narrowest portion of the airway in newborns has a diameter of approximately 4 mm. The thyroid cartilage of infants is relatively shorter and broader than in adults, lies closer to the hyoid and the laryngeal prominence and notch are not well-developed. The cricoid ring in an infant is elliptical, not circular being of larger diameter in the anteroposterior dimension.

The infant’s vocal cords are 4.0–4.5 mm long, relatively shorter than those of the child or adult. They have a lower (caudal) attachment anteriorly than posteriorly, whereas in an adult, the axis of the vocal cords is perpendicular to the trachea. One may encounter difficulty during nasal intubation as the endotracheal tube tends to lodge in the anterior commissure rather than slide into the trachea. Usually by about the 3rd year of life boys develop a longer and larger internal larynx while the angle of the thyroid laminae becomes greater in girls. At puberty these changes are accentuated as the size of the male larynx increases more rapidly. The angle of the thyroid laminae develops to about 90° in males and to about 120° in females, the difference being responsible for the laryngeal prominence in males.

LOWER AIRWAY

In a child, the trachea is smaller, more deeply placed and more movable than in the adult. The length of the trachea in full-term neonates is 4 cm and increases to 12 cm in adults. Because of the shorter length of the trachea endobronchial intubation and accidental extubation are more common with head and neck movement. At birth the trachea is smaller in diameter with immature tracheal rings. Flexible cartilaginous rings of the trachea can predispose to dynamic obstruction with negative pressure ventilation especially when any partial airway obstruction exists. Normally there are 16 to 18 tracheal cartilages between the cricoid and carina. In neonates, the trachea is three times more compliant than that of the infant and six times more compliant than that of an adult. Complete or partial occlusion of the airway can occur with hyperextension or hyper flexion of neck. With growth, the adult configuration emerges and the tracheal lumen changes from the cylindrical to the more adult shaped ovoid form. The bifurcation is at a higher level until 10–12, years. Since the main stem bronchi are at lesser angle than adults, aspiration can occur on either side. As the child grows, increase in chest diameter causes angle of left bronchus to increase.

Resistance to airflow is inversely related to the fourth power of the radius during quiet breathing (when airflow is laminar) but is inversely related to the fifth power of the radius when airflow is turbulent, e.g. in a crying child. When respiratory distress is present the child should be kept as calm as possible so as to reduce turbulent flow and airway resistance. Because the lumen of the pediatric trachea is small, relatively small compromise in tracheal radius due to edema can significantly increase resistance to airflow and work of breathing. A long endotracheal tube (ETT) of small diameter or an obstructed ETT increase work of breathing.

In tracheomalacia the cartilaginous rings of the trachea or bronchi are soft and pliable or have abnormal configuration that results in collapse and occlusion of the tracheal lumen. In neonates with tracheoesophageal fistula, there may be a tracheomalacic segment in the vicinity of the fistula. Tracheostomy should be performed keeping in mind that the larynx is higher. If dissection is done low in the neck there is a risk of injury to the innominate vein which can rise into the jugular notch and to the domes of the pleura which are paratracheal in the neck of children. The neonatal cricothyroid membrane is small measuring 3 mm wide and 2.6 mm tall. In infants the hyoid bone may overlap the usually prominent thyroid cartilage and make identification of anatomy difficult. It has been suggested that in these young patients the preferred approach may be direct puncture of the trachea below the level of the cricoid.

Alveoli are fewer in number in infants, and continue to mature and increase in number till first decade of life. This results in small lung volume. The oxygen demand in infants is high due to high metabolic rate. There is tendency to desaturate fast due to less reserve. In neonates and small infants, alveoli tend to collapse even during tidal ventilation, therefore it is advisable to keep small infants on controlled ventilation during anesthesia.

SMALL AIRWAYS, LARGE CONTROVERSIES

Conventional teaching has been that the vocal cords in adults and the cricoid cartilage in children is the narrowest part of the airway. Videobronchoscopic studies have now shown that the narrowest portion of the pediatric airway is at the level of vocal cords or the subglottic region and not the cricoid. Laryngeal dimensions using computerized software demonstrated that the cross-sectional area at the cricoid (48.9±15.5 m²) was larger than that at the glottis.
(30±16.5 m²). The narrowest portion of the airway in about 70% adults is also at the level of the cricoid cartilage but difficulty in passing the ET is not encountered as the opening is proportionately large.

It was thought that hearing a leak with an uncuffed ETT ensures that an appropriate size has been chosen. However due to the elliptical shape of the airway, it is possible to demonstrate a leak and yet have significant pressure on the lateral walls with the use of an ETT that is circular in cross-section.

Uncuffed tubes have always been preferred in children younger than 6 years so as to avoid trauma due to cuff inflation. With the advent of Microcuff ETTs, anesthetists are more comfortable using cuffed tubes in children especially in laparoscopic and video-assisted thoracoscopy surgeries. The cuff in these tubes is located more distally compared to the standard tubes and is well beyond the cricoid cartilage. Also the polyurethane cuff of these tubes is of a softer material and inflates symmetrically resulting in a more uniformly distributed pressure on the tracheal mucosa.

While the Miller blade was thought to provide better intubating conditions in children up to 2 years of age, recent studies claim that both Miller and Macintosh blades provide similar laryngoscopic views and intubating conditions.

**CHEST WALL**

The chest wall of the neonate is highly compliant, floppy and moves inwards with inspiration. There may be inward displacement of the lower edge of the rib cage in infants during sleep even in the absence of airway obstruction. This is due to poorly developed musculature and incompletely calcified ribs that have greater amount of cartilage. The outward recoil of the thorax is less as compared to adults while the elasticity of the lung is only slightly less.

Chest wall collapse during inspiration is prevented by the accessory muscles and functional residual capacity (FRC) is maintained. Younger children have lesser developed accessory muscles making it difficult to increase strength and depth of ventilation. Higher closing volume in neonates also contributes to their tendency to desaturate rapidly. By 6 months of age chest wall compliance is closer to adult values, although anesthesia related changes in FRC are still marked in children up to age of 12.

In infancy ribs extend horizontally from the vertebral column moving little with inspiration and there is increased workload on the diaphragm to maintain the tidal volume. After assuming an upright posture, the child gradually acquires the caudal slant and downward rotation of ribs that are characteristic of adults. Respiratory failure is also more common in neonates and infants due to the difference in constitution of the diaphragm. Type 1 slow twitch, high oxidative capacity muscle fibers which are fatigue resistant make up less than 10% of the total before 37 weeks of gestational age as compared to 50% in adults. Since, the diaphragm is the most significant respiratory muscle in infancy, it is important to decompress the stomach to stop it from impinging on diaphragmatic excursion. Any condition causing distension of the abdomen will prevent the diaphragm from descending during respiration and will hamper breathing.

Rib fractures are less common as compared to adults due to their pliable nature and greater elasticity of the chest wall. Injury to the underlying structures should be suspected even in the absence of rib fractures since the force may be easily transmitted through the cartilaginous ribs. The mediastinal structures show increased mobility as compared to adults, so a tension pneumothorax is more likely to shift the mediastinum and hamper contralateral lung ventilation. Due to the thin chest wall respiratory sounds are conducted over a wide area making it difficult to localize adventitious sounds on auscultation.

**CENTRAL NERVOUS SYSTEM**

The neonatal brain weighs about 1/10th of body weight compared with about 1/50th of the body weight in an adult. 70% of the adult brain weight is achieved at 18 months, 80% at 3 years, 90% at 5–8 years and approximately 95% at the tenth year. At birth about one-fourth of neuronal cells are present. The development of cells in the cortex and brain stem is nearly complete by one year of age. Myelination and elaboration of dentritic processes continue well into the 3rd year and myelination is completed by the 12th year of life. Normal values for Intracranial pressure (ICP) are generally accepted as less than 15 mm Hg. In full-term neonates, normal ICP is 2–6 mm Hg and is probably lower in preterm infants.

The skull is less rigid in infants than in adults. As a result an increase in the volume of its contents can be accommodated to some extent by expansion of the fontanelles and separation of the suture lines. However, herniation is a possibility even with open fontanelles when acute large increases in ICP occur. ICP may remain normal despite a significant intracranial pathologic process and increasing head circumference may be the first clinical sign. A higher ratio of brain content to intracranial capacity places children at higher risk of herniation than adults. In adults cerebral blood flow (CBF) is approximately 55 mL/100 g of brain tissue/min, i.e. about 15% of the cardiac output. CBF in children is approx 100 mL/100
gram of brain tissue/min, i.e. about 25% of cardiac output. CBF in neonates and preterm infants is less as compared to children and adults, i.e. approximately 40 mL/100 g/min. Global cerebral metabolic rate (CMR) for oxygen and glucose is higher in children than in adults (oxygen, 5.8 versus 3.5 mL/100 g brain tissue per minute and glucose, 6.8 versus 5.5 mL/100 g brain tissue per minute respectively).

Neonates are vulnerable to cerebral ischemia and intraventricular hemorrhage because of their narrow autoregulatory range. As a result of a linear correlation between CBF and systemic blood pressure neonates are vulnerable to both cerebral ischemia during hypotension and intraventricular hemorrhage with increased blood pressure. One has to be cautious while providing deliberate hypotension or performing awake intubations and suctioning in infants.

**THE SPINAL CORD**

The spinal cord in the embryo extends the entire length of the vertebral column. At birth the dura mater ends at the level of the third or fourth sacral vertebra and the conus medullaris at the L3 vertebral level. However, the growth of the vertebrae exceeds that of the cord and at the end of the first year of life the adult level is attained, i.e. S1 for the dural sac and L1 for the conus medullaris. Although, the dura mater and arachnoid mater usually end at the S1 vertebra in adults, the pia mater does not. Distal to the caudal end of the spinal cord, the pia mater forms a long fibrous thread, the filum terminale which extends from the conus medullaris and attaches to the periosteum of the 1st coccygeal vertebrae. Injury to the spinal cord is usually prevented by performing the lumbar puncture at L4-L5 interspace and avoiding epidural approaches above L3 whenever possible.

Unlike adults who exhibit 2 lordotic and 2 kyphotic curves, there is a single spinal flexure in neonates. Cervical lordosis develops with head holding at 3–6 months followed by lumbar lordosis at 8–9 months after the child assumes the sitting position.

Since, the spinous processes are parallel the orientation of the epidural needle does not vary and remains the same irrespective of the intervertebral space chosen.

In adults, the thoracic kyphosis limits cephalad spread of the drug. As it is not well-developed in newborns and infants higher levels may be achieved and hence care should be taken while elevating the limbs immediately after spinal injection.

The vertebrae of neonates being mostly cartilaginous and poorly calcified are prone to damage during regional anesthesia. Bone growth could be hampered when the advancing needle hits the ossification nuclei. Therefore the paramedian approach where the needle is walked off the laminae is to be avoided and the midline approach is preferred.

Newborns have a narrow subarachnoid space (6–8 mm) and greater precision is needed while locating it. The loss of resistance feel during spinal anesthesia is less marked as the ligaments are less densely packed.

Children have a smaller pelvis compared to adults and the sacrum is placed more cephalad relative to the iliac crests. Intercristal line or Tuffiers line is at L3–L4 in adults while it passes through L5–S1 in neonates and L4–L5 in infants.

The highly vascular piamater combined with high cardiac output is responsible for rapid reabsorption of the local anesthetic and shorter duration of block. However, the onset of drug action is faster as the loose endoneurium offers little barrier to drug diffusion.

The head should remain extended so as to avoid airway compromise when the infant is positioned lateral during the block. The CSF hydrostatic pressure is lower in infants in the recumbent posture and the spinal needle should be progressed slowly to detect the backflow of CSF. There are greater chances of obtaining a good CSF flow with the infant in the sitting position due to increased hydrostatic pressure. The normal estimated pressure in the recumbent posture is 100–200 cm H$_2$O and pressure is higher in the erect position.

The volume of CSF varies from more than 10 mL/kg in neonates to 4 mL/kg in infants weighing less than 15 kg, 3 mL/kg in children and 1.5 to 2.0 mL/kg in adolescents and adults. In children half the CSF volume is located within the spinal subarachnoid space as compared to only...
one-fourth in adults and so the local anesthetics injected intrathecally are quickly diluted by the CSF. On a weight basis, the drug dosage in neonates needed to achieve similar dermatome levels as adults is about 10-fold higher. However due to the greater volume of CSF per kg and rapid turnover of CSF, the duration lasts only about one-third to one half as long as in the adult.

THE EPIDURAL SPACE

Epidural space in children is divided into the cervical, thoracic, lumbar and sacral. The epidural space is superficial, the ligamentum flavum is much thinner and less dense than in adults due to which the penetration feel is subtle. Risk of unintentional dural puncture is greater during placement of epidural catheter. Whereas the epidural space in an adult is characterized by densely packed fat lobules and fibrous strands, the epidural fat of neonates and young children is spongy in nature with discrete spaces between the epidural fat lobules. The distance from the skin to the epidural space in children aged 6 months to 10 years is approximately 1 mm/kg body weight.

In infants, the spinal cord can be in close proximity to the vertebral column in the sitting position as it moves backwards. In the lateral flexed position, the spinal cord tends to move forward and away from the ligamentum flavum making it easier to thread the catheter into the expanded epidural space. Myelination begins prenatally at 30 weeks of gestation. Delayed myelination of nerve fibers and increased distance between the nodes of Ranvier permits easier intraneural penetration of local anesthetics. Hence, their onset time is shortened and diluted local anesthetic is as effective as more concentrated drug in adults. Also the local anesthetic solution spreads over an extended area due to the loose attachment of the perineural vascular sheath. However comparatively large volumes of epidural local anesthetic are needed in children up to 6–7 years of age. This can be attributed to the fluid nature of epidural fat and loose attachment of the sheaths surrounding the spinal roots which favor consistent leakage of LA injected within the epidural space. Due to the narrow epidural spaces there is sometimes a backflow of the drug when injected rapidly. However, this leak around the epidural catheter may be visible even with slow injection. Thoracic epidurals are not commonly used due to risk of spinal cord damage. The lumbar and thoracic epidural spaces are compact as compared to the caudal. This permits use of smaller drug volumes when the catheter tip is close to the site of incision. The epidural space can also be approached via the S2–S3 interspace which is 0.5–1 cm below the line joining the two posterior superior iliac spines.

THE CAUDAL CANAL

The sacrum in children is narrower and flatter than in adults. Owing to absence of fat over the sacrum in children the anatomy is well-delineated. The five sacral vertebrae which are identifiable until one year fuse by 2–6 years of age as the child stands and walks. The sacral hiatus is a U or V-shaped opening that occurs as a lack of dorsal fusion of the fifth and sometimes the fourth sacral vertebral arches. The hiatus forms the apex of an equilateral triangle and the base is formed by the line joining the two posterior superior iliac spines. The hiatus is covered by the sacrococcygeal membrane which is the sacral continuation of ligament flavum and is bounded laterally by the sacral cornua. The sacral canal which is a continuation of the lumbar spinal canal is triangular in shape. It contains the cauda equina, the spinal meninges, adipose tissue and the sacral epidural venous plexus which generally terminates at S4. Most of the vessels are concentrated in the anterolateral portion of the canal. Since the caudal space communicates with the perineural spaces a lower concentration of local anesthetic is effective. The loosely packed fat in the epidural space permits easy passage of the catheter. Spread of the drug is more predictable in children although a higher volume/kg as compared to adults is needed to fill the loosely packed space. Distance from the skin to the caudal space in neonates is minimal. There are high chances of entering the dural sac which may extend to S3. Needles 25 mm are long enough to reach sacral epidural space and short enough to prevent dural puncture in most patients. The distance separating the summit of the sacral hiatus and the dural sac ending is 30±10.4 mm (range 13.6 to 54.7 mm) in children 10 months to 18 year. Mean distance from skin to anterior sacral wall is 21 mm (extremes 10 to 39 mm) between 2 months and 7 year of age. At around 7 year, the child’s caudal space begins to become more angulated and difficult to enter. As the child grows and vertebrae begin to ossify midline ultrasound imaging becomes difficult.

HEART

The heart occupies much of the thoracic cavity. It weighs approximately 20 grams at birth. As the body weight doubles during the first year of life, weight of the heart increases by 80% and by puberty it is about 0.5% of the body weight. In the full-term neonate, the right and the left ventricles are symmetrical cone-like structures with both chambers circular in cross-section. At birth and thereafter numerous changes occur as the fetal circulation transits to adult type. The left ventricle (LV) increases in size due to enhanced afterload. The right ventricle (RV) works against

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a much lower afterload and lags behind in weight and size. The adult ratio of about 3:1 for LV:RV weight is attained by 3 months. The neonatal period is characterized by significant development of the LV and a shift from RV to LV predominance. The fetus and neonate have the highest cardiac output with respect to weight, i.e. about 400 mL/kg/min. The ratio of RV to LV output is 1.3:1.  

The newborn myofibrils have a chaotic and scattered arrangement with nuclei and mitochondria in the center of the cell. In the adult, the myofibrils are arranged in parallel with alternate rows of mitochondria and sarcoplasmic reticulum. This variation in mitochondrial arrangement is responsible for the difference in contractile function of the myocardium. Intracellular release of calcium is responsible for contraction of adult myocardium whereas flux of calcium from outside to the inside across the sarcolemma causes contraction of the immature myocardium. Sarcomere volume is only 30% of newborn myocyte compared with 60% in the adult. However, the immature myocardium has higher activity of Na⁺-Ca²⁺ exchanger which can bring calcium either into or out of the cell. As a consequence of the underdeveloped sarcoplasmic reticulum and T tubules the newborn heart has decreased contractile reserve and reduced compliance. They are more dependent on normal cytosolic ionized calcium levels and tolerate hypocalcemia poorly. They are more sensitive to calcium channel blockers, potent inhalational agents with calcium channel blocking activity and rapid blood transfusions where serum calcium concentration can decrease causing myocardial dysfunction. The immature myocardium utilizes carbohydrate as the primary energy source. As the mitochondria mature, long chain fatty acids take over as the energy source.

There is increased activity of the Na⁺ - H⁺ exchanger in the immature myocardium and this has been implicated as a factor in the greater resistance of the immature myocardium to acidosis. The high collagen content may be responsible for the relative noncompliance of the neonatal heart and its limited capacity to respond to volume loading. This nondistensible heart has limited capacity to increase stroke volume and augment cardiac output (CO) when faced with an increased preload. They are more sensitive to increase in afterload as compared to adults. The heart rate is the primary means of increasing CO in the newborn. Since, the normal heart rate is high, increasing the rate has limited effect on CO but decrease in the heart rate dramatically decreases output.

**NEUROMUSCULAR JUNCTION**

It is incompletely developed at birth. Synaptic transmission is slow and the rate at which acetylcholine (ACh) is released during repeated nerve stimuli is limited in the infant. The immature or fetal ACh receptor subtypes differ in the structure of one subunit from the adult subtype receptor. Neonates have a mix of both adult and fetal receptors but at term the adult subtypes are more common. Muscle membranes of older infants, children and adults also have a small proportion of extrajunctional acetylcholine receptors; the production of which ceases with nerve activity. Mature receptors are localized to the end plate region and are metabolically stable with a half life of 2 weeks. Fetal receptors are metabolically unstable with a half life of 24 hour and present at junctional and extrajunctional sites. Neonates and infants require higher dose of succinylcholine as compared to older children whereas they are more sensitive to non-depolarizing muscle relaxants.

**ABDOMEN**

As compared to adults, the abdominal wall in children is thin with less muscle and subcutaneous tissue. The abdominal cavity is relatively small and compact. The diaphragm which is flatter and less dome-shaped tends to push the large organs like the liver and spleen below the rib cage making them more susceptible to injury. Since, the pelvis is shallow, the bladder is an intra-abdominal organ and could be damaged in cases of abdominal trauma. The kidneys are placed lower in the abdomen and
are large relative to body size. Development of the renal system begins by the 3rd week, the tubules and collecting system are functional with formation of urine by 10 weeks and majority of nephrons are formed by 36 weeks of gestation. Hyperplasia continues for about 6 months of postnatal life; thereafter increase in renal size is due to cellular hypertrophy. Between ages of 6 months and 1 year, normalized renal plasma flow is half that of an adult but increases progressively to reach adult levels at about 3 years of age. The liver and the biliary tree begin to develop during the late third to early 4th week and accounts for 10% of the fetal weight by 9 weeks of gestation. At birth it is about 4% of the body weight.

**GASTROINTESTINAL SYSTEM**

Swallowing reflex is seen in the fetus at 10–11 weeks followed by suckling at 18–24 weeks and breathing at 32–37 weeks of gestation. The ability to coordinate swallowing with respiration does not fully mature until infants are 4–5 months of age resulting in a high incidence of gastroesophageal reflux in newborns. Lower esophageal sphincter (LES) pressures are diminished and take 3–6 weeks to achieve adult levels. Peristaltic waves seen in the lower esophagus in adults are absent in infants. At birth gastric pH is alkalotic; it attains the normal range for the lower esophagus in adults are absent in infants. At 37 weeks to achieve adult levels. Peristaltic waves seen in the lower esophagus in adults are absent in infants. At birth gastric pH is alkalotic; it attains the normal range for older children. Premature infants born after 36 weeks to achieve adult levels. Peristaltic waves seen in the lower esophagus in adults are absent in infants. At birth gastric pH is alkalotic; it attains the normal range for older children. Premature infants born after 36

REFERENCE

Physiological Characteristics and Anesthetic Implications

INTRODUCTION
Growth and development are the most clinically relevant features differentiating children from adults. Although, anatomical differences are quite obvious, the pediatric anesthesiologist needs to be more familiar with the various physiological and functional changes that occur from newborn period to infancy to childhood and adolescence. This chapter provides a brief review about the developmental physiological aspects of pediatrics and its implications to pediatric anesthesia practice.

Children can be classified into different groups depending on their age as follows:

- Newborns: First 24 hours
- Neonates: 1–28 days
- Infants: Up to 1 year
- Small children: 2–5 years
- School-aged children: 6–14 years
- Adolescents: 14–18 years

Amongst the above age groups neonates require a special mention especially preterm neonates, because they are more prone for complications related to prematurity.

PREMATURITY
Neonates born before 37 weeks of gestation are referred to as preterm neonates. Preterm babies can be further subdivided into mutually nonexclusive subgroups as:

1. Gestational age between 35–37 weeks—Late preterm
2. Gestational age between 30–34 weeks—Moderately preterm
3. Gestational age between 27–29 weeks—Very preterm
4. Gestational age less than 26 weeks—Extremely preterm.

Preterm infants have various physiological derangements which necessitate an understanding of preterm neonatal physiology and its subsequent effects on the developing immature organ systems. Table 1 provides a brief summary of problems encountered in preterms when they present for anesthesia.

Infants can also be classified based on their gestational age and weight into three categories. Appropriate for gestational age (AGA) babies are newborn infants with birth weight ranging between 10th and 90th percentile on a fetal growth chart. Small for gestational age (SGA) babies are the ones with weight less than 10th percentile because of intrauterine growth retardation. Large for gestational


### Table 1: Common medical issues in preterms

<table>
<thead>
<tr>
<th>System</th>
<th>35–37 weeks</th>
<th>30–34 weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory system</td>
<td>Apnea, respiratory distress syndrome (RDS)</td>
<td>Apnea, RDS, bronchopulmonary dysplasia (BPD)</td>
</tr>
<tr>
<td>Cardiovascular system</td>
<td>-</td>
<td>Patent ductus arteriosus (PDA), anemia</td>
</tr>
<tr>
<td>Gastrointestinal system</td>
<td>Feeding intolerance, hyperbilirubinemia</td>
<td>Feeding intolerance, necrotizing enterocolitis (NEC)</td>
</tr>
<tr>
<td>Central nervous system</td>
<td></td>
<td>Intracranial hemorrhage (ICH)</td>
</tr>
<tr>
<td>Metabolic</td>
<td></td>
<td>Hypoglycemia, hypocalcemia</td>
</tr>
<tr>
<td>Others</td>
<td>Sepsis</td>
<td>Temperature instability, sepsis</td>
</tr>
</tbody>
</table>

### Table 2: Physical examination to determine gestational age

<table>
<thead>
<tr>
<th>Physical examination</th>
<th>Preterm (&lt;37 weeks)</th>
<th>Term (&gt;37 weeks)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ear</td>
<td>Shapeless, pliable</td>
<td>Firm, well formed</td>
</tr>
<tr>
<td>Skin</td>
<td>Edematous, thin skin</td>
<td>Thick skin</td>
</tr>
<tr>
<td>Soles of feet</td>
<td>Creases on anterior third</td>
<td>Whole foot creased</td>
</tr>
<tr>
<td>Breast tissue</td>
<td>Less than 1 mm diameter</td>
<td>More than 5 mm diameter</td>
</tr>
<tr>
<td>Limbs</td>
<td>Hypotonic</td>
<td>Tonic</td>
</tr>
<tr>
<td>Grasp reflex</td>
<td>Weak</td>
<td>Can be lifted by grasp reflex</td>
</tr>
<tr>
<td>Moro reflex</td>
<td>Complete but exhaustible</td>
<td>Complete</td>
</tr>
<tr>
<td>Sucking reflex</td>
<td>Weak</td>
<td>Strong and synchronous</td>
</tr>
<tr>
<td>Genitalia</td>
<td>Males: scrotum poorly developed with undescended testis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Females: large clitoris with gaping labia majora</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Males: rugated scrotum with descended testis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Females: well developed labia majora</td>
<td></td>
</tr>
</tbody>
</table>

### Table 3: Frequently encountered problems in small and large for gestational age babies

<table>
<thead>
<tr>
<th>SGA</th>
<th>LGA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congenital anomalies</td>
<td>Birth injury</td>
</tr>
<tr>
<td>Chromosomal abnormalities</td>
<td>Asphyxia</td>
</tr>
<tr>
<td>Chronic intrauterine infections</td>
<td>Meconium aspiration</td>
</tr>
<tr>
<td>Heat loss</td>
<td>Metabolic abnormalities (hypoglycemia, hypocalcemia)</td>
</tr>
<tr>
<td>Asphyxia</td>
<td>Polycythemia</td>
</tr>
<tr>
<td>Metabolic abnormalities (hypoglycemia, hypocalcemia)</td>
<td>Hyperbilirubinemia</td>
</tr>
<tr>
<td>Polycythemia</td>
<td>Hyperbilirubinemia</td>
</tr>
</tbody>
</table>

**GENERAL GROWTH AND DEVELOPMENT PATTERNS**

Growth denotes a net increase in size or mass of tissues and development refers to maturation of its function. Different tissues of the body grow at different rates (Fig. 1). Order of growth in human beings is cephalocaudal and from distal to proximal. Thus, during fetal life growth of head occurs first, later neck and arms and lastly legs. Distal body parts like the hands increase in size before the proximal ones like the arms develop. Head control develops early, followed by coordination of spinal muscles and use of hands. Creeping and crawling which involve lower limb musculature develop later.²

**Weight**

Weight is a measurement of growth and is a sensitive index of growth and any associated illness. The average weight of a newborn is 3 kilograms. In the first few days after birth, the neonate loses extracellular fluid upto 10% of body weight.
which is subsequently regained by day ten of life. For the first 3 months, infant gains 25–30 g/day; subsequently 400 grams/month till the end of the first year (Table 4).

**Implication:** Percentile charts are more reliable than single recordings of weight to monitor the child's growth and development. Children with features of failure to thrive need to be thoroughly investigated for the underlying medical disorder and optimized well before anesthetising them.¹

### Height
- Birth: 50 cm
- Three months: 60 cm
- Nine months: 70 cm
- One year: 75 cm
- Two years: 90 cm.

### Head Circumference (HC)
Head growth is rapid during first half of infancy.
- Birth: 33–35 cm
- 3 months: 2 cm/month
- 3–6 months: 1 cm/month
- 6–12 months: 0.5 cm/month

**Implication:** Abnormal head size should be evaluated for either normal/familial/pathological conditions.¹

### Chest Circumference (CC)
- At birth: 3 cm less than HC
- At 1 year HC = CC
- After 1 year CC >HC.

### Eruption of Teeth
**Primary teeth:** The mandibular teeth erupt earlier than the maxillary counterparts, the central incisors appear by 6–7 months of age. The primary teeth eruption sequence is complete by 2 years of age and totals 20 teeth.

**Permanent teeth**
- 1st molar: 6–7 years
  - Central and lateral incisors: 6–8 years
- Canine and premolars: 9–12 years
- 2nd molar: 12 years
- 3rd molar: 18 years and above.

**Implication:** Airway assessment should include looking for the presence of any loose teeth especially in the age group of 5–10 years of age.³

### Body Surface Area (BSA), Basal Metabolic Rate and Caloric Requirement
BSA is recommended as the principal basis for drug dosage calculation in children. At birth, BSA is 0.2 m², in adults, it is 1.75 m². Caloric needs in relation to BSA in an infant is 30 kcal/m²/h, at two years of age 50 kcal/m²/h and then gradually reaches adult values of 35–40 kcal/m²/h.

### DEVELOPMENTAL PHYSIOLOGY

#### Cardiovascular System
In the 4th week of gestation, a pair of angioblastic cords develop from the mesoderm to form a pair of endocardial tubes which results in the formation of a primitive heart tube. Fetal heart activity can be detected by day 22 of gestation. From the fifth to eighth week the primitive tube is further transformed to a four chambered organ. Blood cell production on day 18 switches from yolk sac to the liver, spleen, thymus and finally the bone marrow.

In fetus, gas exchange occurs in the placenta bypassing the fetal lungs. Various intracardiac and extracardiac shunts ensure that well oxygenated blood is diverted to organs with higher metabolic demands like the brain and heart. Despite the relatively hypoxic environment in utero, the fetus develops and thrives because of compensatory mechanisms like presence of high levels of HbF with high oxygen affinity, low levels of 2, 3 diphosphoglycerate, and increased cardiac output with greater blood volume.

At birth, with the placenta removed, the neonate’s lungs take over as organs of oxygenation which impose a severe stress on the fragile developing cardiorespiratory system. Transitional circulation is used to describe the changes observed in the fetus as it adjusts to circulatory and respiratory changes and establishes neonatal circulation. The neonatal circulatory pattern is described as “series” circuit of pumps and resistance beds, whereas the fetal circulation is likened to a “parallel” circuit.

The pulmonary vascular resistance drastically decreases after birth, the foramen ovale closes functionally...
with increasing levels of oxygen in blood. The neonatal circulation in the face of hypoxemia and acidosis can revert back to fetal circulation.

Cardiac output increases with increasing birth weight. The resting cardiac output is 2–3 times that of adult values to meet the metabolic and oxygen demands of infancy and childhood. Autonomic control of heart rate is immature and predominantly parasympathetic in nature.\(^1,3,4\)

**Renal System**

Nephrogenesis though complete is immature in newborns. Renal function is markedly diminished because of low perfusion pressure and immaturity of both glomerular and tubular function. Adult values are gradually attained by two years of age. Concentrating and diluting capacity is low in neonates and are sensitive to fluid and solute loads.\(^1,3\)

**Hepatic System**

The fetal and neonatal liver is capable of storing glycogen, forming plasma proteins and is involved in controlling carbohydrate, protein and lipid metabolism. Hematopoiesis occurs in liver peaking at 7 months of gestation.

Hepatic function remains immature because of low hepatic blood flow, immature enzyme systems and low concentration of serum proteins like albumin, prothrombin and carrier proteins. Adult values are attained by one year of age.\(^1,3,4\)

**Gastrointestinal Tract**

Maturation of the gastrointestinal tract occurs from the proximal to the distal end. The foregut and the hindgut develop by 4–5 weeks of gestation. The midgut elongates and herniates into the umbilical cord by 7–12 weeks of gestation. By the 12th week, the abdominal cavity is large enough for the developing gut to exit the cord and re-enter the abdomen.

Various anomalies arising from maldevelopment include tracheoesophageal fistula, intestinal atresia, duplication, bands, omphalocele, gastroschisis and Hirschspring’s disease. Pharyngoesophageal sphincter and the lower esophageal sphincter is immature resulting in frequent regurgitation and gastroesophageal reflux.\(^1,3\)

**Neurological Development**

By 3–4 weeks of gestation the primary tube is developed followed by development of prosencephalon. Neuronal proliferation then occurs from ventricular to subventricular region by 2–3 months. From the 5th month onwards glial development occurs which continues up to second year of life. Myelination continues into the third year of life. Nociceptive pathways with development of sensory receptors occur by 7 weeks of gestation and by 20th week with the formation of thalamocortical connections the fetus can probably perceive pain. The response to pain in neonates is integrated with somatic, neuroendocrine and autonomic pathways.

Developmental milestones represent the average age at which children accomplish various tasks which indicate maturation of different biological functions. The Denver Development Screening Test assesses child’s development in four separate segments—gross motor, fine motor and adaptive, social and language functions. The summary of important milestones is presented in Tables 5 to 8.\(^1,3\)

**Table 5:** Key developmental milestones—gross motor

<table>
<thead>
<tr>
<th>Age</th>
<th>Milestone</th>
</tr>
</thead>
<tbody>
<tr>
<td>3 months</td>
<td>Neck holding</td>
</tr>
<tr>
<td>5 months</td>
<td>Sits with support</td>
</tr>
<tr>
<td>8 months</td>
<td>Sits without support</td>
</tr>
<tr>
<td>9 months</td>
<td>Standing with support</td>
</tr>
<tr>
<td>10 months</td>
<td>Walking with support</td>
</tr>
<tr>
<td>11 months</td>
<td>Crawling/creeping</td>
</tr>
<tr>
<td>12 months</td>
<td>Standing without support</td>
</tr>
<tr>
<td>13 months</td>
<td>Walking without support</td>
</tr>
<tr>
<td>18 months</td>
<td>Running</td>
</tr>
<tr>
<td>24 months</td>
<td>Walking upstairs</td>
</tr>
<tr>
<td>36 months</td>
<td>Riding tricycle</td>
</tr>
</tbody>
</table>

**Table 6:** Key developmental milestones—fine motor

<table>
<thead>
<tr>
<th>Age</th>
<th>Milestone</th>
</tr>
</thead>
<tbody>
<tr>
<td>4 months</td>
<td>Grasps a rattle</td>
</tr>
<tr>
<td>5 months</td>
<td>Reaches for objects with bidextrous grasp</td>
</tr>
<tr>
<td>7 months</td>
<td>Palmar grasp</td>
</tr>
<tr>
<td>9 months</td>
<td>Pincer grasp</td>
</tr>
<tr>
<td>2 years</td>
<td>Imitates vertical line</td>
</tr>
<tr>
<td>3 years</td>
<td>Copies circle</td>
</tr>
</tbody>
</table>

**Table 7:** Key developmental milestones—language

<table>
<thead>
<tr>
<th>Age</th>
<th>Milestone</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 month</td>
<td>Turns head to sound</td>
</tr>
<tr>
<td>3 months</td>
<td>Cooing</td>
</tr>
<tr>
<td>6 months</td>
<td>Monosyllables</td>
</tr>
<tr>
<td>9 months</td>
<td>Bi-syllables</td>
</tr>
<tr>
<td>12 months</td>
<td>Two words with meaning</td>
</tr>
<tr>
<td>18 months</td>
<td>Ten words with meaning</td>
</tr>
<tr>
<td>24 months</td>
<td>Simple sentence</td>
</tr>
<tr>
<td>36 months</td>
<td>Telling a story</td>
</tr>
</tbody>
</table>

**Table 8:** Key developmental milestones—personal and social

<table>
<thead>
<tr>
<th>Age</th>
<th>Milestone</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 months</td>
<td>Social smile</td>
</tr>
<tr>
<td>3 months</td>
<td>Recognizes mother</td>
</tr>
<tr>
<td>6 months</td>
<td>Smiles at mirror image</td>
</tr>
<tr>
<td>9 months</td>
<td>Waves bye bye</td>
</tr>
<tr>
<td>12 months</td>
<td>Plays a simple ball game</td>
</tr>
<tr>
<td>36 months</td>
<td>Knows gender</td>
</tr>
</tbody>
</table>

Children with developmental delay should be evaluated for the cause of delay and appropriately managed.
“Child is not a small adult, neonate is not a small child”. Studies have shown that anesthesia-related complications are more in children than adults, more so in neonates and infants. In order to reduce the morbidity and mortality, anesthesiologists should have a sound knowledge of the physiological differences, immaturity of the organ systems and their implications in various age groups of children from premature neonates to school going children. The differences begin to recede around 10th year.

RESPIRATORY PHYSIOLOGY

This section on respiratory physiology aims to acquaint the reader with various neonatal adaptations in the respiratory system which takes place as soon as the fetus is delivered ex utero, to describe the qualitative and quantitative differences in respiratory physiology of infants and children from that of an adult and their inherent implications affecting pediatric anesthesia practice.

The main function of the human respiratory system is to maintain blood oxygen and carbon dioxide levels within physiological limits. Additional functions include regulation of acid base balance, thermoregulation and also to a certain extent, metabolism.

Rhythmic respiratory movements begin in the fetal stage, which is episodic and varies in frequency. With clamping of the umbilical cord and increasing levels of arterial oxygen tension, the rhythmogenesis is initiated and sustained. The first breath of a neonate is the most crucial one and requires negative pressures of 30 to 70 cm H$_2$O to push fluid out of the lungs which then gets filled with air.

Delayed clearance of this lung fluid could result in a benign self-limiting condition termed as transient tachypnea of newborn (TTN). TTN usually occurs in term infants with tachypnea lasting for 24–72 hours and responds well to symptomatic treatment. With increase in lung volumes, pulmonary vascular resistance rapidly decreases, thus allowing increase in pulmonary blood flow and establishment of alveolar and arterial blood gas exchange.

Morphological development of lung and its implications pertinent to anesthesia are described in Table 9.

Before understanding the various patterns of breathing in neonates and how their response to changes in blood oxygen and carbon dioxide tensions is different from that of adults, a brief about the regulatory mechanisms of respiration.

Control of Breathing

Respiratory rhythmogenesis is initiated by the respiratory neurons in the medulla and pons. The pontine respiratory group of neurons, dorsal respiratory group of neurons, ventral respiratory group of neurons and pre-Bötzinger complex are involved in rhythm generation. The axons of these neurons travel down the spine and innervate the muscles of inspiration and expiration via the interneurons and motor neurons which results in rhythmic inspiration and expiration. A number of receptors which sense a change in lung volume, arterial oxygen tension, carbon dioxide or H$^+$ ion concentration provide a feedback to medullary and higher respiratory centers which influences the depth and frequency of breathing.

Inputs to respiratory center are described in Table 10. The peripheral and medullary chemoreceptors are involved in providing ascending inputs to the higher respiratory centers in medulla and pons.

<table>
<thead>
<tr>
<th>Stages</th>
<th>Weeks of gestation</th>
<th>Development</th>
<th>Implications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Embryonic</td>
<td>0–10</td>
<td>Groove on ventral aspect of foregut to form a pouch. Formation of lung buds</td>
<td>Tracheoesophageal fistula, A-V malformation, congenital lung cysts can occur if growth disturbed</td>
</tr>
<tr>
<td>Pseudo-glandular</td>
<td>10–17</td>
<td>Budding of bronchi and lung growth to form loose mass of connective tissue</td>
<td>CDH, CCAM, pulmonary sequestration</td>
</tr>
<tr>
<td>Canalicular</td>
<td>18–24</td>
<td>Development of respiratory bronchioles with growth of capillaries around it</td>
<td>Preterm birth results in severe respiratory distress (poorly developed peripheral airways and immature lung cells)</td>
</tr>
<tr>
<td>Saccular</td>
<td>25–32</td>
<td>Saccules and capillary network around it develops for pulmonary gas exchange. Development of type II pneumocytes</td>
<td>HMD seen in preterm neonates born during this phase</td>
</tr>
<tr>
<td>Alveolar</td>
<td>32 weeks–18 month</td>
<td>Airspace wall thickness decreases</td>
<td>Extraterine viability is likely after 26 weeks of gestation</td>
</tr>
</tbody>
</table>

Abbreviations: CDH, congenital diaphragmatic hernia; CCAM, congenital cystic adenomatoid malformation; HMD, hyaline membrane disease
Other neural inputs to the respiratory control system are via the respiratory muscle proprioceptors, upper airway and lung stretch receptors, laryngeal and tracheal receptors, irritant receptors throughout the airway and pulmonary J receptors. These receptors as described in Table 11 are sensitive to inflation, deflation, mechanical and chemical stimulation.8

**Response to Hypoxemia**

Biphasic response to hypoxemia is seen in neonates up to 2–3 weeks of age and in preterms up to 25 days after birth. Initial transient hyperapnea for approximately 1 minute followed by decrease in ventilation leading to apnea is seen in thermoneutral environment.9

**Response to Carbon Dioxide Levels**

Increase in ventilation as a response to hypercapnia is seen in neonates, but the slope of response is decreased. However, in contrast to adults and older children, in the presence of hypoxemia, the CO₂ slope is decreased and shifted to right.9

**Upper Airway Receptors**

The upper airway receptors are extremely sensitive to noxious stimuli and result in inhibitory response causing apnea or ventilatory depression. This sensitivity has been attributed to immaturity of central nervous system.

---

**Table 10: Chemoreceptors and their role**

<table>
<thead>
<tr>
<th>Receptor</th>
<th>Location</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carotid body chemoreceptors</td>
<td>Bifurcation of common carotid artery</td>
<td>Type I glomus cells respond to changes in partial pressure of oxygen. Hypoxemia (PaO₂ &lt; 60 mm Hg) results in increase in respiratory rate in all age groups except in newborns especially preterm neonates. Hypocapnia increases ventilation by about 9–10% above which respiration is decreased. Hypoxemia potentiates the CO₂ response curve.</td>
</tr>
<tr>
<td>Central medullary chemoreceptors</td>
<td>Ventrolateral medulla</td>
<td>Respond to changes in H⁺ ion concentration in cerebrospinal fluid</td>
</tr>
</tbody>
</table>

**Table 11: Peripheral receptors**

<table>
<thead>
<tr>
<th>Stimulus/receptor area</th>
<th>Receptor response</th>
<th>Larynx</th>
<th>Bronchi</th>
<th>Mucous</th>
<th>CVS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nose</td>
<td>Apnea/Sneeze</td>
<td>Constriction</td>
<td>Constriction/dilatation</td>
<td>Secretion</td>
<td>Blood pressure ↑</td>
</tr>
<tr>
<td>Epipharynx</td>
<td>Aspiration</td>
<td>Constriction</td>
<td>Dilatation</td>
<td>Secretion</td>
<td>Blood pressure ↑</td>
</tr>
<tr>
<td>Larynx</td>
<td>Cough, apnea, aspiration</td>
<td>Constriction</td>
<td>Constriction</td>
<td>Secretion</td>
<td>Blood pressure ↑</td>
</tr>
<tr>
<td>Tracheobronchial stretch</td>
<td>Apnea</td>
<td>Dilatation</td>
<td>Dilatation</td>
<td>Nil</td>
<td>Heart rate ↑</td>
</tr>
<tr>
<td>Airway irritant</td>
<td>Cough/hyperpnea</td>
<td>Constriction</td>
<td>Constriction</td>
<td>Secretion</td>
<td>Blood pressure ↑</td>
</tr>
</tbody>
</table>

**Types of Breathing**

*Periodic breathing:* Periodic breathing is defined as regular breathing interspersed with repetitive short apneic spells lasting 5–10 seconds without oxygen desaturation or cyanosis. It is seen in neonates and young infants during wakefulness, sleep (REM, NREM stages). It usually decreases by 10–12 months of age.

*Apnea of prematurity (AOP):* AOP is described as apnea for 15 seconds or a shorter duration respiratory pause with either bradycardia (HR <100), cyanosis or pallor. The incidence is about 55% in preterm infants especially with weight less than 2 kilograms.

Apnea in premature infants is exacerbated by hypoxia, sepsis, intracranial hemorrhage, metabolic abnormalities, hypothermia, upper airway obstruction, heart failure, anemia, vasovagal reflexes and drugs, including prostaglandins and anesthetic agents.

Apneic episodes can be either central apnea with absence of breathing efforts or an obstructive pattern in the presence of breathing. Central apnea results from immaturity of respiratory centers causing decrease in central respiratory output whereas obstructive apnea is usually the result of upper airway obstruction, most commonly at the level of pharynx.

Most apneas in neonates are however, mixed in origin with poor respiratory drive as well as inability to maintain a patent upper airway as contributing factors.12
Apparent life-threatening events (ALTE): Sudden onset episode of color change/tone change and apnea which requires immediate resuscitation to revive the infant and restore normal breathing.

Postoperative apnea: Prolonged apnea >15 seconds or brief pause with bradycardia, arterial oxygen desaturation in the postoperative period is commonly encountered in preterm and ex-preemies undergoing elective surgery. Higher incidence is seen with lower postconceptual age, co-existing anemia with hematocrit less than 30% and after major procedures such as laparotomy. Ex-premature infants up to 60 weeks of postconceptual age require monitoring after both regional and general anesthesia for a minimum of 12 apnea free hours after surgery.

Apneic spells are treated by stimulation, bag-mask ventilation and the use of respiratory stimulants such as caffeine or theophylline, neonatal continuous positive airway pressure (CPAP) or ventilation.

Effects of Anesthesia on Control of Breathing

1. Upper airway receptors are more sensitive in infants and thus in response to inhalational induction, reflex coughing, breath holding and laryngospasm are commonly encountered.

2. The upper airway muscles especially the pharyngeal dilator muscles which help to keep the airway open are easily depressed by anesthetics, thus airway obstruction in infants and young children during inhalational induction should be anticipated and can be easily prevented by placement of an oropharyngeal airway/addition of 5–6 cm H₂O of positive end-expiratory pressure (PEEP).

Physiological Aspects of Development of Chest Wall Mechanics and Lung Volumes

High chest wall compliance is seen at birth because of floppy cartilaginous rib cage with horizontally placed ribs. Thus, the rib cage becomes more circular and mechanically less efficient. With ossification and gradual decrease in thoracic index, the cross sectional shape of thorax changes to ovoid improving the efficiency of rib cage.

The major muscle of inspiration which is the diaphragm is prone to respiratory muscle fatigue because of various reasons:

- The angle of insertion is horizontal resulting in reduced contractility when compared to adults where in the insertion is oblique.
- Smaller area of apposition which tends to suck the chest wall inward rather than draw air into the lungs.
- The infantile diaphragm lacks the Type I fibers which are slow twitch with high oxidative capacity and are thus fatigue resistant.

Implication: Infants are more susceptible to respiratory failure with increase in demand.

In neonates, a high ratio between chest and lung compliance decreases the resting lung volumes and transpulmonary pressures promoting alveolar collapse and a further decline in lung volumes (Table 12).

Table 12: Respiratory variables in neonates and adults and implications for anesthesia

<table>
<thead>
<tr>
<th>Variable</th>
<th>Newborn</th>
<th>Adults</th>
<th>Implication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency /minute</td>
<td>30–50</td>
<td>12–14</td>
<td>Maintain an I:E ratio of 1:1</td>
</tr>
<tr>
<td>Tidal volume mL/kg</td>
<td>6–8</td>
<td>6–8</td>
<td>Pressure controlled ventilation preferred to prevent volutrauma and barotrauma</td>
</tr>
<tr>
<td>Minute ventilation mL/kg/min</td>
<td>200–260</td>
<td>90</td>
<td>Control ventilation (spontaneous breathing only for very brief procedures)</td>
</tr>
<tr>
<td>Vital capacity (mL)</td>
<td>120</td>
<td>4000</td>
<td></td>
</tr>
<tr>
<td>Functional residual capacity mL/kg</td>
<td>30</td>
<td>30</td>
<td>Add PEEP</td>
</tr>
<tr>
<td>Oxygen consumption mL/kg/min</td>
<td>6–8</td>
<td>3–4</td>
<td>Preoxygenate before laryngoscopy, prone to hypoxemia</td>
</tr>
<tr>
<td>Physiological V̇ₚ/̇Vₚ</td>
<td>0.3–0.5</td>
<td>0.3</td>
<td></td>
</tr>
<tr>
<td>Physiological Q̇O₂/Q̇ĊO₂</td>
<td>0.1 (10%)</td>
<td>0.01–0.03 (1–3%)</td>
<td>Add PEEP</td>
</tr>
<tr>
<td>Lung compliance mL/cm H₂O</td>
<td>5 (1/20 of adult)</td>
<td>100</td>
<td>Assist ventilation during both induction and emergence</td>
</tr>
<tr>
<td>Chest wall compliance mL/cm H₂O</td>
<td>260 (5 times adult)</td>
<td>60</td>
<td></td>
</tr>
<tr>
<td>PO₂ mm Hg</td>
<td>60–90</td>
<td>80–100</td>
<td>Keep FiO₂ as low as possible to maintain SpO₂</td>
</tr>
<tr>
<td>PCO₂ mm Hg</td>
<td>30–35</td>
<td>37–42</td>
<td></td>
</tr>
</tbody>
</table>
Mechanics that maintain lung volume in newborns are an increased rate of breathing, a dynamic increase in FRC and sustained tonic activity of the inspiratory muscles throughout respiratory cycle. Dynamic increase in FRC is brought about by termination of expiration at substantial flow rates by postinspiratory diaphragmatic activity (diaphragmatic braking) and laryngeal narrowing during expiration (laryngeal braking), thus generating an auto PEEP effect.14

Under static conditions like under general anesthesia or during apnea, the FRC decreases further and thus it is essential to provide a PEEP of 4–6 cm of H2O to prevent alveolar collapse and maintain FRC.

**Closing Volume and Closing Capacity**
Closing volume is the volume of air remaining in the lung when small alveoli and airways in independent regions of lung are collapsed. Infants have a closing capacity greater than FRC in supine position leading to V/Q mismatch.

**Implication:** Atelectasis can be prevented by controlled ventilation and PEEP to recruit the collapsed alveoli.

**Airway Resistance**
Upper airway resistance (mouth, nose, pharynx, and larynx) contribute to 65% of total airway resistance. Large airways (trachea, large bronchi) contribute to 30% and the peripheral airways contribute to less than 5% of total airway resistance.

**Implication:** Infants are prone to airway obstruction of both upper and lower airways. Upper airways are more compliant and prone to dynamic collapse during forceful inspiration (Bernoulli Effect). The lower airways have smaller diameters leading to higher flow resistance in accordance to Poiseuille’s Law.

The cephalad position of the epiglottis and its close proximity to the soft palate imposes a higher resistance to mouth breathing when compared to nasal breathing. Hence, it is essential to keep nasal passages clear at least up to 3–6 months of age.

**Ventilation and Pulmonary Gas Exchange**
Alveolar ventilation is higher per unit of lung volume in infants to meet the higher oxygen requirements. There is uneven distribution of ventilation within the lungs more due to regional mechanical properties of lung than gravitational forces. Gas exchanging surface area is smaller in relation to body size compared to adults. Diffusion capacity is flow limited in newborns and gradually increases linearly with height.

**Implication:** Infants desaturate more rapidly because of increased oxygen demands and high FRC compared to TLC. In infants with unilateral lung disease in lateral decubitus position, oxygenation improves with healthy lung “up” or nondependant position.

**Surfactant**
Surfactant is a complex lipoprotein with dipalmitoyl-phosphatidylcholine and phosphatidylglycerol as surface active component. Surfactant is essential to keep small air sacs open and to prevent lung collapse. It is secreted from Type II pneumocytes of alveolar wall by 22 weeks of gestation. A peak increase is seen by 30–35 weeks and at birth.6

Surfactant production is significantly decreased in preterm infants, in term infants born to diabetic mothers and may lead to respiratory distress syndrome (RDS).

Surfactant production can be enhanced by antenatal administration of steroids to mothers in preterm labor. Surfactant replacement therapy is indicated for preterm infants with HMD, neonates with persistent pulmonary hypertension of the newborn (PPHN), congenital diaphragmatic hernia (CDH), meconium aspiration syndrome (MAS) and in children with acute respiratory distress syndrome (ARDS).

**Oxygen Transport**
Most of the oxygen is carried in blood by combining reversibly with hemoglobin and very small amount is dissolved in plasma. In newborns, the oxygen affinity of fetal hemoglobin is very high and P50 is low (18–19 mm Hg) because of low 2, 3 DPG levels and HbF which reacts poorly with 2,3 DPG.7

By 2 to 3 months of age, most of the HbF is replaced with HbA and P50 increases rapidly and is above adult values (P50 30 mm Hg) up to first decade of life.

High levels of inspired oxygen are to be avoided to reduce oxygen toxicity, absorption atelectasis, BPD and ROP. Clinically SpO2 levels of 90–95% in preterm and 95–97% in term neonates and infants should be aimed.

**CARDIOVASCULAR PHYSIOLOGY**
Children differ from adults in various ways as far as cardiovascular system is concerned. The immature organ system continues its maturation through childhood and it is a continuum. Therefore, understanding the limitations of the system is of utmost importance while anesthetizing children. It is also important to understand the physiology of fetal circulation, the changes that happen soon after birth, the sympathetic and parasympathetic innervations and the effects of anesthetic agents on the cardiovascular system.
Fetal Circulation (Fig. 2)

Placenta forms the organ of respiration in prenatal life. Fetal circulation is a parallel circulation with a low resistance system having low systemic vascular resistance and high pulmonary vascular resistance due to fluid-filled lungs and hypoxic environment. Lungs do not play any role in gas exchange, and there is minimal pulmonary blood flow. The oxygenated blood with a saturation of 80% is carried from placenta through umbilical vein with a high velocity. On reaching the fetal liver it divides into two pathways. About 50–60% of the umbilical blood bypasses the liver via ductus venous. The remainder perfuses the left lobe of the liver. The right lobe of the liver receives its blood supply from portal circulation. Right and left hepatic veins along with ductus venous join supra hepatic IVC. In IVC there are two streams of blood one with high velocity and the other with low velocity from right hepatic vein and abdominal IVC. On reaching the right atrium predominantly high velocity blood from umbilical vein and ductus venous which is more oxygenated enters left atrium through foramen ovale and low velocity blood enters the right ventricle through tricuspid valve. From left ventricle the blood is distributed to the heart and brain through ascending aorta. Blood from the SVC enters the right atrium then to the right ventricle. Because of high pulmonary vascular resistance, the blood from right ventricle enters the aorta through ductus arteriosus which connects right pulmonary artery to arch of aorta. Descending aorta carries blood which is a mix of oxygenated and deoxygenated blood. The kidneys, gut and lower limbs receive blood with a saturation of 55%. Two umbilical arteries return blood to the placenta.9

Circulatory Changes at Birth (Fig. 3)

Transitional Circulation

Expansion of the lungs due to cry increases the partial pressure of oxygen and release of vasoactive substances leads to decrease in pulmonary vascular resistance. As a result pulmonary blood flow and oxygenation increase. Clamping of placenta leads to increase in systemic vascular resistance, functional closure of ductus arteriosus at 15–18 hrs, but the anatomical closure occurs at 2–3 weeks. Increase in pulmonary blood flow results in increased left atrial filling and functional closure of foramen ovale. Anatomical closure occurs at 6 weeks.15

Persistent Fetal Circulation

It is a state of circulation where in the neonates return back to fetal circulation because of hypoxia, hypercapnia, acidosis, hypothermia, increasing the pulmonary vascular resistance. This increase in PVR will cause the opening of ductus arteriosus (DA) and foramen ovale leading to increased right to left shunting of blood and decreased oxygenation. This can happen till the anatomical closure of DA occurs.15
Chapter 2: Physiological Characteristics and Anesthetic Implications

The conditions causing persistent fetal circulation in neonates are respiratory distress syndrome (RDS) which is common in premies, congenital diaphragmatic hernia and the meconium aspiration syndrome. It is diagnosed by the difference in the PaO₂ of >20 mm Hg between pre- ductal and postductal sites, i.e. preductal is right radial artery and postductal is left radial, umbilical artery, posterior tibial, or dorsalis pedis artery.

Pulmonary Vascular Resistance

Understanding the physiology of pulmonary circulation is important because pulmonary vasculature and pulmonary vascular resistance play an important role in the smooth transition from fetal to the neonatal circulation. Many factors in vitro and in vivo influence pulmonary vascular resistance.

At midgestation pulmonary vascular resistance is tenfold higher than it is at 24 hours after birth. During the last trimester it decreases to 7–8 times greater than at 24 hours after birth. This reduction is due to the increase in the cross-sectional area. The theory is that once there is development of normal structure, there is corresponding increase in the blood flow, which in turn stimulates normal growth. In fetal life pulmonary vasculature must grow normally despite reduced flow, but will retain the ability to vasoconstrict. The main factor responsible for vasoconstriction is the hypoxic environment. The PaO₂ of the pulmonary artery is approximately 20 mm Hg.

After birth there will be rapid phase of pulmonary vasodilation followed by a period of remodelling which is completed by 6 months of age. Factors such as hypoxia, hypercarbia, acidosis, pain will increase pulmonary vascular resistance leading to PPHN. This is also seen in neonates with meconium aspiration syndrome, sepsis, polycythemia and surgical condition like congenital diaphragmatic hernia.

Cardiac Output and the Myocardium

The cardiac output is about 200–300 mL/kg/min in neonates. In vitro, it is a combination of right ventricular and left ventricular output. Right ventricle has to work more because of the increase in pulmonary vascular resistance and so there is right ventricular hypertrophy. After birth right ventricle remodels its size and left ventricle takes over.

Myocardial Performance

The neonate has immature myocardium which completely matures by 1 year. After this age, the child responds to preload, after load and contractility similar to adults. The heart rate and blood pressure values will not reach the adult level till adolescence (Tables 13 and 14).

Neonates have more of type 1 myofibrils which have less elasticity and are more rigid, whereas type II isomer which contributes to elasticity is 30% which is less than adult value of 60%. As the myocardium becomes more mature the number of type II fibers increases and gradually exceeds type 1 fibers. These type 1 fibers lack sufficient sarcoplasmic reticulum, contains incompletely developed 'T' tubules which are responsible for calcium influx and myocardial contraction. Hence, cardiac output mainly depends on heart rate than stroke volume in neonates and infants. Maintaining heart rate is of utmost importance in neonates. Bradycardia will drastically decrease the cardiac output.

Autonomic Nervous System

Autonomic nervous system is immature in the neonatal period. Although, both sympathetic and parasympathetic innervation can be demonstrated at birth, the sympathetic system is incomplete whereas parasympathetic system reaches maturity few days after birth. This relative imbalance leads to parasympathetic predominance in neonates leading to vagal sensitivity and preponderance to bradycardia.

Developmental maturation of human heart is completed by 6 months of age. Till then one has to be careful about the cardiac depressant action of volatile anesthetic.

Table 13: The relationship of age and heart rate

<table>
<thead>
<tr>
<th>Age</th>
<th>Heart Rate (beats/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preterm</td>
<td>120–170</td>
</tr>
<tr>
<td>0–3 months</td>
<td>100–150</td>
</tr>
<tr>
<td>3–6 months</td>
<td>90–120</td>
</tr>
<tr>
<td>6 months–1 year</td>
<td>80–120</td>
</tr>
<tr>
<td>1 year–3 years</td>
<td>70–110</td>
</tr>
<tr>
<td>3–6 years</td>
<td>65–110</td>
</tr>
<tr>
<td>6–12 years</td>
<td>60–90</td>
</tr>
</tbody>
</table>

Table 14: Relationship between age and blood pressure

<table>
<thead>
<tr>
<th>Age</th>
<th>Mean systolic BP (mm Hg)</th>
<th>Mean diastolic BP (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Premature</td>
<td>55–85</td>
<td>35–45</td>
</tr>
<tr>
<td>0–3 months</td>
<td>65–85</td>
<td>45–55</td>
</tr>
<tr>
<td>3–6 months</td>
<td>70–90</td>
<td>50–55</td>
</tr>
<tr>
<td>6–12 months</td>
<td>80–100</td>
<td>55–65</td>
</tr>
<tr>
<td>1–3 years</td>
<td>90–105</td>
<td>55–70</td>
</tr>
<tr>
<td>3–6 years</td>
<td>95–110</td>
<td>60–75</td>
</tr>
<tr>
<td>6–12 years</td>
<td>100–120</td>
<td>60–75</td>
</tr>
</tbody>
</table>
agents. Because of increased alveolar ventilation there is rapid rise in the concentration which can lead to bradycardia and sudden cardiac arrest especially at the time of induction of anesthesia.9

**PHYSIOLOGY OF CENTRAL NERVOUS SYSTEM**

The central nervous system is immature in neonates and infants due to the decreased cell differentiation, immature blood brain barrier and incomplete myelination. The brain of the neonate is large compared to the body weight and weighs 1/10th of body weight whereas in adults it is 1/50th of body weight. Neuronal cell differentiation, axon and dendritic remodulations continue into the postnatal life. Incomplete myelination is responsible for primitive reflexes in neonates. Myelination is complete by 2 years of age.9,15

**Blood-brain Barrier**

Blood-brain barrier (BBB) separates brain parenchyma from blood. It is formed by the tight junctions between capillary endothelial cells of the blood vessels. These cerebral capillaries have no fenestrations and demonstrate selective permeability after maturation. It allows the diffusion of glucose, essential amino acids, water and carbon dioxide. BBB is immature in neonates allowing the passage of lipid soluble molecules and various drugs. Because of the immaturity free bilirubin in the blood can cross the BBB causing kernicterus and any osmotic changes like transfusion of hypertonic solutions can cause cerebral damage.15

**Physiology of Cerebrospinal Fluid**

Cerebrospinal fluid (CSF) is produced by choroid plexus and absorbed through arachnoid villi and ependymal lining of ventricles. Derangements of CSF production and reabsorption account for significant mortality in children. The CSF physiology is well-described in adults but it is not clear in children. Rate of CSF production is age dependent and is around 0.3–0.5 mL/min in children and increases during first year of life reaching 60% of adult value (400–500 mL/day) by 2 years of age. Total CSF volume in a neonate is approximately 4mL/kg as compared with 2 mL/kg in an adult (Table 15).9,15

<table>
<thead>
<tr>
<th>Age</th>
<th>CSF pressure (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Children</td>
<td>3–7.5</td>
</tr>
<tr>
<td>Adults</td>
<td>4–13.5</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Age</th>
<th>CSF volume (mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infants</td>
<td>40–60</td>
</tr>
<tr>
<td>Young children</td>
<td>60–100</td>
</tr>
<tr>
<td>Older children</td>
<td>80–120</td>
</tr>
<tr>
<td>Adults</td>
<td>100–160</td>
</tr>
</tbody>
</table>

**Cerebral Blood Flow**

Neonatal brain receives 25% of the cardiac output whereas adult brain receives 15% of the cardiac output. Cerebral blood flow (CBF) in a neonate is 30–40 mL/100 g/min, but in infants and older children it is 80–100 mL/100 g/min which is higher than the adult value of 50 mL/100 g/min. CBF is regulated to meet the cerebral metabolic rate for oxygen (CMRO₂). CBF increases with increase in CMRO₂, e.g. fever, seizures. Likewise CBF decreases with decrease in CMRO₂ in conditions such as hypothermia or when barbiturates are administered. CMRO₂ is higher in children (5 mL/100 g/min) than adults (3–4 mL/100g/min).9,15

**Autoregulation of Cerebral Blood Flow**

Autoregulation is a protective mechanism to maintain a constant CBF across a range of systemic blood pressure. Pressure autoregulation allows the brain to steal and shunt blood flow to and from the systemic vasculature. The upper and lower limit of pressures for auto regulation in adults is well-documented. In pediatric population it has not been documented. The autoregulatory range is narrower in neonates compared to infants and older children and hence they are prone to cerebrovascular injuries. CBF depends on cerebral perfusion pressure, arterial oxygen saturation (PaO₂) and carbon dioxide concentration (PaCO₂).9,15

**Effects of Blood Pressure**

In adults cerebral blood flow remains relatively constant within a mean arterial pressure range of 50–150 mm Hg. Beyond this range the cerebral blood flow becomes pressure dependant. In neonates, the limit of auto-regulation is at a lower range.

When cerebral perfusion pressure decreases there will be dilatation of cerebral vessels thereby increasing the cerebral blood flow. Likewise whenever cerebral perfusion pressure increases there will be cerebral vasoconstriction leading to decrease in blood flow.

Any acute and sudden changes in the blood pressure can cause rupture of the fragile vessels leading to intra-
cranial and intraventricular hemorrhages which are most commonly seen in preterm neonates.

**Effects of Oxygen**

When the partial pressure of oxygen (PaO₂) falls, there is cerebral vasodilatation and increase in cerebral blood flow. Evidence suggests that hyperoxia decreases cerebral blood flow. Breathing 100% oxygen causes 10% decrease in cerebral blood flow in adults whereas the decrease in neonates is about 33%.

**Effects of Carbon Dioxide**

The changes in the arterial partial pressure of carbon dioxide cause a linear change in cerebral blood flow. In patients with raised intracranial pressure (ICP), this direct effect is the basis for hyperventilation to reduce ICP. Likewise hypercarbia causes cerebral vasodilatation and increase in cerebral blood flow. There is no data to suggest the limits at which it occurs in neonates and infants.

**Development of Pain Pathways**

The development of nociception and pain pathways starts in the second trimester of pregnancy and completes by 2 years of age. Studies have shown that the fetus can experience pain and all fetal surgeries require sedation or anesthesia. Evidence has shown that neonates can experience pain, but the response varies from facial grimace to reflex withdrawal of limbs. Experience of severe pain in neonatal period can lead to long-term adverse consequences to sensory processing mechanisms resulting in exaggerated response to subsequent pain stimulus for months to years later. Hence pain relief and perioperative pain management is very essential in neonatal period.15,16

**Spinal Cord and Dura Matter**

At birth spinal cord ends at the level of third lumbar vertebra and dura mater ends at the level of third sacral vertebra. By 1 year the cord ascends to adult level of first lumbar vertebra and dura at first sacral vertebra.

**Implications**

- Spinal anesthesia to be performed between L4-L5, L5-S1 in infants
- Lack of myelin, reduced size of nerve fiber and shorter distance between the nodes of Ranvier favor penetration of local anesthetics and rapid onset of nerve blockade. Hence, children require lower concentrations of local anesthetics and are prone for local anesthetic toxicity.

**RENAL PHYSIOLOGY**

Kidneys play a vital role in the maintenance and homeostasis of fluids and electrolytes. The developmental abnormalities of the kidney and urinary tract are the major cause for early renal failure in children. Renal function in children, especially in neonates is characterized by reduced renal blood flow, glomerular filtration rate (GFR), solid excretion and concentrating ability (Table 16).

**Renal Blood Flow**

Fetal kidney receives 2–3% of cardiac output which increases to 4–6% during first 12 hrs of life and 8–10% by one week. Effective renal plasma flow reaches adult levels by 12–24 months. Neonatal kidneys can autoregulate the renal blood flow at lower systemic perfusion pressures. The internal distribution of renal blood flow is slightly

<table>
<thead>
<tr>
<th>Table 16: Renal function in relation to age</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Measurement</strong></td>
</tr>
<tr>
<td>GFR (mL/min/1.73m²)</td>
</tr>
<tr>
<td>RBF (mL/min/1.73 m²)</td>
</tr>
<tr>
<td>TmPAH(mg/min/1.73 m²)</td>
</tr>
<tr>
<td>Maximal concentration ability (mOsm/kg)</td>
</tr>
<tr>
<td>Serum creatinine (mg/dL)</td>
</tr>
<tr>
<td>TmG(mg/min/1.73 m²)</td>
</tr>
<tr>
<td>Fractional excretion of sodium (%)</td>
</tr>
<tr>
<td>TmP/GFR(mg/dL)</td>
</tr>
</tbody>
</table>

Abbreviations: GFR, Glomerular filtration rate; RBF, renal blood flow; TmPAH, tubular maximum for para aminohippuric acid; TmG, tubular maximum for reabsorption of glucose; TmP, tubular maximum for phosphorous
different in neonates. Higher proportion goes to juxtamedullary nephrons leaving cortical nephrons at a higher risk for ischemia. With maturation there will be gradual rise in renal blood flow due to improved cardiac output, perfusion pressure and decrease in renovascular resistance. Renal blood flow is about 20 mL/min/1.73 m² at 30 weeks, 80 mL/min/1.73 m² at term, and 770 mL/min/1.73 m² at 5 months of age.6

**Glomerular Filtration**

Among the main functions performed by the kidneys is the process of glomerular filtration. The glomerular filtration is low in children especially in neonates compared to adults. Glomerular filtration begins in the fetus during 9th week. It reaches 12 mL/min/1.73 m² at 30 weeks to 20 mL/min/1.73 m² at term. Adult values of 60–80 mL/min/m² are reached by 18–24 months of age.9,15

**Serum Creatinine**

At birth serum creatinine reflects maternal values and is greater than that of 2 week old term neonate (0.4 mg/dL). For the first 4 weeks the serum creatinine values will be more in preterm neonates than term neonates. This variability in GFR and creatinine clearance indicates that the drugs which depend mainly on kidneys for elimination will have a variable half-life during the first weeks to months of postnatal life.

**Tubular Function**

The proximal tubule is the site of re-absorption of large quantities of solutes and filtered fluid. The solutes include sodium, potassium, calcium, phosphorus, magnesium and amino acids. This tubular function is immature especially in neonates making them obligate salt losers.

**Sodium**

The absorption of sodium depends on Na⁺-K⁺ ATPase pump. Fractional excretion of sodium (FENa) gradually falls as the gestational age advances, from 12.8% at 30 weeks to 3.4% at 38 weeks as compared to adult’s value of 1%. Hence preterm neonates are salt losers and at a higher risk of hyponatremia. Term neonates have a limited capacity both to conserve and to excrete sodium load.11 Implication is that excessive administration of sodium may cause extracellular volume expansion, edema and hyponatremia.

**Glucose**

Glucose is reabsorbed in the proximal convoluted tubule and depends on Na⁺-K⁺ ATPase pump activity. In preterm neonates there is decreased Na⁺-ATPase pump activity leading to glucose wasting.15

**Implications**

- Meticulous management of perioperative fluids is important
- Avoid hyperglycemia as it causes osmotic diuresis
- Effects of drugs excreted through kidney will be prolonged, e.g. pancuronium, morphine, digoxin and antibiotics.

**HEPATIC PHYSIOLOGY**

Liver is immature in neonates which makes them prone to hypoglycemia, hyperbilirubinemia and impaired drug metabolism. Understanding the physiology and principles of development of liver is of utmost importance for anesthesiologists.15

**Functional Development**

The liver weighs about 10% of the body weight at 9 weeks of gestation, reducing to 4% in neonates and 2% in adults. The neonatal liver has immature hepatocytes which lack many enzyme systems and are deficient in glycogen.

**Carbohydrate Metabolism**

In intrauterine life fetus gets glucose from the placenta. Fetal hepatocytes are able to synthesize glycogen by 9th week of gestation. At birth term neonate has a glycogen store of 40–60 mg/g of the liver. This serves as a source of glucose for 12 hours after birth by which time breast-feeding should ideally be initiated and established. This phenomenon of glycogen storage and gluconeogenesis is not seen in preterm neonates and hence they are prone to hypoglycemia.15

**Protein Synthesis**

Liver is the site for protein synthesis. By 12 weeks of gestation there is evidence of protein synthesis. The main serum protein in fetal life is alpha-fetoprotein which reaches a peak by 12–13 weeks. Albumin synthesis starts at 3–4 months of gestation and approaches adult value by 15–18 months of age. In preterm neonates this level will be lower.12

**Synthesis of Clotting Factors**

Liver is responsible for the synthesis of Vitamin K dependant coagulation factors viz. factor II, VII, IX, and X. This in turn depends upon the enteral feeding and
colonization of the gut with bacteria which is an important source of vitamin K. The current guidelines recommend that all healthy neonates should receive vitamin K supplementation of 0.5–1 mg IM soon after birth. This will significantly reduce the risk of bleeding.16

**Principles of Hepatic Drug Metabolism**

Metabolism and detoxification of drugs and other toxic substances is the main function of the liver. Lipophilic drugs are difficult to excrete. The major role of the liver is to transform lipid soluble drugs into water soluble compounds which can be easily excreted through kidneys. It is a complex process and depends on various factors like the liver mass, blood flow, protein binding, enzymatic activity and the clearance process.15

The metabolism of the drugs involves two stages. Phase I is hydroxylation, which modifies the structure and Phase II is conjugation which makes them more water soluble and easily excreted through kidneys.

**Phase I Reactions**

Cytochrome P450 is the enzyme responsible for phase I reactions in the metabolism of the drugs. Cytochrome P450 is found in endoplasmic reticulum and mitochondria. They are NADPH-nicotinamide adenine dinucleotide phosphate and NADPH-cytochrome C reductase. They are responsible for oxidation and reduction. Phase I also includes hydrolysis of esters and amides and sulfation, dehalogenation and demethylation.

Cytochrome P450 enzymes are active in the fetal liver from 8 weeks of intra-uterine life, gradually increasing till term. CYP 3A4 is responsible for metabolism of majority of the drugs. Its activity is low in neonates, increasing to 50% of the adult level by 1 year and not fully expressed until 10 years.10 15

**Phase II Reactions**

This involves conjugation which makes them more water soluble, e.g. glucuronide, sulfation/acetylated derivative.

Glucuronide compounds are excreted in urine or through bile. Glucuronyl transferase activity is low in neonates. Hence, if there is significant hemolysis, it will lead to unconjugated hyperbilirubinemia. The level is low in neonates, i.e. 10–20% of adult level reaching full maturity by 1 year.10 15

**Physiological Jaundice**

It is a transient condition wherein there is an increase in the level of bilirubin. In term neonates it is commonly seen within the first three days of life with the bilirubin level of 6–8 mg/dL. In preterm neonates it occurs on 5–7th day of life with peak levels of 10–12 mg/dL. The cause of non-hemolytic hyperbilirubinemia is excessive bilirubin production from breakdown of RBC and increased enterohepatic circulation of bilirubin due to depressed glucuronyl transferase activity. It is a self-limiting condition, and usually resolves by 2 weeks. If it is slow to clear, phototherapy is needed to prevent kernicterus and encephalopathy.10

**Implications**

- There will be more free drug with increased elimination half-life
- Children, especially neonates are prone to hypoglycemia
- Coagulation abnormalities can occur; hence vitamin K supplementation is required in the initial days
- Neonates and small infants are unable to handle large protein loads, and hence prone to acidosis.

**GASTROINTESTINAL SYSTEM**

Most biochemical and physiological functions are present at birth but remain immature. The hydrolytic enzymes can digest a carbohydrate load sufficiently. The proteolytic enzymes required to digest proteins are present but immaturity of renal function poses a hazard to excess protein intake. The absorption coefficient of fats is 90–95% in neonates and more than 95% after 2 months of life. Though bile is present by 22nd week of gestation in fetus, bile secretion is inadequate in newborns. Fat malabsorption is significant in premature neonates who require formula feeds containing medium chain triglycerides whose metabolism is independent of bile salts.

Swallowing is an autonomic reflex for the first three months of life, later on with the development of striated muscles in the pharynx it has both voluntary and involuntary component. The lower esophageal sphincter and the pharyngoesophageal sphincters are immature which lead to regurgitation of food for the first weeks of life.10

**HEMOPOEITIC SYSTEM**

The hemopoietic system in children differs from adults because of the variation in the blood volume, hemoglobin and hematocrit values in various age groups.10 The blood volume in children varies according to the age as shown in the following chart.
Blood Volume in Relation to Age

- Preterm neonate: 90–100 mL/kg
- Term neonate: 85–90 mL/kg
- Infant: 80–85 mL/kg
- School age: 75–80 mL/kg
- Adults: 70 mL/kg

Implications

- Blood volume is relatively larger, but absolute volume is smaller
- Relatively small volumes of blood will constitute significant blood loss in small children, hence monitoring and documenting all blood loss, including amounts that would be insignificant in the adult patient is important
- The size of the child will also determine the amount of fluid required for fluid resuscitation
- Transfusion is generally recommended when 15% of the circulating blood volume has been lost
- Children have very efficient compensatory mechanisms, and will remain normotensive in spite of losing large intravascular volumes (up to 25%)
- Hypotension is a late sign of hypovolemia.

The normal values of hemoglobin in different age groups is depicted in Table 17. The site of sampling is important in the interpretation of the values. Capillary sampling (heel prick) in neonates generally gives higher values as high as 6 g/dL, hence venipuncture is preferred.

Physiological anemia is the normal physiological adjustment of term neonate to extrauterine life resulting in fall in Hb during second month of life to a nadir of 10–11g/dL. This is exaggerated in preterm neonates, nadir being reached earlier at 4–8 weeks to as low as 8 g/dL.

Polycythemia is defined as a condition where the hematocrit value is more than 65%. It occurs in 3–5% of full-term neonates. If persistent, it can lead to hyper- viscosity syndrome wherein there will be increase in the systemic and pulmonary vascular resistance and decreased cardiac output. Partial exchange transfusion is done to lower the hematocrit, to decrease the blood viscosity and to improve the organ blood flow.

Table 17: Hemoglobin and hematocrit values in relation to age

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Preterm</th>
<th>Term</th>
<th>1 year</th>
<th>Child</th>
<th>Adult</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hb (g/dL)</td>
<td>12–13</td>
<td>16</td>
<td>12</td>
<td>13</td>
<td>15</td>
</tr>
<tr>
<td>Hematocrit(%)</td>
<td>40–45</td>
<td>55</td>
<td>36</td>
<td>38</td>
<td>45</td>
</tr>
</tbody>
</table>

Fetal Hemoglobin

At birth neonate will have 80% of its hemoglobin as hemoglobin F (HbF). It gradually reduces to 5% by 6 months of age.

Characteristics of HbF

- Life span is 90 days compared 120 days in adult
- It has more affinity for oxygen

Implication

There will be good oxygen transport but poor delivery to the tissues. Fetal RBCs have decreased 2,3 DPG levels and hence oxyhemoglobin dissociation curve (ODC) is shifted to the left.

White Blood Cells

Neonates have an increased susceptibility to infections due to immaturity of leukocytic functions. There may be a minimal leukocytic response sometimes with leukopenia in presence of sepsis. The normal white blood cell (WBC) count is 21,000/mm³ in first 24 hrs falling to 18,000/mm³ by the end of first week. The neutrophils and lymphocytes are equal in number at birth, reaching adult values by puberty.

Platelets

Normal platelet count in children is 2.5–3 lakhs/mm³. Thrombocytopenia can occur in neonates due to mechanical ventilation, hyaline membrane disease and sepsis. The severity of platelet reduction is inversely proportional to the gestational age/birth weight. Infants with thrombocytopenia are at a greater risk of bleeding.

To conclude, one needs to understand the physiological differences and immaturity of organ systems in various age groups of children and its implications to plan and conduct safe anesthesia in children.

REFERENCES

INTRODUCTION
Pharmacokinetics and pharmacodynamics of drugs are different in children from those in adults. These differences make children respond differently to drugs, including anesthetics and other perioperative medications. Many perioperative medications used in children, especially in neonates and infants, are not extensively clinically studied and data is frequently extrapolated from adult literature. However, doing this in clinical practice may result in inappropriate selection of drugs, therapeutic doses and frequency of administration and may cause overdosing and serious drug toxicity in children. Thus, clear understanding of these pharmacokinetic and pharmacodynamic differences between children and adults is mandatory for anesthesiologists to provide safe anesthesia to children.

PHARMACOKINETIC DIFFERENCES OF DRUGS IN CHILDREN
Children, especially neonates and infants have a different body composition, reduced hepatic metabolic and renal function, decreased body fat and muscle mass as a proportion of body weight and lower protein binding of drugs than in adults. These differences together with some other fairly less significant factors (Table 1) produce differences in pharmacokinetics of drugs in children from those in adults. The pharmacokinetic differences are most marked in neonates and young infants and may persist over the first decade of life.

Neonates and preterm infants have delayed gastric emptying rate until 6–8 months of age. Thus, absorption as well as peak bioavailability of orally administered premedicants and postoperative pain medications are slower in this group of children. Sublingual and nasally administered drugs (e.g. sublingual fentanyl citrate and nasal midazolam) produce rapid onset of action as these agents skip first pass hepatic metabolism and quickly attain peak plasma level. Locally applied medications (such as local anesthetic creams, analgesic patches etc.) are rapidly absorbed in neonates and infants because of their highly perfused, relatively large and thin skin surface area compared to older children and adults. There is potential for drug overdose by this route. Absorption and bioavailability of rectally administered drugs are variable and unpredictable. Drugs administered into the lower part of the rectum initially bypass the liver. Intramuscularly administered drugs exhibit more rapid onset of action with higher bioavailability in children because of higher cardiac output and increased muscle blood flow. However, intramuscular injection is painful and is usually avoided in children. In infants, epidural space is highly vascular with smaller absorptive surface. Consequently, systemic absorption of epidurally administered drugs is faster in neonates and in infants until six months of age.

Volume of distribution is high in infants because of increased total body water content (as increased ECF) than in older children and adults. Preterm and term infants have higher proportion of extracellular fluid (ECF) (approximately 63% and 45%, respectively) compared to older children and adults (20%). Consequently, they
require higher bolus doses of water soluble drugs like succinylcholine, atracurium, etc. compared to older children and adults. In neonates and infants a higher percentage of cardiac output is distributed to their relatively larger cerebral and hepatic masses which causes rapid induction of anesthesia with intravenous agents. Recovery on the other hand is slower because redistribution to well-perfused and deep under-perfused tissues is more limited. Preterm and term neonates have reduced body fat (3% and 12%, respectively as a proportion of body weight which doubles by 4–5 months of age) and muscle mass relative to their body weight than older children and adults. Thus, they have reduced volume of distribution and hence increased and prolonged plasma level of drugs, such as thiopentone that redistribute into fat and muscles (lipid soluble drugs).

The activities of most of the liver enzymes as well as the hepatic blood flow are reduced in neonates. Microsomal activity of cytochrome P450 enzyme system is reduced at birth. Phase-I reactions reach adult levels a few days after birth whilst phase-II reactions mature by 3 months. Consequently, hepatic drug metabolism and biotransformation are reduced in neonates. However, drug metabolism and clearance is higher in children between 1 and 2 years of age because of mature hepatic enzyme systems and increased hepatic blood flow. The renal function in preterm and term infants is less efficient than in adults because of reduced glomerular filtration and tubular function. In term neonates, renal drug clearance becomes normal (adult level) by 3 and 4 weeks of age while glomerular filtration rate and tubular function reach adult level between 20 weeks and 2 years of life.

Neonates have lower albumin and α1-acid glycoprotein (AAGP) concentrations. This results in decreased protein binding and increased plasma level of free active form of highly protein bound drugs, such as thiopentone, bupivacaine, etc. and may cause enhanced clinical effects.

Blood-brain barrier (BBB) function attains adult level by the end of neonatal period. Protein-bound drugs do not normally cross the BBB. Relatively lipid insoluble (partially ionized) drugs, such as morphine cross immature BBB of premature neonates easily. Term neonate’s mature BBB allows passive diffusion of protein unbound, fat soluble drugs, such as bupivacaine to pass across it. Fentanyl moves in and out through the BBB with the help of ATP dependent active transport systems. CNS pathology, such as cerebral ischemia and inflammation may influence opioid transport and distribution across BBB.

**PHARMACODYNAMICS OF DRUGS IN CHILDREN**

Older children respond to pharmacological agents much the same way as adults. However, infants and neonates exhibit different pharmacodynamic responses towards many preoperative medications. Both in premature and term neonates neuromuscular blocking agents are more sensitive because of underdeveloped neuromuscular junction. MAC of all inhalational anesthetics is reduced in neonates than in infants (Table 2). Spinal block remains for shorter duration and requires higher dose of amide local anesthetics to produce same dermatomal level of anesthesia as in adults. While, bronchodilators are not effective because of paucity of bronchial smooth muscles, prokinetic drugs are fully effective only in older children. Neonatal heart stores less calcium and life-threatening bradycardia and hypotension may follow administration...
of calcium channel blockers, such as verapamil. Also externally administered calcium produces exaggerated responses. One should be aware that at birth, in both preterm and term infants, coagulation cascade is immature and may influence perioperative hemostatic effects. And, responses to benzodiazepines vary in children until the age of 10 years because of immature inhibitory GABA receptors.

COMMONLY USED PHARMACOLOGIC AGENTS IN PEDIATRIC ANESTHESIA

Inhalational Agents

Pharmacokinetics

The rate of rise of alveolar anesthetic concentration (wash-in) of inhalational agents in infants and children is more rapid than in adults. This is caused by a greater alveolar ventilation relative to functional residual capacity (5 to 1 in neonates, 1.5 to 1 in adults), distribution of a greater proportion of the cardiac output to the vessel-rich group of tissues, and lower blood/gas as well as tissue/gas solubility of more soluble inhaled anesthetics, such as halothane and isoflurane in children than in adults.

Rapid “wash-in” of volatile agents causes relatively rapid induction of anesthesia in younger children than in adults. Induction is more rapid with poorly soluble agents (such as sevoflurane) because of their reduced uptake from the lungs. However, as MAC of poorly soluble agents is higher in infants and young children thus, the speed of induction also depends on the rate of increase of inspired concentration as well as maximum inspired concentration of these agents. Therefore, the rate of induction is accelerated if the inspired concentration (of poorly soluble agents) is rapidly increased to a very high level in larger increments. Addition of 60% nitrous oxide decreases the MAC values of sevoflurane by 20 to 25%, halothane by 60%, isoflurane by 40% and desflurane by 25%. A reduction in body temperature, cerebral palsy and cognitive disorders also decreases MAC of volatile anesthetics in children.

Pharmacodynamics

Minimum alveolar concentration (MAC): In children, MAC varies significantly with age (Table 2). MAC is highest during the first year of life although at different time periods for different agents. MAC of all volatile agents except sevoflurane is lower in neonates compared to both children and adults. MAC value of sevoflurane is 3.3% (highest) in both neonates as well as in infants younger than 6 months. MAC in preterm neonates is studied only for isoflurane and found to be lower than for full-term infants. Beyond first year of life MAC value for all volatile anesthetics decreases as age increases. MAC of all volatile agents is 15 to 40% higher in children between 1 and 12 years of age than in young adults. MAC, however, does not vary significantly (10% or less) between children of this age group.

Addition of 60% nitrous oxide decreases the MAC values of sevoflurane by 20 to 25%, halothane by 60%, isoflurane by 40% and desflurane by 25%. A reduction in body temperature, cerebral palsy and cognitive disorders also decreases MAC of volatile anesthetics in children.

Individual Inhalational Agents

The most widely used inhalational anesthetics in the practice of pediatric anesthesia are nitrous oxide, sevoflurane, and isoflurane. Halothane is low-priced and thus is still being used, albeit less commonly, in many lesser developed and developing countries.

Table 2: MAC of commonly used inhalational agents in various age groups and effects of N₂O on MAC reduction

<table>
<thead>
<tr>
<th>Agent</th>
<th>MAC with oxygen (%)</th>
<th>MAC with 60% N₂O (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Neonates</td>
<td>Infants</td>
</tr>
<tr>
<td>Halothane</td>
<td>0.87</td>
<td>1.2</td>
</tr>
<tr>
<td>Isoflurane</td>
<td>1.6</td>
<td>1.86</td>
</tr>
<tr>
<td>Sevoflurane</td>
<td>3.3</td>
<td>3.3</td>
</tr>
<tr>
<td>Desflurane</td>
<td>9.2</td>
<td>9.92</td>
</tr>
</tbody>
</table>
Nitrous Oxide

Nitrous oxide (MAC 104% at sea level, in adults) is commonly used as a “carrier agent” to facilitate induction and intubation of children under deep volatile anesthesia. It reduces cardiovascular side effects of some volatile anesthetic agents, such as halothane by decreasing their MAC during induction. Nitrous oxide is a powerful analgesic and provides excellent analgesia for short painful procedures. Analgesic effect of nitrous oxide is especially useful when opioids are either not justified or contraindicated for pain management in children undergoing painful procedures. Nitrous oxide tends to diffuse into the closed spaces and may be relatively contraindicated in endoscopic procedures, gastrointestinal surgery, in middle ear and some ocular procedures, and in children with emphysema. N₂O is less emetogenic in children.

Halothane

Halothane is less commonly used even in third world countries nowadays because of its poor cardiovascular profile. Halothane blunts baroreceptor reflex response and thereby makes neonates and infants vulnerable to direct cardiorespiratory depressant actions of anesthetics. Halothane depresses myocardial contractility in children more than isoflurane and sevoflurane. Benign arrhythmias (such as PVC’s, nodal rhythm, bigemini, as well as supraventricular arrhythmias, etc.) occur frequently, especially in spontaneously breathing children and in presence of increased plasma catecholamines and respond well to deepening of anesthesia and correction of hypercarbia. Halothane decreases heart rate, particularly markedly in neonates, and may cause junctional rhythm, bradycardia and even cardiac asystole. Halothane-induced bradycardia needs early correction with intravenous atropine (10 µg/kg). Rapid rise in alveolar concentration because of its relatively reduced solubility and high potency may produce unintended cardiorespiratory instability in neonates and infants during induction, especially if inspiratory concentration is raised in larger increments and if ventilation is controlled rather than allowing spontaneous ventilation. Control of depth of anesthesia by changing inspiratory concentration (i.e. by changing vaporiser dial setting) is poorer with halothane because of its slower “wash-out” due to higher solubility. MAC of halothane is highest during first six months of age (maximum 1.2%) and comes down by 30% in full term neonates. Unlike in adults, halothane causes less sensitisation of myocardium to catecholamines in children in the presence of hypercarbia. Consequently, during halothane anesthesia, low dose epinephrine (10 µg/kg) can be safely administered to children who otherwise have a normal cardiovascular system. Hepatotoxicity following halothane occurs much less frequently in children and current evidence is not enough to disapprove repeat anesthetic with halothane.

Isoflurane

Isoflurane is commonly used only for maintenance of anesthesia in children. It is pungent and causes irritation of the respiratory tract. However, following induction with other agents children tolerate addition of isoflurane and emerge from it smoothly during recovery. Lesser blood solubility permits better control of depth of anesthesia by changing (increasing or decreasing) inspiratory concentration. Fall in alveolar concentration and thus recovery from isoflurane anesthesia is faster than halothane. The MAC of isoflurane varies with age and is highest in infants aged between 1 and 6 months (1.86%). In preterm newborns MAC is 10–12% lower than it is in term neonates. Sudden increase in the inspired concentration of isoflurane does not cause tachycardia and hypertension in children because of central sympathetic response as is seen in adults. Cardiac output is better preserved during isoflurane than during halothane anesthesia. In children with severe liver disease isoflurane is the anesthetic of choice as it maintains total hepatic perfusion better than halothane and only a little amount (0.2–0.3%) is metabolized in the liver. During isoflurane anesthesia, cerebral blood flow velocity (CBFV) varies proportionately with end tidal carbon dioxide.

Sevoflurane

Sevoflurane has a pleasant odor, does not irritate the respiratory tract and has little effect on hemodynamics.
Sevoflurane’s low blood solubility causes rapid wash-in consistent with rapid induction and faster recovery from anesthesia in children. Anesthetic depth can be controlled more easily. MAC is highest (3.3%) and alike in both neonates and infants up to the age of 6 months (2.5 for older infants and children). This very high MAC value more than offsets the reduced solubility of sevoflurane in neonates and infants. Therefore, excessive concentration of sevoflurane cannot be administered to neonates and infants with current vaporizer design which is advantageous as anesthetic overdose is very unlikely to occur during induction. However, inability to deliver large concentration causes difficulty in rapidly inducing deep level of anesthesia with sevoflurane in neonates and infants compared to older children. Sevoflurane induction requires rapidly increasing the inspired concentration from 0 to 8% following administration of 70% nitrous oxide in oxygen for 1–2 minutes in spontaneously breathing children. Ventilation should not be controlled with higher inspired concentration, as in during induction, as it may produce cardiovascular instability especially in neonates and infants. Attaining a deeper state of anesthesia with sevoflurane is difficult and IV anesthetic may be required. Sevoflurane produces mild cardiovascular depression. Cardiac arrhythmias are uncommon. Compared to halothane, sevoflurane causes more respiratory depression and upper airway narrowing in spontaneously breathing children. Incidence of emergence delirium during recovery is higher than with halothane.

Desflurane

Desflurane is least soluble (blood/gas solubility 0.42) of all modern volatile anesthetics. Uptake from lungs is minimal and alveolar concentration rises very rapidly to near inspiratory concentration. Induction of anesthesia is faster and precise control of depth of anaesthesia is easy. However, induction in children is associated with higher incidences of coughing, breath holding and laryngospasm as desflurane is more pungent than sevoflurane and irritates the airway. Thus, desflurane is not recommended for induction of anesthesia in children. Of all volatile anesthetics, desflurane is least potent with a MAC between 8% and 10% in infants. Desflurane MAC is highest (9.92%) in infants between 6 months and 1 year. It is minimally metabolized (0.02%) in the body. Desflurane rarely causes arrhythmias and does not alter cardiovascular variables at 1 MAC. It exaggerates airway narrowing markedly in susceptible children. Recovery from desflurane is faster than sevoflurane but is associated with higher incidences of emergence delirium than with sevoflurane and halothane. Desflurane anesthesia may be beneficial in neonates as it does not cause post-anesthetic apnea, particularly in preterm neonates and infants.

Intravenous Anesthetic Agents

Propofol

Propofol, an alkylphenol, is widely used for both induction and maintenance of general anesthesia in children. It is also used in children undergoing brief radiological as well as medical procedures. Because of larger volume of distribution children need higher doses to achieve the same plasma concentration as adults. Recommended dose for induction of anesthesia in infants and children is 2.5–3.5 mg/kg over 20–30 seconds. Children may require larger doses (4–5 mg/kg) for acceptance of face mask. More rapid injection may produce sudden hypotension. Hypovolemia, sedative premedication and induction with sevoflurane and nitrous oxide reduce the propofol dose requirement for induction of anesthesia. More than 5 mg/kg (4.7–6.8 mg/kg) of propofol is necessary for successful LMA insertion when opioid is not coadministered. Infusion dose requirement to prevent anesthetic apnea, particularly in preterm neonates and infants who probably require lower doses. Neonates and infants have lower clearance of propofol because of reduced hepatic metabolism. Intermittent bolus or continuous infusion may lead to propofol accumulation and delayed awakening from anesthesia. Initial propofol dose for continuous infusion (Table 3) during short radiological or medical procedures is 200–250 µg/kg/minute (12–15 mg/kg/hour). Infusion dose requirement to prevent movement during procedure is higher for younger children and for children having cognitive impairment. For ICU sedation a lower initial dose (1–4 mg/kg/hour) should be administered which can be titrated later to the desired response. Pain with IV injection is a major disadvantage and is best reduced by pretreatment with...
intravenous lidocaine (0.5–1.0 mg/kg) after applying tourniquet proximal to the site of injection 30 seconds prior to administration of propofol. Propofol decreases mean arterial pressure by 20–30% from baseline and causes apnea in up to 40% unpremedicated children following induction. Anaphylaxis to propofol itself is rare. Propofol causes dose dependent bronchodilatation and is beneficial in asthmatic children. It depresses laryngeal and pharyngeal reflexes and can be used at a dose of 1–2 mg/kg bolus to avert cough and to successfully break laryngospasm. Propofol also possesses useful antiemetic properties. Prolonged sedation with propofol at a rate of more than 5 mg/kg/h (70 µg/kg/min) for more than 48 hours may produce propofol infusion syndrome (PRIS) manifested as insidious onset of lipemia, metabolic acidosis, hyperkalemia and rhabdomyolysis. This may result in profound myocardial instability and cardiovascular collapse and does not usually respond even to advanced resuscitative efforts.

**Thiopentone**

Thiopentone is a short-acting barbiturate with sedative hypnotic and anticonvulsant properties. It is beneficial when cardiovascular stability is necessary as it maintains SVR and causes less reduction in MAP than propofol. Unlike propofol, IV injection of thiopentone is painless. Induction of anesthesia is smooth and occurs in one arm brain circulation time. Awakening from anesthesia is quiet although occasionally interrupted by shivering. Thiopentone induction dose is inversely related to the age. Because of reduced protein binding neonates require a lower induction dose (3–5 mg/kg) of thiopentone. Infants have higher volume of distribution and require higher doses (6–8 mg/kg) than older children (4-6 mg/kg). Elimination is slow in neonates because of reduced metabolism by immature hepatic enzyme systems. Thiopentone may accumulate following repeated injection or continuous infusion because of its low hepatic extraction ratio (0.3) and thus may cause prolonged sedation. Acute tolerance to thiopentone may also occur in children. Recovery of psychomotor skills is delayed following thiopentone induction than following propofol. Thiopentone does not suppress laryngeal and pharyngeal reflexes. It causes direct myocardial depression and mild peripheral vasodilatation and may precipitate hypotension in hypovolemic children. Thiopentone is absolutely contraindicated in presence of variegate (South African genetic porphyria) as well as acute intermittent porphyria. To safeguard from life-threatening complications, however, thiopentone is best avoided in presence of all kinds porphyria in children.

**KEY POINT**

Thiopentone should not be used in children coming for day care surgery.

**Ketamine**

Ketamine, phencyclidine derivative, produces dissociative anesthesia and is an excellent analgesic and amnesic. After IV (Table 4) administration, onset of anesthesia occurs within 30 seconds and duration of action persists for 5–8 minutes. However, analgesic effects of bolus ketamine is more prolonged and may prevail for as long as 4 hours. Top-up doses (0.5–1.0 mg/kg) may be administered as and when necessary. Recovery is relatively slow and is associated with higher incidences of nausea and vomiting and emergence reactions. Coadministration of an antisialagogue is necessary to prevent excessive airway secretion and thereby reduce the consequent risk of laryngospasm. Ketamine in low doses (1–2 mg/kg IV or 3–5 mg/kg IM) is frequently used for short procedures outside the operation theatre because of relative preservation of airway tone. Coughing and airway obstruction may occur even with low doses especially when combined with other sedating medications. Heart rate increases and so does cardiac output and systemic blood pressure. Ketamine maintains peripheral vascular resistance and pulmonary to systemic blood flow ratios and has little effect on shunting magnitude or direction in children with cyanotic heart disease. It is beneficial for induction of anesthesia in hypovolemic children or in children with congenital cyanotic heart disease. Ketamine is a significant bronchodilator and has been an induction agent of choice in children with severe bronchial asthma. Intramuscular Ketamine (3–5 mg/kg) is useful as a premedicant or preinduction agent for

**Table 3:** Continuous infusion scheme for propofol

<table>
<thead>
<tr>
<th>Infusion rate (mg/kg/h)</th>
<th>Time</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0–15 min</td>
</tr>
<tr>
<td>Propofol</td>
<td>15</td>
</tr>
</tbody>
</table>

**Table 4:** Doses of ketamine through various routes

<table>
<thead>
<tr>
<th>Type</th>
<th>Oral (mg/kg)</th>
<th>IM (mg/kg)</th>
<th>IV (mg/kg)</th>
<th>Epidural/ Caudal (mg/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Premedication</td>
<td>5</td>
<td>3–5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Short procedure</td>
<td>3–5</td>
<td>1–2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Induction</td>
<td>5–10</td>
<td>1–3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>As adjuvant</td>
<td></td>
<td></td>
<td></td>
<td>1 or 0.5 (S (+) ketamine)</td>
</tr>
</tbody>
</table>

35
children who are developmentally delayed or those who are too scared to be separated from parents or to come to the operating theatre. Other uses of ketamine include as oral premedicant (5 mg/kg), as an adjunct to local anesthetic (1 mg/kg or 0.5 mg/kg, preservative free S (+) ketamine) to increase duration of epidural block, or as an adjunct to opioids for postoperative pain relief. Ketamine increases ICP and IOP and thus should not be used in children with intracranial hypertension and perforating eye injury. Ketamine is associated with higher incidences of laryngospasm in children with URTI. Ketamine has psychotropic and epileptogenic effects and hence should be avoided in children with psychiatric or seizure disorders.

**Etomidate**

Etomidate is a rapidly acting carboxylated imidazole derivative with a short duration of action. In children, induction occurs within 20–30 seconds after IV injection (0.2–0.3 mg/kg) and action lasts for 2–6 minutes. Etomidate is highly protein bound and has a longer terminal half-life (4.6 hours). Induction dose is 30% higher in children compared to adults because of higher volume of distribution. IV etomidate is painful and is better administered through large veins. Induction is frequently associated with involuntary muscle movement, increased muscle tone and myoclonus. On the other hand, etomidate has minimal cardiovascular and respiratory effects and is suitable for induction in children with poor cardiac reserve. In children with traumatic brain injury induction with etomidate reduces intracranial pressure, improves cerebral perfusion and decreases cerebral oxygen consumption. Because of its better hemodynamic profile etomidate is also frequently used in emergency department for rapid sequence intubation in children. However, etomidate is known to suppress adrenal function and inhibit cortisol synthesis. Even a single bolus injection may block cortisol production or negatively influence adrenal function in critically ill children for at least 24 hours.

**Clonidine**

Clonidine, a mixed alpha-1 and alpha-2 adrenoceptor agonist with predominant alpha-2 actions has dose dependent sedative, anxiolytic and analgesic effects. Oral clonidine (3–5 µg/kg) is used as a premedicant in children. Orally administered clonidine is absorbed slowly and peak effects occur only after 60–90 minutes. Smooth induction of anesthesia under sedation (“steal induction”) is possible in most of the children after oral premedication. Clonidine premedication reduces MAC, prolongs postoperative analgesia, and attenuates surgical stress responses in children. Clearance of clonidine in neonates is about one-third of that described in adults. Intravenous clonidine (1–2 µg/kg) is used in children to prevent or treat emergence dysphoria. Clonidine is also used as an adjunct to local anesthetics to prolong block duration and decrease analgesic requirement. Low dose clonidine (1–2 µg/kg) does not significantly reduce heart rate and blood pressure in children and prophylactic atropine is probably not necessary.

**Sedatives**

**Midazolam**

Oral midazolam (0.3–0.5 mg/kg, maximum 20 mg) is commonly used as premedicant in children to reduce separation anxiety and distress at induction. Peak plasma level is achieved after 30–60 minutes. Maximum sedation and anxiolysis after intranasal administration (0.1–0.2 mg/kg) is obtained at 20 minutes. However, nasal midazolam causes nasal mucosal irritation and has a long lasting bitter after taste and is better avoided. IV midazolam (0.1–0.2 mg/kg, up to 0.5 mg/kg) is used for sedation of ventilated children in ICU. Bolus midazolam injection in neonates and infants may cause profound hypotension particularly if the infant is receiving fentanyl. Neonates and infants are susceptible to have prolonged sedation because of reduced hepatic metabolism and clearance than older children. Older children have higher clearance and require higher sedating doses. Intravenous midazolam reduces hypoxic ventilatory response and may cause respiratory arrest. It reduces pharyngeal muscular tone and may cause upper airway obstruction, especially in children with obstructive sleep apnea.

**Dexmedetomidine**

Dexmedetomidine, a selective α-2 adrenergic agonist, has 8-fold higher affinity for α-2 compared to α-1 adrenergic
receptors. Predominant α2 effects of dexmedetomidine may cause hypotension and bradycardia in children in a dose dependent manner apart from its primary effects of sedation and anxiolysis.116,117 High dose dexmedetomidine may produce hypertension especially in infants and children who require top-up bolus doses to prolong sedation.118,119,120 It is gaining popularity as a sedative agent analgesia and decreases opioid requirement during surgery.118,119,120 It is gaining popularity as a sedative agent for ICU patients, and for procedures outside the operation room in children mainly because of its minimal effects on respiration and a shorter half-life than clonidine. Even though change in airway diameter is minimal and possibility of airway obstruction is very unlikely, measures to rescue compromised airway during dexmedetomidine sedation is mandatory.122 A loading dose of 1 µg/kg intravenously over 10 minutes is followed by infusion at 0.5–1 µg/kg/hour. Recovery profile following dexmedetomidine infusion is slower compared to propofol for sedation during MRI.122,123 Dexmedetomidine sedation provides definite advantages for awake procedures (such as awake fiberoptic intubation as well as for awake craniotomy) as it permits arousal with gentle stimulation.14,124 Dexmedetomidine has also been found to be useful in reducing the incidence of emergence delirium.126

**KEY POINT**

Dexmedetomidine induced bradycardia and hypotension should be managed by decreasing the infusion dose and administering a bolus of balanced salt solution, if necessary

**Opioids**

**Morphine**

Intravenous morphine, commonly in the form of continuous infusion (0.05–0.2 mg/kg/hour), is most frequently used to manage acute postoperative pain in children.127 PCA device is used to administer morphine intravenously commonly in children older than five years of age.128 As gestational and postnatal age increase, postoperative morphine requirement also increases.129,130 Critically ill children receiving supplemental analgesics and hypnotics, and/or who experience fall in hemoglobin saturation at night of obstructive sleep apnea should receive lower doses.131,132 Morphine can also be administered orally (as short or long acting formulations), rectally and as intermittent IV bolus. Because of first pass hepatic metabolism oral bioavailability of morphine is only approximately 35%.9 As adjuvant to local anesthetics, or even sometimes alone, morphine can also be administered into epidural or caudal space either as a single bolus or as continuous infusion.127 Clearance of morphine is age dependent, being lowest in preterm infants and highest in young children. Critically ill children also have reduced clearance of morphine. Respiratory depression is the most important unwanted effect of all opioids in infants and children. Morphine causes respiratory depression by diminishing respiratory rate and tidal volume. However, at reduced infusion rates (10–30 µg/kg/h) morphine renders adequate postoperative analgesia and does not induce respiratory depression.133 Rapid IV administration of morphine may result in systemic hypotension because of histamine release and consequent vasodilation.134 Morphine-induced vomiting in postoperative children is dose dependent. Higher incidence (>50%) of vomiting occurs in children who receive more than 0.1 mg/kg of morphine.135,136

**Fentanyl**

Fentanyl is commonly used as perioperative analgesic in infants and children during general anesthesia. Perioperative fentanyl (1–3 µg/kg IV) blunts surgical stress response and provides better hemodynamic stability than morphine.137 High dose fentanyl (10–100 µg/kg IV) maintains cardiovascular stability and is often administered as a sole anesthetic in high risk neonatal and in pediatric cardiac surgical procedures.9 A continuous infusion (0.5–2.5 µg/kg/h) is frequently used in ICU setting to provide sedation and analgesia in mechanically ventilated infants and children.138 Clinical effects of fentanyl last shorter because of its redistribution and rapid hepatic clearance. Fentanyl clearance is greater in older infants and children than in adults but is prolonged in neonates and in children with congenital cyanotic heart disease.138,139 Respiratory depression is less in neonates after doses as high as 10 µg/kg because of increased volume of distribution.29 Rapid bolus injection of fentanyl may produce chest wall and glottic rigidity and bradycardia due to increased vagal tone.141,142 These can be reversed by administration of naloxone or using a muscle relaxant and control ventilating the child.142 Fentanyl may sometimes depress the baroreceptor reflex control of heart rate in newborns.143 Bolus administration of midazolam and fentanyl together may produce severe hypotension.81 Oral transmucosal fentanyl (OTF) was previously used for premedication in children. However, this product is no longer marketed in US.137 Fentanyl patch provides extended release of fentanyl and is used in children who require long-term fentanyl to combat chronic pain but are either unable to take orally or are noncompliant to oral medications. For this, fentanyl transdermal therapeutic system (TTS) has been developed that releases fentanyl at a lower dose (12.5 µg/h) required to control cancer pain in children.144 Fentanyl is also used as an adjuvant to local...
anesthetics for supplementation of caudal or epidural analgesia.

**Remifentanil**

Remifentanil is a highly potent synthetic opioid with rapid onset of action. Remifentanil provides intense analgesia and anesthesia with cardiovascular stability. It is particularly beneficial for sick neonates and infants because of faster clearance than older children. Remifentanil has a very brief elimination half-life (3–6 minutes) and clinical effects disappear within 10 minutes of intravenous injection. For prolonged analgesia, a loading dose of 0.1–0.25 µg/kg is followed by a continuous infusion 0.25–0.50 µg/kg/min. Remifentanil propofol combination can be effectively used to provide anesthesia in children for a variety of short surgical and nonsurgical procedures. Bolus remifentanil (3 µg/kg) can also be used to supplement propofol (4 mg/kg) to facilitate endotracheal intubation without the use of a muscle relaxant. Initial bolus dose of remifentanil may be associated with bradycardia and hypotension because of direct negative chronotropic action. Respiratory depressant effect of remifentanil is less in younger children.

**Buprenorphine**

Buprenorphine is a partial µ agonist, 25–50 times more potent than morphine. It is highly protein bound and cleared from plasma mostly by hepatic extraction and extensive first pass hepatic metabolism. Decreased hepatic blood flow prolongs duration of action of buprenorphine. After a single bolus dose buprenorphine clearance is higher in children than in adults. Buprenorphine produces similar analgesic and CNS effects as morphine. SUBLINGUAL buprenorphine is rapidly absorbed and produces clinical effects in 30–60 minutes and has been used as a premedicant in children. Intravascular buprenorphine produces equivalent but longer postoperative analgesia compared to morphine. However, respiratory depressant effect of parenteral buprenorphine is greater and longer lasting compared to morphine. Other adverse effects, such as sedation, nausea and vomiting, dizziness, and sweating, are similar to those produced by other morphine like opioids. Caudal buprenorphine (2.5–4 µg/kg), either alone or combined with caudal bupivacaine provides postoperative analgesia up to 24 hours in children without any major opioid-related side effects except higher incidence of nausea and vomiting.

**Tramadol**

Tramadol is a weak opioid and is effective against moderate to severe pain in children. It is used as peroperative analgesic (1–2 mg/kg) in children and to treat postoperative shivering. Clearance in children is similar to that in adults. Compared to other opioids, tramadol causes lower incidence of respiratory depression and similar incidence of other opioid-related unwanted effects, such as nausea, vomiting, pruritus, etc. Rapid IV injection may result in flushing, sweating, dizziness and nausea. Tramadol lowers seizure threshold. Coadministration with 5-HT3 antagonist results in reduced efficacy of tramadol. Oral route is useful as a transition after IV administration. Initial oral dose of 2–3 mg/kg is followed by 1–2 mg/kg 4–6 hourly. Tramadol has also been used caudally and epidurally as adjuvant to local anesthetics to prolong postoperative analgesia in children.

**Non-Opioid Analgesics**

**Ketorolac**

Ketorolac is useful as an adjuvant to prolonged postoperative pain management in children, mostly as a transition from IV (0.2–0.6 mg/kg stat followed by 0.2–0.4 mg/kg 4–6 hourly for 5 days) to oral route (0.2 mg/kg 4–6 hourly). Ketorolac suppresses platelet function and may increase surgical bleeding. Increased incidence of bleeding is seen especially in children undergoing adenotonsillectomy and particularly when administered during or at the beginning of the surgical procedure before hemostasis is achieved. Ketorolac is safe in preterm and term infants and can also be safely used to treat postoperative pain in children undergoing cardiac surgery. Ketorolac is also safe for orthopedic procedures except spinal fusion.
Acetaminophen (Paracetamol)

Acetaminophen has opioid sparing effects. Oral acetaminophen (20–40 mg/kg) administered before induction is rapidly absorbed and produces effective therapeutic blood level at the time of emergence even after very brief surgical procedure. Absorption via rectal route (40 mg/kg) is unpredictable and time delays of 60 minutes or so has been observed before peak clinical effects occur after achieving peak plasma concentration. For long duration surgical procedures rectal administration at the beginning is helpful to provide necessary blood concentration at emergence until the child is ready for oral medication. The superiority of IV route over oral and rectal routes has not been proven. However, IV paracetamol is being increasingly used in neonates. The recommended dose is 7.5 mg/kg/6 hour for neonates after 10 days of age through to infants of 10 kg and a dose of 15 mg/kg/6 hour (max daily dose 60 mg/kg) is recommended for infants and children between 10 and 40 kg. Neonates have larger volume of distribution and smaller clearance. Hepatotoxicity, even though rare, occurs commonly because of overdose. IV paracetamol is painful. However, amongst two IV paracetamol formulations (i.e. acetaminophen and propacetamol) acetaminophen is less painful and should be administered over a period of 15 minutes. IV paracetamol is expensive and should be restricted to use in infants and children who do not tolerate enteral or rectal preparations.

Diclofenac

Diclofenac is effective for treatment of acute pain in children and can be used as a component of balanced analgesic regimen perioperatively in pediatric anesthesia practice. It is readily absorbed via oral, rectal and intramuscular routes. Diclofenac is highly bound to plasma proteins (mostly albumin, >99.5%), has a smaller volume of distribution and undergoes extensive first pass hepatic metabolism. Intramuscular route should be avoided as it produces long-term muscular pain. Timing of administration via oral or rectal route is important for optimal therapeutic effects during surgical procedure or at the time of awakening from anesthesia. When administered before surgery, diclofenac significantly reduces the number of children requiring rescue pain medications post operatively. Diclofenac is associated with less postoperative nausea and vomiting probably because of its better analgesic effects that reduces necessity of other emetogenic rescue analgesics. Incidence of serious adverse effects, such as GI bleeding and allergic-type reactions in children following administration of diclofenac is low (<3/1,000 children). Therapeutic doses of diclofenac do not inhibit platelet aggregation and thus is not associated with increased perioperative bleeding that requires surgical intervention. In asthmatic children single therapeutic dose of diclofenac has been shown to produce no significant incidence of bronchospasm. There have been no recommended optimum doses of diclofenac for acute pain in children. A 2009 Cochrane review revealed a range of 0.5–2.5 mg/kg (both oral as well as rectal) in the single dose being used by different researchers.

Neuromuscular Blocking Agents

Neuromuscular blocking agents are less frequently used in present day pediatric anesthesia practice. Indications for tracheal intubation are decreasing as in many cases airway is managed with supraglottic devices, such as LMA or i-Gel without using a muscle relaxant. A volatile agent when combined with propofol and an opioid (such as remifentanil) gives optimum intubating conditions and also provides sufficient muscle relaxation during surgical procedure and is frequently used in many cases instead of a neuromuscular blocking agent. Regular use of regional anesthesia has also reduced the necessity of neuromuscular blocking drugs in children under general anesthesia. However, in some cases use of muscle relaxant is necessary for smooth and rapid endotracheal intubation, to paralyse skeletal muscles to make surgical procedure easier or to facilitate mechanical ventilation in intensive care. Monitoring of neuromuscular function is essential as clinical clues of depth of neuromuscular blockade are absent during inhalational anesthesia until the child is fully awake afterwards. Dose requirement for neuromuscular blocking agents is dependent on required plasma concentrations and volume of distribution of these agents. These two factors are very much related to the age of the child and consequently
doses of different muscle relaxants also vary with the age of the patient. Neonates and young infants require lower plasma concentration and hence lower doses as they are more sensitive to nondepolarizing agents because of immature neuromuscular junction. Onset is early and actions persist for longer because of slow elimination of the drugs. Children require higher loading doses of muscle relaxants because of their higher volume of distribution (increased ECF) compared to adults. Newer neuromuscular blocking drugs have rapid onset and shorter duration of action in children because of their high cardiac output compared to adults. Onset times are prolonged in children with poor cardiac output or reduced muscle perfusion. Low body temperature and addition of volatile agents potentiate the action of neuromuscular blocking agents. Thus, smaller doses and less frequent administration of neuromuscular blocking agents are required when a volatile agent is used in children. Opioids, barbiturates and inhalation of nitrous oxide have only minimal effects on neuromuscular blocking agents.

### Succinylcholine

Succinylcholine is no longer used routinely in pediatric anesthesia practice primarily because of its potential for causing hyperkalemic cardiac arrest especially in children with undiagnosed muscular dystrophy. However, succinylcholine provides both rapid and ultra-short muscle relaxation and therefore is indicated for rapid sequence intubation, for brief procedures, and for treatment of laryngospasm in children. IV dose requirement to achieve appropriate intubating conditions in neonates and infants is higher (3 mg/kg) because of their increased volume of distribution than in older children and adolescents (2 mg/kg). IV dose of succinylcholine needed to treat laryngospasm is very small, 0.1-0.5 mg/kg IV. When IV access is not available IM succinylcholine (4–5 mg/kg) can be used to treat laryngospasm. Vocal cord paralysis is complete within 4 minutes and clinical effects persist for about 20 minutes following IM injection. Intraligual or sublingual (3 mg/kg) administration is also effective when IV line is not in place and provides faster onset than IM succinylcholine. High-dose rocuronium (1.2 mg/kg) provides equivalent intubating conditions as succinylcholine and can be used for rapid sequence intubation in children with full stomach who are also at risk of life-threatening complications of succinylcholine.

Apart from hyperkalemic cardiac arrest, succinylcholine has also been known to cause some other significant adverse effects. Second dose of succinylcholine may produce, albeit rarely, severe bradycardia or asystole and must be prevented by pre-treatment with IV atropine. Reduced cholinesterase activity may prolong duration of succinylcholine induced muscle paralysis. Children homozygote for atypical gene may have muscular paralysis for 6–8 hours. Increased jaw rigidity (“jaws of steel”) and inability to open mouth and intubate the trachea following administration of succinylcholine especially during halothane anesthesia occurs rarely and may be the premonitory sign of malignant hyperthermia. Muscle fasciculations are rare in infants and milder in older children while postoperative muscle pain is observed in adolescent and adults only. Succinylcholine has been shown to increase IOP only transiently and does not cause further ocular damage following its administration.

### Atracurium

Atracurium is frequently used for routine endotracheal intubation in children of all ages at an initial dose of 0.3–0.6 mg/kg. Children require more atracurium per kilogram than adults and generally recover faster. Satisfactory intubating conditions are achieved within 2 minutes. In neonates and in infants maximum neuromuscular blockade occurs quicker than in older children and adolescents. Clinical recovery is usually complete within 40 to 60 minutes. Atracurium is broken down spontaneously by “Hofmann elimination” and “ester hydrolysis”. Laudanosine, the major metabolite of atracurium is excreted freely in urine and bile and does not possess neuromuscular blocking properties.

Atracurium, therefore, can be safely administered by continuous infusion (Table 5). At clinical doses, when injected slowly, side effects of atracurium are minimal and less in children than in adults. However, rapid intravenous injections of higher doses may produce

### Table 5: The infusion requirement of atracurium to maintain 90 to 99% twitch depression in children

<table>
<thead>
<tr>
<th>Anesthesia technique</th>
<th>Dose of atracurium (µg/kg/minute)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Isoflurane anesthesia</td>
<td>6.0</td>
</tr>
<tr>
<td>Halothane anesthesia</td>
<td>7–8</td>
</tr>
<tr>
<td>N₂O:O₂:Opioid technique</td>
<td>9.0</td>
</tr>
</tbody>
</table>

**Key Point**

Succinylcholine should not be used in children with myopathy, recent burn or spinal cord injury.
Chapter 3: Essentials of Pharmacology in Neonates, Infants and Children

Cutaneous flushing reactions and hypotension because of release of histamine.181,197

**Cisatracurium**

Cisatracurium, a stereoisomer of atracurium, is three times more potent than atracurium with similar duration of action.224,225 Appropriate intubating conditions are achieved at a dose of 0.15–0.20 mg/kg.226,227 Cisatracurium has slightly slower onset of action and similar duration of clinical effects and recovery profiles as that of atracurium.9 Cisatracurium provides a more stable hemodynamic profile as it does not release histamine.197

**Vecuronium**

Vecuronium is devoid of any adverse cardiovascular effects even in large doses.228 Because of higher sensitivity to vecuronium neonates and infants require less initial bolus dose (0.07 mg/kg) compared to older children and adolescents (0.1 mg/kg).197 Onset of action of vecuronium is slower and is longer than twice that of succinylcholine.229 Infants require longer time for recovery of neuromuscular function compared to older children.230 Residual motor weakness persists after continuous infusion because of prolonged elimination half-life and therefore vecuronium is not indicated for mechanically ventilated children in ICU.231,232 Vecuronium is painful when injected through a small vein in children.9

**Rocuronium**

Rocuronium has lower potency and faster onset of action (60–90 seconds following 2×ED95 dose) compared to other intermediate acting non depolarising muscle relaxants.233,234 However, quick onset time of rocuronium is not as significant in older children as those in adults while in infants it is nearer to that after succinylcholine.235 Tracheal intubation can be performed within 60 seconds following an intravenous dose of 0.6 mg/kg in all children.236,237 Increasing the dose of rocuronium improves the intubating conditions at 60 seconds and at 1.2 mg/kg dose intubating conditions are similar to that after succinylcholine.238-240 At a dose of 0.3 mg/kg effective intubating conditions are achieved within 2 to 3 minutes in children under sevoflurane anesthesia and can be used for short cases satisfactorily.241 Neonates are more sensitive to rocuronium. They require lower intubating dose (0.45 mg/kg) and have prolonged clinical effects (1 hour).242 Rocuronium behaves like a long acting agent in infants with prolonged duration of action because of lower plasma clearance, increased volume of distribution and reduced required plasma concentration.233,197 Sevoflurane anaesthesia significantly decreases onset time and increases the duration of action of rocuronium.243 Rocuronium is primarily eliminated by liver and kidney and clinical duration of action after 0.6 mg/kg dose lasts for 21 to 29 minutes. Large doses of rocuronium cause increase in heart rate and blood pressure. Rapid injection is associated with local pain and can be reduced by pretreatment with remifentanil, alfentanil or low dose ketamine.244-246

**Mivacurium**

Mivacurium has a very short duration of action and similar onset time as atracurium. Because of short elimination half-life and fast receptor de-occupancy recovery of clinical effects is rapid irrespective of its dose and duration of administration and does not require pharmacological reversal.197 Clinical effect lasts for 7 to 9 minutes in infants and children after a 0.2–0.3 mg/kg dose and complete recovery occurs within 14 to 19 minutes. Spontaneous and rapid recovery of neuromuscular function makes mivacurium ideal for short surgical cases (day care cases) when tracheal intubation is necessary.247,248 However, mivacurium is not often used in children as tracheal intubation even for very brief surgical procedures can be performed with low dose atracurium combined with volatile anesthetic or with combination of opioid and propofol.29 Mivacurium-induced neuromuscular block can persist significantly longer in children with reduced level of plasma cholinesterase or in the presence of an atypical plasma cholinesterase.197 More than 0.25 mg/kg rapid bolus injection of mivacurium may release large amount of histamine.249,250

**Reversal of Neuromuscular Blockade**

Reversal of neuromuscular blockade is necessary in neonates and infants because neonates remain at a greater risk of residual neuromuscular paralysis while elimination of neuromuscular blocking drugs could be delayed in infants.197 Older children receiving intermediate acting muscle relaxant, particularly atracurium or cisatracurium, may not require neuromuscular blocking reversal depending upon TOF responses (objective

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**KEY POINT**

Advantages of cisatracurium over atracurium are increased potency, and lack of dose-related histamine release.

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**KEY POINT**

High-dose rocuronium (1.2 mg/kg) provides equivalent intubating conditions as succinylcholine and can be used for rapid sequence intubation in children.114
measures of residual neuromuscular blockade) especially if sufficient time has elapsed since the last top-up dose. However, the rate of NMB blockade monitoring among practitioners is not satisfactory and residual paralysis, in most cases, is highly possible at extubation. Thus, in the absence of neuromuscular monitoring, it seems prudent to antagonise neuromuscular blockade in all children receiving intermediate or long acting muscle relaxant either by bolus or by continuous infusion.

Neostigmine

Neostigmine is recommended for routine antagonism of neuromuscular blockade in children as it provides greater final recovery of neuromuscular function than edrophonium. Infants and children require smaller doses of neostigmine than adults. Pre- or simultaneous administration of atropine or glycopyrrolate is necessary to minimize the muscarinic effects of neostigmine. 30 to 50 µg/kg of neostigmine (repeated if required up to a total dose of 70 µg/kg) combined with 10–20 µg/kg of atropine or 5–10 µg/kg of glycopyrrolate is usually sufficient for all pediatric age groups to completely reverse the neuromuscular blockade. Peak effects of neostigmine are reached in between 5 to 10 minutes after IV administration. Plasma clearance is highest in infants and decreases as age increases.

Sugammadex

Sugammadex encapsulates (chelates) rocuronium, vecuronium and pancuronium and removes them from their site of action at neuromuscular junction. The newly formed complex does not have any pharmacological effects and is excreted in urine unchanged. Sugammadex strongly binds to rocuronium than to vecuronium and pancuronium. However, literature relating the use of sugammadex in children is scarce and safety of its use in this age group of patients is yet to be confirmed. A dose of 2 mg/kg has been shown in one study to reverse moderate neuromuscular blockade produced by rocuronium in children and adolescents.

ANTIEMETICS

Children who are at high-risk for postoperative nausea and vomiting (PONV) should receive prophylactic antiemetic therapy along with measures to reduce the baseline risks. Antiemetic medications are necessary when symptoms persist in established post-operative nausea and vomiting. Serotonin receptor (5-HT<sub>3</sub>) antagonists, dexamethasone and metoclopramide are most commonly used in pediatric anesthesia practice.

Ondansetron

Among all 5-HT<sub>3</sub> antagonists ondansetron is most commonly used in children. Adverse effects of ondansetron are rare and clinically unimportant. A dose of 0.10–0.15 mg/kg of ondansetron intravenously is highly effective for both prevention as well as a first line treatment of established PONV. Oral route is as effective as intravenous route and oral ondansetron (4 mg) can be used in older children (weighing >20 kg) when IV line is not available. Combination therapy with a second agent, usually dexamethasone, may improve its efficacy in treating established PONV in children. Ondansetron is more effective than droperidol or metoclopramide and equally effective as dexamethasone for early PONV.

Dexamethasone

Dexamethasone is particularly effective in preventing late PONV (>6 hr). A single dose of 150 µg/kg provides good reduction in PONV with no adverse effects. Dexamethasone (150 µg/kg) combined with ondansetron (50 µg/kg) is highly effective and is recommended for situations at high-risk of PONV or where single agent therapy has failed previously. In susceptible children, particularly in children after receiving cytotoxic drugs for hematological malignancies, dexamethasone can induce tumor lysis syndrome.

Metoclopramide

Metoclopramide is less effective than 5-HT<sub>3</sub> antagonist and dexamethasone. Its clinical efficacy has been inconsistent. However, it may be used as a rescue antiemetic in established PONV at a dose of 0.15 mg/kg IV. Metoclopramide is associated with higher incidence of extrapyramidal syndromes, especially in adolescent girls.

KEY POINT

Drug from a different class should be used for failed antiemetic prophylaxis

ANTICHLINERGICS

The use of anticholinergics is declining in pediatric anesthesia practice for several reasons. Intramuscular injection is painful and the optimal effects of anticholinergic drugs may not coincide with induction of anesthesia. At the same time induction with modern volatile agents such as halothane and sevoflurane is not associated with troublesome secretions, succinylcholine for endotracheal intubation is less often used and
Atropine is never used to treat bradycardia associated with hypoxia. Furthermore, dry and friable mucosa resulting from administration of anticholinergics causes discomfort and reduces effective mucociliary function for several hours in children. Also, atropine is notorious for hyperthermia and decreases lower esophageal sphincter tone. And lastly, better sedatives instead of scopolamine such as midazolam, clonidine and dexmedetomidine etc. are widely available and are safer with less prominent adverse effects when judiciously used in children. Therefore, anticholinergic drugs should be used in children only if and when indicated.

Atropine (10–20 µg/kg, IV) is commonly used to prevent or treat perioperative drug induced (e.g. succinylcholine, halothane or neostigmine) bradycardia or bradycardia associated with oculocardiac reflex during some ophthalmic procedures. Atropine (10–20 µg/kg, IV, IM or SC) is also used as a premedicant to reduce secretions as well as to block laryngeal and vagal reflexes. Newborns and young infants up to the age of 6 months require higher doses. Intramuscular atropine is painful while intravenous atropine is more effective in blocking laryngeal reflexes. If IV access is absent, atropine (50–100 µg/kg) can be administered intratracheally as it is rapidly absorbed and promptly increases heart rate. Atropine should be very carefully used in children with narrow angle glaucoma and hyperthermia.

Glycopyrrolate is a potent anticholinergic and is commonly used to antagonise the parasympathetic side effects of neostigmine at a dose of 5–10 µg/kg. Anticholinergic effects of glycopyrrolate are more pronounced and prolonged than that of atropine. Glycopyrrolate is a synthetic quaternary ammonium compound and thus, unlike atropine and scopolamine, does not cross the blood brain barrier. Intravenous administration does not produce significant changes in heart rate or detrimental arrhythmias. Glycopyrrolate is equally effective as atropine in preventing oculocardiac reflex.

LOCAL ANESTHETICS

Amide local anesthetics (lidocaine, bupivacaine and its isomer levobupivacaine, and ropivacaine) are frequently used in children for local or regional blocks. In plasma, amide local anesthetics are mainly bound to serum α1-acid glycoprotein (AAGP) and human serum albumin (HSA). The AAGP concentration is low at birth and increases gradually in the first year of life and during childhood. Thus, neonatal plasma will have more free drug compared to older children or adults. Again, clearance of amide local anesthetics is poor in neonates and increases slightly during the first 6–9 months of life. Therefore, neonates and young infants are vulnerable to accumulation of local anesthetics following repeated injection or continuous infusion and consequently to excessive plasma concentration and systemic local anesthetic toxicity. Also, immature blood-brain barrier of neonates and young infants allows free local anesthetics to cross into the brain more easily.

Racemic bupivacaine is most commonly used in pediatric anesthesia practice. Newer long acting amides levobupivacaine and ropivacaine have a wider margin of safety compared with racemic bupivacaine and are gaining popularity. Racemic bupivacaine is highly protein bound and primarily metabolized in the liver. 0.25% solutions are most commonly used for peripheral nerve blocks while 0.0625 to 0.125% solutions are used for continuous epidural analgesia. After single injection analgesia persists for 4 hours in older children. Infants and neonates have shorter duration of analgesia because of increased volume of distribution.

Levobupivacaine has similar onset, efficacy and duration of blockade as the racemic bupivacaine but is less cardiotoxic and neurotoxic compared to racemic bupivacaine. Systemic toxicity occurs at a much higher blood concentration compared to racemic bupivacaine.

Ropivacaine is less lipophilic, half as potent and produces less cardiovascular and CNS toxicity compared to racemic bupivacaine. In children ropivacaine provides prolonged analgesia compared to bupivacaine even when a less potent solution is used. Evidence is not sufficient that ropivacaine produces only partial motor block at equianalgesic potency to other local anesthetics. Clearance is low in neonates and infants and increases during the first 2–6 years of life. In pediatric anesthesia practice a 0.2% solution containing 2 mg/mL is usually used. Volume required is similar to that of bupivacaine but mainly depends on the type of block and size/weight of the child.

Lidocaine has a relatively shorter duration of action and is not commonly used for single injection blocks in children.

The dose of local anesthetics (Table 6) is basically calculated based on lean body weight of the child. Various factors including pharmacokinetics of drugs, the age of the child and his/her physical status and the area to be anesthetized may play important role in determining...
blood level of free fraction of local anesthetics. Site of injection plays an important role in blood concentration of local anesthetics. More cephalad injections such as scalp infiltration and intercostal blocks and injection into highly perfused areas are associated with higher blood concentration in children. However, absorption and thereby blood concentration of local anesthetics can be reduced by addition of vasoconstricting agents such as epinephrine, which also prolongs the duration of analgesia. In children 10 µg/kg (maximum 250 µg) of epinephrine (1:200,000 or less) can be safely used. A conservative dosing approach of LA’s should help preventing overdose and systemic toxicity. When a large volume is required, a relatively dilute solution of local anesthetics should be used. Doses should be reduced by approximately 30% in infants younger than 6 months of age.

Systemic toxicity because of excessive blood concentration may occur following local or regional anesthesia in children. Racemic bupivacaine is more toxic compared to ropivacaine and levobupivacaine. Thus, to minimize the possibility of systemic toxicity levobupivacaine and ropivacaine instead of racemic bupivacaine should be routinely used for regional blocks in children. Cardiovascular and central nervous systems are mostly affected when blood concentration exceeds threshold level. CNS is more susceptible as LA readily crosses the blood brain barrier and produces CNS manifestations consistent with their plasma level before appearance of cardiovascular toxicity. Many a time, early symptoms are not apparent as most of the blocks in children are placed under general anesthesia or under deep sedation. Rapid heart rate in children and concomitant use of volatile anesthetic increase the risk of cardiac toxicity. Bupivacaine cardiotoxicity may also occur simultaneously with CNS toxicity in infants and children or may sometime even precede it because of bupivacaine’s lower threshold for cardiac toxicity. Primary cardiac manifestation is impaired ventricular conduction. Tachyarrhythmias and cardiovascular collapse may follow QRS widening, bradycardia, and torsades de pointes.

The specific treatment of LA toxicity is bolus administration of 20% lipid emulsion (Intralipid) 1.5 mL/kg over 1 minute, repeated every 3 to 5 minutes (up to maximum of 3 mL/kg) and then continued, if necessary, as IV infusion at a rate of 0.25 mL/kg until hemodynamic stability is achieved.

**ADJUVANTS**

Adjuvants should be routinely used in pediatric regional anesthesia. Adjuvant drugs, when used in combination, prolong the duration as well as improve the quality of blocks produced by long-acting local anesthetics. They also reduce the possibility of potential adverse effects of local anesthetics as lower concentration of these agents can then be used to provide effective regional blocks. The most commonly used adjuvant drugs in children are clonidine, ketamine, and opioids.

**Clonidine**

Clonidine (1–2 µg/kg) added to caudal or lumbar epidural local anesthetics (bupivacaine 0.25% or ropivacaine 0.2%) improves the duration and quality of postoperative analgesia and decreases analgesic requirement. Clonidine 1 µg/kg as a single shot caudal does not cause hypotension, bradycardia or undue sedation. Epidural or caudal clonidine should not be used in young infants (<3 months), particularly at a dose greater than 2 µg/kg, for they are prone to develop apnea. Clonidine also prolongs duration of peripheral blocks in children. IV clonidine has similar effects to that of epidural clonidine.

**Ketamine**

Preservative free ketamine (1.0 mg/kg of S (+) ketamine) is preferred to racemic ketamine (0.5 mg/kg) because it is less neurotoxic. Ketamine increases duration of analgesia of both bupivacaine (0.25%) as well as ropivacaine (0.2%). Psychotomimetic and behavioral symptoms are not rare after single shot epidural/caudal ketamine. Some authors do not recommend the use of neuraxial ketamine because of its potential for neurotoxicity. Also, drugs such as clonidine and opioids with fewer adverse effects than ketamine when administered as adjuvant to local anesthetics are available and should be considered before ketamine is administered neuraxially, especially in neonates and in small infants.

**Opioids**

Opioids are commonly used as adjuvants for postoperative pain management in infants and children. They prolong

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**Table 6: Local anesthetic doses and their duration of action.**

<table>
<thead>
<tr>
<th>Local anesthetic</th>
<th>Concentration used (for peripheral blockade) (%)</th>
<th>Maximum dose (mg/kg)</th>
<th>Duration of action (minutes)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bupivacaine</td>
<td>0.25</td>
<td>2.5</td>
<td>180–600</td>
</tr>
<tr>
<td>Ropivacaine</td>
<td>0.20</td>
<td>3.0</td>
<td>120–240</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>1.00</td>
<td>7.0</td>
<td>90–200</td>
</tr>
</tbody>
</table>
analgesia for up to 24 hours in children over 6–9 months.\textsuperscript{290} Epidural and subarachnoid opioids are however associated with higher incidence of urinary retention.\textsuperscript{9} Other adverse effects of opioids such as respiratory depression and pruritus are dose dependent.

Morphine is the most commonly used opioid as an adjuvant to local anesthetics. Both single dose epidural and caudal morphine provide excellent and prolonged postoperative analgesia.\textsuperscript{280,281} Preservative free morphine (25–30 µg/kg, bolus) spreads rostrally and can be placed at a lower segmental level.\textsuperscript{290} The bolus dose may be followed by continuous infusion of 1 µg/kg/hour. However, side effects such as nausea, urinary retention, pruritus, and respiratory depression are common and thus the role of opioids in neonates and infants is not clear.\textsuperscript{322,323}

Children having epidural morphine should be monitored in a high dependency area for at least 24 hours. Morphine should be administered in children undergoing only major surgery and avoided in day stay children. Naloxone in a high dependency area for at least 24 hours. Morphine is the most commonly used opioid as an adjuvant to local anesthetics. Both single dose epidural and caudal morphine provide excellent and prolonged postoperative analgesia.\textsuperscript{280,281} Preservative free morphine (25–30 µg/kg, bolus) spreads rostrally and can be placed at a lower segmental level.\textsuperscript{290} The bolus dose may be followed by continuous infusion of 1 µg/kg/hour. However, side effects such as nausea, urinary retention, pruritus, and respiratory depression are common and thus the role of opioids in neonates and infants is not clear.\textsuperscript{322,323}

Fentanyl has also been used as an adjuvant for continuous infusion along with local anesthetics (1–2 µg/mL of LA). Fentanyl (0.5–1.0 µg/kg) may prolong postoperative analgesia when coadministered with LA caudally.\textsuperscript{290} Fentanyl and sufentanil needs to be placed in the same segmental level where pain will occur.\textsuperscript{325}

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Understanding the Pediatric Chest Radiograph

INTRODUCTION
Chest radiography is a commonly performed basic investigation. Children should, however, not be subjected to unnecessary radiation. Nevertheless, in the presence of clinical symptoms and/or auscultatory findings, this simple, inexpensive investigation can provide valuable information.

IMAGING A CHILD
Chest radiograph is obtained during quiet inspiration in uncooperative infants and young children and during full inspiration in cooperative older pediatric patients.

- Optimized techniques of digital chest radiography in children are different from those in adults.
- Automatic exposure control and grids are not very useful in small pediatric patients.
- In most cases, kV is kept below 70.
- Use of an air gap between the child and the film to reduce scatter instead of a grid will significantly reduce radiation dose.
- Pediatric protocols limits the number of views and regions covered.
  - For most clinical situations, frontal view of chest alone is appropriate. Posteroanterior (PA) projection is preferable unless the child is uncooperative, when anteroposterior (AP) projection can be taken
  - Lateral projection may be taken to demonstrate:
    - Mediastinal lesion
    - Lesion in the lung base
    - Localization of a lesion seen on frontal view

- Supine AP projections are taken routinely for babies
- Horizontal beam projection is necessary to demonstrate hydropneumothorax, e.g. in the setting of intubated neonate with sudden deterioration because pleural fluid and pneumothoraces are difficult to detect on supine projection
- Expiratory views may be used to confirm air trapping due to an underlying airway obstruction
- Lateral decubitus view is used to:
  - Detect freely shifting pleural effusion or air
  - Demonstrate an air-fluid level in an intraparenchymal cavitatory lesion
  - Demonstrate air trapping in the dependent lung
- Oblique views may be helpful for evaluating:
  - Rib and soft tissue
  - Hilar, carinal, and peripherallung abnormalities

- Unnecessary radiation to structures such as the lower neck, proximal upper extremity, and upper abdomen should be avoided by using proper collimation and shielding.

Digital radiographic imaging system can achieve 85% dose reduction compared to conventional film screen systems to produce reproducible and high quality radiographs.

READING A CHEST RADIOGRAPH
A systematic approach is important in evaluation of a chest radiograph to ensure that important clues to the diagnosis are not overlooked.
• Check the patient’s name, date of examination and side marking.
• Note the projection (supine or erect, AP or PA), phase of respiration and presence of any rotation. Rotation is the most common cause for inequality in the translucency of the two lungs. Rotation also makes the hilum appear prominent on one side. It must be differentiated from air trapping. Rotation can be assessed by measuring the distance of medial borders of clavicles from line joining the spinous processes of cervical vertebrae. The clavicles must be equidistant from this line. If one of the clavicles is farther away from this line, it suggests rotation to that side.
• Check the phase of respiration. The diaphragms are projected over 5th to 7th rib ends in a well-inspired examination. An expiratory film will lead to exaggeration of heart size and prominence of bronchovascular markings which may be misdiagnosed as cardiac failure and bronchopneumonia. High position of diaphragm may obscure abnormalities of lung bases.
• Note the age of patient. There is gradual change in the appearance of chest radiograph from infancy to adulthood. In a baby, the shape is more triangular and deeper in the AP diameter. Air bronchograms may be normally seen projected through the cardiac shadow and should be considered abnormal when seen more peripherally. The costophrenic angles are shallower in infants compared to adults.
• Identify abnormal signs by reviewing all regions which are trachea, carina and major bronchi, mediastinal outlines, hilar regions, cardiac size and contour, pulmonary vascularity, size and translucency of lungs, position of major fissures, height of diaphragms, costophrenic angles, soft tissues and visualized bones. There are four hidden areas where lesions are commonly missed—lung apices, retrocardiac region, lung hila and below the domes of diaphragm.
• Suggest the possible differential diagnosis based on the radiological findings and clinical features.

NORMAL CHEST RADIOGRAPH

Structures Seen in a Normal Chest Radiograph

• Radiolucent trachea which branches at the carina to right and left main bronchi which can be traced as they branch further beyond the hila into the lungs. Normally, the trachea is placed centrally or mildly to the right. Deviation from the normal position may be due to rotation or due to a pathological process causing push or pull (Figs 1 and 2).

• The lung hila containing major bronchi and pulmonary vessels. Normally, the left bronchus is higher or at the same level as the right. Left hilum appearing lower than the right or marked asymmetry of the size or density of the two hila suggests some pathology.
• Lung fields are divided into upper, middle and lower zones, each roughly occupying one-third of the lung height for the purpose of description. These do not correspond to anatomical lung lobes. Each zone should be compared to its contralateral side to look for asymmetry and the abnormal side should be determined.
• Pleura are only visible when there is some abnormality such as pleural thickening, pleural effusion, pneumothorax, pleural calcifications or plaques.
• Lung lobes and fissures—the right lung is divided into three lobes, upper, middle and the lower by horizontal and oblique fissures and the left lobe into upper and lower by the oblique fissure. These are reflections of
the visceral pleura (Fig. 3). Occasionally, pathologies are limited by the fissure which can help to localize them to lung lobes rather than zones. Abnormal position of the fissure may be seen—upper zone fibrosis causing upward pull of horizontal fissure; or expansion of a lobe due to exudates causing bulging of the fissure, classically seen in klebsiella pneumonia.

- Costophrenic angles—angle formed by the dome of each hemidiaphragm with the chest wall. Normally, the costophrenic angles must be acute and pointed. Pleural fluid, pleural thickening or lung overexpansion causing downward push of the diaphragm cause blunting of these angles (Fig. 4).
- The hemidiaphragms are domed structures that divide the comparatively radiolucent lungs above and denser abdomen below. However, it should be remembered that the lung bases extend below the diaphragm and pathology here should not be missed. Normally, the right dome of diaphragm is higher than the left (Fig. 5).
  - Both domes may be elevated in case of ascites, pregnancy or abdominal lump
  - Both may be depressed in cases of emphysema
  - Unilateral elevation of diaphragm may be seen in conditions causing volume loss of lung like collapse and fibrosis or subphrenic collection or organomegaly
  - Unilateral depression of the diaphragm may result from pneumothorax or giant emphysematous bulla
- Heart size—cardiothoracic (CT) ratio = cardiac width/thoracic width (Fig. 6). The normal CT ratio is 1/2 in a posteroanterior chest radiograph. Ratio more than this suggests cardiomegaly.
- Cardiac contours—the right heart border is formed by SVC, right atrium and small part of IVC. The left heart border is formed by aortic knuckle, pulmonary conus, left atrial appendage and left ventricle (Fig. 7).
- Mediastinal contours—Thymus gives rise to prominent anterior mediastinal shadow in infancy which is quite variable in size and can be recognized by characteristic sail shape or wavy margins resulting from interdigitation of the soft thymic tissues in the intercostal spaces. It becomes
less evident between 2–8 years after which it cannot be seen on frontal radiograph. Sometimes the thymic shadow may be unusually large and can be confused with mediastinal pathology or area of lung consolidation. Lateral projection or other modalities like ultrasound, CT or MRI may be required for distinction (Fig. 8).

The Felson method of division of mediastinum is different from anatomical division. It is based on findings on a lateral chest radiograph (Fig. 9).
- A line extending from the diaphragm to the thoracic inlet along the back of the heart and anterior to the trachea separates the anterior and middle mediastinal compartments.
- A line that connects points 1 cm behind the anterior margins of the vertebral bodies separates the middle and posterior mediastinal compartments.
A line that connects points 1 cm behind the anterior margins of the vertebral bodies separates the middle and posterior mediastinal compartments.

- Soft tissue—any soft tissue lesion on the chest wall can give clue to the diagnosis and must be looked, e.g. neurofibromas.

- Bones—ribs, spine, clavicles, scapula must be looked for congenital deformities or other abnormalities in cases like thalassemia, rickets and scurvy.

**SIGNS IN A CHEST RADIOGRAPH**

**The Silhouette Sign**

An intrathoracic opacity if in anatomic contact with the heart or the aorta and of similar density, will obscure its border. The sign does not apply to lesions that differ in density to the structure they are in contact with (Fig. 10).

- For chest radiographs only
- Used for localization and not characterization of lesions
- Can be applied to frontal or lateral views
- Has been described for heart, aorta and the diaphragm.

**Cervicothoracic Sign**

A lesion clearly visible above the clavicles in the frontal view must lie posteriorly and be entirely within the thorax (Fig. 11).

 Conversely, if the cephalic portion of a shadow disappears as it approaches the clavicles, it is cervicothoracic, i.e. it lies partly in the anterior mediastinum and partly in the neck (Fig. 12).

**Thoracoabdominal Sign**

Convergence of the lower lateral margin of a lesion indicates that it is probably entirely intrathoracic (Fig. 13).

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![Fig. 10: Right heart border obscured by middle lobe pathology whereas left heart border seen through the opacity in left lower lobe](image1)

![Fig. 11: Border traced above clavicle—posterior mediastinum](image2)

![Fig. 12: Border not traced above clavicle—anterior location](image3)
Chapter 4: Understanding the Pediatric Chest Radiograph

Lack of convergence or actual divergence of the lower lateral margin indicates an iceberg configuration with a segment hidden within the water density of the abdomen (Fig. 14).

**Spine Sign**
The radiolucency of spine should increase as we go from superior to inferior. Increasing whiteness indicates lower lobe pathology (Figs 15 and 16).

**ABNORMAL CHEST RADIOGRAPH**

**Congenital Lung Anomalies**

**Congenital Lobar Emphysema**
- Fifty percent cases present with respiratory distress in the newborn period. Eighty percent cases present by the age of 6 months.
- Males are affected more commonly than females.
- Upper lobes are affected more often than the lower lobes.
• Left lung affected more often than the right.
• Supposed to be caused by an intrinsic or extrinsic bronchial narrowing, which results in subsequent air trapping. Intrinsic bronchial narrowing can be caused by abnormalities of underlying bronchial cartilage, whereas extrinsic bronchial narrowing is commonly caused by compression from adjacent mediastinal masses or dilated vessels.
• CLH usually is diagnosed by its typical radiographic features:
  – Progressive lobar hyperexpansion and increased translucency.
  – Displacement or compression of adjacent structures.
  – CLH initially may appear as an area of increased opacity due to retained fetal lung fluid, in the immediate postnatal period, which will clear on subsequent studies (Fig. 17).
• Surgical lobectomy is the current management of choice for symptomatic pediatric patients. Expectant management can be advocated for cases with minimal or no symptoms.

**Pulmonary Agenesis, Aplasia, and Hypoplasia**

• Classification of pulmonary underdevelopment into three main types:
  – Lung agenesis—absence of the lung, bronchus and pulmonary artery (Fig. 18).
  – Lung aplasia—presence of a rudimentary bronchus but no pulmonary artery and lung tissue.
  – Lung hypoplasia—presence of bronchial tree and pulmonary artery with a variable amount of lung parenchyma. The bronchial tree and pulmonary artery are hypoplastic (Fig. 19).
• Left lung agenesis is more common than right lung agenesis.
• May be asymptomatic or present with varying degrees of respiratory distress, depending on the extent of lung underdevelopment.
• On chest radiographs:
  – Severe cases typically present with small, radiopaque hemithorax
  – Ipsilateral pull of mediastinal structures and elevation of the hemidiaphragm usually seen
  – Compensatory hyperinflation of the normal contralateral lung which may cross the anterior midline, which is best seen on the lateral chest radiograph as a band of increased retrosternal lucency.
• Associated congenital malformations must be sought for as they may be seen in 50% to 80% of cases. Most commonly involved are heart, gastrointestinal tract, skeleton and vascular and genitourinary systems.

**Hypogenetic Lung Syndrome**

• Partial anomalous pulmonary venous return (PAPVR) refers to a condition where one or more, but not all (in contrast to total anomalous pulmonary venous return) pulmonary veins return anomalously to the systemic circulation which may be into the hepatic veins, portal veins, azygous vein, coronary sinus, or right atrium, instead of the left atrium.
• Hypogenetic lung syndrome refers to an anomalous pulmonary vein draining a part of or the entire right lung into the inferior vena cava frequently associated with various degrees of right lung hypoplasia and abnormal lobation, along with dextroposition of heart. The anomalous vein resembles a scimitar, a curved Turkish sword, hence the name “scimitar syndrome” (Fig. 20).
• Clinical signs and symptoms:
  – May be an incidental finding
  – May be related to congestive heart failure from right-heart volume overload
  – May manifest as recurrent right basilar pneumonia.
• Chest radiograph shows a typical vertically oriented curvilinear opacity, representing the scimitar vein, in the right lower hemithorax, almost parallel to the right heart border associated with a hypoplastic right lung.
• Confirmation and characterization of hypogenetic lung syndrome requires CT or MRI.
• Symptomatic pediatric patients with scimitar syndrome are treated with surgical techniques that are currently available. They aim to reconnect the anomalous vein to the left atrium with or without creation of an intracardiac shunt.

**Congenital Emphysematous Bulla**

• Emphysema is defined as abnormal and permanent dilatation of air spaces distal to terminal bronchiole, hallmark of which is hyperinflation and destruction of alveolar walls.
• Bullae are air-filled, thin-walled spaces greater than 2 cm in diameter in distended state. Giant bullae are those which encompass more than one-third of the lung volume. They are either congenital without general lung disease or a complication of chronic obstructive lung disease.
• Giant bullae are uncommon but when present can cause compression of adjacent normal lung (Fig. 21).

**Bronchogenic Cyst**

• Bronchogenic cysts typically are unilocular, fluid-filled, or mucus-filled cysts lined by respiratory epithelium.
• They are attached to but do not communicate with the tracheobronchial tree.
• Most bronchogenic cysts are located within the mediastinum (most commonly near the carina).
• May be found within the lung parenchyma, predominantly in the lower lobes.
• Clinical symptom depends on mass effect on neighboring structures including the airway, GI tract, and vessels. Small bronchogenic cysts having no mass effect are often detected incidentally in adults undergoing chest radiograph for other purposes.
• On chest radiograph a bronchogenic cyst is seen as: 
  – Round or oval-shaped soft tissue density
  – In middle mediastinum typically near the carina
- Air-fluid level, thick wall, or surrounding haziness often is associated with superimposed infection (Figs 22A and B).

- Symptomatic patients are treated with complete surgical resection. Palliative procedures such as transparietal, transbronchial, or mediastinal aspiration may be considered in symptomatic pediatric patients who cannot tolerate surgery.

### Congenital Pulmonary Airway Malformation

- Previously known as congenital cystic adenomatoid malformations of the lung (CCAM).
- Group of congenital cystic and noncystic lung masses that communicate with an abnormal bronchial tree lacking supporting cartilage.
- These are classified into 3 types:
  - Type I CPAMs—consist of cysts larger than 2 cm
    - bronchial/bronchiolar origin
  - Type II CPAMs—consist of cysts smaller than 2 cm
    - bronchiolar origin
  - Type III CPAMs—appear solid
    - bronchiolar/alveolar origin.
- Postnatal imaging findings of CPAMs usually correlate with underlying histopathologic features.
  - Type I CPAMs
    - Large cyst type
    - Present with one or several larger air-filled cysts. The cysts are larger than 2 cm and may be associated with microcysts
    - Intervening solid, unaerated lung parenchyma (Fig. 23)
  - Type II CPAMs
    - Partially air-filled multicystic masses. The cysts are smaller than 2 cm
    - Variable degrees of solid-appearing, unaerated lung tissue.

**Fig. 21:** Giant congenital emphysematous bulla

**Fig. 22A and B:** Bronchogenic cyst: (A) frontal projection; (B) lateral projection
Type III CPAMs
- Appear as solid lesions because of microscopic cysts that can be identified only at histologic evaluation.
- CPAMs are more common in the lower lobes although any lobe may be affected.
- Complicated CPAMs like those resulting from superimposed infection may appear similar to pneumonia or a lung abscess.
- Management of choice is surgical resection of the involved lobe in symptomatic cases and by 1 year of age even in antenatally detected asymptomatic cases because of the potential risk of associated complications, such as infection, pneumothorax and even malignant transformation, especially in type I lesions.

Congenital Heart Diseases

Situs
- Assessment of position of heart and visceral situs by noting the sides of liver and fundic bubble should be done.
- Dextrocardia in situs solitus (Fig. 24) is more strongly associated with cardiac abnormalities than dextrocardia with situs inversus.
- Airway anatomy and lung morphology are also valuable tools. Symmetrical bronchi, i.e. the left lung showing three lobes and horizontal fissure is seen in almost all patients with right isomerism (asplenia). It is associated with liver positioned in the midline. Splenic dysfunction and intestinal malrotation occur in children with left isomerism (polysplenia).

Diagnosis
- Chest radiography was once a major tool in the assessment of heart disease.
- Echocardiography now serves as the major primary investigation after physical examination.
- Importance of chest radiograph in congenital heart disease:
  - It may provide the first indication of cardiovascular disease in a child who presents with recurrent lower respiratory tract infection and heart disease is undiagnosed.
  - In pediatric patients with known cardiac disease, radiography helps in assessing status of pulmonary circulation and complications.
  - Chest radiography is important in the early postoperative period to look for pleural or pericardial effusions. Gross post operative pericardial effusion may indicate hemopericardium and cardiac tamponade.
  - Useful in the follow-up of heart disease.
- Chest radiographic findings in patients with cardiac disease are:
  - Cardiomegaly
  - Pulmonary vascular changes (plethora or oligemia)
  - Signs of pulmonary venous hypertension and edema.
- Children with mild structural defects and even some children with severe or complex disease may have normal chest radiographs.
- Systematic approach—Assessment of:
  - Heart size—increase in intracardiac shunts causing volume overload (e.g. VSD), cardiac failure,
pericardial effusion. But it is not significantly affected in Tetralogy of Fallot (TOR).

- Heart shape—e.g. boot-shaped heart in TOF, egg on side in TGV, and box shape in Ebstein’s anomaly. These signs are nonspecific. However, plain film findings may be specific for supracardiac total anomalous pulmonary venous return, aortic arch anomalies, pulmonary stenosis, and coarctation of the aorta.

- Pulmonary vasculature, e.g. oligemia in pulmonary stenosis, TOF and plethora in TAPVC, TGV, VSD, ASD.
- Visceral situs—due to association with complex congenital cardiac anomalies.
- Associated skeletal abnormalities in syndromic cases.

- This approach will help in narrowing down the differential diagnosis in a given clinical setting (Figs 25 to 28).
OTHER CONGENITAL LESIONS ON A PEDIATRIC CHEST RADIOGRAPH

Congenital Diaphragmatic Hernia

Classification

Congenital diaphragmatic herniation can be classified into two basic types on the basis of location:

- **Bochdalek hernia** (Fig. 29)
  - Most common fetal congenital diaphragmatic hernia
    - Commoner on the left: 75–90%
    - Posterior
    - Large and associated with poorer outcome
    - Presents earlier.

- **Morgagni hernia** (Fig. 30):
  - Less common
  - Anterior
  - Presents later.

Radiographic features include indistinct hemidiaphragm with dilated bowel loops or stomach with air-fluid level. Depending on the severity the mediastinum may be pushed to the opposite side with ipsilateral lung hypoplasia. This is more common on the left side (Bochdalek). Anterior hernias show retrosternal air-fluid level.

Congenital Chest Anomalies

- Disorganization of ribs like fusion, bifid ribs.
- Posterior closure defects of cervicodorsal vertebrae—segmental anomalies like hemivertebrae, spina bifida.
- Associated with VACTERL, Klippel Feil syndrome, Cleidocranial dysplasia (Fig. 31).

Kartagener Syndrome

- A subset of primary ciliary dyskinesia.
- Autosomal recessive condition.
- Characterized by abnormal ciliary structure or function leading to impaired mucociliary clearance.
- No gender predilection.
- Clinical triad of situs inversus, chronic sinusitis with nasal polyposis and bronchiectasis.
• Other features include telecanthus, infertility in males and subfertility in females.
• Radiographic features:
  - Predilection to involve right middle lobe, left lingular lobe and both lower lobes
  - Bronchiectasis
  - Bronchial wall thickening
  - Bronchial dilatation with loss of normal peripheral tapering
  - Mucus plugs maybe visible (finger in glove sign)
  - Consolidation may also be seen
  - Situs inversus points to the diagnosis (Fig. 32).

INFECTIVE LUNG DISEASES

Bacterial Pneumonias
• Streptococcal
  - Newborns generally affected following infection with measles
  - Patchy bronchopneumonia
  - Usually lower lobes involved
  - Empyema may develop.
• Staphylococcal
  - Rapidly developing lobar/multilobar consolidation
  - 90% associated with pleural effusions
  - 50% associated with pneumatoceles.
• Pneumococcal
  - Rare in children
  - Usually involves one lobe only
  - Lower lobe prediliction
  - Extensive lobar consolidation abutting against the visceral pleura (lobar/however may go beyond confines of one lobe through pores of Kohns)
  - Slight expansion of involved lobe
  - Twenty percent have prominent air bronchograms (Fig. 33).

COMPLICATIONS OF PNEUMONIA
• Lung abscess.
• Bronchiectasis.
• Pneumatocele.
• Loculated chronic pleural effusions.

Lung Abscess (Figs 34 to 39)
• Collection of pus within the lung.
• Primary—as a result of necrotizing pneumonias like Staphylococcus and Klebsiella.
• Secondary—bronchial obstruction by a foreign body
  - Infective endocarditis
  - Direct extension from rupture of liver abscess/chest wall abscess.
• Chest radiograph shows thick-walled cavity with air-fluid level appearing similar on frontal and lateral projections with or without surrounding consolidation (Fig. 39).
Chapter 4: Understanding the Pediatric Chest Radiograph

Figs 33A and B: Air bronchogram seen in left midzone and lowerzone suggestive of consolidation

Figs 34A and B: Radiograph showing right upper lobe consolidation in a case of staphylococcal pneumonia. Follow-up radiograph taken 3 days later shows development of pneumatoceles and loculated hydropneumothorax

Figs 35A and B: Consolidation collapse of right lower lobe with right pleural effusion
**Fig. 36:** Bronchopneumonia

**Fig. 37:** Right subpulmonic pleural effusion. There is elevation of right diaphragmatic outline, however the highest point is lateralized suggestive of subpulmonic effusion

**Fig. 38:** Left hydropneumothorax. Hyperlucency of the left hemithorax with absence of pulmonary marking and air-fluid level. Mediastinum is shifted to the opposite side. Nodular infiltrates in the right lung points to infective etiology

**Fig. 39:** Lung abscess—thick walled cavity with air-fluid level. Right pleural effusion

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**Bronchiectasis**

- Permanent localized dilatation of bronchial tree.
- Congenital causes include Kartagener syndrome (abnormal mucociliary clearance), Cystic fibrosis (abnormal secretions), alpha 1 antitrypsin deficiency (immune deficiency).
- Postinfectious causes include measles, whooping cough, allergic bronchopulmonary aspergillosis and tuberculosis.

- Types
  - Tubular/Cylindrical/Fusiform
  - Cystic/Saccular
  - Varicose.
- Chest radiograph shows cystic spaces less than 2 cm in diameter with or without air-fluid levels. Increase in size of lung marking with loss of definition of lung markings is seen due to retained secretions and peribronchiolar fibrosis. Honeycomb pattern seen in severe cases (Fig. 40).
• Clinically, there are frequent exacerbations in a chronically unwell child due to superimposed infections.

**Viral Pneumonia**
- Rhinovirus, respiratory syncytial virus, adenovirus, influenza virus, parainfluenza virus.
- Most common cause of pneumonia in children under 5 years.
- Usually bilateral.
- Hyperinflation with air trapping.
- Perihilar linear densities due to bronchial wall thickening with interstitial pattern.
- Twenty percent associated with pleural effusion, however there is striking absence of pneumatoceles, lung abscess and pneumothorax.
- Radiographic resolution lags behind symptomatic resolution by 2–3 weeks (Fig. 41).

**Pulmonary Tuberculosis**
- Children suffer from primary pulmonary tuberculosis due to inhalation of airborne droplets.
- Lower lobes, middle lobes and anterior segments of upper lobes are commonly affected.
- Areas of ill-defined airspace consolidation 1–7 cm in diameter.
- Cavitation is rare in children.
- Massive hilar, paratracheal and subcarinal lymphadenopathy more commonly on the right side (80%).
- Pleural effusion may develop.
- Pneumonic reaction with segmental/lobar consolidation.

• Calcified lung lesion (Ghon lesion) and calcified hilar lymph node called Ranke complex.
• Simon focus—healed site of primary infection in lung apex (Fig. 42).

**Differential Diagnosis of Hilar Lymphadenopathy (Fig. 43)**
- Tuberculosis
- Lymphoma
- Sarcoidosis
- Histoplasmosis
- Metastasis

**Traumatic Pneumothorax**
- Air in pleural cavity is called pneumothorax.
- Common causes include:
  - Direct trauma
  - Barotrauma in an intubated child
  - Rupture of pneumatocele.
- Chest radiograph shows radiolucent hemithorax with absence of lung markings between the lung edge and chest wall (Fig. 44).
- Signs of pneumothorax in a supine patient:
  - Hyperlucent upper quadrant of the abdomen due to air collecting at the lung base overlies the liver
  - Deep sulcus sign—due to air situated in the lateral costophrenic sulcus
  - Sharply outlined dome of diaphragm.
Principles and Practice of Pediatric Anesthesia

TENSION PNEUMOTHORAX

Characterized by

- Depression of ipsilateral dome of diaphragm.
- Push of mediastinum to the opposite side.

_Urgent management is lifesaving._

PNEUMOMEDIASTINUM

- The continuous diaphragm sign—The entire surface of diaphragm is outlined due to dissection of mediastinal gas along tissue planes (Fig. 45).
- Black ring appearance around great vessels (Fig. 46).
- Black halo around the heart.
- Thymic angel wing sign may be seen in young children.
- To differentiate from pneumopericardium—air in pericardial cavity does not extend beyond pericardial reflection, i.e. above the ascending aorta whereas air in pneumomediastinum will (Fig. 47).

Miscellaneous

Foreign Body

- Age—fifty percent occur in less than 3 years.
Eighty five percent are of vegetable origin, and hence radiolucent.
Almost always in lower lobes.
Right side is more common than the left.
Radiographic features:
- Radioopaque foreign body seen in less than 10%
- Seventy percent cases show overinflation due to air trapping
- Collapse of the involved lobe may occur due to resorption of trapped air
- Expiratory film can demonstrate air trapping better (Fig. 48).

Hyaline Membrane Disease
- Respiratory distress in premature neonates.
- Bilateral and symmetrical.
- Reticulogranular with air bronchograms—early.
- Diffusely white out lungs—late (Fig. 49).

Lipoid Pneumonia
- Caused by aspiration of vegetable/animal/mineral oil.
- Initially, there is hemorrhagic bronchopneumonia followed by giant cell foreign body reaction.
- Predilection of right middle and lower lobes.
- Homogeneous segmental consolidation is most common pattern (Fig. 50).
- Rarely reticulonodular pattern.

Paraffinoma—circumscribed peripheral mass due to chronic granulomatous reaction with fibrosis.

Normal Position of Endotracheal Tube
- ET tube should have walls parallel to that of trachea.
- It should be placed several centimeters above the carina so that it is in the trachea in all phases of respiration. Placement of tube in a bronchus will lead to collapse of the contralateral lung (Fig. 51).
• The cuff after inflation should not cause expansion of the trachea.
• Few specks of air in mediastinum after intubation may be considered normal.

**Normal Position of Central Venous Pressure (CVP) Line**

- The tip of central line should be placed in the SVC RA junction. SVC commences at the level of right first anterior intercostal space.
- The subclavian and jugular veins have valves and placement in these veins will give false reading of CVP.
- Placement in the right atrium gives rise to risk of arrhythmia (Fig. 52).

**Thalassemia Major**

Disorder of hemoglobin alpha or beta chain leading to hemolytic anemia. Chest radiograph can show widening of medullary space with coarsening of the trabecular pattern of bones (Fig. 53).
SUGGESTED READING

1. Coley BD (Ed.). Caffey’s Pediatric Diagnostic Imaging. Professor, Departments of Radiology and Pediatrics, University of Cincinnati College of Medicine, Radiologist-in-Chief, Department of Radiology, Cincinnati Children’s Hospital Medical Center, Cincinnati, Ohio


3. Radiology review article by Wolfgang Dahnert, MD, Department of Radiology, Good Samaritan Regional Medical Center.
INTRODUCTION

Electrocardiography (ECG) records the electrical activity of the heart and is a useful, though under-utilized investigation in pediatric practice. A systematic approach to ECG reading is helpful for a complete interpretation and this chapter will address a simple and systematic approach to pediatric ECG diagnosis.

INDICATIONS

Common indications for an ECG in children include chest pain, dysrhythmias (diagnosis and management), seizure, syncope, cardiac drug exposure, electrical burns, electrolyte abnormalities and abnormal cardiac findings. Its role is somewhat limited in the diagnosis of structural heart disease. However, it does provide important clues regarding the changes in chamber dilatation and supplements information required for diagnosis along with clinical examination and chest radiography.

The limitations of pediatric ECG are as follows:
- A single set of criteria can’t be applicable for all ages as age dependent ECG changes occur.
- Chamber enlargement criteria are borrowed from adult data.
- Poor sensitivity, e.g. a child with large ventricular septal defect (VSD) may not have large left ventricular (LV) forces.
- There are no specific guidelines for chest lead placements.
- Congenital heart diseases have very few lesion specific changes on ECG.
- Pediatric training generally is a little soft on ECG interpretation, particularly arrhythmias.

ECG RECORDING AND INTERPRETATION

The ECG may be recorded in a single channel or multichannel. The ECG is a graphical plot of voltage on the vertical axis against time on the horizontal axis. In standard ECG, the paper speed is 25 mm/sec indicating that 1 sec is divided into 25 small divisions of 1 mm each. Thus, each small block measures 0.04 seconds and a large block (comprising of 5 small ones) correlates to 0.20 seconds. The usual standardization is two big boxes tall with normal gains of 10 mm/mV. If the QRS voltage is very large, then the gain may be halved (Fig. 1A).

The basis for a properly performed ECG is lead placement. The limb leads are placed on the right and left arm and the left leg as marked on the leads. The placement of leads must be more proximal in children to avoid limb-motion artifacts. Arm lead reversal will cause false P wave abnormalities; hence one should exercise caution while recording ECG. The usual 12 lead ECG is not enough in pediatrics, and a 14 lead ECG to include V4R or V3R is necessary in cases of congenital heart diseases. The other leads include standard limb leads (I, II, III), augmented limb leads (aVR, aVL, aVF), which represent the frontal plane and the precordial, leads (V1-V6) which represent the horizontal plane.
Steps to read Pediatric Electrocardiograms

The ECG must be read systematically to extract the maximum information. The 7 steps to interpreting pediatric ECGs are:

1. Heart rate
2. Rhythm
3. P wave analysis: Representing atrial events
4. P-QRS-T axis: Mean vector of depolarization for representative waves (Fig. 1B)
5. Intervals-PR, QRS duration and QT/QTc
6. QRS wave analysis, q wave, R/S ratio
7. ST segment and T wave changes.

**Step I: Heart Rate Calculation**

The best method to calculate heart rate (HR) is dividing 1500/Number of small squares between 2 R’s (R-R interval). For e.g. if there are 15 small squares between two consecutive R’s, then the heart rate is 100/min (1500/15) or one could calculate the approximate heart rate by counting the number of large boxes between 2 QRSs, e.g. HR = 300 ÷ number of large boxes between QRSs. RR interval may slightly vary but usually less than 3 small divisions. If variation is more than 3 divisions (120 msec) it is labeled as sinus arrhythmia. The usual cause is the physiological respiratory sinus arrhythmia. The heart rate rises and falls with inspiration and expiration, and this variation is more pronounced in young children. Age and activity-appropriate heart rates must thus be recognized. Heart rates grossly outside the normal range for age should be scrutinized closely for dysrhythmias. Normal heart rate in the neonates varies between 120-220/min and decreases to about 120 beats/min at 1 year, 100 at 5 years and reaches adult values by 15 years. Persistent HR more than 180 beats/min (older children) and 220 beats/min (neonates) indicates tachyarrhythmia. Persistent HR <60 beats/min (older children) and <80 beats/min (neonates) denotes bradyarrhythmia (Figs 2 and 3).4,5

**Step II: Determining the Rhythm**

Sinus rhythm is present, if the PR interval is constant throughout the tracing, and the P wave deflection is positive in leads II and aVF but negative in aVR.5

“Sinus bradycardia” is a slow sinus rhythm seen normally in aerobically trained individuals, but also occasionally in hypothyroidism and long QTc conditions. “Sinus tachycardia” is a fast sinus rhythm that is consistent with anxiety, crying, fever, and occasionally hyperthyroidism.

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**Fig. 1A and B:** (A) Normal ECG grid box with wave, intervals and segments; (B) Axis determination and interpretation: Based on net deflection of individual wave forms in lead I and aVF the P’ QRS’ T’ axis is located in one of the four quadrants and thus interpreted as normal or abnormal

**Abbreviation:** N, normal; NW, north west; LAD, left axis deviation; RAD, right axis deviation

**Fig. 2:** Sinus tachycardia with HR around 150 beats/min

**Fig. 3:** Sinus bradycardia with HR below 60 beats/min
Sinus rhythm is not present if the P wave is negative in lead II and aVF but positive in aVR. This pattern may be consistent with a non-sinoatrial rhythm, such as when the intrinsic cardiac pacemaker is in the low right atrium or in the left atrium. Occasionally, a child may have a pacemaker, and a paced rhythm will then be found (Fig. 4). The normal sinus rhythm may be occasionally disturbed by frequent or infrequent ectopic beats, which may be narrow QRS ectopics-premature atrial contractions (PACs)(Fig. 5) or wide QRS ectopics-premature ventricular contractions (PVCs)(Fig. 6). Also, the normal rhythm may be disturbed by sinus pauses or junctional escapes (narrow QRS complex without preceding P waves) seen during sleep, feeding and defecation. Analyzing the P wave additionally helps us to know whether the rhythm is sinus or not, as detailed below in step III.

**Step III: P Wave Analysis**

P wave represents atrial depolarization. This is the time taken by an electrical impulse to spread from the sinoatrial node through the atrial musculature. The first half of P wave represents the right atrial (RA) and second half the left atrial (LA) depolarization respectively. As the pacemaker of the heart is situated in SA node, all the P waves look similar and are uniform. P waves are best read in Lead II, aVF, and I. In situs solitus (normal) the P wave is always positive in I, II, aVF, V4 and V6 and negative in aVR (Normal P axis). In situs inversus P wave is positive in aVR and negative in I, II, V5-6. Morphology of P wave can vary. It can be monophasic, biphasic, notched, inverted etc. Wandering pacemaker is a benign finding where there are continual changes in P wave morphology, amplitude and PR interval. Abnormal or non uniform P waves, more than one in number, P waves that are not followed by QRS, varying PR interval or abnormal P axis represent non-sinus rhythm and should make one suspect dysrhythmias. Widest P wave can be up to 100 msec. Abnormal width of P wave >120 msec (3 small divisions) suggest LA enlargement. Ventricular septal defect (VSD), patent ductus arteriosus (PDA), aortopulmonary window and mitral atresia demonstrate left atrial enlargement. Maximum amplitude of P wave is 2.5 small divisions So amplitude of >3 mV (3 mm) is abnormal, i.e. tall and peaked P wave suggest right atrial enlargement. Tricuspid atresia, pulmonary atresia with intact ventricular septum and severe pulmonary stenosis are associated with right atrial enlargement.

**Step IV: Axis Detection**

Axis detection is crucial in evaluation of congenital heart disease (CHD). QRS axis helps to interpret net deflection of ventricular depolarization, P axis, the atrial depolarization (Normal P axis at all ages lies between 30 to 60 °) and T axis, the ventricular repolarization (T axis closely follows the QRS axis). Two important leads to determine all axes are leads I and aVF. E.g. for determining QRS axis, note if the net QRS voltage is positive or negative in these leads. (Figs 1 and 7). Then applying the simple rule locate which quadrant the QRS axis is located as shown in Table 1:

At birth, right axis deviation of the mean QRS vector is the rule. The axis becomes normal by 6 months of age. Hence, normal or leftward QRS axis is abnormal

<table>
<thead>
<tr>
<th>Lead I</th>
<th>Lead aVF</th>
<th>Interpretation</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive</td>
<td>Positive</td>
<td>Normal axis (0 to +90°)</td>
<td>Abnormal in neonates and early infancy</td>
</tr>
<tr>
<td>Negative</td>
<td>Positive</td>
<td>Right axis deviation (+90° to +180°)</td>
<td>Normal in neonates and in early infancy</td>
</tr>
<tr>
<td>Positive</td>
<td>Negative</td>
<td>Left axis deviation (0 to -90°)</td>
<td>Abnormal at any age</td>
</tr>
<tr>
<td>Negative</td>
<td>Negative</td>
<td>North-west axis / right upper quadrant axis (-90° to ±180°)</td>
<td>Abnormal at any age</td>
</tr>
</tbody>
</table>

**Table 1: Location of QRS axis**

![Fig. 4: Single chamber atrial pacemaker. Rhythm is regular, with heart rate of 60 bpm. The P wave, PR interval and QRS is normal](image)

![Fig. 5: Premature atrial beat with noncompensatory pause](image)

![Fig. 6: Premature ventricular complex quadrigeminy after every third normal beat](image)
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in the neonatal period and early infancy. Right Axis deviation (+90° to +180°) is seen in neonates (due to right ventricular dominance in fetal life), mechanical shifts, emphysema, right ventricular hypertrophy, right bundle branch block, dextrocardia, ventricular ectopic rhythms and pre-excitation syndrome (Wolff-Parkinson-White). Common conditions with leftward axis of QRS vector are CHD like tricuspid atresia, atrioventricular septal defects or univentricular hearts. ‘T’ axis closely follows corresponding ‘QRS’ axis and QRS-T angle is acute (both axes in the same quadrant). If the axes are in different quadrants, it indicates there is a strain on the ventricle.

Step V: Intervals

PR interval represents the time taken by an impulse to travel from the atria through the AV-node to the bundle of His. This is measured from the beginning of the P wave to the beginning of the QRS complex. PR segment is the interval between end of P wave and beginning of QRS complex and is an isoelectric segment. The PR interval varies with age. (Neonates-0.08–0.15, adolescents- 0.12–0.20 seconds). Atrioventricular blocks result in prolongation of the PR interval (Figs 8A, B and 9). Second-degree block of the Mobitz type II and third degree block are pathological and carries a high mortality risk. Congenital complete heart block (CHB) may be seen in children born of mothers with systemic lupus, and in children with congenital corrected transposition of great arteries and AV canal defects. Acquired CHB can be seen in myocarditis, digitoxicity and following cardiac surgery. Short PR interval is seen in Pompe’s disease, WPW syndrome, and ectopic lower atrial pacemaker.

WPW Syndrome (Pre-excitation)

In children, WPW syndrome is the commonest cause of paroxysmal supraventricular tachycardia. Pre-excitation of ventricular tissue is caused by premature conduction of atrial impulses to ventricles through accessory pathways causing the delta wave and a fusion complex in the ECG. The pre-excitation may be subtle and noted in a resting ECG in the mid-precordial leads. Congenitally corrected transposition of great arteries, Ebstein’s anomaly, hypertrophic cardiomyopathy and cardiac rhabdomyoma have a higher incidence of WPW syndrome. The incidence
of sudden death from cardiac arrest is high and may be the initial presentation. Hence, it is important to have a high index of suspicion for this electrical abnormality. Digoxin and verapamil must be avoided and beta-blocker therapy is the drug of choice (Fig. 10).

QRS duration is the time taken for ventricular depolarization through the bundle of His, its branches and Purkinje system. The QRS complex measures 0.04–0.08 seconds in neonates and 0.05–0.10 (1–3 small divisions) seconds in adolescents. Thus, even a slight prolongation of what may seemingly appear as normal QRS complex can indicate a conduction abnormality or bundle branch block (BBB) in children. To identify bundle branch block, look for ‘M’ sign. If ‘M’ pattern of QRS is seen on V1 then suspect RBBB, if ‘M’ pattern of QRS is seen on V6 then diagnosis is LBBB (Fig. 11A and B).

QTc (Corrected QT interval): The QT interval extends from the beginning of the QRS complex to the end of T wave and represents the total time for ventricular depolarization and repolarization. This interval is best measured in leads II, V5 and V6 and the longest interval is used. QT varies with heart rate, so the corrected QT interval or QTc is calculated by using Bazett’s formula: QTc = QT interval (in milli secs) /√ RR in seconds. Normal QTc is <440 msec in older children and up to 490 msec in early infancy. Prolonged QTc is seen in hypokalemia, hypocalcemia, hypothermia, hypomagnesemia, cerebral injury and certain drugs like macrolide antibiotics, cisapride, etc. There are certain syndromes of long QTc (LQTS) like Jervell and Lange-Nielsen syndrome, Romano-Ward syndrome and other inherited channelopathies. An abnormal QTc can result in sudden death; therefore, the QTc must be calculated in every ECG (Fig. 12). Short QTc is also abnormal when it measures less than 330 msecs.

**Step VI: QRS Wave Analysis**

QRS complex: It follows the PR interval. The q wave represents septal depolarization, which occurs from right to the left and thus is best seen in leads I, aVL, V4-V6 and aVF. The depth is <3 mm and width <1 mm. A q wave >4 mm deep is abnormal and represents ischemia or volume overload of that ventricle. The RS wave represents the ventricular depolarization. QRS duration is traditionally measured in limb leads or V1–V2. Low voltage QRS complexes are defined as <5 mm height in limb leads and, <10 mm in precordial leads. Right ventricular hypertrophy (RVH) is present, if any of the following are present: R wave >98% in V1 or S wave >98% in I or V6, increased R/S ratio in V1 or decreased R/S in V6, RSR’ in V1 or V3R in the absence of complete RBBB, Upright T wave in V1 (>3 days), presence of q wave in V1, V3R, V4R. The differential diagnosis of RVH is ASD, total anomalous pulmonary venous return (TAPVR), pulmonary stenosis, tetralogy of Fallot (TOF), large VSD with pulmonary hypertension (HTN). Left ventricular hypertrophy (LVH) is defined if any of the following are present: R >98% in V6, S >98% in V1, Increased R/S ratio in V6 or decreased R/S in V1, q >5 mm in V6 with peaked T waves. The differential diagnosis
includes—VSD, PDA, complete AV block, aortic stenosis, and systemic hypertension (HTN)(Fig. 13).

**Step VII: T Wave and ST Changes**

The T-wave represents ventricular repolarization and follows the S wave and ST segment. ST segment is an isoelectric segment (like the PR segment). The elevation of ST segment up to 4 mm with the concavity facing upwards is a normal variation and represents early repolarization. Abnormal elevation of ST segment is seen in pericarditis, hyperkalemia, pneumothorax or pneumopericardium. ST depression is suggestive of pressure overload/strain.

The T waves in leads V1–V3 are generally inverted after the first week of life till 8 years of age (“juvenile” T wave pattern). Hence, upright T waves in these leads suggest RVH. T wave inversion in leads I, V5 and V6 are seen in ischemic conditions like ALCAPA (Anomalous Left Coronary arising from the Pulmonary artery)(Fig. 14) and pressure overload/strain. Tall T wave is a common ECG manifestation of hyperkalemia. The other manifestations include disappearance of P wave, broadening of QRS wave, disappearance of ST segment resulting in sine wave. In hypokalemia, there is gradual reduction in the amplitude of T wave with eventual disappearance of T wave and appearance of U wave (Fig. 15).

**SOME DISEASE SPECIFIC ECG CHANGES**

The common complex congenital heart diseases like tetralogy of Fallot, D-transposition of great arteries, total anomalous pulmonary venous connection, truncus arteriosus, pulmonary atresia, hypoplastic left heart syndrome show q waves in inferior leads (leads II, III and aVF) with RVH and right axis deviation of the QRS vector. The ECG pattern, however, is not specific for these lesions nor helps to distinguish one lesion from the other. Importantly, absence of above mentioned features point towards some other diagnosis.

- **Common AV canal defect:** q wave in leads I and aVL, left axis deviation of QRS.
- **Tricuspid atresia:** q wave in leads I and aVL, right atrial enlargement with left axis deviation.
**Ebstein's anomaly**: Giant P waves, low voltage complexes, ECG characteristics of WPW syndrome and a RBBB pattern.

**Corrected transposition of great arteries**: q waves are seen in V1 and V2, but absent in left precordial leads (V5, V6). In this cardiac lesion, the ventricles are inverted and the septal depolarization is reversed, thus explaining the ECG pattern.

### ARRHYTHMIAS IN CHILDREN

Arrhythmias can be broadly classified based on HR as bradycardia and tachycardia. Arrhythmias can also be grouped based on the site of origin (atrial, junctional or ventricular). The characteristics of various abnormal rhythms seen in children are listed in Table 2. The clues for interpretation of major arrhythmias and Atrioventricular blocks are shown in Table 3.

All tachycardias with its origin above the bundle of HIs are called supraventricular tachycardias. (SVT/narrow complex arrhythmias) and all that arise below are called ventricular tachycardia (VT/broad complex arrhythmias). SVT is much more common than VT. Any wide QRS tachycardia should be considered as VT until proved otherwise. Answering the following questions helps one to arrive at proper ECG diagnosis of the tachycardia:

1. Is the QRS complex narrow/wide?
2. Is the rhythm regular/irregular?
3. Is the P wave present/absent?
4. If P wave is seen, what is its relation with QRS?
5. Effect of vagal maneuvers/administration of adenosine?

#### 1. Is the QRS complex narrow or broad?

Generally, narrow QRS complexes suggest SVT and broad complex suggests ventricular tachycardia (Figs 16 and 17).

#### 2. Is the heart rate regular or irregular?

Tachycardia with irregular rhythm is atrial flutter, atrial fibrillation or ectopic atrial tachycardia with varying block, multifocal atrial tachycardia and polymorphic ventricular tachycardia [which is a medical emergency (Fig. 18)]. All the other tachycardias usually have regular RR interval.

#### 3. Is the P wave seen?

P waves are not seen in – AV nodal re-entrant tachycardia (AVNRT), Junctional ectopic tachycardia (JET).

- If T waves are tall and peaked or ST segment is depressed in the inferior leads, one should consider the superimposition of P wave on the T wave or on the ST segment respectively.

**Table 2**: ECG characteristics of abnormal rhythms

<table>
<thead>
<tr>
<th>Premature atrial contraction (PAC)</th>
<th>Premature ventricular contraction (PVC)</th>
<th>Atrial flutter</th>
<th>Atrial fibrillation</th>
<th>Ventricular tachycardia</th>
<th>Ventricular fibrillation</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Length of two cycles (R-R) usually shorter</td>
<td>• Premature, wide QRS, no P waves, T wave opposite to QRS</td>
<td>• Rapid atrial rate (around 300 bpm) with varying ventricular rate</td>
<td>• Very fast atrial rate (350–600 bpm)</td>
<td>• Wide, unusually shaped QRS</td>
<td>• Very irregular QRS</td>
</tr>
<tr>
<td>• Preceded by P wave, followed by normal QRS</td>
<td>• May be multifocal, bigeminy, trigeminy, and couplets. Normal if uniform and decreases with exercise</td>
<td>• Sawtooth pattern</td>
<td>• Irregularly irregular</td>
<td>• T waves opposite direction of QRS</td>
<td>• Rate is rapid and irregular</td>
</tr>
<tr>
<td>• No hemodynamic significance</td>
<td></td>
<td>• Suggests significant pathology</td>
<td>• No P waves, normal QRS</td>
<td>• HR 120–200 bpm</td>
<td>• Terminal arrhythmia</td>
</tr>
</tbody>
</table>

**Table 3**: The ‘ECG rules’ of major arrhythmias and atrio-ventricular (AV) blocks

<table>
<thead>
<tr>
<th>Arrhythmias</th>
<th>AV Blocks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinus</td>
<td>First degree</td>
</tr>
<tr>
<td></td>
<td>Simple prolongation of PR interval</td>
</tr>
<tr>
<td>Atrial</td>
<td>Second degree</td>
</tr>
<tr>
<td></td>
<td>QRS complexes do not follow some P waves</td>
</tr>
<tr>
<td>Nodal</td>
<td>Mobitz type I</td>
</tr>
<tr>
<td></td>
<td>Progressive lengthening of PR interval with eventual dropping of QRS complex (Wenkebach type)</td>
</tr>
<tr>
<td>Ventricular</td>
<td>Mobitz type II</td>
</tr>
<tr>
<td></td>
<td>The PR interval is normal and constant in successive beats, but some P-waves are not conducted, e.g. every other or every third P waves is conducted. (2:1 or 3:1 block)</td>
</tr>
<tr>
<td></td>
<td>Third degree (or complete)</td>
</tr>
<tr>
<td></td>
<td>P waves and QRS complexes are entirely independent; the RR interval is perfectly regular and occurs at a slower rate than PP interval</td>
</tr>
</tbody>
</table>

Source: Jasmine Lam. Approach to Pediatric ECG. Learn Pediatrics @learnpediatrics.com.
In atrial flutter, the isoelectric line is replaced with continuous regular P waves giving rise to a ‘sawtooth’ appearance and in atrial fibrillation, with fibrillatory waves.

4. If P waves are seen, what is its relation with QRS?
- If P waves are prior to QRS complex, then the tachycardia is of atrial origin. With a normal P axis, the rhythm is sinus tachycardia or sinus node re-entrant tachycardia. With an abnormal P axis (different P wave morphology), diagnosis is ectopic atrial tachycardia (EAT).
- If P waves are seen prior to each QRS but inverted in the inferior leads, the possibilities are: EAT, paroxysmal junctional reciprocating tachycardia and atypical AVNRT.
- If the P waves are seen after the QRS complex, generally it would be inverted in leads II, III and aVF. This is due to retrograde activation of atria from the ventricles.
- In AVNRT: Inverted P wave is seen on the terminal portion of QRS or just after QRS complex. This is also seen in JET with 1:1 retrograde conduction. In AVRT (WPW syndrome) inverted P is seen on the ST segment.

If P waves are seen with no consistent relation with QRS complex, it indicates AV dissociation.

5. What is the effect of vagal maneuvers or administration of adenosine?
Adenosine administration can be both diagnostic as well as therapeutic. It causes a marked slowing of the AV- node conduction with the effect lasting for a few seconds only. It is imperative to obtain an ECG record during adenosine administration. (a) In sinus tachycardia there is a gradual slowing of sinus rate and gradual return to the original level; (b) In ectopic atrial tachycardia, the ventricular rate slows, P waves unmasked; (c) In atrial flutter, the ventricular rate slows, flutter waves are seen better; (d) AV node dependent (AVNRT and AVRT) the tachycardia terminates; (e) In junctional ectopic tachycardia, adenosine is ineffective.

SUMMARY
Interpreting pediatric ECG is easy if a systematic approach of heart rate, intervals, axis and waveform morphologies is applied. Knowing what are the normal ranges in pediatric age helps to identify what is abnormal.
LEARNING POINTS

- Heart rate ranges from 80–220 in neonates and 60–180 in children.
- Single P wave of normal and uniform morphology in front of the QRS with a constant PR interval defines a sinus rhythm. Sinus arrhythmia is seen normally in children.
- Rightward QRS axis > +90° is normal in early infancy
- Normal PR interval is (0.12–0.20 sec) and QRS duration (0.08–0.10 sec)
- Normal QTc is 0.44 sec in older children and up to 0.49 sec in early infancy
- P waves are 3 small divisions broad (0.12 sec) and 3 divisions tall (3 mm)
- Q waves best seen in the inferior and left precordial leads
- T wave inversions in leads V1-3 ("juvenile T-wave pattern") is normal after 7 days of life

REFERENCES


Anesthetic Management

Chapter 6: Preoperative Evaluation and Premedication
Chapter 7: Induction, Maintenance and Emergence
Chapter 8: Monitoring in Pediatric Anesthesia
Chapter 9: Perioperative Fluid and Electrolyte Therapy
Chapter 10: Assessment and Management of the Difficult Pediatric Airway
Chapter 11: Pediatric Pain Assessment and Management
Chapter 12: Ventilatory Strategies in the Operating Room
Chapter 13: Regional Anesthesia in Infants and Children
Chapter 14: Transfusion Therapy and Bleeding Disorders
INTRODUCTION

Anesthesiologists should play a leading role in the medical evaluation and psychological preparation of children before surgery or other procedures requiring anesthesia. The primary steps in preoperative preparation are to determine whether the child is in the best possible state of health, given the child’s underlying medical condition, and to manage any concurrent acute interceding illness. The main aim is that the patient’s medical condition should be optimized when he or she presents to the operating room.

Preoperative goals of care are as follows:

- To clearly define the child’s medical issue
- To delineate the physiologic effects and limitations imposed by each condition
- To optimize the management of any comorbid conditions
- To bring patients from a higher risk stratum to a lower one when possible, or at least to ensure that the patient is in the best condition within his or her physical status category
- To present information that will encourage and facilitate communication among surgeons, anesthesiologists and pediatricians
- To provide guidelines to pediatricians and other primary health care providers, who are preparing patients and families for anticipated procedures.

Preoperative evaluation and preparation of pediatric patients involves coordination of anesthesiologist, surgeon and pediatrician. Each of them has their own perspective, but many issues are common to all. It is important to educate the family members about the entire process. The family should be given an opportunity to express anxieties and concerns on an individual basis. Written and audiovisual material could be given to parents. Young children are concrete thinkers, so questions have to be phrased carefully in order to allay their fears.

ROLE OF SURGEON

Patients present to the surgeon in three typical situations:

1. Treatment of a healthy child who is posted for elective surgery.
2. Chronically ill child scheduled for surgery.
3. Acutely ill or injured child undergoing surgery.

The surgeon has to explain to parents the need and details of the surgical procedure, discuss potential complications, postoperative care, anticipated outcome and follow-up. The family should be explained that if a concurrent illness develops which would compromise the procedure, anesthesia and recovery, then surgery could be cancelled for that day.

ROLE OF ANESTHESIOLOGIST

Anesthesia blunts input and output of sensory, motor, reflex and mental functions. There is a temporary alteration in physiology. So, for an anesthesiologist the knowledge of details of present surgical illness, present and past medical history, birth history, current medications, past anesthesia experiences, history of allergies are all important and mandatory.
Perioperative morbidity could be decreased with a thorough preoperative assessment. It is crucial to detect any underlying risk factor that may lead to an unexpected adverse event in the perioperative period. At the same time, preoperative assessment should not involve a number of unnecessary tests which create a stressful environment for the child and family prior to anesthesia.1

ROLE OF PEDIATRICIAN

The pediatrician's examination sets a baseline against which the perioperative physical status can be compared. Highlighting neurologic findings, cardiac murmurs, wheezing, rashes, congenital anomalies and vital signs is helpful for perioperative care providers. The key concept is that the patient’s medical condition should be optimized before surgery.

*A word of caution:* A detailed medical history and physical examination by any other healthcare professional cannot make a patient fit for anesthesia. It only provides additional information to the anesthesiologist and aids decision-making regarding whether the patient can withstand anesthesia safely.

THE PREANESTHESIA CONSULTATION2

Ideally all preoperative evaluations should be well in advance of surgery, so as to avoid last minute cancellations due to missing data critical to optimizing patient’s condition for surgery. If child is examined some time before surgery, a quick short evaluation of the child’s health must also be made on the day of surgery to detect any newly developed problems like an upper respiratory tract infection (URTI).

Just by observing the child from a distance while talking to the parents, important information can be gathered, e.g. breathing difficulties, running nose, cough, basic neurologic development and general health and activity levels.

General Considerations

*History of present illness:* Duration of present illness and the degree of incapacitation with respect to hemodynamic, respiratory, renal and fluid status.

*Medical history:* Presence of other comorbid conditions, and whether a chronic condition has led to the current problem for which surgery is planned.

*Medications:* We must be aware of all the medications that the child is currently taking. Anticonvulsant therapy is optimized prior to surgery. Most of the anticonvulsants have a long half life, so if one dose is missed it should not affect blood levels significantly.3

Ninety percent of diabetic children present with type I diabetes. For these children, surgery should be scheduled early in the morning to avoid long fasting periods. They require about 0.1–0.2 U/kg/h of insulin and the amount should be adjusted as per glucose level.4

Children on long-term steroid therapy, should receive their daily dose orally or parenterally on the day of surgery. An additional dose to counteract the stress of surgery would depend on duration and severity of surgery. Unnecessary large doses are avoided to prevent side effects, such as poor wound healing, inadequate glucose control, fluid retention, hypertension, electrolyte imbalance, immunosuppression, etc.1 As adrenal glands need one year to completely recover following discontinuation of corticosteroids, it is recommended to substitute until one year following discontinuation of corticosteroids.

*Herbal medicines:* Literature highlights potential dangers of herbal medicine. An increasing number of pediatric patients do receive herbal medications and it is not reported at the time of preoperative visit. Herbal remedies like fish oil, ginger, garlic, grape seed extract, Ginkgo bilboa, inhibit platelet aggregation. Echinacea used for URTI may cause immunosuppression. Kava kava and passion flower can lead to CNS depression in perioperative period. Ma Huang contains ephedrine and has the potential to interact with volatile anesthetics and cause arrhythmias.

A report of the World Health Organization Monitoring Center with nearly 5000 cases of adverse events associated with herbal medications included approximately 100 events in children below 10 years.5

*Immunization:* In elective cases, one can wait if vaccination is due to be given in the near future. Vaccinations may be followed by local swelling, pain, fever, headache, rash, malaise and myalgia. These symptoms may last between 2 days to 2–3 weeks.5 There is paucity of literature on anesthetic implications of a recent vaccination. It is probably better to postpone elective surgery for at least 3 days following a vaccination with killed organisms (pertussis vaccine) or inactivated toxins (tetanus and diphtheria) and 2 weeks following attenuated live organisms (measles, mumps, rubella, poliovirus vaccine) to reduce the coincidence of peak systemic reactions to the vaccine with surgery.

Anesthesia, stress and trauma are known suppressors of immune system. So, elective surgery should be postponed in case of an active disease or following contact with another child with active disease.6,7
Special Preoperative Considerations and Implications in Pediatric Patients

Respiratory System

Respiratory events contribute to majority of morbidity and mortality in pediatric patients. The known factors responsible for these are asthma, bronchial hyperreactivity (BHR), URTI and passive smoking. All of these should be elicited in the preoperative history.

Age is an independent risk factor for respiratory complications. The infant has a highly compliant chest wall with relatively low transpulmonary pressures at end expiration with consequent increased tendency for collapse of small peripheral airways even during normal tidal ventilation. In the older child chest wall compliance decreases as age advances, hence tendency for airway collapse reduces.

Infants comparatively have a higher vagal tone, so higher chances of laryngospasm and apnea following vagal stimulation due to irritation of airway receptors by suctioning, secretions or intubation.

Incidence of asthma is quite high in children and BHR persists for many weeks after an acute asthmatic episode. Child may require hospitalization and increase in medication for optimization. Treatment with corticosteroids preoperatively has shown to decrease respiratory adverse events. Inhaled steroids should be started well before surgery. Methylprednisolone 1mg/kg can be given orally before surgery. Nebulization with beta-2 agonists in asthmatic children decreases airway hyperreactivity. Anticholinergic drugs can prevent perioperative bronchospasm through their antimuscarinic effect on the airway receptors via the parasympathetic system.

Upper respiratory tract infection: Upper respiratory tract infection is a frequent accompaniment of most children. There is still an uncertainty regarding the duration of time that elective surgery should be deferred after an episode of URTI. A child with URTI is prone to adverse respiratory events, such as laryngospasm, bronchospasm, desaturation and atelectasis due to altered airway reactivity. Hyperreactivity of airway can persist up to 4–6 weeks, hence the rationale to postpone surgery for 4 weeks in severe URTI. Some authors suggest that acute uncomplicated URTI has no increased morbidity. On an average there are about 6–7 URTI episodes in a year, hence repeated cancellation is quite impractical and can have an adverse economic and emotional impact on the family.

Decision to proceed with surgery depends on urgency of surgery, careful risk-benefit analysis, age of the child and expertise of the anesthesiologist (Table 1).

Table 1: Child with runny nose

<table>
<thead>
<tr>
<th>Schedule surgery/Intubation</th>
<th>Cancel surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clear runny nose</td>
<td>Child &lt;1 year</td>
</tr>
<tr>
<td>Dry cough</td>
<td>Profuse runny nose</td>
</tr>
<tr>
<td>Minor surgery</td>
<td>Productive cough</td>
</tr>
<tr>
<td>No tracheal intubation</td>
<td>Wheezing</td>
</tr>
<tr>
<td></td>
<td>General symptoms: fever&gt;38.5°C, headache, irritable child, feeding problems, stopped playing</td>
</tr>
</tbody>
</table>

Passive and active smoking: Smoking parents are advised not to smoke in the child’s presence for at least 48 hours so as to decrease carboxyhemoglobin levels in the child. It also eliminates stimulant effect of nicotine on the cardiovascular system and improves respiratory ciliary function. For older child who smokes, guidelines similar to adults are applicable.

Obstructive sleep apnea: Obstructive sleep apnea syndrome (OSAS) is a breathing disorder characterized by repeated collapse of upper airway with periods of apnea. Magnetic resonance imaging (MRI) studies show that these patients have smaller volume of the upper airway and significantly large tonsils and adenoids. Soft palate is thickened in these kids. Children with history of snoring, apnea, obesity are prone to OSAS, they can have lower mean oxygen saturation during perioperative period. They are also prone to worsening of OSAS in the postoperative period with more episodes of apnea and hypoxemia. Untreated long standing OSAS can result in pulmonary hypertension and cor pulmonale. Polysomnography is the gold standard, but high cost and lack of consensus on interpretation limits its use in children. Interdisciplinary clinical management is more apt.

Bronchopulmonary dysplasia (BPD): Infants born with weight between 500 and 1500 grams are predisposed to develop bronchopulmonary dysplasia (BPD). It is defined as oxygen dependence at 36 weeks postconceptional age with a total duration of oxygen therapy of <28 days. They are prone to perioperative bronchospasm and oxygen desaturation in the first year of life. There is increased chance of pulmonary vasoconstriction, ventilation perfusion mismatch and greater risk of hypoxemia. One has to suspect BPD if child was born premature and was mechanically ventilated in the neonatal period. In these patients, optimization with bronchodilators, steroids, diuretics and antibiotics may be required. Serum electrolytes should be measured in a child receiving diuretics.

Cystic fibrosis: These children often have malnutrition and chronic pulmonary infections. They are on long-term antibiotic treatment, and hence, at higher risk of nosocomial infection. They may have difficult venous access. Preoperative physiotherapy and antibiotics may be...
started before elective surgery.\textsuperscript{1,13} For emergency surgery, child may have to be treated on the lines of BHR.

**Airway:** Simple bedside tests may be helpful to predict a difficult airway. Child should be asked to open the mouth wide and to extend the neck to rule out cervical spine problems. A high arched palate with a narrow mouth opening may point towards a difficult laryngoscopy. In a child the thyromental distance should be at least the size of three middle fingers of the child’s hand joined together. In patients with craniofacial malformations and syndromes, such as Pierre-Robin, Goldenhar, Klippel Feil, Down’s, etc. the airway difficulties are obvious.

Evaluation of infant airway can be challenging. Dr G Lane introduced the COPUR airway score (Table 2).\textsuperscript{14}

Significant anatomical or physiologic variables are given points from 1 to 4, past history or pertinent medical history are taken into consideration. The more points a child accumulates, the more challenging the airway will probably be.

**Prediction points based on COPUR scale:**
- 5–7: Easy normal intubation
- 8–10: Laryngeal pressure may help
- 12: More difficult, fiberoptic may be less traumatic
- 14: Difficult intubation, fiberoptic or other advanced technique
- 16: Dangerous airway, consider awake intubation, potential tracheostomy.

**Cardiovascular System**

Cardiac murmurs: Incidence of serious congenital disease is less than 1%. It is important to identify and evaluate pathological murmurs by preoperatively eliciting appropriate and relevant medical history (Box 1).

Some syndromes are associated with congenital heart disease, such as Down’s syndrome, CHARGE association (coloboma of the eye, heart defects, atresia of choanae, retardation of growth, genital and/or urinary abnormalities, ear anomalies and deafness), VACTERL syndrome, Turner’s syndrome, etc.

A family history of sudden death should make one suspicious of hypertrophic obstructive cardiomyopathy (HOCM), which has an autosomal dominant inheritance. It is often asymptomatic, with electrocardiogram (ECG) showing left axis deviation and left ventricular hypertrophy. Critical aortic stenosis may also be asymptomatic in a child.\textsuperscript{15}

Examination of peripheral pulses in all four limbs for equality and delay as in coarctation of aorta, chest inspection for pulsations, palpation for thrills and auscultation of heart sounds for intensity and chronology. Murmurs have to be evaluated for location, timing, duration, intensity, quality and variation with posture, in every child who has a murmur.

In a child with an innocent murmur (soft, early systolic, without thrill, normal ECG and asymptomatic), it may be safe to proceed with surgery and investigate further postoperatively. If nature of murmur is doubtful, an echocardiography may be done to rule out organic cause. The major risk factors for increased mortality are cyanosis, younger age, complex cardiac anomalies and poor general health. Patients with corrected anomalies like ventricular septal defect or patent ductus, and who are well compensated, can be safely anesthetized.

**Table 2:** COPUR airway scale

<table>
<thead>
<tr>
<th>Variable</th>
<th>Grades</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>C—Chin From the side view the chin is:</td>
<td>Normal</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Small, moderately hypoplastic</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Markedly recessive</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Extremely hypoplastic</td>
<td>4</td>
</tr>
<tr>
<td>O—Opening Interdental space between front teeth</td>
<td>&gt;40 mm</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>20–40 mm</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>10–20 mm</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>&lt;10 mm</td>
<td>4</td>
</tr>
<tr>
<td>P—Previous Intubation or OSA</td>
<td>Previous attempt easy</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>No previous attempt, no history OSA</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>OSA, previous history of difficult intubation</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Extremely difficult previous intubation, tracheostomy, or patient unable to lie supine</td>
<td>4</td>
</tr>
<tr>
<td>U—Uvula Mouth open, with tongue protrusion</td>
<td>Tip of uvula visible</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Uvula partially visible</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Uvula concealed soft palate visible</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Soft palate not visible</td>
<td>4</td>
</tr>
<tr>
<td>R—Range Observe line from eye to orbit, estimate range of motion looking up and down</td>
<td>&gt;120°</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>60°–120°</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>30°–60°</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>&lt;30°</td>
<td>4</td>
</tr>
</tbody>
</table>

**Box 1:** Symptoms of cardiac disease

- History of recurrent chest infections
- Tachypnea
- Feeding difficulties: Does the child take a long time to feed or is he distressed on feeding or is there cyanosis
- Cyanosis: Does the child turn blue during feeding, crying, playing
- Is there loss of consciousness or squatting episodes while playing
- Failure to thrive
- Sweating of infant during normal care
Children with symptoms of cardiac disease, e.g. failure to thrive, low exercise tolerance, recurrent respiratory tract infection, etc. should undergo thorough cardiac evaluation. Postchemotherapy, cardiac evaluation should be done. An ECG will aid in diagnosing arrhythmias or conduction defects (Box 2).

Patients with muscular dystrophies should also be subjected to cardiac evaluation as they can have rhythm disturbances, mitral valve prolapse, conduction defects, ventricular hypokinesia, etc. Anesthetic implications of systemic conditions have been summarized in Table 3.

**Box 2: NICE guidelines for infective endocarditis prophylaxis**

**Cardiac conditions at high risk for infective endocarditis (IE):**
- Acquired valvular heart disease with stenosis or regurgitation
- Valve replacement
- Structural congenital heart disease, including surgically corrected or palliated structural conditions, but excluding isolated atrial septal defect, fully repaired ventricular septal defect, or fully repaired patent ductus arteriosus, and closure devices that are judged to be endothelialized
- Previous IE
- Hypertrophic cardiomyopathy

**Antibiotic prophylaxis against IE is not recommended:**
- For patients undergoing dental procedures
- For patients undergoing nondental procedures at these sites: Upper and lower GI tract, genitourinary tract, upper and lower respiratory tract—including ENT procedures and bronchoscopy

**Infection in patients at risk of developing endocarditis:**
- Any episodes of infection should be promptly treated to reduce risk of developing IE
- If a patient is undergoing any gastrointestinal or genitourinary procedure at a site with suspected infection, antibiotic therapy should cover organisms causing IE

**Respiratory**
- Prematurity: Increased risk of postoperative apnea
- Respiratory infection: Reactive airways, bronchospasm
- Bronchopulmonary dysplasia: Lower airway obstruction, reactive airway disease, subglottic stenosis, pulmonary hypertension
- Snoring: Obstructive sleep apnea
- Asthma: Beta agonist or theophylline therapy, steroid use
- Cystic fibrosis: Drug interactions, pulmonary dysfunction and VQ mismatch, reactive airway
- Croup: Subglottic stenosis

**Gastrointestinal**
- Vomiting, diarrhea: Dehydration, electrolyte imbalance, risk of aspiration
- Gastroesophageal reflux disease: Aspiration, repeated upper respiratory infection and reactive airway disease
- Jaundice: Altered drug metabolism, risk of hypoglycemia

**Central nervous system**
- Seizures: Drug interactions of medications, liver effects
- CNS tumors: Elevated intracranial pressure, effects of chemotherapy, chronic steroid use
- Hydrocephalus: Elevated intracranial pressure

**Neuromuscular disease**
- Muscle dystrophy: Malignant hyperthermia, altered response to relaxants

**Renal system**
- Chronic renal failure, patient on dialysis: Electrolyte disturbance, volume status, anemia, hypertension
- Nocturia, increased frequency: Occult diabetes mellitus, urosepsis

**Endocrine**
- Diabetes: Insulin requirement, hypoglycemia, hyperglycemia
- Steroid therapy: Adrenocortical suppression

**Hematology**
- Anemia: Transfusion may be required
- Sickle cell disease: Hydration, tourniquet use, anemia, avoid hypoxia

**Dental**
- Loose tooth: Risk of aspiration of the tooth

**Hypertension in children:** Mild to moderate hypertension does not seem to negatively affect perioperative outcome. Secondary causes of hypertension need to be identified and corrected before surgical intervention. Anti-hypertensive medications should be continued perioperatively. The exceptions to this rule are the angiotensin-converting enzyme inhibitors, which are generally withheld on the day of the procedure.

**PREOPERATIVE INVESTIGATIONS**

Preoperative routine blood testing in healthy children is done at many centers though there is lack of evidence for the same. Blood tests are justified only when there is clinical doubt or clinical evidence of disease. This strategy...
reduces cost and patient discomfort. It also reduces the postponement of surgery and further tests due to false positive results. Routine blood testing in children revealed about 2.5–10% abnormal results which did not have significant impact on scheduled surgery.17-19

**Hemoglobin:** In India, childhood anemia continues to be a significant public health problem in school children and iron deficiency either alone or in combination is the commonest nutritional cause of anemia. Mild anemia is a common finding, but not associated with significant perioperative morbidity.

The sickle cell gene in India was first described among tribal groups in South India. The highest frequency of sickle cell gene in India is reported in Orissa followed by Assam, MP, UP, Tamil Nadu and Gujarat.20 Heterozygous sickle cell trait is unlikely to increase perioperative risks during minor surgery. Severe sickle cell disease is a risk factor for perioperative adverse events because intraoperative hypoxemia, hypercarbia, acidosis, hypothermia, hypovolemia can promote sickling.1 Sickle cell disease is often associated with severe anemia. In severe cases, a decrease of hemoglobin S by means of transfusion or exchange transfusion could be necessary.21 Prior to surgery, it is desirable to have a hematocrit level of 30% and Hb S <30%. Optimal hydration and prevention of hypothermia decreases postoperative morbidity.

**Tests of coagulation:** There is clear evidence that coagulation studies have poor predictive value in detecting occult bleeding disorders or an increased risk of perioperative hemorrhage, though they are often done in detecting inherited diseases, although it can be useful in detecting inherited deficiencies, even if factor XI can be missed, but would not change management for minor surgery. Thus, normal coagulation values, do not completely rule out a coagulation disorder.22

The commonly used prothrombin time tests the extrinsic coagulation pathway while its sensitivity to detect inherited defects is minimal. In contrast, the partial thromboplastin time examines the intrinsic pathway and is therefore more useful in detecting inherited diseases, although it can be normal even in the presence of disease.23,24 History of excess bruising in a child is very subjective, and large bruises, hematomas, simultaneous bruising of several parts of the body or unusual forms of bleeding (frequent, prolonged epistaxis, excess bleeding after minor trauma) are more suggestive of a clotting disorder than bruising itself.

**Urine examination:** Purpose of a urine examination is detection of an undiagnosed renal disorder or urinary tract infection. If clinical assessment is thorough, urine analysis is not always necessary as collection of a clean, noncontaminated specimen is difficult to obtain in pediatric age group.

**Other tests:** Children with renal disease or history of prolonged medication which could lead to water and electrolyte imbalance, could be subjected to further tests like serum electrolytes, renal or liver function tests.

**Chest radiographs:** Chest radiographs rarely reveal clinically important abnormalities which were not already suggested by a thorough history and physical examination. Accordingly, in order to minimize radiation exposure in children, the American Academy of Pediatrics now recommends no chest radiographs unless there is a clear indication that it will have significant impact on the perioperative period.1 Children with cervical lymph nodes suspected to have lymphoma and posted for biopsy, should have chest radiographs as they can have a clinically silent fast growing mass in the anterior mediastinum.

**Pulmonary function testing:** In asthmatic kids, symptoms may not be enough to assess disease severity. In such children of more than 5 years of age, simple noninvasive tests, such as peak flow or forced expiratory volume in one second can be easily performed and help in quantifying severity of respiratory impairment. Spirometry is useful when there is uncertainty about presence of pulmonary impairment which would affect anesthesia, thus one can optimize lung function preoperatively.

The American Society of Anesthesiologists (ASA) has an established risk stratification system mentioned in Box 3. Class 1 and class 2 are considered low risk. The ASA-physical status (PS) is routinely used to assess the preoperative health status and anesthetic risk of adult patients. Recent studies have found only modest agreement among anesthesiologists in assigning ASA-PS to children and there are no data regarding its validity in predicting risk. These data suggest the need for an improved measure of health status and risk for children undergoing anesthesia. The NARCO SS is a pediatric specific risk classification tool. The tool includes 5 systems:

**Box 3: ASA physical status classification system**

- ASA physical status 1: A normal healthy patient
- ASA physical status 2: A patient with mild systemic disease
- ASA physical status 3: A patient with severe systemic disease
- ASA physical status 4: A patient with severe systemic disease that is a constant threat to life
- ASA physical status 5: A moribund patient who is not expected to survive without the operation
- ASA physical status 6: A declared brain-dead patient whose organs are being removed for donor purposes

*Abbreviations: ASA, American Society of Anesthesiologists*
Neurologic, airway, respiratory, cardiovascular, other (NARCO) and a surgical severity score (SS). Comparison of the NARCO with ASA scores and correlation with patient outcomes in 340 pediatric surgical patients, suggested that all measures of outcome have acceptable to excellent reliability with a slight improvement in agreement for the NARCO compared with the ASA-PS.25

LEARNING POINTS

- Optimal preparation of the child is required for safe conduct of anesthesia
- Children need physiological as well as psychological preparation preoperatively
- A detailed history from parents, right from antenatal period is mandatory
- Before detailed physical examination, merely observing the child reveals a lot of information
- Tests should be performed which have direct impact on perioperative management
- Preoperative optimization of any underlying disease is best achieved with a multidisciplinary approach

PREMEDICATION

Preoperative period is a stressful event especially in pediatric age group due to parental separation and limited understanding regarding the illness and reasons for surgery. 50% children have preoperative fear and anxiety, hence primary aim is anxiolysis.26

Advantages
- Reduces patient anxiety
- Provides anterograde amnesia
- Prevents physiological stress
- Parental separation is eased
- Reduces postoperative behavioral changes and adverse outcomes.

Disadvantages
- Noncompliance in the child may worsen anxiety
- Certain drugs like midazolam may cause a paradoxical reaction
- Respiratory depression with coadministration of other drugs, e.g. opioids.

Conditions where One has to be Cautious in Prescribing Premedication
- Anticipated difficult intubation
- Obstructive sleep apnea
- Raised intracranial pressure with altered consciousness
- Previous allergic reaction to sedative premedication
- Risk of aspiration
- Severe systemic illness, e.g. sepsis
- Renal or hepatic impairment.

PREOPERATIVE FASTING

Intake of water and other clear fluid up to two hours before induction of anesthesia for elective surgery is safe in healthy infants and children, and improves patient well-being.27 Clear fluid is the one through which newsprint can be read. The volume of administered fluids does not appear to have an impact on patients’ residual gastric volume and gastric pH when compared to a standard fasting regimen. Therefore, patients may have water and other clear fluid up to two hours before induction of anesthesia. Breast milk may be given up to four hours before induction of anesthesia. Formula milk or cow’s milk may be given up to six hours before induction of anesthesia. A minimum preoperative fasting time of six hours is recommended for food. Chewing gum should not be permitted on the day of surgery. Sweets (including lollipops) are solid food. Regular medication taken orally should be continued preoperatively unless there is advice to the contrary. Up to 0.5 mL/kg (up to 30 mL) of water may be given orally to help children take their medication.

If an elective operation is delayed, consideration should be given to giving the patient a drink of water or other clear fluid to prevent excessive thirst and dehydration. Higher risk patients should follow the same preoperative fasting regime as healthy infants and children, unless contraindicated. Patients undergoing emergency surgery should be treated as if they have a full stomach. The routine use of H2 receptor antagonists is not recommended for healthy children.

PSYCHOLOGICAL PREPARATION OF THE CHILD

Seventy five percent of children exhibit significant psychological and physiological manifestations of anxiety during the perioperative period. They are worried about parental separation, pain, injections, strange environment with unfamiliar faces. The older child has anxiety about going to sleep and the surgical process. The younger child is more worried about parental separation. Psychological preparation programs were introduced about 50 years ago for preparing the child by letting him observe other children who underwent similar procedures. Since then these programs have been modified and improvised for best outcomes.
Currently, the programs in use are narrative preparation, operating room tours, play therapy, printed material provision, etc. The most comprehensive is child-life preparation and relaxation. These programs may not be effective during induction.

Parental presence during induction is a suggested alternative to preoperative sedation. Benefits include reduction in need of preoperative sedatives, minimizing the child’s anxiety on separation and improving his compliance. Majority of the parents feel satisfied to be with their child. This is a common practice in the western world though not here in India. There have been isolated reports of disruptive behavior in children due to presence of parents.

**Nonpharmacological Measures to Reduce Anxiety**

*Music therapy:* Anxiolytic effects of music therapy have been well established in adults. Anxiety in a child depends on personality factors, noise levels in the operating room, bright lights and number of people interacting with the child. Kain et al. showed that combination of music, dim lights and only the attending anesthesiologist interacting with the child was effective in reducing anxiety at the time of induction. A recent study by Kain et al. gave contrary results and concluded that anxiolysis was achieved in the holding area and separation, but not effective for induction.

*Acupuncture:* Wang et al. studied the effects of acupuncture in mothers of children posted for surgery. Children of mothers who were subjected to acupuncture therapy were less anxious than those who were in control group.

*Preoperative interview:* Preoperative interview is a good psychological intervention. Some parents ask for relevant information, some have information seeking nature and some have information avoiding coping style. The anesthesiologist has to be discreet in choosing which parents need to be provided with a detailed anesthetic information.

**Pharmacological Measures**

*Benzo diazepines*

*Midazolam:* It is the most common premedicant administered in the preoperative holding area. As an oral preparation it has a short onset and offset of action. Oral midazolam has been shown to be more effective in reducing a child’s anxiety than parental presence and parental presence combined with oral midazolam was not superior in reducing a child’s anxiety than sedation alone.

Higher doses of midazolam appear not to offer any additional benefits, and may cause more side effects, such as paradoxical response. Child may become restless, agitated and dysphoric. Commercially, prepared midazolam formulation is rapidly absorbed with patients demonstrating a satisfactory degree of sedation and anxiolysis within 10 minutes of consumption with a higher percentage at 20 minutes. A bitter taste has been described after oral and nasal administration. The dose of oral midazolam should be adjusted in children taking depressants or inducers of the cytochrome oxidase system, such as anticonvulsants or barbiturates (Table 4).

As a standard syrup preparation, it exists in both an open and closed ring structure, the proportion of which is pH dependent. At lower pH values there is a greater proportion of drug in the open ring, and at higher pH values there is a reduction in the proportion of the drug in the open ring configuration. Since only the open ring formulation is lipophilic and physiologically active, bioavailability is highly sensitive to changes in pH. Therefore, the combination of any “home made” diluents with the intravenous midazolam formulation could significantly alter both the absorption rate and the bioavailability. Midazolam has the advantage of producing anterograde amnesia. Memory usually becomes impaired within 10 minutes after oral midazolam.

*Diazepam:* Diazepam is an unpopular choice as a preoperative premedicant in young children because of immature liver function that would lead to a prolonged half life. The average oral dose for premedicating healthy children with diazepam ranges from 0.1 to 0.3 mg/kg. It is metabolized to desmethyldiazepam with a pharmacologic activity equal to the parent compound.

*Lorazepam:* May be administered orally, intravenously, or intramuscularly and is metabolized by the liver to inactive metabolites. The intravenous formulation has been reported to be neurotoxic in neonates. It has a slow onset and offset of action and so better to use in inpatients. Usual oral or IV dose in older children is 0.05 mg/kg, but a dose of 0.025 mg/kg is good enough to reduce preoperative anxiety.

**Table 4:** Routes of administration and doses of midazolam in children

<table>
<thead>
<tr>
<th>Route of administration of midazolam</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intravenous</td>
<td>0.025–0.1 mg/kg</td>
</tr>
<tr>
<td>Intramuscular</td>
<td>0.1–0.2 mg/kg</td>
</tr>
<tr>
<td>Intranasal</td>
<td>0.2 mg/kg</td>
</tr>
<tr>
<td>Rectal</td>
<td>1 mg/kg</td>
</tr>
<tr>
<td>Oral</td>
<td>0.25–0.75 mg/kg</td>
</tr>
<tr>
<td>Sublingual</td>
<td>0.2 mg/kg</td>
</tr>
</tbody>
</table>
Ketamine
A phencyclidine derivative, antagonizes the N-methyl-D-aspartate (NMDA) receptor and causes dissociation of cortex from the limbic system. Antisialogogue should be given along to reduce the secretions and laryngospasm. Co-administration of benzodiazepines reduces incidence of hallucinations and emergence delirium. Duration of action of a single IV dose is 5–8 minutes (α-elimination half-life of 11 minutes and a β-elimination half-life of 2.5–3.0 hours). Ketamine is administered in very low doses intravenously (0.25–0.5 mg/kg) or intramuscularly (1–2 mg/kg) either alone or preferably in combination with low-dose midazolam (0.05 mg/kg) along with atropine (0.02 mg/kg) for sedation.

Bioavailability of ketamine is approximately 93% after intramuscular administration. The intramuscular route of administration is very useful for children who are uncooperative, refuse oral sedation and become combative. These children become adequately calm in around 3 minutes and accept a mask inhalation induction of anesthesia. Ketamine has also been administered via oral, nasal and rectal routes. After oral administration and nasal administration, bioavailability is 17% and 50%, respectively. The combination of oral ketamine (3 mg/kg) and midazolam (0.5 mg/kg) does not seem to prolong recovery time for procedures longer than 30 minutes.

Nasal transmucosal ketamine at a dose of 6 mg/kg is also effective in sedating children within 20–40 minutes before induction of anesthesia. Only preservative free ketamine should be given nasally to avoid neurotoxicity. The 50 mg/mL concentration is preferable to minimize the volume for nasal administration. Rectal ketamine (5 mg/kg) produces anxiolysis and sedation within 30 minutes of administration.

Opioids
Opioids may be used as a preanesthetic medication to children with preoperative pain; however, one has to be watchful about opioid-related side effects, such as respiratory depression, dysphoria, pruritus and nausea/vomiting. If opioids are used in combination with other sedatives such as benzodiazepines, the dose of each drug should be appropriately adjusted to avoid serious respiratory depression. Opioids are not advisable in neonates. Fentanyl may be administered by parenteral, transdermal, nasal and oral routes. Oral dose is 10–15 µg/kg, IV and transnasal is 1–2 µg/kg.

Sufentanil is 10 times more potent than fentanyl. Nasal sufentanil, in kids, has a higher incidence of reduced chest wall compliance, nausea and vomiting and a prolonged discharge time when compared to nasally administered midazolam. These potential side effects and prolonged hospital stay after nasal sufentanil makes it an unpopular choice for premedication.

Nonbarbiturate Sedatives
Chloral hydrate: It is a nonbarbiturate sedative given orally or rectally in a dose of 20–75 mg/kg, maximum up to 2 grams. It is devoid of analgesic properties, has a bitter taste, prolonged elimination half life and slow onset. It gives a good sedation within 30–45 minutes. It is not advised in neonates and child with liver disease. The metabolites, namely trichloroacetic acid and trichlorepethol may lead to metabolic acidosis, renal failure and hypotonia. It can cause airway obstruction in children with large tonsils. Deaths after chloral hydrate sedation have been reported.

Anticholinergic Drugs
Anticholinergic drugs are useful for their vagolytic, antisialogogue and central sedative effects. They were initially used when inhalational anesthetics caused severe bradycardia. Current inhalational anesthetics are not associated with bradycardia and do not stimulate salivary or tracheobronchial secretions, therefore, the routine use of an anticholinergic drug is not recommended. The recommended doses of anticholinergics are scopolamine, 0.005–0.010 mg/kg atropine, 0.01–0.02 mg/kg and glycopyrrolate 4µg/kg IV, IM. Atropine is more commonly used and is a better vagolytic agent than scopolamine, whereas scopolamine is a better sedative, antisialalagogue, and amnestic. Glycopyrrolate is the only agent that does not cross the blood-brain barrier.

α2 Agonists
Clonidine, an α2 agonist, causes dose-related sedation by its effect in the locus ceruleus through its inhibition of adenylyl cyclase. The plasma concentration peaks at 60–90 minutes after oral administration and at 50 minutes after rectal administration. The need to administer clonidine 60 minutes before induction of anesthesia makes its use impractical in most clinical settings. An oral dose of 3 µg/kg given 45–90 minutes before the surgery produces comparable sedation to that of diazepam or midazolam.

Dexmedetomidine is a highly selective alpha-2 agonist that provides sedation, anxiolysis and analgesia without causing respiratory depression. Recently, it has been explored extensively in the pediatric population. In the dose of 1–2 µg/kg through transmucosal routes (intranasal, sublingual and buccal) or 2.5–4 µg/kg orally, 30–75 minutes prior to surgery, it provides satisfactory separation from parents and reduces postoperative analgesic requirement.
Antihistaminics

As their sedative effects are variable, they are not commonly used. Promethazine in an oral dose of 0.25–0.5 mg/kg has the advantage of being an effective sedative, antiemetic, antimotion sickness, and an anticholinergic, but due to dystonic reactions it is unpopular premedicant in children.

Topical Anesthetics

EMLA cream: It is eutectic mixture of two local anesthetics (2.5% lidocaine and 2.5% prilocaine). One-hour prior application of EMLA cream to intact skin with an occlusive dressing provides adequate topical anesthesia for an intravenous catheter insertion. It causes venuconstriction and skin blanching, making intravenous cannulation more difficult. Methemoglobinemia may occur secondary to prilocaine. However, a 1-hour application of EMLA cream and a maximum dose of 1 g did not induce methemoglobinemia when applied to intact skin of full-term infants younger than 3 months of age.

Other topical anesthetics available are 4% tetracaine with onset time of 30–40 minutes. There is no risk of methemoglobinemia, skin blanching or venuconstriction. 4% lignocaine cream is also available which dilates veins better.

LEARNING POINTS

- Psychological preparation programs are devoid of harmful effects and are effective in allaying anxiety and preoperative stress. It is also an opportunity to win the confidence of the parents or guardians accompanying the child
- Sedative drugs should be provided in a safe environment where resuscitation equipment and drugs can be easily accessed
- A sedated child should be appropriately monitored at all times, including when transferred from the day surgery or inpatient area to the pre-operative holding area and through to operating theatre
- Intranasal route has a quicker onset and may be used when oral premedications are refused. Drug administered via this route may traverse directly into the central nervous system through the cribiform plate via the olfactory nerves. So, preservative free drugs should be administered
- Successful outcome is possible by effectively dealing with physiological, emotional and behavioral aspects in a child

REFERENCES

Chapter 6: Preoperative Evaluation and Premedication


**INTRODUCTION**

Transfer to the operation theater and induction of anesthesia are stressful for children. They are taken away from their parents by strangers to an unfamiliar, intimidating place full of bright lights and strange noises where a frightening mask is put on their face. This can be extremely disturbing and fearful for most children. Pediatric anesthesiologists must strive to alleviate these fears and make this experience as comfortable and pleasant as possible.

**PREPARATION**

Before transferring the child to the operation theater (OT), the anesthesiologist must ensure that the child is prepared for the surgery, a written consent is signed by the parents and the OT is ready (Box 1). Once inside the OT, the child should not be made to wait.

**INDUCTION OF ANESTHESIA**

It is difficult to predict the behavior of the child at the time of transfer and induction of anesthesia. It depends upon the child's age, temperament, previous experience, current state of mind and the preoperative preparation. The anesthesiologist should, therefore, be flexible. She should reassess the child just before transfer and modify the anesthetic plan to suit the child. She must acquire the skill of communicating with children of all ages for smooth induction of anesthesia. She should be caring, sensitive and gentle, and make every effort for the induction of anesthesia to be pleasant for the child. A stormy induction is a traumatic experience for the child and may result in immediate postoperative and long-term behavioral problems. Some of the methods that can be used to make parental separation and induction of anesthesia smooth are given in Box 2. Parents are allowed to be present during the induction of anesthesia in some hospitals. However, parental presence is beneficial only if an anxious child is accompanied by a calm parent.¹

**Choice of Induction Technique**

The child’s preference regarding the method of induction should always be taken into consideration. In general, anesthesiologists use inhalational induction for older children who object to an injection, small children and infants. The child should be warned about the odor

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**Box 1: Preparation required before the start of anesthesia**

- Check the fasting status of the child
- Ensure premedication has been given if required and adequate time has elapsed for its optimal effect
- Review the preanesthetic evaluation regarding any special concerns
- Confirm child’s assent (whenever applicable) and written parental consent for the procedure
- Confirm OT readiness
  - Anesthetic, surgical and nursing teams
    - Personnel
    - Drugs
    - Equipment
  - OT environment
    - Cleanliness
    - Temperature
MAC is the minimum alveolar concentration of an anesthetic gas at 1 atmospheric pressure which prevents movement on noxious stimulus in 50% of subjects.

**Table 1:** Properties of commonly used inhalational anesthetic agents

<table>
<thead>
<tr>
<th></th>
<th>Boiling point (°C)</th>
<th>Blood-gas partition coefficient</th>
<th>Brain-blood partition coefficient</th>
<th>MAC: Adults / Infants / Children</th>
<th>Metabolism (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nitrous oxide</td>
<td>-88</td>
<td>0.47</td>
<td>1.1</td>
<td>104</td>
<td>0.004</td>
</tr>
<tr>
<td>Halothane</td>
<td>50</td>
<td>2.40</td>
<td>1.9</td>
<td>0.76</td>
<td>15-20</td>
</tr>
<tr>
<td>Isoflurane</td>
<td>48</td>
<td>1.40</td>
<td>1.6</td>
<td>1.2 / 1.7 / 1.6</td>
<td>0.2</td>
</tr>
<tr>
<td>Sevoflurane</td>
<td>57</td>
<td>0.65</td>
<td>1.7</td>
<td>2.0 / 3.3 / 2.5</td>
<td>3</td>
</tr>
<tr>
<td>Desflurane</td>
<td>23</td>
<td>0.45</td>
<td>1.3</td>
<td>6.0 / 9.4 / 8.0</td>
<td>0.02</td>
</tr>
</tbody>
</table>

Note: MAC is the minimum alveolar concentration of an anesthetic gas at 1 atmospheric pressure which prevents movement on noxious stimulus in 50% of subjects.

**Box 2: Methods for smooth induction of anesthesia**

- Prepare the child and her parents psychologically by describing what to expect
- Premeditate the child if appropriate
- Allow a parent of an anxious child to be present during induction of anesthesia. However, this is helpful only if the parent is calm
- Make friends with the child and accompany her during the transfer to the OT. Carry an infant or a small child yourself. Transfer an older child on a trolley if premedicated, otherwise walk her to the OT
- Distraction during parental separation and induction decreases the child’s anxiety. Talk to the child during transfer and induction. Use toys, videos, games, cartoons, clowns, stories, etc. as available
- Do not restrain the child physically, but make sure she does not hurt herself
- Most unsedated toddlers and small children prefer to sit. Seat them on the OT table with their back supported against your chest till they are asleep
- Some infants cry when placed on the OT table. Keep them in your lap till they fall asleep and then transfer them to the table
- Ways to make the face mask more acceptable:
  - Use scented masks
  - Hold the mask a little away from the face
  - Use a cupped hand in place of a mask
  - Use 50% nitrous oxide along with inhalational anesthetic
  - Let the child hold the mask herself
  - Let infants suck a pacifier or your gloved finger during induction to prevent crying
  - Ask older children to “blow up the balloon” or “breathe oxygen through a pilot’s mask”

Inhalational Induction

The MAC of inhalational agents varies with age for unknown reasons. It increases from preterm to term neonates to infants and then decreases to adult values by 1–2 years (Table 1). Induction of anesthesia is faster in children than in adults due to a higher respiratory rate and cardiac index and a greater perfusion of vessel-rich organs.

Sevoflurane and halothane are the inhalational agents used for induction of anesthesia in children. Isoflurane and desflurane are unsuitable as they are pungent and cause airway irritation. Sevoflurane is preferred over halothane as it leads to faster induction due to a lower blood-gas partition coefficient (Table 1) and causes less cardiovascular depression. If the child is cooperative and accepts the mask, the inhaled concentration is increased incrementally till the child sleeps. For rapid induction in children older than a year, 8% sevoflurane can be used from the outset. Some anesthesiologists add nitrous oxide to the carrier gas in a concentration of 50–70%. Being odorless, nitrous oxide is well-tolerated by children. It increases the speed of induction due to its own anesthetic effect and the increased uptake of the inhalational agent due to second gas and concentration effects. Once the child is asleep, nitrous oxide is stopped and intravenous access is secured. If the child is sleeping when transferred to the OT, she is not disturbed and a ‘steal induction’ is carried out. The mask is placed close to the face without contact and the anesthetic concentration gradually increased. Once the child is anesthetized, the mask is held on the face.

As the depth of anesthesia increases, the tone of pharyngeal and laryngeal muscles decreases and may lead to upper airway obstruction. If jaw thrust and chin lift do not relieve the obstruction, CPAP of 5–10 cm H₂O can be used to splint the airway patent.

Controlled ventilation with a high concentration of volatile agents, especially halothane, can result in cardiovascular collapse and cardiac arrest. Therefore, once the child is anesthetized, the inspired concentration is reduced to 1–1.5% halothane or 3–4% sevoflurane before controlled ventilation is started.

Both sevoflurane and halothane cause dose-dependent respiratory depression. Sevoflurane causes tachycardia in older children. Halothane causes systolic hypotension.
echolamine-induced arrhythmogenicity and myocardial depression are less with sevoflurane than halothane. Premature and term neonates in that order are more sensitive to cardiac depression than older children due to myocardial immaturity and lower calcium stores. Excitation, abnormal movements, coughing, breath holding and laryngospasm can occur during induction with either agent.

**Intravenous Induction**

**Intravenous Cannulation**

EMLA (eutectic mixture of local anesthetics, 2.5% of prilocaine and lignocaine each) cream provides good topical anesthesia for painless IV cannulation. However, it takes 45–60 minutes to act and also causes vasoconstriction. It is contraindicated in children with methemoglobinemia or G6PD deficiency. Iontophoresis of lignocaine provides topical anesthesia in 5–15 minutes, but is not popular because it causes local burning sensation, petechiae, ecchymosis, stinging and itching.

**Intravenous Drugs**

**Propofol:** The duration of action of propofol may be increased in neonates and small infants due to delayed clearance caused by their immature liver enzyme system. Propofol does not affect cerebral blood flow, intracranial pressure or myocardial contractility. It decreases vascular tone and depresses ventilation. It reduces the incidence of postoperative vomiting. The major side-effects are shown in Table 2. Pain on injection of propofol can be minimised by injecting lignocaine (0.5–1 mg/kg) prior to or mixed with propofol into a large antecubital vein. Propofol should be avoided in children with known anaphylactic reaction to eggs.

**Thiopentone:** Thiopentone acts rapidly. It is used as a 2.5% solution in neonates and 1% in infants and neonates. Thiopentone does not increase the cerebral blood flow and decreases the intracranial pressure. It depresses respiration, myocardium and vascular tone.

**Ketamine:** Ketamine produces dissociative anesthesia and analgesia. Because of a high incidence of side-effects its use is usually reserved for children with cyanotic heart disease and preoperative shock (Table 2). The incidence of emergence agitation can be reduced by the concomitant use of benzodiazepines. Since it increases secretions, an antisialagogue agent, such as glycopyrrolate is coadministered.

**Etomidate:** Etomidate is usually used in children with cardiovascular compromise as it causes no adverse effects on the cardiovascular system.

**Modified Rapid Sequence Induction and Intubation**

The incidence of pulmonary aspiration is low in children. It usually occurs when the child is with full stomach and is not adequately anesthetized, or is coughing, straining or retching during induction and intubation. Rapid sequence induction and intubation should be performed by an experienced pediatric anesthesiologist in an adequately anesthetized and paralyzed child.

The child should be pre-oxygenated if possible. A large pre-calculated dose of thiopentone or propofol is administered followed by succinylcholine or rocuronium. Cricoid pressure can distort the airway causing difficult ventilation and intubation and is not recommended. Gentle mask ventilation is carried out with the airway open and the pressure limited to 12 cmH₂O. The trachea is intubated after 60 seconds and the cuff inflated (if applicable) before recommencing ventilation.

**AIRWAY EQUIPMENT AND TECHNIQUES**

**Suction**

Availability of a wall suction outlet or an electrically powered suction machine should be ensured. A rigid Yankauer suction tip is used to remove thick secretions, blood or particulate matter and a flexible suction catheter for thin secretions and suction through an airway device. Prolonged suction at any one point and too high a suction pressure in small children should be avoided.
Face Masks

A correctly sized mask covers the nose and mouth from the nasal bridge above to the groove between lower lip and chin below, while leaving the eyes uncovered. Masks of different shapes are needed to fit the varied facial contours of children (Fig. 1). Rendell-Baker Soucek masks fit well in neonates and infants and have low dead space. Transparent masks allow rapid detection of secretions and vomitus.

The mask is positioned on the child’s face, the jaw is elevated with the last three fingers, and the mask is pressed down with the thumb and index finger to make a tight seal. The fingers should not compress the submandibular soft tissue to avoid airway obstruction. The mouth is kept slightly open to avoid the tongue sticking to the palate and causing obstruction.

Oropharyngeal and Nasopharyngeal Airways

These devices are used to relieve airway obstruction if head-tilt and jaw thrust are not successful. Oropharyngeal airway is used in children if the airway reflexes are absent. The length of the airway should equal the distance between the angle of the mouth and the angle of the mandible. It is introduced with the concavity facing the palate and rotated by 180° after it has crossed the tongue.

Nasopharyngeal airway can be tolerated by children with intact airway reflexes. The length of the airway should be equal to the distance between the tip of the nose and the tragus and the diameter of the child’s little finger. The airway should be lubricated well and advanced gently through the nostril in a posterior direction.

Supraglottic Devices

Supraglottic devices (SGDs) can be used for short (1–2 hour) surgical procedures not involving a body cavity. They can also be used as a rescue device and as a conduit for tracheal tube in patients with difficult airway. The second generation devices have an additional lumen allowing gastric decompression and reducing the risk of regurgitation. Many SGD are now available in pediatric sizes including classic, flexible, AMBU, Proseal, i-gel, laryngeal tube suction II and air-Q intubating laryngeal airway (Table 3 and Fig. 2).

SGDs are introduced in a deep plane of propofol or inhalational anesthesia usually without a neuromuscular blocking drug (NMBD). A relaxed jaw is an approximate indicator of adequate depth of anesthesia. An SGD of appropriate size (Table 4) is introduced with the patient in supine position and the head extended. In the classic technique for introduction, a fully deflated classic LMA is held like a pen with the index finger extended and introduced along the palate with its dorsal surface facing the palate till the finger can go in no more. It is then pushed in further with the other hand till it meets resistance. The cuff is inflated with the recommended amount of air. A correctly placed device moves out of the mouth slightly with inflation. The intracuff pressure should be less than 60 cmH₂O. Alternative techniques include insertion of LMA with a partially inflated cuff, with the dorsal surface facing laterally or towards the tongue.

The correct placement of SGD is confirmed by the ability to ventilate adequately (determined clinically and by ETCO₂) without inflating the stomach, or by fiberoptic bronchoscopy. Selection of an SGD of an appropriate size, adequate depth of anesthesia and proper technique

![Fig. 1: Commonly used face masks: (A) Transparent anatomical mask; (B) Anatomical mask; (C) Rendell-Baker Soucek mask; (D) Round infant mask; (E) Scented mask](image1)

![Fig. 2: Commonly used supraglottic devices: (A) i-gel; (B) Proseal LMA; (C) AMBU LMA; (D) Classic LMA](image2)
increase the chances of successful placement. Neonates and small infants usually have higher failure rates. SGDs are frequently displaced during surgery. This can be minimized by maintaining an adequate depth of anesthesia throughout the anesthetic and avoiding movement of the head and neck.

**Tracheal Tubes**

Both cuffed and uncuffed tracheal tubes (TT) are used in children at present. Traditionally, uncuffed TT were preferred in children younger than 6 years based on the following assumptions: cricoid cartilage is the narrowest part of the conical larynx; a reasonable seal can be made at this level by uncuffed tubes; cuffed tubes resulted in placement of smaller tubes; and the cuff caused difficulty in positioning of the tube within the trachea. We now know that the narrowest portion is the glottis or the immediate subglottis which is elliptical in cross-section with a longer antero-posterior diameter. Also, TTs designed for children with thin, short and distally placed polyurethane cuffs (Microcuff tracheal tubes, Fig. 3) are available now. These changes have led to the successful use of cuffed TTs even in small children. The minimal leaks allow better ventilation, oxygenation, and monitoring of ventilation and anesthetic gases, with decreased consumption of gases and contamination of the environment. Use of cuffed tubes also reduces the attempts needed to place a TT of the correct size. The major disadvantages are the increased risk of damage to tracheal mucosa and high cost. Whenever cuffed tracheal tubes are used, intracuff pressure should be monitored and maintained at < 20–30 cm H₂O to minimize tracheal damage.

Length-based formulas for selecting the size of TT are more accurate than age-based formulas. Nevertheless, the age-based modified Cole's formula is the commonest formula in use: Inner diameter of the uncuffed tube in mm = (age/4) + 4. A cuffed tube would be one size smaller. A 3.0 mm tube is used in a term neonate and 2.5 mm in a preterm neonate weighing <1500 g. Tubes of three sizes should be kept ready on the airway trolley: calculated size, one size smaller and one larger. A TT of appropriate size.
size should allow a small leak at an airway pressure of 20 cmH₂O.

The tip of a correctly positioned TT lies in mid-trachea, preventing both accidental extubation and endobronchial displacement due to extension or flexion of the child’s head and neck. Most TTs have black marks or bands. When these are positioned at the level of vocal cords under vision, the TT is correctly positioned within the trachea. The position of the TT should always be confirmed by auscultation of the chest and by observing the ventilation parameters (tidal volume and peak airway pressure) after both tracheal intubation and any change in position of the patient.

**Drugs Used for Tracheal Intubation**

Succinylcholine is the only depolarizing NMBD in use nowadays. It provides good intubating conditions in about 60 seconds with a short duration of action (Table 5). In the absence of intravenous access it can be given intramuscularly (4 mg/kg; onset 3–4 minute, duration 20 minute) or intralingually in an emergency. However, its use has declined due to its adverse effects including raised intracranial, intraocular and intragastric pressure. It can cause rhabdomyolysis, hyperkalemia and sudden cardiac arrest in children with muscular dystrophy and other neuromuscular conditions with increased extra-junctional acetylcholine receptors. It can trigger masseter spasm and malignant hyperthermia in susceptible children. It is also associated with bradycardia and sinus arrest usually after a second dose. Its present indications are laryngospasm, rapid sequence intubation and difficult airway.

Nondepolarizing NMBD can also be used for tracheal intubation, provided that they are required intraoperatively, and a difficult airway is not anticipated. If NMBD are not indicated, tracheal intubation can be performed in a deep plane of anesthesia. This can be achieved by any of the following combinations: sevoflurane + propofol (2 mg/kg), sevoflurane + remifentanil (1–2 µg/kg), sevoflurane + lignocaine (2 mg/kg), or propofol + remifentanil (4 µg/kg).¹⁰

**Technique of Tracheal Intubation**

Extension of the head without a pillow underneath is usually adequate for tracheal intubation in children up to the age of 8 years. Because of their large heads, a small roll beneath the shoulders is advantageous in neonates and infants. A head ring can be used to stabilize the head, but it should be ensured that it does not flex the head and exaggerate the natural attitude of flexion, which can make laryngoscopy and intubation difficult.

**Laryngoscopes**

A complete range of laryngoscopes with blades of sizes 0, 1, 2 and 3 should be available. Straight blade laryngoscopes (Miller, Wisconsin) are usually preferred to lift the epiglottis in neonates and infants, as it is omega-shaped and angled over the cephalad placed larynx. Both curved (Macintosh) and straight blades are used for older children.

Videolaryngoscopes have a high-resolution wide-angle digital camera near the tip of the laryngoscope blade to provide a good view of the glottis. Glidescope, Storz DCI and Airtraq optical laryngoscope are available in pediatric sizes. Truview has a prism and a lens system instead of a camera. The utility of these devices is limited by their size in patients with decreased mouth opening: Airtraq 12–13 mm, Glidescope 10 mm, Storz DCI 5 mm, and Truview 8 mm. Individual devices require different techniques of usage.

**Breathing Systems**

Non-rebreathing systems (e.g. Ayre’s T piece with Jackson-Rees modification) are commonly used in children, as they allow rapid equilibration of anesthetic agents, offer low resistance and permit manual assessment of lung compliance. However, they require high fresh gas flows.

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**Table 5: Doses and chief routes of elimination of neuromuscular blocking drugs**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Loading dose (mg/kg)</th>
<th>Re-injection dose (mg/kg)</th>
<th>Infusion rate (µg/kg/minute)</th>
<th>Metabolism</th>
<th>Elimination</th>
</tr>
</thead>
<tbody>
<tr>
<td>Succinylcholine</td>
<td>1–2</td>
<td></td>
<td></td>
<td>Butyrylcholinesterase</td>
<td></td>
</tr>
<tr>
<td>Atracurium</td>
<td>0.4–0.8</td>
<td>0.1–0.2</td>
<td>5–15</td>
<td>Hofmann degradation &amp; ester hydrolysis</td>
<td>Liver and kidney</td>
</tr>
<tr>
<td>Vecuronium</td>
<td>0.05–0.1</td>
<td>0.02</td>
<td>1.0–1.2</td>
<td>Liver</td>
<td>liver and kidney</td>
</tr>
<tr>
<td>Rocuronium</td>
<td>0.4–0.6</td>
<td>0.1–0.15</td>
<td>5–15</td>
<td>Minimal metabolism</td>
<td>Liver and kidney</td>
</tr>
<tr>
<td>Cis-atracurium</td>
<td>0.1–0.2</td>
<td>0.03–0.5</td>
<td>1–3</td>
<td>Hofmann degradation &amp; ester hydrolysis</td>
<td>Liver and kidney</td>
</tr>
</tbody>
</table>
do not allow monitoring of airway pressure and tidal volume, and cause OT pollution.

Rebreathing systems with CO$_2$ absorbers can take a longer time to equilibrate, have a higher circuit compliance leading to loss of volume and inaccurate manual assessment of lung compliance. However, low fresh gas flows can be used, ventilatory parameters can be monitored and OT pollution is less.

**MONITORING AND DOCUMENTATION**

It may not be possible to establish all the monitoring before the start of anesthesia in children. The pulse oximeter probe is the only monitor that an awake child usually allows. ECG electrodes and noninvasive blood pressure cuff can be placed after the child is asleep and temperature probe after the airway device is in position. Monitoring of ETCO$_2$, inhalational agents, and neuromuscular junction is also recommended. Precordial and esophageal stethoscopes are simple but useful monitoring devices in children. Invasive monitoring is governed by the child’s condition and the surgical procedure. All the monitored parameters, intraoperative drugs, fluids and events should be documented contemporaneously in the anesthesia record, usually every 5–10 minutes.

**MAINTENANCE OF ANESTHESIA**

This phase of anesthesia consists of maintaining an appropriate depth of anesthesia, preventing awareness, providing adequate analgesia, amnesia and immobility, maintaining hemodynamic stability, adequate ventilation and normothermia. A balanced technique of anesthesia is commonly used with a combination of inhalational or intravenous anesthetic agents, opioids and/or a regional block for analgesia and a neuromuscular blocking agent as required.

**Inhalational Anesthetics**

Fifty to seventy percent nitrous oxide is commonly used with oxygen as it decreases the amount of other inhalational agent required. It is odorless and rapid in onset and recovery. However, it causes expansion of air-filled cavities, increases the incidence of postoperative nausea and vomiting, depresses methionine synthase and causes atmospheric pollution.

The use of halothane has declined as safer alternatives have become available. The adverse effects of halothane include arrhythmias, cardiovascular and respiratory depression, delayed recovery and a high metabolism (Table 1). In addition, halothane vaporizers are capable of delivering dangerously high doses.

Sevoflurane is less arrhythmogenic and myocardial depressant. Nephrotoxicity has not been reported, though about 5% of the drug is metabolized to inorganic fluoride. Prolonged use in the presence of a desiccated CO$_2$-absorbent can result in the production of compound A which is nephrotoxic in experimental animals. Though such toxicity has not been observed in humans, use of gas flows <1 L/min is not recommended.

Isoflurane and desflurane decrease the blood pressure, but not the cardiac output as they decrease the SVR without myocardial depression. They may cause tachycardia without arrhythmias. They do not sensitize the myocardium to catecholamines. Both depress ventilation. Desflurane produces bronchoconstriction at >1 MAC, while all others cause bronchodilatation.

**Intravenous Anesthetics**

Total intravenous techniques deliver controllable anesthesia with no OT pollution. Propofol is an ideal agent for this. Its other benefits include decreased PONV, airway reactivity, and emergence agitation, faster and better recovery, and amnesia. It is the anesthetic of choice in patients susceptible to malignant hyperthermia and rhabdomyolysis, during airway endoscopy, and for sedation during transport. However, the cost-effectiveness of total intravenous techniques in routine practice has been questioned.

Infants less than 6 months of age require a larger dose of propofol in the first 30 minutes (due to a larger central compartment) and a lower infusion rate later (due to slower metabolism in the immature liver) compared to older children and adults. Manual or computer-controlled target controlled infusion (TCI) can achieve an effect site concentration of 3 µg/mL in children older than 3 years. One suggested manual regimen consists of a bolus of 2.5 mg/kg, followed by an infusion of 15 mg/kg/h for 15 minute, 13 for the next 15 minute, 11 for 30–60 minute, 10 for 1–2 hour, and ending with 9 mg/kg/hour for 2–4 hour. Computer-controlled techniques may be open- (no feedback from the patient), or closed-loop (usually with processed-EEG feedback).

Prolonged propofol infusion at a high rate can occasionally cause propofol infusion syndrome characterized by lactic acidosis, rhabdomyolysis, renal and cardiac failure. Propofol can also cause hypertriglyceridemia, myocardial depression and peripheral vasodilatation. It can be contaminated by ambient bacteria. Lack of intravenous access, unavailability of appropriate infusion pumps and the unreliability of processed EEG in children are other limitations of the technique.
Chapter 7: Induction, Maintenance and Emergence

Analgesia
Multimodal analgesia using different drugs and techniques is commonly used.

Opioids
The large inter-patient variability of opioid response has to be kept in mind during their use.

**Morphine:** An initial bolus of 50 and 100 μg/kg is used in infants and older children followed by 25 μg/kg titrated to clinical effect. Respiratory depression is more common in neonates and small infants due to increased permeability of the blood-brain barrier and decreased clearance of the drug.

**Fentanyl:** Fentanyl with its rapid onset and short duration of action, is the most commonly used opioid in neonates and small children. Increased intra-abdominal pressure, PEEP and vasopressors can reduce its elimination by decreasing hepatic blood flow. The usual initial bolus dose is 1–2 μg/kg.

**Remifentanil:** Remifentanil has a very short half-life even after prolonged infusion in neonates and infants. It shows little inter-patient variability. It can be associated with bradycardia and hypotension which are amenable to vagolytic drugs. 5 μg/mL concentration is used in infants and small children. In older children, 50 μg/mL can be used. Initial rates of IV infusion are 0.1 μg/kg/min in neonates and infants, and 0.4 μg/kg/min in older children. It is important to administer another analgesic before terminating the remifentanil infusion to avoid breakthrough pain.

Nonopioid Analgesics
Paracetamol and nonsteroidal antiinflammatory drugs (NSAIDs) are part of the perioperative multimodal analgesic regimen. The antiinflammatory, antipyretic and analgesic paracetamol is administered intravenously in a dose of 15 mg/kg 6 hourly. The safe dose of paracetamol in neonates, infants and children weighing <10 kg is 7.5 mg/kg with a maximum daily dose of 30 mg/kg.13

Among NSAIDs ibuprofen, diclofenac and ketorolac are commonly used. Their effect on platelet function and increased postoperative bleeding is still controversial.

Regional Anesthetic Techniques
Wound infiltration, plexus and nerve blocks, and neuraxial blocks are used wherever feasible and safe. Their popularity is increasing due to the availability of ultrasound guidance. Peripheral nerve blocks are associated with fewer life threatening complications compared to neuraxial blocks. The total dose of the local anesthetic should be calculated carefully based on the child’s age and weight to avoid systemic toxicity. Use of adjuvants can decrease the dose of local anesthetic required and prolong the duration of postoperative analgesia. Adrenaline, opioids, clonidine and ketamine are commonly used.

Neuromuscular Blocking Drugs (NMBD)
Succinylcholine is not recommended for maintenance because of its side-effects including phase II block. Intermediate- to long-acting atracurium, vecuronium, rocuronium and cis-atracurium are the commonly used nondepolarising NMBD. Their doses, metabolism and routes of elimination are shown in Table 5. They can also be administered as continuous infusion during long surgical procedures. They need to be given to infants in the same dose as to adults but may act longer. Because of their dual mechanisms of metabolism, atracurium and cis-atracurium are preferred in neonates and in children with hepatic or renal dysfunction. Elimination of vecuronium may be prolonged in neonates. Atracurium and vecuronium have minimal cardiovascular effects, while rocuronium causes tachycardia. Atracurium causes histamine release and has been replaced by its metabolite cis-atracurium in the USA. Rocuronium can provide rapid muscle relaxation similar to succinylcholine with an intravenous dose of 1.2 mg/kg. It can also be administered intramuscularly.

Temperature Maintenance
Neonates and infants are at a high risk of hypothermia due to their larger surface-to-weight ratio and thin skin. Hypothermia increases oxygen consumption, causes acidosis and affects coagulation. Methods to prevent hypothermia include maintenance of ambient temperature at 27°C for neonates, keeping the child covered, using hot air mattress and/or blanket, humidification of inspired gases, and warming of fluids used for surgical preparation, cavity irrigation and intravenous infusion. Neonates should be transported in a temperature-controlled incubator. Temperature should be monitored throughout the surgical procedure. Overheating should be avoided.

EMERGENCE FROM ANESTHESIA
Good emergence from anesthesia consists of rapid and smooth recovery of consciousness with adequate spontaneous respiration, a patent airway, good airway reflexes, adequate analgesia and minimal adverse events.
Recovery: Delivery of anesthetic agents is stopped at the end of the surgical procedure. Desflurane results in the fastest recovery followed by sevoflurane, with halothane being the slowest. Propofol also results in rapid and clear-headed recovery. The clinical criteria for adequate emergence from anesthesia in preverbal children are grimacing, spontaneous eye opening and purposeful movement of the upper limbs.

Residual neuromuscular blockade is antagonized by neostigmine (50 µg/kg), and atropine (20 mg/kg) or glycopyrrolate (10 µg/kg). Sugammadex (4 µg/kg) can bind to steroidal NMBD such as rocuronium and vecuronium and reverse even profound neuromuscular blockade with no cardiovascular side effects. However, it is not used routinely due to its cost.

Removal of airway device: SGD or tracheal tube should be removed after adequate respiration is established, either in a completely awake or deeply anesthetized child depending on the anesthesiologist’s skill and preference. Airway complications such as laryngospasm, airway obstruction, coughing, oxygen desaturation, excessive salivation, biting of the device, breath-holding, and vomiting are more frequent if the device is removed in a light anesthetic plane. If the device is removed in an awake state, the child is able to maintain the airway and the risk of aspiration is low. However, the child may cough, strain and struggle before removal. Deep removal on the other hand is smooth, but the airway needs to be kept patent by the anesthesiologist for some time. In case of difficult airway, full stomach, and in small infants and neonates, it is safer to remove the device in the awake state.

A positive pressure of 15–20 cmH₂O can be applied with the reservoir bag and held while the tracheal tube is gently removed. It increases the child’s oxygen reserve. As the child coughs on extubation it also removes any secretions. It may also reduce the risk and severity of laryngospasm by the anesthesiologist for some time. In case of difficult airway, full stomach, and in small infants and neonates, it is safer to remove the device in the awake state.

During transfer to the PACU the child should be accompanied by an anesthesiologist who should brief the PACU team regarding the child’s anesthetic course. If the child is drowsy, she should be transferred in the recovery position.

INTRAOPERATIVE COMPLICATIONS

Arterial Oxygen Desaturation

Oxygen saturation <94% in a healthy child requires immediate attention. Common causes are airway device displacement, secretions/mucus plug in the airway, laryngospasm, bronchospasm, airway obstruction, and atelectasis besides equipment malfunction. Hundred percent oxygen should be administered immediately, and the cause identified and treated. If low saturation persists, a PEEP of 5–10 cmH₂O can be added to open atelectatic alveoli. Diffusion hypoxia can also occur if oxygen is discontinued early in children who had been breathing nitrous oxide intraoperatively. It is prevented by continuing oxygen administration for 5–10 minutes.

Laryngospasm

Laryngospasm is a potentially life-threatening reflex closure of vocal cords, seen more often in children than in adults. Some risk factors are younger age, airway stimulation during a light plane of anesthesia, hyper-reactive airway, current or recent upper respiratory tract infection, exposure to second-hand smoke, use of LMA, and inexperience of the anesthesiologist. It presents as stridor in an intubated child, usually during induction of or emergence from anesthesia. Diagnosis is made by quickly ruling out other causes of airway obstruction. Treatment consists of administering 100% oxygen with 5–10 cmH₂O of continuous positive airway pressure (CPAP) using a tight-fitting face mask maintaining head tilt and jaw thrust. Gentle positive pressure breaths may also be given. One must be careful as these manoeuvres carry the risk of inflation of stomach. If unsuccessful, either the depth of anesthesia is increased with a bolus of propofol, or succinylcholine is used to produce a short period of muscle relaxation. The diagnosis and treatment should be prompt as fatal desaturation can occur rapidly.

Bronchospasm

Perioperative bronchospasm can occur in children with asthma, respiratory infection, prior exposure to second hand smoke, airway foreign body, and gastroesophageal reflux disease. Bronchospasm after intubation may be due to endobronchial placement of the TT, light plane of anesthesia or secretions in the airway. Pneumothorax may be another cause of bronchospasm. If bronchospasm persists after correcting these problems, salbutamol is delivered in the tracheal tube using a metered dose inhaler. Halothane and sevoflurane are bronchodilatory and are preferred over isoflurane and desflurane to deepen the plane of anesthesia. If there is no response to these measures, 1–2 µg/kg of adrenaline can be given intravenously.

Airway Obstruction

During anesthesia there is a loss of pharyngeal and laryngeal muscle tone leading to obstruction of the upper
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Airway. Airway obstruction can be detected by a noisy inspiration, retraction of the chest and distension of the abdomen during the patient's inspiratory effort (see-saw movement), suprasternal and intercostal retraction, decreased movement of the reservoir bag and absent or low ETCO2 waveform. Airway obstruction can also occur at the end of anesthesia if the child is drowsy. Obstruction can be relieved in most instances by head tilt and chin lift or jaw thrust maneuvers and application of 5–10 cmH2O of CPAP. If this is not successful, an oro- or naso-pharyngeal airway can be used to relieve the obstruction.

Bradycardia

The definition of bradycardia is age-specific: <100 bpm in infants, <80 in small children and <60 in children older than 5 years. Some common causes are hypoxia, vagal reflex, drugs (halothane, succinylcholine), heart disease, raised intracranial pressure and dyselectrolytemia. As cardiac output is heart-rate-dependent in infants and small children, the cause of bradycardia should be corrected and bradycardia treated immediately with 20 µg/kg of atropine, intravenously. In case bradycardia proceeds to asystole, CPR is initiated and adrenaline administered.

Emergence Agitation

Behavioral disturbances such as crying, disorientation, excitation, agitation and delirium seen following anesthesia are termed emergence agitation (EA). Though the exact cause of EA is not known, it is seen most commonly following sevoflurane and desflurane anesthesia. Other predisposing factors are rapid awakening from anesthesia, pre-school age, pain and preoperative anxiety. EA is self-limiting and short-lived, but can result in self-injury pre-school age, pain and preoperative anxiety. EA is termed emergence agitation (EA). Though the exact cause of EA is not known, it is seen most commonly following sevoflurane and desflurane anesthesia. Other predisposing factors are rapid awakening from anesthesia, pre-school age, pain and preoperative anxiety. EA is self-limiting and short-lived, but can result in self-injury and displacement of indwelling cannulas, catheters and dressings. It distresses the parents and other caregivers, and may delay discharge from the PACU. Propofol, fentanyl, ketamine, midazolam and α2 adrenoreceptor agonists have been used prophylactically at the end of anesthesia.

Treatment usually consists of benzodiazepines.

LEARNING POINTS

- Separation from the parents and induction of anesthesia are distressing to children. The anesthesiologist should ensure that this process is pleasant and smooth by psychologically preparing the child and her parents, using premedication when necessary and individualizing the induction of anesthesia to suit each child.
- Most children prefer inhalational technique for induction of anesthesia. Sevoflurane, with or without nitrous oxide, is preferred because of its sweet smell, nonirritancy, rapidity of onset and minimal cardiovascular depression.

†Contd…

- Drug dosages should be modified taking into consideration the variation of their pharmacokinetics and dynamics with age
- Appropriate pediatric equipment should be available. A large number of surgical procedures can be performed using supraglottic devices
- Airway complications such as arterial oxygen desaturation, laryngospasm and airway obstruction are common in children. Prevention, rapid detection and immediate treatment are essential to avoid a fatal outcome

Acknowledgment

I thank Dr LN Yaddanapudi, Professor, Department of Anesthesia and Intensive Care, PGIMER, Chandigarh for his critical review of the manuscript which made it more comprehensive, accurate and easily comprehensible.

References

INTRODUCTION
Wide array of monitoring devices have proved to be the backbone of safety in pediatric anesthesia. However, one should not develop false sense of security as no monitoring device is perfect. Children’s physiologic status is dynamic. Along with child’s condition due to disease and surgical procedure and the effect of anesthetic drugs, there can be rapid changes in physiological parameters. Hence, monitoring plan should be tailored to individual patient. Monitoring devices are used during anesthesia to detect deviation from normal physiological parameters and unexpected life threatening events and to help fine tuning of anesthetic management. Although with ever changing technology the accuracy and sensitivity of monitoring devices showing upward trend, there is no substitute for continuous vigilant clinical observation by anesthesiologist. Anesthesiologist needs to interpret the numbers and curves displayed on the screens according to child’s physical status and concurrent events.

Continuous observation of the child and surgical field are of immense importance as there are physical signs which remain outside the scope of monitoring equipment as monitors do have lag time before they show the changes.

PHYSICAL SIGNS
- Status of the eyes—lacrimation, divergence of gaze, size of pupils indicate light plane of anesthesia
- Color, temperature and dryness of skin
- Signs of airway obstruction—paradoxical chest and abdominal movement, stridor do not get detected immediately by monitors
- Palpation of fontanel in the infant indicates state of hydration and intracranial pressure
- Capillary refill time indicates state of peripheral circulation
- Pulsatility of mesenteric vessels provides assessment of splanchnic perfusion
- Quality of peripheral pulses may vary in children despite stable BP measurement
- Color and volume of urine indicate hemodynamic status.

So in all cases "Look, Listen and Feel" these three vital perceptions along with display of physiological parameters on monitors offer valuable information and a warning in impending accidental events indicating prompt action. Our goal comprises of integration and application of the data to achieve safety and prevent morbidity in the management of anesthesia in children.

The special characteristics of infants and young children which include bigger surface area compared to weight, narrow airways, immaturity of respiratory center, decreased respiratory reserve, higher cardiac output and metabolic rate and immaturity of hepatic and renal function make these children vulnerable to hypothermia, hypoxia, bradycardia, hypoglycemia emphasizing the importance of monitoring.

Proper recording of patient’s status right from the time of arrival in the operating room and continuing through intra-operative period until the child is shifted
to recovery becomes a medicolegal document and allows trend analysis of vital parameters. Guidelines for intraoperative monitoring of patient under anesthesia are published by ASA (2003). Basic mandatory monitoring for every child undergoing anesthesia includes stethoscope, pulse oximetry, electrocardiography, end-tidal carbon dioxide (ETCO₂), noninvasive blood pressure (NIBP) and temperature. Additional equipment can be chosen for specific procedures as neurosurgery or cardiovascular and transplant surgery and critically-ill children.

**PRECORDIAL/ESOPHAGEAL STETHOSCOPE**

Stethoscope can help experienced anesthesiologist to detect arrhythmias and also changes in blood pressure and cardiac output guided by intensity of the tone of heart beat. It is also extremely useful in babies during induction of anesthesia when awake baby may not allow application of basic monitors. Changes in heart sounds as well as ventilation can be detected instantaneously. Stethoscope offers cheap, safe and continuous monitoring. Precordial stethoscope (plastic for MRI) can be stabilized with double adhesive tape for distant monitoring during bronchography, CT or MRI imaging. Both heart sounds and breath sounds can be optimally heard at the apex of the heart (between 2nd and 4th intercostal space at left sternal border). Stethoscope placed at the suprasternal notch during induction and emergence helps to detect airway obstruction or laryngospasm much early guiding anesthesiologist to take corrective action with PEEP. A venous air embolus can result in a new mill wheel murmur which can be detected by auscultation.

Esophageal stethoscope should be used only if trachea has been intubated. It can be introduced gently avoiding trauma in neonates and infants while listening to heart and breath sounds to ascertain the optimal position. Often the optimal position is midesophagus. It is useful in cases of burns, chest trauma and thoracic surgery.

Disadvantages of esophageal stethoscope are:
- Occasionally traumatic
- Airway obstruction
- Compression of aortopulmonary collateral vessels in infants with complex cyanotic heart disease
- Misidentification of an esophageal stethoscope as an endotracheal tube during tracheostomy.

**ELECTROCARDIOGRAPHY**

Electrocardiography offers help in accurate measurement of heart rate and diagnosis and treatment of arrhythmias. In neonates and infants normal ECG may be present in spite of significant fall in cardiac output (during use of halothane). So ECG may give false sense of security in young children. Heart rate decreases and PR and QT intervals and duration of QRS increase with increasing age.

**Electrocardiography Features in Children**
- Right ventricle predominates
- T wave is larger in infants causing erroneous double counting of HR
- Rightward axis is normal in infants and children as electrodes are closer to heart
- Arrhythmias are rare but if they occur, it is a sign of danger
- Dangerous levels of hypoxia or hypercapnea can occur without accompanying arrhythmias.

**Danger Signals on ECG**
- Increase in T wave amplitude of more than 25% suggesting intravascular injection of local anesthetic drug with epinephrine
- Prominent T waves seen in hyperkalemia, succinylcholine injection, use of halothane in patients with muscular dystrophy and after massive blood transfusion
- Prolonged QT interval in hypocalcemia during rapid transfusion of citrated blood and blood products
- Prolonged QT with T wave flattening in hypokalemia
- ST segment changes in cardiac ischemia—rare.

For routine ECG monitoring in children, lead 2 is recommended as it provides good view of atrial activity for diagnosis of arrhythmias. Many children have noticeable variation in heart rate with respiratory cycle (sinus arrhythmia). The risk of ECG monitoring includes burns, electrical shock injury, and inappropriate therapy due to misdiagnosis of arrhythmias.

**PULSE OXIMETRY (SpO₂)**

Pulse oximetry is mandatory noninvasive monitor which provides beat to beat oxygen saturation of patient’s hemoglobin. Oxygen saturation is defined as the oxygen content expressed as a percentage of the oxygen capacity. During systole of cardiac cycle there is greater volume of blood in the pulsatile arterial vascular bed. This vascular bed is positioned between a sensor and light emitting diode. Oxygenated and deoxygenated blood absorbs different quantities of light depending upon percent saturation. Arterial saturation is determined by using the ratio of light absorption at two different wavelengths.
(660 and 940 nm). At each wavelength both non-pulsatile and pulsatile absorbance are measured and this allows the calculation of ratio and it is empirically corrected to oxygen saturation. Within 80% to 100% saturation range arterial saturation values determined by this method correlated well with in vitro measurements. There is poor correlation between the observation of cyanosis and true desaturation.

There are different sites where pulse oximeter probes can be placed. Peripheral sites are—finger, palm, toe, sole. Central sites are—ear, tongue, wrist. Proper size of probe for each patient is very important. Lingual probe has the advantage as it is not affected by use of cautery or intense vasoconstriction. However, child must be intubated and paralyzed for use of tongue oximetry. Lingual probes need to be disposable to avoid cross-infection. The sole has better perfusion compared to finger and toe, hence it is often used in NICU setting for better performance. The author has experienced, the flexi probe strapped around the wrist remains quite stable and offers accurate reading in neonates and small infants. All pulse oximeters become inaccurate when saturation falls below 80%. Measurement of preductal (right hand) and postductal (any of the feet—sole or toe) oxygen saturation facilitates the diagnosis of reversal of shunt across atrial or ventricular septal defect (Fig. 1). In peripheral vascular disease and infusion of vasopressor drugs SpO₂ readings are unreliable.

**Physiological Limitations**

- Pulse oximetry cannot display hyperoxia as there is a plateau above PaO₂ of 80 mm Hg seen with oxygen dissociation curve
- In children with β thalassimia and infants with fetal hemoglobin, O₂ dissociation curve is shifted to left resulting in lower PaO₂ for similar SpO₂
- In cases of sickle cell anemia and acidosis. O₂ dissociation curve is shifted to right
- In neonates with increased levels of carboxyhemoglobin due to hemolysis and also in burns and carbon monoxide poisoning SpO₂ is overestimated
- IV dyes like methylene blue, indocyanine green and indigo carmine affect the light absorption displaying low SpO₂
- Nail polish and meconium staining produce low SpO₂ reading and presence of methemoglobin hinders accuracy.
- SpO₂ overestimates SaO₂ in polycythemia and underestimates SaO₂ in anemia
- In hypovolemia with reduced perfusion there is loss of signal and in hypothermia reading may be normal if core temperature is above 30°C
- In obstructed venous return, elevated intrathoracic pressure and presence of venous pulsations in tricuspid regurgitation falsely low SpO₂ is displayed.

![Fig. 1: Various types of SpO₂ probes](image-url)
In case of hypoventilation, PaCO$_2$ (arterial) and PACO$_2$ (alveolar) both will rise and according to alveolar gas equation PAO$_2$ and PaO$_2$ will fall leading to desaturation. If FiO$_2$ is increased PAO$_2$ and PaO$_2$ will rise whatever is the reading of PACO$_2$. Thus, hypoventilation will not be detected. This has significance in postoperative recovery period as every patient is given O$_2$ by mask or nasal prong. Pulse oximeter that changes the tone with changes in SpO$_2$ provides immediate aural warning to the personnel in the vicinity. Pulse oximeter with clip on type of probe is prone to easy displacement and produce artifactual data.5

**Technical Limitations**

- Inaccuracy increases when SpO$_2$ is <80% as in cyanotic heart disease
- There is a delay in response by 30–60 seconds when probe is placed peripherally (finger, toe) as compared to centrally (ear-cheek-tongue) placed
- Pulse oximeter shows motion artifact as inaccurate or no reading
- Ambient light, other light sources, incorrect position of probe affect the accuracy
- Electrical and magnetic interference affect the reading, so especially designed monitors for MRI suite are required.

**Points to Remember**

- Probes of one brand should not be mixed with different brand
- Check the probe and its wiring
- Verify synchrony of SpO$_2$ waveform and heart rate with ECG display on monitor
- Size and type of probe should be selected according to age and size of the patient
- In case of doubt, check the patient’s condition before blaming the equipment
- Check the probe site frequently and keep under direct visual control.

**Possible Complication**

As infants and young children have delicate skin, they may get burns or pressure necrosis if probe is left at one site for a long time. To avoid this, probe site can be changed at regular intervals.

**PLETH VARIABILITY INDEX (PVI)**

From the plethysmographic display of SpO$_2$ valuable information can be obtained regarding hemodynamic status. Respiratory variations in the plethysmographic waveform amplitude have been correlated with respiratory variations in arterial pulse pressure and can predict fluid responsiveness in mechanically ventilated patients under general anesthesia. Cannesson and colleagues found that baseline pulse oximeter plethysmograph amplitude was correlated with percent change in cardiac index induced by volume expansion.8 Perfusion index (PI) is the numerical value of the plethysmograph waveform amplitude. It is the ratio of pulsatile to nonpulsatile blood flow through the peripheral capillary bed. PVI is an automatic measure of the dynamic change in PI that occurs during the respiratory cycle. It can be calculated automatically by using the formula: PVI=100 × (PI$_{\text{max}}$ - PI$_{\text{min}}$)/PI$_{\text{max}}$. Shelley et al. demonstrated that the ear and forehead may be better monitoring sites for detection of variation in respiratory waveform.9

Bringing PVI into clinical field would necessitate devices allowing for continuous automated real-time calculations. Such devices will facilitate and guide fluid optimization during surgery in infants.

**SIGNAL EXTRACTION TECHNOLOGY (SET)**

Innovative pulse oximetry technologies have claimed better performance during poor perfusion and movements of extremity. Studies comparing conventional pulse oximetry with SET during pediatric anesthesia demonstrated superior performance with SET.10

**CUTANEOUS OXYGEN TENSION PsO$_2$**

A miniature Clark polarographic oxygen electrode can be applied to skin to measure PsO$_2$. It is similar to the one used in blood gas analysis. The probe heats the skin to 42–44°C so PsO$_2$ approaches arterial oxygen tension as a result of increase in skin blood flow and permeability.11 The correlation between arterial and cutaneous oxygen tension is better in neonates as they have thin less keratinized skin with dense cutaneous capillary bed.

**Disadvantages of PsO$_2$ Monitor**

- It requires frequent calibration
- There is warm up time of 10–20 minutes
- Skin needs meticulous preparation and probe placement
- It is sensitive to electrosurgical interference and mechanical manipulation
- Because of probe heating burns can develop at the site.

In NICU, PsO$_2$ monitor is useful in premature neonates to detect hyperoxia.
NONINVASIVE BLOOD PRESSURE MONITORING (NIBP)

Blood pressure measurement is a fundamental aspect of monitoring for the hemodynamic status of the patient in the perioperative period. Blood pressure cuff is commonly placed on the upper arm but it can be placed on the thigh, calf, forearm when upper arm is not accessible. The size of the cuff is of paramount importance to get accurate reading (Fig. 2). As a rule width of the inflatable cuff should cover approximately two-third of the distance between child’s axilla and antecubital fossa or other extremity to which it is applied. Length of the inflatable cuff should cover 90–100% of the arm circumference. The inflatable cuff that is too small or too narrow occludes the artery incompletely, resulting in early return of detectable flow, and hence overestimating BP measurement. The error can be as high as 30 mm Hg. Inflatable cuff that is too wide or has residual air in the cuff may dampen the arterial wave, and thus showing spuriously low BP. BP measured from the cuff placed on the calf is usually lower than that measured from arm especially in children up to 4 years old. Most brands of NIBP monitors display mean arterial blood pressure (MAP). MAP in neonates and premature babies correlates with their gestational age in weeks. MAP = 1/3 SBP + 2/3 DBP and Pulse pressure = SBP – DBP; (SBP—systolic and DBP—diastolic BP).

LEARNING POINTS
• Use BP cuff of proper size
• Squeeze out all the air from the cuff
• BP cuff should be wrapped snugly around limb
• Check for the leak in the tubing and connections
• Avoid extrinsic compression of the cuff
• BP cuff and heart should be at the same horizontal level
• In case of malfunction or bizarre reading, check the child’s condition
• Check for motion artifact

INVASIVE OR DIRECT BLOOD PRESSURE MEASUREMENT

When there is a need for precise beat-to-beat BP monitoring or for frequent checking of arterial blood gas values, arterial catheter placement offers great assistance. Although arterial cannulation is commonly performed in the radial artery alternate sites include ulnar, brachial, femoral, dorsalis pedis, posterior tibial artery and umbilical artery (in newborns).

Indications (IBP)
• Presence of unstable hemodynamics in critically ill children
• Surgical procedure resulting in profound hemodynamic alterations related to blood loss (acute loss >10% of EBV and total loss >50% of EBV) or fluid shifts
• Deliberate hypotensive anesthesia
• Cardiopulmonary bypass, organ transplant surgery, neurosurgery
• Significant abnormalities in gas exchange due to pre-existing disease or major thoracic procedure.

Arterial catheter placement is done after considering risk-benefit analysis for a particular child.

CENTRAL VENOUS PRESSURE MONITORING (CVP)

CVP is the result of complex interaction between intravascular volume status, ventricular compliance and intrathoracic pressure. To measure CVP right internal jugular vein is common site for catheter placement, but other sites such as left internal jugular vein (IJV), subclavian vein on either side or femoral vein on either side, or external jugular vein can be used when right IJV is not accessible.

Indications for CV Cannulation
• Inadequate peripheral venous access
• CVP monitoring
• Infusion of hyperosmolar or sclerozing substances, drugs or fluids
• High risk of venous air embolism during certain surgical procedures
• Large estimated blood loss (>50% EBV) or fluid shifts
• Deliberate hypotensive anesthesia
• Cardiopulmonary bypass
• Critically-ill patients with renal failure or congestive heart failure.

Normal values of CVP in children range between mean 2–6 mm Hg. CV cannulation requires proper technique and maintenance or else it can result in complications such as infection, thrombosis, embolism, catheter malfunction, occlusion, dislodgement, breakage, perforation, inadvertent puncture of carotid artery or thoracic duct and pneumothorax. Alderson and others reported 18% prevalence of anatomic variations in children younger than 6 years that would significantly hinder successful cannulation of IJV using anatomical landmarks alone. USG-guided cannulation facilitates to achieve greater success.

NONINVASIVE RESPIRATORY GAS MONITORING

Endtidal Carbon Dioxide (ETCO₂)

Capnometry is the instantaneous measurement of carbon dioxide partial pressure in inspired and expired air during each breath. Capnography is the graphic display and analysis of it helps to evaluate quality and adequacy of ventilation.

Uses of Capnography

• Confirmation of placement of endotracheal (Et) tube or laryngeal mask (LMA) in the correct position
• Gives information on respiratory rate, breathing pattern, Et tube patency
• Analysis of degree of neuromuscular blockade
• Gives warning of faulty anesthesia delivery system, ventilator equipment, disconnection, malfunction of valve, exhausted CO₂ absorber
• Helps diagnosis of metabolic (malignant hyperthermia), respiratory (bronchospasm), and hemodynamic (embolism or arrest) events
• Helps to calculate physiological parameter—dead space.

ETCO₂ measurement can be done by different types of analyzers—infrared analysis, mass spectrometry, acoustic spectrometry or Raman scattering. Expired gases can be sampled at different sites in the breathing circuit. If expired gas passes through a sensor placed in the breathing circuit, it is mainstream capnography. If expired gas is aspirated from the airway by a fine tubing and transported to distant analyzing chamber, it is sidestream capnography. If ETCO₂ is measured in the Et tube or LMA distal to the connection with breathing circuit, it is called distal ETCO₂. If it is measured at the connection of breathing circuit, it is called proximal ETCO₂.

Features of Mainstream Capnography

• There is no response delay
• It shows accuracy at rapid ventilation rates
• It measures only ETCO₂ and no other respiratory gases
• It adds to the dead space—disadvantage in infants
• It cannot be used in nonintubated patients and patients in recovery
• Delicate sensor is nearer to child’s face, so there is a risk of Et tube kinking, disconnection (due to weight) and pressure injury or burns
• Its cuvette needs cleaning between cases.

Features of Side Stream Capnography

• As it has light adapter, distal sampling at Et connector is easy
• It can be used in nonintubated patients and in recovery area with nasal sampling
• It can be used for measurement of other respiratory gases
• Choice of sampling site is critical for accuracy as in re-breathing accuracy is lost
• Transport of gases to distant analyzer causes response delay depending upon length of sampling line
• There is a risk of obstruction in the sampling line due to moisture and secretions
• Accuracy is affected by rapid respiratory rate and capnographic baseline is erroneously elevated above 40 breaths/min.

Proximal sampling of expired gases shows accuracy in ETCO₂ in children weighing more than 12 kg. Distal sampling can be done via catheter inserted through wall of Et, sampling port of elbow connector or Et with special sampling line. If bacterial filter or moisture exchanger is included in the breathing circuit, sampling should be done at the machine side of the filter to avoid ingress of water and infection.

Factors Influencing Measurement of ETCO₂ in Children

• Physical
  - Response time
  - Type of capnometer—side or mainstream
  - Site of sampling—proximal or distal
  - Weight of the child
  - Type of ventilator or circuit used
  - Presence of secretions or moisture
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- Physiological
  - Small tidal volume (TV)
  - High respiratory rate (RR)

- Pathological
  - Pulmonary disease
  - Acyanotic or cyanotic heart disease.

Response time should be less than respiratory cycle time of infant to get predictable ETCO2. Mainstream capnometer is more accurate than sidestream for proximal site ETCO2 but may cause unacceptable rebreathing. Use of rebreathing circuits produce distorted capnogram with no or flat alveolar plateau due to dilution of ET gas. Sidestream capnometer with high flow rate of 150 mL/min results in erroneous ETCO2 and distorted waveform in neonates and infants with small tidal volume and high RR. Microstream technology is superior in infants and young children.20

Interpretation of Capnogram (Figs 3A to C)

- In infants and children ventilated with circle absorber circuit, capnograph shows sharp upstroke at the beginning of expiration followed by slightly ascending plateau phase interrupted by next inspiration
- Sudden disappearance of capnograph occurs due to disconnection, obstruction, cardiac arrest or pulmonary embolism
- Slow increase in ETCO2 occurs due to exhausted CO2 absorbent, hypoventilation and fever
- Rapid increase in ETCO2 suggests malignant hyperthermia
- Increase in inspiratory CO2 (PiCO2) occurs due to rebreathing or artifact due to high RR
- Gradual increase in ETCO2 and PiCO2 suggest faulty unidirectional valve
- Sharp upstroke of the plateau suggests bronchospasm or kinking of Et tube
- Curare cleft suggests residual effect of muscle relaxant and spontaneous breathing attempts
- Overshoot in ETCO2 reflects the washout of CO2 accumulated in venous blood and tissues during circulatory arrest
- Transient increase in ETCO2 occurs during transfusion of blood or NaHCO3
- Abnormally low ETCO2 indicates increase in dead space or a state of low pulmonary perfusion
- Error due to dead space ventilation occurs in children with cyanotic congenital heart disease.21

CUTANEOUS CARBON DIOXIDE TENSION (PsCO2)

PsCO2 is not routinely measured in operating room as there is reasonably good correlation of endtidal and arterial carbon dioxide tensions in all, except extremely small neonates. PsCO2 is always higher than PaCO2 as a result of tissue CO2 production and increased metabolism caused by a heating sensor. The predictable gradient from arterial to cutaneous CO2 tensions enables the monitors to calculate the gradient and display the corrected value. Using a variant of the Severinhaus electrode, measurement of PsCO2 tension is available.22

MEASUREMENT OF OTHER RESPIRATORY GASES

Multigas analyzer offers valuable information about cardiopulmonary physiology. Monitors offering multigas analysis are based on techniques such as mass spectrometry, ultraviolet and infrared light absorption or absorption into lipophilic substances.

Figs 3A and B: (A) Phases of normal capnograph; (B) Graph showing abrupt increase in ETCO2 suggesting spontaneous return of circulation during cardiopulmonary resuscitation
Advantages of Multigas Analysis

- Confirmation of elimination of nitrogen and adequacy of preoxygenation
- Detection of leak in anesthesia delivery system
- In combination with ETCO₂ detection of venous air embolism
- Demonstrates uptake and elimination of anesthetic gases
- Confirms the purity and accuracy of vaporizers
- Assessment of prolonged emergence from anesthesia from quantity of residual anesthetic gases.

Other respiratory monitors, useful in anesthesia include spirometer in ventilators which can display exhaled tidal volume and even flow-volume loop and airway pressures indicating respiratory mechanics (Fig. 4). The data is extremely useful for adjustment of settings of ventilation.

BISPECTRAL INDEX MONITOR (BIS)

BIS monitor is an electroencephalogram (EEG) based device that is used to predict level of unconsciousness or hypnosis in anesthetized patients (Figs 5A and B). The patch is affixed to patient’s forehead and connected to monitor which integrates various EEG descriptors into a single calibrated number ranging from 0 to 100. Hundred represents full wakefulness whereas 0 represents electrical silence. BIS value less than 60 in unconscious state ensures a lack of intraoperative recall. BIS values in anesthetized children are inversely proportional to the endtidal concentration of sevoflurane. However, this association is not precise in infants. During scoliosis surgery BIS can predict voluntary patient movement in response to commands when wake up test is carried out. Because of
age-related differences in brain maturation and synapse formation throughout childhood, BIS monitoring is not as reliable in children as in adults. Other sophisticated processed EEG monitors relying on forehead electrodes are Entropy and Narcotrend. They also have algorithm to process EEG and create score from 0 to 100 representing extremes of coma and wakefulness correlating with depth of sedation and anesthesia. Narcotrend, Entropy and BIS are similar in monitoring conscious level in children older than 1 year.

NEAR INFRARED SPECTROSCOPY (NIRS)

This noninvasive device measures oxygenation of blood in the underlying tissues, including arterioles, capillaries and venules. It is widely used to measure nonpulsatile regional cerebral oxygen saturation (rSatHbO₂). The probe is placed on the child’s forehead to measure cerebral oxygenation which depends on factors that affect oxygen transport, cerebral blood flow, Hb saturation, and Hb-O₂ binding affinity. Tissue consumes O₂, so has less than 100% rSatHbO₂.

LEARNING POINTS

- Vasoconstriction and dilatation of cerebral vessels, capillaries, arteries and veins have large influence on rSatHbO₂
- Normal rSatHbO₂ in noncyanotic infants on air is about 70% and in cyanotic infants between 40–60%
- During CPB rSatHbO₂ is close to 100% as cerebral O₂ demand is reduced by anesthesia and hypothermia
- ↓rSatHbO₂ is seen when there is cerebral metabolism as a result of metabolic debt

Scoff and Hoffman have strongly recommended use of NIRS in pediatric anesthesia in a recent review.

PULSE OXIMETRY-BASED HEMOGLOBIN DETERMINATION

It is a new technology that uses absorbance spectrography in real-time on living tissue to determine the absolute concentration of Hb. When multiple wavelengths of light are used in combination with a lot of clinically derived constants, then it is possible to derive quantitative measurement of Hb. Two devices are currently available—the Masimo SpHb® (Fig. 6) and NBM-200MP (Oursense, Nes Ziona). These devices need to be modified for pediatric and infant use and evaluated in them.

Indications for Use

- Anticipated significant blood loss during cardiac, neuro, craniofacial and oncology surgery and organ transplantation
- When faced with difficult blood sampling, e.g. premature babies and children coming for chemotherapy frequently.

There are chances for inaccuracy in ambient light, inconsistent path length (fat, bone, cartilage, digit size),
movement, vasoconstriction with hypovolemia, cyanosis, Hb varients including fetal Hb.

TEMPERATURE MONITORING

Intraoperative temperature monitoring is essential to detect hypothermia or hyperthermia so as to avoid their consequences. It is also useful to manipulate body temperature whenever indicated, e.g. brain protection during cardiac surgery. In infants and young children thermoregulation is less efficacious during anesthesia. They can invariably become hypothermic in cold environment in OR. Hypothermia directly depresses the level of consciousness and interferes with the recovery from anesthesia due to delayed metabolism. So with the help of warming devices core temperature should be maintained above 36°C to avoid complications. The core temperature can be monitored with adequate accuracy with the probe placed in the distal esophagus, nasopharynx just behind soft palate, per rectum or tympanic membrane. Tympanic membrane temperature can be taken intermittently, so useful in recovery room. The skin temperature is influenced by many factors such as ambient temperature, anesthetic technique, and cardiac output. Large gradients in skin and core temperature are indicative of non-uniform heat distribution or poor cardiac output.

To maintain normothermia in neonates and infants during surgery ambient temperature should be kept at 26°C approximately.31

NEUROMUSCULAR BLOCK (NMB) MONITORING

A nerve stimulator offers assessment of depth of neuromuscular blockade and to determine if the reversal can be given at the end of surgery and effectiveness of reversal agents. It allows muscle relaxant drug titration to the individual patient’s response and to determine the best time to intubate. It helps to diagnose unusually prolonged duration or incomplete reversal of NMB. While evaluating new muscle relaxant, nerve stimulator helps to define its characteristics such as time of onset of action, duration and dose-response curve.

Nerve Muscle Unit Used for Assessment of NMB (Figs 7A to D)

- Ulnar nerve stimulation and assessment of thumb adduction (adductor pollicis muscle response)
- Posterior tibial nerve stimulation behind middle malleolus and assessment of plantar flexion of the big toe (flexor hallucis brevis response)
Facial nerve stimulation anterior to the ear lobe and assessment of muscular response of ipsilateral eyelids contraction.

Facial nerve stimulation may result in false positive response as muscles may be easily stimulated directly potentially causing administration of overdose of muscle relaxant.

**Modes of Stimulation and Evaluation**

- Single twitch stimulation (ST) (Fig. 8A)
- Train of four stimulation (TOF) (Fig. 8B)
- Post-tetanic count (PTC)
- Double burst stimulation (DBS) (Fig. 8C).

Select the current of low amplitude, such as 10–20 mA in the beginning and turn on the nerve stimulator. Increase current in increments of 10 mA until 4 twitches are observed. Supramaximal stimulation is the level at which 4 vigorous twitches are observed, e.g. if 4 strong twitches are observed at 50 mA but when raised to 60 mA, there is no further increase in the response, then supra-maximal stimulation is 50 mA. In TOF mode of stimulation for monitoring NMB 4 successive stimuli are delivered (less than 40 mA in children) every 5 seconds.

**Measurement of Response**

- When 4 twitches are seen—0–75% of receptors are blocked
- When 3 twitches are seen—at least 75% receptors are blocked
- When 2 Twitches are seen—80% receptors are blocked
- When 1 Twitch is seen—90% receptors are blocked
- When no twitch is seen—100% receptors are blocked.

**Post-tetanic count:** NMB can be quantified by PTC when there is profound NMB observed after ST or tetanic or TOF stimulation. Tetanic stimulation (30–50 mA) is applied for 5 seconds, then after 3 seconds ST is applied with 1 mA. The number of post-tetanic twitches observed is called PTC. PTC of 8–11 indicates imminent return of TOF stimulation following muscle relaxant. PTC is used to evaluate intensity of NMB when there is no response to ST or TOF.

**Double burst stimulation (DBS):** It consists of two short bursts of 30–50 mA stimulation separated by 0.75 seconds. Responses are perceived as 2 separate twitches. DBS is mainly used to detect residual NMB. It is more sensitive than TOF for assessment of recovery from NMB as 2 DBS elicited contractions are stronger and easier to compare than 1st and 4th response of TOF.

**Fig. 8A:** Single twitch stimulation
Neuromuscular junction continues to develop morphologically and biochemically in infants and small children. So NMB agents even in small doses, substantially impair NM function. Clinically, this reveals as a lower train of four values in neonates than in older infants and children.\textsuperscript{32} With the availability of shorter and intermediate acting relaxants and their least dependency on renal and hepatic function, postoperative residual curarization is unusual in pediatric anesthesia practice. However, nerve stimulator is of great assistance to achieve appropriate level of muscle relaxation in surgeries of long duration with muscle relaxant drug infusion.

**URINE OUTPUT**

Indwelling urinary catheter facilitates precise measurement of urine output.

**Indications**

- Large shifts of body fluids are anticipated
- Anticipated blood loss is in excess of 25% of estimated blood volume
- Long surgical procedures to prevent bladder distension
- Cardiac, neurosurgery, organ transplantation and oncosurgery.

Urine output of 1–2 mL/kg indicates adequate renal perfusion vis a vis adequate hydration. As neonatal kidneys cannot concentrate or dilute urine, urine output alone is not a good indicator of intravascular volume or cardiac output. Large amounts of urine can occur in patients with metabolic disorders, diabetes insipidus, postobstructive diuresis, nephropathy and head injury.

**BLOOD CHEMISTRY**

During surgeries of long duration and when indicated by patient’s disease, it is essential to measure blood glucose, electrolytes and blood gases so that appropriate action can be taken to optimize physiological values. In neonates, normal values of arterial O\textsubscript{2} tension and blood glucose are low compared to adults. But at the same time neonates are susceptible to become hypoglycemic. It is safer to maintain blood glucose >45 mg% for a neonate. Hyperglycemia should be avoided as blood glucose more than 125 mg% may produce osmotic diuresis. PaO\textsubscript{2} at birth is 70–75 mm Hg and higher values >80 mm Hg for prolonged periods can cause retinopathy.

**OTHER MONITORING AIDS**

Pulmonary artery pressure, cardiac output monitor, transesophageal echocardiography, gastric tonometry, neurophysiological monitoring and transcranial doppler are used during specific surgical procedures. These monitors are described in other chapters in the book.

**CONCLUSION**

With the availability of various monitoring gadgets we can now monitor our pediatric patients more effectively
and determine their clinical status in greater exactitude alerting us to take appropriate action. High risk patients’ preoperative testing can provide clues as to which child is likely to need enhanced care, support and management. Anesthesiologist’s consistent approach to outcome directed care is important for safe anesthesia practice.

REFERENCES

INTRODUCTION

In the perioperative period, fluid has to be administered at the correct rate and should be of right composition with respect to water, electrolyte, glucose, osmolarity and in vivo tonicity. This is very essential to avoid iatrogenic complications like postoperative hyponatremia. As neonatal physiology is markedly different from children and adults, they have unique requirements of fluid and electrolytes.

NEONATAL PHYSIOLOGY

Developmental Changes in Body Composition and Fluid Compartments

As gestation progresses, there is gradual reduction of total body water (TBW) and extracellular fluid (ECF) compartment. In the 16-week-old fetus, TBW is 94% of total body weight, and two thirds of the TBW is distributed in the ECF. By term, TBW is 75%, and half of this is in ECF. Whereas in adults, TBW is 60%, and one third of this is in ECF. ECF has two components—fixed component is intravascular volume; variable component is interstitial volume. Premature neonates, therefore, have expanded interstitium.

In the initial few days after birth, there is absorption of the interstitial fluid into the intravascular compartment resulting in increase in circulating blood volume. This stimulates the release of atrial natriuretic peptide (ANP), which leads to renal sodium and water excretion resulting in reduction in TBW, and hence, weight loss. Healthy term and preterm newborns lose an average of 5–10% and 15% respectively of their birth weight during the first 4–7 days of life.

Blood Volume

In premature infants it is 100 mL/kg, for full-term infants 85–90 mL/kg, for 2 year old 80 mL/kg, and for younger children and adolescents 75–80 mL/kg.

Changes in Capillary Permeability

Capillary permeability to proteins is increased during the early stages of development. It is further increased under pathologic conditions. When sick neonates are treated with frequent albumin boluses, it leaks out causing interstitial edema, further depletion of intravascular volume, and hence, impairment of tissue perfusion.

Maturation of the Skin

Full maturation of the epidermis takes more than 28 days of age. Insensible water loss (IWL) (mL/kg/day) is inversely proportional to birthweight during the first few days of life. (Weight <1000 g: 60–70, 1000–1250 g: 60–65, 1251–1500 g: 30–45, 1501–1750 g: 15–30, 1751–2000: 15–20 mL/kg/day).

Factors increasing IWL are:

- Increased body temperature: 30% increase in IWL per degree centigrade rise in temperature
- High ambient temperature: 30% increase in IWL per degree centigrade rise in temperature
- Use of radiant warmer and phototherapy: 50% increase in IWL
- Decreased ambient humidity
• Increased respiratory rate
• Increased motor activity, crying: 50–70% increase in IWL
• Surgical malformations: Gastroschisis, omphalocele, neural tube defects.

Factors decreasing IWL are:7
• Use of incubators
• Increased ambient humidity
• Humidification of inspired gases
• Use of plexiglass heat shields
• Thin transparent plastic barriers.

Renal Function
Renal blood flow (RBF), glomerular filtration rate (GFR) and maximal concentration ability (mOsm/kg) increase with age. RBF is 40±6 (mL/min/1.73 m²) in premature newborn compared to 620±92 (mL/min/1.73 m²) in adults. GFR is 14±3 (mL/min/1.73 m²) in premature newborn compared to 125±15 (mL/min/1.73 m²) in adults. Maximal concentration ability is 480 (mOsm/kg) in premature newborn compared to 1400 (mOsm/kg) in adults. Fractional excretion of Na is 2–6% in premature newborn compared to <1% in adults. Because of immaturity of renal function, preterm infants have excessive sodium and bicarbonate losses.8 During the first few weeks of life, hemodynamically stable, but extremely immature infants produce dilute urine and may develop polyuria because of their renal tubular immaturity. As tubular functions mature, their concentrating capacity gradually improves from the 2nd to 4th week of life.1

Bicarbonate loss due to incomplete tubular reabsorption leads to decreased serum bicarbonate concentrations (12–16 mEq/L for 26–28 weeks gestation; 18–20 mEq/L for 30–35 weeks gestation; 20–22 mEq/L for term infant; 25–28 mEq/L for adult). Mild hyperkalemia coexists with metabolic acidosis.5 Because of high concentration of arginine vasopressin (AVP), newborns have low urine output during the first 24–48 hours of life. Hypoxia, lung injury (bronchopulmonary dysplasia) and central nervous system injury (intraventricular hemorrhage) increase AVP secretion in both term and preterm infants, which cause free water reabsorption and hyponatremia.5 Preterm infant is unable to excrete sodium or volume load, and therefore, is susceptible to extracellular volume overload with edema formation.

Maturation of End-organ Responsiveness to Hormones Involved in the Regulation of Fluid and Electrolyte Balance
Hormones like rennin-angiotensin-aldosterone system, vasopressin, ANP and brain (B-type) natriuretic peptide, directly regulate the volume or composition of the extracellular compartment. There is decreased responsiveness of the immature kidney to the sodium and water retaining effect of hormones.9

Heart
 Differences in myocardial ultrastructure (e.g. receptors, channels, transporters, pumps, contractile proteins) and the immaturity of various intracellular structures (e.g. myofibrils, sarcoplasmic reticulum, microtubules) lead to impaired contractility. In preterm babies, maximal contractility is more dependent on extracellular calcium than in adults. There is less increase in cardiac output with volume loading compared to older ages. Since, the resting heart rate of the newborn is high, increasing the heart rate above normal has less effect on cardiac output. Decreasing heart rate drastically reduces cardiac output.5

Central Nervous System
In preterm infants, cerebral blood flow is autoregulated over narrower range of arterial blood pressures and is easily disrupted by hypoxia, acidosis, seizures, and by the low diastolic blood pressures of patients with a patent ductus arteriosus.10 Overzealous fluid administration can increase blood pressure leading to rupture of the fragile immature brain vessels. At the same time, hypovolemia by causing hypotension can result in cerebral ischemia.

Glucose Metabolism
Glycogen storage and glycogenolysis are developed in the last trimester of pregnancy. Therefore, preterm newborns often develop hypoglycemia, especially in the first 24–48 hours of life.5

Metabolism
Infants have significantly higher metabolic rates, and therefore, higher oxygen consumption than older children.

Requirements of Neonates

Water
As number of days of age increases, water requirement increases to compensate for urinary losses (Table 1).11–12

Glucose
Term and preterm neonates require about 3–5 mg/kg/min and 5–6 mg/kg/min of glucose, respectively.12
Sodium
Sodium is not required in intravenous maintenance fluid of term and late preterm neonates during the first 24 hours of life. In the first 24 hours, 10% dextrose is administered. On day 2 of life, sodium is added to the intravenous fluids. In the extremely low birth weight (ELBW) infant, sodium-containing fluid is often required early, i.e. at 12–24 hours of life. Subsequently, in full-term infants and older children, 2–3 mmol/kg per day is needed. Premature neonates require 3 to 5 mmol/kg per day as they have high fractional excretion of Na+.

Potassium
Higher serum potassium concentrations are noted in the early postnatal period in neonates, especially in preterm babies. Potassium supplementation is usually started by the third postnatal day once urine output is established. It is started at 1–2 mEq/kg/day, and then, increased over 1–2 days to the normal maintenance requirement of 2–3 mEq/kg/day. Due to high aldosterone concentrations, prostaglandin excretion and very high urine flow rates, preterm neonates may need more potassium after the completion of their postnatal volume contraction.

Calcium
Intravenous 40 mg/kg/day of elemental calcium (4 mL/kg/day of 10% calcium gluconate) is usually required in preterm (≤32 weeks), asphyxiated babies or newborns of diabetic mothers until they establish adequate enteral nutrition. Ideally, calcium is infused into a central venous catheter.

Bicarbonate
Premature infants have a lower renal threshold for bicarbonate and limited tubular excretion of weak organic acids. Sodium bicarbonate at 1–2 mmol/kg per day is generally recommended for the very small premature infant.

Precautions
Fluid should be administered with burette sets or infusion pumps. Fluid used for flushing catheters or to administer medications must be taken into consideration.

In the neonatal ICU, main focus of fluid management is to maintain electrolyte balance, provide adequate nutrition and limit fluid overload. Overhydration can increase chances of pulmonary edema, prolonged ductal patency, necrotizing enterocolitis and chronic lung disease. In operation room, higher amount of fluid is administered as main considerations are maintenance of hemodynamics despite anesthetic-induced vasodilatation and raised venous capacitance, replacement of potentially massive evaporative losses and restoration of circulating blood volume after third space accumulation plus ongoing blood loss. However, excessive administration of fluid should be avoided.

Preoperative Evaluation of the Neonate should include:
- Gestational age, birthweight, growth appropriate for gestational age
- Trend in daily weight
- Trend in intravenous and oral intake rate and composition
- Urine output and specific gravity
- Trend in other output (gastrointestinal, cerebrospinal fluid, etc.)
- Hemodynamic status—trends in heart rate, pulse volume, blood pressure, capillary refill
- Presence of patent ductus arteriosus or other cardiovascular dysfunction (e.g. tricuspid regurgitation after asphyxia)
- Central nervous system insult (presence of intraventricular hemorrhage)
- Blood gases, electrolytes
- Current hemoglobin concentration and recent trend correlated with hemodynamic function
- Coagulation profile and requirements for blood components
- Adequacy of intravenous access.

INTRAOPERATIVE FLUID MANAGEMENT IN NEONATES
During short surgeries involving mild-tissue trauma, e.g. herniotomy, lactated Ringer’s (LR) solution with 5% dextrose at maintenance rate will be enough. If the surgery is major, involving moderate to severe tissue

Table 1: Water requirements of newborns

<table>
<thead>
<tr>
<th>Birth weight (g)</th>
<th>Water requirements (mL/kg/24 h) by Age</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 day</td>
</tr>
<tr>
<td>1501–2000</td>
<td>60</td>
</tr>
<tr>
<td>1251–1500</td>
<td>70</td>
</tr>
<tr>
<td>1001–1250</td>
<td>75</td>
</tr>
<tr>
<td>751–1000</td>
<td>85</td>
</tr>
</tbody>
</table>
trauma or extensive blood loss, then whatever dextrose and electrolyte containing fluid neonate is receiving for daily maintenance in neonatal intensive care unit (NICU) can be continued at maintenance rate as discussed earlier. Replacement solution has to be glucose free isotonic solution like LR at a rate of 6-50 mL/kg/h. For example, during a laparotomy to treat necrotizing enterocolitis, 10–50 mL/kg/h (or more) of nonglucose containing crystalloid may be required to compensate for insensible losses from inflamed, exposed bowel and peritoneum. Intraoperative free water intake of more than 6.5 mL/kg was associated with reductions in postoperative plasma sodium measurements by 4 mMol/L in newborns. At some point after delivering large volumes of crystalloid (30–40 mL/kg/h) for several hours, colloid infusion may be more appropriate.3

THREE COMPONENTS OF INTRAVENOUS FLUID THERAPY

The three components of intravenous fluid therapy include: (1) maintenance water and electrolytes to cover insensible and urinary losses; (2) replacement of preexisting deficits, either because of nil per os (NPO) orders or due to excessive losses; (3) replacement of ongoing fluid losses because of exposure, blood loss and third space loss.

Maintenance Fluid Requirement in Children

Maintenance requirements are the amount of water and electrolytes lost during normal basal metabolism. Metabolism creates two by-products, heat and solute, which is dissipated through IWL and via urine, respectively. Volume of maintenance requirements (mL of H2O/100 calories) for insensible losses is 45 mL (skin 30 mL, lungs 15 mL), renal losses 50 mL, sweat losses 10 mL, stool losses 5 mL. 10 mL of H2O is produced because of oxidation of food.12,15 Thus, 100 mL of water is required for each 100 calories of expended energy. Based on this, Holliday-Segar presented a practical method in 1957. It takes into consideration the fact that smaller the child, higher is the metabolic demand and body surface area to weight ratio. A weight-based, hourly IV fluid rate, extrapolated from the above formulas, led to what is most frequently used today in pediatric practice, the “4-2-1 rule” (Table 2). Considering the electrolyte composition of human milk and cow’s milk, they recommended 2 mEq/100 kcal/day of both potassium and chloride and 3 mEq/100 kcal/day of sodium. These electrolyte requirements are theoretically met by the hypotonic maintenance fluid like 5% dextrose with 0.2% normal saline. This amount does not include fluid deficits, third-space losses, modifications because of hypothermia or hyperthermia, or requirements caused by unusual metabolic demands. Hypotonic fluid should be avoided for replacement of third space losses and other deficits. Now, most anesthesiologists have adopted the use of either normal saline or LR for both maintenance and deficit fluid replacement in the operating room setting. There has been little controversy regarding the acceptable maintenance fluid rate as described in Table 2.

Identification and Correction of Fluid Deficit in Children

Fluid Deficit because of Nil Per Os Orders

During preoperative NPO period, children are presumed to develop preoperative fluid deficits secondary to continuing insensible losses and urine output. Furman et al. suggested multiplication of the hourly rate, as per Holliday and Segar method, by the number of NPO hours. Half of this volume is to be replaced in first hour of surgery and other half over the next 2 hours. Many centres continue this practice till date. Berry in effort to further simplify suggested that otherwise healthy children of ≤ 3 years, >3 and ≤4 years, and >4 years should receive a bolus of 25, 20 and 15 mL/kg fluid, respectively. Both these groups had prepared guidelines with assumption that patients had been NPO for at least 6–8 hours. In 1999, the American Society of Anesthesiologists (ASA) published new fasting guidelines for elective surgery. This allows administration of clear liquids up to two hour before procedures requiring anesthesia. Yet, at times, children may be starving for more than two hours. There is dearth of data determining the exact amount of fluid deficit that occurs in normal fasting children. After a 4–6 hour (or longer) fast, older infants or children without significant preexisting disease usually have no evidence of intravascular compromise. If ASA fasting guideline is followed, and/or absence of evidence for intravascular fluid deficit or hypotension in response to induction of general anesthesia, then we need not replace fasting deficit. So, assessment of the current status is more important than simple calculations.5

Table 2: Daily and hourly (4-2-1) water requirements

<table>
<thead>
<tr>
<th>Body weight</th>
<th>Daily fluid requirement</th>
<th>Hourly fluid requirement</th>
</tr>
</thead>
<tbody>
<tr>
<td>3–10 kg</td>
<td>100 mL/kg</td>
<td>4 mL/kg/h</td>
</tr>
<tr>
<td>10–20 kg</td>
<td>1000 mL + 50 mL/kg for each kg from 11 to 20 kg</td>
<td>40 mL/h + 2 mL/kg/h for each kg from 11 to 20 kg</td>
</tr>
<tr>
<td>&gt;20 kg</td>
<td>1500 mL + 20 mL/kg for each kg &gt;20 kg</td>
<td>60 mL/h + 1 mL/kg/h for each kg &gt;20 kg</td>
</tr>
</tbody>
</table>
Principles and Practice of Pediatric Anesthesia

Perioperatively, it is important to differentiate between replacement need for hypovolemia and that for "maintenance" (e.g. to compensate for NPO status). Boluses of either LR or normal saline are appropriate for correction of hypovolemia (i.e. hemodynamic instability, poor perfusion).

Replacement of Preexisting Deficit

Preexisting fluid deficit may be present because of vomiting, diarrhea, fever, or third space sequestration (e.g. intestinal obstruction). It is very common in children undergoing emergent abdominal surgery.

Rate and volume of replacement infusion for correction of preexisting deficit depends on the type and indication of surgery, hemodynamic status and coexistent medical problems. In many cases, preoperative hypovolemia can be corrected by giving boluses of normal saline or LR (10–20 mL/kg). In cases with ongoing losses (e.g. traumatic hemorrhage), fluid resuscitation is initiated before surgery and is further continued intraoperatively.

After achieving hemodynamic stability, maintenance fluids are started. In addition, sufficient crystalloid/colloids are given to replace the ongoing fluid losses and to maintain hemodynamic stability.

If surgery is not considered urgent, correction of metabolic and fluid status should be done before transfer to the operating room. The best example of this is the infant with hypertrophic pyloric stenosis. This should never be considered an urgent surgical procedure. They have a hyponatremic hypochloremic hypokalemic metabolic alkalosis, with variable degrees of dehydration. Correction of the dehydration is mandatory before induction of anesthesia and surgery should proceed only when the metabolic derangements have been corrected. This can be confirmed by measuring urinary chloride excretion.

Ongoing Losses and Third Spacing

During surgery following losses can occur:

- **Whole blood loss:** It needs to be replaced with 1:1 volume of blood or colloids or 2.5:1 volume of crystalloids
- **Surgical trauma to cell membranes:** Causes hypoxic injury creating a capillary leak resulting in extravasation of isotonic, protein-containing fluid into interstitial compartments (the so-called third space)
- **Anesthetic-induced vasodilation:** (increased capacitance) and relative hypovolemia (a virtual loss)
- **Direct evaporation:** in very small infants.

Ongoing losses are very difficult to quantify. Estimates are usually quite rough. Nevertheless, one has to be very vigilant and prompt in action. Smaller the child, lesser is the circulating volume. Therefore, safety margin is very low. A small loss for an adult becomes very significant for a tiny infant.

Isotonic crystalloid is used to replace third space losses. Depending on mild, moderate or severe tissue trauma, fluid replacement of 3–4, 5–7, ≥10 mL/kg/h may be needed. Third space losses from the vascular compartment include both evaporation and redistribution of fluid. Too much of crystalloids can cause hemodilution and increased capillary pressures ultimately causing whole-body salt and water overload. In a relatively healthy child, most excess fluid is excreted over the first two postoperative days. However, in children with impaired pulmonary, cardiac or renal function, it can lead to pulmonary edema, bowel swelling and laryngotracheal edema. Many studies have showed improved outcomes with conservative fluid management in adults undergoing abdominal surgery. There is little evidence in pediatric patients for this issue. Perhaps a combination of crystalloid and colloid might be helpful.

Composition of Replacement Fluid

Fluid losses during major surgery are from the ECF compartment, and therefore, should be replaced by solutions containing the similar electrolyte composition. The constituents of these losses (i.e. high sodium and chloride, and low potassium, bicarbonate and calcium concentrations) differ from the composition of the solutions used for maintenance. Simply hiking the rate of infusion (i.e. volume) used for maintenance to compensate for these losses can be hazardous during major surgery. During minor surgery, this is not as crucial and the same solution can be used for both maintenance and replacement. As such there is increase in antidiuretic hormone (ADH) intraoperatively which causes free water retention. Large volumes of hypotonic solutions may rapidly diminish serum sodium and hence osmolality, ultimately resulting in undesirable fluid shifts. Finally, the volume expansion needed to counteract anesthetic-induced vasodilatation is difficult to achieve even with isotonic fluids.

Isotonic normal saline can be used for replacement, but hyperchloremic metabolic acidosis can occur if large amounts are administered. A balanced salt solution, such as LR can be used. Lactate gets rapidly degraded into bicarbonate and is a buffer.
Intraoperative Fluid Glucose Composition

Severe hypoglycemia can cause permanent neuro-developmental impairment if goes unrecognized and untreated, especially in neonates. Animal experiments have shown that mild hypoglycemia plus mild hypoxia or ischemia can cause cerebral injury in immature brains. Neonates need glucose in the maintenance fluid. Question arises in bigger infants and children. The estimated incidence of preoperative hypoglycemia is between 0% and 2.5% and is usually associated with fast durations from 8 to 19 hour, which should not occur if one follows the current ASA recommended fasting guidelines. However, most anesthesiologists are afraid of unrecognized hypoglycemia in children, particularly in those who have prolonged fast prior to surgery. Therefore, they continue to administer glucose containing fluids in the intraoperative period.

Most of the time there is stress-induced hyperglycemia, which will get further aggravated with administration of dextrose containing solutions. Hyperglycemia induced osmotic diuresis can cause dehydration and electrolyte abnormalities. In the presence of ischemia or hypoxia, there is impaired metabolism of excess glucose leading to accumulation of lactate and intracellular acidosis with subsequent cell death and neurologic impairment. Hyperglycemia should be prevented especially in neurosurgery.

Glucose containing solutions should not be used to replace intraoperative fluid losses. If dextrose containing maintenance solutions are used, it should be administered as a separate piggyback infusion using an infusion pump or other rate- or volume-limiting device to avoid accidental bolus administration. All studies involving very low dextrose solutions (0.9 or 1%) have demonstrated that, in healthy young children, hypo- and hyperglycemia are avoided during surgery. These solutions have to be isotonic. However, availability of such a solution is a problem in most parts of the world. After the age of 5 years, glucose-free isotonic solutions can be administered, as in adults.

Indications for Vigorous Glucose Monitoring and Need for Higher Glucose Supplementation

- Neonates and infants <6 months of age
- Debilitated infants
- Malnourished children
- Children undergoing cardiac surgery
- Children with endocrinopathies
- Children receiving beta-adrenergic blockers
- Neonates, children receiving hyperalimentation
- Prolonged surgical procedures
- Regional anesthesia combined with general anesthesia (reduction of the stress response to surgery).

Children with mitochondrial disease: They have higher glucose requirements. So, glucose 5–10% containing solutions are administered at maintenance rates and lactate containing solutions are avoided.

Children receiving hyperalimentation: Critically-ill children may be receiving hyperalimentation solutions preoperatively in the form of lipids and concentrated glucose/protein as two different solutions. It is advisable to stop the lipid solution during surgery. The concentrated glucose/protein solution should be continued at the same rate (because circulating insulin concentrations have acclimated accordingly). Some clinicians recommend reducing the established intravenous alimentation fluid infusion rate by 33–40% (to compensate for the reduced metabolic rate under anesthesia) and frequently checking blood glucose. This avoids the waste of the current alimentation fluid. Highly concentrated glucose solutions (D10 or D20) should not be abruptly stopped. This can precipitate hypoglycemia because of high levels of circulating insulin. Remember to deduct same volume from isotonic maintenance fluid volume.

European consensus statement for intraoperative fluid therapy in children was declared in Berlin in September, 2010. It was recommended that an intraoperative fluid should have an osmolarity close to the physiologic range in children in order to avoid hyponatremia, an addition of 1–2.5% instead of 5% glucose in order to avoid hypoglycemia, lipolysis or hyperglycemia and should also include metabolic anions (i.e. acetate, lactate or malate) as bicarbonate precursors to prevent hyperchloremic acidosis. Table 3 summarizes guidelines for intraoperative fluid administration. Table 4 depicts constituents of plasma and common intravenous fluids.

INTRAOPERATIVE COLLOIDS

Colloids can be considered whenever there is requirement for aggressive intraoperative fluid resuscitation, e.g. large-volume blood loss and nonavailability of blood or excessive insensible losses. The choice of colloid is mainly albumin in USA, starches in France, gelatins in Europe. In India blood and blood products are commonly used. Albumin is the gold standard most frequently used plasma expander for maintenance of colloid osmotic pressure in infants and neonates.
Hydroxyethyl starch (HES) was well tolerated and effective in preserving global tissue oxygenation during normovolemic hemodilution in children undergoing pediatric tumor resection.55,56 Newer, low molecular weight (MW) /low molecular substitution (MS) solutions have much less effect on hemostatic mechanisms than older, higher MW/higher MS solutions.23,57 HES 130/0.42/6:1 is very safe in children scheduled for surgery who have normal renal function; it maintains hemodynamic stability and produces only mild to moderate changes in acid–base status.58 In pediatric cardiac surgery, the data are quite varied.59,60 A meta-analysis of children and adults receiving HES during cardiac surgery, showed increased blood loss in patients receiving HES compared with albumin.61 The data supporting the use of gelatin in children are limited. Dextran are not used because of their negative coagulation effects and high anaphylactic potential.62

Hypertonic saline solutions (3%) (4 mL/kg) have been used in the treatment of refractory hypovolemic shock. It can rapidly mobilize fluid into the intravascular

<table>
<thead>
<tr>
<th>Table 3: Guidelines for intraoperative fluid administration</th>
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<tbody>
<tr>
<td>Maintenance fluid fasting compensation</td>
</tr>
<tr>
<td>Infant &lt; 3 month Hypoglycemia risk</td>
</tr>
<tr>
<td>Infant &gt; 3 month No hypoglycemia risk Major surgery</td>
</tr>
<tr>
<td>Infant &gt; 3 month Hypoglycemia risk Major /Minor surgery</td>
</tr>
<tr>
<td>Infant &gt; 3 month No hypoglycemia risk Minor surgery</td>
</tr>
</tbody>
</table>

Low dextrose means 0.9–1% dextrose solution

<table>
<thead>
<tr>
<th>Table 4: Constituents of plasma and common intravenous fluids</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constituent</td>
</tr>
<tr>
<td>-------------</td>
</tr>
<tr>
<td>Plasma</td>
</tr>
<tr>
<td>LR</td>
</tr>
<tr>
<td>0.9% NaCl</td>
</tr>
<tr>
<td>0.45% NaCl</td>
</tr>
<tr>
<td>5% D</td>
</tr>
<tr>
<td>DS ½ NS</td>
</tr>
<tr>
<td>DS ¼ NS</td>
</tr>
<tr>
<td>4% Dextrose /0.18% NaCl</td>
</tr>
<tr>
<td>PLASMA-LYTE A⁺ pH 7.4</td>
</tr>
<tr>
<td>PLASMA-LYTE 148/Normosol-R</td>
</tr>
<tr>
<td>PLASMA-LYTE R</td>
</tr>
<tr>
<td>Sterofundin **</td>
</tr>
<tr>
<td>3% NaCl</td>
</tr>
<tr>
<td>Isolyte P</td>
</tr>
<tr>
<td>Isolyte G</td>
</tr>
<tr>
<td>Isolyte E***</td>
</tr>
<tr>
<td>Isolyte M</td>
</tr>
</tbody>
</table>

* PLASMA-LYTE is a trademark of Baxter International Inc., its subsidiaries or affiliates. NaOH is present for pH adjustment to 7.4
** Sterofundin has maleate 5
*** Isolyte E contains citrate 8 mEq/L
space, improve preload, and thereby, cardiac output. They can improve organ blood flow and microcirculation. They are beneficial in the treatment of traumatic brain injury. Animal and clinical studies have suggested use of hypertonic saline in pediatric burn patients.

### POSTOPERATIVE HYponATREMIA

#### Incidence and Evidence

Hypotonic IV fluid induced hyponatremia have been reported in children undergoing surgery. Death and permanent neurologic injury have also been reported.

#### Causes

1. AVP secretion: Anxiety, prolonged fasting, inhalation anesthetic agents, positive pressure ventilation, haemorrhage, relative hypovolemia, pain, stress, nausea, sleep, nonsteroidal anti-inflammatory drugs and narcotic administration, are some of the factors causing release of AVP in perioperative period. Stress response has been noted even in minor surgery.

2. Many of these children have preexisting condition, (e.g. leukemia/chemotherapy, congenital heart disease/diuretic, therapy, chronic lung disease/ ex-premature) making them prone to excessive AVP secretion.

3. Lack of electrolyte monitoring and delay in recognition adds further to the problem.

4. Neuroendocrine activation by stress and hypovolemia-induced renin secretion, leading to water retention.

5. Hypotonic fluids: Children are prescribed IV fluids like 5% dextrose with 0.22%/0.45% NaCl. Though they are iso-osmolar, dextrose is metabolized in vivo, and therefore, is effectively hypotonic.

6. Replacement of ongoing losses (from wound, chest tubes, nasogastric suction, etc.) with hypotonic instead of isotonic fluid.

#### Reasons for Children being more Susceptible than Adults to the Effects of Hyponatremia

1. Relatively, large brain: Intracranial volume ratio; thus, there is a greater increase in intracranial pressure for any given increase in brain volume.

2. Immaturity of Na⁺/K⁺-ATPase mechanism in prepubertal age group.

3. Relatively smaller volume of cerebral spinal fluid, and hence, buffer capacity.

4. The brain intracellular concentration of sodium is about 27% higher in children than adults.

5. Low index of suspicion, and hence, delayed treatment.

#### Symptomatology and Outcome

The early symptoms of lethargy, headache, nausea, and vomiting are not specific to hyponatremia, and hence, are missed. Many a times, respiratory arrest is the first detected sign. Hyponatremia draws excess water into cells and causes them to swell. It presents as central nervous system symptoms, such as headache, malaise, nausea, lethargy, irritability and muscle weakness. The dreaded complications include encephalopathy with seizures, irreversible brain damage or brain death from cerebral herniation. Death has even been reported after minor surgery.

#### Investigations

SIADH consists of hyponatremia, low plasma osmolality, production of inappropriately concentrated urine with elevated urine osmolality (>200 mOsm/kg) and excessive urine sodium excretion (U Na >30 mEq/L).

#### Solution

There are two schools of thought: One is to give hypotonic fluid in less volume. Proponents of this theory say that extracellular deficits should be replaced first with isotonic fluids, followed by only the amount of fluid and electrolytes required to replace insensible losses and urine output. They argue that isotonic fluids could lead to hypernatremia or fluid overload. Acute volume expansion with saline is sufficient to suppress AVP production and prevent hyponatremia. Finally, it is preferable to give hypotonic fluids in less volume in states of AVP excess rather than to administer isotonic fluids.

Second theory is to give isotonic solutions. Advocates of isotonic fluid say that there are a number of problems in the Holliday and Segar approach to parenteral therapy. First of all, it was meant for calculation of fluid and electrolyte requirements for healthy children. Whereas, in acute disease or following surgery, there is 50–60% reduction in caloric expenditure, IWL, and urine output. In addition, production of endogenous water from tissue catabolism (water of oxidation) may be increased in acute disease. Secondly, this approach fails to recognize the importance of tonicity with its central role in the distribution of water between fluid compartments (intracellular and extracellular space).

It is essential to understand difference between osmolality and tonicity. The osmolality of a solution is the number of osmoles of solute per liter of solution. The
tonicity of a solution refers to the total concentration of solutes that exert an osmotic force across a membrane in vivo. For example, 5% dextrose with in vitro osmolarity of 286 mOsm/L H2O is rapidly metabolized in blood to water. Thus it’s in vivo tonicity is zero. Every liter of 5% dextrose infusion results in the expansion of the TBW by one liter (two thirds of this distributes to the intracellular space and one third to the extracellular space). Similarly, every liter of 0.22% and 0.45% saline results in formation of 800 mL and 500 mL of electrolyte free water, its respectively, which will expand the intracellular fluid (ICF) compartment.

POSTOPERATIVE FLUID MANAGEMENT

Type of Fluid

Use of isotonic fluid has been recommended. Multiple studies and two meta-analysis published in 2014 concluded that in hospitalized children in intensive care and postoperative settings, the administration of hypotonic maintenance fluids increases the risk of hyponatremia when compared with administration of isotonic fluids.90-93 Many editorials have been written on the subject.76,94-98 In 2007, The National Patient Safety Agency of the United Kingdom issued an alert recommending the removal of 4% dextrose with 0.18% saline from general use in children.99 The preferred fluids for maintenance therapy are either 0.45% saline with dextrose or isotonic fluids. Hypotonic fluids should be reserved for patients with either hypernatremia or ongoing extrarenal or renal free-water losses, such as voluminous diarrhea or renal concentrating defect.96

Type of Isotonic Fluid

There is a possibility of hypernatremia, edema, hypertension and chloride-induced acute kidney injury with 0.9% saline. Though randomized controlled studies have not identified these effects, none of the studies done till 2013 had sufficient power to detect these associations.100 Use of LR rather than saline might ameliorate this to a certain extent. Dextrose may be added to these isotonic solutions (commonly in concentration of 5–10%), when clinically indicated to avoid hypoglycemia without changing the solution’s in vivo tonicity.

Postoperative Pulmonary Edema

Children who receive large volume of fluid intraoperatively are at risk. Usually, second to fourth postoperative day fluid is mobilized back into circulation resulting in pulmonary edema. This is usually seen in children with burn injuries101 or in pediatric patients receiving large amount of fluid during resuscitation from trauma or sepsis.102

Postoperative Fluid Rate

Postrecovery IV fluid therapy should consist of an isotonic solution infused at half the rate described in the original 4-2-1 fluid regimen (i.e. 2 mL/kg for the first 10 kg, 1 mL/kg for the next 10 kg and 0.5 mL/kg for each additional kilogram thereafter). If the child does not or cannot tolerate oral intake after 6–12 hours, standard maintenance fluid therapy using hypotonic saline (e.g. 0.45% saline) at 4-2-1 rule rate should be initiated to avoid hypernatremia from prolonged administration of the isotonic solutions.31 This regimen should limit the ADH response and reduce the risk for postoperative hyponatremia and hypernatremia.103

Whenever ambient temperature is high, e.g. in summer in tropical countries, higher amount of fluid may need to be administered. Serial monitoring of serum electrolyte (at least once daily) is very important.104

CORRECTION OF WATER AND ELECTROLYTE ABNORMALITIES IN PERIOPERATIVE PERIOD

There are two ways of doing this:

- Find out the type of fluid lost. Volume of replacement should be equivalent to the amount lost. Composition should be same as the constituents of the fluid lost (Table 5)
- The degree of dehydration can be judged from the vital signs, general appearance and urine flow plus specific gravity (Table 6). Each 1% of dehydration corresponds to 10 mL/kg fluid deficit. Electrolytes can be measured. Deficits can be calculated and accordingly replaced.104
  - Untreated illness <3 days: 80% (0.8) ECF deficit, 20% (0.2) ICF deficit
  - Untreated illness ≥3 days: 60% (0.6) ECF deficit, 40% (0.4) ICF deficit.

Deficit Replacement Strategy

Phase I

Rapid fluid resuscitation with isotonic fluid (NS or LR) can be given. A bolus of 20 mL/kg represents only a 2% bodyweight replacement. Consider subtracting fluid and electrolytes given during resuscitation from the total deficits when calculating replacement of fluid and electrolytes.
### Table 5: Composition of various body fluids

<table>
<thead>
<tr>
<th>Body fluid</th>
<th>Electrolytes (mEq/L)</th>
<th>pH</th>
<th>Osmolarity (mOsm/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Na⁺</td>
<td>K⁺</td>
<td>Cl⁻</td>
</tr>
<tr>
<td>Gastric</td>
<td>50–70</td>
<td>5–15</td>
<td>120–150</td>
</tr>
<tr>
<td>Pancreas</td>
<td>140</td>
<td>5</td>
<td>50–100</td>
</tr>
<tr>
<td>Bile</td>
<td>130</td>
<td>5</td>
<td>100</td>
</tr>
<tr>
<td>Ileostomy</td>
<td>130</td>
<td>15–20</td>
<td>120</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>50</td>
<td>35</td>
<td>40</td>
</tr>
<tr>
<td>Sweat</td>
<td>50</td>
<td>5</td>
<td>55</td>
</tr>
<tr>
<td>Urine</td>
<td>0–100*</td>
<td>20–100*</td>
<td>70–100*</td>
</tr>
</tbody>
</table>

* Variable depending on fluid intake.

### Table 6: Clinical features for estimation of severity of dehydration

<table>
<thead>
<tr>
<th>Weight loss (%)</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infants &lt;10 kg</td>
<td>5</td>
<td>10</td>
<td>15</td>
</tr>
<tr>
<td>Child &gt;10 kg</td>
<td>3</td>
<td>6</td>
<td>9</td>
</tr>
<tr>
<td>Consciousness</td>
<td>Alert</td>
<td>Restless or lethargic, but arousable</td>
<td>Drowsy to comatose</td>
</tr>
<tr>
<td>Behavior</td>
<td>Normal</td>
<td>Irritable</td>
<td>Hyperirritable to lethargic</td>
</tr>
<tr>
<td>Thirst</td>
<td>Slight-Moderate</td>
<td>Moderate-Intense</td>
<td>Intense / drowsy and apathetic, so may not complaint</td>
</tr>
<tr>
<td>Anterior fontanel</td>
<td>Flat</td>
<td>Possibly sunken</td>
<td>Sunken</td>
</tr>
<tr>
<td>Eyes</td>
<td>Normal</td>
<td>Sunken</td>
<td>Markedly sunken</td>
</tr>
<tr>
<td>Tears</td>
<td>Present</td>
<td>Normal to reduced</td>
<td>Absent</td>
</tr>
<tr>
<td>Skin turgor</td>
<td>Normal</td>
<td>Decreased</td>
<td>Markedly decreased</td>
</tr>
<tr>
<td>Skin touch</td>
<td>Normal</td>
<td>Dry</td>
<td>Clammy</td>
</tr>
<tr>
<td>Mucous membranes</td>
<td>May be normal</td>
<td>Dry</td>
<td>Parched</td>
</tr>
<tr>
<td>Pulse rate</td>
<td>Normal</td>
<td>Slightly increased</td>
<td>Increased</td>
</tr>
<tr>
<td>Pulse volume</td>
<td>Normal</td>
<td>Weak</td>
<td>Feeble / impalpable</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>Normal</td>
<td>Normal or low</td>
<td>Reduced and orthostatic hypotension</td>
</tr>
<tr>
<td>Respiration</td>
<td>Normal</td>
<td>Deep</td>
<td>Deep and rapid</td>
</tr>
<tr>
<td>Capillary refill</td>
<td>&lt; 2 seconds</td>
<td>2–3 seconds</td>
<td>&gt;3 seconds</td>
</tr>
<tr>
<td>Urine flow (mL/kg/h)</td>
<td>&lt;2</td>
<td>&lt;1</td>
<td>&lt;0.5</td>
</tr>
<tr>
<td>Specific gravity</td>
<td>1.020</td>
<td>1.020–1.030</td>
<td>&gt;1.030</td>
</tr>
</tbody>
</table>

### Phase II

Deficit repletion, maintenance and ongoing losses. After initial stabilization, the remaining deficit is replaced over the next 24–48 hours. Table 7 depicts equations for calculating fluid and electrolyte deficits.

In isonatremic dehydration, equation no. 1, 2, 3 are taken into consideration. In hyponatremic dehydration, additional equation no. 4 is taken into consideration. Half of deficit is replaced over eight hours and remaining half over next 16 hours. In addition, maintenance fluid is administered.¹⁰⁴

In hypernatremic dehydration, equation no. 5, 6, 7, 8 are taken into consideration. Half of free water deficit is replaced over first 24 hours and remaining half over next 24 hours. Solute fluid, Na⁺, K⁺ deficit are replaced over first 24 hours. In addition, maintenance fluid is administered.¹⁰⁴

**Exercise 1:** A 15-kg (pre-illness weight) child who has been ill for >3 days is 9% dehydrated, with serum Na⁺ 138 mEq/L. Determine fluid schedule.
Principles and Practice of Pediatric Anesthesia

Exercise 2: A 15-kg (pre-illness weight) child who has been ill >3 days is 9% dehydrated, with serum Na+ 160 mEq/L. Determine fluid schedule.

Answer:
Total fluid deficit = (90 mL/kg) (15 kg) = 1350 mL; Free water deficit = 4 mL/kg × 15 kg × (160 - 145) = 900 mL; Solute fluid deficit (SFD) = Total fluid deficit – Free water deficit = 450 mL; Solute Na+ deficit = 0.45 × 0.6 × 145 = 39 mEq; Solute K+ deficit = 0.45 × 0.4 × 150 = 27 mEq
First 24 hours: 24 h maintenance + ½ of free water deficit + Solute fluid and electrolyte deficit = 2150 mL H₂O, 38 mEq Na+, 25 mEq K+. This can be supplied with 2.150 Liter 5%D with 0.22% NaCl + 8 mL KCl/L @ = 90 mL/h × 24 h.
Next 24 hours: 24 h maintenance + ½ of free water deficit = 1700 mL H₂O, 38 mEq Na+, 25 mEq K+. This can be supplied with 1.7 Liter 5%D with 0.18% NaCl + 7 mL KCl/L @ = 71 mL/h × 24 h. Rapid decrease in the serum Na+ by >15 mEq/L per 24 hours should be avoided, otherwise cerebral edema can occur.

DISTURBANCES OF POTASSIUM METABOLISM

Hypokalemia

Definition: Serum K+ < 3.5 mEq/L.

Causes: Inadequate potassium intake, nasogastric drainage, diarrhea, diuretics, renal dysfunction or alkalosis.

Electrocardiogram: Flattened T waves, prolongation of the QT interval or the appearance of U waves.

Table 7: Equations for calculating fluid and electrolyte deficits

<table>
<thead>
<tr>
<th>Eq No.</th>
<th>Name</th>
<th>Formula</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Fluid Deficit ml</td>
<td>(% dehydration) (10mL/kg) (weight in kg)</td>
</tr>
<tr>
<td>2</td>
<td>Na+ Deficit mEq</td>
<td>fluid deficit L × proportion from ECF × Na+ concentration mEq/L in ECF</td>
</tr>
<tr>
<td>3</td>
<td>K+ Deficit mEq</td>
<td>fluid deficit L × proportion from ICF × K+ concentration mEq/L in ICF</td>
</tr>
<tr>
<td>4</td>
<td>Excess Electrolyte Deficits mEq</td>
<td>(concentration desired mEq/L - concentration present mEq/L) × FD × weight in kg - pre-illness FD: distribution factor as fraction of body weight (L/kg): HCO3⁻ (0.4 - 0.5); Cl⁻ (0.2 - 0.3); Na⁺ (0.6 - 0.7)</td>
</tr>
<tr>
<td>4a</td>
<td>Additional Na⁺ Deficit</td>
<td>(desired Na⁺ concentration mEq/L - present S. Na⁺ concentration mEq/L) × 0.6 × weight kg - pre-illness</td>
</tr>
<tr>
<td>5</td>
<td>Free Water Deficit (FWD) mL</td>
<td>4 mL/kg × pre-illness weight kg × (concentration present mEq/L - concentration desired mEq/L)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>It requires 4 mL/kg to decrease serum Na⁺ by 1 mEq/L. If serum Na⁺ is &gt;170, estimate decreases to 3 mL/kg.</td>
</tr>
<tr>
<td>6</td>
<td>Solute Fluid Deficit (SFD) ml</td>
<td>The amount of additional fluid volume loss beyond free water loss in a patient with hyponatremic dehydration</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Total fluid deficit - Free Water deficit</td>
</tr>
<tr>
<td>7</td>
<td>Solute Na⁺ Deficit mEq/L</td>
<td>SFD L × proportion from ECF × Na⁺ concentration mEq/L in ECF</td>
</tr>
<tr>
<td>8</td>
<td>Solute K⁺ Deficit mEq/L</td>
<td>SFD L × proportion from ICF × K⁺ concentration mEq/L in ICF</td>
</tr>
</tbody>
</table>
Complications: Cardiac arrhythmias, ileus and lethargy.

Treatment of hypokalemia: Slow replacement of potassium either intravenously or orally, usually in the daily fluids. In extreme emergencies, potassium can be given as an infusion over 30-60 minutes of not more than 0.3 mEq/kg potassium chloride. If hypokalemia is secondary to alkalosis, the alkalosis should be corrected prior to potassium replacement.

Hyperkalemia

Definition: Serum K+ >6 mEq/L.

Causes: Potassium supplements, acidosis, renal dysfunction, intraventricular hemorrhage or tissue trauma, intravascular hemolysis and congenital adrenal hyperplasia.

ECG manifestations: Peaked T waves, widened QRS complexes, bradycardia, supraventricular tachycardia, ventricular tachycardia and ventricular fibrillation.

Treatment of hyperkalemia: Stop potassium intake, monitor ECG.

Medication:
- Calcium gluconate, 100 mg/kg IV over 2-5 min, has immediate onset, protects the myocardium from toxic effects of potassium
- Sodium bicarbonate, 1-2 mEq/kg has immediate onset, shifts potassium intracellularly
- Insulin 0.1-0.15 U/kg IV plus dextrose 0.5 g/kg IV has onset in 15-30 min, shifts potassium intracellularly. Duration of effects is 2-6 hour
- Albuterol inhalation, 0.15 mg/kg every 20 min for three doses, has onset in 15-30 minutes, shifts potassium intracellularly. Duration of effects is 2-3 hours
- Furosemide, 1-2 mg/kg/dose given every 12-24 hours, has onset in 15 minutes to 1 hour, increases renal excretion of potassium. Duration of effects is 4 hours
- Kayexalate, 1 g/kg per rectal every 6 hours, has onset in 1-hour (rectal route faster), removes potassium from the gut in exchange for sodium. Duration of effects is 4-6 hour.

DISTURBANCES OF CALCIUM METABOLISM

Hypocalcemia is much more common than hypercalcemia in perioperative settings.

Hypocalcemia

Definition: Plasma ionized calcium <1.0 mmol/L, or total calcium <7.0, 8.0 and 8.8 mg/dL or <1.7, 2.0 and 2.2 mmol/L in preterm, term newborns and children, respectively. 

Causes: Early onset neonatal hypocalcemia (ENH) (within the first 3-4 days of life) is seen in about 33% of infants <37 weeks gestation due to decreased parathyroid hormone (PTH) secretion, in 50% of neonates of insulin-dependent diabetic mothers due to hypomagnesemia, and in 30% of neonates with asphyxia due to limited calcium intake. Late onset neonatal hypocalcemia (LNH) is rare compared to ENH and presents at the end of the first week of life. This is usually caused by high phosphate intake (ingestion of Cow’s milk, renal insufficiency), hypomagnesemia, vitamin D deficiency, PTH resistance, hypoparathyroidism and iatrogenic reasons (citrated blood products, lipid infusions, bicarbonate therapy, loop diuretics, glucocorticosteroids, phosphate therapy, use of aminoglycosides mainly gentamicin as single dose, alkalosis, phototherapy).

Childhood hypocalcemia is mainly due to vitamin D or PTH deficiency, calcium malabsorption, hyperphosphatemia, hepatic rickets, acute pancreatitis, and renal osteodystrophy.

Symptoms: ENH is usually asymptomatic. Occasionally, symptoms of neuromuscular irritability, i.e. myoclonic jerks, jitteriness, exaggerated startle and seizures are seen. Apnea, cyanosis, tachypnea, vomiting and laryngospasm are other signs. LNH is usually symptomatic in the form of neonatal tetany or seizures.

Electrocardiogram: QTc (QT interval is measured from origin of q wave to end of T wave) = QT interval in seconds / square root of R-R interval in seconds >0.45 seconds QoTc (QoT is measured from origin of q wave to origin of T wave) = QoT interval in seconds / square root of R-R interval in seconds >0.22 seconds.

Complications: Seizures, laryngospasm, cardiac failure.

Treatment of Hypocalcemia

1 mL calcium gluconate 10% = 100 mg calcium gluconate = 9 mg / 0.46 mEq / 0.22 mmol elemental calcium.
1 mL calcium chloride 10% = 100 mg calcium chloride = 27 mg/1.36 mEq/0.68 mmol elemental calcium.

Neonates with asymptomatic hypocalcemia: 80 mg/kg/day elemental calcium (8 mL/kg/day of 10% calcium gluconate) should be administered for 48 hours. The dose should be tapered to 50% for next 24 hours and then stopped. If baby is tolerating oral feeds, then IV preparation can be used orally.
Neonates with symptomatic hypocalcemia: A bolus dose of 10% calcium gluconate 2 mL/kg/dose diluted 1:1 with 5% dextrose should be administered over 10 minutes under cardiac monitoring, preferably through central vein. This should be followed by a continuous IV infusion of 80 mg/kg/day elemental calcium for 48 hours. Calcium infusion should be reduced to 50% of the original dose for the next 24 hours and then discontinued. Intravenous cannula sites should be checked for extravasation to avoid subcutaneous tissue necrosis.

Tetany or seizure in neonate, infant and child: Calcium gluconate 100–200 mg/kg dose IV over 5–10 min, repeat dose 6 hour later if needed; maximum dose: 500 mg/kg/24 h.[106]

LEARNING POINTS

- Neonates have unique requirement of fluid and electrolytes because of high TBW, ECF and blood volume, increased capillary permeability, immaturity of skin leading to increased IWL, immature renal and cardiac function, decreased end-organ responsiveness to hormones involved in the regulation of fluid and electrolyte balance, and higher glucose and metabolic needs
- Premature and small-for-gestation age neonates have more water requirement
- Intravenous fluid administration has at least three components: (1) maintenance water and electrolytes to cover insensible and urinary losses; (2) replacement of preexisting deficits, either because of nil per os (NPO) orders or due to excessive losses; (3) replacement of ongoing fluid losses because of exposure, blood loss and third space loss
- Intraperative maintenance fluid requirement is calculated with 4-2-1 formula
- Replacement of deficit should be as per fluid lost
- Ongoing fluid losses are to be replaced with isotonic fluid like lactated Ringer’s solution
- Low dextrose solutions (0.9 or 1%) can prevent hypo- and hyperglycaemia in healthy young children intraoperatively
- Postoperative hypocalcemia is common because of raised AVP and faulty use of hypotonic solutions
- Children are more susceptible than adults to the effects of hypocalcemia because of multiple reasons
- Isotonic fluid at half the maintenance rate should be used in the initial 12 hours of postoperative period
- Both hypo- and hypernatremia have to be corrected slowly
- Hypokalaemia and hyperkalaemia require immediate, but careful correction for prevention of dangerous arrhythmias
- Hypocalcemia induced seizures require immediate treatment with intravenous calcium gluconate

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Assessment and Management of the Difficult Pediatric Airway

INTRODUCTION

Difficult airway (DA) management in children can be challenging even for an experienced anesthesiologist. Number of children with anticipated difficult airway has been increasing, both for airway and non-airway surgery. Further, unanticipated difficult airway after induction of anesthesia is one of the most dreaded clinical scenarios, which can rapidly result in life-threatening complications. Several reviews including ‘Perioperative cardiac arrest (POCA) registry’ data have found difficult airway and respiratory complications to be important contributors to perioperative cardiac arrest in children. The term ‘difficult airway’ represents a heterogeneous group of clinical conditions where different aspects of airway management are likely to be difficult for a conventionally trained anesthesiologist. Fortunately, present day anesthesiologists are equipped with better knowledge and understanding of managing difficult airway, wider range of age and size specific airway devices and well established guidelines and algorithms.

This chapter aims to cover the different aspects of pediatric difficult airway in a practically useful manner.

Pediatric Airway: Unique Anatomical and Physiological Features and their Implications

A summary of important differences are presented below (Table 1) along with their implications for the anesthesiologist. These are more prominent in smaller children, especially less than 2 years.

Table 1: Anatomical and physiological characteristics of pediatric patients

<table>
<thead>
<tr>
<th>Anatomy Feature</th>
<th>Implications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Large head</td>
<td>Towel roll under shoulder may be required for laryngoscopy</td>
</tr>
<tr>
<td>Narrow, sloping, omega (Ω) shaped epiglottis</td>
<td>Straight blade may offer better glottic view in small children</td>
</tr>
<tr>
<td>Cricoid ring narrowest part of the airway (this view is challenged now)</td>
<td>Trauma causes airway edema, disproportionately reducing airway diameter compared to adults</td>
</tr>
<tr>
<td>Asymptomatic subglottic stenosis may be present</td>
<td></td>
</tr>
<tr>
<td>Airway abnormalities can be part of syndromes</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Physiology Feature</th>
<th>Implications</th>
</tr>
</thead>
<tbody>
<tr>
<td>More than 50% premature newborns and 30% full term newborns are obligate nasal breathers</td>
<td>Difficulty in adjusting to oral breathing after surgery</td>
</tr>
<tr>
<td>Immature respiratory center</td>
<td>Increased risk of laryngospasm</td>
</tr>
<tr>
<td>Fatigue sensitive intercostal muscles, diaphragm and ribcage</td>
<td>Increased risk of airway obstruction, which is multi-level</td>
</tr>
<tr>
<td>Highly compliant chest wall and trachea</td>
<td>Rapid onset of hypoxia</td>
</tr>
<tr>
<td>High oxygen demand (6–7 mL/kg/min)</td>
<td>Surgeries like adenotonsillectomy, cleft lip and palate repair further increase the risk of airway related complications</td>
</tr>
</tbody>
</table>
ASSESSMENT

This is the first step of DA management. A DA can be obvious (e.g., severe micrognathia), doubtful (mild retrognathia or a small cystic hygroma) or may be easily missed leading to unanticipated DA. Goals of airway evaluation include the following:

a. Identification and confirmation of the presence of DA.

b. Establish the nature of the DA in terms of its impact on difficult mask ventilation, use of supraglottic airway devices (SAD), laryngoscopy and intubation, surgical access and patient cooperation.

c. Develop an appropriate plan and prepare the patient for airway management.

History, clinical examination of the airway, goal directed investigations and review of records are the cornerstones of assessment.

**History**

Majority of the children with anticipated DA could be asymptomatic and associated only with clinical findings.

Symptoms like snoring, noisy breathing, stridor, day time sleepiness, hoarseness of voice and adopting specific positions for sleeping indicate the possibility of acute or chronic airway problems. These can be caused by conditions like obstructive sleep apnea (OSA), tracheal stenosis, adenotonsillar hypertrophy, mediastinal mass or foreign body obstruction. Stridor can be inspiratory or expiratory. History of previous surgery or ICU admission along with details of endotracheal intubation and complications should be enquired into.

**Clinical Examination**

Predictors of DA should be identified during examination (Table 2). They can be congenital, inflammatory, and neoplastic in nature. In addition, presence of stridor, noisy breathing, wheeze, chest or suprasternal retraction and respiratory distress should be looked for. If there is evidence of airway obstruction, further attempt should be made to evaluate the patient to identify the site of obstruction, its nature and cause. Airway obstruction can be fixed or dynamic and intrathoracic or extrathoracic. The details are important for the management of the patient. Presence of an obstructed or compromised airway, congenital cardiac defects, syndromes, poor physical status and respiratory infection can adversely affect difficult airway management (Tables 3 and 4). Oxygen saturation in room air should be measured. Figures 1 to 6 illustrate common conditions causing DA in children. A list of syndromes and associated airway anomalies is depicted in Table 4.

<table>
<thead>
<tr>
<th>Table 2: Predictors of difficult airway, associated clinical conditions and their implications</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Predictor</strong></td>
</tr>
<tr>
<td>Stridor, respiratory distress</td>
</tr>
<tr>
<td>Large tongue</td>
</tr>
<tr>
<td>Reduced or absent mouth opening</td>
</tr>
<tr>
<td>Micronathia/retrognathia</td>
</tr>
<tr>
<td>Neck swelling</td>
</tr>
<tr>
<td>Defects in lip and palate</td>
</tr>
<tr>
<td>Facial defects and abnormal shapes</td>
</tr>
<tr>
<td>Short neck</td>
</tr>
<tr>
<td>Micronathia</td>
</tr>
<tr>
<td>Reduced mobility of neck</td>
</tr>
<tr>
<td>Short neck</td>
</tr>
</tbody>
</table>
Table 3: Factors complicating difficult airway management

- Recent or active respiratory infection. Lower respiratory infection is a contraindication for elective surgery
- Bronchial asthma
- Small age
- Enlarged tonsils and adenoids
- Radiotherapy
- History of prolonged intubation
- Loose teeth, missing teeth
- Congenital cardiac defects
- Poor ASA physical status (PS)
- Medical conditions like epiglottitis
- Full stomach
- Foreign body
- Tracheomalacia and bronchomalacia

Table 4: Syndromes and associated airway anomalies

<table>
<thead>
<tr>
<th>Syndromes</th>
<th>Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apert syndrome</td>
<td>Premature closure of cranial sutures, midfacial hypoplasia, cervical spine fusion</td>
</tr>
<tr>
<td>Beckwith-Wiedemann syndrome</td>
<td>Macrosomia, macroglossia, omphalocele, hemihyperplasia, nephromegaly, cardiomegaly</td>
</tr>
<tr>
<td>Crouzon syndrome</td>
<td>Beaked nose, mandibular prognathism, overcrowding of upper teeth, cleft palate, hypoplastic maxilla</td>
</tr>
<tr>
<td>CHARGE syndrome</td>
<td>Coloboma of the eye, choanal atresia or stenosis, cranial nerve abnormality, short wide ears</td>
</tr>
<tr>
<td>De Lange syndrome</td>
<td>Thin eyebrows that meet in the middle, long eyelashes, short upturned nose, thin downturned lips</td>
</tr>
<tr>
<td>Freeman-Sheldon syndrome</td>
<td>Microstomia, prominent forehead, mid face hypoplasia, long philtrum, chin dimple shaped like an ‘H’ or ‘V’</td>
</tr>
<tr>
<td>Goldenhar syndrome</td>
<td>Maxillary hypoplasia, dermoid cyst over the eye, cleft lip or palate</td>
</tr>
<tr>
<td>Hallermann–Streiff syndrome</td>
<td>Hypoplastic mandible, high arched palate, mal-implantation of teeth</td>
</tr>
<tr>
<td>Langer- Giedion syndrome</td>
<td>Short stature, thin upper lip, rounded nose</td>
</tr>
<tr>
<td>Tricho-rhino-phalangeal syndrome</td>
<td>Short stature, thin upper lip, rounded nose</td>
</tr>
<tr>
<td>Mobius syndrome</td>
<td>Micrognathia, microstomia, unusually shaped tongue, cleft palate</td>
</tr>
<tr>
<td>Mucopolysaccharidosis (Hurler, Hunter, Sanfilippo, Pourquoi Maroteaux-Lamy syndrome)</td>
<td>Flat nasal bridge, thick lips, glaucoma, hearing loss, enlarged mouth and tongue</td>
</tr>
<tr>
<td>Pierre – Robin sequence</td>
<td>Micrognathia, glossoptosis, horseshoe shaped cleft palate</td>
</tr>
<tr>
<td>Treacher Collins syndrome</td>
<td>Down slanting eyes, notched lower eyelids, malformed ears, absence of cheek bones</td>
</tr>
<tr>
<td>Sticklers syndrome</td>
<td>Flattened facial appearance, V shaped cleft palate, macroglossia</td>
</tr>
</tbody>
</table>

Note: Above is partial list of syndromes with airway anomalies. For individual syndromes also, only important airway abnormalities are mentioned. Impact of these findings on airway management should be considered on individual basis. Lastly, presence of congenital cardiac defects should be clearly ruled out.
ILLUSTRATIVE PICTURES OF DIFFICULT AIRWAY IN CHILDREN

Pierre Robin Syndrome
Problems: Small age, micrognathia, glossoptosis and cleft palate
- Difficult mask ventilation, difficult direct laryngoscopy and intubation. Nasopharyngeal airway will be helpful. Intubation fiberscope and video laryngoscope may be useful. SAD may be useful
- Can present for emergency glossopexy in neonatal period
- Difficulty decreases with age

Complicated bilateral cleft lip and palate
Problems: Cleft lip and palate, protruding pre-maxilla
- Mask ventilation may be difficult, larger size may be required; direct laryngoscopy difficult, left side laryngoscopy can be considered
- SAD may be useful as rescue device

Goldenhar syndrome
Problems:
- Difficult mask ventilation. Direct laryngoscopy difficult. Bougie, video laryngoscopy may be useful, fiberscope may be required in some patients
- SAD may be useful

Congenital TMJ ankylosis
Problems: No mouth opening, severe micrognathia
- Difficult mask ventilation
- Oral techniques not possible
- Fiber-optic guided nasal intubation is gold standard
- Retrograde intubation can be considered

Fig. 1: Pierre Robin syndrome
Fig. 2: Complicated bilateral cleft lip and palate
Fig. 3: Goldenhar syndrome
Fig. 4: Congenital TMJ ankylosis
Chapter 10: Assessment and Management of the Difficult Pediatric Airway

**Treacher Collins Syndrome**

Problems: Anteriorly placed larynx
- Difficult mask ventilation unlikely
- Direct laryngoscopy difficult
- Bougie, video laryngoscopy may be useful
- Fiberscope may be required in some patients
- SAD could be used

**Burns contracture**

Problems: Contracture of neck in extreme flexion
- Difficult mask ventilation
- Direct and video laryngoscopy difficult
- Fiber-optic techniques and SAD also may be difficult
- Contracture release under ketamine and tumescent anesthesia followed by definitive airway management

---

**PLANNING AND PREPARATION**

A. Consider the impact of the DA on
   - Mask ventilation
   - Endotracheal intubation
   - Insertion of SAD
   - Emergency procedures across the neck.

   In addition, the impact of co-morbidity, patient’s ability to cooperate and risk of aspiration are also to be considered (ASA practice guidelines 2013) as these factors also guide in decision making regarding type of anesthesia for the airway management.

B. Consider whether endotracheal intubation is required or can the airway be managed with SAD or whether airway management can altogether be avoided by choosing regional anesthesia. As a backup plan, preparation for endotracheal intubation or SAD should be always in place.

C. If endotracheal intubation is required for airway management, plan should be included.
   - Primary and alternate plan for endotracheal intubation
   - Strategies for management of failure of endotracheal intubation

   - Management of ‘cannot intubate, cannot ventilate’ situation.

   **Failure to plan for failure (of intubation, mask ventilation, etc.) is often the cause for complications.**

   **Factors affecting the choice of technique for endotracheal intubation are:**
   - Surgical procedure
   - Route of intubation: oral or nasal
   - Ease of mask ventilation
   - Ease of insertion of SAD
   - Risk of aspiration
   - Approach; supraglottic or infraglottic (anterograde or retrograde)
   - Choice of anesthesia; awake, sedation or general anesthesia with or without relaxant
   - Need for and feasibility of airway anesthesia
   - Availability of appropriate equipment and skill for a particular technique.

   Alternates to endotracheal intubation are use of supraglottic AD and surgical access to airway.

D. Preparation of the child should begin with detailed discussion with the parents about the proposed technique, type of anesthesia, complications and also the availability of alternate techniques. The child
should also be involved in decision making if he or
she is of appropriate age. Proper premedication and
airway anesthesia are crucial for success.
Premedication is used to provide anxiolysis and
antisialaglogue effects. Sedative premedication is
avoided in children with obstructive sleep apnea,
anticipated difficult mask ventilation and those who
are at risk of aspiration. Common premedications
include oral midazolam (0.5 mg/kg) or a combination
of midazolam with atropine or midazolam, ketamine
and atropine combinations. Midazolam can be
administered orally or by nasal route, in a dose of 0.2–
0.3 mg/kg for premedication.12
Facility for continuous supplementation of oxygen is
an absolute requirement for difficult airway manage-
ment, irrespective of the technique, equipment,
approach or type of anesthesia.

TECHNIQUES OF DIFFICULT AIRWAY
MANAGEMENT
These include (a) mask ventilation (b) endotracheal
intubation (c) supraglottic airway devices (d) surgical
access and (e) rescue techniques.

Mask Ventilation
It is the basic technique of airway management and a
very important determinant of specific technique and
device of airway management and the type of anesthesia.
If mask ventilation is not anticipated to be difficult,
further airway management can be performed under
general anesthesia, with or without muscle relaxation. In
case of anticipated difficulty in mask ventilation, awake
techniques should be considered and expert help should
be available.
Difficult mask ventilation is indicated by inadequate
mask fit, inadequate chest expansion, abdominal
distension, absence of good capnographic waves
and fall in oxygen saturation from the baseline value.
Management strategies are (a) optimizing jaw thrust,
chin lift and head tilt (b) use of oral or nasal airway
(c) two hand ventilation (d) two person ventilation
(e) lateral position and (f) use of continuous positive
airway pressure (CPAP) by partial closure of the APL
valves. During mask ventilation, the mouth should always
be kept open.
If difficulty persists, depending on the urgency of
surgery, the options are (a) waking up patient (b) to
further deepen the level of anaesthesia (c) paralysing the
patient (d) use of supraglottic airway device.

Endotracheal Intubation
In spite of advances in supraglottic airways devices
(SAD) endotracheal intubation is required for several
surgical procedures, either for perioperative management
or for postoperative ventilation. Also, presence of
endotracheal tube ensures a safe, stable and patent airway
during the procedure. Endotracheal intubation can be
performed awake, under sedation, general anesthesia
with spontaneous ventilation and with muscle relaxant.
Positioning for laryngoscopy and intubation may need
placement of a roll of towel under the shoulder. In
syndromic children with involvement of neck, or in those
with unstable cervical spine, obesity, etc. positioning related
difficulties predispose to difficulty in airway management.

Techniques for Endotracheal
Intubation Include
• Fiberoptic guided nasal or oral endotracheal intubation
• Video laryngoscopy assisted oral or nasal endotracheal
  intubation
• Endotracheal intubation with direct laryngoscopy
• Endotracheal intubation through SAD
• Retrograde intubation
• Use of intubation aids.
The above techniques are not mutually exclusive as they
can be used in combination. For example, intubation aids
can be used with any technique of intubation. Similarly,
video laryngoscope and fibrescope can be used together
in certain types of DA. Individual techniques are discussed
in more detail below.

Choice of Anesthesia
Awake techniques are practically not feasible in smaller
children. Also, neonatal awake intubation, once
considered common and acceptable practice, is no longer
recommended. When chosen in older and cooperative
child, it should be performed by an appropriately
experienced person and help should be available. Airway
anesthesia and controlled sedation should be considered
for improved success and patient comfort. Drugs like
dexmedetomidine, low dose ketamine and midazolam
can be used for sedation.13
General anesthesia (GA): It is often required for any type
of difficult airway management in small children and
uncooperative children of older age. GA can be safely used
for DA management in the absence of anticipated difficult
mask ventilation (adequate mouth opening, normal
mandible and intraoral contents) and risk of aspiration.
Even in majority of children with anticipated difficult mask ventilation, inhalational induction can be used for endotracheal intubation. Sevoflurane is the preferred inhalation agent.

Whether to paralyze or not before intubation is another important decision to make in pediatric DA management. If the difficulty is restricted to laryngoscopy and intubation and the center is well equipped to manage such situations, paralysing with either succinylcholine or even atracurium or rocuronium can allow the anesthesiologist to perform a smooth intubation without compromising patient safety. Atracurium is safer compared to rocuronium if sugammadex is not available as the former undergoes spontaneous degradation in the body.

Airway anesthesia is achieved using nerve blocks or nebulization or a combination of both with lignocaine. When multiple preparations of lignocaine are used by different routes, the total dose administered and consequently risk of local anesthetic toxicity should be kept in mind.

### Techniques of Airway Anesthesia

Airway anesthesia is indicated when airway needs to be intubated awake or under sedation, with fiber-optic guided techniques, blind intubation or retrograde intubation. The different methods of airway anesthesia are:

a. Nebulization of lignocaine: Advantages are simplicity and anesthesia of the lower airways.

b. Nerve blocks: It includes superior laryngeal nerve block, glossopharyngeal nerve block (less commonly used) and transtracheal injection of lignocaine.

c. Spray as you go (SAYGO), through intubation fiberscope.

#### Superior laryngeal nerve block
- Internal branch is blocked
- Given bilaterally
- Landmark: just beyond the tip of greater horn of hyoid bone
- 1% lignocaine 0.5–1 mL on each side
- Can cause hematoma
- Predisposition to airway obstruction in susceptible patients

#### Transtracheal injection
- Anesthetizes the surface of the airway in the infraglottic region
- Entry point is through the cricothyroid membrane or through the cricotraheal membrane with a syringe filled with lignocaine 1% and a 23G cannula attached. Direction of needle entry is caudal and the entry into the trachea is confirmed with aspiration of air.
- At this point, the needle is removed and local anesthetic injected through the catheter that is left behind. Patient may cough and this helps in spreading the local anesthetic

It is important to remember (a) to restrict the total dose of lignocaine to maximum allowable dose of 5 mg and 7 mg/kg, respectively for plain and adrenaline containing solutions (b) that land marks for nerve blocks are more difficult, structures are small and more closely placed (c) that airway blocks can predispose to airway obstruction in susceptible patients and are consequently contraindicated in children with actual or potential obstruction and (d) small children are unlikely to be cooperative to allow safe and smooth nerve blocks.

### Supraglottic Airway Devices

Broad guidelines which can help in making optimal use of these devices are (Table 5):

1. Choose the one which is most appropriate to the age and size of the patient.
2. Familiarity of the anesthesiologist with the particular device is important.
3. SAD is usually inserted after inhalational induction in children, particularly sevoflurane.
4. Propofol and other intravenous induction agents and small dose of succinylcholine (0.75–1 mg/kg) can also be used to facilitate SAD insertion.
5. In older children, awake insertion after topicalization of oral cavity is possible, followed by induction of anesthesia.
6. If a particular SAD cannot be positioned in the first or second attempts, a change should be considered to a different size of the same group (one size higher) or an altogether different device.
7. SAD can be considered in all children with difficult airway and its role should be defined (primary or back up, ventilation or intubation aid or rescue device).

### EQUIPMENT FOR MANAGEMENT OF DIFFICULT AIRWAY MANAGEMENT

#### Specialized Masks

Patil-Syracuse mask: Facilitates simultaneous ventilation while performing fiberoptic guided intubation.

#### Alternate Laryngoscopes and Blades for Direct Laryngoscopy

- **Miller Blade**: Straight blades may have advantage in terms of better visualization of glottic opening, in children less than 2 years. The epiglottis is lifted with the tip of the blade, unlike with Macintosh blade
- **Oxiport Blade** (Fig. 7): Modification of conventional Macintosh blade with provision for oxygen supplementation
**McCoys Blade:** Blade with flexible tip (Fig. 8). Pediatric size not available

**McMorrow’s Blade (Mirrored laryngoscope):** It is a new device with a mirror incorporated at the angulation of a Macintosh type blade, increasing the angle of vision when the blade is bent. The movement of the blade is controlled by a handle which stays posterior to laryngoscope handle.

**Video Laryngoscopes**

- **Airtraq:** Disposable, optical video laryngoscope with a channel; available in all sizes from neonate. Color coded. Neck extension is not required (Fig. 9).

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Table 5: Supraglottic airway devices

<table>
<thead>
<tr>
<th>Device</th>
<th>Sizes</th>
<th>Advantages</th>
<th>Disadvantages/ Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Classic LMA</td>
<td>1, 1.5, 2, 2.5, 3, 4, 5, 6</td>
<td>Simple, easy to use</td>
<td>No gastric channel, Not ideal for intubation, mal-positioning common</td>
</tr>
<tr>
<td>I gel</td>
<td>1, 1.5, 2, 2.5, 3, 4, 5</td>
<td>Pre-shaped cuff, simple, easy to use, color coded</td>
<td>Gastric channel present, intubation difficult</td>
</tr>
<tr>
<td>ILMA</td>
<td>3, 4, 5</td>
<td>Inserted without manipulation of head and neck, reduced stress response compared with TT</td>
<td>Airway less effectively protected from aspiration</td>
</tr>
<tr>
<td>Proseal LMA</td>
<td>1, 1.5, 2, 2.5, 3, 4, 5</td>
<td>Has a gastric drainage channel, double cuff and bite block. Airway tube is narrow</td>
<td>Higher sealing pressure and allows positive pressure ventilation Ideal in “full stomach” situations</td>
</tr>
<tr>
<td>Flexible LMA</td>
<td>2, 2.5, 3, 4, 5, 6</td>
<td>Airway management in restricted mouth opening</td>
<td>Smaller LMA’s can dislodge easily</td>
</tr>
<tr>
<td>Ambu Aura LMA</td>
<td>1, 1.5, 2, 2.5, 3, 4, 5, 6</td>
<td>Cuff is soft and flexible</td>
<td>Limit peak airway pressure to 20 cm H₂O, and tidal volume to 8 mL/kg</td>
</tr>
<tr>
<td>Air Q mask</td>
<td>1, 1.5, 2, 2.5, 3.5, 4.5</td>
<td>Creates a simple easier design for everyday use, no inflation line necessary, cuff inflates with positive pressure ventilation</td>
<td>Not much clinical evidence AirQ has a self-pressurizing cuff</td>
</tr>
<tr>
<td>Laryngeal tube</td>
<td>0, 1, 2, 2.5, 3, 4, 5</td>
<td>Used in patients with DA</td>
<td>Different types are available LTSII is the most recent one</td>
</tr>
<tr>
<td>Baska airway</td>
<td>3, 4, 5, 6</td>
<td>Used with spontaneous and controlled ventilation</td>
<td>Recently introduced. Has two gastric drainage channels, gastric sump and variable pressure cuff</td>
</tr>
</tbody>
</table>
Chapter 10: Assessment and Management of the Difficult Pediatric Airway

- C-Mac video laryngoscope is a rigid video laryngoscope with both Macintosh and Miller blades. There is a special D blade with unique angulation for difficult airway (Figs 10 and 11A).
- GlideScope: It has a blade with 60 degree angulation in the middle and a miniature camera mounted on blade. A special stylet, verathon stylet is available with the GlideScope. Blades are available for different ages from neonates. Different models are GlideScope titanium, cobalt, AVL and ranger (Fig. 11C).
- McGrath video laryngoscope (Fig. 11D).
- King Vision video laryngoscope has a monitor which can be mounted on the handle. It has both curved and channeled blades (Fig. 11B).

General comments on Video Laryngoscopes

- Multiple products, each with range of blades, design and specifications
- Blades can be channeled, curved, straight and angulated
- Stylet may be required
- Channeled blades require wider mouth opening than the non-channeled ones
- Recording facilities, disposable blades or covers for the blades are available
- VL should be considered as first choice in any difficult airway with mouth opening as per new ASA guidelines
- Every anesthesiologist is expected to be familiar with at least one device

Fig. 10: C-Mac video laryngoscope with straight curved and D blade

Figs 11A to D: Video laryngoscopes
**Intubation Aids (Fig. 12)**

- **Guide wire**: Useful for indirect technique of intubation through fiber-optic or through SAD. Also used for retrograde intubation.
- **Bougie**: Adult and pediatric sizes are available. Acts as intubation guide and are invaluable in difficult laryngoscopy and intubation through SAD in older children.
- **Airway exchange catheter**: useful for exchange of tubes and extubation of difficult airway.
- **Frova introducer**: Another intubation aid with an angulated tip.
- **Aintree intubation catheter**: Used as a guide for fiber-optic intubation.

**Optical Stylets or Lighted Stylets (Fig. 13)**

- **Bonfils retromolar intubation scope**: with a rigid angulated tip (Fig. 14)
- **Shikani stylet** (Fig. 15)
- **Trachlight**.

**Rescue Devices**

- **Cricothyrotomy set**
- **Surgical tracheostomy**
- **Jet ventilation**.

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**Fig. 12**: Intubation aids

**Fig. 13**: Optical stylets or lighted stylets

**Fig. 14**: Bonfils retromolar scope

**Fig. 15**: Shikani stylet
TECHNIQUES OF ENDOTRACHEAL INTUBATION IN DIFFICULT AIRWAY

Techniques with Direct Laryngoscopy

Due to either non-availability or lack of training with other devices, this is the commonest technique used in difficult airway management also. DL is not possible or difficult in significantly reduced mouth opening (TMJ ankylosis), intraoral mass, severe retrognathia, bilateral cleft lip and cleft palate, cystic hygroma, connective tissue disorders like Hunter’s syndrome, severe macroglossia, morbid obesity, etc.

- Modifications like change of blade (from Miller to Macintosh and vice versa), use of optimal positioning (rolled towel between the scapulae), optimal external manipulation, use of intubation aids like bougie should be considered to increase the success with DL, especially if the first attempt is not successful
- Number of attempts should not be more than three under any circumstance, as this could convert a difficult airway into a “failed airway.” Further, the first attempt should be optimally performed (position, equipment, performer) and any subsequent attempts should always be with a well thought change in any of the factors affecting the success of DL. Help should be called for immediately after the unsuccessful first attempt
- Backup plan options include (a) change over to video laryngoscopy (b) fiber-optic guided intubation or (c) SAD for temporary or definitive airway management (d) retrograde intubation and (e) waking up of the patient.

Choice of anesthesia depends on the ability to mask ventilate, risk of aspiration and cooperation of the child. In case of general anesthesia, spontaneous ventilation is preferable.

Video Laryngoscopy

Video laryngoscopes now available in pediatric sizes, are very useful in difficult airway management.14-16

Updated ASA algorithm recommends consideration for video laryngoscopy as the initial choice. This is logical in a difficult airway as attempting a VL after repeated attempts of DL can reduce the success rate. Now, wide variety of VL are available with pediatric blades, such as C-Mac video laryngoscope, glidescope, King Vision VL, etc. The one with which the anesthesiologist is familiar is the best one.

Technique of VL consists of four basic steps: (a) look at the mouth and introduce the VL blade, in midline or from the angle (b) position the tip appropriately, looking at the monitor (c) place the appropriately shaped and proper size endotracheal tube at the glottic opening looking at the mouth and lastly (d) observe the passage of endotracheal tube on the monitor. Thus, VL is a sequence of ‘look at mouth’ – ‘look at monitor’ – “look at mouth” – “look at the monitor”.

As described above, VL can be combined with fiberoptic guided intubation and also can be used to pass an intubation aid, bougie or Frova introducer, in case the tube cannot be directly passed in spite of the good laryngoscopic view.

Fiberoptic Guided Intubation Techniques

Fiberoptic guided or fiber-optic aided airway management is not a single technique, but a group of techniques for primarily performing the endotracheal intubation, using the intubation fiberscope, also called fiberoptic bronchoscope.17,18 Intubation fiberscope can also be used for other aspects of airway management, such as preoperative evaluation of a difficult airway, change of nasal to oral endotracheal tube and vice versa and also to aid in extubation of a difficult airway. The instrument, fiberscope, is a versatile instrument providing a clear picture of the airway as it is advanced down towards the carina from the nose or from the oral cavity. It is available in different sizes ranging from 1.8 mm diameter to 5.7 mm diameter to cater to different ages of the patient (Fig. 16).

Fiberoptic guided nasotracheal intubation is considered as gold standard of difficult airway management. Overall advantages of fiberoptic guided techniques of airway management in difficult airway, indications and contraindications are listed in Table 6.
A child with difficult airway can be intubated with intubation fiberscope by nasal, oral or fiberoptic assisted retrograde approaches.

**Preparation of the Equipment and Patient**

Success of fiberoptic aided techniques, like any other technique of difficult airway management, depends a lot on the selection and preparation of the proper equipment, patient and having a backup plan for failed fiberoptic intubation. Irrespective of the techniques, following general principles should always be kept in mind.

- **Proper size fiberscope is the one over which an appropriate size endotracheal tube can be loaded smoothly, without being too tight or too slack over the insertion cord**
- **Proper patient selection is important and so is the experience of the performer**
- **Fiberoptic aided intubation is neither the sole technique in all cases of difficult airway management nor can it be always successful.** It implies that (a) role of fiberoptic technique should be well defined in a given child with DA and (b) alternate plan should be ready to manage failure
- **Fiberoptic technique, if chosen, should be the first airway technique to be performed in a given patient, preferably first in the surgical list as well**
- **Preparation should be meticulous shown in Table 7.**

**Fiberoptic Guided Nasotracheal Intubation (NTI)**

It is preferred for patients with limited or no mouth opening or for surgeries in and around the airway. Also, this is the commonest method of intubating using a fiberscope. Different techniques are:

- **a.** Direct nasotracheal intubation with a preloaded tube over the intubation fiberscope.
- **b.** Indirect technique of using one nostril for passing the fiberscope and the other nostril for passing the endotracheal tube.
- **c.** Indirect technique for passing a guide wire, epidural catheter or bougie through the fiberscope into the trachea, removing the fiberscope and railroading the endotracheal tube blindly over the above mentioned intubation aids.

Indirect techniques are chosen when the adult size fiberscope is available and the child needs a tube whose size is smaller than the diameter of the fiberscope. Thus, a
2-year-old child’s airway can be intubated with a 3.7 mm diameter fiberscope, by passing the fiberscope through one and the tube through the alternate nostril.

Steps of the direct nasotracheal intubation technique in addition to the common preparation shown in Table 7:

- Insert the scope into the chosen nostril which has already been prepared with vasoconstrictor and local anesthetic
- Identify the turbinate and proceed gently with forward movement of the scope, lateral movement of the entire scope, and up and down movement of the control lever to keep the air passage in the center
- Identify nasopharynx where some resistance may be encountered
- Identify the epiglottis in the oropharynx. If required, jaw thrust or protrusion of the tongue can be helpful
- Additional dose of local anesthetic may be injected if required
- Pass the scope through the glottic opening during maximum inspiration when the opening is widest

- Trachea is confirmed by visualization of tracheal rings anteriorly
- Visualize the carina and stop the advancement. Stabilize the fiberscope
- Railroad the endotracheal tube over the scope gently. If there is resistance, usually by the right arytenoid, gentle anticlockwise rotation and pressure will help to overcome the same
- The scope is gently removed. If there is significant resistance, the entire scope with the tube should be removed and the whole procedure repeated
- Confirm the placement of the tube with capnogram
- While doing the scopy, a nasopharyngeal airway kept in the other nostril may be connected to a pediatric circuit and anesthesia can be maintained.

Techniques of indirect nasotracheal intubation

- Technique 1: The fiberscope is passed through one nostril and endotracheal tube is passed through the other. When the glottic opening is visualized, the scope is stabilized and endotracheal tube is guided into the trachea
• **Technique 2:** 2 stage technique. Initially, fiber-optic scopy is done and glottic opening is visualized. An Aintree catheter, preloaded over the fiberscope, is passed into the trachea and scope is removed. Oxygen can be connected to Aintree catheter. Endotracheal tube is railroaded over the Aintree catheter.

• **Technique 3:** 3 stage technique. Initially, guide wire is passed through the working channel of the fiberscope and fiberscope removed. Since, it is difficult to pass endotracheal tube directly over a guide wire, an Aintree catheter is passed initially over the guide wire and followed by railroading of the endotracheal tube.

The techniques 2 and 3 described above can be used for orotracheal intubation and fiber-optic assisted intubation through a SAD also.

**Fiberoptic Guided Orotracheal Intubation (OTI)**

Though less commonly performed than nasal, oral route provides a shorter route to airway and avoids the difficulty of passing through the nasopharynx. It can be chosen in any patient in whom nasal intubation is not required and mouth opening is adequate. Different techniques OTI are:

a. Direct oral intubation
b. Fiberoptic assisted placement of an intubation aid, followed by railroading of the tube
c. Intubation through SAD
d. VL assisted fiberoptic guided intubation.

The indications for alternate techniques (a, b and c above) are failure of the direct technique and non-availability of the appropriate size fiberscope.

**Fiberoptic Assisted Retrograde Intubation**

Retrograde intubation is described below in detail. Guide wire passed retrograde from the trachea can be threaded into the working channel opening at the tip of the fiberscope which is passed through the nasal route. The fiberscope should be preloaded with the endotracheal tube. The guide wire then acts as a guide for the advancement of the fiberscope.

**Intubation through SAD**

Trachea can be intubated through a SAD (Fig. 18) under certain circumstances:

1. Electively after inducing and stabilizing general anesthesia with a SAD.
2. After the failure of the initial attempts at intubation, SAD can be inserted, patients ventilation stabilized and then intubation attempted through SAD.

3. Direct intubation through SAD is easier with ILMA (Fastrach) and Air Q intubating airway. When direct intubation is not possible through a SAD, indirect techniques can be used. They are:
   • Fiberoptic guided intubation through SAD
   • Passing of a bougie, guide wire or airway exchange catheter under fiber-optic vision or blind and then railroading of tracheal tube.

**Confirmation of Endotracheal Intubation**

ETCO₂ is the gold standard of confirmation and proper positioning of endotracheal tube and at least 6 cycles of capnographic waves should be observed. Observation of passing of the endotracheal tube between the vocal cords, tracheal rings and carina with intubation fiberscope also provides a definitive confirmation of proper placement of the tube. Desaturation is a late sign of failed intubation. The dictum is *when in doubt remove the tube and reintubate. No patient dies because of inability to intubate, but can die because of failure to recognize esophageal intubation.*

**Trouble Shooting During Endotracheal Intubation**

**Difficult visualization of glottic opening with direct laryngoscopy**

Reposition, change of blade, external pressure, *bimanual laryngoscopy* (operator performing both laryngoscopy and external manipulation and assistant passing the endotracheal tube), use of intubation aids, change to VL.

**Inability to pass the tube, in spite of proper visualization**

Use of bougie, external manipulation, VL, change of blade.
Failed first attempt at intubation
Second and maximum third attempt with (a) mask ventilation with 100% oxygen in between attempts (b) modification/change in equipment, person, use of additional drugs (relaxant, propofol, fentanyl) and (c) continued monitoring of the patient.

Not more than 3 attempts of intubation are acceptable, the reason being that (a) further attempts increase chances of airway edema, bleeding and obstruction, leading to 'can’t ventilate, can’t intubate’ situation which can result in significant morbidity and mortality.

Exit Strategies
It refers to options to be considered in case of failed intubation. They include: (a) use of SAD for ventilation and continuation of anesthesia and proceed with surgery (b) use of SAD for ventilation and subsequent intubation through the same (c) use of SAD for ventilation and waking up the patient (d) continue with face mask ventilation and wake up patient or proceed with surgery, if it is short or life saving.

Rescue Procedures
These are life-saving and often temporary procedures. They are described as ‘Plan D’ and ‘procedures across the neck’ (Difficult Airway Society Guidelines). Rescue techniques include needle/surgical cricothyrotomy with manual or transtracheal jet ventilation (TTJV) and surgical tracheostomy. Other than tracheostomy, all the rescue procedures are meant to provide ventilation temporarily till such time a definitive airway is established. This can lead to air trapping and carbon dioxide build up. If SAD is in place, then it can provide at least a passage for expired air.

Cricothyroid membrane should be identified before the beginning of airway management as it could be difficult in a child in emergency. The area can be painted and draped and the ENT or pediatric surgeon familiar with tracheostomy can be scrubbed up during non-invasive airway management. The purpose is to minimize the time of desaturation if intubation and/or ventilation fails. This strategy has been referred to as ‘double step intervention’.

Needle cricothyrotomy is preferred over surgical, in children less than 5 years of age.

Role of Supraglottic Airway Devices
Availability of and advances in SAD have made significant difference in pediatric difficult airway management (Figs 19 to 23). SAD can be used as;

- Primary ventilation device to provide general anesthesia, without intubation.
- Emergency ventilation device to manage (a) difficult mask ventilation (b) difficult endotracheal intubation (c) ‘cannot ventilate, cannot intubate’ situation.
- Conduit for endotracheal intubation, most commonly fiber-optic guided direct intubation.
- To aid extubation in difficult airway (Bailey’s technique).

Advantages of SAD
- It can be alternative to both mask ventilation and endotracheal intubation
- Ease of insertion, can be inserted blindly with a faster learning curve
- Less depth of anesthesia, can be inserted awake also
- No need for muscle relaxation
- Reduced incidence of laryngospasm and desaturation in presence of hyper reactive airway
- It can be left in place till patient is completely awake without significant cardiovascular stimulation
- It can be used as a conduit for intubation
- It can be used to perform diagnostic fiberoptic bronchoscopy to evaluate infraglottic pathology
- Avoids complications specific to endotracheal intubation.
Extubation of a Difficult Airway

In a difficult airway, extubation is as important, if not more, as intubation. This is because, during extubation there are additional factors which will affect the patient safety such as the changes in the airway due to surgery, trauma, or rarely prolonged intubation and effect of residual anesthetics and relaxants. Consequences of improperly planned extubation include risk of postoperative airway obstruction, aspiration, hypoventilation, etc. all potentially requiring reintubation.22,23 All techniques of airway management, mask ventilation, SAD insertion and intubation could become difficult in the immediate postoperative period.

To ensure smooth extubation, children with DA can be classified as low and high risk (Table 8).

‘Plan, prepare, perform and postoperative care’ are the steps in extubation (DAS guidelines for extubation)24

Key components of safe extubation are (a) inspection of the airway at the end of surgery to rule out edema, bleeding, secretions (b) thorough suctioning (c) complete reversal from muscle relaxants, as evidenced by clinical criteria and train of four (TOF) ratio of more than 0.9 (d) complete recovery from anesthetics (e) use of airway exchange catheter if required.

Patient should be monitored for at least half an hour in high dependence area after an uneventful extubation.

Potential complications associated with extubation in a child with difficult airway are similar to those with normal airway and include airway obstruction, laryngospasm and hypoxia. But the management in former can be much more difficult, both for mask ventilation and reintubation.

Management of Post-extubation Complications

Post-extubation Complications

- Laryngospasm
- Bronchospasm
- Airway obstruction
- Aspiration
• Post-intubation croup
• Negative pressure pulmonary edema.

These complications can be mild or life threatening and managed aggressively. By far, the most important complication is laryngospasm.

**Laryngospasm: Causes and management**

Persistent, pathological and involuntary closure of the glottic opening in response to certain stimuli.

**Causes:** Extubation in light plane of anesthesia, presence of secretions and blood in the airway.

**Prevention:** Extubation in deep plane or fully awake, ensuring adequate reversal and clearing the airway before reversal. Administration of small dose of propofol (0.5 mg/kg) IV before extubation also can be helpful.

**Management:** Early recognition is very important. Laryngospasm is indicated by difficult mask ventilation after extubation, fall in SpO2, and subsequently bradycardia:

- CPAP with 100% oxygen
- 2 hand ventilation may be required
- Propofol IV (1mg/kg)
- Endotracheal intubation, if laryngospasm does not respond
- Succinylcholine 0.5 mg/kg
- Pressure at Larson’s point

**Post-obstructive pulmonary edema (POPE)**

- Occurs after relief of prolonged obstruction
- Also called negative pressure pulmonary edema

Treatment is with endotracheal intubation, positive pressure ventilation with PEEP, fentanyl or morphine and diuretics

**Postintubation croup**

Risk factors: Tight fitting endotracheal tube, repeated attempts during intubation, changes in position during the procedure, non-supine positions, traumatic intubation, 1–4 years of age, long duration surgery, coughing on the tube during recovery and respiratory infection.

Treatment is with nebulized adrenaline and dexamethasone

Subglottic stenosis is a delayed complication of airway management and predisposing factors include too large endotracheal tube, laryngeal trauma, prolonged intubation (more than 25 days), repeated intubation, sepsis and chronic inflammation.

**Difficult Airway Algorithms**

Algorithms and guidelines help in decision making, planning for airway management and also minimizes errors.

ASA difficult airway algorithm is meant primarily for adult patients undergoing elective general anesthesia. It emphasises a systematic approach to management of difficult airway. The principles are applicable to pediatric patients as well.

**“Take home” messages from Algorithms**

- Airway assessment is the starting point of difficult airway management
- Identification of nature of difficulty: Mask ventilation, intubation, SAD, aspiration, patient cooperation
- Clear and detailed primary and backup plan
- Facility for continuous oxygen administration during DA management
- To consider role of VL and SAD clearly in a ‘customized’ way
- Multiple attempts of the same techniques (DL, SAD insertion) should be avoided. Any repetition of the same technique should be based on sound reasoning as to the reason of failure of first attempt. It should incorporate changes in positioning, person and/or equipment
- Adequate local anesthesia, analgesia, sedation or depth of anesthesia for muscle relaxation (as per the plan) should be ensured
- Exit plan should be ready for every failed technique
- Training in rescue devices is essential
- Extubation strategy should be included in the plan

**Approach to Unanticipated Difficulty in Intubation**

1. Is mask ventilation possible?
   If yes, choices are:
   a. Further intubation attempts with changes in position, technique and/or person. The change can be in the form of an alternate size or type of blade, intubation assist device (bougie, Frova introducer, etc.), change in position or optimizing external laryngeal manipulation.
   b. Use of video laryngoscope for intubation.
   c. Use of supraglottic airway device as a definitive airway device or as a conduit for intubation, blind or fiber-optic guided.
   d. Retrograde intubation.
   e. If first 2 or 3 attempts fail consider waking up as exit strategy if permitted by the surgical condition.

2. If mask ventilation is difficult/progressively becomes difficult/impossible, then it is an emergency situation. Goal is to prevent ‘can’t intubate, can’t ventilate’ situation.
   **Available options are:**
   a. Improve mask ventilation with (a) two hand technique (b) two person technique (c) oral/nasal airway insertion (d) lateral position and (e) use of CPAP.
   b. Consider paralyzing the child (very critical judgment based on the experience of the anesthesiologist and expertise available). Usually limited to the situation where the difficult mask ventilation is likely to be due to supraglottic
Increased use of supraglottic airway devices

**Controversies in Difficult Airway Management**

- Use of succinylcholine in difficult airway: When there is no known contraindication, succinylcholine can be safely used for endotracheal intubation in presence of difficult airway
- Whether to paralyze or not in presence of difficult mask ventilation: In presence of unanticipated difficult mask ventilation, muscle relaxant can be administered if (a) the anatomy of the airway during assessment was found to be reasonably normal (b) there is no risk of aspiration (c) no subglottic pathology suspected (stenosis, obstruction, mediastinal mass etc.) and (d) anesthesiologist is well experienced in difficult airway management (e) help is available and (f) supraglottic airway can be inserted if required. *Otherwise, waking up is the safest option*
- Can regional anesthesia be the choice of anesthesia for difficult airway management? Again, answer largely depends on (a) nature of the surgical procedure (b) likely success of regional anesthesia (c) expertise and most importantly, ability of the anesthesiologist to manage the airway, in case of either failure or complications of regional anesthesia or the surgical procedure outlasting the duration of regional anesthesia.

**Recent Advances**

- Advent of 3rd generation of supraglottic devices, many of them in pediatric sizes
- Micro cuff tubes. The advantages are reduced leak and atmospheric pollution, better positive pressure ventilation and reduced need for change of endotracheal tube
- Changing concepts about the standard teaching that “subglottic region is the narrowest part of the pediatric airway”

**Changing Trends in Practice**

- Increased use of supraglottic airway devices
- Increased use of video laryngoscopes as the first choice for laryngoscopy.

**REFERENCES**

1. Jeffrey PM, Bhananker SM. Recent findings from POCA registry. American society of anesthesiologists article. 2005;69(2).
INTRODUCTION

The International Association for the Study of Pain (IASP) defines pain as “an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage.” Undiagnosed and undertreated pain leads to long-term physiological, biochemical, and psychological complications. As pain is a subjective experience, small children, physically and mentally handicapped children are unable to describe it and hence their pain assessment becomes difficult. This is one of the major causes of inadequate pain management in children. Other causes of poor pediatric pain management include myths that infants cannot feel pain because of their immature nervous system, young children do not respond to or remember the painful experience as much as adults, use of mild analgesics and inadequate doses for fear of harmful effects of these drugs in pediatric age group and parental misconceptions regarding pain assessment and management with the belief that ‘no pain, no gain’.2,3

In last 2-3 decades, with increase in research and awareness about pediatric pain management, we are better equipped to assess and control the severity of pain. This has led to development of pediatric pain services under the direction of pediatric anesthesiologists. They provide pain management for acute, postoperative, terminal, neuropathic and chronic pain. This should help in reducing the incidence of inadequate pediatric pain management. This chapter reviews different modalities of pain assessment and treatment of pediatric pain with the recent advances.

PAIN ASSESSMENT

Pain should be monitored as ‘the fifth vital sign’ as it is very important to optimally manage the sick child. Goal of pain assessment is to provide accurate information about location, duration and intensity of pain, as well as the effectiveness of measures used to alleviate or abolish pain. When treating pain at any age, it is essential to monitor the response to therapy with an objective scoring system. Pain scores should be regularly recorded on the patient’s vital signs chart.

Children may not report pain because of fear of talking to strangers, disappointing or bothering others, receiving an injection or returning to the hospital if they admit to pain. Children with persistent unrelieved pain may behave differently, depending on their understanding and coping capabilities. Child may scream, grimace, thrash around in the bed, or may withdraw from the surrounding and appear very quiet. Maladaptive behavior and reduction in functions may indicate pain in the cognitively impaired child. Hence in children, parents or caretakers become the key assessor of pain.

Pediatric pain can be measured by any of the three strategies and their combinations.

- Self-report measures: What children report about their experience
- Behavioral measures: The way that children react in response to pain
- Biological/Physiological measures: How children’s bodies respond to pain.
Depending on these, multiple validated scales or scores are available to measure and assess pain in children of all ages (Table 1). Most of them are validated for acute or postoperative pain. In older children, self-reporting measures like numbers (visual analogue scale, numerical rating scale), pictures (Oucher scale, six face pain scale of Wong and Baker and its modification) (Fig. 1), color, word graphics and poker chips are utilized. These cannot be used in cognitively impaired, preverbal, or sedated, intubated patients. In them pain is assessed by observational measures by seeing changes in heart rate, blood pressure, levels of adrenal stress hormones or by utilizing behavioral approaches like facial expressions, posture, body movements and crying intensity (CRIES scale, revised FLACC scale). Combination of behavior (six) and physiologic (two) parameters as in COMFORT scale, improves the assessment. To accurately locate the pain, dolls or action figures or drawings of body outlines, both front and back are used for the child to point out the painful area.

### NEUROPHYSIOLOGY OF PAIN

Pain pathway in humans develop in 2nd and 3rd trimester and by 24 weeks of gestation they are sufficiently mature for premature neonate and even fetus to respond to painful stimuli. The pathway like in adults consist of nociceptive fibers in peripheral nerves entering the spinal cord via the dorsal horn which are then transmitted via ascending spinothalamic, spinoreticular, and spinomesencephalic tracts to the thalamus, limbic system, and sensory cortex. From the sensory cortex, descending control pathways modulate pain in the spinal cord and in the periphery (Fig. 2).

### PATHOPHYSIOLOGY OF PAIN

Although, pain pathways are present at birth, they are often immature and hence compared to adults; young infants have exaggerated reflex responses to pain. Inflammatory mediators cause peripheral and central sensitization after
acute injury. This untreated pain can lead to short term autonomic changes and increased pain perception, chronic pain and exaggerated response with long-term behavioral changes when exposed to subsequent painful stimuli.

**Physiological Response**
- Increased sympathetic tone, ↑ heart rate, ↑ Blood pressure, ↑ Respiratory rate, shallow breath holding respiration, ↓ O₂ saturation, palmar sweating delayed return to GI function
- Immune system—immunosuppression
- Coagulation and haemostasis—hypercoagulable state.

**Hormonal Response**
- ↑ Catecholamines
- ↑ Glucagon
- ↑ Cortisol
- ↑ Aldosterone
- ↑ Corticosteroids
- Sodium and water retention.

**Metabolic Response**
- Hyperglycemia
- Utilization of fat stores/↑ Ketone bodies
- Protein breakdown.

**Psychological Response**
- Fear/helplessness
- Crying spells
- Anxiety
- Sleep disturbance
- New onset nocturnal enuresis
- Others: eating disturbances, mood swings, unexplained crying spells, etc.

**PAIN MANAGEMENT**

Best practice for pediatric pain management is decided by child’s age, developmental skills, associated medical problems, surgical procedure: type and site, expected severity of postoperative pain, predicted recovery course, patient and family needs, desires, technical aspects of interventions and environment. Knowledge of age based anatomic, physiologic and pharmacologic differences help in planning the safe pain management for the child.

**Applied Anatomy, Physiology and Pharmacology**

Infants have larger extracellular and total body water which is responsible for greater volume of distribution for water-soluble drugs. These reduce as child grows. Newborns have smaller skeletal muscle mass and fat stores which decreases the amount of drug bound to muscle and fat resulting in more circulating free drug. As child grows, the muscle mass and body fat increases; which leads to greater binding of drugs to these inactive sites.

Higher cardiac output in infants and children allow rapid equilibrium of drug concentrations to be achieved. This associated with immature blood brain barrier of infancy increases passage of more water-soluble medications, leading to higher central nervous system drug concentrations and more side effects of a drug like morphine at a lower plasma concentration. Also, lower levels and binding capacity of serum albumin and alpha-1 acid glycoprotein in infants up to 6 months of age lead to higher levels of free drug, with greater drug effect and toxicity at lower overall serum levels. Before the age of one year, immature liver and kidney function may be responsible for decreased drug metabolism and excretion. Infants are more sensitive to respiratory depressant effect of opioids because of μ1 to μ2 receptor imbalance and reduced ventilation response to hypoxia/hypercapnea.

The spinal cord and dura mater in the newborn and infant extend to L3 and S3 vertebral level respectively. The line connecting the posterior superior iliac crests, used as a surface landmark during needle insertion, crosses the spinal column at the S1 level in neonates which makes them more vulnerable to injury during needle insertion. The epidural fat below the age of 5 years is loosely...
connected and gel like which makes threading of sacral or lower lumbar catheter to thoracic level very easy. Extensive sympathetic block in child <5 years is not associated with unstable hemodynamics viz ↓ in HR and BP and hence no preloading is necessary in this age group. This is due to smaller venous capacitance in lower extremities with less blood pooling and immature sympathetic system. Larger turnover of cerebrospinal fluid (CSF) is responsible for shorter duration of subarachnoid block and reduced incidence of postdural puncture headache in younger children.

Pediatric regional blocks are usually given under deep sedation or general anesthesia and hence the early central nervous system (CNS) manifestations of LA toxicity may not be apparent or may be misinterpreted. The first sign of toxicity of LA may be dysrrhythmia/cardiovascular collapse. Use of peripheral nerve locator and ultrasound for giving peripheral nerve blocks increase both safety and success. This is responsible for increasing popularity of lower extremity peripheral nerve blocks against central neuraxial blocks.

**PEDIATRIC PAIN MANAGEMENT STRATEGIES**

Pediatric pain management requires team approach which should include pediatric anesthesiologists, child psychologists, child psychiatrist, child life therapists, pediatric pain nurses and pediatric physical therapists. The aim of the team is to have aggressive and proactive pain management in children.

**Preemptive, Preventive and Multimodal Analgesia**

Preemptive analgesia is provided prior to a noxious event in order to prevent or reduce the magnitude and duration of post injury pain and/or the development of chronic pain. Preventive analgesia in addition treats intra and postoperative/injury pain for the same purpose. Multimodal or balanced analgesia targets multiple sites along the pain pathway (Fig. 3) using smaller doses of opioid and nonopioid analgesics to maximize pain control and minimize individual drug-induced adverse side effects. Multimodal pain management also uses nonpharmacologic complementary and alternative medicine therapies. These behavioral techniques which address the emotional component of pain should be applied whenever feasible. Figure 3 depicts multimodal pain therapy which can help in tackling pain at different levels of injury cascade.

**Pharmacological Approaches**

Conventional pain killers include acetaminophen, aspirin, NSAIDs (Table 2), opiates/opioids (Table 3) and local anesthetics as in regional anesthesia. The majority of children with cancer can be made comfortable by using the World Health Organization analgesic “ladder” approach, with weight-based adjustments in dosing.

Unconventional pain killers include antidepressants (tricyclic antidepressants, selective serotonin and norepinephrine reuptake inhibitors), anticonvulsants (gabapentin, pregabalin, carbamazepine, valproic acid, and phenytoin), psychotropic drugs and miscellaneous drugs like magnesium sulfate, muscle relaxants (cyclobenzaprine and baclofen), cannabinoids, capsaicin and oral lidocaine (mexiletine). Not much information is available regarding the prescription of these adjuvant medications, anticonvulsants, and antidepressants for neuropathic pain in children; most prescribing is currently based on extrapolation from adult studies.

**Patient-Controlled Analgesia**

Patient-controlled analgesia (PCA) is being used since late 1980’s in children older than 5 to 6 years of age for acute pain, as well as chronic pain associated with cancer or sickle cell disease. It uses relatively smaller doses of opioids to eliminate the peaks and valleys of analgesia and pain. It results in greater patient satisfaction than with conventional methods as they do not have to ask for pain relief and are “in control.” Younger children can be helped by parent-controlled or nurse-controlled analgesia.
Table 2: Dosing guidelines for commonly used nonopioid analgesics

<table>
<thead>
<tr>
<th>Drug</th>
<th>Oral dose</th>
<th>Per rectal dose</th>
<th>Intravenous dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetaminophen</td>
<td>10–15 mg/kg, 6 hourly, daily dose 75 mg/kg</td>
<td>First dose 35–45 mg/kg then 20 mg/kg 6 hourly</td>
<td>10 mg/kg over 15 mins Maximum daily dose 40 mg/kg in premies, 60 mg/kg in newborn, 75 mg/kg in infants and 100 mg/kg in children</td>
</tr>
<tr>
<td>Ibuprofen</td>
<td>6–10 mg/kg, 6 hourly, Daily dose 40 mg/kg</td>
<td>Not available</td>
<td>Not available</td>
</tr>
<tr>
<td>Diclofenac sodium</td>
<td>1 mg/kg, 8 hourly</td>
<td>2 mg/kg, 8 hourly</td>
<td>1 mg/kg, 8 hourly</td>
</tr>
<tr>
<td>Ketorolac</td>
<td>0.5 mg/kg, 8 hourly, for a maximum of 5 days or 20 doses</td>
<td>Not available</td>
<td>0.5 mg/kg, 8 hourly, for a maximum of 5 days or 20 doses</td>
</tr>
<tr>
<td>Ketamine</td>
<td>5 mg/kg</td>
<td>Not available</td>
<td>IV/IM 0.5 mg/kg                    In regional anesthesia along with local anesthetic 0.5 mg/kg</td>
</tr>
<tr>
<td>Clonidine</td>
<td>2–3 mcg/kg</td>
<td>Not available</td>
<td>0.5–1 mcg/kg IV                    In regional anesthesia along with local anesthetic 0.1–0.5 mcg/kg/hr</td>
</tr>
</tbody>
</table>

Table 3: Dosing guidelines for commonly used opioid analgesics

<table>
<thead>
<tr>
<th>Drug</th>
<th>Oral dose</th>
<th>Intravenous dose</th>
<th>In regional analgesia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Codeine</td>
<td>0.5–1 mg/kg, 4 hourly</td>
<td>Not available</td>
<td>Not available</td>
</tr>
<tr>
<td>Oxycodone</td>
<td>0.1–0.2 mg/kg, 4 hourly or sublingual</td>
<td>0.1–0.2 mg/kg, 6 hourly or intramuscularly</td>
<td>Not available</td>
</tr>
<tr>
<td>Morphine</td>
<td>0.3 mg/kg, 3–4 hourly</td>
<td>Bolus: 0.1 mg/kg, 2–4 hourly</td>
<td>1–5 mcg/kg/hr along with local anesthetic</td>
</tr>
<tr>
<td>Hydromorphone</td>
<td>0.04–0.08 mg/kg, 3–4 hourly</td>
<td>Bolus: 0.02 mg/kg, 2–4 hourly</td>
<td>1–2.5 mcg/kg/hr along with local anesthetic</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>Transmucosal: 10–15 mcg/kg Nasal: 1–2 mcg/kg Transcutaneous patch: 12.5, 25 mcg/hr</td>
<td>Bolus: 0.5–1 mcg/kg, 1–2 hourly Infusion: 0.006 mg/kg/hr</td>
<td>In combination with local anesthetic, 0.25–0.5 mcg/kg/hr</td>
</tr>
<tr>
<td>Pethidine</td>
<td>Not available</td>
<td>0.8–1 mg/kg 3–4 hourly</td>
<td>Not available</td>
</tr>
<tr>
<td>Methadone</td>
<td>0.1–0.2 mg/kg every 12–36 hr</td>
<td>0.1–0.2 mg/kg every 12–36 hr</td>
<td>Not available</td>
</tr>
<tr>
<td>Nalbuphine</td>
<td>Not available</td>
<td>Bolus: 25–50 mcg/kg every 2–4 hr Infusion: 10–15 mcg/kg/hr</td>
<td>Not available</td>
</tr>
<tr>
<td>Tramadol</td>
<td>1–2 mg/kg oral/rectal</td>
<td>1–2 mg/kg bolus 8 hourly</td>
<td>1 mg/kg with local anesthetic</td>
</tr>
</tbody>
</table>

Table 4: Patient Controlled Analgesia Dosing Guidelines

<table>
<thead>
<tr>
<th>Drug</th>
<th>Demand dose</th>
<th>Lockout interval</th>
<th>CBI (mcg/kg/hr)</th>
<th>4 hour limit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morphine</td>
<td>10–20 mcg/kg</td>
<td>8–15 minutes</td>
<td>0–20</td>
<td>250–400 mcg/kg</td>
</tr>
<tr>
<td>Hydromorphone</td>
<td>2–4 mcg/kg</td>
<td>8–15 minutes</td>
<td>0–4</td>
<td>50–80 mcg/kg</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>0.5 mcg/kg</td>
<td>5–10 minutes</td>
<td>0–0.5</td>
<td>7–10 mcg/kg</td>
</tr>
</tbody>
</table>

Patient controlled analgesia (PCA) devices are microprocessor-driven pumps which allow programming of the individual dose of opioid to be administered, the minimal interval between doses (lockout interval), maximum number of doses per hour and the maximal cumulative allowable dose over a 4-hour period. They also allow continuous basal infusion (CBI) in addition to the demand dose (Table 4). All PCA pumps have a locking mechanism, so that neither the settings nor the medication cartridge can be changed without using a key, making the device tamper proof. In IVCA these pumps are connected to child’s existing IV line via Y tubing or can be connected to a separate IV line so that an unintended delivery of a large bolus of opioid is avoided. PCA can also be used with continuous regional blocks like epidural analgesia where it can have combination of opioid and local anesthetic attached to epidural catheter as in PCEA (patient-controlled epidural analgesia).
Regardless of the method of administration, all opioids commonly produce unwanted side effects such as respiratory depression, pruritus, nausea and vomiting, constipation, urinary retention, cognitive impairment, tolerance, and dependence. One should be aware of these side effects and should have strategy in place to take care of them.

**Regional Analgesia**

Regional analgesia (RA) is a use of local anesthetic solution(s) to produce circumscribed areas of loss of sensation (Table 5). It can be central neuraxial technique or peripheral technique and can be either single shot or continuous catheter technique. Regional analgesia is most commonly used in perioperative set-up in pediatric age group. RA offers multiple benefits in pediatric age group as it improves surgical outcome by minimizing autonomic, hormonal, metabolic, immunologic or inflammatory and neurobehavioral responses. It is associated with better operating conditions, reduction in surgical blood loss, profound postoperative analgesia with minimal physiological side effects, alert, calm and cooperative child at the end of surgery. It also minimizes postoperative pain syndromes. Contraindications to RA in pediatric patients are same as that of adults.

Commonly used regional analgesia techniques in pediatric age group can be divided into central neuraxial techniques and peripheral techniques.

**Neuraxial blocks:** Epidural block (caudal, lumbar, thoracic), spinal block, CSE block.

**Peripheral techniques:** Plexus and nerve blocks, Bier’s block, topical surface anesthesia, infiltration, field blocks, intracapsular, transtracheal, intraperitoneal and intrapleural blocks.

Increased use of ultrasound guidance is associated with preference for peripheral nerve blocks wherever applicable. Unlike old times when central neuraxial blocks were the norm for thoracic, abdominal and lower extremity analgesia, now they are limited for thoraco-abdominal surgeries. Caudal epidural block is safe and easy, ‘one technique fits all’ and lumbar epidural block remains gold standard for postoperative pain management in abdominal surgery.20 Peripheral nerve blocks are preferred for their advantages like longer and more localized pain relief, increased safety and success rate, preservation of hemodynamic stability, reduced incidence of PONV, urinary retention, itching, respiratory depression, reduced PACU time and early discharge.21 Patients can be discharged home with continuous peripheral nerve blocks. Overall morbidity is 6 times lower compared to neuraxial blocks.22 Use of adjuvants in pediatric regional analgesia is currently based on extrapolation from adult studies. It is beyond this chapter to describe each regional block in detail, the reader is referred to regional analgesia chapter.

**Nonpharmacological approaches:** It is used in children whenever applicable. Mainly used for chronic pain management23,24 but when used in combination with pharmacologic strategies, it also helps children with acute pain. Breastfeeding25 and administration of oral sucrose26 with and without non-nutritive sucking is frequently used as a nonpharmacological intervention for procedural pain relief of needles or heel pricks in neonates.

- **Behavioral approaches** include distraction27, desensitization, medical staff and parent coaching, modeling, parent training, positive reinforcement and rehearsal.
- **Cognitive approaches**27 include breathing exercises, mental distraction, comforting/reassurance, coping self-statements, hypnosis, imagery, progressive muscle relaxation, providing information/preparation, relaxation training, suggestion, thought-stopping and virtual reality.
- **Complementary approaches** include medical play, therapeutic art, therapeutic play, therapeutic uses of music28 and medical clowns.29
- **Physical approaches** include comfort positioning/iyengar yoga, healing/therapeutic touch, heat/cold therapy, massage, spot-pressure/counter-irritation / acupuncture.30

### Table 5: Local anaesthetics used for Regional anaesthesia

<table>
<thead>
<tr>
<th>Local anesthetic*</th>
<th>Bolus dose, concentration</th>
<th>Infusion dose, concentration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lignocaine+</td>
<td>4–7 mg/kg, 0.5–2%</td>
<td>Not recommended</td>
</tr>
<tr>
<td>Bupivacaine</td>
<td>2–2.5 mg/kg, 0.25–0.5%</td>
<td>0.2–0.4 mg/kg/h, 0.625–0.125%</td>
</tr>
<tr>
<td>Ropivacaine</td>
<td>2–2.5 mg/kg, 0.2–0.5%</td>
<td>0.2–0.5 mg/kg/h, 0.1–0.2%</td>
</tr>
<tr>
<td>Levobupivacaine</td>
<td>2–2.5 mg/kg, 0.25–0.5%</td>
<td>0.2–0.5 mg/kg/h, 0.625–0.125%</td>
</tr>
<tr>
<td>Chloroprocaine</td>
<td>1.5–2 mL/kg, 3%</td>
<td>0.2–0.8 mL/kg/h (neonates), 1–1.5%</td>
</tr>
</tbody>
</table>

* Reduce dose by 30% in infants younger than 6 months of age
+ Higher dose with adrenaline 1: 200,000
CHRONIC PAIN IN CHILDREN

Chronic pain, such as back pain, abdominal pain, headaches, complex regional pain syndrome (CRPS), fibromyalgia, limb pain, chest pain, and joint pain, is common in children and affects their quality of life. Several children with chronic medical conditions like sickle cell disease, cystic fibrosis, epidermolysis bullosa, cancer, require hospital admission for severe pain and acute pain management. All these children require long-term multidisciplinary approach for pain management. Most important part of the chronic pain evaluation process is to look for red flags, which are signs or symptoms that may indicate the presence of a serious illness. Once these serious illnesses are ruled out, these children are mostly benefited by nonpharmacological approaches in addition to conventional and unconventional pain killers. Nerve ablation and destruction are rarely done in pediatric age group as to avoid permanent nerve destruction in a growing child with long life ahead.

FUTURE OF PEDIATRIC PAIN MANAGEMENT

Future of pediatric pain management looks bright because of development and validation of pain assessment tools, advances in developmental neurobiology and pharmacology, knowledge of new analgesics and newer applications of old analgesics, availability of portable ultrasound machine for regional analgesia, infusion pumps for patient/parent/nurse controlled analgesia and structured psychological approach for pain treatment with well-organized pediatric pain services.

SUMMARY

Pediatric pain management depends on anticipation and early recognition of pain in children. Use age, procedure and cognition appropriate pain assessment tools. Record the assessment, treat the pain and reassess whether the treatment given is effective (Fig. 4).

LEARNING POINTS

- Pain should be measured as ‘fifth vital sign’ with age and developmentally appropriate pain assessment tools
- Aggressive and proactive pain management is a must to overcome the historic under treatment of pain in pediatric patients
- Analgesic therapy should depend upon age, weight, comorbidity, and have a multimodal approach
- It is best to avoid intense, single modality treatment. Best results can be achieved by applying number of therapies, each aimed at counteracting the pain in a different way
- Sedative, analgesic, and local anesthetics should form important components of multimodal analgesic regimens
- If analgesic medications are synergistic with sedating agents, then appropriate monitoring must be used during the procedure and recovery
- Behavioral techniques, for addressing the emotional component of pain, should be applied whenever feasible in children
- Interventional procedures, medication management, physical therapy and psychological support are the most important four pillars of the pediatric pain management

REFERENCES

INTRODUCTION

Understanding of respiratory physiology is essential to provide optimal ventilatory strategies in infants and children. The primary goals of ventilation are the maintenance of adequate oxygenation and clearance of carbon dioxide from the body to maintain cellular homeostasis in various clinical scenarios, regardless of the age of the patient. Anesthesia as well as anesthetic equipment like breathing circuits, filters and airway devices may influence decisions regarding appropriate ventilation techniques in children. Laparoscopy and thoracoscopy demand special ventilation strategies, which shall also be discussed.

RESPIRATORY PHYSIOLOGY

We will briefly touch upon the relevant topics in physiology, which are important in understanding the practical application of ventilation strategies.

Resistance and Airflow

For air to move in and out of lungs, gas flows from area of high pressure to one of lower pressure. According to Ohm’s law, the pressure gradient (P) is equal to product of flow rate (V) and the resistance (R) to flow. ($P = V \times R$). The major contribution of resistance is from the frictional resistance of the airways (80%) followed by resistance of lung tissue (20%).

Resistance during laminar flow is calculated using the Hagen-Poiseuille formula:

$$Resistance = \frac{8\eta l}{\pi r^4},$$

where $\eta$ = viscosity, $l$ = length, $r$ = radius.

- Sixteen fold increases in resistance follows a 50% reduction in radius
- When the flow changes from laminar to turbulent, resistance becomes proportional to density rather than viscosity and fifth power of radius (rather than fourth)
- In infants, nasal resistance alone makes up to 30–50% of airflow resistance. It is important to note that occlusion of one nare by insertion of a nasogastric tube in neonates can increase resistance by as much as 50%, and hence earlier consideration of ventilatory support may be needed.
- Resistance caused by laryngeal mask airway (LMA), when measured in a bench study, is less than equivalent endotracheal tube (ETT), presumably because of increased internal diameter of LMA.

Compliance

Compliance is defined as a change in lung volume per unit change in transmural pressure gradient ($C = \Delta V/\Delta P$). It is measured in mL/cm H$_2$O. It has two components—static and dynamic. Static Compliance is measured during the inspiratory pause following delivery of predetermined volume. Dynamic compliance is measured during periods of actual flow. Static compliance is derived from the plateau pressure whereas dynamic compliance from the peak pressure.

During spontaneous ventilation, normal tidal volumes are produced by a transpulmonary pressure of 4–6 cm H$_2$O. However, when positive pressure ventilation is employed, the inflation pressures in most patients are...
approximately twice (8–12 cm H\textsubscript{2}O) because the chest wall has to be passively expanded as well. Chest wall compliance in neonates is high and thus lower pressures are to be expected.\textsuperscript{4}

**Functional Residual Capacity and Closing Capacity**

**Functional residual capacity (FRC)** is defined as the gas remaining in the lungs at end tidal volume breath and acts as a reservoir of oxygen during expiration. FRC in neonates and infants is dynamically maintained by the sustained activity of inspiratory muscles throughout the respiratory cycle, narrowing of glottis during expiration (expiratory laryngeal breaking), and a high respiratory rate relative to expiratory time. These factors in effect create auto-PEEP (Positive End Expiratory Pressure). However, this is lost with anesthesia, intubation and muscle relaxation; hence it is essential to apply PEEP under anesthesia.\textsuperscript{5}

**Closing capacity (CC)** is the volume of gas present when small conducting airways begin to collapse.\textsuperscript{6} As FRC decreases in comparison to CC, airway closure, atelectasis and hypoxemia follow. Closing capacity is higher than FRC in neonates and infants owing to compliant rib cage and low resting volume. CC is higher than FRC in supine children upto the age of 6 years, implying that airway closure is present even before induction of anesthesia.

Anesthesia is further instrumental in the formation of atelectasis as explained by the following mechanisms:\textsuperscript{7-9}

- Airway closure, as CC exceeds FRC (FRC decreases with anesthesia)
- Compression of the lung tissue caused by cephalad displacement of diaphragm in the supine position
- Gas resorption, as increasing the FiO\textsubscript{2} leads to alveolar de-nitrogenation and subsequent loss of alveolar volume.

The major goal should be to prevent and treat atelectasis. Proinflammatory changes in lungs that occur after a major surgery may be limited by minimizing atelectasis with the application of PEEP.\textsuperscript{10} PEEP of 5 cm H\textsubscript{2}O applied early after induction of anesthesia appears adequate to prevent the onset of atelectasis in children.\textsuperscript{11} If 100% oxygen is used, a more aggressive approach like recruitment maneuver may be required along with PEEP.

**Dead Space**

Physiological dead space is the sum of all parts of tidal volume that do not participate in gas exchange. These include anatomical, alveolar and apparatus dead space. Anatomical dead space in an infant (3.3 mL/kg) is higher as compared to adults (2 mL/kg). Apparatus dead space due to connectors, filters and so on further add to this, and this factor must be considered when planning ventilator strategies.

**EQUIPMENT**

Anesthesia equipment acts as the interface between the ventilator and the pediatric airway and hence choice of the various components influences ventilator strategies.

**Laryngeal Mask Airway**

Work of breathing (WOB) was compared in anesthetized children breathing spontaneously under four conditions. WOB was least with an endotracheal tube, higher with an LMA, and face mask with oral airway, and even higher using a facemask without an oral airway. Continuous positive airway pressure (CPAP) reduced WOB in the latter three conditions but not with an endotracheal tube, suggesting that this decrease in WOB is due to decreased supraglottic obstruction rather than any change in lung mechanics.\textsuperscript{12}

In nonparalyzed children managed with ProSeal LMA, WOB was further decreased by 40% using pressure support ventilation of 10 cm H\textsubscript{2}O rather than CPAP.\textsuperscript{13} Positive pressure ventilation with LMA sometimes raises the concern regarding seal of LMA cuff to allow adequate ventilation and prevent gastric distension. ProSeal LMA has consistently been shown to have a more effective seal than classic LMA, with excess seal pressure of about 5 cm H\textsubscript{2}O.\textsuperscript{14, 15}

**Endotracheal Tube**

Uncuffed endotracheal tubes (ETT) have been traditionally used in children up to 8 years of age, irrespective of indication or duration of intubation. Old teaching regarding need for uncuffed ETT in infants and small children is giving way to a more balanced approach as more suitable cuffed ETT are now easily available.\textsuperscript{16}

**Advantages of Cuffed Tubes**

- Less frequent tube changes
- Ability to monitor ventilation (tidal volumes) and carbon dioxide accurately
- Less air leak
- Less OT pollution and less wastage of inhalational agent (more economical despite higher cost of the cuffed tubes).

Use of uncuffed tubes was recommended on the assumption that cricoid is the narrowest portion of pediatric airway; however this is no longer valid.
Recent magnetic resonance imaging (MRI) and video-bronchoscopic studies have revealed that the narrowest portion of pediatric airway is the rimaglottidis and not the cricoid. When using a cuffed ETT, one half size smaller than uncuffed ETT is recommended. Cuffed endotracheal tubes were not popular because of their poor design, however this problem has been circumvented with the use of microcuff high volume low pressure cuffed tubes. It is recommended to monitor the cuff pressure and to limit it 20 cm H2O or below. The authors routinely use cuffed ETT in their practice, even for neonates.

- With uncuffed ETT, pressure controlled ventilation (PCV) is preferable to volume controlled ventilation (VCV), because it compensates better for the inevitable leak which is present with an uncuffed tube.
- When using cuffed ETT, VCV may also be considered with modern anesthesia ventilators, because volume is guaranteed even if lung compliance changes. This is possible only when the ventilator is capable of ‘breathing circuit compliance compensation’. Another important factor is the accurate measurement of the expired tidal volume. For the small tidal volumes in infants and neonates, a flow sensor placed at the Y connector is more accurate.
- When VCV is used, a pressure limit is to be set to protect against barotrauma.

Filter
Airway filters significantly increase the work of breathing associated with their use, mainly owing to the increase in dead space, leading to increased minute ventilation to maintain constant alveolar ventilation. Modern design of filters has ensured extremely low dead space. Neonatal breathing filters with low dead space of about 2–3 mL are now available. Mechanical ventilation offsets the additional burden caused by the use of filters.

BREATHING CIRCUITS
Breathing systems carry gases to and from the machine to the patient. Two main breathing systems used in children are the T piece system (Jackson-Rees modification/Mapleson F system) and the circle system.

T Piece System (JR Circuit)(Fig. 1)
The Jackson–Rees modification of Ayre’s T-piece (JR Circuit/Mapleson F system) incorporates an open-ended bag attached to the end of the reservoir tubing. T piece system is still preferred by some practitioners for induction of anesthesia and transport in children up to 5 years of age or weight less than 20 kg, often without scavenging. This is not because of any advantage of the JR circuit, but lack of awareness and apprehensions of resistance with the circle system.

Advantages
- Less resistance and dead space (no valves and presence of fresh gas flow (FGF) entering the circuit close to the patient)
- Easy assembly.

Disadvantages
- Performance of this system is dependent on the FGF, respiratory rate, tidal volume and duration of expiratory pause. (Rebreathing occurs with low flows and wastage with high flows)
- Scavenging issues and OT pollution
- Requires high flows and hence wastage of inhalation agent
- Cold dry gases delivered to the patient
- In inexperienced hands, bag and mask ventilation with the T piece may be associated with increased gastric insufflations in small children compared to the circle system
- In the absence of pressure relief valve on the expiratory limb, barotrauma and tension pneumothorax may result from over-enthusiastic manual ventilation.

Flows required:
- Spontaneous: 2–3 times minute ventilation (at least 4 L)
- Controlled: 1000 mL +200 mL/kg.

Manual ventilation with T piece may have been the optimal method of ventilation in neonates and infants several years ago, but this practice has changed across the
world. Because of improvements in anesthetic ventilator design which can be used to deliver safer and more accurate tidal volumes compared to hand ventilation, more and more anesthetists are moving away from the T piece circuit, especially since most ventilators are designed for the circle system. Authors no longer use this circuit during general anesthesia.

Circle System (Figs 2 and 3)

Improvement in the performance of circle systems has made them universally applicable for use in children of all ages, including neonates. Owing to improvements in design and the changes in way of practice, the earlier concerns regarding increased dead space and resistance are not as relevant. The circuit, unidirectional valves and the soda lime absorber contribute to the resistance in a typical circle system. The main contribution of resistance is from the unidirectional valves, whereas the pressure decrease across the system at normal adult peak flows is less than 0.75 cm H₂O and for infants it is even less (0.25 cm H₂O). This figure is ten times lower, compared to resistance across an equivalent ETT.

Compression volume of older circuits was also an issue, which amounted to approximately 7–10 cm H₂O and it required the tidal volume to be set in excess of 25 to 125 mL/kg to produce adequate ventilation in neonates while using VCV. Modern pediatric circle systems use light weight 15 mm diameter tubing in comparison to 22 mm adult tubing accounting for less volume loss. The compression volumes as well as resistance of the newer circle systems are no different from the T-piece systems.

![Circle System Diagram](source)


**Figs 3A and B:** Anesthesia circuit—circle system
Advantages of Circle System
- Low flow anesthesia
- Less environmental pollution as well as cost savings
- Improved humidity and heat conservation
- Ability to monitor inspiratory and expiratory gas concentrations
- Almost all new anesthesia machines are designed to be used with circle systems.

Disadvantages of Circle System
As mentioned earlier the notion of increased resistance and decreased compliance is no more true in modern anesthesia work stations.

MODES OF VENTILATION
Various modes of ventilation are available in clinical practice; there is no universally accepted mode of ventilation for all patients. There is no unanimously accepted classification and terminology of different modes. However from clinical perspective, patient’s ventilation and WOB may be controlled or assisted by ventilator.

CONTROLLED MODES
Most common forms of continuous mandatory ventilation are volume controlled ventilation (VCV) and pressure controlled ventilation (PCV) (Fig. 4).

Pressure Controlled Ventilation
In various centers, PCV has become the customary mode of ventilation in pediatric patients.
- Peak inspiratory pressure is preset which is reached early in inspiration and maintained during residual inspiratory phase.
- Tidal volume delivered depends on compliance and resistance of both the circuit and of the patient’s lungs.
- Decelerating flow pattern is observed in this mode of ventilation as compared to constant flow in VCV.

Advantages
- Less Barotrauma
- Better V/Q matching in less compliant lung tissues (decelerating flow pattern)
- Less loss of tidal volume to the circuit (compensates for compression volume and compliance of circuit-added advantage in neonates)
- Delivered tidal volume not influenced by fresh gas flows
- Compensates for inevitable leaks while using uncuffed ETT.

Disadvantages
- Volume delivered is not guaranteed
- Adjustments have to be made in surgeries associated with changes in lung compliance/resistance (e.g. thoracic and laparoscopic surgeries).

Initial Settings
Rate: 30–40 for neonate, 20–30 for infant/small child, 15 for adolescent/child. Peak inspiratory pressure: for neonates 15–22 cm H2O, for children 15–25 cm H2O (can be varied according to lung compliance and pathology to achieve adequate tidal volume of 6–8 mL/kg.)

Inspiratory time: to achieve I:E ratio of 1:2 to 1:3.

Volume Controlled Ventilation
This mode of ventilation is common in adult anesthesia, though less frequently used in pediatric anesthesia.
- Tidal volume is preset in this mode of ventilation, and inspiratory flow rate determines the inspiratory time and the peak inspiratory pressure
- Machine factors (inspiratory flow and time) and patient factors (respiratory rate and compliance) influence the peak inspiratory pressure which is variable
- Constant flow pattern is observed.

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Fig. 4: Pressure, volume and flow tracings during volume controlled and pressure controlled ventilation.

**Advantages**

- Volume delivered is generally guaranteed
- No requirement of adjustments in face of changing lung compliance.

**Disadvantages**

- More chances of barotrauma (in more compliant tissues)
- More loss of tidal volume to circuit (more loss in smaller babies)
- Fresh gas flows can alter peak inspiratory pressures.

In order to achieve a target tidal volume of 6–8 mL/kg, slightly higher tidal volume (8–10 mL/kg) need to be preset owing to losses due to circuit compliance. It is essential to monitor airway pressures and limit peak airway and plateau pressures to less than 35 and 25 cm H₂O respectively by limiting the tidal volume. When using uncuffed ETT, tidal volume delivered to the patient further decreases due to frequent leaks in the system. This will however be readily detected because most ventilators measure both inspired and expired tidal volume.

**Pressure Support Ventilation**

It is the most common type of patient triggered continuous support ventilation available with anesthesia ventilators. Like all support ventilation modes, it requires the patient to trigger the ventilator in order to provide the breath.

- PSV improves gas exchange and decreases WOB when applied correctly in comparison to spontaneous ventilation in both adult and pediatric population.⁷⁻¹⁰
- Flow trigger is associated with greater sensitivity and short trigger delays and is considered a better trigger than pressure in neonates and children. (Flow trigger in adults is set around 1–2 L/min but should be set lower for children with a range of 0.2–0.6 L/min as recently suggested for use in anesthetized patients).¹¹
- Degree of pressure support required depends on the patient’s respiratory drive and desired tidal volume.
- Pressure support required in infants is higher as compared to children to achieve same tidal volume of 6–8 mL/kg, probably because of increased work of breathing as imposed by anesthesia airway equipment in neonates.

**COMBINED MODES OF VENTILATION**

The clear division between controlled and spontaneous ventilation is becoming indistinct because of the increased sophistication afforded by electronics and microprocessors.

**Synchronized Intermittent Mandatory Ventilation**

Synchronized intermittent mandatory ventilation (SIMV) is IMV synchronized with the patient’s inspiratory efforts. For it to work efficiently, a trigger window is created before the next mandatory breath. If patient takes spontaneous breath during trigger window, a mandatory breath is delivered, synchronized with patients effort. This breath is of same tidal volume that would have been delivered by the ventilator if patient had not spontaneously initiated it. Every time a synchronized breath is delivered, machine recalculates the time required to deliver the next mandatory breath. With this mode of ventilation total number of mandatory breaths is equal to preset frequency of breaths despite any patient initiated events. If patient fails to trigger a breath within the allotted time period, the ventilator will deliver a machine breath to the patient.

Spontaneous respiratory efforts outside the trigger window will not result in mandatory breath but will allow the spontaneous breath either to continue unsupported or be assisted by PSV, depending on settings.

- Most appropriate mode of ventilation in neonates and children who are allowed to breathe spontaneously (without use of relaxant) would be PSV with a backup of mandatory pressure regulated breaths (SIMV-pressure controlled). This provides a safety backup of mandatory ventilation in case patient ceases to have spontaneous respiration that might occur with bolus doses of narcotics or induction agent. It also allows for resumption of spontaneous PSV once effects of depressant drugs wear.
- While SIMV with pressure control is always preferable in pediatric practice, volume controlled SIMV can be used in older children.
- The time lag between last spontaneous breath and onset of mandatory ventilation can be set. It is important to decrease this time window in neonates and infants from 30 seconds delay as set for adults.

**Volume Targeted Ventilation**

The fact that volume as well as pressure is involved in the development of ventilator induced lung injury led to growth of this mode of ventilation.
Pressure controlled volume guaranteed (PCV-VG) is one mode offered with some modern anesthesia ventilators. The tidal volume is set and the ventilator delivers the preset volume but using a decelerating flow with the lowest peak inspiratory pressure possible. The ventilator then adjusts the inspiratory pressure required to deliver the preset tidal volume on a breath to breath basis. A maximum pressure is preset, however pressure limit fluctuates between 0 cm H₂O above the PEEP level and 5 cm H₂O below the upper-pressure alarm setting. Inspiratory pressure and time will vary in order to compensate for changes in compliance, resistance and any leak in ETT. With increasing use of cuffed ETT in neonates and infants, it is likely that this mode of ventilation will be increasingly used in operation room settings as it becomes incorporated in newer anesthesia systems.

SPECIAL SITUATIONS

Laparoscopy

Important physiological changes affecting ventilation during laparoscopic surgery are due to increased intra-abdominal pressure, changes in positioning and effects of carbon dioxide insufflations.

- Increase in intra-abdominal pressure which is usually set at 10–12 cm H₂O causes cephalad displacement of diaphragm resulting in decreased FRC and pulmonary compliance. In children, compliance decreases almost by 40% and resistance increases by 20%.
- Trendelenburg position limits diaphragmatic excursion as well as increasing the chances of endobronchial intubation
- Carbon dioxide is commonly used to produce pneumo-peritoneum, and its absorption leads to increase in PaCO₂. Increase in minute ventilation required to maintain normocarbia is variable. Some patients require no increase, but most require a modest increase (20–60%) in MV. Some also require up to 100% increase in minute ventilation.
- With PCV, a significant increase in driving pressure will be required to maintain preinsufflation tidal volume, whereas, while using VCV an increase in peak pressures will be noticed. This increase in inspiratory pressure must be promptly lowered at the end of pneumo-peritoneum to avoid overdistension of the lung
- Increase in minute ventilation if required can be attained with increasing respiratory rate, increasing tidal volume or peak pressures. It is preferable to increase respiratory rate as compared to tidal volume or peak pressures to limit lung injury

- Mild degrees of permissive hypercarbia is generally well-tolerated by the vast majority of patients and pose no significant clinical threat
- The reliability of EtCO₂ to accurately direct ventilation raises some concern as both increase as well as reversal of PaCO₂-EtCO₂ gradient has been reported.
- Intubation with ETT is not always required regardless of the physiological changes described. Proseal LMA may be used with positive pressure ventilation as an alternative to ETT. Proseal LMA size 1.5 and greater have been shown to reliably provide seal with leak pressures >25 cm H₂O.
- PEEP has been shown to be beneficial in adult patients undergoing prolonged laparoscopic surgery in preventing atelectasis and hypoxemia and possibly will be of equivalent benefit in children.

Thoracoscopy

Increasing number of thoracic procedures are being performed thoracoscopically with advances in surgical techniques in neonates and infants (e.g. congenital diaphragmatic hernia, tracheoesophageal fistula repair, decortications of empyema, etc.).

- Insufflation of gas in the pleural cavity tends to decrease the lung volumes, especially the FRC and therefore increases the risk of hypoxemia and atelectasis demanding routine use of PEEP
- Increased inflation pressure is required to maintain tidal volume. However, excessive pressures or volumes should be avoided to prevent barotrauma and inflammation within lung
- Increase in minute ventilation can be achieved by increasing the respiratory rate
- PCV with PEEP may be the optimal mode of ventilation in these patients
- In some situations in which increased pressures are needed, permissive hypercapnea is an option to decrease the risk of barotrauma
- When reinflating the nondependent lung at end of the procedure, it is important to perform recruitment maneuvers and maintain additional PEEP to prevent a recurrence of airway collapse.

Neonates

Management of neonates for minor surgery like open hernia reduction need not entail intubation and controlled ventilation. Spontaneous ventilation with smallest size LMA may be used for short procedures provided the LMA is positioned adequately. If ventilation with LMA is not optimal, an ETT should be substituted.
PSV with low trigger flow and support pressure of 12–15 cm H₂O may be advantageous in decreasing WOB
Dead space minimization may be needed requiring movement of filter from Y connector to expiratory limb of circle system
For major surgery, controlled ventilation with PCV with PEEP is the most popular technique
The increased use of cuffed ETT has made leaks less of an issue, but cuff inflation pressures should be monitored carefully to minimize the risk of subglottic damage.
Adequate humidification is important for neonates undergoing lengthy anesthetic procedures to maintain ETT patency and to enhance mucociliary function.

Magnetic Resonance Imaging
Improvements in MRI compatible equipment have simplified the technique of anesthetizing patients for MRI procedures, allowing anesthetic equipment to be in close proximity to the patient.
- Increased circuit length affects the performance of breathing system
- Some of the concerns are: increased resistance, compression volume and compliance whether a circle, T piece or bain system is used
- With a 5 m length increase in circuit of low compliance (0.5 mL/cm H₂O) and assuming inflation pressure of 16 cm H₂O, the volume loss would be approximately 40 mL, which is a significant amount for a neonate
- With VCV, if system is not compliance compensated, hypoventilation is likely unless the set minute ventilation is increased
- With PCV, an increase in driving pressure is required to compensate for the loss caused by circuit compliance and gas compression
- Adequate monitoring of tidal volume and EtCO₂ is therefore important in this environment
- Maintaining spontaneous ventilation and avoiding need of positive pressure ventilation may be the best choice.

Air Trapping
Asthma, tracheobronchomalacia, bronchiolitis and external airway compression may result in airway trapping. The principles of ventilation are to maintain oxygenation, provide PEEP to splint the airways open, and allow adequate time for expiration in order to prevent dynamic hyperinflation.

Patients with airway collapse may be better breathing spontaneously with pressure support rather than being paralyzed and ventilated
In patients with asthma, controlled ventilation should be set to minimize airway plateau pressure by limiting tidal volume, shortening inspiratory time, and prolonging expiratory time.

LEARNING POINTS
- Narrowest portion of pediatric airway is rima glottidis and not cricoid
- Cuffed ETT are a more cost effective option which provide better airway seal and ventilation
- Newer pediatric circle systems are less compliant and have almost similar compression volume as of Mapleson F and should be used even for induction of anesthesia (with high flows for induction and low flows for maintenance) rather than changing circuits in between
- Application of PEEP early after induction helps prevent atelectasis and hypoxemia in children
- PCV is better than VCV in pediatrics owing to decelerating flow, less barotrauma and more even distribution of flow
- Pressure support required in infants is higher than in children to achieve same tidal volume while using PSV
- Increase in minute ventilation is required during laparoscopy which can be achieved with either increasing respiratory rate, tidal volume (while using VCV) or inflation pressure (with PCV)
- PCV with PEEP and permissive hypercapnia are the ideal ventilator strategies for thoracoscopic procedures

ACKNOWLEDGMENT
We sincerely thank Dr Gitanath, MD, FRCA, for her thorough review of this script. Her suggestions and her critical comments were invaluable.

REFERENCES
Regional Anesthesia in Infants and Children

Chapter 13

INTRODUCTION
Regional anesthesia has wide-ranging benefits. Its use, both as a sole anesthetic and in combination with general anesthesia, continues to increase in neonates, infants and children. The long-term effects of general anesthetics on developing brain and neurocognitive function are a matter of serious concern. Techniques involving regional anesthesia are emerging as an attractive and safe option in this vulnerable population.1, 2

Newer drugs, pediatric specific equipment, nerve localization techniques, and the use of ultrasound guidance have all contributed to the increasing safety of regional anesthesia in children.

REGIONAL ANESTHESIA—BENEFITS3
- Excellent analgesia with minimal side-effects
- Alternative to general anesthesia (GA), when GA is risky, e.g. preterm neonates or premature infants for hernia surgery or other minor procedures; those with neuromuscular, metabolic, cardiac, or chronic lung disease; children at risk of malignant hyperthermia or in emergency situations when patients are at risk of aspiration
- Gastrointestinal function: Early return of gut function, Reduced postoperative nausea and vomiting (PONV); vasodilatation following autonomic blockade improves gut perfusion, e.g. in necrotizing enterocolitis
- Permits reduction in anesthetic depth (MAC), when combined with GA
- Permits early extubation following thoracic and upper abdominal procedures
- Permits early discharge
- Obtunds the neuroendocrine stress response to surgery
- Reduces intraoperative blood loss
- Other uses: Vasodilatation in ischemic limbs, chronic pain
- Cost savings due to reduced ventilation needs, shorter ICU stay and early discharge.

There are several differences between adults and children, which impact the performance of regional blocks. An understanding of these is important for increasing safety and success of regional techniques in children. Table 1 lists the differences in anatomy, physiology, pharmacology and psychology between adults and children pertinent to regional anesthesia procedures.

REGIONAL ANESTHESIA IN CHILDREN—AWAKE OR ANESTHETIZED?
In adults, regional blocks, especially central neuraxial blocks are almost always performed with the patient in the awake state. The awake patient may be better able to appreciate pain, paresthesia and alert the anesthesiologist to possible nerve injury. Quite contrary to the situation in adults, it may be impossible or dangerous to perform
a block in an awake, frightened child because the child can have uncontrolled movements during the block performance. Also, self-report of paresthesias may not be possible in the pediatric age group. Several studies have clearly demonstrated the safety of regional blocks under deep sedation or anesthesia in children.4-7

### FACTORS INFLUENCING THE CHOICE OF TECHNIQUE

These include informed consent, the age and general condition of the patient, and the presence of comorbidities (respiratory, cardiac, neuromuscular, etc.), the severity and site of the pain, the skill of the anesthesia provider, and whether any contraindication to regional anesthesia is present. In making the choice, the anesthesiologist should also take into account the equipment, facilities, and the level of monitoring and nursing care available.

### CONTRAINDICATIONS TO REGIONAL ANESTHESIA

Parental consent and patient assent, where appropriate is a must before performing any regional technique. Absolute contraindications to central neuraxial blocks include coagulopathy, meningitis with increased intracranial pressure, infection at the needle insertion site, and true local anesthetic allergy. Abnormal anatomy or landmarks, e.g. lumbosacral myelomeningocele may preclude performance of central neuraxial block. Progressive
neurologic disease is a relative contraindication primarily because of medicolegal concerns. Contraindications to peripheral blocks are fewer; these include local infection, generalized sepsis, coagulopathy and risk of compartment syndrome.

**COMPLICATIONS OF REGIONAL BLOCKS**

Complications are rare, as reported in several large studies. The incidence seems to be more frequent in children < 6 months and 6 times more common with central neuraxial blocks as compared to peripheral blocks. In general, a peripheral block is safer than a central neuraxial block. However, performing a regional block may result in different complications, most of which can be avoided by learning the correct technique, using appropriate equipment, and applying the basic safety rules.

**Complications of Central Blocks**

- Traumatic—nerve damage, hematoma, paraplegia.
- Related to medium used to identify epidural space—dilution and increase in the injected volume of LA if saline is used and headache, patchy anesthesia, lumbar compression, multiradicular syndrome, subcutaneous cervical emphysema, or embolism if air is used.
- Infective—Meningitis, arachnoiditis, epidural abscess, radiculopathies, discitis, vertebral osteitis.
- Total spinal anesthesia—due to inadvertent dural puncture and injection of large volume of epidural local anesthetic into intrathecal space.
- High level of block—due to large volumes of local anesthetic, head low position immediately following drug injection.
- Postdural puncture headache (PDPH)—has been reported in children as in adults. Catheter related complications—malposition, kinking, knotting, breakage (especially if attempts are made to withdraw the catheter through the epidural needle). Migration into the subarachnoid space, accidental removal, leakage around the puncture point especially with smaller catheters, inadvertent removal, catheter related infection.

**Complications of Peripheral Blocks**

- Related to technique—innervation of nerves, vessels, hematoma, pneumothorax (interscalene block), respiratory paralysis due to epidural/spinal diffusion of LA (interscalene block, lumbar plexus block), diaphragmatic paralysis (interscalene block).
- All these can be minimized by use of ultrasound guidance, and with appropriate training.
- Catheter related—these are fewer than with central catheters, and include kinking, knotting, inadvertent removal, blockage and infection.
- LA toxicity—this has been dealt with in Section II of this chapter.

**ADJUVANTS TO LOCAL ANESTHETICS**

Local anesthetics, even the long acting ones, e.g. Bupivacaine, Ropivacaine have a limited duration of action (4-8 hour). This is a limitation especially with the single shot caudal block. Adjuvants are commonly used to prolong the duration of action and to augment analgesia, while allowing lower concentrations of LA to be used.

**Epinephrine**

Epinephrine 1:200,000 (5 μg/mL) has been used as a caudal adjuvant for many years. It prolongs the duration of caudal block by reducing the systemic absorption of LA. This prolongation is greatest in children less than 5 years of age and its effect on the duration of analgesia decreases with increasing age. Its effect is more pronounced with Lignocaine, compared to Bupivacaine or Ropivacaine. This may be due to high lipid solubility of Bupivacaine and Ropivacaine, which causes them to be deposited in epidural fat and released slowly, combined with the relatively short duration of action of epinephrine. The effect of epinephrine on the duration of action of epidural bupivacaine also depends on the concentration of bupivacaine used; marked prolongation is seen with 0.125% concentration.

More importantly, addition of epinephrine to epidural solutions may help early detection of inadvertent intravascular injection.

**Opioids**

Opioids are commonly used in epidural blocks, with or without LA. Injection of opioids into the epidural space enables provision of analgesia without the sympathetic or motor block associated with LA. This analgesic effect is attributable to a local action of the opioid at the spinal cord level rather than to an effect after systemic absorption. There are 2 distinct classes of opioids—hydrophilic and lipophilic. Hydrophilic opioids, e.g. morphine are capable of rostral spread, and consequently associated with a greater incidence of sedation and respiratory
depression. Lipophilic opioids, e.g. fentanyl remain more localized to the site of injection.

Morphine has been used in the dose range of 30–100 μg/kg, in several studies. Morphine produces significant prolongation of analgesia, with a mean duration of 20–24 hours in some studies. A dose of 30 μg/kg has been recommended in children as the optimal dose, enhancing analgesia while reducing risk of side-effects, especially respiratory depression. The higher dosage may be used in children undergoing cardiac surgery.

The synergistic effect with local anesthetics has also been demonstrated with other opioids e.g. Fentanyl, in the dose of 1 μg/kg. However, the disadvantage of lipophilic opioids is their shorter duration of action compared to morphine. Indeed, several studies demonstrate no significant prolongation of analgesia with fentanyl added to caudal LA in children.

Buprenorphine is a partial agonist with a very high affinity for the μ-opioid receptors in the spinal cord, and has a potency 25–50 times that of morphine. Following inguinal surgery, caudal buprenorphine, in dose of 4 μg/kg prolonged analgesia by 5 hour 40 minutes longer than with morphine 0.05 mg/kg. Combination of buprenorphine 2.5 μg/kg and bupivacaine 0.5% provided analgesia for up to 24 hours after lower-body surgery without any serious complications.

Respiratory depression is a risk with neuraxial opioids; it is higher with patients less than 1 year of age. Delayed respiratory depression is reported with morphine (mean-time 3.8 hours, up to 12 hours), resulting from rostral spread of opioid to the brainstem with subsequent depression of the medullary respiratory centers. In contrast, there are no reports of delayed respiratory depression with fentanyl or buprenorphine. These highly lipophilic drugs are rapidly cleared from the cerebrospinal fluid, thereby making rostral spread less likely.

Neuraxial opioids are, therefore, best avoided in ambulatory surgical patients. Patients who receive neuraxial opioids should be closely monitored postoperatively. Other side-effects include pruritus, nausea and vomiting, and urinary retention.

**Clonidine**

Clonidine is an α₂-adrenoceptor agonist, and produces analgesia by direct stimulation of pre- and postsynaptic α₂-adrenoceptors in the dorsal horn gray matter of the spinal cord, thereby inhibiting the release of nociceptive neurotransmitters.

Clonidine, in the dose of 1–2 μg/kg, has been shown to prolong duration of caudal Bupivacaine. The duration of analgesia achieved by the addition of clonidine to bupivacaine varied widely (5.8–16.5 hours) in these studies. This may be the result of a number of factors—dose of clonidine used; differences in premedication and volatile anesthetic used; type of surgery; indications for rescue analgesia, assessment of pain, and statistical analysis. Similar prolongation of analgesia has also been reported when clonidine was added to Ropivacaine in caudal block.

Side-effects of epidurally administered clonidine are hypotension, bradycardia and sedation, and they are dose dependent; seen with doses of 5 μg/kg. Life-threatening apnea has been reported with its use in neonates, hence clonidine is avoided in neonates and small infants.

**Ketamine**

Ketamine, a phencyclidine derivative, exerts its effects mainly via NMDA receptor blockade. In addition, it also binds to the opioid receptors. It not only produces analgesia after systemic administration, but also exerts a profound analgesic effect when administered in central neuraxial block. When added to LA in doses ranging from 0.25–1 mg/kg, it has been shown to prolong analgesia from 8–16 hour. The higher dose is associated with behavioral side-effects, hence the optimum recommended dose is 0.5 mg/kg.

There is a concern regarding neurotoxicity of ketamine, which may be related to the preservative (benzethonium chloride). It is recommended that only preservative free ketamine should be administered in neuraxial block. Preservative-free S (+)-ketamine has twice the analgesic potency as its racemate and may be promising as an alternative to racemic ketamine.

**OTHER ADDITIVES**

**Midazolam**

Epidural midazolam exerts its analgesic effect through the GABA-benzodiazepine system in the spinal cord. Midazolam in the dose of 50 μg/kg, added to Bupivacaine has been shown to prolong duration of analgesia, with minimal postoperative sedation.

**Tramadol**

Tramadol is a synthetic analogue of codeine. It has a moderate affinity for opioid receptors, and inhibits serotonin and norepinephrine receptors. Caudal tramadol has been used in the dose of 2 mg/kg in children. However, its efficacy seems to be doubtful, the effect
may be due to its systemic absorption and subsequent analgesic action.\textsuperscript{37}

**INDIVIDUAL BLOCKS**

The subsequent text describes central neuraxial blocks in detail, followed by abdominal nerve blocks and certain other commonly performed nerve blocks. For peripheral nerve blocks of the upper and lower limb, the reader is referred to the chapters on orthopedic surgery and ultrasound guided regional blocks.

**CAUDAL BLOCK**

The “single-shot” caudal block is the commonest central block performed in children, as it is technically simple, easy to learn and has a good safety record. It is used to provide intraoperative and postoperative analgesia for surgery below the umbilicus. These include orchidopexy, circumcision, inguinal herniotomy, lower limb and pelvic orthopedic surgery and lower abdominal surgery in neonates and infants where low thoracic dermatomes may be blocked. It has been used as the sole technique for inguinal hernia repair in prematures at risk of postoperative apnea. Contraindications include anatomical anomalies of the sacrum, local infection or a bleeding diathesis.

**Anatomy**

The sacral hiatus results from the failure of fusion of the posterior arches of the fourth, fifth and occasionally, the third sacral vertebrae. This deficiency in the neural arch is covered by a ligamentous membrane known as the sacrococcygeal membrane. The landmarks for this hiatus are the sacral cornua superiorly and the coccyx inferiorly. Posterior fusion of the sacrum becomes more complete with age and in children beyond 8 years, the membrane is usually ossified.

**Technique**

The block is performed with the patient in the left lateral position with the upper hip flexed more than the lower. Strict aseptic precautions must be followed, as it is a potentially contaminated area. The posterior superior iliac spines and the sacral hiatus form the edges of an equilateral triangle (Fig. 1). Epidural puncture is achieved at the apex of the sacral hiatus with the needle inclined 45–60° to the skin. The palpating finger of the left hand lies on the spinous process of S3. In neonates and small infants, this angle can be dropped even further to prevent the needle traversing the epidural space and hitting the vertebral body (Fig. 2). In chubby infants, where the landmarks are obscure, the sacral hiatus can be located by first palpating the coccyx, then sliding the palpating finger cephalad until a depression in the skin is felt. On piercing the sacrococcygeal membrane, a distinct “pop” or give is appreciated. The needle is then advanced 1–3 mm on a plane parallel to the spinal axis. After aspiration to rule out dural puncture or a bloody tap, the local anesthetic solution is injected in aliquots.

**Equipment**

A variety of needles and cannulae have been used to perform caudal blocks. Hypodermic needles are still being used; they are easily available and inexpensive. However, there is a theoretical risk of introducing epidermal cells into the epidural space. The extension tubing in the butterfly needle or scalp vein (Fig. 3) makes injection easier. Specially designed caudal needles with stylet and...
Methods to Confirm Localization of Caudal Space

- “Give” or “pop” of the advancing needle
- Free flow of LA solution, without resistance
- Lack of subcutaneous swelling after injection
- Swoosh or Whooosh test—auscultation over the lumbar spine for an audible sound, following injection of saline or air
- Heart rate reduction by 3 or more beats per minute as a result of stimulation of pressure receptors in the epidural space
- Electrostimulation—if the stimulating needle is in the caudal space, anal sphincter activity will be visible with a stimulation current of 1 to 10 mA
- Ultrasound—to identify the sacral hiatus and to monitor the cephalad spread of LA solution
- Laxity of anal sphincter—signifies a “working” caudal block
- In neonates and small infants, immediate relaxation of the lower extremities occurs after a successful block.

Drug Dosing—Volume of LA

- 0.5 mL/kg: Sacral block
- 1 mL/kg: Thoracolumbar block
- 1.25 mL/kg: Mid-thoracic block
- (Doses described by Armitage).

However, the 1.25 mL/kg dose leads to excessive rostral spread and 0.75–1 mL/kg suffices for the majority of procedures.

Takasaki’s formula:
Volume of drug = 0.056 mL/kg/dermatome to be blocked.

Maximum recommended volume is not to exceed 20 mL. Bupivacaine in the concentration of 0.125–0.25%, up to a maximum dose of 2.5 mg/kg is the commonest local anesthetic used. Ropivacaine and Levobupivacaine have a better safety profile, with less unwanted motor block in the postoperative period, and are emerging as attractive alternatives to Bupivacaine. A limitation of the single shot caudal block is the short duration of action. Adjuvants are almost always added to prolong the duration of the caudal block; the reader is referred to the section on “Adjuvants to local anesthetics”.

Complications

When performed by experienced anesthetists, caudal blocks are remarkable safe. Also, the block is very easy to learn; high success rates have been reported after a limited period of training.

- Venous puncture—incidence is higher with sharp beveled hypodermic needles, hence short beveled, blunt needles are recommended.
- Subperiosteal/intraosseous injection into the marrow cavity of the sacrum—the result would be similar to intravenous injection of LA.
- Dural tap—the needle should not be advanced more than a few millimeters after the ‘give’ of the ligament is appreciated. Negative aspiration for cerebrospinal fluid is possible despite a dural puncture; hence the needle should be left open to atmosphere after entering the space before injection of LA when the flow of cerebrospinal fluid can be ruled out.
- Total spinal block—a rare but serious complication of caudal epidural blockade caused by inadvertent subarachnoid injection of a significant volume of local anesthetic. The risk is increased due to the variable distance between the sacrococcygeal membrane and the lower extremity of the dural sac.
- Motor block—Ropivacaine is associated with less motor block than Bupivacaine, and is an attractive option in ambulatory surgeries. In children undergoing hypospadias repair, caudal block with a “high volume, low concentration” regimen produces prolonged analgesia and less motor block, compared to a “low volume, high concentration” regimen.
- Others—rectal penetration, urinary retention.

LEARNING POINT

Inadvertent dural puncture is possible during performance of a caudal block if the block needle is advanced too far into the caudal epidural space.
LUMBAR EPIDURAL BLOCK

Indications
The common indications are major abdominal surgery, open thoracic surgery, spinal surgery and for long-term pain management. All other surgeries can be better managed with specific techniques such as paravertebral block, transabdominal plexus blocks, rectus sheath blocks and lower limb blocks.

Anatomy
At birth, the vertebral spinous processes are parallel to each other, at all levels, due to the spine having one single concavity (kyphosis). The lumbar lordosis develops later, when the child starts walking. Hence, the orientation of epidural needles is perpendicular to the back for all approaches. The epidural space in infants and small children contains fat that is fluid in nature, permitting easy passage of epidural catheters from the lumbar and caudal approaches to a higher level. The line joining the iliac crests, the Tuffier’s line is a good marker for performance of epidural blocks. In infants, it crosses the L5-S1 vertebra as compared to L4-L5 vertebral level in older children and adults. Blocks below this line are considered safe, as the spinal cord terminates at a higher level. In children, the elasticity of the ligaments provides greater mobility to the vertebral column. In the lateral position, flexion moves the spinal cord forward and expands the epidural space. This position is preferred for epidural blocks in children. The lumbar epidural space may be entered via a midline or a paramedian approach. The midline approach is used most widely, because the ligamentum flavum is at its widest in the midline, and the risk of inadvertent epidural vessel puncture is low.

Sites of Action of LA
- Dorsal and ventral spinal roots within their dural sleeves
- Dorsal roots-spinal ganglion
- Periphery of spinal cord after diffusion into subarachnoid space and CSF
- Spinal nerves, after the LA passes into paravertebral and perineural spaces through intervertebral foramina.

Equipment
Currently, there are three sizes of Tuohy needles suitable for children: 18 G (standard 10 cm); 19 G and 20 G (short 5 cm) for infants and children < 10 kg. Shorter needles are easier to handle and the smaller bevel is more appropriate for the narrow epidural space (Fig. 4).

Technique
The technique of lumbar epidural block is essentially similar to adults. Usually, the midline approach is used, since the space is wider here, and the risk of vessel puncture is lower. The child is placed in the lateral position after administration of anesthesia.

Loss of resistance (LOR) technique is used to determine entry into the epidural space. The medium to be used (air or saline), is a matter of debate. Loss of resistance to air (LORA) has been reported to be associated with significant complications, such as nerve root compression, pneumocephalus, a greater incidence of incomplete analgesia, paresthesia, and venous air embolism. Loss of resistance to normal saline (LORNS) has been advocated by experts, but there are some problems. In direct contrast to adult patients in whom the continuous pressure of the saline technique pushes the dura away and so reduces the risk of dural puncture, the incidence of dural puncture in pediatric patients has been reported to be greater with LORNS. Also, air may permit easier detection of a dural tap compared to saline. Despite the potential for serious complications, it has been suggested that LORA is safer than saline for the identification of the epidural space in children less than 2 years of age. The amount of air in the syringe should be limited to a maximum of 1-2 mL and used only to detect the change of resistance, releasing the pressure on the plunger immediately upon entry into the epidural space. A combination of air and saline has been suggested as the best technique, because this allows improved feeling.
while reducing the risk of injecting air when the epidural space is entered.  

*Depth from the skin to the epidural space can be determined as follows:*

- Neonate ≈ 1 cm
- Children 10 kg–25 kg ≈ 1 mm/kg
- Children > 25 kg: 0.8 + (0.05 × weight [kg]) = depth in cm

After locating the space, the LOR syringe is disconnected from the epidural needle and there should be no reflux of CSF or blood. The local anesthetic solution is injected either as a “single shot” block or a catheter is threaded into the epidural space. Catheters can frequently be threaded from the lumbar to the thoracic level with the Tuohy bevel directed cephalad. If catheters will be threaded to the thoracic level, the distance must be measured prior to insertion. An epidural catheter should never be withdrawn with the epidural needle in place; this can shear the catheter. If there is difficulty in threading a catheter, the entire assembly (the epidural needle with the catheter) should be removed and the procedure attempted again.

The Meniscus test can be performed as an additional test to confirm correct epidural catheter placement. This test involves three steps: (i) Following negative aspiration for blood or CSF, saline is injected into the epidural catheter through the filter. The filter is then removed, and the open end of the epidural catheter is lifted and the liquid meniscus present in the catheter is observed to drop rapidly; (ii) the open end of the epidural catheter is lowered and the liquid meniscus is again observed to fill the catheter with clear liquid and no blood; (iii) the presence of air in the catheter during backflow confirms the correct position in the epidural space as opposed to a position in the subarachnoid space.  

Ultrasonography can be used to judge the skin epidural distance, to locate the position of epidural catheter, and to confirm the spread of local anesthetic in the epidural space.

**Test Dose**

Early symptoms of systemic local anesthetic toxicity cannot be detected in children because the majority of blocks are performed in children under general anesthesia or heavy sedation. Negative aspiration of blood is performed to rule out intravascular placement of needle or catheter, but this has low sensitivity. A small dose of epinephrine (0.5 μg/kg) in 0.1 mL/kg volume of lignocaine is injected as test dose. An increase in heart rate by 10 beats per minute after injection is suggestive of intravascular injection; however, this response varies with the type of anesthetics used.  

T-wave amplitude or ST segment changes are more sensitive means of identifying inadvertent systemic injection. Additionally, development of nodal or sinus Bradycardia is an uncommon but specific sign of systemic injection. The recommendation is to inject LA in small fractions, while observing the ECG for ST-T changes, in addition to heart rate. The indicators of inadvertent systemic injection may be delayed for up to 60–90 seconds after the fractional test dose, hence a period of 90 seconds observation is recommended after test dose, prior to injecting the bolus dose. Even after a negative test dose, the remaining volume of local anesthetic should be administered in incremental volumes of 0.1–0.2 mL/kg.

**Approach to the Epidural Space**

The intervertebral level from where to approach the epidural space depends both on the age of the patient and the experience of the anesthesiologist. Table 2 lists the recommendations for approaching the epidural space.

**Drug Dosing**

Volume of injectate depends on the level needed for surgery. General estimate of dosing for caudal or epidural injections is 0.5–1 mL/kg bolus, maximum 20 mL. 0.1 mL/year of age is needed to block a dermatome. A weight-based regimen for lumbar epidural block; volume of injectate = weight × 0.04 mL/dermatome. Table 3 lists the standard dosing regimen for epidural block.

**Complications**

Increased incidence of epidural catheter migration in smaller children has been observed. The cause could be related to decreased subcutaneous tissue leading to less friction/gripping on the catheter. Also, catheters in smaller children leak more frequently, thereby affecting the integrity of fixation.

**Table 2: Recommendations for approach to the epidural space**

<table>
<thead>
<tr>
<th>Surgery</th>
<th>Technique</th>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Surgery below umbilicus</td>
<td>Single shot technique</td>
<td>Caudal approach in infants and children &lt; 8 years. Lumbar approach in older children</td>
</tr>
<tr>
<td>Catheter technique</td>
<td>Lumbar approach (Caudal catheters are associated with possibility of contamination)</td>
<td></td>
</tr>
<tr>
<td>Surgery above umbilicus</td>
<td>Catheter technique</td>
<td>Experienced anesthesiologist — Thoracic approach; with catheter</td>
</tr>
<tr>
<td>(Thoracic/upper abdominal)</td>
<td>Less experienced anesthesiologist — Lumbar or caudal (in neonates and infants) approach; with catheter</td>
<td></td>
</tr>
</tbody>
</table>
Chapter 13: Regional Anesthesia in Infants and Children

### SPINAL BLOCK

Spinal anesthesia is an easy and effective technique; a small amount of local anesthetic injected in the lumbar cerebrospinal fluid provides highly effective anesthesia, analgesia, and motor block in the lower part of the body.

#### Indications

- Surgery on the lower part of the body—lower abdominal, inguinal, urologic, lower limb procedures
- Surgical procedures in high risk, preterm neonates, as a sole technique, e.g. inguinal hernia
- Children with recent URTI to avoid GA and airway instrumentation
- Child with difficult airway
- Child with a “full stomach”
- Associated comorbidities: Pulmonary, neuromuscular disease, MH susceptibility
- Others: Cardiac surgery, to suppress the stress response, resource poor centers.

#### Technique

The puncture is performed with the child in lateral position with the lower extremities flexed and the neck extended. Alternately, it can also be performed with the child seated with support. Careful attention must be paid to avoid excessive neck flexion in young infants, which causes airway obstruction. After sterile preparation, a short (1.5–2 inch) 25 or 26-gauge spinal needle should be introduced caudal to L3 to avoid possible damage to the spinal cord. After the local anesthetic has been injected, the stylet should be reinserted, and the needle may be left in the position for a few seconds to prevent the drug from tracking back into the tissues and site of skin puncture.63

#### Drug Dosing

Table 4 lists the local anesthetic dosages for spinal in infants and children. One of the major limitations of single-injection spinal anesthesia is the relatively short duration of action. Different classes of adjuvants have been combined with spinal anesthetics to modify the onset, intensity, and duration of spinal block. Table 5 lists the commonly used adjuvants.64

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### Table 3: Dosing regimen for epidural block

<table>
<thead>
<tr>
<th>Drug</th>
<th>Initial dose</th>
<th>Infusion</th>
<th>Repeat doses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bupivacaine/L Levobupivacaine</td>
<td>Concentration: 0.25% with 1:2 lakh epinephrine &lt; 20 kg: 0.75 mL/kg 20–40 kg: 0.1 mL/year/dermatome OR 8–10 mL &gt; 40 kg: Same as adults</td>
<td>&lt; 4 months: 0.2 mg/kg/h (0.15 mL/kg/h of 0.125% solution or 0.3 mL/kg/h of 0.0625% solution) 4–18 months: 0.25 mg/kg/h (0.2 mL/kg/h of 0.125% solution or 0.4 mL/kg/h of 0.0625% solution) &gt; 18 months: 0.3–0.375 mg/kg/h (0.3 mL/kg/h of 0.125% solution or 0.6 mL/kg/h of 0.0625% solution)</td>
<td>0.1–0.3 mL/kg 6–12 hourly Concentration: 0.125–0.2%</td>
</tr>
<tr>
<td>Ropivacaine</td>
<td>Concentration: 0.2% Dose similar to Bupivacaine</td>
<td>Concentration: 0.1–0.2% Doses similar to Bupivacaine</td>
<td>0.1–0.3 mL/kg 6–12 hourly Concentration: 0.1–0.2%</td>
</tr>
<tr>
<td>Adjuvants</td>
<td>Fentanyl: 1–2 μg/kg Clonidine: 1–2 μg/kg Avoided in neonates &amp; small infants</td>
<td>Fentanyl: 1–2 μg/mL Clonidine: 1 μg/mL Morphine: 10 μg/mL</td>
<td>Morphine: 25–30 μg/kg 8 hourly</td>
</tr>
</tbody>
</table>

### Table 4: Local anesthetic dosages for spinal in infants and children

<table>
<thead>
<tr>
<th>Type of LA</th>
<th>&lt; 5 kg</th>
<th>5–15 kg</th>
<th>&gt; 15 kg</th>
<th>Duration of action (mean)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.5% Bupivacaine/ Levobupivacaine</td>
<td>0.5–0.6 mg/kg (0.1–0.12 mL/kg)</td>
<td>0.4 mg/kg (0.08 mL/kg)</td>
<td>0.3 mg/kg (0.06 mL/kg)</td>
<td>80 minutes</td>
</tr>
<tr>
<td>0.2% Ropivacaine</td>
<td>0.5–1 mg/kg (0.1–0.2 mL/kg)</td>
<td>0.5 mg/kg (0.1 mL/kg)</td>
<td>0.3–0.4 mg/kg (0.1 mL/kg)</td>
<td>96 minutes</td>
</tr>
</tbody>
</table>

### Table 5: Adjuvants in spinal block

<table>
<thead>
<tr>
<th>Adjuvant</th>
<th>Dose</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epinephrine</td>
<td>2–5 μg/kg</td>
<td>Prolongs effect by 50%</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>0.2–1 μg/kg</td>
<td>Higher doses have been used in cardiac surgery</td>
</tr>
<tr>
<td>Clonidine</td>
<td>1–2 μg/kg</td>
<td>Higher doses associated with hypotension, bradycardia and respiratory depression</td>
</tr>
</tbody>
</table>
Possible Complications

The most common complication related to spinal puncture in children is postdural puncture headache (PDPH). The reported incidence ranges from 4% to 15%. Similar to adults, needle design and size affects the incidence of PDPH, with smaller, pencil point spinal needles associated with a lower incidence of PDPH. Other complications include backache (5–10%) and transient neurological symptoms (3–4% with Bupivacaine).

LEARNING POINTS

• Do not lift the child’s legs in the air after the block or a high thoracic block will occur
• Although the local anesthetic dose may appear large, recall that children have a large cerebrospinal fluid volume relative to adults
• The duration of the block increases with the patient’s age

ABDOMINAL WALL BLOCKS

Abdominal wall blocks include the ilioinguinal/iliohypogastric nerve block, the rectus sheath block, and the transversus abdominis plane (TAP) block.

ILIOINGUINAL /ILIOHYPOGASTRIC NERVE BLOCK

Ilioinguinal/iliohypogastric nerve blocks provide ipsilateral analgesia in the inguinal area.

Indications

Analgesia for hernia repair, orchiopexy, hydrocele surgery, as a supplement to general anesthesia.

Technique

A short beveled 22 G needle is inserted just below and medial to the anterior superior iliac spine (ASIS). The medial distance from the ASIS depends on the size of the child, varying from 0.5 cm in the infant to 2 cm in the adolescent. The needle is slowly advanced, until a “pop” of the external oblique aponeurosis is felt. LA is injected in a fan shape subcutaneously and immediately deep to the external oblique aponeurosis. A volume of 0.4–0.5 mL/kg of 0.5% Bupivacaine is used. The landmark technique, however, results only in 14% of injections being made in the correct anatomical location.

Ultrasoundographic-guided ilioinguinal/iliohypogastric nerve block has been proven to be considerably more effective than the traditional landmark-based technique. Using real-time imaging, the exact position of the needle tip between the ilioinguinal and iliohypogastric nerves within the correct fascial plane is possible. In addition, ultrasonographic guidance enables the visualization of the spread of local anesthetic around the targeted nerves, and allows significant reduction in the volume of local anesthetic needed.

Complications

Bowel puncture, failure of block.

RECTUS SHEATH BLOCK

Rectus sheath blocks provide efficient pain relief for umbilical or other midline surgical incisions.

Indications

Analgesia for umbilical and epigastric hernia repair, single port laparoscopic surgery, pyloromyotomy and other small midline incisions.

Technique

This blocks the terminal branches of 9th, 10th, and 11th intercostal nerves. Courreges and Poddevin suggest a subcutaneous fan-shaped injection around the umbilicus in combination with a second injection, which is made immediately after the rectus sheath has been pierced. The correct needle position is identified by a fascial click triggered when the needle pierces the anterior rectus sheath. Alternatively, the local anesthetic is injected deep to the rectus muscle within a potential space between the rectus abdominal muscle and posterior aspect of the rectus sheath (Fig. 5).

Use of ultrasound is strongly recommended to avoid complications and improve success rates. The linear probe is positioned just below the umbilicus. The anterior and posterior aspects of the rectus sheath and the enclosed rectus abdominis muscle are visualized. A short bevel needle is inserted in an in-plane approach, and the
needle tip is directed inside the rectus sheath near the posterior aspect of the rectus muscle. 0.1 mL/kg of local anesthetic is sufficient to produce analgesia.

Complications
Block failure, puncture of bowel, mesenteric vessels.

TRANSVERSUS ABDOMINIS PLANE (TAP) BLOCK
This blocks the segmental nerves T9, T10, T11, T12, and L1 within the plane between the transverse abdominal and the internal oblique abdominal muscle, and provides analgesia of the abdominal wall (Fig. 6).73,74

Technique
The lumbar triangle of Petit (a space bounded by the iliac crest, latissimus dorsi muscle, and external oblique muscle) is used as a landmark, and a two-‘pop’ sensation indicates the correct needle position. The first ‘pop’ occurs after penetration of the fascia of the external oblique muscle, and the second ‘pop’ occurs after penetration of the internal oblique muscle. 0.3–0.4 mL/kg of LA is injected on either side. Use of ultrasound is recommended to improve success rates, and reduce complications. With the ultrasound probe in the midaxillary line between the iliac crest and the costal margin, the block is performed in an in-plane technique.75

Complications
Bowel and vessel puncture, liver trauma,76,77 failure of block.

OTHER NERVE BLOCKS
Penile Nerve Block
The penis is innervated by the pudendal nerve (S2-S4). This nerve divides into the right and left dorsal nerves of the penis that pass under the pubic symphysis to travel just below the Buck’s fascia to supply the sensory innervation to the penis. This is a simple block to learn and is associated with a low incidence of complications.78

Indications
Phimosis, paraphimosis, circumcision, distal penile hypospadias repair.

Technique
The anatomical landmark is the pubic symphysis (Fig. 7) The penis is gently pulled downward and 2 puncture sites are identified at 10O’ clock and 2 O’clock positions, at the base of the penis, immediately below the pubic symphysis. The needle is introduced perpendicular to the skin, and advanced until loss of resistance is felt to suggest that the tip of the needle is within the Buck’s fascia. The procedure is repeated on the opposite side. Depth of insertion ranges from 0.5 cm for a neonate to 3 cm in an adolescent. 0.1 mL/kg of LA (0.25% Bupivacaine without epinephrine) is injected on each side, after negative aspiration for blood. Midline approach to the penile block is better avoided, due to the risk of injuring the dorsal artery of penis.79 Epinephrine is contraindicated, because the dorsal artery of penis is an end artery, and injury can cause ischemia and necrosis.

Ultrasound guidance can be used to improve success rates. The probe is placed along the shaft of the penis, along the sagittal plane. The subpubic space is visualized as a triangle, with the pubic symphysis superiorly and the deep penile fascia inferiorly.

PARAVERTEBRAL BLOCK
Paravertebral block provides unilateral analgesia at specific dermatomes. The advantages are localized pain control, and requirement of small volumes of LA. It has been used in children undergoing thoracotomy, renal surgery, cholecystectomy and inguinal surgery.80,82
The paravertebral space is a wedge-shaped area alongside the vertebral column, containing the intercostal nerve, its dorsal ramus, the rami communicantes, and the sympathetic chain. It is bounded anteriorly by the parietal pleura, and posteriorly by the superior costotransverse ligament (Fig. 8). Free communication between adjacent spaces allows LA to spread to several dermatomes with a single injection.

**Abbreviation:** Oes: Esophagus; Ao: Aorta; Td: Thoracic duct; Az: Azygous vein; Bd: Vertebral body; Tp: Transverse process; Sp: spinous process

**Technique**

The child is positioned lateral, with the block side up. The spinous process of the level to be blocked is identified. The point of needle insertion from midline is approximately equal to the distance between 2 spinous processes. The needle (short spinal needle or Tuohy needle if catheter is planned) is inserted perpendicular to skin. It will contact the transverse process, following
which it is walked over its cephalad margin. Loss of resistance to saline is felt once the costotransverse ligament is pierced. A catheter can be threaded 2–3 cm into the space if continuous analgesia is desired. Compared with epidural catheterization, slightly more force is required to thread a paravertebral catheter. A bolus dose of 0.5 mL/kg of LA blocks four dermatomes. For continuous infusions, a volume of 0.2–0.25 mL/kg/h provides reliable analgesia.

**Some Useful Formulae**

- Point of needle insertion (distance from spinous process in mm) = Weight (kg) × 0.12 + 10.2
- Depth of paravertebral space from skin (mm) = Weight (kg) × 0.48 + 18.7

**Complications**

These include vascular and pleural puncture, pneumothorax and block failure. Hypotension is uncommon, so is the risk of dural puncture.

**INFRAORBITAL NERVE BLOCK**

The infraorbital branch of the trigeminal nerve exits the infraorbital foramen and supplies the sensory supply to the upper lip, tip of the nose and the maxillary process. A formula for location of infraorbital foramen is:

\[
\text{Distance from midline} = 21 \text{ mm} + 0.5 \times \text{age (in years)}
\]

**Indications**

Cleft lip repair, nasal septum surgery.

**Technique**

The patient is positioned supine, with a shoulder roll for extension. There are two approaches described:

**Intraoral Approach**

The upper lip is everted, and a 26G hypodermic needle is inserted at the root of the canine tooth and directed toward the infraorbital foramen. 0.5–1 mL of local anesthetic is deposited at the level of the infraorbital foramen where the infraorbital nerve exists.

**Extraoral Approach**

The needle is introduced at the point of bisection of lines drawn through center of pupil and the alae nasi.

**Complications**

Hematoma formation, persistent paresthesia of the upper lip, intravascular injection.

**GREATER PALATINE NERVE BLOCK**

The greater palatine nerve is a branch of the infraorbital division of the trigeminal nerve as it comes off the pterygopalatine ganglion.

**Indication:** Cleft palate surgery

**Technique**

The patient is placed supine, with the head extended on a shoulder roll. The Dingman’s mouth gag is inserted by the surgeon to open the mouth, and visualize the palate. The greater palatine foramen is located lateral to the midline, just anterior to the junction of the soft and hard palate. In patients with dentition, the nerve is located just medial to the second molar. LA (1–1.5 mL) of 0.25% Bupivacaine is injected using a 26G needle, anterior to the greater palatine foramen.

**Complications**

Intravascular and intraneural injection.

**SAFETY RULES FOR PERFORMING REGIONAL ANESTHESIA**

All blocks should be conducted with intravenous access and with standard monitoring applied. The electrocardiogram should be adjusted so that the P wave, QRS complex, and upright T wave is clearly visible. Baseline systolic blood pressure and heart rates should be noted. Appropriate resuscitation equipment should be available.

**Skin Preparation**

Bacterial colonization of epidural and caudal catheters in children is common; more common with caudal catheters and in small children. However, there are no reports of systemic infection following catheter placement for postoperative analgesia. Insertion of an epidural catheter should be performed under strict aseptic conditions with a daily observation of exit site. Before any block, meticulous skin preparation is mandatory. Both 0.5% chlorhexidine and povidone iodine in alcohol are effective antiseptic agents. Chlorhexidine has a faster onset and longer duration of action than povidone iodine, and it retains its efficacy in the presence of blood. It also has a
lower incidence of skin reactions than povidone iodine, and therefore may be preferred over iodine. Both agents should be kept well away from the drugs and equipment to be used for the block, to prevent contamination and the possibility of neurotoxicity. The solution must be allowed to dry before the skin is palpated or punctured.

Optimum aseptic technique requires the use of barrier precautions. These include thorough hand washing with surgical scrub solution, the wearing of a cap, mask, sterile gown and gloves and the use of a large sterile drape.

Injection technique—(for all pediatric regional anesthesia except the subarachnoid block)

- Aspirate prior to injection
- Inject test dose of local anesthetic solution containing 0.5 μg/kg of epinephrine
- Look for signs of positive test dose
- Inject the remaining volume of local anesthetic slowly (120–180 seconds)
- Aspirate every 3 to 5 mL
- Continue to closely monitor electrocardiograph and blood pressure during injection
- Carefully test dose any catheter prior to bolusing or starting a continuous infusion
- Direct visualization of the location of the needle tip and the injectate with ultrasound may provide additional confirmation of lack of intravascular injection.

ROLE OF ULTRASOUND

A most promising recent advance is the use of ultrasound for performance of regional blocks in children. Real-time ultrasound guidance allows the demonstration of the target, whether it is a nerve, fascial plane, or anatomical space, and allows monitoring of the distribution of the injected local anesthetic. In doing so, use of ultrasound may increase the accuracy, success rate of regional blocks and reduce the time needed for achieving the block.

Ultrasound Imaging and Central Neuraxial Blocks

The margin of safety for central neuraxial block is narrow in children due to a number of factors. The block is almost always performed under deep sedation or anesthesia, preventing the report of paresthesia or pain. The anatomical structures are closely positioned, and the epidural space can be very narrow. The spinal and epidural space are at a variable depth from the skin, across age groups. The soft tissues and ligaments may hinder appreciation of loss of resistance to the advancing needle. Ultrasound could be great value in the young pediatric population where there is limited ossification, thus allowing good visual resolution of the anatomy and block-related equipment or solutions.

Ultrasound imaging before neuraxial blockade is useful for determining the angle and depth from the skin to the epidural space. This may be particularly helpful in situations where landmarks may be difficult to palpate such as in obese children.

In neonates and infants, ultrasound visualization of the ligamentum flavum, dura and the spinal cord may enable more rostral puncture points than those often recommended for this age group. This could allow higher success with epidural catheters, because of decreased incidence of catheter coiling during cranial advancement.

In caudal blocks, ultrasound imaging helps the anesthesiologist to appreciate the position of the sacral hiatus and the dural sac, view the needle as it pierces the sacrococcygeal membrane and view the spread of a test dose, either directly or as it displaces the posterior dura mater in an anterior direction.

Ultrasound Imaging and Peripheral Nerve Blocks

Ultrasound visualization of the nerve plexus and the advancing needle, allows for deposition of LA very close to the nerves to be blocked. This allows for reduction in LA needed to produce effective limb blocks. Also, visualization of nearby structures (vessels, pleura) reduces the incidence of complications.

In performing truncal nerve blocks (TAP, rectus sheath block, ilioinguinal nerve block), ultrasound visibility of the musculature, related fascia and aponeuroses, and visualization of LA spread may improve the success rates and allow for administration of minimal volumes of local anesthetic.

ROLE OF NEUROSTIMULATION

Neurostimulation has been used for nerve localization in children for several years, and has been proven to have high degree of safety and success rates. With the advent of ultrasound, should we continue to use neurostimulation?

Neurostimulation may reveal information on proximity to other nerves, e.g. phrenic nerve stimulation in supraclavicular block, and alert the anesthesiologist to intrafascicular injection when motor response is elicited at very low current strength (< 0.2 mA). The consensus is that both neurostimulation and ultrasound offer...
complementary evidence of needle to nerve proximity, and increase success rates, while reducing the time needed to achieve the block.101-103 These dual modalities should be used, whenever feasible when performing regional blocks.

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77. Schuepfer G, John M. Generating a learning curve for penile block in neonates, infants and children: An empirical evaluation of technical
INTRODUCTION

Well-placed local anesthetics (LAs) given in appropriate doses can yield great clinical benefits. But exceeding the dose resulting in increased blood level of LA can be devastating. Prevention is the most important element in avoiding morbidity and mortality associated with local anesthetic systemic toxicity (LAST). Hence, we need to know pharmacokinetics of LAs, factors influencing toxicity of LAs, clinical features of toxicity, prevention and management.

All local anesthetic drugs are broadly divided into two groups:

1. Amino esters, e.g. procaine, 2-chlorprocaine, tetracaine. These are degraded by plasma pseudocholinesterases and nonspecific esterases.
2. Amino amides, e.g. bupivacaine, lidocaine, ropivacaine. They are more stable molecules and undergo hepatic metabolism.

At the physiological plasma pH of 7.40, about 60–85% of amide molecules and more than 90% of ester molecules are in ionized form resulting in free diffusion in the body’s aqueous compartment.

Mechanism of action of LA is mainly to block sodium channels or by preventing sodium channels from opening, thereby interrupting the propagation of nerve impulses. However, this action is not only localized to sodium channels of nerve tissue, but will have its effect on any tissue containing sodium channels. So inadvertently injected LA into blood vessel will block sodium channels in other tissues such as heart and brain and serious complications may ensue if toxic levels are reached. LAs at higher concentration are also potent potassium, and to a lesser extent calcium channel blockers. Hence it cannot be excluded that K⁺ blockade may add its arrhythmogenic effects and Ca⁺⁺ blockade resulting in its negative inotropic effects to those of Na⁺ blockade during bupivacaine toxicity. High concentrations of lidocaine seem to displace bupivacaine from its binding sites. Similarly, phenytoin (anticonvulsant) has been advocated in the treatment of bupivacaine toxicity as it may displace bupivacaine from its binding sites. LAs impair mitochondrial metabolism. Mitochondrial respiration impairment may play role in the mechanism of LA toxicity. Bupivacaine has been found to uncouple oxidative phosphorylation which has been blamed for its toxicity.

FACTORS INFLUENCING TOXICITY OF LOCAL ANESTHESIA

1. Age of the child—In neonates high hematocrit values and larger size of erythrocytes (physiologic macrocytosis) result in consistent entrapment of LAs, thus lowering Cmax values after single injection but increasing secondary release, thus increasing half-life of all LAs. In infants physiologic anemia reduces red cell storage of LAs and its protective effect against toxicity (after single shot injection) when plasma protein sites are saturated. As neonates do not have normal levels of hepatic enzyme P-450 for metabolism of amide LAs, they have longer plasma half-life and greater risk of toxicity. In older infants and young children, the liver mass as a percentage of body weight is relatively large and contains more metabolic sites for breakdown of LAs resulting in rapid elimination of LAs. In neonates plasma protein concentrations of α acid glycoprotein and albumin for binding of LAs are quite low resulting in more unbound free fraction in the blood, predisposing them to LA toxicity. As bupivacaine has high affinity to protein, it should be used cautiously in the premature and young infants. Prilocaine should be avoided in infants as its biotransformation converts it to 6-hydroxytoluidine resulting in severe methemoglobinemia. Aminoesters are rapidly hydrolyzed by plasma cholinesterases. This enzyme values are low at birth and gradually reach adult levels by 1 year of age. Changing percentage of body fluid compartments in the growing child has influence on pharmacokinetics of LAs. The distribution volume of LAs is markedly increased in the very young, resulting in lower peak plasma concentration following the injection of single dose. However, this creates a danger of drug accumulation in case of repeated injections or continuous infusion. Children have higher cardiac output (2–3 times that of adult), a factor causing rapid increase of LAs in blood especially in the vessel rich groups such as brain and heart.

2. Additives to LA—Vasoactive agents such as epinephrine when added to LA are effective in slowing down the systemic uptake. As with epinephrine, peak plasma concentration of lidocaine is significantly reduced with addition of clonidine in the dose of 1–3 μg/kg. If maximum allowable dose of one LA has
been reached, another LA should not be administered, as the toxicity of mixture of LA is additive.10

3. **General anesthesia**—Pediatric anesthesiologists prefer to sedate or give general anesthesia depending upon surgical procedure before performing any regional blocks, as awake children may not cooperate for regional techniques. General anesthesia doubles the blood concentration at which toxic signs appear for lidocaine.11 Midazolam and diazepam have protective influence against toxicity of LA.12

4. **Type of LA**—LAs are short, moderate or long-acting aminosteres or aminoamides. Commonly used lidocaine is moderate-acting amide and bupivacaine and ropivacaine are long-acting amides. The standard formulation of bupivacaine is a racemic mixture of S and R-enantiomer, while levobupivacaine has only S-enantiomer. Levobupivacaine and ropivacaine have intrinsic vasoconstrictor properties, which prolong the systemic absorption and duration of action. So they are safer than standard bupivacaine. Recently, lidocaine has been shown to be toxic when administered intrathecaclly. Preservatives, (e.g. metabisulffites) epinephrine, antioxidants increase the potential for direct neural toxicity as well as for allergy.13

5. **Dose of LA**—Subcutaneous or intramuscular injection may result in failure of block, but there can be danger of LA toxicity if another block is performed without taking into consideration the amount of LA already injected. Inadvertent dural puncture with subsequent intrathecal injection of LA with epidural dose results in total spinal anesthesia showing clinical picture of LA toxicity.14 Injection of large volume of LA may result in excessive spread, which can reach distant nerves, or too high levels of epidural/spinal anesthesia. This may result in respiratory failure due to intercostal muscles or even diaphragmatic paralysis. Lumbar plexus block, intercostal nerve or brachial plexus block, especially interscalene approach may also lead to similar complications. Malfunction of infusion pumps may deliver high dose of LA resulting into LA toxicity.15 Secondary migration of epidural catheter into subarachnoid space or even blood vessel will result in high dose delivery of LA and toxicity. Repeated injections of highly concentrated LA can result in local myotoxicity.16 Whenever lidocaine spray or aqueous jelly are chosen for topical anesthesia, care should be taken to use calculated dose and volume of LA. Prolonged seizures have been reported with viscous lidocaine.17

6. **Faulty technique**—Injection of LA with high speed in the epidural space quickly displaces CSF and may result in intense headache, loss of consciousness, intracranial hypertension and coma.18 Slower injection reduces the peak plasma LA concentration. It allows earlier detection of intravascular needle placement. Injection of LA given without frequent aspiration test may end up in intravascular placement of LA resulting in toxicity.

7. **Site of block**—Some sites have higher risk of direct intravascular injection, such as interscalene or stellate ganglion block while some sites have higher risk of rapid absorption as those are highly vascularized such as scalp, face, bronchial mucosa and pleura. The order of sites in which lowest to highest chances for absorption and toxicity are: subcutaneous tissue injection, brachial plexus, epidural, caudal and finally intercostals blocks and topical anesthesia.19

8. **Physiological status, comorbidities**—When children are mechanically ventilated, often resulting in respiratory alkalosis, the toxic threshold of LAs is increased. Elevated PaCO2 enhances cerebral blood flow and LA is delivered more rapidly to brain. The risk of toxicity is increased by hypothermia, hypoxia, hypercarbia, acidosis and hyperkalemia.20 Children with severe renal impairment have reduced clearance of LAs. It is recommended that the initial dose be reduced by 10–20% and continuous infusion dose should be adjusted with caution. In patients with liver disease single dose blocks are unaffected but doses for repeat boluses and continuous infusions should be reduced on account of reduced clearance. Increased bilirubin levels in neonates can increase the chances of toxicity by competition at plasma protein binding sites. Patients with cardiac failure are susceptible to LA induced myocardial depression and arrhythmias. In these patients because of lower liver and renal perfusion resulting in slower metabolism and elimination of LAs, initial and maintenance doses of LA should be correspondingly lower. In children with congenital cardiac defect having right-to-left cardiac shunts LA systemic toxicity is increased. Cimetidine and propranolol reduce hepatic blood flow, thus increasing the chances of LAST.

### TOXIC PLASMA LEVELS OF LOCAL ANESTHETICS

Toxic plasma levels of LAs are not clearly established. Commonly used doses of LAs such as 6–8 mg/kg of lidocaine and 1.5–2 mg/kg of bupivacaine lead to peak plasma concentration ranging from 3–5 μg/mL for lidocaine and 0.5–1 μg/mL for bupivacaine.21 Clinical signs of toxicity have been reported with plasma concentration ranging from 7–10 μg/mL for lidocaine and 1.5–2.5 μg/mL.
with bupivacaine. The toxic level of free unbound form of bupivacaine is believed to be around 0.2 μg/mL. Neonates when given lidocaine, manifest symptoms of neurologic toxicity at blood levels as low as 2.5 μg/mL, which is half of that seen in adults. Based on clearance of bupivacaine, plasma concentration should remain below 2.5 μg/mL and recommended level is less than 2μg/mL in children (Table 6).

**CLINICAL PICTURE OF TOXICITY**

**Local toxicity**—Local adverse events result from direct nerve damage due to incorrect placement of needle. Local toxicity includes myotoxicity, i.e. skeletal muscle damage, neurotoxicity because of intraneural injection, i.e. cytotoxicity and transient neurological symptoms. LA induced myotoxicity may involve actions on mitochondria. Single shot spinal anesthesia with usual doses and concentrations of LA such as lidocaine and mepivacaine can produce more limited and transient neurologic symptoms such as backache, radicular pain, paresthesia or hypoesthesia. Toxicity of LA may be seen as localized allergic reaction due to aminoester metabolite e.g. para-aminobenzoic acid or preservatives used in LAs. LA solution with epinephrine when used in areas supplied by terminal arteries can result in tissue ischemia and gangrene.

**Local Anesthetic Systemic Toxicity (LAST)**

This comprises mainly of neurological (CNS) and cardiovascular (CVS) system features. The presenting features of LAST vary widely.

**CNS toxicity**—This can be described as two-stage process. Initial excitatory phase followed by a depressive phase. Early premonitory characteristics signs are confusion, agitation, light-headedness, perioral tingling, slurred speech, tinnitus, visual and auditory disturbances followed by tremors, muscular twitching of face and extremities. This excitatory phase culminates into generalized convulsions of tonic and clonic nature and later leading into depressive stage of respiratory arrest and coma. Early excitatory phase is masked in children as often they are given sedation or general anesthesia before carrying out regional or peripheral blocks. CNS excitation may be the result of an initial blockade of inhibitory pathways in the cerebral cortex. There is correlation between potency of LA and CNS toxicity.

**CVS toxicity**—LAs can exert direct actions on both, the heart and peripheral blood vessels. There is decrease in the rate of depolarization in the fast conducting tissues of Purkinje fibers and ventricular muscle. They also exert indirect actions on the circulation by blocking sympathetic and parasympathetic efferent activity. CVS toxicity is seen classically in three phases:

1. **Initial phase**—tachycardia and hypertension.
2. **Intermediate phase**—myocardial depression and hypotension.
3. **Terminal phase**—severe peripheral dilation and hypotension, arrhythmias such as severe bradycardia, QRS widening, conduction blocks, ventricular tachyarrhythmias culminating into asystole.

The infants of prilocaine toxicity resulting into methemoglobinemia will become cyanotic when methemoglobin level exceeds 20–30% of total hemoglobin. The clinical features are dyspnea, tachycardia, hypoxia, vertigo and rarely proceed to death unless level exceeds 70%.

**LEARNING POINTS TO REDUCE RISK OF TOXICITY**

- Avoid overdosing—consider age, weight and physiological status
- Test dose of LA with epinephrine—even small dose—1–2 μg/kg of epinephrine 1:200,000 solution with 0.25%bupivacaine produces peaked T-waves with ST segment elevation on ECG, particularly in lateral chest leads, indicating intravascular injection
- Repeated aspiration to note needle placement
- Slow injection watching ECG tracing
- EMLA cream to be used only on intact skin, and avoided in infants less than 3 months, and patients taking sulfonamides
- Usage of proper equipment—short bevel needles; ultrasound-guided regional blocks for precise

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**Table 6:** Usual and maximum doses of LAs in children

<table>
<thead>
<tr>
<th>Local anesthetic</th>
<th>Usual concentration (%)</th>
<th>Usual doses (mg/kg)</th>
<th>Maximum dose with epinephrine (mg/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Aminoesters</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Procaine</td>
<td>1–2</td>
<td>7 mg/kg</td>
<td>10 mg/kg</td>
</tr>
<tr>
<td>Chloroprocaine</td>
<td>2–3</td>
<td>7 mg/kg</td>
<td>10 mg/kg</td>
</tr>
<tr>
<td><strong>Aminoamides</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lidocaine</td>
<td>0.5–2</td>
<td>5 mg/kg</td>
<td>10 mg/kg</td>
</tr>
<tr>
<td>Prilocaine</td>
<td>0.5–1.5</td>
<td>5 mg/kg</td>
<td>10 mg/kg</td>
</tr>
<tr>
<td>Bupivacaine</td>
<td>0.25–0.5</td>
<td>2–2.5 mg/kg</td>
<td>3 mg/kg</td>
</tr>
<tr>
<td>Ropivacaine</td>
<td>0.2–1</td>
<td>3–4 mg/kg</td>
<td>Not needed</td>
</tr>
<tr>
<td>Mepivacaine</td>
<td>0.5–1.5</td>
<td>5–7 mg/kg</td>
<td>10 mg/kg</td>
</tr>
<tr>
<td>Etidocaine</td>
<td>1</td>
<td>3–5 mg/kg</td>
<td>7 mg/kg</td>
</tr>
<tr>
<td>Levobupivacaine</td>
<td>0.25–0.5</td>
<td>2–3 mg/kg</td>
<td>3 mg/kg</td>
</tr>
</tbody>
</table>
location of the site intended to be blocked, which reveals spread of the LA for optimal dose to reduce the risk.29

TREATMENT FOR LAST

Immediate treatment is required whenever any early signs of toxicity such as twitching, tachycardia, and hypertension are observed. Injection of LA must be stopped and immediately oxygen supplementation and further treatment carried out depending upon the signs of toxicity. It is important to get additional help and continue vigilant monitoring.

Emergency Treatment of Seizures

1. Airway management: 100% Oxygen supplementation with mask ventilation
2. If seizures persist:
   - IV midazolam in the dose of 0.05 mg/kg, or IV diazepam 0.1–0.2 mg/kg
   - IV thiopentone 4 mg/kg prior to giving relaxant, or
   - IV phenytoin 5 mg/kg slowly over 10 minutes.
   - If mask ventilation is possible—IV atracurium 0.5 mg/kg
   - If mask ventilation is difficult succinylcholine 1.5–2 mg/kg for faster action
   - Tracheal intubation and adequate ventilation to prevent acidosis.

Emergency Treatment of Cardiac Complications

1. Initial treatment for all CVS complications:
   - 100% oxygen with face mask ventilation followed by tracheal intubation
   - If required external cardiac massage to restore hemodynamics
   - Sodium bicarbonate administration to prevent metabolic acidosis—dosage 2 mg/kg over 10 minutes with 4.2% sodium bicarbonate in central vein or 6 mg/kg/min with 1.4% sodium bicarbonate in the peripheral vein.
2. If hypotension persists, hemodynamics not restored:
   - IV Atropine—0.02 mg/kg (maximum 0.6 mg)
   - IV inotropic drugs—Epinephrine: 0.1 mL/kg of 1/10000 solution, dopamine or dobutamine: 2–10 μg/kg/minute or rarely required isoprenaline: 0.1 μg/kg
   - IV Calcium chloride: 10–30 mg/kg.
3. If pulseless ventricular tachycardia or fibrillation develops, defibrillation and the ACLS cardiac arrest algorithm is to be followed.

With the resuscitation measures if hemodynamics recover and coronary perfusion gets well established, bupivacaine wash out from heart speeds up to facilitate recovery from toxicity. Recent developments in treatment of LAST includes the use of intralipid emulsion (ILE). The first two reports of ILE use in human to reverse LA induced cardiac arrest were published in July and August 2006. In both cases ILE restored cardiac output and full recovery without neurological deficit.30,31

Recommended doses of ILE:

- 20% ILE 1.5 mL/kg over 1 minute, followed by IV infusion of 20% ILE at 10–15 mL/kg/h
- If no response after 5 minutes, repeat 2 boluses of 20% ILE 1.5 mL/kg slowly with interval of 5 minutes
- If no recovery, continue infusion of ILE at 30 mL/kg/h
- Do not exceed maximum cumulative dose of 12 mL/kg.

In infants of prilocaine toxicity resulting into methemoglobinemia, the treatment consists of IV injection of methylene blue 1–5 mg/kg. It converts methemoglobin to hemoglobin instantaneously.

CONCLUSION

The events of LA toxicity are generally unpredictable but many are preventable with adequate precautions. If early signs of LAST are seen, immediate institution of appropriate treatment can overcome this emergency and facilitate full recovery.

REFERENCES

INTRODUCTION
Bleeding disorders can pose an additional challenge in the intricate field of pediatric anesthesia. The knowledge and its application, of blood and its components are fast evolving. Knowledge of this field of transfusion medicine can provide the anesthesiologist with a better insight in diagnosing, monitoring and managing patients with bleeding disorders during the perioperative period.

An anesthesiologist may come across a bleeding disorder in different clinical backgrounds.

A. During a routine pre-surgical evaluation
An apparently healthy patient may be diagnosed to have a bleeding disorder during routine pre-surgical evaluation.

For the diagnosis of bleeding disorder in an apparently healthy pediatric patient, the following information may be helpful:

Personal history
- Excessive bleeding after cord cutting, circumcision, minor trauma
- Need for transfusion
- Prolonged antibiotic treatment predisposing to vitamin K deficiency
- Warfarin treatment.

Family history
- Excessive bleeding in family members after minor trauma/surgery
- Menorrhagia in female relatives
- Oral anticonvulsant therapy in mother during pregnancy predisposing the neonate to vitamin K deficiency.

Examination findings
- Petechiae and purpura on extremities and mucosa suggest quantitative or qualitative platelet disorder
- Raised petechiae suggest vascular type of bleeding
- Mucosal bleeds, e.g. epistaxis indicate platelet abnormalities
- Bruises, joint bleed, muscle bleed suggest coagulation factor deficiencies, e.g. hemophilia.

B. A diagnosed case of bleeding disorder may need to be prepared pre-surgically to withstand the hemostatic challenges of the surgery. Evaluation of the following aspects, supplement the therapeutic management of the patient.

i. Platelet abnormality:
   a. Is it quantitative or qualitative? In qualitative abnormalities, platelet transfusions may be needed even with normal platelet counts.
   b. Is there an associated fever/splenomegaly? Response to platelet transfusions is inadequate in the presence of fever, splenomegaly.
   c. Is there an active bleeding? Platelet trigger is higher in the presence of active bleeding.

ii. Severity of the disease, e.g. Hemophilia can be mild, moderate or severe.

iii. Presence of inhibitors to coagulation factor, e.g. inhibitors to Factor VIII in a case of hemophilia A.

iv. Response to pharmacological agents, e.g. some cases of von Willebrand’s disease respond to DDAVP reducing the need for transfusion.
v. Affordability and availability:
Recombinant factors are the safest with reference to infectious disease transmission. Also bedside leukoreduction filters, though at an additional cost, aid in achieving safety and efficacy of transfusion therapy.

Factor concentrates prepared by fractionation and pathogen inactivation of human plasma are safer and more efficacious, but costlier than the cryoprecipitate and fresh frozen plasma (FFP).

vi. Level of medical expertise and facilities provided by the blood bank.

C. **The anesthesiologist may be faced with an unanticipated hemostatic catastrophe**, e.g. disseminated intravascular coagulation (DIC) or a dilutional coagulopathy associated with massive transfusion of stored blood. In such a condition, clinical assessment of bleeding is critical in early diagnosis and helps the investigational findings in deciding the further management. Clinical presentation of acute DIC is with bleeding from surgical or venipuncture sites, bruising and purpura.

**TRANSFUSION THERAPY**

Transfusion therapy comprises of blood, its components, and the plasma derivatives. It is supplanted by the recombinant products and pharmacological agents. Transfusion trigger varies depending on whether the transfusion is for prophylaxis or for therapeutic management of the deficiency. It is vital that there must be clear medical justification for every blood transfusion.

The benefits of each transfusion must be weighed against the potential infectious, immunologic and metabolic risks. Overuse of transfusion should be avoided. It is required to monitor for thromboembolism when patient is on multiple therapies, e.g. Factor VIIa, cryoprecipitate, antifibrinolytic agent.

Blood sample of the patient is sent to the blood bank for ordering blood components. Red blood cells need to be grouped and cross-matched. For neonatal transfusion, plasma, platelets and cryoprecipitate should be group specific. Plasma can be either frozen and used as fresh frozen plasma or it can be fractionated into plasma derivatives. Transfusion trigger is defined as the level of the blood constituent below which most patients require transfusion. Group O red blood cells and Group AB plasma can be transfused to all ABO groups. Aspirin, NSAIDs should be avoided in patients with low platelet counts.

Special needs during transfusions in pediatric patients:
Infants younger than 4 months of age, preterm and low-birth weight infants require special consideration. This is due to their immature hemostatic systems, limited ability to tolerate thermal and metabolic alterations from transfusion, and susceptibility to iatrogenic anemia with frequent blood sampling.

**BLOOD AND BLOOD COMPONENTS**

**Red Cells**

*Available as*
- Whole blood
- Red cell concentrate
- Red cells resuspended in additive solution/saline/albumin/thawed FFP
- Washed red blood cells.

**Transfusion Trigger**

a. Hemoglobin <8 g/dL or hematocrit <24%

b. Hemoglobin <10 g/dL or hematocrit <30% in patients with pulmonary disease, cyanotic heart disease, preterm infants.

*Dose:* 10 mL/kg of whole blood or red cell concentrate administered over 2 to 4 hours raises the hemoglobin by 1–2 g/dL or hematocrit by 3 to 6%.

Children older than four months of age who have good cardiac and vascular function, requiring red cell transfusion, should be transfused with 10 to 20 mL/kg.

**POINTS TO REMEMBER**

- During a routine red cell transfusion, donor blood should be gently agitated every 15 minutes to prevent red cell sedimentation in the bag
- During acute hemorrhage, hematocrit and hemoglobin levels may underestimate the blood loss
- Rapid transfusion or transfusion greater than one blood volume in 24 hours needs special care, as this can lead to hyperkalemia, hypothermia, citrate toxicity with hypocalcemia

**Platelets**

Thrombocytopenia in neonates is defined as platelet count less than 150 \( \times 10^9 /L \), similar to that in adults. A platelet count of 100 \( \times 10^9 /L \) or lesser, must be evaluated.

**Platelet Trigger**

- Platelet count <10,000/µL and decreased platelet production, without other risk factors for bleeding
- Platelet count <50,000/µL with active bleeding or planned procedure in patient with decreased platelet production
- Platelet count <1,000,000/µL with hemorrhage, DIC, or other coagulation disorder
• Bleeding with qualitative platelet defect at any platelet count.

**Administration:** A 170 micron filter should be used for administration of platelets to prevent the infusion of microaggregates and fibrin clots. Platelets should be administered over 30 to 60 minutes.

**Dose of platelet suspension:** 10 mL/kg or one whole-blood derived platelet unit per 10 kg of body weight in older children.

**Response:** Increase in the platelet count by 40 to 60 × 10^9/L.

Platelet refractoriness is defined as consistent inability to increase platelet counts following platelet transfusion and can be due to alloimmunization or other causes like fever, splenomegaly, etc.

**Contraindications**

**Absolute:** Thrombotic thrombocytopenic purpura, heparin-induced thrombocytopenia.

**Relative:** Uremia, immune thrombocytopenic purpura. Platelet transfusion should be given only for excessive bleeding and significant thrombocytopenia.

**Fresh Frozen Plasma**

Fresh frozen plasma contains all the coagulation factors, the inhibitors, e.g. antithrombin III, protein C, protein S, and fibrinolytic enzymes.

**Indications**

- Broad-spectrum replacement of both coagulation factors and inhibitors, e.g. in DIC
- Multiple coagulation factor deficiency, e.g. liver disease, hemorrhage due to vitamin K deficiency, or warfarin effect
- Replacement of specific coagulation factor in the event of nonavailability of its concentrate form
- Antithrombin III deficiency.

**Contraindications**

- FFP should never be used as a volume expander, to supply albumin or other nutrients to the patient, as it carries the risk of disease transmission.

- In the absence of clinical bleeding, FFP should not be transfused for a complete correction of coagulation profile.

**Dose and Response**

- During massive transfusion, FFP is transfused in the dose of 10–15 mL/kg
- As the plasma volume in a neonate is about 40 mL/kg, transfusing 20 mL/kg of FFP provides about 50% of normal factor levels
- In children and adults, 10 to 20 mL/kg of plasma will increase a coagulation factor concentration to approximately 30% of normal.

**Cryoprecipitate**

It is prepared from fresh frozen plasma. It contains Factor VIII (80–150 U/unit), von Willebrand factor (100–150 U/unit), fibrinogen (150 to 250 mg/unit) and Factor XIII.

**Indications**

1. To provide fibrinogen when blood levels are below 80–100 mg/dL.
   - Congenital fibrinogen deficiencies
   - DIC
   - Liver failure
   - Post-streptokinase therapy (hyperfibriogenolysis).
2. Factor XIII deficiency.
3. Non-availability of factor concentrates:
   a. von Willebrand’s disease: in children in the dose of 1 unit/6 kg every 12 hourly
   b. Hemophilia A: 1 unit /6 kg gives an approximate factor VIII level of 35% if the patient has <1% at initiation of therapy.

**Dosage:** 5 to 10 mL/kg or 1 unit per 5 to 10 kg body weight. This increases the fibrinogen level by 60 to 100 mg/dL. In a neonate 1 unit will increase fibrinogen by >100 mg/dL.

**Plasma Derivatives**

Plasma derivatives prepared in fractionation centers from plasma pools are—albumin, immunoglobulins, factor VIII concentrates, Factor IX concentrates, antithrombin III and prothrombin complex concentrates.

**Purified Factor VIII Products**

In addition to factor VIII, the enriched products also have high amount of vWF. These products are also useful in treating von Willebrand disease.
Prothrombin Complex Concentrate (PCCs)

It consists of plasma fractions that are enriched for the vitamin-K dependent clotting factors.

**Indications**
- Hemophilia B
- Reversal of medication with warfarin
- Overcoming factor VIII inhibitors
- Severe vitamin K deficiency with life-threatening hemorrhage.

**Adverse events:** Thrombosis, thrombophlebitis, pulmonary embolism and DIC.

**Contraindication:** A severe allergic or anaphylactic reaction to infusion of a preparation makes the next dose contraindicated.

Topical Agents

Topical fibrin and thrombin combinations are adjuncts to surgical hemostasis and are useful for diffuse surgical bleeding. There is a risk of anaphylactic reaction to bovine proteins with the topical hemostatic agents which use bovine constituent.

Recombinant Products

Recombinant factors provide the lowest risk of transmission of infectious agents. The recombinant products in clinical use include factor VIIa, factor VIII, and factor IX. Recombinant factor VIIa is used for factor VII deficiency and for the management of Hemophilia A with inhibitors.

PHARMACEUTICAL PREPARATIONS

Tranexamic Acid

Tranexamic acid is a fibrinolytic inhibitor and an adjunct to therapy to reduce mucosal bleeding in conditions like hemophilia and von Willebrand disease. The oral dose is 25 mg/kg every 6 to 8 hourly. Other antifibrinolytic agents are alpha aminocaproic acid and aprotinin.

DDAVP

Desmopressin (1-deamino-8-D-arginine-vasopressin) is a vasopressin analogue.

**Indications**

1. To increase circulating levels of factor VIII in Hemophilia A
2. To increase vWF levels in type 1 and in some patients with type 2A vWD
3. To control bleeding in cases of uremia.

DDAVP is administered either by nasal aerosol or intravenous infusion, every 24 hours.

AVAILABLE MODIFICATIONS OF BLOOD COMPONENTS

Open System vs Closed System

When smaller aliquots are prepared using an open system, the shelf life of the blood component reduces. Red cells must be used within 24 hours and platelets must be used within 4 hours of separation. When aliquots are prepared without an environmental exposure, i.e. in a closed system, shelf life remains the same as that of the original component.

Fresh Blood

As fresh blood contains maximum level of 2,3-diphosphoglycerate (2,3-DPG), it is preferred for neonates, especially premature neonates, and neonatal or pediatric patients with impaired pulmonary function. Fresh blood (3–5 days old) also contains less amount of extracellular potassium, compared to older blood.

Plasma Reduction

Plasma is present in whole blood, platelet concentrates and in apheresis platelets.

Plasma is reduced from the components which are to be transfused to patients who develop severe allergic or anaphylactic transfusion reactions. Plasma reduction should be also done when group O component is to be transfused to group A, B or AB. This is because plasma of group O contains anti-A and anti-B which may be responsible for hemolysis of the recipient’s red blood cells. Plasma can be reduced by removing the supernatant and by washing the cells with saline.

Removal of Additive Solution

Some of the additive solutions which may have been added to red cells to increase the shelf life may contain adenine and mannitol. Both these constituents are known to be associated with renal toxicity. Mannitol may cause fluctuations in cerebral flow. For these reasons, additive solution must be removed from the red cells intended for infants younger than 4 months and for those with severe hepatic and renal compromise.

Red cells can be re-suspended in saline, albumin or thawed FFP.
Leukoreduction

Leukoreduction can either be done in the blood bank or can be done using bedside filters. Leukoreduction of red cells and platelets reduces the risk of:

- Febrile nonhemolytic transfusion reactions
- Alloimmunization against human leukocyte antigens and therefore platelet refractoriness and graft rejection
- Transmission of cytomegalovirus, Epstein-Barr virus and human T-cell lymphotropic virus type I.

**KEY POINT**

Leukoreduction does **not** prevent transfusion associated graft vs. host disease

Random Donor Platelets vs Single Donor Platelets

Platelets for transfusion are prepared by two different methods. ‘Random donor platelet concentrate’ is prepared from one unit of whole blood.

Whereas, ‘Single donor platelet unit’ is prepared by multiple cycles of apheresis technique performed on one single platelet donor. This method yields 6–8 times higher number of platelets compared to ‘Random donor platelet concentrate’. It is also called ‘Apheresis platelets’. As it is derived from one single donor, it reduces the risk of multiple donor exposures in reference to HLA alloimmunization and infectious disease transmission. Also, this component differs from Random-donor platelet units as it only contains 10⁴ to 10⁶ leukocytes per suspension.

Irradiation

Irradiation of cellular blood components is done to prevent transfusion associated graft vs host disease. It is indicated in the following:

- Immunodeficiency diseases
- Intrauterine transfusions
- Transfusion from a blood relative
- Bone marrow transplant patients
- HLA matched components
- Patients receiving immunosuppressive therapy.

Limited Donor Exposures

It is important to limit the number of donor exposures to a patient for reducing the risk of transfusion-associated infections as well as allogeneic antigen exposure.

For small children, this can be achieved by using multiple aliquots from the same unit of blood or its component.

Directed Donation

Maternal blood may contain antibody against neonate’s red cells or platelets and paternal blood may contain antigens to which the neonate may have obtained antibodies from the mother. Parental blood may also be responsible for transfusion associated graft vs host disease. It is recommended not to use parental blood in the first few weeks of life. Directed blood donation from blood relatives should be provided only upon request. Blood from blood relatives must be irradiated to prevent graft versus host disease.

Warming

Transfusion of hypothermic blood components especially to premature and low-birth weight infants predisposes them to hypothermia. A blood warming device should be used for rapid (>1 mL/minute) or massive transfusion (>25 mL/kg).

Warming of blood components with lights or any other non-approved methods must not be done as it may lead to thermal injury to blood components.

**TRANSFUSION PROCEDURE**

Informed consent is a vital part of the transfusion process. It is required for all blood product transfusions except during an emergency. The physician must explain to the patient and his or her parents the treatment plan, risks, benefits, and alternative approach to treatment. A witness, along with a parent and/or guardian of the patient, must sign a standardized transfusion consent form along side of the physician acknowledging consent.

Collection and Labeling of Blood Sample

Confirmation of identity of the patient is important while collecting and labeling of the sample for sending to the blood bank.

It is equally important to match patient’s identity with the crossmatch number, and the blood component unit number, while initiating a transfusion, since clerical errors remain the leading cause of fatal hemolytic transfusion reactions.
Types of Blood Release

1. Without grouping: When there is a need for immediate transfusion and it is not possible to send the patient’s sample to the blood bank, the emergency order for blood requires a signature from the physician as the units will be issued without a crossmatch. The red cell concentrates selected are of a Group O Rh negative donor.

Group O, Rh negative blood is rare, preference is given for female patients, who may later become pregnant with an Rh positive fetus, predisposing to hemolytic disease of the fetus and newborn. The next appropriate choice for a male patient is Group O, Rh positive red cell concentrate.

2. Group specific: in semi-urgent situations, with a limited amount of time available, patient’s sample can be quickly sent to the blood bank. ABO and Rh group specific blood (A to A, B to B) can be issued without a crossmatch.

3. Urgent crossmatch: If a patient’s sample can be immediately delivered to the blood bank or if a current, prior sample is available with the blood bank, an urgent cross match can be performed within 15–20 minutes.

4. Routine grouping, antibody screening and cross matching can be performed if time permits.

Rate of Transfusion

Factors which influence hemolysis during transfusion of red cells are, the hematocrit of red cell concentrate, gauge of the needle and the flow rate. When 24-gauge catheter is used for premature infants, transfusion should be given slowly. Infusion pumps which are used for neonates, release consistent and low bolus.

INVESTIGATIONS

Prothrombin time (PT) measures the adequacy of factors VII, X, V, prothrombin, and fibrinogen (the extrinsic system). Activated prothrombin time test (aPTT) measures the adequacy of factors XII, XI, IX, VIII, X, V, prothrombin and fibrinogen (intrinsic pathway).

Both the PT and aPTT will be prolonged in patients with deficiencies of factors X, V, prothrombin, and fibrinogen and in patients with DIC, severe liver disease, presence of lupus anticoagulant, vitamin K deficiency.

In the event of an acute blood loss, hematocrit does not equilibrate immediately and hence cannot be relied upon.

Bleeding Disorders and Transfusion Therapy

Von Willebrand Disease

It is an autosomal dominant disease and both males and females with von Willebrand’s disease present with bleeding symptoms. The characteristic findings of this disease are mucocutaneous bleeding including epistaxis, bleeding after dental extractions or ear, nose, throat procedures and easy bruising. A characteristic feature of the disease is the variability in bleeding symptoms at different times, in the same patient. The vWF is responsible for the adhesion and aggregation of platelets to the injured vessel wall. vWF also acts as a carrier for the factor VIII in circulation and localizing it to the site of vascular injury.

Therapy: Virally inactivated vWF/factor VIII concentrates are preferred over cryoprecipitate. In the patients who respond to DDAVP, transfusion needs are reduced.

Hemophilia

Hemophilia A and B are X linked recessive disorders. Deficiency or absence of factor VIII activity is seen in Hemophilia A and factor IX activity for hemophilia B (Christmas disease). Hemophilia is classified as severe hemophilia (factor level of 1% or less), moderate (2% to 5% factor level) and mild (more than 5% factor level). It is most often associated with bleeding into joints and soft-tissue ecchymosis. Acquired inhibitors of factor VIII can clinically mimic hemophilia. Factor XI deficiency is known as hemophilia C.

Before surgery, it is essential to perform tests to ensure that the patient has not developed a specific inhibitor of clotting. Doses of the concentrate are usually given at 12–hourly intervals in hemophilia A and at 24-hourly intervals in hemophilia B.

On the morning of surgery, a 100% correction of clotting factor is given, and the factor level is maintained at greater than 50 to 60% for the next 5 to 7 days. After that, factor levels can be maintained at 30% for the period of time needed for postsurgical healing and rehabilitation to take place.

The low titer inhibitors, less than 5 Bethesda units in activity, can be treated with higher doses of factor. These patients receive 40 units of factor VIII/kg body weight for each Bethesda unit of inhibitor. The high-titer inhibitor (>5 Bethesda units in activity) patients may receive 100 to 200 units/kg/hour of continuous infusions of factor VIII, which may control the bleeding. Alternatively, Prothrombin complex concentrate, recombinant factor VIIa and porcine factor VIII concentrate can be useful. Repeated
plasmapheresis to reduce the inhibitor concentration and infusion of factor IX concentrates have been found useful.

**Other Congenital Deficiencies**

Dysfibrinogenemia is a rare congenital hemorrhagic disorder with an autosomal dominant inheritance. It is characterized by secretion of functionally abnormal fibrinogen. Patients may be either asymptomatic, have hemorrhage or have arterial or venous thrombosis. A pattern of delayed bleeding is seen in homozygous factor XIII deficiency as clots repeatedly form and then break down 24 hours later. Presentation of homozygous deficiency of factor VII, is often in infancy with bleeding symptoms, including intracranial hemorrhage. Clinical presentation of factor X deficiency is with muccutaneous or post-traumatic bleeding. Prothrombin complex concentrates are recommended for use in complicated surgical procedures where higher levels of factor X are required. Factor II (Prothrombin) deficiency is a bleeding disorder with mild to moderate bleeding.

Severe deficiency of alpha-2-antiplasmin is associated with a severe bleeding disorder caused by excessive fibrinolysis. Although FFP contains alpha-2-antiplasmin and plasminogen activator inhibitor-1, bleeding due to these deficiencies can be controlled with antifibrinolytics epsilonaminocaproic acid or tranexamic acid. Plasminogen activator inhibitor-1 deficiency presents with severe bleeding. This is caused by lack of regulation of plasminogen activity and excessive fibrinolysis.

**Acquired Coagulation Factor Inhibitors**

Hemorrhagic lupus anticoagulant syndrome has been described in the clinical setting of a previously well child who develops bleeding symptoms of variable severity following a viral infection.

Acquired antibodies to factor V develop in some patients after exposure to bovine thrombin as part of fibrin glue, most often used during cardiac surgery. Bleeding is variable, ranging from none to significant hemorrhages.

**Acquired Platelet Disorders**

Decreased platelet numbers result from many conditions that decrease platelet production, increase destruction, increase sequestration, or occur due to dilutional thrombocytopenia during massive transfusion.

Platelet function may be adversely affected by factors such as drugs, sepsis, liver or kidney disease, increased fibrin degradation products, cardiopulmonary bypass, and primary marrow disorders.

Since intracranial hemorrhage has been reported in 10 to 15% of cases of neonatal alloimmune thrombocytopenia (NAIT), it is extremely important that effective therapy be provided as soon as possible to infants with suspected or confirmed NAIT and severe thrombocytopenia (platelet counts <30 × 10^9/L).

**Vitamin K Deficiency**

Vitamin K deficiency leads to multiple factor deficiencies. Similar clinical manifestations also are seen with warfarin treatment.

**Correction**

- Within 6 to 8 hours with oral replacement
- Within 2 to 6 hours with subcutaneous vitamin K
- Urgently by transfusing FFP
- Prothrombin complex concentrates, recombinant factor VIIa can be used in life-threatening hemorrhage, e.g. intracranial hemorrhage.

**Disseminated Intravascular Coagulation (DIC)**

In infants with severe DIC, mortality can reach up to 80%. DIC is associated with shock, trauma and other forms of tissue damage. An increase in the D-dimer and schistocytes and helmet cells on peripheral smear when present, help in differentiating DIC from dilutional coagulopathy. Treatment of DIC depends on resolution of the underlying disorder.

Transfusion of FFP is indicated for replacement of clotting as well as inhibitor factors. FFP is transfused at a dose of 10 to 20 mL/kg and repeated every 6 to 8 hourly. Cryoprecipitate is transfused at a dose of 10 mL/kg to maintain fibrinogen >100 mg/dL. Platelets are given at a dose of 5 to 10 mL/kg to maintain platelet count >50,000/mL.

**Dilutional Coagulopathy**

In massive transfusion, hemostasis may be compromised by replacement of plasma volume with crystalloid ‘or’ colloid solutions or stored blood. However, dilutional coagulopathy does not usually occur until replacement of one to two blood volumes within 24 hours. The coagulopathy of massive blood transfusion is primarily related to dilution of either clotting factors or platelets or both.

Coagulopathy is proportional to severity of hypothermia, severe metabolic acidosis, poor tissue perfusion, and release of tissue factors, thus the importance of maintaining body temperature through the
use of efficient blood-warming devices and the need to restore circulation and clear the acidosis.

Liver Diseases and Warfarin Effect
Liver failure from congenital infectious hepatitis, sepsis or other factors may be a major cause of coagulation laboratory test abnormalities and bleeding in neonatal patients. Patients with severe liver disease frequently exhibit a dysfibrinogenemia.

Oral or intravenous vitamin K should be started early in the management of liver disease, avoiding intramuscular injections due to the risk of hematomas. Vitamin K administration will reverse warfarin effects within 12 hours in a patient with normal liver function and is appropriate treatment when time permits. FFP can be used to correct the hemostatic defect of warfarin. As in liver failure, initial FFP doses of 10 to 15 mL/kg are recommended. Efficacy should be assessed by careful clinical assessment of bleeding and laboratory monitoring with the PT and aPTT.

Uremia
Blood urea nitrogen levels greater than 40 to 50 mg/dL are associated with platelet function defects, causing bleeding from mucosal surfaces. The corrective measures recommended to improve hemostasis in uremia patients with excessive bleeding are, transfusion of red blood cells to a hematocrit value >30% and administration of 1-deamino-8-D-arginine (DDAVP). As platelet dysfunction is known to occur in uremia, excessive surgical bleeding in these patients may prompt consideration of platelet transfusion. However, since transfused platelets are rapidly impaired by uremia, their use should be considered secondary.

ADVERSE TRANSFUSION REACTIONS
During the transfusion, and especially within the initial 15 minutes, it is important to observe for signs of bacterial infection, fever, allergy, and anaphylactic reactions.

Allergic Reactions
Allergic reactions to blood components can vary in severity. Anaphylaxis occurs within minutes of transfusion. There may be marked hypotension and respiratory distress due to laryngeal edema or bronchospasm. Airway obstruction and shock need an urgent management. Transfusion should be stopped and should not be restarted. If an allergic reaction to blood component transfusion is limited to the development of hives or urticaria, the transfusion must be stopped temporarily for administration of an antihistamine and transfusion can be resumed after resolution of the cutaneous symptoms. Patients with a history of allergic reactions are more likely to develop urticaria following blood component transfusions. In these patients, premedication with antihistamines or corticosteroids should be considered. Allergic reactions to food, drugs, or latex may mimic allergic transfusion reaction. Factor IX infusions are associated with anaphylaxis.

Hemolytic Transfusion Reactions (HTRs)
Hemolytic transfusion reaction due to ABO incompatible transfusion is one of the three most common causes of fatal transfusion reactions, the other two being post transfusion sepsis and transfusion related acute lung injury. It results most commonly from failure of identification of the recipient, either at the time of transfusion, or at the time of initial phlebotomy for sample collection or due to error while labeling. Effort must be made to strengthen the system of patient identification and to prevent other technical errors. Acute hemolysis can result from transfusion of ABO incompatible red blood cells or ABO incompatible plasma containing blood component. Characteristic features of ABO incompatible transfusion and hemolytic reaction are hemoglobinemia and hemoglobinuria. Hemolytic transfusion reaction can lead to shock and circulatory collapse. Hypotension and renal ischemia may give rise to acute tubular necrosis and renal failure. HTR may also culminate into DIC due to the activation of the coagulation and fibrinolytic systems. The management of the hemolytic transfusion reaction includes discontinuation of the transfusion, maintenance of IV access, maintaining the blood pressure, promoting adequate renal flow and performing clinical evaluation and laboratory investigations.

Hypocalcemia
The anticoagulant sodium citrate which is used for anticoagulation of the donor blood, binds ionized calcium from the recipient’s blood. This may result in transient hypocalcemia, especially after transfusion of fresh frozen plasma. Hypothermia and immature liver function impair citrate metabolism. Frequent monitoring of ionized calcium, pH, and potassium may be necessary during high volume transfusion (>25 mL/kg). Corrective measures include calcium gluconate (100 mg/kg) for routine replacement and calcium chloride (10 to 20 mg/kg) for rapid or emergent replacement. Caution is advised, as excessively rapid infusion of calcium chloride has been associated with death.
Hyperkalemia
The extracellular potassium content of red cell units increase with storage. In small volume neonatal transfusions, i.e. at 15 mL/kg, administered slowly over 3 to 4 hours, the potassium content does not pose a problem. When stored blood is transfused in large volumes or rapidly, i.e. when it exceeds 1.5 to 2.0 mL/kg/min, the electrocardiogram must be closely monitored. The high concentrations of extracellular potassium content can result in fatal cardiac disturbances in small infants, especially when accompanied by acidosis and administration of other potassium containing drugs or intravenous fluids. It is preferable to use washed, extracellular volume reduced or fresh (<5 to 7 days old) RBC components in these situations. Irradiated components are known to have increased levels of extracellular potassium levels.

Alloimmunization
Transfusion exposes the recipient to foreign red blood cell antigens, human leukocyte antigens (HLA) as well as human platelet antigens. This foreign antigen exposure initiates an alloimmunization against the antigen. In the event of re-exposure to identical antigen, there is an immune destruction of the antigen bearing cells. This alloimmunization is responsible for delayed hemolytic transfusion reactions, platelet refractoriness, increased risk of allograft rejection and post-transfusion purpura.

Post-transfusion Purpura
Post-transfusion purpura is due to alloantibodies to platelet specific glycoproteins, most commonly to HPA-1a. Post-transfusion purpura is characterized by the acute onset of thrombocytopenia approximately 5 to 14 days after a transfusion. Previous transfusions may sensitize patients to foreign platelet antigens. This results in the destruction not only of the transfused platelets, but also of the patient’s own platelets.

Transfusion Associated GvHD
Donor’s blood and its cellular components contain viable lymphocytes. In the event that the recipient cannot inactivate them, these lymphocytes give rise to Transfusion associated graft-vs. Host disease (TA-GVHD), which is almost always fatal. Cellular components of blood must be irradiated to prevent TA-GVHD. Irradiated components must be provided for intrauterine transfusions, low-birth weight infants, patients with Immunodeficiency syndromes, immunosuppressed patients, bone marrow transplant recipients, patients on extracorporeal membrane oxygenation and those receiving directed donation from blood relatives and HLA matched components.

Transfusion-related Acute Lung Injury (TRALI)
Transfusion-related lung injury should be a consideration in any patient who develops respiratory distress and hypoxemia during or shortly after transfusion. TRALI occurs 1 to 6 hours after transfusion and is due to the presence of antibodies to leukocytes that are contained in the donor plasma. Patients present with hypotension, fever, and pulmonary infiltrates associated with severe hypoxemia.

LEARNING POINTS
- Factors affecting the clinical manifestation of a bleeding disorder are: its type, severity, presence of active bleeding, response to pharmacological agent, presence of inhibitors, and availability of therapeutic agents
- Clinical assessment of bleeding is critical in early diagnosis and management of an unanticipated hemostatic catastrophe
- Judicious use of pharmacological agents reduces the requirement of blood transfusion
- Transfusion trigger is the level of blood constituent below which most patients need transfusion. Transfusion trigger for red cells is higher for patients with cardiopulmonary compromise. Transfusion trigger for platelets is higher for patients with active bleeding, associated coagulation disorder, fever and splenomegaly
- Hemolytic transfusion reactions can be prevented by careful patient identification during sample collection and transfusion
- Transfusion of blood and its components must be considered only when the benefits outweigh the risks

SUGGESTED READING
Section 3

Subspecialty Anesthesia

Chapter 15: Anesthesia for Surgery in the Neonate
Chapter 16: Anesthesia for Ear, Nose and Throat Procedures in Children
Chapter 17: Anesthesia for Plastic and Reconstructive Surgery
Chapter 18: Anesthesia for Pediatric Dentistry
Chapter 19: Anesthesia for Ophthalmic Procedures
Chapter 20: Anesthesia for Major Burns and its Consequences
Chapter 21: Anesthesia for Pediatric Neurosurgical Procedures
Chapter 22: Anesthesia and Pediatric Liver Diseases
Chapter 23: Anesthesia for Pediatric Urologic Procedures
Chapter 24: Anesthesia for Pediatric Laparoscopic Surgeries
Chapter 25: Anesthesia for Pediatric Thoracic Surgery
Chapter 26: Anesthesia at Remote Locations
Chapter 27: Ambulatory Anesthesia in Children
Chapter 28: Anesthesia for Pediatric Orthopedic Surgery
Chapter 29: Anesthesia for Pediatric Trauma
Chapter 30: Anesthesia for Pediatric Cardiac Surgery
Chapter 31: Anesthetic Management of Children with Congenital Heart Disease for Noncardiac Surgery
Anesthesia for Surgery in the Neonate

INTRODUCTION
Surgical conditions in the neonate cause significant physiologic perturbations and the perioperative management is a daunting task for the anesthesiologist. The newborn requires anesthesia to prevent pain and stress responses, while maintaining physiologic stability. This chapter discusses neonatal physiology and pharmacology as applicable to the neonate for surgery, and anesthesia management of common neonatal general surgical procedures.

NEONATAL PHYSIOLOGY AND THE SURGICAL NEONATE
Respiratory System
The increased oxygen requirement (5–8 mL/kg/min versus 2–3 mL/kg/min in adult), coupled with reduced reserve, makes the neonate exquisitely prone to hypoxemia. The relatively large abdomen displaces the diaphragm cephalad, placing the lungs’ closing capacity within the functional residual capacity. Increased intra-abdominal pressure due to abdominal distension (mask ventilation, obstructive conditions of the bowel), surgical retraction or replacement of bowel into the abdominal cavity (gastrochisis, omphalocele, congenital diaphragmatic hernia) can further reduce lung volumes. Preterm neonates, in addition, have surfactant deficiency and reduced lung volumes due to atelectasis.

The resistive work of breathing in the neonate is nearly 6 times greater than the adult due to the small size of the airways. Breathing through a tracheal tube further increases work of breathing, since resistance is directly proportional to the length of the tracheal tube and inversely proportional to the fifth power of the radius of the tube. Thus the recommendation is that, while a circle system to deliver anesthesia can be safely used in neonates, assisted/controlled ventilation with adequate inflation pressures are necessary to ensure optimum ventilation without increasing work of breathing.1

Apnea in neonates can be classified as:
- Central apnea: Resulting from immaturity or depression of respiratory drive
- Obstructive apnea: Due do the inability to maintain a patent airway
- Mixed: A combination of both.

Susceptibility to central apnea occurs in preterm neonates, and even in term neonates with anemia, sepsis, hypothermia, and metabolic disturbances such as hypoglycemia and hypocalcemia. Opioids increase the risk of central apnea. Xanthine derivatives (caffeine, theophylline) are useful for management.2 Obstructive apnea is common in preterm neonates and is managed with changes in positioning (lateral/prone), neck extension, use of oral or nasal airways and use of continuous positive airway pressure (CPAP).3

Postoperative Apnea
Risk of postoperative apnea should be considered in all patients <44 weeks postconceptional age (PCA) regardless of the anesthetic technique. This risk persists until 60 weeks PCA for infants born less than 37 weeks PCA.4 Apnea
may result from prolonged action of anesthetic agents, a shift of the CO₂ response curve, immaturity of respiratory control, or respiratory muscle fatigue. Increased risk for postoperative apnea include younger gestational and PCA, history of apnea events, hemoglobin <10 g/dL, opioid administration, coexisting disease and physiologic abnormalities. Spinal anesthesia in the absence of any other anesthetics/sedatives/analgesics may decrease the risk of postoperative apnea in the former premature infant.5,7

**Cardiovascular System**

During intrauterine life, gas exchange function is conducted by the placenta, and the pulmonary circulation is marked by low flow and high vascular resistance. At birth, the systemic vascular resistance (SVR) rises due to elimination of the placental circuit, and the pulmonary vascular resistance (PVR) falls as the infant's lungs expand with the onset of respiration resulting in closure of the ductus arteriosus and foramen ovale. Pain, hypothermia, hypoxemia, hypercarbia, acidosis, use of nitrous oxide, and high inflating pulmonary pressures result in increased PVR and may result in the return of fetal circulation leading to severe hypoxia perioperatively. SVR is relatively fixed in infants at birth due to incomplete sympathetic innervation, resulting in the large arteries being in a relatively dilated state and unable to fully contract in response to hypovolemia. Maintenance of blood pressure is related to maintenance of cardiac output via heart rate, as the neonatal ventricles are poorly compliant with a fixed stroke volume. Maintenance of circulating volume and heart rate are essential to maintenance of blood pressure in newborns.

**Temperature Regulation**

Neonates easily become hypothermic in the operating room (and during transport) due to their high body surface area to weight ratio, thin skin, decreased body fat, and inability to shiver.4 Anesthetics further impair non-shivering thermogenesis from brown fat, which is the newborn's only mechanism to maintain body heat. Differentiation of brown fat cells begins at 26–30 weeks gestation; hence premature neonates are exquisitely prone to hypothermia. The consequences of hypothermia include pulmonary hypertension, right to left shunting of blood, hypoxemia, delayed drug metabolism, and apnea.

**Box 1: Strategies to maintain body temperature**

- Transport baby in an incubator
- Warm the operating room to > 27°C
- Use of warming mattress, forced air warming
- Plastic drapes
- Warm scrubbing and irrigating solutions
- Heated airway humidification
- Warm intravenous fluids and blood
- Overhead radiant warmer (pre- and postprocedure)

**Fluid and Electrolyte Management**

The basic principles of fluid and electrolyte management in neonates have been dealt with in a separate chapter. Increased insensible fluid losses often occur in the operating room. Congenital anomalies (e.g. gastroschisis, omphalocele) will markedly increase insensible losses by exposure of large mucosal surfaces. Bowel obstruction will lead to third space losses of fluid and electrolyte abnormalities. Urine volume may be difficult to determine intraoperatively, and may not always correlate with volume status. Anesthetics may mask subtle cardiovascular changes that occur with altered volume status. Renal immaturity, variable body fluid composition, and the effect of the neuroendocrine stress response to surgery make accurate administration of fluids to maintain circulating volume a challenging task. Conversely, overzealous administration of fluids can easily overload the neonate and worsen third spacing. Maintenance fluid consists of hypotonic glucose solutions (D₁₀ in either water or 0.2% NS). For replacement of insensible and small volume blood loss, isotonic fluid should be administered separately. Accuracy in fluid administration can be achieved by using infusion pumps.

**Glucose Homeostasis**

Development of glycogen stores in the fetus does not occur until late in gestation. Neonates who are prone to hypoglycemia include preterm and small for gestational age infants, full term infants who have been excessively fasted, infants of diabetic mothers, and neonates on total parenteral nutrition. Glucose infusion at rates of 8–10 mg/kg/min and 5–8 mg/kg/min prevents hypoglycemia in preterm and term neonates respectively. Perioperative hyperglycemia can occur easily when high glucose containing fluids are given in combination with operative stress.9 During surgery, it is recommended to administer glucose containing solutions using a constant infusion device and regularly monitor blood sugar to avoid both hyper and hypoglycemia. All other fluids used
intraoperatively (to replace third space losses, blood loss, fluid deficit) should be glucose free.

**Calcium Homeostasis**

Hypocalcemia is common in critically-ill neonates. Causes include parathyroid hormone (PTH) insufficiency, peripheral resistance to PTH, inadequate calcium supplementation, and altered calcium metabolism caused by transfusion of citrated blood products, bicarbonate administration or diuretics (furosemide). Neonatal hypocalcemia is defined as a serum Ca $^{2+}$ less than 1 mmol/L in full term infants and 0.75 mmol/L in preterm infants. Symptomatic hypocalcemia is treated with 100 mg/kg calcium gluconate by slow intravenous infusion, followed by a maintenance dose of 100–200 mg/kg/day.

**Neurologic Development**

Immaturity of the brain and its blood vessels, especially in the preterm neonate, increases the risk of intraventricular hemorrhage (IVH) in the neonatal period. Factors that increase the risk of IVH include fluctuations of blood pressure, hypoxia, hypercarbia, and pain. This association has been a concern in “awake laryngoscopy and intubation” in neonates. Questions have also been raised about the vulnerability of the developing neonatal brain to injury due to anesthetic and/or analgesic agents such as isoflurane, nitrous oxide, ketamine and midazolam. Hyperoxia has been associated with retinopathy of prematurity. The optimal saturation range has not been defined in preterm neonates; a preductal saturation range of 91–95% seems appropriate.

It is now clear that neonates, even the very preterm, can perceive pain. Emerging studies provide convincing clinical evidence for an adverse impact of neonatal pain/stress in infants at a time of physiological immaturity, rapid brain development, as well as programing of the hypothalamic-pituitary-adrenal axis. Exposure to prolonged and repetitive pain-related stress in neonates, especially those born very preterm may potentially have long-term effects contributing to altered neurobehavioral development.

**Monitoring**

Minimal monitoring for all neonates undergoing surgery includes pulse oximetry, capnography, non-invasive blood pressure (NIBP), ECG and core temperature. The pulse oximeter should be placed on a preductal site. A second oximeter probe may be placed for back up, or when right-left shunting may be a possibility. Monitoring of urine output is recommended in neonates having major surgery. Intra-arterial catheters are useful in critically ill neonates who are at risk for large intraoperative blood and/or fluid losses and who are hemodynamically unstable. It allows for continuous monitoring of blood pressure and periodic assessment of blood gases, glucose, and electrolytes. Central venous catheters are useful for both monitoring and as a secure IV access, particularly in procedures with anticipated large blood losses or fluid shifts. Peripherally inserted central catheters (PICC) although not able to measure CVP are useful especially for administration of medication. Precordial stethoscope can give valuable information regarding correct position of endotracheal tube, and heart rate, rhythm, and volume status by quality of heart sounds.

**NEONATAL PHARMACOLOGY AND CONSIDERATIONS FOR ANESTHESIA**

The pharmacologic differences in neonates (body fluid composition, renal and hepatic function) impact choices and doses of medications. Other factors affecting drug metabolism in the perioperative period include cardiac function and factors affecting liver and kidney blood flow (hypovolemia, fluid and blood loss, sepsis, effect of anesthetics).

**Intravenous Anesthetics and Analgesics**

Neonates have increased sensitivity to and more prolonged effects of barbiturates and opioids, due to the immaturity of the blood-brain-barrier, allowing greater penetration and higher concentration of these drugs in the brain. Additionally, immaturity of respiratory control mechanisms result in greater respiratory depression leading to apnea at lower levels of these drugs.

Plasma clearance of opioids correlates well with maturity, postnatal age, and weight. Infants with higher intraabdominal pressure (omphalocele, CDH) have a further increase in elimination half-life. Due to the large variability seen in clearance in neonates of different ages, it is recommended that opioids be titrated to each patient carefully. Remifentanil, is an ultra-short acting opioid metabolized by plasma and tissue esterases. Its organ independent elimination, and consequently, its predictable half-life makes it an ideal drug for neonates. Thiopental is highly protein bound, resulting in higher free drug levels for any given dose, however, the larger volume of distribution results in higher induction doses (5–6 mg/kg) being used in neonates. Ketamine requirements are greater (mg per kg of body weight)
in infants than in older children. Although propofol is commonly administered to infants, recommendations are not available regarding its use in neonates. There is considerable inter individual variability of propofol clearance, and preterm neonates and neonates in the first week of postnatal life are at an increased risk for accumulation during either intermittent bolus or continuous administration of propofol. Midazolam has been extensively used in term and preterm infants, and clearance in preterm infants is reduced.

**Inhaled Anesthetic Agents**

Neonates have a faster uptake of inhaled anesthetic agents due to higher alveolar ventilation and cardiac output. Because of difference in solubility and smaller muscle and fat group, they attain a higher \( F_{1} / F_{r} \) ratio than adults. MAC is lower in neonates than in older infants. Sevoflurane has less effect on hemodynamics, compared to halothane, hence it is preferred. Time to emergence is significantly faster with desflurane than any other volatile anesthetic agents; this may be particularly beneficial in the neonate in whom extubation is planned.

**Muscle Relaxants**

Synaptic transmission is slow at birth, the rate at which acetylcholine is released during repetitive stimulation is limited, and neuromuscular reserve is reduced. The increased volume of distribution results in lower concentrations of non-depolarizing muscle relaxants at the neuromuscular junction, however, neonatal neuromuscular receptors are more sensitive so there is no difference in initial dosage. The longer elimination half-life implies that subsequent doses need to be smaller and less frequent. Benzylisoquinoline muscle relaxants (atracurium or cisatracurium) are preferred, as they have organ independent elimination and a more predictable duration of action, provided the patient is normothermic and has a normal acid-base status.

**Local Anesthetics**

The use of bupivacaine, the drug most commonly used for caudal epidural anesthesia, is associated with higher blood levels in neonates, due to immature liver metabolism and decreased protein binding. Initial doses of local anesthetics should be reduced in neonates by around 50% from the equivalent adult dose per kg body weight and should be given slowly in increments. Maintenance infusion rates should also be reduced by about 50% from those in older children and adults.

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**Box 2: General rules for all emergent surgery in neonates**

- No sedative premedication; consider atropine prior to induction.
- Discuss anesthesia plan with the parents/caregivers, including possibility of postoperative ventilation
- Have a trained anesthetic assistant
- Prepare appropriate equipment, e.g. size 3.0–3.5 tracheal tubes for a term newborn, 2.5 for a premature infant. Straight blade laryngoscope size 0,1, infant facemask, appropriate breathing circuit and pediatric ventilator. Confirm all drugs drawn up in appropriate dose. Use a burette or syringe infusion pump for intravenous fluids
- Take all precautions to prevent hypothermia
- Monitoring—ECG, NIBP, saturation, EtCO\(_2\), temperature monitoring as minimum; consider intra-arterial monitoring, CVP monitoring in select cases
- Check adequacy of IV access. Place a second line after induction for most cases

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**SPECIFIC NEONATAL SURGICAL LESIONS**

**Gastrointestinal Problems**

**Obstructive Lesions**

Neonates present with vomiting, abdominal distension, and delayed passage of meconium. There may be associated dehydration, electrolyte abnormalities, aspiration pneumonia and respiratory distress. Unless a life-threatening compromise of organ blood flow occurs, the priority is to re-establish euvolemia and correct metabolic derangements prior to anesthetic induction.

**Lesions with Compromise in Intestinal Blood Supply**

Neonates with compromise in bowel perfusion are very ill. They present with tender distended abdomen, bloody stools, vomiting, hypotension, metabolic abnormalities, anemia, leukopenia and thrombocytopenia. Emergency surgery is needed to remove necrotic tissue, close perforations and reestablish blood supply to the bowel. These patients are poor candidates for central neuraxial block in view of coagulopathy. Blood and blood products should be available in the OR. In case poor perfusion persists in spite of volume resuscitation, sympathomimetics may improve cardiac output and organ perfusion.

**Abdominal Wall Defects—Omphalocele and Gastroschisis**

Although similar in appearance, these two lesions have distinct differences (Table 1). Ultrasound can confirm the diagnosis as early as 12 weeks gestation, allowing for planned delivery at a well-equipped facility.
### Table 1: Abdominal wall defects—Omphalocele and gastroschisis

<table>
<thead>
<tr>
<th></th>
<th>Gastroschisis</th>
<th>Omphalocele (Exomphalos)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Incidence</strong></td>
<td>1: 3000</td>
<td>1: 5000</td>
</tr>
<tr>
<td>Maternal age often &lt; 20 years</td>
<td></td>
<td>Maternal age often &gt; 40 years</td>
</tr>
<tr>
<td><strong>Location of defect</strong></td>
<td>Herniation of abdominal contents from a defect lateral to the umbilicus (usually right sided) (Fig. 1)</td>
<td>Herniation of abdominal contents into the umbilical sac through a central defect of the umbilical ring (Fig. 2)</td>
</tr>
<tr>
<td><strong>Covering sac</strong></td>
<td>Absent</td>
<td>Present</td>
</tr>
<tr>
<td><strong>Condition of bowel</strong></td>
<td>Bowel wall is thickened, with a fibrin 'peel' due to exposure to amniotic fluid</td>
<td>Bowel looks normal</td>
</tr>
<tr>
<td><strong>Contents of hernia</strong></td>
<td>Only small and large bowel in most cases</td>
<td>May be a small 4–8 cm defect (exomphalos minor, with sac containing stomach and intestinal loops) or a large defect, including liver, with poorly developed abdominal and thoracic cavities and pulmonary hypoplasia (exomphalos major)</td>
</tr>
<tr>
<td><strong>Associated anomalies</strong></td>
<td>Rare, but majority of neonates are low birth weight</td>
<td>Cardiac defects, genitourinary abnormalities, Beckwith Wiedemann syndrome, Prune Belly syndrome, prematurity, IUGR, malrotation</td>
</tr>
</tbody>
</table>

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### Initial Management Goals

- Fluid resuscitation
- Minimizing heat loss
- Treating sepsis
- Avoiding direct trauma to the herniated viscous

### Preoperative Management

The immediate treatment is similar for both conditions. The abdomen or exteriorized intestine is wrapped with sterile gauze, and plastic wrap to minimize heat and fluid loss. A nasogastric tube decompresses the stomach, and prevents regurgitation, aspiration pneumonia and further bowel distension. No attempt should be made to reduce the contents into the abdomen. This maneuver may rupture the sac (in case of omphalocele), cause trauma to the viscous, interfere with venous return, and/or impede the infant’s respiratory effort. Intravenous fluid therapy with isotonic fluid, e.g. Ringer’s lactate or 0.9% normal saline at 3–4 times the usual maintenance rate (150–300 mL/kg/day) is started to compensate for the large evaporative losses. Acid base and electrolyte status should be closely monitored in view of large fluid requirements. Regular assessment of capillary refill, heart rate, urine output, core- periphery temperature gradients, and correction of acid-base disturbances will guide resuscitation. Glucose monitoring and maintenance is specially important because of the association with prematurity, IUGR. Antibiotics are administered to combat infection. Endotracheal intubation and ventilation may be necessary in case of respiratory distress.

Appropriate investigations (Chest X-ray, cardiac echo, renal ultrasound) should be performed to rule out associated abnormalities. Gastroschisis requires urgent surgery as the bowel is exposed and fluid and heat loss is significant; also gut circulation may be compromised with ischemia and infarction.

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**Fig. 1:** Gastroschisis, with intact umbilical cord. Bowel loops are thickened, matted, and covered with inflammatory peel

**Fig. 2:** Omphalocele. The sac is intact, and the umbilical cord is seen emerging from the mass
Defects without rupture of the membranous sac may be allowed to epithelialize using topical agents (silver sulfadiazine or antibiotics) and a silo, and the surgery may be deferred.\textsuperscript{23}

**Surgical Procedure**

Small defects can be closed by primary closure. If primary closure is not possible, a staged procedure is planned, including use of the “silo” consisting of a silastic or Teflon mesh that is used to cover the lesion, and then sutured to the fascia of the defect. Sterile urinary bags and intravenous fluid bags have been used to form an improvised silo (Fig. 3). After the silo is in place, it is suspended above the incubator, allowing gravity to ease the extra-abdominal organs in the silastic chimney into the peritoneal cavity over 4–7 days. Once reduced, surgical closure of the abdomen is performed.

‘Abdominal compartment syndrome’ may occur if the abdominal contents are reduced under pressure, particularly in exomphalos major with small abdominal cavity. There is an upward shift in the diaphragm interfering with ventilation, and a decrease in abdominal organ blood flow. Reduction in renal perfusion can result in oliguria or even anuria, vascular injury to the intestine may result in gut necrosis, and reduction in liver perfusion will result in hepatic impairment. Forceful closure of the abdominal wall will also cause significant tension of the skin resulting in a high incidence of necrosis with secondary infection. The feasibility of primary closure can be assessed intraoperatively by measurement of intragastric, bladder, and central venous pressure or changes in ventilatory mechanics. If intragastric pressures are >20 mm Hg or the inspiratory pressures are >30 cm H\textsubscript{2}O, staged surgery is required.\textsuperscript{24}

**Anesthesia Technique**

After aspiration of stomach contents, anesthesia is induced with inhalation or intravenous agents. Nitrous oxide is avoided to prevent further bowel distension. Maintenance is with oxygen-air and low concentrations of an inhalational anesthetic. Neuromuscular blockade facilitates reduction of eviscerated organs.

Opioids, e.g. fentanyl (3–4 µg/kg) or morphine (20–100 µg/kg), if postoperative ventilation is anticipated can be given for analgesia. A regional block, e.g. caudal epidural or TAP block provides good analgesia. An epidural or caudal catheter provides very good intra and postoperative analgesia and has the added benefit of abdominal wall relaxation. Hepatic clearance of local anesthetics may be reduced if there is any abdominal compression. Spinal anesthesia as a sole technique has been used successfully in patients with gastroschisis.\textsuperscript{25}

IV access in the upper limbs is preferable as abdominal pressure may restrict venous return temporarily post-operatively. Third space and evaporative loss is very large, 15–20 mL/kg/h of isotonic fluid may be required to maintain the intravascular volume.

**Postoperative Care**

Mechanical ventilation is often required for 24–48 hours due to temporary deterioration of lung function due to the raised intra-abdominal pressure. In neonates with placement of a silo or similar device, ventilation may be continued for an extended period. Those with a small defect may be extubated at the end of surgery. All patients must be monitored closely for complications such as abdominal compartment syndrome, bowel ischemia and respiratory compromise.

**Complications**

Postoperative ileus is common. It is prudent to establish appropriate intravascular access in the OR for parenteral nutrition, which may be required for days to weeks. Early complications include necrotizing enterocolitis, renal insufficiency, pneumonia, patent ductus arteriosus, cellulitis of the abdominal wall, abdominal wall breakdown, gastroesophageal reflux and cholestasis.

**Learning Points**

- Large volume fluid resuscitation is required
- Abdominal compartment syndrome must be watched out for
Intestinal Obstruction
Intestinal obstruction is a surgical emergency, requiring immediate intervention. Delay in diagnosis and treatment can result in significant abdominal distension, with respiratory compromise and risk of aspiration, and fluid shifts with disturbances in fluid and electrolytes. Intestinal perforation, bowel ischemia, necrosis and septicemia can occur if surgery is delayed.

Surgical Conditions Causing Obstruction
Duodenal obstruction is commonly associated with anomalies such as Down’s syndrome, cystic fibrosis, congenital cardiac defects, midline defects such as esophageal atresia and imperforate anus, and renal anomalies. Radiograph shows the classic ‘double bubble’ with dilated stomach and dilated proximal duodenum (Fig. 4). Neonates present with bilious vomiting, but have minimal abdominal distension. Delayed diagnosis can lead to dehydration, and hypochloremic alkalosis.

Jejunoileal atresia: These neonates are frequently preterm, however, other associated anomalies are infrequent. Radiographs show air-fluid levels throughout the abdomen (Fig. 5).

Meconium ileus is a luminal obstruction of the distal small bowel by abnormal meconium. This condition is found almost exclusively in patients with cystic fibrosis. Respiratory symptoms of cystic fibrosis are not present, since the obstruction manifests in the neonatal period. Medical management involves diatrizoate meglumine enemas, which loosen the meconium, facilitating its expulsion. Surgery involves an enterostomy to evacuate the meconium.

Malrotation and volvulus: Rotation of intestine around the mesentery causes compromise of the vascular supply. Neonates present with bilious vomiting, tender distended abdomen; bloody stools are an ominous sign. Immediate surgery is necessary, as delay may result in necrosis of the entire small bowel (Fig. 6).

Hirschsprung disease and Large bowel obstruction: Absence of parasympathetic ganglion cells creates a non-peristaltic segment of large bowel. The resultant functional obstruction leads to bowel distension; severe distension may result in bowel ischemia, perforation and peritonitis. Toxic megacolon syndrome results when enteric bacteria enter the blood stream. Affected patients present with severely distended tender abdomen, hypotension and need massive volume replacement and vasopressor support (Fig. 7).

Anesthesia Technique
Despite varied etiology, the implications for anesthesia management are similar in all these conditions. Initial stabilization is directed at correcting the hypovolemia and electrolytes. A nasogastric tube may reduce gastric distension, however, if the obstruction is below the duodenum, nasogastric aspiration may not be very effective in reducing the distention. Rapid sequence induction is carried out in hemodynamically stable conditions.
neonates, with normal airway anatomy. Awake intubation may be performed in very sick neonates or those who are actively vomiting. \( \text{N}_2\text{O} \) is avoided to minimize bowel distension. If the patient is volume depleted, inhalation agents are poorly tolerated. Neuromuscular blockade facilitates abdominal exploration, and also reduces the need for high concentrations of inhalational anesthetics.

**LEARNING POINTS**
- All precautions for ‘full stomach’ and prevention of aspiration should be taken
- Perioperatively, aggressive correction of fluid and electrolyte abnormalities is required

**Anorectal Malformations**

Anorectal malformations (Imperforate anus/anal atresia) can range from a mild stenosis to a complex syndrome with other associated anomalies, including genitourinary abnormalities and tethered spinal cord. Male babies generally need surgery immediately after birth to relieve the obstruction. In females, presence of a rectovaginal fistula prevents total bowel obstruction, so surgery can be deferred.

**Surgical Procedure**

Complexity of surgery varies from a simple perineal anoplasty (this may be curative in case of anal membrane), a temporary colostomy or an extensive abdominoperineal repair. The standard surgical approach is diverting colostomy performed in neonatal period, followed by definitive repair in infancy, and take down colostomy once the definitive repair has been performed. Primary repair without a colostomy, and laparoscopic assisted pull through are other options.\(^\text{26}\)

**Anesthesia Considerations**

- ‘Full stomach’ and risk of aspiration
- Marked abdominal distension, with impairment of ventilation
- Fluid and electrolyte derangements, due to third space loss
- Associated anomalies (VACTERL association)
- Sepsis, bowel perforation, peritonitis.

Anesthesia technique is similar to neonates with intestinal obstruction. A caudal block, in the absence of any contraindications, e.g. sacral vertebral anomalies, coagulopathy provides excellent analgesia. Large fluid shifts due to bowel manipulation, peritonitis are expected, and replacement with isotonic solutions at rates of 10 mL/kg or more may be required.

**Postoperative Care**

Neonates with sepsis, cardiovascular instability may need postoperative ventilation and cardiovascular support. Many neonates also need TPN.

**Necrotizing Enterocolitis**

This condition predominantly affects preterm babies, and is associated with a high mortality. The proposed physiologic mechanism is intestinal mucosal ischemia due to reduced mesenteric blood flow. Birth asphyxia, hypotension, RDS, intestinal ischemia, umbilical vessel cannulation, systemic infections and early feeding have been implicated as causative factors.\(^\text{27}\)
Clinical Presentation

Affected neonates appear very ill, with poor feeding, lethargy, temperature instability, vomiting, malabsorption, or overtly bloody stools. Examination may reveal a distended, tender abdomen. In fulminant cases, neonates rapidly progress to a state of hemodynamic instability and septic shock. Radiograph showing gas in the intestinal wall (pneumatosis intestinalis) (Fig. 8) and in the hepatobiliary tract or portal venous system is pathognomonic. Free gas under the diaphragm will be seen if perforation occurs.

Laboratory Investigations

Anemia, thrombocytopenia, coagulopathy are common. Metabolic acidosis and prerenal azotemia occur in severe cases.

Initial Management

Initial management includes discontinuation of oral feeds, decompression of the stomach, and administration of broad-spectrum antibiotics. Vasopressor infusion may be started to improve cardiac output and gut perfusion. Surgery is indicated when there is perforation or signs suggestive of bowel ischemia and necrosis.

Anesthesia Technique

A narcotic based anesthetic, rather than inhalational agent may be better tolerated by these very ill infants. Platelets and fresh frozen plasma are very often required to correct the coagulopathy. Large volume of fluid, as well as blood transfusion are required to manage generalized hemorrhage and third space losses. Placement of a central venous catheter for administration of pressors, fluids, antibiotics, and blood products is prudent.

Postoperative Care

Mechanical ventilation and cardiovascular support are required. These neonates have a prolonged hospital course, with long-term requirements for intravenous alimentation.

LEARNING POINTS

- NEC is associated with significant mortality and long-term morbidity
- Predisposing factors are prematurity, gut ischemia and infection
- Surgery is indicated for bowel perforation, gangrene
- Blood, blood products, cardiovascular support, and postoperative mechanical ventilation are universally required

Pyloric Stenosis

Pyloric stenosis, or infantile hypertrophic pyloric stenosis (IHPS), is a condition characterized by hypertrophy of the muscularis layer of the pylorus causing functional obstruction of the gastric outlet. It may be associated with other congenital abnormalities but usually affects healthy full term babies. The precise cause is unknown but there is a genetic component. The condition is more commonly seen in males (4:1), presenting typically between 2 and 6 weeks of age.

Clinical Presentation

The cardinal features are non-bilious projectile vomiting immediately after feeding, where the infant is hungry soon after vomiting. The child may present with clinical signs of dehydration if the diagnosis is delayed and vomiting prolonged for a number of weeks.

Pathophysiology

The classical picture is hypochloremic metabolic alkalosis due to loss of hydrochloric acid in vomited gastric fluid. The renal response to vomiting is twofold. Initially the kidneys excrete alkaline urine with sodium and potassium loss in an effort to retain hydrogen ions and maintain serum ph. Later, with depletion of these electrolytes, acidic urine is excreted (paradoxical aciduria) worsening the metabolic alkalosis. Persistent vomiting and dehydration may also result in either hyper- or hyponatremia. Pre-renal failure can occur, with hypovolemic shock and metabolic acidosis.
Diagnosis

Diagnosis is made on the basis of the typical history and palpation of an olive-like mass in the upper abdomen. Confirmation is done by ultrasonography, or rarely with a barium swallow and radiographic examination.

Management

Initial management is aimed at correction of dehydration, as well as electrolyte and acid-base abnormalities. An initial bolus of 20 mL/kg 0.9% saline is used if the infant is dehydrated. Thereafter, maintenance fluid is started, the purpose of which is to maintain adequate hydration whilst protecting against hypernatremia and hypoglycemia. An appropriate maintenance fluid is 5% glucose/0.45% saline. Potassium chloride is added to the fluid as required once urine output has been established. An accurate fluid balance chart should be kept and urinary catheterization should be considered if the child is dehydrated. Serial electrolyte, acid-base and blood glucose measurements will guide appropriate therapy. A nasogastric tube is passed to decompress the stomach. Most infants respond to therapy within 24–48 hours after which surgery can be conducted.

Surgical Procedure

The classical operation is a Ramstedt pyloromyotomy; a longitudinal incision of the pylorus with blunt dissection to the level of the submucosa, relieving the gastric outlet obstruction. Pyloromyotomy may also be performed laparoscopically with the advantages of reduced analgesia requirements, a more rapid return to enteral feeding and shorter hospital stay.

Anesthesia Goals

- Preoperative stabilization with treatment of dehydration and metabolic abnormalities
- Prevention of pulmonary aspiration of gastric contents.

Anesthesia Technique

The stomach is aspirated immediately before induction using a wide bore gastric tube. Suctioning with the infant in the left and right lateral positions, in addition to the supine position ensures near complete emptying of gastric contents.

Anesthesia is most often induced with intravenous agents, followed by a rapid-acting muscle relaxant, succinylcholine or rocuronium. Gas induction has also been reported without any adverse sequelae. Anesthesia is maintained with volatile agents carefully titrated to effect, since the surgical procedure is brief and these patients are prone to delayed recovery from anesthesia. Extubation is carried out in the operating room when the infant is fully awake and vigorous.

Postoperative pain is not usually severe, and good analgesia may be achieved intra-operatively with an intravenous opioid such as fentanyl and paracetamol. Ultrasound guided rectus sheath block may provide superior perioperative analgesia compared with infiltration at the surgical site.

The risks of anesthesia in the baby who is inadequately resuscitated are: hypotension due to hypovolemia, potentiation of muscle relaxants because of hypokalemic alkalosis and hypovolemia, and postoperative apnea because of a compensatory respiratory acidosis.

LEARNING POINTS

- IHPS is a common condition, presenting typically between 2–6 weeks of age
- Initial management is aimed at resuscitation, correcting of dehydration, alkalosis and electrolyte disturbances before definitive surgery

Tracheoesophageal Fistula and Esophageal Atresia

Tracheoesophageal fistula (TEF) is a congenital anomaly that is characterized by a connection between the esophagus and the trachea, with or without a disruption of the esophagus. An isolated disruption of the esophagus is termed esophageal atresia (EA).

Coexisting congenital abnormalities occur in 50% of patients. VACTERL association (vertebral abnormalities, imperforate anus, congenital heart disease, tracheoesophageal fistula, renal and limb abnormalities) may coexist in various combinations. Non-VACTERL anomalies include gastrointestinal defects (duodenal atresia, imperforate anus, malrotation, pyloric stenosis, omphalocele), respiratory tract anomalies (tracheobronchomalacia, pulmonary hypoplasia), association with CHARGE and Potter’s syndrome, and chromosomal abnormalities.

Anatomy of TEF

The anatomical classification was pioneered by Vogt, and modified by Ladd and Gross (Fig. 9). Commonest is type C (90%), with upper blind esophageal pouch, and fistula between lower esophageal segment and trachea.
Clinical Presentation and Diagnosis

Prenatal ultrasonographic suspicion is based on polyhydramnios and absent or small stomach bubble. Newborns present with excessive oral secretions, coughing and choking, regurgitation of feeds, cyanosis and respiratory distress exacerbated by feeding. Sometimes, in H type fistula, the diagnosis may be missed and recurrent pneumonia is the presenting symptom. Inability to pass an orogastric tube into the stomach, and chest radiograph showing a nasogastric tube curled up in the upper chest or neck confirm the diagnosis (Fig. 10). A pure EA with no fistula will present with a scaphoid abdomen with no gastric bubble on X-ray. A TEF presents with an inflated enlarged gastric bubble on X-ray. Contrast studies are not necessary as the risk of chemical pneumonitis is high.

Preoperative Management

Immediately after diagnosis, measures are promptly instituted to prevent aspiration and to stabilize the neonate.

- Avoid feeding
- Nurse in semiupright position
- Intermittent suctioning of upper pouch/Replogle tube (sump suction catheter) in upper pouch connected to constant suction
- IV fluids to prevent/correct dehydration and hypoglycemia
- Antibiotic therapy to treat sepsis or aspiration pneumonia.

Echocardiographic examination should be performed to demonstrate any cardiac or vascular abnormality that could affect anesthetic management or surgical approach. Thoracotomy is usually performed opposite to the side of the aortic arch.

Surgical Procedure

Surgery is urgent, and performed within 24 to 72 hours in otherwise healthy neonates. Delay in surgical correction increases the risk of aspiration of saliva as a result of accumulation in the upper esophageal pouch. Reflux of gastric acid through the lower pouch and a TEF can cause pneumonitis.

![Fig. 9: Anatomical classification of tracheoesophageal fistula]
Surgical approach for open repair is through a right thoracotomy, unless the aortic arch is right sided. A total repair involves ligation of the fistula and primary anastomosis of the esophagus. If the gap between the proximal and distal esophagus is too wide, then following fistula ligation, a gastrostomy and exteriorization of upper pouch through an esophagostomy is performed. The definitive repair is then deferred till a later date, depending on the infant’s growth and cardiorespiratory status. A staged repair is an alternative for neonates that are unable to tolerate surgery due to pneumonia and/or other congenital anomalies.

Thoracoscopic repair is being performed in some centers, and adds to the complexity of anesthesia. Preoperative cardiopulmonary evaluation can help determine whether one-lung ventilation will be tolerated by these neonates. CO2 is insufflated to collapse the lung on the operative side. During one-lung ventilation, endtidal CO2 is falsely low, and arterial blood sampling permits monitoring of ventilation.

The Waterston risk stratification system is useful for timing surgery (Table 2).

<table>
<thead>
<tr>
<th>Category</th>
<th>Weight/Comorbidities</th>
<th>Surgical Timing</th>
<th>Survival (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Category A</td>
<td>&gt; 2500 gram</td>
<td>Can undergo surgery</td>
<td>100</td>
</tr>
<tr>
<td>Category B</td>
<td>1800–2500 gram or pneumonia or congenital anomaly</td>
<td>Short term delay, needs stabilization prior to surgery</td>
<td>95</td>
</tr>
<tr>
<td>Category C</td>
<td>&lt; 1800 gram or severe pneumonia or congenital anomaly</td>
<td>Requires staged repair</td>
<td>35</td>
</tr>
</tbody>
</table>

Spitz designed a prognostication index depending on birth weight and presence or absence of cardiac disease.38
- Birth weight >1500 g and no major cardiac anomaly: 98% survival.
- Birth weight <1500 g or major cardiac anomaly: 80% survival.
- Birth weight <1500 g and major cardiac anomaly: 50% survival.

**Anesthesia Concerns**
- Ineffective ventilation, with gastric distension
- Respiratory distress due to aspiration, prematurity
- Coexisting anomalies, especially cardiac defects.

**Anesthesia Technique**
Anesthetic management depends on patient comorbidity, type of defect, and local hospital practice. A wide range of anesthetic techniques have been used.39-43

The upper pouch tube is aspirated and then removed. Induction with sevoflurane and intubation during spontaneous ventilation is preferred by many as it avoids the need for positive pressure ventilation. The negative intrathoracic pressure generated during spontaneous ventilation causes gas to preferentially enter the lungs rather than the fistula. It also allows the option of wake-up if there is major difficulty with ventilation. Conversely, pre-intubation neuromuscular blockade offers the benefit of optimal airway conditions without coughing or straining. Usually ‘gentle bagging’ will ventilate the lungs, unless the fistula is large or the lung compliance very low. A rapid acting muscle relaxant, e.g. succinylcholine or rocuronium can be used for neuromuscular blockade. Specific ventilation strategy is less crucial in patients with small fistula.

Some anesthetists still prefer an awake intubation technique to safely position the endotracheal tube (ETT). Avoidance of positive pressure ventilation will prevent gastric insufflation. Titrating small doses of fentanyl (0.5-1 µg/kg) or morphine (0.05-0.1 mg/kg) can permit tracheal intubation without hemodynamic compromise.

Rigid bronchoscopy prior to surgery can help assess the location and size of the TEF, to exclude multiple TEFs, to place a Fogarty catheter to either aid in surgical identification of the TEF or to occlude the TEF to aid ventilation, and to assess other potential airway problems such as tracheomalacia that may impact the decision for extubation.44,45 Most surgeons reserve preoperative bronchoscopy for neonates with a confusing clinical picture.
To correctly position the ETT, an intentional right main stem intubation is sometimes performed, and the tube is then slowly withdrawn till breath sounds are bilaterally equal. This position of the tube, just above the carina, and beyond the fistula, ensures that ventilation will not inflate the stomach in most cases. Fibreoptic bronchoscopy can aid correct positioning of the ETT, obviating the need for rigid bronchoscopy.

A preoperative gastrostomy was used in the past to manage stomach distension due to ventilation preferentially passing through the fistula (the path of least resistance, especially when the lung compliance is poor). However, although this may prevent gastric rupture due to over distension, it results in effective ventilation as a result of the bronchocutaneous fistula. Gastric insufflation can be better managed by needle decompression of the stomach, followed by urgent surgery.

Strategies to ensure adequate ventilation include one-lung ventilation, tracheal intubation distal to fistula or Fogarty catheter occlusion of the fistula. One-lung ventilation facilitates surgical exposure, but may result in an unacceptable fall in oxygen saturation. If a staged gastrostomy has been performed, a Fogarty catheter passed retrograde through the gastrostomy has been used to block the esophagus. Precise positioning of the balloon is difficult to perform and maintain, and migration of the catheter into the trachea can result in compromise of ventilation.

Usually, the neonate is positioned for a right thoracotomy in a lateral position, with the right arm raised across the head. Use of appropriate padding, tapes, and gel blocks ensure secure and safe positioning of the patient.

Lung retraction during surgery can cause steep falls in oxygen saturation and interfere with ventilation. Prematurity, preexisting pneumonia and congenital heart disease increase the risk of hypoxemia. Management measures include increasing inspired oxygen concentration and gentle manual ventilation, while asking surgeon to stop retraction. Hypoxemia may also result from intubation of the right main stem bronchus, and endotracheal tube obstruction by secretions or blood.

Methods for analgesia include use of opioids, caudal catheters, intercostal blocks, paravertebral blocks, local infiltration and paracetamol.41

Monitoring
In addition to standard monitors, invasive arterial monitoring is indicated in patients with complex congenital heart disease or pulmonary disease, and in patients undergoing thoracoscopic surgery. A precordial stethoscope taped to the left axilla (during a right thoracotomy) can provide valuable information. Confirmation of breath sounds on left side ensures that the dependent lung is ventilated, and that the ETT has not migrated. Prior to ligating the fistula, the surgeon test clamps the fistula; auscultation of breath sounds on the left side confirms that the left bronchus has not been accidentally clamped. Pre and postductal saturation monitoring with two pulse oximeters can provide information regarding shunting.

Postoperative Care
Early extubation is preferred to minimize the time that the repair site is exposed to the pressure of the ETT and ventilation. Decision regarding postoperative extubation is based on patient prematurity and size, associated comorbidity, perioperative status, ease and duration of surgery, tension of esophageal anastomosis, ease of intraoperative ventilation, and presence of tracheomalacia. Opioid sparing techniques, and thoracoscopic surgery facilitate earlier tracheal extubation.46

Complications
Surgical postoperative complications include anastomotic leak, stricture, gastroesophageal reflux, tracheomalacia, and recurrent TEF.

LEARNING POINTS
- Preoperative stabilization is warranted prior to surgery
- Coexisting cardiac defects are common
- Risk stratification systems are useful for timing surgery and to predict outcome
- A wide range of anesthetic techniques have been used for induction and airway management

Congenital Diaphragmatic Hernia
Congenital diaphragmatic hernia (CDH) is a defect in the diaphragm that occurs during development, associated with intrusion of intra-abdominal organs into the thoracic cavity. Most common defect is the posterolateral (Bochdalek’s hernia) occurring in 90%, majority of these are left sided. The other types are anterior (Morgagni hernia), paraesophageal hernias and eventrations. A small percentage may be bilateral.47

Association with Other Anomalies
Associated anomalies such as congenital heart disease, genitourinary or gastrointestinal malformations and chromosomal anomalies occur in 40–60% of patients.
There may be a syndromic association with Beckwith-Wiedemann, CHARGE, Cornelia de Lange, and Denys-Drash syndromes.

**Pathophysiology**

The herniated abdominal contents occupy the thorax, and interfere with the development of the lungs. Varying degrees of pulmonary hypoplasia occur depending on when the herniation and compression occurred during fetal development. The herniated abdominal contents shift the mediastinum to the opposite side, and thereby interfere with the development of the contralateral lung. Severe morphologic derangements in lung development occur, with the lungs demonstrating fewer alveoli with thickened walls, smaller area for gas exchange, decreased pulmonary vasculature with medial hyperplasia of the pulmonary arteries, and altered arteriolar reactivity, causing pulmonary hypertension. Decreased pulmonary blood flow prevents normal transition of the neonate from the intrauterine to the extra uterine circulatory pattern. Right-left shunting and resultant hypoxemia poses an immediate threat to the newborn.

**Prenatal Diagnosis and Treatment**

Ultrasound scan in the second trimester shows polyhydramnios, stomach or loops of bowel within the thoracic cavity, and mediastinal shift away from the side of the lesion.

Intrathoracic position of the liver, lung-to-head circumference ratio (LHR) less than 1, and lung-to-thorax transverse area ratio (L/T ratio) less than 0.08 indicate poor prognosis. The size of the diaphragmatic defect is a reliable surrogate marker of underlying pulmonary hypoplasia and poor outcome. Right-sided defects and bilateral defects have poor outcome. Other proposed predictors are fetal lung volumetry, fetal body volume, left ventricular mass and pulmonary artery diameters as determined by fetal MRI.

Various fetal surgical procedures have been developed to improve the primary pathology, i.e. pulmonary hypoplasia. Surgical occlusion of the fetal trachea results in gradual distension of the hypoplastic lung by preventing the normal efflux of intraluminal lung liquid. Open tracheal occlusion (EXIT) procedure has been superseded by a fetoscopic approach (fetal endoluminal tracheal occlusion, FETO), with the occlusive balloon being removed a few weeks before delivery. However, these procedures are still under investigation, with a recent randomized, controlled trial of tracheal occlusion in fetuses with no liver herniation demonstrating no survival benefit over standard postnatal therapy.

**Postnatal Diagnosis and Management**

The classical triad of CDH consists of cyanosis, dyspnea and apparent dextrocardia. Neonates born with severe pulmonary hypoplasia will demonstrate symptoms immediately after birth. Physical examination will reveal a scaphoid abdomen, bulging chest, reduced breath sounds, bowel sounds in the chest and heart sounds deviated towards contralateral side. Chest radiograph shows bowel gas in the chest and mediastinal shift (Fig. 11). Neonates with Morgagni hernia may present with less severe respiratory symptoms, but with symptoms of bowel obstruction.

**Preoperative Stabilization and Timing of Surgery**

Preoperative care of the neonate with severe CDH should start in the delivery room. Positive pressure ventilation by bag-mask can damage the hypoplastic lung by over distension, and can distend the intrathoracic stomach and bowel. If spontaneous ventilation is inadequate, then the neonate should be immediately intubated and ventilated. A large bore nasogastric tube is passed to decompress the gut. Once these initial procedures have been performed, the neonate is transferred to the pediatric intensive care unit.

In the past, CDH was considered an emergency, and surgery was performed urgently. However, repair of the defect does not result in an improvement in gas exchange, and thoracic compliance and oxygenation and ventilation parameters tend to deteriorate in the...
extracorporeal membrane oxygenation (ECMO) has been used as rescue therapy in neonates who have persistent ductal hypoxemia despite ventilatory (including HFOV, NO) and hemodynamic support. Risks with ECMO are considerable, and include bleeding at surgical site or at chest drain site, intracranial bleeding, and sepsis. Since the likelihood of intracranial hemorrhage is higher in low-birth-weight and premature neonates, use of ECMO is limited to infants >2 kg, born higher than 35 weeks gestation, with no evidence of intracranial hemorrhage.

Surgical Procedure

Open repair is usually achieved via an abdominal incision with gentle reduction of the abdominal viscera from the thorax. The diaphragmatic defect is closed by primary repair or, in the case of a large defect, using a prosthetic patch. If there is difficulty closing the abdominal wound because of adverse changes in thoracic compliance, a prosthetic patch may also be incorporated into the abdominal wall. Thoracoscopy for repair of CDH is now performed at many centers. Careful patient selection is important, as pulmonary dysfunction limits the ability of the neonate to tolerate thoracoscopy and one-lung ventilation.

Anesthesia Technique

In most cases, neonates with moderate to large CDH are on mechanical ventilator support preoperatively. The goals of ventilation in the intraoperative period are similar to that of the preoperative period; small tidal volume, PEEP to prevent atelectasis, adequate oxygenation without hyperoxia and permissive hypercapnia, while maintaining pH >7.25. Volume status needs to maintained to minimize right-left shunt and maintain hemodynamics. Special care needs to be taken to maintain normothermia, as hypothermia increases PVR and can worsen right-left shunting. A sudden deterioration in ventilatory or hemodynamic parameters could occur due to a contralateral pneumothorax. A high index of suspicion must be maintained and immediate needle decompression followed by chest tube placement undertaken to manage the condition.

Nitrous oxide is avoided as it can diffuse into the bowel and worsen the lung compression. Anesthesia is maintained with low concentrations of inhalational agents (Sevoflurane or Isoflurane). These agents may have favorable effects on the pulmonary circulation by causing pulmonary vasodilatation; however, if the SVR falls more than the PVR shunting might worsen. Alternatively, the

Immediate postoperative period. Most centers now delay surgery for at least 24–48 hours, to allow for a period of clinical stabilization and a fall in pulmonary vascular resistance. Surgery may be delayed further if the infant has significant pulmonary hypertension despite appropriate treatment; there is no evidence that timing of surgery affects outcome.

The goal of initial management is to avoid a surgical intervention when the neonate is hypoxic, acidic and hemodynamically unstable. Medical management is directed towards stabilizing the cardiorespiratory function by improving oxygenation, correcting metabolic acidosis, reducing right-left shunting and improving pulmonary perfusion.

Current ventilator strategies focus on methods of improving oxygenation while avoiding injury to the hypoplastic lung. These include limiting the peak inspiratory pressures, maintaining a preductal SaO2 of >85%, and tolerating hypercapnia. The range of ‘acceptable’ peak inspiratory pressure with conventional mechanical ventilation is wide, ranging from 25 to 35 cm H2O; ‘acceptable’ mean airway pressures range from 12 to 15 cm H2O. Permissive hypercapnea is acceptable up to a pCO2 of 60–65 mm Hg. Spontaneous respiration is encouraged while the pH is maintained at more than 7.25 using sodium bicarbonate if necessary, since metabolic acidosis will worsen shunting. Surfactant therapy seems to offer limited benefit. Sedation with opioids and benzodiazepines allow the neonate to tolerate mechanical ventilation, as catecholamine response to airway instrumentation will further increase the PVR. Volume optimization and inotropic support is used to maintain hemodynamics.

High frequency oscillatory ventilation (HFOV) has been used both as rescue therapy when conventional ventilation fails, and as the primary modality of ventilation. The rationale for HFOV is its ability to preserve end expiratory lung volume while avoiding over distension, thereby limiting ventilator induced lung injury. HFOV can provide adequate gas exchange when using mean airway pressures lower than 15 cm H2O. Surgical repair has also been successfully performed while using HFOV.

Management of persistent pulmonary hypertension remains a challenge. A variety of therapeutic modalities have been used in an attempt to manage reversible pulmonary hypertension while ‘gentle ventilation’ is used. These include inhaled nitric oxide, sildenafil, prostacyclins, prostaglandins and nonspecific endothelin inhibitors, all of which act through different second messengers and have been used alone or in combination with varying success.
neonate may be maintained on opioid infusion (Fentanyl), which is generally continued into the postoperative period.

It is important to keep a watch for abdominal compartment syndrome, with compression of the inferior vena cava and reduction in venous return. Venous access sites should be secured in the upper extremities. Stepping up of monitoring with internal jugular vein cannulation for monitoring central venous pressure and arterial line for invasive blood pressure measurement and blood gases is recommended.

Complications

Despite several management strategies, overall survival in CDH remains low, with widely varying mortality rates across institutions. The optimal therapy remains unclear. Survivors often have chronic pulmonary disease, gastroesophageal reflux, growth retardation, developmental delay, and neurocognitive defects.68,69

**LEARNING POINTS**

- Stabilization prior to surgery is currently considered the best practice
- Hemodynamic and ventilator support and correction of metabolic disturbance are invariably required
- ‘Gentle ventilation’; HFOV, inhaled nitric oxide and ECMO improve oxygenation without causing pulmonary barotrauma

Inguinal Hernia

Inguinal hernia is common in the premature neonate; approximately 1/3rd of preterm babies are afflicted compared to 3–5% incidence in term newborns. The continued patency of the processus vaginalis is the principal factor in occurrence of congenital inguinal hernias.

The hernia should be repaired as early as possible to reduce the risk of bowel obstruction and incarceration.70,71 However, these risks must be balanced against the risk of potential operative and anesthetic complications. Younger corrected gestational age is associated with a greater risk of apnea.72

**Surgical Procedure**

Inguinal hernia repair is one of the most common procedures performed by pediatric surgeons. Laparoscopy has been advocated to examine the contralateral side for a patent processus vaginalis.73 The timing of preterm infant inguinal hernia repair varies widely in practice, with surgeons repairing hernias just before discharge from the NICU, at a specific corrected gestational age, or when it is convenient.74

**Anesthesia Technique**

Herniorrhaphy is generally performed as an elective procedure. However, in neonates with signs of bowel obstruction or incarceration an emergency surgery with a rapid sequence induction of anesthesia may be required. For elective hernia repair, anesthesia can be induced by inhaled anesthetics via facemask or by intravenous technique. For open hernia repair, an LMA can be safely used as an alternative to the endotracheal tube. Adequate depth of anesthesia must be maintained while the spermatic cord is handled, as laryngospasm and bradycardia can occur with lighter planes. Ilioinguinal/iliohypogastric nerve block or caudal epidural block provides excellent pain relief. In premature neonates, surgery under spinal or caudal epidural block, without general anesthesia and endotracheal intubation has been advocated to reduce the risk of postoperative apnea.75

In the premature neonate with bronchopulmonary dysplasia, general anesthesia may exacerbate the BPD making tracheal extubation difficult. In this situation, a sole regional technique is advantageous.

**LEARNING POINTS**

- Inguinal hernias are common in neonates born preterm.
- A sole regional technique reduces the risk of postoperative apnea

Sacrococcygeal Teratoma

Sacrococcygeal teratomas (SCTs) are common congenital tumors. They are germ cell tumors arising from the tip of the coccyx and vary greatly in size and the amount of internal and external extension. 10% of SCTs are malignant by the time the neonate reaches 2 months of age, and 50% are malignant by 1 year. Although not emergent, the resection is performed early in the neonatal period (Fig. 12).

**Pathophysiology**

Perinatal mortality is high, and causes are high output cardiac failure due to the rich blood supply of the tumor, preterm delivery due to polyhydramnios, anemia due to hemorrhage into the tumor, dystocia and tumor rupture. A vascular steal phenomenon may occur, with a large part of the fetal cardiac output going to the tumor, rather than the fetus. Fetal surgery has been performed in fetuses that are at risk of significant secondary morbidity.76 For large tumors, planned cesarean delivery is necessary.
Anesthesia Technique

Surgical treatment involves complete resection of the tumor along with the coccyx. Surgery is performed with the patient in the prone position, and may be difficult and lengthy due to the mass effects of the tumor. Anesthesia concerns include potential for massive hemorrhage, cardiovascular instability, hypothermia, and coagulopathy. For large tumors, adequate vascular access for transfusion and monitoring (arterial line, central venous line) are essential.

Complications

Perioperative morbidity is related to massive blood loss and coagulation dysfunction. Long-term complications include urologic problems and incontinence.

LEARNING POINTS

- Sacrococcygeal teratoma is the commonest congenital neoplasm
- Excision of large tumors is associated with massive blood loss

Myelomeningocele

The incidence of myelomeningocele (MMC) is 1 in 3000-4000 live births. It commonly occurs in the lumbosacral region, but can occur at any level in the neuraxis.

Pathophysiology

It results from failure of the posterior neural tube closure in the fetus around 28 days of gestation. Failure of closure of caudal end results in either spina bifida characterized by defects of the vertebral bodies; or meningocele characterized by herniated sac containing meninges; or myelomeningocele characterized by a sac containing neural elements. The sac containing cord structures is just covered by thin skin layer or has no cover besides dura mater. MMC is usually associated with Arnold-Chiari malformation (most commonly Type II) and hydrocephalus. Chiari II malformation is defined as the caudal herniation of the vermis, brainstem, and fourth ventricle through the foramen magnum. These patients may present with features of brainstem compression such as apnea, bradycardia, vocal cord palsy causing stridor, autonomic instability, abnormal respiration, swallowing dysfunction, nystagmus, torticollis, hypotonia and spasticity. Children with MMC have a higher incidence of intestinal malformations, renal anomalies, orthopedic abnormalities (scoliosis, club foot), cardiac malformations and tracheoesophageal fistula.

Surgical Procedure

Surgery for closure is usually undertaken within 24–48 hours after birth to reduce the risk of infection and further neurological damage. The spinal cord below the defect of MMC is often tethered, which over a period of time results in distal neurological defects. Untethering of the cord needs Electromyography (EMG) monitoring to identify functional nerve roots. Surgery is carried out in complete prone position.

Anesthesia Technique

Positioning for induction and intubation in order to avoid physical trauma to the neuroplaque can be challenging. Covering the MMC with sterile towel and positioning the patient’s back on a doughnut ring cushion is recommended. Alternately, intubation can be done with the baby in lateral position. Right-handed anesthesiologists often find it easier to intubate with the patient in left lateral position, especially when using a Macintosh blade for laryngoscopy. After inhalational or IV induction, endotracheal tube should be taped securely and maintenance can be done with inhalational agent and opioids. Muscle relaxant can be used if surgeon does not expect to use nerve stimulator. For prone position, routine precautions should be taken to avoid pressure on the abdomen, eyes, nose, ears and genitalia.

The intraoperative goal is to maintain normovolemia, and thus hemodynamic stability. Normal saline is the most commonly administered crystalloid as it is mildly hyperosmolar and hence prevents cerebral edema. Glucose-containing fluids are traditionally avoided in...
neurosurgical procedures as hyperglycemia worsens reperfusion injury. However, in neonates and premature infants, the danger of hypoglycemia should be borne in mind. Blood glucose should be monitored closely, along with continuous infusion of glucose at 5–6 mg/kg/min. Blood loss may be significant for large defects and if surgery involves undermining of large area of skin and fascia to achieve primary closure. Accurate estimation of blood loss can be challenging.

**Complications**

Perioperative respiratory complications such as hypoventilation, apnea, bronchospasm, laryngospasm, prolonged breath holding are common as a result of structural derangement of respiratory control center or in its afferent and efferent pathways.

Brainstem compression and coning can cause cardiac complications including bradycardia and even cardiac arrest when Chiari malformation is associated with MMC. Hypotension can occur due to sudden loss of CSF from a large MMC leading to increased craniospinal pressure gradient and brain herniation. Intravenous fluid replacement of the calculated deficit may reduce the incidence of hypotension. There may be sudden blood loss from an abnormal vessel or occurrence of venous air embolism. Hypothermia is frequent, due to the surface area of tissue exposed.

Postoperative complications include hydrocephalus, CSF leak, wound infection, and wound dehiscence. Lifelong disabilities including paraplegia, incontinence, sexual dysfunction and mental impairment.

There have been advances in intrauterine fetal MMC repair, but fetal endoscopic surgery while associated with spinal segmental neuroprotection, results in complications such as choioamnionitis, premature rupture of the amniotic membranes, and oligohydramnios.

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**LEARNING POINTS**

- Arnold-Chiari malformation and hydrocephalus are commonly associated with meningomyelocele
- Surgery is performed within 24–48 hours of birth to prevent infection and limit neurologic damage

**REFERENCES**


Anesthesia for Ear, Nose and Throat Procedures in Children

INTRODUCTION

Ear, nose and throat surgeries are some of the most commonly performed procedures in infants and children. They may be diagnostic or therapeutic in nature. Understanding the concepts and learning to administer safe anesthesia for these surgeries form an important part of post-graduate training in anesthesiology. In this chapter we have attempted to present the anesthesia considerations for frequently performed procedures like adenotonsillectomy, grommet insertion, etc. and also for complex surgeries like laryngotracheal surgeries performed in tertiary care centers.

ANESTHESIA FOR EAR SURGERY

Ear surgeries are commonly performed under operating microscope and include the following:

Myringotomy

Myringotomy with tympanostomy tube placement is a surgical procedure performed for fluid drainage from middle ear following persistent middle ear effusions due to acute or chronic otitis media, eustachian tube dysfunction and/or chronic upper respiratory tract infection (URTI). A small incision is made in the tympanic membrane, usually in both ears. Ear tubes (grommet) or tympanostomy tubes are small tubes open at both ends that are inserted into the incisions in the eardrums during myringotomy to allow ventilation of the middle ear.

Anesthesia Concerns

The child undergoing this procedure is usually a healthy child, but may have associated respiratory tract infection, craniofacial anomalies, and/or obstructive airway problems requiring special care. Generally, it is a short general anesthesia (GA) procedure in expert hands, done on an outpatient basis, under face mask or laryngeal mask airway (LMA). The patency of the airway is to be ensured with the head turned to either side. Endotracheal tube (ETT) may be required in some cases. Standard monitoring and postoperative care is necessary. Pain can be treated with paracetamol or non-steroidal anti-inflammatory drugs (NSAIDs).

Above procedure is at times performed along with adenoidectomy and/or tonsillectomy.

Myringoplasty, Tympanoplasty and Mastoidectomy

Myringoplasty involves repair of a tympanic membrane perforation in a dry ear. Tympanoplasty is performed when there is extensive middle-ear damage and involves reconstruction of the tympanic membrane and the ossicular chain. The approach to the ear can be permeatal or post-aural. Mastoidectomy is performed to
eradicate chronic suppurative middle-ear disease. Above procedures require GA with controlled ventilation.

**Anesthesia Considerations**

- Careful rotation of the head and neck while positioning the patient under GA to avoid the risk of atlanto-axial rotational subluxation.²
- The patient’s arm is tucked adjacent to their body with adequate padding to protect ulnar nerve and to facilitate the surgeon’s work. An extension tubing for intravenous line is helpful.
- Muscle relaxants are to be avoided in patients requiring facial nerve monitoring. Deep levels of anaesthesia will be required to achieve intermittent positive pressure ventilation (IPPV) without relaxants.
- A head-up tilt of 10°–15°, epinephrine infiltration, avoidance of tachycardia, relative hypotension [mean arterial pressure (MAP) 10–20% <normal] and control of the end-tidal CO₂ can minimize bleeding during surgery.
- Nitrous oxide (N₂O) is avoided during tympanoplasty as it accumulates in closed gas space, increases middle ear cavity pressure and lifts the graft away from its new site.³

**Preanesthetic Concerns**

- Communication with the child may be problematic and a parent can be allowed to accompany the child
- Child may read lip movements so our masks should be kept down
- Surgery takes 2–3 hours and anesthetic considerations are the same as for mastoidectomy
- Monitoring of the facial nerve requires timing of the muscle relaxant or avoiding it totally.

**ANESTHESIA FOR RHINOLOGIC PROCEDURES**

Common rhinologic surgeries include nasal polypectomy, functional endoscopic sinus surgery (FESS), rhinoplasty, septoplasty, and foreign body removal. Common anesthetic considerations are as follows:

**Preoperative**

- Varying degrees of nasal obstruction and nasal discharge may be caused by polyps, deviated septum, or mucosal congestion and make the child an obligatorily mouth breather. Mask ventilation may be difficult especially in obese children, and those with Obstructive sleep apnea syndrome (OSAS) and/or maxillofacial deformities.
- Nasal pathology is often associated with allergic disorders and gastroesophageal reflux disease (GERD). Nasal polyps are common with cystic fibrosis.
- Coagulation problems must be ruled out as nasal mucosa is very vascular.
- Child/caregiver must be informed about nasal packing and the need to breathe through the mouth postoperatively.

**Intraoperative**

- Topical oxymetazoline or epinephrine pack can be used for local vasoconstriction and for reducing blood loss
- GA with regular or Ring, Adair, Edwin (RAE) tube (preferably), pharyngeal packing and eye protection
- Oral airway during mask ventilation to mitigate nasal obstruction
- Sevoflurane/isoflurane preferred, though 10 μg/kg of epinephrine, 1:200,000 solution (5 μg/mL, maximum 2 mL/kg) has been used with halothane without apparent myocardial irritability.⁵ Careful monitoring during and after epinephrine injection is mandatory

**Cochlear Implantation**

It is a feasible choice of surgery in children with severely impaired hearing having intact auditory nerve fibres but absent sensory neuroepithelium in the cochlea. The causative factors are genetic, infection and/or cochlear ossification.⁸

The implant has 8–22 electrodes and is placed through a cochleostomy. An external microphone receives and converts sound into electrical signals which are sent via electrodes to cells adjacent to the auditory nerve. The cochlea attains its full size at birth; hence electrode placement even in small children does not pose any problem.
• Must confirm pharyngeal pack is removed before extubation; also inspection of the pack to evaluate blood loss
• Extubation is carried out when child is completely awake and after return of airway reflexes.

Postoperative Concerns
• Nasal packing may affect ventilation and oxygenation. Packing around a nasopharyngeal airway helps to maintain nasal patency. Oxygen supplementation, and monitoring are mandatory
• Postnasal trickling of blood is followed by swallowing or risk of aspiration; and can be avoided by cautious use of analgesics, especially narcotics.

REDUCTION OF NASAL BONE FRACTURE
Nasal bone fractures usually result from trauma and could be associated with facial, head and other body part injuries.
• Associated head injury must be ruled out preoperatively
• Nasal fracture can cause considerable bleeding and blood may be swallowed: consider patient with full stomach for urgent or emergency surgery, and GA with ETT is mandatory to avoid aspiration of blood into the lungs
• LMA (preferably flexible type as it does not come in the way of surgery) can be used if the surgery is a scheduled procedure.10

NASAL POLYPECTOMY
Nasal polyps often occur in children having cystic fibrosis.11 They are usually multiple and often recur after removal. Cystic fibrosis is usually associated with obstructive lung disease with recurrent pulmonary infection and thick secretions.

Special Anesthetic Considerations
• Preoperative optimisation with nebulisation, physiotherapy, and bronchodilators
• Monitor for hypoxemia, ventilation and rise in airway pressure (AWP). May require recurrent endotracheal lavage and suction
• Avoid N₂O and give positive end-expiratory pressure (PEEP) of 5–7 cm H₂O to prevent atelectasis. Avoid NSAIDs as polyposis could be a part of Samter’s triad (polyposis with asthma and aspirin sensitivity).13

ENDOSCOPIC SINUS SURGERY
Endoscopic sinus surgery (ESS) is performed in children with chronic or recurrent sinusitis as a last resort when medical treatment followed by adenoidecetomy with or without tonsillectomy has failed.14 ESS focuses on enlarging the natural ostia of the maxillary and ethmoid sinuses for better drainage while preserving the sinus mucosa.14 Preoperatively CT scan may be required to delineate the pathology.

Tachycardia and hypertension should be avoided, with adequate depth of anesthesia and analgesia to achieve a good surgical field. Additionally, infusion of dexmedetomidine or propofol can be used to achieve this end.

CHOANAL ATRESIA
It is a congenital deformity with absence of connection between the nasal cavity and aerodigestive tract15 and causes upper airway obstruction. Incidence is 1:7000 births. Atresia is bony in 30% or mixed (membranous and bony) in 70%.16 It may be associated with other craniofacial anomalies, especially Treacher-Collins syndrome and CHARGE association (i.e. coloboma, heart defects, atresia choanae, retardation of growth and development, genitourinary problems, and ear anomalies).17

Symptoms depend on the extent of obstruction. Neonates with unilateral atresia (50–60%) have minimal symptoms and can go undiagnosed, or present later in life with unilateral anterior nasal discharge.

Neonates with bilateral disease present with acute respiratory distress and is diagnosed by inability to pass a nasal catheter through the nasal cavity. CT scan and nasal endoscopy confirm the diagnosis.18

Neonates with bilateral atresia usually require urgent corrective surgery. As an alternative, the mouth can be kept open with an airway or McGovern nipple19 strapped, as neonates are obligatory nasal breathers.19 Emergency intubation or at times tracheostomy may be required.

If the neonate can adapt to oral breathing, then surgery can wait till infant grows to an age of ten weeks, weighs ten pounds and has a hemoglobin of 10 g/dL (rule of tens).19 Corrective surgeries for this defect include endoscopic, transnasal, transseptal or transpalatal procedures.

Anesthesia Considerations
• The neonate’s mouth must be kept open during IV/ inhalational induction, if not already intubated
• GA is given with RAE tube (preferably) and controlled ventilation. Stents are placed at the end of surgery
• Neonate must be observed postoperatively in the ICU for airway obstruction, especially after bilateral correction. They may come for dilatation later
• Topical application of mitomycin-C (IV antineoplastic agent) may be used after repair to inhibit fibroblast growth, granulation tissue formation and restenosis.20

ANESTHESIA FOR THROAT PROCEDURES

Tonsil Adenoid Resection

Tonsils and adenoids are part of Waldeyer’s ring of lymphoid tissue around the pharynx and are often the sites of acute and chronic inflammation. Hyperplasia and obstruction merit removal and is the most frequently performed procedure. Tonsillectomy alone is performed in 71% of cases and adenotonsillectomy in 27% of cases. In children less than 5 years of age, 61% of tonsil adenoid resection (TAR) are for infective and 33% for obstructive etiology.21

Indications for adenotonsillectomy are severe, recurrent pharyngotonsillitis, hemorrhagic tonsillitis, enlarged adenoids, recurrent rhinosinusitis, nocturnal upper airway obstruction with or without obstructive sleep apnea syndrome (OSAS) and otitis media.22

Sleep apnea is a sleep-related breathing disorder in children characterized by periodic cessation of air exchange, with apnea episodes lasting >10s and an apnea/hypopnea index (AHI, i.e. total number of obstructive episodes per hour of sleep) >5.23 Air flow cessation is confirmed by auscultation or oxygen desaturation <92%.

Causes include central (absent gas flow), upper airway obstruction (paradoxical movement of rib cage and abdominal muscles) and mixed (due to both CNS and obstructive problems).24

Clinical diagnosis is suggested by history of snoring and mouth breathing, restless sleep, and excessive daytime sleepiness. Polysomnography (PSG) is the gold standard for diagnosis but is not always available. Nocturnal pulse oximetry may be useful in discriminating between primary snoring and OSAS but does not assess the severity of the problem.24 OSAS can cause significant cardiac, pulmonary, and CNS impairment due to chronic oxygen desaturation if untreated.25

Preanesthetic Evaluation

Aim is to identify patients with recent URTI, and also primary, simple snorers from children with OSAS as the latter have higher incidence of perioperative complications.20,27

• To assess nasal and oral airway, and orofacial characteristics for ease of mask ventilation and intubation, presence of loose teeth and dental implants. Associated craniofacial anomalies, neuromuscular diseases, and congenital syndromes pose additional risks
• Obese children have higher risk of OSAS
• History of epistaxis, easy bruising, bleeding gums, or positive family history, point to abnormalities in coagulation status and merits further investigation. Hematologic abnormalities like sickle cell disease, Von Willebrands disease and hemophilia A should be optimized preoperatively with blood transfusion, specific factors or desmopressin20
• Investigations: CBC, bleeding and clotting time, urine analysis. Further tests like chest radiography, coagulation profile, etc. depending on history and examination
• Standard nil by mouth (NBM) guidelines. Premedication preferably avoided in children with airway compromise.

Intraoperative Considerations

• Standard monitoring along with airway pressure monitoring
• Induction can be inhalational or intravenous followed by antisialogogue, sedative, analgesic and dexamethasone for its anti-inflammatory and antiemetic effect
• Relaxation of pharyngeal muscles along with nasal airway obstruction can result in complete airway obstruction and hence the mouth must be kept open during induction. Oropharyngeal airway and moderate continuous positive airway pressure (CPAP) of 10 to 15 cm of H2O also helps to overcome this problem
• Depending on the ease of ventilation, intubation is carried out with or without muscle relaxant with ETT, preferably cuffed, to avoid aspiration. Preformed oral Ring, Adair and Elwyn (RAE) tube or a reinforced tube or a regular ETT can be used. Regular polyvinyl chloride (PVC) plain or cuffed ETT requires repeated shifting for adequate surgical exposure (Fig. 1). Constant vigilance is required throughout the surgery to avoid kinking, occlusion of the ETT, accidental disconnection or endobronchial intubation
• Pharyngeal packing is done to avoid aspiration
• Flexible LMA is another choice in experienced hands with the advantage of avoiding neuromuscular blocking drugs (NMBDs) and aiding smooth emergence29
Adequate depth of anesthesia is maintained on spontaneous or controlled ventilation with inhalational agents, propofol, and narcotics to prevent reflex hypertensive response. Children with OSAS should receive short acting and lower dosage of opioids for fear of prolonged apnea and postoperative upper airway obstruction as they have diminished ventilatory response to CO₂ rebreathing.

Ringer lactate is the fluid of choice with additional dextrose in small children.

After confirming hemostasis, pharyngeal pack must be removed and inspected for blood soakage.

Child is extubated in deeper plane (to be avoided in OSAS) or when fully awake.

Vital capacity manoeuvre (high positive pressure ventilation, 35–40 cm H₂O) and extubation under positive pressure prevents postoperative atelectasis and desaturation.

Laryngospasm post-extubation is more common in the pediatric age group and may occur due to blood and/or secretions. Positive pressure on the upper airway walls attenuates the excitation of superior laryngeal nerve and may decrease the incidence and intensity of laryngospasm. Also, the increase in intrathoracic pressure during CPAP/IPPV may depress the excitatory adductor after-discharge activity and increase the threshold of reflex that precipitates laryngospasm. Complete account of laryngospasm is presented in Chapter 35 on acute complications.

Postoperative Complications

- Respiratory: Airway obstruction and hypoxemia. Problems are more common in children less than 3 years of age, history of OSA, obese, hypotonic or with structural abnormalities
- Bleeding: Most common complication and a surgical emergency
- Tonsillar or parapharyngeal abscess is a late complication.

Post-tonsillectomy Bleeding

It is a surgical emergency. Post-tonsillectomy bleed can be primary, occurring within 24 hours, and commonly within first six hours and is profuse in nature. The secondary bleed occurring between 24 hours to 10 days is due to sloughing of tonsillar bed.

Postoperative Pain Management

Paracetamol (oral, rectal, IV) is safe and opioids have to be used judiciously especially in children with OSAS. NSAID’s have not been found to be responsible for postoperative bleeding. Practice is varied with some centers using them routinely and some totally avoiding them depending on their protocol. Infiltration of tonsillar fossa with 0.25% bupivacaine is another option.
Principles and Practice of Pediatric Anesthesia

Bleeding occurs over several hours from the tonsillar bed and is partly swallowed making blood loss estimation difficult. Excessive loss leads to hypovolemia, anemia, agitation, tachycardia, tachypnea, delayed capillary refill, and decreased urine output. Hypotension and altered sensorium indicate advanced volume depletion.

The anesthetic considerations are hypovolemia, risk of pulmonary aspiration from swallowed blood/oral intake, and difficult intubation because bleeding and edema from prior instrumentation obscures the laryngeal view. The goals are prompt and vigorous fluid resuscitation before and during anesthesia and airway management.

Anesthetic Management

- Oxygen, two IV lines with large bore cannula for fluid resuscitation and to collect blood for CBC, hematocrit, coagulation profile, blood grouping and cross matching
- Resuscitation is carried out with crystalloids/colloids (packed RBC, FFP) depending on blood loss, coagulation status and hemodynamic condition. Induction of anesthesia in a hypovolemic child can precipitate cardiovascular collapse
- Difficult airway cart, good wide bore suction and trained assistant
- Standard monitoring. Anticipate difficult laryngoscopy and intubation
- Plan rapid sequence induction, considering full stomach
- Gentle oropharyngeal and nasal suction
- Pre-oxygenation; rapid sequence induction with adequate doses of propofol, thiopentone, ketamine or etomidate depending on the hemodynamic status. Cricoid pressure and succinylcholine 1–2 mg/kg or rocuronium 0.8–1.2 mg/kg to facilitate intubation with preferably a smaller sized, styletted, cuffed ETT and immediate inflation of the cuff before releasing the cricoid pressure, followed by endotracheal suctioning if aspiration is suspected. If one is not sure of achieving ventilation, then inhalational induction and intubation under spontaneous breathing must be considered
- Controlled ventilation for achieving good hemostasis
- A large bore gastric tube is inserted to aspirate the stomach contents after intubation and at the end of the procedure
- Child is extubated when totally awake in lateral, head down position. Monitoring is continued in HDU/ICU.

PERITONSILLAR ABSCESS

It is a common deep neck space infection, commoner in older children, occurring due to spread of the primary tonsillar infection into the peritonsillar space.

Signs and symptoms are: sore throat, dysphagia, odynophagia, otalgia, high fever, trismus, drooling, peritonsillar bulge, uvular deviation, muffled voice and cervical lymphadenopathy.

Investigations: CT scan with contrast and USG of tonsillar area to determine the extent of spread, and assess airway distortion.

Management: NBM, IV fluids, antibiotics and a close watch on airway as the condition may cause airway obstruction. Needle aspiration followed by incision and drainage will be required. At times emergency (quinsy) tonsillectomy may be required.

SUPRAGLOTTIS

It is acute bacterial infection of epiglottis, aryepiglottic fold and arytenoids in a previously healthy child and common in the age group 2–6 years. Causative organisms are Haemophilus influenzae type B-Hib (75% of cases) and Beta hemolytic streptococci. Incidence has dropped with advent of Hib vaccination.

Signs and Symptoms

- Rapid onset of dysphagia, dysphonia, dyspnea, drooling, fever, tachycardia, tachypnea and inspiratory stridor which can proceed to acute, life-threatening airway obstruction without treatment. Agitation due to examination can worsen airway obstruction and so diagnosis is based mainly on presenting symptoms and signs
In selected cases, fibreoptic laryngoscopy, and CT scan of the neck are helpful. X-ray neck lateral view shows supraglottic air space narrowing in supraglottitis and subglottic narrowing in laryngotracheobronchitis. AP view shows 'Steeple sign' in the latter. The key aspects of the treatment are immediate humidified oxygenation with SpO2 monitoring, epinephrine and steroid nebulisation, antibiotics and being ready for emergency resuscitation. Child will require ICU admission for observation. In case of severe obstruction, child may have to be shifted to the OR for emergency intubation or tracheostomy for airway maintenance.

**Anesthetic Management**

- Inhalational induction in sitting position (which is mostly preferred by the child) under monitoring. CPAP of 10–15 cm H2O prevents collapse of upper airway
- Intubation in deep plane of anesthesia on spontaneous respiration with styletted ETT 1–2 size smaller for age. Cherry red epiglottis on laryngoscopy confirms diagnosis. Visualization can be difficult due to inflammed structures
- CPAP is maintained as sudden release of obstruction can cause pulmonary edema
- Throat swabs and blood are sent for culture and sensitivity and child is shifted to PICU
- Child can generally be extubated within 24–48 hours of antibiotic therapy after confirming reduction of airway edema by leak test or laryngoscopy.

**STRIDOR**

Stridor is noisy breathing caused by turbulent air flow through narrowed airway and may occur in various congenital or acquired conditions. Inspiratory stridor is more common and is due to extra-thoracic obstruction caused by cysts or masses, hemangiomas, foreign body or vascular rings. Expiratory stridor is due to intra thoracic obstruction caused by tracheal anomalies.

Craniofacial dysmorphology in syndromic child, laryngomalacia, laryngeal webs, cyst or papilloma, subglottic stenosis, supraglottic infections, laryngitis, foreign body, airway edema postintubation or extubation, and laryngeal nerve paralysis due to birth trauma or CNS diseases are some of the common causes.

**Anesthetic Considerations**

- History pertaining to age, previous intubation, feeding problems, severity and rapidity of progression helps to get to diagnosis
- Stridor worsens with agitation, URTI and in supine position, and improves in prone position, and as age advances to 4–5 years
- Child is usually posted for diagnostic or therapeutic endoscopy. Good communication with the surgeon is important
- Standard NPO guidelines and monitoring; Record SpO2 on room air pre-procedure
- Difficult airway cart to be kept ready
- Glycopyrrolate as anti-sialogogue and to suppress vagally mediated bradycardia
- Midazolam, propofol or dexmedetomidine as sedative
- Inhalational induction, decongestant and lidocaine spray on nasal mucosa followed by flexible nasopharyngoscopy and laryngoscopy while on spontaneous respiration to visualize the vocal cord movements and to rule out paralysis
- Deepening the anesthesia on spontaneous respiration with CPAP (reverses pressure gradient and reduces obstruction) and topical 2–4% xylocaine over nasal mucosa followed by flexible nasopharyngoscopy and laryngoscopy while on spontaneous respiration to visualize the vocal cord movements and to rule out paralysis
- Anesthesia can be continued further on spontaneous or controlled ventilation with an intermediate muscle relaxant and ventilation continued through the side arm of the ventilating bronchoscope. The surgeon intermittently withdraws the bronchoscope above the carina so that both the lungs can be ventilated
- Once the bronchoscope is removed a mask is held if patient is on spontaneous respiration and intubated if paralysed to continue ventilation till reversal.

**ENDOSCOPY OF AIRWAY**

It is performed for diagnostic and/or therapeutic purpose. It is diagnostic for laryngotracheomalacia, subglottic stenosis, vascular anomalies, vocal cord palsies, papillomas, hemangiomas, cysts, granuloma and foreign bodies. Therapeutic uses are for removal of foreign body and bronchial lavage.
Preoperative Examination Aims to Assess

- Degree of airway obstruction and the time of obstruction—during sleep, crying or feeding
- Amount of respiratory distress
- Maneuvers that relieve the obstruction
- Investigations: chest radiography, MRI and CT scans of the head and neck, PFT, ABG.

Anesthesia Concerns

- Sharing of compromised airway with endoscopist
- To provide adequate ventilation, clear laryngeal view and access to the structures.

Direct Laryngoscopy

- Commonly done under GA
- Sedative premedication avoided or given under monitoring; antialagogue is given
- Induction: IV, preferably propofol (as it depresses airway reflexes) or inhalational (Sevoflurane/Halothane in FiO₂ 1.0)
- Topical anesthesia of airway with 2–4% lignocaine (do not exceed 3–5 mg/kg body weight)
- Assess vocal cord dynamics and airway status
- For distal endoscopy, if in doubt of losing the airway, maintain on spontaneous respiration or intermittent doses of suxamethonium.

RIGID BRONCHOSCOPY

The rigid bronchoscope has an optical telescope, a fibroptic light source with an instrument channel and a side arm to which the anesthesia circuit can be attached for ventilation. It is available in different diameters (3 to 6 mm) and lengths (20–30 cm).

Anesthetic goals are: control over airway, reduce secretions, suppression of airway reflexes, amnesia, clear, motionless field, aspiration prevention, and a safe extubation.

- Standard monitoring
- IV antialagogue and dexamethasone 0.4 to 1 mg/kg to prevent postprocedure laryngeal edema
- IV or inhalational induction can be performed. Check laryngoscopy to assess the Cormack Lehane view. If intubation seems easy, an intermediate duration NMBD can be given for complete relaxation and atraumatic introduction of the bronchoscope
- Anesthesia circuit is then attached to the ventilating port of the bronchoscope and IPPV continued with air/O₂ and anesthesia maintained with inhalational agent sevoflurane/iso-flurane or IV propofol (Fig. 2)

Surgery for Laryngeal Stenosis

Laryngeal stenosis could be supraglottic, glottic or infraglottic. Commonest in children is subglottic stenosis, 2–3 mm below true vocal cord which could be congenital or acquired and is defined as airway diameter of less than 4 mm in term and 3 mm in premature at the level of cricoid.

Congenital failure of laryngeal lumen to recanalize leads to complete atresia, stenosis or webs. Severe congenital subglottic stenosis will require endotracheal intubation or tracheostomy for survival. Other causes of neonatal stridor are laryngomalacia (70%) and vocal cord...
Infants with mild stenosis are prone to URTI and croup. Acquired stenosis can occur in neonates on long-term intubation care because of trauma and inflammatory response.

Treatment for subglottic stenosis with glottic or tracheal involvement is tracheotomy followed by surgical reconstruction of trachea. Anterior cricothyroidotomy is an effective procedure in the absence of glottic, tracheal or pulmonary pathology.

The procedure is performed under GA with ETT and muscle relaxant. Cricoid ring, first two tracheal rings and lower 1/3rd of thyroid cartilage are incised and stay sutures are taken. The nasotracheal ETT already present is replaced with one size bigger ETT and guided distal to the slit by the surgeon. ETT is then left in place for seven days to stent the subglottic aperture. This allows fibrosis preventing obliteration due to granulation. In a modified approach thyroid, alar or auricular cartilage is placed over the slit for faster sealing. Patient has to be shifted to PICU and kept sedated with or without ventilation.

Laryngotracheal reconstruction (LTR) will be required for children with symptomatic subglottic stenosis. GA is induced through the pre-existent tracheostomy which is replaced after induction by a cuffed armoured ETT. Postoperatively this is replaced by a nasotracheal tube which is left as a stent. The child is sedated and observed in the pediatric intensive care unit (PICU).

MICROLARYNGEAL SURGERY

Microlaryngeal surgery (MLS) constitutes removal of abnormal tissue from the vocal folds (cords) using precision microlaryngeal instruments and/or laser (Light Amplification by Stimulated Emission of Radiation).

The MLS is commonly performed for vocal cord lesions like congenital or acquired cysts, intubation granulomas, laryngeal atresia, laryngeal webs and respiratory papillomas. Papillomas are caused by human papillomavirus and have a high recurrence rate requiring repeated excision.

Preoperative Concerns

- Children come repeatedly for surgery and may be psychologically disturbed
- Child may or may not be in obstruction (stridor)
- Size, mobility, and location of the lesion has to be assessed by direct or indirect laryngoscopy (DL or IDL)
- Anticipate problems in ventilation: supraglottic lesion obscures laryngeal inlet and visualization of the cords and a subglottic lesion obstructs passage of ETT
- Investigations like CXR, CT and MRI scans required to confirm the clinical findings.

Considerations and Precautions During Microlaryngeal Laser Surgery

- The laser is a beam of coherent electromagnetic radiation that can be focussed to a very small spot with precision resulting in controlled coagulation, incision, or vaporization of the target tissue without affecting the neighboring tissues. CO₂ laser is most commonly used for laryngeal surgery as the laser beam targets tissue surface causing vaporization with minimal damage to the underlying tissues. Other lasers that are used are potassium (kalium) titanyl phosphate (KTP), argon and neodymium-yttrium-aluminium-garnet (Nd:YAG) and these are generally used for the treatment of hemangiomas and granulation tissues in the tracheobronchial tree.

- GA requires using a laser-resistant ETT with metal exteriors, e.g. Laser Flex tube, Fome Cuff tube and Laser Shield II. Red rubber tubes wrapped completely without wrinkles with reflective aluminium or copper adhesive backed tape can also be used. Laser Guard, a metallic foil wrap for ETTs specially manufactured for laser is also available.

- GA requires using a laser-resistant ETT with metal exteriors, e.g. Laser Flex tube, Fome Cuff tube and Laser Shield II. Red rubber tubes wrapped completely without wrinkles with reflective aluminium or copper adhesive backed tape can also be used. Laser Guard, a metallic foil wrap for ETTs specially manufactured for laser is also available.

- During GA FiO₂ should be reduced to 0.3 or less by diluting oxygen with nitrogen, helium or medical air to keep SpO₂ 98% or above, to reduce combustibility of endotracheal tubes. N₂O is avoided as it supports combustion.

- Warning signage on the operating room (OR) doors, safety goggles that are specific for the laser in use is required for all the personnel in OR as errant CO₂ laser beams can cause serious corneal and retinal damage. Patient’s eyes should be taped closed and covered with saline soaked eye pads or metal shields or both.

- Effective smoke evacuation system for laser plume and special high efficiency masks to filter laser plume particles are necessary as viral transmission may take place.

- Laser energy transfer to an inappropriate location like ETT, anesthesia circuits, sponges, drapes, oil based lubricants, ointments, etc. can cause ignition to produce toxic combustion fumes. Airway fires have to be managed with extraction of the ETT, and elimination...
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and extinguishing the fire. A bronchoscopy is advisable later to evaluate the airway and help guide further management.73

ANESTHESIA FOR MICROLARYNGOSCOPY

- All the above mentioned precautions for laser surgery must be scrupulously followed
- Sharing of airway with surgeon, maintaining oxygenation and ventilation, avoiding aspiration and providing a dry, clear, motionless, good surgical field, at times for long duration
- Smooth induction and maintenance, cardiovascular stability and safe emergence with no coughing, bucking, breath-holding or laryngospasm and a pain-free, comfortable child at emergence
- Premedication as per respiratory status of the child and under monitored conditions
- Standard routine and airway pressure monitoring
- GA through the tracheostomy (if pre-existing) is safe and easy. Metal tracheostomy tube must be used if laser is planned. Preoxygenation, is followed by inhalational/IV Induction, IV glycopyrrolate, fentanyl and dexamethasone. GA is continued with or without intubation, on spontaneous or controlled ventilation
- GA with intubation: Technique involves use of special PVC, flexible, kink resistant, cuffed ETT for MLS without laser, or laser resistant ETT which must be of a smaller size. It provides complete control over airway, ventilation and maintenance of anesthesia, but reduces surgical access and visibility; it can cause trauma to vocal cords and also seeding of the papilloma down the respiratory tract. Smaller size ETT requires higher inflation pressures and ventilation must be with long, slow, inspiration with sufficient time given for expiration
- GA is maintained with air/O₂, inhalational agent, or with propofol, dexmedetomidine and/or narcotic infusion on controlled ventilation. Patient’s chest on which suspension laryngoscope rests should be padded and protected
- GA without intubation:
  a. Technique offers unobstructed surgical field without causing trauma to the cords; and without danger of seeding of papilloma to the lower respiratory tract. But a child on spontaneous respiratory mode requires deeper planes of anaesthesia as chances of coughing, gagging, patient movement and laryngospasm are of concern.
  b. Apneic technique can be used for short procedures: Technique involves regular induction with muscle paralysis and periods of ventilation with face mask alternated with short intervals of apnea (duration determined by SpO₂) and can be repeated a few times for completing the procedure. Technique is simple; but dangers of hypoventilation, hypercarbia, and unprotected airway are present.
  c. Insufflation technique can be used for longer duration procedures. GA is maintained on spontaneous respiration with O₂ and inhalational agent with insufflation through a nasopharyngeal airway, or a small catheter through nasopharynx placed above the glottis or through the side port of the laryngoscope (OR pollution will be a problem) or with continuous IV infusion of propofol/dexmedetomidine/fentanyl. It is supplemented by topical lignocaine on vocal cords.78
  d. Jet ventilation technique: In this technique, the patient is paralyzed and ventilated using a supraglottic venturi (Saunders jet injector) through the side port of the suspension laryngoscope.78

Air oxygen mixture is used and maintenance is by total intravenous anesthesia (TIVA). Ventilation must be done carefully, observing for chest expansion and giving enough time for passive expiration to avoid volutrauma/barotrauma of the lungs, gastric distension and regurgitation.78 A subglottic jet ventilation anesthesia system having a laser-safe, subglottic jet ventilation tube and an automatic jet ventilator with provision for monitoring of tracheal pressure and end tidal carbon dioxide is also used at some centres.79

SURGICAL TRACHEOSTOMY

It is a procedure involving the creation of an artificial airway through the trachea. Common indications include airway obstruction, chronic assisted ventilation, pulmonary toilet.80

Commonly available PVC tracheostomy tubes are Portex and Shiley brands for both neonatal and pediatric age groups. Tracheostomy tubes of same external diameter but with different lengths are available and must be chosen correctly to avoid endobronchial placement. PVC tubes are preferred as they cause minimal tissue reaction. Metal tubes are also available.
Anesthetic Considerations

Tracheostomy is generally performed as an elective procedure. Most of the time, patient is already intubated for ventilatory support. Check IV line and attach monitors. Before commencing anesthesia, confirm ETT position and patency clinically and with capnogram as ETT may have slipped out or slipped into the esophagus or become endobronchial during transport from ICU.

If patient is not already intubated, then one can proceed with preoxygenation, induction with oxygen and inhalational agent, local spraying of larynx, followed by ETT placement with the child breathing spontaneously. ETT also helps to identify the trachea during surgery. ETT placement using flexible fiberoptic bronchoscope (FFB) is another option. Esophageal stethoscope and nasogastric tube can occasionally be misidentified as ETT and therefore must be removed before start of procedure to avoid oesophageal incision. LMA can be placed if intubation is not possible; sometimes surgeon may prefer to use a rigid bronchoscope held in place instead of an ETT.

Patient is positioned with shoulders elevated and neck hyperextended and head supported in a ring. Adequate depth of anesthesia is mandatory to prevent movement of the child during the procedure. During tracheostomy tube insertion, ETT is pulled back but not taken out completely. Ensure adequacy of ventilation through tracheostomy tube before removing the ETT.

Postoperatively: Care in ICU/HDU; chest radiography to check the position of the tube and to rule out complications.

Possible complications are more in young infants. Early complications include hemorrhage, pneumomediastinum, subcutaneous emphysema, pneumothorax, accidental decannulation and blocking of the tube. Late complications include subglottic and tracheal stenosis and persistent tracheocutaneous fistula. Early tracheostomy tube change is avoided as the tract is unstable and the soft tissue can close the tract. Forceful placement can create false passages, inability to ventilate and a critical situation.

BRONCHOSCOPY FOR FOREIGN BODY REMOVAL

Most common indication for bronchoscopy is removal of aspirated foreign body (FB) in young children (1 to 4 years), though older children can also aspirate; diagnostic bronchoscopy follows next. Positive history of aspiration may or may not be present and a foreign body may be revealed only on bronchoscopy after failure of medical treatment for lower respiratory tract infection (LRTI).

Commonly aspirated objects include peanuts or other improperly chewed food, plastic or metal toy parts, and coins. Symptoms and signs depend on the site of obstruction, the type of foreign body and its size, and the duration of aspiration. Most common is bronchial aspiration (right more than left) presenting with cough, wheeze, dyspnea and reduced air entry on the affected side.

Laryngeal/tracheal foreign bodies, though less frequently encountered, present with acute respiratory distress demanding immediate retrieval of the FB.

Organic foreign body creates a lot of mucosal irritation, edema and a greater incidence of pneumonia distal to the bronchial obstruction than most other foreign bodies and presents earlier than nonorganic FB.

Chest radiograph: (a) may be normal especially if obtained within the first 24 hours after the aspiration, (b) radiolucent FB will not be seen but secondary pathologic changes such as atelectasis or obstructive emphysema signs point to probable FB aspiration. Bronchoscopy should be emergent if child is in distress or urgent in properly prepared child (NBM guidelines, investigations, etc.).

Anesthesia Plan

- Monitoring is extremely important as there are periods of apnea, hypoventilation
- Preoxygenation is a must. Inhalation or IV induction is followed generally by NMBD and gentle controlled ventilation through the side arm of the ventilating bronchoscope. If the FB is in the trachea, it may be necessary to push it into one of the bronchus to achieve ventilation. Adequate depth of anesthesia has to be maintained to prevent unexpected movement, cough, laryngospasm or bronchospasm.
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- Spontaneous ventilation techniques with topical anesthesia of the vocal cords is advocated by some to avoid the risk of FB being forced into the smaller airways with IPPV. Risk for the spontaneously breathing patient is unexpected movement or cough. Propofol, dexmedetomidine or inhalational agent can be used for maintenance during spontaneous respiration.

- FB can be taken out piecemeal with multiple attempts. Sometime the forceps holding the FB and the scope are taken out together.

- Postprocedure, an ETT can be inserted to maintain ventilation till reversal and also for tracheal suctioning.

- Complications include hypoxia, arrhythmias, pneumothorax, laryngospasm, and bronchospasm.

- IV dexamethasone 0.4–1 mg/kg, and smaller doses for following 2 to 3 days to minimize the subglottic swelling caused by the procedure.

- Nebulization with epinephrine and steroid under ECG monitoring.

ESOPHAGEAL FOREIGN BODY (EFB) EXTRACTION

Retention of ingested foreign body is much more common than aspiration of foreign body in young children. Commonly ingested foreign bodies are coins, food, bones, buttons, button batteries, toys, and pins (Fig. 4). Quite often they are asymptomatic and unrecognized and may be excreted out especially once they reach the stomach.

Sites of lodging of EFBs are: (a) most commonly at the level of cricopharyngeus muscle at the thoracic inlet and seen on X-ray chest at the level of the clavicles, (b) the middle esophagus at the level of the carina and aortic arch, (c) the distal esophagus just proximal to esophago-gastric junction (seen on X-ray at 2–4 vertebral bodies above the stomach bubble). Presence of a previous stricture (e.g. operated cases of esophageal atresia) is a predisposing factor for obstruction.

If the child is asymptomatic, then the child can be observed for 12–24 hours for spontaneous excretion of the EFB. If the EFB has not passed down to the stomach in this period, then its removal under GA must be planned. If the duration is more than a week, then there is significant risk of erosion into the surrounding structures and the surgical team for possible thoracotomy/laparotomy should be kept ready before attempting endoscopic removal. In cases with unavailable history of FB ingestion, high index of suspicion must be kept in children presenting with chronic symptoms.

EFBs lodged in the esophagus, can produce the following symptoms: dysphagia, odynophagia, drooling, gagging, retching and vomiting, coughing, choking or airway compromise. Situations meriting immediate removal are EFB producing airway obstruction in a dyspneic child, button battery or sharp object ingestion.

The EFBs retained for a long time can result in serious complications like erosion, perforation, broncho-esophageal fistula, aorto-esophageal fistula, mediastinitis, esophageal diverticulum, and lobar atelectasis. Such EFBs may require a thoracotomy to remove them. Sharp bodies lodged in stomach may require gastrotomy for extraction.

Anesthesia Considerations

- Preoperative X-ray chest and neck (AP and lateral views) to detect position of FB. CT scans of chest is indicated in longstanding cases or with suspected radiolucent FB
- If the foreign body is suspected in the hypopharynx, child must be sedated to prevent gagging and coughing that can dislodge it into the larynx creating an emergency situation.
- In elective cases:
  - Adequate NBM status, sedation and an antisialogogue
  - Inhalational or IV induction, cricoid pressure is avoided to prevent dislodgement of FB into the trachea
  - A smaller size ETT is used to prevent the mucosa over the cricoid cartilage getting traumatized between the ETT and the rigid esophagoscope. ETT secured on the left side to facilitate endoscopy
  - IPPV with NMBD and adequate depth of anesthesia to prevent esophageal perforation by the scope with patient movement.
- Antiemetic and dexamethasone (0.4–1 mg/kg) to prevent edema and postextubation stridor. Careful monitoring postprocedure for chest pain, pyrexia, and subcutaneous emphysema is warranted
- If esophageal perforation is suspected, then child is kept nil by mouth; IV antibiotics and IV fluids are started and child is cared for in ICU.

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Chapter 16: Anesthesia for Ear, Nose and Throat Procedures in Children


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INTRODUCTION

The commonly performed plastic surgical procedures in children include repair for cleft lip and palate and reconstruction procedures for craniofacial anomalies, temporomandibular joint ankylosis, anomalies of the foot and hands and burns (see Chapter 20). Anesthesia considerations for these procedures require a thorough assessment of the existing anomaly and prevention and management of airway difficulties, blood loss, aspiration of blood and secretions, adverse respiratory events like bronchospasm, laryngospasm and respiratory obstruction. In addition, these children may have associated congenital anomalies and medical illnesses which have an adverse impact on anesthesia management. It therefore becomes very important that these children are thoroughly evaluated and optimized before surgery for a good outcome.

CLEFT LIP AND PALATE

The condition is present since birth with difficulty in feeding and swallowing, nasal regurgitation, history of (H/O) repeated upper respiratory infection (URI), pulmonary aspiration, chest infection and hearing problems, delayed dentition or maloccluded teeth and nasal speech. A child with a cleft lip is unable to suck as negative pressure cannot be established; he is unable to make consonants like B, D, K, P, and T and has typical cleft palate voice and audiometrically detected hearing loss of 10 decibels is present due to inflammation from regurgitated food. During the antenatal period, mother may have history exposure to X-ray, intake of drugs like cortisone, diazepam and phenytoin, vitamin deficiency and viral infection like rubella in 1st trimester. In these children, milestones are delayed and in 10% of cases associated congenital anomalies are present (Table 1).

Preoperative Assessment

The child should be assessed for:
- Presence of other congenital anomalies
- Eustachian tube dysfunction and chronic serous otitis with clear rhinorrhea
- Anemia
- URI may be difficult to control in preoperative period in children with cleft palate. In these children, an effective dose of antibiotics can be given before surgery
- Undernourishment and dehydration because of poor intake

Table 1: Congenital anomalies commonly associated with cleft lip and palate

<table>
<thead>
<tr>
<th>Anomaly</th>
<th>Syndrome</th>
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<tbody>
<tr>
<td>Hypertelorism</td>
<td>Vander Woude syndrome</td>
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<tr>
<td>Congenital heart disease</td>
<td>Down syndrome</td>
</tr>
<tr>
<td>Hand and foot anomalies</td>
<td>Pierre – Robin syndrome</td>
</tr>
<tr>
<td>Hydrocephalus</td>
<td>Klippel Feil syndrome</td>
</tr>
<tr>
<td>Congenital blindness</td>
<td>Treacher Collins syndrome</td>
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<tr>
<td>Mental deficiency</td>
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</table>
Investigations

- Routine—complete blood count and urine examination
- Chest X-ray if there is fever, running nose, purulent secretions and noisy chest
- Investigations dictated by associated congenital anomalies.

ANESTHESIA MANAGEMENT

All children should be fasted according to ASA guidelines. Oral midazolam 0.5 mg/kg, 20–30 min before induction can be used for parental separation provided difficult airway does not contraindicate its use. Children are monitored during surgery with precordial stethoscope, ECG, pulse oximetry, end-tidal CO₂ and end-tidal anesthetic agents, noninvasive blood pressure (NIBP), temperature and fluid balance and blood loss.

Children may be anesthetized with inhalational or intravenous routes utilizing oxygen, sevoflurane or halothane followed by securing of IV access or with thiopentone or propofol if IV access is available. Before administering a muscle relaxant, confirm effective mask ventilation and use a tooth guard/rolled gauze piece over the defect while performing laryngoscopy and intubation to avoid trauma to the lips and gums. It also prevents the laryngoscope blade from falling into the cleft (Fig. 1). Any non-depolarizing muscle relaxant can be used for intubation but atracurium in a dose of 0.5 mg/kg is preferred. Intubation may be difficult in presence of syndromes and bilateral cleft where succinylcholine (1–1.5 mg/kg) may be administered. After intubation with an appropriate RAE endotracheal tube, check bilateral air entry, introduce pack and protect eyes. The surgeon introduces a mouth gag before performing cleft palate surgery and care should be taken to see that the endotracheal tube is not compressed when it is opened. We use hypodermic needle cover to prevent tube compression (Fig. 2). One should auscultate for the breath sounds and chest compliance during placement and manipulation of the mouth gag during manual ventilation. Tube compression can be detected if there is an increase in airway pressures if patient is on a ventilator and by decreased bag compliance if manually ventilating.

Anesthesia can be maintained with oxygen, nitrous oxide and inhalational agent (desflurane, sevoflurane, isoflurane or halothane) and intermittent doses of non-depolarizing muscle relaxants. Analgesia can be provided with morphine 0.1 mg/kg or fentanyl 1–2 µg/kg. Intermittent positive pressure ventilation (IPPV) decreases bleeding and also maintains tidal volume which may be compromised because of head down tilt. During spontaneous ventilation the abdominal viscera presses upon the diaphragm and so increases work of breathing which is prevented by IPPV. At the end of surgery, muscle relaxation is reversed by atropine/glycopyrrolate (0.025 mg/kg/0.01 mg/kg) and neostigmine (0.05 mg/kg). After suction of the throat under vision, remove pack and then remove endotracheal tube (ETT) after child is fully awake, responding to commands and has full muscle tone. Child should be nursed in lateral or semiprone position to keep the airway unobstructed and allow blood...
to trickle out. After palate repair blood tends to gravitate towards hypopharynx and larynx. Auscultate the chest for any aspiration. Elbow sleeve should be applied to avoid the child touching the repaired area. Postoperative pain management can be achieved by paracetamol,\(^4\) non-steroidal anti-inflammatory drugs (NSAIDs),\(^5\) infiltration of cleft repair site with local anesthetic and additives like ketamine and dexmedetomidine;\(^6-8\) infraorbital nerve block\(^9\) and maxillary nerve block.\(^10,11\) The adverse events for which an anesthetist should be alert are summarized in Table 2.

### Table 2: Perioperative problems with cleft lip and palate surgery

<table>
<thead>
<tr>
<th>Preoperative</th>
<th>Intraoperative</th>
<th>Postoperative</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Difficult veins</td>
<td>• Difficult intubation</td>
<td>• Airway obstruction due to pack left inadvertently, tongue and pharyngeal edema, tongue fall and bleeding</td>
</tr>
<tr>
<td>• Difficult mask ventilation</td>
<td>• Accidental extubation during positioning</td>
<td>• Postoperative nausea and bleeding</td>
</tr>
<tr>
<td></td>
<td>• Malposition of mouth gag in relation to ETT leading to partial or complete airway obstruction</td>
<td>• Pain</td>
</tr>
<tr>
<td></td>
<td>• Obstruction due to pharyngeal pack – tube compression</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Anrhthymias when using halothane and adrenaline infiltration</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Blood loss</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Problems of hypothermia and hypoglycemia</td>
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</table>

### CRANIOFACIAL SURGERY

Craniosynostosis is a condition where there is premature fusion of one or more cranial sutures (Fig. 3) leading to a failure of normal bone growth perpendicular to the suture and a compensatory growth at other suture sites resulting in a characteristic abnormal head shape. Most syndromic craniosynostoses show autosomal dominant inheritance, although the majority is attributed to new mutations from unaffected parents. Mutations in genes coding for fibroblast growth factor receptors (FGFRs) are responsible for the most common syndromes.\(^12\) The condition may be isolated (80%) or occurring in association with many syndromic conditions (20%). Both of them can lead to raised intracranial pressure (ICP) due to hydrocephalus, airway obstruction or abnormalities in the venous drainage of the brain.\(^12\) Raised ICP presents with visual difficulties, nausea and vomiting, somnolence or headaches and “sun-downing” appearance. In children with Apert’s and Crouzon’s syndromes, maxillary hypoplasia leads to narrowing of the nasal cavity and nasopharynx. Glossoptosis may cause obstruction of the hypopharynx in children with mandibular hypoplasia.\(^13\) The common syndromes which can cause craniosynostosis are shown in Figure 4. The various surgical procedures which can be performed for craniosynostosis are shown in Table 3.

### Table 3: Types of surgical procedures for craniosynostosis

<table>
<thead>
<tr>
<th>Surgery for sagittal synostosis</th>
<th>Frontal orbital advancement and remodeling</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. Extended strip craniectomies</td>
<td>Posterior expansion and remodeling</td>
</tr>
<tr>
<td>b. Spring-assisted cranioplasty</td>
<td>Midface advancement (Le Fort III and monobloc procedures)</td>
</tr>
<tr>
<td>c. Total calvarial remodeling</td>
<td></td>
</tr>
</tbody>
</table>

---

Fig. 3: Normal cranial bones and sutures in a neonate
Fig. 4: Various craniofacial syndromes

- Apert’s syndrome
- Pierre Robin syndrome
- Treacher Collins syndrome
- Goldenhar syndrome
- Crouzon’s syndrome
PREOPERATIVE ASSESSMENT

During the preoperative visit rapport and trust is established with the patient and family to reduce anxiety. Parents should be told about the possibility of intraoperative blood loss and possible need for mechanical ventilation postoperatively. The child should be evaluated for pre-existing medical conditions (congenital heart disease), medication history, allergies, family history of problems with anesthetics, problems with previous anesthetics and a physical examination.

Children with major congenital craniofacial abnormalities may present with upper airway obstruction because of involvement of the cranium, midface and mandible (Table 4). A history of abnormal sleep patterns like noisy snoring, restless sleep and frequent arousals during sleep, sleep apnea and daytime somnolence identifies patients who are likely to develop airway obstruction during sedation and induction of anesthesia. Children should be assessed for signs of raised ICP. Many syndromic craniosynostosis may produce difficulty in intubation and therefore should have a thorough airway assessment. Oral and nasal cavities should be examined if fiberoptic intubation (FOB) is planned. The mobility of the cervical spine should be evaluated if Goldenhar’s syndrome is suspected. In Apert’s syndrome, there is midface hypoplasia and proptosis which can make face mask ventilation difficult. Because of small nares and a degree of choanal stenosis there may be high resistance to airflow through the nasal route and so these patients are obligate mouth breathers. Thus, face mask ventilation with a closed mouth can lead to obstruction which can be relieved by simple airway adjuncts such as an oropharyngeal airway (OPA) or nasopharyngeal airway (NPA) and continuous positive airway pressure (CPAP). Children with Apert’s syndrome also have fused cervical vertebrae. Children who have undergone frontofacial advancement may have difficulty in intubation as a result of the altered relationship between the maxilla and mandible and reduced temporomandibular joint movement.

Children may show signs of upper respiratory infection presenting as wheeze. Almost 50% of patients with Apert, Crouzon, or Pfeiffer syndromes develop obstructive sleep apnea (OSA). The obstruction may be due to midface hypoplasia, causing a distortion in the nasopharyngeal anatomy. Chronic upper airway obstruction may lead to an increase in ICP and a subsequent decrease in cerebral perfusion pressure (CPP). A negative effect on neurological and cognitive development occurs because of recurrent episodes of intermittent reduction in CPP.

Investigations

These should include a preoperative hematocrit (Hct), platelet count, coagulation studies, serum electrolytes and urea and creatinine along with routine CBC and urine. X-ray chest for assessment of lung fields and heart size and radiograph of the cervical spine to rule out fusion/atlantoaxial dislocation of spine are essential. Blood is grouped and cross-matched and appropriate volume of fresh packed blood and blood products like fresh frozen plasma, platelets, fibrinogen is kept ready.

ANESTHESIA MANAGEMENT

Young infants do not require any premedication but older children may be administered oral midazolam 0.5 mg/kg half an hour before induction of anesthesia to alleviate separation or situational anxiety. A child with history of OSA or difficult intubation should not be premedicated. In this group of patients, an intravenous line may be secured after application of topical anesthetic cream 1 hour before the expected time of induction. If FOB is planned the child should be administered atropine or glycopyrrolate for drying of the oral secretions. Inhalational (sevoflurane preferred because of its rapid uptake and removal) or intravenous induction can be used followed by endotracheal intubation with or without the use of non-depolarizing muscle relaxants. Inhalational induction is preferred because of risk of difficult ventilation in syndromic children and difficulty in securing IV access. Various techniques of intubation have been described in the literature depending on the difficulty in securing the airway. These may range from rigid laryngoscopy to FOB via oral or nasal route. Since awake intubation may not be feasible in children because of lack of cooperation LMA guided FOB may be an alternative technique. Other intubation techniques like retrograde intubation and use of bougies in children have also been described in literature. A preformed oral (RAE) tube or an armored tube is preferred for intubation which should be fixed securely (using a suture or wired to the tooth) to avoid the possibility of dislodgement during manipulation of head during craniotomy.

A balanced neurosurgical technique using opioids and inhalational agents and controlled ventilation is
the anesthetic technique of choice to avoid increase in ICP. Isoflurane is the anesthetic of choice for maintenance of anesthesia since it causes the least rise in ICP. Various authors have utilized remifentanil as well as a combination of sevoflurane and remifentanil for surgical repair of craniosynostosis with good results. Nitrous oxide should be avoided because of the risk of venous air embolism (VEA).

**Intraoperative Problems**

The intraoperative body temperature should be maintained at 35°–37°C by warming all IV fluids, wrapping the non-exposed body parts in plastic sheets, using forced air warming device or warm-water heating pad and using heated humidifiers or HME devices in airway circuit to minimize evaporative heat loss from respiratory tree. Additional protective padding should be used at pressure points to avoid nerve injury.

The child has a larger body surface area-to-volume ratio compared with the adult (head comprises nearly 18% of the surface area vs 9% in adults). This results in proportionally greater fluid and heat losses in a child. The fluid loss may vary from 6–8 mL/kg (extradural procedure) to 10–12 mL/kg (intradural procedure). Fluid is administered to provide maintenance requirements, replace third space losses and to replace a portion of the blood loss. The fluid requirement and therapy can be monitored by central venous pressure (CVP) and urine output.

The surgical procedure may carry a risk of air embolism when venous structures are exposed to the atmosphere, causing the subatmospheric intravascular pressure to entrain air. Mass spectroscopy of end-tidal gases (elevation of end-tidal nitrogen concentration and a sudden decrease in PetCO₂) is the most sensitive indicator of this entrainment. Precordial Doppler is recommended for monitoring of air embolism. However in small children, the technique is cumbersome and offers little benefit.

Pediatric craniofacial surgery commonly requires blood transfusion therapy because extensive scalp dissection and calvarial and facial osteotomies result in significant blood loss. In infants and children, the estimated blood volume ranges between 75–80 mL/kg. Therefore, intraoperative blood transfusion is inevitable in craniosynostosis repair and depends on type of suture repaired and the type of surgical procedure performed. Measures to reduce blood loss and use of alternative techniques for blood conservation can be utilized.

**Monitoring**

Routine monitoring includes ECG, oxygen saturation (SpO₂), end-tidal carbon dioxide (ETCO₂), core temperature and urine output. Invasive arterial pressure monitoring is essential because of potential for massive blood loss. Adequate venous access is essential and requires two large bore IV cannulae. Central venous pressure monitoring is desirable in those cases where excessive blood loss is anticipated. Intraoperative assessment of coagulation parameters may be sometimes required where massive blood transfusion has occurred. Routine use of precordial Doppler for early diagnosis of venous air embolism is essential.

**TEMPOROMANDIBULAR JOINT ANKYLOSIS**

The causes of temporomandibular joint (TMJ) ankylosis in children may be congenital or acquired due to trauma. Anesthesia management is challenging in children because of their restricted mouth opening with near total trismus, and the need for general anesthesia before making any attempts to secure the airway (Fig. 5).

**PRESENTATION**

The child usually presents with inability to open mouth and protrude his jaw with oral intake limited to only fluids with passage of time. If the condition is congenital it may be associated with hypoplasia of the mandible. The main issues are related to the various methods to secure the airway for the surgical repair. FOB guided intubation is the best, but other methods like blind nasal intubation, use of track light, retrograde intubation and tracheostomy...
can also be utilized. Rest of the anesthesia management is based on the basic principles of pediatric anesthesia.

OTHER PLASTIC SURGICAL PROCEDURES

Surgical procedures on the hand and foot are required for syndactyly, burn contracture and club foot. Children may also present with ear deformities, arteriovenous malformation and hemangioma which require surgery. The anesthetic management of these surgical procedures may include general anesthesia which is administered via supraglottic airway devices (LMA, PLMA, I-gel and Air Q) or endotracheal tube. General anesthesia can be combined with ultrasound guided upper limb nerve blocks or caudal block depending upon surgical procedure for perioperative pain relief.

CONCLUSION

Anesthesia for children undergoing plastic surgery procedures can be challenging for an anesthesiologist. It involves focus on airway assessment and management of difficult airway; assessment of blood loss and replacement and intensive perioperative and postoperative monitoring for a favorable outcome.

REFERENCES

Anesthesia for Pediatric Dentistry

INTRODUCTION
American Academy of Pediatric Dentistry (AAPD) defines pediatric dentistry as an age defined specialty that provides both primary and comprehensive preventive and therapeutic oral health care for infants, and children through their adolescence, including those with special medical needs.1 The anesthesia requirements in pediatric dental patients may lie anywhere along the spectrum of monitored anesthesia care (MAC) to sedation or general anesthesia. Complications like obstructive airway, hypoventilation, apnea, laryngospasm, and cardiopulmonary changes are known to occur and hence it should be standard practice to have a separate sedation provider.2 The challenges faced by the anesthesiologist are rare syndromes, a shared airway with the dental surgeon, and working outside the comfort zone of the operation theater with untrained assistants who may not be competent enough to help in the event of some catastrophe. It is likely to be the first anesthesia experience for the child and his parents, hence we should put in our best efforts to make it pleasant and safe.

CLINICAL PRESENTATION
Pedodontists treat a large base of healthy children. They may also deal with other patients such as:3
- Disabled children and adolescents
- Psychologically challenged children
- Medically compromised children
- Children with orofacial trauma
- Children requiring orthodontic care.

PEDiatric DENTAL PROCEDURES
- Operative restorations, including amalgam and composite resin
- Stainless steel crowns
- Pulpal treatments
- Extractions
- Orthognathic plates for cleft palate patients.

THE DENTAL CHAIR
Pediatric dental chairs are usually smaller than conventional dental chairs, hence may be incapable of accommodating larger children (Fig. 1). Many
pedodontists thus use conventional dental chairs along with wooden or papoose board. Dental chair must be capable of head-down tilt even in the event of power failure. When the patient is placed supine pooled saliva or blood can trickle behind and induce coughing. Upright position in the dental chair predisposes to postural hypotension, there is a risk of cerebral hypoxia consequent to unrecognized fainting. The most common position used is semisupine, where the airway is maintained along with distinct cardiovascular and respiratory advantage.4

**LOCAL ANESTHETICS**

Regional and local blocks are usually stand-alone techniques or combined with procedural sedation or general anesthesia (GA) in children. Most procedures are done under infiltrative anesthesia. Maxillary and mandibular nerve blocks are given for extensive work. Reduced bone density of the maxilla and mandible in children may lead to rapid diffusion and absorption of local anesthetic hence toxicity occurs at doses well below the toxic level in adults. To minimize sensation of needle prick, topical lignocaine gel/spray can be applied on the dried mucosa and left in place for at least one minute to achieve effect. In patients allergic to bisulfates local anesthetic without a vasoconstrictor agent is preferred (Table 1).

**LOCAL ANESTHETIC ALLERGY**

With local anesthetics, genuine anaphylactic reactions are rare. Allergic reactions have been caused by coincidental exposure to antigens such as preservatives (e.g. methyl-p-hydroxybenzoate), antioxidants (e.g. bisulfate), antiseptics (e.g. chlorhexidine), and other antigens such as latex, as well as local anesthetic drugs.6 Allergy tests used are skin tests (patch test and/or prick test and/or intradermal reaction) and/or challenge tests. In event of drug allergy in a patient, skin tests should be carried out 4 to 6 weeks after the reaction. Skin prick-tests and intradermal tests are done with dilutions of commercially available drugs. Control tests using saline (negative control) and codeine (positive control) must accompany skin tests. Skin tests are read in 15–20 minutes.6 Prick test is viewed positive, if diameter of the wheal is at least equal to half of the positive control test and at least 3 mm greater than the negative control. Intradermal tests are considered positive, when the diameter of the wheal is twice or more the diameter of the injection wheal (Table 2).

**PROCEDURAL SEDATION**

Children are fearful and uncooperative during dental procedure. The pedodontist however is able to negotiate with behavior management techniques in most of them. Those children where this is not possible, sedation may help avoid the need for general anesthesia (Table 3).

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**Table 1: Doses of local anesthetics**

<table>
<thead>
<tr>
<th>Local Anesthetic</th>
<th>Maximum dose (mg/kg) without epinephrine</th>
<th>Maximum dose (mg/kg) with epinephrine</th>
<th>Approximate duration (minutes)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lidocaine</td>
<td>5.0</td>
<td>7.0</td>
<td>90–200</td>
</tr>
<tr>
<td>Bupivacaine</td>
<td>2.0</td>
<td>3.0</td>
<td>180–600</td>
</tr>
<tr>
<td>Levobupivacaine</td>
<td>2.0</td>
<td>3.0</td>
<td>180–600</td>
</tr>
<tr>
<td>Ropivacaine</td>
<td>2.0</td>
<td>3.0</td>
<td>180–600</td>
</tr>
<tr>
<td>Articaine</td>
<td>7.0</td>
<td></td>
<td>60–230</td>
</tr>
</tbody>
</table>

**Table 2: Concentrations of local anesthetic agents for skin tests**

<table>
<thead>
<tr>
<th>Available agents</th>
<th>Prick-tests</th>
<th>Intradermal tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>Max. conc. and/or dilution</td>
<td>mg.mL-1</td>
<td>mg.mL-1</td>
</tr>
<tr>
<td>Bupivacaine</td>
<td>2.5</td>
<td>Undiluted</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>10</td>
<td>Undiluted</td>
</tr>
<tr>
<td>Ropivacaine</td>
<td>2</td>
<td>Undiluted</td>
</tr>
</tbody>
</table>

**Table 3: Sedation continuum**

<table>
<thead>
<tr>
<th></th>
<th>Minimal sedation/Anxiolysis</th>
<th>Moderate sedation/Analgesia (“Conscious sedation”)</th>
<th>Deep sedation/Analgesia</th>
<th>General anesthesia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Responsiveness</td>
<td>Normal response to verbal stimulation</td>
<td>Purposeful response to verbal or tactile stimulation</td>
<td>Purposeful response following repeated or painful stimulation</td>
<td>Unarousable even with painful stimulus</td>
</tr>
<tr>
<td>Airway</td>
<td>Unaffected</td>
<td>No intervention required</td>
<td>Intervention may be required</td>
<td>Intervention often required</td>
</tr>
<tr>
<td>Spontaneous ventilation</td>
<td>Unaffected</td>
<td>Adequate</td>
<td>May be inadequate</td>
<td>Frequently inadequate</td>
</tr>
<tr>
<td>Cardiovascular function</td>
<td>Unaffected</td>
<td>Usually maintained</td>
<td>Usually maintained</td>
<td>May be impaired</td>
</tr>
</tbody>
</table>
Only patients categorized into ASA class I and II are acceptable as candidates for conscious sedation. Even children below 2–3 years can be treated on day care basis. Generally patients of ASA III and IV are better managed in a hospital setting. The equipment and monitoring is similar to the operating room. Standard ASA monitors are mandatory. Appropriate sizes of oral and nasal airways, laryngoscope with blades, endotracheal tubes, laryngeal mask airways (LMAs), difficult airway cart and suction should be available.

**Sedation Techniques**

Sedative drugs may be administered by oral, submucosal, intramuscular, rectal, inhalational or intravenous routes. Inhalational sedation is preferred by pedodontists because of reliability in terms of onset and recovery. Fasting guidelines need to be followed for sedation procedures.

**Nitrous Oxide Sedation**

Nitrous oxide/oxygen sedation is useful in children who are 4 years and older for mild-to-moderate anxiety. It is used in children with a strong gag reflex, as well as with muscle tone disorders, such as cerebral palsy, in order to avoid unintentional movements. Contraindications include uncooperativeness, claustrophobia, maxillofacial deformities that prevent nasal hood placement (Fig. 2), nasal obstruction, deviated nasal septum, etc.

**Technique**

According to the American Academy of Pediatrics Guidelines, nitrous oxide delivery equipment should have the capacity of delivering 100% oxygen concentration. It is to be used in conjunction with a calibrated and functional oxygen analyzer. With this type of minimal sedation, the child is able to maintain communication throughout the procedure. The delivery tubes are usually secured behind the chair, nasal hood is fixed and the child is asked to breathe through the nose with his mouth closed. At induction the breathing bag is filled with 100% oxygen and delivered to the patient at 4–6 liters per minute for 2–3 minutes. Once the appropriate flow rate is reached, nitrous oxide is introduced slowly at increments of 10 to 20% to achieve the desired level. Local anesthetic is injected when the eyes take on a distant gaze with sagging eyelids. Then the concentration can be reduced to 30% N₂O and 70% O₂ or lower. Recovery is achieved quickly by reverse titration and the patient is allowed to breathe 100% oxygen for 3–5 minutes. Child is instructed to remain in the sitting position for a brief period to ensure against dizziness on standing. The incidence of diffusion hypoxia is minimal after the use of nitrous oxide and oxygen alone as opposed to nitrous oxide supplementation to parenteral or oral sedatives.

**SEDATIVE DRUGS COMMONLY USED**

**Midazolam**

Oral midazolam helps in calming children and does not increase gastric pH or residual volume (Table 4). Disadvantages of this route are delayed onset of action, variable absorption in the gastrointestinal tract and bitter
taste which is difficult to mask. Children sedated with intranasal midazolam (preservative free 5 mg/mL) are passive and moderately drowsy but usually do not fall completely asleep. The efficacy may be decreased in the presence of nasal secretions, larger volume may result in coughing, and sneezing and expulsion of part of the drug.\textsuperscript{13} It produces a burning sensation and a bitter taste on reaching the oropharynx. Chiaretti and colleagues used a single puff of lidocaine spray (10 mg) to provide a local anesthetic effect before administering 0.5 mg/kg intranasal midazolam which found high acceptance rate.\textsuperscript{14} It is speculated, intranasal midazolam may be absorbed into the brain and cerebrospinal fluid directly through the cribriform plate to achieve proportionately higher concentrations.\textsuperscript{13} Absorption via the rectal route has been found to be poor, irregular and associated with rectal pain, itching, and defection.\textsuperscript{15} Secondary and adverse effects of midazolam may include a paradoxical effect, with behavioral changes, agitation and hiccups. Ketamine 0.5 mg/kg IV has been shown to reverse the agitation.\textsuperscript{17}

### Chloral Hydrate and Trichlofos

Chloral hydrate is a popular drug for management of anxiety in pediatric dentistry. The gastric irritation it produces may be minimized by diluting the drug or following it immediately with milk or water. It does not possess any analgesic properties, therefore the drug should not be administered to patients who are in pain because their response may become quite exaggerated. Half life of chloral hydrate is 7–9.5 hour. The dose is 50 mg/kg with a suggested range of 40–60 mg/kg.

Trichlofos is a closely related drug which is metabolized in the liver to the same active metabolite trichloroethanol which is responsible for CNS depression. It is more palatable than chloral hydrate. The oral solution is well absorbed, proves effective within 30–40 minutes, and produces hypnosis for 6–8 hour in doses of 25–75 mg/kg.

<table>
<thead>
<tr>
<th>Route of administration</th>
<th>Age (year)</th>
<th>Dose (mg/kg)</th>
<th>Maximum (mg)</th>
<th>Minutes before procedure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intramuscular or nasal</td>
<td>0.5–2</td>
<td>0.2</td>
<td>3</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td>2–6</td>
<td>0.15</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td></td>
<td>6–12</td>
<td>0.1</td>
<td>7.5</td>
<td></td>
</tr>
<tr>
<td></td>
<td>&gt;12</td>
<td>0.075</td>
<td>7.5</td>
<td></td>
</tr>
<tr>
<td>Rectal</td>
<td>0.5–2</td>
<td>0.7</td>
<td>10</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td>2–6</td>
<td>0.6</td>
<td>15</td>
<td></td>
</tr>
<tr>
<td></td>
<td>6–12</td>
<td>0.4</td>
<td>15</td>
<td></td>
</tr>
<tr>
<td></td>
<td>&gt;12</td>
<td>0.3</td>
<td>15</td>
<td></td>
</tr>
<tr>
<td>Oral</td>
<td>4–6</td>
<td>0.6</td>
<td>10</td>
<td>30</td>
</tr>
<tr>
<td></td>
<td>6–12</td>
<td>0.4</td>
<td>10</td>
<td></td>
</tr>
</tbody>
</table>

**Promethazine (Phenergan)**

It is a sedative, antihistaminic which is administered orally, IV or IM 0.25–0.5 mg/kg. Intramuscular route onset of action is 15–60 minutes with a peak at 1–2 hour and duration of 4–6 hours. Phenothiazines should be avoided in seizure prone patients as they lower the seizure threshold. It is not popular in pediatric ambulatory anesthesia because of dystonic reactions.

**Alpha-2 Adrenergic Agonists**

The major advantages of alpha -2-agonists are an absence of respiratory depression and fewer paradoxical reactions. Oral clonidine (3–4 mcg/kg) is effective as premedication but its slow onset (>60 min) and prolonged duration of action precludes its use in ambulatory pediatric settings. Intranasal dexmedetomidine 1 mcg/kg provides more effective sedation than oral midazolam 0.5 mg/kg or oral dexmedetomidine, 1 mcg/kg.\textsuperscript{18}

**Ketamine**

Ketamine has been used via oral (4–6 mg/kg), intramuscular (3–4 mg/kg) and intranasal (3–5 mg/kg) routes. It is usually used in combination with midazolam and atropine to avoid side-effects.\textsuperscript{19} Sedation is achieved within 10–20 minutes after oral ketamine, 5–10 minutes after IM and 10–15 minutes after intranasal routes. Duration of action is 30–60 minutes. A concentrated solution of preservative free ketamine (50 mg/mL) minimizes the volume administered in the nose. The common adverse reaction is postoperative vomiting which occurs in 33% of children.\textsuperscript{20}

**Opioids**

They provide analgesia and sedation during painful procedures. Their effects are dose dependent. Fentanyl is a potent analgesic with shorter duration of action and hence ideal in day care dental setup. Beware of rigid chest syndrome though usually not seen in procedural sedation.

**Propofol**

Propofol has a rapid onset of action, dose dependent levels of sedation with rapid return to consciousness. *Usual dose:* Loading dose of 2 mg/kg in infants/toddlers, 1 mg/kg in older children and then bolus of 1 mg/kg in younger or 0.5 mg/kg in older children until targeted sedation endpoint is reached. It has a narrow therapeutic range. Efficacy is excellent when used in conjunction with opiates or ketamine for short painful procedures. This should be administered only by persons trained in the administration of general anesthesia and who are
not simultaneously involved in surgical or diagnostic procedures. Full vigilance should be devoted to sedated patient. There is no reversal agent.

REVERSAL DRUGS

Flumazenil is usually reserved for reversal of respiratory depression caused by benzodiazepines. The recommended dose is 10 mcg/kg up to 0.2 mg every minute to a maximum cumulative dose of 1 mg intravenously. Onset time is 1–2 minutes and lasts 30–60 min. The child has to be monitored for at least 2 hours since re-sedation may occur after 1 hour.

Naloxone is an opioid antagonist, given IV or IM 0.01 mg/kg titrated to effect every 2–3 minutes with maximum 2 mg/dose. Onset time is 1–2 minutes and duration of action 20–40 minutes with IV and 60–90 minutes with IM route. The child has to be observed for a minimum of 2 hours as renarcotization can occur within 1 hour after naloxone.

GENERAL ANESTHESIA

Guidelines for general anesthesia (GA) management of pediatric patients referred for dental extractions are:

1. Dental extractions should be performed under GA only when it is considered to be the most clinically appropriate method of management.
2. Children undergoing GA for dental extraction should receive the same standard of assessment, preparation and care as those admitted for any other procedure under GA. They should be managed in a hospital setting that provides space, facilities, equipment and appropriately trained personnel required to enable resuscitation should the need arise. Agreed protocols and communication links must be in place both to summon additional assistance and for the timely transfer of patients to dedicated areas of critical care if necessary.
3. Unless contraindicated, NSAIDs and/or paracetamol should be used to provide analgesia for dental extraction under GA. These drugs may be combined or given separately before, during or after surgery. Opioid drugs are not routinely required for uncomplicated dental extractions.

Indications for GA

1. Extensive dental restoration planned on deciduous teeth in young children.
2. Neurological disorders, such as poorly controlled seizures, athetoid cerebral palsy or postencephalitic syndromes where patient movement is involuntary and uncontrollable.
3. Patients with communication disorders, e.g. autism, mental retardation, etc.
4. Allergy to local anesthetics.
5. Acute local inflammation limiting the effectiveness of local anesthetic agents owing to lower tissue pH.
6. Previous failure of LA or sedation.

PREOPERATIVE EVALUATION AND OPTIMIZATION

The aim is to optimize the child medically prior to anesthesia. History of birth, developmental milestones, previous illnesses and surgical interventions is obtained. The child’s emotional and psychological status is assessed and clinical examination performed. Patency of external nares, a deviated nasal septum, sinusitis, adenoids, loose teeth, enlarged tonsils should be evaluated. Patients with more than 50% of the pharyngeal area occupied by tonsils are at increased risk of developing airway obstruction. Children taking anti-seizure medication will generally benefit from preoperative assessment to ensure therapeutic levels. Those with severe underlying medical condition in categories ASA 3 or ASA 4 should be admitted to a pediatric ward and clinical care shared with a pediatric team. Children with a suspected syndrome should also be evaluated by the pediatrician and the anesthetist should plan the management accordingly.

Blood and biochemical investigations are as required for any other procedure under GA.

Presence of facial swelling due to infection or trauma may limit mouth opening.

Child should wear loose, comfortable clothing (preferably with opening in front to facilitate placement of ECG leads) and diapers. He should preferably be accompanied by two adults who are explained the possibility of hospital admission if need arises. Oral analgesics (paracetamol and/or NSAIDs) given an hour prior to the procedure are shown to reduce requirement of local anesthetics.

Antibiotic Prophylaxis for Bacterial Endocarditis

Children having cardiac defects (congenital or acquired) are believed to be at high risk for developing bacterial endocarditis as the dental procedure or nasotracheal intubation causes a transient bacteremia (Table 5, Box 1 and 2).
is planned. A rubber dam placed around the dental arch to prevent saliva from contaminating the surgical site. Dental impressions may be taken if orthodontic treatment is planned. A rubber dam placed around the dental arch to provide a dry environment and acts as a barrier which prevents entry of dental materials into the pharynx. The pedodontist places cotton rolls along the lingual, buccal, palatal and facial margins of the adjacent tissues. While extractions may not take very long, restorations which involve root canals, fillings and repeat intraoperative X-rays can prolong duration of anesthesia. Topical fluoride application with light cure may be performed as prophylaxis against caries. Premedication reduces airway secretions, blocks vagal reflexes and provides prophylaxis against pulmonary aspiration of gastric contents, in addition to allaying anxiety and facilitating induction. Dexamethasone in addition to antiemetic effect has anti-inflammatory action that reduces swelling, postoperative cough and sore throat. Induction of anesthesia can be preferably done on the parents lap. Regardless of the method of induction, IV access should be considered in all cases and obtained at the earliest opportunity. In cases where securing an intravenous route before inhalational induction is necessary EMLA (lidocaine 2.5% and prilocaine 2.5%) application on the skin 60 minutes prior is useful. It may cause blanching of skin which can make IV access difficult. The duration of action is 1–2 hours after the cream is removed; adverse reactions include erythema, itching, rash and methemoglobinemia. Short acting fast emergence agents, e.g. propofol, sevoflurane and atracurium are used unless contraindicated.

**Airway:** A child with a recognized syndrome associated with difficult airway may be best managed in an operation room where there are fiberoptic bronchoscopes or video laryngoscopes. It may be wise to also ensure the availability of an ENT surgeon competent to perform an emergency tracheostomy. Nasal intubation is preferred as it provides stability and unobstructed access to all four quadrants of the mouth, allowing the evaluation of tooth alignment and occlusion. Epistaxis is the most common complication with an incidence as high as 80% and adequate nasal preparation is necessary to prevent bleeding. Several methods have been described to reduce the incidence of traumatic nasal intubation including selection of the more patent nostril, use of lubricating gel, progressive dilatation with nasopharyngeal airways, thermosoftening of the tube, telescoping the tracheal tube into catheters, etc. Manual assisted ventilation after application of lidocaine jelly and xylometazoline drops ensures adequate nasal spread. Lidocaine gel decreases systemic absorption of vasoconstrictor and reduces postoperative nasal pain. North pole RAE tube is preferred. If not available, conventional endotracheal tube with Magills connector and catheter mounted connected to the pediatric circuit may be used. There should be no pressure around external nares while fixing the tube. One needs

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**Table 5: Regimens for dental procedures**

<table>
<thead>
<tr>
<th>Administer single dose 30 to 60 minutes before procedure</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Situation</strong></td>
</tr>
<tr>
<td>Oral</td>
</tr>
<tr>
<td>Unable to take oral medication</td>
</tr>
<tr>
<td>Allergic to penicillin or ampicillin—oral</td>
</tr>
<tr>
<td>Allergic to penicillin or ampicillin and unable to take oral medication</td>
</tr>
</tbody>
</table>

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**ANESTHESIA MANAGEMENT**

Communication with the dental surgeon about the procedure helps to plan anesthesia. After intraoral examination, radiographs of the teeth are usually obtained. Dental impressions may be taken if orthodontic treatment is planned. A rubber dam placed around the dental arch to

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Box 1: Cardiac conditions associated with the highest risk of adverse outcomes from endocarditis for which prophylaxis prior to dental procedures is recommended

- Prosthetic cardiac valve
- Previous bacterial endocarditis
- Congenital heart disease (CHD)
  - Unrepaired cyanotic CHD, including palliative shunts and conduits
  - Completely repaired CHD with prosthetic material or devices, whether placed by surgery or catheter intervention within the first 6 months after the procedure
  - Repaired CHD with residual defects at the site or adjacent to the site of a prosthetic patch or prosthetic device (which inhibit endothelialization)
- Cardiac transplantation recipients who develop cardiac valvulopathy

Box 2: Dental procedures for which endocarditis prophylaxis is/is not recommended for patients in Box 1

**Recommended:** All dental procedures that involves manipulation of gingival tissue or the periapical region of the teeth or perforation of oral mucosa

**Not Recommended**

- Dental radiographs
- Routine anesthetic injections through no infected tissue
- Placement of removable prosthetic or orthodontic appliance
- Adjustment of orthodontic appliance
- Placement of orthodontic brackets
- Shedding of deciduous teeth
- Bleeding due to trauma to lip and tongue

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to be vigilant as chances of disconnection are high with this arrangement. Silicone-based tubes may be superior to PVC tubes in prevention of epistaxis. A correct size uncuffed tube starts to leak at a positive airway pressure of 20 cm H2O. Usually, an endotracheal tube 0.5–1 mm less than that used for oral intubation is recommended for smooth and atraumatic passage of the nasal tube. This could result in inadequate airway seal. Formation of air bubbles in the oropharynx during the use of the irrigation drill may be disturbing to the pedodontist. This can be overcome by using a larger endotracheal tube, repacking the pharynx or may be using a microcuffed tube if possible. The National Patient Safety Agency advises that whenever a throat pack is inserted there should be visual and documented evidence of its presence.26 Oral route may be a throat pack is inserted there should be visual and documented evidence of its presence.26 Oral route may be used when nasal intubation is contraindicated to or avoid trauma to adenoid tissue in younger children. Preformed oral RAE tube provides access to either side of the mouth. The preformed orotracheal RAE tube is designed to be a midline tube, moving it to either side of the mouth may cause an eccentric position within the trachea. Reinforced tube resists kinking. Conventional endotracheal tube may be used, taking care to prevent endobronchial intubation as the tube is moved from one angle of the mouth to the other. Eye pads are used to prevent ocular injuries.

**Laryngeal Mask Airway:** An LMA makes the surgery difficult because it leaves little space for the dental drill and suction.27 Although, it protects the larynx from contents of the oropharynx to some extent, a throat pack is still required to absorb any blood and particulate matter. Displacement of the LMA may occur after insertion of the throat pack or positioning of the mouth prop. The flexible LMA is sometimes more difficult to insert in children, however this device allows more versatility and better access to teeth.

**Anesthesia Maintenance:** For short procedures and in cases where airway problems are anticipated, the anesthesia technique should allow maintenance of spontaneous ventilation. Oxygen with nitrous oxide and sevoflurane usually suffices. Incremental doses/continuous/target controlled infusions of propofol can be used for maintenance of anesthesia. For extensive and complicated restorations, it is better to use muscle relaxants and control ventilation.

**Extubation:** It is preferable to extubate awake in the lateral position and after the cough reflex has returned. Intravenous dexamethasone (0.4 mg/kg) and inhaled epinephrine may be used to reduce airway edema following extubation. Removal of the LMA while the child is still deeply anesthetized has been associated with lower oxygen saturation in dental patients.28 Classic recovery position without a pillow is ideal for keeping the airway open and preventing aspiration of blood and debris. Gauze which is left inside the dental cavities for hemostasis should be taped to the cheeks following extubation.

**Recovery**

A study of deaths related to dental anesthesia found that more than half occurred in recovery.29 Significant desaturation is common after brief dental anesthesia and the principal cause is airway obstruction. Oxygen supplementation ameliorates the severity of desaturation but does not prevent it.30 No oral fluids are given for 2–3 hours to avoid vomiting and aspiration. Ondansetron 100–150 mcg/kg is effective in lessening the severity of postoperative nausea and vomiting (PONV). Several scales to evaluate recovery have been devised and validated. A recently described simple evaluation tool may be the ability of the child to remain awake for at least 20 minutes when placed in a quiet environment.

**Postoperative Analgesia**

Extraction of deciduous teeth and conservative dentistry are not painful. Extraction of permanent teeth where a gingival flap and bone drilling are needed can be given opioids intraoperatively and paracetamol with NSAIDs continued orally in the postoperative period. Acetaminophen suppository (30–40 mg/kg) given shortly before the end of the procedure confers analgesia with minimal side effects. The nonsteroidal anti-inflammatory agent ketorolac (0.2–0.5 mg/kg) given IV as a single dose may be helpful. Regional blocks performed intraoperatively will alleviate immediate postoperative pain. A long-acting local anesthetic, e.g. bupivacaine is not recommended for the child or the physically or mentally disabled patient since the prolonged numbness may increase the risk of soft tissue injury.

**COMPLICATIONS**

**Arrhythmias:** They may occur during extraction of teeth; but are transient, seldom require treatment and respond to cessation of pull on the tooth.31 It can be attributed to elevated levels of catecholamines and stimulation of the sympathoadrenal system via trigeminal nerve during dental extraction. Other causative factors are hypoxia, hypercarbia, light anesthesia, use of halothane and epinephrine containing local anesthetics.

**Subcutaneous emphysema** of face and cervical areas, although rare, can occur due to the use of air driving ultra-high speed dental instruments. The air enters along
the mandibular periosteum at the operative site. \( N_0 \) should be discontinued on detection of emphysema and respiratory parameters closely monitored.\(^{32} \)

*Injury to the neck* may occur as a result of intraoperative positioning. Dislocation of temporomandibular joint may occur if the mouth is opened widely and can predispose to airway obstruction due to alteration in the position of the tongue.\(^{33} \) It can be easily reduced at the end of surgery.

**Dental complications of anesthesia:** Careful laryngoscopy is essential so as to avoid dislodgement of loose primary teeth. In case of lost tooth, gentle compression must be applied to the bleeding sockets. One should be cautious during laryngoscopy and oropharyngeal airway insertion. Excoriation or laceration of gum pads has been noted in patients with hypoplastic enamel defects in the primary maxillary incisors.\(^{34} \) If an anesthetist is using the incisors to accomplish mouth opening before laryngoscopy, there are chances for dental avulsion. Hence molars by virtue of their dental stability should be used for effective mouth opening.\(^{35} \) Children with conditions like amelogenesis imperfecta or dentinogenesis imperfecta may have dental fractures even with the most careful manipulation.

**Hyperthermia:** Tissue destruction, environmental temperature during surgery, administration of atropine, dehydration and bacteremia have all been implicated in fever following dental anesthesia.\(^{36} \) Procedures provoking bacteremia (e.g. extractions) can be managed by routine administration of antibiotics.

**Operating Room Pollution:** Air pollution due to anesthetic gases is common as scavenging systems and efficient ventilation with more than 12–15 air changes per hour are usually not present.

**Postoperative nausea and vomiting:** Gastric irritation from swallowed blood is a common cause and may be prevented by gently suctioning the stomach prior to extubation. Abdominal distension during bag-mask ventilation and use of opioids are contributory factors. A cause of nausea and emesis unique to dentistry is the inadvertent ingestion of intraoperatively administered topical fluorides used to reduce dental caries.\(^{37} \)

**Emergency agitation:** Several factors including pain, personality traits of the child, type of surgical procedure, too rapid awakening, etc. have been implicated as etiology of emergence delirium. Studies demonstrate that regional block, opioids, NSAIDS decrease the incidence, however, it has been found to occur even after adequate pain relief or procedures not associated with pain.\(^{38} \) Meta-analysis revealed that sevoflurane more often resulted in emergence agitation than did halothane in pediatric patients. Addition of ketamine 0.25 mg/kg at the end of sevoflurane anesthesia has been tried to decrease its incidence and severity without increasing time to meet recovery room discharge criteria.\(^{39} \) Rapid awakening after propofol has not been associated with emergence.\(^{40} \) Many patients are intolerant of having an IV or monitors attached once alert. They might be removed when the necessary doses of analgesics, antiemetics and antibiotics have been given. Parental presence in the room on awakening may have a calming effect.

**Discharge Criteria**

The child is fit to go home when the following criteria are met:

1. Cardiovascular and psychomotor parameters have returned to preoperative status
2. Airway patency is uncompromised and satisfactory
3. Patient is easily arousable and protective reflexes are intact
4. State of hydration is adequate
5. Able to swallow and retain water/juice/ice cream
6. No pain, no active bleeding from the sockets
7. Able to void urine.

The child is advised ice application for facial swelling, a soft smooth diet, nothing too warm or too cold to avoid discomfort and further bleeding. The accompanying adult is informed that after GA, there is a period of about 24 hours in which the child’s judgment, performance and reaction time are affected even though the child may feel normal. The child should not be allowed to do anything potentially dangerous, e.g. swimming, cycling, etc. and should remain in immediate care of a responsible adult.

The time and condition of the child at discharge should be documented. Some sedation medications are known to have a long half-life and may delay a patient's complete return to baseline or pose the risk of resedation; these patients might benefit from a longer period of less-intense observation (e.g. a stepdown observation area) before discharge from medical supervision.

**Solving Common Problems**

**Cerebral Palsy**

Children of cerebral palsy with coexisting mental retardation often present for dental treatment.

- Obtaining intravenous line may be a little tricky due to spasticity, dystonia or simply refusal
- Children may be dehydrated due to abnormal cognitive response to thirst in addition to prolonged preoperative fasting
Positioning in the dental chair may be difficult due to fixed contractures and involuntary movements and special care should be taken to avoid nerve and muscle damage (Fig. 3). Minimize lights, sound and sudden movements that trigger primitive reflexes or uncontrolled movements.

Latex allergy in these children occurs probably as a result of the many operative procedures to which they are exposed.

Intraoperative hypothermia secondary to hypothalamic dysfunction may be compounded by a lack of muscle and fat deposit in the malnourished child.

The airway may be compromised due to excessive secretions (combination of hyperactive salivary glands and disturbed coordination of orofacial and palatolingual muscles), reactive airway disease, regurgitation and silent aspiration.

Physical endurance cannot be assessed due to neurological impairment and respiratory pathology may go unnoticed.

Visual defects like cortical blindness and hearing problems can contribute to postoperative irritability.

Approximately 30% of these children have coexisting epilepsy. Children on anticonvulsants may be unable to have their medication due to postoperative nausea and vomiting. Most anticonvulsants however have a long elimination half-life of 24–36 hours and if their levels are in the therapeutic range, a 24 hours period can elapse without significantly increasing the risk of seizures.

Certain inhaled volatile anesthetics (sevoflurane), local anesthetics (lidocaine, bupivacaine), opioids (fentanyl, alfentanil, sufentanil, meperidine) and hypnotics (propofol, etomidate, ketamine) are known to lower the seizure threshold. The MAC of halothane is 0.9 in healthy children, in children who have CP it is 0.71, children with CP on anticonvulsants have an even lower MAC of 0.63.

Botulinum neurotoxin injected into hamstrings or gastrocnemius muscle (to decrease spasticity) has onset of effect in 12 hours to 7 days with effect for 2 to 6 months. Generalized weakness from systemic toxicity is rare. Potentiation of muscle relaxants has not been substantiated clinically.

Baclofen (an agonist at GABA baroreceptors in the dorsal horn of the spinal cord) is used to reduce pain associated with muscle spasms and may delay development of contractures. Most patients are on oral baclofen which crosses the blood brain barrier poorly. Intrathecal baclofen delivered through pumps reduces spasticity at lower doses than are required orally with fewer side-effects. Drug overdose may produce drowsiness, depressed respiration and progressive hypotonia or loss of consciousness. Abrupt withdrawal from oral or intrathecal baclofen may result in seizures, hallucinations, disorientation, dyskinesia and itching with symptoms lasting upto 72 hours.

CONGENITAL BLEEDING DISORDERS

Patients with Hemophilia A (deficiency of factor VIII), Hemophilia B (deficiency of factor IX), Von Willebrand Disease (deficient or abnormal plasma protein Von Willebrand Factor), Factor XI deficiency, etc. are at increased risk of significant bleeding from invasive dental procedures. Factor replacement therapy may be provided on a prophylactic basis to prevent bleeds or on demand.
when a bleed occurs. Since the factor levels decline rapidly, the procedure should be performed within 30–60 minutes of administration of factor concentrate. The synthetic antidiuretic hormone desmopressin stimulates release of endogenous FVIII and vWF from the stores in patients with mild hemophilia and vWD. DDAVP can be administered one hour pre-procedure subcutaneously or intravenously. Antifibrinolytic agents like tranexamic acid (IV, oral and mouthwash) has been tried perioperatively.42

**LEARNING POINTS**

- Children for dental rehabilitation should be assessed preoperatively in order to investigate and plan the appropriate anesthesia management.
- Conscious sedation may be used as an alternative to general anesthesia for dental treatment in older children. Vigilant monitoring is mandatory, as hypoventilation and upper airway obstruction can occur with changing levels of sedation.
- Practitioners who sedate patients must be skilled in advanced airway management and pediatric advanced life support. The sedation/anesthesia provider should not be the person performing the procedure.
- Insertion and removal of the throat pack should be documented.
- Good medical backup should be available for both preoperative and in case of postoperative complication.

**ACKNOWLEDGMENT**

The authors wish to thank Dr Aadesh Kakade, Head, Department of Pedodontia, Nair Dental Hospital, Mumbai, Maharashtra, India.

**REFERENCES**

Anesthesia for Ophthalmic Procedures

Chapter 19

INTRODUCTION

Main ocular pathologies in children needing surgery include strabismus, congenital and traumatic cataract, glaucoma, orbital tumors, nasolacrimal duct obstruction, and penetrating eye injuries. These children need multiple general anesthesia exposures for diagnosis, treatment and further evaluation of the disease.

Due to early diagnosis, preterm infants have been coming regularly for eye examination, laser and surgery for retinopathy of prematurity (ROP), cataract, glaucoma etc.

Apart from concerns of pediatric age group, children coming for ophthalmic surgery may have different sets of problems, i.e. congenital anomalies, syndromes, limited vision, mental retardation, difficult airway etc. Skillful anesthetic management should be done, as complications can be life-threatening or vision threatening. It is necessary to understand physiological ocular responses as well as the interaction of ophthalmic drugs and anesthetic agents to prevent complications.

PATHOPHYSIOLOGY

The knowledge of eye anatomy, physiology of intraocular pressure (IOP) and effects of anesthetic drugs on IOP, systemic effects of the ophthalmic drugs, mechanism of various ocular reflexes, effects of surgical manipulation are important for proper anesthetic management during ophthalmic surgery.

Intraocular Pressure

The most important influences on IOP are movement of aqueous humor, changes in choroidal blood volume, central venous pressure and extraocular muscle tone. A rise in the IOP decreases intraocular volume by causing drainage of aqueous or extrusion of vitreous through the wound, which can lead to permanent visual loss (Table 1 and Box 1).

Oculocardiac Reflex

Oculocardiac reflex (OCR) occurs during ocular procedures like strabismus surgery, enucleation, scleral banding, vitreoretinal surgery, orbital block and ocular compression. It can lead to cardiac dysrhythmia (bradycardia, ventricular ectopics, sinus arrest, ventricular fibrillation) (Fig. 1). Hypercapnia increases its sensitivity. Routine prophylaxis with anticholinergic is controversial. It is self-extinguishable with repeated traction on the extraocular muscles. The management of OCR is listed in Box 2.

Table 1: Intraocular pressure variations

<table>
<thead>
<tr>
<th>IOP (mm Hg)</th>
<th>At birth</th>
<th>5 years</th>
<th>Diurnal variation</th>
<th>Blinking</th>
<th>Squeezing</th>
<th>Open globe (traumatic perforation, surgery)</th>
</tr>
</thead>
<tbody>
<tr>
<td>9.5</td>
<td>10–20</td>
<td>Increase by 3–6</td>
<td>Increase by 5</td>
<td>Increase by 26</td>
<td>Atmospheric pressure</td>
<td></td>
</tr>
</tbody>
</table>
Oculorespiratory Reflex (ORR) afferent arc is same as in OCR, the efferent arc is from pneumotaxic center in the pons and the medullary respiratory center. ORR results in shallow breathing, bradypnea, tachypnea or respiratory arrest and is commonly seen in strabismus surgery. Atropine has no protective effect.6

Oculoemetic Reflex (OER) is a vagus mediated response to surgical manipulation of extraocular muscles and is responsible for the high incidence of postoperative nausea and vomiting (PONV) associated with strabismus surgery.6

ANESTHETIC IMPLICATIONS OF DRUGS USED IN OPHTHALMOLOGY: TOPICAL AND SYSTEMIC (TABLE 2)

Topical ocular drugs trickle through the punctum into the nasolacrimal duct and are absorbed through the nasal mucosa into the systemic circulation. Infants and children are more susceptible due to lack of availability of lower concentration of ophthalmic eye drops. Systemic effects can be minimized by use of micro dropper, instillation of 1–2 drops, occlusion of punctum during instillation of drops, instillation of drops towards lateral canthus, lower concentration, wiping of excess drug, increase in viscosity of drug, use of alternative drugs etc.7

Phenylephrine (10%, 5%): A single drop of phenylephrine 10% contains 4 µg of drug hence 2.5% solution is recommended in children. If beta blocker is used in response to iatrogenic hypertension, it induces unopposed alpha adrenergic stimulation, can exacerbate symptoms and produce life-threatening consequences.10 Systemic side effects of phenylephrine should be managed with titration of anesthetics, opioids, vasodilator and reduction of undesired autonomic reflex responses.11

Box 1: Factors affecting IOP

Factors increasing IOP
- Obstruction to aqueous humor outflow
- External pressure on the eye (tightly fitted face mask)
- Raised venous pressure (coughing, vomiting, valsalva maneuver)
- Increased choroidal blood volume (respiratory acidosis, hypoxia, hypercarbia, hypertension)
- Rise in the sphere content (injection of large volume of local anesthetic during block)
- Decrease in the size of the globe
- Succinylcholine: Due to the contraction of extraocular muscles during fasciculation. The effect is maximal at 2–4 minutes returning to normal within 7 minutes
- Ketamine
- Laryngoscopy and endotracheal intubation: increases IOP (10–20 mm Hg)

Factors lowering IOP
- Reduced venous pressure (Head up)
- Lowered arterial pressure (systolic pressures <90 mmHg), hypocarbia
- Intravenous induction agents (except ketamine), inhalational agents, nondepolarizing muscle relaxants
- Reduction in aqueous volume (acetazolamide which inhibits production)
- Reduction in vitreous volume (mannitol which exerts osmotic effect)

Box 2: Management of OCR

- Temporary cessation of surgical stimulation until heart rate increases
- Ensure adequate ventilation, oxygenation and depth of anesthesia
- IV atropine 10–20 µg/kg or glycopyrrolate 10 µg/kg as prophylaxis or treatment
- Infiltration of the rectus muscle with local anesthetics (strabismus surgery)7
- Regional block
ANESTHESIA GOALS
Apart from the general goals while anesthetizing children, other goals are shown in Box 3.

ANESTHESIA TECHNIQUE

Premedication
Midazolam, ketamine, dexmedetomidine, clonidine have been used for premedication in children. Darlong et al. concluded that combination of midazolam (0.25 mg/kg) with oral ketamine (3 mg/kg) provided earlier sedation with less time taken for parental separation, and recovery with minimal side effects in comparison to oral midazolam (0.5 mg/kg) alone, or oral ketamine (6 mg/kg) in children planned for ophthalmic surgery.

Monitoring and oxygen supplementation facility should be available in the preoperative room. Effect of clonidine and midazolam premedication on prevention of PONV and emergence delirium is conflicting.

Induction
These children usually do not have intravenous access preoperatively. School going children should be asked for their preference about needle prick or acceptability for face mask. In a child with normal airway, both inhalational induction (sevoflurane or halothane) or intravenous induction (propofol or thiopentone) can be performed.

In intellectually disabled or blind children with normal airway, premedication can be administered in the preoperative room along with eutectic mixture of local anesthetics (EMLA) application on the dorsum of both hands. Both, inhalational or intravenous (IV) induction can be chosen depending on the anesthesiologist’s choice. Presence of parents in the operating room is debatable. However, in children with intellectual disability

<table>
<thead>
<tr>
<th>Name of drug</th>
<th>Class</th>
<th>Route of administration</th>
<th>Concentration</th>
<th>Action</th>
<th>Side effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atropine</td>
<td>Anticholinergic</td>
<td>Topical (drop/ointment)</td>
<td>0.5%, 1%</td>
<td>Mydriasis</td>
<td>Tachycardia, fever</td>
</tr>
<tr>
<td>Tropicamide</td>
<td>Anticholinergic</td>
<td>Drops</td>
<td>0.5%</td>
<td>Mydriasis</td>
<td>Dry mouth, drowsiness, tachycardia</td>
</tr>
<tr>
<td>Phenylephrine</td>
<td>Alpha agonist</td>
<td>Drops</td>
<td>10%, 5%, 2.5%</td>
<td>Mydriasis</td>
<td>Transient hypertension, bradycardia, pulmonary edema, cardiac arrest</td>
</tr>
<tr>
<td>Adrenaline</td>
<td>Catecholamine</td>
<td>Topical (Infusion bottle)</td>
<td>0.1 mg in 500 mL ringer lactate</td>
<td>Mydriasis</td>
<td>Tachycardia, hypertension, tachyarrhythmias</td>
</tr>
<tr>
<td>Timolol</td>
<td>Non-selective beta blocker</td>
<td>Drops, gel</td>
<td>0.25%, 0.5%</td>
<td>Reduces IOP</td>
<td>Bradycardia, hypotension, congestive heart failure, exacerbation of asthma</td>
</tr>
<tr>
<td>Betaxolol</td>
<td>Cardioselcive beta blocker</td>
<td>Drops</td>
<td>0.25%</td>
<td>Reduces IOP</td>
<td>Bradycardia, sinus arrest</td>
</tr>
<tr>
<td>Pilocarpine</td>
<td>Parasympathomimetic</td>
<td>Drops</td>
<td>1%</td>
<td>Reduces IOP</td>
<td>Bronchospasm, bradycardia, increased mucous secretion</td>
</tr>
<tr>
<td>Mannitol</td>
<td>Osmotic diuretic</td>
<td>Intravenous</td>
<td>20% 0.5 gm/kg</td>
<td>Reduces IOP</td>
<td>Hypervolemia, electrolyte imbalance, CHF</td>
</tr>
<tr>
<td>Acetazolamide</td>
<td>Carbonic anhydrase inhibitor</td>
<td>Tablet</td>
<td>8–30 mg/kg/day</td>
<td>Reduces IOP</td>
<td>Metabolic acidosis, hypokalemia, dehydration</td>
</tr>
<tr>
<td>Dorzolamide</td>
<td>Carbonic anhydrase inhibitor</td>
<td>Topical</td>
<td>2%</td>
<td>Reduces IOP</td>
<td>Headache, strange taste in mouth, dizziness</td>
</tr>
</tbody>
</table>

Abbreviation: CHF, congestive heart failure

Box 3: Goals of pediatric ophthalmic surgery

- Complete ocular akinesia
- Smooth induction
- Prevention of oculocardiac reflex
- Prophylaxis and treatment of PONV
- Maintenance of IOP and end tidal carbon dioxide (EtCO2) within normal range especially in intraocular surgery
- Caution about interaction of ophthalmic drugs with anesthetic drugs
- Prevention and management of side effects of topical ophthalmic drugs
- Prevention of ocular pressure by using proper size face mask
- Maintenance of asepsis
- Multimodal analgesia to prevent perioperative pain
- Extubation without coughing & bucking

Abbreviations: PONV, postoperative nausea and vomiting; IOP, intraocular pressure
or blindness, if possible, parents should accompany the child to the operating room.18

If child’s airway is difficult, along with other systemic anomalies, IV access can be secured in the preoperative room after EMLA application and titrated dose of inhalational or intravenous agent can be administered for induction.19

Intravenous induction is preferred in the child with big orbital mass due to chances of compression of the mass with face mask.

**Induction Agents**

**Thiopentone** reduces IOP by about 40% of base line by its central depressive effect and improves outflow of aqueous humor.

**Propofol** reduces IOP and limits its increase during intubation. Rapid onset and short duration of action of propofol ensures optimal titration for total intravenous anesthesia. It has a low incidence of side effects and PONV.

**Ketamine** should be avoided in open eye injuries, as a sole agent. It can be used with small doses of benzodiazepine to blunt its excitatory effects and ventilation should be controlled with a muscle relaxant for IOP control. Nystagmus with contraction and squeezing of the eyelids limits its use in ophthalmology.

**Inhalational Agents**

Inhalational anesthetics decrease IOP in proportion to the depth of anesthesia due to drop in blood pressure, which reduces choroidal volume; relaxation of the extraocular muscles lowers wall tension; pupillary constriction facilitates aqueous outflow; and an effect on the hypothalamic centers in the brain. The reduction in IOP is greater with controlled ventilation.

**Airway Management**

Endotracheal intubation and various supraglottic devices (SGD) have been used for ophthalmic surgeries.20,21 Among supraglottic devices flexible LMA provides a better surgical field for the surgeon as it can be taped on the chin (Fig. 2).21

Supraglottic devices can also be used for fiberoptic guided intubation in children with difficult airway without the need for laryngoscopy and danger of hypoxia.22–25 Videolaryngoscopes are also helpful in difficult airway with an experienced anesthesiologist. Face mask can be used for short ocular examination under anesthesia. However, ocular pressure should be avoided with face mask as it can lead to spurious IOP readings.

**Neuromuscular Blockers**

Administration of neuromuscular blockers (NMB) in ophthalmic surgery is the anesthesiologist’s choice especially with the increased use of SGD. In children with muscular dystrophy and short surgery NMB can be avoided. NMB are required in oculoplasty surgery and viteroretinal surgery. NMB is administered in preterm infants undergoing retinopathy of prematurity (ROP) surgery as they are usually intubated in view of prolonged surgery and possibility of need for postoperative ventilation.26

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Fig. 2: Difference between flexible LMA and classic LMA in ophthalmic surgery
Succinylcholine increases IOP, principally through prolonged contracture of extraocular muscles, due to congestion of the choroidal vessels and distortion of the globe with axial shortening. IOP increase will cause spurious IOP measurements during examination under anesthesia, and may cause extrusion of ocular contents through an open surgical or traumatic wound. The prolonged contracture of the extraocular muscles may lead to abnormal forced duction test for 20 minutes and may influence the type of strabismus surgery performed.

Precurarization with nondepolarizing blockers has little or no effect on this increase. However other factors, such as inadequate anesthesia, elevated systemic blood pressure, and insufficient neuromuscular blockade during laryngoscopy, and tracheal intubation might increase IOP more than succinylcholine.

Nondepolarizing muscle relaxants either reduce IOP or have no effect on it. Atracurium has no significant effect whilst vecuronium produces a small but significant reduction in IOP. Rocuronium in a dose of 0.9–1.2 mg/kg can be used for intubation in open eye injuries to prevent rise in IOP.

Maintenance

Both total intravenous anesthesia (TIVA) (propofol and fentanyl/remifentanil) and balanced inhalation anesthesia [oxygen, nitrous oxide/air, sevoflurane/isoflurane/desflurane] can be used depending on availability and anesthesiologist’s choice.

In healthy adults, there is no change in IOP with the use of nitrous oxide, however, its effect in children is unclear.

Analgesia

It can be provided with opioids, non-steroidal anti-inflammatory drugs (NSAIDs), regional blocks, topical anesthesia depending on the type of surgery.

Opioids

Intravenous administration of potent opioids (fentanyl and remifentanil) results in a significant reduction in IOP. A combination of fentanyl and droperidol also reduces the IOP by 12% in normocapnic patients.

Topical Ophthalmic Anesthesia

Topical anesthesia has been used in cataract and strabismus surgery and can be administered via eye drops, eye drops plus intracameral anesthesia and gel anesthesia. The main advantages of topical anesthesia are no complication of needle block and low cost.

Regional Block

Peribulbar block after general anesthesia has been administered to reduce the opioid requirement, to decrease OCR incidence and PONV. They are safe and have minimal complications in children.

Technique of peribulbar block: Two injection or one injection technique is performed similar to adult block. Total volume of local anesthetic is 0.3 mL/kg. A 26 G, 1/2 inch hypodermic needle is used and half volume is administered just superior to the infraorbital rim in the inferotemporal quadrant and rest half of the volume is injected just lateral to the supratrochlear notch, beyond the equator of the globe. The authors use only infraorbital approach to provide intraoperative analgesia.

Sub-Tenon’s block has been administered in ophthalmic surgeries and laser for ROP. Sub-Tenon’s block is devoid of needle complications and needs less amount of local anesthetic agent. Chemosis and subconjunctival hemorrhage are the main side effects.

Technique of sub-Tenon’s block: Sub-Tenon’s space is commonly accessed through the inferonasal quadrant as it allows good fluid distribution superiorly while avoiding area of surgery and damage to the vortex veins. After instillation of topical local anesthetics, at 5–7 mm away from the limbus, conjunctiva and Tenon’s capsule are gripped with non-toothed forceps (Moorfield forceps). A small incision is made with Westcott scissors to expose the sclera. A blunt 19G, 25 mm curved cannula is inserted through the incision beyond the equator of the globe and local anesthetic is injected [1.5–2.0 mL (children aged 5–10 year) and 2.0–3.0 mL (older children)].

Extubation

Extubation or removal of SGD should be without coughing and straining to prevent increase in IOP and bleeding from the surgical site. Deep extubation in lateral position can be done. Small dose of propofol or lignocaine can be administered at the time of extubation. Operative side should be kept up in the lateral position to prevent pressure on the eye.

CLINICAL PRESENTATION, SURGICAL PROCEDURE AND ANESTHESIA MANAGEMENT

Cataract (congenital, traumatic): Children may have white reflex which can be appreciated by parents or pediatrician. Congenital cataracts are usually bilateral and commonly associated with systemic disease and syndromes. Nystagmus may be present (Fig. 3).
Surgery should be performed as early as possible. In children, lens aspiration is done along with the anterior vitrectomy. Intraocular lens implantation is usually done after the age of 2 years. Good mydriasis is needed for lens aspiration and topical ophthalmic drugs (atropine, tropicamide, phenylephrine) are used preoperatively.

Anesthesia should provide complete akinesia and meticulous control of IOP with controlled ventilation. Opioid, topical lignocaine gel, sub-Tenon’s block, peribulbar block have been used for intraoperative analgesia. Postoperative pain after cataract surgery is minimal and can be managed with NSAIDs.

Glaucoma: Infantile glaucoma can develop within the first 3 years of life and has the classic triad of tearing, photophobia and blepharospasm. Macrocornea and hazy cornea is seen in children. It may be associated with congenital abnormalities, i.e. craniofacial dysostosis, chromosomal trisomies, Sturge-Weber syndrome, Crouzon syndrome etc (Figs 4A and B).

General anesthesia is required for surgery and IOP measurement repeatedly to titrate antiglaucoma drugs. If medical management fails, trabeculectomy with trabeculotomy and mitomycin C application is performed. Side effects of antiglaucoma drugs should be ruled out.

The aim of anesthetic management in glaucoma surgery is to maintain IOP within the normal range and prevent its increase during anesthetic procedure, i.e. laryngoscopy, intubation, extubation. Both endotracheal intubation and extubation lead to increase in IOP especially during coughing and straining on endotracheal tube which may lead to visual damage. SGD provides definitive advantage as it does not increase IOP during insertion and provides smooth extubation. Postoperative pain is minimal.

Strabismus: Strabismus is a misalignment disorder of extraocular muscles characterized by amblyopia with or without anisometropia (Fig. 5). Strabismus may be inherited, developmental or acquired and can be associated with comorbidities particularly neuromuscular disorders, cerebral palsy, undiagnosed cardiomyopathy. There may be an increased risk of malignant hyperthermia (MH).

Rectus muscles and oblique muscles resection or recession is done to correct the alignment of eye. Strabismus surgery can be done as early as 6 months of age.

Strabismus surgery has increased incidence of OCR, ORR, OER and postoperative nausea and vomiting (PONV). Incidence of OCR is more with traction on medial rectus. Succinylcholine and halothane should be avoided to reduce the risk of MH. Body temperature, electrocardiogram (ECG) and EtCO₂ should be monitored. TIVA is an alternative to inhalational anesthesia in children susceptible to MH and PONV.

Awake-sleep-awake technique has been used for pediatric adjustable suture strabismus surgery under propofol-sufentanil and propofol-remifentanil anesthesia. Propofol based anesthesia with peribulbar...
Socket reconstruction (microphthalmos, anophthal- mos), sling surgery (ptosis) and skin grafting (lid colo- boma) is done. Premedication should be administered to allay anxiety. The tumor may be large with proptosis. Face mask should be soft with proper fit as poor fitting mask will lead to pressure on the eye. In case of orbital swelling, triangular Rendell-Baker mask is better as it hugs the bridge of the nose and tapers away from the eyes.

Children with big ocular tumor also come for biopsy to confirm the diagnosis. Face mask application after the biopsy is difficult in these cases as any pressure on the eye can lead to bleeding from the tumor. Eye protection should be done during mask ventilation to prevent injury (Fig. 9).

Enucleation and oculoplasty surgeries are more extensive. Extent of surgery should be discussed with the ophthalmologist as intraoperative bleeding is more in some ocular tumors, which can trickle through nasolacrimal duct (NLD) into the oropharynx. In case of breach in medial or inferior orbital wall, blood can

Retinoblastoma: In children, retinoblastoma is the predominant primary eye neoplasm. In early stage, they have cat eye reflex (intraocular), however in later stages there may be big orbital mass (extraocular)(Figs 6A and B).

Children with retinoblastoma are anxious due to repeated visits to the hospital for fundus examination, ultrasound, radiological imaging, laser, cryotherapy, thermotherapy and surgery (enucleation, socket reconstruction). These children are on chemotherapy and immunocompromised and have difficult venous access. Complete blood count should be done before surgery as they are prone to infections. View vein is helpful in difficult IV cannulation (Fig. 7).

Incidence of OCR is high and intraoperative blood loss may be significant during enucleation and exenteration surgery. Postoperative pain is significant.

**Oculoplastic Disorders**

They include anophthalmos, microphthalmos, cryptophthalmos, ptosis, lid coloboma, lymphangiomas, nasolacrimal duct (NLD) obstruction, dermoid, burn etc. These children may also have other congenital anomalies (Figs 8A to C).
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In both the cases endotracheal intubation with oropharyngeal packing should be done to prevent chances of aspiration. A multimodal analgesia with opioid and NSAIDs should be administered to reduce perioperative pain.

In case of NLD blockade, initially syringing and probing is tried. Methylene blue mixed saline is injected through either of lid punctum to check patency of the NLD. If saline reaches into ipsilateral nasal cavity then procedure is successful. In case of failure, dacryocystorhinostomy (DCR) is performed.

Children for syringing and probing usually come on day care basis. Endotracheal intubation and SGDs both can be used with continuous suction through ipsilateral nasal cavity or pharynx. Child should be positioned with a pillow under the shoulders to divert irrigation fluid away from the larynx. Chances of respiratory complications are more during this procedure.

For dacryocystorhinotomy, topical vasoconstrictors are used to minimize bleeding at the nasal mucosa and endotracheal intubation along with pharyngeal packing is required. Perioperative analgesia should include opioid, infraorbital block and NSAID, as it is painful.

Vitreoretinal Disorders
Preterm infants may develop retinopathy of prematurity (ROP). It is a disease of neovascularization of the retina.
and may result in poor visual acuity, retinal detachment, amblyopia and ultimately blindness. Children with Marfan syndrome, Kniest syndrome and Stickler syndrome may also have vitreoretinal pathologies.

Initial stages of ROP can be treated with laser photocoagulation, however stage IV, and V need vitreoretinal surgery. These premature babies have higher incidence of bronchopulmonary dysplasia, cardiac anomalies, episodic bradydysrhythmias, anemia, intraventricular hemorrhage and necrotizing enterocolitis. They are prone to hypothermia, bradycardia and apnea postoperatively.

Perioperative risk of apnea is dependent on postconceptual age, gestational age and prior history of apnea at home. Postoperatively, preterm infants should be observed with pulse oximetry and apnea—monitoring as inpatient setting. A pediatric transport team and facility for postoperative ventilation should be arranged before the day of surgery.

Infants with ROP usually come for laser under anesthesia, intravitreal injection and vitreoretinal surgery depending on the ROP stage. Endotracheal intubation is preferred as they may need postoperative ventilation. Short acting opioid, paracetamol with topical and regional anesthesia is preferred to prevent postoperative apnea.

Laser treatment for ROP has been done under sub-Tenon’s block successfully without the need for intubation. Topical anesthesia, ketamine, morphine, endotracheal intubation has also been used with or without intubation for laser surgery. LMA and sevoflurane anesthesia has also been used safely for intravitreal injection and photocoagulation.

Other vitreoretinal procedures include scleral buckling, retinopexy, pars plana vitrectomy, vitrectomy with silicon oil insertion etc.

$N_2O$ should be avoided in retinal detachment surgery as sulfur hexafluoride ($SF_6$) or perfluoropropane ($C_3F_8$) gas are injected in the posterior chamber to create tamponade. These gases are inert, water insoluble and poorly diffusible. $N_2O$ is 34 times more soluble than nitrogen and 117 times more soluble than $SF_6$ and rapidly diffuses into the intraocular gas bubble and causes rapid expansion with subsequent rise in IOP. This may lead to retinal artery occlusion, retinal ischemia and eventually visual loss. If $N_2O$ administration is continued even after gas injection within 19 minutes, IOP increases from 14 to 30 mm Hg and both bubble size and IOP decreases (from 29 to 12 mm Hg) within 18 minutes of discontinuation of $N_2O$. This rapid and wide variation in bubble size during general anesthesia may adversely affect the outcome of surgery. Administration of $N_2O$ should be discontinued at least 20 minutes before an intravitreal injection of gas. It is preferable to avoid $N_2O$ altogether when intravitreal injection of gas is planned. $SF_6$ gas bubble remains for at least 10 days. Other intravitreal gases may remain for as long as 21 to 28 days. It is recommended to avoid nitrous oxide within 3 to 4 weeks of surgery with vitreoretinal injection of gas as a second exposure to $N_2O$ might cause re-expansion of the bubble and elevate IOP.

Recent data suggest that $N_2O$ should be avoided altogether for patients who have recently undergone retinal surgery, unless there is evidence by indirect ophthalmoscopy that the gas has been totally reabsorbed.

Corneal Surface Disorders

Children with Steven-Johnson syndrome, burn, acid injury, alkali injury, keratomalacia, corneal opacity, dry cornea may also need surgery.

Salivary gland implantation is done in Steven-Johnson syndrome cases and requires nasal intubation with oropharyngeal packing. Keratoprosthesis is performed for corneal surface disorders. Patient with burns and difficult airway may also need nasal intubation.

Keratoplasty

There are different types of keratoplasties. Donor cornea is transplanted on the patient cornea. Type of keratoplasty depends on the availability of tissue and requirement of the patient. IOP should be controlled to obtain a better outcome.

Examination Under Anesthesia

In children general anesthesia is required for ocular disease evaluation, refraction, IOP measurement, ultrasonography, fundus examination, suture removal, corneal tattooing, etc.

These children usually come as day care for eye examination. Proper preoperative evaluation and fasting status should be checked. Associated anomalies and adequate control of other systemic anomalies should be recorded. Previous anesthesia records should be seen before anesthesia. Both, intravenous or inhalation anesthesia can be used depending on the anesthesiologist’s choice. Sevoflurane is preferred for inhalational induction, while propofol is used for intravenous induction.

Face mask and SGD can be used as airway device depending on the duration and type of procedure. Face mask holding can be modified by lifting the chin to facilitate eye examination (Fig. 10). For short examination,
insertion of an intravenous cannula is debatable.\textsuperscript{56,59} Spontaneous or assisted ventilation with $O_2$, $N_2O$/air and inhalational agent (1.0–1.3 MAC) is preferred.

**Emergency Eye Surgery**

It can be traumatic or chemical burn eye injury, endophthalmitis, foreign body and should be treated urgently to prevent visual loss.\textsuperscript{1,6,8} It may be associated with head injury or injuries of other organs.

Corneal and or scleral perforation repair, globe repair, lid repair is performed depending on the extent of injury. Decision for delay in surgery should be taken after discussing the degree of urgency with the ophthalmologist, to prevent the risk to the eye. Penetrating injuries may need to be dealt more urgently due to the risk of infection, endophthalmitis, vitreous loss and retinal detachment. Proper preoperative evaluation should be done to rule out any other medical or surgical disease and other injuries. Comorbidities should be optimized prior to surgery if time allows.

Fasting for solid food and clear fluids will depend on the age of the child. Time interval between the last meal and the time of the injury is also important. In case of urgent surgery, full stomach child should be induced with rapid sequence induction technique. There is always a caution for the use of succinylcholine with open globe due to increase in IOP. Rocuronium can be used if the child has normal airway. SGD with gastric drain or endotracheal intubation can be done depending on the anesthesiologist’s choice. Deep extubation can be done if the airway is normal, to prevent increase in IOP.

**MONITORING**

Standard monitoring including ECG, heart rate (HR), arterial oxygen saturation ($SpO_2$), noninvasive blood pressure (NIBP), $EtCO_2$ is routinely applied for ophthalmic surgery. In case of SGD, cuff pressure monitoring can be used. Bispectral index, entropy and neuromuscular monitoring can be used according to the anesthesiologist’s choice and individual case requirement.

Temperature monitoring and blood glucose monitoring is done in preterm infants. Radiant warmer, second pulse oximeter is also used in small babies. Monitoring is difficult in ophthalmic surgery as airway is away from the anesthesiologist, and head end area is also crowded with two ophthalmologists, microscope and surgical trolleys. Displacement of SGD or circuit disconnection can be detected early with inspired and expired tidal volume and $EtCO_2$. There should be no movement of table as ophthalmic surgery is microscopic surgery.

**POSTOPERATIVE CARE**

Most of the children can be monitored in postoperative care unit (PACU). Emergence delirium, PONV and pain are the most common complaints and need active management. Emergence delirium is more common with newer inhalational agents.\textsuperscript{28} It can be managed with midazolam. Child may be restless due to closure of both the eyes, especially after strabismus surgery. Children should be nursed in lateral position with nonoperative...
side down to prevent pressure on the operated eye. Children after vitreoretinal surgery need to lie prone, and prone position can be given once the child starts following command.

Postoperative pain is usually managed with intravenous fentanyl and NSAIDs.

**Postoperative Nausea and Vomiting**

Incidence of PONV after eye surgery is high 41–88%\(^{40,61}\) and may lead to dehydration, electrolyte imbalance, needing prolonged stay and delay in discharge from hospital.

Eberhart et al. developed a simplified risk score using four variables in children for PONV. It includes duration of the surgical procedure ≥30 minutes, age ≥3 years, immediate relative (Table 3).\(^{62}\)

### POSSIBLE COMPLICATIONS

Intraoperative arrhythmias, bradycardia and even asystole are not uncommon due to OCR. Bradycardia, hypertension and pulmonary edema can occur after phenylephrine eye drops which may need intensive care admission.\(^{11,10}\) Postoperative apnea and bradycardia may occur in preterm infants coming for ophthalmic surgery.\(^{26}\)

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Anesthesia for Major Burns and its Consequences

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INTRODUCTION

Burns is a complex trauma and the second leading cause of death in children <1 year of age. Children are particularly prone to burns due to the inability to recognize danger or due to risk taking behaviors. Children lose proportionately more fluid, are more prone to hypothermia and mount a greater systemic inflammatory response than adults, making them prone to increased morbidity and mortality from major burns. Temperatures as low as 40°C can cause significant injury in children leading to quick cell death.

Burn care needs continuous and prolonged multidisciplinary team management until the functional and social rehabilitation of the child. Apart from physical and functional disability, need for repeated dressings and surgeries and prolonged intensive care, there is so much physical and emotional pain and insult in the early years of their lives that management of these patients requires thorough understanding of the pathophysiology of burns.

UNDERSTANDING BURN INJURY

Skin is the largest organ of the body. Burns directly affect the skin and subsequently alter the physiological functions of virtually all other body organs. Burns injury had a mortality rate of 50% with involvement of 40% total body surface area (TBSA) during World War II. While today, 80% TBSA burns produce a similar mortality rate in adults, though, mortality rate is higher in pediatric age group. The reduction in mortality rate is attributed to better understanding of pathophysiology, fluid resuscitation, critical care, early enteral nutrition, early excision of eschar, skin substitutes, antimicrobial agents, dedicated burns centers and multifaceted team work.

Burns care team typically includes general surgeon, plastic surgeon, anesthesiologist, intensivist, pediatrician, nursing staff, occupational therapist, physiotherapist, clinical psychologist, speech therapist, social worker and dietician.

Factors that determine the severity of burns and its consequences are the temperature and the time of contact, which in turn affect the size or extent and depth of burn area. Further, patient’s age, site of burns, pre-existing disease and associated non-burn injuries impact the overall patient outcome.

Burns could be due to thermal, electrical or chemical injury. Inhalational injury doubles the risk of mortality and must be suspected in victims of facial burns. Ionizing radiation could be one of the causes of burns. Frost bite is also categorized as burns. Cutaneous burns from thermal injury can be either scalds caused by contact with hot liquids or flame, or contact with flames of flammable liquids.

It is important to understand pathophysiology, classification, severity grading, fluid resuscitation, early surgical management and recent updates in burn science.

BURN INJURY—LOCAL EFFECTS

A central zone of coagulation and necrosis, surrounding zone of venous stasis and outermost zone of hyperaemia are the three concentric recognized zones of burns injury. The central zone of coagulation is due to
irreversible coagulation of tissue proteins and this area is unsalvageable. The zone of stasis is characterized by decreased tissue perfusion. Optimum initial fluid resuscitation can improve the blood circulation in this area and prevent the extension of injury. The outermost zone of hyperaemia, as the name suggests, has increased blood flow. This zone generally recovers well unless there is infection.

**CLASSIFICATION OF BURNS**

American Burn Association has classified burns depending on the depth (Table 1).

The assessment of burn depth is based on clinical evaluation using a combination of characteristics such as pain, appearance, color, blisters and capillary refill. In superficial and partial thickness superficial dermal burns, nerve endings remain intact and exposed. Stimulation of these from movement or touch causes intense pain. In partial thickness deep dermal injuries, some nerves may be completely destroyed decreasing the experience of pain. Nevertheless, the damaged nerve endings being exposed to inflammatory mediators in the zones of stasis and hyperemia can produce moderate to severe pain in response to even non-painful stimuli (Fig. 1).

**BURN SEVERITY GRADING**

As per American Burn Association Burn Injury Severity Grading System, a child is labeled as suffering from:
- Minor burn—if involved area is <5% total body surface area (TBSA) of superficial or partial thickness burn or <2% TBSA of full thickness burn
- Moderate burn—when the involved area is 5-10% TBSA of superficial or partial thickness burn or 2-5% TBSA of full thickness burn or there is high voltage burn, suspected inhalation injury, circumferential burn or comorbid systemic condition
- Major burn—if involved area is >10% TBSA of superficial or partial thickness burn or >5% TBSA of full thickness burn or there is high voltage injury, known inhalational injury, significant burn to face, eyes, ears, genitalia, hands, feet, joints or significant associated injury like fracture.

The extent of burn injury is expressed as a percentage of a total body surface area having either second or third degree burns. The rule of "Nine" which is used in adults cannot be used in children as area of head and neck is larger than 9% and lower extremities are smaller. So, Lund and Browder chart is used which considers the changing proportions of the body from infancy to adulthood and makes age-appropriate corrections (Table 2 and Fig. 2).
PATHOPHYSIOLOGY OF BURN INJURY

Major burns cause massive tissue destruction and initiate systemic inflammatory response that subsequently leads to massive fluid shifts and systemic physiological rearrangements making patient critical. Burn injury involves two distinct phases. Initial phase of hypovolemia and burn shock followed by prolonged phase of hypermetabolism.²

Burn Injury Shock

Within minutes to hours of injury, there is release of inflammatory and vasoactive mediators including prostaglandins, histamine, kinins, interleukins, thromboxane, nitric oxide from the injured burned tissues. The mediators increase the capillary permeability and cause local tissue edema. Massive protein extravasation occurs in one hour of the burn injury.³ Damaged skin no longer retains heat and water, allows large evaporative losses as large as 200 mL/m²/hr and results in acute hypovolemic shock following burn injury.⁴ Protein depletion and crystalloid resuscitation predisposes patients to edema in burned as well as nonburned tissues.⁵

Inflammatory mediators like TNF-α further cause myocardial depression with decrease in cardiac output and increase in peripheral vascular resistance in first 24 hours. At cellular level, there is depression of adenosine triphosphate with decrease in transmembrane potential leading to increase in intracellular sodium and extracellular potassium, cellular swelling and acidosis.⁶

There is decrease in splanchnic perfusion with mucosal damage. There could be decrease in glomerular filtration rate and myoglobinuria. Burns injury shock is thus distributive, hypovolemic and or cardiogenic shock, leading to tissue and end-organ hypoperfusion as a consequence.⁷ Hematological changes depict the picture of hemoconcentration, thrombocytopenia and hemolysis.

Likelihood of upper airway edema and potential airway obstruction is always a threat if inhalational injury is present. Bronchoconstriction can occur due to inflammatory mediators.⁸ Alterations in pulmonary physiology accompany all major burn injuries even in the absence of inhalation injury predisposing patients to acute lung injury and acute respiratory distress syndrome.⁹

Also, there is impairment of humoral as well as cell mediated immune responses. Hypokalemia may occur immediately after burn injury due to massive epinephrine release. Although, cellular injury and acidosis can lead to hyperkalemia, there may be low magnesium, hypophosphatemia contributing to cardiac dysfunction, and low red blood cell survival.¹⁰ Subsequent reperfusion of ischemic tissues produces reactive free oxygen radicals, toxic cell metabolites that cause further cellular membrane dysfunction and propagation of the immune response.

Hypermetabolic Phase

If patient sustains the initial phase with fluid resuscitation, there is more severe and sustained hypermetabolic phase that generally develops after 24–48 hours.⁴ There is 10–50 times rise in plasma catecholamines and corticosteroids. These are the primary mediators of the hypermetabolic response that may last for up to 2 years.⁷ This can lead to increase myocardial oxygen consumption and cardiac work. Persistent tachycardia, systemic hypertension, hyperglycemia, insulin resistance, increased muscle protein degradation are all features of the catabolic phase. Left untreated, hypermetabolism leads to physiologic exhaustion and death.⁴

Loss of barrier function of skin and blunting of immune response results in increased susceptibility to infection and bacterial overgrowth within the eschar. A toxic lipid protein isolated from burned skin, is 1,000 times more immunosuppressive than endotoxins. Sepsis is a leading cause of death in patients who survive the acute burn injury.

Impairment of gastrointestinal integrity leads to increased bowel permeability and translocation of bacteria and absorption of endotoxins into the bloodstream. Burn wound infection, intravenous catheters especially central lines or peripherally inserted central catheters associated septicemia and ventilator-associated pneumonia are particularly common in burned children.

Fever, tachycardia, leukocytosis are almost universal in burn patients and cannot be considered signs of sepsis. Blood cultures may be negative in up to 50% of septic patients. Hypotension, intolerance of enteral feeds and rising serum lactate in the non-acute phase favor the diagnosis of infection. Coagulated dermis can become rigid and constricted allowing fluid to accumulate beneath, causing vascular compromise and compartment syndrome.¹

Inhalational Injury

Inhalation of hot dry gases mainly results in supraglottic injury, whereas steam inhalation results in deeper, parenchymal injury as well. The shockwave from a blast can cause chest barotrauma, contusion to the lung and blunt trauma. Most inhalational injuries occur due to inhalation of smoke. The severity of inhalation injury depends on the fuels burnt (chemical composition), intensity of combustion, particulate size of inhaled smoke, duration of exposure, confinement and the patient’s tidal volume during inhalation.
Inhalational burns can cause injury by three mechanisms—thermal, chemical and systemic. Thermal injury results in destruction of epithelial layer, denaturation of proteins and activation of complement cascade. Release of free oxygen radicals lead to increased endothelial permeability causing edema formation and rapid progression to airway obstruction. Chemical injury occurs due to incomplete products of combustion leading to bronchial epithelial sloughing, impaired mucociliary clearance and surfactant inactivation leading to airway blockage. Fall in pulmonary compliance, microatelectasis, ventilation perfusion mismatch cause poor tissue oxygenation. Systemic toxicity may occur due to the absorption of toxic gases such as carbon monoxide.

**Chemical Injury**

Chemical burns occur when skin or eye comes in contact with irritants, either by inhalation or ingestion. The main types of irritants are acids, bases, oxidizers and solvents. Chemicals can diffuse into tissue without apparent damage on skin surface producing severely painful burns. Patient presents with itching, bleaching, coughing blood or tissue necrosis. These are more commonly seen as occupational hazard of mining, fabrication and medical industry.

**Electrical Injury**

Electric burns occur when body comes in contact with electric current. Hands are most commonly affected. Contact with high voltage current can cause cardiac arrest due to ventricular fibrillation, fluid loss into swollen tissue, renal failure due to myonecrosis and infection.

**MANAGEMENT OF BURNS**

Anesthesiologists are involved in the burn patient management right from their arrival in the emergency room for resuscitation, pain management, dressing change, early excision and skin grafting, to contracture release surgeries in the rehabilitative phase. During management of burn patient, the main goals are:

- To ensure optimum resuscitation in emergency period
- To provide adequate pain control and alleviation of anxiety
- To provide procedural anesthesia for dressing change, excision, debridement, synthetic tissue cover or skin grafting
- To provide surgical anesthesia for postburn contracture release surgeries, cosmetic surgeries for better quality of life and functional rehabilitation.

**RESUSCITATION DURING BURN SHOCK PHASE**

All types of burns require immediate cooling. Cold water and ice should be avoided as these can cause vasoconstriction and increase the depth of injury. Prolonged cooling of >15% burns body surface area (BSA) can cause hypothermia in children.

In minor burns, the injury is cleaned and blisters are debrided to allow full assessment of the wound after appropriate analgesia. Burnt area should be then covered with a sterile non-adherent dressing. Some centers use autoclaved potato or banana peels for covering major burn area.

In patient with major burns, initial management follows trauma resuscitation guidelines of ABC including assessment of airway, breathing and circulation. Simultaneously, background history should be noted including events leading to the injury, smoke inhalation or thermal injury of the upper airway, other injuries, medical problems and vaccination status of child and allergies.

**Airway**

Airway management is first priority because patients with major burn can develop upper airway obstruction over the initial 12–24 hours. Intubation is recommended if there is stridor, wheeze, or voice changes. Smoke inhalation injury is suspected if patient has facial burns, history of entrapment, carbonaceous particles in sputum and signs of respiratory distress. These patients are prone to develop ARDS. Bronchoscopy may reveal carbonaceous endobronchial debris and or mucosal ulceration. Bronchopulmonary lavage may be required to remove viscous secretions. Cervical spine is to be immobilized if any trauma is suspected.

**Breathing**

Adequate ventilation and oxygenation must be ensured. All patients must be administered oxygen and continually monitored by pulse oximetry. Patients with carbon monoxide poisoning necessitate high inspired oxygen concentrations. In patients with smoke inhalational injury with respiratory distress, ventilatory support may be required to maintain adequate ventilation, oxygenation and to decrease work of breathing. Low tidal volume and permissive hypercapnia is preferred. Other modalities, such as inhaled nitric oxide and increased PEEP can be used in cases of refractory hypoxemia.

In critically ill children, a cuffed endotracheal tube may be a better choice during mechanical ventilation.
Frequent suctioning will be required for clearing the mucus and debris. Before tracheal extubation, an air leak should be present around the endotracheal tube, indicating resolution of edema. The patient should be closely monitored during the subsequent 24–48 hours as there are high chances of reobstruction of airway.

**Circulation**

Increased capillary permeability leads to loss of circulating volume into the burnt and unburnt areas. For burns up to 10% BSA in children, oral fluid replacement may suffice. For burns >10%, prompt intravenous fluid resuscitation is required. Parkland formula is the most commonly used formula. 3 mL/kg/%TBSA of lactated Ringer’s solution is administered over 24 hours with half given in the first 8 hours and half in the subsequent 16 hours. If the urine output is less than 1 mL/kg/hr, then increase the infusion by 33% of the hourly calculated fluid requirement. Daily maintenance fluids should be added to above calculated fluid as there is no linear correlation between weight and BSA. Also glucose-containing solutions should be added as necessary, in infants because hepatic glycogen stores are depleted after 12–14 hours of fasting. Patients with inhalation injuries, electrical burns, and those with delayed resuscitation, require additional fluid.

Burn edema is maximal in the first 18–30 hours. Crystalloids are useful in early shock. Resuscitation with large volume of crystalloid can cause compartment syndrome, worsening of edema and conversion of superficial to deep burns. This is called as fluid creep occurring due to overzealous crystalloid resuscitation according to Parkland formula and inefficient titration.

Hypertonic saline generates higher osmotic pressure and shifts intracellular water to intravascular compartment, hence effective in treating shock. But it is deferred for resuscitation in burn patients due to risk of hypernatremia and renal failure.

Use of colloids in burns resuscitation is the subject of debate. During later phase of resuscitation, colloids can be useful (e.g. albumin, hydroxyethyl starch preparations) as they replenish plasma proteins and stay longer in the intravascular space and improve hemodynamic stability as compared to crystalloids. During early phase colloid is either ineffective or destructive. It shifts into extravascular space through leaky capillaries, exacerbates total body edema, making mobilization of that edema fluid difficult. It also causes late pulmonary complications and increases mortality with inhalational injury. Hypersensitivity reactions, interference with coagulation and impairment of renal functions are some of the worrisome consequences with use of colloids for burn resuscitation.

All fluids have potential for both benefit and detriment; the best fluid for resuscitation should be ultimately decided by an individual patient’s unique physiologic needs. Cochran and other clinicians used albumin in early postburn period and demonstrated successful resuscitation with decreased mortality. It decreased fluid requirements and lowered weight gain. Tricklebank has also reported the successful use of crystalloids with albumin or plasma to decrease fluid requirements and thus avoided adverse effects associated with over resuscitation such as abdominal compartment syndrome. Parkland formula should be used as a guide only, for fluid resuscitation in burn patients. Fluid volume should be modified by continuous assessment and reassessment of vitals. This is called as goal directed therapy. Adequate resuscitation is reflected by normal mentation, stable vital signs, and a urine output of 1–2 mL/kg/hr. Judicious use of crystalloids and colloids with monitoring of glucose and electrolytes are important steps.

**Fluid Therapy Between 24 and 48 Hours**

As capillary permeability reduces significantly and extravascular fluid loss is also reduced, lesser fluid is required almost 25–50% less than first day. So 5% dextrose can be given in appropriate volume. Colloids can be given at this stage at rate of 0.3–0.5 mL/kg/% burn area.

**Fluid Therapy After 48 Hours**

It includes maintenance fluid and evaporative losses from wound surface (5% dextrose at rate of 1 mL/kg/% burnt area). Albumin is required if hypoalbuminemia (<2.5 gm/dL) develops. Packed red blood cell (RBC) is infused to maintain PCV around 35%.

**Early Enteral Feeding**

In the catabolic phase, BMR can increase up to 40% after a significant burn, so nutritional support is vital. Early enteral feeding decreases infections and sepsis, improves wound healing and nitrogen balance and, reduces stress ulceration and duration of hospitalization. So in major burns, nasogastric tube should be inserted and feeding has to be started within 6–18 hours.

**Antibiotics**

In minor burns local antibiotics suffice, but for major burns local and IV antibiotics should be started. In pediatric patient, injection tetanus toxoid should be administered after assessing the vaccination status.
ANALGESIA AND ANXIOLYSIS

Burns produce pain by direct injury of nerve endings and specialized receptors in the skin, with both primary and secondary hyperalgesia. Thus, severe pain is an inevitable consequence of a major burn injury. Anxiety and depression are common components in a major burn and can further decrease the pain threshold. Pain management should be offered during all stages of treatment including in the emergency department, during procedures such as dressing changes and after discharge when complex neuropathic pain syndromes may develop.

During resuscitation, pain scores should be assessed hourly, frequency of assessment can be eased while managing background pain. Along with pain assessment, monitoring of vital signs and sedation level is also required.

For assessing pain the child’s self-report should be used. For children ≥7 years, a visual analogue scale is an excellent tool. Wong-Baker FACES Pain Rating Scale is recommended for children ≥3 years of age. In infants or in those with cognitive impairment or language difficulties the FLACC tool should be used. FLACC is a behavioral assessment tool with five categories (Face, legs, activity, cry, and consolability).2

Pain management should be based on an understanding of the type of burn pain. Burn pain can be background, breakthrough or procedure-related. Background pain can be managed with oral opioids. For breakthrough pain, faster, short acting opioids or paracetamol is preferred whilst procedural pain requires more intense analgesia with more potent opioids and other anesthetic agents.5

Pharmacological Treatment

The ideal analgesic agent in a child with burn should be easy to administer, well tolerated, with rapid onset of analgesia, short duration of action and minimal side effects.

Various routes such as parenteral, oral, rectal and intranasal are available for administration of analgesia. For severe pain, intravenous route is preferred. However, intranasal route is a good alternative.

Opioids

Opioids are the mainstay of pain management. They are potent, effective, can be titrated to effect; but have unwanted side-effects, such as nausea, vomiting, pruritus, respiratory depression, tolerance and dependence. Morphine is currently the most widely used drug. It can be given as IV 0.1 mg/kg every 6 hourly in children >6 months of age. But it has slightly delayed onset of action (10 minutes) and long lasting effects (8-12 hours). So intraoperative titration is difficult during procedure to meet individual needs. Therefore, short-acting medications such as fentanyl, alfentanil and remifentanil are more appropriate for pain relief in burn patients during procedures.5 Alfentanil is a short acting opioid with the peak effect reached within a minute. It undergoes hepatic metabolism to inactive metabolites that are excreted via the kidneys; it is a safer option in children with impaired renal function. Fentanyl lollypops 15–20 μg/kg and intranasal fentanyl 1.5 μg/kg are more interesting alternatives for use in pediatric burns dressing changes either alone or in combination with oral morphine as a top up agent.5,11,12 Remifentanil is an ultrashortacting opioid, but it does not provide post-procedural pain relief. It is useful in spontaneously breathing, nonintubated burn patients and preferred in neonates where risk of postprocedure sedation is more.13 Tramadol, a weak opioid, in the dose of 1-2 mg/kg IV/IM can be used for background pain (sustained release preparation) and breakthrough pain. For patients who have become intolerant to morphine through prolonged treatments, oral methadone can be used as an alternative.

Ketamine

Along with opioids other agents are also used to provide multimodal analgesia and avoid opioid related side effects. Ketamine offers the advantage of maintaining stable hemodynamics and providing analgesia and has been used extensively as the primary agent for both general anesthesia and analgesia for burn dressing changes. Oral dose of 3–10 mg/kg of Ketamine produces sedative effect in 20–45 minutes. To avoid agitation and delirium it is co-administered with benzodiazepines or opioid.

Nonsteroidal Anti-inflammatory Drugs

This group of drugs modifies the systemic inflammatory response. Antiplatelet and nephrotoxic effects of nonsteroidal anti-inflammatory drugs (NSAIDs) are side effects of significant concern in burn patients. NSAIDs are not the drug of choice for pain management in burn patient.

Acetaminophen can be used for minor and superficial burns in the acute setting, as a good adjunct along with opioids. It can be administered as syrup 20 mg/kg 6 hourly or suppository 40 mg/kg.

Nitrous Oxide

50:50 of O₂:N₂O (Entonox) is a safe agent for providing analgesia in burn patients. It is contraindicated in situations, such as decreased consciousness, pneumothorax or air embolism.
Antidepressants and Anticonvulsants

These are new drugs for use in children. They improve the sleep patterns. Antidepressant Amitriptyline enhances opiate-induced analgesia while anticonvulsant Gabapentin is useful in the treatment of neuropathic pain following burns. Gabapentin is also effective in the management of itch in children (common after burn injury) unresponsive to simple anti-itch medications.14 It is started at 10 mg/kg and titrated up to 40–50 mg/kg/day. Midazolam acts synergistically with opioids to reduce mental awareness. But it potentially increases the risk of respiratory depression.

Alpha-2 Agonists

Clonidine and dexmedetomidine provide conscious sedation, and thus are used as possible adjuncts to the standard opioids and benzodiazepines regimen. They are also effective in treatment of sympathetically mediated pain. The dose of clonidine used in pediatric practice is 1–3 μg/kg orally three times a day.

Dexmedetomidine is a novel alpha-2-adrenergic agonist. Apart from sedation, anxiolysis, and analgesia, it also inhibits insulin secretion by stimulation of alpha-2 receptors on pancreatic beta cells which is helpful in catabolic phase.15 It is used as 1 μg/kg bolus followed by infusion in titrated doses 0.2–0.7 μg/kg/hr. It is considered as an excellent adjuvant to ketamine and propofol combination for pediatric wound dressing changes. Sympathomimetic effect of ketamine is balanced by central sympatholytic effect of dexmedetomidine and maintains stable hemodynamics.16 It does not result in respiratory depression. Dexmedetomidine is effective for sedation of pediatric burn patients on mechanical ventilation with close cardiovascular monitoring.17

Nonpharmacological Treatment

An anticipation of pain increases pain intensity and discomfort, which can be decreased by diverting the patient’s attention. Distraction, hypnosis are commonly used techniques. These supportive techniques may be time-consuming, but they can help to reduce the feelings of fear and anxiety, especially during long procedures. However, they must always be used in conjunction with pharmacological treatment.5

EARLY EXCISION AND GRAFTING SURGERY

Excision and grafting involves tangential excision of the burn wound, in which the eschar is shaved off from the burn until a plane of viable tissue is reached, followed by covering the excised wound with a split thickness skin graft. Early excision (within 7 days) is preferred. It is one of the ways to modulate the hypermetabolic response in severe burns. It decreases the bacterial load, prevents protein loss and catabolism and resting energy expenditure. It also helps in wound healing and decreases the number of further dressing and pain. In major burns, excision of >15% of burn area may exaggerate the blood loss, hypothermia and consequences. Thus, sequential excision can benefit the patient. General anesthesia is preferred for this surgery in small children; while total intravenous anesthesia (TIVA) can be used in older children.

ANESTHESIA CONCERNS IN EARLY EXCISION

Preoperative assessment for patients with major burn injuries, particular attention should be paid to following:

- **Airway assessment:** All patients with face, neck, and upper chest burns are considered potential difficult airways due to facial and airway edema that may distort the normal anatomy and/or limit neck and mandibular mobility.

- **Pulmonary status:** If patient requires ventilator assistance, ventilator mode, settings and ABG should be noted. Mechanism of injury and time elapsed since injury should also be enquired. After 24–48 hours of injury, patient can develop pulmonary edema, ARDS.

- **Extent of injuries** (percentage of total body surface area burnt), burn depth and distribution should be noted.

- **Current physiologic status:** Intravascular volume status, vasopressor requirements, urine output, respiratory rate, temperature (fever), any cardiorespiratory event or insult during resuscitation should be known.

- **Derangements in investigations:** Complete blood count with hemoglobin and hematocrit, electrolytes, acid-base disturbances should be documented.

- **Surgical plan:** Patient positioning, estimate of areas of excision, donor sites to harvest should be discussed with surgeon.

- **Previous anesthetic records**

- **Vascular access:** Two wide bore peripheral lines or a central line is recommended.

- **Associated comorbidities**—including conditions that increase risk of infection, presence of fractures which may limit the sites available for vascular access or monitoring should be enquired.
• **Enteral feeding:** Volume of Ryle’s tube (RT) aspirate before giving the next RT feed, frequency of stool. Delayed gastric emptying and paralytic ileus increase the chances of aspiration.

• Availability of blood and blood products.

### Anesthetic Technique

Balanced general anesthesia with the combination of an opioid, muscle relaxant, and a volatile agent is the most widely used technique. General anesthesia with endotracheal intubation is preferred in pediatric patient for burn debridement.

Before induction of anesthesia, nasogastric tube should be suctioned to prevent aspiration.

Propofol and ketamine can be carefully titrated to minimize dose dependent cardiac and respiratory depression. But if airway control is a concern or venous access difficult, inhalation induction is recommended. During shock phase, lower doses of agents are required because of prolonged duration of action and slower rates of renal clearance. Anesthetic requirements are increased during catabolic phase due to altered protein binding and increased renal clearance. Benzodiazepine and dexmedetomidine significantly reduce anxiety, produce sedation during burn procedures and may be helpful in ameliorating opioid tolerance.

Burn injury causes proliferation of extrajunctional nicotinic acetylcholine receptors leading to increased resistance to nondepolarizing muscle relaxants and increased sensitivity to depolarizing muscle relaxants, i.e. suxamethonium. Within 24 hours of burn, suxamethonium can be used for endotracheal intubation if difficult airway is anticipated. But after 24 hours for upto 2 years, it is unsafe to use suxamethonium, as it can cause lethal hyperkalemic response leading to ventricular dysrhythmias.

Rocuronium can be used (up to 1.2 mg/kg) for rapid-sequence induction after 24 hours of burn injury.

Resistance to nondepolarizing muscle relaxants may develop within a week of burn injury and persist for upto a year and is proportional to TBSA burned. Burn patients may require a 2–5 fold greater dose of nondepolarizing muscle relaxant than nonburned patients.

Burn injuries are intensely painful due to direct tissue injury and inflammation-mediated hyperalgesia. So, multipronged approach including opioids, acetaminophen and tumescent local infiltration is required to manage pain.

Tumescent local anesthesia with lidocaine and epinephrine has been shown to be safe and effective local anesthesia technique for the surgical treatment of noncontiguous pediatric burns.

Regional anesthesia is used in combination with general anesthesia in patients with small burns or for reconstructive procedures. For procedures on lower extremities, spinal anesthesia or lumbar epidural or caudal catheters can be used to provide intra- and postoperative analgesia in absence of contraindications. Role of regional blocks is limited in acute burns because of site of burn and local or systemic infection.

Airway management in pediatric burn patients for surgical anesthesia is challenging. Mask ventilation is difficult with facial burns. Depending on the duration of the burns, edema, scarring or contractures can cause narrowing of the mouth opening. Neck burns cause restriction of the neck movements. In cases of facial burns, laryngeal edema is also expected in early stages. Difficult airway cart with various sizes of facemasks, oropharyngeal airways and endotracheal tubes, ventilating and intubating bougie, supraglottic airway devices, intubating LMA, videolaryngoscope and fiberoptic bronchoscope should be available. Surgical airway equipment and ENT surgeon, who is expert in pediatric tracheostomy should be kept standby. Anesthesiologist and team should have plan A, plan B and plan C ready to execute with clarity for difficult airway.

Crystalloids are mainstay of therapy during early phase of burn injury. Colloids are preferred 24 hours after burn injury. Urine output is an important guide for fluid management.

Multiple burn sites and donor skin sites require frequent changes in position, repositioning of the patient during operation. This skin exposure can cause hypothermia. Thus, active warming is required, including raising theater temperature, heated mattresses, convective warming systems, and covering exposed areas wherever possible.

Monitoring is known to be difficult with lack of sites available for pulse oximeter probes, NIBP cuffs, and ECG electrodes. Oximeter ear probes work well, and electrodes can be sited away from the burnt area on chest or attached to surgical clips.

### Blood Loss

Burn excision can cause massive and sudden blood loss. Measuring blood loss is difficult in pediatric patients, so transfusion is given according to hematocrit estimations. Adequate venous access should be secured before procedure. Blood should be available in the operating room before excision begins. Intraoperative tourniquet on burned extremities reduces the blood loss. Subcutaneous infiltration of lignocaine with epinephrine at donor area
facilitates skin harvesting, and at burn area reduces blood loss during debridement and produces a bloodless surface for placement of skin grafts. Post-excision compression dressings also reduce blood loss. Hematocrit between 20 and 25% is preferred to maintain high metabolic demands for oxygen. If blood loss is excessive, then coagulation abnormalities should be ruled out.

Simple hand wash technique and strict asepsis should be followed for infection prevention. Sepsis is a leading cause of death in patients who survive the acute burn injury. Cultures of wound biopsy will help in identifying the offending pathogen. Systemic antibiotics are reserved for treatment of proven infection and in the perioperative period.

**ANESTHESIA CONCERNS FOR DELAYED EXCISION**

When patient presents late (30–45 days) for skin grafting, laryngeal edema is not the concern but beginning of contracture formation may pull the larynx anteriorly making intubation difficult and virtually impossible by direct laryngoscopy. In such a scenario, video laryngoscope e.g. Airtraq, intubating laryngeal mask airway (ILMA) or fiberoptic bronchoscope can be used. Difficult airway cart containing smaller size ETT, oral airways, good suction should be kept ready.

Pediatric patients require deep sedation for fiberoptic intubation, which can be facilitated by inhalation induction with a volatile anesthetic or intravenous ketamine, 1 to 2 mg/kg.

**ANESTHESIA CONCERNS FOR CONTRACTURE RELEASE**

Pediatric burn patients have continued growth retardation long into the rehabilitative phase. Patients are exposed to multiple repetitive surgeries either for contracture release or for cosmetic purposes with their growing age. In patients with burn >40% of their TBSA, the hypermetabolic stress response remains for longer time. It causes severe catabolism, immune dysfunction, and profound physiologic perturbations. It cannot be completely abolished by nonpharmacologic interventions. So, pharmacologic agents like recombinant human growth hormone (rHGH), insulin-like growth factor-1 and insulin-like growth factor binding protein-3, oxandrolone, fenofibrate, glucagon-like peptide-1, beta-antagonist, ketoconazole can be used either alone or in combination. But long-term use of rHGH can cause hyperglycemia, dyslipidemia. So when such patient comes for repetitive surgeries, anesthesiologist must keep in mind effects of such drugs.

Anesthesiologists continue to face the issues of difficult airway, liver enzyme induction and its effects on drug metabolism and problems of hypercatabolic state. Non painful, pleasant experience of anesthesia and recovery can improve patient rapport, cooperation and may bring about positive outcome in the pediatric burn survivors.

Most of the burns in children can be prevented. Widespread education and safety devices such as smoke alarms can avoid many thousands of deaths per year. The educational material on safety and first aid like extinguishing fire by stop, drop and roll and cool burn part by pouring tap water will generate awareness and help in reducing accidental burns in pediatric patients.

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**LEARNING POINTS**

- Children suffering from major burn injuries present unique challenges for management. Burn survival primarily depends on severity of burn injury, patient age, site of burns, presence of inhalational burns, pre-existing disease and associated non burn injuries.
- Children are not miniature adults. They have different percentage representation of body surface area, thinner skin, lose proportionately more fluid, are prone to hypothermia, and mount a greater systemic inflammatory response.
- Anesthesiologists are involved in the burn patient management right from their arrival in the emergency room for resuscitation, pain management, dressing change, early excision and skin grafting to contracture release surgeries in the rehabilitative phase. Knowledge about the advances in treatment of major burns, early debridement, better intensive care, early enteral feed and management of inhalation injury can bring about marked improvement in survival from major burns.

**REFERENCES**

INTRODUCTION
Pediatric neuroanesthesia deals with patients whose central nervous system (CNS) is in a constant state of development. Infants have incomplete brain development and myelination, altered cerebral blood flow and metabolism, affecting their responses to pain as well as anesthesia techniques. In addition, CNS pathology leads to further local, general or systemic alterations. Hence, a pediatric neuroanesthesiologist should have thorough knowledge of pediatric neurophysiology and neuropharmacology, surgical pathology and a good understanding of neuroanesthesia principles so as to avoid any additional insult on stressed brain.

PATHOPHYSIOLOGY

Intracranial Pressure (ICP)
Normal ICP in children is 3–7 mm Hg and in neonates 1.5–6 mm Hg as compared to 10–15 mm Hg in adults. The cranial cavity contains relatively incompressible brain tissue, blood and cerebrospinal fluid (CSF). In small infants, any increase in intracranial volume can expand the unfused sutures and increase head size without much change in intracranial pressure. In older children with fused sutures and fontanels, any volume increase is accommodated by shift in CSF or blood to some extent. Larger or sudden increases in volume are associated with increased ICP leading to secondary brain injury and herniation with neurological deterioration.

Cerebral Blood Flow (CBF)
Infants and children receive a larger percentage of blood flow than adults (55 mL/100 g of brain tissue/minute). The CBF in children of 3–12 years is approximately 100 mL/100 g/min, in children of 6 months to 3 years, it is 90 mL/100 g/min and in neonates and infants, it is lower (40 mL/100 g/min). Cerebral autoregulation allows constant blood flow within a set range of cerebral perfusion pressure (CPP). In adults, this range is approximately 60–160 mm Hg. In children, the autoregulation occurs at a lower and narrower range and at lower and upper limit of autoregulation, the CBF dips and rises steeply. In term neonates, this range is 20–60 mm Hg. This implies that at low blood pressure, they are at higher risk of cerebral ischemia and higher blood pressure puts them at greater risk for intraventricular hemorrhage. Carbon dioxide (CO₂) has a direct vasodilatory effect on cerebral vessels and CBF is directly proportionate between pCO₂ between 25–55 mm Hg range. Therefore, hyperventilation may precipitate cerebral ischemia especially in neonates. Cerebral blood flow increases significantly at pO₂ less than 50 mm Hg. Raised ICP has a detrimental effect on cerebral perfusion.

Cerebral Metabolism
Younger children and infants have a higher cerebral metabolic rate for oxygen (CMRO₂) approximately 5.2 mL/100 g/minute and 5.8 mL/100 g/min, respectively as
compared to adult values of 3.2 mL/100 g/min. Neonates in view of lesser metabolic demand have a lesser CMRO₂, approximately 2.8 mL/100 g/min. Hypothermia reduces CMR by 6–7% per °C reduction in body temperature.  

**Effects of Anesthetic Agents**

Various drugs affect cerebral blood flow by affecting the CBF, ICP and cerebral metabolism. All anesthetic agents reduce cerebral metabolic demand for oxygen as they produce unconsciousness.

*Intravenous agents:* Thiopentone and propofol reduce CMRO₂ by 50%, and also reduce CBF and ICP. Etomidate produces similar effect to a lesser extent. Cerebrovascular autoregulation in response to MAP and PCO₂ is not affected by propofol. Ketamine increases CMR, CBF and ICP. Opioids and benzodiazepines produce modest reduction in CMR.

*Inhalational anesthetic agents:* Inhalational anesthetic agents are vasodilators and thus increase cerebral blood flow and therefore ICP. Halothane causes most rises in CBF. They also produce dose-dependent reduction in CMR. Nitrous oxide increases CBF without reducing CMR and therefore has tendency to increase ICP.

**CLINICAL PRESENTATION**

Clinical presentation varies with the specific pathology, age of the child as well as the rapidity of change in the volume of intracranial components. The presenting child could be healthy with mild symptoms, critically ill or be developmentally delayed. Signs of raised ICP in infants could be history of irritability, failure to feed, lethargy whereas older children may present with headache, nausea, vomiting. Excessive sleepiness, unconsciousness, seizures, motor weakness may also suggest possibility of increased ICP.

In addition to routine general examination, neurological assessment like assessing the level of consciousness, motor, sensory and cranial nerves functions, pupillary signs and reaction to light should be performed. Signs of increased ICP are bulging fontanelle and cranial enlargement in infants and in older children diplopia, hypertension, bradycardia, pupillary dilatation and papilloedema. In unconscious patients, gag and cough reflex and evidence of pulmonary aspiration should be checked. Fluid status should be assessed in children with raised intracranial pressure. Evaluation of other systems for associated comorbidities, especially in a syndromic child should be done.

*Investigations:* Apart from the baseline hemogram and routine urine microscopy, additional tests like coagulation studies, serum creatinine, arterial blood gases, chest radiograph and electrocardiogram may be asked on an individual basis. Serum electrolyte levels are required in children with suprasellar lesions and in those on diuretic therapy. Liver function tests are required for children on anticonvulsants and antitubercular treatment. Children with intracranial abscesses should be evaluated for co-existing cyanotic heart diseases.

**ANESTHESIA GOALS**

The fundamental goals of pediatric neuroanesthesia are:
- To avoid worsening of existing neurological injury by maintaining normal ventilation, body temperature, fluid electrolyte balance and glucose levels. Disruption of homeostasis in these areas can lead to secondary neurological injury
- Thorough knowledge of every presenting pathology and hence a judicious choice of various induction and maintenance techniques
- To provide excellent operating conditions for the surgeon
- Prompt emergence and neurologic assessment at the end of surgery.

**ANESTHETIC MANAGEMENT**

Individual concerns for specific conditions are discussed later though some common principles apply to most cases.

**Premedication**

A child with normal ICP may benefit from sedative premedication to reduce separation anxiety, most popular being oral/nasal midazolam. However, children with raised ICP are extremely sensitive to even a small dose of sedative leading to airway obstruction or respiratory depression causing hypercarbia and further elevation of ICP. In such patients sedative drugs are preferably avoided or should be administered under strict supervision.

**Monitoring**

Standard ASA monitors should be used for all the cases. Additionally, there should be temperature monitoring and precordial or esophageal stethoscope for small children. Sudden severe hemodynamic changes may be
seen in large tumors, craniofacial surgeries and invasive blood pressure monitoring is recommended. The latter is also recommended when tight control of BP is required, e.g. elevated ICP, cerebrovascular procedures. A central venous catheter is useful when venous air embolism is anticipated or as an intravenous access when peripheral vein is not available. Urine output monitoring is considered in all large intracranial masses, patient on osmotic diuretics, sellar and suprasellar tumors (for risk of diabetes insipidus). Neuromuscular junction monitoring is helpful in patients on antiepileptics. Neurophysiologic monitoring like electrocorticography (ECoG), electroencephalography (EEG) or evoked potential (EP) monitoring may be needed in certain complex surgeries.

**Induction of Anesthesia**

It is prudent to avoid episodes of hypoventilation, hypoxia, hypercarbia, hypotension, coughing and straining during induction. Intravenous induction is preferred when an intravenous access is present with either thiopentone or propofol.5,7,10 Etomidate though having an advantage in hemodynamically unstable patients, reduces seizure threshold and suppresses adrenocortical axis.11 Ketamine is not preferred in neuroanesthesia practice as it can cause neuroexcitation and increases ICP.

Inhalational induction techniques are used when the child presents without IV access. Sevoflurane is the popular agent for induction. Even though inhalational agents cause increased CBF and hence raised ICP, mild hyperventilation (ETCO₂ of 30–35 mm Hg) offsets this increase in CBF. However in a child with impending herniation, it is best to avoid high doses of inhalational agent for securing IV access. Rapid sequence induction or modified RSI12 is necessary for patients with raised ICP as they have delayed gastric emptying. Rocuronium (1–1.2 mg/kg) is the preferred drug in this regard.

**Airway Management**

Induction agents are supplemented with opioids, muscle relaxant for ideal intubating conditions. Preexisting hypercarbia and hypoxia can be corrected by gentle ventilation. Intravenous fentanyl, preservative free lignocaine or nitroglycerine sublingual spray may be used to prevent the hyperdynamic response of tracheal intubation. Tracheal tube with a minimum leak should be inserted and appropriately fixed so as to avoid endobronchial migration after surgical position is given. In some cases where transoral or trans-sphenoidal approach is planned wire reinforced tubes are preferred. Constant monitoring of airway pressures can identify inadvertent displacement. A pharyngeal pack is inserted in case where oral bleeding or CSF leak is expected.

**Maintenance of Anesthesia**

A wide variety of maintenance agents can be used in pediatric neuroanesthesia. This includes low dose of inhalational agents, propofol, dexmedetomidine and opioids. Inhalational agents increase ICP and decrease MAP and CPP in doses larger than 1 MAC. This effect is more pronounced when nitrous oxide is added.11 In children with elevated ICP, desflurane and isoflurane may increase ICP to a greater extent than sevoflurane.10 On the other hand, intravenous propofol will reduce CMRO₂, CBF and ICP.14 Inhalational agents and propofol may cause dose dependent myocardial depression which should be kept in mind while patient is in head high or sitting position. A balanced technique combining inhalational agents and intravenous drugs may reduce side effects associated with high doses. Use of nitrous oxide may be relatively contraindicated in presence of pneumocephalus, need for neurophysiologic monitoring and in cases at high risk for venous air embolism.

Dexmedetomidine with its hemodynamically stable profile, anesthetic sparing effect and lack of respiratory depression is being increasingly used in pediatric neuroanesthesia. Its effects on CMRO₂, CBF and ICP are still being investigated.15 Vecuronium, rocuronium or pancuronium can be used in older children whereas atracurium or cisatracurium is preferred in neonates and infants for neuromuscular blockade.

**Positioning**

To obtain the best surgical conditions, various positions like supine with head elevation, head turned, lateral, prone or sitting position are employed. When the head is fixed using pins care should be taken to give small dose of opioids or a deeper anesthetic to avoid movement or injury. Excessive head rotation or flexion may impede venous return causing raised ICP and increased surgical bleeding. Prone position for posterior fossa tumors is common in children. It involves significant cervical flexion to aid surgical exposure which may result in kinking of endotracheal tube or massive tongue and uvular edema with inability to extubate. Wire reinforced tube may be preferred. Adequate padding of the pressure points, checking of peripheral pulses should be done and stretch of peripheral nerves should be avoided.
Intravenous Fluids and Blood Administration

The goal is to maintain normovolemia throughout the procedure. Children presenting for neurosurgery are often volume-depleted. Ringer’s Lactate and normal saline are considered a suitable maintenance fluid during neurosurgery. Glucose containing fluids are avoided in neurosurgery in general due to the deleterious effect of hyperglycemia on the brain. Blood sugar level monitoring is needed during long procedures, prolonged fasting, diabetic children, infants and neonates as both hypo- and hyperglycemia can exacerbate secondary neurological injury in acute cerebral damage.

The estimation of blood loss can be difficult to measure and can be more accurate if the operative field is in view, suctioned bottles are calibrated and hematocrit is estimated intraoperatively. The anesthesiologist should predetermine the maximum allowable blood loss. Most commonly hematocrit is maintained above 24 percent. Prompt replacement of blood loss with packed RBCs and other blood products helps to prevent setting of coagulopathy.

Temperature Management

Small children are prone to hypothermia. The operation theater should be warmed to near ambient temperature. Cleaning solutions, irrigating and intravenous fluids should be close to body temperature. Warming mattress and blanket should be used to maintain normothermia. Hyperthermia is also frequently seen in pediatric neurosurgery in presence of certain mid-brain or thalamic lesions and infection. This should be treated using antipyretics and passive cooling.

Emergence

Smooth emergence without coughing, straining and hypertension is desirable in neurosurgery. Adequate analgesia and adequate reversal of neuromuscular blockade should be ensured before allowing patient to wake up. Adjuncts like fentanyl, dexmedetomidine, esmolol may be used prior to extubation. Extubation should be planned if the child is alert, spontaneously breathing, hemodynamically stable and neurologically well. If there is delayed awakening with apparently no anesthesia cause, urgent CT scan may be required. Elective ventilator support is considered in case of facial swelling, airway edema, and posterior fossa surgery close to vital structures in brain stem or poor preoperative neurological status.

Postoperative Management

Postoperative pain can be treated with intravenous opioids, paracetamol, nonsteroidal anti-inflammatory drugs and care should be taken to avoid drugs causing respiratory depression or interfering with neurological assessment.

POSSIBLE ISSUES AND COMPLICATIONS

1. Intraoperative blood loss: Some tumors are vascular, some very close to major vessels, so it is worthwhile to read the CT and MRI plates preoperatively to anticipate blood loss and prepare in advance.
2. Tight Brain: During surgery, there could be acute rises in ICP which need to be promptly managed to ensure good surgical exposure and to prevent cerebral ischemia. Various measures to be used:
   - Ensure normoxia and normocarbia
   - Ensure adequate depth of anesthesia and muscle relaxation
   - Recheck head position for adequate venous drainage from head
   - Propped-up position, if hemodynamically stable
   - Use hyperosmolar agent—mannitol 0.5–1 g/kg or 3% NaCl infusion 1–2 mL/kg over 1 hour
   - Small bolus dose of thiopentone or propofol to deepen anesthesia. This can reduce blood pressure, putting the ischemic brain more at risk.
   - Surgical drainage of cerebrospinal fluid
   - Hyperventilation for very short period only; It is important to note that small children are prone to inadvertent hyperventilation and are prone to cerebral ischemia.
3. Venous air embolism (VAE): VAE is a common problem when the head is above the heart level mainly in sitting position. Even in supine position, the large occiput in an infant’s head keeps the head above the level of heart and a head elevated position compounds the problem. The incidence of venous air embolism is found to be 33% in children below 12 years. In view of higher incidence of intracardiac shunt in pediatric population, preoperative screening 2D echo is recommended in all children to prevent paradoxical air embolism. Sitting position is avoided in such patients with intracardiac shunts. A sudden drop in ETCO2, blood pressure or presence of air on Doppler monitor should alert of VAE. Though least sensitive, an esophageal stethoscope may pick up ‘Mill-Wheel” murmur in absence of other sensitive monitors. If VAE occurs, FiO2 should be increased to
100%, surgical field should be flushed with saline, and if possible, air should be aspirated from central venous line, with head positioned below the heart level.

4. Postoperative complications following neurosurgery include delayed recovery from unconsciousness, airway obstruction, respiratory depression, increased ICP secondary to cerebral edema, hematoma or hydrocephalus, continued blood loss or seizures. All children who have undergone major craniotomy should be shifted to intensive care unit for observation and care.

**ANESTHESIA CONSIDERATIONS FOR SPECIFIC SURGICAL PROCEDURES**

Apart from the general considerations discussed above, anesthesia management differs as per the unique demands of each surgery.

**CSF Diversion Surgeries**

Obstructive hydrocephalus is the commonest indication for CSF diversion surgeries to reduce intracranial CSF volume. Ventriculoperitoneal shunt is the commonest procedure done and other procedures include ventriculoatrial, ventriculotheal, ventriculopleural shunts, ventriculostomy or external ventricular drain.

Infants with hydrocephalus have a large head and difficult intubation is anticipated. A roll under the shoulder or a small mattress under the torso would help in the neck extension and visualization of larynx. The position for surgery involves head rotation and care should be taken to avoid extreme rotation as it can impede venous return, increase ICP and bleeding. All IV lines should be within anesthesiologist’s reach. Dilated scalp veins can be used for intravenous access if other sites are not available. The most stimulating part of surgery are incision and tunneling of shunt and additional dose of opioids should be given along with deepening plane of anesthesia. On cannulating the ventricle, blood pressure may drop as brain stem pressure is suddenly released.

Complications of shunt procedures include hypotension, dysrhythmias, pneumothorax, accidental bowel injury, shunt infection, malposition. Venous air embolism may occur if the shunt system is not kept flushed continuously. Overdrainage of CSF leads to risk of subdural hematoma or reverse herniation.

While doing ventriculoatrial shunt, use of endocardial ECG on atrial tip helps in correct placement. Sudden drainage of large amount of CSF into right atrium may lead to congestive heart failure. These children should not receive excessive intravenous fluids.

Endoscopic third ventriculostomy (ETV) is a procedure to connect the cerebral ventricles with the subarachnoid space below the surface of brain for obstructive hydrocephalus. Ringer’s lactate is commonly used as irrigating fluid. Deeper plane of anesthesia with complete paralysis is mandatory to prevent any movements. Bradycardia is most frequently seen besides tachycardia, hypotension and hypertension. Electrolyte disturbances may be seen. As significant air can get entrapped in ventricles, nitrous oxide should be avoided to prevent problems related with pneumoencephalus and pneumoventricle. Injury to basilar artery may occur due to its close proximity leading to severe bleeding. Control of such bleeding is difficult as vision through endoscope is lost. Cross-matched group specific blood should always be kept readily available.

**Intracranial Tumor Excision**

Intracranial tumors in pediatric age group are most commonly seen in the midline and have tendency to produce obstructive hydrocephalus and raised ICP.

**Supratentorial Tumors**

Supratentorial tumors include craniopharyngiomas, gliomas, pituitary adenomas, choroid plexus papillomas and cerebral tumors. Craniopharyngiomas require detailed preoperative endocrine evaluation including pituitary, thyroid and adrenal functions as hypothalamic-pituitary-adrenal axis may be impaired necessitating peroperative hormone replacement therapy and corticosteroid supplements. Common findings are hypernatremia with dilute urine. Replacement with antidiuretic hormone (ADH) substitute desmopressin (DDAVP) is often necessary. Anticonvulsants should be provided. Fluid intake should match the urine output and should be devoid of excess sodium load. Hence 0.45% NS/RL alternatively can be used, till patient is able to take orally.

**Infratentorial Tumors**

Common ones include medulloblastoma, ependymoma, cerebellar astrocytoma, hemangioma and brainstem glioma. Preoperative assessments includes looking for signs of raised ICP, cranial nerve involvement like history of regurgitation, choking on food, change in voice, loss of protective airway reflexes or cerebellar symptoms.

Surgical excision of a posterior fossa tumor is usually done in the prone position with lateral and sitting position being other alternatives. Pins are avoided in small children as they may cause skull fractures or dural tears.
and horseshoe rest is generally preferred. While resecting tumors involving the brainstem or around it, arrhythmias and hemodynamic fluctuations may occur and should be immediately notified to the surgeon. Tissue handling and retraction should be stopped till hemodynamics are normal.

Extubation may be deferred if intraoperative course had multiple hemodynamic changes, impaired lower cranial nerves or expected postoperative respiratory insufficiency.

**Pediatric Head Injuries**

Surgery may be needed for major scalp injuries, depressed skull fractures, extradural or subdural hematomas and decompression of severe cerebral edema or cerebral contusions. Thorough head to toe assessment should be performed. Spine injury should be ruled out. Airway should be secured in all children with Glasgow Coma Score ≤ 8. Intubation is done with manual inline stabilization whenever in doubt. Basilar skull fractures should be suspected in presence of bleeding from nose, ears, CSF rhinorrhea, bilateral black eyes or ecchymosis behind ears. Nasal intubation or insertion of nasogastric tube should be avoided.

The goal of anesthesia should be to minimize secondary insults to the injured brain due to hypotension, hypoxia, hypocarbia and hypercarbia, further increases in ICP, seizures, hypo- or hyperglycemia. Lower dose of intravenous agents is required in hypovolemic patients. Etomidate can also be used. Inhalational agents <1 MAC can also be used. Age appropriate CPP should be maintained above 40–50 mm Hg. Strict normocapnia is targeted. Hyperventilation can only be used for short term control of ICP to facilitate surgical exposure. Hyperglycemia is associated with poor outcomes; sugars should be frequently monitored and maintained between 80–180 mg/dL. Prophylactic anticonvulsant therapy can be considered for seven days post-injury to reduce incidence of post-traumatic seizures.

**Craniofacial Surgeries**

Craniofacial anomalies encompass wide range of defects of cranium and facial skeleton. Craniosynostosis results from premature fusion of cranial sutures affecting brain growth. They may be associated with other congenital anomalies like Apert’s and Crouzon’s syndrome. The child may present with signs of raised ICP, optic atrophy, papilloedema which mandate urgent surgery. The associated syndromes may present with difficult airway management issues. These patients may have obstructive sleep apnea and in long-standing cases pulmonary hypertension. Detailed preoperative assessment identifying airway difficulties, cervical vertebral anomalies (mainly fusion), limb anomalies, difficulty of vascular access, speech, communication problems, cardiac, renal or urogenital anomalies is must. Possibility of postoperative ventilatory support needs to be explained to parents.

Close communication and planning with surgeons is mandatory. In anticipated difficult intubation, it is best to have the patient breathe spontaneously till airway is secured. Backup plan should be ready at all times. Reinforced orotracheal tubes are used, ensuring optimal positioning, firmly fixed and a pharyngeal pack is inserted. In difficult airway, fiberoptic technique may be needed. Submental intubation can be considered as an option to tracheostomy for procedures involving extensive facial osteotomies. The greatest intraoperative challenge is the long duration of surgery and major blood loss. During surgery, there is significant ongoing blood loss or sudden loss due to venous sinus bleeding. Blood should be cross matched and meticulously replaced. Fresh frozen plasma and platelet transfusions are often needed to prevent coagulopathy. Bradycardia during orbital manipulation should be watched for. Hypothermia should be prevented. The child can be extubated postoperatively if intraoperative course is uneventful and blood loss is adequately replaced. In patients with prolonged surgery, unstable hemodynamics, airway edema and difficulty in securing the airway at the start, postoperative ventilator support is indicated. In view of facial edema, head up position is given postoperatively.

**Encephalocele Excision**

Either frontal or occipital encephalocele, both make endotracheal intubation challenging as the anesthesiologists have to intubate the patient in a suboptimal position while taking care to prevent accidental rupture of the lesion. Attempts should be made to have a stable ICP to prevent changes in its size. Anesthesia management is otherwise similar to craniofacial anomalies, though reconstruction is not as extensive. Postoperative CSF rhinorrhea may occur following repair of frontal encephalocele.

**Surgeries for Vascular malformations**

Pediatric cerebrovascular disease is rare and may present as arteriovenous malformations, aneurysm or arteriopathies like Moyamoya disease.
Arteriovenous Malformations

Small children with arteriovenous malformation (AVM) may present with congestive cardiac failure and hydrocephalus. During surgery it is essential to maintain CPP, in spite of the fact that hypotension is desirable to prevent rupture. Induced hypotension in such patients may produce ischemia in adjacent brain tissue and may also lead to venous thrombosis. Adequate wide bore intravenous access and arterial line are necessary as blood loss can be rapid in case of a rupture. Normal perfusion pressure breakthrough can occur in previously hypoperfused vessels adjacent to AVM postoperatively especially in high-flow AVMs.

Moyamoya Disease

Moyamoya disease is a vaso-occlusive disease, affecting mainly the internal carotid artery causing ischemia in the vessel territory. The goal of anesthesia is to maintain normocarbia and avoid hypocarbia during revascularization surgery. Children should be adequately premedicated with good anxiolysis in order to prevent crying which causes hyperventilation and aggravation in symptoms. Anesthetic techniques should avoid hypotension, dehydration and hypothermia to maintain cerebral perfusion through small collateral vessels.

Epilepsy Surgery

Surgery to treat the medically unresponsive and intractable seizures by excision or stimulation of epileptic focus is now increasingly practised. Detailed history should include type of seizure, onset, duration and treatment. Longstanding anticonvulsant use predisposes the patient to liver dysfunction, enzyme induction and coagulopathies. They also alter the metabolism and clearance of opioids and neuromuscular blocking agents. The procedure may involve placement of cortical mapping electrodes after craniotomy to identify epileptogenic focus.

Anesthesia goals are similar to other neurosurgical procedures. For induction, sevoflurane is commonly used. In children with intravenous access, propofol 2–4 mg/kg or thiopentone 5–7 mg/kg may be used. Propofol is also epileptogenic in low doses, but in anesthetic doses, it suppresses seizure activity. Use of peripheral nerve stimulator is recommended. If motor area is to be identified, neuromuscular blockade needs to be withheld. Anesthesia is usually maintained with inhalational agents with or without nitrous oxide. Phenytin may cause hypotension and arrhythmias if given rapidly.

In case of older cooperative children, an awake craniotomy with scalp nerves block for excision of the epileptic focus may be undertaken. Preoperatively rapport must be established with the child. Procedural sedation is commonly provided using dexmedetomidine, propofol, midazolam, fentanyl or remifentanil can be used with child breathing spontaneously and alert, obeying at the time of cortical stimulation. The face should be accessible to the anesthesiologist. Intraoperative seizures may occur when target area is stimulated. In such situation, bolus dose of thiopentone or propofol and irrigation of the area with cold Ringer lactate may be used.

Anesthesia for Surgeries involving Neurophysiological Monitoring

Many neurosurgical procedures involve intraoperative neurophysiological monitoring for assessment of functional status of different neural pathways. Spontaneous electrical activities like electrocorticography (ECoG) or electroencephalography (EEG) are used to identify specific cortical areas during excision of tumor involving eloquent areas or epilepsy surgery. Similarly evoked potential monitoring like somatosensory evoked potential (SSEP) monitoring and motor evoked potential (MEP) monitoring is used to assess the integrity of sensory and motor pathways respectively when surgical manipulation poses a risk to these pathways in cranial or spinal surgeries. Auditory evoked potentials (AEP) are often used for posterior fossa surgeries. All anesthetic agents affect EP to more or less extent. Hence, it is essential to use an appropriate strategy and to maintain constant levels of anesthetic drug during recordings. In small babies, the monitoring as well as its interpretation is different due to immature nervous system.

All fluorinated inhalational anesthetic agents increase latency and decrease amplitude of cortical signals in a dose dependant manner. Nitrous oxide also produces depressant effects on SSEPs especially in neurologically impaired patients. Intravenous agents produce lesser effects on evoked responses as compared to inhalational agents. Thiopentone is shown to preserve SSEPs even in larger doses. Propofol, though it increases the latency and decreases the amplitude of evoked potentials, it is considered as acceptable for use as a part of neuroanaesthesia technique. Sudden boluses of any agent should be avoided. Opioids do not affect SSEP significantly and hence opioid-based anesthesia provides better monitoring conditions. However, use of multiple drugs may produce additive effect. Use of dexmedetomidine is acceptable with evoked potential monitoring, though data is limited. MEPs are affected significantly by all inhalational agents.
agents. Though neuromuscular blocking agents can affect MEPs, recordings can be done when two twitches are present on train-of-four monitoring. Brainstem potentials like auditory evoked potentials are more resistant to changes by anesthetic agents and amplitude of signals are not affected in clinical doses. Therefore, most anesthesia techniques used in neuroanesthesia are acceptable. It is to be noted that apart from anesthetic agents, EEG and SSEPs are also affected by severe hypoxia, hypotension, regional ischemia and hypothermia. A constant depth of anesthesia should be maintained and hemodynamic manipulations if needed should be done with vasoactive drugs rather than anesthetic agents.

**LEARNING POINTS**

- Pediatric patients should not be treated as small adults as they have distinct neuroanatomy and neurophysiology
- Thorough preoperative documentation of preexisting neurological deficits is required—Associated syndromes should be ruled out
- Increased ICP may cause unconsciousness, hypertension, bradycardia, aspiration and hypoxia. Sudden deterioration in status may occur
- Main goals are to maintain normal ICP, maintain CPP, MAP, normocarbia, normoglycemia, normothermia, thereby avoiding further neurological injury. Providing optimum surgical condition and rapid and smooth emergence are equally important
- Apart from these, knowledge of concerns pertaining to individual surgeries enables the anesthesiologist to provide excellent perioperative care on a case to case basis
- When intraoperative electrophysiological monitoring is planned, a team approach involving anesthesiologist, surgeon and neurophysiologist with appropriate alteration in anesthesia technique is needed

**REFERENCES**

INTRODUCTION
Pediatric liver diseases encompass a wide group of disorders ranging from congenital abnormalities, inherited metabolic disorders, abnormalities in bilirubin metabolism and excretion, to overt liver failure from acute or chronic conditions. The purpose of this chapter is to provide a functional classification of liver disease for understanding to the anesthesiologist. Disorders have been grouped as liver diseases for incidental surgery or treatment and end stage liver disease needing transplantation.

Children with Liver Disease for Anesthesia

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GENERAL PRINCIPLES OF PEDIATRIC ANESTHESIA IN LIVER DISEASES

Functions of the Liver
The functions of the liver include maintenance of carbohydrate, protein and fat metabolism. It also plays a role in the synthesis of clotting factors, acute phase reactants and proteins. As an end organ most of the drugs are metabolized by the liver by oxidation or hydrolysis. The liver is also the end organ for the breakdown of lactate to bicarbonate and lactate from the skeletal muscle, kidney and red blood cells is broken down to maintain the normal levels of 0.2–2.4 mmol/L.

Anesthetics and Liver Functions
The hepatic blood flow is determined by dual flow through the portal vein and hepatic artery, the two having a semi-reciprocal relationship called the hepatic arterial buffer response (HABR). This implies that a fall in portal vein flow is compensated by an increase in hepatic arterial flow through the release of adenosine but the converse cannot occur. A fall in blood pressure can reduce the liver blood flow and this can be offset by drugs that increase the mean arterial pressure (MAP). Desflurane, sevoflurane and isoflurane are safe for use in patients with liver disease in decreasing order of safety. Among induction agents, although propofol, etomidate and ketamine are lipid soluble with a high hepatic extraction ratio, their duration...
of action is not prolonged and elimination of half lives are similar to normal children. Regional anesthesia is safe in the absence of coagulopathy but significant reductions in blood pressure proportionately decrease hepatic blood flow. Dexmedetomidine has a decreased half-life and prolonged clearance in patients with hepatic dysfunction part of which could be due to altered protein binding. Fentanyl is lipid soluble with a short half-life because of redistribution to storage sites and with prolonged administration cumulative effects could occur. Sufentanil has a similar profile to fentanyl and pharmacokinetics safe for use in liver diseases. Alfentanil has a prolonged half-life, almost double that of normal in patients with liver diseases and is best avoided in this group. Remifentanil is metabolized by tissue esterases that hydrolyze the ester linkage with elimination that is independent of liver function and is the safest opioid for use in children with liver diseases. Amongst muscle relaxants, succinylcholine and mivacurium are metabolized by pseudocholinesterase, the levels of which are altered in chronic liver disease. Vecuronium is metabolized by hepatic metabolism and its action prolonged with liver disease. Atracurium and cisatracurium have a non-end organ linked route of elimination by ester hydrolysis and are considered to be safe for use in liver disease. Sugammadex can be safely used in liver disease as its excretion is through the kidney.

Paracetamol is a weak nonsteroidal anti-inflammatory drug (NSAID) that in large doses produce hepatotoxicity due to depletion of glutathione in the hepatocytes. It can be used in smaller doses or for a shorter time in liver diseases. Other NSAIDs (cox inhibitors) can rarely produce hepatotoxicity by an idiosyncratic reaction.

**Fluid Management**

Perioperative fluid management is according to standard guidelines and the Holliday-Segar formula. It has been our practice to avoid the use of half normal saline as hyponatremia can occur. Normal saline is slightly hypertonic and is known to be associated with hyperchloremic metabolic acidosis when large volumes are used. Ringer’s lactate (RL) is a balanced salt solution and most optimal substitute, even if the sodium is less than that present in plasma. However, the base in Ringer’s lactate is lactate that is normally converted to bicarbonate by the liver, hence the standard use in children with liver disease cannot be recommended. Plasmalyte is the alternative balanced solution that is available and contains 140 mEq/L sodium, 98 mEq/L chloride, 27 mmol/L acetate and 24 mmol/L gluconate as a base; this has an advantage of extrahepatic metabolism. It has been extensively used in adults. Although reports of its use in children are still lacking. Plasmalyte 148 (Baxter-IL) is emerging as an alternative fluid in pediatric surgeries.

Certain groups that need glucose supplementation during surgery include preterms, neonates, children in sepsis or receiving parenteral nutrition, liver disorders and prolonged surgeries supplemented by regional anesthesia. We use 2% dextrose in RL for children less than 1 year and 1% for those older. In case the liver metabolism contraindicates the use of lactated solutions then either Plasmalyte-148 or normal saline could be used.

**Liver Function Tests and Interpretation**

Bilirubin elevations are seen with overproduction (hemolysis) or obstruction, either intra- or extrahepatic. Among amino transferases, alanine aminotransferase (ALT) is specific for hepatocyte necrosis while aspartate aminotransferase (AST) is common to the liver, skeletal and cardiac muscle, kidney and the brain. Highest elevations of both enzymes are seen with viral hepatitis, toxic and ischemic hepatitis. Alkaline phosphatase (ALP) is elevated in obstructive jaundice and simultaneous measurement of 5’ nucleotidase helps in establishing a hepatic etiology to the elevations. Prothrombin time (PT) and albumin levels are tests for synthetic functions of the liver; prothrombin time is affected earlier than albumin levels.

The PELD score is a pediatric end stage liver disease scoring for children less than 12 years old. This calculates scores based upon bilirubin, albumin, INR, age and growth failure and is used for prognostication and for allocation on transplant waiting lists. It was found that PELD scores above 17 correlates with survival benefit after transplantation and increasing values of PLED has increasing benefit after transplant. There is no value of PELD above which survival is decreased and hence this score should not be used as a marker for futility.

**Physiological Jaundice in the Neonate**

Jaundice is not an uncommon biochemical finding in neonates scheduled for surgery. The immaturity of the liver and kidneys poses concerns as this reflects metabolism and excretion of administered anesthetic drugs. Unconjugated hyperbilirubinemia in the newborn is due to defect in the conjugation of bilirubin to bilirubin glucuronide. This may be due to a transient defect in conjugation, along with the increased turnover of red blood cells from resolution of bruises and cephalhemosmas. It is commonly seen in preterm infants, Rh isoimmunization, Gilbert’s syndrome and in exclusively breastfed infants (breast milk jaundice). Coexistence of Gilbert’s syndrome
with diseases producing hemolysis like G-6PD deficiency may cause high elevations in bilirubin levels predisposing to kernicterus. Levels for treatment vary with the weeks of gestation and the intensity of jaundice; a preterm child may need attention earlier than a full-term neonate. Phototherapy alters the shape of bilirubin and converts it to a soluble form that is excreted even if not conjugated. It is important for the anesthetist to be aware of hyperbilirubinemia and consequent implications of hepatic enzyme immaturity.

**Biliary Atresia**

The presentation is usually of a term infant feeding normally with weight gain but with increasing jaundice and pale stools. The incidence varies between 1 in 9600 in Japan and China to 1 in 16,000 in UK and Europe. It is classified as type I that affects the common bile duct and proximal cystic duct, type II that affects the common hepatic duct and type III that affects the entire extrahepatic biliary tree. Type III is the most commonly encountered form in infants. Investigations reveal obstructive jaundice with Alkaline phosphatase (ALP) elevations. The prothrombin time and serum albumin are usually normal at this stage of presentation and preemptive vitamin K administration can correct coagulopathy. The diagnosis is made with an abdominal ultrasound and confirmed peroperatively with a cholangiogram (Fig. 1).

The surgical procedure is a portoenterostomy called the Kasai’s procedure, which involves excision of the entire extrahepatic biliary tree with transection of the fibrous portal plate near the hilum of the liver. Bilioenteric continuity is then re-established with a Roux-en Y limb. The ultimate goal of the procedure is to allow drainage of the bile from the liver into the Roux limb via microscopic ductules in the portal plate. Surgical results are best when the surgery is performed prior to 65 days of age; the surgery is palliative and these children grow to need a transplant in the future. Vitamin K should be administered preoperatively to correct coagulation abnormalities due to obstruction in the flow of bile. Fasting guidelines are standard and the child allowed clear fluids until 2 hours prior to surgery. Separation is not a problem for children less than 3 months of age.

**Anesthetic Concerns**

- **Anesthetic drugs and hepatic metabolism:** To be titrated according to general principles given above.
- **Coagulopathy due to vitamin K malabsorption:** corrected by parenteral vitamin K.
- **Prolonged surgery:** Hypothermia. Use warm fluids, forced air warmer, wrapping the child’s limbs in plastic to maintain temperature.
- **Fluid and Electrolytes:** A central line is useful to monitor volume status and metabolic profile.
- **Children can usually be extubated at the end of the procedure. Postoperative complications are mainly related to cholangitis. Breastfeeds through a nasogastric tube are commenced as breast milk contains lipases and bile salts that aid lipid hydrolysis and micelle formation. Levels of bilirubin higher than 6 mg/dL three months post-portoenterostomies are indications for early referral for liver transplant.**

**Disorders of Bilirubin Metabolism**

**Gilbert’s, Crigler Najjar, Dubin Johnson syndromes:** Gilbert’s syndrome is a benign disorder of bilirubin metabolism due to deficiency in UDP glucuronosyltransferase 1 polypeptide (A1) (UGT1A1) enzyme in the range of 70–80%. The condition is often detected incidentally during evaluation of an unrelated complaint. Jaundice manifests after fasting, stress, fever, exercise or infection as an increase in unconjugated bilirubin. These children do not pose any problem during anesthesia; symptomatic relief from jaundice can be obtained by enzyme inducing agents like phenobarbital and carbamazepine. A subgroup of Gilbert’s syndrome is known to have increased sensitivity to paracetamol.

Crigler-Najjar syndrome is a rare autosomal recessive disorder due to total or near total deficiency of the enzyme UGT1A1. In type I, there is a total deficiency of this enzyme manifesting as jaundice with kernicterus and death if untreated. In type II, small amounts of the enzyme are
present and may be managed with phenobarbital therapy
and are associated with survival till adulthood.

Dubin Johnson’s syndrome is caused by a gene
mutation responsible for transport of conjugated bilirubin
across the hepatocyte and is autosomal recessive in
transmission. This usually manifests as intermittent
obstructive jaundice in adolescence and is confirmed by
an increased urinary finding of coproporphyrin I to III
when erythropoietic porphyria or arsenic poisoning are
excluded.

**Alpha1antitrypsin Deficiency**

Although this is reportedly a common cause for end stage
liver disease in children, the occurrence in India is low.
A variant gene coding for alpha-1 antitrypsin results in
decreased levels of circulating antitrypsin. Liver diseases
in this group vary from chronic hepatitis to cirrhosis that
may need a liver transplant. Coexistent emphysema may
add to the morbidity in this group.

**Viral Hepatitis**

Viral hepatitis is a common cause for infective hepatitis
in developing countries and infections with A and E are
common in our country. Vertical transmission can occur
with hepatitis B and hepatitis C, and is associated with
transfusions. Acute infection is associated with fever,
malaise, jaundice, elevated transaminases (>1000 IU/L)
and sometimes, acute fulminant hepatic failure. None
other than emergency surgeries need to be performed
in the acute phase. Elective surgeries are deferred until
complete recovery with return of enzymes to normal.

In an emergency surgery during acute infection
with viral hepatitis, the management of anesthesia will
depend upon the clinical presentation of the patient.
Anesthetic agents will reduce blood supply to the liver
and inhalational agents can produce hepatocyte injury.
The choice of regional versus a general anesthetic will
depend upon the nature of surgery. Regional anesthesia is
not contraindicated if the patient is not decompensating
and the INR less than 1.5 without corrective transfusions.
When anesthetizing a patient with a history of jaundice,
serological testing is important. Besides standard
screening for serology, persistent HBsAg, HBeAg may
indicate an infectious state and poses a risk for health
care.

**Wilson’s Disease**

Wilson’s disease is a familial, autosomal recessive
transmitted disease of copper metabolism characterized
by decreased levels of serum ceruloplasmin and
deposition of copper in the brain, cornea and kidneys.
The gene encodes a metal transporting ATPase that is
found on the hepatocytes and regulates transmembrane
transport of copper resulting in inability to excrete copper into the bile. Initial testing reveals decreased
levels of ceruloplasmin (<20 mg/dL), increased serum
free copper (>25 mcg/dL), increased urinary copper
(>100 mcg/dL) and Kayser-Fleischer (KF) rings. Further
evaluation includes evaluation of the liver copper content,
ultrastructural and gene studies.

Children may be asymptomatic or may present with
hepatomegaly and raised aminotransferases detected
incidentally. Children are usually treated with a chelating
agent, penicillamine or trientene both of which act by
binding to copper. Zinc is also used and it acts by blocking
the intestinal absorption of copper by stimulating synthesis
of endogenous copper chelators like metallothioneins.

The presentation is either as a chronic liver disease
or as neurological or psychiatric manifestations. Some
patients with Wilson’s may present with an acute liver
failure (ALF) that has a characteristic pattern. They
present with a Coomb’s negative hemolytic anemia,
coagulopathy unresponsive to vitamin K, tendency for
progression to renal failure, elevation in transaminases,
normal or decreased alkaline phosphatase and a female
predilection.

Management of a child with Wilson’s disease is on the
standard line of management with liver abnormalities.
Cautious use of drugs that undergo hepatic metabolism
and vigilance in the postoperative period for worsening
of hepatic function may be needed. The response to
pharmacological management is excellent even with
established evidences of hepatitis and transplant is
indicated in irreversible chronic liver failure or acute
fulminant failure.

**Non-alcoholic Steatohepatitis (NASH) and
Obesity syndromes in children**

Obesity is not uncommon even in developing countries,
the terms obese and overweight defined as weight more
than 2 standard deviations (SD) and 1 SD above the WHO
median for age respectively. Waist-height ratio, body
mass index and Z score of BMI have all been used for
predicting obesity in children and studies have shown that
the waist-height ratio has the greatest prediction with the
development of metabolic syndrome in children. NASH
is emerging as a leading cause of chronic liver diseases in
children.

The presence of elevated liver enzymes AST and ALT
in an obese child presenting for incidental surgery should
prompt an evaluation for steatohepatitis. NASH in children
is defined as the presence of more than 5% steatosis in hepatocytes (liver biopsy) in the absence of other causes. The spectrum of steatohepatitis varies from intrahepatic fat accumulation to varying degrees of inflammation and fibrosis in the liver, the end stage being cirrhosis.

Anesthetic concerns in the management include obstructive sleep apnea, difficult intravenous lines and difficult airway in addition to deranged hepatic metabolism of anesthetics. Sometimes, fatty liver may be identified during incidental surgery. Although the liver is not dysfunctional and can metabolize lactate present in Ringer’s lactate, acetate containing solutions may be preferred. The anesthesia management is on the standard lines for the management of liver disease.

**Familial Hypercholesterolemia**

Familial hypercholesterolemia (FH) is a common genetic disorder affecting 1 in 200 to 1 in 250 births and is a cause for premature coronary artery disease. The disease exists as heterozygous and homozygous forms with an autosomal dominant transmission. Genetic disorder produces a defective receptor or reduction in the low density lipoprotein cholesterol (LDL-C) receptor on the surface of the hepatocytes resulting in increased levels of circulating LDL-C. Cholesterol deposits on the vascular endothelium, causing inflammation, accelerated atherogenesis and premature coronary vascular disease.

This entity is under diagnosed in our country and only the homozygous forms are picked up when the child develops ischemic heart disease or symptoms of aortic stenosis due to subvalvular fat deposition. Early screening and detection with diet modification and statin therapy restores near normal life expectancy in the heterozygous population. Family history of coronary artery disease with elevated LDL-C is a screening criterion. Values of LDL-C more than 190 mg/dL on two successive readings after 3 months of dietary interventions are suggestive of FH. In case of a positive family history or elevated LDL-C the setting is suggestive of FH and the need for intensive therapy. If there is a positive genetic testing along with a reading of LDL-C more than 130 mg/dL this would also suggest FH in a child.

An elevated LDL-C level >500 mg/dL with premature manifestations of coronary artery disease (pulmonary edema/MI) along with aortic valve disease, tendon xanthomas in the hands, elbows and Achilles tendon is a presumptive diagnosis of homozygosity. Children with elevated LDL-C may present for incidental surgery, however a diagnosis is made on the exclusion of secondary causes like hypothyroidism, nephrotic syndrome, obesity, anorexia nervosa and iso-retinoid administrations. If a child presents for an elective surgery, it can be deferred for 1–2 months with dietary modifications and exercise. If the levels persist for more than 3 months, then statin administration may be warranted. Occurrence of IHD in heterozygous states in children is rare but studies have documented increase in the intimal carotid lining suggesting an increased risk. Homozygous states may present for aortic valvular surgery and rarely for liver transplantation.

**Choledochal Cyst and Cholelithiasis**

Choledochal cyst is a congenital abnormality in the biliary tract that can involve both intra- and extrahepatic biliary radicles. The symptoms and age at onset are variable, but are usually associated with right upper quadrant pain and vomiting and features of cholecystitis or pancreatitis. The surgery in types I, II and IV involve excision of the dilated biliary duct and restoration of biliary enteric continuity either by a hepatic Roux en Y or by closure of the defect in the common bile duct. Type III is a choledochocele and endoscopic sphincterotomy can be a therapeutic option for cysts lesser than 3 cm. Large cysts need surgical excision sometimes with a reimplantation of the pancreatic ducts in the duodenum (Fig. 2). Type V is also known as Caroli’s disease characterized by cystic dilation of the intrahepatic bile duct, whereas Caroli’s syndrome refers to the presence of associated congenital hepatic fibrosis. The treatment for Caroli’s disease involves supportive care for cholangitis.
and hepatolithiasis. Surgical resection has been used successfully in patients with monolobar disease. For patients with diffuse involvement, the treatment of choice is orthotopic liver transplantation.

Management of anesthesia for types I to IV is not very different from standards followed for children undergoing abdominal surgery. Surgery is to be scheduled after treatment for episodes of cholangitis. An epidural catheter can be inserted for intra- and postoperative analgesia, lower thoracic and upper lumbar sites are commonly chosen. If a liver lobe resection is also included in the surgery, the remnant liver functions should be considered while siting the epidural, as there is a possibility of coagulopathy postoperatively. In smaller children, the epidural can be substituted with a caudal analgesia, and clonidine as an additive to bupivacaine or ropivacaine in doses of 1.0–1.5 mcg/kg to increase duration of analgesia.

The management of fluids and choice of anesthetic agents will follow the general principles for liver diseases.

**Cholelithiasis**

Gallstones in children are encountered in some forms of hemolytic anemia. A thorough hematology workup is essential prior to planned surgery. Laparoscopic cholecystectomy is indicated in symptomatic gallstones. Elective cholecystectomy is indicated even in the absence of symptoms in sickle cell anemia.

**Neoplasms**

Benign cystic tumors of the liver are rare and may need a surgical excision (Fig. 3). Amoebic liver abscesses and hydatid cysts are seen in tropical countries. The risks of surgery depend upon the volume of liver involved and immunologic reactions mediated by rupture. Anaphylactic reactions can occur with rupture of hydatid cysts while pyogenic cysts can result in peritoneal reaction.

Among malignant tumors, hepatoblastomas are most common, with a reported incidence of one per million. Children may present at about 3 years with lethargy and a large abdominal mass. These children may need chemotherapy with doxorubicin or cisplatinum prior to surgical excision or transplant if there is no extrahepatic spread. Children receiving doxorubicin need cardiac evaluation prior to surgery on account of drug related cardiotoxicity.

Anesthetic concerns besides the standard precautions in liver disease include blood loss, hypotension, venous embolism and coagulopathy. A 5.0 Fr triple lumen catheter in the IJV provides venous access for volume replacement and inotropes. If peripheral veins are inaccessible on account of chemotherapy, an additional 4 Fr or 5.0 Fr catheter can be inserted alongside the IJV with the guide wires of both inserted first. Blood and plasma can be replaced commensurate with losses at 10–15 mL/kg body weight. In addition to standard laboratory tests for coagulation, dynamic tests like thromboelastography can guide transfusion of products.

Hepatic dysfunction also results from large-volume parenchymal resection, which is suggested by increasing metabolic acidosis, hypoglycemia, and coagulopathy. Management is supportive until the liver recovers and may take 2–5 days. N-acetylcysteine is used as an antioxidant and as a precursor for liver glutathione and may have a role in liver recovery. Epidural analgesia may be inadvisable in cases involving large parenchymal resections. The postoperative issues relate to pain management, coagulopathy and residual liver dysfunction.

**Liver Diseases Presenting for Transplant**

Multiple etiologies of liver diseases in children converge into transplant as a treatment option. In India, biliary atresia, Wilson’s disease, autoimmune hepatitis are the most common indications for non-emergent transplant. Acute fulminating hepatitis following viral hepatitis, autoimmune hepatitis and accidental poisonings (rat poison: zinc phosphide) are causes for liver disease in small children. Metabolic disorders such as primary hyperoxaluria (requires combined liver and kidney transplant), glycogen storage disorder can present for transplant.

**Chronic liver disease (CLD)** in children involves multiple systems and is classified in severity according to the PELD score. The pathophysiology in cirrhosis is the toxic injury to the hepatocyte from inflammatory mediators resulting in fibrosis with nodular regeneration.
Consequence of fibrosis is the development of portal hypertension and splenomegaly with changes in various organ systems.

**Cardiovascular system:** Hyperdynamic circulatory state with decreased systemic vascular resistance is a feature of CLD. Cirrhotic cardiomyopathy is identified by global dyskinesia and poor systolic function on transthoracic echocardiography. Pericardial effusions are sometimes seen associated with hypoalbuminemic states.

Portopulmonary hypertension (POPH) refers to the increase in pulmonary artery pressure (PAP) and pulmonary artery resistance (PVR) in the background of CLD. It is classified as mild, moderate and severe when the mean PAPs are greater than 25, 35 and 45 mm Hg respectively with a PVR more than 240 dyne/sec/cm\(^5\) in the presence of normal left atrial pressures. Values of mPAP >40 mm Hg would contraindicate a transplant while values between 30–45 mm Hg can be considered for reassessment after a trial of pulmonary vasodilators. However changes in the portal circulation take about 7 years after establishment of CLD and are probably the reason why POPH is not very common amongst children with CLD.

**Respiratory System:** Children with large ascites show restrictive lung disease and decreased functional residual capacity. Hepatic hydrothorax refers to a collection of pleural fluid in the absence of cardiac, lung or pleural disease. Hepatopulmonary syndrome (HPS) refers to a triad of CLD, pulmonary arteriolar dilations and hypoxemia and is classified as mild, moderate, severe and very severe if the PaO\(_2\) is more than 80 mm Hg, between 60–80 mm Hg, 50–60 mm Hg, less than 50 mm Hg and not improving to more than 300 mm Hg with 100% oxygen administration. Hepatopulmonary syndrome is confirmed by contrast echocardiography with microbubble echocardiography and quantified by a macrolabeled albumin scan. In rare cases, the dilatations are discrete and may need coil embolization. Resting PaO\(_2\) < 50 mm Hg is associated with higher morbidity postoperatively and poorer outcomes (Fig. 4).

**Gastrointestinal System:** The development of portal hypertension results in portosystemic collateral circulation and bleeding esophageal varices can result in encephalopathy and anemia. Banding may be required for children with varices and GI bleed.

**Central Nervous system:** Encephalopathy occurs secondary to GI bleed or an infection and ammonia is the neurotransmitter incriminated. Antibiotics, control of GI bleed, lactulose are usual measures in management.

**Renal System:** Hepatorenal syndrome (HRS) is the renal dysfunction associated with extreme splanchnic vasodilatation and renal vasoconstriction, treatment for which is a liver transplant. This is not very common in children with CLD, but ALF with renal failure can occur and carries a poorer prognosis. Splanchnic vasoconstriction with terlipressin can correct HRS as it improves renal perfusion. In ALF, mannitol is used to reduce the cerebral edema along with hypertonic saline.

**Hematopoietic:** The liver synthesizes all clotting factors except factors VIII and vWF. Liver failure is associated with coagulopathy and needs adequate clotting factors for correction.

**Fulminant hepatitis** from HAV is common in India and presents with encephalopathy, deranged liver function tests, coagulopathy and jaundice. The major concern in these patients is cerebral edema with intracranial hypertension with potential for coning and brain death. The decision for transplantation is based upon the King’s College Criteria for non-paracetamol poisoning.

- Single Criterion: INR >6.5 (PT >100 seconds)
- Any 3 of the following: Age <10 or >40 years; etiology non-A, non-B hepatitis, or idiosyncratic drug reaction; duration of jaundice before hepatic encephalopathy >7 days; INR >3.5 (PT >50 seconds); serum bilirubin >300 micromol/L (>17.6 mg/dL).

The management during transplant is beyond the scope of this chapter. The highlights include identification of an optimal time free from infection and decompensation. Intraoperative management needs correction of coagulation, use of vasopressors and inotropes and support of the metabolic state until functions of the new graft take up.
LEARNING POINTS

• Children with liver diseases may present for surgeries that range from minor or incidental to major resections and transplant
• Improvisations in anesthetic techniques and safer drugs have improved outcomes for these patients
• Children with end stage liver diseases are best cared for in tertiary care centers with multidisciplinary backup including support systems for emergency transplant

ACKNOWLEDGMENT

I wish to thank Dr Bindu Sudarsan (Pediatric surgeon) and Dr Geetha Saril (Pediatric gastroenterologist) for their valuable inputs in the preparation of this chapter.

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INTRODUCTION

Pediatric urologic procedures make up about 40% of all procedures in a pediatric OT. Most of them are day care procedures. Often as with other surgeries, understanding the surgical procedure and its nuances goes a long way in providing a suitable anesthetic. In this chapter, we have attempted to briefly explain the surgical problem before discussing the anesthetic implications.

Most pediatric urologic cases are elective procedures giving us ample time to optimize the patient prior to surgery. The few emergencies include testicular torsion, suprapubic cystostomy for drainage in acute urinary retention, and percutaneous nephrostomy in an infected system. Table 1 lists common conditions and procedures encountered and age at presentation.

RENAL PHYSIOLOGY AND ANESTHESIA

The kidney controls body fluid composition, blood pressure, hematopoiesis, calcium metabolism, besides excreting important waste products. Glomerular filtration rate (GFR) is the volume of plasma filtered by the kidneys per minute and this is about 125 mL. This value is reached at 2 years of age. The GFR is maintained at a constant rate over a wide range of blood pressures by the mechanism of renal autoregulation. Various drugs and anesthesia can interfere with this autoregulation predisposing to acute kidney injury (AKI) or acute renal failure (ARF). Inhalational anesthetics generally reduce GFR and urine output, mainly by extrarenal effects that can be reduced by adequate hydration preoperatively. Opioids, barbiturates and benzodiazepines also reduce GFR and urine output. The hemodynamic effects of regional anesthesia seem to be less in children compared to adults, as the sympathetic system is not well developed up to 12 years of age. These perioperative alterations of renal function are usually

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**Table 1: Common conditions and procedures encountered and age at presentation. Timing of surgery is variable, depending on age at which diagnosis is made***

<table>
<thead>
<tr>
<th>Surgical Condition/Procedure</th>
<th>Age at presentation*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cystoscopy, Posterior Urethral Valve (PUV) fulguration</td>
<td>Newborn, older child</td>
</tr>
<tr>
<td>Bladder Extrophy closure</td>
<td>Newborn/older child</td>
</tr>
<tr>
<td>Pyeloplasty for Pelviureteric junction (PUJ) obstruction (usually unilateral)</td>
<td>Infants usually upto 3 months</td>
</tr>
<tr>
<td>Ureteric reimplantation (maybe unilateral/bilateral)</td>
<td>Usually above 2 years</td>
</tr>
<tr>
<td>Orchidopexy, Unilateral/bilateral</td>
<td>Above 1 year</td>
</tr>
<tr>
<td>Hypospadias repair</td>
<td>Around 11 months to 3 years. Repeat procedures in older children</td>
</tr>
<tr>
<td>Clitoroplasty/Vulvovaginoplasty</td>
<td>3 months/older child</td>
</tr>
<tr>
<td>Laparoscopy - to identify testis/staged repair/to identify gonads in intersex</td>
<td>Newborn to older child</td>
</tr>
<tr>
<td>Tumor surgery - Laparotomy/ Biopsy/excision for Wilm’s tumour/Adrenal Adenoma</td>
<td>Infants, upto 5 years</td>
</tr>
<tr>
<td>Central line/port insertion</td>
<td>Any age</td>
</tr>
<tr>
<td>Peritoneal Dialysis (PD) catheter insertion</td>
<td>Newborn to older child</td>
</tr>
</tbody>
</table>

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transient and may not be clinically relevant in normal patients. But in those patients with compromised renal function they become important.

Creatinine is an end product of skeletal muscle metabolism. It is freely filtered by the body and not reabsorbed in the kidneys. It is used as a marker of kidney function. When the GFR reduces by 50%, serum creatinine rises. The trend in the serum creatinine is important as individual value is related to skeletal mass. So, if we have a preoperative value of creatinine and the value rises postoperatively, we know there has been some renal injury.

On the other hand, urea being a waste product of protein metabolism in the liver is freely filtered, but also reabsorbed. It is a poor marker of renal function being increased with increased protein intake and catabolism. Its reabsorption is increased in response to vasopressin in dehydration, so it is used as a marker in dehydration.

Various factors can predispose to acute kidney injury (previously called ARF) in the surgical patient. Preoperative dehydration, sepsis, increased insensible losses due to fever, intraoperative blood loss, third space loss and loss through ryle’s tube or drains can all contribute to a situation leading to AKI.

Some of the drugs should be used with caution in patients with renal dysfunction preoperatively.

Nonsteroidal anti-inflammatory drugs are analgesics which have a significant effect on renal function. They inhibit prostaglandin mediated dilatation of the afferent glomerular arteriole, and hence, autoregulation, the purpose of which is to maintain renal blood flow in the face of systemic vasoconstriction, e.g. in hypovolemia. They can also cause interstitial nephritis. They must be used with caution in patients with renal dysfunction.

Angiotensin converting enzyme (ACE) inhibitors and angiotensin receptor blockers prevent local action of bradykinin, which again affects renal autoregulation. These drugs are stopped on the day of surgery, for renal protection and to prevent hypotension.

High doses of aminoglycosides (gentamicin) can cause renal tubular toxicity and are best avoided in patients with renal dysfunction.

Acute interstitial nephritis (AIN) can also occur with certain antibiotics, e.g. penicillin, cephalosporin and flouroquinolone.

IV contrast agents can cause serious vasoconstriction in vulnerable patients and should be used with caution. Smaller doses (as small as is necessary) and prehydration with IV fluids can overcome this problem.

Among the anesthetic agents, the induction agents drop the blood pressure with the exception of ketamine.

### Table 2: RIFLE criteria for acute renal dysfunction (adults)

<table>
<thead>
<tr>
<th>Category</th>
<th>GFR creatinine</th>
<th>Urine output (UO)</th>
<th>Sensitivity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Risk</td>
<td>Increased creatinine x 1.5 or GFR decrease &gt;25%</td>
<td>UO &lt;0.5 mL/kg/h x 6 h</td>
<td>High sensitivity</td>
</tr>
<tr>
<td>Injury</td>
<td>Increased creatinine x2 or GFR decrease &gt;50%</td>
<td>UO &lt;0.5 mL/kg/h x 12 h</td>
<td></td>
</tr>
<tr>
<td>Failure</td>
<td>Increase creatinine x3 or GFR decrease &gt;75%</td>
<td>UO &lt;0.3 mL/kg/h x 24 h or anuria x 12 h</td>
<td>High specificity</td>
</tr>
<tr>
<td>Loss</td>
<td>Persistent ARF = complete loss of kidney function &gt;4 weeks</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ESKD</td>
<td>End Stage Kidney Disease (&gt;3 months)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Source: Adapted from ADQI 2004 consensus

Abbreviations: GFR, glomerular filtration rate; ARF, acute renal failure; ESKD, end stage kidney disease

### Table 3: Pediatric RIFLE criteria—pRIFLE

<table>
<thead>
<tr>
<th>Estimated CCI</th>
<th>Urine output</th>
</tr>
</thead>
<tbody>
<tr>
<td>Risk</td>
<td>eCCI decrease by 25%</td>
</tr>
<tr>
<td>Injury</td>
<td>eCCI decrease by 50%</td>
</tr>
<tr>
<td>Failure</td>
<td>eCCI decrease by 75% or eCCI &lt;35 mL/min/1.73 m²</td>
</tr>
<tr>
<td>Loss</td>
<td>Persistent failure &gt;4 weeks</td>
</tr>
<tr>
<td>End stage</td>
<td>End-stage renal disease (persistent failure &gt;3 months)</td>
</tr>
</tbody>
</table>

Abbreviations: eCCI, estimated creatinine clearance; pRIFLE, pediatric risk, injury, failure, loss and end-stage renal disease

Volatile anesthetic agents like isoflurane and sevoflurane release nephrotoxic fluoride, which theoretically harms the kidney, although there is little evidence to suggest we avoid these agents.

In the event of AKI, the RIFLE criteria (Table 2) helps delineate the extent of injury. It looks at serum creatinine or GFR and urine output to quantify the extent of renal injury. A 1.5–2 fold increase in creatinine and a fall in urine output to 0.5 mL/kg over 6 hours puts the patient at increased risk for renal dysfunction. In children since most often a baseline creatinine value is not available, estimated creatinine clearance is used (pRIFLE)(Table 3).

### Renal Function in the Newborn

- Growth of the kidney is completed at 36 weeks gestation and no further nephrons are produced. Any further increase in renal size is due to the growth of tubules.
- Urine in infants is dilute and adult values of osmolality are reached by 1 year of age. Infants tolerate fluid restriction poorly and are prone for dehydration.
The GFR at term is low and reaches adult values only at 2 years of age. Creatinine at birth reflects the mother’s creatinine and falls to reflect renal function of the baby by 1 week of age.

- The neonate's limited renal function is appropriate to the period of rapid growth after birth. However, in the postoperative period, the neonate is more vulnerable to renal insufficiency and cannot handle fluid or sodium overload.4

### PREOPERATIVE ASSESSMENT

A good history and clinical examination is often sufficient in most routine cases. Some of the patients coming for urologic procedures might have had many visits to the hospital for a previous urinary tract infection (UTI) (frequent fliers) or for associated medical renal disease. In such children care must be taken to make the visit to the operation theatre less traumatic. IV access may also be difficult in such children. Usual sedative premedicant drugs we use in children are Syr. triclofos sodium (Syr. Pedichloryl 50–100 mg/kg) or Syr. midazolam (0.5 mg/kg P.O.) or intranasal midazolam (0.1–0.2 mg/kg).

In infants, antenatal history, whether the infant is term or preterm and the nature and duration of neonatal intensive-care unit (NICU) stay are concerns. Prolonged NICU stay may mean that care must be taken to ensure there are no airway issues like subglottic stenosis. A preoperative chest X-ray may also be useful in such patients. A complete blood count is usually done. Anemia is an important contributing factor for postoperative apnea, especially in the preterm infant. Prothrombin time (PT), partial thromboplastin time (PTT) and international normalized ratio (INR) are done to look for any coagulation defects. PT can be prolonged in newborns. Injection vitamin K given (3 doses), helps in maturation of clotting factors. Renal function tests (RFT) give an indication of the compromise on renal function. Normal serum creatinine value in newborn is 0.3–0.5 mg/dL. Usually, a period of catheterization preoperatively helps reduce the serum creatinine (in obstructive uropathy) and stabilize electrolytes.

Children with end-stage renal disease (ESRD) coming for a peritoneal dialysis (PD) catheter insertion are usually on dialysis and are under the care of the nephrologists. It is ideal to consult the nephrologist regarding the timing of dialysis. Usually, the dialysis is planned about 12–24 hours before surgery. This way the child’s system equilibrates and the effects of the heparin, if used, have worn off. Besides a complete blood count, the RFT like serum urea, serum creatinine, electrolytes and platelet count are done after dialysis. Recommended daily allowance of fluid and the daily weight of the child help us to plan the IV requirement for this child. 5% dextrose solution is the common fluid of choice for maintenance in these children. Often anemia (Hb up to 7 grams) may be acceptable in ESRD.

Many of the children with ESRD may be hypertensive on multiple drugs. Vesicoureteral reflux (VUR) or a dysfunctional kidney may also be the cause for the hypertension. Often hypervolemia is an important cause of hypertension and can be treated preoperatively. A preoperative echocardiogram should be included as part of the evaluation to rule out a pericardial effusion and to look for cardiac dysfunction.5

As far as antihypertensives are concerned, our practice is to check blood pressure on the morning of surgery and decide on the need for medication. A discussion with the parent or caregiver will give us the normal values for this child since they would be checking the blood pressure on a regular basis. We usually avoid long acting drugs on the morning of surgery. Most of the time, following induction of anesthesia, there is a drop in blood pressure. This along with the regional anesthetic makes for a good control of the blood pressure during surgery. ACE inhibitors can cause profound hypotension intraoperatively and are best avoided.

Manipulations of the kidney or adrenal during surgery can cause significant fluctuations in blood pressure. As regional anesthetics (central neuraxial blocks) are commonly used for urologic procedures, a coagulation profile in the form of PT, PTT and platelet count is included in the preoperative workup.

Children on corticosteroid medication for congenital adrenal hyperplasia (CAH) or medical renal disease will need steroid supplementation IV during the period of surgery till they resume their oral medication. ‘Stress dose’ steroids are given as 5–10 times their usual dose of oral hydrocortisone for major procedures. For instance, if the child is getting 10 mg hydrocortisone, 50 mg IV hydrocortisone is given the night before, at induction and 8 hours after surgery. Subsequently, 3–5 times the usual dose of hydrocortisone can be continued for the next 2 days till the child is back on his oral medication. These patients (CAH) also require serum electrolytes to be done on the day of procedure to look for dyselectrolytemia.

Children with renal disease might have delayed gastric emptying time and need to be adequately fasted for elective procedures.

Either a central neuraxial block or a peripheral block is planned depending on our concerns regarding the coagulation parameters (ESRD). Understanding of
the sensory innervation of the genitourinary system is essential to this plan (Table 4).

**POSITIONING FOR UROLOGICAL PROCEDURES**

Many urological procedures are done with the child in the lithotomy position. Cystoscopic/ureteroscopic procedures require the legs be put up in lithotomy to enable access per urethra.

**Points to Remember while Positioning the Child in Lithotomy (Fig. 1)**

Lithotomy position involves abduction of the legs, flexion of the knees and support being given at the thigh. The patient is moved to the end of the table and the legs are put on leg holders. Depending on the size of the baby, the leg supports are customized. An infant will only require a folded towel while in the older child the leg holder holds the leg up at the popliteal fossa (knee crutch). Some leg supports provide support at the calf. Compression of the calves can lead to compartment syndrome, especially if the procedure takes long. This is characterized by severe pain, paresthesia, absence of pulses and pallor of the limb. Other stirrups holding the legs up by the heel will prevent this complication.

Besides causing a transient increase in venous return to the heart, lithotomy causes a decrease in lower leg perfusion and pressures. Often lithotomy is combined with a head down tilt. This further predisposes to venous thrombosis in the lower limb.

Putting the legs up compresses the abdominal viscera and causes limitation of respiratory excursion. This causes a decrease in tidal volume (VT) and vital capacity (VC). With a head down tilt, this can increase further. This may be relevant in newborns under anesthesia breathing spontaneously.

Among the various neuropathies that we encounter, sciatic nerve injury can occur with direct compression of the nerve or as part of compartment syndrome. Common peroneal nerve may be affected as it passes around the head of the fibula, if the legs are placed inside the stirrups or if the assistant is leaning on the leg. Saphenous nerve near the medial malleolus is prone to injury from pressure.

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**Table 4: Innervation of the genitourinary system and the regional blocks**

<table>
<thead>
<tr>
<th>Nerves</th>
<th>What do they innervate</th>
<th>What blocks can be used</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pelvic parasympathetic nerves via pelvic plexus (S2, 3, 4)</td>
<td>Innervate detrusor muscle and inhibit the internal sphincter of the bladder. Afferent fibers from the urinary bladder for pain and distension travel via these fibers</td>
<td>Blocked by CNB</td>
</tr>
<tr>
<td>Pelvic sympathetic nerves from the hypogastric plexus (presacral nerves)</td>
<td>Inhibit the detrusor muscle and they are motor to the internal sphincter of the bladder (responsible for painful reflex spasms)</td>
<td>Blocked by CNB</td>
</tr>
<tr>
<td>T10–L1</td>
<td>Kidney</td>
<td>CNB, paravertebral block</td>
</tr>
<tr>
<td>T11–L1</td>
<td>Ureter</td>
<td>Same as above</td>
</tr>
<tr>
<td>T10</td>
<td>Testes</td>
<td>Same as above</td>
</tr>
<tr>
<td>Dorsal nerve of the penis, a branch of the pudendal nerve (S2, 3, 4); dorsal part of root of the penis–ilioinguinal nerve (L1)</td>
<td>Penis</td>
<td>CNBs, Penile block</td>
</tr>
<tr>
<td>Anterior one-third of the scrotal skin is supplied by ilioinguinal (L1), posterior two-third of the skin is supplied by the perineal nerve (S2) and lateral by the posterior cutaneous nerve of the thigh (S3)</td>
<td>Scrotum</td>
<td>Ilioinguinal block plus LIA (local infiltration analgesia) CNB</td>
</tr>
</tbody>
</table>
All these areas have to be padded with cotton wool or the commercially available gel pads. When placing the leg on stirrups, the limbs have to be moved out symmetrically and simultaneously to prevent low back pain. Femoral neuropathy can occur from extreme abduction of the thighs with external rotation of the hip. Hip dislocation can occur with slippage of lithotomy poles.

**Lateral Position** \(^7\) (Fig. 2)

Once the child is in the lateral/semiprone position (usually for pyeloplasty), the head and neck are supported to keep them neutral in relation to the body. A roll is placed caudal to the axilla (under the upper chest) to prevent pressure on the neurovascular bundle in the axilla. The upper leg is flexed at the knee and hip while the lower leg is kept in the extended state. Pads are placed between the legs and the body is allowed to fall forward with gravity. The child is now fixed with straps/bandages or Velcro—one just below the axilla and the other above the iliac crest. Care should be taken to ensure the straps are not too tight to compromise respiration.

Once the child is in this position, the table is flexed (broken) at just above iliac crest to obtain the kidney position. In a smaller child or infant, a roll may be placed under the child above the iliac crest to widen the space between the iliac crest and lateral costal margin (Fig. 2). In this lateral or semiprone position, the arms are kept flexed and away from the field above the head, padded adequately and supported by arm rests.

Adequate downside padding can prevent injury to the ear, eye and facial nerve. Head and neck are supported adequately to be in the neutral position. The endotracheal tube can move towards the carina or can come out 0.9–1.7 cm with movements of the neck position. In the absence of an axilla roll, compression of the brachial plexus can occur if the lower shoulder and arm lie under the chest. \(^7\)

The common peroneal against the fibula and the lower sciatic nerve against the ischio pubic ramus, both are liable to injury due to compression if the child is not padded adequately.

**Trendelenburg Position** \(^7\)

Some of the laparoscopic urologic procedures require steep trendelenburg position, which includes a head down tilt of varying degrees. Though this causes an increase in cardiac output due to the increase in venous return, this effect is short lived (about 10 minutes). Respiration should be controlled in this position as work of breathing increases during spontaneous breathing and the reduction in lung volume predisposes to atelectasis. The use of head down position does not predispose to gastroesophageal regurgitation per se, but in patients with GERD (gastroesophageal reflux disease) the problem is compounded.

**ANESTHETIC MANAGEMENT FOR INDIVIDUAL PROCEDURES**

In general, as with most other invasive or surgical interventions in children, urologic procedures are done under general anesthesia. General anesthesia with controlled or spontaneous ventilation is used in most urology cases, along with a regional anesthetic. The exception is in preterms, where cystoscopy may be done “awake” with a caudal or spinal anesthetic to avoid the various problems associated with general anesthesia. \(^8\) Due to the short duration of action of the spinal, only brief procedures like vesicostomy or posterior urethral valve (PUV) fulguration may be done. In order to prolong the duration of the spinal, combined spinal and caudal analgesia may be given.

**Posterior Urethral Valves**

**Presentation/Diagnosis**

Posterior urethral valve disease is an obstruction to the free flow of urine caused by a membrane, which is obliquely placed in the posterior urethra. The membrane allows retrograde passage of a catheter, but the antegrade flow of urine is severely obstructed. The disease manifests in the antenatal period with bilateral hydrourereteronephrosis (HUN), absence of bladder cycling (filling and emptying) and in severe cases, reduced amniotic fluid volume. In its most severe manifestation, it is accompanied by pulmonary hypoplasia, azotemia and limb deformities. Neonatal ascites may be the presenting feature in some babies, where there has been rupture of a renal fornix into
the retroperitoneum (as a method of decompression) and this tracks forward as ascites.

Diagnosis is usually made by the lack of a good urinary stream in the newborn with a palpable bladder supplemented by the antenatal finding of HUN. Babies with progressive oligohydramnios may be considered for antenatal vesicoamniotic shunting (done as outpatient procedure in mother). Here a catheter is placed from the infant’s urinary bladder draining into the amniotic sac, in order to decompress the urinary bladder (foetal intervention). This is done percutaneously in the mother under ultrasound guidance. Unfortunately, this procedure, if done late, does not reverse renal damage.

Management
At birth, babies present with dribbling of urine and a palpable bladder. If facilities are available and the baby’s condition allows, cystoscopy and primary fulguration of the valve can be attempted. However, this is entirely dependent on surgeon’s expertise and the availability of appropriately sized equipment. If primary fulguration is not possible, a diversion procedure in the form of a vesicostomy may be needed.

Anesthetic management of a newborn baby coming for cystoscopy and fulguration of PUV includes general principles as for any newborn coming for surgery. In most centers, a period of initial catheterization is done to allow renal indices to stabilize. General anesthesia with controlled ventilation using an endotracheal tube/PLMA would be appropriate for this procedure.

Caudal blocks are very popular (0.25% bupivacaine) and provide up to 6–8 hours of analgesia. 0.2% ropivacaine is used off-label and has a better safety profile. Alternately, spinal or combined spinal and caudal as described earlier may be used. Spinal anesthesia is given, especially in preterms. In former premature infants, Wellborn et al. showed a reduction in apnea from 36% to 0% with use of spinal.

Dose of bupivacaine 0.5% (heavy) for spinal anesthesia is 0.1 mL/kg up to 5 kg. Antibiotic prophylaxis is important for any cystoscopic procedure. Since cold fluids may be used for irrigation, it is important to keep the infant warm and to monitor temperature. Warming the irrigation solution is ideal. The procedure usually lasts 30–60 minutes. Bladder perforation can occur while instrumenting the bladder. Care should be taken not to allow the child to cough or strain while the scope is in situ. Postoperatively these babies may go through a period of post obstructive diuresis, where they put out a lot of urine. So fluid replacement has to be adjusted in consultation with the nephrologist. Analgesia is usually with paracetamol suppositories at 20 mg/kg/dose 8 hourly (PR), 10 mg/kg/dose 6 hourly per oral.

The dose of IV paracetamol is controversial in neonates and infants. The BNF for Children (BNFC) suggests a dose of 7.5 mg/kg every 8 hours (maximum 25 mg/kg daily) in preterm neonates over 32 weeks postmenstrual age, 10 mg/kg every 4–6 hours (maximum 30 mg/kg daily) in neonates.11

IV paracetamol in infants <10 kg is 7.5 mg/kg, maximum daily dose of 30 mg/kg.

Cystoscopy for other Pathology: Anesthetic Management
In older children, cystoscopy for diagnostic purposes can be done, holding a mask or inserting an LMA/PLMA/Igel and maintaining the child on inhalational/IV agents, breathing spontaneously. In the female, the urethra is short and the stimulus for insertion of the cystoscope is minimal. Just a combination of ketamine 1 mg/kg and propofol 2–3 mg/kg or midazolam/fentanyl would be sufficient, while maintaining the airway. Dexmedetomidine is now gaining popularity in combination with ketamine for these procedures. Male children might need caudal analgesia, especially if the scope needs to be introduced a few times. Caudal analgesia is preferred, especially if any interventional procedure is to be done cystoscopically like fulguration. Antibiotic prophylaxis is given at induction. Postoperative pain relief is with paracetamol IV at 15 mg/kg or PO at 15 mg/kg on discharge.

Exstrophy Epispadias Complex (Fig. 3)
Presentation, Diagnosis
The diagnosis of exstrophy epispadias complex (EEC) is invariably done after the baby is born when a reddish fleshy mass is seen below the umbilicus through which
free egress of urine is seen. The anterior abdominal wall including the anterior wall of the bladder and the dorsal wall of the urethra are absent.

**Surgery/Anesthetic Management**

In most centers an attempt will be made to obtain bladder closure, leaving the penile defect for later surgery. The surgery involves extensive dissection of bladder and bladder neck, involving considerable blood loss. Centers with adequate expertise may attempt a single stage complete repair of bladder and penis which is called complete primary repair of exstrophy. In cases in which there is severe pubic diastasis, an innominate osteotomy or a pubic ramotomy may be done, further adding to the morbidity of the procedure. If an iliac osteotomy is done, the child is initially placed in the prone position and subsequently turned supine. The child might come back for multiple procedures.

Anesthetic management in these infants would include invasive monitoring in the form of an arterial line, a central venous line (in some patients) and two good peripheral lines anticipating blood transfusions. An epidural catheter is inserted before start of surgery, and fixed away from the osteotomy site posteriorly and covered with waterproof plastic dressing like tegaderm, over which the surgeon can paint if necessary. Care must be taken to replace third space and evaporative losses. Since the urine output cannot be determined and blood loss is also difficult to assess, these babies pose quite a challenge. Systolic blood pressure is a good indicator of volume in infants in the absence of central venous pressure (CVP) measurement. In the postoperative period, watch for ongoing blood loss, anemia and hypotension.

Epinephrine infusion of bupivacaine 0.125% at a rate of 0.1–0.2 mL/kg/hour may be continued for 24–48 hours to cover pain. Alternatively, ropivacaine 0.2% may be given and has a very good safety profile when given as infusion over many days. IV infusion of fentanyl at 0.5–1 μg/kg/hour would keep the baby quiet and improve outcome of repair. If the hip spica is planned following osteotomy in the infant, special precautions must be taken to bring the epidural catheter out, so it can be removed either by traction from above the spica or a window is cut in the spica plaster at the site where the catheter is fixed to facilitate easy removal, 3–5 days later.

Postoperative period may be long drawn out and often IV access may become an issue. In such patients, it may be worthwhile inserting a central venous line initially at surgery. This can be maintained for the entire period of hospitalization. Ketorolac aids in relieving the child of bladder spasms, which can cause a lot of pain postoperatively.12

**Pyeloplasty**

**Presentation/Diagnosis**

Pyeloplasty is a procedure done to relieve pelviureteric junction obstruction, which is often diagnosed antenatally by ultrasound. Our patients are usually 1.5–3 months of age at the time of surgery unless diagnosed late. The obstruction produces backpressure effects on the kidney and compromises renal function in the long-term. Most patients have normal renal parameters at surgery. The pathology is usually unilateral, but can be bilateral. Usually, one side is operated upon at any one time.

**Surgical Aspects/Anesthetic Management**

The classic open procedure involves a subcostal or lumbotomy incision a transperitoneal approach where the child is supine or a retroperitoneal approach (lateral position). General endotracheal anesthesia with controlled ventilation and caudal analgesia is adequate. Alternately, an USG-guided TAP block may be used. Postoperative analgesia can be maintained with a combination of IV/PO paracetamol and opioids. Alternatively, a combination of oral paracetamol and triclofos sodium (50 mg/kg) bd for sedation in an option. These patients might have associated anomalies like cardiac or spinal cord defects (spina bifida), which have to be identified preoperatively and appropriate precautions taken. The laparoscopic technique involves small incisions and is less painful. Robot assisted pyeloplasty is becoming popular and the results are very promising.

**Vesicoureteric Reflux**

**Diagnosis/Surgical Treatment**

Vesicoureteric reflux (VUR) can be suspected by noting renal pelvic dilatation or ureteric dilatation and peristalsis antenatally. Management of VUR depends on grade, presence of renal scarring and breakthrough infections inspite of antibiotic prophylaxis. Definitive surgical treatment of VUR (ureteric reimplantation) is generally advised only after toilet training in a child. However, the advent of subureteric injection of dextranomer (Deflux®) has made it possible to treat VUR cystoscopically as a day care procedure at any age.

**Anesthetic Management**

Bilateral/unilateral ureteric reimplantation is usually a procedure lasting about 60–120 minutes. Patients with renal scarring may be hypertensive and on medication. Hospitalization for recurrent UTIs makes
it difficult to get IV access. It is advisable to type and crossmatch one unit of blood. Lumbar epidural with catheter in situ helps reduce intraoperative anesthetic requirements and can be continued for postoperative pain relief. Injection tranexamic acid 15 mg/kg given at start of procedure helps reduce blood loss. Clonidine as an adjuvant to the epidural provides for a bloodless field, prolongs analgesia, reduces intraoperative anesthetic requirements and ensures the child wakes up calm and quiet. Patients usually have at least one perurethral catheter and a suprapubic catheter after surgery. Postoperatively besides the epidural infusion, oxybutynin hydrochloride in a dose 0.2 mg/kg/dose is usually given twice daily. Tab. diazepam PO at 0.2 mg/kg and ketorolac are all useful to help prevent and control bladder spasms. Bladder spasms are most common after 24 hours and is a difficult problem to treat. Epidural catheter may be removed after 2–3 days.

Hypospadias (Figs 4 and 5)

Surgical Aspects

Hypospadias is a common congenital condition in which male babies are found to pass urine from an abnormal opening on the undersurface of the penis. There may be an associated ventral bending of the penis called chordee. The position of the orifice can range anywhere from the perineum to just below the tip of the penis. If associated with nondescent of the testis, an intersex state has to be suspected. Repair of the condition is usually done after 6 months of age. Repair may be attempted in a single stage, although a staged repair may be required in severe cases. In those children who have had multiple failed procedures with complete absence of prepucial skin, substitution procedures may be needed.

Anesthetic Management

General anesthesia with spontaneous ventilation using a PLMA/igel should be adequate for most hypospadias surgery as long as a good regional block is in place. Penile block can be used for distal and coronal hypospadias (Fig. 4). The more proximal hypospadias warrant a caudal block with ropivacaine 0.2% and clonidine 1–2 μg/kg. Clonidine prolongs duration of the caudal block and keeps the child quiet in the immediate postoperative period due to its sedative properties.

In the severe cases and in perineal hypospadias, we usually insert a lumbar epidural catheter for intraoperative and postoperative pain relief. Perineal hypospadias repair usually takes much longer, about 2–3 hours, and we can expect significant blood loss. Where a buccal mucosal graft procedure is planned, nasotracheal intubation with a throat pack is needed. The graft is removed from the inside of the lower lip or cheek. These children will usually be nil per oral up to 12 hours after the procedure.

Following hypospadias surgery, children may return for dressing change or repeat procedures. Sudden dislodgement of per urethral catheter or blockage of catheter with retention of urine is an emergency and requires urgent suprapubic cystostomy under GA.

Cryptorchidism

Surgical Aspects

Children with testicular maldescent are generally taken up for surgery after 6 months of age. The surgery called orchidopexy involves a herniotomy, creating a space in the scrotum and fixing the testis securely. This can be done as a daycare procedure in children with a palpable testis. In those where the testis is not palpable, laparoscopy is
done to assess position, size and viability. When the testis is far away from the internal inguinal ring, a two-stage laparoscopic procedure may be resorted to.

**Anesthetic Management**

General anesthesia with a caudal block with the child breathing spontaneously via an LMA/Igel is adequate for a child with a palpable testis. Laparoscopic procedures need controlled ventilation with a PLMA or an endotracheal tube. Bilateral rectus sheath block with TAP block or TAP block alone on the side of procedure is an alternative to caudal analgesia. Ilioinguinal nerve block combined with local infiltration analgesia for scrotal skin incision is another option.

Postoperative nausea and vomiting is common following orchidopexy. Injection ondansetron 0.1 mg/kg IV and injection dexamethasone 0.1 mg/kg IV given during the procedure will take care of this. Children undergoing orchidopexy are now treated as day care in many centers so it is worthwhile to check if the child is staying overnight before we give long acting additives like clonidine which may delay discharge.

**Torsion Testis**

Testicular torsion occurs when the testis twists around the spermatic cord, cutting off its blood supply.

**Signs and Symptoms**

Severe pain and swelling on one side of the scrotum. There is also often redness/tenderness of the scrotal skin. Child may also have nausea and vomiting. If the blood supply is not restored within 6 hours the testis will undergo necrotic changes.

**Anesthetic Management**

Patient may be full stomach so precautions need to be taken at induction accordingly. General anesthesia with controlled ventilation with caudal analgesia would be a suitable anesthesia plan. If the child is fasted, a PLMA/Igel with spontaneous ventilation and caudal analgesia can be used. A good antiemetic needs to be included. The opposite testis is usually fixed. If the testis is not salvageable, orchidectomy may be done. Appropriate consent should be obtained at the beginning of the procedure.

**Intersex Disorders**

Procedures for intersex may be diagnostic or therapeutic. Diagnostic procedures are minimally invasive and include assessment of the pelvic organs and biopsy of the indeterminate/dysgenetic gonads. Often this is done laparoscopically in the first few months of life.

**Cloaca**

Cloaca is a condition where there is a single opening in the perineum which leads into the urethra, vagina and the rectum. The first surgery is right transverse colostomy done in the newborn period in order to divert the fecal stream. Rarely, accumulation of secretions in the vagina may necessitate vaginal drainage. Subsequent total repair of cloaca is done between 9 months and 1 year of age. This is a major procedure lasting several hours.

**Wilms’ Tumor**

Wilms’ tumor or nephroblastoma is the commonest renal neoplasm affecting children. Most children (90%) present before the age of 8 years (mean age at presentation 3.5 years). Clinical staging and histology determines the treatment plan, which is multimodal. Anesthetists are involved in the care of these patients during sedation for MRI or CT contrast studies, which are vital in looking for metastasis in liver and lung, for staging. We are also involved in the primary or delayed excision of the tumor and for central venous access or port insertion for chemotherapy.

The decision to resect surgically is based on renal size, friability (risk of rupture), vascular invasion and pulmonary metastasis. Large friable tumors, those with vascular thrombi, bilateral tumors and the presence of liver and pulmonary metastasis generally warrant preoperative chemotherapy.

Anesthetic concerns are those of major surgery in small infants—potential for major blood loss, fluid management and temperature maintenance. An epidural catheter placed early helps provide analgesia along with reducing the blood pressure fluctuations that may occur. If the patient has been on chemotherapy, a complete blood count is necessary. Some of the chemotherapy drugs like Adriamycin may have side effects (cardiac), which need to be assessed. A preoperative ECHO is a must. The Echo also reveals the extent of thrombus, if any. 50% of patients are hypertensive and require control of blood pressure before surgery. An arterial line and CVP monitoring are essential in large tumors. For smaller tumors without intravascular extension, 2 large peripheral lines (upper limb preferred) and non invasive blood pressure measurement may suffice. The external jugular vein is a good access in
all these patients. A large supraumbilical transverse, bilateral rectus cutting incision is made to minimize renal handling. Vessels are preferably brought under control in the beginning and the tumor is excised along with the adrenal without spillage. Tumor neovasculature and the need to mobilize the aorta and IVC add an element of risk to the operation. Extensive node sampling is done as part of the surgical procedure.

When there is a tumor thrombus in the renal vein extending into the infrahepatic IVC, complete control of IVC is obtained by extensive dissection and mobilization. Prior to cross clamping the IVC, we need to preload the patient to make sure there is no fall in CVP from the decrease in venous return. Once cavotomy is done and the tumor thrombus removed, deairation is carefully done to avoid air embolism. Upon release of cross clamp we need to watch for the surge in CVP. In the event of extensive tumor thrombus in the IVC, the patient might have to go on cardiopulmonary bypass for its removal.

Nephrectomy may also be done for other indications like a dysplastic nonfunctioning kidney causing repeated infections. The concerns and management are similar.

Laparoscopy and Robotic Surgery

Laparoscopy is routinely used for urologic procedures as it credited with improved patient outcomes, early mobilization and discharge. It is used for identification and bringing down of an impalpable undescended testis, in intersex for diagnosis and biopsy of the internal structures and for nephrectomy or nephroureterectomy. Pyeloplasty can also be done laparoscopically, retroperitoneally or transperitoneally. With low inflation pressures of CO₂, 8–10 cm in newborn and up to 15 cm in older child, the physiological changes are minimal. All children require endotracheal intubation with controlled ventilation for both laparoscopy and robotic surgery, though we do use Proseal LMA with controlled ventilation successfully for short laparoscopy procedures in healthy fasted children. Regional blocks in the form of rectus sheath block, TAP block or caudal block have been found to have a significant advantage in these types of surgeries.

Robotic surgery in children has been found to have improved outcomes compared to open surgery.

The da Vinci robotic interface enhanced the laparoscopic surgical experience by introducing a series of revolutionary changes:

- Three-dimensional view of the organs in ‘real color’
- Complete control of the camera by the surgeon who can zoom in or out or pan around as he sees fit
- Ten times magnification, which allows precise and delicate surgery on tissues without undue disturbance of neighboring structures
- Instruments which bend, twist and turn like the human wrist, enabling suturing to be as precise as in open surgery.

Today many major urologic procedures are being done robotically in children as well. The surgeries are more aptly called robot-assisted laparoscopic procedures as pneumoperitoneum is used as in laparoscopy, the ports are inserted, the robotic arms attached to the ports and the surgeon sits at a console (computer) to direct the robotic arms.

The large arms entering the small child through ports, lack of access to patient once the arms are docked and relatively long duration of procedure (in early stages of learning), often with steep trendelenberg position, are some of the challenges faced. These are compensated for by excellent patient recovery due to minimal tissue handling, and hence, minimal analgesic requirement, early feeding and discharge.

Urolithiasis

In endoscopic procedures for stone removal, anesthesia is similar to cystoscopic procedures. They may, however, be longer and require controlled ventilation via endotracheal tube or SGA. Imaging is often a part of the procedure. Children can become hypothermic from prolonged procedure and need warming devices. Depending on the stone size and impaction, the children have varying degrees of pain. Better analgesia, intraoperatively and postoperatively, will help reduce the incidence of nausea and vomiting. Antiemetics are given intraoperatively.

RENAL TRANSPLANTATION IN CHILDREN

Epidemiology

ESRD is less common in children than adults. Obstructive uropathy, hypoplastic kidneys, focal segmental glomerulosclerosis, reflux nephropathy and polycystic kidney disease are common causes of ESRD in children. Many of the diseases causing chronic renal failure (CRF) do not recur, which means in children successful transplantation gives a permanent cure.

Both hemodialysis (HD) and peritoneal dialysis (PD) are used as renal replacement therapy in children with ESRD. Hemodialysis is more challenging in younger children due to issues with vascular access and smaller circulating volume.
The chances of success with transplant are greater in living related transplants in 2–5 year olds. Some factors have been associated with a poor outcome—multiple transfusions preoperatively, pretransplantation dialysis and retransplantation. Laparoscopic donor nephrectomy has higher rate of delayed graft function and 6 month rejection. Pediatric deceased donors (<5 years) are not preferred for pediatric recipients due to higher rates of graft thrombosis and technical failures.

Pathophysiology of Renal Failure

Kidney plays an important part in regulation of acid base. It is also responsible for hormone production and metabolism. With the onset of renal failure, growth and development are affected. Metabolic acidosis, hyperkalemia, hyperphosphatemia, hypocalcemia are some of the derangements seen in children with ESRD. Many are on long-term renal replacement therapy in the form of bicarbonate, calcium and vitamin D supplements. Often they are on treatment for hypertension, this being the result of increased renin levels. Other problems in these patients include altered platelet function and anemia as a result of decreased erythropoietin levels. Volume overload leading to congestive heart failure can occur. Seizures can occur as a result of electrolyte imbalance and many children are on antiepileptic drugs.

Indications for Renal Transplantation\textsuperscript{19}

- ESRD not responsive to medical management.
- Failure to thrive in spite of optimal medical management.
- Patients with developmental delay.
- Children with progressive renal osteodystrophy in spite of optimal renal replacement therapy.

Contraindications for Renal Transplantation

Contraindications for renal transplantation include active infection or malignancy, patients at high risk of damage to transplanted kidney from primary disease, patients who have poor reserve to tolerate the anesthetic and those with poor life expectancy. Patients with malignancy, have to be in remission for 2–5 years. Hepatitis B/C positivity is not an absolute contraindication.\textsuperscript{19}

Optimal age for renal transplantation: \textsuperscript{20,21}

In infants and children less than 2 years it is technically difficult, with higher risk of arterial thrombosis and graft failure. Usually, transplant is deferred till the child is >2 years of age or the weight >15–20 kg. Graft loss is seen more in patients who received deceased donor transplant as against living related donor transplant. Recent studies show that a kidney from a deceased pediatric donor could be transplanted into a child and the kidney will grow with the patient.

Surgical Technique

Renal transplantation is done with a retroperitoneal approach in children >20 kg. The renal artery is anastomosed to the common iliac or hypogastric artery, renal vein to common iliac or external iliac vein. The ureter is then anastomosed to the bladder. Often these children would have undergone other procedures like augmentation cystoplasty (where the urinary bladder capacity is increased by attaching a loop of bowel or ureter to its dome). If they have urosepsis from a dysplastic kidney or vesicoureteric reflux, a nephrectomy is done pretransplant to prevent infection in the transplanted kidney.

If an adult kidney is transplanted into a small child, the vessels are anastomosed to the aorta or IVC. A retroperitoneal approach may not always be possible, as it might compress the adult graft (Becket et al. 2006). Therefore, transperitoneal approach may be used. Due to pooling of a large amount of blood in the adult organ, hemodynamic changes in the infant on reperfusion can be profound.

Anesthesia Concerns

In the preanesthetic evaluation (as already detailed earlier in this chapter), the effects of renal failure on other organs have to be evaluated. Babies with congenital aplasia of the kidney may have associated cardiac defects. The VACTERL association is well known. Here, renal agenesis or aplasia maybe associated with VSD, ASD, PDA. Anemia, hypertension and electrolyte abnormalities are common. Children with ESRD often have reactive airways and care must be taken to avoid airway irritation during intubation and extubation. Congestive heart failure from volume overload, uremia is treated with dialysis. Children with ESRD often are on some form of dialysis, initially peritoneal, then hemodialysis. Dialysis removes excess fluid and brings electrolytes to near normal values. A hemodialysis catheter may already be in situ and can be used intraoperatively for vascular access/CVP measurement. This has to be safeguarded for future use in the immediate post operative period. Arterial/venous cannulation may be difficult in some edematous children, who are on long-term steroids for nephrotic syndrome.

Anesthetic Technique

Preanesthetic medication is usually in the form of midazolam, IV or PO. Antihypertensive drugs are continued on the day of the surgery after recording the...
BP on the morning of the surgery. A slightly higher BP for age is preferred, so in consultation with the nephrologist the antihypertensive medication may be adjusted keeping in mind that the BP is likely to fall at induction and with reperfusion.

IV induction is with propofol or thiopentone. Atracurium is a suitable muscle relaxant in these children. The duration of action of vecuronium or rocuronium may be prolonged due to partial renal excretion. Maintenance of anesthesia can be with isoflurane or desflurane. Sevoflurane is avoided due to concerns about nephrotoxicity in the transplanted organ. Air oxygen mixture is preferred as use of nitrous oxide can cause bowel distension and problems with closure in a child, especially with a large transplanted kidney in place. Fentanyl is used along with IV paracetamol for analgesia.

Besides standard monitoring in the form of ECG, SpO$_2$, temperature, NIBP, CVP and arterial monitoring are very useful. If the child has a Hickmann catheter for dialysis already in place, this may be used for CVP measurement and to give immunosuppressant.

Preloading is done with crystalloid/colloid to increase the CVP (up to 12–14 cm water in older child and up to 18 cm water in infant/smaller child) so that with release of cross clamp the hypotension is not significant. Preloading can be with 5% albumin, FFP along with crystalloids as the situation demands. Also, systolic BP is kept at about 20% higher than preoperative values. An infusion of dopamine at 5 μg/kg/min may be required to keep the blood pressure up if preloading alone is insufficient. Sometimes, surprisingly large volumes of fluid need to be transfused to bring up the blood pressure after reperfusion (Beebe et al. 1991).

Temperature maintenance is of paramount importance. This is done by warming fluids and using forced air warming devices. Since, both aorta and IVC are cross clamped in small children it is not advisable to warm the lower extremities during the period of cross clamp as it can increase the quantity of ischemic metabolites that enter the circulation upon release. In the immediate period following cross clamp release atropine 20 μg/kg and calcium chloride 10 mg/kg are given to prevent vagal response to hypotension and to counter potassium excess which enters the circulation on reperfusion. The preservative solution used to perfuse the kidney has high potassium content. This is usually washed out before anastomosis. Sodium bicarbonate 1 mmol/kg is also given to combat acidosis. Urine output is replaced with 0.45 NS for up to 2 days postoperatively.

A low flow state can promote thrombosis of the allograft. Mannitol 20% at 0.5 g/kg and Furosemide 1–2 mg/kg IV are given at surgery to induce osmotic diuresis. In living related transplants the allograft kidney starts making urine almost immediately. Insensible losses are replaced with 5% dextrose while urine is replaced with 0.45 NS. CVP is maintained at 5–10 cm water.

### Postoperative Complications

Although pulmonary edema from excess administration of fluid is seen in many patients, very few need mechanical ventilation. Generally in children, living related transplants do better than cadaver recipients. Several immediate, acute and long-term problems can occur. Primary nonfunction of allograft can occur as a result of hyperacute rejection (within few minutes to an hour, due to antibodies to HLA and ABO antigens). Renal artery thrombosis is common in children receiving adult kidneys and acute tubular necrosis. Urinary obstruction due to blood clots may mimic this. Other complications include hypertension, anemia, erythrocytosis as a result of the various drugs. Delayed acute rejection occurs after 6–12 months usually due to noncompliance with medication.

### CONCLUSION

Patients coming for urologic surgery form a wide spectrum from the normal infant to the sick child awaiting transplant. Preoperative preparation in the form of blood pressure control or correction of acid base and electrolyte imbalance goes a long way in improving outcome in these patients. Intraoperatively most children get a general anesthetic with regional analgesia, taking into account the coagulation profile. Clonidine is a common adjunct used with the caudal local anesthetic when patients are going to stay in. Many patients are done as day case procedures. Many major procedures require continuous catheter techniques wherever possible. Renal transplantation in children enjoys better success with the live donor.

### ACKNOWLEDGMENTS

I thank Dr V Sripathi, MS, MCh, FRACS (Pediatric Surgeon) for his valuable inputs in the preparation of this chapter. I also thank Dr VK Sairam, MD, AB (Pediatric Nephrologist), Dr S Ramesh, MD, DA (Pediatric Anesthetist) and Dr Narendra Tajne, MBBS, DNB (Pediatric Anesthetist) for proofreading the manuscript.
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Anesthesia for Pediatric Laparoscopic Surgeries

INTRODUCTION
Minimally invasive surgery has been widely adopted for the management of various surgical conditions in all age groups. Advances in instrumentation and techniques have led to a remarkable development in the field of laparoscopy involving almost any organ, and have become standard procedures in pediatric population. Initially laparoscopy was confined to short, diagnostic procedures which were conducted on young healthy children. Now neonates and high risk pediatric population who were earlier considered unfit for laparotomy are being accepted for laparoscopy due to its documented benefits. The main advantages of laparoscopic surgery for patients are less postoperative pain, reduced wound complications, minimal scarring, a shorter hospital stay, and an earlier return to normal activities including feeding, bowel movements, and work/school.1

Laparoscopic surgery has inherent limitations for the surgeon. These include a two dimensional visual image, a loss of touch sensation, difficulty in controlling bleeding (limited suction, no manual pressure), limitation in the number and directions of instruments and difficulty in suturing. Introduction of “Robotic laparoscopic surgery” in pediatric patients will eliminate few of the above mentioned problems, however new skills have to be acquired and these can translate into longer operating times.

Although visually minimally invasive to the patient, laparoscopic surgery produces significant pathophysiological changes. These changes require meticulous observation and possible alterations in the standard anesthetic techniques.2

The anesthetic management in these surgeries poses special challenges due to the creation of carbon dioxide pneumoperitoneum and the need for the extreme degrees of positioning. Understanding this physiology is particularly important in the planning and conduct of anesthesia and to prevent a turbulent perioperative course.

INDICATIONS FOR LAPAROSCOPIC PROCEDURES

Diagnostic
Non-palpable testes, liver biopsy, direct cholangiography, disorders of sexual differentiation, recurrent pain in abdomen, lower GI bleeding, blunt/sharp abdominal trauma, seromuscular bowel biopsy.

Therapeutic
• Orchidopexy for intra-abdominal testes
• Cholecystectomy
• Choledochal cyst excision
• Antireflux surgery—fundoplication
• Resection of benign ovarian tumor
• Pyloromyotomy
• Gonadectomy for dysgenic gonads
• Small bowel resection
• Laparoscopic assisted pull through for Hirschsprung’s disease
• Anorectal malformations
• Pyeloplasty
• Nephrectomy for non-functioning kidney
• Partial nephrectomy for duplex system
• Ureteric reimplantation
• Laparoscopic-assisted bladder reconstructive surgery
• Adrenalectomy
• Excision of pheochromocytoma
• Varicocelectomy
• Appendectomy
• Meckel’s diverticulum excision
• Ventriculoperitoneal (VP) shunt placement
• Uncomplicated liver cysts
• Splenectomy.

CONTRAINDICATIONS

Absolute contraindication is patient refusal. VP shunt once thought as relative contraindication is no longer considered so, because of availability of pressure regulator shunts. Children who have an unstable hemodynamic status are not suitable for laparoscopic procedures, which involve prolonged operating times. Laparoscopy should be avoided in patients with severe cardiac diseases, pulmonary insufficiency or uncorrected bleeding disorders. Multiple previous abdominal surgeries can be also a relative contraindication purely from a surgeon’s perspective.

ANESTHETIC CHALLENGES

The anesthetic challenges are discussed briefly here:
• Physiological effects of carbon dioxide (CO₂) pneumoperitoneum and raised intra-abdominal pressure
• Effects of patient positioning
• Risk of heat loss and hypothermia
• Early recognition and management of intraoperative complications.

Physiological Effects of CO₂ Pneumoperitoneum and Raised Intra-abdominal Pressure (IAP)

Pneumoperitoneum creation involves the intraperitoneal insufflation of CO₂ through a Veress needle, or commonly in children by open incision and trocar insertion.

CO₂ almost fulfills the criteria of ideal gas for laparoscopy. CO₂ has remained the insufflation gas of choice because of its ready availability, noncombustible properties and a high Ostwald’s blood/gas partition coefficient (0.48). Due to its high solubility, the incidence of gas embolism is rare (0.0016–0.013%). There are concerns about other gases which could be used for insufflation. Nitrous oxide may cause explosion, while helium is linked with higher risk of gas embolism and subcutaneous emphysema and argon may cause cardiac depression.

The insufflation of gas to create pneumoperitoneum causes increase in IAP which is the major determinant of cardiovascular and respiratory changes during laparoscopy. The magnitude of these changes is influenced by the patient’s age, underlying cardiorespiratory status and the anesthetic agents used.

Effects on Cardiovascular System

Raised IAP will cause changes in the preload, systemic vascular resistance (SVR) and the myocardial contractility. If IAP is <10 mm Hg, there is milking effect on the veins leading to increase in the venous return and cardiac output. If IAP is >12 mm Hg, there is 10–30% reduction in the cardiac output due to compression of IVC and decreased venous return. Raised IAP will cause compression of abdominal aorta increasing after load and SVR. An increase in blood pressure and tachycardia is often seen resulting from an increase in systemic vascular resistance and increased PaCO₂ due to absorption of insufflated CO₂. Reflex increase in vagal tone due to excessive stretching of the peritoneum may produce bradycardia.

Effects on Respiratory System

Pulmonary effects include cephalad shift of diaphragm and reduced diaphragmatic excursions, reduced thoracic compliance and decreased functional residual capacity (FRC), leading to early closure of the smaller airways and increased peak inspiratory pressures. This increased airway pressure can lead to barotrauma during intermittent positive pressure ventilation (IPPV). In neonates and infants, the reduction in FRC may be significant. Coupled with the high closing capacity and oxygen consumption, reduced FRC makes neonates and small infants prone to develop hypoxemia following increase in intra-abdominal pressure. The short trachea in children increases the risk of endobronchial intubation. The tip of the tracheal tube has been observed to have a tendency to be displaced caudally with the cranial displacement of lung tissue and carina during the establishment of pneumoperitoneum. This risk is greater when the Trendelenburg tilt is employed to facilitate laparoscopy of the lower abdomen. These changes get compounded by frequent alterations
in position during surgery. All these factors will increase ventilation perfusion (V/Q) mismatch.\textsuperscript{10-14}

Significant intravascular absorption of insufflating gas i.e. CO\textsubscript{2} can occur in children due to the smaller distance between the capillaries and the peritoneum and the large peritoneal surface area. PaCO\textsubscript{2} increase is generally within tolerable limits in most children. Most patients need ventilatory interventions i.e. increase in minute volume to restore acceptable end tidal CO\textsubscript{2}.

As the cardiorespiratory changes produced by raised IAP are seen at lower values in children, it has been recommended that the IAP should be limited to 5–8 mm Hg in neonates, about 10–12 mm Hg in infants, less than 15 mm Hg in older children with the gas flow of about 0.9–1 L/min to maximum of 5 L/min. The effects of general anesthetics and intravascular volume on hemodynamic function during creation of pneumoperitoneum also augment the above perturbations.

**Effects on Renal System**

The renal blood flow and glomerular filtration rate decrease because of an increase in renal vascular resistance, reduced glomerular filtration gradient and reduced cardiac output, leading to decrease in urine output. An IAP of >20 mmHg reduces the renal and mesenteric blood flow significantly. There is an overall reduction of blood supply to all the intra-abdominal organs except the adrenal glands. Intraoperative anuria was noted in 88% of infants undergoing laparoscopy, and oliguria in 32% above 1 year of age.\textsuperscript{15} This was found to be completely reversible within 5 to 6 hours after surgery with no significant associated reduction in renal blood flow or rise in serum creatinine and urea levels. For this reason, intraoperative urine output in children undergoing laparoscopic procedures is not a reliable indicator of intravascular volume or need for intravenous therapy until 5 hours after surgery.

**Neurological Effects**

Hypercarbia, Trendelenburg positioning, and a decrease in CSF absorption due to the high IAP are all responsible for the increase in cerebral blood flow and intracranial pressure noted during laparoscopy.\textsuperscript{16} Laparoscopy may be relatively contraindicated in patients with poor intracranial compliance.

**Effects on Gastrointestinal System**

Patients undergoing laparoscopy are usually considered as high risk for acid aspiration syndrome because of gastric regurgitation, which might occur due to the rise in intra gastric pressure consequent to the increased IAP. However, during pneumoperitoneum, the lower esophageal sphincter tone far exceeds the intragastric pressure and thus the raised barrier pressure limits the incidence of regurgitation (normal barrier pressure: 30 cm H\textsubscript{2}O).

**PATIENT POSITIONING**

Patient positioning can cause cardiorespiratory compromise, and the severity depends on the degree of the tilt and the IAP. Trendelenburg position will increase venous return and cardiac output whereas with reverse Trendelenburg position both will decrease. Trendelenburg position causes decrease in the lung compliance by reducing the FRC and reverse Trendelenburg position will improve the respiratory compliance.

**HYPOTHERMIA**

Children are at risk of heat loss and hypothermia during laparoscopic surgery especially during prolonged surgeries. It is mainly due to the insufflation of cold CO\textsubscript{2} gas. The fall in temperature is approximately 0.3°C/50 mL CO\textsubscript{2}. This can be minimized by using warming mattress, warm intravenous fluids and the use of pre-warmed CO\textsubscript{2} for insufflation.

Laparoscopy elicits a classic stress response elucidated by a rise in secretion of ACTH, cortisol, insulin and glucagon. DVT prophylaxis is recommended for patients undergoing laparoscopic procedures of long duration in the reverse Trendelenburg position. The pressure points should be padded with great care to prevent nerve compression injuries.

**ANESTHETIC MANAGEMENT**

The pre-operative assessment and pre-operative preparation are same as open surgery. The standard fasting protocols should be followed in elective cases. In case of emergency, precautions should be taken to prevent aspiration of gastric contents. Parents should be informed about the indication for surgery, as well as other treatment options, risks and complications. The possible risk of conversion to an open procedure needs to be discussed. Blood loss is usually minimal in laparoscopic surgery, but hemorrhage is a possibility and a blood group and cross match should be available for major procedures.

**Premedication and Induction**

A number of drugs have been used as premedicants; the author prefers any of the following:
• Oral midazolam syrup (0.5 mg/kg) 20 minutes before the surgery
• Oral clonidine (4–5 μg/kg), 45–60 minutes before the surgery
• Intranasal dexmedetomidine (1–2 μg/kg), 30–45 minutes before the surgery.

However, each institution has a protocol which can be used accordingly. Atropine or glycopyrrolate may be included in the premedication in order to prevent the reflex bradycardia that can occur on abdominal insufflation, in addition to their effect of decreasing airway secretions.

General anesthesia with muscle relaxant and controlled ventilation is the preferred technique. The choice of the induction agent and technique is based on the clinical status of the patient and the preference of the anesthesiologist. Induction of anesthesia can be performed either by inhalational technique with sevoflurane or halothane, or with one of the available intravenous induction agents. This is followed by a nondepolarizing neuromuscular blocking agent, such as atracurium or vecuronium, to facilitate endotracheal intubation.

**Maintenance**

Anesthesia is maintained with controlled ventilation with an inhalational agent such as isoflurane or sevoflurane supplemented with an intermediate acting nondepolarizing neuromuscular blocking agent and intravenous opioids as required. Halothane should be avoided for maintenance, where possible, because the hypercarbia and the subsequent sympathetic stimulation that occurs due to CO2 absorption from the peritoneal surface can cause arrhythmias when combined with halothane. It is common to avoid the use of nitrous oxide since this may distend bowel, particularly during prolonged procedures.

**Management of the Airway**

Although, the laryngeal mask airway has been used safely for short laparoscopic procedures, endotracheal intubation is preferred for long duration laparoscopic surgery in children. The ProSeal laryngeal mask airway (PLMA) provides protection against regurgitation and prevents gastric insufflation when correctly placed. However, it may render the upper esophageal sphincter incompetent either due to reflex relaxation or a direct mechanical effect.

Specially designed cuffed endotracheal tubes have been studied in children and may have an advantage over the uncuffed endotracheal tube. The cuffed tube may result in more effective ventilation and may offer better protection against aspiration in the face of increased IAP.

**Mechanical Ventilation during Laparoscopy**

Mechanical ventilation during laparoscopy has a potential to cause harm. Distribution of tidal volume to a smaller end expiratory lung volume causes lung strain. Protective lung strategy may also be extended to the operating room in children undergoing laparoscopic procedures with possible benefit.

Volume-controlled ventilation in the setting of laparoscopy with reduced lung compliance and increased airway resistance may result in high tracheal pressures. Pressure controlled ventilation provides a lower peak inspiratory pressure and is a popular mode especially in neonates and infants. The combination of decelerating flow and a constant airway pressure maintained over time improves ventilation and reduces intrapulmonary shunt and thereby improves gas exchange.

The Pressure Regulated Volume Control mode is available with new anesthesia ventilators and may have an advantage in laparoscopy. The combination of volume controlled ventilation with decelerating flow offers the advantages of pressure-controlled ventilation but with the guarantee of a minimum tidal volume.

The open lung strategy with application of a vital capacity maneuver after induction and thereafter every 30 minutes during the procedure with a minimum positive end expiratory pressure (PEEP) of 5 cms of H2O will aid in airway recruitment. PEEP has been used to offset the hypoxemia due to increased intra-abdominal pressure and the Trendelenburg position. Hyperoxia has been associated with lung toxicity in the form of oxygen free radicals leading to atelectasis and increased shunt. Protective lung ventilation with low tidal volumes may lead to hypercapnia but is safe in the absence of raised intracranial pressure or pulmonary hypertension. Increasing the respiratory rate is the rational approach to counteract hypercarbia, but very high rates could worsen the intrinsic PEEP.

There is no clear evidence yet of the superiority of one ventilation mode over the other as regards outcome after general anesthesia. As long as attention is paid to oxygenation, ventilation and prevention of lung strain, personal preference and expertise may influence the choice of ventilation mode.
**Intraoperative Monitoring**

Standard monitoring for these procedures includes pulse oximetry, capnometry, electrocardiography, non-invasive blood measure and temperature monitoring, and monitoring of urine output. In hemodynamically unstable or compromised patients, and in patients with cardiorespiratory diseases, careful invasive monitoring of cardiovascular system [CVP, IBP] and blood gases is indicated. Anesthetic gas concentration, ventilator performance monitoring are useful for safe anesthetic practice. There is need for a urinary bladder catheter and nasogastric tube to decompress the viscera and thus avoid injury during trocar insertion. The insufflators must be in view, and the anesthesiologist should be mindful of the insufflation pressure. Blood glucose measurement, especially in very small children, will help guide intravenous fluid administration. Isotonic fluid, such as Ringer’s lactate is administered intraoperatively. ETCO₂ is most commonly used as a non-invasive substitute for PaCO₂ in evaluating the adequacy of ventilation during laparoscopic surgery. However, it should be remembered that ETCO₂ may differ considerably from PaCO₂ because of ventilation-perfusion (V/Q) mismatching.

Abdominal insufflation of gas causes changes in respiratory mechanics which is reflected as an increase in airway pressure. The rise in airway pressure is due to both splinting of the diaphragm and stiffening of the chest wall and from the narrowing and distortion of the large conducting airways. The reduction in lung volume due to the upward shift of the diaphragm causes tendency to atelectasis in the dependant lung regions with the passage of time. Monitoring airway pressure continuously allows detection of dynamic hyperinflation or atelectasis formation. Monitoring of exhaled tidal volumes become important especially when pressure controlled ventilation is used.

**Analgesia**

A multimodal approach for perioperative analgesia, using a combination of local anesthetic infiltration of the trocar insertion sites, opioids, paracetamol and nonsteroidal anti-inflammatory drugs is recommended. Caudal epidural block has been demonstrated to be effective following laparoscopic inguinal herniorrhaphy in children. Alternatively, transversus abdominis plane (TAP) blocks can replace port site infiltration. At the end of the procedure child is extubated after the effects of muscle relaxants are worn off. Nerve stimulator can be used to confirm the adequacy of reversal.

**Postoperative Care**

The rate of CO₂ absorption is about 70 mL/min during the first 30 minutes of pneumoperitoneum and may increase to 90 mL/min. CO₂ elimination during laparoscopy is age dependant. The younger or smaller the child, the greater the increase in CO₂ elimination. The reason for this is not clear but is thought to be because of the different characteristics of their peritoneal surface. Also, CO₂ elimination continues well after desufflation, hence small children warrant close monitoring in the postoperative period. This is explained by the systemic redistribution of CO₂ rich blood after relief of the tamponade effect of the pneumoperitoneum on the venous return from the lower limbs, or the sudden increase in minute ventilation upon desufflation. Respiratory changes persist into the postoperative period when the ability to increase ventilation is often impaired by residual anesthetic drugs, effects of intravenous opioids and pain.

**Postoperative Nausea and Vomiting**

Postoperative nausea and vomiting can occur in children after laparoscopy and antiemetics should be prescribed for the postoperative period on an “as required” basis.

*PONV can be minimized by the following methods:*

- Avoiding N₂O; use of oxygen/air mixture for prolonged surgeries
- Avoid/limit the use of inhalational agents
- TIVA with propofol and dexmedetomidine
- Antiemetics: Injection ondansetron (100 μg/kg) and injection dexamethasone (0.25–0.5 μg/kg).

**COMPLICATIONS OF LAPAROSCOPY**

**Hypoxia and Hypercapnea**

Causes include endobronchial intubation, blockage/kinking of endotracheal tube, pneumothorax/pneumomediastinum, excessive IAP/patient position leading to splinting of diaphragm, and subcutaneous emphysema.

**Pneumothorax and Pneumomediastinum**

This is more common during procedures around the diaphragm e.g. fundoplication. Congenital defects of the diaphragm (patent pleuroperitoneal canal through which the insufflated gas passes into the thoracic cavity) or pleural tears have been suggested as the underlying mechanism.
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CO₂ Embolism

It is a lethal complication but is very rare, diagnosed by a sudden fall in EtCO₂ and hypotension. Transesophageal echocardiography, esophageal Doppler, whenever available, are sensitive monitors to detect embolism.

Other complications include reflex bradycardia. It may be due to vagal mediated reflex, which may be exaggerated by drugs like vecuronium, halothane, fentanyl and succinylcholine. It can be overcome by anticholinergic premedication.

Surgery-related Complications

Hemorrhage is a dreaded complication because intraperitoneal bleeding is more difficult to control laparoscopically and children respond poorly to hemodynamic disturbances. Inadvertent visceral injury during trocar insertion is another feared complication. The use of an open technique for the insertion of the first trocar and placement of subsequent trocars under direct vision minimizes unintentional major vessel and visceral injuries. New designs of trocars with safety mechanisms further reduce such risks. Overall, in large centers, the complication rate is low (1–2%).

Failure to evacuate CO₂ at the end of surgery can impair spontaneous ventilation by splinting the diaphragm causing hypercarbia, shoulder pain and vomiting.

ADVANCES IN LAPAROSCOPY

Single port laparoscopy is now gaining popularity among surgeons. It has limitations in pediatric surgery. The small size of the peri-umbilical region, skin fold around it and small intra-abdominal cavity in children pose technical difficulties.

The other recent development is three dimensional imaging systems which improve the spatial depth perception of target organ. The use of heated humidified carbondioxide for creation of pneumoperitoneum is gaining popularity.

CONCLUSION

Laparoscopy is safe in pediatric population. The anesthesiologist must be well aware of the implications of peritoneal insufflation. Patient selection must be made wisely and clinical monitoring, anesthesia technique and ventilation must be tailored to make these procedures safe for patients.

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2. Laseroxhn L. Anaesthetic considerations for paediatric laparoscopy. SAJS. 2011;49 (1).
INTRODUCTION

Thoracic anesthesia in infants and children poses special challenges for the anesthesiologist. These include assessment of the patient’s clinical condition, preoperative preparation, obtaining and maintaining single lung ventilation, maintaining adequate oxygenation and hemodynamics while allowing the surgeon access to a very limited intrathoracic cavity. Children may suffer from a wide variety of conditions, and may present with different degrees of pulmonary compromise which may not be evident from a cursory examination. A thorough understanding of pediatric physiology as well as of the principles of pediatric thoracic anesthesia and lung isolation techniques is important in order to conduct safe anesthesia. Surgical interventions with video-assisted thoracoscopic surgeries, have become increasingly common in the neonatal and pediatric populations. However, a successful VATS (video-assisted thoracic surgery) requires well-executed single-lung ventilation (SLV).1

PHYSIOLOGY OF SINGLE LUNG VENTILATION (SLV) IN CHILDREN

Ventilation is normally distributed preferentially to dependent regions of the lung, so that there is a gradient of increasing ventilation from the most non-dependent to the most dependent lung segments. Because of gravitational effects, perfusion normally follows a similar distribution, with increased blood flow to dependent lung segments. Therefore, ventilation and perfusion are normally well matched. During thoracic surgery, several factors influence the ventilation/perfusion (V/Q) ratio:

a. Compression of the dependent lung in the lateral decubitus position may cause atelectasis.
b. Surgical retraction and/or SLV result in collapse of the operative lung.
c. Hypoxic pulmonary vasoconstriction acts to divert blood flow away from underventilated lung regions, thereby minimizing V/Q mismatch. This beneficial effect may be countered by inhalational anesthetic agents and other vasodilating drugs.

The overall effect of the lateral decubitus position on V/Q mismatch, however, is different in infants compared to older children and adults.2,3

In adults with unilateral lung disease, oxygenation is optimal when the patient is placed in the lateral decubitus position with the healthy lung dependent (‘down’) and the diseased lung nondependent (‘up’). This is related to an increase in blood flow to the dependent, healthy lung and a decrease in blood flow to the nondependent, diseased lung due to the hydrostatic pressure (i.e. gravitational) gradient between the two lungs. So 40% of the cardiac output is used for perfusing the nondependant lung and 60% is directed to the dependant lung when both the lungs are being ventilated. During SLV blood flow to the nondependant lung is further reduced to 22.5% while 77.5% is directed to the dependant lung.4 Therefore, the dependant lung is better ventilated and better perfused. This phenomenon promotes V/Q matching in the adult patient undergoing thoracic surgery in the lateral decubitus position.
Ventilation-Perfusion in Lateral Decubitus (Figs 1A and B)

a. Atelectasis of non-ventilated lung.

b. Blood flow distribution
   - Gravity
   - Direct compression
   - HPV.

In infants with unilateral lung disease, however, oxygenation is improved with the healthy lung ‘up’. Several factors account for this discrepancy between adults and infants:

a. Infants have a soft, easily compressible rib cage that cannot fully support the underlying lung. Therefore, functional residual capacity is closer to residual volume, making airway closure likely to occur in the dependent lung even during tidal breathing.

b. The infant’s small size also results in reduced hydrostatic pressure gradient between the non-dependent and dependent lungs. Consequently, the favorable increase in perfusion to the dependent, ventilated lung is reduced in infants.

c. Finally, the infant’s increased oxygen requirement, coupled with a small functional residual capacity due to big abdominal contents and comparatively small thoracic cage predisposes to hypoxemia. Infants normally consume 6–8 mL of O₂/kg/min compared with oxygen consumption in adults of 2–3 mL/kg/min. For these reasons, infants are at an increased risk of significant oxygen desaturation during surgery in the lateral decubitus position.

Common indications for thoracic surgery and single lung ventilation (SLV) in adult as well as pediatric patients are:

Chronic empyema, lung cysts, mediastinal mass, pneumonectomy, lobectomy, bronchopleural fistula, unilateral bronchopulmonary lavage.

Indications specifically for pediatric patients are:

Congenital lobar emphysema, congenital pulmonary airway malformations, tracheoesophageal fistula, esophageal atresia, congenital diaphragmatic hernia, eventration of diaphragm, PDA ligation.

PREOPERATIVE EVALUATION

A thorough preoperative evaluation, including appropriate imaging and laboratory studies according to the lesion involved, is essential in caring for the pediatric patient scheduled for thoracic surgery. The history in older children focuses on complaints of dyspnea, cyanosis, wheezing, coughing, and weight loss. Infants often show less specific signs, such as poor feeding, irritability, or change in sleep habits. The chest is inspected for asymmetric expansion and use of accessory muscles and then is auscultated for wheezes, rales, rhonchi, and absent breath sounds in both the supine and sitting positions. Measurement of oxygen saturation by pulse oximetry and evaluation of venous HCO₃⁻ elevated in children with chronic CO₂ retention, generally supplant the need for arterial blood gas analysis. Preoperative CT scan of the chest is useful in children with anterior mediastinal mass. While pulmonary function testing may be useful in infants and children for monitoring progress of their underlying pulmonary process, it cannot be routinely done for perioperative assessment. Simple bedside spirometry may be performed in older children to assess the degree of restrictive or obstructive lung disease. ECG and echocardiography is obtained based on patients’ medical history.

PREOPERATIVE PREPARATION

Chest physiotherapy, bronchodilators, antibiotics, steroids supplementation helps in optimizing the patients
Preoxygenate adequately with 100% oxygen. Inhalation agents are especially useful in patients with bronchospasm but may precipitate hypotension in patients with poor cardiac function. There is some evidence that propofol does not inhibit hypoxic pulmonary vasoconstriction.7

PERIOPERATIVE MANAGEMENT

The goals of anesthesia should be to minimize the airway reactivity, optimize gas exchange, maintain adequate cardiovascular function, and provide adequate pain relief in the postoperative period. The choice of anesthetic agents depends on both the patient’s status and the surgical lesion. Nitrous oxide can accumulate in cysts with air–fluid levels and should be avoided in such cases or in patients requiring a high fraction of inspired oxygen (FiO2). Preoxygenate adequately with 100% oxygen. Inhalation agents are especially useful in patients with bronchospasm but may precipitate hypotension in patients with poor cardiac function. There is some evidence that propofol does not inhibit hypoxic pulmonary vasoconstriction.7

Muscle relaxants are routinely used along with controlled ventilation employing warm, humidified gases which can prevent blockage of the narrow lumen of very small double lumen tubes. Pressure controlled ventilation is preferred in pediatric patients to prevent barotrauma. Intermittent manual ventilation may provide useful information to the anesthesiologist about changes in chest compliance or airway resistance. Maintain a tidal volume of 8–10 mL/kg. Inspiratory-to-expiratory time ratio (I:E) of 1:2 prevents air trapping. Moderate hypercapnea, PaCO2 between 45 mm Hg and 60 mm Hg is acceptable unless this degree of hypercapnea cannot be tolerated because of other physiologic factors like concomitant hypoperfusion and metabolic acidosis. Isoflurane may be preferred due to less attenuation of hypoxic pulmonary vasoconstriction (HPV) compared with other inhalational agents, although this has not been studied in children.8 Intravenous opioids may facilitate decrease in the concentration of inhalational anesthetics used, and therefore limit impairment of hypoxic pulmonary vasoconstriction. Thoracic epidural anesthesia preserves this condition, and the combination of general anesthesia with regional anesthesia and postoperative analgesia is particularly desirable for thoracotomy.9 Thoracic epidural blockade may be achieved with greater safety and efficacy by placing the epidural catheter tip in proximity to the spinal segment associated with surgical incision. Segmental anesthesia may then be achieved with lower doses of local anesthetic than those needed when the catheter tip is distant from the surgical site. In infants, a catheter can reliably be advanced from the caudal to the thoracic epidural space. A variety of regional anesthetic techniques have been described for intraoperative and postoperative analgesia, including intercostal and paravertebral blocks, intrapleural infusions, and epidural anesthesia.

VIDEO-ASSISTED THORACOSCOPIC SURGERY (VATS)

In video-assisted thoracoscopic surgery, CO2 insufflation10 into the operative hemithorax (capnothorax) is used as a technique to facilitate collapse of the lung on the operative side. This is particularly useful in smaller patients where lung isolation is not possible and there is inadequate separation of the two lungs with overflow ventilation into the operative side. This approach offers the advantages of a smaller incision, less postoperative pain, a faster postoperative recovery and shorter hospitalization as compared with thoracotomy. Meticulous cardio-pulmonary monitoring is mandatory as displacement of intrathoracic contents and creation of an excessive pneumothorax can lead to significant cardiovascular
compromise from decreased venous return or high left ventricular afterload. The effects of the artificial pneumothorax can be minimized by slowly adding the CO₂ (flow rate 1 L/min) and limiting the inflating pressure to 4 to 6 mm Hg. Direct insufflation of CO₂ into the lung parenchyma can cause sudden rise in end-tidal CO₂. Subcutaneous emphysema and CO₂ embolism can occur.

Detection techniques for gas embolism that can identify the problem prior to the onset of cardiovascular changes include transesophageal echo (0.1 mL of gas), precordial Doppler (0.5 mL) and capnometric end-tidal nitrogen monitoring. The continuous insufflation of large volumes of cold, non-humidified CO₂ into the thoracic cavity causes hypothermia in small children. The combination of standard tracheal intubation with prone position and CO₂ insufflation may provide good exposure in some cases like pulmonary lobectomy in presence of infected secretions to prevent soiling of the healthy lung or repair of esophageal atresia. The anesthesiologist not only must be prepared for each complication but must also notify the surgeon immediately if there is loss of airway, difficulty in ventilation, or sudden hypotension. Utmost vigilance is needed as one encounters hypercapnea, arrhythmias, mediastinal shift, re-expansion pulmonary edema, atelectasis during surgery. Possibility of major vessel injury with torrential bleed should be kept in mind.

MEASURES TO MANAGE DESATURATION DURING SLV

If adequate oxygenation cannot be maintained after induction, the following measures can be instituted:

- Administer 100% O₂
- Check tube position, circuit, ventilator
- Rule out hypotension as the cause
- Reduce inhalation agent concentration (to less than 1 MAC)
- Suction the dependant lung
- Apply CPAP to nondependant lung
- PEEP to dependant lung, recruitment maneuver
- High frequency jet ventilation at low driving pressures 10–12 psi to the operative lung
- Ipsilateral pulmonary artery can be clamped
- Revert to intermittent both lung ventilation.

Fluid administration during thoracoscopic surgery should be done judiciously because excessive administration of intravenous fluids can cause increased shunting and subsequently lead to pulmonary edema of the dependent lung, particularly during prolonged surgery. Intravenous fluids are administered to replace the volume deficits, the maintenance fluids and the “third space” loss. The common dictum is “Don’t drown the down lung”. The fluid of choice is isotonic fluid, either Ringer’s lactate or normal saline.

SLV is an ideal technique in all pediatric patients due to the following advantages:

a. Provides an ideal surgical field and uninterrupted surgery.
b. Prevents bacterial contamination of the contralateral lung.
c. Prevents blockage of contralateral bronchus by blood clots or tissues.
d. Active inflation of the collapsed lung at the end of surgery is possible with a DLT or a bronchial blocker (BB) with a lumen.
e. Surfactant therapy for collapsed alveoli, e.g. pulmonary hypoplasia.
f. N₂O can be used in congenital lobar emphysema (CLE) without the risk of barotrauma.

METHODS OF SINGLE-LUNG VENTILATION IN PEDIATRIC PATIENTS (FIG. 2)

a. Endobronchial intubation was described first in 1932 by Gale and Waters. The main bronchus is intubated with a conventional single-lumen ETT by advancing it until breath sounds over the contralateral, operative lungs disappear. A fiberoptic bronchoscope (FOB) can be passed through or alongside the ETT to confirm or guide placement. Problems include failure to achieve
an adequate seal if an uncuffed tube is used. This may also prevent the operated lung from collapsing completely or fail to protect the healthy lung from contamination.\textsuperscript{13}

b. Synchronized independent lung ventilation (SILV) where both bronchi are intubated independantly and connected to two separate ventilators with different ventilatory settings, e.g. in bronchopulmonary dysplasia, CDH (congenital diaphragmatic hernia). In 1983, Hedenstierna used a bilumen tube and two servo ventilators for SILV (Fig. 3).

c. Balloon tipped bronchial blockers: The bronchus on the operative side is initially intubated with an ETT. A guidewire is then advanced through the ETT into that bronchus. The ETT is then removed and the blocker catheter is then advanced over the guidewire into the bronchus. The ETT is then reinserted into the trachea alongside the blocker catheter. Blockers which have been utilized are Fogarty embolectomy catheter, Magill’s, Foley’s or Swan-Ganz catheters (Figs 4A and B). The disadvantage of bronchial blocker is it requires a fiberoptic bronchoscope for proper placement. It takes a long time to position it. Lung may not collapse completely and suction is impossible if there is no lumen in the blocker. The high pressure, low volume cuff may damage the airway and a small amount of postextubation subglottic edema significantly increases the work of breathing in an infant. The blocker may get dislodged and block the airway completely. Repositioning during surgery in lateral position is difficult.\textsuperscript{14,15}

Cohen bronchial blocker has an angled tip, a high volume low pressure cuff, a Murphy’s eye and a proximal control wheel to adjust tip deflection (Fig. 5).

Arndt endobronchial blocker (invented by Dr Arndt, an anesthesiologist) has a multiport adaptor with three ports which allows simultaneous insertion of FOB, the blocker and gases for mechanical ventilation. This blocker can be used for children only above 2 years of age (Figs 6A to C and Table 1).
Table 1: Armst blocker: Size selection recommendations

<table>
<thead>
<tr>
<th>Armst size (Fr)</th>
<th>External diameter (cm) cuff down</th>
<th>Best patient age (years)</th>
<th>Smallest ETT size (mm) for placement within ETT</th>
<th>Cuff inflation volumes (mL)</th>
<th>Fiberoptic bronchoscope (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>50</td>
<td>1.7</td>
<td>&lt;8</td>
<td>4.5</td>
<td>0.5–2</td>
<td>2.2 or 2.8</td>
</tr>
<tr>
<td>70</td>
<td>2.3</td>
<td>8–12</td>
<td>6.5</td>
<td>2–8</td>
<td>2.8</td>
</tr>
<tr>
<td>90</td>
<td>3.0</td>
<td>&gt;12</td>
<td>8</td>
<td>Spherical 4–8</td>
<td>2.8</td>
</tr>
</tbody>
</table>

Abbreviations: ETT, endotracheal tube; Fr, French.

**Fuji uniblocker** shaft has a lumen to collapse the operated lung by aspirating air. These are "torque control" blockers, with easy to direct malleable shaft for smooth manipulation to position into the required bronchus. They are latex free so can be used for patients with allergy to latex (Fig. 7).
- 5F (Cuff volume 3 mL)
- 9F (Cuff volume 8 mL)

d. **The univent tube** is a conventional ETT with a second lumen containing a small tube that can be advanced into a bronchus. The balloon located at the distal end of this small tube, when inflated serves as a blocker (Figs 8A to D). These tubes require FOB for proper placement. The smallest size available is 3.5 mm internal diameter but the 8 mm outer diameter limits its use only for children above 6 years of age. The displacement of the univent blocker balloon is less likely. Univent torque controlled blocker (TCB) is a modification which facilitates easier introduction. The shaft is more flexible and easier to direct into the target bronchus and the blocker is made from a softer medical grade silicone material that is more compliant.

e. **All DLTs** are essentially two tubes of unequal length molded together. The shorter tube ends in the trachea and the longer tube in the bronchus. DLTs for older children and adults have cuffs located on the tracheal and bronchial lumens. The tracheal cuff, when inflated, allows positive pressure ventilation. The inflated bronchial cuff protects the lung from contamination from the contralateral side. Conventional plastic DLTs, once only available in adults sizes (35, 37, 39, and 41 Fr), are now available in smaller sizes. The smallest cuffed DLT is a 26 Fr which may be used in children as young as 8 years old. DLTs are also available in sizes 28 and 32 Fr and are suitable for children 10 years of age and older (Table 2).

In children, the DLT is inserted using the same technique as in adults. The tip of the tube is inserted just past the vocal cords and the stylet is withdrawn. The tube is rotated through 90 degrees to the appropriate side and then advanced into the bronchus. In the adult population, the depth of insertion is directly related to the height of the patient. No equivalent measurements are yet available in children. If FOB is to be used to confirm tube placement, a bronchoscope with a small diameter and sufficient length must be available. A DLT offers the advantage of ease of insertion, ability to suction and oxygenate the operative lung with CPAP. Left tubes are preferred to right DLTs because of the shorter length of the right main bronchus.
Chapter 25: Anesthesia for Pediatric Thoracic Surgery

Table 2: Tube selection for single-lung ventilation in children

<table>
<thead>
<tr>
<th>Age (year)</th>
<th>ETT (ID)\textsuperscript{a} (mm)</th>
<th>BB\textsuperscript{b} (Fr)</th>
<th>Univent (ID)\textsuperscript{c} (mm)</th>
<th>DLT\textsuperscript{d} (Fr)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.5–1</td>
<td>3.5–4.0</td>
<td>5</td>
<td>5</td>
<td>26</td>
</tr>
<tr>
<td>1–2</td>
<td>4.0–4.5</td>
<td>5</td>
<td>5</td>
<td>28–30</td>
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<tr>
<td>2–4</td>
<td>4.5–5.0</td>
<td>5</td>
<td>5</td>
<td>30</td>
</tr>
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<td>4–6</td>
<td>5.0–5.5</td>
<td>5</td>
<td>5</td>
<td>32</td>
</tr>
<tr>
<td>6–8</td>
<td>5.5–6</td>
<td>6</td>
<td>3.5</td>
<td>32</td>
</tr>
<tr>
<td>8–10</td>
<td>6.0 cuffed</td>
<td>6</td>
<td>3.5</td>
<td>26</td>
</tr>
<tr>
<td>10–12</td>
<td>6.5 cuffed</td>
<td>6</td>
<td>4.5</td>
<td>32</td>
</tr>
<tr>
<td>12–14</td>
<td>6.5–7.0 cuffed</td>
<td>6</td>
<td>4.5</td>
<td>32</td>
</tr>
<tr>
<td>14–16</td>
<td>7.0 cuffed</td>
<td>7</td>
<td>6.0</td>
<td>35</td>
</tr>
<tr>
<td>16–18</td>
<td>7.0–8.0 cuffed</td>
<td>7</td>
<td>7.0</td>
<td>35</td>
</tr>
</tbody>
</table>

Abbreviations: ETT, endotracheal tube; BB, bronchial blocker; ID, internal diameter; DLT, double lumen tube.
\textsuperscript{a}Sheridan\textsuperscript{®} Tracheal Tubes, Kendall Healthcare, Manhfield, MA
\textsuperscript{b}Arrow International Corp, Reddeg, PA
\textsuperscript{c}Fuij System Corporation Tokyo, Japan
\textsuperscript{d}26F Rusch, Dulush GA, 28–35F Mallinkrodt Medical Inc, St Louis, MO

Right DLTs are more difficult to accurately position because of the greater risk of right upper lobe obstruction. DLTs are relatively safe and easy to use. Their high volume, low pressure cuffs should not damage the airway if they are not overinflated with air or distended with nitrous oxide while in place.

Dr. Marraro described a bilumen tube made of radiopaque blue line PVC for infants to 5 year olds in 1985. It consists of two separate circular, uncuffed tubes of different lengths and sizes attached laterally to each other. Murphy's eye on the longer tube prevents exclusion of upper lobe bronchus. The disadvantage of pediatric DLT's is, confirmation of proper placement is difficult as FOB of very small sizes are difficult to acquire and too delicate. The smallest size available 1.8 mm which can fit through 2.5 size ETT. Fiberoptic scopes only larger than sizes 2.8 outer diameter have a side port for oxygen insufflation. Chances of blockage of the DLT lumen are high unless humidified and warm gases are used. Leak occurs around the uncuffed tube if proper size is not used (Figs 9A and B).

The most common problems and complications associated with DLT are essentially due to the inexperience in intubation, dislodgement and obstruction of the tube, trauma to trachea and bronchi. A malpositioned DLT will fail to collapse the operative side lung or partially collapse the ventilated lung causing hypoxia. There are at least six ways by which the proper positioning can be confirmed.

a. Visible chest wall expansion.
b. Auscultation.
c. Chest X-ray.
d. X-ray image intensifier (C-arm).
e. FOB.
f. Ultrasound.

POSTOPERATIVE MANAGEMENT

At the end of surgery, lung recruitment with manual ventilation is important to prevent atelectasis and hypoxia. Exubation immediately after the surgery is
usually preferred. However, depending on the patient’s underlying cardiopulmonary reserve, the course of the surgery, and the expected postoperative course, elective postoperative ventilation in PICU can be considered. Before extubation the patient must be awake, breathing well, able to cough and maintain an airway, and able to maintain oxygenation. A chest radiograph should be obtained as soon as possible after surgery to detect any significant pneumothorax or atelectasis.16

POSTOPERATIVE COMPLICATIONS

The expected postoperative course depends on both the surgical procedure and the underlying disease. Persistent air leak is the most common complication following VATS which can lead to subcutaneous emphysema or residual pneumothorax. Atelectasis is common and usually responds to humidification, encouragement to cough, and, if necessary, endotracheal suction.

Down lung syndrome is the term used for increased secretions and pneumonia that can develop in either lung following SLV. Lung herniation through the chest wall is possible. Major complications include airway obstruction, perforation of the airway, and massive blood loss. Infection ranges from local wound infection to a pulmonary abscess or empyema.17

POSTOPERATIVE PAIN CONTROL

Good postoperative pain relief can minimize the discomfort. Thoracoscopic procedures offer the advantage of small incisions without either splitting of the serratus anterior or latissimus dorsi muscles or spreading of the ribs which markedly contribute to postoperative pain. In order to minimize pain, patients breathe rapidly with small tidal volumes. This type of breathing promotes atelectasis, retention of secretions, decrease in FRC and increase V/Q mismatching causing hypoxemia. Oral NSAIDs, paracetamol or diclofenac rectal suppositories, parenteral opioids, intercostal nerve blocks, intrapleural instillation or local infiltration at port sites or epidural catheters are different modalities for pain relief.18

SALIENT FEATURES IN MANAGING PEDIATRIC THORACIC LESIONS

Pulmonary Sequestrations

Result from disordered embryogenesis producing a nonfunctional mass of lung tissue supplied by anomalous systemic arteries needing surgical resection. These generally do not get hyperinflated during positive pressure ventilation but N2O administration may result in expansion of these masses and should be avoided.

Congenital Cystic Lesions

These are classified into three categories: (a) Bronchogenic cysts, (b) Dermoid cysts, and (c) Congenital cystic adenomatoid malformation (CCAM) which is a common congenital lung lesion usually occurring in a single lobe. These lesions cause ipsilateral lung compression, pulmonary hypoplasia and mediastinal shift. They communicate with the airways causing overdistension due to gas trapping leading to respiratory distress. Treatment is surgical resection of the affected lobe. CCAM may contain fluid varying from clear to purulent in nature. SLV will allow better surgical access, minimize trauma to the limited residual lung tissue and protect normal lung from contamination. Prognosis depends on the amount of remaining lung tissue, which may be hypoplastic because of compression in utero.

Congenital Lobar Emphysema

Presents with respiratory distress shortly after birth due to overdistension of lung with fetal lung fluid. The resultant
emphysematous lobe may compress the lung bilaterally resulting in various degrees of hypoplasia. About 15% of these neonates have cardiac deformities. Lungs can get hyperinflated with positive pressure ventilation. \( N_2O \) is contraindicated and SLV is advocated.

### Congenital Diaphragmatic Hernia

In congenital diaphragmatic hernia, failure of a portion of the fetal diaphragm to develop allows abdominal contents to enter the thorax, interfering with normal lung growth. In 70 to 80% of diaphragmatic defects, a portion of the left posterior diaphragm fails to close, forming a triangular defect known as the foramen of Bochdalek. Hernias through the foramen of Bochdalek occurring early in fetal life usually cause respiratory failure immediately after birth because of pulmonary hypoplasia. Distention of the gut postnatally with bag-and-mask ventilation exacerbates the ventilatory compromise by further compressing the lungs. Neonates present with tachypnea, a scaphoid abdomen, and absent breath sounds over the affected side. Chest radiography typically shows bowel in the left hemithorax with deviation of the heart and mediastinum to the right and compression of the right lung. In the presence of significant respiratory distress, bag-and-mask ventilation should be avoided and tracheal intubation should be performed immediately. Pulmonary hypertension with right-to-left shunting contributes to severe hypoxemia in neonates with CDH. Surgical correction through a subcostal incision, or alternately via thoracoscopy may be performed. Hyperventilation to induce a respiratory alkalosis and 100% oxygen should be administered to decrease pulmonary vascular resistance.

The anesthetic should be designed to minimize sympathetic discharge, which may exacerbate pulmonary hypertension (e.g. a high-dose opioid technique). Infants should be ventilated with small tidal volumes and low inflating pressures to avoid pneumothorax on the contralateral side. Mechanical ventilation may be continued postoperatively.

### Tracheoesophageal Fistula

In majority of infants, this lesion includes esophageal atresia with a distal esophageal pouch and a tracheal fistulous connection. The neonates present with spillover of pooled oral secretions from the pouch and may develop progressive gastric distention and tracheal aspiration of acidic gastric contents through the fistula. Esophageal atresia is confirmed when an orogastric tube passed through the mouth cannot be advanced more than approximately 7 cm. The tube should be secured and placed on continuous suction. Mask ventilation and tracheal intubation are avoided prior to surgery because they may exacerbate gastric distention and respiratory compromise. When the trachea is intubated, an attempt is made to occlude the tracheal orifice of the fistula with the tracheal tube. The tip of the tracheal tube is positioned just above the carina.

Surgical repair usually involves a right thoracotomy and extrapleural dissection of the posterior mediastinum. In most cases, the fistula is ligated and primary esophageal anastomosis is performed. The trachea may be intubated with the patient breathing spontaneously or during gentle positive-pressure ventilation with small tidal volumes to avoid gastric distention.

Alternatively, the tracheal tube may be positioned in the main-stem bronchus opposite the side of the thoracotomy incision until the fistula is ligated. The advantages of the thoracoscopic approach include reduction in the musculoskeletal sequelae that often develop following open thoracotomy in the newborn period. These have been well described as “winged” scapula, asymmetry of thoracic wall and thoracic scoliosis. In addition, surgeons have described superior visualization of fistula and surrounding structures including vagus nerve with the thoracoscopic approach. The ability to see all structures clearly allows less traction on the trachea and surrounding structures which can perhaps decrease postoperative stridor and risk of recurrent laryngeal nerve injury.

### Mediastinal Mass

Elicit history of cyanosis, stridor and whether sleep, excitement, position, movement of the head and neck, or coughing changes the degree of obstruction. Anesthetic induction in these patients can lead to severe airway obstruction, hemodynamic compromise and death. Cardiovascular involvement may be related to direct compression of the heart or of the great vessels. If the child has arrhythmias, pulsus paradoxus, hypotension, or superior vena caval syndrome, the risk of general anesthesia increases dramatically. Mask induction with a volatile agent and 100% oxygen is preferred to intravenous induction if there is concern about airway obstruction. It is preferable to maintain spontaneous ventilation till the chest is opened.

### Pectus Excavatum

Children with pectus excavatum (funnel chest) or pectus carinatum (pigeon breast) deformity often appear asymptomatic, but may have cardiac or pulmonary compromise related to the structural abnormality.
Patients with pectus excavatum may present with reduced forced vital capacity and total lung volume. The heart may be displaced to the left and compressed, leading to arrhythmias, right axis deviation on electrocardiogram, and a functional murmur. There is also an increased incidence of mitral valve prolapse in patients with pectus deformities. SLV is usually not necessary but a thoracic epidural is ideally placed immediately after induction of anesthesia for intraoperative and postoperative pain control.

**Empyema**

Thoracoscopic decortication is effective in the early treatment of pediatric parapneumonic empyema. Thoracoscopy facilitates proper visualization, evacuation, and mechanical decortication of the thickened pleura with no additional morbidity and may lead to reduced time for chest tube drainage, shorter hospitalization, and more rapid clinical recovery. After successful adhesiolysis, the affected lung can be recruited and inflated with positive pressure ventilation to check for any air leak. Small air leaks generally improve after closure.

A chest tube or intercostal drain is inserted through the chest wall into the pleural space postoperatively to remove air (pneumothorax), fluid (pleural effusion, blood, chyle) or pus (empyema) from the intrathoracic space into drainage canister which has a water seal chamber.

Air bubbling through the water seal chamber is usual when the patient coughs or exhales, but if persistent may indicate air leak from the lung. The chest tube clogging due to thrombus formation inside can cause blood or air to be retained around the heart and lungs causing complications like pericardial tamponade, hemothorax, pleural effusion, tension pneumothorax and empyema. While transferring the patient from the operation table to the ward or ICU, the tube has to be temporarily clamped to prevent the backflow of water from the chamber into the pleural cavity. After the clamp is released, confirm that the water column in the tube is moving freely.

**CONCLUSION**

Thoracic anesthesia in infants and children poses special challenges for the anesthesiologist. These include careful preoperative preparation, obtaining SLV in a small airway, and maintaining a delicate balance of ventilation and hemodynamics while allowing the surgeon access to a very limited intrathoracic cavity. Ensuring adequate monitoring, intravenous access, blood replacement, and constant communication with the surgeon about developing situations are essential to success.

**REFERENCES**

Anesthesia at Remote Locations

INTRODUCTION
Administering anesthesia outside the operating theater at an alternate site has become part and parcel of the anesthesiologists’ domain. Demand to anesthetize patients including children at these locations is increasing due to the advances in diagnostic and therapeutic procedures. These procedures frequently require huge and heavy equipment, which cannot be transported to the operating rooms. The comfort and smooth organization of the operating theater will not be present at such locations. Therefore, there is always a chance of breach in safety in such situations unless the anesthesiologist is competent, knowledgeable and well prepared to handle these challenges.

GOALS
The goals of sedation/anesthesia for remote locations:
- Guard the patients safety and welfare
- Minimize physical discomfort and pain
- Control anxiety, minimize psychological trauma and maximize the potential for amnesia
- Control behavior and movement of the patient to allow safe completion of the procedure
- Return the patient to a state in which safe discharge from medical supervision is possible.

PROBLEMS
Unfamiliar locations and working conditions pose certain problems:
- Related to physical layout of the facility
- Remoteness from available help
- Difficult or limited access to patients
- Unfamiliar or outdated anesthesia equipment
- Untrained personnel.

In this chapter, we will discuss the strategy we should follow to deliver anesthesia at locations remote from operating theater effectively and safely under following headings:
- Pre-procedure planning
- Medications
- Documentation
- Anesthesia techniques
- Monitoring
- Recovery and discharge planning.

PRE-PROCEDURE PLANNING
Ideally anesthesiologist should be involved in organizing and designing the suite, so that the same uniform standard of anesthesia care can be taken in these remote locations as in our operating rooms.

Pre-procedure planning is the key to any anesthesia administered. A thorough history taking, clinical examination and parental counseling in the form of information brochures and procedure specific requirement should be considered. Preoperative fasting guidelines should be followed strictly and a written informed consent for anesthesia is a must.
Guidelines for Anesthetic Care Delivered Outside the Operating Room: American Society of Anesthesiologists (ASA) 1994

Guidelines for Non–operating Room Anesthetizing Locations

1. A reliable oxygen source with backup as required—piped gas or cylinders
2. A working suction source with all proper connections and suction catheters
3. Waste gas scavenging system
4. Adequate monitoring equipment to meet the standards for basic anesthesia monitoring and, in addition, a self-inflating hand resuscitator bag/transport ventilator
5. Sufficient safe electrical outlets
6. Adequate illumination of the patient and anesthesia machine with battery-powered backup
7. Sufficient space for the anesthesia care team
8. An emergency cart with a defibrillator, emergency drugs, and other emergency equipment
9. A means of reliable two-way communication to request assistance
10. Compliance of the facility with all applicable safety and building codes

It is the responsibility of the anesthesiologist providing care to ensure that the anesthetizing location in which the care is delivered meets all applicable standards.

Patient Evaluation

Clinicians should be familiar with the sedation related aspects of the patient's medical history.

These include:
- Abnormalities of major organ systems
- Previous adverse effects with sedation and general anesthesia
- Drug allergies, current medications, and drug interactions
- Time and nature of oral intake

A focused physical examination including vital signs, auscultation of the heart and lungs and evaluation of the airway is recommended.

Preprocedural Fasting Guidelines

Sufficient time should elapse before a procedure to allow gastric emptying in elective procedures. Minimum fasting period recommended for healthy children:

<table>
<thead>
<tr>
<th>Type of Food</th>
<th>Fasting Period</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clear liquids</td>
<td>2 hours</td>
</tr>
<tr>
<td>Breast milk</td>
<td>4 hours</td>
</tr>
<tr>
<td>Infant formula, nonhuman milk, light meal</td>
<td>6 hours</td>
</tr>
</tbody>
</table>

If urgent, emergent, or other situations impair gastric emptying, the potential for pulmonary aspiration of gastric contents must be considered in determining the target level of sedation or the best technique to adequately protect the airway during the procedure.

MEDICATIONS

Anesthesia drugs should be chosen judiciously to facilitate the procedure with minimal patient movement and anxiety, without compromising patient safety. An ideal agent should have rapid onset, titrable effect, be efficient in achieving loss of consciousness and amnesia without compromise of hemodynamic functions, be painless to administer, with minimal side effects, easy reversal, and fast recovery. Many agents have these properties but none possess all. Numerous protocols and guidelines are available which involve the use of a single agent or a combination of medications; these vary from institute to institute and as per local guidelines and practices. A combination of drugs give the desired ideal effect in a shorter period with smaller doses of individual drugs, but require increased monitoring and preparedness for unforeseen adverse reactions and rapid changes in the level of anesthesia. The ideal approach in any situation remains to individualize the protocol for every patient, taking into consideration the patients' medical condition and titrating the drugs to ensure patient safety, achieving the desired goal for the procedure.

A brief pharmacology of the agents commonly used for procedural sedation and anesthesia is described in Table 1, with their dosages, route and properties.

Inhalational Agents

Nitrous Oxide: It is an effective analgesic and sedative agent, useful at all ages. It can be used up to 70% with oxygen 30%. Side effects are nausea, vomiting, and diaphoresis.

Sevoflurane: It is a sweet smelling agent, nonirritant to airway, and has a fast induction and recovery.

DOCUMENTATION

Documentation is a must for following reasons:
- Medicolegal purposes
Table 1: Medications used during procedural sedation and anesthesia with dose, route and important properties

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Route</th>
<th>Dose</th>
<th>Properties</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chloral hydrate/Triclofos</td>
<td>PO/PR</td>
<td>25–100 mg/kg</td>
<td>Onset of action 20-30 min, Prolonged sedation, unpredictable effect</td>
</tr>
<tr>
<td>Midazolam</td>
<td>IV/IM/PO/PR/IN</td>
<td>IV:0.02–0.05 mg/kg; IM:0.07–0.15 mg/kg; PO/PR:0.25–0.75 mg/kg; IN:0.2–0.4 mg/kg</td>
<td>Excellent amnesia and anxiolysis, rapid and short acting, reversal with flumazenil</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>IV</td>
<td>1–4 µg/kg</td>
<td>Excellent analgesia, causes respiratory depression, reversal with naloxone</td>
</tr>
<tr>
<td>Ketamine</td>
<td>IV/IM</td>
<td>IV:1–2 mg/kg; IM: 2–5 mg/kg</td>
<td>Dissociative anaesthetic, excellent analgesia, minimal respiratory depression, increases bronchial secretion</td>
</tr>
<tr>
<td>Propofol</td>
<td>IV</td>
<td>1–1.5 mg/kg loading dose, 0.25–0.5 mg/kg every 3–5 min or 50–150 µg/kg/min infusion</td>
<td>Rapid induction of general anesthesia, short acting, causes apnea, effect not reversible</td>
</tr>
<tr>
<td>Dexmedetomidine</td>
<td>IV</td>
<td>1–2 µg/kg loading dose followed by 0.05–1 µg/kg/h</td>
<td>Short acting sedative, no respiratory depression, hypotension and bradycardia at higher doses</td>
</tr>
<tr>
<td>Flumazenil</td>
<td>IV</td>
<td>0.02 mg/kg every minute, maximum 1 mg</td>
<td>Reverses benzodiazepine induced respiratory depression</td>
</tr>
<tr>
<td>Naloxone</td>
<td>IV/IM</td>
<td>0.1 mg/kg/dose every 2–3 min, maximum 2 mg</td>
<td>Reverses opioid induced respiratory depression</td>
</tr>
<tr>
<td>Atropine</td>
<td>IV/IM</td>
<td>0.01 mg/kg</td>
<td>Antisialagogue, caution in cystic fibrosis</td>
</tr>
<tr>
<td>Glycopyrrolate</td>
<td>IV/IM</td>
<td>0.04 µg/kg</td>
<td>Antisialagogue</td>
</tr>
</tbody>
</table>

Table 2: The sedation continuum

<table>
<thead>
<tr>
<th>Responsiveness</th>
<th>Airway</th>
<th>Spontaneous Respiration</th>
<th>Hemodynamics</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Minimal sedation</strong></td>
<td>Normal response to verbal stimulation</td>
<td>Unaffected</td>
<td>Unaffected</td>
</tr>
<tr>
<td><strong>Moderate sedation</strong></td>
<td>Purposeful response to verbal or tactile stimulation</td>
<td>No intervention required</td>
<td>Adequate</td>
</tr>
<tr>
<td><strong>Deep sedation</strong></td>
<td>Purposeful response following repeated or painful stimulation</td>
<td>Intervention required SOS</td>
<td>May be inadequate</td>
</tr>
<tr>
<td><strong>General anesthesia</strong></td>
<td>Not arousable, to painful stimuli too</td>
<td>Intervention often required</td>
<td>Frequently inadequate</td>
</tr>
</tbody>
</table>

- Record the anaesthesia technique and vital parameters
- To develop intradepartmental procedure specific protocol
- To aid in audit and research

The consent form, preoperative questionnaire, preprocedure evaluation, anesthesia chart and postprocedure monitoring charts should be maintained.

**ANESTHESIA TECHNIQUE**

Minimal, moderate and deep sedation/analgnesia and general anesthesia are usually considered for remote locations (Table 2).4,5

With deeper sedation an oral airway may be required and sometimes securing the airway with a laryngeal mask airway or endotracheal tube is a better and safer option.

The patient’s condition, the anticipated level of stimulation, and patient position during the procedure must be considered in choosing the technique.

**MONITORING**

ASA standards for basic anesthesia monitoring require:7
- Presence of qualified anesthesia personnel throughout conduct of the course of anesthesia.
- Continuous evaluation of the patient’s oxygenation, ventilation, circulation, and temperature.
- Provision is made for the absence of anesthesia personnel from the immediate vicinity of the patient if required for safety (i.e. in the presence of radiation hazards), provided that adequate patient monitoring is continued.
- Oxygen concentrations of inspired gas should be monitored with the use of a low FiO2, audible and visual
alarm; blood oxygenation should be monitored with pulse oximetry and ventilation should be monitored by observation of the patient.

- The position of the endotracheal tube must be verified by observation and by detection of end-tidal carbon dioxide (CO\textsubscript{2}). Continuous end-tidal carbon dioxide analysis should be performed.
- When mechanical ventilation is used, a disconnect alarm with an audible signal must be present.
- Circulation is monitored by continuous display of the electrocardiogram, as well as by measurement of arterial blood pressure at a minimal interval of 5 minutes, in addition to other assessments, such as auscultation, palpation of pulse, invasive blood pressure monitoring, or oximetry.
- When changes in body temperature are anticipated or suspected, patient temperature should be assessed.

There should be no hesitation to use invasive monitoring if the patient condition warrants it.

RECOVERY AND DISCHARGE PLANNING\textsuperscript{5,6}

The patient must be medically stable before transport and must be accompanied to the recovery area by the individual providing the anesthesia or sedation/analgesia care. Appropriate recovery facilities should be provided with adequate oxygen delivery and monitoring while the patient is in the recovery room. Trained staff must be provided to continually assess, monitor and document the patient’s condition. Availability of personnel trained in advanced cardiac life support as well as an emergency crash cart should be ensured. Patient with multiple comorbidities and complicated procedure should be shifted to full-fledged postanesthesia care unit.

Many of these procedures are done on day-care basis so discharge criteria such as the Postanesthetic Discharge Scoring System (PADSS), should be met before sending patient home.

INDIVIDUAL SCENARIOS

MRI Anesthesia

Though there is no radiation hazard in the MRI suite like in the other radiological suites, it has its own challenges.

Magnetic Field

There is a very strong magnetic field of 0.5–3.0 tesla present in scanner room (gantry). The magnetic field of the earth is only 0.5 gauss. (1 tesla = 10,000 gauss).

No ferromagnetic objects are allowed within the 50 gauss line as they will not only malfunction, but also damage the magnet of the MRI machine. In the scanner room, following things are banned for their missile like effect: scissors, laryngoscopes, gas cylinders.

Four zones have hence been delineated around the scanner:\textsuperscript{8}

<table>
<thead>
<tr>
<th>Zone</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>No magnetic field. Reception, relative waiting area, dressing rooms and lockers</td>
</tr>
<tr>
<td>II</td>
<td>Pre and post recovery</td>
</tr>
<tr>
<td>III</td>
<td>Console room for radiology and anesthesia staff</td>
</tr>
<tr>
<td>IV</td>
<td>MRI gantry (Magnet)</td>
</tr>
</tbody>
</table>

Patient

Since even slight movement can disrupt the MRI images, children need to be sedated unless they are older and co-operative.

- Children who come for these studies can have multiple problems.
  a. Premature babies should be preferably taken after 60 weeks of conceptual age, as the incidence of post-procedure apnea reduces significantly.
  b. Caution should be taken in patients with:
     - Developmental delay (cerebral palsy)
     - Neuromuscular disorders
     - Chromosomal abnormalities
     - Obstructive sleep apnea
     - Down’s syndrome with cardiac abnormalities
     - Cervical instability (Atlantoaxial subluxation)
     - Cardiac patients should have a cardiac evaluation within 1 year of MRI date, and current use of cardiac medication should be noted.
     - Patients with reactive airway and patients having URTI should be evaluated clinically.
  c. Patients with artificial heart valves, pacemakers, aneurysmal clips, cochlear implants, and steel splinters in the eye are absolute contraindications for MRI, unless they are MRI compatible.

Auditory Damage

The dynamic magnetic field causes mechanical vibration and noise production up to 90 db which can cause potential cochlear damage in children as well as relatives. The child and the relative should be provided with earplugs.

Duration

One single MRI sequence can last up to 15 minutes, so a complete scan can take more than 3 hours and the child needs to be immobile for that long.
Postprocedure Recovery

This is a day-care procedure and recovery has to be prompt, as the child has to be clear headed without side effects before discharge.

Anesthesia Considerations

- Pre-procedural evaluation and consent forms to be filled by relatives
- All metallic objects including pagers and credit cards to be left outside the MRI gantry
- Cotton earplugs or earmuffs to be used for both patient and relative
- Allergy to the intravenous contrast agents, gadopentetate or gadodiamide, may cause some adverse effects such as severe hypothermia, hypotension, local erythema, or vomiting.

Equipment for MRI

Equipment should be such that there will be least interference in the monitoring signal quality due to the magnetic field, along with the least disruption of the MRI images. Eddy currents constantly produced by this magnet, get conducted through the ECG leads and the local heat production may potentially cause burns. Most MRI scanning rooms are protected from signal disruption. All electrical equipment within the scanner is shielded. Electrical cables are filtered with radiofrequency filters.

- ECG: Placing the electrodes in the center of the chest can reduce interference with ECG waves, avoiding looping of the wires and emphasizing on V5-V6 leads. Telemetric ECG may be used when possible to avoid the antenna-like effect of the leads.
- Liquid crystal display (LCD) of the monitors to minimize distortion of the waves.
- Pulse oximeter: Fiberoptic cables can be used.
- Capnography: Side-stream sampling with long sampling set required.
- Blood pressure equipment: Connector should not be made of metal, and it should have an extended length of tubing.
- Invasive monitoring: The transducer should be radiofrequency filtered and kept as close to the patient as possible.
- Plastic laryngoscope with lithium batteries should be available.
- MRI compatible ventilator and anesthesia machine, aluminum cylinders can be used.
- Defibrillator should be placed beyond the 50 Gauss line along with other resuscitation equipment. In case of cardiovascular collapse, only basic life support measures should be started in the gantry. Advanced cardiac life support and other CPR measures should be done after getting the patient outside the scanner room. The patient can be transported on an aluminum or a stainless steel trolley (Figs 1 and 2).

CT Scan

CT scan is faster than MRI, and is preferred in emergency situations such as intracranial hemorrhage, abdominal or thoracic mass, encephalopathy and head injury patients. Since the actual time required for CT scan is very short, 10–40 seconds, often presence of parents is enough.

Indications for GA are:
- Complicated cases along with other abnormalities
• To evaluate choanal atresia, craniofacial abnormalities
• Scanning with extreme head extension or absolute neck immobility for 3D reconstruction
• HRCT Chest, which requires apnea.

Anesthetic Considerations
• Patients should follow fasting guidelines. Oral contrast like gastrograffin (1.5%) may be used. It is considered a clear fluid and GA can safely be given two hours after ingestion taking due precaution against vomiting and aspiration.
• CT chest may require apnea, which necessitates deep sedation with assisted ventilation.
• CT-guided biopsies in children require sedation with analgesia and local anesthesia, or even complete anesthesia depending on the site, duration, and pain of the procedure.
• Watch for allergic reactions to contrast media.

Interventional Radiology
Interventional radiology procedures are becoming increasingly popular as compared to surgical intervention (Fig. 3).3

Indications
• Diagnostic angiography and WADA tests
• Embolization and sclerotherapy of vascular malformations like AVM, hemangiomas, vascular tumors
• Peripheral angioplasty
• Needle biopsies
• Nephrostomy, gastrostomy tube placement.

Anesthetic Considerations
• Depending on the procedure and patient, anesthesia can range from sedation to general anesthesia with controlled ventilation.
• High flow lesion like AVM should be evaluated for high output cardiac failure, congestive cardiac failure and pulmonary edema. Repeated anesthesia may be required for occlusion of large AVM like the vein of Galen aneurysmal malformation.
• Diagnostic and WADA tests usually require only light sedation with midazolam, fentanyl or propofol, but in therapeutic procedures GA with endotracheal intubation is preferred.
• Precautions should be taken to protect personnel from radiation exposure by utilizing lead aprons, lead collars, dosimeters and lead glass screens.

Goals of Anesthesia
• Immobile patient
• During the procedure, manipulation of the blood pressure may be required using vasodilators like NTG
• Controlled hypercarbia causes vasodilation and better visualization of blood vessels
• Heparin is required to prevent thrombosis and ACT needs to be monitored and maintained >300 seconds. Protamine should be available to reverse the anticoagulation.

General anesthesia allows easy control of blood pressure and ventilation, and the patient is immobile. However, periodical neurological assessment is not possible.

Preoperative history of seizures, bleeding risks, anticoagulants, anticonvulsants, signs of increased ICP, should be noted. Routine preoperative investigations including coagulation, renal and cardiac profiles and blood grouping and cross matching are mandatory.

Since large volumes of saline flush are used during the procedure, it is necessary to guard against hypothermia with monitoring of temperature and use warming devices.

Postembolization, vasodilators like nimodipine or nitrate infusion may be used to maintain the blood pressure.

Complications include stroke, cranial nerve palsies, skin necrosis, infection and pulmonary embolism. A postoperative neurological assessment should be documented.

Special Requirements
Portable lights, temperature monitors, radiant heating devices, long breathing circuits, long intravenous line extensions, ACT monitoring.
The agents used for the permanent occlusion of AVM are polyvinyl alcohol foam, ethanol, GDC coils and cyanoacrylate glue. Temporary occlusion is done using absorbable gelatin pledgets or powder. Massive systemic embolization can cause systemic intravascular coagulation (SIC) with increased PT with decreased coagulation factors. Other complications include sudden intracranial hemorrhage, acute thrombosis, incomplete occlusion and catheter balloon migration.

Cardiac Catheterization

**Indications**
- Percutaneous cardiac interventions—cardiac catheterizations, endovascular closure of intracardiac defects, like ASD, VSD and PDA
- Insertions of AICD.

**Anesthesia Considerations**
- Patients’ anomalies vary from simple atrial or ventricular septal defects to complex congenital cardiac and non-cardiac anomalies. Patients may be cyanotic.
- Moderate to deep sedation with propofol, fentanyl and midazolam with or without a LMA, may be enough for angiography.
- Closure of intracardiac defects requires general anesthesia with endotracheal intubation, as a TEE probe is used for these procedures.
- Heparin is administered to maintain ACT >300s.
- Monitoring of temperature, blood gases, blood sugar, and hematocrit is necessary.
- Insertion of an automated cardioverter defibrillator (AICD) needs close monitoring of hemodynamics.

Ultrasound Procedures

- Needle biopsies kidney, liver, lung, muscle, unknown mass and aspiration of fluids.
- Drainage procedures—percutaneous drainage of abscess, cysts, pancreatic pseudocyst.
- Central venous line insertion.

**Anesthesia Considerations**
Mild sedation to complete general anesthesia with controlled ventilation depending on:
- Duration of procedures
- Location
- Age of patient
- Associated risk
- Procedure requirement.

Nuclear Medicine (PET/SPECT)

**Indications**
- To locate epileptic foci in refractory epilepsy
- Evaluation of moyamoya disease
- Evaluation of cognitive and behavioural disorders

**Single Photon Emission Computed Tomography (SPECT):**
Radionuclide dye: Technitium radionuclide is an ideal dye that remains intracellular for a prolonged period, and can measure the regional blood flow. It is seen even 6 hours after the seizure. There is a hazard of contact radiation and requires protective wear such as gloves. This scan is used to map the epileptic foci prior to surgical resection. Due to the presence of radiation, remote monitoring from the console is required.

**Positron Emission Tomography (PET):**
It should be performed during the seizure or 1 hour after the seizure, as the radionucleotide tracer is glucose or oxygen with a short half-life.

**Anesthesia Considerations**
The child should remain immobile at least for 1 hour. Moderate to deep sedation with midazolam or propofol is usually sufficient.

Scopy

**Indications**

**Upper Gastrointestinal Endoscopy**
- Biopsy
- Foreign body removal
- Esophageal strictures for dilation or stenting
- Percutaneous endoscopic gastrostomy.

**Colonoscopy**
- For diagnosis of chronic diarrhea
- Failure to thrive.

**Anesthesia Considerations**
- General anesthesia with intubation is required for foreign body removal
- Aspiration can occur during inflation of stomach during upper gastrointestinal scopy
- Use of antispasmodics cause tachycardia
- Positioning—Lateral/semiprone/prone
- Time taken for the procedure varies
- Injury to teeth must be anticipated. Prevent injury to the scope in case the patient bites by using a bite block
- Colonoscopy can be performed under deep sedation.
Radiation Oncology

External beam radiation therapy (EBRT) is a painless brief procedure, which requires immobile child for a very short duration. It is performed daily or twice daily for up to 6 weeks. Simulation for planning and preparation of moulds for radiation prior to actual radiation requires at least 1-2 hours. However, there is no radiation hazard present in this room during the simulation, so, anesthesia personal can be present inside the suite.

Anesthesia Considerations

- Immobile patient with rapid onset anesthesia, and a rapid recovery
- Drugs: Oral triclofos, combination of IV/IM midazolam and ketamine can be used. Total intravenous anesthesia with propofol, maintaining spontaneous respiration with a rapid recovery is preferable
- If airway cannot be maintained, then GA with LMA should be the modality of choice
- Patients may develop tachyphylaxis because of repeated propofol use, necessitating a frequent change of drugs and dosages
- Temperature monitoring is important, as there can be a tendency towards hyperthermia
- Patients’ vitals are monitored by closed circuit cameras due to inability of the anesthesiologist to be present in the therapy room due to the radiation hazard. These treatments are very short and can be interrupted at anytime in case of an emergency.

Gamma Knife (Fig. 4)

Indications

- Brain tumors, e.g. ependymoma, medulloblastoma, germinoma, astrocytoma and vascular malformations.
- It can be an alternative to surgery or is performed for residual tumor postsurgery. Advantage of radiosurgery over conventional EBRT is that the remainder of the brain receives a small dose of radiotherapy.
- Residual vascular malformation postsurgery or deep-seated lesion can also be treated with radiosurgery.

Three steps are involved:

- Application of metal frame with four pins to skull; this can be done under local anesthesia with deep sedation with midazolam, dexmedetomidine or propofol
- MRI/cerebral angiography for planning coordinates for treatment; sedation may be required. A MRI compatible spanner to loosen the bolts of the frame must be available in case of emergency, e.g. loss of airway or accidental extubation, to allow access to the airway.
- Actual radiotherapy: Usually does not require any sedation, but in uncooperative patients an infusion of dexmedetomidine or propofol is adequate. General anesthesia with intubation or LMA is difficult because of the radiation hazard to the anesthesiologist.

The total procedure may last up to 2 hours.

Note: Dental procedures and burns have been covered in other chapters in detail; the key points are highlighted here.

Dental Procedures

Depending on age, anxiety, mental disability, the patient may require minimal to moderate sedation/analgesia or even general anesthesia.

Apart from the common challenges for remote location, dental anesthesia is characterized by:

- Sharing of airway with surgeon
- Use of small instruments like burrs, implants, files, which may fall in and get aspirated into the oropharynx
- Potential for bleeding due to the rich blood supply in the head and neck region
- Possible intense pain transmitted by maxillary and mandibular divisions of trigeminal nerve.
- Coexisting cardiorespiratory anomalies and airway abnormalities such as macroglossia, hypoplastic maxilla, palatal malformations, prognathism, micrognathia
- Due to the sitting position in the dental chair, the vasodilatory effects of the anesthetic agents are
exaggerated. Close monitoring of the hemodynamic parameters is important
• For general anesthesia, nasotracheal intubation with throat pack is preferred.

**Burn Dressings**

It is a short but very painful procedure which may be performed in the casualty room or in the wards. Most cases require sedation with some analgesia. Always confirm starvation status and consent. Airway assessment is important if the burns are on the head, neck or chest area. A combination of midazolam and ketamine is commonly used.

**LEARNING POINTS**

• As the number and complexity of procedures being performed under anesthesia at remote areas increase, the standards of care should not vary with the location.
• Advances in drugs and monitoring equipment help in the administration of safe anesthesia.
• Good communication between all the personnel, both physicians and staff involved, is necessary and ensures a smooth and successful outcome.

**REFERENCES**

INTRODUCTION
In the era of increasing cost of medication and hospitalization, and also the increasing number of the patients in the hospital, ambulatory or outpatient anesthesia has come as a boon to both the patients and also to the hospitals. This is more so in view of the increasing safe practices of anesthesia and monitoring. The concept of ambulatory anesthesia is not new. It has been in practice from the days of Crawford Long who used ether in 1842 to do short procedures in his clinic.1

The publication on ambulatory anesthesia by Dr James H Nicholes, a surgeon for the Royal Hospital for Sick Children in Glasgow, was done as early as 1909.2 He made a presentation to the British Medical Association, of 8,988 operations over a period of 10 years, though his paper did not discuss the morbidity and mortality. Ambulatory anesthesia again started gaining popularity in the early 1990s.3

The availability of safe and short-acting anesthetic drugs also contributed to this increasing number of ambulatory procedures. These facilities of ambulatory anesthesia may be attached to main hospital itself, or office based or free standing.

Children are excellent patients for ambulatory anesthesia as they are relatively healthy as compared to adults and free from chronic illness like hypertension, diabetes, etc.4 They also have care taking parents with them.

ADVANTAGES
The advantages of ambulatory anesthesia in pediatric practice include:

- Early return of the child to the comfortable and familiar surroundings
- Minimal parental separation
- Decreased nosocomial infections
- Decreased financial burden to the family
- Lack of dependence on the availability of hospital beds
- Shorter surgical waiting lists
- Reduced overheads and increased turnover to the hospital. The cost is expected to be 25–75% lesser than that of a similar inpatient procedure.5

LIMITATIONS
- Responsibility shifts from hospital to parents postoperatively
- In countries like India lack of adequate health education of the parents
- Lack of transport and healthcare facilities in villages
- Lack of rapid access to the hospital in case of emergency.

In view of these limitations extra care and definite policies should be established in carrying out the procedures on an outpatient basis so as to prevent surgical and anesthetic morbidity or mortality.

GUIDELINES
The guidelines or the prerequisites for the safe and effective outpatient anesthesia will be discussed under the following heads:
Selection of the procedures
Selection of the patient
Preoperative preparation
  - Preparation of the patients and family
  - Preanesthetic evaluation
  - Screening tests
Premedication
Anesthetic management
Discharge criteria
Postdischarge instructions
Complications and hospitalization
Evaluation of patient and family satisfaction.

Selection of the Procedure
The convenience and low overhead costs continue to attract more surgeries to be conducted in an ambulatory setting. Experts predict that in the years to come, nearly 80% of all surgeries performed in the United States will be on an ambulatory basis.6

In a country like India where access to emergency health care facilities and transport are problems, the number of procedures selected for ambulatory anesthesia is less than those of developed countries.

Appropriate procedures for ambulatory surgery are those associated with postoperative care that is easily managed at home, and with low rates of postoperative complications that require intensive physician or nursing management.7

List of ambulatory procedures quickly become outdated simply because the procedures which were excluded before become routine after a short time depending on the ambulatory settings and experience of the operating team.8

Length of surgery is not a main criterion for ambulatory procedures because there is little relationship between length of anesthesia and recovery.9

Procedures done on outpatient basis are given in Table I.

Selection of Patient
1. ASA physical status I or II
2. ASA physical status III as long as they are stable and well-controlled
3. The risk of complications can be minimized if preexisting medical conditions are stable, for at least 3 months before the scheduled operation.10
4. Even morbid obesity (BMI >40 kg/m²) is no longer considered an exclusion criterion for day-case surgery if adequate precautions are taken.11

<table>
<thead>
<tr>
<th>Table 1: Procedures commonly done on outpatient basis10</th>
</tr>
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<tbody>
<tr>
<td><strong>Especially</strong></td>
</tr>
<tr>
<td>General surgery</td>
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<td>Diagnostic/Therapeutic procedures</td>
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Special Risk Factors
1. Prematurity and age:
   a. The risk of apnea in healthy ex-premature infants is low (1%) and not zero.12
   b. Ex-premature infants (gestational age < 37 weeks) have an increased risk of cardiac and respiratory complications.13,14 Factors which increase risk of apnea should be excluded from the ambit of ambulatory anesthesia (Table 2).
   c. Risk for postoperative apnea usually persists until the 60th post-conceptual week.15
   d. Decision should be taken on case-to-case basis and also the policy of the hospital.
2. Upper respiratory tract infection (URTI): This is a frequently seen condition in children. Sometimes, it is difficult to get a symptom-free period in children. The presence of URTI increases the complications such as laryngospasm and bronchospasm which usually occur in the intra or immediate postoperative period; if managed properly will not have long-term consequences. In the authors’ experience, a patient can be taken up for the procedure only if the child does not have fever of more than 38°C and the lungs are clinically clear.

3. Chronic medical illness:
   a. Asthma: Well-controlled asthma or symptom-free period is ideal for outpatient anesthesia. Pretreatment with bronchodilators and nebulization during postoperative period may be necessary for some patients.
   b. Heart diseases: Patients with well-stabilized congenital heart disease do not have an increased risk of complication. The heart disease should be investigated and the general condition should be evaluated. Outpatient anesthesia may be given in stable, well-controlled patients. Antibiotic prophylaxis either orally two hours before or intravenously 30 minutes before the procedure is indicated.
   c. Seizures: Children with a history of febrile convulsions are not a contraindication for ambulatory procedures. Any other form of seizures also does not come as a contraindication when patient is on medication and well-controlled. Regular doses of medication for seizures should be taken with clear fluids before and after surgery.
   d. Diabetes mellitus: Type I diabetes mellitus is usually seen in children. The ambulatory procedures usually do not need more than 4 hours of preoperative fasting period for liquids and the child comes back to oral feeds after 2–4 hours post operatively. DM usually does not present with any morbidity for outpatient procedures unless there are associated comorbid conditions.
   e. Mental handicap: These children usually present for dental restoration procedures under general anesthesia. These children rely heavily on parents and hence outpatient anesthesia is preferable for them. Generally, they do not present with any complications postoperatively.
   f. Obesity and sleep apnea syndrome (OSAS): Obesity in children, is on the rise in India due to the change in the eating habits of the urban children. Obstructive sleep apnea (OSA) is a syndrome characterized by periodic, partial, or complete obstruction of the upper airway during sleep with episodic sleep-associated oxygen desaturation, episodic hypercarbia, and cardiovascular dysfunction. This is seen more commonly in younger children. If they have associated URTI or other medical conditions such as Down syndrome, craniofacial disorders and genetic disorders, then they are not suitable for outpatient anesthesia. Otherwise healthy children usually have an uneventful postoperative course and can be discharged the same day.

Exclusion Criteria for Outpatient Surgery

1. Major blood loss
2. Major surgery with significant fluid shifts and requiring postoperative monitoring
3. ASA III or IV and requiring complex or long-duration monitoring postoperatively
4. Morbidly obese patients who have OSAS (OSAS alone not a contraindication)
5. Infants
   - <56 weeks postconceptual age and <32 weeks post-gestation when born (56:32)
   - <54 weeks postconceptual age and <35 weeks post-gestation when born (54:35)
   - Ex-premature infants less than 60 weeks’ post-conceptual age requiring general endotracheal anesthesia
   - History of apnea
   - Complex congenital heart diseases and cyanotic congenital heart diseases
   - Anemia
   - No responsible adult at home to care for the patient after the discharge from the hospital.
Chapter 27: Ambulatory Anesthesia in Children

Preoperative Preparation

These include preanesthetic evaluation of the child, preparation of the patients and family, and screening tests. This will help:

1. To identify potential medical problems in advance, determine their etiology, and if indicated, initiate appropriate corrective measures. Certain disorders such as undiagnosed OSAS and congenital heart diseases may be relatively common in an ambulatory surgical population and these patients demand a vigilant perioperative care. It will also minimize the numbers of cancellations and complications.

2. In discussing the risks and benefits of anesthesia options with the parents and reducing their anxiety about the surgery and anesthesia.

3. Written and verbal instructions can be given regarding arrival time and place, fasting instructions, chronic oral medications and information concerning the postoperative course, and the need for a responsible adult to care for the patient during the early post-discharge period (<24 hours).

4. Parents are advised to inform the concerned doctors the night before admission, if there is any fever, severe URTI, so that the surgery may be rescheduled to some other time.

5. Planning the technique of anesthesia.

6. Side effects and complications can be discussed with the parents.

7. Basic minimum laboratory investigations can be conducted during the above period.

   The basic screening tests which are advised in pediatric patients are complete blood picture, routine urine examination, PT, APTT (for unsuspected bleeding disorders), HIV and HBsAg. Any other tests are as per the requirements.

   Fasting time should be adjusted to the scheduled time of procedure and the parent should be explained to adhere to the fasting recommendations and advised not to allow excessive fasting hours as it can lead to dehydration, discomfort and asymptomatic hypoglycemia. Fasting guidelines are given in Table 3.

   Examples of clear liquids include water, fruit juices without pulp, carbonated beverages, clear tea, and black coffee. Because nonhuman milk is similar to solids in gastric emptying time, the amount ingested must be considered when determining an appropriate fasting period.

   A light meal typically consists of toast and clear liquids. Meals that include fried or fatty foods or meat may prolong gastric emptying time. Additional fasting time (e.g. 8 hours or more) may be needed in these cases. Both the amount and type of food ingested must be considered when determining an appropriate fasting period.

Premedication

Generally infants younger than 6 months separate easily from parents. Older children may need oral premedication. Children between 6 months and 4 years benefit by the presence of the parent during induction. If there is an intravenous access preoperatively, the child may be given intravenous inducing agent in the lap of the parent and then wheeled into the operating room. Otherwise oral premedication is the choice. Intranasal, intramuscular and intrarectal routes are not routinely used.

a. Midazolam: Dose of oral midazolam is 0.5 mg/kg body weight of injectable preparation mixed in sugar flavored juices or oral flavored preparation at least 30 minutes before the procedure.

b. Ketamine: It can also be given orally at 6 mg/kg either alone or in combination with midazolam. Intramuscular ketamine may be used occasionally.

c. Analgesic creams like EMLA as occlusive dressing at two potential sites at least one hour prior is effective for the painless venepuncture.

General Anesthesia

1. Outpatient surgery requires the same basic equipment as inpatient surgery for delivery of anesthetic drugs, monitoring, and resuscitation. Standard intraoperative monitoring equipment for outpatient operations should include an ECG, blood pressure cuff, pulse oximeter, and capnograph.

2. The ideal outpatient anesthetic should have a rapid and smooth onset of action, produce intraoperative amnesia and analgesia, provide good surgical conditions with a short recovery period, and have no adverse effect.

3. Inhalational induction is widely used in pediatric practice, the most suitable agents being sevoflurane and halothane.
a. Sevoflurane has a pleasant smell, maintains hemodynamic stability, and is preferred for rapid, smooth induction and recovery.

b. Halothane also has smooth and rapid induction but may produce bradycardia and cardiac depression at higher concentrations. Intravenous atropine is needed to prevent or treat this complication.

c. Isoflurane is not generally tolerated due to its pungent smell and respiratory side effects. Desflurane though having rapid induction is not used as it is irritant and not well tolerated. It also produces airway complications and involuntary movements during induction.

4. Intravenous induction: This is the ideal choice for those having a secured IV access.

a. Propofol is the preferred agent for IV induction due to its rapid action and smooth recovery and shorter half-life. The problem of pain on injection is minimized by giving lidocaine intravenously directly or mixing with propofol.

b. Thiopentone also can be used for induction but the recovery time is prolonged as its half-life is more than propofol.

5. Maintenance of anesthesia: It can be done by using short-acting anesthetic agents, analgesics and muscle relaxants.

a. The inhalational agents which can be used are sevoflurane, desflurane, isoflurane and halothane.

b. Propofol is the agent of choice for maintenance, if total intravenous anesthesia (TIVA) is planned. The infusion rates can be as high as 300–500 µg/kg/min. The advantage of propofol is rapid and smooth recovery and reduced incidence of PONV. Analgesics which can be used include fentanyl and remifentanil.

Airway Management

There are many ways of airway management and are similar to inpatient anesthesia.

- For short procedures holding the mask is best
- For long procedures LMA is preferable especially in asthmatics and those having recent history of URTI
- There is still a controversy regarding preference of uncuffed or cuffed endotracheal tubes for intubation. If a Microcuff tube is available, it is preferred.

Regional Anesthesia

Regional anesthesia along with general anesthesia or sedation allows the child to have rapid recovery and pain free postoperative period. The type of block chosen should be such that the motor block should be minimal or the child has rapid recovery of motor function. Many types of regional and local anesthetic techniques can be used.

- Epidural and spinal anesthesia is preferred for older children
- Caudal block can be used for procedures below the level of umbilicus especially in children less than six years. Both bupivacaine and ropivacaine can be used and the dose used should not exceed the toxic limit. Use of additives in regional anesthesia for outpatient procedures is still controversial.
- Penile block is extensively used for circumcision and distal penile hypospadias
- Ilioinguinal and iliohypogastric nerve blocks are commonly used for herniotomy in children
- Upper and lower limb blocks give an excellent pain relief and should be given for peripheral limb surgeries
- Intravenous regional anesthesia for upper limb can be given in older children
- Local infiltrations are given wherever it is possible.

Monitored Anesthesia Care

The combination of local anesthesia with intravenous sedative and analgesic drugs is extremely popular in the ambulatory setting.

Many different sedative-hypnotic drugs have been used during monitored anesthesia care. The most commonly used sedation techniques are a small dose of midazolam (0.03–0.1 mg/kg) or analgesic dose of ketamine (0.5 to 1 mg/kg), or propofol followed by a propofol infusion at 25 to 100 µg/kg/min.

Discharge Criteria

- Early recovery is the time interval during which patients emerge from anesthesia, recover control of their protective reflexes, and resume early motor activity (Aldrete score).
- Intermediate recovery starts in recovery room – patient begins to ambulate, drink fluids, void, and prepare for discharge.
- Late recovery period starts when the patient is discharged home and continues until complete functional recovery is achieved and the patient is able to resume normal activities of daily living.
  1. Anesthetics, analgesics, and antiemetics can affect the patient's early and intermediate recovery
  2. The surgical procedure has the highest impact on late recovery
3. Before ambulation, patients receiving a central neuraxial block should have normal perianal (S4–5) sensation, have the ability to plantar flex the foot, and have proprioception of the big toe.

4. Postanesthesia discharge scoring (PADS) system: This is helpful in making the decision to discharge the child easy (Table 4).

**Post-discharge Instructions**

- Child should go home accompanied by the parent, who should not be the driver of the car.
- Written instructions are to be given to the parent about the home care, the operation and the sequel the following days.
- The parent should be explained about the diet, care of the operation site, physical activity of the child and pain relief.
- Telephone numbers of the hospital, surgeon and anesthetist are given for any emergency.

**Complications and Hospitalization**

- Usually ambulatory anesthesia is safe except for minor incidences like nausea and vomiting, pain and rarely postoperative croup. These generally subside over a period with oral medication and do not require hospitalization.
- There may also be some transient behavioral changes and sleep disturbances.

**Evaluation of Patient and Family Satisfaction**

Follow up of the patient and evaluation and parent satisfaction add to the experience and help to modify the techniques for better quality of ambulatory anesthesia practices.

**GUIDELINES FOR A GOOD AMBULATORY SURGICAL FACILITY**

- Employment of appropriately trained and credentialed anesthesia personnel.
- Availability of properly maintained anesthesia equipment appropriate to the anesthesia care being provided.
- Complete documentation of the care provided as that required at other surgical sites.
- Use of standard monitoring equipment according to the ASA policies and guidelines.
- Provision of a PACU or recovery area that is staffed by appropriately trained nursing personnel and provision of specific discharge instructions.
- Availability of emergency equipment (e.g. airway equipment, cardiac resuscitation).
- Establishment of a written plan for emergency transport of patients to a site that provides more comprehensive care should an untoward event or complication occur that requires more extensive monitoring or overnight admission of the patient.
- Maintenance and documentation of a quality assurance program.
- Establishment of a continuing education program for physicians and other facility personnel.
- Safety standards that cannot be jeopardized for patient convenience or cost savings.

**LEARNING POINTS**

- The successful management of children undergoing outpatient surgery requires that the anesthetist understand and be actively involved in all aspects of management.
- Specific criteria for selection of patients, careful choice of agents and techniques for operative anesthesia, and use of postoperative analgesia should ensure rapid and comfortable emergence from anesthesia and early discharge from the hospital.

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**Table 4: Postanesthesia discharge scoring**

<table>
<thead>
<tr>
<th>Vital signs</th>
<th>Score</th>
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<tbody>
<tr>
<td>Within 20% of preoperative baseline</td>
<td>2</td>
</tr>
<tr>
<td>20–40% of preoperative baseline</td>
<td>1</td>
</tr>
<tr>
<td>40% of preoperative baseline</td>
<td>0</td>
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</table>

<table>
<thead>
<tr>
<th>Activity level</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Steady gait, no dizziness, consistent with preoperative level</td>
<td>2</td>
</tr>
<tr>
<td>Requires assistance</td>
<td>1</td>
</tr>
<tr>
<td>Unable to ambulate/assess</td>
<td>0</td>
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</table>

<table>
<thead>
<tr>
<th>Nausea and vomiting</th>
<th>Score</th>
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</thead>
<tbody>
<tr>
<td>Minimal: mild, no treatment needed</td>
<td>2</td>
</tr>
<tr>
<td>Moderate: treatment effective</td>
<td>1</td>
</tr>
<tr>
<td>Severe: treatment not effective</td>
<td>0</td>
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<table>
<thead>
<tr>
<th>Pain</th>
<th>Score</th>
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<tr>
<td>VAS = 0–3 the patient has minimal or no pain prior to discharge</td>
<td>2</td>
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<tr>
<td>VAS = 4–6 the patient has moderate pain</td>
<td>1</td>
</tr>
<tr>
<td>VAS = 7–10 the patient has severe pain</td>
<td>0</td>
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<table>
<thead>
<tr>
<th>Surgical bleeding</th>
<th>Score</th>
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<tbody>
<tr>
<td>Minimal: does not require change</td>
<td>2</td>
</tr>
<tr>
<td>Moderate: required up to two dressing changes with no further bleeding</td>
<td>1</td>
</tr>
<tr>
<td>Severe: required three or more dressing changes and continues bleed</td>
<td>0</td>
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**Note:** Max 10 Score ≥9 fit for discharge.
REFERENCES

INTRODUCTION

Pediatric orthopedics is a rapidly progressing branch because of evolution of new techniques and modern instruments. Children undergoing orthopedic procedures range from otherwise healthy children to those with complex abnormalities. They may undergo surgery for fixation of a simple fracture or correction of severe complex defects where the orthopedic problem is one of many other issues.

Pediatric Orthopedic conditions can be classified as follows:

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Conditions</th>
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<tbody>
<tr>
<td>Congenital</td>
<td>Club foot, congenital dislocation of the hip (CDH), radial club hand, Scoliosis</td>
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<tr>
<td>Neurological</td>
<td>Cerebral palsy, spina bifida, etc.</td>
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<tr>
<td>Muscle Dystrophies</td>
<td>Duchenne's muscular dystrophy, myotonia congenita, myotonic dystrophy, progressive muscular atrophy, ppolimyelitis, scoliosis</td>
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<tr>
<td>Growth disorder</td>
<td>Achondroplasia</td>
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<td>Infections</td>
<td>Osteomyelitis, septic arthritis</td>
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<tr>
<td>Immune</td>
<td>Juvenile rheumatoid arthritis</td>
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<tr>
<td>Metabolic</td>
<td>Mucopolysaccharidoses, renal osteodystrophy, rickets and osteomalacia, juvenile osteoporosis, gaucher's disease, etc.</td>
</tr>
<tr>
<td>Other Conditions</td>
<td>Arthrogryposis multiplex congenita (AMC), osteogenesis imperfecta (OI), gargoylism, neurofibromatosis</td>
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<tr>
<td>Idiopathic</td>
<td>Scoliosis</td>
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<tr>
<td>Bone cysts and tumors</td>
<td>Benign: Osteochondroma, bone cysts, granulomata Malignant: Sarcoma</td>
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</table>
• Severe postoperative pain, either due to the magnitude of the surgery or because of muscle spasm (e.g. muscle release operations)
• Frequent use of intraoperative X-rays or image intensifiers can cause radiation hazards to patient and or personnel.

PREOPERATIVE EVALUATION AND PREMEDICATION

Anesthesiologists should perform usual preoperative assessment in children keeping above issues of major concern in mind. Most of the children are healthy. Anemia is a relatively common problem in children of low-socioeconomic class and infants born prematurely. Prematurity is an important cause of intraoperative hypoxemia and postoperative apnea. In children with upper respiratory infections, there is increased incidence of laryngo/bronchospasm, intra- and postoperative desaturation. To decrease these untoward incidences they should be treated with antibiotics and nebulization. Syndromic children should be thoroughly examined for airway abnormality and involvement of other systems.

Minimal laboratory tests for minor surgeries are complete blood count and urine analysis. X-ray chest should be advised when respiratory symptoms are present and for major procedures. Apart from these, other investigations such as serum electrolytes and creatinine, clotting studies and blood sugar (in adolescents) should be done for major procedures. Electrocardiogram is advised, if clinical situation warrants. Pulmonary function tests should be done to test the lung capacity in cases of scoliosis. If it is not possible, arterial blood gases should be done. For major procedures where significant blood loss is expected, blood and blood products should be reserved.

Children on long-term drug therapy like anti-convulsants, steroids, chemotherapy, insulin, etc. should be advised for continuation or discontinuation as appropriate preoperatively. Also their side effects and drug interaction with anesthetic agents should be kept in the mind.

Premedication is an important part of preoperative preparation because children come frequently for correction of deformities. Parental separation and fear of pain causes tremendous anxiety. Children with impaired intellectual communication may be a great problem. Midazolam, oral (0.5 mg/kg) or nasal (0.3 mg/kg) 20–30 minutes before surgery can be administered to allay the anxiety.

TOURNIQUETS

They are used for peripheral limb surgeries to reduce blood loss. The tourniquet should be applied very carefully to the proximal part of the limb at the greatest circumference because the muscle bulk at that site is the greatest, and hence it affords a greater protection against nerve injury. The tourniquet should be first deflated and smoothened out. Adequate padding should be done at that site. Any solution applied to the skin must not be allowed to run underneath the tourniquet as it may lead to skin burns. The limb is exsanguinated before a pneumatic tourniquet is applied with pressure exceeding arterial systolic pressure. The surgeon should be informed when tourniquet has been on for 1 hour or longer. Tourniquet inflation pressure should be kept to the minimum effective pressure. Inflation time should be kept to a minimum. The patient should be monitored continuously with special consideration to parameters like hypertension and tachycardia as a surrogate of tourniquet pain.

Common pediatric orthopedic conditions are discussed below.

CONGENITAL DEFORMITIES—LOWER LIMB

Clubfoot [Congenital Talipes Equino Varus (CTEV)]

Overview

The incidence of clubfoot is 1 in 1000 live births with male: female ratio of 1:2. Other associated problems are neuropathies and myopathies such as myelodysplasia, cerebral palsy, arthrogryposis, spinal muscular atrophy and muscular dystrophy.

Pathophysiology

Clubfoot is idiopathic in majority of cases. Various causes are vascular deficiencies, environmental factors, in utero positioning, abnormal muscle insertion and genetic factors. Secondary clubfoot is seen in paralytic disorders like polio, spina bifida, and myelodysplasia or in cases of arthrogryposis multiplex congenita.

Clinical Presentation

Clinical features include deformed talus (i.e. ankle) and calcaneum (i.e. heel) such that the foot points downward (i.e. equinus), inward (i.e. varus) while the forefoot curls.
towards the heel (i.e. adduction); and tightening of the Achilles tendon (Fig. 1).

Nonsurgical Procedures
Ponseti's method—It is done immediately after birth, involves serial manipulation and casting with Plaster of Paris (POP) or soft fiberglass.3,4

Anesthesia Concerns
• These corrective procedures are performed in neonatal age
• Spina bifida causing clubfoot can pose problems with regional blocks and difficult positioning.

Anesthesia for Surgical Treatment
• Manipulations and percutaneous tendo achillis tenotomies and subsequent serial change of POP casts are done on day care basis under general anesthesia with mask using inhalational agent and narcotics and propofol. For pain relief local anesthetic infiltration of 0.25% of Bupivacaine is done.
• Soft tissue release and lengthening of the tight tendons/ligament around the joints are done under general anesthesia using face mask or supraglottic airway devices and caudal block. In Cincinnati approach, soft tissue release is done in prone position, hence to secure airway endotracheal intubation is preferred.
• For bony procedures, such as osteotomies/arthrodesis, single shot caudal or lumbar epidural along with GA can be given. Epidural catheter can be placed for intermittent or continuous infusion of local anesthetics along with additives.
• Paracetamol can be given in the form of intravenous bolus/infusion or suppositories for multimodal analgesia.

Congenital Dislocation of Hip
Also known as developmental dysplasia of the hip (DDH).

Overview
DDH refers to spontaneous dislocation of the hip occurring before, during or shortly after birth. The incidence is 1.5 to 20 of 1,000 live births.4 It is more commonly seen in females, first born children and newborns with breech presentation.
The two variants are:
• Teratologic hip dysplasia is associated with various syndromes (e.g. Ehler Danlos, Down syndrome, arthrogryposis)
• Neuromuscular hip dysplasia refers to weakness and/or spasticity in some or all of the hip muscle groups (e.g. in spina bifida, cerebral palsy).

Pathophysiology
In DDH, the anatomical relationship between the femoral head and the acetabulum is incorrect. Persistently poor alignment of the ball and socket during the development of the hip joint can lead to premature arthritis.6,7

Clinical Presentation
The leg may turn outward, and appear shorter on the side of the dislocated hip, hence the space between the legs may look wider than normal. The folds in the skin of the thigh or buttocks may appear uneven (Fig. 2).

Anesthesia Concerns
• DDH can be associated with arthrogryposis multiplex congenita (AMC)
Associated cardiac anomalies should be given due consideration if DDH is associated with Down’s Syndrome.

**Surgical Procedures and Anesthesia**

- The goal of treatment is to relocate the head of the femur into the acetabulum
- Closed reduction and hip spica in infants is done under general anesthesia under mask using inhalational agent and/or propofol with small dose of short-acting narcotics, if required
- Open reduction—If the above option fails or the child is much older when DDH is diagnosed, acetabular reconstruction with femoral osteotomy is performed. Supraglottic airway devices or conventional balanced anesthesia with endotracheal intubation and single shot, continuous caudal or lumbar epidural is the best choice of anesthesia.8,9

### CONGENITAL DEFORMITIES—UPPER LIMB

#### Radial Club Hand

**Overview**
The incidence of radial club hand is estimated to be 1 in 30,000 to 1 in 100,000 live births. It is bilateral in 38% to 58% of cases.

**Clinical Features**
Deformity includes significant shortening of the forearm, and generalized underdevelopment of the extremity. The thumb is usually absent or hypoplastic and the ulna is typically of normal length and bowed due to the absence of radius (Fig. 3A). 40% of cases may have associated thrombocytopenia and anemia.

**Treatment**
Soft tissue distraction followed by reconstructive wrist stabilization and pollicization is the most acceptable treatment. It is usually done at age of 9 months (Fig. 3B).

**Anesthesia**
General anaesthesia with brachial plexus block by axillary or infraclavicular approach is preferred.

#### Cerebral Palsy

**Overview**
Cerebral palsy (CP) is a nonprogressive disorder of posture and movement often associated with epilepsy and abnormalities of speech, vision and intellect resulting from a defect or lesion of the developing brain.10

**Pathophysiology**
Cerebral palsy occurs as a result of interruption of oxygen supply to fetus or brain asphyxia. In preterm infants, the damage is predominantly in white matter and in full term infants damage is in gray matter and the brainstem nuclei.10

Figs 3A and B: (A) Child with left radial Club hand; (B) External fixator for wrist stabilization
Clinical Features
- Isolated neurological abnormality
- Severe neurological deficit associated with cognitive impairment, sensory deficits, seizures, communication disorders, behavioral and emotional problems
- Muscle spasticity/‘scissor’ gait
- Joint pain when walking and sitting due to muscle contracture (Fig. 4).

Swedish Classification for CP
1. Spastic (70%): Most common type. Lesion in cerebrum. It includes quadriplegia, diplegia, hemiplegia.
2. Dyskinetic (10%): Lesion in basal ganglia. It includes dystonia (twisting position of torso and extremities), athetosis (slow, purposeless, distal movements), and chorea (quick, jerky, movements of proximal extremity). Often intellect is normal, but difficulties with communication exist.
3. Ataxic (10%): Lesion in cerebellum. It includes intention tremor and head tremor, i.e. titubation, and speech abnormalities.
4. Mixed (10%): Lesion in cerebrum and cerebellum, e.g. spastic athetoid.

Note: Complex cases may DEFY Classification

Common Surgical Procedures
Major multilevel surgery involving tenotomies/osteotomies, soft tissue release or tendon transfers are done. Many times Botulinum toxin injections are given.

Anesthetic Issues

Neurological deficiencies, cognition, and communication:
- Up to 50% have either focal or generalized forms of epilepsy
- Visual and auditory impairment and abnormal touch and pain perception.

Respiratory System
- Respiratory muscle hypotonia, chronic respiratory infections, respiratory failure.

Gastrointestinal System
- Esophageal dysmotility, abnormal lower esophageal sphincter tone, and spinal deformity leading to gastroesophageal reflux.

Airway
- Increased incidence of temporomandibular joint dislocation due to muscle spasticity.

Musculoskeletal and Skin
- Fixed flexion deformities of the limbs and trunk as a result of muscle contractures, causing difficulties in attaining intravenous access and positioning
- Cerebral palsy muscle contracts poorly when surgically incised. This can result in significant blood loss during major surgery.

Anesthetic Management
- Good premedication, sedation or general anaesthesia along with regional blocks are mainly used.
- Assessment of postoperative pain is difficult because of communication problems and mental retardation. Hence, it is preferable to use multimodal approach, i.e. narcotics, local or regional techniques, paracetamol, and anti-inflammatory drugs for postoperative pain relief.

MUSCULAR DYSTROPHIES

The muscular dystrophies are a group of genetically determined, progressive diseases of skeletal muscle. The commonest dystrophies are Duchenne’s, Becker’s, Scapulohumeral—"Limb girdle", Myotonia congenita, Dystrophia myotonica, Paramyotonia congenita, etc.

Muscular dystrophies lead to progressive weakness and debility. In later stages, respiratory muscle weakness leads to decrease in respiratory reserve. Operative risk is very high if vital capacity is 30% of normal. Cardiac changes include right ventricular hypertrophy, sinus tachycardia, mitral valve prolapse, and diminution of the QRS wave. An echocardiographic examination is recommended yearly in these patients. There may be associated cardiomyopathy, hence it is safer to avoid suxamethonium. It is important to remember these patients have tendency to bleed more than normal even if hypotensive techniques are used. It may be because of abnormality of blood vessels.
GROWTH DISORDER

Achondroplasia

This is a syndrome of disproportionate short stature also known as Dwarfism.\textsuperscript{15}

Overview

Achondroplasia is the most common form of dwarfism with an incidence of 150 per 1 million live births.

Clinical Presentation (Figs 5A and B)

- Normal trunk and short limbs (rhizomelic) dwarfism
- Frontal bossing, small and depressed nasal bridge
- Waddling gait due to hypotonia
- Normal intelligence but delayed motor milestones
- Lumbar stenosis
- Excessive lordosis (short pedicles with decreased interpedicular distances).

Surgical Procedures

- Osteotomy and fibular shortening
- Limb lengthening of up to 40–50% per segment
- Spine decompression may be required in children with cord compression due to spinal stenosis.

Anesthesia Issues

- Due to facial deformity, face mask fit may be inadequate
- Regional blocks specially central neuraxial blocks may be difficult because of spinal stenosis.

INFECTIONS

Osteomyelitis

Overview

Osteomyelitis is an infection of the bone and bone marrow most frequently occurring in infants and young children, although no age group is immune. Patients with reduced immunity, sickle cell disease, and organ transplant patients are known to be particularly susceptible to infection.

Clinical Features

- The child may be toxic and febrile. Movements of involved limb are painful.

Surgical Treatment

- Prompt surgical decompression of bone is essential to avoid permanent sequelae.

Anesthesia

- GA with neuraxial or appropriate peripheral nerve block
- Coagulopathy associated with infection increases bleeding tendency and limits the use of regional blocks.

METABOLIC DISORDERS

The common metabolic disorders are listed below:

- Mucopolysaccharidosis
- Renal osteodystrophy
- Rickets and osteomalacia
- Juvenile osteoporosis
- Thyroid disorders
- Gaucher’s disease
- Hypervitaminosis
- Hypophosphatasia
- Parathyroid disorders
- Scurvy.

Mucopolysaccharidosis

Mucopolysaccharidosis (MPS) (i.e. Hurler’s syndrome [MPS I-H], Hunter’s syndrome [MPS II], Morquio’s syndrome [MPS IV], and Scheie’s syndrome [MPS V]) are rare conditions, but pose significant anesthetic challenges.

Pathophysiology

Mucopolysaccharidosis is genetically determined disease in which mucopolysaccharides are stored in tissues in abnormal quantities due to deficiency of specific lysosomal enzymes.\textsuperscript{15}
Clinical Presentation and Anesthetic Challenges (Figs 6A and B)

Upper airway abnormalities
- Macroglossia/micrognathia, temporomandibular joint immobility, tonsillar and adenoidal hypertrophy
- Narrow nasopharynx, laryngomalacia, short neck.

Pulmonary dysfunction
- Restrictive lung disease, pulmonary hypertension, obstructive sleep apnea.

Cardiovascular dysfunction
- Congenital heart disease, acquired valvular disease.

Neurologic complications
- Atlanto-occipital instability, intracranial hypertension.

Hematologic dysfunction
- Disorder of platelet aggregation.

Surgical Procedures
Limb-lengthening techniques, cervical decompression, joint replacement, limb realignment, and bone marrow transplantation for patients with mucopolysaccharidosis are but a few of the procedures that are increasingly being performed on these patients.

Renal Osteodystrophy

Pathophysiology
- Glomerular damage leading to phosphate retention and a reduction in production of 1,25-dihydroxyvitamin D. Gut absorption of calcium is reduced
- Marked secondary hyperparathyroidism results; end result is a combination of rickets and secondary hyperparathyroidism.

Clinical Presentation
- Knock-knees.

Anesthesia Concerns
- Elevated creatinine and blood urea nitrogen.
- Renal excretion of drugs might be impaired.
- Function of cholinesterase may be impaired, resulting in prolonged respiratory muscle paralysis if neuromuscular blocking agents are used.

Rickets

Overview
The term rickets, implies a decrease in calcium, phosphorus, or both, which is of such magnitude that it interferes with epiphyseal growth and mineralization.

Pathophysiology
Decreased vitamin D causes decreased intestinal absorption of calcium. Hypocalcemia in turn leads to secondary hyperparathyroidism resulting in decreased tubular reabsorption of phosphorus leading to hypophosphatemia. Thus, a recurrent cycle ensues.

Clinical Presentation
- Delayed walking and bowing of legs (Fig. 7).
Anesthetic Concerns
- Complications due to hypocalcemia (QT widening, laryngospasm)
- Osteoporosis leading to recurrent fractures even during positioning for laryngoscopy and intubation.

OTHER CONDITIONS

Arthrogryposis Multiplex Congenita (AMC)

Overview
Arthrogryposis multiplex congenita is a condition characterized by multiple joint contractures found throughout the body at birth. AMC may be associated with multiple congenital anomalies. The incidence is 1 in 3,000 live births.

Pathophysiology
Abnormalities, such as neurological or connective-tissue disorders or physical restriction, prevent normal development of joints in embryonic life leading to AMC. The muscles are partially or completely replaced by fat/fibrous tissue. The most common form, accounting for 40% of cases, is amyoplasia. Associated syndromes:
1. Freeman-Sheldon syndrome.
2. Osteochondrodysplasias.
3. Chromosomal disorders.

Clinical Presentation (Fig. 8)
- Upper limb and lower limb deformities
- Facial deformities (including asymmetry, flat nasal bridge hemangioma, micrognathia, trismus).

Nonsurgical Care
- Physical therapy to improve the range of motion and to stretch surrounding tissues.
- Serial casting after physical therapy with weekly changes of cast and gentle manipulation.

Surgical Procedures
- To correct soft-tissue contractures and joint deformities
- To stabilize dislocated hips, spinal deformities, and correct foot deformities.

Anesthesia Issues
- **Difficult airway**: Micrognathia, high-arched palate, cervical spine instability, and airway worsening with age.
- **Malignant hyperthermia (MH)**: Avoid triggering agents, such as Succinylcholine and inhalational anesthetic agents.
- **Difficult IV access**: Reduced subcutaneous tissue and tense skin.
- Regional anesthesia although technically difficult due to anatomical abnormalities, caudal analgesia has been successfully used for postoperative pain in Freeman-Sheldon syndrome. Fascia iliaca block for muscle biopsy in patients with AMC and central as well as peripheral blocks are used extensively.

Fig. 7: Bowing of legs in Rickets

Fig. 8: Arthrogryposis multiplex congenita
Osteogenesis Imperfecta (OI)

Overview
Osteogenesis imperfecta (OI) is a heritable disorder of bone formation characterized by bone fragility and low-bone mass. It was first termed as Lobstein’s disease. Later, the name was changed to osteogenesis imperfecta, which means imperfect bone formation. Incidence is one in 10,000 births.27,28

Pathophysiology
Osteogenesis imperfecta (OI) is associated with abnormalities in the synthesis or structure of type I collagen. This leads to increased bone fragility.29

Clinical Presentation (Figs 9A and B)
- Fractured bones following even trivial trauma
- Blue or gray sclera, thin skin, opalescent teeth (dentinogenesis imperfecta)
- Hearing loss, hyperextensible joints (hypermobility), cardiac valve anomalies, and thoracic and spinal deformities.

Treatment
Goals
- Controlling the symptoms, maximizing independent mobility, and developing optimal bone mass and muscle strength
- Care of fractures, extensive surgical and dental procedures, and physical therapy
- Cyclic bisphosphonate therapy.

Surgical Procedures
- Fixation of fracture (most common).

Anesthetic Considerations30-32
Positioning
- The table should be padded with careful consideration to pressure points, avoid overextension during positioning
- During insertion of an intravenous catheter, light pressure should be applied above the site of insertion.

Airway
- A short neck, a protruding mandible, proportionately large tongue and the presence of a pigeon chest makes airway difficult
- A delicate mandible and the presence of dentinogenesis imperfecta can lead to jaw fracture and easily dislodged teeth
- Overextension of the cervical spine can lead to odontocervical dislocation or fracture and must be avoided
- Basilar invagination, can distort the airway anatomy, increases risk of brainstem damage
- Fiberoptic intubation is ideal in such circumstances.

Spine deformities and pulmonary mechanics
- Kyphoscoliosis and thoracic cage deformity leads to a restrictive defect of respiration.

Cardiac anomalies
- Decreased ventricular stiffness and major alterations in stress-strain relationship
- ASD, VSD, PDA, aortic dissection, left ventricular rupture, and aortic or mitral valve incompetence, aortic root dilatation.33,34

Hemopoietic anomalies
- Platelet dysfunction, increased bleeding tendency.35,36

Hyperthermia
- Hyperthermia is thought to be a result of a hypermetabolic state of which the pathogenesis is unknown.37

Anesthesia
- Nondepolarising muscle blockers preferred as fasciculations caused by succinyl choline can produce fractures
- Regional anesthesia is preferred.

SCOLIOSIS

Scoliosis refers to a lateral curvature of the spine. Kyphosis, another structural deformity refers to an anterior flexion to the spinal column (Figs 10A and B).
### Classification\(^{38,39}\)

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Idiopathic</td>
<td>About 70% of cases, strong genetic component</td>
</tr>
<tr>
<td>Congenital</td>
<td>Associated with other defects, particularly renal and cardiac defects</td>
</tr>
<tr>
<td>Neuromuscular</td>
<td>Associated with upper or lower motor neuron diseases</td>
</tr>
<tr>
<td>Neurofibromatosis</td>
<td>Von Recklinghausen's Disease</td>
</tr>
<tr>
<td>Trauma</td>
<td>Vertebral fracture, irradiation</td>
</tr>
</tbody>
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### Pathophysiology

#### Respiratory System
- Decrease in lung volume and a restrictive pattern in thoracic scoliosis
- Elevated pulmonary vascular resistance and pulmonary hypertension secondary to chronic hypoxemia/Cor-pulmonale.

#### Cardiovascular
- Mitral valve prolapse.

### Measurement of Curve Magnitude

It is done by Cobb's method. The measurement is done by selecting the end-vertebrae of the curve deformity (vertebrae at the upper and lower limits of the curve). Lines are drawn perpendicular to the end plates of the vertebrae. The angle formed at the intersection of these lines is the Cobb's angle. The treatment depends on magnitude of the curve. Curve >45° requires surgical intervention (Fig. 11).

### Clinical Manifestations

- Uncorrected scoliosis results in curve progression, cosmetic deformity, back pain and cardiorespiratory compromise
- The worse the curve and the more compromised the cardiorespiratory function, the greater the risk for perioperative morbidity and mortality
- Pulmonary dysfunction is exacerbated by frequent respiratory infections, predilection to aspiration, and impaired ability to clear pulmonary secretions\(^{39-42}\)

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**Fig. 11:** Cobb's angle measurement.
Preoperative Assessment and Preparation

Apart from general preoperative assessment, detailed examination of following systems should be done.38,40

Respiratory Function
- Treating any reversible cause of pulmonary dysfunction, including infection, with physiotherapy and nebulized bronchodilators as indicated
- Pulmonary Function Testing (PFT): If preoperative vital capacity is less than 30–35% of predicted and any blood gas abnormality, postoperative ventilation is likely to be required.

Cardiovascular System
- ECG and echocardiography to assess left ventricular function and pulmonary arterial pressures.

Neurological System
- A full neurological assessment of the patient should be made preoperatively to rule out coexisting neurological disease and impairment.

Anesthetic Management
- Conventional balanced anesthesia is required. Airway secured with preferably nonkinkable endotracheal tube
- Blood loss in this procedure may exceed 50% of patient’s total blood volume and is directly related to the number of vertebrae fused and surgical time. Hypotensive anesthesia is used to reduce intraoperative blood loss by using various drugs like using narcotics, propofol, alpha- and beta-blockers. Continuous monitoring of blood pressure and peripheral perfusion is essential. Two large bore IV access and central venous catheterization along with intra-arterial cannulation are recommended
- Continuous urine output and temperature monitoring is essential and meticulous thermoprotective strategies to prevent intraoperative hypothermia should be used
- Careful positioning with the goals of protecting the patient from neurologic damage and optimizing chest ventilation and venous return
- Apart from standard monitoring, blood sugar and blood gases are monitored.39,41,43

Neurologic Injury During Surgery
- Direct contusion of the cord by implant or instrument
- Reduction of spinal cord blood flow by stretching or compression of vessels or direct interruption of radicular blood flow
- Distraction injury of the spinal cord
- Epidural hematoma.40,44
  - To prevent spinal cord injuries spinal cord monitoring is essential.

Methods of Spinal Cord Monitoring

Wake-up Test
The wake-up test allows intraoperative emergence sufficient to test lower limbs’ gross motor function, but not sensory functions.44 Patient is counseled and explained regarding the test preoperatively. 30–45 minutes before the test muscle relaxants are withheld and the inhalational agents are stopped. A syringe loaded with propofol is kept ready in case of stormy emergence. Patient is asked to move feet/hands. Depth of anesthesia is increased and muscle relaxants administered as soon as the test is over.

Clonus Test
Clonus can normally be elicited in patients with intact spinal reflexes and lack of central inhibition. Eliciting clonus of the ankles is attempted just prior to wakeup. If clonus is present spinal cord integrity is assumed.43

Somatosensory Evoked Potential (SSEP) Monitoring
It monitors dorsal sensory pathways. This test may be adversely affected by changes in the anesthetic level and perfusion.38

Motor Evoked Potential (MEP) Monitoring
This involves recording of spinal cord motor tract.39

Other Complications
- Blood loss and coagulopathy
- Venous air embolism
- Visual loss
- Superior mesenteric artery syndrome
- Problems with fluid management
• Respiratory complications (Atelectasis and deterioration in respiratory function).

**Postoperative Pain Management**
• Systemic opioids by continuous infusion or using patient controlled analgesia (PCA) and NSAIDs are the major components
• Intrathecal morphine has been shown to be associated with reduced intraoperative bleeding and better postoperative analgesia than systemic morphine alone in scoliosis surgery
• Epidural analgesia using low dose opioids also provides excellent pain relief.

**BONE CYSTS AND TUMORS**
Simple bone cyst or aneurysmal bone cysts can present as bony lumps in children (Fig. 12). Benign bone cysts, such as osteoid osteoma or osteochondroma are very painful and they require excision. Malignant bone tumors like Ewing’s sarcoma and osteosarcoma require extensive excision. The blood count should be checked as these patients are on chemotherapy. These patients may be resistant to nondepolarizing muscle relaxants (Fig. 13).

**TRAUMA**
Children commonly present with fractures which can be simple or associated with major trauma. Simple fractures need manipulation and plaster. Simple fractures do not usually need to be treated urgently but if they are treated as an emergency, inadequate fasting times with risk of vomiting and aspiration should be kept in mind. Children with fracture associated with major trauma should be operated after hemodynamic stabilization.

**COMMONLY USED REGIONAL BLOCKS FOR ORTHOPEDIC PROCEDURES**
Regional blocks have revolutionized the perioperative anesthetic management of pediatric orthopedic surgery. Except for fractures where nerve block may mask pain from compartment syndrome, blocks are extremely useful as they reduce the requirement of general anesthesia and use of muscle relaxants. Another advantage of regionals is—it gives good pain relief, which can be continued in the postoperative period by placing catheter and giving continuous infusion of local anesthetic agents. With full doses of NSAIDs and paracetamol, they enable minimal opioids to be used in most procedures, hence reduces incidence of postoperative nausea and vomiting.

Most of the blocks that are used in adults can be used in children and even in infants with only minor modification. The use of a peripheral nerve stimulator and ultrasonography enhances accuracy of regional blocks.

Regional blocks can be divided into:
1. **Central Neuraxial Blocks**—a) Epidural: Caudal/Lumbar/Thoracic approach, b) Spinal.
2. **Peripheral Nerve Blocks**—a) Upper limb: Brachial plexus block (Interscalene, Supraclavicular, Infraclavicular, Axillary approach), b) Lower limb: Sciatic/Femoral/Popliteal/Ankle block.
Central Neuraxial Blocks

Indications: Lower extremity surgeries.

Caudal Epidural Block

It can be given as single shot for minor procedures. In a neonate or infant the epidural catheter can be placed through the sacral hiatus and threaded up to a higher level. The catheter can be tunnelled to avoid soiling when it is to be left in situ for a prolonged period.

Position: Lateral

Landmark: Sacral hiatus which forms an equilateral triangle with two posterior superior iliac spines (Fig. 14).

Technique: Sacral hiatus is identified. A 22G/23G needle inserted at 45° between the two cornuae until a distinct ‘pop’ or give is felt.

Drug/Dosage: 0.25% bupivacaine up to a maximum of 20 mL can be given after negative aspiration test for CSF and blood (Armitage Formula).
- 0.75 mL/kg for a lumbosacral block
- 1.0 mL/kg for a thoracolumbar block
- 1.25 mL/kg for a midthoracic block.

Lumbar Epidural Block

Position: Lateral or sitting in cooperative and adolescent patient (Fig. 15).

Technique: (a) Appropriate space is identified, (b) Tuohy’s needle 18G/19G or 20G are used according to age of the child, (c) Epidural space is identified by loss of resistance using normal saline, (d) Epidural catheter inserted to appropriate depth, (e) Approximate depth(in cm) = age(in years)/10.

Drug dosages: (a) 0.25% bupivacaine or 0.2% ropivacaine along with additives, (b) Single bolus 0.5 mL/kg, (c) Continuous infusion 0.1–0.3 mL/kg/h.

Spinal Block

Position: Preferably should be given in lateral under sedation or sitting position in cooperative child.

Technique: (a) L4–5 or L5–S1 interspinous space is targeted in infants. L3–4 in older children (Tuffier’s line crosses L4–5 in infants), (b) A short stiletted, 24/25G spinal needle is introduced. Drug is given once free-flow of CSF achieved.

Drug dosages: 0.5% hyperbaric bupivacaine: 0.5 mg/kg in infants up to 5 kg (0.1 mL/kg), 0.4 mg/kg for children between 5–15 kg (0.08 mL/kg), 0.3 mg/kg for children >15 kg (0.06 mL/kg)

LOWER EXTREMITY BLOCKS

Femoral Nerve Block

Indications
- Surgeries on the anterolateral aspect of the thigh, e.g. fracture femur shaft, quadriceps muscle biopsy, etc.

Technique (Fig. 16)
- The femoral nerve is located immediately lateral to the femoral artery and deep to both the fascia lata and fascia iliaca
• A blunt 22-gauge needle is advanced lateral to the pulsation of the femoral artery
• Two fascial planes can be located by two distinct “pops” (of fascia lata and fascia iliaca)
• Block is achieved by depositing adequate drug volume deep to fascia iliaca.

Dosage
• 0.5 mL/kg of 0.25% bupivacaine or 0.2% ropivacaine.

Complications
Intravascular injection, hematoma.

Fascia Iliaca Block (Alternative to Femoral Nerve Block)
Blocks lateral cutaneous nerve of thigh and obturator nerve in addition to femoral nerve.

Indication
Surgeries on thigh.

Technique
• Inguinal ligament is divided in three equal parts
• Needle is entered 0.5–1 cm below the junction of middle and lateral thirds, at 90°
• “Pops” are felt as the needle traverses fascia lata and fascia iliaca
• While exerting pressure just caudal to the needle (via tourniquet) the drug is injected.

Dosage
0.7 mL/kg of 0.25% bupivacaine or 0.2% ropivacaine.

Complications
Intravascular injection, hematoma.

Sciatic Nerve Block

Indications
For surgeries of lateral aspect of lower leg, lateral aspect of ankle and entire foot, also anesthetizes posterior aspect of thigh (Fig. 17).

Contraindication
Local skin sepsis.

Technique
• Position—Lateral decubitus with operative leg on top, slightly flexed on hip and knee
• A line is drawn from PSIS to greater trochanter
• A second 3 cm line is drawn perpendicular from the mid-point of first line in caudal direction
• Needle with nerve stimulator is inserted at the end of 2nd line to hit the bone and subsequently moved towards PSIS till muscle contractions elicited.

Drug Dosage
0.5 mL/kg of 0.25% bupivacaine or 0.2% ropivacaine.
Popliteal Nerve Block

*Indications*
For surgeries of lateral aspect of lower leg, lateral aspect of ankle and entire foot (Fig. 18).

*Contraindication*
Local skin sepsis.

*Technique*
- Position—lateral decubitus or prone
- Popliteal fossa identified by palpating bifurcation of hamstring and biceps femoris
- Popliteal artery is palpated and nerve is blocked just lateral to it, either blindly or via nerve stimulator.

Ankle Block

*Indications*
Procedures on foot.

*Technique*
Five nerves are blocked (Fig. 19):
- Deep peroneal nerve—needle inserted through the skin at the ankle crease until it contacts the tibia; 5 mL of local anesthetic is injected, additional amount as the needle is being withdrawn
- Superficial peroneal nerve—subcutaneous infiltration from the anterior border of the tibia to the lateral malleolus
- Saphenous nerve—subcutaneous infiltration around the great saphenous vein at the level of the medial malleolus
- Tibial nerve—blocked at the level of the medial malleolus, posterior to posterior tibial artery
- Sural nerve—blocked at the lateral aspect below the lateral malleolus.

**UPPER EXTREMITY BLOCKS: BRACHIAL PLEXUS BLOCK (FIG. 20)**

Supraclavicular Approach

*Indications*
Surgeries of forearm and elbow.

*Technique*
- Position—supine with head turned to opposite side
- Subclavian artery is palpated lateral to the lateral border of sternocleidomastoid
- Nerve bundle is located superior and lateral to the artery using nerve stimulator
Axillary Approach

Indications
Surgeries of hand and forearm.

Technique
- Position—supine with shoulder abducted and elbow flexed
- Axillary artery is pinned to humerus using nondominant hand
- Axillary sheath is entered just superior to artery (recognized by slight give/needle pulsations) and half the dose is given
- Axillary sheath is again entered, just inferior to artery, and the remaining dose given.

Intraclavicular Approach

Indications
Surgeries of upper arm and elbow.

Technique
- Position—supine with head turned to opposite side
- Coracoid process is palpated at the lateral end of clavicle
- Nerve bundle is located 1 cm caudal and medial to coracoid process, using nerve stimulator.

Dosage
0.5 mL/kg of 0.25% bupivacaine or 0.2% ropivacaine.

Interscalene Block

Indications
Surgeries of shoulder and upper extremity.

Technique
- Turn the head to opposite side with neck slightly extended
- Needle is inserted in interscalene groove at the level of cricoid perpendicular to all planes
- Using a nerve stimulator, hand contractions (pronation-supination movement) are elicited at 0.5-0.6 mA and drug injected.

Wrist Block

It involves following three nerves:
- Radial Nerve—performed as a field block just above the radial styloid, infiltrating medially.
- Median Nerve—LA is deposited deep to the fascia (identified as a pop) between the tendons of palmaris longus and flexor carpi radialis.
- Ulnar Nerve—performed by inserting the needle under distal part of tendon of flexor carpi ulnaris.

Intravenous Regional Anesthesia (IVRA)

Bier’s Block
- Can be used in a cooperative child with sedation.

Indication
Upper and lower limb surgeries of approximately 1 hour duration.

Technique
- IV line is secured on the limb to be operated. A reliable double cuff on the limb is applied to ensure that pressure will not drop inadvertently. The limb should be exsanguinated before upper cuff is inflated with pressure exceeding arterial systolic pressure.
- Lignocaine 3 mg/kg is injected, after 2–3 minutes the lower cuff is inflated and the upper is deflated so that the tourniquet pain is avoided as the area under the second cuff is anesthetized. The cuff should not be deflated at least for 20 minutes.
- IV access on the opposite limb and resuscitation equipment should be ready in case of cuff failure.

ACKNOWLEDGMENT

Special thanks to Dr Atul Bhaskar, Pediatric Orthopedic Surgeon, for photographs and images of few conditions.

REFERENCES

Chapter 28: Anesthesia for Pediatric Orthopedic Surgery

INTRODUCTION

Pediatric anesthesia is a challenging specialty and anesthesia for pediatric trauma is an even greater challenge. Pediatric trauma can affect any of the organ systems of the body. Trauma is the most common cause of death in children and injury is the most serious public healthcare problem in pediatric population. Apart from considering the anatomical and physiological differences between pediatric and adult patients, all aspects of trauma care need to be implemented in children as for adults. Cerebral, abdominal and thoracic injuries account for most of the disability and death among injured children. The order of priority among injuries is related to the degree to which survival is threatened by each injury. It must be remembered that, assessment and resuscitation must proceed simultaneously. The key to success is repeated assessment to diagnose and treat injuries that are not immediately obvious on initial presentation. One has to be well versed with the basic principles of pediatric advanced life support (PALS) and pediatric advanced trauma life support (PATLS), in addition to understanding of basic concepts of pediatric anesthesia, so as to improve patient outcome.

ROLE OF ANESTHESIOLOGIST IN TRAUMA CARE

- Initial assessment and resuscitation of the injured child
- Securing intravenous or intraosseous access.
- Airway management in the emergency department
- Sedation or anesthesia for diagnostic imaging
- Anesthetic management for surgical intervention
- Ultrasound-guided techniques for acute and chronic pain management
- Postoperative intensive care management
- During intra- or inter-hospital transfer.

TYPES AND PATTERNS OF PEDIATRIC TRAUMA

Death due to trauma occurs in one of the three periods or peaks after injury, described as the trimodal distribution of deaths (immediate, early and late). Following the Advanced Trauma Life Support (ATLS) principles in resuscitation can significantly reduce the mortality in the second peak or the golden hour. The importance of pre-hospital trauma care cannot be over-emphasized. Road-traffic accidents or motor-vehicle injuries are the commonest causes of death in children of all ages. Other causes include fires, falls, drowning, homicides and child abuse. Even though falls account for maximum pediatric injuries, they infrequently result in death. Due to smaller body mass, greater force is applied per unit of body area of the child, resulting in multiple injuries.

Multisystem injury is common in blunt trauma. The most serious pediatric injury is blunt trauma to brain. Hence, apnea and hypoxia are much more common than hypovolemia in severe pediatric trauma, emphasizing the importance of airway and ventilatory management. The
bones of children are incompletely calcified and more pliable, resulting in greater incidence of internal organ damage without overlying bony fracture. Injury following falls depends on the height of fall (low, medium or high) causing extremity fractures, head, neck and spine injuries. Pattern of injury in automobile accidents depend whether the child was a restrained or an unrestrained occupant. Presence or absence of helmet determines the pattern of injury in two-wheeler or bicycle accidents. In thermal injuries, it must be remembered that the percentage of total body surface of the infant’s head is twice that of the normal adult. Early airway management and fluid resuscitation are of paramount importance. Burns of certain areas of the body can point towards suspected child abuse. Electrical burns result in serious internal body trauma as the heat generated results in thermal injury to the tissue. Severe burn injuries require early referral to a dedicated burn center. Special care must be taken in all trauma victims to prevent hypothermia. It is imperative to examine the entire body of an injured child to rule out occult trauma and injury marks. The revised pediatric trauma score (PTS) can be used as a predictor of injury severity. It gives a score of +2, +1 and -1 to each of the following 6 variables: Weight, Airway, Systolic blood pressure, Level of consciousness, Fracture and Cutaneous lesions (contusion/abrasion/laceration/tissue loss). A PTS score less than 8 mandates referral to a dedicated pediatric trauma center.

Note:
- Apnea and hypoxia are much more common than hypovolemia in severe pediatric trauma.
- Early airway management and fluid resuscitation are of paramount importance in burn victim.

INITIAL ASSESSMENT: PEDIATRIC ADVANCED TRAUMA LIFE SUPPORT PRINCIPLES

The principles of pediatric ATLS are similar to the adult ATLS protocols. Treat the injury causing immediate threat to life first. The standard ABCDE’s need to be followed diligently.

A – Airway maintenance with cervical spine control
B – Breathing with oxygenation and ventilation
C – Circulation with hemorrhage control
D – Disability limitation and neurologic status
E – Exposure/Environment control: Complete patient undressing with prevention of hypothermia.

Under emergency situations, it is preferable to use a readily available Broselow’s tape for determining the correct tube or airway sizes, drug doses and fluid boluses required (Figs 1A to C). It is a color-coded tape measure used in pediatric emergencies, especially trauma. It correlates a child’s height as measured by the tape to his/her weight to provide age-appropriate drug doses, size of airway equipment needed, intravenous fluids and defibrillator shock voltage. This provides quick and easy information where time is of essence in prompt management. It is designed for children up to 12 years or up to a weight of 36 kg. It has 9 color zones (gray, pink, red, purple, yellow, white, blue, orange and green) corresponding to the child’s weight in kilograms or pounds. The child must be lying down and the tape must be placed with the red portion towards the head-end. The tape mark that corresponds to the child’s heels indicates the child’s weight and color zone.

Note: Keep Broselow tape handy in emergency wing for rapid use.

There are several anatomical and physiological differences between an adult and child’s airway and respiratory system, which make children exquisitely prone to hypoxemia. All children must be adequately pre-oxygenated before any airway intervention.

PRIMARY SURVEY AND RESUSCITATION

In primary survey, life-threatening conditions are identified and prioritized management is instituted immediately. The priority remains as airway, breathing, circulation and neurological assessment and management simultaneously. Inability to establish and/or maintain a patent airway with lack of oxygenation is the most common cause of cardiac arrest in children. Both basic and advanced airway management tools, including large bore suction should be kept ready and functional. Supraglottic devices like laryngeal mask airway (LMA) and its variants, though cannot prevent aspiration, can serve as a reasonable alternative in difficult airway, till a definitive airway is secured. Rapid sequence induction (RSI) with cricoid pressure is the gold standard even in pediatric trauma airway management. Every trauma victim needs to be considered as full stomach, until proved otherwise. This is more so in children who swallow
a lot of air (aerophagy) under such circumstances. Air, blood or foreign bodies may be swallowed causing gastric distension, and trauma can further delay gastric emptying.14 Difficult airway needs to be anticipated in all trauma victims and difficult airway cart must be available in the emergency department (ED) and operating room (OR). If the child’s trachea is already intubated, then the anesthesiologist has to check for correct tube placement: type, size, cuffed/uncuffed, bilateral equal air entry and fixation length at mouth or nose. The newer microcuff endotracheal tubes with ultra-thin high volume, low pressure polyurethane cuff can be used to reduce chances of endobronchial intubation.15 Cuff pressure in all cases should be maintained below 20 cm of H₂O. Selection of tube size is according to age, for which several formulas are popular (Table 1).

Note: Priority for trauma management is airway first and then breathing, circulation and neurological assessment subsequently and management simultaneously.

The increased physiologic reserve in children allows for maintenance of systolic blood pressure within normal limits, even in the presence of shock. Increased heart rate and decreased skin perfusion may be the only signs of hypovolemia. Other features of blood loss include weak peripheral pulses, narrowed pulse pressure (<20 mm Hg), skin mottling, cool extremities and decrease in consciousness level. The lower limit of normal systolic pressure in a child = 70 + 2 × Age (in years). Fall in blood pressure is a late sign and presence of hypotension in a child indicates a state of decompensated shock with severe blood loss (>45% of the blood volume).16 The goal of fluid resuscitation in trauma victim is rapid replacement of blood loss to achieve optimal circulating blood volume. Usually three fluid boluses of 20 mL/kg to a total of 60 mL/kg are given for initial replacement therapy. Loss of > 20% blood volume is replaced by blood at 10 mL/kg. In hypovolemic children, etomidate (dose 0.1–0.2 mg/kg intravenous) is preferred for drug-assisted intubation due its cardiovascular stability. Urinary output is one of the best indicators of organ perfusion and adequacy of resuscitation. The normal urine output in infants = 2 mL/kg/hour; in younger children 1.5 mL/kg/hour, and in older children = 1 mL/kg/hour.17 Urinary catheters with in-built temperature probes are available for use in critically ill pediatric patients. A recent concept in fluid resuscitation during shock is to accept a lower than normal blood pressure. This is known as controlled, hypotensive or balanced resuscitation.18 It can be used as a temporizing measure to contain the bleeding, till definitive surgical control is undertaken.

Note: Increased physiologic reserve in children allows for maintenance of systolic blood pressure within normal limits, even in the presence of shock.

Intraosseous (IO) needles are life-saving tools in pediatric trauma.19 They are indicated in children with no or difficult venous access, in emergent situations like severe trauma or cardiorespiratory arrest with urgent fluid and drug administration. Contraindications for IO use are targeted bone fracture, infection at insertion site or anatomical limitations. IO needles are generally placed in the proximal and distal ends (epiphysis) of long bones such as tibia, humerus and femur. Within the epiphysis or medullary space lies a vast system of canals, which the fluids can traverse to reach the central circulation. Any fluid infused into the intraosseous space gains access to the central circulation within just few seconds. The IO needle lengths available include: 15 mm, 25 mm and 45 mm. Its size is 18 gauge in infants and 15 gauge in young children. The needle is inserted at 90 degrees to the skin initially and later tilted to 45 degrees and directed caudally towards the medullary canal (Fig. 2). Any drug that can be given intravenously can be given safely through IO and their doses are same.

Neurological evaluation is established by assessing level of consciousness, pupillary size and reaction, lateralizing signs and spinal cord injury level.20 The level of consciousness can be assessed by Glasgow Coma Scale score (GCS). The maximum score is 15 and minimum score is 3. The lower the score, more is the neurological insult. This score also aids in serially monitoring the child for any deterioration or improvement. The main difference in GCS for children assessment is in verbal response score, as the eye-opening (E) and motor responses (M) are similar to adults. The verbal response (V) can be summarized according to the age of the injured child (Table 2).

### Table 1: Formulas for pediatric endotracheal tube selection:

<table>
<thead>
<tr>
<th>Name</th>
<th>Formula for endotracheal tube size</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Penlington</td>
<td>ID (mm) = [age/3]+3.5</td>
<td>Age less than 6 years</td>
</tr>
<tr>
<td></td>
<td>ID (mm) = [age/4]+4.5</td>
<td>Age more than 6 years</td>
</tr>
<tr>
<td>Cole</td>
<td>ID (mm) = [age/4]+4</td>
<td>For uncuffed tubes</td>
</tr>
<tr>
<td>Modified Cole</td>
<td>ID (mm) = [age/4]+4</td>
<td>For uncuffed tubes</td>
</tr>
<tr>
<td>Morgan and Steward</td>
<td>ID (mm) = [age+16]/4</td>
<td>For uncuffed tubes</td>
</tr>
<tr>
<td>Motoyama</td>
<td>ID (mm) = [age/4]+3.5</td>
<td>For cuffed ETT in children 2 years or older</td>
</tr>
<tr>
<td>Khine</td>
<td>ID (mm) = [age/4 + 3]</td>
<td>For cuffed tubes, one-half smaller size than the calculated uncuffed ETT diameter</td>
</tr>
</tbody>
</table>

Age in years
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It is important to prevent the occurrence of the lethal triad (acidosis, coagulopathy and hypothermia) in pediatric trauma patients by providing adequate oxygenation, airway-breathing control, improving the circulatory status and maintaining normothermia.21

SECONDARY SURVEY

Secondary survey begins after primary survey is completed and optimization of vital signs has been demonstrated. It is the head-to-toe evaluation of the injured child, including history-taking, reassessment of vitals, complete neurologic evaluation. History-taking is simplified by the pneumatic: AMPLE (A – allergies, M – Medications used, P – Past illness/Pregnancy, L – last meal time, E – events/environment related to injury).22 History about mechanism of injury can tell us the type or severity of trauma and the possible body areas affected. The child’s ABCDE’s should be re-evaluated and all interventions done noted. The child may require additional blood tests, X-rays and imaging studies during the secondary survey. A decision to transfer the severely injured child may also be taken at this stage for more definitive care.

MONITORING

Basic monitoring in the ED and OR should include pulse oximeter (SpO2), blood pressure, electrocardiogram (ECG), temperature and end-tidal carbon-dioxide monitor (EtCO2). In certain situations, invasive monitoring (arterial line, central venous catheter, intracranial pressure monitor) can be undertaken. The use of ultrasound in securing invasive lines can be very helpful in small children. Other monitors, which can be used in the perioperative setting include: precordial stethoscope, neuromuscular monitoring, bispectral index (BIS), multigas monitor, airway pressure, urine output and cardiac output measurements. In the intensive care unit (ICU), apart from the above monitors, serial GCS scores, pupillary size measurements and arterial blood gas (ABG) measurements need to be undertaken. However, it must be remembered that no monitor can substitute for a good clinical acumen.

ANESTHETIC CONSIDERATIONS FOR SPECIFIC TRAUMA

Anesthesia for Head Injury

Head injury is an important cause of death and disability worldwide. The two phases of traumatic brain injury (TBI) include: Primary injury (tissue or mechanical damage incurred at the time of trauma) and Secondary or delayed injury (inflammatory and excitotoxic processes leading to cerebral edema and raised intracranial pressure (ICP)).23 Anesthesiologists need to contain secondary injury by preventing hypoxemia and hypotension as well as by maintaining normoglycemia, normothermia and normocarbia. Nasotracheal intubation or nasogastric tube is contraindicated in base of skull fractures.

The 2012 guidelines for acute medical management of severe TBI in pediatric population highlight the following:24

- **Intracranial pressure (ICP):** Consider ICP monitoring and treatment at an ICP threshold of 20 mm Hg
- **Cerebral perfusion pressure (CPP):** A minimum CPP of 40 mm Hg is recommended in TBI; CPP threshold of 40–50 mm Hg may be considered.
- **Brain oxygenation:** Maintenance of oxygen tension \( \geq 10 \) mm Hg.
- **Hyperosmolar therapy:** 3% hypertonic saline (0.1–1 mL/kg/h) should be considered for treatment of intracranial hypertension (ICH).
- **Hyperventilation:** Avoid prophylactic hyperventilation to ETCO2 <30 mm Hg in the initial 48 hours after injury.
However, if hyperventilation is used for refractory ICH, advanced neuromonitoring is to be considered for evaluation of cerebral ischemia.

- **Temperature control:** After severe TBI, moderate hypothermia (32–33°C) should be initiated within first 8 hours and continued for up to 48 hours. This helps in reducing intracranial hypertension. Shorter duration (i.e., less than 24 hours) hypothermia should be avoided. If hypothermia is required for any scenario then rewarming should not be done >0.5°C/h.

- **Cerebrospinal fluid (CSF) drainage:** CSF drainage through an external ventricular drain may be considered in management of raised ICP.

- **Barbiturates:** Barbiturate therapy may be considered in hemodynamically stable, intubated patients with refractory ICH, despite maximal medical and surgical management. When high dose barbiturates are being given, continuous arterial blood pressure monitoring and cardiovascular support to maintain adequate cerebral perfusion are required.

- **Corticosteroids, analgesics, sedatives and neuromuscular blockade:** The use of corticosteroids is not recommended to reduce ICP in children with severe TBI. Etomidate may be advocated to control severe ICH, considering the risk of adrenal suppression.

- **Anti-seizure prophylaxis:** Anti-epileptics are not recommended for children with severe TBI for preventing late post-traumatic seizures. It may be considered for preventing early post-traumatic seizures in children with severe TBI.

- **Decompressive craniectomy:** Decompressive craniectomy with duroplasty may be considered for children with early signs of neurologic deterioration or refractory ICH during the initial stages of their treatment.

- **Nutrition:** There is no evidence supporting the use of immune-modulating diet to improve outcome.

The principles of anesthetic management in TBI include the following: cervical spine protection; definitive airway management in children with low GCS scores (<9); maintain adequate mean arterial pressures so that the CPP is more than 50 mm Hg in 6–17 year olds and more than 40 mm Hg in 0–5 year olds; and use of appropriate sedative, analgesic and neuromuscular blockers according to institutional protocols to mitigate secondary brain injury. There is recent evidence to avoid the use of propofol infusion and advocate the use of ketamine in pediatric TBI with refractory ICH.25

All inhalational anesthetics decrease cerebral metabolic rate (CMR) and increase cerebral blood flow (CBF), thereby uncoupling CBF from CMR.26 It is better to avoid succinylcholine due to concerns regarding undiagnosed myopathies in pediatric population and due to the rise in ICP caused by fasciculations. Nevertheless, succinylcholine is not contraindicated and may be used for securing a difficult airway with aspiration risk. Care needs to be taken while applying cricoid pressure, as it can lead to subluxation at injury site, laryngotracheal compression and improper laryngeal view. Anterior part of the rigid cervical collar needs to be removed temporarily to facilitate intubation and in-line stabilization performed for cervical spine immobilization.

### ANESTHESIA FOR CHEST/AIRWAY INJURY

Airway trauma results in edema and airway obstruction. One mm of edema can narrow a child’s airway caliber by 60% (as per Hagen-Poiseuille’s equation).27 Also, the pressure of endotracheal tube cuff or a very tightly inserted endotracheal tube may cause mucosal trauma leading to airway compromise on extubation. So, a leak is advisable around the endotracheal tube to prevent subsequent subglottic stenosis and post-extubation stridor. Laryngeal trauma is characterized by the triad of clinical signs: hoarseness of voice, subcutaneous emphysema and palpable fracture.28 Emergent intubation is warranted in such situations and emergency tracheostomy or even surgical cricothyroidotomy may be a life-saving option. Most pediatric chest trauma is caused by blunt injury, while penetrating trauma is more common in adolescents. Serious intrathoracic injuries can occur without rib fractures and the mortality rate increases with the number of ribs fractured. Management involves standard ATLS protocols of ABCDE.4 Lung contusion is the commonest chest trauma in pediatrics.29 It causes bleeding and fluid leakage into lung tissue, leading to stiffening, pulmonary edema and hypoxia. Mild pulmonary contusions are managed by good pain control, supplemental oxygen, judicious fluid therapy, pulmonary toilet and early mobilization. Anesthesiologists may administer thoracic epidural catheter for analgesia. Severe contusions necessitate mechanical ventilation, application of positive end-expiratory pressure (PEEP), preventing overhydration and sometimes, even extracorporeal membrane oxygenation (ECMO). Rib fractures in children should alert the anesthesiologist to the possibility of significant chest or abdominal injury. In flail chest, the main concern is adequate pain relief to facilitate adequate respiration. Pneumothorax is the most common life-threatening thoracic injury. Since the mediastinum is highly mobile and compliant in children, its contents easily shift to the contralateral side, compromising venous return and cardiac output. Tension pneumothorax is a clinical diagnosis requiring immediate needle decompression,
followed by chest tube insertion. It must be remembered that positive pressure ventilation can convert a simple pneumothorax into a tension pneumothorax, if undetected. Selective one lung ventilation may rarely be required in children with complex tracheobronchial tree injury. This can be quite challenging due to unavailability of smaller sized double lumen tubes, in which case bronchial blockers or univent tubes may be inserted. Massive hemothorax may warrant an early thoracotomy. Blunt chest trauma can also cause cardiac tamponade, characterized by the Becks triad (venous pressure elevation, decline in arterial pressure and muffled heart tones), Kussmaul’s sign and pulseless electrical activity (PEA).30 Nowadays, with the use of ultrasound, focused assessment sonography in trauma (FAST) can rapidly and accurately identify pericardial fluid.31 Pericardiocentesis can be used as a temporizing measure till the patient is prepared for definitive surgery. Thoracotomy is not generally needed in pediatrics and should be undertaken only in the presence of a qualified surgeon. Other serious thoracic injuries like traumatic aortic disruption, diaphragmatic injury, blunt esophageal rupture and cardiac contusions are rarely seen in childhood.

**ANESTHESIA FOR ABDOMINAL INJURY**

Majority of pediatric abdominal injuries are due to blunt trauma. Oro-gastric decompression is preferred in infants to decrease gastric distension. Bladder decompression also facilitates abdominal examination. Several diagnostic adjuncts can be used for abdominal trauma assessment like FAST, diagnostic peritoneal lavage (DPL) and computed tomography (CT) scanning.32 FAST can pick up even small quantities of intra-abdominal blood in children, which is unlikely to be associated with significant injury. Nowadays, e-fast or extended FAST for detecting hemothorax is done in-addition to the standard four point scan. It is vital to understand that operative treatment is not indicated by the amount of intraperitoneal blood, but by the degree of hemodynamic disturbance and its response to therapy. DPL may be used only if FAST or CT (computed tomography) scanner is not available or if the child is hemodynamically unstable to be transported to the radiology suite. The mere presence of intraperitoneal blood does not mandate a laparotomy. The presence of feces, vegetable fibers or bile warrants a laparotomy. At time of obtaining the intravenous access itself, blood should be taken for baseline investigations (hematocrit, coagulation screen) and sent for cross-matching. The prime indication for operative intervention in children is persistent hemodynamic abnormality despite resuscitation and transfusion requirements exceeding one-half of the child’s blood volume or 40 mL/kg in the first 24 hours post-injury. Among the specific visceral injuries, duodenal hematomas are common in children due to thinner abdominal wall and right upper quadrant injury from bicycle handlebar or striking of elbow. It is managed conservatively with gastric decompression and parenteral nutrition. Other common injuries in children include blunt pancreatic injuries, small bowel perforation and mesenteric avulsion. Children restrained by seat belts are at risk of enteric disruption, which requires early surgery.33 RSI with cricoid pressure is advocated. Bladder rupture is common due to shallow depth of the child’s pelvis. Straddle injuries to the perineum occurs with falls onto prominent objects and may cause intraperitoneal injuries. Injury to solid organs can occur in blunt trauma. Usually, they do not require operative repair, but the child should undergo serial CT scan after stabilization and admitted for continuous monitoring. A massive splenic rupture may require splenectomy, with the risk of post-splenectomy sepsis and sometimes death.

**ANESTHESIA FOR UROLOGICAL/PELVIC INJURY**

Nearly 12% of pediatric trauma victims suffer urogenital injury. Most of these require conservative and supportive treatment, except for penetrating injuries, which warrant operative intervention. Kidney, bladder and urethral injuries are common in pediatric patients in view of their body habitus. The reasons include relatively large kidney compared to body size; lower positioning of kidneys, not fully protected by ribcage; reduced retroperitoneal fat cushion around the kidney; less elastic intima of the renal artery, predisposing to traction-distraction injury. The management should be as per standard ATLS protocol for resuscitation, use of pelvic binder to contain pelvic hematoma, ruling out urethral injury (blood at the urethral meatus, blood in scrotum or blood on the per-rectal examination during primary survey) before catheterization, excluding other congenital anomalies of the genitourinary system and arranging for blood and blood products. The anesthesiologists after resuscitation may be involved in advanced imaging studies of the injured child (abdominopelvic computed tomography, CT cystogram, retrograde urethrogram and Doppler flow studies of renal vessels). In the OR, attention must be paid to securing large bore intravenous access, preventing hypothermia, avoiding movement of the pelvis, maintaining adequate urine output and avoiding drugs which are excreted by the kidneys.
ANESTHESIA FOR ORTHOPEDIC INJURY; DAMAGE CONTROL RESUSCITATION

Musculoskeletal injuries are the commonest indication for operative management in most trauma centers. It involves initial resuscitation as per ATLS protocol (ABCDE) and early stabilization of long bone, pelvic and acetabular fractures. Early total care (ETC) is definitive fixation of all long bone fractures within 24 hours of injury after physiologic and metabolic stabilization of the patient.

Damage control resuscitation (DCR) is a new concept consisting of time-limited permissive hypotension; early bleeding control; advocating massive hemorrhage protocols (MHP’s) to correct shock and coagulopathy; damage control surgery (DCS) for hemorrhage control; and use of antifibrinolytics like tranexamic acid. DCS involves emergency surgery to save life or limb of a trauma patient, while delaying the time consuming reconstructive surgery later on.34

Preoperative evaluation must rule out concurrent medical problems and organ system involvement. Low cardiac output state or hypovolemia must be optimized prior to surgery. Chest trauma and pneumothorax must be excluded before giving positive pressure ventilation. Associated obstructive sleep apnea or pulmonary hypertension must be looked in injured obese child. The chest and pelvic X-ray taken during primary survey need to be reassessed. Rapid sequence induction (RSI) with cricoid pressure and cervical stabilization, after adequate preoxygenation with ready difficult airway cart and suction is recommended for airway management. Apart from active warming techniques, intravenous fluids and blood need to be warmed. Intravenous induction can be done either with ketamine or etomidate for preserving temperature and fluid balance. Ultrasound guided block procedures in children. These surgeries can be time consuming, emphasizing the importance of maintaining hemodynamic stability. Regional anesthesia can supplement general anesthesia and help in postoperative analgesia as well, in addition to decreasing the risk of thromboembolism. Early definitive stabilization of femur fracture within 24 hours of injury is advantageous in decreasing pulmonary complications, incidence of deep vein thrombosis (DVT) and overall hospital stay/costs. Pelvic ring fractures are associated with torrential hemorrhage and sometimes fatal retroperitoneal bleeding. Successful treatment involves volume resuscitation, external fixation of the unstable pelvis and angiography. Dislocation of hip is an emergency resulting from high impact trauma. Failure of its timely diagnosis and reduction can lead to avascular necrosis and neurologic injury. General anesthesia with endotracheal intubation and standard monitoring is recommended. Open fractures are surgical emergencies due to increased rate of infection. If the child is not stable enough to go to the OR for open fractures management, then it may be done at the bedside, after basic ‘ABCDE’ control. Traumatic amputations require immediate treatment with pressure, bleeding control, early intravenous antibiotics, tetanus prophylaxis and surgical debridement. Other indications for emergency surgery after trauma include vascular or arterial injury and compartment syndrome. Such limb threatening injuries must be attended immediately after initial stabilization and resuscitation. Intraoperative complications that must be avoided include hypotension or shock, hazards of massive transfusion, coagulopathy, hypothermia and fat embolism syndrome. Crush injuries can result in rhabdomyolysis and acute renal failure, which has to be treated with vigorous volume replacement and maintaining a good urine flow.35 All orthopedic injuries are very painful and pain management must be given paramount importance.

ANESTHESIA FOR REIMPLANTATION

Children are good candidates for reimplantation surgeries because of their greater ability to heal and regrow tissue. Injuries to other body parts must be actively sought. Reimplantation of an amputated part is best done within 4–6 hours of the injury, but can be successful up to 24 hours after the injury if the amputated part is cooled.36 After aligning the preserved digit in place, the surgeon stabilizes the bone with wires or plates. Microvascular reconstruction with revascularization of the amputated part is then done. Usually general anesthesia with RSI supplemented with regional block is given for such procedures in children. These surgeries can be time consuming, emphasizing the importance of maintaining temperature and fluid balance. Ultrasound guided block given after general anesthesia for postoperative analgesia in a child also facilitates microvascular flow by increasing limb blood supply. Care must be taken while performing central neuraxial blocks as these surgeries may require anticoagulation or use of dextran.

ANESTHESIA FOR SPINAL INJURIES

Fortunately, spinal cord injuries are uncommon in children. It may present as SCIWORA (spinal cord injury without radiologic abnormalities).8 Patient’s neurological status should be documented.37 The American Spinal Injury Association (ASIA) score is the grading scale for essential elements of neurological assessment in spinal injury.38 Grade A is complete loss of motor and sensory function; grades B, C and D refer to lesser degrees of
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INJURIES

ANESTHESIA FOR OPHTHALMIC INJURIES

Eye injuries are common in pediatric population and can be isolated or occur as part of multisystem injury. They can go undetected if not specifically looked into, which can lead to vision loss. Globe perforation can present for emergency surgery. Such children should be considered full stomach (RSI with cricoid pressure) and care should be taken to prevent further rises in intraocular pressure (IOP). Gentle mask application, laryngoscopy, intubation and extubation should be practiced. Drugs which affect ventilation and airway exchange catheters should be used for a smooth extubation.

ANESTHESIA FOR FACIOMAXILLARY INJURIES

Faciomaxillary injury is usually associated with difficult airway and cribiform plate fractures. Since infants and neonates are obligate nose breathers, occluded nasal passages from such injuries can be problematic. Objective signs of airway obstruction must be recognized in the form of agitation or obtundation, use of accessory muscles of ventilation, abnormal or noisy breathing sounds and tracheal shift. Awake fibreoptic intubation application. Most of these surgeries are done in prone position and all considerations for prone position need to be strictly followed. The objective of MILS is to apply sufficient opposite forces to the head and neck to limit the movement during airway intervention. The prime perioperative goal is to prevent iatrogenic deterioration of existing injury as well as secondary injury, whilst providing generalized organ support. Significant blood loss may occur in some spine surgeries and coagulopathy may follow massive blood transfusion. Emergency hemorrhage panel (EHP) assessment includes estimation of prothrombin time, fibrinogen, platelet count and hematocrit levels for guiding transfusion decisions. Many of these patients may require postoperative elective ventilation and airway exchange catheters should be used for a smooth extubation.
is recommended and cervical spine injury must be ruled out. Anesthesiologist may also be required to give sedation for computed tomographic scan of the face with three-dimensional reconstruction in the injured child. It may be associated with traumatic brain injury or base of skull fracture, where nasotracheal intubation or nasogastric tube insertion is contraindicated. There may be blood in the oral cavity, which may be accidentally swallowed by the injured child. Dislodged teeth following facial trauma can serve as foreign bodies, which can lodge either in the food or the wind pipe, necessitating removal under anesthesia. Rapid sequence induction with cricoid pressure along with full preparations for cricothyroidotomy or tracheostomy must be adopted in such situations. Surgical cricothyroidotomy is not recommended in children less than 12 years of age to avoid damage to the cricoid cartilage. Definitive plastic surgery and fixation of Lefort fractures must be carried out only after stabilization of the child.

**FLUID MANAGEMENT**

In trauma, initial control of circulation is with warm isotonic or balanced salt solutions. Priority must be given to establish at least one or preferably two reliable intravenous accesses in children, failing which intraosseous catheter can be inserted. A maximum of three fluid boluses of 20 mL/kg each of normal saline or ringer lactate can be infused to restore the circulating blood volume. After the second bolus (i.e. after infusing 40 mL/kg), consideration for blood transfusion must be given in hemodynamically unstable children. Blood for grouping and cross-matching along with baseline investigations can be drawn at the time of securing the intravenous access. In the OR, fluid replacement must include fasting deficit, maintenance requirements and replacement of losses. The standard Holliday-Segar formula for pediatric fluid calculations can be applied, after initial resuscitation in the ED. For maintenance, normal saline or N/2 saline with 5% Dextrose can be given. In neonates, 10% dextrose can be added to maintenance fluid requirements, especially after blood sugar monitoring. Children are prone to develop hyponatremia and hypoglycemia. Urine output serves as a useful guide to adequacy of fluid management.

**PAIN MANAGEMENT**

Any treatment for trauma is incomplete without pain relief. Adequate analgesia not only blunts the sympathetic nervous system stimulation, but also allays anxiety of the injured child and parents as well. Musculoskeletal injuries are so painful that they may hinder comprehensive examination and diagnostic assessment. Analgesics, like paracetamol (preferably intravenous route at 10–15 mg/kg, every 4–6 hours) and NSAID’s (nonsteroidal anti-inflammatory drugs) are the starting points in pediatric pain management. Opioids (fentanyl and morphine) can be given once airway is secured and hemodynamic parameters are stable. Single shot and continuous nerve or plexus blocks are very helpful in limb injuries, where local anesthetics with or without adjuvants can be infiltrated. Plexus catheters require careful tunneling in children and complete asepsis needs to be maintained. Epidural analgesia can be given for thoracic and abdominal injuries after ruling out coagulopathy. In patients in whom central neuraxial blocks are contraindicated or peripheral blocks not given, intravenous patient controlled analgesia (IV-PCA) can be given in a monitored set-up, once the patient is stabilized. If the child is too young or unable to operate such electronic pain pumps, nurse-controlled analgesia or continuous infusors can be advocated. Splinting of fractures itself can decrease the pain significantly. Multimodal analgesia has been shown to improve overall patient outcome and prevents the occurrence of chronic pain syndromes.

**CHILD ABUSE**

Child abuse is on an increasing trend these days and may be missed if not specifically looked into. Any child who sustains an intentional injury as a result of acts by parents, guardians, or acquaintances is a battered or abused child. Homicide is the commonest cause of injury and death in infancy. It must be suspected whenever there is a discrepancy between history and degree of physical injury, long interval between time of injury and seeking medical attention, repeated trauma or ED visits, history of doctor shopping or inappropriate parental support. It presents in the form of multi-colored bruises, perioral or perianal or genital injuries, green-stick fractures, buckle fractures, multiple subdural hematomas, retinal hemorrhages, burns and bizarre injuries. These children should be dealt with compassion and medicolegal authorities should be informed.

**TRANSFER AND TRANSPORT OF PEDIATRIC TRAUMA PATIENT**

Safe intra- and interhospital transfer of the injured pediatric patient is a challenging task. The foremost aspect is to identify patients who require transfer from a primary care facility to an institution capable of providing necessary level of trauma care. Once the need to transfer is recognized, precious time should not be wasted.
waiting for test results or performing more diagnostic procedures. The referring doctor is responsible for initiating transfer of the patient to the receiving hospital and selecting a suitable mode of transportation. There must be clear communication between the referring and receiving doctor. Transfer agreements must be established between institutions under the legal framework for safe transfer of trauma patients. It is important to ensure that the patient is stabilized and his ABCDE’s taken care-of, before and during transfer. Tracheal tubes and intravenous lines can get dislodged or displaced during transfer. A simple pneumonic to remember in such situations is DOPE (Dislodgement, Obstruction, Pneumothorax and Equipment). Complete information about the patient needs to be provided in the form of identification, history of incident, mechanism of injury, initial findings and their response to therapy, airway maintenance, circulatory status and fluid given, and neurologic status (GCS and revised trauma score). Complete monitoring must be continued during transfer, with the aim of preventing mishaps or physiologic deterioration.

**PEDIATRIC INTENSIVE CARE AFTER TRAUMA SURGERY**

Many injured children may require admission to a dedicated intensive care unit (ICU) after initial stabilization in the ED. This is mainly for monitoring, close observation, mechanical ventilation and nursing care. These children may undergo various diagnostic tests either in the ICU or outside for finalizing line of further management. It is imperative to look into the "ABCDE’s" of the child on receiving in the ICU. A detailed history from the parents or guardians can be taken about the past history and mechanism of present injury of the child. Some children may have associated congenital anomalies, especially heart disease. In mechanically ventilated children, precautions should be taken to prevent barotrauma and ventilator associated pneumonia (VAP). Nosocomial infections and sepsis must be prevented and treated. Several children may be observed in a high dependency unit after solid organ injury and serial CT scans may be required to assess progress of injury. Few children with polytrauma and multiorgan failure may be given a trial of ECMO where facilities exist, when other modalities have failed. Critical care may also be required after surgery in a child with low GCS scores or fluctuating hemodynamics. Blood and blood product transfusion may need to be continued. Invasive monitors inserted previously need to be taken care-of and monitoring continued. Active warming needs to be instituted and hypothermia has to be prevented. Electrolyte imbalances occur frequently in such children and they have to be corrected. There has to be a coordinated effort between the anesthesiologist, pediatric intensivist and the surgeon for a successful outcome. Trauma also has long-term effects on the injured children in the form of blunted growth and development, post-traumatic stress disorder, disability and chronic pain syndromes.

**BRAIN DEATH AND ORGAN DONATION**

Severe TBI’s can result in brain stem death. Determination of brain death in term newborns, infants and children is a clinical diagnosis based on the absence of neurologic function. Hypotension, hypothermia and metabolic derangements must be corrected and drugs that interfere with neurologic testing should be discontinued. Two examinations including apnea testing with each examination separated by an observation period are required. The observation period is 24 hours for term newborns, and 12 hours for infants and children up to 18 years of age. In apnea test, an arterial PCO2 of 20 mm Hg above baseline and value ≥60 mm Hg with no respiratory effort should be documented. Ancillary cerebral studies are not mandatory to establish brain death and are not substitutes to neurologic examination. Organ donation and transplantation in children have an important life-extending benefit to the pediatric organ recipients and a great emotional effect on the donor and recipient families. The chief concerns are numerous and sometimes difficult to overcome: availability and access; pediatric medical and surgical consultation throughout donation-transplantation process; ethical, social, financial and follow-up issues; and public awareness about the growing need for organ donors of all ages.

**LEARNING POINTS**

- Pediatric ATLS principles form the cornerstone of trauma management in children.
- In trauma, life-threatening injuries need to be assessed and managed before detailed evaluation. Treat the injury causing immediate threat to life first. Secondary survey begins after primary survey is completed and optimization of vital signs has been demonstrated.
- Assessment, resuscitation and treatment must go hand-in-hand to maximize chances of survival.
- If peripheral intravenous access cannot be obtained, then intraosseous needles can be life saving in children.
- Child abuse is a serious problem, which can lead to delayed psychosomatic development in affected children.
REFERENCES

Chapter 29: Anesthesia for Pediatric Trauma

INTRODUCTION

The anesthetic management of children undergoing surgeries for congenital heart defects (CHD) has always been a challenging topic for discussion all over the world. This is mainly due to the ongoing evolution in the field of pediatric cardiac surgery. Not only are the surgical techniques refined and innovated over a period of time but also thorough preoperative evaluation and stabilization, newer techniques for the conduct of the cardiopulmonary bypass, intraoperative monitoring as well as advances in the postoperative management have slowly but definitely improved the outcomes of these children. Global literature reports prevalence of CHD between 6–13 per 1,000 live births.1-5 or nearly 25% of all congenital malformations.6 Hence, it is important for an anesthesiologist to understand the perioperative management of certain basic heart defects.

UNDERSTANDING THE CARDIO-PULMONARY PHYSIOLOGY IN CHILDREN

At birth, the blood flow pattern in the systemic and the pulmonary circulations undergo series of alterations. Also, the neonatal myocardium has certain limitations and one has to understand them to alter the perioperative anesthetic management. These differences in the cardiopulmonary physiology are explained below.

Restoration of Fetal to Normal Systemic Circulation

During fetal life, blood flow returning to the right atrium bypasses the unventilated fluid-filled lungs. Blood is then preferentially shunted across the patent foramen ovale (PFO) into the left atrium or passes from the right ventricle (RV) across the patent ductus arteriosus (PDA) to the systemic circulation (Fig. 1).
Chapter 30: Anesthesia for Pediatric Cardiac Surgery

The physiological closure (Functional closure) of the PDA occurs within 12–18 hours after birth. This occurs by abrupt contraction of the muscular wall of ductus arteriosus, which is associated with increases in the partial pressure of oxygen (PaO₂) coincident with reduction in pulmonary vascular resistance (PVR) caused by the first breath and cry. The anatomical closure may take 2–3 weeks.

Similarly, the PFO closure also occurs within few hours after birth. However, approximately 27% of adults still show probe patent foramen ovale. The PFO closure occurs due to fall in the PVR facilitating the blood flow to the lungs, thereby increasing left atrial blood volume and the left atrial pressure more than the right atrial pressure pushing the flap against the atrial septum and this functionally closes the foramen ovale.

The presence of certain congenital heart defects or pulmonary disease can disrupt this normal adaptive process, creating a Transitional Circulation, in which right-to-left shunting persists across the PFO or the PDA.

**Alterations in Pulmonary Circulation**

The pulmonary circulation undergoes significant change during the first month of life characterized by regression of the hypertrophied medial smooth muscular layer in the pulmonary arteries that exists in utero, resulting in a concomitant drop in PVR. In the immediate newborn period, the large decrease in PVR is due to lung expansion and the vasodilatory effects of a higher PaO₂. Further decline in PVR throughout the next 2 months of life is attributable to regression of the smooth muscle layer in the pulmonary arterioles. A corresponding fall in pulmonary artery pressure (PAP) occurs as PVR declines. Acute physiologic stress in the newborn period, such as hypoxemia or acidosis, can increase PAP and thus PVR.

**Myocardial Reserve**

Another unique feature of the normal neonatal and infant cardiovascular system is that the left ventricular function is restricted by a reduced number of beta-receptors, high resting levels of circulating catecholamines, limited recruitable stroke work, an immature calcium transport system and decreased ventricular compliance.

**Ventricular Mass**

In neonatal heart, there is a 50% reduction in the number of myofibrils arranged in a nonlinear, disordered array. As a direct result, the contractile mass of the heart is effectively reduced; resulting in a ventricle with low compliance. Preload augmentation is effective at low filling pressures (1–7 mm Hg). As a consequence, neonates are more dependent on heart rate (HR) and to a lesser extent on preload to maintain cardiac output (CO) at filling pressures of 7–10 mm Hg or greater.

**Poor Calcium Reserves**

The calcium transport system in the neonatal myocardium is underdeveloped. The transverse tubular system is absent and the sarcoplasmic reticulum which has to store and release calcium is small and inefficient. The neonatal heart is therefore more dependent on extracellular calcium levels than the adult myocardium. Because intracellular calcium concentrations play a central role in myocardial contractility, normal or even elevated plasma levels of ionized calcium may be necessary to augment or maintain an effective stroke volume (SV).

**PATHOPHYSIOLOGY**

It is extremely important to understand the inherent physiological differences in a pediatric heart as mentioned above and then understand the pathophysiology of various lesions in congenital heart defects. Broadly, the pathophysiology of any lesion can be understood on the basis of following anatomical and hemodynamic characteristics for that particular lesion:

1. Level of defect and direction of shunting of blood across them (Table 1).
2. Site of obstruction.
3. Dominance of systemic vascular resistance (SVR) or pulmonary vascular resistance (PVR) based on which the blood flows across the defect. The ratio of pulmonary blood flow (Qp) to systemic blood flow (Qs) is determined by the resistance in either circulation, blood flow favoring the path of least resistance (Table 2).

Based on the above information it is extremely simple to understand the pathophysiology for a given lesion, thereby facilitating the conduct of anesthetic management.

**CLASSIFICATION**

Congenital heart defects (CHD) have been classified into various categories by different authors. However, for the sake of convenience and for the ease of identifying the drugs to be used, the authors here propose a broad classification after understanding the pathophysiology of shunts. Mainly, CHD comprises of shunt lesions and obstructive lesions. In fact, it can be explained as, “It’s all about blood flowing across the communications (defects) and changing its path when it has nowhere to go.
(obstruction) in the direction of least resistance (SVR and PVR).” This leaves us with a simple way to classify lesions based on the direction of blood flow:

- Left-to-right shunts (Acyanotic): Atrial septal defect (ASD), ventricular septal defect (VSD), patent ductus arteriosus (PDA), atrioventricular canal (AVC) (Figs 2 to 5)
- Right-to-left shunts (Cyanotic): Tetrology of Fallot (TOF), tricuspid atresia, pulmonary atresia, Ebsteins anomaly (Fig. 6)
- Bidirectional shunts (Complex cyanotic): transposition of great arteries (TGA), total anomalous pulmonary venous connection (TAPVC), truncus arteriosus, single ventricles (Figs 7 and 8)
- Isolated obstructive lesions: Coarctation of aorta (COA), aortic stenosis (Fig. 9).

### PREOPERATIVE EVALUATION

The preoperative assessment of a child with CHD should include the following apart from the routine preoperative evaluation which an anesthesiologist would conduct:

1. **Detailed birth history:** Birth weight, current weight, birth asphyxia, NICU/hospital admission, if yes why and frequency?
2. **Congenital anomalies:** Craniofacial: microgastria, cleft lip/palate, macroglossia, large head, short neck, single kidney or history of any surgeries performed after birth for congenital non-cardiac/cardiac anomalies.
3. **The current status of the child:** Stable/unstable; intubated, if yes why?
4. **Medications:** Diuretics, digoxin, propranolol, PGE1 infusion, inotropes-Continue all.
5. **Investigations:** Complete blood count (CBC), renal function tests (RFT), liver function tests (LFT), arterial blood gases (ABG) in ventilated neonates, coagulation profile, Chest X-ray (CXR), 2-dimensional echocardiography (2D-Echo), cardiac catheterization report.
6. **Septic profile** work-up especially in neonates transported from peripheral centers on ventilator or sick neonates. These include baseline Procalcitonin levels (PCT) and blood cultures.
Once the above information is available, it can be interpreted and necessary interventions can be performed (Table 3).

**PREOPERATIVE PREPARATION**

**Premedication**

The aim of premedication is to prevent a child’s anxiety during separation from their parents. This separation from the dear ones not only further disturbs the crying child, but also disturbs the parents and the operating room environment; often seen in inadequately premedicated older children between the age groups of 1 to 5 years. They can be well-sedated with oral syrup Chloral hydrate 50 mg/kg or 0.5 mL/kg given in the ward 30 minutes prior to shifting in the operating room. It is best to have an intravenous (IV) line in the wards which can be taken at the time of sampling for the preoperative blood investigations, a day prior to the surgery. The same IV line can also be used the next day in the holding area if the child is crying despite Chloral hydrate premedication, to administer Injection Ketamine, Injection Midazolam and Injection Glycopyrrolate. This will keep the child calm.
when he/she is still in the mothers’ arms. Neonates do not need premedication, not even anticholinergics. They are disturbed only if they sense cold; hence nursing with warm hands, use of warming mattresses, warm air blowers and increasing the ambient temperature of the operating room for induction keeps the baby warm and sedated.

The authors do not prefer intramuscular premedication especially in cyanotic children for the risk of hematomas due to coagulation abnormalities. Also, intranasal, rectal routes are cumbersome to give, have unpredictable absorption and the discomfort might further make the child anxious. Sick neonates or infants do not need premedication as they can easily decompensate with respiratory depression and desaturations resulting in bradycardia.

Oral medications like diuretics, digoxin, propranolol should be continued. Infusion of ionotropes and PGE1 (0.02-0.1 μg/kg/min) should be on flow. Various practices are followed and the commonly used drugs for premedication with their doses and routes are given in Table 4.

**Fasting Guidelines**

The fasting guidelines are similar to those followed for other pediatric procedures and are clearly mentioned in
### Table 3: Interpretation and interventions based on history and investigations

<table>
<thead>
<tr>
<th>History and Investigations</th>
<th>Interpretation</th>
<th>Interventions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prematurity</td>
<td>Physiologically immature and have incomplete development of several organ systems, hence at a greater risk of mortality</td>
<td>They are not routine neonates, maintain temperature, titrate doses for the liver/kidneys to handle it, maintain sugar levels</td>
</tr>
<tr>
<td>Birth asphyxia</td>
<td>Neurological damage may have occurred</td>
<td>Ultrasound of head (neonates) or MRI with neurologist consult</td>
</tr>
<tr>
<td>NICU/ hospital admission</td>
<td>Respiratory cause, physiological jaundice or other birth related problems</td>
<td>To confirm whether completely treated and recurrent</td>
</tr>
<tr>
<td>Craniofacial anomalies</td>
<td>Difficult intubation</td>
<td>Awake-sevoflurane induction</td>
</tr>
<tr>
<td>Single kidney</td>
<td>Reduced kidney function</td>
<td>Maintain better perfusion pressures on CPB</td>
</tr>
<tr>
<td>Previous surgeries</td>
<td>Congenital or palliative shunts (assess sites for vascular/ arterial access)</td>
<td>Evaluate the system and for shunts maintain good hydration to keep hematocrit around 45 or perform phlebotomy</td>
</tr>
<tr>
<td>Stable</td>
<td>Better outcomes</td>
<td>Routine parental counselling</td>
</tr>
<tr>
<td>Unstable</td>
<td>Prolonged postoperative stay in PICU</td>
<td>High-risk consent</td>
</tr>
<tr>
<td>Diuretics, digoxin</td>
<td>Antifailure medications</td>
<td>To be continued</td>
</tr>
<tr>
<td>PGE1</td>
<td>To keep the PDA patent in case of TGA with intact septum, pulmonary atresias, hypoplastic left hearts and coarctation of aorta</td>
<td>To be continued</td>
</tr>
<tr>
<td>Ionotropes</td>
<td>Compromised perfusion and ventricular function</td>
<td>To be continued and hemodynamics optimized</td>
</tr>
<tr>
<td>CBC</td>
<td>Low hemoglobin indicates nutritional anemia (failure to thrive)</td>
<td>Nasogastric feeding for low Hb</td>
</tr>
<tr>
<td></td>
<td>High hemoglobin indicates polycythemia and tendency for spells</td>
<td>Phlebotomy to maintain Hb around 16–18 g/dL.</td>
</tr>
<tr>
<td>LFT, RFT</td>
<td>Hepatic congestion secondary to hepatomegaly in large L-R shunts</td>
<td>Can bleed more, consider giving vitamin K 0.1 mg/kg IV/IM preoperative single dose</td>
</tr>
<tr>
<td></td>
<td>Elevated BUN/creatinine indicates dehydration, should be normalized post catherization if dye used</td>
<td>Hydrate the child well</td>
</tr>
<tr>
<td>ABG</td>
<td>Low PO2 needs higher FiO2, High PCO2, may warrant preoperative intubation, high lactates or metabolic acidosis indicate poor tissue perfusion</td>
<td>Electively ventilate</td>
</tr>
<tr>
<td></td>
<td>Start dopamine/dobutamine</td>
<td>Sodium bicarbonate</td>
</tr>
<tr>
<td>Coagulation profile</td>
<td>If deranged may bleed in postoperative period</td>
<td>Arrange for adequate blood products</td>
</tr>
<tr>
<td>CXR</td>
<td>Cardiomegaly, pneumonias, oligemic lung fields indicate decreased PBF and congested indicate increased PBF or pulmonary arterial/venous hypertension</td>
<td>Therapies to improve PBF by adequate hydration preoperatively, premedication and avoid tet spells</td>
</tr>
<tr>
<td>2D echocardiography</td>
<td>Size of defects and degree of obstruction guides us to select appropriate ionotropes</td>
<td>Severe PAH and tachypnea may warrant preoperative intubation</td>
</tr>
<tr>
<td>Cardiac catheterization report</td>
<td>Branch pulmonary artery confluence, pressures and saturations for operability plan</td>
<td>Helps to anticipate intraoperative and postoperative problems</td>
</tr>
<tr>
<td>Septic profile</td>
<td>Poor outcomes as preoperative infections can cause profound vasodilatation when subjected to CPB</td>
<td>Start sensitive antibiotics if blood culture positive</td>
</tr>
</tbody>
</table>

**Abbreviations:** NICU, Neonatal intensive care unit; CBC, Complete blood count; LFT, Liver function test; RFT, Renal function test; ABG, Arterial blood gas; CXR, Chest X-ray; PICU, Pediatric intensive care unit; CPB, Cardiopulmonary bypass; PDA, Patent ductus arteriosus; TGA, Transposition of great arteries; BUN, Blood urea nitrogen; PBF, Pulmonary blood flow; MRI, Magnetic resonance imaging; PGE1, Prostaglandin E1; PAH, Pulmonary Hypertension
standard textbooks. The only major difference is that the fasting period should be covered with fluids, preferably 0.9% normal saline, at the rate of 3 mL/kg/h (75% of fluid requirement, under hydration) for left-to-right shunts and 5 mL/kg/h (125% of fluid requirement, over hydration) for right-to-left shunts.

### Infective Endocarditis Prophylaxis

Bacterial endocarditis is an infrequent but severe complication of CHD. An anesthesiologist should be aware of the potential patients at risk for developing infective endocarditis (IE) and appropriate antibiotic coverage should be given at least 30–60 minutes prior to the procedure. Patients undergoing surgical and dental procedures or instrumentations involving mucosal surfaces or contaminated tissues commonly suffer from transient bacteremia.\(^3\) As it is impossible to predict which patients will develop this infection or which particular procedures will be responsible, prophylactic antibiotics (Table 5) are recommended for all patients at risk for developing endocarditis. The current American Heart Association (AHA) guidelines recommend IE prophylaxis for the following patients:\(^2\)

1. Unrepaired cyanotic heart defects including palliative shunts and conduits.
2. Completely repaired heart defects with prosthetic material or device, whether placed by catheterization or surgery, during the first 6 months after surgery.
3. Repaired heart defects with residual defects, such as peripatch leaks.

### CHOICE OF ANESTHETIC DRUGS AND GENERAL PRECAUTIONS

The choice of anesthetic drugs can be simplified by understanding the pathophysiology of the lesion and the direction of shunt flow (Tables 1 and 2) with emphasis on the fact that shunts have to be balanced. Theoretically, drugs with an opposite effect on the basic shunt flow direction have to be used (Table 2). This means choosing drugs that reduce the SVR in left-to-right shunts and increase the SVR in right-to-left shunts. However, in the clinical scenario the above theory holds true in the broader aspect and not in the practical aspect; implying the use of same induction agents for all the lesions in the same doses.

---

**Table 4: Commonly used drugs for premedication with doses and route**

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Dosages</th>
<th>Route</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chloral hydrate</td>
<td>50 mg/kg</td>
<td>Oral</td>
</tr>
<tr>
<td>Midazolam</td>
<td>0.5 mg/kg</td>
<td>Oral</td>
</tr>
<tr>
<td>Diazepam</td>
<td>0.2–0.4 mg/kg</td>
<td>Oral</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>15–20 μg/kg</td>
<td>Transmucosal (lozenge)</td>
</tr>
<tr>
<td>Ketamine</td>
<td>5 mg/kg</td>
<td>Nasal, IM</td>
</tr>
</tbody>
</table>

**Table 5: AHA recommendations for antibiotics and dosages to be used**

<table>
<thead>
<tr>
<th>Situation</th>
<th>Agent</th>
<th>Adults</th>
<th>Children</th>
</tr>
</thead>
<tbody>
<tr>
<td>Able to take oral medication</td>
<td>Amoxicillin</td>
<td>2 g</td>
<td>50 mg/kg</td>
</tr>
<tr>
<td>Unable to take oral medication</td>
<td>Ampicillin</td>
<td>2 g IM or IV</td>
<td>50 mg/kg IM or IV</td>
</tr>
<tr>
<td></td>
<td>Cefazolin or Ceftriaxone</td>
<td>1 g IM or IV</td>
<td>50 mg/kg IM or IV</td>
</tr>
<tr>
<td>Allergic to Penicillin or Ampicillin</td>
<td>Cephalexin</td>
<td>2 g</td>
<td>50 mg/kg</td>
</tr>
<tr>
<td></td>
<td>Clindamycin</td>
<td>600 mg</td>
<td>20 mg/kg</td>
</tr>
<tr>
<td></td>
<td>Azithromycin</td>
<td>500 mg</td>
<td>15 mg/kg</td>
</tr>
<tr>
<td></td>
<td>Clarithromycin</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Allergic to Penicillin or Ampicillin</td>
<td>Cefazolin or Ceftriaxone</td>
<td>1 g IM or IV</td>
<td>50 mg/kg IM or IV</td>
</tr>
<tr>
<td>and unable to take oral medication</td>
<td>Clindamycin</td>
<td>600 mg IM or IV</td>
<td>20 mg/kg IM or IV</td>
</tr>
</tbody>
</table>

**MONITORING**

The basic monitoring includes electrocardiogram (ECG) for monitoring heart rate and detecting arrhythmias, noninvasive blood pressure monitor for initial phase of induction, temperature probes (nasopharyngeal and/or rectal), inspired O\(_2\) concentration monitor, continuous-pulse oximetry, and end-tidal CO\(_2\) measurement, arterial blood pressure, central venous pressure monitoring and activated clotting time (ACT).

Advanced monitoring includes transesophageal (TEE) echocardiography for preoperative assessment of the lesion and postoperative evaluation of surgical correction, near-infrared spectroscopy (NIRS) for monitoring cerebral perfusion on cardiopulmonary bypass especially in complex aortic arch surgeries and bispectral (BIS) monitoring to ensure adequate depth of anesthesia. Certain cases like COA, large PDA and hypoplastic left heart syndrome (HLHS) warrant the need for invasive monitoring in both upper limb (proximal to the PDA) and lower limb (distal to the PDA). Also, upper limb arterial lines should be avoided on the same limb, in cases operated previously for COA with subclavian flap plasty and cases of Blalock-Taussig shunt (BT shunt) as these lines would show inaccurate blood pressures.
The obvious cardiovascular effects of certain anesthetic agents cannot be neglected. Anesthetic induction may be influenced by factors that alter the uptake and distribution of inhalation or intravenous agents. The speed of induction with inhalation anesthetic or intravenous agents may differ based on the direction of the shunt flow. Inhalation induction in a patient with a right-to-left shunt can be prolonged because the blood is shunted away from the lungs thereby reducing its uptake by lungs. This effect can be reduced by hyperventilation and combination with appropriate intravenous agents. Also, expect delayed induction in sick neonates due to poor peripheral perfusion. Hence, it is important to have patience and not to exceed doses in order to achieve a rapid induction in an irritable child. The effects of shunting on the speed of inhalation induction is more pronounced for nitrous oxide than for the more soluble volatile anesthetics.\(^2\) Nitrous oxide is best avoided due to concerns over expansion of air emboli and cardiac depression. Certain lesions with increased pulmonary blood flow (PBF) need lower FiO\(_2\). Air is preferred over nitrous oxide to reduce the concentrations of oxygen.

With intravenous agents the induction would be rapid in right-to-left shunts as the systemic venous blood bypasses the pulmonary circulation and the drug reaches the brain sooner than predicted.

Strict de-airing of syringes and IV tubings should be done for patients with right-to-left shunts for the risk of air-embolism and also for patients with left-to-right shunts as there can be a transient reversal of shunt for a brief period in the cardiac cycle. Air filters can be used.

The authors have a routine protocol of giving IV premedication in the holding room (Ketamine 1 mg/kg, midazolam 0.1 mg/kg, glycopyrrolate 0.004 mg/kg) followed by sevoflurane (6-8%) with scented mask and IV Fentanyl 2-5 mcg/kg in the operating room. Pancuronium 0.1-0.2 mg/kg IV is used to facilitate intubation. Pancuronium has the benefit of counteracting the bradycardia caused by fentanyl. Maintaينence of anesthesia is done with isoflurane, fentanyl, midazolam and pancuronium boluses. Nasal intubation is preferred in neonates and children who would stay on the ventilator for longer duration postoperatively. This prevents accidental extubation in the ICU due to loosening of uncuffed endotracheal tube (ETT) fixation caused by oral secretions, especially during the weaning mode when the child is awake. However, oral intubation can be performed if cuffed ET tubes are used. In older and cyanotic children with higher hematocrits, oral intubation is preferred. Nasal intubation is avoided in many centers due to the risk of translocation of bacteria from the nasal passages directly to the lungs while passing the ETT.

### INDUCTION AGENTS

#### Opioids

The role of opioids to provide hemodynamic stability during the intraoperative period is well-proven. They attenuate the stress response associated with intubation, surgical incision, sternotomy, ETT suctioning and also blunt the pulmonary hypertensive response to prevent crisis in children with severe pulmonary hypertension. Techniques vary from usage of high-dose, sole-narcotic based induction with inhalational agent or low-dose narcotic with other intravenous induction agents and good analgesics intraoperatively. These include fentanyl, sufentanil, alfentanil and morphine.

#### Propofol

The use is justified in cardiac surgical cases needing fast-tracking and early recovery.\(^2\) It is not much popular in pediatric patients as it significantly decreases mean arterial pressure (MAP) and SVR, however CO, HR, PAP and PVR are maintained.\(^2\) In patients with intracardiac shunts the net result is significant increase in right to left shunt and significant decrease in left to right shunt due to fall in SVR. Propofol has no effect on SA and AV node therefore, it is desirable as primary agent during electrophysiological studies.\(^2\) Although, Propofol is used for cardiac catheterization and short-term ICU sedation, its use as long-term sedative in pediatric patients is not recommended. It can also be used in cases of PDA ligation to induce hypotension and thus reduce the ductal pressures to prevent accidental rupture of the duct during ligation.

#### Thiopental

It is well-tolerated in normovolemic patients with compensated congenital heart disease. However, it has to be used with caution and in reduced doses in patients with compromised circulatory function. Induction with Thiopentone in children decreases MAP due its mild negative inotropic effect and peripheral venodilation. The increase in HR that accompanies Thiopentone administration results from baroreceptor mediated stimulation of heart. Therefore, induction with Thiopentone in CHD is reserved for patients with good cardiac reserve and intact baroreceptor reflex. It is preferable to use it as an adjunct to reduce the requirements of opioids.

#### Etomidate

It has minimal effect on cardiovascular function. A bolus of 0.3 mg/kg in patients with CHD undergoing device closure and radiofrequency ablations showed...
no significant changes in HR, MAP, filling pressures, vascular resistances and Qp:Qs. Thus, Etomidate is best utilized in patients with limited cardiac reserve. Single dose of etomidate used for induction in pediatric patients undergoing cardiac surgery with CPB causes temporary adrenocortical suppression which is not significant as cortisol level returns to normal after 24 hours.

**Ketamine**

The sympathomimetic properties of ketamine are beneficial to maintain the contractility and SVR. Hence, it is useful in defects with right to left shunts like TOF to reduce the magnitude of shunt. Contrary to the traditional belief that ketamine increases the PVR, it has been noted that in the absence of hypoventilation (hypercarbia), a ketamine dose of 2 mg/kg intravenously does not increase PVR even in children with pulmonary hypertension. Use of ketamine is associated with emergence delirium, excessive salivation and increase in intracranial pressures. In spite of all these adverse effects ketamine is a popular anesthetic agent for short-term sedation during cardiac catheterization and minor ICU procedures.

**Dexmedetomidine**

It is a highly selective alpha-2 adrenergic agonist. It reduces the doses of other agents like opioids and inhalational agents. Dexmedetomidine should be used with caution in children with pre-existing hypotension and bradyarrhythmias requiring pacing as it causes bradycardia and interferes with pacemaker activities due to its negative chronotropic effects. However, these properties can benefit children with junctional ectopic tachycardia (JET) usually seen in cases of TOF postoperatively.

**Inhalational Agents**

Neonates and infants less than 6 months exhibit exaggerated myocardial depression and blood pressure reduction with inhalational agents especially with halothane. Halothane at 1 and 1.5 MAC causes significant myocardial depression resulting in a decline in MAP, ejection fraction (EF) and CO. Sevoflurane 1.5 MAC causes minimal myocardial depression resulting in slight fall in MAP, EF, CO and HR. Isoflurane at 1.5 MAC preserves myocardial function with no change in CO, EF, minimal fall in MAP and increases in HR. No significant differences have been found between effect of isoflurane and sevoflurane on cardiovascular parameters in children with CHD. Halothane, isoflurane and sevoflurane do not affect Qp:Qs ratio. Among all inhalational agents halothane is most arrhythmogenic. Sevoflurane has 6-12% incidence of atrial or junctional arrhythmia. Also, high dose sevoflurane induction is known to cause junctional bradycardia. Desflurane commonly produces tachycardia and hypertension in children during induction, followed by slight decrease in HR and systolic blood pressure at 1 MAC.

**Nitrous Oxide**

Its use in children with CHD is still controversial. A study has shown minimal reduction in HR, MAP and CI with the use of 50% Nitrous oxide; however mean PAP and PVR were not significantly changed. The use of nitrous oxide for its analgesic properties is negated by the use of opioids during cardiac surgery.

**Benzodiazepines and Muscle Relaxants**

The preferred benzo diazepine in children with CHD is Midazolam. It can be given as an oral premedication or intravenously in the operating room as an adjunct to other induction agents for amnesia.

The choice of specific muscle-relaxant is usually made on the basis of its predicted cardiovascular effects as well as its duration of action. Pancuronium is a popular choice for its vagolytic effect which supports the HR and CO, particularly in children who receive high dose opioids. If tachycardia is not desirable and the procedure is short in duration, atracurium, vecuronium or rocuronium can be used. Succinylcholine is rarely used in patients with CHD as it is associated with malignant hyperthermia, hyperkalemia, nodal rhythm, bradycardia and rarely asystole after intravenous use. The frequency of these arrhythmias increases with second dose to produce bradycardia or sinus arrest in children. If used, it should be preceded by, or combined with, atropine.

**CENTRAL LINES AND ARTERIAL LINES**

The dilemma about selection of catheters based on the patients age, weight and height is well-known. The authors follow a protocol for central venous (Table 6) and arterial catheters (Table 7) based on site, weight and age of the child in order to avoid confusion. Both catheters are inserted once the child is intubated and after appropriate positioning, keeping a close watch on the hemodynamics and temperature. The choice of the catheter site depends on the type of surgical procedure. The preferred site for central venous cannulation is right internal jugular vein (IJV) followed by left IJV, femoral vein and subclavian vein. IJV cannulation should be avoided in an operated case of Glenn shunt. For arterial cannulations in neonates, femoral artery is preferred; however the practice varies
in different institutions. Left radial artery cannulation should be avoided in cases of COA or left BT shunts as the left subclavian artery is clamped during these surgeries. Similarly, right radial artery should be avoided for right BT shunts.

**ANESTHETIC CONSIDERATIONS DURING CARDIOPULMONARY BYPASS**

The steps involved in the conduct of cardiopulmonary bypass (CPB) should be known, in order to intervene at the appropriate time and facilitate the smooth separation from CPB (Box 1). It is beyond the scope of this chapter to explain the routine steps involved in initiation and the components of CPB. The authors recommend standard CPB textbooks for the details. However, there are few important steps during this period which need special considerations and understanding, mentioned below.

- Cannula placement is critical as significant amount of blood loss can occur during cannulation of the aorta, SVC and IVC. Due to smaller blood volumes in children and systemic heparinization, it is important to have blood available in the operating room prior to cannulation to compensate for the hemodynamic instability caused by blood loss. Accidental dissections of aorta or puncture of the posterior wall due to the small intraluminal caliber can also occur. This can be confirmed by checking the CPB aortic line pressures by the anesthesiologist and the perfusionist. Similarly, the cardioplegia delivery pressure has to be monitored and maintained between 75–100 mm Hg.
- Moderate hypothermia (25–28°C) is generally employed in most of the cases. However, temperatures

### Table 6: Selection criteria for central venous catheter

<table>
<thead>
<tr>
<th>Age</th>
<th>Weight (kgs)</th>
<th>French</th>
<th>Length (cms)</th>
<th>Lumen</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neonate</td>
<td>1–5</td>
<td>3, 4, 5</td>
<td>6</td>
<td>Triple, dual</td>
</tr>
<tr>
<td>Infant</td>
<td>5–10</td>
<td>5, 5.5</td>
<td>8</td>
<td>Triple, dual</td>
</tr>
<tr>
<td>1–3 Years</td>
<td>10–20</td>
<td>5.5</td>
<td>11</td>
<td>Triple, dual</td>
</tr>
<tr>
<td>3–8 Years</td>
<td>20–30</td>
<td>5.5, 7</td>
<td>13</td>
<td>Triple, dual</td>
</tr>
<tr>
<td>&gt; 8 Years</td>
<td>30+</td>
<td>7</td>
<td>13</td>
<td>Triple</td>
</tr>
</tbody>
</table>

### Table 7: Selection criteria for arterial catheters

<table>
<thead>
<tr>
<th>Site</th>
<th>Age</th>
<th>Weight (kgs)</th>
<th>Gauge</th>
<th>Length (cms)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Femoral</td>
<td>Neonate</td>
<td>1–5</td>
<td>22</td>
<td>4, 6</td>
</tr>
<tr>
<td></td>
<td>Infant</td>
<td>5–10</td>
<td>22</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>&gt; 1 Year</td>
<td>&gt; 10</td>
<td>20</td>
<td>8</td>
</tr>
<tr>
<td>Radial</td>
<td>Neonate</td>
<td>1–5</td>
<td>24</td>
<td>1.9</td>
</tr>
<tr>
<td></td>
<td>1–4 Years</td>
<td>5–20</td>
<td>22</td>
<td>4.0</td>
</tr>
<tr>
<td></td>
<td>&gt; 4 Years</td>
<td>&gt; 20</td>
<td>20</td>
<td>3.2</td>
</tr>
</tbody>
</table>

**Box 1: Cardiopulmonary bypass checklist**

**Pre bypass checklist**
- Baseline ABG after intubation
- Aprotinin or tranexamic acid after confirming with the surgeon in cyanotics
- Give heparin 3 mg/kg in acyanotics and 4 mg/kg in cyanotics (1 mg = 100 units)
- Check ACT after 3 min and keep it above 480 sec
- Maintain arterial blood pressure on the lower side of normal during aortic cannulation
- Ventilate with 100% oxygen
- Check for air bubbles in the aortic line after cannulation

**Initiating bypass**
- Ensure all venous/arterial clamps are removed from the CPB circuit and patient end
- Check aortic line pressure and Check good venous return
- Clinically heart is empty
- Stop IV fluids, ventilation on full flows
- Record temperature to which patient is cooled
- If inotropes started pre-bypass, stop on bypass
- Bolus or continuous infusion of sedation and muscle relaxation

**Monitoring on bypass**
- Pupillary size should be small and equal
- MAP should be 30–50 mm Hg
- ABG done 5 mins after commencement of bypass and then every 30 mins
- Maintain ABG within normal limits
- Hematocrit maintained around 25% in children
- K+ maintained around 4.00–5.00 mEq/L
- ACT checked every hour and heparin administered to keep ACT >450 sec
- Cardioplegia every 20 min unless requested otherwise.
- Urine output 1–2 mL/kg/h
- Blood sugar every hour. Values upto to 200 mg% acceptable in children
- Record aortic cross clamp time and CPB time
- Keep forehead open and watch for congestion
- SNP/Sevoflurane/Sevoflurane continued on pump for uniform warming
- Rewarm slowly in order to keep an acceptable temperature gradient between the core and peripheral temperature

**Coming of Bypass Checklist**
- Is ABG satisfactory?
- Serum K+ levels are acceptable
- Na+, Cl-, Ca++ and Mg++ levels are normal
- PCV >30%
- Nasopharyngeal temperature >37°C
- Acceptable peripheral temperature
- Meticulous de-airing done
- Ventilation with 100% oxygen and both lungs inflated
- No bleeding and suctioning of pleural spaces done by surgeon
- Inotropes/Vasodilators on flow if required and dose calculated
- ECG pattern and pacemaker box and cables available
- Blood and blood products available
- Adequate surgical repair and myocardial function confirmed by TEE
- Watch for visual contractility of the heart
can be drifted down to as low as 18°C in some cases to facilitate reduction in the pump flows and provide bloodless field for the surgeon. For example, TOF repairs with increased blood return on the pump due to major aorto-pulmonary collateral arteries (MAPCAs) or TAPVC repairs. Deep hypothermic circulatory arrest (DHCA) or total circulatory arrest (TCA) is used in some centers for neonates and complex congenital cases. It allows more controlled complex surgery in a bloodless field with better myocardial protection as oxygen consumption falls 2–2.5 times per 10 degree fall in temperature. Methylprednisolone 30 mg/kg, Thiopentone 5 mg/kg and ice packs around head should be used during DHCA.

- Monitor two temperatures on CPB: Rectal and nasopharyngeal (brain temperature). Monitor the nasopharyngeal temperature during rewarming as the rectal temperature is 1°C less than the brain temp; which means that when the rectal temperature is 37°C, the brain temperature would already be 38°C. It is best to stop rewarming at a nasopharyngeal temperature of 35.5–36°C and wean the CPB. This holds true especially in cases of TOF where junctional ectopic tachycardia (JET) is sometimes seen in the postoperative period due to extensive resection of the right ventricle, worsened further by hyperthermia. Adequate and uniform cooling as well as rewarming can also be ensured if both the temperatures coincide throughout the CPB period. This can be facilitated by titrating doses of sodium nitroprusside (1–5 μg/kg/min) or isoflurane on pump.

- Use of ultrafilters has significantly improved the outcomes in sick children undergoing complex surgeries. Ultrafiltration is a technique which removes excess fluid volume from the circuit in the pre-CBP period (pre-ultrafiltration/PUFF), improves hematocrit on the CPB (conventional ultrafiltration/CUFF) and removes inflammatory mediators in the post-CBP period (modified ultrafiltration/MUFF) after separation from the CPB machine. MUFF improves hemodynamics as seen by improvement in systemic blood pressure and reduction in the PAP. Temperature has to be monitored because it can drift down during MUFF as most of the ultra-filtration circuits do not have heat-exchangers.

- When milrinone infusion is used, a bolus of 50 μg/kg should be given on CPB at 34°C during the rewarming phase. This facilitates vasodilation on the CPB ensuring uniform rewarming and prevents profound hypotension caused by the bolus, if given in the post-CBP period. Infusion of 0.5–0.75 μg/kg/min can then be safely continued.

### DIFFICULTY IN WEANING CPB

The identification of residual defects or issues mentioned below and rectifying them before leaving the operating room significantly reduces the morbidity. Following are some of the common problems encountered:

- **Inadequate repair-resection or residual lesions:** It can be picked up on transoesophageal echocardiography (TEE), by direct needle pressures taken by the surgeon (RV, LV, PA, Ao, LA) or by step-up saturations on ABG taken from right atrium and pulmonary artery, e.g. TOF repairs, VSD closures.

- **Pulmonary arterial hypertension (PAH):** When the pulmonary artery pressures are systemic or supra-systemic as seen by direct PA line monitoring, e.g. TAPVC repair, truncus arteriosus repair.

- **Left or right ventricular dysfunction:** LV dysfunction secondary to prolonged pump run or ischemia due to coronary issues, e.g. arterial switch operations for TGA. This can manifest as pulmonary edema and bleeding in the ETT. RV dysfunction secondary to excessive RV resection, e.g. TOF repairs or high PA pressures, e.g. TAPVC repair.

### ANESTHETIC CONSIDERATIONS FOR COMMON LESIONS

Generally, surgeries performed for CHD offer either palliation or total correction of the lesion. Most of these defects if diagnosed early can be repaired in infancy itself. Anesthesia can be planned according to the nature of the planned surgery. The steps involved in the most commonly performed operations are given in Table 8.

The two important factors determining whether to offer palliation or total correction are the type of the lesion and the age of presentation. Palliative surgeries include procedures which restore the physiological functions of the circulation rather than anatomical corrections. Hence, they are performed either to increase the pulmonary blood flow (Blalock-Taussig shunt, Glenn shunt, Fontan surgery) or to decrease the pulmonary blood flow (PA band). Certain palliative procedures that improve the intracardiac mixing include balloon atrial septostomy (TGA, obstructed TAPVC) and can be performed in cardiac catheterization laboratory.

Most of the total corrections are performed in the neonatal age group or by one-year of age. As explained earlier, the basic physiological differences in the neonatal myocardium should be taken into consideration during administration of anesthesia. This group includes arterial switch operation for TGA, TAPVC repair and
**Chapter 30: Anesthesia for Pediatric Cardiac Surgery**

**Table 8: Surgical procedures in some common congenital heart defects**

<table>
<thead>
<tr>
<th>Common lesions</th>
<th>Surgical procedure performed</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Open Heart Surgeries (on CPB)</strong></td>
<td></td>
</tr>
<tr>
<td>ASD, PAPVC</td>
<td>Direct closure or pericardial patch closure, rerouting of the anomalous pulmonary vein/veins to left atrium by suturing the ASD patch in a way that the pulmonary venous drainage is redirected to left atrium</td>
</tr>
<tr>
<td>VSD</td>
<td>Pericardial patch closure via right atriotomy (perimembranous), other approaches include closure via main pulmonary artery (subpulmonic VSD) or left ventriculotomy (large apical muscular VSD)</td>
</tr>
<tr>
<td>AV canal</td>
<td>Pericardial 2-patch technique of ASD and VSD closure with repair of the atrioventricular valves</td>
</tr>
<tr>
<td>TOF</td>
<td>Corrective-VSD closure, right ventricular outflow widening by pericardial patch and infundibular resections with/without patch pulmonary artery augmentations</td>
</tr>
<tr>
<td>TAPVC</td>
<td>Total correction of rerouting all the pulmonary veins into left atrium by suturing of a common chamber in which all 4 veins open to the left atrium directly depending on the type (supracardiac, cardiac, infracardiac, mixed)</td>
</tr>
<tr>
<td>TGA</td>
<td>Corrective - Arterial switch operation involves switching of the great arteries to their respective ventricles with reimplantation of coronary arteries in the neo-aorta (LeCompte Technique) with/without VSD closure Palliative – Sennings and Mustards in older children involves redirecting the blood flow from the atrial chamber to the respective ventricles over baffles (patients own pericardium or Goretex)</td>
</tr>
<tr>
<td>Truncus Arteriosus</td>
<td>Resection of the pulmonary artery from the aorta and reimplanting it on the right ventricle directly or via a valved conduit depending on the type (Type 1-4 Collet-Edwards)</td>
</tr>
<tr>
<td>Valve Atresias</td>
<td>Palliation by conversion to single ventricle physiology by performing bidirectional Glenn shunt (suturing the SVC to Pulmonary artery) and subsequently Fontan surgery, sometimes even primary extracardiac (directing the IVC flow to the PA by a Goretex tube)</td>
</tr>
<tr>
<td><strong>Closed Heart Surgeries (Without CPB)</strong></td>
<td></td>
</tr>
<tr>
<td>PDA</td>
<td>Ligation of the duct with suture or clipping via left thoracotomy</td>
</tr>
<tr>
<td>COA</td>
<td>Resection of the coarcted segment with end-to-end anastomosis or subclavian artery flap plasty or anastomosis of an interposition graft if the resected segment is long via left thoracotomy</td>
</tr>
<tr>
<td>TOF</td>
<td>Palliative- Modified Blalock Taussig shunt with Goretx grafts sutured between subclavian artery and branch pulmonary artery (midline sternotomy or thoracotomy)</td>
</tr>
<tr>
<td>PA band</td>
<td>Application of a mersilene tape based on Trusslers formula on the PA to restrict the pulmonary blood flow (midline or thoracotomy)</td>
</tr>
</tbody>
</table>

**Abbreviations:** ASD, atrial septal defect; VSD, ventricular septal defect; PAPVC, partial anomalous pulmonary venous connection; TOF, tetralogy of Fallot; TAPVC, total anomalous pulmonary venous connection; TGA, transposition of great vessels; PDA, patent ductus arteriosus; COA, coarctation of aorta; CPB, cardiopulmonary bypass; SVC, superior vena cava; PA, pulmonary artery.

Truncus arteriosus repair. Total correction for TOF, VSD is usually offered by 3 months of age although some centers are performing it as early as one month. Such centers are usually geared with extracorporeal membrane oxygenation (ECMO) as a back-up for difficult weaning from CPB.

With the thorough knowledge of the cardiovascular effects of anesthetic drugs provided under the section of choice of anesthetic drugs and induction agents; and with the knowledge of classifying the heart lesion on the basis of shunt direction, level of obstruction and the play between SVR-PVR, it is extremely simple to choose the appropriate drugs for induction and maintenance of anesthesia. Also, based on the need to reduce the SVR or PVR in the post-bypass period, the choice of catecholamines can be simplified (Table 9). Thus, the choice of the anesthetic agents, drugs and catecholamines depends on the hemodynamic changes desired in the perioperative period for the given lesion under treatment.

The factors and drugs that reduce or increase the PVR and SVR should be remembered. An anesthesiologist can to some degree manipulate the magnitude of intracardiac shunting by altering the PVR and SVR.

**Factors that Increase PVR**
- Hypoxia
- Hypercarbia
- H+ acidosis
- Hyperinflation (PEEP)
- Hypothermia
- Atelectasis
- Sympathetic stimulation

**Factors/ Drugs that Decrease PVR**
- Hypoxia
- Hypocarbia
### Table 9: Commonly used cardiac drugs in surgeries for congenital heart defects

<table>
<thead>
<tr>
<th>Drug</th>
<th>Mechanism of action</th>
<th>Use</th>
<th>Dose</th>
<th>Adverse effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epinephrine</td>
<td>α and β receptor agonist, low dose-β2 agonist medium dose-α1 and β1 agonist, high dose –α1 agonist</td>
<td>LCOS, systolic dysfunction</td>
<td>0.05–0.2 µg/kg/min</td>
<td>Tachycardia, arrhythmia</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>Potent α action</td>
<td>BT –shunt, LCOS with low SVR</td>
<td>0.01–0.3 µg/kg/min</td>
<td>Hypertension, reflex bradycardia</td>
</tr>
<tr>
<td>Dopamine&lt;sup&gt;41&lt;/sup&gt;</td>
<td>Dopaminergic, α and β receptor agonist, low dose- DA 1 and DA 2 agonist, medium dose- DA 1 and β1 agonist, high dose—α agonist</td>
<td>Provides modest inotropic support</td>
<td>2–20 µg/kg/min</td>
<td>Tachycardia, AV conduction abnormality, pulmonary vasoconstriction</td>
</tr>
<tr>
<td>Dobutamine&lt;sup&gt;41&lt;/sup&gt;</td>
<td>Selective β1 and β2 agonist</td>
<td>Prevention and treatment of LCOS</td>
<td>2–15 µg/kg/min</td>
<td>Tachycardia, arrhythmia</td>
</tr>
<tr>
<td>Milrinone&lt;sup&gt;42&lt;/sup&gt;</td>
<td>PDE-III inhibitor, inodilator increases myocardial contractility and decreases SVR</td>
<td>Prevention and treatment of LCOS</td>
<td>Loading: 50–75 µg/kg F/b 0.5–0.75</td>
<td>Tachycardia, hypotension, arrhythmia</td>
</tr>
<tr>
<td>Levosimendan&lt;sup&gt;43&lt;/sup&gt;</td>
<td>Calcium sensitizer and K-ATP channel opener, inodilator, increases myocardial contractility without increasing oxygen consumption, decreases SVR</td>
<td>Management of LCOS, CCF, cold shock in sepsis</td>
<td>Loading: 6–12 µg/kg F/b 0.05–0.2</td>
<td>Hypotension, arrhythmias rarely</td>
</tr>
<tr>
<td>Vasopressin&lt;sup&gt;44&lt;/sup&gt;</td>
<td>Acts on V1 receptors and produces vasoconstriction in skeletal, splanchnic vasculature</td>
<td>Catecholamine resistant vasodilatory shock</td>
<td>0.0001–0.0003 U/kg/min</td>
<td>Severe vasoconstriction</td>
</tr>
<tr>
<td>Sodium nitroprusside (SNP)&lt;sup&gt;41,45&lt;/sup&gt;</td>
<td>Interacts with oxy-Hb and releases nitric oxide which causes vasodilation through cGMP</td>
<td>Post coarctation HTN, for uniform cooling and rewarming on CPB</td>
<td>0.3–5</td>
<td>Severe hypotension, cyanide toxicity @ dose &gt;2 mcg/kg/min.</td>
</tr>
<tr>
<td>Amiodarone&lt;sup&gt;41&lt;/sup&gt;</td>
<td>Class –III antiarrhythmic, prolongs effective refractory period in all cardiac muscles</td>
<td>Supraventricular tachycardia (SVT) Junctional ectopic tachycardia (JET), Ventricular tachyarrhythmia (VT) ventricular fibrillation (VF) torsades de pointes</td>
<td>Loading: 5 mg/kg over 30 min. F/b 5–15 µg/kg/min</td>
<td>Hypotension, bradycardia, pulmonary alveolitis, long QTc syndrome, Thyroid dysfunction</td>
</tr>
<tr>
<td>Esmolol&lt;sup&gt;45&lt;/sup&gt;</td>
<td>Selective β1 adrenergic antagonist</td>
<td>Hypercyanotic spell in TOF, post coarctation HTN</td>
<td>Loading: 500 µg/kg over 20 min F/b 50–200 µg/kg/min</td>
<td>Hypotension, bradycardia, contraindicated in asthma</td>
</tr>
<tr>
<td>Phenylephrine&lt;sup&gt;46&lt;/sup&gt;</td>
<td>Directly acting synthetic non –catecholamine, acts on α1 adrenergic receptor</td>
<td>Hypercyanotic spell in TOF, topically applied as nasal decongestant</td>
<td>Intermittent boluses- 1 µg/ kg infusion – 0.1 – 0.5 µg/kg/min</td>
<td>Reflex bradycardia</td>
</tr>
<tr>
<td>Lignocaine&lt;sup&gt;46&lt;/sup&gt;</td>
<td>Class- I B antiarrhythmic, shortens action potential duration and refractory period</td>
<td>Suppression and treatment of ventricular tachyarrhythmia</td>
<td>Loading: 1 mg/kg F/b 20–50 µg/kg/min</td>
<td>High dose causes myocardial depression, bradycardia</td>
</tr>
<tr>
<td>Sildenafil&lt;sup&gt;41&lt;/sup&gt;</td>
<td>PDE –V inhibitor increases cGMP and causes relaxation of vascular smooth muscles</td>
<td>Pulmonary hypertension</td>
<td>IV infusion 0.067 mg/kg/hr oral dose 0.5–2 mg/kg/dose every 6 hourly</td>
<td>Headache, flushing, diarrhea, vomiting</td>
</tr>
<tr>
<td>Heparin&lt;sup&gt;41&lt;/sup&gt;</td>
<td>Heparin binds with Antithrombin and enhances its activity by 1,000 times</td>
<td>For on-pump and off-pump cardiac surgery, postoperative BT shunt</td>
<td>On pump surgery 400 U/kg Off pump surgery 200 U/kg Post-op BT shunt 10–20 U/kg/h infusion</td>
<td>Bleeding, thrombo-cytopenia, allergy</td>
</tr>
</tbody>
</table>

Note: β1 receptors (heart) stimulation has positive inotropic, chronotropic and dromotropic effect. β2 receptor (blood vessel) stimulation has vasodilatory effect. α receptor (blood vessel) stimulation has vasoconstrictory effect.

Abbreviation: F/b, followed by
Chapter 30: Anesthesia for Pediatric Cardiac Surgery

3

Factors/Drugs that Increase SVR

- Sympathetic stimulation
- Alpha-adrenergic agonists (Table 9).

Drugs that Decrease SVR

- Anesthetic agents (Refer to the section of anesthetic drugs)
- Vasodilators (Table 9)
- Alpha-adrenergic antagonists (Table 9)
- Beta-adrenergic antagonists (Table 9).

Managing Cyanotic/Hypercyanotic/Tet Spell

Clinical Profile

- Profound cyanosis and hyperpnea, followed by limpness, convulsions and loss of consciousness
- Peak incidence is at 1–3 months of age
- Usually lasts for 15–60 minutes, but may extend to hours
- Episodes occur primarily in the morning at awakening. This is because the vulnerable respiratory center which is sensitive after prolonged sleep in the night reacts to sudden increase in cardiac output and heart rate, if the child cries. This increases the venous return which further increases the right-left shunt, diverting the deoxygenated blood away from the lungs leading to desaturation.
- Frequently induced during cardiac catheterization.
- May be initiated by crying, feeding and bowel movements
- Spontaneous regression of spells may occur in some children after 3–6 years, whether or not surgery is performed. This is due to anatomic (MAPCA’s) and physiologic adaptation of the child to chronic hypoxia.

Postulated Mechanisms

- Infundibular spasm or hypercontractility
- Paroxysmal hyperpnea with increased right to left shunt
- Postural changes with volume redistribution
- Sudden shift in SVR: PVR ratio (drop in SVR secondary to hypoxia)
- Right ventricular mechanoreceptors.

Acute Treatment

- Supplemental oxygen
- Knee-chest position
- Morphine
- Ketamine (1 mg/kg IV bolus)
- Sodium bicarbonate (1 mL/kg IV bolus)
- Volume (Crystalloid bolus NS 5 mL/kg IV)
- Beta-blockers (Esmolol: loading dose 500–600 µg/kg iv over 2 minutes followed by infusion of 200 µg/kg/min)
- Phenylephrine 10 mcg/kg i.v boluses. (Dilute 1 ampoule/1 mL = 10 mg in 9 mL NS in syringe. Take 1 ml of this dilution and further dilute it in 9 mL NS in syringe. This makes the dilution to 100 µg/mL)
- Urgent BT shunt.

POSTOPERATIVE CARE IN PEDIATRIC CARDIAC PATIENT

The field of pediatric cardiac intensive care continues to evolve due to collaborative effort from anesthesia, surgery, cardiology, and critical care. The general principle is to anticipate potential problems by understanding the lesion and postoperative physiology (especially in the palliative corrections) as well as the common postoperative issues. The commonly encountered complications with their management are described below.

Bleeding

The causes of postoperative bleeding are anastomotic site bleeding, pre-existing coagulopathy and CPB induced coagulopathy. Most of the centers have fixed their limits for intervention in case of ongoing bleeding (Table 10). The protocol for management of on-surgical bleeding is mentioned below (Table 11). The authors routinely use tranexamic acid in cyanotic heart defects in anticipation to bleeding. A regimen of IV tranexamic acid bolus 3 times in a dose of 10 mg/kg prior to skin incision, on CPB and after protamine is followed. In rare cases iv bolus of

Table 10: Interpretation based on chest-tube drainage

<table>
<thead>
<tr>
<th>Quantity of drainage</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1–4 mL/kg/h</td>
<td>Acceptable</td>
</tr>
<tr>
<td>5–10 mL/kg/h</td>
<td>Clotting problem / surgical bleeding—be watchful</td>
</tr>
<tr>
<td>&gt;10 mL/kg/h</td>
<td>Surgical bleeding – Reexploration</td>
</tr>
</tbody>
</table>
Table 11: Interventions based on investigations

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Target</th>
<th>Intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin</td>
<td>Acyanotic &gt;10 g/dL</td>
<td>Packed RBC 10 mL/kg or 1:1 replacements for the loss</td>
</tr>
<tr>
<td></td>
<td>Cyanotic &gt;12 g/dL</td>
<td></td>
</tr>
<tr>
<td>PT, aPTT</td>
<td>&lt;1.5 times normal</td>
<td>FFP 10–20 mL/kg</td>
</tr>
<tr>
<td>Fibrinogen</td>
<td>&gt;0.75 g/L</td>
<td>Cryoprecipitate 5 mL/kg</td>
</tr>
<tr>
<td>Platelet counts</td>
<td>&gt;1 lakh/dL</td>
<td>Platelets 10 mL/kg (single donor preferred)</td>
</tr>
</tbody>
</table>

Abbreviation: PT, prothrombin time; aPTT, activated partial prothrombin time; PRBC, packed red blood cells; FFP, fresh frozen plasma

Table 12: Clinical, laboratory and hemodynamic parameters for identification of LCOS

<table>
<thead>
<tr>
<th>Physical examination</th>
<th>LCOS</th>
<th>Adequate cardiac output</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peripheral perfusion</td>
<td>Poor capillary refill</td>
<td>Good capillary refill &lt;3 sec</td>
</tr>
<tr>
<td>Core/peripheral gradient</td>
<td>&gt;3</td>
<td>&lt;3</td>
</tr>
<tr>
<td>Pulses</td>
<td>Weak</td>
<td>Good peripheral pulses</td>
</tr>
<tr>
<td>Urine output</td>
<td>&lt;1 mL/kg/h</td>
<td>&gt;1 mL/kg/hr</td>
</tr>
<tr>
<td>Mental status</td>
<td>Disoriented</td>
<td>Cooperative</td>
</tr>
<tr>
<td>Arterial waveform</td>
<td>Small area under curve</td>
<td>Large area under curve</td>
</tr>
<tr>
<td>Metabolic acidosis</td>
<td>Base excess &gt;5 mmol/L</td>
<td>Base excess &lt;5 mmol/L</td>
</tr>
<tr>
<td>Lactate</td>
<td>&gt;4 mmol/L</td>
<td>&lt;2 mmol/L</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>Normal or low</td>
<td>Normal</td>
</tr>
<tr>
<td>Rhythm</td>
<td>Tachycardia or bradycardia</td>
<td>Normal sinus rhythm</td>
</tr>
<tr>
<td>Mixed venous oxygen saturations</td>
<td>Low or decreasing trend</td>
<td>Normal</td>
</tr>
</tbody>
</table>

recombinant factor VIIa can be used in a dose of 10–30 µg/kg. TEG or sonoclot should be considered for diagnosing the cause of bleeding and appropriate blood products should be replaced. Also ensure that patient is not hypertensive as this will worsen the bleeding. Warm up the patient to 36.5°C as clotting cascade is more effective at normal temperature.

**Arrhythmias**

Postoperative arrhythmias occur commonly (15–48%) following pediatric cardiac surgery and can result in significant hemodynamic deterioration. Arrhythmias are more likely in the immediate postoperative period due to direct injury to the conduction system, myocardial injury, ischemia, high catecholamine levels, electrolyte disturbances, scars, sutures and stress response. Common, post-operative arrhythmias include:

**Junctional Ectopic Tachycardia**

Junctional ectopic tachycardia (JET) is self-limiting tachycardia, occurs within 72 hours and resolves within 8 days. ECG shows narrow QRS complex at rate of 170–260/min and AV dissociation where atrial rate is slower than ventricular rate. ECG may show retrograde 1:1 ventriculoatrial conduction. If P wave is not visible, atrial ECG can be performed. If diagnosis of JET is not clear, Adenosine can be used to differentiate JET from other supraventricular tachycardia. Adenosine does not affect the ventricular rate in JET. It is commonly seen in postoperative cases of TOF, VSD, AVSD, ASO, and TAPVC. Management includes correction of acidosis, hypovolemia, hypomagnesemia, hypokalemia, hypocalcemia, core body cooling to 33°C, sedation and paralysis to decrease oxygen demand. A loading dose of Amiodarone (5 mg/kg over 20 min) can be started followed by continuous infusion (5–15 µg/kg/min). Atrial over drive pacing can be used to break JET.

**Supraventricular Tachycardia**

Supraventricular tachycardia (SVT), which is hemodynamically stable is managed with vagal maneuvers (carotid sinus massage), adenosine 0.1 mg/kg or amiodarone. However, if it is unstable cardioversion 1–2 J/kg has to be considered.

**Complete Heart Block**

Complete heart block (CHB) is common after surgical closure of large VSD, AVSD, DORV, subaortic AS due to
injury to conduction system. It is a standard practice to wait for 10 to 14 days for the normal sinus rhythm to return. During this period temporary epicardial pacing is done and steroids are given to decrease edema near conduction tissue. If normal sinus rhythm doesn’t return within 14 days, permanent pacemaker is to be implanted.

**Low Cardiac Output Syndrome**

Low cardiac output syndrome (LCOS) is most important cause of morbidity and mortality in the early postoperative period. The risk is greatest for neonates and infants undergoing complex surgical repair (ASO, TAPVC, Fontan, TOF repair, large VSD). Main risk factors are prolonged aortic cross clamp, reperfusion injury, inflammatory response to CPB, pre-existing ventricular dysfunction, inadequate myocardial protection, residual defect, extensive suture line bleeding etc. Early identification of changes in the clinical, laboratory and hemodynamic parameters for this complex syndrome (Table 12) with timely interventions are often rewarded by positive outcomes.

Since, \[ BP = CO \times SVR \] and \[ CO = HR \times SV \]

\[ BP = HR \times SV \times SVR \]

Hence, all the three components; heart rate, stroke volume and SVR are extremely important to maintain the blood pressure and avoid the LCOS. Heart rate should be increased by AV sequential pacing, isoprenaline to maintain sinus rhythm and AV synchrony. Stroke volume can be increased by increasing force of myocardial contraction with catecholamines, phosphodiesterase inhibitors and digoxin resulting in more effective ventricular ejection. Inodilators like dobutamine, milrinone, levosimendan are generally helpful in LCOS with elevated SVR due to vasoconstriction, whereas dobutamine and norepinephrine is a preferred combination in LCOS with low SVR situation. Some recommend the prophylactic use of high loading dose of milrinone 75 µg/kg followed by 0.75 µg/kg/min for 35 hours postoperatively to significantly reduce the risk of LCOS.

**Pulmonary Hypertension Crisis**

Paroxysmal increase in pulmonary artery (PA) pressures exceeding systemic pressures causes low cardiac output and this phenomenon is called as pulmonary hypertensive crisis (PHC). This is typically seen in lesions with left-to-right shunts postoperatively. Common conditions being AP window, Truncus, AVSD, VSD, TGA, TAPVC, cor-triatriatum. The triggering factors include fall in arterial or alveolar oxygen, rise in CO₂ metabolic acidosis, pain, tracheal suctioning, lung infection and stimulation in partially sedated child. It manifests as elevation in PA pressures and CVP, hypotension, desaturation, bradycardia and increase in the peak airway pressure. Treatment includes:

- Manual hyperventilation with 100% FiO₂ using short inspiratory and long expiratory time
- Correction of metabolic acidosis & maintenance of pH between 7.40–7.45 with normocarbia
- Tablet sildenafil (2 mg/kg) via nasogastric tube
- Start inhalation of nitric oxide iNO (20–80 ppm) which is a selective pulmonary vasodilator and continue for at least 48 hours postoperatively
- Increase milrinone infusion up to 0.8–1 µg/kg/min to reduce the PVR
- Sedate with fentanyl, midazolam and paralyze with muscle relaxant
- Recent use of IV sildenafil infusion (0.007 mg/kg/h; max 1.6 mg/kg/day)
- IV Prostacycline 5–20 µg/kg/min infusion and IV magnesium sulfate 25–50 mg/kg/dose slowly over 1 hour every 6–8 hourly can be considered for their pulmonary vasodilatory properties
- Sternum to be left open: Delayed sternal closure once the heart and lung edema settles 24–48 hours postoperatively. This may again cause a rise in PAP, hence medications for reducing PAH should be continued in ICU.
- Tablet Bosentan 5 mg/kg OD can be added on the next day after confirming normal liver function tests
- Patent foramen ovale—Atrial level shunt left open by surgeon in anticipation, so expect desaturation due to R-L shunting; can expect lower PO₂ on ABG
- If no PA lines, watch for PFO shunting on TEE.

**ANESTHESIA FOR CLOSED HEART PROCEDURES**

These procedures do not need CPB as they are performed on extracardiac structures (mainly arteries and veins) and do not warrant the need for opening of the cardiac chambers. Most of the surgeries under this category are performed via lateral thoracotomy. Hence, one has to remember the possible complications involved with the lateral positions and the physiology of one-lung ventilation (described in other sections of the book). Procedures like PDA ligation, COA repair, PA band and sometimes left BT shunts are performed via left thoracotomy. Right BT shunts are performed via right thoracotomy. With the advent of BT shunts performed in the neonatal age group, majority of the centers perform midline sternotomy for BT shunts. The benefits of midline BT shunt surgery are:
1. Reduction of the episodes of desaturation, hypercarbia, atelectasis caused by mechanical compression of the lungs with retractors during shunt placement via thoracotomy, thereby avoiding fluctuations in the PVR.

2. The surgeon has the choice to choose either of the pulmonary arteries for shunt placement based on his visual assessment of its dimensions.

3. Easier to perform pulmonary artery plasties if needed along with BT shunts.

4. Better access to the PDA if performing a right BT shunt (PDA is difficult to access from the right thoracotomy). This is important if the PDA needs to be ligated in case of shunt overflow.

5. Easier access for cannulation to go on emergency CPB in cases of excessive desaturation or hemodynamic instability after clamping of pulmonary arteries.

Other closed heart procedures performed via midline sternotomy are Glenn shunts (Palliative procedure involving anastomosis of the superior vena cava to the right pulmonary artery for atretic lesions of the right heart to divert venous blood to the lungs), occasionally PA bands for the reasons similar to one mentioned above and permanent pacemaker insertion.

The authors can describe the anesthetic management of each procedure, however one can easily remember certain principles involved in closed heart procedures. The authors recommend some salient practical points to be remembered based on their clinical practice, as given below:

1. Complications of thoracotomy position like compression injuries to nerves, eyes and pressure points are to be avoided.

2. Central venous access and arterial line access to be secured with proper dressings and the back-flow of blood confirmed after positioning.

3. Auscultation of both lungs for confirming bilateral air-entry and detection of accidental displacement of the tube too far in or too far out.

4. As mentioned earlier most of the procedures involve application of clamps on arteries and veins in order to facilitate surgical anastomosis and prevent blood loss. This simply means if the clamps are placed on the arterial side-aorta (COA repair) one would see brief periods of hypertension proximal to the clamp which can be managed by vasodilators (Table 9) followed by reperfusion phase marked by hypotension which can be managed by IV boluses of Injection Calcium chloride 10–20 mg/kg slowly over 5–10 mins, Injection Sodium bicarbonate 1 mEq/kg, Injection Phenylephrine 1–2 μg/kg, fluids (Normal Saline) and rarely a vasopressor infusion (Table 9). Also, mild hypothermia (35–35.5°C) is preferred for protection of the organs distal to clamp.

5. Similarly, if the clamps are placed on the venous side (Glenn shunt) one would have to watch for signs of venous congestion over the face and assess the neurological status postoperatively. Some centers prefer doing these shunts on CPB as they may involve atrial septectomy for mixing at the atrial level.

6. If the clamps are placed on the arterial side-pulmonary (BT shunt) one would see brief periods of desaturation which can be managed by hyperventilation with 100% oxygen, boluses of atropine for bradycardia and sodium bicarbonate for metabolic acidosis caused by desaturation.

7. Due to the involvement of clamps and arteriotomies, Heparin is needed in reduced doses 1 mg/kg to maintain the ACT around 200 seconds and thus prevent thrombosis in the vessels due to stasis leading to shunt blockages.

8. The target saturations on the pulse oximeter should be between 75–85% for BT shunts, Glenn shunts and PA bands, the FiO₂ has to be adjusted accordingly.

**ANESTHESIA FOR CARDIAC CATHETERIZATION**

In recent times due to refining in the catheter-based techniques, the need for corrective cardiac surgeries for simpler lesions is slowly but steadily declining. Other benefits include providing data in complex cases with inadequate information derived from echocardiography to help the surgeons before operation. It is therefore important for an anesthesiologist to understand his role in the cardiac catheterization laboratory. Most aspects about challenges faced by the anesthesiologist are due to the laboratory’s remote location and restricted space similar to MRI or endoscopy suites (would be covered in other sections). The authors therefore limit themselves to the practical aspects of providing anesthesia for CHD in the catheterization laboratory. Detailed preoperative assessment for fitness should be done. Investigations can be limited to a hemoglobin, total leukocyte counts, viral markers, blood urea nitrogen and serum creatinine. Blood should be reserved for patients undergoing transcatheter device placements. Interventions can be divided into:

1. Diagnostic
2. Therapeutic
3. Electrophysiological studies and ablation techniques.
Diagnostic Procedures

They are performed in the preoperative period and occasionally in the postoperative period:

- To define the anatomy in complex cardiac diseases
- To measure pressure in different chambers of heart, pulmonary artery and pulmonary veins
- To derive hemodynamic information such as PVR, SVR, CO.

The usual approach for diagnostic catheterization is through femoral vein and femoral artery, however in patients undergoing Fontan surgery, catheterization of IJV is preferred to study pulmonary vasculature and PA anatomy (as the SVC is connected to the PA via a Glenn shunt which is invariably performed prior to Fontan surgery except in cases of primary Fontan). One must also remember and discuss with the cardiologist the need for reduction of FiO2 whenever necessary. This is vital to avoid errors in hemodynamic calculations based on saturations and pressures. For example, if a child with large left to right shunt receives high FiO2, PVR would significantly drop and there would be a resultant increase in Qp:Qs. Therefore, the key is to maintain patient on room air, avoid airway obstruction and hypercarbia especially during diagnostic catheterization procedures.

Most of diagnostic procedures are done under sedation. The authors perform these procedures under sedation with glycopyrrolate, ketamine, midazolam and fentanyl in titrated doses. However, anesthetics like propofol, etomidate and opioids can also be used. It is interesting to note that a switch from positive to negative pressure ventilation increases CO by 11% in healthy children and by 28% in postoperative cardiac patients.

Therapeutic Procedures

The following procedures can be done for correction of certain heart defects:

- Transcatheter device closure of PDA, Ostium secundum ASD, VSD
- Balloon dilatation of cardiac valves of pulmonary, aortic and mitral valvuloplasties
- Balloon angioplasty of neonatal COA, pulmonary arteries
- Stents placements in recoarctation of aorta, systemic venous stenosis, pulmonary artery stenosis
- Balloon atrial septostomy for TGA, TAPVC, tricuspid atresia, mitral atresia
- Coil occlusion of AP collaterals, pulmonary AV fistula and veno-venous collaterals.

Majority of the therapeutic procedures are carried out under sedation with or without the use of laryngeal mask airway (LMA). However, intubation should be performed if TEE has to be used, especially in the cases of device closures and extubation performed at the end of procedure in the catheterization laboratory. This warrants the use of short-acting anesthetic agents and muscle-relaxants.

Possible complications include dislodgement of devices leading to catastrophes and urgent surgical intervention if the devices occlude the left ventricular outflow, bradyarrhythmias especially in VSD device closures and during dilatation of arteries, tachyarrhythmias due to catheter irritation, blood loss and hypothermia especially in neonates.

Electrophysiological (EP) Studies and Ablation Techniques

The purpose of electrophysiological (EP) studies is to identify the mechanism of arrhythmias by recording signals from electrodes placed within the heart. Usually children presenting for EP studies are healthy with well-tolerated SVT. They rarely present with life-threatening arrhythmias. Pacing via high RA and apical RV lead allows arrhythmias to be provoked or terminated, thus permitting the measurement of EP properties of conduction tissue. If CHB occurs during procedure, ventricular pacing (VVI) can be done. Medications commonly used to define EP studies are:

i. Adenosine—blocks the AV conduction exposing concealed abnormal pathway.

ii. Isoprenaline—increases SA node rate and speeds up AV conduction and reduces the refractory period and increases the automacity of other contractile tissue.

Once diagnosed, these abnormal pathways and automatic foci need to be destroyed by radiofrequency ablation in order to abolish the arrhythmias. EP studies are generally done under sedation. Antiarrhythmic drugs should be ‘discontinued’ before EP study. Anesthetic agents and techniques that can cause suppression of the arrhythmias should be avoided. Number of clinical studies have shown no direct effect of Propofol on conduction tissue at doses 100–150 µg/kg/min. The usual strategy is to avoid high doses of narcotics and propofol as well as to limit the dose of inhalational agents during induction of arrhythmia and replace the endogenous sympathetic activity with sympathomimetics like isoprenaline.

ANESTHESIA FOR CARDIAC RADIOLOGY PROCEDURES

Recently, imaging studies have emerged as an important noninvasive modality in confirming the diagnosis of certain
complex CHD. These include computed tomography (CT) angiographies and magnetic resonance imaging (MRI). CT angiography helps to define the complex anatomy of blood vessels, relation of the cardiac chambers, abnormal branching patterns of the arterial system and the extent of the collateral circulation apart from the diagnosis made by 2-D echo. In addition to the above, MR angiography gives information about the systemic to pulmonary blood flow ratio, cardiac output, stroke volume, ventricular volumes and ejection fraction.60

The decision to administer general anesthesia or sedation depends on the duration required to perform these studies. CT angiographies are performed within 15-20 minutes and can be done under sedation as described earlier in the section of cardiac catheterization. The challenge is to provide anesthesia in the MRI suites. This is mainly due to:

• Duration of procedure: 45–60 minutes
• Requires intermittent periods of breath-holding (apnoea): 30 seconds
• Requires maintenance of heart rate between 100–115/minute
• Difficult access to the airway (head-end)
• Need for MRI compatible anesthesia machine, monitor and infusion pumps
• Remote location and low-temperatures in these suites
• Performed as a Day-care procedure done on OPD basis.

Apart from the MRI compatible anesthesia set-up required, one has to remember that the child undergoing cardiac MRI usually has a complex heart defect and the anesthetic agents or drugs chosen should maintain hemodynamic stability. Also, it is important to note that in an enthusiasm to reduce the heart rate, overdoses of anesthetic drugs like propofol, opioids etc. should not be used as this would delay the recovery and extubation at the end of the procedure. The authors therefore routinely avoid giving premedication with atropine or glycopyrrolate and use esmolol infusion titrated between 100–200 µg/kg/min for controlling heart rate. The aim is to use short-acting anesthetic agents and muscle-relaxants such as sevoflurane, propofol, atracurium which also avoid increases in heart rate and facilitate early extubation.

**FAST-TRACKING AND REGIONAL ANESTHESIA**

The benefits of early extubation are well-established. The era where children undergoing cardiac surgeries were ventilated for 48–72 hours is far gone. Most of the patients, with few exceptions, are extubated within 24 hours after surgery. This duration has further shortened to extubations in the operating room or within the first few hours, depending on the complexity of the lesion. Children who have undergone ASD closure, small-sized VSD closure, TOF repairs with mild degree of pulmonary stenosis and PDA ligations can be safely extubated in the operating room after giving reversal, good analgesia, confirming hemodynamic stability and hemostasis. The surgeon should be informed about the decision of extubation. A recent study performed in a large center has demonstrated that even neonates with complex congenital heart defects were extubated in the operating room with a success rate of 87.1%.61 The role of regional anesthesia to fast-track these patients and facilitate early extubation has been also highlighted in the same study.

Regional anesthesia provides good pain relief facilitating better hemodynamic stability in the perioperative period. Opioids can be given intrathecally, as a continuous epidural infusion or as a single shot (caudal) with or without a local anesthetic agent. The authors routinely use caudal morphine 100 µg/kg and clonidine 4 µg/kg diluted in a total volume of 1 mL/kg normal saline without local anesthetics, post-induction in all children, cyanotics and acyanotics, below the age of 8 years. The contraindications for these procedures include spinal deformity, neurodevelopmental abnormalities and altered preoperative coagulation profile (INR >1.5). Till date, there are no reports of epidural hematoma in pediatric cardiac patients who have received neuraxial intervention followed by systemic heparinization prior to CPB.

**LEARNING POINTS**

Anesthesia for congenital heart defects can be managed with ease if we understand the pathophysiology and keep things simple. Better perioperative outcomes can be achieved with:

• Knowledge about the unique differences in the cardiovascular system of children
• Understanding the anatomy and pathophysiology of the defect prior to and after corrective/palliative surgery
• Better preoperative evaluation specific to the defect and stabilisation if required
• Planning for vascular access and advanced monitoring
• Choice of drugs based on the desired hemodynamic goals
• Anticipation of the surgical steps for the given defect and alterations in the management accordingly
• Knowledge about the steps involved in the conduct of CPB
• Anticipation and management of complications specific to the defect
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INTRODUCTION

Congenital heart disease (CHD) is the commonest of all birth defects in children and is estimated to be 1 in every 125 live births. Among these, almost 30% require noncardiac surgery either immediately after birth or within one year. With advances in anesthesia and surgery, more than 90% grow into adults.

The anesthesiologist managing patients with CHD coming for extracardiac surgery (usually noncardiac anesthesiologist) should not only have knowledge of pediatric anesthesia but also of the pathophysiology of the cardiac lesions so that a rational management plan can be made. They have to consider the patients’ age, complexity of the heart lesion, along with patients’ capacity to compensate, urgency of surgery and multiple coexisting diseases. By and large, these children who present for noncardiac surgery do have an increased risk of morbidity, perioperative cardiac arrest and a higher 30 day mortality.

THE NEONATE

The cardiovascular system changes markedly at birth because of dramatic alteration in blood flow patterns which occur with the first breath. During fetal life, blood flow returning to the heart bypasses the unventilated, fluid-filled lungs. Blood is then preferentially shunted across the patent foramen ovale into the left atrium, or it passes from the right ventricle across the patent ductus arteriosus to the systemic circulation. At the time of birth, physiologic closure of the patent ductus arteriosus and the foramen ovale initiates normal adult circulatory patterns. All the blood returning to the right atrium is diverted to the lung, and all pulmonary venous blood returns to the left atrium and is pumped via the left ventricle to the body. Certain congenital heart defects can obstruct this normal adaptation process, creating a transitional circulation in which right-to-left shunting across the foramen ovale or the patent ductus arteriosus persists. The persistence of continued transitional circulation leads to severe hypoxemia, acidosis, and hemodynamic instability, which are poorly tolerated in the neonate. Nevertheless, in the initial treatment of some forms of cyanotic congenital heart disease, patency of the PDA is essential as it permits pulmonary blood flow and postnatal viability. An example is pulmonary atresia, in which pulmonary blood flow is temporarily supplied by the patent ductus arteriosus. Closure of the patent ductus arteriosus results in absent pulmonary blood flow, hypoxemia, and death. Patency can be maintained with the administration of prostaglandin E1.

Another difference in the cardiovascular system of infants is the decreased compliance of the neonatal myocardium which limits augmentation of stroke volume. Therefore, these patients are highly dependent on heart rate and adequate circulating blood volume to maintain cardiac output. Also, the response to inotropic drugs is limited and sensitivity to β-adrenergic blockade is increased. All these changes in the cardiovascular system of the young patient affect anesthetic management. The pulmonary vasculature also undergoes significant change during the first year of life. In the immediate newborn period, the large decrease in pulmonary vascular resistance
is the result of lung expansion and the vasodilatory effects of a higher partial arterial oxygen pressure (PaO₂) than in utero. Further decline in pulmonary vascular resistance and pulmonary pressures throughout the first month of life is attributable to regression of the smooth-muscle layer in the pulmonary arterioles. Any stress such as hypoxemia or acidosis or presence of VSD or ASD can delay regression of medial muscle hypertrophy. The presence of elevated pulmonary artery pressure in the neonate and infant adds complexity to the perioperative anesthetic management and significantly increases the risk of mortality. Immaturity of the liver and kidneys adds to the problem due to delayed drug metabolism, clearance and excretion.

**CLASSIFICATION**

There are several classifications of CHD but the most common and acceptable are:

1. **Based on Presence/Absence of Cyanosis: Acyanotic or Cyanotic**
   - **Acyanotic Heart Disease**
     - Ventricular Septal Defect (VSD).
     - Atrial Septal Defect (ASD).
     - Patent Ductus Arteriosus (PDA).
     - Atrioventricular Septal Defect (AVSD).
     - Pulmonary Stenosis (PS).
     - Aortic Stenosis (AS).
     - Coarctation of the Aorta.

   - **Cyanotic Heart Disease**
     - Tetralogy of Fallot.
     - Transposition of the Great Arteries (TGA).
     - Total Anomalous Pulmonary Venous Return (TAPVR).
     - Tricuspid Atresia.
     - Truncus Arteriosus.
     - Uncommon, each <1% of CHD, pulmonary atresia, Ebstein’s anomaly.

2. **Classification on the Basis of Pulmonary and Systemic Flow**
   - **Excessive Pulmonary Blood Flow**
     - VSD, ASD, PDA, PAPVR.
   - **Inadequate Pulmonary Blood Flow**
     - Tetralogy of Fallot, pulmonary atresia.
   - **Inadequate or Obstruction to Systemic Blood Flow**
     - Coarctation of Aorta.

   - **Abnormal mixing**
     - TGA.

**PREOPERATIVE CONSIDERATIONS**

Perioperative anesthetic considerations include preoperative evaluation, management of hypoxemia, shunt, polycythemia, pulmonary hypertension and ventricular dysfunction. Other than the risk associated with the heart disease itself and its associated complications, other risk factors include type of surgery, age, ASA physical status and length of previous hospital stay. Children with CHD presenting for noncardiac surgery can be grouped into three categories:

a. Nonoperated patient
b. Previous palliative surgery
c. Previous corrective surgery

To fully optimize the patient, information must be obtained about the cardiac lesion, its altered physiology and its implications under anesthesia. This includes knowledge about whether the patient is on parallel or single ventricle physiology and relies deeply on relative resistance between systemic and pulmonary circulation.

A thorough preoperative evaluation and preparation is necessary in these patients focusing on history and physical examination, available investigations such as echocardiogram and ECG, previous surgical and catheterization procedures, etc. Common medications in the CHD population include ACE inhibitors in patients with single ventricle, or those with significant CHF or mitral regurgitation; beta blockers are given to TOF patients or those with atrial arrhythmias such as paroxysmal supraventricular tachycardia; patients with significant atrial or ventricular arrhythmias are on amiodarone; and diuretics are given to patients with CHF. Digoxin is not as popular now due to the lack of effectiveness over a period and side-effects. Endothelin antagonists, phosphodiesterase-5 inhibitors, or prostaglandin analogs are used in pulmonary hypertensive patients. Many patients with CHD are on aspirin or other antiplatelet therapies to decrease risk of thrombus formation in shunts or conduits. Patients with mechanical valves are on coumadin, which will involve careful planning of perioperative anticoagulation regimens. Many patients, particularly older children with CHD will have implanted transvenous or epicardial pacemakers or automated
defibrillators, and it is critical to understand the patient’s underlying cardiac rhythm, the reason for placement of the device and the current modes and settings of the device.

Echocardiography is the mainstay of diagnostic testing in the CHD population and the latest echocardiography results should be reviewed. Presence of cardiac rhythm abnormality should be reviewed with a recent ECG or Holter examination. Cardiac catheterization has become uncommon as a diagnostic test, and is most often performed for interventional procedures. However, if it is performed on the child, very important anatomic and physiologic diagnostic data would be available. Cardiac MRI, being noninvasive, provides information on the anatomy, function, and progression of pathophysiology of the disease. If the child has a simple lesion such as ASD or PDA or a moderate complex lesion which is completely corrected (e.g. closure of VSD—surgical or nonsurgical), mandatory cardiologists consultation prior to the noncardiac surgery is not necessary as the child is well-compensated. All other patients, e.g. cyanotics or uncompensated noncyanotics should be reviewed by a cardiologist.

Preparation of the psychological aspect of patient and the family is essential for a successful outcome. Surgeons, cardiologists, anesthesiologists, intensivists, and nurses must work as a team in preparing the patient and the family for surgery and postoperative recovery. This team-oriented approach not only prepares the patient and family but also prevents errors and omissions in preoperative, intraoperative and postoperative care. The preoperative visit offers the family the opportunity to meet the surgeon and anesthesiologist as well as help in preparing the patient and the family for surgery.

**RISK STRATIFICATION**

Children may present with a range of heart disease for a variety of noncardiac procedures. This makes risk stratification difficult. Factors such as physiological status, disease complexity, type of surgery, and age help define children with higher risk. The most important factors are the physiological status and complexity of heart disease. Physiologically well-compensated patients with CHD can undergo elective operations at a low operative risk, whereas poorly compensated patients undergoing urgent or major operations are at high risk. In order to provide a practical and structured approach to management, Michelle C White and James M Peyton have classified children into high-, intermediate-, and low-risk groups (Table 1).

Individual risk factors are discussed below.

**Physiological Status**

Physiological status can be divided into four major risk factors: Cardiac failure, pulmonary hypertension (PHT), arrhythmias and cyanosis.

**Cardiac Failure**

Children with severe cardiac failure must be identified and optimized because they are at very high risk. Cardiac failure can be due to a volume-overloaded heart, pressure-overloaded heart or both. Volume overload may result from residual shunts or incompetent valves (e.g. after tetralogy of Fallot repair) and pressure overload (e.g. residual outflow tract obstruction). The incidence of perioperative inotropic support requirement is as high as 86% and cardiac arrest is 10%. Cardiac failure

<table>
<thead>
<tr>
<th>High risk</th>
<th>Intermediate risk</th>
<th>Low risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physiologically poorly compensated and/or presence of major complications</td>
<td>Physiologically normal or well-compensated</td>
<td>Physiologically normal or well-compensated</td>
</tr>
<tr>
<td>a. Cardiac failure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>b. Pulmonary hypertension</td>
<td></td>
<td></td>
</tr>
<tr>
<td>c. Arrhythmias</td>
<td></td>
<td></td>
</tr>
<tr>
<td>d. Cyanosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Complex lesions (single-ventricle or balanced circulation physiology) cardiomyopathy, aortic stenosis</td>
<td>Simple lesions</td>
<td>Simple lesions</td>
</tr>
<tr>
<td>Major surgery (intraperitoneal, intrathoracic) anticipated major blood loss requiring transfusion</td>
<td>Major surgery (intraperitoneal, intrathoracic) anticipated major blood loss requiring transfusion</td>
<td>Minor (or body surface) surgery</td>
</tr>
<tr>
<td>Under 2-year-old</td>
<td>Under 2-year-old</td>
<td>Over 2-year-old</td>
</tr>
<tr>
<td>Emergency surgery</td>
<td>Emergency surgery</td>
<td>Elective surgery</td>
</tr>
<tr>
<td>Preoperative hospital stay more than 10 days</td>
<td>Preoperative hospital stay more than 10 days</td>
<td>Preoperative hospital stay less than 10 days</td>
</tr>
<tr>
<td>ASA physical status IV or V</td>
<td>ASA physical status IV or V</td>
<td>ASA physical status I to III</td>
</tr>
</tbody>
</table>
in children manifests as poor feeding, failure to gain weight, sweating, tachypnea, tachycardia, hepatomegaly and cool peripheries. These children should be shifted to a tertiary center for any noncardiac surgery, however minor it may be. If the child presents with an emergency condition needing immediate surgery, help should be sought from an experienced pediatric anesthesiologist or at least a second experienced anesthetist and cardiologist. Procuring a vein may be difficult in a puffed up child. Anesthesia induction can be either inhalational or IV route but induction times will be prolonged. Hence, excessive administration of drugs should be avoided as prolonged use of 8% sevoflurane or excessive propofol may cause a profound decrease in cardiac output. Ketamine is the i.v. agent of choice\textsuperscript{1} as it neither causes myocardial depression nor alters the systemic vascular resistance.

**Pulmonary Hypertension**

Pulmonary hypertension (PHT) is defined as having a PA pressure (PAP) \( >25 \) mm Hg at rest or \( >30 \) mm Hg during exercise. Documented PHT is a clear predictor of perioperative morbidity.\textsuperscript{11} Children with PHT have increased airway resistance and poor pulmonary compliance causing increased work of breathing. Therefore, presence of respiratory tract infections will be poorly tolerated and have a greater impact on PVR. They are eight times more likely to experience a major complication. Hence, they should be transferred to a specialist center equipped with pediatric intensive care. PHT is treated with 100% oxygen, inhaled nitric oxide, IV prostacyclin, inotropic support of the right ventricle and other measures to maintain cardiac output and PBF.

**Arrhythmias**

A preoperative ECG is mandatory in all children with CHD. Presence of right bundle branch block (RBBB) is not serious as it usually does not progress to complete heart block. However, ventricular ectopics are dangerous as \( \sim30\% \) of children with ventricular ectopics die suddenly.\textsuperscript{12} Furthermore, arrhythmias leading to death arise in \( \sim30\% \) of patients with a single-ventricle circulation. Therefore, children with ventricular arrhythmias on ECG or a single-ventricle circulation should be referred for surgery in a specialized center having pediatric cardiology and pediatric intensive care facilities.

Children who have undergone ventriculotomy or had a right ventricle to PA conduit performed are more at risk of ventricular arrhythmias than other groups of children. If these children have a normal preoperative ECG, they would be classed as low risk (Table 1) and can be anesthetized in the local hospital.

**Cyanosis**

Most of the cyanotic children often have concurrent cardiac failure, PHT and arrhythmias, making them a very high-risk group and merit being cared in a specialist center. Anesthesiologists who understand the physiology and perioperative problems related to cyanosis can proceed with anesthetizing the child if there are no additional complications and is presenting for a minor procedure. The problems related to cyanosis are polycythemia and coagulopathy. This can affect both anesthesia and surgery. Children are at risk of hyperviscosity causing cerebral vein and sinus thrombosis. Dehydration, fever and iron deficiency anemia increase the risk. Preoperative IV fluid therapy can minimize risk. FFP should be kept ready in presence of deranged coagulation profile. Aspirin should be continued if the child is receiving it, as the risk of thrombosis is usually greater than that of bleeding.

**Complexity of Heart Disease**

Children with complex heart disease are more prone to perioperative morbidity and mortality. Complexity\textsuperscript{5} can be defined as:

i. Parallel circulation physiology.
ii. Single-ventricle physiology.
iii. Cardiomyopathy.
iv. Aortic stenosis.
v. Presence of long-term sequelae (cardiac failure, PHT, arrhythmias and cyanosis).

**Parallel or ‘Balanced’ Circulation**

The pulmonary and systemic circuits instead of being separate, communicate with each other and are parallel. The flow in either of the circuits is delicately balanced and depends on the relative resistance of each, i.e. SVR to PVR ratio (hence, the name “balanced”). Excessive PBF causes pulmonary edema and poor systemic perfusion (which may compromise coronary and splanchnic perfusion); insufficient PBF causes profound cyanosis. Examples of children with this type of circulation are those with unrepaired VSD, palliative surgery (Modified BT shunt) in a case of TOF, truncus arteriosus and hypoplastic Left heart syndrome. These patients are very difficult to manage as high concentrations of oxygen will increase PBF and reduce systemic perfusion; conversely, large doses of induction agent may reduce SVR so much that shunt flow is reversed causing desaturation. The excessive PBF also makes the child vulnerable to developing pulmonary hypertension (PHT).
**Single-ventricle Circulation**

A child with a single ventricle is not amenable to full anatomical correction. Therefore, these children will be palliated by creating a circulation based upon a single ventricle. The single-ventricle pumps oxygenated blood around the body, while blood flows passively to the lungs down a pressure gradient from the pulmonary artery (PA) to the left atrium (LA). A BT shunt or Glenn shunt is placed first to improve pulmonary blood flow. At a later stage, the Fontan procedure (inferior vena cava is connected to the RPA) is performed for normalizing oxygen saturation after disconnecting the earlier shunt. The pressure gradient from the PA to the LA is now the sole determinant of PBF.

In the single-ventricle circulation, increases in PVR and intrathoracic pressure can compromise PBF. Spontaneous breathing causes negative intrathoracic pressures and augments PBF; however, positive pressure ventilation allows better control of oxygenation and minute ventilation, and avoids hypoxia and hypercapnia. Positive end-expiratory pressure should be optimized; peak inspiratory pressures and inspiratory times minimized to facilitate PBF.

**Cardiomyopathy**

Pediatric cardiomyopathy is rare but could be undiagnosed leading to sudden cardiac arrest under anesthesia. About 68% is dilatation variety (DCM) and 25% is of hypertrophic type (HCM). A thorough assessment of cardiac function is needed before taking up the noncardiac case. Anesthetic management and response to anesthetic drugs are different in each type. Broadly speaking, the management goals are to optimize myocardial function by maintaining the patient’s baseline hemodynamic variables of preload, heart rate, contractility, and afterload. Anesthetic principles for DCM include maintenance of normal diastolic arterial pressure to optimize coronary perfusion, maintenance of preload, avoidance of tachycardia, avoidance of decreased myocardial contractility, and ensuring that the systemic vascular resistance (SVR) is not elevated. A poorly contracting left ventricle will not maintain CO in the presence of a high SVR. If inotropic support is required, dilating agents such as milrinone, dobutamine, or low-dose epinephrine may be titrated to clinical effect. The anesthetic management of patients with HCM aims to minimize any increase in systolic left ventricular outflow tract obstruction (LVOTO). This is achieved by maintaining a normal to slightly elevated SVR, preventing hypovolemia, and avoiding a state of increased myocardial contractility (either through endogenous or exogenous catecholamine stimulation). Heart rate is usually kept at a normal to low rate, aimed at optimization of diastolic filling time and enhanced stroke volume. Sinus rhythm is essential. As a disease of diastolic dysfunction, these patients rely on atrial contraction to fill a non-compliant ventricle. Atrial fibrillation or supraventricular arrhythmias are, therefore, poorly tolerated.

**Aortic Stenosis**

Majority of the children have bicuspid valves. A preoperative 2D Echo should be done to assess the severity of stenosis and left ventricular function as intraoperative sudden cardiac arrests are common. Perioperative invasive monitoring is advised for patients with an aortic valve area <1.0 cm² or a mean aortic valve gradient >30 mm Hg. Hypotension and arrhythmias should be avoided at all cost. Hypotension leads to myocardial ischemia and a downward spiral of reduced contractility causing further falls in blood pressure and coronary perfusion. Anything that reduces systemic vascular resistance (e.g. regional neuraxial techniques) must be used with extreme caution. Limb blocks can be useful, either alone or combined with general anesthesia, as they do not alter the sympathetic tone. Drugs to maintain the systemic vascular tone such as norepinephrine, phenylephrine or metaraminol must be at hand. Hypotension should be treated aggressively with these drugs initially, followed by management of the underlying cause, e.g. hemorrhage. Maintenance of sinus rhythm and adequate intravascular volume is vital to ensure ventricular filling. Arrhythmias must be treated promptly. New onset atrial fibrillation may require cardioversion. Sinus tachycardia can also be detrimental as it reduces the diastolic time for myocardial perfusion.

**Type of Surgery and Age**

The mortality of children with heart disease undergoing major surgery is 16% compared with 3% for minor surgery. Major surgery is classified as intraperitoneal, intrathoracic, or vascular reconstructive surgery. Furthermore, the most common cause of cardiac arrest in noncardiac surgery is hypovolemia including the consequences of massive blood transfusion. However, given that major surgery increases perioperative risk from 3% to 16%, children with physiologically well-compensated disease or simple disease undergoing major surgery present intermediate risk. The presence of additional risk factors such as age under 2 years or prolonged hospital stay may be additive risk factors.

Children should be fed light meal 6 hours prior to major surgery. Breast fed children should get the last feed 3 hours prior to surgery. Children who are otherwise normal and well-compensated can have clear fluids 2 hours prior to surgery. Fasting instructions should be clearly written.
with timing. Dehydration is avoided in cyanotic patients. If timing of surgery is uncertain, IV fluids should be started.

Sympathetic stimulation due to crying can increase oxygen consumption and myocardial work; this might be poorly tolerated in a child with limited cardiac reserve. Intranasal 0.2 mg/kg or oral midazolam 0.5 mg/kg is the preferred premedication half an hour before surgery to reduce oxygen consumption. If IV line is present, then incremental doses of 0.1–0.25 mg/kg midazolam can be given, provided airway and breathing issues were addressed accordingly. 18

In the recent guidelines from American College of Cardiology/American Heart Association (ACC/AHA), underlying cardiac rhythm modes and settings of patients with implanted pacemakers and/or automated defibrillator must be investigated prior to and after operation. Intraoperative troubleshooting of the device must be managed by a trained personnel. 16

Endocarditis prophylaxis has recently been revised for dental procedures. AHA recommends antibiotic prophylaxis for following patients:
- When gingival tissue is manipulated, or periapical region of teeth or perforation of oral mucosa
- Prior history of infective endocarditis
- Nonrepaired cyanotic CHD, including shunts and conduit
- Complete CHD repair within the previous 6 months
- Repaired CHD with residual defects.

Antibiotics for infective endocarditis prophylaxis is no longer indicated in patients with:
- Aortic stenosis, mitral stenosis, or symptomatic or asymptomatic mitral valve prolapse
- Genitourinary and gastrointestinal tract procedures (transesophageal echocardiography, esophagogastroduodenoscopy, colonoscopy, etc.) also do not warrant infective endocarditis prophylaxis unless active infection is present. 17 Box 1 lists the key features of preoperative assessment in children with CHD.

Monitoring

Apart from using standard monitors usually used in any pediatric surgical case, one should step up monitoring depending upon the merit of the case. ECG, Spo₂, ETCO₂, respiration, temperature, anesthetic gas monitoring, NIBP or invasive BP as per case, CVP, urine output, ABG, serum electrolytes and blood sugar are monitored.

Anesthetic Technique

No specific technique is superior over the other. 18 Left-to-right shunts are the most common lesions representing over 50% of children with CHD. Examples include atrial septal defects, ventricular septal defects, patent ductus arteriosus, AV canal defects, PAVD and Blalock Taussig shunt (BT Shunt). Left-to-right shunts lead to excess pulmonary blood flow. Patients are acyanotic and regular inhalational or intravenous induction is usually safe in these patients but deterioration in gas exchange may result from pulmonary congestion. 100% oxygen and hyperventilation in patients with L-R shunt will result in pulmonary vasodilation, which in turn will further increase pulmonary congestion, and thus should be avoided. Propofol, thiopentone and ketamine are the agents used commonly in children with CHD undergoing noncardiac surgery. Propofol dramatically reduces SVR and mean arterial pressure (MAP). In children with right-to-left shunt lesions, propofol may cause a clinically significant reduction in oxygen saturation by increasing the shunt flow. 19,20 Ketamine has minimal effect on SVR, MAP, PVR and PAP. Hence, this agent is preferred when a reduction in SVR is undesirable as in TOF or in children with severe PHT (Eisenmenger complex). A TET SPELL should be promptly treated with 100% oxygen, IV fluids, Alpha agonist like phenylephrine 0.01 mg/kg and beta blocker (Injection metoprolol 0.1 mg/kg) to release infundibular spasm.

Children may present with pulmonary hypertension and it should not get aggravated by the stress of anesthesia, such as pain, hypothermia, acidosis, hypercarbia, hypoxia or elevated thoracic pressures. 21 Measures to treat the

**Box 1: Key features in preoperative assessment**

- Knowledge of the underlying lesion and type of circulation. Are changes in SVR/PVR likely to be of significant importance? Are the oxygen saturations what is to be expected for the type of lesion?
- Evidence of long-term complications and other features that put children into a high-risk category.
- Evidence of recent upper or lower respiratory tract infections: This may cause changes in airway reactivity and PVR which may be poorly tolerated in children with reduced pulmonary compliance or PHT and particularly those with a Glenn or Fontan circulation.
- Venous access: May be problematic and alter the choice of anesthetic technique. Many children have had multiple peripheral and central venous lines in the past.
- Routine drug therapy: Most cardiac medications should be continued before operation. Some anesthetists prefer to omit ACE inhibitors based on adult literature. Cardiac medications are not associated with electrolyte disturbances so routine preoperative blood testing is unnecessary. Aspirin should be continued to prevent shunt thrombosis, and children on warfarin need admission for monitoring and establishment on IV heparin.
- Sedative premedication: Commonly used to avoid stress, minimize oxygen consumption, and may also reduce the amount of induction agent so minimizing reductions in SVR.
- Endocarditis prophylaxis: National guidelines must be followed.
cause should be immediately instituted and pulmonary hypertension should be treated as mentioned earlier in the chapter.

Inhalation induction with sevoflurane or high dose opioid induction can be used for children with CHD. In high-risk children, prolonged inspired concentrations of 8% are not advocated and slow induction with lower concentrations will often work. Both isoflurane and sevoflurane have minimal effect on myocardial contractility or shunt fraction and can be used for maintenance. The effects of desflurane in children with CHD are less known. Airway management may range from sedation with spontaneous respiration, to mask or LMA general anesthesia, to endotracheal anesthesia. Injection rocuronium, vecuronium or atracurium can be used safely for providing muscle relaxation. One should be mindful of the effects of hyper- or hypocarbia, and postive pressure ventilation for the individual patient.

Laparoscopic procedures can cause further insults due to pneumoperitoneum, hypercarbia and extreme positions. Children with compensated physiology tolerate these procedures quite well. However, the intra-abdominal pressures should be maintained below 12 cm H2O.

Dexmedetomidine is increasingly used for sedation in patients with CHD and is usually well-tolerated if the patient can withstand bradycardia and hypotension that sometimes results from the use of this agent. Single shot caudal and nerve block techniques can also be used, even with low dose aspirin use. Major neuraxial techniques, i.e. lumbar/thoracic epidural and spinal are best avoided with aspirin use.

The presence of scars due to previous surgery, atrial or ventricular overdistension can trigger arrhythmia. Only lethal rhythms should be treated with antiarrhythmics.

Use of spinal anesthesia is not completely contra-indicated and has been used successfully in patients with ASD, VSD or PDA with no pulmonary hypertension. Opioid infusions and epidural anesthesia have also all been used successfully. Peripheral blocks are safer and used whenever possible for intraoperative as well as postoperative pain relief.

Pain management is crucial and should not be neglected in these patients. Opioid infusion or nurse-controlled analgesia for major operations has been the primary postoperative intervention for pain in patients with CHD. Regional blocks or caudal epidural are safe and effective methods for pain relief. NSAID can be given orally or as rectal suppositories for minor/day care procedures. The children are observed in high-dependency bed or intensive care unit to treat complications which may arise such as arrhythmias, cardiac ischemia, dehydration, pain, ventilation issues, etc.

CONCLUSION

With technological and medical advances, more and more children survive and may require anesthesia for noncardiac surgeries. Multidisciplinary approach involving the surgeon, cardiologist, intensivist and the nurse is required in intermediate and complex cases. The caring anesthesiologist should have a complete knowledge of the pathophysiology of the lesion and should be able to assess the patient thoroughly and anesthetize the child as per requirements for a safe outcome.

LEARNING POINTS

- The anesthesiologist managing patients with CHD coming for extracardiac surgery should not only have knowledge of pediatric anesthesia but also of the pathophysiology of the cardiac lesions as the presentation may be diverse depending on the congenital cardiac lesion
- A complete history and physical examination is essential taking into consideration the physiological factors such cardiac failure, PHT, arrhythmias and cyanosis so that appropriate treatment can be given and the child is optimized in the preoperative period
- The risk associated with the non cardiac surgery varies depending upon the type of surgery and the congenital cardiac lesion. For example, dental extraction in a child with tetralogy of Fallot is less risky than laparoscopic procedures in a VSD patient
- Anesthetic technique should be modified depending upon the merit of the lesion. No single anesthesia technique is superior over the other
- Both inhalational and opioid induction are safe in children. Injection propofol can be used only in a well-compensated patient as it causes a significant fall in cardiac output
- Pain management is very essential in these patients as the sympathetic response associated with pain may decompensate the child in the postoperative period

REFERENCES


SECTION 4

Special Problems and Situations

Chapter 32: Common Pediatric Medical Conditions: Anesthetic Considerations
Chapter 33: Anesthesia Management in Special Conditions
Chapter 34: Cardiopulmonary Resuscitation in Neonates and Children
Chapter 35: Acute Complications During Anesthesia
Common Pediatric Medical Conditions: Anesthetic Considerations

Chapter 32

INTRODUCTION

"Upper respiratory tract infection (URI)" broadly encompasses rhinosinusitis (common cold), pharyngitis/tonsillitis, sinusitis, ear infections and laryngitis. Young children commonly have around 6-8 episodes of URI in a year, most of which are common cold and viral in origin. The common cold is generally heralded by sore throat, followed by nasal obstruction, rhinorrhea, and occasionally cough. Examination findings are increased nasal secretions and swollen or erythematous nasal turbinates. It usually lasts for about 1 week, occasionally extending to a fortnight in 10%. Certain conditions like allergic rhinitis, foreign body, sinusitis, pertussis, etc. may mimic a common cold, with disastrous consequences if missed; hence, they should be differentiated in any child presenting as an URI.2

PATHOGENESIS3

The airway hyper-reactivity (Flowchart 1) could persist for up to 6 weeks after the URI thereby affecting management decisions of children requiring anesthesia in the acute and convalescent phase. Often, children who present for an elective surgery with an URI have their procedure rescheduled until they are asymptomatic.4 Though the risk of serious events is low, numerous studies and anecdotal reports have demonstrated around a two to ten fold increase in risk of bronchospasm, laryngospasm and hypoxemia, which further increase to eleven fold if tracheal intubation is done.1

PREOPERATIVE OPTIMIZATION

Studies have attempted to preoperatively score children with URI to identify variables and predict the probability of an adverse anesthetic event.5 Identifying such factors preoperatively may guide in making decisions regarding anesthesia in such children, some of which are summarized in Table 1.1

Often the parental assessment of severity of illness in their child correlates well with the risk of perioperative complications. Besides, it is prudent to involve a senior anesthetist in managing such children.

Flowchart 1: Pathogenesis of upper respiratory tract infection

Chhaya A Divecha, Chandrika Bhat, Milind S Tullu
Consolidating the above factors, the following steps outline the preoperative assessment and management of a child with URI:

• When a child presents for an emergency procedure, the anesthetist should be prepared for potential complications during induction and emergence, before proceeding with the surgery.

• However, when a child with URI of a suspected infectious etiology has to undergo anesthesia for an elective procedure, a detailed history, physical examination and necessary investigations (such as complete blood count, chest X-ray) can be done to exclude a lower respiratory tract infection.

• If the child has a lower respiratory tract infection or severe symptoms (fever >38°C, mucopurulent secretions, productive cough or signs of respiratory involvement), antimicrobial agents are started and surgery is postponed for at least 4 weeks.

• Also, children should be screened for the risk factors mentioned earlier (Table 1) before proceeding with the surgery.

INTRAOPERATIVE MANAGEMENT

Management of a child with URI should be targeted at reducing secretions and preventing stimulation of a potentially sensitive airway.

Reducing Secretions

• Adequate intravenous hydration, especially if procedure is prolonged.

• Adequate suction of the airways under deep anesthesia (reduces airway stimulation and mucus plugging).

• Humidification of inhaled air.

• Use of anticholinergics like atropine or glycopyrrolate (attenuates vagal-stimulated hyper-reactiveness).

Prevention of Airway Stimulation

• Minimize intubation wherever feasible and opt for face mask or LMA as a safer alternative.

• If intubation is obligatory, it should be performed in a deep plane of anesthesia and with an ETT that is one size smaller than the age determined value.

• Bronchodilator premedication may minimize autonomic triggered airway complications.

• Adjunctive agents like intravenous lidocaine, an opioid, or both may be used to decrease airway reflexes.

• Appropriate choice of anesthetic for induction and maintenance (sevoflurane > halothane) based on rapid recovery profile and fewer complications.

The policy of blanket cancellation of surgery in a child with URI should undergo reconsideration. It is known that children with active/recent URI are prone to perioperative complications; however, these can be anticipated, identified, and treated in most cases. Issues, such as the need to expedite surgery and the emotional and financial burden on parents also need to be addressed. Anesthesia in a child with URI can be optimized by deciding on a case-to-case basis, after reviewing risk/benefit profiles and the anesthesiologist’s comfort and expertise in dealing with such a child with URI.

### Table 1: Factors which increase the risk of adverse anesthetic event in a child with URI

<table>
<thead>
<tr>
<th>Factors related to the URI</th>
<th>Factors pertaining to the child</th>
<th>Surgery and anesthesia related factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fever (&gt;38°C)</td>
<td>Age under 1 year</td>
<td>Airway instrumentation (ETT &gt; LMA &gt; face mask)</td>
</tr>
<tr>
<td>Purulent and copious secretions</td>
<td>History of prematurity</td>
<td>Airway surgery</td>
</tr>
<tr>
<td>Productive cough</td>
<td>History of sickle cell disease</td>
<td>Inhalational induction</td>
</tr>
<tr>
<td>Malaise, lethargy, decreased appetite</td>
<td>History of snoring</td>
<td>Major surgery</td>
</tr>
<tr>
<td>Lower respiratory tract signs</td>
<td>History of reactive airway disease (asthma)</td>
<td>Anesthetist with limited pediatric anesthesia experience</td>
</tr>
<tr>
<td></td>
<td>Parental smoking</td>
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</table>

### REACTIVE AIRWAY DISEASE/ASTHMA

INTRODUCTION

Asthma is a chronic inflammatory condition of the airways and is a leading cause of morbidity in children worldwide. It is characterized by reversible hyper-responsiveness, inflammation and obstruction of airways; manifested by symptoms ranging from wheeze, shortness of breath, chest tightness, and cough. Upper respiratory tract infection (URI), aeroallergens, emotion, exercise and cold air are known to precipitate an exacerbation. Classification of the severity of asthma is usually based on level of treatment needed for control of symptoms. Global
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Initiative for Asthma (GINA) updated 2015 guidelines can be referred for further details regarding stepwise control of asthma. Due to risk of perioperative respiratory adverse events, preoptimization of treatment and appropriate perioperative measures are essential to reduce such risks.1

PREOPERATIVE EVALUATION

Optimization of management of a child with reactive airway disease should ideally begin 1–2 weeks prior to elective surgery. This encompasses a prudent history, physical examination and few laboratory tests (Table 2). Any patient with uncontrolled symptoms should be referred to the primary physician/asthma specialist for modification of treatment for better asthma control. Medical management generally involves intermittent use of short acting β2 agonists, and may be escalated as necessary to add inhaled corticosteroids in increasing doses, long acting β2 agonists, leukotriene receptor antagonists, methylxanthines and oral glucocorticoids.5

PERIOPERATIVE MANAGEMENT1,3,5

Presurgery optimization enables the anesthetist to be well versed with the profile of the child with asthma and parental concerns, making him prepared to deal with intraoperative issues. The following plan may be followed with some individual variations on a patient-to-patient basis:

1. Preoperative Medication Management: All asthma medications should be continued up to, as well as on day of surgery (except theophylline due to risk of cardiac arrhythmias). In children on long-term steroids (more than 5 mg/day of prednisolone for more than 3 weeks in preceding year) or severely, poorly controlled asthma, preoperative systemic steroids can be given. Prednisolone 1 mg/kg orally for 3–10 days or intravenous hydrocortisone (4 mg/kg 6 hourly) or methylprednisolone (1–2 mg/kg once daily) can be administered.5

2. Premedication: Children with asthma should be administered inhaled short acting β2 agonists (salbutamol at 2.5 mg if <20 kg weight and 5 mg if >20 Kg weight via nebulizer OR two to eight puffs via metered dose inhaler (MDI) with spacer) 20–30 minutes prior to induction of anesthesia. This attenuates increased airway resistance associated with tracheal intubation. Sedation may be used to prevent anxiety-related hyperventilation and subsequent bronchospasm. Midazolam is generally preferred either transmucosally or intravenously (0.1 mg/kg/dose).3,5

3. Decision regarding choice of anesthesia (Regional versus General): As endotracheal intubation can be a difficult procedure in children with reactive airway disease, regional anesthesia is the preferred method. A regional block can be performed before induction of general anesthesia to provide additional sedation and analgesia. In cases where general anesthesia is necessary, it is important to preoptimize the child with asthma and ensure that all necessary medications are given beforehand. The anesthetist should be prepared to deal with any respiratory complications that may arise during the procedure. 

Table 2: Pre-operative evaluation in a child with asthma1,3

<table>
<thead>
<tr>
<th>History</th>
<th>Specific history</th>
<th>Intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>Focus on key points as:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Triggering factors</td>
<td>If the child has experienced an acute exacerbation or used rescue medication in the preceding 4–6 weeks</td>
<td>Surgery is rescheduled to a date 4–6 weeks later to alleviate the effects of residual hyper-reactiveness and impaired gas exchange</td>
</tr>
<tr>
<td>• Frequency and severity of symptoms</td>
<td>Recent upper respiratory tract infection (URI) in a child with asthma may precipitate an acute episode</td>
<td>Consider postponing the surgery for 4–6 weeks</td>
</tr>
<tr>
<td>• Time since the previous attack</td>
<td>Lower respiratory tract infection in previous 6 weeks</td>
<td>Essential to reschedule surgery due to exaggerated airway reactivity</td>
</tr>
<tr>
<td>• Activity level</td>
<td>Children with a history of poorly controlled symptoms even with maximal therapy and frequent oral steroid courses</td>
<td>Short preoperative course of oral steroids especially if intubation planned. (Prednisolone 1 mg/kg/day for 3 days inclusive of the day of surgery) However, consider postponement of elective surgery till treatment is optimized</td>
</tr>
<tr>
<td>• Current medications (maintenance and rescue; including recent oral steroids)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• History of admissions to hospital and intensive care/emergency room visits</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• History of recent URI</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Any previous reactions to anesthesia or non-steroidal inflammatory drugs (NSAIDs)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Physical examination

Comprehensive physical examination to detect tachypnea, wheezing, crepitations, accessory muscle use, prolonged expiration and decreased air entry (severe asthma)

As asthma patients (up to 15%) may have nasal polyps as comorbidity, it should be ruled out if nasal intubation is considered

Baseline oxygen saturation on room air using a pulse oximeter should be documented to identify pre-existing hypoxemia

Laboratory tests

In children above 6 years of age, lung function tests are performed to assess the degree of airway obstruction

If younger or uncooperative, peak expiratory flow rates can be measured and compared to the baseline (or to that estimated by height)

Reversibility with bronchodilators can also be assessed
potentially trigger bronchoconstriction, it is preferable to use noninvasive airway devices (LMA) whenever feasible. Bronchoconstriction during general anesthesia can occur due to mechanical stimulation of airway by endotracheal tube, histamine release by administered medications and vagal stimulation during surgery. Supplementation of inhalation anesthesia (sevoflurane or isoflurane) with regional anesthesia should be considered wherever feasible.5,6

4. Strategy for General anesthesia:
   a. Airway: Noninvasive devices are preferred to endotracheal intubation whenever possible. Also a cuffed endotracheal tube is preferable as it allows better mechanical ventilation.2 Heated humidification is recommended especially in infants and young children as the anesthetic dry gas mixture has propensity to precipitate intraoperative bronchospasm.3
   b. Induction of anesthesia: Adequate depth of anesthesia should be ensured to blunt physiological response to airway manipulation. Also intravenous induction agents and inhalational anesthetic agents which blunt airway reflexes (during induction and airway manipulation) and facilitate bronchodilatation (while maintaining anesthesia) should be used. Intravenous drugs which can be used are propofol, ketamine and etomidate.5
   c. Maintenance of anesthesia: Inhalational agents, such as sevoflurane, isoflurane, halothane or nitrous oxide can be used, while desflurane should be avoided. Intravenous agents, such as propofol, ketamine, clonidine and dexmedetomidine can be used. Neuromuscular agents such as rocuronium, vecuronium and cisatracurium are safe to use while histamine- releasing drugs like atracurium, mivacurium, thiopentone, morphine and suxamethonium should be avoided. Synthetic opioids like fentanyl, sufentanil, remifentanil and hydromorphone can also be safely used.
   d. Strategy for ventilation: Asthma patients require controlled ventilation to minimize air trapping. Though a well controlled child requires routine ventilation, modification of ventilation strategy may be required during bronchospasm. Tidal volumes should be kept between 6 and 8 mL/kg while respiratory rates should be near or even less than physiological rates. Inspiration to expiratory ratio should be prolonged (up to 1:4 to prevent breath stacking, Positive End Expiratory Pressure (PEEP) should be low (3–5 cm H₂O) and Plateau pressures should be <35 cm H₂O.

MANAGEMENT OF INTRAOPERATIVE WHEEZING/BRONCHOSPASM

Despite the above preoperative and intraoperative measures, wheezing may still occur. Wheezing that develops in the intraoperative period could be due to numerous reasons, such as inadequate depth of anesthesia, endotracheal tube obstruction, foreign body in the airway, airway secretions, pulmonary edema, embolus, aspiration or exacerbation in a known asthmatic child. The following steps should be followed when such a situation is encountered as shown in Box 1.

EXTUBATION

Exubation should be performed aiming to achieve a smooth and controlled emergence. This can be done in the awake or deep state after weighing the pros and cons. (Table 3).

**Box 1: Intraoperative management of bronchospasm**1,2,5

- Auscultate the chest again to exclude mainstem bronchus intubation. Also rule out pneumothorax, mucous plug, anaphylaxis and congestive cardiac failure.
- Step up the concentration of inhaled oxygen to 100% and initiate careful hand ventilation with slow respiratory rate and adequate expiratory time to prevent barotrauma.
- Increase the depth of anesthesia using volatile anesthetics and intravenous propofol or ketamine (a known bronchodilator at 0.5 to 2 mg/kg intravenously).
- Airway reflexes/reactivity may be blunted with the use of intravenous lidocaine (1 mg/kg).
- In an intubated patient, the ETT should be suctioned to clear the secretions and/or muscle relaxant may be administered.
- Bronchodilators like β2 agonists, e.g. salbutamol (<5 years—2.5 mg >5 years—5 mg) or ipratropium bromide (250 µg) should be administered through a nebulizer or a metered dose inhaler and adapter to the ETT followed by bagging with vital capacity precision to ensure widespread dissemination of the aerosol, this can be repeated two to three times.
- In cases of severe bronchospasm, supplemental oxygen (100%) along with two to three puffs of salbutamol should be administered 20 minutes apart. If bronchospasm does not abate, concentration of volatile anesthetic agents (excellent bronchodilators) can be increased. Systemic corticosteroids like hydrocortisone (8 mg/kg loading dose followed by 2 mg/kg/dose 6 hours apart) or methylprednisolone (1–2 mg/kg), magnesium sulfate (iv, 40–50 mg/kg) or aminophylline (5 mg/kg loading dose followed by 1 mg/kg/hour infusion) can be tried.
- Anticholinergics such as Glycopyrrolate (4 mcg/kg iv), atropine (0.02 to 0.03 mg/kg iv) and ipratropium (250 to 500 mcg by nebulizer or four to eight puffs [18 mcg/puff] via metered dose inhaler- MDI) can be used due to their bronchodilator properties.
- Epinephrine 0.01 mL/kg/dose of 1:1000 dilution can be administered intramuscularly/intravenously for refractory bronchospasm and suspected anaphylaxis.
- Ventilation should be modified to prevent breath stacking, air trapping and barotrauma.

**Abbreviation:** IV, Intravenous
In case of an uneventful intraoperative course and well-managed pain and pulmonary status, the postoperative management of an asthmatic child is the same as a nonasthmatic child. The general care is summarized below:

- Continue maintenance oral/inhaled asthma medications. Replace oral with intravenous steroids and metered dose inhalers (MDI) with nebulized medications, if required
- Adequate pain control, bronchodilation, use of incentive spirometry in co-operative children, deep breathing maneuvers and early mobilization should be done to minimize postoperative pulmonary complications
- Postoperative ventilation and delay in extubation can be done in the event of severe bronchospasm during the surgery
- If wheeze persists postoperatively, trial of high-flow humidified oxygen or noninvasive ventilation (NIV) may be considered.

**Table 3**: Deep state extubation versus awake extubation

<table>
<thead>
<tr>
<th>“Deep” state extubation</th>
<th>“Awake” extubation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Risk of soft tissue collapse in the pharynx causing upper airway obstruction</td>
<td>Risk of invoking airway reflexes resulting in bronchospasm, increased incidence of coughing and oxygen desaturation</td>
</tr>
<tr>
<td>Successful deep extubation is more likely if the upper airway is patent and clear of secretions prior to intubation while inducing anaesthesia</td>
<td>This can be overcome by prophylactic inhaled β2 agonist (over and above their use prior to or during anesthesia)</td>
</tr>
<tr>
<td>If not the case, it may be enabled by placing a well lubricated nasopharyngeal or oropharyngeal airway prophylactically while patient is still in deep anaesthesia</td>
<td>Adequate tracheal suctioning prior to emergence (prevent mucus plug migration)</td>
</tr>
<tr>
<td>Intravenous Lidocaine (1 mg/kg) at emergence to reduce tracheal stimulation on patient awakening</td>
<td>Intravenous atropine (0.02 mg/kg) prior to extubation as a vagolytic and bronchodilator</td>
</tr>
</tbody>
</table>

**Emergency Surgery**

Unless the situation is life-threatening, there is usually enough time to do basic evaluation (identifying triggers of asthma, current medical management, level of control and previous history of bronchospasm during anesthesia), administer inhaled bronchodilators, and if needed, perioperative intravenous steroids. While managing trauma with hemodynamic instability, achieving deep level of anesthesia may be difficult. A modified rapid sequence induction may be used using cricoid pressure and gentle bag and mask ventilation. Combination of ketamine and propofol may be useful due to their bronchodilator properties. In children with chest and airway trauma, conditions like tension pneumothorax, pulmonary hemorrhage, and partial tube obstruction by clots may mimic bronchospasm and need to be ruled out.

**Asthmatics with Increased Intracranial Pressure**

The management of such children may pose a challenge. Hyperventilation to decrease the intracranial pressure may lead to air trapping, whereas use of higher concentration of volatile anesthetics in an asthmatic without hyperventilation may increase the intracranial pressure. In such children propofol, or etomidate may be used for induction, after taking adequate measures to maintain blood pressure.

**NUTRITIONAL ANEMIA**

**INTRODUCTION**

Anemia is defined as a reduction in red blood cell mass or blood hemoglobin (Hb) concentration to less than lower limit of reference range for age. Iron deficiency anemia (IDA) is the most common nutritional disorder worldwide. Data from studies conducted by the National Family Health Survey (NFHS)-3 shows that 7 out of 10 children in the age group of 6–59 months in India are anemic.

**ETIOLOGY**

The etiology of iron deficiency anemia revolves between iron intake, physiologic requirements and potential blood loss.

- Inadequate iron intake due to low overall dietary intake compounded by poor intake of iron rich food. The bioavailability of iron in breast milk is more than that in cow’s milk. Hence, a diet consisting of cow’s milk predisposes a child to IDA
### Box 2: Three tiers of patient blood management (PBM)

<table>
<thead>
<tr>
<th>Tier</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Optimization of the (preoperative) erythrocyte volume</td>
</tr>
<tr>
<td></td>
<td>- Identifying anemia in preoperative outpatient clinic at least 3–4 weeks prior to a planned surgery</td>
</tr>
<tr>
<td></td>
<td>- Treatment of underlying conditions</td>
</tr>
<tr>
<td></td>
<td>- Dietary modifications like increased consumption of iron rich food and reduction in bovine milk intake</td>
</tr>
<tr>
<td></td>
<td>- Specific treatment of iron deficiency anemia</td>
</tr>
<tr>
<td></td>
<td>Iron supplementation using oral formulations of iron like ferrous sulfate (elemental iron of 20%) or ferrous fumarate (elemental iron of 33%) at doses of 3 mg/kg/day up to a maximum of 6 mg/kg/day. Blood counts and indices can be repeated after 4 weeks wherein the Hb should have increased by at least 1 g/dL. The rate of rise in Hb is the same with oral and parenteral iron therapy</td>
</tr>
<tr>
<td>2.</td>
<td>Reduction of diagnostic, therapeutic, or intraoperative blood loss</td>
</tr>
<tr>
<td></td>
<td>- Spinal anesthesia advantageous (reduced blood loss)</td>
</tr>
<tr>
<td></td>
<td>- Total intravenous anesthesia preferable to inhalational anesthesia (reduced blood loss)</td>
</tr>
<tr>
<td></td>
<td>- Limiting intensity and duration of controlled hypotension</td>
</tr>
<tr>
<td></td>
<td>- Notifying the operating surgeon of the child’s hematological state so that blood loss during the surgery is minimized</td>
</tr>
<tr>
<td></td>
<td>- Use of minimally invasive surgery, modern coagulation techniques and other measures to reduce intraoperative blood loss</td>
</tr>
<tr>
<td></td>
<td>- Optimizing hemostatic and pharmacological means to reduce blood loss and transfusions</td>
</tr>
<tr>
<td></td>
<td>- Improving circulatory status and treating cardiac risk factors preoperatively</td>
</tr>
<tr>
<td>3.</td>
<td>Increasing individual tolerance towards anemia and accurate blood transfusion triggers</td>
</tr>
<tr>
<td></td>
<td>- Assessing pulmonary functions and administering oxygen even if minor reduction in hemoglobin is encountered</td>
</tr>
</tbody>
</table>

### CLINICAL FEATURES

The manifestations of nutritional anemia are varied and may range from anorexia, irritability and pallor (Hb between 6 to 10 g/dL; mild to moderate anaemia) to palpitations, easy fatigability and systolic murmur (Hb less than 5 g/dL; severe anemia). Diagnosis is based on parameters, such as hemoglobin, red blood cell indices, reticulocyte count, peripheral smear and biochemical indicators, such as total iron binding capacity (TIBC), transferrin saturation, serum iron and serum ferritin.

### PREOPERATIVE AND INTRAOPERATIVE OPTIMIZATION

The presence of anemia reduces the oxygen carrying capacity of blood. Transfusions enable to improve the oxygen transport capacity and increased regional or global availability of oxygen. However, the availability of outcome data analyses has clearly shown a direct association between transfusion of blood products and increased frequency of perioperative complications. Thus, Patient Blood Management (PBM) has been introduced as a multidisciplinary and evidence-based therapeutic approach to optimize use of blood products (Box 2).

**INDICATIONS OF BLOOD TRANSFUSIONS**

1. If Hb is less than 11 g/dL, in chronic oxygen dependent state.
2. If Hb is 7 g/dL or less, in patients who do not have any chronic illness/diseases.
3. Patients symptomatic due to anemia.
4. An intraoperative blood loss of ≥15% of total blood volume or a hematocrit of <24% with signs and symptoms of anemia, intraoperative blood transfusion may be needed.
5. First 24 hours and neonates in intensive care if Hb is less than 12 g/d (Hct = 0.36).

Thus, based on the above knowledge, a decision to transfuse should be made in adjunction to the assessment of clinical parameters, only if tissue oxygenation is likely to be compromised. Any decision to transfuse should be made with awareness of the fact that administering erythrocytes is likely to have medium to long-term disadvantages for that patient.
Chapter 32: Common Pediatric Medical Conditions: Anesthetic Considerations

EOSINOPHILIA

INTRODUCTION

Eosinophils are specially designed cells of the innate immune system. They are formed in the bone marrow and then move to the blood vessels. Eosinophils are implicated in numerous inflammatory processes and possess antiparasitic and bactericidal activity. Eosinophilia is said to be present when the eosinophil count exceeds 500 cells/cu mm. Accordingly, eosinophilia can be categorized as mild (500–1500/cu mm), moderate (1500–5000/cu mm) or severe (>5000/cu mm). Peripheral eosinophilia can be classified as primary, secondary or idiopathic (Box 3).12

EVALUATION OF EOSINOPHILIA

Eosinophilia is further evaluated on basis of history, examination and laboratory investigations as described in Table 4. Some of the investigations are mandatory in work up of a child presenting with eosinophilia, whereas further testing can be planned on an individual basis.

PREOPERATIVE OPTIMIZATION

Patients with eosinophilia when exposed to general anesthesia, have a risk of developing complications like urticaria, bronchospasm, coagulopathy and acquired respiratory distress syndrome during the perioperative period.15 Hence, it is necessary to investigate and treat the eosinophilia prior to an elective surgery. Management of eosinophilia is usually dependent on the underlying disease state and severity of organ dysfunction. Treatment of the underlying inciting infection or parasitic infestation generally controls the hypereosinophilia.14 Since parasitic infestation is very common in tropical countries, and often latent, it is prudent to empirically treat a child with eosinophilia with antihelminthics while awaiting further investigations. Albendazole can be administered at a single dose of 200 mg (if less than 2 years of age/10 kg weight) or 400 mg (if more than 2 years of age/10 kg weight) or Mebendazole can be given at dose of 100 mg twice daily for 3 days. In case there is no identifiable cause and no response to antihelminthics, an empiric trial of Diethylcarbamazine at a dose of 6 mg/kg/day can be given for 10–21 days. If there is no response to DEC and a clonal disorder is ruled out, a trial of steroids is warranted (can/may be given in consultation with a hematologist). Based on the response, the steroids can be tapered over 4–6 weeks. In the event of persistent eosinophilia, trials with immunosuppressants, such as imatinib mesylate, vincristine, hydroxyurea and mepolizumab have been recommended with specialist opinion.

INTRAOPERATIVE MANAGEMENT

Eosinophilia promotes entry of inflammatory mediators by inducing mast cells and basophils to release mediators and cationic proteins, thereby increasing the risk of intraoperative bronchospasm.16 Intravenous hydrocortisone is thus recommended at 2 mg/kg in three divided doses prior to surgery as well as on the morning of surgery to prevent life-threatening complications.6 Histamine-releasing drugs like atracurium, mivacurium, thiopentone, morphine, suxamethonium should be avoided, whereas drugs which facilitate bronchodilation, such as propofol, ketamine, vecuronium, rocuronium and fentanyl are preferred for anesthesia. Despite the above measures, if bronchospasm does occur intraoperatively, it should be managed symptomatically with inhaled β2 agonists, corticosteroids, magnesium sulfate or aminophylline.

POSTOPERATIVE MANAGEMENT

Due to description of patients with hypereosinophilia developing severe life-threatening complications postoperatively, diligent monitoring of such patients undergoing anesthesia is essential.
### Table 4: Evaluation of eosinophilia\textsuperscript{13,14}

<table>
<thead>
<tr>
<th>History</th>
<th>Physical Examination</th>
<th>Laboratory Investigations</th>
</tr>
</thead>
</table>
| Allergic symptoms  
International travel  
Recent/current medications  
Constitutional symptoms  
Symptoms of end organ involvement (Skin, Lungs, Gastric, Cardiac, Neurologic) | Allergic manifestations  
(atopic tendency, rhinitis, wheezing)  
Evidence of end organ involvement (eczema, angioedema, bronchospasm, myocarditis, right/ left ventricular failure, hepatosplenomegaly) | Complete Blood Count with differential leucocyte count\*  
Liver and kidney function Tests\*  
Stool microscopy\* (at least 3 specimens for ova/ parasites)  
Urine analysis\*  
Blood microscopy for microfilaria\*  
Serological tests for parasites\*  
Chest X-ray\*  
Further Investigations if unexplained/ persistent hypereosinophilia:  
- Studies specific to focal findings (imaging studies, spinal fluid, blood smear, tissue biopsy, etc.)  
- Viral serologies, Antinuclear antibodies (ANA), Antineutrophil cytoplasmic antibody (ANCA)  
- Serum total immunoglobulin E, and specific tests for allergy (skin prick tests and allergen specific IgE tests) if indicated  
- Bone marrow aspiration and biopsy  
- Cytogenetic analysis on bone marrow aspirate  
- Molecular analysis on peripheral blood cells for PDGFRα, PDGFRβ and FGFR1 gene rearrangements  
- Serum tryptase, serum erythropoietin, serum vitamin B12 and JAK2 mutation analysis  
- Investigation of blood T-cells (immunophenotyping and molecular analysis) for possible cytokine-driven eosinophilia (T-HES)  
- Imaging studies (CT scan, ultrasound) of chest and abdomen for underlying lymphoma or non-haematological malignancy  
- Serum troponin and ECG/echocardiogram  
- Pulmonary function tests and bronchoalvelolar lavage if clinically indicated  
- Serum interleukin 5 concentration (if available) |

*Recommended in all cases at baseline

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**INTRODUCTION**

Diabetes mellitus (DM) is a common, chronic, metabolic syndrome characterized by hyperglycemia as a cardinal biochemical feature. Type-1 diabetes is caused by pancreatic β cell destruction, usually immune-mediated, that results in absolute insulin deficiency. Type-2 diabetes, previously a disorder that predominantly affected middle-aged and elderly adults, results from a combination of insulin resistance and a relative deficiency of insulin.\textsuperscript{18}

In children, other causes for diabetes include genetic defects of β cell function (Maturity-onset diabetes of the young (MODY), Mitochondrial disorders), disease of the exocrine pancreas (Cystic fibrosis-related diabetes), drug-induced diabetes (Steroids, Chemotherapeutic drugs), genetic syndromes (Prader-Willi syndrome, Down syndrome, Turner syndrome, Wolfram syndrome) and endocrinopathies (Autoimmune polyglandular syndrome, Cushing syndrome).\textsuperscript{19}

**MANAGEMENT OPTIONS FOR DIABETES MELLITUS IN CHILDREN**\textsuperscript{18}

Type-1 diabetes will always require insulin treatment. Various preparations of insulin are available, such as bolus rapid acting (Aspart, Glulisine, Lispro), bolus short acting (Regular), basal intermediate acting (NPH) and basal long acting (Detemir, Glargine). Type-2 diabetes—most pediatric patients are managed with insulin and/or metformin. Metformin is an FDA approved drug for pediatric use.\textsuperscript{20}

**RISKS ASSOCIATED WITH DIABETES MELLITUS**\textsuperscript{20}

Insulin being an anabolic hormone promotes reuptake of glucose by the muscle and adipose tissue. However, during periods of stress like surgery, its production is suppressed and counter-regulatory hormones are released. These hormones, such as cortisol, glucagon, growth hormone
and epinephrine inhibit glucose uptake and use muscle and fat, stimulate gluconeogenesis, glycolysis (liver), lipolysis and ketogenesis. Along with the enhanced catabolism precipitated by stress of surgery and the starvation preceding and during surgery, these changes predispose a diabetic child to hyperglycemia and diabetic ketoacidosis. Hyperglycemia, on the other hand may adversely affect neutrophil function and impair collagen production, thereby increase the risk of postoperative infections and delay in wound healing, respectively. Due to this adverse metabolic response to surgery and anesthesia and untoward consequences of hyperglycemia, it is deemed essential to maintain normal levels of blood glucose (BG) in standard perioperative care of patients with diabetes.

**PREOPERATIVE ASSESSMENT**<sup>20-22</sup>

When a diabetic child is scheduled for an elective surgery, the preliminary assessment should review parameters such as operative risks (General risk factors, such as respiratory, cardiac, renal, hematological and Diabetes associated risks such as macrovascular, microvascular and neuropathic complications), diabetes treatment regimen (type, dosage and timing of medication), timing of meals and carbohydrate content, routine activity levels, frequency, severity and signals of hypoglycemia, and details of planned surgery (type of procedure, anesthesia, start time and duration of surgery).

After a careful assessment and evaluation of the laboratory parameters (blood glucose, electrolytes, ketones and glycosylated hemoglobin [HbA1C]) at least 10 days presurgery, the child is posted for surgery if the metabolic control is acceptable, i.e. no ketonuria, normal electrolytes and glycosylated hemoglobin (HbA1C) close to or within therapeutic range (6.5–7.5 %). If not, surgery has to be cancelled and rescheduled till an adequate glycemic control is achieved. For convenience of management, surgeries are divided into major surgery (prolonged general anesthesia, higher risk of metabolic decompensation and warranting at least an overnight hospital stay) and minor surgery (heavy sedation or brief general anesthesia, usually less than an hour duration and discharge on same day). It is prudent to post any diabetic patient requiring surgery as first case in the morning to prevent prolonged fasting and easy modification of diabetes treatment schedules.

**PREOPERATIVE MANAGEMENT OF A CHILD POSTED FOR A MAJOR SURGERY**<sup>22</sup>

The preoperative management of a child with Type 1 or Type 2 diabetes on insulin and undergoing a major surgical procedure is summarized in Box 4.

---

**Box 4:** Preoperative management of diabetes mellitus in children<sup>2</sup>

**General recommendations**

- Admission for general anesthesia
- Insulin continued (even if fasting) to avoid ketoacidosis; concomitant glucose infusion started if fasting >2 hours
- Careful capillary blood glucose monitoring to adjust insulin appropriately
- Infuse normal saline 0.9% (NS) or Ringer’s lactate rapidly in case of an unexpected drop in blood pressure due to vasodilatation during anesthesia

**Evening prior to surgery**

- Frequent blood glucose monitoring
- Measure blood β-hydroxybutyrate and/or urinary ketone concentration if blood glucose is >250 mg/dL
- The usual evening/bedtime insulin and bedtime snack to be given
- Overnight IV insulin infusion may be needed if there is ketosis/ hyperglycemia

**On day of surgery**

- No solid food at least 6 hours prior to surgery
- Clear fluids up to 4 hours before surgery
- Omit the usual morning insulin dose unless BG > 250 mg/dL
- Insulin infusion with 5% glucose to be started at least 2 hours before surgery
- If hypoglycemia- use 10% glucose
- If hyperglycemia (>250 mg/dL) use 1/2 Normal Saline (NS) or NS without glucose and increase rate of insulin infusion; add 5% dextrose once BG falls below 250 mg/dL

- Measure blood ß-hydroxybutyrate and/or urinary ketone concentration if blood glucose is >250 mg/dL
- The usual evening/bedtime insulin and bedtime snack to be given
- Overnight IV insulin infusion may be needed if there is ketosis/ hyperglycemia

**The intraoperative fluid management is summarized in Box 5.**

**PREOPERATIVE MANAGEMENT OF A CHILD POSTED FOR MINOR SURGERY (TABLE 5)**<sup>23</sup>

General instructions include posting the procedure first on the list in the morning, fasting from solids at least 6 hours prior and from clear fluids 4 hours prior to surgery.

**EMERGENCY PROCEDURE**<sup>22</sup>

Often, children with diabetes require emergency procedures. In such cases, it is prudent to keep the child nil by mouth; necessitating decompression of gastric contents in certain situations. At admission, an IV access should be achieved and parameters, such as weight, blood glucose, serum electrolytes, blood gases and serum/ urinary ketones should be evaluated prior to anesthesia. Any evidence of diabetic ketoacidosis should be managed as per protocol and surgery can be performed after volume and electrolyte deficits are rectified. In the absence of diabetic ketoacidosis, the management using IV fluids and insulin infusion is similar to an elective procedure.
**Table 5:** Preoperative management of diabetes mellitus prior to a minor surgery²³

<table>
<thead>
<tr>
<th>Patients treated with twice daily insulin regimen</th>
<th>Patients treated with basal bolus regimen</th>
<th>Patients on insulin pumps</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Morning operation between 8:00 AM to 9:00 AM</strong></td>
<td><strong>Morning operation between 8:00 AM to 9:00 AM</strong></td>
<td><strong>Morning operation between 8:00 AM to 9:00 AM</strong></td>
</tr>
<tr>
<td><em>On day prior to surgery:</em> Usual dose of short/rapid-acting insulin and intermediate insulin</td>
<td><em>On day prior to surgery:</em> Continue basal insulin before minor procedures; decrease previous night dose of long-acting insulin by 20–30% if pattern of low morning blood glucose values</td>
<td>The SC infusion site has to be secured properly to prevent dislodgement and interruption of insulin supply</td>
</tr>
<tr>
<td><em>If BG between 100 and 200 mg/dL—continue the same infusion with blood glucose monitoring.</em></td>
<td><em>If BG &gt; 200 mg/dL—Administer correction factor</em>²⁴ dose of rapid acting insulin or adjust dose of regular insulin IV infusion as needed to keep blood glucose between 200 and 300 mg/dL</td>
<td>For short procedures (GA &lt; 1 hour), SC insulin delivery using insulin pump at usual basal rate for time of day with maintenance fluid containing 5% glucose</td>
</tr>
<tr>
<td><em>If BG falls below 100 mg/dL—increase the rate of glucose infusion.</em></td>
<td><em>If BG falls below 4 mmol/L (55 mg/dL); temporarily discontinue insulin for 10–15 minutes.</em></td>
<td>Monitor BG levels every 30–60 min and give correction doses of rapid insulin with pump OR Extra IV insulin to maintain perioperative targets of blood glucose A meal bolus when patient can feed orally Alternatively instead of continuous subcutaneous insulin infusion, IV insulin and glucose infusion can be given, till feeding is resumed</td>
</tr>
</tbody>
</table>

| **Breakfast is permitted** | The usual dose of rapid-acting insulin OR 50–60% of the usual short-acting insulin with usual dose of basal intermediate- or long-acting insulin IV fluids with 5% dextrose 2 hours after breakfast | Continue the usual morning regimen with small doses of rapid-acting insulin sos Once child can eat—give bolus insulin dose with food |
| **Light breakfast can be permitted** | IV fluids are started 2 hours before surgery (Table B) | |
**TYPE 2 DIABETES**

In children with Type-2 diabetes and on treatment with insulin, management during surgery is based on insulin guidelines for an elective procedure depending on insulin regimen (Box 4 and 5). For those on oral medications, discontinuation of Metformin (24 hours prior to surgery) and sulfonylureas/thiazolidinediones (on day of surgery) is mandatory. Blood glucose should be measured hourly and treatment with IV insulin (elective surgery) or SC insulin (minor procedure) should be initiated, if blood glucose rises above 10 mmol/L (180 mg/dL).

**POSTOPERATIVE MANAGEMENT**

Postprocedure, glucose infusion is continued with 1–2 hourly BG and ketone (urine or blood) monitoring till oral intake is tolerated. Thereafter, discontinue the glucose infusion and resume the normal diabetes regimen. If oral intake is not possible, IV glucose solution with electrolytes are continued. IV regular insulin infusion or intermittent boluses of SC rapid-acting insulin is used, with regular BG and ketone monitoring; aiming a BG of 100–200 mg/dL. Prior to discharge, variable metabolic responses due to stress of surgery, poor oral intake due to pain/nausea/vomiting, decreased activity, drugs and postoperative infections should be anticipated and addressed.

The management of diabetes mellitus in the perioperative period is thus a dynamic process with need to target near normal blood glucose levels to minimize complications. The choice of regimen depends on the individual patient, the clinical profile and resources available.

**HYPOTHYROIDISM**

**INTRODUCTION**

A decrease in the production of thyroid hormone is termed hypothyroidism. The cause for hypothyroidism may be due to a pathology in the thyroid gland (primary hypothyroidism—congenital/acquired), pituitary gland (secondary hypothyroidism) or in the hypothalamus (tertiary hypothyroidism). Its manifestations are varied and often a delay in the diagnosis of hypothyroidism in infancy leads to retardation of both physical and mental development. Diagnosis of hypothyroidism requires estimation of serum TSH, T3 and T4 levels (Table 6).

Additional tests to determine the etiology include levels of serum thyroglobulin, antiperoxidase antibodies or antithyroglobulin antibodies and a thyroid scan in children with a palpable goiter. Treatment of choice for children with hypothyroidism is levothyroxine at a dosage of 10–15 µg/kg/day in infancy, 4–6 µg/kg/day for children 1–3 years; 3–5 µg/kg/day for 3–10 years; and 2–4 µg/kg/day for 10–16 years.

**ANESTHETIC IMPLICATIONS OF HYPOTHYROIDISM**

The physiological changes induced by hypothyroidism may lead to anesthesia related issues which are summarized in Table 7. Though mild or well-controlled hypothyroidism hardly affects anesthesia, severe hypothyroidism could lead to numerous complications.

**PREOPERATIVE EVALUATION**

- Thyroid functions should be assessed prior to surgery to ensure that the patient is euthyroid. If the disease process is under adequate control in previous 3–6 months, no other tests are required
- In a patient recently diagnosed with thyroid disease or hypothyroid despite medications, surgery is preferably deferred till a euthyroid state is achieved. Those who do not respond to oral replacement should be treated with intravenous T3 (loading dose of 0.7 mcg/kg) followed by an infusion titrated to T3 and TSH levels.
- Assessment for severe hypothyroidism which includes patients with myxedema, coma, features of chronic hypothyroidism (altered mental status, pericardial effusion and heart failure) or a total thyroxine of <1.0 mcg/dL or free thyroxine <0.5 ng/dL is essential. Due to instances of sudden death in children with myxedematous heart disease, they should receive one fourth of the maintenance dose of thyroid hormone with increments over 2–4 weeks till maintenance dose achieved.
Epilepsy

INTRODUCTION

Epilepsy is a disorder characterized by predisposition to generate seizures. It is twice as common in children as compared to adults. The clinical diagnosis of epilepsy requires the occurrence of at least one unprovoked epileptic seizure with either a second such seizure or electroencephalogram (EEG) and clinical findings that demonstrate a predisposition to develop recurrences.

Surgical procedures in an epileptic patient may pose
Chapter 32: Common Pediatric Medical Conditions: Anesthetic Considerations

a multitude of problems for the anesthetist during the perioperative period. A thorough evaluation of the child with due attention to medication and triggering agents is essential prior to anesthesia. Furthermore, seizure disorders are frequently accompanied by behavioral difficulties, multisystem abnormalities and syndromic associations which may have also anesthetic implications.1

PHYSIOLOGICAL AND PHARMACOKINETIC CONSIDERATIONS

Chronic administration of antiepileptic medications can have several implications with regard to drug interactions and adverse effects with concomitant anesthetic use. Given below are a few aspects to be considered during anesthesia in an epileptic patient (Box 6).28

PREANESTHETIC EVALUATION

Preoperative evaluation should include a targeted but thorough history including the history of the last seizure, type of seizure, frequency, factors triggering seizures, risk factors for aspiration and the medications used. Attention should be paid to the disease states associated with seizure disorders like cerebral palsy, trisomy 21, Angelman syndrome, Sturge-Weber syndrome, DiGeorge syndrome, tuberous sclerosis, neurofibromatosis, craniosynostosis and Lesch Nyhan syndrome. In addition, the child should be evaluated for the adverse effects of certain antiepileptics that may add to the comorbidity. Regular antiepileptics should be continued up to the time of surgery and restarted immediately after surgery. If enteral intake cannot be restored in the immediate postoperative period an alternative antiepileptic medication should be selected after discussion with the neurologist or attending pediatrician. Since hyperventilation can trigger seizures, premedication with benzodiazepines is beneficial in anxious children.1

The role of anesthesiologist can be discussed under the following modalities:
1. Management of Status Epilepticus.
2. Perioperative management for epilepsy surgery.
3. Perioperative management for nonepilepsy surgery.
4. Postoperative management.

Management of Status Epilepticus

Status epilepticus is defined as a seizure lasting longer than 30 minutes or recurrent seizures without an interictal return to the baseline clinical state. Seizures during anesthesia are difficult to diagnose and may be missed.

**Box 6: Aspects to be considered during anesthesia in an epileptic patient**

**Antiepileptic drug and anesthetic implications**
- Phenobarbitone—Hepatic microsomal enzyme induction
- Valproic acid—Displaces diazepam from the binding site, hepatic microsomal enzyme inhibitor
- Phenytoin—Induces oxidative metabolism of drugs including thiopentone, propofol, midazolam, opioids and nondepolarizing neuromuscular blockers
- Carbamazepine—Enhances hepatic oxidation and conjugation of liposomal drugs, hyponatremia
- Lamotrigine—Induces hepatic microsomal system
- Primidone—Chronic use induces metabolism of enflurane, halothane and isoflurane
- Zonisamide—Metabolic acidosis
- Clonazepam—Sialorrhea and increased bronchial secretion
- ACTH and Corticosteroids—Addisonian crisis during surgery

**Anesthetic drugs and epileptic implications**
- Benzodiazepines—First-line anticonvulsant besides sedation purpose
- Propofol—Antiepileptic activity; used for sedation, induction and maintenance of general anesthesia, however, can cause abnormal movements (opisthotonus, myoclonia) in normal as well as epileptic patients
- Etorphidol—Anticonvulsant (high-doses) as well as proconvulsant (clinical doses)
- Ketamine—Epileptogenic; best avoided
- Droperidol—No proconvulsant activity
- Dexmedetomidine—Neither proconvulsant nor anticonvulsant activity. Beneficial as prolongs analgesia of local anesthetics in neuraxial blocks, attenuates pressor response when used before induction, reduces postoperative analgesic need and shivering postoperatively after general anesthesia
- Clonidine—Similar to dexmedetomidine when used as above but has lesser affinity at alpha receptors
- Barbiturates—Significant anticonvulsant activity (except methohexital)
- Opioids—Meperidine is proconvulsant. Morphine, may cause tonic-clonic activity when used in epidural space. High doses of phentanyliperidine derivatives (fentanyl, alfentanil, sufentanil and remifentanil) should be avoided in epileptic patients
- Nitrous oxide—Epileptogenic potential is very low and safe in epileptic patients
- Halothane, Isoflurane—Potent anticonvulsant activity
- Enflurane—Contraindicated in epileptic patients
- Sevoflurane—Avoid use at concentration above 1.5 MAC and in presence of hypoponxia
- Desflurane—No proconvulsant activity

**Neuromuscular blockers**
- i. Nondepolarizing blockers: Hepatic clearance of rocuronium, pancuronium, vecuronium, and cisatracurium is increased by chronic use of phenytoin and carbamazepine, while atracurium and mivacurium are unaffected
- ii. Depolarizing blocker succinylcholine: Duration of action is slightly increased
- Anticholinesterases—No proconvulsant activity
- Anticholinergics—Atropine, scopolamine and glycopyrrolate can be used in epileptics

Local anesthetics—Can induce seizures at higher concentrations
- Antiemetics—Dopamine antagonists can cause extrapyramidal effects and dystonia; confused with epileptic activity
If suspected, management should be immediately initiated with 100% oxygenation, deepening of anesthesia, administering anticonvulsants and correcting precipitating factors. Treatment for status epilepticus should be initiated when a seizure lasts for more than five minutes. The management of status epilepticus is outlined below (Box 7).

**Epilepsy Surgery**

Children with refractory seizures often require surgical resection of epileptogenic focus or seizure pathway. This may be done under general anesthesia or local anesthesia with sedation. The anesthetist may be involved in various ways ranging from monitoring anesthesia during awake craniotomy, inducing intraoperative seizures for electrocorticography (ECoG), maintaining intracranial hemodynamic, facilitating early postoperative recovery for neurological evaluation and managing refractory seizures. It may also extend to anesthesia for preoperative evaluation, such as radio-imaging, stereotactic electrode insertion, localization of seizure focus (thiopentone test) and detection of dominant cerebral hemisphere (Wada test using amobarbital). The intraoperative management during epilepsy surgery is summarized in Box 8.

**Non-epilepsy Surgery**

The monitoring of children undergoing nonepilepsy surgery is dictated by the surgical procedure and the clinical condition. Intensive monitoring is needed in major surgeries and critical clinical states. For induction thiopental, propofol and benzodiazepines are the preferred agents. Ketamine and etomidate should be avoided. Total intravenous anesthesia with propofol and fentanyl or neuraxial opioids (other than meperidine) can be safely used. For maintenance, isoflurane is used due to its anticonvulsant property. Other alternatives include sevoflurane at low concentrations, halothane and desflurane. During the surgery, changes that can reduce seizure threshold like hypoxia, hypotension, hypocapnia and hyponatremia should be avoided. Coagulation parameters need to be monitored due to increased risk of coagulation disturbances by certain anticonvulsants. Postoperatively anticonvulsants should be restarted early, if fasting exceeds 24 hours, parenteral replacement is necessary.

**Postoperative Management**

Children with risk factors, such as poorly controlled epilepsy, surgery prolonged beyond effective period of last antiepileptic dose, pre-existing brain anomaly and use of certain epileptogenic anesthetic drugs and metabolic abnormalities, are more likely to have seizures in the post-operative period; often with deleterious consequences due to cardiorespiratory impairment causing brain damage. Prophylaxis is hence the mainstay of management. However, postoperative pseudoepileptic
conditions, such as postoperative shivering should also be ruled out. Phenytoin is usually used for post-operative prophylaxis. Currently, levetiracetam is gaining popularity over phenytoin for postoperative prophylaxis as it does not require serum drug level monitoring. Alternative agents which can be used are phenobarbital, carbamazepine, and valproic acid. Postoperative status may be controlled with lorazepam (0.1 mg/kg bolus IV over 2 minutes) or diazepam (0.5 mg/kg rectally). Lorazepam can be repeated, if necessary after 10 minutes and supplemented with Fosphenytoin (20 mg/kg IV or IM) if seizures persist. Phenobarbitone at 20 mg/kg may also be used as first line drug. Refractory seizures may be controlled by midazolam infusion (0.1–0.3 mg/kg in 2–5 minutes, followed by infusion of 0.05–0.4 mg/kg/h), propofol (1–2 mg/kg followed infusion of 2–10 mg/kg/h), thiopental (5–10 mg/kg in 10 minutes, followed by infusion of 100–400 mg/h), lidocaine (1.5–2 mg/kg in 2–5 minutes, followed by infusion of 2–3 mg/kg/h for 12 hours), isoflurane (0.5–1.5%).

PREPARING FOR SPLENECTOMY

INTRODUCTION

The spleen filters senescent and abnormal red blood cells and contributes to immune function and hosts defences against certain infectious agents; making individuals with asplenia/postsplenectomy predisposed to fulminant infections. Indications for splenectomy can be absolute (hereditary spherocytosis, primary splenic cancers) or relative (idiopathic thrombocytopenic purpura, trauma, abscesses, thalassemia, autoimmune disorders, hereditary elliptocytosis, and Hodgkin’s lymphoma). Splenectomy can be complete or partial and can be done through an open surgery or laparoscopic mediated.31

ANESTHETIC CONSIDERATIONS32,33

The laparoscopic approach is preferred unless certain contraindications like liver failure with portal hypertension, ascites or unmanageable coagulaopathy coexist. Open surgery is preferred where there is a probability of hemodynamic instability like in splenic trauma. The risks of laparoscopic surgery are by and large due to the physiological changes dictated by the creation of pneumoperitoneum and the position of the patient during the surgery.

PREOPERATIVE MANAGEMENT32

Parents should be counseled regarding the risks of splenectomy especially infection from encapsulated organisms. Vaccination against Pneumococcus, Meningococcus and Haemophilus influenzae should be administered at least four weeks prior to an elective surgery.35 The vaccination schedule is elaborated in Table 9.

Patients with thrombocytopenia should have a preoperative platelet count above 30,000/cu mm. In children with ITP, platelet transfusion is avoided until after surgery. However, if a rise in platelet count is desired prior to surgery, a steroid bolus or parenteral IVIG can be given. Hemoglobin of at least 10g/dL has to be maintained. Children who are chronically on steroids for the primary illness may need stress doses of steroids in the peri-operative period.

INTRAOPERATIVE MANAGEMENT34

• The gastric contents should be emptied to reduce the risk of visceral injury during trocar insertion. A stethoscope should be placed over the left sternal border at the line of the nipple to detect this complication
• Nitrous oxide is usually avoided as it supports combustion.
• Laparoscopy is associated with a high incidence of postoperative nausea and vomiting, which can be minimized by using a combination of propofol and remifentanil instead of inhalation anesthesia. Prophylactic treatment with antiemetics and histamine blockers can also be given
• Postoperative pain can be minimized by infiltrating bupivacaine at the incision site. Low dose intrathecal morphine, acetaminophen, non-steroidal anti-inflammatory agents, and other non-opioid analgesics may also help to reduce pain
• Neuromuscular blockade, endotracheal intubation, and positive pressure ventilation may help to reduce the pulmonary effects of the Trendelenburg position
• Intra-abdominal pressure should be maintained below 15 mm Hg, to target normal oxygen saturation
• Hypothermia is avoided by warming the insufflating gas or by maintaining insufflating flows of less than 2 L/min.
**Table 9: Vaccination of a child posted for splenectomy**

<table>
<thead>
<tr>
<th>Vaccine</th>
<th>Age</th>
<th>Schedule</th>
</tr>
</thead>
<tbody>
<tr>
<td>PCV 13</td>
<td>0–24 months</td>
<td>If a child has completed the primary course PLUS a booster dose (PCV7 or PCV10) after 12 months of age, give only one dose If aged 12–24 months and has not received a complete primary course, give two doses 8 weeks apart</td>
</tr>
<tr>
<td></td>
<td>24 months to 18 years</td>
<td>If aged &lt;5 years and has received a complete primary course PLUS a single booster dose (PCV7 or PCV10) after 12 months, give one dose If aged 24 months—5 years and has not received a complete primary course, give two doses 8 weeks apart If aged 5–18 years—one dose</td>
</tr>
<tr>
<td>PPSV 23</td>
<td></td>
<td>One dose from 2 years of age, at least 8 weeks after the last PCV13 dose. If aged &lt;10 years at time of first 23PPV, revaccinate once after 3 years. If aged ≥10 years at time of first 23PPV, revaccinate once after 5 years</td>
</tr>
<tr>
<td>Hib</td>
<td>0–18 months</td>
<td>Age appropriate immunization</td>
</tr>
<tr>
<td></td>
<td>18 months to 5 years</td>
<td>Primary schedule—6 weeks, 10 weeks, 14 weeks and a booster dose at 1.5 years of age</td>
</tr>
<tr>
<td></td>
<td>More than 5 years</td>
<td>If the child has not received a single Hib dose after 12 months of age, give one dose Give one dose even if fully vaccinated</td>
</tr>
<tr>
<td>Meningococcal vaccine (ACWY-135)</td>
<td>From 2 years of age</td>
<td>One dose from 2 years of age If aged ≤5 years at time of first dose, revaccinate once after 2–3 years If aged ≥6 years at time of first dose, revaccinate once after 3–5 years</td>
</tr>
</tbody>
</table>

Abbreviation: PCV, Pneumococcal conjugate vaccine; PPSV, Pneumococcal polysaccharide vaccine; Hib, *Haemophilus influenzae* type B vaccine.

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**POST-OPERATIVE MANAGEMENT**

Acute complications post-splenectomy include postoperative bleeding, subphrenic abscess, pulmonary atelectasis, bronchopneumonia and left pleural effusion. It is important to identify these events in due time and call for adequate conservative or surgical treatment. After splenectomy, there is also an increase in the number of thrombocytes, which might lead to thromboembolic complications. Anticoagulation therapy is thereby suggested. Delayed complication includes postsplenectomy sepsis which can be avoided by early detection of infections and use of prophylactic oral penicillin V (125 mg twice daily for children younger than 5 year; 250 mg twice daily for children 5 year or older) for at least 2 year after splenectomy (to at least 6 year of age).

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**SYNDROMIC CHILD**

**INTRODUCTION**

More often than not, one encounters a child who appears ‘different’. While evaluating these children, a few terminologies should be borne in mind. A syndrome is defined as a consistent pattern of multiple abnormalities that are related by pathophysiology and result from a common, defined etiology. Sequences consist of multiple malformations that are caused by a single event that can have many etiologies. An association refers to a nonrandom collection of malformations in which there is an unclear relationship among the malformations such that they do not fit the criteria for a syndrome or sequence.

**ETIOLOGY**

The cause for dysmorphism can be attributed to genetic or nongenetic disorders.

- Genetic disorders
  1. Chromosomal disorders like aneuploidy, deletions.
  2. Single gene disorders
  3. Multifactorial disorders
- Nongenetic disorders like teratogens which cause irreversible damage to the developing embryo.

**PREANESTHETIC EVALUATION**

The family history or pedigree should be outlined in detail to determine the pattern of inheritance, if any. A three generation pedigree is sufficient. The perinatal history with particular reference to the obstetric history, exposure to potential teratogens and antenatal factors like oligohydramnios has to be obtained. It is prudent to have a pediatric consult, often requiring a specialist opinion if the syndrome is complex. A thorough physical examination should be performed to note all the...
dysmorphisms. Imaging studies are ordered depending on the physical aberrations detected. This may include a skeletal survey, ultrasound evaluation, echocardiography or neuroimaging. Cytogenetics with Giemsa-banded peripheral leukocyte karyotype analysis is the gold standard in the evaluation of the dysmorphic child.

**ANESTHETIC CONCERNS**

**Systemic Abnormalities**

Many of the syndromes are multisystemic in origin. The systemic involvement and associated anesthetic issues are summarized in Box 9:

<table>
<thead>
<tr>
<th><strong>Box 9: Systemic involvement in syndromic children</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cardiac disease</strong></td>
</tr>
<tr>
<td>Usual abnormalities detected are cardiomyopathies,</td>
</tr>
<tr>
<td>valvular lesions, endocardial cushion defects,</td>
</tr>
<tr>
<td>hypertension, cardiac failure, arrhythmias</td>
</tr>
<tr>
<td>Intraoperative cardiac instability thereby</td>
</tr>
<tr>
<td>can lead to disastrous consequences</td>
</tr>
<tr>
<td><strong>Renal disease</strong></td>
</tr>
<tr>
<td>May be a primary feature or syndromic association</td>
</tr>
<tr>
<td><strong>Skeletal deformities</strong></td>
</tr>
<tr>
<td>Pulmonary function tests are indicated in such</td>
</tr>
<tr>
<td>children Imaging of the cervical spine is a must to</td>
</tr>
<tr>
<td>preclude a difficult airway Chest radiographs and</td>
</tr>
<tr>
<td>pulmonary function tests may be needed Perioperative</td>
</tr>
<tr>
<td>phyotherapy and bronchodilators are important in</td>
</tr>
<tr>
<td>certain conditions like cystic fibrosis</td>
</tr>
<tr>
<td><strong>Respiratory problems</strong></td>
</tr>
<tr>
<td>These can be secondary to skeletal deformities or</td>
</tr>
<tr>
<td>myopathies Concomitant gastroesophageal reflux can</td>
</tr>
<tr>
<td>worsen the condition</td>
</tr>
<tr>
<td><strong>Metabolic abnormalities</strong></td>
</tr>
<tr>
<td>Liver function tests, urea and electrolytes</td>
</tr>
<tr>
<td>are important preoperative investigations in such</td>
</tr>
<tr>
<td>children</td>
</tr>
<tr>
<td><strong>Hemopoietic Abnormalities</strong></td>
</tr>
<tr>
<td>Abnormalities can range from depression of a single</td>
</tr>
<tr>
<td>cell line to pancytopenia</td>
</tr>
<tr>
<td>Blood loss during surgery can aggravate the</td>
</tr>
<tr>
<td>condition and should be anticipated</td>
</tr>
<tr>
<td><strong>Neuromuscular system</strong></td>
</tr>
<tr>
<td>Sudden cardiac arrest, idiosyncratic reactions to</td>
</tr>
<tr>
<td>neuromuscular blockers are known to occur</td>
</tr>
<tr>
<td><strong>Central nervous system</strong></td>
</tr>
<tr>
<td>Children with neurodegeneration are</td>
</tr>
<tr>
<td>predisposed to seizure disorders and need</td>
</tr>
<tr>
<td>anticonvulsant therapy</td>
</tr>
</tbody>
</table>

**Malignant Hyperthermia**

A few neuromuscular disorders place children at a higher risk for malignant hyperthermia, which is characterized by rising end tidal carbon dioxide, tachycardia, muscle rigidity, acidosis and hyperthermia and most often occurs during anesthetic administration. Dantrolene is the mainstay in treatment of malignant hyperthermia. A trigger free anesthesia can prevent the aforementioned complication.

**Difficult Airway**

An airway assessment and anticipation of possible difficult intubation should be done in children with syndromes and dysmorphism, where a difficult airway is expected to be present due to anatomical abnormalities and hypotonia.

**Difficult Intravenous Access**

The veins of children in certain syndromes are difficult to cannulate due to increased subcutaneous tissue. Care must be taken to prevent hypothermia during this period, particularly in an infant or neonate.

**Patient Anxiety/Poor Cooperation**

Children with syndromes may have mental retardation and behavioral abnormalities like autism or hyperactivity making induction of anesthesia tough. Establishing a rapport, judicious use of premedication and the presence of a parent can help to make the experience less traumatic for the child.

Thus, many congenital anomalies are inter-related. The presence of one anomaly should prompt the anesthesiologist to search for others, particularly those that may influence anesthetic outcome. Recognition of patterns will help to exclude the important life-threatening anomalies that can influence choice of anesthetic and long-term outcome.

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INTRODUCTION

Cerebral palsy (CP) is a set of neurological disorders characterized by varying degrees of motor, sensory, and intellectual impairment.¹

The incidence of ~1 in 500 live births has changed little over the past 30 years, despite improved public health, antenatal care, and an increase in rate of cesarean section to prevent neonatal asphyxia.² This constancy is attributed to an increase in survival rate of premature babies, in whom there is a recognized 10–50 fold increase in prevalence of CP.³

PATHOPHYSIOLOGY

Majority of CP cases (~70%) are associated with one or more pathogenic factors outlined. In the remaining 30%, no pathogenesis is identified.

Pathogenic Risk Factors for CP⁴⁻⁹

Congenital CP (80%)

Fetal pathogenic factors
- Vascular maldevelopments
- Prenatal ‘TORCH’ infections
- Congenital genetic/metabolic disorders
- Low Apgar Score
- Microcephaly
- Prematurity (<32 weeks)
- Fetal trauma
- Multiple births
- Neonatal asphyxia in the peripartum period (6%)
- Low-birth-weight (<2.5 kg).

Maternal pathogenic factors
- Breech presentation
- Preeclampsia
- Peripartum hemorrhage
- Maternal hyperthyroidism
- Fetal alcohol syndrome.

Acquired CP (20%)—Develops during the First 2 Years of Life
- Intracerebral hemorrhage
- Viral encephalitis
- Bacterial meningitis
- Hyperbilirubinemia (Kernicterus)
- Head injury
- Neonatal seizures.

The series of structural and functional changes in the brain can be disrupted by different pathogenic insults at different stages of its development. As a result, unique patterns of injury are produced which are clinically associated with characteristic neurological signs.
Bilateral spastic CP is the most common subtype of CP. Periventricular lesions can be identified on MRI in ~90% of those children born prematurely who go on to develop the clinical signs of this subtype of CP (diplegias and tetraplegias) in the post-natal period.\(^\text{10}\)

**CLASSIFICATION**

The CP has been classified according to a patient’s resting muscle tone:

- **Spastic 70%:** Lesion in cerebrum (most common clinical manifestation)
  - Quadriplegia 27% of all cases of CP
  - Diplegia 21%
  - Hemiplegia 21%.

- **Dyskinetic 10%:** Lesion in basal ganglia (normal intellect, difficulties with communication)
  - Dystonia (maintained twisting position of torso and extremities)
  - Athetosis (slow, purposeless, distal movements)
  - Chorea (quick, jerky, proximal).

- **Ataxic 10%:** Lesion in cerebellum intention tremor and head tremor (titubation)

- **Mixed 10%:** Lesion in cerebrum and cerebellum, e.g. spastic athetoid.

A more comprehensive classification system that incorporates assessment of a patient’s motor function, physical activity, and psycho-social ability supersedes the above. These classifications enable clinicians to plan better day-to-day care for individual patients.\(^\text{11}\)

**CLINICAL PRESENTATION**

**Muscle Spasticity:** 70–80% of patients exhibit some degree of muscle spasticity. The lower limbs, when affected manifest as an abnormal ‘scissor’ gait.

Tendency to walk on ‘tip-toes’ if still ambulatory.

**Muscle Contracture:** Develops as a result of differential growth rates between long bones and spastic muscle groups.

Results in joint deformities and dislocations which go on to cause patients considerable problems with joint pain when walking and sitting.

Although CP is primarily a disorder of posture and movement, the more severe forms impact on patients’ neurological, respiratory, gastrointestinal, musculoskeletal, and urological functions.

**Neurological Disorders**

The CP happens to be the commonest cause (60%) of motor impairment in childhood. Two-third of patients have some degree of impaired intellectual and cognitive function, the magnitude of which may be difficult to determine because of problems with communication arising from expressive language disorders and or oromotor problems affecting speech.

Perioperative anxiety in patients could be heightened by communication issues hence due consideration should be given to anxiolytic premedication where appropriate.

Up to 50% of patients have either focal or generalized forms of epilepsy (particularly those with a history of neonatal encephalopathy) which may be poorly controlled. Other problems include visual and auditory impairment and abnormal touch and pain perception.

**Respiratory System**

Amongst those born prematurely, a significant number has underlying chronic lung disease secondary to neonatal respiratory distress syndrome. With increased risk of aspiration pneumonitis, there occurs chronic lung scarring because of swallowing difficulties, esophageal dysmotility, abnormal lower esophageal sphincter tone, and spinal deformity which lead to gastroesophageal reflux. Decreased immunity, secondary to poor nutrition, respiratory muscle weakness and a weak cough in conjunction with gastroesophageal reflux make patients susceptible to recurrent chest infections which exacerbate the underlying chronic lung disease. In the long-term truncal muscle spasticity can lead to scoliosis, restrictive lung defects, pulmonary hypertension and ultimately cor pulmonale and respiratory failure.

**Gastrointestinal System**

Generally small for their age, inability to chew or swallow food, or both coupled with hyperactive salivary gland\(^\text{12}\) leads to drooling and poor feeding. This in turn leads to malnutrition, dehydration, anemia, and electrolyte imbalances. Awkwardly placed loose teeth and poor dental hygiene are common, and coupled with TM joint dislocation secondary to muscle spasticity can make airway management during anesthesia challenging. A review of previous anesthetic records may highlight previous difficulties with airway maintenance or intubation.

**Musculoskeletal and Skin**

Fixed flexion deformities of the limbs and trunk as a result of muscle contractures, joint dislocations, scoliosis, and decubitus skin ulcers can make patient positioning, vascular access, and invasive monitoring for anesthesia challenging.
Nonweight-bearing long bones become osteopenic and vulnerable to fracture. CP muscle contracts poorly when surgically incised. This can result in significant blood loss during major surgery which may necessitate the use of red cell salvage devices, antifibrinolytic drugs such as tranexamic acid, and homologous blood transfusion. Heat loss is a major consideration in CP patients because they have thin skin, very little subcutaneous fat, and atrophic musculature. Morphologically, they can be likened to ‘giant neonates’ because they have large surface area to body weight ratios and cannot conserve heat effectively. Often these patients come to theater with borderline hypothermia (<35°C) and they should be actively warmed throughout the perioperative period.

Immobility limits a patient’s ability to exercise and the anesthesiologist’s ability to assess their cardiorespiratory reserve. It is worth noting that the incidence of ischemic heart disease in the adult CP patient population is higher than in the general adult population.

Urological Disorders

Neuropathic bladder, general immobility, communication or learning disability may lead to significant number of patients being incontinent. Patients who have undergone intermittent or indwelling urinary catheterization can be at increased risk of latex allergy as a result of multiple previous exposures.13

SURGICAL PROCEDURE

Surgical procedures for patients with CP are considered for two main reasons: to reduce tone in the extremities or the trunk and to correct the acquired skeletal deformities of long-standing spasticity.

Common surgical interventions for patients with CP include:
- Orthopedic procedures: Major multilevel surgery involving tenotomies/osteotomies, soft tissue release/tendon transfers, and botulinum toxin injections
- Dental extractions and restorations
- Gastrostomy and antireflux procedures
- Imaging
- Others: Neurosurgery, urology, ophthalmology, ENT and insertion of infusion devices.

ANESTHESIA TECHNIQUE

Preoperative Management

Communication and cognition happen to be the greatest challenges faced by anesthesiologist. Parent or guardian can make invaluable contribution, not only in terms of their expert knowledge of the patient’s past medical history, but also their ability to facilitate communication. Patients may present with significant comorbidity.

Assessment of hydration state, renal function, drug history, and cardiorespiratory functions are of vital importance. Detailed history and examination should guide the preoperative investigations and treatments required. Complete blood count, renal indices, electrolytes, blood grouping and cross-match would be a basic requirement for patients undergoing major surgery. Chest X-ray and ECG may be indicated in patients suspected of or known to have CHD or acute respiratory problems. Medications, particularly anticonvulsants and antispastics, should be continued in the perioperative to avoid problems with acute withdrawal and worsening of seizure control. Many of anticonvulsants have long half-lives and a tendency to accumulation. They can reduce MAC values by up to 30%14 and cause over-sedation, a slower recovery from anesthesia, and airway compromise.

Sedative premedication is best avoided in hypotonic patients because it may accentuate problems with upper airway muscle tone and increase the risk of aspiration perioperatively.

Intraoperative Management

Propofol is the preferred anesthetic induction agent in patients with reactive upper airways, because unlike thiopental, it decreases tone in the smooth muscle of the airway. Succinylcholine is not contraindicated in patients with CP. Although some studies have demonstrated the presence of extra-junctional Acetylcholine (ACh) receptors in up to 30% of CP patients, other studies have demonstrated no significant difference in potassium release after succinylcholine administration to children with CP when compared with non-affected children.15

Non-depolarizing muscle relaxants are less potent and have a shorter duration of action in patients with CP owing to the up-regulation of ACh receptors. Clinically, this is offset by the fact that these highly water-soluble drugs are redistributed through a smaller volume of total body water because these patients are often dehydrated. In those patients with proven gastroesophageal reflux, it would seem prudent to secure the airway with a tracheal tube.

Postoperative Management

The CP patients are prone to hypoxia, hypovolemia, and hypothermia. They often have a poor cough reflex and reduced respiratory drive, making them prone to secretion
retention, basal atelectasis, and pulmonary collapse. Attention is often required with regard to blood loss, hydration, circulatory status and temperature control.

Communication difficulties can make the assessment of postoperative pain difficult and postoperative analgesia should be based on ‘continuous’ rather than ‘on demand’ regimens. Acetaminophen and non-steroidal anti-inflammatory drugs should be given regularly and supplemented with intravenous morphine infusions or local anesthetic-based epidural infusions or other regional blocks as appropriate. Systemically and extradurally administered opioids should be used with caution in CP patients because they can accumulate resulting in oversedation, suppression of the cough reflex, and depression of respiration in this already vulnerable patient group.

Hypothermia, postoperative pain, and anxiety can all trigger acute muscle spasms which are often more painful and distressing to the patient than the operation itself. Caudal or epidural analgesia using a combination of clonidine with a local anesthetic agent are beneficial to counter both, the postoperative pain and the pain of muscle spasms.16

SPECIFIC PROBLEMS (PREOPERATIVE)

Regular oral anticonvulsants should be continued. Incase there is a nil per oral period indicated for the past 24 hour period on account of generalized convulsions, IV doses of same should be supplemented.

Severe muscle spasms due to spinal reflexes initiated by pain are frequent after lower limb orthopedic surgery. Diagnosis of compartment syndrome may be delayed by regional analgesics and impaired communication. Vigilant observation is required. Fixed contractures, prolonged pressure over bony prominences and malnutrition may lead to troublesome skin breakdown. Meticulous pressure care and frequent repositioning is required to prevent pressure sores.

Chronic pain requires a multidisciplinary approach. Differentiation of surgical pain from other causes of pain is important.

Perioperative chest physiotherapy helps those with poor cough, poor secretion clearance, and recurrent chest infections. Patients with poor respiratory function may need postoperative ventilation and even tracheostomy during the weaning process. Preoperative respiratory function tests are unreliable and not predictive of outcome.

Cerebral palsy children are prone to constipation because of reduced gut motility and fluid intake, and this is worsened by perioperative opioids. Use opioid sparing analgesia where possible and consider normal bowel habits for that child. The prescription of laxatives may be necessary.

LEARNING POINTS

- Cerebral palsy describes a spectrum of movement and posture disorders which result from pathological injury to the developing fetal or infant brain.
- Commonly associated co-morbidities include dehydration, malnutrition, epilepsy, gastro-esophageal reflux and impaired lung function.
- Frequently encountered perioperative problems include difficulties with patient positioning and vascular access.
- Regional analgesic techniques to reduce postoperative pain, muscle spasms, and respiratory complications are particularly beneficial.

REFERENCES

INTRODUCTION
Neuromuscular disorders (NMDs) in children have posed a wide variety of challenges for the anesthesiologists. They affect muscle strength by either directly weakening the muscle fibrils or indirectly by a degenerative nerve supply and weak neuromuscular junction. Majority of NMDs are genetic in origin. The anesthetic management of pediatric patients with NMDs can be very complicated and requires careful peri-operative planning. These children commonly present for anesthesia for diagnostic procedures (muscle biopsy, MRI), or surgery relating to their underlying disorder (gastrostomy, corrective orthopedic procedures, strabismus surgery), or incidental surgery.

PATHOPHYSIOLOGY
The normal skeletal muscle contraction involves:
- Input from efferent somatic nerves
- Acetylcholine release
- Motor end-plate stimulation
- Calcium release from the sarcoplasmic reticulum
- Contraction coupling of actin and myosin (myofibrils).

Abnormalities affecting normal muscle function can occur in the:
- Release or action of acetylcholine (myasthenic syndromes)
- Post-synaptic membrane and sarcoplasmic reticulum (channelopathies)
- Myofibrils (dystrophies and myotonias)
- Mitochondria (mitochondrial myopathies).

A vast number of muscle disorders in children have been described; however, the important ones as mentioned under have been dealt with here with an overview of the various causes, specific pathophysiology, and their impact on anesthetic management, to facilitate a sensible approach towards the perioperative management of these children, complications and cautions.

- Muscular dystrophy
- Myotonias
- Congenital myopathies
- Mitochondrial myopathies
- Channelopathies.

MUSCULAR DYSTROPHY
Duchenne Muscular Dystrophy (DMD)
An X-linked recessive inherited disorder, this is the most common childhood dystrophy with an incidence of 1:3500 live male births. There is marked abnormal or absent dystrophin, a large protein necessary to stabilize and link the myofibrils and cytoskeleton in skeletal, cardiac and smooth muscle. This defect results in chronic muscle fiber necrosis, degeneration and regeneration, as manifested in muscle weakness and pseudohypertrophy of calves. It also accounts for cardiac manifestations and mental retardation. DMD is progressive. Although a neonate may appear normal, serum creatine kinase (CK) will be elevated. A positive family history (90%) and delayed motor milestones are evident: late walkers, difficulty in climbing stairs by the age of 5 years, and wheelchair bound from age 6 year onwards. Paraspinal weakness leads to progressive kyphoscoliosis. Respiratory weakness causes decreased vital capacity and recurrent chest infections. Degenerative changes in cardiac muscles cause conduction deficits and cardiac failure/dilated cardiomyopathy. Patients also develop gastric hypomotility, delayed gastric emptying, and a hypertrophied tongue. They succumb to cardiac or pulmonary manifestations of the disease in their late 20s to early 30s.

Preoperative Assessments
- Chest X-ray: Signs of pulmonary congestion and cardiac enlargement
- ECG: Right ventricular strain, tall R waves, deep Q waves and inverted T waves
- PFT: Patients tend to develop restrictive respiratory pattern with reduction of forced vital capacity (FVC)
- Blood gases: Hypoxemia and CO₂ retention suggest necessity of postoperative ventilation
- Echocardiogram: Ventricular systolic dysfunction progressing to dilated cardiomyopathy.

Anesthetic Considerations
The clinical manifestations of DMD are bimodal: during early childhood when skeletal muscle is being destroyed, rhabdomyolysis and hyperkalemia can occur in response to triggers. Hence it is mandatory to ensure a trigger-free anesthetic and ‘clean’ anesthesia machine. Most literature reviews suggest a total intravenous technique (ITIVA).

Anesthetic drugs should be short-acting and rapidly metabolized. There is an increased sensitivity to non-depolarizing muscle relaxants. Opioids should be used sparingly.
Succinylcholine has been implicated in intraoperative cardiac arrests secondary to rhabdomyolysis and hyperkalemia, hence it is contraindicated. There has been much talk on the risks of hyperkalemic cardiac arrest with inhalational agents and there are continued reports of arrest, even during the recovery period. The most ‘at risk’ group is the patients under eight years of age, with still some muscle generation. This anesthetic-induced rhabdomyolysis (AIR) is unrelated to malignant hyperthermia (MH).

Drugs causing cardiac and respiratory depression need to be avoided or used with caution. Prone positioning and large blood losses may unmask cardiac dysfunction dramatically. Close postoperative monitoring is needed and ventilation is often indicated.

Becker’s Muscular Dystrophy (BMD)
This is the second most common form of dystrophy, occurring in 1:30,000 live male births. Dystrophin is abnormal, but still partly functional. The dystrophy is much milder with a slower onset. Symptoms start at around 11 years of age, often with a history of delayed motor milestones for walking, running and jumping. Later, they struggle with climbing stairs or getting up, fall frequently or walk on their toes. Life expectancy is virtually normal; however, they can develop severe dilated cardiomyopathy as adults.

Anesthetic Considerations
Similar to those in DMD
- Respiratory: Optimize chest infections, and assess lung function (deterioration may occur as a result of scoliosis, muscle weakness, and aspiration pneumonia)
- CK level elevation: Less marked than DMD: 50–100 times
- ECG/ECHO: Dilated cardiomyopathy often presents before skeletal muscle symptoms, and arrhythmias can also occur
- Postoperative ventilatory assistance may be necessary.

MYOTONIAS
Myotonic Dystrophy (Steinert’s Disease)
An autosomal dominant dystrophy, it is characterized by consistent contracture of muscle following stimulation. An abnormal nucleotide sequence on chromosome-19 causes prolonged stimulation of the actin-myosin complex due to a larger sodium current, causing delayed relaxation of contracted muscle. It may manifest in early childhood and is a multisystem disease.

Preoperative Assessment
CK levels: Do not correspond with the level of severity, often highest in infants.
Chest X-ray: Recurrent aspiration pneumonia due to bulbar weakness.
ECG: Cardiac conduction defects in 50–90% of cases, with 1st degree AV block and left anterior hemiblock.
ECHO: May reveal cardiac enlargement and mitral valve prolapse (30%). Significant left ventricular function impairment occurs later.
ABG: If SpO2 may be below 94%. Involvement of the intercostal muscles and diaphragm lead to poor cough and chronic alveolar hypoventilation, necessitating postoperative ventilation.

Anesthetic Considerations
- Sedatives preoperatively can alleviate fear and anxiety in an aggressive child prone to myotonic episodes, but has the risk of serious respiratory depression.
- TM joint contractures might limit mouth opening and make direct laryngoscopy difficult.
- Neither muscle relaxants nor regional anesthesia prevent or reverse myotonic contractions.
- Volatile anesthetics and steroids might attenuate contractions. Infiltration of muscles with LA can help.
- Depolarizing muscle relaxants and cholinesterase inhibitors exacerbate myotonia.
- Patients are at increased risk for perioperative aspiration.
- Succinylcholine has resulted in fatal rhabdomyolysis with hyperkalemia.
- Postoperative cardiac, respiratory and apneic complications can be dramatic.
- Prevent perioperative hypothermia and pain to limit myotonia.
Anesthesia in patients of myotonic dystrophy is associated with a higher than normal complication rate, even in mildly affected patients.
Complications could be precipitated by:
- Respiratory depressant effects of sedative analgesic/anesthetic agents
- Cardiovascular depressant effects
- Precipitation of myotonia by succinylcholine or neostigmine.
These patients are particularly vulnerable because of respiratory and bulbar muscle weakness, central and obstructive sleep apnea, cardiac conduction abnormalities and, sometimes, impaired left ventricular function. They remain at risk for several days after surgery.
Respiratory depressant effects of anesthetic, sedative or analgesic agents may cause gradual onset of respiratory failure and can go undetected in the surgical ward. Close monitoring even in late postoperative period is required, with appropriate intervention, such as NIV, as necessary.\(^2\)\(^5\)

**CONGENITAL MYOPATHIES**
These are characterized by early presentation of hereditary generalized hypotonia, small muscle mass and dysmorphic features. Patients present with weakness and delayed milestones. There is no muscle necrosis or degeneration and the disorders are non-progressive. The CK levels are normal or slightly increased. The best known of this group is central core disease (CCD).\(^2\)

**Central Core Disease**
It is an autosomal dominant, nonprogressive congenital myopathy with abnormalities of sarcoplasmic reticulum and T-tubules. Patients are hypotonic at birth, have proximal muscle weakness and get tired when feeding. It is characterized with delayed motor milestones secondary to generalized hypotonia and muscle weakness. Histopathological diagnosis is made on finding characteristic central cores on muscle biopsy. The ‘central core’ is an area of central clearing in the muscle with the loss of myofibrils, mitochondria, and glycogen. Central core disease has been consistently associated with malignant hyperthermia (MH). MH and CCD both involve abnormalities in genetic coding of RYR1 receptor on chromosome 19.\(^6\)

Evidence from the literature would suggest that any patient with an undefined myopathy that has not been confirmed by histopathology, genetic mutation analysis, or both is a possible candidate for central core disease and therefore of developing MH.

Due considerations for anesthetic management (awareness and avoidance of ‘trigger agents’) revolve around the cautious approach and safe conduct of anesthesia in view of the impending risks of MH in such patients.

**MITOCHONDRIAL AND METABOLIC MYOPATHIES**
This heterogeneous group of disorders is amongst the commonest cause of muscle weakness in children, with an incidence of 1 in 4000. Mitochondria are responsible for aerobic respiration and energy generation, in the form of ATP, via oxidative phosphorylation. Impaired electron transport chain (ETC) function results in decreased ATP production and an increased production of free radicals. The acidosis and excess free radicals further damage the mitochondria. Tissues that are dependent on high metabolic demand, like that of the skeletal muscle and central nervous system are most affected. It is now evident that mitochondrial defects are associated with variable dysfunction in virtually every organ system.\(^5\)\(^7\)

- Mitochondrial DNA mutations include:\(^2\)
  - MELAS: Mitochondrial encephalopathy, lactate acidosis and stroke like episodes
  - MERRF: Myoclonic epilepsy with ragged-red fibers syndrome.
- Severe forms of disorder can present in the neonatal period with profound weakness, liver and renal failure and substantial neurological impairment.
- The typically floppy infant, a poor feeder with small stature, displays developmental delay, hypotonia or hypoglycemia, with or without positive family history. Mild weakness can present later in adulthood.
- Metabolic derangements can include increased serum and CSF lactate and pyruvate.
- In lipid storage deficiencies, patients are susceptible to hypoglycemia, acidosis, general muscle weakness, rhabdomyolysis, and progressive cardiac insufficiency.
- Patients have exaggerated metabolic responses to prolonged fasting, fever and illness.
- ECG and ECHO might reveal cardiomyopathy or conduction deficits, and ventricular dilatation can compress the airway.\(^2\)\(^8\)

**Anesthetic Considerations**
- Preoperative
  - Evaluation of cardiac and respiratory status
  - Metabolic status: glucose, lactate, liver enzymes and serum creatinine.
- Overnight fasting could precipitate hypoglycemia, dehydration and mild metabolic acidosis. Preoperative intravenous infusion of glucose and electrolyte could avoid such a situation.
- Avoid lactate-containing fluids.
- There is an increased sensitivity to sedatives, barbiturates and propofol
- Avoid succinylcholine
- Variable sensitivity to nondepolarizing muscle relaxants could exist.
- Pain management is essential, as the response to pain may heighten the risk of lactic acidosis from depletion of energy stores and increased oxygen demand. Use opioids sparingly and consider NSAIDs, nonopioid analgesics and regional techniques. Prevent hypothermia.\(^2\)
Choice of Anesthetic Technique in Mitochondrial Disorders

Inhalation anesthesia has found favor over TIVA for known mitochondrial diseases. Recent data suggests, propofol, a lipid carrier with long-chain fatty acids, may adversely affect fatty acid oxidation and impair mitochondrial respiratory chain function. This may, in patients with mitochondrial disorders, lead to a clinical picture similar to propofol infusion syndrome (PRIS). Hence it has been suggested to avoid propofol in children with mitochondrial disease. If used, it should be combined with remifentanil or regional techniques, infused at doses lower than the recommended 4 mg/kg/h, and for short duration of time (< 48 hours). Other TIVA agents under investigation include ketamine, etomidate, and dexmedetomidine. Reports about increased sensitivity to these drugs are inconsistent. Sevoflurane has now been considered as the agent of choice in these patients.2,7,8

CHANNELOPATHIES

Channelopathies are characterized by disturbance in the transfer of ions across the sarcolemma. Channels are protein complexes which control the transfer by voltage or ligand gating.2,9

Familial Periodic Paralysis

Hyperkalemic

An autosomal dominant disorder, it is characterized by early onset, sometimes in infancy. Periodic paralysis is marked by brief episodes of flaccid weakness, which occur variably and resolve spontaneously. Respiratory and cranial muscles are typically spared. Attacks are precipitated by food high in potassium, cold, exercise and fasting, but not stress. Genetic mutation that affects sodium channels causes sustained sodium currents which don’t allow the formation of action potentials during these brief attacks.

Anesthetic considerations2,9
• ECG: Signs of hyperkalemia, also ectopic beats or paroxysmal tachycardia21
• Preoperative management consists of potassium-free dextrose containing solutions
• Avoid cold, hyperkalemia and carbohydrate depletion
• Succinylcholine is contraindicated.

Hypokalemic

An autosomal dominant disorder, it is the most common type of periodic paralysis with onset during adolescence.

It results from a mutation in a calcium channel. The attacks can be severe, resulting in respiratory compromise and cardiac disturbances. Triggers are strenuous exercise, high carbohydrate intake, low serum potassium, mental stress, cold, trauma and infection.2,9

Anesthetic considerations
• Patient should avoid overeating the day before surgery
• Adequate premedication should be administered to avoid stress
• Maintain normal serum potassium, glucose and acid-base status perioperatively
• Avoid intravenous fluids with dextrose and sodium
• Maintain normothermia.

Ligand-Gated Calcium Channelopathy

Malignant Hyperthermia

Inherited as an autosomal dominant trait, malignant hyperthermia (MH) is an occult myopathy unmasked by triggers such as depolarising muscle relaxants and inhalation anesthetics. Abnormalities in intracellular calcium homeostasis result in muscle rigidity, increased metabolism, rhabdomyolysis, hyperkalemia, acidosis and cardiac arrest, if untreated. The inheritance ranges from 1:15000 in children to 1:50000 in adults. Multiple mutations in the ryanodine receptor (RYR1) have been identified. When exposed to ‘triggering’ anesthetic agents, this abnormal calcium release causes sustained muscle contraction and rhabdomyolysis.

A definite association has been found with CCD, King-Denborough syndrome and Evans myopathy. Though there is an unclear link, but rhabdomyolysis has been reported in other neuromuscular disorders. In vitro caffeine-halothane contracture tests are recommended to assess MH susceptibility, particularly in cases with a strong family history.2,10

Anesthetic considerations:
• Avoid triggers in high-risk patients.
• Dantrolene if used early → Reduces mortality from 60% to 10%.10,11

Complications

Rhabdomyolysis

Depolarizing neuromuscular blockers can cause rhabdomyolysis in most patients with neuromuscular disorders. They could lead to massive changes in ion distribution with muscle contraction, swelling, and consequent damage which lead to rhabdomyolysis. Myotonia may also spontaneously induce rhabdomyolysis.
due to sustained muscle contraction. Volatile agents have been implicated with rhabdomyolysis in the past, due to their association with malignant hyperthermia, but it is now thought that this is a separate disease process to most neuromuscular disorders other than central core disease.

Rhabdomyolysis is characterized by:
- Metabolic acidosis
- Hyperkalemia
- Myoglobinuria
- Creatine kinase > 10,000 units/litre.

Treatment: The anesthesiologist should withhold the potentially causative drugs and correct life-threatening hyperkalemia. Aggressive volume resuscitation to remove myoglobin should commence to maintain a urine output of >1 mL/kg/h. The urine may be alkalinized using sodium bicarbonate. Administration of dantrolene (2.5 mg/kg to maximum 10 mg/kg) should be considered if hyperthermia is present.

Autonomic Dysfunction

Autonomic dysfunction can result in severe hypotension on induction and after regional anesthesia. Gastric dysmotility can lead to regurgitation and aspiration during general anesthesia. Sympathomimetic drugs should be available for use but doses may need to be reduced due to increased sensitivity of alpha and beta-receptors.

Myotonias

Myotonic contractures are caused by repeated action potentials leading to a permanent sodium influx or chloride efflux across the muscle membrane, rendering it hyperexcitable. Amongst the anesthetic agents, succinylcholine, anti-cholinesterases, and opioids could precipitate myotonic contractures. Environmental factors also play a part with alterations in temperature, acidosis, and shivering capable of triggering a contraction.

Myotonic contractures are not classically responsive to neuromuscular block, regional or peripheral nerve blockade. After correction of environmental and physiological conditions, drugs which block sodium channels, such as local anesthetics and antiarrhythmic agents may abort such episodes.

Cardiorespiratory Complications

Cardiomyopathies and conduction abnormalities can precipitate in the peri and postoperative period. Perioperative catecholamine release may further precipitate arrhythmias and potentiate cardiac failure. Thorough preoperative screening should aid the diagnosis of underlying abnormalities, but regardless of findings the patient should be treated as a high cardiac risk.

Respiratory failure remains the commonest cause of death in patients with neuromuscular disorders. The reasons could be:
- Bulbar muscle weakness leading to repeated aspirations
- Poor pharyngeal and respiratory muscle tone
- Obstructive sleep apnoea and progressive spinal deformities leading to a restrictive lung defect.

**LEARNING POINTS**
- Children with neuromuscular disorder may present for a variety of surgical procedures.
- A thorough preoperative history and assessment is necessary to evaluate functional capacity with a high suspicion of co-existing cardiac and respiratory disease, as well as metabolic disease.
- Preoperative optimization, fluid and electrolyte correction, and careful postoperative planning is vital.
- The dilemma remains with the anesthetic management of the undiagnosed floppy child.
- An educated guess as to the etiology of the neuromuscular disease is all that can be made followed by the diagnostic workup.
- It would be appropriate to choose a TIVA technique for the child with muscular dystrophy, and an inhaled agent for the child with mitochondrial myopathy.
- The risk of rhabdomyolysis or malignant hyperthermia when using inhalational anesthesia looms large at 1.09% and a total intravenous technique may be used in children with undiagnosed myopathy.
- Myotonia congenita, hypokalaemic periodic paralysis, DMD, central core myopathies, and King Denborough syndrome have all been associated with malignant hyperthermia in the past.
- It is now felt that there may be two distinct conditions - true malignant hyperthermia and a contracture-related rhabdomyolysis and acidosis. The latter has been described as ‘Anesthesia Induced Rhabdomyolysis’ (AIR), with regard to rhabdomyolysis and hyperkalemia seen after administration of succinylcholine and volatile agents in patients with DMD.
- A recent literature suggests that the risk of myotonic patients developing malignant hyperthermia is equivalent to that in general population, with the exception of hypokalemic periodic paralysis and central core disease.
- If using propofol as the primary agent, risks for propofol infusion syndrome and rhabdomyolysis are minimized by using moderate doses (<4 mg/kg) and for short duration (< 48 hours).

**REFERENCES**

INTRODUCTION

Obesity in children is on the rise. The risk of associated comorbid conditions increase with obesity. There is an increased risk of perioperative complications especially in obese children. Difficult mask ventilation and perioperative desaturation remains a potential life-threatening nightmare and challenge for the anesthesiologist in this group of patients. Further, obesity can impact the pharmacokinetics of most anesthetic drugs. This has important implications on how to estimate the optimal drug dose. A conservative approach is advocated when dosing drugs in overweight children, especially when using drugs with a narrow therapeutic index.1-5

DESCRIPTION: CHILDHOOD OBESITY

Unlike in adults, where a BMI > 25 kg/m² defines overweight and >30 kg/m² obesity, in children, median BMI varies with age and sex. WHO defines childhood overweight and obesity according to standard deviations (BMI Z-scores) from the mean BMI. The American definition of childhood obesity describes a BMI ≥ 85th percentile as overweight and a BMI ≥ 95th percentile as obesity.6-8

PREVALENCE

Worldwide statistics indicate that 20 million children under the age of 5 years are obese. More than 90% of childhood obesity cases are primary, caused by excessive calorie consumption. The remaining cases are secondary to underlying diseases like endocrine disorders, neurological dysfunction, and syndromes (e.g. Prader Willi).9,10

PATHOPHYSIOLOGY, CLINICAL PRESENTATION, ANESTHETIC ISSUES AND PHARMACOKINETICS OF ANESTHETIC AGENTS

There appears to be a correlation between pediatric obesity and the reduction in lung functions—FRC, ERV, FEV1 and DLCO (Diffusing Capacity of the Lung for Carbon Monoxide) which result in high closing volume. This is followed by atelectasis, air trapping, and intrapulmonary right to left shunting with possible hypoxemia. Supine position of the patient potentiates these changes, when the abdominal pressure on the diaphragm is highest.11,12

Obesity may induce bronchial hyperreactivity. Approximately 30% of obese children between 8–18 years have asthma that shows a rise in both incidence and severity with increasing BMI.13 Obese children are more prone to URTI in comparison to those with normal weight.14

Approximately 13–59% of obese children suffer from obstructive sleep apnea (OSA), compared to 1–2% of normal weight children.15 Children with OSA are at increased risk of postoperative respiratory depression, airway obstruction and diminished ventilatory response to CO₂ compared to other children.16-17 Postoperative management of obese children with OSA includes positioning, use of a nasal airway and in severe cases use of continuous positive airway pressure (CPAP) and prolonged postoperative (overnight) monitoring.16

The cardiac output remains high in obese children due to the increased volume of circulation and stroke volume.16 20–30% of obese children have hypertension.3 The cardiac risk factors: LVH, hypercholesterolemia, and hyperlipidemia are common in obese children.19 A detailed history of effort tolerance in obese children should be obtained to determine the cardiopulmonary status.

Metabolic syndrome is known to occur in 40–50% of obese children.20 They are consequently at a higher risk of developing type 2 diabetes mellitus.21 These children should therefore be tested preoperatively for fasting blood glucose level.

Gastroesophageal reflux (GER) in obese children has been described with symptoms in as much as 20% of severely obese children compared to 2% of normal weight children.22

Comorbidities that may be associated with childhood obesity are:

- Asthma
- Obstructive sleep apnea
- Hypertension
- Type II diabetes
- Gastroesophageal reflux
- Low self esteem.

Pharmacokinetics of most anesthetics are altered by obesity.5 Extrapolations must be made from studies on obese adults, because of a lack of pharmacokinetic studies in obese children. Optimal doses for induction and maintenance of anesthesia are based on the patients total body weight (TBW), ideal body weight (IBW), and lean body weight (LBW).1

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IBW = BMI at the 50th percentile for the child’s age × [height (m)]^2
LBW = IBW + 0.3 × (TBW – IBW).

Compared to children with normal weight, LBW is higher in obese children as 20–40% of the excessive weight is due to an increase in muscles and bones. 23

**ANESTHESIA TECHNIQUE**

The preanesthetic checkup in obese children needs to include: height, weight, BMI, airway assessment. HR, BP and preoperative pulse oximetry should be recorded, and fasting blood glucose be obtained. Screening for obstructive sleep apnea may indicate the need for overnight oximetry, formal sleep studies, and other respiratory function tests, including arterial blood gas analysis. If cardiac involvement is suspected, an ECG and 2D ECHO should be obtained. 1 Additionally, assessment for gastroesophageal reflux with the commencement of suitable medication should be considered.

Individualizing anesthetic drug doses in obese patients maybe difficult. 24 Considering total body weight for dose calculation of many of the anesthetic agents may lead to overdose and subsequent adverse physiological sequelae. 1 Hence, ideal body weight or even lean body weight may be preferred for dose calculation. Succinylcholine is an exception, however, and should be administered taking total body weight into consideration despite its hydrophilicity. The reason for this is the increased pseudocholinesterase activity in this population (Table 1). 25

Intravenous (IV) induction of anesthesia is preferred in obese children, however, venous access may be difficult for induction due to increased subcutaneous fat deposits. Prolonged and repeated cannulation attempts are difficult and distressing, even with distraction techniques and topical anesthesia. These children may co-operate for a short period, so the inhalation induction can be considered.

Sevoflurane, amongst the volatile agents, has a higher hemodynamic stability, is less irritant to the airway when compared to desflurane and can be safely used for induction. 26,27 Desflurane has lower blood-lipid solubility with faster return of protective airway reflexes and perhaps more rapid recovery profile than sevoflurane in obese patients. 28,29 Isoflurane has a high blood-lipid solubility, which markedly increases recovery time in obese individuals compared to sevoflurane and desflurane. 20,29

Succinylcholine happens to be the only anesthetic agent whose pharmacokinetics have been studied in obese children. 25 All other dose suggestions are based on studies on obese adults and normal weight children. There is no data on optimal dosage of midazolam and clonidine in obese children. It has been suggested that IBW be referred to for optimal doses of either, to avoid respiratory depression. 1 The non-depolarizing muscle relaxants should be given at an ideal body weight dose with careful reversal of neuromuscular block at the end of the procedure. Dosing of IV paracetamol at total body weight may result in overdose and subsequent liver toxicity; therefore, it is safer to dose same using ideal body weight.

**Postoperative Care**

Length of stay in PACU may be significantly prolonged and there may be an increased risk of postoperative upper airway obstruction in obese children. 4,31 Nursing the obese child in propped up position reduces the incidence of atelectasis and improves oxygenation.

Supplementation with regional analgesia for postoperative pain relief seems rational than administration of opioids (risk of respiratory depression). 32 Otherwise, nurse-controlled or patient-controlled analgesia (NCA or PCA) with dosage according to IBW and monitoring of respiration can be used after major surgery.

Drug clearance in obese children might be altered. 5 Monitoring the depth of anesthesia could be helpful in titrating anesthetic doses in obese children.

A low-risk of postoperative respiratory depression and airway compromise makes both peripheral nerve blocks and neuraxial blockade seemingly attractive for obese children. 32

**POSSIBLE RISKS AND COMPLICATIONS (PERIOPERATIVE)**

Few studies have evaluated the airway management and ventilation of obese children especially those younger...
than 2 years. In such studies, children with normal weight were not found to be different from overweight children with respect to the adverse airway incidences, while obese children had a significantly higher risk of airway-related complications compared to the other groups. Obesity, age <10 years, OSA, and procedures involving the airway were identified as risk factors for perioperative respiratory complications.3,34

Hypoxemia precipitates faster in children than in adults due to a higher consumption but low reserves of oxygen in children. Obese children are more prone to intraoperative desaturation than normal weight children. Obese children are not at an increased risk of pulmonary aspiration intraoperatively when compared to normal weight children. There is no evidence that rapid sequence induction (RSI) imparts safety against aspiration in all obese children.

Upper airway collapse during sedation and general anesthesia remains a perpetual risk in children. Difficult mask ventilation compounds the problem further, which has been found to be higher in proportion in obese than in normal weight children. Incidence of difficult laryngoscopy in obese and children with normal weight seem to be the same.

LEARNING POINTS

- Anesthesia in obese children poses unique challenges.
- Managing the obese child is becoming a common scenario for the anesthesiologist as the problem reaches epidemic proportions.
- Anesthesia at any stage may be associated with higher risk (airway obstruction, intraoperative oxygen desaturation, difficult mask ventilation) in these children, some of which may be life threatening.
- Accompanying comorbidities in the obese child further add up to the anesthetic problems and require due expertise for safe and successful conduct of anesthesia.
- There is no evidence to support the recommendation of longer fasting hours, RSI or the use of an endotracheal tube instead of a LMA in the obese child scheduled for anesthesia.

REFERENCES

INTRODUCTION

Obstructive sleep apnea (OSA), a sleep-related breathing disorder, is characterized by repeated episodes of apnea and hypopnea during sleep. Apnea is defined as complete cessation of airflow for more than 10 seconds, and hypopnea as airflow reduction more than 50% for more than 10 seconds. This repetitive upper airway obstruction often results in oxygen desaturation and arousal from sleep.1 Sleep disordered breathing (SDB) in children comprises of disorders ranging in severity from primary snoring (seen in 20% of children), upper airways resistance syndrome, obstructive hypopnea to obstructive sleep apnea (seen in 2% of children).2-4

Children with OSA can have significant associated morbidity. The airway obstruction due to OSA can cause abnormal gas exchange leading to hypoxemia, hypercapnia, sleep fragmentation with their associated physiological and behavioral consequences. The degrees of hypercapnia, hypoxemia, and upper airway airflow reduction are the primary factors determining the severity of OSA. The mainstay of treatment of pediatric OSA is adenotonsillectomy. Anesthesia for same can be challenging and a safe and successful anesthetic practice revolves around good and ideal airway management. A safe anesthetic management of a child with OSA for the proposed surgery requires an anesthetic technique tailored to the underlying aetiology and severity of OSA and the surgical procedure.5

ETIOLOGY OF SLEEP DISORDERED BREATHING IN CHILDREN

- Adenotonsillar hypertrophy → Most common etiology
  → Significant in 2–6 year age group as adenoids and tonsils enlarge but absolute size of airway still remains small.
- Chromosomal abnormalities → Downs syndrome (mild mid-face hypoplasia, large tongue, generalized hypotonia).
- Craniofacial abnormalities → Aperts or Crouzons syndrome (severe mid-face hypoplasia) → Treacher-Collins or Pierre Robin sequence (micrognathia).
- Cerebral Palsy (Hypotonia).
- Sickle cell disease (Lymphoid hyperplasia)
- Papillomatosis.
- Cystic hygroma (Foreign body).
- Obesity is becoming an increasingly common cause of OSA in older children.3

PATHOPHYSIOLOGY

The definite pathophysiologic mechanism of pediatric OSA is still not well understood. Obstructive cycling, increased respiratory effort, flow limitation, tachypnea, and/or gas exchange abnormalities result in variable abnormal respiratory effort and disturbance of sleep homeostasis. Imbalance in neuromuscular activation, ventilatory control, and arousal threshold cause the instability of the upper airway.

Hypertrophy of the tonsils and adenoid are frequently encountered in childhood OSA, and adenotonsillectomy reduces adverse respiratory events and the risks of related morbidities.6 Residual morbidity after adenotonsillectomy, if any, indicates that other factors needed to be implicated for OSA. For example, obesity increases airflow resistance and pharyngeal collapsibility.7 Craniofacial deformities increase upper airway resistance which may be decreased by craniofacial reconstruction.8-10

Accordingly, OSA is the consequence of one or more disorders with comorbid diseases that mandate further evaluation and management.

CLINICAL PRESENTATION

The distinctive symptoms of OSA in children are remarkably scarce and usually require a high level of suspicion and require systematic implementation of explorative screening questions to enable their detection.11

OSA in children shows distinctive differences from OSA in adults. The typical clinical picture of severe OSA (Type I OSA) is a young child where the parents are concerned about the child’s breathing at night, the child has noisy breathing associated with ‘snorts’ after periods of apnea, and the parents have to shake the child or reposition them during sleep. The child is not gaining weight despite good appetite, may be hyperactive with behavioral difficulties by day, habitually mouth breathes and has a blocked nose. Cardiovascular complications are of particular interest to the anesthesiologist. Severe OSA with repeated nocturnal hypoxia is associated with pulmonary hypertension which if severe and long-standing may result in right heart failure. Clinical examination reveals the cause of OSA (e.g. signs of adenotonsillar hypertrophy, narrow midface) and signs of cardiomegaly that may be confirmed on CXR. The CXR may also demonstrate prominent central pulmonary arteries. An ECG reveals signs of right ventricular
hypertrophy (right axis deviation, peaked P waves, tall R waves in lead V1). Echocardiography confirms the diagnosis of pulmonary hypertension, right ventricular hypertrophy ± dilatation. These children are at high risk during anesthesia (see below) but signs and symptoms resolve completely within months of treating the OSA.12 It has recently been recognized that there is an emerging subset of obese adolescents with OSA (Type II OSA) who have similar clinical features to adults with OSA.2

**DIAGNOSIS**

Overnight sleep study in a sleep laboratory (Polysomnography) has been the ‘gold standard’ for diagnosis of SDB in children. This includes measures of nasal airflow, chest wall and abdominal wall movement, a continuous monitor of expired CO₂, and oxygen saturation. This, however, is expensive, not widely available and is only indicated where there is some diagnostic uncertainty.

‘Mini sleep studies’ using overnight pulse oximetry and heart rate monitoring give an excellent measure of severity of OSA in children, and may be performed on the ward or more usefully at home.

**Clinical diagnosis of OSA:** Some key questions need to be asked in the clinical history.13

- Does the child snore at night?
  - Do they stop breathing?
  - Do they sweat?
  - Are they restless?
- Does the child breathe through the mouth when awake?
- Is there a family history of OSA?
- Does the child have behavioral problems?

**OSA SEVERITY SCORING**

<table>
<thead>
<tr>
<th></th>
<th>Mild OSA</th>
<th>Moderate OSA</th>
<th>Severe OSA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical signs</td>
<td>Mouth breathing, slight increased respiratory effort, ± snoring, sleeps quietly at night</td>
<td>Mouth breathing with respiratory effort, ± snoring or ‘snorting’, restless sleep</td>
<td>Mouth breathing, markedly increased respiratory effort, loud snoring and ‘snorting’, disrupted sleep</td>
</tr>
<tr>
<td>Sleep study correlate</td>
<td>SpO₂ in normal limits, ± minor dips</td>
<td>Normal baseline SpO₂, repeated desaturation to mid 80s</td>
<td>Frequent prolonged episodes of paradoxical breathing, frequent prolonged desaturation</td>
</tr>
</tbody>
</table>

**SURGICAL PROCEDURE**

Although OSA has multiple etiologies in children, with the diagnosis being established and its severity assessed, adenotonsillectomy is usually the first line of treatment.19 It should be implemented along with weight normalization in obese children.

In children who manifest residual OSA after adenotonsillectomy or in those who present with minimally enlarged upper airway or lymphadenoid tissues or who opt not to undergo surgery, positive airway pressure, in the form of CPAP or BiPAP (bilevel positive airway pressure), has been recommended.15 Although positive airway pressure can undoubtedly be a highly effective treatment,16,17 adherence can be particularly challenging in children, particularly those with behavioral problems or developmental delays.

In selected patient populations, some orthodontic procedures, such as rapid maxillary expansion, have been proposed as efficacious.18-20 Procedures such as tongue-base suspension and uvulopalatopharyngoplasty have also been studied in children with cerebral palsy and OSA.21

In complex or persistent cases of OSA, sleep endoscopy is a technique that enables the exact level of obstruction in the child to be identified, thus facilitating site-specific surgical therapy.22,23

**ANESTHETIC CONSIDERATIONS**

**Preoperative Assessment**

Screening of patients should begin with the question to the parents: Does your child snore? A history of nightly snoring is a sensitive (91%) but not completely specific (75%) marker of OSA.14 If a child regularly snores, additional focused questions may help to clinically identify those with OSA especially in patients with known OSA risk.

Presenting signs are snoring, apnea, failure to thrive, developmental delay, and recurrent respiratory infections.25 It is not clear if infants are in a subset of OSA with more severe disease, more comorbidities, or genetic predisposition.

The physical examination should include an airway assessment: nasal anatomy, ability to breathe through the nose, presence of elongated facies, oral aperture size, mandibular size, intermaxillary distance, thyromental distance, tonsillar size, tongue volume, body habitus, and Mallampati score.26 The examination of the patient should also include an assessment of muscle tone, handling of oral secretions, and observation of facial malformations. When history, physical examination, and sleep laboratory data are combined, risk assessment is more accurate.27
Cardiac evaluation though not indicated for most of the children posted for adenotonsillectomy, should be carried out in any child with signs of right ventricular dysfunction, systemic hypertension, or multiple episodes of desaturation below 70%. ECG and chest radiograph are not sensitive evaluation tools; echocardiography is recommended. Routine ABG is not recommended, however the metabolic parameters can identify a compensatory metabolic alkalosis in response to chronic hypercarbia, and hemoglobin estimation may identify the patient with severe chronic hypoxemia.

The CPAP in the preoperative period is known to reduce the postoperative complication rate and increase airway patency in adult OSA patients. It may be beneficial for certain pediatric patients. Effective CPAP/BiPAP therapy may improve pulmonary hypertension and reduce the patient’s surgical risks.

**Intraoperative Management**

No specific anesthetic technique is preferred for adenotonsillectomy.

Sedatives/anesthetics alter the CO₂ response curve, hence OSA patients may be at higher risk of sedative/anesthetic induced respiratory complications. Patients with OSA rescue themselves during obstructive episodes by arousal from sleep, but sedatives or residual anesthetics may make it impossible for patients to arouse themselves during obstructive episodes. Hence, short-acting anesthetics are preferred.

Sedatives used before the induction of general anesthesia may delay emergence in patients, especially for short cases. Patients with OSA can receive sedatives but require monitoring until complete recovery. There is no preferred anesthetic induction strategy in OSA, some children with OSA may require approaches different from those used with normal patients. Children without maxillofacial malformations are often easily mask-ventilated once an oral airway is placed, and endotracheal intubation should not pose a problem. IV induction can be used to rapidly induce a deep plane of anesthesia ready for airway instrumentation. This technique may be preferable for patients with very severe OSA. Children with OSA in addition to craniofacial abnormalities or other significant airway disorders must be evaluated for potential difficult intubation. Induction of anesthesia with volatile anesthetics results in airway collapse from relaxation of the genioglossus muscle, thus placing the OSA patient at high-risk for airway obstruction. Positioning in an upright or lateral position, use of a jaw thrust maneuver, delivery of positive pressure by face mask and placement of an oral airway may aid in relieving the obstruction.

Upon conclusion of the procedure, patients should be awake and have adequate strength to maintain the upper airway before tracheal extubation. Before extubation, it may be advisable sometimes to place nasal airways in patients with severe OSA.

The child who continues to have significant obstructive episodes after extubation can be positioned in the lateral decubitus or prone position to help relieve the obstruction. CPAP or BiPAP can be used to assist ventilation and relieve airway collapse. Reintubation may be required in an occasional patient.

Postoperative vomiting and pain after adenotonsillectomy add up to the morbidity. Anesthetic techniques using sevoflurane and propofol with muscle relaxant and fentanyl have shown no statistical difference in postoperative vomiting. Steroids (Dexamethasone) have been shown to improve postoperative oral intake and reduce pain and vomiting. Ondansetron (0.1 mg/kg) has been found superior to metoclopramide (0.5 mg/kg) in countering PONV.

**POSTOPERATIVE COMPLICATIONS**

Chronic hypoxemia in children with OSA renders them more susceptible to the respiratory depressant effects of opioids in the postoperative period. Children with OSA were seen to have a higher incidence of apnea after administration of 0.5 μg/kg of fentanyl, and diminished minute ventilation during spontaneous ventilation under general anesthesia with inhaled anesthetics, when compared with non-OSA patients. If high doses of opioids are required for an OSA patient after a surgical procedure, intensive cardiopulmonary monitoring must be carried out. When possible, regional anesthesia and/or analgesia should be used. Postoperative intensive care unit admission is reserved for very severe OSA, very young children and those with comorbidities.

**POSTOPERATIVE PAIN RELIEF**

For short procedures, one approach is to minimize opioids intraoperatively and then titrate them to effect when the child is awake, extubated, and in a monitored setting. Less painful procedures may only require non-opioid analgesics, such as acetaminophen or nonsteroidal antiinflammatory drugs (NSAIDs). The use of NSAIDs in posttonsillectomy patients has been avoided because of reports of associated postoperative bleeding. The bulk of evidence supports the use of nonaspirin NSAIDs for postoperative analgesia.
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PHEOCHROMOCYTOMA

INTRODUCTION

Rare tumors in the pediatric age group, pheochromocytomas arise from chromaffin cells of adrenal medulla or extra-adrenal paraganglionic tissue which are responsible for synthesis, metabolism, and secretion of catecholamines. They are more frequently familial, extra-adrenal, bilateral, and multifocal compared to adults. They have been commonly associated with heritable conditions such as multiple endocrine neoplasia (MEN) types IIa and IIb, von Hippel-Lindau and hereditary PCC/PGL syndromes. Approximately 40% of pheochromocytomas in children have a hereditary basis, hence proper genetic testing should be performed, with appropriate implications for surgery, future follow-up and treatment options.

PATHOPHYSIOLOGY

With an incidence of 2/million/year, 0.8–1.7% of hypertensive children can be detected to be suffering from pheochromocytoma. Children around 11–12 years of age are usually affected with a male preponderance (2:1) and most are familial. Majority being benign, extra-adrenal (~40%) and bilateral (~25%) tumors are more common.

Pheochromocytoma releases large amounts of:
- Catecholamines (CCA): Adrenaline, noradrenaline and dopamine
- Various peptides
- Ectopic hormones: Enkephalins, somatostatin, calcitonin, oxytocin, vasopressin, insulin and adrenocorticotropic hormones.

Adrenal pheochromocytoma secretes adrenaline and noradrenaline, while extra-adrenal tumors secrete only noradrenaline. Generally, the secretion of noradrenaline is greater than adrenaline. Less than 10% are malignant. During embryonic development the chromaffin cells settle mainly near the sympathetic ganglia, vagus nerve, paraganglia and carotid arteries but some chromaffin tissue may be present in the bladder wall, prostate, rectum, gonads, renal and hepatic hilus. Thus, pheochromocytomas can arise at any of these sites. Excessive secretion of catecholamines (norepinephrine, epinephrine, or dopamine) into the circulation results in the variety of symptoms in patients.

Biosynthesis and Metabolism of Catecholamines

\[
\begin{align*}
\text{Phenylalanine} & \rightarrow \text{Tyrosine} \\
\text{Tyrosine} & \rightarrow \text{DOPA} \\
\text{DOPA} & \rightarrow \text{DOPAMINE} \\
\text{DOPAMINE} & \rightarrow \text{NOREPINEPHRINE} \rightarrow \text{EPINEPHRINE} \\
\text{Normetanephrine} & \rightarrow \text{Vanillylmandelic} \rightarrow \text{Metanephrine Acid}
\end{align*}
\]

The actions of catecholamines are mediated by the \( \alpha \) adrenergic and the \( \beta \) adrenergic receptors. \( \alpha_1 \) receptors cause vascular constriction while \( \alpha_2 \) receptors mediate the presynaptic feedback inhibition of norepinephrine release and decrease insulin secretion. \( \beta_1 \) receptors increase cardiac rate and contractility and \( \beta_2 \) receptors lead to arteriolar and venous dilation and relaxation of tracheobronchial smooth muscle.

The clinical manifestation of pheochromocytoma is determined by the profile of catecholamine secretion. In the norepinephrine secreting tumor, clinical picture is associated with hypertension whereas if epinephrine is the major secreted catecholamine, tachycardia, tachydysrhythmias and hypotension often result. Increased dopamine secretion by a pheochromocytoma may suggest malignancy.

CLINICAL PRESENTATION

Clinical presentation in pheochromocytoma is heralded by excessive secretion of catecholamines. The most characteristic feature is hypertension. 70–90% present with HTN, usually sustained
- 70–90% present with HTN, usually sustained
- Sweating, visual problems, weight loss, nausea/vomiting, and polyuria/polydipsia are common
- Classic triad of paroxysmal sweating, palpitations, and headaches are less common.

Occasionally, children with sustained hypertension also have paroxysmal episodes, precipitated by excitement.

\[\text{Chap-33.indd} \quad 440\]
or a particular physical activity, such as bending over or lifting a heavy object. Convulsions secondary to hypertensive encephalopathy may occur.

Wide fluctuations in blood pressure are characteristic, and marked increases may be followed by hypotension and syncope.

Pallor is usually present because of the intense alpha-receptor-mediated peripheral vasoconstriction, which causes cool, moist hands and feet, and facial pallor.

Palpitations, mediated by beta-1 receptors, reflect increased cardiac output and heart rate.

Hyperthermia or flushing secondary to decreased heat loss and increased metabolism leads to reflex sweating.

Poor weight gain or severe cachexia may develop because of hypermetabolism. The child may have a good appetite but, because of hypermetabolism, does not gain weight.

Polyuria and polydipsia may result from increased glycolysis and alpha-receptor-mediated inhibition of insulin release. This insulin inhibition causes an increase in blood sugar levels and glucose intolerance. As a result, patients may present with hyperglycemia or glucose intolerance, most commonly during paroxysms.

Hypercalcemia is uncommon but well-recognized complication that may reflect associated hyperparathyroidism, particularly in familial cases.

Affected children are often emotionally labile and have an anxious expression. Occasionally, these children are labeled hyperactive with an attention deficit disorder.

**Preoperative Workup**

The anesthesiologist should take relevant history, assess the severity of hypertension, cardiac manifestations and look for any end-organ damage especially catecholamine induced cardiomyopathy and cardiac failure, which are associated with a high mortality.6

The baseline investigations pertaining to individual organ system functions should be sought. A baseline full blood count and haematocrit followed by serial monitoring provides an assessment of the adequacy of volume expansion when alpha adrenergic blockade has been started.

Serum electrolytes, blood urea, and serum creatinine provide a useful insight into the metabolic and renal function status. If hypercalcaemia is present, the presence of MEN type II should be suspected.

Blood sugar estimation can determine the dose of insulin requirement as few patients have uncontrolled hyperglycemia pre-operatively.

Chest X-ray and spirometry are valuable adjuncts to determine the structural and dynamic aspects of lungs especially in the patients in whom parenchyma of lungs and right sided involvement of heart is anticipated.

ECG may be supplementary in diagnosing arrhythmias, ischemia, cardiomyopathy, but echocardiography is a must to evaluate the status of cardiac tissue.

The specific diagnostic current protocol recommends patients to be tested for fractionated elevations of Catecholamine breakdown products - metanephrine and normetanephrine - in plasma, urine or both.22 Although 24 hours measurements are most accurate, overnight measurement is usually adequate for screening purposes.

(Normal 24 hours urinary: Free catecholamines< 100 mg; VMA< 7 mg, Metanephrine< 1.3 mg).

On confirmation of diagnosis of pheochromocytoma by chemical analysis, the tumor should be located. Although USG is the usual method for evaluating the abdominal masses in children, CT identifies almost 95% of Pheochromocytomas. MRI is more accurate than CT or USG in the diagnosis of pheochromocytoma and other adrenal tumors, hence, is considered the method of choice for definite evaluation of adrenal neoplasms in children.15 Radioisotope studies with 131 labelled metiodobenzylguanidine (MIBG) have also been useful in localizing abnormal medullary tissue in Pheochromocytoma.14

**Preoperative Medical Management**

Preoperative medical preparation of the patient to block the effects of released catecholamines is the prerequisite to definitive surgery. Prior to surgery it is imperative to control arterial pressure, heart rate and arrhythmias and to restore the blood volume to normal.

Phenoxybenzamine and phentolamine block the alpha-adrenergic receptors of epinephrine and norepinephrine. To counter the potential danger of hypertensive paroxysms, the patient should be started on alpha-adrenergic blocking agents as soon as the diagnosis of Pheochromocytoma is made and at least 3 to 7 days before surgery.15 Normalization of metanephrine levels are the most reliable predictors and the best indication that the patient is adequately controlled.

Therapy with phentolamine 1 to 2 mg/kg/day is recommended in four divided doses which can be increased until blood pressure returns to normal. Phentolamine can be used for rapid alpha adrenergic blockade, whereas phenoxybenzamine therapy (0.2–1 mg/kg/day in divided doses) for 1 to 2 weeks can be used when surgery is not urgent.16
β-adrenergic blockers have been used to prepare patients for surgery or to control tachycardia and prevent arrhythmias resulting from α adrenergic blockade during surgery. β blockade should never be instituted until α adrenergic blockade is fully established as unopposed α stimulation may lead to severe hypertension. Nitroprusside has occasionally been used before surgery in patients who have crescendo symptoms or who have become refractory to oral and intravenous α adrenergic blocking agents.

As an alternate preoperative treatment regimen, α methylparatyrosine (AMPT) - 250 mg 3 to 4 times per day, gradually increased to a total dose of 1.5 to 4.0 g/day, an inhibitor of tyrosine hydroxylase has been recommended to decrease catecholamine synthesis. It has not been tested in children less than 12 years of age.

Prolonged medical therapy for pheochromocytoma is not recommended. Replacement of intravascular volume is often required as alpha blockade is achieved because, patients with Pheochromocytoma tend to be hypovolaemic, with an average 15% reduction in the normal plasma volume. Carefully monitored preoperative re-expansion of the vascular system helps to minimize fluctuation in blood pressure and intractable cardiac arrhythmias during surgery.

Nutritional supplements may also be necessary as the patients may be catabolic as a result of frequently elevated BMR.

**SURGICAL RESECTION OF THE TUMOR**

It is the only curative procedure.

**Surgical Procedures**

- Open Adrenalectomy– Large tumors
- Laparoscopic Adrenalectomy–Anterior transperitoneal/Posterior retroperitoneal
- Cortical Sparing – VonHippel-Lindau Syndrome (VHL, MEN - II, etc.)

**ANESTHESIA GOALS**

General anesthesia for excision of pheochromocytoma may be divided into two stages. The 1st is characterized by efforts to keep the systemic blood pressure down while the tumor is isolated and its blood supply and drainage are ligated. The 2nd involves efforts to keep the systemic blood pressure elevated thereafter. Main goals of anesthesia are:

- Normalization of blood pressure, heart rate and functions of other organs
- Restoration of volume depletion
- Prevent surgery induced catecholamine storms.

**ANESTHESIA TECHNIQUE**

It is a good practice for the anesthesiologist to accompany the patient from the preoperative ward to the operating room with administration of sedation as necessary. Combined epidural and general anesthesia is the most preferred anesthesia technique. Preoperative benzodiazepine and reassurance reduces anxiety and pressure fluctuations. Various anesthetic techniques have been employed successfully. ECG, NIBP, and SPO₂, are the mandatory monitors attached before induction of anesthesia. Invasive arterial pressure and central venous pressure monitoring are essential and are ideally employed after anesthesia induction in a pediatric patient. Temperature and urine output should also be monitored.

Induction of general anesthesia is particularly critical because inadequate sedation may produce severe hypertension, whereas excessive α blockade with inadequate blood volume/re-expansion may result in severe hypotension. Thiopentone or propofol can be used for induction of anesthesia along with fentanyl. Both have a good hemodynamic profile. Nitrous oxide has no contraindications and has been used routinely. Sevofluane and isoflurane are commonly used while desflurane is best avoided due to its ability to cause significant sympathetic stimulation. Halothane has the ability to sensitize the myocardium to the arrhythmogenic effects of catecholamines hence is no longer used for anesthesia for pheochromocytoma resection. Vecuronium is the preferred muscle relaxant for cardiovascular stability but atracurium and rocuronium have been used without any untoward effect.

Intraoperative hypertensive spikes during tumor manipulation can be impeded by deepening anesthesia and infusions of SNP (0.5–5 μg/kg/min), NTG (0.5–1.5 μg/kg/min), phenolamine (as an infusion or in incremental doses of 1 to 2 mg to control acute hypertensive crises because it acts fast and has a short half-life), esmolol (Bolus 500 μg/kg/min, Infusion 50 to 200 μg/kg/min), dexmedetomidine (1 μg/kg over 10 min followed by 0.5 μg/kg/h), or magnesium sulphate (loading dose of 40–60 mg/kg followed by an infusion of 1–2 g/h). A single drug or combination regimens might be employed depending on severity, familiarity, and availability.

Epidural infusion of bupivacaine 0.1–0.125% with fentanyl 1–2 μg/mL at the rate of 5–10 mL/h (0.2–0.3 mL/kg/h) can be used for postoperative analgesia.
POSSIBLE COMPLICATIONS

Post-resection, patients should be monitored for complications of hypotension, hypoglycemia, and persistent hypertension. Hypotension can be severe, and might need phenylephrine, adrenaline/noradrenaline, or vasopressin infusions, especially in patients receiving phenoxybenzamine. Hypertension might persist for few days to elevate circulating catecholamines. Hypoglycemia might ensue after removal of tumor, secondary to increase in insulin levels due to cessation of pancreatic beta cell suppression. Decision for post-operative extubation or elective ventilation depends on hemodynamic stability and other vital parameters. Post-operative ICU care is necessary for close monitoring of the complications. In the event of persistent hypertension beyond 7–10 days, presence of residual tumor or extra adrenal tumor should be considered. Biochemical assay and imaging studies are repeated for confirmation and further management.

LEARNING POINTS

- Anesthesia for pheochromocytoma is challenging and requires multidisciplinary approach for optimal care and successful outcome.
- Proper preoperative pharmacological preparation has greatly improved perioperative outcome.
- Modern anesthetic drugs combined with advanced monitoring contribute to intraoperative stability, though, it is difficult to extract any consistent recommendation from the literature regarding intraoperative anesthetic management of pheochromocytoma resection.
- Despite certain advances to come, it is likely that a specific intraoperative anaesthetic protocol for pheochromocytoma will never exist across the institutions. A diligent, attentive anesthesiologist and a well prepared patient is almost certainly more important than which drug or drug combination is selected.

REFERENCES

INTRODUCTION

Pediatric vascular anomalies (vascular ‘birthmarks’) are abnormal formations or growths within the vascular system. Subcategorized into vascular tumors and malformations, each anomaly is characterized by specific morphology, pathophysiology, clinical behavior, and management approach. Hemangiomas are the most common vascular tumors. Lymphatic, capillary, venous, and arteriovenous malformations make up the majority of vascular malformations. With a morbidity of about 2.5%, most of the lesions occur in oral and maxillofacial regions. In brief, hemangiomas are vascular tumors that are rarely apparent at birth, grow rapidly during the first 6 months of life, involute with time and do not necessarily infiltrate but can sometimes be destructive. Vascular malformations, in contrast to hemangiomas, are present at birth, slow growing, infiltrative, and destructive. Almost all vascular malformations and nearly 40% of hemangiomas eventually require intervention. Approximately two-thirds are predominantly venous.

PATHOPHYSIOLOGY

Diffuse or localized defects in embryonic development have been considered to lead to vascular malformations. They have been traditionally attributed to sporadic mutations. However, recent evidence points to a possible familial hereditary component.

Unlike hemangiomas, vascular malformations do not have a growth cycle and subsequent spontaneous regression but rather persist throughout a person's lifetime, growing slowly, sometimes in response to injury, changes in blood or lymph pressure, infections, hormonal changes, etc. Characteristically, these lesions progressively produce ectasia of vascular structures, increasing the diameter of vessels without increasing their number. Expansion is therefore by hypertrophy but not by hyperplasia, as is the case for hemangiomas. Pathogenesis of vascular malformations hints at their formation and progression being closely related to angiogenesis.

CLASSIFICATION

The International Society for the Study of Vascular Anomalies Classification

Vascular Tumors

- Hemangioma at infancy.
- Congenital hemangioma
  - Rapidly involuting congenital hemangioma
  - Noninvoluting congenital hemangioma
  - Tufted angioma
  - Kaposiform hemangioendothelioma.

Vascular Malformations

- Slow flow:
  - Capillary
  - Port-wine (venular)
  - Venous malformation
  - Lymphatic malformation
  - Complex combined
    - Klippel-Trenaunay syndrome (KTS)
    - Parkes-Weber Syndrome (PWS).
- Fast flow:
  - Arterial malformation
  - Arteriovenous fistula
  - Arteriovenous malformation.

CLINICAL PRESENTATION

Vascular malformations may present as high flow or low flow lesions. High flow lesions include arteriovenous fistula and arteriovenous malformations. Extensive lesions with an arterial component may lead to high output cardiac failure with resultant CHF and pulmonary edema. Low flow lesions, those with venous and lymphatic malformations do not present such a risk. There may be accompanying pain, tissue ulceration, disfigurement, multiorgan compromise, impairment of limb function, claudication, hemorrhage and progressive nerve degeneration or palsy. The important vascular malformations have been outlined here.

Venous Malformation

Most common type of symptomatic vascular malformation, venous malformations can be single or multiple. Skin is the most common appendage involved, however, any tissue or organ in the body can be affected. They are composed of abnormally formed, dilated veins with very thin walls due to a relative lack of smooth muscle cells. Venous malformations are often mistakenly called hemangiomas. They may not be obvious until later in childhood or adulthood. The involved skin is usually soft with compressible dark blue bulges. Large ones can bleed spontaneously or can be associated with bleeding and
clotting abnormalities due to consumption of platelets and clotting factors.\textsuperscript{3,4,5}

Venous malformations are amenable to treatment with sclerotherapy or surgical debulking. Surgery is more invasive than sclerotherapy and is rarely curative.\textsuperscript{4,5}

**Arteriovenous Malformation**

Arteriovenous malformations are abnormal communications between arteries and veins. Brain is the most common appendage involved, however, head and neck, limbs, trunk and internal organs can be affected too. The microscopic capillary bed which normally intervenes between arteries and veins is absent resulting in higher than normal blood flow. They can be sporadic or can occur in conditions such as hereditary hemorrhagic telangiectasia (HHT or Osler-Weber-Rendu syndrome), and Parkes-Weber syndrome.

Arteriovenous malformations of skin and superficial tissues present with gradual enlargement and color changes. Local warmth associated with an enlarging pulsatile mass is a common presentation. Pain, ulceration or bleeding is not uncommon with progression. In rare instances, the blood flow through the malformation with time, can get so fast that a hyperdynamic circulation may result which could ultimately precipitate high output cardiac failure.\textsuperscript{3,4}

Arteriovenous malformations can be treated by angiographically guided endovascular and percutaneous embolization and sclerotherapy. This involves delivering the embolic/sclerosant material to the malformations either through a catheter inserted through an artery in the groin or through a needle placed directly through the skin. Surgical resection can sometimes be ‘curative’ for suitable small focal arteriovenous malformations.\textsuperscript{1,5}

**Lymphatic Malformation**

Lymphatic malformations have been described as lymphangioma, cystic hygroma, lymphangioma circumscriptum, and lymphangiomatosis. They are sponge-like collections of lymphatic channels and cystic spaces that contain clear lymphatic fluid. They can be macrocystic or microcystic depending on whether they contain large or microscopic fluid spaces. They most commonly occur in the head and neck and upper chest but can occur in any location throughout the body. They usually present as localized swellings which can worsen with a generalized viral or bacterial illness. Mucosal lesions that affect the floor of the mouth and tongue belong to the microcystic type and are hard to eradicate with surgery. The cervical lesions, also known as cystic hygomas, are macrocystic and easier to resect. The latter presents as painless nonpulsatile masses with a rubbery consistency that is covered by normal colored skin.\textsuperscript{1,2}

Infection and bleeding within a lymphatic malformation can cause periodic worsening of symptoms. Depending on the location, leakage of lymphatic fluid may cause excess fluid in the abdomen or chest (chylous ascites or chylothorax) and generalized protein loss. MRI is arguably the best overall imaging modality for characterizing lymphatic malformations.\textsuperscript{5,9}

Treatment modalities are either surgical excision for small focal lesions or sclerotherapy which involves directly injecting the lymphatic malformation with sclerosing agent that results in sclerosis and regression over a short period of time. GA may be required in young children for the procedure. Patients with extensive disease who are treated with surgery have a high potential for significant complications including cranial neuropathies. Increasingly, lymphatic malformations are being diagnosed prenatally. Large lymphatic malformations of the head and neck can be an indication for EXIT (ex-utero intrapartum treatment) procedure for initial airway control.\textsuperscript{2,10}

**Capillary Malformation**

Capillary malformations are flat, sharply demarcated, reddish-purple staining of the skin, sometimes referred to as a “port-wine stain”. They generally present after birth and most commonly involve the head and neck. Most are harmless birthmarks but there could be underlying abnormality if upper eyelid, forehead or the spinal column is involved. They are often present as part of combined vascular malformations associated with limb overgrowth. Capillary malformations are treated with pulsed-dye laser and are generally not treated by Interventional Radiology techniques.\textsuperscript{1,7}

**Complex Combined Vascular Malformations**

Complex combined malformations affect the extremities and trunk and are associated with limb overgrowth in both girth and in length. They involve more than one tissue/channel type. They can be accompanied by overgrowth of soft tissue and bone in the affected region/extremity. They sometimes affect more than one extremity and often involve the perineum and trunk. The most common forms are Klippel-Trenaunay syndrome (KTS) and Parkes-Weber syndrome (PWS).\textsuperscript{4,7}
KTS → It is a Capillary-lymphaticovenous malformation (CLVM). It usually involves the lower extremity and trunk in combination but can also involve an upper extremity. The lymphatic component of the syndrome is prone to infection and internal bleeding while the venous component can be complicated by blood clot formation which can migrate to the lungs.

PWS → It is a capillary-arteriovenous malformation (CAVM). The arteriovenous malformation component of this syndrome is most troublesome with pain, skin breakdown, bleeding and often high output cardiac failure due to the number and size of the arteriovenous shunts.

Treatment of complex combined malformations includes interventional radiological techniques discussed previously. A multi-disciplinary approach with inputs from surgeons, dermatologists, hematologists and radiologists is vital.5

TREATMENT

In view of the risks of significant intraoperative bleeding and coagulopathy in addition to the surgical and anesthetic risk, sclerotherapy and invasive embolization of lesions have become popular alternatives to surgical resection when possible.5

Sclerotherapy/Embolization Procedures

Radiologically guided treatment for all types of vascular malformation involves percutaneous (through the skin) and endovascular (through blood vessels) placement of needles and catheters to deliver the sclerosant or embolic material to the affected lymphatic or vascular channel. Ethanol, stainless steel coils, antibiotics, absorbable gelatin pledgets and powder, polyvinyl alcohol foam are often used by the radiologist when embolizing vascular malformations. The choice of agent depends on the clinical situation and the size of blood vessel.1,3

Delivery of sclerosant can be painful and procedures can be lengthy. For both these reasons, it is preferable to perform sclerotherapy under general anesthesia. Treatment is usually followed by admission for observation with patients normally discharged the day following the procedure.

Anesthesia Goals

The goals of anesthesia are management and protection of the airway (difficult airway cart to be kept ready), maintaining cardiovascular and neurological stability, manipulating systemic and regional blood flow, and managing anticoagulation.

Anesthesia Technique

Anaesthesia for vascular malformation may be complicated due to the proximity to the airway, size, presence of congestive heart failure, thrombocytopenia and blood loss.11 A dedicated anesthesia team committed to interventional radiology has an advantage. The patients and parents often return for multiple procedures and are comforted by the sight of familiar anesthesiologists.

Hemangiomas or capillary malformations such as port-wine stain without a systemic involvement are amenable to treatment in the outpatient department. An inhalational anaesthetic via a face mask may suffice for the conduct of the therapeutic procedure. Lesions on face or back may warrant the use of supraglottic airway device or endotracheal intubation to secure the airway.11

ANESTHETIC CONCERNS AND COMPLICATIONS

Absolute ethanol is a powerful sclerosing agent, which has particular implications for the anesthesiologist. It causes denaturation of blood proteins and may produce a coagulum of blood with endothelial necrosis. A post procedure coagulopathy marked by positive d-dimers, elevated prothrombin time and decreased platelets may result. Extensive ethanol injection (>0.5 mL/kg) can cause hematuria.

Pulmonary embolism from thrombus dislodgement at the site of vascular malformation may occur with mild oxygen desaturation and prolonged (24–48 hours) hypoxemia presumably from micro-thromboembolism. These patients benefit from systemic anticoagulation. Narcotics may produce a synergistic effect with the potential for respiratory depression.

Large malformations may be associated with a coagulopathic condition called Kasabach-Merritt syndrome. In this condition, the hemangioma traps and destroys platelets and clotting factors resulting in thrombocytopenia with an increased risk of hemorrhage. Such patients may not be ideal candidates for regional anaesthesia.11 A condition described as systemic intravascular coagulation can occur after the embolization of extensive vascular malformations. It is similar to DIC but specific for coagulopathy which results from embolizations. During extensive embolizations, cryoprecipitate or platelets may be administered to promote clotting and successful sclerosis.

Extensive embolization procedures frequently lead to tissue necrosis and swelling. Postembolic swelling may impact the perioperative airway management following procedures in head and neck. Children may need to remain
intubated after such procedures particularly when edema in the floor of mouth, tongue, hypopharynx, oropharynx, or anterior neck could compromise a patent airway.\textsuperscript{11}

Finally, with the use of iodine containing radiocontrast media, sclerosing and embolizing agents, consideration must be given to adequate volume resuscitation, the risk of a contrast reaction and bladder catheterization for detection of oliguria, polyuria or hematuria.

### Learning Points
- Vascular anomalies embody a myriad of blood vessel abnormalities that are thought to occur perinatally.
- Correct diagnosis is imperative for appropriate treatment.
- Differentiating between highflow and low flow lesions is vital to diagnosis and therapy of vascular malformations.
- Most vascular malformations are palliated, not cured and may require multiple treatments and multi-disciplinary approach.

### References

   
   


INTRODUCTION

Cardiopulmonary resuscitation in neonates and children is different from adults. The differences in size and physiology, etiology of cardiac arrest, its effects, techniques used and the available resources make pediatric cardiopulmonary resuscitation a challenge. An indirect estimate of the need for resuscitation in India can be gauged from the number of neonatal deaths (approximately 7–8 lakhs annually), and an under-5 mortality rate of 21%. The mortality is higher in pediatric out of the hospital cardiac arrests (OHCA) as compared to in hospital cardiac arrests (IHCA), of which anesthesia and surgery related arrests are a significant majority. A 2012 systematic review documented worldwide anesthesia related mortality at <1 death per 10,000 pediatric anesthetics administered, with developing countries having 10.7–15.9 deaths per 10,000 anesthetics. This chapter discusses the latest recommendations for cardiopulmonary resuscitation in neonates and children as mentioned in the AHA update 2015.

PRIMARY CAUSES OF PEDIATRIC CARDIAC ARREST (TABLE 1)

<table>
<thead>
<tr>
<th>Neonatal</th>
<th>Infants</th>
<th>Child ≥ 1 year of age</th>
</tr>
</thead>
<tbody>
<tr>
<td>Perinatal asphyxia</td>
<td>Pneumonia</td>
<td>Infections</td>
</tr>
<tr>
<td>Severe infections</td>
<td>Diarrhea</td>
<td>Injury</td>
</tr>
<tr>
<td>Preterm births</td>
<td>Congenital malformations</td>
<td></td>
</tr>
</tbody>
</table>

STRATEGIES AND GUIDELINES TO IMPROVE SURVIVAL

Guidelines for the optimal management of neonatal and pediatric cardiac arrest have been issued by various national and international organizations in a bid to improve survival. The links are as follows:

- Prevention
- Early cardiopulmonary resuscitation (CPR)—Emphasis on chest compressions
- Early access to emergency medical system (EMS)
- Early advanced life support (ALS)
- Post-resuscitation care.

Prevention

Neonatal deaths can be prevented through implementation of the Newborn Resuscitation Program: First Golden Aparna A Nerurkar, Naina P Dalvi
Minute Project by having one person trained in neonatal resuscitation at every delivery, identifying and treating causes of birth asphyxia like intrauterine growth retardation, prematurity, infections, congenital malformations, tetanus, etc. The Back to Sleep campaign to reduce SIDS, swimming pool safety, use of seat belts and car seats, helmets and protective gear during sports, child-proofing of medicine bottles and targeted public health interventions are important preventive strategies. Early identification of congenital diseases, screening for channelopathies and cardiomyopathies can prevent SCA in adolescents and young adults. Formation of dedicated pediatric medical emergency team can reduce in-hospital mortality.

Early Detection and Treatment of Life Threatening Illness in Child

The AHA Pediatric Advanced Life Support (PALS) course recommends an evaluate, identify and intervene sequence to identify life threatening illnesses followed by Primary ABCDE (Airway/Breathing/Circulation/Disability/Exposure) survey and Secondary survey with SAMPLE (Signs and Symptoms/Allergies/Medications/Past illness/Last meal/Events), Focused examination, Head-to-Toe examination, diagnosis and investigations with ongoing evaluation, assessment and treatment for management of pediatric emergencies.

Early recognition of the symptoms and signs and early intervention can reverse the child’s deterioration to cardiorespiratory arrest. It is thus important to know normal parameters in children (Table 2).

Warning signs include increased respiratory rate or effort like nasal flaring, retractions, seesaw breathing or grunting, inadequate respiratory rate or effort, gasping or diminished chest excursion, especially with depressed mental status, pallor or cyanosis despite supplementary oxygen. Tachycardia, cool and pale distal extremities, prolonged (>2 seconds) capillary refill (despite warm ambient temperature), weak peripheral pulses compared with central pulses and normal systolic blood pressure suggest compensated shock.

Table 2: Normal pediatric parameters

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Breaths/min</th>
<th>Age</th>
<th>Heart rate/min</th>
<th>Age</th>
<th>Hypotension threshold (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infant &lt;1</td>
<td>30–60</td>
<td>Newborn-3 months</td>
<td>80–205</td>
<td>Term neonates</td>
<td>60</td>
</tr>
<tr>
<td>Toddler (1–3)</td>
<td>24–40</td>
<td>3 months-2 years</td>
<td>75–190</td>
<td>Infants</td>
<td>70</td>
</tr>
<tr>
<td>Pre schooler (4–5)</td>
<td>22–34</td>
<td>2–10 years</td>
<td>60–100</td>
<td>Child (1–10 yrs)</td>
<td>70 + (2 x age in yrs)</td>
</tr>
<tr>
<td>School age (6–12)</td>
<td>18–30</td>
<td>&gt; 10 years</td>
<td>50–100</td>
<td>Child ≥10 yrs</td>
<td>90</td>
</tr>
<tr>
<td>Adolescent (13–18)</td>
<td>12–16</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Early Cardiopulmonary Resuscitation (CPR)

In an effort to improve bystander CPR and simplify guidelines, AHA has recommended that CPR be started with compressions, even for infants and children. The European guidelines however recommend giving five rescue breaths prior to checking for signs of life.

As per the AHA algorithm; the infant guidelines apply to children beyond neonatal age to approximately 1 year of age, child guidelines from age of 1 year to puberty (breast development in females and the presence of axillary hair in males) and adult guidelines apply at and beyond puberty.

Early CPR starts with identification of arrest. Assure safe environment for victim and responder. Establish unresponsiveness by tap and talk i.e. gently tap and ask a question looking for a response in the form of answer, moaning, movement or breathing, call for help verbally or using mobile devices, if available; to gain access to EMS/automated external defibrillator (AED)/manual defibrillator. Identify apnea or gasps while simultaneously performing a quick pulse-check taking not more than 10 seconds. Pulse check is performed on umbilical artery in neonates, brachial or femoral in infants and carotid or femoral in child/young adult.

If pulse is absent or rate is less than 60/min or signs of poor perfusion are present (i.e. signs of decompressed shock) start compressions at a rate of 100–120/min. Position the patient supine and deliver chest compressions on a firm surface. Neonatal and infant compressions are preferentially delivered with thumb encircling technique (Fig. 1) when two rescuers are present and two finger technique (Fig. 2) just below the inter-mammary line when 1 rescuer is present. In children heel of one hand (Fig. 3) or two hands like in adults can be used just below the inter-mammary line taking care not to compress the
xiphisternum or ribs. Sternum should be compressed to a depth of at least one third the anterior-posterior (AP) diameter of the chest or approximately 1.5 inches (4 cm) in infants and 2 inches (5 cm) in children; not exceeding upper limit of 6 cm in adolescents. Compression to relaxation ratio should be 50:50 allowing adequate time for recoil and cardiac filling. Interruptions in compressions should be minimised to allow good coronary perfusion. Rescuers should change roles of compressor and ventilator every 2 minutes or sooner if fatigued to ensure good quality of chest compressions. Compression: Ventilation ratio is 30:2 with single rescuer and 15:2 with 2 rescuers present.

If pulse is present with adequate rate and perfusion; check and open the airway.

If patient has an open airway and has adequate breathing; give recovery position after confirming that there is no history of trauma; and continue monitoring. Give recovery position in neonate or infant by cradling the patient with both arms while keeping their head tilted downwards (Fig. 4). In case of children, place the child in lateral position with elbow of the nearest hand bent at a right angle and palm facing upwards, back of other palm below nearest cheek, head tilted with chin lift, dependent leg extended and the other leg flexed (Fig. 5). This position

If airway is not patent, open airway with head tilt, chin lift, jaw thrust maneuver and removal of foreign body as required. If child is apneic or gasping with adequate pulse-deliver rescue breaths mouth to mouth and nose or mouth to mouth with pinched nose or mouth to nose with patient’s mouth closed or mouth to mask as required; at a rate of one every 3–5 seconds (12–20 breaths/min). For adolescents give rescue breaths at a rate of 1 breath every 5–6 seconds (10–12 breaths/min). Each breath should take about 1 second with adequate chest rise. Recheck pulse every 2 minutes. Consider Naloxone if opioid overdose is suspected.11

Early Access to EMS
AHA recommends an early call for help and access to EMS to improve survival. In a lone rescuer, witnessed arrest situation; call for nearby help, activate EMS via mobile device if appropriate, look for no breathing or gasping with simultaneous pulse check; perform 2 minutes of CPR, then activate EMS if not already activated and get AED. Return to victim as soon as possible; resume CPR starting with chest compressions and use AED when available. In a two rescuer witnessed arrest situation, first rescuer remains with the victim, confirms arrest as above and starts CPR with 30:2 ratio of compressions to ventilation. The other rescuer activates the EMS and gets AED and emergency equipment and returns to the victim and CPR is continued with a 15:2 ratio. In sudden witnessed arrest (e.g. sudden collapse in an adolescent or a child identified at high risk for arrhythmia or during an athletic event), assume ventricular fibrillation (VF) induced pediatric cardiac arrest; follow above protocols as per number of rescuers with an aim of obtaining the AED as soon as possible. Attach the AED pads after switching on the power and follow the audiovisual prompts. The AED analyzes the rhythm, selects the dose and charges to the appropriate energy. Press the shock button after clearing the patient when indicated.

GUIDELINES REGARDING USE OF AED IN CHILDREN
For children between 1–8 years, pediatric dose attenuator system with AED is preferred. If not available, standard AED can be used. For infants, a manual defibrillator is preferred. If unavailable, AED with dose attenuator can be used or standard AED is used.

If a manual defibrillator is obtained in hospital setting; it is attached and rhythm identification is done to detect shockable rhythm. If the rhythm is shockable, i.e. ventricular fibrillation (VF) or ventricular tachycardia (VT), shock at 2 J/kg is delivered after clearing the patient. CPR is continued starting with compressions. A 2 minute cycle comprising of 5 sets of compressions and ventilations are delivered prior to next rhythm and pulse. Second Shock at 4 J/kg and subsequent shocks at ≥4 J/kg not exceeding 10 J/kg or adult dose can be given if required. Defibrillation can be performed using adult defibrillator pads if the affected child weighs more than 10 kg. There should be a distance of at least 3 cm between the defibrillation pads or paddles. Infant paddles should be used for children younger than 1 year or weighing less than 10 kg.

If the rhythm is non-shockable i.e. asystole or pulseless electrical arrhythmia (PEA), CPR is continued.
with rhythm check every 2 minutes with aggressive efforts to detect the cause of arrest.

**Early Advanced Life Support**

This consists of advanced airway management, oxygen supplementation, obtaining intravenous or intraosseous access, attachment of monitors with administration of medications as required. Use oropharyngeal and/or nasopharyngeal airways, as required to obtain a patent airway. Use bag-mask ventilation to deliver breaths. Avoid delivering excessive ventilation during cardiac arrest. Use adequate force to get just a visible chest rise. Bag-mask ventilation can be effective and safer in infants and children where early transportation to advanced care centers is anticipated. When bag-mask ventilation is unsuccessful and when endotracheal intubation, the gold standard, is not possible, experienced providers may use LMA to provide a patent airway and support ventilation. However, LMA may be associated with a higher incidence of complications in young children. Other supraglottic advanced airway devices (e.g. laryngeal tube, I-gel), used successfully in pediatric anesthesia, may also be useful in an emergency but there are few data on the use of these devices in pediatric emergencies. Endotracheal intubation by skilled personnel with appropriate sized cuffed or uncuffed endotracheal tube is recommended for securing airway. Five point auscultation, capnometry, Carbon dioxide (CO₂) indicator, direct visualization with laryngoscopy, X-ray in hospital setting are used to confirm proper tube placement. An esophageal detector device (EDD) may be considered to confirm endotracheal tube placement in children weighing 20 kg with a perfusing rhythm.

Capnometry may be used to evaluate the quality of chest compressions, but specific values to guide therapy have not been established in children. Once the victim is intubated, ventilate at a rate of about 1 breath every 6 seconds (10 times per minute) without interrupting chest compressions. Use maximum feasible oxygen concentration initially and then use appropriate equipment to titrate oxygen administration to maintain the oxyhemoglobin saturation ≥94%. If the patient has a perfusing rhythm, use pulse oximeter to monitor oxyhemoglobin saturation continuously.

Intraosseous (IO) access is a safe, effective, and acceptable initial route for vascular access in children. Peripheral intravenous (IV) access if it can be placed rapidly is also acceptable. Lipid-soluble drugs, such as Lidocaine, Epinephrine, Atropine, and Naloxone (mnemonic LEAN) can be administered via an endotracheal tube if IV/IO is not possible. Give epinephrine 0.01 mg/kg IO/IV (0.1 mL/kg of 1:10,000) or via ETT 0.1 mg/kg (0.1 mL/kg of 1:1,000) every 3–5 mins. Give IV amiodarone 5 mg/kg or lidocaine 1 mg/kg in case of VF/VT. Detect and treat the reversible causes of arrest - the 6 H’s—Hypovolemia, Hypoxia, Hydrogen ion (acidosis), Hypo-/hyperkalemia, Hypothermia, Hypoglycemia and 5 T’s—Tension pneumothorax, Tamponade cardiac, Toxins, Thrombosis-pulmonary and coronary.

**Extracorporeal cardiopulmonary resuscitation** (ECPR) may be considered for pediatric patients with cardiac diagnoses who have IHCA in settings with existing Extracorporeal Membrane oxygenation (ECMO) protocols, expertise, and equipment.

**Postresuscitation Care**

The goal is to preserve brain function and limit the risk of secondary neuronal injury. AHA 2015 update has adopted the term targeted temperature management (TTM) as a strategy of temperature control postresuscitation. For children who are comatose in the first several days after cardiac arrest (in hospital or out of hospital), temperature should be monitored continuously and fever should be treated aggressively. For comatose children resuscitated from OHCA, it is reasonable to maintain either 5 days of normothermia (36°C to 37.5°C) or 2 days of initial continuous hypothermia (32°C to 34°C) followed by 3 days of normothermia. For children remaining comatose after IHCA, there is insufficient data to recommend hypothermia over normothermia. Prehospital cooling of patients after ROSC with rapid infusion of cold intravenous fluids is not recommended. Therapeutic hypothermia is to be considered for comatose postresuscitation adolescent survivors, infants and neonates born at >36 weeks gestation with evolving moderate to severe hypoxic-ischemic encephalopathy. Avoidance of hyperthermia (≥38°C), maintenance of oxygen saturation 94–99%, avoidance of hyperventilation unless used as rescue therapy to treat signs of impending cerebral herniation, use of fluids and inotropes/vasopressors to maintain a systolic blood pressure above the fifth percentile for age, continuous intraarterial pressure monitoring, when available, to identify and treat hypotension, avoidance of hypoglycemia and maintenance of blood chemistry need to be followed for postresuscitation care.

The pediatric resuscitation protocol is summarized in the Flowchart 1.
Chapter 34: Cardiopulmonary Resuscitation in Neonates and Children

FLOWCHART 1: Pediatric life support (health care providers) 5,6,11

**SPECIAL SITUATIONS**

**Arrest Under Anesthesia**

**Causes**

The major risk factors for anesthetic related cardiac arrests are age-newborn or less than 1 year old, ASA III or worse physical status, and children undergoing emergency surgery, general anesthesia, or cardiac surgery.1,9,10,13,14 Causes of cardiac arrest during anesthesia can be mainly divided into four categories; those resulting from preoperative status; surgical procedures; intra-operative pathological events; and those attributed to anesthetic management.21 Anesthesia-related events are due to airway or ventilation, cardiovascular-related events, medication accidents and infusion/transfusion mishaps.9,13,20 However, cardiac arrest during anesthesia differs from other situations in that the arrest occurs in an setting ideal for optimal quality resuscitation. The cardiac arrest is witnessed, patient is being monitored, intravenous accesses are secured, and oxygen, emergency drugs and defibrillator are immediately available.

**Prevention**

Follow 1999 AAP Guidelines to prevent anesthetic mishaps.12 Identify high risk groups, clinical conditions like preoperative anemia, airway difficulties etc. Decrease the potential for human error by increased vigilance, use of a team approach in the form of two anesthesiologists in anticipated high-risk cases, prompt investigation of abnormalities and early intervention.

Specific strategies like use of supraglottic airway devices, use of microcuffed tubes, sevoflurane over halothane, careful use of Bradygenic drugs like propofol, opioids, muscle relaxants, proper use of test doses during regional anesthesia, anticipating and treating blood loss with the freshest packed red blood cells (PRBCs) available and avoidance of whole blood, use of cell saver, treating concomitant temperature and electrolyte changes, use of pulse oximetry, capnography can help prevent arrests.
**Treatment**

Evaluate airway breathing circulation (ABC). However, remember difficulties in ABC are responsible for most arrests. Hence critically assess and uncover difficulties in airway, ventilation, oxygenation or perfusion. Re-establish ABC/CAB as per age. Do not assume that ABCs are in order just because that was the case earlier. Do a quick assessment of the 6 H’s and 5 T’s. Peri-arrest arrhythmias—bradycardia which is often a cause of arrest; or tachycardia need to be identified and treated.

**Neonatal Resuscitation**

Follow the Flowchart 2 as described considering the following points:
- Delaying umbilical cord clamping for longer than 30 sec is reasonable for term and preterm newborns not requiring resuscitation.
- Maintain the temperature of newly born non-asphyxiated infants between 36.5–37.5 degree centigrade after birth. Complete initial steps of resuscitation under the radiant warmer for infants.

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**Flowchart 2**: Basic neonatal resuscitation: the golden minute management

1. **Birth**
   - No meconium—Dry the baby
   - If meconium present—Suction mouth, then nose (if baby not crying)
   - Dry the baby
   - Assess breathing

2. **Breathing well/crying**
   - Routine care: Warmth: suction mouth, nose if required; cut cord in 1–3 mins; baby to mother, breastfeeding

3. **Not breathing well**
   - Cut cord, place baby on firm, flat surface with neck slightly extended; warmth; suction mouth then nose; stimulate, reposition

4. **Reassess breathing (by 30 seconds of birth)**

5. **Breathing well/crying**
   - Observational care: Warmth; observe breathing, temperature; look for complications—convulsion, coma, poor feeding, lethargy, respiratory distress and refer as needed; if well-breastfeeding advised

6. **If not breathing well**
   - Provide bag-mask ventilation at 40–60/min for 30 seconds ensuring chest rise

7. **Reassess breathing**

8. **Breathing well/crying**
   - Observational care as above

9. **If not breathing well**
   - Call for help; continue bag-mask ventilation, add oxygen if available

10. **Assess heart rate (by 1 minute of birth)**

11. **HR <100**—Continue ventilation with oxygen; provide advanced care—compressions, 120 beats/min at 3:1 compressions/ventilations; intubation, medications—epinephrine/volume expansion; referral

12. **HR ≥100**—continue ventilation

13. **Reassess breathing—Observational care; if breathing well**

14. **Postresuscitation care**
   - Warmth; Observe breathing, temperature, color, capillary refill time, blood sugar; look for complications and refer as needed; if well-breastfeeding
born through meconium-stained amniotic fluid presenting with poor muscle tone and inadequate breathing efforts.

- Perform endotracheal suction only in nonvigorous babies with meconium-stained amniotic fluid.
- Titrate oxygen supplementation in term or preterm babies being resuscitated at birth with pulse oximetry. Resuscitate preterm newborns of less than 35 weeks of gestation with low oxygen (21% to 30%), and titrate the oxygen concentration to achieve a preductal oxygen saturation approximating the interquartile range measured in healthy term infants after vaginal birth at sea level. These are 60–65% at 1 minute with successive 5% increase at 2, 3, 4 and 5 minutes and saturation of 85–95% at 10 mins.\(^7\)
- It is reasonable to use 3 lead ECG monitoring to assess heart rate.
- If the baby is bradycardic (HR <60 per minute) after 90 seconds of resuscitation with a lower concentration of oxygen, give 100% oxygen till recovery of normal heart rate.
- Positive pressure ventilation should be initiated if the infant is not breathing or the heart rate is less than 100/min after the initial steps are completed. Assist ventilation at the rate of 40 to 60 breaths per minute to achieve or maintain the heart rate of >100 per minute.
- Laryngeal mask airway can be considered in case of unsuccessful/failed bag-mask ventilation or endotracheal intubation.
- Endotracheal intubation, may be indicated for nonvigorous meconium-stained newborns, ineffective or prolonged bag-mask ventilation, babies requiring chest compressions, newborns with congenital diaphragmatic hernia or extremely low birth-weight neonates.
- Compressions, when required, are administered at 120 events/min with a ratio of 3:1 compressions/ventilations.
- Consider hypovolemia or pneumothorax if heart rate persistently below 60/min.

**Drowning\(^22\)**

- Take the victim out of water immediately without endangering rescuer's life
- Consider the possibility of cervical spine injury
- Consider hypothermia if icy water drowning. Attempt to rewarm if core temperature <30°C
- Wipe chest before defibrillation to prevent electrical arcing between paddles
- Suction airway if water is vomited out. Decompress stomach with nasogastric tube
- Transport all victims of drowning who require any form of resuscitation (including rescue breathing alone) to the hospital for evaluation and monitoring, even if they appear to be alert and demonstrate effective cardiorespiratory function at the scene.

**Foreign Body Airway Obstruction (FBAO)**

**Flowchart 3:**

<table>
<thead>
<tr>
<th>Partial FBAO</th>
<th>Complete FBAO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Child able to make sounds or cough</td>
<td>Child unable to make sound or cough or breathe</td>
</tr>
</tbody>
</table>

- Allow child to clear the airway by coughing
- Check responsiveness

**Child responsive**

- Try age appropriate maneuver till foreign body is out or child becomes unresponsive
- Neotones and Infants
  - Give 5 back slaps followed by 5 chest thrusts
- Child > 1 year of age
  - Heimlich manoeuvre/abdominal thrusts:
    - Make a fist with one hand, position it just above navel, grasp this fist with other hand and thrust upward with the intention to remove the FB

**Child unresponsive or Partial FBAO child becomes unconscious**

- Begin CPR with chest compressions
- Perform additional step of looking for dislodged foreign body in mouth and removing it prior to delivering rescue breaths
Anaphylaxis

Intense vasodilatation leading to relative hypovolemia, severe bronchoconstriction causing hypoxia and impaired tissue oxygen delivery are important manifestations. Management involves standard CPR protocol with early and rapid advanced airway management in view of impending oropharyngeal/laryngeal edema, treatment of asystole or PEA, large volumes of isotonic crystalloids using two large bore IV/IO lines and epinephrine (0.01 mg/kg max 0.5 mg IM) to be repeated every 5 to 15 minutes in the absence of clinical improvement.

Poisoning

Drug overdose or poisoning can cause cardiac arrest either due to direct cardiotoxicity or secondary effects of respiratory depression, peripheral vasodilatation, arrhythmias and hypotension. Cardiac dysfunction will persist till the drug is metabolized or reversed. As the toxicity may be temporary, prolonged resuscitation efforts and advanced support system like extracorporeal membrane oxygenation (ECMO) may give good results. Early search for the toxin and specific treatment along with standard CPR will improve the outcome. Naloxone for opioid toxicity, intravenous lipid emulsion for systemic local anesthetic toxicity have been deemed reasonable therapies as per the AHA 2015 update.

Trauma in Children

The common mechanisms of injury are low/high speed pedestrian struck accidents, unrestrained/restrained automobile occupant, fall from height, fall from bicycle-with or without helmet or due to striking handlebar. Child can have chest trauma, abdominal trauma, head injury, spinal cord trauma or musculoskeletal trauma. The same trauma ABCDE is followed:

- Airway with cervical spine control
- Breathing with identification and management of life-threatening chest injury
- Circulation with bleeding control and shock management
- Disability with recognition and management of altered mental status
- Exposure with maintenance of body temperature.

Electrical injuries

**Manifestations**

- Victims of alternating current (AC) contact electrical shock may have tetanic muscle contractions locking victim to the current, VF and multiple injuries
- Lightning strike victims may have primary VF or asystole; thoracic muscle spasm, suppression of the respiratory center; extensive catecholamine release; peripheral and central neurological injuries.

**Treatment**

- Dislodge victim with non-conducting material while maintaining scene safety
- Remove smouldering contact material
- Spinal stabilization during extrication and treatment
- Early intubation in electric burns of the face, mouth, or anterior neck
- Rapid IV fluid to maintain diuresis; facilitate excretion of myoglobin, potassium, and other by-products of tissue destruction.

Periarrrest Arrhythmias

**Tachyarrhythmias**

- Identify and treat cause while assessing and supporting ABCs
- Identify as narrow complex (QRS ≤ 0.09 sec) or wide complex (QRS ≥ 0.09 sec)
- For narrow complex tachycardia consider vagal maneuvers like asking cooperative child to blow through an obstructed straw. Unilateral carotid sinus massage or icewater bag application over upper half of face taking care not to obstruct the airway can be tried in other children. Avoid ocular pressure due to fear of retinal injury
- IV adenosine (0.1 mg/kg, 2nd dose 0.2 mg/kg) is given for narrow complex tachycardias
- Consider IV amiodarone (5 mg/kg) or procainamide (15 mg/kg), lignocaine (1 mg/kg) and magnesium 25–50 mg/kg for stable patients with wide complex tachycardias
- Synchronized cardioversion with 0.5–1 J/kg is done for unstable wide complex tachycardias.

**Bradyarrhythmias**

- Identify and treat cause while assessing and supporting ABCs
- CPR if heart rate ≤ 60/min
- Therapies include IV/IO epinephrine 0.01 mg/kg, atropine 0.02 mg/kg with minimum 0.1 mg dose and pacing as required.

RECENT AHA UPDATE 2015

The updated recommendations for CPRC mentioned in the AHA update 2015 have been summarized in the following Table 3.
### Table 3: AHA 2015 update important points

<table>
<thead>
<tr>
<th>BLS</th>
<th>Algorithms</th>
<th>Separate algorithms for 1- and 2-person healthcare provider CPR</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Compression rate</td>
<td>Chest compression rate of 100/min to 120/min for infants and children</td>
</tr>
<tr>
<td></td>
<td>Compression depth</td>
<td>Depth of at least 5 cm, but no more than 6 cm, for the adolescent of average adult size</td>
</tr>
<tr>
<td></td>
<td>Compression only CPR</td>
<td>Conventional CPR (chest compressions and rescue breaths) provided for pediatric cardiac arrest. Compression-only CPR if rescuers unwilling or unable to deliver breaths</td>
</tr>
</tbody>
</table>

| ALS | Ventilation | Once intubated, ventilate at a rate of about 1 breath every 6 seconds (10 times per minute) without interrupting chest compressions |
|     | Shock refractory VF/pVT treatment | Both amiodarone and lignocaine acceptable for treatment of shock refractory ventricular fibrillation/ pulseless ventricular tachycardia |
|     | Atropine | No routine use of atropine for emergency tracheal intubation in non-neonates, unless specific risk of bradycardia, no minimum dose of atropine for this indication |
|     | Blood pressure monitoring | If invasive hemodynamic monitoring is in place at the time of a cardiac arrest in a child, use it to guide CPR quality |
|     | ECPR (Extracorporeal cardiopulmonary resuscitation) | ECPR may be considered for pediatric patients with cardiac diagnoses who have in-hospital cardiac arrest in settings with existing extracorporeal membrane oxygenation protocols, expertise, and equipment |

| Neonate | Cord clamping | Delay umbilical cord clamping for longer than 30 sec for term/preterm newborns not requiring resuscitation |
|         | Temperature | Maintain the temperature of newly born non-asphyxiated infants between 36.5–37.5°C after birth |
|         | Complete initial steps of resuscitation under the radiant warmer for infants born through meconium-stained amniotic fluid presenting with poor muscle tone and inadequate breathing efforts |
|         | Oxygen supplementation | Resuscitate preterm newborns of less than 35 weeks of gestation with low oxygen (21% to 30%), and titrate oxygen to achieve a preductal oxygen saturation approximating the interquartile range measured in healthy term infants after vaginal birth at sea level |
|         | ECG monitoring | Use 3 lead ECG monitoring to assess heart rate |
|         | Respiration | Positive pressure ventilation to be initiated if the infant is not breathing or the heart rate is less than 100/min after the initial steps are completed |
| Post-resuscitation | TTM (Targeted temperature management) | Temperature should be monitored continuously and fever should be treated aggressively in comatose children after cardiac arrest. For comatose children resuscitated from OHCA, maintain either 5 days of normothermia (36°C to 37.5°C) or 2 days of initial continuous hypothermia (32°C to 34°C) followed by 3 days of normothermia. After IHCA, there are insufficient data to recommend hypothermia over normothermia |
|         | Hemodynamic management | Use of fluids and inotropes/vasopressors to maintain a systolic blood pressure above the fifth percentile for age. Continuous intra-arterial pressure monitoring when available to identify and treat hypotension |
| Special circumstances | Poisoning | Naloxone for opioid toxicity, intravenous lipid emulsion for systemic local anesthetic toxicity have been deemed reasonable therapies |

### CONCLUSION

A systematic approach with protocol-based resuscitation can improve survival in cardiopulmonary arrest in children.

### REFERENCES


INTRODUCTION

Providing safe anesthesia for infants and children without doubt requires skill and expertise. Reviews on anesthesia-related morbidity and mortality have highlighted problems which if recognized and promptly managed can avoid perioperative cardiac arrest and mortality.1-9 These are most commonly due to cardiovascular, respiratory, equipment failure and medication related causes. Children aged less than 2 years of age with underlying heart disease, respiratory infections and sepsis are more likely to have untoward events under anesthesia. However, critical incidents can occur in normal children as well. This review outlines the common acute problems one can encounter during the process of anesthetizing young children and how one can best manage them.

CAUSES OF ACUTE PROBLEMS DURING ANESTHESIA IN CHILDREN

Cardiovascular

• Hypovolemia: pre-existing, blood loss, inability to keep up with hemorrhage, inaccurate assessment of volume loss, inadequate IV access, inadequate or inappropriate fluid replacement
• Electrolyte imbalance (hyperkalemia, hypocalcemia)
• Sudden arrhythmia
• Air embolism
• Anesthesia medication related: halothane, sevoflurane, isoflurane, propofol, narcotic-benzodiazepine combinations

• Perioperative cardiac arrest
• Myocardial ischemia.

Respiratory

• Airway obstruction: laryngospasm, bronchospasm
• Inadequate ventilation or oxygenation
• Difficult intubation, esophageal or endobronchial intubation
• Aspiration
• Pneumothorax, pulmonary edema
• Premature extubation.

Medication Related

• Wrong drug or dose
• Adverse drug reaction
• Malignant hyperthermia.

Equipment and Monitoring

• Line complications: arterial line, central line, peripheral line extravasation
• Breathing circuit obstruction or malfunction
• Endotracheal tube obstruction or kinking
• Faulty gas delivery
• Faulty warming equipment.

Miscellaneous

• Hypothermia
• Delayed awakening.
ACUTE CARDIOVASCULAR PROBLEMS\textsuperscript{10,11}

When sudden and unexplained changes in heart rate occur during surgery, one has to first rule out the possible anesthetic or patient-related problems. Discuss with the surgeon for any surgical related causes, i.e. bleeding, injected air, traction or pressure on viscera, or drug injected (adrenaline, hypertonic saline, antibiotic, local anesthetic or chemotherapeutic agents). Acute onset bradycardia, tachycardia or muscle weakness is known to occur if residual succinylcholine is present in the extension tubing between the injection port and the IV cannula following subsequent injection into the line. To ensure that no drug is retained in the dead space an immediate saline flush should follow drug injection.

Bradycardia\textsuperscript{12,13}

Bradycardia can occur with and without heart block or as an acute cardiovascular event along with hypotension. What is defined as bradycardia in children varies with age;

- Age <30 days: heart rate (HR) <100 bpm
- Age >30 days <1 year: HR <80 bpm
- Age >1 year: HR <60 bpm.

Causes

Include hypoxemia, heart disease, conduction defects, surgical stimulation under light planes of anesthesia and inhalation agent overdose.

Management

- Call for help, stop surgical stimulation
- Hypoxia is often the common cause of bradycardia. Give 100% oxygen, initiate manual ventilation, check ventilator, ensure oxygen delivery
- Rule out airway obstruction; kinked airway device or tubing disconnect
- If laparoscopy, desufflate
- Start cardiopulmonary resuscitation (CPR), chest compression if pulse is weak or absent or pulseless electrical activity
- Adrenaline 2–10 μg/kg IV push, atropine 0.01–0.02 mg/kg IV push, if vagal etiology.

Tachycardia

Tachycardia may be associated with hypotension, hypertension or a painful stimulus and appropriate management is initiated based on the cause. Electrocardiogram (ECG) characteristics and diagnosis of supraventricular tachycardia will determine the management of the same. Sinus tachycardia: narrow complex, p waves present before QRS; Supraventricular tachycardia: narrow complex, no p waves or p waves not associated with QRS; Ventricular tachycardia: wide complex, polymorphic or monomorphic.

Management

- If no pulse, start CPR
- If pulse present, observe ECG:
  - Narrow complex: Vagal maneuver: Valsalva, carotid massage
  - Amiodarone 5 mg/kg IV bolus over 20–60 minutes
  - Lignocaine 1 mg/kg IV bolus.

Acute Hypotension

- Definition: Systolic blood pressure <5 percentile for age or 20–30% fall from baseline
- 5 percentile for age
  - Term neonate <60 mm Hg
  - Infant >30 days to <1 year <70 mm Hg
  - Child 1 to 10 years <70 mm Hg + (2 x age in years)
  - Child ≥10 years <90 mm Hg.

Causes

- Hypovolemia Negative inotropic drugs Drug-induced vasodilation
- Venodilation Anesthetic agents Sepsis
- Impaired venous return Arrhythmias Anaphylaxis
- Tamponade Hypoxemia Endocrine crisis
- Pulmonary embolism Heart failure

Management

- Ensure oxygenation and ventilation
- Discontinue anesthetic agents, inform surgeon
- Verify blood pressure cuff size, position
- Administer fluids rapidly to expand circulating blood volume, head down position, place or replace IV cannula, consider interosseous needle (IO)
- Start inotrope infusion IV, IO:
  - Dopamine: 2–20 μg/kg min
  - Adrenaline: 0.01–1 μg/kg min
  - Milrinone: loading dose: 50 μg/kg over 10–60 min, infusion 0.25–0.75 μg/kg min
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- Review ECG for rhythm disturbances or ischemia
- Send blood sample for arterial blood gases (ABG), hemoglobin (Hb) and electrolytes
- Start vasopressor infusion:
  - Phenylephrine: dose (age 1–12 years) 5–20 μg/kg (repeat after 15 minutes to max dose of 500 μg)
  - Noradrenaline: dose (age 2–12 years) 0.1–2 μg/kg/min, titrate to effect
- Start anaphylaxis treatment if appropriate
- Administer corticosteroids for endocrine crisis
  Have a high index of suspicion for cardiac tamponade, concealed large hemorrhage or pneumothorax, in a trauma victim, if after initial fluid resuscitation and stabilization of vital signs and following rapid sequence induction and intubation, significant hypotension and fall in CO₂ trace develops.

**Acute Hypertension**

In infants and small children, hypertension is diagnosed if the systolic blood pressure is >99 percentile for age + 5 mm Hg or a 20–30% rise from baseline value.

**Causes**

Pain, light anesthesia, measurement error (faulty transducer level, small BP cuff size), medication error, other medical causes and unsuspected endocrine diseases.

**Management**

In addition to adequate analgesia and adequate depth of anesthesia, if hypertension persists, consider the following therapy:

- Beta-adrenergic blockade—
  - Esmolol 100–500 μg/kg over 5 min, then 50–200 μg/kg/min
  - Labetalol 0.2–1 mg/kg in divided doses at 10 minute intervals
  - Propranolol 10–100 μg/kg slow IV
- α₂-agonist: clonidine 0.5–2 μg/kg
- Calcium channel blockade:
  - Nicardipine 0.5–5 μg/kg/min
  - Clevidine 0.5–3.5 μg/kg/min
- D-1 agonist: Fenoldopam 0.3–0.5 μg/kg/min (maximum 2.5 μg/kg/min)
- Direct smooth muscle relaxation:
  - Sodium nitroprusside 0.5–10 μg/kg/min
  - Hydralazine 0.1–0.2 mg/kg

**Massive Hemorrhage and Transfusion**

In pediatric patients massive transfusion has variable definitions which include the following: (i) transfusion of 100% total blood volume (TBV) within 24 hours, (ii) transfusion support to replace ongoing hemorrhage of >10% TBV per minute (iii) replacement of >50% TBV by blood products within 3 hours.

**Management**

- Call for help
- Obtain additional vascular access
- Send blood samples for blood type and crossmatch, Hb/PCV, platelets, PT/PTT/INR, fibrinogen, ABG, electrolytes and lactate
- Notify blood bank of immediate massive transfusion requirements
- Transfuse in a ratio of 1:1:1 for packed red blood cells (PRBCs): fresh frozen plasma (FFP): platelets. Use a 140 micron filter for all products and blood warmer for RBC and FFP transfusion (not for platelets) and a rapid transfusion pump if available
- Use uncrossmatched O negative blood until crossmatched blood is available
- Transfuse cryoprecipitate to maintain fibrinogen >100 mg/dL
- Consider rFactor VIIa for refractory hemorrhage if above measures are corrected
- Maintain hematocrit (HCT) >21% or Hb >7 g/dL, platelet count >50,000, INR <1.5
- Monitor temperature and prevent hypothermia with active warming methods.

**Hyperkalemia**

**Causes**

Massive or old blood transfusion. Extracellular potassium concentration in stored RBC products increases with duration of storage. Transfusion of fresh red blood cells <7 days old and saline washed irradiated RBCs are recommended in neonates to avoid hyperkalemia. Other causes include cardioplegia, crush injury, burns, succinylcholine, malignant hyperthermia, acidosis and renal failure.

**Diagnosis**

ECG displaying tall peaked T waves, heart block, ventricular arrhythmias, ventricular fibrillation or asystole.
Management

Acute management includes driving K into the cells and removing the potassium from the body

- Call for help
- Stop potassium containing fluids (Ringer’s lactate, PRBCs) change to normal saline, or washed PRBCs
- If hemodynamically unstable, initiate CPR
- Hyperventilate with 100% oxygen
- Sodium bicarbonate 1–2 mEq/kg IV/IO
- Calcium chloride 20 mg/kg or calcium gluconate 60 mg/kg IV/IO
- Glucose/insulin: dextrose 0.25–1 g/kg IV and regular insulin 0.1 U/kg SC/IV
- Albuterol/salbutamol nebulization
- Terbutaline 10 μg/kg IV bolus, then infusion 0.1–10 μg/kg/min
- Furosemide 0.1 mg/kg IV.

Air Embolism

Diagnosis

Rapid fall in end-tidal carbon dioxide (ETCO₂), oxygen saturation (SpO₂) and blood pressure. This is noted more commonly during neurosurgical, head and neck and laparoscopic surgery and in the sitting position.

Management

- Ventilate with 100% oxygen
- Call for help, inform the surgeon
- Stop nitrous oxide and volatile agents
- Find entry point of air, stop source and limit further entry, irrigate surgical field with N. saline
- Check for open venous lines or air in tubing
- Turn off all pressurized gas sources (laparoscope, endoscope)
- Perform the Valsalva maneuver
- Compress jugular veins intermittently if head and neck or cranial surgery
- Position left side down, head down, once entry source controlled
- Consider: Vasopressors (adrenaline, noradrenaline), chest compressions: 100 per min (to force air through lock, even if not in cardiac arrest). If central venous line in place, attempt to aspirate air. Transesophageal echocardiography if available, can confirm the diagnosis.

Sudden Intraoperative Decrease in Electrocardiogram Amplitude

Decreased ECG amplitude can be produced by spontaneous pneumothorax.

Management

- Give 100% oxygen, turn off nitrous oxide
- If pneumothorax suspected, needle decompression
- Request for portable chest X-ray
- If pneumothorax confirmed, insert intercostal tube.

ACUTE RESPIRATORY PROBLEMS

Hypoxemia (Desaturation and/or loss of ETCO₂ trace)

In this urgent situation, follow the algorithm as described below:

Initial Response

- Give 100% oxygen, turn off inhalation anesthetic agents
- If ventilator on; check ventilator rate, volume, pressure and/or change to manual ventilation
- Confirm oxygen delivery from wall or cylinder.

Determine Cause

- Dislodged: Check endotracheal tube (ETT) position: endobronchial, kinked, not in trachea.
- Obstructed: Suction ETT: kinked, mucous plug, blood.
- Pneumothorax: Listen to breath sounds, decompress with needle.
- Equipment: Check: oxygen flow, valves, CO₂ canister, disconnections, obstructions.
- Rule out acute embolus: air, blood, fat.

Auscultate for breath sounds (bilateral sounds, chest movement, quality of breath sounds, wheezing and crepitations). Check gas analyzer and ETCO₂ line connections, oxygen saturation (SpO₂) probe, BP, perfusion. If abnormal: call for help and start CPR.

Management

If circuit leak present and bag does not fill:

- Check for disconnect from patient to machine or soda lime canister leak.
- Is the NG tube in the trachea?
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• Is the ETT/Laryngeal mask airway (LMA) cuff seal inadequate?
• Is the ETT not in the trachea?
• Is the ETT or LMA damaged?
  If above causes ruled out, change to a self-inflating ventilation bag.

*If bronchi are present with a supraglottic device in situ:*
• Suspect a light plane of anesthesia or aspiration, deepen plane of anesthesia and suction the pharynx
• If no improvement: Remove airway device, establish bag mask ventilation and re-secure airway with ETT
• If cardiac arrest, follow Pediatric Advanced Life Support (PALS) algorithm
• Support ventilation.

**Hypercarbia**
• Increase the fresh gas flow, increase minute ventilation by manual ventilation, change soda lime
• Remove the heat and moisture exchanger (HME increases dead space)
• During laparoscopic surgery, may need to reduce CO₂ flow or intra-abdominal pressure, reconfirm position of ETT (may be endobronchial).

**Elevated Airway Pressures**
• Manually ventilate to confirm high airway pressures
• Examine the patient’s respiratory system and call for help
• Check ETT, valves, connections and HME for kinking or obstruction
• Exclude light anesthesia and/or inadequate muscle relaxation (laryngospasm may occur in an un-intubated patient)
• Connect self-inflating bag directly to airway device
• Do a systematic anesthetic circuit check, Check position and patency of the airway, suction the airway device, if in doubt replace
• Review other causes: bronchospasm, pulmonary edema, pneumothorax, pneumomediastinum, hemothorax, chest wall rigidity.

**Laryngospasm**

Laryngospasm is the sustained closure of the vocal cords resulting in the partial or complete loss of the airway. It is a primitive protective airway reflex that exists to protect against aspiration but can occur in light planes of anesthesia and surgical stimulus. Laryngospasm can rapidly result in hypoxemia and bradycardia. In order to re-establish oxygenation, a clear management plan is required to avoid significant morbidity and even mortality.

**Diagnosis**
Laryngospasm may present with simple airway obstruction, regurgitation and vomiting or desaturation. Signs and symptoms of laryngospasm include inspiratory stridor which may progress to complete obstruction, increased respiratory effort, tracheal tug, paradoxical respiratory effort and oxygen desaturation with or without bradycardia.

**Management**
• Discontinue nitrous oxide, give 100% oxygen
• If pulses present, continue inhalation agent to deepen anesthetic
• Remove airway device and suction
• Apply gentle continuous positive airway pressure (CPAP) with a jaw thrust, an oral airway may help if not in place
• If IV present give succinylcholine (0.3–1 mg/kg), propofol 1 mg/kg, to break laryngospasm in a hemodynamically stable patient
• Attempt intubation if laryngospasm persists and desaturation continues and the child is flaccid.

*If the patient’s tone prevents an intubation attempt and no IV line is available*
• IM or submental atropine 0.02 mg/kg (0.1 mg minimum dose), IM, submental, or intralingual succinylcholine 2–4 mg/kg (maximum dose 150 mg)
• Chest compressions may be needed to circulate the medications if cardiac output is low. If IV or ETT access is unavailable and the child arrests, then IO access should be established immediately and the appropriate resuscitation algorithm followed
• Consider stomach deflation.

**Severe Bronchospasm**

**Causes**
Patients with bronchial asthma and chronic obstructive pulmonary disease (COPD) may have hyperreactive airway responses to mechanical and chemical irritants. A combination of constriction of bronchial smooth muscle, mucosal edema and mucous hypersecretion with plugging of smaller airways occurs. Exposure to tobacco smoke, history of atopy and recent viral upper respiratory tract infection, (URTI) all increase the risk of bronchospasm during anesthesia. Bronchospasm
Principles and Practice of Pediatric Anesthesia

during general anesthesia can present in isolation or as a component of a more serious underlying pathology such as anaphylaxis.

Clinical Suspicion and Diagnosis

Bronchospasm during anesthesia usually manifests as, bilateral expiratory wheeze, in severe bronchospasm wheeze and breath sounds may be quiet or absent. With IPPV, increased peak airway pressures, reduced tidal volume, rise in end tidal CO₂ occur. The capnogram may show a delayed rise ‘shark fin’ appearance (representing expiratory obstruction) and prolonged expiration. Positive pressure ventilation delivered before completion of exhalation can lead to ‘breath stacking’ or intrinsic PEEP, which increases intrathoracic pressure, reduces venous return and cardiac output.

Causes of Wheezing Under Anesthesia

These include partial obstruction of the tracheal tube with mucous plug and tube malposition (e.g. kinked, abutting the carina, bronchial intubation), pulmonary edema, aspiration of gastric contents, pulmonary embolism, tension pneumothorax and foreign body (e.g. a tooth) in the tracheobronchial tree.

Causes of Increased Peak Airway Pressure During IPPV

These include excessive tidal volume, high inspiratory flow rates, small diameter tracheal tube, endobronchial intubation, tube kinked or blocked, obesity, head-down position, pneumoperitoneum, tension pneumothorax and bronchospasm.

Management

Suspected bronchospasm during anesthesia should be assessed and treated promptly. Ongoing management should address the underlying cause.

- Give 100% oxygen, stop all potential precipitants and increase depth of anesthesia and call for help early
- Hand ventilate, check tube placement
- Exclude mechanical obstruction or occlusion of the breathing circuit
- Provide salbutamol and ipratropium bromide puffs (2.5–5 mg every 20–30 min via the breathing circuit)
- If ventilating with an LMA consider replacing with an ETT
- Drugs and dosages (Table 1).

Intravenous salbutamol should be considered when there is poor response to inhaled β₂ agonists. Aminophylline should only be used in severe life-threatening exacerbations that fail to respond to conventional therapy. Magnesium sulfate is safe in asthma and recommended for refractory bronchospasm. Adrenaline should be promptly used if anaphylaxis is suspected in doses of 1–10 μg/kg, depending on the response.

- Ventilation strategies include: a longer expiratory phase to allow complete exhalation and prevent air trapping. Long inspiration time to allow adequate gas exchange, intermittent disconnection and low PEEP to reduce hyperinflation. Hand ventilation with permissive hypercapnoea may be required to avoid high pressure ventilation.
- Plan for ICU admission.

Aspiration

Causes

Aspiration can occur with inadequate fasting periods, delayed gastric emptying or gastric distension during mask ventilation and occurs at the time of induction or in the intraoperative period, during sedation, general anesthesia with a face mask, LMA, following esophageal intubation or change of ETT intraoperatively. Light or inadequate anesthesia and increased intra-abdominal pressure due to coughing, can also result in aspiration. Aspiration may

Table 1: Drug dosages in management of bronchospasm

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Bolus dose IV Age &lt;2 years</th>
<th>Bolus dose μg/kg IV Age 2–18 years</th>
<th>Infusion rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adrenaline</td>
<td>0.1–1.0 μg/kg</td>
<td>0.1–1.0 μg/kg</td>
<td>0.1 μg/kg/min</td>
</tr>
<tr>
<td>Salbutamol</td>
<td>5 μg/kg</td>
<td>15 μg/kg(max 250 μg)</td>
<td>100–300 μg/kg/h</td>
</tr>
<tr>
<td>Aminophylline</td>
<td>5–7 mg/kg over 15 min</td>
<td></td>
<td>0.5 mg/kg/h</td>
</tr>
<tr>
<td>Magnesium Sulfate</td>
<td>40–50 mg/kg over 20 min (max dose 2 g)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hydrocortisone</td>
<td>4–8 mg/kg</td>
<td>4–8 mg/kg</td>
<td>Maximum: 250 mg; 2 mg/kg per dose every 6 hours</td>
</tr>
</tbody>
</table>

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go undetected clinically but can be suspected when bile or gastric contents are observed in the face mask or airway or when rapid oxygen desaturation occurs.

Aspiration is likely to occur when there is a failure to recognize risk factors and not using a modified rapid sequence induction when indicated and during maintenance of anesthesia in patients with risk factor when an LMA is in situ. Anxiety, opioids or gastrointestinal pathology and any factor which increases intra-abdominal pressure or prolonged gastric emptying time should raise suspicion. Second generation supraglottic airway devices with a channel should be preferred to a classic LMA. It is preferable to secure the airway with an ETT for all emergencies with good assistance at hand.

**Management**
- Call for help, inform the surgeon
- Position the child head down and lateral, suction the pharynx. Intubate and suction via ETT. Ventilate with 100% oxygen
- Empty the stomach with wide bore stomach tube
- If the patient is asymptomatic, the chest X-ray is clear and SpO₂ is normal, mild aspiration usually resolves without specific treatment
- If aspiration is severe or with particulate matter suctioned, proceed with bronchoscopy and lavage. Defer surgery, when possible if significant aspiration with persistent desaturation
- Consider ICU admission.

**Airway Leak Around the ETT following Change in Position, e.g. Prone Position**

**Management**
- To reduce intragastric pressure insert orogastric tube and suction the stomach
- If the situation does not improve and ventilation is compromised, reposition supine, do a check laryngoscopy to rule out partial extubation or expulsion of cuff above the glottis. If required reintubate.

**Difficulty in Extubation**

**Cause**
In an attempt to rapidly deflate the ETT cuff, at times the pilot balloon and valve assembly are pulled off the pilot tube prior to extubation in an awake and alert patient. In doing so the pilot tube is stretched, occluding the stump of the tubing that is still attached to the inflated cuff. The cuff remains inflated and removal of the ETT is not possible. This practice of deflating the tracheal cuff should be strongly discouraged.

**Management**
In this situation reassure the patient verbally, encourage to breathe through the tube. Cut the pilot tube proximal to the obstruction, deflate the cuff with a needle and syringe past the occlusion in the pilot tube.

**Airway Fires**

**Cause**
Fire in tracheal tube, circuit, canister.

**Management**
- Call for help
- Disconnect breathing circuit and stop all gas flow (oxygen and nitrous oxide)
- Pour saline into ETT, remove ETT
- Remove sponges and other flammable materials from airway
- Reintubate and re-establish ventilation
- If intubation difficult, obtain a surgical airway
- Consider bronchoscopy to assess thermal injury, look for tracheal tube fragments and remove residual material
- Inspect all equipment.

**LOCAL ANESTHETIC TOXICITY**

**Clinical Diagnosis**
The first signs of toxicity may be electrocardiogram changes with prolonged PR interval, progressive bradycardia and cardiac conduction block leading to hypotension, decreased contractility and altered consciousness, seizures.

Local anesthetic toxicity may be more difficult to recognize in children than in adults because, unlike adults, children are usually under general anesthesia during injection of the local anesthetic. The central nervous system changes (agitation, confusion, twitching, and seizures) would likely be masked under general anesthesia, particularly if the patient has received muscle relaxants.

**Management**
- Stop local anesthetic injection and call for help
- Ask for intralipid kit
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- Secure airway and ventilation, give 100% oxygen, monitor ECG, BP, SpO₂ and establish IV access
- **Seizure treatment:** midazolam 0.05–0.1 mg/kg IV, or thiopentone 3–5 mg/kg IV bolus, additional bolus doses of 1–2 mg/kg every 3–5 min until a clinical response is achieved to a maximum of 10 mg/kg. A continuous infusion of 0.2–0.3 mg/kg/min till the seizures are controlled, or propofol 0.5 mg/kg.
- If cardiac instability, start CPR. Initial doses of adrenaline for resuscitation should be reduced as it can decrease the efficacy of lipid emulsions. The recommended antiarrhythmic drug is amiodarone.
- Start Intralipid 20% IV: Bolus dose 1.5 mL/kg over 1 minute. Start an infusion at 0.25 mL/kg/min. Repeat bolus every 3–5 minutes to a maximum dose of 3 mL/kg, till circulation is restored. Increase infusion rate to 0.5 mL/kg/min if BP remains low. Maximum total dose: 10 mL/kg over first 30 minutes.
- Consider cardiopulmonary bypass to support circulation as these patients may frequently re-arrest until the local anesthetic is metabolized.

**GENERALIZED SEIZURES**

**Differential Diagnosis**
Local anesthetic toxicity, undiagnosed epileptic, hypoglycemic coma.

**Management**
Seizures may be short lived and resolve spontaneously within a few minutes. The treatment regime starts with airway management. If the seizure continues beyond 5 minutes.
- Start CPR
- If airway compromised or respiratory depression, consider early intubation
- Secure IV, send venous sample for blood sugar and electrolytes analysis
- If hypoglycemia, glucagon IV, IM, SC; 0.5 mg for <12 year of age, 1 mg for >12 years age or 10–30 μg/kg. If glucagon is not available, IV dextrose should be administered slowly over several minutes; dextrose 10–25% at 200–500 mg/kg to reverse the hypoglycemia (dextrose 10% is 100 mg/mL). Rapid administration, or excessive concentration of dextrose, i.e. dextrose 50%, may result in an excessive rate of osmotic change
- Midazolam 0.5 mg/kg, lorazepam 0.1 mg/kg IV or rectal diazepam 0.5 mg/kg
- If seizure continues 10 minutes after administering above drugs:
  - Administer: Phenytoin 20 mg/kg IV over 20 minutes. If the child is already receiving phenytoin, give a smaller dose of 5 mg/kg over 5 minutes.
  - If unsuccessful, administer phenobarbital 10–20 mg/kg IV (not to exceed 700 mg IV) Phenobarbital may be used in infants before phenytoin; be prepared to intubate and closely monitor hemodynamics and support blood pressure. If seizure continues 20 minutes after starting phenobarbitone or phenytoin, start an infusion.
  - If IV access cannot be achieved promptly, IM fosphenytoin or rectal paraldehyde can be used with persistent seizures, proceed to a rapid sequence induction with thiopentone.

**ANAPHYLAXIS**

Anaphylaxis is a severe, acute and potentially life-threatening condition, often in response to an allergen. Patients experiencing anaphylaxis can present with cutaneous, respiratory, cardiovascular or gastrointestinal manifestations.

**Clinical Diagnosis**
Manifestations of anaphylaxis include hypotension, rash, bronchospasm, pulmonary edema, pulmonary hypertension, arrhythmias, increased peak inspiratory pressures, hypoxemia, stridor, hives and angioedema.

**Common Causative Agents**
Neuromuscular blockers, latex, chlorhexidine, plasma substitutes, antibiotics, morphine-like substances.

**Management**
- Call for help, supplement 100% oxygen
- Remove suspecting trigger, e.g. if latex is suspected, thoroughly wash area or stop drug
- Ensure adequate ventilation, oxygenation, preferably hand ventilate
- If hypotensive, turn off anesthetic agents
- Restore intravascular volume by rapid infuse of normal saline or Ringer’s lactate (10–30 mL/kg IV)
- Adrenaline 1–10 μg/kg IV, up to 0.5 mg per dose every 20 minutes as needed to restore BP, 10 μg/kg dose IM, if no IV. Adrenaline infusion 0.02–0.2 μg/kg/min
- Adjuvants:
  - Beta agonists for bronchoconstriction: Albutarol 4–10 puffs
Chapter 35: Acute Complications During Anesthesia

- Corticosteroids: Methylprednisolone 2 mg/kg, maximum 60 mg or hydrocortisone 2 mg/kg, maximum 100 mg
- $H_1$ antagonist: Diphenhydramine 1 mg/kg IV, maximum 50 mg
- $H_2$ antagonist: Famotidine 0.25 mg/kg IV or ranitidine 1 mg/kg

- For laboratory confirmation of anaphylaxis, send mast cell tryptase level within 2 hours of the event and plasma histamine level within 30 minutes of the event.
- Perform allergy testing later.

**MALIGNANT HYPERTERMHA**

This manifests as rapid rise in temperature usually triggered by an anesthetic and is potentially lethal. It is an inherited myopathy due to a genetic mutation. In children with the muscle abnormality, muscle cells have an abnormal protein on their surfaces. The protein does not affect muscle function significantly. That is, until the muscles are exposed to a drug that can trigger a reaction.

When a person with this condition is exposed to one of these drugs, calcium stored in muscle cells is released. The muscles contract and stiffen at the same time, there is a dramatic and dangerous increase in body temperature (hyperthermia). Malignant hyperthermia usually occurs during or after surgery or wherever anesthetic medications are used. Potent inhalation anesthetic agents are the main triggers. Halothane, desflurane, sevoflurane and isoflurane can cause florid malignant hyperthermia (MH) reactions. Other drugs which may trigger MH include succinylcholine, serotonergic drugs.

**Clinical Diagnosis**

A dramatic rise in body temperature, sometimes as high as 40°C, rigid or painful muscles, especially in the jaw, flushed skin, sweating, an abnormally rapid or irregular heart rate, rapid respiration or uncomfortable breathing, brown-colored urine, very low blood pressure (shock).

**Management**

- Call for help, stop procedure, request for chilled saline.
- Stop volatile anesthetic, change to non-triggering anesthetic like propofol, ketamine
- Hyperventilate 2–4 times the patient's ventilation to reduce CO$_2$
- Mix dantrolene 20 mg vial with 60 mL sterile water, administer 2.5 mg/kg IV every 5 minutes until symptoms resolve
- Sodium bicarbonate 1–2 mEq/kg IV to maintain pH >7.2
- Rapid cooling, if temperature >39°C: by nasogastric tube lavage with cold water, cool mist and fans, cooling blankets, cold packs or external ice application in the groin, neck regions and forehead, cold saline IV infusion. Stop cooling if <38°C
- Treat hyperkalemia: IV calcium gluconate 30 mg/kg or calcium chloride 10 mg/kg.
- Dextrose 0.5 g/kg (maximum 50 mL D50), regular insulin 0.1 U/kg IV (max 10 U) and sodium bicarbonate 1–2 mEq/kg IV
- Treat dysrhythmias: do not use calcium channel blocker
- Send arterial or venous blood gas sample, electrolytes, serum calcium, serum/urine myoglobin, coagulation profile
- Insert urinary catheter, monitor urine output
- Inform ICU.

**TRANSFUSION REACTIONS**

Blood transfusion reactions typically occur when the recipient's immune system launches a response against blood cells or other components of the transfused product. These reactions may occur within the first few minutes of transfusion (acute reaction) or develop hours to days later (delayed reaction). If red blood cells are destroyed, the reaction may be classified further as hemolytic and all other types of reactions are broadly classified as nonhemolytic.

Safe practice to prevent these reactions include accurate collection of pre-transfusion blood samples for typing and cross matching, before hanging the blood product, double-check the patients identification and verify the actual product and check the unit to be transfused against patient identifiers. Blood products should be infused with normal saline solution only, using tubing with filters.

**Management**

1. Stop transfusion and disconnect product and IV tubing
2. Call for help
3. Infuse 0.9% saline through a new infusion set
4. Examine blood product ID, determine if correct patient, send blood to blood bank

**Management of Hemolytic Reaction**

- Furosemide 0.1 mg/kg
- Mannitol 0.5 g/kg (2 mL/kg of 25% mannitol)
Dosing being administered. In documentation may also result in inadvertent repeat effect may be excessive and potentially lethal. Omission from retention of injected medication in the dead space of IV tubing. Additional doses may be then given and the effect may be excessive and potentially lethal. Omission in documentation may also result in inadvertent repeat dosing being administered.

**Anaphylactic Reaction**
- Stop transfusion
- Support airway and circulation
- Adrenaline 10 μg/kg IV
- Diphenhydramine 1 mg/kg IV
- Hydrocortisone 2–5 mg/kg IV
- Maintain intravascular volume.

**MEDICATION RELATED PROBLEMS**

Administration of wrong drug, wrong dose, wrong route, or right drug inadvertently repeated, can lead to drug related acute side effects in children during anesthesia. The most common drugs that cause problems include analgesics and antibiotics and local anesthetic drugs. Apnea, bradycardia, hypotension and hypotonia may arise from retention of injected medication in the dead space of the IV tubing. Additional doses may be then given and the effect may be excessive and potentially lethal. Omission in documentation may also result in inadvertent repeat dosing being administered.

**REFERENCES**

11. Critical events check list on www.pedsanesthesia.org
23. www.asthma.org.uk
SECTION 5

Anesthetic Techniques

Chapter 36: Vascular Access in Infants and Children
Chapter 37: Ultrasound-guided Regional Blocks
INTRODUCTION

Obtaining a vascular access is a part of the daily routine for pediatric anesthesiologists. Nevertheless, it can still be challenging in some of the pediatric patients. Following are some of the reasons, why vascular access can be more difficult in pediatric patients:

- Less cooperative patient
- Smaller size of vessels
- More superficial and mobile vessels
- Anatomical variations.

Hence, we need to train and equip ourselves with the necessary knowledge and skill. In this chapter, we will go through the various options available, techniques and limitations. The choice of site and the type of vascular access device depends on the type of medication to be administered and duration of therapy.

PERIPHERAL VEIN CANNULATION

- **Introduction:** Peripheral venous access is the most preferred and commonly used method for drug and fluid therapy, venous sampling for investigations, anesthesia, resuscitation and blood product administration. Transillumination technique may be used in neonates or infants to improve visualization of veins in chubby and dark skinned children. A cold light source like torch or fiber-optic scope and a low-lighted room is desirable for success using this technique.

- **Indications:** It is preferred when the anticipated IV therapy is for less than a week and does not involve medications with high osmolarity. Peripheral catheters may need to be replaced every 72–96 hours or earlier if there is evidence of thrombophlebitis.

- **Sites:** Dorsum of hand, cubital fossa, dorsum of foot, medial aspect of ankle, and scalp are the common sites.

- **Contraindications:** Should not be attempted where skin integrity is doubtful as in cellulitis, burns or severe edema.

- **Complications:** Thrombophlebitis and extravasation at the insertion site are the most common complications of the peripheral line. They need to be detected promptly and the line should be replaced immediately. Else, may lead to grievous complication like necrosis requiring debridement and skin grafting. Figure 1 depicts such a complication.

- **Recent developments:** Ultrasound-guided venous access can be used to improve the accuracy of catheter placement particularly in infants and obese children. Infra red devices have been recently available for enhanced vein visualization particularly in dark skinned and obese children. Even though these devices improve visualization, they may not improve success to cannulation, and more experience is required with these devices.

ULTRASOUND-GUIDED VASCULAR ACCESS

- **Introduction:** Ultrasound is being increasingly used and becoming the standard of care, for a variety of vascular access in adults as well as children.
Familiarity with the equipment, knowledge of the relevant anatomy and the basic principles of vascular puncture should be learnt to use ultrasound for any vascular access.

- **Advantages:**
  - Visualization of anatomy helps to identify the vessel accurately even in the presence of anatomical variations, improving the chances of successful cannulation.\(^7\)
  - Ultrasound helps optimization of puncture point as well guides the direction of needle advancement.
  - Real-time ultrasound assessment helps confirm placement of needle, guidewire and catheter in the vessel.
  - It helps reduction of complications due to inadvertent puncture of surrounding anatomical structures.\(^7,8\)

- **Disadvantages:**
  - Extra equipment is needed.
  - More time-consuming,\(^9\) especially during the learning curve.
  - Maintaining asepsis requires effort and expense.
  - Complex hand–eye coordination is required for real-time imaging.
  - Sometimes, ultrasound artifacts may complicate the technique.

- **Principles of ultrasound-guided technique:**
  - Ultrasound should be used in 2D mode. Doppler color may be added to further confirm the presence of flow in a certain anatomical structure.
  - Longer catheters are preferred for ultrasound-guided technique to allow easy maneuverability.
  - High frequency linear array probe should be used.
  - The orientation marker on the ultrasound probe should correspond to the same side of the patient.
  - For the single operator, hold the ultrasound probe in nondominant hand and needle in the dominant hand.

- **Techniques:**
  - Static ultrasound-guided technique—where ultrasound is used to mark the anatomy and then the vessel puncture is blind. This may be done in very small infants or neonates where the equipment size may have limitations.
  - Dynamic or real-time ultrasound technique—where ultrasound is used to visualize the needle tip as the puncture is made and catheter as it is advanced into the vessel. This technique is safer, and hence, preferred over static technique.\(^10\)
  - This technique can be ‘single operator’ or ‘two operators’, based on the convenience and experience. Figure 2 depicts dynamic ultrasound technique for central venous access.

- **Orientations:**
  - Long axis (LAX) / ‘in-plane’ orientation—LAX view may be difficult to perform in smaller areas and the learning curve is steep. LAX view is preferred whenever artery lies immediately posterior to the vein\([\text{e.g. as in carotid artery lying posterior or posteromedial to internal jugular vein (IJV)}]\), intended to be cannulated. This technique prevents double puncture of the vein, as long length of the needle including its tip is visible.
PERIPHERALLY INSERTED CENTRAL CATHETER INSERTION

- **Introduction:** Peripherally inserted central catheter (PICC) lines are central lines from a peripheral route—so they have all the advantages of central line with a lower incidence of infective complications than the nontunneled central lines.\(^{12}\)
- **Indications:** PICC line is preferred, when a central venous access is desired for administering hyperosmolar medications or medications in a home care setup for longer duration and in patients with limited venous access.
- **Contraindications:** Doubtful skin integrity over the intended vein, preexisting thrombosis in the vein and when infusion therapy is expected to last for more than a couple of months.
- **Sites:** Arm veins—antecubital, brachial and cephalic are very frequently used. In infants, saphenous vein may be used with the tip of the catheter in the inferior vena cava (IVC) above the diaphragm. In neonates, axillary vein has been used with success.\(^{13}\)

**Procedure description:** There are two techniques of PICC line insertion and separate kits are available for each.

- **Peel away cannula:** In this technique, a cannula is placed in the desired vein, needle is removed and the PICC catheter is advanced through the cannula. After the desired length of catheter is advanced, the cannula is pulled out and peeled away. The catheter is then fixed appropriately.

- **Modified Seldinger technique:** A guidewire is advanced through a needle that enters the desired vein. Needle is removed and introducer sheath with dilator is advanced over the guide wire. Catheter is then negotiated in the sheath after removing the dilator over guidewire.

With either technique, correct placement of the catheter needs to be identified fluoroscopically or with a postprocedure X-ray. Figure 4 shows PICC line in-situ.

**Complications:** Vein thrombosis,\(^{14}\) phlebitis, catheter migration, kinking or breaking, local inflammation and infective complications have been noted.

EXTERNAL JUGULAR VEIN CANNULATION

- **Introduction:** External jugular vein cannulation is not frequently done and has limited indications. Its proximity to the heart allows the medications to reach the heart early even in circumstances of low flow.

- **Indications:** External jugular vein cannulation may be an option in patients with poor peripheral veins or thrombophlebitis of the limb veins, undergoing short duration surgical procedures or IV therapy. External jugular vein may also be used by some for central venous access with around 90% success rate.\(^{15}\)
• **Anatomy:** The external jugular vein runs obliquely on each side of the neck roughly from the angle of mandible towards the middle of clavicle, before it perforates the deep fascia and opens into the subclavian vein.

• **Limitations:** External jugular vein cannulation may require the head to be turned to the opposite side for adequate flow. This limits the use of this access for longer duration. Also, it often has valves, which may pose difficulty in threading the catheter.

• **Contraindications:** Polytrauma with neck or spine injuries precludes the use of this site for venous cannulation.

• **Procedure description:** The child needs to be well sedated or preferably anesthetized. The anesthesiologist should be at the patient’s head end. Head needs to be turned to the opposite side and about 30° head low, facilitates filling up of the vein and easy visualization. Firm pressure on the clavicular side of the vein may further accentuate the vein. The proximal end of the vein needs to be stretched firmly to prevent slipping of the vein as the cannulation is performed. Once the vein is punctured, the catheter is threaded into the vein.

• **Complications:** This vein can generate significant negative pressure particularly during deep breathing. Hence, there are chances of air embolism during the placement of canula in this vein as well as later during the use. Thrombophlebitis and extravasation can occur and have to be monitored for. Intrathoracic fluid accumulation and mediastinal tamponade have also been reported.

**CENTRAL VENOUS CATHETERIZATION**

• **Introduction:** Central venous catheter is placed in one of the central veins in neck, thorax or groin—e.g. IJV, subclavian vein and femoral vein. The central venous catheter can be tunneled or nontunneled, based upon the indication and the duration of catheter dwell time required. Different products are available for each. This section refers to nontunneled catheters and tunneled catheters are discussed in the Table 1.

• **Indications:**
  - Need for frequent blood sampling
  - Monitoring of central venous pressure.

• **Contraindications:**
  - Infection over desired insertion site
  - Thrombosis in the desired vein
  - Uncorrected coagulopathy
  - Tumor obstructing the intended vein.

• **Sites:**
  - Internal jugular vein
  - Subclavian vein
  - Femoral vein.

• **Practice guidelines:** Every unit should develop their own protocol for insertion, maintenance and removal of central venous lines based on evidence base and good practice guidelines. American Society of Anesthesiologists (ASA) task force in 2012 has detailed the practice guidelines for central line placement. Though most of the evidence is in adult patients, it can be extrapolated to children. Chlorhexidine is preferred for skin preparation except in neonates, where povidone iodine or alcohol may be used.

  - Carina can be used as a radiologic landmark for the central venous line tip position in children.

**Internal Jugular Vein Cannulation**

**Procedure description:** Lie the child supine in about 15° Trendelenburg position. Liver compression may be used to assist filling up of IJV further. A small shoulder roll may be placed to extend the head and neck. Head should be turned towards the opposite side.

**Right IJV is preferred over left IJV as—**

a. Left dome of lung is placed higher

b. Thoracic duct empties in left IJV

c. The course of the right IJV towards the heart is straighter than that of left IJV.

Use of static or real-time ultrasound is preferred to locate and cannulate IJV. Long-axis view is preferred, especially when the internal carotid artery lies posterior to the IJV. There are three traditional approaches—anterio, central and posterior for IJV cannulation, as depicted in the Figure 5.

**Anterior Approach**

The needle entry point is on the anterior border of sternocleidomastoid muscle at the level of cricoid cartilage. The direction is posterior and lateral towards ipsilateral nipple. This approach is less commonly used.
### Table 1: Comparison of various vascular access devices

<table>
<thead>
<tr>
<th>Vascular access device</th>
<th>Duration of use</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peripheral intravenous (IV) cannula</td>
<td>Few days</td>
<td>Can be placed by nurse</td>
<td>Shorter duration</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Less cost</td>
<td>Cannot use highly osmolar medications</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Less complications</td>
<td>Thrombophlebitis</td>
</tr>
<tr>
<td>Peripherally inserted central catheter (PICC)</td>
<td>Few weeks</td>
<td>Requires training, but can be taught to a nurse</td>
<td>Costlier than a peripheral canula</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Can be kept for days or weeks</td>
<td>Thrombotic and phlebitic complications</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Both for peripheral and central venous indications</td>
<td>Needs X-ray for documentation of the tip</td>
</tr>
<tr>
<td>External jugular vein cannula</td>
<td>Few days</td>
<td>Requires training, but can be taught to a nurse</td>
<td>Needs restraining the child when not under anesthesia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Quicker to access than a central line</td>
<td>Temporary access for a couple of hours only</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Used in an emergency till better access is secured</td>
<td></td>
</tr>
<tr>
<td>Nontunneled central venous catheter</td>
<td>Few days</td>
<td>Short-term access for vasopressors and CVP measurement</td>
<td>Needs to be placed by a doctor</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Can use high osmolarity medications</td>
<td>Needs strict asepsis and general anesthesia in younger kids</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Cannot be used longer than 10–14 days</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Risk of infections</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Needs X-ray documentation</td>
</tr>
<tr>
<td>Tunneled central venous catheter</td>
<td>Few weeks to few months</td>
<td>Long-term access for antibiotics, nutrition or chemotherapy</td>
<td>Needs to be placed by a doctor</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Less thrombogenic than nontunneled counterparts</td>
<td>Needs strict asepsis and general anesthesia in younger kids</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Less infectious complications</td>
<td>Needs fluoroscopy for placement</td>
</tr>
<tr>
<td></td>
<td></td>
<td>More cosmetic and less lifestyle restriction</td>
<td>Costlier than nontunneled counterparts</td>
</tr>
<tr>
<td>Implantable port</td>
<td>Few months to few years</td>
<td>Long-term access for antibiotics, nutrition or chemotherapy</td>
<td>Needs to be placed as well as removed by a doctor</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Less thrombogenic</td>
<td>Needs strict asepsis and general anesthesia in younger kids</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Less infectious complications</td>
<td>Needs fluoroscopy for placement</td>
</tr>
<tr>
<td></td>
<td></td>
<td>More cosmetic and less lifestyle restriction</td>
<td>Most expensive</td>
</tr>
<tr>
<td>Intraosseous needle</td>
<td>Few hours to few days</td>
<td>Requires training, but can be taught to a nurse</td>
<td>Needs to change into another vascular access as soon as feasible</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Useful in emergency when other accesses seem poor</td>
<td>Risk of osteomyelitis</td>
</tr>
<tr>
<td>Arterial line</td>
<td>Few days</td>
<td>Accurate measurement of blood pressure in major surgeries and sepsis</td>
<td>Risk for arterial thrombosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Frequent sampling for arterial blood gas is feasible</td>
<td>Cannot be kept longer than a few days</td>
</tr>
</tbody>
</table>

### Central Approach (Called as Anterior Approach by Many in the Literature)

The needle entry point is at the apex of the triangle (Sedillot's triangle) formed by the two heads of sternocleidomastoid muscle, sternal head and the clavicular head. The direction is towards the ipsilateral nipple. This approach is preferred for tunneled catheters.

### Posterior Approach

The needle entry point is where the external jugular vein crosses the posterior border of sternocleidomastoid muscle. The direction should be towards the sternal notch or the ipsilateral sternoclavicular joint. This is the most commonly practiced blind technique.

### Complications:

Carotid artery puncture, pneumothorax, hydrothorax, arrhythmias and air embolism. Kinking of
stiffer catheters like dialysis catheters may occur from IJV approach as depicted in Figure 6.

**Subclavian Vein Cannulation**

*Procedure description:* Right subclavian vein cannulation is preferred as the dome of pleura is higher on left side. Slight Trendelenburg position is preferred with a small roll between the two scapulae, to open up the space between the clavicle and first rib. The puncture point is a little lateral and inferior to the junction between the middle third and lateral third of the clavicle. The needle is advanced below the clavicle and directed towards the suprasternal notch. If the needle hits the first rib, the entry point may be moved more inferior and lateral.

*Complications:* Incidence of pneumothorax is higher than IJV cannulation. Other complications reported are subclavian artery injury, arrhythmias and air embolism.

**Femoral Vein Cannulation**

*Procedure description:* The child lies supine or a little reverse Trendelenburg. The desired leg is abducted and externally rotated. A small pillow may be placed under the hip joint to make the joint taut and stabilize the femoral vessels. Static or real-time ultrasound may be used to locate and puncture the femoral vein. Short-axis view is preferred. Femoral vessels run below the inguinal ligament, roughly at the midpoint of anterior superior iliac spine and pubic symphysis. Femoral arterial pulse can be palpated. Femoral vein lies medial to the femoral artery and the femoral nerve lies lateral. The puncture point should be at least 1–2 cm below the inguinal ligament and in the direction of the expected vein course.

*Complications:* Infectious complications are the highest with this site and hence, avoided whenever feasible. Other complications are deep vein thrombosis, injury to femoral artery, A-V fistula, femoral nerve injury.

**TUNNELED CENTRAL VENOUS CATHETERIZATION**

- **Introduction:** Tunneled central venous catheters are preferred over nontunneled catheters when the duration of treatment is longer. There is a cuff in the tunneled part of the catheter, which allows tissue ingrowth. This not only helps secure the catheter in place, but also it seals and prevents bacterial growth along the length of the catheter proximal to the cuff. This allows these catheters to be kept for a longer duration.
- **Advantages:** Tunneled central venous catheters offer the advantage of reduced infectious complications as compared to their nontunneled counterparts.\(^\text{12}\)
- **Indications:** Chemotherapy, nutrition, long-term antibiotic therapy, frequent blood sampling, frequent transfusions or dialysis.
- **Contraindications:** Coagulopathy is a relative contraindication and needs to be corrected before attempting placement. INR <1.5 and platelet count >50,000 are aimed by some.\(^\text{22}\) Skin over the desired insertion site should be healthy.
- **Sites:** Subclavian vein and IJV are the preferred sites for tunneled catheters. The exit port is often planned in the infraclavicular area as there is lesser restriction of mobility and can be well covered by clothing. Tunneled catheters may also be placed in femoral vein in sick children and they are tunneled on the thigh.\(^\text{23}\)
- **Types:** Hickman and Broviac are the two main types of tunneled catheters. Both have soft silicone catheters, which are less traumatic and less thrombogenic allowing longer duration of use. Broviac catheters are single lumen and being smaller in size, are often preferred in children. Sizes available are 2.7 Fr to 6.6 Fr only. Hickman catheters are available as single, double or triple lumen and vary from 9.6 Fr to 12.5 Fr to suit all patient sizes and needs.
- **Procedure description:** These catheters are usually placed in the operation theater and under local or general anesthesia. Antibiotic prophylaxis is administered. The venous access is performed using modified Seldinger’s technique in the desired vein followed by the dilator sheath insertion. Tunneling of the catheter is performed using a tunneling device in...
Chapter 36: Vascular Access in Infants and Children

the kit. The catheter is threaded into the sheath after cutting at a desired length calculated fluoroscopically during the procedure, so that the tip of the catheter lies at the sino-atrial junction. The dacron cuff should lie in the tunnel closer to the exit port. The tunnel direction should allow a smooth curve of the lay of the catheter to prevent kinking and obstruction. Figure 7 depicts Hickmann’s catheter inserted via subclavian approach.

- **Complications:** Immediate complications of these catheters are similar to central venous lines placed in the respective veins. Delayed complications include infections and thrombosis of the veins.

**PORT IMPLANTATION**

- **Introduction:** Totally implantable vascular access devices also called ports have been in use for more than three decades. Ports if handled carefully are associated with the lowest infection rates and hence, can be maintained for years allowing permanent access. Ports are the most cosmetic option and associated with least interference of day-to-day activities. Port placement is done in the operation theater and under general anesthesia. The children are evaluated before procedure and complete blood picture, coagulation screen and viral screening are ordered. The side and site of port implantation is planned in consultation with the treating oncologist.

- **Indications:** The usual indications are chemotherapy and long-term nutrition support.

- **Contraindications:** Coagulopathy is a relative contraindication and needs to be corrected before planning port implantation procedure. Skin over the intended puncture and the port pocket should be healthy. Ports cannot be placed in neonates and technically difficult in infants due to the size of the port.

- **Sites:** Central venous access sites for ports are IJV and subclavian vein with the port pocket in the infraclavicular area. Basilic vein has also been used with port pocket in the arm.

- **Types:** Ports are available in various sizes and shapes. Catheters could be silicone or polyurethane and the port body could be titanium metal or plastic. Some ports are MRI compatible and others can be used for power infusions (where it can withstand 5 mL/s of contrast medium injection during contrast enhanced CT scan procedures). While most ports are single chambered, double chambered ports are available too. Ports have a self-sealing silicone diaphragm, which allow punctures more than a few hundred times. A noncoring side holed Huber needle should be used to reduce the damage to the diaphragm with each puncture.

- **Procedure description:** Antibiotic prophylaxis should always be used for this procedure. Figure 8 depicts chemoport set ready for insertion. Venous access and tunneling is similar to the tunneled catheters and performed under fluoroscopy. Gentle curve of the silicone catheter should be maintained. Port pocket is created in the infraclavicular area, which hosts the port body. The tunneled catheter is secured to the port with a locking device as per the manufacturer’s instructions. Post procedure chest X-ray should be done for documentation of the correct placement.
INTRAOSSEOUS NEEDLE PLACEMENT

- **Introduction:** Our body has a network of noncollapsible veins in our bone marrow, which can be accessed in times of emergency. This makes intraosseous access, the most preferred route in an emergency, when regular venous access is unavailable or appears difficult. In the newborn, this route is found to be faster than accessing the umbilical vein.

- **Indications:** It is mostly used in Code Blue events or polytrauma resuscitation, till another stable venous access can be secured. Earlier, this was recommended only in children less than 6 years due to thinner bone and larger marrow cavity, but now can be used in older children and adults too.

- **Contraindications:** Loss of skin integrity or infection over the desired area, bone diseases like osteogenesis imperfecta, osteopetrosis, etc.

- **Sites:** The most preferred site is proximal tibia followed by distal femur, distal tibia, proximal humerus, iliac crest and sternum.

- **Uses:** This route can be used to infuse fluids, drugs and blood products too. It can also be used to draw samples for blood chemistry and acid–base status.

- **Procedure description:**
  - **Proximal tibia**—Support the knee with a folded towel under it. Palpate the tibial tuberosity and select a flat surface slightly (2 cm) caudal and medial to it. Apply skin antisepsis and infiltration of local anesthetic if patient is awake. Direct needle perpendicular to the plane of bone or a slight caudal tilt of 10–15° may be given to avoid injury to growth plate. Holding the needle close to the skin, firm pressure with twisting movement...
should be done till a loss of resistance is felt. Aspiration of bone marrow is diagnostic, but a free flow without local swelling may be considered adequate too.

- **Distal femur**—Maintain similar position to the above and feel the epicondyles of femur. The needle insertion point should be 1–3 cm above the line joining epicondyles, in anterior midline. The direction should be slightly cranial, away from growth plate. Rest of the procedure is same.

**Complications:** Misplacement of the intraosseous needle and its complications like extravasation, tissue edema, necrosis, compartment syndrome are early complications. Late complications like cellulitis, osteomyelitis, fractures and growth plate injuries are possible.

**Learning points:** Bone marrow consists of non-collapsible network of vasculature, which can be used even in severe hypovolemia.

**Practicalities:** Dedicated intraosseous needles are available in the market like Cook’s intraosseous needle, EZ-IO, bone injection gun, etc. But, in an emergency, options like bone marrow aspiration needle or wide bore spinal needle may also be used. Figure 12 depicts bone marrow aspiration needles.

**Points to remember:** Intraosseous needle should be removed once another stable access is available. Ideally, within 3–4 hours, though it may be used for 72–96 hours. Remember that longer the intraosseous needle stays, higher the chances of complications.

### ARTERIAL CANNULATION

**Introduction:** Securing arterial line can be difficult and cumbersome in very small children due to smaller vessel sizes.

**Indications:** Arterial cannulation is done for continuous real-time blood pressure monitoring and cardiac output monitoring in patients where hemodynamic instability is expected. It also helps to withdraw blood sample for arterial blood gases and other tests.

**Contraindications:** Coagulopathy due to any reason could be a relative contraindication. Unhealthy skin over the proposed insertion site should be ruled out.

**Sites:** Radial artery is the most common site of arterial cannulation (Table 2), though some of the other sites preferred are—brachial femoral, dorsalis pedis, etc. Radial artery has low complication rates as compared to other sites. Femoral artery is often used in very small kids.

**Guidelines:** Traditionally, modified Allen’s test is suggested to test the collateral circulation in the hand, before radial arterial cannulation. Release of ulnar artery after blocking both radial and ulnar arteries, should allow skin color of the palm to normalize within 10 seconds. But, recent studies suggest that the sensitivity and specificity of this test is inadequate and hence, it is not universally recommended.

**Procedure description:** For radial artery cannulation, place the hand comfortably in 30–45° dorsiflexion by supporting the wrist with a small roll. Aseptic technique should be used by using sterile drape and gloves. There are two insertion techniques:

- **Seldinger’s technique:** Seldinger’s technique involves puncture of one wall of radial artery and passing the guidewire over which the canula is advanced.

- **Transfixation technique:** Both walls of the artery are punctured and then the needle is gradually withdrawn till a good flash back of blood is seen and the catheter is advanced.

Remember not to force the catheter or the guidewire, if resistance is encountered. It is best to remove the needle and start afresh.

**Table 2:** Recommended central and radial arterial line sizes appropriate for the age and the weight of the child

<table>
<thead>
<tr>
<th>Age and weight of the child</th>
<th>Recommended central line—size and length</th>
<th>Recommended radial arterial line</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infants &lt;5 kg</td>
<td>3 Fr, 5 cms</td>
<td>24G</td>
</tr>
<tr>
<td>Larger infants/ Toddlers 5–10 kg</td>
<td>4 Fr, 8 cms</td>
<td>24G–22G</td>
</tr>
<tr>
<td>Preschool children 10–25 kg</td>
<td>5 Fr, 12 cms</td>
<td>22G</td>
</tr>
<tr>
<td>Older children &gt;25 kg</td>
<td>7 Fr, 15 cms</td>
<td>22G–20G</td>
</tr>
</tbody>
</table>

Fig. 12: Bone marrow aspiration needles can be used as intraosseous needles.
Both the techniques are associated with equal success rates and no difference in complications like thrombosis, but the time required is longer with transfixation technique.

- **Practicalities:** Ultrasound guidance for radial artery cannulation is acceptable technique in adults, but makes it more technically difficult in pediatric patients. Nakayama et al. demonstrated injection of saline in the subcutaneous tissue to obtain optimum depth of radial artery 2–4 mm below the skin for reduced procedure time as well as increased success rate.

**UMBILICAL VEIN CATHETERIZATION**

- **Indications:** This is a life-saving procedure in neonatal resuscitation, particularly when peripheral access is difficult. It can be used as central venous access and for exchange transfusions in the first 10 days of life.
- **Contraindications:** Umbilical sepsis or intra-abdominal infections.
- **Procedure description:** This procedure should be done under aseptic precautions. The cut end of the cord has two thick walled lumens of umbilical artery and one thin walled umbilical vein roughly at 12 O’clock position. Figure 13 depicts the anatomical orientations of umbilical vessels. Umbilical cord stump may be refreshed by cutting it and stabilized using a tape. Umbilical catheter or infant feeding tube 5 Fr should be inserted after dilating the vein with small artery forceps. After about 4–5 cm insertion, back flow should be checked. The catheter may be advanced till back flow is good and is then secured using umbilical tape. For using it as a central line, it is advanced further by 4–5 cm, so that the tip lies at the junction of inferior vena cava and right atrium. Documenting the catheter tip position may be done with an X-ray, where the tip is seen just at the level of diaphragm or at T9–10 vertebrae. There is no evidence supporting the use of procedural prophylactic antibiotics.
- **Complications:** Vessel rupture, false tract, air embolism and infections may occur. Portal vein thrombosis and hepatic necrosis may occur with deeper catheters, hyperosmolar medications and longer duration of use.

**UMBILICAL ARTERY CATHETERIZATION**

- **Indications:** This is usually done for monitoring in a very sick neonate and can be often used for the first 5–7 days only. It is used for continuous arterial pressure monitoring, sampling of blood gases, exchange transfusions and angiography.
- **Contraindications:** Like umbilical vein catheterization, this cannot be done in conditions of umbilical sepsis or intra-abdominal infections.
- **Procedure description:** The procedure is very similar to umbilical vein catheterization, except that umbilical artery is dissected in the umbilical stump aseptically and 3.5 Fr or 5 Fr catheter is advanced. Side holed catheters have higher incidence of aortic thrombosis and hence, end hole catheters should be preferred. The tip of the catheter is placed above the diaphragm at ‘high position’ (i.e. tip between T6–T9 vertebrae on X-ray) or just above the aortic bifurcation at ‘low position’ (i.e. tip near L4–5 on X-ray). Recent evidence suggests that ‘high position’ has lesser complications than ‘low position’ and hence, must be preferred. For the high position, the insertion depth of the catheter may be calculated using Shukla’s or Wright’s formula. Also, there is no evidence supporting the use of procedural prophylactic antibiotics.
- **Complications:** False passage, malposition, vessel rupture, thrombosis, vasospasm of related vessels, infarction and infection.

**RECENT DEVELOPMENTS**

“Pediatric Vascular Access Teams” have been formed in major centers abroad that cater to vascular access needs of all pediatric patients. Nurses and doctors on the team are specially trained. These teams have reported positive response and improved care and might become the way to go in future.
REFERENCES


INTRODUCTION
Ultrasound imaging for nerve localization has been one of the exciting advances in regional anesthesia. This chapter provides a practical approach to the performance of regional blocks in infants and children using ultrasound guidance.

ADVANTAGES OF ULTRASOUND IMAGING IN REGIONAL BLOCKS
- Ultrasound defines the target nerve
- Identifies the tip of the needle close to the nerve (first important factor in achieving successful block)
- Notifies the spread of the local anesthetic in the perineural area
- Reduces the incidence of vascular, pleural and neural punctures.

STEPS IN PERFORMING ULTRASOUND-GUIDED NERVE BLOCKS
- Ensure a good resolution ultrasound machine is available, adequately charged and working correctly
- A linear probe of greater than 10 MHz and a suitable footprint is chosen:
  - Probe of 25 mm width for patients less than 15 kg
  - Probe of 50 mm width for patients greater than 15 kg
- Adjust the machine settings frequency, gain and depth
- Check the probe orientation
- Apply enough ultrasound gel to prevent air interference
- Position ergonomically
- The procedure should be carried out under aseptic conditions
- Perform an initial “scout” scan to locate the target
- Identify nerve/fascial plane and vascular structures
- Apply color Doppler in all scans to identify vessels
- Adjust the target in the middle of the screen
- Employ in-plane needling techniques that give greater needle visibility
- The out of plane technique needs more experience
- Flush needle with saline to remove any air.

The author follows the following protocol.
Patients are induced with intravenous fentanyl, propofol and a supraglottic airway device is introduced. Anesthesia is maintained with sevoflurane. Muscle relaxant, if required, is administered only after the block. Neurostimulation is utilized as an adjunct to ultrasound.
The specifications for all the blocks are:
- Current amplitude: 0.3 mA
- Duration: 0.1 ms
- Frequency: 1 Hz
UPPER LIMB BLOCKS

Brachial Plexus Block

The brachial plexus arises from ventral rami of the lower four cervical nerves (C5-C8) and the first thoracic nerve (T1). The roots form the trunks and divisions in the supraclavicular area. Below the clavicle are the cords and terminal nerves.

The anatomical landmarks popularized by Dalens and colleagues\(^1\) made it easy for supraclavicular approaches, but the fear of puncturing vascular structures or the pleura persisted. It was only after the pictures of sonoanatomy of the neck appeared in children, that pediatric anesthesiologists started appreciating the role of ultrasound (Fig. 1).

Interscalene Approach

Indications
- The main indication for interscalene approach is shoulder surgery, which is uncommon in the pediatric population
- Deformities of the arm, forearm and hand
- Chronic osteomyelitis of the neck and head of humerus
- Fracture of neck of humerus.

Technique

The head is slightly turned to the contralateral side. A small pillow is positioned below the scapula on the same side of block to be performed.

The probe is deployed in the transverse oblique plane so that the beam is perpendicular to the brachial trunks (Fig. 2). The brachial trunks are sandwiched between the anterior and middle scalene muscle (Fig. 3). The brachial plexus trunks are seen as three hypoechoic shadows stacked one above the other, and are surrounded by hyperechogenic epineurium. Note the bulk of the middle scalene muscle, which is larger than the anterior scalene muscle. The perimysium of the scalene muscle engulfs the brachial trunks.
The needle (white) is inserted in plane and squeezes in through the middle scalene muscle. The tip of the needle should lie just lateral to the hyperechoic (epineurium) ring. At times the needle tip penetrates the hyperechogenic epineurium and drug deposition becomes subepineural.

The in-plane technique allows complete visualization of the needle shaft and the tip. The needle tip is stabilized at the junction of the superior and the middle trunk and a test volume is injected to assess the spread of the local anesthetic.

**Neurostimulation**

At this point, neurostimulation will evoke biceps and deltoid contractions at 0.4 mA. This is the appropriate response at which point the local anesthetic is injected.

The needle is seen approaching through the upper part of the middle scalene muscle and is directed towards the superior trunk.

**Volume of local anesthetic**

0.1–0.2 mL/kg of 0.2–0.37% bupivacaine.

**Complications**

- Phrenic nerve block—This can occur even with smaller volumes and under ultrasound guidance
- Recurrent laryngeal nerve block
- Stellate ganglion block
- Some of the dreaded complications due to needle misplacements can be avoided under ultrasound imaging, e.g. epidural/spinal injection, vertebral artery puncture, bilateral spread, spinal cord injury and pneumothorax.

**POINT TO REMEMBER**

- In an “Out-of-plane technique”, the needle shaft and tip are not visualized clearly. An “In-plane technique” allows the needle advancement to be accurately visualized
- Use a high-frequency linear or hockey stick probe
- Hydro dissection is performed once needle is close to plexus
- Drug injection is performed by either surrounding the brachial plexus or lateralizing the drug from superior to inferior trunk
- Aspirate intermittently to exclude intravascular injection

**Supraventricular Approach**

**Indications**

Procedures on the arm, forearm and hand, excluding the shoulder.

**Technique**

The patient is positioned as for the interscalene approach (head slightly turned to the contralateral side). The probe is kept in coronal oblique plane. Thus, the probe is parallel to and just behind the clavicle.

The subclavian artery is seen medially, lying on or just anterior to the first rib. Color Doppler is mandatory as there are numerous vessels that are in close proximity to the brachial plexus (the dorsal scapular artery and the ascending circumflex artery). The brachial plexus is found posterior to the subclavian artery as a cluster of hypoechoic nodules, described as a “bunch of grapes” (Fig. 4).

The subclavian artery and the lower part of the divisions of brachial plexus are observed to be resting on the first rib (hyperechogenic structure; blue).

The cervical pleura is seen at both sides of the rib and is seen to “slide” with respiration. Thus, both important structures, the subclavian artery and the pleura are visualized and can be avoided. An in-plane technique is recommended for supraclavicular block (Fig. 5).

The needle (white) is inserted in plane and is close to the midpart of the brachial plexus divisions (yellow).

Confirm the tip of the needle is not in vascular lumen (red). Many-a-times the needle shaft disappears in the brachial plexus, and the tip is not visualized. It is important to assess the tip of the needle before the injection is made close to the supraclavicular brachial plexus.

It is important to understand which areas (divisions of brachial plexus) to target in supraclavicular block (Fig. 6), hence the needle tip is initially guided to 5 o’clock position and a test volume of 0.5 mL is injected. This lifts the brachial plexus away from the rib and the subclavian...
artery. Once an adequate spread is noted, another 1.5 mL is injected at the same point.

The needle is then withdrawn and slowly guided to the superior aspect of brachial divisions and 2 mL of local anesthetic is injected. Single injection techniques generally suffice for the block. However, multiple injections of local anesthetic can be performed if needed with the needle redirected to ensure sufficient circumferential spread around the plexus.

**Neurostimulation**

At this point, neurostimulation will evoke extension or flexion of metacarpophalangeal joints.

**Volume of local anesthetic**

0.2 to 0.3 mL/kg of 0.2–0.37% Bupivacaine.

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**Complications**

There is a greater risk of pneumothorax, as the cupola of the lung lies just medial to the first rib, not far from the plexus.

In patients with severe respiratory compromise and infants who are dependent on diaphragmatic function, phrenic nerve palsy may lead to respiratory distress and therefore, the supraclavicular blocks are best avoided in these populations.4

**Infraclavicular Approach**

This is an advanced block. The axillary neurovascular bundle lies deeper, with the large axillary vein lying medial and caudal to the artery. The lateral, posterior and the medial cords lie according to their relations to axillary artery.

**Technique**

The child is placed supine with the arm adducted, elbow flexed and forearm placed on the abdomen. This is a comfortable position for all patients with elbow and forearm fractures. A linear probe is placed parasagittal, i.e. perpendicular to the clavicle, and medial to the coracoid process (Fig. 7).

**Parasagittal plane**

Probe is perpendicular to the clavicle—it is slide from the coracoid process, medially until the axillary artery is in view.

The orientation marker is cephalad. The pectoralis major and minor muscles are separated by a hyperechoic lining (perimysium); the pectoralis major muscle lies superficial and lateral to the pectoralis minor muscle. A short-axis view of the brachial plexus cords and axillary vessels can be visualized in Figure 8.
The lateral cord of the plexus is readily visualized as a hyper echoic oval structure; the medial and posterior cords can be difficult to identify because the medial cord lies between the axillary artery and vein, whereas the posterior cord is often hidden deep to the axillary artery. The posterior cord is delineated in the Figure 8.

Occasionally with abduction of the arm the medial cord pops out. In addition, the medial cord can be posterior or even slightly cephalad to the axillary artery.

This author prefers to insert the needle from the superior aspect of the probe and aim toward the posterior cord (Figs 7 to 9).

The needle is carefully placed below the axillary artery. The needle shaft and tip disappear after some distance, hence a cautious hydrodissection is recommended with 0.2 mL of LA, if neurostimulation is not used.

Alternatively, neurostimulation should evoke flexion or extension of the metacarpophalangeal joints. The hydrodissection should separate the posterior cord from the axillary artery. The injectate should then surround the lateral cord and dissect the plane between the axillary artery and the vein to soak the medial cord.

Volume of local anesthetic
0.3–0.4 mL/kg 0.2 to 0.37% bupivacaine.

Note: The spread in the infraclavicular area is posterolateral to the axillary artery with a minimal spread between the axillary artery and the vein. It is critical to visualize adequate spread of local anesthetic rather than to inject the lowest potentially effective volume.

**POINT TO REMEMBER**
- The linear probe is medial to the coracoid process and inferior to the clavicle (perpendicular)
- The axillary artery is noted just below the inferior part of fascia of pectoralis minor
- The cords (hyperechoic figures) of the brachial plexus surround the artery
- The needle is in plane, with the tip of the needle aimed toward the posterior cord. The angle is steep towards the posterior cord
- The injection should hydrodissect the fascia surrounding the artery from the posterior cord
Axillary Approach

Axillary approach is a commonly performed block, easy to learn for a novice.

Indication

Procedures on the elbow, forearm and hand.

Technique

The arm is abducted to 90° and externally rotated. The probe is placed transversely, across the axilla (Fig. 10). The scan should identify the axillary artery, vein and then the individual nerves. The nerves are around the vessels. The radial nerve is inferior, median is superior, and ulnar nerve is posterior to the axillary artery. These nerves can be blocked individually via a single insertion site (Fig. 11).

The probe is placed in transverse axis, proximal in the axilla. The beam is perpendicular to the axillary artery and the nerves in the axilla. The needle is in plane to the perivascular-perineural structures.

The musculocutaneous nerve (green) is between the biceps and coracobrachialis and at times in the mass of coracobrachialis. This may require a second insertion of the needle, but in the pediatric population is usually accomplished with single puncture.

In the lower part of the scan the humerus (blue) stands out as the hyperechoic rim with hypoechoic dorsal shadowing. Directly above is the axillary artery (red—hypoechoic shadow). This is pulsatile. Superiorly is the median nerve at 1 o’clock position (yellow—multiple hypoechoic shadows), posteriorly is the ulnar nerve at 2 o’clock position and inferiorly is the radial nerve (yellow—multiple hypoechoic shadows).

A small volume of LA <1 mL (blue) delineates the median nerve (yellow)(Fig. 12 and 13).

The needle tip is then directed to the posteroinferior aspect of the axillary artery to target the radial and ulnar neural elements.

The needle placement (white) is inferior to the axillary artery to localize the radial nerve. The median nerve (yellow) is visualized superior to the axillary artery and bathed in local anesthetic (blue).

In pediatric population the neural elements are very superficial and at a depth of less than 0.5 cm. In this case, the subcutaneous fat will camouflage the inserted needle.

Neurostimulation

- Median and radial nerve stimulation
- Flexion or extension of metatarsophalangeal joints.

Volume of local anesthetic

0.3–0.4 mL/kg of 0.2–0.37% bupivacaine.

POINT TO REMEMBER

The shallow depth of the neurovascular structures and the easy compressible arterial lumen needs to be carefully evaluated before advancement of the needle.

LOWER LIMB BLOCKS

Lumbar Plexus Block

This is an advanced block. The lumbar plexus (LP) arises from ventral rami L1–L4 and after emerging through the intervertebral foramina they form the plexus in front of the transverse process (TP) and posterior one-third of the psoas
major. Lumbar plexus blocks are considered to be difficult blocks to perform in view of the potential risks involved.\textsuperscript{5-7}

**Technique**

The needle tip after encountering the TP is made to slide cephalad or caudad 0.5 cm beyond the TP. The distance from the TP and the LP is fixed.

Lines passing through above points intersect at one point. This point is the point of needle insertion (Fig. 14).

The transverse processes are not fully developed in children.\textsuperscript{5} If the TP is used as an anatomical landmark for needle placement, the needle tip might be too medial.\textsuperscript{5}

This increases the risk of puncturing the dural cuff of the spinal roots.\textsuperscript{5}

**Indications**

Perioperative analgesia for procedures on hip, thigh and knee.

**Contraindications**

Retroperitoneal pathology.

**The Lumbar Paravertebral Ultrasound**

In a transverse (axial) view using a linear probe in infants, the following are visualized (Figs 15 and 16).
**Bony structures:** Spinous processes, laminae and transverse processes.

**Muscles:** External oblique, internal oblique, transverse abdominis, erector spinae, quadratus lumborum and the psoas major.

The linear probe guides in visualizing the lumbar plexus in the pediatric age group. The probe is initially placed in the midaxillary line and slowly shifted posteriorly to the back of the patient. Initially, the abdominal muscles will be observed, and as they terminate the quadratus lumborum (QL) will be seen emerging. The QL is followed medially to observe the transverse process (TP). In front of the TP is the psoas major and medially is the vertebral body. The lumbar neural elements (white streak on ultrasound) will be emerging from the intervertebral foramina (at times not seen on ultrasound). The lumbar neural elements are concentrated in the posterior one-third of the psoas major.

**Technique**

A scout scan of paravertebral space is performed to identify the various structures. The TP is visualized and attempts are made to note the lumbar neural elements in the posterior one-third of the psoas major.

Note (yellow dotted, Fig. 16) the neural elements in lumbar area. These are the lumbar neural components sandwiched in the psoas muscle. The paravertebral and abdominal muscles are easily identifiable. The needle is inserted according to anatomical landmark mentioned earlier.

The probe is placed in transverse oblique plane (Fig. 17). The ultrasound beam is exactly in plane to the stimulating needle. Thus, the entire shaft and the tip of the needle should be visualized as the tip encounters the TP and goes beyond the TP. The needle is placed just beyond the TP (Fig. 18). The needle shaft and tip are identified in the posterior quadrant of psoas major.

**End-point**

The end-point is quadriceps contractions at 0.4 mA.

The local anesthetic is then injected in small boluses of 1–2 mL and the spread is noted on ultrasound. The
psoas muscle is displaced as the sheath of the neural elements gets filled with the local anesthetic (light brown).

Volume of local anesthetic: 0.5–0.6 mL/kg of 0.25–0.375% bupivacaine.

Kirchmar L and colleagues visualized the lumbar paravertebral region with a curved array probe. The authors placed the probe in longitudinal and transverse planes to view the lumbar plexus and to measure the skin-to-plexus distance. The strongest positive correlation existed between skin-plexus distances and the children’s weight.*

**Outcome**

The ultrasound-guided lumbar plexus blocks could be performed fast and showed rapid onset times. Effective anesthesia and analgesia of the area innervated by the lumbar plexus during the surgical procedure as well as for postoperative pain relief could be provided.* The major benefit of ultrasound-guided techniques is the monitoring of local anesthetic spread as a reliable predictor of success.

**Femoral Nerve Block**

The femoral nerve is the largest branch of the lumbar plexus arising from the dorsal division of the second to fourth lumbar nerves. It emerges from the lower border of the psoas muscle, runs between the psoas and the iliacus muscle, and passes underneath the inguinal ligament into the femoral triangle. At the level of the inguinal ligament, it lies deep to fascia lata and iliaca in a groove between the iliacus and the psoas muscle and is separated from the femoral vessels, which lie in a separate fascial compartment medial to the nerve.

It supplies the anterior compartment of the thigh and the medial aspect of the leg.

**Indications**

- Analgesia for femoral fracture for intra- and inter-hospital transfers
- Postoperative analgesia for hip, femur and knee surgery
- Correction of hip and femur deformities and exploration of knee joint and quadriceplasty
- In combination with sciatic nerve block, it can provide analgesia for all procedures below knee.

**Technique**

The probe is placed just below and parallel to the inguinal ligament. This is the transverse axis scanning. The color Doppler identifies the femoral vein and artery, and the superficial circumflex iliac vessels as they pass directly over the femoral nerve. The nerve is found lateral to the femoral artery in a triangular hyperechoic area. The femoral nerve lies over the iliacus muscle and is engulfed by the fascia iliaca sheath (Fig. 19).

The needle is inserted in-plane (from lateral to medial) technique. Two pops are felt as this needle pierces the fasciae.

Initially, the needle tip is inserted below the femoral nerve and a bolus is injected to lift the femoral nerve. Later the needle tip is deployed superior to the femoral nerve. The nerve should be surrounded with local anesthetic. Ensure that the local anesthetic is deposited under the fascia iliaca. Distally, if ultrasound scan is performed, femoral nerve branching is visualized.

**Neurostimulation**

The end-point is quadriceps contractions at 0.4 mA.

**Volume of local anesthetic**

0.5–0.6 mL/kg of 0.25–0.375% bupivacaine.

**Sciatic Nerve Block**

The sciatic nerve has tibial and common peroneal components. It enters the gluteal region from the pelvis through the greater sciatic foramen, before running down the leg between the ischial tuberosity and the greater trochanter of the femur.

It usually divides at the apex of the popliteal fossa, though this can occur proximally. As it exits the gluteal region, it is accompanied by the posterior cutaneous nerve of the thigh on its medial aspect. The nerve supplies the skin on the posterior part of the thigh, hamstring, and...
biceps femoris muscles and the leg below the knee joint, except for the area of skin supplied by the saphenous nerve (medial aspect of leg).

**Indications**
- Isolated sciatic block—Ankle and foot surgery
- In combination with a saphenous or femoral nerve block, provides analgesia for all procedures below the knee.

**Technique**
Following approaches can be considered depending on the site of surgery:
- Subgluteal
- Midthigh
- Popliteal.

**Popliteal Sciatic Nerve Block**

The popliteal approach is most commonly used and is facilitated by placing the patient in the lateral position, with the operative side uppermost. The upper leg is slightly bent and rested on the flexed lower leg. The probe is placed transversely on the popliteal crease.

Color Doppler identifies the popliteal vessels (Fig. 20). The tibial nerve is visualized just posterior to the popliteal vein; as the probe is scanned proximally, the common peroneal nerve can be seen laterally moving medial to join the tibial nerve. The probe is now purposefully made longitudinal to the sciatic nerve. The fascicular pattern (Fig. 21) of the sciatic nerve is noted (yellow). A hypoechoic shadow is observed to spread on the superior aspect of the sciatic nerve (light blue).

Apart from a routine use of neurostimulation, it is mandatory to use nerve stimulator to locate the nerves in conditions like cerebral palsy and muscular dystrophies, since the nerves are difficult to locate because of fatty infiltration.

**Neurostimulation**

Plantar or dorsiflexion of the foot.

**Volume of local anesthetic**

0.5–0.6 mL/kg of 0.25–0.375% Bupivacaine.

Ultrasound guidance for sciatic and femoral nerve blocks in children increases the duration of sensory blockade in comparison with nerve stimulator guidance. Prolonged sensory blockade is achieved with smaller volumes of local anesthetic (0.3 mL/kg) when using ultrasound guidance.10

**Thoracic Paravertebral Block**

**Anatomy of the paravertebral space:** The paravertebral space is wedge-shaped; the base is formed by the posterolateral part of the vertebral body, the disc, and the intervertebral foramina with its contents. The anterolateral boundary is formed by the parietal pleura and the posterior wall by the superior costotransverse ligament (Fig. 22).

The aim of this block is to inject local anesthetic into the wedge-shaped paravertebral space found on either side of the vertebral column. This blocks the motor, sensory and the sympathetic chain.

**Indications**
- Unilateral thoracic surgery
- Upper abdominal surgery.
Chapter 37: Ultrasound-guided Regional Blocks

Contraindications
- Empyema and tumor occupying the paravertebral space
- Kyphoscoliosis
- Scarring from previous thoracotomy or inflammation.

Technique
The patient is placed in lateral position with the operative side uppermost. The level of block is dictated by the surgery; T5 for thoracotomy and T10 for abdominal surgery. USG can be used to assess the depth to the transverse process and pleura prior to the landmark technique.

Approaches
- In-plane technique provides better visualization of the needle and is the accepted technique for paravertebral block
- Out-of-planes offers visualization of the pleural depression though the needle movements cannot be observed clearly.

Out-of-plane Technique
The needle is inserted out of plane, lateral to the spinous process. The tip encounters the costotransverse junction (CTJ). The needle tip is skirted beyond this CTJ.

The volume of LA depresses the pleura as it occupies the wedge-shaped space in the paravertebral area. The probe is placed longitudinal to assess the spread of the LA. The spread is observed over several intercostal spaces.

Volume
A volume of 0.5 mL/kg is injected; its craniocephalad spread is then assessed.

Complications
- Pneumothorax
- Contralateral paravertebral spread
- Epidural spread
- Dural tap.

In-plane Thoracic Paravertebral Block
Alternatively, the probe is placed on the lateral thorax and parallel to the intercostal spaces. The needle is inserted in plane to the probe. Local anesthetic injection expands the paravertebral space (Figs 24 and 25).

The landmark-based techniques in children are now being replaced with ultrasound-guided techniques. Unilateral and bilateral catheters can be inserted.16

Transversus Abdominis Plane (TAP) Block
The lateral abdominal wall is made up of the external oblique, internal oblique, and transversus abdominis muscles. It is between these inner two muscles that the anterior primary rami of the lower six thoracic and the first lumbar nerves pass.

Indications
- Unilateral TAP block
- Appendicectomy, inguinal hernia repair and iliac crest bone graft
- Bilateral TAP block
- Laparoscopic operations and lower abdominal incisions.

Note: TAP block does not provide visceral analgesia.
Anatomy

The intercostal nerves are the anterior primary rami of the spinal nerves T1–T11. The lower five thoracic nerves (T5–12) in conjunction with L1 nerves supply the abdominal wall. The neural plexus lies in the sheath between the internal oblique and transversus abdominis muscles.

Technique

The patient is positioned supine. The probe is placed in the transverse plane on the lateral abdominal wall midway between the costal margin and the iliac crest. All three layers of muscles and the peritoneum can be visualized (Fig. 26). The subcutaneous fat in larger patients can mimic a muscle layer to the inexperienced practitioner.

A 22 g insulated block needle is inserted in plane (Figs 27 and 28).

The angle of insertion is such that it will traverse through the external and internal oblique muscles to reach the plane posterior to the mid-axillary line, thus blocking the lateral branches of the nerves. In the Figure 28, the needle tip penetrates the sheath of internal oblique and tent the transversus abdominis. Local anesthetic deposited here spreads along the sheath between the IO and TA.
A small volume of LA is injected to confirm correct placement of the needle seen as splitting of the two muscle layers. A total of 0.5 mL/kg/side is injected.

**Complications**

Intraperitoneal injection, bowel perforation, visceral injury.

The use of ultrasound guidance is strongly recommended to facilitate correct needle placement and adequate spread of local anesthetic for truncal blocks.17

**REFERENCES**

ULTRASOUND-GUIDED NEURAXIAL BLOCKS

SONOANATOMY OF THE SPINE

Real-time sonography reveals not only the non-radiopaque parts of the spinal column such as cartilage, spinal cord, subarachnoid space, and nerve roots; but, more importantly, it helps to visualize cord movements and vascular pulsations and permits visualization of suspected pathology in transverse and longitudinal sections. This, in turn, provides a three-dimensional understanding of spinal anatomy and pathology.

Osteochondral components of the vertebral bodies and posterolateral elements are best visualized on transverse sonograms (Fig. 29). The bodies are visible at all levels, and the posterolateral elements are consistently visible with bilaterally symmetric obliquity from their dorsal to ventral aspects.

Absolute symmetry of the posterolateral element is the rule, regardless of age-dependent variations of spinal anatomy. The spinal cord is anechoic, but its central canal is echogenic, most likely the result of imaging the anterior and posterior aspects of the wall of the central canal as a single structure because they are too closely spaced to be resolved.

SINGLE-SHOT CAUDAL BLOCK

The spread of the single shot caudal can be assessed to limit the height of the block (Figs 30 and 31). The volume of the drug is of utmost importance as an inadvertently injected large volume can reach higher levels of the epidural space.

Indications
Lower abdominal, pelvic and lower limb surgeries.

Fig. 29: Sonoanatomy of the thoracic spine
Abbreviation: SP—spinous process; TP—transverse process; SC—spinal cord

Fig. 30: Longitudinal axis assessment of caudal epidural block

Fig. 31: Spread of LA in caudal block
Abbreviation: LA—local anesthetic; FT—filum terminale; ITS—intrathecal space
ULTRASOUND-GUIDED CATHETERS

Catheters can be introduced from the caudal epidural space to the thoracic level. The caudal space is identified with a loss of resistance technique after an appropriate sized Tuohy needle is inserted. The needle is identified with a hockey stick probe and catheter is inserted up to the desired level (Fig. 32).

The progression of the catheter along the epidural space is monitored. At the level of T12–L1 a resistance is generally felt as the catheter is inserted, a small manipulation is required to overcome this. In real-time, the catheter is observed progressing cephalad.

The subarachnoid space is anechoic, but contains echogenic structures, probably representing the nerve roots and vascular branches. In infants, unlike adults, the transverse diameter of the canal is wider than the vertebral bodies at all levels of the spine.

Once the catheter is sited, a small bolus of 1 mL of LA is injected. The LA spread is identified as it depresses the posterior dura anteriorly. The spread of the LA provides an idea of the possibility of the number of nerve roots blocked.

The longitudinal view (Fig. 33) of the spinal canal in the thoracic area depicting the following structures from dorsal to ventral: Subcutaneous tissue, erector spinae, spinous process, epidural catheter, posterior dura, spinal cord, anterior dura, vertebral column.

Ultrasound imaging can be an excellent tool to identify neuraxial structures in infants and children, particularly as their epidural spaces are superficial and the posterior elements of the spinal canal are less ossified than those of adults. The spinal cord often appears to occupy the center of the canal in the cervical region, to pass more anteriorly in the thoracic region, and to return toward a more central position in the lumbar canal.

The infant cord is round in the cervical region, becoming oval in the distal thoracic region. At the level of T10–T11, the cord widens into a bulbous conus medullaris, while at L1–L2, the conus narrows to form the filum terminale.

More distally, the nerve roots forming the cauda equina are visible as multiple echogenic linear structures.

Visualization of the dura mater is seen best, although the ligamentum flavum, intrathecal space, spinal cord, nerve roots and fibers can all be seen adequately via a soft tissue window among the bony structures. The precise catheter tip visualization has been suggested to only be possible in infants younger than one year or six months of age.

EQUIPMENT

The children’s epidural spaces are more superficial than those of adults, lending to somewhat higher resolution imaging (5 MHz to 10 MHz). In ultrasound-guided lumbar epidural catheter placement, a good correlation of ultrasound-measured depth of the epidural space with that from LOR was observed.
Limitations of Ultrasound

Ultrasound imaging for regional anesthesia takes considerable time and effort to learn.
- Long learning curve to optimally view the needle tip and shaft
- The need for an assistant
- The expense of the equipment
- The role of strict sterility is essential.

Neurostimulation versus ultrasound for epidural in infants.

The benefit and safety profile of ultrasound guidance during epidural anesthesia over that of epidural electrical stimulation, has yet to be determined. Neurostimulation detects intrathecal placement, on this particular aspect it may score over ultrasound, at least with current technology and knowledge.

Combining Neurostimulation and Ultrasound

Anesthesiologists can now confirm catheter placement in the epidural space with neurostimulation and also accurately determine where the catheter tip is in the central neuraxis with ultrasound imaging.

ACKNOWLEDGMENTS

The images are with the permission of the Director, Sancheti Hospital, Pune. My sincere thanks to Dr Parag Sancheti, Director, Dr Sandeep Patwardhan, Pediatric Orthopedic Surgeon, Sancheti Hospital, Pune and Dr. Sudhakar Sane, Pediatric surgeon, Miraj.

SUGGESTED READING

Notes on Allied Topics

Chapter 38: Safety and Quality in Pediatric Anesthesia
Chapter 39: Ethical Issues in Pediatric Anesthesia
Chapter 40: Simulation in Pediatric Anesthesia
INTRODUCTION

Safety and quality, these two words are interlinked. When the quality of delivery of anesthesia improves, the safety is taken care of. When safety is considered as the primary goal, quality needs to be maintained.

Precise definition of quality is difficult to express. However, there are three components of framework for measuring quality described by Donabedian.

1. **Structure** refers to the setting in which anesthesia is provided. This will include personnel and facilities used to provide anesthesia care. Organizational structure includes qualified manpower and technical facilities.

2. **Process** involves what is actually done by anesthetic care provider. This can be interpreted in terms of evaluation, diagnosis and anesthetic care (for surgical therapy).

3. **Outcome** of care can be evaluated by effects of care on the status of the patient. As anesthesia is not therapeutic, good care can be stated, if there is no worsening of the disease or condition of the patient and poor outcome involves adverse events and complications.

So, in short we can say, “High quality structure facilitates high quality process of care which will result in high quality outcome as patient’s wellbeing and satisfaction”.

Anesthesiologists work with numerous specialists in various branches of medical field. In addition, the field of pediatric anesthesia has depth of complexity stemming from patient comorbidities, surgical intricacies and evolving medical technology. So, for pediatric anesthesiologists, improvement in quality is a complex challenge. Their involvement is multifaceted in nature with care in operating rooms, intensive care unit and remote locations. They deal with diversity of patients such as tiny premature neonates to robust adolescents. All these factors confound simple definition of high quality anesthesia care.

HIGH QUALITY ANESTHESIA CARE HAS SIX ATTRIBUTES

1. **Safety**: Do no harm and avoid injuries
2. **Effectiveness**: Providing service guided by scientific knowledge
3. **Patient centeredness**: Clinical decisions guided by patient’s values and preferences, and respectful and responsive care offered to each patient
4. **Timeliness**: Harmful delays must be avoided which may affect diagnosis, treatment and satisfaction
5. **Efficiency**: Waste of material supplies, equipment and energy must be avoided
6. **Equity**: Anesthesia care should not vary in quality because of gender, age, ethnicity, and socioeconomic status.

Quality of care should be monitored from time to time at predetermined intervals. To monitor quality, there should be—a. Hospital standardization program, b. Gaps in quality are recognized and lessons learnt translated to bring in improvement.
ASSESSMENT OF QUALITY OF ANESTHESIA CARE

A. Assessment of structure of anesthetic care requires evidence of clear link of improved infrastructure to better performance.

B. Assessment of processes is dependent on scientific knowledge of meaningful effect of processes on outcomes.

C. Assessment of outcomes relates to complications, rehospitalization, morbidity and mortality.

Regular assessment of these is truly meaningful for improving the quality of care and identification of gap in the quality, but this is no easy feat. Clinical science incorporates three great branches such as “diagnosis, prognosis and treatment”. Now our clinical practice in anesthesiology has the fourth branch, which incorporates measuring and improving the quality of care we deliver. This is a continuously evolving science.

IMPLEMENTATION OF QUALITY IMPROVEMENT

1. Improvement is facilitated by collaboration with other colleagues and institutions. There is a need to involve colleagues to find ways to improve anesthesia care through regular education, learning technology and emphasis on importance of team work.

2. Monitoring our processes and outcomes are prerequisites to improving performance. Analysis of retrospective data on processes and outcomes of care with automated anesthesia information systems offers to improve learning process from wide group of practices. Collection and analysis of the data can serve as a quality improvement tool.

3. Finding leaders from both institution and frontline staff to have alliance to work together for successful outcome.

4. Development of strategies to improve quality, identification to overcome barriers is an important step. At local level certain aspects of hospital culture may create barriers which need to change to improve quality.

So, the framework for improvement of quality can be outlined as—identifying problems in various areas of institution, testing, evaluating them, and implementing changes. Appropriate quality measures must be established and tracked over time to determine if the changes implemented are successful.

Excellence is a dynamic change of any performing science including anesthesiology. Only through multi-disciplinary education, anesthesiologists can understand and appreciate the knowledge and valuable opinions of other colleagues who take care of an individual patient or trying to improve patient care on a system wide level. Without emotional bond with patient, point of excellence (highest quality) cannot be attained. We need to grow more friendly, communicative, and compassionate.

“To err is human” and it is a part of human cognitive function. Errors producing conditions can be enumerated as stress, fatigue, distraction, interruption, time pressure, poor planning, unfamiliarity of the place, and most important, lack of knowledge. Analysis of errors may reveal causative factors as—casual approach, callousness, ignorance, impatience, carelessness. Errors can be of three types: A. Errors of commission caused by “wrong doing”; B. Errors of omission caused by “not doing right thing”; C. Errors of execution caused by “doing right thing incorrectly”.

Definition of patient safety—Prevention of health care errors (anesthetic) and elimination or mitigation of patient injury caused by anesthesia delivery during perioperative period. Adverse events can be defined as undesirable clinical outcome during the whole process of evaluation, diagnosis and therapy (anesthetic management). Focus on prevention of adverse events can be dealt by proper human factors approach such as involvement, proper communication and coordination of different departments. A number of new steps have been taken to ensure perioperative patient safety. Implementation of standards for basic monitoring with advanced technology has been suggested. After proper inspection, documentation of the quality assurance process has been emphasized for accreditation of the hospital. A number of national and international organizations have been created that include International Committee on Prevention of Anesthesia Mortality and Morbidity (Cooper 1988) and the Anesthesia Safety Foundation (Cooper and Pierce 1986), and Guidelines for the provision of anesthetic services (GPAS).

KEY POINT

Crisis management can be enumerated as: Know your environment, anticipate and plan, call for help early, exercise your leadership for team work, distribute workload, mobilize resources, communicate effectively, use available information, cross and double check situation, use cognitive aids, re-evaluate repeatedly, allocate attention and set priorities.
COMMON CAUSES OF UNDESIRABLE EVENTS DURING ANESTHESIA

1. Risk factors:
   A. Age—infants because of the small size and physiologic peculiarities are at higher risk of developing complications than older children.
   B. Physical status—there is significant correlation between perioperative adverse events and number of coexisting diseases and ASA physical status.
   C. Emergency Surgery—The French study showed a three-fold increase in adverse events in pediatric emergency cases as compared to elective surgeries.
   D. Training—Adequate and proper training in pediatric anesthesia is particularly important to minimize incidence of complications.

2. Judgment errors: Most anesthetic adverse events involve human judgment errors, which are preventable. These include inadequate ventilation, equipment failure, wrong choice of anesthetic, inadequate preparation and postoperative management, lack of experience, lack of skilled assistance, and lack of attention. Most of these events can be prevented by adequate monitoring with pulse oximetry, capnography, cardiography, and noninvasive blood pressure monitoring.

3. Urgent or emergency cases: Inadequate pre-anesthetic preparation of patient for urgent surgeries is often responsible for adverse events. Appropriate hydration, electrolyte balance and optimum hemoglobin level should be achieved before surgical procedure to prevent complications.

4. Timing of occurrence: It has been observed that more than 50% of cardiac arrests occurred during induction of anesthesia and the rest during maintenance and emergence or in PACU.

5. Anesthetic over dosage: Incidence of overdose has decreased over the last two decades with adequate monitoring. Earlier reported incidents of cardiac arrests in children were due to overdose of halothane.

6. Training: The incidence of adverse events can be minimized mainly by proper training and adequate experience in pediatric anesthesia. It has been observed that incidence of bradycardia was significantly higher when pediatric anesthesiologist was not supervising the trainees administering anesthetic to infants.

Along with skilled anesthesiologists, technical advances have certainly reduced the incidence of adverse events. Technical advances can be enumerated as newer anesthetic agents, more precise mode of administration, better monitoring methods, and availability of equipment for resuscitation and textual knowledge of clinical and electronic monitoring.

ANESTHETIC TRAINING SHOULD INCORPORATE

- Focus on basic knowledge and technical skill
- Emphasis on avoidance of problems
- Protocol for emergencies
- Frequent testing of emergency equipment
- Management of crisis situation protocol
- Fire drill training
- Nontechnical skills such as decision making, assertiveness, communication skills and situational awareness.

Anesthesia training should include checklists for:

**Patients:** Allergies, loose teeth, tendency for reflux, neurological condition (epilepsy), asthma, respiratory and heart disease, medication, bleeding disorder and surgeries in the past, and history of any diseases in family.

**OR Equipment:** Anesthesia machine, breathing circuit, ventilator, airway equipment, suction, and monitoring equipment, warming equipment.

Anesthesia training and proper checklists can help to reduce errors which include improper anesthesia setup, judgment errors, medication errors, (e.g. omission, repetition, substitution, incorrect dose, incorrect route or wrong time) inadequate ventilation, monitoring error, technical mishap, aspiration, inadequate resuscitation, inadequate reversal, hypoxic mixture.

Training in simulation has shown that checklists reduce the amount of crucial steps that may be missed in intraoperative emergencies. The checklists for pediatric anesthesia emergencies developed by the quality and safety committee are—air embolism, anaphylaxis, cardiac arrest, bradycardia, difficult airway, hypertension, hypotension, hyperkalemia, hypoxia, local anesthetic toxicity, malignant hyperthermia, tachycardia, transfusion reactions, trauma, loss of evoked potentials, myocardial ischemia, fire-airway and operating room.

Timely recognition of any adverse event is extremely important to initiate appropriate measures for treatment. So, continuous vigilance or sustained attention is essential for patient safety during perioperative period. Vigilance can be defined as sustained attention composed of alertness, selection of information from monitors and readiness for action. Anesthesiologists should aim for
near perfection by minimizing factors that adversely affect vigilance such as distraction, boredom, overwork, fatigue and lack of motivation. Once identified, the error should be reported promptly and honestly. Analysis of critical incident reporting can be instrumental in improving patient safety.

SPECIALTY PROFESSIONAL TRAINING HAS EVOLVED INTO MULTIDISCIPLINARY EDUCATION (MDE) INVOLVING MANY STEPS

1. Isolation—education in only your branch.
2. Awareness—teacher’s awareness about other branches.
3. Consultation—discussion of teaching programs between teachers of different branches.
4. Nesting—information about work in other branch.
5. Temporal coordination—teaching program for two or more branches.
7. Correlation—planning program for professionals from many branches.
8. Complimentary program—along with single branch, teaching of complimentary branches.
9. Multi-professional—emphasis on integration of multiple branches.
10. Inter-professional—teaching about each branch from the perspective of its own and other branches.
11. Transprofessional—based on the experience of real world education of multiple branches.

For any given situation or potential scenario, anesthesiologist considers thoughtfully before selecting a suitable technique. Preferences may vary widely among anesthesiologists. So, safe performance cannot be achieved by only regulation and standardization but is based on resilience. Incorporating self-assessment on an individual level is an important aspect for the situation. Cuvelier and Falzon P study mention that anesthesiologists’ resilience is fundamental in maintaining patient’s safety, and anesthesiologists differ in the strategies they use to create resilience. Successful performance usually does not attract attention. Failure is often studied— and is called “Safety 1 study”, while “Safety 2” refers to the study and promotion of success in complex situation. Recognition and importance of allowing anesthesiologists to be flexible, innovative and adaptable are important to understand success. So, reactive Safety 1 to proactive Safety 2 culture needs rebalancing for planning strategies to promote success.

When many professionals are involved in maintenance of safety and quality in the anesthesia department, good leadership can inspire creativity, ingenuity, and excellent productivity.

CONCLUSION

Safety and quality in pediatric anesthesia can be maintained by “Anticipation” of a problem, “Precise” skillful delivery of anesthesia, continuous “Vigilance” of physiological scenario, and “Prompt action” to avert the adverse event. All these are possible only by proper training in pediatric anesthesia, continuous quality improvement programs and frequent multidisciplinary teaching programs.

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INTRODUCTION

Anesthesiologists often face ethical dilemmas while managing pediatric surgical patients. These are somewhat different from those encountered by treating physicians and surgeons. Anesthesiologists’ responsibility includes respecting the wishes of the child undergoing surgery, allaying anxiety of both the child and parents, obtaining assent and informed consent, as well as conveying postoperative issues. The pediatric anesthesiologist faces ethical dilemmas about how much information should be offered and disclosed to the parents. The predicament increases as many anesthetic drugs are not approved for use in children and are used as “off-label”, and the long-term effects of anesthetic drugs on the developing brain are still unknown. This chapter covers these ethical issues in brief, explaining the process of informed consent and assent, and research in pediatric patients. The Institutional Ethics Committee plays a vital role in an institute ensuring adherence to guidelines and resolving ethical issues.

Consider the situation of an anesthesiologist who recommends deferring surgery in an infant because of an upper respiratory infection (URI). How should the anesthesiologist respond to a parental request to proceed? In an Indian context, surgery may have been scheduled after a big waiting list in government hospitals, parents may have travelled large distances or incurred loss of wages. These decisions are often taken as medical decisions based on the characteristics of the URI and the nature of surgery. But within the decisions lie the ethical components of informed consent and obligations to the child and family.

Ethical principles help anesthesiologists to identify critical factors, apply the relevant principles in the interest of the child and the family, and have an individualized case-based approach for formulating practical solutions. This is especially true today, when medicolegal suits are common. The law, however, is not a desirable substitute for resolving ethical dilemmas. The law represents a lower boundary for acceptable behavior, whereas ethics formulate a standard to which one should adhere.

Elements of consent and assent as defined by the American Academy of Pediatrics Committee on Bioethics (Box 1).

INFORMED CONSENT

Taking an informed consent for any medical care is a process. The process of pediatric informed consent depends on the age of the child. Minors are not considered legally competent for giving an informed consent. Children are considered to lack the maturity, experience, and capacity to make appropriate decisions. The “natural bonds of affection” between parents and children imply parents will act in the best interest of their children. Therefore, parents (or legal guardians) are considered as best decision makers for their children.

The anesthesiologist is obligated to provide the parents with a “reasonable amount of information” for them to make an informed decision. The parents should know before surgery what they are consenting for. This consent should be taken after explaining the proposed procedure and possible alternative treatments. The associated common complications, the nature and degree
of the risks and benefits involved in decision of accepting or rejecting the treatment should be explained. Consent is then considered “informed” when given “knowingly, competently and voluntarily” (Box 1).

knowingly means that the anesthesiologist has an obligatory duty of disclosure to provide adequate information to the parents in a manner in which they can comprehend. “Adequate” means the amount and kind of information that the average person in the parent’s position would want to have regarding the child in reaching an informed decision. This information should be given in the language the parents understand and should be documented.

Competently means that the parents have the capacity to understand “information relating to the surgical procedure and to appreciate the consequences of such a decision”. The challenge is to assess the ability of the parents, whether they can understand the relevant medical information and make a rational decision based on it. This depends on the educational level and other sociocultural factors.

voluntarily means the parents should take the decision free from manipulation or undue influence. Due to the vulnerable situation of the child and parents, danger of undue influence from the health care providers or persuading the parents or child when assent is taken for any procedure should be avoided.

An appropriately taken informed consent protects the anesthesiologist in the legal proceedings in the court of law. This is particularly important with increasing medical litigation cases as health care professionals come under the Consumer Protection Act.

asSENT

Children younger than the age of 7 years have insufficient decision-making capacities to effectively participate in the informed consent process.

The American Academy of Pediatrics uses the term “informed permission”, when the parent provides legal consent and ethical decision making for the child. For children between the ages of 7 and 13, anesthesiologists should seek both informed permission from the parent and assent and participatory decision-making from the child. It is an ethical obligation for the pediatric anesthesiologist to involve children in the informed consent process to the maximum extent depending on their level of understanding and maturity.

Children with significant decision-making capacity (perhaps around the age of 10 years but certainly by the age of 13 years) might refuse some procedures. Anesthesiologists should respect this refusal of assent and consider explaining the child to relieve his doubts and anxiety. Anesthesiologists may emphasize that nothing will happen without the child’s approval, but only if that is true. Moving the discussion away from the preoperative area or letting the child dress in street clothes, showing a video about the operating room and procedure, respecting the child’s wish for oral sedation, using local anesthetic for taking an IV, explaining about patient controlled analgesia for providing pain relief; will often reduce stress and improve communication.

Extra care should be taken conscientiously to avoid pressuring the child. Coercing or manipulating a child into having a procedure damages the child’s trust of the medical profession and impairs future cooperation. Maintenance of trust is particularly important in children with chronic medical conditions. Strategies for resolving conflicts should include maintaining communication, clarifying misunderstandings about the anesthetic and surgical experience, and decreasing the anxiety of both the child and parents. The goal is to resolve the problem without impairing relationships among clinicians, patient, and parents.

MATURE MINOR

When minors reach a specific age, especially adolescence or nearing maturity, treatment for specific conditions like pregnancy, sexually transmitted diseases or substance...
abuse can raise ethical issues. Many times, they do not disclose such information to their parents.

The “mature minor doctrine” ensures treatment of minors when parental consent may be difficult to obtain or may cause family conflict, and protects physicians who treat “mature minors.” Indian law does not recognize the mature minor doctrine. In such situations, anesthesiologist along with treating physician/surgeon should encourage the adolescent to share relevant information with the parents, counsel the parents to handle the situation and suggest appropriate solutions; thereby obtaining consent from minor and parents.

**The Emancipated Minor**

Minors are deemed emancipated when they have the right to consent or refuse medical treatment without the involvement of the minor’s parent, guardian, or other third party, regardless to whether the minor has reached the age of majority. The legal authorities of the state award this status to patients who are in the military, who are married, who have children and who are economically independent.

**SPECIAL SITUATIONS**

**Children of Jehovah’s Witnesses**

Jehovah’s Witnesses believe the biblical scripture that anyone who takes blood will be “cut off from his people” and not receive eternal salvation. Anesthesiologists should directly discuss the issue of transfusion therapy while handling child of Jehovah’s Witness. The anesthesiologist should clarify that attempt to follow the family’s wishes would be done within the standard of care. Various acceptable interventions like deliberate hypotension, deliberate hypothermia, hemodilution, and use of cell saver blood, should be explained. Use of medications like synthetic colloid solutions, dextran, erythropoietin, desmopressin, and preoperative iron should be offered. These options are feasible in slightly bigger children and adolescents. Fewer options may be available for smaller babies considering the narrow safety margin for transfusion. The family should be informed that in critical situations, the anesthesiologist would transfuse as per the standard of care for wellbeing of the child. The anesthesiologist may, in cases with increased likelihood of transfusion, preoperatively obtain order from the local legal authority as well as take help of institutional ethics committee.

**Battered Baby Syndrome**

In case of child abuse, parents or guardians may not consent in the best interest of the child. The anesthesiologist is ethically right in going ahead with emergency life-saving care. For further management, help of the local legal authorities should be taken.

**Do-not-Resuscitate Orders/Do not Intubate/Life Sustaining Medical Therapy**

There are no laws formulated in India for such orders. Suffering of the child, consent of family and the availability of resources are to be taken into consideration and management of such cases should be individualized.

**RESEARCH IN PEDIATRIC PATIENTS**

Research in pediatric patients requires greater caution and care than research in adults, as they are considered a vulnerable population. Their inability for right decision making makes them vulnerable to abuse and exploitation during research. Pediatric research exposes children to unknown risks of long-term harm because research interventions are usually done during growth and development of the child.

Any research should be carried after approval from the Institutional Ethics Committee (IEC) or Institutional Review board (IRB). IEC or IRB should strictly follow guidelines laid by international federation for research as well as Indian Council of Medical Research. The IRB should facilitate appropriate unbiased research, not hindering necessary and beneficial research, and regularly supervising and protecting the rights of the vulnerable pediatric population.

**Federal guidelines define four categories of pediatric research (Box 2).**

- **Minimal risk** is defined as “the probability and magnitude of harm or discomfort anticipated in the research are not greater in and of themselves than those ordinarily encountered in daily life or during the performance of routine physical or psychological examinations or tests”.

- The category “greater than minimal risk, and no prospect of direct benefit to individual subjects, but likely to yield generalizable knowledge about the subject’s disorder or condition . . . which is of vital importance” defines when it is acceptable to expose a child to a “minor increase over minimal risk”. *Minor increase over minimal*
Adequate justification is needed in subjects who have

Interventions to provide direct diagnostic, therapeutic

Research should be conducted in the settings in

Assent should be obtained to the extent of the child’s

Children will not be included in research that could

the investigator must ensure that:

Research state that before undertaking research in

human subjects issued by the Indian Council of Medical

Scrutinized for all potential risks, including discomfort,

the intervention or procedure presents experiences to subjects that

IRB determines the risk represents a minor increase over minimal risk

The intervention or procedure presents experiences to subjects that

IRB finds and documents that adequate provisions are made for

soliciting assent from children and permission from their parents or

risk has been interpreted as pain, discomfort or stress that is

Studies to be carried out in children must be

Ethical guidelines for the biomedical research on

human subjects issued by the Indian Council of Medical

Research state that before undertaking research in

the investigator must ensure that:

• Children will not be included in research that could

be carried out equally well in adults.

• Assent should be obtained to the extent of the child’s

capability.

• A parent or a legal guardian of each child has given

the proxy consent.

• Research should be conducted in the settings in

which child and parent can obtain adequate medical

and psychological support.

• Interventions to provide direct diagnostic, therapeutic

or preventive benefit of the individual child subject

must be justified in relation to anticipated risks

involved in the study and anticipated benefit to the

society.

• Adequate justification is needed in subjects who have

reduced autonomy (prisoner, students, orphans, etc.).

• Child’s refusal to participate in the research must

always be respected unless there is no medically

acceptable alternative to the therapy provided/
tested, provided the consent has been obtained from

parents/guardians.

The following guidelines should be undertaken before

conducting any research in pediatric population:

1. Participation in drug research should always be of

free choice. It is unethical to obtain agreement for

participation on the basis of coercion, inducement, or

reward.

2. A well-informed consent, permission, assent should

be obtained after explaining all relevant information

in language easily understood by the consentor,

permission giver, and assenter.

The relevant information as decided by the IRB and

investigator to be provided is:

- Aim and method of research
- The expected duration
- The benefit that might be expected as an outcome
to the child or the others
- Any alternative procedures or course of treatment

that might be advantageous to the child as the

procedure or treatment to which he/she is being

subjected
- Confidentiality of the records
- The anticipated consequences of the breach of

confidentiality, identity of the research teams

including their contact details
- Foreseeable extent of the information on possible

current and future uses of the biological material

and of the data to be generated from the research.

If the material is likely to be used for the secondary

purposes or would be shared with others, clear

mention of the same, risk for discovery of the

biologically sensitive information
- Free treatment for research related injuries.

3. The parents, guardians or the child can anytime

withdraw consent or assent at any time during the

research and the investigator should respect their

decision; there would be no penalty or loss of benefits

which the child would otherwise be entitled to.

4. The research would cause no additional costs to the

family.

5. The investigator or IRB would provide compensation

in case of any unforeseen complication.

OBLIGATIONS OF PEDIATRIC
ANESTHESIOLOGISTS

Pediatric anesthesiologists are obligated to patients,
local community and other members of the same faculty.
Chapter 39: Ethical Issues in Pediatric Anesthesia

“Units” of anesthesiologists such as private practitioners, academic departments, and state societies should fulfill the following obligations collectively:

- Be active in national organizations and contribute to growth and development of specialty organizations, like the Indian Association of Pediatric Anesthesiologists (IAPA).
- Teach, conduct research, and support teaching and research in the field of pediatric anesthesia.
- Facilitate high standard quality training by providing state and national level accredited fellowship and degree programs for pediatric anesthesia.
- Participate in the initiatives of relevant professional organizations.
- Foster patient care by surveillance data audits, use of safety check lists, search problems and implement solutions, formulate and comply with policies to improve patient care and safety.

CONCLUSION

Ethical issues in pediatric anesthesia are multi-faceted and challenging to handle. It is an ethical obligation for the pediatric anesthesiologist to involve children in the informed consent process to the maximum extent, depending on their level of understanding and maturity. Proper communication and simple strategies help resolve most issues, improving trust in the relationships among clinicians, the child, and parents. Institutional ethics committee plays an important role in formulating standards and guidelines for providing quality and safe child care services.

SUGGESTED READING

INTRODUCTION

Recent medical literature highlights that over two thirds of serious medical errors were primarily caused by failures in communication. Healthcare providers tend to be trained as individuals, yet function almost exclusively as teams, creating a gap between training and reality.

The sphere of medical education is undergoing a profound change in the new millennium. There is an increasing demand for public accountability and a shift to competency-based curriculum, with an emphasis on more objective, performance-oriented tests of clinical competence. There is decreased acceptance of practicing skills on patients, as well as the development of a culture of safety, with a reduced tolerance for errors.

The following questions have challenged medicine for years. How can clinicians gain experience without putting patients at undue risk? How can we assess the abilities of clinicians as individuals and teams when each patient is unique? These approaches focus on simulation, a technique well known in the military, aviation, space flight, and nuclear power industries.

WHAT IS SIMULATION?

Simulation is a method of training or research that attempts to create a realistic experience in a controlled environment.

Medical simulation mimics clinical care, allowing individual health professionals and teams to inculcate skills and cultures in preparation for safe and effective clinical care, all the while gaining confidence and becoming more efficient. Practice is a key component of learning and maintenance of skills, and is fundamental in many disciplines. Medical education has recently applied the concept of deliberate practice to the acquisition and retention of medical skills.

Simulation offers systematic training, rehearsal, performance assessment, and refinement in practice. Beyond training, simulation may provide indirect ways to improve safety, including facilitating recruitment and retention of skilled personnel, and improving quality and risk management activities (Box 1).

**Box 1: Advantages of simulation training in healthcare**
- No harm to real patient
- Predictable, programmable, standardizable, reproducible scenarios can be presented
- Allows for skill acquisition through repeated practice
- Simulation can be stopped or restarted for teaching
- Videotaping allows for review
- Useful to learn management of rare and infrequent events
- Team training and crisis management can be taught
- Assessment and competency testing possible

TYPES OF SIMULATION (BOX 2)

Fidelity is the extent to which the appearance and behavior of the simulator matches the appearance and behavior of the simulated system. In simpler words, it depicts how close the simulation is to reality. Realism addresses
the question of how closely a replication of a situation resembles the target.

Medical simulators are located on a continuum of fidelity, ranging from simple task trainers to full-body, computer-driven mannequins with sophisticated physiologic features that respond to pharmacologic and mechanical interventions.

Part task trainers are designed to replicate only part of the environment, e.g. arms for phlebotomy practice, intubation models. These are relatively inexpensive.

Computer systems can be used to model aspects of human physiology or pharmacology, simulated tasks or environments and allow interaction with these through a computer interface. These systems generally produce data on student interaction and can provide the student with feedback during or after the interaction, e.g. Gasman for understanding inhalational agent uptake and distribution. Screen-based simulators can more readily be used by a trainee alone, without the need for a tutor or actors, which also reduces cost. The disadvantage of screen-based simulators is that they cannot be used to practice psychomotor skills or teamwork.

Virtual reality and haptic systems present virtual objects or environments to all human senses in a way that is identical to their natural counterpart. Haptic (touch) feedback is used to produce a feeling of resistance when using instruments within the simulated environment, e.g. laparoscopy trainers, epidural space identification using loss of resistance technique.

Standardized patients are real persons trained and calibrated to portray patients with a variety of presenting complaints and pathologies. They are useful in teaching of communication and interpersonal skills.

Simulated Environments
This refers to creation of a realistic working environment used to increase the psychological fidelity of scenarios when using higher-level simulators. Recreating the working environment in which multi-disciplinary teams can work together and create a powerful learning experience.

Integrated Simulators
These are high fidelity patient simulators, which combine sophisticated life-like manikins with computer programs driven by scientifically derived complex mathematical models of human physiology and pharmacology e.g. Human-patient simulator (HPS), PediaSim, Birthing simulator. These simulators allow clinicians to interact with the ‘patient’ as they would in the real clinical environment. Loudspeakers in the manikin’s head create the impression of the ‘patient’ talking, and physical signs including pulses, breath and heart sounds, pupillary reactions and urine output are produced. Physiological signals generated by the manikin are fed to clinical monitors allowing simple (ECG, non-invasive blood pressure, oxygen saturation, end-tidal carbon dioxide) and complex (CVP, pulmonary artery and intracranial pressure, cardiac output) monitoring to be carried out. Injected drugs will be automatically ‘sensed’ and have appropriate effects as in a real patient. These simulators are expensive due to their complexity. Hardware-based simulators usually require more space, as well as other equipment (such as anesthesia machines and defibrillators). The ongoing costs, such as repairs and maintenance, are therefore higher for hardware-based simulators.

The choice of simulator will depend on the intended application and the available budget.

CONCEPTUAL ISSUES ABOUT PATIENT SIMULATION
The key is the program, and not the hardware. Merely creating a realistic simulation does not guarantee meaning or utility. Simulators have to be used in a goal-oriented way. Obtaining the best results, i.e. learning, is as much about the technique as it is about the technology of the simulation devices. Understanding of the conceptual and theoretical aspects of the use of simulation techniques is extremely important for conduct of simulation exercises.

SETTING OF THE SIMULATION EXERCISE
Simulation exercises are theme based, and are generally integrated into a larger training context (often a course or a series of exercises). A simulation exercise is an activity resembling a clinical situation in which participants, role-play the part of medical caregivers. Elaborated below is a typical course flow.

Setting Introduction
The introduction delivers general information on how the exercise will be conducted, the aims and objectives of the
course, logistical information, and some of the known limitations of the course.

**Simulator Briefing or Familiarization**

Participants get familiar with the simulator and the simulated environment through explanations, demonstrations, and hands-on time. They learn how to use the simulator, what it can and cannot do, what is normal (e.g. normal breath sounds), and how they can interact with the environment (e.g. how to call for help, how to request information about the patient that is not directly available in the simulation environment). The familiarization session also is an opportunity to discuss realism issues, e.g. a simulator cannot sweat or demonstrate cyanosis. Here, the “voice of God” (i.e. the simulation scenario controller) is used to indicate such events.

**Theory Input**

Most exercises have didactic theory components on relevant topics. Sometimes this material is made available in advance online exercises. It may be presented before or after a simulation session.

**Case Briefing**

In many simulation scenarios, participants receive a briefing about the upcoming case. Sometimes, this is done explicitly before entering the scenario; sometimes, it is presented as a natural hand-off of the case from one clinician to another.

**Simulation Scenario**

Most simulation exercises involve a scenario that presents a clinical situation to the participants. Generally, the possible evaluations of the scenario are delineated in advance by instructors, although occasionally an intrinsically challenging situation is started and then allowed to play out naturally, depending on how the participants react. Depending on the response by the participants, the scenario evolves, until the end is called for by the instructor.

**Debriefing**

Debriefing is the process of reviewing a simulation scenario after it is complete to optimize any lessons that can be learned. Debriefing allows the team to learn where errors occurred and how they could potentially have been prevented, but it also allows recognition of areas of appropriate performance. Debriefing has been shown to be a critical element in the observation of improved performance following simulation training. The instructor/facilitator conducts the debriefing session, in as non-judgemental a manner as possible, applying adult learning principles of mutual respect and protection of participants self-esteem. Participants are encouraged to speak and actively involve themselves in the discussion. Learning occurs through self-reflection and analysis of the events that happened during the scenario.

**Ending**

This is a separate final session included to end the course, especially for multiple-scenario courses. This is an opportunity to summarize issues that were covered, to address questions, and to consider how best to apply the principles covered to real patient care.

**RESOURCE MANAGEMENT IS A CRUCIAL SKILL FOR ANESTHESIA PROFESSIONALS**

The concept of resource management is borrowed from the field of aviation. For many years “high-risk industries”, such as aviation, nuclear power and military have recognized that maximizing safety and productivity requires an understanding of individual and group cognitive psychology aimed at changing organizational structure, equipment design, operational protocols, and crew training.

Investigations into airline mishaps revealed that communication errors, inefficient leadership and coordination skills, and faulty decision-making in crisis situations were more often to blame than lack of technical skills. Subsequently, the aviation industry embraced a training philosophy originally called “cockpit resource management” (CRM)—later “crew resource management.” In the CRM approach, crews are instructed not only in the technical aspects of managing crises such as engine fires, but also in how to manage their individual and collective resources to work together optimally as a team.

David Gaba and colleagues pioneered the application of simulation and CRM to anesthesia crisis situations, naming their approach as anesthesia crisis resource management (ACRM). Successful conduct of anesthesia depends on more than just having the requisite medical knowledge and technical skills. These must be translated into effective management of the situation. ACRM encompasses the ability of the anesthesiologist to command and control all the resources at hand to execute the anesthesia as planned and respond to problems that arise. This is, in essence, the ability to translate the
knowledge of what needs to be done into effective team activity in the complex and ill-structured real world of perioperative settings. "Crisis resource management" (CRM) is now widely adopted within the healthcare community (Box 3).

**KEY POINTS**

**Anaesthesia crisis resource management**

Cognitive components of dynamic decision making

- Know the environment
- Anticipate and plan
- Use all available information and cross check it
- Allocate attention wisely
- Mobilize resources
- Use cognitive aids

Team management components

- Call for help early enough to make a difference
- Designate leadership
- Establish role clarity
- Distribute the workload
- Communicate effectively

**Box 3: Advantages of simulation in teaching CRM**

- Allows practice in a controlled, psychologically safe environment
- Allows situations that challenge behavioral aspects e.g. communication, leadership
- Allows self-reflection with feedback
- Allows discussion of hierarchy

**SIMULATION IN PEDIATRIC ANESTHESIA**

Pediatric anesthesia is particularly suited to application of simulation methodologies. We know that children are not down-sized adults, and that they differ from adults in anatomy, physiology, pharmacology as well as psychology. The pediatric anesthesia spectrum includes patients from the premature neonate, to the term neonate, infant, toddler, and adolescent. The pediatric patient, is therefore an individual whose anatomy and physiology is continuously evolving. Hence, the skill-set needed to appropriately manage this diverse group is especially challenging.

In our country, the specialty of pediatric anesthesia is still evolving. Large numbers of children are admitted and treated in general-care settings. Often, non-specialist anesthesiologists are required to care for infants or small children. In hospitals or institutions with low volume of pediatric work, the issue of acquiring and maintaining competency in safely managing pediatric patients is particularly challenging.

The occasional pediatric anesthesiologist, as also students of anesthesia, need specific training to perform seemingly simple tasks such as maintaining airway and ventilation, securing vascular access, and performing regional blocks in children. The inability of the small, preverbal child to collaborate and communicate is another difficulty faced by anesthesiologists caring for children. Communicating with parents, the caregivers, and winning the confidence of the child, where appropriate, is a special skill set needed when managing children.

**PEDIATRIC SIMULATORS**

Earlier, in the absence of pediatric specific simulators, instructors had to make do with manikins designed for young adults. Nowadays, neonate, infant and child simulators are available, thereby increasing the realism and accuracy of pediatric simulation.

PediaSim is a high fidelity full body simulator with highly developed pediatric patient physiological models that generate realistic and automatic responses to clinical interventions specific to pediatric patients (Fig. 1). Combining intricate systems design with flexible, user-oriented software achieves a high-tech, interactive synergy that creates realistic learning experiences.

The ultra-sophisticated system captures the complexities of human physiology with heart and breath sounds, palpable pulses and a myriad of other features that create a true and accurate representation of the human body. The intuitive design of the PediaSim and the realistic physiological models make it easy for instructors as well as learners to use the system.

**Fig. 1:** PediaSim & BabySim.

*Courtesy: Dr DY Patil Medical Simulation Laboratory, Navi Mumbai, Maharashtra*
APPLIcATIONS OF SIMULATION IN PEDIATRIC ANESTHESIA

Clinical Skills
Airway and difficult airway management, central venous and vascular access, chest tube placement, regional anesthetic techniques, ultrasound-guided regional blocks and vascular access.

Conduct of anesthesia, managing pediatric and neonatal ventilation, management of anesthetic emergencies, e.g., loss of airway, laryngospasm, bronchospasm, foreign-body aspiration, hypotension, shock etc.

Team Training
Pediatric advanced life support (PALS), neonatal resuscitation program (NRP), pediatric trauma.

Soft Skills/ Non Technical Skills
Communication with parents and caregivers, breaking bad news

Competency Testing
Competency testing in clinical as well as non-technical skills.

Retraining and Certification
This is especially useful for occasional pediatric anesthesiologists, and in centers with low volume pediatric work.

IN SITU PEDIATRIC SIMULATION
In situ simulation involves scenarios that are run in participants usual clinical environment. The advantages over events run at simulation centers are affordability and easier access to personnel, by bringing the simulation experience to the participants rather than asking them to travel to an offsite simulation center. The working environment can be examined for system flaws, and the participants’ experience is improved by increased credibility and comfort working within their usual setup.

LEARNING POINTS
- Pediatric anesthesia involves a wide range of patients, from the newborn to the adolescent. Anesthesiologists need specific training in clinical skills as well as non-clinical skills to manage this diverse patient group.
- Simulation offers a safe environment in which learners may develop and improve skills through sustained deliberate practice.
- Appropriate simulators must be chosen to address identified learning objectives.
- Integration of simulator-based training with clinical practice is the key to successful delivery of training.

SUGGESTED READING
Appendices

**Appendix 1:** Index of Syndromes and Anesthetic Implications  
**Appendix 2:** Pediatric Drug Index  
**Appendix 3:** Quick Reference Tables and Formulae
## Index of Syndromes and Anesthetic Implications

<table>
<thead>
<tr>
<th>Name</th>
<th>Key characteristics</th>
<th>Anesthetic implications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abetalipoproteinemia</td>
<td>Absent apolipoprotein B, malabsorption of lipids, and neuropathy</td>
<td>Deficiency of fat soluble vitamins, elevated prothrombin time, succinylcholine contraindicated with demyelination</td>
</tr>
<tr>
<td>Achondrogenesis</td>
<td>Defect in bone and cartilage formation, micrognathia, short stature, large cranium, micromelia</td>
<td>Difficult airway, care with positioning, difficult venous access</td>
</tr>
<tr>
<td>Achondroplasia</td>
<td>Early fusion of bones, scoliosis, spinal stenosis, macrocephaly, brain stem compression, short stature, obesity, normal intelligence</td>
<td>Difficult airway, care with positioning, restrictive lung disease</td>
</tr>
<tr>
<td>Adrenogenital syndrome</td>
<td>Deficient synthesis of hydrocortisone and aldosterone, virilization of female. May have salt wasting, hypoglycemia, hypokalemia, hypertension.</td>
<td>Check electrolytes. Hydrocortisone supplementation required</td>
</tr>
<tr>
<td>Alagille syndrome</td>
<td>Congenital heart disease, biliary hypoplasia or atresia, hypertension, renal dysplasia, vitamin K deficiency</td>
<td>Associated portal hypertension, cirrhosis, preoperative cardiac and renal evaluation</td>
</tr>
<tr>
<td>Alport’s syndrome</td>
<td>Nerve deafness, renal failure, myopathy, peripheral neuropathy</td>
<td>Hyperkalemia, preoperative cardiac and renal evaluation</td>
</tr>
<tr>
<td>Alstrom syndrome</td>
<td>Vision and hearing loss, cardiomyopathy, renal dysfunction, obesity, diabetes</td>
<td>Renal and cardiac evaluation. Evaluation for diabetes in older children</td>
</tr>
<tr>
<td>Antley-Bixler syndrome</td>
<td>Craniosynostosis, choanal atresia, proptosis</td>
<td>Airway intervention for choanal atresia, eye protection</td>
</tr>
<tr>
<td>Apert syndrome</td>
<td>Craniosynostosis, raised ICP, mid face hypoplasia, fusion of cervical vertebrae, tracheal stenosis, CHD, mental retardation, renal anomalies</td>
<td>Difficult airway, difficult venous access, cardiac evaluation</td>
</tr>
<tr>
<td>Arthrogryposis multiplex</td>
<td>Multiple contractures, small mandible, short neck, myopathy, neuropathy</td>
<td>Difficult airway, difficult venous access</td>
</tr>
<tr>
<td>Bardet-Biedl syndrome</td>
<td>Obesity, mental retardation, polydactyly, retinitis pigmentosa, hypogenitalism</td>
<td>Difficult venous access, regional blocks due to obesity</td>
</tr>
<tr>
<td>Bartter syndrome</td>
<td>Abnormalities in Na, K, Cl transport, metabolic alkalosis, mental retardation, muscle weakness</td>
<td>Hypokalemia, hypochloremia, ileus</td>
</tr>
<tr>
<td>Beckwith-Wiedemann syndrome (infantile gigantism)</td>
<td>Macroglossia, macrosomia, hypoglycemia, omphalocele, CHD</td>
<td>Persistent neonatal hypoglycemia, increased incidence of Wilm’s tumor, adrenal cysts, hyperplasia of insulin producing pancreatic cells</td>
</tr>
<tr>
<td>Blackfan Diamond syndrome</td>
<td>Congenital red cell hypoplasia, hypoplasmenism, thrombocytopenia</td>
<td>Patients on steroid therapy</td>
</tr>
</tbody>
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<table>
<thead>
<tr>
<th>Name</th>
<th>Key characteristics</th>
<th>Anesthetic implications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Branchio-ocular-facial syndrome</td>
<td>Defects of branchial arch involving eye, face, pseudocleft lip, renal anomalies</td>
<td>Renal evaluation</td>
</tr>
<tr>
<td>Carpenter syndrome</td>
<td>Craniosynostosis, raised ICP, hypoplastic mandible, CHD</td>
<td>Difficult airway, cardiac evaluation</td>
</tr>
<tr>
<td>Cat-eye syndrome</td>
<td>Colobomas of iris, choanal atresia, CHD, anal atresia, renal and radial anomalies</td>
<td>Renal and cardiac evaluation. Radial artery catheterization difficult</td>
</tr>
<tr>
<td>Cerebrocostomandibular syndrome</td>
<td>Micrognathia, small thoracic cage, CHD</td>
<td>Difficult airway, restrictive lung disease</td>
</tr>
<tr>
<td>Charcot-Marie-Tooth syndrome</td>
<td>Peripheral neuropathy, muscle atrophy</td>
<td>Respiratory insufficiency, hyperkalemia, caution with succinylcholine</td>
</tr>
<tr>
<td>CHARGE syndrome</td>
<td>Coloboma of the eye, Heart disease, Atresia of choanae, Retarded growth, Genital anomalies, Ear anomalies</td>
<td>Difficult airway, respiratory distress due to choanal atresia, cardiac evaluation</td>
</tr>
<tr>
<td>Chediak-Higashi syndrome</td>
<td>Defect in granular cells (WBC’s), platelet abnormalities, partial albinism, peripheral neuropathy</td>
<td>Immunodeficiency, risk of infections, hyperkalemia-caution with succinylcholine, stress dose steroids</td>
</tr>
<tr>
<td>CHILD syndrome</td>
<td>Congenital hemidysplasia, ichthyosiform erythroderma, limb defects, CHD, hypomelia, renal agenesis</td>
<td>Difficult venous access, cardiac and renal evaluation</td>
</tr>
<tr>
<td>Cohen syndrome</td>
<td>Micrognathia, mitral valve prolapse, mental retardation, hypotonia, obesity</td>
<td>Difficult airway, SBE prophylaxis</td>
</tr>
<tr>
<td>Cornelia de Lange syndrome</td>
<td>Micrognathia, short neck, CHD, mental retardation, seizures, aspera, GERD, micromelia</td>
<td>Difficult airway, difficult venous access, post operative respiratory complications</td>
</tr>
<tr>
<td>Costello syndrome</td>
<td>Short neck, oral, nasal papillomas, cardiomyopathy, arrhythmias, CHD, mental retardation, swallowing difficulty</td>
<td>Difficult airway, postoperative respiratory complications due to GERD</td>
</tr>
<tr>
<td>Cri du chat syndrome (partial deletion of chromosome 5-p)</td>
<td>High pitched, cat-like cry, micrognathia, laryngeal deformity, microcephaly, CHD, mental retardation</td>
<td>Difficult airway, risk of aspiration, airway obstruction</td>
</tr>
<tr>
<td>Crouzon syndrome</td>
<td>Craniosynostosis, proptosis, hypoplastic maxilla</td>
<td>Difficult airway, care of eyes</td>
</tr>
<tr>
<td>Dejerine-Sottas syndrome</td>
<td>Motor and sensory neuropathy, distal muscle atrophy, autonomic dysfunction</td>
<td>Hyperkalemia, caution with succinylcholine, thermal lability</td>
</tr>
<tr>
<td>Di George syndrome (CATCH 22) syndrome</td>
<td>Abnormalities of thymus, parathyroids, great vessels, choanal atresia, micrognathia, short trachea, CHD, immunodeficiency</td>
<td>Difficult intubation, hypocalcemia, susceptibility to infections</td>
</tr>
<tr>
<td>Down's syndrome (Trisomy 21)</td>
<td>Macroglossia, pharyngeal hypotonia, atlantoaxial instability, small trachea, OSA, duodenal atresia, CHD, congenital hypothyroidism</td>
<td>Perioperative airway obstruction, atlantoaxial subluxation, bradycardia common</td>
</tr>
<tr>
<td>Dubowitz syndrome</td>
<td>Small faces, micrognathia, growth and mental retardation, CHD</td>
<td>Difficult airway</td>
</tr>
<tr>
<td>Edward's syndrome (Trisomy 18E)</td>
<td>CHD, micrognathia, renal anomalies</td>
<td>Difficult airway, evaluate cardiac and renal function</td>
</tr>
<tr>
<td>Ehlers-Danlos syndrome</td>
<td>Collagen abnormality with hyperelasticity and fragile tissues and blood vessels, dissecting aneurysm of aorta</td>
<td>Spontaneous rupture of vessels, GI hemorrhage, cardiac conduction abnormalities, spontaneous pneumothorax</td>
</tr>
<tr>
<td>Ellis-Van Creveld syndrome (chondroectodermal dysplasia)</td>
<td>Skeletal defects, dwarfism, micrognathia, CHD, restrictive lung disease, hepatosplenomegaly</td>
<td>Difficult airway, poor lung function</td>
</tr>
<tr>
<td>Epidermolysis bullosa</td>
<td>Abnormality of dermis and mucous membranes, bullae formation, scarring, limited mouth opening, airway bleeding</td>
<td>Protect skin and mucous membranes; use nonadhesive fixation for venous access, endotracheal tube, monitors Difficult airway due to oral scarring. Care of eyes and pressure points</td>
</tr>
<tr>
<td>Fanconi syndrome</td>
<td>Renal tubular dysfunction progressing to renal failure</td>
<td>Evaluate renal function and electrolytes</td>
</tr>
<tr>
<td>Fetal alcohol syndrome (in utero exposure to alcohol)</td>
<td>Micrognathia, short neck, CHD, mental and growth retardation</td>
<td>Difficult airway</td>
</tr>
<tr>
<td>Fetal hydantoin syndrome (in utero exposure to Dilantin)</td>
<td>Microcephaly, midface hypoplasia, webbed neck, CHD, growth and mental retardation, hirsutism</td>
<td>Difficult airway</td>
</tr>
</tbody>
</table>
## Appendix 1: Index of Syndromes and Anesthetic Implications

<table>
<thead>
<tr>
<th>Name</th>
<th>Key characteristics</th>
<th>Anesthetic implications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fetal rubella syndrome (in utero exposure to rubella infection)</td>
<td>Microcephaly, hearing loss, mental retardation, CHD, anemia, thrombocytopenia in neonates, congenital cataract and glaucoma</td>
<td>Check cardiac function, hemoglobin and platelet count</td>
</tr>
<tr>
<td>Fetal valproate syndrome (in utero exposure to valproic acid)</td>
<td>Meningomyelocele, radial anomalies, CHD</td>
<td>Cardiac evaluation</td>
</tr>
<tr>
<td>Fetal warfarin syndrome (in utero exposure to warfarin)</td>
<td>Microcephaly, mental retardation, seizures, CHD</td>
<td>Upper airway obstruction, cardiac evaluation</td>
</tr>
<tr>
<td>Fragile X syndrome</td>
<td>Acromegaly, large ears, prognathism, mitral valve prolapse, mental retardation, seizures</td>
<td>Cardiac evaluation</td>
</tr>
<tr>
<td>Freeman-Sheldon syndrome (whistling face syndrome)</td>
<td>Progressive myopathy, microstomia, micrognathia, upper airway obstruction, dysphagia, joint contractures</td>
<td>Difficult airway, aspiration risk, hyperkalemia</td>
</tr>
<tr>
<td>Fryns syndrome</td>
<td>Micrognathia, CHD, CDH, renal anomalies, mental retardation</td>
<td>Difficult airway, respiratory distress</td>
</tr>
<tr>
<td>Goldenhar syndrome</td>
<td>Hemifacial microsomia, rib, vertebral, scapular and radial anomalies, microtia with hearing loss, CHD, renal anomalies, hydrocephalus</td>
<td>Difficult mask ventilation and intubation, cardiac and renal evaluation</td>
</tr>
<tr>
<td>Gorlin syndrome</td>
<td>Pigmented and atrophic skin changes, oral papillomas, CHD, GERD, renal dysplasia</td>
<td>Difficult ventilation and intubation. Caution with positioning due to joint hypomobility</td>
</tr>
<tr>
<td>Guillan–Barre syndrome (acute idiopathic polyneuritis)</td>
<td>Progressive, ascending polyneuropathy involving cranial nerves. Autonomic dysfunction with bulbar palsy, hypoventilation, hypotension</td>
<td>Hyperkalemia, avoid succinylcholine, may require support of ventilation and blood pressure</td>
</tr>
<tr>
<td>Holt-Oram syndrome</td>
<td>Upper limb anomalies, CHD (commonly ASD, sudden death due to pulmonary embolus, coronary occlusion)</td>
<td>Conduction abnormalities, rhythm disturbances</td>
</tr>
<tr>
<td>Hunter syndrome (Mucopolysaccharidosis II)</td>
<td>Coarse facial features, soft tissue stiffness of lips and mouth, joint stiffness, coronary artery narrowing, hydrocephalus</td>
<td>Difficult intubation, care with positioning</td>
</tr>
<tr>
<td>Hurler syndrome (Mucopolysaccharidosis I)</td>
<td>Features similar to Hunter syndrome; but severe and rapid clinical course. Mental retardation</td>
<td>Difficult intubation, care with positioning</td>
</tr>
<tr>
<td>Jarcho-Levin syndrome</td>
<td>Dwarfism, limited cervical movement, rib abnormalities, CHD</td>
<td>Difficult intubation, restrictive lung disease</td>
</tr>
<tr>
<td>Jeune syndrome</td>
<td>Thoracic cage deformity, lung hypoplasia, pulmonary HTN, chronic renal failure</td>
<td>Respiratory insufficiency progressing to cor pulmonale</td>
</tr>
<tr>
<td>Kartagener syndrome</td>
<td>Situs inversus, immotile cilia, chronic sinusitis, respiratory tract infections, dextrocardia bronchiectasis, asplenia</td>
<td>Respiratory complications due to thick secretions. Reverse placement of ECG leads and defibrillator pads</td>
</tr>
<tr>
<td>Kasabach-Merritt syndrome</td>
<td>Hemangioma, thrombocytopenia, high output cardiac failure, microangiopathic hemolytic anemia, may develop DIC</td>
<td>Blood and blood products may be needed</td>
</tr>
<tr>
<td>Klinefelter syndrome (47 XXY syndrome)</td>
<td>Tall stature, vertebral collapse due to osteoporosis, hypogonadism, behavioral problems</td>
<td>Care during positioning</td>
</tr>
<tr>
<td>Klippel-Feil syndrome</td>
<td>Congenital fusion of cervical vertebrae, short neck, limited cervical mobility, micrognathia, CHD, renal anomalies</td>
<td>Difficult intubation, neurologic injury with neck hyperextension</td>
</tr>
<tr>
<td>Klippel-Trenaunay-Weber syndrome</td>
<td>Unilateral extremity hypertrophy, arteriovenous fistula, hemangiomas, high output multiple joint failure</td>
<td>Bleeding from arteriovenous fistula, thrombocytopenia. Caution with central neuraxial block</td>
</tr>
<tr>
<td>Larsen syndrome</td>
<td>Flat face, subglottic stenosis, tracheomalacia, CHD, cervical spine instability, multiple joint dislocations</td>
<td>Care with neck extension, intubation, positioning, smaller size endotracheal tube</td>
</tr>
<tr>
<td>Laurence-Moon-Biedl syndrome</td>
<td>Obesity, retinitis pigmentosa, polydactyly, mental retardation</td>
<td>Association with cardiac defects, renal dysfunction, diabetes insipidus</td>
</tr>
<tr>
<td>LEOPARD syndrome</td>
<td>Lentigines (multiple large freckles), ECG abnormalities, Ocular hypertension, Pulmonary stenosis, Abnormal genitalia, Retarded growth, Deafness</td>
<td>Cardiac evaluation</td>
</tr>
</tbody>
</table>
### Name | Key characteristics | Anesthetic implications
--- | --- | ---
Lesch-Nyhan syndrome | Developmental delay, hypertonia, spasticity, self-mutilation, hyperuricemia, renal stones, megaloblastic anemia | Check hematocrit and renal function
Liddle syndrome | Na-channel defect; HTN, hypokalemic metabolic alkalosis, renal failure | Check blood pressure, electrolytes, and renal function
Lowe syndrome | Cataract, mental retardation, renal failure, rickets | Check renal function, electrolytes (calcium)
Lown-Ganong-Levine syndrome | Accessory cardiac conduction pathway; atrial tachyarrhythmias | Cardiac evaluation. Reentrant tachycardia treated with adenosine or β blockade
Maffucci syndrome | Enchondromatosis and hemangiomas with malignant change | Pathologic fractures, bleeding from hemangiomas and gastrointestinal tract; sensitivity to vasodilator drugs
Marfan's syndrome (arthro也知道）dactyly) | Connective tissue disorder, pectus excavatum, dilatation of aorta and pulmonary artery, mitral valve prolapse, aortic regurgitation, joint and lens dislocation | Poor lung function, risk of pneumothorax with IPPV, care with myocardial depressant drugs, caution during positioning
Maroteaux-Lamy syndrome (Mucopolysaccharidosis IV) | Short stature, decreased joint mobility, kyphoscoliosis, OSA, recurrent respiratory infections, heart failure, hepatosplenomegaly, anemia, thrombocytopenia | Poor cardiopulmonary reserve, difficult intubation
McAndie syndrome (Glycogen storage disease V) | Myopathy, possible cardiac involvement | Cardiac evaluation, caution with succinylcholine
McCune-Albright syndrome | Fibrous dysplasia of bones, café-au-lait spots, precocious puberty, possible thyrotoxicosis and hyperadrenalism | Caution with positioning, difficult IV access in cushingoid patients
McKusick-Kaufman syndrome | Polydactyly, hydrometrocolpos, CHD (ASD, VSD, complex CHD) | Cardiac evaluation
Meckel-Gruber syndrome | Microcephaly, micrognathia, short neck, CHD, occipital encephalocele, hydrocephalus, renal dysplasia | Difficult intubation, cardiac and renal evaluation, may be on anticonvulsant medication
McKusick-Kaufman syndrome | Microcystic dysplasia of the kidney, renal failure, cardiac anomalies | Difficult intubation, care with positioning
Menkes syndrome | Abnormal copper transport, characteristic kinky hair, microcephaly, seizures, GERD | Difficult ventilation and intubation
MERRF syndrome | Myoclonus, epilepsy, ragged red fibres-defect in mitochondrial DNA, hearing loss, CNS degeneration | May have lactic acidosis, caution with succinylcholine
Miller syndrome (craniofacial syndrome) | Malar hypoplasia, eyelid colobomas, micrognathia, CHD, limb and renal anomalies | Difficult ventilation and intubation, cardiac and renal evaluation, difficult IV access
Mobius syndrome (congenital facial diplegia) | 6th and 7th cranial nerve palsy, micrognathia, feeding difficulties and aspiration, CHD, limb defects | Difficult intubation, excessive secretions, perioperative hypoventilation and apnea
Morquio syndrome (Mucopolysaccharidosis IV) | Short neck, reduced neck and spine mobility, atlantoaxial instability, thoracic deformities, short stature, aortic valve pathology | Difficult intubation, care with positioning
Multiple pterygium syndrome | Multiple pterygium, micrognathia, reduced neck mobility, CHD rare | Difficult intubation, MH association possible
Myotonia congenita | Anterior horn cell degeneration | Respiratory depression with narcotics, anesthetic agents. Caution with neuromuscular blocking drugs
Nager syndrome (Mandibulofacial dysostosis) | Absent zygomatic arches, down sloping palpebral fissures, colobomas, micrognathia, hearing loss, CHD, radioulnar synostosis | Very difficult intubation, difficult IV access
Noack's syndrome | Craniosynostosis, digital anomalies, obesity | Difficult intubation
Neu-Laxova syndrome | Microcephaly, micrognathia, short neck, canine facies, cardiac and renal anomalies | Difficult intubation, cardiac and renal evaluation

Contd...
### Appendix 1: Index of Syndromes and Anesthetic Implications

<table>
<thead>
<tr>
<th>Name</th>
<th>Key characteristics</th>
<th>Anesthetic implications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neurofibromatosis (von Recklinghausen disease)</td>
<td>Neurofibromas of central and peripheral nervous system, café-au-lait spots, association with pheochromocytoma, bone lesions</td>
<td>Airway and neuraxial space involvement, sensitivity to neuromuscular blocking drugs</td>
</tr>
<tr>
<td>Noonan syndrome</td>
<td>Webbed neck, low set ears, micrognathia, pectus excavatum, CHD—pulmonary stenosis, chylothorax, lymphedema, mental retardation, coagulation disorders, renal dysfunction</td>
<td>Difficult intubation, difficult IV access, possible bleeding diathesis</td>
</tr>
<tr>
<td>Oculodentodigital syndrome</td>
<td>Micro-ophthalmia, cleft lip or palate, micro-or macrognathia, small nose, syndactyly</td>
<td>Difficult intubation</td>
</tr>
<tr>
<td>Oral-facial-digital syndrome</td>
<td>Cleft lip and palate, micrognathia, choanal atresia, mental retardation, hydrocephalus, digital anomalies, polycystic kidney disease, affects females</td>
<td>Upper airway obstruction, difficult intubation</td>
</tr>
<tr>
<td>Olsr-Weber-Rendu syndrome (hereditary hemorrhagic telangiectasia)</td>
<td>Vascularpathy with multiple telangiectases, pulmonary AV fistula with right-left shunting, paradoxical emboli, CNS AV fistula and aneurysms, gastrointestinal bleeding</td>
<td>Severe bleeding from nose, lung, brain, GI tract. Care with airway, avoid nasal instrumentation and neuraxial blocks</td>
</tr>
<tr>
<td>Pallister Hall syndrome</td>
<td>Hypothalamic hamartoblastoma, panhypopituitarism, thyroid hypoplasia, micrognathia, cleft lip and palate, dysplastic tracheal cartilage, lung hypoplasia, CHD, imperforate anus</td>
<td>Difficult intubation, thorough evaluation of hypothalamic-pituitary axis, perioperative steroids</td>
</tr>
<tr>
<td>Pearson syndrome</td>
<td>Sideroblastic anemia, exocrine pancreatic dysfunction</td>
<td>Check electrolytes and hematocrit</td>
</tr>
<tr>
<td>Pendred's syndrome</td>
<td>Deafness, goiter, deranged thyroxine production</td>
<td>Evaluate thyroid function</td>
</tr>
<tr>
<td>Pentalogy of Cantrell</td>
<td>Defect in ventral midline growth; cystic hygroma, cleft lip, palate, CDH, pulmonary hypoplasia, omphalocoele, renal and cardiac anomalies</td>
<td>Evaluate for renal, cardiac function and pulmonary HTN</td>
</tr>
<tr>
<td>Peter's plus syndrome</td>
<td>Ophthalmic anomalies, micrognathia, dwarfism, developmental delay, CHD</td>
<td>Difficult intubation, caution with NMB in patients on anticonvulsant drugs</td>
</tr>
<tr>
<td>Pfeiffer's syndrome (Acrocephalosyndactyly)</td>
<td>Coronal and sagittal synostosis, mid face hypoplasia, proptosis, OSA, CHD, broad thumbs and great toes, fused elbows, normal intelligence</td>
<td>Difficult intubation, raised ICP</td>
</tr>
<tr>
<td>Pierre Robin syndrome</td>
<td>Cleft palate, micrognathia, glossoptosis, CHD</td>
<td>Difficult intubation, prone positioning and tongue stitch advised postoperatively</td>
</tr>
<tr>
<td>Pompe's disease (glycogen storage disease)</td>
<td>Macroglossia, respiratory and swallowing muscle weakness, cardiomegaly, hypotonia</td>
<td>Difficult ventilation and intubation, risk of perioperative respiratory complications</td>
</tr>
<tr>
<td>Porphyria (Heme synthesis defect)</td>
<td>Precipitating factors—infecion, starvation, drugs, pregnancy, Neuropathy, psychiatric problems, electrolyte abnormalities. May have hepatic dysfunction. Symptoms during acute attack-abdominal pain, hypertension</td>
<td>Minimize preoperative fasting. Avoid barbiturates, etomidate, pentazocine. Safe anesthetics—Inhalational agents, opioids, propofol, ketamine, muscle relaxants Aspiration risk</td>
</tr>
<tr>
<td>Potter syndrome</td>
<td>Secondary to oligohydramnios. Low set ears, beaked nose, micrognathia, hypoplasia, renal agenesis</td>
<td>Difficult intubation, risk of respiratory failure</td>
</tr>
<tr>
<td>Prader-Willi syndrome (partial deletion of chromosome 15)</td>
<td>Obesity, OSA, hypotonia, developmental delay, hypogonadism, pulmonary HTN, diabetes mellitus</td>
<td>Difficult mask ventilation, intubation. Decreased FRC, respiratory complications. Monitor glucose</td>
</tr>
<tr>
<td>Prolonged Q-T interval syndrome (Jervell-Lange-Nielsen syndrome)</td>
<td>Prolonged Q-T on ECG, congenital deafness</td>
<td>Tachyarrhythmias, sudden death, torsades de pointes Avoid drugs that prolong Q-T interval (volatile anesthetics), avoid epinephrine</td>
</tr>
<tr>
<td>Proteus syndrome</td>
<td>Hemihypertrophy, hypertrophic cardiomyopathy, emphysema of lung, cervical spine and renal anomalies</td>
<td>Evaluate cardiopulmonary and renal function</td>
</tr>
<tr>
<td>Prune belly syndrome (Eagle-Barrett syndrome)</td>
<td>Deficient abdominal wall musculature, cryptorchidism, lung hypoplasia secondary to in utero urinary tract obstruction, urinar tract dilatation, CHD</td>
<td>Risk of perioperative respiratory complications</td>
</tr>
<tr>
<td>Rett syndrome</td>
<td>Females affected. Progressive encephalopathy, seizures, spasticity, behavioral problems</td>
<td>On anticonvulsant medication</td>
</tr>
</tbody>
</table>

Contd...
<table>
<thead>
<tr>
<th>Name</th>
<th>Key characteristics</th>
<th>Anesthetic implications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Riley-Day syndrome (Familial dysautonomia)</td>
<td>Deficiency of dopamine hydroxylase. Hypersensitivity to catecholamines—blood pressure lability. Decreased respiratory drive to hypoxia and hypercarbia, decreased pain sensation. Reduced sweating and tear formation</td>
<td>Risk of aspiration. Increased response to inotropes and narcotics. Reduced analgesic requirements</td>
</tr>
<tr>
<td>Riley-Smith syndrome (lipid storage myopathy)</td>
<td>Macrocephaly, developmental delay</td>
<td>Caution with NMB</td>
</tr>
<tr>
<td>Robinow syndrome (fetal face syndrome)</td>
<td>Frontal bossing, micrognathia, CHD, renal and genitourinary abnormalities</td>
<td>Difficult intubation, check renal and cardiac function.</td>
</tr>
<tr>
<td>Romano-Ward syndrome</td>
<td>Prolonged Q-T interval, sudden heart block</td>
<td>Arrhythmias. Avoid drugs that prolong Q-T interval</td>
</tr>
<tr>
<td>Rubella syndrome</td>
<td>Mental retardation, deafness, CHD</td>
<td>Cardiac evaluation</td>
</tr>
<tr>
<td>Rubinstein-Taybi syndrome</td>
<td>Defect in CAMP mediated induction of protein synthesis</td>
<td>Difficult intubation, caution with NMB, may have delayed recovery from anaesthesia</td>
</tr>
<tr>
<td>Russell-Silver syndrome</td>
<td>Short stature, micrognathia, café-au-lait spots, tendency to hypoglycemia</td>
<td>Difficult intubation, monitor glucose</td>
</tr>
<tr>
<td>Sanfilippo syndrome (mucopolysaccharidosis III)</td>
<td>Coarse facies, mental retardation, seizures</td>
<td>Difficult intubation, caution with NMB in patients on anticonvulsants</td>
</tr>
<tr>
<td>Scheie's syndrome (mucopolysaccharidosis I)</td>
<td>Corneal clouding, glaucoma, joint stiffness, OSA, aortic valve involvement, hernias</td>
<td>Difficult intubation due to macroglossia, difficult IV access due to thick skin and contractures</td>
</tr>
<tr>
<td>Schinzel-Giedion syndrome</td>
<td>Growth and mental deficiency, seizures, CHD, renal anomalies, choanal atresia</td>
<td>Cardiac and renal evaluation caution with NMB</td>
</tr>
<tr>
<td>Schwartz-Jampel syndrome</td>
<td>Myotonia due to sodium channel defect. Micrognathia, joint contractures</td>
<td>Difficult intubation. Caution with succinylcholine. Susceptible to MH</td>
</tr>
<tr>
<td>Scimitar syndrome</td>
<td>Partial anomalous venous return, hypoplastic right lower lobe, pulmonary HTN, dextrocardia</td>
<td>Recurrent pulmonary infections</td>
</tr>
<tr>
<td>Shwachman syndrome</td>
<td>Metaphyseal chondrodysplasia, hypotonia, short stature, pancreatic insufficiency, pancytopenia, immunologic abnormalities</td>
<td>Monitor hematocrit and platelet count</td>
</tr>
<tr>
<td>Shy-Drager syndrome</td>
<td>Degeneration of CNS and autonomic nervous system, orthostatic hypotension, decreased sweating, hypersensitivity to epinephrine</td>
<td>Labile pulse and blood pressure, caution with halothane and isoflurane. Treatment of hypotension with phenylephrine.</td>
</tr>
<tr>
<td>Silver's syndrome</td>
<td>Short stature, skeletal asymmetry, micrognathia, abnormal sexual development</td>
<td>Difficult intubation</td>
</tr>
<tr>
<td>Sipple's syndrome (MEN type II)</td>
<td>Pheochromocytoma, medullary thyroid carcinoma, parathyroid adenoma, CNS tumors, schwannoma of mediastinum, Cushing's disease</td>
<td>Problems of multiple endocrine disorders, pheochromocytomas often bilateral. Evaluate for mediastinal mass</td>
</tr>
<tr>
<td>Soto's syndrome</td>
<td>Cerebral gigantism, mental retardation, CHD</td>
<td>Cardiac evaluation</td>
</tr>
<tr>
<td>Stevens-Johnson syndrome</td>
<td>Erythema multiforme, urticarial lesions, erosions of mouth, eyes, genitalia. Hypersensitivity to drugs, infections</td>
<td>Placement of monitors difficult, intubation trauma due to lesions. Care with positioning.</td>
</tr>
<tr>
<td>Stickler syndrome</td>
<td>Flat facies, hearing loss, cleft palate, micrognathia, pectus excavatum, hyperextensible joints</td>
<td>Difficult intubation</td>
</tr>
<tr>
<td>Sturge-Weber syndrome</td>
<td>Capillary hemangiomas over distribution of 5th cranial nerve, port wine stain, glaucoma, progressive neurologic deficit, seizures</td>
<td>Difficult intubation due to hemangiomas in airway</td>
</tr>
<tr>
<td>TAR (Thrombocytopenia-absent radius) syndrome</td>
<td>Thrombocytopenia precipitated by stress, infection, surgery, CHD, bilateral absent radial bones</td>
<td>Check platelet count, CHD evaluation</td>
</tr>
<tr>
<td>Treacher Collins syndrome</td>
<td>Mandibulofacial dysostosis, malar hypoplasia, down sloping palpebral fissures, micro-ophthalmia, low-set ears, micrognathia, OSA, CHD</td>
<td>Difficult airway</td>
</tr>
</tbody>
</table>
# Appendix 1: Index of Syndromes and Anesthetic Implications

<table>
<thead>
<tr>
<th>Name</th>
<th>Key characteristics</th>
<th>Anesthetic implications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trisomy 8 syndrome</td>
<td>Strabismus, micrognathia, webbed neck, CHD, mental retardation, seizures</td>
<td>Difficult intubation, caution with NMB, opioids in patients on anticonvulsants</td>
</tr>
<tr>
<td>Trisomy 9 syndrome</td>
<td>Microcephaly, mental retardation, micrognathia, cleft lip, palate, CHD</td>
<td>Difficult intubation, caution with NMB, opioids in patients on anticonvulsants</td>
</tr>
<tr>
<td>Trisomy 13 (Patau) syndrome</td>
<td>Occipital scalp defect, low set ears, micrognathia, cleft lip, palate, CHD, mental retardation, renal anomalies, hemangiomas, radial anomalies</td>
<td>Difficult intubation, caution with NMB, opioids in patients on anticonvulsants, difficult placement of radial catheter</td>
</tr>
<tr>
<td>Trisomy 18 (Edwards) syndrome</td>
<td>Prominent occiput, microcephaly, micrognathia, short sternum, clenched hands, rocker bottom feet, CHD, renal anomalies</td>
<td>Difficult intubation, check renal and cardiac function</td>
</tr>
<tr>
<td>Turner’s syndrome (single X chromosome)</td>
<td>Micrognathia, short webbed neck, broad chest, short stature, CHD (coarctation), HTN, Hypothyroidism, lymphedema of hands and feet, renal anomalies</td>
<td>Difficult intubation, difficult iv access, check renal and cardiac function</td>
</tr>
<tr>
<td>VACTER association (Trisomy 18)</td>
<td>Vertebral anomalies, Anal atresia, CHD, Tracheoesophageal fistula, Esophageal atresia, Radial and renal anomalies</td>
<td>Check radiographs of vertebrae, radii, check renal and cardiac function</td>
</tr>
<tr>
<td>Velocardiofacial (Sprintzen) syndrome (deletion of chromosome 22)</td>
<td>Microcephaly, micrognathia, CHD, developmental delay, T cell immunodeficiency, hypocalcemia</td>
<td>Difficult intubation, cardiac evaluation, irradiated blood if T cell immunodeficiency, check calcium</td>
</tr>
<tr>
<td>Von Hippel Lindau syndrome</td>
<td>Retinal, CNS and visceral hemangioblastomas, association with pheochromocytoma, may have renal, pancreatic or hepatic cysts, cerebellar tumors</td>
<td>Evaluation for pheochromocytoma, may have raised ICP</td>
</tr>
<tr>
<td>Walker-Warburg (HARD) syndrome</td>
<td>Retinal defects, microtia, cleft lip and palate, CNS anomalies, hydrocephalus, mental retardation, muscular dystrophy, seizures</td>
<td>May have raised IOP, ICP. Caution with NMB, opioids in patients on anticonvulsants, Caution with succinylcholine in muscular dystrophy</td>
</tr>
<tr>
<td>Watson syndrome</td>
<td>Valvular pulmonary stenosis, neurofibromas, café au lait spots, mental retardation</td>
<td>Cardiac evaluation</td>
</tr>
<tr>
<td>Weaver syndrome</td>
<td>Macrocephaly, large tongue, short neck, developmental delay, seizures, behavioural problems</td>
<td>Difficult intubation, caution with NMB, opioids in patients on anticonvulsants</td>
</tr>
<tr>
<td>Weill-Marchesani syndrome</td>
<td>Glaucoma, blindness, sub valvular aortic stenosis</td>
<td>Cardiac evaluation</td>
</tr>
<tr>
<td>Williams-Beuren syndrome (deletion of elastin gene on chromosome 7)</td>
<td>Elfin facies, severe CHD, sudden death, developmental delay, hypercalcemia</td>
<td>Cardiac evaluation</td>
</tr>
<tr>
<td>Wiskott-Aldrich syndrome</td>
<td>Recurrent pulmonary infections, vasculitis-coronary, cerebral arteries, renal dysfunction, thrombocytopenia, immune deficiency, eczema</td>
<td>Check hematocrit, platelet count. Irradiate blood products. Cardiac and renal evaluation. Stress dose steroids</td>
</tr>
<tr>
<td>Wolf-Parkinson-White (WPW) syndrome</td>
<td>Reentrant tachycardia, structural heart disease</td>
<td>Adenosine and β blockade for tachycardia</td>
</tr>
<tr>
<td>Zellweger syndrome (abnormal peroxisomes)</td>
<td>Micrognathia, CHD, hypotonia, respiratory insufficiency, adrenal atrophy, contractures</td>
<td>Cardiac evaluation, Stress dose steroids, care with positioning</td>
</tr>
</tbody>
</table>

Abbreviations: CHD, congenital heart disease; CNS, central nervous system; GERD, gastroesophageal reflux disease; HTN, hypertension; ICP, intracranial pressure; IOP, intraocular pressure; MH, malignant hyperthermia; NMB, neuromuscular blockers; OSA, obstructive sleep apnea; AV fistula, arteriovenous fistula
Drugs and dosages are mentioned as general guidelines only. Dosages should be adjusted based on clinical situation: Hepatorenal function, cardiopulmonary and other factors.

### ANTIBIOTICS

<table>
<thead>
<tr>
<th>Drug</th>
<th>Description</th>
<th>Side effects, caution©</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ampicillin</td>
<td>Broad-spectrum Penicillin-Endocarditis Prophylaxis</td>
<td>Nausea, diarrhea, rash</td>
<td>IV 10–25 mg/kg, qds</td>
</tr>
<tr>
<td>Amoxicillin + Clavulanic acid (Augmentin)</td>
<td>Broad spectrum</td>
<td>Rash, gastrointestinal upset</td>
<td>IV 25–50 mg/kg tds</td>
</tr>
<tr>
<td>Cefazolin (Reflin)</td>
<td>1st generation Cephalosporin</td>
<td>Hypersensitivity,© Renal impairment</td>
<td>IV 25 mg/kg tds</td>
</tr>
<tr>
<td>Cefaclor</td>
<td>1st generation Cephalosporin</td>
<td>Oral only</td>
<td></td>
</tr>
<tr>
<td>Cefadroxil</td>
<td>1st generation Cephalosporin</td>
<td>Oral only</td>
<td></td>
</tr>
<tr>
<td>Ceftriazone</td>
<td>2nd generation Cefalosporin</td>
<td>Allergy</td>
<td>IV 20–30 mg/kg tds</td>
</tr>
<tr>
<td>Cefotaxime</td>
<td>3rd generation Cephalosporin</td>
<td>Allergic reaction, ©Severe renal failure</td>
<td>IV 25 mg/kg tds 50 mg/kg qds in severe infections</td>
</tr>
<tr>
<td>Ceftriaxone</td>
<td>3rd generation Cephalosporin</td>
<td>IV 50–100 mg/kg once daily</td>
<td></td>
</tr>
<tr>
<td>Clindamycin (Cleocin)</td>
<td>Aminoglycoside Endocarditis prophylaxis</td>
<td>Thrombophlebitis, anaphylaxis, ©Renal dysfunction, neutropenia</td>
<td>IV 5–10 mg/kg qds slowly</td>
</tr>
<tr>
<td>Gentamicin</td>
<td>Aminoglycoside Endocarditis prophylaxis</td>
<td>Nephrotoxicity Ototoxicity</td>
<td>1.5–2.5 mg/kg tds Neonates: 2 mg/kg 8–12 hrly</td>
</tr>
<tr>
<td>Vancomycin (used as bactericidal in severe infection)</td>
<td>Glycopeptide against aerobic, anaerobic gram +ve bacteria</td>
<td>©Renal impairment, phlebitis, ototoxicity, hypotension</td>
<td>10-20 mg/kg slowly over 1 h dose—6–8 hrly</td>
</tr>
<tr>
<td>Metronidazole</td>
<td>For anaerobic bacterial infection</td>
<td>Nausea, vomiting</td>
<td>7.5 mg/kg slowly qds</td>
</tr>
<tr>
<td>Tobramycin</td>
<td>Anaphylaxis, nephrotoxicity</td>
<td>2.5–5 mg/kg tds slowly over 30 mins</td>
<td></td>
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</table>

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### Appendix 2: Pediatric Drug Index

#### Anticonvulsants

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<th>Drug</th>
<th>Description</th>
<th>Side effects, caution©</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amikacin</td>
<td>Aminoglycoside</td>
<td>Less nephrotoxic than gentamicin in preterm babies</td>
<td>&lt;28 PCW 7.5 mg/kg/24 h&lt;br&gt;&lt;32 PCW 7.5 mg/kg/16 h&lt;br&gt;&lt;37 PCW 10 mg/kg/12 h&lt;br&gt;&gt;1 month 7.5 mg/kg/12 h</td>
</tr>
<tr>
<td>Phenytoin (Eptoin)</td>
<td>50 mg/mL</td>
<td>Induces oxidative metabolism</td>
<td>10–20 mg/kg slowly (max 1–3 mg/kg/min)</td>
</tr>
<tr>
<td>Diphenyl hydantoin (Dilantin)</td>
<td>Hydantoins</td>
<td>Hypotension, skin necrosis, ataxia</td>
<td>12–20 mg/kg over 30 mins</td>
</tr>
<tr>
<td>Phenobarbitone</td>
<td>Hepatic microsomal enzyme induction</td>
<td>Sedation</td>
<td>20 mg/kg IV</td>
</tr>
<tr>
<td>Valproic acid</td>
<td>Enhances hepatic oxidation</td>
<td>©Liver impairment</td>
<td>20–40 mg/kg IV</td>
</tr>
<tr>
<td>Carbamazepine</td>
<td></td>
<td>©Liver impairment</td>
<td>10–20 mg/kg PO</td>
</tr>
</tbody>
</table>

**Note:** Benzodiazepines—Midazolam and Diazepam can be used as anticonvulsants.

### Antiemetics

<table>
<thead>
<tr>
<th>Drug</th>
<th>Description</th>
<th>Side effects, caution©</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metoclopramide (Perinorm)</td>
<td>Increases gastric emptying and lower esophageal sphincter tone (Benzamide)</td>
<td>Extrapyramidal signs, inhibits plasma cholinesterase</td>
<td>0.1–0.2 mg/kg qds</td>
</tr>
<tr>
<td>Ranitidine</td>
<td>H₂ receptor antagonist Reduction in gastric acid secretion</td>
<td>Tachycardia ©-in porphyria</td>
<td>1 mg/kg slowly tds</td>
</tr>
<tr>
<td>Ondansetron</td>
<td>5-HT₄ receptor antagonist, more selective, longer elimination half-life</td>
<td>Flushing, headache, hypotension</td>
<td>0.1 mg/kg slowly tds</td>
</tr>
<tr>
<td>Granisetron</td>
<td>5-HT₄ receptor antagonist, more selective, longer elimination half-life</td>
<td></td>
<td>20–40 μg/kg 12 hrly</td>
</tr>
<tr>
<td>Droperidol</td>
<td>Butyrophenone Dopamine receptor antagonist</td>
<td>Sedation, drowsiness, agitation, extrapyramidal reaction</td>
<td>50–75 μg/kg</td>
</tr>
<tr>
<td>Promethazine (Phenergan)</td>
<td>Phenothiazine group</td>
<td>Sedation</td>
<td>IV/IM 0.1–0.5 mg/kg qds</td>
</tr>
<tr>
<td>Prochlorperazine</td>
<td>Phenothiazine group</td>
<td>Extrapyramidal signs, sedation, hypotension</td>
<td>0.1/mg/kg IM 0.4 mg/kg/day -divided doses</td>
</tr>
</tbody>
</table>

### Antipruritics

<table>
<thead>
<tr>
<th>Drug</th>
<th>Description</th>
<th>Side effects, caution©</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diphenhydramine (Benadryl)</td>
<td>H₁ receptor inhibition Antihistaminic</td>
<td>Sedation, paradoxical excitement</td>
<td>0.5 mg/kg qds IV&lt;br&gt;1.25 mg/kg 6 hrly oral</td>
</tr>
<tr>
<td>Hydroxyzine (Atarax)</td>
<td></td>
<td>Drowsiness, thrombosis, ©Renal impairment</td>
<td>0.5–1 mg/kg qds</td>
</tr>
<tr>
<td>Nalbuphine</td>
<td>Synthetic narcotic agonist-antagonist analgesic</td>
<td>Sedation</td>
<td>0.1 mg/kg slowly over 20 mins</td>
</tr>
</tbody>
</table>
### BRONCHODILATORS

<table>
<thead>
<tr>
<th>Drug</th>
<th>Description</th>
<th>Side effects, Caution ©</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aminophylline</td>
<td>Methylxanthine</td>
<td>Tachycardia, nausea, vomiting, restlessness</td>
<td>Loading dose 5–7 mg/kg IV slow over 1 h infusion—0.5–1.5 mg/kg/h PO 100 mg/kg 6 hry</td>
</tr>
<tr>
<td>Isoproterenol (Isoprenaline)</td>
<td>Synthetic catecholamine β-receptor agonist, ionotrophic, chronotropic, vasodilator</td>
<td>Tachycardia, arrhythmias</td>
<td>1 μg/kg IV bolus Then 0.05 μg/kg/min infusion</td>
</tr>
<tr>
<td>Epinephrine (1:1,000) Racemic epinephrine</td>
<td>Endotracheal nebulization 100 μg/kg</td>
<td>Tachycardia, Palpitations</td>
<td>10 μg/kg SC 0.05 μg/kg/min infusion</td>
</tr>
<tr>
<td>Salbutamol (Albuterol)</td>
<td>β-receptor agonist</td>
<td>Tachycardia, tremors, hypokalemia</td>
<td>5 μg/kg IV bolus over 10 mins, infusion 1–5 μg/kg/min</td>
</tr>
<tr>
<td>Terbutaline</td>
<td></td>
<td>Hypertension, arrhythmias, palpitations, hypokalemia, muscle cramps</td>
<td>10 μg/kg SC Max 250 μg</td>
</tr>
</tbody>
</table>

Note: Initial treatment of bronchospasm comprises nebulization with
(1) Albuterol 0.5% solution—0.01 mL/kg/2.5 mL NS
   6 mg/kg in 500 mL of NS for nebulization 20 mL/h gives dose of 4 μg/kg/min Or
(2) Metaproterenol 5% solution—0.01 mL/kg/2.5 mL NS Or
(3) Terbutaline 10 μg/kg SC

### ANTIHYPERTENSIVES

<table>
<thead>
<tr>
<th>Drug</th>
<th>Description</th>
<th>Side effects, caution ©</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Esmolol</td>
<td>Cardioselective β-blocker</td>
<td>Bradycardia, ©Asthma, heart failure</td>
<td>0.5 mg/kg IV bolus over 1 min, infusion 50–300 μg/kg/min</td>
</tr>
<tr>
<td>Propranolol</td>
<td>Nonselective β-adrenergic antagonist</td>
<td>©Asthma, bronchospasm, WPW Syndrome, AV block, verapamil</td>
<td>0.01–0.15 mg/kg IV slowly</td>
</tr>
<tr>
<td>Hydralazine</td>
<td>Direct acting arteriolar vasodilator</td>
<td>Tachycardia</td>
<td>0.1–0.2 mg/kg IV qds</td>
</tr>
<tr>
<td>Phentolamine (Regitine)</td>
<td>α, and α, adrenergic antagonist Potent vasodilator</td>
<td>Reflex tachycardia, flushing</td>
<td>0.1–0.2 mg/kg IV then, infusion 0.25–0.5 μg/kg/min</td>
</tr>
<tr>
<td>Labetalol</td>
<td>α (mild) β-adrenergic receptor antagonist</td>
<td>Bradycardia, bronchospasm ©Asthma, AV block, verapamil</td>
<td>0.2–1 mg/kg/h IV infusion</td>
</tr>
<tr>
<td>Nifedipine</td>
<td>Calcium channel Blocker</td>
<td>Flushing, headache</td>
<td>0.01–0.02 mg/kg IV</td>
</tr>
<tr>
<td>Nitroglycerin</td>
<td>Potent vasodilator</td>
<td>Tachycardia, methemoglobinemia</td>
<td>0.5–5 μg/kg/min IV infusion</td>
</tr>
<tr>
<td>Sodium nitroprusside(SNP)</td>
<td>Nitric oxide generating potent peripheral vasodilator</td>
<td>Methemoglobinemia, tachycardia, acidosis, cyanide toxicity</td>
<td>0.3–0.5 μg/kg/min IV Infusion</td>
</tr>
<tr>
<td>Nicardipine</td>
<td>Calcium-channel antagonist Limited effect on ionotropy, chronotropy</td>
<td>Prolonged effect after discontinuation</td>
<td>1–5 μg/kg/min IV Infusion</td>
</tr>
</tbody>
</table>

Abbreviations: WPW, Wolff-Parkinson-White syndrome; AV, atrioventricular.
### CARDIAC DRUGS

<table>
<thead>
<tr>
<th>Drug</th>
<th>Description</th>
<th>Caution©, side effects</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amiodarone</td>
<td>Antiarrhythmic useful in SVT and VT, Tachyarrhythmias class I &amp; III mixed</td>
<td>SA block, thyroid dysfunction, hypotension</td>
<td>5 mg/kg IV bolus over 10 min then infusion—5–10 μg/kg/min</td>
</tr>
<tr>
<td>Digoxin</td>
<td>Cardiac glycoside, weak inotrope</td>
<td>Toxicity in hypokalemia, nausea, fatigue, arrhythmias</td>
<td>4–25 μg/kg IV loading, 5–10 μg/kg/dose IV qds, Then 5 μg/kg bid</td>
</tr>
<tr>
<td>Diltiazem</td>
<td></td>
<td></td>
<td>0.25 mg/kg over 2 mins, repeat after 15 mins</td>
</tr>
<tr>
<td>Dopamine</td>
<td>Catecholamine—α and β, Dopaminergic action, inotropic agent</td>
<td>Tachycardia, ectopy</td>
<td>2–10 μg/kg/min</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>β adrenergic agonist, positive inotrope and chronotrope</td>
<td>Arrhythmia, hypertension</td>
<td>2–10 μg/kg/min</td>
</tr>
<tr>
<td>Adenosine (endogenous nucleoside)</td>
<td>Antiarrhythmic, slow conduction through AV node useful in acute SVT</td>
<td>2nd and 3rd degree heart block and asthma flushing, dyspnea</td>
<td>0.05, mg/kg increase up to 0.3 mg/kg</td>
</tr>
<tr>
<td>Adrenaline</td>
<td>Endogeneous catecholamine—α and β action useful in anaphylaxis, bronchospasm, severe hypotension</td>
<td>Arrhythmias with halothane, S/E— hypertension, tachycardia, hyperglycemia</td>
<td>0.1 mL/kg IV of 1:10,000 (10μ/kg), ET and intraosseous: 0.1 mL/kg of 1:1,000, infusion: 0.05–1 μg/kg</td>
</tr>
<tr>
<td>Phenylephrine</td>
<td>Selective direct-acting α adrenergic agonist causes peripheral vasoconstriction</td>
<td>Reflex bradycardia, arrhythmias</td>
<td>2–10 μg/kg then 1–5 μg/kg/min—infusion</td>
</tr>
<tr>
<td>Vasopressin</td>
<td>Extract from posterior pituitary, used in septic shock and GI bleed</td>
<td></td>
<td>Add 2–5 U in 1L fluid, replace IV= urine output+ 10% or 2-SU—IM or SC</td>
</tr>
<tr>
<td>Milrinone</td>
<td>Selective phosphodiesterase inhibitor—used in cardiac failure</td>
<td>©myopathy, valvular stenosis</td>
<td>20–50 μg/kg over 10 mins, maintenance 0.3–0.7 μg/kg/min—infusion</td>
</tr>
<tr>
<td>Ephedrine</td>
<td>Use—hypotension in central neuraxial block</td>
<td>Bradycardia, confusion</td>
<td>IV 0.2–0.3 mg/kg</td>
</tr>
<tr>
<td>Magnesium sulfate</td>
<td>For torses de pointes, hypomagnesemia, arrhythmias</td>
<td>CNS depression, hypotension, ©heart block, potentiates muscle relaxants</td>
<td>25–50 mg/kg over 10–20 mins</td>
</tr>
<tr>
<td>Metaraminol (Aramine)</td>
<td>Direct acting adrenergic sympathomimetic</td>
<td>Reflex bradycardia arrhythmias, renal perfusion</td>
<td>IV 5–10 μg/kg then 0.1 μg/kg/min—if required</td>
</tr>
<tr>
<td>Calcium chloride</td>
<td>Positive inotrope, uses—hyperkalemia, hypermagnasaemia, 10% contains Ca²⁺ 680 μmol/mL</td>
<td>Arrhythmias, hypertension, necrosis if extravasation</td>
<td>10–30 mg/kg IV slowly</td>
</tr>
<tr>
<td>Calcium gluconate</td>
<td>10% contains Ca²⁺ 220 μmol/mL</td>
<td>Same as calcium chloride</td>
<td>60–100 mg/kg IV slowly</td>
</tr>
</tbody>
</table>

**Abbreviations:** SVT, supraventricular tachycardia; SA block, sinoatrial block; VT, ventricular tachycardia; CNS, central nervous system; GI bleed; gastrointestinal bleed

### RESUSCITATION DRUGS

<table>
<thead>
<tr>
<th>Drug</th>
<th>Description</th>
<th>Caution©/side effects</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atropine</td>
<td>Anticholinergic</td>
<td>Dry mouth, tachycardia</td>
<td>0.01–0.03 mg/kg IV</td>
</tr>
<tr>
<td>Sodium bicarbonate</td>
<td>For acidosis</td>
<td></td>
<td>1–2 mEq/kg IV (0.3 × kg × BE)</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>Amide type Local anesthetic</td>
<td>Arrhythmias, overdose- seizures</td>
<td>1 mg/kg IV, IC 20–50 μg/kg/min ET 3–4 mg</td>
</tr>
<tr>
<td>Dextrose</td>
<td>For hypoglycemia</td>
<td></td>
<td>0.5–1 g/kg IV, 2–4 mL/kg of D 25%</td>
</tr>
<tr>
<td>Dextrose –saline</td>
<td>For hypovolemic and hypoglycemic shock</td>
<td></td>
<td>10–20 mL/kg over 30 min</td>
</tr>
<tr>
<td>Intralipid 20%</td>
<td>For LA toxicity</td>
<td></td>
<td>IV 1.5 mL/kg over 1 min 0.25 mL/kg/min infusion</td>
</tr>
</tbody>
</table>

Contd...
## Drug Description

### Caution / side effects

### Dose

<table>
<thead>
<tr>
<th>Drug</th>
<th>Description</th>
<th>Caution / side effects</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Flumazenil</strong></td>
<td>Benzodiazepine antagonist</td>
<td>Arrhythmias, changes in blood pressure</td>
<td>IV 0.01 mg/kg</td>
</tr>
<tr>
<td><strong>Naloxone (narcan)</strong></td>
<td>Stimulant if opioid overdose</td>
<td>Hypertension</td>
<td>IV/IM 1–10 μg/kg titrated</td>
</tr>
<tr>
<td><strong>Caffeine</strong></td>
<td>Stimulant useful in apnea in neonates</td>
<td>Tachyarrhythmias</td>
<td>PO 5–10 mg/kg</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>IV 5–10 mg/kg</td>
</tr>
</tbody>
</table>

### Abbreviations:
- LA, Local anesthetic

## ANALGESICS

### Drug-NSAIDs

<table>
<thead>
<tr>
<th>Drug</th>
<th>Description</th>
<th>Caution / side effects</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Acetaminophen</strong></td>
<td>Tylenol</td>
<td>For pain, fever, available as drops, elixir, tablet, suppository</td>
<td>PO 10–15 mg/kg qds</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hepatotoxicity if used for long period</td>
<td>PR 20–40 mg/kg</td>
</tr>
<tr>
<td>Ibuprofen</td>
<td>Analgesic, anti-inflammatory</td>
<td>Nephrotoxicity, inhibition of platelet aggregation</td>
<td>6–10 mg/kg qds PO</td>
</tr>
<tr>
<td>Keterolac</td>
<td>Prevensts sensitization of peripheral nociceptors by prostaglandins</td>
<td>SE—bradycardia</td>
<td>0.5 mg/kg IV qds</td>
</tr>
<tr>
<td></td>
<td></td>
<td>© bleeding diathesis, renal failure</td>
<td>PO 1 mg/kg qds</td>
</tr>
<tr>
<td>Acetaminophen</td>
<td>Intravenous preparation- Parfalgan</td>
<td></td>
<td>IV 7.5 mg/kg 6 hrly in neonates 10–15 mg/kg 6 hrly in older children</td>
</tr>
<tr>
<td>Aspirin</td>
<td></td>
<td></td>
<td>PO 10–15 mg/kg qds</td>
</tr>
</tbody>
</table>

### Abbreviations:
- SE, side effects

## OPIOIDS

### Drugs

<table>
<thead>
<tr>
<th>Drug</th>
<th>Description</th>
<th>Caution / side effects</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Codein</strong></td>
<td>Oral opioid</td>
<td>Unpredictable analgesia</td>
<td>PO 0.5–1 mg/kg 4 hrly</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>Synthetic opioid, intraoperative analgesia adjunct for induction</td>
<td>Apnea, cough, chest wall rigidity, bradycardia, laryngospasm</td>
<td>0.5–2 μg/kg IV 30 mins to 1 hrly</td>
</tr>
<tr>
<td>Fentanyl-lollipop orale</td>
<td></td>
<td>SEDation, pruritus</td>
<td>PO 5–15 μg/kg</td>
</tr>
<tr>
<td>Meperidine (pethidine)</td>
<td>Synthetic opioids for rigors</td>
<td>Seizures if overdose, No—infusion, © causes renal dysfunction, © liver disease</td>
<td>IV 0.1 mg/kg once for rigors 1–2 mg/kg IV</td>
</tr>
<tr>
<td>Methadone</td>
<td>Sedation, nausea, vomiting, respiratory depression</td>
<td></td>
<td>IV 0.1 mg/kg 8–12 hrly or sliding scale according to severity of pain</td>
</tr>
<tr>
<td>Morphine</td>
<td>Potent analgesic</td>
<td>Apnea, histamine release, nausea, vomiting, sedation</td>
<td>0.05–0.1 mg/kg IV, IM 6–8 hrly 0.3–0.5 mg/kg/dose PO qds</td>
</tr>
<tr>
<td>Remifentanil</td>
<td>Ultrashort-acting</td>
<td>Apnea, bradycardia</td>
<td>0.1–0.5 μg/kg/min by Infusion: 1–4 μg/kg—bolus</td>
</tr>
<tr>
<td>Sufentanil</td>
<td>Short-acting</td>
<td>Chest wall rigidity, apnea</td>
<td>IV 0.25–1 μg/kg—bolus 0.1–1 μg/kg/hr—infusion</td>
</tr>
<tr>
<td>Hydromorphone</td>
<td>Potent analgesic</td>
<td>Nausea, vomiting, respiratory depression</td>
<td>IV 0.01–0.015 mg/kg</td>
</tr>
<tr>
<td>Tramadol</td>
<td>Synthetic phenyl-peperidine analog of codein, mixed agonist antagonist effect</td>
<td>Nausea, vomiting, sweating, ©seizures, head injury</td>
<td>IV 0.5–1 mg/kg qds</td>
</tr>
<tr>
<td>Oxycodone</td>
<td>Codein+Ibuprofen better analgesia</td>
<td></td>
<td>PO 6–10 mg/kg tds</td>
</tr>
<tr>
<td>Ibuprofen</td>
<td>Analgesic, anti-inflammatory</td>
<td>↑risk of bleeding</td>
<td>Max—40 mg/kg/day</td>
</tr>
</tbody>
</table>

### Contd...

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**Principles and Practice of Pediatric Anesthesia**

Contd...
### Appendix 2: Pediatric Drug Index

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Description</th>
<th>Caution©, side effects</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diclofenac</td>
<td>Analgesic-anti-inflammatory</td>
<td>©-renal diseases</td>
<td>2–3 mg/kg rectal suppository</td>
</tr>
<tr>
<td>Naproxen</td>
<td>Analgesic-anti-inflammatory</td>
<td>GI upset</td>
<td>5–6 mg/kg–PO</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Max–25 mg/kg/day</td>
<td></td>
</tr>
<tr>
<td>Pentazocine</td>
<td>Benzomorphone derivative, mixed agonist, antagonist</td>
<td>↑blood pressure if given as IM premedication</td>
<td>IV 0.3–0.5 mg/kg intraoperative analgesia</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Buprenorphine</td>
<td>Thebaine derivative, 5 fold higher, lipophilic effect—extremely potent</td>
<td>Respiratory depression, long lasting sedation, cannot be reversed by naloxone</td>
<td>1.5–3 μg/kg IV</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Epidural dose—3–4 μg/kg</td>
</tr>
</tbody>
</table>

#### SEDATIVES/ANESTHETICS

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Description</th>
<th>Side effects, Caution©</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chloral hydrate</td>
<td>Mild sedative</td>
<td>Arrhythmias, occasional apnoea</td>
<td>25–75 mg/kg PO/PR</td>
</tr>
<tr>
<td>Triclofos (Pedichloryl)</td>
<td>Mild sedative—Premedicant</td>
<td>©Hepatic, renal disorders</td>
<td>25–50 mg/kg 1-1.5 h before surgery PO</td>
</tr>
<tr>
<td>Midazolam (Fused)</td>
<td>Water soluble benzodiazepine gr. Oral premedication Nasal spray</td>
<td>Hypotension in neonates</td>
<td>0.05–0.1 mg/kg IV</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.5–1 mg/kg PR</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.5–0.75 mg/kg PO</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.2 mg/kg IN</td>
</tr>
<tr>
<td>Diazepam</td>
<td>Benzodiazepine</td>
<td>Neurotoxicity in neonates</td>
<td>0.1–0.2 mg/kg orally or rectally</td>
</tr>
<tr>
<td>Lorazepam</td>
<td>Sedative Anxiolysis</td>
<td></td>
<td>0.04 mg/kg</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Max 4 mg IV</td>
</tr>
<tr>
<td>Clonidine</td>
<td>α, adreno-receptor agonist, sedative, enhances neuraxial blockade</td>
<td>Bradycardia, Hypertension with loading dose. ©Digoxin ↓Cardiac output 10%</td>
<td>4 μg/kg orally</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.5–1 μg/kg IV</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1–2 μg/kg epidural</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.1, 0.2, 0.3 mg—TD patch every 7 days</td>
</tr>
<tr>
<td>Clonidine</td>
<td>α adrenergic agonist</td>
<td>Bradycardia, Hypertension with loading dose. ©Digoxin ↓Cardiac output 10%</td>
<td>Loading dose 1 μg/kg IV over 10 mins</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Infusion: 0.2–1 μg/kg/h</td>
</tr>
<tr>
<td>Ketamine</td>
<td>Sedative, analgesic NMDA receptor antagonist</td>
<td>Tachycardia Hallucinations ↑Intracranial and intraocular pressure ↑salivation</td>
<td>3–6 mg/kg PO</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>6–10 mg/kg PR</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>3 mg/kg intranasal</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1–2 mg/kg IV</td>
</tr>
<tr>
<td>Etomidate</td>
<td>Anesthetic induction</td>
<td>Apnea, hypotension, painful injection, myoclonus, yachycardia sdrenal supression</td>
<td>0.3–0.4 mg/kg IV</td>
</tr>
<tr>
<td>Pentobarbital (Nembutal)</td>
<td>Barbiturate</td>
<td>Respiratory depression</td>
<td>2 mg/kg IM, IV, PO</td>
</tr>
<tr>
<td>Propofol</td>
<td>Short-acting anesthetic</td>
<td>Apnea, hypotension, Pain on injection, muscle movement</td>
<td>1–3 mg/kg IV</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Infusion: 100–250 μg/kg/min</td>
</tr>
<tr>
<td>Methohexitol</td>
<td>Barbiturate sedative, short-acting anesthetic</td>
<td></td>
<td>1–2 mg/kg IV</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>20–30 mg/kg PR</td>
</tr>
<tr>
<td>Thiopental</td>
<td>Barbiturate, anesthetic induction</td>
<td>Hypotension</td>
<td>3–7 mg/kg IV</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>20–40 mg/kg PR</td>
</tr>
</tbody>
</table>

**Abbreviations:** NMDA, N-methyl-D-aspartate
## MUSCLE RELAXANTS

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Description</th>
<th>Side effects, Caution</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cisatracurium</td>
<td>Nondepolarizing Neuromuscular Blocker</td>
<td>Histamine release</td>
<td>0.1–0.2 mg/kg/dose IV for intubation</td>
</tr>
<tr>
<td>Atracurium</td>
<td>Histamine release</td>
<td>0.1–0.2 mg/kg/dose IV for intubation</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.5–0.6 mg/kg/dose IV for intubation</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>5–10 μg/kg/min infusion</td>
<td></td>
</tr>
<tr>
<td>Pancuronium</td>
<td>Long-acting</td>
<td>Tachycardia</td>
<td>0.1 mg/kg/dose IV for intubation</td>
</tr>
<tr>
<td>Mivacurium</td>
<td>NMB</td>
<td>Histamine release</td>
<td>0.2–0.3 mg/kg for intubation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>10–15 μg/kg/min infusion</td>
<td></td>
</tr>
<tr>
<td>Rocuronium</td>
<td>Long-acting</td>
<td></td>
<td>0.4–1.0 mg/kg IV for intubation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.6 mg/kg/hr for infusion</td>
<td></td>
</tr>
<tr>
<td>Vecuronium</td>
<td>Long-acting</td>
<td></td>
<td>0.1 mg/kg IV</td>
</tr>
<tr>
<td></td>
<td></td>
<td>60 μg/kg/hr for infusion</td>
<td></td>
</tr>
<tr>
<td>Succinylcholine</td>
<td>Depolarizing Short-acting Useul in Laryngospasm</td>
<td>Hyperkalemia, Rhabdomyolysis, Histamine release, Cardiac arrest ©Burn, tissue trauma</td>
<td></td>
</tr>
<tr>
<td>Tubocurarine</td>
<td>Long-acting NMB</td>
<td>Histamine release</td>
<td>0.3–0.6 mg/kg</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hypotension</td>
<td></td>
</tr>
</tbody>
</table>

**Abbreviations:** NMB, Neuromuscular blocking drug

## REVERSAL AGENTS

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Description</th>
<th>Side effects, Caution</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Edrophonium</td>
<td></td>
<td>Bradycardia</td>
<td>0.5–1 mg/kg IV</td>
</tr>
<tr>
<td>Neostigmine</td>
<td></td>
<td>Bradycardia, Excessive secretions</td>
<td>0.04–0.07 mg/kg IV</td>
</tr>
<tr>
<td>Physostigmine</td>
<td></td>
<td>Bradycardia</td>
<td>0.01 mg/kg IV slowly</td>
</tr>
<tr>
<td>Sugammadex</td>
<td>γ Cyclodextrin selective binding agent—Reversal only for rocuronium</td>
<td>Renal impairment</td>
<td>2–4 mg/kg IV Max 16 mg</td>
</tr>
</tbody>
</table>

## MISCELLANEOUS

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Description</th>
<th>Side effects/caution</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aprotinin</td>
<td>To decrease bleeding in cardiac surgery</td>
<td>Anaphylaxis</td>
<td>IV loading dose—Test dose 10,000 unit 30,000 units/kg over 30 mins</td>
</tr>
<tr>
<td>Alprostadil</td>
<td>For patency of ductus arteriosus</td>
<td>Fever, apnea</td>
<td>0.05–0.1 μg/kg/min</td>
</tr>
<tr>
<td>Aminocaproic Acid-Amicar</td>
<td>To reduce bleeding in cardiac and orthopedic surgery</td>
<td>Hypotension, arrhythmias</td>
<td>Cardiac 200 mg/kg IV Ortho 100–150 mg/kg IV Later 10–15 mg/kg/h</td>
</tr>
<tr>
<td>DDAVP</td>
<td>For platelet dysfunction</td>
<td>Hypotension</td>
<td>0.3 μg/kg over 15 mins IV</td>
</tr>
<tr>
<td>Dantrolene</td>
<td>Malignant hyperthermia</td>
<td>Drowsiness, Muscle weakness</td>
<td>2.5 mg/kg IV Max 10 mg/kg IV</td>
</tr>
<tr>
<td></td>
<td>Slow to solubilize</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Contd...
<table>
<thead>
<tr>
<th>Drugs</th>
<th>Description</th>
<th>Side effects/caution</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Furosemide (Lasix)</td>
<td>Diuretic—Pulmonary edema Hypervolemia</td>
<td>Hyponatremia</td>
<td>0.5–1 mg/kg IV, PO</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hypokalemia</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Volume depletion</td>
<td></td>
</tr>
<tr>
<td>Heparin</td>
<td>CP Bypass</td>
<td></td>
<td>50–100 U/kg IV</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Then 10–20 U/kg/h</td>
<td></td>
</tr>
<tr>
<td>Insulin</td>
<td>Hyperglycemia, diabetic ketoacidosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hyperkalemia—Insulin+Dextrose</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hyponatremia</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hypokalemia</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Volume depletion</td>
<td></td>
</tr>
<tr>
<td>Mannitol</td>
<td>↑ intracranial pressure, diuresis</td>
<td></td>
<td>0.25 to 1 g/kg over 20 mins</td>
</tr>
<tr>
<td>Methyleneblue</td>
<td>Antidote for drug-induced methemoglobinemia</td>
<td>Stains skin</td>
<td>1–2 mg/kg</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Repeated after 1 h</td>
</tr>
<tr>
<td>Prostaglandin E₁</td>
<td>To keep PDA open</td>
<td>Fever, apnea</td>
<td>0.05–0.1 μg/kg/min</td>
</tr>
<tr>
<td>EMLA cream</td>
<td>Topical anesthesia</td>
<td>Methemoglobinemia</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Blanching of skin</td>
<td></td>
</tr>
<tr>
<td>Protamine</td>
<td>Heparin neutralization</td>
<td></td>
<td>1 mg for every 100 U heparin to be neutralized</td>
</tr>
<tr>
<td>Scopolamine</td>
<td>Amnesic, antisialagog</td>
<td>Tachycardia</td>
<td>6–10 μg/kg IV/IM</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Confusion</td>
<td></td>
</tr>
</tbody>
</table>

**STEROIDS**

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Description</th>
<th>Side effects/ Caution</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dexamethasone (Decadron)</td>
<td>Useful in croup, airway edema Cerebral edema</td>
<td>Headache, hypertension, Hyperglycemia</td>
<td>0.5–1 mg/kg IV, Max 10–20 mg</td>
</tr>
<tr>
<td>Hydrocortisone (Ecocrin)</td>
<td>For asthma, bronchospasm adrenal supression</td>
<td>Hypertension</td>
<td>Anti-inflammatory 0.5–mg/kg 6 hrly IV Asthma 4–8 mg/kg then 2–4 mg/kg qds</td>
</tr>
<tr>
<td>Methylprednisolone</td>
<td>Anti-inflammatory, For status asthmatic</td>
<td>Hypertension</td>
<td>0.04–0.2 mg/kg IV qds Asthma 2 mg/kg load then 0.5–1 mg/kg qds</td>
</tr>
<tr>
<td>Solumedrol</td>
<td></td>
<td>Psychosis, glucose intolerance</td>
<td></td>
</tr>
<tr>
<td>Tolazoline</td>
<td>Pulmonary hypertension</td>
<td>Hypotension</td>
<td>1–2 mg/kg IV slowly Infusion 2 mg/kg/hr</td>
</tr>
</tbody>
</table>

**Abbreviations:** PDA, Patent ductus arteriosus; DDAVP, Desmopressin; CP, cardiopulmonary; EMLA, Eutectic Mixture of Local Anesthetics; ET, endotracheal; IC, intracardiac; IM, intramuscular; IO, intraoesous; IV, intravenous; PO, per OS (orally); PR, per rectum; SC, subcutaneous; TD, transdermal; μg, microgram; bds-twice a day; tds, three times a day; qds, four times a day, yrs, years, hrly, hourly, mths, months
## Endotracheal tube size [Internal diameter (ID) in mm]

<table>
<thead>
<tr>
<th>Formula</th>
<th>Calculation</th>
</tr>
</thead>
<tbody>
<tr>
<td>(16 + Age in years)/4</td>
<td>Internal diameter of endotracheal tube</td>
</tr>
<tr>
<td>Age in years/4 + 4</td>
<td></td>
</tr>
<tr>
<td>Age in years/4 + 3 (Cuffed endotracheal tube)</td>
<td></td>
</tr>
</tbody>
</table>
| Penlington's formula | Below 6.5 years = Age (years)/3 + 3.5  
Above 6.5 years = Age (years)/4 + 4.5 |

## Endotracheal tube insertion depth (length up to mid-trachea) in cm

<table>
<thead>
<tr>
<th>Category</th>
<th>Calculation</th>
</tr>
</thead>
</table>
| Over 1 year | Distance from lip = ETT size (ID) × 3  
Orotracheal = age/2 + 12  
Nasotracheal = age/2 + 15 |
| Under 1 year | Orotracheal = age/2 + 8  
Nasotracheal = age/2 + 9 |
| Under 4 years | Nasotracheal = weight (kg)/2 + 10.5 |
| Yates formula (>3 kg weight) | Nasotracheal = (tracheal tube size × 3) + 2 |

## Endotracheal Tube ID (mm) in neonates

<table>
<thead>
<tr>
<th>Gestational age in weeks divided by 10</th>
<th>ID (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1 kg</td>
<td>2.5 mm</td>
</tr>
<tr>
<td>1–2 kg</td>
<td>3.0 mm</td>
</tr>
<tr>
<td>2–3 kg</td>
<td>3–3.5 mm</td>
</tr>
<tr>
<td>&gt;3 kg</td>
<td>3.5–4.0 mm</td>
</tr>
</tbody>
</table>

Approximate ETT insertion depth from middle of upper lip = (weight in kg + 6 cm)

## NORMAL RESPIRATORY VALUES

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Newborn</th>
<th>Adult</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory frequency breaths/min</td>
<td>30–50</td>
<td>12–16</td>
</tr>
<tr>
<td>Tidal volume (Vt) mL/kg</td>
<td>6–8</td>
<td>7</td>
</tr>
<tr>
<td>Dead space (Vd) mL/kg</td>
<td>2–2.5</td>
<td>2.2</td>
</tr>
<tr>
<td>Vital capacity (Vc) mL/kg</td>
<td>35–40</td>
<td>50–60</td>
</tr>
<tr>
<td>Functional residual capacity (FRC) mL/kg</td>
<td>27–30</td>
<td>30</td>
</tr>
</tbody>
</table>

Contd...
**Appendix 3: Quick Reference Tables and Formulae**

### Lung compliance mL/cm H$_2$O
- **Newborn:** 5–6
- **Adult:** 200

### Alveolar ventilation (VA) mL/kg/min
- **Newborn:** 100–150
- **Adult:** 60

### Oxygen consumption (VO$_2$) mL/kg/min
- **Newborn:** 6–8
- **Adult:** 3

### Dead space/Tidal volume ratio (Vd/Vt)
- **Newborn:** 0.3
- **Adult:** 0.3

### Airway resistance cm H$_2$O/L/sec
- **Newborn:** 25–30
- **Adult:** 1.6

### Relation of Age, Weight and Body Surface Area

<table>
<thead>
<tr>
<th>Age</th>
<th>Weight</th>
<th>Surface area</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birth</td>
<td>3.0 Kg</td>
<td>0.2 m$^2$</td>
</tr>
<tr>
<td>6 months</td>
<td>7.5 Kg</td>
<td>0.38 m$^2$</td>
</tr>
<tr>
<td>1 year</td>
<td>10 Kg</td>
<td>0.5 m$^2$</td>
</tr>
<tr>
<td>3 years</td>
<td>13–15 Kg</td>
<td>0.62 m$^2$</td>
</tr>
<tr>
<td>5 years</td>
<td>18.4 Kg</td>
<td>0.75 m$^2$</td>
</tr>
<tr>
<td>8 years</td>
<td>30.0 Kg</td>
<td>1.0 m$^2$</td>
</tr>
<tr>
<td>10 years</td>
<td>32.6 Kg</td>
<td>1.12 m$^2$</td>
</tr>
<tr>
<td>14 years</td>
<td>49.0 Kg</td>
<td>1.50 m$^2$</td>
</tr>
<tr>
<td>Adult</td>
<td>60–80 Kg</td>
<td>1.7–1.9 m$^2$</td>
</tr>
</tbody>
</table>

### Blood Pressure During 1st Year of Life

<table>
<thead>
<tr>
<th>Age (Months)</th>
<th>Systolic (mm Hg$^*$)</th>
<th>Diastolic (mm Hg$^*$)</th>
<th>Mean (mm Hg$^*$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>84 (65–103)</td>
<td>52 (35–69)</td>
<td>61 (45–80)</td>
</tr>
<tr>
<td>3</td>
<td>89 (70–108)</td>
<td>52 (36–68)</td>
<td>64 (47–81)</td>
</tr>
<tr>
<td>6</td>
<td>92 (72–112)</td>
<td>53 (36–70)</td>
<td>66 (48–84)</td>
</tr>
<tr>
<td>9</td>
<td>92 (72–112)</td>
<td>54 (37–71)</td>
<td>67 (48–84)</td>
</tr>
<tr>
<td>12</td>
<td>92 (72–112)</td>
<td>55 (38–72)</td>
<td>67 (48–84)</td>
</tr>
</tbody>
</table>

Mean blood pressure is estimated as diastolic + (systolic/3) OR [Systolic + (2 x diastolic)/3]

Hypotension is a systolic blood pressure less than the 5th percentile of normal for age, namely:

- <60 mm Hg in term neonates (0 to 28 days)
- <70 mm Hg in infants (1 month to 12 months)
- <70 mm Hg + (2 x age in years) in children 1 to 10 years
- <90 mm Hg in children ≥10 years of age

### Total Body Water and its Distribution in Percentage of Weight—According to Age

<table>
<thead>
<tr>
<th></th>
<th>Preterm neonate</th>
<th>Term neonate</th>
<th>1–3 years</th>
<th>Adults</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total body water</td>
<td>85%</td>
<td>80%</td>
<td>65%</td>
<td>65%</td>
</tr>
<tr>
<td>Extracellular fluid</td>
<td>55%</td>
<td>45%</td>
<td>25%</td>
<td>25%</td>
</tr>
<tr>
<td>Intracellular fluid</td>
<td>30%</td>
<td>35%</td>
<td>40%</td>
<td>40%</td>
</tr>
</tbody>
</table>

### Endotracheal Epinephrine Doses During Cardiac Arrest

<table>
<thead>
<tr>
<th>Age</th>
<th>Volume</th>
</tr>
</thead>
<tbody>
<tr>
<td>Newborn</td>
<td>0.7 mL</td>
</tr>
<tr>
<td>Infant</td>
<td>1–2 mL</td>
</tr>
<tr>
<td>Small child</td>
<td>2–5 mL</td>
</tr>
<tr>
<td>Large child</td>
<td>5–10 mL</td>
</tr>
</tbody>
</table>

Use 1:1000 epinephrine solution 0.1 mL/Kg mixed with equal volume of normal saline.
### AGE RELATED K⁺ (POTASSIUM) LEVELS IN PLASMA

<table>
<thead>
<tr>
<th>Age</th>
<th>K⁺</th>
</tr>
</thead>
<tbody>
<tr>
<td>30–32 weeks</td>
<td>6.5 ± 0.5 mmol/L</td>
</tr>
<tr>
<td>33–35 weeks</td>
<td>5.6 ± 0.2 mmol/L</td>
</tr>
<tr>
<td>36–38 weeks</td>
<td>5.3 ± 0.3 mmol/L</td>
</tr>
<tr>
<td>39–41 weeks</td>
<td>5.1 ± 0.2 mmol/L</td>
</tr>
<tr>
<td>1–12 months</td>
<td>5 ± 0.5 mmol/L</td>
</tr>
<tr>
<td>2–20 years</td>
<td>4.3 ± 0.4 mmol/L</td>
</tr>
<tr>
<td>Adults</td>
<td>3.5–5 mmol/L</td>
</tr>
</tbody>
</table>

### AGE AND WEIGHT-BASED SIZING CHART

<table>
<thead>
<tr>
<th>Age or Weight</th>
<th>Plain tube</th>
<th>Cuffed tube</th>
<th>Laryngoscope blade</th>
<th>Double lumen tube</th>
<th>Central venous line</th>
<th>Arterial line</th>
<th>LMA</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 kg</td>
<td>2.5</td>
<td>Miller 00</td>
<td>3 Fr</td>
<td>24 G</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 kg</td>
<td>3</td>
<td>Miller 00</td>
<td>3 Fr</td>
<td>24 G</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 kg</td>
<td>3–3.5</td>
<td>Miller 0</td>
<td>3 Fr</td>
<td>24 G</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Full term</td>
<td>3–3.5</td>
<td>3</td>
<td>Miller 1</td>
<td>3 Fr</td>
<td>24 G</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>5 kg</td>
<td>3.5–4</td>
<td>3.5</td>
<td>Miller 1</td>
<td>4 Fr</td>
<td>24 G</td>
<td>1.5</td>
<td></td>
</tr>
<tr>
<td>1 year</td>
<td>4–4.5</td>
<td>4</td>
<td>Miller 1</td>
<td>5 Fr</td>
<td>24 G</td>
<td>1.5</td>
<td></td>
</tr>
<tr>
<td>2 years</td>
<td>4.5–5</td>
<td>4–4.5</td>
<td>Miller/Mac 2</td>
<td>5 Fr</td>
<td>24 G</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>3 years</td>
<td>4.5–5</td>
<td>4.5</td>
<td>Miller/Mac 2</td>
<td>5 Fr</td>
<td>22 G</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>4 years</td>
<td>5–5.5</td>
<td>4.5</td>
<td>Miller/Mac 2</td>
<td>5 Fr</td>
<td>22 G</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>5 years</td>
<td>5.5</td>
<td>5</td>
<td>Miller/Mac 2</td>
<td>5 Fr</td>
<td>22 G</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>6 years</td>
<td>5</td>
<td>Miller/Mac 2</td>
<td>5 Fr</td>
<td>22 G</td>
<td>2.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7 years</td>
<td>5–5.5</td>
<td>Miller/Mac 2</td>
<td>5 Fr</td>
<td>22 G</td>
<td>2.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8 years</td>
<td>5.5</td>
<td>Miller/Mac2</td>
<td>26 Fr</td>
<td>5 Fr</td>
<td>22 G</td>
<td>2.5</td>
<td></td>
</tr>
<tr>
<td>9 years</td>
<td>5.5–6</td>
<td>Miller/Mac 2</td>
<td>26 Fr</td>
<td>7 Fr</td>
<td>22 G</td>
<td>2.5</td>
<td></td>
</tr>
<tr>
<td>10 years</td>
<td>6</td>
<td>Miller/Mac 3</td>
<td>28 Fr</td>
<td>7 Fr</td>
<td>20 G</td>
<td>2.5</td>
<td></td>
</tr>
<tr>
<td>11 years</td>
<td>6</td>
<td>Miller/Mac 3</td>
<td>28 Fr</td>
<td>7 Fr</td>
<td>20 G</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>12 years</td>
<td>6</td>
<td>Miller/Mac 3</td>
<td>28 Fr</td>
<td>7 Fr</td>
<td>20 G</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>13 years</td>
<td>6–6.5</td>
<td>Miller/Mac 3</td>
<td>32 Fr</td>
<td>9 Fr</td>
<td>20 G</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>14 years</td>
<td>6.5–7</td>
<td>Miller/Mac 3</td>
<td>32 Fr</td>
<td>9 Fr</td>
<td>20 G</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>15 years</td>
<td>7</td>
<td>Miller/Mac 3</td>
<td>35 Fr</td>
<td>9 Fr</td>
<td>20 G</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>16 years</td>
<td>7</td>
<td>Miller/Mac 3</td>
<td>35 Fr</td>
<td>9 Fr</td>
<td>20 G</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>17 years</td>
<td>7</td>
<td>Miller/Mac 3</td>
<td>35 Fr</td>
<td>9 Fr</td>
<td>20 G</td>
<td>4</td>
<td></td>
</tr>
</tbody>
</table>

### TEMPERATURE REGULATION IN NEWBORNS AND INFANTS

<table>
<thead>
<tr>
<th>Age</th>
<th>Neutral temperature</th>
<th>Critical temperature</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preterm infant</td>
<td>34°C</td>
<td>28°C</td>
</tr>
<tr>
<td>Term infant</td>
<td>32°C</td>
<td>23°C</td>
</tr>
<tr>
<td>Adult</td>
<td>28°C</td>
<td>1°C</td>
</tr>
</tbody>
</table>

**Neutral temperature**: The ambient temperature that results in minimal oxygen consumption.

**Critical temperature**: The temperature below which the unanesthetized patient cannot maintain normal core temperature.
FLUID REQUIREMENTS IN PREMATURE BABIES

<table>
<thead>
<tr>
<th></th>
<th>1250 grams</th>
<th>1500 grams</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st and 2nd day</td>
<td>90 mL/kg/day</td>
<td>80 mL/kg/day</td>
</tr>
<tr>
<td>3rd to 15 days</td>
<td>120 mL/kg/day</td>
<td>110 mL/kg/day</td>
</tr>
<tr>
<td>15 days onward</td>
<td>130 mL/kg/day</td>
<td>130 mL/kg/day</td>
</tr>
</tbody>
</table>

5% Dextrose
- 4 mL/kg/h = 0.2 g/kg/h
- 6 mL/kg/h = 0.3 g/kg/h
- 8 mL/kg/h = 0.4 g/kg/h
- 10 mL/kg/h = 0.5 g/kg/h

Note: Full-term neonates require glucose 3–5 mg/kg/min. Preterm neonates require sick infants require 7-10 mg/kg/min. 5-6 mg/kg/min.

PAIN ASSESSMENT SCALES

1. Children’s Hospital of East Ontario Pain Scale (CHEOPS). It is an observational pain scale for pain measurement in children 1–7 years of age. CHEOPS has a minimum possible score of 4 points (no pain) to a maximum of 13 points (the worst pain).

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Score 0</th>
<th>Score 1</th>
<th>Score 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cry</td>
<td>No Cry</td>
<td>Crying, moaning</td>
<td>Scream</td>
</tr>
<tr>
<td>Facial</td>
<td>Smiling</td>
<td>Composed</td>
<td>Grimace</td>
</tr>
<tr>
<td>Verbal</td>
<td>Positive</td>
<td>None or other complaints</td>
<td>Pain complaint</td>
</tr>
<tr>
<td>Torso</td>
<td>Neutral</td>
<td>Shifting, tense, upright</td>
<td>Restrained</td>
</tr>
<tr>
<td>Legs</td>
<td>Neutral</td>
<td>Kicks, squirm, drawn up</td>
<td>Restrained</td>
</tr>
</tbody>
</table>

2. FLACC Behavioral Pain Assessment Scale, used to assess pain for children between 2 months and 7 years or individuals who are unable to communicate their pain. Each category is scored on the 0–2 scale, which results in a total score of 0–10.

0= Relaxed and comfortable; 1–3= Mild discomfort; 4–6= Moderate pain; 7–10= Severe discomfort or pain or both

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Score 0</th>
<th>Score 1</th>
<th>Score 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Face</td>
<td>No particular expression or smile</td>
<td>Occasional grimace or frown, withdrawn, disinterested</td>
<td>Frequent to constant quivering chin, clenched jaw</td>
</tr>
<tr>
<td>Legs</td>
<td>Normal position or relaxed</td>
<td>Uneasy, restless, tense</td>
<td>Kicking, or legs drawn up</td>
</tr>
<tr>
<td>Activity</td>
<td>Lying quietly, normal position, moves easily</td>
<td>Squirming, shifting back and forth, tense</td>
<td>Arched, rigid or jerking</td>
</tr>
<tr>
<td>Cry</td>
<td>No cry (awake or asleep)</td>
<td>Moans or whimpers; occasional complaint</td>
<td>Crying steadily, screams or sobs, frequent complaints</td>
</tr>
<tr>
<td>Consolability</td>
<td>Content, relaxed</td>
<td>Reassured by occasional touching, hugging or being talked to, distractible</td>
<td>Difficult to console or comfort</td>
</tr>
</tbody>
</table>

3. CRIES: Neonatal pain assessment score. The tool is a 10 point scale, an acronym of 5 physiological and behavioral variables associated with neonatal pain.

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Score 0</th>
<th>Score 1</th>
<th>Score 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crying</td>
<td>No</td>
<td>High pitched</td>
<td>Inconsolable</td>
</tr>
<tr>
<td>Requires oxygen</td>
<td>No</td>
<td>&lt;30%</td>
<td>&gt;30%</td>
</tr>
<tr>
<td>Increase in vital signs</td>
<td>Heart rate and blood pressure less than or equal to preoperative state</td>
<td>Heart rate and blood pressure increase less than 20% of preoperative state</td>
<td>Heart rate and blood pressure increase greater than 20% of preoperative state</td>
</tr>
<tr>
<td>Expression</td>
<td>None</td>
<td>Grimace</td>
<td>Grimace/grunt</td>
</tr>
<tr>
<td>Sleepless</td>
<td>No</td>
<td>Wakes at frequent intervals</td>
<td>Constantly awake</td>
</tr>
</tbody>
</table>

Source: Krechel S, Bildner J, RNC, CNS, 1995, from University of Missouri-Columbia, with permission.
**CONGENITAL MALFORMATIONS DUE TO DEVELOPMENTAL PATHOLOGY AND RELATIVE TIMING**

<table>
<thead>
<tr>
<th>Malformations</th>
<th>Defect</th>
<th>Fetal age</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Head and Neck</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Branchial sinus</td>
<td>Resolution of branchial cleft</td>
<td>8 Weeks</td>
</tr>
<tr>
<td>Cleft lip</td>
<td>Closure of lip</td>
<td>36 Days</td>
</tr>
<tr>
<td>Cleft palate</td>
<td>Fusion of maxillary palatal shelves</td>
<td>10 Weeks</td>
</tr>
<tr>
<td><strong>Cardiovascular System</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Transposition of great vessels</td>
<td>Development of bulbus cordis septum</td>
<td>34 Days</td>
</tr>
<tr>
<td>Ventricular septal defect</td>
<td>Closure of septum</td>
<td>6 Weeks</td>
</tr>
<tr>
<td>Patent ductus arteriosus</td>
<td>Closure of ductus</td>
<td>9-10 months</td>
</tr>
<tr>
<td><strong>Gastrointestinal System</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tracheo-esophageal fistula and esophageal atresia</td>
<td>Septation of foregut into trachea and foregut</td>
<td>30 Days</td>
</tr>
<tr>
<td>Diaphragmatic hernia</td>
<td>Obliteration of vitelline duct and closure of pleuroperitoneal canal</td>
<td>10 Weeks, 6 Weeks</td>
</tr>
<tr>
<td>Duodenal atresia</td>
<td>Recanalization of duodenum</td>
<td>7-8 Weeks</td>
</tr>
<tr>
<td>Malrotation of gut</td>
<td>Rotation of intestinal loop</td>
<td>10 Weeks</td>
</tr>
<tr>
<td>Rectal atresia with fistula</td>
<td>Septation of cloaca into rectum and urogenital sinus</td>
<td>6 Weeks</td>
</tr>
<tr>
<td>Meckel's diverticulum</td>
<td>Failure of proximal segment to close, Return of midgut from yolk sac to abdomen</td>
<td>10 Weeks</td>
</tr>
<tr>
<td>Omphalocele</td>
<td>Non-return of bowel from extraembryonic celom</td>
<td>10 Weeks</td>
</tr>
<tr>
<td>Gastrochisis</td>
<td>Occlusion of omphalomesenteric artery and failure of development of lateral folds</td>
<td>Later in fetal life</td>
</tr>
<tr>
<td><strong>Genitourinary System</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bladder extrophy</td>
<td>Migration of infraumbilical mesenchyme</td>
<td>30 Days</td>
</tr>
<tr>
<td>Bicornuate uterus</td>
<td>Fusion of lower part of Mullerian duct</td>
<td>10 Weeks</td>
</tr>
<tr>
<td>Hypospadias</td>
<td>Fusion of urethral folds</td>
<td>12 Weeks</td>
</tr>
<tr>
<td>Undescended testis</td>
<td>Descent of testicle</td>
<td>7-9 Months</td>
</tr>
<tr>
<td><strong>Central Nervous System</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anencephaly</td>
<td>Closure of anterior neural tube</td>
<td>26 Days</td>
</tr>
<tr>
<td>Meningomyelocele</td>
<td>Closure of part of posterior neural tube</td>
<td>28 Days</td>
</tr>
<tr>
<td><strong>Bony Structure</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Radial aplasia</td>
<td>Genesis of radial bone</td>
<td>38 Days</td>
</tr>
<tr>
<td>Syndactyly</td>
<td>Separation of digital rays</td>
<td>6 Weeks</td>
</tr>
</tbody>
</table>
Photo Gallery

Figs 1A and B: Anterior meningocele

Fig. 2: Dorsal meningocele

Fig. 3: Large occipital encephalocele
Fig. 4: Encephalocele

Fig. 5: Cystic hygroma

Fig. 6: Cystic hygroma—face

Fig. 7: Cystic hygroma—chest and abdomen

Fig. 8: Hemangioma—chest wall

Fig. 9: Hemangioma—neck
Fig. 10: Hemangioma

Fig. 11: Lymphangioma—chest and arm

Fig. 12: Lymphangioma—forehead

Fig. 13: Lymphangioma—neck and chest

Figs 14A to C: Lymphangioma—neck
Fig. 15: Lymphangioma—tongue

Fig. 16: Exomphalos with associated abnormality

Figs 17A and B: Exomphalos

Fig. 18: Hydrocephalus

Fig. 19: Hydrocephalus—Sunsetting sign
Figs 20A and B: Sacrococcygeal teratoma

Fig. 21: Teratoma—chest and Exomphalos

Fig. 22: Teratoma—stomach in newborn

Fig. 23: Rhabdomyosarcoma—shoulder

Fig. 24: Thigh—sarcoma
Fig. 25: Osteogenesis imperfecta

Fig. 26: Limb anomalies

Fig. 27: Pectus deformity

Fig. 28: Conjoint twins

Fig. 29: Trichobezoar specimen

Fig. 30: Trichobezoar X-ray
Fig. 31: Umbilical hernia

Fig. 32: Patent Urachus

Fig. 33: Pyloric stenosis wave

Fig. 34: Necrotizing enterocolitis with air in intestinal wall

Fig. 35: Ovarian cyst with ascitis in newborn

Fig. 36: Bilateral hydrenephrosis
Fig. 37: Ectopia cordis
Fig. 38: Ranula
Fig. 39: Treacher collins—operated

Fig. 40: Hemangioma
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Fig. 42: Epidermolysis bullosa
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