

Dr. Azam's....

Notes in Anesthesiology

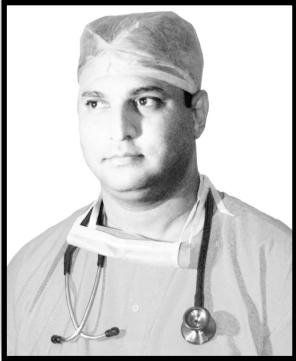
Updated up to December 2013, 3rd Edition

Postgraduates appearing
for MD, DNB & DA Exams

Pediatrics

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Dedication

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

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

Finally, I would like to thank my teachers (**Dr. Manjunath Jajoor & team**) & **Dr T. A. Patil**. The dream begins with a teacher who believes in you, who tugs and pushes and leads you to the next plateau, sometimes poking you with a sharp stick called "truth."

A NOTE TO THE READER

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

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Dr. Azam

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Classification by Age:

- Neonate: 1-28 days of age
 - Infant: 1-12 months of age
 - Toddlers – 2-3 yrs
 - Children – 1-12 yrs
- Pediatric patients deserve special considerations with respect to anatomic, physiologic and pharmacologic differences from adults.
 - Premature infants (gestational age less than 37 wks) and low birth weight infants (birth weight less than 2,500g) can have abnormal organogenesis / abnormal organ function / smaller organs / reduced muscle and fat mass.

1) Body size:

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

2) Airway anatomy:

- Large head, short neck, narrow shoulders → neutral / slightly flexed position of head for intubation.
- Nasal passages narrow → smaller size of ETT.
- Large tongue in relation to oropharynx → difficulty in laryngoscopy / visualization of larynx / ↑ chance of airway obstruction.
- Epiglottis – short and stubby, hard and narrow folded into an 'Ω' omega or 'V' shaped → angled at 45° over laryngeal inlet
- difficult to lift with the tip of a laryngoscope blade
- Vocal cords – angled forwards and downwards blindly passed ETT may lodge in the ant. Commissure. → flexion of head.

- Larynx high – C₄ vertebrae – glottis higher and anterior than adults → straight blades more useful than curved blades.
- Narrowest portion – cricoid cartilage (adults-glottis)
- Subglottic region easily damaged by large ETT
- Any edema at this region ↓ airway diameter by as much as 60-70% of neonates.
- Un-cuffed ETT used in children younger than 10 yrs.
- Trachea 4cm in length and 6-8 mm diameter → term infants
- Bronchial intubation more likely
 1. Chest relatively small in relation to abdomen.
 2. Poorly developed body support (bone and muscle) with disproportion.
 - Difficulty in positioning
 - Sitting position – craniotomies – head secured safely as neck is weak for heavy head.
 - Prone position – shoulders need adequate support – rolls underneath both shoulder.

3) Respiratory physiology:

1. Ribs are more horizontal → less A-P and lateral chest expansion.
2. Sternum and thoracic cage-soft and compliant → Negative -ve intrathoracic pressure poorly maintained.
3. Intercostal muscles weak.
 - Diaphragm is high and moves like piston → abdominal distention – diaphragm splinting.
 - Diaphragm and intercostal muscle are deficient in type I – type II fibers are predominant.
 - Type II fibers – rapidly stimulated, easily fatiguable, glycolysis metabolism → any ↑ work of breathing (WOB) – fatigue – apnea and CO₂ retention – respiratory failure.
 - Alveolar malnutrition incomplete until late childhood

- And small alveoli are associated with low lung compliance
- **CL = Change in volume/Change in broncho pulmonary pressure**
- Small diameter of airways - $\uparrow$ resistance to airflow ($R \propto 1/r^4$) spontaneous ventilation not advised as $\uparrow$ work of breathing – fatigue.
- Tidal volume 6-8ml/kg and dead space to T.V – 0.3.
- Alveolar ventilation – 150ml/kg/min (twice of adults)
- RR - $\uparrow$ 35/min.
- O₂ consumption 5-6ml/kg/min – higher than adults
- FRC 27-30 ml/kg – smaller than adults

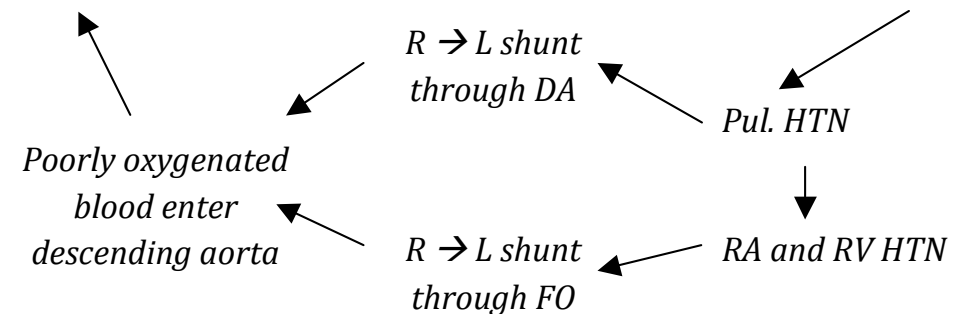
4) Cardiovascular system:

- Birth and the initiation of spontaneous ventilation initiate circulatory changes, permitting neonates to survive in an extra uterine environment.
- Fetal circulation – high PVR, low SVR (placenta) and R - L shunting through foramen ovale and ductus arteriosus.
- At birth – placental removal from circulation – SVR $\uparrow$ rapidly exposure of ductus arteriosus to $\uparrow$ O₂ conc. - ductus arteriosus closure.
- SVR - $\downarrow$ PVR $\rightarrow$ $\uparrow$ left side pressure $\rightarrow$ foramen ovale closure.
- Closure of ductus arteriosus: Functional closure 10-15 hrs after birth, Anatomic closure 2-3 wks after (ligamentous Arteriosus)
- Closure of foramen ovale: Functional closure – at birth, Anatomic closure – 6 wks
- Closure of ductus venosus: Functional closure – 3 to 7 days after birth, Anatomic closure – 2-3 months of age (ligamentous Venosum)
- Transitional circulation – until true mechanical closure of ductus arteriosus at 2-3 wks of age adult circulation is not establishment.

Persistent fetal circulation / persistent Pulmonary HTN of newborn

1. Diaphragmatic hernia
2. Meconium aspiration
3. Polycythemia.

Severe hypoxemia $\rightarrow$ intense pulmonary vasoconstriction $\rightarrow$ $\uparrow$ PVR



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

5) Hematology:

- Blood volume $\rightarrow$ Preterm 90-105 ml/kg
- Full term neonate 80-90ml/kg
- Hb - 19g% at 8-12 wks $\rightarrow$ 10-11g% at 2-3 months – physiologic anemia.
- Fetal Hb (HbF)- at birth HbF 80% HbA 20%
- [HbF $\rightarrow$ P₅₀ 18-20mmHg, $\downarrow$ 2,3 -DPG, $\downarrow$ affinity to 2,3-DPG - $\uparrow$ O₂ affinity

Hematological Importance Continuation:

- ODC – left → better O₂ transport but poor O₂ delivery.
- By 5 months HbF ↓ to 5%, 2,3-DPG ↑ by about – 37%
- HbA ↑ → ODC – right
- Leukocyte function impaired – prone for infection.
- Platelet count (NL) – defective function
- Prothrombin time prolonged (liver immature and minimal placental transfer of vitamin K dep. factors)
- Corrected with vitamin K treatment IM preoperatively and at birth.

6) Renal:

- Nearly complete maturation of glomerular filtration and tubular function – 20 wks (5 months) of age.
- Complete maturation of renal function – 2 yrs of age.
- Diminished renal function in neonate – low perfusion pressure and immature glomerular and tubular function.
- Newborn – GFR 21 ± 4 ml/min/BSA
 - 6mon – 1 yr – 77 ± 14 ml/min/BSA
 - Adults – 125 ± 15ml/min/BSA
 - Half life of drugs excreted by glomerular filtration prolonged (pancuronium, digoxin, antibiotics).
 - Lower renal threshold for glucose (glycosuria – osmotic diuresis)
 - avoid hyperglycemia
- Neonates obligate Na⁺ loser and cannot conc. urine → exogenous Na⁺ and H₂O during perioperative period.

7) Hepatic:

- At term → functional maturity incomplete
- Neonates – conjugation reactions impaired - ↓ degradation reactions
- long t_{1/2} of drugs
- Neonates – plasma albumin and other proteins lower →
- Greater levels of free drug (thio ↓ dose), neonatal coagulopathy
- Minimal glycogen stores – prone for hypoglycemia (avoid prolonged fasting)
- Unable to handle large protein loads – prone for academia.

8) Body composition:

- Total body water content and ECF volume - ↑ proportionately in neonates ↓ with age (premature infant > term infant > 2yr old)
- ECF vol. – 40% of body weight in neonates (20% of adults)
- 18-24 months become same as adults.
- Fat and muscle content ↑ with age
 - Water sol. drugs – large Vd → higher initial doses
 - Drugs that redistribute to muscle – longer clinical effect e.g. Fentanyl
 - Drugs that redistribute to Fat for termination – longer clinical effect Thiopentone

9) Nervous system:

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

Note: Vitamin K = Causes Hemolysis

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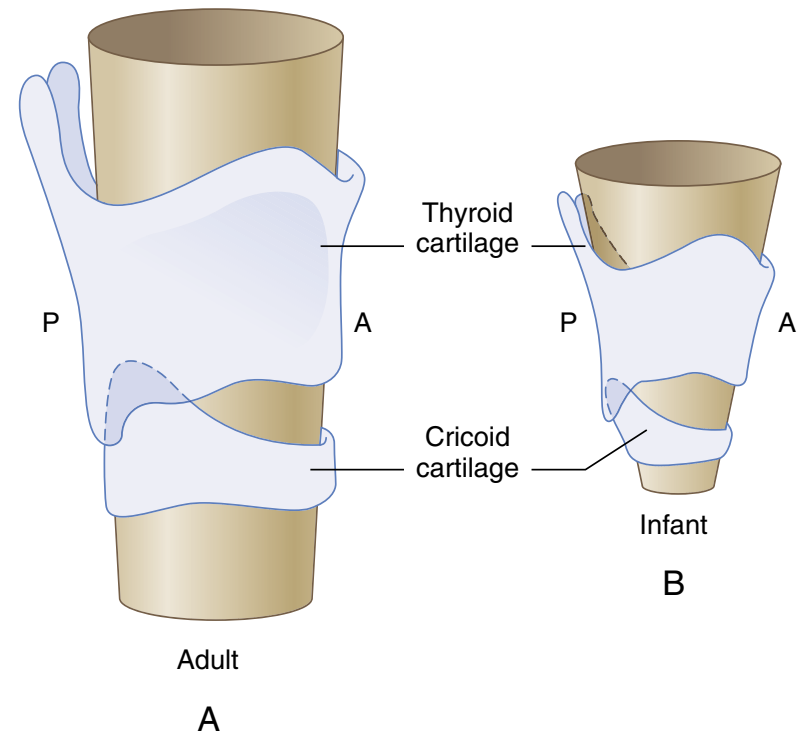
- Parasympathetic – fully developed at birth
- Sympathetic – not fully developed until 4-6 months age
- Infants and newborns more prone for bradycardia (with hypoxia, hypovolemia, laryngoscopy, pharyngeal suctioning, drugs – halothane, scoline) – atropine

10) Thermoregulation:

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

Grid:

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



Classification according to gestational age:

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

Classification according to birth weight

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

Airway:

- Airway in Pediatrics, unlike the adult airway is not a uniform entity, but encompasses a huge spectrum of assorted heterogenous entities.
- The difficulties of management of a normal airway in a neonate are very different and complex compared to the airway of a two year old and that of an adult.
- The neonatal airway is at the most difficult end of spectrum and as the infant grows into childhood the normal airway becomes easier to handle; but in many varieties of the abnormal airway, the difficulties may grow with the child. A thorough understanding of the normal airway, the mechanism of laryngoscopy and facilitation of tracheal intubation is mandatory.
- Pediatric patients have a proportionately larger head and tongue, narrow nasal passages, and anterior and cephalad larynx (at a vertebral level of C3-C4), a long epiglottis and a short trachea and neck.
- These anatomic features make neonates and most young infants' obligate nasal breathers until about 5 years of age. The cricoid cartilage (subglottis) is the narrowest point of the airway in children younger than 5 years of age.
- One millimeter of edema will have a proportionately greater effect in children because of their smaller tracheal diameters. Also, due to the shorter length of trachea endobronchial intubation and accidental extubation are more common with head movement.

- During laryngoscopy optimal head positioning displacement of tongue and soft tissues into mandibular space and pressure on the larynx are needed to achieve a straight line of vision between the eye and larynx. These are difficult to achieve in Pediatric airway. Certain modifications in the technique may aid in better visualization.
- Due to the large occiput, a small pillow placed under the occiput (similar to adults), will flex the head on neck instead of extending it for "sniffing position".
- Thus it is preferable to place a pad under the neck and shoulders, with a large ring under the occiput to stabilize the head to aid in optimum head positioning for laryngoscopy.
- The relatively large tongue can pose difficulty in being pushed into the "mandibular space". It can also cause obstruction to ventilation.
- The visualization of larynx becomes further difficult in presence of abnormalities like Pierre Robin syndrome, Goldenhar syndrome, Cystic hygroma etc.

Respiratory system:

- Independent life is not possible until gestational age is 2-26 weeks. Alveoli increase in number and size until the child is approximately 8 years old.
- Further growth is seen as an increase in the size of the alveoli and airways. At term, a full complement of surface active proteins help to maintain patency of the airway.
- In premature children respiratory failure (respiratory distress syndrome) is common due to deficiency of these surface active proteins.
- Oxygen consumption in the neonate 7 ml/kg-1 min-1 is almost twice that of adult value. This is seen as increased minute ventilation ($200 \text{ ml/kg-1 min-1}$) at puberty. As tidal volume remains constant at 7 ml/kg-1 throughout life, increased ventilation is brought about by an increase in respiratory rate; approximately 30/min at birth which progressively falls to adult values by adolescence.

Respiratory System Continuation:

- FRC in young infants at complete relaxation (central apnea, under general anesthesia, use of muscle relaxants) decrease to mere 10-15% of TLC.
- This low FRC is substantially below the closing capacity and results in small airway closure, atelectasis, ventilation/perfusion imbalance and hemoglobin desaturation.
- The small diameter of airways increase resistance to airflow. The airway of the infant is highly compliant and poorly supported by the surrounding structures.
- The chest wall is also highly compliant, so that the ribs provide little support for the lungs, thus the negative intra thoracic pressure is poorly maintained.
- Thus the work of breathing increases to approximately three times of the adult.
- Another important factor is the composition of the diaphragmatic and intercostals muscles.
- Type I muscle fibers which are fatigue resistant and able to perform repeated exercise are deficient in newborn and infants.
- The adult configuration is reached only by approximately 2 years of age. Any factor increasing the work of breathing contributes to early fatigue of the respiratory muscles.
- This fatigue can lead to apnea or carbon-dioxide retention and respiratory failure.

Control of breathing:

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

- The biphasic depression of ventilation during hypoxemia is due to central depression, rather than depression of peripheral chemo receptors.
- Both full term and premature neonates breathe irregularly. Periodic breathing i.e. rhythmic breathing inter-spread with a series of short apneic spells lasting less than 10 seconds without cyanosis or bradycardia, occurs both during REM and non REM sleep and even during wakefulness. Periodic breathing is about 80% in full term neonates, whereas it is nearly 1000% in premature neonates. The frequency of periodic breathing diminishes after 44 weeks post conception and with maturation during the first year of life.
- Central apnea is the cessation of breathing activity lasting longer than 15 to 20 seconds or a shorter apnea associated with bradycardia (HR<100) cyanosis or pallor. The mechanism is not understood but may be related to an immature respiratory control mechanism. Central apnea is rare in full term neonates but occurs in the majority of premature infants.

Postoperative apnea:

- Postoperative apnea is an important clinical issue in Pediatric anesthesia. Prematurely born infants, less than 44 weeks post conception, especially those with a history of apnea, are at high risk (20 to 40%) of developing postoperative apnea. Apnea can occur mostly within 12 hours postoperatively.
- There are a number of compounding factors associated with the development of postoperative apnea, such as the extent of surgery, anesthetic techniques, anemia and postoperative hypoxemia. High risk of postoperative apnea is associated in anemic infant (HCT <30%) regardless of post conceptional age.
- Both caffeine and theophylline are known to be effective in reducing the incidence of apnea in premature infants, strengthen muscle contractility and prevent fatigue and stimulate respiration.

- Apnea or hypoventilation unrelated to neuronal or central apnea can occur in infants and children of all ages who are predisposed to upper airway obstruction and may be exaggerated due to the residual depressant effect of anesthetics, opioids or sedatives.

Cardiovascular system:

- The fetal circulation, well adapted to the hypoxic intrauterine milieu, differs from the postnatal counterpart in a number of significant ways.
- At birth, the fetal circulation begins the transition to the postnatal type. With the first breath, the lungs become aerated and pulmonary vascular resistance falls, resulting in an increase in pulmonary blood flows. Left atrial pressure increases above right atrial pressure. Leading to closure of foramen ovale. Increased arterial oxygen tension causes constriction of ductus arteriosus. The ductus venosus and the umbilical arteries also constrict over several days.
- Fetal hemoglobin (HbF) in the intrauterine life is beneficial as it allows oxygen extraction from the maternal hemoglobin even at relatively low venous oxygen tension. This HbF is a disadvantage postnatally, as it impairs oxygen delivery to the tissues. Resting cardiac output is high in the neonate as compared to that of older child and adult. This allows the infant to meet oxygen demand, but the ability of the newborn to further increase the cardiac output during stress is limited. Stroke volume is relatively fixed due to a noncompliant and poorly developed left ventricle in the neonate and infant. The contractile apparatus comprises only about 30% of the neonatal heart compared to 60% in the adult heart. Thus the immature ventricle is characterized physiologically by both poor compliance and reduced contractility.
- Cardiac output at birth is 200 ml/kg-min which progressively decreases to 100 ml/kg-min by adolescence. Resting stroke volume remains fairly constant at about 1 ml/kg. The increased cardiac output in younger patients is being maintained by an increase in the heart rate. Normal heart rate which is approximately 150 beats per minute in neonates, progressively decreases throughout childhood. Activation of the parasympathetic nervous system, anesthetic overdose or hypoxia can cause bradycardia and profound reductions in cardiac output. The sympathetic nervous system and baroreceptor reflexes are not fully mature. The infant has reduced catecholamine stores and displays blunted responses to exogenous catecholamines. Thus vasoconstriction in response to hypotension is less manifested and hypotension without tachycardia is the hallmark of intravascular fluid depletion in neonates and infants.
- Systolic blood pressure which is around 80 mmHg at birth, progressively increases to 120 mmHg at puberty, keeping pace with the perfusion demands as the child assumes sitting and standing positions. Diastolic blood pressure also increases associated with an increase in myocardial mass and to ensure adequate coronary blood flow during diastole.
- ECG findings in neonates and children are different from those of adults. The ECG changes with age reflect the development of the myocardium. Due to the right-sided predominance of the fetal heart, the neonatal ECG shows a marked right axis deviation (+300 to +1800) compared to adults (-300 to +1050). Also seen are tall 'R' waves in the right leads and deep 'S' waves in the left leads, shorter QRS duration, shorter PR interval, T waves inverted toward the left.
- Innocent murmurs are common in children and may be present in up to 80% of children. Innocent systolic murmurs include vibratory Still's murmur, basal systolic ejection murmur, cardiorespiratory murmur and murmur of physiologic peripheral pulmonary stenosis. Venous hum is a continuous murmur heard throughout the cardiac cycle.

- **Cycle Murmurs heard only during diastole are pathologic.**
Other innocent heart sounds, present in childhood include carotid bruit and third heart sound.
- Certain risk factors increase the likelihood of reversion from adult circulation to a fetal type of circulation, known as transitional circulation. Factors include hypoxia, hypercarbia, anesthesia induced changes in peripheral vascular tone. When this reversal occurs pulmonary artery pressure increases to systemic levels. Blood gets diverted past the lungs via the patent foramen ovale, and opening of the ductus arteriosus, allowing blood to shunt at the ductus levels. A rapid downhill course may occur, causing severe hypoxia.
- Certain risk factors increasing the likelihood of prolonged transitional circulation include prematurity, infection, acidosis, pulmonary diseases resulting in hypercarbia or hypoxemia, hypothermia and congenital heart disease. Thus care must be directed to keeping the infant warm, maintaining normal arterial oxygen and CO₂ tension, and minimizing anesthetic induced myocardial depression.
- Developmental myocardial immaturity accounts for the tendency toward biventricular failure, sensitivity to volume loading poor tolerance to increased after load and heart rate dependant cardiac output.

Physiological differences between neonatal and adult myocardium

	Neonate	Adult
Cardiac output	Heart rate dependant	Stroke volume and heart rate dependent
Contractility	Reduced	Normal
Starling response	Limited	Normal
Compliance	Reduced	Normal
After load compensation	Limited	Effective
Ventricular interdependence	High	Relatively low

Age-related changes in vital signs.

Age	Respiratory rate	Heart rate	Arterial blood pressure	
			Systolic	Diastolic
Neonate	40	140	65	40
12 months	30	120	95	65
3 years	25	100	100	70
12 years	20	80	110	60

Nervous System	Spinal Cord	Dural Sac
At Birth	L3	S3
In Adult	L1	S2

Blood:

- Blood volume is highest in new born averaging 86 ml/kg-1 in term neonates and 106 ml/kg-1 in premature and small for gestational age infants. Normal adult values are 70-80 ml/kg-1.
- In the neonates hemoglobin is primarily of the fetal type (HbF) which has a higher affinity for oxygen than adult hemoglobin. This combined with low 2,3-DPG levels, results in a left shift of the oxy-hemoglobin dissociation curve and poor oxygen delivery to the tissues. Hemoglobin levels which are at 16 – 19 gm/dl and high hematocrit of approximately 55 at birth progressively decrease, reaching the lowest values at about 2 months. This is due to a relatively hypoxic environment in utero, which stimulates the production of erythropoietin and red cell production. After birth, there is a sharp fall in erythropoietin activity due to the greater availability of oxygen. By 6 months of age, mean hemoglobin is 12.5 gmdl-1 and maintained till 2 years of age. Thereafter there is gradual increase upto puberty.

Renal function:

- The kidneys are immature at birth and both glomerular filtration and tubular function are reduced. The GFR is low in the newborn, rises sharply in the first 2 weeks of life, and reaches adult values by 2 years, when complete maturation of renal function occurs. The ability to handle free water and solute loads is impaired and half life of drugs excreted through glomerular filtration will be prolonged.
- Premature neonates often possess multiple renal defects, including decreased creatinine clearance, impaired sodium retention, glucose excretion, bicarbonate reabsorption and poor diluting and concentrating abilities. Thus, meticulous attention is in fluid administration.

Fluid balance:

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

Age related changes in TBW and its distribution

	Preterm	Term	1-3 years	Adults
TBW	85%	80%	65%	65%
ECF	53%	45%	25%	25%
ICF	30%	35%	40%	40%

- Maintenance fluid requirements are related to metabolic rate. In general 1 ml fluid is required for every 1 Kcal expended. Requirements for sodium, potassium and chloride are usually quoted as 30, 20 and 20 mmol 1000 kcal-1. This requirement of both fluids and electrolytes can be met with infusion of a solution of 0.18 % NaCl, 4% dextrose and 20 mmol KCl/lit at a rate equal to caloric expenditure based on Holiday and Segar rule of 4:2:1. The rationale behind adding dextrose in Pediatric maintenance fluid is that though it provides only 20% of total calories required for a child <10 kgs, it is sufficient to prevent ketosis.
- Perioperative fluid management is divided into three phases maintenance, deficit and replacement of losses.
- Maintenance fluid requirement can be managed as outlined above by Holiday and Segar rule. Fluid deficits are calculated and replaced based on duration of fasting, presence of associated conditions like fever, vomiting, diarrhoea, sweating and particular disease state or surgical problem likely to affect fluid status (bowel obstruction, peritonitis etc).
- Intraoperative losses are subdivided into third space loss and blood loss.

- Third space losses surgical trauma, blunt trauma, infection and may surgical conditions are associated with the isotonic transfer of fluid from the ECF to a non functional interstitial compartment. This is called third space loss and is impossible to measure, and may be estimated by the extent of surgery and the clinical response to appropriate fluid replacement. The magnitude of third space loss is usually highest in infants undergoing intra-abdominal procedures, and least in superficial surgery or neurosurgery and approximate ranges are:
- Intra-abdominal surgery: 6-10 ml/kg-1h-1.
- Intra-thoracic surgery: 4-7 ml/kg-1h-1.
- 1 - 2 ml/kg/hr in following patients:**
- Eye surgery
- Neurosurgery
- Superficial surgery
- The aim is to replace sequestered plasma volume and Ringer's lactate is an appropriate replacement fluid. The clinical response to appropriate replacement is a sustained and adequate blood pressure and heart rate adequate tissue perfusion and uterine output of 1-2 ml/kg-1h-1.
- All blood loss in Pediatric patients requires replacement. The anesthesiologist should have a preoperative plan regarding blood loss replacement, based on the patient's preoperative condition, preoperative hematocrit and nature of surgery. The concept of an allowable blood loss (ABL) is a useful approach. Generally, a hematocrit of 28-30% is acceptable, although in neonates a value of 40% is more appropriate. In determining ABL, an estimate of blood volume (EBV) must be first made:
- Premature neonate – 90-100 ml/kg-1.
- Term neonate – 80-90 ml/kg-1.
- 3 months to 1 years -75-80 ml/kg-1.
- 3-6 years – 65-70 ml/kg-1.
- ABL is calculated using the formula:
- $ABL = \text{Weight} \times EBV \times (H_o - H_1) / H_a$
- H_o is the starting hematocrit
- H_1 is the lowest acceptable hematocrit

- H_a is the average hematocrit $H_o + H_1/2$

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

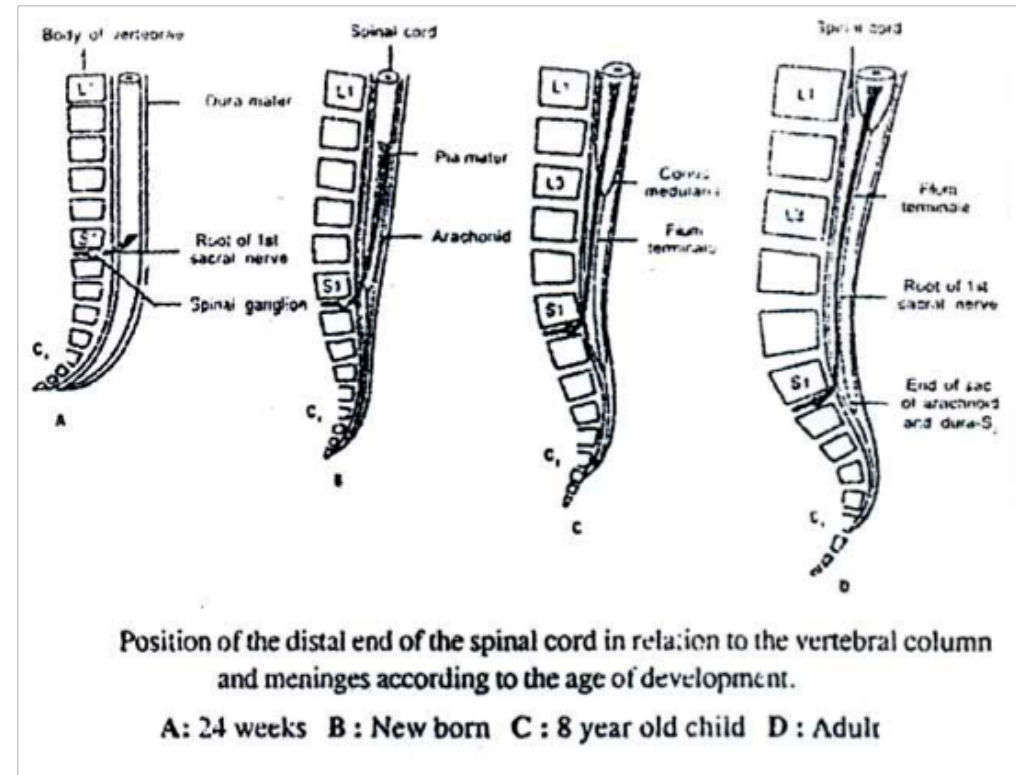
Central and autonomic nervous system:

- Although the nervous system is anatomically complete at birth, myelination continues, and functionally it remains immature. Myelination of the nervous system is rapid during the first two years of life and is accompanied by rapid advances in motor function. These advances occur in a rostrocaudal fashion. Myelination is complete by 7 years of age.
- Brain is solely dependent on glucose for its energy source as it is the only molecule capable of crossing the blood brain barrier (BBB). Despite the enormous glucose requirement (6.8 mg glucose 100 mg -1 min-1 in child verses 5.5 mg glucose /100mg/min in adult), brain does not store glucose and does not elaborate any glycogen. The brain glucose reserve only secures 3 minutes of energy supply, enough to maintain normal cerebral function.
- Basic glucose consumption - 0.3 to 0.8 mmol 100g-1min-1.
- Oxygen consumption (CMRO2) - 3.5 ml O2 100 g-1min-1 (adults)
5.5 ml O2 100 g-1min-1 (children)
- Increased O2 consumption is related to the energy requirements of growth.

Cerebral blood flow:

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

- Age related reduction in cerebral metabolism and CBF is mainly due to loss of synapses or a synapse or a drop in their activity rather than to a neuronal anatomic loss.
- Cerebral blood flow is matched to CMRO₂. Cerebral autoregulation is present and capable of rapid response in infants and young children.
- The blood brain barrier (BBB) consists of tight junctions and is impermeable to electrolytes. This BBB is immature at birth, but develops rapidly in postnatal life. The less mature BBB permits the passage of larger, lipid soluble molecules and various drugs.
- Sympathetic and parasympathetic functions do exist in neonates but do not mature until later in infancy. There is a predominance of parasympathetic response system.
- New born also respond to noxious stimuli with facial grimaces as well as cardiovascular and metabolic stress responses, suggesting perception of pain. Thus, the anesthesiologist should aim at attenuating the stress response as well as to prevent the perception of pain by administering sufficient analgesia.
- **Spinal cord following retrogressive differentiation at birth, the spinal cord ends at the intervertebral level L3. Reaching the adult level of L1 to L3 at the age of 8 years.** During embryonic life, the spinal cord fills the spinal canal, but from the fetal period onwards, the growth of osseous structures exceeds that of neural structures, thus, the cord and dural sac terminates at progressively higher levels.
- Due to lower termination of these essential structures, lower intervertebral approaches to the equidural and subarachnoid spaces are recommended in infants to avoid any inadvertent neurologic damage. Myelination begins in cervical neuromeres and progressively extends downwards and upward, but is not achieved until 12 years shorter distance between successive nodes of Ranvier favor penetration of local anesthetics and rapid onset of nerve blockade even with the use of diluted solutions.



Thermoregulation:

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

- The infant is particularly vulnerable to hypothermia because of both large ratio of body surface area to weight and a limited ability to cope with stress. The premature infant is even more susceptible because of very thin skin and limited fat stores. Compensatory mechanisms to this may be shivering and non shivering (cellular metabolic) thermogenesis. Thermogenesis by shivering is minimally developed during the first three months of life, making non shivering thermogenesis (metabolism of brown fat) the principle method of heat production. This non shivering thermogenesis depends on:
 1. Intact surface and central responses to the difference between the skin temperature and that of the environment.
 2. Intact sympathetic nerve endings to release the catecholamines that stimulate the catabolism of fat
 3. The cardiopulmonary capacity to increase oxygen uptake and delivery and
 4. The availability of fat stores to provide free fatty acids which undergo complete metabolism and generate calories.
- Oxygen consumption also increases in direct relation to the increasing differences between skin temperature and environmental temperature as the difference exceed 40C. Thus all steps should be undertaken to minimize heat loss. This can be achieved by various devices like; use of warming. This can be achieved by various devices like; use of warming mattresses, blankets, warm IV fluids and blood, warming mattresses, blankets, warm IV fluids and blood, warming and humidifying anesthetic gases, over head radiant heaters of incubators for transport, use of plastic wrap of decrease evaporative loss, warming of preparation solution, and increasing the operating room temperature.

Gastro intestinal system:

- At birth, the functional maturity of the liver is incomplete. Most enzyme systems for drugs metabolism although developed, are not yet induced (stimulated) by the agents they metabolize. As the infant grows, the ability to metabolize drugs increases rapidly in two ways.
 1. Hepatic blood flow increases and more drug is delivered to the liver and
 2. The enzyme systems develop and are induced.
- Conjugation reactions are often impaired in the neonates, resulting in jaundice, decreased degradation reaction leading to long drug half lives. Thus longer drug elimination half life is seen in the neonate, whereas infants and older children have shorter drug half life.
- Also seen are minimal glycogen stores, inability to handle large protein loads, lower levels of plasma albumin and other drug binding proteins. These factors account for tendency to hypoglycemia and acidosis.
- The lower albumin levels contribute to neonatal coagulopathy, decreased drug binding and higher levels of free drug.
- At birth, the gastric pH is alkalotic, and is gastric acid production increases it reaches normal adult values by the second day.
- In neonates, infants lower esophageal sphincter tone is decreased.
- Also the ability to co-ordinate swallowing with respiration is not fully matured till 4-5 months of age.
- These two factors increase the incidence of gastroesophageal reflux.

Conclusion:

- Pediatric anesthesia involves perioperative and critical care of patients of all ages ranging from preterm infants to teenagers. The differences in physiological characteristics makes anesthetic management different and extremely challenging for the anesthesiologist.
- It is imperative to have a good knowledge of the anatomic and physiologic patient for conduct of safe anesthesia.

Introduction

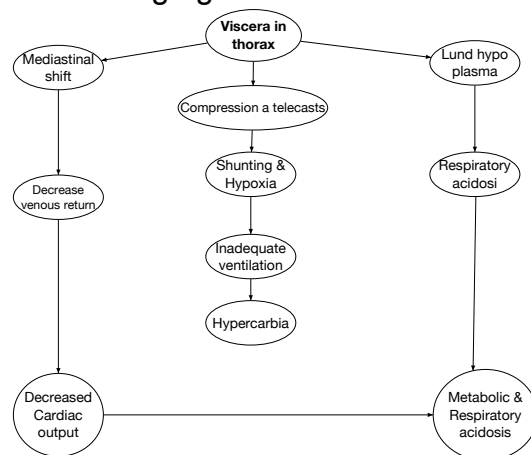
- Congenital diaphragmatic hernia (CDH) may present as a life threatening emergency which warrants rapid resuscitation; correction of acidosis fluid deficit, hypothermia and baby prepared for emergent surgery. CDH occurs in approximately 1:2500 live births, with a male to female ratio of 2:1. Left diaphragm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

Embryology

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

Pathophysiology:

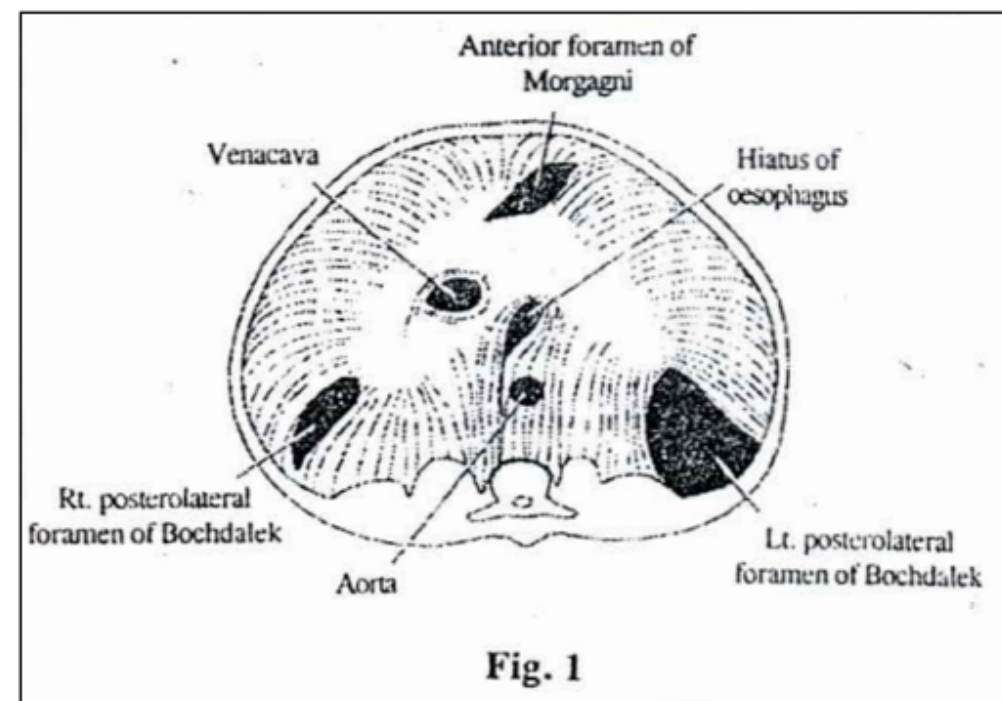
- The spectrum of clinical presentation varies, depending upon the period of lung invasion and pulmonary agenesis. Developmental defect in the diaphragm leads to herniation of the abdominal viscera into the pleural cavity leading to pulmonary hypoplasia and mediastinal shift with consequent effects. The commonest site being right or left foramen of Bochdalek.



- As the contralateral lung is always spared, the infant is capable of sustaining respiration and gradually ipsilateral lung expands and becomes functional. Subsequent picture depends on the development of pulmonary hypoplasia and pulmonary vasculature.

Causes of hypoxemia:

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



Definition:

- CDH is the herniation of the abdominal viscera into the chest through the defect in the diaphragm.

Classification based on anatomical defect in the diaphragm:

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

Assessment of severity of pulmonary hypoplasia

- $PAO_2 - PaO_2 > 500$ mmHg breathing 100% O_2 - predicts non survival; 300-500 mmHg - survival uncertain and values <400 mmHg; has better prognosis.
- Cardiac catheterization, echocardiography, colour doppler pulmonary angiography.
- Bohn's index prognosticates information as 'ventilatory index' (V.I.): product of mean airway pressure X respiratory rate..
- Neonate with $PaCO_2 < 40$ mmHg and VI < 1000 always survive, while $PaCO_2 > 50$ mmHg, VI < 1000 or $PaCO_2 < 40$ mmHg, VI > 1000 usually die.

Signs and symptoms

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

Monitoring

- Precordial / oesophageal stethoscope, pulse oximeter both above / below the nipple for preductal and postductal SpO_2 , capnography, inspiratory pressure, FiO_2 , acid base status, ECG, invasive blood pressure in right radial artery for preductal PaO_2 , CVP volume status and right ventricular performance, thermoregulation - oesophageal, rectal temperature.

Surgical Correction

- Repair of hernia is not a surgical emergency unless the contents of hernia are incarcerated. Improved ventilation and maintenance on ECMO or other treatment strategies to control pulmonary hypertension, ductal shunting and ineffective oxygenation are continued till pH is reversed and lung functions improve (7-10 days). Patients not amenable to improve on ECMO or with 3 weeks treatment have bad prognosis.
- Subcostal or thoraco-abdominal incision is taken, herniated abdominal contents are pulled back and the muscular defect in the diaphragm repaired. If closure of abdomen exacerbates respiratory distress only skin may be sutured or a separate pouch be created temporarily. Sudden hypotension, bradycardia, decreased SaO_2 and pulmonary compliance suggest tension pneumothorax. Contralateral pneumothorax can be disastrous. Decreased venous return due to tight abdomen should be suspected if no improvement is seen after treatment of pneumothorax.

Cantrell's Pentalogy:

- Omphalocele
- Diaphragmatic Hernia
- Intracardiac defects - PDA
- Failure of closure of Posterior pleuroperitoneal canal - PPP - 8 weeks 1:2500

Preoperative management

- Assess associated anomalies.
- Hypothermia increases O₂ consumption, optimal environmental temperature suggested is 30° C-40°C.
- Arterial blood gases, complete blood count, serum electrolytes, blood sugar, cross matching.
- Correct metabolic acidosis (glucose, fluids) and respiratory acidosis (proper ventilation/sodium bicarbonate) prior to surgery. Sodium bicarbonate may be given empirically as diluted 0.5 mEqml⁻¹, (2-4 mEqkg⁻¹) I.V. infusion at < 1 mEqKg⁻¹min⁻¹.
- Venous access must be secured in the upper arm; neck veins reserved for ECMO. Central vein access is via umbilical or femoral veins.
- Use of vasodilators - tolazoline, prostacycline, dipyridamole, nitric oxide.
- Minimize sympathetic discharge by high dose opioids.
- Nasogastric suction for gastric decompression.
- Transport to OT with manual ventilation.

Anesthetic considerations

- Infant in semi-sitting position is warmed with warming devices, humidified inspired gases and warm transfused fluids at 37°C. After pre-oxygenation, atropine 0.02 mg/kg is administered I.V. Awake intubation is preferred. Alternatively infant is induced with halothane / Sevoflurane in O₂ and intubated while breathing spontaneously. Avoid mask ventilation, to obviate gastric distension and respiratory embarrassment.
- Gentle hand ventilation is preferred to avoid ipsilateral or contralateral pneumothorax. Anesthesia is maintained with volatile agent in 100% O₂ or with 0.5 mg/kg ketamine with titrated dose 1-3 µkg⁻¹ fentanyl. PPV is avoided till intubation.
- Patient in shock and severe hypoxemia is maintained on supplemental O₂, non depolarizing muscle relaxants (pancuronium / vecuronium) and analgesics. Inhalation agents, opioids (fentanyl) and muscles relaxants (pancuronium) are added in titrated doses.

- N₂O is avoided. It may produce intrathoracic gut distension, inability to close abdomen and increased intra abdominal pressure, leading to precipitous hypotension.
- Selection of appropriate FiO₂ depends upon severity of pulmonary dysfunction. To avoid hypoxia increasing R → L shunt of Desaturated blood, inhaled gases with higher O₂ content is suggested. PaO₂ should be optimally kept between 80-100 mmHg or arterial O₂ saturation at 95-98%.

Ventilation:

- Infant is ventilated with small tidal volume and low inflation pressure (< 20 mmHg) to prevent contralateral pneumothorax. Respiratory rate (60-120 min⁻¹) is adjusted to achieve hypocarbia (PaCO₂ 25-30 mmHg), lower pulmonary vasoconstriction and minimize R→ L shunt through the ductus arteriosus.

Pulmonary hypertension and management

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

Treatment

- Continue ventilation in ICU using fentanyl 3µg/kg/hr, pancuronium 0.1 mg/kg/hr to blunt autonomic cardiovascular response (pulmonary vasoconstriction) to stimulation.
- Minimize endotracheal suction, to avoid transient hypoxemia.
- Hyperventilate the neonate with low VT and high RR (60-120 min⁻¹) to maintain pH of 7.55-7.60 and induce pulmonary vasodilation.
- Restrict fluids to 2-4 mg/kg/hr
- Administer pharmacologic pulmonary vasodilators morphine, chlorpromazine, prostaglandin E1 and inhaled nitric oxide.
- Severe lung hypoplasia and refractory pulmonary hypertension with arterial O₂ saturation <50 mmHg at FiO₂ of 1.0 needs ECMO therapy immediately to avoid progressive pulmonary injury. ECMO is associated with 50-60% survival rate.
- Nitric oxide (NO) at 20-80 ppm a endothelial derived relaxing factor is selective pulmonary vasodilator with no effect on systemic circulation and is immediately inactivated on exposure to Hb.
- High frequency oscillatory ventilation (HFOV) has been used as pulmonary ventilatory therapy to improve oxygenation.

Fluid replacement

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

Postoperative management:

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

Introduction

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

Risk categorizations

Spitz classification

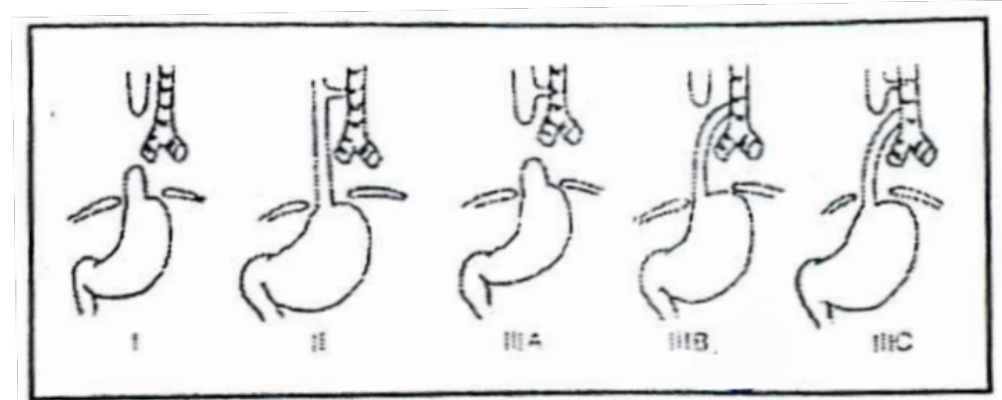
- Grade I - Body weight > 1500 g without cardiac disease (survival 97%)
- Grade II - Body weight > 1500 g and major cardiac disease (survival 59%)
- Grade III - Body weight < 1500 g and major cardiac disease (survival 22%)

Waterston and colleagues

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

TOF types: Cross and Vogt classification

- Type I - esophageal atresia with no fistula
- Type II - No atresia, communication between trachea oesophagus (H-type fistula)
- Type III - esophageal atresia, upper segment communicating with trachea
- Type IIIB - esophageal atresia with blind upper pouch and lower segment communicating with the trachea (commonest)
- Type IIIC - esophageal atresia with both upper and lower segments communicating with the trachea.



Pathophysiology

- Physiology of swallowing is disturbed and secretions/ saliva accumulates in the blind upper pouch. Fistulous communication entrains air into the stomach via the trachea causing gastric distension. Overflow of saliva feed from the blind upper pouch and regurgitation of gastric contents via the fistula leads to clinical pneumonitis.
- Two main pathological entities in TOF are dehydration and aspiration pneumonitis.

Clinical presentation and diagnosis:

- 3Cs – choking, coughing, cyanosis. Cyanosis of oesophageal atresia are secondary to oesophageal discontinuity and respiratory complications.
- Every feed is regurgitated
- Abdomen usually distended but in the absence of fistula, respiratory symptoms are less and abdomen is Scaphoid.
- Diagnosis is established when there is inability to pass suction catheter beyond 10cm Radiography shows a coiled catheter in the blind proximal pouch, and air accumulation in the stomach. Instillation of radio-opaque dye into the proximal blind loop is avoided for fear of pulmonary aspiration.

Evaluation

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

Monitoring

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

Preoperative preparation

- The priorities are to save life, achieve alimentary continuity and preserve esophagus

Prevention of aspiration:

- Avoid feeding, nurse the baby in propped up position, keep proximal pouch empty by aspirating the pooled saliva every 15 minutes. Catheter should be left in upper blind pouch.

- Before shifting, the operating room temperature should be above 27°C.
- Optimize pulmonary status: chest physiotherapy, tracheal suction, supplemental oxygen and antibiotics.
- Secure appropriate intravenous and arterial access.
- Maintain volume and metabolic status.
- Arrange blood.

Surgery

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

Anesthetic considerations:

- Aspiration pneumonia, securing airway, oxygenation, gastric over distension, and problems associated with coexisting anomalies. Management may be staged, if TOF co-exists with prematurity, pneumonitis or other congenital anomalies.

Problems

- Inadequate oxygenation and ventilation; leakage of gases through the fistula, ETT misplacement or of endobronchial intubation.
- ETT blockade; periodic ETT suctioning; in case of blocked ETT, replace with a new ETT over a tube exchanger.

Problems. Continuation:

- Ventilation/perfusion (V/Q) mismatch in anesthetized lateral decubitus position may be compounded by atelectasis secondary to retraction of the non-dependent lung, bronchial traction and ETT kinking. A high FiO₂ is desirable to keep the SpO₂ between 95-98%. The right lung is intermittently inflated to prevent hypoxemia.
- Vagal response to tracheal manipulation is avoided by prior atropine administration.
- Hypothermia produces peripheral vasoconstriction, non-shivering thermogenesis and increased O₂ consumption. It also affects pharmacokinetic and pharmacodynamics profile of anesthetics leading to over dosage, postoperative hypoventilation, apnea, coagulopathy and metabolic acidosis. Therefore, body and ambient temperature is continuously monitored. Infant must be protected by heating devices and use of warm infusion fluids. Proper ETT positioning is achieved by placing the tip beyond the fistulous opening and just short of carina to avoid gastric distension during positive pressure ventilation (PPV), and endobronchial intubation [confirmed by flexible fiberoptic bronchoscope (FOB)].
The tube is inserted with bevel facing posteriorly to avoid entering the fistula and then secured with the bevel facing anteriorly. Prevention of gastric distension to prevent respiratory embarrassment is achieved by;
 - Preoperative gastrostomy or an emergency procedure if massive gastric distension produces respiratory embarrassment and/or difficult PPV.
 - Retrograde placement of balloon-tipped catheter in the fistula, through the gastrostomy under guidance with FOB or antegrade occlusion of TOF with a balloon tipped Fogarty catheter advanced through the trachea.
 - Application of snug abdominal binder.

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

Anesthetic technique

- Precordial stethoscope is fixed in left axilla, infant is placed in left lateral position with right upper limb positioned above the head (pulse oximetry is important to diagnose dislodgment of ETT). Awake intubation is preferred. No anesthetic agent is contra indicated. Infant is maintained on spontaneous or assisted ventilation. (Paw <10-15cmH₂O) till the fistula is ligated. Inhalational induction with sevoflurane or halothane is preferred alternative. 90-95% SpO₂ saturation must be maintained. Adequate oxygenation should never be compromised to avoid retrolental hyperplasia.
- Use of N₂O in anesthetic mixture is debatable owing to possibility gastric distension.
- Narcotics are given for analgesia. Anesthesia is maintained with O₂ in volatile anesthetic agents (sevoflurane or halothane) along with small titrated doses of non depolarizing muscle relaxants.
- Atraumatic suction of the dependent lung and tracheo-bronchial toilet is essential at the end of surgery, to avoid atelectasis or accumulation of secretions/blood.

Postoperative Care

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

Fluid management

- Maintenance of fluid should be with 5% dextrose in 0.2 - 0.25% saline solution. Insensible loss should be replaced with a balanced salt solution at the rate 6-8 ml/kg/hr.
- Blood loss should be carefully monitored and replaced.

Pain management

Neonates require adequate pain control by neuraxial or parenteral route.

- Morphine : 0.1 mg/kg/hr in full term and, 0.05 mg/kg/hr in premature babies.
- Epidural Bupivacaine 0.1%, Fentanyl 0.5µml-1 at the rate 0.1-0.2 mg/kg/hr.
- Acetaminophen: Suppositories 35-40 mg/kg followed by 20 mg/kg.
- Infiltration of surgical incision with 0.25% bupivacaine in a dose of 0.5 ml/kg.

Postoperative complications**Immediate:**

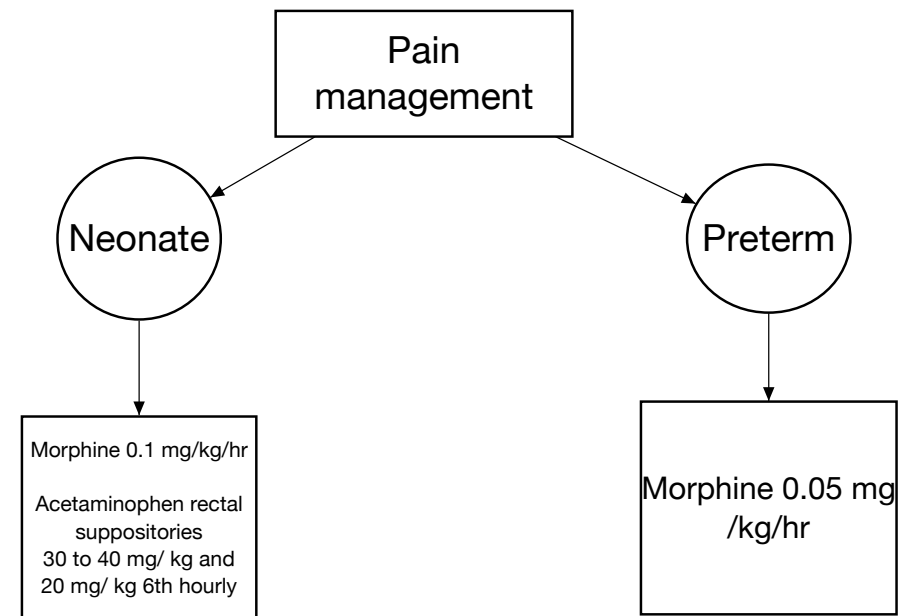
- Anastomatic leak (16%), symptomatic stricture (35%), gastro-oesophageal reflux (58%), tracheomalacia (15%).

Late:

- Due to food trapping in lungs, recurrent bronchitis, lung infection and recurrence.

GRID: Question to be answered under following headings:

1. what is TEF
2. Incidence and associated anomalies.
3. Diagram & Classification
4. Risk Factors and diagnosis
5. General Management
6. Surgical Management
7. Preoperative evaluation
8. Monitoring
9. Preoperative preparation
10. Anesthetic Management and Consideration.
11. Post operative complications
12. Post operative pain management



Introduction

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

Pathophysiology:

- Sequelae and effects of FB aspiration depend on the site degree and duration of airway obstruction.

Acute phase:

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

Latent phase: (few hrs to months):

- Careful examination, expiratory wheeze or obstructive symptoms may localize FB.

Chronic phase:

- Is demonstrated by productive cough or superadded infection.

Four types of obstruction and their effects

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

Pharmacology

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

Symptoms and signs

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

Problems

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

Goals

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

Preoperative considerations

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

Monitoring

- Precordial stethoscope -inspection of chest movements. SpO₂, ETCO₂
- Neuromuscular transmission with TOF nerve stimulator.
- One overhead spotlight towards anesthetic machine, second over the child's feet to assess saturation and perfusion.

Anesthetic management**Acute emergency**

- GA is always required because of fighting irritable, child removal of distally lodged invisible FB and if prolonged bronchoscopic procedure is contemplated. Based on feeding history, anesthesia can be induced with inhalational or intravenous technique.
- Spontaneous technique is safer than apnoeic technique with complete neuromuscular blockade for fear of distal dislodgement. Sevoflurane is preferred over halothane because of smooth induction, its antitussive and cardio-respiratory stability properties.
- Anesthesia is induced with sevoflurane 3-5% in 100% oxygen and reinforced with topical lidocaine 1-4 mgkg⁻¹ prevent laryngospasm.
- Anesthesia is then maintained with 100% oxygen in halothane or isoflurane as these are more soluble and eliminated slowly, giving more time for airway manipulation, avoiding lighter plane and associated hemodynamic reflexes. Deeper plane of anesthesia with spontaneous respiration is required during attempts to extract FB.
- During instrumentation or sanction of FB if the child coughs, small increments of propofol or fentanyl are administered.
- Propofol based IV anesthesia supplemented with topical spray of lidocaine can alternatively maintain a steady level of anesthesia, independent of ventilation or O.T. pollution.

Stable child

- After premedication with atropine, anesthesia is induced with thiopentone (4-6 mgkg⁻¹), fentanyl (0.5-1 µgkg⁻¹), followed by atracurium (0.3-0.5 mgkg⁻¹) vecuronium (0.07-0.1 mgkg⁻¹) / mivacurium (0.2-0.3 mgkg⁻¹) depending on the estimated duration of endoscopy.
- Muscle relaxant and PPV enables a lighter plane of anesthesia and ensures quieter field for the surgeon. Anesthetic gases are attached to the side port of the ventilating bronchoscope and flow adjusted to control respiration, keeping in mind the peri-bronchoscope gas leakage.
- Continual monitoring of chest excursions, breath sounds and oxygen saturation is essential because of widespread-atelectasis, low FRC and low O₂ reserve, increased shunting with a possibility of sudden drop in SpO₂ and rise in PaCO₂ during apneic spells.

- During active ventilation, the telescope/forceps is withdrawn to mid tracheal level, proximal end of endoscope is blocked with glass obturator or thumb for optimal ventilation. Apnoea time should not exceed one minute.
- Irrespective of the anesthetic technique (spontaneous/controlled), once the FB is grasped, bronchoscope holding forceps and FB are withdrawn as a single unit. Depth of anesthesia may need to be increased or child temporarily paralysed with succinylcholine 0.25-0.5 mg/kg to provide quiet, relaxed upper airway

Child with respiratory distress and emphysema

- 100% O₂ is supplemented, anesthesia induced in sitting position with sevoflurane and O₂ with facemask. IV access established and atropine given. Anesthesia deepened while ventilation assisted with mask and bag, bronchoscopy, carried out. Muscle relaxants are avoided till the airway is secured.
- When the large FB removed from the bronchus gets dislodged in the trachea or larynx, blocking the entire airway, it must be removed immediately or pushed back into the bronchus and the child ventilated.

Child with suspected full stomach / severe respiratory distress

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

3 Ventilation technique

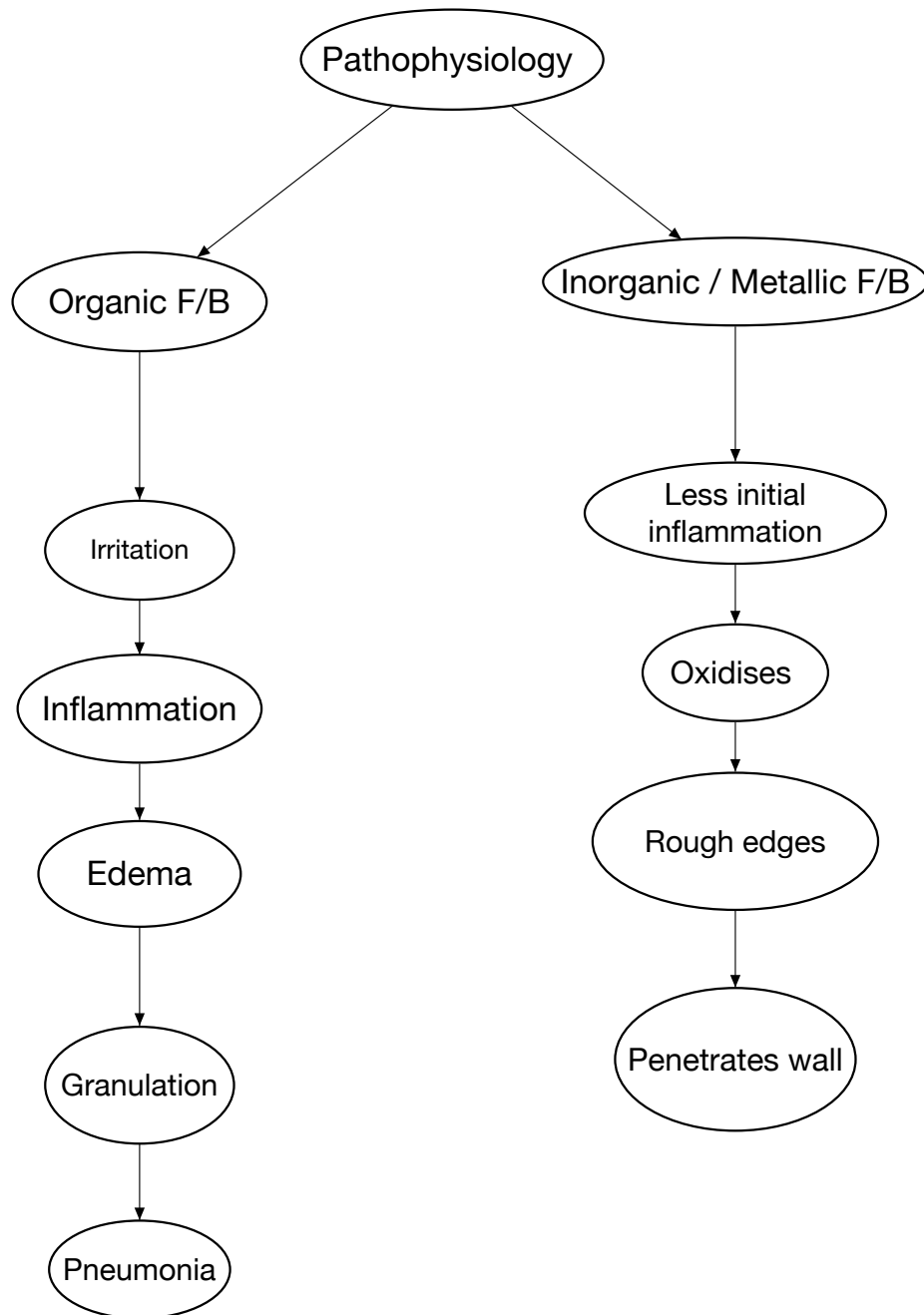
- The ventilation can be manual, IPPV, HFJV or HFPPV. HFJV is preferred through 14G needle attached to bronchoscope with respiratory rate of 120-140 min⁻¹, flow rate of 5-15 L/min with 30-40% inspiratory time. Uninterrupted oxygenation and free, quiet surgical field is provided to visualize the FB.

Intraoperative complications

- Laryngospasm, bronchospasm, pneumothorax and cardiac arrhythmias may occur. Atropine to minimize secretions; muscle relaxation for adequate ventilation and oxygenation; pulse oximetry to monitor O₂ saturation are preferred. Arrhythmias associated with hypercarbia are treated with hyperventilation and IV lignocaine (1 mg/kg). Chest tube is inserted if pneumothorax is confirmed.

Postoperative care

- Bronchoscopy is repeated to evaluate residual FB fragments and the impact site for trauma, bleeding or granulations. Child may be intubated for tracheobronchial toilet and ventilation until adequate spontaneous breathing is established.
- Minimize postoperative stridor and distress by Dexamethasone (0.1-0.25 mg/kg) for laryngeal, oedema, humidified oxygen, nebulized racemic epinephrine (2.25% solution given 1:6 to 1:10 dilution) under ECG monitoring, β -agonist bronchodilators such as albuterol for postoperative wheezing.
- 24-48 hrs intubation may be necessary till oedema subsides.



Introduction

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

Physiological changes

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

Pneumoperitoneum (PNP)

- Carboperitoneum permits improved visualization of the operative field. The volume of insufflating gas required in children is typically 0.9 L/1.0 kg-1 as against 2.5-5.0 L in adults. Low insufflating pressures help control hypercarbia, allow better ventilation and improved oxygenation, limit increased peripheral venous pressure following insufflation and reduce the risk of CO₂ embolism.
- Complications of Veress needle puncture of hollow viscera and blood vessels with consequent visceral complications must be kept in mind.

- Ideal gas for PNP should not support combustion, be rapidly excreted, have high blood solubility and allow minimal absorption, physiologic perturbation and possibility of embolism.
- CO₂ is safe for use during electrocautery, and gets eliminated through the lungs quickly. But smaller distance between capillaries and peritoneum and greater absorptive area of peritoneum in relation to body weight leads to significant vascular absorption in children.
- Hypercapnia leads to increased sympathetic activity sensitizes the myocardium to volatile anesthetic agents induced catecholamines (arrhythmia). In the postoperative period ventilation is impaired by residual anesthetic drugs and diaphragmatic dysfunction. Slow CO₂ insufflation might need increased minute ventilation (upto 60%) to restore EtCO₂ to base line specially during prolonged surgery.

Other means for pneumoperitoneum

- Helium (costly, CVS complications) and N₂O (supports combustion) induced PNP did not gain popularity. Alternatively, gasless technique lifts the abdominal wall without implications on IAP.

Intra-abdominal Pressure (IAP)

- Pneumoperitoneum raises the IAP with significant effects on all systems.
- Cardiovascular effects during laparoscopy are due to IAP and patient positioning.
- IAP < 15 mmHg - Venous return (VR) is augmented as blood is squeezed out of the splanchnic bed producing increased cardiac output (CO). It benefits infants with hypovolemia who cannot tolerate volume loss from the sequestration of blood.
- IAP > 15 mmHg - Decreases VR as IVC is compressed, decreasing CO and arterial BP. The compensatory flow via by the capillaries is ineffective as the collateral vessels are simultaneously compressed.
- There is a progressive decrease in cardiac index (CI) with an increase in IAP (fall in CI is 55% and 38% of baseline at 20 and 30 mmHg IAP respectively). There is simultaneous (35%) fall in renal, hepatic and mesenteric blood flow at an IAP of 25 mmHg.

IAP.Continuation:

- Low IAP in cardiac compromised patients is recommended as IAP upto 12 mmHg had minimal effect on CI (13% reduction) while at 6 mmHg IAP neither cardiac parameter nor any surgical condition is effected.

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

Positioning**Cardiovascular effects**

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

Respiratory effects

- Raised IAP shifts the diaphragm cephalad. Splinting results in closure of smaller airways, increased airway pressure, reduced thoracic /compliance and FRC. V/Q mismatch increases because of preferential ventilation of non dependent lungs. Effects are accentuated during trendelenburg position and positive pressure ventilation (PPV).

- In children FRC is low (10% of TLC) and quickly falls below the closing capacity producing small airway closure, atelectasis, intrapulmonary shunting and hypoxemia. These effects are aggravated in steep head down tilt because of the weight of the abdomen viscera on the diaphragm. Use of PEEP reverses this action and improves O2 saturation.
- At low IAP (10 - 15 mmHg) minimal changes are seen in increased PAP (<20%), EtCO2 (20-27%) and lung compliance. All changes revert back to normal within 10 minutes of insufflation.
- Post operative hypoxemia is due to diaphragmatic dysfunction rather than creation of pneumoperitoneum because changes seen during fundoplication exceed that during inguinal Herniorraphy. High IAP may result in leakage of gases to tissue spaces leading to pneumothorax, Pneumomediastinum, especially during Nissans fundoplication. A post operative chest radiograph is, therefore, advisable.
- Diagnostic laparoscopy in young children with facemask and spontaneous ventilation shows no significant CVS alteration or V/Q mismatch. Elevation of VT, EtCO2 and RR return to normal within 10 minutes of insufflation. It is advantageous in patients with irritable tracheobronchial tree but should be restricted to healthy children for brief procedures in supine position.

Neurologic effects

- Elevated IAP raises ICP and reduces the cerebral perfusion pressure (CPP). IAP of 25 mmHg increases the ICP from 7.6 to 21.4 mmHg (mean) and reduces the CPP from 82 to 62 mmHg. Laparoscopic procedures are avoided in patients with reduced intracranial compliance. Hypercapnia, increased SVR and head down tilt further aggravate the condition.

Endocrine effects

- Like any conventional surgery, laparoscopic procedures increase blood levels of insulin, cortisol, prolactin, epinephrine. Blood levels of lactate, glucose and interleukin-6 too are raised.

Anesthetic management Preoperative evaluation

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

Premedication

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

Monitoring

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

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

Induction of Anesthesia

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

Perioperative care

- Balanced anesthetic technique involves controlled ventilation, inhalational agents (Halothane, Isoflurane, sevoflurane, desflurane), short acting IV opioids, neuromuscular blockers (vecuronium, rocuronium, cisatracurium, mivacurium, or rocuronium). TIVA may be opted if myocardial depression is the concern. Use of N₂O is debatable because of possibility of venous gas embolism, distension of bowel, increased incidence of nausea /vomiting.
- Halothane should be discontinued once the trachea is intubated as it sensitizes myocardium to hypercapnia. Reduced hepatic blood flow following pneumoperitoneum can make the children prone to halothane hepatotoxicity.
- Controlled ventilation facilitates removal of exogenous CO₂ and minimizes the reduction in FRC during increased IAP, head down tilt and use of volatile agents.
- Minute ventilation may be increased by 20% by increasing RR. In children ventilated with uncuffed ETT, peri- ETT gas leakage reduces VT and should be compensated by increasing minute ventilation by about 30%.

Perioperative Care. Continuation:

- Insufflation should be complete to minimize hypo ventilation (due to delayed excretion of CO₂) referred shoulder pain (diaphragmatic irritation).
- Euthermia should be maintained with warmers, heated humidified gases to offset heat loss during surgery (high body surface: mass ratio, continuous insufflation of cold, dry gases).

Postoperative care

Monitor signs of hypoventilation. Maintain euthermia. Chest X-ray should be done following laparoscopic fundoplication, O₂ therapy is continued. The incidence of PONV can be decreased by H₂, blocked 5HT₃ antagonist; ondansetron (100µkg-1, maximum 4 mg), dexamethasone 150µkg-1, droperidol 25µkg-1 (maximum 0.625 mg).

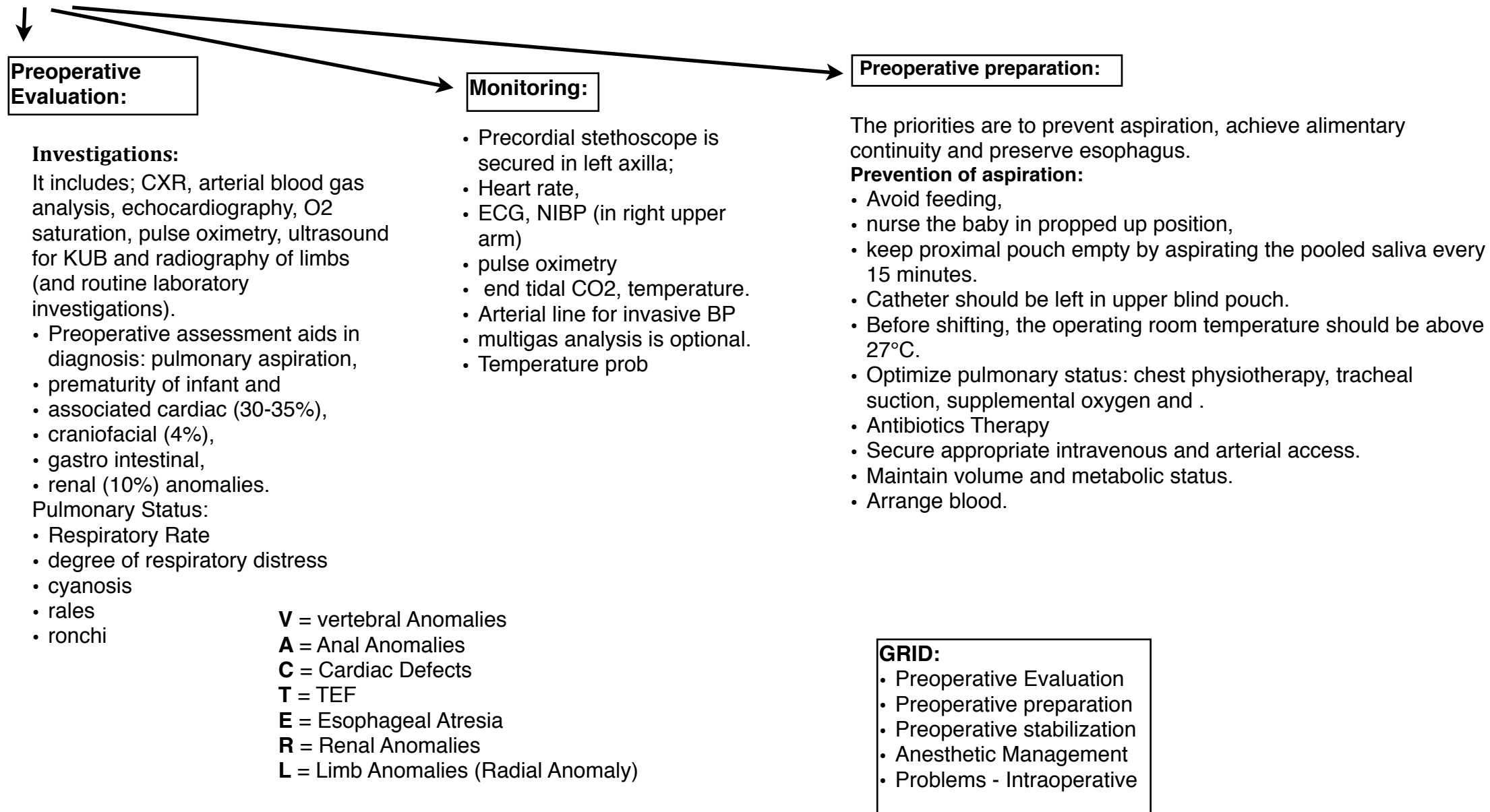
Pain management

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

Use of laryngeal mask airway (LMA)

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

7. Peri-operative problems and Anesthetic management of of two days old child scheduled to undergo TOF repair



Anesthetic Management:**• Problems:**

- Aspiration pneumonia,
- securing airway,
- oxygenation, gastric over distension,
- problems associated with coexisting anomalies.
- Management may be staged, if TOF co-exists with prematurity, pneumonitis or other congenital anomalies.
- Inadequate oxygenation and ventilation; leakage of gases through the fistula, ETT misplacement or of endobronchial intubation.
- ETT blockade; periodic ETT suctioning; in case of blocked ETT, replace with a new ETT over a tube exchanger.
- Ventilation/perfusion (V/Q) mismatch in anesthetized lateral decubitus position may be compounded by atelectasis secondary to retraction of the non-dependent lung, bronchial traction and ETT kinking. A high FiO₂ is desirable to keep the SpO₂ between 95-98%. The right lung is intermittently inflated to prevent hypoxemia.
- Vagal response to tracheal manipulation is avoided by prior atropine administration.
- Hypothermia produces peripheral vasoconstriction, non-shivering thermogenesis and increased O₂ consumption. It also affects pharmacokinetic and pharmacodynamics profile of anesthetics leading to over dosage, postoperative hypoventilation, apnea, coagulopathy and metabolic acidosis.

A major cause of morbidity in these patients, detailed Systemic examination is a must:

Goals: Preoperative stabilization

Establish IV route

PaO₂ of 50 to 70 mm of Hg,

Correct dehydration, hypovolemia

Hypoglycemia (10% dextrose 5mg/kg/min)

Warm to 37°C,

Correct acidosis if pH < 7.3

Anesthetic Management:

- Awake Intubation - allows appropriate positioning of ETT without IPPV
- Inhalation anesthetic agents with or without muscle relaxation may be used
- Ensure distal positioning of the tube with the bevel facing anteriorly and the posterior wall of the ETT occluding the fistula

Intraoperative problems:

- As the lungs are retraced - ventilation may become difficult
- Blood clot or secretion may block the ETT and require frequent suctioning
- Kinking of Trachea - during surgical resection may obstruct ventilation
- Positioning of the tube beyond the fistula opening.

8. A 2 days old child with congenital diaphragmatic hernia is scheduled for primary repair. Outline the pre-operative evaluation preparation & anesthetic consideration for this case.

Definition: CDH denotes herniation of abdominal viscera into the chest through the defect in the diaphragm.

Classic Triad of CDH: C = Cyanosis, D = Dyspnea, H = Dextrocardia

S/S: 1st hour of life or 24 hrs after birth = physical exertion, Scaphoid abdomen, Bulging chest, Right side heart, Decreased breath sounds, Bowel sounds in the chest.

Preoperative evaluation & preparation:

- Assess associated anomalies: PDA, Cantrell's pentalogy - Omphalocele, Diaphragmatic hernia & Intra cardiac defects
- Hypothermia increases O_2 consumption, optimal environmental temperature suggested is 30°C - 40°C .
- Arterial blood gases, complete blood count, serum electrolytes, blood sugar, cross matching.
- Correct metabolic acidosis (glucose, fluids) and respiratory acidosis (proper ventilation/sodium bicarbonate) prior to surgery. Sodium bicarbonate may be given empirically as diluted 0.5 mEqml^{-1} , ($2\text{-}4 \text{ mEqkg}^{-1}$) I.V. infusion at $< 1 \text{ mEqKg}^{-1}\text{min}^{-1}$.
- Venous access must be secured in the upper arm; neck veins reserved for ECMO. Central vein access is via umbilical or femoral veins.
- Use of vasodilators - tolazoline, prostacycline, dipyridamole, nitric oxide.
- Minimize sympathetic discharge by high dose opioids.
- Nasogastric suction for gastric decompression.
- Transport to OT with manual ventilation.

Assessment of severity of pulmonary hypoplasia:

- $\text{PAO}_2 - \text{PaO}_2 > 500 \text{ mmHg}$ breathing $100\% O_2$ - predicts non survival;
- $300\text{-}500 \text{ mmHg}$ - survival uncertain and values $< 400 \text{ mmHg}$; has better prognosis.
- Cardiac catheterization, echocardiography, colour doppler pulmonary angiography.
- Bohn's index prognosticates information as 'ventilatory index' (V.I.): product of mean airway pressure X respiratory rate..
- Neonate with $\text{PaCO}_2 < 40 \text{ mmHg}$ and $\text{VI} < 1000$ always survive, while $\text{PaCO}_2 > 50 \text{ mmHg}$, $\text{VI} < 1000$ or $\text{PaCO}_2 < 40 \text{ mmHg}$, $\text{VI} > 1000$ usually die.

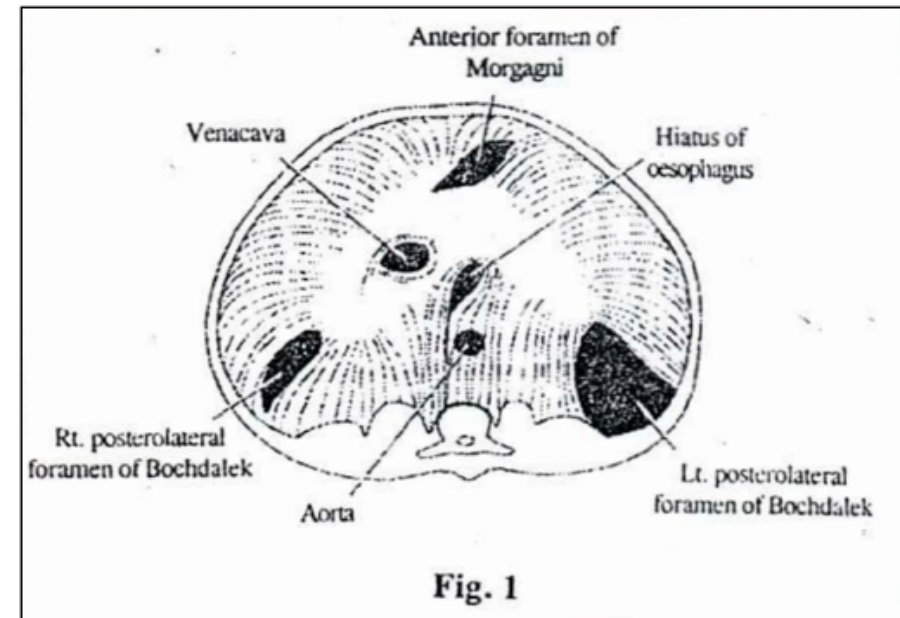
Chest X-Ray: Bowel gas shadow in the chest & mediastinal shift.

Pre-operative Care:

- Decompression of Stomach
- Improving Oxygen
- Correction of Acid base balance
- Decrease right to left shunt
- Increasing pulmonary perfusion.

In-spite of this if Condition decreases:

- Indicates Pulmonary HTN
- Right to left shunt
- Pneumothorax of contralateral lung
- If Hypoxia & Hypercarbia persists awake oral intubation and maintain High respiratory Rate



A 2 days old child with congenital diaphragmatic hernia is scheduled for primary repair. Out line the pre-operative evaluation preparation & anesthetic consideration for this case. Continuation:
Monitoring

- Precordial / esophageal stethoscope,
- Pulse oximeter both above / below the nipple for preductal and postductal SpO₂,
- Capnography,
- Inspiratory pressure,
- FiO₂,
- Acid base status,
- ECG,
- Invasive blood pressure in right radial artery for preductal PaO₂,
- CVP volume status and right ventricular performance,
- Thermoregulation - esophageal, rectal temperature.

Anesthetic consideration & Management:

- No IV line in lower limbs, because it may obstruct after reduction
- Infant in semi-sitting position is warmed with warming devices, humidified inspired gases and warm transfused fluids at 37°C. After pre-oxygenation, atropine 0.02 mg/kg-1 is administered I.V. Awake intubation is preferred.
- Alternatively infant is induced with halothane / Sevoflurane in O₂ and intubated while breathing spontaneously. Avoid mask ventilation, to obviate gastric distention and respiratory embarrassment.
- Gentle hand ventilation is preferred to avoid ipsilateral or contralateral pneumothorax. Anesthesia is maintained with volatile agent in 100% O₂ or with 0.5 mg/kg-1 ketamine with titrated dose 1-3 µg/kg-1 fentanyl. PPV is avoided till intubation.
- Patient in shock and severe hypoxemia is maintained on supplemental O₂, non depolarizing muscle relaxants (pancuronium / vecuronium) and analgesics. Inhalation agents, opioids (fentanyl) and muscles relaxants (pancuronium) are added in titrated doses.
- ***N₂O is avoided. It may produce intrathoracic gut distension, inability to close abdomen and increased intra abdominal pressure, leading to precipitous hypotension.***

Anesthetic consideration & Management: Continuation:

- Selection of appropriate FiO₂ depends upon severity of pulmonary dysfunction. To avoid hypoxia increasing R to L shunt of Desaturated blood, inhaled gases with higher O₂ content is suggested. PaO₂ should be optimally kept between 80-100 mmHg or arterial O₂ saturation at 95-98%. Prevent Hypothermia OT temperature to be more than 27°C.

Ventilation:

- Infant is ventilated with small tidal volume and low inflation pressure (< 20 mmHg) to prevent contralateral pneumothorax. Respiratory rate (60-120 min⁻¹) is adjusted to achieve hypocarbia (PaCO₂ 25-30 mmHg), lower pulmonary vasoconstriction and minimize R to L shunt through the ductus arteriosus.

Pulmonary hypertension and management

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

Treatment

- Continue ventilation in ICU
- Minimize endotracheal suction, to avoid transient hypoxemia.
- Hyperventilate the neonate with low V_T and high RR (60-120 min⁻¹) to maintain pH of 7.55-7.60 and induce pulmonary vasodilation.
- Restrict fluids to 2-4 ml/kg⁻¹hr⁻¹.
- Administer pharmacologic pulmonary vasodilators, chlorpromazine, prostaglandin E₁ and inhaled nitric oxide.
- Severe lung hypoplasia and refractory pulmonary hypertension with arterial O₂ saturation <50 mmHg at FiO₂ of 1.0 needs ECMO therapy immediately to avoid progressive pulmonary injury. ECMO is associated with 50-60% survival rate.
- Nitric oxide (NO) at 20-80 ppm a endothelial derived relaxing factor is selective pulmonary vasodilator with High frequency oscillatory ventilation (HFOV)

A 2 days old child with congenital diaphragmatic hernia is scheduled for primary repair. Out line the pre-operative evaluation preparation & anesthetic consideration for this case. Continuation:

Fluid replacement

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

Postoperative management

- Postoperative intubation and ventilation should be planned. The FiO₂ is adjusted to maintain PaO₂ over 150 mmHg till the infant is slowly weaned in 48-72 hrs to avoid honeymoon phenomenon, characterized by early smooth course followed by development of pulmonary vasoconstriction and lethal persistent pulmonary hypertension, hypercarbia and acidosis. Do not extubate till the child is fully awake, breathing spontaneously and rhythmically, open eyes and maintains SpO₂ of 100%.
- Mortality hypothermia, hypoxia.. acidosis, left to right shunt, shock and pneumothorax.

Pulmonary Vasodilators:

- PgE₁
- Dipyridamole
- Inhaled NO
- Chlorpromazine

9. A 2 year old child is scheduled for removal of organic foreign body in right main bronchus. What is the anesthetic management.

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

Four types of obstruction and their effects

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

Symptoms and signs

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

Problems

- N₂O should be withheld to limit further pulmonary inflation and potential rupture.
- Sharing the airway with endoscopist while maintaining alveolar ventilation and providing an unobstructed surgical access.
- Rigid ventilating bronchoscope equipped with an optical telescope and fiberoptic light source is essential.
- Coughing, gagging, laryngo-bronchospasm, hypertension, arrhythmias and secretions must be controlled.
- Anticholinergics reduce secretions and attenuate vagal mediated bradycardia and reflex bronchoconstriction.
- Local anesthetics (lidocaine IV/spray) diminish airway reflexes secondary to endoscopic manipulations.

Problems Continuation:

- Opioid analgesics (fentanyl 1 μgkg^{-1}) suppress airway reflexes and administered only after the airway is secured.
- Sevoflurane with 100% oxygen provides smooth induction and can be replaced by isoflurane once deeper plane of anesthesia is achieved.
- Propofol with good reflex suppression has rapid action, quick recovery.
- N₂O should be withheld to limit further pulmonary inflation and potential rupture.
- Sharing the airway with endoscopist while maintaining alveolar ventilation and providing an unobstructed surgical access.
- Rigid ventilating bronchoscope equipped with an optical telescope and fiberoptic light source is essential

Goals

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

Preoperative considerations

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

Monitoring

- Precordial stethoscope -inspection of chest movements.
- SpO_2 ,
- ETCO_2
- Neuromuscular transmission with TOF nerve stimulator.
- One overhead spotlight towards anesthetic machine, second over the child's feet to assess saturation and perfusion.

Postoperative care

- Bronchoscopy is repeated to evaluate residual FB fragments and the impact site for trauma, bleeding or granulations. Child may be intubated for tracheobronchial toilet and ventilation until adequate spontaneous breathing is established.
- Minimize postoperative stridor and distress by Dexamethasone (0.1-0.25 mgkg^{-1}) for laryngeal, oedema, humidified oxygen, nebulized racemic epinephrine (2.25% solution given 1:6 to 1:10 dilution) under ECG monitoring, β -agonist bronchodilators such as albuterol for postoperative wheezing.
- 24-48 hrs intubation may be necessary till oedema subsides.

Acute emergency

- GA is always required because of fighting irritable, child removal of distally lodged invisible FB and if prolonged bronchoscopic procedure is contemplated. Based on feeding history, anesthesia can be induced with inhalational or intravenous technique.
- Spontaneous technique is safer than apnoeic technique with complete neuromuscular blockade for fear of distal dislodgement. Sevoflurane is preferred over halothane because of smooth induction, its antitussive and cardio-respiratory stability properties.
- Anesthesia is induced with sevoflurane 3-5% in 100% oxygen and reinforced with topical lidocaine 1-4 mgkg^{-1} prevent laryngospasm.
- Anesthesia is then maintained with 100% oxygen in halothane or isoflurane as these are more soluble and eliminated slowly, giving more time for airway manipulation, avoiding lighter plane and associated hemodynamic reflexes. Deeper plane of anesthesia with spontaneous respiration is required during attempts to extract FB.
- During instrumentation or sanction of FB if the child coughs, small increments of propofol or fentanyl are administered.
- Propofol based IV anesthesia supplemented with topical spray of lidocaine can alternatively maintain a steady level of anesthesia, independent of ventilation or O.T. pollution.

10. Draw a labelled diagram to illustrate Fetal circulation. What are the circulatory changes that occur at birth.

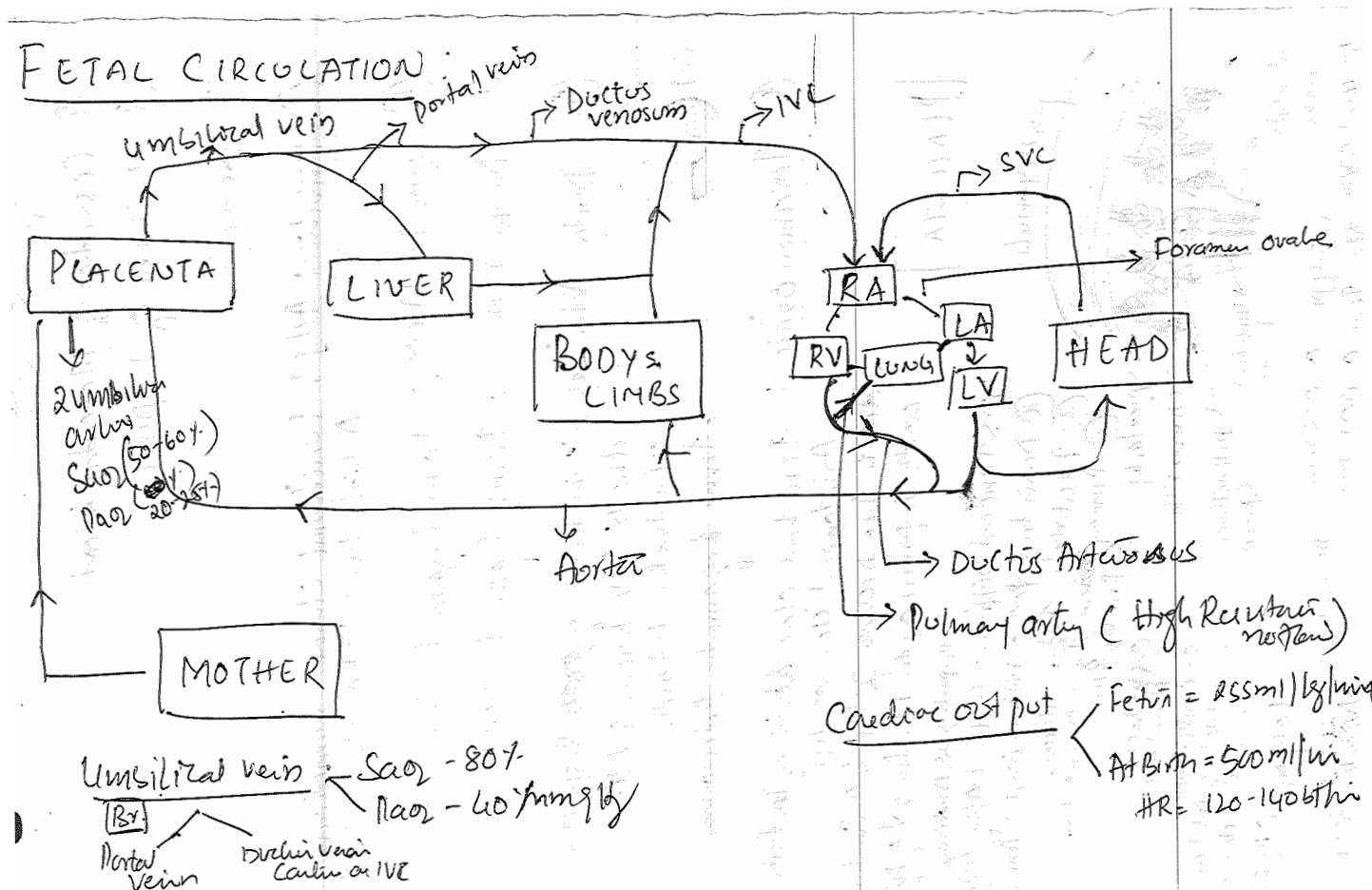
The placenta which receives nearly one half of the fetal cardiac output, is responsible for respiratory gas exchange. Lung receive little blood flow, pulmonary & systemic circulations are parallel instead of in series.

This arrangement is possible by two cardiac shunts:

- Foramen Ovale
- Ductus arteriosus

4 Shunts:

1. Placenta
2. Ductus Venosum
3. Foramen Ovale
4. Ductus Arteriosus



After birth & After Clamping:

1. Increases in SVR
2. Decrease in PVR
3. Increase Pulmonary blood flow
4. Decrease in Pulmonary artery pressures.

Key Points:

- The fetal circulation supplies the fetal tissues with oxygen and nutrients from the placenta. It bypasses the fetal lungs.
- The presence of fetal haemoglobin (which has an oxygen dissociation curve shifted to the left compared with adult haemoglobin) ensures that oxygen delivery is maintained despite low oxygen partial pressures.
- At birth, the circulation of the fetal blood through the placenta ceases, acute changes in the pulmonary and systemic vascular resistance occur and the lungs begin to function.
- Subsequently the foramen ovale, ductus arteriosus and umbilical vessels close or transform into the corresponding ligaments.
- Neonates may revert to a persistent fetal circulation (PFC) with decreased pulmonary blood flow and a right to left shunt in response to certain stimuli such as hypoxia, hypercarbia, acidosis and cold.

Circulation in the fetus

1. Deoxygenated fetal blood is conducted to the placenta via the two umbilical arteries. The umbilical arteries arise from the internal iliac arteries.
2. Gas exchange occurs in placenta.
3. Oxygenated blood from the placenta passes through the single umbilical vein and enters the inferior vena cava (IVC).
4. About 50% of the blood in the IVC passes through the liver and the rest bypasses the liver via the ductus venosus. The IVC also drains blood returning from the lower trunk and extremities.
5. On reaching the heart, blood is effectively divided into two streams by the edge of the interatrial septum (crista dividens) (1) a larger stream is shunted to the left atrium through the foramen ovale (lying between IVC and left atrium) (2) the other stream passes into right atrium where it is joined by blood from SVC which is blood returning from the myocardium and upper parts of body. This stream therefore has a lower partial pressure of oxygen.
6. Because of the large pulmonary vascular resistance and the presence of the ductus arteriosus most of the right ventricular output passes into the aorta at a point distal to the origin of the arteries to the head and upper extremities. The diameter of the ductus arteriosus is similar to the descending aorta. The patency of the ductus arteriosus is maintained by the low oxygen tension and the vasodilating effects of prostaglandin E₂.
7. Blood flowing through the foramen ovale and into left atrium passes into the left ventricle where it is ejected into the ascending aorta. This relatively oxygen rich blood passes predominantly to the head and upper extremities.

Physiological transition of fetus at birth:

1. Catecholamine in the neonate during labor may augment release of surfactant from type II pneumocytes.
2. At term fetal lungs are developed but contain 90 ml of plasma ultra filtrate. During normal delivery the fluid is squeezed from the lung by the force of pelvic muscles & vagina - vaginal squeeze.
3. Respiratory efforts normally initiated within 30 sec, becomes sustained within 90 sec.

Closure:

- Umbilical artery: Functional closure immediate. Actual closure takes about 2 - 3 months
- Umbilical Vein: Forms ligamentum teres
- Ductus Venosum: Forms Ligamentum venosum
- Ductus Arteriosus: Within a few hours of respiration due to increase oxygen tension & decrease PG's, Obliterates 1 - 3 months forms ligamentum arteriosum
- Foramen Ovale: Functional closure soon after birth. Anatomical closure at 1 year, due to increased pressure of the LA combined with decreased pressure on the right atrium & decrease in PVR & SVR.

Difference between the Adult & Fetal Circulation:

Criteria	Adult	Fetal
Artery	Carries oxygenated blood away from the heart	Carries Non oxygenated blood away from the heart
Vein	Carries Non oxygenated blood towards the heart	Carries oxygenated blood to the heart
Exchange of gas	Takes place in Lungs	In Placenta.
Pressure	Increase pressure on the left side of the heart	Increased pressure on the right side of the heart
SVR PVR PBF PAP	↑ SVR ↓ PVR ↑ PBF ↓ PAP	↓ SVR ↑ PVR ↓ or NO PBF ↑ PAP

Closure	Functional Closure	Anatomical Closure
Ductus Arteriosus (PDA)	10 - 15 Hr	2 - 3 weeks Ligamentum arteriosus
Foramen Ovale	At Birth	6 wks to maximum 1 year
Ductus Venosum	3 - 7 days after birth	2 - 3 months of age Ligamentum Venosum
Umbilical Artery	At Birth	2 - 3 months of age
Umbilical Vein	Forms ligamentum teres	

- PDA is an abnormal communication between PA and the descending aorta after birth.
- It is present in fetal life in all, but closes both functionally and anatomically after birth.
- Definition: PDA is present when the ductus arteriosus fails to close spontaneously after birth.
- Obliteration takes 1 to 3 months and becomes ligamentum arteriosum.
- Associated anomaly: CDH
- Its a Acyanotic heart disease with left to right shunt.

Embryology:

- Connects between left pulmonary artery & descending aorta

Pathophysiology:

- It arises just distal to subclavian artery
- PDA is present in fetus because of under developed lungs, - Pulmonary artery to aorta & placenta
- Failure to close in pre term

Signs & Symptoms:

- Dyspnea
- Collapsing pulse
- Apex beat down and out ward
- Continues thrill
- Continues Machinery murmur
- LVH on ECG
- Increase risk of infective endocarditis
- MDM & S3
- Pulmonary HTN
- CCF end stage

Diagnosis:

- Echocardiogram with doppler studies
- Chest x ray - prominent pulmonary vasculature
- Cardiac Catheterization

Management:

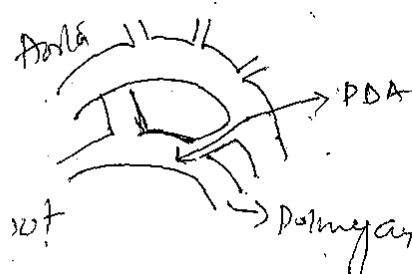
- Medical
 - For preterm delivery before 28 weeks of gestation
 - Cyclo-oxygenase inhibitor (Cox1 & Cox 2) - inhibits Prostaglandin synthesis
 - Indomethacin - Non selective Cox inhibitor
 - Ibuprofen -
- Surgical - Once pulmonary hypertension develops surgical or percutaneous closure is contraindicated.

Anesthetic Management:

- Infective endocarditis prophylaxis:
 - Ampillicin - 50 mg/kg 30 min before procedure and then 25 mg/kg 6 hrs after initial dose or Vancomycin - 20 mg/kg IV one hour before procedure
- Premedication:
 - Midazolam nasal - 0.2 to 0.3 mg (max 5 mg) 10 min before
 - Oral - 0.5 to 0.75 mg/kg 20 to 30 min before
 - IM - 0.08 mg/kg 10 min before
 - Rectal - 0.3 to 1mg/kg 20 to 30 min before

Monitoring:

- Pulse oximeter
- NIBP
- ECG
- EtCO₂
- CVP
- TEE - Risk of embolization arising from surgical site
- Temperature probe
- Urine output



Anesthetic Management Continuation:

Induction:

- IV induction is relatively slower due to additional dilution by recirculating blood volume, as increased doses of induction agents may be required. Special attention should be paid to risk of over dose.
- Left to right shunt - IV induction is slow & Inhalation induction is faster
- Severely ill patients the administration of fentanyl and a non-depolarizing muscle relaxants provides adequate analgesia and surgical operating conditions.
- Ventilation is maintained with a mixture of air and oxygen.
- This combination is well tolerated even by critically ill infants with congestive heart failure.
- We use on induction dose of fentanyl of 5-10 micrograms/kg and 30-50 micrograms/kg in total for maintenance.
- In haemodynamically stable children, this technique usually allows extubation at the end of procedure.
- Thiopentone 2 - 3 mg/kg, isoflurane and midazolam are useful drugs that can be use to supplement the anaesthetic.
- Fluid boluses of 5 to 10 ml/kg to improve tolerance to induction agents.
- Ligation is done through left thoracotomy approach.
- Blood loss should be anticipated

Maintenance:

- O₂ + N₂O (to be avoided in Pulmonary HTN & increases PVR) + NDMR + Isoflurane + IPPV
- PaCO₂ is maintained between 30 to 40 mm of Hg
- Reversal: Neostigmine (0.05 mg/kg) + Glycopyrolate (0.01 mg/Kg)

During PDA ligation the systemic pressure needs to be decreased with the help of:

- NTG/SNP
- Esmolol
- Increasing isoflurane/sevoflurane/halothane

Risk of surgical closure:

- Intracrainal hemorrhage
- infections
- Recurrent laryngeal nerve palsy.

Post operative pain relief:

- NSAID
- Intermittent opioids

	Preload	HR	Contractility	SVR	PVR
PDA	↑	N	N	↓	↑

12. Fasting guidelines for neonates & infants

Age	Milk & Solids	Liquid
< 6 months	4 hours	2 hours
6 - 36 months	6 hours	3 hours
> 36 months	8 hours	3 hours

Oral Medications: Allowed

- Adult: 150 ml 1 hr before.
- Children: 75 ml 1 hr before.

Fasting guidelines in neonates

- Breast milk may be given until 4 h preoperatively.
- Dextrose solutions may be given until 2 h preoperatively.
- Formula milk feeds may be given until 6 h preoperatively. These milks contain cow protein and are more slowly digested than breast milk.
- Babies having continuous feed via jejunal catheters should have these stopped 4 h preoperatively.

Clear fluids may be taken up to 2 h before the induction of anesthesia.

The mandatory fasting period after solid foods should be 6 h.

Breast milk can be taken up to 4 h before anesthesia.

Oral premedication can be taken with 150 mL of water in adults up to 1 h before anesthesia.

Oral premedication can be taken with 75 mL of water in children up to 1 h before anesthesia.

Chewing gum, candies, and tobacco should be avoided for the last 2 h before anesthesia.

Elective cesarean section and children are included.

Clear fluids are defined as non-particulate fluids without fat. Non-human milk is defined as solid.

The guidelines apply to patients without known delay of gastric emptying.

Infant Airway	Adult Airway
• Infant larynx is more cephalad and anterior, At birth, the Rima glottidis is opposite interspace of C3 & C4, with lower border of cricoid cartilage opposite the lower border of C4. • At age of 13 adult position is achieved	• At the age of 13, the lower cricoid cartilage is opposite the C7 and rima glottidis is opposite the C4-C5 interspace.
• Infantile epiglottis is short & stiffer, which is U or V shaped	• Adult epiglottis is flatter & flexible.
• Hyoid bone in infant is attached to thyroid cartilage, causing base of the tongue to depress the epiglottis leading to increased protrusion into the pharyngeal cavity.	• Hyoid & thyroid cartilage separate, & epiglottis with anterior pharyngeal wall changes from 45° to become much closer to the base of the tongue in adult.
• Infant tongue is relatively larger and bulky	
• To visualize larynx in an infant or child the laryngoscope blade may have to be passed perpendicularly with the head in neutral position. Lifting an infants upper back & shoulder area is helpful in obtaining proper neck extension.	• Vocal cord are minimally concave
• The epiglottis should be visualized to avoid folding it down over the larynx with tip of the blade. Use of excessive force is unnecessary & leads to trauma & airway injury.	
• Vocal cords are composed of vocal ligament anteriorly & the cartilaginous vocal process of the arytenoid posteriorly, with age the increase in vocal cord length increases in the ligamentous portion, which increases from 50% to 80% of the entire length. As the cartilaginous portion is angled down the trachea & inward, the infantile cords are concave.	

Infant Airway	Adult Airway
• With growth there is forward movement of the thyroid cartilage. The concavity of the cords in the infant may impede passage of a curved ETT. Pushing the tube slightly posteriorly with the blade will aid advancement. • With vigorous laryngoscopic lifting, occasionally the trachea is angled forward so much that tube advancement is prevented even when the tip of the ETT may be beyond the cords. • Slight relaxation of the blade allows the tube to be advanced successfully.	
• Narrowest portion of the infant larynx is at the level of cricoid cartilage. (subglottic area)	• Rima glottidis (glottic area) is the narrowest point in the upper respiratory tract.
• A tube size that permits a slight leak when positive pressure is applied will usually be preferred.	• A normal size endotracheal tube is preferred.

Physiological:

- Heart rate dependent cardiac output
- Faster heart rate
- Lower blood pressure
- Faster respiratory rate
- Lower lung compliance
- Greater chest wall compliance
- Lower functional residual capacity
- Higher ratio of body surface area to body weight
- Higher total body water content
- Predominate parasympathetic activity more developed.

Anatomic:

- Non compliant left ventricle
- Residual fetal circulation
- Difficult venous & arterial cannulation
- Large head and tongue
- Narrow nasal passages
- Anterior & cephalad larynx
- Long Epiglottis
- Short trachea & neck
- Prominent adenoids & tonsils
- Weak intercostal & diaphragmatic muscles

Pharmacological:

- Immature Hepatic biotransformation
- Decrease protein binding
- rapid raise in FA/FI
- Rapid induction & recovery
- Increased MAC
- Larger volume of distribution for water soluble drugs
- Immature neuromuscular junction

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

Characteristics:

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

Upper airway:

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

GIT: chronic duodenal atresia – 300 times more frequency

CNS: microcephaly, small brain mass, mental retardation

Eye: oblique palpebral fissures, brush field spots, cataracts and strabismus → surgical correction.

Ear: otitis media and hearing loss common → freq. ear examination and myringotomies.

Teeth: dental caries → surgical repair.

Hematologic: polycythemia - ↑ risk for transitional circulation, hypotonia

Musculoskeletal: (20% - care during manipulation during laryngoscopy) asymptomatic dislocation of atlas on axis → screening for atlanto axial instability lateral radiographs in flexed, extended, and neutral position.

Distance ant. arch of atlas and adjacent odontoid process > 5mm – diagnostic.

Anesthetic management / Consideration:

- Care should be taken during manipulation during laryngoscopy.
- Preop neurologic evaluation
- Premedication
- Anticholinergic drugs (atropine / glycopyrrolate) to ↓ airway secretions.
- Sedatives – response unpredictable oral midazolam commonly used.
- IM ketamine to prepare stubborn children
- Obesity and folds of skin at wrists and ankles → venous cannulation technically difficult.
- Patency of upper airway difficult to maintain (short neck, small mouth, narrow nasopharynx, large tongue)/
- Tracheal intubation usually not difficult – atlanto axial instability → extreme movement of the head and neck during laryngoscopy to be avoided → spinal cord compression.
- Size of ETT required is smaller – subglottic stenosis
- Possibility of associated CHD must always be considered
- Avoid air bubbles in IV line because of possible R → L shunts (paradoxical air embolus).
- Respiratory complications such as post op stridor and apnea and post op pulmonary complications.

16. Fetal Hemoglobin.

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

HbF: P50 – 18 to 20 mmHg

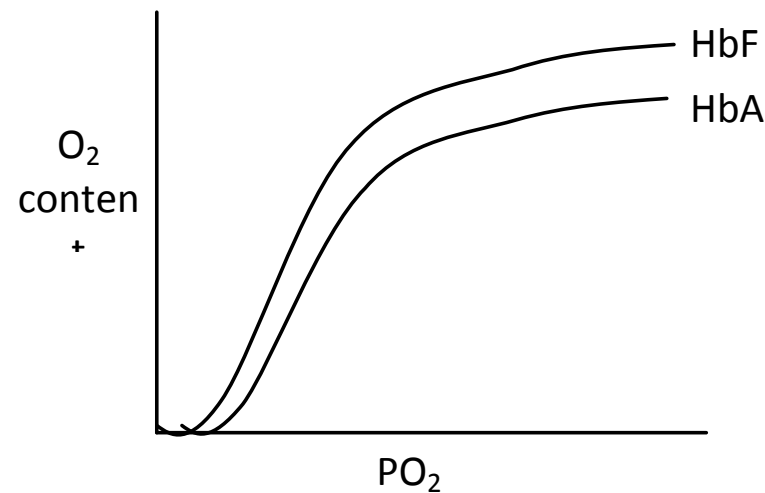
- $\uparrow$ O₂ affinity
- $\downarrow$ Concentration of 2,3-DPG
- $\downarrow$ Affinity to 2,3-DPG

Adults

HbA $\uparrow$
 P50 27mmHg
 $\uparrow$ affinity to 2,3-DPG
 2 α and 2 β

The ODC is shifted to left

- This results in $\uparrow$ O₂ affinity and $\uparrow$ O₂ transport necessary to extract O₂ from the adult HbA from the placental circulation but the disadvantage is poor O₂ delivery to fetal tissue.



The ODC is shifted to left

17. Fluid Management in Infants.

- Fluid management in infants is the most important & critical as smaller volumes involved.
- Normal daily water consumption in infant is 10% - 15% of body weight.

Fluid calculation is based on the **Holiday Segar Formula:**

It is based on energy requirement.

- 1- 10 kg infant need about 100 cal/kg/24 hr
- 10 - 20 kg infant need about 1000 + 50 cal/kg/24 hr
- > 20 kg infant need about 1500 cal + 20 cal/kg/24 hr

Approximately 1ml of water is needed for each calorie expended:

- 1 - 10 kg infant requires 100ml/24hr/kg
- 10 - 20 kg infant requires 1000 ml + 50 ml/24hr/kg
- > 20 kg infant requires 1500 + 20 ml/24hr/kg

Estimated Fluid requirements - 4:2:1 Formula:

Body Weight	Fluid
First 10 kg	4ml/kg/hr
Second 10 kg	2 ml/kg/hr
Subsequent (Remaining)	1 ml/kg/hr

- The Fluid of choice is controversial: Hartman's Solution, 1/2 Normal saline
- Neonates requires 3-5mg/kg/min of glucose infusion to maintain euglycemia (40 - 120 mg/dl)
- Premature requires 5 - 6 mg/kg/min of glucose infusion.

- After using 4:2:1 Formula the amount obtained is **Maintenance fluid**
- To calculate deficit the Maintenance fluid is multiplied by number of hours of fasting
 - **Deficit = Maintenance fluid X number of hours of fasting**
- The deficit is administered in aliquots of 50% in the first hour, 25% in 2nd 3rd hour respectively.

Replacement requirements:

- It can be divided into blood loss & third space loss.

Blood Loss:

- Blood loss is typically replaced by non-glucose containing crystalloids (e.g: 3 ml of HRL with each ml of blood)
- Because their small intravascular volume, neonates & infants are at increased risk for electrolyte disturbance (Hyperglycemia, Hyperkalemia & Hypocalcemia)
- Platelets One unit per 10kg & FFP 10-15ml/kg should be given when blood loss exceeds 1-2 blood volumes.
- Cryoprecipitate is 1U/10 kg.

Third Space Loss:

Degree of Blood loss or Tissue Trauma	Additional fluid requirements
Minimal (e.g. herniorrhaphy)	0 - 2 ml/kg
Moderate (e.g. Appendicitis)	2 - 4 ml/kg
Severe (e.g. Bowel resection)	4 - 8 ml/kg

- Magnitude of perioperative third space loss is proportional to the amount of tissue manipulation.
 - E.g. Burns, trauma, infected tissues, etc.

- Symptom complex rather than a specific disease.
- Comprises a group of non progressive, but often changing, motor impairment syndromes secondary to lesions or anomalies of brain that arise during early stages of development.

Cerebral palsy is classified according to:

Depending upon extremity involved :

- Monoplegia, hemiplegia, diplegia, quadriplegia

Depending on characteristics of neurologic dysfunction:

- Spastic, hypotonic, dystonic, athetotic

Incidence and risk factors:

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

During labor and delivery:

- Premature separation of the placenta.

During the early postnatal period:

- Newborn encephalopathy
- Rate of cerebral palsy is 25 to 31 times higher among infants who weigh less than 1500g at birth than among full size new born.

S/s: cerebral palsy

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

Management of anesthesia:

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

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

Signs and symptoms:

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

Associated congenital anomalies:

Neurologic: Arnold – Chiari II malformation

(caudal displacement of cerebellar vermis than Foramen magnum

- Caudal displacement medulla oblongata and cervical spine)
- Kinking of medulla
- Obliteration of cisterna magna
- Brain – stem dysfunction.
- S/s – stridor, apnea and bradycardia, aspiration pneumonia, vocal cord paralysis, in-coordination, spasticity, Hydrocephalus.
- Orthopedic – club foot – dislocation of hips – scoliosis
- Urogenital: extrophy of bladder, prolapsed uterus, severe dilatation of upper urinary tract (urinary diversion procedure)
- Recurrent UTI → septicemia gram negative
- Congenital cardiac defects

Latex allergy:

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

Preoperative care:**Focus on**

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

Anesthetic management Meningomyelocele:

- Meningomyelocele operated with 72 hrs of life – to prevent development of ventriculitis and prevent neurologic deficits.
- Closure performed under local / general anesthesia.
- Positioning for induction – lateral decubitus position – intubation is challenging. Supine position – with the defect – resting in a “dough nut” support to minimize trauma.
- Awake tracheal intubation.
- IV line present – I.V induction + succinylcholine to facilitate intubation without risking hyperkalemia.
- Difficult IV access – inhalation induction
- Surgery in prone position
- Maintenance with inhaled / opioids anesthetics. Avoid muscle relaxants as identification of neural tissue may require nerve stimulation.
- Surgical closure tight enough to prevent CSF leakage – confirmed by increasing the pressure in sac with positive airway pressure.
- Monitor temperature – prolonged surgery – tendency for hypothermia. Post operatively prone position – respiratory complications (stridor, apnea and bradycardia, cyanosis, respiratory arrest → in brainstem abnormality)
- High index of suspicion for raised ICT → shunt insertion.
- Meningocele: protrusion of meninges, contains only CSF
- Meningomyelocele: protrusion of meninges with spinal cord / cauda equina and they may be adherent to the post aspect of sac.
- Encephalocele: protrusion of the brain
- Meningoencephalocele: protrusion of meninges as well as brain. Common sites – root of nose, occipital region, anterior fontanells

- Syringomyelocele: central cord of spinal cord becomes dilated and the cord lies within the sac and becomes adherent to the post part of sac.
- Myelocele: central portion of the spinal cord opens out on the surface and discharges CSF continuously. Commonest type of spina bifida but incompatible with life. Associated with paralysis of foot and incontinence of feces and urine.

Precautions:

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

20. Hydrocephalus.

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

Classification:

Non-obstructive / communicating hydrocephalus

- I. Due to overproduction or abnormal absorption of CSF
- II. No obstruction to flow of CSF

Obstructive hydrocephalus

- I. Obstruction to the flow of CSF and its absorption in SAS
- II. Due to congenital, neoplastic, post traumatic and post inflammatory lesions.

Congenital causes of obstructive hydrocephalus

- a. Arnold – chiari malformations – basilar subarachnoid pathways under development.
- b. Aqueductal stenosis – between 3rd and 4th ventricle.
- c. Dandy-Walker syndrome – occlusion at the outlet of 4th ventricle by a congenital membrane.

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

Signs and symptoms:

Depends on age of child and rapidity with which ICP↑.

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

Diagnostic tests:

- Serial head circumference measurements
- Skull radiographs – CT scan of head

Treatment:

Depends on mechanisms responsible for hydrocephalus

- Operative excision of lesions responsible for obstructing flow of CSF performed if feasible.
- Shunting procedures – necessary if obstruction cannot be relieved surgically – shunt system employs a one-way valve, that directs flow of CSF away from ventricles.
 - a. Ventriculosternostomy (Torkilden's procedure)
 - b. Ventriculoatrial shunts
 - c. Ventriculoperitoneal shunts (V-P shunts)
- Less common → ventriculocholecystostomy and ventriculospinal shunt
- Ventriculoatrial shunts for obstructive / non-obstructive hydrocephalus
- Distal end of catheter placed in right atrium while monitoring the changes in venous pressure wave patterns while advancing the catheter in right atrium through SVC.
- Complications: thrombosis of IJV /SVC, septicemia, meningitis, pleural effusion, pulmonary embolism and pulmonary hypertension.
- Growth of child displaces the cardiac end of catheter into SVC revision of shunt / V-P shunt

Anesthetic management of Hydrocephalus:

- Operative procedures – placement, revision or removal CSF shunt system.
- Hydrocephalic infants and children with (NL) intracranial pressures.
 - a. Induction with short acting induction drugs + muscle relaxants → tracheal intubation
 - b. Maintenance with volatile anesthetics / opioids + N₂O + MR
- Hydrocephalus with co-existing intracranial hypertension – precautions during anesthesia.
- Shunt is to be inserted before craniotomy to excise an intracranial tumor.
- Potential for further increases in ICP in association with use of scoline.
- Nevertheless, scoline induced ↑ in ICP does not always occur and its use can be justified if there is need for rapid onset of muscle paralysis.
- Sudden hypotension sometimes occurs if tensely distended cerebral ventricles are decompressed.
- Venous air embolism or and ↑ blood loss can occur when large neck veins are opened to place an atrial catheter.
- Postoperatively – slightly head up position – to permit free drainage of CSF.
- During surgery in children with V-P shunts – excessive pressure on the skin of scalp overlying the shunt avoided by rotating the head to the opposite side of shunt.
- Awake tracheal intubation, crying, struggling and straining - ↑ ICP.

- **Major concerns** – protection of airway and control of ICP. Rapid sequence induction pretreatment with atracurium 0.05mg/kg I.V. thio – 3-4mg/kg / propofol 2mg/kg IV + scoline 2mg/kg + cricoid pressure.
- Hyperventilation + Barbiturates – rapidly controls ICP.
- Maintenance – volatile anesthetics, N₂O and opioids
- Post op - trachea should remain intubated and receive PEEP if they are experiencing period of apnea / bradycardia before surgery because of intracranial abnormalities. Hydrocephalus.

21. Pyloric Stenosis.

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

Signs and symptoms:

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

Anesthetic management:

- Aspiration of gastric contents – definitive risk, further ↑ after radiographic examination of upper GIT with barium.
- Large orogastric tube is passed and stomach contents aspirated as much as possible.
- Awake tracheal intubation / after induction of anesthesia.
- IV line already obtained – rapid seq. tracheal intubation (explain with cricoid pressure).
- IV line not in place – stomach emptying → inhalational induction with N₂O + Halothane / Sevoflurane. N₂O discontinued after loss of lid reflex → if vein becomes evident IV line placed – rapid seq. induction with cricoid pressure. If IV line still not placed – 2 options exist

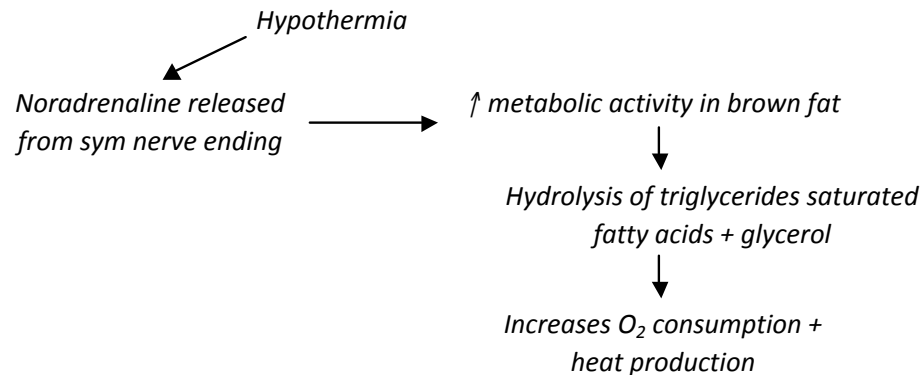
1. Deepen inhalational anesthesia with halothane / Sevoflurane, insert laryngoscope, topically anesthetize the vocal cords with 4mg/kg lidocaine, and intubate the trachea.
2. Administer 4mg/kg scoline IM before intubation
3. Secure IV line before beginning surgery – if needed cut down
 - Maintenance: volatile anesthetic + opioids ± N₂O + skeletal muscle relaxants + mechanical ventilation.
 - Surgeons need muscle relaxation twice
1. When they deliver the pylorus at the beginning of surgery
2. When they replace the pylorus into the abdomen at the end of surgery, shortly before closing the peritoneum → short acting drugs like succinylcholine / mivacurium are considerations.
- Caudal anesthetic (1.25ml/kg 0.25% bupivacaine with epinephrine) after induction of GA with tracheal intubation.
 - Provides intraoperative relaxation
 - Reduces anesthetic requirement
 - Provides postoperative analgesia

Postoperative management:

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

- In adults shivering generates heat, and when there is hypothermia. But in infants shivering mechanism is not well developed, hence rely on "Non Shivering Thermogenesis".
- NST is brought about by brown adipose tissue (BROWN FAT) in infants.
- Very-LBW babies have very little brown fat.
 - Brown fat constitutes 25% of body weight
 - Location of brown fat: scapular, mediastinum, around kidneys and adrenal glands.
 - Speciality / contents of brown fat
 - These contain ↑ no of mitochondria and fat vacuoles and rich blood supply and autonomic nerve supply.

Mechanism:



- **Importance:**
- NST → increases O₂ and glucose utilization → acidosis
- NST is lost ↓ anesthesia
- Brown fat deposits decline during the first few wks of extra uterine life.

Maintenance of Body Temperature / Prevention of Heat Loss:

- Conduction Loss:** Maintaining/warming OT Temperature, Use of warm mattress.
- Convection Loss:** Keep infant in incubator, Cover infant with cloth role/Warm blanket.
- Radiation Loss:** Use of incubator during transport.
- Evaporation loss:** Humidification of inspired gases, warming IV Fluids, Warm skin disinfectant, using warming blanket /Bear Hugger.

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

Signs and symptoms:

Over distended lobe – compression atelectasis of (NL) lung parenchyma – mediastinal shift – impaired venous return – arterial hypoxemia and hypotension.

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

Anesthetic management:**Surgical lobectomy / Pneumonectomy:**

- Cardiovascular and pulmonary changes during mechanical ventilation.
- PPV during induction – rapid expansion of emphysematous lobes (gas enters but cannot leave)
- - Sudden mediastinal shifts
- - Cardiac arrest
- Spontaneous breathing with minimal positive airway pressure recommended.
- GA supplemented with local anesthesia until chest is opened and emphysematous lobes are delivered.
- Thereafter – infants paralyzed and lungs mechanically ventilated.
- Nitrous oxide not used as its diffusion into diseased lobes ↑ distention.
- Severely decompensated infants may require emergency needle aspiration or thoracotomy for decompression of the affected lobe or lobes.

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

Incidence and risk factors:

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

During labor and delivery:

- Premature separation of the placenta.

During the early postnatal period:

- Newborn encephalopathy
- Rate of cerebral palsy is 25 to 31 times higher among infants who weigh less than 1500g at birth than among full size new born.

Signs and symptoms:

- Most common manifestation of cerebral palsy is skeletal muscle spasticity.
- Extrapyramidal cerebral palsy is associated with Choreoathetosis and dystonia and cerebellar ataxia is characteristics of atonic cerebral palsy.
- Varying degrees of mental retardation and speech defects can accompany cerebral palsy.
- Seizures disorders co-exist in approximately 1/3rd of individuals affected with cerebral palsy.
- Children with cerebral palsy may have varying degree of spasticity of different skeletal muscle groups, resulting in contractures and fixed deformities of several joints of both upper and lower extremities.
- Stereotactic surgery may be performed in attempts to decrease skeletal muscle rigidity, spasticity and dyskinesia.
- Gastroesophageal reflux is common in children with CNS disorders and antireflux operations may be recommended.
- Children with cerebral palsy frequently receive antiseizure medications and dantrolene for relief of skeletal muscle spasticity.
- Phenytoin → gingival hyperplasia and megaloblastic anemia.
- Phenobarbitone → hepatic microsomal enzymes – alters liver metabolism.

Management of anesthesia:

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

S/s: cerebral palsy

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

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

Sign	0	1	2
1. Heart rate (beats / min)	Absent	< 100	> 100
2. Resp. effort	Absent	Slow irregular	Good, crying
3. Reflex irritability	No response	Grimace	Crying
4. Muscle tone	Limp (flaccid)	Flexion of extremities	Active
5. Colour (Appearance)	Pale cyanotic	Body pink extremities cyanotic	Pink

1) Score 8 to 10 (Normal):

- Achieved by > 90% of all neonates.
- Nothing is required except nasal / oral suctioning drying of skin, maintenance of body temperature.
- Re-evaluate at 5 mins of age.

2) Score 5 to 7: Mild

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

3) Score 3 to 4: Moderate

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

4) Score 0 to 2: Severe

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

First Steps for every newborn

1. Proper antenatal maternal history to be taken about any infections, drug intake, past history prolonged labour or premature birth of baby
2. Equipments for resuscitation to be checked
3. Immediately after delivery, place the baby under radiant heat on a pre-warmed mattress. Dry the infant properly and remove the wet linen
4. Assess the newborns response to ABCs of resuscitation.

Airway:

- If the neonate is obviously depressed, the cord is clamped early and resuscitation is initiated immediately.
- Breathing normal begins in 30 seconds and sustained within 90 seconds. Respiratory rate should be 40-60 breaths/min and heart rate 120-160 beats/min.
- A relatively large cranial occiput and moulded head shape, typically hinders placing the newborn in sniffing position.
- Placing a rolled cloth behind the neck as shoulders helps ensure proper positioning.
- Suction the mouth then the nose
- If then also sufficiently depressed it may require assisted ventilator or if thick meconium is present in the amniotic fluid, then prompt tracheal intubation is done, which facilitates thorough suctioning.
- Once the airway is clear, assess newborns breathing. In addition to breathing, heart rate, colour, reflex, irritability should be evaluated.
- Apgar score is recorded at 1 minute (correlates with survival) and at 5 minutes (correlate with neurological outcome)
- But irrespective of heart rate if the newborns colour is poor as if breathing efforts seem inadequate, the next step is to assist breathing.

Breathing:

- If the newborn has good respiratory effort, proceed to checking heart rate and colour. Mask ventilation will be necessary if breathing efforts are poor or absent. Further stimulation of baby may only delay ventilator assistance.
- Recheck the head position and we have to give several positive pressure breaths by mask. After effective breaths at a rate of 40-60 breaths/min for 15-30 seconds, assess heart rate.

Circulation:

Check the heart rate with a stethoscope or by feeling the base of umbilical cord, if beats are < 100/min effective positive pressure ventilation should be initiated or continued.

- If Heart Rate > 100 beats/min, assess colour
- If central cyanosis is present give supplemental O₂ with FIO₂ 80-100%
- If heart rate is between 60-100 beats/min, continue assisting ventilation for another 30 seconds, and then recheck the heart rate.
- If heart rate now exceeds 80 beats/min or appears to be rapidly increasing continue PPV and with for improvement in heart rate, colour and onset of spontaneous respiration.
- If heart rate remains below 80 beats/min and not increasing initiate chest compressions.
- If heart rate < 60 beats/min, continue assisted ventilation and begin chest compressions at 100 compressions/min. recheck the heart rate every 30 seconds continue chest compressions until the heart exceeds 80 beats/min. if heart rate remains below 80 minutes, the infant should be intubated and given an endotracheal dose of epinephrine and preparations should be made for umbilical venous catheter placement.

Techniques of chest compressions:**I. Two thumb encircling hands technique:**

- It is generally preferred because it appears to generate higher peak systolic and coronary perfusion pressures.

II. Two-finger technique:

- Is alternative method 1/3rd to 1/2 AP diameter. The depths of compressions should be approximately 1/3rd of antero posterior diameter of the chest and enough to generate a palpable pulse.
- Compressions should be interposed with ventilation in a 30-32 ratio such that are given 100/minute. Heart rate should be checked periodically. Chest compressions stopped when heart rate exceeds 80 beats/min.

Vascular Access:

- Cannulation of umbilical vein with a 3.5F or 5F umbilical catheter is easiest and preferred technique. The tip of the catheter should be just below skin level and allow free back flow of blood.
- Further advancement may result in infusion of hypertonic solutions directly into the liver. A peripheral vein or ET tube can be used as an alternative route for drug administration. Cannulation of one of two umbilical arteries allows measurement of blood pressure of facilitates blood gas measurements.
- Care must be taken not to introduce any air into either the artery or the vein.

Volume resuscitation:

- Some of the neonates at term and 2/3rd of premature infants requires resuscitation because of hypovolemia at birth. Diagnosis is based on physical examination (low BP and pallor) and poor response to resuscitation.
- Normal Blood pressure depends on birth weight and varies from 50/25mmHg for neonates weighing 1-2kg to 70/40mmHg for those weighing over 3kg.
- Volume expansion may be accomplished with 10ml/kg of either RL; NS or type O-ve blood cross matched with maternal blood is less common causes of hypotension includes treat hypocalcaemia, hypo mg2+ and hypoglycemia.

Drug therapy:

- If a new born needs a medication urgently, adrenaline can be given intra-tracheal other medications can be given through parenteral access.

Adrenaline: 0.01 to 0.03 mg/kg 1:10000 = 0.1 to 0.3 ml/kg

- Indicated in a systolic or bradycardia heart rate < 80beats/min despite 30 seconds patient chest compressions.

Dose:

- 0.01 to 0.03ml/kg (0.1 to 0.3ml/kg of 1:10,000 solution) and repeated every 3-5 minutes.

Epinephrine + NS 1ml saline drain given through ET Tube**Naloxane: 0.1 mg/kg or 0.2 mg/kg; (0.25 to 0.5 ml/kg of 0.4 mg/ml Concentration)****Indications:**

- Maternal history of narcotics less than 4 hours before delivery and evidence of respiratory depression in newborn.

Dose: 0.1g/kg (iv) or 0.2g/kg (im) it can given by endotracheal tube or subcutaneous

Bicarbonate:**Indication:**

- Prolonged arrest not responding to other drug 2mEq/kg of a 0.5mEq/kg 4.2% solution.
- The infusion rate should not exceed 1mEq/kg/min to avoid hypertonicity and intracranial /hemorrhage.

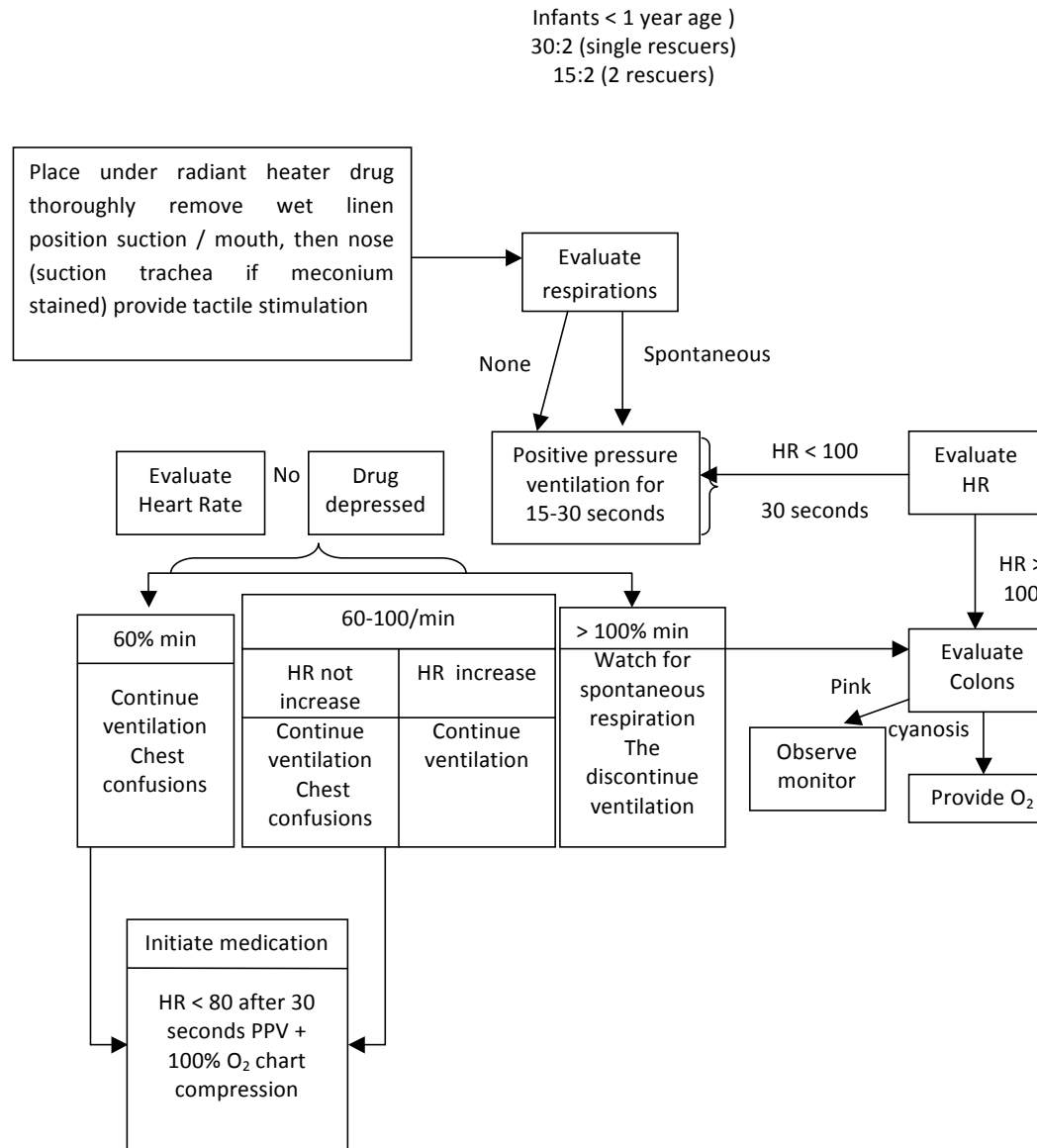
Calcium gluconate: Indication; hypocalcaemia, Suspected Mg toxicity.

Dose: Cacl2 30mg/kg, calcium gluconate 100mg/kg.

Glucose: Indicated in hypoglycemia 8mg/kg/min of a 100% solution.

Dopamine: 5µg/kg/min to support arterial blood pressure.

STEPS IN NEONATAL RESUSCITATION



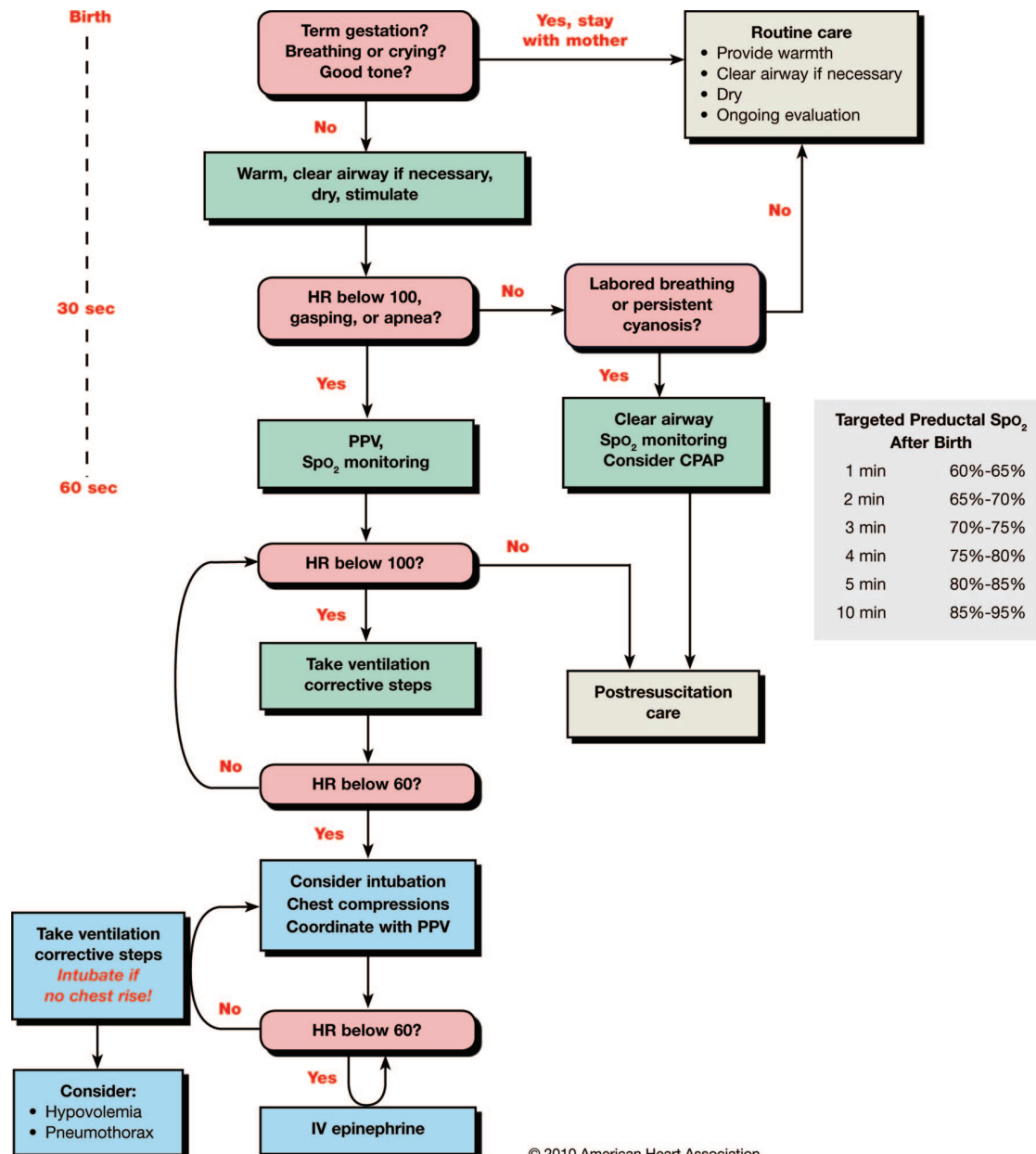


Figure. Newborn Resuscitation Algorithm.

Points:

- Brachial artery
- Technique - 2 finger for single rescuer and 2 thumbs - encircling two rescuer
- Compression depth = 4 cm or 1.5 inches
- Compression ventilation ratio: at least 2 rescuer 15:2 and 1 rescuer 15:2
- Rate 100 bts/min.

Dose:

- Adrenaline = 0.01 to 0.03 mg/kg
- Naloxone = 0.25 to 1 microgram/kg increments
- Bicarbonate = 1 mEq/kg
- calcium Gluconate = 100 mg/kg
- Glucose = 8 mg/kg/min
- Dopamine = 5 microgms/kg/hr

CPR: Guidelines

- C-A-B
- 4 cm = Infants
- 2 inches or 5 cm in children
- Infants < 1 year age - Single rescuer - 30:2
- 2 Rescuer - 15:2



Pediatric - Chain of Survival

Narcotics:**1) Pethidine:****Crosses placenta**

- Safe dose – 50-100mg
- Administration during 1st trimester – polydactyly, hypospadias.
- Higher doses – changes in FHR variability, Apgar scores – low, changes in oxygen saturation, convulsions.

Dose delivery interval:

- < 1hr: less neonatal depression
- > 5 hrs: lower APGAR scores

Elimination $\frac{1}{2}$ life:

- In adults 3 hrs
- In neonates 24-30 hrs

Pentazocine:

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

Morphine:

- Causes greater neonatal depression and FHR variability than other narcotics.
- Longer duration of action.
- Dose delivery of 3 $\frac{1}{2}$ hrs causes neonatal depression.
- Teratogenic effects - ↓ in fetal brain size.

Fentanyl:

- Rapidly crosses placenta
- Safe dose 1 μ gm/kg – no effect on Apgar and neuro behaviour scores.

Naloxone:

- Crosses placenta and detected in fetus in min.
- Reverses respiratory depression in neonates exposed

- To narcotics in utero in dose of 0.01mg/kg to 0.1mg/kg (IM/IV/ETT/SC)
- Should not be given prophylactically to mother to antagonize the narcotic effects because it reverse maternal analgesia.

Propofol:

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

Benzodiazepines:**Diazepam:** crosses placenta rapidly

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

Midazolam: transfer is less than diazepam

- Produces neonatal depression < diazepam but more than thiopentone

INHALATION AGENTS:**Nitrous oxide:** rapid placental transfer

- Reaches 87% of maternal concentration in 20min. Its use can adversely affect a severely asphyxiated fetus.

Halogenated volatile agents:**Safe concentration:**

- Halothane 0.5%, enflurane 1%, and isoflurane 0.75%
- No change in uterine tone, responsive to oxytocin
- No effects on neonatal acidosis and oxygenation.

Halothane:

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

Enflurane:

- At concentration > 1% causes maternal and fetal bradycardia, ↓ uterine blood flow and fetal acidosis.
- Metabolized to inorganic fluoride – negligible chance for nephrotoxicity.

Isoflurane:

- 2 Mac isoflurane causes ↓ cardiac output and uterine blood flow resulting in fetal acidosis.
- Studies following use of halogenated agents reveal – carcinogenicity, teratogenicity and mutagenicity in fetus.

Barbiturates:**Thiopentone:**

- Rapid transfer because largely unionized at physiological pH. But fetal concentration is less due to extensive protein binding in mother.
- During concentration in fetal brain is less because –
 - Major portion of fetal blood from placenta enters portal circulation and drug is extracted before it enters systemic circulation.
 - Dilution of blood (from viscera and lower extremities) before the drug reaches the brain.
- Thus, with a dose of 4mg/kg no fetal CNS depression occurred.
- Dissociative anesthetic ketamine
- Safe dose 0.2-0.4mg/kg (analgesia)
- 1mg/kg (induction)
- Doses > 1.5 mg/kg → low APGAR scores and hypertonia noted in 1st trimester – cranial anomaly (rare).

Local anesthetics:

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

Lignocaine:

Rapidly crosses placenta

Produces indirect effects on fetus following epidural and spinal anesthesia resulting in fetal bradycardia and acidosis.

When used in cont. epidural analgesia – tachyphylaxis drug accumulation → ↑ placental transfer.

Bupivacaine:

- Limited transfer due to high protein binding and ionization.
- Drug accumulation is less.
- Following. Spinal, very small amounts, cross placenta and do not have any detrimental effects on fetus.
- Following Epidural maternal peak concentration – 15-30min and the umbilical vein concentration is < 30% maternal concentration even after repeated injections.

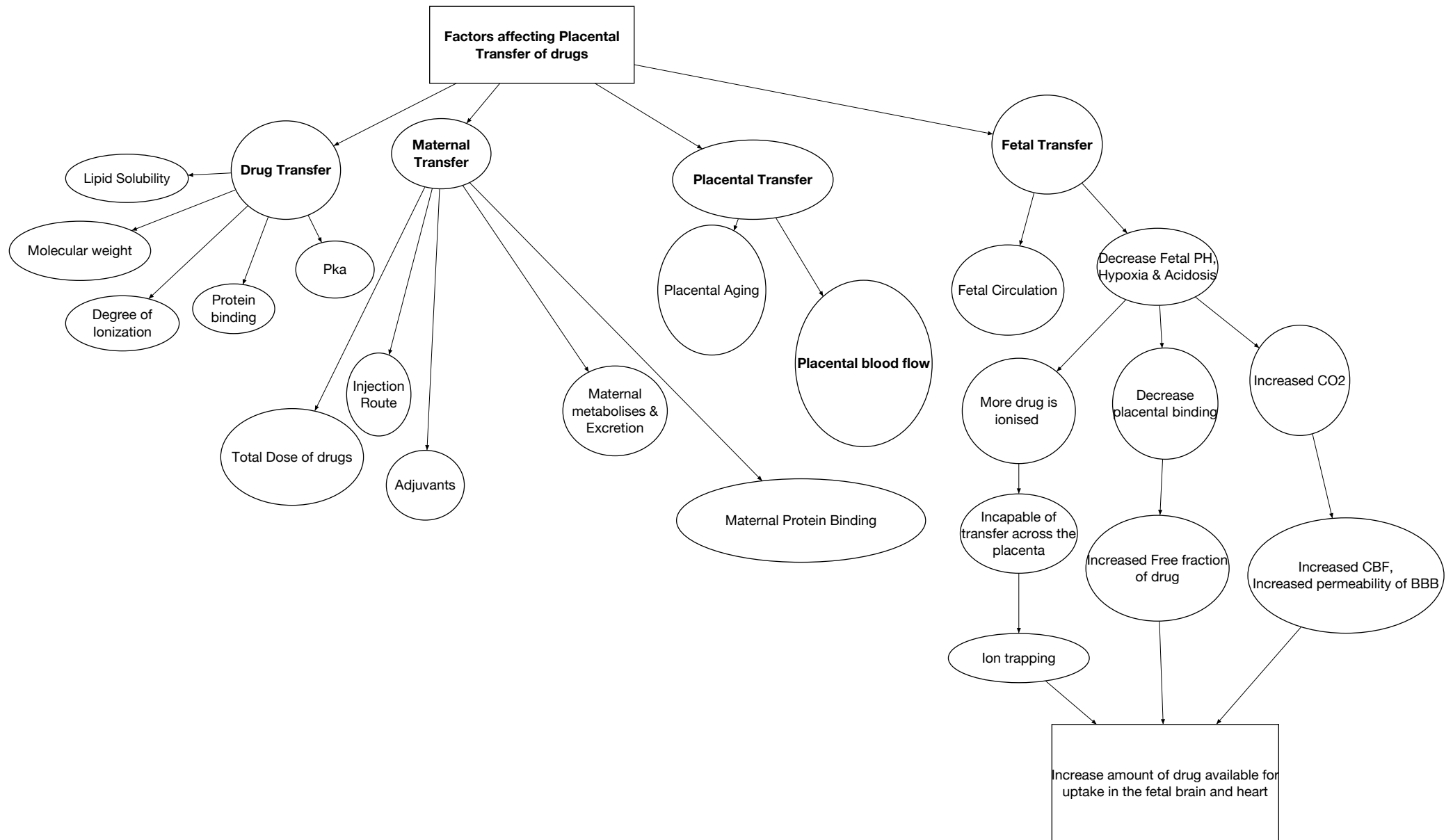
Magnesium sulphate:

- Given during severe eclampsia may cause hypotonia and respiratory failure in neonate.

Adrenaline:

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





Assessment of pain in infants is very difficult for post operate analgesia, multimodal approach is commonly used.

- Common modalities of pain management
 - **Mild pain- NSAIDS**
 - **Moderate pain-**
 - NSAIDS with opioid
 - IV opioid(consider addition of fixed interval NSAIDS)
 - IV opioid by PCA
 - Continuous infusion of opioid with prn rescue doses opioid
 - Fixed interval dosing of opioid
 - Regional anesthetic technique- caudal epidural
 - Topical lidocaine gel
 - **Severel pain-**
 - IV opioid by PCA
 - Regional anesthetic technique-caudal
 - Peripheral nerve blocks
 - Topical lidocaine gel repeatedly
- Paracetamol(non opioid analgesic):-for mild moderate pain
- Good safety margin in infants and neonates
 - Oral dose:-10-15mg/kg QID
- **Maximum dose:**
 - 180mg/kg/day <12 years
 - 75mg/kg/day in infants
 - 40mg/kg/day in neonates
- **Rectal dose:**
 - 30-40mg/kg initially
 - Followed by 20mg/kg 6 hourly.
- **NSAIDS:-** increase risk of bleeding
 - Ibuprofen 6-10mg/kg to maximum of 40mg/kg oral 8 hourly
 - Diclofenac 2-3/kg(>1 year child) oral 8 hourly
 - Ketorolac 0.8mg/kg

- **Cognitive behavioral techniques:**

- Preparation and rehearsal
- Distraction
- Relaxation
- [Ketamine - 6mg/kg oral/1-2mg/kg iv]

- **Opioids:**

- Used for moderate to severe pain Mx
- But blood level maintained with in a therapeutic range
- For optimal analgesia there should be a steady state level of opioid in plasma
- Can be administered im, iv, oral, sc, caudal/epidural

- 1. **Recommended dosage of morphine**

- 50-100micrograms/kg iv incremental boluses
- Morphine infusion-1mg/kg morphine in 50ml saline i.e.
- 20micrograms/kg/ml, rate 1-2ml/hour(20-40microgram/kg/ml)
- Morphine PCA
 - Initial bolus 20micrograms/kg Q 10min till 0.1mg/kg
 - PCA dose-10-25microgram/kg/hour
 - Basal rate-10-25microgram/kg/hour
 - 4 hourly maximum-300micrograms/kg
 - Lockout time-6-12min

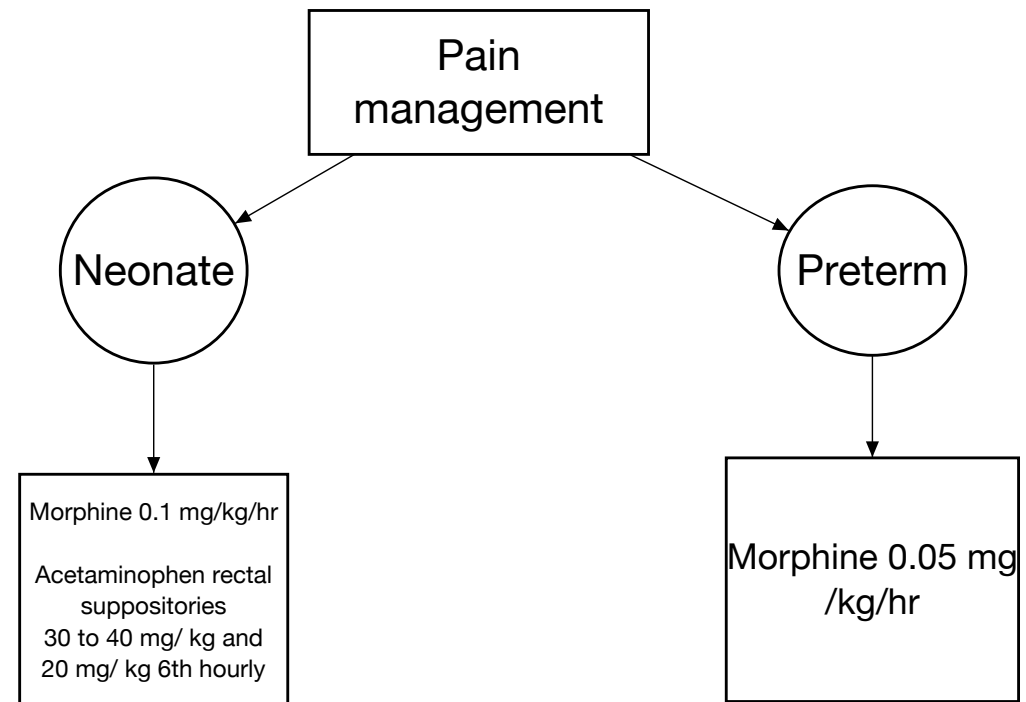
- 2. **Transmucosal fentanyl:** is used for acute pain relief

Transdermal fentanyl for chronic pain relief

- 3. **Codeine phosphate:** 1mg/kg orally/i.m/PO 6 hourly

3. Oromorph: 400 microgram/kg PO 4th hourly

- Topical EMLA/cream can be applied for pain relief repeatedly by parents but toxic dose should not reach.
- Caudal epidural/is technically easier in infants and children producing good post operating pain relief. Along with opioid/ $\alpha 2$ agonist
- Penile block/ring block/: dorsal nerve of penis and posterior scrotal branches f pudendal nerve (s2-4)with branches from ilioinguinal and genitofemoral and post. Cutaneous nerve of thigh are blocked. Local anaesthesia injected deep to superficial fascia of penis and s.c.around root of penis. Avoid adrenaline solutions.



- Fluid management in infants is the most important & critical as smaller volumes involved.
- Normal daily water consumption in infant is 10% - 15% of body weight.

Fluid calculation is based on the **Holiday Segar Formula:**
It is based on energy requirement.

- 1- 10 kg infant need about 100 cal/kg/24 hr
- 10 - 20 kg infant need about 1000 + 50 cal/kg/24 hr
- > 20 kg infant need about 1500 cal + 20 cal/kg/24 hr

Approximately 1ml of water is needed for each calorie expanded:

- 1 - 10 kg infant requires 100ml/24hr/kg
- 10 - 20 kg infant requires 1000 ml + 50 ml/24hr/kg
- > 20 kg infant requires 1500 + 20 ml/24hr/kg

Estimated Fluid requirements - 4:2:1 Formula:

Body Weight	Fluid
First 10 kg	4ml/kg/hr
Second 10 kg	2 ml/kg/hr
Subsequent (Remaining Weight)	1 ml/kg/hr

- The Fluid of choice is controversial: Hartman's Solution, 1/2 Normal saline
- Neonates requires 3-5mg/kg/min of glucose infusion to maintain euglycemia (40 - 120 mg/dl)
- Premature requires 5 - 6 mg/kg/min of glucose infusion.

- After using 4:2:1 Formula the amount obtained is **Maintenance fluid**
- To calculate deficit the Maintenance fluid is multiplied by number of hours of fasting
 - **Deficit = Maintenance fluid X number of hours of fasting**
- The deficit is administered in aliquots of 50% in the first hour, 25% in 2nd 3rd hour respectively.

Replacement requirements:

- It can be divided into blood loss & third space loss.

Blood Loss:

- Blood loss is typically replaced by non-glucose containing crystalloids (e.g: 3 ml of HRL with each ml of blood)
- Because their small intravascular volume, neonates & infants are at increased risk for electrolyte disturbance (Hyperglycemia, Hyperkalemia & Hypocalcemia)
- Platelets One unit per 10kg & FFP 10-15ml/kg should be given when blood loss exceeds 1-2 blood volumes.
- Cryoprecipitate is 1U/10 kg.

Third Space Loss:

Degree of Blood loss or Tissue Trauma	Additional fluid requirements
Minimal (e.g. herniorrhaphy)	0 - 2 ml/kg
Moderate (e.g. Appendicitis)	2 - 4 ml/kg
Severe (e.g. Bowel resection)	4 - 8 ml/kg

- Magnitude of perioperative third space loss is proportional to the amount of tissue manipulation.
 - E.g. Burns, trauma, infected tissues, etc.

Introduction:

- The gastrointestinal tract is a flexible, compliant and motile tube. Clinical presentation of intestinal obstruction in neonates and infants is based on signs and symptoms which occur as a result of the level of obstruction of the tract. Intestinal obstruction can occur due to structural (atresia, adhesions) or functional (dysmotility, Hirschsprung's) defects. Distension of the gut occurs proximal to the point of obstruction, and is more pronounced the more distal the obstruction. In general, surgery is indicated when clinical signs of obstruction are evident. The presence of the 'double-bubble' sign or loops of distended bowel seen in the plain X-ray or contrast study of the abdomen are indications for surgical intervention.

EVALUATION OF HYDRATION STATUS, ELECTROLYTE IMBALANCE, FLUID AND ELECTROLYTE RESUSCITATION:

- Fundamental to any approach to fluid management are the answers to three basic questions:
 1. What is the fluid and electrolyte deficit?
 2. What fluid is required to maintain the patient?
 3. What are the ongoing losses?
 - a. Based on the answers to these questions, there are three basic principles of management

Fluid replacement is based on the 3 R's

- a. Repair of deficits
- b. Regular maintenance
- c. Replacement of losses

FLUID THERAPY:**Replacement of fluid deficit:****This is based on the 3D's**

1. Degree of dehydration estimated
2. Determine type of fluid lost
3. Develop an approach to correct deficit

Estimation of degree of dehydration:

- Dehydration is estimated on the basis of history, e.g., of losses/poor feeding, etc., and the clinical examination (Table 1)
- Mild dehydration (1-5%) The decision is made mainly on the history of gastrointestinal losses.
- Moderate dehydration (6-10%) is associated with clinical signs, e.g., tenting of skin, dry mucous membranes, loss of weight, lethargy, sunken eyes and fontanel.
- Severe dehydration (>10%) is associated with cardiovascular instability, e.g., skin mottling, cold peripheries, tachycardia and hypotension. In addition, there may be neurological features such as irritability or coma.
- There are no laboratory tests for dehydration, although an inappropriately high haematocrit may indicate significant dehydration.

Table 1. Signs and symptoms of dehydration

	Mild dehydration	Moderate dehydration	Severe dehydration
Fluid deficit	<5%	6-10%	>10%
Sensorium	Well, alert	Restless, irritable	Lethargic or floppy
Eyes	Normal	Sunken	Very sunken
Tears	Present	Absent	Absent
Mucous membranes	Moist	Dry	Very dry
Anterior fontanel	Normal	Sunken	Deeply sunken
Thirst	Drinks normally	Thirsty	Drinks poorly
Turgor (skin pinch)	Normal, goes back quickly	Reduced, goes back slowly	Reduced, goes back very slowly
Urine output	Normal	Reduced and dark	Severe oliguria/anuria
Capillary refill	Normal	<2 seconds	>3 seconds

Determining the type of fluid loss:

The history gives a clue to the type of fluid lost. The serum osmolality and electrolyte profile may also reflect the type of fluid lost.

The role of osmolality:

- The serum electrolyte concentration may reflect the type of losses incurred and the amount of deficit requiring correction. This is reflected in the serum osmolality which may be isotonic, hypotonic or hypertonic. If isotonic, (serum osmolality 270-300 mOsmol L⁻¹, serum sodium 130-150mmol L⁻¹) problems are minimal. However, hypotonic and especially hypertonic serum may have important clinical implications. Hypertonic dehydration (serum osmolality >310, serum sodium, >150 mmol L⁻¹) requires special care as cerebral oedema may occur during rehydration.

Developing an approach to replace volume and electrolyte deficits:

The volume of fluid required to correct the deficit must be calculated before commencing replacement.

Rule of thumb for volume loss

- 5% dehydration -50mlkg⁻¹
- 10% dehydration – 100mlkg⁻¹
- Special care must be taken in states of malnutrition (especially kwashiorkor), disproportionately small or chronically ill children and in hypertonic dehydration. These situations may require a slower rate of fluid resuscitation along with careful monitoring.

SODIUM**Hyponatremia:**

- <130mmol L⁻¹ may result in clinical signs
- <120mmol L⁻¹ may be life-threatening due to cerebral swelling, oedema and coning Associated oliguric renal failure may be difficult to reverse and is best prevented.

Formula for correction of Na⁺ deficit:

Sodium deficit (mmol) = {Normal serum [Na⁺]-observed serum [Na⁺]} x 0.6 x body weight (kg) Half this calculated amount is given and the situation is reassessed

Potassium (K⁺):

- Potassium plays an extremely important role in the homeostasis of acid-base balance in the body. Only 50-60 mmol K⁺ exists in ECF, the plasma concentration being between 3.4-5.6 mmol L⁻¹. The remaining K⁺ is intracellular, two thirds being in muscle.
- Potassium requirement is 2-4mmol kg⁻¹day⁻¹ (approx 3mmol kg⁻¹day⁻¹ depending on the gestational age). The minimum urinary loss of K⁺ is 20mmol/day. Urinary K⁺>30mmol/day and a serum level below 3.5 mEqL⁻¹ indicates excessive loss.
- An increase in renal excretion of K⁺ occurs in respiratory and metabolic alkalosis where it competes with H⁺ for Na⁺ retention. More K⁺ will be required in alkalosis because K⁺ will be excreted with bicarbonate (HCO₃) until acid-base status equilibrates. In alkalosis, the H⁺ ions also move into the cells and K⁺ moves out into the ECF. This will often correct itself in conditions such as pyloric stenosis following treatment. In acidosis, there is the reverse situation and the K⁺ is retained.
- Hypokalemia may also be a problem in patients managed with total parenteral nutrition and can be prevented by supplementation. Anuria must be excluded prior to the administration of potassium chloride (KCl).

K⁺ administration in hypokalemia

- Great care must be taken in the administration of K⁺ intravenously. The maximum rate of administration of potassium is 0.5mmolhr⁻¹ KCl and usually not more than 40mmol is added to 1 litre of fluid.

Sodium bicarbonate (NaHCO₃):

- Sodium bicarbonate is used with caution. In acidosis, correction is generally carried out for pH<7.10 or a standard bicarbonate less than 5mmolL⁻¹

The following formula can be used:

- **Base excess (deficit) x 0.3 x body weight (kg) = mmol NaHCO₃**
- **[1ml 8.4% NaHCO₃ =1mmol]**

- Avoid giving NaHCO_3 as a bolus but add it to the rehydration fluid. Initially correct 25% over 4 hours. Stop correction with 8.4% NaHCO_3 when pH is >7.15 and standard bicarbonate is 8-10mmol/L. The potential dangers of NaHCO_3 include hypokalemia, cerebral oedema and an increased oxygen affinity resulting in peripheral anoxia.

Regular maintenance fluid replacement:

This depends on the following: (i) volume required to be replaced, (ii) electrolyte requirement, (iii) rate of administration, (iv) composition of fluid and (v) frequent clinical review.

Volume of fluid:**Rule of thumb**

- 0-10kg-100ml $\text{kg}^{-1} \text{ day}^{-1}$
- 10-20 kg 50 ml $\text{kg}^{-1} \text{ day}^{-1}$
- $>20 \text{ kg}$ 20ml $\text{kg}^{-1} \text{ day}^{-1}$

Neonate:

- Day 1 60ml $\text{kg}^{-1} \text{ day}^{-1}$
- Day 2 90ml $\text{kg}^{-1} \text{ day}^{-1}$
- Day 3 120 ml $\text{kg}^{-1} \text{ day}^{-1}$

Electrolyte requirements:

- Electrolytes such as Na^+ , K^+ , Cl^- should be replaced by administering approximately 3 mEq $\text{kg}^{-1} \text{ day}^{-1}$

Rate of administration:

- Replacement of intestinal losses
- The electrolyte contents of gastrointestinal fluid in various regions of the tract are shown in Table 2.

Table 2. Electrolyte profile of intestinal contents in mmol^{-1} .

	Na^+	K^+	H^+	Cl^-	HCO_3^-
Gastric juice	40 ± 20	10 ± 5	85 ± 5	85 ± 5	-
Small intestine	110 ± 10	10 ± 5		115 ± 15	25 ± 5
Bile	130 ± 10	10 ± 5		70 ± 20	50 ± 20
Ileostomy	100 ± 130	10 ± 5		80 ± 40	
Colostomy	70 ± 20	20 ± 10	-	50 ± 20	-

Nasogastric losses:

Nasogastric losses reflect mostly gastric losses in normal postoperative patients. In intestinal obstruction however, excessive small bowel contents may be drained via the nasogastric tube. Replacement is tailored to replace gastric losses but does not fully match gastric juice in content.

	Gastric juice mEq/L	Replacement fluid 0.4% NaCl + 30mEq/L-1KCL
Na^+	60 (60-75)	77
Cl^-	160 (105 -170)	107
K^+	10(5-30)	30
H^+	0-65	-
HCO_3^-	-	-

Replacement of ileostomy losses:

- Small bowel content differs from gastric juice mainly in its sodium and bicarbonate content. Replacement of ongoing losses is important. There may be sudden and dramatic increase in ileostomy losses with massive fluid shifts. This requires careful monitoring and may require a volume expander, e.g., plasmalyte B. Ileostomy sodium losses may exceed intake necessitating spot checks of urine sodium (Normal urine $\text{Na}^+ > 10 \text{ mmol/L}$).
- If $< 10 \text{ mmol/L}$, add sodium i.e., NaHCO_3 or isotonic saline ($0.9\% \text{ NaCl}$).

Pyloric stenosis:

- Pyloric obstruction is characterized by ongoing losses of gastric contents particularly hydrogen (H^+) and chloride (Cl^-). The morbidity is directly related to the degree of dehydration and duration of symptoms (patient is malnourished). It is a clinical emergency largely due to the degree of dehydration and the biochemical profile (hypochloraemic, hypokalemia, metabolic alkalosis).
- The homeostatic mechanisms control mainly the H^+ ion. When chronic losses of H^+ occur, the body tends to retain H^+ at the renal tubule, in exchange for K^+ . This results in an alkalotic urine rich in K^+ . In an attempt to maintain the pH, H^+ is removed from the cell, driving K^+ intracellularly. This step is important as this process is reversed with resuscitation resulting in further shifts of K^+ . As a result, despite the fact that gastric juices are low in K^+ , a state of hypokalemia exists with its attendant risks to the heart.
- Treatment is with intravenous replacement of electrolyte losses i.e., Cl^- and glucose (we use $0.45\% \text{ NaCl}$ in $5\% \text{ dextrose}$). K^+ is restored. Do not replace K^+ until there is urine production. Operative correction of the pyloric stenosis (pyloromyotomy) is delayed until biochemical normally has been restored.

Peritonitis:

- Peritonitis results in a massive sequestration of fluid resulting in a third – space loss which is mainly in the ECF fluid compartment. Patients with peritonitis are often severely dehydrated as a result of third space loss plus the effects of their primary pathology and vomiting. The degree of dehydration is related to the duration of symptoms. Ischaemic bowel results in particularly excessive third-space losses and requires urgent correction (preferably with colloid e.g., $10\text{--}20 \text{ ml Kg}^{-1}$ of albumin or plasma). Babies with peritonitis must have their fluid status aggressively corrected as rapidly as possible prior to surgery or else vasodilatation on induction of anaesthesia may lead to shock. Mortality results from multiorgan failure due to shock and sepsis. Adequate hydration and prevention of shock are thus vital in the management of patients with peritonitis.

Definition of third-spaces loss:

- This can be defined as internal loss of ECF into a non-functional space (dysfunctional second-space).
- This loss of fluid may be:
 - Extracellular, e.g., peritonitis –sequestration
 - Intracellular, e.g., haemorrhagic shock
 - Both extracellular and intracellular, e.g., major burns
- If the ECF is depleted of sodium, water will move into the intracellular fluid (ICF) until osmolality is equalized. Fluid balance cannot be restored because of an osmotic / hydrostatic pressure difference following diffusions of fluid out of intravascular compartment into the ECF.

Estimation of third-space losses:

- To estimate third-space losses, the abdominal cavity is divided into four quadrants and one fourth of the maintenance volume calculated for fluid requirement for each quadrant involved.
- Additional factors which contribute to third space loss are obstruction, inflammatory reaction, and surgical intervention. Requirements to restore third space loss may be twice the normal calculation.

Intussusceptions and intestinal obstruction:

- Intussusceptions are the most common form of intestinal obstruction which is seen between infancy and 5 years of age. It is caused by a segment of bowel 'telescoping' into a more distal bowel segment. Following a viral infection, Peyer's patches in the bowel wall enlarge and these become the focal or precipitating point for ht telescoping to take place. This condition presents as any other case of intestinal obstruction with an 'acute abdomen'. Often, there is a history of the baby passing bloody or 'red currant jelly' stools.

GENERAL CONSIDERATIONS FOR ANESTHETIZING NEONATES / INFANTS

- All children with intestinal obstruction require anaesthesia for surgery. Infants less than 1 year of age have a higher morbidity and mortality than other patients. Children in the age group differ from the older child and adults in several ways (Table 3).

Pulmonary and airway differences:

- The trachea is short
- The larynx is higher in the neck
- Large tongue
- Floppy epiglottis
- Small mouth
- Short neck
- Apparently anterior larynx
- Neonates and infants have a higher incidence of airway obstruction
- Hence there is greater tendency to desaturate with a high incidence of hypoxia
- Faster respiratory rate
- Higher alveolar ventilation / functional residual capacity ratio
- Greater oxygen consumption (>6 mlkg-1min-1)

- Decreased ventilatory response to CO₂
- Periodic breathing with episodes of apnoea
- Increased work of breathing because of non-compliant lungs and a very compliant chest wall
- Fewer type I diaphragmatic muscle fibres (tire more quickly)
- Stress, surgery, anaesthesia and sepsis may accentuate periodic breathing and apnoea.

Cardiovascular physiology:

- Cardiac output is dependent on heart rate and ventricular diastolic filling.
- Myocardial performance is near maximum.
- Decreases in blood volume are poorly tolerated
- Parasympathetic innervations to the heart if fully developed at birth whereas the sympathetic supply continues to develop for 6 weeks.
- Possibility of shunting via a patent ductus arteriosus or foramen ovale
- Foetal haemoglobin with a resultant leftward shift of the oxyhaemoglobin dissociation curve

Table 3. Differences in cardiovascular physiology in neonates, infants, children and adults:

	Neonate	Infant	1 year	5 years	Adults
O₂ consumption (mlkg-1min-1)	6	5	5	5	3
Systolic BP (mmHg)	65	90	95	95	120
Heart rate (beats min-1)	130	120	120	90	77
Blood volume (ml Kg-1)	85	80	80	75	70
Haemoglobin (g dL-1)	17	11	12	13	14

Renal function:

- Decreased glomerular filtration rate
- Limited concentration ability
- Drug clearance may be delayed.

Thermoregulation:

- Large surface area
- Small mass
- Thin layer of subcutaneous fat
- Limited thermogenesis

Miscellaneous:

- The arteries and veins have an elasticity that enables them to cope with a range of intravascular volume
- The brain and heart are more resistant to hypoxia as compared to the older child and adult. Full recovery is common after cardiac arrest
- Stress is poorly tolerated by premature babies
- Both respiratory and metabolic acidosis are common
- Hepatic insufficiency is reflected by the limited ability to conjugate or detoxify drugs, maintain blood glucose levels, synthesise coagulation factors abnormal bilirubin loads.

Preoperative management:

- Any child with suspected intestinal obstruction should be rehydrated at once since circulatory collapse can occur within a few hours of the onset of symptoms. Fluid losses into the bowel, especially in intussusceptions, can be quite significant and are commonly underestimated. Infants who present with signs of hypovolaemia should be resuscitated rapidly with fluids including colloids and sometimes blood.
- In severe cases, 20ml/kg, of fluid should be administered as rapidly as possible using a syringe with careful assessment of clinical response. In severe cases, as much as 30-40ml/kg i.e., half the circulating volume may be needed.

- Vascular access may be difficult in children below two years of age and an introsseous needle may be required before intravenous access is secured. The haematocrit should be checked before surgery, especially if large volumes of colloid have been used in resuscitation.
- Surgery can usually be delayed while gastric decompression and rehydration and correction of electrolyte imbalance take place. However, in some circumstances, e.g., intussusceptions or volvulus, there may be a compromised blood supply to the bowel and delay can increase the chances of danger from ischaemia, necrosis, perforation and septic shock. Occasionally the metabolic acidosis cannot be corrected until the necrotic segment of bowel has been resected.

RAPID SEQUENCE INTUBATION (RSI)

- Rapid sequence intubation is the rapid induction of general anaesthesia that results in rapid unconsciousness and muscle relaxation to facilitate intubation under optimal conditions with minimal side effects. This technique is mainly used in patients with full stomach or intestinal obstruction in order to prevent pulmonary aspiration of gastric contents.
- RSI should not be initiated until equipment is available and ready to use such as two laryngoscopes, a stylet, a large-bore suction catheter and appropriate sizes of endotracheal tubes (ETT).

Steps of RSI:

- Brief history and airway assessment
- Preparation of equipment and medications
- Preoxygenation
- Premedication with adjunctive agents (atropine, defasciculating agents)
- Sedation and induction of unconsciousness
- Cricoids pressure
- Muscle relaxation
- Intubation
- Verification of ETT placement
- Release of cricoids pressure
- Secure the ETT

Commonly used muscle relaxants in RSI are shown in Table 4

Table 4: Muscle relaxants used for rapid sequence intubation

	Succinylcholine	Vecuronium	Rocuronium
Onset	30 to 60 seconds	90 to 240 seconds	30 to 90 seconds
Duration	4 – 6 min	30 to 90 min	25 to 60 min
IV dose	1.5 to 2 mgkg-1	0.15 to 0.2 mgkg-1	0.6 to 1.2 mgkg-1

- Atracurium is preferred in sick children especially with hepatic or renal impairment as it dissociates spontaneously into inert metabolites but intubating conditions are not very good compared to the muscle relaxants mentioned above. It may also cause histamine release if injected rapidly.

REGIONAL ANALGESIA:

- Regional analgesia combined with general anaesthesia in neonates and infants with intestinal obstruction is ideal for both intraoperative and postoperative analgesia. If the platelet count is less than 50,000 mm³, the risks and benefits of central neuraxial blockade should be considered. The commonly employed regional analgesic techniques in abdominal surgeries include single dose caudal epidural, continuous caudal epidural, continuous lumbar epidural, wound infiltration.

Prematurity and anesthetic considerations.

- Anesthetic morbidity increases directly with the degree of prematurity
- Prematurity means if infants born before 37 weeks of gestation or weigh less than 2500gm at birth
- Intra uterine asphyxia is common in premature infants and is often cause for early labour and premature delivery
- Pre term infants are more prone to asphyxia because of their reduced oxygen carrying capacity
- Most of the premature infants will have multi organ pathology like
- Airway:- subglottic stenosis, tracheomalacia
- Lungs:- chronic lungs disease, reactive pulmonary vasculature, bullae, high risk pneumonia
- Heart:-cardiomyopathy, persistent PDA
- CNS:- delayed development, seizures, hydrocephalus
- GI tract:- disordered swallowing/sucking, GE reflux, bowel obstructions
- Liver:- hepatic failure, hyperalimentation hepatitis
- Kidney:- chronic renal failure, renal tubular acidosis.
- Eyes:- retinopathy of prematurity
- Other:- malnutrition
- Premature will have high preoperative complications rate following even minor surgery
- Preoperative complications:-apnea, atelectasis, aspiration pneumonia, extubation stridor, excessive secretions coughing and cyanosis

Anesthetic considerations:**1. Impaired temperature regulation****Causes:**

- Increased surface area to volume ratio- heat loss
- Thin skin- heat and water loss
- Lack of fat insulation
- Fewer brown fat cells

Consequences:

- Hypoglycemia
- Apnea
- Bradycardia
- Metabolic acidosis

2. High risk for respiratory distress syndrome (hyaline membrane disease)

- Most common after cesarean section and it is rare after 34 weeks of gestation
- Due to deficiency in surfactant production before 26 weeks of gestation which results in alveolar collapse, shunting, hypoxemia and metabolic acidosis

- **Treatment:-** immediate artificial surfactant

3. Broncho pulmonary dysplasia(BPD):- defined as a continued oxygen requirement at 28 days of life in an neonate with a h/o RDS

- Risk factors associated with BPD- increased FIO₂, PPV, infection, PDA, fluid over load in first 5-6 days of life
- BPD is characterized by:-
 - Increased airway pressure
 - Decreased pulmonary compliance
 - V/Q mismatch
 - Decreased Pao₂, tachypnea
- Increased oxygen consumption, increased pulm. Infections
- Pulmonary hypertension and cor-pulmonale can result from severe BPD

4. Apena: defined as cessation of breathing that lasts for 15-20sec or produces cyanosis and bradycardia
 - The more the prematurity, greater chances of apnea spells post operatively
 - Post operative monitoring is recommended for next 12 hrs after surgical procedures
5. Patent Ducts Arteriosus (PDA):-
 - It may result in CHF and respiratory distress
 - Close the PDA with indomethacin before surgical intervention
6. Infection:
 - Reduced cellular and tissue immunity- pneumonia, sepsis and meningitis are common
 - Sepsis can develop in the absence of a positive blood culture, increased WBC count or fever
 - First sign infection- apnea, Bradycardia or acidosis
7. Necrotizing enterocolitis- multifactorial etiology because of hypo perfusion of the GIT, results in ischaemia high risk in small preterm infant (<32 weeks and 1500 gm weight)
 - Abdominal distension and bloody feces shock hypovolemic and require fluid resuscitation before induction
 - Rapid fluid administration may cause intracranial hemorrhage or reopening of ductus arteriosus
 - It is associated with DIC and thrombocytopenia
 - avoid nitrogen
 - avoid rapid fluid administration
 - preload adequately
 - pre operative BP maintain
 - nitrogen to be avoided and pre operative BP maintained
8. Retinopathy and prematurity
 - it is to be maintained between in infants < 1000 gm weight
 - Paco₂ to be maintained between 60-80 mm Hg whenever possible (don't give 100% oxygen for prolonged time)

- Penile block has become increasingly accepted as a better technique especially in pediatrics when compared to general anaesthesia. When the block is combined with general anesthesia it reduces intra operative requirement of anaesthetic agents and allows more rapid return of preoperative status, at the same time providing effective postoperative pain relief with minimal sedation.

For blocking a nerve.

1. Before you give any anaesthetic safely, follow the Maurice H. King's TEN GOLDEN RULES OF ANAESTHESIA.
2. Pre or paramedicate patient thoroughly according to patient's wishes
3. Follow the details of each block exactly
4. Before you start, explain carefully to the patient, what you are going to do to him
5. Always aspirate before injecting
6. Observe the patient during latent period,
7. Since the patient is conscious, talk to him, explaining all the time what is happening
8. Don't put the towel clips on the unanaesthetized skin!
9. If the block has failed don't give him anymore, if you have already given the full dose of local anaesthetics and above all don't try to operate when local anaesthesia has obviously failed.
10. Always nil per mouth before you give him a regional one.

INDICATIONS

1. Circumcision
2. Dorsal preputial slit
3. Meatotomy
4. Cutaneous biopsy of penis
5. Reduction of PARAPHIMOSIS

Special precautions

DO NOT ADD ADRENALINE to the local anaesthetic solutions

Advantages

1. Less local anaesthetic solution
2. Minimal complications
3. Prolonged postoperative analgesia
4. Ideal for day care surgeries

Disadvantages

- Some patients may require minimal sedation for block

Complications

1. Hematoma formation from piercing the dorsal veins or arteries.
2. Injecting large volume of local anaesthetic may compromise blood flow to the penis
3. Necrosis of the penile skin after circumcision and penile nerve block – remote

ANATOMY

- The penis is innervated primarily by the two dorsal nerves of the penis with a few sensory fibres from the genitofemoral (supplies skin over base of penis) and ilioinguinal (supplies base of penis) nerves. The dorsal nerves of the penis emerge from the midline from under the symphysis pubis and run together the dorsal arteries of the penis along the inner aspect of Buck's fascia (deep fascia of the penis) at the 10:30 and 1:30 positions.

Methods of blockade

- I. Technique of Bacon
- II. Technique of Dalens
- III. Technique of Yaster and Maxwell
- IV. Ring block of the penis

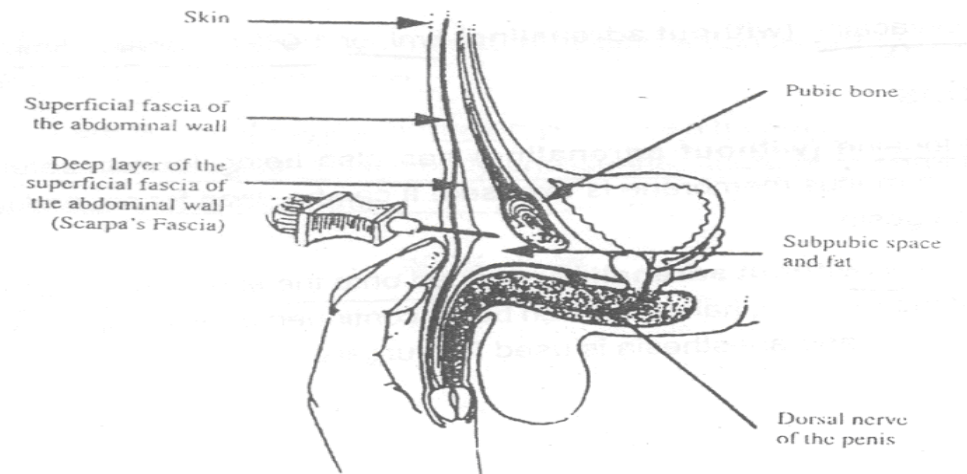
Technique of Bacon

- Midline technique. Using a 22G block needle with gentle downward traction on the penis the needle is inserted midline, perpendicular to the skin, to strike the caudad border of the symphysis. The needle is withdrawn upto the skin and redirected to the point where it is just below the caudad border of the symphysis pubis, still perpendicular to the skin. The needle should be midway between the symphysis and the base of the penis. After confirming negative aspiration for blood, and without moving the needle, the appropriate dose of local anaesthetic is injected.
- Dosage for 0.5% bupivacaine (maximum of 2mg/kg) **without adrenaline**

0 – 3	Years	-	1	ml
3 – 6	Years	-	2	ml
6 – 9	Years	-	3	ml
9 – 12	Years	-	4	ml
12 – 15	Years	-	5	ml
> 15	Years	-	7	ml

Technique to Dalens

- It refers to the technique where blockade of the dorsal nerves is performed at the subpubic space, bounded above by the pubic bone and perineal membrane, below by the crura of the corpora cavernosa covered by a fibrous tissue which fuses with Buck's fascia, and anteriorly by skin, subcutaneous tissue, and the fatty and deep membranous layers of the superficial abdominal fascia. The subpubic space may be separated into two noncommunicating compartments by a septum at the level of the suspensory ligament.


Dosage

- Two injections one on either side of midline to block the dorsal nerve at this level using 0.25% to 0.5% bupivacaine (without adrenaline) in a dose of 0.1mL/kg for each side, up to 5mL total per side.

Technique of Yaster and Maxwell

- Injecting 0.8mL of 1% lidocaine in the newborn, and 1 to 3 mL of 0.25% bupivacaine (without adrenaline) in the older child using a 25 or 26 G needle at the 0' clock and 11' clock positions just beneath the Buck's fascia, approximately 3 to 5 mm below the skin surface.

Ring block of the penis

- The simplest penile block technique and this technique does not require the level of skill required by the dorsal penile nerve block. It avoids the potential damage to dorsal blood vessels of corpus cavernosum.

Method

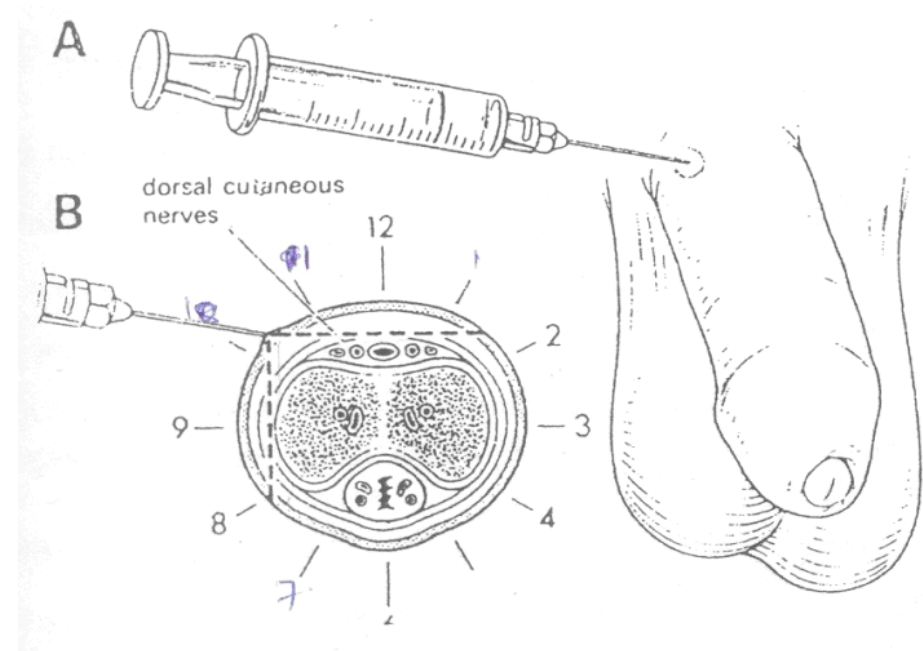
- Make a subcutaneous skin wheal. Advance the needle subcutaneously across the patient's penis. Aspirate to make sure you have not entered his corpora cavernosa, and then inject 3 to 5 ml of solution in the one O'clock position. Do the same thing for the 8 O'clock position. Then take the needle out and do the same thing on the other side, until you have anaesthetized all the four quadrants.

Dosage

- 0.25% bupivacaine (without adrenaline) 5 ml, or 1.0% lignocaine 5 ml.

OTHER METHODS

- Topical lidocaine (without adrenaline) has also been found useful once foreskin is amputated and the mucous membrane is exposed. It can be used for several days for additional postoperative analgesia.
- If 1% lidocaine (without adrenaline) dripped onto the exposed mucous membrane following amputation of the foreskin, halothane can be discontinued often completely for the remainder of the surgery, if halothane anesthesia is used for surgery.

RING BLOCK OF THE PENIS**Conclusion**

- Although the Anesthesiologist is accustomed to providing a consistent type of anesthetic, the "customary" approach may not be the best.
- Stephen C. all – 'There is an old saying that when you have a hammer everything starts to look like a nail. The Anesthesiologist must clearly analyze the nature of the procedure to properly plan what technique is most appropriate.

Prerequisites for fetal Surgery:

1. Prenatal diagnostic techniques should identify the malformation and exclude other lethal malformations with a high degree of certainty.
2. The defect should have a defined natural history and cause progressive injury to fetus that is irreversible after deliver
3. Repairs of the defect should be feasible and should reverse or prevent injury process.
4. Surgical repair must not entail excessive risk to the mother or her future fertility.

Risks associated with surgery:

1. Surgery and anesthesia can lead to fetal death and morbidity
2. Altered coagulation factors predispose the fetus to bleeding and cause difficulty in surgical hemostasis during surgery.
3. Premature labour and birth.
4. Risk in mother are hemorrhage, infection, airway problems, amniotic fluid embolism, maternal mirror syndrome (preeclampsia symptoms).

Only ASA Class I and II mothers with very sick fetuses are taken up for fetal surgery.

There are three basic types of surgical intervention

1. **Ex utero intrapartum treatment EXIT or (OOPS) generation on placental support.**
 - These interventions are done on vaginal delivery or cesarean section only a portion of fetus is delivered and brief procedures such as endotracheal intubation or examination of neck mass done while the fetus is connected to the placenta through umbilical cord. Only brief procedures were possible as placental support rarely lasts for more than 10 minutes during routine births procedure done are:
 - ECMO for CDH
 - Tracheostomy for congenital high air way obstruction syndrome
 - Resection of giant cervical neck mass
 - Laryngoscopy tracheostomy for anticipated difficult intubation

2. **Mid-gestation open procedure** – fetal defects and placental location is determined by USG and low transverse abdominal incision with wide uterine incision is put. Fetal part is exteriorized for surgery and after completion of surgery fetus is placed back into the uterus which is closed. The fetus continues to grow for rest of gestation with reversal of disease process.

Indications are:

- Surgical repair of meningocele
- Surgical resection of sacro coccygeal teratoma
- Resection of intrathoracic mass
- Temporary tracheal occlusion in CDH
- Lobectomy pneumonectomy in cystic adenoma

3. Minimally invasive mid-gestation procedures:

- Aberrant placental vessels providing imbalance of blood flow to twins can be identified and ligated with the help of endoscopes and can prevent death due to twin transfusion syndrome

Other surgeries

- Radio frequency ablation or coagulation of non viable twins umbilical cord
- Division of amniotic bonds
- Laser ablation of posterior urethral valves by fetal cystoscopy

Anesthetic goals of fetal surgery:**• Maternal anesthetic considerations:**

1. Left uterine displacement to prevent aortocaval compression must be done.
2. Avoid hypoxemia and aspiration because of increased O₂ demand and decreased FRC during pregnancy.
3. Increased risk to pulmonary edemas when MgSO₄ is used for tocolysis because of decrease in capillary oncotic pressure and increased capillary permeability.
4. Increased sensitivity to the anesthetic agents should consider the dosage of adjustments.

Fetal anesthetic consideration:

1. Anesthetic agents (Inhalation) rapidly crosses the placenta & can anesthetize fetus, but fetus requires less muscle relaxant & anesthetic agent.
2. Less cardiac contractile tissue & fetal manipulation can result in hypotension, bradycardia & cardiac collapse. Hence low level of inhalational agents to be used.
3. Surgical blood loss poorly tolerated because of low blood volume & rate dependent cardiac output.
4. Thin fetal skin predisposes to hypothermia; avoid it by limiting fetal surgical time & warm fluids.
5. Maintain uteroplacental blood flow by relaxing uterus & avoid kinking of umbilical cord, avoid increase in maternal pH & hypocapnia.

Preoperative Preparation:

1. H/O anesthetic problems previously, evaluate airway & concurrent medical problem.
2. Placenta location & fetal CVS function are evaluated by USG, ECHO & MRI.
3. Patient is ordered NPO for 6 to 8 hrs.
4. Operating theater is warmed to 80 degree F.
5. Type specific packed red cells for mother & O⁺ -ve packed red cells for the fetus are made available.
6. Sodium citrate orally & metoclopramide IV given for aspiration prophylaxis.
7. Indomethacin suppository for post operative tocolysis in midgestation procedure

Fetal Monitoring:

- Pulse oximeter probe is placed on the limb & wrapped with foil to decrease interference with ambient light.
 - Normal fetal saturation is 60 to 70%, values above 40% during surgery represents adequate ventilation.
- Echocardiography to monitor fetal HR & stroke volume.
- Fetal arterial and blood gas monitoring: blood is withdrawn by surgeons through umbilical or central vessel puncture & acidosis is corrected
- Warm fresh O⁺ blood administered to fetus to correct anemia.

Anesthetic plan for fetal surgery procedures**1. Ex utero intrapartum treatment procedures**

- EXIT procedures requires maternal laparotomy and hysterotomy while maintaining uteroplacental circulation. General anesthesia is preferable as it accomplishes both maternal and fetal anesthesia.
 - Adequate preoxygenation, rapid sequence indication
 - High concentration volatile anesthetics to maintain uterine atony but this leads to maternal hypotension which is treated by iv fluids and sympathomimetics to maintain MAP within 20% of baseline to preserve uteroplacental blood flow.
- After hysterectomy, operative site of fetus is delivered
 - Prevent kinking of umbilical cord
 - Fetal monitoring done with pulse oximetry and echocardiography
 - ET intubation done in fetus with severe cardiac defects to prevent hypoxemias at birth. LMA can also be put, but in the fetus has airway problem, tracheostomy performed.
 - Fetal anesthesia supplemented by im or iv vecuronium and fentanyl.
 - If blood loss is present, surgery can perform iv cannulation to give drugs and fluids.
 - After completion of procedures infant is delivered, cord is divided and baby placed in the care of a neonatologist
 - Following this uterine, atony reversed by decreasing concentration of volatile anesthetic and giving methergin₁₀
 - Adequate fluids and blood replaced.
 - Epidural catheter may be placed for postoperative analgesia.

2. Mid gestation open fetal surgery:

- This is similar to EXIT procedures as far as the exposure of the fetus is concerned but is done in the second trimester. Since the fetus is returned to uterus after completion of intervention, the parturient requires tocolysis and fluid restriction.
- Mother is induced by rapid sequence technique. An arterial line taken before increasing concentration of volatile anesthetic upto 2½ MAC's. Fetal monitoring done with pulse oximetry and echocardiography.
- Fetal analgesia supplemented with iv or im medication. After completion of intervention fetus is returned to uterus. The amniotic fluid is replaced by warmed Ringer's solution containing antibiotics and uterus is closed.
- Aggressive tocolysis done with iv infusion of MgSO₄ to prevent reflex uterine contractions. MgSO₄ can increase risk of pulmonary edema which is managed by use of adrenergic agents. MgSO₄ also increases sensitivity to muscle relaxants hence NM monitoring to be done.
- During postoperative period in the ICU, uterine activity and FHR are continuously monitored by tocodynamometry. Epidural catheter reduces the maternal stress response and early preterm labor.

3. Minimally invasive surgery

- Most procedures can be done with large bore needle under USG or a 5mm trocar has a camera and a port for a laser fibre to coagulate placental vessels in twin, twin transfusion syndrome. In fetoscopy NS irrigation is used inside the uterus. Maternal incision is small and such procedures can be performed decrease local anaesthesia with infiltration of both skin and peritoneum epidural spinal or combined epidural spinal anesthesia can be given.

- Iv sedation given for maternal anxiolysis for fetoscopy because of the neonatal use of surgery and awkward position of the mother. Midazolam fentanyl, remifentanyl or propofol infusion used but deep sedation avoided to prevent aspiration & can lead to pulmonary edemas as the patient is also receiving tocolytics. This can be treated with diuretics. fetoscopy involves manipulation of placenta and umbilical cord and there is no fetal incision because fetal anesthesia is not required.

- Anesthetic management is essentially same for gastroschisis and omphalocele, but knowledge of the associated / anomalies will influence anesthetic decisions.
- Usually diagnosed in utero, overall incidence is 1 : 3000-4000
- Gastroschisis is a defect in anterior abdominal wall usually on the right causing herniation of abdominal contents without a covering sac. It is common on right side, rarely, associated with other congenital anomalies but does have an increased incidence of 'prematurity'.
- Omphalocele: it has a 75% prevalence of other defects including cardiac anomalies (VSD is most common), trisomy 21 and Beckwith Wiedeman syndrome (omphalocele, organomegaly, macroglossia and hypoglycemia). Epigastric omphalocele associated with cardiac and lung anomalies. Hypogastric omphalocele are associated with exstrophy of bladder and other genitourinary anomalies.
- Antenatal diagnosis by ultrasound can be followed by elective cesarean section at 38 weeks and immediate surgical repair to be done.

Preoperative care

1. The exposed area of viscera must be covered with a sterile plastic bag or film to limit evaporative heat loss from exposed bowel.
2. Fluid and electrolytes deficit corrected aggressively.
3. Hypoglycemia should be corrected slowly with a glucose infusion (6-8mg/kg/min). Severe rebound hypoglycemia may occur after bolus dosage of glucose.
4. Decompress the stomach using NGT.
5. A full cardiology assessment should be performed.
6. Two iv cannulae to be put in upper arms for maintenance and volume replacement.

Intra operative management

- Pre oxygenation followed by awake or rapid sequence intubation is preferred with or without muscle relaxant.
- N₂O should be avoided to prevent further bowel distension.
- Muscle relaxation is required for replacing the bowel into the abdominal cavity.
- IPPV with combined volatile narcotic techniques are most commonly used.
- Routine monitors arterial catheters and +/- CVP are used.
- A one stage closure (primary closure) is not always advisable, as it can cause an abdominal compartment syndrome. A staged closure with a temporary Dacron-reinforced silasticsilo may be initially necessary, followed by second procedure a few days later for complete closure.
- Suggested criteria for a staged closure include
 - Intragastric or intravesical pressure more than 20cm H₂O,
 - Peak inspiratory pressure more than 35cm H₂O or
 - End tidal carbon dioxide more than 50mmHg
- Third space fluid losses are aggressively replaced with a balanced salt solution and 5% albumin (6-10ml/kg)

Post operative management

- The neonate remains intubated after the procedure and weaned from the ventilator over the next 1 to 2 days in ICU.
- Patient is placed in head up position.
- Fluid loss and infection are major issues, which should be corrected.

Special considerations

- Manual ventilation is useful to assess the effect of replacement of abdominal contents on lung compliance to determine the correct degree of abdominal reduction.

Common congenital heart diseases and anesthetic management of these patients coming for non cardiac surgeries

- Fetal circulation and its transformation into adult type
- Individual cardiac lesions

• Atrial septal defect (ASD) • Ventricular septal defect (VSD) • Patent ductus arteriosus (PDA) • Tetralogy of fallot (TOF)	• Definition • Incidence • Pathophysiology and hemodynamics • Clinical manifestations • Diagnosis • Treatment
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Anesthetic management of patients with CHD coming for non-cardiac surgeries.

- Pre-operative diagnosis, evaluation and preparation
- Premedication and antibiotic prophylaxis
- Anesthetic management
- Post-operative management
- CHD and pregnancy

Fetal and perinatal circulation:

- Knowledge of fetal and perinatal circulation is an integral part of understanding the natural history, Pathophysiology and the approach to anesthetic management patients with CHD.

Fetal circulation:

Is a parallel circulation and has 4 low resistant shunts

- Placenta
- Ductus venosus
- Foramen ovale
- Ductus arteriosus

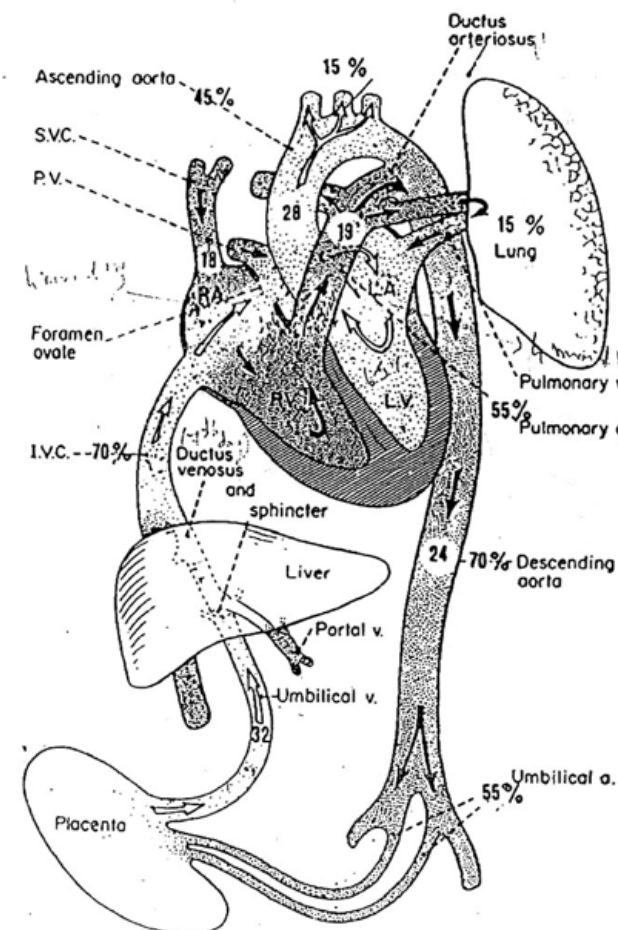
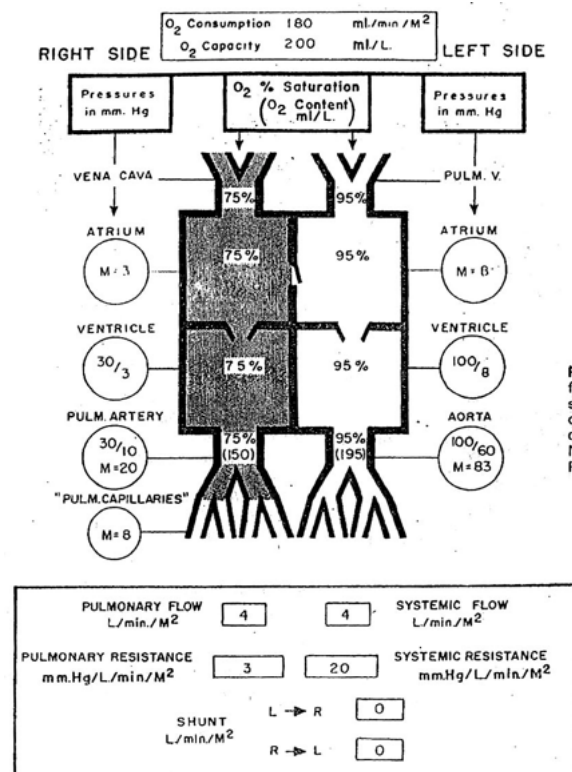


Figure 8-1. Diagram of the fetal circulation showing the four sites of shunt: placenta, ductus venosus, foramen ovale, and ductus arteriosus. Intravascular shading is in proportion to oxygen saturation, with the lightest shading representing the highest PO_2 . The numerical value inside the chamber or vessel is the PO_2 for that site in mm Hg. The percentages outside the vascular structures represent the relative flows in major tributaries and outlets for the two ventricles. The combined output of the two ventricles represents 100%. a, artery; IVC, inferior vena cava; LA, left atrium; LV, left ventricle; PV, pulmonary vein; RA, right atrium; RV, right ventricle; SVC, superior vena cava; v, vein. (From Guntheroth WG, et al: *Physiology of the circulation: Fetus, neonate and child*. In Kelley VC [ed]: *Practice of Pediatrics*, vol 8. Philadelphia, Harper & Row, 1983.)

Changes in circulation after birth:

- The primary change in circulation after birth is a shift of blood-flow for gas exchange from the placenta to the lungs.
- Placental circulation disappears and the pulmonary circulation established.


Etiology of CHD:

Etiology of majority of CI-ID is not known, advances in molecular biology have provided new understanding of the genetic basis of CHI). Hereditary plays an important role in the etiology of CHD.

Sexes are equally affected though in individual lesions there may be predominance of one sex over the other, but in general.

Right, sided lesions → Female

Left sided lesions → Male

Genetic and chromosomal aberrations are also known to predispose to CF-1D.

- **CATCH 22:** (CHD, abnormal facies, thymic hypoplasia, cleft palate and hypocalcemia) due to defect in chromosome 22.
- **VATER AND VACTERL SYNDROMES**
- Pierre robin syndrome, Treacher Collins syndrome, Goldenhar syndrome, Nagars syndrome.

Extra-cardiac anomalies associated with cardiac lesions:

Extra-cardiac anomalies	Most likely congenital cardiac lesion
Abnormalities of forearm like absence of radius or ulna	Ventricular septal defect
Syndactyly and polydactyly	Ventricular septal defect
Down syndrome (Mongolism)	Atrial septal defect of the endocardial cushion type
Arachnodactyly	Atrial septal defect
Turner syndrome	Coarctation of aorta, pulmonic stenosis and aortic stenosis
Ellis-van Creveld syndrome	Atrial septal defect, single atrium
Rubella syndrome	Patent ductus arteriosus and/or pulmonic stenosis
Moon facies and hyperaldosteronism	Pulmonic stenosis
Holt Oram syndrome	Familial atrial septal defect
Marfan's syndrome	Aortic or pulmonary artery dilation
Hurler syndrome	Mitral or aortic regurgitation
Trisomy 13-15	Ventricular septal defect
Trisomy 17-18	Ventricular septal defect, patent ductus arteriosus

- Environmental factors: like high altitude may predispose higher incidence of PDA and ASD.
- Alcohol and drug abuse (thalidomide) during pregnancy
- DIABETIC mother
- Infections: congenital rubella (PDA and PS)
- Metabolic: idiopathic Hypercalcemia

Classification of CHD:

CHD have been traditionally classified into CYANOTIC and ACYANOTIC heart defect.

Defect	Incidence %
Acyanotic defect	
• Ventricular septal defect	35%
• Atrial septal defect	9%
• Patent ductus arteriosus	8%
• Pulmonary stenosis	8%
• Aortic stenosis	6%
• Coarctation of aorta	6%
• Atrioventricular septal defect	3%
Cyanotic defects	
Tetralogy of fallot	5%
• Transposition of great vessels	4%
• Tricuspid Atresia	

Depending upon the Pathophysiology of the defects CHD are classified as:

1. Simple shunts Restrictive
 Nonrestrictive
 2. Obstructive lesions
 3. Complex shunt lesions (shunt + obstruction)
- Simple shunts: These are lesions with an abnormal communication between systemic and pulmonary circulation.
 - Ex. ASD, VSD, PDA
 - The amount of blood flow across the shunt depends upon the size of the orifice and the relative difference in the PVR and SVR.

Restrictive (non dependent):

- Small defects, with pressure gradient across the defect and flow largely fixed by orifice size.
- Changes in downstream impedance, to flow have little influence (i.e. changes in SVR & PV). Ex. small ASD, VSD

Nonrestrictive (Dependent):

- Large defects with very little or no pressure gradient across the defect and shunt flow is largely dependent on the downstream impedance to flow (dependent shunts). Ex: Large ASD, VSD

Obstructive lesions:

- May be right sided (PS) or left sided (AS)
- May be valvular, subvalvular or Supravalvular, Dynamic Fixed

Complex-shunt lesions

- Simple shunts + obstructive lesions Ex: Tetralogy of fallot

Classification of CHI by their effect on blood flow:

- Volume overload of the ventricle or atrium resulting in the increased pulmonary blood flow.
 - ASD, VSD, PDA, endocardial Cushion defect
- Cyanosis resulting from obstruction to PBF
 - TOF, tricuspid atresia, pulmonary stenosis
- Pressure overload of the ventricles
 - AS, coarctation of aorta, PS

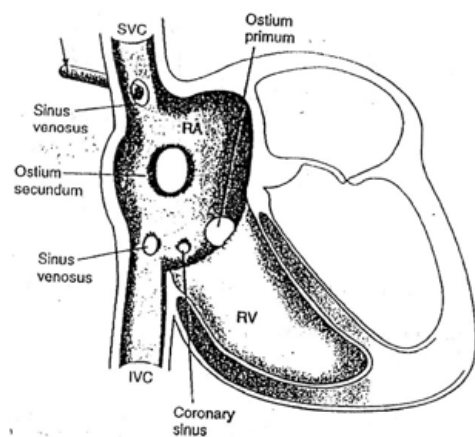
- Cyanosis due to common mixing chambers
 - Total anomalous venous return
 - Truncus arteriosus
 - Double outlet right ventricle
 - Single ventricle
- Cyanosis due to separation of the systemic and pulmonary circulation
 - Transposition of the great vessels

Atrial septal defect (ASD):

- Is an abnormal communication between the 2 atria.
- ASD occurs as an isolated anomaly in 5-10% of all congenital heart defects, and 30-50% of children with CHDs have ASD as part of the cardiac defect.
- More common in females with a ratio of 2:1

Pathology:**Types:**

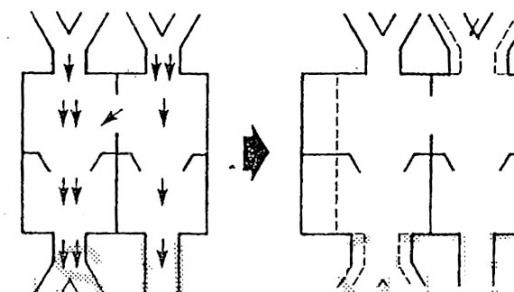
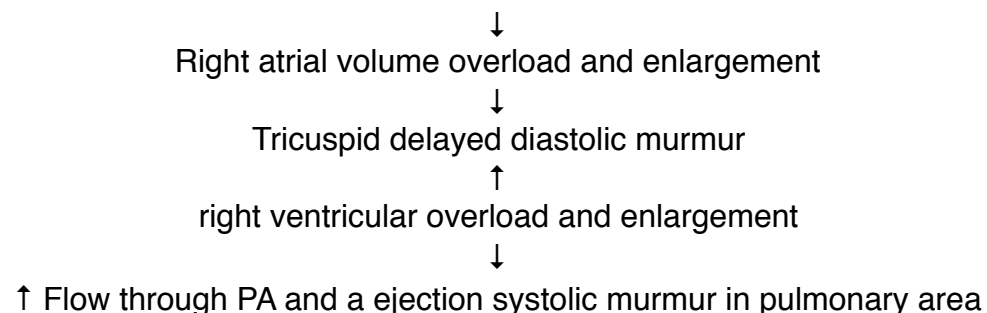
1. Septum secundum type
2. Septum Primum type
3. Sinus venosus Superior venaecaval type
 IVC type
 rarely a coronary sinus type

**Pathophysiology and hemodynamics of ASD:**

Physiologically atrial septal defect results in leaking of oxygenated blood from the left to right atrium at a minor pressure difference between the two atria.

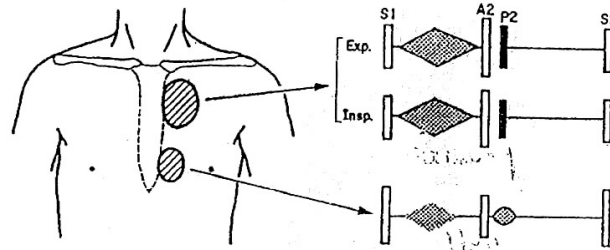
In an ASD the magnitude of the L-R shunt is determined by the size of the defect and the relative compliance of the right and left ventricle.

L → R shunt through ASD

**Clinical Picture:**

- Children with ASD are generally asymptomatic or mild effort intolerance, with dyspnea on exertion, frequent chest infections may be the only symptoms with relatively slender body build (< 10th percentile).

Auscultation:



- A widely split and fixed S_2 with delayed and accentuated P2 .
- A ejection systolic murmur in pulmonary area.
- Delayed diastolic murmur in tricuspid area.
- Typical auscultatory findings may be absent in infants with large ASD.
- Severity of ASD is directly proportional to intensity of the 2 murmurs and the cardiac enlargement.

Natural history prognosis and complications:

- All most all ASD of < 3mm closes spontaneously by 1 ½ years of age.
- ASD of > 8mm rarely closes spontaneously.
- A large untreated ASD may lead to CHF and pulmonary hypertension in adults who are in their 20s and 30s.
- Atrial arrhythmias can occur.
- SABE doesn't occur in children with isolated ASD.
- Cerebrovascular accident from paradoxical embolization through an ASD is a rare complication.

Diagnosis:

ECG:

- shows right axis deviation and RVH
- RBB with rsR pattern in V1

Chest x-Ray:

- Cardiomegaly with right sided enlargement
- Prominent PA segment and ↑ pulmonary markings with plethoric lung fields.

Echocardiography and color Doppler studies:

- Shows position and size of the defect
- Reveals characteristic flow patterns and direction of the shunt.

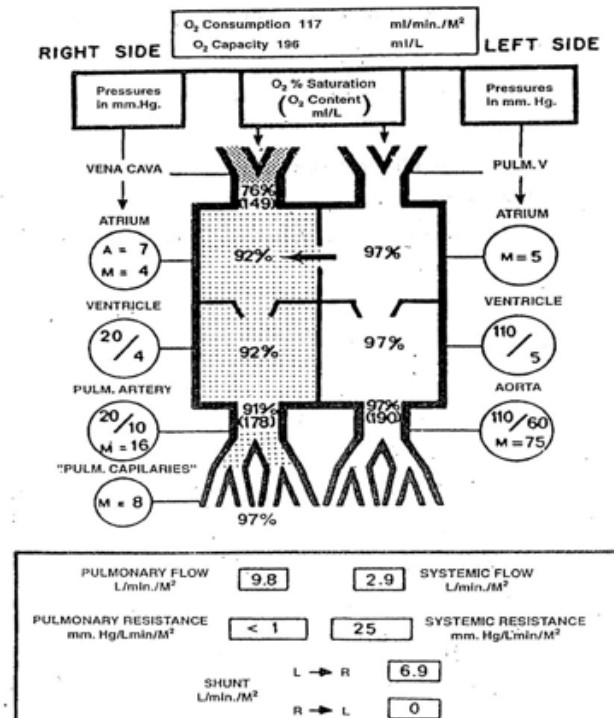


Figure 23-15. Catheterization findings in a patient with an ASD. (From Nadas AS, Fyler DC: Pediatric Cardiology. Philadelphia, WB Saunders Company, 1972.)

Treatment:**Medical management**

- Treatment of chest infection.
- Prophylaxis for SABE if associated with other defects.
- In infants with CHF, medical management is recommended because of its high success rate and possibility of percutaneous closure of the defect.

Surgical treatment:**Indications:**

- Not responding to medical management
- QP: QS $\rightarrow$ 1.5:1

Contraindications

- 11 PVR (> 10 units m^2 or > 7 units / m^2 with vasodilators) and development of PVOD
- Timing of surgery: usually until 3-4 years of age because of high rates of spontaneous closure.
- Procedure: Traditionally repaired through a midsternal incision under CPB by either a single suture of Teflon graft.
- Nonsurgical closure; for secundum ASD with an adequate septal rim (< 20 mm)
 - Ex: 1. Clamshell device
 - 2. Angel wings ASD device
 - 3. Cardio-selective device

Advantage being rapid recovery with < 24 hr hospital stay, no risk of thoracotomy.

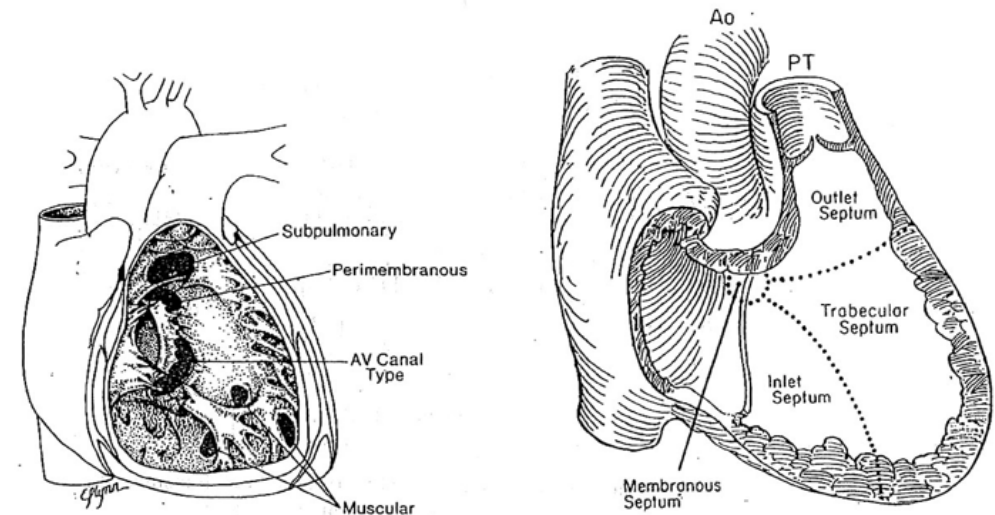
- Complications: CV accidents and arrhythmias in immediate post operative period.
- MR may occur in patients who have undergone repair of an ASD primum.

Ventricular septal defect (VSD):

- Is a communication between the 2 ventricles.
- VSD is the commonest CHD and accounts for about 30-35% of all CHD.

Pathology:**Types of VSD:**

- Perimembranous
- Inlet
- Outlet (infundibular)
- Muscular



- Anatomically 90% of VSD are located in the membranous septum with variable extension into adjoining muscular part of the septum.
- May be multiple and the defect may vary in size ranging from tiny defects without hemodynamic significance to large defects with accompanying CHF and pulmonary HTN.

Pathophysiology and hemodynamics

- AVSD results in shunting of oxygenated blood from the left to right ventricle (Acyanotic)
- The magnitude of the shunt is determined by the size of the shunt and level of PVR and not the location of the defect.
- With a small VSD, a large resistance to L-R shunt is offered at the defect and the shunt does not depend on the level of PVR.
- With large VSD, the resistance offered by the defect is minimum and L-R shunt depends on the level of PVR.

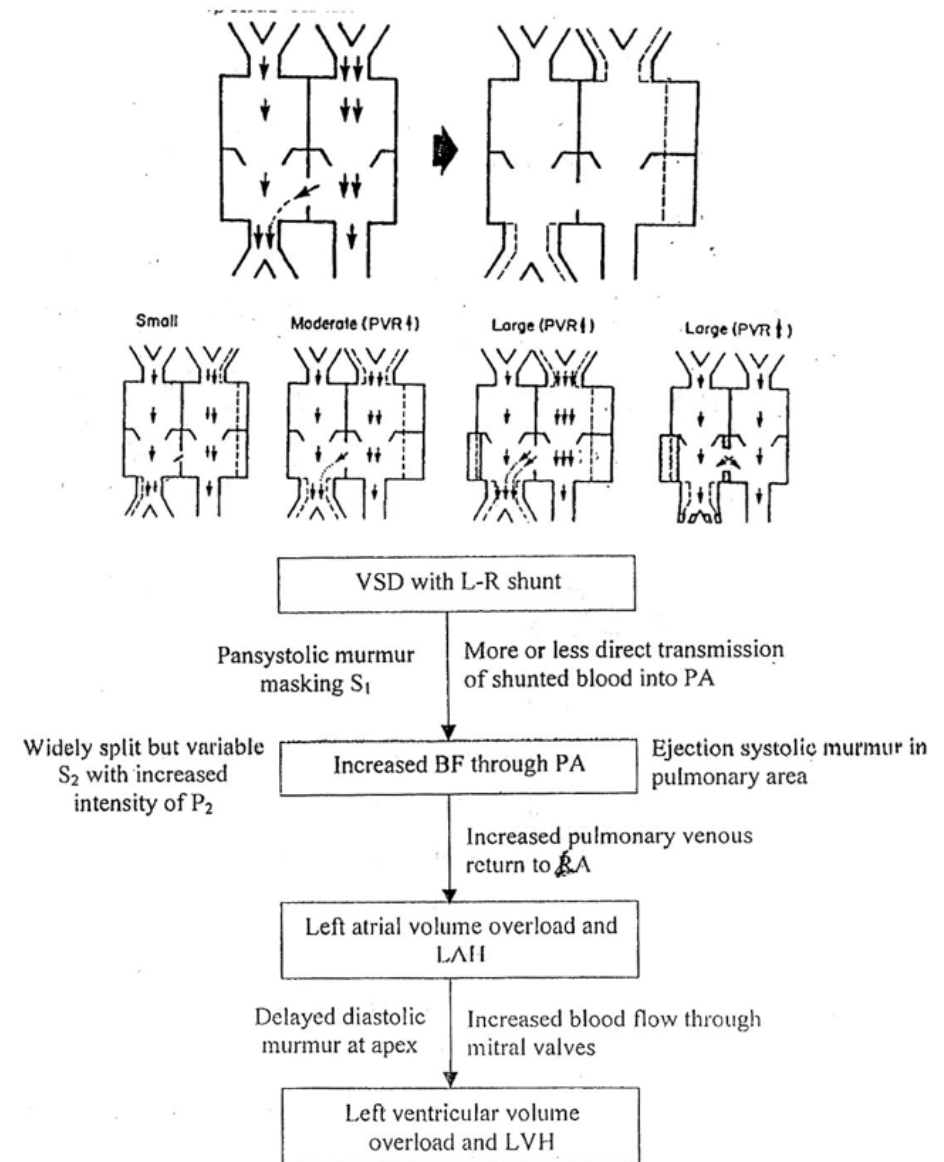
CLINICAL MANIFESTATIONS:

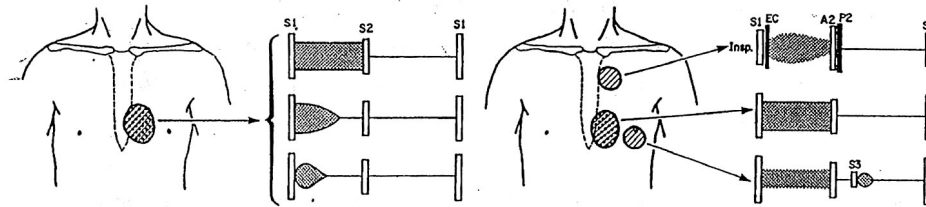
History: with a small VSD, the child is asymptomatic with normal growth and development.

- With moderate to large VSD, delayed growth, and development, decreased exercise tolerance, repeated pulmonary infections and CHF are relatively common in infancy.
- With long standing cases of large VSD pulmonary HTN, history of cyanosis and decreased level of activity may be present.

On physical examination:

- With large VSD, signs of CHF may be present.


Pathophysiology and hemodynamics

On auscultation:

- Murmurs: a pan-systolic murmur due to shunting of blood across the defect, which starts early in systolic and masks S1.
- Ejection systolic murmur due to increased PBF.
- Delayed diastolic murmur at the apex.
- S2 is widely split but varies with respiration and intensity of P2 is increased.

Natural history, prognosis and complications of VSD:

- Spontaneous closure occurs in 30-40% of patients with small membranous and muscular VSDs during first 6 months of life.
- CHF develops in infants with large VSDs, but usually not until 6-8 weeks of age.
- Pulmonary vascular obstructive disease may begin to develop as early as 6-12 months of age in patients with large VSDs. But the resulting R-L shunt usually does not develop until the teenage years.
- Infundibular stenosis may develop in some infants with large defects and result in ↓ in the magnitude of L-R shunt (i.e. Acyanotic TOF) with occasional production of R-L shunt.
- Infective endocarditis rarely occurs.

Diagnosis:**ECG**

- In small VSD, ECG is normal
- In moderate VSD LVH with occasional LAH may be seen.
- In large VSD, ECG shows combined VH with or without LAH.
- With development of pulmonary vascular obstructive disease (PVOD), ECG shows only RVH.

Chest x-ray

- Cardiomegaly of varying degrees is present and involves LA, LV and RV (some times), pulmonary vascular markings increase. The degree of Cardiomegaly and the I in PV markings directly related to the magnitude of L-R shunt.
- With development of PVOD, the main PA and the hilar pulmonary arteries enlarge, but the peripheral lung fields are ischemic and the heart size is usually normal.

Echocardiography:

- 2 dimensional and Doppler echo can identify the number, size and exact location of the defect and to estimate the PA pressure and identify other associated defects and estimate the magnitude of the shut.

Cardiac catheterization:

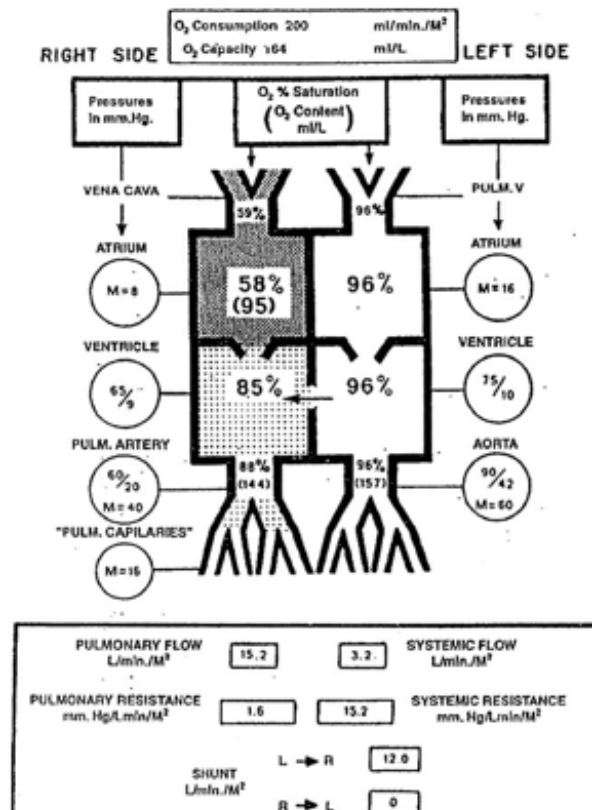


Figure 23-17. Catheterization findings in a patient with a VSD and PA hypertension. (From Nadas AS, Fyler DC: Pediatric Cardiology. Philadelphia, WB Saunders Company, 1972.)

Management:

Medical management:

- Treatment of CHF → with digoxin and diuretics for 2-4 months to see if the growth failure can be improved, frequent feedings with high calorie formulas either by Naso-gastric tube or oral feeding may help and correction of anemia if present with oral iron therapy.
- Maintenance of good dental hygiene and prophylaxis against SABE.
- Nonsurgical closure of selected muscular VSDs is possible using "UMBRELLA" device.
- PA bonding as a palliative procedure (rarely)

Surgical management:

Indications:

- CHF in infancy not responding to medical management with first 6 months of age.
- After 1 year of age, QP: QS of at least 2:1 indicates for surgical closure regardless of pulmonary artery pressure.

Contraindicated:

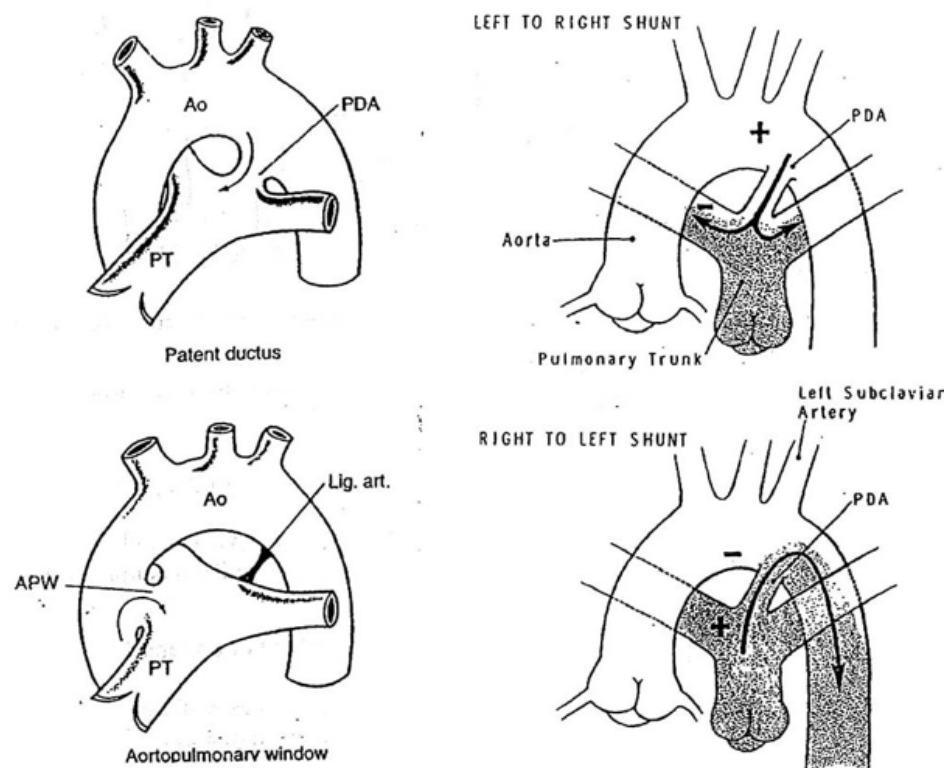
- With the development of PVOD and reversal of shunt
- Direct closure of the defect with a use of DACRON patch, ↓ CPB and or deep hypothermia preferably carried out through and RA approach or right ventriculotomy.

Complications:

- Right and left ventricular failure.
- Complete heart block.
- RBB
- Residual VSD
- Cerebrovascular accidents rarely

Patient ductus arteriosus (PDA):

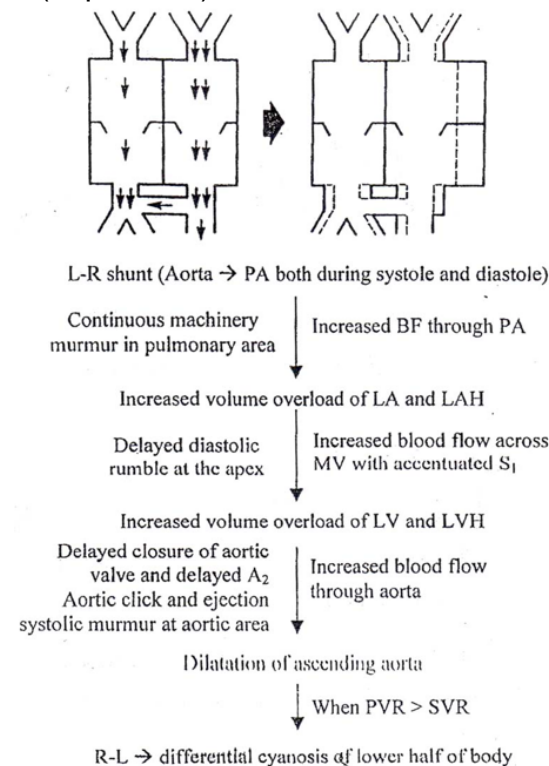
- PDA is an abnormal communication between PA and the descending aorta after birth.
- It is present in fetal life in all, but closes both functionally and anatomically after birth.
- PDA occurs in 5-10% of CHD, excluding premature infants.
- More common in females with a ratio of 3:1
- More common in children born at high altitudes.

Pathology:


- There is a persistent patency of a normal fetal structure between left PA and descending aorta, that is 5-10mm distal to the origin at left subclavian artery.
- Usually cone shaped with a small orifice to PA which is restrictive to flow.
- Duct may be short or long, straight or tortuous.

Pathophysiology and hemodynamics:

- Hemodynamics of PDA is similar to those of VSD.
- The magnitude of L-R shunt is determined by the resistance offered by the ductus (diameter, length, tortuosity) when the duct is small (nondependent) and depend upon level of PVR when ducts is large (dependent).

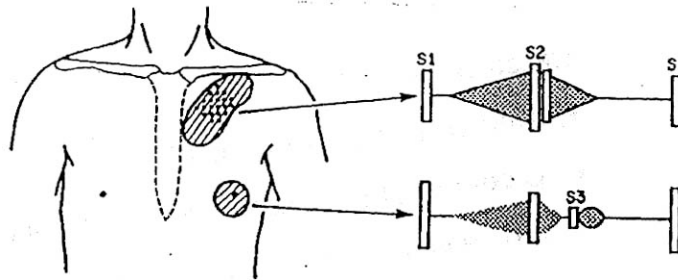


Clinical manifestations:**History:**

- Patients are usually asymptomatic when the duct is small, a large shunt PDA may cause recurrent LRT, atelectasis and CHF accompanied by poor weight gain.

On physical examination:

- Tachycardia and exertional dyspnea in children with large shunt PDA.
- With chronically elevated PA pressure results in development of PVOD and reversal of shunt ($R \rightarrow L$) leading to differential cyanosis (lower half of the body).
- Hyperactive pericardium with systolic thrill at upper left sternal border.
- Bounding peripheral pulse with wide pulse pressure.

**Auscultation:**

- $S_1 \rightarrow$ Accentuated
- $S_2 \rightarrow$ normally split
- $S_3 \rightarrow$ small L – R shunt

Murmurs:

- Shunt murmur $\rightarrow$ continuous machinery murmur
- Flow murmurs
- Delayed diastolic murmur in mitral area
- Aortic ejection systolic murmur

Assessment of severity:

- Heart size
- 3rd heart sound (S_3) (mild)
- Delayed diastolic murmur large
- Wider the pulse pressure larger is the L – R shunt

Natural history, prognosis and complications:

- Unlike PDA in premature infants, spontaneous closure of a PDA does not usually occur in full term infants. This is because PDA in full term infants results from a structural abnormality of the ductal smooth muscle cells rather than decreased responsiveness of premature ductus to oxygen.
 - CHF and recurrent pneumonia if shunt is large.
 - Subacute bacterial endocarditis.
 - Aneurysm of PDA and rupture is a rare complication.

Diagnosis:**ECG:**

- A normal ECG or LVH with small to moderate PDA
- CHV with large shunt PDA
- Only RVH with the onset of PVOD

Chest-x-ray:

- Normal with a small shunt PDA
- Cardiomegaly of varying degrees occurs with enlargement of the LA, LV and ascending aorta, with increased pulmonary vascular markings.
- With PVOD, the heart size is normal with marked prominence of PA segment, hilar vessels and peripheral lung fields are ischemic.

Echocardiography:

- 2 dimensional and Doppler echocardiography can help in assessing the size, functional information.
- The dimensions of LA, LV provide indirect assessment of the magnitude of L-R shunt.

Management

- If diagnosis is made within 2 weeks and in premature infants.
- Indomethacin 0.1 mg/kg/dose orally 12th hourly for 3 doses.
- But not useful in term infants.
- Medical management of CIIF with digoxin and diuretics.
- SAGE prophylaxis.
- Non surgical closure: with stainless coils for PDA of up to 5 mm in length.
- Surgical closure: anatomic existence of a PDA, regardless of its size is an indication for surgery.

Timing:

- Between 6 months to 2 years age in premature infant and soon after the diagnosis is made in older children.

Procedure:

- Ligation and division of PDA through left posterolateral thoracotomy without cardiopulmonary bypass (CPB).
- Recently Video assisted thoroscopic surgery (VATS) is being used.

Complications:

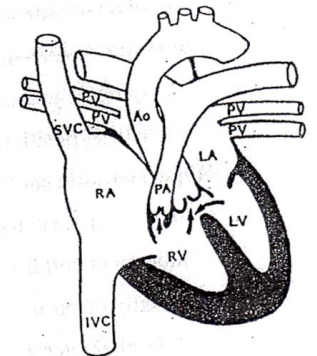
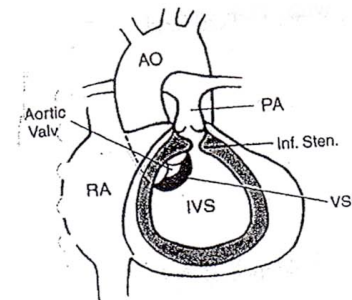
- Injury to recurrent laryngeal nerve, left phrenic nerve and thoracic duct.
- Re-opening of the duct is rare.

Tetralogy of fallot (TOF):

TOF is the most common cyanotic CHD accounting for about 10% of all CHDS.

Anatomically TOF consists of

- A large VSD
- Right ventricular out flow obstruction
- Dynamics
- Fixed RHV
- Overriding of aorta (25%)

**Pathophysiology and hemodynamics:**

A large VSD with right ventricular outflow obstruction

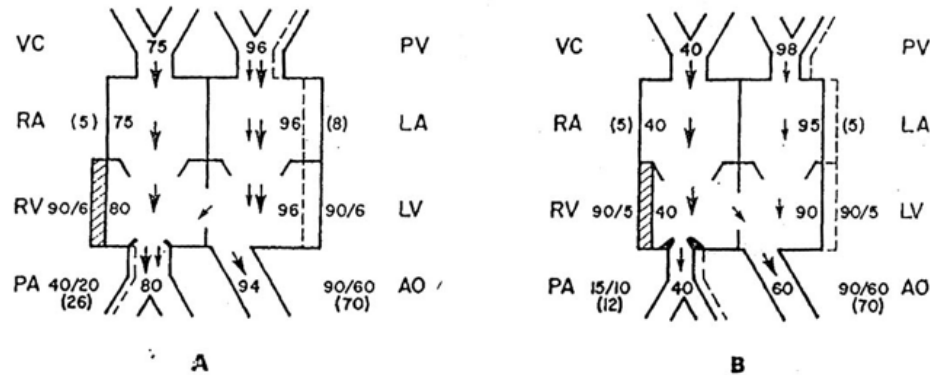
↓ Continuous murmur in large L – R shunt
concentric RHV and RV pressure over load



Reversal of shunt and decreased pulmonary artery blood flow



cyanosis


CLINICAL MANIFESTATIONS:
History:

- A heart murmur is audible at birth.
- Most patients are symptomatic with cyanosis at birth or shortly thereafter.
- Dyspnoea on exertion, squatting or hypoxic spells develop later in life.
- Infants with Acyanotic TOF may be asymptomatic or shows signs of CHF due to large L-R shunt.
- Severe cyanosis soon after birth is seen in infants with TOF and pulmonary atresia.

Physical examination:

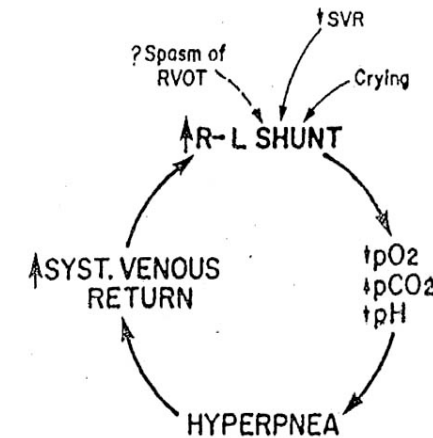
- Varying degree of cyanosis; tachypnea and clubbing.
- Squatting position for relief of Dyspnoea.

Hypercyanotic spells - Tet Spells

- It is seen specially ii) first 2 years of life with a peak incidence at 2-4 months in children with TOF.
- Usually occur in the morning, after crying, feeding or defecation.
- It is characterized by a paroxysm of hyperapnoea, arterial desaturation, associated with worsening cyanosis and even loss of consciousness, seizures, CV accidents and even death in untreated cases.

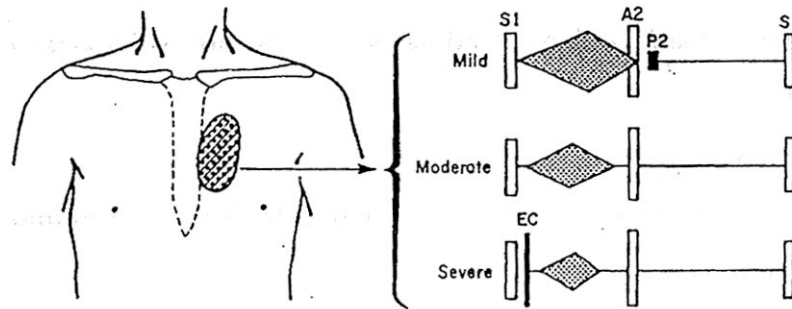
Mechanism:

- Mechanism of hypoxic spell. A decrease in the arterial PO₂ stimulates the respiratory center, and hyperventilation results. Hyperpnoea increases systemic venous return. In the presence of a fixed right ventricular outflow tract (RVOT), the increased systemic venous return results in increased right-to-left (R-L) shunt, worsening cyanosis. A vicious circle is established. SVR, systemic vascular resistance.


Treatment of Hypercyanotic spells:

- Infant should be picked up and held in knee-chest position.
- 100% oxygen supplementation
- Injection morphine 0.2 mg/kg SC or IV.
- Acidosis corrected with sodium bicarbonate 1 mEq/kg IV.
- If not responding to the above measure.
 - Vasoconstrictors → phenylephrine – 0.02 mg/kg/IV
 - Inj ketamine 2 mg/kg IV
 - Inj propranolol – 0.05 mg/kg/KV
 - Inj esmolol – 300-500 µgm/kg/IV

Auscultation:



Sounds:

- S1 is normal
- S2 is single → only A2 is heard
- P2 being soft, delayed and inaudible.

Murmurs:

Shunt murmur: usually absent

Flow Murmur: Pulmonary ejection systolic murmur (intensity inversely proportional to the severity of right ventricular out flow tract obstruction)

Aortic ejection click.

Natural history, prognosis and complications:

- Infants with Acyanotic TOF gradually become cyanotic; patients who are already cyanotic become more cyanotic as a result of the worsening condition of infundibular spasm or pulmonary stenosis.
- Polycythemia secondary to chronic hypoxemia (Hct > 65%) may lead to systemic thromboembolism (renal, CNS)
- Relative iron deficiency anemia.
- Growth retardation
- Brain abscess and anoxic infarction in CNS and hemiplegic are not uncommon.
- SABA
- Coagulopathy in long standing cases with chronic hypoxemia

DIAGNOSIS:

Chest-X-ray:

- Heart size is normal or even small with decreased pulmonary vascular markings.
- Boot shaped heart (Coeur en sabot)
- Right aortic arch is 25% of patients.

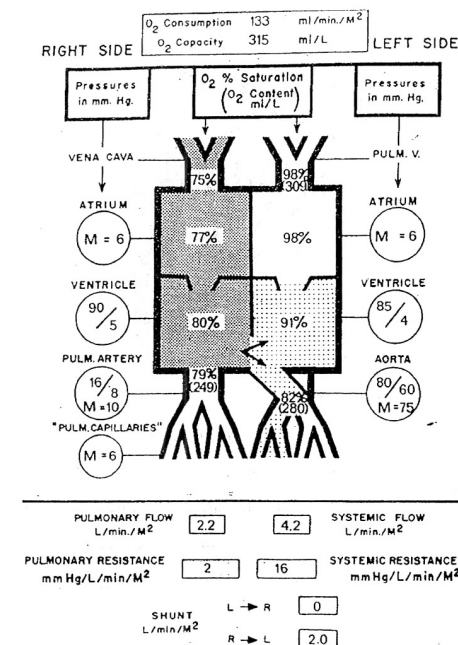
ECG:

- RVH with right axis deviation
- 'P' pulmonary maybe seen
- CVH in Acyanotic TOF (large L-R shunt)

Echocardiography:

- 2 dimensional and Doppler echo studies can snake the diagnosis and quantative the severity of TOF.

Cardiac catheterization:



Management:**Medical management:**

- Medical management of CHF and other complications.
- Maintenance of good dental hygiene with SABA-prophylaxis.
- Correction of relative iron deficiency anemia.

Surgical management:

- Palliative shunt surgery
- Conventional repair surgery

Palliative shunt procedure:**Indications:**

- Neonates with TOF and pulmonary atresia
 - Infants with hypoplastic pulmonary annulus which requires a transamural patch for complete repair.
 - Children with hypoplastic pulmonary artery stenosis.
 - Severely cyanotic infants < 3 months of age.
-
- Infants of 3-4 months with unsuccessful medical management of hypoxic spells.

Types:

- Blalock - Taussig shunt → between subclavian artery and the ipsilateral pulmonary artery.
- Gore-Tex interposition shunt (modified B-T shunt)
- The Waterston shunt (ascending aorta and right pulmonary artery)
- Potts operation (between descending aorta and left pulmonary artery)

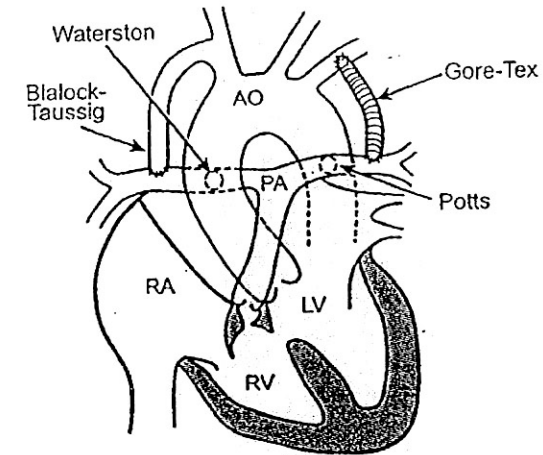


Figure 14-20. Palliative procedures that can be performed in patients with cyanotic cardiac defect with decreased pulmonary blood flow. The Gore-Tex interposition shunt (or modified Blalock-taussig shunt) is the most popular systemic-to-pulmonary artery shunt procedure. AO, aorta; LV left ventricle; PA, pulmonary artery; RA, right atrium; RV, right ventricle.

CONVENTIONAL REPAIR SURGERY**Indications and timing**

- Symptomatic infants who have favorable anatomy of right ventricular out flow tract, PA, without coronary artery anomalies may have primary repair at any time after 3-4 months of age.

Procedure:

- Total repair of the defect is carried out under CPB and circulatory arrest.
- Procedure includes patch closure of VSD and widening of the right ventricular outflow tract by resection of the infundibular tissue and placement of a fabric patch.

Complications:

- Bleeding problems in postoperative period, especially in polycythemia patients.
- Right and left ventricular failure.
- Complete heart block, RBB.
- Residual VSD.
- Pulmonary valve regurgitation.

ANESTHETIC MANAGEMENT OF PATIENT WITH CHD COMING FOR NON CARDIC SURGERIES

Approach to the patient with CHD is the same whether the procedure is cardiac or non cardiac.

Pre-operative preparation:

- Successful anesthetic management of patients with CHD is based on complete and accurate pre-operative assessment, adequate pre-operative preparation and early post-operative management.
- The approach to CHD patients must emphasize cardiac concerns but the basic tests of pediatric anesthesia cannot be overlooked because they form the formulation on which more complex interventions are built.
- In general, patients with CHD requiring non-cardiac surgery may present in the following situations.
 - Before the congenital lesion is diagnosed.
 - After the diagnosis but before surgical intervention.
 - After surgical palliation
 - After surgical correction
 - Inoperable cardiac lesions
- The anesthesiologist caring for the patient with CHD should understand.
 - The patient specific intracardiac and extra-cardiac defects and resulting pathophysiology of the hemodynamic changes.
 - The sequels of surgery or residual defects after the surgery.
 - Cardiovascular effects of the anesthetic agents to be administered.

Pre-operative diagnosis and evaluation:

Goals of pre-operative evaluation are as follows

- To develop a detailed understanding of the patient cardiac anatomy and its physiologic consequences.
- Ascertain anesthetic problems related to the patient non-cardiac medical conditions or concomitant congenital syndromes.
- Educate the patient and family concerning the expected course of the planned anesthetic.
- Reduce anxiety and fear relating to the operative procedure through psychological preparation of both patient and family.
- In addition to the routine history about the patient, anesthesiologist should be particularly concerned about the
 - Functional status of the patient
 - Details about surgery to be taken & kind its complications.

Physical examination:

- Apart from the regular examination, airway assessment requires special attention in patients with CHD.
- For symptoms arising from the compression of major airways.
- Anticipate difficult airway for tracheal intubation in patients with facial dimorphic changes such as
 - Large tongue, hypoplastic mandible and teeth
 - Patients with Down's syndrome are more prone for cervical dislocation.
- Special attention should be paid to the cardiopulmonary status of the patient and any abnormalities that pertain to the planned surgical procedure.

Symptoms and signs of cardiac-failure in a neonate and infant:

1. Failure to thrive - poor feeding, diaphoresis
2. Increased work of breathing: tachypnea, wheezing, grunting, flaring of ala nasi and chest wall retraction.
3. Altered cardiac output: tachycardia, gallop rhythm – Cardiomegaly, hepatomegaly, cyanosis.

4. Pulse and BP should be recorded in all the 4 extremities as patients with CHD are at a increased risk for an abnormal origin of the arterial supply to the extremities and for unsuspected stenosis of peripheral vessels.
5. Special attention should be paid for the presence of upper and lower respiratory tract infections and should be treated promptly.

Laboratory studies:**Hemoglobin (Hb%)**

- For relative iron deficiency anemia due to polycythemia.
- Chronic hypoxemia in CHD patients lead to the development of Hb% > 20gm% and such high levels of hematocrit (>65%) results in hyperviscosity and peripheral sludging of blood leading to reduced tissue perfusion, acidosis, ↑ level of 2,3 DPG and right ward shift of oxyhaemoglobin dissociation curve.
- If the hematocrit > 65%, pre-operative isovolumic exchange transfusion or RBC pheresis is considered.

Coagulation profile

- Patients with CHD especially with polycythemia are at a risk of having coagulation abnormalities due to
 - Reduced number of functional platelets.
 - Increased fibrinolysis
 - Decreased concentration of clotting factors.
- Hence all the patients with CHD requires a detailed coagulation profile evaluation which-includes
 - Prothrombin time (PT)
 - Activated partial thromboplastin time (APTT)
 - Fibrinogen levels
 - Platelet count

Serum electrolytes

- Pre-operative electrolytes evaluation, especially for K⁺ in patients on digitalis and diuretics and prompt treatment of the imbalance.

Blood glucose

- Pre-operative blood glucose should be evaluated and monitored as the patients under stress and with CHD are more likely to acquire hypoglycemia.

Blood gases: ABG

- For patients with respiratory compromise and severe cyanosis.
- A PaO₂ of 30-40 mm of Hg and SPO₂ of >70% is an indication for the development of metabolic acidosis.

Renal function test

- Blood urea
- Serum creatinine

Chest x-ray:

- For cardiac size, signs of pulmonary congestion or pulmonary vascularity.
- X-ray may give a clue of any underlying airway problem including major airway compression from the enlarged pulmonary vessels.

Electrocardiogram (ECG):

- For information about rhythm, volume and pressure burdens on the ventricles due to anatomic defect.
- The pre-operative ECG should be evaluated for pre-existing arrhythmias and as a baseline for intraoperative and postoperative wave forms.

Echocardiography and color Doppler studies:

- For the anatomic location of the defect
- To know the direction and magnitude of the shunts and pressure gradients.
- To access the wall motion abnormalities.
- For intraoperative and postoperative assessment of the cardiac function.

ECG gated Magnetic resonance imaging and angiography (MRI/A):

- MRA has emerged as an important diagnostic modality in the evaluation of cardiovascular system.
- Provides an excellent 3 dimensional images to qualitatively assess valve and ventricular function, and to quantify flow, ventricular volume, mass and ejection fraction.

Cardiac catheterization

- Cardiac catheterization remains the gold standard for assessing anatomy and physiologic function in congenital heart disease.
- Important catheterization data for anesthesiologist include
 - Patient response to sedative medications.
 - Pressure and oxygen saturation in all chambers and great vessels.
 - Location and magnitude of intra- and extracardiac shunt (QP:QS).
 - Pulmonary vascular resistance, systemic vascular resistance,
 - Chamber size and function.
 - Valvular anatomy and function.

PREOPERATIVE ORDERS:
Fasting ORDERS:

- Standard guidelines for the fasting interval based on the age must be adjusted according to the individual needs.
- Preoperative dehydration should be avoided especially in children with cyanotic CHD and elevated hematocrit.
- Current recommendations for fasting interval in children and infants are
 - Solids and milk products until 6-8 hrs prior to surgery.
 - Clear liquid until 2 hrs prior to surgery.
 - Breast milk until 3 hrs prior to surgery.

Medications:

- Patients with CHD should receive all cardiac medications till the day of surgery except anticoagulants and diuretics.

Antibiotics: for SABE prophylaxis

Surgical procedures that do & do not require endocarditis prophylaxis

Endocarditis prophylaxis recommended	Endocarditis prophylaxis not recommended
Dental procedures known to induce gingival or mucosal bleeding, including professional cleaning	Dental procedures not likely to induce gingival bleeding, such as simple adjustment of orthodontic appliances or fillings above the gum line.
Tonsillectomy or adenoidectomy	Injection of local intraoral anesthesia (except intraligementary injections)
Surgical operations that involve intestinal or respiratory mucosa	Shedding of primary teeth
Bronchoscopy with a rigid bronchoscope	Thmpanostomy tube insertion
Sclerotherapy for esophageal varices.	Oral endotracheal intubation
Gallbladder surgery	Cardiac catheterization
Cystoscopy	Endoscopy with or without gastrointestinal biopsy.
Urethral dilatation	Cesarean section
Urethral catheterization if urinary tract infection is present	In the absent of infection for urethral catheterization, dilatation and curettage, uncomplicated vaginal delivery, therapeutic abortion, sterilization procedures, or insertion or removal of intrauterine devices.
Urinary tract surgery if urinary tract infection is present	
Prostatic surgery	
Incision and drainage of infected tissue	
Vaginal hysterectomy	

Table 23-5. Recommendations For Prophylaxis Against Subacute Bacterial Endocarditis

Oral Regimen for Dental, Oral, or Upper Respiratory Tract Procedures
Standard

- Amoxicillin, 50 mg/kg, orally 1 hr before procedure Penicillin-Allergic Patients
- Clindamycin, 20 mg/kg, orally 1 hr before procedure Azithromycin or Clarithromycin, 15 mg/kg, orally 1 hr before procedure

IV/IM Regimen for Dental, Oral, or Upper Respiratory/ Tract Procedures
Standard

- Ampicillin, 50 mg/kg, IV/IM 30 min before procedure Penicillin-Allergic Patients
 - Clindamycin, 20 mg/kg, IV/IM 30 min before procedure
- Regimens for Genitourinary and Gastrointestinal Procedures
- Standard
- Ampicillin, 50 mg/kg, IV/IM + gentamycin, 1.5 mg/kg, 30 min before procedure; 6 hrs later, ampicillin, 25 mg/kg, IM/ IV or amoxicillin, 25 mg/kg, orally
 - Penicillin-Allergic Patients
 - Vancomycin, 20. mg/kg, IV over 1 hr + gentamicin, 1.5 mg/ kg, IV/ IM completed 30 min before procedure
 - Adapted from Dajani AS, Taubert KA, Wilson W: Prevention of bacterial endocarditis. Circulation 96:358-366,1997.

Pre-medication:

- The potential advantages of pre-operative sedative patients with CHD
 - Easy separation from points and less crying.
 - Decreased sympathetic activity
 - Decreased oxygen demand and consumption
 - Decreased requirements of intraoperative anesthetics.
- But this should be balanced against the respiratory depressant effects of the drugs used.
- Thus, premedication should procedure a sedate and cooperative patient plus maintenance of airway reflexes and a smooth induction of anesthesia.

Premedication Recommendations For Children With Congenital Heart Disease Over 1 Year Of Age*

Drug	Dose	Minutes prior to induction to be given
Oral premedicant	Combinations	
Meperidine	3 mg/kg (maximum 100mg)	60-90
pentobarbital	4mg/kg (maximum 100mg)	
Atropine	0.02 mg/kg (maximum 0.4 mg)	
Or		
Meperidine	1.5 mg/kg (maximum 100mg)	30-60
Diazepam	0.15 mg/kg (maximum 10mg)	
Atropine	0.02 mg/kg/ (maximum 0.4 mg)	
Or		
Meperidine	2-3 mg/kg (maximum 100mg)	60
Diazepam	0.1 mg/kg (maximum 10mg)	
Pentobarbital	2-4 mg/kg (maximum 100mg)	
Fentanyl (oral/ transmucosal)	15-20 µg/kg	30-45
Midazolam	0.5-0.75 mg/kg (maximum 5 mg)	20-30
Intramuscular premedicant combinations		
Scopolamine	0.01 mg/kg (maximum 0.4 mg)	60
Pentobarbital	2 mg/kg (maximum 100mg)	
Morphine	0.1 mg/kg (maximum 10mg)	
Atropine	0.02 mg/kg (maximum 0.4 mg)	60
Pentobarbital	2 mg/kg (maximum 100mg)	
Morphine	0.1 mg/kg (maximum 10mg)	
Or		
Midazolam	0.08 mg/kg (maximum 5 mg)	10
Nasal premedication		
Midazolam	0.2-0.3 mg/kg (maximum 5mg)	10
Or		
Ketamine	1-5 mg/kg	5-15
Sufentanil	0.3-3 µg/kg	10
Rectal premedication		
Midazolam	0.3-1.0 mg/kg	20-30

Operating room preparation:

- An operational anesthesia machine with suitable breathing circuit.

- Airway and intubation instruments

Variety of pharmacologic agents

Cardiac resuscitative drugs and a defibrillator.

Equipment for thermal hemostasis

Functional suction apparatus

Venous access:

- Consideration should be given to placement of an IV catheter in all the patients with CHD undergoing any surgical procedure because rapid administration of cardiac medications may be required.
- EMLA cream can be used for painless access especially in pediatric cases.
- The introduction of air bubbles is extremely dangerous in patients with Shunt lesions, precautions must be taken to prevent the inadvertent introduction of venous air.
 - Meticulous preoperative dabbling of all invasive tubing and intravascular monitoring catheters.
 - Allowing free flow of fluid from tubing and intravascular catheters before connection.
 - Ejection of any air from syringe and needle before injecting drug into an IV catheter.
 - Avoiding injection of the last milliliter of fluid from I syringe because of micro bubbles on the plunges.
 - Using IV air traps whenever possible.
 - Positioning syringes vertically before injection to allow air to rise away from the outlet.
 - Never allowing arty central venous catheter to be open to air.
 - Avoiding use of nitrous oxide whenever possible and discontinuing its use if air embolization is suspected.

Monitoring:

Standard monitoring devices for patients with CHD undergoing noncardiac surgeries include.

- Pulse oximetry
- Precordial stethoscope
- NIBP
- ECG
- ETCO₂
- Temperature monitoring
- CVP monitoring
- Urinary catheter
- Transoesophageal echocardiography is reserved for procedure in which patient is placed at risk of embolization arising from the surgical field.
- Interatrial blood pressure, pulmonary artery catheterization and PCWP are reserve for more complex surgeries and in patients with compromised cardio-respiratory status.
- **Thermal stabilization:** Low environmental temperature brings about cutaneous vascular constriction and a significant increase in blood viscosity. Therefore children already polycythemia encountering the combination of peripheral constriction and sludging of blood secondary to viscosity increase, will be predisposed to metabolic acidosis, increased oxygen consumption and cardio respiratory depression. Hence all anti hypothermic actions should be taken.
- **Fluid management:** Fluids must be individualized For each patient based on the patients cardiac physiology, age specific fluid needs, preoperative deficit and expected third space fluid and blood losses associated with the planned surgical procedure. Care should be taken not to fluid overload a child with borderline myocardial dysfunction. The ultimate goal of fluid management is to produce a hemodynamically stable child during the anesthesia while maintaining a urine flow of 0.5-1.0 ml/kg/hr.

- Maintenance of an optimal preload is essential in these patients, ideally guided by CVP monitor.
- In R-L shunt preload must be increased.
In L-R shunt fluid overload must be avoided.

Anesthetic management:

- A variety of pharmacologic and physiologic differences exist for each anesthetic agent and muscle relaxant. The choice of which agent to use for a specific patient is based on NPO status, hemodynamic status, and requirement of the patient determined by cardiac grid, age of the patient, venous access, airway competency, level of anxiety of expertise of the anesthesia.

General anesthesia:

- General anesthesia with tracheal intubation and positive pressure ventilation should be used in all but shortest procedure to avoid the potential complications of transient hypoxia and hypercarbia in patients with reduced pulmonary blood flow and pulmonary hypertension.

Breathing system:

- Jackson Rees circuit (<20kg)
With a fresh gas flow twice the minute ventilation.
- Bain's circuit: 70ml/kg for controlled ventilation
- Closed circuit: 2-4 ltr of fresh gas flow

Induction techniques:

- No drug is absolutely contraindicated, slow and smooth induction of general anesthesia should be done avoiding myocardial depression, excessive vasodilatation and hypotension.
- The technique of induction depends on various factors including age, NPO status, level of anxiety and pathophysiology of anatomic defect, cardio respiratory status of the child, IV access and the expertise of the anesthesiologist.

Inhalation induction:

- Is preferred for children with good cardiac reserve and without an IV access.
- Halothane is used for a awake and steal mask induction in children because of its easy acceptance and minimal effect on PVR and SVR and ability to titrate depth of anesthesia.
- Other agent such as sevoflurane is gaining more popularity as an induction agent in children because of its less myocardial depressant effect.

Nitrous oxide:

- Can be used for induction and maintenance along with other inhalation agents in 50% concentration.
- Nitrous oxide increases SVR and PVR so should be avoided in patients with pulmonary hypertension.

Intravenous (IV) induction:

- Is usually used in patients with significant cardiovascular compromises
- Inj. Thiopentone - 3-5 mg/kg/IV
- Inj. Ketamine -1-2 mg/kg/IV
2-10 mg/kg/IM
- Inj. Midazolam - 0.05-0.1 mg/kg/IV
- Inj. Etomidate -0.3 mg/kg/IV
- High dose narcotic techniques are useful in patients with minimal cardiac reserve and when post-operative mechanical ventilation is planned.
- Inj. Fentanyl - 25-75 µg/kg/IV
- Inj. sufentanyl - 5-20 µg/kg/IV
- A useful combination that can be used in a sick cyanotic CHD child is IV ketamine + IV phenylephrine + IV fentanyl with glycopyrrolate Which maintains the best arterial oxygenation during anesthesia.

Speed of induction:**1) Left-Right shunt**

- L-R shunts have a relative over perfusion of the pulmonary circuit. The blood from lungs which already contains anesthetic agent is recirculated, thereby acquiring additional anesthetic, the result is a higher peak concentration of the anesthetic agent in the blood allowing a more rapid induction with inhalation agent.
- IV induction agents have a slower onset of the action as a result of a delay in reaching the brain during the period of recirculation through the pulmonary circuit.

2) Right-Left shunts

- R-L shunts slow the rate of rise in alveolar concentration of inhalational agents and prolong induction and emergence of anesthesia.
- IV induction should have a more rapid onset of action in patients with limited PBF, since the agent would more rapidly reach the systemic circuit.

MUSCLE RELAXANTS**For tracheal intubation****Inj. Succinylcholine 1-2mg/kg/IV**

- But can cause bradycardia so reserved only for rapid sequence induction.
- Rapid onset and intermediate acting non-depolarizing muscle relaxant such as inj. Rocuronium – 0.06-0.8 mg/kg IV can be used as an alternative.

Other non-depolarizing NMB agents

- Inj. pancuronium -0.1-0.15 mg/kg/IV
- Inj. Vecuronium – 0.07-0.1 mg/kg/IV
- Inj. Atracurium – 0.3-0.5 mg/kg/IV

Maintenance:

- O₂ + N₂O + inhalational agent + muscle relaxants + analgesia + IPPV.
- N₂O is avoided in patients with pulmonary HTN.
- Controlled ventilation is used in all patients. PaCO₂ is maintained between 30-40mm HG.
- In TOF, excessive positive pressure ventilation is avoided as it may lead to increased R-L shunt across the VSD and decreased PaO₂.

Analgesia:

Narcotic analgesics such as

- Inj. Morphine – 0.1-0.2 mg/kg/IV
- Inj. Fentanyl – 1.2 µg/kg/IV
- Inj. Remifentanyl - 1µg/kg/min IV.
- Postoperative mechanical ventilation should be considered in patients with preoperative CCF and in case of prolonged and extensive surgeries involving massive fluid shifts.

Reversal:

- At the end of the surgery the patient is reversed with
- Inj. Neostigmine – 0.05 mg/kg

+

Inj. Glycopyrrrolate – 0.01-0.02 mg/kg

Regional anesthesia:

- Is used whenever possible and suitable.
- But, care should be taken when initiating the surgical blockade, which may cause decrease in SVR and increase R-L shunts resulting in rapid desaturation.
 - Coagulation abnormalities are contraindicated for regional anesthesia.
- Regional anesthetic techniques are useful adjuncts to general anesthesia in patients with CHD. The advantage included
- Decrease requirements of other general anesthetics that might cause delay in extubation.
 - Provision for postoperative analgesia.

POSTOPERATIVE MANAGEMENT:

- Immediate post-operative care of the patients with CHD who has undergone surgery is an important period in the overall sequence of anesthetic and surgical management.
- Patients with CHD are susceptible to deleterious effects of hypoventilation and decreased oxygen saturation. Therefore it is advisable for postoperative oxygen administration.
- As a member of the operating team, it is necessary the anesthesiologist understand and become involved during the immediate postoperative care.
- One important area in which anesthesiologist can aid the recovery of these patients is in the form of providing adequate postoperative analgesia and sedation. The main aim is to reduce cardio-respiratory work by attenuation of stress response.
- Patients with CHD and poor cardio-respiratory reserve, who have undergone major coincident surgeries should be ventilated for some hours and should be under the supervision of an anesthesiologist familiar with the specific cardiac disease.
- Postoperative pain relief is considered as part of the anesthesia plan.
- Regional analgesia such as caudal epidural analgesia in pediatric patients for genitourinary surgery - Inj. bupivacaine 0.25% 1 ml/kg Inj morphine 0.05-0.75 mg/kg
- Epidural analgesia with injection bupivacaine 0.1 % with
 - Inj. fentanyl – 2 µg/ml
 - Inj. morphine – 30 µg/kg/hr
- NSAIDs - provide effective analgesia for mild to moderate postoperative pain. The drugs most commonly used are acetaminophen - 15mg/kg po, Diclofenac - 1-3mg/kg/day PO/PR and ketorolac - 0.5mg/kg.
- Systemic analgesia: patient controlled analgesia (PCA) is a method of giving adult patients small doses of opioids at frequent intervals to minimize the side effects.

CHD And Pregnancy::

- CHD has overtaken RHD as the most common type of heart disease seen in pregnancy women with a L-R shunt tolerate pregnancy, labour, delivery and surgery well ; whereas women with a R-L shunt are associated with a high maternal and fetal mortality. The risk of the fetus is in the proportion to the degree of maternal hypoxemia with a 50% risk of fetal death if maternal oxygen saturation is less than 85%.
- L- R shunts: pregnancy creates an additional burden by increasing blood volume, heart rate and cardiac output. Parturient with small shunts who are asymptomatic prior to pregnancy tolerate this additional stress; whereas those with large shunts develop symptoms of congestive cardiac failure.

Anesthesia consideration:

- Should receive antibiotic prophylaxis for SBE.
- Avoid hypotension which may cause shunt reversal.
- Conditions that increase PVR such as hypoxemia, hypercarbia and acidosis are avoided.
- Avoid aortocaval compression by left uterine displacement.
- Epidural analgesia and anesthesia is appropriate as it brings about minimal changes in vascular resistance.
- R-L shunts: A few women with these lesions reach child bearing age and are associated with high maternal and fetal mortality. Pregnancy results in up to 50% maternal mortality and 80% fetal loss. Pregnancy in these women is not advised. The decreased SVR in pregnancy promotes increased R-L, shunting and cyanosis.

Anesthesia consideration:

- Prime importance is to avoid myocardial depression and to maintain normal SVR, venous return and blood volume.
- Epidural analgesia is chosen as it allows a gradual onset of block. Epidural opioids are used which improve quality of block and allow use of lower concentrations of local anesthesia.
- General anesthesia is preferable during cesarean section as it allows optimal airway management and titration of drugs to maintain vascular resistance and volume.

CONCLUSION:

- Anesthesiologists should be familiar with the anatomy and pathophysiology of the specific CHD, before anaesthetizing these patients, a discussion with the cardiologist prior to anesthesia provides valuable information regarding the status of the individual cardiac pathophysiology and help to anticipate intraoperative problem.
- To goals of anesthesia for these patients with CHD coming for non cardiac surgeries are the maintenance or even improvement of the hemodynamic status in the face of destabilizing surgical manipulation.

ASSESSMENT OF PAIN

- Although self reporting of pain is the gold standard for assessment of the site, nature, and severity of pain, it is not precisely applicable in children below 3 years of age.

Table 1 Responses of infants to pain

Physiological changes	Behavioural changes	Biochemical changes
Increase in: - Heart rate - Blood pressure - Respiratory rate - Oxygen consumption - Mean airway pressure - Muscle tone - Intracranial pressure	Change in facial expression ¹² : - Grimacing - Screwing up of eyes - Nasal flaring - Deep nasolabial groove - Curving of the tongue - Quivering of the chin	Increased release of: - Cortisol - Catecholamines - Glucagon - Growth hormone - Renin - Aldosterone - Antidiuretic hormone
Autonomic changes ^{10 11} : - Mydriasis - Sweating - Flushing - Pallor	Body movements ¹³ : - Finger clenching - Thrashing of limbs - Writhing - Arching of back - Head banging	Decreased secretion of: Insulin

Box 5: Basis of management strategies for infant pain

- Awareness of infants' capacity to perceive pain.
- Sensitivity to situations where infants may experience pain.
- Prevention of pain.
- Assessment of cause and severity of pain.
- Pharmacological interventions.
- Non-pharmacological interventions.
- Modification of techniques used for diagnostic and therapeutic procedures.

Box 3: Pain assessment scales in infants**Based on behavioural changes**

- Neonatal Facial Coding System (NFCS).
- Infant Body Coding System (IBCS).
- Neonatal Infant Pain Scale (NIPS).
- Pain assessment in Neonates (PAIN).
- Liverpool Infant Distress Scale (LIDS).
- Modified Behavioural Pain Scale.
- Children's Hospital of Eastern Ontario Pain Scale (CHEOPS).
- Neonatal Assessment of Pain Inventory (NAPI).
- Behavioural pain score.
- Clinical scoring system.

Combination of physiological and behavioural changes

- CRIES (acronym for crying, change in transcutaneous oxygen saturation, heart rate, blood pressure, facial expression and alteration in sleep pattern).
- Pain Assessment Tool (PAT).
- Premature Infant Pain Profile (PIPP).
- Scale for Use in Newborns (SUN).
- COMFORT Score.

Box 4: Consequences of pain in infants

Immediate effects

- Irritability.
- Fear.
- Disturbance of sleep and wakefulness state.
- Increased oxygen consumption.
- Ventilation-perfusion mismatch.
- Diminished nutrient intake.
- Increased gastric acidity.

Short term effects

- Enhanced catabolism.
- Altered immunological function.
- Delayed healing.
- Impaired emotional bonding.¹⁸

Long term effects

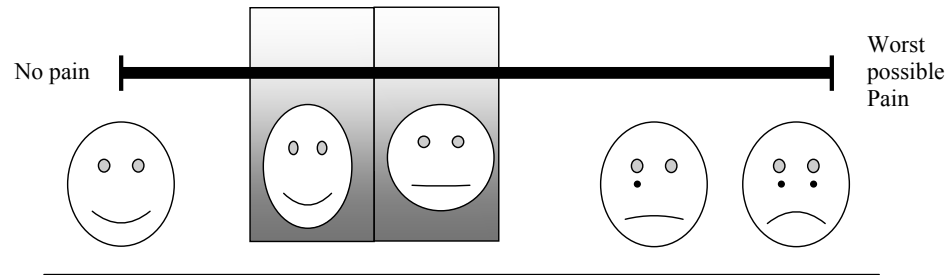
- Memory of pain.
- Developmental retardation.
- Alteration in response to subsequent painful experience.

Box 6: Non-pharmacological interventions to relieve infant pain

- Positioning and restraining the infant in a relatively flexed posture.²⁹
- Stimulation of nerve fibres transmitting tactile and thermal sensations.
- Combining these methods with soothing vocal stimulation.¹³
- Breast feeding.^{30 31}
- Feeding of sweet compounds such as sucrose,^{32 33} glucose,^{34 35} and saccharine.
- Non-nutritive sucking on pacifiers.^{36 37}

1. The Wong Baker Face Pain Scale:

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2. Oucher Scale: Combines a photographic faces scale with a 0 - 100 mm vertical numeric scale.
3. The Manchester pain Scale
4. Visual Analog scale: For children > 7 years.
5. Children's Hospital of East Ontario Pain (CHEOPS): 6 Categorioc of Pain from 1 - 3, > 6 Indicates Pain.

Cystic hygroma:

- Lymphangiomas of head & neck region
- Potency for continuous growth
- Manifestation depends on size & adjacent structures involved
- Cosmetic concern to acute life threatening emergency.

Pathophysiology:

- Failure of lymphatics to connect to the venous system
- Abnormal budding of lymphatic tissue & sequestered lymphatic rests with retained embryonic growth potential.
- Lymphatic rests can penetrate adjacent structures
- Spaces retain their secretions develop cystic components
- The nature of the surrounding tissue determines whether the lymphangioma is capillary, cavernous, or cystic

Anatomical sites:

- Neck (75%), Posterior triangle of neck
- Axilla (20%)
- Others :
 - Mediastinum
 - Groin
 - Retroperitoneum

History:

- Soft swelling in neck, face, oral cavity
- Progressive growth
- Acute increase in size (infection, bleed)
- OSA
- Stridor, cyanosis
- Feeding difficulty, failure to thrive
- Recurrent pneumonia, bronchiectasis

Demography:

- Frequency :1 case per 6,000-16,000 live births
- Mortality: 2-6% in some series (pneumonia, bronchiectasis airway compromise)
- Morbidity: Cosmetic disfigurement, Impingement on nerves, vessels, lymphatics
- Sex : Distribution is equal
- Age: 50-65% evident at birth, 80-90% by 2 yr, Abdominal ultrasonography by 10 weeks' gestation.

Physical examination:

- Painless, soft, compressible, fluctuant
- Transillumination positive
- Tracheal deviation & noisy breathing
- Close inspection of oral cavity large tongue, lax pharyngeal tissue
- Extension
 - Mediastinum - get below the swelling
 - Anterior neck (overlies trachea)

Associated syndromes in neonatal CH:

- Multiple pterygium
- Turners
- Noonan
- Pena-Shokeir
- Roberts Syndrome
- Downs Syndrome
- Cleftlip & palate, imperforate anus, cardiac defects and hydronephrosis

Specific preoperative investigation:

- Plain radiography: high-kilovolt antero posterior and lateral neck x-rays or airway fluoroscopy
- MRI: study of choice. best soft tissue detail, delineate the relationship of CH to underlying structures
- CT scanning: faster and readily available, the risk of radiation exposure, poor soft tissue delineation
- Ultrasonography: least invasive study demonstrates the relationship of CH to the surrounding structures limited ability in assessing mediastinal and retropharyngeal structures used to detect CH in utero.

Medical Management:

- Watchful waiting in asymptomatic pts
- Intralesional sclerosing agents
 - OK-432 (an inactive strain of group A Streptococcus pyogenes)
 - Bleomycin
 - Pure ethanol
 - Sodium tetradecyl sulfate
 - Doxycycline

Anesthetic concerns:

- Emergency vs. elective surgery
- Airway : difficult ventilation, intubation & tracheostomy
- Intubation under ketamine:
 - Rigid bronchoscopy & bronchoscopy (ENT Help).
- Post operative respiratory obstruction
- Pediatric age group concerns
- Significant blood loss Grouping & X matching

Airway management options:

- Inhalation induction (sevoflurane in O₂)
- Awake intubation under LA application
- Intubation under Ketamine sedation
- Rigid laryngoscopy/bronchoscopy (ENT help)
- Fiberoptic bronchoscopy
- Tracheostomy
- LMA

Preoperative preparation:

- Assess hydration – iv fluid therapy
- Identify maximum comfortable position – adapt it during induction
- Feeding difficulty & rec. cough – impending airway obstruction
- High risk parental consent
- Premedication – limit to antispasmodic only
- ENT surgeon standby (rigid bronchoscope)
- Check functional airway equipments
- Close communication with surgeon

Anaesthetic concern- extend of lesion:

- Extension into mouth
 - Poor feeding – dehydration, malnourishment pre op I.V fluids
 - Possibility of difficult airway.
 - Expert assistance/help/anesthesiologist.
 - Full range of pediatric airway should be available.
 - Facility for emergency tracheostomy should be kept ready.
 - Awake intubation
 - Surgeon should be stand by during induction.
 - Fibre optic intubation
 - Partial aspiration of the cyst should be done before intubation
 - Spontaneous respiration.

- Intra thoracic extension X ray chest
 - Wide mediastinum Prepare for sternotomy/ thorocotomy
 - PEEP can impede the thoracic duct flow.
Traction of airway by surgeon
- Pre tracheal region
 - Difficult tracheostomy, needs cyst aspiration
 - Post operative respiratory obstruction
 - Don't extubate
- Pharynx
 - Lax, redundant tissue
 - OSA & airway obstruction on induction
- Observation for complications.

Management of Post operative pain in Pediatric age group:**I. Minor Surgery:**

- Acetaminophen and NSAIDS +
- Peripheral nerve block, Caudal block +
- Wound infiltration when applicable.

II. Major Surgery:

- Abdominal, Thoracic, Orthopedic or Urogenital surgeries:
- Acetaminophen and NSAIDS +
- Opioids - Continuous IV opioids in children < 6 years, and PCA in a child > 6 years. OR
- Regional Anesthesia
 - Continuous epidural analgesia
 - Continuous paravertebral analgesia.

Non-Opioid analgesia for relief of pain in neonates and Infants:

Medicine	Neonates 0 - 29 days	Infants 1 month to 12 months	Maximum drug dosage
Paracetamol	5 - 10 mg/kg every 6 months	10 - 15 mg/kg every 4 - 6 hours	Maximum 4 Doses per day
Ibuprofen (Avoided in less than 1 month)	Avoided	5 - 10 mg/kg, Avoided in less than 3 months of age	4 mg/kg/day

Caudal anaesthesia (CA) is epidural anaesthesia of the cauda equina roots in the sacral canal, accessed through the sacral hiatus. CA is a common paediatric regional technique that is quick to learn and easy to perform, with high success and low complication rates. CA provides high quality intraoperative and early postoperative analgesia for sub-umbilical surgery. In children, CA is most effectively used as adjunct to general anaesthesia and has an opioid-sparing effect, permitting faster and smoother emergence from anaesthesia.

Indications for caudal anesthesia

The indications for single shot CA are:

- Abdominal,
- urologic
- orthopaedic surgical procedures located in the sub-umbilical abdominal,
- pelvic and genital areas,
- Lower limbs,
- Procedures where postoperative pain does not require prolonged strong analgesia. Examples of appropriate surgery include inguinal or umbilical herniorrhaphy, orchidopexy, hypospadias and club foot surgery.
- CA is useful for day case surgery, but opioid additives to the local anesthetic agent should be avoided in this setting.

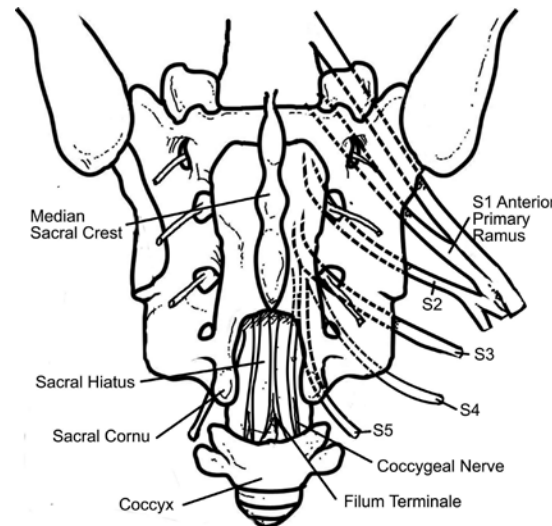
Contraindications

The usual contraindications to regional anaesthesia such as:

- coagulation disorders,
- local or general infection,
- progressive neurological disorders and
- patient or parental refusal apply to CA.
- Cutaneous anomalies (angioma, hair tuft, naevus or a dimple) near the puncture point require radiological examination (ultrasound, CT or MRI).

Anatomy**Anatomical landmarks:**

- The sacrum is roughly the shape of an equilateral triangle, with its base identified by feeling the two posterosuperior iliac processes and a caudal summit corresponding to the sacral hiatus.
- The sacrum is concave anteriorly.
- The sacral hiatus is located at the caudal end of the median crest and is created by failure of the S5 laminae to fuse
- The hiatus is surrounded by the sacral cornua, which represent remnants of the inferior S5 articular processes and which face the coccygeal cornua. Palpation of the sacral cornua is fundamental to locating the sacral hiatus and to successful caudal block.



- The sacral hiatus is the shape of an inverted U, and is covered by the sacro-coccygeal ligament, which is in continuity with the ligamentum flavum. It is large and easy to locate until 7-8 years of age.

The sacral canal

- The sacral canal is in continuity with the lumbar epidural space.
- It contains the nerve roots of the cauda equina, which leave it through anterior sacral foraminae.
- During CA, leakage of local anaesthetic agent (LA) through these foraminae explains the high quality of analgesia, attributable to diffusion of LA along the nerve roots.
- Spread of analgesia cannot be enhanced above T8-T9 by increasing injected LA volume.
- The dural sac (i.e. the subarachnoid space) ends at the level of S3 in infants and at S2 in adults and children. It is possible to puncture the dural sac accidentally during CA, leading to extensive spinal anaesthesia.
- Therefore the needle or cannula must be cautiously advanced into the sacral canal, after crossing the sacro-coccygeal ligament.
- The distance between the sacral hiatus and the dural sac is approximately 10mm in neonates.

Technique**Preparation**

- Obtain consent for the procedure either from the patient or, if appropriate, from the parents. After induction of general anaesthesia and airway control, the patient is positioned laterally (or ventrally), with their hips flexed to 90°.
- Skin disinfection should be performed carefully, because of the proximity to the anus.

- Aseptic technique should be maintained.
- According to the child's size, needle diameter and length are respectively between 21G and 25G, and 25mm and 40mm. A short bevel improves the feeling of sacrococcygeal ligament penetration and decreases risk of vascular puncture or sacral perforation.² Use of a needle with a stylet avoids risk of cutaneous tissue coring, and the (theoretical) risk of epidural cutaneous cell graft. If a stylet needle is not available, a cutaneous 'pre-hole' can be made with a different needle prior to puncture with the caudal needle. Another solution is to puncture with an IV catheter, the hollow needle of which is removed before injection through the sheath.

Puncture

- After defining the bony landmarks of the sacral triangle, the two sacral cornuae are identified by moving your fingertips from side to side.
- The gluteal cleft is not a reliable mark of the midline.
- The puncture is performed between the two sacral cornuae.
- The needle is oriented 60° in relation to back plane, 90° to skin surface.
- The needle bevel is oriented ventrally, or parallel to the fibers of the sacro-coccygeal ligament.

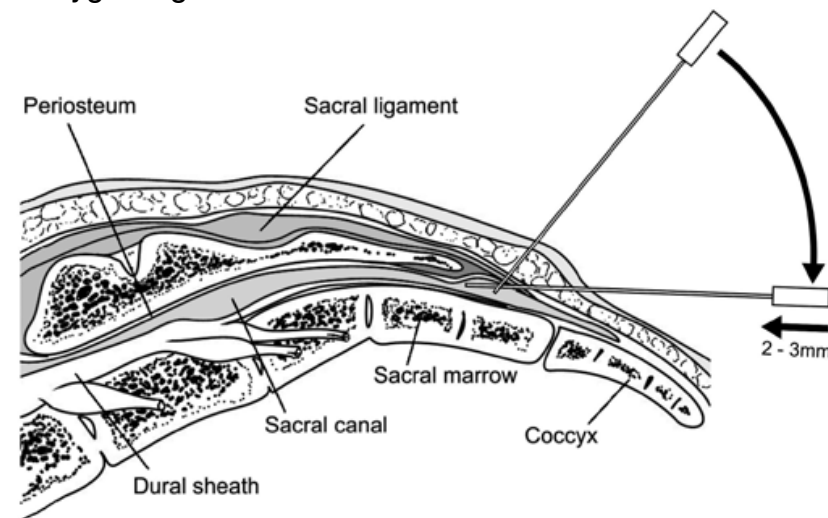


Figure 4. Puncture - orientation of the needle and reorientation after crossing the sacro-coccygeal ligament.

- The distance between the skin and sacro-coccygeal ligament is between 5 and 15mm, depending on the child's size. The sacro-coccygeal ligament gives a perceptible 'pop' when crossed, analogous to the ligamentum flavum during lumbar epidural anaesthesia. After crossing the sacro-coccygeal ligament, the needle is redirected 30° to the skin surface, and then advanced a few millimeters into sacral canal. If in contact with the bony ventral wall of sacral canal, the needle must be moved back slightly.
- After verifying absence of spontaneous reflux of blood or cerebrospinal fluid (more sensitive than an aspiration test), injection of LA should be possible without resistance. Inject slowly (over about one minute).

Local anaesthetic agents:
Test dose:

- A test dose of epinephrine 0.5mcg.kg⁻¹ (administered as 0.1ml.kg⁻¹ lidocaine with epinephrine 1 in 200 000) allows detection of intravenous injection with sensitivity and specificity close to 100%, under halogenated anaesthesia.

Full dose

- The volume of caudally injected LA determines the spread of the block and this must be adapted to surgical procedure (Table 1). Analgesic spread will be two dermatomes higher on the down positioned side at the time of puncture. Injected volume must not exceed 1.25 ml.kg⁻¹ or 20 to 25ml, in order to avoid excessive cerebrospinal fluid pressure.

Table 1. *Spread of block as a function of caudally injected local anaesthetic volume¹⁸*

Volume (ml.kg ⁻¹)	Dermatomal level	Indication
0.5	Sacral	Circumcision
0.75	Inguinal	Inguinal herniotomy
1	Lower thoracic (T10)	Umbilical herniorraphy, orchidopexy
1.25	Mid thoracic	

Table 2. *Maximal allowable doses of local anaesthetic agents*

	Plain local anaesthetic (mg.kg ⁻¹)	With epinephrine (mg.kg ⁻¹)	Neonates
Bupivacaine	2	2	
Lidocaine	3	7	↓ 20%
Ropivacaine	3	3	

Significant complications, in order of decreasing frequency, are:

- **Dural tap.** This is more likely if the needle is advanced excessively in the sacral canal when subarachnoid injection of local anaesthetic agent may cause extensive spinal anaesthesia. Under general anaesthesia this should be suspected if non-reactive mydriasis (pupillary dilation) is observed.
- **Vascular or bone puncture** can lead to intravascular injection and consequently LA systemic toxicity. Preventative measures are use of a test dose, cessation of injection if resistance is felt and slow injection under hemodynamic and ECG monitoring. Sacral perforation can lead to pelvic organ damage (e.g. rectal puncture).
- **Exceeding the maximal allowed LA dose** risks overdose and related cardiovascular or neurological complications.
- **Delayed respiratory depression** secondary to caudally injected opioid.
- **Urinary retention** - spontaneous micturition must be observed before hospital discharge.
- **Sacral osteomyelitis** is rare (one case report).

I. Problems Anticipated in pediatrics:

- Full stomach
- Open eye injury
- Airway Securing/Difficult airway
- IV Line access
- Hypothermia
- Hypoxia & Hypercarbia

II. Factors responsible for IOP:

Variables		Effect on IOP
CVP:	Increases	Increases IOP
	Decreases	Decreases IOP
Arterial:Blood Pressure	Increases	Increases IOP
	Decreases	Decreases IOP
PaCO₂	Increases	Increases IOP
	Decreases	Decreases IOP
PaO₂	Increases	No Effect
	Decreases	Increases IOP

Variables		Effect on IOP
Inhalational Agents:	Volatile Agents	Decrease IOP
	Decreases	Decreases IOP
Intravenous Agents	Barbiturates	Increases IOP
	BNZ	Decreases IOP
	Ketamine	Increases IOP/?
	Opioid	Decrease IOP
Muscle Relaxants	Depolarizers	Increases IOP
	Non - Depolarizers	Decreases IOP

III. Anesthesia & IOP:

- Causes Increase in IOP are:
 - Laryngoscopy & Endotracheal Intubation, coughing staining the valsalva or vomiting.
 - CNS Depressants: Decrease IOP relaxes extra ocular muscular tone, depress the CNS(i,e The Diencephalon) improves the out flow of aqueous humor decreases aqueous production & lower venous and arterial blood pressure.
 - Succinylcholine increases IOP for 4 - 6 min and ketamine increases IOP as well.

Succinylcholine:

- Transient Increase in IOP
- 8 to 10 mm Hg
- Increase within 1 min & Peaks within 6 min.
- Tonic contractions of extra ocular muscles.
- Vascular event - choroidal vascular dilatation.
- Increased Drainage secondary to increased CVP.

Eye Injuries:

- Traumatic
 - Penetrating
 - Non - Penetrating
- Non - Traumatic

Aim of Anesthetic Management:

- Safety
- Akinesia
- Profound Analgesia
- Minimal bleeding
- Avoid ocular reflex
- Prevent Increase IOP
- Smooth Emergence

Open eye - Full Stomach Problems:

- Concerns about airway
- Extrusion of ocular contents
- Aspiration Prophylaxis
- RSI
- Succinylcholine? with pretreatment
- Avoid cough/straining/HTN

Anesthetic Management:**Premedication:**

- Aspiration Prophylaxis
 - Inj. Metoclopramide 10 mg IV
 - Inj. Ranitidine 50mg IV - H2 Antagonist

Intraoperative Management:

- Pre oxygenation X 3 min
- Rapid sequence Intubation
- Succinylcholine - Adequate induction, pre-treatment with NDMR
- Smooth anesthesia & Extubation
- Rocuronium 0.6 mg/kg - Good substitution for rapid sequence induction.
- A dose of 0.9 to 1.2 mg/kg dose of rocuronium - equivalent intubating condition to succinylcholine.
- Disadvantage is recognized or unrecognized difficult airway.

Priming Principle:

- 1/10th of an intubating dose of NMDR, followed 4 min later by intubating dose, after waiting an additional 90 sec, trachea may be performed.
- Stomach decompression during surgery to be done.

Extubation:

- Stomach decompression during surgery
- Narcotic 10 - 15 min before extubation and lignocaine 5 min before extubation.
- Awake extubation.

Post Operative Management:

- Control of post operative pain
- Non - Opioids - Paracetamol, Ibuprofen, Ketoralac, Diclofenac
- Opioids and Anti emetic
- Droperidol = 0.5 to 1mg in adults up to 3 times a day
- Post operative Nausea & Vomiting
- Ondansetron - Children 0.1mg/kg
- Dexamethasone.

Grid: If question asked in the exam answer under following headings:

1. Problems in pediatric patients
2. Factors responsible for IOP
3. Anesthesia for IOP
4. Problems with Succinylcholine
5. Types of eye injuries
6. Aims of Anesthetic management
7. Open eye with full stomach
8. Anesthetic management:
 - 8.1. Pre - Medication
 - 8.2. Intraoperative Management
 - 8.3. Priming Principle
 - 8.4. Extubation
 - 8.5. Post operative Management.

I. Pre assessment & Planning:

- Interdisciplinary communications & co-operation is vital to the success of separation operation.
- Dedicated team for anesthesiologist for each child
- Duplication of all monitoring equipment.

II. Pre operative evaluation:

- Routine blood and urine analysis.
- Coagulation screening & Plain X - Ray USG
- CT - Scan, Digital subtraction angiography to delineate anatomic & bone details, demonstrate organ position, shared viscera and limited vascular anatomy.
- Planning of post operative period in ICU.
- Planning of reconstructive & Rehabilitation
- Arrangement of blood & Blood products.
- 8 weeks prior to actual separation surgery, Plastic surgeons place tissue expanders, in the place of separation to gain skin expansion for the closure of raw area
- Anesthesia with LMA placement 3 times for tissue expanders may be required.
- Presence of cross circulation can be tested by given atropine administration to one twin and no change in the heart rate observed in other twin.
- Sequential Induction when there is little or no cross circulation.

Pre-Medication:

- Inj Glycopyrolate 0.01 mg/kg
- Inj Ranitidine
- Midazolam 2 mg to each child.

Monitors: Two monitors for two children

- 2 ECG
- 2 SpO₂ probes
- 2 urine out put
- 2 Temperatures
- 2 NIBP are attached

Induction:

- IV Fluids - Warmed
- Antiseptic - Warmed
- OT temperature to be warmed
- Oxygen + N₂O + Sevoflurane once in deeper planes good IV access to be used. (20 G if possible 18G cannula)
- Fentanyl 3micrograms/Kg
- Propofol 2mg/kg
- Pancuronium 0.05 mg/kg
- Mask Ventilation + Nasotracheal/oral intubation done with appropriate size tube
- Lines Radial artery & CVP with help of USG.
- Urine output, temperature, antibiotics for both the babies are given.
- Aprotinin 1ml/kg 100000 KIU to prevent massive blood loss.

Maintenance of Anesthesia:

- O₂ + N₂O + Isoflurane + Intermittent Pancuronium + Fentanyl to each twin.
- Pressure controlled ventilation
- Peak plateau pressure maintained at 15 - 16 cm of H₂O

Fluids:

- D5 =0.45% NS using 4:2:1 formula to replace deficits
- RL for maintenance fluid
- 3rd Space replaced by RL

- RL:Blood Ratio = 3:1
- Colloid : Blood Ration = 1:1
- Blood : Blood Ration = 1:1
- Acid Base balance to be corrected
- Electrolytes to be corrected.
- Blood pressure to be maintained.

Post operative:

- Ventilation for 24 to 48 hours
- Adequate pain relief
- Extubate only when good hemodynamics are maintained
- Look for neurological deficits

In Presence of Cross Circulation:

- Cross circulation to be established by Tc99 m injection of micro-colloidal human serum albumin & its excretion in urine of the other twin.

Recommended IV Dose:

- Dose o anesthetic agents for combined body weight of the twin are usually halved and then divided into two equal dose to be administered to each twin.
- Reduced incremental dose are titrated against response and help minimize the dangers of compound drug effects in one twin.

Anesthetic Consideration:

- Unusual position
- Blood loss
- Prolonged surgery
- Same operating table
- Difficult airway
- Cross circulation
- Drug dosing
- Dual Equipment.

- Dual Anesthetics
- Dual Monitoring